TUTORIAL 6

GENERAL PHYSICAL EXAMINATION

OVERALL OBJECTIVES

At the end of this module the student should be able to do:

- A full general physical examination
- Able to differentiate abnormal from normal findings in different age groups children

By careful observation more information can be obtained than by any single form of examination in children. Each system must be fully observed before it is examined. You need to have the followings in your possession before starting a clinical examination.

- Pupil torch
- Stethoscope
- Tape measure
- Patella hammer
- Blood pressure apparatus
- ENT set with ophthalmoscope
- Growth charts (Z-Score) and a ruler
- Pen and exam pad to document your findings
- Weighing scale, Stadiometer and Infantometer (these are usually provided in exams)

SEQUESNCE OF GENERAL PHYSICAL EXAMINATION

There is no hard and fast rule but you may follow the following sequance when doing a General Physical Examination

Scene & Surroundings
General out look & facies
Posture & attitude
General impression and vital signs

J	Jaundice
A	Anaemia
C	Cyanosis
C	Clubbing
O	Oedema
L	Lymph nodes
S	Splinter haemorrhages
T	Thyroid gland
E	Eyes
E	Ear Nose Throat
S	Skin
T	Tanner staging

1. SCENE & SURROUNDINGS

Look out for clues around the bedside

- Pale stool in a nappy: think obstructive jaundice
- Cola colored urine in a container: think post streptococcal glomerulonephritis

Position for examination

*Patient should be approached from **right** hand side!*

- Under 6 months: on the couch or the bed
- 6 months to 3-4 yrs: start with child on mother's lap
- Over 4 yrs: standing up, lying down or sitting up

2. GENERAL OUTOOK AND FACIES

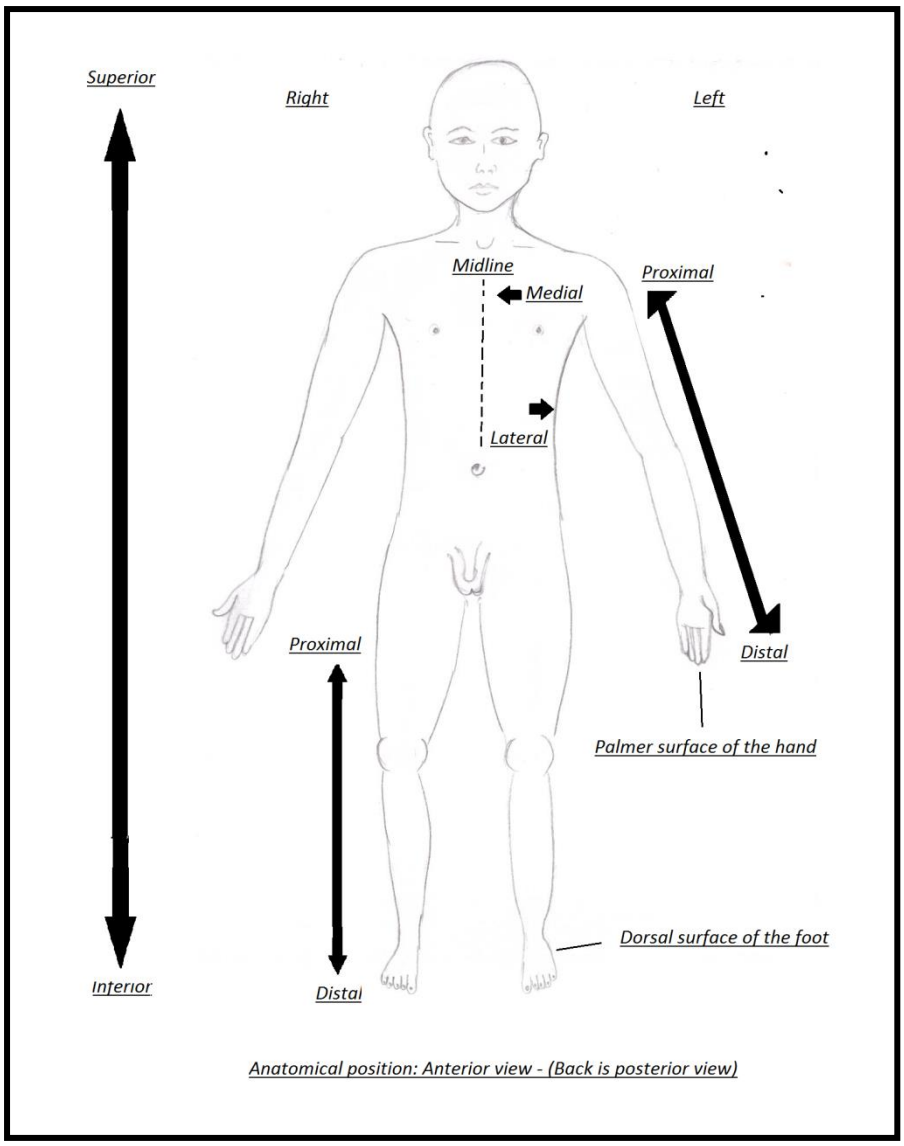
Ill looking, in severe respiratory distress or comfortable at room air

- Moon-like face: Cushing's syndrome
- Idiotic face: Mental retardation
- Pale faec: Anaemia
- Reddish face: Polycythemia, mitral stenosis, high grade fever
- Dusky face: Uraemia
- Yellow face: Jaundice
- Bluish face: Cyanosis

3. POSTURE AND ATTITUDE

Look for adopted posture by the patient on the bed.

- Patient sitting upright with back rest: heart failure (congestion of lungs)
- Patient with extended neck, head bore into pillow: photophobia; meningitis
- Patient with fever and peculiar aspect of helplessness, the limbs lying motionless, the joints being swollen, stiff and painful: rheumatic fever



2. GENERAL IMPRESSION AND VITAL SIGNS

Does the child has any obvious abnormality i.e. a child with a big head

Does this suggest some syndrome; strange looking child

Does the child look mentally retarded

Is he co-operative or un-coperatice, abnormally irritable

Note if the child is well cared for or not

Note if the child is on iv fluids, nasogastric feeds or nasal prongs oxygen or getting nebulised.

