Tutorials on Clinical Methods in Paediatrics

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Preface

Due to Covid-19 lockdown most of the Medical Students and Interns who are under training in the discipline of Paediatrics and Child Health, may find it difficult to have a face to face tutorial in their respective hospitals. Therefore, I have developed this document in such a way that each tutorial discusses basic information related to that system first and then explains clinical methods with photographs and lively illustrations.

It was presumed that students learning Paediatrics have had previous exposure of clinical methods therefore in-depth description of basic clinical terms is not done in this review. It is also not intended to be a text book or encyclopaedia, instead to serve as a guide to what need to be done at the bedside.

Although main emphasis is made on history taking and clinical examination, students are also guided to make logical deductions and differential diagnosis on the basis of their clinical findings. Since my approach is basically clinical that will be confined largely to clinical signs and symptoms in children of varying ages, it does not include pathophysiology, laboratory investigations and treatment protocol. Readers are therefore directed to main text books of Paediatrics and Neonatology if detailed information is required on a particular condition. In keeping with the need of the reader I have tried to retain a problem-oriented approach where possible, and I hope that this manual fills the need for a distant learning concise set of tutorials on Clinical Methods in Paediatrics.

I am indebted to my team of doctors which include Dr Yajna Ramdass, Dr Riaz Bux and Dr Irfaan Saib for assisting me for lively illustrations and photographic shots. I would also like to thank the parents of little boy for allowing us to include him for illustrating the clinical examination.

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SOURCE OF INFORMATION & ACKNOWLEDGEMENTS

1. Macleod's Clinical Examination 11th ed
2. Students Clinical Methods: M Ali 4th ed
3. Clinical Examination: NJ Tally et al 5th ed
4. Examination Paediatrics: Wayne Harris 3rd ed
5. Coovadia's Paediatrics and Child Health 6th ed
6. Weiner and Levitt's Pediatric Neurology 4th ed
7. Parks Paediatric Cardiology for Practitioners 5th ed
8. Clinical Paediatrics for post graduate students 3rd ed
9. Paediatric Guidelines: Mc Karrow, Patrick & Stephen et al
10. Bates Guide to Clinical Examination and History Taking 4th ed
11. Handbook of Clinical Methods Course for 4th year UKZN Students 2018
12. The History and Clinical Examination of Children: University of Pretoria 2009

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TUTORIAL 1

HISTORY TAKING

OVERALL OBJECTIVES:

At the end of this module, students are expected to acquire the art of appropriate history taking by:

a. Eliciting the chief complaints i.e. the reasons for presentation
b. Evaluating each complaint through questioning
c. Assessing relevant positive and negative aspects in the history that contribute to the most likely diagnosis

Followings need to be taken into account when taking a paediatric history

History taking from both parents and the child is an important art for the interpretation of clinical signs in children of varying ages.

SYMPTOMATOLOGY

A symptom is a normal physiologic response to a harmful stimulus. Every symptom and sign has a beginning and a course of development that may be progressive.

Symptoms and signs are products of the body that produced them. Each body creates symptoms and signs in a unique way, and each personality adapts to them its own way.

One symptom by itself usually means very little. It is its relationship to other symptoms that is significant. For instance, vomiting accompanied by abdominal pain in the lower right quadrant may suggest appendicitis, while vomiting with headache and failing vision could lead one to suspect something causing increased intracranial pressure.

Symptoms are either subjective or objective, or both. Subjective symptoms are those perceptible only to the patient such as sensory disturbances i.e. pain, tenderness, headache, nausea, itching and numbness. Pain and itching are pure subjective symptoms.

Objective symptoms which are evident to the observer are called physical signs like temperature, pulse rate and rhythm, respiratory rate and character, oedema, and gait.

In order to elaborate a symptom fully, we need to ask the relevant questions and duration of each symptom related to patients' complaints. Paediatric patients presenting with the following symptoms should be enquired about:

1. FEVER

- Mode of onset of fever: sudden or gradual
- Associated with rigors or sweating
- High or low grade
- Pattern of fever: continuous, remittent or intermittent
- Associated with vomiting, diarrhoea, abdominal pain, headache or burning on micturition

2. COUGH

- Severity and frequency
- Sudden vs gradual (foreign body)
- Occurs during a special time of day or night
- Seasonal or associated with dust, pollen or perfumes
- Abnormal sounds while coughing (croupy, whooping cough)
- Colour changes while coughs (cyanosis)
- Dry or productive
- Colour of sputum or presence of blood

3. VOMITING

- Frequency of vomiting
- Forceful or projectile
- Relationship between food and vomiting (may be meningitis)
- Special time of vomiting (early morning: may be space occupying lesion)
- Amount, smell, colour and contents of vomitus: yellow, bile stained or feculent
- Any other associated symptoms like heart burn or epigastric pain

4. DIARRHOEA

- Loose motions: intermittent or continuous
- If intermittent: duration which patient is free from symptoms
- Frequency of stools passed per day (count per 24hrs period)
- Colour, quantity, odour and contents of stool
- Any blood in stools: red or black / separate or mixed in stools
- Any history or eating out / bottle feeding/ formula feeding
- Any history of diarrhoea in any other family member staying with child
- History of pain during defecation
- When last did the child passed urine?
- History of administration of ORS and if made appropriately
- Check if the child is vaccinated for Rota virus vaccination

5. PAIN

- Site of pain
- Localised or diffuse
- Continuous or intermittent
- If radiates to which direction
- Dull, burning, colicky or stabbing
- Intensity of pain: mild, moderate or severe /excruciating
- Factors which aggravate or relieve pain
- Does it affect sleep, play and activity (growing pains vs pathological pains)

6. FITS

- Generalised or localised
- First episode or repeat
- Previous history of fits and the time duration

- Frequency and time duration of each fit
- Type: myoclonic/tonic/clonic/ atonic/absence
- Any loss of consciousness or history of aura
- Weakness or hemiparesis thereafter
- Any history of fall, head trauma or loss of sphincteric control
- Any other associated symptoms like tongue bite, froth in the mouth, deviation of eye balls during the attack
- Family history of epilepsy or fits

7. WEAKNESS

- Mode of onset: sudden or gradual
- Extent of weakness: generalised or localised to one part of body like one arm or one leg (monoplegia), one half of the body (hemiplegia), both legs (paraplegia), or one half of the face (facial palsy)
- Grade of weakness:
    - **Grade** 0: No contraction or muscle movement
    - **Grade 1:** Trace muscle activation, such as a twitch, without achieving full range of motion
    - **Grade** 2: Movement at the joint with gravity eliminated
    - **Grade** 3: Movement against gravity, but not against added resistance
    - **Grade** 4: Movement against external resistance with less strength than usual
- Any other associated symptoms like loss of consciousness, fever, headache, neck rigidity, loss of sensations or presence of tremors
- History or trauma / head injury or fever in the past
- Feeling week at rest or after prolonged use of muscles (myasthenia gravis)

8. HEADACHE

- Character: dull, shooting or throbbing
- Continuous or intermittent
- If intermittent: duration and frequency of each attach
- Related to any particular time of the day
- Relieving or aggravating factors
- Part of head involved: one half (migraine), frontal or occipital region
- Any associated symptoms like vomiting, nausea, impairment of vision or consciousness
- History of head injury, hypertension, neck rigidity, or loss of consciousness
- History of blurred vision or unable to see black boards in school children

9. LUMP OR MASS

- Site, size and number of lumps
- Then note its surface, consistency, mobility, tenderness and fluctuation
- Note skin over the mass: fixed or mobile
- Any sinus formation over the swelling
- Note lymph nodes: swollen, separate or matted together

HISTORY TAKING

In order to have correct diagnosis we need a perfect history, good clinical examination and laboratory investigations. Following general but important points need to be noted before starting an interview with the child, parents or the caregivers.

1. See the patient in private room if possible and listen carefully to everything that is said.
2. Introduce yourself and make the acquaintance of both the child and parent.
3. Light banter about the weather or how they found the travelling to hospital help to break the ice.
4. A detailed discussion with the parents and the child is very important step towards correct diagnosis, identification of the problems and further management.
5. Don't jump to conclusions prematurely or you may come with wrong answers.
6. Let the child or the parents tell the story in their own way & words and make notes as you go along. At the same time an occasional guiding question will keep the story going in the right direction. Show interest and concern for the patient's problems.
7. Listen to the voice, but also watch the facial impressions, body movements and hand positions. These may provide additional information. The actions will often provide information about the underlying emotional state as well as giving an indication as to the reliability of information being supplied.
8. Collect the patient's information without expressing surprise or making judgments. It is unethical to comment adversely on another practitioner's or clinical nurse's previous treatment. Neither you nor the patient has enough information to pass the judgment. The information you receive may sometimes be falsified by the parents for fear of possible accusation of neglect, for compensation or even insurance purposes. Information can also be suppressed because of fear. Mothers may misinterpret symptoms but rarely invent them.
9. Avoid "why did not you" questions – they may lead to hostility and consequent lack of co-operation as the parent / guardian feels threatened. The same question can be phrased in a less accusing form.
10. Parents experience reactions to health problems like fear and anxiety. Reassurance must be given and fears relieved whenever possible.
11. Be sure that you understand what the patient or parents mean and that they understand what you mean. This is of greater importance especially when interpreters have to be used. Always use lay terms for questions and explanations.
12. When taking history, make notes of both positive and negative findings and define the problems. From this you should be able to decide which system must be examined in detail. This history will often produce a working diagnosis but keep an open mind until full examination is completed.
13. After taking history and doing clinical examination define the patient's problem. Explain each problem and its management to the parents and when they should see you again.
14. Warn the patients / parents of possible complications or danger signs which may occur and what to do in that event.
15. Remember there are three assessments to be made:
    a. The physical diagnosis and the plan of management
    b. The assessment of patients hidden but unspoken fear about the child's problem

c. The assessment of parents' capability of understanding the nature of the problem and the likelihood of their following your advice

BACKGROUND AND BASIC INFORMATION

A complete database entails the following:

1. **Name & surname:** for identification
2. **Age & date of birth:** certain conditions are common in early childhood others in later life like Wilms' tumour is present in childhood and malignancy and vascular disorders common in older patients
3. **Gender:** Certain conditions present in males others in females like Haemophilia never occur in females and gout and ankylosing spondylitis has preponderance in males
4. **Religion & Church affiliation:** to understand the problems that may arise in certain situations like: blood transfusions are not accepted by the people practicing Jehovah's Witness; Muslims & Jews both practice circumcision in early childhood
5. **Parents' Name and Surname:** their cell and telephone numbers at home & at work
6. **Residential address:** to understand social circumstances around the child and the fact that certain diseases & conditions are common in certain areas. Secondly a future correspondence may be required.
7. **Informant:** name and relation to the patient. If the informant is other than the mother, elaborate the reason?
8. **Details of Doctor who referred the patient:** family physician & his/her address. Document if patient is not a referral case – self referral?
9. **Place of consultation:** consulting rooms, outpatient department, causality or paediatric ward
10. **Date and time** of your consultation
11. Name of the ward or the unit (if admitted)
12. Name and qualifications of admitting doctor

PRESENTING COMPLAINTS

A presenting symptom (chief complaint) is that symptom, or group of symptoms, about which the patient complains or seeks relief. Chief complaints which have brought the patient to the doctor are arranged in chronological order i.e. complaint of longest duration at the top and that of shortest duration in the last.

Each complaint should be written in one line and should be brief. Where possible, write down the information offered in their own word like if the Parents say that the child has shortness of breath or difficulty in breathing do not decide to write "dyspnoea".

Date the commencement of symptoms precisely and completely and use adjectives with each complain like fever with rigors for 5 days instead of fever for 5 days.

HISTORY OF PRESENT ILLNESS

Details of each complaint are asked in chronological order by asking additional questions to bring out full description of individual symptom. Avoid asking leading questions and do not use technical terminologies, instead if possible, write down the history in the patient's own words. While describing the symptoms, following general points need to be noted:

1. **Duration of symptoms:** ascertain the duration by asking "were you alright before such period of time". Date the commencement of symptoms precisely and completely, like diarrhoea started on 25th Dec 2019 – that is 5 days ago.
2. **Health status:** immediately prior to the present illness & how long ago the patient was completely well
3. **Mode of onset:** to ask if the symptoms appear suddenly or gradually
4. **Disease course, sequence and period separating new symptoms:** to ask if the symptoms have been present continuously since their onset or have there been intervals of freedom – note length of these intervals.
5. **Factors** aggravating or relieving symptoms
6. **Associated phenomena:** to ask if any other symptoms associated with the main complaints like abdominal pain due to intussusception may be associated with vomiting, diarrhoea or blood in stools and pain in right upper quadrant may be associated with jaundice & pale stools in obstructive jaundice.
7. **Treatment received and duration** - whether the symptoms are improving or getting worse or of the same nature. History of drug allergy should be asked as well.
8. **Possible exposure to infectious diseases** or recent travel or visits to malaria areas
9. Relevant negative data obtained by direct questioning e.g. no visits to possible malaria areas
10. **If the symptoms occur in clusters or attacks –** a typical attack should be described with regard to frequency, duration and degree of symptoms

SYSTEMIC ENQUERY

Questions related to all body systems can be asked but special emphasis should be made on systems involved.

- **SKIN:** Skin temperature, skin colour, pruritus, hydration, eczema and other skin rashes – associated medication / environmental exposure etc.
- **EYES:** Vision, glasses, infection, allergies and strabismus, swelling discharge
- **ENT:** Hearing, pain, nose obstruction, snoring, runny nose, epistaxis, sneezing, caries teeth, stomatitis, sore throat, tonsillitis, adenitis, mouth breathing etc.
- **RESPIRATORY SYSTEM:** Cough, wheeze, shortness of breath, strider, cyanosis, sputum, haemoptysis, asthma, foreign body, snoring, mouth breathing and enquiry about precipitating factors like exercise, dust, animals. Ability to speak full sentence with wheeze
- **GASTROINTESTINAL SYSTEM:** Weight loss or abnormal weight gain, appetite, vomiting, haemochezia, constipation, diarrhoea, jaundice, melena, worms in stool, pain and thirst.
- **CARDIOVASCULAR SYSTEM:** Shortness of breath, tires while feeding, excessive sweating (in infants it is sign of CCF), limitation of exercise, oedema, palpitations, haemoptysis and cyanosis.
- **CENTRAL NERVOUS SYSTEM:** Headache, mental state, seizures, tremors, paralysis, sight, hearing, behaviour, inattention or hyperactivity, ataxia, weakness, gait, co-ordination, dizziness, muscle and joint swelling & pains, weakness and postural deformity.
- **GENITOURINARY SYSTEM:** Frequency, bladder control, dysuria, character of stream, urine colour (be aware of the pigments resembling blood) enuresis, and vaginal discharge.

- **ENDOCRINE SYSTEM:** Growth, polyuria, polydipsia, apathy, hoarse voice, muscularity, menstruation, breast and testes and growth of pubic and axillary hair.
- **HAEMOPOITIC SYSTEM:** Pallor, tiredness, shortness of breath, bleeding tendency, bruising, enlarged glands and spleen.

PAST HISTORY

This should not be confused with the earlier symptoms of the present condition but this includes attacks of a similar nature when a long interval has elapsed. Following points need to be kept in mind:

1. Diseases of childhood such as rheumatic fever, whooping cough, mumps, measles or primary pulmonary tuberculosis should be enquired or any history of relevant disease in the past.
2. Any history of trauma, accident, surgery, visit abroad, intake of "herbal medications, herbal enemas or scarification from traditional healers.
3. History of any previous admission in the hospital may indicate the seriousness of the disease like child with uncontrolled chronic persistent asthma admitted twice since last month.

BIRTH HISTORY

Birth history is particularly important in young infants which should be confirmed by looking into the Road to Health Booklet. Salient points should include:

1. **ANTENATAL HISTORY**
    - Age of mother
    - Gravida, para, abortions, perinatal deaths
    - Birth weight and health of previous infants
    - Gestational age at birth of the index patient
    - Planned or unplanned pregnancy
    - Foetal growth and wellbeing
    - Complications of pregnancy e.g. eclampsia, gestational diabetes
    - HIV status and CD_4 count and viral load if HIV infected
    - Antiretroviral prophylaxis or treatment detail
    - Results of WR or RPR – treatment if syphilis was diagnosed
    - Blood group of mother
    - Exposure to medicines, smoking, alcohol
2. **NATAL HISTORY**
    - Date and place of delivery
    - Duration and mode of delivery
    - History of foetal distress
    - Other problems during labour and delivery
    - Medication received by mother
    - Apgar score and if baby needed resuscitation
    - Estimated gestational age
    - Birth weight, length, head circumference
    - Vitamin K and medications given to the baby
    - Congenital abnormalities
3. **POSTNATAL HISTORY**
    - History of admission in nursery, NICU – duration

- Breathing problems
- Jaundice, anaemia, cyanosis
- Skin rashes or history of haemorrhage

4. FEEDING PROBLEMS

- A 24-hour dietary history is useful when assessing a feeding problem of child with signs and symptoms of malnutrition
- Breast feeding problems: poor latching, small nipples or cracked nipples
- Formula feeding: formula type, change of formula, volume, frequency, preparation and dilution of feeds
- Weaning age, solid foods and supplements and appetite
- Food intolerance, food allergy, weight gain or loss

CHILD HEALTH RECORD

1. IMMUNIZATION

Check if the immunisation is up to date on road to health booklet. Following is the current schedule of EPI South Africa

Birth	OPV (0)	BCG			
6 W	OPV (1)	DTaP-IPV // Hib (1)	Hep B (1)	PCV (1)	RV (1)
10 W		DTaP-IPV // Hib (2)	Hep B (2)		
14 W		DTaP-IPV // Hib (3)	Hep B (3)	PCV (2)	RV (2)
9 M	Measles (1)			PCV (3)	
18 M	Measles (2)	DTaP-IPV // Hib (4)			
6 Y (M/F)		Td vaccine			
12 Y (M/F)		Td vaccine			

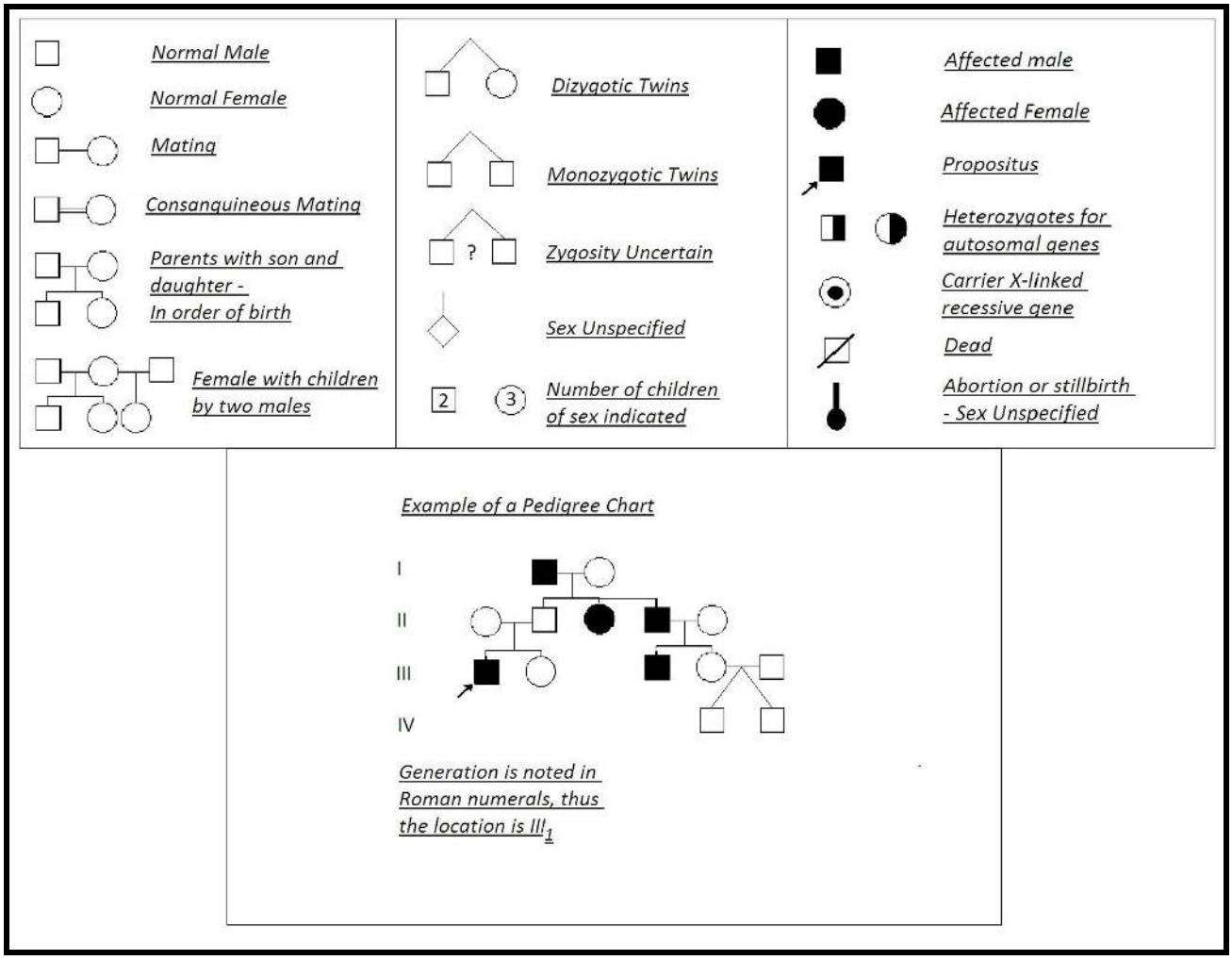
Non-EPI vaccines: MMR & Varicella: @ 15 months & Cervirex (HPV) for F @ 12 yrs.
RV vaccine is not administered after 24 months

Preterm infants: if acutely ill, vaccinations are deferred until recovered. All vaccinations are given at chronological age and not the corrected age. BCG is aslo deferred until infant is ready to be discharged. Side effects like skin rash and fever are not worse than term infants. Of note use of acellular pertussis in current EPI-SA is safe and does not cause apnoea as it was the case with cellular pertussis vaccine.

2. CLINIC VISITS: Growth monitoring, vitamin A administration and deworming

FAMILY HISTORY

Ask about the number of family members, both males and females and their health. In case of suspected heriditary or familial type e.g. haemophilia, mental retardation, and myopathies a detailed family history is required including consanguinity, pregnancy or child loss. Pedigrees are often used to determine the mode of inheritance of genetic diseases. A sample pedigree is as follows:



Diseases like hypertension, diabetes, epilepsy, certain renal and cardiovascular diseases, rheumatic fever also have familial background. Tuberculosis is very common in South Africa, thus any history of tuberculosis in the family should always be enquired.

SOCIAL HISTORY

A detailed history about the psycho-social circumstances of the family where child functions must be taken as follows:

- Primary care giver
- Occupation and employment history of parents
- Housing, sanitation and movement
- Financial situation like paternal support, grants and employment
- Long parental absences from home and support systems: family, friends
- Marital state and stability
- Psychiatric diseases, substance abuse, alcohol and smoking
- Family violence, traumatic family episodes, deaths, divorce or accidents
- Recreation as a family

The aspects listed above are sensitive, thus indirect questioning should be used. By observing family interaction and body language good amount of information can be gathered. A simple question like “what do you as a family do for recreation and leisure,” can provide sufficient information about interpersonal relationships within the family.

TUTORIAL 2

ANTHROPOMETRY AND NUTRITION

OVERALL OBJECTIVES

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

a) Take a dietary history and growth history of children
b) Use growth parameters to assess nutritional status (normal or abnormal)
c) Describe the clinical features of malnutrition
d) Recognise by appropriate history taking and examination, common and life threatening complications of nutritional deficiencies in infants and children and investigate and manage these complications.
e) Use and impart the significance of Road to Health Booklet to parents.

ANTHROPOMETRIC MEASUREMENTS

A normal distribution pattern for weight, length and head circumference has been established for every month of age in the first 2 years of life and for every 2 months untill the age of 12 years. This was done by measuring hundreds of normal children in each group. From the above range of normal measurement Z-score charts have been developed for weight, length/height, BMI and head circumference.

Weight:

The most rapid period of growth after intrauterine period is during the first 2 years of life. Average birth weight doubles by 5-6 months and tripples by 1 year. Ideally the weight of every child seen at the clinic or hospital should be plotted on a Z-score chart than trying to rely on memory. Weight is easy to measure than height and shows rapid changes. Remember that weight, but not height, can be lost therefore weight is considered to be a more sensitive measure. Idealy children should be undressed when weighed but light clothing need not to be removed and weighing scale should be checked regulary.

Average weight values at certain age groups

Age	Weight in Kg
Birth	3.5
5-6 months	7
1 Year	10
2 yrs	12

Formula to calculate an average weight from 1 year therefater: Age (in years) + 4 x 2 = Kg

For example: approximate weight of a 2 years old child: 2 + 4 x 2 = 12 kg

Length:

Length is measured by infantometer and height by stadiometer and it follows the similar pattern of weight.

Average length/height in certain age groups

Age	**Length/height in cm**
Birth	50
6 months	65
1 year	75
2 years	87
3 years	95
4 years	100

Approximate height of a child at the age of 4 years is considered as 100 cm and therafter every year should add 6 cm per year till the age of 15 years. For example a 5 years old child's estimated height will be calculated as follows: 100+6 = 106 cm

Head Circumference or Occipito-Frontal Circumference (OFC):

Head circumference is of importance as increase in head size is almost entirely dependent on brain growth. The most rapid period of brain growth is before birth and during the first into the second year of life. More brain growth occurs during the first year after birth than in the total period from 1 year of age untill addulthood. In fact most growth occurs during the first 6 months of life. If severe nutritional insult or prolonged illness occurs during this very vulnerable period "catch up" brain growth may be insufficient for the potential of that individual ever to be achieved. Measurement of OFC is also useful if some chronic brain abnormality or abnormal growth of head is suspected.

Average head circumference in certain age groups

Age	OFC in cm
Birth	35
3 months	40
6 months	44
1 year	47
2 years	50
5 yrs	52
Adult	56+

PHYSICAL GROWTH

Children grow, and if they don't, there is a problem. Their growth depends on *varuis factors*, the most important being the heriditary, genetic, environmental, emotional health, chronic diseases, endocrine and timing of puberty .

1. **Heriditary and constitutional factors:** this refers to the type of body build inherited from the parents. If both parents are small and short, the children are likely to be small whereas tall parents tend to have tall children. However, temperament, manifested as the behavioural style of the child, is influenced by child-rearing practices as well as by the continuous interaction between partents and children.

2. **Intrauterine period:** During intra-uterine life, maternal influences play an important part in the growth of the fetus. Foetal growth may be affected by maternal nutritional, socioeconomic status, habbits and medical problem.
3. **Postnatal period:** The major part of infancy is cheratcterized by a rapid growth rate, which becomes increasingly related to the genetic background. This period of rapid growth is largely determined by nutritional rather than endocrine factors.
4. **Nutrition:** Growth is profoundly affecetd by nutrition. Linear growth, expressed as length or height measurement, is a sensitive indicator of the physical health of a child.
5. **Health status:** physical health and emotional status of a child or of his family may have direct or indirect effect on his growth and development. Chronic illness often leads to disability.
6. **Socioeconomic status:** Poverty, poor education, and social adversity increases the possibility of having complications from pregnancy, birth, infancy and childhood.
7. **Cultural factors:** Child-rearing beliefs and practices vary markedly from one culture to another and have profound effect on growth and development of children.
8. **Environmental:** this includes our surroundings, and factors that can influence our life and growth. A diet which is poor in quantity or quality will prevent normal growth. This is what can often be detected on properly completed growth charts.

Poor health includes infections, as well as any serious disturbances of physiological function such as heart disease or severe congenital abnormalities. If the vital organs will not function properly, the child will not grow normally. Health and nutrition influence each other. In turn, unhealthy child may become malnourished and the poorly nourished child suffers with more frequent infections. Thus a serious cycle is set in motion, the one factor aggravating the other.

Sprcific factors may be affecting at specific age groups:

- Infancy (upto two years): Food, nutrition and chronic diseases
- Childhood: Genes, growth hormones and chronic diseases
- Puberty: Sex hormones

Types of growth charts

- Road-to-Health Booklet growth charts: contains Weight for age & Height for age
- Longitudinal growth charts: Percentile graphs & Z-Score graphs
- BMI charts
- Weight for length/height charts
- Growth velocity Charts
- Combined prenatal and postnatal growth charts
- Growth charts for special populations: Down syndrome, Turner syndroms
- WHO Anthro: for personal computers – software for assessning growth and developemnt of world's children. www.who.int/growth

Uses and advantages of growth charts

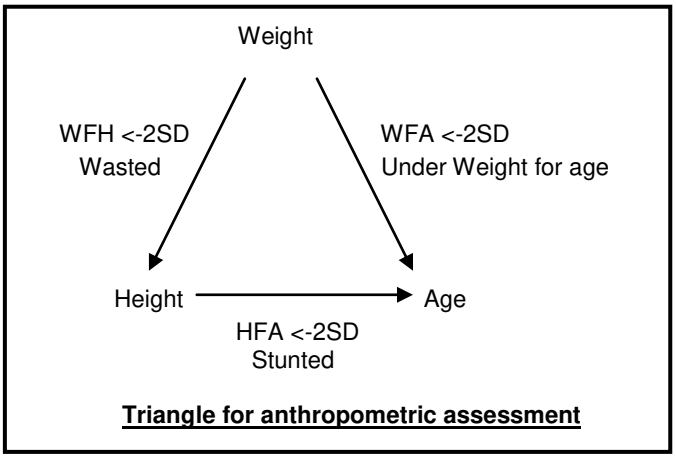
1. **To check whether the child falls into the normal range** for his age. Many children are found whose weight falls below -2SD line. They do not necessarily look ill or obviously malnourished. They are just small for their ages and if their weights were not plotted on growth charts, they would have not been noticed. These children are underweight for age (UFA) and represent the largest group of undernutrition. Steps must be taken to improve their nutrition because they are more likely to develop infections and obvious malnutrition.
2. **Recording growth repeatedly at intervals**, gives the most valuable information as to whether the rate of growth is satisfactory. The child's charted weight graph should run parallel to one of the normal growth lines given on the graph i.e parallel growth = good (normal) growth.

Z-SCORE GROWTH CHARTS

Measurement is the only way to recognise whether growth is normal or not. In order to record child's growth and physical development, special Z-Score growth charts are used in South Africa.

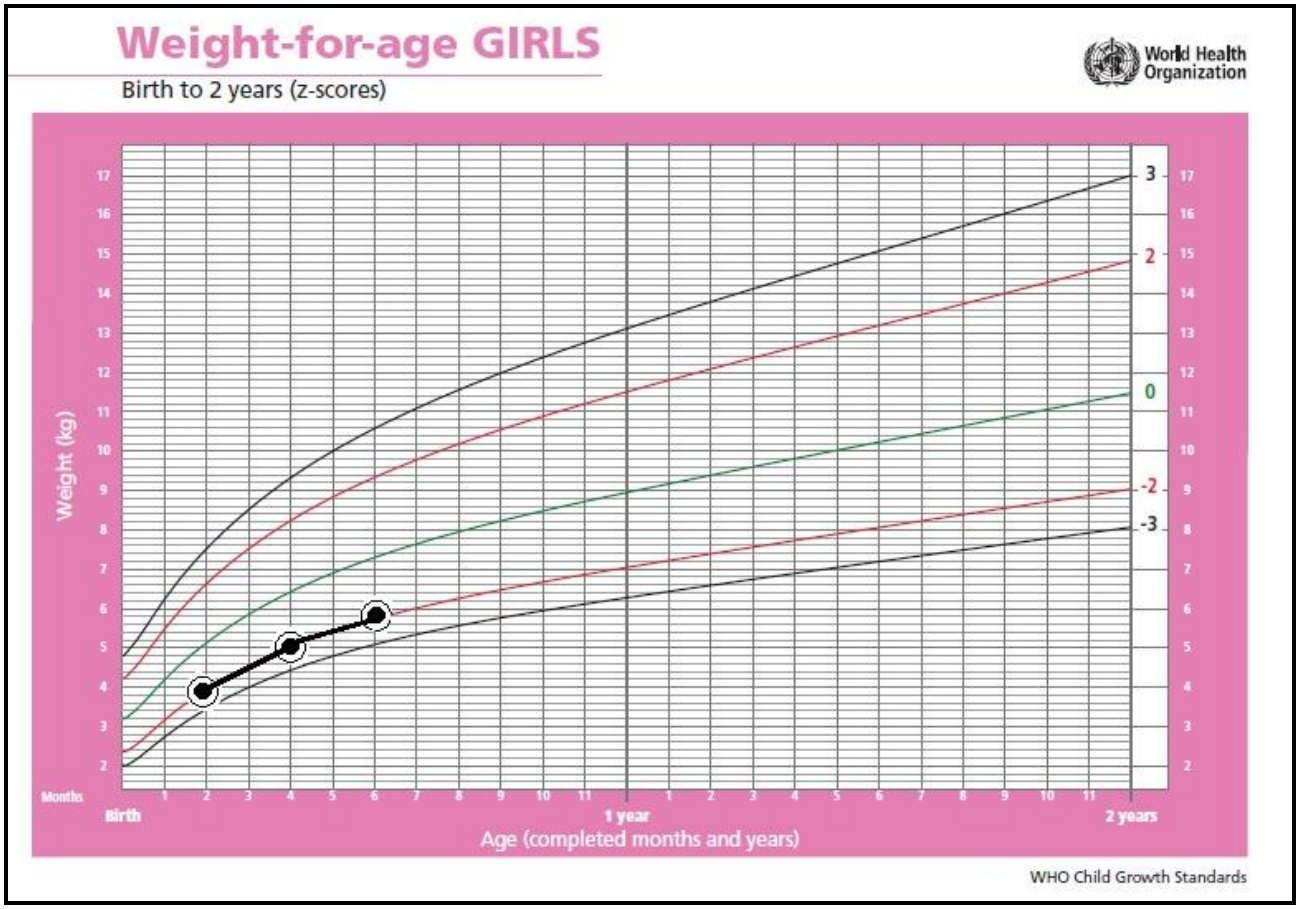
In a graph one factor is plotted against another but related factor e.g. wight for age, weight for height and height for age. Repeated observations are recorded at regular intervals. Following triangle for anthropometric measurements is helpful tool for plotting the variables on the growth charts.

- Weight for age (WFA Z score)
- Weight for height (WFH Z score)
- Height for age (HFA Z score)



Triangle for anthropometric assessment

Increasing age should be accompanied by increase in weight. Following graph shows this and the rate at which it occurs. Increase in weight by itself is not enough as the time period over which it occurred must also be taken into account.



Explanation of above graph:

A graph is made indicating the baby's growth.

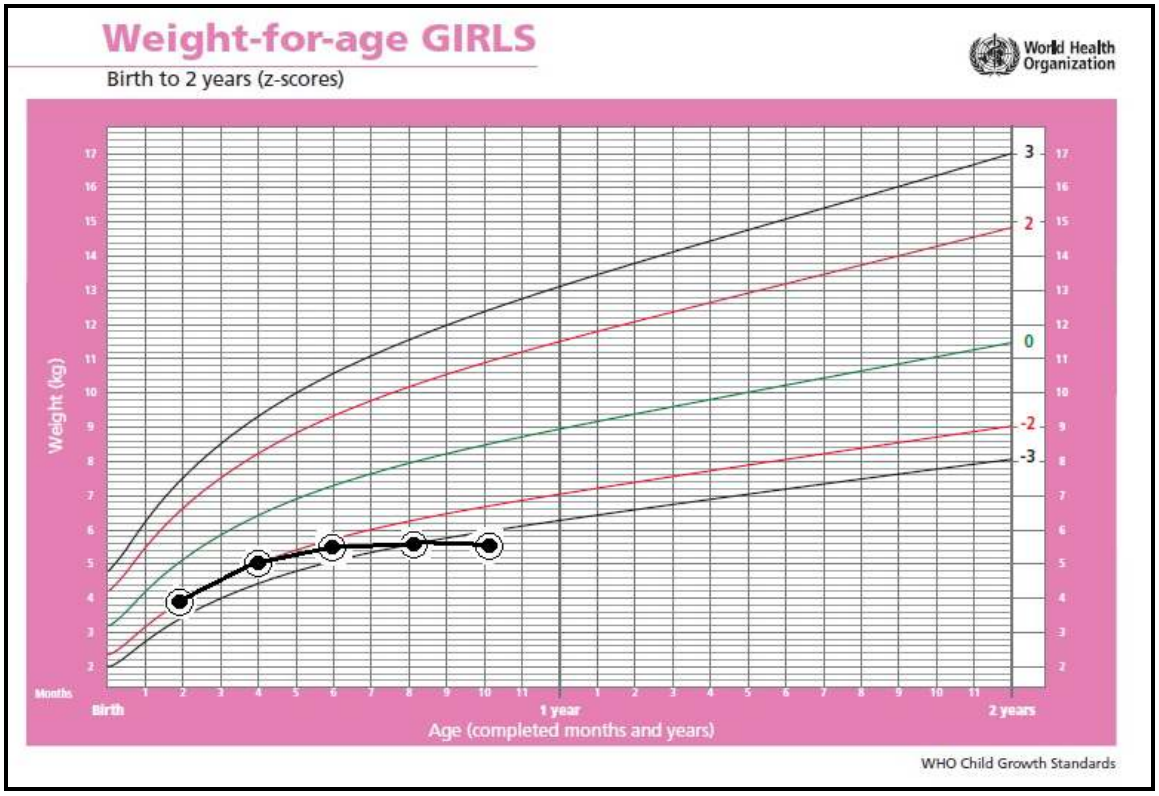
When seen at 2 months female baby is found to have a weight of 4kg. Following a straight line up from 2 months and across from 4 kg a point can be plotted.

At the age of 4 months the baby weighs 5 kg and at 6 months 6 kg. Find and mark these points on the graph and then join these points. In a similar way height and age or head circumference and age can be plotted.

Babies grow very rapidly in the first year of life. A normal infant should double its birth weight by 5-6 months and it should triple by the age of 1 year. In later childhood growth is generally slower but increases again markedly at puberty.

Human beings of any given age are not all the same size even though they may be perfectly normal and healthy. It can therefore not be said that a normal newborn should weigh 3.5 kg. There is always a range of normal and in this case, it is from 2.5 to 4 kg.

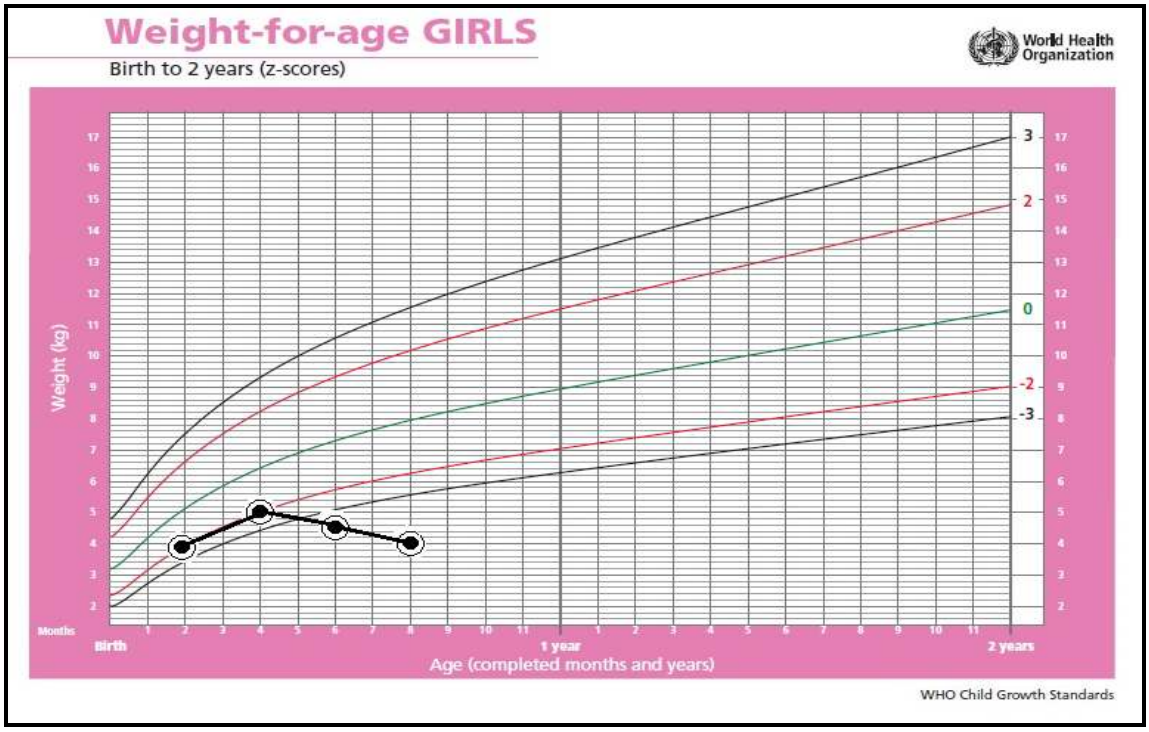
It is difficult to remember all these normal variations for all ages; therefore, growth charts are used with normal lines showing both the normal range and the rate of growth.



Graph above showing abnormal growth pattern

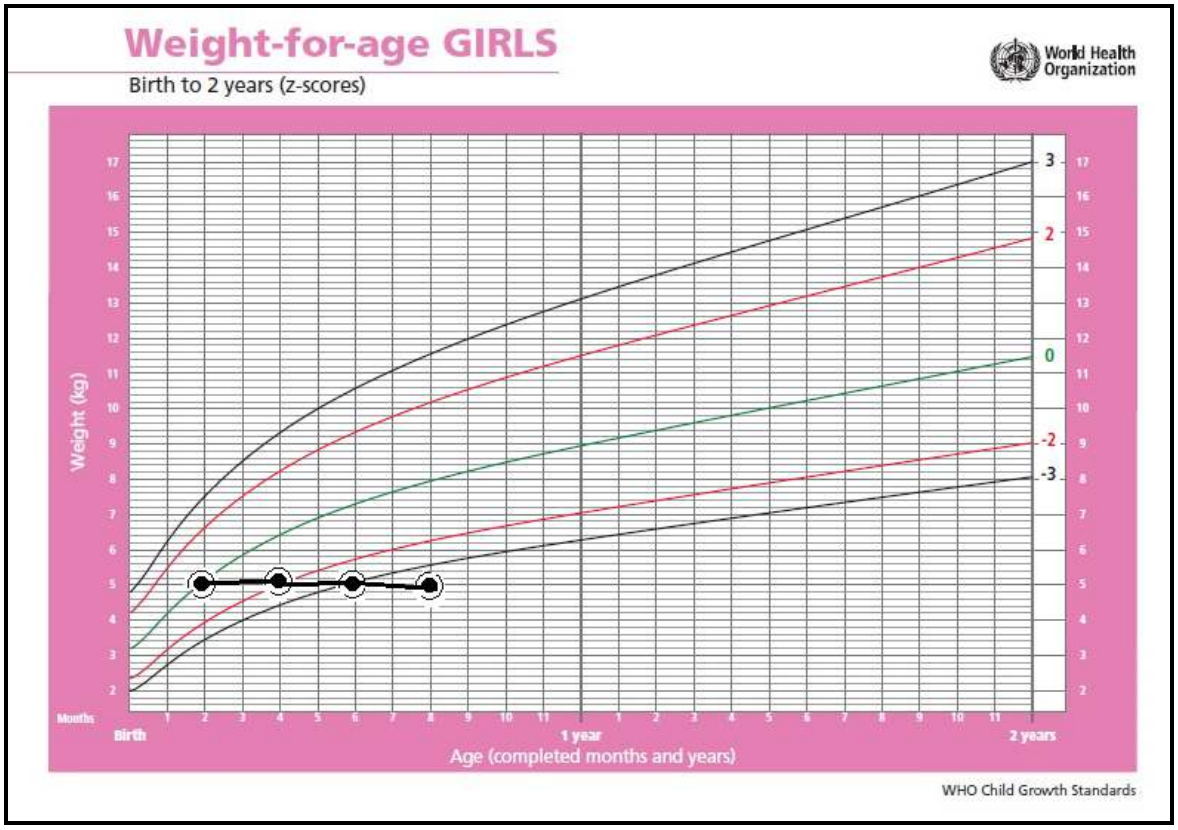
If a child was supposed to gained 1 kg over a certain period of time but has only gained ½ kg, this is slowing of growth, and is an early important sign of inpending malnutrition.

Following graph The example is the following graph



1. Abnormal growth pattern of weight loss

Graph above showing well-nourished infant showing loss of weight. The continued recording of weight at intervals is of far greater value than any single recording. Regular recording of weight will show progress the child is making, i.e., pattern of growth.



