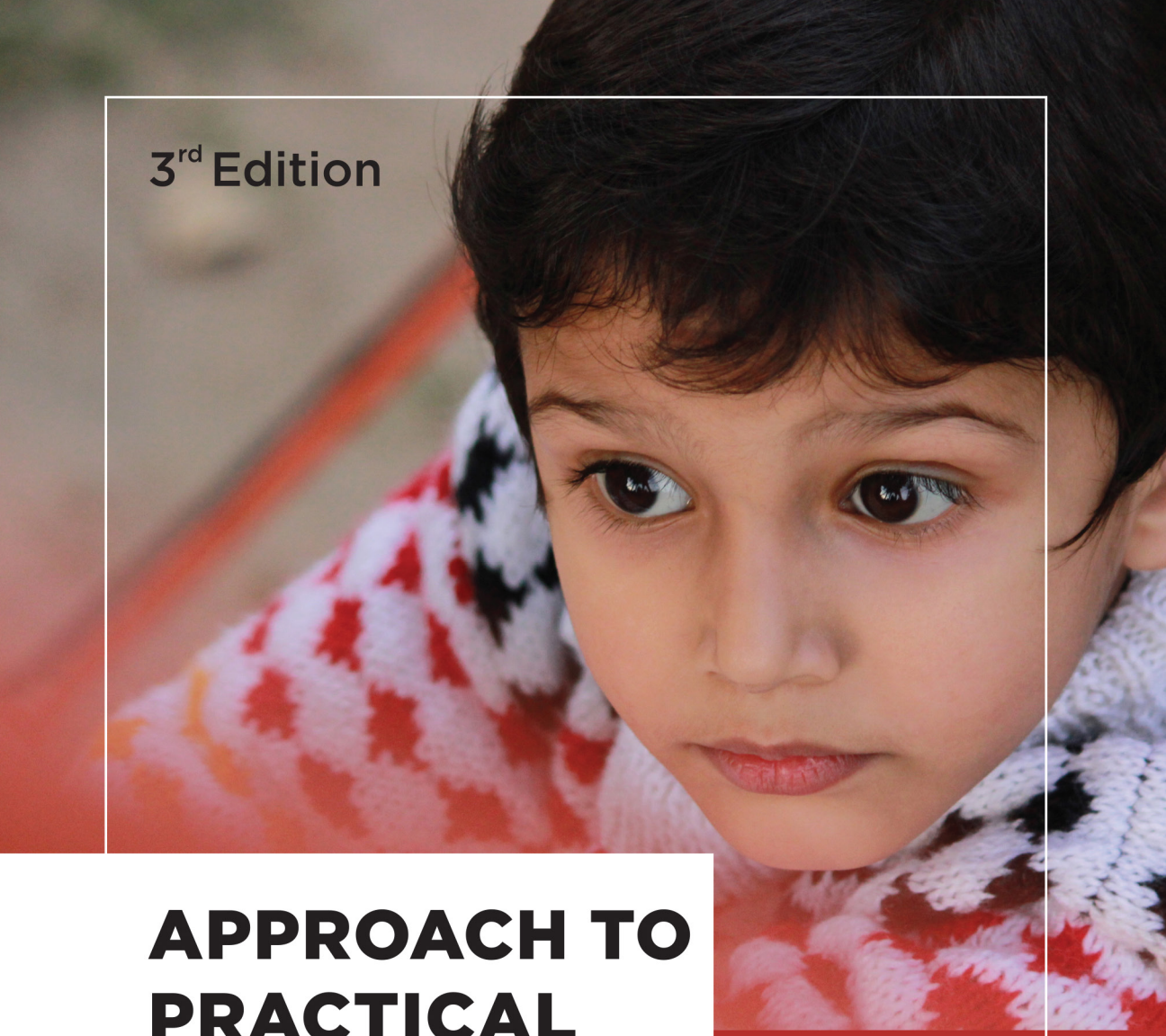

3rd Edition

APPROACH TO PRACTICAL PEDIATRICS

**Long Cases, Short Cases,
Neonatal Resuscitation,
Instruments and X-Rays
(As per Competency Based Curriculum)**

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Instruments

Bone-Marrow Aspiration Needle

Bone marrow examination is pathologic analysis of bone marrow samples obtained by bone marrow aspiration and bone marrow biopsy (trephine biopsy).

