Motor Systems Clinical

Cases and Examples
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Notes: MotorClinicalReview.pdf

Lecture Goals
• Review components of symptoms and tests as a whole
• Track through the motor system reviewing disorders and constellations of symptoms
• Lab Cases and Review Questions
• Notes available as download and Cases and Review via the FAQ

Neuro-components of a Clinical exam

• Mental status
  – Cognitive
  – Emotional state
  – Speech
• Motor status
• Sensory status

Motor Clinical testing

• Muscle and reflex function:
  – spinal and cranial systems including eyes
• Muscle group:
  – Strength
  – Endurance
  – Tone
  – Voluntary control
  – Reflexes
• Presence of unusual involuntary motions or postures

Clinical strength and reflex testing

Issues in both voluntary and involuntary

• Coordination
  – Strategy
  – Muscles used
  – Quality
• Accuracy
• Initiation
  – Speed of initiation
  – Context appropriate initiation
• Sequencing
**Muscle and reflex function: spinal and cranial systems**

**A Spinal: segmental pattern**
- 1. Fasciculations, muscle weakness, hypotonia: lower motoneuron or peripheral nerve damage
- 2. Voluntary weakness, hypertonia, hyperreflexia, clasp knife, clonus, (spasticity), Jendrassik modulation of spastic reflexes: Corticospinal/reticulospinal damage (pyramid or upper MN syndrome).
- 3. Unusual reflexes: Babinski, grasp; corticospinal damage

**Muscle and reflex function: spinal and cranial systems**

**B Cranial : cranial nerve patterns**
- 1. Fasciculations, muscle weakness, hypotonia: motoneuron or peripheral nerve damage
- 2. Voluntary weakness, hypertonia, hyperreflexia, clasp knife, clonus (spasticity): Corticobulbar/reticulobulbar damage (pyramid or upper MN syndrome)
- 3. Unusual reflexes: routine reflex: corticobulbar damage

**Lower Motor Neuron Signs:**
- Hyporeflexia
- Hypotonia
- Fasciculations
- Denervation-induced muscle atrophy
- Flaccid paralysis

**Upper Motor Neuron Signs:**
- Hyperreflexia
- Hypertonia
- Clonus
- Clasp-knife
- Spastic paralysis

**Somatotopy of Corticospinal tract:**

*Effect of Lesion of medullary pyramids:* Babinski sign, contralateral weakness, permanent loss of fine movements of the digits
Presence of unusual involuntary motions or postures

A. Hyperkinetic
- 1. Myoclonus: muscle spasms: subacute spongiform encephalopathy (e.g. slow viruses, Jakob-Creutzfeld, BSE or Mad Cow disease). Epilepsy.
- 2. Athetosis: writhing sinuous motions: basal ganglia: striatal lesions, mostly putamen (or rarely globus pallidus) from perinatal brain damage.
- 3. Chorea: jumpy rapid motions, dance-like: basal ganglia: Huntington’s, striatal lesions, mostly caudate, L-dopa overdose (intoxication), (historically, idiopathic: chronic classical neuroleptics: Tardive dyskinesia, other drugs, new drugs). Excessive transplant DA.
- 4. Hemiballismus: flinging ballistic limb motions: basal ganglia: Subthalamic nucleus degeneration or lesion
- 5. Eye motions disrupted by random saccades: basal ganglia
Presence of unusual involuntary motions or postures

- Dystonias: inappropriate prolonged deforming contractions: basal ganglia: L-dopa intoxication. Neonatal brain damage. Focal dystonias may arise from repetitive use but involve basal ganglia / cortex: e.g. typist hands cramp in bizarre posture on typing.

Presence of unusual involuntary motions or postures

B. Hypokinetic
- 7. Resting tremor: disappears with motion: Parkinson’s (degeneration SNc)
Presence of unusual involuntary motions or postures

C: Cerebellar

• 8. Intention tremor: begins with movement: Cerebellar damage
• 9. Postural tremor: fatigue, stress, benign familial central tremor (alleviated by small quantities of alcohol)

Voluntary control tasks

A Posture

• 1. Dystonic and athetotic postures: basal ganglia: neonatal striatal damage, late untreated Parkinsonism.
• 2. Swaying posture: vestibular disorders, Cerebellar disorders: vermal/fastigial or flocculonodular damage.
• 3. Failed Romberg test: vestibular, Cerebellar disorders: vermal/fastigial or flocculonodular damage.
• 4. Trunk tremor (titubation): cerebellar fastigial effects.
• 5. Rigid postures: Rigid cocontracted muscles: "cog-wheel" or "lead pipe" rigidity, masked facies: flat expression, e.g. Head drop test, no Jendrassik effects: basal ganglia disorders (see Sequences of Movements: akinesia below, Parkinson's disease, SNc degeneration)