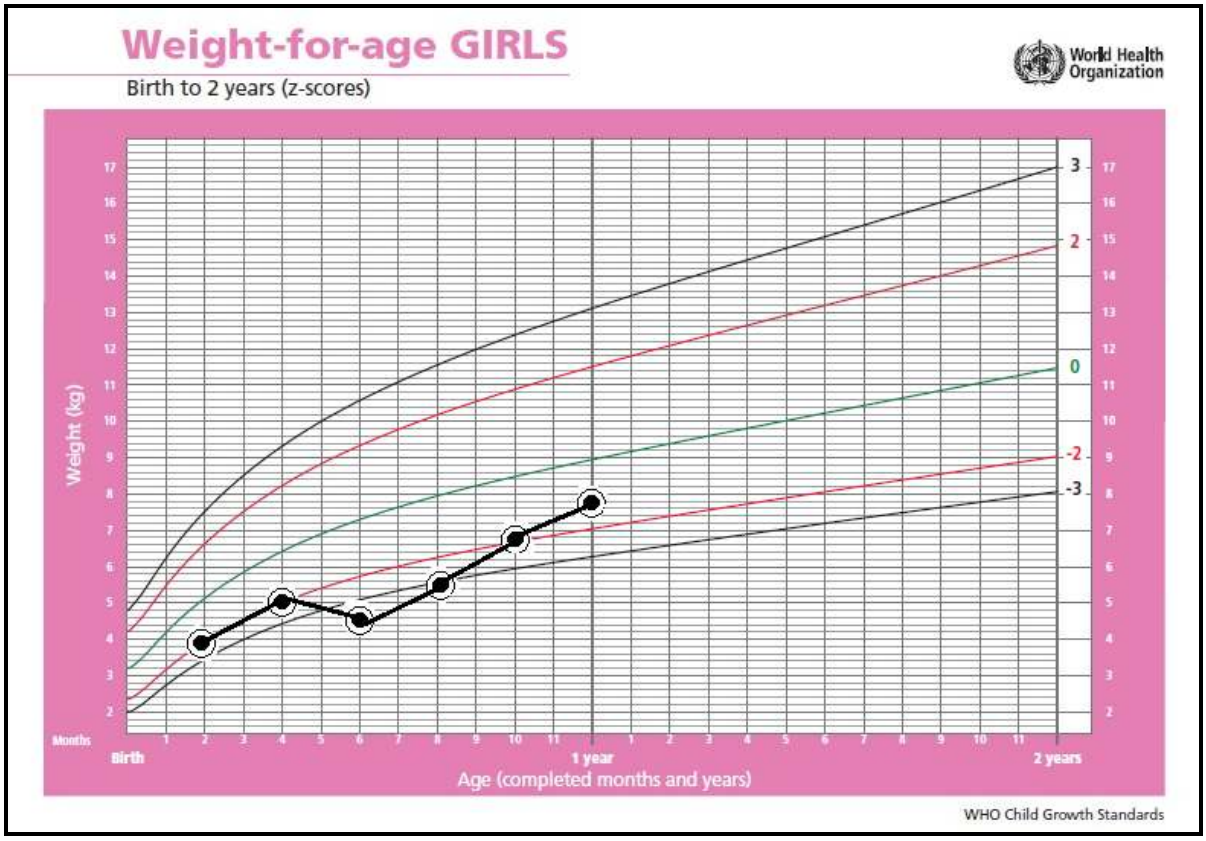
2. Abnormal growth pattern of poor weight gain

Graph above showing poor weight gain leading to malnutrition. Plotting once off on the weight chart may be misleading as it gives no indication of direction whereas repeated recordings give both the direction and adequacy of weight curve which is more important in assessing progress. Stopping breast feeding and giving only porridge may be seen as plotted in the above graph.

While most children with severe acute malnutrition (SAM) with oedema have weight for age below -2SD, fluid may falsely increase the weight. A gain of 500g due to fluid retention will however show as obvious oedema. Since SAM with oedema can develop rapidly, the child may still be within normal z-scores but will have deviated from his previous growth curve.

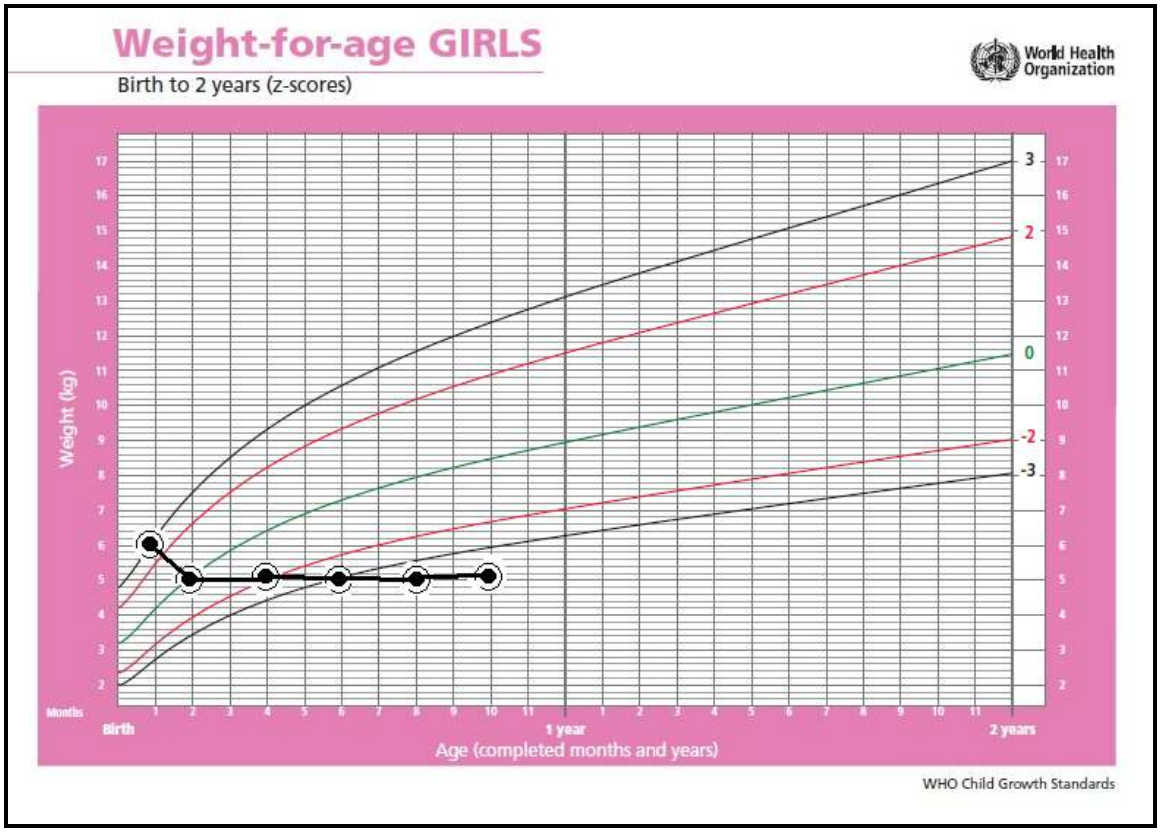
3. Infection and growth:

In babies who are weighed regularly and growth charts used, the frequent relationship between infection and poor growth is noted. Every time an infant suffers from gastro-enteritis, a respiratory tract infection and particulaly having measles, there will be weight loss. As poorly nourisehd children tend to get more frequent infections, so one problem leads to other. If recovery after an illness is satisfactory and complete, there should be a rapid regaining of the lost weight i.e. “catch up growth” so that the child continues again on his previous normal growth line.



Graph above indicates catch up growth

If this catch up growth doesn't occur, e.g. after measles, suspects some complication. In this case the probability of TB, which is common after or reactivated by measles. This can be picked up on the growth chart and early diagnosis or even permanent brain damage from TB meningitis.

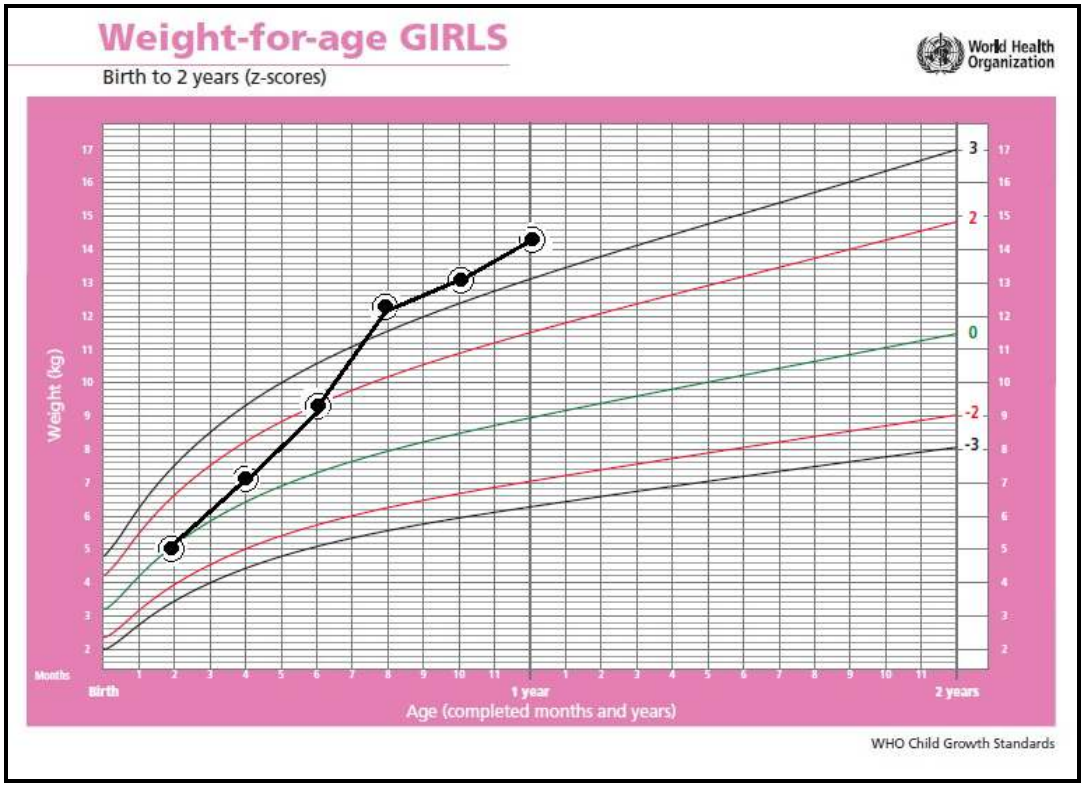


Graph above shows failure to regain weight and return to normal growth line.

If weight gain is inadequate, check for underlying disease, enquire about diet (type and frequency of feeds, and advise about food supplements). If above attempts to promote growth are unsuccessful – consider underlying infection or chronic illness.

4. Excessive growth

In infancy, particularly with artificial feeds and the early introduction of excessive amounts of cereals, a rapid and marked weight gain is undesirable and obesity may develop. This may persist into adult life causing problems such as hypertension, diabetes and cardiac disease and should be avoided.



Above graph growth shows pattern of excessive growth

NB! Standard Road to Health Booklets should be used for all babies wether rich or poor, state or private health care system. The same comprehensive record must be used to record not only growth but immunization, milestones of development, and other important information. Road to heath bookelt is an important document and parents should be encouraged to take it with them when they go for a clinic or a hospital visit.

In summary, the growth charts in road to health booklet can be used to;

- Determine and promote adequate growth
- Detect children at risk of malnutrition
- Introduce comprehensive health care to the family

In a busy clinic or POPD, can time be found for all this? Time must be found!

EXAMINATION OF A CHILD FOR ANTHROPOMETRIC ASSESSMENT

Look for obvious clinical features

- Child with visible severe wasting
- Child with generalised oedema
- Or is this is a chubby child

Measure the child's weight by relaible weighing scale

- Small infants with baby scale
- Older children with standard scale

Measure the child's

- Length: supine with infantometer while head position is in vertical Frankfurt plane

- Height: standing up with stadiometer while head position is in horizontal Frankfurt plane

Frankfurt plane: Frankfurt plane is an imaginary line from the center of the external auditary meatus to the lower border of the eye. It should be vertical in supine position and horizontal in standing position while measuring the length or heigth respectively.

Measure child's occipito-frontal circumference (OFC) with measuring tape. Plot the child's weight, height, and head circumference on the growth charts provided. Before plotting make sure that measurements do make sense to you and you have the correct growth charts in hand.
Separate growth charts are available for:

- VLBW infants (wt <1500g)
- Turners & Downs Syndromes
- Achondroplasia

Plotting

- Plot completed weeks, months, years on the vertical line not in between lines like 5½ month – plot at 5 months
- Plot length or height on or between horizontal lines precisely
- Judge if the plotted point seems sensible

Z score lines on growth charts are numbered:
Positively +1, +2, +3 or
Negatively -1, -2, -3

Plotted point that is far from Median in either direction i.e. close to +3 or -3 Z score, may represent growth problem, although other factors should be considered like:

- Growth pattern on road to health booklet i.e. acute drop in weight, plateau pattern or crossing of a Z score
- General health of the child
- Height and weight of parents in case of abnormality noted in child's anthropometry

Crossing a Z score line

- If child's line crosses a Z score line, it means there has been a significant change in the child's growth:
    - If shift is towards a higher median – it is possibly a good change
    - If shift is away from the median above – this signals a problem or a risk

Growth Patterns

- If child has been ill or severely undernourished, a sharp incline is expected during re-feeding period as the child experiences catch-up-growth. Otherwise sharp incline is not good which may indicate change in feeding practices, which may result in the child being overweight.

- If a child has gained weight rapidly, then look at the height. If there is no change in height then there may be a problem of overweight
- Flat growth line is called stagnation which indicates a problem
- For children in age groups where the growth rate is fast as shown by steep growth curves i.e. during 1st 6 months of life, even one month's stagnation in growth represents a problem.

BODY MASS INDEX

BMI is an index for classifying adiposity and is recommended as a screening tool for children and adolescents to determine wether an individual is overweight. Growth charts for ploting of BMI are also available.

Calculate BMI especially in children >5 years old as follows and plot on the growth charts:

BMI = Weight / Height^2

BMI	
Under weight	<18.5
Normal	18.5 – 24.9
Overweight	25 – 29.9
Obese	30 – 39.9
Morbid obese	≥40

MUAC

- MUAC is measured at a mid-point between acromion and olecranon
- Measurement is usually done every 3 months from 6 months of age
- If MUAC is <11.5 cm, child is classified as SAM and must be referred to a hospital for assessment & treatment
- If MUAC is 11.5 - <12.5 cm and child has no oedema, this indicates moderate acute malnutrition and child can be managed as an outpatient according to IMCI guidelines.

MUAC REFERENCE RANGE ACCORDING TO AGE (IMCI GUIDELINES)

Age of patient	< -3 Z score Sever acute malnutrition	< -2 Z score Moderate acute malnutrition
6 – 12 months	<11.0 cm	<12.0 cm
1 – 5 yrs	<11.0 cm	<13.0 cm
6 – 9 yrs	<13.5 cm	< 14.5 cm
10 – 14 yrs	<16.0 cm	< 18.5 cm

Classification of malnutrition according to WHO

WHO classification				
Assessment	**Weight for age**	**Weight for Height**	**MUAC**	**Other**
Moderate acute malnutrition	WFA Z score < -2 SD	WFH Z score < -2 SD	Yellow band 11– 12.5 cm	
Sever acute malnutrition	WFA Z score < -3SD	WFH Z score < -3 SD	Red band < 11 cm	Visible wasting or oedema of both feet
	Under wight for age (UWFA)	Wasting	Wasting	SAM with oedema

HEAD CIRCUMFERENCE

- Measure the OFC of your patient and plot it on the growth chart
- Comment as microcephaly (Z-score <2SD) or macrocephaly (Z-score >2 SD)
- Small OFC - parents may be of smaller head size (genetic heritage). Baby's brain may not be growing well during early crucial period of brain growth spurit that lasts until 2 yrs of age which can lead to microcephaly due to failure of brain development.
- OFC can be small in sever chronic malnutririon but increased in vitamin D deficiency

CAUSES OF MICROCEPHALY (Small head)

1. Trisomies 21, 13, 18
2. Intrauterine infections like TORCH, syphilis, HIV, varicella
3. Head injury, perinatal hypoxia, stroke
4. Post meningitis, encephalitis
5. Teratogens like radiations or alcohol (foetal alcohol syndrome)
6. Undernutrition or chronic illness (renal, cardiac, pulmonary disease)
7. Hypothyroidism or hypopituitarism
8. Agenesis of carpus callosum
9. Craniosynostosis totalis
10. Grey matter dx, White matter dx

CAUSES OF MACROCEPHALY (Big head)

1. Rickets (vitamin D deficiency)
2. Osteogenesis imperfecta
3. Galactosaemia
4. Leukodystrophy
5. Head injury, haematoma (NAI)
6. Infections
7. Perinatal infections like TORCH, syphilis, HIV
8. Post natal infections like meningitis, encephalitis
9. IVH, Stroke or intravascular haemorrhage
10. Tuberous sclerosis, Sturge Weber Syndrome

Organs which are obligate glucose users

- Brain
- Errythrocytes
- Cells of renal medulla

TYPES OF HYDROCEPHALUS WITH CAUSES

1. **Obstructive hydrocephalus**
   - Acqueduct stenosis
   - Spina bifida
   - Dandy-Walker malformation
   - Brain tumors
2. **Non-obstructive hydrocephalus**
   - Fibrosis secondary to haemorrhage
   - Brain malformations
   - Neurofibromatosis
   - Tuberous sclerosis
   - Papilloma of choroid plexus

ASSESSMENT OF ANTHROPOMETRY

- If the child has oedema or ascities it does not reflect the true weight so oedema and ascities need to be resolved for the assessment of true weight
- If stunted or wasted: interprete as a chronic problem
- If the weight and height are both normal look for other signs of chronic disease like clubbing and say even though anthropometry is normal, child still shows signs of chronic disease.

Now see the trend of growth in the Road to Health Booklet

- Common finding is poor weight gain
- Height of the child can be significantly small in:

1. Chronic disease
2. Hypothyroidism
3. Hypopituatarism
4. Chronic malnutrition
5. Zinc deficiency
6. Rickets
7. Achondroplasia

INTERPRET THE GOWTH PARAMETERS

Z-scores	Growth indicators			
	Length/height for age	**Weight for age**	**Weight for length/height**	**BMI for age**
Above 3	(See note 1)	(See note 2)	Obese	Obese
Above 2			Overeweigth	Overweight
Above 1			Possible risk of overeweight (See note 3)	Possible risk of overweight (See note 3)
0 (median)				
Below -1				
Below -2	Stunted (See note 4)	Underweight	Wasted	Wasted
Below -3	Severely stunted (See note 4)	Severly underweight (See note 5)	Severely wasted	Severely wasted

Notes:

1. A child at this range is very tall. Tallness is rarely a problem, unlessnit is so excessivetahtit may indicate an endocrine disorder such as growth hormone tumor. Refer a child in this range for assessment if you suspect an endocrine disorder (i.e. if parents of normal height have a child who is excessively tall for his or her age).
2. A child whose weight for age falls in this range may have a growth probplem, but this is better assessed from weight for length/height ot BMI for age.
3. A plotted point above 1 shows possible risk. A trend towards the 2 z-score line shows definite risk
4. It is possible for stunted or severely stunted child to become overweight.
5. This is referred to as very low wight in IMCI training modules.

Make your final assessment:

- Child has normal anthrpomerty and normal growth pattern
- Child has severe or moderate malnutrition or at risk of malnutrition
- It is acute or chronic malnutrition

TUTORIAL 3

MICRONUTRIENTS

OVERAL OBJECTIVES

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

a) Take a dietary history and growth history of children
b) Describe the signs and symptoms of micronutrient deficiency
c) Recognise by appropriate examination, common and life threatening complications of micronutrient deficiencies in infants and children and investigate and manage these complications.

Signs & symptoms of micronutrient deficiency

The specific examination of child, who grows poorly, needs full clinical examination focusing on the cause, degree and complications of malnutrition. It is therefore important, to learn the signs and symptoms of deficiency of individual vitamins, minerals and trace elements.

Classification of micronutrients

1. Vitamins
    a. Fat soluble vitamins
    b. Water soluble vitamins
2. Minerals
3. Trace elements

1. VITAMINS

Vitamins are organic dietary constituents necessary for life, health, and growth that do not function by supplying energy. These are the substances that act as co-enzymes and are regulators of metabolic processes.

a. Fat soluble vitamins

i. Vitamin A: Retinol

It is constituent of visual pigments, necessary for foetal development and for cell development throughout the life.

Signs of deficiency

- Night blindness
- Xerophthalmia – dry eyes due to inadequate production of tears
- Conjuctival xerosis – dryness of eye membrane
- Corneal xerosis – dryness of cornea
- Keratomelacia – softening of cornea – seen in infancy
- Bitots spots in school children
- Follicular hyperkeratosis of skin

Signs of hypervitaminosis

- Irritabilty & anorexia
- Headache and bone pain
- Hepatosplenomegaly
- Scaly dermatitis & patchy loss of hair

ii. Vitamin D: Cholecalciferol

It increases intestinal absorption of calcium and phosphate and promotes normal bone formation and mineralization

Signs of deficiency

- Rickets:
    - Epiphyseal enlargement – painless – over 6 months of age
    - Beading of ribs – rachitic rosary
    - Bowed legs and knocked knees
- Persistanty open anterior fontanelle after 18 months
- Frontal bossing
- Craniotabes < 1 yr
- Delayed dentician/poor dentition
- Rachitic tetany caused by hypocalcaemia

Signs of hypervitaminosis

- Weight loss
- Calcification of many soft tissues, and eventual renal failure

iii. Vitamin E: Tocopherol

- Cholestasis and Cystic fibrosis are important causes of deficiency
- Clinical deficiency does not appear until 1 year of age, even in children with cholestasis since birth
- It functions as an anti-oxidant; precise biochemical functions unknown
- It is also a co-factor in electron transport in cytochrome chain

Signs of deficiency

- Loss of deep tendon reflexes
- Limb ataxia, intention tremor, dysdiadochokinesia
- Truncal ataxia: wide based unsteady gait
- Ophthalmoplegia: limitation of upward gaze
- Nystagmus
- Decreased proprioception: positive Romberg test
- Visual field constriction: may progress to blindness
- In premature infants: haemolysis of RBCs during 2nd month of life
- Haemolytic anaemia in preterm infants: oedema may also be present

- Young children with congenital cholestasis: a genetically inherited rare disease – familial isolated vitamin E (FIVE) deficiency. Despite normal lipid absorption they have low undetactable level of plasma vitamin E. Reduced tendon reflexes occur at 3-4 yrs of age and disabling cerebrospinal symptoms occur by early adolescence.

iv. Vitamin K: Phytomenadion

It catalyzes gama carboxylation of glutamic acid residues on various proteins concerned with blood clotting. It is essential for production of coagulation factors: 2, 7, 9 , 10 and other factors like protein C, S, Z

Signs of deficiency

- Easy bruisability: look for patechiae and bruising
- Conjunctival haemorrhages
- Mocosal bleeding
- GIT haemorrhage, haematuria and epistaxis

Signs of hypervitaminosis

- Gastrointestinal disturbances
- Anaemia

b. Water soluble vitamins

i. Vitamin B1: Thiamine

It is cofactor for decarboxylations.

Signs of deficiency

Beriberi & neuritis

- **Dry beriberi**
  - Pain - tingling or loss of sensation in hands and feet – peripheral neuropathy
  - Muscle wasting – loss of function and paralysis of lower extremities
- **Wet beriberi**
  - Oedema - lung congestion
  - Cardiomegaly – Congestive cardiac failure (CCF)
- **Infantile beriberi**
  - Convulsions and acute cardiac failure in early months of life

ii. Vitamin B2: Riboflavin

It is constituent of flavoprotein enzymes which are important in oxidation reduction reactions.

- It act as co-enzyme in carbohydtrate metabolism
- It is also needed to process fats and amino acids
- It activates vitamin B6 and folic acid

Signs of deficiency

- Cataracts – corneal vascularization – blurred vision
- Burning and itching of eyes
- Reddening of lips – cheilosis
- Glossitis – magenta tongue
- Delayed mental response

iii. Vitamin B3: Niacine

It is the constituent of NAD & NADP: coenzymes in numerous oxidative reduction reactions.

Signs of deficiency

- Pallegra
- 3D: diarrhoea, dementia, dermatitis
- Tongue fissuring
- Malar and supraorbital pigmentation
- Hyperpigmented skin rash in sun exposed areas

iv. Vitamin B5: Pantothenic acid

It is an important component of co-enzyme A and Acyle carrier protein involved in fatty acid metabolism.

Signs of deficiency

- Dermatitis
- Alopecia
- Numbness
- Paresthesias
- Muscle cramps
- Adrenal insuffficiency

v. Vitamin B6: pyridoxine

It forms prosthetic group of certain decarboxylases and transaminases and gets converted in body into pyridoxal phosphate & pyridoxamine phosphate. It is also constituent of co-enzymes for fatty acid metabolism, haem synthesis and is essential for homocystein metabolism.

Signs of deficiency

- Peripheral neuritis in patients receiving INH
- Convulsions in infants
- Hyperirritability
- Hypochromic anaemia

vi. Vitamin B9: Folic acid

- It is important for synthesis of DNA, RNA and nuclear proteins
- It is used in the metabolism of homocysteine so its deficiency leads to accumulation of homocystein in the body

Signs of deficiency

- Sprue
- Pale conjunctiva: megaloblastic anaemia
- Congenital abnormalities in the new born: spina bifida
- Coronary artery disease and stroke: due to accumulation of homocysteine

vii. Vitamin B12: Cyanocobalamine

It act as coenzyme in amino acid metabolism & stimulates erythropoiesis. It plays an important role in DNA synthesis and neurological function.

Signs of deficiency

- Pale conjunctiva: megaloblastic anaemia
- Loss of appetite intermittent diarrhoea or constipation
- Numbness and tingling sensation in hands
- Optic neuritis: optic nerve inflammation
- Romberg's sign
- Jaundice

B1, B6, B12 deficiency: main features: peripheral neuropathy and decreased knee & ankle jerks

viii. Biotin

It catalyzes carbon dioxide "fixation" (in fatty acid synthesis).

Signs of deficiency

- Dermatitis
- Enteritis
- Seborrhoea
- Alopecia
- Conjunctivitis

ix. Vitamin C: Ascorbic acid

- It maintains prosthetic metal ions in their reduced form and scavenges free radicals.
- It protect folic acid reductase, which converts folic acid to folinic acid

- It maintains non-haem Fe absorption and Fe transfer from transferrin to ferritin

Signs of deficiency

- Scurvy: haemorrhagic manifestations and abnormal bone and dentine formation
- Spongy and bleeding gums, infection, gangrene, losening of teeth at later stage
- Slow healing of wounds
- Bowing of long bones epiphyseal enlargement (painful)
- Haemorrhages under the skin intramuscular and subperiosteal haematoma
- Follicular hyperkeratosis

Infentile scurvy

- Haematoma formation
- Painful epiphyseal enlargement especially at costochondral junction
- Spongy bleeding gums: does not occure in the absence of teeth

2. MINERALS

i. Calcium

- It has important role in cell permeability, bone and teeth formation
- It is essential for blood co-agulation, transmission of nerve impulses and normal muscle contraction

Signs of deficiency

- Irritability – reptitive muscle spasms
- Twitching corpopedal spasm tetany
- Cardiac arrythemias
- Chvostek's sign / Troussea's sign
- Decreased muscle tone – weakness – lethargy

ii. Phosphorous

- It forms structure of teeth, bones and cell membrane
- It is an important component of ATP and forms RNA and DNA
- Deficiency of Phosphate can cause reduction of 2,3 DPG levels in RBCs which decreases the oxygen delivery to tissues. It can also cause Haemolysis & dysfunction of white blood cells

Signs of deficiency

- Rickets
- Delayed dentition and tooth abscesses
- Short stature without clinical evidence of bone disease
- Rhabdomyolysis

- Proximal muscle weakness and atrophy
- Cardiomegally / cardiac failure

iii. Magnesium

Hypomagnesaemia causes secondary hypocalcaemia by imparing release of PTH by parathyroid gland and through blunting the tissue response to PTH.

Signs of deficiency

- Tetany
- Positive Chvostek and Trousseu signs
- Seizures

iv. Sodium

Most of the body sodium is located in blood and in the fluid round cells. It helps the body keep fluids in a normal balance, plays a key role in normal nerve and muscle function.

Signs of deficiency

Brain cell swelling is responsible for most symptoms

- Anorexia, nausea and vomiting
- Confusion and lethargy
- Headach, seizures and coma and decreased reflexes
- Cheyn Stokes respiration
- Muscle cramps and weakness
- Chronic hyponatraemia may be asymptomatic: owing to a compensatory decrease in brain cell osmolality which limits brain swelling

v. Potassium

It helps regulate fluid balance, muscle contractions and nerve signals.

Signs of deficiency

- Ventricular fibrillation
- Skeletal muscle weakness and cramps
- Paralysis: if potassium < 2.5 – starts with legs followed by arms
- Respiratory paralysis: some may develop rhabdomyolysis
- Constipation: ilius, due to slow GIT motility
- May cause polyuria by producing secondary nephrogenic diabetes insipidus

3. TRACE ELEMENTS

By definition a trace element is the one which is required in <0.01% of the body weight to maintain normal body growth

i. Copper

It is the component of enzymes involved in:

- Energy production through cytochrome oxidase
- Protection of cells from free redical damage through supperoxide dismutase
- Brain neurotransmitters through dopamine hydroxylase
- Also involved in absorption, storage and metabolism of iron

Signs of deficiency

- Microcytic anaemia
- Neutropenia
- Osteoporosis
- Depigmentation of hair and skin

ii. Iron

It is the main component of haemoglobin and myoglobin.

Signs of deficiency

- Pale conjunctiva: Fe deficiency anaemia
- Kiolonychia: spoon shaped nails – in older children and adults
- Toungue – atrophic lingula papillae
- Angular stomatitis
- Decreased alertness
- Impaired learning

iii. Zinc

It is essential for body's immune system. It plays a role in cell devision, cell growth, wound healing and breakdown of carbohydrates. Its supplementation is beneficial in diarrhoea and to improve neurodevelopmental outcome.

Signs of deficiency

- Hypogonadism: delayed puberty
- Skin changes: particularly associated with a rash – dermititis of extremities and around orifices
- Impaired immunity, poor wound healing
- Diarrhoea
- Mental lethargy

iv. Manganese

It is an enzyme co-factor. It is essential for connective tissue, bones, clotting factors and sex hormones. It is also necessary for normal brain & nerve function and blood sugar regulation.

Signs of deficiency

- Hypercholesterolaemia
- Decreased clotting proteins
- Memory disturbance
- Tiredness: mysthenia gravis
- Ataxia: fainting
- Hearing loss

v. Iodine

Component of thyroid hormone

Signs of deficiency

- Goiter
- Coarse facies, dry skin
- Lethargy
- Jaundice
- Large tongue
- Bradycardia
- Wide posterior fontanelle
- Hypothermia
- Constipation

vi. Fluroide

It is the main constituent of bone and forms an active componenet of teeth enamel.

Signs of deficiency

- Brittle bones
- Dental caries

[enamel defects (vit D), looseness (vit C)]

vii. Selenium

It plays a role in metabolism & thyroid function and prevents oxidative damage

Signs of deficiency

- Myopathy
- Fatigue
- Hyperthyroidism
- Lack of mental dexterity
- Keshan disease: it is congestive cardiomyopathy (myocardial necrosis which leads to weakneing of the heart muscle) caused by a combination of dietary deficiency of selenium and the presence of a mutated strain of Coxsackievirus.

EXAMINATION OF A CHILD FOR MICRONUTRIENT DEFICIENCY

After full anthropometric assessment child need to be assessed for specific deficiencies of micronutrients including vitamins, minerals and trace elements. Expose the child from head to toe. Put up the nappy or undergarments after examining the genitalia and perineum. Avoid undue and long exposure.

Following clinical signs may be noted in micronutrient deficiency

Hair changes	Reddish brown, brittle and sparse hair	Sever acute malnutrition
	Depigmentation of hair and skin (Kinky hair)	Copper deficiency
Skull	Microcephaly	Chronic malnutrition
	Bossing of skull Craniotabes Delayed closure of fontanels	Vitamin D deficiency
Eye changes	Night blindness Xerophthalmia Xerosis conjunctivae Xerosis cornia Keratomalacia Corneal perforation	Vitamin A deficiency
Mucous membranes lesiosns	Angular stomatitis Atrophic glossitis	Fe deficiency
	Reddish cracks in lips (cheilosis)	Vitamin B2 defiency
	Swolen, spongy and bleeding gums	Vitamin C deficieny
	Patechiae Mocosal bleeding	Vitamin K deficiecny
Tongue	Smooth reddish tongue (Glossitis)	Severe acute malnutrition Vitamin B2 deficiency Pellagra
Teeth	Brittle bones and dental caries	Floride deficiency
Neck	Goiter Coarse facies Large tongue Bradycardia Wide posterior fontanelle or delay in closure of fontaneles	Iodine deficiency Hypothyroidism
Chest	Rickety-rosary Harrison sulcus	Vitamin D deficiency Phosphate deficiency

	Pectus carinatum or Pectus excavatum	
Skeletal abnormalities	Broadening of wrists & ankles Kyphosis, scoliosis or lordosis Legs: X or O or laterlised deformaity Tetanic spams	Vitamin D deficiency Phosphate deficiency
	Loss of deep tendon reflexes Limb ataxia Truncal ataxia	Vitamin E deficiency
	Peripheral neuropathy and decreased knee & ankle jerks Numbness and tingling sensation of hands	Vit B1, B6 and B12 Deficiency
	Abnormal bone and dentine formation Pseudoparalysis Tender epiphyseal enlargement	Vitamin C deficieny
	Twitching & corpopedal spasm (tetany) Chvostek's sign Troussea's sign	Calcium and or magnisium deficiency
	Skeletal muscle weakness and muscle cramps	Potassium deficiency
Skin	Follicular hyperkeratosis (shouldrs, gluteal area and extensor surfaces of extremities	Vitamin A deficiency
	Hyperpigmentation of sun exposed parts	Nicotinic acid deficiency: Pellegra
	Depigmentation or hyperpigmentation of flanks with wet and raw lesions in diaper area	Sever acute malnutrition
	Easy bruisability Patechiae and bruising Conjunctival haemorrhages Mocosal bleeding	Vitamin K deficiecny
	Poor wound healing Hypogonadism Delayed puberty Impaired immunity Mental lethargy	Zinc deficiency
Palmer pallor	Pale palms Pale conjunctivae	Fe, folate or B12 deficiency anaemia
Nails	Brittleness of nails	Fe deficiency

	Kiolonychia: spoon shaped nails (in older children and adults)	
Organomegaly	Hepatomegaly	Severe acute malnutrition
	History of pica & Spleenomegaly	Fe deficiency
Non specific symptoms	Irratability Loss of appetite Easy fatiguability Decreased alertness Impaired learning	Fe deficiency Vitamin D deficiency Vitamin C deficiency Sever acute malnutrition

Lastley make summary of your assessment and exclude Refeeding Syndrome (RFS) in the child if he is already receiving treatment.

Refeeding Syndrome (RFS)

- There is no agreed international definition of RFS.
- It is a syndrome of severe electrolyte and fluid shifts associated with metabolic abnormalities in malnourished patients undergoing refeeding, whether orally, enterally, or parenterally
- Hypophosphataemia is the adopted surrogate marker for diagnosing RFS though low phosphate is not pathognomic as hypophosphataemia is uncommon in hospitalised patient population
- Symptoms occur because changes in serum electrolytes affect the cell membrane potential impairing the function in nerve, cardiac and skeletal cells
- Spectrum of symptoms ranges from simple nausea, vomiting, and lethargy to respiratory insufficiency, cardiac failure, hypotension, arrythmias, delirium, coma and death
- Prevention is the key to successful management: early identification of high risk individuals, monitoring during refeeding, and an appropriate feeding regimen
- Principles of management are to correct biochemical abnormalities and fluid imbalances returning levels to normal where possible

TUTORIAL 4

NEURO DEVELOPMENTAL ASSESSMENT

Overall objective

1. The student should be able to perform a neuro-developmental assessment on all children i.e hearing and vision, gross motor & fine motor, language & speech, performance assessment, personal & social assessments.
2. At the end of this examination student should be able to make an assessment about the aproximate age at which the child is functioning and if that is equal or less than the other children of the same age.

Ask the following questions to caregiver when doing neurodevelopmental assessment and proceed with the examination

- Is your child able to see?
- Is your child able to hear and communicate as other children of the same age?
- Is your child doing the same things as the other children of the same age?

PHYSICAL

Vision

- Optical blink
- Menace response
- Follow light , tracking
- Can count fingers

Hearing

- Accoustic blink
- Turn to sound
- Rinnie's & Webber's tests in children >4 yrs old

Gross motor

- Sitting and crawling
- Standing, and walking
- Running – up and down stairs, tendom walking (forward, backward), kicking a ball

Fine motor

- Palmer grasp, reaching for objects
- Pincer grip, handedness, scribbles, writing, geomatrical figures
- Feeding / dressing

INTELLECTUAL

Language

Expresive: being able to produce speech and communicate a message

Comprehension	1 yr, 1 word
Expression	2 yrs, 2 words sentence
Articulation	3 yrs, 3 words sentence

Receptive: being able to follow a series of commands (listening and understanding what is communicated) i.e. responds to name like give me a toy, give me a brush

Speech: Babbles, coos, do distraction test, check if say words, phrases or sentences and ask for name, age, color etc.

Performance assessment

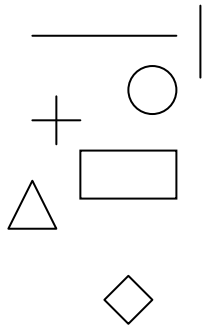
Basic concepts: objective performance 6-12 months

Take a cup and marble – retreive marble from under the cup

Motor constructive: bricks and blocks building >1 yr

Perceptual motor: geomatric designs > 2 yrs

- ± 2 yrs stright line
- ± 2.5 yrs – vertical line
- ± 3 yrs – round circle
- ± 4 yrs – cross
- ± 4 ½ yrs – rectangle
- ± 5 yrs - cross like multiplication
- ± 5 ½ yrs - triangle
- ± 6 yrs – diamond

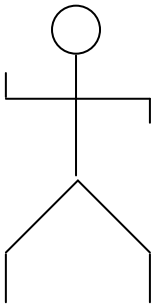


Draw a man test – good enough test

Total of five items or body parts where each scores 3/12. Add this total score to a basic age of 3yrs to obtain developmental age as follows:

- Head 3/12
- Neck 3/12
- Arms 3/12
- Spine 3/12
- Legs 3/12

Developmental age: 15/12 + 3 = 5/4 + 3 = 17/4 = 4 3/12



PERSONNEL / SOCIAL

- Face regard, responsive smile, response to images, stranger anxiety
- Feeding, drinking by holding a cup, exchanging
- Dressing up with help, alone
- Plays peak a boo
- Co-operative play - plays together
- Parallel play – plays alone – along side each other

NEURODEVELOPMENTAL MILESTONES ACCORDING TO THE AGE

AGE	VISION & HEARING	GROSS MOTOR CONGNITIVE	FINE MOTOR	SPEECH & LANGUAGE	PERSONAL / SOCIAL	WARNING SIGNS
New Born	Closes eyes to sudden bright light Still to sounds	Ventral suspension Head droops Hips flaxed Limbs hang Moro + Palmer grasp +	Fisted hands	Startles to sudden loud sound	Alternates between drowsiness and alert wakefulness	Increased or decreased tone Asymetry Sever head lag Poor suck
Screening 6 weeks	Follows Stares Red reflex Pupillary reactions Rattle or bell	Some head control Prone (pelvis up), Moro + Ventral suspension (head up, hips extension)	Keeps hands open 50% of the time	Startles - on history	Smiles Turn to face	Not fixing or following Asymetry Floppy Not smiling Poor sucking No response
3 months	Follows through 180 degree Turns head towards sounds	Pull to sit – no head lag Prone: rise on elbows Rolls over	Holds rattle Hands open Watches hands Pulls at clothes	Coos (sound like a pigeon) Chuckles Laugh quitely	Responds to bottle Excited when fed	
6 months		Prone (extends arms, lifts chest) Pull to sit (braces shoulders) Sits with support Supine (plays with feet)	Reaches Transfers Mouths	Initiates conversation	Responds to mirror Starts to hold bottle Shows likes and dislikes	
Screening at 9 months	No squint Normal eye movements Near vision Follows dropped toy	Rolls over Weight bears Sits without support Crawls Pulls to stand	Distraction Hearing test Holds cubes in both hands Immediately reaches out	Vocalises delibrately Bebbles Responds to name	Stranger anxiety Holds cup and bottle Plays: 'peek – a boo' with mother	Not sitting Hand preference Fisting Squint Primitive reflexes + No resonse to sound
10 months		Pulls to stand	Picks up	Waves	Plays peek-a-	

		Walks with assistance	small object b/w thumb and index finger	bye – bye Shakes head for no	boo with mother	
12 months		Bear walks Walks holding on	Pincer grip Releases object on request	Knows name Simple words: come, go	Finger feeds Arm into sleave	Unresponsive to sound Abnormal grasp
15 months		Walks alone	2 cubes tower	Jabbers	Holds & drinks from cup Attempts feeding with spoon Spills most	
Tests screening 18 months	Near vision Far vision	Pulls and carries toy Climbs into chair Walks well	3 cube tower Pincer grip Scribbles	3 or more words – excluding mama dada Obeys simple commonds	Indicates toilet needs – wet nappy	Failure to walk No pincer grip Inability to understand simple commonds No spontaneous vocalisation Mouthing Drooling
2 years	Near vision test	Runs Stairs up and down- 2 feet per step Kicks ball	6 cube tower Train with cubes Immitates vertical line Hand preference	Short phrases Uses pronouns	Clean and dry in day Spoon feeds - no spilling	Not understnding Tremor Incordination
3 years		Rides bicycle Stairs: Up 1 foot step Down 2 feet step	9 cubes tower Bridge Copies circle	Sentences Knows name and sex Talks incessantly	Toilet trained Dresses without supervision	Ataxia Using simple words only
4 years		Hops on preferred foot	Builds gate with cubes	Knows full name, age,	Eats with spoon and fork	Poor articulation

		Stairs: up and down 1 foot step	Copies cross	home address Matches colours	Washes and dries hands Make-believe play Dresses and undresses	
Screening 5 years	Near and far vision test Hearing test history	Walks easily along narrow line - heel to toe Hops on either foot Bounces ball	6-10 cube steps Copies square and triangle Draws a man with full features (6 parts)	Knows birthday Fluent speech	Uses knife and fork well competently Chooses own friends Dresses and undresses alone	Emotional immaturity
6 years		Walks backward on straight line (10 paces)	10 cube steps Copies diamond	Word definition Composition	Co-operative play	Clumsy Poor posture
7 years		Adds Substratcs 2 digit numbers Counts 2's, 100's	Draw a man Diamond	Defines words & nouns Similarities & differences	One special friend	Poor pencil grip
8 years		Counts 5's, 100's Simple multiplication	Cursive writing	Knows days of the week Talks sentences of 10 syllables	Dresses, undresses completely withot help	
9 years		Simple division 2 digit multiplication fraction	Cylinder	Understands obsurdity		
10 years		Division Runs downstairs	Writes three word sentences	Complex meaning Produces all speech sounds including 's', 'z' and 'ng'	Takes full responsibility for personal care	Speech sound difficulty

AN APPROACH TO A HANDICAP CHILD

Parents may visit with one or more of the following complaints:

- The child is delayed in sitting or walking i.e. delay in motor development
- The child is slow in all aspects of development (motor and social)
- The child is slow to talk (language delay)
- The child repeatedly falls in the early grades at school
- The combination of all above – global developmental delay

Past history

Take full past history.
Look for incidents that may have caused neurological damage.
Note the following in particular:

Maternal: Age of mother (Down syndrome in older women)
Illness of mother during pregnancy – eclampsia, rubella, drugs, alcohol

Delivery: Abnormal means of delivery – forceps, vacuum etc.
Condition of the baby at birth, was there a prolonged period of cyanosis, or did he/she take long time to cry and breath normally.

Prematurity: The degree can be assessed by the duration of stay in the hospital after birth

Postnatal: Jaundice/kernicterus - any history of exchange transfusion
Fits in newborn period have a close association with later handicap

Past illnesses:

- Ask for history of severe diarrhoea & dehydration. This may have caused a cerebral cortical vein thrombosis or electrolyte abnormalities more specifically hypo or hypernatraemia.
- Has there been a serious illness needing hospital admission such as pneumonia (hypoxic episode) or meningitis either bacterial or TB meningitis?
- Has there been a head injury?

Family history:
Is there a history of developmental delays or hearing loss or other handicapping disorder?
If so, there may be a genetic basis

Developmental history:
Most parents do not remember minor milestones such as rolling over or holding head clear of the bed. It is best to enquire about the major milestones such as smiling, sitting unaided, standing, walking alone and saying single intelligible words. Remember there is never a fixed normal age for reaching a particular milestone. There is always range of normality.

