Edema:

• Is it truly edema or not?
• It may be due to steroid, allergy, or other causes.
• Is this edema localized or generalized
• If it’s generalized the most common cause $\rightarrow$ nephritic syndrome especially if there is peri-orbital edema.
• Then $\rightarrow$ protein losing enteropathy $\rightarrow$ cardiac $\rightarrow$ liver

Pathophysiology:

• $\uparrow$ hydrostatic pressure
  o Acute nephritic syndrome.
  o Congestive cardiac failure (CHF)

• $\downarrow$ plasma oncotic pressure
  o Protein energy malnutrition (PEN), Nephrotic syndrome and protein loosing enteropathy.

• $\uparrow$ capillary leakage
  o Allergy, sepsis, angioedema.

• Impaired venous flow
  o Venacaval obstruction, hepatic vein obstruction.

• Impaired lymphatic flow
  o Congenital lymphedema, Wuchereria bancrofti infection (elphantaiasis)
Clinical approach to edema:

renal:
Periorbital edema, history of collagen vascular disease (SLE, RA, rash, joint pain frothy urine due to protein)

Cardiac: Ask about:
- Palpitation: if the child is > 3 year
- Fainting, bluish episode (TOF).

Hepatic:
Jaundice, umbilical infection (omphalitis) → neonatal sepsis.

Sign of adequacy of breast milk intake:
1. Urine output: a well-hydrated infant voids six to eight times a day. Each voiding should soak, not merely moisten, a diaper, and urine should be colorless.
2. Stool: By 5 to 7 days, loose yellow stools should be passed at least four times a day.
3. Growth: Rate of weight gain provides the most objective indicator of adequate milk intake in mother: let down reflex.

The way of sterilization:
- First wash the bottle with cold water + detergent (to remove protein - albumin)
  → Brush it
- Wash it by hot water (to remove lipids - carbohydrate)
- Take off the tit and put the bottle in boiling water for 10-15min.
- Put the tit for 3-5 min in the boiling water.
- Then put the bottle in the refrigerator till you will use it.
- Types of sterilization: by Boiling or Steaming Sterilizer or using chemicals (specialized for sterilizing baby feeding equipments)
- Number of bottles = number of feeds + 1.
Assessment of degree of dehydration:
(3-6 %) of body weight mild
(6-10 %) moderate
(> 10 %) severe
(>14 %) incompatible with life

Indication for hospital admission in diarrhea:
1. Moderate – severe dehydration.
2. Persistent vomiting.
3. Social background.
4. Diagnose in doubt (e.g meningitis, parenteral diarrhea)
5. Food poisoning.

Treatment:
ORS: oral rehydration should be given to infants and children slowly, especially if they have emesis. It can be given initially by a dropper, teaspoon, or syringe, beginning with as little as 5mL at a time. The volume is increased as the pt. tolerates the ORS.

Assessment of Developmental age:
If there is discrepancy between chronological age and developmental age→ think about:
1. UMNL due to any cause: trauma for example.
2. Cerebral palsy.
3. Degenerative brain disorder.
4. Kernicterus

Anti-motility drugs: should be avoided in children as it may cause respiratory depression:
- <1 year (Absolutely contraindicated)
- >1 year (relatively contraindicated)

When to give antibiotics in pt. diarrhea:
1. Less than 3 month.
2. Marsmus to avoid septicemia.
4. Food poisoning.

Note: Normal increment in weight below 3 months 600-900 g or
Session notes  

Important steps should not be forgotten in exam:
1. Greeting the parent.
2. Introduce yourself.
3. Take permission.
4. Thank them finally.

**GIT examination:**
General exam (related to GIT) + abdominal exam:

**Abdominal examination:**
1. **Inspection:** Scar of previous surgery, hernia, dilated veins, abdominal distension, shape of umbilicus
2. **Palpation:** first of all ask about any pain and try to avoid painful area
   A. **Superficial:** for tenderness, rigidity, mass.
   B. **Deep:** for any mass, tenderness, and organomegally.

| Liver: start from RIF upward till you feel the live → if palpable: comment on: |
|---------------------------------|-------------------------------------------------------------|
| Surface: smooth or nodular.     | Consistency: Soft, firm or hard.                           |
| Border: sharp or blunted.       | Tender or not (tender in RV heart failure, hepatitis, liver abscess) |
| Then → measure how many cm below costal margin. | -3cm BCM is significant. -Liver is always palpable in neonate |
| Then → measure liver span (differ according to age of child). | The liver may be palpable but not enlarged as in hyperinflated lungs |

| Spleen: palpate for spleen from RIF and ascend diagonally till you reach the left costal margin → if you couldn’t feel the spleen → turn the baby to the Rt. Then palpate. → If you felt the spleen → comment on (as in case of liver). |
|-------------------------------------------------|-------------------------------------------------|
| **Q/ How to differentiate between palpable spleen and the left kidney?** |

<table>
<thead>
<tr>
<th>Features</th>
<th>Organ</th>
<th>Spleen</th>
<th>Lt. kidney</th>
</tr>
</thead>
<tbody>
<tr>
<td>Movement with respiration</td>
<td>yes</td>
<td>No</td>
<td></td>
</tr>
<tr>
<td>Direction of enlargement</td>
<td>Diagonally toward RIF</td>
<td>Vertically toward LIF</td>
<td></td>
</tr>
<tr>
<td>Can get above it?</td>
<td>No</td>
<td>yes</td>
<td></td>
</tr>
<tr>
<td>Presence of notches?</td>
<td>yes</td>
<td>No</td>
<td></td>
</tr>
</tbody>
</table>
**Febrile convolution:** Age 6-60 months (6m-5 years)

**Types:**

a. **Simple** (generalized, < 15 min, occurs 1 time in 24 hours and does not recur)

b. **complex** (focal, > 15 min, > 1 time in 24 hours, recurrent)

c. **status epilepticus:** seizure that is lasting >20-30 min or recurrent convulsive attacks without retaining consciousness in between.

**Risk factor of recurrence (in febrile convolution):**

1. Age < 1 year.
2. Duration between fever and seizure < 24 hours.
3. Temperature > 39 C.
4. Family Hx of epilepsy.
5. Male gender.
6. Hypernatremia (young age).

**Causes of status epilepticus:**

1. Missed dose of anti-epileptic drug.
2. Febrile convolution “most common”.
3. Meningitis.
4. Hypoglycemia
5. Electrolyte disturbance: (↓Na⁺, ↓Ca++, ↓Mg++).
7. Intracranial hemorrhage.
8. Drugs "naldixic acid = nigram" and aminophyllin.

**Convulsion (status epilepticus): emergency management:**

1. ABC (airway, breathing, circulation)
2. Recovery position (lateral)
3. Don’t put any thing in the mouth
4. Suctioning.
5. Diazepam IV: 0.1-0.3 mg\kg. slowly + O2 →

   #Fosphytoin → rapid rate of administration + ↓ irritation
   #never use 5% dextrose as it causes crystal
   #Diazepam should be given slowly (5-10 min) as it may cause respiratory depression.
   #Rout: IV or rectally using insulin syringe.

   #If no response after 20 min → give additional 10mg/kg phenytoin.
   #If no response → Give Phenobarbital “luminal” (20 mg\kg slowly).

   #Used with caution when co-administered with benzodiazepines as it may cause ventilator failure

   #Note: staring of the eye: 
   # upward → generalized
   # laterally → focal
If no response → Admit the pt. to ICU and call anesthetist for general anesthesia (propofol or halothane or ketamine).

**Investigation:**
1. Blood sugar
2. Serum electrolyte
3. CBC
4. Blood culture
5. CRP
6. **Lumbar puncture**: before you do it, do fundoscopy looking for papilledema:
   - If present → do CT scan to exclude brain mass.
   - If absent → do LP safely.

**Q/ If we give antibiotics before doing lumbar puncture what changes seen in CSF?**
CSF analysis will not change significantly but the culture always changes.

**Procedure of LP:**
1. Patient sitting position or leaning forward or laterally directed.
2. Sterilization of the area in circular pattern
3. Draw an imaginary line between the 2 iliac crests → insert the needle just above it between L4-L5.
4. Collect CSF (15 drops are enough) and check the pressure.

**What to do after collection of CSF sample?**
1. Inspection: clear crystal or turbid (infected).
2. Send for biochemistry: glucose, protein.
3. Send for Gram stain and microscopy.
4. Send for Culture and sensitivity test.

**Drugs induced fever:**
1. Pencillin.
2. Cephalosporin.
3. Quinidine.
4. Alfa-Methyldopa.
5. Nitrofurantin and INH.

---

**NOTES:**

# Turbid CSF contains: > 350 cell
# In acute bacterial infection, at early phase (1st to 12 hours) lymphocyte predominant, while later on, neutrophils predominant.
# CSF glucose should = ½ - ⅔ of blood glucose
# ↓ CSF glucose is called hypoglycorrhagia.

**NOTES:** In any convulsion do not forget to ask about:

# Fever, trauma, and symptoms of meningitis.
# Dysentery (shigellosis may cause convulsion).
# Cough and SOB → pneumonia may cause convulsion by (Cerebral anoxia and SIADH → cerebral edema as a result of hyponatremia)
# Skin rash → measles & roseola infantum can cause convulsion.
# Family Hx. (Febrile convulsion and epilepsy)
**Meningitis:**
Symptoms: pyrexia, headache, vomiting photophobia or even convulsion develops over few days.
Signs: meningism (brudizinski sign, neck stiffness, kernoing sign).

**CSF: Findings:**

<table>
<thead>
<tr>
<th>Type</th>
<th>Pressure in mm H2O</th>
<th>LEUKOC (cmm)&lt;5./75% ympho</th>
<th>Protein (mg/dl)</th>
<th>Glucose (mg/dl)&gt;50,75% of serum</th>
<th>Gram stain and culture</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute bacterial</td>
<td>Elevated 100-300</td>
<td>100-10,000 PMN</td>
<td>100-500</td>
<td>DECREASED &lt;40mg</td>
<td>positive</td>
</tr>
<tr>
<td>Partially treated m.</td>
<td>normal</td>
<td>5-10 mostly mononuclear</td>
<td>Same 100-500</td>
<td>normal</td>
<td>sterile</td>
</tr>
<tr>
<td>Viral men</td>
<td>80-150 slight increase</td>
<td>&lt;1000, mainly lymphocyte</td>
<td>50-200</td>
<td>Normal, except in mumps it decrease</td>
<td>sterile</td>
</tr>
<tr>
<td>Tuberculous meningitis</td>
<td>elevated</td>
<td>10-500 lymphocytes</td>
<td>100-3000</td>
<td>Decreased &lt;50</td>
<td>Acid-fast or culture positive for Tb.bacterial culture; ve</td>
</tr>
</tbody>
</table>

**Galactossemia:**
Decrease in G-1-PH UT enzyme which essential in glucose metabolism.

**Presentation:**
1. jaundice
2. hepatomegaly
3. feeding poorly
4. hepatic failure
5. vomiting

Q/Patient with galactosemia can’t lie for long time?
A/Because they are susceptible for sepsis with E.coli

**Indirect hyperbilirubenia:**
1. Gilbert syndrome
2. Criggler syndrome (type I or II)
Gilbert syndrome: Benign condition → causes indirect hyperbilirubinemia at time of stress such as fasting \ infection because of deficient of hepatic conjugate enzyme.

Direct hyperbilirubinemia:

1. **Biliary atresia** (diagnosed by US).
   - surgical treatment: (Kasai porto-enterostomy)
   - Should be done once biliary atresia is suspected.
2. **Neonatal sepsis**
3. **Hepatitis**
4. **Cystic fibrosis**
5. **Galactosemia**

Investigation:
1. TSB (total serum bilirubin) + differentiation (direct or indirect).
2. Blood culture
3. WBCs.
4. Reticulocyte count.
5. US of abdomen
6. PT + PTT
6. TFT (T3, T4 and TSH)

**Neonatal jaundice: DDx**
1. Biliary atresia
2. Cystic fibrosis
3. Metabolic disease (+ve family history, +ve consangity)
4. TORCH infection (most important → CMV)

**Stigmata of chronic liver disease:**
1. Jaundice
2. Spider naevi
3. Ascites
4. Palmar erythemia
5. Internal bleeding (intra abdominal, in joint) because of clotting factor deficiency
Causes of acute liver injury:
1. Viral hepatitis (A > B & others)
2. Acetaminophen poisoning > 200 mg/kg (dose in pediatric) while in adult is (7 gm)
4. Alpha anti-trypsin deficiency.
5. Glycogen storage disease: type 1 & 2 cause hetaptomegaly only.

Rx of chronic liver disease:
- Albumin 5 cc/kg
- vit.K, blood transfusion
  - If there is infection → antibiotics.

Respiratory distress syndrome: lack of surfactant due to prematurity.
- Surfactant formation starts at 28 wks and complete at 37 wks.
- For maturation of lung: give betamethasone before delivery 1 injection \ 24 hours

To differentiate RDS of respiratory source from CVS sourc:
→ Respiratory: respond to O₂
→ CVS: doesn’t respond to O₂
Side effect of ↑O₂ administration → bronchopulmonary dysplasia (long term complication)

DDx of RDS:
- Pneumonia, bronchiolitis, asthma, PE, ARF, DKA, anemia.

DDx of hyperinflated chest
1. Asthma.
2. Bronchiolitis.
3. Emphysema.

Infecitve Endocarditis:
Symptoms:
Prolonged fever, malaise, anorexia, weight loss, dyspnea, cough, SOB, myalgias, nightsweets, and joint pain.

NOTES: about Auscultation of heart:
#S1 beast heard at mitral area.
#S2 beast heard at pulmonary area.
#Loud S1 → ASD
#Loud S2 → Pul. HT, ASD and VSD
#S3 → (after S2) normal or anemia
#S4 → (before S1) always abnormal
#Murmur of aortic coarctation → interscapular
#HF → base of the lung.
Signs:
- Petechiae: not specific
- Splinter hemorrhage
- Jane way lesion: Non-tender macules on the palms and soles.
- Osler nodes: tender subcut. Nodules usually found on distal pads of the digits.
- Roth spots: Retinal hemorrhage (found in 5% of Pt.)
- Heart murmurs (85%): change in characteristics of previously heard murmur (10%).
- Signs of embolic stroke
- Signs of systemic septic emboli.
- Splenomegaly

Investigations:
2. ECHO → vegetation.
3. US.

Pathogen: S. viridians, fungal infection, s.epidermis (in catheterized pt.)

Indication for prophylaxis:
1. Prosthetic heart valve.
2. CHD.
3. Coronary heart stent.
4. Dental and genitourinary procedures.
5. Procedure on infected skin or musculoskeletal tissue.
6. Procedures involving incision in respiratory mucosa
7. History of IE

Developmental hx: من المحاظرة
In rota virus vomiting is before the diarrhea but not always.
Diarrhea due to small intestinal cause → low amount stool.
In colitis → large amount stool
One of the features of mal-absorption is → large bowel diarrhea
Shigella, sallmonilla, E.coli, campylobacter, pesdomembranous colitis, associated listeria.
Clostridium toxin for rapid assessment, skip AB
Antibiotics cause (watery diarrhea) eg: ampicillin

Q/How can you differentiate bloody diarrhea due to bact. or parasite?
A/ by chronicity: short period → bacterial, long period → parasitic

**Cow’s milk allergy:** cause colitis and bloody diarrhea.

Diarrhea is of two types:
1. non-infectious
2. infectious (viral, bacterial, parasitic, non-viral)
   * Bed dysentery (bacterial).
   * Walking dysentery (E. histolytica), IBD.

**Cow’s milks allergy findings:**
- Cough, skin rash, GIT problem, wheezes
- Bloody diarrhea Caused by → bovine, protein (casein)
- Can be replaced by: Hydrolyzed formula
- Side effect of this formula is → the taste (Not sweet)
- If not benefit → give amino acid Formula (elemental formula)
- From the mother transmitted by the breast when she ingested the cow’s milk then to the baby by breast feeding.
- Soy formula → isomil → Should not give it below 6 monthes, **because it contain estrogen**
- We can administer hypo-allergic diet

**Lactose intolerance: (2ndry)**

occur secondary to acute gastro-enteritis → chronic diarrhea with peri-anal excoriation, flatulence and distended abdomen.

#this is occurred due to mucosal damage of GIT by disturbance in lactase enzyme function. (The enzyme is located in the brush border of epithelial cells)

**Investigation:**

1. culture for bacteria
2. ELISA for viral mainly rota virus
3. microscopic for parasitic showing trophozoite
4. clostridia difficile.

**Investigation for Lactose**

1. ph stool: acidic
2. reducing substance in stool: +ve
   in urine >>> -ve mean galactosemia

**Convulsion associated with diarrhea:**

-(UTI, meningitis, Febrile convulsion, parental diarrhea)
- Electrolyte disturbance (hyponatremia).
- Hypoglyemia
- Shigllosis (convulsion + bloody diarrhea)

**How to prove?**

- Urine, stool and blood culture
- CSF.
- Serum electrolyte.
- RBS (random blood sugar)
**General exam:**
First of all ask about permission
Try not to disturb the child
a. General appearance:
1. General look (looks comfortable, ill, distress, dyspneic, irritable, semiconscious, drowsy, unconscious).
2. Dysmorphism (looks normal, any dysmorphic features).
3. Body built (looks thin, emaciated, good body built, thriven, I have to plot his parameters on growth chart)

**Hand:**
-Looks for clubbing but at least 6 months, peripheral cyanosis
-Nail $\rightarrow$ koilonychia (iron deficiency anemia), Leukonychia (hypoalbuminemia), splinter hemorrhage, capillary refill (2 sec), pale or not.

**Face:**
Eye: looks for conjunctiva for pallor, sclera for jaundice.
Mouth: (hygiene, color of tongue, dental caries (source of IE), ulceration, aphthous, pigmentation (addison disease) talk about any striking abnormality.

