Answers of

PEDiATRIC SHEET

EXCEPTIONAL LEARNERS

https://www.facebook.com/azharpulse.cairo
1. Polyarticular R.A.
2. SLE.
3. T.B.
4. Dermatomyositis.
5. Malignancy.
6. IBD (Chron’s – ulcerative colitis).


### D.D.

1. Other Rh. Diseases
   - SLE, juvenile dermatomyositis, sarcoidosis.
   - Scleroderma, vasculitic syndrome.
   - Auto immune hepatitis, lyme disease.
   - Trauma – infection.
2. Single joint
   - Bacterial infection.
   - Osteomyelitis.
   - Psoriatic arthritis.
   - Chondromalacia of patella and femoro patellar syndrome.
   - Chondrolysis of hip.
3. Persistent arthralgia
   - Juvenile episodic arthritis.
   - Hyper mobility syndrome.
   - IBD, enteric infection.
   - School phobia.
4. Less commonly
   - Leukemia.
   - Cystic fibrosis.
   - D.M. – skeletal dysplasia.
   - H.I.D. (humoral immune deficiency).
   - Glycogen storage disease.

### Diagnosis

1. No pathognomonic finding.
2. Clinical subtypes
   - Pauci articular 50 % (one – two).
   - Poly articular (35 %) (one –two).
   - Systemic onset (15 %) felty syndrome.
3. Exclusion of other articular diseases
4. CBC
   - ESR: increased.
   - CRP: increased.
   - WBCs: leucocytosis.
   - RBCs: anemia.
   - Platelets: thrombocytosis.
Henoch Schenolin pupura (HSP) (clinical pictures) 1996

Clinical pictures

- Onset: acute or insidious.
- Course: over weeks or months.
- General: low grade fever – fatigue.
- Local: angioedema ➔ pinkish maculopapular rash (pathognomonic) ➔ palpable purpura.

Skin

Palpable purpura (100 % of cases) on buttock, flexor of lower limb, extensors of upper limb

- Occur in crops and as long as
  - 3 - 10 days.
  - 3 – 4 months. or
  - Even a year or more.

Systemic

1. Arthritis
   - Poly articular.
   - Large joints. { No deformity
   - Exudative lesion.

2. GIT: vasculitis of mesentric artery
   - Abdominal pain.
   - Bleeding per rectum.
   - Intussusception.
   - Submucosal hemorrhage.
   - Intestinal infarction.
   - Peritoneal exudate.
   - HSM.
   - Lymphadenopathy.
   - Pancreatitis.

3. Renal: Focal G.N.
5. Pulmonary and intra muscular hemorrhage.
6. Rh. Like nodules – non neuropathies.
7. Cardiac involvement.

SLE (clinical pictures) 1997

2. Cutaneous:
   - Malar rash: erythema over cheeks.
   - Butterfly rash: erythema over cheeks and nose.
   - Photo sensitivity: skin rash (unusual reaction to sunlight).
   - Alopecia.
   - Vasculitis
     - Purpura – leg ulcer. ✓
     - Raynaud's phenomenon. ✓

3. Musculoskeletal
   - Arthalgia (pain only) and myalgia.
   - Renal: lupus nephritis.
   - Cardiac:
     ✓ Libman sacks endocarditis ➔ AR – MR.
     ✓ I.H.Ds. – myocarditis ➔ heart failure.
   - Chest
     ✓ Chronic fibrosis.
     ✓ Pulmonary infarction (shrinking lung syndrome).
   - Serositis: pleural, pericardial, peritoneal.
- C.N.S: neuropathy – memory loss.
- Neuro psychiatric: psychosis (severe).
- GIT
  - Vasculitis → abdominal pain, infarction.
  - Painless mouth ulcer.
  - HSM – Hepatitis.
  - Lymphadenopathy, IBD.
- Arterial and venous thrombosis
  - Anti phospholipid syndrome.

**Juvenile R.A. (clinical pictures) 1995**

**Clinical pictures**

1. Chronic arthritis.
2. Early morning stiffness or arthalgia for > 6 – 8 weeks.
3. Deformity
   - Spindle shaped fingers.
   - Ulnar deviation of hands.
4. Classification (clinical types)
   
   According to onset and number of joint affection.

   a) **Poly articular (35 %)**
   - Female > male.
   - No systemic manifestation.
   - Affect small and large joint ≥ 5 joints.
   - Rheumatoid nodules.
   - Micoglnathia → due to TMJ (Tempro mandibular joint) involvement.
   - Atalanto axial sublaxation.
   - Hoarse voice → due to crycoarnoid joint involvement.

   b) **Pauci articular (50 %)** < 5 joints affected
   (knee – ankle) most affected – two types (one – two)

   c) **Systemic onset disease (15 %)** Felty's syndrome
   - Arthritis → prominent visceral involvement.
   - HSM – lymphadenopathy – serositis.
   - Fever for ≥ 2 weeks with daily spikes at least 39.5 C.
   - Associated with Salmon colored lesions over trunk and proximal extremities.
**C.N.S.**

**MR (causes, common presentation) 2008-03-02 ★★★**

**Causes**

1. Genetic syndrome.
2. Chromosomal disorder.
4. Pre natal and post natal causes.
5. Neurodegenerative disorders.
6. In born error of metabolism.
7. Developmental brain abnormality.
8. Unknown.

**Common presentation by age**

1. New born
   - Dysmorphism.
   - Major organ dysfunction (MOD).
2. Early infancy (2 – 4 months)
   - Failure to interact with environment.
   - Concerns about vision and hearing impairment.
3. Late infancy (6 – 18 months)
   - Gross motor delay.
4. Toddlers (2 – 3 years)
   - Language delay or difficulties.
5. Pre scholar (3 – 5 years)
   - Language delay or difficulties.
   - Behavioral difficulties including (play).
   - Delayed in motor skills (cutting – drawing).
6. School age (> 5 years)
   - Academic under achievement.
   - Behavioral difficulties
     - Attention.
     - Anxiety.
     - Mood.
     - Conflict.

**Febrile convulsions 2011-08-06-04 ★★★**

**Definition**

Generalized tonic clonic convulsions occasionally occur at onset of acute extra cranial infections.

**Etiology**

1. At onset of acute extra cranial infections e.g. tonsillitis.
2. Underlying serious acute infections.
3. Association with high environmental temperature.
Clinical pictures = criteria for diagnosis

1. Patient type
   - Age: 6 months – 6 years.
   - Sex: female > male.
   - F.H.: strong positive.
   - Neurologically and metabolically.

2. Seizure type
   - Pre ictal: occur at onset of temperature ≥ 39 C.
   - Ictal: generalized tonic clonic.
   - Short duration: (5 – 15 min).
   - Course: usually one conclusive fit (attack) during same illness (one attack at time).
   - Post ictal: short post ictal stupor (short stupor).

Treatment

1. Immediate 1st aid measures.
2. Leaving temperature measures
   - Cold fomentation.
   - Anti pyretic.
3. Treatment of cause of fever e.g.: antibiotic for tonsillitis.
4. Short acting anti convulsant (Diazepam 0.3 mg / kg / dose).

Grand mal epilepsy 2001-99
(clinical pictures, treatment)

Clinical pictures

1. Aura: unusual behavior: recognized by mother.
2. Tonic phase: painful sustained contraction (5 min) → patient falls to ground stiff with flexed arms and extended legs.
3. Clonic phase: rhythmical contraction of muscles of limbs and face
   - Biting the tongue.
   - Incontinence (may occur).
   - Variable duration but, if > 20 min → status epilepticus
4. Post epileptic phase
   - Child falls in deep sleep.
   - May be confused or irritable.

Treatment

1. Anti convulsants :
   - Sodium valporate (20 – 40 mg / kg) (drug of choice)
   - Phenytoin (3 – 5 mg), carbamazepine (10 – 20 mg / kg).

For (1 or 2 years) after last convulsion in otherwise normal child
Or longer period or even for life long in child with associated problems.

2. Advice to parents and child:
   - Full information about drug therapy.
   - Importance not stopping drug without medical advice.
   - Allow normal activities but, should be attended by responsible adult only while bathing or swimming.
   - Clear instructions about 1st aid measures:
     ✓ Ensure patent airway.
     ✓ Avoid biting tongue.
     ✓ Putting child in prone position with head down.
1. Paralytic spinal poliomyelitis.
2. Guillain Barre syndrome. (GB syndrome).
3. Transverse myelitis.
4. Post diphtheria paralysis.
5. Botulism.
6. Acute spinal cord compression.
7. With movement disorders due hypotonia
   - Acute cerebellar ataxia.
   - Rheumatic chorea.

<table>
<thead>
<tr>
<th>Poliomyelitis</th>
<th>G.B. syndrome</th>
<th>Transverse myelitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Site</td>
<td>Acute LMNL</td>
<td>Acute LMNL</td>
</tr>
<tr>
<td>Cause</td>
<td>Viral</td>
<td>Auto immune</td>
</tr>
<tr>
<td>Equality</td>
<td>Asymmetrical</td>
<td>Symmetrical</td>
</tr>
<tr>
<td>Type</td>
<td>Purely motor</td>
<td>25 % sensory</td>
</tr>
<tr>
<td>Course</td>
<td>Stationary or regressive</td>
<td>Progressive or ascending</td>
</tr>
<tr>
<td>Autonomic disturbance</td>
<td>Transient autonomic disturbance</td>
<td>No autonomic disturbance</td>
</tr>
<tr>
<td>Gait</td>
<td>Limbing gait</td>
<td>High steppage gait</td>
</tr>
<tr>
<td>C.S.F.</td>
<td>Pleocytosis – increased cell + N.ptns.</td>
<td>Cytoalbominous dissociation, increased ptn + N. cells</td>
</tr>
</tbody>
</table>

**Petit mal epilepsy (treatment) 2000**

1. Anti convulsant:
   - Drug of choice
     - Ethosuximide (20 – 40 mg / kg / day).
     - Sodium valporate (20 – 40 mg / kg / day).
   - Duration: as grand mal.
2. Advice to parents and child: as grand mal epilepsy.
   - Full information about drug therapy.
   - Importance not stopping drug without medical advice.
   - Allow normal activities but, should be attended by responsible adult only while bathing or swimming.
   - Clear instructions about 1st aid measures:
     - Ensure patent airway.
     - Avoid biting tongue.
     - Putting child in prone position with head down.
Clinical types

1. Spastic type (pyramidal type): (commonest type)
   Criteria
   a) UMN → spasticity – hypertonia – hyper reflexia, positive Babinski signs – clonus.
   b) Gait: scissoring – circumduction gait.
   c) Pseudo bulbar palsy: weak cry – difficult swallowing, positive gag reflex.

2. Athetoid (extra pyramidal) type:
   • Choreathetotic C.P.
   • Hypotonia.
   • Athetosis.
   • Dystonia.
   • Associated hearing defect.
   • Abnormal posture of limbs and trunk.

3. Ataxic C.P. (cerebellar)
   • Hypotonia, hyporeflexia.
   • Weakness.
   • Incoordinated movement and tremors.

4. Hypotonic (atonic) C.P.
   • Hypotonia and hyporeflexia.

5. Mixed C.P.

Prevention

• Early diagnosis is essential.
• Close observation.
• Correction of risk factors (LBW – hyper bilirubinemia – birth asphyxia).
• Physiotherapy.

Early detection by early signs of C.P.

1. Persistence of neonatal reflexes
   • Moro’s reflex after 6 months age.
   • Grasp reflex after 4 months age.
   • Presence of prominent tonic neck reflex in neonatal period.

2. Defective development and locomotion
   • Delayed motor development.
   • Clenched hand after 2 months.
   • Movement of one limb < other.

3. Early signs of spasticity
   Difficult abduction of thighs during change of diapers.
Causes of 1ry inability to walk (with hypotonia) 2003 - 1997

Primary cause ➔ i.e. infant never walk up to 18 months.

A. Central (congenital):
   • C.P.
   • MR.
   • Down syndrome.
   • Hydrocephalus.
   • Cretinism.

B. Peripheral causes
   • S.C. ➔ meningeocele, traumatic birth injury.
   • A.H.C. ➔ poliomyelitis, werding Hoffman disease.
   • P.N. ➔ congenital peripheral neuropathy.
   • M.E.P. ➔ congenital myasthenia gravis.
   • Muscles ➔ congenital myopathy.

Bone ➔ rickets and osteogenesis imperfecta.

D.D. of large head 2003

1. Cranial
   • Familial large head.
   • Rickets.
   • Chronic hemolytic anemia.
   • Cretinism.
   • Osteogenesis imperfecta.
   • Achondroplasia.

2. Intra cranial
   • Hydrocephalus.
   • Hydroencephaly.
   • Megalencephaly.
   • Causes on increased intra cranial tension.
     ✓ Tumor.
     ✓ Mass (tuberculoma).
     ✓ Chronic ascites.
     ✓ Hemorrhage.
     ✓ MPS (muco poly sacharridosis).

Causes of floppy infant 2002

1. C.P. (atonic – cerebellar).
2. Congenital
   • Poliomyelitis.
   • Peripheral neuropathy.
   • Myopathy.
   • Myasthenia gravis.
3. Benign congenital hypotonia.
4. Down syndrome.
5. Werding Hoffman disease.
6. Severe PEM.
BRAIN TUMOURS 2010*

- Predominantly infratentorial involving cerebellum, midbrain, brainstem
- Glial (astrocytomas most common) or primitive neuroectodermal, medulloblastoma, germ cell tumors, ependymomothera

**Signs and symptoms**
- Infratentorial: vomiting, morning headache, increased head circumference, ataxia, diplopia, nystagmus, papilledema
- Supratentorial: focal deficits, seizure, long tract signs

**Evaluation**
- History, physical exam including complete neurological exam
- CT and/or MRI of head as indicated

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## Renal

**Nephrotic syndrome (diagnostic criteria) 2008 – 2002**

### Clinico laboratory (4)
1. Heavy proteinuria.
2. Hypo albuminemia.

- Massive selective proteinuria ≥ 2 gm / day mainly albumin.
- Nephrotic edema (generalized, severe, ± ascites).
- Hypo albuminemia \(\Rightarrow\) total serum ptn < 5 gm % and serum albumin < 2.5 gm %.
- Hyper lipidemia (hypercholesterolemia) > 250 mg / dl.

+ Clinico laboratory diagnosis
- Age: 2 – 8 years.
- Good response to corticosteroids.
- Pathology: renal biopsy:
  - By L.M. \(\Rightarrow\) normal.
  - By E.M. \(\Rightarrow\) fusion of podocytes of glomeruli.
**Diagnosis**

A. Clinical pictures
   1. History of streptococcal infection (1 – 2 weeks) before onset of symptoms.
   2. FAHM: fever, malaise, lethargy, abdominal pain.
   3. Edema, HTN, oliguria.

Usually resolve after 4 – 6 weeks.

B. Investigations
   1. Urine
      - Microscopic hematuria.
      - RBCs casts.
      - Proteinuria.
   2. Mild anemia.
   3. Decreased serum C3.
   4. Kidney function tests: may be normal or impaired.
   5. Throat culture: positive streptococci.
   6. Serological evidence of streptococcal infection:
      - Increased ASOT.
      - Anti DNase.
      - Anti streptokinase.

**Complications**

1. Hypertensive encephalopathy.
2. Congestive heart failure.
3. Electrolyte disturbance.
4. A.R.F.

**Treatment**

1. Penicillin course for 10 days.
2. Bed rest during acute stage.
5. Treatment of hypertension by CCB, VDs, Diuretics, ACEIs.
6. Convulsions by diazepam.
As before (PSGN)

**Lab and clinical difference between nephritic and nephrotic syndrome 2005**

<table>
<thead>
<tr>
<th>Clinical picture</th>
<th>Nephritic syndrome</th>
<th>Nephrotic syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>3 – 10 years</td>
<td>2 – 8 years</td>
</tr>
<tr>
<td>Lab</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Urine</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Volume</td>
<td>Decreased</td>
<td>Normal</td>
</tr>
<tr>
<td>• Hematuria</td>
<td>Positive</td>
<td>Negative</td>
</tr>
<tr>
<td>• Proteinuria</td>
<td>Mild</td>
<td>Massive</td>
</tr>
<tr>
<td>• Casts</td>
<td>Red cell</td>
<td>Lipoid</td>
</tr>
<tr>
<td>2. Blood</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Albumin</td>
<td>Normal</td>
<td>Decreased</td>
</tr>
<tr>
<td>• Cholesterol</td>
<td>Normal</td>
<td>Increased</td>
</tr>
<tr>
<td>• C3</td>
<td>Decreased</td>
<td>Normal</td>
</tr>
<tr>
<td>3. Renal function</td>
<td>Disturbed</td>
<td>Normal</td>
</tr>
<tr>
<td>4. Electrolyte</td>
<td>Hypo natrema</td>
<td>Normal</td>
</tr>
<tr>
<td>5. ASOT</td>
<td>Increased</td>
<td>Normal</td>
</tr>
</tbody>
</table>

**A.R.F. (causes) 2002**

1. Pre renal
   • Severe dehydration.
   • Hemorrhage, Burns, sepsis.
2. Renal
   • Infection: post S.G.N., pyelonephritis.
   • Systemic disease: SLE.
   • Vascular: R.V. or R.A. thrombosis.
   • Hematological: acute and severe hemolysis and hemolytic uremic syndrome.
   • Acute tubular necrosis.
   • Drugs & toxins: NASID – Aminoglycosides.
   • Hypoxic ischemic injury.
3. Post renal: due to obstruction
   • Congenital: post ureteral valve.
   • Acquired: bilateral stones or tumors.

**C.R.F. (clinical pictures) 1998**

1. Non specific symptoms
   • Headache (FAHM).
   • Fatigue.
   • Lethargy.
   • Anorexia and vomiting.
2. Signs
   • Hypertension
   • Oliguria.
   • Edema and pallor.
   • Growth retardation
Urinary tract infection (clinical pictures) 1999

1. Newborn: generalized septicemia
   - Poor feeding even refusal.
   - Temperature instability (hypothermia > hyperthermia).
   - Jaundice.

2. Children: according to site of infection
   a) Upper urinary tract infection (pyelonephritis)
      - Acute: fever, rigor and loin pain.
      - Chronic:
        ✓ Prolonged fever.
        ✓ Loss of weight.
        ✓ Manifestations of renal failure:
          - Anemia.
          - Bleeding tendency.
          - Hypertension.
          - Neurological manifestations.
          - Pericarditis and cardiomyopathy.
   b) Lower urinary tract infection (cystitis, uretheritis)
      - Dysuria.
      - Frequency.
      - Dripping.
      - Urgency.
      - 2ry nocturnal enuresis.
      - ± hematuria

Hematuria (causes) 1995

1. G.N.
   - Acute post S.G.N.
   - HSP.
   - Lupus nephritis. (SLE).
   - (membraneous, proliferative, membrano proliferative) G.N.
   - Ig A nephropathy.
   - Good pasture syndrome.
   - Hemolytic uremic syndrome.

2. Infection
   - Acute pyelonephritis.
   - I.E.
   - Hemorrhagic cystitis.

3. Blood causes
   - Thrombocytopenia.
   - DIC.
   - Coagulation defect.
   - SCAN.
   - Renal vein thrombosis.

4. Drugs
   - Salicylates.
   - Anti coagulant.
   - Cyclophosphamid

5. Stone.
7. Miscellaneous
   - Bilharziasis.
   - Trauma.
   - Severe exercise.
Clinical pictures

1. Neonatal period
   - Female > male (2 : 1).
   - Relative increase in head size and large post fontanel.
   - Hypothermia, lethargy, poor feeding.
   - Abdominal distention, jaundice, umbilical hernia.
   - Large tongue, cyanosis, respiratory distress.
   - Dry skin, hoarse cry, mottled skin.

2. Later on
   - Delayed bone age.
   - Overall progress for normal intellectual development is excellent (when treatment is initiated within 1 – 2 months).
   - If therapy is initiated after 6 months or later: return of normal intellectual functions is markedly decreased, however, growth improves after thyroid displacement.

Investigations

1. T3 – T4 – TSH.
2. Serum cholesterol ➔ elevated.
3. E.C.G. ➔ bradycardia.
5. Thyroid scan.
**Diagnosis**
1. Hyper glycemia: glucose > 300 mg / dl.
2. Ketonemia (mouth acetone odor).
5. Acidosis
   - PH < 7.3.
   - HCO3 < 15 mEq / L.

**Clinically**
Patient may be
1. Consciousness: oriented or sleepy or comatosed.
2. Kussmaul respiration, polyurea, polydepsia, dehydration.

**Treatment**
Immediate aim of therapy
- Correct dehydration and expansion of I.V. volume.
- Correction of electrolytes and acid base balance status.
- Initiation of insulin therapy to correct intermediary metabolism.
1. Fluid therapy: according to body weight and body surface area 
   \[ 30 \text{ كيلو جرام} \]
   
   1st hour: saline 0.9% (500 ml / m² / hr to correct shock).
   
   2nd hour: saline (0.45 % ml / m² /hr + add K Cl 20 mEq.
   
   3rd hour up to 12 hours: (0.2 % saline + glucose 5 %  4 : 1) ➞ 200 ml / m² / hour (2000 ml / m² / 10 hours).
   
   Next 24 hours: saline + glucose (1 : 1) ➞ 100 ml / m² / hour (2,400 ml / m² / 24 hours ) + add K phosphate ➞ 40 mEq / L.

2. Insulin therapy
   - Start with 0.1 u crystalline insulin / kg / dose I.V. bolus.
   - Then add 0.1 u / kg / hour I.V. drip until glucose reach > 300 mg /dl.
   - Then 0.1 u / kg / Sc / hour.
3. To prevent hypo kalemia: start K Cl from 2nd hour from treatment  40 mEq / m2 / I.V.
4. Na HCO3: to correct acidosis 
   \[ \left( \frac{1}{3} \text{ body weight X (12 – observed HCO3)} \right) \]
5. Prophylactic antibiotic for associated infection.
6. Treatment of precipitating factors.
**Pathophysiology**

1. Progressive destruction of B cells $\rightarrow$ progressive $\downarrow$ of insulin.
2. Insulin deficiency $\rightarrow$ $\uparrow$ stress hormones (epinephrine, cortisone, G.H. and glucagon) $\rightarrow$ $\uparrow$ metabolic decompensation.
3. $\uparrow$ stress hormones $\rightarrow$ impairment secretion of insulin and antagonize its action by promoting
   - Glycogenolysis and gluconeogenesis.
   - Lipolysis and ketogenesis.
4. Hyperglycemia when exceed renal threshold $\rightarrow$ glucosuria $\rightarrow$ osmotic diuresis and polyurea, urinary loss of electrolytes and compensatory polydipsia.
5. Acute stress and infection $\rightarrow$ acceleration of metabolic decompensation $\rightarrow$ ketoacidosis.
6. Lipolysis $\uparrow$ $\rightarrow$ $\uparrow$ plasma concentration of
   - Total lipids.
   - Cholesterol and F.F.A.
7. $\downarrow$ insulin and $\uparrow$ glucagon $\rightarrow$ shunt of F.F.A. into ketoacidosis $\rightarrow$ metabolic acidosis $\rightarrow$ compensatory deep rapid breathing (Kussmaul respiration).
8. Conversion to acetoacetate $\rightarrow$ characteristic fruity odor of breath.
9. With progression
   - Hyper osmolarity and dehydration.
   - Acidosis.
   - $\downarrow$ cerebral O2 utilization and consciousness impaired & patient becomes comatose.

**Clinical picture**

1. Poly urea, polydipsia with loss of weight.
2. Eneuresis in a previously toilet-trained child.
4. Ketoacidosis:
   a) Early manifestations
      - Vomiting.
      - Polyurea.
      - Dehydration.
   b) In severe cases
      - Resp: Kussmaul respiration & acetone odor in breath.
      - Abdominal pain or rigidity.
      - C.V.S.: impaired consciousness & coma.

**Diagnosis**

1. History.
2. Clinical symptoms and signs.
3. Hyperglycemia in association with glucosuria with or without ketoacidosis.
4. Hb A1C (Glycohemoglobin).
Treatment of DKA

1. Immediate aim of therapy
   - Correct dehydration and expand I.V. volume.
   - Correction of electrolytes & acid base status.
   - Correct inter meal dairy metabolism by initiation of insulin therapy.

A. Fluid and electrolyte therapy: according to body weight and body S.A.
   a) Initial hydration fluid (isotonic saline 0.9 %).
   b) 1st hour: 10 – 20 ml /kg / I.V. of (0.9 % Na Cl) + insulin drip at (0.1u / kg / hour).
   c) 2nd hour: and until DKA resolution = delicate 85 ml / kg / 24 hours + maintenance + continue insulin drip.
   d) Administration of K should start early to correct hypo kalemia.

B. Alkali therapy: to correct acidosis (rarely used as it risk of K and cerebral edema).

C. Insulin therapy
   a) Regular insulin (0.1 u / kg / hour ) until blood glucose reach 300 mg / dl reduce to (0.05 u / kg / hour).
   b) After child eating: regular insulin (0.2 – 0.4 u / kg / 6 – 8 hours) before meals.
   c) Insulin adjusted according to monitoring glucose level before and 2 hours after meal to keep in range of (80 – 180 mg /dl).
   d) Insulin dose should be adjusted (1 – 2 days) to estimate daily requirement of patient.

D. Insulin regimen.

E. Nutritional management.

F. Exercise encouragement.

G. Monitoring.

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Emergency

Basic life support in cardio pulmonary arrest 1997

Aim is to restore spontaneous breathing & circulation (A, B, C)

A. Air way control
1. Open air way:
   - Place on hard flat surface.
   - Pull chin forward by lifting angle on mandible.
   - Open mouth.

2. Clear airway:
   - Remove F.B.
   - Suction of mouth & oropharynx.

3. Maintain patent airway:
   - Oropharyngeal airway.
   - Endotracheal tube.

B. Breathing support (with artificial ventilation)
   - Mouth to mouth (20 C / min).
   - Bag and mask.
   - Endotracheal tube.

C. Circulation support (with cardiac compression)
1. External cardiac massage by 2 bingers.
   compression on lower sternum.
   or „„, heal of hand on lower 1/3 of sternum in a child.
2. Rate 60 – 100 compression / min.
3. Ratio of compression to ventilation (5 : 1).
4. Palpate femoral pulse to see response.
Allergic disease

As regard bronchial asthma ★★★★★

Pulmonary index score for acute asthma 2008

It is a score for assessment of acute asthma composed of 4 symptoms
1. R.R.
2. Wheezing.
3. Use of accessory muscles.
4. Inspiration : expiration ratio

<table>
<thead>
<tr>
<th>Score</th>
<th>Respiratory rate</th>
<th>Wheezing</th>
<th>Inspiration : expiration ratio</th>
<th>Accessory muscles</th>
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<tr>
<td></td>
<td>&lt; 6 years</td>
<td>&gt; 6 years</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>&lt; 30</td>
<td>&lt; 20</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>1</td>
<td>31 – 45</td>
<td>21 – 35</td>
<td>End exp.</td>
<td>+</td>
</tr>
<tr>
<td>2</td>
<td>46 – 60</td>
<td>36 – 50</td>
<td>Entire exp.</td>
<td>++</td>
</tr>
<tr>
<td>3</td>
<td>&gt; 60</td>
<td>&gt; 50</td>
<td>Insp., exp., or none !!!</td>
<td>+++</td>
</tr>
</tbody>
</table>

- Score < 6 ➔ mild.
- Score 6 – 10 ➔ moderate.
- Score > 10 ➔ severe.

Emergency room management of acute asthma ★★★


1. Inhaled B2 agonist (0.15 mg / kg up to 3 doses in first hour).
2. O2 therapy to achieve O2 saturation ≥ 92 %.
3. Oral corticosterone if no immediate response 2 mg / kg / day (prednisolon).
4. For severe exacerbation
   - Continuous B2 agonist inhalation for 1 hour.
   - Ipratropium bromide / 6 hours.
5. For impending R.F. or respiratory arrest ➔ admission to I.C.U.


A. Clinical features and suggestive history

2. Clinical feature
   a) Symptoms
      - Difficulty in breathing, shortness of breathing.
      - Dry cough, tight wheezy chest, tachypnea.
      - Cyanosis in severe cases.
   b) Signs
      - Inspection: increased R.R. – chest indrawing.
      - Palpation: palpable ronchi.
      - Percussion: hyper resonance.
      - Auscultation
        - Diminished air entry.
        - Breath sounds: harsh vesicular with prolonged expiration.
        - Adventitious sounds: sibilent expiratory ronchi.
B. Laboratory testing for allergic disorders
1. Pulmonary function tests.
2. Skin testing.
3. Quantization of total and specific Ig E.
4. Provocation (challenge) testing and elimination diet.

Causes of acute exacerbation 2004
1. Viral infection of respiratory tract.
2. Air pollution (irritants and chemicals).
3. Food allergy.
4. Emotional stress and exercise.
5. Animal contact.
6. Weather changes and cold air.

Definition
It is a clinical diagnosis defined by: increasingly severe asthma not responsive to drugs that are usually effective.
i.e. "acute severe asthma not responding to usual treatment".