Then note the vital signs

- Temperature (normal 36.5 to 37.5 ^{O}C)
- Pulse: rate, rhythm, character and volume
- Blood Pressure
- Respiratory Rate
- GCS
- Check GM (Glucose) if necessary

a. Perfusion & hydration

Comment if the child is ill, distressed or toxic. If the child is ill, comment if critical or stable.

Remove clothing gradually but undress fully, after asking permission from the mother and/or the examiners. Do up the nappy once you have checked the perineum and genitalia

3. JAUNDICE

When serum bilirubin level rises more than 45 µmol/L, jaundice appears clinically.

Method of examination

Retract upper eye lid upward with the help of your thumb. Now ask the patient to look down towards his/her feet without tilting head (difficult in small children). See the colour of sclera which will be yellow in case of jaundice. In addition, examine the under surface of tongue and mucous membranes of the mouth. Also check stool colour, urine and size of the liver (hepatomegaly).

NB! Patient should be examined in sunlight and never in artificial light especially when looking for jaundice.

CAUSES OF JAUNDICE IN CHILDHOOD

Conjugated Jaundice

1. **Obstructive:** Biliary attresia, bile duct stenosis and choledochal cyst
2. **Infectious:** TORCH, EBV, measles and varicella, hepatitis A, B, C, UTI especially gram negative, cholecystitis
3. **Metabolic:** Galactosaemia, tyrosinaemia, cystic fibrosis
4. **Toxic:** TPN, salicylates, Iron & INH, Valproic acid & Phenytoin sodium
5. **Autoimmune:** Autoimmune chronic hepatitis, Graft versus host disease

Unconjugated jaundice

1. **No haemolysis**
   - Physiological jaundice of the new born
   - Breast milk jaundice & breast-feeding jaundice
   - Crigler-Najjar Syndrome: deficiency of glucuronide transferase
   - Gilbert syndrome: Decreased hepatic levels of glucuronide transferase
   - Hypothyroidism
   - Pyloric stenosis
2. **Haemolysis and reticulosis**
   - Positive Coombs test
     - ABO and Rh incompatibility
     - Auto immune (SLE)
   - Negative Coomb's test
     - RBC Enzyme defect: G6PDD
     - Haemoglobinopathy: sickle cell anaemia
     - RBC membrane defect: hereditary spherocytosis
     - Haemolytic uraemic syndrome

6. PALLOR (ANAEMIA)

A condition of having a lower than normal number of red blood cells and quantity of haemoglobin resulting in pallor and weariness. It is exmined from the palms, nails, conjuctiva of lower eye lids, undersurface of tongue, mucous membrane of cheeks and hard palate.

Method 1: Examine the palms and nails of the patient and compare them with your own palms and nails. In anaemia there will be pale nails and palmer pallor.

Method 2: Ask the patient to look upward and pull his lower eye lid down (be gentle). See the anterior of the eyelids. Normally this is pink but in anaemia it will become pale.

Method 3: Ask the patient to open the mouth and examine the colour of the tongue and mucous membranes of the cheeks and hard palate. In anaemia it will look pale.

Picture: Anaemia - Methods 1 , 2 , 3

In order to investigate anaemia we need to check haemoglobin, indices, retics count and red cell morphology to classify as follows:

CAUSES OF ANAEMIA

1. **Hypochromic, microcytic anaemia**
    - Iron dfeiciency (chronic blood loss, poor diet, cow's milk protein intollerence)
    - Thalassemia (alpha & beta)
2. **Normochromic, normocytic anaemia**
    - Chronic inflammatory disease

- Recent blood loss
- Malignancy
- Chronic renal failure

3. **Macrocytic anaemia**
    - Vitamin B12 and folic acid deficiency
    - Hypothyroidism
    - Chronic liver disease
4. **Haemlyitc anaemia**
    - Sickle cell disease
    - G6PD deficiency
    - Heriditary spherocytosis
    - Haemolyitc uraemic syndrome

7. CYANOSIS

When amount of reduced Hb reaches up to 5g % or more, cyanosis appears clinically**.** It can be peripheral or central cyanosis depending upon the underlying cause. A bluish tinge can be found in the nails, lips, tongue, tip of the nose and mucous membrane of mouth.

Peripheral cyanosis: occurs due to vasoconstriction and other conditions leading to the stasis of blood in the blood vessels. The peripheries will be cold, and cyanosis will disappear on making the patient warm. Common causes are:

- Exposure to cold
- Poor perfusion
- Venous congestion
- Raynand's disease (excessive vasomotor stimulation)

Central cyanosis: all the circulating blood is blue therefore extremities, tongue, mucous membranes are blue due to mixing of venous and arterial blood or impaired diffusion of oxygen due to pulmonary disease.

COMMON CAUSES OF CENTRAL CYANOSIS

1. Respiratory
    - Consolidation (pneumonia)
    - Cor pulmonale
    - Chronic obstructive pulmonary disease
    - Bronchial Asthma
    - Persistent pulmonary hypertension
2. Haematological
    - Methemoglobinaemia
    - Sulph-heamoglobinaemia
    - Sever sepsis
3. Cardiac
    - Transposition of great arteries
    - Truncus arteriosis

- Tricuspid atresia
- Tetralogy of Fallot
- Total anomalous pulmonary venous return (TAPVR)

8. CLUBBING

In finger clubbing, the soft tissues at the base of the nails are thickend and the angle between the base of the nail and the adjacent skin of the finger (Lovibond angle) is obliterated.

Schamroth's window test: Originally demonstrated by South African Cardiologist Dr Leo Schamroth on himself is a popular test for clubbing.

