Growth, Development, Malnutrition

- Sequence of sexual characters:
  - Girls: Thelarche (10 years) → Pubarche → Peak Growth → Menarche
  - Boys: Testicular growth (11.5 years)→ Pubarche → Peak growth → Beard/Spermarche

- 1st peak growth → IU period - infant → Insulin is the main hormone responsible for IU growth.
- 2nd peak growth → Adolescent/Pubertal

- Growth Assessment Markers:
  1. Weight of a child
  2. Head circumference of a child } Age dependent marker
  3. Length/height of a child
  4. Mid-arm circumference of a child → Age independent marker

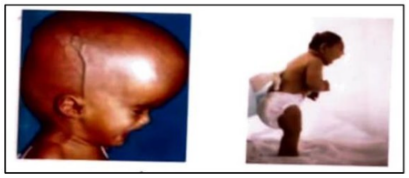
1. Weight of a child:
   - Term: fall of 5-10% body weight → Regain birth weight by 10 days of life
   - Preterm: fall of 10-15% body weight → Regain birth weight by 14 days of life
   - Weight doubles by 5 months, three times at 1 year, four times at 2 years, five times at 3 years, six times at 5 years, 7 times at 7 years, 10 times at 10 years.

2. Length/height of a child
   - Length measured in supine position till 2 years of age by Infantometer
   - Height measured in standing position after 2 years of age by Stadiometer
   - Length at birth: 50cm
   - Length at 1 year of age: 75cm
   - Length at 2 years of age: 85-90cm
   - 2-12 years of age: Increases at 6cm/year

3. Head circumference:
   - At birth: 35cm
   - 3 months: 40cm
   - 6 months: 43cm
   - 1 years: 46cm
   - 2 years: 48cm
   - When child is born: HC > Chest circumference by 3cm
   - Head circumference = Chest circumference at about 9 months - 1 year.

4. Mid Arm Circumference of a child:
    - Age independent marker
    - Measured between 6 months - 5 years (1 year - 5 years)
    - Shakir's tape (3-color tape) used to measure MAC:
        - Red: <11.5cm
        - Yellow: 11.5-12.5cm
        - Green: >12.5cm

- Large Head (macrocephaly) vs Small head (microcephaly):



Macrocephaly Microcephaly

    - Macrocephaly: HC >2SD
    - Microcephaly: HC <3SD

- Causes of macrocephaly:
    - Hydrocephalus
    - Hydrancephaly
    - Hemorrhage (brain)
    - Leukodystrophy: Alexander's disease & Canavan disease
    - Lysosomal storage disease (some)
    - Neurocutaneous disorder: Neurofibromatosis and Tuberous sclerosis
    - Sotos syndrome (Cerebral gigantism)

- Microcephaly:
    - Isolated Microcephaly
    - Trisomy 13, 18, 21
    - TORCH infection
    - Fetal alcohol syndrome
    - Fetal hydantoin syndrome
    - Teratogen
    - HIE (Hypoxia)
    - Rett syndrome

SHORT STATURE IN CHILDREN

29:42

- Static height of child <2SD of expected

- It is divided into:
  I. Normal variant (Physiological short stature) → Growth velocity is normal
     A. Familial short stature: Child's final stature remains short because of decreased growth in first 2 years.
        - Short parents
        - Bone age = chronological age
     B. Constitutional delay (in growth and puberty): Final stature is normal because of compensation for slow growth in 1-3 years by growing faster during adolescent period.
        - Delay in puberty
        - It is the most common cause of short stature in children
        - Bone age < Chronological age

  II. Pathological short stature → Growth velocity is reduced
     Based on US/LS segment:
        - 1.7 at birth
        - 1.3 at 3 years
        - 1.1 at 6 years
        - 1 at 7-10 years
        - <0.9 after 10 years
     A. Disproportionate:
        1. Congenital hypothyroidism
        2. Achondroplasia
        3. Rickets
     B. Proportionate:
        1. Failure of growth: SGA and malnutrition
        2. Endocrinal disorders
        3. Extended diseases (CKD, Celiac disease)
        4. Turner syndrome (chromosomal disorders)

- Chromosomal disorders causing short stature:
  1. Turner syndrome
  2. Noonan syndrome
  3. Down syndrome
  4. Prader Willi syndrome
  5. Russel Silver syndrome

6. Seckel syndrome

MILESTONES IN CHILDREN 43:50

- All milestones develop in a cephalocaudal sequence.

- It is broadly divided into:
  1. Gross motor
  2. Fine motor
  3. Language
  4. Social

- Gross motor milestones:
  - Sitting with support: 5 months
  - Sitting without support: 8 months

  - Crawling: 8 months
  - Creeping: 10 months
  - Creeping stairs: 15 months

  - Stands with support: 9 months
  - Stands without support: 12 months
  - Walks with support: 10-11 month
  - Walks without support: 13-15 month

  - Climbs stairs: 2 years (maturely by 4 years)

Crawl Creeping

- Fine motor milestones:
  - Bidextrous approach: 4 months
  - Unidextrous approach: 6 months

- Ulnar grasp: 6-7 months
- Radial grasp: 8-9 months

- Pincer grasp (immature): 9 months
- Pincer grasp (mature): 12 months

- Transfer objects: 5-7 months

- Tower of 2 cubes: 15 months
- Tower of 6 cubes: 2 years
- Tower of 9-10 cubes: 3 years

- Copies line: 2 years
- Copies circle: 3 years
- Copies square/rectangle: 4.5 years
- Copies triangle: 5 years
- Copies hexagon: 6 years
- Copies rhombus: 7 years
- Copies cylinder: 9 years
- Cup/spoon with spilling: 10-12 months
- Turns each page singly: 2 years

- Language milestones:
  - Monosyllables: 4-5 months
  - Babbling: 6 months
  - Bisyllables: 9 months
  - 2-3 words with meaning: 1 year
  - Jargons: 15 months (4-10 words)
  - Sentences: 2 years

- Social milestones:
  - Social smile: 6-8 weeks
  - Recognizes mother: 12 weeks
  - Mouthing: 5-6 months
  - Plays with mirror image: 6 months

  - Waves bye bye: 9 months
  - Casting: 1 year
  - Plays Peek-a-boo: 10-12 months

- Dresses/undresses himself: 3 years

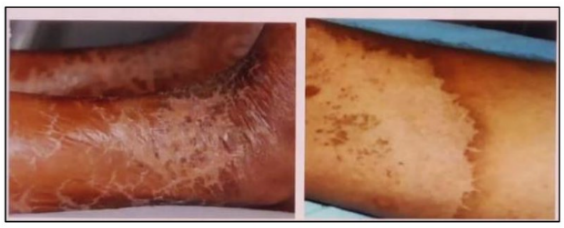
- Gender/name/color identification: 3 years
- Right/left hand discrimination: 4 years???
- Bladder control (complete): 5 years → Diagnosis of enuresis after 5 years
- Bowel control (complete): 4 years → Diagnosis of encopresis after 4 years

- Enuresis:
  - Best: Alarm therapy
  - Pharma: Desmopressin and anticholinergic [Oxybutynin and Tolterodine]
  - Oral desmopressin is the drug of choice for enuresis in children

MALNUTRITION IN CHILDREN

01:01:37

Kwashiorkor	Marasmus
Pitting edema	Severe wasting/no edema
Fat sugar baby appearance	Monkey facies Baggy pant appearance
Apathetic or irritable	Alert
Poor appetite	Voracious appetite
Hair lustreless, loss, easily pluckable, alternating hypo and hyperpigmentation (Flag sign)	
Skin changes "Crazy pavement dermatosis" and "Flaky paint dermatosis"	



Crazy Pavement appearance Flaky Paint appearance

- Severe acute malnutrition:
  - Diagnostic criteria: For children between 6 months-59 months of age.
    - Bilateral pedal edema
    - Weight for height < -3SD scores/-3 Z-score
    - MAC <11.5cm

- Management:
  - Hospital admission criteria: appetite absent, complications, severe edema, danger signs [unconscious, lethargic, persistent vomiting, unable to drink, seizures]
  - Home: above features are absent

- Complications of malnutrition:
  a. Hypoglycemia: Blood sugar <54mg/dl
  b. Hypothermia
  c. Infections
  d. Electrolyte abnormality: hypernatremia, hypokalemia and hypomagnesemia
  e. Dehydration
  f. Deficiency of micronutrients

- Never give iron in first week of management as it is known to compromise infection management.
- Calories: 75 Kcal/kg/day → 100 Kcal/kg/day → 175 Kcal/kg/day
- Starting with high calories can lead to Refeeding syndrome: Hypophosphatemia, hypokalemia, hypomagnesemia, Vitamin B_1 deficiency

Update - Milestones

1. Gross motor
2. Fine motor
3. Language
4. Social → "Handedness"
    - At the age of 3 years it develops

Neonatology

NEWBORNS

00:40

- L – 50 cm
- HC – 35 cm
- HR – 110–160/min
- RR – 40–60/min
- Normal temperature – 36.5 – 37.5°C; <36.5 – Hypothermia
  > 37.5°C – Hyperthermia

1. 36–36.5°C: Mild/cold stress
2. 32–36: Moderate
3. <32°C: Severe

- SGA or IUGR –B wt < 10th percentile (Small for gestational age)
- AGA – B wt 10th – 90th percentile (appropriate for gestational age)
- LGA – B wt > 90th percentile (large for gestational age)

- Preterms < 37 weeks
- Terms 37–42 weeks
- Postterm > 42 weeks

Neonate passes meconium by 24 hours
Urine by 48 hours

IUGR has signs of intrauterine malnutrition like- small umbilical cord wrinkles on skin

- IUGR subdivided into:-
  1. Asymmetrical IUGR – later part of pregnancy (Better prognosis)
  2. Symmetrical IUGR – early pregnancy problems