Upper limits of normality – if abnormal refer for assessment

- Not smiling by 8 weeks
- Not sitting unaided by 9 months
- Not standing unsupported by 12 months
- Not walking alone by 18 months
- Only single word with meaning at 36 months

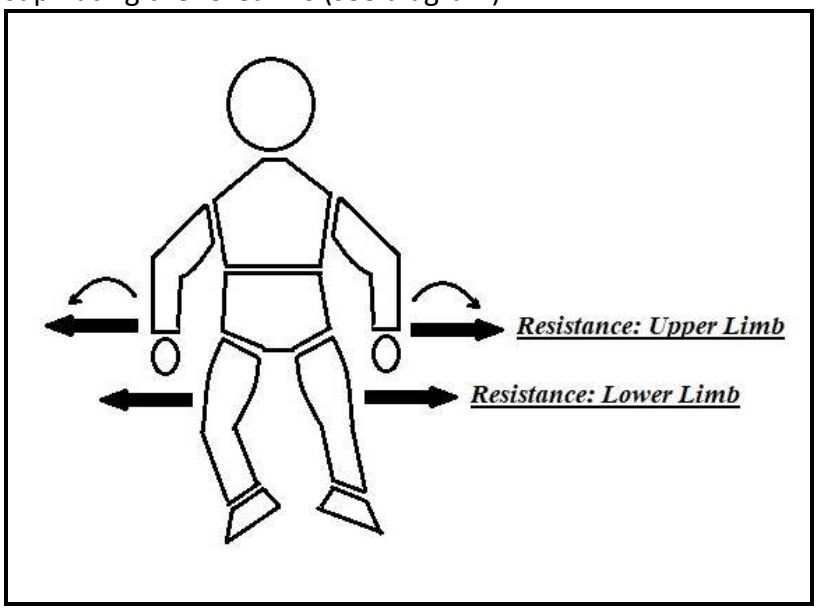
Delay in sitting or walking: the main causes of motor delay are:

1. **Severe illness:** a child recovering from severe acute malnutrition or measles may be static in his motor progress.
2. **Mental retardation:** these children are usually delayed in all aspects of development
3. **Cerebral palsy:** this is a group of motor disorders caused by an insult to the brain at or near the time of birth. Three important types are:
    a. Spastic (increased tone)
    b. Athetoid (abnormal movements)
    c. Hypotonic (decreased tone)
4. **Rare neuromuscular disorders:** cause severe hypotonia like spinal muscular atrophy

EXAMINATION OF MOTOR SYSTEM

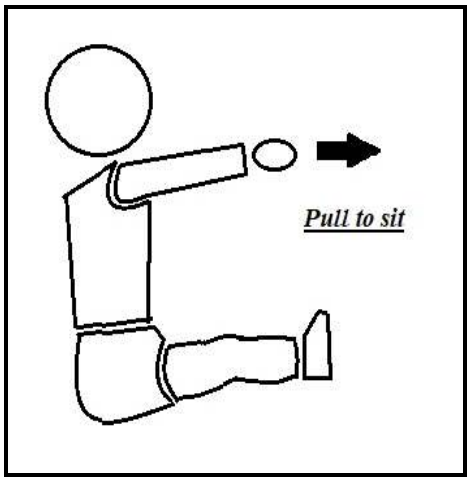
1. When the child is lying on his/her back

a. Observe the child's position
    - In spastic cerebral palsy (CP) the limbs are usually extended
    - In hypotonic states the limbs lie flat on the bed
b. Perform passive abduction with knees flexed and straight to test tone
    In spastic CP there is usually increased tone especially on abducting the knees and supinating the forearms (see diagram)



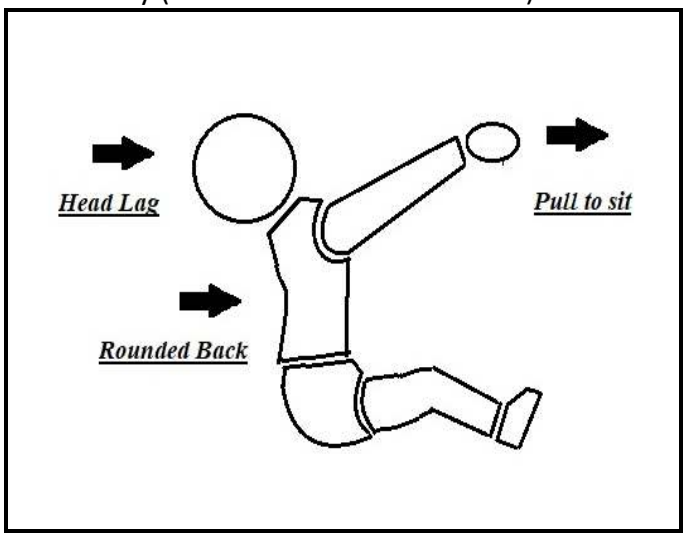
In hypotonic states tone is reduced and the limbs are floppy

2. Pull-to-sit:

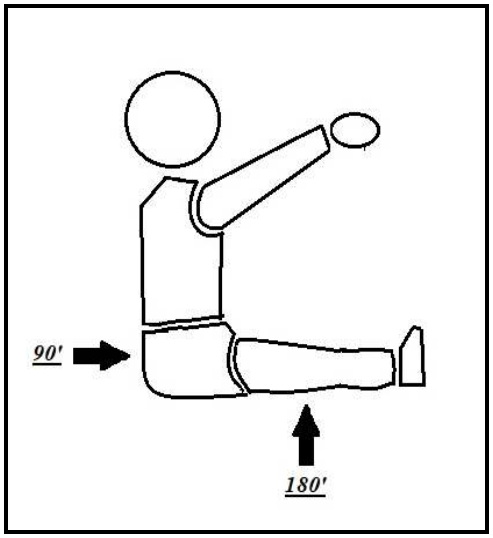


Start with the child lying on his back.
Take his hands and gradually pull up to a sitting position.
Observe (diagram)

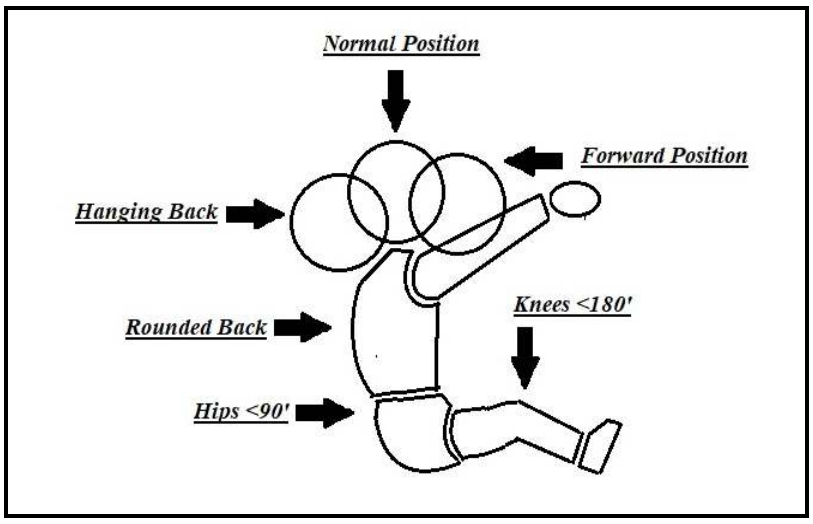
a. **Head:** does it fall back or stay in line with the baby or even come forward ahead of the baby (as is normal after 6 months).



b. **The curve of the back:** is it very rounded like a newborn baby or straight (which is normal after 6 months of age)
In both spastic and hypotonic cerebral palsy, the back is abnormally rounded



c. **Position of the hips and knees:** the normal position after 6 months of age is as seen in the Diagram



In spastic CP the hips are at less than a right angle and the knees remain flexed. Head may be in normal position, forward or hanging back in the young infant

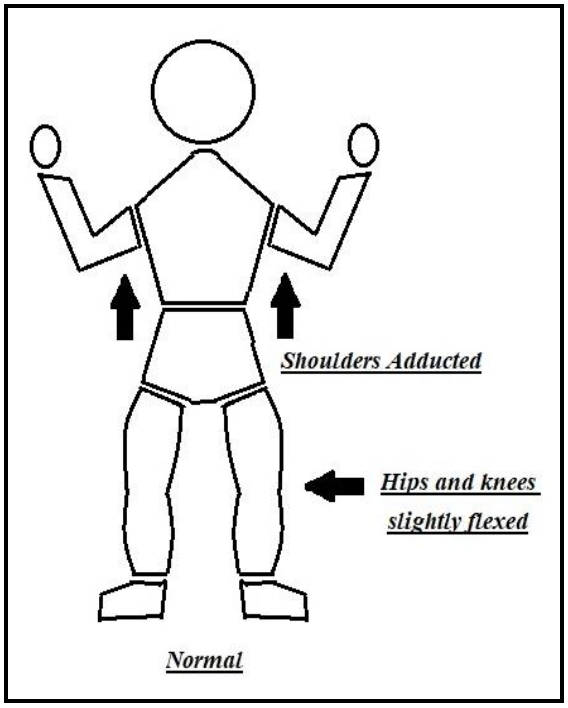
d. Can the child sit unaided? This is normal after 6-9 months of age.

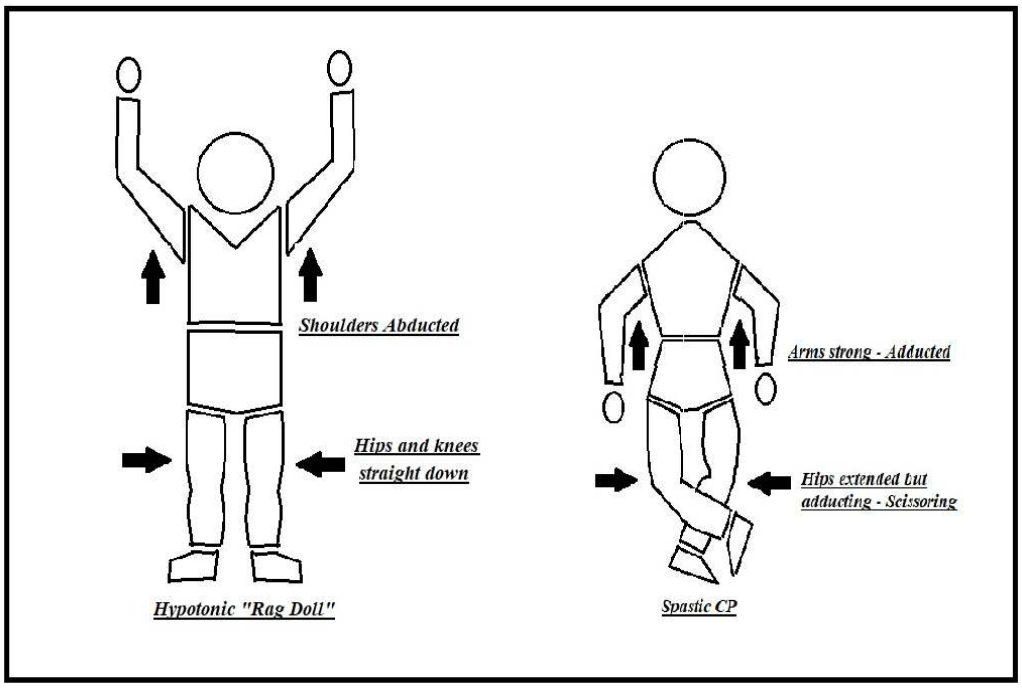
3. Suspension

Hold the child under the armpits and lift him clear of the floor or couch.

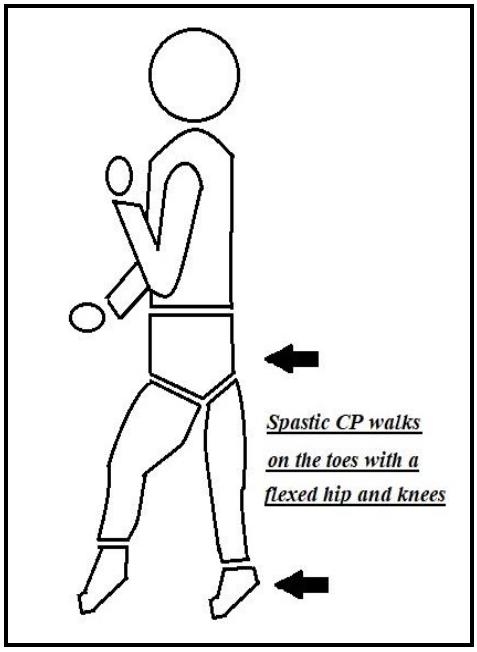
Observe:

a) Does he slip through your hands like a rag doll? This is typical of hypotonic states.
b) The position of the legs: in spastic CP the legs and feet extend abnormally and tend to cross over each other. This is known as scissoring (see diagram).





4. ***Walking:*** if the child can walk get him to walk up and down your consulting room or the corridor and observe:
   a. Movements of arms: do they swing freely as is normal or does one arm not moving. In hemiplegia one arm may be held at the side or comes up and is held in the position without swinging
   b. The legs: in spastic CP the walk is on the toes with a flexed hip and knee (see diagram). Take very small steps and legs are internally rotated.



Management of children with motor disorders

- If your assessment suggests motor disorder, the child should be referred to specialist centre for diagnosis and treatment purpose.
- In the case of CP, it is vital to begin expert physiotherapy as soon as possible.
- Intensive therapy can make a great difference to the child's final degree of handicap.
- The therapist usually sees the mother and child weekly for a prolonged period.
- The mother must be taught the techniques and exercises so that therapy can continue at home.
- In many remote areas it is quite impossible for the pair to come regularly. In these cases, it is useful to admit the child together with mother to hospital for a week or two several times a year.
- During these times the mother is taught the correct positioning and exercises for the child which she then carries out when they return home.
- The therapist can also supply them with simple aids such as positioning wedges and chairs made out of cardboard or other disposable material.
- Children who are unable to walk eventually become too heavy for the mother to carry and need a wheel chair.
- The collapsible very light 'Buggy-Major' or similar model is particularly suitable as it can be folded up for easy storage, fits in the boot of a car or can be taken on a bus.

AN APPROACH TO A CHILD WITH SPEECH DELAY

Take a full history as above and try to establish if there is really a significant speech delay;

Its normal development if the child is:

- 10 months: uses one word with meaning
- 1 year: uses 2-3 words with meaning
- 21 months: joins 2 words, repeat things said, ask for food, drinks
- 2 years: uses I, me, you and talks incessantly
- 3 years: knows name and sex, uses 3 words sentences

There is quite a wide range around these normals but it is always abnormal to be using only single words at 3 years. Always ask for a speech development of siblings and parents as benign delay in speech tends to run in families.

Carry out full physical examination. This may reveal an associated handicap such as CP. If delay is present, consider the possible common causes. Speech is learned by imitation of sounds the child hears. It is not part of normal natural development such as walking.

Deafness or hearing loss: It is essential to assess the hearing of any child with speech delay. Details of simple screening tests are given below. If your screening test suggests hearing loss, the child must be referred for formal audiometry at the ENT department of a hospital with the necessary staff and equipment.

Mental retardation: in this case there will be evidence of major delay in most other aspects of development such as motor and social.

Environmental deprivation: a child who is not frequently exposed to the speech of adults and children will be delayed in speech development.

Isolated delay in speech development: in this disorder other aspects of child's development including hearing are normal. It is an isolated disability, akin to dyslexia.

MANAGEMENT

This depends upon the cause. With deafness or hearing loss, it is important to place the child in therapy as soon as the deafness has been detected. If this is delayed the child may subsequently have greater difficulty in learning to speak.

Babies attend group parent sessions with the speech therapists who teach how the baby can be stimulated. From 3 years the child may attend a special nursery school and later a special school for the deaf. These schools are equipped with special apparatus and the staffs are specially trained in teaching the deaf. The child with isolated speech delay needs intensive speech therapy. The mentally retarded deaf child is largely managed as discussed in the section of mental retardation.

Screening tests for hearing loss: (see in ENT examination section)

- 0-6 months: Observe if the young infant quietens to the sound of his mother's voice or if he turns his eyes towards a sound.
- 6-12 months

The rattle test: it is essential to use the proper acoustic rattle which is obtainable from the ENT department, Tygerberg Hospital, Parow, Cape Town.

Method: the child sits on the mother's lap facing forward. The room must be quiet. The examiner stands behind the mother out of sight of the child. The rattle is held about 5 cm from the ear. Then turn the rattle through 90 degrees. This will produce a soft high frequency sound. If the child can hear it, he will usually turn his head to look at the source of the sound. The other ear is then tested. If the result is doubtful refer the child for normal audiometry at the ENT Department of a suitably equipped hospital.

Older children: the easiest screening method is to get the child to repeat the words that have been spoken (not whispered) into the ear at close range (10 cm). The STYCAR (Screening Test for Young Children and Retardates) screening tests are very useful when screening is done on a large scale. These require some simple special equipment. Further advice and help about deaf or hard to hearing children can be obtained from the local branch of The National Council for the Deaf.

The child who is slow in all aspects of Development (mental retardation)

This situation usually involves mental retardation either alone or in combination with other forms of handicap. Causes may include Down's syndrome, perinatal hypoxia, hypoglycaemia or kernicterus

CAUSES OF SPEECH DELAY

1. Bilingualism
2. Hearing loss
3. Mental retardation
4. Expressive language disorder
5. Psychosocial deprivation
6. Autism or elective autism
7. Receptive aphasia
8. Cerebral palsy

CAUSES OF DEAFNESS

Sensorineural hearing loss

1. Waardenburg syndrome
2. Klipple Feil syndrome
3. Alport's syndrome

Conductive hearing loss

1. Achondroplasia
2. Treacher-Collin syndrome
3. Craniosynostosis

NB! Deafness has aslo been associated with severe hypothyroidism and diabetes mellitus

TUTORIAL 5

DYSMORPHOLOGY

Dysmorphology is the branch of clinical genetics in which clinicians and researchers study and attempt to interprete the patterns of human growth and structural defetcs.

OVERALL OBJECTIVES

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

- Recognise major malformations and describe dysmorphic features
- Interprete the patterns of human growth and structural defects
- To reach an acceptable diagnosis for chidren with dysmorphic features and describe this in keeping with a particular
    - Syndrome
    - Sequence
    - Association

In order to start examination of a child with dysmorphic features you need to have relevevant family history and ages of both parents.

Expose the child in steps as you go along the clinical examination and inspect:

CRANIOFACIAL

Head

- Hair: quality, texture, distribution
- Shape: symmetrical or asymmetrical
- OFC: normo, macro or micocephalic, brachycephalic
- Is there craniotabes? Painful- be gentle. Not of significance under 3 months

Craniotabes: It is thinning of skull outer table. When pressed firmly over occiput or parietal bones, it gives-way as if pressing on Ping-Pong ball. It can be a normal variant or present in newborns, resolves by 1 month of age or may persist normally at suture lines

Differential Diagnosis includes: Rickets, osteogenesis imperfecta, hydrocephalus.

Fontanelle

- **Anterior:** diamond shaped
- **Posterior:** triangular
- **Third fontanelle:** may only be barely perceptable widening of the sagital suture between anterior and posterior fontanelles. It may be a normal varient or clue to Down syndrome
- **Closed or open:** If open – is it normal, under tension or sunken
- Fontanelles usually close around 1 year of age. Delayed closure > 18 months may suggest:

    - Hydrocephalus
    - Down syndrome
    - Hypothyroidism
- Abnormally wide fontanelle at any age may suggest:
    - Rickets
    - Hypothyroidism
    - Cranial synostosis
    - Rare syndromes like:
        - Smith-Lemli-Opitz syndrome
        - Zelweger's syndrome
        - Rubinstein-Taybi syndrome

Sutures: palpate with the finbers and palm of your hand

- Coronal
- Sagital
- Lambdoid
- Metopic: in the midline of the forehead due to early synostosis between the frontal bones (cosmatic problem only)

FACIAL FEATURES

Divide face into 4 regions

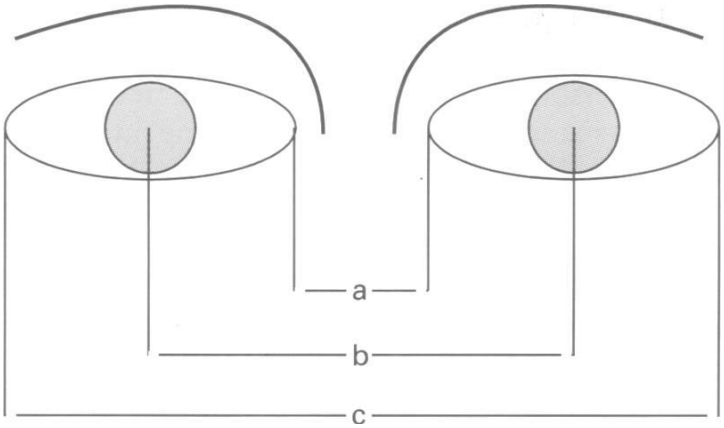
Forehead

- Overt prominance: achondoplasia
- Bitemporal narrowing: high forehead – occasional vertical ridges on forehead → Miller Dieker syndrome
- Sloping appearance - primary microcephaly

Mid face

From eye brows to the upper lip & from outer canthi to the commissures of mouth

- Look at inner canthal distance
- Measure interpupillary distance if suspecting abnormal – ask for charts to plot



- **Check for Hypotelorism**: may be associated with defect in midline brain formation

Hypotelorism is narrowness of the intraorbital distance which may occur as a morphogenetic varient or in association with other anomalies like epicanthic folds (vertical folds of skin on either side of the nose), or secondary to cranial dystrophy.

- **Check for Hypertelorism** (roughly check if 3rd eye can fit in)

Hypertelorism is wide separation of eyes or increased intraorbital distance (> +2 SD). It may be morphogenetic varient, a primary deformity or secondary phenomenon in association with developmental abnormalities like: frontal meningocele, encephalocele and persistance of facial cleft. It may be associated with strabismus, extropia or optic atrophy.

- **Exophthalmos**
  - It is protrusion of eyes commonly due to orbital disease
  - Shallowness of orbits due to craniofacial malformations
  - Increased tissue mass in orbit like neoplasia, vascular, inflammatory disorders

  Occular complications due to exophthalmos include:
    - Exposure keratopathy
    - Occular motor disturbances
    - Optic atrophy – loss of vision
- **Enophthalmos**
  - It is shrinking of the eye back into the orbit that may be due to
    - Orbital fracture
    - Atrophy of orbital tissue
- **Thyroid related ophthalmopathy**
  - It is believed to be secondary to immune mechanisms leading to inflammations and deposition of mucopolysaccharoids and collegen in extraoccular muscles and orbital fat.
  - Involvement of extra occular muscles may lead to restrictive strabismus
  - Complications include corneal exposure leading to inflammation and perforation and compression of optic nerve.
- **If Protuberant upper lip** with thin vermian border, consider Miller Dieker syndrome
- **If short length of pelpebral fissure consider** foetal alcohol syndrome
- If excesive length of pelpebral fissure consider Kabuki make up syndrome (with short stature and mental retardation)

EYES

A direct ophthalmoscopic examination helps assess the status of optic discs and macula. Complete examination of retina requires dilating the pupil and using an indirect ophthalmoscope.

Abnormalities noted

- Coloboma
- Conjunctival telangiectasia
- Upward stant: consider Downs syndrome
- Downwards slant: consider Treacher Collins syndrome

- Prominent epicanthic folds: consider Downs or Foetal alcohol syndrome
- Squint: consider Fanconi's syndrome or 6th nerve palsy with intracranial bleed
- Proptosis: consider Fanconi's syndrome or 3rd nerve palsy or myasthenia gravis
- Synophrys: eyebrows that meet in the midline
- Cataract: consider diabetes mellitus, congenital rubella, galactosaemia

NOSE

- Nasal bridge
  - Flattened: Down or Foetal alcohol syndrome
  - Prominent: Velocardiofacial syndrome

MALAR REGION

It extends from ears to the midline

Note:

- Size and shape
- Low set ears
- Posterior rotation
- Pre-auricular skin tag
- Ear lobe creases (Beckwith-Wiedemann syndrome)
- Note if the child has a hearing aid. Deafness can occure in Treacher Collin, CHARGE, foetal rubella and Waardenburg syndrome

MANDIBULAR REGION

It extends from lower portion of ears bounded out to the chin by the mandible

- Small chin: consider Pierre Robin syndrome

NECK

- Webbing of neck: consider Turner or Noonan syndrome
- Short neck: consder Klippel-Feil syndrome
- Position of posterior hair line is low: consider Klipple-Feil or Noonan syndrome

TRUNK

- Sheild like chest: consdier Turner or Noonan syndrome
- Pectus deformity: consider Marfan syndrome
- Scoliosis: consider Marfan syndrome

EXTREMITIES

- Check knee joints: look for patella (missing patella in "Nail Patella Syndrome")
- Check range of movements of all joints and screen for carrying angle
  - Increased (cubitus valgus)
    - Turner syndrome

        - Noonan syndrome
- Check if joint laxity is present:
    - Hypermobility
        - Osteogenesis imperfacta
        - Marfan syndrome (wrist sign, thumb sign)
        - Ehler-Danlos syndrome
    - Multiple contractures
        - Arthrogryposis multiplex congenita
    - An inability to pronate and supinate elbow
        - radioulner synostosis
            - X chromosome aneuploidy
            - Foetal alcohol syndrome

HANDS

- **Syndactyly:** two or more digits at least partially or completely fused: consider Apert syndrome, Smith-Lemli-Opitz syndrome
- **Polydactyly** (extra digit): consider Ellis-van Creveld syndrome
- **Brachydactyly** (short digits): consider Aarskog syndrome, Downs syndrome
- **Camptodactyly** (digit is bent or fixed in direction of flexion): can be associated with other skeletal anomalies and number of congenital syndromes like Jacobson syndrome and Beals syndrome.
- **Clinodactyly** (digit is crooked, curves towards or away from the other digits): consider Russell-Silver syndrome

DERMATOGLYPHICS

- Single transverse palmer crease and hypotonia during early foetal life: consider Downs syndrome in 50% cases but can be normal in 10% cases.
- Characteristic palmer pattern is seen in Foetal alcohol syndrome

GENITALIA

- Disorder of sexual differentiation may indicate congenital adrenal hyperplasia
- Hernia; especially in neonate and hypospadias; if associated with cryptorchidism, there is strong possibility of a syndrome

MAKE AN ASSESSMENT

Try to recognize if it is Syndrome, Sequence or Association, otherwise say you don't recognize a particular syndrome but would either refer or try to look it up. Chromosome studies are indicated when 3 or more than 3 dismorphic features are present.

Common Syndromes

Down syndrome

- Trisomy 21: non-dysjunction (95%), translocation (4%), mosaicism (1%)
- Flat facial profile, Up slanting eyes, Epicanthic folds, Flat nasal bridge, Small and low set eras, Protruding tongue
- Short stature, Brachydactyly, 5th finger clinodactyly, Single palmer crease, Sandal-gap between 1st and 2nd toes
- Congenital heart defect (40%), Duodenal atresia, Hypotonia, Brachycephaly
- Complications: Hypothyroidism, Acute leukaemia, Alzheimer's disease, Mental retardation
- Diagnosis: QF-PCR aneuploidy test, Karyotyping
- Treatment: growth and neurodevelopment monitoring**.** Medical treatment of cardiac failure and hypothyroidism, neurodevelopmental interventions
- Prevention: Female education, Family planning, Pregnancy screening for advanced maternal age (AMA), Sonar and maternal screening, Screening for hypothyroidism

Turner syndrome

- Appearance: short neck with a webbed appearance
- Low hairline at the back of the neck
- Low-set ears
- Hands and feet that is swollen or puffy at birth
- Soft nails that turns upward.
- Broad chest with widely spaced nipples
- High, narrow roof of the mouth (palate)
- Arms that turn outward at the elbows
- Fingernails and toenails that are narrow and turned upward
- Swelling of the hands and feet, especially at birth
- Slightly smaller than average height at birth
- Slowed growth
- Cardiac defects
- Receding or small lower jaw
- Short fingers and toes

CATCH 22

- **C**ardiac defects – Interrupted aortic arch, truncus arteriosus, VSD, PDA, TOF
- **A**bnormal facial features – wide set eyes, narrow small groove in upper lip, small jaw
- **T**hymus underdevelopment
- **C**left palate
- **H**ypercalcaemia

CHARGE association

- **C**oloboma and cranial nerve abnormalities – defects of the eye ball
- **H**eart anomalies – TOF
- **A**tresia of choanae – bony and or membranous
- **R**etardation of growth – both cognitive and somatic growth
- **G**enital anomalies and or hypoplasia in males

- **E**ar anomalies and or deafness

CHILD syndrome

- **C**ongenital **H**emidysplasia
- **I**chthyosiform erythroderma
- **L**imb **D**efects

LEOPARD syndrome

- **L**entigines (brownish pigmented spots on skin due to increased deposition of melanin & increased number of melanocytes)
- **E**CG abnormalities: axis deviation, unilateral or bilateral ventricular hypertrophy, conduction abnormalities
- **O**cular hypertelorism
- **P**ulmonary stenosis
- **A**bnormal genitalia
- **R**etardation of growth, deafness

PHASE Syndrome *(Frieden et al 1996 – Neuron 2011)*

- **P**osterior fossa anomalies
- **H**aemangioma
- **A**rterial lesions
- **C**ardiac abnormalities – aortic co-arctation
- **E**ye abnormalities

VACTERL / VATER association

- **V**ertebral anomalies
- **A**nal atresia with or without fistula
- **C**ardiac anomalies – VSD
- **T**racheo-**E**sophageal fistula with oesophageal atresia
- **R**enal anomalies – urethral atresia, with hydronephrosis
- **L**imb anomalies – polydactyly, humeral hypoplasia, radial aplasia and proximally placed thumb

CRANIOSYNOSTOSIS *(Nelson p2456)*

- Premature closure of cranial sutures
- **Primary** – closure of one or more sutures due to abnormality of skull development
- **Secondary** – due to failure of brain growth

Crouzen Syndrome (Ziai page 21)

- Craniofacial dysostosis, Shape of head depends upon timing, often is compressed back to front diameter, Orbits are underdeveloped & Ocular proptosis is prominent
- Hypoplasia of maxilla & orbital hypertelorism are typical facial features
- Beak type nose, Exophthalmos, Strabismus, Short upper and protruding lower lip

Apert syndrome

- Syndactyly of 2^{nd}, 3^{rd} & 4^{th} fingers which may be joined to thumb & the 5^{th} finger
- Similar abnormalities often occur in feet
- Progressive calcification & fusion of bones of hands, feet and cervical spine
- Mental retardation, Asymmetric facies – eyes are less proptotic than Crouzen

Carpenter Syndrome

- Many fusions of suture, tend to produce the Kleeblattschadel skull deformity
- Soft tissue syndactyly of hands & feet is always present

- Mental retardation, Obesity, Hypogonadism (Ziai page 22)
- Heart disease, Corneal opacities, Coax valga and genu varum

Pfeiffer syndrome

- Commonly associated with turricephaly, Eyes are prominent (widely spaced)
- Thumbs and great toes are short and broad, Partial soft tissue syndactyly

Each of the above genetic syndromes poses additional anomalies including

- Hydrocephalus and increased intracranial pressure
- Optic atrophy – due to abnormalities of optic foramina
- Respiratory problems secondary to a deviated nasal septum or choanal atresia
- Disorders of speech and deafness
- Craniotomy is mandatory for management of increased intracranial pressure

Hunter's Syndrome *(Forfar p983)*

- Usually between 2-5 years but milder types
- Slowly deteriorating MR, Stunted growth – claw hands
- Hepatosplenomegaly & Cardiac defects due to infiltration into valves & arterial walls
- Papilledema (may lead to blindness)
- Hernias, Progressive deafness, Carpel Tunnel Syndrome
- Differs from Hurler's – no gibbous formation

Kabuki Makeup Syndrome

- Mental retardation, Short stature
- Unusual skin ridges patterns on the fingers, toes, palms of hands & soles of feet

Klipple-Feil Syndrome

- Short neck, low hair line, Sensorineural hearing loss
- Paralysis of external rectus muscles – one or both eyes squint

Miller Dieker Syndrome

- Lissencephaly, Sever hypotonia at birth
- Seizures with corticospinal tract signs

Pierre Robin Syndrome *(Nelson p1534)*

- Triad of micrognathia + glossoptosis (results in) → airway obstruction
- Micrognathia is usually associated with high arched palate & cleft palate. Tongue usually of normal size but floor of the mouth foreshortened. Obstruction of air passage may occur, on inspiration – usually requiring treatment to prevent suffocation
- Tracheostomy or endotracheal intubation may be required
- Mandibular distraction procedures in neonates can improve mandibular size – enhance respiration – facilitate oral feeding
- Sufficient mandibular growth may take place to relieve obstruction – growth often achieves normal profile in 4-6 yrs
- Dental anomalies require individual treatment
- 50% of children with PRS have Stickler Syndrome (AD) that includes other findings – early arthritis and ocular problems
- DD: Treacher Collin (mandibulo-facial dysostosis), Hemifacial microsomia, Goldenhar Syndrome
- Treatment: Address airway, Prone position, do U/E (look for Bicarb retention)

Pradder Willi Syndrome

- Major criteria: Neonatal hypotonia, Excessive weight gain, Feeding problems, Facial features, Hypogonadism, Developmental delay, Hyperphagia
- Minor criteria: Decreased foetal activity, Behaviour problems, Sleep disturbances / sleep apnoea, Short stature, Hypopigmentation, Small hands & feet, Narrow hands & straight ulnar border, Eye abnormalities, Thick viscous saliva, Articular defects and Skin picking
- Diagnosis: Chromosomal analysis for methylation patterns in PWS, PCR or Southern Blot hybridization and FISH analysis – parental

Rubinstein-Taybi Syndrome *(Forfar)*

- Broad thumbs, great toes
- Severe mental retardation
- Characteristic facies: Antimongoloid slants, Mid face Hypoplasia, High arched palate
- Narrow beaked nose

Russell Silver Syndrome

- Growth delays, increased risk of learning disabilities
- Before birth is IUGR and after birth will be growth deficiency
- Clinodactyly, Hemihypertrophy – unequal asymmetrical growth

Smith-Lemli-Opitz Syndrome *(Nelson p2401)*

- Prenatal and post-natal growth retardation
- Ptosis
- Anteverted nares
- Broad alveolar ridges
- Syndactyly of 2^{nd} and 3^{rd} toes
- Microcephaly
- Severe mental retardation
- Types: Classical **type 1** (associated with pyloric stenosis) & **type 2** (associated with Hirschsprung disease, Cleft palate, Skeletal abnormalities

Treacher Collin Syndrome

- Malformation of external ears
- Hearing loss
- Breathing difficulties
- Abnormal down ward slant of palpebral fissures
- Craniofacial abnormalities
- Hypoplasia of certain bones of head
- Zygomatic complex and the jaw & cheek bones

Zelweger Syndrome – Cerebro-hepato-renal syndrome *(B Nelson p 263)*

- High forehead, Flat orbital ridges, Widely opened fontanels
- Hepatomegaly, Hypotonia, FTT, Seizures & nystagmus

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

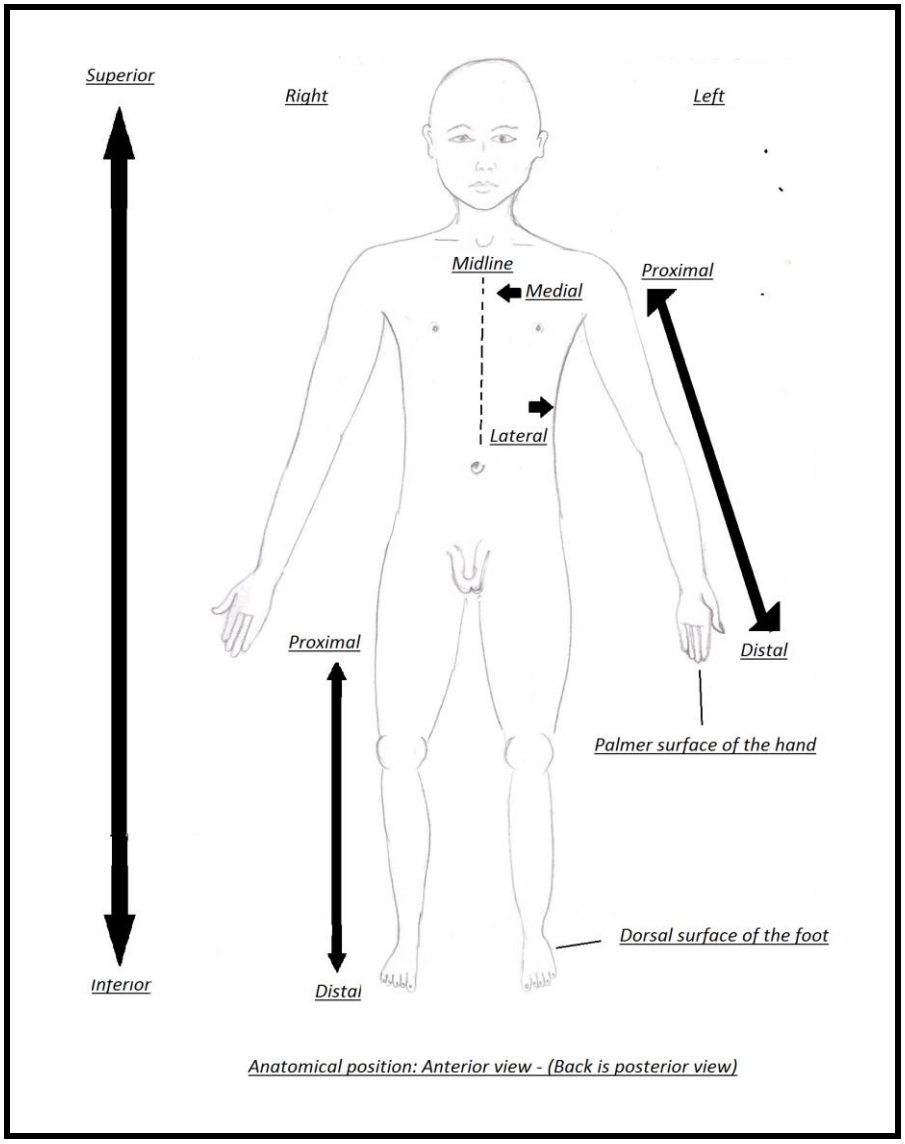
Ill looking, in severe respiratory distress or comfortable at room air

- 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



4. 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

b. 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

5. 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, and 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.

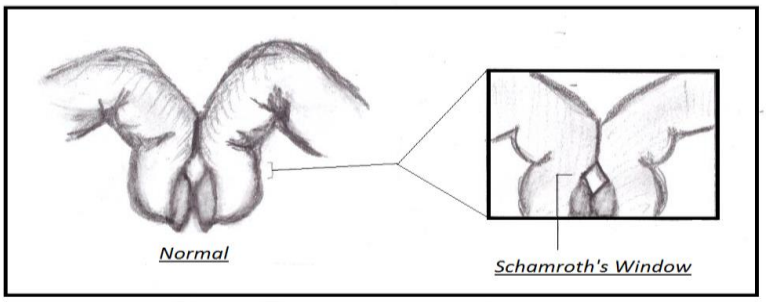
When the distal phalanges of corresponding fingers of opposite hands (preferably thumb nails) are directly apposed (placed against each other back to back), a small diamond shaped window is normally apparent between the nail beds. If this window is obliterated, the test is positive and clubbing is present.

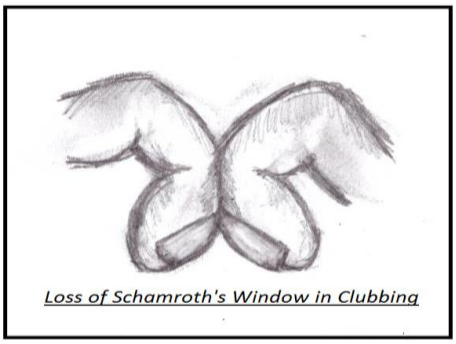
Long standing arterial desaturation (usually longer than 6 months in duration) even if too mild to be detected by an inexperienced person results in clubbing of finger nails and toes. It appears earliest and most noticeably in thumb.

Clubbing develops in 5 steps:

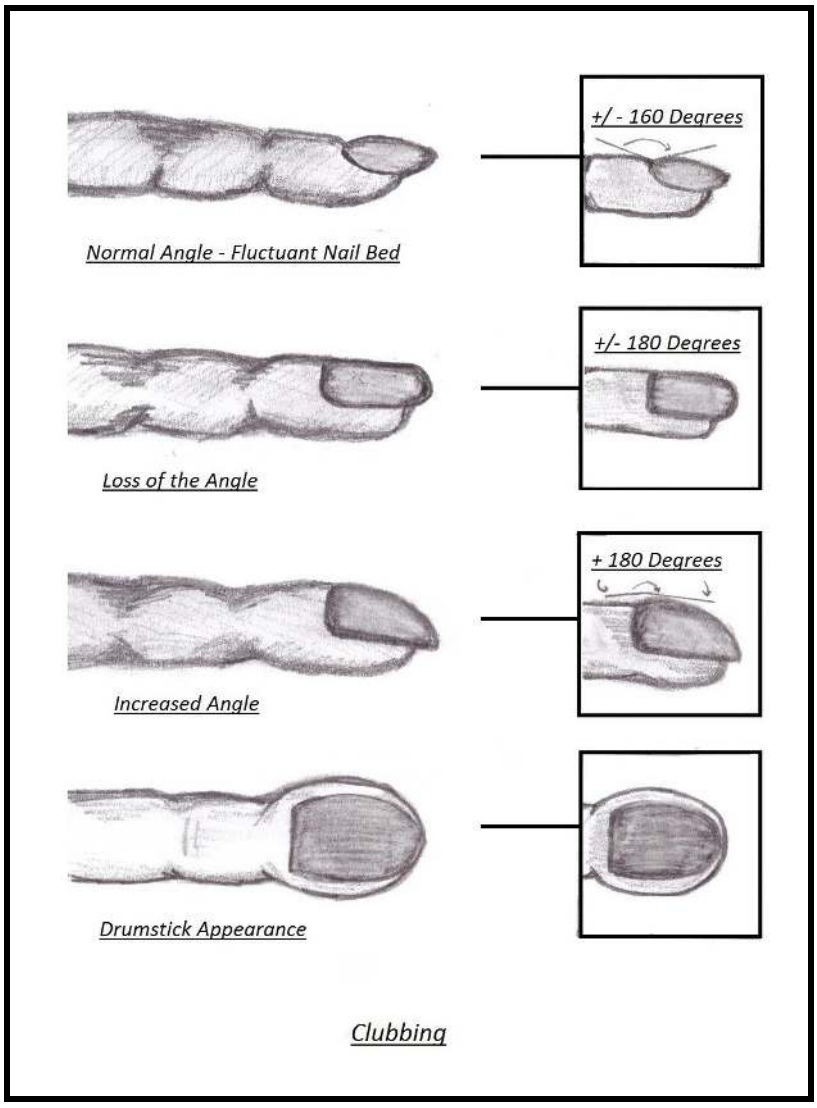
1. Fluctuation and softening of the nail bed
2. Loss of normal Lovibond angle (165^{o})
3. Increased convexity of the nail fold
4. Thickening of the whole distal finger (resembling drum stick)

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

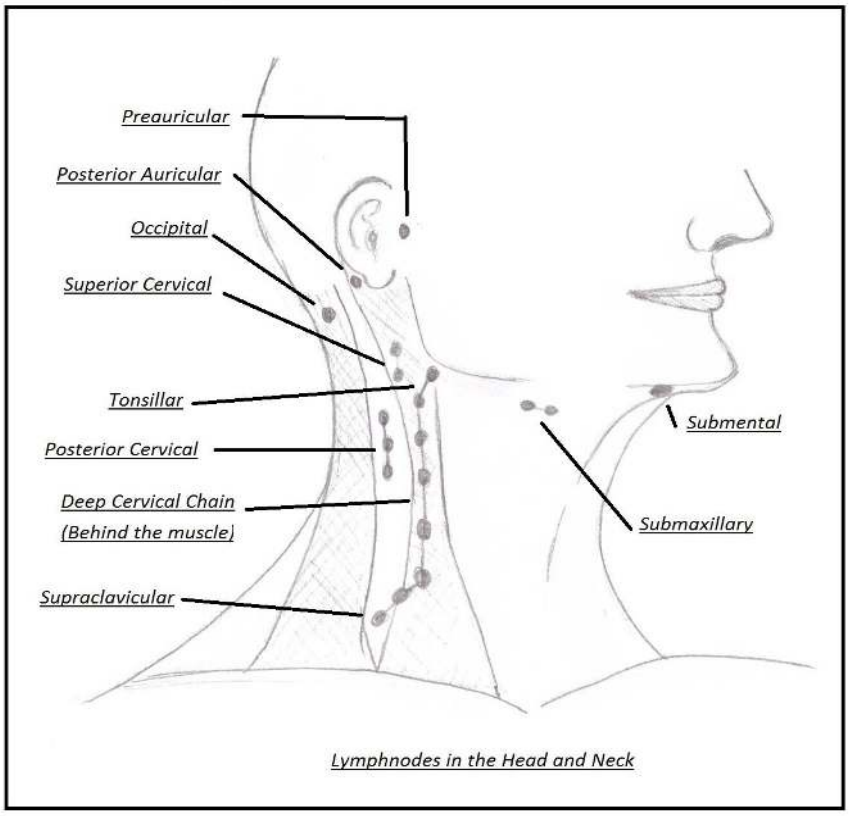
1. **Venous causes –** (pitting – unilateral lower limb oedema)
   - Post-surgical
   - Venous thrombosis
   - Pressure from neighbouring tumour or lymph node
   - Valvular incompetence (varices)
2. **Lymphatic causes – (nonpitting lower limb oedema) +**
   - Lymphoedema
   - Filariasis (lymphatic obstruction 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

11. LYMPH NODES

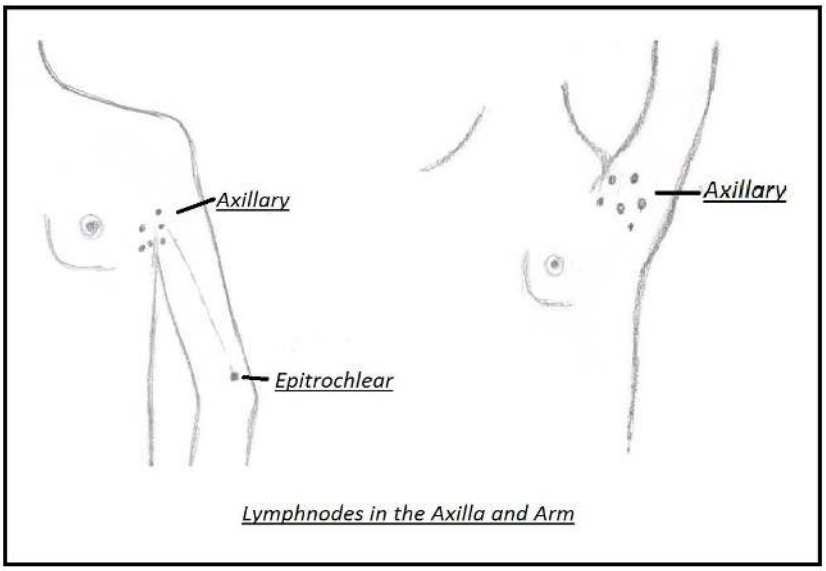
Palpate various groups of lymph nodes.

