Disclaimer: As we all know, we are still students and henceforth bound to make mistakes, however, we will try our very best to convey all knowledge based on the Malaysia protocols. By that, we do not hold any responsibilities should our presentations bear mishaps in the future.
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- Case presentation 2: Grace Ng Pek Kum
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- Urinary tract infections: Chung Soo Bee
- Neonatal resuscitation: Ivan Choo Tze Yong
PART 1: HISTORY TAKING

Purposes of history taking:
1. Used in the care of patients
2. As medico-legal documents
3. For clinical research

Clerking scheme

<table>
<thead>
<tr>
<th>Date of birth:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Date / Time of Clerking:</th>
<th>eg: 18/7/09 1500</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Age:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Source of referral:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Informer: mother / father / grandfather / care taker</th>
</tr>
</thead>
</table>

CHIEF COMPLAINTS & DURATION

Record child’s and parents words. Parents 1st priority
State the MAJOR Problem in one or two of the patient’s own words. Do not use medical terminology.
Eg:

2.5 months / Malay / Female / Wt : 5.6 kg
Fever x 3/7
Cough X 3/7
RN x 5/7

History of Present Illness

- When and How did it start?
- Was she / he previously well before?
- How did it develop?
- What aggravates it or alleviates / relieve it?
- Has there any contact with anyone infection?
- Has the child been to overseas recently?

Eg: PAIN (ask for pain: site, onset, trigger, previous episode, progression, duration, frequency, character, radiation, severity, elevating n relieving factors)

<table>
<thead>
<tr>
<th>Category</th>
<th>Scoring</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Face</td>
<td>No particular expression or smile</td>
</tr>
<tr>
<td>Legs</td>
<td>Normal position or relaxed</td>
</tr>
<tr>
<td>Activity</td>
<td>Lying quietly, normal position, moves easily</td>
</tr>
<tr>
<td>Cry</td>
<td>No cry (awake or asleep)</td>
</tr>
<tr>
<td>Consol ability</td>
<td>Content, relaxed</td>
</tr>
</tbody>
</table>
PAIN ASSESSMENT TOOL FOR UNDER 3 YEARS AND THOSE WHO CANNOT SELF REPORT

FLACC Scale: Each of the five categories (F)ace, (L)egs, (A)ctivity, (C) cry and (C) consol ability is scored from 0-2, resulting in total range of 0-10

- Always has Differential Diagnosis in your mind!!
- Eg: LOC
  - List out few main causes:

Include RELEVANT POSITIVE and RELEVANT NEGATIVE questions to help exclude differential diagnosis!

Past Medical / Surgical History
- Any previous hospitalization? Y/N
- If Yes, state HOsP, Probs, Duration of admission
- Follow-up for any medical problems and treatment

Eg: AEBA previously at ward 6B x 5/7 treated well.

Family History
- Consanguineous marriage Y/N?
- Draw family Tree

Find out any
- Stillbirth, tb, dm, renal ds, seizure, jaundice, malformations, others.
- Are siblings and parents alive and well?
- Find out about late-onset ds with a genetic component

  Eg: A parent has had a MI before 40 years old (with familial hyperlipidemia), do serum lipids on these children will turn out to have same, the sooner it treated, the better.

- Family History of Asthma / Eczema / allergy
- Febrile seizure / Epilepsy
- Other Illness

SOCIAL AND ENVIRONMENT HISTORY
- Living condition, Hygiene, who is caregiver? How is the environment?
- Level of education attained by both parents
- Any pets involved?
- Income of both parents?
- Document social services involvement

DIET HISTORY
- Exclusive breast feeding / mix, adult food, soft food etc
- Duration of breast feeding
- Weaning
- -volume eg. 1 scoop 2 Oz
- Freq / 24H
- Any previous Formula Changed? Why?
BIRTH HISTORY: eg: SVD, cry after delivery

IMMUNISATION HISTORY: eg: Completed up to age

DEVELOPMENT HISTORY

• Younger age group:
  ➢ Gross motor, fine motor, hearing /vision, social behaviour

• Older children (7 years and older):
  ➢ Compare with other siblings or peer group
  ➢ Schooling
  ➢ Following by previous milestones (emphasising on important milestones)

• Schooling:
  ➢ Which school and what sort
  ➢ Whether he is in a special school or has any remedial help
  ➢ Assess academic performance
  ➢ Participation in extra-curricular activities
  ➢ Has he missed school? Press the parent for an estimate of this
    • Eg: 3 months in the last year
  ➢ How does he get on with the other children

PRESENTING SUMMARY

Example:

A 5 years old chinese girl, previously well, no known any medical cases, 1st hospitalization, presented with complaints of vomiting for 2 days, diarrhea for 2 days, itchy nonpainful rash for 1 day started from LE to UL. Patient has no history of AGE contact, no fever, no URTI contact, not from dengue endemic area. At this stage my provisional diagnosis is HSP with DD of ITP and AGE.
### PAEDIATRIC CLERKING SHEET

<table>
<thead>
<tr>
<th>Parents</th>
<th>Age (years)</th>
<th>Education</th>
<th>Occupation</th>
<th>Income (permonth)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Father / guardian</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mother</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

#### Antenatal
- [ ] Home
- [ ] Hospital
- [ ] Private center

#### Birth
- Antenatal
- [ ] Home
- [ ] Hospital
- [ ] Private center

#### Neonatal Period
- Birth Wt: _____ kg
- Gestation (wks): ___

#### Neonatal problems:
- [ ] Nil
- [ ] Yes
- [ ] Asphyxia
- [ ] R.D.S
- [ ] Jaundice
- [ ] Feeding problems
- [ ] Others

#### Complications:
- Type of Delivery:
  - [ ] SVD
  - [ ] Vacuum
  - [ ] Forceps
  - [ ] LSCS
- [ ] Term
- [ ] Preterm

#### Care:
- [ ] Yes
- [ ] No

#### Immunisation

<table>
<thead>
<tr>
<th>Age (months)</th>
<th>Previous infectious disease</th>
<th>Date</th>
<th>Feeding History</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Whooping cough</td>
<td></td>
<td>Breast feeding</td>
</tr>
<tr>
<td>0</td>
<td>Measles</td>
<td></td>
<td>Duration:</td>
</tr>
<tr>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- BCG
- HepB
- DPT
- Polio
- Hib
- MMR

#### Others:
- [ ] Weaning (mouth)
- [ ] Any feeding problem:

#### Developmental milestone

<table>
<thead>
<tr>
<th>Age</th>
<th>Development milestone</th>
<th>Age</th>
<th>Allergy:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Smile spontaneously (6 weeks)</td>
<td></td>
<td>Food</td>
</tr>
<tr>
<td></td>
<td>Head help up (16 weeks)</td>
<td></td>
<td>Drugs</td>
</tr>
<tr>
<td></td>
<td>Roll over (6 months)</td>
<td>Bladder control</td>
<td>Hives</td>
</tr>
<tr>
<td></td>
<td>Sit unsided (7-8 months)</td>
<td>Dry by day(2 yrs)</td>
<td>Asthma</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dry by night(3 yrs)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Complete control</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Crawl (8-9 months)</td>
<td>Nursery/schooling performance</td>
<td>Eczema</td>
</tr>
<tr>
<td></td>
<td>Pull to stand (1 year)</td>
<td>Any other concern</td>
<td>Other</td>
</tr>
<tr>
<td></td>
<td>Stand unaided (15 months)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
General Physical Examinations

GENERAL CONDITIONS

- Well? ill?
- Ankle edema?
- Dehydration ? %
- Perusions (CRT) < 2sec ?
- Respiratory Distress?

Example: Alert, Comfort, Pink, Good Hydration, CRT <2sec

CARDIOVASCULAR SYSTEM

- Pulse Normal / Abnormal / Character:
- Surgical scars?
- Apex beat non-displaced? Displaced?
- Thrills / parasternal heave
- Heart sound S1 S2 normal / abnormal
- Added heart sounds S3 ? S 4?
- Heart:
- Femoral Pulses well felt / Delay

Eg: Pulse Volume Kept, Regular Rhythm

Heart: DRNM

RESPIRATORY SYSTEM

- Throat injected? Tonsil Enlarged? Exudate? Follicle?
- Ears – Right _____ Left _________
- Chest wall deformity?
  - Pectus carinatum / Excavatum / harrison sulci / Hyperinflated
  - Recession – Suprasternal / Intercostal / subcostal
- Nasal flaring?
- Stridor? Wheeze?
- Lungs – Breath sounds – vesicular ? Bronchial breathing? Prolonged expiratory phase?
- Air entry – equal? Reduce at ?
- Added sounds – Rhonchi ? Crepts?

GASTROINTESTINAL SYSTEM

- Jaundice? Oral cavity _____ Palate _________
- Abdominal Distension?
  - Fat, Flatus, Fecal, Fluid,
- Surgical Scar?

• VITAL SIGNS
  - BP ____ mmHG
  - HR ____ / min
  - RR ____ / min
  - Temp ________ C
  - SpO2 ________%

• Weight ___kg
• Height / Length ____cm
• COH ____cm  BMI ______  BSA ______
• Visible peristalsis?
• Soft? Guarded?
• Non-tender? Tender?
• Liver? Spleen? Kidney? Mass?
• Ascites?
• Hernia? Ambiguous?
• Per-rectal examination (if indicated) _______

NEUROLOGICAL SYSTEM
• GCS EVM?
• ORIENTATION TO TIME / space / person?
• Neck Stiffness? Pupils R ___ mm Reactive ?
• L ___ mm Reactive ?
• Fundoscopy – R ?? L ??
• Cranial nerves intact? Deficit?
• Contractures? Pes cavus?
• Sensory (if indicated)
  ➢ Light touch ___ Pain ____ Vibration ______
• Spinal Level ______
• Cerebellar signs? Gait ______ Rombergn sign +ve / -ve
• Spine – dimples? Tuff of hairs? Defect?

Distended bladder? Lax anal tone?
HOSPITAL SULTANAH BAHYAH
PAEDIATRIC CLERKING SHEET

Date of birth .................................................................
Date/Time of clerkng ........................................................
Age ..............................................................................
Source of referral ...........................................................
Chief Complaint ................................................................

History of Present illness ...................................................
......................................................................................
......................................................................................
......................................................................................
......................................................................................
......................................................................................
......................................................................................
......................................................................................
......................................................................................
......................................................................................
......................................................................................
......................................................................................

Past History .....................................................................

Previous Treatment history: Self/Traditional/GP/Health Centre/Hospital (Duration prior to admission)
Family History

Social History

Feeding History

Others (Birth/Developmental/Immunisation/etc.)

Physical Exam

<table>
<thead>
<tr>
<th>Centile</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight</td>
</tr>
<tr>
<td>Height</td>
</tr>
<tr>
<td>Head Circumference</td>
</tr>
<tr>
<td>General Condition</td>
</tr>
<tr>
<td>Temperature</td>
</tr>
<tr>
<td>Pallor/Cyanosis/Jaundice/etc</td>
</tr>
<tr>
<td>Head/Neck</td>
</tr>
<tr>
<td>Eyes</td>
</tr>
<tr>
<td>Ears</td>
</tr>
<tr>
<td>Throat</td>
</tr>
<tr>
<td>CVS:—BP.</td>
</tr>
<tr>
<td>Respiratory R/R.</td>
</tr>
<tr>
<td>Alae nasi</td>
</tr>
</tbody>
</table>
Abdomen

Genitalia

Lymph Nodes

CNS — level of consciousness

Cranial Nerves

Motor System

Others

CNS

Musculoskeletal System

Developmental Assessment

Summary

Diagnosis: (1)

(2)

Name of Doctor

Signature
# CASE DISCUSSION 1

## CASE 1 (a)

A 10 year old boy with the recent history of multiple episodes of respiratory difficulty presents to you with tachypnoea, perioral cyanosis, likely pulsus paradoxus, use of accessory muscle of breathing, slight wheezing, delayed capillary refill, and drowsiness.

## CASE 1 (b)

2 years old Indian female
- Previously healthy, Presented in the department at 2p.m.

