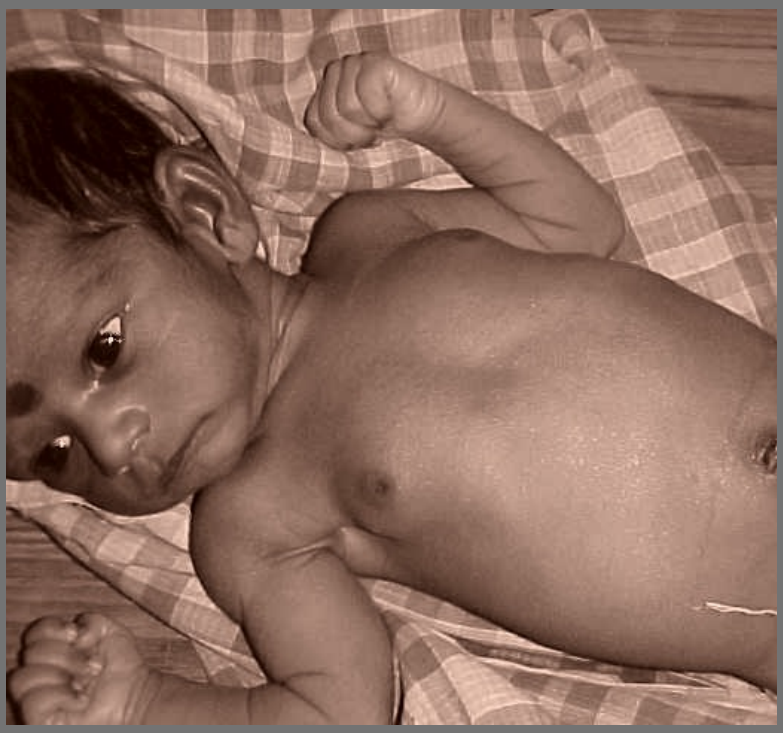
Clinical Pediatrics



CLINICAL PEDIATRICS

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Introduction

Children are a unique population and not just little adults

Pediatric population consists of:

1. Embryo: the product of conception upto 9 weeks of gestation.
2. Fetus: product of conception which is not completely expelled or extracted from mother irrespective of gestational period.
3. Infant: 0-365 days; “Infant” is derived from the Latin word, “infans,” meaning “unable to speak.”
    i. Early neonate: 0-7 days of post natal life.}
    ii. Late neonate: 8-28 days
    iii. Post neonate or infant: 29-365 days
4. Toddler : 1-3 years
5. Pre school or under 5: 3-5 years
6. Late childhood: 6 to 10 years
7. **Preadolescence**: The period of childhood just before the onset of puberty, often designated as between the ages of 10 and 12 in girls and 11 and 13
8. **Adolescence**: WHO defines adolescents as individuals in the 10-19-year age group and “youth” as the 15-24- year age group. These two overlapping age groups are combined in the group “young people”, covering the age range 10-24 years

Perinatal period:

- The perinatal period is defined as commencing at 28 completed weeks of gestation (>1000 gm) and ends seven completed days after birth.
- The extended perinatal period commences at 22 completed weeks (>500 grams) of gestation and ends at 7 completed days after birth.

Early neonatal period:

- The health of the NB during this period depends on events in pregnancy, child birth and interventions during and soon after birth. Maximum mortality in paediatrics occurs during this period.

Late neonatal period:

- The health of late neonate is dependant not only the effects of birthing process but also the exposure to infections, feeding practices and rearing practices.

Infancy:

- 1-12 mo; this is the age of maximum growth in terms velocity; child gains 3 times the birth weight; ½ the length; head circumference increase by 1/3 by 1 year.

Under 5 children:

1. 15% of population
2. Period of growth and development
3. Prone for:
    a. Malnutrition
    b. vaccine preventable diseases
    c. Nephrotic syndrome
    d. Febrile fits
    e. More deaths due to malnutrition, ari, pneumonias and tuberculosis, measles, pertusis and other VPDs

Adolescence:

- 20% of population
- Characterized by a rapid rate of growth and development
- Period of sexual maturation: puberty and menarche
- Prone for sexual behaviour and its consequences like RTI, pregnancy etc
- Prone for obescity
- Prone for aberrant behaviours like drug abuse, drinking, smoking,delinquency and violence

PEDIATRIC HISTORY

1. **Patient Profile:**
    1. Name:
        i. Name is important for identity
        ii. Name sometives gives a clue about the backround of the child
        iii. Names like Mannankatti, Kuppusamy and Pitchandi indicate that he is a precious child as all previous siblings died; it may also indicate that some hereditary disorder or Rh incompatibility could be the cause for such deaths.
        iv. Names like Salammal and and Pothum ponnu indicate that there are too many female siblings and the possibility of being neglected in nutririon and illness.
    2. Sex of the child:

i. There are sex specific diseases like :
    1. Fragile X syndrome in male
    2. Rett syndrome in female
    3. X-linked recessive disorders manifest in male child
ii. A mild CHD in a female child will later produce severe manifestation during her pregnancy

3. Birth order:
    i. This indicates that no of children dependant on the family
    ii. Congenital hypertropic pyloric stenosis is more prevalent in first born male child.
4. Age:
    i. The health problems in paediatrics are mostly age dependant;
    ii. Preterm succumbs to infections more easily than a full term baby
    iii. Edema and oliguria in 2 year old child could be nephrotic syndrome while the same featurtes in 10 year old may be due to acute nephritis.
    iv. Between 1 ½ to 3 years the following diseases are more common:
        1. Nephrotic syndrome
        2. Kwashiorkor
        3. Febrile seizures
        4. ITP
        5. Spasmodic croup
        6. Acute childhood hemiplegia

2. Informant: usually the mother is the informant who gives a fairly reliable history. We need more care while taking history in situations like:
    i. Illiterate parent
    ii. Medicolegal problem
    iii. Munchuhasan syndrome by proxy
    iv. Battered child syndrome
    v. Parental disharmony etc
3. Presenting complaints:

   The **Chief Complaint** or **Presenting Complaint** (PC), is the wors of mother describing the symptom about which she is more concerned. Here no medical terms are used and reproduced what actually the mother complained about thye child's illness.

   Eg. A 5 year old child brought by his mother from ariyur with fairly reliable history with the following chief complaints:

Fever	since 7 days
Cough and cold	since 5 days
Occasional vomiting	since 3 days
Head ache	1 day.

3. History of Presenting Illness (HPI):

This is the narration of entire aspects of illness for which the child has been brought to hospital. It includes probing questions and eliciting other positive symptoms which the mother might have omitted and eliciting some negative histories which may help in differential diagnosis.

Eg:

1. The fever started acutely and it was moderately high grade from day one. The fevere is intermittant in nature and not associated with chills. On day 4 and 5 the fever has got reduced in its intensity
2. Ther is nasal stuffiness and running nose; it is muco purulant in nature and no stinged with blood;
3. Cough is moderate in intensity; it is dry and hacking; more in nights; not associared with woop; no post tussive syncope or vomiting; (no need to mention about expectration as children often swallow sputum)
4. Vomiting is occasional; not projectile; not bilious; not blood stained; comes after intense cough.
5. Headache mis mild to moderate in intensity; mostly frontal; relived by analgesics; less felt during sleep.
6. Ther is pain in throat while swllowing; the nostrils are dry and itching with occasional sneezing.
7. The appetite is reduced
8. The child does not sleep adequately.
9. No history suggestive of aspiration of foreign body by the child
10. No history suggestive of GERD
11. No history sugeestive of allergy, bronchial asthma
12. There was no history of convulsions or unconscious states leading aspiration pneumonia.

4. Socio economic status:

Kuppusamy scale (modified)

(A) Education	**Score**
1. Profession or Honours	7
2. Graduate or post graduate	6
3. Intermediate or post high school diploma	5
4. High school certificate	4
5. Middle school certificate	3
6. Primary school certificate	2
7. Illiterate	1

(B) Occupation	**Score**
1. Profession	10
2. Semi-Profession	6
3. Clerical, Shop-owner, Farmer	5
4. Skilled worker	4
5. Semi-skilled worker	3
6. Unskilled worker	2

7. Unemployed	1

(C) Family income per month(in Rs)- **Modified for convenience**

Income	**Score**
> 20,000	12
10 to 20,000	10
7 to 10 000	6
5 to 7000	4
3 to 5000	3
1 to 3000	2
< 1000	1

Total Score	**Socioeconomic class**
26-29 Upper	(I)
16-25 Upper Middle	(II)
11-15 Middle Lower middle	(III)
5-10 Lower Upper lower	(IV)
<5 Lower	(V)

Importance of socio economic status and percapita income:

1. Infant mortality is more
2. Frequent illnesses due to malnutrition, Vit.A and Zinc deficiency, overcrowding, lack of personnel hygiene etc

5. Maternal literacy:

Mother's educational status and employment are important factors for regularity in immunization, utilization of health interventions, nutrition of the child and caring the child during illness.

6. Occupation:

Occupation has a bearing on childhood illness in some situations.

1. Brucellosis is possible if the family is rearing cattle's and selling milk.
2. If the father is dealing with used automobile batteries lead poisoning is a possibility.

7. Immunization:

State whether child has received vaccines up to the age; a fully immunized child is one who receives 0 opv, BCG, 3 doses of DPT, OPV, and measles vaccines before first birthday for "6 killer diseases".

State whether child received state programs like:

I. Immunizations as per age
II. Vit A prophylaxis (9, 18, 24, 30, 36, 42, 48, 54,and 60 months)
III. FST small
IV. Deworming
V. nutrition supplementation from Anganwadi centres (3-5 years)

VI. Noon meal (>5 years)

8. Nutrional history: state whether child received:
    1. Colestrum feeding initiated within ½ hour after birth. And no prelacteal feedings like ass milk, sugar water, cow milk etc are given soon after birth.
    2. Excusive breastfeeding up to 6 months
    3. Weaning or supplementary feeding after 6 months
    4. Calculate calory and protein intake and deficits for the age and weight
9. Family history: state:
    1. No of siblings
    2. Birth order of the child
    3. Exposure to TB
    4. Any communicable disease in the family
    5. Any hereditable disease in the family
    6. Parental disharmony and conflicts
    7. Loss of a parent
    8. Drug abuse & Alcoholism among parent
    9. Joint or nuclear family
10. Developmental history:

    Five Domains of development:

    1. **Motor:**
        a. **Gross motor:** using large groups of muscles to sit, stand, walk, run, etc., keeping balance, and changing positions.
        b. **Fine motor:** using hands to be able to eat, draw, dress, play, write, and do many other things.
    2. **Language:** It refers to speaking, using body language and gestures, communicating, and understanding what others say and also reading and writing
    3. **Cognitive:** Thinking skills: including learning, understanding, problem-solving, reasoning, and remembering.
    4. **Adaptive:** Adaptive skills refer to the skills used for daily living, such as dressing, eating, dressing, undressing and using toilet independently. It also includes avoiding dangers from any sources.
    5. **Social:** Interacting with others, having relationships with family, friends, and teachers, cooperating, and responding to the feelings of others.
11. State important mile stones:
    1. Motor:
        a. 5 months: head control; rolls over
        b. 8 mo: sits with support
        c. 10 mo: stands with support
        d. 15 mo: walks
        e. 2 yrs: climbs up stairs

f. 3 yr: stands on one foot
g. 4 yr: hops
h. 5 yr: walks in straight line back and forth

2. Fine motor:
    a. 3 ½ mo: persistant grasp reflex
    b. 5 mo: holds rattle
    c. 7 mo: holds object in each hand
    d. 10 mo: Pincer grasp
    e. 15 mo: puts objects in and out of a container
    f. 20mo: removes socks
    g. 2 yr: stacks 5 blocks
    h. 3 yr: stacks 8 blocks; draws a straight line
    i. 4 yr: stacks 10 blocks; draws circle
    j. 4 ½ yr: draws a square
    k. 5 yr: draw a cross
3. Speech:
    a. 5-6 mo: babbles
    b. 8-9 mo: da, ba
    c. 11 mo: dada, mamma
    d. 2 yrs: two word sentence
    e. 3 yrs: 3 word sentence
    f. 4 yrs: understands prepositions
    g. 5 yrs: use syntax in short sentances
4. Social :
    a. 2-3 mo: recognizes mother
    b. 6 mo: shows likes and dislikes
    c. 9 mo: less dependent on mother
    d. 12 mo: comes when called
    e. 15 mo: feeds self
    f. 18 mo: tells mother about wetting
    g. 2 yr: imitates others
    h. 3 yr: knows one's name
    i. 4 yr: differentiates shorter from longer line
    j. 4 ½ yr: count numbers
    k. 5 yr: identify colours; domestic role play; toilet control
    l. 5 ½ yr: remembers birth day and address
5. Behavior:

a. 3-4 mo: social smile
b. 1 yr: hard to console
c. 2-3 yrs: Temper tantrum
d. 4-5: group play

6. State wether there is any behaviour or developmental disorders:
    a. Neonatal seizures; Febrile seizures; Epilepsy
    b. Breath holding spell
    c. Thum sucking
    d. Pica
    e. Temper tantrums
    f. Hyper activity
    g. Short attention span
    h. Bed wetting after 5 years

11. Antenatal history:
    i. Is mother registerd by village health nurse or private practioners
    j. Did mother receive:
        i. TT two doses to prevent neonatal tetanus
        ii. Iron and Folic acid to prevent LBW, preterm labour and neural tube defects.
        iii. Nutrition supplementation from Anganwadi center.
        iv. Nutrition in Noon meal centers
    k. Did mother have exposure to:
        i. X radiation
        ii. Drugs like hydantoin, vit A
        iii. Smoking
        iv. Alcohol
    l. Any history suggestive of congenital infections?
        i. Pet animal like cat in the household: Toxoplasmosis
        ii. Fever with rash and lymphadenopathy: Rubella; CMV
        iii. Genital ulcer: Syphilis; Herpes
        iv. Genital discharge: PID; Gonorrhea; moniliasis
        v. Weheter mother was tested for GBS in the vaginal canal
    m. Any infection in the mother that may lead to LBW:
        i. UTI
        ii. Dental carries
        iii. RTI

12. Intra natal history:
    n. Place of birth

o. Birth attendant

p. Nature of delivery:

i. Spontaneous or induced

ii. Natural/forceps/vaccum/LSCS

13. Postnatal history:

a. Wether child cried immediately after birth and turned pink.

b. Meconium aspiration

c. APGAR at I,V and X minutes

d. Nature of Resussciation if any: suction, bag and mask, ET intubation; CPAP; ventilator

e. Duration O2 administration

f. Medications

g. H/o intensive care

14. Anthropometry and nutritional asessment: Refer WHO charts and specify interms of Z score or Standard deviation

1. Weight actual and expected:

i. Weight doubles at 5 months(3x2=6 kg)

ii. Tribles at 1 year (3x3=9kg)

iii. Quadruples at 2 years (3x4=12 kg)

iv. Formula: in Kgs

1–6 yr	Age (yr) × 2 + 8
7–12 yr	$\frac{\text{Age(yrs)x7-5}}{2}$

2. IAP classification of malnutrition:

Nutritional Grade	Percentage of standard weight for age
Normal	> 80%
Grade 1	71 – 80%
Grade 2	61 – 70%
Grade 3	51 – 60%
Grade 4	Less than 50%

3. Height actual and expected:

Height gain pattern: Formula: Age (yr) × 6 + 77

At birth: 50 cm

1year: + 25 cm= 75 cm

2 year: + 12.5 cm= 87.5 cm

3 year: + 6 cm= 93.5 cm

4 year: + 6 cm= 100cm

8 year: 125 cm

12 year: 150 cm

4. OFC: occipito frontal circumference- actual and expected:
5. MAC: Mid arm circumference- actual and expected
6. Upper segment to lower segment ratio:

The **lower body segment** is defined as the length from the symphysis pubis to the floor, and the **upper body segment** is the height minus the lower body segment.

I. The ratio of upper body segment divided by lower body segment (U/L ratio) equals approximately:

a. 1.7 at birth,

b. 1.3 at 3 yr of age, and

c. 1.0 after 7 yr of age.

II. Higher upper segment is characteristic of short-limb dwarfism or bone disorders, such as rickets; Highr lower segment is seen in Marfan syndrome

HEAD CIRCUMFERENCE (HC) -

Measurement of HC -

By the crossed tape technique- the occipitofrontal diameter is measured.

Importance of HC -

When the HC is less than the third percentile for that age & sex, it is called `microcephaly' when the HC is more than 97th percentile; it is called `macrocephaly'

Increase in HC:

At Birth 34 cm

First 3 months: 2 cm increase per month= 40 cm at 3 mo

Next 3 months: 1 cm increase per month= 43 cm at 6 mo

Next 6 months: 0.5 cm increase per month= 46 cm at 12 mo

2^{nd} year: + 2 cm= 48 cm

3^{rd} year: + 1 cm= 49 cm

CHEST CIRCUMFERENCE (CC)

Measurement of CC

Crossed tape technique at the level of nipples anteriorly & inferior angle of scapula posteriorly.

At birth: HC is > the CC by about 2 cms.

At 6-9 months: HC is equal to CC

1 year: CC exceeds HC: **This transition is delayed in malnutrition.**

MID ARM CIRCUMFERENCE (MAC) -

Measurement of MAC -

It is taken at the midpoint (between tip of the acromian process & olecranon process of ulna) while the arm is hanging by the side.

Importance of MAC -

It is used as an age independent criterion for detecting malnutrition as it remains almost constant between 16-17 cm for about 5 yrs (from 1 yr to 6 yrs),It is because of gradual replacement of the fat of infancy by muscle mass. When less than 13.8 cms it indicates malnutrition & when less than 12.5 cms it indicates severe malnutrition.

Primary Dentition:

	Mandibular	Maxillary
Central incisors	5–7 mo	6–8 mo
Lateral incisors	7–10 mo	8–11 mo
Cuspids (canines)	16–20 mo	16–20 mo
First molars	10–16 mo	10–16 mo
Second molars	20–30 mo	20–30 mo

PYSICAL EXAMINATION:

VITAL SIGNS

1. **Temperature:** axillary, rectal or oral
2. **Pulse: Quiet child,** Rate, rhytm, volume, Chatarecter (collapsing; bigeminis etc) palpability in all peripheral areas, radio femoral delay and pulse temperature dissociation.
3. **Respiration: quiet child,** rate, rhthm, nature (abdomial, thoracic), paradoxical respiration, working of axcessory muscles, dyspnea, tachypnea, grunting, stridor, depth (normal, shalow or deep) symmetrical or asymmetrical and chest retraction.
4. **Blood Pressure: quiet child,** cuff complete encircling, covering 2/3 of upper arm, palapatory followed by ascultatory, muffling point as diastolic pressure. Measure in both arms and thighs if coarctation is suspected.

Anthropometry:

5. **Height: use** stadiometer; state actual and expected height for the age;
6. **Body proportion:**
7. **Weight:** make o correction of weighing scale; Wt actual and expected, BMI
8. **Chest circumference:** measure along nipple lines; compare with HC; HC grossly more than CC is macrocephly and hydrocephalus; CC smaller than a normal HC indicates malnutrition.

GENERAL APPEARANCE

State wether child is:

1. Conscious, alert and active; or acute
2. Chronically ill looking, toxic, lethargic or having altered sensorium.
3. Quiet or irritable
4. State of hydration
5. Well or mal nourished
6. Normotonic, Floppy, spastic or opisthotonic
7. Normal or anemic
8. Polycythemic
9. Cyanosed
10. Jaundiced: lemion yellow or greenish yellow
11. Having Edema

Look for:

a. Dysmorphic features
b. Neuro Cutaneous markers:
    i. Nevi
    ii. café-au-lait spots
    iii. Neurofibroma
    iv. White leafy macule
    v. Shagreen patch
c. Haemtological conditions:
    i. Purpura
    ii. Petchiae
    iii. Echymosis
d. Infective:
    i. Skin rashes
    ii. Scabies
    iii. Impetigo
    iv. Moluscum
    v. Ulcers

Head and Face:

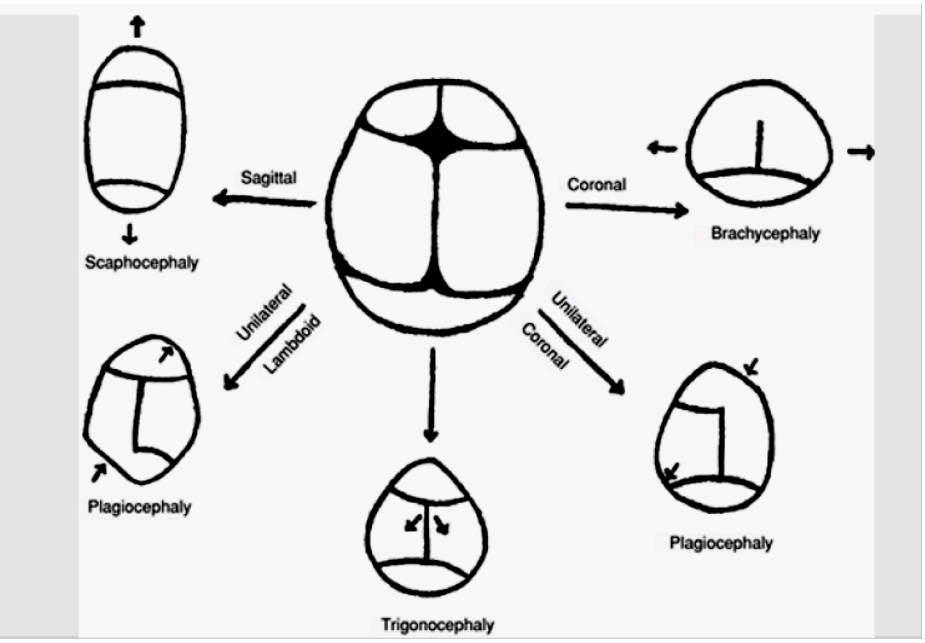
a. **Head Circumference: occipito frontal;** actual and expected and state as normal, micro or macrocephaly;
b. **AF**: state condition of AF and sutures in infants; Eg. AF admits 2 finger tips; soft and pulsations felt; sutures are palpable, not fused (prematurely fused, widely separated and overriding sutures are abnormal). Look for PF and 3rd fontanel.
c. **Shape**: abnormal shape occurs in Crouzon, Apert, Carpenter, Chotzen, and Pfeiffer syndromes.
    a. Scaphocephaly - Early fusion of the sagittal suture

b. Anterior plagiocephaly - Early fusion of 1 coronal suture

c. Brachycephaly - Early bilateral coronal suture fusion

d. Posterior plagiocephaly - Early closure of 1 lambdoid suture

e. Trigonocephaly - Early fusion of the metopic suture



d. Facial dysmorphism:

FEATURE	CONDITIONS
Hypertelorism	DiGeorge syndrome; Crouzon syndrome; Mucoploysachharidoses; Wardenburg; Cri du chart etc
Hypotelorism	craniostenosis; Trisomy 13; microcephaly etc
Mangoloid slant:	Down and Noonan; Prader willi
Antmangoloid slant:	Treacher-Collins syndrome; Apert; Cerebral gigantism
Low set ears:	Down syndrome; Turner syndrome; Beckwith-Wiedemann syndrome; Potter syndrome; Rubinstein-Taybi syndrome; Smith-Lemli-Opitz syndrome; Treacher Collins syndrome; Trisomy 13; Trisomy 18
Upturned nose	William syndrome; Fetal alcohol syndrome Corenelia de lange syndrome
Beaking of nose	Crouzan syndrome; Rubinstein –Taybi
Downturned mouth	Cri du chart; Prader willi
Syntrichosis	Corenelia de lange syndrome
Saddle nose	Congenital syphilis; Chr 7 p deletion
Long filtrum	Williams; Digeorge

e. Face appearence:
    a. Grotesque facies: mucopolysacharidosis
    b. Protruding toung: cretinism; Down
    c. Mask like: bilateral facial nerve palsy (eg. Mobius syndrome)
    d. Moon facies: Cushing and steroid therapy

Eyes:

a. Cataract : Rubella
b. Exoptholmos : hyperthyroididsm
c. Blue sclera: osteogenesis imperfecta
d. Corneal clouding: Hurler
e. Hazy cornea: Glaucoma
f. Conjunctival telangiectasia: Ataxia Telangiectasia

Ears:

a. low set: Turner
b. Large: fragile X
c. Small: Down

Nose:

a. Abnormal shape: beaked in Crouzan
b. Bifid nose: Pai syndrome
c. Bulbous nose: Trisomy 13
d. Depressed nasal bridge: Down, cong. syphilis
e. Nasal discharge: Rhinitis; Forien body

Mouth and Throat:

a. Macroglossia:
    a. Hurler
    b. Cretinism
    c. Beckwith-Wiedemann syndrome
    d. Lingual thyroid
    e. Gargoylism
    f. Ganglioside storage disease type I
b. High arched palate: Marfan
c. Cleft palate: Trecher colin; Pierre Robin
d. Pegged teeth: Ectodermal dysplasia
e. Tonsil and adenoid hyperplasia and inflammation: acute and chronic tonsillitis and adenoiditis.

Neck:

a. Webbing: Turner and Noonan
b. Short neck: Turner; Kliffel-Feil

c. Goitre: Hypothyroidism; Iodin deficiency; pubertal

d. Stiff neck: Meningitis

LYMPH NODES

1. In addition to the lymph nodes in the neck, palpate inguinal, epitrochlear, supraclavicular, axillary, and posterior occipital nodes.
2. Normally, inguinal nodes may be up to 1 cm in diameter: the others are nonpalpable or less than 5 mm.
3. **Generalized adenopathy** (enlargement of >2 noncontiguous node regions) is caused by systemic disease and is often accompanied by abnormal physical findings in other systems.
4. In contrast, **regional adenopathy** is most frequently the result of infection in the involved node and/or its drainage area.
5. A firm, fixed node should always raise the question of malignancy (Hodjkin and Lymphoma), regardless of the presence or absence of systemic symptoms or other abnormal physical findings.

COMMON CAUSES OF LYMPHADENOPATHY

INFANT	CHILD	ADOLESCENT
Syphilis	Viral infection	Viral infection
Toxoplasmosis	EBV	EBV
CMV	CMV	CMV
HIV	HIV	HIV
Tuberculosis	Toxoplasmosis	Toxoplasmosis
		Syphilis
RARE CAUSES		
Chagas disease (congenital)	Serum sickness	Serum sickness
Congenital leukemia	SLE, JRA	SLE, JRA
Congenital tuberculosis	Leukemia/lymphoma	Leukemia/lymphoma/Hodgkin disease
Reticuloendotheliosis	Tuberculosis	Lymphoproliferative disease
Lymphoproliferative disease	Measles	Tuberculosis
Metabolic storage disease	Sarcoidosis	Histoplasmosis
Histiocytic disorders	Fungal infection	Sarcoidosis
	Chronic granulomatous disease	Drug reaction

INFANT	CHILD	ADOLESCENT
	Sinus histiocytosis	Castleman disease
	Drug reaction	

CHEST:

1. Observe the chest for shape and symmetry.
2. The chest wall is almost round in infancy and in children with obstructive lung disease. Respirations are predominantly abdominal until about 6 years of age, when they become thoracic.
3. Note suprasternal, intercostal, and subcostal retractions, which are signs of increased respiratory work.
4. Edema of the chest wall occurs in children with superior vena cava obstruction.
5. Asymmetry of expansion occurs with diaphragmatic paralysis, pneumothorax.
6. Broad chest widely placed nipples: Turner syndrome
7. Barrel shape in cgronic obstructive airway disese; Ricketty rosary (Swelling at the costochondral junctions) in Rickets; scorbutic rosary in Vit.C deficiency;
8. Congenital chest wall deformities:
    1. Pectus Excavatum (sunken chest)
    2. Pectus Carinatum (and a protuberant or “pigeon chest”)

BREASTS:

1. Neonatal breast enlargement- secondary to increase endogenous steroids at the end of pregnancy. May express milk ("witches' milk").
2. Supernumerary breasts and nipples- very common and located along the "milk line" from the axilla to the symphis pubis
3. Premature Thelarche- isolated breast development without signs and symptoms of other secondary sexual traits such as estrogen effects, osseous development, acne, etc. Common between the ages of 2-5 and usually not progressive.
4. Gynecomastia- defined as the presence of mammary tissue in males.
    a. a. Approximately 2/3 of males will have breast tissue development during puberty
    b. Etiology:
        1. Familial
        2. Exogenous estrogens
        3. Exogenous steroids
        4. Klinefelter's syndrome
        5. Peutz Jegher Syndrome
        6. Pituitary tumors
        7. In adults associated with chronic liver disease, Marijuana use, other drugs
        8. INH, phenothiazines, Valium, Ketoconazole, street drugs

5. **Genitals:**
    a. **Look for:**
        1. Hydrocoel
        2. Hernias
        3. Undescendant testis
        4. Epi/hypospedias
        5. Long penis in Fragile X syndrome
        6. Underdeveloped genitals in Turner, Down, Lawrence Moon Biedel etc

Examination of relevant systems:

1. Follow Ossler's sequence:
    a. Inspection,
    b. Palpation,
    c. Percussion and
    d. Auscultation.

16. Investigations:
17. Diagnosis:
18. Differential diagnosis
19. Management
20. Complications
21. Prevention

GROWTH PEARLS

WEIGHT, HEIGHT, AND HEAD CIRCUMFERENCE: 5TH- 50TH-95TH PERCENTILES

Boys

Age*	**Height (cm)**	**Weight (kg)**	**FOC (cm)**
0 mo	46-50-54	2.5-3.5-4.3	32-36-39
0.5 mo	49-53-57	3.0-4.0-4.9	34-37-40
1.5 mo	53-57-61	3.8-4.9-6.0	36-39-42
3.5 mo	58-62-67	5.2-6.4-7.8	39-42-44
6.5 mo	64-68-73	6.7-8.2-9.9	42-44-46
9.5 mo	68-72-77	7.9-9.5-11.4	43-45-48

1 yr	72-76-81	8.8-10.5-12.6	44-46-48
2 yr†	82-88-94	10.7-12.7-15.3	46-49-51
3 yr	89-95-102	12.0-14.3-17.4	47-50-52
4 yr	96-103-110	13.6-16.3-20.3	—
5 yr	101-109-117	15.2-18.5-23.5	—
10 yr	128-139-150	24.9-32.1-46.1	—

ENERAL C L I N I C AL INFORMATION

Girls

Age*	**Height (cm)**	**Weight (kg)**	**FOC (cm)**
0 mo	46-49-54	2.5-3.4-4.2	32-35-38
0.5 mo	48-52-56	2.9-3.8-4.6	34-36-39
1.5 mo	51-55-59	3.5-4.5-5.5	36-38-41
3.5 mo	56-60-65	4.7-5.9-7.1	38-40-43
6.5 mo	62-66-70	6.1-7.5-9.0	41-43-45
9.5 mo	66-71-75	7.2-8.7-10.4	42-44-46
1 yr	69-74-79	8.1-9.7-11.6	43-45-47
2 yr†	80-86-92	10.3-12.1-14.7	45-48-50
3 yr	88-94-101	11.6-13.9-17.2	46-49-51
4 yr	94-101-108	13.1-15.9-20.4	—
5 yr	100-108-116	14.7-18.0-23.8	—
10 yr	127-138-150	24.8-33.1-48.2	—

Weight

- Average birth weight: 3.2 kg (girls); 3.6 kg (boys)
- Regain birthweight by 7 to 14 days (7–10 days for term infants; 10–14 days for preterm infants)
- Doubles in 4 mo, triples in 12 mo, quadruples in 24 mo

Weight (Rate of Gain)

- 0–3 mo → 20–30 g/d
- 3 mo–6 mo → 20 g/d
- 6 mo–1 yr → 10 g/d or 1 lb/mo
- 2 yr–puberty → 0.5 lb/mo or 2 kg/yr*

(Abnormal prepubertal velocity = <1 kg/yr wt gain)

Height

- Average birth length, 50 cm
- Doubles in 3 to 4 years; triples by 13 years

• Infant growth rate, 0.8 to 1.1 cm/wk

• Often grow in 8-wk spurts separated by periods of slow growth or stasis (~18 d)

• Reach half of adult height by 2 to 2.5 years

• First 6 mo: Growth rate influenced by intrauterine environment

• Male growth spurt during Tanner 4–5; female during Tanner 3–4

Height (Rate of Gain)

• Rule of thumb: 10-4-3-3-2 (inches gained per year until 5 years)

• Gain average of 10 inches in fi rst year of life, 4 inches in second year, 3 inches in third year, 3 inches in fourth year, and 2 inches in fi fth and each subsequent year until puberty

• Abnormal prepubertal height velocity <2 in/yr ht gain

Head Circumference (FOC)

• Average birth FOC: Girls, 35 cm; boys, 36 cm

• Usually 1 to 2 cm greater than chest circumference at birth

• Most head growth complete by 4 years

• Brain weight doubles by 4 to 6 months and triples by 1 year (similar to overall weight)

FOC (Rate of Gain)

• 0–3 mo → 2 cm/mo

• 3–6 mo → 1 cm/mo

• 6–12 mo → 0.5 cm/mo

• 12–24 mo → 2 cm total

Fontanelle

• The posterior fontanelle closes by age 4 mo.