They are significantly enlarged if following groups are:

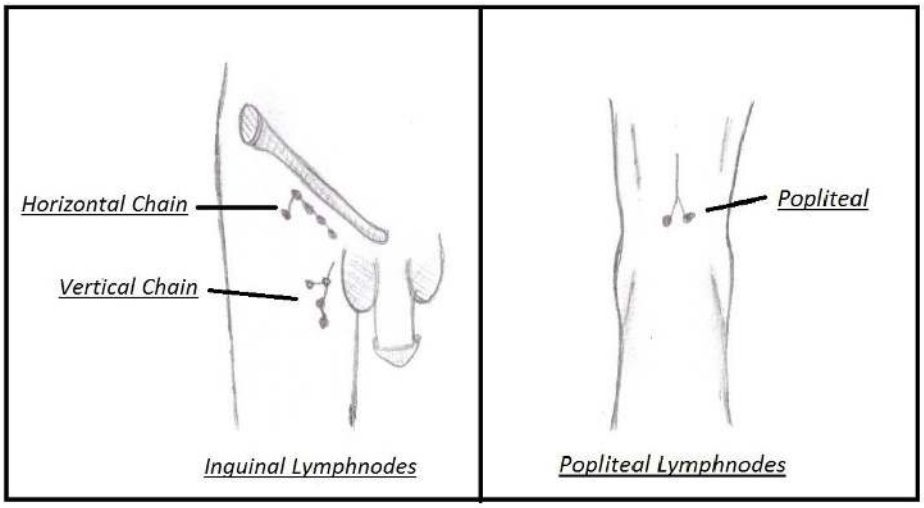
- More than 1 cm
  - postrior cervical
  - submandibular
  - axillary
  - femoral
- More than 1.5 cm
  - Inguinal
- Any size
  - Supraclavicular
  - Epitrochlear (mention if no peripheral inflammation)

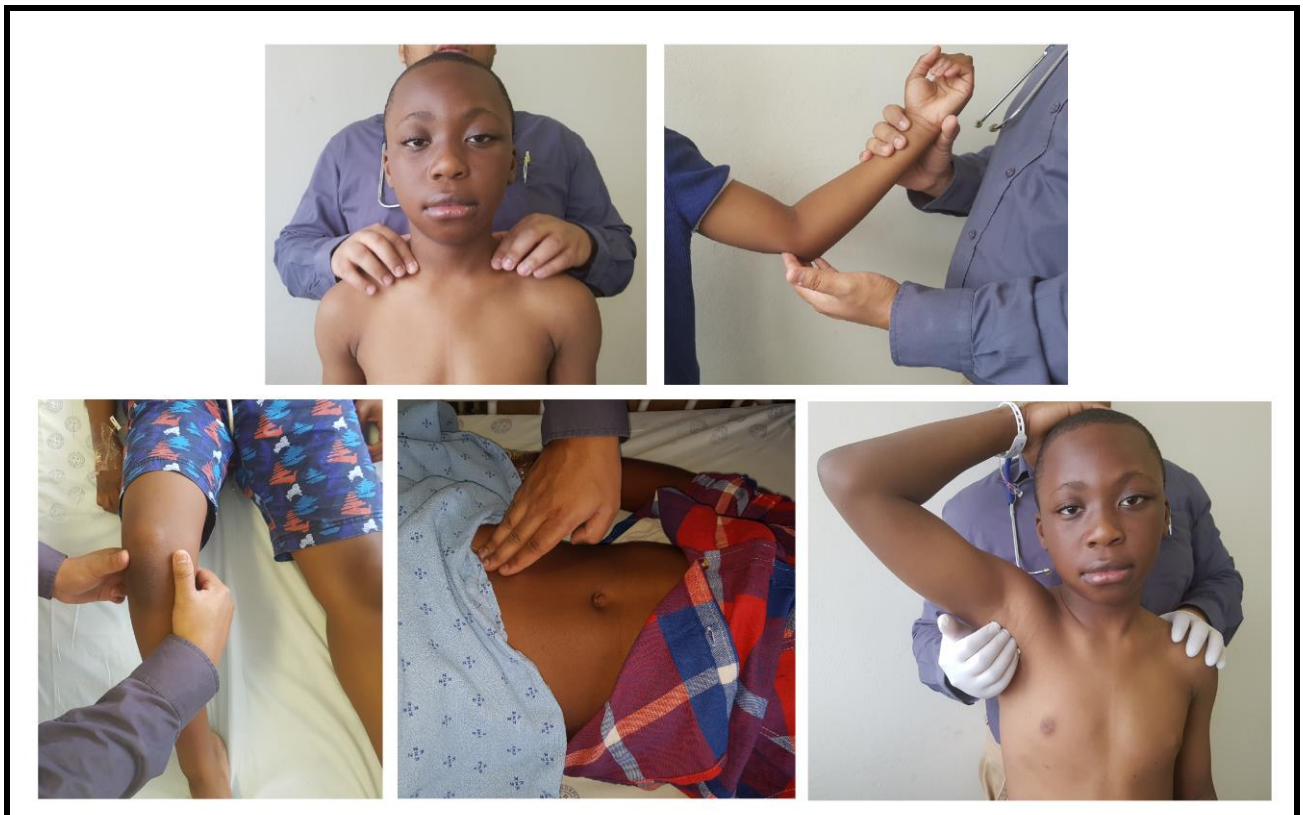


Lymphnodes in the Head and Neck



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

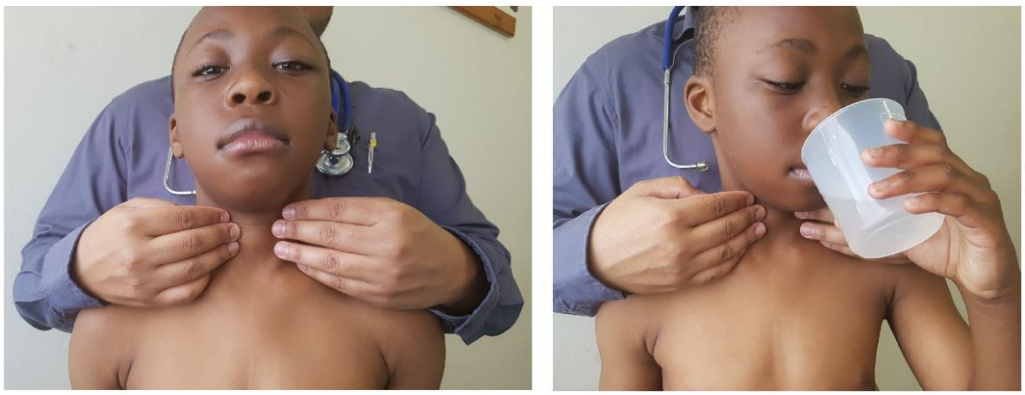
1. Local pyogenic infections like pharyngitis, dental abscess, otitis media
2. Viral infection: cat scratch fever, lyphogranuloma venereum
3. TB (lymph nodes matted and there may be sinus formation)
4. Non- Hodgkin's lymphoma
5. Secondary carcinoma – hard regular and fixed lymph nodes
6. Virchow's gland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

12.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.

13.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.

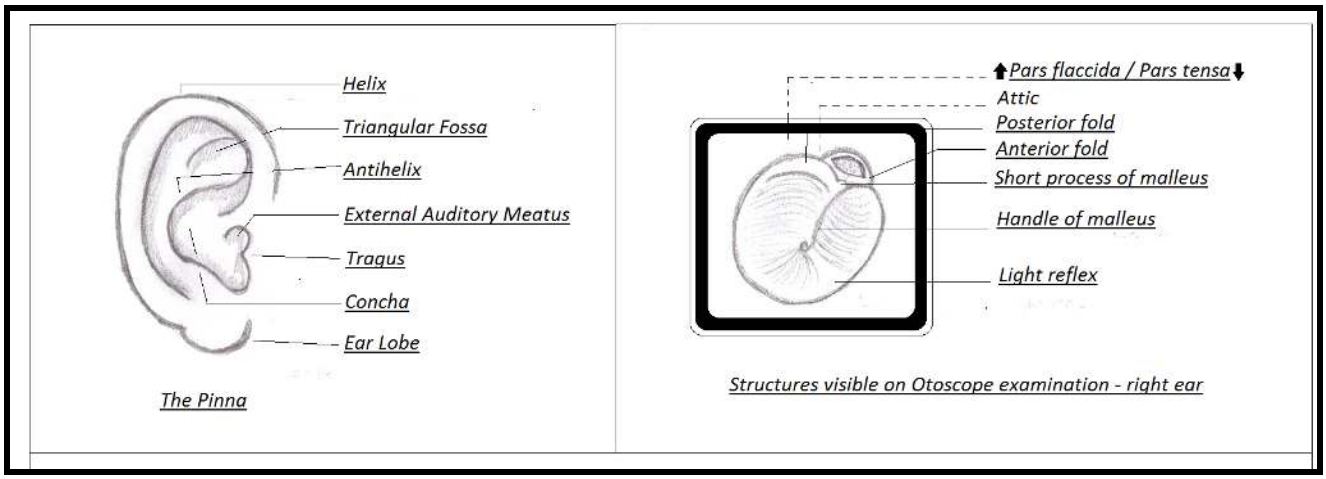
Changes that are associated but are not directly measured by Tanner Staging include bone growth and fusion, body composition and linear growth and haematocrit values. In males, the onset of puberty ranges from 9 to 14 years of age. In females, the normal onset of puberty ranges from 8 to 13 years old 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 <u>≤</u>3 ml
G2 Testes <u>≥</u> 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>

14. 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



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

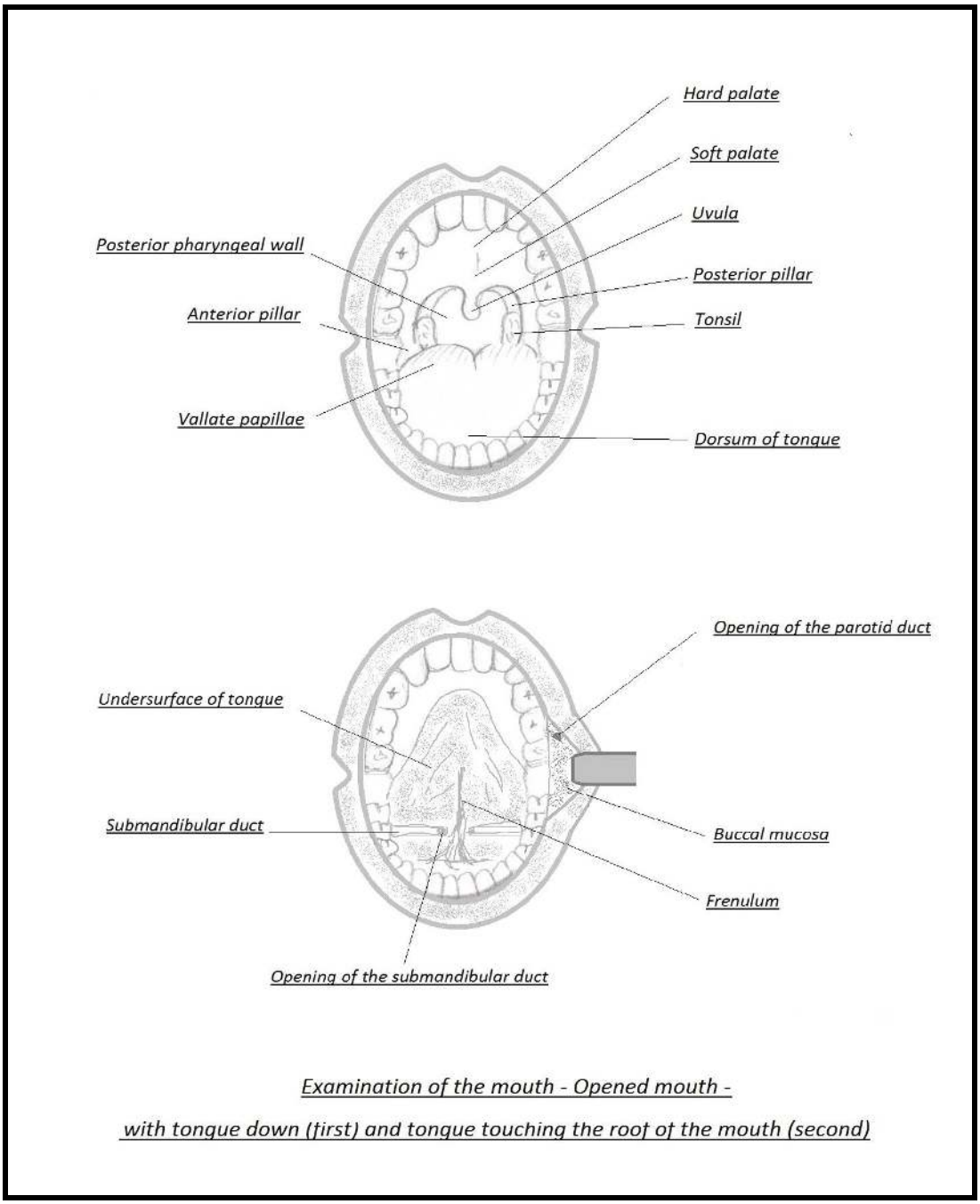
Inspect gums for

- 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

15. 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

TUTORIAL 7

THE EXAMINATION OF RESPIRATORY SYSTEM

OVERALL OBJECTIVES

At the end of this module the student should be able to differentiate, diagnose and manage common respiratory problems like:

1. **Upper airway obstruction** [laryngotracheobronchitis (LTB), foreign body (FB) in airway] and
2. **Lower airway obstruction** [pneumonia and its complications (pleural effusion, pneumothorax, cysts, collapse), chronic chest disease (pulmonary TB, bronchiectasis)]

To look for extra general features:

1. **Cough:**
   - Productive: results from an inflammatory or infective exudate on the bronchial mucosa: Cystic fibrosis, Pulmonary Tuberculosis (PTB)
   - Non-productive nocturnal: post nasal drip, asthma
   - On exercise: asthma
   - Dry intermittent or persistent: may imply irritation of the upper respiratory tract or of the bronchial wall by a foreign body, or an enlarged lymph node.
   - Productive more than 2 weeks: Consider PTB
   - Croupy: barking associated with strider and hoarseness – Croup, acute laryngotracheobronchitis (LTB)
   - Whooping: inspiratory gasp, prolonged distressing cough, ending in a whoop, may follow vomiting – pertussis
   - During or after feed: inhalation, aspiration
   - Bovine brassy: Tracheitis
   - Absent during sleep – psychogenic
2. Hallitosis and a productive cough – Chronic suppurative lung disease
3. Sputum:
   - Clear mucoid or tacky tenacious – indicative of asthma
   - Green yellow, grey (dirty) – infection most common is PTB
   - Haemoptysis: rare in children – may indicate cystic fibrosis or PTB
4. Parotid enlargement in lymphocytic interstitial pneumonia (LIP)
5. Delerium, pleural pain, rusty sputum and herpes labilais – suspect pneumococcal pneumonia
6. Look for plethora

1. INSPECTION

Comment on general inspection

To avoid anxiety in children, mother should hold the child on her lap during the initial examination. Inspection should be done first before doing anything else which may disturb the child. Clothing need to be removed and chest exposed as the examination proceeds.

Scene: look around if there is a specimen container and note its contents like sputum, blood, mucous or pus

Expose the chest and look for:

- Scars: post op, post chest drain insertation
- Pigemntation: Addisons disease
- Rashes: on the chest wall

Features of allergic facies

Note tired looking face, open mouth breathing, double folds of lower eye lids (Denne-Morgan lines), allergic shiners, allergic salute (nasal crease) and nasal discharge.

Shape of the chest

Normal chest is elliptical i.e. its anterioposterior diameter is lesser than its transverse diameter that is 3:5 respectively. Abnormal forms can be as follows:

1. **Asymmetry: Retraction** of chest wall on one side may be due to;
    - lung collapse, fibrosis, spinal deformity
2. **Asymmetry: Bulging** of chest wall on one side may be due to:
    - Pleural effusion, pneumothorax, massive cardiomegaly, kyphosis or intrathoracic tumour.
3. **Deformity:** Check for scoliosis or kyphosis and ask older children to touch their toes to determine whether the deformity is fixed or postural
4. **Barrel shaped chest:** Increased AP diameter and angle of Louis becomes more prominent: consider air trapping
5. **Harrison's sulcus:** This is transverse constriction which begins at the level of the xiphisternum, passes outward and slightly downwards. The sulcus is formed along the line of diaphragm (normal diaphragm pulls on soft ribs). Causes:
    - Chronic airway obstruction
    - Left to right cardiac shunt
    - Rickets
6. **Hoover sign**: it is isolated subcostal recession associated with hyperinflation of the lungs in a patient with peripheral airway disease. It causes diaphragm to flatten and to contract laterally against the lower chest wall resulting in indrawing below the rib cage.
7. **Pectus carinatum** (pigeon chest)
8. **Pectus excavatum** (funnel chest)
9. **Rachitic rosary:** Rickets
10. **Absent clavicles**: Cleido-cranial dysostosis
11. **Absent pectoralis:** Poland's syndrome

Movements of chest:

1. Stand at the end of the bed and compare two sides of the chest – pathology is usually on the side with lesser movement
2. Note the signs on one side of chest and compare with the other side of chest on the respective point
3. Count respiratory rate for a full minute – say if its normal or abnormal
4. Note nasal flaring, difficulty in breathing or shotness of breath
5. Note respiratory movements – normal or abnormal
6. State if patient is in respiratory distress. Ist it mild, moderate or severe
7. **Look for intercostal & subcostal recession.** It is also due to airway obstruction or decreased lung compliance and by using accessary muscles, especially sternocleidomastoids.

Common causes of fast breathing

1. Airway obstruction
2. Bronchopneumonia
3. Metabolic acidosis
4. Atelectasis
5. Pneumothorax
6. Fever and anxiety
7. Drugs like salicylates

8. **Note if tracheal tug is present:** Tracheal tug is downward pull of the trachea, manifested by a downward movement of the thyroid cartilage. In children it is due to lower airway obstruction probably due to lung compliance problems. This recession is due to greater negative intrathoracic pressure which draws in the soft tissue.
9. **Audible sounds:**
   - Stridor, stertor, snoring, hoarseness, wheeze
   - Grunting (breathing out against a closed glottis) increases pressure in the lungs and small airways to prevent collapse and improve oxygen transfer.

Acute laryngotracheobronchitis (stridor)

Common between 6 months to 2 years of age

Grading:

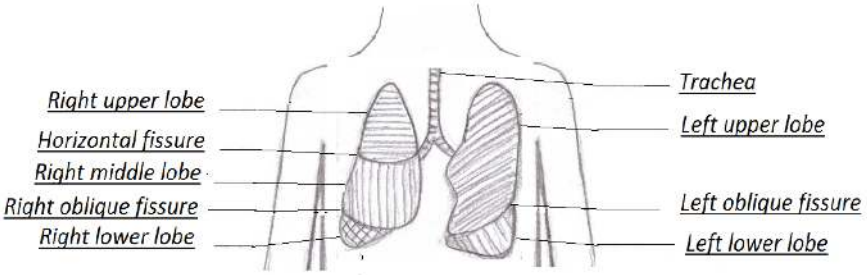
- Grade 1: inspiratory strider
- Grade 2: Inspiratory and expiratory strider
- Grade 3: Inspiratory and expiratory strider and pulsus paradoxus
- Grade 4: Impending apnoea, cyanosis, apathy and marked retractions

Type of breathing patterns

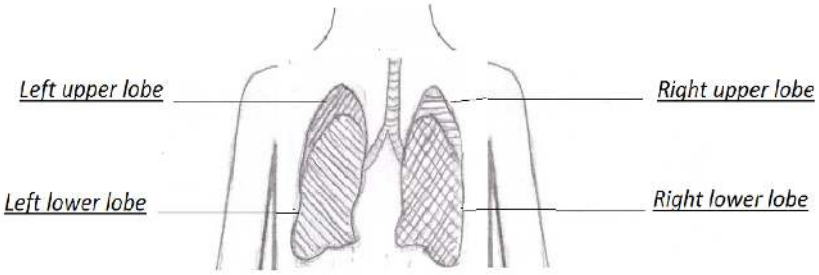
1. **Cheyne- Stokes breathing:** It is terminal pattern of respiration which dependent on CO_2 drive. It is characterised by progressively deeper and faster breathing followed by a gradual decrease in breathing until apnoea. The pattern repeats, with each cycle and usually takes 30 seconds to 2 minutes.
   - First there is rapid and deep breathing which causes fall in PCO_2 in pulmonary blood. When this blood reaches the brain, it inhibits the respiratory centre and causes apnoea.

- As a result of apnoea, alveolar pulmonary PCO_2 gradually builds up. When this blood carrying high CO_2 reaches in the brain, it causes stimulation of respiratory centre and in this way respiration begins again. Thus, initiating the new cycle.
- Causes include left cardiac failure, increased intracranial pressure (trauma, cerebral haemorrhage), and acute kidney injury or sever pneumonia.

2. **Kussmal respiration:** It is deep and rapid breathing pattern that indicates that the body or organs have become too acidotic. The respiratory centre is stimulated and body works constantly to maintain a normal temperature and neutral blood pH. Causes are the conditions which lead to metabolic acidosis like diabetic ketoacidosis, chronic kidney injury etc.
3. **Sleep apnoea:** cessation of air flow for more than 10 seconds, more than 10 times at night during sleep
4. **Shallow and painful breathing:** pleuritic
5. **Paradoxical respiration:** it is paradoxical inward movement of abdomen during inspiration. Causes: diaphragmatic paralysis, COPD etc.

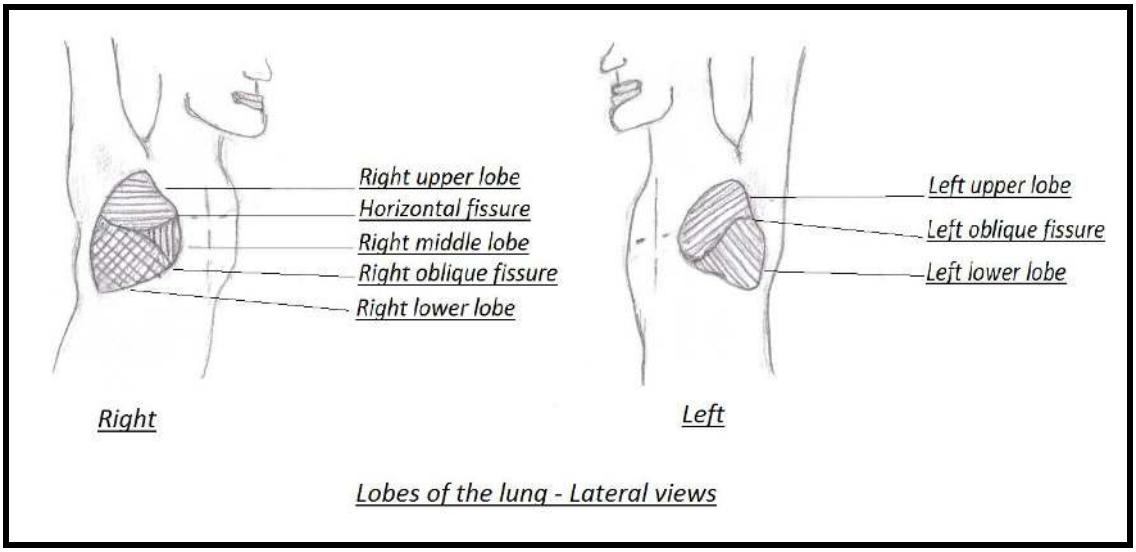


Anterior view



Posterior View

Lobes of the lung



Lobes of the lung - Lateral views

2. PALPATION

Feel for any tendeness, to exclude any local inflammatory condition like Teitze's disease (costochondritis) or fractured rib. Crepitations are felt in case of surgical emphysema. Note the position of mediastinum:

Palpation of trachea (to assess the upper mediastinum)

Stand in front of the patient and place your 3 fingers (ring, middle, and ring finger) in a way that index and ring fingers rest on the right and left sternoclavicular joints respectively while the middle finger is free to feel the gap between the sternomastoid muscle (of either side) and trachea. Reduced gap indicates the shifting of trachea toward that side. A tracheal position 1-2 mm to the right is considered as normal due to aortic arch curving over from right to left displacing the trachea

Conditions which pull the trachea

1. Atelactasis or collapsed upper lobe of lung
2. Fibrosis of lungs
3. Pneumonectomy

Conditions which push the trachea

1. Pneumothorax
2. Pleural effusion
3. Upper mediastinal tumour
4. Retrosternal goiter
5. Bochdalek hernia

When trachea is not displaced to the side of fibrosis or colapse of lung or to the opposit side in case of pleural effusion, this is called as **splinting of trachea**.

Palpation of apex beat (to assess the lower mediastinum)

Apex beat is localised at the most lateral and inferior point at which palpating fingers are raised with each heart beat. Place the palm of your hand over the pericardium and feel the apex beat. If not palpable, then try to feel it by turning the patient to his/her left side.

Following may be the cause when it is not palpable:

1. Thick muscular chest wall
2. Pneumothorax of left side
3. Pleural effusion of left side
4. Pericardial effusion
5. Emphysema
6. Dextrocardia

If apex beat is palpable then localise it by placing the index finger over it vertically. The apex beat is normally situated in the 5^{th} intercostal space in midclavicular line in small children & 1 cm medial to left midclavicular line in older children & adolescents. Now assess if it is at the right position or displaced. This may be displaced "pushed" outward only or outward and downward. Apex beat may also be "pulled" mean displaced inward.

Causes which push the apex beat – outward/downward

1. Left ventricular enlargement
2. Right ventricular enlargement (outwards only)
3. Pleural effusion of right side
4. Pneumothorax of right side
5. Pectus excavatum

Causes of displacement of apex beat inward (pulled)

1. Collapse of right lung
2. Fibrosis of right lung
3. Dextrocardia – apex beat on the right hand side

Vocal fremitis

This is feeling of voice vibration with the flat hand on chest wall. Its not easy to demonstrate in small children.

Method: Place the palm of your hand flat on the identical points on either side of the chest wall and ask the patient to say "One, one, one" or "ninty nine" and feel the vibration. The vocal fremitus may be nornal or

- **Increased vocal fremitus:** in cavitation, collapse with patent bronchus and consolidation (better conduction to the surface)
- **Diminished vocal fremitus:** in asthma, bronchiolitis, pneumothorax, pleural effusion, fibrosis and collapse with obstructed bronchus

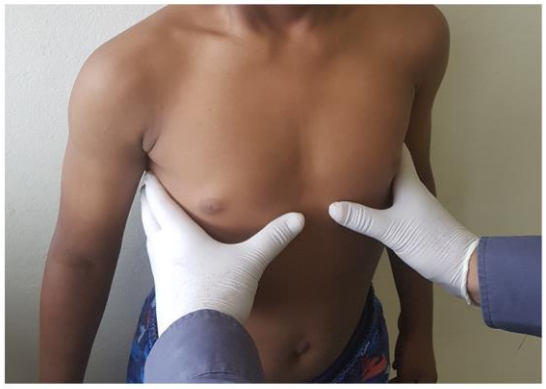
Chest expansion

The angle of Louis is impalpable in children so the first space below the clavicle is the 1st intercostal space. Assessment of symmetrical chest expansion by placing the hands around the thorax of a child is usually not done as breathing in children is shallow.

Following methods ca be used in older children and adolescents

Method 1: Fix the fingertips of either hand at the patient's side and make the thumbs just meet in the midline, in front of the chest. Now ask patient to take deep breaths. See the distance of departure of each thumb from midline and compare them.

Method 2: Encircle the chest with a tape-measure and bring the end in front of the chest below the nipples. Ask the patient to take a deep breath and measure the chest expansion. The normal chest expansion in a 5-year old child is 1cm or more, 2.5 cm in older children and more than 5 cm in adolescents and adults.



Picture: Testing for chest expansion

Note if the chest movement is symetrical or asymetrical:

Asymmetry of chest movement can be due to:

1. Pleural effusion
    - excudate (pus)
    - transudate(clear fluid)
    - or blood
2. Collapse or consolidation
3. Pneumothorax
4. Fibrosis

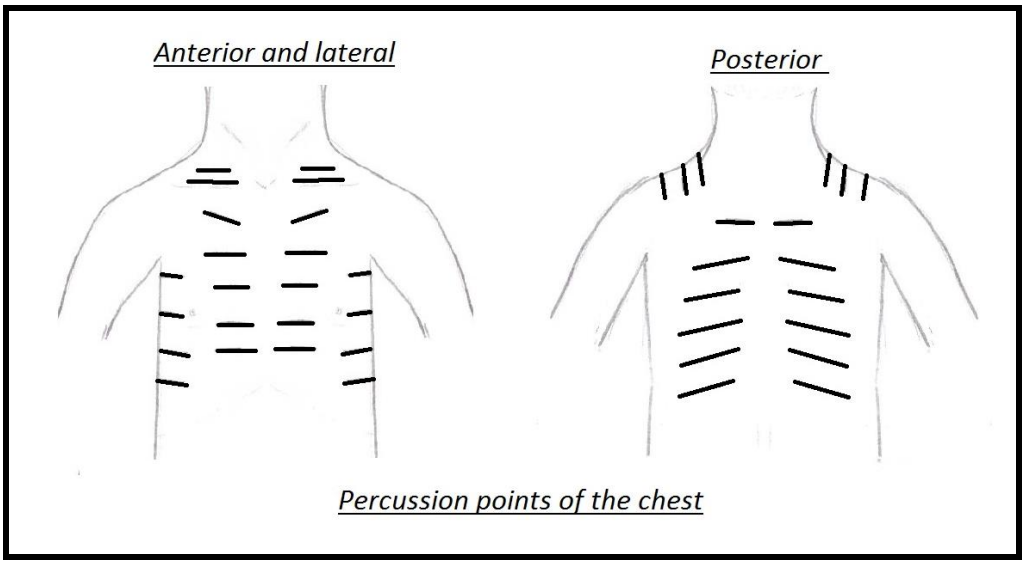
3. PERCUSSION

The most important reason to percuss the chest of a child is to assess for hyperinflation or dullness. The technique for a right-handed person is as follows:

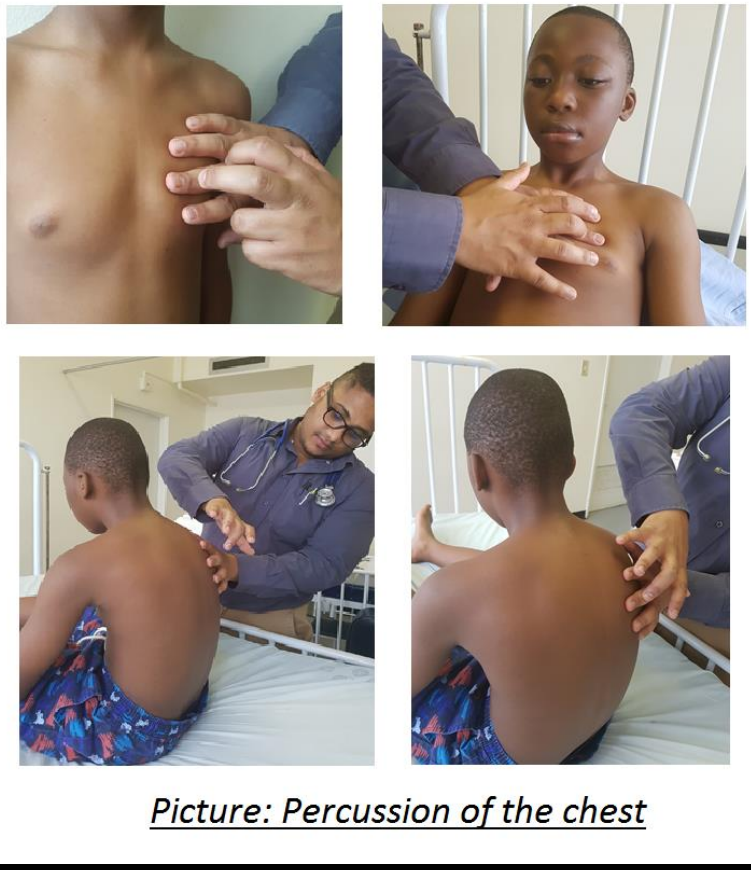
Rules of percussion:

1) The middle finger of left hand is called as **Pleximeter** & and middle finger of right hand is called as **Plexor**

2) Percussion is done from resonant to dull area and comparison is done with respective point on the other sides.
3) Pleximeter should be placed parallel to the border of organ to be percussed in close contact with the body surface and Plexor should strike the middle phalanx of Pleximeter perpendicularly
4) Movements should take place at the wrist and finger joints
5) Strike the Plexor twice and then lift it off the Pleximeter i.e. it should not remain in contact with Pleximeter after striking it because it stops the overtones and hence changes the character of the sound produced.
6) The blow should be no heavier than is necessary to elicit the resonance of the part being examined, and the wrist joint must move loosely.
7) The character of the sound varies quantitatively and qualitatively producing different types of percussion notes.



Percussion points of the chest



Picture: Percussion of the chest

Methods of percussion:

Chest is percussed anteriorly, laterally and posteriorly.

Apical percussion

Rotate the head of the patient to the opposit side and place the pleximeter above the medial third of clavicle and percuss it.

Clavicular percusson

Place a finger above and below the clavicle and stabilise it. Now percuss the clavicle directly without pleximeter.

Anterior percussion

After apical and clavicular percussions move in front of the chest

Precaution should be taken to keep the pleximeter away from the cardiac dullness

Upper border of the liver must be percussed and marked before starting to percuss for the anterior of the chest. Look for obvious differences between left and right chest

Posterior percussion

Patient sits up on the bed and crosses his hands in fron of the chest places on the shoulders and bends forward. Now start percussion and look for obvious difference between left and right chest.

Lateral percussion

Patient sits up on the bed and place his/her hands on the head. Now percuss the lateral sides of chest from above downward and look for obvious difference between left and right chest.

Types of percussion notes

- **Normal resonance**
  A percussion note heard over a normal air-filled lung will be resonant
- **Hyperresonant:** Hyperresonance on percussion indicates too much air within the lung tissue note, that may be due to:
  - Pneumothorax
  - Air trapping in bronchospasm
  - Bronchiolitis
  - Emphysema
- **Decreased resonance** – may be due to:
  - Consolidation
  - Pleural thickening
  - Fibrosis
  - Collapse of lung
- **Dull note**
  - Percussion note over a solid organ like liver, spleen and heart
- **Stony dull -** percussion note over the fluid filled cavity for example:
  - Pleural effusion
  - Ascities
- **Tympanitic note**
  Percussuin note over a cavity containing air. A best example to percuss over an empty stomach.

Signs of hyperinflation

1. Loss of cardiac dullness
2. Upper border of liver pushed down (usually in 4^{th} to 5^{th} intercostal space)
3. Barrel shaped chest – increased AP diameter

Causes of hyperinflation

1. Chronic asthma
2. Emphysema
3. Cystic fibrosis
4. Bronchiolitis
5. Chronic lung disease

4. AUSCULTATION

Patient should sit or lie down supine on the bed with upper garments removed. Now ask the patient to relax, open the mouth and take deep breaths.

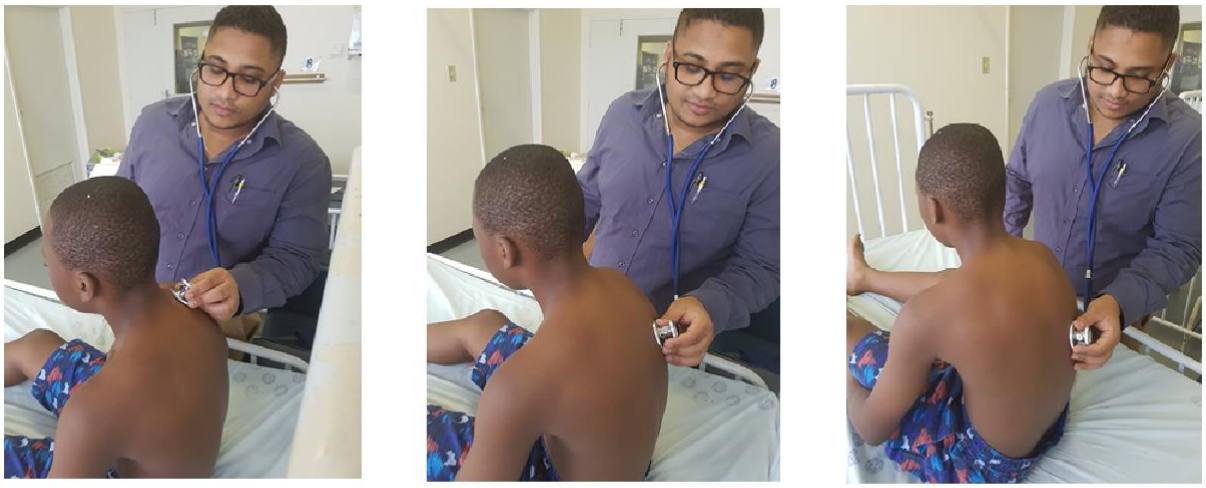
Small children can be examined in their mothers' lap and auscultation can just be done while they are breathing normally.

Warm the chest piece of stethoscope by rubbing it on the palm of your hand and out it on the chest wall in a way that it is in full contact with the chest and should not move on the skin while patient is breathing.

First auscultate anteriorly and then posteriorly. When auscultating, first note character of breath sounds, second character of vocal resonance, and third the presence or absence of other sounds.

Auscultate:

- Anteriorly from clavical down to 6th rib
- Laterally from axilla to the 8th rib
- Posteriorly down to the 11th rib



Picture: Posterior auscultation (example of auscultation)

Breath sounds are two types: vesicular and bronchial

Vesicular breath sound

This is produced by the passage of air in and out of normal lung tissue. It is heard all over the healthy chest and most typically in the axillary and intrascapular regions. Throughout inspiration the sound is fairly intense and of low pitch with a characteristic rustle.

Bronchial breathing

This is pathological sound. It is blowing and hollow in character. Expiration may be prolonged and is harsher and more intense than inspiration. There is pause between inspiration and expiration. When auscultating the chest, the abnormal side is usually the side with decreased breath sounds.

Broncheal breathing may also be produced by the passage of air through the trachea and large bronchi. It is very intense when heard over the trachea but far less intense and of same quality when heard over a diseased lung. The inspiratory sound of bronchial breathing is harsh and becomes inaudable shortly before the end of inspiration. Not to say the child has bronchial breathing if listening near the trachea. Always look for Cor Pulmonale in Chronic Lung Disease

Causes of bronchial breathing

- Consolidation of lung
- Cavitation
- Collapse (with patent bronchus)
- Top of pleural effusion

Vocal resonance

It is difficult to assess in small children but easy in older children and adolescents.

Method: Auscultate when patient say “one, one, one” or “ninety-nine”. The ear perceives, not the distinct syllables, but a resonant sound, the intensity of which depends on the loudness and depth of the patient’s voice and the conductivity of his lungs; the nearer the stethoscope is to a large bronchus, the more intense the sound. Each point examined on one side of the chest should be at once compared with the corresponding point on the other side. Vocal resonance of normal intensity generally conveys the impression of being produced just at the chest-piece of the stethoscope. If it seems to be nearer the ear than this, the resonance is increased. Increased resonance occurs when the lung substance conducts the sound waves set up by the voice more clearly than usual from the bronchi.

Decreased vocal resonance may be due to:

- Reduced conduction
  - Pleural effusion
  - Marked pleural thickening
  - Pneumothorax
- Reduced air flow
  - Generalised COPD
  - Localised lung collapse

Increased vocal resonance may be due to:

  - Consolidation
  - Cavitation
  - Collapse with patent bronchus

Crackles

These are discontinuous crackling or bubbling sounds produced due to the presence of mucous or fluid in bronchi or alveoli. These may be fine (due to the presence of fluid in the alveoli as occurs in heart failure or early stages of pneumonia when there is exudate in alveoli) or coarse (due to secretions in the bronchi in bronchitis or bronchiectasis).

- Early inspiratory: small airway disease – bronchiolitis
- Mid inspiratory: pulmonary oedema
- Late inspiratory: pulmonary fibrosis

Crackles in the inspiration and expiration (biphasic) are present in bronchiectasis

Wheeze

These are prolonged uninterrupted noises, arising in the bronchi due to the partial obstruction of lumen, by swelling of mucosae, by viscous secretion or by constriction of bronchial smooth muscle often associated with turbulent air flow within the lumen. These are high pitched and can be heard without the use of stethoscope.

- Asthma (high pitched and expiratory)
- Bronchiolitis
- COPD

A pleural rub/friction rub:

It is described as grating or creaking sound made when roughened inflamed surfaces of the pleura move over one another during respiration. It has a superficial course, grating, creaking quality surfaces from just beneath the bell/diaphragm of the stethoscope. It is a sign of pleurisy (pleuritic).

Transmitted sounds

These are sounds transmitted from oropharynx to the chest and are common in infants and toddlers with lots of mucous and secretions in their upper respiratory tract. They are rough, sometimes leathery sounds often mistaken for a pleural rub and first hearing.

At end summarise your findings and make a reasonable provisional diagnosis in the light of history and your clinical examination.

Summarise your findings and make a provisional diagnosis:

- Identify the anatomical section of the system affected
- Work out possible aetiology and pathology of the disease process
- Is the organ function is compromised and to what degree?
- Look for complications of the disease process

For chronic symptoms note some of the clues on general examination

- Anthropometry
- Finger clubbing
- Chest deformity
- Generalised lymphadenopathy

Upper respiratory tract infection	Middle respiratory tract infection	Lower respiratory tract infection
• Rhinitis • Otitis media • Sinusitis • Tonsillitis • Mastoiditis • Pharyngitis	• **Spasmodic laryngitis** • Laryngotracheobronchitis • Epiglottis	• Tracheitis • Tracheobronchitis • Bronchitis • Bronchiolitis • Pneumonia • Empyema

Differential diagnosis of airway obstruction according to signs & symptoms

Acute upper airway obstruction	Persistent upper airway obstruction	Lower airway obstruction
• Laryngotracheobronchitis • Acute bacterial epiglottitis • Foreign body aspiration • Retropharyngeal abscess • Bacterial tracheitis • Subglottic oedema after intubation • Angioneurotic oedema	• Laryngomalacia • Vocal card paralysis • Subglottic stenosis • Laryngeal papilomatosis • Obstructive sleep apnoea	• Acute viral bronchiolitis • Asthma • Cystic fibrosis • Foreign body inhalation

Foreign body inhalation should be considered in DD if:

- Strider not responding to therapy
- Asthma not responding to bronchodilators
- Pneumonia not responding to treatment
- Repeated episodes of pneumonia occurring in the same lobe
- Unexplained chronic cough
- Unexplained lobar collapse
- Localised bronchiectasis

Differential diagnosis of pneumonia according to signs and symptoms

Condition	Symptoms	Signs
Bronchiolitis	Cough Wheeze Rapid breathing Poor feeding	Oral frothing Respiratory difficulty Hyperinflated chest Diffuse crackles Bilateral rhonchi
Bronchopneumonia	Cough Wheeze Irritability Fever Poor feeding	Flaring of alar nasi Grunting Respiratory difficulty Unilateral or bilateral crackles Occasional rhonchi
Wheezy bronchitis	Cough Wheeze Low grade fever Irritability	Tachypnoea Recessions Audible wheeze Bilateral rhonchi
Tuberculosis	Contact with a PTB patient Persistent cough may be associated with wheeze or strider Failure to thrive Weight loss and night sweats	Mild fever and tiredness Erythema nodosum Pleural effusion Phlyctenular conjunctivitis Reactive (Porcet) arthritis Hepatosplenomegaly Generalised Lymphadenopathy
Pneumocystis Jirovecii	Fever Non-productive cough	Tachypnoea Intercostal & subcostal recession Prominent hypoxia Respiratory distress with hypoxia out of proportion to the chest findings Fine crackles
Asthma	Family history of asthma or allergies Coughing specially at night Shortness of breath Whistling sound when breathing out Chest congestion and tightness	Breathing difficulty Intercostal and subcostal recessions Hyperinflated chest Wheezing especially during expiration

If an atopic child has chronic cough, who is otherwise thriving, asthma should be considered in differential diagnosis. But if such a child is losing weight or failing to thrive then tuberculosis, cystic fibrosis and HIV related lung disease should be considered.