Umbilical cord has 2 arteries + 1 vein; falls by 7–10 days (max 2 weeks)

Single umbilical artery – congenital genitourinary defects such as U/L renal agenesis

- Timing of appearance and disappearance of primitive neonatal reflexes

		Appears	Disappears
1.	Grasp reflex:	28 wks IU life	3 months
2.	Rooting reflex:	32 wks IU life	1 month
3.	Moro's reflex	28–32 wks	3–6 months
4.	Asymmetric tonic neck	35 wks	6 months

NOT PRIMITIVE (AFTER BIRTH)

21:14

1. Symmetrical tonic neck reflex
2. Parachute reflex

Preterm child	Postterm child
• Thin, red skin • Abundant hairs (lanugo) • Sole creases superficial/absent • Poor elastic recoil of ears	• Pale desquamated skin • Long nails • Absent lanugo • Loose skin folds • Often meconium stained

COMPLICATIONS OF PRETERM (PREMATURE BABIES) *29:00*

A - Apnea: temporary stoppage of breathing > 20s or <20s associated with: bradycardia, cyanosis, pallor
Apnea of prematurity T/E- caffeine citrate

B - Bronchopulmonary dysplasia (BPD) - O_2 induced inflammatory lung injury
- Continuous O_2 requirement till D28
- Mild, moderate, severe states
- O_2 requirement at time of discharge
  Mild - room air
  Moderate - 22-29% O_2
  Severe - $\geq$ 30% O_2

C - Colitis (Necrotising enterocolitis)
↳ Inflammatory injury to intestine
↳ Preterm + Formula feeding ↑ risk
- Symptoms start from 2nd - 3rd week

Abdominal distension
Bleeding in stools
↓ bowel sounds
Anemia
Neutropenia
Thrombocytopenia
Metabolic acidosis

- When gas enters intestinal wall - Xray abdomen
  ↓
  Pneumatosis Intestinalis } Pathognomic sign

D - Ductus Arteriosus (PDA)

E - Extended hospitalisations

F - Feeding difficulties
>34 wks - Breast feeds
32 - 34 wks - spoon / Paladai feeds
28-31 wks - orogastric feeds
>28 wks - I.V fluids

G - M/c intracranial bleeding - "Germinal matrix haemorrhage"

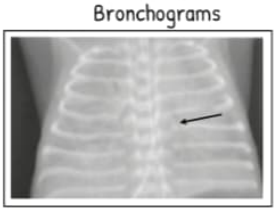
H - Hypoglycemia: Blood sugar < 40mg / dl → T/t
Hypocalcemia: Serum Ca^{2+} < 7mg/dl (Preterm)
< 8mg/dl (Term)
Hyaline membrane disease
Respiratory distress syndrome

m/c cause of RD in preterm (<34 wks)
or, surfactant deficiency

Hypoglycaemia
- Symptomatic → IV dextrose
- Asymptomatic
  - <20mg/dl → IV
  - 20-40 mg/dl → Oral feeds

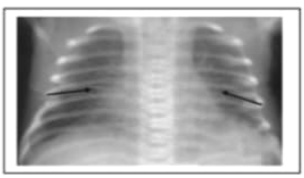
Hyaline membrane disease / RDS
- Common <34wk
- M/c cause of RD in preterm
- Chest x-ray:-
  1. Ground glass opacity
  2. Reticulogranular opacity
  3. Air bronchogram

Ground glass opacity with Air Bronchograms



Transient Tachypnea of newborn (TTB)
- M/c cause of RD in late preterm or term
- Persistence of fetal lungs fluid
  Seen in: C-section
  Precipitous delivery

Chest X-ray: Fluid in lungs
↓
Prominent interlobar fissure



T/t: CPAP – for mild
Mechanical ventilation
For severe RD

- Surfactant administration
  - Prophylactic
  - Therapeutic
- Via MIST (minimally invasive surfactant therapy)
- Or via LISA (less invasive surfactant administration

T/t: Symptomatic management

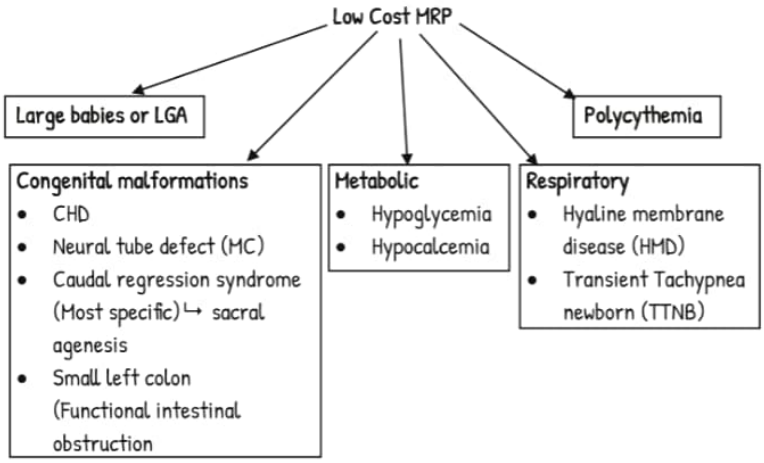
I – Ischaemic brain injury
↓
Perinatal asphyxia

- Apg score <3 for >5 mins — Brain involvement
- Cord artery pH <7 — HIE
- Multiple organ dysfunction — Hypoxic ischaemic Encephalopathy

H/E m/c cause of neonatal seizures

- Periventricular leukomalacia

Complications in infants of diabetic mothers



NEONATAL SEPSIS *1:38:52*

Early-onset sepsis (within 72 hours of birth) – Klebsiella, E coli, (organism from maternal genital tract)
Grp B streptococcus, CONS

Late-onset sepsis (after 72 hours of birth) – Coagulase – negative Staph (CONS), Staphylococcus
↳ Organism environment

Diagnosis approach – Sepsis screen + Lumbar Puncture + Blood culture

- TLC < 5000/mm^3
- ANC < 1800/mm^3
- Immature to Total Neutrophil ratio > 20%
- CRP > 1 mg/dl
- microESR ≥ 15 mm in first hour

if ≥ 2 of sepsis screen positive – NEONATAL SEPSIS

Treatment – Antibiotics

NEONATAL RESUSCITATION *1:45:06*

Born
↓
Term / Breathing Regular / Tone good

Yes → 90% Routine care

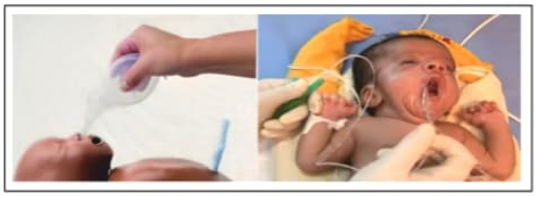
No → Resuscitation (10%)

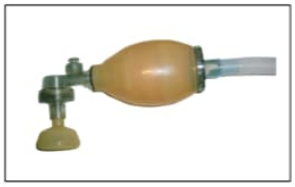
- Warmer
- Clean secretion (no routine suction)
- Dry
- Stimulate

>Primary apnea: HR↓
BP (N)
Respond tactile management

>Secondary Apnea: HR ↓
BP ↓
Respond to PPV
↓
Apnea HR <100/m (within 60 s of birth)
↓
PPV (positive pressure ventilation)

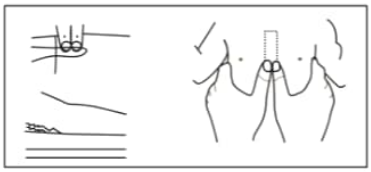
Suction bulb | Suction catheters





Self Inflating Ambu Bag (for PPV)

Start → 30–40cm H_2O
Maintain → 15–20 cm H_2O
Rate – 40–60/min
>35 wk & Term – 21% O_2
<35 wk Preterm– 21–30% O_2



Chest compression
Chest compression to ventilation ratio 3:1

Revaluate after 15s → No improvement in HR

M – Mask adjust
R – Reposition baby
S – Suction
O – Open mouth
P – Pressure
A – Alternative airways (intubation / Laryngeal Mask airway)
↓ Total PPV 30s reassess HR < 60/m
Chest compression (3:1 ratio)
↓ Reassess HR; no improvement
Epinephrine

NEONATAL JAUNDICE

02:03:44

Physiological jaundice
- Usually appears D2/D3
- Disappears by 2 weeks
- TSB < 15 mg/dl

Pathological jaundice
- Appears first 24 hours
- Persists beyond 2 weeks
- TSB >15 mg/l
- TSB rises >0.2mg/dl/hr OR 5mg/dl/day

Important pathological causes of jaundice in newborns
1. ABO / Rh incompatibility / TORCH infection - first 24 hr
2. Congenital hypothyroidism
3. Hereditary detects in bilirubin metabolism ——— Crigler- Najjar syndrome
   Gilbert syndrome
   Dublin Johnson syndrome
   Rotor's syndrome
4. Cephalhematoma
5. Cholestasis

BREAST MILK JAUNDICE vs BREAST FEEDING JAUNDICE
↳ Persists 3-10 wks └— (Persist for 2-3 wks)

Phototherapy – Best treatment approach for neonatal jaundice
Mechanism by which phototherapy reduces bilirubin
- Structural isomerization (forms lumirubin; most effective method)
- Configurational isomerization (Z - isomers to E-isomers)
- Photo - oxidation products

Wavelength: Blue-Green light (460-490 nm)

Side effects
- Dehydration
- Retinal damage
- Hypocalcemia
- Bronze baby syndrome

Exchange Transfusion - Aggressive reduction in neonatal jaundice when kernicterus risk likely.