• The anterior fontanelle is smaller by age 6 mo and is closed by age 9 to 18 mo (workup if open at 18 mo).

Sutures

• Usually all closed by age 12 to 24 mo, ossified by 8 years, and, completely fused by early adulthood

PREMATURITY

• Catch-up: FOC by 18 mo, weight by 24 mo, height by 40 mo (correct for gestational age until these age limits when plotting)

• Exception: VLBW (very low-birth weight) infants: Girls catch up by 20 years, but boys remain shorter and lighter than control subjects

TEMPERATURE AND WEIGHT CONVERSION

$°F = (°C \times 9/5) + 32$

$°C = (°F - 32) \times 5/9$

Kg = lb/2.2

Pounds = kg × 2.2

Grams = lb × 454

BODY SURFACE AREA

Mosteller's Formula

$$BSA(m^2) = \sqrt{\frac{height(cm) \times weight(kg)}{3,600}}$$

A child with 30 kg weight will have 1 suare meter body surface area.

Nutritional anthropometry:

This is a valuable index of assessment of nutritional status. 2/3 of children with PEM does not present with clinical signs but are diagnosed by anthropometry.

1. **Weight for age:**

   Most sensitive method when recorded serially. A decrease in weight gain / loss in weight can be seen within 1 month.

2. **Height for age:**

   Compares the child's height with the expected height for the age.

3. **Weight for height:**

   This compares a child's weight with the expected weight of the same height. It is useful for differentiating between acute and chronic malnutrition.

   Acute malnutrition:

   - Child is wasted i.e. weight for age and height is low but height for age is normal.

   Chronic malnutrition:

   - Child is stunted i.e. weight for age is low and height for age is low.

4. **Mid upper arm circumference (MUAC):**

   Normal MUAC for a child between 1-5 years of age is greater than 13.5 cm. If the MUAC is 12.5-13.5, the child has mild to moderate malnutrition and if it is less than 12.5 cm it is suggestive of severe malnutrition. This is useful for screening a large number of children but less useful in long term growth monitoring.

5. **Chest/ Head circumference**

   Chest circumference = Head circumference at one year of age.

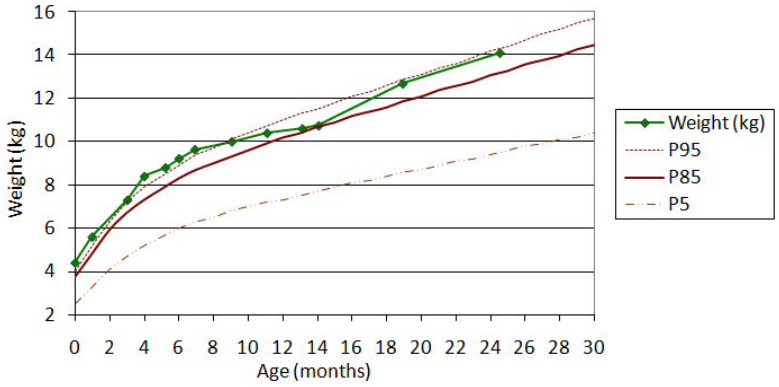
   Chest circumference > Head circumference after 2 years of age.

   In PEM, chest circumference is less than Head circumference even after 2 years of age.

6. **Skin fold thickness:**

It is an indication of the availability of caloric stores in the form of subcutaneous fat. Sites for measurement are triceps and subscapular region.

7. Body mass index: BMI is calculated as weight in Kg/height in M^2; E.g. a 6 year old girl with BMI of 21 is overweight whereas 16 year old girl with BMI 21 is just above the 50^{th} percentile
8. **The most important measurement for malnutrition is the growth curve:** weight for age is plotted as percentiles curves and growth is monitored over a period of time; it is also called road to health chart



Age Independent Criteria

1. Weight for Height
2. **MID ARM CIRCUMFERENCE (MAC) -**
    a. **Measurement of MAC -**
    b. It is taken at the midpoint (between tip of the acromian process & olecranon process of ulna) while the arm is hanging by the side.
    c. **Importance of MAC -**
    d. It is used as an age independent criterion for detecting malnutrition as it remains almost constant between 16- 17 cm for about 5 yrs (from lyr to 6 yrs),It is because of gradual replacement of the fat of infancy by muscle mass. When less than 13.8 cms it indicates malnutrition & when less than 12.5 cms it indicates severe malnutrition.
3. **Quack stick -**
    a. It is a rod with 2 sets of markings. One indicates the height & the other shows the expected MAC of a normal child for the corresponding height. The MAC of the child being examined is obtained & the stick is placed behind the standing child. If the height of the child is more than the height expected for the measured MAC, the child is considered to be malnourished.
4. **Shakir's tape for MAC -**
    a. It is a plastic tape with coloured zones -
    b. Green (>13.5 cms), corresponding to the MAC for normal Yellow (12.5 to 13.5 cms) borderline
    c. Red (<12.5 cms) wasted arms.
5. **Bangle test -**

a. A bangle with an inner diameter of 4 cms is slipped up the arm of the child. If it crosses the elbow, the child is considered malnourished. It is a less sensitive method as the width of the arm also depends on the thickness of the bones besides the muscle mass.

6. **SKIN FOLD THICKNESS (SFT) -**

a. **Measurement of SFT -**

b. Over triceps muscle or the subscapular region by using Herpenden's calipers.

c. **Importance -**

d. The SFT varies according to age & nutritional status of the patient. Normally it is 10 mm or more. SFT < 6 mm indicates severe malnutrition.

Nutritive value of common food:

S.No	Food	Measure	Protein	K.Calories	Remarks
1	Cow's milk 1 glass	200 ml	6	120	
2	Cooked rice 150 cc	1 cup	4	175	
3	Cooked dhal	1 tsp	0.5	10	Deficient in metionine
4	Egg	1	6	80	
5	Fish	1 oz-1 piece	6	80	
6	Mutton	1 oz- 8 pieces	6	50	
7	Bread	1 slice	2	70	
8	Dosai	1	2	70	
9	Chapathi	1	2	70	
10	Puri	2	2	70	
11	Idli	1	2	50	
12	Vada/bonda	1	1	50	
13	Upma	1 cup 150 cc	6	250	
14	Sugar	1 tsp	-	20	
15	Jaggery	1 tsp	-	20	
16	Ghee/butter	1 tsp	-	36	
17	Meshed potato	1 tsp	-	40	
18	Plantain	1	0.5	50	
19	Groundnut	10	1	20	
20	Pappadam	1	0.5	20	
21	Biscuit	1	0.5	20	
22	Coffee	1 cup	1.8	80	
23	Tea	1 cup	1	60	

24	Orange	1	0.5	50	
25	Oil /ghee	5 ml	-	40	

Nutritional advice for 1 year child:

Childs expected weight is 10 kg; actual weight is 7 kg; Gr II malnutrition (IAP)

Nutrition requirement is calculated for expected weight and the recommended diet is slowly stepped up to this level as per tolerance of the child

Calories: 10x100= 1000

Proteins: 2x10=20 gm

Diet plan for 1 year old child:

		Calories	Protein
Early Morning:	100 ml of milk	60	3
Breakfast:	2 idli	100	4
	Dhall 3 tsp	30	1.5
	Ghee/oil 1 tsp	40	-
Noon	2 biscuits	40	1
	100 ml milk	60	3
Lunch:	1 cup rice	170	4
	Dhall 3 tsp	30	1.5
	Ghee/oil 1 tsp	40	-
Evening:	1 egg or 1 vada	80	6
	100 ml milk	60	3
	1 banana	40`	-
Night:	1 cup rice	170	4
	Dhall 3 tsp	30	1.5
	Ghee/oil 2 tsp	80	-
Total		1030	32.5

Breast milk: Not to be included; considered as extra

EXAMINATION OF CARDIOVASCULAR SYSTEM

I. CHD due to expossure to maternal infections and drugs:

No	Antenatal exposure	Foetal condition
1	Cong. rubella infection:	PDA; P.S
2	CMV; Herpes; Coxackie; HIV	Cardiomyopathy
3	Ampetamine	VSD; PDA; ASD; TGA
4	Phenytoin	PS; AS; Coarctation; PDA
5	Trimethadione	TGA; TOF; Hypoplastic Lt. heart syndrome
6	Lithium	Epstein anomaly
7	Retinoic acid	Cono truncal anomaly
8	Valproic acid	ASD; VSD; AS; CoA;; P.atresia with intact VS
9	Progesteron and estrogen	VSD; TOF; TGA
10	Alcohol	VSD; PDA; ASD; TOF; Fetal alcohol syndrome
11	Smoking	IUGR
12	Diabetes	Cardiomyopathy; TGA; VSD; PDA
13	SLE	Cong. Heart block
14	CHD	1% increased to 15% CHD

II. CHD in infant condition and syndromes:

No	Infant condition	Heart disease
1	IUGR	Foetal alcohol syndrome; Rubella
2	Heavy for gestational age	TGA
3	**Carpenter syndrome**: Brachycephaly; craniosynostosis;	AR

	poly and syndactyly	
4	**Cornelia de lange**: Hirsutism; IUGR; microcephaly; up turned nose; down turned mouth; mental retardation	VSD
5	**Cri du chat**: Cat cry; microcephaly; downward slant of palpebrum	VSD; PDA; ASD
6	**Crouzon's disease**: Ptosis; shallow orbits; craniosynostosis; maxillary hypoplasia	PDA; COA
7	**Down syndrome**	Endocardial cushion defect; VSD
8	**Ellis van creveld**: short limbed dwarf; narrow thorax; polydactyly; neil hypoplasia; neonatal teeth	AR
9	**HaLt. Orum:** defective or absent thumb or radius	ASD; VSD
10	**KaRtagener**: situs inversus; sinusitis; otitis media; bronchiactasis; immotile celia; immotile sperm; AR	Dextrocardia
11	**Pierre Robin**: micrognathia; glossoptosis; cleft palate	COA; PDA; VSD; TOF
12	**Turner**: short female; broad chest; wide placed nipples; lymphedema of dorsum of hands and feet;	COA
13	**Noonan**: male turner; no chromosomal defect; AD	PS
14	**Weber**: facial hemangioma	Pulmonary AV fistula
15	**Osteogenesis imperfecta**: fragile bone; fractures; blue sclera; AR/AD	AR; MVP
16	**Rubella**: triad deafness; cataract; CHD; microcephaly	PDA; PS
17	**Rubinstein Taybi**: broad thumbs & toes; hypoplastic maxilla; beaked nose; short stature; MR ; sporadic	PDA; VSD; ASD
18	**Trecher Collin:** Cleft palate; defect in lower lids; malar hypoplasia; down slanting; ; malformed ear; Mutattion or AD	VSD; PDA; ASD
19	**Trisomy 13:** LBW; central facial hypoplasia; polydactyly; hemangioma; low set ears; genital defects;	VSD; PDA; dextrocardia
20	**Trisomy 18:** LBW; Microcephaly; micrognathia; rocker bottom feet; closed fist; overlapping index finger;	VSD; PDA; PS
21	**VATER: v**ertebral anomaly; **a**nal atresia; **T**EF; **r**enal dysplasia; radial dysplasia	VSD
22	**Williams:** MR; elfin facies; upturned nose; flat nasal bridge long fiLt.rum; wide mouth; sporadic	Supravalvular AS; PS

III. CVS: symptoms:

1. Chest pain:
    a. Coronary artery disease.
    b. Coronary vasospasm: not induced by exertion and may awaken the patient from sleep. Some patients report emotional stress as a trigger.
    c. Oesophageal disease
        i. Reflux oesophagitis
        ii. Oesophageal motor disorders- spasm
    d. Mitral valve prolapsed
    e. Pericarditis
    f. Pneumonia and pneumothorax
    g. Pleurisy
    h. Costochondritis
    i. Anxiety
2. **Dyspnoea**: Dyspnoea refers to subjective experience of breathing discomfort
    a. Exertional dyspnoea:
        i. Unaccustomed exercise
        ii. Accustomed exercise
    b. Dyspnoea at rest
    c. Orthopnoea is the sensation of breathlessness in the recumbent position, relieved by sitting or standing. caused by pulmonary congestion during recumbency
    d. Paroxysmal nocturnal dyspnoea (PND) is a sensation of shortness of breath that awakens the patient, often after 1 or 2 hours of sleep, and is usually relieved in the upright position. The failing left ventricle is suddenly unable to match the output of a more normally functioning right ventricle; this results in pulmonary congestion.
3. **Palpitation**: is unpleasant awareness of heartbeats; due to changes in heart rate or rhythm.
    a. Single "skips" suggest isolated premature extrasystoles,
    b. Regular palpitations characterize paroxysmal supraventricular or ventricular tachycardia.
    c. Paroxysmal atrial fibrillation is often described as beats irregular in both rhythm and strength,
    d. Sinus tachycardia is regular increase in heart rate.
    e. Syncope following an episode suggests sinus node dysfunction
4. **Syncope**: is a sudden transient loss of consciousness associated with loss of postural tone.
    a. **Non cardiac causes:**
        i. Micturition syncope
        ii. Defecation syncope
        iii. Swallow syncope
        iv. Cough syncope
        v. Orthostatic hypotension

vi. Drug-induced syncope: antihypertensive; nitrates; quinidine; antidepressants

b. **Cerebro vascular disease**:

i. Carotid sinus syncope

ii. Glossopharyngeal or trigeminal neuralgia with asystole: trigeminocardiac reflex

c. **Cardiac Causes:**

i. Reduced cardiac output

ii. Obstruction to left ventricular outflow: aortic stenosis and hypertrophic cardiomyopathy

iii. Obstruction to pulmonary flow: pulmonary stenosis, pulmonary hypertension, pulmonary embolism, Tetralogy of Fallot

iv. Pump failure: massive myocardial infarct Cardiac tamponade, Atrial myxoma, Aortic dissection

v. Arrhythmias:

1. Bradyarrhythmias

a. Second and third degree atrioventricular block

b. Ventricular asystole

c. Sick sinus syndrome

2. Tachyarrhythmia

a. Ventricular tachycardia

b. Supraventricular tachycardia

5. **Oedema** (hydropsy): refers to a discernible excess of interstitial fluid.

a. Pitting oedema gives way on palpation, leaving persistent impressions in the skin;

b. Brawny oedema offers resistance and leaves no impressions.

c. Causes:

i. Disorders associated with inappropriate renal sodium retention

1. Intrinsic renal disease

2. Acute glomerulonephritis

3. Acute renal failure

4. Chronic renal failure

ii. Drug-induced

1. Non steroidal anti-inflammatory agents

2. Insulin

3. Estrogens and oral contraceptives

4. Exogenous mineralocorticoids

iii. Venous occlusion and its Sequelae

1. Peripheral deep vein thrombosis

2. Post phlebitis venous insufficiency

3. Inferior vena caval occlusion

4. Superior vena caval occlusion

iv. Idiopathic capillary leak syndrome

v. Disorders associated with lymphedema

1. Milroy's disease: hereditary defect in lymphatics with oedema
2. Filariasis
3. Neoplastic obstruction of lymphatics
4. Surgical interruption of lymphatics

vi. Disorders associated with a high cardiac output

1. Arteriovenous fistula
2. Beriberi
3. Anemia
4. Thyrotoxicosis
5. Paget's disease

vii. Disorders in which the pathogenesis is unclear

1. Idiopathic oedema
2. Premenstrual syndrome
3. Toxaemia of pregnancy
4. Hypothyroidism

viii. Disorders with complex pathogenesis

ix. Congestive heart failure

x. Nephrotic syndrome

xi. Cirrhosis

6. **Claudication:** is a pain, cramp or sense of fatigue in a muscle group of the lower extremity related to sustained exercise and relieved promptly by a few minutes of rest; it is an indicator of arterial insufficiency of the leg.

a. Causes:

i. Atherosclerosis

ii. Arterial embolism,

iii. Buerger's disease: due to smoking; autoimmune to tobacco factors; thrombosis of arteries and veins.

iv. Takayasu's arteritis: pulseless disease; unknown inflammatory arterial stenosis or thrombosis; more in female

v. Arteriovenous fistula,

vi. Coarctation of the aorta,

vii. Leriche's syndrome: saddle thrombus at bifurcation of abd. Aorta with absent femorals, claudication, absent sperms etc.

4. INSPECTION

1. FACIES

A. Skin Color and Texture:

1. Malar flush: long-standing MS,
2. Butterfly rash across the nose in SLE.
3. Brick red colour of polycythemia (may cause hypertension, thrombosis, MI)
4. Bronze skin in hemochromatosis(Cardiomyopathy)
5. Brown + buccal pigmentation in Addison's disease (hypotension)
6. Flushing & telangiectasia in carcinoid syndrome (tricuspid & pulmonary valve disease)
7. Moon face in Cushing's disease (hypertension)
8. Coarseness & dryness in myxedema (bradycardia, heart failure)
9. Central cyanosis (right to left intra cardiac shunt or lung disease)
10. Pallor: anemia or shock

B. Eyes and Lids

1. Xanthelasma : hypercholesterolemia, Diabetes Mellitus
2. Lid edema : myxedema, nephrotic syndrome, SVC syndrome
3. Exophthalmos, lid retraction in thyrotoxicosis : A.F, high output failure
4. Corneal arcus in young people indicates severe hypercholesterolemia
5. Blue sclera in Marfan syndrome, Ehlers-Danlos syndrome : AR, MVP, ASD
6. Lenses : subluxation in Marfan syndrome - superior, homocystinuria- inferior
7. Pupils (Argyll Robertson sign) react to accommodation not to the light seen in neurosyphilis : AR, calcification in the ascending aorta

C. **Bony Developmental Abnormality**

1. Large head: Paget's disease→ High-out failure; defect in remodelling of bone
2. Acromegaly : hypertension, CHF
3. Marfan syndrome (with long narrow face, lens subluxation, long arm, arachnodactyly):AR, aortic dissection, MVP
4. Williams syndrome (small elf-like forehead, turned up nose, egg shaped teeth, low set ears): supravalvular aortic stenosis
5. Noonan syndrome (widely set eyes, web neck):PS

D. Hands

1. Tremor may indicate thyrotoxicosis (AF, CHF)
2. Clubbing of the fingers (cardiac cause: congenital heart disease, bacterial endocarditis)
3. Capillary pulsation (AR, thyrotoxicosis, pregnancy)
4. Splinter haemorrhage (bacterial endocarditis, acute glomerulonephritis)
5. Osler's nodes (0.5-1 cm painful reddish-brown subcutaneous papules occur on the tip of the fingers or toes, palm of the hand, planter aspect of the feet (bacterial endocarditis)
6. Arachnodactyly (long slender hand and fingers) Marfan syndrome

E. **Forehead sweating**: In CHF due to sympathetic overlay;

F. **Acanthosis Nigricans**: dark pigmentation over neck, axilla and groin creases→ type II diabetes;

2. BREATHING PATTERNS

1. Using accessory muscles of respiration? (Pulmonary oedema, asthma, COPD, fulminant pneumonia)
2. Breathlessness + wheezing (asthma, COPD, LV failure)
3. Stridor (indicating upper airway obstruction) life-threatening situation
4. Chyne-stokes respiration (CHF, strokes, over sedation, uraemia)

3. CYANOSIS

1. Cyanosis Is not apparent if Hb is < 5g/dl (central)
2. In CHD cyanosis is observed if R to L shunt and not improved by 100% of O2
3. Cyanosis becomes deeper on crying

FOUR TYPE OF CYANOSIS

1. **Central cyanosis** blue tongue, lips, and extremities with warm peripheries (CHD, lung disease as emphysema, pneumonia, ARDS, chronic bronchitis, sometimes CHF)
2. **Peripheral cyanosis** (result. from sluggish circulation in the peripheries) reduction in oxygenated Hb occur in capillaries(extremities are blue & cold) aetiologies: low CO, hypovolemic shock)
3. **Differential cyanosis** (lower limb cyanosed, upper limb pink) in CHD: PDA with revered shunt due to pulmonary hypertension
4. **Reversed differential cyanosis.** The cyanosis of the fingers exceeds that of the toes; seen in transposition of the great vessels (blood from RV ejected into the AO reaches the upper limbs and head, blood from LV ejected into PA reaches the lower limb via PDA)

5. PALPATION

1. **PULSE**: determine
    1. Rate
    2. Rhythm
    3. Character
    4. Symmetry : Examine both radial, carotid, femoral, tibial, and dorsalis pedis pulses

2. **Rate**

    At rest > 100/min (tachycardia) seen in anxiety, pain, CHF, hyperthyroidism, anemia, fever, medications

    Rate < 60/min (bradycardia) due to (medications, MI, hypothyroidism, hypothermia

3. **Rhythm** (regular or irregular indicating AF, frequent Premature AC's, Premature VC's)

4. **Character**

    1. **Collapsing pulse:**water hammer pulse; jerky pulse with full volume followed by sudden collapse (AR, PDA, A-V fistulas, pregnancy, Paget's disease, thyrotoxicosis, anemia)
    2. **Pulsus alternans:** regular rate, amplitude varies from beat to beat; seen in LVF
    3. **Pulses bisferiens:** two strong systolic peaks separated by a mid systolic dip seen in cardimyopathy, AS/AI
    4. **Anacrotic pulse:** slow rising pulse in A.S.

5. **Dicrotic pulse**: two systolic and diastolic peaks; sepsis, hypovolemic, cardiogenic shock

6. **Paradoxic pulse;** amplitude decreases with inspiration and increases during expiration; seen in cardiac tamponade, COPD, massive P.E.

7. **Pulse in COA**:

   1. Week femoral and radio femoral delay: COA
   2. Stronger Rt. brachial than left: pre Lt. subclavian COA
   3. Week Rt. brachial: aberrant Rt. subclavian distal to COA

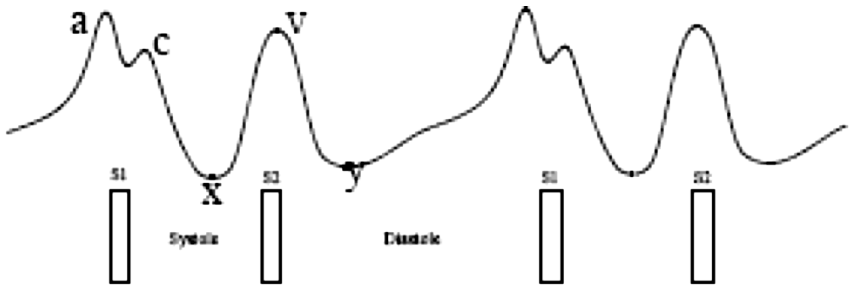
5. PERIPHERAL VEINS

JVP:

Differentiation of the jugular and carotid pulse wave

	Jugular	**Carotid**
1. Character	3 positive waves	1 wave
2. Effect of respiration	present	No effect
3. Venous compression	easily eliminate pulse wave	No effect
4. changing position	more prominent When recumbent, less prominent when sitting	No effect
5. Abdominal pressure	JV is more visible	No effect

Normal JVP:



The pulse seen in the jugular veins represents atrial pressure changes during the cardiac cycle

- 'a' wave: atrial contraction
- 'c'' wave: transmitted pulsation from the carotid artery or ventricular systole (not normally visible)
- 'v' wave : atrial filling
- 'x' descent: atrial relaxation
- 'y' descent: tricuspid valve opens with rapid emptying of the atrium and filling of RV

Abnormalities of Venous Wave

1. Giant "A" wave seen in RA contraction against an obstructed TV (TS, atresia, myxoma) high resistance to RA emptying (RVH, P.hypertension, PS, PE,)
2. Cannon "A" wave: (RA contracts against closed TV) seen in CHB.
3. Prominent "V" wave (V wave caused by RA filling against TV closure coincide with S2 and T wave on the ECG) seen in significant TR, VSD, ASD causing diastolic RA overloading
4. Kussmaul's sign: paradoxical rise in JVP with inspiration (constrictive pericarditis, severe RHF)

Chest:

Precardial bulge: Chronic Cardiomegaly;

Pectus excavatum: depressed sternum; no significance

Harrison's groove: poor lung compliance as seen in L to R shunt.

Palpatation:

1. APEX BEAT:

- Supine, sitting, and left lateral decubitus position.
- Normal apical impulse occurs during early systole
- Palpable at 4 th intercostal medial to midclavicular line in less than 7 yrs; intersection of left mid clavicular line and 5th intercostal space after 7 yrs;
- Apex beat > 2cm indicate LV enlargement.
- Double apical impulse caused by LVH and forceful Left Atrial contraction.

2. Left parasternal lift

Best appreciated by the distal palm or with the finger tip; Palpable anterior systolic movement sustained up to S2indicate RVH; Giant presystolic lift seen in Hypertrophic cardiomyopathy.

3. Abdomen

- Abdominal aorta (aneurysm)
- Liver (hepatomegaly, pulsatile liver)
- Ascites

6. **Auscultation**

1. AREAS TO AUSCULTATE:

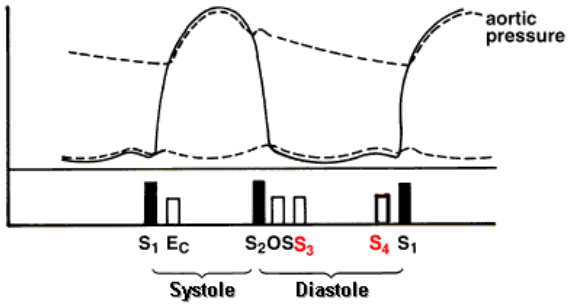
1. Apex (mitral area) murmur originated from the MV are best heard
2. Lower sternal edge (tricuspid area)
3. Lower left parasternal (4th intercostal space) murmur of AR is best heard
4. Upper left parasternal (pulmonary area, 2nd left intercostals space)
5. Upper right parasternal (aortic area, 2nd right intercostal space murmurs arising from aortic valve area best heard)
6. below the left clavicle: continuous murmur of PDA is best heard
7. Posterior chest for bruits caused by bronchial collaterals in case of coarctation of the aorta

8. Other areas: (abdominal aorta, renal arteries, carotid, femoral arteries for bruit)

2. Heart sounds:

Heart Sound	Occurs during	Associated with
S1	Isovolumetric contraction	Closure of mitral and tricuspid valves
S2	Isovolumetric relaxation	Closure of aortic and pulmonic valves
S3	Early ventricular filling	Normal in children; in adults, associated with ventricular dilation (e.g. ventricular systolic failure)
S4	Atrial contraction	Associated with stiff, low compliant ventricle (e.g., ventricular hypertrophy)

Timing of Heart sounds in cardiac cycle:



3. FIRST HEART SOUND (S1)

Produced by M&T valve closure

Best heard at the apex

Occurs just before the palpable upstroke of the carotid pulse

S1 is loudest when onset of ventricular systole finds mitral leaflets maximally recessed into LV cavity as in short PR, A.F. with short cycle length.

Factors influencing intensity of S1

1. PR interval: - short PR-loud S1 & long PR-soft S1
2. Mitral Valve Disease: MS typically causes loud S1 but when the valve becomes calcified and immobile, S1 intensity decreases. Soft S1 occurs also in AR due to premature closure of MV.
3. Intensity of S1 increase in (MS, TS, myxoma, short PR)
4. Intensity of S1 decrease in (fibrosis or calcification of MV, prolong PR, heart failure, MR, AR)
5. Variable intensity of S1 in A.F., CHF, VT

4. **SECOND HEART SOUND (S2)**

Closure of semi lunar valves AV & PV (other theory is that deceleration of a column of blood in the root of aorta & PA at termination of systole leads to sound producing vibration audible as S2).

Normally AV closes before PV. Inspiration splitting of S2 is due to delay in P2 due to:

1. Increase capacitance of pulmonary vascular bed
2. Increase-RV volume

Normally intensity of A2 exceeds that of P2.

A) Loudness of A2 or P2 is proportional to the respective pressures in Aorta or PA at onset of diastole, i.e., higher the pressure, louder the A2 or P2

A2 is louder with HYPERTENSION, dilated aorta.

P2 is louder with pulmonary HYPERTENSION, dilated PA

B) Decrease intensity of A2 or P2 is due to decrease pressure beyond the valve, stiff semilunar valves (A.S, P.S.), chest wall or lung deformity (emphysema)

Splitting S2:

1. A widely split and fixed S2 is found in conditions that prolong the RV ejection time or that shorten the LV ejection. Therefore, it is found in:

a. ASD or partial anomalous pulmonary venous return (PAPVR) (conditions in which the amount of blood ejected by the RV is increased; volume overload).

b. PS (the valve stenosis prolongs the RV ejection time; pressure overload).

c. RBBB (a delay in electrical activation of the RV) delays the completion of the RV ejection.

d. MR (a decreased forward output seen in this condition shortens the LV ejection time, making aortic closure occur earlier than normal).

e. An occasional normal child, including “prolonged hangout time” seen in children with dilated PA (a condition called idiopathic dilatation of the PA). In dilated PA, the increased capacity of the artery produces less recoil to close the pulmonary valve, which delays closure.

2. A narrowly split S2 is found in conditions in which the pulmonary valve closes early (e.g., pulmonary hypertension) or the aortic valve closure is delayed (e.g., AS). This is occasionally found in a normal child.

3. A single S2 is found in the following situations.

a. When only one semilunar valve is present (e.g., aortic or pulmonary atresia, persistent truncus arteriosus)

b. When the P2 is not audible (e.g., transposition of the great arteries [TGA], tetralogy of Fallot [TOF], severe PS)

c. When aortic closure is delayed (e.g., severe AS)

d. When the P2 occurs early (e.g., severe pulmonary hypertension)

e. In an occasional normal child

4. A paradoxically split S2 is found when the aortic closure (A2) follows the pulmonary closure (P2) and therefore is seen when the LV ejection is greatly delayed (e.g., severe AS, left bundle branch block [LBBB], sometimes Wolff-Parkinson-White [WPW] preexcitation).

5. THIRD HEART SOUND (S3)

1. Normal findings in younger patients
2. Due to passive diastolic filling of the ventricle (occurs 0.14 to 0.22 s after S2)
3. Best heard at the apex patient on the LLP
4. Causes of S3 include:
    1. Abnormal ventricular relaxation
    2. Ventricular failure
    3. Dilated and hypertrophic cardiomyopathy
    4. Severe TR, MR
    5. LV dyskinesia or aneurysm
    6. Hyperdynamic states (AV fistula, thyrotoxicosis)

6. **FOURTH HEART SOUND (S4)**

1. Due to vigorous atrial contraction to propel blood into a stiff ventricle (absent in AF)
2. Best heard at the apex, patient on LL decubitus
3. S4 is heard in:
    1. LVH
    2. Acute MI
    3. HOCM (hypertrophic obstructive cardiomyopaty), DCM(dilated cardiomyopathy)
    4. Severe AS, PS
4. S4 occurs just before the S1 (diagnosis with split S1: firm pressure by the diaphragm eliminates S4 but not split S1)

7. EJECTION CLICKS:

Three mechanisms:

1. Follows S1; occurs at the onset of ventricular ejection
2. Audible at the base
3. Due to stenosis of AV or PV (congenital bicuspid AV, congenital P.S.)
4. Dilated great artery (hypertension, p. hypertension, dilated PA, aortic aneurysm)
5. Increase flow states (ASD: pulmonic EC) (truncus arteriosus: aortic EC)

Mid to Late Systolic Clicks:

1. Commonly heard in MVP
2. Sharp high pitched sound
3. Manoeuvres that decrease LV volume move the click earlier.
4. Manoeuvres that increase LV volume move the click later.