Treatment
1. O2 therapy
   • Dose: 2 – 3 L / min.
   • Method: nasal prong or mask.
   • Aim: to keep O2 saturation > 92 % & O2 tension > 70 mmHg.
2. Rapid
   • ABG.
   • CBC.
   • Electrolytes.
   • Chest X-ray.
3. Monitoring (cardiac and respiratory)
   • If H.R. > 160 / min ➔ stop B agonist temporarily and re evaluate heart condition for heart failure.
4. I.V. fluid for rehydration
   • 70 % of maintenance dose.
   • K as B2 agonists ➔ hypo kalemia.
5. Continue B2 agonist
   • Inhalation /20 min or continuous ipratropium bromide 250 – 500 ug / 6 hours.
6. Aminophylline 5 mg / kg
   • I.V. over 20 min / 6 hours if needed.
   • Or ,, 0.75 – 1.25 mg / kg / hr continuous infusion after loading dose.
Aim: to maintain serum level (12 – 15 ug / ml)
Under sample analysis giving before next dose or every 6 hours in .... Infusion.
7. I.V. B2 agonist infusion if no response
   • S.C. terbutaline 0.01 mg / kg (max. 0.3)
   • Or ,, I.V. terbutaline 10 ug bolus 0.6 ug / kg / min continuous infusion).
Dose can be increased by {0.2 ug / kg / min until 6 ug / kg / min} in severe cases.
8. Mehtypredinisolone I.V. (1 – 2 mg /kg / dose for 48 hours then 1 – 2 mg / kg / day).
10. If no response: reevaluate by serial ABG ➔ mechanical ventilation (volume cycled).
Anaphylaxis

**Definition**
Is an acute, life-threatening systemic reaction caused by an IgE-mediated hypersensitivity reaction and characterized by urticaria, acute airway obstruction, and circulatory collapse.

**Etiology**
The most common causes are:

1. **Foods**: Peanuts, tree nuts, milk, eggs, fish, shellfish, seeds, fruits, grains
2. **Drugs**: Penicillins, cephalosporins, sulfonamides, non-steroidal anti-inflammatory drugs, opiates, muscle relaxants, vancomycin, dextran,
3. **thiamine**, vitamin B12, insulin, thiopental, local anesthetics
4. **Hymenoptera venom**: Honey bee, yellow jacket, wasp, hornet, fire ant
5. **Latex**
6. **Allergan immunotherapy**
7. **Exercise**: Food-specific exercise, postprandial (non-food-specific) exercise
8. **Vaccinations**: Tetanus, measles, mumps, influenza, Radio contrast media, immunoglobulin, cold
9. **chemotherapeutic agents**, blood products, inhalants
10. **Miscellaneous**
11. **Idiopathic**

**Clinical features:**
- **onset**: within a few minutes to hours after antigen exposure.
  Systemic manifestations are caused by the release of inflammatory mediators from mast cells and basophils.
- **Skin manifestations**, such as urticaria and angioedema.
- **Respiratory manifestations**, such as edema of the larynx and epiglottis (causing hoarseness and stridor), bronchospasm, hypoxia, nasal congestion, sneezing, and rhinorrhea.
- **Circulatory manifestations**, such as vasodilation, loss of intravascular volume, hypotension, circulatory collapse, arrhythmias, palpitations, and syncope.
- **Gastrointestinal manifestations**, such as vomiting, diarrhea, dysphagia, and abdominal cramps
- **Genitourinary manifestations**, such as urgency.

**Management:**

**Immediate:**
1. Rapid assessment of airway, breathing, circulation; dermatologic examination; mental assessment
2. Epinephrine, 0.01 mg/kg (1: 1000) 1M; repeat every 20 min as needed*
3. Oxygen 100%, secure and maintain airway
4. Start large-bore IV line for venous access and fluids
5. IV isotonic fluids, 20 ml/kg, repeat as necessary
6. Frequent vital signs, cardiac monitor, pulse oximetry
7. Rapid history for acute triggering event, known allergy and anaphylaxis history, current medications, history of asthma symptoms, concomitant medical conditions.

**Sub-acute:**
1. H₁ antagonist, diphenhydramine, 1-2 mg/kg PO, IM, IV
2. Corticosteroids. Administered PO, prednisone, 1 mg/kg, or IV, methylprednisolone, 1-2 mg/kg
3. Nebulized albuterol, 1.25-2.5 mg every 20 minutes or continuously.

**Prevention:**
- Avoidance of known antigen, (e.g., drugs, latex, foods).
- The use of emergency epinephrine self-administration kits, and identification bracelets.

---

**Immune deficiency disorders**
Clinical pictures

1. Category E (perinatally exposed infant): describes (HIV exposed infants) in whom infection has not been
   - Confirmed.
   - Or,„„, excluded.
2. Category N (infected infant) (HIV infected infant) asymptomatic.
3. Category A (mildly symptomatic infections)
   - Parotitis – otitis media.
   - Lymphadenopathy.
   - Recurrent urinary tract infections.
   - HSM.
4. Category B (moderately symptomatic infection)
   - Oral candidiasis & failure to thrive.
   - Anemia & thrombocytopenia.
   - Cardiomyopathy.
   - Lymphocytic interstitial pneumonitis. (pneumonitis).
   - Diarrhea.
5. Category C (severely symptomatic infections) (AIDS defining condition)
   - Recurrent serious bacterial infections.
   - Opportunistic infections.
     - P.Carini pneumonia.
     - Candida esophagitis.
     - Toxoplasmosis encephalitis.
   - Wasting syndrome.
   - Malignancies: lymphoma.
   - Progressive neurologic disease e.g.
     - Developmental delay.
     - Encephalopathy.
     - Paresis.
     - Dystonia.
     - P.N.

Diagnosis of HIV infections

A. Infant born to HIV infected mother (category E)
   1. Serologic testing (ELISA & Western blot): maternal antibodies may persist up to 18 months in exposed infant.
   2. HIV P24 antigen detection
      - Not all infected infant positive.
      - Negative not exclude infection.
      - But, Positive test generally makes diagnosis
   3. HIV culture of blood lymphocytes:
      When available "most reliable diagnostic test) but, negative culture doesn't rule out HIV infection.
B. Infants & children without known perinatal HIV exposure:
   - Presenting symptoms and signs may raise suspicious of infection.
   - Children may present with AIDS defining infections.

General features

A. Also called cell mediated immune deficiency.
   - Results from: abnormalities in T. Cell function.
   - Antibody production may also affected as T. Cells are important for B. Cells differentiation and function.

B. Causative organism (agents): (intra cellular pathoges) e.g.
   - Herps viruses.
   - Mycobacteria.
   - Fungi (candida).
   - Protozoa (toxoplasma).

C. Sites of infection: variable may be
   - Local.
   - Systemic.
   - Or both.

D. Congenital cell mediated immune deficiency include ➔ severe combined immune and isolated defects that affects only cell mediated immunity to one particular pathogen e.g. (chronic mucocutaneous candidiasis).

Examples

1. Severe combined immune deficiency

Clinical pictures
   - Recurrent infections, wasting.
   - Chronic diarrhea, failure to thrive.
   - Graft versus host disease (GVHD).
   - Anemia, alopecia.

Therapy
   - B.M. transplantation.
   - Antibiotics & I.V. (lg).
   - Blood transfusion must be irradiated to guard against GVHD.

2. Defects of purine salvage pathway
   - Adenosine deaminase deficiency (ADA).
   - Purine nucleoside phosphorylase deficiency.

Clinical pictures
   - Recurrent infections, dysostosis.
   - Anemia, MR, adinosine deaminase deficiency.
   - Purine neucloside deficiency.
   - Rib and scapula abnormality in ADA.

Therapy
   - B.M. transplantation.
   - Enzyme replacement therapy.


Clinical pictures
   - Hypo para thyroidism (hypo calcemia).
• Facial abnormalities.
• Cardiovascular abnormalities.
• Infections and mental deficiency (in some patients).
• GIT malformation (some patients).

**Therapy**
Thymus graft or thymosin therapy.

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**Genetics**

**Down syndrome (clinical pictures, genetic types)**

**Genetic types**

1. **Non dysjunctional**: (95%) of children with Down syndrome have trisomy with an extra 21 chromosome present in all cells, total chromosomal count (47).
   Chromosomal non dysjunction during (maternal meiosis) is responsible for 80 – 90% of cases of trisomy 21.

2. **Translocation** (4%): involving 21 chromosome
   - Extra chromosome is attached to chromosome (13 – 14 – 15 – 21 – 22).
   - Parental karyotype analysis (should be undertaken) it revealed apparent with a balanced translocation (20 – 40% of time).

3. **Mosaic** (3%)
   - Some cells display (trisomy 21) but others have a normal karyotype.

**Clinical pictures**

1. Head and neck
   - Skull: small and with flat occipit (brachycephaly).
   - Eye.
     - Lateral upward slanting.
     - Prominent epicanthic fold.
     - Brush field spots in iris.
   - Ears: small with over folding helix.
   - Nose: flat nasal bridge and up turned nose.
   - Tongue: protruded (due to small oral cavity) and fissuring (scrotal and tongue).
   - Teeth: delayed eruption.
   - Neck: short, thick with excess nuchal fat.

2. Trunk ➔ abdomen: protuberant (due to hypotonia of abdominal muscles).

3. Extremities:
   - Hands:
     - Short and broad.
     - Single transverse (Simian) crease in both hands in 50%.
     - Short in curved little finger (clinodactyly).
     - Middle phalanx of little finger is short or absent.
     - Joints are hyper flexible.
   - Feet:
     - Wide gap between bid and 2nd toe.
     - Longitudinal planter crease.

4. Poorly developed genitalia.
5. Delayed puberty.
6. Mental handicap: IQ is variable, range from (20 – 75%), average 50%.
7. New born: may show
   - Prolonged physiologic jaundice.
• Polycythemia.
• Or, transient leukemoid reaction.

**In genetics: what is (Meiosis, carrier, non dysjunction) 1999**

**Meiosis**
Reduction cell division which occur in gametes resulting in haploid number.

**Carrier**
Heterozygous of an autosomal recessive (AR) disease.

**Non dysjunction**
Non separation of two chromosomes during meiosis.

**monosomy**
Absence of a sex chromosome (monosomy X as in Turner's syndrome).

**Trisomy**:
Presence of an extra somatic chromosome (trisomy, as in Down syndrome)
Trisomy syndromes (21, 13, and 18).

**Karyotype:**
The karyotype is an individual's chromosomal constitution.

“Each chromosome is formed of 2 identical chromatids held together by a centromere. The chromosomes are classified according to their length and position of their centromere into 7 groups A, B, C, D, E, F and G together with the pair of sex chromosomes.”
**Growth & Development**

**Q1: Factors affecting growth  11-10*- 08**

**Def.**
It is an increase in size of the whole body or of its separate parts measured in Kg as weight & cm as length due to increase of cells size or number (Multiplication).

**Factors Affecting Growth:**

1. **Genetic or Hereditary Factors:**  (Down ↦ short stature)
2. **Endocrinal Factors:**
   - Linear growth: Growth hormone (Somatotropin)
   - Skeletal maturation: Thyroid hormone
   - Growth in adolescence
     - Boys: Adrenal and testicular androgens
     - Girls: Adrenal androgens and Ovarian estrogen
3. **Nutritional Factors:**
   A- **Nutrition:**
   - Deficiency of energy, protein, minerals & vitamins.
   - Diseases interfering with adequate nutrition e.g. Parasites & chronic diarrhea.
   B- **Nutrition:**
   - Over nutrition causes obesity (Obese children are slightly taller than average).
4. **Socioeconomic Factors:**
   - Poverty & its associated environmental factors (↓ Exposure to sunlight) e.g. Poor hygiene, Poor living Standards.
5. **Psychological Factors** (Stress):
   - Psychological stress can Inhibit growth by altering endocrine function.
6. **Pathological Factors** (Chronic disease):
   - e.g. T.B., Chronic asthma, liver Cirrhosis, Chronic renal failure.
   - If the disease is properly treated before adolescence ↦ accelerated growth rate for a period.

**Q2: Ant. Fontanel examination  2011-02-01-96**

**Size:** 3 x 2 in early months of life & gradually decreases until it closes at 9-18 months.

**Clinical significance:-**
- **Large sized:** Rickets, hydrocephalus & cretinism
- **Delayed closure** (still open at 18 months)
  - General: (MACRO- H):
    - Mongolism - Achondroplasia - Cretinism - Rickets - Osteogenesis imperfecta.
  - Local: ↑ ICT <hydrocephalus>
- **Premature closure** (in early months of life with small head): Microcephaly & Craniostenosis.
- **Depressed:** Dehydration
- **Bulging and tense:** ↑ ICT e.g. hydrocephalus, meningitis.

**Q3: Factors affecting Development  2000**

**Def.** It is Maturation of systems & organs as well as gain of skills & Abilities "Mental, emotional, Social & Motor as well as ability to adapt & assume responsibility.

**Factors affecting development:**

1. **Genetic or Hereditary factors.**
2. **Environmental factors:**
   - Emotions & lack of stimulations.
   - Disorders early in life e.g. "Rubella in 1st trimester of pregnancy, Perinatal Asphyxia, Cretinism (untreated), Malnutrition (severe)". 
### Q4: Assessment of motor development in the 1st 3yrs of life  2010 -2001

<table>
<thead>
<tr>
<th>Age¹ (Mon)</th>
<th>Gross Motor</th>
<th>Fine Motor</th>
<th>Social skills</th>
<th>Language skills</th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>- On Prone: Raise Chest &amp; supports weight with for arm. - On erect: Head support.</td>
<td>- Opens hands spontaneously.</td>
<td>- Smiles appropriately on social contact.</td>
<td>- Coos مقع شديد. Makes sounds like pigeon says &quot;aah, ngah&quot; - Laughs laud</td>
</tr>
<tr>
<td>6</td>
<td>- Sits momentarily, Supported by his arms</td>
<td>- Transfer objects. (From hand to another)</td>
<td>- Show Likes &amp; dislikes. - Recognize mother.</td>
<td>- Bubble &quot;ba, ba&quot; sounds</td>
</tr>
<tr>
<td>9</td>
<td>- Sits alone, back straight - Creeps or crawls محفوف بfinger.</td>
<td>- Grasp objects by: Thumb &amp; fingers. (Pincer Grasp) - Plays (Peek-a-boo) (Hiding the face, then suddenly uncovering it)</td>
<td>- Double Bubble &quot; Dada &amp; mama&quot; sounds</td>
<td></td>
</tr>
<tr>
<td>12 = 1 Y</td>
<td>- Walks supported with One hand held. - Walks alone well at 13 – 15 mon.</td>
<td>- Replaces object to mother on Requests.</td>
<td>- Comes When Called Plays : (simple ball game)</td>
<td>- 1:2 meaningful words.</td>
</tr>
<tr>
<td>18 = 1.5 Y</td>
<td>- Seats himself in a small chair. - Ascend stairs With: One hand held.</td>
<td>- Build tower of 3 cubes. - Points to body parts (On request). - Feeds with spoon by him.</td>
<td>- Imitates &quot;Mimics actions of other&quot;.</td>
<td>- At least 6 words.</td>
</tr>
<tr>
<td>36 = 3 Y</td>
<td>- Tricycle (Ride &amp; pedal). - Climb up stairs well. - Jumps on spot.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>60 = 5 Y</td>
<td>- Jump on one foot. - Walks Heel to Toe along line.</td>
<td>- Draws a man (6 parts) With: pencil.</td>
<td>- Chooses own friends. - Dramatic group play.</td>
<td>- Fluent Speaker. - Asking about: Words &amp; Things meaning</td>
</tr>
</tbody>
</table>

### Causes

1. Familial Predisposition.
2. Environmental: emotional deprivation & lack of stimulation by parents.
3. Nutritional: severe protein energy malnutrition (PEM)
4. Systemic: Congenital heart disease. (CHD)
5. CNS: Cerebral palsy, Mental handicap
7. Muscular: Myopathies.
8. Neurological: Neuropathies, poliomyelitis
9. Perceptual: Blind child

### Q5: Delayed sitting   2001

1. Familial Predisposition.
2. Environmental: emotional deprivation & lack of stimulation by parents.
3. Nutritional: severe protein energy malnutrition (PEM)
4. Systemic: Congenital heart disease. (CHD)
5. CNS: Cerebral palsy, Mental handicap
7. Muscular: Myopathies.
8. Neurological: Neuropathies, poliomyelitis
9. Perceptual: Blind child

### Q6: 1ry inability to walk at 2yrs(Limping)  2003 - 97

1. **Painful causes**
   a) Trauma: Fracture.
   b) Infection: T.B.
   c) Inflammation: Rh. Diseases.
   d) Malignancy: Leukemia & Lymphoma.
   e) Hematology: SC&A hemophilia
   f) Orthopedic: Avascular necrosis.
   g) Metabolic: Lipid storage .

2. **Painless causes**
   a) Neurological disorders: Poliomyelitis
   b) Muscle disorders: Muscle dystrophy.
   c) Joint disorders: Congenital hip dislocation.
   d) Bony disorder: Leg length discrepancy.

3. **Others**
   a) Psosas abscess    b) Synovitis.   c) F.M.F.   d) Acute inguinal lymphadenitis.
### Warning signs of poor development during 1st y of life

<table>
<thead>
<tr>
<th>Age</th>
<th>sign</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 W</td>
<td>- Poor feeding</td>
</tr>
<tr>
<td></td>
<td>- Hypotonia</td>
</tr>
<tr>
<td></td>
<td>- Abnormal Posture</td>
</tr>
<tr>
<td>6 W</td>
<td>- No Face Regard.</td>
</tr>
<tr>
<td>3 m</td>
<td>- No Smiling responding.</td>
</tr>
<tr>
<td></td>
<td>- Clenched hands.</td>
</tr>
<tr>
<td>4 m</td>
<td>- Poor head control.</td>
</tr>
<tr>
<td></td>
<td>- No interest in own hands.</td>
</tr>
<tr>
<td>6 m</td>
<td>- Head lag on pull up.</td>
</tr>
<tr>
<td></td>
<td>- Persistent hand play.</td>
</tr>
<tr>
<td></td>
<td>- Persistent Moro's reflex.</td>
</tr>
<tr>
<td>9-10 m</td>
<td>- Can’t sit alone.</td>
</tr>
<tr>
<td></td>
<td>- Can’t reach / transferring.</td>
</tr>
<tr>
<td></td>
<td>- Baulks on solids.</td>
</tr>
<tr>
<td>11-12 m</td>
<td>- Not mobile.</td>
</tr>
<tr>
<td></td>
<td>- No pincer grip.</td>
</tr>
<tr>
<td></td>
<td>- Poor attention.</td>
</tr>
<tr>
<td>12-15 m</td>
<td>- Not crawling.</td>
</tr>
<tr>
<td></td>
<td>- Persistent mouthing.</td>
</tr>
<tr>
<td></td>
<td>- Not responding to Speech.</td>
</tr>
<tr>
<td>18 m</td>
<td>- Not walking.</td>
</tr>
<tr>
<td></td>
<td>- Clumsy hand.</td>
</tr>
<tr>
<td></td>
<td>- No useful speech.</td>
</tr>
<tr>
<td></td>
<td>- No sustained interesting in toys.</td>
</tr>
<tr>
<td>2 Y</td>
<td>- Over activity. Aimless (little constructive play).</td>
</tr>
<tr>
<td>3-4 Y</td>
<td>- Doesn’t follow clear Orders E.g. &quot;Put this on the table&quot;.</td>
</tr>
<tr>
<td></td>
<td>- doesn’t know Pictures of common things.</td>
</tr>
<tr>
<td></td>
<td>- can’t use a pencil (scribble / draw a line).</td>
</tr>
<tr>
<td>4-5 Y</td>
<td>- Can’t tell his full Name.</td>
</tr>
<tr>
<td></td>
<td>- Doesn’t use Plural’s.</td>
</tr>
<tr>
<td>6 Y</td>
<td>- Can’t know Colors. (Yellow, Green, Red &amp; Blue).</td>
</tr>
</tbody>
</table>

### Q8: Developmental screening tests 2008

**Def.:**
These are simple preliminary tests performed by the physician to determine if a child should be referred for more extensive examination for developmental delay so these are not intended for final diagnosis of Mental Retardation which include:

**Advantages:** Ease of administration & demonstrated reliability.

**Types**
1. **Gesell figures:** as shown in the following figures:
2. **Denver Developmental Screening Test (DDST):** (Best screening method)
   - It includes the 4 major development fields (Gross & fine motor, social & language).
3. **Good enough draw a person test:**
   - **Technique:**
     - Child (3 years or older) is supplied with: pencil + sheet of blank paper and instructed to draw a person without any additional instructions.
   - **Interpretation:**
     - 1 item (e.g. head present, legs present, arms present...) = 1 point
     - every 4 points = 1 year
     - Mental age score = items/4 + basal age (3 years)"
   - *E.g.* if drawing show 11 items are present → Mental age score = \( \frac{11}{4} + 3 = 5 \frac{3}{4} \) years
**Definition:**
It is a persistent eating of non-nutritive substances for at least one month e.g. Earth (Geophagia), Ice (Pagophagia)
- It must be developmentally inappropriate behavior.
- It must be differentiated from the practice of mouthing inanimate (not living inorganic) objects which is normal between 6-12 months of age.

**Aetiology:**
- Associated circumstances.
- Parental neglect and deprivation.
- Nutritional deficiencies "Iron, Zinc"
- Higher incidence occurs in the relatives of people with this phenomenon.
- Mental retardation.

**Incidence:**
- Occur in 10 to 32 % of children between 1 - 6 years of age.

**Management:**
1. No definitive treatment, most ttt aimed at education & behavior modification:
   - Determine the cause whenever possible.
   - Treatment of the cause.
   - Eliminate lead if it is present in the surroundings.
2. TTT of complications as lead poisoning.
3. Several behavioral techniques have been used with some effect as mild electric shock, unpleasant noise, and emetic drug.
   - Positive and negative reinforcement can be used.
### Definition

It is marked abnormal development in social interaction, restricted activities & interests

#### C/P:

| a. | Boundedness: Many of them have failure of lateralization at proper age. |
| b. | Higher incidence of: |
| c. | Febrile seizures. |
| d. | React differently to illness: as they may not have elevated temperature with infectious diseases, may not complain of pain, may not show the malaise of ill child. |

#### C: Qualitative impairments in social interaction

| 1) | Language deviance & delay. |
| 2) | Make little use of meaning in their memory. |
| 3) | Their conversations are not characterized by reciprocal responsive interchange. |
| 4) | Autistic children say more than they understand |
| 5) | Words and even sentences may drop. |
| 6) | May use a word once and then not use it again for a week, a month or years. |
| 7) | Their speech contains echolalia and stereotyped phrasing |
| 8) | Pronominal refer (You) instead of (I). |
| 9) | Peculiar voice quality and rhythm. |
| 10) | Usually never have useful speech. |
| 11) | Read without any comprehension of whatever. |

#### b: Qualitative impairments in communication & language

| a. | In the 1st yr, much of the exploratory play is absent. |
| b. | Toys and objects are often manipulated in an unintended way, with little variety creativity & imagination. |
| c. | They can't imitate whatever. |
| d. | Ritualistic & compulsive phenomena are common in early and middle childhood. |
| e. | They often spin, bang, line up objects and become attached to inanimate objects. |
| f. | They are resistant to transition and change, when the reverse was the routine may result in panic or temper tantrums. |

#### d: Instability of mood & affect: |

They usually exhibit sudden mood changes, with burst of laughing or crying without apparent reason

#### e: Other behavioral symptoms

| a. | Over-responsive or under-responsive to sensory stimuli |
| b. | Many have lightened pain threshold. |
| c. | They seem to enjoy music. |
| d. | Hyperkinesia, hyperactivity, aggressiveness, temper tantrum, self-injurious behavior, short attention span, insomnia, feeding and eating problems and enuresis. |
| e. | Intellectual functioning(IQ) |
| a. | 40 % below 50. |
| b. | 30 % at 50-70. |
| c. | 30 % at or above 70. |
| d. | Some have splinter functions or islets of precocity, music, play & math. |

### Management

- Socially acceptable and prosaically behavior.
- Odd behavioral symptoms.
- Aid in the development of verbal and non-verbal communication.
- Children with mental retardation need more simplistic behavioral intervention

---

**Q2: Autistic disorders (C/P & D.D) 2007**
Human Genetics

Q1: Down $ (C/P & Genetic types) 2001-96
(Trisomy of chromosome 21)

Causes (Genetic types)

1- Regular Down: (92%)
   - Chromosomal non-disjunction during maternal meiosis
   - Total number of chromosomes is 47 instead of 46.
   - Common in elderly mothers>35 yrs. (47xy +21)

2- Translocation Down (5%)
   - Translocation of chromosome 21 occurs on any chromosome specially (13, 14, 15, 21 or 22).
   - Total chromosome count is 46 but functionally the child has 47 chromosomes.
   - Common in young mothers with liability for translocation.

3- Mosaic Down (3%)
   - Chromosomal non-disjunction during 1st or 2nd mitotic division of the zygote.
   - 2-3 cell lines are present.

Clinical pictures

- Head & neck
  - Skull: Small with flat occiput (Brachycephaly)
  - Eyes: Lateral upward slanting, Prominent epicanthic folds, Brush field spots on iris
  - Ears: Small with over folding helix
  - Nose: Flat nasal bridge, upturned nose.
  - Mouth
    - Tongue: Protruded due to small oral cavity $\rightarrow$ fissuring (scrotal tongue)
    - Teeth: Delayed eruption
  - Neck: Short, thick, with excess nuchal fat.

- Heart: High incidence of congenital HD (Cushion defect $\rightarrow$ VSD)

- Abdomen: Protuberant d.t. hypotonia of abdominal muscles.

- Extremities:
  - Hands:
    - Short & broad, Single transverse (Simian) crease in both hands in 50%.
    - Short incurved little finger (Clinodactly)
    - Middle phalanx of little finger is short or absent.
    - Joints are hyper flexible
  - Feet
    - Wide gap between the big and 2nd toe.
    - Longitudinal plantar crease.

- The genitalia are poorly developed & puberty is delayed.

- Mental handicap: The IQ is variable, ranges from 20-75%
**Nutrition**

**Breast feeding**

**Q1: Physiological mechanisms of lactation**

2011

1- **Maternal reflexes:**
   a. **Prolactin reflex (Milk secreting reflex):**
      
      Suckling \(\rightarrow\) Nipple \(\rightarrow\) Vagus nerve \(\rightarrow\) Hypothalamus \(\rightarrow\) Anterior pituitary \(\rightarrow\) Prolactin hormone \(\rightarrow\) **Stimulation of milk secretion.**
   
   b. **Oxytocin reflex (Milk ejection or let-down reflex):**
      
      Suckling \(\rightarrow\) Nipple \(\rightarrow\) Vagus nerve \(\rightarrow\) Hypothalamus \(\rightarrow\) Posterior pituitary \(\rightarrow\) Oxytocin hormone \(\rightarrow\) Contraction of the smooth muscles around the acini \(\rightarrow\) **Milk ejection.**

2- **Infant’s reflexes:**
   a. **Rooting reflex:**
      
      Turning of the head to the side in which the nipple is felt *then* the infant draws it into his opened mouth then to oropharynx by action of buccal muscles.
   
   b. **Suckling reflex:**
      The tactile stimulus caused by the nipple and areolar tissues filling the mouth \(\rightarrow\) milking action by the tongue against the hard palate.
   
   c. **Swallowing reflex:**
      This enables the baby to ingest the milk that is obtained by suckling and allows interruption of breathing to prevent choking during swallowing.

**Q2: Advantage of breast feeding**

2009-06-02-01

A. **To the mother:**
   1- No **Cost** & **Economic- Inexpensive**
   2- **Convenient** & **Available at anytime, anywhere, suitable, fresh**
   3- Natural method of **Contraception**
   4- **Incidence of Cancer breast.**

B. **To both mother& infant:**
   - Helps emotional security of infant & mother and establish strong psychological relationship between them.
   - The infant is afforded close & comfortable physical & sensual contact essential for his emotional development.

C. **To the infant:**
   i. **General:**
      - **Sterile** & free from contamination
      - **Adequate amount (Quantity)**
      - **Adequate composition (Quality)**
   
   ii. **Composition:**
      Breast milk is nutritionally superior to any alternative & is easily digestible.
   
   iii. **Anti-allergic:**
      Breast milk gives partial protection against some allergic conditions as infantile eczema.
   
   iv. **Anti-infective properties (Biological):**
      1- **Cellular:** (Human milk is alive fluid contains)
         - 4 million cells per ml colostrum.
         - The important cells are **WBCs Imp.** \(\rightarrow\) Infant protection against pathogens.
   
      2- **Humoral:**
         High amount of **secretory IgA** \(\uparrow\uparrow\) colostrum & \(\downarrow\downarrow\) in mature milk.
         - **Imp.** \(\rightarrow\) GIT & respiratory protection against viruses & bacteria.
   
      3- **Others:**
         - **Lactose (bifidus factor):** inhibits growth of E-coli and vibrio-cholera in colon by changing to lactic acid so growth of lactobacillus bifidus causing diarrhea).
         - **Lysozyme:** Lyses of bacteria CW (Bactericidal).
         - **Lactoferrin:** deprive bacteria from the growth factors (iron) by binds them (Bacteriostatic).
**Problems and Difficulties during Breast Feeding**

**a) Related to the mother (2 Nipple - 3 Milk - 4 Mother conditions)**

- **Fissured (Cracked) Nipple**
- **Retracted (flat) Nipple** = Non-projectile
- **Insufficient (Scanty) breast milk**
- **Delayed Appearance of Milk**
- **Mother Breast Engorgement** (Tender full breast)
- **Contraceptive & breast feeding**
- **Pregnancy & breast feeding**
- **Twins & breast feeding**
- **Work & breast feeding**

**b) Related to the Infant**

- Suckling in a poor position
- Inability to Suckle or Refusal to Breastfeed
- Regurgitation after breast milk

---

**Q3: Fissured (Cracked) Nipple 2002**

When the infant is not taking enough of the areola inside his mouth while suckling

**Cause**

- Improper position
- Milk engorgement
- Retracted nipple

**Correct the position (Change position)**

- The part of the nipple which is subjected to repeated trauma from suckling is allowed to rest and heal.

