CLINICAL EXAMINATION IN HEMIPLEGIA

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I. INTRODUCTION

Hemiplegia is paralysis of one half of the body-which includes arm, leg and often face on the affected side.

Terms used to describe weakness
Hemiplegia: Total Paralysis on one side of the body
Hemiparesis: Weakness on one side of the body.

MAIN POINTS: (Hemiplegia)

- Usually acute in onset
- Results from upper motor lesion/ most commonly pyramidal tract lesion
- Associated symptoms and signs aid the diagnosis of level of the lesion
- A detailed history taking is of great value
- Initially -weakness is flaccid; later-spastic

Diagnostic Features:
- Involvement of one half of the body, hypertonia, hyper-reflexia, extensor plantar- response and characteristic hemiplegic gait.

- Speedy reach to hospital at the earliest warning signs is a must.
- Treatment of risk factors- ideal for prevention
- Refer chart 1 below
II. Points of importance in History Elicitation

HISTORY

1. Presenting symptoms

- **Motor** symptoms
  - Decreased movements - Paresis /paralysis
  - Increased movements; involuntary movements
  - Feeling of stiffness or flailness

- **Sensory** symptoms
  - Reduced sensations – hypoesthesia /anesthesia
  - Increased sensation – occurs in-
    - Thalamic pain – where threshold for pain is increased (Hyperpathia)-
    - it is a boring, diffuse, unpleasant and spontaneous pain
    - Exacerbated by touch of clothing – occurs in stroke involving thalamus

- **H/o** **Higher function disorders**
  - Altered consciousness
  - Speech disturbance

- **cranial nerve** Disorders
  - Especially VII cranial nerve- (commonest involvement): Lower half of face involved due to UMN lesion

- **Pearls for practice:**
(Following Activities of daily life affected in hemiplegia must be asked for in history)
1. upper limb: using a pen, tying shoe lace, using comb/tooth brush, dressing
2. Lower limb: difficulty to lift the foot in front of the other and difficulty in walking
3. Trunk: Difficulty in turning over in bed, in getting on to bed, in seating himself on a chair or lavatory seat
4. Face: Angle of mouth pulled to opposite side, unable to whistle

Elaboration of presenting complaints

1. Onset – sudden or insidious
   Sudden onset indicates vascular or traumatic etiology. (Rarely demyelination)
   NOTE THE TIME WHEN PATIENT WAS LAST FOUND NORMAL

2. Duration

3. Progress- (types)
   a. Stationary or b. progressive with amelioration later c. rapidly progressive
d. slowly progressive  e. Stuttering
   Traumatic lesions are stationary; do not progress
   Vascular lesions progress at first and then start ameliorating.
   Carotid hemiplegia is stuttering in progress (‘step ladder pattern’)
   Malignant lesions are rapidly progressive
   Degenerative conditions are slowly progressive.

4. Rapidity of development of the symptoms–rapid in cerebral hemorrhage

5. Part of the day of development of symptoms –
   During sleep / early morning hours – thrombosis
   Day time – hemorrhage or embolism
   After exertion –hemorrhage

Accompanying symptoms

Fits, headache, vomiting, coma – indicate severe hypertension. (h/o hypertension+)
Symptoms of raised ICT are- headache, vomiting, blurring of vision, fits
Symptoms of other systems (CVS, RS, ABDOMEN)

Precipitating factors:
   Hemiplegia precipitated by epileptic fits – Todd’s palsy.
   Precipitated by fever in – meningitis, encephalitis.

Differential diagnosis to be kept in mind

-Migraine, Todds palsy, TIA, hypoglycemia, Multiple Sclerosis, hysterical hemiplegia
History of migraine, fits, hypoglycemia, episodic weakness and relapses of MS, hysteria
Chart: 2  History elicitation and clinical examination

**Past history**
(\(\text{age of the patient has a bearing}\))

- **Elderly**: h/o hypertension, diabetes mellitus, IHD, Hypercholesterolemia, Recent M.I, Embolic source (cardiac, artery to artery), large cell arteritis like Mitral stenosis, MVPS, prosthetic valve insertion,

- **Young adults**:  
  1. Cardiac diseases – Mitral stenosis, MVPS, prosthetic valve insertion, Fallot’s, arrhythmias, cardiomyopathy, Infective endocarditis

