Section 4

Individual Muscle Testing

A very common complaint encountered in general medical practice is that of weakness. As discussed in the section on the neurological history, this complaint needs further clarification since weakness may be used to denote fatigue, malaise or other non-specific symptoms to certain patients. If the patient does indeed complain of loss of strength in an extremity or elsewhere, then it is the task of the examiner to determine the distribution, degree and type of weakness. The distribution of weakness (e.g., extensors in the arm, flexors in the leg); associated deep tendon reflex (DTR) changes (e.g., increased); presence or absence of atrophy (e.g., absent); and type of motor tone (e.g., spasticity) are the characteristics used to define type of weakness. The previous example defines upper motor neuron weakness. The following is a summary of types of weakness commonly encountered in clinical practice.

<table>
<thead>
<tr>
<th></th>
<th>Upper Motor Neuron</th>
<th>Lower motor Neuron</th>
<th>Myopathy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distribution</td>
<td>Extensors in arm</td>
<td>Follows root or nerve innervation pattern</td>
<td>Proximal Symmetric</td>
</tr>
<tr>
<td>Atrophy</td>
<td>Absent</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td>DTR</td>
<td>Increased</td>
<td>Decreased</td>
<td>Decreased</td>
</tr>
<tr>
<td>Muscle Tone</td>
<td>Increased</td>
<td>Decreased</td>
<td>Not affected</td>
</tr>
</tbody>
</table>

Exceptions to the above may occur in certain specific disease states such as motor neuron disease (amyotrophic lateral sclerosis) where weakness patterns vary, but the above patterns serve in most clinical situations.

How muscle strength is tested is an extremely important and often under emphasized clinical skill. Many extraneous factors may influence the examiner’s interpretation as to whether or not the patient has weak muscles. Patients may not exert full effort because of pain, their wish to emphasize their own impairment, or lack of understanding as to what is desired of them during the examination. Individual doctors, as well as patients, vary in their own physical strength. This often leads to inter-examiner variability. The best one can do is to strive for standardization of how he or she performs the test from one patient to the next. By doing this you eventually develop a feel for how strong various types of patients should be when compared to yourself.

A cardinal practice should be that **you are the one exerting the force** against the muscle being tested. The force you exert becomes the gauge for normality or abnormality. It is also easier to detect **break-away** or **give-away** weakness. Here the patient suddenly gives up on the force they exert and the examiner feels a sudden decrease in resistance in the muscle being tested. This is in contradistinction to true weakness where there is a **smooth decrease** in resistance as the examiner exerts increasing force. Your ability to recognize this will increase with experience in testing muscle strength. The key to achieving this experience is **standardization** of your performance of the examination.
each muscle tested, it should be placed in the position of maximal mechanical advantage (vide infra) and then you begin exerting force to try and overcome the muscle. With true weakness there is a smooth movement of the extremity in the direction in which you are exerting force; at the same time you feel a constant steady counter-resistance on the part of the patient.

What follows are descriptions and illustrations of commonly tested muscles as well as their innervations. There will be additional demonstrations of how to test the muscles on the video portion of the module and by your clinical preceptor. Some examiners may vary in just how the test is performed, and you may be exposed to more than one technique. Select the one that works best for you keeping in mind that you are striving for reliability and reproducibility in assessing muscle weakness.

The most common rating system for muscle strength gives a score of 5 for normal, (100 percent) strength, and 0 for total paralysis. 1, 2, 3, etc. note increasing strength in approximately 20 percent increments.

Muscles
The underlined root carries the majority of innervation to the listed muscle.

Neck Flexors (C 1-6)
Test: The head is flexed to the chest. The examiner places his hand on the patient’s forehead and exerts backward pressure, trying to place the head in the normal upright position. The patient resists. (Figure 2-46)

Neck Extensors (C1–T1)
Test: The patient extends the head backward and resists the examiners attempt to push the head forward (Figure 2-47).

Neck flexors and extensors usually are affected by myopathies, and not by root lesions because of the number of different roots innervating these muscles.