B Walking / gait

• 1. Spastic interference, foot drop in walking: corticospinal, reticulospinal lesions
• 2. Wide base of support, ataxic gait, asynergia of limbs, dysmetric stepping: lesion of DSCT, spinocerebellum (includes some hypertonia (fastigial, vermis lesions), acute alcohol intoxication, chronic alcohol abuse).
• 3. Small shuffling steps (marche a petit pas), difficulty initiating, locomotor akinesia, bradykinesia, alleviated by target steps: basal ganglia, Parkinson's (degeneration SNc)

C Targetted limb and eye movements, speech

• 1. Weak spastic motions: Premotor Cortex, or pyramidal tract and reticulospinal lesions.
• 2. Lack of fractionation of motion with normal tone: Non-trunk primary motor cortex or low pyramid lesions.
• 3. Ataxic, asynergic, dysmetric arm reaches with intention tremor, possible hypotonia: Cerebellar lateral lobes and dentate/interpositus lesions.
• 4. Ataxic, asynergic, dysmetric arm reaches, acute alcohol intoxication.
• 5. Dysmetric eye motions: cerebellum damage
• 6. Nystagmus: vestibular or cerebellar damage.
• 7. Dysarthria: slurred speech (head area of fastigial, vermis lesions), acute alcohol intoxication, damage to cerebellum, premotor or Broca's areas etc (see Cortex lectures)
Voluntary control tasks

• D Sequences of movements
  1. Akinesia, bradykinesia: inability or slowness in initiation of movements, worsened in sequences, helped by external cues: Basal ganglia, Parkinsons (SNc degeneration)

• E Skills of life: tie shoe, write etc.
  1. Micrographia: progressively smaller writing until nearly illegible: basal ganglia: Parkinson's (SNc degeneration).
  2. Inability to sequence otherwise normal limb use: apraxias: supplementary motor cortex.
  3. Neglect of limb use, denial of limb possession: non-dominant parietal cortex (see Cortex lectures).

Case 1

• A 45 year old carpenter came to his physician with "a trembling right hand" ...

Answer

• This lesion is in the lateral hemisphere of the posterior lobe of the cerebellum. [right side.]
• This lesion also includes the deep cerebellum nuclei, which is indicated by the presence of an intention tremor.

Case 2

• A man was brought to the hospital after collapsing in the street...

Answer

• This is a lesion that involves the posterior limb and genu of the internal capsule on the right side. The pathways damaged that result in the described deficits include
  1. corticospinal (paralysis, Babinski)
  2. corticoreticular (spasticity, increased deep tendon reflexes)
  3. corticobulbar (forehead wrinkled, but mouth drooped on left side)
  4. thalamocortico (specifically from VP to parietal lobe).
Case 3

• An elderly housewife went to her doctor complaining of involuntary movements in her right arm...

Answer

• Hemiballism: This lesion is caused by damage to the subthalamic nucleus (left side.)

Case 4

• A 61 year old architect suddenly lost his ability to speak and within a few minutes he was unable to move his right arm...

Answer

• This involves a lesion in the cortex on the left side. Specifically, motor and premotor cortex in the area to which the mouth and arm are somatotopically mapped. Sudden onset indicates a cerebrovascular accident or sudden trauma.

• (1) Corticospinal (paralysis)
• (2) Corticoreticular (spasticity)
• (3) Corticobulbar (there is bilateral representation to upper part of facial motor 7 which innervated upper 1/2 of face. Thus, forehead can wrinkle even if one side of cortex is damaged

Symptoms by motor system component and lesion

• Spinal cord
• Decussation
• Brainstem
• Mid Brain
• Cerebral peduncles
• Cerebellum and peduncles
• Basal Ganglia
• Thalamus
• Internal capsule
• Cortex

Spinal cord

• Ventral root: Ipsilateral myotome faccial paralysis, fasciculation, hypotonia, areflexia
• Dorsal root: Ipsilateral myotome and dermatome areflexia.
• Ventral horn: Ipsilateral fasciculations, flaccid paralysis, hypotonia, areflexia in myotonmes affected
• Dorsal horn: Ipsilateral pain and temperature loss in dermatomes, perhaps fine touch
• Central canal: Bilateral loss of pain and temperature in dermatomes, flaccid trunk paresis
• Dorsal columns: Ipsilateral loss of fine touch and proprioception
• Lateral funiculus: Ipsilateral spastic paralysis, contralateral pain and temperature loss
• Hemisection: Ipsilateral spastic paralysis, loss of fine touch and proprioception, contralateral pain and temperature loss
• Motoneuron diseases: Ipsilateral fasciculations, flaccid paralysis, hypotonia, areflexia in myotonmes affected. Pattern of damage may be diffuse and patchy.
• Amyotrophic Lateral Sclerosis: Ipsilateral fasciculations, eventual flaccid paresis, combined with spastic paralysis/paresis (upper and lower motoneuron signs) in motor system
• Multiple Sclerosis: Sensory and motor signs of CNS lesions in long myelinated tracts, NO lower motoneuron signs

Pyramidal decussation:

• alternating hemiplegia (special case)
  – Ipsilateral upper arm and contralateral lower leg paresis. Tone may or may not be normal
• Hemiplegia without tone deficit
Brain stem level: see Cranial nerves, Brainstem notes

Cerebral peduncles:
- contralateral spastic paresis in pattern based on portion of somatotopic pyramidal system involved in lesion.