Parts

- Stillete
- Thick body with nail
- Guard 2 cm from the tip (guard prevents through and through penetration of the bone)

Uses

Bone marrow aspiration

Indications

- Diagnostic:
  - Investigation for unexplained abnormal red blood cells (megaloblastic anemia)
  - Staging and follow-up of malignancies (acute and chronic leukemias, lymphoms, myelodyplastic syndrome and myeloproliferative disorders)
  - Investigation of abnormal peripheral smear morphology
  - Infection e.g. kala azar
  - Pyrexia of unknown origin
- Therapeutic:
  - Bone-marrow transplantation

Contraindications

- Coagulation disorders like hemophilia, disseminated intravascular coagulation
- Infection at aspiration area

Complications

- Infection
- Bleeding
- Cardiac injury (if deep penetration occurs in sternal aspiration)

Sites

- Posterior iliac crest (both aspiration and biopsy)
- Upper 1/3rd of medial aspect of tibia (in children <2 years of age)

Procedure

Prior to the procedure

- Obtain informed consent for
- Investigate for prothrombin time, partial thromboplastin time, platelet count and blood group

Aspiration is generally done from the posterior superior iliac spine

- The patient is placed in the prone position

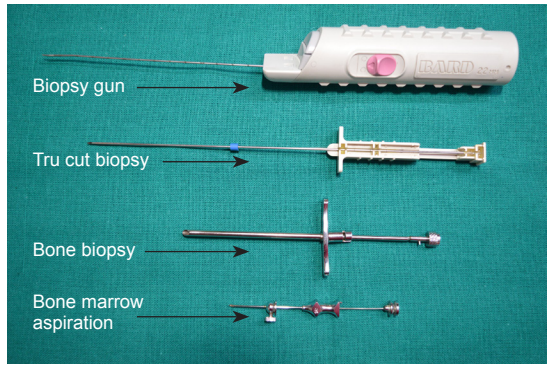

Fig.1.42: Biopsy gun, tru cut biopsy, bone biopsy and bone marrow aspiration.

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Neonatal Resuscitation

Chest Compressions

Indication of Chest Compressions

Chest compressions are initiated if after 30 seconds of effective PPV, the heart rate remains below 60 bpm.

Rationale

In babies with heart rate below 60 bpm despite PPV, the oxygen level drops to cause acidosis and myocardial dysfunction. Chest compressions supplements mechanical ability of heart to maintain circulation till the time myocardium is oxygenated to provide adequate function and deliver oxygen to the brain.

Note:

- Baby is firmly supported in back
- Neck is slightly extended
- Compressions should
  location, depth
- Chest
  always be
  ilation with 100%

niques

Two techniques have been described:

- *Two-thumb technique*: Compression with 2 thumbs with the fingers encircling the chest and supporting the back [preferred method] or
- *Two-finger technique*: Compression with 2 fingers with a second hand supporting the back. Two finger technique is no longer recommended

Thumb technique [Fig.3.14]

WHY: The 2-thumb technique generates higher blood pressures and coronary perfusion pressure with less rescuer fatigue. The 2-thumb technique can be continued from the head of the bed while the umbilicus is accessed for insertion of an umbilical catheter.

- Positioning of thumb or site of compression: It is done in lower third of sternum in midline. The area to be compressed lies between a line drawn between nipples and the xiphoid. This can also be located by running fingers along costal margin and localizing the xiphoid and placing the fingers above xiphoid **[Fig.3.15]**. Thumbs can be placed side by side or in
  baby one above another. Thum
  flexed at the first joint
  vertically to pre
  and spi
  of anterio-posterior
  .3.16]
  Duration of downward stroke < duration of release
  - Thumb and fingers should remain in contact with chest all the time

Method of chest compression and how is it coordinated with PPV?