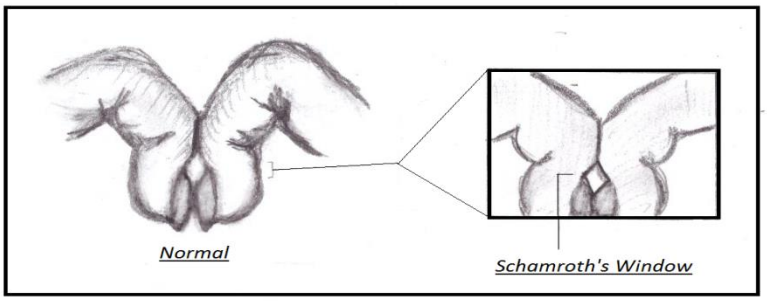
When the distal phalanges of corresponding fingers of opposite hands (preferably thumb nails) are directly apposed (placed against each other back to back), a small diamond shaped window is normally apparent between the nail beds. If this window is obliterated, the test is positive and clubbing is present.

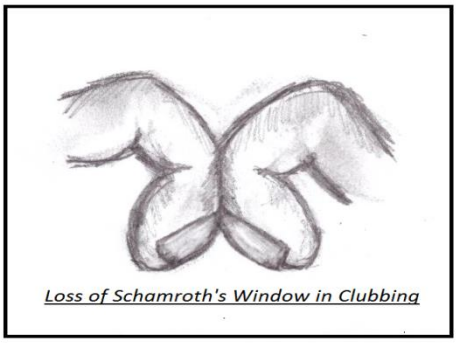
Long standing arterial desaturation (usually longer than 6 months in duration) even if too mild to be detected by an inexperienced person results in clubbing of finger nails and toes. It appears earliest and most noticeably in thumb.

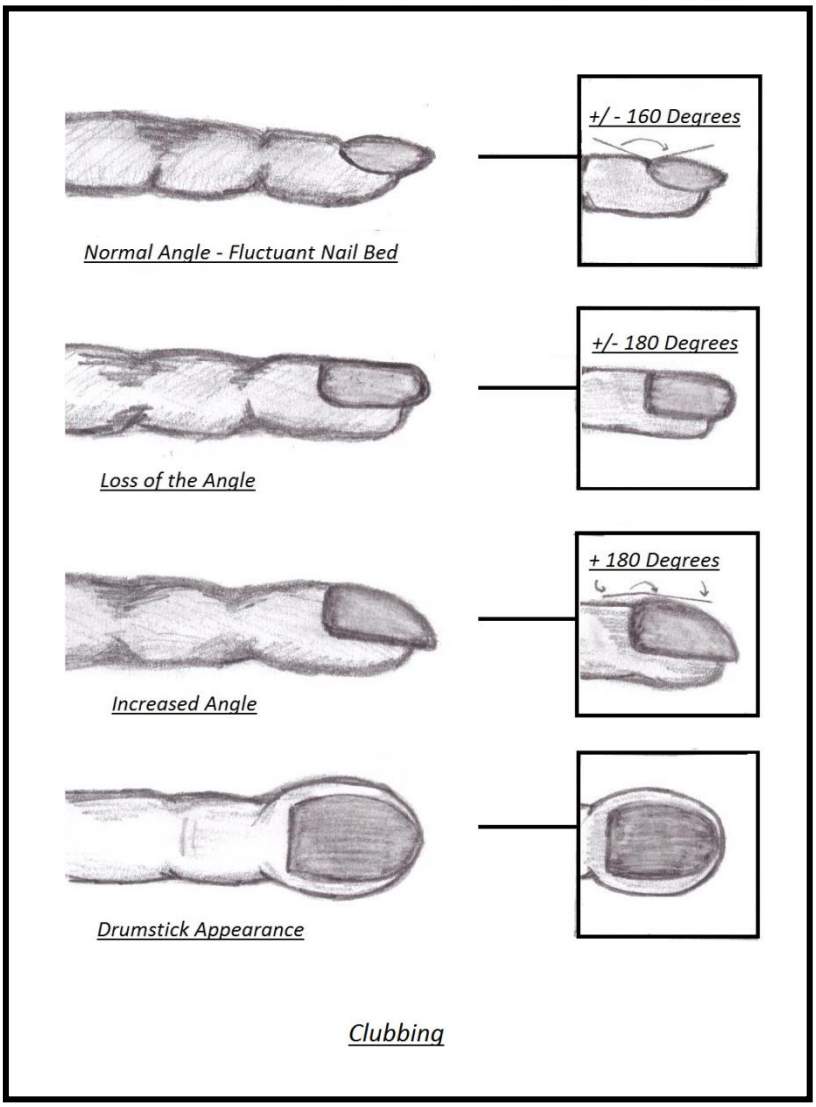
Clubbing develops in 5 steps:

1. Fluctuation and softening of the nail bed
2. Loss of normal Lovibond angle (165^{o})
3. Increased convexity of the nail fold
4. Thickening of the whole distal finger (resembling drum stick)

5. Shiny aspect and striation of the nail and skin







Stages of finger clubbing:

1st stage: obliteration of angle between nailbed and forefinger
2nd stage: fluctuation at the base of nail
3rd stage: drumstick appearance

CAUSES OF FINGER CLUBBING

Idiopathic or primary: Familial, hypertrophic osteoarthropathy

1. **Lungs:**
   - Interstitial lung disease
   - Pulmonary HTN
   - Cystic fibrosis
   - Bronchiectasis
   - Lung abscess
   - Empyema
2. **Heart** (any disease featuring chronic hypoxia)
   - Cyanotic disaeses of the heart like TOF etc.
   - Subacute bacterial endocarditis
3. **GIT and endocrine**
   1. Malabsorption syndrome
   2. Crohn's disaese
   3. Ulcerative colitis
   4. Hyperthyroidism
4. **Liver**
   - Hepatopulomonary syndrome
   - Biliary cirrhosis (primary)
   - Hepatic cirrhosis
   - Intrahepatic biliary atresia
   - Axillary artery aneurysm (unilateral clubbing)

9. KOILONYCHIA

The nails become soft, brittle and spoonshaped

CAUSES

1. Iron deficiency anaemia
2. Plummer-Vinson's syndrome
3. Familial
4. Excessive use of detergents and soap
5. Idiopathic

10.LEUKONYCHIA

Abnormal whiteness of nails, either total or in spots or streaks

Cause may be chronic liver disease

11.SPLINTER HEAMORRHAGES IN THE NAILS

These are small, reddish, dark brown, vertical lines in the nails.