**Legs:**
Scar, color, edema.

#General examination is introduction for every systemic exam:
#Edema could be due to respiratory problem caused by right-sided heart failure (cor pulmonale).
**GIT exam:**

**General exam:**
Hand looks for clubbing (Look for angle between nail and nail bed → do shamroth’s test), palmor erythema, nail changes, pallor of creases

→ Causes of clubbing:
liver cirrhosis, cyanotic heart disease, lung (bronchiactasis, cystic fibrosis, empyema, pulmonary fibrosis (fibrosing alveolitis), familial, hereditary.

**Abdominal exam:**
Inspection, palpation, percussion and auscultation. (like adults)

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**Case: Cerebral palsy → findings:**
- Head → microcephalus.
- CP posture spastic → scissoring of legs.

**General exam:**
- looks ill, thin and emaciated, dysmorphic face, abnormal posture

**CNS exam:** Upper and lower limbs:
**Inspection:** abnormal posture, wasting, joint swelling, fixed deformity.
**Palpation:** tone of each limb, reflex, power and sensation.
**Hyper-reflexia** and **hypertonia** → due to upper motor neuron lesion.
**Babinski sign** → +ve

**Cranial nerve examination:**
Abnormal C.N 9 +10 (psudobulbar palsy) → inability to swallow, uveola deviated to side, loss of gags reflex, and loss of taste is post. ½ of the tongue.

**Hx of pt. with CP: important pointes:**
2. Per-natal → Rash, fever during pregnancy (cong. inf.)
4. Post-natal: Birth wt., crying, NICU, jaundice, hx of head trauma and CNS infection.
5. Family Hx: consanguinity, same condition.

**Diaplegia means:**
affection of both upper and lower limbs with lower limb predominance.
Lower limbs > upper.

**Psudobulbar palsy** → UMN lesion of cranial nerves 9+10 → paralysis of pharyngeal, laryngeal and soft palate muscles → dysphagia (as in patient with CP).
**bulbar palsy** → same as pseudobulbar palsy but it’s due to LMN lesion of cranial nerves 9+10.
Why pt. with CP is liable for recurrent chest infection?
- Because of recurrent aspiration, which is due to:
  1. Psudobulbar palsy.
  2. GERD (many pts. with CP have concurrent GERD)

Cerebellar exam:
1. Speech (scanning or dysarthric speech).
2. Eyes (Nystagmus).
4. Rapid alternating movement (dysdiadochokinesia).
5. Finger–nose test → dysmetria (dysmetria).

Rx: -Medical → PPI, H₂ blockers and domperidon.
- Surgical (Niesson fundiplication)

Important causes of CP:
Prenatal: cerebral malformation, congenital infection.
Natal: prematurity and birth asphyxia
Post – natal: Kernicterus, non specific head trauma and CNS infection.

Meningism: consists of headache, photophobia and neck stiffness, often accompanied by other signs of meningeal irritation including:
Kernig’s sign (extension at the knee with the hip joint flexed causes spasm in the hamstring muscles) and Brudzinski’s sign (passive flexion of the neck causes flexion of the hips and knees).

#Meningism is not specific for meningitis.
#Causes of meningism:
  1. meningitis.
  2. Subarachnoid hemorrhages.
#Conditions that may mimic meningism:
  1. cervical spondylosis.
  2. Tetanus.
  3. ↑ICP

Examination steps:
- Position the patient supine with no pillow
- Expose and fully extend both the legs.

Neck stiffness:
- Put one hand under the occiput of the baby and support the body with the other hand → try to flex the head forward aiming to make the chin touches the front of the chest.
- If there is neck stiffness → there will be resistance to flexion movement.
- If the pt. is old enough → ask him to touch the front of his chest by his chin.
**Brudzinski sign:** While you are examining for the neck stiffness, if the flexion of the head was accompanied by flexion of the knees → +ve Brudzinski sign.

**Kernig’s sign:**
- Flex the hip and knee of one side to 90°.
- Put your left hand over the hamstring muscles (posterior thigh) → extend the flexed knee → if the there is any resistance to extension or the hamstring muscle spasm → +ve Kerning’s sign.

↑ICP: can cause meningism: Symp. and Signs →

**Note:**
• 6th C.N palsy → **Convergent** squint (can’t abduct the eye of the affected.
• 3rd C.N palsy → **Divergent** squint (can’t adduct the eye of the affected side) with ptosis and dilated pupil (No parasympathetic).
• Squint caused by nerve affection is called → paralytic squint.
• How to differentiate between paralytic and non-paralytic squint?
  - By cover test: Cover the normal eye:
    → if the squint eye is corrected → non-paralytic squint.
    → if the squint is NOT corrected → Paralytic squint.

**Facial palsy (VII):** due to UMNL or LMNL.

**In UMNL:**
1. Deviation of angle of mouth (toward normal side).
2. Flattening of the nasolabial fold.
3. Inability to blow cheeks.
4. Preservation of the upper face (check it by asking the pt. to elevate his eyebrows and see the wrinkles in the forehead).

**In LMNL:**
1. Deviation of angle of mouth toward normal side
2. Flattening of the nasolabial fold.
3. Inability to blow cheeks.
4. **Dropped eyebrow (of the affected side).**
5. Loss of forehead wrinkles.
6. Inability to close the eye and +ve Bell’s phenomenon: when you ask the pt. to close his eyes → the eye of the affected side will not be closed, so the pt. elevate his eye upward in order to seem like he is closing his eye.

# Most common cause of VII C.N palsy is Bell’s palsy.
# Bell’s palsy: is LMNL of facial nerve which is mostly idiopathic.
# Other causes of facial n. palsy include:
2. Complicated otitis media.
3. Gullian-Barre syndrome (GBS)
4. Hypertension: also can cause facial palsy.
5. Trauma.

So, when you face a case of facial palsy (LMNL) → the next step will be:
→ Examination of ear looking for herpetic vesicles (In RHS) and signs of otitis media.
→ Examination of the lower limbs for → hypotonia and hyporeflexia (ascending paralysis in GBS).
→ Examination of blood pressure.
→ Examine for any sign of trauma.

# Examination of the cranial nerves

Start by general exam for example: the baby is conscious alert (small child) or oriented (older child), normal posture or lying supine or in the lab of his/her mother, no squint, no nystagmus, no facial asymmetry, no any dysmorphic features.

Olfactory nerve (I):
1. Exam each nostril separately
2. You should confirm that the nostril is patent (Rhinitis (increased secretion), foreign body obstructing the nostril interfere with smell)
3. Bring familial odors as apple or tea or others in tubes specific for smell examination
4. Never use irritant smell because its sensed by the ophthalmic division of the V cranial nerve not olfactory nerve.
Optic nerve (II):

Examination: It has five components to be examined:

1-Visual acuity: Above 4 months you can use Fixation and follow test, this done by holding an interesting toy about one meter in front of the baby, once baby fixes his vision to it, you start to move it in an arc, if the baby follow it, consider that this baby has normal vision.

Below 4 months, the baby can follow faces and light only. Above 7 years (cooperative child), we can use Snellen chart Sometimes Allen’s chart (pics of animals with different sizes) can be used below 7 years.

2-Visual field (in cooperative child): One meter distance between doctor and baby then the doctor closes one eye by his hand and the child asked to close the eye opposite to the doctor eye, then the doctor start to test upper, lower and right and left fields of each eye separately by comparing with his eye considering that the doctor has normal visual fields.

3-Color vision: (in cooperative and old child): This can be tested by using Ishihara test, which consist of book containing different figures with different colors, and the child is asked about these figures (considering that the child knowing these figures e.g. numbers, pictures etc.)

4-Pupillary reflex: Optic nerve form the sensory (afferent) part of this reflex, while the motor (efferent) part is oculomotor (will be discussed next)

Oculomotor, trochlear and abducent (III, IV, and VI):

Examination: These nerves should be examined simultaneously, their examination consist of three components:

1-Eyeball movement: One meter between the child and examiner:
   -In small child move an interesting toy in H shape pattern and look at the child if he follows the toy by his eyes, in the same time you should look for sequent and nystagmus with each movement.
- In older cooperative child you can move your finger or any object in H shape pattern and asking him/her to follow, in every movement you should look for sequent and nystagmus and ask about diplopia.

2-Accommodation:
- In small child, move an interesting toy toward the nose, normally there will be ptosis, conversion and meiosis of both eyes. 2-In older child, you can move your finger or any other object same as above. 3-Pupillary reflex: Afferent: optic nerve Efferent: Oculomotor nerve This test has two parts:

1-Direct: By using light torch, come out of the visual field and direct the light toward the pupil, normally the pupil size will decrease

2-Indirect (consensual): the same maneuver but this time you put a barrier between the two eyes, then direct the torch toward one eye and look at the other eye for pupil constriction (normal consensual reflex).

Notes: Types of squint: divergent squint and convergent squint. Diplopia: as patient looks downward like step down a ladder. Dilated pupil

3rd cranial nerve lesion lead to: Downward divergent squint (inferolaterally) + ptosis + lack of accommodation (mydriasis) + lacrimation also affected.

Trigeminal nerve (V): Function: motor, sensory, reflexes.

1-Motor: supply the muscles of mastication by the motor fibers of the mandibular division which includes:
   a-Masseter muscle, temporalis muscle: closing the mouth (clenching the mouth)
   b-Lateral pterygoid muscles: open the mouth and moving the lower jaw from side to side (lesion in the nerve causes deviation of the jaw toward it).

2-Sensory: Supply the skin of the face by the three division as follow:
   a-Ophthalmic; supply the skin above the imaginary line that runs from the middle of cranium downward to the lateral angle of the eye.
   b-Maxillary; supply the skin below the imaginary line as described above and above an imaginary line from anterior third of the lateral part of the cranium to the angle of the mouth.
   c-Mandibular: supply the skin below the imaginary line as described in b.
3-Reflexes:

a-Corneal reflex: Afferent: Ophthalmic division of trigeminal. Efferent: Branch from facial nerve causes blinking of the eye.

Two ways of examination:
1-Blowing on the child eye (usually not done due to risk of transmit infection to child as respiratory infection) its positive when the child blink his eyes.
2-By using cotton, bring it out of the field of the child and touch rapidly the corneoscleral junction, positive when the child blink his eye(also not done due to risk of corneal ulceration)

b- Jaw jerk:
Afferent: 5th CN mandibular division.
Efferent; 5th CN mandibular division.
Ask the child to slightly open his mouth, put your index finger on the the chin and by taping it by the hummer, you will notice slight or no upward movement of the jaw normally, while brisk upward movement seen in upper motor neuron lesion.

Facial nerve (VII):

Function: motor supply for facial muscles and sensation of taste of the anterior two thirds of the tongue.

Anatomy: nucleus in the pons.

Examination: In facial nerve you should examine 3 components:

1-Motor: facial muscles (4 muscles which are; frontalis, orbicularis oculi, cheek muscles and orbicularis oris) and this done as follow:
   a-Ask the child to look upward while fixing his neck in normal anatomical position, check for forehead wrinkles bilaterally
   b-Ask the child to close his eyes as much as he can till the eyebrow is buried, then try to open his eyes by your hands to check for resistance.
   c-Ask the child to blow against closed mouth, also check the resistance by pushing his cheeks with your fingers
   d-Ask the child to smile, look for the angle of the mouth if they are normal or there is mouth deviation. If there is a lesion in the right fascial nerve, the mouth will be deviated toward the normal side. All muscles affected in upper motor neuron lesion, while only cheek muscles and orbicularis oris muscles are affected in lower motor neuron lesion,
because the upper two muscles supplied by nerve fibers that come from the upper half of fascial nucleus, and the lower two muscles supplied by nerve fibers that originate from the lower half of fascial nucleus, the upper half has innervation from both cerebral hemispheres therefore when the fibers come from one hemisphere affected, the branched from the other side will take its place, while lower half of the nucleus take innervation only from the ipsilateral hemisphere, therefore when its fibers damaged nothing take its place!

2-Sensory: it takes taste sensation of the anterior two thirds of the tongue by its sensory branch, chorda tympani. To examine this typically the tongue should be pulled by forceps out of the mouth, and then put drops of specific taste on its specific area of sensation (sweet on the anterior part of the tongue, salty and sour on the lateral side and biter on the posterior part of the tongue)

#Note: Ramsay-Hunt syndrome, its herpes zoster of the sensory part of the facial nerve of part of the external auditory canal.

- **Facial nerve palsy**: Upper motor neuron lesion: only lower part affected (toward lesion). Lower motor neuron lesion: Bell's palsy, all sites of face, no wrinkling, deviation of mouth toward same side.

**Vestibulocochlear nerve (VIII):**

1-Hearing:
- a-Audiometry; it’s a device that needs cooperative child > 5 years
- b-Tonic fork; used in children > 5 years, also needs cooperative child.

2-Balance:
- a-Nystagmus: there are 3 types, transverse, vertical and arc movement. You can examine for it by two methods:
  1-Water caloric test: a-warm water (44°C and above) introduced in the external auditory canal, head will turn to the ipsilateral side, both eyes will turn toward contralateral side with horizontal nystagmus toward the ipsilateral ear. b-Cold water (30°C or below) introduced in the external auditory canal, head will turn to the contralateral side, both eyes will turn toward the ipsilateral side with horizontal nystagmus toward the
contralateral ear.
2-Hallpike test; Check Macleods for further information b-Ataxia: Incoordination of body movement.

**Glossopharyngeal and vagus nerves (IX and X):**
Examination: You should examine these nerves together and start as follow:
1-The Uvula, should be central, if there is lesion in one side the uvula should deviated to the normal side. Ask the baby to say Ahh to demonstrate the uvula clearly.
2-Gag reflex; by touching the posterior wall of the pharynx by tongue depressor (afferent by glossopharyngeal), this will cause contraction of the pharyngeal muscles (efferent by vagus nerve), this reflex induces sense of vomiting.

**Accessory nerve (IX):**
1-Ask the baby to elevate or shrug his shoulder, and push against his shoulders by your hands.
2-If the child is small, move an interesting toy in front of him from side to side to check for sternocleidomastoid muscles
3-If the child is old and cooperative ask him to look to the side, the push his jaw by your hand and ask him to push against your hand.

**Hypoglossal nerve (XII):**
Examination: (you need cooperative child):
1-Examine the tongue in resting and look for:
   a-Wasting
   b-Fasciculation (fibrillation when seen on EMG, but in tongue examination we can use both words)
2-Examine the tongue after asking the baby to protrude his tongue out of the mouth and look for a-Weakness b-Deviation (deviation is toward the abnormal side)
3-Then ask the baby to close his mouth and push his cheeks by his tongue.

**#Baby during feeding uses 9 cranial nerves:**
- Eye to eye contact with mother by optic nerve (indirectly related).
- Eye movement by 3rd, 4th and 6th cranial nerves (indirectly related).
- Muscles of mastication by V CN (directly related).
- Facial muscles (sucking muscles) by VII CN (directly related).
- Muscles of deglutition by IX and X CNs (directly related).
- Swallowing (tongue) by XII CN (directly related).
If more than 7 years: You can proceed in examination as follow:

#Olfactory

#Optic:
- Visual acuity: snellen chart
- Visual field: confrontation test or perimetry
- Papillary reflex

#3, 4, 6 (H-shape)

#5 cranial nerve (trigeminal):
- Sensory
- Motor ---> mastication
- Jaw jerk reflex (+ve UMNL)
- Corneal reflex

#Facial deviation to normal side, flattening of nasolabial fold to the affected.

#8 cranial (vestibulocochlear):
- Whisper in child ear

#9, 10 cranial nerve: swallowing, say ahh there is uveal move

Bulbar palsy (UMNL), pseudobulbar (LMNL)

- Pseudobulbar (supra-nuclear), bulbar (infra-nuclear)
- NG tube or gasrostomy
- Post 1/3 of 9 GN taste

#Accessory n.

#12 cranial nerve: tongue affected deviation, fasciculation, wasting

- Vagus: uveal pulled to normal side

In infant:

#Sucking 9, 10 (swallowing) look to his mother

#Move a toy in front if him for 3, 4, 6

#Crying for facial palsy deviation

#Jaw jerk
Exam limb:

**Appearance**: Scissoring of leg: CP, wasting of muscle.

**Feel**:  
- Tone (ankle, knee, hip)  
- Power  
- Reflex  

#How to examine proprioception?  
- Explain to patient → close the eye → ask the pt. to tell you to which direction his joint is moving (upward and downward)  
- Romberg sign: pt. is doing well when eyes are opened → ask him to close eyes → if the pt. start to deviate and lost his balance → Romberg sign +ve → defect in proprioception (dorsal column of spinal cord) and not cerebellum as pt. with cerebellar ataxia has unbalance even when his eyes are opened.  
- Plantar reflex (Babinski sign): Normally +ve in children <2 years.  
  one of the reflexes occur in infants, responses when body receives certain stimulus, after sole of foot firmly stroked, big toe upward and others fan out  
- normal in children up to 2 years and disappear as child gets older, may disappear as early as 12 months.
The degree at which the pt. is considered febrile = 38.3 centigrade.
* The tool for measuring temperature is "thermometer"
* Sites of measuring the body temperature:
  1. Oral.
  2. Axillary: the most commonly used in children.
  3. Rectal: the most accurate route
  4. From tympanic membrane: otometry

* Axillary temperature = 0.5°C less than that of oral & 1°C less than that is of rectal

* Be careful drug fever? But which drugs?
  - Injections like ampicillin or ceftriaxone even Intravenous fluid could elevate our body temperature,

* Investigations you may send in feverish pt.:
  - CBC
  - ESR
  - CRP
  - CXR (if cough is present or any sign of RD)
  - US
  - Even B.M exam

* PUO: pyrexia of unknown origin: 8 days of elevated body temperature (fever) with basic physical examinations & investigations (as inpatient or outpatient) but there is no clear cause.