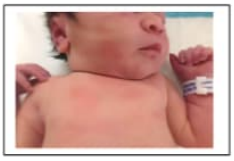
Phototherapy | Exchange Transfusion

Pinch to zoom

Kernicterus – bilirubin induced brain damage (basal ganglia)

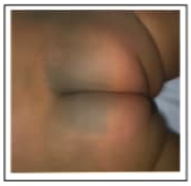
S/E: hypoglycemia
Hypocalcemia
Hyperkalemia
Metabolic alkalosis
Problems related to catheterisation

Pictorial diagnosis in newborns



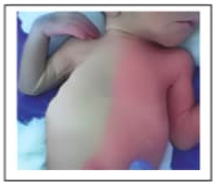
Erythema Toxicum

- Mc erythematous rash in newborn
- Eosinophils
- No T/t required



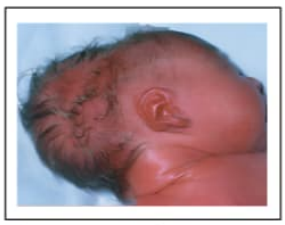
Mongolion spots

- Blue patches
- Particularly common in lumbosacral area
- Disappear on own by 1 yr



Harlequin color

- Due to immature tone of blood vessels



Caput secundum vs Cephalhematoma

Caput Secundum	Cephalhematoma
• Fluid collection	• Blood collection
• Appear in 12-24 hr of birth	• Late appearance
• Disappear in 2-3 days	• Disappear in 3-6 wks
• Gross sutures	• Limited by sutures

Pediatric Cardiology

FETAL CIRCULATION 00:43

- Placenta oxygenates → Umbilical vein → Ductus venosus (bypasses hepatic circulation) → Enters Inferior Vena Cava → Oxygenated blood enters right side of heart (Right atrium); Eustachian valve is present just at entry of Right atrium → Enters Left atrium through foramen ovale → Left ventricle → Aorta → Provides oxygenated blood to brain, heart and rest of body
- Deoxygenated blood from upper half of the body → Superior Vena Cava → Right atrium → Right Ventricle → Pulmonary artery → Fetal lungs are in a state of physiological pulmonary hypertension (only 5% of deoxygenated blood enters lungs) → Remaining 95% of blood shunts through Ductus arteriosus from pulmonary artery to aorta; It attaches just distal to left subclavian artery → Umbilical artery → Placenta (for oxygenation)

AFTER BIRTH 11:25

- Superior vena cava and Inferior vena cava bring deoxygenated blood into right atrium. → Enters right ventricle through tricuspid valve → Pulmonary artery arises from right ventricle → Lungs (blood gets oxygenated) → 4 pulmonary veins enter into left atrium → Enters left ventricle via bicuspid/mitral valve → Aorta: 3 arteries arise:
  - i. Brachiocephalic artery → Right subclavian artery and right carotid artery
  - ii. Left carotid artery
  - iii. Left subclavian artery

CONGENITAL HEART DISEASES 16:52

- Classification:
  - A. **Acyanotic:**
    - I. Left to Right shunts:
      1. ASD
      2. VSD
      3. PDA
      4. Atrioventricular septal defect (Endocardial cushion defects)
    - II. Ventricular obstructions:
      1. Aortic stenosis
      2. Pulmonary stenosis
      3. Coarctation of aorta

B. **Cyanotic:**
   I. Decreased pulmonary blood flow:
      A. Right Ventricular Hypertrophy: TOF
      B. Left Ventricular Hypertrophy: Tricuspid atresia

   II. Increased pulmonary blood flow: associated with recurrent chest infection.
      1. Transposition of great arteries/great vessels
      2. TAPVR/TAPVC (Total Anomalous Pulmonary Venous Return)
      3. Truncus arteriosus: common trunk serves as aorta and pulmonary artery
      4. Hypoplastic Left Heart Syndrome: 3 structures of left side of heart do not develop properly:
         i. Ascending aorta
         ii. Left ventricle
         iii. Mitral valve

- Exam points: Some CHD:
  - **Atrial Septal Defect:**
    - Defect in atrial septum which allows blood to move from left atrium to right atrium.
    - It is of 3 types:
      a. Ostium Primum defect (lower part) → MC type ASD seen in Down Syndrome
      b. Ostium Secundum defect (middle part) → Most common type
      c. Sinus venosus defect (upper part)
    - Size of RA, RV and pulmonary artery increases
    - Size of LA is not increased (unlike VSD)
    - Wide fixed splitting of S_2

  - **Ventricular Septal Defect**
    - Overall, most common CHD
    - Defect allows blood to move from LV to RV
    - It is of three types:
      a. Muscular VSD (lower part):
         - Multiple defects
         - Swiss Cheese Septum

b. Infracristal/Perimembranous VSD (middle part) → Most common type
c. Supracristal VSD (upper part) → Associated with collapse of right coronary leaflet if AV → Associated with aortic regurgitation.

- Presentation: symptoms of heart failure from 4-10 weeks of age.
- High risk of development of PAH
- Increased risk of infective endocarditis [MC CHD]

- **Patent Ductus Arteriosus:**
  - Open connection between PA and aorta (just distal to subclavian artery)
  - Presents with symptoms of heart failure from 4-10 weeks of age
  - Normal DA closes by 10-21 days after birth
  - In Intrauterine life: high prostaglandins and low O_2 of uterus keep the Ductus arteriosus patent.
  - After birth: Prostaglandins disappear and High O_2 in environment → Ductus arteriosus closes.
  - There are 2 ways in which ductus arteriosus can be closed:
    a. Medical: Prostaglandin inhibitors –
       i. Indomethacin
       ii. Ibuprofen
    b. Surgical
  - Medical therapy can be attempted within 2 weeks after which only surgical treatment should be done.
  - Continuous murmur:
    a. PDA
    b. Severe CoA
    c. Systemic AV fistulas
    d. Coronary AV fistula
    e. Rupture of sinus of valsalva

- Eisenmenger syndrome:
  - Left to right shunts → Pulmonary arterial hypertension and RVH → R to L shunt (reversal of shunt)
  - Eisenmenger complex: Eisenmenger syndrome + VSD

- Coarctation of aorta:
  - Constriction in arch of aorta distal to origin of left subclavian artery; Hence, it is called as Juxta-ductal narrowing.
  - Symptoms occur due to difficulty of blood going in lower half of body:
    a. Poor pulses in lower extremity.
    b. Upper extremity hypertension
    c. Radiofemoral delay
    d. Pain of intermittent claudication
  - How collaterals help bypass the obstruction?

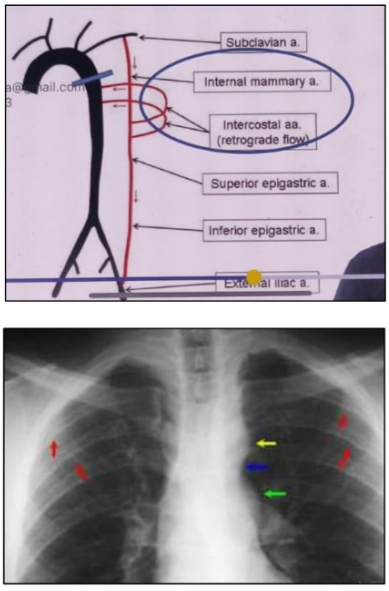


Figure of 3 sign (CoA)

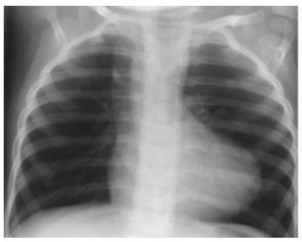
  - Notching of lower end of ribs due to dilatation of intercostal arteries.

CYANOTIC HEART DISEASES *01:10:20*

A. Tetralogy of Fallot (TOF)
  - Most common cyanotic CHD in children
  - Cyanosis after 1 year of age
  - Trilogy of Fallot: PS + RVH + ASD
  - Tetralogy of Fallot: PS (Subvalvular, valvular or supravalvular) + RVH + VSD + Aorta overrides VSD

- Pentad of Fallot: TOF + ASD
- Large VSD: Heart failure are very rare → CHD which carries least risk of Heart failure.
- During infancy: "Tet spells"/" Hypercyanotic spells" (2-6 months of age)
- During periods of crying/irritability →Increased release of catecholamines → Closure of pulmonary infundibulum (Subvalvular)
- Blood moves from RV to LV → Cyanosis spells

- **Treatment of Tet spells:**
  1. Squatting
  2. Knee chest position
  3. Systemic vasoconstrictors (IV Phenylephrine)
  4. β-blockers (propranolol, esmolol)
  5. Oxygen
  6. Subcutaneous morphine
  7. Sodium bicarbonate

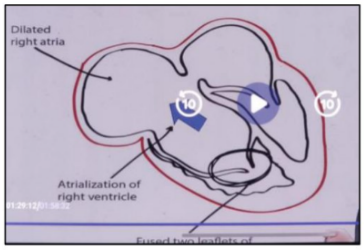


Boot shaped heart (Cour en Sabot)

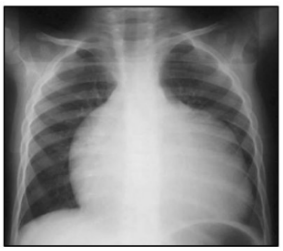
- **Important examination points:**
  1. No cardiomegaly
  2. Very low risk of heart failure
  3. Second most common CHD that is associated with right aortic arch
     Most common CHD associated with Right aortic arch: Truncus arteriosus

B. Tricuspid atresia:
- Cyanotic CHD associated with reduced pulmonary blood flow and LVH.
- Absence of tricuspid valve → All blood from IVC & SVC → RA → LA → LVH
- Right ventricle becomes very small or hypoplastic

C. Ebstein anomaly:
- Disorder of tricuspid valve: Two leaflets of tricuspid valve are fused with the apex of heart



- When RV contracts, it sends blood back to RA as blood is unable to enter pulmonary artery.