Opening snap

1. Caused by the opening of the stenotic but pliable MV or TV (disappears in severe calcified MS or TS). Higher LA pressure leads to short S2-OS
2. Occurs 0.08 s after S2
3. Best heard between apex and left sternal border
4. DD: Split S2. However, having the patient stands help to differentiate the two. The S2 – OS internal widens, while split S2 does the change or narrow.

8. Murmurs

1. Grading of intensity of murmurs:

Grade:

Grade I - So faint and heard only with special effort

Grade II - Soft but readily detected

Grade III - Prominent but not loud

Grade IV - Loud usually with palpable thrill

Grade V - Very loud with thrill

Grade VI - Heard without stethoscope on the chest wall

2. High pitch sounds (use diaphragm of stethoscope)

S1, S2, murmurs of valvular regurgitation

OS, clicks, obstruction of semilunar valves (AV, PV)

Pericardial knock

3. Low pitch sound (use of bell of stethoscope)

S3, S4 obstruction of AV valve (MV, TV)

Classification of Murmurs: (Systolic, Diastolic and Continuous)

I. Systolic Murmurs (SM)

- Stenosis of semilunar valve causes delay in peaking of SM related to prolongation of ejection. The duration not the intensity is proportional to severity of obstruction.
- Commonly encountered clinic problem is the differentiation of AS Vs benign aortic sclerosis. With aortic sclerosis there should be no clinical, ECG, or radiological evidence of heart disease. The carotid upstroke is normal. The SM peaks early with normal S2.
- MR murmur is usually pansystolic, It can be late systolic in timing (suspect MVP, papillary muscle dysfunction). It can also be early systolic in acute severe MR with markedly increased LA pressure reducing late systolic LV-LA gradient.
- The SM of TR is best heard at LLSB or over xyphisternum and associated with large V-wave.
- The murmur of VSD parallels the pressure difference between the two ventricles. The murmur is typically pansystolic and associated with thrill. With significant pulmonary hypertension, the murmur duration shortens.

- SM heard in the back may be caused by Coarctation, aortic dissection, peripheral PA stenosis or pulmonary AV fistula.

II. Diastolic Murmurs

Early diastolic murmurs:

1. Aortic regurgitation: configuration of the murmur reflects volume and rate of regurgitation flow. Therefore, with chronic AR, the aortic diastolic pressure consistently exceeds the LV diastolic pressure and the murmur is heard throughout diastole. However, with acute AR, the LV diastolic pressure is very high (because the LV is not dilated and unprepared), so the murmur tends to be short.
2. Pulmonary regurgitation murmurs secondary to pulmonary hypertension (Graham-steel murmur) is high velocity blowing murmur that can last throughout diastole. While PR without elevation of PA pressure results in mid diastole murmur because the diastolic pressure exerted on PV is minimal in early diastole.

Mid diastole murmurs

1. Majority originates across mitral or tricuspid valves.
2. Murmur is easily appreciated with increase flow through the valves.
3. Duration of the murmur correlates with severity.
4. Mid diastolic murmurs occur with: (1) obstruction of AV valves (MS, TS, Austin flint) and (2) Increase flow across AV valves (functional obstruction), e.g., ASD, VSD.

Late diastolic murmur (pre systolic)

1. Represented by increased flow through obstructed mitral or tricuspid valves during atrial contraction
2. Can be heard (opposite to common belief) in MS or TS in patients with atrial contraction (AF)

III. Continuous Murmurs – due to

1. Aortopulmonary connection (PDA)
2. AV connection (AV fistula, anomalous origin of left coronary from PA)
3. Disturbance of flow in arteries or veins (mammary soufflé, cervical venous hum)

IV. **Manoeuvres to Differentiate Between Different Murmurs**

Valsalva:

During active strain phase most murmurs decrease in intensity except murmur of: 1) HOCM– typically gets louder and 2) MVP – longer and louder

Respiration:

Right sided sounds and murmurs get louder with inspiration (except for pulmonary ejection click)

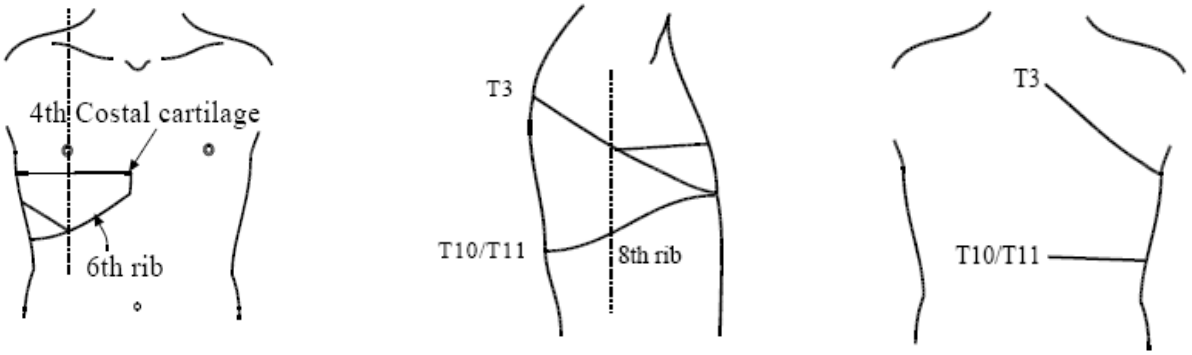
Handgrip:

By increasing BP (after load), augments murmur of MR, AR but does not affect murmur of AS and tends to decrease murmur of HOCM

EXAMINATION OF THE RESPIRATORY SYSTEM

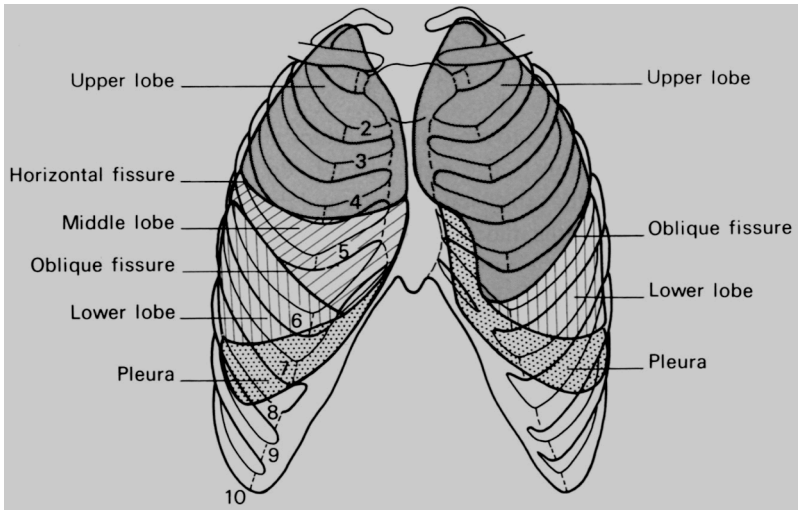
1. **General considerations:**
   - I. The patient must be properly undressed and gowned for this examination.
   - II. Ideally the patient should be sitting on the end of an exam table.
   - III. The examination room must be quiet to perform adequate percussion and auscultation.
   - IV. Try to visualize the underlying lobes of the lungs as you examine the patient.
   - V. Observe the patient for general signs of respiratory disease (finger clubbing, cyanosis, air hunger, etc.).
   - VI. The physical examination of the lung is referenced to the time frames of inspiration and expiration, just as the cardiac examination is referenced to systole and diastole.
   - VII. The major characteristic of the normal respiratory physical examination is symmetry. What is encountered on one side should be encountered on the other. What is perceived anteriorly should be perceived similarly when examining posteriorly.

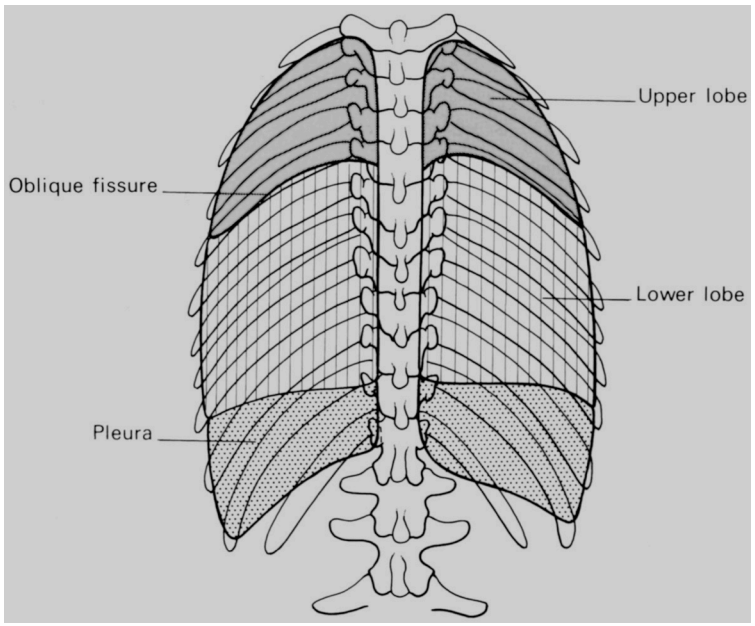
2. **Surface anatomy:**

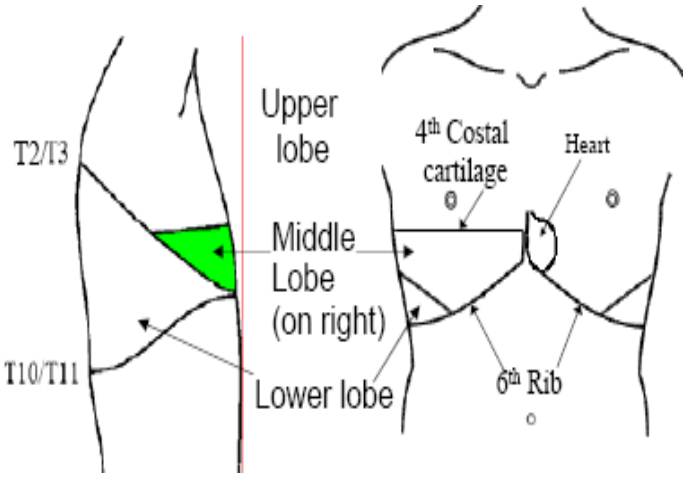


- The lower border of the lung (at rest) extends down to the 6th rib
- The oblique fissure is marked anteriorly by the point at which the midclavicular line crosses the sixth rib
- The horizontal fissure on the right is marked by the position of the 4th costal cartilage
- The oblique fissure curls upwards towards the 3rd thoracic vertebrae
- The horizontal fissure extends as far as the oblique fissure in the midaxillary position
- The lower border of the lung extends to the eight rib in the mid-axillary line
- Posteriorly the oblique fissure reaches up to the level of the 3rd thoracic vertebra
- The lower most border of the lung is marked by the 10th or 11th thoracic vertebra

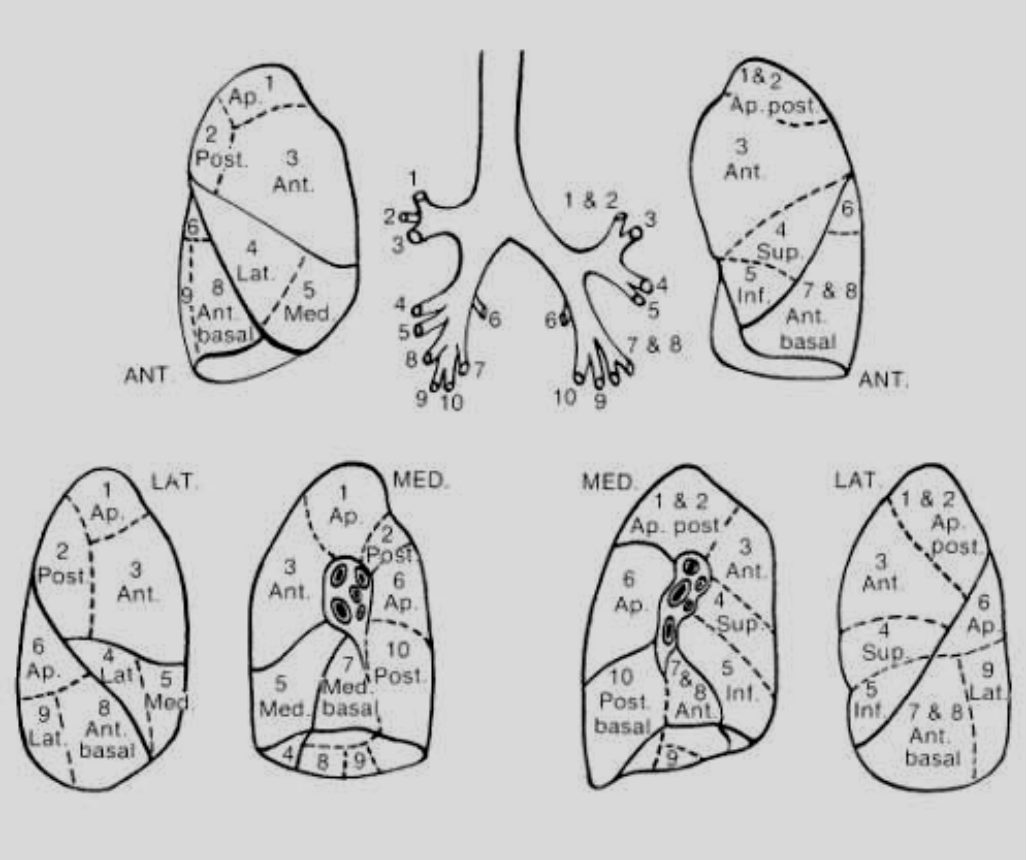
1. Lobes o the lung:







2. Bronchopulmonary segments:



3. Respiratory symptoms:

1. **Dyspnea**: is an uncomfortable abnormal awareness of breathing

   a. Grading:

Grade	**degree of dyspnoea**
0	No dyspnea except with strenuous exercise
1	Dyspnea when walking up an incline or hurrying on the level
2	Walks slower than most on the level, or stops after 15 minutes of walking on the level
3	Stops after a few minutes of walking on the level
4	Dyspnea with minimal activity such as getting dressed, too dyspneic to leave the house

   b. Cuses:

      i. Obstructive:

         1. Chronic obstructive pulmonary disease (COPD)

            a. Emphysema

b. Chronic bronchitis,

2. Bronchial asthma, (periods of dyspnea frequently alternate with periods of normal breathing.)

ii. Restrictive pulmonary disorders:

Reduced lung volume, either because of an alteration in lung parenchyma or because of a disease of the pleura, chest wall, or neuromuscular apparatus.

1. Intrinsic lung diseases or diseases of the lung parenchyma:

a. Interstitial lung disease

b. Pneumonitis

c. Fibrosis

d. Sarcoidosis

2. Extrinsic disorders or extraparenchymal diseases.

a. The chest wall-kyphoscoliosis

b. Pleura-pleurisy and effusion

c. Respiratory muscles- poliomyelitis; myasthenia gravis

2. Wheeze:

Is a high-pitched, musical, adventitious lung sound produced by airflow through an abnormally narrowed or compressed airway(s). A wheeze is synonymous with a high-pitched or sibilant rhonchus. Common causes of wheezing are:

1. Asthma is a heterogeneous syndrome characterized by variable, reversible airway obstruction and abnormally increased responsiveness (hyper reactivity) of the airways to various stimuli.
2. Bronchiolitis
3. Chronic obstructive pulmonary disease
4. Medication-induced bronchoconstriction
5. Pulmonary edema
6. Tracheobronchitis
7. Vocal cord dysfunction
8. Anaphylaxis

3**. Stridor**:

A high-pitched musical or "crowing" sound localized in the larynx or trachea, resulting from turbulent air flow in the upper airway. It is primarily inspiratory; Stridor may occur as a result of:

- Foreign bodies (e.g., aspirated peanut, aspirated food bolus),
- Tumour (e.g., laryngeal papillomatosis, squamous cell carcinoma of larynx, trachea or esophagus),
- Infections (e.g., epiglottitis, retropharyngeal abscess, croup),
- Subglottic stenosis (e.g., following prolonged intubation or congenital),
- Airway edema (e.g., following instrumentation of the airway intubation, drug side effect, allergic reaction),

- Subglottic hemangioma (rare),
- Vascular rings compressing the trachea
- Many thyroiditis such as Riedel's thyroiditis
- Vocal cord palsy
- Tracheomalacia or Tracheobronchomalacia (e.g., collapsed trachea)
- Congenital anomalies of the airway are present in 87% of all cases of stridor in infants and children.

4. **Cough**:
    a. Acute or chronic
    b. Dry or productive
    c. Pertussoid

    Many viruses and bacteria benefit evolutionarily by causing the host to cough, which helps to spread the disease to new hosts. Most of the time, coughing is caused by a respiratory tract infection but can be triggered by choking, smoking, air pollution, asthma, gastro esophageal reflux disease, post-nasal drip, chronic bronchitis, heart failure and medications such as ACE inhibitors
5. **Hemoptysis**:
    a. Coughing up bright red blood or blood clots: carcinoma of the lung, tuberculosis, pulmonary embolism
    b. Blood-streaked, purulent sputum : bronchitis, bronchiectasis, or pneumonia
    c. Blood-tinged, white, frothy sputum : congestive heart failure); or
    d. Foul-smelling, bloody sputum: anaerobic lung abscess
    e. Red sputum that contains no blood: Serratia marcescens pneumonia , in glass sanders with sputum discolored by iron oxide, and in ruptured hepatic amebic liver abscess with its "anchovy paste" sputum.
6. **Halitosis**, or **bad breath**:
    a. Is a term used to describe noticeably unpleasant odors exhaled in breathing – whether the smell is from an oral source due to bacteria or otherwise.
7. Smoking:
    a. Is the most important determinant of chronic obstructive lung disease. In cigarette smokers, the risk of developing COLD is 10 times that of non smokers,
8. Household smoke:
    a. Produce frequent ARI in children

4. General inspection:

1. **Respiratory distress:** this will be signified by an increased respiratory rate, nasal flaring and intercostals recession
2. Pink puffer has predominantly emphysema. They are thin, have an increased respiratory rate and breathe through pursed lips.
3. Blue bloaters however are cyanosed from hypoxia and oedematous as a result of Cor Pulmonale.
4. **Cushingoid:** a clue that the patient is a severe asthmatic on oral steroids.
5. Cachexic: This could indicate carcinoma of the lung or chronic disease such as COPD

6. One constricted pupils and ipsilateral wasting of the muscles of the hand due to an apical lung cancer **(Pancoast's syndrome)**

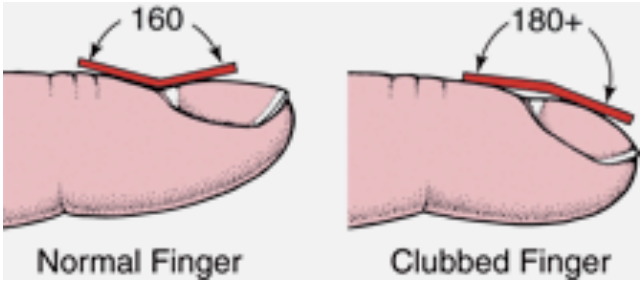
5. Nails and hands

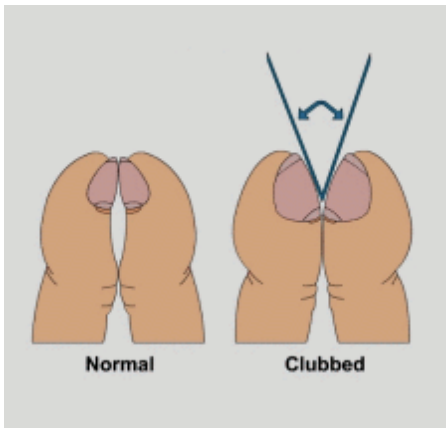
1. Nicotine staining
2. Clubbing:

   Clubbing develops in five steps:

   1. Fluctuation and softening of the nail bed (increased ballotability)
   2. Loss of the normal <165° angle (Lovibond angle) between the nailbed and the fold (cuticula)
   3. Increased convexity of the nail fold
   4. Thickening of the whole distal (end part of the) finger (resembling a drumstick)
   5. Shiny aspect and striation of the nail and skin

   Schamroth's test or **Schamroth's window test** (originally demonstrated by South African cardiologist Dr Leo Schamroth on himself) is a popular test for clubbing. When the distal phalanges (bones nearest the fingertips) of corresponding fingers of opposite hands are directly apposed (place fingernails of same finger on opposite hands against each other, nail to nail), a small diamond-shaped "window" is normally apparent between the nail beds. If this window is obliterated, the test is positive and clubbing is present.
3. **Hypertrophic pulmonary osteoarthropathy** (or **Bamberger-Marie disease**) is a medical condition combining clubbing and periostitis of the long bones of the upper and lower extremities. Distal expansion of the long bones as well as painful, swollen joints and synovial villous proliferation are often seen. The condition may be primary or secondary to diseases like lung cancer.
4. Peripheral cyanosis
5. Wasting of the intrinsic muscles of one hand
6. A fine finger tremor related ti use of beta-agonist bronchodilators.





7. **Asterixis**-this is a coarse tremor which looks like the flap associated with liver failure. It signifies CO2 retention, the other features of which are arm peripheries, a bounding pulse, Papilledema and headache.

6. The radial pulse

1. **Pulsus paradoxus**. A fall in the systolic blood pressure by more than 10mmhg on inspiration. It occurs in severe asthma, constrictive pericarditis and pericardial effusion. Take the blood pressure at the end of inspiration and expiration.

7. R**espiratory rate**. A normal rate is between 12-16/minute.

8. The Eyes:

Pancoast's syndrome is the association of a Horner's Syndrome with a cancer at the apex of the lung. Other features are related to the destruction of the brachial plexus and include arm pain and wasting of the intrinsic muscles of the hand. If the recurrent laryngeal nerve is also affected, a hoarse voice and bovine cough may also occur.

Horner's syndrome:

- **Miosis** (pupil constriction)
- **Enopthalmos** (sunken eye)
- **Ptosis**
- And ipsilateral **anhydrosis** (loss of sweating)

9. The Mouth

- **Central cyanosis.** Central cyanosis develops when there is at least 5g/dl unsaturated haemoglobin in the blood.
- Peripheral cyanosis: eg: Raynaud's Phenomenon

10. The Neck

Examination of the neck as part of the respiratory system focuses on the following three points:

1. JVP
2. Trachea
3. Cervical Lymphadenophaty

11. The Trachea

- Check that the tracheal deviation by placing the index and ring fingers either side of the trachea at the Sterna and using the middle fingers to feel the tracheal rings.
- Keeping the middle fingers in place over the tracheal rings, try to detect tracheal tug by asking the patient "could you please breathe in deeply". Tracheal tug is present if your middle finger moves upwards when the patient breathes. It signifies lung pathology.

12. **Cricosternal distance**.

- This is the distance between the cricoids cartilage and the sterna angle. If it is less than 3 finger breadths this indicates hyperinflation of the lungs.

13. Cervical Lymphadenopathy

- The lymph nodes of the neck should be examined from behind with the patient sitting upright.
- Start off by placing the fingers in the submental region . Work backwards and laterally, feeling the submandibular glands and finishing behind the ears where you may detect enlarged posterior auricular nodes.
- Then place the fingers just medial to the top of the sternocleidomastoid muscle. Work down the medial border of the muscle, palpating the cervical lymph node chain. Finish in the supraclavicular fossae. An enlarged node in the

left supraclavicular fossa is known as **Virchow's Node** and its presence is called **Troissier's sign**. It is indicative of stomach cancer.

14. The Chest

1. **Inspection**
    a. A **barrel chest: chronic lung hyperinflation** (e.g. Asthma, COAD) it has an increased AP diameter and a tracheal tug is often also present.
    b. **Pectus carinatum** or **"pigeon chest":** the sternum is prominent; caused by **chronic childhood asthma** and **rickets**.
    c. **Harrison's sulcus: chronic childhood asthma** and **rickets;** groove deformity of the lower ribs at the point of attachment to the diaphragm.
    d. **Kyphosis** is an increased forwards spinal convexity
    e. **Scoliosis** is an increased lateral curvature of the spine

15. B**reathing pattern**:

1. **Cheyne-Stokes :** consists of cycles of increasing hyperventilation followed by apnoea; indicates a **brainstem lesion** (CVA or raised ICP) or an **increased lung to brain circulation time** (poor cardiac output).
2. **Biot respiration**: or cluster breathing, is also periodic in nature but does not have the crescendo–decrescendo pattern seen with Cheyne–Stokes respiration. It is clusters of irregular breaths that alternate with periods of apnea. This breathing pattern is seen in individuals with pontine lesions.
3. **Ataxic breathing**: is one of varying tidal volumes and rates. These individuals can frequently keep their rate more rhythmic if they try consciously. The abnormality is in the medullary chemoreceptor or the medullary respiratory control center.
4. **Paradoxical movement**: In normal persons, therefore, there is a coordinated movement of the chest wall and abdomen moving outward on inspiration and inward on expiration.
    a. Paralysis of the intercostals results from cervical spinal cord injury. In this group of patients on physical exam there is a paradoxical movement of the chest wall inward and the abdomen outward during inspiration
    b. Diaphragmatic paralysis can be diagnosed or suggested by an inward movement of the diaphragm during inspiration.
5. Check for **intercostals recession**, the indrawing of the intercostals spaces as the patient breathes in.
6. Also note whether **movement** of the chest wall is symmetrical. If it isn't, the pathology lies on the Side of restricted movement.

16. **Palpation**

1. Assess chest expansion by placing the palms of the hands symmetrically on either side of the chest wall with the thumbs pointing towards the midline. Ask the patient "Could you please take a deep breath in?" And feel whether the fingers move apart symmetrically. The thumbs should separate by at least 5cm.
2. **Tactile vocal fremitus**. This is performed by placing the ulnar borders of each hand in intercostals spaces on either side of the sternum at 3 levels. Ask the patient "would you mind saying ninety nine for me as this helps me

to assess how your lungs are working." Tactile vocal fremitus is affected by conditions in the way as the breath sounds are (see later).Therefore, you should expect :

- Vesicular breathing over areas of normal tactile vocal fremitus
- Decreased breath sounds over areas of decreased tactile vocal fremitus
- Bronchial breathing over areas of increased tactile vocal fremitus

17. Percussion

1. Percuss in both supraclavicular fossae, the clavicles and at 3-4 levels in either side of the anterior aspect of the chest.
2. Dullness over the liver extends as high as the sixth costal cartilage and over the heart. In emphysema, the liver may be pushed down by the hyperinflated lung and the area of cardiac dullness may be lost.
1. The following percussion notes may occur :
    a. **Resonant**-normal
    b. **Dull**-consolidated lung, collapsed lung or a lung abscess or neoplasm.
    c. **Stony dull**-pleural effusion
    d. **Hyper-resonant**-this can be generalized, when it signifies lung hyperinflation, or localized when the cause may be a pneumothorax or a large emphysematous bulla.

18. Auscultation:

1. It is customary to use the bell of the stethoscope when auscultating the supraclavicular fossa but the diaphragm elsewhere. If the patient is very thin, the bell may have to be used throughout.
2. When auscultating you are listening for both the **breath sounds** and for **added sounds**.

3. Breath sounds:

1. These can be vesicular**, decreased** or **bronchial**.
2. **Vesicular breath sounds** are heard over **normal** lung. There is no gap between the inspiratory and expiratory parts and expiratory part is shorter.
3. Localised decreased breath sounds occurs in the following conditions :
    i. Pneumothorax
    ii. Pleural effusion
    iii. Tumour
    iv. Collapse
    v. Pleural thickening
4. Generalised decreased breath sounds occurs in :
    i. Emphysema
    ii. Asthma
    iii. Muscular chest wall
    iv. Obesity
    v. Fibrotic lung disease

5. **Bronchial breathing**: there is a gap between the inspiratory and expiratory parts and both parts are of the same length. The quality of the sounds is "harsher" than vesicular breathing. Bronchial breathing is heard in the following situations :
    i. Consolidation
    ii. Abscess near the chest wall
    iii. Bronchial neoplasm (when the airway is still patent)
    iv. Dense fibrosis
    v. The top of a pleural effusion

4. Added sounds

1. **Wheezes:** usually occur in **expiratio**n and indicates narrowing of an airway. The smaller the airway, the higher the pitch of the wheeze. Thus **high pitched polyphonic wheeze** occurs in **asthma** and **COAD** where asthma is a component of the disease. On the other hand, a **low pitched monophonic wheeze** indicates narrowing of a single large airway due to **bronchial carcinoma**.
2. **Crepitations** are produced when airways "pop" open as the chest expands in inspiration. As a general rule, the smaller the airway, the finer the crepitation and the later it occurs in inspiration.
    vi. The following conditions cause fine, late inspiratory crepitations that affect both lung bases equally :
        1. Pulumonary oedema
        2. Fibrosis
    vii. The following conditions cause coarse crepitations that tend to be present thoughout inspiration
        1. COAD
        2. Bronchiectasis
    viii. Pneumonia causes a mixture of fine and coarse crepitations which tend to be localised to one lobe of the lung.

i. Pleural Rub

This is caused by the 2 inflamed layers of the pleural membrance rubbing against each other. If it present the patient may also have pleuritic chest pain at the same site. It can be caused by:

1. Pneumonia
2. Pulmonary embolus

Adventitious (extra) lung sounds	
Crackles	These are high pitched, discontinuous sounds similar to the sound produced by rubbing your hair between your fingers. (also known as **rales**)
Wheezes	Wheezes are continuous polyphonic sounds heard during expiration that reflect narrowing of intrathoracic airways.
Rhonchi	Rhonchi are low-pitched continuous snoring sounds heard in either inspiration or expiration associated with uncleared secretions in the airways

Stridor	Stridor is a wheezing sound most commonly encountered in extrathoracic airways and heard in inspiration. Stridor usually reflects extrathoracic airway obstruction but may be caused by a fixed obstruction anywhere in the tracheobronchial tree.
Pleural rubs	Pleural rubs may be heard in either respiratory phase and sound like the rubbing of leather.

4. Vocal resonance

Place the stethoscope in all the areas you auscultated previously and listen to see whether vocal resonance is **normal, decreased** or **increased**. Vocal resonance is the "speaking equivalent" of the breath sounds and can be used to confirm what you suspected on auscultation. Therefore you should expect:

1. Normal vocal resonance over areas of vesicular breathing
2. Decreased vocal resonance over areas decreased breath sounds
3. Increased vocal resonance over areas of bronchial breathing

All this information can be summarised in the following table, which should be learnt:

	Chest Movement	**Trachea**	**Percussion Note**	**Breath sounds**	**Added sounds**
Pleural Effusion	Decreased	Central or away	Stony dull	Decreased, Bronchial breathing At surface	None
Pneumothorax	Decreased	Central or away	Hyper-resonance	Decreased	None
Consolidation	Decreased	Central	Dull	Bronchial if airway Patent	Coarse Crepitation +- rub
Fibrosis	Decreased	Central	Normal	Decreased or Bronchial if severe	Fine late Inspiratory Crepitation
Collapse (blocked Bronchus)	Decreased	Toward lesion	Dull	Decreased	None

19. Examination of the Posterior Aspect of the Chest

1. Inspection

Look for:

a. kyphoscolisis
b. surgical scars
c. drains
d. Radiotherapy tattoos. Check for **intercostals recession a**nd whether **movement** of the chest wall is symmetrical.

2. Palpation

Assess chest expansion at 2 levels on the back of the chest. And feel whether the fingers move apart symmetrically. The thumbs should separate by at least 5cm.You may also palpate for changes in **tactile vocal fremitus**.