- Positive pressure ventilation should always be

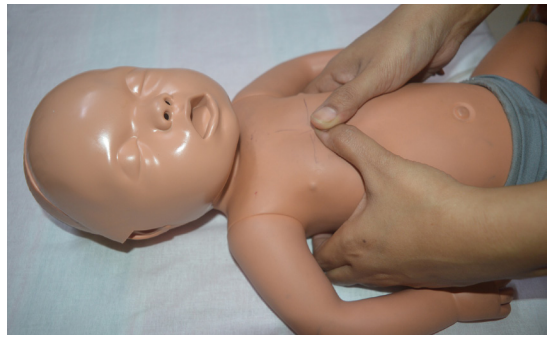

Fig.3.14: Chest compression technique.

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Severe Acute Malnutrition (SAM)

History

Preparation for History

- Introduce yourself to the patient
- Check the patient is not in any pain or respiratory distress (ask for oxygen if patient has visible respiratory distress)

Introduction of the Patient

- Name
- Age
- Sex
- Resident of
- Informant is ..... who is reliable and educated up to .....

Chief Complaints

- Poor weight g
- Swe
- ..rity in respiration, diarrhea

..ry of Present Illness

The typical history can be described as

"Sonu, a 4 year old boy presented to emergency department with loose motions and vomiting for last 2 days. The child had 10-12 watery stools over the previous 24 hours, during which he became quite irritable, crying a lot, and drinking half his usual amount of liquids. Additionally, he had several episodes of non-bloody, non-bilious, non-projectile vomiting. His mother denies any episodes of fever, night sweats, chills or bleeding episodes. For the past few months, Sonu's mother has been feeding him a thin dalia (porridge), but in the last one month he has not been eating well. He has become miserable and irritable, and prefers to be left alone, not moving at all unless his mother carries him. Three days back, mother became worried because his stomach was distended, and gave him medicine brought from local doctor. That night Sonu passed three loose stools and was restless. He drank the water quickly that his mother gave him and then vomited three times. Subsequently, mother brought Sonu to the hospital ....."

History of disease

- Fever:
  - ..ocumented)
  - ..attern of fever: Intermittent, remittent, continuous
  - Associated with chills and rigor, sweating, malaise or apathy, loss of appetite, evening rise of temperature
  - Responds to medications
  - History of convulsions associated with fever
  - History of immunization
- Diarrhea:
  - Duration and frequency of diarrhea
  - Number of stools per day
  - Characteristics of stools (bloody, mucus, watery, formed, oily, foul odor)
  - Precipitating factors: Recent travel, antibiotic course, change in diet
  - Urine output (suggests severity of dehydration)

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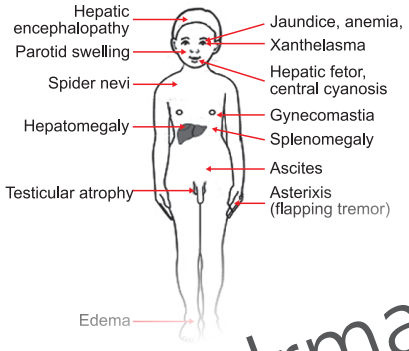
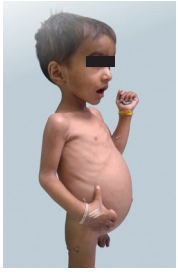
Gastrointestinal System