Box shaped heart (Ebstein Anomaly)

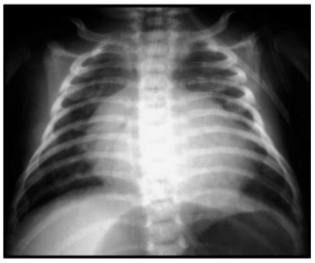
- **Important points:**
  1. Associated with lithium intake in pregnancy
  2. Most common CHD associated with arrhythmia
  3. Box shaped heart

D. Transposition of Great Arteries/Vessels (TGA)
- Most common cyanotic CHD in newborn period.
- Aorta arises from right ventricle and PA arises from left ventricle.

- **Types**:
  a. d-TGA: Aorta lies anterior and to right of PA. → Most common type
  b. l-TGA: Aorta lies anterior and to left of PA

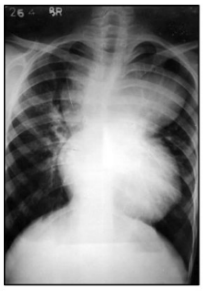
- **There are 2 parallel circulations happening without any mixing:**
  - RV → Aorta → Body → RA → RV
  - LV → PA → Lungs → LA → LV
- Unless there is mixing, child cannot survive.
- Procedure which is helpful in improving symptoms: Atrial septostomy → Mixes deoxygenated and oxygenated blood.
- Another option: Keep Ductus arteriosus patent → allows mixing of blood.

- Duct dependent CHD: Dependent on patent DA for mixing of blood (PGE1 infusion: Alprostadil)
  - **Duct dependent CHD are of Three types:**
    a. Maintain pulmonary circulation: Tricuspid atresia, TOF
    b. Mixing of oxygenated and deoxygenated blood: RGA
    c. Maintain systemic circulation: Hypoplastic left heart syndrome and severe COA.



Egg on a string appearance (d-TGA)

E. TAPVR/TAPVC:
  - **4 pulmonary veins enter into RA instead of LA either:**
    a. Supracardiac: Going above the level of heart (Most common type)
    b. Infracardiac: Going below the level of heart through ductus venosus
      - Since Ductus venosus closes after birth, hence Infracardiac TAPVR can become obstructive.
    c. Cardiac: Directly open into RA

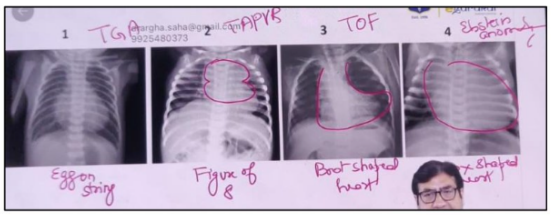


Snowman/Figure of 8 appearance (Supracardiac TAPVR)

  - RA receives all the blood → Some of which may enter LA through foramen ovale.

F. Hypoplastic Left Heart Syndrome:
   - Hypoplasia of 3 structures:
     1. Ascending aorta
     2. Left ventricle
     3. Mitral valve

   - Circulation: 4 PV → LA → RA → RV → PA → Through ductus arteriosus, some systemic circulation can be maintained. Hence, it is Duct dependent CHD.

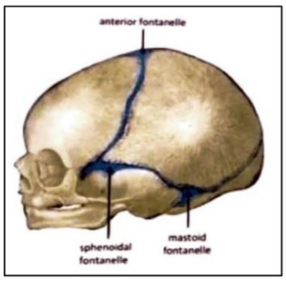


- Some syndromes and important CHD:
   - Holt Oram Syndrome: ASD (with absent radius bone + heart blocks)
   - Lutembacher syndrome: ASD with MS
   - Down syndrome: Endocardial/Atrioventricular cushion defect; Others: ASD, VSD, TOF
   - Turner syndrome: Bicuspid aortic valve [MC] (type of valvular aortic stenosis), Coarctation of aorta [2nd MC]
   - Noonan syndrome: Valvular PS
   - William syndrome: Supravalvular aortic stenosis
   - DiGeorge syndrome/CATCH22 syndrome: TOF [MC], TGA (Conotruncal abnormalities)
     - CHD
     - Abnormal facies
     - Thymus hypoplasia
     - Cleft palate
     - Hypocalcemia

Pediatric Neurology

SKULL FONTANELLE IN CHILDREN 00:50

- There is a total of 6 fontanelle in children:
  - 1 Anterior fontanelle
  - 1 Posterior fontanelle
  - 2 Mastoid fontanelles
  - 2 Sphenoidal fontanelles



- Timing of closure of fontanelles:
  - Posterior fontanelle is the first to close: 0-3 months
  - Sphenoid fontanelle: 3-6 months
  - Mastoid fontanelle: 6-18 months
  - Anterior fontanelle: 18-24 months (Last)

NEURAL TUBE DEFECTS 04:35

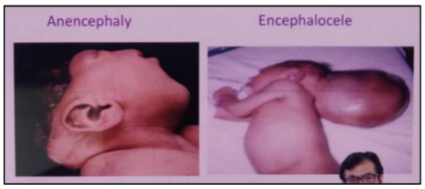
- Failure of neural tube to close by 3-4 week of IU life.

- Risk factor:
  - Diabetes/obesity in mother
  - First-generation antiepileptics during pregnancy
  - Previously affected pregnancy

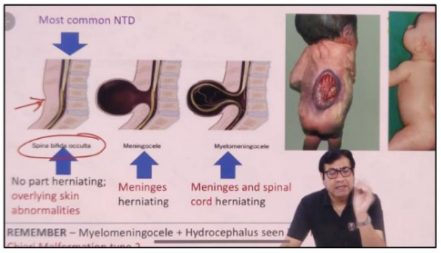
- Antenatal diagnosis (amniotic fluid)
  1. Acetylcholinesterase (most specific and best marker)
  2. Alpha-fetoprotein (most sensitive)

- Folic acid dose to reduce risk of NTD:
  - Started 1 month before conception till 12 weeks of pregnancy.
  - Primary prevention: 0.4mg/day
  - Secondary prevention: 4mg/day

- Types of Neural Tube Defects:
  - **A. Craniospinal:**
    - i. Craniorachischisis
  - **B. Cranial:**
    - a. Closed
      - i. Encephalocele
    - b. Open
      - i. Anencephaly



  - **C. Spinal:**
    - a. Closed
      - i. Spina bifida occulta: overlying skin may have –
        - Tufts of hair
        - Cyst
        - Dimple
    - b. Open
      - i. Spina Bifida Cystica or Aperta:
        - Meningocele
        - Myelomeningocele



- Combination of Myelomeningocele + Hydrocephalus seen in Arnold Chiari Malformation type 2.

HYDROCEPHALUS 20:17

- Obstructive or non-communicating (Obstruction to CSF flow)
- **Most common**: Congenital Aqueductal stenosis

- **Acquired Aqueductal stenosis:**
  - Mumps
  - Toxoplasmosis

- Non-obstructive or communicating (Poor absorption of CSF)
  - **Most common**: subarachnoid hemorrhage

- **Flow of CSF:** Lateral ventricle → 3rd ventricle via foramen of Monro → 4th ventricle via Aqueduct of Sylvius (narrowest part of CSF) → comes out of 4th ventricle and absorbed by subarachnoid villi.
  - Tuberculous meningitis is an important cause of non-obstructive hydrocephalus is children.

- **Treatment**: Shunt procedures –
  1. Ventriculoperitoneal shunt
  2. Ventriculoatrial shunt

- **No shunt procedures:**
  1. Third ventriculostomy

FEBRILE SEIZURE 31:02

- Most common provoked seizures of childhood.
- Seizures associated with high fever in an otherwise neurologically normal child (6 months – 5 years)

Simple febrile seizures	Complex febrile seizures
Generalized	Focal
Single seizure in 24 hours of fever	Multiple seizures
Duration <15 minutes	Duration >15 minutes

- 3-12% of febrile seizures progress to epilepsy.

- Children with high risk of developing epilepsy in future:
  1. Complex febrile seizure

2. Developmental delay
3. Family history of epilepsy

- Treatment:
  - 1st line management for all seizures presenting in emergency: IV Midazolam or IV Lorazepam → 0.1 mg/kg
  - Newborns: IV phenobarbitone

- Prophylaxis strategy for preventing febrile seizures:
  - Routine prophylaxis is not recommended
    1. Paracetamol
    2. Seizures are recurrent: ≥3/6months OR ≥4/1year → Oral diazepam/Oral Clobazam/Oral clonazepam → During first three days of fever.

EPILEPSY IN CHILDREN *44:55*

- **Epilepsy**: 2 seizures occurring 24 hours apart.

Epilepsy	Features	Drug of choice
Absence seizures Petit mal epilepsy	• Sudden interruption of activity • Blank • No post ictal confusion	Ethosuximide Valproate
Infantile spasms (West syndrome) Salaam seizures	• Epileptic spasms • Developmental regressions • Characteristic EEG: Hypsarrhythmia	ACTH (Drug of choice) Vigabatrin (2nd line; DOC: Infantile spasm associated with tuberous sclerosis)
Juvenile myoclonic epilepsy (Janz syndrome)	• 12-18 years of age • Myoclonic jerks occurring in early morning hours. • Gene mutations: CACNB4 and GABRA1	Valproate
Lennox Gastaut syndrome	• Mixed seizures + cognitive impairment + slow waves EEG	Valproate, Lamotrigine

- EEG:
  - Absence seizure: 3Hz spike + wave
  - Infantile spasm: Hypsarrhythmia

PEDIATRIC MENINGITIS *56:40*

- Most common cause:
  - 0-2 months: GBS (Worldwide), E. coli & Klebsiella (India), Staph, Listeria
  - 2 months-2 years: H. influenzae B, S. pneumonia
  - After 2 years: S. pneumonia, Meningococcus

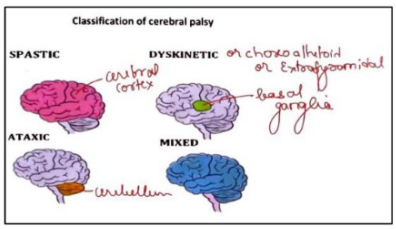
- Complications:
  - Subdural effusion: after H. influenzae meningitis
  - Most common complication of bacterial meningitis in children - Sensorineural deafness → BERA (Brainstem Evoked Response Audiometry)

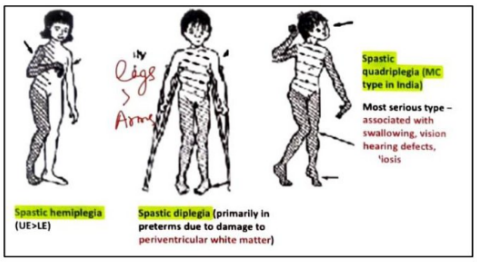
- Treatment:
  - Duration of antibiotics:
    - Meningococcus: 5-7 days
    - Hib: 7-10 days
    - S. pneumonia: 10-14 days
  - Meningococcus: Prophylaxis of household contacts is necessary with Rifampicin.
  - Invasive Hib: prophylaxis to selected contacts with Rifampicin:
    - Children < 4 years of age and unimmunized
    - Immunocompromised

CEREBRAL PALSY *01:05:05*

- Non-progressive, neuromotor disorder resulting from brain injury early in life.