* Always in fever ask about travelling to other areas.
*Some of researchers put priority for certain diseases as causative agents of PUO:
1-UTI
2-Kawasaki disease
3-EBV infection

*rash types also can give you clues about the underlying disease.
*is there any rigor? "Rigor in brucellosis is common"
*is there any sweating “as in T.B"
*2 spikes of increased body temperature: Falciparum malaria
*fever at morning & evening: typical of Kala – azar.

**Kawasaki disease**: fever for 5 days, injected eyes, redness of mouth with fissuring of lips, ↑ESR, ↑CRP and thrombocytosis.

*most PUO remit with time ""pyrexia of unknown source"

NOTES: important points in Hx that shouldn't be forgotten:-
1-where does the pt. lives?
2-Who diagnose the fever, mothe or doctor? And how?

*In convulsion Hx taking:-

- Ask about vaccination recently? DPT can lead to convulsion.
- Vomiting and diarrhea? because this can result in electrolyte disturbance→convulsion
- Look for signs of rickets→hypocalcemia→tetany.
- Sometimes tonsillitis or otitis media + fever→convulsion, so don't miss the examination of tonsils and ear.

*Causes of big head:
1- Congenital hydrocephalus.
2- Storage disease.
3- Arnold-Chiari malformation→herniation of cerebellar tonsils through the foramen magnum→Non-communicating hydrocephalus.

#Q/when you face a child with big head, what is the next step in examination?
A/-Fontannelles .
   -Sutures
   -Back for spina bifida (meningocele or meningomyelocele). → Most important.

#Dandy-walker syndrome: complete absence of cerebellar vermis with enlarment of the 4th ventricle→leads to enlargement of skull posteriorly (bulging at back of head)
→ unlike hydrocephalus which enlargement of entire head.
In pt. sudden cessation of urination: try to concentrate on these points in the history: could be → **anuria or retention of urine**.

1- Is the child constipated? → Hard stool can press on the urethra → cessation of urine (especially in females).

2- Vomiting and/or diarrhea present? → Dehydration → pre-renal failure.

3- Evidences of UTI.

4- Menstrual Hx and puberty in young girls → if you suspect **hematocolpus** (imperforated hymen), and examine the breast for Tanner staging.

**Menstrual cycle usually start at 3rd or 4th Tanner stage.**

**Other causes of sudden cessation of urination:**

1- acute renal shutdown
2- drugs
3- GN
4- renal tubular necrosis
5- HUS

⇒ the pt. is edematous + tired + acidotic.

Investigations you may send in such a pt. : according to the condition you suspect:

- CBC/Blood culture
- RFT
- US
- CT scan.

#Oliguria <1ml/kg/hr #Anuria <180ml/24 hrs.
Questions from previous years:

- A neonate 1month age , with persistent jaundice , hard stool , posterior fontanlle open
  
The result of investigations:
  
  TSB( total serum bilirubin ) =? , Hb=14g/dl , T3=low , T4=low , TSH= high
  
  Dx:-Cretinism (1ry or congemenital hypothryoidism).

- A child 2years old ,presented with fever for 2 weeks ,
  on exam:-
  
  his temperature = 38.9 degree centigrades , spleen ,L.N all are NOT palpable ,liver
  1finger palpable.
  
  1. mention two possible differential diagnose?
  2. mention most important investigation ?

  
  Hint:
  Platlets count=60
  Hb=6g/dl
  Bilirubin=normal
  WBCs =5000 (5 x 10⁹/L)

  
  DDx:
  1-leukemia
  2-Aplastic anemia
  Others (rarely parvovirus infection, infectious mononucllosis)

  
  Investigation:-Bone Marrow exam.

  
  Note: always consider B.M exam in pancytopenia ,(very imp)
  When two line of cell affected in bone marrow then we need BM biopsy
Neonatal jaundice:

65% of infants are having jaundice in 1st wk of life. In most cases it is benign condition & resolved spontaneously. Others are pathological and can end with persistent damage and kernicterus.

Pathophysiology of physiological jaundice:
-the liver is incapable of dealing with the excess bilirubin, this excess bilirubin resulted from the normal polycythemic state of the infant in the intrauterine life, when baby is delivered there will be no need for the polycythemic state (lazy circulation is being established), and hyperviscosity syndrome so the excess RBCs will be degraded & bilirubin level will start to raise.
-This bilirubin needs mature hepatocytes to be dealt with & sufficient amount & activity of transferase enzymes.
-Prematurity predisposes neonates to more severe jaundice.

Notes:
• Dusky color of a neonate masks the yellow tint of jaundice and that is explaining the late presentation of jaundice baby.
• Hb in neonate = 18 ± 2.
• There is physiological anemia in 2 months infant
  ➔ [Hb] = 9g/dl (full term baby) and 7.5g/dl (preterm baby).

| If [Hb] is <9 g/dl (in full term) or <7.5 g/dl (in premature) ➔ Consider the baby as anemic. |

Bilirubin is one of the most potent anti-oxidant, when it is raised in the early days of life this will help in getting rid of the free radicals.

• 1g/dl of Hb will give 34mg/dl of bilirubin.
• Papkin's reflex: palmomental reflex, if persisted beyond 4-6mo, this is mostly due to frontal lobe damage ➔ "CP".
• Glanzmann disease: platelets dysfunction.
• In first days of neonate life Hb < 18 mg/dl considered anemic
**Hx of baby with jaundice:**

1. **Cause**
2. **Effect of this jaundice**
3. **Duration** (in hours not days)
4. **LMP/EDD**
5. **Birth weight, Rh, ABO of father and mother**
6. **Prenatal** (mother's disease, chemical exposure, drugs, Hx of maternal hepatitis.)
7. **Natal** (mode of delivery)
8. **Postnatal Hx**
   - Did he cry immediately after birth? Needed resuscitation?
   - Time of start of feeding? Type?
   - Child now receives adequate nutrition?
9. **Ask about signs & symptoms of kernicterus?**
   - Poor sucking
   - Poor reflexes (by examination ➔ absent Moro reflex)
   - Lethargy
10. **Family History:**
    - Any of siblings have the same condition?
    - Needed phototherapy? Exchange transfusion?
    - Hx of hemolytic diseases (G6PD" & hereditary spherocytosis) or early cholecystectomy.

\[\text{#β-thalassemias and sickle cell disease can't present as neonatal jaundice as there is minimal HbA in the first 3-6 months of life an the majority of Hb is HbF.} \]

\[\text{# α- thalassemia can present as neonatal jaundice.} \]

At last you can say: the health of the baby is severely disturbed.

**Example:**

A baby who was delivered by NVD at (39) wks was presented to hospital with jaundice started at 2\textsuperscript{nd} day as it was observed by the family.

**Physical exam:**

- Is it jaundice really??
  
  By gentle pressure on the nose or forehead or sternum.
- What is the cause?
- What is the effect?
• Eye exam: if the eyes are closed by holding the baby from axillae, bending him forward & backward, he will open the eye & we can see the sclera. Don’t try to open eye forcefully because this can lead to orbicularis oculi muscle damage.
• Measure the wt?
• Mature or premature?
• Dysmorphic features?
• Anterior & posterior fontanelles? "wide posterior fontanelle in hypothyroidism"
• Birth markers? (bruises & cephalohematoma).
• Hepatosplenomegaly?
• Rash? "TORCHS infection & severe Rh incompatibility"
• Anemia "pallor"
• Activity "primitive reflexes"

**Important definitions:**

**Small for gestational age:** birth weight <10th centile mostly due to IUGR as a result of placental insufficiency "fetal hypoxia" → 2ry polycythemia → increase Hb breakdown → increase bilirubin → jaundice

**Large for date:** birth weight >90th centile → infant of diabetic mother. Poorly controlled maternal diabetes → intermittent periods of hyperglycemia → Hyperglycemia in the fetus → stimulation of insulin, insulin like growth factors, growth hormone, and other growth factors → stimulate fetal growth and deposition of fat and glycogen → Macrosomia

**LBW:** Birth weight <2500kg.

**Premature:** baby NOT completed 37wks gestational age calculated from 1st day of LMP.

**Full term:** 37-42 wks

Rash + jaundice = TORCH infection

Criteria of physiological jaundice? Important من محاضرة النظري
**Rh incompatibility:**
- No naturally occurring Anti-D, it should be given once there is suspicion of feto-maternal bleeding: abortion, birth trauma, previous still birth.
- Anti-D must be given at 28-32 wks of gestation within 72 hrs after delivery to prevent sensitization of maternal blood.
- Once sensitization occurs (↑ anti-D titer in maternal blood) → Anti-D injection is useless.

Note: we give anti-D to the mother to get rid of the RBC, that is escaped from baby’s blood to maternal blood TO PREVENT MATERNAL IMMUNE SYSTEM FROM BEING SENSITIZED AND PRODUCTION OF IT’S OWN Ab.

**History:** yellowish discoloration started from the 1st day, his mother Rh-ve.
His brother has the same condition, or one of his brothers has kernicterus.

4 investigations:-
1- TSB: direct and indirect
2- CBC and retic count.
3- hematocrit (PCV)
4- Rh & ABO testing
4- coomb's test: strongly +ve.

**ABO incompatibility:** >common than Rh incompatibility.
Mother =O
Infant=A/B/AB.

**Investigations:**
Coomb’s test = weakly –ve or weakly +ve.
Retic count: ↑
Hb: ↓

In the film of ABO incompatibility:
Brisk spherocytosis, this condition is also encountered in:
1- Autoimmune Hemolytic anemia.
2- Burns.

When there is sign of hemolysis with ve coomb’s test you should think about:
- ABO incompatibility.
- non immune cause of hemolysis as:
  1. Enzyme deficiency (G6PD, PK deficiency).
  2. Cell membrane disease spherocytosis
  3. alpha Thalasaemia.

<table>
<thead>
<tr>
<th>Phenotype</th>
<th>Genotype</th>
<th>Antigens</th>
<th>Naturally occurring antibodies</th>
<th>Frequency (UK) (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>O</td>
<td>O0</td>
<td>O</td>
<td>Anti-A, anti-B</td>
<td>46</td>
</tr>
<tr>
<td>A</td>
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<td>Anti-B</td>
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</tr>
<tr>
<td>B</td>
<td>BB or BO</td>
<td>B</td>
<td>Anti-A</td>
<td>9</td>
</tr>
<tr>
<td>AB</td>
<td>AB</td>
<td>AB</td>
<td>None</td>
<td>3</td>
</tr>
</tbody>
</table>

#Mother A (or B) have baby with B( or A) → is there any sensitization?
- Although Anti- A & Anti - B Ab are naturally present without previous immunization that is usually in the form of IgM that can not cross the placenta, but when the mother exposes to A or B Ag (during first ABO incompatibility pregnancy or abortion) leading to maternal sensitization & formation of IgG which can cross the placenta leading to fetal hemolysis during 2nd or next ABO incompatibility pregnancy.
G-6-PD deficiency

Characteristics:
- ↓ Hb
- ↑ Retic. count.
- Family Hx: +ve X-linked recessive (Brother, mother, aunt)
- Coomb's test ➔ -ve.

The mother is carrier in 40-60% of cases.

Management of jaundice in the 1st week of life:
- 25 mg/dl Bilirubin is considered the limit border
Either there is no need for Tx especially if there is no hemolysis, full term baby, healthy active and the jaundice is not deep with criteria of physiologic jaundice.
- For safe management start at 20 mg/dl to prepare for blood transfusion

Double volume exchange

Technique: a double volume exchange removes approximately twice the infant circulating blood volume 170 ml/kg of the (infant is approximately 80-90 ml/kg body wt.) replacing it with cross matched whole blood. The procedure involves placement of central catheter and removing and placing blood in a volume that is approximately 10% or less of the infant blood volume.
Most of bilirubin is extravascular, as a result exchange transfusion remove approximately 25% of the total body bilirubin, after the procedure serum bilirubin falls to approximately ½ of pre exchange value then increases to ⅔ of that levels as extravascular and vascular bilirubin re-equilibrate.

Side effects of exchange transfusion
1. Hyperkalemia (if old blood is being used > 72 hours).
2. Hypocalcemia (if Ca++ hadn’t given)
3. Hypoglycemia (Glucose must be monitored especially in Rh incompatibility because of hyperinsulinemia or pancreatic islet hypertrophy).
4. Volume overload (if your transfusion wasn't accurate).
5. Shock (if large amount had been drawn at first).
6. Anemia (if you didn’t shake well the contents, RBCs precipitate downward)
- you should monitor the heart
- Should be in a controlled environment to avoid hypothermia
- Acid glucose-phosphate added
- Phosphate chelate the calcium can lead to convulsion because of hypocalcemia.
- Bicarbonate added to resolve acidosis.
- MR (mortality rate) is 1-2%
- Advice mother to prevent feeding before 1 hour of procedure.
- Check blood glucose.
- Q/Pt. with Rh incompatibility ➔ glucose + hypothermia, why?

A/ patient Rh incompatibility has Nesidioblastosis which means hyperinsulimic hypoglycemia (due to hyperplasia of pancreatic islet cell this lead to surge secretion of insulin which cause hypoglycemia).

**Phototherapy**
- visible spectrum of light (it is NOT ultraviolet light).
- The most appropriate spectrum is blue light with 450nm wave length.

**Mechanism:** bilirubin in the skin absorbs the light energy which by photoisomerization converts the toxic unconjugated bilirubin to a product which can be excreted in bile without the need for conjugation, also phototherapy converts unconjugated bilirubin to lumirubin which is excreted in the kidney.

**Side effects of phototherapy:** (very important).

| i | Hypo/hyperthermia |
| ii | Dehydration |
| iii | Diarrhea |
| iv | Rash |
| v | Bronze baby syndrome (if the pt. has direct hyperbilirubinemia) |
| vi | Eye injury & nasal obstruction |

**Note:**

- TSB at first 7hrs, begin to rise, then it goes down as the phototherapy continued.
- As you perform phototherapy, you should increase the maintainance fluid by 10%-30%.
Jaundice (prolonged neonatal jaundice): ➔>10days
Divided into two divisions:

<table>
<thead>
<tr>
<th>Indirect hyperbilirubinemia</th>
<th>Cholestatic jaundice</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conjugated (direct) bilirubin &lt;20% of total Serum bilirubin.</td>
<td>Conjugated (direct) bilirubin &gt;20% of total serum bilirubin</td>
</tr>
</tbody>
</table>

**4 causes prolonged jaundice:**

1- Hemolysis
2- crigler-Najjar syndrome
3- Breast milk jaundice
4- Hypothyroidism.

![Prolonged jaundice + indirect hyperbilirubinemia ➔ screening for hemolysis is indicated.](image)

**Direct hyperbilirubinemia**

1- Extrahepatic biliary atresia: clay color stool, portal hypertension.
# Surgery is the only treatment: kasai-portojejunostomy. (Alkaline phosphatase raised as there is biliary obstruction)
# Mal-rotation of mid gut ➔ diagnosed by: Ba enema.
# Choledochal cyst ➔ diagnosed by Ultrasound.

2- idiopathic neonatal hepatitis ➔ mostly male, normal color stool & consistency for some time before the condition deteriorates//ALT & AST will be raised.

![How to differentiate between the two?](image)

By HIDA test: Radio-isotope in vein:

➔ if the uptake is Normal but the excretion is slow ➔ biliary atresia.
➔ idopathic neonatal hepatitis

Rx:-
1- Medium chain triglycerides.
2- Vitamins ➔ injection (A, K, E, D).
3- Cholestramine
4- Supportive care.
5- Liver transplant.
Others:
- TORCH → serology
- HBV/HCV → serology
- Sepsis (Blood, CSF & urine cultures)
- UTI → urine culture.
- Cystic fibrosis → sweat chloride test
- Alpha -1- anti-trypsin deficiency → (immunoassay).
- Galactosemia (deficiency in Glucose -1-PUT): this enzyme is estimated in RBCs & fibroblasts // if there is +ve reducing substance in urine other than glucose means → galactosemia → (Isomil is prescribed for them)

**Notes:-**
- Glucose deposition in the eyes → cataract.
- In brain → convulsion
- In kidney → RTA
- In liver → jaundice
Shortness of breath:

- **NOTES:**
  - Duration ➔ acute or chronic
  - If patient presented with recurrent pneumonia and received antibiotics With no improving think about **Foreign body aspiration**
  - **Asthma a common cause for recurrent admission**
  - Using accessory muscle of respiration mainly sternocleidomastoid muscle in respiratory distress causes head nodding.
  - Frontal bossing is one of the features of rickets.
  - **Infant with breast feeding more liable to rickets.**
  - Vit. D deficiency patient more risky for asthma.
  - Bowing of the leg during the period (8 – 24) months is physiological.
  - Delayed tooth eruption can be due to Rickets

Ask also about: Bluish discoloration, whooping cough.

**Whooping cough: rare in the 1st year of life.**
#Post – tussive emesis is not characteristic (but not pathognomonic)
#Ask about feeding, sleeping if there is disturbance, ➔ indicate severity of disease.
#Ask about convulsion, whooping cough can lead to convulsion by inducing cerebral hypoxia.

**IN reveiw:**
**Renal:**
- Ask about: urine output ➔ Very important.
- ➔ UOP in pt. with SOB ➔ is a sign of dehydration.
- Dehydration ➔ may cause pre-renal failure.

---

**Characteristic:** one of the disease features.
**Pathognomonic:** specific only for this dis.

**Causes of post-tussive emesis:**
Asthma, Bronchiolitis and whooping cough.

**Pneumonia can cause convulsion by:**
1. Cerebral hypoxia or anoxia.
2. SIADH ➔ Hyponatremia ➔ cerebral edema

**Causes of dehydration in pt. with SOB:**
1. ➔ food and water intake.
2. ↑ insensible loss by sweating (due to fever) and rapid breathing
**Prei-natal Hx:** ask about:
- Maternal fever, rashes and any illness, leaking liquor, gestational age, prematurity?, cry immediately and NICU admission ➔ RDS
- Meconium aspiration, usage of mechanical ventilation ➔ may cause bronco-pulmonary dysplasia.
- Oligohydramnios ➔ hypovoluemia ➔ heart failure
- Polyhydramnios ➔ chest compression ➔ affects growth of lung ➔ hypoplastic lung.
- Hypoplastic kidney in intrauterine life ➔ oligohydramnios ➔ renal failure and metabolic acidosis at birth.