Before testing the neck flexors and extensors make sure there is no bony neck injury that might be worsened by these maneuvers. Patients with rheumatoid arthritis may have lax ligaments binding the C1 and C2 vertebrae and the above maneuvers may cause vertebral subluxations.
Upper Extremity

Shoulder Girdle

**Infraspinatus** (C 5,6: Suprascapular nerve)

Action: External rotation at the shoulder.

Test: The patient flexes at the elbow, with his elbows at his side. The examiner exerts force at the dorsal wrist or forearm, trying to push the forearm inwards towards the patient’s abdomen (Figure 2-48).

**Figure 2-48**: Infraspinatus muscle (external rotation at shoulder).

**Pectoralis major** (C 5–T 1)

Action: Internal rotation at the shoulder.

Test: Same position as above, but the examiner pushes outward against resistance (Figure 2-49).

**Deltoid** (C 5,6: Axillary nerve)

Action: Shoulder abduction.

Test: The patient holds his proximal arm out laterally at 90 degrees of abduction, and the examiner exerts force in a downward direction (Figure 2-50).

![Figure 2-49: Pectoralis major (shoulder adduction).](image1)

![Figure 2-50: Deltoid muscle (arm abduction and elevation).](image2)

Arm

**Biceps** (C 5,6: Musculocutaneous nerve))

Action: Flexion of the forearm at the elbow.

Test: The patient flexes the arm to about 45 degrees, forearm supinated, and the examiner tries to extend it against resistance (Figure 2-51).

**Triceps** (C6, 7, 8: Radial nerve)

Action: Extension of the forearm at the elbow.

Test: The forearm is flexed to about 70 degrees with the forearm fully supinated. The examiner tries to push it in the direction of flexion against resistance by the patient (Figure 2-52).
Forearm

**Brachioradialis** (C 5,6: Radial nerve)

Action: Flexion of the forearm at the elbow.

Test: The forearm is flexed to about 70 degrees with the forearm midway between pronation and supination. The examiner again pulls in the direction of forearm extension, against patient resistance (Figure 2-53).

**Extensor Carpi Radialis Longus and Brevis** (C 6,7: Radial nerve)

Action: Extension of the hand at the wrist.

Test: The patient extends the wrist and holds that position while the examiner pushes downward in the direction of flexion (Figure 2-54).

**Extensor Digitorum Communis** (C 7,8: Radial nerve)

Action: Extension of the fingers.

Test: The patient keeps the fingers extended. While supporting the wrist with his left hand the examiner exert downward pressure on the extended fingers, pushing them in the direction of flexion (Figure 2-55).

**Pronator Teres** (C 6,7: Median nerve)

Action: Pronation of the forearm.

Test: The arm is flexed, with elbow at the side of the trunk. The forearm is pronated. The examiner grips the patient’s hand and tries to supinate the forearm against resistance (Figure 2-56).
Flexor Carpi Radialis (C 6, 7: Median nerve)
Action: Flexion of the wrist at the hand.
Test: The patient flexes the hand at the wrist. The examiner pushes in the direction of extension against resistance by the patient (Figure 2-57).

Flexor Digitorum Sublimis and Profundus (C 7, 8: Median nerve, [ulnar nerve supplies the profundus to the forth and fifth fingers])
Action: Flexion of the fingers.
Test: Flexion of the fingers, Examiner tries to open them against resistance (Figure 2-58).

Hand
Abductor pollicis brevis (C 8, T 1: Median nerve)
Action: Moves the thump perpendicular to the plane of the palm (palmar abduction).
Test: The thumb is placed in palmar abduction and the examiner pushes it towards the dorsum of the hand. (Figure 2-59).

Figure 2-59: Abductor pollicis brevis (median nerve).

Interossei (C 8, T 1: Ulnar nerve)
Action: Abduction of the fingers.
Test: It is easiest to test the index finger. The second–fifth fingers are held to support the hand and the index finger is pushed inwards to overcome abduction (Figure 2-60).
Hypothenar (C 8, T1: Ulnar nerve)


Test: (ADQ) Push the abducted fifth finger towards adduction. (FDQ) Extend fifth finger, against attempt to keep it flexed (Figure 2-61).

Figure 2-60: Interossei (1st Dorsal) (finger abduction).