- Classification of cerebral palsy:
  1. Spastic (Most common)
  2. Dyskinetic (Second Most common) [Kernicterus]
  3. Ataxic
  4. Mixed





Periventricular leukomalacia

BRAIN TUMORS IN CHILDREN *01:12:38*

- Divided into 2 broad categories:
  A. Supratentorial
     - Most common in <1 years
     - Most common in > 10years
  B. Infratentorial:
     - Overall, most common
     - Most common in children of 1-10 years of age

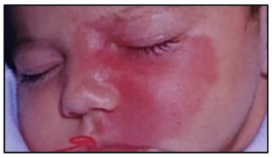
- Overall, most common brain tumor: Cerebellar astrocytoma (periphery of cerebellum > centre)

- Most common malignant brain tumor: Medulloblastoma (Centre of cerebellum > Periphery)
  - Comes under category of embryonal tumors

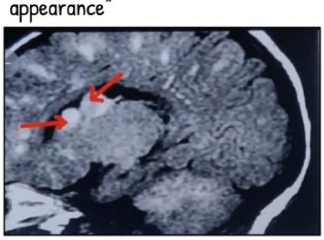
- Most common brain tumor in infants: Choroid plexus tumor
- Most common brain tumor in adolescent (>10 years): Diffuse Astrocytoma (Debatable)
- Most common brain tumor associated with suprasellar calcification: Craniopharyngioma

NEUROCUTANEOUS SYNDROMES *01:22:17*

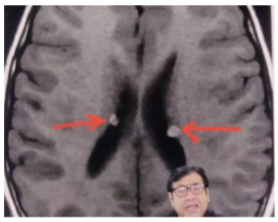
- Sturge Weber Syndrome:
  - CNS: Leptomeningeal angiomas
  - Skin: Port-wine stain + Leptomeningeal angiomas + Glaucoma
  - X-ray skull/CT brain: Tram Track calcification
  - Sporadic development pattern



- Tuberous sclerosis:
  - CNS: Cortical tubers, subependymal nodules → Subependymal giant cell astrocytoma, Subependymal nodules calcified appear as "Candle wax dripping appearance"

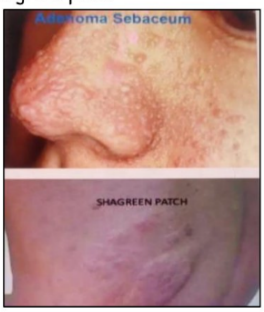


Candle Wax Dripping



Subependymal nodules

  - Skin: "Sebaceous Adenoma" + "Ash Leaf macules" (Most common skin manifestation) + "Shagreen patch"



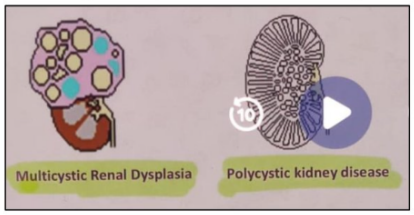
- DOC for Infantile spasm with tuberous sclerosis: Vigabatrin

Pediatric Nephrology & Pediatric Respiratory Disorders

PEDIATRIC NEPHROLOGY 00:12

- Congenital Renal Anomalies:
  - **Disorders of renal development:**
    - Bilateral renal agenesis: Most common associated with Potter Syndrome:
      - Pulmonary hypoplasia
      - Oligohydramnios
      - Twisted skin
      - Twisted face
      - Extremity defects
      - Renal malformation
    - Unilateral renal agenesis: Most common associated with VACTERL syndrome:
      - Vertebral Defects
      - Anorectal malformation
      - Cardiac defects
      - Tracheoesophageal malformation
      - Renal defects
      - Limb defects
  - **Congenital cystic disorders of the kidney:**
    1. Multicystic dysplastic kidney
       - Most common congenital cystic disorder of kidney
       - Abdominal lump in newborns (MC cause after hydronephrosis)
       - Sporadic development
       - Large randomly arranged non-communicating cysts in one kidney
    2. Polycystic kidney disease (Infantile polycystic kidney disease)
       - Autosomal recessive disorders
       - Associated with mutation in PKHD1 gene
       - Congenital liver fibrosis is commonly co-associated.
       - Multiple small cysts lead to bilateral enlarged kidneys.

Congenital cystic disorders of Kidney

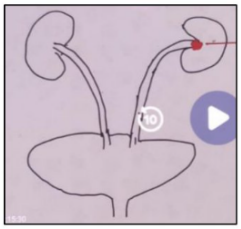


VESICOURETERIC REFLUX (VUR) *11:22*

- Congenital disorder
- Retrograde reflux of urine from bladder to ureter → Kidney (recurrent UTI/pyelonephritis and injuries to the kidney)
- Females >> Males
- Autosomal Dominant Disorder
- IOC: MCU/VCUG [Micturating cystourethrogram or Voiding cystourethrogram]
- MC a result lower urinary tract obstruction
- How to screen for VUR and possible renal injury after first UTI in children:
  - First UTI: Test depends on age of child -
    - < 1 year:
      - USG Urinary tract
      - MCU
      - DMSA (Renal structural damage)
    - 1-5 years:
      - USG urinary tract
      - DMSA:
        - Normal: Do nothing
        - Abnormal: MCU
    - > 5 years:
      - USG urinary tract:
        - Normal: Nothing to be done
        - Abnormal: MCU & DMSA

CONGENITAL OBSTRUCTION OF URINARY TRACT *21:50*

- Ureteropelvic junction obstruction:
  - Unilateral > Bilateral
  - USG: Hydronephrosis with normal ureter
  - Grade III/IV hydronephrosis with poor renal function: Pyeloplasty is recommended



- Ureterocoele:
  - Dilatation of intravesical part of ureter with pinpoint orifice.
  - This can lead to Vesicoureteral Reflux.
  - Females > Males
  - Often associated with duplicated ureters (almost in all females)
  - MCU: Filling defect in bladder
  - IVP: Adder shaped/Cobra head shaped ureter.

- Posterior Urethral Valve (PUV):
  - Most common cause of lower urinary tract obstruction in male infants.
  - It is an obstruction present in prostatic part of urethra.
  - Urine starts getting accumulated inside the bladder leading to Vesicoureteric reflux eventually.
  - USG: Keyhole sign

- How are VUR managed?
  - VUR → MCU is investigation of choice. → Grade I- Grade V VUR.
  - As grade increases, severity of VUR increases.
  - Management:
    - Antibiotic prophylaxis/Wait and watch
  - Take history of bladder bowel dysfunction (BBD) → High risk of UTIs.
  - Duration of prophylaxis:
    - Low grade VUR → 1 year
    - High grade VUR → 5 years
    - Associated Bowel Bladder Dysfunction → > 5 years
  - Antibiotic prophylaxis is recommended in most infants with VUR.
  - Indications for surgery:
    i. Treatment adherence is doubtful
    ii. Renal functions are deteriorating
    iii. High grade reflux on antibiotic prophylaxis with breakthrough UTI

GLOMERULONEPHRITIS ("NEPHRITIC SYNDROME") 43:55

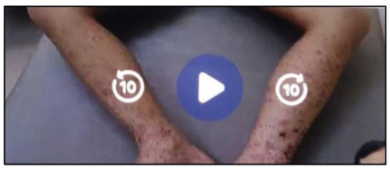
- Glomerular Injury:
  1. Hematuria (with or without proteinuria)
  2. Hypertension
  3. Edema → Main cause of edema → Na reabsorption

- Types of glomerulonephritis:
  a. **Isolated**
     1. Alport syndrome: Glomerulonephritis, bilateral sensorineural deafness and anterior lenticonus.
     2. Post streptococcal GN
     3. IgA nephropathy
     4. MPGN
     5. Rapidly progressive GN

  b. **Part of systemic disorder:**
     1. Lupus nephritis
     2. Henoch Schönlein Purpura associated nephritis
     3. Good Pasture Syndrome (Adults > Children)

- Post streptococcal GN:
  - MC cause of acute GN in children
  - Hematuria 1-3 weeks after URTI, C3 reduced
  - GN associated with Low C3 level:
    i. PSGN
    ii. MPGN
    iii. Lupus Nephritis
  - Treatment symptomatic

- IgA Nephropathy and Henoch Schönlein Purpura (HSP) Nephritis
  - IgA Nephropathy usually associated with chronic GN
  - Hematuria 1-3 days after URTI, C3 Normal
  - Renal biopsy mimics HSP
  - Treatment: ACE inhibitors (1st line) → Corticosteroids (2nd line)
  - HSP also called IgA vasculitis (small vessel vasculitis in children)
  - Most common manifestations: Abdominal pain + arthralgia + palpable purpura in dependent position
  - 30-50% have nephritis (can develop till 3 months after onset of illness)



Rashes of HSP

- Rapidly progressive GN (RPGN): MC cause is pauci-immune ANCA positive vasculitis.
  - It is also called as Crescentic Glomerulonephritis.
  - Pauci-immune ANCA positive small vessel vasculitis include:
    i. Microscopic Polyangiitis
    ii. Granulomatosis with polyangiitis
    iii. Eosinophilic granulomatosis with polyangiitis

NEPHROTIC SYNDROME 01:04:23

- Pathological proteinuria: Urine protein excretion > 3.5 gram/day or urine protein excretion > 40mg/m^2/hr or Urine Protein: Creatinine ratio >2.
- Only low molecular weight proteins are being lost. → Selective Proteinuria

- There is:
  - Decreased albumin
  - Increased cholesterol
  - Increased risk of thrombosis (most common cause of renal vein thrombosis in children)
  - Increased risk of infection

- Causes:
  - Minimal change disease/Lipoid Nephrosis: MC cause in children
  - Focal segmental glomerulosclerosis: MC cause in adolescent and adults
  - Other causes

- Treatment of nephrotic syndrome in children:
  - **Corticosteroids (Prednisolone):**
    - First 6 weeks: 2mg/kg/day (daily)
    - Next 6 weeks: 1.5mg/kg/day (alternate days)

- Steroid dependent nephrotic syndrome:
  - Significant Proteinuria (2 relapses minimum) on alternate day steroids or within 2 weeks of stopping the steroids.