**Feeding Hx:**
- Is there any aspiration during feeding?

**Past medical Hx:**
Recurrent problems, asthma, heart failure, pneumonias.

**Family Hx:**
Child contact with TB patients, asthma, cystic fibrosis, kartagener syndrome.

**Social Hx:**
Domestic animals, overcrowding, sewage, air conditioning.

**ON EXAM:**
- Face of the child ➔ looks ill or will.
- Color: cyanosis?, pink color or pale
- Posture: patient can’t lie in asthma, tripod position in Severe SOB (acute epiglotitis).
- Sign of respiratory distress (flaring of ala nasi, subcostal, suprasternal resscion and use of extra muscles of respiration), deviated trachea.
- Hydration state, level of consciousness.

**Investigations:**
- CXR
- Blood gas analysis (PO$_2$ ➔ normal (60-90 mmHg) if < 60 mmHg, OR PCO$_2$ (normal 35-45 mmHg) if > 45 mmHg ➔ Mechanical ventilator.

---

**Causes of aspiration:**
- **Direct causes:** Cleft palate, Tracheoesophageal fistula (H type), bulbar or (psudobulbar palsy as in CP).
- **Indirect causes:** Over feeding, GERD, esophageal dysmotility (transient).
Neonatal Emergencies:

- **Birth asphaxia**

  First of all the asphyxiated newborn baby should be put on a resuscitation trolley where the baby put under a radiant heater to avoid hypothermia then drying up of the baby, the head is positioned down & slightly extended, the airway is cleared by suctioning, and also gentle tactile stimulation provided (slapping the foot or rubbing of the back).

  If spontaneous respiration started and the cardiac output improved where the color of the baby becoming pink, then there is no need now to go onto further steps of resuscitation, but if these measures fails to improve the condition of the baby and the heart rate is < 100/min so we need:

  2- Positive pressure ventilation with a 100% oxygen is given through a tightly fitted mask & bag for 15-30 sec, subsequent breaths are given at a rate of 40-60/min with pressure of 15-20 cm water. Successful ventilation is determined by good chest rise symmetric breath sounds, improved pink color, heart rate of >100/min, spontaneous respiration and improved tone. If no response within 15-30 sec.

  the next step is:- Ambu bag Traditionally, the inspired gas for neonatal resuscitation has been 100% oxygen. Resuscitation with room air (or 30%) is equally effective and may reduce the risk of hyperoxia, which is associated with decreased cerebral blood flow and generation of oxygen free radicals. Currently 100% O2 is recommended. Room air (or 30%) may become the preferred initial gas for neonatal resuscitation in the future.

  3- Insert an endotracheal tube and start to push an oxygen through the tube by an ambu bag, if after 15-30 sec of doing that & the baby does not improve: (no spontaneous respiration, heart rate is < 100/min, no improvement in the color of the baby, so the next step is:-

  4- Starting chest compression (cardiac compression to improve circulation) the compression is exerted to the lower third of the sternum at a rate of 120 per min. the ratio of compression to ventilation is 3:1 simultaneously the color, the heart rate the respiration and muscle tone should be assessed, if the baby did not respond after 15-30 sec of chest compression & oxygen supply through an endotracheal tube then:-
An intravenous drugs are used after an insertion of an intravenous (usually umbilical) catheter and as follows:

1. Epinephrine 1/10000 (0.1-0.3) ml/kg IV or intratracheal is given for asystole or for failure to respond to 30 sec of combined resuscitation and the heart rate is < 60/min, this can be repeated every 5 min.

2. Volume expanders 10 - 20 ml/kg of (normal saline, blood, 5% albumine, or ringers solution) should be given for hypovolemia, pallor, E.M dissociation (weak pulses with normal heart rate), history of blood loss, suspicion of septic shock, hypotension or in poor response to resuscitation.

3. Sodium bicarbonate (1-2 meq/kg) should be given slowly in case of metabolic acidosis and resuscitation is prolonged.

4. Calcium gluconate (2-4 ml/kg of 10% solution) if there is evidence of hypocalcemia.

5. Naloxone given in a dose of 0.1 mg/ kg repeated as needed when there is CNS depression due to maternal narcotic analgesic administration during labor which will results in respiratory depression & failure to initiate spontaneous respiration.

6. Dopamine or dobutamine may be given in a dose of 5-20 microgram / kg/ min. this drug may be used in severe asphyxia when there is depressed myocardial function.

**Kernictus**

تفاصيل الموضوع موجودة في محاولة د.ياسمين النظري.

**Sepsis and meningitis**

Check for hyperthermia or hypothermia (hypothermia > hyperthermia), lethargy, hardening of subcutaneous tissue.

Urgently start with empirical antibiotics:

- Either Ampicillin (300 mg/kg) + Gentamicin for 2-3 weeks
- Or 3rd generation cephalosporin (Cefoxime) + gentamicin
- Most common pathogen (Group B streptococcus, E.coli, H.influenzae)
- Neonatal sepsis carries poor prognosis (↑ fatality rate).
**Neonatal hypoglycemia:**
- It is dangerous because it may end with brain damage.
- **Risk factors** are: hyperinsuleniemia as in pt. with Rh incompatibility, premature baby, IUGR, low birth weight, polycythemia.
- **Can be presented with seizure, lethargic, apnea.**
- **Tx:** dextrose 10% by IV infusion avoid > 10% dextrose as it is irritant to vein (thrombophilibitis), if >10% dextrose is needed ➔ use central vein.
- Initially start wit **bolus** dose 200-400 mg/kg
- **Maintenance** 6 mg /kg

**Hemorrhagic disease:**
- Check for petechea, melena, heamtemesis, Caused by clotting factor deficiency (hepatic immaturity, vitamin K deficiency).
- **Vit . K deficiency result from insufficient amount of normal intestinal flora** Patient with breast feeding more liable for Vit . K deficiency.
- **Tx : Vit. K replacement 5-7 unit, 1 unit prophylaxis**
  If there is severe hemorrhage or no response vit. K ➔ give Fresh frozen plasma

**Neonatal seizures:**
- Stop convulsion urgently to avoid cerebral anoxia
- usually occur 12–48hr after delivery.
- Can be generalized or focal, and tonic, clonic, tonic-clonic or myoclonic.
- Startle or Moro reflexes, normal jittery movements (fine, fast limb movements that are abated by holding affected limb), Sleep myoclonus (REM movements).

**Tx :**
- IV phenobarbital (10–20mg/kg bolus; give further 10–15mg if seizures persist after 30min ➔ maintenance dose 5mg/kg/day)

**Non-accidental injury (child abuse):**
- Check for :
  - Delay in seeking medical attention.
  - The details of the mechanism of injury are implausible, different stories with different informants, injury inconsistent with the story mentioned by family.
- Lack of concern by the person accompanying the child.
- Abnormal behavior or demeanor by the child e.g. withdrawn, avoiding eye contact. This should be observed in the context of the child's background – for example it is usual to avoid eye contact in some Pacific cultures.
- Direct disclosure by the child that the injury was deliberately inflicted. Bruises, Thermal burns and multiple fractures shown by X-ray at different stages of healing

**Congenital malformation:**
- **Bilateral choanal atresia:** presented with difficult feeding, cyclical apnea and cyanosis that is relieved once the child cry (become pink and cyanosis disappear) as the neonate is obligatory nasal breather.
  
  Dx ➔ 1. Simply by inserting an NG tube through the anterior nostrils ➔ resistance to flow through posterior choana.
  2. CT-scan.

- **Esophageal atresia.**
- **Neonatal intestinal obstruction:** Cause gangrene ➔ sepsis ➔ death
  - Usually result from duodenal atresia, imperforated anus, volvulus and malrotation.
  
  In upper intestinal obstruction vomiting preceding the constipation in lower intestinal obstruction constipation preceding the vomiting
- **Necrotising enterocolitis:**
- **Cardiac malformation:** TGA, critical coarctation of aorta, hypoplastic right ventricles, tricuspid atresia

  - Presented with early birth cyanosis
  Rx ➔ prostaglandin to maintain duct opening.
- **Renal:**
  - Hypoplastic or agensis of kidney treated by kidney transplantation
  - If urine not pass check for bladder may be due to posterior urethral valve obstruction if the cause in the kidney may be pelvic ureteric junction obstruction, mass (teratoma).
  - **Meningiomyocele**

    More risky if leaking ➔ closed by surgery

**Life threatening birth injury:**
- **By forceps** during delivery cause depressed skull fractures or intra-cerebral hemorrhage, Subgial hemorrhage may result in ➔ hypovolemia
  - Check blood to exclude if there is bleeding tendency
  - Give the child BT or NS or any volume expander
- **Bilateral phrenic nerve palsy:** Transient and patient need assisted ventilation

<table>
<thead>
<tr>
<th>Duct dependent CHD:</th>
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<tbody>
<tr>
<td>1. Pulmonary atresia</td>
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<tr>
<td>2. Severe a stenosis</td>
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<tr>
<td>3. TOF with severe pulmonary stenosis</td>
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<tr>
<td>4. Coarctation of Aorta (severe)</td>
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<tr>
<td>5. Interrupted Aortic Arch</td>
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<tr>
<td>6. Hypoplastic Left Heart</td>
</tr>
</tbody>
</table>
- **Splenic, hepatic injury:**
  - Need urgent correction to stop bleeding
  - Check for ecchymosis, hypovolemia
  - Investigate by US

- **Bilateral femoral fracture**

- **Congenital adrenal hypoplasia**
  - In children with the more severe form of the disorder, symptoms often develop within 2 or 3 weeks after birth.
  - Poor feeding or vomiting
  - Dehydration
  - Electrolyte changes ($\downarrow$Na+, $\uparrow$K+) $\rightarrow$ due to $\downarrow$aldosterone level.
  - Abnormal heart rhythm
  - Girls with the milder form will usually have normal female reproductive organs (ovaries, uterus, and fallopian tubes). They may also have the following changes:
  - Abnormal menstrual periods or failure to menstruate
  - Early appearance of pubic or armpit hair
  - **Excessive hair growth** or facial hair
  - Failure to menstruate
  - Some enlargement of the clitoris

**Exams and Tests**

Your child's health care provider will order certain tests. Common blood tests include:

- Serum electrolytes
- Aldosterone
- Renin
- Cortisol

The goal of treatment is to return hormone levels to normal, or near normal. This is done by taking a form of cortisol, most often hydrocortisone. People may need additional doses of medicine during times of stress, such as severe illness or surgery.
Notes:
- Melena: Shiny black tarry stool
- Viral hepatitis: presented with jaundice, hepatomegaly, abdominal pain and the most important symptom is **loss of appetite**, so try to save the patient with good feeding, well dehydration, encourage **carbohydrate** intake and avoid diet restriction

**Respiratory ER:**
- **Croup**: is not an emergent condition, but need monitoring in the severe cases to resuscitate the patient by endotracheal intubation, or tracheostomy if there is any total upper air way obstruction that may be associated with disturbed level of consciousness
- Use steroid on demand (don’t let the patient die before giving him steroid)
- Steroid can be used until 2 weeks safely (1 week on textbooks) and stoppe without tapering.

**Acute epiglottitis:**
- It is life threatening condition (Total airway obstruction).
- Severe bacterial infection of epiglottis and subepiglottic fold
- Bacteria: Hemophilus influenzae type b, Strep.pyogens
- Features: sudden onset, high fever, toxic, sore throat, dysphagia, tripod position, **drooling of saliva**, dyspnea, collapse, coma, death (in few hours).
- Clinical diagnosis (not use tongue depressor † lead to respiratory obstruction)
- Don’t take history, don’t do x-ray.
- Blood culture investigation is hazardous.
- Do examination in theater room with available tools for intubation, tracheostomy and anesthesia.
- All children need intubation for 2-3 days.
- Antibiotics: for H.influenzae (amoxicillin or ceftriaxone) for 7-10 days
- Then send child to home.
- Give rifampicin to house hold members for 2 days to prevent meningitis due to H.influenzae.

Avoid ceftriaxone in children especially with breast feeding
Procaine pencilllin best drug for H.inf
Chloramphenicol also can be used ( S/E : aplastic anemia )
Some medical schools administer steriod for aryepiglottic fold inflammation

**Foreign body inhalation (aspiration)**
- Common in infants and toddlers
• (Infant can swallow F.B because they explore environment by their mouth).
• Inhale things like: فستق، حب شمس، خرز
• History: very important, healthy baby, sudden onset, parent denies something (social circumstances).
• Cause acute strider.
• First stage: severe paroxysm of cough, cyanosis, chock, sneezing, gagging.
• Second stage: Misleading (like a recovery state).
• Third stage: symptoms of complications because F.B go to the right lung and lead to atelectasis, pneumonia, tachypnea, cyanosis, retractions, fever, and other symptoms.
• Diagnosis clinically
• Investigations: CXR should be done in deep inhalation see localized hyperinflation, most are radio-lucent.
• Treatment: upside down, big thrust on baby back, laryngoscope or bronchoscope, tracheostomy (trans-thoracic approach).

In presistent pneumonia think about FB inhalation
Or H-type TEF fistula in slowly resolving pneumonia

Penmonia: The commonest bacteria in pneumonia in all age groups are strepto pneumoniae, H.influenzae.

• Neonate (less than 3 or 4 weeks): group B strepto, E.coli, G+ bacillus. Pneumonia in neonate is like septicemia: give parenteral antibiotics for two weeks then admission.
• After neonate: viral infection.
• After 3 months: chlamydia, uroplasma, mycoplasma.
• After age of 5 years: most common is strepto pneumonia.

Diagnosis by CXR opacity, patchy infiltrate (viral), lobar infiltrate (viral).

• Staph. pneumonia:
  o High fever, toxic, dramatic and progressive course.
  o CXR very characteristic: lung abscess, empyema, plural effusion, pneumatocele.
  o Come with septicemia and coma.
  o Blood culture (+ve in 10% only)
  o Give anti-staph drugs. (vancomycin, gentamicin, fluxacilliin) with supportive treatment, IV fluid, monitoring.

• bronchiactasis beast treated by physiotherapy and antibiotics
• For mycoplasma pneumoniae azithromycin or clarithromycin.
• For pneumococcus amoxicillin for 7-10 days (40-100 mg/kg in day)

**Indication for hospital admission:**

- Need O2.
- Less than 6 months age.
- Need fluid and supplements.
- Immuno-deficient baby.
- Slowly resolving pneumonia.
- Multiple infections. umoniae then mycoplasma pneumonia.
**Status asthmaticus**

Chest X-ray (CXR):
- Is indicated in:
  1. First attack to exclude other DDx (no need to repeat CXR in the other attack).
  2. If we treating the patient with his good compliance but the patient condition still NOT stable, perform CXR to diagnose complications or to exclude other Diseases.
- Findings like pneumothorax, atelectasis, mediastinum widening
- Peak expiratory flow (PEF):
  - Very important but is indicated in children who are 6 years old age or

**Short acting B agonist (SABA).**
- Inhaled Side effects less than oral one.
- Side effects like tachycardia, hyperkalemia, tremor.
- It is bronchodilator.
- Like salbutamol, albuterol.
- 0.5 ml \ for less than 5 years // 1 ml \ for more than 5 years ((only ml, not ml/kg)
- Very effective.
- Give it with 2 ml of normal saline \ use nebulizer.
- Oral is as effective as parenteral.

**Treatment:**
- Admission to ICU
- Monitoring
- Two rescue treatment
  - Inhaled and systemic corticosteroids
  - aminophylline infusion
  - Mg sulfate (IV 75 mg/kg) \ ipratropium bromide
  - terbutaline
  - Adrenaline (0.01 mg/kg) SC or IM (very painful)
- Ventilator
  - No need for \ Oral beta 2 agonist / Ketotifen (anti-histamine) / Antibiotics /Oral bronchodilators (side effects).

**Aminophylline:**
- Give it by infusion
- The bolus dose 5 mg/kg (slowely)
- Maintenance 0.7-1 mg/kg by infusion pump
- Don’t give aminophylline in supine position, give it in lying postions
- S/E: seizure, arrhythmia, vomiting, hypotension
-Social management with action plan (how they use the spacer, allergen avoiding, sign of severity)
-Spirometry not used in children < 7 years not – cooperative

**Bronchiolitis:**
- Common wheezy infection
- Occur in few months up to 2 years
- Above 5 years rare.
- Viral infection (RSV)
- Rarely mycoplasma pneumoniae.
- More in boys
- Breast feeding is protective.
- **Neonate (1 month) rarely have bronchiolitis and rarely have viral infection**
- Diagnosis clinically
  - Features: rhinorrhea, cough, sneezing, common cold, low grade fever, respiratory distress, cyanosis, tachypnea (120/min), wheezing, flaring ala nasi, recession, tired, hyperinflated chest, air trapping, auscultation (wheezing, fine bilateral crackles), may feel liver and spleen (due to hyperinflation), poor appetite, refuse eating.
- Not diagnose H.F with radiological evidence of cardiomegaly.
- CXR: Flat diaphragm, narrow mediastinum.

**Clinical cases notes:**
-In convulsion exclude meningitis
-1st attack of convulsion + fever, < 18 months
-Lumbar puncture is mandatory because of meningitis suspicion in this age and its signs not specific.
-If CSF in lumbar puncture is turbid by eye and exit under high pressure give him intensive antibiotics
-Don’t say febrile convulsion if the baby is < 6 months, check for electrolyte disturbance, hypoglycemia or idiopathic convulsion.