CAUSES

1. Subacute bacterial endocarditis
2. Haemorrhagic disorder
3. Trichurus trichura
4. Sickle cell anaemia
5. Atrial myxoma

12.OEDEMA

It is an excess of fluid present in the interstitial tissues. It can be pitting type or non-pitting.

Pitting oedema

The swollen part is pressed with thumb for 15 to 20 seconds. If it leaves a pit which stays for half a minute the oedema is called as pitting oedema. Oedema is demonstrated in the dependent parts of the body like sacrum and tibial aspect of lower legs.

Non pitting oedema

In the above method if pit doesn't form, this is called as non-pitting oedema.

CAUSES

GENERALIZED OEDEMA

1. **Hypoproteineamia** (inadequate protein intake/absorption)
   - Severe malnutrition with oedema
   - Crohn's disease
   - Coeliac disease
2. **Inadequate protein production**
   - Chronic liver disease causing hypoalbuminaemia (cirrhosis)
3. **Excessive protein loss**
   - Nephrotic syndrome (>5g/day)
   - Sepiated removal of ascitic fluid
   - Protein losing enteropathy (coeliac disease)
   - Thiamine deficiency (wet beriberi)
4. **Fluid overload**
   - Congestive cardiac failure, cor-pulmonale, constrictive pericarditis
   - Secondary hyperaldosteronism
   - Acute glomerulonephritis
   - Excessive fluid replacement (iatrogenic)
5. **Hypothyroidism**

LOCALISED OEDEMA

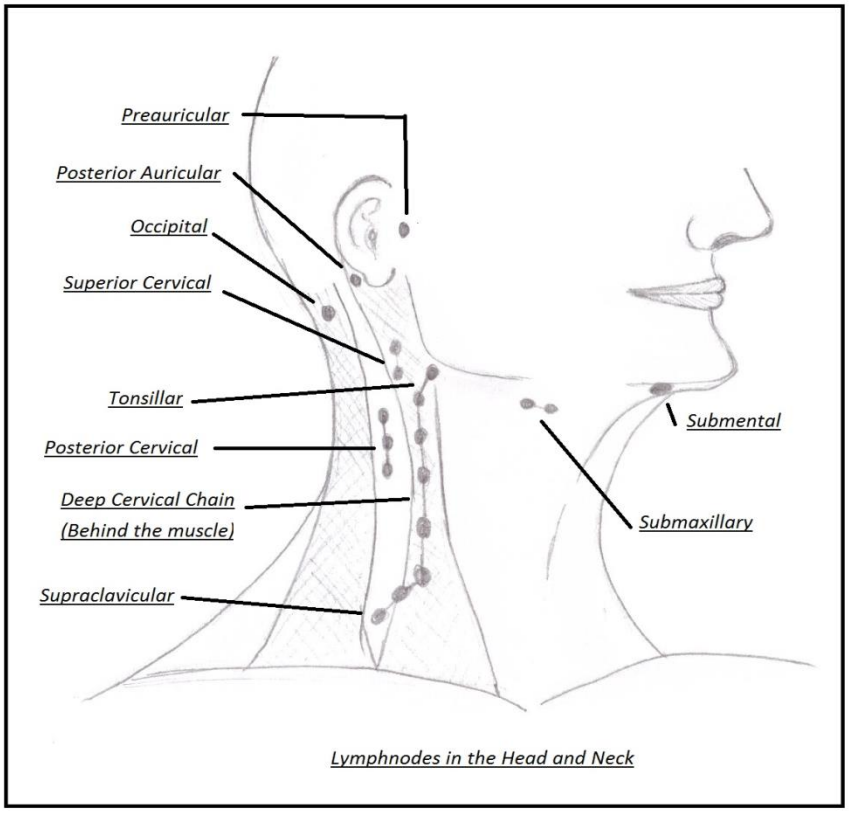
1. **Venous causes –** (pitting – unilateral lower limb oedema)
   - Post-surgical
   - Venous thrombosis
   - Pressure from neighbouring tumour or lymph node
   - Valvular incompetence (varices)
2. **Lymphatic causes – (nonpitting lower limb oedema) +**
   - Lymphoedema
   - Filariasis (lymphatic obstruction by filarial worms)
   - Milroy's disease (unexplained lymphoedema at puberty common in females)
   - Congenital lymphoedema (lymphatic agenesis or hypoplasia of lymphatics of legs)
3. **Inflammatory causes**
   - Infection
   - Injury or ischaemia (histamine, bradykinin, cytokinin) causing inflammation, redness, heat, pain
4. **Allergic causes**
   - Angio-oedema

1. LYMPH NODES

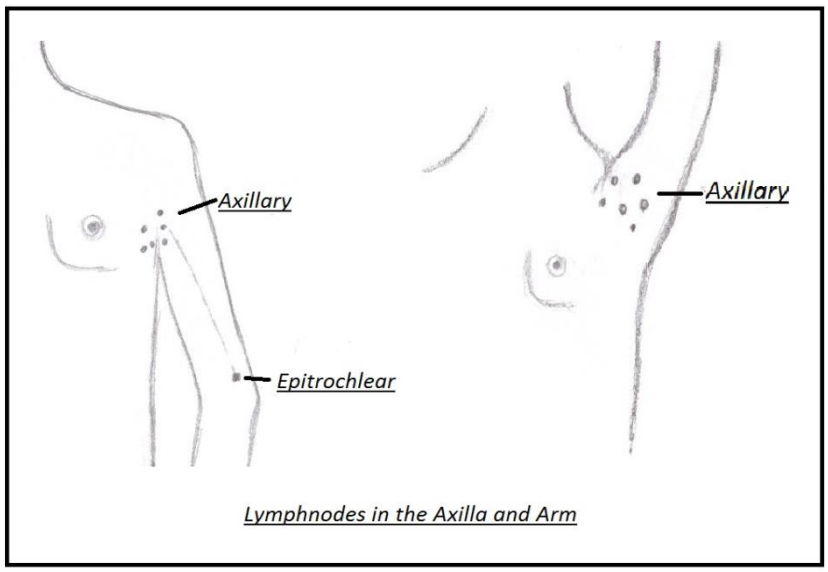
Palpate various groups of lymph nodes.