3. Auscultation

Listen for the **breath Sounds** and whether there are **added sounds**.

Also test for changes in **vocal resonance**.

20. Special tests

1. Peak flow monitoring

Peak flow meters are inexpensive, hand-held devices used to monitor pulmonary function in patients with asthma. The peak flow roughly correlates with the fev1.

1. Ask the patient to take a deep breath.
2. Then ask them to exhale **as fast as they can** through the peak flow meter.
3. Repeat the measurement 3 times and report the average.

2. Voice transmission tests

These tests are only used in special situations. This part of the physical exam has largely been replaced by the chest x-ray. All these tests become abnormal when the lungs become filled with fluid (referred to as **consolidation**).

3.Tactile fremitus

1. Ask the patient to say "ninety-nine" several times in a normal voice.
2. Palpate using the ball of your hand.
3. You should feel the vibrations transmitted through the airways to the lung.
4. Increased tactile fremitus suggests consolidation of the underlying lung tissues.

4. Bronchophony

1. Ask the patient to say "ninety-nine" several times in a normal voice.
2. Auscultate several symmetrical areas over each lung.
3. The sounds you hear should be muffled and indistinct. Louder, clearer sounds are called bronchophony.

5. Whispered pectoriloquy

1. Ask the patient to whisper "ninety-nine" several times.
2. Auscultate several symmetrical areas over each lung.
3. You should hear only faint sounds or nothing at all. If you hear the sounds clearly this is referred to as whispered pectoriloquy.

6. Egophony

1. Ask the patient to say "ee" continuously.
2. Auscultate several symmetrical areas over each lung.
3. You should hear a muffled "ee" sound. If you hear an "ay" sound this is referred to as "e -> a" or egophony.

7. Sucussion's splash:

A succussion splash describes the sound obtained by shaking an individual who has free fluid and air or gas in a hollow organ or body cavity. It is usually elicited to confirm intestinal or pyloric obstruction due to pyloric stenosis or gastric arcinoma, but may also be heard with hydropneumothorax, a large hiatal hernia, or over a normal stomach.

Pleural Fluid in absence of Pneumothorax is silent; In hydropneumothorax, chest contains air and fluid; When patient moves, splash sound is audible

Technique: Examiner auscultates lungs while patient moves or assistant grasps patient's shoulders and shakes.

NEUROLOGICAL HISTORY AND PHYSICAL EXAMINATION

"History tells you what it is, and the examination tells you where it is."

Steps in the neurologic examination

1. Higher functions
2. Cranial nerves (CNS)
3. Sensory system
4. Motor system
5. Cerebellum
6. extrapyramidal
7. Meninges
8. System survey

1. Examination of the Higher Functions

Gait: Gait is the attitude of a person in the upright position.

1. Hemiparetic gait

2. Gait:

The shoulder adducted; elbow flexed; forearm pronated, wrist and fingers flexed; circumducts the affected leg while walking.

3. Simple hemiplegia:

The lesion is of the contralateral upper motor neurons above the brainstem in the cortical, subcortical, or capsular region.

4. Crossed hemiplegia:

Cranial Nerve paralysis is ipsilateral and body paralysis is contralateral. CN paralysis is of the lower motor neuron type.

1. Weber syndrome:

Paralysis of CN III with crossed hemiplegia indicates a midbrain lesion

2. Millard-Gubler syndrome

Lesion of CN VII with crossed hemiplegia indicates a pontine lesion

3. Jackson syndrome:

CN XII paralysis with crossed hemiplegia indicates a lower medullary lesion -

2. Ataxic gait

- Patient spreads his legs apart to widen the base of support to compensate for the imbalance while standing or walking.
- The heel-to-toe or tandem uncovers subtle forms of ataxia.

Cause:

- Ataxia results from midline lesions of the cerebellum and may be isolated or associated with other cerebellar findings
- When the lesion is unilateral, the patient may veer to the side of the lesion.
- With bilateral cerebellar involvement, the patient may fall to either side.

3. Shuffling gait

- The individual takes short steps and practically not moving forward or making little progress.
- Festinating gait: short steps with a tendency to accelerate as he walks.
- Both types are seen in Parkinson disease and may be associated with other extrapyramidal signs.

4. High stepping gait:

- Steps as if climbing of stairs while walking on a level surface.
- Seen in chronic peripheral neuropathies and can be the result of bilateral foot drop.

5. Spastic or scissor gait

- The legs in adduction at the hip and the thighs rub against each other
- This is typically seen in cerebral diplegia, a form of cerebral palsy.

6. Antalgic gait

- Patient favours the affected painful (usually lower) extremity and walks, putting weight on the normal leg.
- The hand held over hip on the affected side is typical in patients with radicular pain.

7. Waddling gait:

- Dug like walking in myopathy due to weekness of gluteus muscles

SPEECH

1. Dysphonia or aphonia

- Impairment or inability to phonate.
- Causes:
    1. Inflammation of the larynx.
    2. Hypothyroidism, thickening of the vocal cords from amyloid deposits.
    3. Unilateral recurrent laryngeal nerve paralysis and lesions of the vagus nerve.

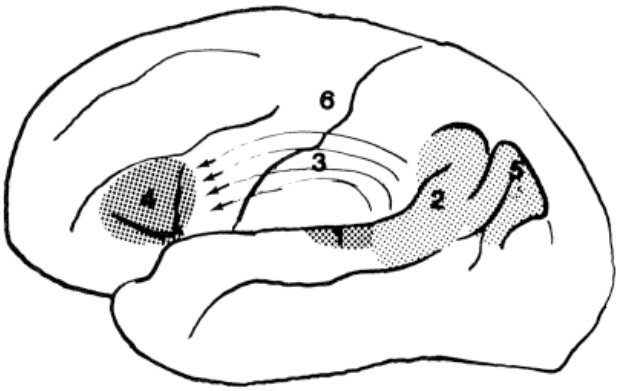
2. Dysarthria

- Inability to articulate spoken words.
- Ability to understand and synthesize speech remains intact.
- Results from paralysis of pharyngeal, palatal, lingual, or facial musculature.
- Observed with cerebellar lesions (scanning or staccato speech).

3. Dysphasia or aphasia

- Language includes: spontaneous production; response to commands; the ability to repeat complex phrases; the ability to name objects; and the ability to read and write
- Dysphasia: inability to process language; abnormal language expression, repetition, and comprehension; inability to read and write;
- Lesion in dominant hemisphere of all rt handed and >70% of left handed individuals.
- Sensory or Wernick's or fluent aphasia:
    i. The lesion is in superior temporal gyrus area 39 & 40;
    ii. Associated with embolic strokes affecting the inferior division of the middle cerebral artery,
    iii. Inability To understand spoken(word deafness) and written (Dyslexia) language
    iv. Fluency with no meaning of speech - unintelligible jargon
    v. Neologism: meaningless new words
    vi. Paraphrasing: incorrect words
    vii. Nominal aphasia: inability to name objects
    viii. Repetition is affected
    ix. Neighborhood signs:
        1. Superior quadrantanopsia due to involvement of optic radiations,
        2. Limb apraxia a disorder of skilled movement due to involvement of the inferior parietal lobule
        3. Finger agnosia, acalculia, or agraphia due to involvement of the angular gyrus.
    x. No motor paralysis
- Expressive, motor, or Broca's or non fluent aphasia:
    i. Lesion in frontal lobe areas 44, 45.
    ii. Can understand, write and read language.
    iii. Inability to express words ;
    iv. Agrammatism.
    v. Repetition is impaired
    vi. Neighbourhood sign: Right hemi paresis
- conduction aphasia :
    i. Inability to transfer signals from the Wernicke to the Broca area
    ii. Lesion in arcuate fasciculus
    iii. Auditory and reading comprehension is fairly good.
    iv. Can follow command
    v. Repetition is absent
    vi. Spontaneous speech is usually fluent
    vii. Articulation is better than Broca's aphasia
- The combination of Broca and Wernicke aphasias - global aphasia or mutism.

- The transcortical aphasias:
    1. Broca's area, Wernicke's area and the arcuate fasciculus are undamaged but are cut off from the rest of the brain by infarcted tissue.
    2. Patients cannot read and also cannot write
    3. No comprehension
    4. **Repetition is intact**.



Key: 1, Heschel's gyrus (auditory cortex); 2, Wernicke's area; 3, arcuate fasciculus; 4, Broca's area; 5, angular gyrus; 6, motor cortex.

- **"split brain":**
    - Damage to the entire corpus callosum;
    - Inability to verbally tell you what an article is, if it is placed in the left hand (assuming left hemisphere dominance and that the patient is prevented from looking at it).
    - Additionally, this individual will be unable to understand written language if the writing is presented only to the left visual field. This material reaches only the right hemisphere and cannot be transferred to the left or verbal hemisphere, for interpretation.

Type of dysphasia	Spontaneous speech	Compre-hension	Repetition	Naming	Associated signs
Transcortical					
Sensory	Fluent				Variable (signs of "watershead" infarct); may have Gerstmann's syndrome
severe	Echolalic	Poor	Good	Poor	
mild-anomia	Circumlocution	Good	Good	Poor	
Motor	Nonfluent except when repeating	Good	Good	Can be good	May have right hemiparesis
Mixed sensorimotor "isolation of	Nonfluent	Poor	Good	Poor	Signs of "watershead" lesion (weakness in proximal upper limb

speech area"					etc.)
Wernicke's	Fluent, paraphasic	Poor	Poor	Poor	May be normal; may be visual field defect or coprtical sensory loss; may have hemiparesis.
Broca's	Nonfluent	Good	Poor (but can be better than spontaneous speech)	Poor	Right hemiparesis
Conduction	Fluent, paraphasic	Good	Poor	Poor	Variable; often cortical sensory loss or lost pain sensation on right side
Global	Nonfluent	Poor	Poor	Poor	Right hemiparesis

Mental status:

1. **Memory**
    1. **Memory** is an organism's ability to store, retain, and recall information
    2. Sensory memory: The ability to look at an item, and remember what it looked like with just a second of observation
    3. **Recent memory.** Ask the patient to recall three items or a brief story after a delay of 3 to 5 minutes; difficulty with recall after about 3 to 5 minutes usually signifies damage to the limbic system located in the medial temporal lobes and medial diencephalon
    4. **Remote memory (retrograde amnesia):** Ask the patient about historical or verifiable personal events. **Cortical area:** first stored in the temporal lobes and becomes more widespread in both hemispheres. Remote memory is relatively preserved in chronic dementing processes until late in the course of the disease.
    5. All aspects of memory are impaired in acute encephalopathy.

Orientation

1. Cognitive sense of his status in time, place, and person.
2. The sense of time is first to be impaired in organic dysfunction, and the sense of person is the last to be lost.
3. Who does not know who he or she is, but can tell the time and is oriented in place, hasa psychological disturbance than to have an organic aetiology for the condition.

Intelligence

1. Ability to apply previous knowledge to a new situation and to use reason in solving problems.

2. Vocabulary, calculations (eg, serial-7 calculations), abstraction (eg, use of proverbs), and judgment (eg, what to do with a found wallet) are good indicators of intelligence.

Psychological disturbances

1. Illusions: distorted reality:
2. Hallucination : perception in the absence of a reality
3. Delusions of grandeur: one believes oneself possessed of great importance, power, wealth, intellect, or ability.

EXAMINATION OF THE CRANIAL NERVES

Olfactory nerve - CN I

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1. Olfactory pathway:
    a. Olfactory epithelium →Unmyelinated axons →the cribriform plate → the olfactory bulb → the olfactory tract →the medial and lateral olfactory straie : Medial olfactory straie → the olfactory tract of opposite side;The lateral olfactory straie → the primary olfactory cortex in the temporal lobe
    b. The primary olfactory cortex:
        i. Periamygdaloid
        ii. The prepyriform :
            1. Uncus,
            2. Parahippocampal gyrus.
    c. Pain from the nasal epithelium is carried to the central nervous system by the trigeminal nerve
2. Olfaction:
    a. Olfaction, taste and trigeminal receptors together contribute to flavor. The human tongue can distinguish only among five distinct qualities of taste, while the nose can distinguish among hundreds of substances, even in minute quantities.
    b. Dogs in general have an olfactory sense approximately a hundred thousand to a million times more acute than a human's. Olfaction helps animals for mating partner and their prey.
3. Lesinos:
    a. Lesions of the nerve result in parosmia (altered sense of smell) or anosmia (loss of smell).
    b. Olfactory fatigue or adaptation is the temporary, normal inability to distinguish a particular odor after a prolonged exposure to that airborne compound[
    c. Olfactory hallucinations (phantosmia) occur in brain trauma, tumors and temporal lobe epilepsy.
4. Causes:
    a. Common cold
    b. Fractures of the cribriform plate of the ethmoid bone.
    c. Frontal lobe tumors
    d. Blunt trauma, such a coup-contra-coup damage, meningitis and tumors of the frontal lobe.
5. Test:

a. Olfactory function is tested by having the patient smell common objects such as coffee or perfume.

6. Pheromone:

a. Chemicals capable of acting outside the body of the secreting individual to impact the behavior of the receiving individual. There are alarm pheromones, food trail pheromones, sex pheromones, and many others that affect behavior or physiology.

b. The **terminal nerve**, or **cranial nerve zero**:

i. Discovered by German scientist Gustav Fritsch in 1878 in the brains of sharks. It was first found in humans in 1913,

ii. It projects from the nasal cavity, enters the brain just a little bit ahead of the other cranial nerves as a microscopic plexus of unmyelinated peripheral nerve fascicles.

iii. Although very close to the olfactory nerve, nerve zero is not connected to the olfactory bulb, where smells are analyzed. This fact suggests that the nerve is either vestigial or may be related to the sensing of pheromones.

iv. nerve zero projects to the medial and lateral septal nuclei, and the preoptic areas, all of which are involved in regulating sexual behavior in mammals.

Optic nerve - CN II

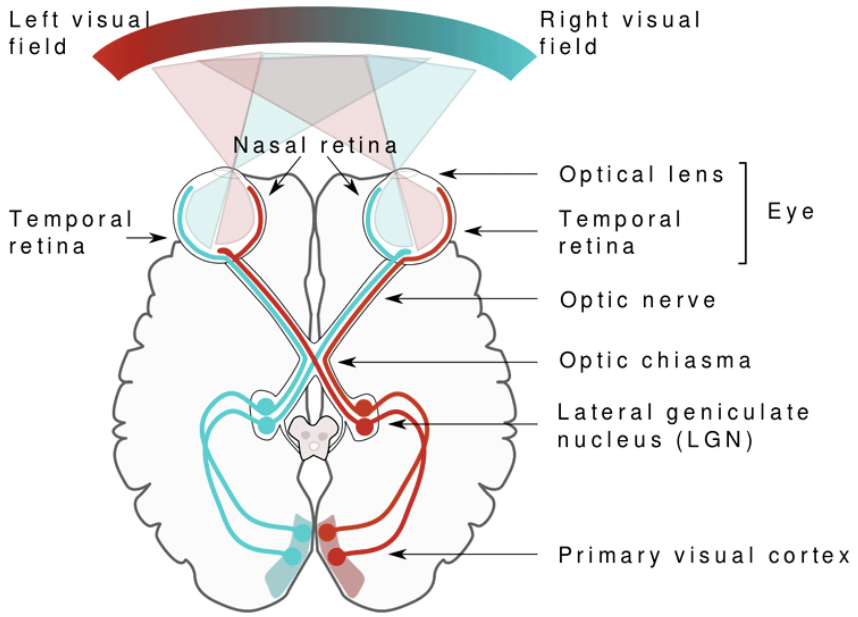
Embryology:

1. Derived from an outpouching of the diencephalon;
2. Considered to be part of the central nervous
3. Covered with myelin produced by oligodendrocytes rather than the schwann cells;
4. Encased within the meninges.
5. Peripheral neuropathies like guillain-barré syndrome do not affect the optic nerve.

Pathway:

1. **Vision**:

The rods (dim) and cones (colour) of the retina → the temporal derivations reach the ipsilateral and the nasal derivations the contralateral optic tract by crossing at optic chiasm → optic nerve → The lateral geniculate nucleus → optic radiation traversing the temporal (Myer loop) and parietal lobes via post limb of internal capsule → visual cortex (Broadman areas 17,18,19) located in and around the calcarine fissure in the occipital lobe



2. Light reflex
3. Accommodation reflex
4. Gaze
5. Fixation
6. The primary circadian "clock" suprachiasmatic nucleus in the hypothalamus which regulate the circadian rhythms;

The **optic disc** or **optic nerve head:**

- Location where ganglion cell axons exit the eye to form the optic nerve.
- No light sensitive rods or cones.
- A break in the visual field called "the blind spot"

Papilledema:

- Venous engorgement (usually the first signs)
- Loss of venous pulsation
- Hemorrhages over and / or adjacent to the optic disc
- Blurring of optic margins
- Elevation of optic disc
- Paton's lines = radial retinal lines cascading from the optic disc
- visual field: enlarged blind spot;
- The visual acuity may remain relatively intact until papilledema is severe or prolonged.

Causes:

- Raised intracranial pressure: brain tumor, pseudotumor cerebri or cerebral venous sinus thrombosis, Intracerebral hemorrhage

The **macula** or **macula lutea:**

- Oval-shaped highly pigmented yellow spot near the center of the retina
- Diameter of around 5 mm
- Histologically two or more layers of ganglion cells.
- Its center is the fovea, a small pit that contains the largest concentration of cone cells in the eye and is responsible for central vision,
- Also contains the parafovea and perifovea.

Macular degeneration:

Dry (atrophic) form: This type results from the gradual breakdown of cells in the macula, resulting in a gradual blurring of central vision. Single or multiple, small, round, yellow-white spots called drusen are the key identifiers for the dry type.

Wet (exudative or neovascular) form: In the wet form of macular degeneration, newly created abnormal blood vessels grow under the center of the retina. These blood vessels leak, bleed, and scar the retina, distorting or destroying central vision.

Cherry-red spot:

The choroid appearing as a red spot through the fovea centralis surrounded by a contrasting white edema; it is noted in cases of infantile cerebral sphingolipidosis.

Tests:

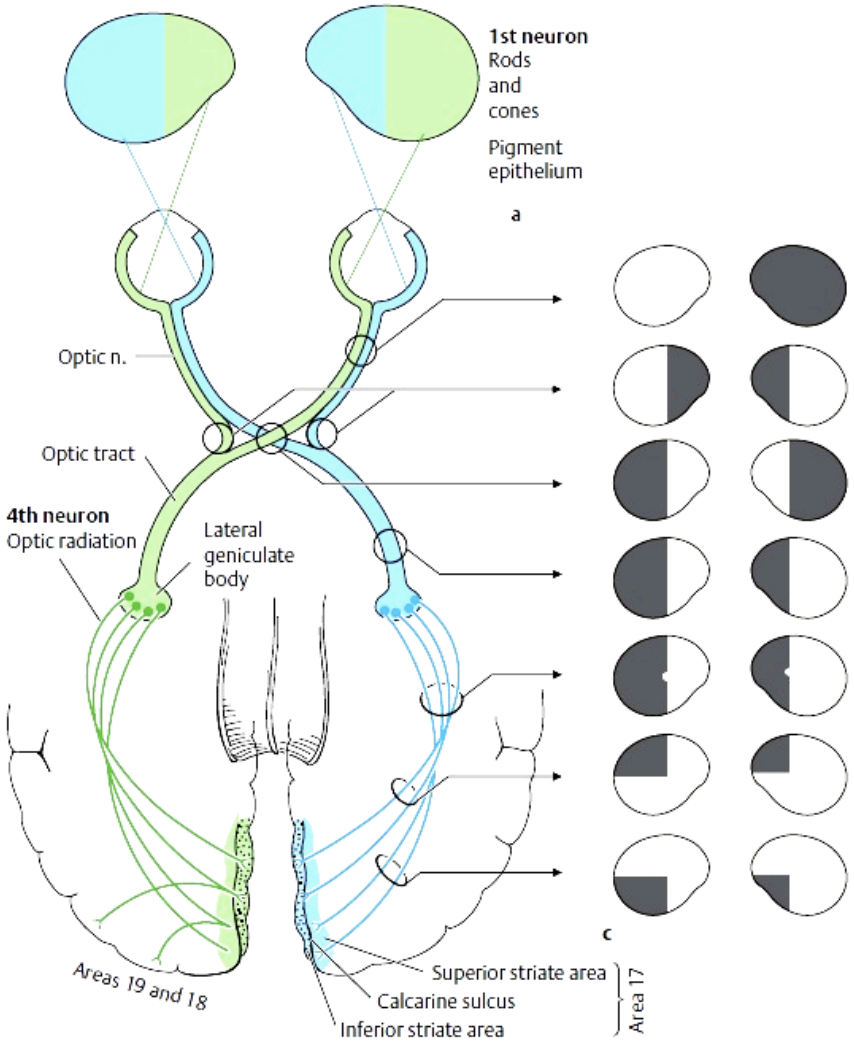
- Acuity - the Snellen chart (near and distant vision)
- Visual fields - confrontation or perimetry
- Color - Ishihara chart
- Fundoscopy

Visual defects:

1. Deficits in vision resulting from a single lesion can either be homonymous or nonhomonymous, i.e., affecting the same or different parts of the two eyes' visual field.
2. Relatively small visual field deficits are called scotomas, while large ones are called anopsias.

Lesions of the visual pathways:

1. Right optic nerve: total loss of vision in the right eye; right afferent pupil deficit (Marcus Gun pupil).
2. Optic chiasm: loss of vision in the temporal half of both visual fields: bitemporal hemianopsia.
3. Right optic tract: complete loss of vision in the left hemifield: contralateral hemianopsia.
4. Right optic radiation just after the LGN: loss of vision in the left hemifield: contralateral hemianopsia.
5. Right visual cortex: complete loss of vision in the left hemifield: contralateral hemianopsia.
6. Calcarine cortex: Hemianopsias with macular sparing



Oculomotor nerve - CN III

Oculomotor nuclei:

1. Originates in midbrain at the level of the superior colliculus. Two nuclei: Oculomotor and Edinger-Westphal
2. Several subnuclei: innervate extraocular muscles, the eyelids, and the pupils except superior oblique and the lateral rectus muscle.
   - The superior rectus subnucleus innervates the contralateral superior rectus.
   - The levator subnucleus is a single central caudate nucleus and innervates both eyelids.
   - Other subnucleii supply the ipsilateral muscles.
3. The Edinger-Westphal nucleus supplies parasympathetic fibres to the iris via the ciliary ganglion, and thus controls pupil constriction.

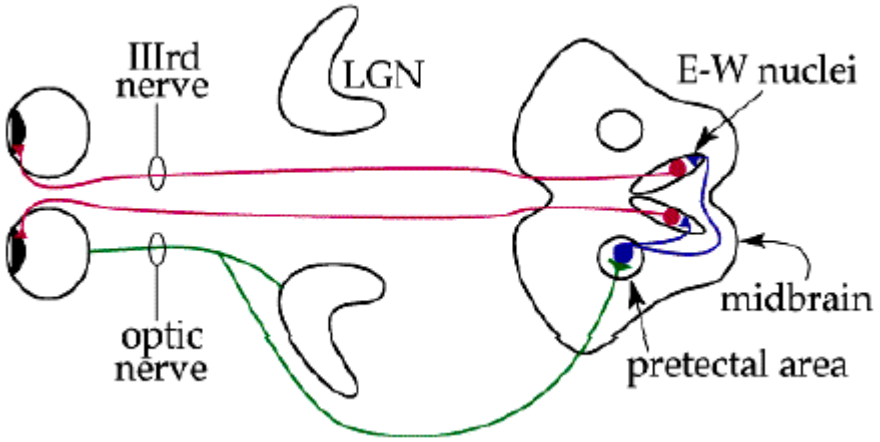
Pathway:

1. Arises from the anterior aspect of midbrain.
2. Passes between the borders of the tentorium cerebelli.
3. Runs along the lateral wall of the cavernous sinus,

4. Receives filaments from the cavernous plexus of the sympathetic, and a communicating branch from the ophthalmic division of the trigeminal.
5. Divides into two branches, which enter the orbit through the superior orbital fissure, between the two heads of the lateral rectus.
6. Placed below the trochlear nerve and ophthalmic nerve.

Lesions of CN III:

1. Nerve paralysis:
    1. Ipsilateral ptosis
    2. Dilatation of pupil
    3. Eye ball turned down and out due to paresis of adductor, superior and inferior rectus
    4. In subtle cases, patients complain of only diplopia or blurred vision.
2. Nucleus of the third nerve:
    1. Cause bilateral ptosis,
    2. Findings mentioned above.
7. Outward deviation of eye: D.D:
    a. In internuclear ophthalmoplegia, convergence is preserved.
    b. Paralysis of CN III : diplopia in more than 1 direction, distinguishing itself from CN IV paralysis
    c. Pupillary involvement is an additional clue to involvement of CN III.
8. Pupil-sparing CN III paralysis: diabetes mellitus, vasculitides of various etiologies ,multiple sclerosis.
9. In transtentorial herniation, the uncus can squeeze the third cranial nerve, causing the pupil to dilate and precedes III nerve compression.
10. **The pupillary light reflex:**
    a. Retina →Optic nerve →The pretectum, → Edinger wespal nucleus (para sympathetic) bilateral → ciliary ganglion → Ciliary muscle → light reflex.



Normal Pupillary reflex:

1. **Light Reflex:**
    a. The **pupillary light reflex** controls the diameter of the pupil, in response to light. Greater intensity light causes the pupil to become smaller, whereas lower intensity light causes the pupil to become larger.
    b. Light thrown to one eye makes the opposite pupil to constrict- consensual light reflex
    c. The optic nerve is responsible for the afferent limb and the oculomotor nerve (bilateral) for the efferent limb

2. Accommadationreflex :

- Active process; bilateral; iii CN is the main nerve
- Pathway:

  Each retina → optic nerve →optic radiation →occipital lobe → frontal eye field → Edinger-Westphal nucleus → 3rd cranial nerve → ciliary muscle, the medial rectus muscle and (via parasympathetic fibres) the sphincter pupillae muscle.
- **Triad:**

  1) Contraction of ciliary muscle

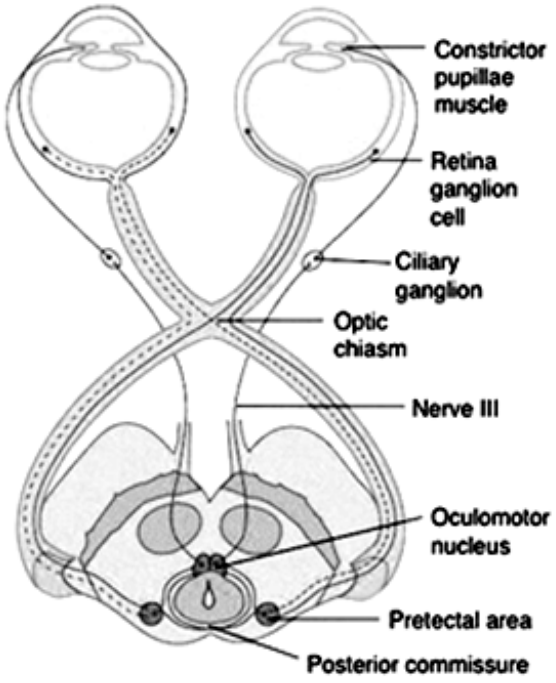
  2) Convergence of eye balls

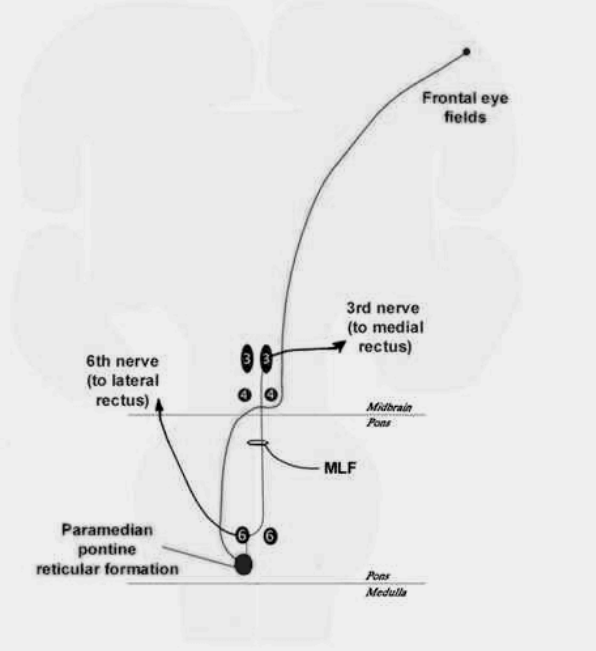
  3) Constriction of Pupils

3. Conjugate gaze:

a. Voluntary gaze:

Frontal eye field area 8 and parieto occipital eye field area 19 → opposite paramedian pontine reticular formation → same side VI nerve→lateral rectus and via medial longitudinal fasciculus → opposite iii nerve nucleus→medial rectus





b. Horizontal gaze:reflex:

Cerebral hemispheres, cerebellum, vestibular nuclei, and neck→ horizontal gaze center i.e paramedian pontine reticular formation→6th cranial nerve nucleus→lateral rectus on the same side, and → the medial longitudinal fasciculus (MLF)→contralateral 3rd cranial nerve nucleus and the medial rectus

c. Vertical gaze:

vestibular system; → MLF on both sides → 3rd and 4th cranial nerve nuclei, the interstitial nucleus of Cajal, and the rostral interstitial nucleus of the MLF.

cerebral hemispheres→pretectum→ the 3rd and 4th cranial nerve nuclei.

The rostral interstitial nucleus of the MLF integrates the neural input into a final command for vertical gaze.

d. Eye fixation:

The superior colliculus (Tectum) bilateral → visual cortex and cervical spinal cord which control orienting head and eye movements towards object:

1) Fixation, in which the eyes are directed toward a motionless object,
2) Smooth pursuit, in which the eyes move steadily to track a moving object;
3) Saccades, in which the eyes move very rapidly from one location to another;

e. Doll's eye movement:

- When the oculocephalic reflex is present (positive doll's eyes), the eyes do not turn with the head; it is maintaining fixation on a single point in space. The eyes thus appear to be moving relative to the head in the direction opposite to the head movement.

- This reflex is usually suppressed (and therefore not usually tested) in conscious patients, but it is a normal finding in comatose patients.
- Absence or asymmetry of this reflex in a comatose patient indicates dysfunction somewhere in the reflex pathway:
- afferent limb: labyrinth and vestibular nerve, and neck proprioceptors
- the efferent limb: cranial nerves III and VI and the pathways connecting them in the pons and medulla.

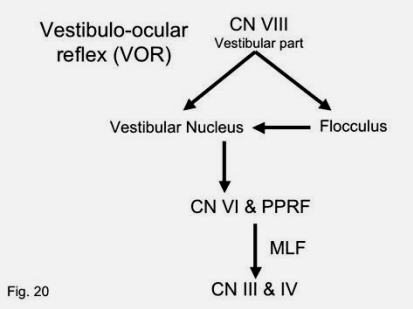


Fig. 20

4. Abnormal Pupils:

1. Amaurotic,: "blind eye," with no light perception as a result of an optic nerve lesion.

A.) Pupils are of equal size.

B.) Neither pupil reacts when the defective eye is stimulated.

1.) There is no direct light response.

2.) There is no consensual light response to the contralateral eye.

C.) Both pupils react when the contralateral eye is stimulated.

1.) Direct light response is present.

2.) Consensual light response is present.

D.) Accommodation reflex is normal.

2. The Marcus-Gunn pupil:

Stimulation of the normal eye produces full constriction in both pupils. Immediate subsequent stimulus of the affected eye produces an apparent dilation in both pupils since the stimulus carried through that optic nerve is weaker. It occurs when there is a unilateral lesion in the afferent visual pathway anterior to the chiasm.