Mid-brain / pons:
- Suppression / lesion above red nucleus:
  - Decorticate posture (arms flex, legs extend)
- Suppression / lesion between red nucleus and life support systems of medulla:
  - Decerebrate posture (arms extend, legs extend)

Cerebellum:
- Vermis, Fastigial, ICP:
  - Ipsilateral hypertonia, loss of balance, Romberg sign, gait ataxia
- Dentate, Interpositus, SCP:
  - Ipsilateral hypotonia, intention tremor, dysmetria, reach ataxia
- PICA syndrome: lateral medullary syndrome (see brainstem lectures):
  - Ipsilateral nystagmus, dysmetria, ataxia, dysarthria, loss of gag reflex, ipsilateral homonymous syndrome
- AICA syndrome: lateral inferior pontine syndrome (see brainstem lectures):
  - Ipsilateral weakness of face, contralateral spastic hemiparesis, Ipsilateral gait ataxia
- SCA syndrome: lateral superior pontine syndrome (see brainstem lectures):
  - Ipsilateral limb and trunk ataxia, intention tremor, dysmetria

Cerebellar function tests

Basal ganglia:
- Striatum: caudate:
  - Contralateral chorea, some athetosis. If degenerative a bilateral effect occurs
- Striatum: putamen:
  - Athetosis, some chorea, (affective disorders, dementia, see cortex lectures)
- Globus pallidus, internal:
  - Contralateral hyperkinesia.
- Globus pallidus, external:
  - Contralateral akinesia
- Substantia Nigra Pars Compacta:
  - Parkinson’s symptoms: akinesia, bradykinesia, resting tremor, rigidity, dystonia.
- Subthalamic nucleus:
  - Contralateral hemiballism
**Thalamus:**
- VL: Contralateral hypotonia, paresis,
- VA: Contralateral akinesia

**Internal Capsule:**
- Contralateral spastic paresis in pattern based on portion of somatotopic organized pyramidal system in capsule involved in lesion (see Cortex lectures).

**Motor Cortex:**
- Primary motor (except trunk):
  - Contralateral paresis, normal tone, babinski or grasp, routing
- Premotor and primary trunk:
  - Contralateral spastic paresis, some apraxias
- Supplementary:
  - Contralateral/bilateral apraxias
- Frontal eye fields:
  - Temporary gaze paralysis

**Arteries**
- ACA syndrome
  - Spastic paralysis of leg more than arm/face and incontinence other See Cortex lectures
- MCA syndrome
  - Spastic paralysis of arm/face more than leg, no incontinence
  - Speech effects, other See Cortex lectures
- PCA syndrome
  - Motor effects minimal. See Cortex lectures

**Upper Motor Neuron Signs**
- Hemiparetic gait

**Lower Motor Neuron Signs**
- Muscle stretch reflexes
  - Hyporeflexia
Upper Motor Neurons Signs

Muscle stretch reflexes
Hyperreflexia

Lower Motor Neurons Signs

Muscle stretch reflexes
Hyperreflexia
### Differentiation between Spasticity and Rigidity

<table>
<thead>
<tr>
<th>Normal</th>
<th>Spastic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clonus</td>
<td>Present</td>
</tr>
<tr>
<td>Absence of antagonist reflexes</td>
<td>Present</td>
</tr>
<tr>
<td>Absence of Babinski sign</td>
<td>Present</td>
</tr>
<tr>
<td>Absence of Patellar reflex</td>
<td>Absent</td>
</tr>
<tr>
<td>Absence of Dorsal flexion reflex</td>
<td>Absent</td>
</tr>
<tr>
<td>Absence of Knee jerk reflex</td>
<td>Absent</td>
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</tbody>
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### Reflex balances

<table>
<thead>
<tr>
<th>Tone:</th>
<th>Length</th>
</tr>
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<tbody>
<tr>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Spastic</td>
<td>Spastic</td>
</tr>
</tbody>
</table>

### Electromyographic findings (EMG)

- Normal activity at rest
- Electrical activity with the result of movement of the affected limb.

### Figure 4

- **Fig. 11-6:**
  - (A) Lead pipe
  - (B) Clasp knife
  - (C) Ileum

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