  2. Vascular diseases - Hypertension, collagen vascular diseases Arteritis –TB, syphilis

  3. Hematological: leukemia, lymphoma, polycythemia, sickle cell anemia

  4. CNS diseases: epilepsy, migraine

  5. Respiratory diseases: h/o malignancy, tuberculosis

  6. Infections: CSOM, cerebral abscess, TB, syphilis

- **In females**:  
  H/o oral contraceptives, h/o recent delivery (which can cause- -cortical vein thrombosis), collagen vascular diseases, migraine

- **In infants**:  
  Intra natal and perinatal h/o mother, mile stones, h/o involuntary – movements

- **In extremes of age**: H/o dehydration (diarrhea, vomiting).- which can predispose to cerebral thrombosis
**Family history**

- hypertension, diabetes mellitus, ischemic heart diseases,  
  Epilepsy, migraine  
- Stroke runs in families but no inheritance-pattern

**Personal history**

- Alcoholism,  
- smoking,  
- exposure to toxins,  
- consumption of drugs  
- H/o sexually transmitted infections/diseases

Alcoholism by definition is the pattern of drinking which is harmful to the individual and the family.  
*Permissible amount of alcohol:*  
20 units per week for males.  
15 units per week for females.  
1 unit = 10 gms

Ill effects of smoking on CNS: increases the incidence of cerebro vascular lesion.

**III.CLINICALEXAMINATION**

**III.A.GENERAL EXAMINATION**

*General appearance* including posture,motor activity

*Vital signs*-Level of consciousness,pulse,BP,look for pupil size,conjugate deviation of eyes  
Meningeal signs

▶*Neuro cutaneous markers:*

1. Neurofibroma over the skin (may have associated Tuberous sclerosis of brain)  
2. Caféaulait spots-associated with neuro fibramatosis, tuberous sclerosis, and Ash leaf spots  
3. Sebaceous adenoma  
4. Sturge Weber syndrome – facial nevus (port wine stain) involving one half of face with upper eyelid - associated with atrophy and calcification of ipsilateral cerebral hemisphere .It is caused
by errors in ectodermal and mesodermal development. Manifests with fits and hemiplegia on the opposite side
► Lymphadenopathy in young: leukemia/lymphoma/disseminated tuberculosis
► Cyanosis – suggests Fallot’s
► Clubbing – TB, infective endocarditis, bronchiectasis
▲ Shortening of hemiplegic limb – indicates it is dating from early childhood
► Irregular pulse of atrial fibrillation.
BP must be recorded always.

**IIIB. HIGHER FUNCTIONS**

A. Consciousness
B. Orientation
C. memory
D. intelligence
E. speech
F. Emotion
G. Judgment
H. behavior
I. Presence of hallucination /delusion /illusion.

**Levels of altered sensorium:**
1. Drowsy 2. Stuporous- Not easily arousable 3. Semiconscious-responding to painful stimuli only. 4. Unconscious-No response to even painful stimuli.

**Condition with hemiplegia and altered level of consciousness**

1. Cerebral hemorrhage
2. Extensive cerebral infarct
3. Raised ICT due to cerebral edema
4. Infarct turning hemorrhagic
5. Meningitis or encephalitis
6. Brainstem lesions
7. Infra tentorial herniation
8. Trauma
9. Space occupying lesion: tuberculoma or cerebral abscess

**Speech disturbances: -Aphasia**

Aphasia: Definition: partial or total loss of ability to communicate verbally or by written words.
HANDEDNESS

The hemisphere in which speech area is located is referred to as dominant hemisphere. In majority of right handed persons dominant hemisphere is on left side and vice versa. Damage to speech areas in dominant hemisphere causes aphasia. In a right handed person damage to left hemisphere can cause hemiplegia with aphasia. In 95% of right handed individuals, left dominance occurs with speech area on left. In 70% of left handed, right dominance occurs.