3. Argyll Robertson Pupil (ARP):

a. Are bilateral small pupils that constrict when the patient focuses on a near object

b. Do not constrict when exposed to bright light

c. "Prostitute's Pupils" because of their association with tertiary syphilis and because of the convenient mnemonic that, like a prostitute, they “accommodate but do not react.”[1]

d. They are a highly specific sign of neurosyphilis

e. The vicinity of the sylvian aqueduct of the third ventricle as the likely region of damage. A lesion in this area would involve efferent pupillary fibres sparing the accommodation fibres

f. **Adie's (Tonic) Pupil:**

1. The lesion is in the ciliary ganglion by a viral or bacterial infection and characterized by a tonically dilated pupil.

Adie syndrome presents with three hallmark symptoms:

1. Dilated pupil

2. Deep tendon reflexes absent

3. Diaphoresis (excessive sweating).

4. **Parinaud's Syndrome**:

- This syndrome occurs with injury to the superior colliculus, pretectal area**,** and posterior commissure. It occurs in encephalitis, and in tumors of the pineal gland, posterior 3rd ventricle**,** and midbrain. A vascular etiology is occlusion of the collicular branches of the posterior choroidal artery which is a branch of the posterior cerebral artery.
- Bilateral paralysis of upward gaze and a questionable weakness of convergence. The pupils constricted upon convergence but not in response to light.

5. Horner syndrome:

1. Damage to the ipsilateral sympathetic nervous system
2. Congenital or iatrogenic.
3. Pancoast tumor or thyrocervical venous dilatation.
4. Signs found in all patients on affected side of face include;
   - Ptosis
   - Miosis
   - Anhidrosis
   - Enophthalmos
   - Sometimes there is flushing of the face

5. Causes:

a. Transection of the cervical spinal cord (hypothalamospinal pathway)

b. Compression of the sympathetic chain by a lung tumor (Preganglionic lesions)

c. A tumor in the cavernous sinus (Postganglionic lesions)

6. Internuclear optholmoplegia:

1. During horizontal gaze, the medial longitudinal fasciculus (MLF) on each side of the brain stem enables abduction of one eye to be coordinated with adduction of the other.
2. The MLF connects the following structures:
   - 6th cranial nerve nucleus (lateral rectus)
   - Pontine reticular formation (horizontal gaze center)
   - Contralateral 3rd cranial nerve nucleus (medial rectus)
   - The MLF also connects the vestibular nuclei with the 3rd cranial nerve nuclei.
3. Internuclear ophthalmoplegia results from a lesion in the MLF.

4. Caused by:

   Multiple sclerosis and may be bilateral.

   Stroke and is unilateral.

   Neurosyphilis,

7. Cortical eye blindness:

- Bilateral lesions of the primary visual cortex may cause cortical blindness.
- Patients have no vision but the response of the pupil to light is intact (as the reflex does not involve the cortex).
- Fundoscopy is normal.
- Cortical blindness can be associated with visual hallucinations, denial of visual loss (Anton–Babinski syndrome)
- Side effect of anti-epilepsy drugs (AEDs) over time.
- The posterior cerebral artery supplies the occipital lobe, and can be associated with cortical blindness.

Amblyopia:

- One eye obviously drifts off target ("wandering eye"). However, the patient is unaware of this and does not see double.
- Due to severe muscle weakness or poor vision (usually in one eye).
- No double vision as the brain "turns off" input from the bad eye.

Trochlear nerve - CN IV

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1. Nucleus:
   a. The nucleus of the nerve is located in the midbrain.
   b. It innervates the superior oblique muscle,
   c. The superior oblique muscle depresses, rotates inward, and abducts the eye.
   d. Allows a person to view the tip of his or her nose.
2. Unique features:
   a. Smallest nerve.
   b. Has the greatest intracranial length.
   c. Decussates before innervating its target.
   d. Exits from the dorsal aspect of the brainstem.
3. Lesions:
   1. An isolated right superior oblique paralysis results in :
      i. Exotropia to the right (R),
      ii. Double vision that increases on looking to the (L),
      iii. And head tilt to the right (R).
      iv. No ptosis and/or pupillary involvement in contrast to CN III paralysis.
   2. A lesion of the trochlear nucleus affects the contralateral eye.

4. Causes :
    a. Head trauma.
    b. A generalized increase in intracranial pressure:
        i. Hydrocephalus,
        ii. Pseudotumor cerebri,
        iii. Hemorrhage,
        iv. Edema – will affect the fourth nerve, but the abducens nerve (VI) is usually affected first
        v. Infections (meningitis, herpes zoster),
        vi. Demyelination (multiple sclerosis),
        vii. Diabetic neuropathy and
        viii. Cavernous sinus disease can affect the fourth nerve,
        ix. Orbital tumors and
        x. Tolosa-Hunt syndrome
            1. Unilateral headaches with extraocular palsies, assumed to be, associated with inflammation of the areas behind the eyes (cavernous sinus and superior orbital fissure).
            2. In general, these diseases affect other cranial nerves as well. Isolated damage to the fourth nerve is uncommon in these settings.
        xi. The most common cause of chronic fourth nerve palsy is a congenital defect, in which the development of the fourth nerve (or its nucleus) is abnormal or incomplete.

Trigeminal nerve - CN V

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1. Nucleus:
    a. The nucleus of the nerve stretches from the midbrain through the pons to the cervical region.
    b. It provides sensory innervation for the face and supplies the muscles of mastication.
2. Branches:
    a. The ophthalmic nerve (V_1),- sensory
    b. The maxillary nerve (V_2), - sensory
    c. The mandibular nerve (V_3). – motor and sensory
3. Pathways:
    a. The three branches converge on the trigeminal ganglion (semilunar ganglion or gasserian ganglion), in Meckel's cave.
    b. Exit of skull: (The mnemonic **s**tanding **r**oom **o**nly)
        i. The ophthalmic- the superior orbital fissure
        ii. Maxillary - the foramen rotundum
        iii. Mandibular-- foramen ovale

- Innervation:

- The ophthalmic nerve: scalp, forehead, the upper eyelid, the conjunctiva , cornea, nose except alae nasi, the nasal mucosa, the frontal sinuses, and parts of parts of the meninges
- The maxillary nerve: the lower eyelid, cheek, the nares, upper lip, the upper teeth and gums, the nasal mucosa, the palate and roof of the pharynx, the maxillary, ethmoid and sphenoid sinuses, and parts of the meninges.
- The mandibular nerve: the lower lip, the lower teeth and gums, the chin and jaw (except the angle of the jaw, which is supplied by C2-C3), parts of the external ear, and parts of the meninges.
- The mandibular nerve: sensation from the mouth but not taste sensation and motor branches supply:
    - Muscles of mastication
    - Masseter
    - Temporalis
    - Medial pterygoid
    - Lateral pterygoid
- Other
    - Tensor veli palatini
    - Mylohyoid
    - Anterior belly of digastric
    - Tensor tympani

Paralysis:

1. Ophthalmic:
    a. Superior orbital fissure syndrome: fracture of the superior orbital fissure leading to diplopia, paralysis of extraocular muscles, exophthalmos, and ptosis.
    b. Sensory loss over the forehead along with paralysis of CN III and CN IV.
2. Maxillary:
    a. Loss of sensation over the cheek and is due to lesions of the cavernous sinus; additional paralysis of V1, CN III and CN IV.
3. Complete paralysis of CN V:
    a. Sensory loss over the ipsilateral face and weakness of the muscles of mastication.
    b. Attempted opening of the mouth results in deviation of the jaw to the paralyzed side.
4. **Wallenberg syndrome** (lateral medullary syndrome) due to the posterior inferior cerebellar artery thrombosis::
    a. Loss of pain/temperature sensation from one side of the face and the other side of the body.
    b. This is due to fact that the ascending spinothalamic tract (which carries pain/temperature information from the opposite side of the body) is adjacent to the descending spinal tract of the fifth nerve (which carries pain/temperature information from the same side of the face)

Abducens nerve - CN VI

1. Nucleus:

a. Located in the pons, on the floor of the fourth ventricle, at the level of the facial colliculus.

b. Axons from the facial nerve loop around the abducens nucleus, creating the facial colliculus

2. Pathway:

a. Leaves the brainstem at the junction of the pons and the medulla, medial to the facial nerve.

b. Runs alongside the internal carotid artery in the cavernous sinus

c. Enters the orbit through the superior orbital fissure and innervates the lateral rectus muscle of the eye.

d. The long course of the abducens nerve between the brainstem and the eye makes it vulnerable to injury at many levels.

3. Lesions of VI nerve palsy:

i. Fractures of the petrous temporal bone

ii. Aneurysms of the intracavernous carotid artery.

iii. Tentorial herniation by stretching it between the point where it emerges from the pons and the point where it hooks over the petrous temporal bone.

iv. Demyelination, infections (e.g. Meningitis), cavernous sinus diseases and various neuropathies.

v. Diabetic neuropathy.

vi. Wernicke-Korsakoff syndrome:

1. Is caused by thiamine deficiency, due to alcoholism.

2. Abnormalities are nystagmus and lateral rectus weakness.

vii. Tolosa-Hunt syndrome:

1. Idiopathic granulomatous disease that causes painful oculomotor palsies,unilateral headaches, pain around the sides and back of the eye.

2. Cause not known, thought to be, associated with inflammation of the areas behind the eyes (cavernous sinus and superior orbital fissure).

viii. Brain tumor, hydrocephalus, pseudotumor cerebri, hemorrhage, edema that exerts downward pressure on the brainstem, causing the nerve to stretch.

ix. A right-sided brain tumor can produce either a right-sided or a left-sided sixth nerve palsy as an initial sign. “False localizing signs.”

x. Isolated sixth nerve palsies in children are assumed to be due to brain tumors until proven otherwise.

4. Lesion of the Nucleus:

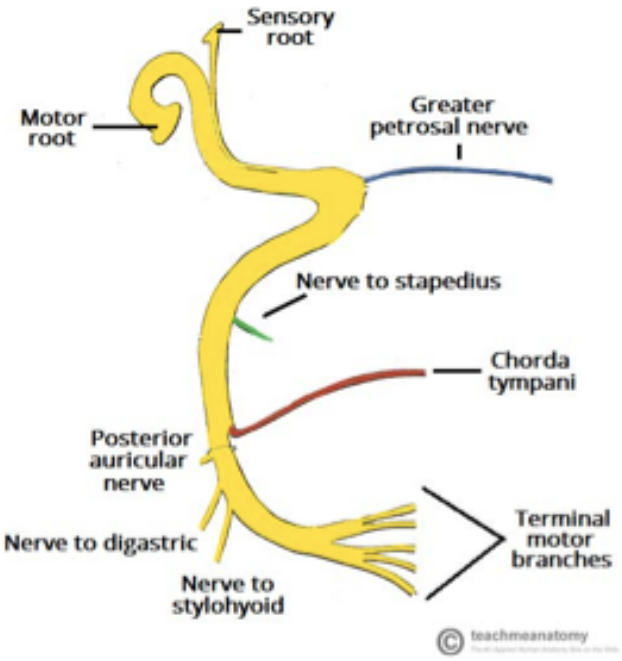
a. A horizontal gaze palsy; The abducens nucleus controls the lateral rectus muscle on the same side, and contralateral the medial rectus muscle of the opposite eye.

5. Internuclear ophthalmoplegia:

- The Median Longitudinal Fasciculus in midbrain connects the following structures:
    - 6th cranial nerve nucleus (lateral rectus)

        - Adjacent horizontal gaze center (paramedian pontine reticular formation)
        - Contralateral 3rd cranial nerve nucleus (medial rectus)
        - The MLF also connects the vestibular nuclei with the 3rd cranial nerve nuclei.
    - MLF lesion results in paresis of eye adduction in horizontal gaze but not in convergence. It can be unilateral or bilateral.
    - Cause: Multiple sclerosis; stroke
6. Gaze can be controlled by smooth pursuit or by saccades.
    - "Conjugate gaze" refers to the motion of both eyes in the same direction at the same time, and conjugate gaze palsy refers to an impairment of this function.
    - The lateral gaze is controlled by the paramedian pontine reticular formation (PPRF).[1]
    - The vertical gaze is controlled by the rostral interstitial nucleus of medial longitudinal fasciculus and the interstitial nucleus of Cajal.
    - Horizontal gaze:
        - Input from the cerebral hemispheres, cerebellum, vestibular nuclei, and neck.
        - Neural input from these sites converges at the horizontal gaze center (paramedian pontine reticular formation)
        - Integrated into a final command to the adjacent 6th cranial nerve nucleus, which controls the lateral rectus on the same side, and, via the medial longitudinal fasciculus (MLF), to the contralateral 3rd cranial nerve nucleus and the medial rectus it controls. Inhibitory signals to opposing eye muscles occur simultaneously.
        - The most common and devastating impairment of horizontal gaze results from pontine lesions that affect the horizontal gaze center and the 6th cranial nerve nucleus. Strokes are a common cause, resulting in loss of horizontal gaze ipsilateral to the lesion.
    - Vertical gaze:
        - Vestibular system → MLF on both sides → the 3rd and 4th cranial nerve nuclei, the interstitial nucleus of Cajal
        - Vertical gaze palsies commonly result from midbrain lesions, usually infarcts and tumors.
        - Parinaud's syndrome (dorsal midbrain syndrome), a conjugate upward vertical gaze palsy, may result from a pineal tumor or, less commonly, a tumor or infarct of the midbrain pretectum. This syndrome is characterized by impaired upward gaze, lid retraction (Collier's sign), downward gaze preference (setting-sun sign)
        - Downward gaze palsies: Impaired downward gaze with preservation of upward gaze usually indicates progressive supranuclear palsy.

Facial nerve - CN VII



The facial nerve is associated with the derivatives of the second pharyngeal arch. It is a mixed nerve which contains motor, sensory and parasympathetic fibers. It is more important as a motor than as a sensory nerve. It is a mixed nerve only while it is found inside the skull, but after leaves the skull is becomes a purely motor nerve and supplies muscles of facial expression.

Supranuclear pathways:

a. Somatomotor cortex: controlling motor component of facial nerve lies in precentral gyrus (Broadmann area 4,6,8)
b. Volitional component: Corticonuclear tracts descend and cross to supply both ipsilateral and contralateral facial (mainly to the contralateral side) nucleus i.e. frontal branch components of the facial nucleus receives bilateral cortico-nuclear tract innervation.
c. Emotional component: Input to the facial nucleus from the basal ganglia and limbic system control involuntary facial expression associated with emotion
d. From visual system (involved in blink reflex)
e. From trigeminal nerve and nuclei (involved in corneal reflex)
f. Auditory nuclei (involuntary closure of eye in response to loud noise)

Nucleus:

1. Motor nucleus: The motor nucleus lies deep in the reticular formation of the lower pons. The part of the nucleus that supplies muscles of the lower part of the face receive corticonuclear fibers only from the opposite cerebral hemisphere. The part of the nucleus that supplies muscles of the upper part of the face receives corticonuclear fibers from the motor cortex of both the right and left sides.

2. Superior salivatory nucleus (pons) – parasympathetic component to nerve of Wrisberg or Nervus intermedius. They go to *(a)* the lacrimal, nasal, and palatine glands (via the greater superficial petrosal nerve) and *(b)* the submandibular and sublingual salivary glands (via the chorda tympani nerve).
3. Nucleus of solitary tractus (medulla) – receives sensory component from nerve of Wrisberg or Nervus intermedius. These fibers go to taste buds on the anterior two-thirds of the tongue (via the chorda tympani nerve).
4. Before leaving brainstem: motor fibers wind around the abducens nucleus (CN VI) to form an internal genu
5. It leaves brainstem at pontomedullary junction.Facial nerve leave brainstem in close association with vestibulocochlear nerve at Cerebello-pontine angle (CP angle). Vestibular schwannoma and other tumors arising in the region of the cerebellopontine angle, may compress the facial nerve.
6. After leaving brainstem: motor fibers align with nervus intermedius and together enters Internal acoustic meatus, along with vestibulo-cochlear nerve (CN VIII) .

Roots of the facial nerve:

1. **Facial Nerve (motor nerve):** is a purely motor nerve supplies all the muscles of facial expression, the posterior belly of the digastric, the stylohyoid and the stapedius muscles.
2. **Nerve Intermedius:** consists of sensory and parasympathetic fibers
    i. Parasympathetic to all glands of head (submandibular, sublingual and lacrimal glands) except **the parotid gland** (glossopharyngeal nerve).
    ii. Sensory for auricle and tympanic membrane.
    iii. Special sensory: Taste for anterior 2/3 of the tongue

Course: Intracranial

The two roots travel through the internal acoustic meatus, a 1cm long opening in the petrous part of the temporal bone. Here, they are in very close proximity to the inner ear. Still within the temporal bone, the roots leave the internal acoustic meatus, and enter into the **facial canal**. The canal is a 'Z' shaped structure. Within the facial canal, three important events occur:

- Firstly the two roots **fuse** to form the facial nerve.
- Next, the nerve forms the **geniculate ganglion** (a ganglion is a collection of nerve cell bodies).
- Lastly, the nerve gives rise to the **greater petrosal nerve** (parasympathetic fibres to glands), the nerve to **stapedius** (motor fibres to stapedius muscle), and the **chorda tympani** (special sensory fibres to the anterior 2/3 tongue).

The facial nerve then exits the facial canal (and the cranium) via the **stylomastoid foramen**. This is an exit located just posterior to the styloid process of the temporal bone.

Extracranial

After exiting the skull, the facial nerve turns superiorly to run just anterior to the outer ear. The first extracranial branch to arise is the **posterior auricular nerve**. It provides motor innervation to the some of the muscles around the ear. Immediately distal to this, motor branches are sent to the posterior belly of the **digastric** muscle and to the **stylohyoid** muscle.

The main trunk of the nerve, now termed the **motor root** of the facial nerve, continues anteriorly and inferiorly into the parotid gland **(note – the facial nerve does not contribute towards the innervation of the parotid gland, which is innervated by the** *glossopharyngeal nerve***)**. Within the parotid gland, the nerve terminates by splitting into five branches:

1. Temporal branch
2. Zygomatic branch
3. Buccal branch
4. Marginal mandibular branch
5. Cervical branch

These branches are responsible for innervating the muscles of facial expression.

Motor Functions

Branches of the facial nerve are responsible for innervating many of the muscles of the head and neck. All these muscles are derivatives of the **second pharyngeal arch**.

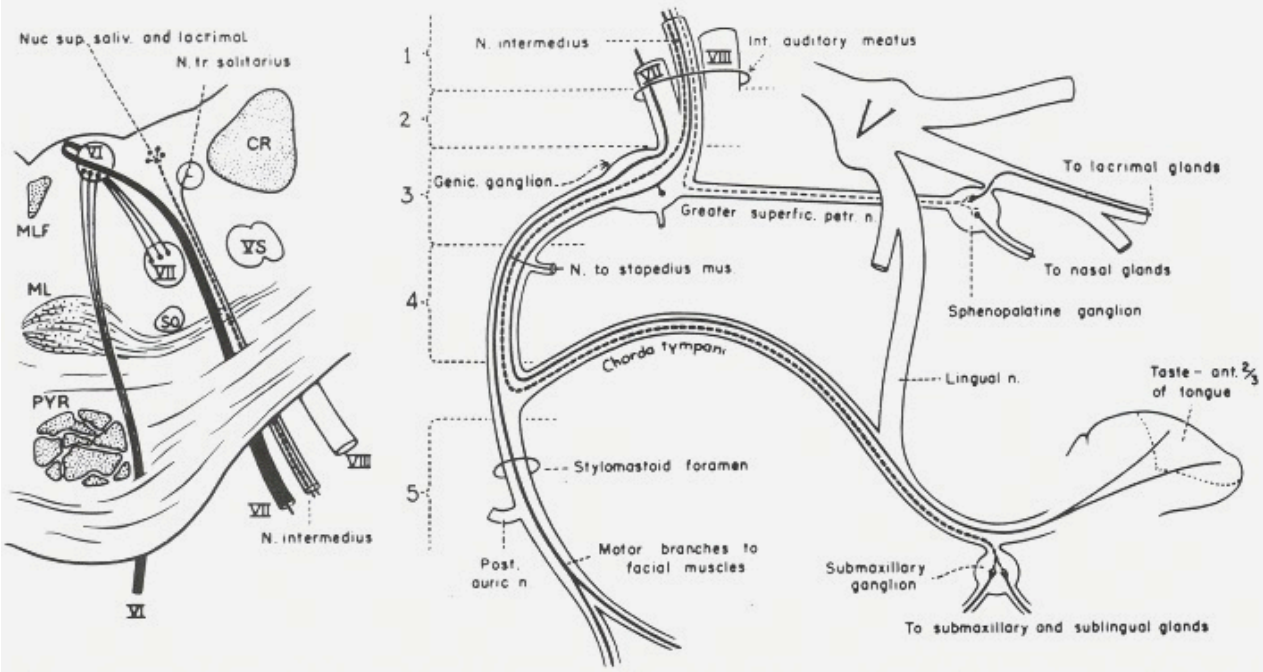
The first motor branch arises within the facial canal; the **nerve to stapedius**. The nerve passes through the pyramidal eminence to supply the stapedius muscle in the middle ear.

Between the stylomastoid foramen, and the parotid gland, three more motor branches are given off:

a. **Posterior auricular nerve** – Ascends in front of the mastoid process, and innervates the intrinsic and extrinsic muscles of the outer ear. It also supplies the occipital part of the occipitofrontalis muscle.
b. **Nerve to the posterior belly of the digastric muscle** – Innervates the posterior belly of the digastric muscle (a suprahyoid muscle of the neck). It is responsible for raising the hyoid bone.
c. **Nerve to the stylohyoid muscle** – Innervates the stylohyoid muscle (a suprahyoid muscle of the neck). It is responsible for raising the hyoid bone.

Within the parotid gland, the facial nerve terminates by bifurcating into five motor branches. These innervate the muscles of facial expression:

1. **Temporal branch** – Innervates the frontalis, orbicularis oculi and corrugator supercilii
2. **Zygomatic branch** – Innervates the orbicularis oculi.
3. **Buccal branch** – Innervates the orbicularis oris, buccinator and zygomaticus muscles.
4. **Marginal Mandibular branch** – Innervates the mentalis muscle.
5. **Cervical branch** – Innervates the platysma.



Lesions:

Lesion location	Manifestations
Above the facial nucleus (Supranuclear lesion)	Contralateral paralysis of lower facial muscles with relative preservation of upper muscles. Lesion located either in brainstem or cortex.
Pons (nuclear or fascicular lesion)	1. Ventral pontine lesion (of Millard-Gubler): ipsilateral facial monoplegia, lateral rectus palsy (VI), contralateral hemiplegia (corticospinal fibers). 2. Pontine tegmentum lesion (of Foville): ipsilateral facial monoplegia; contralateral hemiplegia (corticospinal fibers); paralysis of conjugate gaze to side of lesion (pontine paramedian reticular formation).
Cerebellopontine angle (peripheral nerve lesion).	1. Ipsilateral facial monoplegia, loss of taste to anterior two-thirds of tongue, impairment of salivary and tear secretion, hyperacusis (if VIII is not affected). 2. Additional cranial nerves may be involved: deafness, tinnitus, vertigo (VIII): 3. sensory loss over face and absence of corneal reflex (V); 4. ipsilateral ataxia (cerebellar peduncle).
Facial canal between internal auditory	Same as above except cranial nerves other than VII are not

Lesion location	Manifestations
meatus and geniculate ganglion	involved.
Facial canal between geniculate ganglion and nerve to stapedius muscle.	Facial monoplegia; impaired salivary secretion; loss of taste; hyperacusis.
Facial canal between nerve to stapedius and leaving of chorda tympani.	Facial monoplegia; impaired salivary secretion; loss of taste.
After branching of chorda tympani.	Facial paralysis, distribution related to site of lesion.

Syndromes:

FOVILLE'S SYNDROME:

- Blockage of the perforating branches of the basilar artery in the region of the pons.
- Structures affected by the infarct are the pontine reticular formation, nuclei of cranial nerves VI and VII, corticospinal tract, medial lemniscus, and the medial longitudinal fasciculus.
- This produces ipsilateral horizontal gaze palsy and facial nerve palsy and contralateral hemiparesis, hemisensory loss, and internuclear ophthalmoplegia.

MILLARD-GUBLER SYNDROME

- Contralateral hemiparesis, abducens palsy, and variablefacial nerve palsy. Lesions lie in the brain stem

MOBIUS' SYNDROME

- Nuclear agenesis of nerves VI and VII with facial and abducens palsy; often bilateral; with musculoskeletal abnormalities, cardiac anomalies, craniofacial defects, and mental retardation.

CEREBELLOPONTINE ANGLE LESIONS

- The cranial nerves VII and VIII are enclosed in a common sheath as they leave the brain stem in the Cerebellopontine angle
- Combination of facial weakness, tinnitus, hearing loss,
- Common tumors are acoustic neuroma, and meningioma

TRAUMATIC FACIAL NERVE PALSY

- Forcefs injury
- Trauma to the head

RAMSAY HUNT SYNDROME.(GENICULATE HERPES')

- Facial paresis
- Herpetic eruptions in geniculate and along the ipsilateral external auditory meatus

MELKERSSON-ROSENTHAL SYNDROME

- Triad of recurrent infranuclear facial paralysis,
- Orofacial edema (predominately of the lips), and

- Lingua plicata (fissured toungue) but all three features need not be present; only very rarely do they appear in combination. Onset can be at any age.
- Several causes have been put forth, such as inheritance, infection, autoimmunity, and allergic reaction.
- Treatment includes several drugs, such as clofazimine and steroids, as well as the surgical reduction of granulomatous tissue

BELL'S PALSY (IDIOPATHIC. FACIAL PALSY)

- Affects adults 20 to 40 years,
- Acute unilateral infranuclear facial palsy.
- Usually self-limiting.
- Bilateral resulting in total facial paralysis in around 1% of cases.
- Inflammation of the facial nerve, pressure is produced on the nerve where it exits the skull within its bony canal,
- Diminished taste
- An infectious process probably accounts for the majority of cases; vascular and genetic causes account for some cases.

Vestibulocochlear nerve - CN VIII

1. Cochlear nerve - hearing, vestibular nerve - balance.
2. Nucleus:
    a. Emerges from the medulla oblongata and enters at the pontomedullary junction via the internal acoustic meatus in the temporal bone, along with the facial nerve.
    b. The cochlear nerve:
        i. Organ of Corti → the spiral ganglia → cochlear nerve→ cochlear nucleus→primary auditory cortex in temporal lobe(41,42)
        ii. Vestibular system of the inner ear→ vestibular ganglion, ganglion of Scarpa,→vestibular nerve →vestibular nucleus on the lateral floor and wall of the fourth ventricle in the pons and medulla→ vestibular cortex?
3. Lesion:
    i. Hearing loss
    ii. Vertigo
    iii. False sense of motion
    iv. Loss of equilibrium (in dark places)
    v. Nystagmus
    vi. Motion sickness
    vii. Gaze-evoked tinnitus.
4. Tests for hearing:

a. The Weber test:
    i. Involves holding a vibrating tuning fork against the forehead in the midline. The vibrations are normally perceived equally in both ears because bone conduction is equal.
    ii. In conductive hearing loss, the sound is louder in the abnormal ear than in the normal ear. In sensorineural hearing loss, lateralization occurs to the normal ear.
b. Rinne test:
    i. The vibrating tuning fork is placed over the mastoid region until the sound is no longer heard. It is then held at the opening of the ear canal on the same side.
    ii. A patient with normal hearing should continue to hear the sound.
    iii. In conductive hearing loss, the patient does not continue to hear the sound, since bone conduction in that case is better than air conduction.
    iv. In sensorineural hearing loss, both air conduction and bone conduction are decreased to a similar extent.
c. Schwabach test:
    i. The patient's hearing by bone conduction is compared with the examiner's hearing by placing the vibrating tuning fork against the patient's mastoid process and then to the examiner's. If the examiner can hear the sound after the patient has stopped hearing it, then hearing loss is suspected.

5. Tests for balance:
    a. The Romberg test:
        i. When standing with feet placed together and eyes closed, the patient tends to fall toward the side of vestibular hypofunction.
        ii. When asked to take steps forward and backward, the patient progressively deviates to the side of the lesion.
        iii. Results of the Romberg test may also be positive in patients with polyneuropathies, and diseases of the dorsal columns, but these individuals do not fall consistently to 1 side as do patients with vestibular dysfunction.
    b. Ask the patient to touch the examiner's finger with the patient's hand above the head. Consistent past pointing occurs to the side of the lesion.
    c. Provocative tests:
        i. Nylen-Bárány test :
            1. Is performed with the patient sitting upright with the legs extended. The patient's head is then rotated by approximately 45 degrees. The clinician helps the patient to lie down backwards quickly with the head held in approximately 20 degrees of extension
            2. If rotational nystagmus occurs then the test is considered positive for benign positional vertigo.

ii. Caloric testing:

1. Hot and cold water produce currents in opposite directions and therefore a horizontal nystagmus in opposite directions in patients with an intact brainstem.

Glossopharyngeal nerve - CN IX:

Nucleus and course:

- From the nucleus in the medulla oblongata, nerve passes laterally across or below the flocculus, and leaves the skull through the central part of the jugular foramen. It passes between the internal jugular vein and internal carotid artery.
- Its superior and inferior (petrous) ganglia contain the cell bodies of pain fibers.
- Projects into many different structures in the brainstem:
    1. Solitary nucleus: Taste from the posterior one-third of the tongue and information from carotid baroreceptors and carotid body chemoreceptors
    2. Spinal nucleus of the trigeminal nerve: Somatic sensory fibers from the middle ear.
    3. Lateral Nucleus of Ala Cinerea: Visceral pain.
    4. Nucleus ambiguus: The lower motor neurons for the stylopharyngeus muscle.
    5. Inferior salivatory nucleus: Parasympathetic input to the parotid and mucous glands.

Branches

- Tympanic
- Stylopharyngeal
- Tonsillar
- Nerve to carotid sinus
- Branches to the posterior third of tongue
- Lingual branches
- A communicating branch to the Vagus nerve
- The glossopharyneal nerve contributes in the formation of the pharyngeal plexus along with the vagus nerve.

Functions:

- Mostly sensory; aids in tasting, swallowing and salivary secretions.
- Receives general sensory fibers from the tonsils, the pharynx, the middle ear and the posterior 1/3 of the tongue.
- Receives special sensory fibers (taste) from the posterior one-third of the tongue.
- Receives visceral sensory fibers from the carotid bodies.
- Supplies parasympathetic fibers to the parotid gland via the otic ganglion.
- Supplies motor fibers to stylopharyngeus muscle, the only motor component of this cranial nerve.
- Contributes to the pharyngeal plexus.

Lesions:

- Loss of taste in the posterior third of the tongue and loss of pain and touch sensations in the same area, soft palate, and pharyngeal walls.
- CN IX and CN X travel together, and their clinical testing is not entirely separable.

Vagus nerve - CN X

1. **Nucleus and course:**
    a. Start in the nucleus ambiguous,
    b. Motor supply to the pharyngeal muscles (except the stylopharyngeus and the tensor veli palati), palatoglossus, and larynx.
    c. Carries somatic sensation from the back of the ear, the external auditory canal, and parts of the tympanic membrane, pharynx, larynx, and the dura of the posterior fossa.
    d. Innervates the smooth muscles of the tracheobronchial tree, esophagus, and GI tract up to the junction between the middle and distal third of the transverse colon.
    e. Provides secretomotor fibers to the glands in the same region and inhibits the sphincters of the upper GI tract.
    f. Participates in vasomotor regulation of blood pressure by carrying the fibers of the stretch receptors and chemoreceptors (ie, aortic bodies) of the aorta and providing parasympathetic innervation to the heart.
2. The pharyngeal gag reflex:
    a. Tongue retraction and elevation and constriction of the pharyngeal musculature in response to touching the posterior wall of the pharynx, tonsillar area, or base of the tongue
3. Palatal reflex:
    a. Elevation of the soft palate and ipsilateral deviation of the uvula on stimulation of the soft palate are decreased in paralysis of CN IX and CN X.
    b. In unilateral CN IX and CN X paralysis, touching these areas results in deviation of the uvula to the normal side.
4. Recurrent laryngeal nerve:
    a. Unilateral paralysis results in hoarseness of voice.
    b. Bilateral paralysis results in stridor and requires immediate attention

Spinal accessory nerve - CN XI

1. Nucleus:
    a. Spinal accessory nucleus, located in the lateral horn of the upper spinal cord and continue With the nucleus ambiguus of the medulla oblongata, from which the cranial component of the accessory nerve is derived.
    b. The fibers of the spinal accessory nerve originate in these neurons

c. Enters the skull through the foramen magnum, the large opening at the base of the skull.
d. Exits the skull through the jugular foramen with the glossopharyngeal and vagus nerves
e. Only cranial nerve both to enter and exit the skull.
f. From the nucleus ambiguous, the spinal accessory nerve joins the vagus nerve in forming the recurrent laryngeal nerve to innervate the intrinsic muscles of the larynx.
g. The spinal portion of the nerve provides motor innervation to the sternocleidomastoid and the mid and upper thirds of the trapezius.