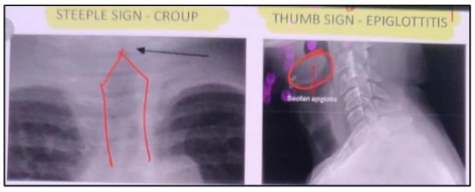
- DOC: Levamisole (best)
  Others: MMF > Cyclophosphamide

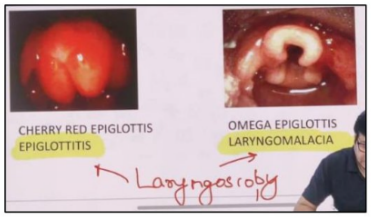
- Steroid resistant NS:
  - Proteinuria persisting despite 6 weeks of daily steroids treatment.
  - DOC: Calcineurin inhibitors - Cyclosporine > Tacrolimus

PEDIATRIC RESPIRATORY DISORDERS *01:17:08*

- Important points for some common childhood respiratory infections.
  - Most common cause of common cold: Rhinovirus
  - Most common cause of stridor in infants is laryngomalacia (supraglottic larynx poor cartilage framework)
  - In laryngomalacia, symptoms (stridor) improve with position change
  - In choanal atresia, symptoms (cyanosis, RD) improve with crying
  - MC cause of croup (Fever + URTI → Seal barking cough + stridor) - Parainfluenza virus.
  - DOC in croup for mild stridor → Corticosteroid (Dexamethasone: Oral/IM)
  - DOC in croup for severe stridor → Epinephrine by nebulization
  - MC cause of epiglottitis (sudden-onset fever/RD/Stridor/restlessness → Emergency) is S. pneumonia, H. influenza
  - Treatment of choice for epiglottitis → Secure the airways (intubation) followed by antibiotics
  - MC cause of wheezing (first wheezing) in infants is bronchiolitis (1-6 months of age)
  - MC cause of bronchiolitis is RSV

  - Treatment of bronchiolitis is symptomatic; Palivizumab for prophylaxis in high-risk infants:
    - <29 weeks gestation
    - Co-existing hemodynamically significant CHD
    - Co-existing bronchopulmonary dysplasia





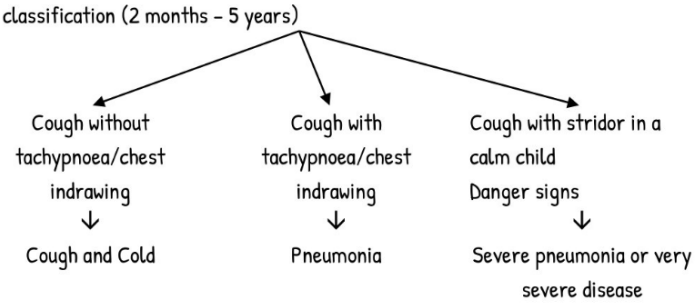
- COVID-19 infection in children:
  - **Mild disease:**
    - Mild symptoms
    - No fast breathing:
      - RR > 60/minute in 0-2 months
      - RR > 50/minute in 2-12 months
      - RR > 40/minute in 1-5 years
      - RR > 30/minute in > 5 years

      } Fast breathing definition
  - **Moderate disease**:
    - Fast breathing OR
    - Oxygen saturation (90-93% on room air)
    - No sign of severe disease
  - **Severe disease:**
    - Pneumonia with any one of these:
      - Grunting/severe retractions
      - Oxygen saturation <90%
      - Increased respiratory effort
    - Lethargy, somnolence, seizures
    - Severe diarrhea, vomiting, abdominal pain

- Management:
  - Mild Covid → Home, isolation, symptom management, RR and O_2 saturation monitoring
  - **Moderate Covid → Managed in hospital:**
    - O_2 saturation is more than or equal to 94% → Treat as Mild illness
    - O_2 saturation is <94% → Treat with O_2 and IV steroids if deteriorating
  - **Severe Covid:**
    - Hospitalize, start on O_2, ventilation
    - IV steroids is a must, treat ARDS/Shock
    - Other treatment as needed

PNEUMONIAS IN CHILDREN *01:39:40*

- Pneumonia – IMNCI [Integrated Management of Neonatal and Childhood illnesses] classification (2 months – 5 years)



- Danger signs:
  - Unconscious
  - Persisting vomiting
  - Unable to drink
  - Seizures
- Cough and Cold:
  - No routine antibiotics
  - Follow up after 5 days
- Pneumonia:
  - Amoxycillin for 5 days
  - Follow up after 2 days
  - Symptomatic management
  - O_2 saturation if less than 90% → Refer to higher centre
- Severe pneumonia or very sever disease:
  - IV ampicillin + IV gentamycin
  - Refer to higher centre

CYSTIC FIBROSIS IN CHILDREN – IMPORTANT POINTS *01:46:54*

- AR disorder associated with mutation in CFTR [Cystic Fibrosis Transmembrane Regulatory] protein (controls Cl and Na transport across hollow ducts)

- Manifestation:
  - Recurrent chest infections + respiratory obstruction thick secretions
  - Exocrine pancreatic insufficiency (Fibrocystic disease of pancreas) - malabsorption, foul stools
  - Obstruction to bile flow (Cholestasis) - obstructive jaundice
  - In newborns - meconium ileus → Intestinal obstruction

- Sweat duct only hollow duct not obstructed

- Diagnosis:
  - Sweat chloride test (atleast two positive results) → Chloride in sweat ≥60Meq/L - positive; 30-59Meq/L: Indeterminate test ; <30Meq/L: Negative test
  - In newborns (screening test): Immunoreactive trypsinogen testing + One CF-specific gene mutation.
  - Detection of atleast two CF-specific mutations (karyotyping)

- Treatment: Supportive

ASTHMA *01:57:04*

- Diagnostic features of asthma in children:
  - Typical symptoms: Wheezing, Respiratory distress (early morning, nocturnal), dry cough
  - FEV1/FVC <80%
  - Improvement in FEV1 >12% after bronchodilator inhalation
  - Diurnal (AM/PM) PEF [Peak Expiratory Flow rate] variability ≥20%

- Asthma management approach in children:
  - **< 5 years:**
    - Step-1: SABA inhalation when needed (rescue therapy)
    - Controller therapy: Step-2 Low-dose Inhalational corticosteroids daily (recommended) or LTRA (montelukast)
    - Persistence of symptoms:
      - Low dose ICS + LTRA
      - Double the dose of low dose ICS
  - **6-11 years:**
    - Step-1: SABA inhaled + Low dose ICS as needed
    - Controller daily: Step-2 - Low dose ICS daily (recommended) or LTRA (less commonly recommended)

- Persistence of symptoms:
  i. Low dose ICS + LABA (daily controller) } SABA as rescue when needed
  ii. Medium dose of ICS
  iii. Budesonide-LABA (Formoterol) as maintenance therapy (controller) and reliever therapy (rescue) → ICS-Formoterol combination

Latest Updates on Childhood Asthma

ASTHMA GUIDELINES IN CHILDREN 2023 *01:05*

- Chronic inflammatory disease of airways associated with hyperresponsive airway.
- Occur in susceptible children. Develop dry cough, recurrent wheezing and breathing difficulty especially at night.

DIAGNOSIS OF ASTHMA *03:14*

- **Spirometry**: best marker of airway obstruction is FEV1 /FVC ratio.
- If ratio is less than the lower limit of normal in children (normal is >0.9) is indicative of asthma.
- If FEV1 improves more than 12% after inhaling bronchodilator then it's reversible obstruction.
- Average diurnal peak expiratory flow rate variability is >13%
  ie PEF is taken two times a day, difference between max and min divided by average reading of the two.
  Indicative of hyperresponsiveness of airways.
- During or after exercise FEV1 falls by more than 12% or PEF falls by more than 15% then indicative of asthma induced by exercise.

MANAGEMENT OF ASTHMA *10:45*

- Rescue treatment for acute presentation and controller treatment daily control and reduce frequency.

- In < 5 yrs children:
  1. Step 1 treatment is inhaled bronchodilators ie SABA. sos when required (for infrequent episodes)
  2. Step 2 treatment: daily controller is inhaled low dose corticosteroids. Also same for older children. It can be inhaled budesonide, beclomethasone, fluticasone etc.
  3. Step 3 : If not improved, double the dose of inhaled corticosteroid or add a leukotriene receptor antagonist on alternate days (Monteluekast recommended for children).