Figure 2-61: Hypothenar (abductor digiti quinti).

Lower Extremity

Hip Girdle

Muscles

Iliopsoas (L 2,3,4: Femoral nerve)

Action: Flexion of the thigh at the hip.

Test: In the lying or sitting position, the patient flexes the thigh at the hip. The examiner pushes downward at the knee, towards hip extension. (Figure 2-62).

Gluteus Maximus (L 5, S 1,2: Inferior gluteal nerve)

Action: Extension of the thigh at the hip.

Test: With the patient sitting or standing the patient pushes, (extends), his thigh downward into the chair or bed, against the examiner’s attempt to elevate the thigh by lifting upwards under the heel. (Figure 2-63).

Gluteus Medius (L 4,5, S 1: Superior gluteal nerve)

Figure 2-62: Iliopsoas.

Figure 2-63: Gluteus Maximus.
Action: Abduction of the thigh.

Test: While sitting or lying the patient holds the thigh in the outward abducted position, against the examiner’s attempt to push it inward towards adduction. (Figure 2-64).

**Thigh**

**Quadriceps Femoris** (L 2,3,4: Femoral nerve)

Action: Extension of the leg at the knee.

Test: The patient extends his leg, at the knee, to about 170 degrees. The examiner tries to flex the leg at the knee while the patient resists. (Figure 2-65).

Hamstrings

External = Biceps Femoris (L 5, S 1,2: Sciatic nerve)

Internal = Semitendinosus; Semimembranosus (L 4,5, S 1,2: Sciatic nerve)

Action: Flexion of the leg at the knee.

Test: The leg is flexed at the knee. The examiner tries to extend the leg against resistance by the patient (Figure 2-66).

**Adductors** (Adductor Magnus, Longus, Brevis) (L 2,3,4: Obturator nerve)

Action: Adduction of the thigh.

Test: The patient holds the knees in fairly close proximity. The examiner tries to individually force them apart against resistance by the patient (Figure 2-67).
Distal Leg

**Anterior Tibial** (L 4,5: Deep peroneal nerve)

Action: Dorsiflexion of the foot at the ankle.

Test: The patient dorsiflexes the foot and the examiner pushes downward towards plantar extension. Alternatively, to detect mild weakness, the patient is asked to walk on his heels. With normal strength each foot should stay equally dorsiflexed and the toes not touch the ground while walking. (*Figure 2-68*).

**Peroneus Longus, Brevis** (L 5, S 1: Superficial peroneal nerve)

Action: Eversion of the foot at the ankle.

Test: The patient holds his foot in the everted position and the examiner pushes inward towards inversion (*Figure 2-69*).  

---

---

**Toe Extensors** (Extensor Hallucis and Digitorum) (L 4,5, S 1: Deep peroneal nerve)

Action: Extension of the toes.

Test: The patient extends the toes upward and holds them there against the examiner’s attempt to push them downwards towards flexion. (*Figure 2-70*).

**Posterior Tibial** (L 5, S 1: Posterior tibial nerve)

Action: Inversion of the foot at the ankle.

Test: The patient holds his foot in the inverted position while the examiner pushes outward towards eversion. (*Figure 2-71*).
Gastrocnemius (L 5, S 1,2: Tibial nerve)

Action: Plantar flexion of the foot at the ankle.

Test: The patient holds his foot plantar flexed while the examiner tries to dorsiflex it against resistance. Subtle weakness may be detected by having the patient walk on his toes and observing if the heel comes closer to the ground when stepping off the affected side (Figure 2-72).

Toe Flexors (Flexor Hallucis and Digitorum) (L 5, S 1: Posterior tibial nerve)

Action: Flexion of the toes.

Test: The patient flexes his toes and the examiner tries to extend them against resistance by the patient. (Figure 2-73).

Abdominal Muscles (T6–L1)

Action: Flexion of the trunk.

Test: The patient lies supine and flexes his neck. The abdominal muscles are observed to tighten. The mid abdomen (umbilical level) is innervated by T-10, a frequent site of spine metastatic lesions. Spinal lesions at this level often cause weakness below T-10. This can be detected in the abdominal muscles by Beevor’s sign. The patient lies supine and flexes his neck while the examiner holds a pen over the umbilicus. When the abdominal muscles tense the stronger upper abdominal muscles pull the umbilicus upward which is made easier to observe by holding a pen over the original umbilical location.