**Local application of hydrous lanolin or panthenol**

To soothe the sore nipple may help.

**N.B:** Wetting the nipple with breast milk is sometimes helpful.

**Let the baby suckles first from the least affected breast**

d.t. the initial suckling is the strongest and thus the most painful.

**Prevention:**

- Proper antenatal care
- Ensure proper feeding position
- Early treatment of engorgement.
Q4: Insufficient (Scanty) breast milk  2010-95

Cause
- Maternal malnutrition, diseases or psychic troubles.
- Early administration of sugary fluids → ↓ Suckling.
- Maternal conditions<Work – Contraception – Pregnancy – Twins>

N.B: The most valuable sign that the mother is producing sufficient milk is adequate → Weight gain of the infant as shown by follow-up of the weight curve.

TTT
1. Reassure the mother.
2. Advise her to increase frequency of feeding.
3. Follow up, weight growth of the baby on the chart.
4. Complementary feeding if true insufficient breast milk (زبادى او لبن صناعى)

Certain rules should be fulfilled with complementary feeding to avoid inhibition of breast milk:
- Use cup and spoon, sucking from a bottle.
- Both breasts should be given and completely emptied at each feed.
- The feed should always be immediately after breastfeeding
- The feed should not be too sweet.
- The amount of the complementary feed is determined by trial & errors

Q5: Signs of suckling in good position 2008-02-99
1. The baby's whole body is facing his mother & his face is close to the breast.
2. His Chin is touching the breast.
3. His Cheeks are round, and not pulled in.
4. His Mouth is wide open.
5. His Lower lip is curled outwards
6. You see more areola above the upper lip than below the lower lip.
7. You can see or hear the baby swallowing.
8. The baby takes slow deep sucks
9. The baby is relaxed, happy & satisfied at the end of a feed.
10. The mother does not feel any pain.

Q6: Inability to Suckle or Refusal to Breastfeed  2010*

Causes
1. Improper position during feeding.
2. Sore mouth due to thrush or ulcer.
3. Giving the baby bottle feeding.
4. Blocked nose from nasal catarrh.
5. Pendulous breasts → obstructing the nostrils.
6. Anorexia due to infection.
7. Sleepy feeder: this is common in the first few days after birth particularly in premature and following heavy maternal analgesia.
8. Scanty breast milk → crying & refuse breast feeding.
These include helping & encouraging mothers to breastfeed & prolong lactation for at least 2 yrs.

<table>
<thead>
<tr>
<th>Infant</th>
<th>Permanent(Absolute) contraindication</th>
<th>Temporary contraindication</th>
</tr>
</thead>
<tbody>
<tr>
<td>a. Inborn errors of Digestion:</td>
<td>Lactose intolerance (CHO)</td>
<td>a. Acute maternal illness as septicemia, typhoid fever, pneumonia until treated</td>
</tr>
<tr>
<td>b. Inborn errors of metabolism:</td>
<td>Galactosemia (CHO) &amp; Phenyl ketonuria (P.t.n)</td>
<td>b. Local breast disease: as bilateral cracked nipple, mastitis, breast abscess</td>
</tr>
<tr>
<td>c. Mental &amp; neurological diseases:</td>
<td>as epilepsy, mania, mental retardation &amp; psychosis.</td>
<td>c. Eclampsia.</td>
</tr>
</tbody>
</table>
| d. Maternal hepatitis B disease: | Unless the new born receive hepatitis B immune globulin & HB vaccine at birth, and then completes the HB vaccination schedule | d. Active tuberculosis
Mother is treated. Infant receives INH and is repeatedly tested with tuberculin test.
- If tuberculin is still negative after 3-4 months of age.
- Mother response to treatment is satisfactory. *** INH is discontinued |
| e. Maternal HIV/ AIDS disease: | Breast feeding is not recommended if a safe alternative is available                             | e. Chicken pox
Zoster immune globulin (ZIG) is given to non-infected neonate. The neonate is separated from mother until she is no longer infectious. |
| f. Intake of dangerous drugs | which are secreted in milk in considerable amounts
- Anticoagulant, anticancer, tiouracil, ergotamine & lithium.
- Radioactive substances, Cocaine, heroin, marijuana. | f. Syphilis
Infant of syphilitic mother is allowed to fed from his mother because he is syphilitic but it’s C.I from other woman to avoid infecting her. |
|                         |                                                                                                    | 1. Acute infant illness: Pneumonia, Persistent vomiting. |
|                         |                                                                                                    | 2. Complete cleft palate, micrognathia |

Q7: Contraindication of Breast Feeding 2001

Q8: Promotion of Breastfeeding 2012

<table>
<thead>
<tr>
<th>Antenatal period</th>
<th>Natal period</th>
<th>Post-neonatal period</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Nipple care:</td>
<td>Avoid unnecessary procedures e.g Episiotomy → Fatigue, anxiety &amp; pain.</td>
<td>Avoid estrogen containing oral and injectable contraceptives.</td>
</tr>
<tr>
<td></td>
<td>Avoid deep anesthesia → Sedated newborn unable to suckle properly.</td>
<td>No solids or semisolids supplements in the first 4 months.</td>
</tr>
<tr>
<td></td>
<td>Avoid bottle feeding → Nipple confusion or refusion.</td>
<td>Mothers with insufficient milk or other problems should be given the extra support, advice &amp; encouragement.</td>
</tr>
<tr>
<td></td>
<td>Start breastfeeding within the first half hour after delivery.</td>
<td>Breastfeeding should be prolonged as much as the mother and child wish preferably till 2 years with proper supplementation from 6 months.</td>
</tr>
<tr>
<td></td>
<td>Allow rooming-in to allay mother’s anxiety and permit frequent suckling.</td>
<td></td>
</tr>
</tbody>
</table>
Q9: Modifications of buffalo milk 2000

<table>
<thead>
<tr>
<th></th>
<th>Milk</th>
<th>Water</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st 3 months</td>
<td>1 Part</td>
<td>1 Part</td>
</tr>
<tr>
<td>3-6 months</td>
<td>2 Part</td>
<td>1 Part</td>
</tr>
<tr>
<td>After 6 months</td>
<td>No dilution</td>
<td></td>
</tr>
</tbody>
</table>

Q10: How you feed an infant aged 3 months weighing 5 kg with powdered milk what are you going to avoid contamination?

The amount of formula needed is calculated according to weight as follows:
- Daily needs = 150 ml formula/kg of body weight! day.
- The calculated amount is divided by the number of feeds per day (usually 6 feeds).

Methods

1. **Amount of feed (ml) = 150 ml x Body weight in Kg Number of feeds per day**
   - For a baby weighing 5 kg the daily needs is: 150 x 5 = 750 ml/day.
   - We give 6 feeds/day, the amount given in each feed = 750/6 = 125 ml.

2. Another less accurate method:
   **Amount of feed in ml = Weight of baby in GRAMS/ 36**
   - For a baby weighing 5 kg, the amount per feed = 5000 / 36 = 139 ml.

Teach the mother about the following:
- Clean the feeding bottle by special brush using soap and water then sterilize by boiling for 5-10 minutes and keep covered until reused.
- Any formula left over should be discarded.

Protein Energy Malnutrition (PEM)

Q11: Welcome classification 2001

It depends on body weight for age & the presence or absence of edema.

<table>
<thead>
<tr>
<th>Body wt 60-80%</th>
<th>Body wt &lt; 60 %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Without Edema (Underweight)</td>
<td>Without Edema (Marasmus)</td>
</tr>
<tr>
<td>With Edema (Kwashiorkor)</td>
<td>With Edema (Marasmic Kwashiorkor)</td>
</tr>
</tbody>
</table>
### Clinical picture

#### Q11: Kwashiorkor (C/P - sure signs - diagnosis & dietetic TTT) 2003-94-92-91

<table>
<thead>
<tr>
<th>Chch</th>
<th>1- Growth failure</th>
<th>2- Mental changes</th>
<th>3- Muscle wasting</th>
<th>4- GIT troubles</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chch</td>
<td>• Weight at 60 - 80 % of standard age.</td>
<td>• The patient → Apathetic, disoriented with his surroundings.</td>
<td>• Weak, thin atrophic muscles</td>
<td>• Soft pitting, bilateral, painless.</td>
</tr>
<tr>
<td></td>
<td>• Failure to gain weight → weight loss.</td>
<td>• Head &amp; height circumference may be affected lastly.</td>
<td>• Mostly affected the biceps &amp; the triceps.</td>
<td>• Starts early at the dorsum of feet and legs → Generalized.</td>
</tr>
<tr>
<td></td>
<td>• Wasted → Weight for length.</td>
<td>• SC is preserved.</td>
<td>• SC is preserved.</td>
<td>• Bouncy checks with moon face appearance.</td>
</tr>
<tr>
<td></td>
<td>• Head &amp; height circumference may be affected lastly.</td>
<td>• Muscle wasting is detected by</td>
<td>• Muscle / fat ratio.</td>
<td>• Not associated with ascites &amp; pleural effusion</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Skin folds thickness.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

#### Cause

1. Variable (Nonessential) features

<table>
<thead>
<tr>
<th>Chch</th>
<th>1- Hair changes</th>
<th>2- Skin changes</th>
<th>3- Hepatomegaly</th>
<th>4- GIT troubles</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Color: lighter progressively, black to dark brown, and light brown to orange to yellow.</td>
<td>• Shape: Darkness &amp; hyperpigmentation followed by desquamation &amp; depigmentation.</td>
<td>- Soft to firm &amp; smooth with rounded border.</td>
<td>• Anorexia, vomiting, diarrhea or steatorrhea</td>
</tr>
<tr>
<td>Chch</td>
<td>• Texture: soft &amp; easily broken.</td>
<td>• Site: in irritated areas (hemorrhage → atack - back)</td>
<td>- Palpable liver is common.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Distribution: sparse.</td>
<td>• Called</td>
<td>- Fatty infiltration in Kwo is reversible &amp; never leads to hepatic cell necrosis or cirrhosis)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Attachment: - Loose easily pickable without pain.</td>
<td>•aky paint dermatitis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>[diagnostic sign]</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>d.t. repeated attacks &amp; affection of hairs in segmental manner which → bands of light color alternating with bands of darkening in the same hair.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cause</th>
<th>1- Hair changes</th>
<th>2- Skin changes</th>
<th>3- Hepatomegaly</th>
<th>4- GIT troubles</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Sulphur containing aa</td>
<td>• Nicotinic acid</td>
<td>• Fatty infiltration results from:</td>
<td>• Anorexia, vomiting, diarrhea or steatorrhea</td>
</tr>
<tr>
<td></td>
<td>• Tyrosine which is needed for melanin pig. synthesis</td>
<td>• Essential fatty acid, VITAMIN A &amp; zinc</td>
<td>• lipotropic factors</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Fat mobilization from adipose tissue</td>
<td></td>
</tr>
</tbody>
</table>

#### Variable (Nonessential) features

<table>
<thead>
<tr>
<th>Chch</th>
<th>1- Anemia</th>
<th>2- Infection</th>
<th>3- Vitamin deficiency</th>
<th>4- Hgic manifestation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chch</td>
<td>1- Microcytic hypochromic anemia:</td>
<td>Most common infections are: gastro enteritis, pneumonia, OM, T.B., UTI &amp; Candida infections</td>
<td>- Vitamin def: Xerosis, keratomalacia, night blindness, corneal opacities.</td>
<td>• Purpura</td>
</tr>
<tr>
<td></td>
<td>2- Normocytic normochromic anemia</td>
<td></td>
<td>- Vitamin def: Glossitis, angular stomatitis.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3- Megaloblastic anemia:</td>
<td></td>
<td>- Vitamin def: Scurvy, bleeding gums.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- Vitamin def: Rickets.</td>
<td></td>
</tr>
</tbody>
</table>

| Cause | 1- Iron deficiency anemia. | 2- Infection & hypoproteinemia | 3- folic acid & vit. B12 deficiency. | |
|-------|----------------|----------------|------------------|
|       | • Epithelial cells lining of the GIT & Respiratory Tract usually becomes unhealthy → invasion by micro-organisms. | | |
|       | | • Impaired immunity. | |

### Complication of Kwo (4H + ABCDEF)

1) Hypoglycemia: d.t. enzymatic deficiency, liver affection & lack of anti-insulin H.  
2) Hyperthermia: d.t. heat loss d.t. edema & heat production d.t. thyroid function  
3) Hypokalemia: d.t. usually present from start ↑ by IV fluid &tt of acidosis  
4) Hypomagnesaemia → tetany  
5) Nephemic HF: other causes of HF in Kwo (Weak myocardium & H2o-Na retention).  
6) Peeding (Hge): d.t. Hypoprothrombinemia, cap. Frailty or Dic.  
7) Urinary excretion: d.t. vit. A def. → Night blindness  
8) Arrhean: d.t. lactase def. vit. def. and infection → dehydration  
9) Electrolytes disturbance: ↑ Na & Ca  
10) frequent infection: Skin, GIT, Respiratory, oral moniliasis, T.B & Septic shock.
Investigation of Kwo:

a- **Plasma proteins:**
   - ↓ Total serum proteins.
   - ↓ Serum albumin (<2.5 gm/dl)
   - ↑ Gamma globulins (d.t. infection)
   - Reversed albumin / globulin(A/G) ratio (N = 4.5/2.5).
   - ↓ Essential aa.
   - ↑ Non-essential aa.
   - ↓ Essential aa/non-essential aa ratio (N = 2).

   **Special proteins:**
   - ↓ Transferrin (used to transfer iron).
   - ↓ Ceruloplasmin (used to transfer copper).
   - ↓ Haptoglobin.

b- **Blood sugar:**
   - CHO Hypoglycemia is common complication.

c- **Serum lipids:**
   - ↓ cholesterol which returns to normal soon after ttt up FFA (d.t. up CHO diet)


d- **Serum Minerals, vitamins & electrolytes**
   - ↓ Mg, Fe, Cu, Zn and all other trace elements.
   - ↑ Na level but water retention is excessive ***Dilutional hyponatremia***.
   - ↓ K level mainly d.t. vomiting & diarrhea
   - ↓ Ca level.

e- **Water**
   - ↑ Total body water (intra and extra cellular).

f- **CBC**
   - Anemia.
   - Leucocytosis may be leucopenia.
   - Thrombocytosis.

g- **Others**
   - Tuberculin test.
   - Chest X-ray.
Q11: Marasmus (Aetiology & prevention) 2009

A. Socio-economic causes: Ignorance, poverty, depression.

B. Dietetic errors (Nutritional- Primary Marasmus):
   - **Quantitative disorders:**
     - Scanty breast milk (in amount or number of feeds).
     - Small milk amount in artificial feeding.
     - Insufficient calories in diet of older children
   - **Qualitative disorders:**
     - Prolonged breast feeding without supplementation (delayed weaning)
     - Over dilutional formula in artificial feeding.
     - Cow’s milk: Allergy- Diarrhea – Vomiting – DM<36%

C. Non-dietetic errors (Secondary Marasmus):
   1) GIT causes:
      a) Failure to assimilate food
         - Mal-digestion e.g. Cystic fibrosis of pancreas
         - Mal-absorption e.g. Mal-absorption syndrome.
      b) Loss of food
         - Recurrent attacks of diarrhea or chronic diarrhea d.t. gastroenteritis
   2) Infections:
      - Recurrent acute or chronic infections: as T.B., pyelonephritis, Chronic suppurative lung disease.
   3) Metabolic disorders:
      - Renal tubular acidosis.
      - Urea cycle defects
      - Galactosemia.
      - Fructosemia.
      - Amino acid defects
   4) Malignant
      - Neurolblastoma
      - Wilm's tumor
   5) Endocrinal disorders
      - Juvenile D.M.
      - Adrenal insufficiency.
   6) Congenital abnormalities:
      - C.N.S: Defective cerebral development.
      - C.V.S: Fallot’s tetralogy, V.S.D.
      - Chest: Congenital interstitial fibrosis.
      - G.I.T: Congenital pyloric stenosis, cleft lip and palate.
      - Liver: Congenital hepatic cirrhosis.
      - Renal: Renal agenesis, obstructive uropathy.
   7) Psychological problems: Maternal deprivation syndrome.
**Growth failure**
- At first, there is failure to gain wt → loss of wt occurs.
- Wt < 60% of the ideal wt for age.

<table>
<thead>
<tr>
<th>Growth failure</th>
<th>Loss of subcutaneous fat</th>
<th>Muscle wasting</th>
<th>Edema</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1&lt;sup&gt;st&lt;/sup&gt; degree:</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Loss of SC fat in the abdominal wall.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>2&lt;sup&gt;nd&lt;/sup&gt; degree:</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Loss of SC fat in limbs, buttocks</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>3&lt;sup&gt;rd&lt;/sup&gt; degree:</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Loss of SC fat in face &amp; buccal pad (senile face)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Hair changes**
- Loose, thin, wrinkled
- Inelastic
- Thrown into folds esp. medial side of thigh

**Skin changes**
- Anorexia.
- Constipation d.t. lack of food intake.
- Diarrhea d.t. gastroenteritis & malabsorption.

**Anemia**
- Manifested by marked pallor

**Infection**
- Pneumonia, Gastroenteritis, OM, U.T.I & T.B

**GIT manifestation**
- The same as KWO

**Hepatomegaly**
- As Kwo +
  - Atrophic ulcers over bony prominence (Pressure sore)
  - Muscle fibrosis in advanced cases
  - Oedema (Marasmic Kwo)

**Complication**
- To detect the causes in cases of 2<sup>nd</sup> marasmus & to detect the complications.

<table>
<thead>
<tr>
<th>Investigation</th>
</tr>
</thead>
<tbody>
<tr>
<td>CBC:</td>
</tr>
<tr>
<td>RBCs: Anemia (all types of anemia can be found).</td>
</tr>
<tr>
<td>WBCs: Leucocytosis or leucopenia.</td>
</tr>
<tr>
<td>Platelets: Thrombocytopenia.</td>
</tr>
<tr>
<td>Total proteins &amp; serum albumin:</td>
</tr>
<tr>
<td>Slightly reduced.</td>
</tr>
<tr>
<td>Urine analysis:</td>
</tr>
<tr>
<td>Culture in case of U.T.I.</td>
</tr>
<tr>
<td>Glucosuria in case of D.M.</td>
</tr>
<tr>
<td>Stool analysis:</td>
</tr>
<tr>
<td>For parasites or steatorrhea.</td>
</tr>
<tr>
<td>Stool PH (↓ PH = lactase deficiency)</td>
</tr>
<tr>
<td>Stool PH (↓ PH = lactase deficiency)</td>
</tr>
<tr>
<td>Other:</td>
</tr>
<tr>
<td>Chest X-ray: For bronchopneumonia or congenital heart disease.</td>
</tr>
<tr>
<td>Tuberculin test.</td>
</tr>
<tr>
<td>Intestinal biopsy: If there is malabsorption.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Kwo</th>
<th>Marasmus</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein malnutrition</td>
<td>Caloric malnutrition</td>
</tr>
<tr>
<td>Wt 60 – 80%</td>
<td>Wt &lt; 60%</td>
</tr>
<tr>
<td>Edema</td>
<td>NO edema</td>
</tr>
<tr>
<td>Mental apathy</td>
<td>Alert irritable</td>
</tr>
<tr>
<td>Preserved SC</td>
<td>Loss of SC fat</td>
</tr>
<tr>
<td>Moon face</td>
<td>Senile face</td>
</tr>
<tr>
<td>Skin &amp; Hair changes</td>
<td>Skin changes only</td>
</tr>
<tr>
<td>Hepatomegaly</td>
<td>NO hepatomegaly</td>
</tr>
<tr>
<td>Poor appetite</td>
<td>Good appetite</td>
</tr>
</tbody>
</table>
Treatment:

Medical Treatment:-

i. Treatment of dehydration: (Golden ttt for dehydration)

1. Mild dehydration
   - ORS therapy
     ✓ Amount: 70-100 ml/kg over 12 hours.
     ✓ At the first 2 hours, the patient receives 10 ml/kg and the remaining amount given over the following 10 hours.
     ✓ Add 50-100 ml after each watery stool.

2. Severe dehydration
   - I.V. fluids:
     ✓ Ringers lactate + glucose 5% in a percentage of 1:1 & add 2.5 ml of 15% kcl for each 1 litter.
     ✓ Amount: 100 ml/kg over 12 hours.

b. Treatment of infections:
   - Appropriate antibiotics even if the signs of infection are not present.
   - In manifest infection: according to culture and sensitivity.

c. Treatment of electrolyte disturbances:
   - Hypoglycemia: Glucose 10% 2 ml/kg I.V. and regular feeding.
   - Hypocalcemia: Ca gluconate 10% slowly I.V. (2ml/kg).
   - Hypokalemia: Add K to the I.V. fluids (2mEq/kg).

d. Treatment of hypothermia:
   - Proper wrappings.
   - Put baby under radiant warmth.

e. Treatment of anemia: If severe and threatening the life we give:
   - Fresh blood in amount of 20 ml/kg.
   - Fresh packed RBCs in case of anemic heart failure in amount of 5-10 ml/kg.
   - Fresh frozen plasma in amount of 10 ml/kg in case of bleeding tendency & Prothrombinemia

ii. Dietetic management:- The main line of Treatment.

1. Types of feeding:-
   - There is 3 formulas:
     a) 100 cc cow milk + 2 ml oil + 5 gm sucrose 67 +18 +20 = 105 cal.
     b) 100 cc yoghurt + 2 ml oil + 5 gm sucrose.
     c) 15 gm full cream powdered milk + 100 cc water + 5 gm sucrose + 1 ml oil.

2. Amount of feeding:
   - Start with 150 ml/ kg /day then 150 Cal/ kg/day for 7-10 days.
   - Until catch up growth then increase the amount to 200 ml/kg/day
     1. Dilution :-
        - Start with half dilution for 1st 2 days, 2/3c one in next 2 days
        - Then gradually strength of feeding to full strength at the 5th day.
     2. Frequency: Start with small frequent diets 10-12 feds / day. Fed /2hrs
     3. Consistency: The diet must be soft to pass easily through Nasogastric tube.
Prevention of PEM:

A. Promotion of breast milk feeding
B. Proper weaning.
C. Programs of food supplementation:
   - Iron to treat anemia.
   - Iodine to treat & prevent hypothyroidism.
D. Early detection of P.E.M. by using weight charts and MAC.
E. Health education through health workers and mass media.
F. Mother's education.
G. Environmental sanitations
H. Immunization.
I. Control of most common diseases as diarrhea.

Q13: Failure To Thrive (FTT) (causes & management) 2005

Definition:
- Physical growth of an infant or child less than his peers.  
  - It is referred to grow below 3rd or 5th percentiles for age & sex.
  - It is change of growth that has crossed two major growth percentiles 
    (i.e. from above the 75th percentile to below the 25th percentile).

Causes
I- Inadequate intake:
   - Lack of appetite:
     - Anemia (e.g. iron deficiency) - Psychosocial problems (e.g. apathy)
     - Chronic disease (e.g. chronic infections)
   - Difficulty with ingestion:
     - Feeding disorder
     - Psychosocial problems (e.g. apathy, rumination)
     - Neurologic disorders (e.g. cerebral palsy, hypertension, hypotonia)
     - Craniofacial anomalies (e.g. choanal atresia, cleft lip and palate)
     - Generalized muscle weakness/pathology (Myopathies)
     - Dyspnea (e.g. congenital heart disease, pulmonary diseases)
     - Tracheoesophageal fistula.
   - Unavailability of food: Inappropriate feeding technique - Inappropriate food for age.

2- Altered growth potential regulation:
   - Prenatal insult - Chromosomal abnormality/genetic syndrome

3- Calorie wasting:
   - Vomiting, malabsorption, infections & renal losses (DM, RTA)

4- Increased caloric requirements:
   - Congenital heart disease/acquired heart disease
   - Chronic/recurrent infection
   - Chronic respiratory disease (Bronchopulmonary dysplasia)
   - Chronic anemia - Drugs/toxins (lead, levothyroxine)
   - Endocrinopathies (Hyperthyroidism, Hyperaldosteronism) - RTA
   - Metabolic disorders (Inborn errors of CHO metabolism) - Insufficient volume of food
Evaluation and Management:

1- Growth data:
   - Current growth parameters and Growth curves over time.
   - Relationship of growth parameters to each other.

2- History:
   a) Medical
      - Prenatal care & complications.
      - Gestational age & growth parameters at birth (SGA, prematurity).
      - Review of systems
        (Vomiting, Stool patterns & mechanics of feeding/swallowing, anorexia).
   b) Nutritional
      Caloric intake: 3-day diet history
      (food/beverage type, method of preparation, quantity consumed)
   c) Psychosocial
   d) Developmental/behavioral

3- Physical examination:
   Complete physical examination.

4- Developmental/behavioral assessment:
   Neuro-developmental assessment of gross/ fine motor, language, socio-emotional, cognitive skills.

5- Observation of a feeding:
   - Feeding environment (home observation)
   - Type & amount of food offered
   - Pace and duration of feeding

6- Laboratory studies:
   CBC, serum electrolytes, serum creatinine, urinalysis (± culture) & total protein/albumin, bone age (if height growth is also poor).

7- Hospitalize if:
   - High risk for abuse and neglect, very disturbed parent & child interaction.
   - Severe malnutrition and/or medically unstable.
   - Outpatient management failure.
**Clinical manifestation**

1. **Eye:** Rarely occur before 2-3 yr of age.
   - Impairment of dark adaptation → Night blindness
   - Drying of the conj. (Xerosis conj.) & cornea (Xerosis corneae)
   - Bitot spots (Dry, silver-gray plaques appear on the bulbar conjunctiva)
   - Wrinkling and cloudiness of the cornea or keratomalacia.
   - Follicular hyperkeratosis and photophobia.

2. **Skin**
   - Dry and scaly
   - Follicular hyperkeratosis: on the shoulders, buttocks & extensor surfaces of the extremities.

3. **Growth:** Apathy, retardation of mental and physical growth.

4. **Other**
   - Anemia with or without hepatosplenomegaly is usually present.
   - ↑ ICT with wide separation of cranial bones.

**Diagnosis:**

1. **Dark adaptation tests** help in diagnosing vitamin A deficiency.

2. **Biomicroscopic examination of the conjunctiva**
   - To detect the Xerosis conjunctivae.

3. **Concentration of carotene & Vit. A in plasma:**
   - Carotene concentration falls quickly.
   - Vitamin A concentration decreases more slowly.

**Prevention:**

- Infants should receive at least 500 µg of vitamin A daily
- Older children should receive 600-1500 µg of vitamin A or carotene.

**Treatment:**

- 1,500 µg of vit. A daily is sufficient for treating latent vit. A deficiency.
- Xerophthalmia is treated by giving 1,500 µg/kg orally for 5 days followed by daily IM injection of 7,500 µg of vit. A in oil until recovery occurs.

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**Vitamin D**

**Q15: Hypervitaminosis D 2003**

**Def**

It is a condition results from excess intake of Vitamin D for a long time (1-3 months).

**Clinical picture:**

- Picture stimulating hypercalcaemia
  - Anorexia, nausea, vomiting, irritability, pallor.
  - Polyuria, polydipsia, constipation.
  - Hypercalcuria.
  - Hypotonia, hyporeflexia.
  - Metastatic calcification of long bones.

**Treatment:**

- Discontinuation of Vitamin D.
- Corticosteroid (prednisone 2mg/kg) which antagonize calcium transport.
Q16: Rickets

(Various forms, commonest forms in Egypt, C/P, diagnosis, ttt & Prevention). 2007-01-94-93-92-91

Pathology

New bone formation is initiated by osteoblasts which are responsible for matrix deposition and with mineralization (Ca & Ph are essential for mineralization).

Types of ossification:

a. Endochondral → ↑ bone length.
b. Subperiosteal → ↑ bone thickness.