2. TEST:
    a. Have the patient push the face against resistance to the right and to the left. When the right SCM is weak, pushing to the opposite (ie, left) side is impaired, and vice versa.
    b. Shrugging of the shoulder is impaired ipsilaterally when the trapezius is weak.

Hypoglossal nerve - CN XII

1. The nucleus:
    a. Hypoglossal nucleus lies in the lower medulla, and the nerve leaves the cranial cavity through the hypoglossal canal
    b. Provides motor innervation for all the extrinsic and intrinsic muscles of the tongue except the palatoglossus.
2. Test:
    a. Tested by getting the person to poke the inside of their cheek, and feeling how strongly they can push a finger pushed against their cheek
    b. Look for signs of lower motor neuron disease, such as fasciculation and atrophy.
    c. Paralysis/paresis of one side of the tongue results in ipsilateral curvature of the tongue (apex toward the impaired side of the mouth) i.e., the tongue will move towards the affected side.

Sensory system

Non cortical sensory system

1. Constituted by the peripheral nerves with their central pathways to the thalamus. Light touch, pain, heat, cold, and vibration sensations included in this group.
2. Light touch:
    a. Tested by touching the skin with a wisp of cotton or tissue.
3. Pain:
    a. Tested by using a sharp object such as an open safety pin.
4. Temperature:
    a. Tested by touching the patient's skin with 2 test tubes, 1 with warm water and the other with cold water. Compare the 2 sides and also to a benchmark, such as the patient's own forehead (assuming sensation there is normal).

5. Vibration:
    a. Tested with a tuning fork, preferably with a frequency of 128 Hz. Compare findings on the 2 sides, and also compare findings with those in the same body part of the examiner.

Cortical sensory system

1. Includes the somatosensory cortex and its central connections.
2. Enables the detection of :
    a. The position and movement of the extremities in space (ie, kinesthetic sensation),
    b. Size and shape of objects (stereognosis),
    c. Tactile sensations of written patterns on the skin (graphesthesia),
    d. Tactile localization
    e. Tactile discrimination
3. Position sensation:
    a. Tested with the patient's eyes closed. The examiner moves various joints, being sure to hold the body part in such a way that the patient may not recognize movement simply from the direction in which the patient may feel the pressure from the examiner's hand.
4. Stereognosis:
    a. Tested by placing some familiar object (eg, ball, cube, coin) in the patient's hand while his or her eyes are closed and asking the patient to identify the object.
    b. Inability to recognize the size or shape is referred to as astereognosis.
    c. Agraphesthesia is the inability to recognize letters or numbers written on the patient's skin. These abilities are impaired in lesions of the right parietal region.
5. Extinction:
    a. Touching the patient in two places on opposite sides of their body, simultaneously. Then ask the patient to point to where they felt sensation. Normally they will point to both areas. If not, extinction is present.
    b. With lesions of the sensory cortex in the parietal lobe, the stimulus is not felt is on the side opposite of the damaged cortex. The sensation not felt is considered "extinguished".

MOTOR SYSTEM

Trophic state

1. Assess the 3 S : size, shape, and symmetry of a muscle.
2. Look for atrophy, hypertrophy, or abnormal bulging or depression in a muscle
    a. Atrophy: LMN lesion
    b. Disuse atrophy: UMN lesion
    c. Hypertrophy: exercise
    d. Psuedo hypertrophy: Duchenne muscular dystrophy.

Muscle tone

1. Muscle tone is the permanent state of partial contraction of a muscle and is assessed by passive movement.
2. Hypotonia: lower motor neuron lesions, spinal shock, and some cerebellar lesions.
3. Hypertonia: may manifest as spasticity or rigidity.
    a. Pyramidal lesions: clasp-knife phenomenon (ie, resistance to passive movement with sudden giving way).
    b. Bilateral frontal lobe lesions: paratonia in which resistance increases throughout flexion and extension.
    c. Extrapyramidal lesions: cogwheel (stepwise) or lead-pipe (uniform) resistance to passive movement.

Power:/Muscle strength

Use this muscle-strength scale when assessing and documenting muscle strength (Table 2).

Score	Description
0	Absent voluntary contraction
1	Feeble contractions that are unable to move a joint
2	Movement with gravity eliminated
3	Movement against gravity
4	Movement against partial resistance
5	Full strength

Involuntary movements

1. Include fibrillations, fasciculations, asterixis, tics, myoclonus, dystonias, chorea, athetosis, hemiballismus, and seizures.
2. Fibrillations: seen only in the tongue.
3. Fasciculations: may be seen; occcurs amyotrophic lateral sclerosis (ALS).
4. Asterixis: can be elicited by having the patient extend both arms with the wrists dorsiflexed and palms facing forward and eyes closed. Brief jerky downward movements of the wrist are considered a positive sign. Asterixis is commonly seen with metabolic encephalopathies.
5. Tics: are involuntary contractions of single muscles or groups of muscles that result in stereotyped movements. Gilles de la Tourette syndrome can manifest with multiple tics and elaborate complex movements and vocalizations.
6. Myoclonus; muscle jerk; it is a brief generalized body-jerk, which is sometimes asymmetric. These occur alone or in association with various primarily generalized epilepsies.

7. Dystonias: are muscle contractions that are more prolonged than myoclonus and result in spasms. Examples include blepharospasm, spasmodic torticollis, oromandibular dystonia, spasmodic dysphonia, and writer's cramp.
8. Athetosis: the spasms have a slow writhing character and occur along the long axis of the limbs or the body itself; the patient may assume different and often peculiar postures.
9. Chorea: means dance. Quasi-purposeful movements affect multiple joints with a distal preponderance.
10. Hemiballismus: violent flinging movement of half of the body. It is associated with lesions of the subthalamic nucleus (ie, body of Louis).
11. Seizures: orofacial or appendicular automatisms, repeated eye blinks, or tonic or clonic motor activity.

Reflexes

Three categories on the basis of their clinical significance.

1. **Primitive reflexes**
    1. These include the glabellar tap, rooting, snout, sucking, and palmomental reflexes. As a rule, these signs are generally absent in adults. When present in the adult, these signs signify diffuse cerebral damage, particularly of the frontal lobes (frontal-lobe release signs).
2. **Superficial reflexes**
    1. Segmental reflex responses that indicate the integrity of cutaneous innervation and the corresponding motor outflow.
    2. These include the corneal, conjunctival, abdominal, cremasteric, anal wink, and plantar (Babinski) reflexes.
    3. The corneal and conjunctival reflexes:
        a. Touching the appropriate structure with a sterile wisp of cotton produce bilateral winking.
        b. Absence: CN V paralysis.
        c. Blinking of 1 eye only: weakness of CN VII
    4. The abdominal reflex:
    a. Stroke from the umbilicus along the diagonals of the 4 abdominal quadrants: draws the umbilicus toward the direction of the line
    5. The cremasteric reflex:
        a. Stroke is along the medial thigh: elevation of the ipsilateral testis.
    6. The anal reflex:
        a. Stroking the perianal skin with a safety pin results in puckering of the rectal orifice owing to contraction of the corrugator-cutis-ani muscle.
    7. The plantar reflex:
        a. Stroking the lateral aspect of the sole with a sharp object: plantar flexion of the great toe, which is considered an absent Babinski sign.
        b. Dorsiflexion of the great toe (Babinski sign present) suggests an upper motor neuron lesion

c. Dorsiflexion of the big toe also may be associated with fanning out of the other toes, as detailed in Babinski's original description.

8. Flexion of the knee and hip may occur in the paretic leg with urinary and fecal incontinence. This is referred to as the en-mass reflex. Lack of either response may indicate absence of cutaneous innervation in the S1 segment or loss of motor innervation in the L5 segment ipsilaterally.

3. Deep tendon reflexes

1. These are monosynaptic spinal segmental reflexes; include the biceps, brachioradialis, triceps, patellar, and ankle jerks. When they are intact, integrity of the following is confirmed: cutaneous innervation, motor supply, and cortical input to the corresponding spinal segment.

Muscle	Spinal Roots	Nerve
Biceps	C5, 6	Musculocutaneous
Brachioradialis	C6	Radial nerve
Triceps	C7	Radial nerve
Knee jerk	L4	Femoral nerve
Ankle jerk	S1 S2	Tibial nerve

Table 4. Reflex-Grading System

Score	Reflexes
0	Absent
1	Hypoactive or present only with reinforcement
2	Readily elicited with a normal response
3	Brisk with or without evidence of spread to the neighboring roots
4	Associated with a few beats of unsustained clonus
5	Sustained clonus

CEREBELLAR SIGNS

1. Coordination of muscle activity by integrating the functions of the cortex, basal ganglia, vestibular apparatus, and spinal cord.
2. Midline cerebellar dysfunction:
    a. Results in ataxia of gait, difficulty in maintenance of upright posture, and truncal ataxia.
3. The following are various cerebellar signs:
    - Ataxia, atonia, and asthenia (the classic triad)

- Intention tremor (tremor that increases on activity)
- Dyssynergia (incoordination)
- Dysmetria (overshooting or undershooting)
- Dysrhythmia (inability to repeat a rhythmic tap)
- Dysdiadochokinesis (difficulty with rapid alternating movements)
- Dysarthria (staccato or scanning speech)

4. Gait: tested by heel to toe walking; tendency to sway or fall to 1 side indicates ataxia, suggesting ipsilateral cerebellar dysfunction.
5. Atonia and asthenia can occur in other lesions of the nervous system and are not specific to the cerebellum;
6. Intention tremor: oscillating tremor that accelerates in pace on approaching the target.
7. Dyssynergia: loss of smoothness of execution of a motor activity.
8. Dysmetria: overshooting or undershooting of a target while attempting to reach an object.
9. All 3 of these can be elicited by having the patient attempt to touch alternately his or her nose and the examiner's finger.
10. Dysrhythmia : inability to tap and keep a rhythm. Tested by tapping the table with a hand and asking the patient to repeat the maneuver.
11. Dysdiadochokinesis: inability to perform rapid alternating movements; tested by asking the patient to tap 1 hand on the other repeatedly while simultaneously pronating and supinating the hand.
12. Dysarthria: poor modulation of the volume and pitch of the speech,

MENINGEAL SIGNS

1. Signs of meningeal irritation indicate inflammation of the dura;
2. Nuchal rigidity: placing the examiner's hand under the patient's head and gently trying to flex the neck. Undue resistance implies diffuse irritation of the cervical nerve roots from meningeal inflammation.
3. The Brudzinski sign: flexion of both knees during the maneuver to test nuchal rigidity. This indicates diffuse meningeal irritation in the spinal nerve roots.
4. The Kernig sign: flexing the hip and knee on 1 side while the patient is supine, then extending the knee with the hip still flexed. Hamstring spasm results in pain in the posterior thigh muscle and difficulty with knee extension. With severe meningeal inflammation, the opposite knee may flex during the test.
5. Straight-leg raising (SLR) sign is elicited by passively flexing the hip with the knee straight while the patient is in the supine position. Limitation of flexion due to hamstring spasm and/or pain indicates local irritation of the lower lumbar nerve roots.

AUTONOMIC NERVOUS SYSTEM:

1. Anatomy:
    a. The autonomic nervous system has two main divisions: the sympathetic and the parasympathetic.
    b. Stimulating body processes - sympathetic division, or
    c. Inhibiting them - parasympathetic division.

2. An autonomic nerve pathway:
    a. Involves two nerve cells. The brain stem or spinal cord→autonomic ganglion→ internal organs
    b. Sympathetic division are located just outside the spinal cord on both sides of it.
    c. The ganglia for the parasympathetic division are located near or in the internal organs.
3. Function:
    a. Controls blood pressure, heart and breathing rates, body temperature, digestion, metabolism (thus affecting body weight), the balance of water and electrolytes (such as sodium and calcium), the production of body fluids (saliva, sweat, and tears), urination, defecation, sexual response, and other processes.
    b. Generally, the sympathetic division prepares the body for stressful or emergency situations—fight or flight.
    c. The parasympathetic division conserves and restores.
    d. Both the sympathetic and parasympathetic divisions are involved in sexual activity,
    e. Two chemical messengers (neurotransmitters), acetylcholine and norepinephrine, are used to communicate within the autonomic nervous system.
4. Symptoms:
    a. Erectile dysfunction an early symptom
    b. Orthostatic hypotension
    c. Urinary incontinence or retention
    d. Constipation
    e. The pupils may not dilate and narrow (constrict) as light changes.
5. Causes ANS disturbances:
    1. **Alcoholism,** can lead to nerve damage.
    2. Amyloidosis, affects the nervous system.
    3. **Autoimmune diseases:**
        - Sjogren's syndrome:
            - **Sjögren's syndrome (**"Sicca syndrome") is an autoimmune disorder in which immune cells attack and destroy the exocrine glands[2] that produce tears and saliva.
            - The hallmark symptoms of the disorder are dry mouth and dry eyes
        - Systemic lupus erythematosus and
        - Paraneoplastic syndrome.
    4. **Diabetes,** most common cause of autonomic neuropathy,
    5. **Multiple system atrophy,** ;
        - A degenerative disorder that leads to loss and malfunction of some portions of the central nervous system.

- MSA is associated with the degeneration of nerve cells in specific areas of the brain. This cell degeneration causes problems with movement, balance and other autonomic functions of the body such as bladder control.
- The cause of MSA is unknown
- When autonomic failure predominates, the term **Shy-Drager syndrome** is sometimes used

6. **Injury to nerves** caused by surgery or trauma.
7. **Medications** used in cancer chemotherapy and anticholinergic drugs, sometimes used to treat irritable bowel syndrome and overactive bladder.
8. Parkinson's disease and HIV/AIDS.
9. Riley-Day syndrome is an autosomal recessive disorder seen predominantly in Jews of eastern European descent. Patients present with sensory and autonomic disturbances. Newborns have absent or weak suck reflex, hypotonia and hypothermia. Retarded physical development, poor temperature and motor in coordination are seen in early childhood. Other features include reduced or absent tears, depressed deep tendon reflexes, absent corneal reflex, postural hypotension and relative indifference to pain. Scoliosis is frequent. Intelligence remains normal. Many patients die in infancy and childhood. Lack of flare with intradermal histamine is seen. Histopathology of peripheral nerve shows reduced number of myelinated and non-myelinated axons. The catecholamine endings are absent.

6. Diagnosis:
    a. Tilt table testing may be done to check blood pressure and heart rate responses to changes in position
    b. Valsalva maneuver with Electrocardiography to determine the heart rate changes
    c. Sweat testing: stimulated by electrodes filled with acetylcholine or stimulated in hot room; volume of sweat is measured;

Neurovascular system

The following may be tested by palpation of the pulses and use of appropriate instruments:

- Brachial plexus and bilateral blood pressures
- Cranial and peripheral pulses
- Arterial bruits

Neurocutaneous system

1. Several neurologic conditions have telltale cutaneous stigmata.
2. Evaluation for the following can provide valuable diagnostic clues:
    a. Loss of skin pigmentation as in vitiligo,
    b. White hair-log in Vogt-Harada-Koyanagi disease,
    c. Cutaneous tumors or ash-leaf spots in tuberous sclerosis, and

d. Cutaneous eruptions over a dermatome which may signify herpes zoster.
e. Coffee-brown pigmented (ie, caf é; au lait) spots of varying sizes, usually greater than 1.5 cm in diameter, and axillary freckling are seen in neurofibromatosis. These are observed in addition to or in the absence of the characteristic blubbery subcutaneous tumors that give the condition its name.
f. Tufts of hair (satyr's tail), dimples, and large moles along the spine may indicate spina bifida occulta or diastematomyelia of the spinal column.

Skeletal system - Cranium, spine, bones, joints

1. Palpation of the skull can reveal congenital anomalies that may indicate underlying abnormalities of the brain. In cephaloplegia, one half of the skull may be smaller than the other, possibly signifying asymmetric brain development.
2. Microcephaly or macrocephaly may be detected by measuring the circumference of the head. Observation of the spine may reveal the presence of myelomeningocele, scoliosis, and/or kyphosis.
3. In cases of prenatal brain injuries, the length of the long bones may be reduced on the side opposite the cephaloplegia.
4. Trophic changes in the joints can be associated with denervation in tabes dorsalis or Charcot-Marie-Tooth (CMT) disease.
5. The distal muscular atrophy seen in CMT disease gives the legs the appearance of inverted champagne bottles.
6. Muscular atrophy seen in the region of the temporalis muscles and facial musculature associated with frontal balding is typical of myotonic dystrophy.
7. Pes cavus deformity can be associated with spina bifida and other spinal dysraphisms. A young person with mental retardation, genu valgum, pes cavus, and stroke may have homocystinuria, an inborn error of metabolism typically associated with mental retardation (usually severe) and intimal thickening and necrosis of the media of blood vessels, resulting in strokes and coronary artery disease.
8. Pes cavus deformity can be associated with many conditions including spina bifida, other spinal dysraphisms, and homocystinuria.

Neurological localization

1. Is the lesion inside or outside the central nervous system? : The following symptoms are seen only if there is a lesion in the CNS:
    a. Aphasia
    b. Homonymous hemianopia
    c. Alternating hemiplegia: Cranial nerve signs on one side of the body and sensorimotor deficits on the other side of the body.
    d. Sign of Babinski
    e. Spasticity
2. If the lesion is in the CNS, is it in the spinal cord or in the brain?: Signs pointing to a lesion of the spinal cord include:
    a. Atrophy of muscles innervated by the spinal nerves;
    b. "fasciculations" and "fibrillations."
3. If the lesion is above the level of the cord, is it in the brainstem (subtentorial) or is it in the forebrain (supratentorial = cerebral hemisphere, thalamus and hypothalamus)?

    Signs of a brainstem locus:
        a) Ipsilateral Cranial nerve signs
        b) Contralateral hemiplegia

    Signs of a forebrain lesion:
        a) Aphasia
        b) Visual field defect
4. If the lesion is in the brainstem, is it in the medulla, pons, or midbrain?
    a. Medulla: bulbar palsy
    b. Pons:
        a) CN 6 or 7
        b) Horizontal gaze palsy or INO
        c) Unilateral facial numbness from CN 5
        d) Unilateral deafness
    c. Midbrain:
        a) CN III: ptosis, dilated pupil,
        b) eye down and out
        c) vertical gaze palsy
5. Cerebral cortex:
    a. Frontal lobe:
        a) Incontinence
        b) Impaired smell
        c) Contralateral hemiparesis

d) Frontal release signs1

b. Parietal dominant:

a) Dysphasia

b) Acalculia

c) Dyslexia

d) Apraxia

e) Agnosia - Inability to recognise familiar objects, e.g. faces.

c. Parietal non dominant

a) Neglect of contralateral side

b) Spatial disorientation

d. Temporal

a) Receptive aphasia

b) Dyslexia

c) Impaired verbal memory

e. Occipital

a) Cortical blindness

Melingering or functional deficits: Clinical Clues to melingering

SYNDROME	***CLUES***
Pseudoparalysis	Drop test: Arm paresis Hold paretic hand above face and drop it. If hand misses face, paresis is nonorganic Hoover test for Leg paresis: Cup heels and have patient press down with paretic limb. Then have patient raise opposite limb. True paresis if no difference in downward pressure at heels Adductor sign: Leg paresis Ask patient to abduct paretic leg to resistance. In true paresis, opposite leg should abduct.
Pseudosensory syndromes	Tuning fork test: Vibratory loss on one half of the skull, sternum or pelvis is thought to be physiologically impossible because of bone conduction. The presence of vibratory loss over these surfaces suggests a pseudosensory syndrome. Bowlus and Currier test: In this test, the patient's arms are extended and crossed with thumbs down and palms facing together. The fingers are then interlocked and the hands rotated downward, inward and up in front of the chest. The fingertips end up on the same side of the body as their respective arms. The thumbs are not interlocked so that they lie on the side opposite the fingers. With true sensory impairment, a patient can quickly identify fingers with normal and abnormal sensation when rapid sharp tactile stimuli are applied.

	Patients with pseudosensory deficits confuse the lateralization, causing them to make many mistakes identifying digits when sharp stimuli are applied to them.
	Yes-no test: When testing the sensation of touch, have patients close their eyes and give "yes" responses when they perceive they are being touched and "no" responses when they perceive that they do not feel a touch. A repeated "no" response when a supposedly numb limb is touched favors a pseudosensory syndrome.
	Big toe. Patients with pseudosensory syndromes may erroneously identify the position of the big toe 100 percent of the time. In contrast, a rate of at least 50 percent accuracy would be anticipated with an organic lesion based purely on chance.
Pseudoseizure	No lateral tongue biting
	No postictal confusion
	Pelvic thrusting
	Geotropic eye movements
	Normal "ictal" EEG
	Normal serum prolactin level
Pseudocoma	semipurposeful avoiding movements; normal pupils, corneal reflexes, plantar reflexes and sphincters Normal caloric-provoked nystagmus
Pseudoblindness	Normal pupils and OKN
Pseudodiplopia	Monocular diplopia
Pseudoptosis	Eyebrow depression
Hysterical aphonia	Pressured whispering
	Normal cough
	Normal laryngoscopy

Neurological signs:

Anosognosia

1. This refers to denial of illness and typically is seen in patients with right frontoparietal lesions, resulting in left hemiplegia that the patient denies.
2. A form of visual anosognosia (Anton syndrome) is seen in patients with bilateral occipital lobe infarctions; these patients with double hemianopsia (bilateral cortical blindness) deny that they are blind.

Beevor sign

This is seen with bilateral lower abdominal paralysis that results in upward deviation of the umbilicus when the patient tries to raise his head and sit up from the supine, recumbent position.

Benediction hand: This is seen with lesions of the median nerve in the axilla and upper arm. When present, the index finger remains straight and the middle finger partially flexes when the patient tries to make a fist (assuming the position of the hand of a clergyman while saying the benediction).

Bielschowsky sign

This refers to increasing separation of the images seen when a patient's head is tilted toward the side of a superior oblique (trochlear nerve) paralysis. This sign by itself is not diagnostic and should be used only as a supplement to other tests in suspected CN IV paralysis.

Chvostek sign

This is seen in hypocalcemia. Tapping the cheek at the angle of the jaw precipitates tetanic facial contractions.

Cogan sign

This is seen in myasthenia gravis. It refers to transient baring of the sclerae above the cornea as the patient resumes the primary eye position after looking down.

Dalrymple sign

This refers to the upper-lid retraction seen in thyroid ophthalmopathy.

Doll's-eye maneuver

This refers to turning the head passively with the patient awake and fixated or when the patient is in a coma. In the former, the eyes remain fixated at the original focus when all gaze pathways are normal; in the latter, the eyes deviate in the opposite direction when the brainstem is intact.

Gower sign

This sign, seen in severe myopathies, occurs when the patient attempts to stand up from the floor. Patients first sit up, then assume a quadrupedic position, and then climb up their own legs by using their arms to push themselves up.

Heterochromia iridis

This term refers to the difference in color of the 2 irides. It indicates early injury to the sympathetic system. Ipsilateral to the injury the iris is blue or green, while the contralateral iris is darker.

Jaw jerk

This is elicited by placing the examiner's index finger on the patient's lower jaw and then striking it with the reflex hammer. An exaggerated reflex indicates the presence of a pontine lesion. When the rest of the examination findings are normal, it may indicate physiologic hyperreflexia.

Kayser-Fleischer ring

This is a brownish ring around the limbus of the cornea. It is best demonstrated during an ophthalmologic slitlamp examination.

Lhermitte sign

This refers to the sensation of electricity associated with cervical spinal cord lesions during passive or active flexion and extension of the neck. Once considered pathognomonic of multiple sclerosis, it simply is the result of electricity generation by the hypersensitive, demyelinated, or injured spinal cord; this sign can be associated with any lesion in or around the cord.

Milkmaid's grip

This refers to the inability to maintain a sustained grip commonly seen in patients with chorea.

Myerson sign

Patients with Parkinson disease, particularly those with bilateral frontal lobe dysfunction, continue to blink with repeated glabellar taps.

Ondine curse

This refers to the failure of autonomic control of breathing when the patient falls asleep.

Oommen sign : Have the patient close the eyes and place a pebble the size of an M&M candy on the palm of the examiner's left hand. Cross the patient's middle finger over the index finger on its dorsal aspect. With the examiner's right hand, hold the patient's crossed fingers and have the patient's 2 (crossed) fingertips touch the pebble at the same time. Ask the patient how many pebbles are in the examiner's hand. With normal stereognosis, the patient should answer that there are 2 pebbles. In cases of astereognosis, the patient reports feeling only 1 pebble.

Opsoclonus

This refers to large-amplitude saccadic oscillations of the eyes in all directions, often exacerbated by refixation. They persist during sleep and are associated with brainstem and cerebellar lesions as well as a remote effect of certain carcinomas.

Optokinetic nystagmus

This is elicited by using a rotating, striped drum or a moving, striped piece of cloth. As the patient's eyes fixate on a stripe, nystagmus seen in healthy individuals is due to the optokinetic reflex. Lesions in the anterior aspects of the visual pathways decrease the response, and lesions of the vestibular system result in a directional preponderance to the elicited nystagmus.

Phalen sign

This refers to the aggravation of paresthesia and pain when the wrist is held in flexion (in patients with carpal tunnel syndrome).

Roger sign

This is numbness of the chin in patients with lymphoreticular (and other types of) malignancies.

Stellwag sign

This refers to decreased blinking frequency seen in thyroid ophthalmopathy.

Summerskill sign

This refers to the bilateral upper- and lower-lid retraction associated with severe liver disease.

Tinel sign

This refers to the tingling sensation elicited by tapping along the path of a regenerating nerve following injury. It helps to delineate the extent of nerve regeneration. The Tinel sign also can be observed in tardy ulnar palsy (palpation at the elbow) and carpal tunnel syndrome (tapping at the wrist).

Trendelenburg sign

This refers to the pelvic tilt toward the side of the unaffected raised leg when walking in patients with lesions of the superior gluteal nerve.

Trombone tongue

This is seen in patients with chorea. It refers to the unsteadiness of the tongue when the patient tries to protrude it outside the mouth.

Tullio phenomenon

This refers to the induction of vertigo and nystagmus with acoustic stimuli in patients with labyrinthine disease.

Von Graefe sign

This refers to the lid lag on down gaze in patients with thyroid ophthalmopathy.

Definition of Terms

Apoplexy - Stroke

Cataplexy - Sudden fall, usually due to loss of muscle tone; may be precipitated by sudden changes in affect or mood in narcolepsy (see definition of Narcolepsy)

Cerebritis - Inflammation of the cerebral hemispheres

Encephalitis - Inflammation of the brain and brainstem structures

Encephalopathy - Dysfunction of the brain

Mononeuropathy - Dysfunction of individual nerves

Mononeuritis multiplex - Dysfunction of multiple single nerves

Myelitis - Inflammation of the spinal cord

Myelopathy - Dysfunction of the spinal cord

Myopathy - Primary muscle disease

Myositis - Inflammation of the muscles

Narcolepsy - Sudden attacks manifesting as an uncontrollable urge to sleep

Neuronopathy - Dysfunction of the cortical, cranial, or spinal neurons

Neuropathy - Dysfunction of the cranial or spinal nerves

Polyneuropathy - Bilateral symmetric ascending (stocking and glove) or descending dysfunction of the peripheral nerves

Radiculopathy - Dysfunction of the nerve roots

Seizure - Subjective or objective behavioral manifestation of an abnormal and excessive electrical discharge in the CNS

Stroke - Sudden onset of a neurological deficit, also known as a cerebrovascular accident

EXAMINATION OF ABDOMEN

Symptoms:

1. Vomiting:

1. Neonate:
    i. Physiological chalazia
    ii. Oesophageal Web
    iii. Intestinal atresia
    iv. Imperforate anus
    v. Meconium ileus or plug;
    vi. Hirschsprung's disease
    vii. Inborn errors: tyrosinemia, organic acidemias, urea cycle defects, fatty acid oxidation defects, and galactosemia.
    viii. Adrenogenital syndrome (21-hydroxylase deficiency.)
2. Infants:
    i. Pyloric stenosis
    ii. Malrotation
    iii. Intussusception
    iv. Milk protein allergy
    v. Gasritis
    vi. gastroenteritis
3. Older children:
    i. Cyclic vomiting: recurring attacks of intense nausea, vomiting and sometimes abdominal pain and/or headaches or migraines. Cyclic vomiting usually develops during childhood usually ages 3–7; although it often remits during adolescence, it can persist into adult life. There is a strong suggestion of maternal inheritance, especially when the family history is significant for a mother with migraines.
    ii. Worm infestation
    iii. Hepatitis
    iv. UTI
    v. CNS causes

2. Rumination: regurgitation food into mouth for re chewing
3. Bulimia: binge eating. These cycles often involve rapid and out-of-control eating, followed by self-induced vomiting or other forms of purging.
4. Hiccups: contraction of the diaphragm that repeats several times per minute. In humans, the abrupt rush of air into the lungs causes the epiglottis to close, creating a "hic" sound. Injury or irritation to the phrenic and vagus nerves, as well as toxic or metabolic disorders affecting the aforementioned systems. Hiccups often

occur after consuming carbonated beverages, alcohol, or spicy foods. Prolonged laughter or eating too fast is also known to cause hiccups.