**Case of nephrotic syndrome:**
-Edema, scrotal edema, eye puffiness
-Check for BP always in Cardiac, CNS, Renal
-Rx: steriod dependent or resistant, free sloute albumin, diuretics, K replacement
**Diarrhea:** increase frequency, consistency and liquidity of stool that is observed by the mother.

**Diarrhea (WHO):** > 3 bowel motions/day (except for babies who are exclusively abreast fed).

**Approach to acute diarrhea: <14 days.**

- **History:** Same history but should include (thirst and UOP) to estimate the severity of dehydration.
  - Feeding Hx: should include:
    - Type of feeding: breast, Bottle or mixed.
    - If Bottle feeding:
      - sterilization method (boiling for 10 min.)
      - preparation of milk.
      - No. o bottles (should be= No. of feds +1)
      - Type of milk.
- **Examination:** we have to look for signs of dehydration:
  1. Ant. Fontanelles → should be: At sitting position + baby not crying (مهم).
  2. Sunken eyes.
  4. Skin Turgor → should be <2 seconds, if >2 seconds → dehydration.
  5. UOP.
- **Investigations:**
  1. Serum electrolytes:
  2. RBS (random blood sugar).
  4. CBC: -RBC and HB: Normal or ↓
  5. -shift to left {e.g: neutrophia +band cells (bandemia)} → suggest shigellosis.
  6. -PCV {normal or ↑(hemoconsitration)}
  7. -platelet: normal or ↓ (in case of HUS)

**HUS:** is a triad of:
1. Microangiopathic anemia (helmet and fragmented RBC in blood film).
2. Thrombocytopenia.
3. Uremia.

Note: presence of 2 out 3 + predisposing factors can make the Dx.
5. General stool exam: Microscopic, chemical and microscopic exam:

✓ Macroscopic: volume, consistency, color ... etc
✓ Chemical: -PH →
  - Normal PH is alkaline (because of HCO₃⁻)
  → Whenever it's acidic → considered abnormal (e.g. lactose intolerance) except in breast fed baby.
  - Reducing agents: if +ve → (e.g. lactose intolerance)
  - Occult blood.
✓ Microscopic:
  1. RBC
  2. Pus cells
  3. Fatty cells
  4. Cysts or trophozoits.

DDx:
1. Infection: Bacterial (shigella, salmonella, yersenia, capelobacter...etc) or protozoal.
2. Cow's milk allergy.
3. IBD.
4. Post antibiotic psuedomembranous colitis.

Approach to a pt. with chronic diarrhea: >14 days

We can classify chronic diarrhea into 3 types:

1) Chronic diarrhea without FTT.
2) Chronic diarrhea with FTT.
3) Chronic diarrhea with blood.

1) DDx of Chronic diarrhea without FTT:
- Lactose intolerance.
- Giardiasis.
- Toddler’s diarrhea.

NOTES:

CASE1: Pt. with diarrhea get improving → develops abdominal distention → what is the Dx? And how to prove it?
A/- Dx: paralytic ileus from hypokalemia.
  - Proof: by
    ## Auscultation → sluggish bowel sounds
    ## Serum K⁺ (If there’s no facility to do serum K⁺) then we can do:
    ## ECG (flattening of T wave, prolonged QT interval)

CASE2: Pt with chronic diarrhea → develops bloody diarrhea → what is the Dx?
A/ Vit. K deficiency, because of antibiotic use → Kills bact. Flora which can produce Vit. K

Color of stool:
- Red → blood
- Non-bilious → Cholera, obst. Jaundice.
- Green → rapid transient time (benign)
**Lactose intolerance:**

- **Types:**
  1. Primary.
  2. Developmental (very rare) → temporary → eg. Prematurity.
  3. Secondary to (very common): celiac dis., Rota virus inf., cow’s milk allergy...etc

- **How to Dx:**
  - **Clinical:** diarrhea, abd. pain & distension, exaggerated bowel sounds and perianal excoriation.
  - **PH** of stool: ↓
  - **Reducing substances:** +ve
  - **Jejunal biopsy:** → Histochemical study → reduced lactose in brush border.
  - **Lactose tolerance test:** flat (no ↑ in glucose after CHO meal).
  - **H’ breath test:** ↑ (as a result of bacterial fermentation of undigested lactose)

- **Rx:** Lactose free formula (LF) → Isomil
  Soy milk (isomil) → for 2-4 wks then re-introduce into ordinary formula.

---

**Giardiasis:**

- **How to Dx:**
  1. Finding trophozoites (not cyst) in FRESH stool: (25% +ve).
  2. Dudenal aspirate: (75% +ve).
  3. Metronidazol trial.

- **Rx:** 20-30 mg/kg metronidazole for 10 days.

---

**Toddler’s diarrhea:**

- **How to Dx:** by exclusion of other causes of chronic diarrhea. (Correlate to IBS in adults.)

---

**Isomil used only in:**
1. Galactossemia.
2. Lactose intolerance.
3. Cow’s milk allergy

---

**Q/What is the difference in metronidazol use in amebiasis and Giardiasis?**

**A/ in amebiasis:**
50 mg/kg metronidazole for 2 wks in divided dose to be followed by diloxanide furoate or paromomycin to get rid of cysts in order to prevent relapses
2) **DDx** of Chronic diarrhea with FTT:

- Lactose intolerance.
- Giardiasis.
- Celiac Dis.
- Cystic fibrosis.
- Bact. Overgrowth.
- UTI.
- Endocrinopathies → thyrotoxicosis, Addison’s dis., VIPomas
- Cow’s milk allergy.

Can cause chronic diarrhea WITH or WITHOUT FTT

---

**Celiac Dis.:**

- **Presentation:** Abd. pain & distension, chronic diarrhea (large, greasy, offensive) poor weight gain, anorexia, vomiting ...etc .
- **How to Dx:**
  1. **Serology:** anti-tissue transglutaminase IgA ab and total IgA.
      
      If anti-tissue transglutaminase Ab is ↑ and total IgA is normal then we have to do:
  2. **Dudenal or jejuna biopsy:** Histopathological findings:
      - Total or subtotal villous atrophy.
      - Deepening of the crypts of langerhans (cryptitis)
      - Intraepithelial and submucosal infiltration of lymphocytes and plasma cells.

      ## If the above findings was found + serology ↑ titer → definitive Dx.

- **Rx:** Gluten-free diet for life long.

---

**NOTE:** celiac dis. Is never diagnosed <9 months age.

**Celiac syndrome:** diseases similar to celiac dis. But it’s not celiac dis.:
- Tropical sprue.
- Giardiasis
- Malnutrition.
- Eosinophilic enteritis.

**Note:** Malignancy (lymphoma and pancreatic CA) is a possible complication of celiac dis.

**Note:** Gluten-free diet dose not eliminate the risk of malignancy.
**Cystic fibrosis:**

- **How to Dx:**
  1. It can be diagnosed 1 hr. after birth because it can cause meconium ileus.
  2. Sweet chloride test \( \rightarrow > 60 \text{ mmol/L} \)
  3. DNA diagnosis.

- **Can cause bronchiectasis as result of recurrent chest infection \( \rightarrow \) can cause death.**

- **Rx:** Antibiotics + physiotherapy.

---

**UTI: Very important \( \rightarrow \) DDx of many complaints.**

- **How to Dx:**
  1. GUE \( \rightarrow > 5 \text{ pus cells/HPF} \)
  2. Urine culture (main diagnostic method) : specimen:
     - Mid-steam urine :
       - \( \#\# > 100,000 \text{ colony/ml of the same M.O} \rightarrow \text{UTI} \)
       - \( \#\# 10,000 \text{ colony/ml} \rightarrow \text{NOT UTI} \).
       - \( \#\# 10,000 - 100,000 \rightarrow \text{Suspicious} \rightarrow \text{Repeat} \) the test or do more accurate method
     - Suprapubic aspiration \( \rightarrow \) if single colony is recovered \( \rightarrow \) definitive Dx of UTI.
     - Or - Urine obtained by foly’s catheter \( \rightarrow \) If single colony is recovered \( \rightarrow \) UTI.
  3. U/S
  4. MCUG

- **If the above mentioned investigations are:**
  - Norml \( \rightarrow \) No need for IVU.
  - Abnormal \( \rightarrow \) we have to do IVU to estimate the remaining functioning part of the kidney \( \rightarrow \) If the kidney is still functioning \( \rightarrow \) it’s worth to do surgery for VUR dis. 
    \( \rightarrow \) If the kidney is scarred \( \rightarrow \) No need to do the corrective surgery.
✓ **Rx:** *cystitis* → symptomatic Rx + Antibiotic (amoxicillin or 3rd generation cephalosporin as cefixime) orally for 7-10 days.

Pyelonephritis → Hospitalization + symptomatic Rx + Antibiotic (combination of 3rd generation cephalosporin + aminoglycoside) parenterally for 10-14 days to be switched into oral therapy once the pt. is able to.

**Cow’s milk protein allergy: (Casein protein):**

✓ **Presentation:** bloody diarrhea, abdominal pain, urticaria, runny nose and even FTT.

✓ **How to Dx:**
   2. Specific Ab

✓ **Rx:** Extensively hydrolyzed formula.

3) **DDx** of Chronic diarrhea with Blood:

Post-antibiotic pseudomembranous colitis.

---

General notes:

- Palpitation: never mention in the history of child (<3 years) who are not old enough to express such a feeling. → "palpitation " because it is subjective (it is feeling by the patient him/herself)
- Feeding Hx:
  - Ask about any new introduction of *unmodified cow’s milk?*
- **Convlusion** is pyrmidal tract in origin, while occulogyric crisis is extra-pyramidal origin.
- **Child with no symptoms, no murmurs upon auscultation and normal ECG and CXR (no cardiomegaly) → unlikely to have cardiac problem (CHD) → NO matter to do ECHO.**
**Chyne-stoke breathing**: special breathing pattern characterized by tachypnea that is slowly come down followed by a period of apnea then tachypnea progress again.

- This is normal condition in premature babies in first few weeks of life and even in healthy full term babies, usually after sleeping deeply.
- This is abnormal in adults → indicating severe brain damage, renal failure.

Home care: supine position, avoid soft pillows and smoking, never shake baby to breath as it may cause brain injury.

**CVS notes**

**Apex beat:**
- Apex beat → outermost, lowermost visible or palpable to the left or right
- If you don’t find the pulsation; look at the axilla (left side), if you still don’t find the pulsation; see the right side (dextrocardia)

**Regions in auscultation:**
- Mitral (Apex) area : 4th left ICS at mid-clavicular line or 5th ICS in older child.
- Aortic area : 2nd ICS right to the sternum
- Pulmonary area : 2nd ICS left to the sternum
- Tricuspid area : left sternal border in 4th ICS or 5th ICS in older child

**Coarctation of aorta is characterised by the following features**
- Usually seen in male patient and they presents with headache, claudication, palpitation, anginal pain or cold extremities.
- The upper extremity and thorax may be more developed compared to lower extremities.
- Radiofemoral delay is present.
- All the peripheral pulses should be examined carefully
- Prominent suprasternal and carotid pulsations are present
- Dilated pulsating collaterals especially intercostal arteries which can be seen in the inter-scapular region posteriorly (Suzman sign), and is best elicited with patient bending forward with arms hanging by the side of the body.
- Systemic hypertension.
- Bruit over the collaterals.
- Left ventricular type of cardiac enlargement and heaving apex is seen.
- A systolic murmur may be heard over the anterior chest and back.

Continuous murmur is heard over the collaterals.

<table>
<thead>
<tr>
<th>Causes of absent apex beat:</th>
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<tbody>
<tr>
<td>1. obesity.</td>
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<td>2. Thick chest wall</td>
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<tr>
<td>3. Pericardial effusion</td>
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<tr>
<td>4. Dextrocardia</td>
</tr>
</tbody>
</table>
**Clinical association of coarctation:**
- Bicuspid aortic valve
- Turner’s syndrome
- Berry aneurysm
- Polycystic kidney

**Radio radial delay:**
To detect the radioradial delay you should simultaneously palpate both the radial arteries by both your hands, using your left hand for patient’s right radial artery and vice versa.
Normal situation radial and femoral pulsations are felt equally and synchronously. The inequality between two radial pulses is known as **Radio radial delay**. The delay between the radial pulse and femoral pulse is called as **Radiofemoral delay**.

**Dorsalis pedis exam:**
The pulse of the dorsalis pedis artery, palpable at the prominent arch of the top of the foot between the first and second metatarsal bones. It can be felt in approximately 90% of people.
Blood pressure measurement:
Measurement of blood pressure

Flush method:
Flush technique for obtaining pediatric BP:
- Used in infants or very young children where lack of cooperation precludes use of auscultation and palpation to determine BP
- The flush technique allows for a value lying between the systolic and diastolic to be determined
- With the cuff in place, an elastic bandage is wrapped around the elevated arm
- Proceeding from the fingers to the antecubital space emptying of the capillaries and venous network occurs
- The cuff is now inflated to a pressure above the expected systolic reading
- The bandage is removed
- The now pallid arm is placed at the patient’s side
- The pressure is allowed to fall slowly until the sudden flush of normal colour returns to forearm, hand and fingers.
- The endpoint is strikingly clear
- This method may also be used on the thigh
First check for brachial blood pressure it is audible or not by the stethoscope then checks for the radial pulse it is palpable or not if neither brachial or radial pulse felt now you can change to the Flush method.

**Pulsus deficit:**
a condition in which a peripheral pulse rate is less than the ventricular contraction rate as auscultated at the apex of the heart or seen on the electrocardiogram, indicating a lack of peripheral perfusion.
pulse deficit the difference between the apical pulse and the radial pulse, obtained by having one person count the apical pulse as heard through a stethoscope over the heart and a second person count the radial pulse at the same time.
Exam the genitalia:
Check for any ambiguous genitalia (congenital adrenal hyperplasia → ↓Na⁺), imperforated anus

**Exam the back**
Exam the skin: for any neurocutaneous disease; eg: neurofibromatosis
Tuberus sclerosis

**Rickets: Vitamin D deficiency rickets.**

*How to Dx:* by X-ray and laboratory tests

*X-ray:*
- Widening and cupping of the metaphysis→ best seen at wrist or ankle.
- Fraying of metaphysis.
- **Bowling** of long bones
- Development of knock-knees, or genu valgum.

**LAB findings:**

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Ca</th>
<th>Pi</th>
<th>PTH</th>
<th>Ca(25-OH)D</th>
<th>ALK PHOS</th>
<th>URINE Ca</th>
<th>URINE Pi</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin D deficiency</td>
<td>N</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
<td>↓</td>
<td>▼</td>
</tr>
</tbody>
</table>

Early on in the course of rickets, the calcium (ionized fraction) is low. However, this level is often within the reference range at the time of diagnosis, as a consequence of increased parathyroid hormone secretion. Although calcidiol (25-hydroxy vitamin D) is low and parathyroid hormone is elevated, determining calcidiol and parathyroid hormone levels is typically not necessary in order to establish a diagnosis. Calcitriol levels maybe normal or elevated because of increased parathyroid activity.

The phosphorus level is invariably low for age, unless recent partial treatment or recent exposure to sunlight has occurred. **Alkaline phosphatase levels are uniformly elevated.**

A generalized aminoaciduria occurs from the parathyroid activity. However, aminoaciduria does not occur in familial hypophosphatemia rickets (FHR).
Treatment

Children with nutritional vitamin D deficiency should receive vitamin D and adequate nutritional intake of calcium and phosphorus. There are 2 strategies for administration of vitamin D. With stoss therapy, 300,000-600,000 IU of vitamin D are administered orally or intramuscularly as 2-4 doses over 1 day. Because the doses are observed, stoss therapy is ideal in situations where adherence to therapy is questionable. The alternative is daily, high-dose vitamin D, with doses ranging from 2,000-5,000 IU/day over 4-6 wk. Either strategy should be followed by daily vitamin D intake of 400 IU/day if 1 yr, typically given as a multivitamin.

Most sensitive alkaline phosphatase enzyme the first one is affected
And the last improved if it is become normal after treatment
So rickets due to vit.d deficiency if not return to normal
Rickets may be due to:
1-vit.D resistant
2-congenital hypophosphatemia
3-renal tubular acidosis

The marker of response to Rx is the reduction in the level of Alkaline phosphatase enzyme.

SHORT STATURE:

#Constitutional growth delay: This condition describes children who are small for their ages but who are growing at a normal rate. They usually have a delayed "bone age," which means that their skeletal maturation (bone age) is younger than their age (chronological) in years. (Bone age is measured by taking an X-ray of the hand and wrist and comparing it with standard X-ray findings seen in kids the same age).
These children don't have any signs or symptoms of diseases that affect growth. They tend to reach puberty later than their peers do, with delay in the onset of sexual development and the pubertal growth spurt. But because they continue to grow until an older age, they tend to catch up to their peers when they reach adult height. One or both parents or other close relatives often had a similar "late-bloomer" growth pattern.

#Familial (or genetic) short stature: This is a condition in which shorter parents tend to have shorter children. This term applies to short children who don't have any symptoms of diseases that affect their growth. Kids with familial short stature still have growth spurts and enter puberty at normal ages, but they usually will only reach a height similar to that of their parents. With both constitutional growth delay and familial short stature, kids and families need to be reassured that the child does not have a disease or medical condition that poses a threat to health or that requires treatment. However, because they may be short or may not enter puberty when their classmates do, some may need extra help coping with teasing or reassurance that they will go through full sexual development eventually. In a few children who are very short or very late entering puberty, hormone treatment may be helpful. Diseases of the kidneys, heart, gastrointestinal tract, lungs, bones, or other body systems might affect growth. Other symptoms or physical signs in kids with these illnesses usually give clues as to the disease causing the growth delay. However, poor growth can be the first sign of a problem in some.