TUTORIAL 8

EXAMINATION OF CARDIOVASCULAR SYSTEM

OVERALL OBJECTIVE

By the end of this module the student should be able to:

- Differentiate between the child with normal heart and the one with a heart disease
- If there is heart disease; is it congenital or acquired
- Recognise and manage common and life-threatening cardiovascular problems in infants and children
- Recognise by appropriate history taking and clinical examination and by doing appropriate investigations and be able to discuss principles of management of:
    - Cardiac failure (acute cardiac failure, pulmonary oedema)
    - Rheumatic fever (active carditis vs rheumatic heart disease)
    - Congenital heart disease (including but not limited to VSD, ASD, PDA, tetralogy of Fallot, transposition of great arteries)
    - Myocarditis, pericarditis, pericardial effusion
    - Cardiomyopathy

BORDERS OF THE HEART

Left border: from superior to inferior is formed by: Arch of aorta, pulmonary artery, left arterial appendix and left ventricle.
Right border: from superior to inferior is formed by: Ascending aorta, superior vena cava, right atrium and inferior vena cava.
When examining cardiovascular system, specially look for

- Plethora: may be due to cyanotic congenital heart defect
- Jaundice: may be due to sever CCF, hepatic congestion or prosthetic heart valve induced
- Mitral facies: rosy cheeks with bluish ting due to dilatation of malar capillaries associated with pulmonary haemorrhage or severe mitral stenosis due to reduced cardiac out put
- Malar rash may be due to mitral stenosis or pulmonary stenosis
- High arched palate: as a feature of Marfan's syndrome consider
    - Aortic regurgitation due to aortic root dilatation
    - Mitral regurgitation due to mitral valve prolapse
- Teeth: can be source of infective endocarditis
- Tongue, lips, and finger tips: look for central or peripheral cyanosis. If room is artificially lit, look again in natural day light. Avoid saying "pink or blue": say 'cyanosed' or 'not cyanosed'. Tongue has same colour in all races with good blood supply therefore, it presents as an important sign for central cyanosis. If the patient is cyanosed – state whether they are peripherally or centrally cyanosed.
- Mucosa: for petechiae, signs of IEC
- Neck
    - JVP (not commonly practical in small children due to short neck).
    - Central venous pressure height
    - Wave forms

- Abdominojugular reflex test
- Carotid pulse character

- **Signs of Infective Endocarditis, Acute Rheumatic Fever and Cardiac Failure:**

Infective Endocarditis	Acute Rheumatic Fever	Cardiac Failure
1. Fever 2. New or changing murmur 3. Pallor 4. Splenomegaly 5. Clubbing 6. Haematuria 7. Splinter hemorrhages 8. Osler's nodes 9. Janeway lesions 10. Arrhythmia 11. Tachycardia 12. Heart failure	Revised Jones criteria <u>Two Major (PECCS)</u> 1. Polyarthritis 2. Erythema marginatum 3. Carditis 4. Chorea 5. Subcutaneous nodules <u>Or 1 major and 2 Minor criteria (FAPPLE)</u> 1. Fever 2. Arthralgia 3. Previous Rh fever 4. Prolonged P-R interval 5. Leucocytosis 6. Elevated CRP & ESR	Two ↑ in number 1. RR – tachypnoea 2. HR – tachycardia may lead to gallop rhythm Two ↑ in size 1. Cardiomegaly 2. Hepatomegaly

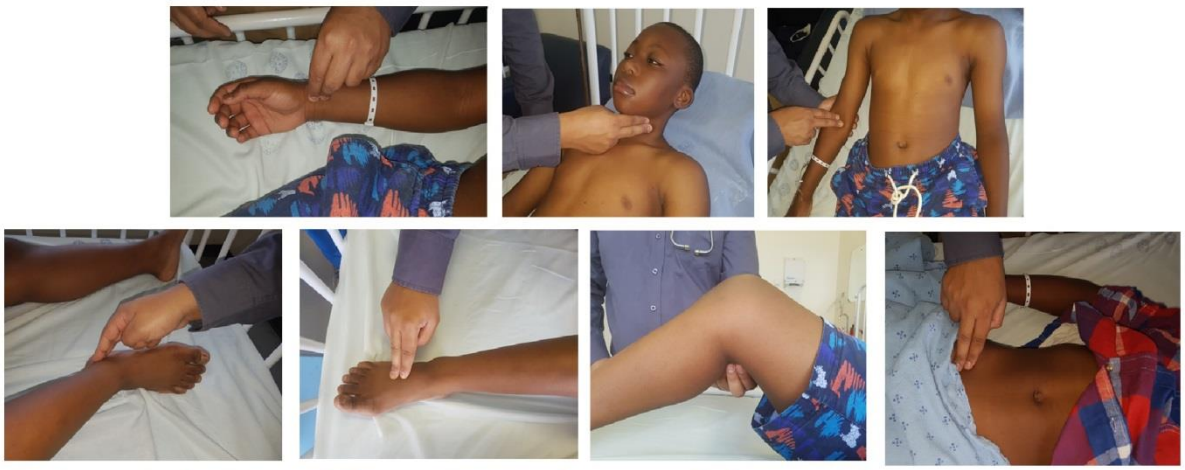
An approach to cardiovascular examination

- Start your examination at the periphery and work towards the heart.
- Look for cyanosis, clubbing, respiratory difficulty, pallor or plethora or polycythaemia.
- Jugular venous pulse and pressure are difficult to appreciate in infancy due to relative shortness of neck.

1. PULSES

Pulse is a wave of increased pressure which passes along the arteries with each beat of heart. Following points need to be noted while examining the pulse:

- **Feel** radial, bracheal, axilary, facial, temporal, carotid, dorsalis pedis, posterior tibial, popliteal and femoral pulses - are all pulses present?



Picture: Palpation of the various pulses - radial, carotid, bracheal, dorsalis pedis, posterior tibial, popliteal and femoral (from top right to left)

- **Radio-femoral delay** (RFD) only relevant in the older child (>6yrs) once collaterals have had time to develop. Reduction of one femoral only is not due to coarctation but due to local trauma/cardiac catheterisation. Looking for RFD is important if there is history of hypertension.

Picture: Radio-femoral delay

- **Pulse rate:** pulse rate is counted by placing the finger tips on the radial artery while the forearm is pronated and wrist slightly flexed. It is counted for 1 minute. Normal pulse rate ranges between:

Age	Beats per minute
Birth to 3 months	120-160
1 yr	80-140
2 yrs	80-130
3 yrs	80-130
Older	70-115
In febrile condition each 1 degree ^{o}C rise in temp above normal increases pulse rate by 10 per min	

Note:

- Tachycardia:
  - Causess: anxious child, fever, anaemia, shock , heart failure, hyperthyroidism and in emotional state.
- Bradycardia:
  - Causes: junior athelete, drugs (beta blockers, digoxin), complete heart block, increased intracranial pressure, and hypothyroidism.

- **Rhythm:**

Here the time interval between the beats is noted which is equal in most individuals. However in some patients there may be some disturbance during respiration. During inspiration, pulse becomes rapid while during expiration this becomes slow. This is a normal phenomenon which is called Sinus Arrythmia. There are other forms of arrythmias like:

- **Regularly irregular heart rate:** here the pulse is irregular but its irregularity is manifested after regular intervals like premature beat which comes after fixed number of beats; sinus arrythmia and 2nd and 3rd degree heart blocks.
- **Irregularly irregular heart rate:** here beats are non-equidistant seen in atrial fibrillitaion, multiple ectopic beats, and atrial flutter with variant block (uncommon in small children)

- **Volume** (small volume, large volume or absent)

This is amplitude of the wave passing through the blood vessel during the ventricular contraction. In other words this is the force by which the palpating fingers are lifted up. Conditions causing increased cardiac out put are associated with increased volume of pulse.

Small volume (narrow pulse pressure)	Full volume (increased/wide pulse pressure)

• Cadiac failure
• Shock: circulatory failure due to hypovolumia
• Out flow obstruction:
  ○ aortic stenosis
  ○ pericardial effusion
	• Anaemia
	• Carbon dioxide retention
	• Thyrotoxicosis
	• PDA & truncus arteriosus
	• Fever – sepsis (vasodilatation)
	• A-V malformation

- **Character**
    - Normal
    - Slow rising - aortic stenosis
- **Collapsing or water hammer pulse**

This is a rapid, high volume nonsustained pulse.

Method: Hold the wrist of the patient with your hand. Feel for radial pulse and then suddenly raise the arm of the patient straight up. The volume of the pulse will increase if it is water hammer pulse. Sudden rise is due to increased stroke volume and collapsing character is due to backward leakage (seen in aortic incompetance) or due to low peripheral resistance which enhances the blood flow to peripheral vasculature during diastolic phase. This is seen in conditions which lead to increase in pulse pressure like:

- Aortic incompetence
- Thyrotoxicosis
- Fever
- Arteriovenous fistula
- Severe anaemia
- PDA (large volume – rapid collapse – often a neonate)
- Truncus arteriosus
- In adults: Beri-Beri and females; pregnancy

Picture: Collapsing pulse

Pulsus deficit

There is difference in the rate of pulse and the apex beat. This is seen in arterial fibrillation and multiple ectopic beats. This is due to the fact that certain beats are not strong enough to push the blood in the peripheral blood vessels. This is also called as missing beat.

Pulsus paradoxus
In normal circumstances there is decrease in the volume of pulse during inspiration due to decrease (5-10 mm of Hg) in cardiac output and the blood pressure. If there is exaggeration of normal response i.e., the volume of pulse decreases markedly or even become impalpable this is called as pulsus paradoxus. On auscultation the heart sounds are still audible even when the pulse becomes impalpable. This is present in the following conditions:

- Constrictive pericarditis
- Pericardial effusion
- Severe asthma and severe strider

Pulsus bisference
This is a combination of anacrotic and collapsing pulses. This is felt twice in one beat and is seen in combined lesions of aortic stenosis and aortic incompetence.

Delayed pulse
Normally, the radial and the femoral pulses can be felt simultaneously but in case of coarctation of aorta, the femoral pulse is delayed.

2 BLOOD PRESSURE

Types of blood pressure (BP)

- **Systolic** blood pressure is maintained by the elasticity of blood vessels and stroke volume
- **Diastolic** blood pressure is regulated by peripheral resistance
- **Pulse pressure** is the difference between systolic & diastolic blood pressure.

Method of checking the blood pressure
There are many types of sphygmomanometers like Mercury, Aneroid, and Electronic. Mercury sphygmomanometer is preferably used and students are also examined on this BP apparatus specifically in adolescents and adults as electronic sphygmomanometers are routinely used in infants and small children.

Patient should be sitting at ease or lying down on the bed comfortably (especially if a small child). Now place the sphygmomanometer at the level of heart and of doctor's eye. Choose the correct size of cuff which should cover 2/3rd of the upper arm. A range of cuff widths – 7cm, 9cm, 11cm and 13cm – will be required. The largest cuff which fits comfortably around the arm should be applied. There are two methods of taking blood pressure i.e., palpatory and the auscultatory.

Krotokof sounds (KS)
KS are blood flow sounds which are observed while taking blood pressure with a sphygmomanometer over an artery (most commonly brachial artery in the antecubital fossa). These sounds appear and disappear as the BP cuff is inflated and deflated. Following are phases of KS and a diagramatic presentation of reading a blood pressure of an adolescent.

Phases of Krotokof Sounds

	Systolic	→	→	Diastolic →	
Silence	Phase 1 Clear tapping sound	Phase 2 Softens and becomes swishing sound	Phase 3 Sharper sounds become criper sound	Phase 4 Muffling and Blowing sound	Phase 5 Silence .. ^ ..
	120	110	100	90	80

Palpatory method
Palpate the radial pulse at the wrist and inflate the armlet. The mercury column will rise in the manometer. Now note the level of mercury column when the pulsation in the radial artery stops. Inflate further so that the column of mercury should further rise. Then deflate the armlet by opening the screw slowly. The level of mercury column at which the first pulse is felt, is noted. Mean of these two is systolic blood pressure.

Auscultatory method
Inflate the armlet and make the mercury column to rise above the level of systolic BP which was determined by the palpatory method. Now put the chest piece of stethoscope gently on the brachial artery in antecubital fossa and deflate the armlet slowly. Note the column of mercury when the sound is heard. This is systolic BP. Now gradually keep on deflating the cuff. The sound will come louder and louder and suddenly this will change its character, i.e., it becomes soft and finally inaudible. Note this point when it becomes soft, which is diastolic BP.

Check four limb BP if the child has primarily a CVS problem. For this patient should lie down on the bed with face downward. The cuff is applied on the lower part of the thigh and the stethoscope is applied on the popliteal artery to note the BP. Systolic is where sound is first heard. Diastolic is where sound softens and not where it disappears

Normal BP

- Normal systolic blood pressure = 2 x age in yrs + 65
- Mean diastolic blood pressure = 55 + age in yrs
- Mean systolic blood pressure = 90 + age in yrs
- Upper limits of normal blood pressure
    - Mean + 20 for diastolic
    - Mean + 18 for systolic

Single raised values are of no significance; they must be repeated several times. Blood pressures recorded on admission to hospital may be unreliable because of combination of anxiety plus obesity in the child.

Age Years	**Systolic Blood pressure**		**Diastolic Blood Pressure**	
	Systolic BP	Upper limit of normal	Diastolic Bp	Upper limit of normal
2-4	90	110	64	80
6	100	120	66	82
8	105	125	70	86
10	110	130	72	88
12	115	135	74	90
14	120	140	76	92
	Mean systolic BP = 90+age in yrs BP rises by aproximately 2.5mmHg per year thereafter.		Mean diastolic BP = 55+age in yrs Up to the age of 12 years there is no appreciable differences between boys' and girls' BP	

3. JUGULAR VENOUS PRESSURE (JVP)

JVP is unhelpful in the infant and small child because of their short necks but is useful in older children and adolescents.

Technique

Patient should recline at an angle of 45 degrees. This is the angle between the bed and the back of the patient. Now look for prominent jugular veins. The prominence of jugular veins in this position indicates some pathology. The basis of this test is that in healthy individuals in upright posture the upper level of venous column corresponds with manubrium sterni. This remains behind or slightly above the level of clavicle when the person is reclining at the angle of 45 degree so we cannot see the prominent veins. But in certain conditions like congestive heart failure (other conditions listed below), the column of blood in jugular veins rises up. So even when patient reclines at an angle of 45 degrees, the veins become prominent and visible.

Hepatojugular reflex (HJR)

When pressure is sustained over hepa for 10 seconds and there is a rise of >2-3 cm in children (4 cm in adults), it is considered significant which is called as HJR also known as abdominal jugular reflex.

The causes of prominent neck veins

- Congestive cardiac failure
- Constrictive pericarditis
- Pericardial effusion
- Cardiac temponade
- Enlarged lymphnode or mass compressing superior vena cava
- Tricuspid stenosis or incompitance
- Volume over load

4. PRECORDIUM

INSPECTION

- Look for congenital chest deformity or pericardial bulge due to bowing forward of sternum and the ribs, giving the chest an overblown appearance.
- Look for visible ventricular impulse:
- The right ventricular impulse may be visible under the xiphisternum.
- The left ventricular impulse or apex beat is frequently visible in children with hyperdynamic circulation (due to fever or excitement) and in children with true left ventricular enlargement.
- Look for any scars on precordium, do not miss thoracotomy or sternostomy scars (complex cardiac surgery), scar at groin (cardiac catheterisation) etc.

PALPATION

Palpation includes localisation of apex beat, appreciation of palpable sounds or murmur and search for right or left venricular enlargement

1. Apex beat:

At the most lateral and inferior point, left hand side of precordium

Method for localising the apex beat

Make the child to lay down and place the palm of your right hand over the left precardium. Feel the apex of heart beating under your palm. Now localise it by placing the index finger over it vertically and count the intercostal space.

The apex beat is normally found in the 4th intercostal space (ICS) in mid clavicular line (MCL) in infants and toddlers. In school children it is at 4-5th ICS in MCL and 1 cm medial to the left MCL in adolescents and adults. Followings may be the causes if it is not palpable:

- Plump, healthy infant and toddler
- Pleural effusion of left side
- Pneumothorax of left side
- Pericardial effusion
- Dextrocardia

Picture: Locating the apex beat

If apex beat is displaced, check position of the trachea to make sure that this is due to cardiomegaly and is not displaced due to a mediastinal shift.

Thrill: a palpable murmur is referred to as a thrill. A thrill in the suprasternal notch may suggest coarctation or aortic stenosis

2. **Search for right or left venricular enlargement**

Right ventricular enlargement (RVE) is outwards and downwards.

Method of assessing ventricular enlargement

Right ventricular enlargement: Place fingertips of your right hand between 2^{nd} , 3^{rd} and 4^{th} ribs along the left sternal border or use ulner border of your right hand and feel for tap of lift. A slight ventricular tap in a thin child may be a normal finding. An epigastric pulsation and a left parasternal lift is indicative of right ventricular hypertrophy (RVH).

Left ventricular enlargement: See method of localising apex beat (above). Apex beat displacement towards left may be due to left ventricular enlargement.

Causes of apex beat displacement

Apex beat displaced to left side

- LV enlargement
- Scoliosis
- Pectus excavatum
- Pneumothorax on the right side
- Pleural effusion of the right side

Apex beat displaced to the right side

- Congenital dextrocardia - feel for the liver (Kartagener's syndrome)
- Acquired dextroposition – heart pushed or pulled to the right
- Left diaphragmatic hernia
- Collapsed lung in the right side
- Percussion is really only used if a large pericardial effusion is suspected and then the dullness will extend beyond the apex beat

Character of the apex beat

Character of apex beat is of two types:

1. **Heaving:** Place index finger of your right hand over the apex beat and note if it gently lifts the palpating finger. If the tip of finger is moving significant upward with apex beat during systole and downward during diastole this is called as heaving of apex beat. This is present in left ventricular hypertrophy.

   Causes of left ventricle enlargement

   - Mitral incompetence
   - Aortic incompetence
   - Aortic stenosis
   - Hypertension

- Coarctation of aorta – older children
- Tricuspid atresia
- AVSD

2. **Tapping:** This is palpable first heart sound. In this the apex strikes the palpating finger and goes back nicely. This is present in case of mitral stenosis or tricuspid stenosis leading to RVH

Causes of right ventricular enlargement

- Secondary to failure of LV
- Mitral stenosis
- Pulmonary stenosis
- Cor-pulmonale
- Tricuspid incompetence
- Pulmonary incompetence
- Primary pulmonary hypertension
- Coarctation in infants

Palpable P2 can also be felt in pulmonary area in patients with pulmonary hypertension

Picture: Palpation of P2

PERCUSSION

Percussion has got limited value in examination of cardiovascular system especially in children but can be of value in an academic clinical exam. Therefore, technique should be leaned which is same as for adults.

Percussion of right border of heart: first percuss the upper border of the heart and then percuss in vertical direction, from lateral to medial side in 4th intercostal space. Mark where percussion note becomes dull this will be the right border of heart.

Percussion of left border of heart: left border of the heart runs from above downward and laterally, so percussion is done in an oblique fashion. Now do the percussion in 4th intercostal space, on left chest from left to medial side till the percussion note becomes dull. Same way percuss the 3rd and 5th intercostal spaces and mark the left border of the heart.

The area of dullness is decreased in case of pneumothorax and increased in pericardial effusion.

AUSCULTATION

By the time auscultation is performed a short list of possibilities should have been compiled:

- Peripheral findings – pulse, blood pressure, JVP
- Cyanotic or acyanotic
- Heart failure or no heart failure
- Precordium findings

Auscultation is done in all four areas of the heart which are as follows:

1) Mitral area
2) Tricuspid area
3) Aortic area
4) Pulmonary area

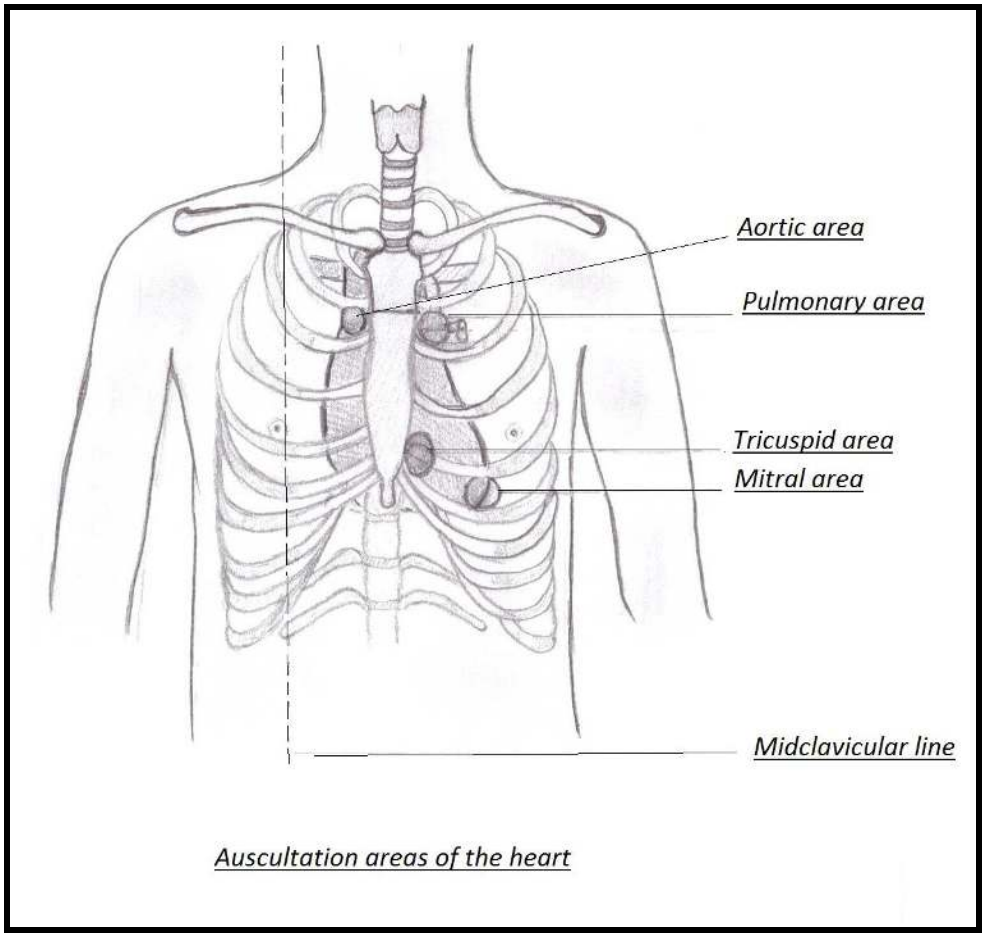
SURFACE MARKING OF AREAS OF THE HEART

Mitral area: it corresponds with the apex beat which is over the 5^{th} intercostal space slightly medial to left mid clavicular line

Tricuspid area: this is near the lower end of left border of sternum in the 4^{th} and 5^{th} intercostal spaces

Pulmonary area: this is over the left second intercostal space near the left border of sternum.

Aortic area: there are 2 aortic areas. First is over the right second intercostal space near the sternum (A1) and the second is over the left third intercostal space near the sternum.



Auscultation areas of the heart

POSTURES DURING AUSCULTATION

When listening child should be calm, use both diaphragm and bell (Paediatric stethoscope is preferable), and note any variation with respiration. Patient may need to resume following three postures if possible, to best hear the different types of murmurs.

1. While patient lies flat on the bed, auscultate all the 4 areas
2. Turn the patient on his left side and auscultate at the apex with bell of stethoscope. In this posture murmur of mitral area becomes more audible.
3. Then let patient sit up and bend forward. Auscultate over aortic and pulmonary areas. Early diastolic murmur of aortic area is best heard in this posture.

Keep in mind the old adage: 'sounds first, murmurs second'. During auscultation note the followings:

- Heart rate and rhythm
- Intensity of heart sounds
- Splitting of heart sounds
- Pericardial friction rub
- Clicks and opening snaps
- Murmurs

1. HEART RATE AND RHYTHM

Two heart sounds are normally heard:

- **First heart sound** is produced by the closure of atrioventricular valves (mitral & tricuspid)

- **Second heart sound** is produced by the closure of pulmonary and aortic valves.

These two heart sounds correspond, the words "Lub" and "Dup". Now count the Lub and Dup for one minute. This will be the heart rate. Note whether rhythm is regular or irregular.

Rhythm becomes irregular in the following conditions:

- Extra systole
- Atrial flutter
- Atrial fibrillation
- Multiple ectopic beats

2. INTENSITY OF HEART SOUNDS

First heart sound

The intensity of first heart sound is increased in:

- Mitral stenosis
- Systemic hypertension
- Tachycardia

The intensity of first heart sound is decreased in:

- Rheumatic carditis
- Mitral incompetence
- 1st degree heart block

Second heart sound

Second heart sound has two components: aortic and pulmonary

Increased intensity of aortic component is seen in:

- Hypertension
- Atheroma of aorta (uncommon in children)
- Aortic aneurysm (uncommon in children)

Decreased intensity of aortic component is seen in:

- Aortic stenosis
- Aortic incompetence

Increased intensity of pulmonary component:

- Feel for palpable P2 at 2nd ICS. PA gets dilated and becomes palpable.
- Palpable pulsation on the epigastric region may be due to pulmonary hypertension.
    - Increased pulmonary blood flow may be due to PDA, ASD, and large VSD.
    - Other causes may be mitral stenosis and cor-pulmonale.

Decreased intensity of pulmonary component is seen in:

- Pulmonary stenosis
- Pulmonary incompetence
- Fallot's tetralogy

3. SPLITING OF HEART SOUNDS

Splitting of first heart sound:

First heart sound is produced by the closure of atrioventricular valves. This sound may normally split as mitral valve closes earlier than tricuspid valve.

Wide splitting may also occur in:

- Mitral stenosis
- Atrial septal defect.

Splitting of second heart sound:

Second heart sound is produced by closure of aortic & pulmonary valves therefore it has two components:

- Aortic (A2)
- Pulmonary (P2)

A2 is audible over whole of the precordium while P2 is audible over pulmonary area. Normally aortic valve closes earlier than pulmonary valve. A widely split and fixed S2 is found in conditions that prolong the RV ejection time or that shorten the LV ejection.

Pathological causes of splitting of 2nd heart sound are:

- Pulmonary hypertension
- ASD or partial anomalous pulmonary venous return (PAPVR) – volume overload
- Pulmonary stenosis (pressure overload)
- RBBB (a delay in electrical activation of the RV) delays the completion of RV ejection
- Mitral regurgitation (a decreased forward output seen in this condition shortens the LV ejection time, making aortic closure occur earlier than normal).

A single S2 is found in the following situations:

- When only one semilunar valve is present (aortic or pulmonary atresia)
- When the P2 is not audible (Transposition of Great Arteries (TGA), Tetralogy of Fallot (TOF), severe Pulmonary Stenosis (PS).
- When aortic closure is delayed (sever Aortic Stenosis [AS]
- When P2 occurs early (sever Pulmonary Hypertension)
- Keep in mind physiological splitting of second heart sound

Third heart sound:

Rapid ventricular filling (normal in healthy children). Childrens' hearts go faster than adults so you may not be able to listen it normally.

4. GALLOP RHYTHM

Third heart sound occurs in early diastole at the time of maximal ventricle filling. This may occur in any healthy young adult but in any other clinical setting its presence indicates abnormal ventricular filling. The most important causes of this are left ventricular failure and mitral regurgitation.

Fourth heart sound occurs when bolus of blood is delivered into the ventricle from atrial contraction. It can be caused by an increased stiffness or non-compliance of the ventricles.

When heart rate is rapid, diastole is shortened and the third and the fourth heart sounds may coincide. When this occurs the amplitude of the sound increases and is more easily detected, giving rise to a summation **gallop rhythm**, so called because it gives the auditory impression of a galloping horse. Third and fourth heart sounds may originate from either right or left ventricles. These diastolic sounds are best heard with the bell of a stethoscope and auscultation should routinely include a search of these sounds with the patient turned slightly onto the left side.

5. MURMURS

Murmurs are due to turbulence in the blood flow at or near a valve or an abnormal communication within the heart. It follows that a loud murmur may originate from a rather small defect such as ventricular septal defect. Equally a soft murmur may originate from a large abnormal orifice as in very sever aortic regurgitation.

Therefore, while it is important to note the intensity of a murmur, one should not make immediate deductions about its importance solely from its loudness. Not all murmurs are produced by a structural disorder of the heart; they may be due to abnormally rapid flow of blood through a normal valve. Such murmurs are called as flow murmurs.

When examining a murmur following points must be noted

1) Decide time of occurrence: note if it is systolic or diastolic or both. This can be made out by synchronising the murmur with carotid pulse or by apex beat.
2) Pitch and quality: high or low, harsh or blowing
3) Grade them according to the intensity of murmur:
    a. **Grading of systolic murmurs:**
        - Grade I: very faint
        - Grade II: medium intensity
        - Grade III: loud without thrill
        - Grade IV: loud with thrill
        - Grade V: very loud (stethoscope must be on the chest wall to listen it)
        - Grade VI: Murmur audible with stethoscope off the chest wall
    b. **Grading of diastolic murmurs**
        - Grade I: barely audible
        - Grade II: Faint but audible
        - Grade III: Easy to hear
        - Grade IV: loud
4) Note point of maximum intensity
5) Note the direction of propagation beyond the precordial area
6) Note the character of murmur
7) Note changes with respiration and posture
8) Then feel for a big liver (CCF) and spleen (IE)
9) Is there a thrill?

Thrill: It is purring sensation which is felt by the palpatory hand which is present in a palpable murmur at suprasternal & supraclavicular areas. It is of two types (systolic or diastolic) which can be made out by synchronising it with apex beat or carotid pulse.

Systolic: It corresponds with systolic murmurs. When apex beat strikes the palpating hand, thrill felt during this time is systolic

Diastolic: When apex beat is away from the hand, thrill felt during this time is diastolic
Same rule applies in case of **carotid pulse:** when you feel the pulse, it is systolic & when you do not feel, it is diastolic

Causes of systolic murmurs

Ejection systolic murmur	
Innocent flow murmur	Left sternal edge or Pulmonary area
Anaemia	Left sternal edge or Aortic area
ASD	Pulmonary area - (mid diastolic) Left 2nd intercostal space
Pulmonary stenosis	Pulmonary area – (can be mid diastolic in peripheral PS) Left 2nd intercostal space
Aortic stenosis or	Aortic area, Right 2nd intercostal space to carotids in AS
Bicuspid aortic valve	
Mitral valve prolapse	Apex
Pansystolic (holosystolic) murmurs	
VSD	Left sternal edge, 4th intercostal space
Co-arctation of aorta	Left sternal edge and between scapulae
Mitral regurgitation	Mitral area - radiates to axilla
Tricuspid regurgitation	Tricuspid area

Causes of diastolic murmurs

- ASD
- VSD
- Mitral stenosis
- Aortic regurgitation

Causes of continuous or machinery murmurs

- PDA
- Coarctation of aorta

CONGENITAL DISORDERS ASSOCIATED WITH HEART DISEASE

1. Major syndromes with cardiovascular abnormalities

- **Downs:** (Trisomy 21): AVSD (30%), VSD, ECD

- **Turner's:** (XO): Coarctation of aorta, bicuspid aortic valve, aortic stenosis, hypertension, aortic dissection later in life,
- **Alagille:** peripheral pulmonary artery stenosis
- **CHARG association:** TOF, truncus arteriosus, aortic arch anomalies
- **Di-George:** interrupted aortic arch, truncus arteriosus, VSD, PDA, TOF
- **Pierre Robin:** occasional VSD, PDA

2. Intrauterine infection

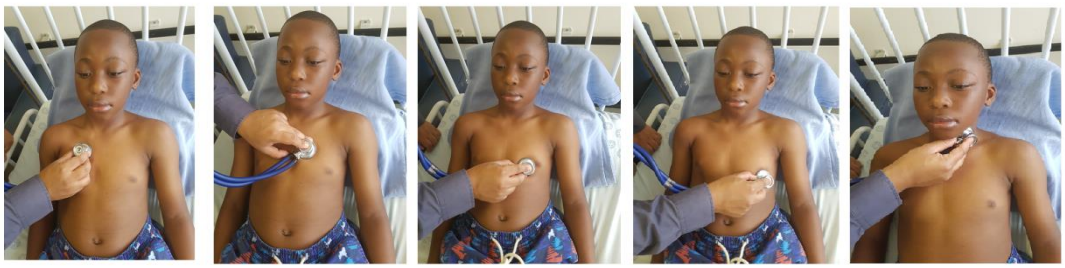
- Rubella (esp. in the 1st trimester): PDA, septal defects, peripheral pulmonary valve stenosis

3. Maternal disease

- Diabetes: Infant of diabetic mother: TGA, VSD, COA, cardiomyopathy, PPHN
- Systemic lupus erythematosis: congenital heart block

4. Drugs in pregnancy

- Anticonvulsants: aortic stenosis, pulmonary stenosis, coarctation of aorta
- Foetal warfarin syndrome: TOF, VSD
- **Foetal alcohol syndrome:** VSD, PDA, ASD, TOF



Picture: Auscultation of the heart and the neck

AT THE END OF CVS EXAMINATION MAKE A REASONABLE ASSESSMENT

1. **What is the lesion?**
   Decide if
   - Congenital (usually a young child) or
   - Acquired (usually an older child)
2. **If congenital**, decide if ACHD or CCHD and offer the most likely lesion with a differential diagnosis
3. **Is there Congestive cardiac failure** (CCF)? (big heart/big liver/fast heart/fast breathing)
4. **Is there pulmonary hypertension** (PHT)? (loud P2 and evidence of RVH)
5. Is there cor-pulmonale
6. **Is there infective endocarditis** (IE)?
7. **If congenital HD**, is there a system associated?
8. **If acquired HD** (usually Rheumatic HD, sometimes CMO) is there Acute Rheumatic Fever at present?
9. **Is there growth**/development failure?

A PRACTICAL APPROACH TO AUSCULTATE A PRECORDIUM OF A CHILD (KZN DOH)

Commence auscultation at apex, with diaphragm of stethoscope then bell (for diastolic)
Work across to and up the sternal border – tricuspid, pulmonary and aortic areas

1. Listen to each component of cardiac cycle carefully
2. Note the intensities of S1, S2
3. Whether S2 splits normally with respiration
4. Listen for added sounds
5. Then for systolic and diastolic murmurs
6. Note radiation of any murmurs to axillae or carotids
7. Next sit the child up and listen to any murmur variation with this change in position
8. Listen with the child in full expiration for the subtle early diastolic murmur of aortic incompetence
9. Listen at the back for radiation of murmur and for any pulmonary adventitious sounds
10. Inspiratory crackles with left ventricular failure
11. Variable findings with coexistent chest infection in Kartagener's syndrome
12. **Lay the child down** and examine the abdomen for:
    a. Hepatomegaly – CCF
    b. Pulsatile liver – tricuspid incompetence
    c. Splenomegaly – Subacute Bacterial Endocarditis (SBE)
13. **Then feel for ankle oedema**
14. Request urine analysis for blood – SBE
15. Then temperature chart – SBE
16. If SBE does appear likely, request for an ophthalmoscope to detect Roth spots
17. Give your succinct diagnosis based on your clinical findings and only after this you request the CXR and ECG.

TUTORIAL 9

THE EXAMINATION OF ABDOMEN

OVERALL OBJECTIVE

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

1. Do a full clinical examination of abdomen and able to make a reasonable provisional and differential diagnosis of vomiting, gastroenteritis, failure to thrive, hepatomegaly, splenomegaly and jaundice in infants and children.
2. To list the complications and principles of management of chronic diarrhoea, malnutrition, chronic liver disease and portal hypertension.

EXTRA GENERAL FEATURES INCLUDE

Signs of Chronic Liver Disease	Signs of Chronic Liver Failure
• Jaundice • Ascites • Spider telangiectasia • Spider navi • Palmer erythema • Finger clubbing • Pigmentation • Itching	• Foetor hepaticus, scratch marks, pruritus • Malabsorption of vitamin D: Rickets • Malabsorption of Vit K: Bleeding/bruising/ haemorrhage/ epitasis/ purpura • Splenomegaly - Portal hypertension • Hepatic encephalopathy • Vit A deficiency: Xerophthalmia, corneal xerosis, follicular hyperkeratosis

Spider telangiectasia

It is a branched group of dilated capillary blood vessels forming a spiderlike image on the skin. It is compressible and blanchable with adequate pressure (glass slide). Generally, as the pressure is released the central feeding vessel can be seen and which often pulsates.

Spider navi

These are spider shaped small reddish marks formed by the dilatation of central arterioles from which small vessels radiate. They are found on the chest above the nipples, face forearm, and sometimes dorsum of the hands. These are present in case of liver cirrhosis.

Complications of cirrhosis (HEPATIC)

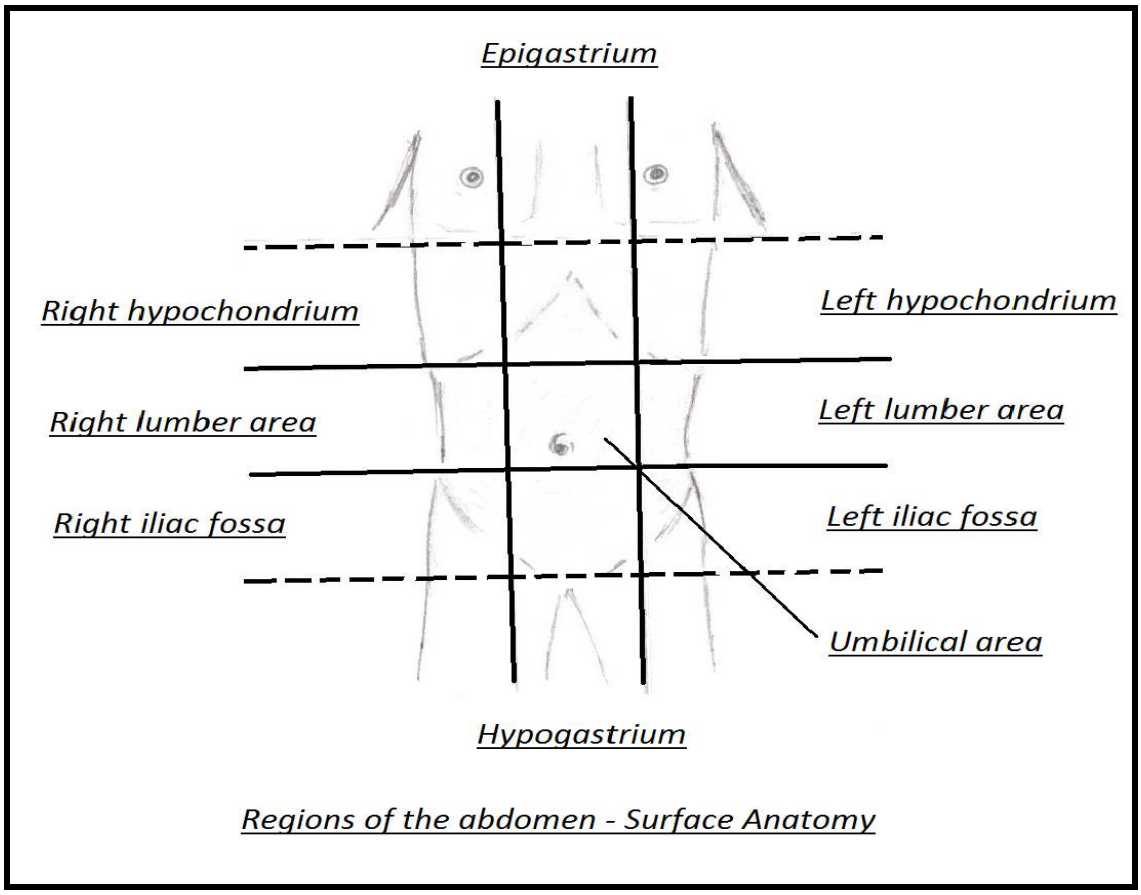
- Hepatic encephalopathy: hepatorenal syndrome, hepatopulmonary syndrome
- Esophageal varices
- Portal hypertension
- Protein energy malnutrition(PEM)
- Ascites
- Thrombosis of portal vein
- Infection/peritonitis
- Coagulopathy

EXAMINATION OF ABDOMEN

After taking permission from the parents, talk to the child and explain what you are going to do. Expose the abdomen and lower chest with child laying on his her back.

In small children, examination is done on the mother's lap if they are anxious, otherwise mother can stand at the upper end next to you, holding the hand of child to make him her more comfortable and relaxed. Leave the genitalia to examine at the end.

The abdomen is divided into 9 areas as described in the following diagram.



Regions of the abdomen - Surface Anatomy

The examination of abdomen will include inspection, palpation, percussion, auscultation, measurement of abdomen and rectal examination.

1. INSPECTION

Bladder should be emptied and nappy changed before the abdominal examination
Child should lie flat, comfortable and fully relaxed, hands by his/her side
No pillow under the head, good light and good exposure: from below nipples to include inguinal area.

Do inspection and note the followings:

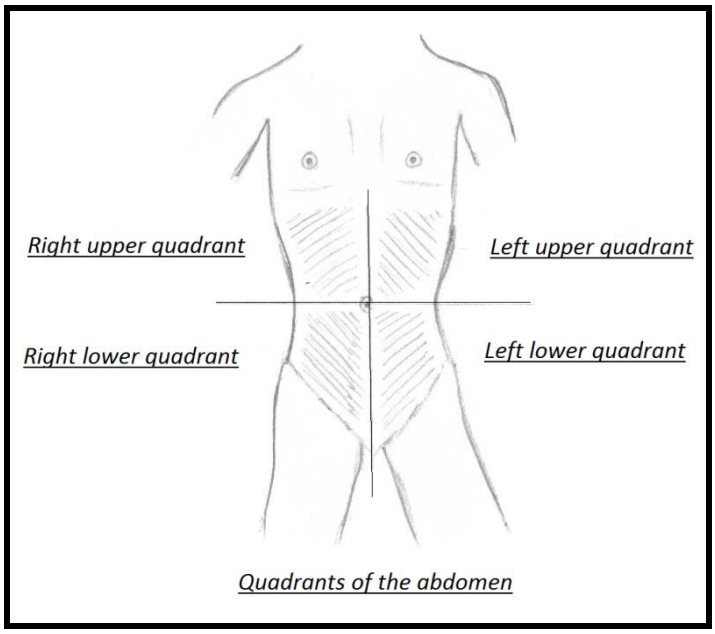
- In new born baby abdomen is symmetric, slightly rounded and moves simultaneously with the chest during respiration
- In older infants, abdomen is more prominent
- In babies, infants and small children, respiratory movements are mostly abdominal

- In older children, adolescents and adults, respiration is mainly thoracic in character
- Absence of abdominal movements may be due to peritonitis
- Paradoxical movements i.e., recession with inspiration on the paralysed side of abdomen can be seen with phrenic nerve palsy.
- Scaphoid abdomen (depressed in the centre or flat) is normal in older children but indicates a diaphragmatic hernia in the newborns
- Abdominal distension may be due to:
    1) Intraperitoneal air or fluid i.e. ascites, blood, lymph, pus, tumours (Wilm's), distended bladder or organomegaly.
    2) Flatus (gas), faeces (constipation), intestinal obstruction, ileus or Hurschsprung's disease
    3) Hypothyroidism and hypotonic musculature i.e., rickets, Downs syndrome etc.
    4) Enlarged abdominal organs or any other masses usually give rise to a localised swelling.
    5) Oedema is noted in nephrotic syndrome etc.
- Prominent veins: Dilated superficial veins also called as caput medusae may be present in the following conditions.
    1) **Portal hypertension:** obstruction in the portal circulation. Blood will be shunted in to the para umbilical veins, umbilical veins, superior and inferior umbilical veins – so these veins will become prominent called as "caput medusae". The flow in these veins is away from the umbilicus to the surroundings.
    2) **Obstruction to the inferior vena cava:** there is venous anastomosis of inferior epigastric vein tributary of femoral vein with the axillary vein. Due to the obstruction of inferior vena cava the connecting veins of the two will become prominent in the antero-lateral part of the trunk – blood flow is from below upward
- Scars of previous surgery or biopsies: stomas
- Pinkish stria may be seen in Cushing's syndrome
- Peristaltic waves may at times be seen in preterm babies but in term babies if present may indicate pyloric stenosis or obstruction to ileocaecal valve
- If secondary hair absent, consider hypopituitarism or hypogonadism
- Examine hernial orifices and look for hernias like: epigastric, inguinal, umbilical, femoral and assess if it's reducible or irreducible hernia
- Suprapubic bulge may be due to full urinary bladder
- Examine the genitalia
    - Males: look for Disorder of Sexual Differentiation (DSD), micropenis, hypospadias, undescended testes or urine voiding stream
    - Females: **Clitoris:** shape and size. Is it unusually enlarged, consider masculinisation, **Labia:** fused, check for DSD, **Vagina:** check for discharge
    - Anus: normal, patent or patulous

2. PALPATION

Child should lie flat on the bed without pillow and should be relaxed if possible. Lower limbs should be flexed at the hips and the knees. Now you should stand on the right hand side of

the patient and rub your hands to warm them up. Start palpation with your right hand; first superficial then deep palpation.



Quadrants of the abdomen

Superficial palpation

First do light supperficial palpation. If ther e is a possible tender or painful area, this should be palpated last. Child need to be made comfortable either by giving a soft toy or asking the mom to stand next to you at the upper end, holding the childs hand. Even if the child is crying, his abdomen can be examined by dipping your fingders during inspirtaion.

In calm and co-operative children start examination from left iliac fossa, moving in anticlockwise direction to find out any tenderness, rigidity or mass.

- Guarding: it is voluntary protective contraction of abdominal wall muscles
- Rigidity: it is involuntary, check if its generalised or localised

Young children cannot precisely indicate painful area and often just point to the umbilicus. The facial impression of the child often helps to determine the degree of tenderness. Babies seldom manifest abdominal rigidity even in the presence of peritonitis.

In case there is tenderness it should be graded as follows:

- Grade 1: when patient complains there is pain on touching
- Grade 2: when patient shows facial expressions for feeling pain
- Grade 3: when patient moves away his part being touched
- Grade 4: when patient does not allow the examiner to touch the affected part

In case there is inflammatory process in abdomen, there may be rebound tenderness (appendicitis) or rigidity (peritonitis). Rebound tenderness is a painful procedure with limited validity in young children.

Other signs of acute abdomen:

Rovsing's sign: is elicited by palpating the left lower quadrant of a patient's abdomen. If it increases the pain felt in the right quadrant, the patients is said to have a Rovsing's sign and may have appendicitis.

Psoas sign: is elicited by asking the patient to lie down on his or her left side while the right thigh if flexed backward. Pain may indicate an in inflamed appendix overlying the psoas muscle.

Obturator sign: is elicited by asking the patient to lie down supine. Now flex the right hip and knee at 90 degrees and rotate it internally and externally. Pain felt by patient may indicate inflamed appendix.

Murphy's sign: is elicited by asking the patient to take in and hold a deep breath while palpating the right subcostal area. If pain occurs on inspiration, when inflamed gallbladder comes into contact with examiner's hand, Murphy's sign is positive. Discomfort is more likely to be nonspecific in paediatric cholecystitis and patients of this age often present with irritability, jaundice and acholic stools.