Types of aphasia:

Broca’s aphasia (Synonyms: Motor aphasia/Expressive aphasia/Non fluent Aphasia):
- Inability to “express” though patient understands what is being talked to.
- ie. Verbal comprehension is normal but unable to express.
- Features affected: naming, fluency, rhythm
- Anomia; Inability to name objects
- Aggramatism: disuse of connecting terms like “and”, ‘the’ and ‘of’

Wernick’s aphasia (Sensory aphasia /Receptive aphasia/Jargon aphasia)
- Comprehension and production of meaningful speech is affected.
- Inability to understand spoken words in a language well known to the patient.

Global aphasia: is a combination of Broca’s and Wernick’s.
- In addition patient is unable to read or write. Comprehension is poor for spoken words and written language.

Speech areas
- Located in the dominant Hemisphere
- Broca’s Area: posterior 1/3rd of inferior frontal convolution-Area44
- Wernick’s Area: upper part of posterior temporal lobe and adjacent parietal lobe.
How to test for aphasia:

Test for:
1. Fluency
2. Naming
3. Repetition
4. Obeying of commands
5. Reading
6. Writing
7. Articulation

TECHNIQUE:
- Engage the patient in conversation and ask him to describe his illness.
- Note if speech is fluent or non fluent.
- Does the patient understand complex commands/simple commands?
- Is he able to read correctly?
- Is he able to write correctly?
- Can he name the objects?
- Can he recognize objects? if not – the condition is agnosia

- **Differentials for Aphasia:**
  - **Confusional** states versus aphasia
    - Ask patient his name, date and whereabouts; this will elicit
      - an inappropriate response if the patient is grossly aphasic
    - If he is merely confused response will be appropriate but incorrect.
  - **Akinetic mutism:**
    - Patient lies apparently awake but is immobile and mute –with eyes open and
      fixing on people or objects around the room.
    - The site of lesion is in frontal lobe or in the region of 3rd ventricle
  - **Dysarthria:** poorly articulated speech; muscles or nerves concerned in production of
    speech (peripheral apparatus of speech) are dysfunctional in dysarthria
  - **Dysphonia:** hoarse or whispered speech.

**Orientation:**
- Orientation-in time, space, person are to be tested
- Organic confusional state: Indicated by dementia and delirium
- Dementia: person is confused in time, place and person but he is alert and awake
- Delirium: patient is similarly confused but is having impaired alertness.
- Delirium occurs in alcoholism and atropine poisoning

**Memory:**
- **a. Immediate memory**: memory lasting up to a minute or so.
  - Test: Ask the patient to repeat 7digit number or 5 digit number
  - Ask him to spell the word ‘world’ backwards or any short word
  - Ask if he can remember telephone number while dialing
  - Give an address to remember and ask to repeat after a short interval or distraction
  - give 3 names to remember and recall after 2-5 minutes
  - Condition where immediate memory is impaired: Korsakoff’s psychosis
b. Recent memory: Memory of events which occurred in the last few days – events should be of personal importance or world news. Check with relatives or friends of the patient if he is correct.
c. Remote memory: is long term memory – is relatively resistant to loss. But in progressive severe dementia, it can get lost.

Intelligence:
Affected in dementia. To test – asks the patient to serially deduct 7 from 100 backwards or add 7 forwards. (If the patient is literate)

Judgment:
Ask him what he would do if fire breaks out or what he would do if an addressed envelope is lying on the road.

Emotion:
Anxious /depressed /elated /swings of moods.

IV. CRANIAL NERVES

Importance of cranial nerve examination in hemiplegia: (Table1)

Optic nerve -  
*Papilledema* in raised intracranial tension  
Tubercular meningitis with hemiplegia can have *optic atrophy*

Oculomotor nerve  
involved in Weber’s syndrome (Fig1a)  
Pinpoint pupils in pontine hemorrhage

Abducens and facial  
ipsilateral 6th and 7th nerve palsy with contra lateral hemiplegia in Millard – Gubler’s syndrome  (fig1c.)