General Physical Examination

Signs of anemia	General	Pallor, particularly conjunctival, hyperdynamic circulation (bounding pulse, flow murmur)
	Signs of iron, vitamin B_{12} and folate deficiency	Kolionychia (iron deficiency only), atrophic glossitis, angular chelitis, hyperpigmentation over joints (B_{12} deficiency)
	Signs of hemolysis	Icterus, dark urine
	Signs of bleeding	Excessive bruising, petechiae, telangiectasiae or larger vascular malformation
	Signs of malignancy	Muscle wasting, edema, organomegaly, lymphadenopathy, palpable soft tissue masses
Signs of liver cell failure **[Fig.17.1]** 		• Fetor hepaticus (sweetish, slight fecal smell of breath seen in hepatic encephalopathy) • Parotid enlargement • Spider nevi (central arteriole with radiating ves… resembling legs of spider, seen in sup… territory) • Asterixis: Patient is a… with fingers e… will be involv… movements at …rist joints. It is usually seen in …pathy, uraemic encephalopathy, or CO_2
Signs of portal hyperte…		Ascites, splenomegaly, dilated veins over abdomen, caput medusa
	…art failure	Tachypnea, wheezing, crepitations, cyanosis
	Right heart failure	Edema, ascites, hepatomegaly, elevated JVP
…ral status [Fig.17.2] 	Signs of malnutrition	• Hair changes: Hypopigmentation, sparse hair, easily pluckable hair, flag sign • Nail changes: Brittle nails, paronychia, koilonychia, platynychia • Skin changes: Hypopigmentation, hyperpigmentation, desquamation, ulceration
	Vitamin A deficiency	Conjunctival or corneal xerosis, Bitot's spots
	Vitamin B deficiency	Angular stomatitis, cheilosis
	Vitamin D deficiency	Bossing of skull, beading of ribs, wrist enlargement (rickets)
	Vitamin E deficiency	Petechiae, purpura
Others	BCG mark	Look for scar mark in left upper arm
	Skin	Petechial hemorrhages
	Stigmata of tuberculosis	Phylectenular conjunctivitis, scars and sinuses, erythema nodosum

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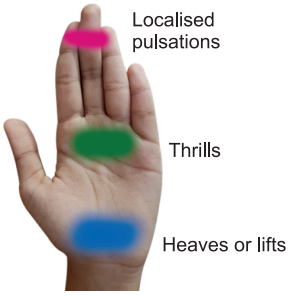
Cardiovascular System

Systemic Examination

Cardiovascular System

Inspection	
Precordium	• Shape and symmetry • Deformity or bulging • Engorged superficial veins • Apical impulse: Apex impulse is lower-most and the outer-most part of the cardiac impulse seen on the precordium. It is normally located in the 4th or 5th intercostal space just medial to the mid-clavicular line
Visible pulsations	Any pulsation present in aortic, pulmonary, parasternal areas, epigastrium, suprasternal area, carotid pulsation, inferior angle of scapula (Suzman's sign in coarctation of aorta)
Back	• Abnormalities of the shape of spine such as a kyphosis, scoliosis gibbus should be noticed • Drooping of the shoulder, winging of scapula
Skin	Look for any sinus, ulcer, venous pro
Palpation	
Apex beat **[Fig.15.4]** 	Maximum upwa ger by the lower-most and the outs Look for: pex beat diffuse beat *Tapping apex beat*: Cardiac impulse just touches the finger and leaves with lifting or without lifting the finger. This is seen in mitral stenosis ◦ *Hyperdynamic apex beat*: Finger will be lifted up less than 2/3rd of the systole i.e. the apex beat is ill-sustained. This is seen in mitral regurgitation and aortic regurgitation ◦ *Heaving apex beat*: Finger will be lifted for more than 2/3rd of the systole. This is seen in aortic stenosis ◦ *Hypodynamic apex beat*: Decreased thrust of the cardiac impulse is felt. This is seen in shock, pleural effusion, pericardial effusion and constrictive pericarditis
Parasternal heave **[Fig.15.5]** 	• Outward movement of the precordium at the left parasternal area felt with the base of the hand • It is seen in right ventricular hypertrophy and massive left atrial enlargement • It is of two types: ◦ Fast ill sustained as seen in right ventricular hypertrophy due to volume overload as in ASD and VSD ◦ Slow sustained as seen in right ventricular hypertrophy due to pressure overload as in pulmonary stenosis • *Grading of parasternal heave* Grade 1: Parasternal heave is visible but not palpable

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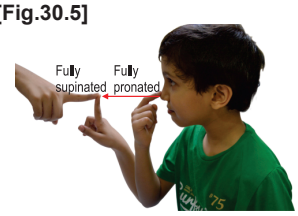
Ataxia

Cerebellar Signs (assessment of ataxia)