- In 6-11 yrs of age
  1. **Step 1 :** if episodes < 2 times per month
     Inhaled SABA with low dose inhaled corticosteroid sos.

2. **Step 2:** if episodes>2 times per month, daily controller is same i.e. low dose ICS.

3. **Step 3**: if symptoms are like daily and nocturnal symptoms more than equal to 1 per week.
   I. Low dose ICS + LABA like Salmeterol
   II. Increase the dose of inhaled dose ICS to medium.
   III. Inhaled ICS + formoterol very low dose as maintenance and reliever treatment.
   MART. No separate drug like SABA for relieving treatment.

4. **Step 4**: daily symptoms and nocturnal symptoms more than or equal to 1 per week along with decreased lung function.
   I. LABA with medium dose ICS
   II. ICS formoterol low dose as MART

5. **Step 5**: uncontrolled asthma. New therapies like anti Ig E like Omalizumab, anti IL4R antagonist like dupilumab, anti IL5 antagonist like mepolizumab and sometimes a very short course of oral corticosteroids.

Pediatric GI Disorders

HYPERTROPHIC PYLORIC STENOSIS 00:52

- Gastric outlet or pylorus gets thickened and get obstructed.
- Symptoms, 3Ps
  1. Palpable abdominal mass ,olive shaped and in epigastric region or on the rt side just below the liver.
  2. Projectile vomiting starting from 3wks of age.
  3. Peristaltic waves from left to right or towards the umbilicus.
- Treatment surgical.
- Diagnosis confirmed by USG and Hypochloremic hypokalemic metabolic alkalosis due to persistent vomiting.

HIRSCHSPRUNG DISEASE 00:45

- MC cause of not passing of meconium (first stool, should pass within 24hrs)
- Investigations should be made if not passed within 48hrs.

- Other causes of delayed meconium passage
  1. Large intestinal disorder; MC Hirschsprung disease.
  2. Small intestinal disease
  3. Hypothyroidism
  4. Maternal drugs
  5. Dehydration

- Also called congenital aganglionosis.
- Ganglion absent in rectosigmoid colon. As a result the intestine can't contract normally. So the aganglionic part is contracted and the proximal part of the normal intestine is dilated.
- It's seen in contrast enema.
- Diagnosis made by rectal suction biopsy. Absence of ganglion, increased acetylcholinesterase activity and hypertrophy of nerve trunks are seen in histopathology.
- Treatment is surgical.

MANAGEMENT OF DEHYDRATION IN CHILDREN 08:07

- Types of dehydration; No, some and severe.

- Severe one is lethargic, unconscious in contrast to some, where restlessness and irritability is seen.

SOME DEHYDRATION	SEVERE DEHYDRATION
• Restless, irritable	• Lethargy, unconscious
• Drinks eagerly	• Not able to drink or drinks poorly
• Sunken dry eyes	• Sunken dry eyes
• Skin pinch goes back slowly	• Skin pinch goes back very slowly

- Skin pinch goes back in more than 2 s for severe dehydration.
- Management of no dehydration:
  According to ORS therapy.
- Management of some dehydration also according to ORS therapy.

- In some dehydration
  1. Daily requirements of fluid should be met.
  2. ORS 75ml/kh over 4 hrs
  3. After each loose stool 10 ml/kg should be given.

- Severe dehydration needs IV fluid. Ringer Lactate is the drug of choice, normal saline if not available.
  1. If child is less than 1 yr:
     30ml/kg over 1 hr
     70 ml/kg over 5 hr
  2. If more than 1 yr:
     30ml/kg over ½ hr
     70 ml/ kg over 2.5 hr

NEONATAL CHOLESTASIS *16:12*

- Obstruction to bile flow ultimately causing conjugated jaundice after two weeks.
- Obstruction can be inside the liver (intrahepatic) or outside (extrahepatic).
- Intrahepatic causes: cystic fibrosis, idiopathic neonatal hepatitis, alpha 1 AT deficiency, metabolic liver disease.
- Extrahepatic causes: Biliary atresia (most common cause overall)
- Investigations: USG, LFT are the initial tests.
- GGT is increased, the best marker of obstruction in the liver, obstruction to bile flow. Most reliable enzyme.

- Most specific investigation is liver biopsy. Can differentiate between intrahepatic and extrahepatic causes.
- Treatment is management of the cause.
- In biliary atresia, there is bike obstruction si fats aren't digested. So high calorie diet should be supplied. Short doses of oral phenobarbitone(enhance bike flow) can also be given for 3-7 days because bile salt accumulation can cause itching.

CELIAC DISEASE

22:12

- Gluten sensitive enteropathy.
- Wheat, barley, rye which contain gluten cause abdominal pain, bloating etc.
- Maize can be taken.
- Hereditary and increased susceptibility in HLADQ2 and HLADQ8 positivity. Reliable biomarkers.
- Other biomarkers are tissue transglutaminase antibody (best marker and useful for early detection) and endomysial antibody.
- Small intestinal endoscopy and biopsy from duodenum can also be done.
- There will be damage to mucosal due to gluten sensitivity.
- Increased intraepithelial lymphocytes, villous flattening and crypt hyperplasia are the three hallmark features in biopsy.
- If TTG is raised more than 10times the normal we can confirm and no need to go for a biopsy.
- If TTG levels are high but one among HLADQ8 and HLADQ2 aren't positive then it's an indication of biopsy. Biopsy is taken to confirm when TTG levels are not that high.
- Treatment is by lifelong abstinence from gluten containing food items.

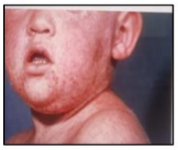
Other Pediatric Disorders and Childhood Infections

TUBERCULOSIS 00:41

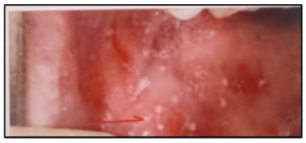
- One of the most common cases and has high morbidity rates even though less mortality rates .
- Out of all the TB cases only 10% occurs in children.
- Childhood infections are often subclinical or sputum negative. So it's hard to identify TB in children.
- Most children acquire infection from adults more than other children because children are often sputum negative.
- Extra pulmonary TB includes TB meningitis, TB lymphadenopathy, intestine TB, bone TB etc. Most common one in children is TB lymphadenitis.
- Congenital TB is by transmission through amniotic fluid, primary complex formed in lungs due to aspiration and placenta via umbilical vein from mother to fetus and primary complex is formed in the liver here. Most common is liver.
- PCR based CBNAAT (cartridge based nucleic acid amplification test), GeneXpert and TrueNAT are the best diagnostic tests. High turnaround time and can give results in 3-4 hrs and are highly reliable.
- Gastric aspirate or the sputum if possible to expectorate is the sample taken.
- Treatment is 2HRZE + 4HRE for all classes.
- Pyridoxine is given when INH is given in the form of treatment or prophylaxis.

MEASLES 06:38

- One of the most common viral infections caused by paramyxoviridae virus.
- Fever and 3Cs follow the incubation period (10-21d).
- 3Cs are Cough, chorizo and conjunctivitis.
- After the incubation period the child enters prodromal phase. Here Cough, chorizo, conjunctivitis and fever is present.
- After the 4th day the fever rash appears.
- There is the appearance of koplik spots opposite to lower molar 2 days after fever in the prodromal phase.



Measles Rash



Spots with red background

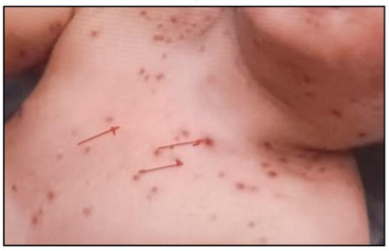
- Complications:
  1. Otitis media (most common)
  2. Diarrhea
  3. Pneumonia
  4. Giant cell pneumonia or hecht's pneumonia
     In immunocompromised.
  5. Meningitis
  6. Subacute sclerosing panencephalitis
     Least common and take 7-13 yrs to occur after primary infection due to alteration in virus or immune system.
     Show myoclonic jerks (lead pipe rigidity)and burst separation episodes in EEG.

CHICKENPOX

10:25

- Herpes virus causes chickenpox.
- Incubation period followed by prodromal phase. Unlike measles there are very nonspecific prodromal symptoms including low grade fever, body ache which is followed by rashes 1 or 2 days after fever.
- Rashes are pleomorphic i.e. in different stages of development. Initially it may be in macular or papular stage, later turning to vesicles with fluid. Fluid is infectious when come in contact.

Chicken pox rash



- When scabs are formed the child is non infectious.
- So there are two modes of transmission ie oro respiratory route and by coming in contact with fluid.
- Rashes are centripetal ie more in center and less in periphery.
- MC complication is secondary bacterial infections.
- Others are Reye syndrome, meningitis and pneumonia (less common in children).

- **Congenital varicella syndrome:** due to infection of the mother in the first trimester and thus the fetus gets infection leading to malformations of the fetus like zigzag scars on skin, limb defects, cerebral atrophy etc. The child is not infectious and doesn't have a viral load because of transfer of antibodies.
- **Neonatal varicella**: When mother is infected from 5 days before to 2 days after delivery there is a chance of passing viral load to child, but protective antibodies could not be transferred. Should give varicella zoster immunoglobulin at birth for protection.

ERYTHEMA INFECTIOSUM (FIFTH DISEASE) *20:02*

- It was the fifth exanthematous illness to be discovered in the sequence.
- Parvovirus B19.
- Rashes on the cheek like slapped cheek appearance.

ROSEOLA INFANTUM *21:12*

- Sixth disease or exanthem subitum.
- Caused by HHV6, sometimes HHV 7
- Fever disappears on the third day followed by a rash appearance.
- Nagayama spots seen on uvula and palate. White to red colored spots.