**C/C:**
- Difficulty in breathing, Noisy breathing
- 1st episode started last night
- Fever x 2/7 38.5°C
- °Cough, Rash x 1/7, °conjunctivitis

**HOPI**
- Morning went to KK – Neb 1 x
- Condition improved
- Then condition worsen
- And admitted to hospital

Family History
No Family History Asthma

## Probable diagnosis:??

### Differential Diagnosis:
- Asthma
- Brochiolitis
- Congenital/ Anatomical Anomalies s.a.
  - Bronchopulmonary dysplasia
- Tuberculosis
- Cystic Fibrosis
- Croup
- Tracheal Stenosis
- FB Aspiration/ GERD
- Laryngotraceomalacia

### Viral Bronchiolitis
- Common respiratory disease in child <2 y.o
- Caused by RVS virus
- Peak incidence: 3-6 months of age

**Clinics:**
- Mild coryza
- Low grade temperature
- Cough and wheeze
- Tachypnoea, chest wall recession
- Hyperinflated chest
- Auscultation: Fine crepitation, rhonchi
- Neonates: maybe apnoea

### Acute asthma

Recognize early sign of worsening of asthma control:
- Increase in symptoms
- Use of bronchodilator more often, less relieved
- PFM: readings getting lower/ difference btw best and worst getting greater

## Viral bronchiolitis

<table>
<thead>
<tr>
<th>Hx</th>
<th>Asthma</th>
</tr>
</thead>
<tbody>
<tr>
<td>URTI contact</td>
<td>Strong family history</td>
</tr>
<tr>
<td>Clinics</td>
<td>Allergy</td>
</tr>
<tr>
<td>Intoxication syndrome well expressed</td>
<td>Intoxication syndrome mild or absent</td>
</tr>
<tr>
<td>Low grade temperature</td>
<td>absent</td>
</tr>
<tr>
<td>Spastic cough all day</td>
<td>Noctural cough predominance</td>
</tr>
</tbody>
</table>

## Asthma

<table>
<thead>
<tr>
<th>CXR</th>
<th>Viral bronchiolitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Perihilar haziness</td>
<td>Sign of emphysema</td>
</tr>
<tr>
<td>Air bronchogram</td>
<td>Lab</td>
</tr>
<tr>
<td>Lymphocytosis</td>
<td>Eosinophilia</td>
</tr>
<tr>
<td>Increase ESR</td>
<td>Instrumental</td>
</tr>
<tr>
<td>HAPPY WHEEZER</td>
<td>PEV increase &gt;15% after bronchodilators</td>
</tr>
</tbody>
</table>
Question 1

What is the most likely diagnosis?

- Case 1 (A)
- Case 1 (B)

Question 2

What is the next step in evaluation?

Management of acute asthma:

- Oxygen via mask or nasal prong if SaO2 <95%
- SABA - salbutamol, terbutaline via Nebulizer
  - In Msia Hosp, we use AVN (Atroven(IB), Ventolin, NS) of ratio 1:1:1, 1:2:1 depends on hospital
- Oral steroids- prednisolone 1-2mg/kg, max 40-60mg/d
- Ipratropium bromide only given in severe cases
- CXR not indicated usually

Management of Viral Bronchiolitis

Table 1. Guideline for hospital admission in viral Bronchiolitis

<table>
<thead>
<tr>
<th></th>
<th>Home management</th>
<th>Hospital management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &lt; than 3 months</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Toxic – looking</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Chest recession</td>
<td>Mild</td>
<td>Moderate/severe</td>
</tr>
<tr>
<td>Central cyanosis</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Wheeze</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Crepitation on auscultations</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Feeding</td>
<td>Well</td>
<td>Difficult</td>
</tr>
<tr>
<td>Apnoea</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Oxygen saturation</td>
<td>&gt;95%</td>
<td>&lt;93%</td>
</tr>
<tr>
<td>High risk group</td>
<td>No</td>
<td>Yes</td>
</tr>
</tbody>
</table>

Principles of management

- Physiological monitoring
- Hydration
- Minimal handling
- Early recognition of complication especially respiratory distress
- Child with severe respi. distress, cyanosis, apnoea should be kept NBM, IV fluids given 100ml/kg/day

Specific Treatment

1. Oxygen therapy – SaO2 <93%
2. Nebulised Bronchodilators
   - Eg: Bricanyl 0.02 ml/kg 4 hourly
3. Corticosteroids therapy
4. Montelukast- Singular
5. Antibiotics are recommended for all infants with:
   - Recurrent apnoea and circulatory impairment
   - Possibility of septicemia
   - Acute clinical deterioration
   - High white cell count
   - Progressive infiltrative changes on CXR

Tips:

- no need cough medicine to suppress cough
- *give hydration to dilute mucous
- *give steroid to reduce oedema of bronchial mucous
- *give salbutamol if cough, wheezing at night, morning or when wake up
- *give neb for acute n severe attack!!
PART 2: NEONATAL JAUNDICE

- Neonatal Jaundice is the physical manifestation of hyperbilirubinemia when serum bilirubin level exceeds 85 µmol/L (>5mg/dL)
- Bilirubin Conversion Unit: 1mg/dL~17.1 µmol/L

Risk factor for bilirubin toxicity
- Preterm infant
- Small for gestational age (SGA)
- Sepsis*
- Acidosis
- Asphyxia
- Hypoalbuminemia
- Jaundice onset <24 hrs

Physiological Jaundice

<table>
<thead>
<tr>
<th>Group</th>
<th>Term Asian Neonates</th>
<th>Preterm Neonates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset</td>
<td>After 24 hr (3-5th day)</td>
<td>After 24 hr (2-3rd day)</td>
</tr>
<tr>
<td>Peak</td>
<td>170-238µmol/L (72-120hr of age)</td>
<td>170-204µmol/L (by 120 hr of age)</td>
</tr>
<tr>
<td>Resolution</td>
<td>7-10 days</td>
<td>10-14 days</td>
</tr>
<tr>
<td>Mech</td>
<td>Deficiency of UDPGT with slow maturation and increase intestinal bilin absorption</td>
<td>Deficiency of UDPGT and delayed maturation</td>
</tr>
</tbody>
</table>

Non-physiologic Jaundice

- Clinics of onset within 24hrs of life
- Increase total serum bilin >85 µmol/L/d
- Full term infant: Total serum [bili]
  - bottle fed >221µmol/L
  - breast fed >257µmol/L
- Preterm infants: Total serum [bili]>150µmol/L
- Conjugated bilin >34 µmol/L
- Clinics of jaundice lasting >1week (term), >2wks (preterm)
- Sign of underlying illness
- Unexplained hemorrhages
- G6PD deficiency
- Sepsis, eg: UTI, septicemia, meningitis
- GIT obstruction: increase enterohepatic circulations

Non-organic: Breast fed, breast milk JAUNDICE
**Hx taking**
- Onset of jaundice
- Age of gestation
- Pervious pregnancy: NNJ, G6PD deficiency, kernicterus, neonatal death, anemia (Rh-/ABO incompatibility)
- Maternal: blood grp, DM, medications
- Antenatal: TORCH infections
- Labor & delivery: traumatic hemorrhage
- Clinics of infant: lethargy, vomit, bad feeding, delay stool, temperature instability

**Physical Examination**
- General condition of infant
- Hydration status
- Weight of child
- Extravascular blood: bruise, hematoma
- Plethora, pallor
- Abdominal distention
- Sign of IU infections: Hepatosplenomegaly, petechia
- Signs of hypothyroidism
- Neurological signs of bilirubin encephalopathy:
  1. Abnormalities of tonus of muscles
  2. Lethargy, difficult arousing the child
  3. High pitch crying
  4. Opisthotonus
  5. Fever

**Kramer’s rule**

*Table 2. Clinical assessment of neonatal jaundice (Kramer’s rule)*

<table>
<thead>
<tr>
<th>Zone</th>
<th>Jaundice (detected by blanching the skin with finger pressure)</th>
<th>Estimated serum bilirubin (µmol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Head and neck</td>
<td>100</td>
</tr>
<tr>
<td>2</td>
<td>Over upper trunk above umbilicus</td>
<td>150</td>
</tr>
<tr>
<td>3</td>
<td>Lower trunk and thighs</td>
<td>200</td>
</tr>
<tr>
<td>4</td>
<td>Over arms, legs and below knee</td>
<td>250</td>
</tr>
<tr>
<td>5</td>
<td>Hands, feet</td>
<td>&gt;250</td>
</tr>
</tbody>
</table>

Note: This maybe difficult in dark skinned infants

**Indications to Hospitalization**
- Jaundice within 24 hrs of life
- Rapid rise serum bilirubin >8.5 µmol/L/hr
- Jaundice below umbilicus/ extending to soles of foot
- Family hx of significant hemolytic ds and kernicterus
- An unwell neonates with jaundice
- Prolonged jaundice >14 days
### Differential Diagnostic of NNJ

**Neonatal Jaundice**

**Unconjugated bilirubin**
- Pathologic
- Physiologic

**Conjugated bilirubin**
- Hepatic
- Post-hepatic

**Intrinsic causes of hemolysis**
- Membranopathy: Spherocytosis, Hereditary ellipsoidal
- Systemic disease: Sepsis, Arteriovenous malformation
- Enzymopathy: G6PD deficiency
- Hemoglobinopathy: sickle cell disease, Alpha-thalassemia

**Extrinsic causes of hemolysis**
- Hemolytic disease of the newborn (ABO)
- Rh incompatibility
- Wrong transfusion of blood to NB

**Non-hemolytic causes**
- Congenital: Hypothyroidism, Gilbert’s syndrome, Crigler-Najjar syndrome
- Acquired: Cephalohematoma, Polycythemia, Sepsis

**Hepatic causes**
- Infections: Sepsis, Hepatitis B, TORCH infections
- Metabolic: Galactosemia, Alpha-1-antitrypsin deficiency, Cystic fibrosis
- Drugs
- Idiopathic

**Post-hepatic**
- Biliary atresia
- Bile duct obstruction

**Non-hemolytic causes**
- Congenital: Hypothyroidism, Gilbert’s syndrome, Crigler-Najjar syndrome
- Acquired: Cephalohematoma, Polycythemia, Sepsis

**Investigation:**
1. FBC
2. G6PD status
3. Thyroid F(x) Test

**For conjugated:**
- FBP
- Blood Culture
- UFEME
- Infant, maternal GXM
- Direct/Indirect Coomb’s test

**For unconjugated:**
- Immunological test
- USE
- HIDA Scan
Phototherapy

- Decrease the need for ET
- Prophylactic in VLBW infants may prevent hyperbilirubinemia
- CI in conjugated hyperbilirubinemia (bronze baby syndr) and congenital porphyria
- Complications: insensible water loss, loose stool, rash, tanning, ROPM

Types of phototherapy:
- Conventional phototherapy
- Fibreoptic type (Biliblanket)
- Intensive phototherapy

How to do it???

- Minimum irradiance of 12µmol/cm²/nm, measure the intensity periodically
- Intensive phototherapy is irradiance >30 µmol/cm²/nm
- Distance of light source 35-50cm
- Expose infant adequately
- Cover infant’s eyes with eye patch
- Turn infant every 2 hrs
- Monitor serum bilirubin levels
- Monitor infant’s temp 4 hrly to avoid chilling or overheating
- Ensure adequate hydration
- Allow parental-infant interaction
- Discontinue phototherapy when bilirubin is 30 µmol/L below phototherapy level

Recommended action level for management of NNJ in healthy term newborn

<table>
<thead>
<tr>
<th>Age of Life (Hours)</th>
<th>Total Serum bilirubin levels (µmol/L)</th>
<th>Phototherapy</th>
<th>ET if Phototherapy fails</th>
<th>ET + Intensive phototherapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;24</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>24</td>
<td>200</td>
<td>340</td>
<td>400</td>
<td></td>
</tr>
<tr>
<td>48</td>
<td>250</td>
<td>400</td>
<td>425</td>
<td></td>
</tr>
<tr>
<td>72</td>
<td>250</td>
<td>420</td>
<td>450</td>
<td></td>
</tr>
<tr>
<td>&gt;72</td>
<td>&gt;250</td>
<td>450</td>
<td>475</td>
<td></td>
</tr>
</tbody>
</table>

Recommended action level for management of NNJ in healthy preterm newborn

<table>
<thead>
<tr>
<th>Age of life (hours)</th>
<th>Wt &lt;1500g</th>
<th>Wt 1500-2000g</th>
<th>Wt &gt;2000g</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PhotoT</td>
<td>ET</td>
<td>PhotoT</td>
</tr>
<tr>
<td>&lt;24</td>
<td>&gt;70</td>
<td>&gt;170-225</td>
<td>&gt;70</td>
</tr>
<tr>
<td>24-48</td>
<td>&gt;85</td>
<td>&gt;170-255</td>
<td>&gt;120</td>
</tr>
<tr>
<td>49-72</td>
<td>&gt;120</td>
<td>&gt;170-255</td>
<td>&gt;155</td>
</tr>
<tr>
<td>&gt;72</td>
<td>&gt;140</td>
<td>&gt;255</td>
<td>&gt;170</td>
</tr>
</tbody>
</table>
Non-organic Jaundice