Posture	Truncal control **[Fig.30.2]** 	Ask the child to sit on edge of a firm surface. Make him lift his feet from the ground with arms crossed (truncal ataxia). Look if the child can keep balance in this position without support from his extremities **[Fig.30.2]**
Gait	Gait is the posture of the patient during walking (*Decubitus* means posture of the patient in bed). **[Fig.30.3]** 	• Ask the child to walk several steps with natural gait. Next ask the patient to walk heel to toe (tandem walking) **[Fig.30.3]**, then on their toes only, and finally on their heels only. Normally, these maneuvers are possible without difficulty. A child with organic cerebellar disease lean towards the side of the lesion • Note the amount of arm swi… arm swinging is a s… …nity weakness • O… …e sitting position. Note
Face	Eye…	H test for extra-ocular muscles and pause at lateral gaze – horizontal nystagmus, towards the side of the lesion (lateral cerebellar lesion)
		Ask the child question or ask him to read (staccato speech/ scanning dysarthria)
…Limbs	Intention tremor **[Fig.30.4]** 	Ask patient to pick a object. The amplitude of an intention tremor increases as an extremity approaches the object **[Fig.30.4]**
	Dysmetria (incoordination of limb while performing a task) **[Fig.30.5]** 	*Finger to nose test*: Assess dysmetria by asking patient to touch his nose with his index finger and then touch examiner's finger **[Fig.30.5]** Challenge by moving your finger to different locations. Dysmetric child will be unable to connect with examiner's finger or his nose

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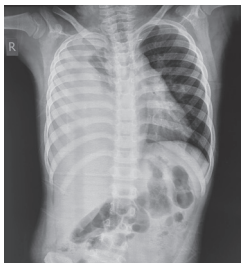
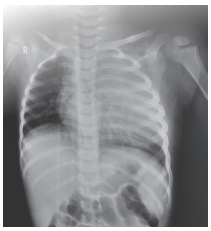
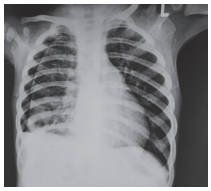
Respiratory System

Differential Diagnosis

Pleural Effusion

Differential diagnosis	Clinical presentation	Differential investigations
Empyema **[Fig.16.11]** 	• Dyspnea, cough, chest pain • Fever with chills • Decreased movement of the chest on the affected side • Decreased vocal fremitus • Dullness on percussion • Diminished breath sounds on the affected side • Decreased vocal resonance and pleural friction rub • Above the effusion, where the lung is compressed, there may be bronchial breathing sounds and egophony	• Pleural tap: Frank pus/organisms on gram stain, positive culture • Chest X-ray: Shift of mediastinum to opposite side, loss of costo-phrenic angle
Tubercular effusion	• Low grade fever, weakness, weight loss night sweats, cough, pleuritic chest • Decreased movement of on the affected percussi vocal , and pleural	uve, AFB stain ised adenosine deaminase say • Chest X-ray: A chest x-ray is typically diagnostic of a pleural effusion. A meniscus sign at the costo-phrenic angle in an upright chest x-ray is diagnostic • CT chest: Pleural thickening and mediastinal lymphadenopathy
olidation 4] 	• Acute onset with high fever, rusty sputum, chest pain and respiratory distress • No mediastinal shift • Resonant on percussion • Bronchial breath sounds on auscultation	• Total leukocyte count: Elevated but non-specific • Sputum cultures and blood cultures may be positive for bacterial pathogens • Chest X-ray: Consolidation
Pneumothorax **[Fig.16.13]** 	• Sudden onset • Decreased movement of the chest on the affected side, hyperresonance on percussion on ipsilateral side of the chest, diminished breath sounds on the affected side, decreased vocal resonance • Mediastinum shift to opposite side	• Visceral pleural line typically identified on chest X-ray • CT chest: Visceral pleural line easily identified; atelectasis of lung