HIV IN CHILDREN *23:11*

- NACO- National AIDS control organization
- Perinatal or vertical transmission is more common, parent to child transmission (PTCT).
- Nevirapine prophylaxis was indicated to start in children at risk.
- Revised guidelines to prevent PTCT indicate a viral load checking in mothers having HIV. Testing should be done between 32-36 wks.
- If viral load is less than low risk child and if high load them high risk child. If the mother is untested and the mother is not under ART, the infant becomes at high risk.
- Nevirapine given for low risk and Nevirapine along with zidovudine for High risk prophylaxis.
- Cotrimoxazole given from 6wks for P.jiroveci prophylaxis.
- For TB prophylaxis INH is given for 6 months by:
  1. Testing after 1 year if no TB exposure
  2. Testing before 1 yr if TB exposure is there.

- Choice of testing:
  1. Total nucleic acid PCR for children below 18m
  2. HIV antibody by ELISA for testing after 18 m. Not done before 18m because passive transfer of antibody can occur from mother to child.

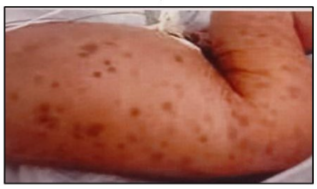
Treatment of HIV in children < 6 years, <20 kg
Abacavir + Lami + Lopinavir/Ritonavir
6-10 years, 20-30 kg
Abacavir + Lami + Dolutegravir
>10 years and > 30 kg
Tenofovir + Lami Dolutegravir

- Dolutegravir is an integrase inhibitor.
- Lopinavir and Ritonavir are protease inhibitors.

CONGENITAL RUBELLA SYNDROME *32:00*

- When a mother has rubella during pregnancy transferred to the child. If infection occurs after 16 wks of gestation the fetal defects are minimal.
- Most prone in the first 11 wks, 11-16wks less chance, more than 16 wks negligible.
- Associated with blueberry muffin lesions in the skin.
  Also occurs in congenital CMV. Indicative of dermal erythropoiesis.
- Cataract, CHD and sensorineural deafness occurs(single most common complication).
- PDA is the most common followed by pulmonary stenosis and ASD is the least common CHD occuring in congenital rubella syndrome.

Blue berry muffins



CONGENITAL CMV 34:35

- Most common congenital infection.
- 80-90% are asymptomatic.
- Can be associated with chorioretinitis, intracranial calcification and microcephaly.
- Sensorineural deafness is most common complication and congenital CMV is the most common non syndromic cause for sensorineural deafness in children.
- Should treat with Ganciclovir to avoid this in all babies.

CONGENITAL TOXOPLASMOSIS 36:39

- Triad of hydrocephalus, intracranial calcification and chorioretinitis.
- IUGR and microcephaly can also occur.
- Pyrimethamine, Sulfadiazine and leucovorin are used for treatment.

NATIONAL IMMUNIZATION SCHEDULE 38:14

National Immunization Schedule	
Birth	BCG, OPV-0, Hep B birth dose
6 weeks	OPV-1, Pentavalent-1, Rotavirus 1, fIPV1, PCV1*
10 weeks	OPV-2, Pentavalent-2, Rotavirus 2,
14 weeks	OPV-3, Pentavalent-3, Rotavirus 3, fIPV2, PCV2*
9-12 months	MR-1, JE 1**, PCV Booster*
16-24 months	MR2, JE 2**, DPT Booster 1, OPV Booster
5-6 years	DPT Booster 2
10 years	Tetanus and adult diphtheria (Td)
16 years	Td
Pregnant women	Td1, Td2 or Td Booster***
* PCV only in selected states, ** JE in endemic districts only; *** 1 dose if previously vaccinated within 3 years	

- Fractional dose of IPV is 1/5th dose of IPV. It's given 2 doses intradermally.
- d is a lesser dose of D, which is a higher dose which is equivalent to 25-30 times low dose and can create immunogenic reactions.

PEDIATRIC ENDOCRINOLOGY 42:53

- Precocious puberty is the appearance of pubertal signs before 8 years in girls and 9 or 9.5 years in boys.

- Delayed puberty has no sexual characters by 13y, no menstrual cycle by 16y and no pubertal sign by 14y in boys.

PRECOCIOUS PUBERTY *44:07*

- Divided into
  1. Central: earlier maturation of hypothalamic pituitary axis, so gonadotropin dependent.
     Seen common in females and usually idiopathic. In males most common cause is hypothalamic hamartoma associated with gelastic seizures causing uncontrolled laughter.
  2. Peripheral: due to increased production of gonadal steroids from gonads or adrenals.

DELAYED PUBERTY *46:26*

Divided to :

- **Primary**; hypergonadotropic hypogonadism. Gonads are not developed properly which cause compensatory increase in gonadotropins. Eg: klinefelter's syndrome, turners syndrome, swyer syndrome.
- **Secondary**; hypogonadotropic hypogonadism, problem in CNS. Not enough gonadotropins are produced.
  Eg: Prader willi syndrome(disease of gene imprinting, deletion of paternal gene on chr. No. 15, have hypotonia and unable to take proper feeds but later develop hyperphagia, GH deficiency, obesity), kallmann syndrome(anosmia).
- MC cause of congenital hypothyroidism is thyroid dysgenesis.
- MC cause of goitrous hypothyroidism is dyshormonogenesis.

METABOLIC SYNDROMES *50:35*

- Mousy or musty odor of urine related to phenylketonuria.
  Deficiency of phenylalanine hydroxylase.
- Cabbage odor urine in tyrosinemia type I. Deficiency of fumarylacetoacetate hydrolase. Cause damage to kidney and liver. Hepatorenal type of tyrosinemia.
- Sweaty feet odor urine in isovaleric aciduria.
- Cat urine odor in multiple carboxylase deficiency.
  Due to deficiency of biotinidase and holo carboxylase synthetase deficiency which cause deficiency of biotin specific carboxylase.

- **Ferric chloride test green** : indicates PKU
  blue color in maple syrup urine disease.
- **Homocysteinuria**: tall stature, long limbs, ectopia lentis, strokes. Dd of marfan syndrome. Deficiency of cystathionine beta synthase enzyme.
- **Alkaptonuria**: deficiency of homogentisic acid oxidase. Cause darkening of urine.
- Hyperammonemia seen in organic acidurias(high anion gap acidosis) and urea cycle defects.
- Organic aciduria, rashes , alopecia seen in multiple carboxylase deficiency. Biotin is used for treatment.
- X linked urea cycle defect is due to OTC deficiency.
- Hartnup disease, defective reabsorption of neutral amino acids in renal tubules. Have pellagra like symptoms ie photosensitive rashes but mental retardation is not usually a feature.

- Glycogen storage disorders:
  Can be either hepatic or muscle or hepatic and muscle disorders.
  Pure muscle disorders are type 5 McArdle disease and type 7 Tauri disease.

- Lysosomal storage disorders are usually autosomal recessive, two X linked are hunters and fabry disease.
- Acroparesthesia and hyperhidrosis (less sweating) are seen in Fabry disease.
- Marked startle response to noise seen in Tay sachs disease.
- Cherry red spots seen in Tay sachs and sandhoff disease. Also seen in 50% of children with Neiman pick disease.

- Treatment:
  1. Tetrahydrobiopterin- PKU
  2. Pyridoxine - Homocysteinuria (cofactor of enzyme)
  3. Betaine- homocysteinuria
  4. Biotin- multiple carboxylase deficiency
  5. Nitisinone-tyrosenimia
  6. Nicotinamide- hartnups disease

RHEUMATIC DISEASE *58:20*

- Suspect in children with fever, joint pain, rashes and fatigue.
- Called collagen vascular disease or connective tissue diseases. Has a multisystemic involvement.

- Malar rash seen in SLE and juvenile dermatomyositis(spare nasolabial folds).
- ANA are highly sensitive for SLE and anti dsDNA and anti Sm are highly specific.
- Anti Ro, SSA
  (Sjogren Syndrome related antigen A)and anti La, SSB involved in neonatal Lupus.
- Anti histone ab associated with drug induced Lupus.
- Most feared complication of neonatal Lupus is heart block.
- Juvenile dermatomyositis is a collagen vascular disorder associated with proximal muscle weakness and malar rashes. Heliotrope rash are purplish and seen around eyelids along with shiny papules in metacarpophalangeal and interphalangeal joints (Gottron's papules) are pathognomonic of juvenile dermatomyositis.
  Also associated with pathological calcification and lipodystrophy.
- Kawasaki disease: medium vessel vasculitis.
  The most common cardiac complication of this is myocarditis(first two weeks), most feared is coronary artery aneurysms(in subacute phase).
  Aspirin first given in anti-inflammatory doses and then in anti thrombotic doses.
- HSP is a small vessel of vasculitis. MC vasculitis seen in childhood.

CHILDHOOD MALIGNANCY *01:04:32*

- MC abdomen malignancy in infants - neuroblastoma (tumor of peripheral sympathetic system)
  (Followed by wilms tumor of kidney, both occur in young children)
- MC site of this is adrenal medulla.
- Neuroblastoma commonly metastasis to bone (also to liver and lymph nodes) while wilms tumor to lungs.
- Neuroblastoma present with abdominal mass, diarrhea, HT, bone pain. There will be increase urine excretion of VMA and HVA.
- Can be seen with opsoclonus myoclonus syndrome. Jerky eyes and myoclonus in limbs. (Paraneoplastic manifestation)
- Wilms tumor: asymptomatic abdominal mass.
- Neuroblastoma diagnosed by increased catecholamine metabolites in urine (VMA and HVA)
  Neuron specific enolase, CT or MRI of abdomen.
- CT abdomen for wilms tumor.
- Most common malignancy is leukemia.
- ALL> AML

- In Children ALL > AML
- Favorable prognostic factors in ALL → Age 1-10 years; t(12:21); hyperdiploidy
- Poor prognostic factors in ALL → Age <1 or > 10 years; t(4:11), t(9:22); hypodiploidy