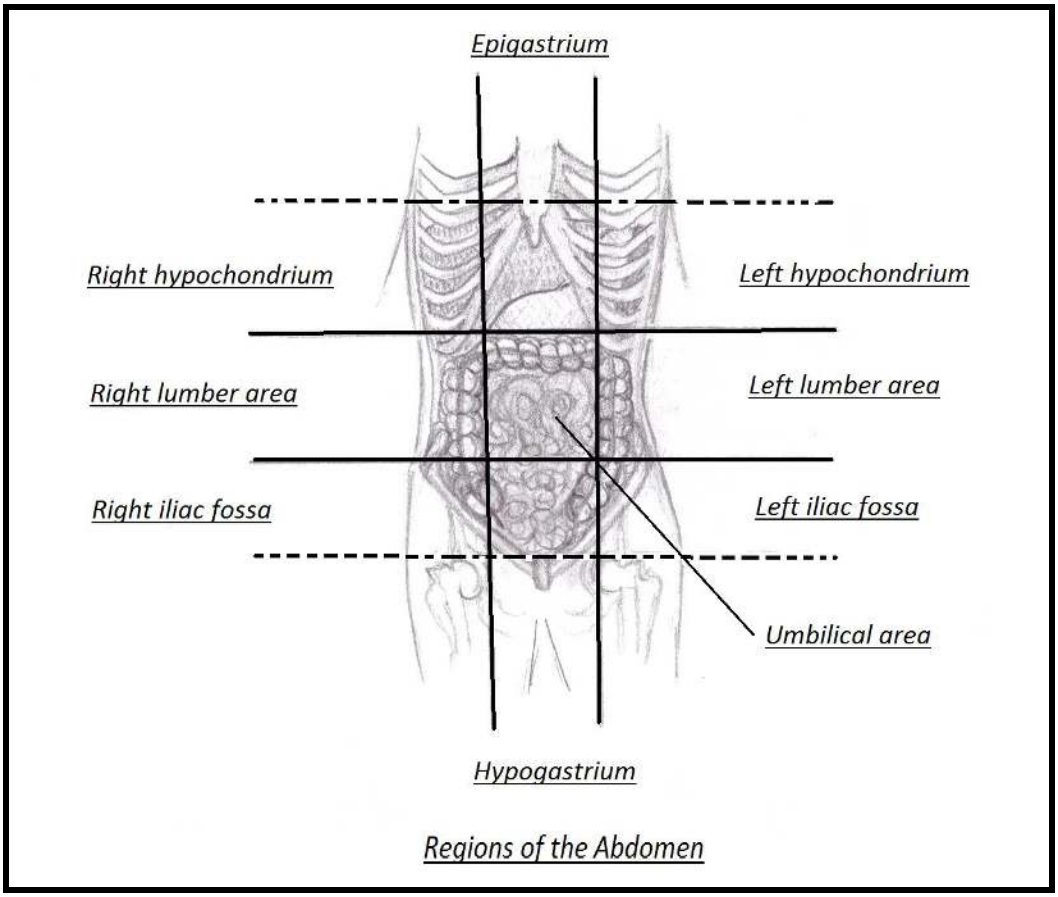
Deep palpation

Deep palpation is done by placing the whole right hand on the abdominal wall in a way that whole hand should touch the abdomen in a relaxed form. Keeping the fingers straight and flexing them slightly at the metacarpophalangeal joints. The abdomen is pressed firmly but gently. The forearm should be in horizontal position in line with the wrist joint. Following scheme can be followed while palpation is done:

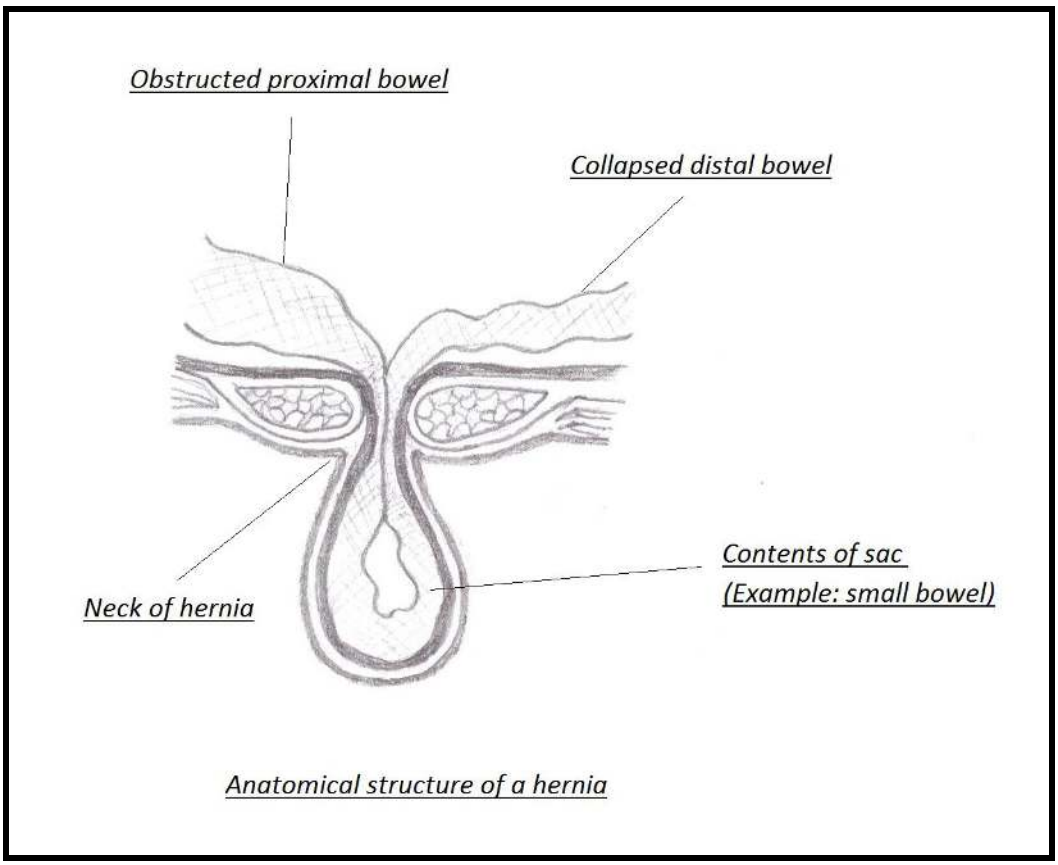
1) Left iliac fossa
2) Left lumber area (for left kidney)
3) Left hypochondrium (for spleen)
4) Right lumber area (for right kidney)
5) Right hypochondrium (for liver & gall bladder) and epigastrium
6) Right iliac fossa and hypochondrium (for urinary bladder and uterus in females)
7) Umbilical area for aortic, para-aortic, and mesentric lymph nodes
8) Palpate both groin areas for lymph nodes and femoral arteries
9) Palpate the hernial orfices: size, contents, reducibility, tenderness etc.
10) Palpate the external genitalia (see next section p 131).
11) Do rectal examination (see the next section p 130)

If mass is palpable, note the following points:

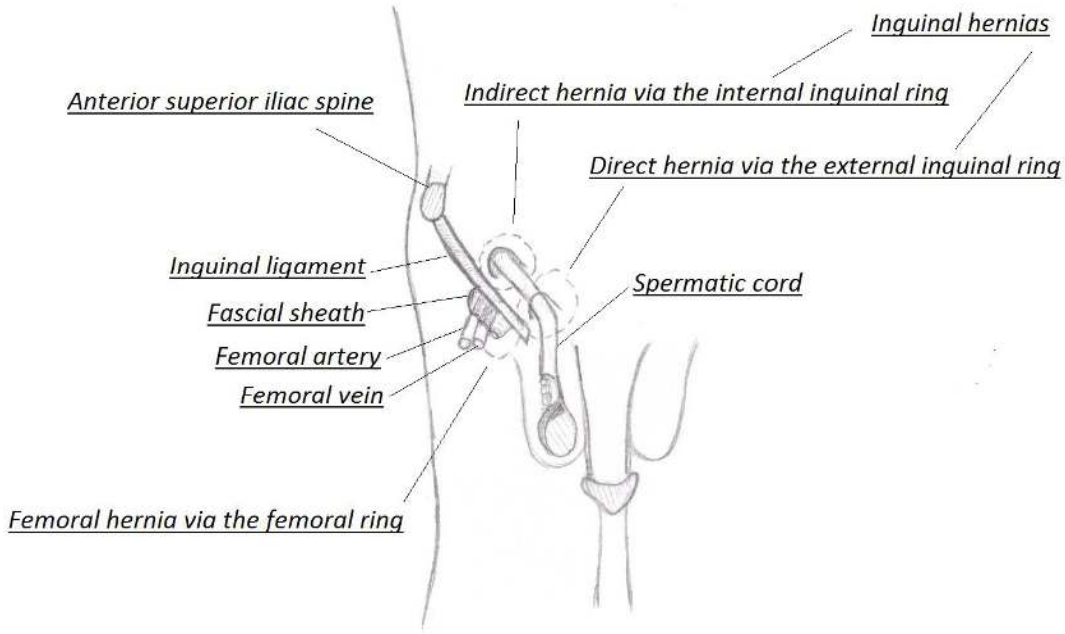
1) Area in which mass is palpable
2) Number of masses
3) Size, shape, consistency, pulsatile, tenderness
4) Mobility during respiration and examination
5) Whether the mass is intra or extra abdominal



Regions of the Abdomen



Anatomical structure of a hernia



Antomy of the inguinal and femoral hernias

METHOD OF PALPATION OF SPLEEN

Spleen is a flat disc which lies in the left upper part of the abdomen between 9th, 10th and 11th ribs. Patient should lie supine with legs flexed and abdomen exposed as stated above. For palpation of spleen with right hand, start palpating from right iliac fossa and go up obliquely to the left hypochondrium as spleen usually enlarges in this direction – infero-medially. In infants, spleen enlarges inferiorly so palpate from left iliac fossa to left hypochondrium.

Older children can be asked to take a deep inspiration. With this, the diaphragm moves downwards so the spleen is pushed down. In order to become palpable, spleen has to be enlarged by 2-3 times of its normal size.

While palpating the spleen in older children, it is sometimes helpful to let them turn slightly on the right side, with legs a little flexed. By putting your left hand in the left loin of the patient and pressing the spleen forward can be helpful at times.

When spleen is palpable describe its:

1) Size: take longest measurement. Spleen usually enlarges downward and to the right but can enlarge in any direction – measure it from the costal margin to the longest direction
2) Tenderness
3) Texture
4) Notch

Common causes of splenomegaly

1) **Cardiac:** Subacute bacterial endocarditis**,** constrictive pericarditis, CCF (Rt)
2) **Connective tissue disease:** Systemic onset juvenile chronic arthritis , serum sickness
3) **Haematological:** Polycythemia, Sickle cell disease, hereditary spherocytosis**,** G6PD deficiency,thalassaemias, haemolytic anaemia, purpura
4) **Infections/infestations:** Infectious mononeucliosis, sub acute bacterial endocarditis, malaria, CMV, kalazar, hydated cyst, typhoid fever
5) **Malignancy:** Leukaemia (myeloid), Hodgkins disease
6) **Portal hypertension:** Post UV line, Budd-Chiari sydrome
7) **Storage diseases:** Gaucher's disease
8) **Liver cirrhosis**

METHOD OF PALPATION OF LIVER

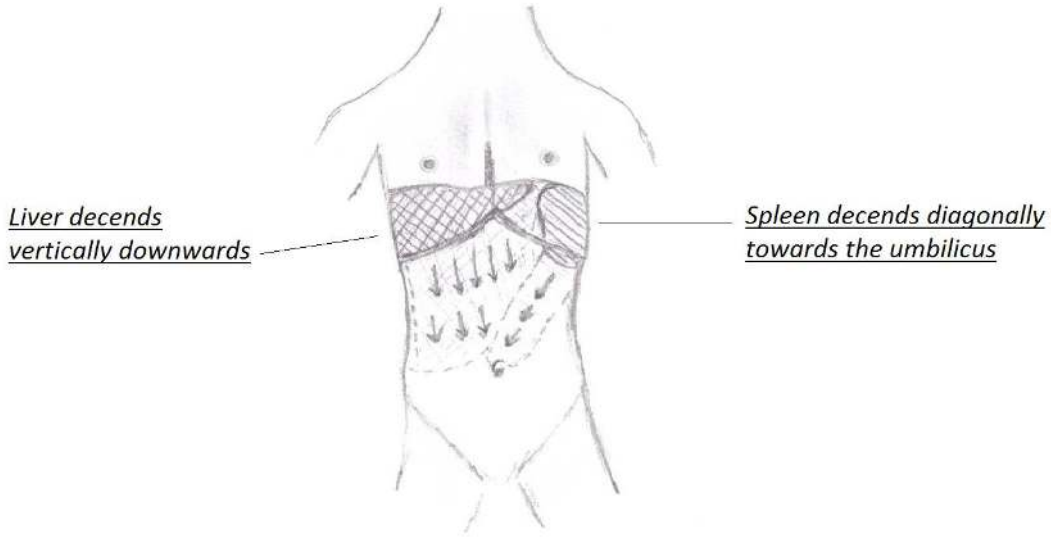
Liver lies in the right hypochondrium. Its upper border lies approximately in the 4^{th} intercostal space or behind the 5^{th} rib in mid clavicular line. Patient lies flat on the bed (pillow removed) with knees flexed. You stand on right hand side. Place your right hand over the right iliac fossa to the outer side of rectus muscle with index finger's edge towards the costal margin. Now palpate the liver by going vertically upward. If liver is palpable, note the following points.

- Feel of edge: is it smooth, sharp, rounded, irregular or thickenend
- Texture: check if it is soft, firm or hard:
    - **Soft** – non pathological
    - **Firm** – pathological
    - **Hard** – more pathological
- Surface of liver: whether smooth or nodular
- Size: measure in cm below costal margin in the midclavicular line
    - Upto 3cm Hepa is normally palpable till 5 yrs of age and 1-2cm till 10 yrs. So if its 1-2cm palpable, say its 1-2cm palpable hepa and not to say hepatomegally.
    - Percuss the upper border, it should be in 4^{th} to 5^{th} ICS
    - Mark the upper border and measure the liver span
- Tenderness: common in liver congestion (CCF) or infective hepatitis
- Pulsation: this is felt by pressing in the right hypochondrium with right hand, while supporting the right lower chest posteriorly by left hand. Pulsatile liver can be seen in tricuspid incompitance.
- Look for the presence of bruit
- Look for sub hepatic masses

Common causes of hepatomagaly

1) **Structural:** extrahepatic billiary atresia, choledochal cyst
2) **Storage and metabolic:** glycogen storage diseases, sever malnutrition
3) **Haematological:** Leukaemia, thalassaemia, sickle cell disease
4) **Heart:** Congestive cardiac failure, Constrictive pericarditis

5) **Infections/parasites:** TORCH infections, neonatal septicaemia, malaria, tuberculosis
6) **Inflamatory:** Chronic active hepatitis, chronic perisistant hepatitis
7) **Reticuloendothelial:** Non-Hodgkin's lymphoma, Hodgkin's disease
8) **Rheumatological:** Systemic onset juvenile idiopathic arthritis, Systemic Lupus Erythematosis
9) **Tumours:** Neuroblastoma/Willim's tumour



Direction of enlargement - Liver and Spleen

METHOD OF PALPATING THE KIDNEYS

Kidneys lie in the lumbar region. The left being 2 inches higher than the right kidney. Both kidneys are palpated bimanually. In newborn and premature babies, they are usually palpable, but as a rule not so in older children.

The patient lies on his or her back without pillow with knees flexed. Place the left hand in the lumber region posteriorly and the right hand anteriorly on respective sides.

First palpate the left kidney and then right. The left hand presses the loin forwards while the right hand pushes the anterior abdominal wall backwards, upwards and inwards. Older children can be asked to take a deep breath and relax abdominal muscles while trying to palpate the kidney between the two hands. NB! Kidneys don't move with respiration.

If kidney enlarged it will be felt between the two hands. In very lean lower pole of right kidney may be felt normally.

Common causes of renal enlargement

1) Hydronephrosis
2) Multicystic kidney (unilateral)
3) Posterior uretheral valve
4) Perinephric abscess

5) Obstructive uropathy
6) Polycystic kidney disase
7) Urolethiasis
8) Wilm's tumour
9) Neuroblastoma
10) Hydronephrosis
11) Renal cysts
12) Renal vein thrombosis
13) Tuberous sclerosis

Differentiating between enlarged left kidney and enlarged spleen

Enlarged kidney	Enlarged spleen
Bean shaped with round edge	Flat disc with sharp edge
Notch is not present	Nocth is present
Kidney enlarges downwards only	Spleen enlarges downward and medially
Moves very little if at all with respiration	Moves down with respiration
Bimanually palpable	Cab be palpable with one hand
Can get above the mass/margin	Cannot get above the mass
Resonant note is present in front of the kidney	Dull note is present in front of spleen

Picture: Examination of Organomegaly - Liver, Spleen, Kidney respectively

PALPATION OF ABDOMINAL LYMPH NODES AND TUMOURS

Method of palpating aortic and para-aortic lymph nodes

In majority of children, aorta can be felt readily. For its palpation finger tips of both hands (in a line from above downward) are pressed deeply in the abdomen at a site which is little above on the left side of umbilicus. After feeling the aortic pulsation, the fingers are displaced to the right so to have an idea about the width of aorta.

When enlarged, aortic and para-aortic lymph nodes can be palpated in the umbilical and epigastric regions, along the left aortic wall as firm and rounded masses. They may be separate or matted together. In most of the cases they are fixed to the posterior abdominal wall.

Mesenteric lymph nodes can be palpated along the medial 2/3rd of a line which joins right iliac spine to the umbilicus and extends about 2-3 cm to the left.

Common causes of abdominal lymph node enlargement

1) Abdominal TB
2) Bacterial infections: E-Coli, Yersinia and Staph species
3) Giardia lamblia and non Slamonella typhoid
4) Viruses like Adeno, Coxsackie, Rubiola, EBV, HIV and Catchscratch disease
5) Lymphoma

Following palpable lymph nodes of any size may indicate significant pathology

1) Femoral
2) Mediastinal
3) Abdominal
4) Epitrochlear
5) Supraclavicular

Hepatospleenomegaly with lymphadenopathy in HIV positive may indicate presence of Kaposi's sarcoma even if there are no skin lesions

PALPATION OF URINARY BLADDER

Normal urinary bladder cannot be palpated. If there is urinary retention and the patient is unable to empty the bladder, it is felt in the supra pubic area as a symetrical oval shaped swelling, firm in consistancy, with smooth surface and regular margins.

Method of palpation: stand on the right hand side of patient and with left hand horizontley from ziphisternum towards the pubic symphysis. By gentle palpation its upper border (which may be as high as umbilicus) and lateral margins can be felt but not the lower border. Now percuss the distended bladder and see if the dullness is present or absent. Pressure over it makes the patient desire to pass urine.

3. PERCUSSION

Percussion is done to confirm the visceral enlargement, fluid accumulation or gas in the gut. Normal percussion note in abdomen is tympanic (like a drum). If the fluid is present in the peritoneal cavity, the percussion note will be stony dull and fluid thrill will be present. When fluid is comparatively less in amount, shifting dullness may be present.

The ability to demonstrate ascites is usually assessed by examiners in clinical exams therefore students should acquire this skill correctly. You should expect gross ascites when abdomen is distended, umbilicus is everted, flanks are full, skin looks oedematous, pressure marks on the skin are obvious and vulva or scrotum are full. Presence of following three signs usually confirms presence of ascites.

Fluid thrill

A fluid thrill is an unreliable sign and can easily be elicited as false positive in very obese children. To find out whether fluid thrill is present or not, patient should lie flat on the bed with knees flexed (to relax abdominal muscles). Two hands method is used i.e. palmer surface of one hand is placed on the flank which is away from examiner and with the finger of the other hand flick the proximal flank. A sensation will be felt by the palpating hand. Thrill can also be conducted through the fat along the skin surface so to rule this out, another person is asked to place the ulnar side of his hand in the middle of the abdomen vertically and same procedure is repeated. If thrill is felt this is due to the fluid, as conduction by fat is checked by the third hand.

Shifting dullness

This is more reliable sign than fluid thrill. This is done to detect a small amount of fluid which is not enough to produce fluid thrill. The child is instructed to lie flat on his/her back and percussion is done from the centre of the abdomen toward the flank till the percussion note becomes dull. Keep the finger there at the point of dullness and ask the child to turn on the other side for 30 seconds and then percuss again. Now the percussion note will be resonant as under the effect of gravity the fluid has moved to the lower flank. Positive shifting dullness indicates free fluid in the peritoneal cavity. If it remains dull, there is no shifting dullness.

Distribution of ascites dullness may be noted as horseshoe shaped especially if small amount of fluid is present. In this case child is asked to stand dullness can be felt in the flanks. Free mobile fluid in the abdomen can be felt in children which is called as ‘jelly belly’.

Puddle sign

This can be elicited in older and co-operative children, which may indicate presence of as little fluid as 120 ml. The child is instructed to lie down on his/her abdomen for few minutes and then is asked to be on the hands and knees. Chest piece of stethoscope is applied over the lower part of the suspended abdomen. Now flick the proximal flank repeatedly with the finger and listen with stethoscope. The sound will be dull and soft. As you move the stethoscope away from the examiner toward the opposite flank, the sound will get louder.

For further confirmation place the chest piece of stethoscope back to the lower most suspended part of abdomen, flick the finger in the flank and keeping the stethoscope there, ask the patient to sit up. Flicking is done again. Now the sound will be louder and clearer as compared to the first.

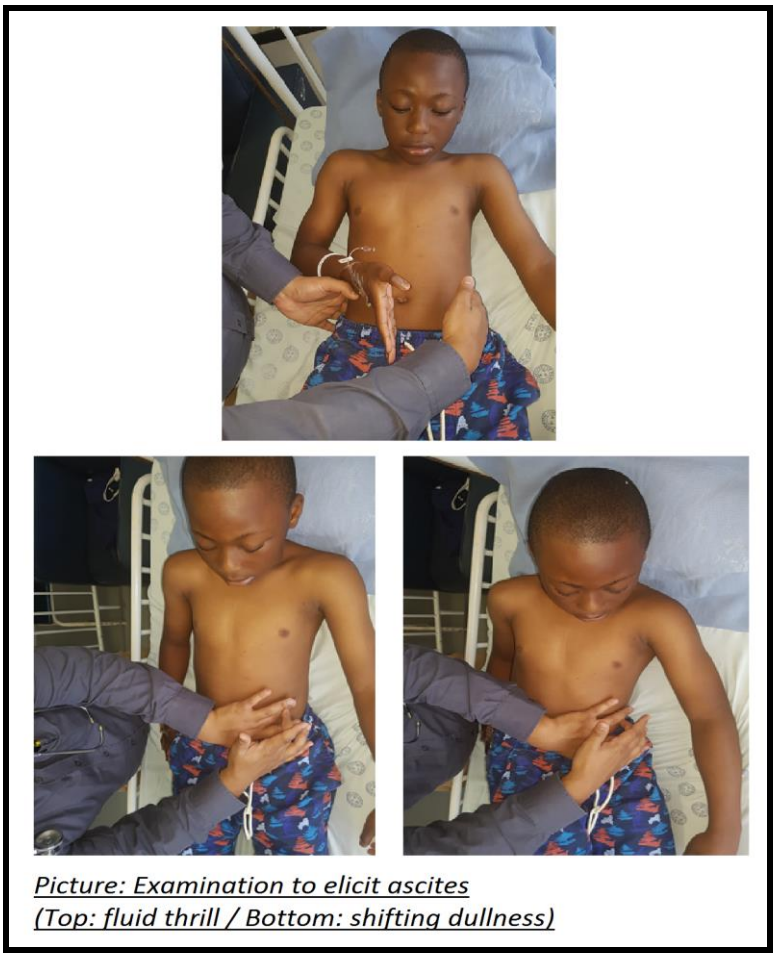
Now make your assessment:

1) Is abdomen resonant or dull
2) Fluid thrill present of absent
3) Shifting dullness present or absent
4) Dullness over distended urinary bladder, present or absent

If signs of ascites are present or the abdomen is distended, do horizontal abdominal measurement at the level of the umbilicus and below it. Circumference of abdomen is taken by measuring tape and is recorded. This can be used for prognostic purpose.

Ascitic fluid in newborn may be:

- A transudate as in hydrops and heart failure
- An exudate in peritonitis
- Biliary – rupture of common bile duct
- Chylous – rupture of lymphatic duct



Picture: Examination to elicit ascites
(Top: fluid thrill / Bottom: shifting dullness)

Common causes of ascities

1) **Hepatic:** Cirrhosis, portal hypertension
2) **Renal:** Nephrotic syndrome
3) **Gastro-intestinal**
   - Protein loosing enteropathy
   - Caeliac disease
   - Inflammatory bowel disease
   - Neutritional: Beri Beri , PEM
4) **Lymphatic:** Acquired chylous ascities (thoracic duct obstruction)
5) **Infection:** Chronic tuberculous peritonitis
6) **Cardiovascular**
   - Right ventricular failure

- Constrictive pericarditis
- Inferior vena cava obstruction
- Hepatic vein obstruction (Budd-Chiari syndrome)

4. AUSCULTATION

Place the chest piece over the abdominal wall and auscultate for peristaltic sounds, called borborygmi. The sounds may increase in case of intestinal obstruction and intestinal hurry i.e. diarrhoea or may be absent in later stages. If the sound is absent even after auscultation for 2-3 minutes at various points, this may indicate paralytic ileus (silent abdomen).

Also listen for bruits

- over the liver – hepatoma
- over the aorta – aneurysm
- over the kidneys – renal artery stenosis (bruit is present in cases of incomplete stenosis but no bruit if complete stenosis)

Picture: Auscaltation for a bruit

Succussion splash

Make the patient to lie down supine on the bed. Now place hands on both sides of the abdomen and shake it/move vigorously horizontally. Splash usually is present in stomach but sound may indicate presence of intestinal obstruction.

Groin

Examine the groins and genitalia for inguinal hernia, strangulated hernia, undescended testes, sexual abuse etc.

Anus and rectum

Examine the anus and if possible, stool in the nappy. Imperforate anus is usually missed. Note volume, colour and consistency of stools as well as the presence of blood – bright red, dark red or black – each one has a particular significance. Stool mcs can be sent for bilharzia, amoebiasis or worm infestation.

Rectal examination is usually not done as a routine in children but in case of acute abdomen, chronic constipation and rectal bleeding. Ask permission from parents and older children and explain the procedure and the reason why you are doing it. Ask the mothers or the nurse to be present during this procedure.

Ask the child to lie down on his left lateral position with left leg straight and right flexed. Use the little finger in small children and index finger for older children and lubricate properly with K-Y Jelly. Relax the child as best as you can and approach the rectum from behind by inspecting the perianal area.

When finger is inserted assess for anal tone. A tight anus resisting your finger is suggestive of anal stenosis. A loose patulous anus usually is usually present in myelomeningocele or other lower spinal lesions.

Common abnormalities found are:

1) Tenderness in retrocaecal appendix
2) Rectal prolapse and rectal polyps
3) Fissure-in-ano at 6 and 12 O' clock or sentinel tags
4) Rectal foreign bodies
5) Roundworms and tapeworms (usually noted by mothers)
6) Condyloma acuminata or genital warts – exclude sexual abuse
7) Constipation with overflow incontinance
8) Explosive release of flatus may indicate Hirschprung's disease

Back

Evaluate the back as well as the dorsal, lumber and saceral areas to rule out intraspinal abnormalities like:

- Myelomeningocele
- Pilonidal tract or sinus
- Hair tufts
- Dimples

Anal wink: Also called as anal reflex, perineal reflex or anocutaneous reflex and is usually done in infants with spina bifida. In order to elicit apply a tactile stimulus around the anus and note a wink contraction of anal sphincter muscles and also flexion. This stimulus is detected by pudendal nerve and response is integrated by the spinal cord sacral segments S2-S4. Absence of this reflex indicates interruption of reflux arc or damage to the lowest sacral segments of the spinal cord.

Genitalia

Familiarise yourself with normal genital appearance and age appropriate size and shape of its parts so that when you examine the child you can differentiate between normal and abnormal genitalia in male and female infants, toddlers and school children. Keep in mind the disorders of sexual differentiation.

Inspection and examination of perineum in girls and penis and testes in boys is part of routine examination but should always be performed in the presence of mother or a nurse.

Female genitalia

Vaginal palpation is not usually performed unless it is clinically indicated for example foreign body, suspected sexual abuse or vaginal discharge.

In premature baby girl's clitoris and labia minora is usually prominent, dark coloured, and swollen soon after birth. It is sometimes fused by a transparent membrane. The clitoris may also appear prominent. If it is abnormally large, virilisation should be considered.

During first week after birth, a vaginal discharge often occurs due to transplacental maternal hormones. The colour could be white, grey or blood stained.

A discharge is abnormal in older children and could be due to bacterial infection, irritating clothing, lack of hygiene, bubble baths, threadworms and foreign body. In children with diabetes mellitus, candidiasis may be a common cause of vaginal discharge.

In some children, hymen is imperforated and there may be accumulation of secretions causing development of hydrocolpos. At puberty haematocolpos may develop. Around the genitalia, also note presence of blisters, or ulceration due to herpes, condylomata due to syphilis tumours like sarcoma botryoides.

Epispadias may also occur in females. There is midline split in the clitoris and mons.
If baby urinates during examination take note of the volume and stream. A weak dribbling stream is abnormal. Uterus and ovaries are not usually palpable in infants and children.

Male genitalia

Examine the size of penis. Enlarged penis may be seen in certain endocrine and neurological conditions. For example, in congenital adrenal hyperplasia the penis is large but the testicular volume is normal. The explanation of usually reported small penis is a normal penis buried in fat. True micropenis is rare.

In small children, prepuce is usually adherent to the glans. Sometime you may see small harmless white spots on distal prepuce which usually disappear in few days. Also, some children have small foreskin opening which does not obstruct the urine flow. We don't need to separate the foreskin from the glans as it occurs naturally with time.

On inspection of scrotum, a normal rugosity and testes should be visible.

Enlargement of scrotum can be seen due to:

1) Enlarged testes
2) A hydrocele
3) An inguinal hernia

If there is any apparent abnormality in the size of testes, an orchidometer can be used to measure the testicular volume. The examination of testes is done in three positions:

- Standing position: toddlers and small children

- Lying flat on the couch
- Squatting position

Following are the common abnormalities seen in children:

Epispadias: the meatus is situated dorsally on the penile shaft or the glans.

Hypospadias: the penis is abnormally shaped and the meatus is on the ventral side accompanies by ventral curving called as chordee.

Undescended testes: examine the scrotum cautiously and you may find one or both testes missing from the scrotum. In order to confirm the presence of testes, put a slight pressure on the superficial inguinal ring and feel the testes in the scrotum.

In normal full-term neonate the testes are usually hiding in the inguinal canal, but can be brought into scrotum. In preterm babies the testes are in the inguinal canals and cannot be moved into scrotum soon after birth.

Torsion of testes: suspect if the testes are swollen, tender and painful. This is an emergency situation since necrosis of testes may ensue. This needs to be differentiated from orchitis and epididymitis, as both have the similar features. Check for cremasteric reflex which is absent in case of torsion but present in epididymitis. Pain relief with lifting the affected testicle points towards epididymitis also called as Prehn's sign.

Inguinal hernia: look for the bulge in the groin. This may only become visible when the child is crying, coughing, or staining during a bowel movement or it may appear larger during these times.

Hydrocele: a soft and non-tender swelling where the testes cannot usually be felt. Presence of fluid is demonstrated by trans-illumination test.

TUTORIAL 10

THE EXAMINATION OF CENTRAL NERVOUS SYSTEM

OVERALL OBJECTIVES

At the end of this module, students should to be able to:

- **Recognise** by appropriate history and examination:
  - The anatomical site of a neurological lesion i.e. to distinguish between upper and lower motor neuron lesion
  - The general pathology of the lesion i.e. to distinguish between vascular, inflammatory, metabolic, neoplastic or degenerative lesions
- **Investigate** appropriately and manage common and life threatening neurological problems in infants and children
- **Describe common indications** for lumber puncture, EEG, cranial ultrasonography, CT and MRI scans

Extra General Features for CNS examination

- Dysmorphology
- Neurocutaneous lesions – café au lait spots etc.
- Meningomyelocele, gibbus and kyphoscoliosis
- Head and abdomen for VP shunt scars
- Hypertrophied calf muscles, wasting, fasciculations and trophic signs

PARTS OF CENTRAL NERVOUS SYSTEM

Cerebral cortex: cortical functions

Frontal lobe (precentral gyrus is motor strip)

- Controls
  - Voluntary movements
  - Personality
  - Social behaviour
- Labile mood → possible frontal lobe involvement

Temporal lobe

- Responsible for memory and perception of smell
- Contains lower fibers of optic radiations

Parietal lobe (post central gyrus is sensory strip)

- Responsible for conscious sensations
- Contains upper fibers of optic radiations

Occipital lobe

- Responsible for analysis of visual information

Broca's area

- Responsible for speech production and articulation

Wernick's area

- Responsible for language comprehension

Motor system

Upper motor neuron (UMN)

1) UMNs pass down from cortex to corona radiata, internal capsule, midbrain, pons, medulla oblongata and spinal cord
2) Axons of UMN arborize around the LMN in the anterior horn
3) Axons of UMN while passing through the internal capsule occupy the genu and anterior 2/3rd of posterior limb
4) In midbrain these fibers occupy the middle 3/5th of the peduncles
5) In pons these fibers are split by the nuclei of the pons and pontine fibers
6) In medulla, these fibers are condensed anteriorly to form a bulge called pyramid
7) Majority of these fibers (90%) docusate in the lower part of medulla (and some uncrossed fibers) forming lateral corticospinal tract
8) While the rest (10%) pass uncrossed to the same side forming anterior corticospinal tract

Lower motor neurons (LMN) consist of:

1) Anterior horn cells & homologous cell in the brain stem
2) Fibers passing through anterior spinal nerve roots and peripheral nerves to muscles
3) Motor nuclei of cranial nerves

Extra-pyramidal system

This includes all the higher centres in the Nervous System

1) Basal ganglia
2) Substantia nigra
3) Red nucleus
4) Sub-thalamic nuclei
5) Reticular nuclei
6) Olivary nuclei

Five major extrapyramidal tracts

1) Reticulo-spinal tract
2) Rubrospinal tract
3) Olivo-spinal tract
4) Tectospinal tract
5) Vestibulo-spinal tract

All 5 tracts run in the anterior column of spinal cord & end around the anterior horn cells. Their functions include: movements, posture and tone in the voluntary muscles.

Lesions in these fibers produce three clinical features:

1) Change in the muscle tone
2) Involuntary movements
3) Slowing of the movements

SENSORY SYSTEM

There are 3 neurons:

1) **The cyton (cell body) of the first neurons** lies in the root ganglia of spinal cord and cranial nerve. It conveys 2 types of sensations:

   Superficial sensation (from the skin):
   1) Sensation of touch
   2) Temperature
   3) Pain

   Deep sensations include:
   1) Sense of position, movements and vibration
   2) Sense of size and shape of an object
   3) Sensations of muscle and joint movements

   Central axon of the first neurone arborises around the cells situated at the base of grey horn of the spinal cord. The fibers which convey deep sensations go up in posterior white column and end in the nucleus gracilis and cuneatus. From these nuclei second axons arise and cross to the opposite side.

2) **Cyton of the second neuron** lies in the nucleus gracilis and nucleus cuneatus in the medulla. They cross over to the opposite side and form two tracts:
   - Anterior spino-thalmic tract – crude touch and pressure signals
   - Lateral spino-thalmic tract – pain and temperature signals.

   Spinothalmic fibers go upward through the spinal cord, the brain stem and finally into thalamus

3) **Cyton of third neuron** lies in the thalamus. From here fibers arise and go to the sensory cortex (by way of the internal capsule) lying in the parietal lobes behind the fissures of Rolando. Sensory tracts passing through the brain stem receive fibers from sensory nucleus of trigeminal nerve.

SPINAL CORD

- Extends from foramen magnum down till upper border of the 2nd lumber vertebra
- Membranes of spinal cord end in the second sacral vertebra.
- Spinal cord terminates at L1 in adults and L3 in infants and children
- In upper cervical regions, spinal nerve roots are short and horizontal but lumber and sacral roots form vertical leash of nerves called as cauda equina
- There are 7 cervical vertebrae but 8 cervical roots
- There are 4 coccygeal vertebrae but one coccygeal spinal root
- Physiologically spinal cord is composed of supper-imposed segments:

Segments:	C 8	T 12	L 5	S 5	C 1-2 (coccygeal) = 31
Vertebrae:	C7	T 12	L 5	S 5	C4 (Three fused) = 30

Summary of Neurologic examination of a child

1) Mental state assessment: Assesses various zones of cerebral cortex
2) Cranial nerves assessment: Evaluate integrity of brain stem
3) Motor examination: Evaluates upper and lower motor neuron function
4) Sensory examination: Assesses peripheral sensory receptors & their central reflections
5) Deep tendon reflexes: Assess upper and lower motor neuron connections
6) Gait assessment: Puts motor system into a dynamic state for functional assessment.

CLINICAL EXAMINATION

Here the process differs from the other systems:

- First just look at the child for abnormal posture and movements
- Let the child stand, walk, sit & then get up from the floor, this will give you important information

1. LEVEL OF CONSCIOUSNESS

Consciousness has two components

- **State of consciousness:** *how awake the child is* – this determines the integrity of ascending reticular activating system
- **Content of consciousness:** *how aware the child is* – this determines integrity of cerebral cortex, thalamus and their connections

Mental state evaluation: consists of alertness and intellectual abilities

Alertness is assessed:

In infants by

- Observing spontaneous activities
- Feeding behaviour
- Fixing and following
- Response to tactile, visual and auditory stimuli

In older children

Orientation to time, place, space and person

Intellectual abilities are assessed by language skills

Receptive (*Wernicke) aphasia:* It is the inability to understand language i.e., fluent speech but nonsensical. For example, Tina had a stroke which involved Wernicke's area. She has difficulty understanding when spoken to.

Expressive (*Broca) aphasia:* It is the partial loss of the ability to produce language (spoken, manual or written) although comprehension generally remains intact. For example, Nandi had a stroke which involved Broca's area. She knows what she wants to say but has difficulty expressing herself. This should not be described as confused.

Receptive and expressive (*Global) aphasia:* It is severe form of non-fluent aphasia caused by damage to the left side of brain, that affects receptive and expressive language skills (needed for both written and oral language) as well as auditory and visual comprehension.

Glasgow coma scale (GCS)

EYE OPENING		VERBAL		MOTOR	
Spontaneous	4	Oriented	5	Obeys	6
Voice	3	Confused	4	Localises pain	5
Pain	2	Inappropriate words	3	Withdaws	4
None	1	Vocalises sounds – no words	2	Flexion	3
		No vocalization	1	Extension	2
				None	1

Severity of head injury:

- GCS <8: Severe
- GCS 9 to 12: Moderate
- GCS >13: Mild injury

AVPU score:

- **A**lert
- Responsive to **V**oice
- Responsive to **P**ain
- **U**nresponsive

P & U scores indicate coma and correspond to GCS of 8 or less

Signs of upper and lower motor neuron lesions

UMNL	LMNL
• Paralysis affects group of muscles	• Individual muscles are paralysed
• Muscle tone increases	• Muscle tone decreases
• Muscle wasting absent	• Muscle wasting present
• Babinski sign positive	• Babinski sign negative
• Deep reflexes exagerated	• Deep reflexes diminished
• Clonus present	• Clonus absent
• Trophic changes absent	• Trophic changes present
• Reaction of degeneration is not present	• Reaction of degeneration is present
• UMNL appears as stiffness	• LMNL is detected by weakness
• Involuntary movements (IM) absent	• IM present: fasciculations
Muscle fasciculations indicate denervation from disease of anterior horn cells or peripheral nerve	

Bulbar palsy

- Bulbar palsy is LMNL
- Bulbar refers to the lower brain stem (medulla oblongata) which is the control centre for cranial nerves 9 to 12
- Cranial nerves 9, 10 & 12 are involved in bulbar palsy; palsy means weakness

- If muscles supplied by these cranial nerves are weak then it referrs to as bulbar palsy
- There is no emotional lability.

Pseudobulbar palsy

- Pseudobulbar palsy is UMNL which affects the corticobulbar fibers
- Corticobulbar fibers are the neurons which connect cerebral cortex to cranial nerve nuclei in the medulla oblongata
- In pseudobulbar palsy these corticobulbar fibers get disrupted causing mess up of voluntary control but there is no intrinsic damage to the cranial nerves or brain stem
- Examples are
    - Stroke involving both hemispheres of brain
    - Degenerative disorders

2. SIGNS OF MENINGEAL IRRITATION

Neck stiffness: There is an increased resistance to passive flexion of neck due to inflammation of meninges. Patient feels discomfort or pain when trying to turn, move or flex the neck.

Kerning's sign: When patient is in supine position with hips flexed at 90 degree, note pain and inability to fully extend the knees.

Brudzinski's sign: When patient is in supine position, do flexion of neck. There will be involuntary flexion at the knees and hips.

Jolt sign: Tell the patient to shake his head. Patient will experience severe headache or will not be able to shake his head due to inflamed meninges.

Picture: Examintion for signs of meningeal irritation: Neck stiffness, Brudzinski's sign, Kernig's sign

Meningism is not a sign of meningitis or meningeal irritation. It is neck stiffness due to non-meningitic causes; for example, neck muscle sprain, or soft tissue injury. It can also be due to an acute febrile illness in children which involves triad of nuchal rigidity, photophobia and headache. It therefore requires differentiation from other CNS problems with similar symptoms, including meningitis and some types of intracranial haemorrhage.

Signs of raised intracranial pressure

1) Irritability
2) Headache
3) Vomiting
4) High pitched cry
5) Full or buldging fontanelle in infants
6) Confusion
7) Somnolence
8) Coma
9) Cushing's triad (Bradycardia, Hypertension, Irregular breathing)

Important to note that:

- Raised ICP produces confusion which may mimic global encephalopathy
- Signs of compression of cranial nerve III: ptosis, anisocoria (unequal pupil size)
- Signs of compression of cranial nerve VI: lateral rectus weakness

Causes of raised ICP

CNS causes

1) CNS infections
2) Transverse myelitis
3) Guillain Bare Syndrome (GBS)
4) Tuberous sclerosis
5) Blood in subarachnoid space
6) Neurofibromatosis type 1

Other causes

1) Trauma / haemorrhage
2) Hyperaemia
3) Hydrocephalus
4) Tumors and abscesses
5) Metabolic abrasions
6) Hypoxic ischaemia

3. STANCE

Patient should stand upright with feet closed together and **eyes opened**

- Observe for swaying or lurching
- If present consider cerebellar ataxia

Patient should stand upright with feet closed together and **eyes closed**

- Observe for swaying/lurching or loss of balance.
- If present consider sensory ataxia. This can be due to proprioceptive (somatosensory) deficit due to dorsal column dysfunction and is not primarily a test for cerebellar dysfunction.
- This is also called as positive Romberg sign.

Romberg's Sign:
It is also called as Romberg's Test or Romberg's Manoeuvre which is used in an examination of neurological function for balance. This sign is also used to test for driving under the influence of an intoxicant.

The examination is based on the premise that a person requires at least two of the three following senses to maintain the balance while standing.

- Proprioception (the ability to know one's body position in space)
- Vestibular function (the ability to know one's head position in space)
- Vision (to monitor and adjust for changes in body position)

A patient who has a problem with proprioception can still maintain balance by using vestibular function and vision.

Procedure: patient should stand upright with slightly opened legs and you observe:

- If already ataxic – will get worse on closing eyes
- If not already ataxic – will become slightly ataxic on closing eyes

If there is sensory ataxia (dorsal column lesion), patient will need to use eyes to position feet and stand upright. These patients on closing eyes rely only on dorsal column to keep balance.

Picture: Method to elicit Romberg's sign

4. GAIT

Advise the opatient to:

- Walk normally for 10 meters & turn through 180^{o} – watch width of steps tendancy to veer away
- Toe walk/heel walk: if there is distal weakness, consider peripheral neuropathy

- Heel to toe walk (tendom gait) in straight line: If there is gait instability (broad based unsteady gait) consider gait ataxia that may be due to cerebellar dysfunction
- Lie down and get up quickly. Patient will role over prone position, get on knees and get up this is called as positive Gower's sign seen in proximal muscle weakness
- Stand and walk normally:
  - Scissors like stance and gait →consider bilateral UMNL
  - Hemiplegic gait → consider unilateral UMNL
  - Waddling gait may be due to myopathic proximal muscle weakness

NB! Access how motor & co-ordination systems are functioning:

- A 6 years old child can tendom walk and walk high on toes and heels
- An infant can creep, crawl or cruise or small child can walk or crawl

5. CRANIAL NERVES

Folowing is the list of Cranial Nerves and their place of origion

- 1, 2 out of the brain
- 3, 4 mid brain
- 5, 6, 7 ,8 pons
- 9, 10, 11, 12 medulla

Motor neuclei of Cranial Nerves are supplied by the pyramidal tract of both sides except the two:

- Hypoglossal and
- Part of the 7th cranial nerve which supplies the lower half of the face

EXAMINATION OF CRANIAL NERVES

1. Olfactory

It is difficult to examine this nerve in small children but we can test it in older & co-operative children.

Method: Close one nostril of the patient and bring some vinegar or mint closer to the other nostril and ask the patient to smell:

- If the patient has loss of smell, this is called as Anosmia
- If the patient has perversion of smell this is called as Parosmia
- When a person smells something which is no there, it's called as Phantosmia

Causes of Anosmia

- Severe head injury
- Upper respiratory tract infection
- Meningitis
- Invasion of cancer i.e., ethmoid tumors etc.