They are significantly enlarged if following groups are:

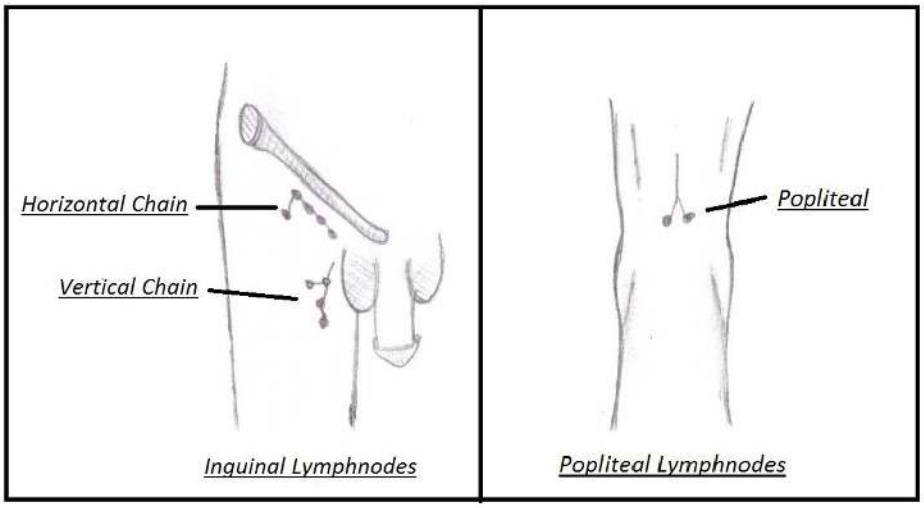
- More than 1 cm
  - postrior cervical
  - submandibular
  - axillary
  - femoral
- More than 1.5 cm
  - Inguinal
- Any size
  - Supraclavicular
  - Epitrochlear (mention if no peripheral inflammation)

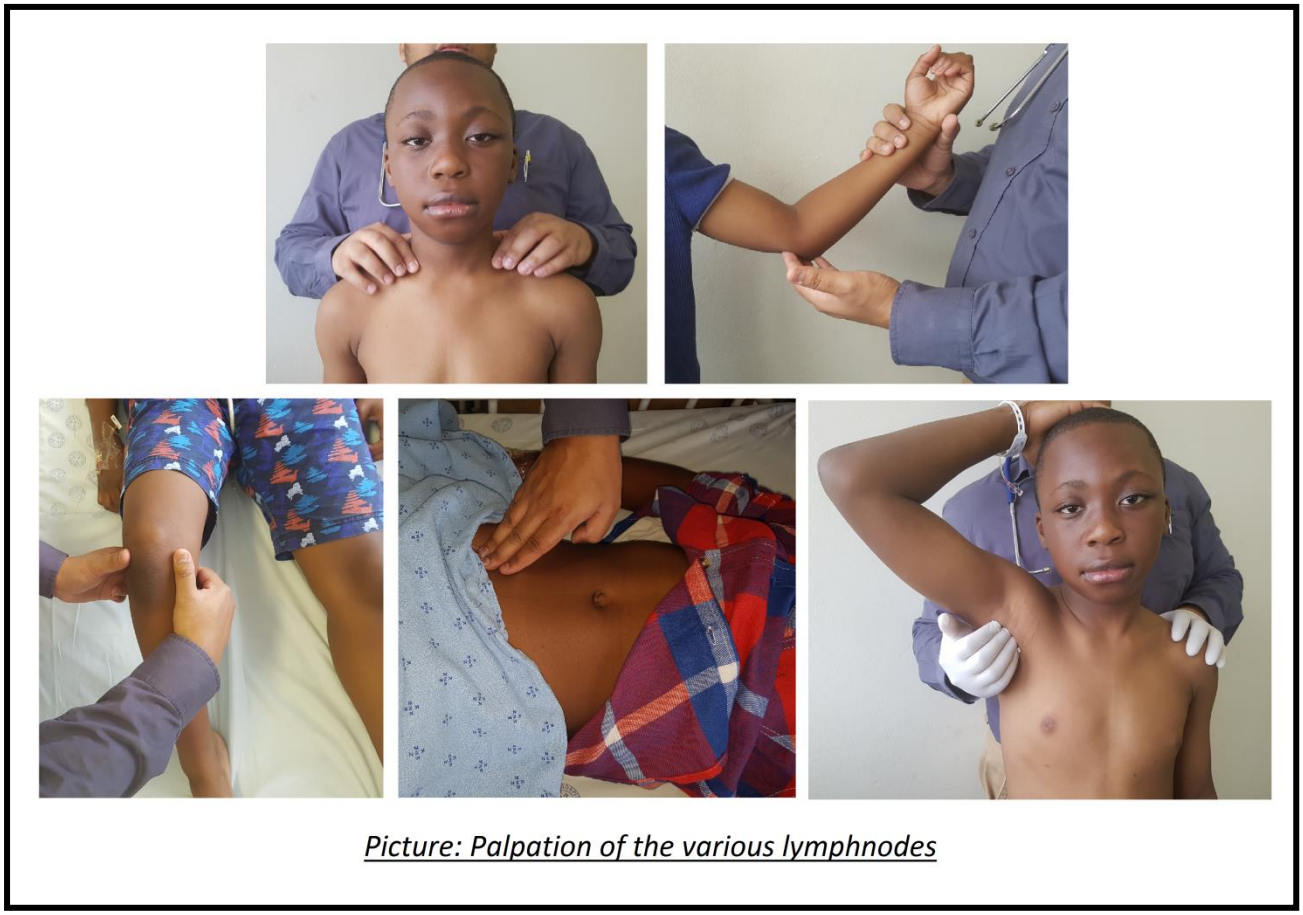


Lymphnodes in the Head and Neck



Lymphnodes in the Axilla and Arm





Picture: Palpation of the various lymphnodes

CAUSES OF GENERALIZED LYMPHADEMOPATHY (LAD)

