Weakness and Numbness

I. CASE HISTORIES

Case 9-1. A 45-year-old man presents to the office because of generalized weakness for several days and intermittent diplopia for several weeks. He reports the weakness worsens at the end of the day. Past history is remarkable except for mild hypertension. Physical examination shows a man with average muscle bulk. Cranial nerves are intact, including extraocular movements. Ptosis is present bilaterally. Grip and gait are normal as are reflexes. The patient ambulates easily.

1. What might be the cause of his symptoms?

Case 9-2. A 38-year-old woman presents to the office reporting numbness in her perineal area for three days. She denies weakness. Past medical history is remarkable for insulin-dependent diabetes for 10 years and chronic back pain. She reports subjective fevers but is afebrile today. Physical examination shows a superficial decubitus on the right buttock; the patient is insensate in that area, and testing with pinprick reveals subjectively diminished sensation across the perineum. Reflexes are normal, and Babinski sign is absent.

1. What additional questions would be of help in evaluating this patient?
2. Where in the nervous system might this clinical picture be potentially located?

II. DISORDERS

Weakness and numbness are often nonspecific complaints used by patients to refer to non-neurologic problems. Weakness often equates with malaise or general fatigue, and systemic causes should be considered before jumping to the possibility of neurologic disease. Likewise, numbness may be used to describe a variety of sensory disturbances from pain to paresthesias and has many systemic, vascular, and non-neurologic causes.

The first step is to clarify the complaint. If the patient's issue is thought to be neurologic in etiology, an anatomic approach attempting to localize the area of dysfunction in the nervous system is useful. The next step is to then infer a potential etiologic cause based on temporal course and the anatomic localization.

Time Course	Brain	Spinal Cord	Nerve	Neuromuscular Junction	Muscle
Acute					
Weakness	Stroke TIA Multiple sclerosis	Spinal cord injury/compression (tumor, abscess), transverse myelitis	Guillain-Barré Neuropathy, compressive	Tick paralysis Botulism	Hypokalemia Toxins
Numbness	Stroke TIA Multiple sclerosis	Same as above	Guillain-Barré		
Subacute					
Weakness	Tumor Mass lesion Multiple sclerosis	ALS Spinal cord injury/compression (tumor, abscess) , compression (see above)	Disc disease	Myasthenia gravis Eaton-Lambert Polymyositis	Myopathies Myositis
Numbness	Multiple sclerosis Tumor Mass lesion	B12 deficiency	Disc disease Neuropathy	N/A	N/A
Chronic					
Weakness	Stroke residual Old CNS insult Multiple sclerosis	ALS Spinal cord injury/compression (tumor, abscess)	Neuropathy	Myasthenia gravis	Myopathy Muscular dystrophies
Numbness	Stroke residua Old central nervous system insult Multiple sclerosis	Spinal cord injury/compression (tumor, abscess)	Neuropathies, diabetic, others	N/A	N/A

TIA = transient ischemic attack; ALS = amyotrophic lateral sclerosis; CNS = central nervous system.

III. HISTORY

Table 10-2 Important Historical Points

Symptom	Ask the Patient
Weakness and numbness	When did you first notice the problem? How rapidly has it changed?
Weakness	Are you having any double vision or trouble swallowing? Any eyelid drooping? Is the weakness fatigable? Are there any specific activities that seem to bring out the weakness? Is there weakness in rising from seated position to standing? (Suggests proximal muscle weakness) Does your strength improve with rest?

Numbness	**How long has this sensation been present?** **Where do you feel the sensory alteration? Is the numbness worse at nighttime? Do you have to shake your hands at night to get rid of the numbness?**

Compression neuropathies are very common, especially in patients with diabetes since their nerves are already compromised. Carpal tunnel syndrome causes numbness in the fingers and hand that is usually more prominent at night. A common complaint is that patients will awaken at night with numb hands and have to "flick" their hands to "wake them up." Weakness can also occur in the grip. Ulnar neuropathy causes numbness in the fourth and fifth digits and can cause diffuse hand weakness. Lateral femoral cutaneous neuropathy (meralgia paresthetica) causes numbness on the lateral thigh between the hip and the knee and is caused by pressure on the nerve as it crosses over the pelvic rim. Peroneal neuropathy can cause numbness on the dorsum of the foot and lateral calf, as well as footdrop. This typically occurs from crossing the legs at the knees ("crossed-leg palsy").