<table>
<thead>
<tr>
<th>Normal intracartilagenous ossify.</th>
<th>Ricketic changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zone of resting cartilage:</td>
<td></td>
</tr>
<tr>
<td>✓ Single layer of columnar cells.</td>
<td>✓ No change.</td>
</tr>
<tr>
<td>Zone of proliferating cartilage:</td>
<td></td>
</tr>
<tr>
<td>✓ 4-6 layers, irregular, avascular with no calcium.</td>
<td>✓ Formation of many layers → enlarged zone.</td>
</tr>
<tr>
<td>Zone of provisional calcification:</td>
<td></td>
</tr>
<tr>
<td>(degenerating cartilage)</td>
<td></td>
</tr>
<tr>
<td>✓ Chondrocyte → swollen, vacuolated.</td>
<td>✓ Chondrocytes don’t die</td>
</tr>
<tr>
<td>✓ Ca &amp; Ph are precipitated in matrix → well defined line (Epiphyseal line).</td>
<td>✓ No Ca deposition in the lower end of long bone → frayed, irregular.</td>
</tr>
<tr>
<td>Zone of ossification:</td>
<td></td>
</tr>
<tr>
<td>✓ Vasculaization of the cells</td>
<td>✓ Poor mineralization of the new osteoid → uncalcified osteoid → yield with pressure → cupping.</td>
</tr>
<tr>
<td>✓ Osteoblasts deposit osteoid materials which will be mineralized → bone formation</td>
<td></td>
</tr>
</tbody>
</table>

1- VITAMIN D DISORDERS

- Nutritional vit. D deficiency
- Congenital vit. D deficiency
- Secondary vit. D deficiency
  ✓ Malabsorption: Celiac disease
  ✓ ↑ Degradation
  ✓ ↓ Liver 25-hydroxylase.
  ✓ Anticonvulsants drug
    (Phenytions, Phenobarbitone)
    Interference with vit. D metabolism.
- Vit. D-dependent rickets
  ✓ Type 1
  ✓ Type 2 (End organ resistance)
- Renal Osteodystrophy (ROD)
  d.t. Chronic renal failure

2- Ca DEFICIENCY

- Low intake
  ✓ Diet
  ✓ Premature infants
    (rickets of prematurity)
- Malabsorption
- Primary disease

3- PHOSPHORUS DEFICIENCY

- Low intake
  ✓ Diet
  ✓ Premature infants
    (rickets of prematurity)
- Aluminum-containing antacids

4- RENAL TUBULAR DEFECTS

- X-linked(Familial) hypophosphatemic rickets
- Autosomal dominant hypophosphatemic rickets
- Hereditary hypophosphatemic rickets with hypercalciuria.
- Fanconi syndrome
- Renal Tubular Acidosis (RTA)

Pediatric Sheet
Clinical picture

i. Early manifestation:-
- **General**
  - Anorexia & Irritability.
  - Excessive sweating (Autonomic disturbance).
- **Craniotabes** :-
  It’s ping pong ball sensation due to softening of the skull bone, can be detect by pressure over occipital or posterior parietal bone just above & post. to the auricle.
- **Rachitic rosary** :-
  - It’s prominent enlargement of the costro chondral junction
  - It’s early palpable <raw of beads d.t. excess osteoid> → later visible.
- **Broadening of wrists & ankles** d.t. epiphyseal enlargement.

ii. Late (Advanced) manifestation:
- **Head:**
  - Anterior fontanelle is wide <large> with delayed closure.
  - Asymmetric skull.
  - Big <large> head if rickets develops early in the 1st year.
  - Bossing <thickening of the central parts> of frontal & parietal bones.
  - Caput quadratum (box shaped skull) results from bossing of skull.
  - Delayed teething with enamel defect and caries may occur.
  - Depressed nasal bridge secondary to frontal bossing.
- **Thorax:**
  - Dr. Harrison has rose pig with long tail
  - **Rachitic rosaries:** Not only palpable but also visible
  - **Longitudinal grooves:** developed posterior to the rosaries with flattening of sides of chest cage.
  - **Harrison sulcus:** Horizontal depression at the lower part of the chest along the costal attachment of the diaphragm which is dragged in during inspiration.
  - **Pigeon chest:** the sternum with its adjacent cartilage appears to project forward.
- **Vertebral spine:**
  - **Kyphosis:**
    - In dorso-lumbar region
    - **Apparent** while sitting due to laxity of spinal muscles and ligament.
    - **Disappears** if the child is suspended from his shoulder
    - DD: Pott's dse
  - **Scoliosis:** lateral curvature of the spine
  - **Lordosis:** may be seen in the lumber region while standing.
- **Pelvis:**
  - Contracted inlet: forward displacement of sacral promontory.
  - Contracted outlet: forward displacement of the lower part of the sacrum
    (Both in female may lead to obstructed labor later on life)
Extremities:

- **Broadening of epiphysis**: of long bone especially at wrist and ankle.
- **Marfan sign**: Transverse groove on medial malleolus d.t. failure of union or fusion of the two ossific centers & passage of the tendon of tibialis anterior on soft med malleolus.
- **Green-stick fracture**: Fracture of the cortex of one side of the long bone with no bone separation.

**Deformities**: d.t. weight bearing at the shaft of bones →

- Bowing of forearm in creeping infants.
- Bowing of legs (Genu varum)
- knock-knees (Genu valgum)
- Over extended knees during walking (Genu recurvatum)

**Muscles and ligaments (Non skeletal) manifestation:**

- Hypotonia of the muscles & laxity of ligaments lead to:
  - Delayed motor milestones e.g. delayed sitting, waddling, walking.
  - Pot belly abdomen<abdominal distention> d.t. weakness of abdominal muscles.
  - Ptosis of liver & spleen: d.t. chest deformity & weak abdominal muscles
  - Constipation (Intestinal hypotonia).

**Complications:** RICKETS

- Respiratory tract infection: due to chest deformity.
- Iron deficiency anemia.
- Dental caries
- GIT complication: Gastroenteritis (GE) is common d.t. stasis of int. contents.
- Tetany: is uncommon with nutritional rickets.
- Skeletal deformities & fracture.

**Diagnosis**

The diagnosis of rickets is based on typical history (of inadequate intake of vit. D & inadequate exposure to sunlight):

a) **Laboratory investigations:**

- **Specific findings**
  - **Serum Ca level**: Normal or low (N: 9 -11 mg/dl) d.t. 2ry hyperparathyroidism
  - **Serum Ph level**: less than 4mg/dl (N: 4.5 - 6.5 mg/dl).
  - **Serum alkaline phosphatase level**: increased > 500 IU/L (N: 50 - 200 IU/dl) d.t.
  - **Serum Parathormone hormone (PTH)**: High(N. 10-60 Pg/ml)
  - **Serum 25-D**: low.

- **Non-specific findings**:
  - Generalized aminoaciduria.
  - Low bone citrate level.
  - Elevated urinary citrate excretion.
  - Impaired renal acidification.
  - Impaired renal acidification.
  - Phosphaturia
### Radiological changes:
By X rays: best seen at the lower end of long bone especially wrist and ankle.

<table>
<thead>
<tr>
<th>Active rickets</th>
<th>Healing rickets</th>
<th>Healed rickets</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lower end:</strong> Triad</td>
<td>2 - 3 wks after ttt</td>
<td>After 4 weeks.</td>
</tr>
<tr>
<td>Broading: The joint space distance between the lower ends of the radius &amp; ulna and carpal bones is wide as uncalcified rachitic metaphysis don’t appear on X-ray.</td>
<td>Concave irregular interrupted line appears at the zone of provisional calcification (preparatory zone) with no fraying.</td>
<td>Straight continuous thick slight irregular than normal</td>
</tr>
<tr>
<td>Cupping: Concavity of the lower end</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fraying: Epiphyseal line is faint &amp;irregular → indistinct</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Shaft:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rarefaction → ↓ bone density.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Double periosteal line: along lateral outline.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Greenstick fracture: may occur in the long bone with no clinical symptoms.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Other features of active rickets are less evident.</td>
<td></td>
</tr>
</tbody>
</table>

### Prevention:
Vitamin D supplementation:
- **Full term:** 400 IU /day from 3rd month.
- **Premature:** 1000 IU /day from 2nd week.

### Treatment:
There are 2 strategies for administration of vitamin D:

#### a) With stoss<shot> therapy:
300,000-600,000 IU of vitamin D is administered orally or IM as 2-4 doses over 1 day. Stoss therapy is ideal in situations where adherence to therapy is questionable.

#### b) The alternative is daily:
Oral vitamin D, with high 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 old or 600 IV/day if >1 yr, adequate dietary Ca& Ph: this dietary intake is usually provided by milk, formula& other dairy products.
**Def.**
- It is a state of hyperexcitability of the central and peripheral nervous system d.t. ionized Ca⁺ resulting in a state of neuromuscular irritability.
- It occurs in severe rickets when calcium level falls below 7 mg/dl.

**Aetiology:**
1. **Hypocalcemia** (Serum Ca⁺ below normal) with Hyperphosphatamia.
   - Vit. D deficiency (Infantile rickets)
   - Other causes of hypocalcemia: Poor intake – Malabsorption – Hypoparathyrodism.
2. **Alkalosis** (↓ ionization of Ca)
   - Excessive vomiting → HCL depletion → respiratory alkalosis → ↓ ionized Ca
   - Hyperventilation → CO₂ wash → respiratory alkalosis → Tetany.
   - Excess alkali intake: e.g. citrate, bicarbonate.
3. **Hypomagnesemia:** - Chronic diarrhea. - Nephrotoxic medication. - Diuretic therapy.

**C/P**
- **Latent tetany** (serum calcium 7-9 mg/dl) detected by:
  - **Chevostek sign:** Not specific {Face}
    - Tapping on the facial n. (ant. to external auditory meatus) → Contraction of facial muscle
  - **Trousseau sign:** More specific {UL}
    - Blood pressure cuff is inflated slightly above the systolic blood pressure for more than 3 min → Carpopedal spasm due to ischemia of motor nerve.
- **Manifest tetany** (serum calcium 5-7 mg/dl):
  - **Convulsions:** only manifestation in neonates and young infants
  - Brief, generalized & occasionally localized with no loss of consciousness bet. seizures.
  - **Carpopedal spasm leads to** Flexion of the wrist & meta-carpophalangeal joints with finger extension.
    - **Spasm of other muscle:**
      - Facial muscles (Risus sardonicus)
      - Mastication muscles (Trismus of the jaw)
      - Laryngeal muscle (Stridor)
      - Diaphragm (Hiccup)
      - Back muscles (Opisthotonus)
  - **Parathesia:** Numbness, tingling in hands, feet, perioral esp. in older children.

**Investigation:**
- In hypocalcemia: Low serum Ca  High serum Ph
- In alkalosis: High PH of the blood
- In hypomagnesemia: Low serum Mg
- **ECG:** prolonged corrected QT interval.

**Treatment:**
1. **Acute attack** {Emergency treatment}
   - Calcium gluconate 10% (1-2 ml/kg solution slowly IV)
   - while monitoring for bradycardia. The dose can be repeated.
2. **Supplementation of the deficit** {Maintenance}
   - **In hypocalcemia:** Oral calcium e.g. Ca carbonate, Ca gluconate.
   - **In alkalosis:** breath into bag (to ↑ CO₂) trying to compensate by ++ respiratory acidosis.
   - **In hypomagnesemia:** Mg SO₄ IM 0.2 ml of 50% solution.
**Introduction**

**Q1: APGAR score 2001**

**Def:-**

It is a practical method to assess the condition of the newborn immediately after birth, done at 1 minute & at 5 minutes, to identify those requiring resuscitation.

**Components:-**

Five objective signs are evaluated and each is given a score of 0, 1, or 2 according to the following illustrative table:

<table>
<thead>
<tr>
<th>Sign</th>
<th>0</th>
<th>1</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appearance (Color)</td>
<td>Blue or pale</td>
<td>Pink body &amp; blue extremities</td>
<td>All pink</td>
</tr>
<tr>
<td>Pulse (HR)</td>
<td>Absent</td>
<td>&lt;100</td>
<td>&gt;100</td>
</tr>
<tr>
<td>Grimace (Resp. to nasal cath.)</td>
<td>No response</td>
<td>Grimace</td>
<td>Cough, sneezing</td>
</tr>
<tr>
<td>Activity (Ms tone)</td>
<td>Limp (Flaccid)</td>
<td>Some flexion</td>
<td>Active motion</td>
</tr>
<tr>
<td>Resp. effort (Breathing)</td>
<td>Absent</td>
<td>Slow - Irregular</td>
<td>Normal &amp; crying</td>
</tr>
</tbody>
</table>

**Significance:**

<table>
<thead>
<tr>
<th>APGAR score</th>
<th>Diagnosis</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-3</td>
<td>Cardiopulmonary arrest</td>
<td>Resuscitation</td>
</tr>
<tr>
<td>4-6</td>
<td>Close attention</td>
<td>Resuscitation</td>
</tr>
<tr>
<td>7-10</td>
<td>Normal infant</td>
<td>Discharge</td>
</tr>
</tbody>
</table>

Assessment should be carried on at: 1 - 5 - 10 - 15 minutes after birth.

**Interpretation**

**At 1 min**

1. Apgar score of 0-3 ➔ Immediate resuscitation.
2. Apgar score of 4-6 ➔ Resuscitation & Observation.
3. Apgar score of 7-10 ➔ Normal.

**At 5 min**

4. Apgar score of 0-3 ➔ Intubation, positive pressure ventilation & admission to NICU
5. Apgar score of 4-6 ➔ Perinatal asphyxia ➔
   - Continuing resuscitation efforts.
   - Assessment of Apgar at 10, 15 & 20 min.
NEONATAL REFLEXES

Definition:
It is primitive reflexes are present since birth which occur at the subcortical level, as the cerebral cortex is functionally deficient and disappear at 4-6 months of age with maturation of the cerebral cortex.

Significance:

- **Normal response**: they indicate a normal neurological condition.
- **Absence/Poor response**: at the time in which they normally present indicates:
  - Marked prematurity.
  - Depression of CNS: (HIE, ICH, sedation or anesthesia given to the mother during delivery).
- **Presence response**: after the normal time for disappearance indicates failure of development of the cortical area which suppress the reflex e.g. cerebral palsy
- **Asymmetrical or unilateral response**: it indicates focal neuromuscular lesion: Erb's palsy, fractured clavicle, dislocated shoulder.
- **Exaggerated response**: it indicates brain irritation, as in early stages of HIE, kernicterus, meningitis.

Q2: Moro reflex 2011-06

**Stimulus**

1. Placing the baby in a semi-upright position, with shoulders and back supported by the examiner's hand. The head is suddenly allowed to fall backward, with immediate support by the examiner's hand.
2. Sudden withdrawal of the blanket from underneath the infant.
3. Sudden loud sound behind the infant's ear.

**Response**

Sudden jerking movements consisting of abduction & extension of both arms, followed by adduction & flexion in an embracing manner.

**Time**: ≥ 28 Wks of GA → 4 mon

Q3: Grasp reflex 93

1) **Palmer grasp reflex**

**Stimulus**: Stroking of the infant's palm with the examiner's finger.

**Response**: Flexion of the infant's fingers, grasping the examiner's finger.

**Time**: ≥ 28 Wks of GA → 6 months

2) **Planter grasp reflex**

**Stimulus**: Touching the sole of the baby's foot with the practitioner's little finger.

**Response**: The baby's toes will flex towards the practitioner's finger.

**Time**: ≥ 28 Wks of GA → 10 months
Def.: It is live-born baby delivered before 37 weeks of GA regardless to his wt.

A/E

1- Idiopathic: Most cases

2- Secondary:
  - Maternal factors:
    - Maternal age (< 20 yrs and > 35 yrs).
    - Maternal malnutrition during pregnancy.
    - Multiple pregnancy
  - Fetal factors:
    - Congenital infection
    - Congenital malformation
    - Twin pregnancy
  - Obstetric factors:
    - Uterine malformation e.g. bicornuate uterus.

Criteria Complication

- Weight < 2500 gm.
- Length < 45 cm
- Head C. < 33 cm.

1) Respiratory:
   - Hyaline membrane disease (HMD)
   - Apnea.
   - Respiratory infections.

2) CVS:
   - PDA
   - Hypotension
   - Bradycardia.

3) CNS:
   - Kernicterus.
   - Intra-cranial hemorrhage.

4) GIT:
   - Necrotizing enterocolitis (NEC)

5) Immunological:
   - More liable to infection and sepsis

6) Nutritional:
   - Hypoglycemia.
   - Hypocalcemia.
   - Hypothermia.
   - Nutritional deficiencies (rickets and iron deficiency)

7) Others:
   - Retro-lental fibroplasias (Retinopathy of prematurity)

---

**Difference between preterm and small for date**

<table>
<thead>
<tr>
<th></th>
<th>Preterm</th>
<th>Small for date</th>
</tr>
</thead>
<tbody>
<tr>
<td>Size</td>
<td>Small &amp; plump</td>
<td>Small and wasted</td>
</tr>
<tr>
<td>Ms tone</td>
<td>Hypotonia</td>
<td>Good muscle tone</td>
</tr>
<tr>
<td>Hair</td>
<td>Lanugo</td>
<td>Dark</td>
</tr>
<tr>
<td>Skin</td>
<td>Thin &amp; transparent</td>
<td>Wrinkled</td>
</tr>
<tr>
<td>Ear &amp; genitalia</td>
<td>Immature</td>
<td>Mature</td>
</tr>
<tr>
<td>Face color</td>
<td>Plethora</td>
<td>Pale</td>
</tr>
</tbody>
</table>
Management

a. **Incubator care will provide:**
   - Regulation of body temperature.
   - Regulation of relative humidity.
   - Regulation of relative oxygen concentration.
   - The possibility of infections.

b. **Feeding:** (breast feeding is the best)
   - If **breast feeding is not possible** start formula feeding (LBW formula) using, bottle spoon, dropper or even nasogastric tube.
   - If **enteral (oral) feeding is impossible** use IV alimentation (Parenteral nutrition).
   - Vitamin and mineral supplements

c. **Prevention of infections:**
   - No or minimal visitors.
   - Strict hand washing for those handling the preterm & LBW babies.
   - Doctors and nursing staff should be free from infection and should wear masks.
   - Proper isolation of the infected baby.
   - Prophylactic antibiotics
   - Prophylactic immunoglobulin
CNS problems of the new born

Q5: (Hypoxic-Ischemic Encephalopathy) Perinatal Asphyxia 2008 - 2006 - 2003

1. Metabolic or mixed acidosis PH (< 7.0 of umbilical cord blood)
2. Low Apgar score (< 5 for > 5 minutes)
3. Neurological manifestation in the immediate neonatal period.
4. Multi organ system dysfunction in the immediate neonatal period.

Impairment of O\textsubscript{2} & CO\textsubscript{2} exchange and inadequate perfusion of tissues → 4 criteria:

- Metabolic or mixed acidosis PH (< 7.0 of umbilical cord blood)
- Low Apgar score (< 5 for > 5 minutes)
- Neurological manifestation in the immediate neonatal period.
- Multi organ system dysfunction in the immediate neonatal period.

Aetiology (Risk factors)
2. Intrapartum: Cord compression, cephalopelvic disproportion, breech presentation.
3. Postpartum: Severe pulmonary disease, congenital HD, large PDA & sepsis.

Clinical manifestations (consequences):
A- General manifestation:
- Depression of the neonate at birth.
- Low APGAR score and acidosis.

B- Multi-organ system dysfunction: Effect of ischemia on various organs
i. CNS: Hypoxic ischemic encephalopathy (HIE): 3 grades:

<table>
<thead>
<tr>
<th>Phenomenon</th>
<th>Grade I</th>
<th>Grade II</th>
<th>Grade III</th>
</tr>
</thead>
<tbody>
<tr>
<td>Consciousness</td>
<td>Irritable</td>
<td>Lethargy</td>
<td>Coma</td>
</tr>
<tr>
<td>Pupils</td>
<td>Dilated &amp; reactive</td>
<td>Constricted</td>
<td>Dilated &amp; Fixed</td>
</tr>
<tr>
<td>MS tone &amp; reflex</td>
<td>↑ Tone &amp; deep</td>
<td>↓ Tone &amp; deep</td>
<td>Flaccid &amp; absent</td>
</tr>
<tr>
<td>Convulsion</td>
<td>No</td>
<td>Controllable</td>
<td>No controllable</td>
</tr>
<tr>
<td>HR</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>EEG</td>
<td>Normal</td>
<td>Abnormal</td>
<td>Abnormal</td>
</tr>
<tr>
<td>Prognosis</td>
<td>Good</td>
<td>Variable</td>
<td>Poor</td>
</tr>
</tbody>
</table>

ii. Endocrine: adrenal failure, SIADH.
iii. CVS: Hypoxic cardiomyopathy and congestive heart failure.
iv. Resp: Persistent pulmonary hypertension of newborn, respiratory distress.
v. GIT: Necrotizing enterocolitis (NEC) & perforation.
vi. Liver: Hepatic necrosis with altered liver function tests.
vii. Renal: Acute tubular necrosis (ATN) leading to oliguria, hematuria.
viii. Hematological: Disseminated intravascular coagulopathy (DIC)

Investigations:
1. ABG: hypoxia, hypercarbia & acidosis.
2. Cranial ultrasonography
3. CT scan
4. MRI
5. EEG.

Management:
- Air way:
  - Open air way → (Position & suction)
  - Baby lie flat on slope table
  - Extension of the neck to open air way
  - Suction of oropharynx, nose.
- Breathing:
  - O\textsubscript{2} administration
    - Method:
      - Face mask
      - Ambu bag
      - Endotracheal intubation & mech. vent
    - Amount 2-3 L /min.
    - Pressure 20 -30 mmH\textsubscript{2}O
- Circulation
  - IV fluid (ringer lactate)
  - Cardiac massage by tip of two fingers on lower part of the sternum (80 -120 min)
- Drugs:
  1. Bradycardia → Atropine 0.1 - 0.3mg/kg/IV
  2. Convulsion → Diazepam 0.2 - 0.4mg/kg/IV
  3. Hypoglycemia → Glucose 10% 2 ml/kg/IV
  4. Acidosis → NaHCO\textsubscript{3} 1-3 mEq/kg /dose IV
  5. Acute morphine poisoning → Naloxone 0.1 - 0.4 mg/kg/dose IV
Definition:
Paroxysmal, time limited & change of motor and or behavior state due to electrical change of the brain.

Causes:
1- Hypoxic ischemic encephalopathy (HIE) 60%
2- Intracranial hemorrhage. 15%
3- Metabolic:
   Hypoglycemia, Hypocalcemia, Hypomagnesemia, Hypo or hyper natremia &
   Pyridoxine (Vit. B6) dependency
4- Infections:- 12%
   Sepsis, Meningitis and TORCH.
5- Maternal Drugs:
   Theophylline & corticosteroids.
6- Chronic Inborn errors of metabolism:- Maple syrup urine disease
7- Others:- Hydrocephalus - Polycythemia

Pathophysiology:
Normal neurons in the CNS undergo depolarization as a result of inward migration of Na.
Abnormal seizures occur due to excessive depolarization as IS:
- Failure of Na-K pump
- Increase excitatory > inhibitory (e.g. GABA) neurotransmitters.
- Inhibition of Na movement.

Clinical picture & classification:
1) Focal clonic seizure:
   Def:- Rhythmic twitching of muscle groups of extremities and face.
   Cause:- Local structural lesions, infections and subarachnoid hemorrhage.
2) Multi focal clonic seizure:- Common
   as before but include many muscle groups.
3) Tonic seizure:-
   Rigid extremities and trunk ± fixed deviation of the eyes.
4) Myoclonic seizure:-
   Brief focal or generalized jerks of distal muscle groups of the extremities or body.
5) Subtle (خفی) seizure:-
   Chewing, excessive salivation, apnea, blinking, nystagmus, bicycling, pedaling, changes in color and tongue thrusting especially in premature.

Investigations:
- Metabolic workup: blood glucose, serum Na, Ca and Mg.
- Infection workup:
  - CBC and differential leukocytic count, CRP.
  - Blood, urine and CSF cultures serum IgM specific for TORCH.
- Serum drug level in special cases e.g. Theophylline.
- Radiographic:
  - Ultrasound examination of the head.
  - CT scan of the brain.
- Lumbar puncture in suspected meningitis.
- EEG.

Treatment:
- Treatment of the attack:-
  - O2 therapy, IV fluid & suction of secretion
  - Anticonvulsants: second end round 2013
    1. Phenobarbital 15-20 mg/kg IV repeat the dose if seizures are not controlled.
    2. If seizures continue we add Phenytoin 20 mg/kg IV.
    3. If seizure still present we give Diazepam 0.3 mg/kg IV.
- After controlling the attack:-
  - Treatment of the cause:
    1. Hypoxic ischemic encephalopathy:
       - Observation
       - Prophylactic Phenobarbital
    2. Hypoglycemia: 10% glucose IV maintenance.
    3. Hypocalcemia: Ca gluconate 10% 2 ml/kg.
    4. Hypomagnesemia: Mg sulfate 50-100mg /kg IM or IV.
    5. Hyponatremia: increase Na in the IV fluids.
    6. Hypernatremia: decrease Na in the IV fluids.
    7. Pyridoxine dependency: pyridoxine 50-100mg IV.

N.B- Jitteriness (ارتعاشة):
- Startle response to minor physical stimuli or loud noises.

<table>
<thead>
<tr>
<th>N.B- Jitteriness</th>
<th>Focal clonic</th>
<th>Tonic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Holding of the hand</td>
<td>Not stop</td>
<td>Stop</td>
</tr>
<tr>
<td>Side</td>
<td>Uni or Bilateral</td>
<td>Bilateral</td>
</tr>
<tr>
<td>Association of acute man.</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>EEG</td>
<td>Abnormal</td>
<td>Normal</td>
</tr>
</tbody>
</table>
Aetiology of NRD:

**A. Respiratory causes:**

1. **Air ways Obstruction:**
   - **Nose:** Bilateral choanal atresia.
   - **Oral cavity:** Macroglossia, Micrognathia, Pierre Robin syndrome.
   - **Larynx:** Laryngeal obstruction (congenital laryngeal web, stenosis, F.B.)
   - **Trachea:** Tracheal obstruction (stenosis, F.B., compression, TOF)
   - **Bronchi:** Bronchial obstruction (F.B.< thick meconium>, compression)

2. **Lungs:**
   - Hyaline membrane disease (RDS)
   - Transient tachypnea of the newborn (TTN)
   - Meconium Aspiration Syndrome (MAS)
   - Neonatal pneumonia
   - Air leak syndrome (Pneumothorax)
   - Chronic lung disease (BPD)
   - Congenital lobar emphysema
   - Pulmonary hypoplasia (Diaphragmatic hernia, Potter syndrome)
   - Diaphragmatic hernia
   - Persistent pul. HTN of the newborn
   - Pulmonary He.
   - Phrenic nerve palsy
   - Pleural disease: Pneumothorax, Hydrothorax, Hydro-pneumothorax)

**B. Non respiratory causes:**

1. **CNS:** (HIE, ICH, Infection)
2. **CVS:** (Congestive HF, PDA, VSD)
3. **Hematologic:** (Anemia, Polycythemia)
4. **Metabolic:**
   - Hypothermia & hyperthermia
   - Hypocalcemia
   - Hypoglycemia
   - Infection
   - Acidosis

**Clinical manifestations of RD:**

- **Tachypnea** (R.R. ≥ 60 breaths/min)
- **Retractions** (Use of accessory muscles of respiration)
- **Grunting** (Self-induced CPAP).
- **Cyanosis** (Failure of the compensatory mechanisms)
- **Apnea** (May be primary or secondary).

**Assessment of RD:** By R.D. scoring system (e.g. Downe’s score)
Q8: Neonatal apnea 2010

Def:
- Absence of breathing > 15 sec. in term infant & > 20 sec. preterm infant with or without bradycardia.
- Cessation of resp. with cyanosis with or without bradycardia regardless the time.

Risk factor:
1. Preterm (most imp)
2. New born with nasogastric tube (+ Vagus) → - RC

Incidence
↑ with ↓ gestational age (G.A) (تناسب عكسي)
- 25% of preterm (< 34 w) → Apnea
- 100% of preterm (< 28 w) → Apnea

Causes
1- Central < CNS causes>
2- Peripheral < Other causes>
I- Pathological
II- idiopathic according to G.A

<table>
<thead>
<tr>
<th>I- Pathological apnea</th>
</tr>
</thead>
<tbody>
<tr>
<td>a) CNS:</td>
</tr>
<tr>
<td>1. Perinatal asphyxia (HIA) اهم سبب</td>
</tr>
<tr>
<td>2. Seizure</td>
</tr>
<tr>
<td>3. I.C hge</td>
</tr>
<tr>
<td>4. Hydrocephalus + ↑ CT</td>
</tr>
<tr>
<td>5. Infection (Meningitis)</td>
</tr>
<tr>
<td>6. Cerebral Infarction</td>
</tr>
<tr>
<td>b) CVS:</td>
</tr>
<tr>
<td>1- Congestive heart failure (CHF)</td>
</tr>
<tr>
<td>2- Congenital heart disease (PDA – TGA)</td>
</tr>
<tr>
<td>3- Congenital heart block</td>
</tr>
<tr>
<td>4- Hypo or hyper tension.</td>
</tr>
<tr>
<td>c) Respiratory:</td>
</tr>
<tr>
<td>1. Hypoxia (RDS)</td>
</tr>
<tr>
<td>2. Air way obstruction</td>
</tr>
<tr>
<td>3. Lung disease e.g. pneumonia</td>
</tr>
<tr>
<td>4. Inadequate ventilation</td>
</tr>
<tr>
<td>d) GIT:</td>
</tr>
<tr>
<td>1- NEC&lt;necrotizing enterocolitis&gt;</td>
</tr>
<tr>
<td>2- GERD</td>
</tr>
<tr>
<td>3- Feeding intolerance</td>
</tr>
<tr>
<td>e) Blood:</td>
</tr>
<tr>
<td>1- Anemia: more common in preterm</td>
</tr>
<tr>
<td>2- Polycythemia: more common in term (WHY?) disappearance of Rolex figure.</td>
</tr>
<tr>
<td>f) Metabolic:</td>
</tr>
<tr>
<td>1- Hypo or hyperthermia</td>
</tr>
<tr>
<td>2- hypoglycemia.</td>
</tr>
<tr>
<td>3- Electrolyte imbalance: ↑ or ↓ Na - ↑ K - ↑ NH3 - ↑ Mg</td>
</tr>
<tr>
<td>g) Other:</td>
</tr>
<tr>
<td>1- Sepsis 2- Vagal reflex 3- Drug: Phenobarbitone – anesthesia agents</td>
</tr>
</tbody>
</table>
II- Idiopathic apnea of prematurity

Onset: Starts in 2nd day and ends in 7th day of life.

Types:
- Central (40%): Respiratory center immature.
- Peripheral<Obstructive> (10%): Passive neck flexion.
- Mixed: (50%): Central & obstructive causes

Frequency: maybe 10 – 100 times per day

Treatment: it can be spontaneously resolved without medication.
1. If apnea produced cyanosis or bradycardia: it needs urgent medication
2. Immaturity of the brain stem RC is manifested by an attenuated response to CO₂ and a paradoxical response to hypoxia.
3. It occurs in the absence of any identifiable predisposing disease.

Diagnosis
According to type of apnea after attack
CNS EEG or - Resp Cest x-ray.