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Central Nervous System

Investigations

Lumbar puncture (done after fundus examination to rule out raised intracranial tension)	Gross appearance	Straw coloured, may form cobweb on standing
	Cytology (total cell counts and differential counts)	High leukocyte count with lymphocytic predominance; neutrophilic response may be seen in early stages
	Biochemistry (CSF protein and sugar)	• Elevated protein (100-800 mg/dL) • Decrease in the glucose levels (20-40 mg/dL), which is generally less than 50% of the serum levels, although is never as low as in pyogenic meningitis
	Gram stain	No organism seen in TBM. This is done to rule out bacterial meningitis (e.g. *N. meningitidis, S. pneumoniae*)
	BACTEC for tuberculosis	Average time required for detection is 9-14 days. It detects growth of AFB radiometrically by measuring the release of CO_2
	Genexpert test	Genexpert test detects the DNA in T genetic mutations associat drug rifampicin
	Cartridge-based nucleic acid amplification test (CBNAAT)	CBNAAT sis specific ic acid amplification R, providing results within
	Adenosine (AD	A is enzyme produced by T lymphocytes. Conditions that trigger the immune system, such as an infection by *Mycobacterium tuberculosis* cause increased amounts of ADA • Results are available in 24 hours and have high sensivity and specificity
	Complete blood count	• Total leukocyte count (lymphocytosis) • ESR is elevated in TB
	Blood glucose	Serum glucose level is required for comparison with the glucose level measured in the cerebrospinal fluid
	Liver function tests	If drug induced hepatitis occurs
	HIV testing	This is done to rule out HIV
Imaging	Chest X-ray	Chest X-ray may be normal or show hilar lymphadenopathy, miliary tuberculosis or patch of pneumonia
	Ultrasound abdomen	Ultrasonography abdomen to look for hepatomegaly, splenomegaly, retroperitoneal lymphadenopathy and free fluid
	CT brain with contrast	Features of TBM include hydrocephalus, basal meningeal enhancement, tuberculoma, or infarcts
	MRI brain with contrast	Magnetic resonance imaging (MRI) provides more detailed information than CT

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Normal Neonate

Neonatal Reflexes

Reflex	Method	Importance
Rooting or Search reflex **[Fig.22.1]** 	• When baby's cheek comes in contact with mother's breast, baby seeks the nipple • When upper lip, lower lip or cheeks are stimulated the baby will turn to that side to find the source of milk. • This reflex is present in normal full term babies and disappears by three months	• This reflex helps the baby for locating the breast as there is no neck control at birth • This reflex disappears when baby develops neck control and can voluntarily turn and find the breast
Sucking and Swallowing reflex **[Fig.22.2]** 	• Sucking reflex can be elicited by introducing finger into the baby's mouth. Baby starts sucking vigorously • Sucking gets well synchronized with swallowing at 34 weeks of ge	• Its absence suggests develop defect • This reflex
Moro's reflex [Fig. 	ine position and back supported on palm of hand, ex the neck by 15 degrees. Release the head to initiate the reflex. Head should be in midline and hands should be open *Reflex Components* Phase 1: Abduction of arms at shoulder and extension of arms at elbows with hands open Phase 2: Adduction of arms and flexion of forearms. In preterm babies phase two is absent because of weakness of antigravity muscles	• Moro's reflex is a vestibular reflex • It appears at 28 weeks of gestation. Reflex is complete after 32 weeks of gestation. It disappears by 3 months • Persistence is seen in cerebral palsy while asymmetrical reflex is seen in Erb's palsy, spastic hemiplegia, fracture of humerus or clavicle, closed hand
Grasp reflex **[Fig.22.4]** 	Touch the ulnar side of palm of baby by your finger to initiate grasp reflex. As you lift your finger, flexor muscles of forearm of baby become tight, and baby supports his whole weight. Phase 2 is present only in term babies	• It appears at 34 weeks of gestation and disappears by three months • Persistence is seen in spastic cerebral palsy and reflex is asymmetrical in hemiplegia and cerebral damage

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Central Nervous System

Acute Flaccid Paralysis

Discussion

Definition of Acute Flaccid Paralysis

Sudden onset of flaccid paralysis in any part of body in a child <15 years of age or paralysis in a person of any age in whom polio is suspected.