<table>
<thead>
<tr>
<th>Breast feeding Jaundice</th>
<th>Breast milk Jaundice</th>
</tr>
</thead>
<tbody>
<tr>
<td>■ Less breast milk in first few days of life</td>
<td></td>
</tr>
<tr>
<td>■ Exhaustion after delivery</td>
<td></td>
</tr>
<tr>
<td>■ Dehydration</td>
<td></td>
</tr>
<tr>
<td>■ Present factor that cause increase production of bilirubin</td>
<td></td>
</tr>
</tbody>
</table>

Algorithm in managing NNJ

- Newborn with Jaundice
  - History taking
  - Physical Examination
  - Assess Severity of Jaundice

  Pathological Jaundice
  - Hospitalized for Ix & evaluation
  - Appropriate Management

  Physiological Jaundice
  - Phototherapy according to level of bilirubin table

For BF and BM Jaundice
- Observe, cont BF
- Supplementary formula milk
- Ensure good hydration of baby
- Phototherapy if needed

Mistakes of doctors.....
- Do not doubt in bilirubin level from lab and delay of treatment
- Don’t delay treatment and interrupting phototherapy for diagnosis test
- Examine for sign of Bilirubin Encephalopathy
- Use Total Serum Bilirubin level for estimation of treatment
- Prevent bilirubin level from reaching dangerous level
- Measure bilirubin and compare with hour specific norms
CASE DISCUSSION 2

A 4-month-old girl presented with fever & rash
CC : High fever x 4/7
HOP: 4/7 prior to admission (PTA), she developed high fever and rhinorrhea. She was taken to see a doctor at a clinic and was diagnosed as "URTI". She was prescribed paracetamol, actifed and amoxicillin.
2/7 PTA, she had semi-solid stool.
1/7 PTA, she developed maculopapular rash at trunk at extremities.
PMH: she had been healthy. Birth weight was 2.8 kgs.

O/E
- Conscious but irritable, body weigh 5.2kgs.
- Vital signs: Temp 39.8°C, HR 160 beats/min, RR 40 breaths/min, BP 98/50 mmHg
- HEENT: mild injected conjunctiva, no icteric sclera, red lips, normal anterior fontanel.
- Lymph node: not palpable
- Heart: DRNM, tachycardia
- Lung: clear, no adventitious sound
- Abdomen: soft, no organomegaly
- Skin: Erythematous rash at trunk & extremities, Indurated and redness of old BCG scar at left shoulder
- Extremities: Swelling of dorsal part of hands and feet

Provisional disease:

Ix
- FBC: Hb 10.6 g/l, Hct 30.7%, WBC 14 PLT 328
- PBS: normochromic normocytic RBC, toxic granulation 1+, vacuolization 1+
- U/A: yellow ,clear, spgr 1.024, PH=5, WBC 20-30/HPF, albumin 1+
- ESR: 97mm/h
- ECG: sinus tachycardia, HR 170/min, no ST-T Δ
- Echo: No structural heart defect, normal left ventricular function (EF 67%), minimal pericardial effusion 5mm. At apex without any fibrin. Normal size coronary arteries (proximal left coronary artery= 1.8 mm, right coronary artery 1.7mm)

Course in the hospital
- On the first admission day, while waiting for U/C, she was started on IV gentamicin for a working diagnosis of "UTI".
- Repeated single catheter for urine exam for gram stain was done.
- The 2nd U/A revealed many WBC in urine and urine gram stain was negative.

Final Diagnosis:

TRO:
Management:

**Follow up course (2 weeks later):**
- She had peeling of skin at finger.
- Ix showed her PLT was 955 and her ESR down to 27mm/h.
- Her repeated echo showed mild dilatation of proximal left coronary artery (3.5mm) without any thrombus.
- She was put on continued low dose aspirin and her next follow up was 6 week later.

**Differential Diagnosis**
- Infantile Polyarteritis Nodosa (commonly involves the kidneys, joints, muscles, peripheral nerve)
- Staphylococcus Aureus Infection (purulent, + bacteria)
- Juvenile Rheumatoid Arthritis (joint, organomegaly, myalgia, serositis)
- Streptococcal Infection, Group A - Rheumatic Fever, Rheumatic Heart Disease (+ thorat swab)
- Leptospirosis (bac test +)
- Toxic Shock Syndrome (GI, Kidney, muscle, CNS involvement)
- Measles (stepwise rash appearance, Koplik’s spot)
- Toxicity, Mercury (occupations, hobbies, seafood intake)
- Rocky Mountain Spotted Fever (epidemio risk, sero+)

**DDx Kawasaki Disease and Steven Johnson Syndrome**

<table>
<thead>
<tr>
<th>Kawasaki Diseases</th>
<th>Steven-Johnson Syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>A vasculitis disease which affect medium arteries</td>
<td>Affecting the skin in which cell death causes the epidermis to separate from the dermis</td>
</tr>
<tr>
<td>There is no ulcer or other lesions presented in Kawasaki disease</td>
<td>Ulcers and other lesions appear in the mucous membranes in the mouth and lips but also in the genital and anal regions.</td>
</tr>
<tr>
<td>Patient mouth is not painful and just present redness of the oropharynx</td>
<td>Patient usually experienced extremely painful in the mouth.</td>
</tr>
<tr>
<td>Rash is polymorphous but non vesicular.</td>
<td>Rash can begin as macules that develop into papules, vesicles, bullae, urticarial plaques, or confluent erythema.</td>
</tr>
</tbody>
</table>

**Note:**
1. Kawasaki should be included in differential diagnosis of all cases who presented with fever with rash.
2. Other main criteria (red lip, oedema of extremities, injected conjunctiva) are helpful for diagnosed Kawasaki in this case. Cervical lymphadenopathy was less helpful (incidence only 50%).
3. Other associated finding such as extremely irritable, indurated and erythema of BCG scar, and sterile pyuria from urine exam should alert physician to suspected Kawasaki disease.
4. Every patient diagnosed Kawasaki disease should be undergo echocardiogram evaluation in 2-3 weeks after onset of disease due to high incidence of coronary artery abnormalities (25 % without IVIG treatment, 5 % in IVIG treatment) which may leading to sudden death.
PART 3: FLUID AND ELECTROLYTES BALANCE

Volume replacements
1) Whole blood and plasma
2) Plasma expanders
3) Colloids
4) Crystalloids

Crystalloids

<table>
<thead>
<tr>
<th>Hypotonic solutions</th>
<th>Isotonic crystalloids</th>
<th>Hypertonic saline solutions</th>
</tr>
</thead>
<tbody>
<tr>
<td>- D5W, 0.45% NaCl</td>
<td>- Lactated Ringer’s, 0.9% NaCl</td>
<td>- 3% NaCl</td>
</tr>
<tr>
<td>- less than 10% remain intravascularly, inadequate for fluid resuscitation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Isotonic crystalloids</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- only 25% remain intravascularly</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Colloids
- Contain high molecular weight
- substances do not readily migrate across capillary walls
- Preparations
  - Albumin: 5%, 25%
  - Dextran
  - Gelifundol
  - Haes-steril 10%

Indication of colloids infusion
- Hypovolemic shock.
- Burns.
- Severe trauma.
- Endotoxin shock.

Contraindications of colloids infusion
- Severe anaemia.
- Cardiac failure.
- Pulmonary oedema.
- Renal insufficiency.

FLUID SELECTION
Maintain: N/2 + D (adult) + K+ 20 mEq
N/4 + D (children) + K+ 20 mEq

Repair: NaHCO₃ 8,4%
KCl 25 mEq/25 ml
NaCl 3%

Calculate fluid required
Volume needed = maintenance + deficit + ongoing loss

Calculating Maintenance Fluids

<table>
<thead>
<tr>
<th>Neonates and infants</th>
<th>Older children (&gt;12months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 day age</td>
<td>The Holliday-Segar Formula</td>
</tr>
<tr>
<td>2 day age</td>
<td>(Burn these numbers into your mind!!)</td>
</tr>
<tr>
<td>3 day age</td>
<td>100-50-20</td>
</tr>
<tr>
<td>&gt;3 days to 6 months</td>
<td>4-2-1</td>
</tr>
<tr>
<td>6 months to 1 year</td>
<td></td>
</tr>
</tbody>
</table>
Calculating Maintenance Fluids

The Holliday-Segar Formula

Case #1

An 32 kg girl is admitted for elective surgery and is NPO. She has normal renal function, no diarrhea and no fever. What would her maintenance fluids be?

Calculating Maintenance Fluids

The Holliday-Segar Formula

First 10 kg  →  100 ml/kg/day x 10  =  1000 ml
Second 10 kg →  50 ml/kg/day x 10  =  500 ml
Last 12 kg  →  20 ml/kg/day x 12  =  240 ml

Total 32 kg  1740 ml/day or 72.5 ml/hr

The Holliday-Segar Formula

First 10 kg  →  4 ml/hr x 10  =  40 ml
Second 10 kg →  2 ml/hr x 10  =  20 ml
Last 12 kg  →  1 ml/hr x 12  =  12 ml

Total 32 kg  72 ml/hr

Maintenance Electrolytes

Electrolyte loss can all be considered urinary

<table>
<thead>
<tr>
<th>Electrolyte</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium</td>
<td>3 mEq/100 ml</td>
</tr>
<tr>
<td>Potassium</td>
<td>2 mEq/100 ml</td>
</tr>
<tr>
<td>Chloride</td>
<td>2 mEq/100 ml</td>
</tr>
</tbody>
</table>

We all need some Sugar...

Glucose is added to:
- Prevent ketosis
- Limit protein catabolism
- 20% of caloric need made up of glucose is sufficient to prevent severe catabolism

5 grams glucose for every 100 cal

D5W (5% dextrose water) is an appropriate base for electrolyte solutions

Putting it Together

Maintenance IVF will need:
Water
Glucose
Sodium
Potassium
Chloride
Your choices:
D5 0.2 NS with 20 mEq KCl/L (<18 month old)
D5 0.45 NS with 20 mEq KCl/L
(PEARL – Do not add KCl until after first void and potassium level is known)

Deficit

Assessment of Hydration and Fluid Requirements

The clinical assessment of hydration is difficult and often inaccurate. In children who are dehydrated the accepted gold standard of assessment is acute weight loss but this is often not possible due to lack of accurate pre-illness weight.

A weight should be recorded at presentation and compared to any recent weight measurements

A Case of Dehydration

A 10 kg infant has had severe diarrhea for the past 2 days, decreased formula intake, a sunken fontenelle, no tears and oliguria.

How dehydrated is this infant?
What laboratory values do you want to obtain?
How do you want to manage this infant?

Management of Dehydration

Step 1 – Determine the presence and degree of dehydration
Step 2 – Obtain appropriate laboratory data (iso/hypo/hyper-natremia)
Step 3 – Bolus 20 mL/kg of NS (isotonic and will stay in the intravascular space)
Step 4 – Determine patient’s needs for next 24 to 48 hours

Maintenance + Deficit + On-going losses

Next
This infant is ~10% dehydrated given the history and PE findings Na 140, K 3.7, Cl 107, HCO3 22

Bolus 20 mL/kg NS → improved urine output
Still refusing po intake and still stooling at a rate of 20 mL/hr
Now what?

PEARL → 1000 mL (1L) = 1000 gm (1 kg)
Maintenance = 1000 mL (100 mL/kg/day)
Deficit = 1000 mL (10% of a 10 kg infant)
1000 mL – 200 mL (bolus given) = 800 mL remains to be given
On-going losses = 20 mL/hr → 480 mL/day

For isonatremic and assymptomatic hyponatremic dehydration

Give HALF of Maintenance and Deficit with minus of bolus in first 11 hours and remainder over the next 12 hours
first 11 hours: (1/2Maintenance )+ Deficit – Bolus = (1/2x1000)+1000- 200=1300 mL
Therefore: Run 1300 mL over 11 hours at 118 mL/hr
Then, next 500 mL over 12 hours at 41 mL/hr

Hypernatremic Dehydration

Total body water losses in excess of sodium losses
Hypernatremia must be corrected SLOWLY
Hyperosmolality causes cells to shrink – especially in the CNS
Correcting too quickly will cause fluid to be rapidly drawn into brain cells
Cerebral edema is BAD
Hypernatremic Dehydration

Case
A 5 kg infant presents with a 5 day history of viral syndrome with fever, vomiting and diarrhea. Signs and symptoms reveal an infant who is 10% dehydrated. Laboratory data reveals a Na of 160.