Rectal Sphincter (S 3,4: Pudendal nerve)

Action: Constriction of the anus.

Test: The examiner performs a rectal examination and notes rectal tone and contractile ability on command. Decreased tone and contractile ability denotes a lower motor neuron lesion. When associated with an atonic bladder (overflow incontinence), it is almost always due to a lesion of the conus medullaris, (distal end of the spinal cord) or the cauda equina (distal lumbar and sacral nerve roots before they exit the spinal canal).

Summary
- Weakness is loss of strength in individual muscles or groups of muscles, not fatigue.
• Weakness should be defined in terms of its pattern. (Table 1)
• When testing muscle strength the examiner should exert the force and note the degree of resistance of individual muscles to determine degree of weakness.

Evaluation of Speech and Language
Disorders of speech and communication are numerous and some of the neuroanatomical pathways are complex. For purposes of this examination we will be dealing with broad concepts and will limit our discussion to clinically relevant and common disturbances.

Aphasia (dysphasia). A disorder of speech where the patient has trouble understanding speech, (in the absence of hearing problems), or in the thought and word-finding processes of speech. There is a defect in the comprehension and/or expression of language. Aphasia refers to the absence of speech and dysphasia to a less complete disorder of speech. There are different types of aphasia depending on where the lesion is located (Figure 2-74).

Wernicke aphasia (sensory aphasia, receptive aphasia, fluent aphasia.). This is caused by lesions of the posterior portion of the superior temporal gyrus (Wernicke’s area). The disorder is characterized by copious speech that is not intelligible because of incorrect word and syllable choice. The patient does not understand what he is saying or what is said to him. If a patient is hungry he will speak volumes but not be able to convey the simple message that he wants to eat. If the lesion involves the surrounding cortex there may be contralateral sensory loss or a homonymous visual field defect.

Broca aphasia (motor aphasia, expressive aphasia, nonfluent aphasia). It is caused by lesions of the inferior portion of the left frontal gyrus and its underlying white matter. The patient understands speech but speech production is distorted. There is difficulty with speech fluency and organization and sentences have few words (telegraphic speech). Unlike the patient with a fluent dysphasia, patients can understand what they themselves and others are saying and can convey ideas. In the example of the starving patient he might communicate his plight by saying “hungry...eat”. If the lesion involves the surrounding cortex the patient will also have upper motor neuron right facial and hand weakness.

Global aphasia. A large lesion affecting both speech areas and their connections leaves the patient mute and unable to comprehend speech. There is also an associated dense contralateral hemiplegia. This can be seen with acute infarcts in the dominant hemisphere, usually left middle cerebral or carotid artery distribution.
**Dysarthria.** Speech comprehension and expression are intact but an articulation problem exists which affects word pronunciation. There are different types of dysarthria, which reflect the level of the neuraxis affected.

**Dysphonia.** A mechanical or psychological disturbance of voice production. This can be seen in patients with laryngectomies, vocal cord paralysis, or laryngitis. It is recognized by the quality of speech and the diagnosis confirmed by demonstration of the suspected underlying cause.

If you think a patient is confused, test him for aphasia by giving him verbal commands to follow. This will test for Wernicke’s aphasia. Be sure not to give the patient visual cues. Families will often insist that an aphasic patient understands them. They demonstrate by asking the patient to wiggle his fingers but at the same time wiggle their fingers in front of him. The patient then responds to the visual cue. If one asks him to wiggle his fingers without simultaneously showing what is wanted, he will not comply.

**References**


**Self-Assessment Questions**

Please choose the correct answer for the following.