Growth disorders include:
Failure to thrive: which isn't a specific growth disorder itself, but can be a sign of an underlying condition causing growth problems. Although it's common for newborns to lose a little weight in the first few days, failure to thrive is a condition in which some infants continue to show slower-than-expected weight gain and growth. Usually caused by inadequate nutrition or a feeding problem, it's most common in kids younger than age 3. It may also be a symptom of another problem, such as an infection, a digestive problem, or child neglect or abuse.
Endocrine diseases: (diseases involving hormones, the chemical messengers of the body) involve a deficiency or excess of hormones and can be responsible for growth failure during childhood and adolescence. Growth hormone deficiency is a disorder that involves the pituitary gland (the small gland at the base of the brain that secretes several hormones,
including growth hormone). A damaged or malfunctioning pituitary gland may not produce enough hormones for normal growth.

**Hypothyroidism** is a condition in which the thyroid gland fails to make enough thyroid hormone, which is essential for normal bone growth.

**Occipitofrontal circumference of the head:**

<table>
<thead>
<tr>
<th>Age</th>
<th>Rate of ↑ of OFC in Cm</th>
<th>OFC(in Cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>At birth</td>
<td></td>
<td>35</td>
</tr>
<tr>
<td>2 months</td>
<td>4</td>
<td>39</td>
</tr>
<tr>
<td>4 months</td>
<td>3</td>
<td>42</td>
</tr>
<tr>
<td>6 months</td>
<td>2</td>
<td>44</td>
</tr>
<tr>
<td>8 months</td>
<td>1</td>
<td>45</td>
</tr>
<tr>
<td>10 months</td>
<td>1</td>
<td>46</td>
</tr>
<tr>
<td>12 months</td>
<td>1</td>
<td>47</td>
</tr>
<tr>
<td>2 year</td>
<td>2.5</td>
<td>49.5</td>
</tr>
<tr>
<td>(whole 2nd year)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3-7 year</td>
<td>1/2</td>
<td></td>
</tr>
<tr>
<td>(each year)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8-12 year</td>
<td>1/3</td>
<td></td>
</tr>
<tr>
<td>(each year)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Hypocalcemia caused by SOB:
Hyperventilation syndrome often occurs under stressful conditions which cause hypocapnia and consequently results in respiratory alkalosis and a wide range of somatic symptoms. Respiratory alkalosis can induce secondary hypocalcaemia (shift of ionized Ca\(^{++}\) into bound Ca\(^{++}\)) that may cause cardiac arrhythmias, conduction abnormalities and various somatic symptoms such as paraesthesia, hyperreflexia, convulsive disorders, muscle spasm and tetany. Acute hypocalcaemia is an emergency that requires prompt attention and management.

#NOTE:
Acidosis causes the reverse of alkalosis (i.e. causing shift of protein bound calcium into ionized ca\(^{++}\)), that is why the pt. with renal failure (acisdotic) does not develop tetany despite the hypocacemia that he has (as result of failure of renal activation of vit. D into active vit. D3).

Difference between modified and unmodified cow’s milk?

- **Unmodified cow’s milk:**
  - Contain protein 4% (human milk 1%) so it exhaust the liver and kidney during metabolism and excretion.
  - ↑↑ Na\(^{+}\) than breast milk → hypernatremic dehydration.
  - ↑ phosphorus → chelate calcium → lead to convulsion.
  - Modified but modified cow’s milk suitable from birth.
  - Water, solids, calories, fat → same level in human and cow milk.
  - Modified cow’s milk is cow milk with less protein and sodium and phosphorus but contains ↑ fat and CHO.
  - Modified and fortified milk is modified milk with vitamins and minerals.

#NOTE:
Modified cow’s milk contains proteins (energy) < cow’s milk but CHO and oils are higher, so the calorie content is equal to cow’s milk and breast milk which 20 Kcal/oz.
Approach to examine a pt with neurological problem?
Start from the head:
1-hair texture: shiny brittle hair easily broken in FTT, sparse with coarse facies in hypothyroidism.
2-fontanelles:-examine the pt in semi-sitting position (45 degrees),avoid supine position as this will depress the fontanelle
While sitting position will bulge the fontanelle so also avoid it.

- DDx of delayed closure of fontanelle?
  1)ricket
  2)hydrocphalus
  3)osteogenesis imperfect
  4)metabolic disease
  5)hypothyroidism.

- 3-eye :nystagmus,squint,upright slanting (as in Down syndrome).
- 4-nose :-shape of nose ,saddle nose in syphilis/ Hypertelorism.
- 5-Chin: Micrognathia
- 6-mouth:-dental carie , state of hydration, tongue size .
  **protruded but not macroglossen tongue in Down syndrome
- 7-Neck → swelling in midline for goiter.
- 8-Hand:-fingers & hand creases.
  9-foot ,legs for peripheral edema ,sandle gap in down syndrome.
  10-peripheral pulses
  11-LAP
  12-vital signs
  13-growth parameters.
**Growth parameters :-**

**Head circumference :-**
- 3 times attempts & take the highest.
- Normal ranges:
  - At 1st (3)mo → increased by 2cm
  - At 2nd (3)mo → increased by 1cm.
  - At (6)mo & later on → 1/2cm
  - After 1yr → 1cm/yr.

The increment stops when we stop growth.

**LENGTH**
- Till 2 yrs we use the term (length) → Then after use the term (height).

**BP :** Start to be measured at (3) yrs & later but with indications:
- Cvs
- Renal problem
- Umbilical problem

**Use WHO chart for BP**

**In child with convulsion → CNS exam :-**

1) general look for → nutrition, well or sick? conscious? alert? oriented?, posture, any deformity, any abnormal movement, tremor, any scar over legs, wasting, hypertrophy, irritability.
2) Tone exam
3) Power exam
4) reflexes
5) Coordination (Heel shin test, finger nose test)

**speech (dysarthria), nystagmus, dysdiadichokinesia, ataxic gait → cerebellar problem.**
In ER, how do you treat pt with convulsion?

- ABC-put pt in recovery position (lateral position) /airway in mouth for avoiding tongue biting/suctioning of mouth.

- Diazepam IV/rectally very slowly with O2.
  
  -(ampoule contain 10mg/2cc), we give (1) mg for each (3)kg or (4)kg.
  
  --Every (1)cc=(5)mg.
  
  --We use insulin syringe
  
  --It is graduated into (100) ,, divide 100 by (5) =(20) units in insulin syringe =(1)mg.

Fill the syringe fully with D.W.

--Give it over(7)min to (10) min (very slowly).

--Wait for (20) min for response

--the microdrip is used for dilution & to control pediatric age group to avoid overload.

--In microdrip, each (1)ml gives(15) drops.

- If NO RESPONSE

- Give 20mg/kg luminal (luminal in ampoul is "200"mg/kg) then wait (20)min,

  NO RESPONSE→ increase the dose by 5, then 10 min after→ increase by 5, till you reach to 40→ no response→ give both diazepam + Phenobarbital.

- If you prescribe AED→ allow pt to take them for 2yrs if no response then change.

ملاحظة:
- تسلسل الأدوية المذكور ليس كما يذكره الكتاب ، لكن دكتورة رنا ذكرته حسب تفريض الأدوية في الطوارئ.
- التسلسل كما يشرح في الكتب موجود في مصنف دكتورة رونة ص 5.
Case of Epilepsy

C.C: abnormal involuntary body movement for 10 days duration.

H.P.I:-

(9mo) old baby, known case of epilepsy on treatment, his condition started as continuous fever, for (2) days duration not associated with sweating or rigor, then suddenly abnormal involuntary body movement for (10) min duration occurred with generalized, spastic posturing.

It was associated with salivation & bluish discoloration of mouth, not passed motion or urine, aggravated by fever with no Hx of trauma, the pt immediately was brought to hospital after admission & receiving Rx, he had developed vomiting & FBM, (6) times/day, watery in nature, no blood or muscus in stool, convulsion attacks continued at hospital although Rx is continuous.

Electrolytes & blood sugar with GUE were performed, MRI has been given as appointment but has NOT been done till now.

Diazepam was given on admission, the pt was on Phenobarbital then converted to sodium valproate before (2) mo.

Review of system:- nothing significant.

Prenatal Hx

Prenatal:- No vaccination, no smoking, mother age 39yrs, not anemic, bad ANC.

Natal: - primigravida, no fever, No APH, No G.D, No Hypertension, no preclampsia, NVD, No PPH, slight leaking liquor, no abortion.

Postnatal: - cyanosed, 2 days in NICU, wt: 3.5 kg, No jaundice, no crying at birth.

Developmental Hx:-

Gross motor: - no sitting, no crawling, no rolling over, he controlled his head at 4th month.

Fine motor: - palmar grasp, not able to reach the object to mouth, not able to transfer objects from one hand to another.
Social :-smile to his mother at 2\textsuperscript{nd} month.

Language & speech:- only pronounce some incomprehensible sounds, not able to say mamma, baba.

Hearing&vision:- no response to his name.

Immunization:- upto date.

Family Hx:- no member in the family with febrile convulsion or epilepsy/no Hx of chronic illnesses

Social Hx:- using tap water, no travel to other areas or countries, no animals in the house, average outcome.
Werdnig-hoffmann disease (SMD):

- Progressive degeneration of AHC (anterior motor horn cell).
- 3 types (early type WHD), (late type Kugelberg-Welander syndrome), intermediate type).
- Autosomal recessive.
- WHD: start as progressive proximal weakness, ↓ spontaneous movement, floppiness, atrophy of muscles, loss of head control, drooling, ↓ facial expression, loss of reflex, eyes remain bright open, engaging, tongue fasciculation (sleep) normal mentality, language, sensation
- Cause of death: respiratory infection, respiratory failure.
- Diagnosis: CPK ↑, EMG.
- TREATMENT: CONSERVATIVE

Hypotonia occur in:

1. Surgical cut of nerve
2. Diabetes mellitus
3. B complex deficiency
4. Myaesthania gravis

In the first week hypotonia is unusual

In case of cerebral palsy hypotonia can occur at first early life and then changed to either spastic paralysis or still in hypotonic pattern
The death is due to respiratory failure → infantile death All lesion result in hypotonia + hyporeflexia.

In case of UMNL it result in hypotonia + hyporeflexia

On Exam:

- **hypotonia**: can be found by:
  1. ventral suspension
  2. Scarf Sign

**Scarf Sign**

- This maneuver tests the passive tone of the flexors about the shoulder girdle.
- With the infant lying supine, the examiner adjusts the infant’s head to the midline and supports the infant’s hand across the upper chest with one hand. The thumb of the examiner’s other hand is placed on the infant’s elbow.
- The examiner nudges the elbow across the chest, felling for passive flexion or resistance to extension of posterior shoulder girdle flexor muscles.
- The point on the chest to which the elbow moves easily prior to significant resistance is noted. Landmarks noted in order of increasing maturity are: full scarf at the level of the neck (-1); contralateral axillary line (0); contralateral nipple line (1); xyphoid process (2); ipsilateral nipple line (3); and ipsilateral axillary line (4).
3. Both Elbow meet on the back
4. Heel reach and touch the ear lobule (Heel – Ear test)
5. Sun set sign during hands rising up

Prognosis: it is a lethal disease
25% of siblings are affected, 50% carrier, 25% normal
Advice family to avoid consanguineous marriage

Notes:
Simian creases is a normal finding

# Heat exhaustion

Heat exhaustion is a heat-related illness that can occur after you've been exposed to high temperatures, and it often is accompanied by dehydration.

There are two types of heat exhaustion:

- **Water depletion.** Signs include excessive thirst, weakness, headache, and loss of consciousness.
- **Salt depletion.** Signs include nausea and vomiting, muscle cramps, and dizziness.

Although heat exhaustion isn't as serious as heat stroke, it isn't something to be taken lightly. Without proper intervention, heat exhaustion can progress to heat stroke, which can damage the brain and other vital organs, and even cause death.

Symptoms of Heat Exhaustion

The most common signs and symptoms of heat exhaustion include:

- Confusion
- Dark-colored urine (a sign of dehydration)
- Dizziness
- Fainting
- Fatigue
- Headache
- Muscle or abdominal cramps
- Nausea, vomiting, or diarrhea
- Pale skin
- Profuse sweating
- Rapid heartbeat

Treatment for Heat Exhaustion
If you, or anyone else, has symptoms of heat exhaustion, it's essential to immediately get out of the heat and rest, preferably in an air-conditioned room. If you can't get inside, try to find the nearest cool and shady place.

Other recommended strategies include:

- Drink plenty of fluid (avoid caffeine and alcohol).
- Remove any tight or unnecessary clothing.
- Take a cool shower, bath, or sponge bath.
- Apply other cooling measures such as fans or ice towels.

If such measures fail to provide relief within 15 minutes, seek emergency medical help, because untreated heat exhaustion can progress to heat stroke.

After you've recovered from heat exhaustion, you'll probably be more sensitive to high temperatures during the following week. So it's best to avoid hot weather and heavy exercise until your doctor tells you that it's safe to resume your normal activities.

Notes on general exam:

Check for:

- Position
- Consciousness
- Respiratory condition
- Color:
  1. Pale: check the eye and palm
  2. Cyanosis: central and peripheral, clubbing, heart
  3. Rash: distribution, nature of rash
  4. Jaundice
- Reaction to examiner
- State of hydration
- State of nutrition
- Vital sign

Cervical and inguinal LN is recurrently enlarged in the infancy
Due to infection

Axillary LN is impt. During exam if there is any LAP, sometimes is enlarged due to BCG vaccine
Notes:

Don’t mix egg with iron it will be non-absorbable in the intestine
Milk + iron = non-absorbable complex

Green leafy vegetable contain iron but is non-absorbable because it is ferric

<table>
<thead>
<tr>
<th>Iron Requirements</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-6 months</td>
</tr>
<tr>
<td>7-12 months</td>
</tr>
<tr>
<td>1-3 years</td>
</tr>
<tr>
<td>4-8 years</td>
</tr>
</tbody>
</table>

Physiological needs of iron:

On Exam check for nutritional status by:
Fat and carbohydrate → check for wasting
Protein → check for edema
Mineral and vitamins → check for rickets
Water → check for dehydration

Anemia: Normal retic count is 0.2-2% when increase indicates BM compensation

Differential diagnosis of Anemia:
1. iron def. Anemia → (serum ferritin test)
2. Thalassemias → Hemoglobin electrophoresis with quantitative hemoglobin A2 and hemoglobin F
3. sideroblastic anemia →
4. lead poisoning → porphyrin in urine test
5. anemia of chronic disease
6. Hodgkin dis → take biobsy
7. Atransferrin anemia (congenital loss of iron binding protein)

Ancylostoma Duodenale:

If patient has iron def anemia and not respond to Tx check
Dx: stool microscopy looking for D-shape ova
Rx: Albendazol
Notes:

In SOB ask about:
- fever
- previous attack of SOB
- Cough at day or at night (nocturnal)?
- Cough with on-off nature?

In systematic review ask about:
1-GIT : vomiting , diarrhehea
2-UT : check for urine output

Indication of admission in SOB:
- Less than 6 months age.
- Severe respiratory distress.
- Need O2.
- Baby with high risk factors \( \rightarrow \) CHD, chronic lung diseases (broncho-pulmonary dysplasia), immune deficiency, and neuromuscular weakness.
- Home condition is bad.
- Carless parents.
  During admission try to identify any life-threatening factors
  If there is cyanosis\( \rightarrow \) give O\(_2\) or even mechanical ventilation
  If there is dehydration \( \rightarrow \) IV fluid
  Foreign body \( \rightarrow \) bronchoscopy
  Fever \( \rightarrow \) try to decrease body temperature
  Pneumothorax \( \rightarrow \) chest tube

Notes on general exam:
- Look for mouth or lips color if it is blue may be due to central cyanosis or gentian violet pigments it is a very effective anti-fungal
- using an accessory muscle eg; sternocleidomastoid muscle is called (head nodding), using suprasternal muscle indicate upper respiratory tract obstruction.
- On chest exam don’t forget to examine the back for ( pulmonary edema in the basal zone ), examine the liver if there is any hyperinflated lung
Signs of hyperinflated lung on CXR:
1- Flat diaphragm.
2- Cylindrical heart shape.
3- Darkening of lung marking.

CXR finding in patient with bronchiolitis:
1-could be normal
2- Hyperinflation
3- Non-homogenous opacity

To assess whether it is good exposure or not, look to the vertebrae it must be just visible.

When to admit patient to the hospital if he has diarrhea?

A. Severe Diarrhea with difficulty maintaining hydration
B. Very young
C. Severe co-morbid illness
D. Paralytic ileus
E. High fever
F. Intractable vomiting.
G. Coma
H. Complicated gastroenteritis eg; renal failure

Antibiotic usage in diarrhea:

A. Contraindications
1. Grossly bloody Diarrhea or other signs of Escherichia coli 0157:H7 (STEC: Shiga Toxin E coli)

B. Indications
1. Findings suggestive of Bacterial diarrhea
   a. Guaiac positive stool (not grossly bloody stool)
   b. Fecal Leukocyte positive
2. Diarrheal illness lasting longer than 10-14 days
3. Immunocompromised patients
4. Severe illness or Sepsis
5. Less than 3 months age.
6. Suspected meningitis

C. Empiric Antibiotics
1. Ciprofloxacin
   a. Empiric dose: 500 to 1000 mg once or 500 mg twice daily for 3 days
   b. Preferred agent for E. coli (ETEC, EIEC), Shigella
   c. Also covers Campylobacter, Salmonella, Yersinia, Cryptosporidium.
2. Trimethoprim-Sulfamethoxazole (Septra, Bactrim)
   a. Empirc dose: One twice daily for 3-5 days
   b. Preferred agent for Cyclospora or Isospora
   c. Also covers E. coli (ETEC, EIEC), Salmonella, Shigella, Vibrio Cholerae, Yersinia (Septra has higher resistance rates)

3. Azithromycin
   a. Empirc dose: 500 mg daily for 3 days
   b. Preferred agent for Campylobacter
   c. Also covers E. coli (ETEC), Salmonella, Shigella, Vibrio Cholerae

D. Other antibiotics used for specific indications

1. Metronidazole
   a. Preferred agent for Clostridium difficile, Entamoeba histolytica, Giardia

2. Doxycycline
   a. Preferred agent for Vibrio Cholerae
   b. Also covers Yersinia (when combined with an Aminoglycoside)
   c. Antiparasitic agents used for specific indications

1. See Metronidazole indications above

2. Albendazole (Albenza)
   a. Preferred agent for Microsporida

3. Tindazole (Tindazole)
   a. Covers Entamoeba histolytica (when treated in combination with Paromomycin)
   b. Also covers Giardia

4. Naldixic acid (very effective)
   SE: gastric upset, if less than 3 mo. It may lead to renal failure
   By toxicity, increase intracranial pressure, bulging fontanelle
   Meningitis may presented as parenteral diarrhoea
   Cerebral edema presented in hypernatremia
   In infancy when there is drowsiness think always in meningitis

Enterostop

Lomotil
• It is anti-diarrheal agent (like entero-stop).
• It is anti-chonergic agent.
• Signs: respiratory depression, Hypotension, hypo-reflexia, coma, and death.
• Don’t give lomotil until age of 4-5 years ➔ due to side effects. • If there is no cause of diarrhea you can give lomotil.
• Treatment
Sagwa poisoning:
- It is lead acetate compounds.
- Symptoms: Convulsion, coma, encephalopathy → death.
- No surviving, it is acute positioning.