5th, 7th and 8th  
Cerebellopontine angle tumor with extension to cortico spinal tract

Pearls for practice

Important cranial nerve signs to look for in hemiplegia
Papilledema (II)  
Ptosis (III)  
Abnormal/asymmetrical pupils II, III)  
Abnormal eye position (III, IV, VI), conjugate deviation of eyes  
Facial droop  
Dysarthria

Learning points

1. UMN palsy of VII nerve lesion above Pons
2. LMN palsy of VII nerve crossed hemiplegia
3. Mimic palsy- of frontal lobe lesion (emotional facial movements lost, voluntary facial movements retained)
4. Hemiplegia without 7th cranial nerve involvement is suggestive of lesion below Pons
5. Almost all the cranial nerves receive bilateral innervations from the pyramidal tract.- except CrNerveVII that innervates lower face.
<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Site</th>
<th>Tract</th>
<th>Signs</th>
<th>Usual causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Webers</td>
<td>Base of midbrain</td>
<td>Cortico spinal tract + III nerve nucleus</td>
<td>Ipsilateral III nerve palsy + contralateral hemiplegia</td>
<td>Vascular, tumor</td>
</tr>
<tr>
<td>2. Benedikt’s</td>
<td>Midbrain</td>
<td>Cortico spinal tract, III nerve nucleus + red nucleus</td>
<td>Ipsilateral III nerve palsy + contra lateral hemiplegia + tremors + ataxia</td>
<td>Vascular, tumor</td>
</tr>
<tr>
<td>3. Millard-Gublar’s</td>
<td>Pons</td>
<td>Corticospinal tract + VI and VII nerve nuclei</td>
<td>Ipsilateral LMN VI and VII nerve palsy + contralateral hemiplegia</td>
<td>Vascular, tumor</td>
</tr>
<tr>
<td>4. Medial medullary syndrome</td>
<td>Medial medulla</td>
<td>Cortico spinal tract + XII nerve nucleus</td>
<td>Ipsilateral tongue paralysis + contra lateral hemiplegia</td>
<td>Vascular</td>
</tr>
</tbody>
</table>

Table 1. Cranial nerve syndromes associated with hemiplegia
V. SPINAL MOTOR SYSTEM

Points to note in the examination of the spinal motor system: (chart3)
Hemiplegia often is a UMN lesion:

1. Nutrition:
Wasting of the muscles is a very late phenomenon in UMN hemiplegia - wasting occurs commonly in chronic hemiplegia in small muscles of hand due to disuse atrophy

2. Power:
Often groups of muscles are involved - (UMN Lesion);
Often distal muscles are involved
- Grading of power
  0. absent movements
  1. -flicker of contraction
  2. Movements with gravity eliminated
  3. Movements against gravity, not against resistance
  4. Movement against gravity and resistance but less than normal
  5. Normal power

Law of dissolution;
Movements which are lost late in evolution are first to be affected and last to recover e.g. Fine movements

3. Tone:
Tone is a state of partial contraction of resting muscles which helps in maintenance of posture.
Spasticity: is Velocity dependant increase of muscle tone in response to passive muscle stretch.
- Spasticity is a sign of pyramidal lesion found commonly in hemiplegia
- Clasp-knife type of spasticity is typical. (resistance to passive movements with sudden giving way, usually towards completion of joint flexion or extension)
- . In upper limbs – tone is more in flexors than extensors (flexor predominance)
- In the lower limbs – extensor predominance
  Thus tone is more in antigravity group of muscles
Clonus: sustained clonus is a sign of pyramidal tract lesion.
Clonus is a series of rhythmical contraction occurring in response to maintenance of tension in muscle tendon (in ‘pseudo clonus’ contractions are not sustained)

Clonus is due to **gamma** neuron discharge.

**Sites** where elicitable:
- Ankle clonus: elicited by forcibly dorsiflexing the foot after flexing the hip and knee
- Patellar clonus: by sharply moving the patella downwards.
- Wrist clonus: by suddenly and forcibly dorsiflexing the wrist.
- Finger clonus: by suddenly extending the fingers
- Pseudo clonus: is ill sustained; and is seen in tense individuals normally.