Treatment:
- A → Air way
- B → Breathing < O₂ by bag & must keep O₂ saturation bet. 88% - 92%.
- C → Circulation
- D → Drug
  1. Theophylline: ينطبغ 70% من الحالات
     - Loading dose 5mg/kg I.V.
     - Maintenance dose 5 mg/kg per day
  2. If fails → 1 + Nasal CPAP<Continue Positive Airway Pressure>
  3. If fails → 1+ 2+ NaHCO₃ to treat acidosis.
  4. If fails
     1 + 2 + 3+ Doxapram (Potent respiratory & CNS stimulant)
     Dose: 1mg/kg/hr IV infusion.
  5. If fails
     1+ 3+ 4+ Mechanical ventilation.
- E → Etiology < ttt of cause>
### Neonatal Hematology
#### Q9: Neonatal Anemia 2010

**Definition:**
Hematocrite (Hct) < 40–45 % (0.40-0.45).

**Causes:**

<table>
<thead>
<tr>
<th>A- Hemolysis</th>
<th>B- Blood loss</th>
<th>C- RBCs production(rare)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Immune hemolysis</td>
<td>• Prenatal causes</td>
<td></td>
</tr>
<tr>
<td>▶ Feto-maternal blood group incompatibility: Rb, ABO&amp; minor blood groups (e.g. C, E, Kell&amp; Duffy)</td>
<td>▶ Fetomaternal transfusion.</td>
<td></td>
</tr>
<tr>
<td>• Non-immune hemolysis</td>
<td>▶ Fetofetal, [twin-to-twin] transfusion</td>
<td></td>
</tr>
<tr>
<td>▶ Hereditary</td>
<td>▶ Obstetric: Placenta previa, abruptio placenta, incision of placenta at CS, hematoma or rupture of cord.</td>
<td></td>
</tr>
<tr>
<td>▶ Spherocytosis &amp;other abnormalities of red cell morphology.</td>
<td>▶ Postnatal causes:</td>
<td></td>
</tr>
<tr>
<td>▶ G6PD deficiency&amp; other red cell enzymes deficiencies.</td>
<td>▶ External bleeding: bleeding from umbilicus, bleeding after circumcision.</td>
<td></td>
</tr>
<tr>
<td>▶ Hemoglobinopathies: e.g. α&amp;γ thalassemia.</td>
<td>▶ Internal bleeding: Cephalhematoma, subgaleal hge, adrenal hge, ICH, pulmonary hge, hepatic subcapsular hge, GIT hge.</td>
<td></td>
</tr>
<tr>
<td>▶ Infections: e.g. Bacterial, Viral (TORCH).</td>
<td>▶ Congenital leukemia.</td>
<td></td>
</tr>
<tr>
<td>▶ Drugs: e.g. penicillin, cephalosporins &amp; vit. K3.</td>
<td>▶ Congenital pure red cell anemia (Diamond Black Fan syndrome)</td>
<td></td>
</tr>
</tbody>
</table>

**Clinical features:**
1. **Acute blood loss:** Pallor and shock with hypotension, poor tissue perfusion, and acidosis.
2. **Chronic blood loss:** Pallor, irritability & poor weight gain, and if severe enough may manifest with tachypnea, tachycardia, and tender liver (HF).
3. **Hemolysis:** Pallor, jaundice & hepatosplenoenegaly.

**Investigation:**
- CBC: Hb, Het, RBCs count.
- Reticulocytes: in hemolysis and acute blood loss, in diminished RBCs production.
- RBCs morphology e.g. spherocytosis & fragility test.
- Total and differential WBCs, platelets count & CRP for infection.
- Infant’s and mother’s blood groups & coomb’s test.
- G6PD assay.
- TORCH screen if suspected.

**Treatment:**
1. **Packed RBCs transfusion:** 15–20 ml/Kg over 2 hours if the baby has Cardio-pulmonary Compromise, (e.g. respiratory distress, congenital heart disease), or severe anemia Hct < 30 %.
2. **Partial exchange transfusion:** using packed RBCs in severely anemic infants, when routine transfusion to correct anemia, would result in circulatory overload.
3. **Iron-fortified formula:** (2-4mg/Kg/day), if they are not breast-fed.
4. **Vitamin E:** (15–25 IU/day, water-soluble) is given to preterm infants until the baby is 38–40 wk post-conception.
5. **Recombinant human erythropoietin:** offers a promise in anemia of prematurity.
**Definition:**
It is a bleeding disorder due to deficiency of vitamin K dependent clotting factors II, VII, IX & X.

**Pathogenesis**
Vit. K is synthesized by the intestinal bacterial flora. Because the newborn's intestine is sterile, and bacterial colonization is delayed by breast-feeding → HDN is common among newborns especially breast-fed ones.

**Clinical features:**

A) **Classic type:** (2nd day → 5th day)
- GIT bleeding (Hematemesis, melena, bleeding per rectum).
- Bruises.
- Bleeding from nose, mouth, venipuncture sites, heel sticks and circumcision.

B) **Early-onset type:** (on the first 24hr of life)
- **Cause**
  - Hereditary clotting factors deficiency.

C) **Late-onset type:** (after the 15th week of life (1-6 mon)).
- **Cause**
  - Malabsorption, cholestasis, biliary artesia, hepatitis & cystic fibrosis.
  - Intracranial hemorrhage is common.

**Diagnosis:**
- CBC: usually within normal range, except if bleeding is severe, normocytic normochromic anemia with mild reticulocytosis is present.
- **Bleeding time:** Normal
- **Clotting time:** PT, and PTT are prolonged.

**Prevention**
All newborn infants should receive vit. K1 1 mg IM at birth.

**Treatment:**
- **Vit. K1:** (1-5 mg slow infusion) and stops bleeding within few hours.
- **Fresh Frozen Plasma Infusion:** 10 ml/kg
  May be required for serious active bleeding & may be repeated every 8-12 hours, if needed.
- **Blood transfusion** in severe bleeding to correct anemia.
### Q11: NEONATAL JAUNDICE (NJ)

#### Def.:  
- **Neonatal Hyperbilirubinemia:**
  Biochemical term indicates elevated Total Serum Bilirubin (TSB) >1 mg/dl.

- **Neonatal Jaundice:**
  Clinical term denotes yellowish discoloration of skin & mucus membranes. 
  - TSB > 2 mg/dl in adults.
  - TSB >7mg/dl in newborns.

#### Types & Causes of Neonatal Jaundice

##### A. Pathological Jaundice

<table>
<thead>
<tr>
<th>Causes</th>
<th>Direct hyperbilirubinemia (CB ≥ 20% of TBS)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Production</td>
<td>1. Defective secretion of CB by hepatocytes:</td>
</tr>
<tr>
<td></td>
<td>• Rotor &amp; Dubin Johnson syndromes (AR)</td>
</tr>
<tr>
<td></td>
<td>• Neonatal hepatitis either Sepsis or Idiopathic or cong. Infection (TORCH)</td>
</tr>
<tr>
<td></td>
<td>• Metabolic as galactosemia, tyrosemia, α1 antitrypsin deficiency &amp; cirrhosis.</td>
</tr>
<tr>
<td></td>
<td>2. Defective Excretion (Obstruction jaundice)</td>
</tr>
<tr>
<td></td>
<td>• Hepatic:</td>
</tr>
<tr>
<td></td>
<td>• Congenital intrahepatic biliary atresia.</td>
</tr>
<tr>
<td></td>
<td>• Viral hepatitis.</td>
</tr>
<tr>
<td></td>
<td>(Viral hepatitis ➔ Defective the uptake, conjugation &amp; secretion ➔ Mixed hyperbilirubinemia)</td>
</tr>
<tr>
<td></td>
<td>• Extrahepatic:</td>
</tr>
<tr>
<td></td>
<td>• Congenital extrahepatic biliary atresia.</td>
</tr>
<tr>
<td></td>
<td>• Biliary tumor or stones.</td>
</tr>
<tr>
<td></td>
<td>• Inspirated bile syndrome:</td>
</tr>
<tr>
<td></td>
<td>(Prolonged UC hyperbilirubinemia ➔ Production of CB by liver which accumulates in biliary canalculi ➔ Obstruction of them ➔ Changing the condition to Conjugated hyperbilirubinemia)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Causes</th>
<th>Indirect hyperbilirubinemia (CB &lt; 20% of TBS)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>2. Enclosed hges: Extensive petechiae, hematomas &lt;Cephalohematoma&gt;, Pulmonary...</td>
</tr>
<tr>
<td></td>
<td>3. Sepsis &amp; TORCH infection</td>
</tr>
<tr>
<td></td>
<td>4. Defective Transport:</td>
</tr>
<tr>
<td></td>
<td>• Albumin (e.g. Nephrosis)</td>
</tr>
<tr>
<td></td>
<td>• Displacement of bilirubin from p.t.n binding sites</td>
</tr>
<tr>
<td></td>
<td>(Ampicillin - Aspirin - Warfarin)</td>
</tr>
<tr>
<td></td>
<td>5. Non-hemolytic:</td>
</tr>
<tr>
<td></td>
<td>• Polycythemia: Materno-fetal transfusion, Feto-fetal transfusion.</td>
</tr>
<tr>
<td></td>
<td>6. Enclosed hges: Extensive petechiae, hematomas &lt;Cephalohematoma&gt;, Pulmonary...</td>
</tr>
<tr>
<td></td>
<td>7. Sepsis &amp; TORCH infection</td>
</tr>
<tr>
<td></td>
<td>8. Defective Transport:</td>
</tr>
<tr>
<td></td>
<td>• Albumin (e.g. Nephrosis)</td>
</tr>
<tr>
<td></td>
<td>• Displacement of bilirubin from p.t.n binding sites</td>
</tr>
<tr>
<td></td>
<td>(Ampicillin - Aspirin - Warfarin)</td>
</tr>
<tr>
<td></td>
<td>9. Defective Uptake:</td>
</tr>
<tr>
<td></td>
<td>• Z,Y proteins (Gilbert’s syndrome &lt;AD, mild dse&gt;)</td>
</tr>
<tr>
<td></td>
<td>10. Defective Conjugation:</td>
</tr>
<tr>
<td></td>
<td>(U-Glucoronyl transferase enzyme)</td>
</tr>
<tr>
<td></td>
<td>• Absence: (Crigler najjar syndrome Type I &lt;AR, severe dse&gt;)</td>
</tr>
<tr>
<td></td>
<td>• Deficiency: (Crigler najjar syndrome Type II &lt;AD, less severe than type I)</td>
</tr>
<tr>
<td></td>
<td>• Lack of stimulation: (Hyperthyroidism)</td>
</tr>
<tr>
<td></td>
<td>• Inhibition: (Breast milk jaundice) due to</td>
</tr>
</tbody>
</table>

- Pregnantriol & non-estrified fatty acids present in breast milk. (Early)  
- Enterohepatic circulation due to presence of β-glucuronides in breast milk that deconjugated bilirubin. (Late)

#### Characteristics

1. Clinical jaundice appears the first 24 hr of life.
2. Clinical jaundice persists ≥ 14 day.
3. TSB: • > 12 mg in full term. • > 15 mg in preterm infants.
4. A rate of rise in TSB • 0.2 mg/dl/hr. • > 5 mg/dl/24hr.
5. **Sings of an underlying Illness:**
   (Lethargy, Poor feeding, apnea, Tachypnea, Temperature instability, vomiting, HSM).
6. Direct or indirect hyperbilirubinemia
Investigations:
- Total & direct serum bilirubin
- CBC (Hb, Hct, RBCs morphology, Retics, WBCs and platelets).
- Coombs test (identifies Ab that RBCs (for both the mother and the baby)
- Blood type and Rh (for both mother and baby)
- Screen for chronic hemolytic anemia (G6PD assay, osmotic fragility test)
- Bilirubin/Albumin ratio: > 8 mg → indicates Exchange transfusion.
- Liver function profile (increased CB, cholestasis).
- Sepsis screen (Cultures, CRP).
- TORCH screen (specific IgM & IgG).

B. Physiological jaundice: 2011

Mechanism (Causes)
1- Short life span of neonatal RBCs{ 90 day} → RBCs destruction → ↑ Bilirubin production.
2- Low Y& Z protein levels in liver cells during the 1st wk. → ↓ Uptake.
3- Immaturity of Glucuronyl transferase enzyme → ↓ Conjugation.
4- High levels of β-glucuronidase, ↓ enteral intake, ↓ Intestinal bacteria &
     ↓ Gut motility → ↑ enterohepatic circulation.

Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Onset</th>
<th>Peak</th>
<th>Disappear</th>
</tr>
</thead>
<tbody>
<tr>
<td>1- Full term</td>
<td>2nd or 3rd day</td>
<td>4th or 5th day</td>
<td>6th or 8th day</td>
</tr>
<tr>
<td>2- Preterm</td>
<td>3rd or 4th day</td>
<td>6th or 8th day</td>
<td>10th or 12th day</td>
</tr>
</tbody>
</table>

3- TBS:
   - In full term: < 12 mg/dl.
   - In preterm: < 15 mg/dl.

4- Rate of bilirubin rise
   - < 0.2 mg/dl/hr.
   - < 5 mg/dl/24 hr.

5- On examination:
   No any abnormal sign (poor feeding, lethargy, hypothermia...).

6- Unconjugated hyperbilirubinemia.

DD: Physiological jaundice must be differentiate from causes of jaundice.

<table>
<thead>
<tr>
<th></th>
<th>Physiological jaundice</th>
<th>Pathological jaundice</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset</td>
<td>2nd or 3rd day (3-4 in preterm)</td>
<td>Any time (even 1st day)</td>
</tr>
<tr>
<td>Duration</td>
<td>About one week in full term (2 wk in preterm)</td>
<td>May be longer</td>
</tr>
<tr>
<td>Rate of rise</td>
<td>&lt; 5 mg/dl/24 hr.</td>
<td>&gt; 5 mg/dl/24 hr.</td>
</tr>
<tr>
<td>Peak level</td>
<td>&lt; 12 mg/dl (&lt;15 in preterm)</td>
<td>&gt; 12 mg/dl (&gt;15 in preterm)</td>
</tr>
<tr>
<td>Examination</td>
<td>Not associated abnormal signs</td>
<td>Associated abnormal signs</td>
</tr>
<tr>
<td>Type</td>
<td>Unconjugated</td>
<td>Unconjugated or conjugated</td>
</tr>
</tbody>
</table>

♥ Prolonged physiological jaundice: Persist > 2 weeks
- Hypothyroidism: Thyroxin is essential for maturation of the enzyme.
- Breast milk jaundice: Pregnandiol → inhibition of the enzyme
- Constipation (↑ enterohepatic circulation)
- Congenital pyloric stenosis (↑ enterohepatic circulation)

TTT: Usually resolves spontaneously, in exaggerated cases:- Phototherapy
Management of Neonatal Jaundice:

A- General:
- Infant’s feeds both in volumes & calories.
- Stop drugs that interfere with bilirubin metabolism.
- Correct associated abnormalities: hypoxia, acidosis, infection...

B- Specific:

Depends on:
- Serum unconjugated bilirubin.
- Gestational age and birth weight.
- Post-natal age.
- Co-morbidity (Complicated or uncomplicated).

<table>
<thead>
<tr>
<th>I- Phototherapy</th>
<th>II- Exchange transfusion</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Idea</strong></td>
<td>Removal of excess UC bilirubin 50-60%</td>
</tr>
<tr>
<td>Exposure of neonate with unconjugated hyperbilirubinemia to blue or white light will convert UC bilirubin → non-toxic photoisomers which are excreted in urine &amp; bile</td>
<td>Removal of Abs from the circulation in case of RH incompatibility.</td>
</tr>
<tr>
<td><strong>Indication</strong></td>
<td>Corrects anemia</td>
</tr>
<tr>
<td>Rise of bilirubin to high levels (But below the critical levels).</td>
<td></td>
</tr>
<tr>
<td>During waiting for exchange transfusion.</td>
<td></td>
</tr>
<tr>
<td>Prophylactic in:</td>
<td></td>
</tr>
<tr>
<td>Very low BW.</td>
<td></td>
</tr>
<tr>
<td>Severely bruised neonates.</td>
<td></td>
</tr>
<tr>
<td>Immediately after birth if Rh incompatibility is suspected</td>
<td></td>
</tr>
</tbody>
</table>

| **Procedure** | |
| It is done through an umbilical vein catheter attached to 3-way stopcock valve with iso-volumic exchange transfusion, withdraw 10-20 ml of the infant blood and infuse 10-20mL of the donor blood till the volume of exchange is completed. | |
| 1) Special blue or green phototherapy bulbs are used (WL of 425-490nm). | |
| 2) The radiance of the bulbs should be >30 μw/cm²/nm. | |
| 3) The nearest distance from the baby (30-40 cm). | |
| 4) Should be continuous with short intervals for feeding | |
| 5) Shield eyes and testicles (using face mask as a diaper). | |
| 6) Change the position of the baby every 2 hr. (supine / prone). | |

| **Side effect** | |
| 1) Dehydration and/or fever, due to increased insensible water loss. | |
| 2) Diarrhea(d.t. bile salts in stool → watery) | |
| 3) Damage to the eye or genitalia. | |
| 4) Erythema (phototherapy rash). | |
| 5) Bronze baby syndrome, in case of rect hyperbilirubinemia. | |

III- Other lines of TTT:

- **Phenobarbione**:
  - In oral doses of 4-5 mg/kg/day, in 2-3 divided doses.
  - It induces hepatic enzymes →↑ bilirubin conjugation& excretion.
  - It is recommended in Crigler-Najjar Type II.
  - It needs 3-4 days to start action.
  - It may cause lethargy& poor feeding, especially in preterm.

- **TTT of the cause**:
  - **1- Inhibition of enterohepatic circulation**:
    - Oral Agar binds CB preventing the formation of UCB.
    - Oral activated charcoal binds CB preventing the formation of UCB.
  - **2- Inhibition of immune hemolysis**:
    - IVIG →↓ bilirubin levels in isoimmune hemolytic disease (Rh, ABO…) by occupying the FC receptors of reticuloendothelial cells → Preventing them from taking up & lysing antibody-coated RBCs.
  - **3- Inhibition of bilirubin production**:
    - Metalloprotoporphyrins(10 mesoporphyrin) are competitive inhibitors of heme oxygenase →↓ conversion of heme to bilirubin.
  - **4- Bilirubin oxidase**: allows for the oxidation of bilirubin → water soluble & excretable.
**Q12: Neonatal Hypoglycemia 2003**

**Definition**
Blood glucose level (< 40mg/dl) irrespective to gestational age.

**Causes**
- Infants of diabetic mothers.
- Infants with IUGR.
- Stressed infants.
- Preterm infants.

**Clinical picture**
♥ Asymptomatic
♥ Lethargy, irritability, jitteriness, poor feeding, seizures, level of consciousness& coma.

**Differential Diagnosis**
- Islet cell hyperplasia-Beckwith- Wiedemann syndrome, erythroblastosis
- Genetic hyperinsulinism.
- Inborn errors-galactosemia, glycogen storage diseases.
- Endocrine(hypopituitarism).
- Complication of birth asphyxia.
- Complication of sepsis.

**Treatment**
♥ Blood sugar < 40 mg/dl with symptoms
   - Start IV within 10 minutes.

♥ Blood sugar <40 mg/dl without symptoms
   - Feed and repeat glucose and give 2 mL/kg of 10% Dextrose bolus followed by 3.6 mL/kg/h constant infusion.
   - Monitor glucose within 30 minutes.

♥ Blood sugar <20 mg/dl with/without symptoms
   - Start IV within 10 minutes and give 2 mL/kg.
   - 10% dextrose bolus followed by 3.6 ml/kg/h constant infusion.
   - Monitor glucose within 30 minutes.
**Definition:**
Neonate born to diabetic mother (Pre-existing (before pregnancy) or gestational (during pregnancy)) usually large for gestational age & preterm.

**Common problems:**

- **Congenital anomalies:**
  - Cardiac anomalies
  - CNS anomalies: Neural tube defect {NTD}
  - Renal anomalies
  - GIT anomalies: Small left colon syndrome : delayed passage of meconium, meconium plug

- **Metabolic:**
  - Hypoglycemia (Serum glucose < 40 -45 mg/dl)
  - Hypocalcemia (Serum total Ca < 7mg/dl or ionized Ca < 4.4 mg/dl)
  - Hypomagnesemia (Serum Mg < 1.5 mg/dl)

- **Blood:**
  - Hyperbilirubinemia
  - Polycythemia

- **Resp.:** RDS

- **Obstetric:**
  - Abortion - still birth - preterm delivery

- **Renal:**
  - Renal vein thrombosis

- **Other:**
  - Macrosomia → Birth Trauma.

**Investigation:**
- Blood glucose level at birth, 1hr, 3hrs, 6hrs, 12hrs, 24hrs, 36hrs & 48hrs.
- Incubator care for at least 48 hrs & monitoring

**Management:**

- IDM is a high risk newborn, thus delivery should be carried out at a hospital equipped for neonatal care.
- Resuscitation and stabilization of the newborn.
- **Treatment of hypoglycemia:**
  Bolus IV dextrose 10% 2-4 ml/kg/min.
- **Treatment of hypomagnesemia:**
  Bolus MgSO₄ 50% 0.1-0.2 ml/kg (50-100 mg/kg)IM.

  Complete physical examination and follow up searching for complications of IDM and treat.
**Definition:**
It is a clinical syndrome of symptoms and signs due to the presence of bacteria & its toxins in the blood stream and /or other body fluids and tissues.

**Risk Factors:**

**A) Maternal Risk Factors:**
1) Premature rupture of membranes ≥18 hrs. {{PROM}}
2) Chorioamnionitis (intrapartum fever ≥ 38°C, foul vaginal discharge, uterine tenderness, leukocytosis & increased CRP.
3) Premature delivery.
4) Maternal UT infection.

**B) Neonatal Risk Factors:**
1) Preterm & low birth weight newborns.
2) Invasive procedures: IV catheters, endotracheal tube, chest tube.
3) Congenital defects: Meningocele, TOF, obstructive uropathy, cleft lip & palate.
4) Male sex

**C) Environmental Risk Factors:**
1) Contaminated place, equipments & personnel.
2) No adherence to infection control measures (nosocomial infection).

**Causative organism:** differ according to the pattern of infection:

<table>
<thead>
<tr>
<th>Early-onset sepsis</th>
<th>Late-onset sepsis</th>
</tr>
</thead>
<tbody>
<tr>
<td>(first 3 days)</td>
<td>(&gt; 3 days)</td>
</tr>
<tr>
<td>Maternally acquired</td>
<td>Environmentally acquired</td>
</tr>
</tbody>
</table>

**Organism:**
GBS, gram-negative enterococci, E coli, Listeria monocytogenes

**Organism:**
Coagulase-negative Staph., Staph. Aureus, E coli, Klebsiella & Pseudomonas

**Problem:**
Mainly pneumonia

**Problem:**
Meningitis

**Clinical picture:**
1) **General:** nonspecific symptoms & signs
   - Poor feeding, jaundice, absent or poor moro’s reflex.
   - Temperature instability (Hypothermia > Hyperthermia)
2) **CNS:** Convulsion, irritability, high pitched cry & pulging AF.
3) **Respiratory:** Tachypnoea, RD, grunting, cyanosis & apnoea.
4) **GIT:** Vomiting, diarrhea, abdominal distension "Paralytic ileus", bleeding per rectum" Necrotizing Enterocolitis (NEC).
5) **Blood:** Bleeding (thrombocytopenia, DIC)
6) **Skin:** Hardening of skin "Scleroderma".
7) **Septic shock:** Tachycardia & hypotension
### Investigation: Sepsis screen

1. **CBC:**
   - **WBC:**
     - Leucocytosis > 20,000/cc or Leucopenia < 5000 cc.
     - Neutropenia <1500/cc or Neutrophilia >8000/cmm,
     - Immature neutrophile "Band cell" > 10%
     - Immature band cell/Mature≥ neutrophil ≥ 0.2%
   - **Platelets:**
     - Thrombocytopenia < 150,000/cc.
   - **RBC:**
     - Anemia, Hct < 40%.

2. **Cultures:**
   - Blood, CSF, urine, local sites of infection.

3. **Radiological:**
   - Chest X-ray: (Pneumonia)
   - CT& MRI

4. **ESR, CPR (> 0.6 mg/dl).**

5. Check blood glucose and serum electrolytes for any abnormality.

### Treatment:

1) **Start IV antibiotics** after collection of samples for cultures.
   - Amoxicillin (200 mg/kg/day)& gentamicin(4-6 mg/kg/day) for early-onset sepsis.
   - Vancomycin and third generation cephalosporin for late onset-sepsis.
   - **Duration:** 10-14 days for proven sepsis, 14-21 days for meningitis.

2) **Immunotherapy:**
   - IVIG
   - GM-CSF
   - Exchange transfusion

3) **Supportive therapy:**
   - Thermoregulation (Incubator care).
   - Nutrition, fluids and electrolytes therapy.
   - Correction of hypoxia and acidosis.
   - Fresh frozen plasma and platelet transfusion for DIC.
   - Phototherapy and exchange transfusion for hyperbilirubinemia.

---

### Treatment of Schistosomiasis

1. **Praziquantel**
   - 40 mg / kg / day → divided bid for 1 day.
   - 60 mg / kg / day → S. Japonicum & S. mekongi.

2. **Niridazole**
   - 15 mg / kg / day for 10 days.

3. **Prevention**
   - Sanitary measures.
   - Eradication of snails.
   - Treatment of individuals.
**Treatment of Giardiasis**

1. Metronidazole ... 15 mg / kg / day ➔ for 7 days.
2. Tinidazole ..... 50 mg  single oral dose.
3. Furazudione ..... 6 – 8 mg ➔ for 10 days.

**Treatment of Chicken pox**

1. Anti histaminic ➔ for allergy.
2. Anti pyretic ➔ paracetamol.
3. Anti septic ➔ calamine lotion.
4. Anti viral ➔ acyclovir.
5. Treatment of complications.

**Treatment of amoebiasis**

1. Metronidazole 30 – 50 mg /kg / day ... orally in 3 divided doses for 10 days.
2. Tinidazole 50 – 60 mg / kg.
3. Test of cure ➔ stool examination after 2 weeks from end of treatment.

**Congenital rubella syndrome**

**Definition**

Intra uterine infection with rubella in the 1st trimester of pregnancy ➔ organogenesis.

**Clinical pictures**

1. C.N.S.
   - Microcephaly.
   - M.R.
   - Convulsions.

2. Eye
   - Cataract.
   - Glaucoma.
   - Retinopathy.
   - Cloudy cornea.

3. C.V.S.
   - P.S.
   - PDA.
   - VSD.

4. Lung
   - Interstitial pneumonia.

5. Ear
   - Sensory neural hearing loss.

6. HSM

7. Skin
   - Rash.
   - Purpura.

8. Bone
   - Rubella osteomyelitis.

**Prevention**

1. Terminate pregnancy if
   - Clinical rubella develops.
   - Antibody titer is rising.
2. Allow pregnancy if Ab titer is
   - Already high.
   - Not rising.
3. IG ➔ not recommended.
Febrile patient at high risk of serious bacterial infection

A. Immune component
   - Neonates.
   - Hyperpyrexia > 40 C.
   - Fever with petichiae.

B. Immune compromised
   - Sickle cell disease.
   - Asplenia.
   - Immune deficiency.
   - Malignancy.

Mumps

Complications
1. C.N.S.
   - Meningeo-encephalitis.
   - Aseptic meningitis.
   - Encephalitis.
   - GBS.
   - Transient facial palsy.

2. Orchitis
   - At the end of 1st week of illness.
   - Unilateral.
   - Red, swollen, painful and tender.
   - Absolute infertility is rare but partial impairment.

3. Oophritis
   - Lower abdominal pain and tenderness.
   - Fertility not affected.

4. Acute pancreatitis

Management
- Prevention ➔ MMR vaccine.
- Treatment ➔ no specific therapy.

Entrobious treatment

<table>
<thead>
<tr>
<th>Drug</th>
<th>Ascaris</th>
<th>Entrobious</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pyrantel pamoate</td>
<td>(of choice) 11 mg / kg once daily for 3 days ... maximum dose 2 gm.</td>
<td>The same</td>
</tr>
<tr>
<td>Mebendazole or flubendazole</td>
<td>100 mg bid for 3 days or 500 mg once</td>
<td>(of choice) 100 mg as single dose</td>
</tr>
<tr>
<td>Albendazole</td>
<td>400 mg as a single dose</td>
<td>400 mg as a single dose</td>
</tr>
</tbody>
</table>

All family must be treated and therapy may be repeated after two weeks in entrobious.
Roseola infantum

**Etiology** .... Human herps virus 6 & 7.

**M O T** .... Droplet.

**I.P.** .... 2 – 3 weeks.

**Clinical manifestations**
- Season ➔ spring.
- Prodrome of
  - Mild diarrhea.
  - Mild redness of conjunctiva, pharynx.
- Clinical presented by

  1. Fever
     - Sudden high.
     - Last 3 – 4 days.
     - Associated with convulsions.
  2. Maculopapular rash
     - Appears on the 4th day with drop of fever.
     - Rainbow flowing of the storm.
     - Starts on the trunk and spreads rapidly on the arms and trunk.
     - Last only 24 hours.

**Diagnosis** ... clinically.

**Treatment**
- Symptomatic.
- Anti pyretic

**FUO**

**Definition**
Documented fever > 38 C lasting for > 14 days with no obvious cause despite
- Complete history.
- Physical examination.
- Routine lab investigations.

**Causes**

A. Infection
- Systemic
  - Bacterial ➔ T.B., typhoid, Brucellosis.
  - Viral ➔ EBV, CMV.
  - Protozoal ➔ Toxoplasmosis.
- Localized
  - Sinusitis.
  - Otitis media.
  - I.E.
  - UTI.