1. Memory can be impaired with:
   - A. decreased motivation
   - B. symptoms of depression
   - C. inattention
   - D. all of the above

2. The anatomy of memory involves all EXCEPT the:
   - A. hippocampus
   - B. subthalamic nucleus
   - C. dorsomedial nucleus of the thalamus
   - D. fornix
   - E. mammillary bodies

3. Disturbances in calculations are seen in lesions of the:
   - A. Non-dominant parietal lobe
   - B. thalamus
   - C. angular gyrus of the dominant hemisphere
   - D. cingulate gyrus
4. Match pupil size with lesion.
   - Metabolic disease: A. Pinpoint pupil
   - Midbrain lesion: B. 4–5 mm fixed pupil
   - Pontine lesion: C. 2 mm and nonreactive
   - Thalamic lesions: D. Sluggishly reactive
   - Mass effect with herniation: E. Unilateral dilated pupil

5. The doll’s eye maneuver: (Please circle the correct answers for the following)
   - should only be done after cervical spine disease or fracture is ruled out
   - is done with the head of the bed raised 30 degree
   - is positive when the eyes move toward the cold water stimulus on the tympanic membrane
   - all of the above

6. Decorticate posturing is:
   - manifest as tonic adduction and extension of the arms and legs
   - suggests a lesion at the level of the pons
   - manifest as tonic adduction and extension of the lower extremities only
   - manifest by tonic flexion of the arms and extension of the legs

7. The primary sensory cortex is located in the:
   - frontal lobes
   - parietal lobes
   - occipital lobes
   - precentral gyrus
   - none of the above

8. Root lesions are:
   - associated with pain
   - most frequent in the thoracic spine
   - never associated with sensory loss
   - none of the above

9. All are true EXCEPT:
   - proprioceptive fibers and touch fibers travel in the ipsilateral dorsal columns.
   - pain and temperature fibers travel in the contralateral lateral spinothalamic tract
   - impairment in 2-point discrimination implies a lesion in the thalamus
   - vibration is tested with a 256 Hz tuning fork on a distal bony prominence
10. The extra pyramidal system:
   A. receives input from the primary motor cortex
   B. consists of subcortical nuclei called the basal ganglia
   C. receives input from the motor cortex
   D. degeneration can lead to movement disorders
   E. all of the above

11. The neurological exam in a patient with Parkinson’s disease will show all EXCEPT:
   A. tremor
   B. rigidity
   C. flexed posture
   D. hyperkinetic speech
   E. Bradykinesia

12. The pyramidal system:
   A. effects voluntary movements
   B. begins in the cortex, the fibers travels in the internal capsule and travel ipsilateral in the spinal cord fibers
   C. descend in the medial corticospinal tract
   D. lesions cause loss of legs tendon reflexes

13. The cerebellum helps control motor coordination. Which are true:
   A. lesions that affect the vermis produce limb ataxia
   B. lesions of the anterior lobe produce gait ataxia
   C. lesions of the lateral hemispheres produce truncal ataxia
   D. lesions are contralateral to the affected side

14. Peripheral nerve lesions may produce all EXCEPT:
   A. muscle atrophy
   B. sensory loss
   C. weakness
   D. increased deep tendon reflexes
   E. distal paresthesias on tapping the lesion site

15. MATCHING
<table>
<thead>
<tr>
<th>Muscle</th>
<th>Nerve Roots</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quadriceps</td>
<td>A. L4-5</td>
</tr>
<tr>
<td>Biceps</td>
<td>B. L2, 3, 4</td>
</tr>
<tr>
<td>Rectal Sphincter</td>
<td>C. L5, S1, S2</td>
</tr>
<tr>
<td>Anterior tibial</td>
<td>D. C5, 6</td>
</tr>
<tr>
<td>Gluteus Maximus</td>
<td>E. S3, 4</td>
</tr>
</tbody>
</table>
16. MATCH TYPE OF APHASIA WITH DEFICIT

- Wernicke’s: A. inability to repeat
- Broca’s: B. mute and unable to comprehend
- Conduction: C. understands, but cannot produce speech
- Global: D. can repeat, but may not make sense or may be able to find words
- Transcortical: E. Copious speech that is not intelligible