Motilium:
Phenothiazine group poisoning:
- Anti-emetics (metochlopromide & domeperidone)
- Treat the cause of vomiting and not give anti-emetic to child side effects of antiemetics are hypotension, ataxia, tachycardia, coma, occulogyric crisis, severe muscle rigidity.
- Vomiting can be seen in any systemic diseases so treat the cause not the symptom.
- Influenza, pneumonia, UTI, gastroenteritis, tonsillitis, infective hepatitis, meningitis... etc → all can lead to diarrhea.
- The only indication for use of anti-emetics in children are GERD and before performing jejunal biopsy in celiac disease
- Treatment → General Measures + Anti-dote (Benztropine) + anti-histamine (chlorpheneramine) or diazepam (IV).
- If you give diazepam only the case could be re-occur.

6th stage Pediatrics lec.3

Session notes

Notes:
Blood group taking in the history is important in hemolytic diseases such as: ABO incompatibility & Rh incompatibility (mother: Rh-ve, baby:Rh+ve)

Cerebral palsy:
In general means disorder of posture & motion
Causes
1) Prenatal
- Prematurity
- LBW
- Intrauterine exposure to maternal infection
- Maternal medical problems
- Sever toxemia and eclampsia
- Bleeding in the 3rd trimester
- Drug abuse, drug therapy and toxic exposure
- Trauma
- Multiple pregnancies
- Placental insufficiency
- Congenital malformations
2) Natal
- Prolonged and difficult labor
- Premature rupture of membrane
- Presentation anomalies
- Vaginal bleeding at the time of admission for labor
- Bradycardia
- Encephalopathy

3) Postnatal
- CNS infection: encephalitis and meningitis.
- Hypoxia.
- Seizure.
- Coagulopathies.
- Neonatal hyperbilirubinaemia (kernicterus): stormy event.
- Head trauma

### Types

<table>
<thead>
<tr>
<th>Classification of Cerebral Palsy by Type of Motor Disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Spastic cerebral palsy</strong>: the most common form of cerebral palsy. It accounts for 70%-80% of cases. It results from injury to the upper motor neurons of the pyramidal tract. It may occasionally be bilateral. It is characterized by at least two of the following:</td>
</tr>
<tr>
<td>Abnormal movement pattern</td>
</tr>
<tr>
<td>Increased tone</td>
</tr>
<tr>
<td>Pathologic reflexes (e.g., Babinski response, hyperreflexia)</td>
</tr>
<tr>
<td><strong>Dyskinetic cerebral palsy</strong>: occurs in 10%-15% of cases. It is dominated by abnormal patterns of movement and involuntary, uncontrolled, recurring movements.</td>
</tr>
<tr>
<td><strong>Ataxic cerebral palsy</strong>: accounts for &lt;5% of cases. This form of the disease results from cerebellar injury and features abnormal posture or movement and loss of orderly muscle coordination or both.</td>
</tr>
<tr>
<td><strong>Dystonic cerebral palsy</strong>: also uncommon. It is characterized by reduced activity and stiff movement (hypokinesia) and hypotonia.</td>
</tr>
<tr>
<td><strong>Choreoathetotic cerebral palsy</strong>: rare now that excessive hyperbilirubinemia is aggressively prevented and treated. This form is dominated by increased and stormy movements (hyperkinesia) and hypotonia.</td>
</tr>
<tr>
<td><strong>Mixed cerebral palsy</strong>: accounts for 10%-15% of cases. This term is used when more than one type of motor pattern is present and when one pattern does not clearly dominate another. It typically is associated with more complications, including sensory deficits, seizures, and cognitive-perceptual impairments.</td>
</tr>
</tbody>
</table>

### Topographic subclassification of spastic CP:

| Hemiparesis (hemiplegia): predominantly unilateral impairment of the arm and leg on the same (e.g., right or left) side |
| Diplegia: motor impairment primarily of the legs (often with some limited involvement of the arms; some authors challenge this specific type as not being different from quadriplegia) |
| Quadriplegia: all four limbs (whole body) are functionally compromised |
• hemiplegic CP :the cause may be emboli or CVA in the intrauterine life.
• in case of CP :concentrate on prenatal Hx,developmental Hx(Global or in certain parts),family Hx is also important.
• Grasping :- begins in (3)mo.
• Grasp reflex :-hold the rattle.
• Don't forget hearing & vision, ask about them.
• He developed well for some periods of time then till (6) months he begin to regress, this is typical Hx of pt with degenerative disease, also called lysosomal storage disease. Ex:-Gaucher's disease/Tay–Sachs disease.

**Meningitis & CP:-**
- **CP**: insult to brain during its development.
- **CP** is one of the complication of **meningitis**, they are:
  1) Deafness/blindness.
  2) Mental retardation
  3) epilepsy
  4) Hemiplegia/monoplegia
  5) Hydrocephalus
  
  **Rx of CP:-** multidisciplinary team ((very important))
  1) Physiotherapist
  2) Orthopedic surgeon
  3) Pediatric surgeon
  4) Ophthalmologist
  5) Occupational therapist :- trying to make the child adapt.

• **Consanguinity** is important in autosomal recessive disorders (galactosemia, Glycogen storage disease, mucopolysacridosis)

  • We share eighth of the genes (1/8 of the genes), especially if 1st degree relative.

**General exam of the child with CP (we saw in the ward**):
- are there any dysmorphic features?
- How is the body built?
- He looks ill or healthy?
- Dyspneic or NOT?
- Cyanosed ?
- Dehydrated ?
- Posture ?
- Instruments ?
- Abnormal body movement?(flexed spastic all joints are contracted for example).
Malnutrition:

Kwashiorkor
1. This disease is caused by the deficiency of protein in the diet of child.
2. Kwashiorkor occurs in children in the age group 1-5 years.
3. The disease is more common in villages where there is small gap period between successive pregnancies.
4. In this disease, swelling of body is observed due to retention of fluids.
5. Wasting of muscles is not evident.
6. Skin changes color and become broken and scaly.

Marasmus
1. This disease is caused by deficiency of protein as well as energy nutrients (that is carbohydrates and fats) in the diet.
2. Marasmus occurs in children below the age of 1 year.
3. This disease is more common in towns and cities where breast-feeding in discontinued quite early.
4. No swelling of body takes place in Marasmus.
5. In Marasmus, wasting of muscles is quite evident. The child is reduced to skin and bones.
6. Skin does not change color and does not break.

Kwashiorkor
Oedema
Psychomotor changes
Growth retardation
Muscle wasting

Usually present signs:
Moon face
Hair changes
Skin depigmentation
Anaemia

Occasionally present signs:
Hepatomegaly
Flaky paint dermatitis
Cardiomyopathy & failure
Dehydration (diarrh. & vomiting)
Signs of vitamin deficiencies
Signs of infections

**Clinical features of marasmus**
Severe wasting of muscle & s/c fats
Severe growth retardation
Child looks older than his age
No edema or hair changes
Alert but miserable
Hungry
Diarrhoea & dehydration

**Investigations** are usually guided by history and examination. Routine tests may include:
- FBC.
- Urinalysis.
- Urine culture.
- U&E and creatinine.
- LFTs, including total protein and albumin.
- Celiac screen.
- Prealbumin which may be used as a nutritional marker.
  The following tests are not usually routine but may be indicated by history and examination:
- Testing for HIV infection.
- Sweat chloride test.
- TFTs.
- Stool studies for parasites or malabsorption.
- Immunoglobulins.
- Purified protein derivative (PPD) skin test (for tuberculosis).
- Radiological studies (bone age may be helpful to distinguish genetic short stature from constitutional delay of growth).
Special tests may be used for coeliac disease or to detect growth hormone deficiency

**Treatment:**
Normal formula contain 100 ml = 67 Kcl
In malnutrition this formula changed to F100 = 100 Kcl (high calories)
Start at F75 milk to avoid vomiting and diarrhea
We can commence from 50% of the expected feeding at that age of child
Until reach 150% of the expected

**Developmental assessment:**
-Gross motor.
-Fine motor.
-Language
-Social.

**Gross Motor:**
A newborn has head lag on pulling to sitting position. In ventral suspension, the head and the limbs are flexed.
By 3 months, the head lag is minimal when pulled to sitting position. The baby can raise the chin in prone position. On ventral suspension, the head are in line with the body.
By 4 months, the baby has head control when held up. In prone position, the baby can raise the head and shoulder.
By 5 months, the baby bears weight on the forearm in prone position and can bear weight on standing, also rolling over can be seen at this age.
By 6 months, the baby bears weight on hands in prone position.
By 7 months, the baby can sit with support and lean forward and also bounce on standing.
By 8 months, the baby crawls on the abdomen, and can sits without support.
By 10 months, most of the babies pull to standing posture and walk holding on to a piece of furniture. This is called “cruising”, they can crawl.

By 1 year, the baby can make a few steps with support.
By 15 months, the baby can walks alone.
By 18 months, the baby can runs.
By 24 months, the baby can climb stairs two feet per step and the baby can also kick a ball.
By 3 years, the baby can climb stairs 1 foot per step and can ride a tricycle.
By 4 years the baby can climb down stairs one foot per step.
By 5 years, the child can skip.

**Fine Motor:**
The newborn can focus on objects.
By 1 month, the baby can follow objects for up to 90° and for 180 degrees
by 2 months.
5 By 4 months the baby can hold objects with both hands and take them to the mouth.
By 5 months, the baby can take the feet to the mouth.
By 7 months, the baby can transfer objects from hand to hand and having a palmar grasp.
By 9 months, the baby can hit two cubes together, can drink by a cup
By 10 months, the baby has a “pincer grasp” by approximating the thumb and the index finger and can uncover hidden things.
By 1 year, the baby can release objects on request and can make a tower of two cubes.
By 13 months, the baby can turn pages of a book, two to three pages at a time.
By 15 months, the baby can feed self with a spoon without much spilling.
By 18 months the baby can build a tower of 3 cubes.
By 2 years, the baby can turn page by page, and can build a tower of 6 cubes also he can scribble lines ( horizontal line ).
The baby can draw a circle by 3 years, a square by 4 years and a triangle by 6 years. Baby can dress and undress alone by 4 years.

- suquare , traingle , cycle drawing is called coping if more than 6 mo.
If less than 6 mo. Is called Emitate
- seizuring حركة المقص حركة مسك أو عد الخرز beating

- Language Development:
  Baby makes cooing sounds by 3 months.
  By 4 months, the baby can babble.
  By 7 months the baby responds to his/her name. By 7-8 months, baby vocalizes monosyllables.
  By 10 months combines monosyllables, and understands spoken speech.
  By 1 year, the baby speaks 2-3 words with meaning. By 18 months, one can speak 20 words.
  By 2 years baby can speaks about 200 words and can make a 2 words sentence.

  Personal Social Development:
  The baby regards faces by 1 month, has social smile by 6 weeks and recognizes the mother and caretakers by 3 months, can laugh loudly by 4 months.
  The baby enjoys looking at the mirror by 6 months and imitates other by 1 year.
  By 6 months, the baby shows sadness when mother leaves and has stranger anxiety by 8 months.
  By 9 months, the baby can drink from a cup and by 15 months the baby can self feed with a cup and a spoon with little spilling.
  By 10 months baby can wave bye bye.
  The baby starts toilet training by around 2 years, being dry during day by around 3 years.
  Bowel control is before urine control, and girls can control before boys. By 2 years of age, baby refers to self as “I” By 3 years, the baby also has gender identity.

#hearing:
  At birth startle
  Ask mother about hearing problem ?
  Distraction test at 6 mo.
  Hearing test at birth till 3mor 6 mo
  9 mo. Listen to his / her name
  PTA ( pure tone audiometry )
  ABR ( auditory brain stem response )
  Autoacustic machine
Febrile convulsion Hx
1. generalized or focal
2. duration
3. vomiting
4. fever
5. LOC
6. deep sleep *post ictal*
7. Hx of trauma
8. cyanosis
9. drooling saliva
10. staring of the eyes upward.

- In physical exam, try first to exclude meningitis; then labile it "febrile convulsion", may be due to: otitis media, tonsillitis, UTI, roseola infantum (Human herpes virus-6: febrile convulsion, high grade fever & morbilliform rash), pneumonia.
- Signs of meningeal irritation → neck stiffness, kernig sign, brudzinski sign. All are done & confirmatively in >1yr old infant.
- The most reliable sign in meningitis is: deterioration of mental status (drowsiness).
- Fever & convulsion in a child (<18mo) for the 1st attack → LP is mandatory.
- We exclude raised ICP by fundoscopy (Atropine is given as dilator).
- If parents refuse to do LP → let them to sign on their responsibility.
- Try to explain the procedure to parents before doing it.
- (30%) recurrence of febrile convulsion
- Educate the parents →
  o put the head in lateral position (to avoid URT obstruction).
  o wait (1-2) min.
  o try to reserve suppositories of anti-pyretics in the home always.
- **Investigations**
  RBS, serum electrolytes, CBC, blood culture, CRP, LP, CXR, GUE and neuroimaging (CT-scan).

  **Procedure of LP:**
  1) pt in sitting position & lean forward or laterally directed.
  2) Sterilization of the area in circular pattern, beginning from centre & directed to periphery.
  3) Transverse line from iliac crest, space above, space below.
  4) 2cc is to be drawn if cloudy.

- Most common organism is strep. pneumoniae
- Try to calm the parents, say its benign problem.
- If no meningitis, put in your mind: viral infection: no need for Rx only anti-pyretic.

- **Rx:**
  - Airway, O2, suctioning, IV cannula.
  - 1/2 mg rectally diazepam.
  - 0.25 mg/kg IV diazepam.
  - *Next time if fever start: prophylactic diazepam is indicated.*
  - **proper position of bottle feeding (to avoid air)**

**POLYCYTHEMIA**
- Emergency in diabetic mother infant.
- Send for PCV, if >65 → venesection.

**Heart Failure**
- *admission to ICU*
  1. O2
  2. IV line ((only give maintenance: 150<6mo, 125>6mo, after 1yr: 100): chart input/output/weighing the child every day is v.imp (to see if the edema resolved or NOT).
  3. Head up, tube feeding "very imp".
  4. Diuretics (1-2mg/kg).
  5. Digoxin

  **don't diagnose H.F without cardiomegaly (is a must), tender hepatomegaly, tachycardia/tachypnea, Galop rhythm.**

**Signs of H.F**
- 1. Dyspnea on exertion.
- 2. Pulmonary rales.
- 3. Tender hepatomegaly.
- 4. Tachycardia.
**Acute chest syndrome**
- Severe and may be fatal due to vasooclusive crises leading to hypoxia and needs urgent exchange blood transfusion and ventilation.
- **Rx**
  - regular monthly blood transfusion to dilute the sickling cells and prevent recurrence. Also give hydroxurea to increase HBF
  - Pneumococcal vaccine should be given and also routine vaccination for H.influenza.
  - Exclusive curative treatment is by BMT.

**Renal failure "anuria"**
- Chart for input /output,lv fluid/protein restriction/weighing every day/if hyperkalemia → correct/diuretics(1mg/kg/dose).
  **then look for the cause.**

**Hypoglycemia**
- Presentation: Hypotonia, lethargy, apathy, poor feeding, jitteriness, and seizure are common.
- Congestive heart failure, tachycardia, cyanosis, pallor, diaphoresis, apnea, and hypothermia.
- **Rx:** Requires IV fluid, hypertonic glucose as initial intravenous bolus infusion of 200mg/kg [2ml/kg] 10%glucose, this should be followed immediately by continuous infusion of 6-8mg/kg/min of glucose.
  - **Note:** aminophylline dose :250mg= 250cc/ 375 mg.
  - Don't use salbutamol in child (<15-12mo)age.

**ITP**
- Hx of previous viral infection (2-3)wks.
- Presentation:- in otherwise healthy infant in the absence of Hepatosplenomegaly/bone pain/anemia/lymphadenopathy.
- **Investigations:** CBC, blood film→ reduction in platelets count+absent of immature leucocytes.
  - Do n't do bleeding time test as this leads to severe bleeding sometimes.
  - If leukemia is suspected → Bone marrow exam is mandatory.
General notes:
No rickets during marasmus due to calcium and minerals deficiency
- Caput quadratum → caused by rickets
- Clubbing → most obvious site → big toe

Cyanotic heart disease:
During history presentation try to avoid using definitive diagnose try to give differential diagnose.