Hypernatremic Dehydration
- Hypernatremic dehydration is corrected EVENLY over 48 hours
- Bolus 20 mL/kg NS to restore intravascular volume
- Maintenance = 100 mL/kg x 5 kg = 500ml/day
- 48 hours of maintenance = 1000 mL
- Deficit = 0.5 kg = 500 mL
- 500 mL – 100 mL (bolus given) = 400 mL remain to be given

Total fluids over a 48 hour period is 1400 mL or 29 mL/hr
Maintenance and deficit is used 0.45%NS D5W

Oral Rehydration Therapy

<table>
<thead>
<tr>
<th>Indications</th>
<th>Contraindications</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;10% dehydrated</td>
<td>&gt;10% dehydrated/circulatory instability</td>
</tr>
<tr>
<td>Following initial volume resuscitation</td>
<td>Severe vomiting</td>
</tr>
<tr>
<td></td>
<td>Abdominal distention/ absent bowel sounds</td>
</tr>
<tr>
<td></td>
<td>Severe hypo- or hyper- natremia</td>
</tr>
</tbody>
</table>

Examples – MOH ORS, Oralite, WHO rehydration solution

Oral rehydration therapy in acute gastroenteritis

<table>
<thead>
<tr>
<th>Assess</th>
<th>Mild dehydration</th>
<th>Moderate dehydration</th>
<th>Severe dehydration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Child general condition</td>
<td>Well, alert</td>
<td>Restless/ irritable</td>
<td>Lethargic/ unconscious</td>
</tr>
<tr>
<td>Sunken eyes</td>
<td>Absence</td>
<td>Presence</td>
<td>Presence</td>
</tr>
<tr>
<td>Thirsty level</td>
<td>Drink normally</td>
<td>Drinks eagerly, thirsty</td>
<td>Not able to drink or drink poorly</td>
</tr>
<tr>
<td>Pinch skin of abdomen</td>
<td>Skin goes back immediately</td>
<td>Skin goes back slowly</td>
<td>Skin goes back very slow (&gt;2 sec)</td>
</tr>
<tr>
<td>Classify</td>
<td>No dehydration (&lt;5%)</td>
<td>≥ 2 above signs present: some dehydration (5-10%)</td>
<td>≥ 2 above signs present: severe dehydration (&gt; 10%)</td>
</tr>
<tr>
<td>Treat</td>
<td>Give fluid and food to treat diarrhoea at home, Treatment Plan A</td>
<td>Give fluid and food for some dehydration, Treatment Plan B</td>
<td>Give fluid for severe dehydration, Treatment Plan C</td>
</tr>
</tbody>
</table>

Note: Children on treatment Plan C may show signs of shock, such as tachycardia, weak peripheral pulses, delayed capillary refill time (>2 seconds), cold peripheries, depress of mental state with or without hypotension.
Plan A/5% dehydration
- Treat at home
- Counsel mom 3 main rules:
  - give extra fluid, continue feeding, when to return
Steps:
- Give mom 8 packets of ORS, 1 pack dissolve in 250ml of cooled after boiled water
- Tell mom how to use ORS
- >2y.o.:50-100ml after each stool
- >2y.o.:100-200ml after each stool
- Or 10ml/kg after each loose stool
- Give by small sips of spoon, if vomit then wait 10 minutes and continue slower.
- Continue breastfeed for breastfed child, and continue give food based fluids for older child
- Avoid food with high in simple sugar as osmotic load will worsen the diarrhea

When to return hospital?
- Unable to drink
- Become sicker
- Develops fever
- Has blood in stool

Plan B/5-10% dehydration
- Give ORS in 4 hours in such amount

<table>
<thead>
<tr>
<th>Age</th>
<th>Up to 4 months</th>
<th>4-12 months</th>
<th>12 months – 2 years</th>
<th>2-5 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight</td>
<td>&lt;6kg</td>
<td>6-&lt;10kg</td>
<td>10-&lt;12kg</td>
<td>12-19kg</td>
</tr>
<tr>
<td>Volume of ORS (ml)</td>
<td>200-400</td>
<td>400-700</td>
<td>700-900</td>
<td>900-1400</td>
</tr>
</tbody>
</table>

Footnote:
1. Use the child’s age only when you do not know the weight. The approximate amount of ORS required (in ml) can be calculated by multiplying the child’s weight (in kg) x 75
2. If the patient wants more ORS than shown, give more

- Method of using same as plan A only it’s done in 4 hours with different amount
- After 4 hours we reassess the child for dehydration and choose plan A, B or C

Plan C/>10% dehydration
- Start IV, give 100ml/kg ringer lactate/Ns as BELOW
- 1st start by 20ml/kg NS bolus. Repeat until perfusion improved
- Give the remaining fluid over 5 hours(<1y.o.) or 21/2 hour(>1y.o.)
- Reassess pt after every bolus and stop the bolus once perfusion improves/overload is suspected
- If perfusion not improve suspect shock, myocarditis, myocardiopathy, pericarditis etc.
- Also give 5ml/kg/hour of ORS as soon as child can drink usually it’s after 4 hours for infant and 2 hours for child.
- Reassess pt after 6 hours for infant and 3 hours for child and determine to continue for plan A, B or C
Electrolytes Disbalance
Norm Sodium: 136-145mEq/L (mmol/L)
Norm Kalium: 3.5-5.0mEq/L (mmol/L)

Hyponatremia

Symptoms
- Mild gradual- asymptomatic
- Moderate- apathy, nausea, vomiting, weakness, headache, change mental status confusion, lethargy, muscle cramp, hyperreflexia, restlessness
- Severe- coma, seizure it’s emergency

Causes
- High osmolality - hyperglycemia (1.6mg%Na/100mg%glucose)
- Normal osmolality - hypertriglyceridemia
  - hyperproteinemia (Pseudo-hyponatremia)
- Low osmolality - multiple cause

Determine osmolality
Direct biochemical measurement
Calculation
- Osmolality = 2 (serum Na +K) + glucose(rbs) + BU all in mmol/L
  Normal osmolality is 275-295

Hyponatremia with high osmolality
- Mechanism - increase solute in blood usually glucose
- Cause osmotic fluid shift from intracellular space into blood
- This dilutes the Na level in blood
- How glucose affects Na level?
- Na fall 1.6 in every rise of glucose 100mg/dL

Hyponatremia with normal osmolality
For 1L of blood, there’s less measured Na in blood d/t elevated protein/lipid
But Na concentration in water compartment is NORMAL therefore this condition also call pseudo-hyponatremia
How to approach hyponatremia?

**Case**

16 y.o. man present with 1 week of vomiting and 2 days of confusion. Take thyroxin, fluoxetine, and chlorpropamide. Na=126, glucose=342, and pneumonia.

What are possible causes of hyponatremia?

What do you need to know to determine the cause?

**Treatment of Hypornatremia:**

- Slow correction!
- up to 2meq/l/h, and not more than 10mEq/day
- Hypovolemic: Add 0.9% NaCl to correct volume deficit
- Isovolemic:
  - correct underlying cause
  - water restriction
- Hypervolemic:
  - water restriction
  - loop diuretics
  - optimize cardiac performance in severe CHF patient
  - hypertonic saline in severe symptomatic patients
Severe hyponatremia
- Severe hyponatremia (serum Na < 109 mEq/L; effective osmolality < 238 mOsm/kg) restriction of water intake.
- Treatment is more controversial when neurologic symptoms (eg, confusion, lethargy, seizures, coma) are present.
- serum Na be raised no faster than 1 mEq/L/h, but replacement rates of up to 2 mEq/L/h for the first 2 to 3 h have been suggested for patients with seizures. Regardless, the rise should be ≤ 10 mEq/day over the first 24 h. More vigorous correction risks precipitation of osmotic demyelination syndrome.

Hypertonic (3%) saline (containing 513 mEq Na/L) may be used but only with frequent (q 2 to 4 h) electrolyte determinations. For patients with seizures or coma, ≤ 100 mL/h may be administered over 4 to 6 h in amounts sufficient to raise the serum Na 4 to 6 mEq/L.

Change in serum Na= \[ \text{infused Na} - \text{Serum Na} \]
\[ \text{TBW+1} \]
Case
50kg boy with seizure Na=109mmol/L (TBW=30L)
What is our plan?

1L 3%Nacl will elevate in Na in blood = \( \frac{513-109}{30+1} \)
= 13mmol/L

increase Na at rate 1mmol/L/h for 1st 3hr=3mmol/L
We need increase Na 3mmol/L=1L x(3/13)=0.23L
Means in 1st 3 hours give 3%NaCl in= 76.9ml/hr
Then next 6hr we halve the initial rate=38.4ml/hr
Also we give frusemide with titration to ensure urine output similar to input

Hypernatremia

Principal Causes of Hypernatremia

### Hypovolemic hypernatremia
- Decreased TBW and Na with a relatively greater decrease in TBW
- GI losses
  - Diarrhea
  - Vomiting
- Skin losses
  - Burns
  - Excessive sweating
- Renal losses
  - Intrinsic renal disease
  - Loop diuretics
  - Osmotic diuresis (glucose, urea, mannitol)

### Euvolemic hypernatremia
- Decreased TBW with near-normal total body Na
- Extrarenal losses from respiratory tract
  - Tachypnea
- Extrarenal losses from skin
  - Excessive sweating
  - Fever
- Renal losses
  - Central diabetes insipidus
  - Nephrogenic diabetes insipidus

### Hypervolemic hypernatremia
- Increased Na with normal or increased TBW
- Hypertonic fluid administration
  - Hypertonic saline
  - NaHCO₃
  - TPN
- Mineralocorticoid excess
  - Adrenal tumors secreting deoxycorticosterone
  - Congenital adrenal hyperplasia (caused by 11-hydroxylase defect)

Hypernatremia symptoms
- Lethargy
- Confusion
- Seizure and coma
- Thirst
- Hyperpyrexia

Hypernatremia treatment
- Initial treatment
  - Treat dehydration
  - Isotonic fluids
  - Slow correction of Na (1mEq/2h)
- If does not stay corrected, consider diabetes insipidus, then we need to do water restriction test

Hypernatremia:

<table>
<thead>
<tr>
<th>Treatment:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Free water deficit (L) = ( 0.60 \times \text{TBW(kg)} \times (([\text{Na}^+]_{\text{serum}}/140)-1) )</td>
</tr>
<tr>
<td>2. Avoid rapid correction - ½ water deficit should be corrected over the first 24 hour, and not more than 10mEq/d. the remainder corrected over the following 2~3 days</td>
</tr>
<tr>
<td>Oral replacement is preferable</td>
</tr>
<tr>
<td>IVs ( \rightarrow ) D5W, D5 + 0.45%Nacl</td>
</tr>
</tbody>
</table>
HyperNa hypervolume—free water deficit can be replaced with 5% D/W, with a loop diuretic. Other electrolytes, including serum K, should be monitored and should be replaced as needed.

Hypernatremia and euvolemia—free water can be replaced using either 5% D/W or 0.45% saline.

Hypernatremia and hypovolemia—0.45% saline with alternative 0.9%NS + 5% D/W to replenish Na and free water.

Note! all hypernatremia should be decreased slowly at rate 0.5mmol/hr in 48hrs

Hypokalemia

Common cause
- GI loss—diarrhea, tube drainage
- Medications
  - Diuretics—renal loss
  - Beta agonist (eg albuterol) increase K entry into cell
  - Insulin— increase K entry into cell

Rare cause
- Hyperaldosteronism—primary conn’s syndrome, cushing syndrome, bartter syndrome
- Licorise—mineralcorticoids effect of glycyrrhizic acid

Case
13 y.o. with CHF had diarrhea for 3 days. Now has increasing SOB. Meds include furosemide and albuterol. Received multiple albuterol treatments in ER
3 possible cause of hypokalemia

Hypokalemia symptoms
- Mild—assymptomatic
- Moderate—muscle weakness, muscle cramp, and non specific T and U wave changes
- Severe—cardiac arrhythmia
- All levels will potentiate digitalis toxicity

DIAGNOSIS
Hypokalemia treatment

- Replace K
  - Orally or parenterally depend on severity
  - Better give iv. Cos po induce vomit
  - Maximum IV K administer: 0.4mmol/kg/h
  - Maximum oral K administer:
    - <5y.o. 1mmol/kg/dose
    - >5y.o. 0.5mmol/kg/dose
  - IV-max 10(20)mEq/hour. Excessive rate causes cardiac conduction defects heart block and/or asystole!!