B. Collagen diseases
- SLE, RA.

C. Malignancy
- Leukemia.
- Lymphoma.
- Neuroblastoma
- Dermatomyositis.

D. Miscellaneous
- IBD ➔ U.C. & C. disease.
- Drugs ➔ salicylate, atropine.

**Investigations**
Treatment
- Tap water fomentation.
- Antipyretic drugs.
- Broad spectrum AB.

Pertussis

Clinical pictures
1. Catarrhal stage
   - 1 – 2 weeks.
   - Nasopharyngitis.
   - Flu like pictures "cough, fever, rhinorrhea, sneezing".
2. Paroxysmal stage
   - 2 – 6 weeks.
   - Gradually increase which is mainly at night.
   - During the attack
     ✓ Protruded tongue.
     ✓ Bulging eye.
   - The attack is followed by
     ✓ Vomiting.
     ✓ Whoop ➔ forceful inspiratory gasp.
   - Infancy ➔ no whoop, but apnea and cyanosis.
3. Convalescence stage
   - 1 – 2 weeks.
   - Cough decrease gradually.
   - Improvement of general condition.

Complications
- Respiratory
  ✓ Pneumonia.
  ✓ Atelectasis or bronchiectasis.
  ✓ O.M.
  ✓ Dissemination of previously existing 1ry T.B.
  ✓ Sinusitis.
  ✓ Forceful paroxysm
  ✓ Emphysema.
  ✓ Rupture alveoli
    ✓ Pneumothorax.
    ✓ Pneumo mediastinum.
  ✗ Rupture of diaphragm.
- GIT
  ✓ Vomiting
    ✓ Malnutrition, dehydration.
    ✓ Alkalosis, tetany.
- C.N.S.
  - Tetany.
  - I.C.H.
  - Convulsion in infants.
- Blood
  - Sub conjunctival hemorrhage.
  - Purpura ➔ rupture capillary.

**Pulmonary T.B.**

### Clinical pictures
1. Asymptomatic in most cases ➔ discovered when doing X-ray chest.
2. Feeding is poor, fatigue, face edema.
3. Non productive brassy cough & cyanosis.
4. Dyspnea.
5. Emphysema, collapse may occur & positive Depsin’s sign.
6. Hyper sensitivity reaction
   - Erythema nodosum.
   - Phlycten.
   - Pleural effusion.
7. Miliary T.B.

### Prophylaxis
1. BCG vaccine
   - 0.1 ml I.D. in left arm.
   - Live attenuated.
   - 60 – 80 % protection against T.B.
   - Prevents ➔ miliary T.B. & T.B. meningitis.
   - Normal reaction ➔ wheal, swelling, abscess, ulcer after 1 ms.
   - Accelerated ➔ red swelling in 2 – 3 days.
   - No reaction ➔ repeated the vaccination after 3 ms.
   - Complicated by ➔ ulcer, adenitis, osteitis, fever, convulsions.
2. Health education.
3. Proper isolation.
4. Good nutrition.
5. Avoid overcrowding.
6. Prevent perinatal T.B. by using INH, RIF, EMB.

### Treatment
- I ➔ I.N.H.  10 – 15 mg / kg / day  6, 9, 12 months.
- P ➔ Pyrazinamide  20 – 40 mg / kg / day  2 months.
- R ➔ rifampicin  10 – 20 mg / kg / day  6, 12 months.
- E ➔ Ethambutol  5 – 25 mg / kg / day  6, 9, 12 months.
- S ➔ streptomycin  20 – 40 mg / kg / day  for 2 months.
- S ➔ steroids  1 – 2 mg / kg / day for 1 – 2 months.

**Interpretation of tuberculin test**

**Method**
0.1 ml of purified protein derivative (PPD) → intra dermal in the front of left forearm.

**Result**

Induration after 48 – 72 hours

- 0 – 5 mm → negative test.
- 5 – 9 mm → doubtful → repeated after 2 weeks.
- > 10 mm → positive test.

**Positive**

- Non vaccinated child < 5 years.
- Highly positive > 15 mm patient in contact to open T.B. case.

**False positive**

- Vaccinated.
- Repetition of the test.

**False negative**

- Destroyed vaccine.
- CMI not developed.

- Atypical mycobacterial infection.
- Miliary T.B.
- Decreased immunity

**Clinical pictures of meningitis**

1. Newborn " non specific "
   - Poor feeding and activity.
   - Temperature instability.
   - Hypotonia.
   - Convulsion.
   - Bulging anterior fontanel.

2. Infant & child up to 5 years.
   - Poor feeding.
   - Fever.
   - Irritability. High pitched cry.
   - Convulsions.
   - Bulging anterior fontanel.
   - Signs of meningeal irritation not reliable.

3. Child > 5 years
   - Fever.
   - Convulsion.
   - Increased intra cranial tension ( bursting vomiting, papilledema, projectile vomiting ).
   - Signs of meningeal irritation
     - Nuchal rigidity.
     - Positive Kernig's sign.
     - Positive Brudzinski's sign.

**D.D. of maculopapular rash**

<table>
<thead>
<tr>
<th></th>
<th>Measles</th>
<th>German measles</th>
<th>Ros. Infantum</th>
<th>IMN</th>
</tr>
</thead>
<tbody>
<tr>
<td>C.O.</td>
<td>Rubeola</td>
<td>Rubella</td>
<td>Human herpes 6</td>
<td>EBV</td>
</tr>
<tr>
<td>MOT</td>
<td>Contact or droplet</td>
<td>Transplacental</td>
<td>Droplet</td>
<td>Droplet</td>
</tr>
</tbody>
</table>

الجداول مهمين comp. vaccine
**Scarlet fever**

### Clinical pictures

1. **Rash**
   - Red.
   - Pin point "goose skin".
   - Blanch on pressure.
   - Pastia's sign ➔ transverse dark lines in creases of antecubital fossa.
   - More intense in the axilla and groin.

2. **Forehead & cheeks**
   - Circumoral pallor.
   - Strawberry tongue.
     - White ➔ coated papillae.
     - Red ➔ after shedding of papillae.
   - Acute follicular tonsillitis.

### Complications

- **Acute ➔ Suppurative.**
  - Acute O.M.
  - Acute sinusitis, mastoiditis & bronchilitis.
  - Arthritis.
- **Remote ➔ non suppurative**
  - Post strept.
    - G.N.
    - Rh.F.
  - Erythema nodosum
    - Red and tender

### Diagnosis

- Typical cases ➔ early diagnosed.
- Mild and atypical ➔ must be differentiated from other causes of maculopapular rashes.
- Leucocytosis ➔ 1500 !!!!.
- ASOT ➔ > 350 Todd units.
Treatment

1. Anti pyretic – analgesics.
2. Penicillin
   - Procain penicillin ... 400,000 u / day I.M. for 10 days.
   - Oral penicillin .... 400,000 u / day oral for 10 days.
   - Benzathine penicillin ... 600,000 – 1,200,000 u / I.M. once.
Erythromycin in penicillin allergy ... 40 mg / kg orally in 4 divided doses for 10 days.

Prevention

- Avoid overcrowding.
- Close observation of contacts.

Measles

Clinical pictures

- I.P ➔ 10 – 12 days.
- Prodromal stage ... Koplik’s spots
  ✓ 3 – 5 days.
  ✓ Grayish white dots on erythematous base
- Final stage ➔ rash with increased fever
  ✓ 1st day ➔ behind the ears and along the hair line ,, then on the face , upper arms and upper part of chest.
  ✓ 2nd day
    ● On the back, abdomen.
    ● Arms and thighs.
  ✓ 3rd day
    ● On feet and begin to fade on the face.
    ● The rash fades downward in the same sequence in which it appeared.

Complications

A. Activation of tuberculous focus.
B. Pneumonia and O.M. ➔ main complications.
C. Corneal ulcers and stomatitis.
D. Diarrhea and dysentery.
E. Encephalitis.
F. Hemorrhagic measles.
G. PEM

Diagnosis

- Clinical pictures.
- Lab is rarely needed.

- Decreased WBCs with relative lypmhocytosis.

Treatment

1. Supportive نزل الحرارة - سباحة - أكل قليل
   - Anti pyretic ➔ paracetamol.
   - Bed rest.
   - Adequate fluid intake.
   - Easily digested diet and proper cleanliness.
2. Vitamin A ➔ single dose ➔ 50,000 to 200,000 u.
3. AB in presence of infection.

**German measles**

**Complications**
- Encephalitis.
- Polyarthritis.
- Congenital rubella syndrome.

**Prophylaxis**
- Active vaccination
  - MMR.
  - Not in pregnant woman.
  - Avoid pregnancy for 3 months after vaccine.
- Passive
  - Indicated in non immune pregnant woman.
  - Immune serum globulin "ISG" I.M. one week after exposure.
**EVIDENCE OF HEMOLYSIS:**

<table>
<thead>
<tr>
<th>CBC</th>
<th>↓(↓): (HB, RBCs, HCT).</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>- Reticulo-cytois.</td>
</tr>
<tr>
<td>↑(↓)</td>
<td>(uro-bilinogen &amp; sterco-bilinogen).</td>
</tr>
<tr>
<td>↑(↓)</td>
<td>(Serum Iron). (↓) TIBC.</td>
</tr>
<tr>
<td>↑(↓)</td>
<td>indirect bilirubin.</td>
</tr>
</tbody>
</table>

- Bone marrow aspiration →
  - erythroid hyperplasia.

**LAB FINDING.**

**Aplastic Anemia (2007 - 02)**

**ETIOLOGY:**

1ry
- Idiopathic.
- Fanconi syndrome.

2ry "acquired"
- Drugs: AB "sulpha", Anti Rh "penicillamine", Anti convulsants.

**CLINICAL PICTURES:**

1ry
- Onset: (3 – 12 years).
- Pallor ➔ purpura, bleeding from mm.
- Short stature & hypo-gonadism.
- Congenital anomalies at birth.

2ry "acquired"
- ↓ RBCs ➔ Anemia "normo / Macro" ➔ pallor.
- ↓ WBCs ➔ recurrent infection "sore throat + scanty pus ".
- ↓ Platelets ➔ purpura & hemorrhage.

**D.D.:**

- Acute leukemia
  - Tender sternum.
  - B.M hypoplasia WITH leukemic cells "Aure's cells".
  - B.M replacement – normoblast / myelocytes (peripheral blood).
- Hypersplenism ➔ ↑ Reticulocytes.
- Auto immune pancytopenia – Ab against blood cells (peripheral blood).

**Causes & D.D. of microcytic hypochromic anemia (2011 - 08 - 04 - 03 - 02)**

<table>
<thead>
<tr>
<th>Fe deficiency anemia</th>
<th>Anemia Of Chronic Dis.</th>
<th>Thalassemia trait (α &amp; β)</th>
<th>Sidero-blastic anemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>M.C.V</td>
<td>↓↓</td>
<td></td>
<td>↓ (Congenital) - ↑ (Acquired)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Serum</th>
<th>Iron</th>
<th>Ferritin</th>
<th>TIBC</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>↓↓</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Iron</th>
<th>BM</th>
<th>RBCs</th>
<th>Present</th>
<th>Present</th>
<th>Ring forms</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
CAUSES:
1. Diet : PEM.
2. Demands (↑) : Infancy & Adolescence.
4. Lack of storage : Preterm & Low birth weight.
5. Chronic blood loss : repeated epistaxis.

DIAGNOSIS:
1. Clinical findings.
2. Lab.
   - Blood picture: micro – hypo chromic.
   - Reticulocytes: normal / moderately ↑.
   - Iron & ferritin (Serum): ↓
   - TIBC : ↑
3. Trial ➔ oral iron.
   - 1st day: improvement of neurologic functions & appetite.
   - 2nd day: ↑ Reticulocytes in B.M.
   - 3rd day: ↑ Reticulocytes in peripheral blood.
   - 4th day: ↑ Hemoglobin 4 g / dl / day.
   - ↑ Ht 1% / day.
   - Repletion of Fe stores in 1 – 3 months.

D.D.: (Anemia)
- α & β thalassemia.
- Sideroblastic Anemia.
- Lead poisoning Anemia.
- Copper deficiency Anemia.
- Anemia of late chronic infection.
- Anemia of chronic blood loss.

TREATMENT:
1. PROPHYLACTIC:
   - Iron (Rich in Diet) ➔ liver.
   - Iron (Prophylactic therapy).
   - Intestinal Parasites TTT.
   - Full term ➔ at 4 ms (1 mg / kg / day).
   - Pre term ➔ at 2 ms (2 mg / kg / day).
2. ACTIVE TTT:
   - Iron (Oral) ➔ 6 mg / kg (elemental).
   - Iron (Parenteral) ➔ - 2 ml in young child
     - 1 ml in infant. "for (3 – 5) days".
   - Packed RBCs transfusion ➔ 10 mg / kg (given slowly).
**HEREDITARY SPHERO-CYSTOSIS (2010)**

**DEFINITION:** Chronic Hemolytic anemia transmitted as AD trait due to dysfunction of cell membrane protein.

**CLINICAL PICTURES:**
- Neonatal Jaundice.
- General features of anemia: Jaundice, Gall stones, Aplastic crisis, Folate deficiency, Splenomegaly, Hemo-siderosis.

**LAB FINDINGS:**
- Anemia \(\rightarrow\) normocytic normochromic.
- Reticulocytes \(\rightarrow\) 5 – 20 %.
- Osmotic fragility.
- Unconjugated hyper-bilirubinemia.
- Peripheral blood \(\rightarrow\) Spherocytes.
- B.M. \(\rightarrow\) erythroid hypoplasia.

**TREATMENT:** S & S
- Supportive: Packed RBCs transfusion, Folic acid supplement.
- Splenectomy: Vaccine (Pneumococcal &H. Influenza), Antibiotics (Long Acting Prophylactic).

**β-thalassemia major TTT (2001 - 91)
"SYMPTOMATIC & CURATIVE"

**SYMPTOMATIC:**
1. Packed RBCs transfusion: 10 – 15 ml / kg / dose - every 4 – 6 weeks - to keep hemoglobin < 9 %.
2. Iron chelation
   - Desfero-xamine \(\rightarrow\) S.C. 30 – 50 mg / kg / dose \(\rightarrow\) 5 days / week.
   - Desfera-zirox \(\rightarrow\) Oral 20 – 30 mg / kg / dose.
   - Low iron diet WITH \(\uparrow\) tea taking.
3. Folate \(\rightarrow\) 1 mg daily.
4. DNA technology:
   - \(\leftrightarrow\) α chain BY Hydroxyl-urea β- Hydroxy-buterate.
   - \(\uparrow\) Hb F \(\rightarrow\) \(\downarrow\) Infective erythro-poiesis.

**CURATIVE:** (Transplantation) • B.M. • Stem cell.

**DIAGNOSIS OF HEMOPHILIA A (97)**

<table>
<thead>
<tr>
<th>CT:</th>
<th>Prolonged</th>
</tr>
</thead>
<tbody>
<tr>
<td>PTT:</td>
<td>Prolonged &gt; 120 sec. &quot;N= 25 – 45 &quot;.</td>
</tr>
<tr>
<td>PT:</td>
<td>Normal</td>
</tr>
<tr>
<td>Factor 8 assay:</td>
<td>(\downarrow)</td>
</tr>
<tr>
<td>Prenatal diagnosis:</td>
<td>Molecular biologic techniques ON chronic villus</td>
</tr>
</tbody>
</table>
**Bleeding diathesis (diagnosis) (2007)**

**PRESENT HISTORY:**
- Duration & Course (Since Birth / Recent).
- Fever following infection, Pain (abdominal & joint).

**PAST HISTORY:**
- Operation ➔ Bleeding (Prolonged & Excessive) After circumcision.

**FAMILY HISTORY:**
- Inheritance of: (any bleeding tendency).

**PHYSICAL EXAMINATION:**
- General condition.
- Rash Distribution.
- Fever.
- Organ enlargement.

**LAB TESTS FOR SCREENING:**
- Full blood picture.
- Hemo-stasis AT small blood vessels.
- Bleeding time.
- Thrombo-plastin generation test.
- Specific factor assay ➔ from factor 1 -TO- 13.

---

**Causes of thrombocytopenia (2007)**

<table>
<thead>
<tr>
<th>↓ Platelets production</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Congenital</td>
</tr>
<tr>
<td>- T.A.R.</td>
</tr>
<tr>
<td>- Fanconi.</td>
</tr>
<tr>
<td>- Congenital leukemia.</td>
</tr>
<tr>
<td>2. Acquired</td>
</tr>
<tr>
<td>- Aplastic</td>
</tr>
<tr>
<td>- Marrow infiltration</td>
</tr>
<tr>
<td>- Nutritional</td>
</tr>
<tr>
<td>- Renal failure.</td>
</tr>
<tr>
<td>➔ idiopathic, drugs, toxins.</td>
</tr>
<tr>
<td>➔ leukemia, lymphoma.</td>
</tr>
<tr>
<td>➔ vit. B12, folic acid.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>↑ Platelets Destruction</th>
</tr>
</thead>
<tbody>
<tr>
<td>3. Immune</td>
</tr>
<tr>
<td>- I.T.P.</td>
</tr>
<tr>
<td>- SLE.</td>
</tr>
<tr>
<td>4. Non-immune</td>
</tr>
<tr>
<td>- DIC.</td>
</tr>
<tr>
<td>- Hemolytic uremic syndrome.</td>
</tr>
<tr>
<td>- Infection, hyper-splenism, drug induced.</td>
</tr>
</tbody>
</table>
**I.T.P (2006 - 98)**

**Definition:**
Auto immune dis. Preceded By (URTI) upper respiratory tract infection.

**Clinical Pictures:**
- Recurrent Bleeding
  - Skin: Petechiae, Purpura, Ecchymosis, Hematoma.
  - Orifice: Epistaxis, Gingival, Uterine, Hematoma.
- No Pallor except in Evan’s syndrome.
- No HSM except if chronic.
- No Lymph nodes enlargement / sternal tenderness.

**Lab Investigations:**
- Platelets \(\rightarrow\) ↓ count. (Ig G against platelets)
- B.M \(\rightarrow\) Hyperplasia of megakaryocyte.

**Management:**
1. Conservative TTT.
2. Whole blood, platelets rich plasma, packed RBCs.
3. Prednisolone 2 mg/kg. (Until bleeding is stopped).
4. I.V. IG 900 mg/kg over 4 – 8 hours in 3 consecutive days.
   - Booster dose (every 2 – 4 weeks) "May be needed".
5. Immune suppressive drugs.
7. Splenectomy "improve 70 – 80% of cases".

---


1. B.M infiltration
   - ↓ RBCs \(\rightarrow\) anemia \(\rightarrow\) pallor.
   - ↓ WBCs \(\rightarrow\) infection.
   1. Fever \(\rightarrow\) sore throat not responding to TTT.
   2. Perianal abscess.
   3. ↓ Platelets \(\rightarrow\) bleeding "Epistaxis & purpura".

2. Tissue infiltration
   - Bone: fracture & sternal tenderness.
   - Liver, spleen & LN: enlargement.
   - Porta-hepatis: obstructive jaundice.
   - L.N: meningeal infiltration.
   - Heart: cardiomypathy.
   - Kidney: tubular damage \(\rightarrow\) ↓ Na & K.
   - Skin: itching.
   - Retina: ↓ vision.
   - Lung: hemoptyisis.
   - Testicular enlargement.
   - Leuco-stasis.

---

**Causes & D.D. of generalized lymphadenopathy (2004 - 02)**

الجدول الموجود في الكتاب صفحة 142 - صورة حلول في كتاب موافي صفحة 56

- Infection:
  - Viral: Hepatitis - IMN.
  - Bacterial: TB, Brucellosis.
  - Protozoa: Toxoplasmosis.
  - Fungal: Histo-plasmosis.

- Others:
  - Amyloidosis, Sarcoidosis, Hyperthyroidism.
  - Drugs: phenytoin.

- Tumor:
  - Leukemia.
  - Lymphoma.
  - Metastasis.

- Immunological:
  - SLE.
  - Sjogran syndrome.

---

**Note:**
أ krat معرف السؤال اللي كان جاي في البابطة بتاع البلد اللي جالها الامام علي.
Respiratory

Rapid breathing (Def.) (1999)

**TACHYPNEA:** ↑ R.R > normal according to age.

<table>
<thead>
<tr>
<th>Age</th>
<th>0 – 2 m</th>
<th>2 m – 1 Y</th>
<th>1 Y – 5 Y</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cycle / Min</td>
<td>≥ 60</td>
<td>≥ 50</td>
<td>≥ 40</td>
</tr>
</tbody>
</table>

Stridor (Def., causes) (2003 - 1999)

**DEF.** inspiratory harsh sound. (↑ On crying).

**CAUSES:** (Air Passing Through: a partially obstructed "larynx / trachea")

1. **Obstruction**
   a) Inside (Lumen): F.B. / mucous.
   b) Wall (edema):
      ✓ Allergic: angioedema.
      ✓ Inflammatory: laryngitis – trachetitis.
   c) outside (Pressure): by
      ✓ Retrosternal goiter.
      ✓ Media-stinal tumors.
      ✓ Anomalous: Aortic arch / Vascular ring.

2. **Neuromuscular:** Hyper excitability of nerves E.G. tetany → (laryngismus stridutus): spasm of laryngeal muscle.

3. **Congenital laryngeal stridor:**
   ✓ Laryngeo-malacia.
   ✓ Tracheo-malacia.

CROUP Syndrome (Def, Causes, C/P, viral type, TTT) (2012-2000)

**DEF.:** Heterogeneous group OF acute infectious conditions characterized by: characteristic brassy coughs & accompanied by:

- Inspiratory stridor, hoarseness & signs of respiratory distress due to varying degree of laryngeal obstruction.

**CAUSES & TYPES:**

<table>
<thead>
<tr>
<th>CROUP Type</th>
<th>Cause</th>
<th>Name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Viral CROUP</td>
<td>Para influenza virus type 1&amp;2.</td>
<td>Acute laryngo tracheobronchitis (ALTB)</td>
</tr>
<tr>
<td>Bacterial CROUP</td>
<td>H.I. type B.</td>
<td>Acute epiglottitis.</td>
</tr>
<tr>
<td>Membranous CROUP</td>
<td>Staph. Aureus. &amp; H.I...... Follows a viral URTI. (Bacterial complication of viral...)</td>
<td>Bacterial tracheitis</td>
</tr>
<tr>
<td>Diphtheric CROUP</td>
<td>Coryne-bacterium diphtheria.</td>
<td>Acute diphtheric laryngitis</td>
</tr>
<tr>
<td>Spasmodic CROUP</td>
<td>Unclear, may be allergic because recurrent nature.</td>
<td>Acute spasmodic laryngitis</td>
</tr>
</tbody>
</table>

**CLINICAL PICTURES OF VIRAL CROUP:** (typical episode) begins after URTI s & Duration: < 5 days.

1. Brassy cough, hoarseness, inspiratory stridor & respiratory obstruction (slowly / acutely).
2. ↑ obstruction → restless WITH ↑ H.R & R.R.
3. Cyanosis (isolated sign & denote complete airway obstruction).
4. C.P: worse at night.
**ETIOLOGY:**
- Respiratory Syncytial virus (RSV) (most common cause).
- Others:  
  - Viruses: (Para influenza - Influenza - Adeno-)
  - Mycoplasma - Chlamydia.

**CLINICAL PICTURES:**
- Rhinorrhea (flu like symptoms) is followed by:
  - Severe respiratory distress.
  - Expiratory wheeze (sibilant rhonchi).
  - Apnea.

**SYMPTOMS:**
- Naso-pharynx. (3-5) days.
- Gradual developing of: (dyspnea – respiratory distress – wheeze ± cyanosis) (2) days.
- Improvement of Symptoms. (7th day)
- Cough may continue. For another week.

**SIGNS:**
- ↑ R.R & Respiratory distress: +ve.
- ↓ Chest movement & T.V.F.
- Trachea: central.
- Percussion: Hyper resonance
- Auscultation:
  - ↓ Air entry & harsh vesicular breath sound.
  - Adventitious sound: wheezed – crepitation.

**Acute epiglottitis (etiology, diagnosis, treatment) (2010)**

**ETIOLOGY:**  
(H. influenza type B).

**DIAGNOSIS:**
- Direct laryngo-scopy:
  - Inflamed & swollen (supra-glottic structures).
  - Cherry red swollen (epi-glottis).
- Lateral neck " air way radiograph":
  - "Thumb sign" of swollen (epi-glottis).
  - Normal (sub-glottis).
- CBC: leukocytosis.
- Blood culture: +ve "80 %". (H.I. type B)
- Latex particle agglutination ➔ rapid diagnosis of (H.I. type B).

**TREATMENT:**
1. Epinephrine (Racemic) & corticosteroids " limited value".
2. Antibiotics (Parenteral): ceftriaxone (cefotaxime) / Ampicillin sulbactam (should be given at once).

**Breathe holding attack (Def., C/P, management) (2002 - 2000)**

**DEF.:** benign condition occur at (6 months: 6 years) with sequence of attack ➔ disturbance of (infant, child).

**CLINICAL PICTURES:**
- Disturbance of child ➔ crying ➔ holding breathing FOR few seconds.
- With attacks of either: - Cyanosis (blue type). / - Pallor (pallid type) & associated with few muscle jerks.
- Precipitated when child is upset.

**MANAGEMENT:**
- Investigations:
  - Good mentality.
  - Normal (EEG, ECG & ECHO).
  - Iron deficiency anemia (Some causes related to / associated with).
- Treatment: Reassurance of mother.
CAUSES OF RECURRENT PNEUMONIA: due to \( \uparrow \) TTT.
- Underlying Condition: e.g. T.B. / bronchiectasis.
- Organism Resistant to: Antibiotic: e.g. Tubercle bacilli.
- Complication Development: e.g. Pleural effusion, Empyema, Lung abscess.

Etiology to Age:

<table>
<thead>
<tr>
<th>Age</th>
<th>Neonates</th>
<th>4 – 16 weeks</th>
<th>&gt; 5 Y</th>
</tr>
</thead>
<tbody>
<tr>
<td>Organism</td>
<td>group B streptococci (GBS) &amp; E.coli</td>
<td>S.aureus.</td>
<td>H.I., S. pneumonia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>H.I., S. pneumonia</td>
<td>S. pneumonia</td>
</tr>
</tbody>
</table>

D.D. of Recurrent Chest Infection:

* PULMONARY CAUSES:

1. F.B. \( \Rightarrow \) History of choking. (X ray)
   - Complete obstruction \( \Rightarrow \) collapse (atelectasis).
   - Partial obstruction \( \Rightarrow \) emphysema.
2. Frequent \( \Rightarrow \) history of choking with feeding
   - Tracheo-esophageal fistula (TOF).
   - Gastro-esophageal reflux disease (GERD).

3. Persistent Collapse: (X-ray): triangular opacity \( \Rightarrow \) apex toward hilum.
4. Chronic infection:
   - Chronic sinusitis \( \Rightarrow \) Aspiration of post-nasal discharge.
   - T.B.
5. Genetic disease:
   - Immotile cilia syndrome = Kartagner syndrome: (Dextro-cardia + azo-spermia + immotile cilia).
   - Cystic fibrosis \( \Rightarrow \) viscid secretion.
   - \( \alpha-1 \) antitrypsin deficiency.

* CARDIAC CAUSES:

1. Pulmonary congestion (LSHF).
2. Pulmonary plethora (LT: RT shunt "ASD, VSD, PDA").
3. Cardiomegaly.

* IMMUNE DEFICIENCY:

1. 1ry (congenital)
   - Defective B cell (humoral immunity) (H.I.).
   - T cell deficiency (CMI).
   - Phagocytosis & complement defects.
2. 2ry (Acquired)
   - AIDS – PEM – N.S.
   - Corticosteroid therapy.
   - Malignancy.

TREATMENT:

1. Antibiotics according to age (Amoxillin 50 mg / kg / ceftiraxone 30 mg /kg).
2. O2 therapy.
3. Hydration.
4. Antipyretics.
5. Causal TTT.

ANTIBIOTICS ACCORDING TO AGE

1. Newborn
   - Ampicillin: 200 mg / kg / day
   - Gentamycin: 4 – 6 mg / kg / day.
   - OR 3rd generation cephalosporin:
     - Ceftriaxone: 100 – 200 mg / kg / day.
   - With staph. Aureus:
     - vanco-mycin: 60 mg / kg / day.

2. Infancy + childhood
   - Not severe pneumonia (just tachypnea) \( \Rightarrow \) TTT at home.
     - Amoxicillin: 50 mg / kg / day (oral) (for 5 – 7 days) & follow up in 2 days.
     - OR Cotrimoxazole.
   - Severe pneumonia (chest Indrawing) \( \Rightarrow \) TTT at hospital.
     - Amoxicillin: 50 mg / kg / day (I.M. /I.V.)
   - IF Suspect H.I. (Resistant to TTT) \( \Rightarrow \)
     - Chloramphenicol: 100 mg / kg / day.
     - Atypical mycoplasma pneumonia \( \Rightarrow \)
     - Erythromycin: 40 mg / kg / day.
CRITERIA FOR DIAGNOSIS:

1. Major criteria (Cancer):
   - Carditis.
   - Polyarthritis.
   - S.C. Nodules.
   - Chorea.
   - Erythema marginatum.

2. Minor criteria (Peace):
   - Arthralgia.
   - Fever (Elevated temperature).
   - Acute phase (ESR, CRP).

3. Evidence of: (Group A β-streptococcal infection): (Recent scarlet fever, "rapid Ag test +ve throat swap, ↑ ASOT")

   For diagnosis
   - 2 major criteria + Evidence of: GABS infection.
   - 1 major criteria & 2 minor criteria + Evidence of: GABS infection.

BUT (EXCEPTION)

✓ Chorea (Rheumatic).
✓ Carditis (Late onset / insidious with no explanation).
✓ Rheumatic recurrence: - Rheumatic heart disease.
   - Presence of 1 major / 1 minor + Evidence of: GABS infection.