1. **Viral:** Infectious mononucleoisis, CMV, measles, rubella, viral hepatitis and HIV
2. **Bacterial:** Syphilis (lymph nodes shotty painless discrete), tuberculosis, bacterial endocarditis
3. **Fungal:** Histoplasmosis
4. **Protozoal:** Toxoplasmosis
5. **Non infectious inflammatory diseases;** Rheumatoid arthritis, SLE

6. **Malignant:** Leukaemias: Chronic lymphocytic , acute lymphocytic leukaemia, non-Hodgkin's lymphoma, Hodgkin's disease

CAUSESS OF LOCALISED LYMPHADEMOPATHY

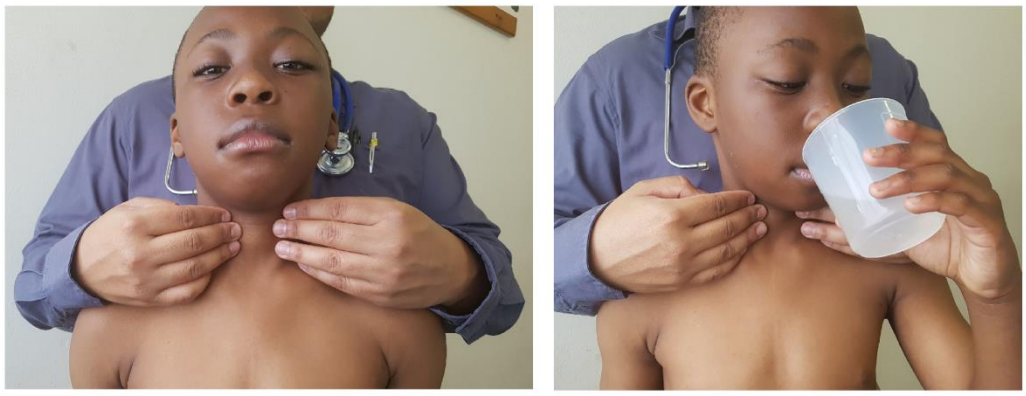
1. Local pyogenic infections like pharyngitis, dental abscess, otitis media
2. Viral infection: cat scratch fever, lyphogranuloma venereum
3. TB (lymph nodes matted and there may be sinus formation)
4. Non- Hodgkin's lymphoma
5. Secondary carcinoma – hard regular and fixed lymph nodes
6. Virchow's gland (supraclavicular nodes, left sided enlarged in carcinoma of stomach – uncommon in children

2. THYROID

Inspect the neck for local or general enlargement of thyroid gland and observe its movement with the larynx as the patient swallows. The thyroid gland will move upward.

Examination of thyroid gland

Stand behind the patient and put your fingers of both hands in front of the neck to palpate the gland.



Picture: Examination of the thyroid gland

If the gland is enlarged, note its:

- Size, shape, surface, tenderness
- Movement of skin over it
- Pulsation
- Systolic bruit (toxic goiter)
- van Graef's sign - in thyrotoxicocis

Von Graefe's sign is the lagging of the upper eyelid on downward rotation of the eyes, indicating exophthalmic goiter (Graves' disease). It is a dynamic sign, whereas lid lag is a static sign which may also be present in cicatricle eyelid retraction or congenital ptosis.

3. TANNER STAGING

Tanner staging, also known as Sexual Maturity Rating (SMR) is an objective classification system that is used to document and track the development and sequence of secondary sex characteristics of children during their puberty. It was developed by Marshal & Tanner, based on their observational data on development of external genitalia:

- Phallus, scrotum, and testes volume in males;
- Breasts in females;
- Pubic hair in both males and females.

Tanner stages are utilized in paediatric and adolescent practice to counsel patients about the timing of anticipated body changes, perform appropriate medical screenings, and monitor for deviations in normal timing and sequence of physical signs of puberty that may represent physiological problems.

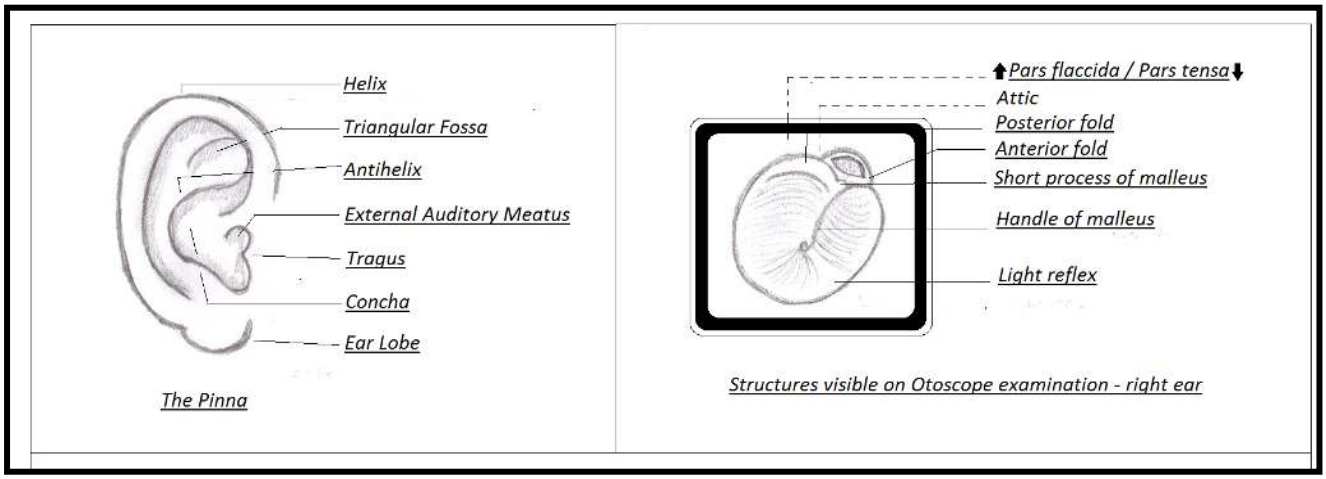
Changes that are associated but are not directly measured by Tanner Staging include bone growth and fusion, body composition and linear growth and haematocrit values. In males, the onset of puberty ranges from 9 to 14 years of age. In females, the normal onset of puberty ranges from 8 to 13 years old averaging 10 years in White Americans and age 8.9 years in African Americans.