**Flaccidity** – in the initial state of neuronal shock

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**Chart3. Factors to be examined under spinal motor system**

**Coordination:** affected if cerebellar lesion is associated

**Involuntary movements:** Accompany if extra pyramidal lesion is associated.

**Reflexes (table2)**

For convenience reflex examination is included after motor system

But ideally must be tested after sensory system examination

DTR can be reinforced by patient performing isometric contractions
(e.g. clenching the teeth)

**Deep tendon reflexes:**

**JAW JERK:**
- Relax the jaw, open 1/3rd and keeping the thumb over the chin, tap the thumb;
- Response is closure of the jaw. Note the speed of the closure
- Exaggerated jaw jerk occurs in pseudo bulbar palsy.
- Pseudo bulbar palsy may be associated with UMN lesion of the limbs.
- Absent jaw jerk or slight response is normal

Afferent: V cranial nerve
Efferent: V cranial nerve motor component

**Biceps Jerk:** (Root value C5C6)  
(Positioning-Elbow flexed, forearm supinated)
Keep the forearm supported on the patient’s thigh, or on examiner’s forearm.
Patient’s forearm is held midway between flexion and extension.
Keeping the thumb firmly over the biceps tendon and with fingers around the elbow, 
-tap the thumb briskly.
Normal response is flexion of the elbow.
Lesion above C5 causes exaggerated biceps reflex

**Supinator reflex:** Root value (C5C6) - (With elbow flexed, forearm pronated)
Strike the radius 2cm above the wrist joint,
Response-flexion of elbow and contraction of brachio radialis and flexion of fingers

**Triceps reflex;** (root value C7 C8)

Hold the arm midway between flexion and extension, resting it on the thigh.
Tap the triceps tendon just above the olecranon.
Normal response is extension of the elbow and contraction of triceps.

**Finger flexion Reflex** (root value C7C8)
Blow upon the palmar surface of the semi flexed fingers
Normal response-flexion of the fingers and thumb.

**Knee Jerk ;** (Root valueL2, L3, L4)
Let the knee swing free by the side of the bed with the patient sitting up.
Tap the quadriceps tendon below the patella
Response: Extension of the knee and contraction of the quadriceps.

**Ankle jerk** (root value L5S1)

Flex the knee, rotate the thigh externally, slightly dorsiflex the foot and tap the Achilles tendon.
Response: plantar flexion of the foot and contraction of gastroconemius

Reinforcement of deep tendon reflexes;Ask the patient to clench the teeth or for lower limb reflexes 
have the patient to hook together their flexed fingers and pull apart-(Jendrassik maneuver)

**Grading of Reflexes:**
Refer Chart 4.

<table>
<thead>
<tr>
<th>REFLEX</th>
<th>ROOT VALUE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jaw jerk</td>
<td>V cranial nerve</td>
</tr>
<tr>
<td>Biceps Reflex</td>
<td>C5C6</td>
</tr>
<tr>
<td>Supinator Reflex</td>
<td>C5C6</td>
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<tr>
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<td>C7, C8</td>
</tr>
<tr>
<td>Knee Jerk</td>
<td>L2, L3, L4</td>
</tr>
<tr>
<td>Ankle Jerk</td>
<td>L5S1</td>
</tr>
</tbody>
</table>

Table 2. Root value of reflexes

Chart 4.

GRADING REFLEXES
0 No response
1 hypoactive
2 normal
3 hyperactive; no clonus
4 hyperactive with clonus
Certain pathological reflexes looked for in hemiplegia:
In the upper limb: clinically significant if markedly active and asymmetrical

**Hoffmann’s Sign**
- It is significant if unilateral and markedly active
- It is an index of muscle hypertonia in upper limb. And is equivalent of Babinski’s in lower limb.
- To elicit, terminal phalanx of middle finger is suddenly flexed and released.
- Positive response is adduction and flexion of the thumb and flexion of fingers.

**Wartenberg’s sign:**
- To elicit - patient’s fingers are interlocked with examiner’s fingers and pulled holding examiner’s palm pronated and patient’s palm supinated.
- Normally thumb extends.
- In pyramidal lesion thumb is flexed and adducted.
- This is a sign of flexor hypertonia occurring in upper limb.
- This sign is equivalent of Babinski of lower limb.

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**Superficial reflexes in hemiplegia:**

Importance of abdominal reflex:
**Abdominal reflex and cremastric reflex** loss is a sign of UMN lesion.
But loss is *not dependent* on the severity of pyramidal lesion.
In congenital diplegia and motor neuron disease – not lost till late.
But lost very early in multiple sclerosis.