5. Dysphagia: difficulty in swallowing; Causes:
    1. cerebral palsy;
    2. bulbar nerve involvement;
    3. myopathy;
    4. Plummer Vinson syndrome, a triad of dysphagia (due to oesophageal webs), glossitis, and iron deficiency anemia.
    5. foreign body
    6. Globus is psychological dysphagia.
6. Diarrhoea: acute& chronic infections , IBS, malabsorption and allergic and inflammatory causes
7. Constipation: passage of hard stools with difficulty; causes:
    1. Megacolon,
    2. Drugs,
    3. Anal stenosis,
    4. Mental retardation,
    5. Cretinism ,
    6. Dehydration
    7. Poor toilet training
8. Abdominal pain:
    1. GIT: infection; infestation, acid peptic disease, constipation, appendicitis, inflammatory bowel disease , hepatitis, Cholecystitis, pancreatitis etc
    2. CNS: abdominal migraine
    3. RS: Lower lobe pneumonia
    4. GUT: UTI, obstructive uropathy, nephrolithiasis
    5. Peritoneum: peritonitis, mesenteric adenitis
9. Haematemesis: fresh blood; Malaenemisis: altered blood
    1. Swallowed maternal blood syndrome (Apt test)
    2. Haemorrhagic disease of newborn
    3. Curling ulcer (birth asphyxia; burns)
    4. DIVC
    5. Mallory-Weiss syndrome: oesophageal erosion due to repeated vomiting
    6. Acid peptic disease; NASID drugs
    7. Oesophageal varices
10. Malaena: altered blood per rectum; causes as above
11. Haematochezia: fresh blood
    1. Necrotizing enterocolitis

2. Intussusception- red current gelly
3. Rectal polyp
4. Bacterial dysentery

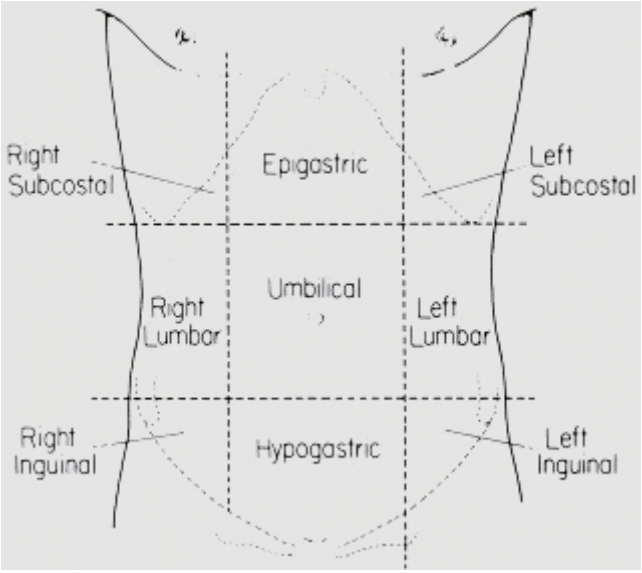
Inspection:

1. General inspection:
    1. Pallor: GI bleed
    2. Jaundice: hepatitis
    3. Hyperpigmentation:
        i. Wilson- increasesd tyrosinase activity and melanin synthesis
        ii. **Peutz–Jeghers syndrome**, also known as **hereditary intestinal polyposis syndrome**, is an autosomal dominant genetic disease characterized by the development of benign hamartomatous polyps in the gastrointestinal tract and hyperpigmented macules on the lips and oral mucosa.
    4. Purpura: hypersplenism
    5. Edema: hypoproteinemia
    6. Telangiactasia: Cirrhosis
    7. Scratch marks: cholestasis
    8. Gynaecomastia: cirrhosis
    9. Kayser-Fleischer ring: Wilson
    10. Catract: Wilson
    11. Perioral rash: Acrodermatitis enteropathica
    12. Palmar erythema: Cirrhosis
    13. Spider naevi: cirrhosis
    14. Asterixis: hepatic encephalopathy
2. Distension:
    1. generalized: obesity; bowel distension by gas or liquid; ascites
    2. localized:
        i. Upper half: pancreatic cyst; tumour; acute gastric dilatation.
        ii. Lower half: pregnancy, ovarian tumour, uterine fibroids, or bladder distension.
3. A scaphoid abdomen is due to malnutrition.
4. Abscence of abdominal muscles: prune belly syndrome
5. Umbilical hernia: Down and cretin
6. Visible peristalsis: VGP and VIP
7. Skin abnormalities:
    1. Bruising: trauma

2. Cullen's and Grey Turner's signs (bluish discoloration of the umbilicus and flanks, respectively) due to intra-abdominal and retroperitoneal bleeding as in severe pancreatitis, trauma, or ruptured ectopic pregnancy.

8. Striae: rupture of the reticular dermis that occurs with stretching: pregnancy, obesity, ascites, abdominal carcinomatosis, and Cushing's syndrome.
9. Surgical scars:
    1. transverse right lower quadrant incision: appendectomy,
10. Enlarged veins:
    1. Emaciation, portal hypertension, and inferior vena caval obstruction.
    2. portal hypertension:
        i. The umbilical vein becomes an outflow tract of the portal system and forms collaterals with the veins of the abdominal wall. This is responsible for the caput medusa that is diagnostic of portal hypertension. The direction of blood flow in these veins in portal hypertension is upward in those above the umbilicus and downward in those below
        ii. The veins of the abdominal wall may be dilated due to obstruction of the inferior vena cava. The direction of blood flow will be reversed below the umbilicus as the blood flows from the femoral vein to the superior vena cava. Obstruction of the inferior vena cava can occur in hepatic malignancy, hepatic vein obstruction (Budd–Chiari syndrome), thrombophlebitis.
11. **Masses**: A mass of the abdominal wall will become more prominent with tensing of the abdominal wall musculature, whereas an intra-abdominal mass will become less prominent or disappear.
12. Looks for flanks for any bulging.
13. spider angiomas

Anatomical areas of the anterior abdominal wall.



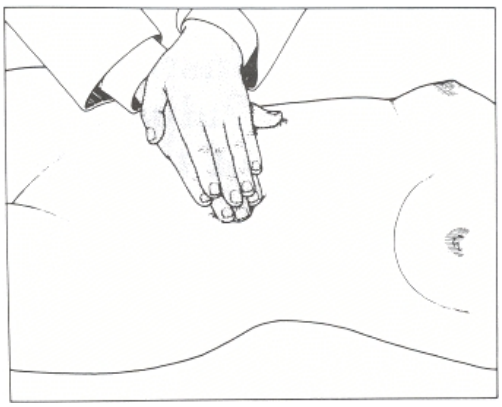
Auscultation:

1. Auscultataion should precede palpation as palpation may stimulate bowel activity and thus falsely increase bowel sounds if performed before auscultation
2. The stethoscope is used to listen over several areas of the abdomen for several minutes for the presence of bowel sounds.
3. Auscultation for abdominal bruits is the next phase of abdominal examination. Bruits are "swishing" sounds heard over major arteries during systole or, less commonly, systole and diastole. The area over the aorta, both renal arteries and the iliac arteries should be examined carefully for bruits.
4. Rubs are infrequently found on abdominal examination but can occur over the liver, spleen, or an abdominal mass.
5. Borborygmi: intestinal sounds increased in enteritis; decreased in peritonitis; ileus.
6. Succussion flash: air and fluid in bowel producing a splashing sound on shaking

Palpation and Percussion;

1. Light palpation:
    a. Gently examine the abdominal wall with the fingertips. This will demonstrate the crunching feeling of crepitus of the abdominal wall, a sign of gas or fluid within the subcutaneous tissues.
    b. When abdominal masses are palpated, decide whether the mass is intra-abdominal or within the abdominal wall. This can be determined by having the patient raise his or her head or feet from the examining table. This will tense the abdominal muscles, thus shielding an intra-abdominal mass while making an abdominal wall mass more prominent.
2. Two-handed deep palpation.
    a. Deep palpation of the abdomen is performed by placing the flat of the hand on the abdominal wall and applying firm, steady pressure. It may be helpful to use two-handed palpation, particularly in evaluating a mass. Here the upper hand is used to exert pressure, while the lower hand is used to feel. One should start deep palpation in the quadrant directly opposite any area of pain and carefully examine each quadrant. At each costal margin it is helpful to have the patient inspire deeply to aid in palpation of the liver, gallbladder, and spleen.

Two-handed deep palpation.



3. In the flanks it is often helpful to elevate the flank to be examined slightly and place one hand on the lower ribs of that flank to "push" the retroperitoneal contents up to the examining hand. In this way, small renal masses that would otherwise be missed may be appreciated.
4. **Tenderness**:
    a. Abdominal tenderness is the objective expression of pain from palpation. When elicited, it should be described as to its location (quadrant), depth of palpation required to elicit it (superficial or deep), and the patient's response (mild or severe).
    b. As tenderness is caused by inflammation of the parietal peritoneum, the etiology of tenderness can be related to the underlying organs.
        i. Right upper quadrant:
            1. cholecystitis,
            2. ulcer disease,
            3. pancreatitis,
            4. Hepatitis.
        ii. Epigastric:
            1. pancreatitis
            2. Peptic ulcer disease.
        iii. Right lower quadrant:
            1. appendicitis
            2. cecal diverticulitis
            3. perforated carcinoma
        iv. Left lower quadrant:
            1. Sigmoid diverticulitis.
        v. Flank :
            1. Pyelonephritis
            2. Perinephric abscess.
        vi. Generalized:
            1. Generalized peritonitis:
                a. Acute perforated ulcer
                b. perforated diverticulitis,
                c. perforated appendicitis, and
                d. Pancreatitis.
5. Spasm or rigidity is the involuntary tightening of the abdominal musculature that occurs in response to underlying inflammation.
6. Guarding, in contrast, is a voluntary contraction of the abdominal wall musculature to avoid pain. Thus, guarding tends to be generalized over the entire abdomen, whereas rigidity involves only the inflamed

area. Guarding can often be overcome by having the patient purposely relax the muscles; rigidity cannot be. Rigidity is thus a clear-cut sign of peritoneal inflammation.

7. Rebound tenderness is the elicitation of tenderness by rapidly removing the examining hand.
8. Palpation of the left lower quadrant may produce tenderness and rebound tenderness in the right lower quadrant in appendicitis (Rovsing's sign).
9. Ascites:
    a. A rounded, symmetrical contour of the abdomen with bulging flanks is often the first clue.
    b. Palpation of the abdomen in the patient with ascites will often demonstrate a doughy, almost fluctuant sensation.
    c. In the supine patient with ascites there should be periumbilical tympany with dullness in the flanks.
    d. A change in the level of dullness is termed shifting dullness and usually indicates more than 500 ml of ascitic fluid.
    e. Another physical sign of ascites is demonstration of a transmitted fluid wave. The patient or an assistant presses a hand firmly against the abdominal wall in the umbilical region. The examiner places the flat of the left hand on the right flank and then taps the left flank with his right hand. In the presence of ascites, a sharp tap will generate a pressure wave that will be transmitted to the left hand.
    f. In addition to detection of ascites, percussion can be used to help define the nature of an abdominal mass.
    g. Tympany of an abdominal mass implies that it is gas filled (i.e., intestine). Percussion is also used to define liver size.
    h. **Puddle sign**: for > 120 mL; shifting dullness and bulging flanks typically require 500 mL.
    The steps are outlined as follows:
        1. Patient lies prone for 5 minutes
        2. Patient then rises to knee chest position
        3. Apply stethoscope diaphragm to most dependent abdomen
        4. Flick near flank with finger. Continue to flick at same spot on abdomen
        5. Move stethoscope across abdomen away from examiner
        6. Sound loudness increases at farther edge of puddle
        7. Sound transmission does not change when patient sits
        8. In relation to auscultatory percussion, the puddle sign is more specific, but less sensitive.
        9. Percuss from flank to centre of abdomen; dullness over the puddle
    i. **Causes**:
        i. Exudative ascites: protein concentration of over 3 gm/dl and a specific gravity above 1.016.

1. occurs in bacterial peritonitis,
2. carcinomatosis, and
3. pancreatic ascites

ii. Transudative ascites: protein concentration is less than 3 gm/dl and the specific gravity less than 1.016.

1. cirrhosis,
2. Budd–Chiari syndrome,
3. constrictive pericarditis,
4. congestive heart failure, and
5. Nephrotic syndrome.

10. Abdominal masses: consider its location, mobility, and the presence or absence of tenderness in order to define its aetiology.

1. a mass in the right upper quadrant:
    a. Hepatomegaly from hepatitis or hepatic tumour,
    b. A distended gallbladder from Cholecystitis or
    c. Pancreatic cancer or a carcinoma in the head of the pancreas.
2. An epigastric mass:
    a. Acute gastric distension.
    b. pancreatic pseudocyst,
    c. pancreatic cancer, or
    d. Aneurysm of the abdominal aorta (which will be pulsatile).
3. Mass left subcostal region:
    a. splenomegaly,
    b. carcinoma of the splenic flexure of the colon
4. flanks:
    a. kidney (cyst or tumour),
    b. lymphoma, sarcoma
5. Lower quadrants: usually arise from the bowel.
    a. right side:
        i. Appendiceal abscess and
        ii. Cecal carcinoma;
    b. left;
        i. Diverticular abscess and
        ii. Sigmoid carcinoma.
6. Central abdominal masses:
    a. Aortic aneurysms and the pulsatile nature of the mass is diagnostic.

11. Liver:

a. liver size:
    i. Increases with increasing age, averaging 5 cm span at 5 years and attaining adult size by age 15. By percussion, the mean liver size is 7 cm for women and 10.5 cm for men. A liver span 2 to 3 cm larger or smaller than these values is considered abnormal. The liver weighs 1200 to 1400 g in the adult woman and 1400 to 1500 g in the adult man.
    ii. The normal liver is smooth, with no irregularities.

b. Causes of palpable liver:
    i. increased diaphragmatic descent;
    ii. presence of a palpable caudate or Riedel's lobe;
    iii. presence of emphysema with an associated depressed diaphragm;
    iv. thin body habitus with narrow thoracic cage;
    v. fatty infiltration (enlarged with rounded edge);
    vi. active hepatitis (enlarged and tender);
    vii. cirrhosis (enlarged with nodular irregularity);
    viii. Hepatic neoplasm (enlarged with rock-hard or nodular consistency).

c. Observation:
    i. Protuberant abdomen harbouring ascites may be the first clue to hepatic cirrhosis or malignancy.
    ii. vascular spiders
    iii. caput medusa
    iv. Fullness of right upper quadrant is observed for the appearance at rest or on deep inspiration.

d. Auscultation:
    i. venous hum: portal vein hypertension
    ii. Hepatic bruit: A is indicative of alcoholic hepatitis or primary or metastatic cancer.
    iii. Hepatic Friction Rub:
        1. Infarction;
        2. Inflammation of the liver or contiguous structures, the commonest causes being infection and cancer, either primary or metastatic.
        3. If detected in a young woman, consider gonococcal peritonitis of the upper abdomen (Fitz–Hugh–Curtis syndrome).
        4. A hepatic rub and bruit in the same patient usually indicates cancer in the liver. A hepatic rub, bruit, and abdominal venous hum would suggest that a patient with cirrhosis had developed a hepatoma**.**

e. Palpation: is performed to determine liver shape and consistency. Single-handed palpation is used for lean individuals, while the bimanual technique is best for obese or muscular individuals and for deep palpation. The normal liver may be slightly tender upon palpation, but the inflamed liver

(hepatitis) is often exquisitely tender. The nodularity, irregularity, firmness, and hardness of the liver can be characterized.

f. Percussion :

i. Percussion should first be performed at the right midclavicular line, then at the midsternal and anterior axillary lines.

ii. The normal upper level of the liver is at the level of the right nipple, whereas the lower margin of the liver is at the right costal margin.

iii. **Tidal percussion**:

- Starting at a level below the umbilicus in the right mid-clavicular line, lightly percuss upward toward the liver. Ascertain the lower border of liver dullness.
- Identify the upper border of liver dullness in the mid-clavicular line by lightly percussing from lung resonance down toward liver.

iv. An alternate method of estimating liver size is the **scratch test**.

- Keep the diaphragm of your stethoscope over the liver dullness.
- Start scratching the abdominal wall starting from the right lower quadrant parallel to the liver edge.
- There will be a sudden transition of increased transmission of sound once the liver edge is reached.
- Similar manoeuvres will determine the upper edge.

- **Liver span in the mid-clavicular line is 6-12 cm.**

12. Examination of GALLBLADDER

- orient hand perpendicular to the costal margin feeling from medial to lateral below the right costal margin (feels bulbous, focally rounded mass that moves downward on inspiration)
- **Murphy's sign (cholecystitis)**
- place left hand with forefinger parallel to and at the RLCM and with thumb pressing on the intersection of the costal margin and the lateral border of the abdominal rectus muscle

  + ve sign → pain elicited around the area pressed during a deep inspiration

13. Spleen:

a. General:

i. Lies immediately under the diaphragm in the left upper quadrant of the abdomen. It ranges in length from 6 to 13 cm and in weight from 75 to 120 g.

ii. The spleen is not normally palpable

iii. When the spleen can be felt below the left costal margin, at rest or on inspiration, splenic enlargement should be assumed

iv. The enlarged spleen in itself is not a problem but is an important clue to the presence of a variety of illnesses.

b. Spleen : Percussion:

i. Percussion of **Traube's Space;**

Boundaries -Left anterior axillary line, 6th rib, costal margin; this area should be resonant on percussion; dullness indicates possible splenic enlargement

ii. Percussion by Castell's method

Percuss in the lowest Left intercostal space in the anterior axillary line (usually the 8th or 9th IC space) ; this space should remain resonant during full inspiration; dullness on full inspiration indicates possible splenic enlargement (a positive Castell's sign)

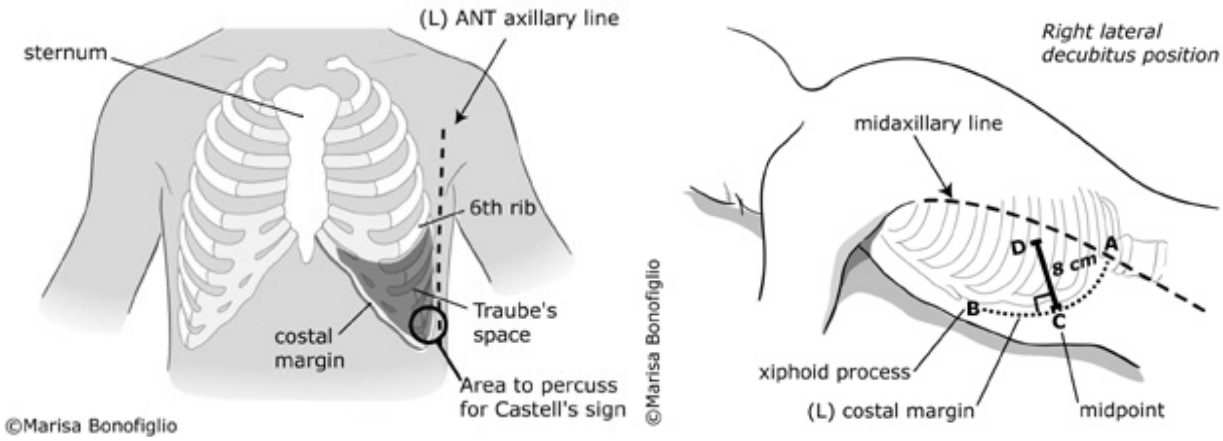
iii. Percussion by **Nixon's method**

Place the patient in Right lateral decubitus

Begin percussion midway along the Left costal margin

Proceed in a line perpendicular to the Left costal margin

If the upper limit of dullness extends >8 cm above the left costal margin, this indicates possible splenomegaly



c. Kidney vs. Spleen

1. Kidney is balottable, spleen is not
2. Notch on anterior border - palpable in spleen, not in kidney
3. Spleen enlarges diagonally towards rt iliac fossa, while the kidney enlarges inferiorly
4. Kidney can be resonant to percussion (d/t overlying bowel), spleen should be dull
5. Upper edge of spleen not palpable, upper edge of kidney is
6. SPLENIC RUB on auscultation (have patient breath in and out)

Causes of splenomegaly:

1. **Vascular congestion:**
   a. Cirrhosis
   b. Splenic vein thrombosis
   c. Portal vein thrombosis
2. **Reticuloendothelial hyperplasia:**

a. Acute infections:
    i. typhoid fever,
    ii. cytomegalovirus,
    iii. Epstein-Barr virus infection
b. Subacute or chronic infections:
    i. bacterial endocarditis,
    ii. brucellosis,
    iii. tuberculosis,
    iv. histoplasmosis,
    v. Malaria
c. Collagen-vascular diseases and abnormal immune responses
    i. systemic lupus erythematosus,
    ii. serum sickness,
    iii. sarcoidosis

3. **Work hypertrophy:**
    a. Hemolytic anemias (e.g., spherocytosis)
4. **Infiltrative or replacement processes:**
        i. Nonmalignant hematologic disorders:
            1. polycythemia vera,
            2. myelofibrosis
        ii. Leukemias
        iii. Lymphomas
        iv. Metastatic solid tumors
        v. Storage diseases (e.g., Gaucher's disease)
        vi. Amyloidosis
        vii. Benign tumor and cysts
        viii. Abscess
        ix. Subcapsular hemorrhage

HEPATOSPLENOMEGALY:

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1. Infective:
    a. Viral:
        i. Hepatotrophic (A,B,C,D,E)
        ii. Other viruses (herpes, cytomegalo, Ebstein-Barr, varicella, HIV, rubella, adenovirus, enterovirus, arbovirus
    b. Protozoal:
        i. malaria,
        ii. kalazar,

iii. amoebic,
iv. toxoplasma

c. Bacterial:
i. sepsis;
ii. tuberculosis,
iii. brucellosis,
iv. syphilis

d. Helminths:
i. hydatid,
ii. visceral larva migrans

e. Fungal: histoplasmosis

2. Hematological:
a. Haemolytic:
i. haemolytic disease of newborn,
ii. thalassaemia

3. Metabolic:
a. Nieman-Pick,
b. Gaucher,
c. gangliosidosis,
d. mucopolysaccharidosis,
e. glycogen storage

4. Malignancies:
a. Leukemia, histiocytic syndromes, myeloproliferative syndromes, lymphomas

5. Immunological:
a. Chronic granulomatous disease, hereditary neutrophilia, Ommen syndrome.

6. Developmental:
Congenital hepatic fibrosis

7. Congestive:
a. Hepatic vein obstruction,
b. constrictive pericarditis

8. Investigations: According to the case, may include
a. CBC, retics
b. Blood film
c. Malaria smear
d. LFT, PT, Ammonia
e. Hepatitis tests
f. Monospot test

g. Mantoux
h. TORCH
i. α-fetoprotein
j. Bone Marrow Aspiration
k. Urinalysis
l. Ultrasound
m. CT scan
n. Angiography
o. Needle aspiration

- Hepatosplenomegaly With jaundice
    1. Hemolytic disease
    2. Neonatal hepatitis
    3. Viral hepatitis
    4. Galactosemia
    5. Biliary atresia
- With anaemia
    1. Hemolytic anemia
    2. Iron deficiency anemia
    3. Leukemia
    4. Malaria
- With lymphadenopathy
    1. Infectious mononucleosus
    2. Disseminated tuberculosis
    3. Leukemia
    4. Lymphoma
    5. Letterer- siwe disease: autosomal recessive; proliferation of Langerhan type histiocytes;ofetn fatal
- With fever
    1. Enteric fever
    2. Infectious mononucleosus
    3. Malaria
    4. Kala Azar: Leismania donovani transmitted by sand fly
    5. Leukemia
    6. Collagen disease:
        1. SLE and
        2. Rheumatoid arthritis
- With ascites

1. Cirrhosis
2. TB peritonitis
3. Malignancy: leukemia, lymphoma, Wilm's tumor

- With arthritis
  1. SLE
  2. Rheumatoid arthritis
  3. Sickle cell disease: septic, gout and avascular necrosis
  4. Leukemia
- With rash
  1. Infectious mononucleosus
  2. CMV
  3. Rubella
  4. Hepatitis B
  5. Typhoid
  6. SLE
- With renal swelling
  1. Leukemic infiltration
  2. Beckwith-widermann syndrome; obescity, visceromegaly and macroglossia
  3. Zellweger syndrome: antimangoloid slant, large fontanel, atonia, cirrhosis
- With Cardiomegaly
  1. Haechromatosis: hemosiderin deposited in organs
  2. Cardiomyopathy
  3. SLE
- With malabsorption
  1. Wolman's disease: acid lipase deficiency; accumulation of cholesteryl esters
  2. Whipphle's disease: due to gm positive bacterium Tropheryma whipplii infection;
  3. With mental retardation
  4. Mucopolysaccharidoses: Hurler, Hunter, Morquio
  5. Gangliosidosis: lipid storage disorders
- With fits:
  1. Goucher's disease: lysosomal accumulation of glucocerebroside
  2. Niemann pick: lysosomal accumulation of sphingomyelin in macrophage-histiocytes
- With fits
  1. Galactosemia:G-1-p-uridyl transferase deficiency; accumulation of galactose
  2. Hereditary fructose intolerance- aldolase B deficiency; accumulation of fructose
  3. Farber's disease-ceramidase deficiency and lipid accumulation
  4. Cerebral malaria

SHRI VENKATESWARA MEDICAL COLLGE AND RESEARCH CENTER

ARIYUR, PONDICHERRI

PEDIATRIC CASE RECORD

HISTORY

I. Informant/Reliability of informant:

II. **Patient Profile:**

1. Name:.....Age:.....Sex:.....
2. Family unit: joint / nuclear
3. Father: Education:.....Occupation:.....
4. Mother: Education:.....Occupation:.....
5. Social status: .....
6. Per capita income: Rs .....

III. **Presenting Complaints:**

IV. H/O Present Illness:

V. Past Medical History

A. Antenatal:

B. Natal

C. Neonatal:

D. Growth and Development:

VI: Past Illnesses:

1. Infections:

2. Past Hospitalizations, including surgery:

3. Allergies:

4. Injuries:

VI. Current medications if any: for epilepsy, anemia, ATT etc.

VII: Immunization history:

VIII: Nutrition:

IX: National/state child programs:

1. **Vit. A prophylaxis :**
2. **FST small :**
3. **Deworming :**
4. **Noon Meal : CM / Anganwadi(ICDS)**

iii. Family history:

XI. Behavioural History:

XII. Family history:

1. **Expossure to TB :**
2. **HIV :**
3. **Hereditiory disoreders :**
4. **Any other illness :**

XII. Family tree (Genogram):

XIII. Social History: tribal/nomadic/semi nomadic etc (if appropriate):

XIV. Environmental History:

General Physical Examination

I. Vital Signs and Measurements

Level opf consciousness:

Temperature:.....pulse rate:.....Respiratory rate:.....Blood pressure:.....

Weight:.....BMI:.....Mid arm circumference:.....Head circumference.....

II. General Inspection:

III. C.V.S:

IV. R.S:

V. Abdomen including genitals:

VI. Neurologic Examination

2. Mental status:

a. Recent memory:

b. **Remote memory:**

c. Orientation:

d. Intelligence:

3. Examination of the Cranial Nerves:

4. Cerebellar Function:

5. Motor System:

6. Sensory System:

6. Reflexes:

1. Deep reflexes –

2. Superficial reflexes –

VII: 1.**Particulars of previous Lab Tests**

2. Lab Tests:

TC: DC:

Smear study:

ESR:

Blood: C&s:

Urine:

Stool:

Mx:

CXR:

Other Tests: specify:

VIII: Provisional diagnosis:

VIII: Differential diagnosis:

IX: Final diagnosis:

X: Treatment:

XI: Follow up:

INSTRUMENTS

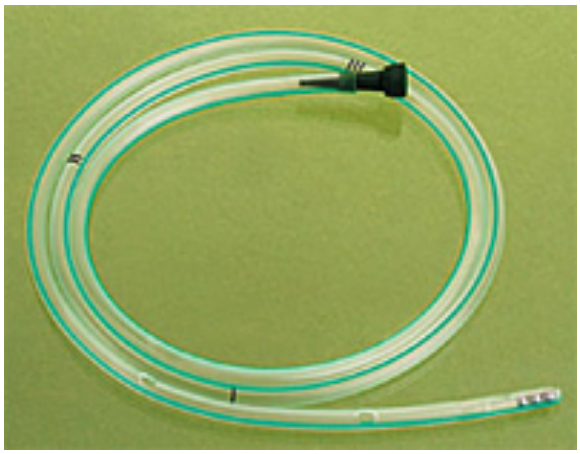
INFANT MUCUS EXTRACTOR

- Specially designed for aspiration of secretion from Oropharynx in newly born babies to ensure free aspiration
- Clear transparent container permits immediate visual examination of the aspirateAlso suitable for obtaining mucus specimen for micro biological examination
- Spare closure cap is provided to seal the container for safe transportation of specimen to the laboratory or aseptic disposal of container
- Low friction surface catheter is provided with open end silk smooth round tip for trauma free insertion
- Capacity 20 to 25ml.,
- Individually packed
- Laminated inner box of 40 pcs
- Disposable, Sterile Ready for use

UMBILICAL CORD CLAMP

- Manufactured from Non toxic Medical grade polymer
- Designed for clamping the Umbilical Cord immediately after birth
- Provided with double purpose security lock click to indicate the correct locking & protect against accidental re-opening
- Finger grip ensure safe and convenient handling, particularly when gloves are wet
- Provided with grooves all along the length to prevent the slipping of the Umbilical Cord and to retain it in the same position
- Individually packed
- Laminated inner box of 100 pcs
- Disposable, Sterile ready for use

RYLE'S TUBE



- Manufactured from non toxic, non irritant PVC
- Specially designed for naso gastric introduction for nutrition and aspiration of intestinal secretion
- Distal end is coned with corrosion resistant stainless steel balls sealed into the tube, to assist the passage of the tubing during intubationFour lateral eyes are provided for efficient aspiration and administration
- 3 openings at the tip
- The tube is marked at 50cm 60 cm & 70cm from the tip for accurate placement
- Super smooth low friction surface facilitates easy intubation
- Provided with X-ray opaque through out the length
- Length : 105 cms
- Sizes: Fg: 8,10,12,14, 16,18 & 20
- Disposable, Sterile ready for use

INFANT FEEDING TUBE

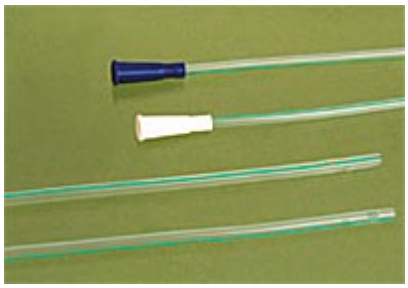
- Manufactured from non toxic PVC non irritant to delicate mucosa
- Suitable for neonates and paediatric nutritional feeding
- Distal end is coned with two lateral eyes
- Proximal end is fitted with female luer mount for easy connection feeding funnel or syringe
- Low friction tubing & super smooth tip ensures trauma free intubation
- Radio –opaque line is provided throughout its length for X-ray visualization
- Length : 52 cm

UMBILICAL CATHETER:

- Manufactured from SOFT PVC white yield easily to tissue contours at body temperature
- Designed for intermittant or continuous access to the umbilical artery or vein of newly born or premature infants
- Individually straight packed in paper pouch packing to ensure aseptic handling
- Laminated inner box of 100 pcs
- Disposable, Sterile ready for use

NASAL OXYGEN CATHETER

- Open distal end with multiple lateral eyes for even dispersion of oxygen and prevention of oxygen burns
- Colour coded for size identification
- Frozen surface
- Length : 40 cms
- Individually packed
- Laminated box of 100 pcs



- Disposable, Sterile Ready for use

OXYGEN MASK MEDI MASK (ADULT & PAEDIATRIC)

- Moulded face mask has adjustable elastic strap and integrated nose clip for proper positioning of mask on the mouth and nasal area
- Specially designed for convenient oxygen therapy
- Provided with 2 meter long star lumen tube to ensure continuous flow of Oxygen
- The tube has funnel shaped connector at the proximal end to facilitate simple & safe connection with the oxygen Tube
- Sizes: Adult, Paediatric

GUEDEL AIRWAYS (PLASTIC)

- Designed to maintain an unobstructed Oro- Pharyngeal airway during or following general anesthesia and in patients who are unconscious for other reasons
- Integral hard bite block avoids airway occlusion and biting of the tongue
- Stepless airway path for easy cleaning
- Bite blocks are colour coded for instant identification of sizes
- Individually packed
- Laminated box of 30 pcs
- Disposable, Sterile Ready for use

SUCTION CATHETER

- Suction catheters are suitable for removal of secretion from mouth oropharynx trachea and bronchial tubes
- Distal eye is open with one lateral eye
- Frozen surface tubing for smooth intubation
- Colour Coded for instant size identification
- Length : 53 cms

Malecot Catheter

Used for percutaneous placement of a malecot catheter in the renal pelvis for nephrostomy drainage.

Key Features:

- The malecot flower design is ideally suited for the drainage of thick viscous fluids
- Highly radiopaque for better visualization

Size: 10 to 18 French

Length: 30 cms

Ambu Bag:

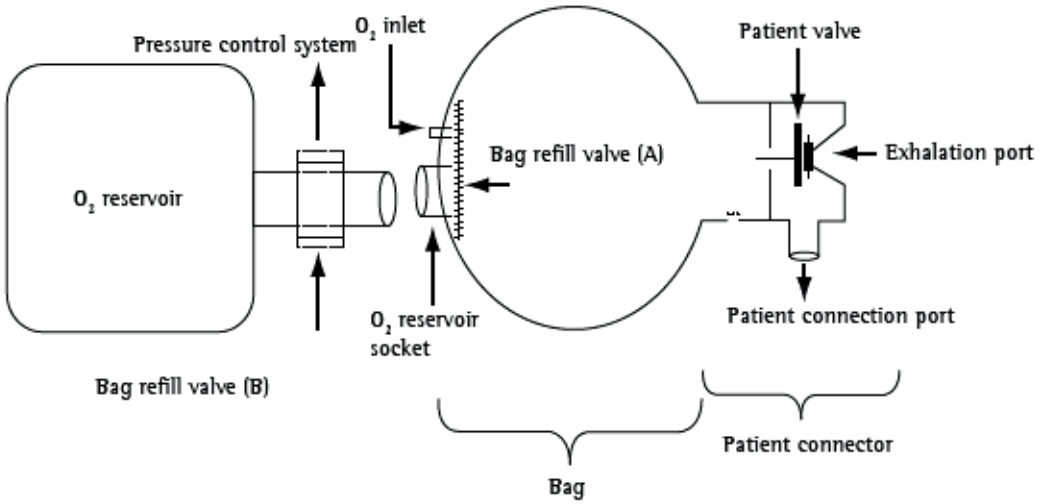


Figure 1 – Basic components of the manually operated self-inflating resuscitation bag and of the oxygen reservoir.