Hyperkalemia

- Increased intake
  - Excesss K administration
  - Dg induced
- Artifact venipuncture(pseudohyperkalemia)
• Movement of K from cell into blood
  o Acidosis (H into cells, K out of cells)
  o Rhabdomyolysis
  o Familial periodic paralysis (rare)
• Decreased renal excretion
  o Renal failure
  o Meds: K-sparing diuretics, ACE inhibitors
  o Hypoaldosteronism
  o Adrenal insufficiency symptoms
• Generally asymptomatic
• May have weakness
• Sudden cardiac conduction defect
  o Execution
  o Preceded by classic EKG changes

**Hyperkalemia treatment**
• Protect heart - if ECG changes
  o CaCl or CaGluconate infusion (10% 5mg/kg in 10 minutes)
• Lower serum potassium
  o Glucose and insulin (IV dextrose 1ml/kg D50%W in 30 minute with actrapid)
  o Bicarbonate (alkalosis drives K into cells) (8.4% NaHCO3 1mmol/kg in 30 minute)
  o Nebulised salbutamol 2.5-10mg/kg/dose
• Remove potassium from body
  o Cation-exchange resin (kayexalate)
  o Dialysis if renal failure
CASE DISCUSSION 3

HOPD
• The parents of a 3 y.o. girl bring her to your office for routine check up.
• A quick review of her chart reveals that she was delivered at term without prenatal or postnatal Problems.
• Her development thus far has been normal.
• Family reports that is a rather “picky” eater.
• She doesn’t like to eat meats, but loves to drink large quantities of whole milk

O/E
• The child is normal with perhaps a bit of paleness to her mucous membranes.
• The family has not travelled and they report no family history of anaemia.

Ix
— FBC shows haemoglobin is 8 g/dl, microcytosis with hypochromia.

**Question 1:** What are the mostly aetiology for this child’s condition?

**Question 2:** What is the most appropriate test to confirm this aetiology?

**Question 3:** What are the treatments?

**Guideline:**
1. First, ask for symptoms of anaemia:
   ✓ Lethargy
   ✓ Easy fatigability
   ✓ Tiredness
   ✓ Dyspnoea on exertion
   ✓ Worsening of angina/intermittent claudication

2. Then, find out the cause:
   • **Any abdominal pain, h/o of melena, hematochezia?** Peptic ulcer disease, Bowel malignancy (in adults)
   • If there's blood loss, ask about frequency and amount
   • Is there any hemoptysis, hematemes, menorrhagia at the same time?
   • Is there h/o of passing greasy, foul-smelling stools which floats up? ♦ Malabsorption
   • Dietary intake of folate? Iron? B12?
     o Sufficient???
• Any excess intake of alcohol (macrocytic anaemia) – adults
• Any h/o of chronic illness??
  o Anaemia of chronic dss (RA, CRF, TB, CCF (adult))
• Any h/o of travelling to endemic areas of malaria/hook worm infestation?
• How about his/her drug intake?
• NSAIDs, Aspirin, sulphonamide, chloramphenicol
• Is it due to haematological malignancy?
• Recurrent infection, bleeding tendency, any one of them?
• Any previous surgery done? (gastrectomy, blind-loop syndrome)
• Any family history of anaemia? (G6PD deficiency, Thalassemia, H-spherocytosis)

3. DDx IDA and Thalassemia

Menzel Index = \( \frac{MCV}{RBC} \)

- < 13 Thalassemia trait
- > 13 IDA
- If between 13 – 15, look at MCHC

IF MCHC < 26 / 27, suggest Thalassemia trait.
PART 4: ACID BASE BALANCE

Parameters for acid base balance
From ABG:
- pH = 7.35 – 7.45
- PaCO₂ = 40 – 45 mmHg / 5.3 – 6 kPa
- PaO₂ = 60 – 75 mmHg / 8 – 10 kPa
- Base excess = 0 ± 5.0 mmol/l
- HCO₃⁻ = 20 – 25 mmol/L

Regulation of acid-base balance
1) Chemical Buffers
   - The Bicarbonate (HCO₃⁻) Buffer
   - The Phosphate Buffer
   - The Protein Buffer
2) Respiratory Control of pH
3) Renal Control of pH *

Characteristics of Primary Acid-Base Disturbances

<table>
<thead>
<tr>
<th></th>
<th>pH</th>
<th>PaCO₂</th>
<th>HCO₃⁻</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>7.4</td>
<td>40 mmHg</td>
<td>24 mEq/L</td>
</tr>
<tr>
<td>Metabolic acidosis</td>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Respiratory acidosis</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Respiratory alkalosis</td>
<td>↑</td>
<td>↓</td>
<td>↓</td>
</tr>
</tbody>
</table>

↑ ↓ Primary Disturbance
↑ ↓ Compensatory Response

Acidosis: < pH 7.35; Alkalosis: > pH 7.45
7.35 -----------7.4-----------7.45

<table>
<thead>
<tr>
<th>Acid Base Disorders</th>
<th>Compensatory Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic acidosis</td>
<td>Hyperventilation</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>Hypoventilation</td>
</tr>
<tr>
<td>Respiratory acidosis</td>
<td>Renal HCO₃⁻ reabsorption</td>
</tr>
<tr>
<td>Respiratory alkalosis</td>
<td>Renal HCO₃⁻ secretion</td>
</tr>
</tbody>
</table>
### Causes of Acidosis

- **Metabolic acidosis**
  - renal failure
  - septicaemia
  - hypoxia
  - hypothermia
  - hypotension
  - cardiac failure
  - dehydration
  - hyperkalaemia
  - hyperglycaemia
  - anaemia
  - intraventricular haemorrhage
  - drugs (e.g. acetazolamide)
  - metabolic disorders (often associated with hypoglycaemia)

- **Respiratory acidosis**
  - asphyxia: damage to respiratory centre
  - obstruction to respiratory tract
    - e.g. secretions, blocked endotracheal tube
  - respiratory distress syndrome (RDS)
  - pneumonia (acute lung disease)
  - pulmonary oedema
  - apnoea
  - Chronic lung disease
  - Opioids, narcotics, sedatives
  - Weakening of respiratory muscles

### Causes of Alkalosis

- **Metabolic alkalosis**
  - administration of sodium bicarbonate
  - pyloric stenosis
  - hypokalaemia
  - use of diuretics like thiazides and furosemide

- **Respiratory alkalosis**
  - asphyxia – overstimulation of respiratory centre
  - over ventilatin while on mechanical ventilation
Effects of Acidosis & alkalosis

**Acidosis:** < pH 7.35  ;  **Alkalosis:** > pH 7.45

<table>
<thead>
<tr>
<th>Acidosis:</th>
<th>Alkalosis:</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Depresses CNS, causing confusion, disorientation, and coma.</td>
<td>- CNS becomes hyperexcitable.</td>
</tr>
<tr>
<td>- Hyperventilation in metabolic acidosis and supressed respiration in respiratory acidosis</td>
<td>- Nerves fire spontaneously and overstimulate skeletal muscles</td>
</tr>
<tr>
<td>- High PaCO₂ in respiratory acidosis increases cerebral blood flow and risk of intraventricular haemorrhage</td>
<td>- Decreased cerebral blood flow causing cerebral ischaemia, convulsions</td>
</tr>
</tbody>
</table>

* Severe acidosis or alkalosis is lethal.

**Management of Metabolic acidosis**
1. Treat the underlying cause (dehydration, shock, DKA)
2. NaHCO₃ only when pH < 7.2 or HCO₃⁻ < 12 mmol/L

\[
\text{NaHCO}_3 \text{ required (mmol/L) = base deficit x BW(kg) x 0.3} \\
(1 \text{ ml of 8.4% NaHCO}_3 = 1 \text{ mmol HCO}_3^-)
\]

- Administer NaHCO₃ IV at a rate < 1 mmol/kg/min
- Rate of correction is depend on clinical picture and desired HCO₃⁻ level
- May be 1-2 mmol/kg initially (4.2% for neonates, 8.4% for older infants and children), then half the calculated HCO₃⁻ deficit for over 24 hours
- Do not give NaHCO₃ unless assisted ventilation is adequate (respiratory compensation by lungs cause hyperventilation)

3. Recheck ABG and electrolytes especially K⁺ periodically
4. Do not treat by hyperventilate (correct pH, but adverse effects on CO and pulmonary blood flow)
5. Do not give volume expansion (bolus NaCl) unless there is hypovolemia (acidosis decreases myocardial contractility)

Management of Metabolic alkalosis
- Treat the underlying causes.
- Usually associated with hypokalemia.
- Usually iatrogenic in premature infants - diuretic use, gastrointestinal losses

Management of Respiratory Acidosis
1. Treat underlying cause (Bronchodilators, O₂, Antibiotics/Drug therapy, Dialysis)
2. Ventilatory support is indicated: -- A steadily rise PaCO₂ at any stage in the disease
3. Correct electrolyte imbalance
4. IV NaHCO₃ in severe case

Special cases in rise of PaCO₂
* A gradual rise at the end of the first week in a LBW infant on ventilator = ?? PDA
* A rapid rise = Acute changes in the infant’s condition
  D.D: Pneumothorax, collapsed lobes, misplaced endotracheal tube
* A rise often accompanied by hypoxia following weaning = infant is not ready for weaning

In cases of low PaCO₂
* Low PaCO₂ in a infant on a ventilator = overventilation.
  So, wean down the ventilation.
* In pulmonary hypertension or cerebral oedema a slightly low PaCO₂ may be necessary in the treatment

Management of Respiratory Alkalosis
1. Treat underlying disease:
   - Removal of ingested toxins
   - Treatment of fever or sepsis (toxin)
   - Treatment of CNS disease
2. Monitor ABG
3. Assist patient to breathe slowly
4. Apply rebreather mask
5. Sedatives
6. In severe respiratory alkalosis: -- Breathing into a paper bag, which helps relieve acute anxiety and increases carbon dioxide levels

Test yourself:

Interpretation of ABG

<table>
<thead>
<tr>
<th>pH</th>
<th>PaCO₂</th>
<th>HCO₃⁻</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.36</td>
<td>25 mmHg</td>
<td>15 mmol/L</td>
</tr>
<tr>
<td>7.44</td>
<td>68 mmHg</td>
<td>49 mmol/L</td>
</tr>
<tr>
<td>7.2</td>
<td>60 mmHg</td>
<td>24 mmol/L</td>
</tr>
<tr>
<td>7.66</td>
<td>45 mmHg</td>
<td>40 mmol/L</td>
</tr>
<tr>
<td>7.22</td>
<td>55 mmHg</td>
<td>17 mmol/L</td>
</tr>
</tbody>
</table>
PART 5: FEBRILE FITS

Febrile fits = Febrile convulsions = Febrile seizures

Definition: A febrile convulsion is a *seizure* associated with *fever* in children between 3 months and 6 years old, in whom there is no evidence of intracranial pathology or metabolic derangement.

✓ Every 1 in 30 child have tendency to develop febrile convulsion.