Causes of Parosmia

- Severe head injury
- Sinus infection

- Chemical exposure
- Side effects of certain drugs
- Certain conditions of Psychosis (especially elderly)
- Brain tumors

2. Optic nerve

This nerve is examined for: Visual acuity, visual field, colour vision and fundus

Visual acuity: This is the sharpness with which the details and contour of the object are perceived. Test it for near vision and far vision.

- For near vision: ask patient to read a book at a distance for about 10 inches
- For far vision: Use standard Snellen's Chart
- Visual acuity of the newborn is 20/200 and for an infant at 6 months is 20/20
- Best method of finding out if a child can see is to check if the child fixes and follows your smiling face. Babies should rapidly fix a large object and follow it
- The examiners face is the best visual target. In infants, the visual acuity can be assessed by the ability to follow rolling white balls of varying sizes
- From the age of above 3 years, a more accurate estimate of visual acuity can be made using the Sheridan-Gardiner Test.

Procedure:

- Mother holds a card with a number or letter on it
- The examiner then shows the child one of the letters on an identical card and asks the child to point to the same letter on his own card
- When the child has understood the test, the examiner moves to a distance of 6 meters and shows the child a series of letters of descending size until the child fails to make a match
- This test is also useful in assessing visual acuity in patients with whom the examiner has no common language.

Causes of blindness

- Sudden blindness in both eyes
  - Bilateral occipital lobe infarction
  - Bilateral occipital lobe trauma
  - Bilateral optic nerve damage
- Sudden blindness in one eye
  - Retinal artery or vein occlusion
  - Temporal arteritis
  - Optic neuritis
  - Migrain
- Gradual blindness bilateral
  - Cataract
  - Acute glaucoma
  - Diabetic retinopathy

- Bilateral optic nerve/optic chiasma compression
- Vitamin A deficiency

Visual fields

The full extent of this vision is called as the visual field. Forexample: when looking at an object, we not only see that object but also a number of other objects in the neighbourhood, more or less distinctly.

The simplest method of assessning the visual field is comparing the extent of the patient's visual field with your own; testing the field of confruntation. Both eyes must be tested together first and then each must be tested separately.

Confrontation test

- Sit opposite the patient, at a distance of about one meter from him.
- If his right eye is to be tested, ask him to cover his left eye with his hand and look stadily at your left eye.
- Cover your right eye with your right hand and gaze at the patient's right eye.
- Hold up your left hand in a plane midway betweeen the patient's face and your own, at almost a full arm's length to the side. Keep moving the fingers of the hand and bring it near until you can just perceive the movements of the fingers 'with the tail of your eye'.
- Ask the patient whether he/she can see the movements, telling him/her meanwhile to be sure not to take his own eye off yours. If he/she fails to see the fingers, keep bringing them closer untill he/she can see them.
- Test the field in this fasion in every direction; upwards, downwards, to the right and to the left; using the extent of your own visual field for the purpose of comparison.
- Followings may be noted:
  - **Central vision defects**: due to disease of macula
  - **Concentric contraction of the field:** due to optic arophy
  - **Hemianopias:** loss of sight in one half of the visual field
    - **Homonymous hemianopia**: same halves of both the visual fields are affected
      - Cerebral haemorrhage
      - Neurosurgical procedure
    - **Hetronymous hemianopia:** opposite halves of both the visual fields are affected
    - **Bitemporal hemianopia:** outer halves of both (temporal or lateral), the right and the left visual field affected
      - Pituitary adenoma
      - Craniopharyngioma
      - Meningioma

Colour vision

Colour vision is most easily tested by the use of pseudo-isochromatis plates, the best known being those of Ishihara. People with defective colour vison confuse certain colours. These plates are constructed in a way that a person with abnormal vision will read a different number to a normal person on the same plate.

Ophthalmoscopy

Examination of the fundus of the eye, i.e. the retina and its associated structures, with an ophthalmoscope is an essential part of every complete medical examination. Valuable infromation can be obtained about the state of the optic nerve head and of the arteries and veins of the retina, in addition to the detection of local ophthalmic disorders.

Method of doing ophthalmoscopy:

- Check that the ophthalmoscope actually works: check batteries
- When switched on, the emitted light should be:
  - **Bright:** turn it to maximum
  - **White:** ignore all other colours
  - **Circular:** ignore all slits and crosses; turn the dial untill you get a round circle
- Ask the patient to remove his/her glasses but you can keep your or remove
- Switch the room lights off or make them dim
- Explain the procedeure and warn the patient that bright light can hurt a little
- Use 1% tropicamide in each eye and wait for 15 minutes to dilate the pupil
- Sit opposite the patient; confrontation position
- Tell patient to fixate on a precise area; corner of the room or any apecific object.
- Instruct the patient to look at this point no matter what; even if someone come in their way
- This spot should be located so that they are looking slightly away from you when they are examined i.e., to the left when you examine the right eye and to the right when you examin the left eye
- Its better that you examine the patient's left eye with your left eye and right eye with your right eye
- Now place your hand on the patient's forehead so that your fingers are splayed but your thumb is on the upper lid
- Look at the structures from front to back using the following ophthalmoscope settings:
  - Cornea +20
  - Iris +15
  - Lense +12
  - Vitreous +4
  - Fundus 0 (rotate the dial of the ophthalmoscope untill the vessels or the optic nerve is clearly seen.

Followings need to be looked for during Fundoscopy

1. **Red reflex:** media opacities obscure the red reflex like corneal scars, cataract, viterious haemorrhage
2. **Optic disc:** look for optic disc size, colour (pallor or congestion), cup disc ratio. Margins, haemorrhages, new vessels, collaterals. Pale and clearly demarcated disc indicates optic atrophy; yellow grey disc with blurred margins/haemorrhages indicates bilateral papilloedema and new vessels on the disc may indicate diabetic retinopathy etc.

3. **Vessels:** Follow the retinal vessels out from disc to periphery. Look for haemorrhages, excudates, abnormal vessels and pigmented lesions. Also look for microaneurysms, blot haemorrhages, hard exudates, cotton wool spots etc.
4. **Macula:** this is a structure temporal to the disc. To examine the macula, ask the patient to look at your ophthalmoscope's light and note the white foveal reflex in the middle of the macula. A circinate ring of hard excudate, haemorrhage or pigment deposition are the most common findings.
5. The ophthalmoscope can also be used for examining the anterior part of the eye by turning the lens dial to +10.
6. Record any abnormalities in a diagram using the disc diameter as a reference measure
7. Explain the patient that he/she may have blurred vision for 2-3 hrs after dilatation

Following may be noted during ophthalmoscopy

- Optic atrophy
- Papillooedema
- Chorioretinitis
- Retinitis pigmentosa

3. Occulomotor, 4. Trochlear, 6. Abduscens

Look for ptosis:

If ptosis is present, consider 3rd nerve involvement. Then see if the eyes move in all directions. If they do then these nerves are intact. A 6th nerve lesion means that the eye on the affected side cannot look laterally.

Check if the pupils are equal and reacting to light (PEARL)

Size of the pupils depends upon a balance of parasympathetic & sympathetic innervation

- Parasympathetic stimulation will cause pupillary constriction
- Sympathetic stimulation will cause pupillary dilatation

CHECK FOR NORMAL PUPILLARY REFLEXES

Direct light reflex:

- Examine each eye separately with the patient in an indirectly illuminated place
- Ask the patient to look at a distance (to relax his/her accomodation)
- Shine a bright light into the eye to be tested
- The pupil should contract immediately and then dilate again a little and after undergoing a few slight oscillations, settle down to a smaller size.
- When the light is switched off, the pupil should rapidly dilate to its previous diamater

Consensual light reflex:

When the bright light is shone into one eye, both pupils contract; and if the light is shut off from one eye both pupils dilate slightly. The former is the **consensual light reflex**.

Method:

- It is tested by keeping one eye in the shade while shining a bright light into the other one.
- The effect on the pupil of the unilluminated eye is then observed. This is due to the decussation of some of the fibers in the optic nerves at the optic chiasma.

Accommodation reaction:
The pupils become smaller on accomodation for a near object (miosis). Accomodation is only rarely lost in brain stem lesions, but may be impaired with lesions of the oculomotor nerve and in certain neuropathies in which there is autonomic involvement.

Method:
- Hold one finger close to the patient's nose.
- Ask him to look away at a distant object.
- Then ask him to look quickly at your finger.
- As the eyes converge to accomplish this pupils become smaller.
- If the patient is blind, the test may still be carried out by getting him to hold up his own finger about a foot in front of his face, and then asking him to 'look at the finger'.

CHECK FOR ABNORMAL PUPILLARY REFLEXES

Argyle Robertson pupils
This is a classical pupillary abnormality in neuro-syphilis. The pupil is small and irregular, reacts briskly to acommodation, but does not react to light. There is loss of direct and consensual light reflexes due to mid brain lesion which is usually due to neuro-syphilis. Near convergence reflex is preserved.

Marcus Gun Pupil (MGP):
When light is swung over the abnormal eye, both pupils dilate inappropriately. This is called as afferent pupillary defect or MGP. This may be due to optic nerve lesion. This is particularly useful sign in retrobulber neuritis and in ischemic or compressive lesions of optic nerve.

Constricted pupils
Sympathetic nerve activity causes pupillary dilatation and elevation of the upper eyelid; the later through the contraction of smooth muscle fibers in the levator palpebrae superioris.
Interruption of the cervical sympathetic nerve supply to the eye causes **Horner's synrome** (oculosympathetic paresis) which is characterised by the classic triad of:

- miosis (contricted pupil)
- partial ptosis and
- loss of hemifacial sweating (anhydrosis)
    - Anhydrosis is less commonly seen. It is the absence of sweating on the corresponding half of the face and neck both in front and behind, extending as low as the third rib and third cervical spine and over the whole of the upper limb on the same side.

- An apparent enophthalmos is often present, and is useful clue to the diagnosis when the patient is first seen.
- Congenital Horner's syndrome is associated with neuroblastoma and urine VMA should be measured.
- Drugs like opiates and cholinesterase inhibitors can also cause constricted pupils and should be considered in the differential dignosis of Horner's syndrome.

Ciliospinal reflex (pupillary-skin reflex)

It is the dilatation of the ipsilateral pupil in response to pain applied to the neck, face and upper trunk. If the right side of the neck is subjected to a painful stimulus, the right pupil dilates (increase in size 1-2 mm from base line). This is due to reflex excitation of the pupil-dilating cervical sympathetic fibers. This reflex is abolished by the lesions of these nerves and by some medullary, cervical and upper thoracic cord lesions.

Dilated pupils:

Causes

- Mydriatic drugs like atropine, tropicamide etc.
- Blindness due to damage to optic nerve

Unreactive pupils:

Causes

- Third nerve palsy
- Cataracts
- Corneal opacities
- Vitreous/retinal haemorrhage
- Prosthetic eye

Eye movements in different nerve lesions

- **3rd nerve lesion** may result in:
    - Ptosis, dilated pupils and loss of light and accomodation reflexes
- **4th nerve lesion** may result in:
    - Inability to look down and medially
- **6th nerve lesion** may result in:
    - Inability of eye ball to move laterally

Red reflex

Transparancy of the media of the eye may be observed by observing the red reflex .

Method to elicit red reflex:

- Set the ophthalmoscope at the zero and stand about 40 cm or an arm's length away from the patient.
- Whilst looking through the ophthalmoscope shine the light into each pupil.
- The vessels and the colour of choroid should elicit a red reflection.
- Red reflex may be dark or absent when cataract or viterous opacity is present

A **white reflex** may be seen in case of retinoblastoma.

Abnormalities of these three nerves may cause diplopia.

Since 6th cranial nerve within subarachnoid space has very long route, raised intracranial pressure (ICP) may cause failure of abduction of one or both eyes; a nonspecific sign of raised ICP.

5. Trigeminal

Major branches: ophthalmic, maxillary and mandibular divisions

Ophthalmic division

The lesions of ophthalmic division result in loss of cutaneous and corneal sensation. Trophic changes in the cornea may occur. Corneal reflex is abolished. Wisp of cotton or an esthesiometer can be used to measure sensation.

Method of testing corneal reflex

- Aske patient to gaze at the distance or onto the ceiling.
- Twist a light wisp of cotton into a fine hair and lightly touch the lateral edge of the cornea at its conjunctival margin with the wisp.
- If the reflex is present, the patient will blink.
- The two sides should be compared.
- Cornea should not be wiped with cotton and central part of cornea should not be touched to avoid the risk of corneal ulceration.

Maxillary division

The lesions of maxillary division, lead to loss of palatal reflex and loss of sensation at:

- the cheek
- the front of the temple
- the lower eye lid and its conjunctival surface

Mandibular division

The lesions of mandibular divison, lead to loss of sensations at:

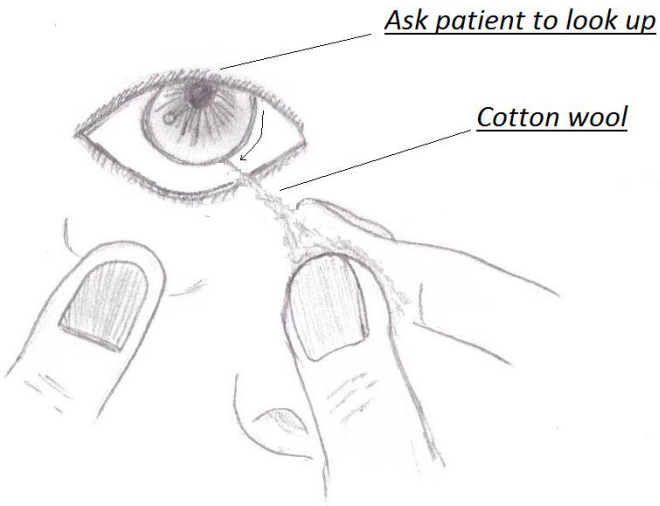
- lower part of the face
- lower lip
- ear
- tongue
- lower teeth

Whole trigeminal nerve

Lesions of the whole trigeminal nerve:

- will lead to loss of sensations in skin & mucous membrane of the face & nasopharynx.
- may develop diminished, salivary, buccal and lacrimal secretations leading to the development of trophic ulcers in the mouth, nose and cornea.

- may spare the taste but lack of oral secretions may result in its subjective impairment.
- will result in weakness of the muscles of mastication.

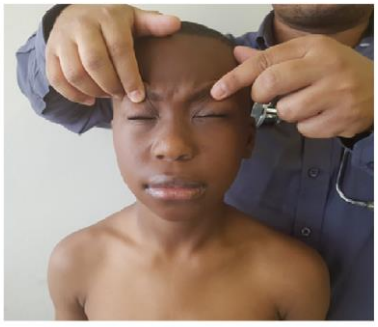


Corneal reflex

7. Facial

Examine the child and look if the face is symmetrical when crying or smiling?

- Can the child wrinkle the forehead and close eyes tightly?
    - A LMN lesion will affect both upper and lower halves of the face whereas an UMN lesion will affect only the lower half.
- Check blood pressure in a child with facial palsy; this may be the first presentation of systemic hypertension.
- Check the sense of taste



Picture: Examination to check if patient can wrinkle the forehead and close eyes tightly

Motor function: facial nerve supplies all the muscles of facial expression

- Any asymetry of the face
    - Opens eyes → Occulomotor - sympathetic nerves

    - Closes eyes → facial nerve
- Spontaneous movements
- Involuntary movements
- Wrinkle forehead
- Screw your eyes
- Show me your teeth
- Blow out cheeks

Common lesions of facial nerve

- Bells palsy and Bells phenomenon
- Ramsay Hunt Syndrome
- Unilateral LMNL: weakness of both upper and lower facial muscles
- Unilateral UMNL: facial paresis is marked in the lower facial muscles – sparing of upper face – because there is bilateral cortical innervation of the upper facial muscles

D/d of bilateral facial weakness, palsy or paralysis

Lower motor neuron lesions of facial nerve

Unilarteral or bilateral

- **Bell's palsy:** idiopathic acute paralysis of facial nerve (most commonly due to herpes simplex infection) affecting all the muscles on ipsilateral side of the lesion
- **Ramsay Hunt syndrome:** acute facial paralysis (most commonly) due to vericella zoster infection of lateral geniculate body
- Vascular lesions
- Bilateral parotid disease
- Myasthenia gravis
- Miller- Fisher varient of GBS

Upper motor neuron lesions of facial nerve

- Stroke, haemorrhagic/thrombotic vascular accidents
- **Brain tumours** causing raised ICP may present with buldging fontanelle & increased OFC
- **Infections:** herpes/ vericella
- **Moebius syndrome:** Palsy of 6 and 7^{th} cranial nerve usually bilateral + TEV (Talipes Equinovarus) & micrognathia. It is a sporadic condition and is the result of a number of different etiologies including developmental abnormalities of brain, peripheral nerve or myopathy
- **Metabolic** (seizure, myopathy):
    - MELAS (Mitochondrial Encephalopathy, Lactic acidosis, and Stroke like episodes)
    - MERF (Myoclonic Epilepsy with Ragged-red Fibers)
- **Di-George syndrome**: associated with facial palsy and congenital heart disease versus cardiofacial syndrome

8. Vestibulocochlear Nerve

Vestibulocochlear division comprises of:

- Auditory fibers which arise from cochlea
- Vestibular fibers which arise from otolith organs & semicircular cannals

Vestibulocochlear nerve runs with facial nerve in internal auditary meatus and both enter the brain stem at the cerebellopontine angle. Both may be affected by posterior fossa tumors or an accoustic neuroma

- Lesions of cochlear component of 8th cranial nerve will produce deafness &
- Lesions of vestibular component of 8th cranial nerve will produce symptoms of
  - Vertigo
  - Nausea
  - Vomiting
  - Diaphoresis
  - Nystagmus

For examination ask mom if the child can hear?

- Jangle the keys away from the vision of the child – see if he/she turns to the sound
- Clap hands and observe if he/she stares or blinks
- Alert neonates blink in response to bell
- Four months old infant turns his head and eyes to lacalise a sound

Cochlear division: to test the hearing loss

The simplest method of testing the hearing is a conversational voice that should be heard at 3.6 m in each ear separately and if this facility extends to 6 m it can be presumed that the patient has normal hearing.

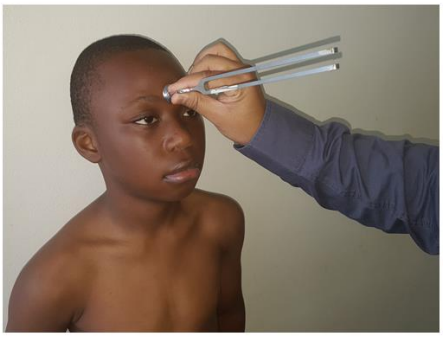
Tuning fork tests: Tuning forks 256 or 512Hz are usually used

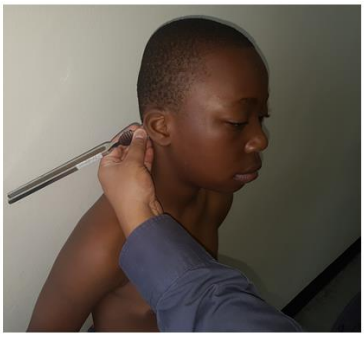
Rinne test:

- It compares the hearing through air conduction with the bone conduction (with base of the fork on the mastoid process).
- In the normal ear, air conduction is better than bone conduction (Rinne positive).
- This also applies to patients who have a sensorineural deafness, where as patients with a conductive deafness will show the opposite response i.e., bone conduction better than air conduction (Rinne negative).

Webber's test:

- The base of tuning fork is placed anywhere on the midline of the skull or mandible.
- In conductive deafness patient will perceive in the deafer ear whilst, in the cases of sensorineural loss, it is referred to the better ear.





Picture: Above - Webber's Test , Below - Rinne Test

CAUSES OF DEAFNESS

CONDUCTIVE DEAFNESS

1. Wax in the ears
2. Chronic secretory otitis media
   - Glue ear
   - Grommets
   - Damage to ear drums

PERCEPTIVE DEAFNESS

1**. Congenital**
   - Inherited – may be isolated or part of a syndrome
   - Waardenburg's syndrome (AD)
   - Congenital malformation of ears
   - Intrauterine infection: TORCHES

- **Perinatal causes**
   - Perinatal hypoxia
   - Kernicterus
   - Use of aminoglycosides

- **Postnatal causes**

1. Meningitis
2. Encephalitis
3. Trauma
4. Ototoxic drugs

Vestibular division

To assess for sense of balance and spacial orientation for the purpose of coordinationg movement with balance i.e., vertigo & nystagmus

- Abnormality of posture or gait
- Induction of positional nystagmus
- Doll's eye reflex
- Oculovestibular reflex
- Dix-Hallpike maneuver

Nystagmus is caused by the lesion of cerebellum, brainstem, cervical cord or inner ear - always offer to examine hearing, cerebellar functions and the occular fundi

Nystagmus

Involuntary & repetitive oscillations of the eye or eyes is called as nystagmus. It is defind by the direction of the fast phase but it is the slow phase which is pathological. Nystagmus may suggest end-organ or intrinsic disease of the nervous system.

The most common cause of nystagmus in children is caused by the centrally acting medications like anticonvulsants

Central nystagmus:
Nystagmus in any direction results from:

- Brain stem lesions
- Vertebrobasilar ischaemia
- Phenytoin toxicity

Pendular nystagmus
This refers to the waveform of involuntary eye movement in which eye moves in sinusoidal trajectory, similar to that of a pendulum. Rhythmic abnormalities that are equal in all planes of gaze; associated with either cerebellar or retinal disease

Jerk nystagmus
Consists of a slow and fast phase and refers to abnormalities of vestibular system

Vertical nystagmus
Primarily seen in abnormalities of the brain stem

Ocular nystagmus
Due to poor macular vision which impairs retinal fixation

9. Glassopharyngeal Nerve

Ask simple questions to the care giver and observe like:

- Can the child swallow?
- Is the child drooling?

Then:

Assess patient's speech for dysarthria or dysphonia

Check for:

- Palatal reflex: tickle the back of the pharynx, and note if contraction occurs. This is also a test for vagus nerve.
- Ask the child to puff out cheeks with lips closed: air leak from nose will indicate lesion
- Examine the taste in the posterior 1/3rd of tongue: Loss of taste at this part of the tongue may occur with a lesion of the trunk of the glossopharyngeal nerve.

10. Vagus Nerve

Following test can be performed:

- Ask the patient to say Aah and look at the palate and note position of uvula. If it is in the center and moving in either side than that's normal and if it is deviating on one side that side is considered normal because of pull by the stronger muscles
- Note nasal twang
- Note regurgitation of food
- Note hoarsenes of voice; due to paralysis of laryngeal muscles

Common causes of bilateral vagus nerve injury

- Progressive bulber palsy
- Bilateral supranuclear lesions
- Cerebrovascular disease
- Multiple sclerosis

9th through 12 cranial nerves are called bulbar cranial nerves. Gag reflex is caused by touching the posterior pharyngeal mucosa. Both 9th and 10th cranial nerves are responsible for gag reflex.

11. Accessary Nerve

It is purely a motor in function. Check bulk and test power of sternocleidomastoid and trapezius muscles.

Method

- **Testing the trapizius muscle**
  - Tell the patient to shrug shoulders while the examiner presses downward on them. Paralysis of the upper part of trapizius muscle is demonstrated in this way.
- **Testing the right sternocleidomastoid muscle**
  - Face the patient and place your right palm laterally on the patient's left cheek. Tell the patient to turn head to the left, resisting the pressure you are excerting in the opposite direction. Paralysis of the sternomastoid causes weakness of rotation towards the opposite side.
- **Testing the left sternocleidomastoid muscle**
  - Face the patient and place your left palm laterally on the patient's right cheek. Tell the patient to turn head to the right, resisting the pressure you are excerting in the

opposite direction. Paralysis of the sternomastoid causes weakness of rotation towards the opposite side.

12. Hypoglossal Nerve

It is purely a motor nerve. It supplies the tongue and the depressors of the hyoid bone.

It is tested by examining the tongue and its movements as follows:

- If the nerve is injured, at rest the tongue may appear to have the appearance of a "bag of worms" (fasciculations) or wasting (atrophy). Fasciculations must be assessed with the tongue relaxed in the mouth, not when protruded.
- Ask the patient to stick the tongue out as far as possible. If the hypoglossal nerve is paralysed, the tongue, instead of being protruded straight, will be pushed over to the paralysed side.
- Ask the patient to move the tongue from side to side and lick each cheek with it; observe whether patient can do so freely.
- Strength may aslo be assessed by pressing against the tongue with a finger as the patient protrudes it into each cheek in turn.
- Patients with hypoglossal nerve disorder have difficulty speaking, chewing and swallowing.
- Assess speech by asking the patient to say "red lorry and yellow lorry" etc.

SUMMARY: IF ALL CRANIAL NERVES ARE NORMAL - SAY

1. Pupils equal and reactive to light
2. Fixes and follows
3. No ophthamoplegia
4. No facial asymetry
5. Hearing appears intact
6. Tongue central
7. Positive gag reflex
8. No drooling

NB! Cavernous sinus thrombosis causes problems in 3 cranial nerves**;** Occulomotor, Trochlear and Abducent (OTA) nerves.

6. EXAMINATION OF MOTOR SYSTEM

Achronym: BPTR: Bulk, Power, Tone, Reflexes

EXAMINE THE MUSCLE BULK

Inspection

- Examine after full exposure and look for deformity
    - Bony or soft tissue
    - Pes cavis/Pes planus or claw of hands
        - Pes mean foot
        - Pes cavis: foot with abnormally high longitudinal arch

        - Pes planus or flat foot: when entire sole of the foot touches the floor on standing
    - Ankle valgus: an insidious deformity that results in pronation of the foot and medial malleolar prominence
- Look for asymmetry: Proximal/distal
- Look for muscle wasting or hypertrophy
    - Loss of muscle bulk may be due to a lower motor neuron lesion, disuse atrophy or generalised wasting
    - Excessive muscle bulk may be due to Duchenne Muscular Dystrophy or Myotonia Congenita, may look like baby Hercules

Examine the posture:

- Shoulder adducted, elbow flexed, hand clinched may be due to pyramidal tract problem
- Erb's palsy (policeman tip hand deformity)
- Claw hand (atrophy and distortion of hand)
- Arthrogryposis (persistant flexion of a joint, tetnoid spasm)

Palpate the muscles to assess bulk

Common abnormalities

- Athetosis: repetitive involuntary, slow gross movements which involve distal parts of the extremities
- Peripheral neuropathy: distal wasting (inverted champaign bottle)
- Check thenar/hypothenar for muscle wasting

EXAMINE THE POWER

Grade the power according to Medical Research Council Scale

- Grade 0: No muscle movement (complete paralysis)
- Grade 1: Muscle flicker, no joint movement
- Grade 2: Full movement is possible when gravity is illiminated
- Grade 3: Movement against gravity but not against resistance
- Grade 4: Power against resistance but can be overcome by resistance
- Grade 5: Normal power against gravity and full resistance is present

Check proximal/distal power

- **Upper limbs**
    - Check thumb for flexion/extension
    - Check abduction/adduction of fingers
    - Squeez hands and check grip/strength
    - Examine wrists – stablise at elbow – extend/flex
    - Examine elbows – stabilize upper arm – extend/flex
    - Check shoulders for abduction/adduction – shrug shoulders

- Elicit pronator drift: when child extends his or her arms out in front with palms upword and eyes closed. Hands on weak side cup and pronate slowly. This is scalled as apronator drift.

- **Examine neck:** patient to lie flat, check for flexion/extension of neck
- **Examine trunk for muscle weekness:** note for pot belly/lumber lordosis
- **Check for proximal weakness:** elicit Gower's sign
- **Lower limbs**
  - Big toe: extend & check for muscle wasting at the dorsum of the foot
  - Foot: invert/evert, dorsiflex/planter flex
  - Hip, knee – extend and flex
- **Compare**
  - UL/LL
  - Right to left
  - Proximal to distal

Picture: Examination - Power

EXAMINE THE TONE

Muscular tone is a state of tension or contraction found in healthy muscles. It represents the dynamic resistance of muscles to movement across a joint (stretch or to gravity). An increase in the tone is called as hypertonia and a diminution as hypotonia. The degree of tone is estimated by handling the limbs and moving them passively at their various joints.

- **Passive tone** is the resistance to stretch felt when limb is flexed and extended manually
    - In LMNL, passive tone is reduced
    - In chronic UMNL, passive tone is increased
- **Active tone** is the posture that an infant adopts when placed in a particular position

Hypertonia

Causes of hypertonia may include upper motor neuron disease, lesion in basal ganglia, and anxiety. Hypertonia following lesions of the corticospinal system (UMNL) is termed as spasticity. The term spasticity is used to describe a state of increased tone which is of claps knife type.

- **Claps knife type hypertonia** is when the limb is fairly rapidly flexed or extended i.e. when limb is flexed, initially it is very rigid but after certain degree of flexion the resistance suddenly disappears & same thing happens when extending it. Clasped knife response or spasticity is seen in upper motor neuron lesions.
- **Cogwheel rigidity** hypertonia results from disease of the basal ganglia also termed as extrapyramidal rigidity**.** In this type, the resistance to passive movements is fluctuant i.e. if you try to flex the elbow, it will flex in a jerky manner described as like a lever rubbing on the teeth of a cogwheel (cogwheel rigidity). This cogwheel rigidity is seen in extrapyramidal disease.
- **Lead pipe rigidity**, the rigidity is uniformly present throughout the passive movements (flexion or extension). This is present in cases in which patient's state of consciousness is impaired.

Hypotonia

In hypotonia, there is little or no resistance to passive movement of the limb and when handled or shaken the unsupported part flops about inertly. Hypotonic muscles are abnormally soft to palpation. Causes of hypotonia may include lower motor neuron disease, unconsciousness, and neural shock i.e. in early stages of UMNL.

Do passive movements of flexion and extension in all the four limbs, slowly and then quickly and note abnormalities of tone:

- Hypotonia
- Spasticity
- Rigidity

EXAMINE THE REFLEXES

There are superficial & deep tendon reflexes.

Superficial reflexes

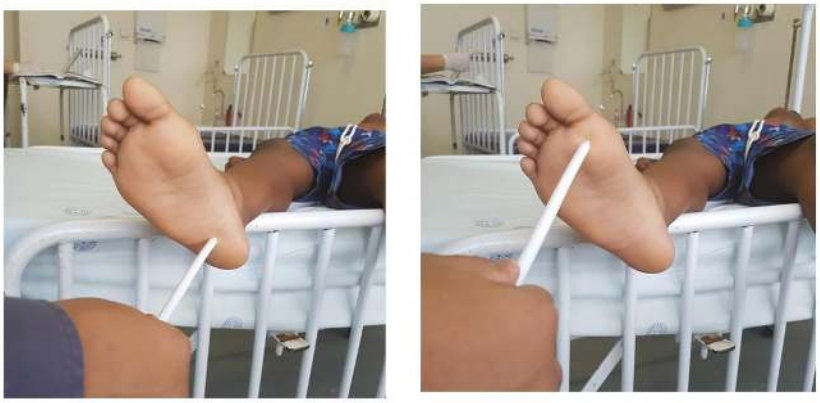
Reflex	How elicited	Result	Level of cord concerned
Anal	Stroking or scratching the skin near the anus	Contraction of anal sphincter	S3, S4
Planter	Stroking sole of foot	Flexion of toes, foot or leg	L5, S1
Cremasteric	Stroking skin at upper and inner part of thigh*	Upward movement of testicle	L1, L2 Lost in UMNL
Abdominal	Stroking abdominal wall below costal margin, at the level of umbilicus and in iliac fossa	Contraction of abdominal muscles	T7 to T12 Lost in UMNL & LMNL
Scapular	Stroking skin in the intrascapular region	Contraction of scapular muscles	C5 to T1
*the cremasteric reflex can often be more easily elicited by pressing over the sartorius in the lower third of **Hunter's canal** (an aponeurotic tunnel in the middle third of the thigh, extending from the apex of the femoral triangle to the opening in the adductor magnus, the adductor hiatus)			

Babinski's sign:

Scratch the outer border of the sole of foot with edge of a key or the stick of a patella hammer. Normally the greater toe goes up and there is flexion and adduction of all other toes and foot is dorsiflexed and inverted. This is called as negative Babinski sign or planter reflex (planters are flexors). In positive Babinski the big toe bends up and back to the top of the foot and other toes fan out. Nerve roots involved are L5, S1.

Positive Babinski is present in:

- Upper motor neuron lesion
- During sleep
- In infants – untill they start walking (about 1 yr)
- Post ictal or coma



Picture: Babinski's sign

Deep tendon reflexes

If the tendon of a slightly stretched muscle is struck (a single, sharp blow) with a soft rubber hammer, the muscle contracts breifly. This is the monosynaptic stretch reflex. It is the test of integrity of the afferent and efferent pathways and of the excitability of the anterior horn cells in the spinal segment of the stretched muscle. When eliciting the tendone reflexes grade responses as follows:

- Grade 0: Absent reflex
- Grade 1: Present normal
- Grade 2: Brisk (as normal knee jerk)
- Grade 3: Very brisk
- Grade 4: Sustained clonus

When eliciting a tendon reflex following need to be kept in mind:

- There should be no joint disease of the limb on which reflex is going to be elicited
- Patient should be fully relaxed and the part of the body to be tested should be fully exposed.
- Concentration should be on the movements of the muscles whose tendon has been struck by the hammer

BICEPS JERK (C5, C6)

Method 1:

- Flex the arm at the elbow and place forearm on the abdomen.
- Put your index finger over the tendon of biceps muscle and press it lightly.
- Now strike the hammer on your finger and note the Biceps jerk.

Method 2:

- Hold the patient's elbow in your left hand in a manner that your thumb should be on the tendon of biceps muscle.
- The patient's upper limb should be fixed at elbow and it should rest on your left forearm relaxed.

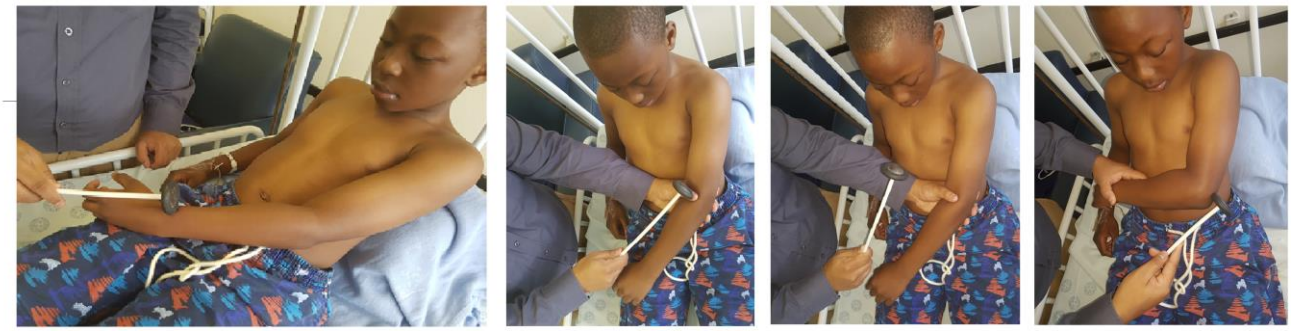
- Now press the tendon of biceps muscle lightly with your thumb and strike the hammer over your thumb.
- Note intensity of reflex by seeing the contraction of the muscle.

TRICEPS JERK (C6, C7)

- Keep the forearm on the abdomen with elbow flexed.
- Strike the hammer on the tendon of triceps muscle just above the olecranon process.
- Note intensity of reflexion by seeing the contraction of the triceps muscle.

SUPPINATOR JERK (C5, C6)

- Keep the forearm placed on the abdomen in semi pronated position with flexed elbow.
- The hand should slightly deviate towards the ulnar side.
- Now strike with patella hammer on the styloid process of the radius (the tendon of brachio-radialis muscle).
- Note intensity of reflex by seeing the contraction of Brachio-radialis muscle.



Picture: Deep tendon reflexes of the upper limbs

KNEE JERK (L3, L4)

There are many methods to elicit the knee jerk. Following three are commonly used:

Method 1:

- Make the patient to sit at the edge of the bed with his legs hanging down.
- Remove trousers from the thigh's and locate the tendon of the quadriceps femoris
- Give a stroke on the tendon with patella hammer and note the grade of reflex.

Method 2:

- Make the patient to lie on the bed with both knee joints flexed.
- Pass your hand under the knee to be tested and bring it to rest upon the opposite knee.
- In this way the knee to be tested will hang on the dorsum of your wrist.
- Now divert the attention of the patient in some way and strike the hammer on the tendon of the quadriceps midway between the tibial tuberosity and the patella.

Method 3:

- Make the patient to lie supine on the bed with lower limbs slightly flexed at hip and knee joints with femur externally rotated in a manner that the heels of both sides are near to each other.
- Now strike the tendon of quadriceps femoris muscle and note intensity of reflex by seeing the contraction of the muscle.

JENDRASSIK MANEUVER

Jendrassik manoeuvre is used to elicit the deep tendon reflexes. In this manoeuvre the patient clenches the teeth, flexes both sets of fingers into a hook-like form, and interlocks those sets of fingers together. The tendon below the patient's knee is then hit with the patella hammer. The elicited response is compared with the reflex result of the same action when the manoeuvre is not in use.

The amplitude of the response is increased because a larger number of motor units have been activated. Clinically some brain stem lesions are manifested by exaggerated spinal cord reflexes. Isometric contractions in another muscle group enhance the response of the reflex. Often this is used to distract the patient and relax the muscles in the quadriceps.

ANKLE JERK (S1, S2):

Method:

- Ask the patient to lie on the bed with legs everted and slightly flexed at the knee.
- Now, with one hand slightly dorsiflex the foot and strike the tendon Achilles.
- There will be contraction of the calf muscles and the foot will undergo planter flexion.

Picture: Deep tendon reflexes of the lower limbs

CLONUS

This is elicited when the tendon reflexes are exagerated.

Clonus ordinarily occurs when the stretch reflex is highly sensitised by facilitatory impulses from the brain. For instance in a decerabrate animal in which stretch reflexes are highly facilitated, clonus develops readily. It is done to determine the degree of facilitation of spinal cord.

Clonus may be sustained or unsustained. Sustained clonus is indicative of hyper-reflexia (UMNL), while unsustaiend clonus is present in nervous and anxious persons. This is of two types: Patellar and Ankle clonus

Patellar clonus

Method:

- Ask the patient to lie down supine on the bed and you hold the tendon of quadriceps by upper border of the patella.
- Now give 2 to 3 tendon jerks downwards. In positive cases, there will be series of clonic contractions of the quadriceps and the patella will move with it.

Ankle clonus

Method:

- Ask the patient to lie down supine on the bed.
- Flex the knee along with slight flexion of hip joint and support the leg with your hand.

- Now grasp the forepart of the foot with other hand and give 2 to 3 sudden dorsiflexion movement to the foot.
- The pressure on the sole of the foot is sustained by keeping it in dorsiflexed position. There will be series of contractions of calf muscles in positive cases. So there will be repeated vibrations at the ankle joint.

Picture: Examination to elicit ankle clonus

Hoffman's reflex

The test involves tapping the nail or flickering the terminal phalanx of the middle finger. Hoffman's test is positive if you see adduction of thumb and flexion of index finger which may indicate an upper motor neuron lesion or a pyramidal sign. Hoffman's sign may be seen in multiple sclerosis and conditions which cause spinal cord compression such as cervical myelopathy, cervical spondylitis, tumors or degenerative arthritis.

Wartenburg's sign

It is a neurological sign consisting of involuntary abduction of the little finger, caused by injury of the ulner nerve which supplies intrinsic hand muscles (particularly palmer interosseous muscle) which results in unopposed action of the extensor digiti minimi. This is commonly seen in ulner nerve neuropathy and cervical neuropathy.

Pendular knee reflex

This is one of the cerebelalr sign where leg keeps swinging after knee jerk more than 4 times (4 or less is normal), which ma be due to cerebellar damage.

Crossed adductor reflex

Contraction of both hip adductors when either knee jerk is elicited. The cross adductor response usually disappears by 7 to 8 months. Its persistance beyond 8 months is a sign of pyramidal tract dysfunction.

Method

- Ask the patient to lie down suppine on the bed with hips and knees partially flexed and abducted with soles of the feet facing each other in the centre.
- Now strike the hammer on the medial aspect of the knee. If both the legs are adducted the test is considered as positive which indicates pyramidal tract dysfunction.

7. EXAMINATION OF SENSORY SYSTEM

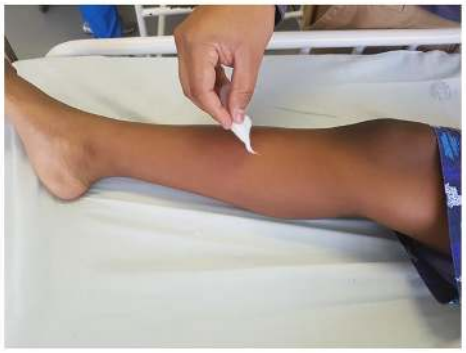
Loss of sensation means there is no feeling of pain, heat, or cold. This can happen in one or more parts of the body. And because of this, one is more likely to hurt himself so look for sores especially on feet and legs of the patient. Main conditions that may cause loss of sensations include problems with the peripheral nerves (neuropathy), stroke, spinal cord injury, tumours and chronic infections.

Following various sensations can be tested:

Sense of light touch

- Expose the part of the skin to be examined.
- Ask the patient to close his eyes.
- Now take a wisp of cotton and touch the identical points of the body of the patient; the upper limbs, then trunk, then back and finally lower limbs.
- Two questions are asked when examining sensation of touch.
    - **Where the wisp of cotton is touching?** This will give you an idea of sense of localisation of touch (tactile localisation) of the patient. Ask the patient to say "yes" every time he feels the touch.
    - **How many wisps of cotton are touching him?** Before asking the question, touch the patient with two wisps of cotton close to each other. By this we can make out the sense of discrimination of touch (tactile discrimination) of the patient.

If there is lesion in the anterior spinothalamic tract, the sense of localisation and discrimination of the patient will be affected.



Picture: Sensory examination - Light touch

Sense of course touch

Method

- Press the limb with finger or blunt probe on identical points.
- Now ask the patient whether he appreciates or not.

Sense of pain

Pain is carried by lateral spinothalamic tract; therefore, its lesion will impair the pain sensation.

Method

- Take a steel pin or sterile needle (rarely done in children).
- The pin is pricked on the identical points and the patient is asked whether he feels pain or not.

Sense of deep pain: Two tests are done

- Squeeze the calf muscles and apply pressure in between the thumb and the index finger. Note if the patient feels pain.
- Deep pain is absent in tabes dorsalis (a form of neurosyphilis in which there is loss of both axons and the myelin in the dorsal roots, with pallor and atrophy in the dorsal columns of the spinal cord).

Sense of temperature

- This is tested by hot and cold water contained in two copper test tubes.
- The naked area under the examination is touched with each, in turn, and the patient is asked whether he feels it hot or cold.
- Lesion in the lateral spinothalamic tract will impair or even abolish this sensation.

Sense of position and passive movements (posterior column integrity):

Method 1

Here the movements of thumb and big toe are tested.

- The patient is made to understand the movements of big toe or thumb which is moved up, down and sideways.
- Ask the patient to close his eyes.
- Now hold the big toe with your thumb and index finger.
- Flex it at the distal interphalangeal joint, slightly without touching the other toes and ask the patient whether it is directed upward or downward.
- If unable to make final assessment test can be repeated number of times.
- Same procedure can be used for upper limb by the movement of thumb.

Method 2

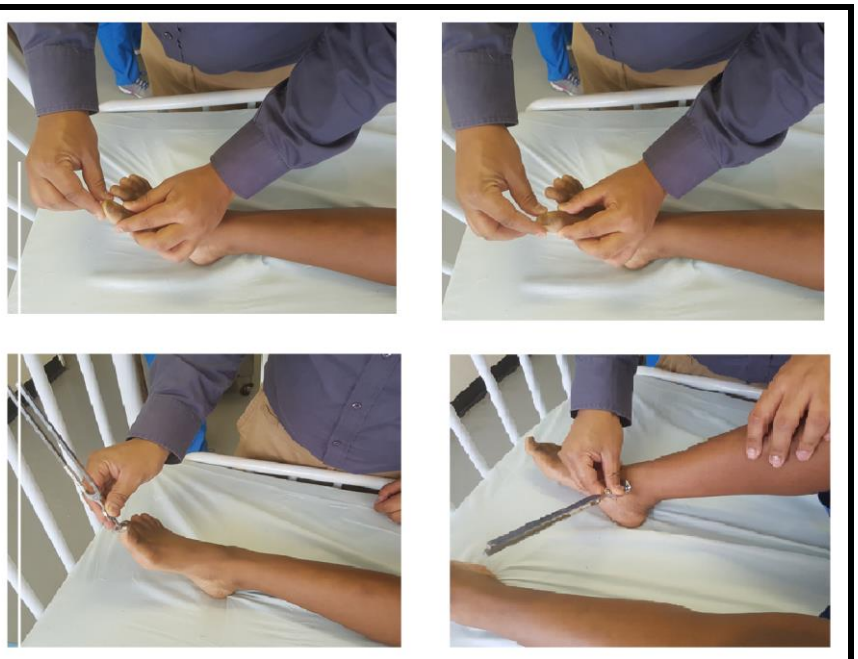
- Ask the patient to close his eyes.
- Hold the thumb of the patient and move it in various directions through the air.
- Now place the thumb of the patient in an abnormal position and ask the patient to place his other thumb in the same position.
- In the case of hand, move the fingers in various directions and ask the patient to imitate with other hand while eyes are closed.

- Testing limb should not touch any other skin surface; otherwise patient will be able to appreciate its position in spite of the lesion.

Sense of vibration

Tuning fork (frequancy 128/sec) is used to test sense of vibration.