TREATMENT:

1. Bed rest.

2. Antibiotics (to eradicate GABS infection)
   - Penicillin / erythromycin orally for 10 days.
   - OR Benzathine penicillin I.M. single dose.

3. Anti-inflammatory agents:
   - a) Salicylates (Arthritis & mild conditions)
     - (100 mg / kg / 24 hours in 4 divided doses orally for 4 – 6 weeks).
   - b) Corticosteroids (severe conditions: H.F. / cardiomegaly)
     - (2 mg / kg / 24 hours in 4 divided doses for 2 – 3 weeks).
     - followed by salicylates
     - (75 mg / kg / day for 4 – 6 weeks).

4. Congestive H.F (3Ds):
   - Bed rest.
   - Diuretics.
   - Digitalis (Small dose).

5. Sydenham Chorea:
   - Pheno-barbital (16 – 32 mg / 6 – 8 hr).
   - Halo-peridol (0.01 – 0.03 mg / kg / 24 hrs divided bid).
   - Sodium valproate (15 – 20 mg / kg / day).

6. In resistant cases:
   - Plasma-pharesis.
   - I.V. Ig.
   - Reserpine.

7. residual V.H.D.S.:
   - Vulpulo-plasty.
   - Valve replacement.

PREVENTION:

1. 1ry: tonsillitis (proper TTT).
2. 2ry: Suggest duration of 2ry prophylaxis.

Category of patient duration of prophylaxis
a) Patient without proven carditis ➔ 5 years after last attack.

b) Patient with carditis / V.H.D.s ➔ lifelong.

c) After valve surgery ➔ lifelong.
Peripheral signs of A.R. (2000)

9 = (3 NECK, 3 U.L., 3 L.L.)

1. De Musset **SIGN:** Nodding of head.
2. Corrigan's **SIGN:** Prominent pulsation of carotid in neck.
4. Big: Pulse pressure.
7. Pistol shot: sound heard over femoral artery in groin.
8. Duroziez **SIGN:** systolic & diastolic murmur heard over femoral artery.

Heart failure (Clinical pictures, treatment) (2012-10-06-02-01)

**Clinical Pictures:**

1. Symptoms: Varies according to age

   A. In infants:
   - Failure to thrive.
   - Feeding difficulties.
   - Fussiness / agitation.
   - Diaphoresis (excessive sweating).
   - Dyspnea on feeding.
   - Ruffiness of face.
   - Persistent cough & wheeze & shortness of breathing.
   - Pedal edema.

   B. Older children:
   - Loss of appetite & abdominal pain.
   - Exercise intolerance.
   - Syncope.
   - Dyspnea on exertion.

2. Signs:

   A. Lung congestion
   - Tachypnea.
   - Respiratory distress.
   - Crepitation (Rales).
   - Wheezes.

   B. Systemic congestion
   - Edema.
   - Ascites.
   - Congested neck veins.
   - Hepatomegaly (tender liver).

   C. Low COP
   - Poor perfusion.
   - Mottling cold extremities.
   - ↑capillary refilling time.
   - Hypotension.
   - Weak pulse.

   D. Cardiac signs
   - Tachycardia.
   - Cardiomegaly (pericordial bulge, apex displacement).
   - Gallop rhythm.
   - A.V. valve regurge (functional regurge).
   - Hyperactive pericordium.

**Treatment:**

1. General measures: \(\rightarrow\) ↓ Cardiac Work.

   A. Hospitalization.
   C. Humidified O2 therapy.
   D. Sedative in acute L.V.F. (only): morphine sulphate.
   E. Diet (high calorie) – low salt (< 0.5 mg / day).
   F. Precipitating factors TTT.
   G. Causal TTT.
   H. Monitoring of: (vital signs – weight – liver size – JVP)

2. Drug Therapy:

   A. Inotropic Agents:

   (improve COP) ↑ contractility.

   - Digitalis
     - Action: ↑ contractility, diuretic effect, ↓ H.R. (A.V. Block).
     - Dose: 30 – 40 mg / kg
     - (0.5 dose "initially" → 0.25 dose "after 8 h" → 0.25 dose "after 10 h from start"
       (يغني بعد 8 ساعات)
     - Maintenance dose: 5 – 10 mg / kg / day, "divided every 12 hours".
     - C.I.: Heart block, P.V.T., junctional (Nodal rhythm).
     - I.V. dose \(\rightarrow\) 75% of oral dose in form of (Drops 0.05 mg / ml. - Tablets 0.25 mg / tab)
   - Sympathomimetic (Dopamine – Dobutamine).
   - PDI: mil-rinone.
B. DIURETICS: (↓ load – cardiac work)

<table>
<thead>
<tr>
<th>Loop DIURETICS</th>
<th>Potassium sparing DIURETICS</th>
<th>Thiazide DIURETICS</th>
</tr>
</thead>
<tbody>
<tr>
<td>furosemide</td>
<td>spironolactone</td>
<td>hydrochlorothiazide</td>
</tr>
</tbody>
</table>

C. VASODILATORS: (↓ preload – after load) / both

<table>
<thead>
<tr>
<th>Arterio DILATORS</th>
<th>Veno DILATORS</th>
<th>Balanced VASODILATORS (ACEI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hydradazine (↓ after load).</td>
<td>Nitroglycerine (↓ pre load).</td>
<td>Captopril (↓ both pre &amp; after load)</td>
</tr>
</tbody>
</table>

3. OTHER MEASURES:
   a) Mechanical counter pulsation.
   b) Cardiac transplantation.
   c) Extra corporal membrane oxygenation bypasses heart.
   d) Peritoneal dialysis with hypertonic solution "in case of fluid retention (APO)".

Congestive heart failure treatment (2001 - 98)

= Lt. & Rt. sided H.F ➔ same TTT.

Digitalization in CHF (1997 - 94)

As Above in TTT of H.F.

Infective endocarditis (Diagnostic criteria & treatment) (2008 - 1999)

A. DIAGNOSTIC CRITERIA
   (Clinical laboratory diagnosis)

A. CLINICAL PICTURES:
   (Classic tetrad of C.P of endocarditis)

1. Toxic
   • FAHM.
   • Weight loss.
   • Arthralgia.
   • Splenomegaly.
   • Clubbing.

2. Embolic
   • Renal infarction ➔ frank hematuria.
   • Splenic infarction ➔ left flank pain.
   • Central retinal artery occlusion ➔ sudden blindness.

3. Immunological
   • Splinter hemorrhage.
   • Roth's spot.
   • Petechial lesions with pale centers.
   • Osler's nodules.
   • Janeway lesions.
   • Diffuse G.N.

4. Cardiac:
   • Signs of preexisting (V.H.D. / shunt).
   • Murmer: Sudden occurrence of new / change in character of already existing.
   • H.F: (3Ts) "Tachycardia - Tachypnea - Tender liver".

B. INVESTIGATIONS:

1. Blood cultures: (2-5) samples on (2 – 3) successive days.
2. CBC: anemia, leukocytosis (May leucopenia).
3. ESR: (↑)
4. ECHO: • Vegetation / peri-valvular abscess. • already pre-existing cardiac defects.

© TREATMENT:

A. PROPHYLAXIS:

1. High dose of antibiotics, (1 hour before & 6 hours after procedure) according to procedure.
   • Dental: Amoxa-cillin 50 mg / kg / dose.
   • Genitourinary: Amoxa-cillin + Genta-mycin 1.5 mg / dose.
2. Oral hygiene.
3. Correction of congenital defects E.G. PDA.

B. CURATIVE:

1. Antibiotics (Immediately AFTER taking sample for blood culture).
   - Crystalline penicillin G. (200,000 – 300,000 u / kg / day) I.V
   - With gentamycin (2 – 4 mg / kg /day). -Then according to culture & sensitivity.
   - TTT Duration: (4 – 6 weeks).
2. Surgical e.g. (Aortic Abscess).
CLINICAL PICTURES:
Carditis = Pan-Carditis ➔ (Peri – Myo – Endo) Carditis

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>- Chest pain</td>
<td>- Palpitaton</td>
<td>- Valves affected (M &gt; A&gt; T&gt; P).</td>
<td></td>
</tr>
<tr>
<td>Signs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Pericardial rub.</td>
<td>- Tachycardia disproportionate to: fever.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Pericardial effusion Signs.</td>
<td>- Weak heart sound.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Gallop rhythm &amp; H.F.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

LAB INVESTIGATIONS:
- ↑ (ESR - CRP - CPK - ASOT).
- Leukocytosis.
- Other investigations (may show)
  - ECHO: Cardiomegaly – vegetation & valve lesions.

CLINICAL DIAGNOSIS:
(Sure signs of rheumatic carditis)
- History of Rheumatic fever.
- Cardiomyopathy.


1. CARDIAC EDEMA: Congestive H.F (Start in L.L)
   - Cardiomegaly, H.F., dyspnea.
   - Gallop rhythm.
   - Congested neck veins.

2. LIVER CELL FAILURE (LCF) "HEPATIC":
   - Ascites.
   - Liver cirrhosis.
   - Sharp border.
   - Splenomegaly.
   - Manifestations of LCF.

3. ALLERGIC: Urticaria (Drug intake).

4. NUTRITIONAL:
   - Hypo-protein-emia (Kwashiorkor & Murmice kwashiorkor)
   - Starts in: dorum of feet &legs.
   - Ascites: No in: kwashiorkor,
   - Rarely in: septic peritonitis & during nutritional recovery syndrome.

5. RENAL (G.N., N. SYNDROME, R.F.):

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Nephrotic</th>
<th>Nephritic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Edema</td>
<td>Severe</td>
<td>Mild</td>
</tr>
<tr>
<td>Proteinuria</td>
<td>Massive</td>
<td>Mild</td>
</tr>
<tr>
<td>Pleural effusion</td>
<td>present</td>
<td>No</td>
</tr>
<tr>
<td>Pericardial effusion</td>
<td>present</td>
<td>No</td>
</tr>
<tr>
<td>B.P.</td>
<td>Normal</td>
<td>Hypertension</td>
</tr>
</tbody>
</table>
Acute diarrhea (etiology, common pathogen, complications, management) (2011-10-08-03-02-95)

**ETIOLOGY:** (Common pathogen)

1. Infectious (most common)
   a) Viral: Rota-, Adeno-, entero-.
   b) Bacterial:
      - Invasive (bloody):
        ✓ Shigella.
        ✓ Salmonella.
        ✓ Campylobacter jejune.
        ✓ Enterohemorrhagic E.Coli.
      - Noninvasive (common) (watery):
        ✓ Enterotoxigenic E.Coli (ETEC).
        ✓ Vibrio Cholera & Parasite.
        ✓ Giradia lambia.
        ✓ Entameba histolytica.
        ✓ Crypto-sporidium.

2. Non infectious
   a) Dietetic
   b) Formula: Over concentrated.
   c) Food: Unsuitable weaning.
   d) Drugs.
   e) Toxins.
   f) Laxatives.
   e) Hurry intestine e.g. (thyrotoxicosis & irritable bowel syndrome).

**COMPLICATIONS:**

1. Dehydration:
   - Shock.
   - Pre renal failure.
   - DIC.

2. Electrolyte disturbance:
   - Hyper / hypo Kalemia. K
   - Hyper / hypo natremia. NA
   - Hypo calcmia. Ca
   - Acidosis (↓ PH). PH

3. Persistent diarrhea & PEM.

**MANAGEMENT:**

1. **PROPHYLACTIC**
   a) Promotion of breast feeding.
   b) Proper weaning
   c) Good Hygiene (for mother & baby).
   d) Proper vaccination (measles & recently Rota virus).

2. **LINES & TTT:**
   a) Diet: breast feeding.
   b) Formula: Low / lactose free.
   c) Drugs According To condition (Causal TTT).

- Anti-microbial: In Case Of:
  - Bloody diarrhea cholera (tetracylin) associated bacterial infection e.g.
  - (co-tri-mextazole) tri-meto-prim - subl-meto-xazole (10 mg - 50 mg / kg) (for 5 days).
  - Metronidazole "for trophozoite" (Giardiasis 15 mg / kg & 30 mg for amoebiasis). (for 10 days).
  - Salmonella ➔ chloramphenicol.
  - Campylobacter jejuni ➔ erythyo-mycin.

- Anti-diarrheal (not elective) e.g.
  - Constipating drugs ➔ ↑ toxemia.
  - Anti-motility drugs ➔ paralytic ileus.
3. **If Persistent**: Hospitalization in following:
   - Age: < 6 months.
   - Dehydration: (2nd degree).
   - Associated other severe infection (viremia).
   - Failure goes home.
   - Presence of PEM.

4. **Complications**:
   - Correct dehydration.
   - Hypernatremia $\rightarrow$ ↓ Na intake.
   - Hyponatremia $\rightarrow$ NaCl.
   - Hypokalemia $\rightarrow$ KCl.
   - Metabolic acidosis $\rightarrow$ Na HCO₃.
   - Convulsions: $\rightarrow$ Diazepam 0.2 – 0.4 mg / kg / dose 1.V.

<table>
<thead>
<tr>
<th>ORS (Composition &amp; function of each component) (2008 - 1998 - 91)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Composition</strong></td>
</tr>
<tr>
<td>Na Cl $\rightarrow$ 3.5 g / L</td>
</tr>
<tr>
<td>K Cl $\rightarrow$ 1.5 g / L</td>
</tr>
<tr>
<td>Tri sodium citrate $\rightarrow$ 2.9 g / L</td>
</tr>
<tr>
<td>Glucose (2%) $\rightarrow$ 20 g / L</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Differences between osmotic &amp; secretory diarrhea (2007)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Character of Diarrhea</strong></td>
</tr>
<tr>
<td><strong>Causes</strong></td>
</tr>
<tr>
<td><strong>Mechanism</strong></td>
</tr>
<tr>
<td><strong>Stool</strong></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
</tr>
<tr>
<td><strong>Response</strong></td>
</tr>
</tbody>
</table>

**INVESTIGATIONS:**

1. Stool PH.
2. Reducing substances.
3. Stool culture.
4. > 72 hours stool collection (For fat).
5. Smear (for WBCs, fat, ova & parasites).
6. Clostridium toxin, blood pictures (CBC), ESR.
7. Electrolytes.
8. BUN & creatinine.
10. Endoscopy & biopsy (for small bowel, sigmoid colon).
12. Hormonal studies: vasoactive intestinal peptide (VIP), gastrin, secretin.

**TREATMENT:**

1. Supportive:
   - Adequate nutritional support: promote growth.
   - ↓ Fluid intake (for chronic nonspecific diarrhea)
   - ↓/ remove Fruit juice.
   - ↓ Lactose / (sucrose & lactose) / (sucrose free diet).
   - ↑ Fat intake (if it was restricted).
   - Predigested formula (pre-gesta-mil).
2. Surgical: For partial obstruction / mal-rotation.
3. Medical:
   - Metronidazole (for giardiasis).
   - Parenteral Penicillin (for bacterial infection).
   - (for other causes).
4. Specific TTT:

### Dehydration (Signs, TTT, fluid therapy in severe form) (2003-99)

#### Signs:

<table>
<thead>
<tr>
<th></th>
<th>No dehydration</th>
<th>Some dehydration</th>
<th>Severe dehydration</th>
</tr>
</thead>
<tbody>
<tr>
<td>General consciousness</td>
<td>Alert</td>
<td>Irritable</td>
<td>Comatose</td>
</tr>
<tr>
<td>Eye</td>
<td>Not sunken</td>
<td>Sunken</td>
<td>Very sunken</td>
</tr>
<tr>
<td>Drink</td>
<td>Normal</td>
<td>Eagerly</td>
<td>Unable</td>
</tr>
<tr>
<td>Skin elasticity</td>
<td>Go back rapidly</td>
<td>Slow &lt; 2 sec.</td>
<td>Very slow ≥ 2 sec.</td>
</tr>
</tbody>
</table>

#### Diagnosis:
depends on presence ≥ 2 signs of dehydration.

#### Treatment:

<table>
<thead>
<tr>
<th>No dehydration</th>
<th>Some dehydration</th>
<th>Severe dehydration</th>
</tr>
</thead>
<tbody>
<tr>
<td>(loss &lt; 5% of body weight) 2 – 4%</td>
<td>(Loss &lt; 10%) 5 – 9%.</td>
<td>(Loss &gt; 10%).</td>
</tr>
<tr>
<td>&quot;Plan (A) TTT&quot; (At home)</td>
<td>&quot;Plan (B) TTT&quot; (At hospital)</td>
<td>&quot;Plan (C) TTT&quot; ➔ shock TTT (At hospital)</td>
</tr>
<tr>
<td>Excess fluid.</td>
<td>ORS:</td>
<td></td>
</tr>
<tr>
<td>✓ &lt; 2 y: 50 - 100 ml / loose stool.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>✓ ≥ 2 y: 100 – 200 ml / loose stool.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>✓ Instruct mother how to prepare ORS.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>✓ Give rice water, yoghurt, semisoluid food (if child eat).</td>
<td></td>
<td></td>
</tr>
<tr>
<td>✓ Continue breast feeding.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ORS:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>✓ 75 ml / kg (over 4 hours).</td>
<td></td>
<td></td>
</tr>
<tr>
<td>✓ By spoon &amp; cup (1 spoon/1 min).</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Continue breast feeding/ formula.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>I.V. fluid therapy.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ringer lactate.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Poly electrolyte solution.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pan solution.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>✓ &lt; 1 year</td>
<td></td>
<td></td>
</tr>
<tr>
<td>100 ml / kg / 6 hours.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30 ml / kg / 1 hour.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>70 ml / kg / 5 hours.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>✓ ≥ 1 year</td>
<td></td>
<td></td>
</tr>
<tr>
<td>100 ml / kg / 3 hours.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>20 – 30 ml / kg / on (0.5–1 hour).</td>
<td></td>
<td></td>
</tr>
<tr>
<td>70 ml / kg / on (2–2.5 hours).</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

#### Treatment of diarrhea without dehydration (2003)

**TREATMENT THROUGH "PLAN A" HOME MANAGEMENT**

- Fluid: ORS
  ✓ 1/4 – 1/2 cup for baby < 2 years. Other fluids
  ✓ 1/2 – 1 cup for child > 2 years. + Plain water – Helba (حلبة) – Anise (بنسون).
  - After each watery stool: (avoid high sugar content)

- Food
  ✓ Feeding (Breast): Continue.
  ✓ Formula: Frequent.
  ✓ Food: Easily digestible.

(Follow up ➔ diarrhea Not Getting better in 3 days).

- Generally: (more sicker).
- Fever: (persists > 2 days / appear).
- Vomiting: (persists / appears).
- Dehydration: (reappears).
- Food &drink: (refusal).
**GERD (Def., clinical pictures, TTT) (2008)**

<table>
<thead>
<tr>
<th>DEF.:</th>
<th>Retrograde movement (regurgitation) of gastric content across (LES) lower esophageal sphincter into esophagus.</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>CLINICAL PICTURES:</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1. Infants</strong></td>
</tr>
<tr>
<td>A. Regurgitation especially postprandial.</td>
</tr>
<tr>
<td>B. Signs of Esophagitis:</td>
</tr>
<tr>
<td>• Irritability.</td>
</tr>
<tr>
<td>• Arching – Chocking.</td>
</tr>
<tr>
<td>• Gagging.</td>
</tr>
<tr>
<td>• Feeding aversion.</td>
</tr>
<tr>
<td>C. Failure to thrive.</td>
</tr>
<tr>
<td>D. Extra esophageal</td>
</tr>
<tr>
<td>• Obstructive apnea.</td>
</tr>
<tr>
<td>• Stridor.</td>
</tr>
<tr>
<td><strong>2. Older children</strong></td>
</tr>
<tr>
<td>A. Regurgitation during preschool years.</td>
</tr>
<tr>
<td>B. Chest &amp; abdominal pain.</td>
</tr>
<tr>
<td>C. Extra esophageal</td>
</tr>
<tr>
<td>• Asthma.</td>
</tr>
<tr>
<td>• Oto-laryngo-logic disease.</td>
</tr>
<tr>
<td>• Laryngitis – sinusitis.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TREATMENT:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. DIETARY MEASURES:</td>
</tr>
<tr>
<td><strong>1. Infants</strong></td>
</tr>
<tr>
<td>• Normalization of feeding:</td>
</tr>
<tr>
<td>✓ Technique.</td>
</tr>
<tr>
<td>✓ Volume.</td>
</tr>
<tr>
<td>✓ Frequency.</td>
</tr>
<tr>
<td>• Formula: <strong>Thickening with</strong> table spoon to ↓ regurgitation.</td>
</tr>
<tr>
<td>• Greater caloric density (30 kcal/ oz).</td>
</tr>
<tr>
<td>• Short trial of hypo-allergic diet:</td>
</tr>
<tr>
<td><em>(To Exclude: milk &amp; soy protein allergy)</em></td>
</tr>
<tr>
<td><strong>2. Older children</strong></td>
</tr>
<tr>
<td>• Avoid acid food (Tomato).</td>
</tr>
<tr>
<td>• Avoid beverage (juices).</td>
</tr>
<tr>
<td>• Weight reduction for obese.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>PHARMACOTHERAPY:</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1. Infants</strong></td>
</tr>
<tr>
<td>• Antacid.</td>
</tr>
<tr>
<td>• H2 receptor blocker.</td>
</tr>
<tr>
<td>• PPI (proton pump inhibitors). 0.7 – 3.3 mg / kg / day (not for long period).</td>
</tr>
<tr>
<td><strong>2. Older children</strong></td>
</tr>
<tr>
<td>• For mild – moderate cases.</td>
</tr>
<tr>
<td>• For severe cases.</td>
</tr>
<tr>
<td><strong>SURGERY:</strong> fundo-plication.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>D.D. of vomiting in 1st month of life (2005)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. ↑ intra cranial (pressure / hemorrhage)</td>
</tr>
<tr>
<td>2. GERD &amp; gastroenteritis.</td>
</tr>
<tr>
<td>3. Intestinal obstruction (anatomical obstruction).</td>
</tr>
<tr>
<td>4. Swallowed amniotic fluid.</td>
</tr>
<tr>
<td>5. Over feeding.</td>
</tr>
<tr>
<td>6. Cow milk allergy.</td>
</tr>
<tr>
<td>7. Inborn error of metabolism e.g. Phenyl ketonuria.</td>
</tr>
<tr>
<td>8. Neonatal sepsis (systemic infection).</td>
</tr>
</tbody>
</table>
**Bleeding per rectum (causes) (2002 - 1998)**

**CAUSES:** (VARY WITH AGE)

1. Newborn
   - Bacterial enteritis (G.E.).
   - Ingested maternal blood.
   - Anal fissure, NEC, volvulus.

2. Infant
   - G.E.
   - Milk protein allergy.
   - Intussusception.

3. Child
   - Bacterial enteritis (G.E.).
   - N.S.A.I.D. injury.
   - Esophogitis – esophageal varices.
   - Mekel's diverticulum.
   - HSP – IBD.
   - Colonic polyps.

4. Adolescent
   - G.E.
   - Gastritis – peptic ulcer.
   - Esophagitis – esophageal varices.
   - Hemorrhoids (piles).
   - I.B.D.
   - Colonic polyps.

**Difference between organic and functional abdominal pain (1999)**

<table>
<thead>
<tr>
<th></th>
<th>Functional</th>
<th>Organic</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>abdominal pain</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>School age.</td>
<td>Any age.</td>
</tr>
<tr>
<td><strong>Severity</strong></td>
<td>Vague.</td>
<td>Severe.</td>
</tr>
<tr>
<td><strong>Site</strong></td>
<td>Peri umbilical.</td>
<td>Away from umbilicus.</td>
</tr>
<tr>
<td><strong>Other symptoms</strong></td>
<td>No other symptoms.</td>
<td>Associated fever, diarrhea, vomiting.</td>
</tr>
<tr>
<td><strong>Organo-megaly</strong></td>
<td>No organo-megaly.</td>
<td>± organo-megaly.</td>
</tr>
<tr>
<td><strong>General condition</strong></td>
<td>Good.</td>
<td>Bad.</td>
</tr>
</tbody>
</table>

**Gastroenteritis (Complications) (1996)**

1. Dehydration ➔ hypovolemic shock.
2. Electrolyte disturbance.
4. Meningitis.
5. Post infectious arthritis – osteomyelitis.
Liver


IT DIFFERS ACCORDING TO AGE OF CHILD:

<table>
<thead>
<tr>
<th>1. Neonates</th>
<th>2. Infants</th>
<th>3. Children</th>
</tr>
</thead>
<tbody>
<tr>
<td>b) Neonatal hepatitis.</td>
<td>b) PEM.</td>
<td>b) Drugs – toxins.</td>
</tr>
<tr>
<td>c) Biliary atresia.</td>
<td>c) Inborn errors of metabolism</td>
<td>c) Metabolic</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- α1 - antitrypsin deficiency.</td>
</tr>
<tr>
<td></td>
<td>f) Hepatic vein thrombosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(Budd Chiari syndrome).</td>
<td></td>
</tr>
<tr>
<td></td>
<td>g) Tumors: hepatoblastoma, lymphoma.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>e) Tumors: Leukemia - Lymphoma-Metastasis.</td>
</tr>
</tbody>
</table>

Viral hepatitis (etiology, diagnosis, prevention) (2012 - 93 - 95)

**ETIOLOGY:**


b) Non Hepato-tropic viruses: (EBV - CMV - Coxakie). (Echo, rubella, varicella & measles).

**INVESTIGATIONS:**

(for acute hepatitis)

1. Liver function test
   - T.P. Ptn ➔ normal.
   - Serum bilirubin ➔ mixed hyper-bilirubinemia.
   - P.T. ➔ Prolonged.
   - ↑ SGOT, SGPT, gamma GT & 5- nucleotidase.

2. Serology
   - HA ➔ HA Ig M (recent infection).
   - HB
     - HBS Ag.
     - HBC Ig M (acute infection).
   - PCR – ELIZA (C- D – E).

(for chronic hepatitis)

1. Chronic persistent
   - Liver enzymes: Normal /slightly ↑.
   - Liver biopsy:
     - Normal hepatic architecture.

2. Chronic active
   - Lab:
     - Liver function tests.
     - Total plasma protein: ↓
     - PT ➔ prolonged.
     - Serum bilirubin: ↑
     - Liver enzymes: ↑
     - Liver biopsy show: same as chronic persistent hepatitis.
   - Abdominal sonar ➔ liver cirrhosis.
PREVENTION:

1. **Hepatitis A**
   - Environmental sanitations.
   - Hepatitis A vaccine: (Plasma derived genetic engineering - Non obligatory)
     - (0.5 ml (10 u) for 2 doses in 6 months interval) "I.M".
   - Close contact: HA Ig G (0.02 ml / Kg / I.M.)

2. **Hepatitis B**
   - Exam of blood products (plasma, platelets, albumin).
   - Exam of disposal syringes.
   - Sterilization of surgical instruments.
   - Hepatitis B vaccine: (Genetic engineering)
     - (0.5 ml (10 u) /dose). "I.M"
     - HB Ig G (sero prophylaxis) (0.06 ml / kg / dose).
     - "I.M"

3. **Hepatitis C**
   - Same hygienic measures as B.
   - No available vaccine against HCV.
   - Caesarian delivery for mothers with HCV.

---

**Hepatitis A (Prevention) (2011)**

1. Infectivity: Contagious (7 days before & after onset of jaundice).
2. Isolation from school: (till 7 days after). **When** "dark color urine & jaundice appear",
3. Hygiene: Careful hand washing **AFTER** (changing disappears & before preparation of food).
4. Fly control.
5. Ig (I.V.). (Pre & post exposure).
6. Hepatitis A vaccine: now available ➔ children > 2 years. (I.V. Ig or vaccine ➔ contacts).
### Infectious diseases

**Q1: Febrile patient at high risk of serious bacterial infection**

**A. Immune component**
- Neonates.
- Hyperpyrexia > 40°C.
- Fever with petichiae.

**B. Immune compromised**
- Sickle cell disease.
- Asplenia.
- Immune defi ciency.
- Malignancy.

**Q2: FUO 2012 - 07**

**Def:**
Persistent elevation on of body temperature over 38.5°C for at least 2 weeks without diagnosis after proper history taking, physical examination & routine investigations.

**A/E:**

<table>
<thead>
<tr>
<th>Infection</th>
<th>Malignancy</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1. Bacterial:</strong></td>
<td></td>
</tr>
<tr>
<td>Systemic</td>
<td></td>
</tr>
<tr>
<td>✓ T.B., Tularemia,</td>
<td></td>
</tr>
<tr>
<td>Brucellosis&amp;</td>
<td></td>
</tr>
<tr>
<td>Campylobacter</td>
<td></td>
</tr>
<tr>
<td>Localized</td>
<td></td>
</tr>
<tr>
<td>✓ Sinusitis, Otitis</td>
<td></td>
</tr>
<tr>
<td>media, I.E&amp; UTI.</td>
<td></td>
</tr>
<tr>
<td><strong>2. Viral:</strong></td>
<td></td>
</tr>
<tr>
<td>EBV – CMV – HIV –</td>
<td></td>
</tr>
<tr>
<td>Hepatitis</td>
<td></td>
</tr>
<tr>
<td><strong>3. Parasitic:</strong></td>
<td></td>
</tr>
<tr>
<td>Amoebiasis – Giardiasis - Malaria</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Collagen disease</th>
<th>Granulomatous disease</th>
<th>Familial- Hereditary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rheumatic fever.</td>
<td>Sarcoidosis.</td>
<td>Ectodermal dysplasia,</td>
</tr>
<tr>
<td>PAN.</td>
<td>Granulomatous hepatitis</td>
<td>Cyclic neutropenia,</td>
</tr>
<tr>
<td>Juvenile RA.</td>
<td></td>
<td>Mediterranean fever.</td>
</tr>
</tbody>
</table>

**Investigations**
- C.B.C. - Culture ➔ blood, urine, CSF - Chest X – ray.

**Treatment**
- Tap water fomentation. - Antipyretic drugs. - Broad spectrum AB.
Q4: D.D of exanthematous diseases. 2002-01
Q5: Maculo-papular rash. (Causes - D.D) 1997-96

i. Maculo-papular exanthematous diseases:
   A. Infectious:
      1- Measles (rubeola)  2- Rubella (German measles)  3- Roseola
      4- Erythema infectiosum  5- Infectious mononucleosis  6- Scarlet fever
      7- Enteroviral infections  8- Typhoid fever
   B. Non Infectious:
      1- Sweat rash  2- Drug rash  3- Connective tissue diseases

ii. Vesiculopapular exanthematous diseases: I
   A. Infectious:
      1) Viral: Varicella (Chicken pox), HSV, Variola virus (small pox), and coxsackievirus
      2) Bacterial: Impetigo and staphylococcal scalded skin syndrome (SSSS).
   B. Non Infectious: Papular urticaria, drug eruption, burns, and insect bites.