<table>
<thead>
<tr>
<th>Simple Febrile Convulsions</th>
<th>Complex Febrile Convulsions</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Duration &lt; 15 min</td>
<td>• Duration &gt;15 min</td>
</tr>
<tr>
<td>• Generalized seizure</td>
<td>• Focal seizure</td>
</tr>
<tr>
<td>• Does not reoccur during febrile episode</td>
<td>• &gt;1 seizure during the febrile episode</td>
</tr>
<tr>
<td></td>
<td>• Residual neurological deficit post-ictally, eg: “Todd’s paralysis”</td>
</tr>
</tbody>
</table>

**Risk factors for recurrent febrile convulsions**
1. Family history of febrile convulsion (first or second degree relative)
2. Age < 18 months
3. Low degree of fever (< 38°C) during 1st febrile convulsion
4. Brief duration (< 1 hr) between onset of fever and convulsion
* No risk factor < 15 % recurrence
* Two or > risk factors > 30 % recurrence
* Three or > risk factors > 60 % recurrence

**Causes**
- Genetic predisposition
- Febrile condition
- Poisoning/toxins
- Meningitis, encephalitis
- Head trauma
- Metabolic disorder
  - hypo/hypernatraemia
  - hypocalcaemia/hypomagnesaemia
  - hypokalaemia
  - hypoglycaemia

**Investigation**
- FBC
- UFEME
- Dextrose stick
- Lumbar puncture*
- CT scan – should be considered in patients with complex seizures
- EEG – usually **UNNECESSARY** for a child with a 1st simple febrile seizure

**Signs & symptoms**
- Febrile convulsions usually occur during 1st Day of febrile illness with significant high fever of ≥38 °C.
- Focal seizure/Generalized tonic clonic in nature (muscles may stiffen or jerk)
- Skin becomes pale and sometimes turn blue for few seconds.
- Convulsion may last for several minutes depends on simple or complex febrile seizures
- Drooling of the saliva
- Up rolling of eyes (bilateral/unalateral)
- Urinary and bowel incontinence
- Changed or derranged consciousness (If febrile convulsion, usually patient loss consciousness)
- The child falls asleep after the fit stops and remains irritable and confused after waking up (post-ictal drowsiness). Child regain consciousness themselves or by parent’s help.
Lumbar puncture

Must be done (unless CI):
- Any signs suspicious of intracranial infection
- Prior to antibiotic therapy
- Persistent lethargy and not fully interactive 6-8 hours after the seizure

Strongly recommended:
- < 12 months old
- First complex febrile convulsion
- In district hospital without paediatrician
- If parents have a problem with bringing the child again to hospital in event of deterioration at home.

If a 1 year old child come with fits........

What must we do...

!!! Rule out intracranial pathology and metabolism derangement

If 1 year old baby fits

Try to confirm is it fit

HOW?? ➔ ask witness to describe what happened

- What was the child doing prior to seizure (triggering factors: previous accidents, head trauma, any changes in feeding of the child, etc.)
- Duration of the seizure and present repetition or not
- Changed/derangement of consciousness
- Present/absent of jerking, tonic stiffness
- What was the position of the hands (straight down or flexed)
- Up rolling of eyes (bilateral or unilateral)
- Drooling of saliva
- Blue coloring of the lips
- Urinary and bowel incontinence
- How the child regain consciousness after seizure
- Any previous episode of seizure
**Home care for the child**

- nothing parents can do to make the convulsion stop
- stay calm - don't panic
- Place child on a soft surface, lying on his or her side or back. Do not restrain child.
- **Do not put anything in their mouth, including fingers.** Child will not choke or swallow their tongue.
- *Try to watch exactly what happens, so that can describe it later to the doctors.
- Do not put a child who is having a convulsion in the bath.

Flow Chart For Children With Febrile Fits

```
Fits with Fever ↓
Note time of onset
Put the child on left lateral position
Loosen clothing

Fit stops in less than 15 minutes
Comfort child
Tepid sponging (CI in child less than 1 year because it can cause vascular collapse)
Give sy. Paracetamol 15mg/kg 6 hourly

Fit lasts for more than 15 minutes
Rectal diazepam available at home
Administer rectal diazepam
0.5 mg/kg to maximum 10mg
Or IV Diazepam (0.2 mg/kg to maximum 3mg if < 5 years,
or maximum 5mg if > 5 years) No rectal diazepam at home
```
**Differential Diagnosis**

| TABLE 1 |
|------------------|------------------|
| **Febrile Seizure** | **Infantile Spasm (West Syndrome)** |
| **Definition** | a convulsion in a child triggered by a fever. A febrile seizure may be as mild as the child’s eyes rolling or limbs stiffening. |
| | Violent flexor spasms of the head, trunk and limbs followed by extension of the arms (so-called ‘salaam spasms’). |
| **Age of onset** | 3 months – 6 years old |
| | 4- 6 months |
| **Etiologies** | 1)Infection of the nervous system (meningitis, encephalitis, or brain abscess) |
| | 2)unrecognized epilepsy triggered by fever |
| | 3) **simple febrile convulsions** (a common genetic predisposition to seizures in infancy that is precipitated by a rapid increase in body temperature) |
| | 1) **Metabolic** |
| | Phenylketonuria |
| | Maple syrup urine disease |
| | Ornithine accumulation |
| | Nonketotic hyperglycinemia |
| | Pyridoxine dependency |
| | Hypoglycemia |
| | Lipidosis |
| | 2) **Developmental malformations** |
| | Polymicrogyria |
| | Lissencephaly |
| | Schizencephaly |
Down syndrome and other chromosomal disorders

3) Neurocutaneous syndromes
   **Tuberous sclerosis**
   Sturge-Weber syndrome

4) Congenital infections
   Toxoplasmosis
   Cytomegalovirus
   Syphilis

5) Encephalopathies
   Post-asphyxia
   Post-traumatic
   Posthemorrhagic
   Postinfectious

<table>
<thead>
<tr>
<th>Classification</th>
<th>1-Simple febrile seizure</th>
<th>2-Complex febrile seizure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical manifestations</td>
<td>- are usually brief, generalised tonic-clonic seizures occurring with a rapid rise in fever (1 min to a max of 5 min) - loss of consciousness - becoming stiff. - stop breathing for about 30 sec. - loses control of their bladder/ bowel. - twitching/ spasms of limbs and face muscles too. - rolling of eyes upwards. - drooling of saliva - head is thrown backwards and the arms and legs begin to jerk. - skin becomes pale and sometimes turn blue for few seconds. - the child falls asleep after the fit stops and remains irritable and confused after waking up (post-ictal drowsiness)</td>
<td>Initial phase: <strong>flexion</strong> and <strong>extension</strong> in various combinations such that the head may be thrown either backward or forward. The arms and legs may be either flexed or extended. Spasms occur most frequently when the child is awakening from or going to sleep. Each jerk is followed by a brief period of relaxation, then repeated multiple times (20-30 spasms) in clusters of unpredictable and variable duration. Many clusters occur each day.</td>
</tr>
</tbody>
</table>

*May be misinterpreted as **colic**. Social interaction often deteriorates - a useful marker in the history.

<table>
<thead>
<tr>
<th>Diagnostic criteria</th>
<th>1) anamnesis</th>
<th>2) clinical picture</th>
<th>3) a full physical and neurologica examination.</th>
<th>4) blood test and urine test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment</td>
<td>most children require no treatment</td>
<td>adrenocorticotropic hormone, oral corticosteroids, benzodiazepines, and valproic acid</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prognosis</td>
<td>excellent</td>
<td>poor prognosis</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**TABLE 2**

<table>
<thead>
<tr>
<th>Definition</th>
<th>Epilepsy</th>
<th>Funny turns</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>a brain disorder that causes recurring seizures, when clusters of nerve cells, or neurons, in the brain send out the wrong signals</td>
<td>a sudden, short-lived episode when a child becomes dizzy, wobbly on the feet, confused or perhaps blacks out (collapse) altogether</td>
</tr>
<tr>
<td>Age of onset</td>
<td>After 1 year till adolescent (15 years)</td>
<td>6 months till adolescent</td>
</tr>
<tr>
<td>--------------</td>
<td>----------------------------------------</td>
<td>-------------------------</td>
</tr>
</tbody>
</table>

**Etiologies**

1) low oxygen during birth  
2) head injuries that occur during birth or from accidents during youth or adulthood  
3) brain tumors  
4) genetic conditions that result in brain injury, such as tuberous sclerosis  
5) infections such as meningitis or encephalitis  
6) stroke or any other type of damage to the brain  
7) abnormal levels of substances such as sodium or blood sugar

**Classification**

**Generalized Epilepsy**

1. Absence seizure  
2. Myoclonic seizures  
3. Tonic seizures  
4. Tonic-clonic seizures  
5. Atonic/Astatic seizures

**Partial Epilepsy**

1. Simple partial seizure  
2. Complex partial seizure  
3. Partial seizure with secondary generalisation

**Clinical manifestations**

**Partial or focal seizures:** Only part of the brain is involved, so only part of the body is affected. Depending on the part of the brain having abnormal electrical activity, symptoms may vary.

*Frontal seizures* - involve the motor cortex. May lead to clonic movements, which may travel proximally (Jacksonian march). Asymmetrical tonic seizures can be seen, which may be bizarre and hyperkinetic and can be mistakenly dismissed as non-epileptic events.

*Temporal lobe seizures*, the most common of all the epilepsies - may result in strange warning feelings or aura with smell and taste abnormalities and distortions of sound and shape. Lip-smacking, plucking at one’s clothing, and walking in a non-purposeful manner (automatisms) may be seen, following spread to the pre-motor cortex. Déjà-vu and jamais-vu are described (intense feelings of having been, or never having been, in the same situation before). Consciousness can be impaired and the length of event is longer than a typical absence.

*Occipital seizures* - cause distortion of vision.  
*Parietal lobe seizures* - cause contralateral dysesthesias (altered sensation), or distorted body image.

#simple partial focal seizures - consciousness is retained  
#complex partial focal seizures - consciousness is

1) Breath-holding attacks  
2) Reflex anoxic seizures  
3) Syncope  
4) Migraine  
5) Benign paroxysmal vertigo  
6) Others: Cardiac arrhythmia, Tics, daydreaming, Night terrors, Self-gratification, Minor stroke, After medication  
- Nonepileptic attack disorder (NEAD):  
  *Pseudoseizures, Fabricated, Induced illness

---

# Note: The table and text are extracted from a source on epilepsy, focusing on age of onset, etiologies, classification, and clinical manifestations. The table lists types of seizures and their characteristics, along with potential causes and diagnostic criteria. The text provides a detailed explanation of each type of seizure, including descriptions of symptoms, triggers, and associated conditions. The information is presented in a structured manner, allowing for easy reference and understanding of the material. The table serves as a valuable resource for medical professionals and patients seeking detailed information on epilepsy.
Children with complex partial seizures may, however, retain some memory of the event.

**Partial focal seizures with secondary generalisation** - focal seizure manifest on EEG and followed by generalised tonic-clonic seizure.

**Generalized seizures:** All areas of the brain (the cortex) are involved in a generalized seizure. Sometimes these are referred to as grand mal seizures.

- To the observer, the person experiencing such a seizure may cry out or make some sound, stiffen for some seconds, then have rhythmic movements of the arms and legs. Often the rhythmic movements slow before stopping.
- Eyes are generally open.
- The person may not appear to be breathing. The person is often breathing deeply after an episode.
- The return to consciousness is gradual and should occur within a few moments.
- Loss of urine is common.
- Often people will be confused briefly after a generalized seizure.

**Diagnostic criterias**

1) **Anamnesis**
2) **Clinical picture**
3) A complete physical and neurological exam of muscle strength, reflexes, eyesight, hearing, and ability to detect various sensations
4) **Blood tests**
5) **EEG**
6) **MRI**

1) **Anamnesis**
2) **Clinical picture** (clear description from witness)
3) **Neurological and physical examinations**
4) **ECG (cardiac arrhythmia) &/ Holter-monitoring**
5) **Blood tests**
6) **EEG**

**Treatment**

1) **Antiepileptic drugs (AEDs):** *Tegretol or Carbathol (carbamazepine), Valproate, Felbatal, Gabitril, Keppra, Dilantin (Phenytoin), Neurontin (Gabapentin), Valium and similar tranquilizers such as Klonopin or Tranxene,*
2) The ketogenic diet helps some children and adults with epilepsy.
3) **Vagal nerve stimulation,** delivered using externally programmable stimulation of a wire implanted around the vagal nerve, may possibly be useful for focal seizures; trials are being conducted.
4) **Surgery.** The main procedure is temporal lobectomy for mesial temporal sclerosis but other procedures include hemispherectomy or hemispherotomy (does not involve hemisphere removal and problems with shifts in space) and focal resections.

*Antiepileptic drugs should not be used as 1st line drug, before you make the diagnosis.
Prognosis

Though there's no cure, the prognosis for most epilepsy is quite good. Up to 80% of people can control their condition with medicine.

Excellent

Febrile convulsions are benign events with excellent prognosis
- 30% recurrence after 1st attack
- 48% recurrence after 2nd attack
- If simple febrile fits - does not affect intellectual performance or no risk of developing epilepsy
- If complex febrile fits, 4-12% risk of subsequent epilepsy.
- No deaths were reported from simple febrile convulsions
HEMATURIA

If patient present with Hematuria /red urine, next steps:
- History
- Physical examination
- Urine analysis

**History** – trauma of external genitalia
- drugs???
- recent UTI infections - ASO titre and C3 level – suspect post infection AGN

What is Pink diaper syndrome?