Vignette

A 66-year-old retired schoolteacher was referred for headaches. The patient’s headaches dated back to age 30, when she developed migraine headaches. They were characterized by right-sided throbbing pain associated with nausea, vomiting, and photophobia. For the most part, her migraines were under good control with propranolol, but occasionally she took sumatriptan subcutaneously for breakthrough headaches. The patient’s headaches worsened in the three to four months before consultation. Although they varied in intensity, the overall severity had increased during this period. The headaches occurred daily and were aggravated by activities such as stooping, bending or straining to have a bowel movement. The pain was localized principally at the back of the head now and was dull in character. Within the previous four to six weeks, she avoided gardening because stooping over to pull out weeds exacerbated the severity of the headaches. During the past few weeks, she also experienced intermittent vomiting. The patient ascribed this to “nerves” as she felt increasingly anxious, but could not identify why. On further questioning, the patient admitted that she suffered from a slight limp for several years, which she attributed to an old back injury.

Neurological exam revealed normal tone and moderate impairment of strength in the left leg. Pin prick, vibration and proprioception were intact. Deep tendon reflexes were equal in the arms, but increased in the left leg, compared to the right. Left Babinski was present while the right Babinski was equivocal. On ambulation, circumduction of the left leg was apparent.

17. What features of the patient’s exam suggest an upper motor neuron lesion?

- A. weakness
- B. hyperreflexia
- C. Babinski
- D. circumduction
- E. all of the above
- F. all but D

18. The most likely cause of the patient’s leg weakness is:

- A. poorly controlled complicated migraines
- B. lumbar cord compression from an old vertebral fracture
- C. meningioma of the falx
- D. ependymoma of the upper cervical cord
- E. pontine glioma
Discussion: Meningiomas are benign, slow growing neoplasms and the brain accommodates to slow growth. Consequently clinical signs may not develop until the tumor reaches significant size. The leg is primarily affected since this tumor overlies the parasagittal primary motor cortex representing the lower extremity. (See Figure 2-22). Parasagittal meningiomas may also produce focal motor seizures (starting in the leg), which may then secondarily generalize.

A 52-year-old housewife presented with generalized weakness. Her illness commenced about ten days ago when she suffered from nausea, vomiting and diarrhea. About four to five days later, she experienced tingling in both hands so that she was unable to hold a cup or use a knife and fork effectively. During the next few days, the weakness extended into her legs. At this stage, she was referred for consultation. Her past medical history was remarkable for a gastric ulcer, which was successfully treated medically. She has had no further symptoms of ulcer and her weight has slightly increased in the past year.

The patient was afebrile and blood pressure was 180/90 mm Hg. Physical examination was remarkable for palpable lymph nodes on both sides of the neck which were discrete, mobile and non-tender, the largest being about 2 cm in diameter. On neurologic examination, facial expression was immobile. She had difficulty holding air in both of her cheeks or pursing her lips. Blinking was diminished. The patient could not close her eyes completely on request and when she attempted to do so, it could be seen that the eyeballs turned upwards. There was hypnotic and weakness of all limbs to the point that the patient had great difficulty lifting her limbs off the bed. Sensory exam revealed loss of pinprick, vibration and proprioception in the hands and feet. Deep tendon reflexes were absent in the arms and legs. Babinski could not be elicited bilaterally. Chest X-ray was normal. CBC demonstrated normal WBC and hemoglobin. Chem 7 revealed mild hyponatremia of 128. Lumbar puncture yielded clear CSF with an opening pressure of 170 mm of water. CSF protein was 220, glucose 60, WBC 0 and RBC 10.

19. The patient’s inability to close her eyes completely is due to:
   A. bilateral upper motor neuron weakness of the facial nerve
   B. bilateral lower motor neuron weakness of the facial nerve
   C. bilateral frontalis muscle weakness
   D. bilateral oculomotor nerve palsies
   E. an abnormality of neuromuscular transmission

20. Weakness of the limbs is due to:
   A. acute inflammatory demyelinating polynévropathy (Guillain-Barré syndrome)
   B. subacute combined degeneration of the spinal cord from B12 deficiency
   C. cytomegalovirus polyradiculopathy
   D. myasthenia gravis
   E. lead neuropathy
21. Loss of pin prick, vibration, and proprioception may be due to:
   A. cytomegalovirus polyradiculopathy
   B. infectious myelopathy
   C. dorsal column dysfunction and sensory neuropathy from B12 malabsorption
   D. the effect of botulinum toxin at the neuromuscular junction
   E. none of the above