A **bag valve mask** (also known as a **BVM** or **Ambu bag**) is a hand-held device used to provide positive pressure ventilation to a patient who is not breathing or who is breathing inadequately. The device is a normal part of a resuscitation kit for trained professionals, such as ambulancecrew. The BVM is frequently used in hospitals, and is an essential part of a crash cart. The device is used extensively in the operating room to ventilate an anaesthetised patient in the minutes before a mechanical ventilator is attached. The device is self-filling with air, although additionaloxygen (O_2) can be added.

Use of the BVM to ventilate a patient is frequently called "**bagging**" the patient. Bagging is regularly necessary in medical emergencies when the patient's breathing is insufficient (respiratory failure) or has ceased completely (respiratory arrest). The BVM resuscitator is used in order to manually provide mechanical ventilation in preference to mouth-to-mouth resuscitation (either direct or through an adjunct such as a pocket mask).

One proprietary brand of a self-inflating BVM resuscitator is called the Ambu bag. The concept for the original Ambu bag was developed in 1953 by the German engineer, Dr. Holger Hesse, and his partner, Danish anaesthetist Henning Ruben. In 1956, the world's first non-electric, self-inflating resuscitator was ready for production by their company, Ambu A/S, which still produces a wide range of single-patient and multi-use resuscitators. The Ambu name has become an example of a Genericized trademark, as all manual bag resuscitators in medical settings are now often referred to generically as "Ambu bags," even though Ambu brand resuscitator bags are still produced and other companies are not allowed to use the Ambu trademark.

I. Device: Self-inflating bag (Bag-valve mask or Ambu Bag)

A. Mechanism

1. Bag fills spontaneously after being squeezed

B. Advantages

1. Does not require an oxygen source
2. Easier to learn to use

C. Disadvantages

1. Can not deliver free flow oxygen

D. Oxygen Delivery with ventilation (Bag-Valve Mask)

No Oxygen Source: Delivers 21% Oxygen (Room air)

Without Oxygen Reservoir: Delivers 30-80% Oxygen at 10 LPM flow

With Oxygen Reservoir (required for high oxygen flow): Delivers 60-95% Oxygen at 10-15 LPM flow

II. Pop-Off Valves (Bag Valve Mask)

Usually set at 30-45 cm H2O

Pop-off should be easily occluded on bags

Higher pressures are needed during CPR

Occlusion of the pop off valve

Depress valve with finger during ventilation or

Twist the pop-off valve into closed position

III. Precautions

. Do not use Bag Valve Mask to deliver free flow oxygen

IV. Technique

Tidal Volume

Term Newborns

Administer 5-8 ml/kg (15 to 25 ml per ventilation)

Bag volume: 200 to 750 ml (usually >450 ml)

Adults and older children

Administer 10-15 ml/kg

Technique:

Hold mask over face with one hand

Mask should fit snugly

Covers mouth, nose and chin

Should not cover eyes

Thumb over nose

Support jaw with middle or ring finger

Avoid submental pressure (risk of airway obstruction)

Head Tilt - chin lift (Avoid if trauma!): Infants/Toddlers

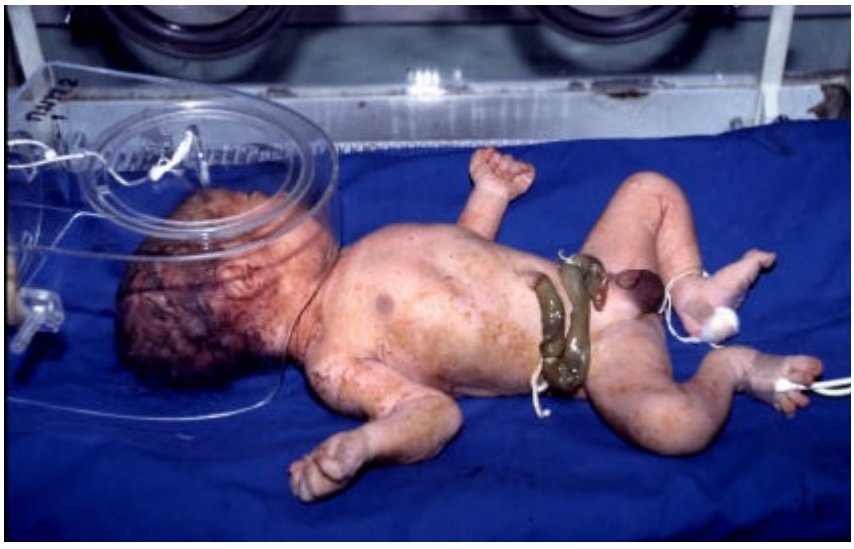
Neutral sniffing position without hyperextension: Children >2yo

Folded towel under neck and head

Observe for adequate ventilation

Adequate chest rise

Oxygen hood



Provides a stable concentration, visibility and access to most of the body ; recommend for acutely ill or unstable infants whorequire a FiO2 > 0.40 ; a minimum flow rate of 3 LPM is recommended in order to prevent CO2retention.

OXYGEN THERAPY.

Oxygen saturation is defined as the ratio of oxyhemoglobin to the total concentration of hemoglobin present in the blood (ie Oxyhemoglobin + reduced hemoglobin).

A hemoglobin molecule can carry a maximum of four oxygen molecules. 1000 haemoglobin molecules can carry a maximum of 4000 oxygen molecules; if they together were carrying 3600 oxygen molecules, then the oxygen saturation level would be (3600/4000)*100 or 90%.

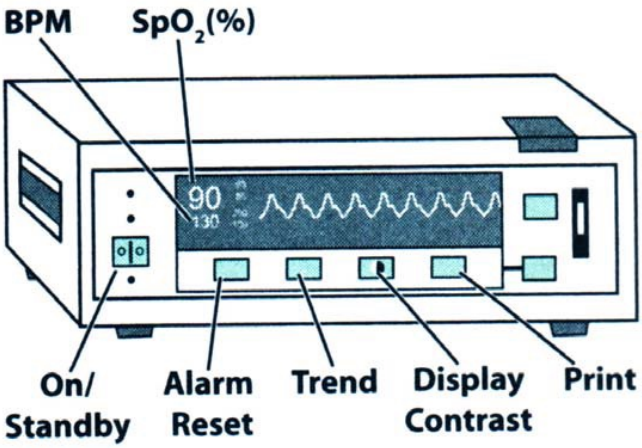
When arterial oxyhemoglobin saturation is measured by an arterial blood gas it is called SaO2.

When arterial oxyhemoglobin saturation is measured non-invasively by pulse oximetry, it is called SpO2

Oxygen therapy-NB

Apparatus	**Flow rate L/Mt**	**% of available Oxygen**
Room Air		21%
Hood	10-12	80-90%
Nasal tube	5-8	Held at distance: .5" : 80% 1" : 60% 2" : 40%
Mask	5-8	Tightly over the face 60-80% Loosely over the face 40%
Ambu Bag	6	Without reservoir 40% With reservoir 80-90%

Pulse oximeter measuring the oxyhemoglobin saturation.



Oxygen is transported from the lungs to tissues by hemoglobin in the red blood cells.

The hemoglobin changes color from blue to red as it carries more oxygen.

This change in color can be measured with a pulse oximeter that is attached to a baby's

Hand or foot.The oximeter gives a reading ranging from 0% to 100% and is useful in determining whether a baby has a satisfactory amount of oxygen in his or her blood.

MODEL CHARTS

A 20 days old male child was admitted for lethargy and poor feeding. With the following test results what would be your diagnosis?

1. Gastric aspirate: 15 polys per HPF
2. TC 4500 / cml
3. Band forms in DC 22 %
4. Micro ESR 25 mm
5. CRP 10 mg/L
6. Thrombocytopenia +
7. Acidosis +

Which blood gas analyses are most indicative of respiratory acidosis?

- pH = 7.22, PCO_2 = 55 mm Hg, HCO_3 = 30 mEq/L
- pH = 7.28, PCO_2 = 45 mm Hg, HCO_3 = 15 mEq/L
- pH = 7.34, PCO_2 = 35 mm Hg, HCO_3 = 25 mEq/L
- pH = 7.40, PCO_2 = 25 mm Hg, HCO_3 = 30 mEq/L

An 18-month-old male patient is brought to the clinic because of a cold. The patient's mother says, "I don't know if his ear hurts or not, but I can't stand it when he holds his head and screams." During the physical examination, the paediatrician notices bruises in various stages of healing on the boy's back, arms, and legs.

What is the probable diagnosis?

A 10-month-old female is brought to your office for a routine health evaluation. Her diet consists of table food and whole milk and she is a “good drinker”. Her weight and length are at the 50th percentile and no changes are noted in her growth curves. Her physical examination is notable for pallor; otherwise there are no abnormalities. Her hemoglobin is 7.5 grams per deciliter and the peripheral blood smear reveals microcytic hypochromic cells.
Which of the following is the MOST likely etiology of this anemia?

A) Thalassemia

B) Sickle cell anemia

C) Transient viral suppression of her bone marrow

D) Anemia of chronic disease

E) Iron deficiency anemia

A 3-year-old boy is seen in the emergency department after coughing and gagging while eating a peanut. He now has difficulty breathing. His physical examination is significant for wheezing on the right side hemithorax. What radiographic finding is classic for this condition?

A) Atelectasis on the side of aspirated content

B) An expiratory film demonstrating air trapping with hyperinflation of the lung shifting away from aspirated side

C) Bilateral hyperexpansion

D) Peanut seen on the radiograph

E) An inspiratory film demonstrating a left lower lobe consolidation

A 10-year-old boy is mentally retarded, but able to carry out activities of daily living, including feeding himself and dressing himself. On physical examination, he has brachycephaly and oblique palpebral fissures with prominent epicanthal folds. On the palm of each hand is seen a transverse crease. Upon auscultation of the chest, there is a loud systolic murmur. Which of the following diseases will he most likely develop by the age of 20?

A ☐ Chronic renal failure

B ☐ Hepatic cirrhosis

C ☐ Acute leukemia

D ☐ Acute myocardial infarction

E ☐ Aortic dissection

A genetic counselor elicits the history that three adult males and one adult female in a family of 10 over 3 generations are mentally retarded, the males more severely so. Physical examination of these affected males reveals no major morphologic anomalies, though their testes appear to be slightly enlarged, without mass lesions present. These males have been healthy, without a history of major illnesses. Which of the following genetic abnormalities is the most likely etiology for these findings?

A ☐ Klinefelter syndrome

B ☐ Gaucher disease

C ☐ Fragile X syndrome

D ☐ Phenylketonuria

E ☐ Trisomy 21

F ☐ XYY karyotype

A 2-week-old-male presents with lethargy and vomiting. His electrolytes reveal sodium of 121 meq/L, potassium of 7.0-meq/l and blood glucose of 40 mg/dl. What is the most likely diagnosis?

a. Dehydration
b. Congenital adrenal hyperplasia
c. Inborn error of metabolism
d. Pyloric stenosis

Vaginal bleeding in a 3-day-old female is:

e. Is always indicative of child abuse
f. May be due to withdrawal of maternal hormones
g. Is suspicious for gonorrhea
h. Is most commonly due to a vaginal foreign body-such as baby wipes

When is an upgoing plantar reflex normal?

i. Never
j. Until 1 month
k. Until 6 months
l. Until 12 months+
m. Until adolescence

You can slightly drop the head of a baby and he responds with arm extension and abduction, followed by arm flexion and a cry. What is this called and in what age groups is it normal?

When does the palmar grasp reflex diminish?

a. 2 months

b. 4 months+

c. 6 months

d. 1 year

A three day-old term infant borne at home, breast- fed exclusively, presented to the ED with lethargy, bulging fontanel, and bright red blood from rectum. What is the most likely etiology of his disease?

1. Fluoride deficiency
2. Ca deficiency
3. Vitamin K deficiency
4. Iron deficiency

In the absence of other concerning associated signs, APNEA is defined as a respiratory pause of greater than:

a. 10 seconds
b. 15 seconds
c. 20 seconds
d. 30 seconds
e. 1 minute

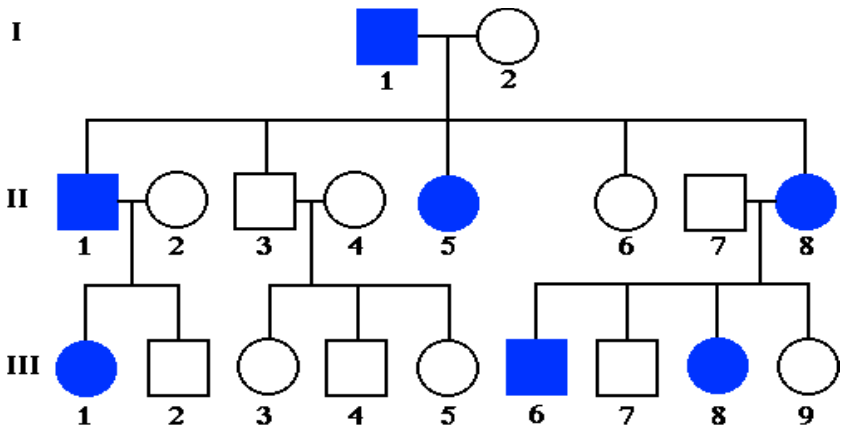
1. 2 year old child had an upper respiratory tract infection with rhinorrhea, pharyngitis, mild cough, and low-grade fever
2. 1 to 3 days later the child then develops "barking" cough, hoarseness, and inspiratory stridor.
3. Symptoms are worse at night
4. Agitation and crying greatly aggravate the symptoms and signs.
5. The child prefers to sit up in bed or be held upright.
    a. What is proble diagnosis?
    b. Mention one important medical management.

Lab Findings:

1. Blood picture:
2. TC: 8200/ ml
3. DC: P 62 L 36 E2
4. Platelet count: 10,000/micro litre
5. Hemoglobin value: 10.4 gm/dl
6. The hemoglobin may be decreased if there have been profuse nosebleeds or menorrhagia.

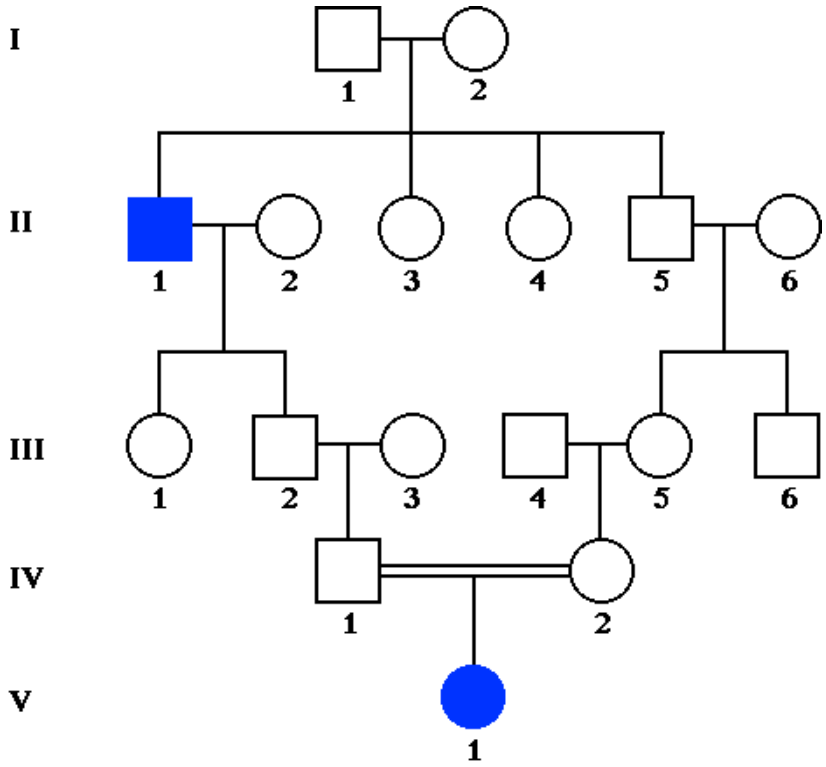
7. The bone marrow examination reveals normal granulocytic and erythrocytic series with increased numbers of megakaryocytes.
    a. What is the probable diagnosis?
    b. Mention any one important mode of treatment.

Comment on the inheritance pattern:



Pedigree 1. An idealized pedigree of a family with hypercholesterolemia, an autosomal dominant disease where the heterozygote has a reduced number of functional low density lipoprotein receptors.

Comment on the inheritance pattern



Comment on the biochemistry and probable diagnois and immediate management of Child A and B based on the following findings:

	pH	**pCO2**	**pO2**	**Bicarb**	**BE**
A	7.40	40	100	24	0
B	7.22	60	70	24	-3

A child with fever for 3 weeks and convulsions since 2 days is investigated. Result of lumbar CSF analysis is as follows. What is the drug management?

1. Leukocytes: 500 cells/dL; predominantly lymphocytes
2. Glucose 54 mg/dL
3. Protein: 400 mg/dL

Lab Findings of 18 mo old female child with puffiness of face since 4 days:

Urine Albumin: +++

Sugar: Nil

Deposits: Pus cells occasional

No RBCs

Epithelial cells 2-3 HPF

CSF findings of 12 mo old child with fever 5 days and convulsions 1 day:

CSF pressure: increased

Colour: opalescent

Cells: 1200 polys/ml

Protein: 180 mg/dl

Glucose: 32 mg / dl

NORMAL BIOCHEMICAL PATTERN IN CHILDREN

BIOCHEMICAL TEST	NORMAL RANGE
TRIGLYCERIDES	35 - 165 mg / dl
BLOOD SUGAR (P.P.) (TRUE GLUCOSE)	Upto 125 mg / dl
F. BLOOD SUGAR (TRUE GLUCOSE)	65 - 110 MG / DL
H.B.D.H.	90 -220 IU / L
C.P.K	10 - 120 IU / L
L.D.H	40 - 110 IU /
S.G.O.T.	7 - 24 IU / L
S.G.P.T.	4 - 25 IU / L
G.G.T.P	0 - 31 IU / L
TOTAL BILIRUBIN	0.2 - 1.0 mg / dl
CONJ. DIR. BILIRUBIN	0.1 - 0.6 mg / dl
UNCONJ. BILIRUBIN	0.1 - 0.4 mg / dl
CHOLESTEROL	125 - 225 mg / dl
TOTAL PROTEIN	6 - 8 Gm / dl
ALBUMIN	3.5 - 5.0 Gm / dl
A / G RATIO	0.9 - 2.0
ALK. PHOSPHATASE	A à 15 - 65 IU / L C à 70 - 150 IU / L
CALCIUM	A à 8.5 - 10.5 mg / dl C à 9.5 - 11.0 mg /dl
PHOSPHORUS	A à 2.5 – 4.5 mg / dl C a 2.5 - 5.5 mg / dl
BLOOD UREA	15 - 45 mg / dl
S. CREATININE	0.5 - 1.5 mg / dl
S.URIC ACID	2.1 - 7.4 mg / dl
SODIUM	137 - 148m.Eq / L

POTASSIUM	3.5 - 5.6m.Eq / L
CHLORIDES	99 - 108m.Eq / L
BICARBONATE	23.7 - 31.4m.Eq / L

South East Asia Regional

NEONATAL - PERINATAL DATABASE

World Health Organization (South-East Asia Region)

Working Definitions

I GENERAL:

1. INTRAMURAL BABY: A baby born within premises of your center
2. EXTRAMURAL BABY: Baby not born within premises of your center
3. FETUS: Fetus is a product of conception, irrespective of the duration of pregnancy, which is not completely expelled or extracted from its mother
4. BIRTH: Birth is the process of complete expulsion or extraction of a product of conception from its mother.
5. LIVE BIRTH: A live birth is complete expulsion or extraction from its mother of a product of conception, irrespective of duration of pregnancy, which after separation, breathes or shows any other evidence of life, such as beating of the heart, pulsation of the umbilical cord, or definite movements of voluntary muscles. This is irrespective of whether the umbilical cord has been cut or the placenta is attached. [Include all live births >500 grams birth weight or >22 weeks of gestation or a crown heel length of >25 cm]
6. STILL BIRTH: Death of a fetus having birth weight >500 g (or gestation >22 weeks or crown heel length >25 cm) or more.
7. BIRTH WEIGHT: Birth weight is the first weight (recorded in grams) of a live or dead product of conception, taken after complete expulsion or extraction from its mother. This weight should be measured within 24 hours of birth; preferably within its first hour of live itself before significant postnatal weight loss has occurred.
8. LOW BIRTH WEIGHT (LBW): Birth weight of less than 2500 gm
9. VERY LOW BIRTH WEIGHT (VLBW): Birth weight of less than 1500 gm
10. EXTREMEY LOW BIRTH WEIGHT (ELBW): Birth weight of less than 1000 gm
11. GESTATIONAL AGE (best estimate): The duration of gestation is measured from the first day of the last normal menstrual period. Gestational age is expressed in completed days or completed weeks.
12. PLEASE PROVIDE THE BEST ESTIMATE OF GESTATION. IT MEANS THAT, IN YOUR JUDGEMENT, BASED ON ALL THE HISTORICAL, ULTRASOUND AND BABY EXAMINATION DATA, THE ESTIMATE AS ENTERED IN THE DATABASE IS MOST ACURATE.
13. PRETERM: Gestational age of less than 37 completed weeks (i.e. less than 259 days)
14. TERM:Gestational age of 37 to less than 42 completed weeks (i.e. 259 to 293 days)

15. POST TERM: Gestational age of 42 completed weeks or more (i.e. 294 days or more).
16. PERINATAL PERIOD: Commences from 22 weeks (154 days) of gestation (the time when the birth weight is 500 g), and ends at 7 completed days after birth.
17. NEONATAL PERIOD: It refers to the period of less than 28 days after birth. Early neonatal period refers to the period before 7 days of age. Late neonatal period refers to the period from completion of 7 days upto 28 days of life.
18. MATERNAL DEATH: A maternal death is the death of a woman known to be pregnant within 42 days of termination of pregnancy, irrespective of the duration or site of the pregnancy from any cause related to or aggravated by the pregnancy or its management, but not from accident or incidental causes.
19. PROLONGED RUPTURE OF MEMBRANES: Rupture of membranes or leaking for > 18 hours.
20. ANTEPARTUM HEMORPHAGE: Bleeding per vaginum after 20 weeks of gestation
21. SEVERE MATERNAL ANEMIA: Hemoglobin of less than 7g/dl
22. FETAL BRADYCARDIA: Fetal heart rate of less than 120 per minute
23. FETAL TACHYCARDIA: Fetal heart rate of more than 160 per minute

II NEONATAL DETAILS

24. BIRTH ASPHYXIA

    (A) Definition I (For extramural babies):

    - Moderate birth asphyxia: Slow gasping breathing at 1-minute of age.
    - Severe birth asphyxia: No breathing at 1-minute of age.

    (B) Definition II (For intramural babies)

    - Birth asphyxia: Apgar score of less than 7 at 1 minute of age
    - Moderate birth asphyxia: Apgar score between 4 to 6 at 1-minute of age
    - Severe birth asphyxia: Apgar score of 3 or less at 1-minute of age.

25. RESPIRATORY DISTRESS:

    Presence of at least 2 of the following criteria:

    1. Respiratory rare > 60/minute
    2. Chest indrawing
    3. Expiratory grunt/groaning

    (Note: the baby should be evaluated in between the feeds and in a quiet state. Respiratory rate should be recorded for at least 1 minute.)

26. TRANSIENT TECHYPNEA/DELAYED ADAPTATION: Respiratory distress in a term or preterm neonate starting within 6 hours after birth, often requiring supplemental oxygen, but recovering spontaneously within 3-4 days and showing characteristic x-ray changes (linear streaking at hila and interlobar fluid).
27. HYALINE MEMBRANE DISEASE:

    (A) Presence of all of the following three criteria

    - Pre-term neonate
    - Respiratory distress having onset within 6 hours of birth

- Amniotic fluid L/S ratio of <1.5, or negative gastric aspirate shake test, or skiagram of chest showing poor expansion with air bronchogram/ reticulo-granular pattern/ ground glass opacity.

(B) Autopsy evidence of HMD.

28. MECONIUM ASPIRATION SYNDROME
29. Presence of two of the following:
    (A) Meconium staining of liquor or staining of nails or umbilical cord or skin.
    (B) Respiratory distress soon after birth, within one hour of birth
    (C) Radiological evidence of aspiration pneumonitis (atelectasis and/or hyperinflation).
30. PNEUMONIA:
    (A) In a neonate with respiratory distress, pneumonia is diagnosed in the presence of a positive blood culture or if any two of the following are present.
    (B) Existing or predisposing factors: maternal fever, foul smelling liquor, prolonged rupture of membranes (>18 hours) or gastric polymorphs more than 5 per high power field.
    (C) Clinical picture of septicemia (poor feeding, lethargy, poor reflexes, hypo, hyperthermia, abdominal distension etc.)
    (D) X-ray picture suggestive of pneumonia.
    (E) Positive septic screen (see septicemia)
31. SEPTICEMIA (SYSTEMIC BACTERIAL INFECTION):
    (A) CULTURE NEGATIVE (CLINICAL)
        - In an infant having clinical picture suggestive of septicemia, the presence of any one of the following criteria is enough for assigning probable diagnosis of infection:
            1. Existence of predisposing factors: maternal fever or foul smelling liquor or prolonged rupture of membranes (>18 hrs) or gastric polymorphs (>5 per high power field).
        - Positive septic screen (two of the four parameters (namely, TLC (<5000/mm, band to total polymorph ratio of > 0.2, absolute neutrophil count less than 1800 / cmm, C-reactive protein >1mg/dl and micro ESR>10 mm 1st hour).
        - Radiological evidences of pneumonia.

    (B) CULTURE POSITIVE SEPSIS
        - In an infant having clinical picture suggestive of septicemia, pneumonia or meningitis along with either of the following.
        - Isolation of pathogens from blood or CSF or urine or abscess(es)
        - Pathological evidence of sepsis on autopsy.
32. EARLY/ LATE ONSET SEPSIS (Pneumonia, septicemia, Meningitis, NEC, UTI etc.)
    (A) Early onset: Onset <72 hours.
    (B) Late onset: Onset >72 hours.

33. MENINGITIS
    (A) In the setting of septicemia, if CSF culture is positive; or CSF microcopy and biochemistry are suggestive of meningitis.
34. NECROTISING ENTEROCOLITIS (NEC)
    (A) In a baby at risk for NEC (pre-maturity, sepsis, umbilical venous/arterial catheterization, birth asphyxia, extreme pre-maturity, formula feeding) presence of any two of the following:
        - Pre feed gastric aspirate of >50% of previous feed or abdominal distension.
        - Bloody stools or occult blood in the stools.
        - Radiological evidence of pneumatosis intestinalis/portal air/free air under the diaphragm.
35. HYPERBILIRUBINEMIA
    (A) Total serum bilirubin level needing phototherapy and/or exchange transfusion
36. HYPOTHERMIA
    (A) Skin temperature <36.0C
37. HYPOGLYCEMIA
    (A) Whole blood glucose of less than 45mg/dL
38. HYPOCALCEMIA
    (A) Any one of the following:
        - Serum total calcium <7 mg/dl. or
        - Serum ionized calcium <4 mg/dl.
        - QOTC >0.2 seconds on ECG which normalizes after calcium therapy.
39. INTRAVENTRICULAR HEMORRHAGE (IVH)
    (A) CLINICALLY SUSPECT if at least 3 clinical criteria in a pre-term infant in whom hypoglycemia and pyogenic meningitis have been excluded:
        - Onset of symptoms within 0-72 hours of age
        - Apneic attacks or seizures
        - Sudden pallor or falling hematocrit
        - Gross hypotonia
        - Flat or bulging fontanel
    (B) CONFIRMED if corroborated by ultrasound or CT or autopsy findings
40. ANEMIA
    (A) Hemoglobin <13 g/dl or PCV <40 percent
41. Vitamin K Deficiency Bleeding
    (A) Bleeding from any site especially from the gastrointestinal tract
    (B) Onset 2nd to 5th day of postnatal life
    (C) Prolonged pro-thrombin time and thrombin time, with normal platelet count.
42. APNEIC SPELL

(A) Period of respiratory arrest of a duration of more than 20 seconds: or of less than 20 seconds if accompanied by bradycardia (<100/minute) and/or cyanosis.

43. POLYCYTHEMIA
    (A) Capillary hematocrit of more than 70% or venous hematocrit more than 65% after 24 hours of age
44. MAJOR CONGENITAL MALFORMATION
    (A) A malformation that is life threatening or requires surgical correction.
45. CHRONIC LUNG DISEASE
    (A) Oxygen requirement at 36 weeks post-menstrual age

III. CAUSES OF NEONATAL DEATH

(This entry should be verified by the PI)

Important Note: You will be first asked the cause(s) of death and you would choose from the following 11 causes of death. You may assign more than one cause of death at this stage. You will then be asked to identify the single most important cause of death. Here you will choose only one cause. This is the primary or underlying cause of death which is defined as disease or injury, which initiated the train of morbid events leading directly to death. You will exercise your judgement to assign this cause keeping in mind this definition

1. Perinatal asphyxia: Death of a neonate in the setting of and with features of perinatal hypoxia and / or birth asphyxia followed by manifestations of or hypoxic ischemic injury of brain (hypoxic ischemic encephalopathy) or other organs.
2. Birth trauma: Death due to birth trauma.
3. Extreme prematurity: Extreme prematurity as a cause of death is assigned to infants having birth weight of less than 1000 gm
4. Hyaline membrane disease: Death in a neonate attributable to hyaline membrane disease
5. Intraventricular hemorrhage: Death in a neonate attributable to intraventricular hemorrhage
6. Pneumonia/Septicemia/Meningitis: Death in a neonate attributable to pneumonia or septicaemia or meningitis
7. Tetanus neonatorum: Death due to tetanus neonatorum
8. Congenital malformations: Death due to lethal congenital malformation.
9. Others: Mention the cause not classified by above
10. Not established : Cause of death not established

IV CAUSES OF STILLBIRTHS

(This entry should be verified by the PI)

Important Note: You will be first asked the cause(s) of stillbirth and you would choose from the following 11 causes of death. You may assign more than one cause of stillbirth at this stage. You will then be asked to identify the single most important cause of stillbirth. Here you will choose only one cause. This is the primary or underlying cause of death which is defined as disease or injury, which initiated the train of

morbid events leading directly to death. You will exercise your judgement to assign this cause keeping in mind this definition

1. Aspyhxia: Death of a fetus in the setting of preeclampsia, hypertension, eclampsia, fetal growth retrdation, oligihydramnios, prolonged / obstructed / precipitate labor, meconium passage, cord around the neck, fetal heart slowing or instrumentation.
2. Trauma : Death of a fetus in the setting of cephalopelvic disproportion or obstructed labor or instrumentation with obvious evidence of traumatic lesions,
3. Infection: Death of a fetus in the setting of intrauterine infections (TORCH group) or
4. chorioamnionitis (maternal fever, abdominal tenderness, foul smelling liquor)
5. Congenital malformations: Death of a fetus due to lethal congenital malformation.
6. Rh Isoimmunization : Death of a fetus attributable to erythroblastosis fetalis
7. Others: Mention the cause not classified by above
8. Not established : Cause of death not established