**Causes of red urine...**

a) Haemoglobinuria
b) Drugs: quinine, sulfonamides
c) Chemicals: CO, CO2, chloroform, fava beans, naphthalene, oxalic acid, henylhydrazine, snake venom, tin
   - Exercise, cold, intravascular hemolysis ~Myoglobinuria
d) Discored urine – Foods and dyes blackberries, beets, vegetable
   - Drugs: phenytoin, phenophthalein, phenothiazides, pyridium, rifampin, chloroquine, deferoxamine
e) Internal pigments: bilirubin, urates (red diaper syndrome), porphyrins, metabolic errors (alcaptonuria, tyrosinosis, homogentisic acid)
f) Hemoglobin – Acute Intravascular Hemolysis : G6PD PNH
   From Intravascular coagulation: Sepsis or hemolytic Uremic syndrome
   Myoglobin – Injury, burn, myositis, asphyxia

**Differential diagnosis**
- GN
- congenital nephritis (syndrome Al-Port)
- interstitial nephritis
- toxico-allergic nephropathy
- vasopathy
- thrombocytopathy
- coagulopathy
- DIC
- secondary hemolytic anemia
- dysplasia, aplasia, dystopia
- Reflux
- tumor of kidney
- TB
URINARY TRACT INFECTION...

DEFINITIONS

UTI is growth of bacteria in the urinary tract.

- **Significant bacteriuria** is > 105 colony forming units of a single organism per ml of freshly voided urine (Kass).
- **Acute pyelonephritis** is infections involving the renal parenchyma which carries a higher risk of renal scarring.
- **Acute cystitis** is infections limited to the lower urinary tract presenting clinically with acute voiding symptoms, including dysuria, urgency, frequency, suprapubic pain, and incontinence.
- **Asymptomatic bacteriuria** is presence of bacteriuria in repeated samples from a otherwise asymptomatic child, usually detected at routine investigations.

Simplified clinics

<table>
<thead>
<tr>
<th>Infants and toddlers &lt;2 years</th>
<th>Children &gt; 2 years old</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fever - unexplained fever</td>
<td>Lower urinary symptoms</td>
</tr>
<tr>
<td>Failure to thrive</td>
<td>Frequency</td>
</tr>
<tr>
<td>Jaundice</td>
<td>dysuria</td>
</tr>
<tr>
<td>irritability</td>
<td></td>
</tr>
</tbody>
</table>

Physical Examination

- General examination, growth, blood pressure
- Abdomen for distended bladder, ballotable kidneys, other masses, genitalia.
- Back for any spina bifida
- Lower limbs - deformities, wasting etc in neurogenic bladder

Investigation for UTI

- Urinalysis
- Urine culture
- Ultrasound
- Micturating Cystourethrogram (MCUG)
- DMSA scan

Urinalysis

- Leucocyte esterase
- Nitrite
- Pyuria
- Bacteria

** the three most useful tests are the leucocyte esterase test, nitrite test and microscopy.

Collection of urine

- **Bag urine specimen** – High contamination rate of up to 70%. A positive culture should be confirmed with a clean catch or suprapubic aspiration specimen (SPS)
- **Clean catch specimen** – in a child who is bladder trained.
- **Catheterisation** - sensitivity of 95% & specificity of 99% compared to SPA. There is a low risk of introducing infection.
- **Suprapubic aspiration (SPA)** - The best technique ("gold standard") of obtaining an uncontaminated urine sample and any growth from SPA specimen is significant.
Ultrasound
❖ Dilatation of upper tracts, renal sizes, bladder anomaly.
❖ Urgent if obstruction is suspected

MCUG
❖ Look for VUR (vesicoureteral reflux)
❖ Posterior urethral valve
❖ Done when child is free of infection
❖ 6 weeks after infection

DMSA scan: 99mTc dimercaptosuccinic acid (DMSA) scan
❖ During infective phase: uptake defect=represent acute inflammation
❖ Post infection: Uptake defect= renal scar
❖ Estimates the differential function of renal function of both kidneys Imaging

Management
General
❖ Encourage child to drink plenty
❖ Empty bladder adequately
❖ Avoid constipation
❖ Proper toilet habits- wipe from front to back
❖ Encourage double micturition

Specific
❖ Acute Form
  o Start antibiotic without delay after collection of urine
  o Oral antibiotic in non-toxic child
  o Intravenous antibiotic for toxic-appearing child or poor oral intake
  o Duration of treatment 7-14 days
  o Repeat urine culture after 2 days of Tx. Change antibiotic if bacteriuria persist.
  o Children treated for UTI should continue antibiotic at prophylactic dosage while awaiting hospital referral and imaging studies
❖ Normal renal tract
  o Prophylactic antibiotic not required unless if there recurrent infections (>3 per year)
  o Urine culture during any febrile illness
  o Follow-up for at least 1 year
❖ No VUR but renal scarring present
  o Repeat DMSA 6-12 months to see if defect is still there
  o Repeat urine culture only symptomatic
  o Annual BP
  o Annual renal function test and urinalysis

Antibiotic Tx
<table>
<thead>
<tr>
<th>Drug</th>
<th>Therapy</th>
<th>prophylaxis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trimetoprim</td>
<td>4mg/kg/dose</td>
<td>1-2mg/kg ON</td>
</tr>
<tr>
<td>Nitrofurantoin</td>
<td>1mg/kg/dose</td>
<td>1mg/kg ON</td>
</tr>
<tr>
<td>Amoxicillin</td>
<td>20-40mg/kg/dose</td>
<td></td>
</tr>
<tr>
<td>Amikacin</td>
<td>15mg/lg/dose</td>
<td>OD</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>5mg/kg/dose</td>
<td>OD</td>
</tr>
<tr>
<td>Cephalexin</td>
<td>50-100mg/kg/day</td>
<td></td>
</tr>
</tbody>
</table>
**Vesicoureteral Reflux**

**Definition:** Retrograde flow of urine from bladder into ureter and collecting system

Can be due to:

- congenital anomaly of ureterovesical junction
- posterior urethral valve, neuropathic bladder or voiding dysfunction → high pressure in bladder → reflux

VUR → recurrent UTI → progressive renal scarring → 1. ESRD  
2. Hypertension

**Classification**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>ureter only</td>
</tr>
<tr>
<td>2</td>
<td>ureter and upper collecting system (w/o dilation)</td>
</tr>
<tr>
<td>3</td>
<td>mild or moderate dilation of ureter &amp; mild and moderate dilation of renal pelvis but no and slight blunting of fornices</td>
</tr>
<tr>
<td>4</td>
<td>moderate dilation/ tortuosity of ureter with moderate dilation of pelvis and calyces and complete obliteration of sharp angles of fornices with maintenance of papillary impression</td>
</tr>
<tr>
<td>5</td>
<td>gross dilation and tortuosity of ureters, renal pelvis and calyces; papillary impression not visible in majority of calyces</td>
</tr>
</tbody>
</table>

**Investigation**

- MCUG (micturating cystourethrogram)
- DMSA scan

**Management**

According to grades:

- **normal renal tracts** → no need antibiotic prophylaxis
- **no VUR but renal scarring present**  
  → uncertain role of antibiotic  
  → urine culture if symptomatic  
  → annual BP surveillance, renal f(x) test and urine for proteinuria
- **VUR grade I-II** → no antibiotic prophylaxis
- **VUR grade III-IV** → a/biotic prophylaxis at least 1 yr or watch and treat each infection with a/biotic
- **VUR V at all ages/ bilateral VUR IV < 1 yr**  
  → start a/biotic. Consider circumcision for boys. After 1 yr, repeat MCUG # persisting VUR III–IV at follow up examination  
  → stop prophylaxis. Treat each UTI with short courses of a/biotics. If recurrent infection r new scars, consider endoscopic injection or surgery
**Acute Renal Failure**

**Definition:** Abrupt rise in serum creatinine and decreased GFR result in inability of kidney to regulate fluid and electrolyte balance

**Common causes of ARF**

a) Prerenal:
   - Hypovolemia (dehydration, bleeding, nephrotic syndrome)
   - CHF

b) Renal:
   - GN (infections, SLE, Chronic GN)
   - ATN (drugs: aminoglycosides, chemotherapy, toxins: Mb, Hb, venom: bee sting)
   - Tumor lysis & uric acid nephropathy
   - Vascular lesions (hemolytic uremic syndrome, renal venous thrombosis)
   - Sepsis
   - Post-renal:
     - Urethral obstruction (posterior urethral valves)
     - Bilateral ureteric obstruction
     - Obstruction of solitary kidney

**Clinical features:** Of underlying causes

- **Oliguria**
  - Children: <300ml/m²/d
  - Neonates: <1ml/kg/hr

- **Non-oliguric**

**Fractional excretion of Na** $\text{FE}_{\text{Na}}$

Useful to differentiate pre-renal from renal causes of ARF

$$\text{FE}_{\text{Na}} = \left( \frac{\text{Urine Na}}{\text{Plasma Na}} \right) \times \left( \frac{\text{Plasma Creatinine}}{\text{Urine Creatinine}} \right) \times 100\%$$

**Management of ARF**

1. Fluid balance
2. Hypertension
3. Metabolic acidosis
4. Electrolytes abnormalities
5. Nutrition
6. Medications

**1. Fluid management**

a) Hypovolemia
   - In prerenal failure secondary to severe volume depletion (hypovolemia) → fluid resuscitation with NS or hartmann’s sol 20ml/kg over 30’ to 60’
   - **BUT** 1ST, WE NEED TO DIFFERENTIATE IS PRERENAL FAILURE OR RENAL FAILURE.
   - ❖ If dehydration is the reason for oliguria, urination should be increases within 2-4 hours after normovolemia is established.

b) Hypervolemia: i/v frusemide 2mg/kg/dose (over 10-15’) tds, max 5mg/kg/dose
   OR i/v frusemide infusion 0.5mg/kg/hr

**Ix**

- **Blood:** FBC, FBP, urea, electrolytes, creatinine, ABG, serum albumin, Ca&P, Uric acid
- **Urine:** protein, UFEME
- **USE:** in emergency if cases unknown
- **Other Ix** as determined by cause
- *blood urea is affected by state of hydration, GI bleed, high protein intake, catabolic state**

**Prerenal ARF** $\text{FE}_{\text{Na}} < 1\% (2.5\% in Neonates)$

**Intrinsic ARF** $\text{FE}_{\text{Na}} > 2\%$ in children
• Dialysis indicated if no response
• monitor: BW, BP, clinical status, nutritional needs, Input/output, hypertension
• If child is oliguric and fluid overload. Frusemide 2-4 mg/kg/dose given slowly i/v (4mg/min)
• If remains oliguric or anuric → dialysis

2. Antihypertensive drugs during emergencies

• Nifedipine 0.25-0.5 mg/kg oral pm
• Labetalol 0.2-0.4mg/kg/dose IV stat OR 0.25-3 mg/kg/hr infusion
• Hydralazine 0.2-0.4 mg/kg
• Diazoxide 1-3 mg/kg, max 150mg/kg
• Minoxidil 0.1-0.2 mg/kg

3. Metabolic acidosis
- if severe i.e. pH < 7.25 treat with NaHCO3 but in anuric child need DIALYSIS...

4. Electrolytes imbalance

(a) Hyperkalemia
- Resonium 1g/kg PO or per rectal
- Nebulised salbutamol 2.5-10mg/kg/ dose
- IV NaHCO3 1-2 mmol/kg
- Glucose & insulin – glucose 0.5mg/kg, insulin 0.1 U/kg
- IV Ca gluconate 0.5 ml/kg, 10% solution

Hyperkalemia on ECG

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Tall peak T waves</td>
</tr>
<tr>
<td>2.</td>
<td>Prolonged PR interval</td>
</tr>
<tr>
<td>3.</td>
<td>Widened QRS complex</td>
</tr>
<tr>
<td>4.</td>
<td>Flattened P wave</td>
</tr>
<tr>
<td>5.</td>
<td>Sine waves QRS complex merge with peaked T waves</td>
</tr>
<tr>
<td>6.</td>
<td>VF or asystole</td>
</tr>
</tbody>
</table>

K > 6
K > 8
K > 7
K > 9
K > 6-7
K > very high

(b) Hyponatraemia
- if secondary to fluid overload, treatment is fluid removal
- if due to sodium depletion, replace it

(c) Hypocalcaemia & hyperphosphataemia
- symptomatic: treat with IV calcium gluconate 10%
- asymptomatic: oral phosphate binders eg calcium carbonate or clacium acetate is given to correct the hyperphosphataemia first.
PART 7: NEONATAL RESUSCITATION

What risk factors are associated with the need for neonatal resuscitation?