D.D. of maculopapular rash

<table>
<thead>
<tr>
<th></th>
<th>Measles</th>
<th>G. measles</th>
<th>Ros. Infantum</th>
<th>IMN</th>
</tr>
</thead>
<tbody>
<tr>
<td>C.O.</td>
<td>Rubeola</td>
<td>Rubella</td>
<td>Human herps 6</td>
<td>EBV</td>
</tr>
<tr>
<td>MOT</td>
<td>Contact or droplet</td>
<td>Transplacental</td>
<td>Droplet</td>
<td>Droplet</td>
</tr>
<tr>
<td>I.P.</td>
<td>2 – 3 weeks</td>
<td>2 – 3 weeks</td>
<td>1 – 2 weeks</td>
<td>30 – 50 d</td>
</tr>
<tr>
<td>Prodroma</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Koplik's spot</td>
<td>Unnoticed</td>
<td>Rhinorrhea</td>
<td>FAHM</td>
</tr>
<tr>
<td></td>
<td>3 – 5 days</td>
<td>Very short</td>
<td></td>
<td>1 – 2 weeks</td>
</tr>
<tr>
<td>Rash</td>
<td>Appear on 2nd day</td>
<td>Appear on 2nd day</td>
<td>Appear on 4th day</td>
<td>Present in 10% of patients</td>
</tr>
<tr>
<td></td>
<td>Progress on 3rd day</td>
<td>Disappear on 3rd day</td>
<td>Disappear on 5th day</td>
<td></td>
</tr>
<tr>
<td></td>
<td>At 7th day ➔ branny desquam.</td>
<td>No branny desquam.</td>
<td>Rainbow flow.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Trunk ➔ arms and shoulder.</td>
<td></td>
</tr>
<tr>
<td>Treatment</td>
<td>Supportive.</td>
<td>MMR.</td>
<td>No anti-viral</td>
<td>Acyclovir.</td>
</tr>
<tr>
<td></td>
<td>Vitamin A.</td>
<td>Passive imm.</td>
<td>Treatment of complications</td>
<td>Steroids</td>
</tr>
<tr>
<td></td>
<td>Vaccine.</td>
<td>Anti-pyretic</td>
<td>Anti-pyretic.</td>
<td></td>
</tr>
</tbody>
</table>
**Viral infection**

**Measles (Rubeola) 2011-03 -01-97**

- **Infectious agent:** Measles virus (RNA Paramyxovirus) - One antigenic type.
- **MOT:** Droplet infection (direct & indirect)
- **Incubation period:** 1-2 weeks
- **Period of Infectivity:** 5 days before and 5 days after rash appearance

**Clinical picture:**

A. **Prodromal stage (3-5 days):** Most infectious stage
   - Fever (↑ 40°C), headache, fatigue, anorexia, Conjunctivitis, Coryza & Cough
   - Koplic’s spots (Sure sign): Bluish white spots on red raised base opposite 2nd molar on the buccal mucosa which disappear soon after rash appears).

B. **Eruptive stage (rash):** (4th day): 
   - Erythematous, maculo-papular, starts (Behind ears → face → neck → trunk → limbs).
   - They fade in order of appearance. - Fever settles after 4-5 days.

**Diagnosis:**

- **Clinical picture.**
- **Complete blood picture:**
  - Leukocytic count: Low with relative lymphocytosis. - High in 2ry bacterial inf.
  - Serology: IgM (1-2 days after onset of rash) IgG (rises after 10 days)

**Complication:** 2ry infection

1) **Respiratory tract:** Otitis media, bronchitis, 2ry pneumonia
2) **GIT:** Gastroenteritis.
3) **Eye:** Keratitis, conjunctivitis
4) **Brain:** Encephalitis
5) **Other:** Activation of a tuberculous focus. - Hemorrhagic measles - PEM

**DD**

- Rubella.
- Roseola infantum,
- IMN
- Toxoplasmosis
- Scarlet fever.
- Meningococcemia
- Kawasaki disease
- Drug rashes.

**TTT**

1) **Supportive therapy:**
   - Antipyretics (Paracetamol) - Bed rest and an adequate fluid intake are indicated.
   - A nourishing easily digested diet & proper cleanliness.
2) **Vitamin A:** A single dose of 50,000 to 200,000 ill.
3) **Broad spectrum antibiotic** in presence of infections.

**Prevention**

i. **General** - Personal hygiene. - Environmental sanitation - Socioeconomic development.

ii. **Specific**

1) **Measles, Mumps &Rubella (MMR vaccine).**
   A. **If exposure is within 48 hour:**
      Administer measles vaccine to prevent the disease and confer lasting immunity. The incubation period of the vaccine is 2 days shorter than that of natural infection.
   B. **If exposure is within 2-5 days:**
      i. **Prevention:** 0.25 ml/kg of immune serum globulin IM.
      ii. **Attenuation:** 0.05 ml/kg of immune serum globulin IM.

2) **Gamma globulin:**( close contacts that not immunized)
Mumps

- **Infectious agent:** Mumps virus (RNA). - One antigenic type.
- **MOT:** Droplet infection (direct & indirect)
- **Incubation period:** 2-3 weeks
- **Period of Infectivity:** 1 day before and 5 days after the swelling of salivary gland

**Clinical picture:**

A. **Prodromal period:** Malaise & anorexia followed by:

1) Rapid enlargement of the parotid glands.
   - Being somewhat painful and tender.
   - Obliterating the angle of the mandible.
   - The opening of Parotid duct is commonly pointed and red.
   - Swelling subsides in 3-7 days.
   - Unilateral at onset but becomes bilateral later.

2) Low grade fever (1 week.)

3) Testes & ovaries affection → No sterility.

- **Diagnosis:**
  ✓ Clinical picture
  ✓ Complete blood picture:
    - Leukocytic count:
      - Low with relative lymphocytosis.
  ✓ Serology: IgM: Recent infection
    - IgG: Establish the diagnosis

- **Complication:**
  ✓ Deafness (hearing loss)
  ✓ Orchitis, Oophoritis
  ✓ Pancreatitis
  ✓ Aseptic meningitis, Encephalitis

- **DD**
  - Acute suppurative parotitis.
  - Acute obstructive parotitis.
  - Cervical lymphadenitis.
  - Other viral parotitis.

- **TTT**
  Nonspecific therapy, only supportive management

- **Prevention**
  - Measles, Mumps & Rubella (MMR vaccine).
German measles (Rubella) 2010-02-01

- **Infectious agent:** Rubella virus. (RNA toga virus) - One antigenic type.
- **MOT:** Droplet infection (direct & indirect)
- **Incubation period:** 2-3 weeks
- **Period of Infectivity:** 7 days before and 7 days after rash appearance

**Clinical picture:**

A. **Prodromal period:** very short and mild
   - Enlarged LN (Lymphadenopathy) especially behind ears & on the back of the head.

B. **Eruptive stage (rash) (1-5 d):** 1st manifestation
   - Fine, pink, start from: forehead & face downwards mainly on the abdomen

**Diagnosis:**
- Clinical picture.
- ELISA to pregnant (4 fold over the normal)

**Complication:**
1. Encephalitis
2. Polyarthritis
3. Congenital Rubella $ 2003$

If rubella develops during the first trimester of pregnancy, (which is the period of organogenesis).

**C/P:**
1. Intrauterine growth retardation, SGA and failure to thrive
2. **CNS:** Microcephaly and mental retardation
3. **CVS:** Congenital heart disease (PDA, VSD)
4. **Eye** Cataract, glaucoma, and cloudy cornea
5. **Ear** Nerve deafness
6. Thrombocytopenic purpura, HSM, and osteopathy

**Prevention**
1. Terminate pregnancy if clinical rubella develops, or the antibody titre is rising (sub clinical infection).
2. Allow pregnancy to continue if the antibody titre is already high (immunity) or is not rising.
3. Routine immune globulin administration for post exposure prophylaxis in pregnancy is not recommended.

**TTT:**
1. Antipyretics for fever.
2. Treatment of complications

**Prevention**
1) **MMR Vaccine:** MMR vaccine should not be given to pregnant $♀$; vaccinated $♀$ should avoid pregnancy for 3 m. after vaccination.
2) **Passive immunization.** It is not indicated except in non-immune pregnant $♀$. Immune serum globulin in big doses is given I.M within one week of exposure.

---

Roseola infantum 2007-2000

- **Infectious agent:** Human herpes virus 6 and 7 (DNA) {{Sixth disease}}
- **MOT:** Droplet infection
- **Incubation period:** 1-2 weeks

**Clinical picture:**

A. **Prodromal period:**
   - Sudden $\uparrow\uparrow$ fever lasts 3-4 days ± convulsions without any localizing sign
   - Mild rhinorrhea, and mild redness of conjunctiva and pharynx.

B. **Eruptive stage (rash)**
   - **Maculo-papular rash** "Rainbow following the storm"
   - Appears on the 4th day with the drop of fever.
   - Starts on the trunk and rapidly spreads over the arms and neck, leaving the face minimally involved, and lasts only 24 hours.

**Diagnosis:** this is only clinical.

**TTT:** - No need for antiviral therapy - Antipyretics - Supportive treatment for febrile convulsions
Chicken Pox 2009-2004

- **Infectious agent:** Varicella-zoster virus (VZV)
- **MOT:** Droplet infection (direct & indirect)
- **Period of Infectivity:** 2 days before the onset of the rash till all lesions are crusted.
- **Clinical picture:** Lack of transplacental immunity, commonly 5-10 years
  
  A. **Prodromal stage:** very mild and short.
  B. **Eruption stage:** **Pleomorphic rashes**
     - The rash appears as crops of **macules** which within hours pass through a **papular** stage, then progress to develop into **vesicles** (fragile, fluid-filled, clear, and oval on an erythematous base).
     - Vesicles → **Pustules** → **Crusts** on the 9th to 14th day.
     - The rash is centripetal in distribution (heavy on the trunk, scarce on extremities).
     - Pruritus may be intense.
     - The rash may appear on mucous membranes with ulceration in the mouth.

- **Complication:**
  1. Secondary bacterial infection of skin lesions.
  2. Hemorrhagic complications: thrombocytopenia, purpura, hematuria, and gastrointestinal hemorrhage.
  3. Encephalitis and cerebellar ataxia.
  4. Reye syndrome: encephalopathy and hepatic dysfunction.

- **DD:**
  - **Herpes zoster:** Unilateral rash occurs along one or more dermatome of peripheral nerves.
  - **Impetigo:** It is pyogenic infection of the skin caused by staphylococci or streptococci.
  - **Papulo-vesicular urticaria:**
    - Allergic reaction, mostly to an insect bite.
    - The lesions are mainly distributed over the extensor surfaces of the extremities.
  - **Scabies:**
    - There is history of contact with a case.
    - Itching is more by night.
    - Thread-like burrows in interdigital spaces of fingers and toes.

- **TTT:**
  1. Local and systemic antihistaminic to alleviate itching
  2. Non-aspirin antipyretics (e.g. paracetamol).
  3. Local application of calamine lotion.
  4. Patients at risk of severe chickenpox should receive **Acyclovir:** 2 µg/Kg
  5. Treatment of complications.
**Bacterial infection**

**Scarlet fever** 2012-11-00-99-98-94

- **Infectious agent:** Pyogenic exotoxin producing group A β-hemolytic streptococci
- **MOT:** Droplet infection (direct & indirect)
- **Incubation period:** 1-7 day
- **Clinical picture:**

A. **Prodromal stage:**
- Sudden onset of fever, headache, sore throat & vomiting
- **Throat:**
  - Edema of the anterior pillars and a beefy red appearance.
  - **Tonsils** are hyperemic, edematous and may be covered with membrane in severe cases.
- **Tongue**
  - Early: Swollen coated with prominent papillae
  - Late: Shedding of the white coat leaving beefy tongue with swollen papillae

B. **Eruption stage:**
- Appears in the 2nd day of fever
- Diffuse maculo-papular producing bright red discoloration of the skin which blanch on pressure
- Starts in the axilla and groin the spared rapidly to trunk, face and extremities
- In the face the perioral area is spared (Circumoral pallor)
- **Pastia’s sign** Transverse dark lines in the creases of antecubital fossa (not fades with compression, not pathognomonic)

**Diagnosis**
1. Leukocytosis up to 15000 with an Antistreptolysin O titre above 350 Todd Units.
2. Throat culture is positive for β-hemolytic streptococci.
3. Rapid antigen detection tests for group A streptococci

**DD:** Other causes of maculo-papular rash

**TTT:**
1. Antipyretic analgesics
2. Antibiotics
   - **Penicillin**
     - Oral:
       - Penicillin V 400,000 U/day for 10 days.
     - Parenteral:
       - Procaine penicillin 400,000 U/day IM for 10 days.
       - Benzathine penicillin 600,000-1,200,000 U IM once.
   - In case of penicillin allergy,
     - Erythromycin: 40 mg/kg/day orally in 4 divided doses for 10 days OR
     - Azithromycin: 10 mg/kg/day orally as a single daily dose for 5 days.

**Prevention:**
1. Avoid overcrowding in schools
2. Close observation of contacts of an individual case of scarlet fever.
3. No vaccine is available as many strains of group A streptococci cause the disease.
**Infectious agent:** Bordetella pertussis

**MOT:** Droplet infection (direct & indirect)

**Incubation period:** 1-2 weeks

**Period of Infectivity:** 1 week before to 3 weeks after the onset of symptoms

**Clinical picture:** Lack of transplacental immunity

a. **Catarrhal stage** (1-2 weeks):
   - Low-grade fever, Congestion and rhinorrhea, sneezing & conjunctival coloring.

b. **Paroxysmal stage** (2-6 weeks):
   - **Episodes of cough**:
     - Repetitive series of severe cough during a single expiration followed by a sudden deep inspiratory whoop (forceful inspiratory gasp).
     - During the attack chin and chest are held forward, tongue protrudes maximally, eyes bulge, and face becomes purple or cyanosed.
     - Vomiting commonly follows the paroxysm and thick tenacious Mucoid sputum is expectorated.
     - Repeated attacks may interfere with sleep and feeding.
     - **Between attacks:** the child does not appear ill and chest signs are minimal.
     - **Complication:** PEM and alkalosis $\rightarrow$ Ionized calcium with manifestations of Tetany (Normocalcemic).
     - **In infant:** Absent in infants and are replaced by attacks of apnea and cyanosis.
     - The infant many die during an attack.

c. **Convalescent stage** (1-2 weeks):
   - Paroxysms of cough gradually $\downarrow$ in number and severity,
   - Vomiting becomes less frequent and appetite returns.

**Diagnosis:**
- Typical manifestation may be enough for diagnosis
- Blood picture: leukocytosis (>20,000/mm3) with predominating lymphocytosis.
- Culture of organism: Nasopharyngeal swab on blood agar media during the early phase of the disease.

**Complications**

A. **Respiratory:**
   - Atelectasis (due to mucus plug),
   - Activation of latent T.B focus
   - Bronchopneumonia. {{Commonest one}}
   - Bronchiectasis Otitis media and sinusitis.
   - Rupture of Emphysematous blebs $\rightarrow$ emphysema
   - Ruptured alveoli $\rightarrow$ Pneumothorax, pneumomediastinum

B. **Gastro-intestinal:**
   - Severe and prolonged vomiting may lead to malnutrition, dehydration, alkalosis and tetany.
   - Rectal prolapse& Hernias.

C. **Nervous:**
   - Convulsions due to cerebral anoxia during paroxysms (common in infants).
   - Tetany and intracranial hemorrhages

D. **Hematological:** Hemorrhage (epistaxis, subconjunctival, hemoptysis, intraventricular and subarachnoid).
• DD: Asthma – Bronchitis – Bronchiolitis – Cystic fibrosis
  
  F.B in larynx or trachea – Others: Postnasal discharge – Mediastinal $ 

• TTT:
  ○ Symptomatic treatment:
    - General supportive measures, bed rest, soft diet and liquids.
    - Anti-tussives for spasmodic cough
  ○ Specific treatment: Erythromycin 40mg/kg for 14 days

• Prevention:
  ✓ Isolation of patients and household contacts for 5 days after starting erythromycin.
  ✓ Active immunization (DTaP).
  ✓ All contacts:
    Erythromycin 40mg/kg /day divided 6 hourly for 10-14 days +
    Booster DTaP if age is less than 6 years.

> Acute Bacterial Meningitis 2002-95

• Infectious agent:
  ○ First 2 months of life: Group B Streptococcus, gram negative bacilli, Streptococcus pneumoniae, Neisseria meningitides, H. influenza type b. & Listeria
  ○ Children 2 mo-12yr of age
    1- Streptococcal pneumoniae
    2- Neisseria meningitides

• MOT: Droplet infection - Blood born (esp. in neonatal meningitis)

• Incubation period: < 7 days

• Clinical picture:
  A. Non-specific Symptoms: Fever, anorexia, poor feeding, myalgia, arthralgia, tachycardia, hypotension and various cutaneous signs such as petechiae, purpura or an erythematous macular rush

  B. Signs of meningeal irritation:
    1. Nuchal rigidity and back pain
    2. Kerning’s sign: Flexion of the hip 90 degrees with subsequent pain with extension of the leg.

  C. Symptoms and Signs of increased ICP:
    1. Headache and vomiting
    2. Bulging fontanel or widening of the sutures
    3. Cranial neuropathies.
    4. Hypertension with bradycardia
    5. Apnea or hyperventilation, stupor and coma.

  D. Seizures (focal or generalized)
    Cause: cerebritis, infarction or electrolyte disturbances.
    Time: occur on presentation or within the first 4 days of onset are usually of no prognostic significance.
• **Diagnosis:** Lumbar puncture for CSF analysis

1. Aspect Turbid.
2. Pressure: ↑
3. Cells: Marked ↑ especially Neutrophil (300-2000/mm³)
4. Protein: Marked ↑ (100-500 mg/dl)
5. Glucose: ↓↓ <<50% of serum glucose>>
6. Chloride: ↓
7. Organisms: detected by Gram stain and culture

• **TTT:**
  - **Supportive ttt:**
    ✓ Correction of dehydration and electrolyte disturbances and proper nutrition
  - **Symptomatic ttt:**
    ✓ Control of seizures. 4- Management of neurological complications
  - **Specific ttt:**
    ✓ **Antibiotic Therapy:**
      Once meningitis is suspected we should start Ht immediately even before culture:
      a. Vancomycin 60mg/kg/ 24hr given every 6 hr in combination with
         ♥ Cefotaxime (Claforan): 200 mg/Kg/24hr IV OR
         ♥ Ceftriaxone : 100 mg/kg/24hr single dose or given every 12 hour.
      b. Chloramphenicol: : 100 mg/Kg/day in penicillin-sensitive patients.

    ✓ **Corticosteroids:**
      o Dose: IV dexamethasone 0.15 mg/kg/dose given every 6hr for 2 days
      o Indication: Children older than 6wk with acute bacterial meningitis caused by H. infl.

• **Prevention:**
  a. Isolation of the patient.
  b. Chemoprophylaxis: Rifampicin for contacts.
     ✓ 10 mg/kg/12hr for 2 days (in N. meningitides)
     ✓ 20 mg/kg/24hr for 4 days (in H. influenzae type b).
  c. Immunoprophylaxis:
     ✓ Meningococcal Live attenuated vaccine for high-risk children older than 2yr.
     ✓ H. influenza type b vaccine for all children.
Pulmonary T.B. 2005-03-01-00:99-97-00 - 97-96

- Clinical pictures

<table>
<thead>
<tr>
<th>Primary pulmonary TB Birth - 7 years</th>
<th>Progressive primary pulmonary TB 7 - 18 years</th>
<th>Reactivation TB 7 - 18 years</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>IP</strong> 2 - 4 weeks</td>
<td><strong>Progressive primary pulmonary TB</strong> 3 - 9 months</td>
<td><strong>Chest:</strong> Hemoptysis, chest pain, <strong>Systemic:</strong> Fever, anorexia, malaise, wt loss, night sweats.</td>
</tr>
<tr>
<td><strong>Symptoms</strong> Chest: Dry cough, difficult breathing, chest pain. Systemic: Fever, anorexia, wt loss, night sweats.</td>
<td><strong>Chest:</strong> Severe cough with expectoration Systemic: High fever, wt loss, night sweats.</td>
<td><strong>Chest:</strong> Decreased breath sounds, rales, Dullness.</td>
</tr>
<tr>
<td><strong>Signs:</strong> Respiratory distress, tachypnea, wheeze</td>
<td><strong>Decreased breath sounds, rales, Dullness.</strong></td>
<td><strong>Dullness over infiltrate, hyper-resonance over the cavity.</strong></td>
</tr>
</tbody>
</table>

- Diagnosis:

  **Tuberculin test**

  - **Method:** 0.1 ml of purified protein derivative (PPD) → intra dermal in the front of left forearm.
  - **Result:** Induration after 48 – 72 hours - 0 – 5 mm → Negative test. - 5 – 9 mm → doubtful - repeated after 2 wks. - > 10 mm → positive test.
  - **Positive:** - Non-vaccinated child < 5 years. - Highly positive > 15 mm patient in contact to open T.B. case.
  - **False positive:** - Vaccinated. - Repetition of the test. - Atypical mycobacterial infection.
  - **False negative:** - Destroyed vaccine. - Decreased immunity. - CMI not developed. - Miliary T.B.

- Treatment

  1. **Main principles** 2C-2P

  - Continuous.
  - Combined.
  - Prolonged periods (6 - 9 months or more).
  - Proper dose.

  2. **Drugs**

  - **The standard drugs:** RESPIRATION
    - **Rifampicin.**
    - **Ethambutol.**
    - **Streptomycin.**
    - **Pyrazinamide.**
    - **INH (Isoniazid).**

  - **DRUG**
  - **DOSE (mg/kg/day)**
  - **5 10 15 25 30**
  - **SIDE EFFECT**

  - **INH** 5 Orally
    - **Hepatotoxicity.**
    - **Neuropathy.**
    - **Sarcoid.**
  - **Rifampicin** 10 orally
    - **Hepatotoxicity**
    - **GIT irritation.**
  - **Streptomycin** 15 IM 2 months.
    - **Ototoxicity.**
    - **Nephrotoxicity.**
  - **Ethambutol** 25 Orally
    - **Optic neuritis (2%)**
  - **Pyrazinamide** 30 Orally 2 months
    - **Hepatotoxicity. Hyperuricemia.**

  3. **Regimens.** (6 - 9 month continuous regimen)

  - **Initial phase:** 2 months of 3 - 4 drugs (Rifampicin + INH + one or two drugs from the standard drugs)
  - **Continuation phase:** 4 - 7 months of Rifampicin + INH.

  - **4.TB & cortisone**

  - **Initial phase:**
    - o TB meningitis.
    - o Genitourinary tuberculosis to prevent ureteric strictures.
    - o Pericarditis, peritonitis & pleural effusion to prevent fibrosis.
    - o Lymph node enlargement.

  - **Continuation phase:**
    - o TB meningitis.
    - o Genitourinary tuberculosis to prevent ureteric strictures.
    - o Pericarditis, peritonitis & pleural effusion to prevent fibrosis.
    - o Lymph node enlargement.

- Prevention

  1. **BCG vaccine**

    - 0.1 ml I.D. in left arm.
    - Live attenuated.
    - 60 – 80 % protection against T.B.
    - Prevents miliary T.B. & T.B. meningitis.
    - Normal reaction → wheal, swelling, abscess, ulcer after 1 ms.
    - Accelerated → red swelling in 2 – 3 days.
    - No reaction → repeated the vaccination after 3 ms.
    - Complicated by ulcer, adenitis, osteitis, fever, convulsions.

  2. Health education.
  3. Proper isolation.
  4. Good nutrition.
  5. Avoid overcrowding.
  6. Prevent perinatal T.B. by using INH, RIF, EMB
Parasitic infection

Protozoa

Treatment of Amoebiasis 2004

1. Metronidazole 50 mg/kg/day orally in 3 divided doses for 10 days.
2. Tinidazole 50 mg/kg orally single dose for 3-5 days
3. Test of cure ➔ stool examination after 2 weeks from end of treatment.

Treatment of Giardiasis

1. Metronidazole 15 mg/kg/day orally in 3 divided doses for 7 days.
2. Tinidazole 50 mg orally single dose.
3. Furazolidone 8 mg/kg/day orally in 4 divided doses for 10 days.

Helminthes

Treptomate

Treatment of Schistosomiasis 2011

1. TTT
   - Praziquantel: Drug of choice
     • 40 mg/kg/day ➔ divided bid for 1 day.
   - Niridazole
     • 15 mg/kg/day for 10 days.
2. Prevention
   - Sanitary measures.
   - Eradication of snails.
   - Treatment of individuals.

Nematode

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Pyrantel Pamoate</td>
<td>11 mg/kg once daily for 3 days ... maximum dose 1 gm.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mebendazole</td>
<td>100 mg bid for 3 days or 500 mg once</td>
<td>100 mg as single dose</td>
<td></td>
</tr>
<tr>
<td>Flubendazole</td>
<td>100 mg bid for 3 days or 500 mg once</td>
<td>100 mg as single dose</td>
<td></td>
</tr>
<tr>
<td>Albendazole</td>
<td>400 mg as a single dose</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Diagnosis of Ankylostoma 2009

- Examination of fresh stool reveals hookworm eggs and occult blood.
- Eosinophilia is marked.
- Hypochromic microcytic anemia.
## Immunization

### Egyptian Compulsory Vaccination Schedule 04-01-97-95-94-91

<table>
<thead>
<tr>
<th>Vaccine</th>
<th>Type</th>
<th>Dose</th>
<th>Mode</th>
<th>Timing</th>
<th>Booster</th>
</tr>
</thead>
<tbody>
<tr>
<td>BCG</td>
<td>Live attenuated V</td>
<td>0.1 ml</td>
<td>ID</td>
<td>0-2 mo</td>
<td>6-10Y</td>
</tr>
<tr>
<td>DTP</td>
<td>Inactive toxoid</td>
<td>0.5 ml</td>
<td>IM</td>
<td>2,4,6 mo</td>
<td>18m- 6Y</td>
</tr>
<tr>
<td>OPV</td>
<td>Live attenuated V</td>
<td>2 drops</td>
<td>PO</td>
<td>2,4,6,9,12 mo</td>
<td>18m- 6Y</td>
</tr>
<tr>
<td>Hib</td>
<td>Conjugated Polysaccharides</td>
<td>0.5ml</td>
<td>IM</td>
<td>2,4,6 mo</td>
<td>12 –15m</td>
</tr>
<tr>
<td>Hep B</td>
<td>Protein derivative</td>
<td>0.5ml</td>
<td>IM</td>
<td>2,4,6 mo</td>
<td>5Y</td>
</tr>
<tr>
<td>MMR</td>
<td>Live attenuated V</td>
<td>0.5ml</td>
<td>IM/SC</td>
<td>12,18mo</td>
<td>5-10Y</td>
</tr>
</tbody>
</table>

### Other vaccines not included in Egyptian schedule. 2003-02-01

<table>
<thead>
<tr>
<th>Vaccine</th>
<th>Type</th>
<th>Dose</th>
<th>Mode</th>
<th>Timing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hib</td>
<td>3D&amp;B</td>
<td>0.5ml</td>
<td>IM</td>
<td>2 - 4 - 6 mo</td>
</tr>
<tr>
<td>RV</td>
<td>2-3D</td>
<td>1 ml</td>
<td>PO</td>
<td>6mo 1- 11/2 mo interval</td>
</tr>
<tr>
<td>Var</td>
<td>2D</td>
<td>0.5ml</td>
<td>IM</td>
<td>12-15 mo - 4 - 6 years</td>
</tr>
<tr>
<td>Hep A</td>
<td>2D&amp;B</td>
<td>0.5ml</td>
<td>IM</td>
<td>&gt; 1Y -1-6 mo</td>
</tr>
<tr>
<td>Influenza</td>
<td>1D</td>
<td>0.5ml</td>
<td>IM</td>
<td>&gt;6mo in selected pt.</td>
</tr>
<tr>
<td>Pneumococcal</td>
<td>1</td>
<td>0.5ml</td>
<td>IM</td>
<td>&lt; 2 Y in selected pt.</td>
</tr>
<tr>
<td>Lyme disease</td>
<td>1</td>
<td>0.5ml</td>
<td>IM</td>
<td>&gt; 15 Y endemic areas</td>
</tr>
<tr>
<td>Rabies</td>
<td>3-5</td>
<td>0.5ml</td>
<td>IM</td>
<td>After exposure</td>
</tr>
</tbody>
</table>

D = dose - B = booster