22. Loss of deep tendon reflexes may due to:
   A. acute inflammatory demyelinating polyneuropathy
   B. sensory neuropathy from B12 deficiency
   C. Subacute combined degeneration of the spinal cord from B12 deficiency
   D. A or B
   E. B or C

The patient is a 58-year-old lawyer who was referred with the complaint of weakness. Apart from an illness affecting her legs at age of 9 years, which had been diagnosed as poliomyelitis, she was in good health until 2.5 years prior to presentation. She first noticed that her left foot and leg became “tired and tended to drag” when she walked for several minutes. After a few weeks she noted a definite weakness in the left leg even at rest. This weakness progressed to involve the right leg and foot similarly within two or three months. Her hands later became weak so that she experienced difficulty writing or unscrewing bottle tops, and frequently dropped objects such as cups and utensils. During the last six months her speech became less distinct and solid foods often stuck in her throat upon swallowing. There was no nasal regurgitation of liquids, but at night, in bed, she frequently had difficulty clearing mucus from the back of her throat. In the past month, she required assistance with ambulation, complaining of easy fatigue. Her fingers felt clumsy and weak such that dressing became laborious, particularly when buttoning was required. During this period of illness, the patient’s weight dropped from 136 lbs. to 100 lbs.

Neurologic examination was remarkable for normal cognitive function. There was nasal intonation of voice and mild slurring of speech. The tongue was wrinkled. Fasciculations appeared to be present when the tongue as at rest in the floor of the mouth. Upon gross observation of the body, generalized loss of muscle bulk was evident. In general, the legs were more wasted than the arms. The intrinsic hand muscles were atrophic. Fasciculations were conspicuous in the shoulder girdle, biceps, triceps, quadriceps and calf muscles. Tone was diminished throughout, particularly in the arms. Strength was diminished throughout, with the greatest weakness noted where muscle atrophy was present. Sensory exam was normal. No difficulty with finger–nose–finger and heel-to-shin tests. Deep tendon reflexes were exaggerated and Babinski was elicited bilaterally. Jaw jerk was brisk. Gait was slow with short shuffling steps and evinced a poverty of knee flexion.
23. The most likely cause of generalized weakness is:
   A. cervical cord compression from a herniated disc
   B. Chronic inflammatory demyelinating polyneuropathy
   C. brainstem glioma
   D. none of the above

24. What feature of the patient’s exam suggests lower motor neuron disease?
   A. wrinkled tongue with fasciculations
   B. diffuse hyperreflexia
   C. slurred speech
   D. slow, shuffling gait
   E. none of the above

25. Which of the following suggests upper motor neuron disease?
   A. brisk jaw jerk
   B. fasciculations
   C. atrophy of intrinsic hand muscles
   D. A and B
   E. A and C

26. What feature(s) of the patient’s exam is compatible with myopathy?
   A. weakness
   B. wrinkled tongue
   C. fasciculations
   D. B and C
   E. none of the above

27. Brisk deep tendon reflexes in the limbs and bilateral Babinski may be due to:
   A. poliomyelitis
   B. C2-3 herniated disc with cord compression
   C. pontine glioma
   D. A or B
   E. B or C
   F. A or C

28. Nasal intonation of speech, slurred speech and difficulty swallowing in this patient is due to pathology involving the
   A. motor cortex
   B. Broca’s area
   C. white matter of the brain
   D. brainstem
   E. none of the above
Answers

1. D
2. B
3. C
4. Metabolic disease D
   Midbrain lesion B
   Pontine lesion A
   Thalamic lesions C
   Mass effect with herniation E
5. A
6. D
7. B
8. A
9. C
10. E
11. D
12. A
13. B
14. D
15. Quadriceps B
   Biceps D
   Rectal sphincter E
   Anterior tibial A
   Gluteus maximus C
16. Wernicke’s E
   Broca’s C
   Conduction A
   Global B
   Transcortical D
17. E
18. C
19. B
20. A
21. C
22. D
23. E
24. A
25. A
26. A
27. E
28. D