Abdominal reflex
- To elicit: Stimulus is a scratch or stroke on anterior abdominal wall
- Trace the scratch from outwards to inwards
- Look for contraction of abdominal muscles and
- Movement of umbilicus towards stimulus
- Contra lateral side – contraction lost in (a). pyramidal lesion
Same side – contraction lost-in (b) LMN lesion (T7 – T12)-in paraplegia

Root value for abdominal reflex
Epigastric region – T7-T9
Upper abdomen -- T9-T11
Lower abdomen-- T11-T12
Superficial abdominal reflex lost also in: obesity, elderly, muscle wasting, rigidity

Cremastric reflex
Root value; L1
Test in lying or standing posture
Stroke over inner upper aspect of thigh from above downwards and inwards
Response: testes moves upwards
Absent in---------pyramidal tract lesion
LMN lesion at L1
Non pyramidal lesion where cremastric reflex is lost/diminished – varicocele
In hemiplegia-superficial reflexes affected are abdominal, cremastric, plantar.

In Lower Limb

Plantar reflex:
Abnormal extensor plantar reflex is positive babinski sign

• Eliciting plantar reflex:
  o With the patient lying supine,
    - ‘flex the knee, fix the foot’ with ankle externally rotated
    Using blunt object
    Stroke on lateral aspect of foot
  o Normal response:
    Plantar flexion of great toe and plantar flexion of other toes

• Root value: L5, S1

Types of Babinski response: (chart 6)

• True Babinski sign:
  Extension of great toe with or without
  -fanning and abduction of other toes;
  Fully developed response involves above response plus
  -dorsiflexion of ankle plus flexion of knee and hip with slight abduction of the thigh
  causing withdrawal of leg.

• Minimal Babinski:
  No movements of the toes
  Contraction of tensor facia lata and of adductor muscles of thigh - can be only felt.

• Equivocal Babinski sign: Initial flexor movement of the toe followed by extension.

Babinski mimickers
Pseudo Babinski sign occurs-
  a. If the short flexors of the toe are paralyzed (Inverted plantar)
b. In athetosis or chorea when the involuntary movement is super imposed at the time of eliciting the reflex, the great toe may extend as a part of athetoid movement.
c. In case of plantar hyperesthesia, application of strong or painful stimulus to the sole of the foot produces ‘withdrawal response’

**Positive bilateral Babinski in absence of** pyramidal lesion:
- a. Infants before age one
- b. deep sleep
- c. deep anesthesia
- d. post ictal state
- e. coma.

**Other methods** of eliciting plantar reflex:
- These methods test positive in extensive pyramidal lesions
  - Oppenheim’s method: firm stroke applied down on either side of anterior border of tibia with finger and thumb.
  - 2. Gordon reflex: the calf muscles are squeezed.
  - 3. Chaddock method: A curved stroke applied below the lateral malleolus.

**Absent Babinski** response in the presence of pyramidal lesion:
In S1 lesion with paralysis of extensor hallucis longus.

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**Chart 6. Types of Babinski response**

**Gait**

Gait in hemiplegia: (circumduction gait)
Patient has difficulty in flexing the hip, knee and ankle since flexor muscles are weak in the lower limbs and tone is increased in the anti gravity muscles. Patient therefore drags the foot. The foot is raised from the ground by tilting the pelvis and the leg is swung forward in semicircle from the hip, so the foot tends to describe an arc, the little toe scraping along the floor.
(Hip- is tilted upwards and abducted.
Knee – stiff and hyper extended
Ankle – excessively plantar flexed and may invert (equino varus)
Upper arm adducted and flexed with minimal swing of shoulder; elbow and wrist flexed
VI. Examination of sensory system

Hemi sensory loss can occur in thalamic or cortical lesion along with hemiplegia not common – hence details of exam of sensory system – not dealt with in this article

VII. Cerebellar Examination:
Abnormal signs in hemiplegia are caused by medial medullary syndrome.

VIII. Extra pyramidal signs in hemiplegia
Occur in pseudo bulbar palsy.

IX. Examination of other systems

Cardio vascular System: look for signs of congenital, valvular, and hypertensive heart diseases
Respiratory system: Look for - Tuberculosis, lung abscess, bronchogenic carcinoma
Abdomen: Look for hepatosplenomegaly, mass abdomen.