<table>
<tr><th colspan="2">TANNER STAGING</th></tr>
<tr><td>Girls</td><td>Boys</td></tr>
<tr><td>BREAST STAGING</td><td>GENITALIA STAGING</td></tr>
<tr><td>B1 Prepubertal
B2 Breast budding
B3 Development of actual breast mound
B4 Areola projects at an angle to breast mound
B5 Adult configuration</td><td>G1 Prepubertal penis, unstretched length 2.5-6 cm, scrotum and testes volume ≤3 ml
G2 Testes ≥ 4 ml and scrotal laxity but no penile enlargement
G3 Penile lengthening and further dvelopment of testes and scrotum
G4 Penile lengthening and broadening further development of testes (volume 10-12 ml)
G5 Adult genitalia</td></tr>
<tr><td colspan="2">PUBIC HAIR STAGING
P1 No pubic hair
P2 Fine hair over mons pubis / scrotum / labia
P3 Adult type hair (curly coarse) but distribution confined to pubis
P4 Extension to near adult distribution
P5 Adult</td></tr>
<tr><td colspan="2">AXILLARY HAIR STAGING
A1 No axillary hair
A2 Hair present not adult amount
A3 Adult</td></tr>
</table>

4. ENT

Ears: Inspection of external ear

- Pinna in general
- Auditory cannal: ask and check if tragus is painful
- Draw outer part of the ear up & back in the older child and down & back in the infant
- Examine auditaory cannal for inflamation, foreign body, wax or discharge
- Do otoscopy for redness, discharge, perforations of tympanic membrane



The Pinna

Structures visible on Otoscope examination - right ear

Nose

- Notice any deformity - nasal septum deviation
- Look for signs of allergic rhinitis
- Nasal polyps, masses, turbinate hypertrophy
- Percussion of sinuses (acute/acute on chronic if tender)

Mouth open

Inspect buccal mucosa for

- Ulceration
- Oral thrush
- Kaposi's sarcoma
- Koplik spots
- Dental caries
- Tongue tie
- Thrush
- Leukoplakia

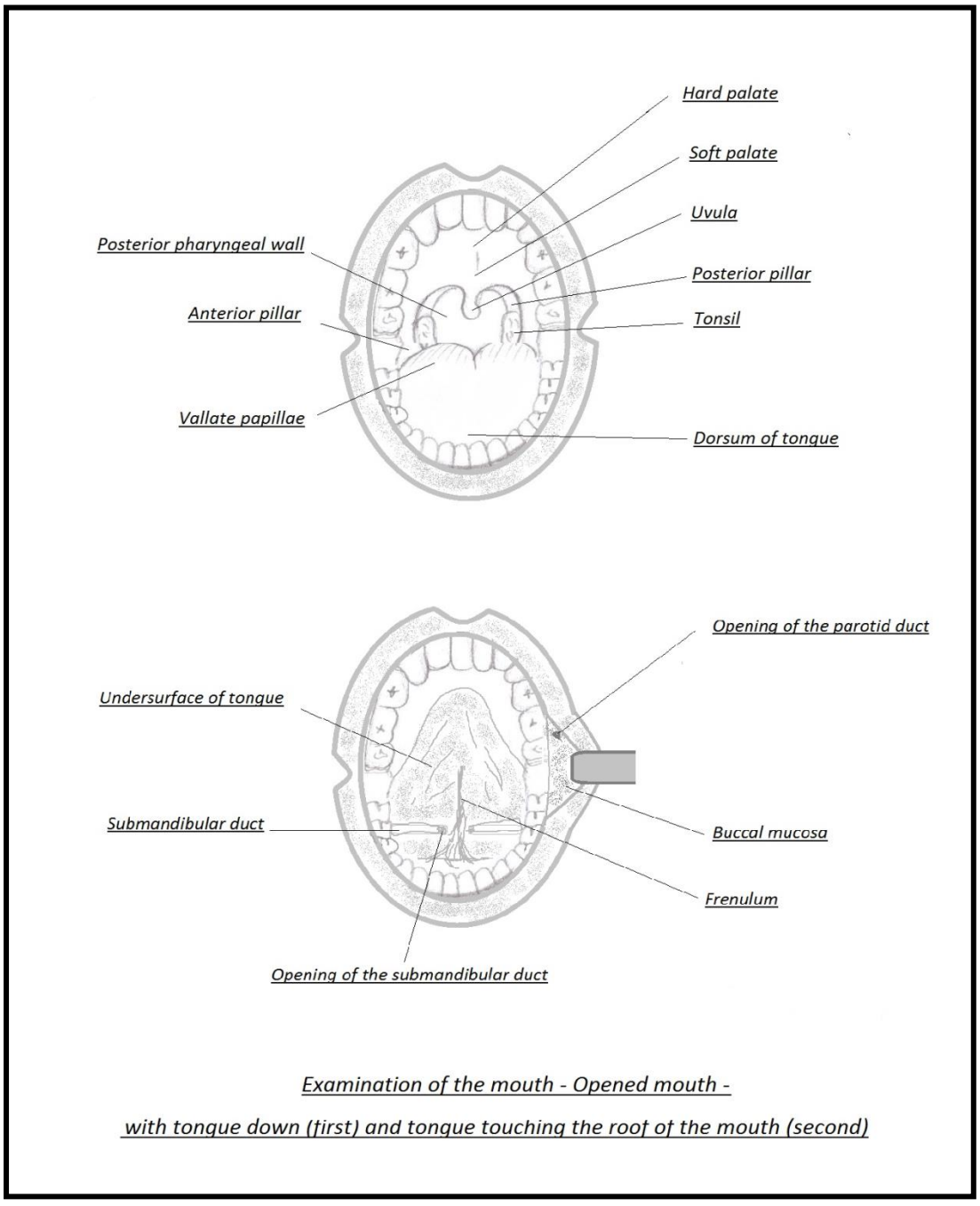
Inspect gums for

- Gum hyperplasia – acute myeloid leukaemia (AML)
- Hypertrophied and firm – treatment with phenytoin
- Soft haemorrhagic – scurvy