Hypercyanotic spell

Management:
- Moist O2 inhalation
- Knee Chest
- Morphin Sulphate 0.1-0.2mg/kg IM/IV
- I.V. Propranolol (0.5 mg/kg)
- NaHCo3 : 1 mEq /kg I.V.
- Phenylephrine 0.02 mg/kg
- Ketamine 1-3 mg/kg I.V.
- Gen. Anaesthesia
- Emergency Surgical Intervention

- Recurrent chest infection occurs in right to left shunt
- Infection also reaches to brain → brain abscess
- This infection can be rise from valve affected by infective endocarditis.
- Circumsicion can result in → bleeding due to secondary polycythemia
- Child with snoring → adenoid hypertrophy.

DDx of cyanotic heart disease:

Table 1. Etiologies of Cyanotic CHD

- Tetralogy of Fallot.
- Total anomalous pulmonary venous return.
- Transposition of the great arteries.
- Tricuspid atresia.
- Truncus arteriosus.
- Pulmonary atresia or stenosis.
- Ebstein's anomaly.

Drugs cause congenital heart disease:
In a newborn which is centrally cyanosed at birth what is your DDx?
1-CNS: convulsions, coma, abnormal breathing, acidic respiration gasping breathing, pupillary dilatation, spastic.
2-haematological: rare may be due to methaemoglobinemia
3-respiratory
4- cardiac.

---

# How to differentiate between cardiac and respiratory cause in cyanosed newborn?
A/ by hyperoxia test.

<table>
<thead>
<tr>
<th>Hyperoxia test- Cardiac or Pulmonary?</th>
</tr>
</thead>
<tbody>
<tr>
<td>paO2 On 100% oxygen</td>
</tr>
<tr>
<td>Pulmonary disease (V/Q mismatch)</td>
</tr>
<tr>
<td>&gt;150mm Hg</td>
</tr>
<tr>
<td>Cardiac disease or PPHN (SHUNT)</td>
</tr>
<tr>
<td>&lt;150 mm Hg</td>
</tr>
<tr>
<td>Tetralogy of Fallot, Tricuspid Atresia</td>
</tr>
<tr>
<td>(Reduced pulmonary flow)</td>
</tr>
<tr>
<td>&lt;50 mm Hg</td>
</tr>
<tr>
<td>Truncus Arteriosus</td>
</tr>
<tr>
<td>(No restricted pulmonary blood flow)</td>
</tr>
<tr>
<td>50-150mm Hg</td>
</tr>
</tbody>
</table>

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# How to differentiate between CP and degenerative brain diseases:

<table>
<thead>
<tr>
<th>Cerebral palsy</th>
<th>Degenerative brain disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Acquired</td>
<td>1. inherited</td>
</tr>
<tr>
<td>2. static</td>
<td>2-progressive</td>
</tr>
<tr>
<td>3-no inheritance</td>
<td>3-autosomal recessive eg, WHD</td>
</tr>
<tr>
<td>4-associated with UTI</td>
<td>4-associated with PKU , glycogen storage diseases</td>
</tr>
</tbody>
</table>

**Leukemia presentation:**

- Systemic: Weight loss, Fatigue, Loss of appetite
- Lungs: Easy shortness of breath, Swelling
- Muscular: Weakness
- Bones or joints: Pain or tenderness
- Lymph nodes: Swelling
- Skin: Night sweats, Easy bleeding and bruising, Purplish patches

---

**Bleeding tendency:**

Check for
1-frequency
2- Local or general
3-any trauma, circumcision
4-history of affected liver or spleen
5-history of bleeding tendency, PUO, haematological dis.
6-family history
7- Ask about menorrhagia a,d metorrhagia in female

On Exam: look for:
1-anemia
2-purpura, petechia, ecchymosis
3-lymphadenopathy
4-hepatosplennegaly
5-uremia from acidotic breathing
6-exam the joint mainly in SLE (bleeding + arthritis)
or recurrent haemarthrosis and leukemia
7-examine the skin, mucous membrane, conjunctiva
Note: if there is any hemorrhagic bullae in the oral cavity or conjunctiva it is due to thrombocytopenia
PT (protrombin time to measure factors (2, 5, 7, 10) abbreviated as (1927)
PTT (prothromboplasin time): to measure any factor except 7

Case: a male baby with GIT bleeding, breast fed baby
Invx: PTT increased, PT increase, BT normal, what is the Dx?
A: Hemorrhagic disease Vitamin K deficency
Invx: PTT, PT, BT
Tx: According to condition:
Life threatening bleeding → blood transfusion, fresh frozen plasma
Mild → Vit.K replacement
After 2 hours pt return normal

Case: male newborn his aunt son with bleeding tendency and hemorrhagthesis?
A: Haemophilia
INVX: factor 8, 9
Tx: factor 8 replacement

Note: all thrombocytopenia associated with mucous membrane bleeding and GIT bleeding more than deeper tissue bleeding
Haemophilia rarely associated with GIT bleeding
Case: a child with influenza, recurrent nose bleeding, skin bleeding
After 2 weeks from starting of infection, on exam normal temp. Hb =12
No hepatosplenomegalnoy LAP your Dx?
A: Idiopathic thrombocytopenias (ITP)
Dx: BM biobsy, increase megakaryocytes
Case: a female child 3 years old with recurrent nose bleeding on exam well growth no anemia no fever, Hb: 12, platelets: 12,000
BT: increase, PTT: increase, her mother with heavy cycle?
Dx: Von willbrand dis. VWB
Invx: PFT (platelet function test)
Exam by: Antibiotics (restocin), ADP
Treatment is to give DDAVP, for mild bleeding, or give plasma-derived FVIII concentrate, which cannot produced by recombinant way, it should be with FVIII because it hold it in the plasma. Also same advice to the pt, not to have intramuscular injection or aspirin or NSAID

Down's syndrome (trisomy 21)
- This is the most common autosomal trisomy and the most common genetic cause of severe learning difficulties. The incidence in live-born infants is about 1 in 650. Cytogenetics
- The extra chromosome 21 may result from non-disjunction, translocation or Mosaicism
Non-disjunction (94%) most cases result from an error at meiosis the pair of chromosome 21s fails to separate, so that one gamete has two chromosome 21s and one has none fertilisation of the gamete with two chromosome 21s gives rise to a zygote with trisomy 21 parental chromosomes do not need to be examined. 

Translocation (5%) When the extra chromosome 21 is joined onto another chromosome (usually chromosome 14, but occasionally chromosome 15, 22 or 21), this is known as an unbalanced Robertsonian translocation. An affected child has 46 chromosomes, but three copies of chromosome 21 material. In this situation, parental chromosomal analysis is essential since one of the parents carries a balanced translocation in 25% of cases. Translocation carriers have 45 chromosomes, one of which consists the two joined chromosomes the risk of recurrence is 10-15% if the mother is the translocation carrier and about 2.5% if the father is the carrier if a parent carries the rare 21:21 translocation, all the offspring will have Down's syndrome if neither parent carries a translocation (75% of cases).

**Electrocardiography (ECG):**

**Definition:**

Recording the electrical activity of the heart

---

**INTERPRETING THE ECG**

- **P wave:** Atrial depolarization.
  - It is normally 2.5mm or less in height
  - 0.11 seconds or less in duration.

- **QRS Complex:** Ventricular depolarization.
  - Less than 0.04 seconds in duration
  - Less than 0.12 seconds in duration.

- **T wave:** Ventricular repolarization.

- **U wave:** Repolarization of the Purkinje fibers.

**SPEED 25-50 mm/sec according to the activity**

- Prolonged PR interval → BBB normal 3-5 small squares
- Short PR interval → WPW
- QRS from beginning of Q till to the end of S
- Prolonged in QRS → BBB
- QTc from end of QRS till to end of T-WAVE
- QTc interval

---

**Paediatric electrocardiographic findings that may be normal**

- Heart rate > 100 beats/min
- QRS axis > 90°
- Right precordial T wave inversion
- Dominant right precordial R waves
- Short PR and QT intervals
- Short P wave and short duration of QRS complexes
- Inferior and lateral Q waves
QT interval prolonged if > 0.45 sec →
ECG reading:
When you ECG read should be done in a structured way:
1-rhythm: regular or irregular
Look for the interval between the R-R wave.
2-Rate:
3-The axis: it is right or left axis
How you going to assess the axis?
Before birth baby’s right ventricle is the predominant and the
left ventricle is not-functioning as baby receives blood from
his circulation and mother and is it is oxygenated blood already
by SVC TO RA AND RIGHT VENTRICLE.
**right ventricular predominance:**
to decide the axis look for the:
lead I, III, avF = limb leads

Example: 1 day
-lead I shaking hand with lead II (lead I ↓ and AVf (or lead III) ↑ → right axis deviation.
If lead I ↑ and lead III (or AVf) ↓ → left axis deviation
If the two leads looks toward the same side → it is normal axis
If the both lead I and lead III are ↓ → it is undetermined axis (extreme right or extreme left)

**Axis:**
0 → 90 normal
90 → 180 right axis
0 → - 90 left axis
0 → - 180 indeterminate right or extreme
Right ventricular predominance
In normal Chest lead cover the right ventricle from V1-V5.
V6 receives the voltage from left ventricle as it is lies
posterior predominantly.
In patient with VSD can be present with right ventricular
hypertrophy and left to right shunt and pulmonary HT
If V6 is negative it is → RVH
IF V6 is positive → it is LVH
AFTER that look for the interval:
QRS, P-wave, QT interval, ST, PR
Right ventricular hypertrophy
Criteria:
1. Prominent right axis deviation
2. Prominent R IN V1
3. Small S in V6
4. UP ward T-wave in (V 1, 2 and 3)

-When child grows right axis deviation changed from right toward the normal site
-During the first year of life right axis effects is resolved
-Normal RS progression proceeds during child growth and changed from right axis deviation toward left axis until in become normal (0 - +90°).
- Prominent R in V1 decrease and increase in V6
- Prominent S1 increase in V1 and decrease in V6

Q/Can MI presented in pediatric age group?
1- Familial hyperlipidemia
2- anomalous origin from left coronary artery
3- Kawasaki dis. → thrombosis of coronary a
4- Thrombophilia → inheretd anti-thrombin deficiency.

-p- pulmonale → right atrial hypertrophy
-p- mital → left atrial hypertrophy

Best reading for ECG from lead II
And take a trace ECG to discover rhythm abnormality
Tented T-wave in lead II indicate → hyperkalemia
V4 R mains V4 on the right side and it is similar to V1
Sinus arrythemia Sinus arrythmia: occur during inspiration by increased heart beats
- Ectopic beat with pause temporary
QRS distorted
Right ventricular hypertrophy: upward T-wave in V1, V 4
Strain pattern in V6 IN left ventricular hypertrophy
Complete bundle branch block or RS-R pattern

This condition presented in ostium primeum canal associated with Down's syndrome
ASD+VSD = AV CANAL

Regarding T wave in V1, 2 and 3:
- 6 days- 6 years → must be inverted (otherwise → it is RVH)
- 6 years- 12 years → may or may not be inverted.

One of the main differences from adult ECG.

QT prolonged in
1- Congenital disease:
   a- Autosomal recessive
   b- Autosomal dominant
2- Acquired:
   Hypocalcemia most common and less in hypokalemia, hypomagnesimia
   The danger when changed to arrythmia and it is bradycardia
Tachycardia + absent p-wave = SVT
Tx:
Short PR, J-wave, wide QRS \(\rightarrow\) Wolf parkinsonian white syndrome
SVT + adenosine
ECG RBBB \(\rightarrow\) IN OSTIUM PRIMEIUM
LBBB \(\rightarrow\) IN CARDIAC SURGERY

**6th stage**

<table>
<thead>
<tr>
<th>Pediatrics</th>
<th>lec.3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Session notes</td>
<td></td>
</tr>
</tbody>
</table>

**Neonatal jaundice**
Prolonged neonatal jaundice means more than the physiological \(\rightarrow\) pathological
Ask about family Hx, TORCH infection
Hemorrhagic disease of newborn indicate \(\rightarrow\) Vit.K deficiency syndrome

**DDx of Generalized bleeding tendency?**
1- ITP (idiopathic thrombocytopenic purpura)
2- Haemophilia
3- Factor 7, 5 deficiency

**Approach to patient with hemorrhagic disease:**
1- Bleeding profile
2- CBC
3- GUE: to search for bile pigment \(\rightarrow\) cholestasis
4- Urine reducing substance: to look for galactosemia
5- Chromatography: to check for amino-acid
6- ASPG, ASPOT liver enzyme \(\rightarrow\) to see if there is hepatitis
7- Alkaline phosphatase enzyme.
8- Blood group: ABO, Rh to exclude inspissated bile syndrome is defined as partial or complete obstruction of the extra hepatic biliary system by impaction of thick bile or sludge in the distal common bile duct during the neonatal period.
9- Congenital infection: TORCH (Toxoplasmosis rubella, Cytomegalovirus, Herpes virus) to measure IgM or IgG

**Hemorrhagic disease classified as:**
1- Early type: 24 hours
2- Classical: 24 – 7 days
3- Late: > 7 days
   eg; liver disease (cholastasis), alpha1-antitrypsin deficiency, galactosemia, malabsorption
   Invx: PTT, PT, CBC, BT (Bleeding profile)
   In Vit.K \(\rightarrow\) ↑ PT, ↑ APTT, BT normal
10-U/S: intrahepatic biliary diltation
11- Cholescitography using Hida scan.

**On Exam:**
1- cataract: associated with galactosemia
2- face: if ugly \(\rightarrow\) Argyll syndrome
3- Heart: look for coarctation of aorta, VSD
4- convulsion: seek for hypoglycemia

**Treatment:**
Vit. K replacement mainly fresh frozen plasma, There is 3 types of vitamin K (1, 2 and 3), given mainly IM and monitor the patient to avoid anaphylactic shock
Surgical operation for biliary atresia is called Kasai portoenterostomy.

---

**Diabetic ketoacidosis**
Random blood sugar should be:
- \(>11\) mmol/l or \(200\) mg/l
- In fasting \(>7\) mmol/l or \(127\) mg / l

Look for this sign in DKA:
1- Tachypnea
2- dehydration
3- Abdominal pain
4- vomiting
5- polyuria, polydepsia

**Management**
1- hospital admission
2- monitoring
3- IV canula
4- Draw bolld sample for: RBS, seum electrolyte
5- blood gas analysis (BGA)
6- Serum BUN

Even patient is severely dehydrated urin still present due to osmotic diuresis
7- HbA1c (glycosalted hemoglobin)
For past 3 months if \(>6.5\) is diagnostic for DKA
There pseudohypernatremia in DKA
Approach to pt with SOB
1- onset of SOB
2- duration of SOB
3- timing (at night or day time)
4- precipitating factors: exercise , perfume , dust.
5- Ass. symptoms : cyanosis or congestion, runny nose, noisy breathing, chest pain , sore throat, sputum , hemoptysis
8- aggravating factors & relieving factors: certain position
9- frequency of the attacks
10- wt. gain , sleeping pattern , interfere with the activity
11- associated with eczema , allergy
12- management received ?response?

Pt with cough
1- Duration
2- Onset
3- Time of occurrence ( night , morning)
4- Frequency&severity
5- Short or Paroxysmal
6- Character : barking , whooping
7- Dry or productive : if there is sputum : color , amount , with blood , smell
8- Aggravating factors relieving factors
9- Associated symptoms( fever, dyspnea, vomiting , convulsion, cyanosis , noisy breathing, hoarse voice, sore throat)
11- Effect on feeding , sleeping , activity
VSD→acyanotic.
CHD inherited as multifactorial.
Accounts for 25% of CHD
harsh pansystolic murmur heard along the LSB, more prominent with small VSD

**Complications:**
1- FTT
2- CHF
3- pulmonary hypertension
4- infective endocarditis
5- CVA
6- arrhythmia

**Investigations:**
CXR
ECG
ECHO

**Treatment**
- Small VSD - no surgical intervention, no physical restrictions, just reassurance and periodic follow-up and endocarditis prophylaxis.
- Symptomatic VSD - Medical treatment initially with afterload reducers & diuretics ± digoxin
- Prophylaxis against infective endocarditis (after dental or GU procedures)

**Indications for Surgical Closure**
- Large VSD with medically uncontrolled symptomatology & continued FTT.
- Ages 6-12 mo with large VSD & Pulmonary HTN.
- Age > 24 mo w/ Qp:Qs ratio > 2:1.
- Supracristal VSD of any size.

ASD → RV heave, fixed widely split S2, systolic ejection murmer/on ECG —right-axis deviation and RVH.
NOTE → RAD normal in infants & children till 4yrs of life.
H.F complications

1- FTT caused by chronic hypoxia + increased caloric needs by the tissue + feeding interruption + drugs taken that leads to anorexia.
2- recurrent chest infection.

Signs of H.F →
1) tachycardia
2) tachypnea
3) pulmonary rales
4) cardiomegaly on XR.

** ask about consanguinity + any family Hx for CHD → for recurrence of same problem in the other siblings.

Rash + abdominal pain + fever + vomiting → suspect HSP + scarlet fever "pastia lines"

The definitive diagnostic method to enteric fever → blood culture, widal test will be positive 2 wks later on.

Connective tissue disease criteria (4 of which should be present):

- Constitutional (eg, fatigue, fever, arthralgia, weight changes)
- Musculoskeletal (eg, arthralgia, arthropathy, myalgia, frank arthritis, avascular necrosis)
- Dermatologic (eg, malar rash, photosensitivity, discoid lupus)
- Renal (eg, acute or chronic renal failure, acute nephritic disease)
- Neuropsychiatric (eg, seizure, psychosis)
- Pulmonary (eg, pleurisy, pleural effusion, pneumonitis, pulmonary hypertension, interstitial lung disease)
- Gastrointestinal (eg, nausea, dyspepsia, abdominal pain)
- Cardiac (eg, pericarditis, myocarditis)
- Hematologic (eg, cytopenias such as leukopenia, lymphopenia, anemia, or thrombocytopenia)