<table>
<thead>
<tr>
<th>Antepartum Factors</th>
<th>Intrapartum Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal diabetes</td>
<td>Emergency cesarean section</td>
</tr>
<tr>
<td>Pregnancy-induced hypertension</td>
<td>Forceps or vacuum-assisted delivery</td>
</tr>
<tr>
<td>Chronic hypertension</td>
<td>Breech or other abnormal presentation</td>
</tr>
<tr>
<td>Fetal anemia or isoimmunization</td>
<td>Premature labor</td>
</tr>
<tr>
<td>Previous fetal or neonatal death</td>
<td>Precipitous labor</td>
</tr>
<tr>
<td>Bleeding in 2md/3rd trimester</td>
<td>Chorioamnionitis</td>
</tr>
<tr>
<td>Maternal infection</td>
<td>Prolonged rupture of membranes</td>
</tr>
<tr>
<td>Maternal cardiac, renal, pulmonary, thyroid, or neurologic disease</td>
<td>(&gt;18 hours before delivery)</td>
</tr>
<tr>
<td>Polyhydramnios</td>
<td>Prolonged labor (&gt;24 hours)</td>
</tr>
<tr>
<td>Oligohydramnios</td>
<td>Prolonged second stage of labor (&gt;2 hours)</td>
</tr>
<tr>
<td></td>
<td>Macrosomia</td>
</tr>
<tr>
<td></td>
<td>Persistent fetal bradycardia</td>
</tr>
<tr>
<td></td>
<td>Non-reassuring fetal heart rate patterns</td>
</tr>
<tr>
<td></td>
<td>Use of general anesthesia</td>
</tr>
<tr>
<td></td>
<td>Uterine hyperstimulation</td>
</tr>
<tr>
<td></td>
<td>Narcotics administered to mother within 4 hours of delivery</td>
</tr>
<tr>
<td></td>
<td>Meconium-stained amniotic fluid</td>
</tr>
<tr>
<td></td>
<td>Prolapsed cord</td>
</tr>
<tr>
<td></td>
<td>Abruptio placenta</td>
</tr>
<tr>
<td></td>
<td>Placenta previa</td>
</tr>
<tr>
<td></td>
<td>Significant intrapartum bleeding</td>
</tr>
</tbody>
</table>

Antepartum Factors:
- Maternal diabetes
- Pregnancy-induced hypertension
- Chronic hypertension
- Fetal anemia or isoimmunization
- Previous fetal or neonatal death
- Bleeding in 2md/3rd trimester
- Maternal infection
- Maternal cardiac, renal, pulmonary, thyroid, or neurologic disease
- Polyhydramnios
- Oligohydramnios

Intrapartum Factors:
- Emergency cesarean section
- Forceps or vacuum-assisted delivery
- Breech or other abnormal presentation
- Premature labor
- Precipitous labor
- Chorioamnionitis
- Prolonged rupture of membranes
- (>18 hours before delivery)
- Prolonged labor (>24 hours)
- Prolonged second stage of labor (>2 hours)
- Macrosomia
- Persistent fetal bradycardia
- Non-reassuring fetal heart rate patterns
- Use of general anesthesia
- Uterine hyperstimulation
- Narcotics administered to mother within 4 hours of delivery
- Meconium-stained amniotic fluid
- Prolapsed cord
- Abruptio placenta
- Placenta previa
- Significant intrapartum bleeding
The compromised baby may exhibit 1 or more of the following clinical findings:

- Poor muscle tone
- Depression of respiratory drive
- Bradycardia
- Low blood pressure
- Tachypnea
- Cyanosis

D.D: Infection, hypoglycemia, narcotics or general anesthetic agents given to the mother before birth.

**Initial Steps**

- Provide warmth.
- Position; clear airway (as necessary).
- Dry, stimulate, reposition

The baby should be positioned on the back or side, with the neck slightly extended in the "sniffing" position.

**Clear Airway**

- Secretions may be removed from the airway by wiping the nose and mouth with a towel or by suctioning with a bulb syringe or suction catheter.
- If the newborn has copious secretions coming from the mouth, turn the head to the side to allow secretions to collect in the cheek where they can more easily be removed.

**Caution:**

- When you suction, particularly when using a catheter, be careful not to suction vigorously or deeply.
- Stimulation of the posterior pharynx during the first few minutes after birth can produce a vagal response, causing severe bradycardia or apnea.
- Brief, gentle suctioning with a bulb syringe usually is adequate to remove secretions. Vigorous stimulation can cause serious injury.
- Shake baby → Brain Damage
- Slap baby → Bruise
- Squeeze rib cage → Fractures, Pneumothorax
- Dilate anal sphincter → Sphincter tear
- Hot/Cold Compress → Burn / Hypothermia

1. Oxygen mask held close to the baby’s face to give close to 100% oxygen
2. Using a flow-2 inflating bag to deliver free-flow oxygen. Hold the mask close to the face, but not so tight that pressure builds up.
3. Oxygen delivered by tubing held in cupped hand over baby’s face
Positive Pressure Ventilation

1. Self inflating Bag
2. Flow-inflating bag
3. T-Piece resuscitator

What ventilation rate should you provide during positive-pressure ventilation?

Breaths should be delivered at a rate of 40 to 60 breaths per minute.

Caution:
- Too large - may cause eye damage and will not seal well.
- Too small - will not cover the mouth and nose and may occlude the nose.

<table>
<thead>
<tr>
<th>Conditions</th>
<th>Actions</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Inadequate seal</td>
<td>- Reapply mask to face and lift the jaw forward.</td>
</tr>
<tr>
<td>2. Blocked airway</td>
<td>- Reposition the head.</td>
</tr>
<tr>
<td></td>
<td>- Check for secretions; suction if present.</td>
</tr>
<tr>
<td></td>
<td>- Ventilate with the newborn’s mouth slight open.</td>
</tr>
<tr>
<td>3. Not enough pressure</td>
<td>- Increase pressure until there is a movement of the chest.</td>
</tr>
<tr>
<td></td>
<td>- Consider endotracheal intubation.</td>
</tr>
</tbody>
</table>

Note: If you still are unable to obtain physiologic improvement and adequate chest movement after going through this sequence, endotracheal intubation and positive-pressure ventilation through the endotracheal tube are usually required.

Chest Compression
- If thumbs/fingers removed at each compression
  - Lose time relocating
  - Lose control of depth of compression
  - Compression of wrong areas
- Avoid giving compressions and ventilation simultaneously
- “One-And-Two-And-Three-And-Breathe-And”
- 30 Breaths : 90 Compressions
- After 30 seconds, Stop and feel pulse at base of umbilical cord
- 1) If pulse above 60
  - Stop chest compressions
  - Continue PPV (40-60 breaths/min)
- 2) If pulse below 60
  - Insert orogastric tube
  - Insert endotracheal tube
**Major Steps Oropharyngeal Tube Insertion**

Measuring the correct distance for inserting an orophageal tube

**Endotracheal Intubation**

- Preterm Trachea = 3cm
- Term Trachea = 5-6cm
- At 15cm end of tracheal tube should be at infant’s lips

<table>
<thead>
<tr>
<th>Tube Size (mm)</th>
<th>Weight (grams)</th>
<th>Gestational Age (wks)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.5</td>
<td>Below 1000</td>
<td>Above 28</td>
</tr>
<tr>
<td>3.0</td>
<td>1000 – 2000</td>
<td>28 – 34</td>
</tr>
<tr>
<td>3.5</td>
<td>2000 – 3000</td>
<td>34 – 38</td>
</tr>
<tr>
<td>4.0</td>
<td>&gt;3000</td>
<td>&gt;38</td>
</tr>
</tbody>
</table>

**Instruments**

1. Laryngoscope Blade
   - Preterm = No. 0
   - Term = No. 1
2. Check Light
3. Prepare suction equipment
4. Prepare device for PPV
5. Connect O2 source
6. Stethoscope
7. Prepare stabilizer

<table>
<thead>
<tr>
<th>Weight (Kg)</th>
<th>Depth of Insertion (cm from upper lip)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>3</td>
<td>9</td>
</tr>
<tr>
<td>4</td>
<td>10</td>
</tr>
</tbody>
</table>

- **Signs of endotracheal tube in the esophagus instead of trachea**
  - Poor response to intubation (continued cyanosis, bradycardia)
  - CO2 detector fail
  - No audible breath sounds
  - Air heard in stomach
  - Gastric distension
  - No mist in tube
  - Poor chest movement

- **Signs of tube being inserted in right main bronchus**
  - Infant’s HR and color show no improvement
  - Breath sounds heard over right side only
  - Breath sounds heard louder over right side of chest

**Complications**

- Hypoxia
- Bradypnea/Apnea
- Pneumothorax
- Contusions / Lacerations
- Obstructed endotracheal tube
- Infection
**Umbilical Vein Catheterization**
Most quickly accessible vein in newborn
1) Clean cord with antiseptic solution
2) Place loose tie around umbilical base
3) Prefill 3.5-5F umbilical catheter with NS
4) Close stopcock to prevent fluid loss and air entry
5) Cut the cord below the clamp placed at birth (1-2 cm from skin line)
6) Be sure to point the catheter upwards

**Umbilical Vein**
- Inject Epinephrine and 0.5ml of Normal Saline
- Epinephrine is only used when HR below 60bpm and after 30 secs of PPV and 30 secs of cardiac compression
- Recommended dose: 0.1 – 0.3ml/kg of 1 : 10000 solution (INTRAVASCULARLY)
- Recommended rate of administration: As fast as possible.
- Check baby’s HR for 30 secs
- If still less than 60bpm, repeat epinephrine every 3 – 5 mins.

N.B: Babies who are pale, delayed capillary fill, weak pulse = Circulatory Insufficiency

**If persistent HR less than 60 and persistent cyanosis:**
   a) Congenital Airway Malformation
   b) Lung problems (Pneumothorax, Diaphragmatic hernia)
   c) Congenital Heart Disease

**Meconium Blockage of Airway**
- Meconium or mucus in the pharynx or trachea
- Choanal Atresia
- Pharyngeal airway malformation
- Laryngeal web

**Impaired Lung Function**
- Pneumothorax
- Congenital Pleural effusions
- Congenital diaphragmatic hernia
- Pulmonary Hypoplasia
- Extreme immaturity
- Congenital Pneumonia

**Pneumothorax**
- In a large pneumothorax – block blood flow in lung → Respiratory distress, cyanosis, bradycardia.
- Breath sounds diminished in the side of pneumothorax.
- Confirm with CXR
- Screen by Transillumination technique.
- Loss of breath sounds in the left side due to endotracheal tube being too far.
  Treatment: Placing percutaneous catheter, needle or chest tube into pleural space
**Congenital Diaphragmatic Hernia**

- Entering of viscera into thoracic cavity
- Usually detected by ultrasound before birth
- If not, baby will have a completely unanticipated respiratory distress
- Persistent respiratory distress
- Flat appearing/ Scaphoid abdomen
- Breath sounds diminished in the side of hernia
- Persistent pulmonary hypertension
- Persistent cyanosis
- If PPV were to be given to the baby, some of the air given will be passed into the stomach or intestines → Decreasing the ability of the lungs to expand
- PPV delivered to the unexpanded lung may cause pneumothorax
- Babies with known congenital diaphragmatic hernia should not receive prolonged PPV with mask
- Should intubate trachea and insert large bore orogastric tube to empty stomach contents
- Consult Paediatric Surgery

**Postresuscitation Managements**

- All babies should have HR > 100bpm and pink centrally
- Close monitoring vital signs(HR, BP, RR, Temp)
- Monitor O2 sat and ABG
- Hcrit, blood glucose
- CXR

**Pulmonary Hypertension**

- Maintain Oxygenation and ventilation
- Consider tertiary therapies such as nitric oxide inhalation or extracorporeal membrane oxygenation(ECMO)

**Pneumonia and Lung Complications**

- A/w pulmonary hypertension
- Begin parenteral antibiotics
- Early pneumonia

**Metabolic Acidosis**

- Sodium bicarbonate
- N.B: Sodium bicarbonate is very caustic and hypertonic → given into large vein
- Usual dose: 2mEq/kg/dose 4.2% solution
- Never give through endotracheal tube

**Other Post Resuscitation Problems**

- Hypotension
- Seizures / Apnea
- Hypoglycemia
- Feeding problems (Ileus, UGIB, NEC)
- Temperature Management
- Fluid Management (SIADH, Renal Agenesis, ATN)