Common Causes of Acute Flaccid Paralysis

AFP can be polio AFP or non-polio AFP:

- Poliomyelitis (polio)
- Transverse myelitis
- Guillain-Barré syndrome
- Traumatic neuritis
- Non-polio enterovirus (Ech… Enterovirus-70,71; Coxsackie B…
- Acute peripheral… diphtheritic…
- Po…

…ssify AFP as Poliomyelitis

…case is classified as polio:

- If wild polio virus is isolated from stool
- AFP case without isolation of wild polio virus is classified as "polio compatible" if:
  - Stool samples were inadequate AND
  - Residual neurologic deficit present on 60 days follow-up, or has died before follow-up, or has unknown follow-up status AND
  - 'Expert review committee' concludes the case cannot be discarded as 'non-polio'

Investigations Required for Differentiation of AFP

CSF study: CSF may take week to show changes, so should be done after one week:

- *Poliomyelitis*: High cell count with lymphocyte predominance and slightly increased CSF protein
- *Guillain-Barré syndrome*: No rise in cell count (mononuclear cells) with high CSF protein (this is known as albumino-cytological dissociation)
- *Transverse myelitis*: Normal or slightly increased cell count with normal or slight… increased CSF protein

Nerve conduction … performed at 3rd w… …eeks for chang…

…generation pattern

- … *syndrome*: Demyelination …tern
- *Transverse myelitis*: No definite pattern, may be normal

Electromyography (EMG) is performed at 3rd week as it takes about 2 weeks for changes to occur:

- *Poliomyelitis*: Abnormal (degeneration of muscle units)
- *Guillain-Barré syndrome*: Normal
- *Transverse myelitis*: Normal

Follow Up Sequel in AFP

- *Paralytic poliomyelitis*: Gradual asymmetric atrophy of affected muscles, skeletal deformity may appear later
- *Guillain-Barré syndrome*: Symmetric atrophy of distal muscles, usually full recovery occurs
- *Transverse myelitis*: Initially flaccidity is seen, which is replaced by spasticity as stage of neurologic shock is over. Subsequently,

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Thalassemia

Questions in MCI Competency-Based Curriculum for Undergraduates

Number	Competency & Learning Objective(s)	Domain K/S/A/C	K/KH/ SH/P	Core	Suggested Teaching Learning Method	Suggested Assessment Method
PE 29.4	Discuss the etiopathogenesis, clinical features and management of hemolytic anemia, thalassemia major, sickle cell anemia, hereditary spherocytosis, auto-immune hemolytic anemia and hemolytic uremic syndrome.	K	KH	Y	Lecture, SGD	Written, Viva voice
PE 29.4.1	Define hemolytic anemia.	K	KH	Y	Lecture, SGD	Written, Viva voice
PE 29.4.2	Enumerate the causes of hemolytic anemia in children.	K	KH	Y	Lecture, SGD	Written, Viva voice
PE 29.4.3	Describe the pathogenesis of different types of hemolytic anemia.	K	KH	Y	Lecture, SGD	Writ
PE 29.4.4	Describe the clinical features of hemolytic anemia, thalassemia major, sickle cell anemia, hereditary spherocytosis, auto-immune hemolytic anemia and hemolytic uremic synd	K	KH	Y		iva voice
PE 29.4.5	List the inv dia		KH	Y	Lecture, SGD	Written, Viva voice
PE 29	emia based ical features and nvestigations.	K	KH	Y	Lecture, SGD	Written, Viva voice
E 29.4.7	Describe treatment of hemolytic anemia thalassemia major, sickle cell anemia, hereditary spherocytosis, auto-immune hemolytic anemia and hemolytic uremic syndrome.	K	KH	Y	Lecture, SGD	Written, Viva voice
PE 29.4.8	Describe the role of chelation therapy and recall the drugs, dosages and side-effects of the drugs.	K	KH	Y	Lecture, SGD	Written, Viva voice

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Whatsup: **9560254259**

Email: **practicalpediatrics2020@gmail.com**