- Spongy and haemorrhagic – congenital cyanotic heart disease
- Punctate blue line – lead poisoning

Teeth

- Primary dentition: 3 yrs – 20 teeth
- Secondary start at 6 years: 32 teeth
- Look for caries or missing teeth
- Floating teeth – Burkitts lymphoma



Examination of the mouth - Opened mouth -
with tongue down (first) and tongue touching the roof of the mouth (second)

Salivary glands

- Protids
- Submandibular
- Sublingual

Throat - pharynx / larynx

- Look hard palate – abnormal arch
- Cleft palate
- Talangiectasia
- Tonsills – size, color, discharge
- Gag reflex

EYES

Conjunctivae: look for purpura, jaundice, anaemia, cyanosis

Cornea: normal or look for scars or ulcers

Cataract: consider diabetes mellitus, congenital rubella, trauma to the eye, galactosaemia

Hypopyon: It is the presence of pus (accumulation of WBCs) in anterior chamber of the eye

In leukaemia there may be infiltration of the iris

Leukocoria: white pupilary reflex – cat eye reflex.

The D/D can be:

- Cataract: galactosaemia, rubella
- Cicatrical retinopathy of prematurity
- Retinal detachment
- Retinoblastoma

5. SKIN

BCG scar: Look for

- BCGioma
- BCG lymphadenitis
- Disseminated BCG disease

Regarding BCG scar:

- There is no associateion between the presence of a BCG scar and immunogenicity or effectiveness of the vaccine.
- Prolonged ulceration at BCG site may occur with lymphadenitis in 1-10% of cases and is more common in HIV infected children

Mantoux test: if present measure and interprete

Two strains of PPD are available in South Africa

- PPD-RT 23 strain: dose 2 TU (currently used in KZN)
- BCG Danish 1331 strain: dose 5 TU

Surgical scars: count and measure – work out what procedure might have been done

Traditional markings: if present ask for any use of traditional medication or herbal enema

Bleeding diathesis: look over the skin, inside mouth (wet petechie - thrombocytopenia)

Scratch marks: haemolysis, jaundice

Trophic changes: occur in soft tissue (skin, fascia, muscle), resulting from interruption of nerve supply

Rash: it is an acute manifestation of disease. When describing a rash assess the following:

- The type of rash
- The distributon i.e. parts of the body involved
- Progression of rash
- How it has regressed
- How it has healed

Types of rash

- Erythema marginatum: pale centered ringlets with pink margins, present over trunk and flexor surfaces of joints i.e., Rheumatic Fever
- Erythema nodosum: conical rounded nodules present just under skin , present on extensor surfaces of the joints – elbows shins and dorsum of the hands and feet

Common causes	**Less common causes**
Rheumatic fever TB GIT infections (yersinia, salmonella, shigella, or systemic fungal infections)	Ulcerative collitis Crohn's disease Drug sensitivity – sulpha, salisylates

Tissue defect: Maceration, Erosion, Ulcer

Deposition of skin: Scale formation, Crust on scab formation

Macule: alteration in colour

Papule: hemispherical elevation of epidermis <5mm

Vesicles bulla: accumulation of serous fluid

Pustule: accumulation of pus

Nodule: involvement of whole thickness of skin by inflammatory infilterate or some new growth of cells – it is harder than papule >5mm

Heberdens nodules: small nodules present at the terminal phalangeal joints in case of osteo-arthrosis

Wheel formation: oedema formation in dermis due to allergy and associated with severe itching – uricaria

Erythematous lesion: capillary dilatation – will blench on pressure

Haemorrhagic spots: these do not blench on pressure: D/D telengiectasia

- Petechial: small pin point lesions in the skin
- Purpura spots: haemorrhagic spots which are larger than petechiae
- Ecchymosis: like large bruises

Kaposi's sarcoma (Human Herpes Virus 8): multipe firm, purple blue or reddish-brown plaques and nodules typically appear initially on the hands and feet and progress up the arms and legs over a period of years and decades, eventually involving the viscera or mucosa in about 10% of patients. Untreated lesions evolve from flat discolourations or

patches to plaques and then to raised nodules that become confluent. Lymphaedema may preced or follow the appearance of lesions.

Neurocutaneous lesions – café a lait spots

Café a lait spots are flat hyperpigmented lesions on the skin due to increased deposition of melanin and an increased number of melanocytes. Differentiate from motherspots (birth marks). Considered significant if:

- 5 or more and >5 mm in prebubertal
- 6 or more and >15 mm in post pubertal

SYNDROMES ASSOCIATED WITH THE PRESENCE OF CAFÉ A LAIT SPOTS

(Acronym: **N**andi **A**nd **F**harnisa **H**ave **T**o **G**o **T**o **B**uy **M**e **M**y **N**ew **R**ing **V**ery **S**oon)

1. Neurofibromatosis
2. Ataxia telangiectasia
3. Fanconi's anaemia
4. Hurler syndrome
5. Tuberous sclerosis
6. Gaucher's disease
7. Turner syndrome
8. Bloom syndrome
9. Marfan syndrome
10. McCun Albright syndrome
11. Noonan syndrome
12. Russel silver syndrome
13. Von Hipple-Lindau disease
14. Sturg Weber syndrome