- Strike the tuning fork on the side of your knee and then place its base over the bony prominances, i.e. medial and lateral maleoli, tubercles of tibia, anterior superior iliac spines and olecranon processes.
- Normal individual will feel the buzzing sensation.
- If unable to feel the vibration sense, consider polyneuropathy or disease of posterior column.



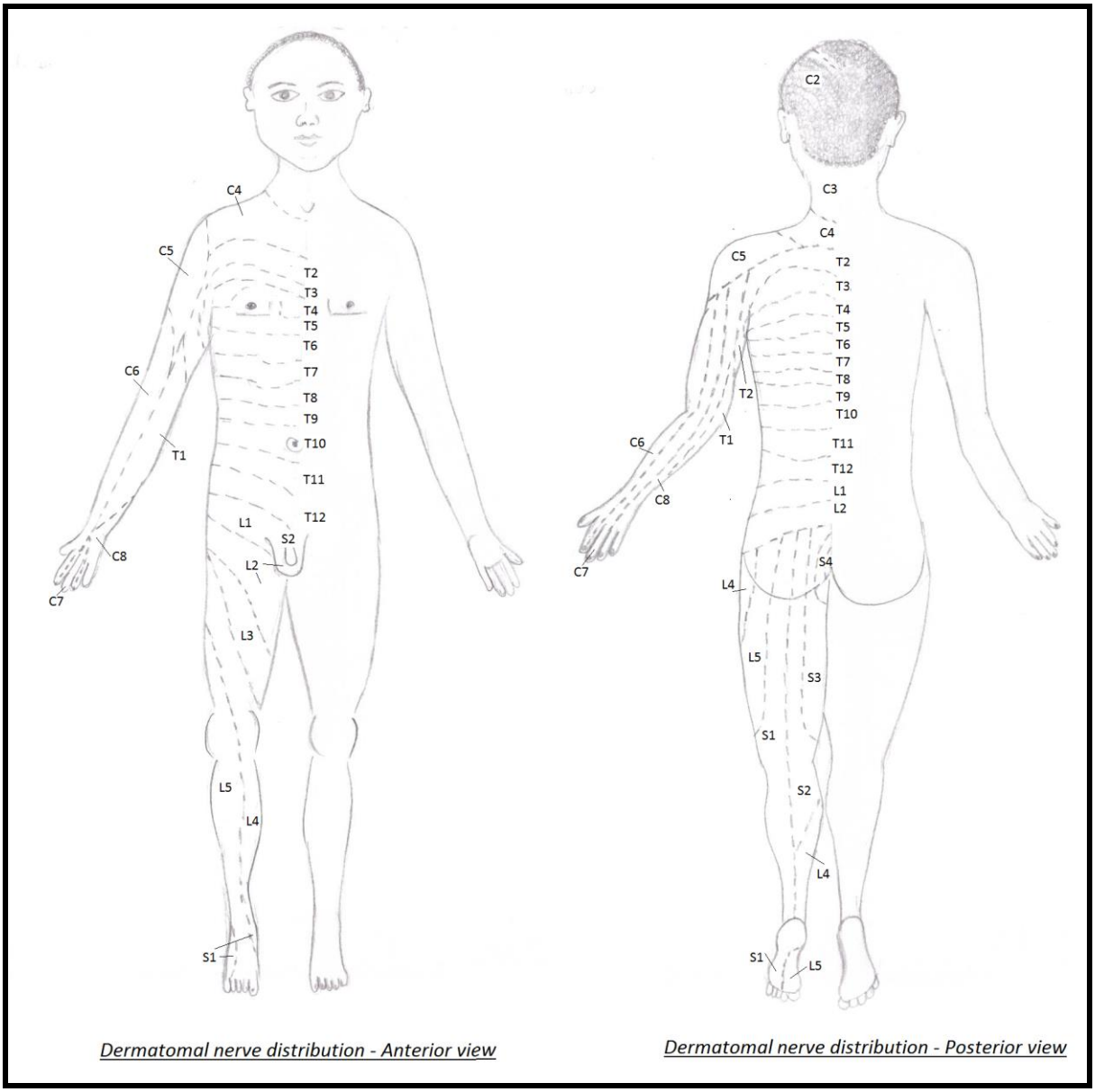
Picture: Sense of position and passive movements

Brown-Sequard Syndrome

It is a rare neurological condition cheracterised by an incomplete lesion in the spinal cord (often in the cervical cord region) which results in weakness or paralysis (hemiparaplegia), spascity and ataxia on one side of the body (upper motor neuron paralysis) and loss of pain and temperature sensation (hemianaesthesia) on the opposite side. There may also be loss of bladder and bowel control.

Papilloedema is observed in few patients usually in association with markedly elevated CSF protein levels that presumably interfere with normal CSF flow dynamics.

Focus mainly need to be on the underlying cause of the disorder. Early treatment with high dose steroids may be beneficial in many cases. Physical, occupational and recreational therapy is important aspects of patient rehabilitation.



8. CO-ORDINATION: CEREBELLAR SIGNS

The cerebellum receives information from the sensory system, the spinal cord and other parts of the brain and then regulates motor movements such as posture, balance, coordination, speech and rate, range, rhythm and force of muscle contraction. Following signs should be lookd for in the lesions of cerebellum:

Ataxia

Ataxia is impaired balance or coordination of movement. Patients with cerebellar lesion will not be able to walk in straight line with the eyes open. Cerebellum and associated pathways modulate volitional movement.

If either afferent cerebellar connections (joint position sense) or efferent cerebellar connections (cerebellum through thalamus through cerebral cortex) are disturbed, the patient experiences ataxia.

Adiadochokinesia (inability to conduct rapid alternating hand movements)

Method 1

To elicit loss of rapid alternate movements.

- Ask the patient to flex his elbows at the right angle and to pronate and supinate his forearm by placing one hand over the other.
- The patient will not be able to do so and his movements will be slow and incomplete.

Method 2

- Ask the patient to tap your palm with his finger tips as fast as possible.
- Patient will not be able to do so.

Asynergia: This means inco-ordination. This is tested in both limbs:

UPPER LIMB

Finger-Nose test (finger nose pointing or dysmetria)

- Ask the patient to touch tip of your index finger which is at adistance of about ½ meter from the patient) with his index finger and then to touch his nose.
- This should be done rapidly. Patient will fail to do so.

Picture: Finger-nose test

Finger-Finger test

- Ask the patient to touch the tips of his both index fingers with extended arms bringing them from a distance rapidly.
- Patient will not be able to do so.

Rebound phenomenon (loss of rebound control):

- Ask the patient to flex his elbow slightly and flex it further firmly against resistance (i.e. you hold his hand) then release it suddenly.
- The patient will slap his chest due to rebound phenomenon but normal individual will be able to prevent it to happen.
- If the results of above tests are doubtful, then patient should repeat the above tests while his eyes closed - irregularity in the movements will become more marked

LOWER LIMB

Heel-Knee Test

- Ask the patient to lie in the bed in supine position

- Then to put the heel of his foot on the knee of other leg and rub it down along the shin
- Then raise the leg in the air and again put the heel on the knee.
- He will fail to do so.


Picture: Heel knee test

Cerebellar gait

- It is a staggering, wide-based, unsteady gait which has lurching movements with a breakdown of normal synergy of walking.
- In the older child one can test ataxic gait by asking the patient to walk by heel to toe (like walking on a tight rope).

Pendulous knee jerk

- Ask the patient to sit in the bed with legs hanging down and elicit the knee jerk.
- The leg after extension will fall down in a pendulous fashion

Charcot's triad [SIN]

- Scanning speech
  - Ask the patient, to speak few words
  - Note slow, slurring or explosive dysarthria, poor or no cordination
- Intension tremor
  - Ask the patient to reach out and touch your finger
  - Note the fine tremor increasing as it approaches the finger
- Nystagmus
  - Ask the patient to look straight ahead at you
  - Note the abnormal movement of eye from slow to rapid phase - nystagmus

9. SPINAL CORD (CERVICAL, THORACIC AND LUMBER SPINE)

Do full examination of the spine

- Look for hairy patch, lipoma or spina bifida
- Palpate the spinous processes and paraspinal tissues
- Note overall alignment and focal tenderness

- Lightly percuss the spine with your closed fist – note any tenderness
- Move and observe flexion, extension, and lateral flexion

Note clinical signs in the case of "Hemisection of Spinal Cord"

- Below the level of lesion
  - Features ipsilateral to the lesion
    - Impaired light touch
    - Impaired vibration touch
    - Corticospinal tract signs
  - Features contralateral to the lesion
    - Impaired pain sensation
    - Impaired temperature sensation
- At the level of lesion
  - Features ipsilateral to the lesion
    - Segmental zone of hyperpathia (pain)
    - Signs of lower motor neuron at this level

Lesion in the anterior horn cells	Lesion in the nerve
• Disease process is gradual in onset	• Disease process is sudden in onset
• Fibrillation and fasciculations are present	• Fibrillation and fasciculations are not present
• Only motor system is involved	• Both motor and sensory systems are involved
• There is no sphinteric disturbance	• Sphinteric loss is present

10.AUTONOMIC NERVOUS SYSTEM

Sympathetic Nervous System

Horner's syndrome:

This is due to the lesions in the cervical sympathetic trunk which leads to:

- Ptosis of the eye lid
- Enophthalmos (eye ball moves inward)
- Miosis (constriction of the pupil)
- Loss of sweating (anhydrosis)

Loss of cilio-spinal reflex:

On pinching the lateral side of the neck, sympathetic trunk gets stimulated which results in dilatation of the pupil. This reflex is lost in the diseases involving cervical sympathetic trunk.

Parasympathetic nervous system

- Cranial nerve III (ciliary ganglion) supply cilliary muscles and the sphincter pupillae
- Cranial nerve VIII (Pteropalatine ganglion + submandibular ganglion) axons projet to the lacrimal glands, mucous membrane and nasal mucosa
- Cranial nerve IX (otic ganglion) scretomotor fibers then pass to parotid gland
- Cranial nerve X (ganglions close to organ) supplies the:
  - Heart
  - Larynx/trachea/bronchi
  - Oesophagus, stomach
  - Abdominal blood vessels
  - Liver & bile duct
  - Pancrease
  - Adrenal gland
  - Small intestine
  - Large intestine

S1 to S4 → Colon, rectum, kidney, bladder, penis, clitoris

11.EXTRAPYRAMIDAL SYSTEM

Lesions of the basal ganglia cause involuntary movements

Chorea (of hands and fingers)

Cheracterised by involuntary, irregular and purposeless movements of the hands and fingers like piano-playing movements

Huntington's chorea: absent caudate shadow on CT brain

Signs

- Protruded toungue
- Milkmaid sign
- Shelving sign

Sydenham chorea

- Post streptococcal
- Increased ASOT
- Cardiac murmur

Athetosis

Slower and writhing movements

Dystonia

Abnormal posture due to periodic sustained contraction of opposing muscle group

Ballism
Sudden massive jerky flinging movements

Basal nuclei
Lesions of basal nuclei do cause cog wheel or lead pipe regidity

12.PERIPHERAL NERVE DAMAGE

Lesions in bracheal plexus

Upper lesion – Erb-Douchenne's paralysis

- Deformity:
  - upper limb hangs by the side
  - internally rotated
  - and forearm pronated
  - also called as policeman's receiving tip hand
- Lesion C5 and C6
- Lesion C7 in 50% cases
- Paralysis of biceps brachialis, brachioradialis and supinator muscles

Lower lesion – Klumpke's paralysis: here lower nerve roots are injured

- This deformity is also called as "Claw Hand" in which there is:
  - Weakness of the flexor muscles of the wrist
  - Weakness of the small muscles of the hand
  - Lesion C8, T1
  - Up to 1/3rd of these patients have Horner's syndrome.

Median nerve lesion: carple tunnel syndrome
Radial nerve lesion: wrist drop
Ulner nerve lesion: claw hand
Common peronial nerve lesion: foot drop
Lateral cutaneous nerve of thigh lesion: loss of sensation over lateral aspect of thigh

GENERAL PRINCIPLE

When hemiplegia is crossed i.e. hemiplegia on the one side and involvement of cranial nerves on the opposite side, the lesion is in the brainstem which may be in the:

- Midbrain
- Pons
- Medulla oblongata

But if the hemiplegia is uncrossed i.e. hemiplegia and the involvement of cranial nerves are on the same side, the lesion is above the brainstem which may be in:

1) Internal capsule
2) Corona radiata

3) Cerebral cortex

AT THE END OF THE EMANIBATION MAKE A REASONABLE ASSESSMENT

What is the dysfunction → like spastic quadriplegia with profound developmental delay

Where is the lesion → like in cerebral cortex

What is the aetiology → like infarct due to hypoxic ischaemic encephalopathy

WHAT IS THE DIAGNOSTIC EVALUATION LIKE

1) Normal or abnormal
2) UMNL or LMNL
3) Site of lesion
4) Nature of lesion
5) Complications
6) Developmentally normal or abnormal
7) Static or progressive
8) Acute or chronic

If it is lower motor neuron lesion, then describe as:

It is lower motor neuron lesion due to

- Hypotonia
- Weakness
- Absent reflexes
- Wasting

And conclude with summary of your findings and make a provisional diagnosis on the basis of your clinical examination.

TUTORIAL 11

THE EXAMINATION OF MUSCULO- SKELETAL SYSTEM

OVERALL OBJECTIVE

At the end of this module, the student should be able to do a full clinical examination of musculoskeletal system and to be able to make a reasonable differential diagnosis of swollen joints, rheumatic fever, systemic lupus erythematosis, dermatomyositis, gait problems, muscular atrophy or hypertrophy in infants and children.

Extra General features include:

- Malar rash of SLE
- Supraorbital rash of dermatomyositis
- Muscular hypertrophy or pseudohypertrophy
- Muscle atrophy
- Muscle fasciculations
- Signs of acute rheumatic fever
- Rickety rosery, craniotabes, widening of wrists – rickets

Screening questions

1) Do you have pain or stiffness in your muscles, joints, back?
2) Can you dress up yourself without difficulty?
3) Can you walk up and down stairs without difficulty?

EXAMINATION OF A JOINT

Introduce yourself to the patient and the caregiver

- Sit in front of the patient: good position is confruntation position
- Do a good observation
  - Ask the patient to walk a short distance and watch gait
  - Ask the patient to take off a jumper or shirt and observe upper limb function
  - Ask the patient to write his name and observe his hand function
  - Ask the patient to undress and observe the upper limb function

General examinatin will include:

- Sequence: Look, Feel, Move, Measure
- GALS screen: Gait, Arms, Legs, Spine

SEQUENCE

1. Look

- General inspection, see if the patient is:
  - Thriving, has any bandages or is on a wheelchair.
  - Having any prosthesis e.g. calipers or special shoes

- Has any spine deformaties e.g. kyphosis, scoliosis or kyphoscoliosis
- Has any limb deformaties like:
    - **Amelia** (birth defect of lacking one or more limbs)
    - **Hemiamelia** (upper part of limb well developed but the lower part is rudementary or absent)
    - **Phocomelia** (the upper part of limb is extremely underdeveloped or missing and lower part is directly attached to the trunk"
    - Contracture
    - Joint swelling

- Also look for any:
    - Skin changes or rash like erythema or trophic skin changes
    - Muscle wasting

2. Feel

Enquire first if any part is painfull and what exacerbates pain.

Then feel for

1) **Move** to assess Temperature
2) Tenderness (watch expression)
3) Enlargement
4) Effusion
5) Synovial thickening
6) Bony enlargment

3. Move to assess

Move to assess active before passive movements

1) Range of active movements (normal or limited)
    a. Range of passive movements (normal or limited)
2) Is movement painful
3) Is movement accompanied by crepitus
4) Is there any fixed deformaity: when joint cannot be placed in the neutral anatomical position due to inability of movement or laxity of joint ligaments.
5) State of peripheral circulation

4. Measure

Limb circumference for muscle wasting

- 5-10 cm above tibial tuberosity
- 5-10 cm below tibial tuberosity

Limb length for length discrepancy

- True leg length: from anterior superior iliac spine to medial maleolus
- Apparent leg length: from umbilicus (or pubics symphysis) to medial maleolus

Causes of true leg length discrepancy

1) Undetected CDH
2) Previous trauma/bone surgery
3) Increased limb growth – arthritis of the knee
4) Ollier's disease
5) Osteogenesis imperfecta
    - Blue sclera
    - Hyperextensible joints
6) Ployostotic fibrous dysplasia
    - Café a lait spots
    - Sexual precosity
7) Causes of apparent leg length discrepancy
    - Severe hemiparesis
    - Adductor spam causing pelvic tilt

GALS SCREENING

1. Gait

- Watch patient walking in straight line
    - Bare foot
    - In shoes
- Walking fast or running will unmask any abnormality
- Evaluate from, behind, front and the side
    - Foot, Ankle, Knee, Hip, Pelvis, Spine
- Note for limp or abnormal gait
    - Pain – antalgic gait – dot dash
    - Structural – limb length discrepancy
    - Weakness
    - Trendlengurg gait – Duchenne sign
    - Drop foot – common peronial nerve palsy – the gait is high stepping to lift the drop foot
- Note for increased tone
    - UMNL – Crouch gait
    - CP
    - CVA

2. Arms

Advise the patient to:

1) Put your hands behind your head. Note elbows going backward. In frozen shoulder or rotators cuff problems there will be painful abduction
2) Bend arms & touch shoulders: note elbow flexion
3) Straight arms: extension at 180 degree

4) Prayer sign: assess wrist 90 degree extension
5) Reverse prayer sign: assess wrists for 90 degree flexion
6) Clinch fists and open hands flat: observe any abnormality in wrists and hands
7) Squeez your index and middle finger: note strength of power grip
8) Touch each finger tip with your thumb; note precision grip or if any problems in co-ordination or concentration
9) Assess pronation & supination for radio-ulner joint
10) Squeez patient's metacarpal heads and note for tenderness like in rheumatic arthritis

3. Legs

- Examine hip joints: stabilise patient's pelvis and passively test abduction, adduction, flexion, internal & external rotation with hip & knee at 90^{o}
- In newborn look for congenital hip dislocation (CDH) by Barlows & Ortolani maneuvers
- Examine knee joint for
    1) Flexion and extension
    2) Bow legs or genu varum
    3) Knock knees or genu valgum
    4) Palpate antheses at 10, 2, 6 oclock positions and patella and synovial thickening or effusion
    5) Buldge sign for effusion
    6) Patellar tap
    7) Check knee stability - Drawer sign for anterior cruciate ligamants and posterior cruciate ligaments
- Examine the ankle joint
    1) Tibiotalor joint for dorsi flexion and planter flexion
    2) Subtalor joint for inversion and eversion
    3) Midtarsal for medial and lateral movement of foot
    4) Metatarsal joints
- Note any abnormalities like
    1) Flat feet: benign, failure of arch to develop in collagen disorders and cerebral palsy
    2) High arched feet in spina bifida, Friedreich ataxia, Charcot-Marie-Tooth disease
    3) In-toeing or out toeing
    4) Toe walking
    5) Talipes EV
    6) Syndactyly – 2^{nd} and 3^{rd} toes
    7) Overlapping of 3^{rd}, 4^{th}, and 5^{th} toes

4. Spine: Neck, Cervical, Thoraco–lumber Spine

Watch child standing and then bending forward: note for scoliosis, kyphosis or kyphoscoliosis. Palpate for tendeness

Neck

1) Flexion, extension, lateral rotation, lateral flexion
2) Common abnormality noted is Klippel Feil syndrome in which there is low hair line and fusion of cervical spine vertibrae or hemivertibrae resulting in short neck
3) Webbing of skin of neck seen in Turner's syndrome
4) Torticollis
5) Cervical spina bifida
6) Thoracic hemivertebra
7) Hypoplasia of shoulder girdle muscles
8) Cervical rib
9) Temporomandibular joint: open mouth as wide as possible and palpate over joint for crepitus
10) Sprengel's shoulder: one scapula fixed high due to failure to descent during fetal development resulting in reduced abduction and bracheal plexus compression

Cervical spine: Check range of movements (ROM)

1) Flexion – chin touches chest
2) Extension – head touches back
3) Rotation – turning chin to be in line with shoulders 80 degree
4) Lateral flexion – era to shoulder 40 degree

Thoracolumber spine: check for

1) Flexion – able to touch toes
2) Extension – arching back 30 degree
3) Lateral bending – 50 degree to each side
4) Lateral rotation with child sitting – 30 degree at each side

NORMAL RANGE OF MOVEMENTS

Wrist				
Flexion	80 ^{o}	Extension		70 ^{o}
Radial deviation	20 ^{o}	Ulnar deviation		30 ^{o}
MCP				
Flexion	90 ^{o}	Extension		30 ^{o}
PIP				
Flexion	100 ^{o}			
DIP				
Flexion	90 ^{o}	Extension		10 ^{o}
Elbows				
Flexion	135 ^{o}	Extension		0-10 ^{o}
Supination	90 ^{o}	Pronation		90 ^{o}
Knees				
Flexion	135 ^{o}	Extension	up to	10 ^{o}
Hips				
Internal rotation	35 ^{o}	Extension		35^{o} (hips flexed 9^{o})
Abduction	50 ^{o}	Adduction		30 ^{o}
Ankles				
Planter flexion	50 ^{o}	Ext dorsiflexion		20 ^{o}
Subtalar joint				
Inversion	50 ^{o}	Eversion		5 ^{o}
Mid tarsal joints				
Abduction	10 ^{o}	Adduction		20 ^{o}
First metatarsophalangeal joint				
Planter flexion	45 ^{o}	Ext dorsiflexion		70 ^{o}

Note any crepitus: pain on moving the 1st MTJ may be selectively involved in spondyloarthropathies

TESTS TO ASSESS CONGENITAL HIP DISLOCATION IN NEONATES

Be gentle in performing these tests as the undue force can cause avascular necrosis of the head. Following are high risk babies with congenital dysplasia

1. Breech deliveries
2. Family history of hip dysplasia
3. Preterm babies

Ortolani test

Baby should lie supine. Now flex the hips and knees at 90 degrees and abduct the legs until each knee touches the examining couch. If there is congenital hip dislocation, a clunk can be felt and it can be audible as the femoral head slips in the acetabulum. This test is difficult to elicit after neonatal period.

Barlow's test

Is the hip is not fully dislocated but is noted o be unstable then this can be diagnosed by pressing the femur (90 degree flexed at the hip join) backwards and outwards. Keep the thumb over the lesser trochanter medially and it is possible to feel the femoral head sliding over the posterior acetabular rim. When the pressure is exerted with the middle finger over the greater trochanter, it can be felt how the femoral head slips back into the acetabulum.

Work out your differential diagnosis for mono & polyarthritis in children

At completion of examination summarise positive findings

1) No of joints involved
2) Symmetry: symmetrical in JRA and asymmetrical in psoriatic
3) Activity of disease: active with pain and redness
4) The functional severity
5) Differential diagnosis

CAUSES OF WEAKNESS AND HYPOTONIA

1) Muscles (myopathy, myositis, muscle dystrophy, steroids use)
2) Neuromuscular junction disorders (Myasthenia, organophosphate poisoning, botulism)
3) Central nervous system (Poliomyelitis, Guillain-Barre Syndrome)
4) Spinal cord: tumours, gibbus formation
5) Electrolyte disturbances: Hypokalaemia, hyponatraemia, hypoglycaemia, hypocalcaemia
6) Hypothyroidism or lax ligaments

TUTORIAL 12

EXAMINATION OF ENDOCRINE SYSTEM

OVERALL OBJECTIVES

At the end of this module, the student should be able to do a full clinical examination of endocrine system and to be able to make a reasonable differential diagnosis of children with common endocrine problems like short or tall stature, and hypo or hyper functioning of pituitary and thyroid glands.

Extra general features

Hyperpigmented skin

Child with short or tall stature

Pot bally, protrude tongue

Coarse skin

Polyuria

CHECK IF THE CHILD IS OF ABNORMAL STATURE

1. Short (achondroplasia/ hypothyroidism)
2. Tall (Marfan's syndrome)

EXAMINE THE CHILD

- Measure child's height
- Measure lower segment (LS) → pubic symphysis to ground
- Calculate upper segment (US) → by subtracting the LS from the total height
- Calculate US/LS ratio

Normal values

- at birth 1.7
- 3 yrs 1.3
- 8 yrs 1
- 18 yrs 0.9

IF THE CHILD IS SHORT

If:		
US/LS ratio ↑	Short lower limbs	skeletal dysplasia, hypothyroidism
US/LS ratio ↓	Short trunk	vertebral radiation, scoliosis
	Short neck	Klipple-Feil sequence

Measure arm span and subtract from it the total height

Calculate AS–H

Normal values are as follows:

- From birth to 7 yrs - 3 cm
- From 8-12 yrs 0 cm
- At 14 yrs
  - Boys +4 cm
  - Girls +1 cm

If:		
AS – H = < N	and US/LS ↑	Short limbs / normal trunk
AS – H = > N	and US/LS ↓	Normal limbs / short trunk
AS – H = < N	and US/LS ↓ or N	Short arms / short trunk

IF THE CHILD IS TALL

If:

US/LS ↓	Lower limbs are disproportionately long
	Marfanoid habitus, eunuchoid habitus
US/LS N	This is more in keeping with pituitary gigantism
	Familial short stature
AS – H ↑	Upper limbs are disproportionately long
	Marfanoid habitus, eunuchoid habitus
AS/H > 1.05	Suggestive of Marfan syndrome

TUTORIAL 13

EXAMINATION OF RENAL SYSTEM

OVERALL OBJECTIVE

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

1) Recognise by appropriate history and examination and investigate and manage common and life-threatening problems of the kidney and urinary tract in infants and children
2) Formulate a likely differential diagnosis and treat appropriately the oedema, oligo/anuria, haematuria, proteinuria, dysuria and hypertension
3) List complications and principles of management of:
    - Glomerulonephritis
    - Nephrotic syndrome
    - Urinary tract infection
    - Acute kidney disease
    - Chronic kidney disease
    - Obstructive uropathy
    - Hypertension

IMPORTANT ADDITIONAL POINTS TO ADD WHEN EXAMINING A CHILD WITH RENAL PROBLEM

1) Assess patient's mental status: for encephalopathy, due to uraemia, or aluminium toxicity or depression due to chronic illness
2) Assess for sever malformation syndromes: check for abnormalities in genitourinary system
3) Look for evidence of rickets, hypertension, ascites
4) Hemihypertrophy may indicated Wilim's tumour
5) Pallor is a sign of anaemia that can be due to chronic kidney disease or haemolytic uraemic syndrome
6) Note periorbital or peripheral oedema in cases of nephrotic syndrome or post streptococcal glomerulonephritis
7) Presence of jaundice may indicate diagnosis of hepatorenal syndrome
8) Bruising, uremic frost, scratch marks (pruritus) may indicate chronic kidney disease
9) Presence of hirsutism may indicate use of steroids or cyclosporine
10) Cushingoid features may indicate use of steroids for transplants or nephrotic syndrome

TUTORIAL 14

EXAMINATION OF HAEMATOLOGICAL SYSTEM

OVERALL OBJECTIVES

At the end of this module, student should be able to

1) Recognise and manage the common haematological problems of neonates, infancy and childhood.
2) Interpret the FBC and state the changes that occur from birth to puberty
3) Recognise by appropriate history taking, clinical examination and relevant laboratory investigations, and approach to the following anaemias:
   - Iron deficiency
   - Folate deficiency
   - B12 deficiency
   - Anaemia of chronic disease
   - Anaemia infection
   - Aplastic anaemia
   - Haemolytic anaemias
4) Tabulate important aspects in prevention and management of above anaemias.
5) **Bleeding disorders / purpura**
   - State and recognise clinically the difference in the clinical presentation of vascular vs platelet vs co-agulation defects
   - Identify important aspects in the history (including patterns of inheritance) of a patient with a bleeding disorder
   - Tabulate important aspects in the aetiology, pathology and management of thrombocytopenia (ITP), haemophilia, A, B, DIC, Henoch Schonlein purpura and haemorrhagic disease of the newborn.
   - Interpret the basic screening tests and their application in the approach to a patient with a bleeding disorder.
6) **Malignancy**
   - Recognise the presenting features (clinical + laboratory) of leukaemia + lymphoma in childhood
   - State the differential diagnosis of above
   - State the steps in the management of these patients
   - Counsel the parents on effects of malignancy (including prognosis) and of cytotoxic therapy
7) **Component therapy**

   List indications for and complications of blood component therapy
8) **Ethics**
   - Malignancy
   - Cytotoxic use

CONSIDER HAEMATOLOGICAL SYSTEM INVOLVEMENT IF FOLLOWING HAS BEEN FOUND IN ABOVE EXAMINATION

- **Pallor:** long list of anaemias: microcytic, macrocytic, normocytic (low Retics, high Retics)
- **Petechiae:** purpura, ecchymosis: ALL, AML, aplastic anaemia, ITP
- **Pigmentation:** thalassaemia
- **Eczema:** Wiskott-Aldrich (with ear discharge)
- Chronic lung disease stigmata
- Joint swelling:
    - Haemophilia
    - Henoch-Schonlein purpura
    - Sickle cell anaemia, leukaemia
    - Irritable bowel disease
    - Juvenile idiopathic arthritis
- **Squint, nystagmus:** Fanconi's anaemia, 6th nerve palsy with IC bleed
- **Ptosis:** Fanconi's anaemia
- **Ataxic gait:** Vit B 12 def
- **Hemiplegia:** haemophilia with IC bleed, sickle cell anaemia with cerebral sickling
- Splenomegaly
    - Haemoglobinopathies
    - Malignancy
    - Infection – SBE
    - Osteopetrosis
    - Storage diseases
    - Congenital spherocytosis
- **Hepatomegaly:** as above, plus
    - Hepatitis
    - Wilson's disease (decrease in clotting factors 1, 2, 5)
- **Delay in Tanner staging:** Thalassaemia, sickle cell anaemia
- **Posterior iliac crest tenderness, scar:** bone marrow aspiration

THEN THINK SPECIFICALLY ABOUT SEX, RACE AND SYNDROMES

- Blackfan-Diamond red cell aplasia
- Fanconi's anaemia
- Thrombocytopenia absent radius
- Wiskott-Aldrich syndrome
- Dyskeratosis congenita

- Triade may become apparent in 1st 10 yrs of life (XLR 85%, AD & AR 15%)
- Reticulate lacy pigmentation of upper body
- Mucosal leukoplakia
- Nail dystrophy
- Swaschman-Diamond syndrome
- Pearson syndrome
    - Refractory sideroblastic anaemia
    - Cytoplasmic vacuolization bone marrow precursors
    - Metabolic acidosis
    - Mitochondrial DNA mutation
    - Exocrine pancreatic insufficiency

THE ASSESSMENT

THIS IS VITAL PART OF YOUR EXAMINATION SO PRESENT IT PROPERLY

Include all problems that have been identified but do not simply list all positive findings.

Assess the child and do not only summarize

For example:

"**My assessment of Jacob is that he is a 4-year-old little boy** who has severe acute malnutrition with oedema. He has chronic suppurative lung disease as evidenced by abnormally shaped chest, clubbing and cough productive of purulent sputum. He also has evidence of Cor-pulmonale on the basis of having epigastric pulsation, a LPSH and a loud P2. He also has generalized lymphadenopathy".

"**Most likely causes of these problems in children are** HIV infection with CLD following recurrent or severe infections. He does not have parotid enlargement but I think it is still important to consider LIP. Other important considerations would include TB as it is important to remember that not everything is due to HIV and to consider other causes like cystic fibrosis, which although uncommon, do occur in black children."

"My approach to manage this child would be":

First, relevant history including the HIV status, TB contacts, any severe/recurrent ARI's and previous pulmonary TB. The relevant investigations would include:

1. HIV status
2. CXR – "I would look for changes suggestive of TB or LIP and most importantly it would be useful to review old CXRs". Lateral view CXR – look for Donut Sign (Cape Town Sign)
3. Mantoux and AFB's
4. CD4% if HIV infected and a review of social circumstances to start ARVs
5. FBC looking for evidence of acute infection (WBC) and the Hb to determine if plethoric from long term hypoxia or anaemia
6. ESR nonspecific but helpful for diagnostic and prognostic purpose
7. Procalcitonin, c reactive protein and albumin will give one an idea of the inflammation

MANAGEMENT

Depends on the diagnosis made

The longer you talk sensibly, the better you will do. To do well, you need to efficiently examine the child so that there is time for your assessment and discussion!

Time is of essence. Good luck and all the best!

TUTORIAL 15

INFANT FEEDING

1. BREAST FEEDING

Physiology:

During pregnancy the glandular tissue in the breasts increases under the influence of the pituitary hormone prolactin as well as oestrogen and progesterone. They prepare the breasts for later work of lactation

Colostrum

Before birth a very small quantity of colostrum is secreted. For the first few days after delivery it alone is secreted and gradually replaced by mature milk. It contains a very high concentration of Immunoglobulins and Immunologically active cells such as macrophages and lymphocytes. It is an important means of providing the newborn baby with passive immunity. The small volume corresponds with the neonate's low fluid requirements in the first few days of life.

Mature milk

Consists of two main portions; the Foremilk which is produced in the early part of a feed. It is relatively dilute containing less protein and fat than the creamy Hindmilk produced later in the feed. Babies are able to regulate the dilution of the milk they drink by varying the proportions of Fore-and Hindmilk. It is incorrect to think that the Foremilk which looks rather bluish and watery is not suitable for feeding.

Prolactin

Between feeds milk is secreted by the milk producing glands in the breasts in preparation for the next feed. The quality of milk produced depends on the amount of prolactin in the circulation. Prolactin secretion is increased by:

- **Sucking by the by the baby:** the more minutes per day of sucking the more prolactin is produced. This is why it is important not to reduce suckling by complementary bottles.
- **Sleep:** rest is important
- **Lack of Mental Stress:** A worried, tense mother will secrete less prolactin than a comfortable and relaxed mother.

Oxytocin

The release to the baby from the storage ducts of milk that has been secreted is controlled by another pituitary hormone called Oxytocin.

Sucking is the most powerful stimulus to its secretion but even the sight or sound of the baby is effective. The action of oxytocin on the breast is to cause contraction myoepithelial cells (resembling muscle fibres) surrounding the breast ducts resulting in ejection of milk. Oxytocin does not cause milk to be made; it only affects its release.

Nipples:

For satisfactory breast feeding it is essential that the baby is able to get the nipple and surrounding areola into its mouth. The nipples should be checked during the antenatal

period and if found inverted or flat, Hoffman's exercises should be taught to the mother. In these exercises the nipples are drawn out and rolled between the forefinger and the thumb. The wearing of a nipple shield which helps to protrude nipple is often used.

THE ADVANTAGES OF BREAST FEEDING

1) **Protection against infection:** as the breast milk is sterile and contains both immunoglobulins and immune cells, it provides protection against viral and bacterial infections, which enter the intestine via oral route as in gastroenteritis.
2) **Protection against allergy:** the fact that human milk protein is less 'foreign' than cow's milk protein accounts for the much lower incidence of allergic disorders in breast fed babies.
3) **Protection against Malnutrition:** Breast milk is always available when the baby needs it, even when the family finances are low. It cannot be diluted as is the case so often with the artificial milk. Breast feeding remains a protection even in the second year of life as it will provide an important protein supplement to a staple carbohydrate **food such as mealie meal or samp.**
4) **Promotion of mother-infant bonding:** Breast feeding brings a mother and the baby very close together, especially in the early weeks when the process of bonding is at **its most delicate.**
5) **Protection against breast cancer:** women who have breast fed babies for a good length of time have a reduced incidence of breast cancer.
6) **Uterine resolution:** the oxytocin secreted during breast feeding has a powerful **effect on the uterus causing it to contract and undergo involution more rapidly.**
7) **Convenience:** Breast milk is always available at the right strength and temperature not needing preparation or sterilisation.

MEANS OF PROMOTING BREAST FEEDING

1) **Early contact:** newborn babies and their mothers are alert and seek each other in the first hour after a normal delivery. They should be allowed to have maximum contact in that period. Routine procedures that keep them apart (such as bathing) should be delayed for at least one hour. It has been shown that increased contact in this period leads to increased length of breast feeding as well as superior mother-infant relationships.
2) **Avoid supplementary/complementary feeds:** during the first few days the normal term newborn has a low fluid requirement. This means that the rather small volumes of colostrum/milk that are produced are enough. Providing extra artificial milk or water is usually not necessary and serves to reduce the production of breast milk. Also, it seems that it is easier for a baby to obtain milk by sucking on rubber teat than from the mother's nipple. So once a baby has experienced a teat it may object to the nipple. It is thus best to keep teats way from breast feeding babies.
3) **Mother's nutrition:** for good lactation it is essential that the mother takes an adequate balanced diet and extra fluids.
4) **Rest and relaxation:** it is very helpful if a mother can take a regular rest period during the day and get adequate sleep at night. Many mothers find it more restful and satisfying to have the baby sleep in bed with them. The dangers of overlying have been greatly over emphasised in the past.

5) **Prolactin stimulators:** certain medications have the effect of stimulating the hypothalamus to secrete prolactin. These may be used to re-stimulate a failing milk supply. Their use must always be accompanied by good diet, extra fluids, and as much sucking on the nipple by the baby as possible. These medications are Maxolone (metoclopramide), Eglonyl (sulpiride) and must be prescribed by the Doctor.

COMMON MISCONCEPTIONS ABOUT BREAST FEEDING

1) **The working mother – cannot breast feed a baby:** This is not so. If the baby is artificially fed by a caretaker during the working day, the mother can breast feed during the evening, night and early morning. Lactation is especially well maintained if the baby sleeps with the mother. Many nurses use this scheme very successfully with their own babies.
2) **Small breasts – cannot produce enough milk:** This is not so as much of the contour of a breast is made up of adipose tissue. Small breasts produce quite as much as larger ones.
3) **A short period of breastfeeding is not worth the trouble:** this is not so. Many of the positive advantages of breastfeeding are of particular benefit to the very young infant. These include the anti-infective and antiallergic effects. Even a period as short as 2-3 weeks can be a lasting benefit to the baby.

COMMON PROBLEMS OF BREAST FEEDING

1) **Breast engorgement:** the breast become enlarged, painful and even oedematous on the 2^{nd}-5^{th} day after delivery. The milk flow is poor and mastitis is often wrongly diagnosed. The correct management is to empty the breasts by manual expression or with a breast pump and then to encourage the infant to suck as much as possible
2) **Mastitis:** Staphylococci may enter the breast either via the milk ducts, Montgomery's tubercles or an abrasion in the nipple. The incomplete emptying of the breasts always makes infection more likely and should be avoided. Mastitis is not a reason to stop breastfeeding. The baby should feed from unaffected breast whilst the infected breast is expressed. If the mastitis is at an early stage treatment with Penicillin or Cloxacillin may be adequate. If an abscess has already formed the patient needs hospital referral for incision and drainage. In either case once mastitis is improved, feeding from that breast may be resumed.
3) **Painful or cracked nipples:** this condition may be due to a variety of causes including; flat nipples, engorged breasts, and tugging by the baby before fixing to the nipple has occurred. Do not advise stopping breast feeding. The mother may need to express her milk for 24-48 hours to give them a chance to recover. An emollient cream such as Vandol or lanolin cream short spells of exposure of sunlight and the treatment of candida infection if present may all help.

Inadequate lactation: the main causes of this are;

1) Use of 'sup' or 'cump' artificial feeds
2) Missing out feeds
3) Anxiety
4) Fatigue and other emotional problems

5) Certain contraceptive pills

MANAGEMENT INCLUDES

1) Ensuing adequate fluid intake and diet
2) Frequent suckling
3) Prolactin stimulators such as Maxolon and Eglonyl

2. ARTIFICIAL FEEDING

Choice of Milk:

0-3 months

Use a humanise formula such as NAN or S26. The main advantage of these milks is that they present a lower solute load to the immature kidneys. Unlike breast milk however, they do not provide protection against infection or allergy

4-6 months

Use a full cream or dried milk e.g. Lactogen, Nespray, Klim, Babymilk Plus.

6-12 Months

a) Skim milk: this is prepared from milk that has had most of the cream (fat) removed. This results in a powder that is high in protein and lactose. If used to provide the same number of calories as full cream milk, the high lactose content may lead to lose stools in some infants – especially in infants with reduced lactose tolerance, such as those with sever acute malnutrition or chronic diarrhoea. It should not be used as routine milk feed for infants less than 9 months of age. Its main use is a high protein supplement to solid foods. For example: skim milk powder sprinkled onto porridge makes a well-balanced energy and protein food.
b) Soya based milk: like Isomil, Infasoya, Sobes, and Mullsoy. These milks all contain soya protein and are lactose free. They are prescribed in cases of lactose intolerance or allergy to cow's milk and have sucrose as their sugar.
c) Special milks for chronic Diarrhoea or Malabsorption e.g. Nutramigen, AL110, Alfare. These are used for specific conditions and should only be prescribed at specialist level.
d) Bottle hygiene in all cases. The bottle should be thoroughly washed with soap or detergent. A bottle brush is of great assistance, teats should be washed and rubbed with salt and both the bottle and the teats are boiled in a pot of water for at least 15 minutes and allow cooling down before filling. Or **prepare a solution of diluted Jik** and leave the bottles and teats submerged for 10 minutes or until the next feed. It is not necessary to rinse the bottle before filling it with milk. Teats perish very quickly when boiled so rather use this method.
e) **To prepare the Jik solution:** place 2 litres of clean water in a suitable container and add 1 table spoon of un-perfumed Jik. This solution should be changed daily.
f) Note:
    - If a fridge is available for storage it is fine to make up a whole day's feeds at once.

- Otherwise make only one feed at a time as the milk is breeding ground for bacteria.
- Throw away the unfinished feed for the same reason.

3. MIXING FEEDS

Use clean cooled water

- Use 1 scoop (as provided by the manufacturer) per 25 ml of water. Avoid heaping or packing the powder as this will produce an over concentrated feed and may lead to salt overload and hypernatraemia.
- The most dangerous error is to prepare a dilute feed as the baby then receives insufficient energy and protein with the result that growth is slowed down leading before long to sever acute malnutrition.
- Volume of feeds: after first 5 days of life 150 ml/kg/24 hrs is a rough guide. The best guide is the baby's weight gain. Adequate weight gain is about 25g per day in the first 4 months and 500g per month from 5-12 months of age.

How many feeds per day?

- Under 6 months: not less than 5
- 6-12 months: not less than 3

Feeding of solids to older infants and preschool children

Food is needed in the body for three main purposes:

The energy requirements of the baby which include the maintaining of body temperature, the work done by muscles including the heart, the carrying out of the many, different chemical reactions involved in the body's metabolism and for many other purposes.

The foods providing most of the body's energy are carbohydrates, (4Kcal/g), and fats (9 Kcal/g): 1 kilojoule = 4.2 Kcal). Proteins are able to provide energy if no other source is available.

The repair of tissues and cells that normally "wear out" i.e. the hair, skin, blood cells, and cells of small intestinal villi etc. this process requires both energy and protein.

The growth of the child and adolescent: this requires both energy and protein. If a child is not getting enough to eat, the first of these processes to suffer will be growth which slows down and in severe cases even stops completely. If the food intake is even more decreased the processes of repair will cease. The skin will become thin, the number of red cells will decline and the lining of the small intestine will become flattened. This stage is typically seen sever acute malnutrition with oedema. The most severe case of all is when the energy supply falls. In this situation the child's activity is reduced, the body temperature falls and death is very close.

Food value of some common foods:

Energy containing foods:

Carbohydrates containing foods:

Mealie meal Samp

Rice Sugar

Potatoes

Fat containing foods such as:

Margarine Cooking oil

Lard Dripping

Peanut butter

Protein containing foods

More expensive

Milk Skim powder

Meat Fish

Eggs

Less expensive

Beans Nuts

Protective foods

These contain vitamins and minerals but generally very little in the way of protein or energy. Some examples:

Green vegetables	yellow vegetables
Spinach	Squash
Cabbage	Carrots
Cauliflower	Pumpkin

Points about individual food stuffs

A mealie seed consists of a large upper sac below which is a small protein rich germ bud. When the seeds are ground coarsely and the meal is left unsifted a useful quantity of protein remains in the meal. The highly relined and sifted meal usually sold in the shops has led almost all the germ bud removed and thus has very low protein content.

Samp:

This is almost pure starch. It requires prolonged cooking, the fuel for which must be added to its cost.

Beans

These are rich in both energy and protein and are one of the most economical sources of protein.

Instant cereals

Nestum, Pronutro, Cerelac etc. are all scientifically prepared cooked foods of excellent nutrients. Their major drawback is cost.

An outline of ideal feeding of preschool children

0-4 months

Breast milk is the ideal

If necessary artificial milk is used

Vitamins: for breast fed babies and babies fed on artificial milks, which do not contain added vitamins, it is important to recommend a multivitamin preparation which contains vitamin D 400 IU, Vitamin C 50 mg, Vitamin A 5000 IU in daily doses.

4-9 months

Start at 4 months with a simple porridge. Later add vegetables one at a time giving the baby time to get used to new tastes and textures. The quantity is gradually increased. The milk feeds continue as before.

9 months to 4 year

600 ml of breast, artificial or fresh cow's milk per day is required throughout this period. In addition, 3-4 good sized solid food meals are given. The small stomach size of the young child makes it necessary to give feeds frequently in this way. The most economical way to provide balanced meals at this age is basic porridge such as mealie meal with small amounts of beans or skim milk powder which are rich in protein, added. Finally add a small quantity of fat or oil.

Some examples are:

- Mealie meal + beans + cooking oil
- Samp + skim milk

The six golden rules of good nutrition for young children are:

1. Breast feed for 2 years of age
2. Start porridge at 6 months, not sooner
3. Add protein and fat/oil to the porridge
4. Give infants and toddlers 4 good-sized meals a day
5. Give protective foods to children over 6 months
6. A sick child needs to be fed as well as any other child

THE END

For comments & suggestions: ghuman@telkomsa.net