IV. EXAMINATION

The anatomic distribution of the weakness or numbness, both from the patient's description and from the examination (Motor Exam Video 10-2; Sensory Exam **Video 10-4**), is a major tool to localize the problem and guide further investigations. Examination starts with inspection. Any loss of muscle bulk or atrophy should be noted.

Fasciculations (Video 10-1) or brief twitches of the muscle fibers, should be noted if present. An assessment of muscle tone should be made. Asking the patient to stand and walk (Chapter 8) is a powerful test if one is a careful observer. Proximal muscle strength is assessed as the patient arises from a sitting position. While walking, circumduction of a leg suggests weakness on that side as may loss of the arm swing associated with walking. The width of the gait and difficulty with turning should be observed.

Asking the patient to stand with the feet together, arms stretched in front and palms turned upward ("like you're catching rain") tests balance, motor strength, and sensation. Unilateral upper extremity weakness may be detected by observing pronation of the outstretched hand or downward drifting of the arm. Any asymmetry of the sustained posture suggests a motor problem. Asking the patient to close their eyes while maintaining this posture may accentuate the findings and also is the method to perform the Romberg test (see Chapter 8). Cranial nerve examination tests some muscle groups. Ptosis may be evident with observation and in some cases may be provoked with repetitive or sustained upward gaze. Extraocular movement testing assesses not only the cranial nerves, but also the functioning of the extraocular muscles. The patient may report diplopia, or disconjugate gaze may be evident. Other facial muscle movements should be observed for any asymmetry. Classically, with a lesion of the facial nerve, a "peripheral seventh" palsy is noted, which means that both the upper and lower face are weak (Bells Palsy Video 10-3). In a "central seventh" palsy, as is common with many strokes, there is weakness of the lower face but preserved function without detectable asymmetry of the upper face.

Muscle stretch reflexes are an often-overemphasized part of the examination. They frequently serve to confirm impressions formed by history and other parts of the examination. Reflexes are valuable when asymmetric in certain patterns, such as brisk reflexes on the right or left side of the body, or upper extremities versus lower extremities. In cases of central nervous system (CNS) injury or disease, reflexes frequently become exaggerated or "brisk," and the pattern of abnormal reflexes may be useful in localization. Often there is some time delay in reflexes becoming abnormal, and they are of limited use in an acute neurologic problem. "Upgoing toes," or the presence of Babinski responses, are a companion sign of CNS injury.

Sensory testing (**Video 10-4**) is the most subjective part of the examination and is frequently frustrating for both the patient and the examiner. Given the time limitations of the office, it is difficult to spend more than a few moments on this portion of the examination. A clean pin may be used to verify the patient's subjective complaints and map out the anatomic distribution of the sensory disturbance. As with the motor examination, patterns of left-side versus right-side, or upper extremities versus lower extremities, should be noted as this may be a clue to localization. If one suspects carpal tunnel syndrome then a Tinnel Sign or Phalen Sign can be helpful.

V. CLUES TO DIAGNOSIS

The history is the most important part of the process. Associated medical problems, medications, the tempo of the illness, and the patient's subjective description of the distribution of the weakness and numbness are vitally important. The physical examination often serves to strengthen or weaken the historical impression.

For common sensory and motor patterns see the diagrams in chapter 1, "Introduction." Patterns of hemisensory alteration or hemiplegia suggest a CNS cause of the complaint, possibly a contralateral brain problem. Thus, a patient with onset of numbness of the face, arm, trunk, and leg of the right or left side may have a process involving the contralateral sensory cortex or the white matter involved with that associated cortex.

Transverse patterns of weakness or numbness with truncal levels suggest possible spinal cord etiology. Often the sphincter is involved in spinal cord diseases and the patient will give a history of urinary or fecal incontinence. Obtaining an unexpectedly large postvoid residual urine volume is sometimes the first indication of the presence of a neurogenic bladder.

Weakness or numbness in part of one extremity might suggest a peripheral nerve problem or a problem anywhere along the pathway from the entry or exit of the nerve fibers in the nerve roots, through the cervical or lumbar plexus, and the peripheral nerve. Brain and spinal cord problems may also present with symptoms or signs involving one extremity, however.

Bilateral paresthesias distal in the arms and legs (stocking-glove distribution) with sensation loss suggest widespread peripheral nerve disease consistent with a neuropathy.

VI. RED FLAGS AND WHEN TO REFER

The rapidity of symptom onset and associated symptoms and physical signs will govern the tempo of referral. Generally speaking, anatomic localization within the brain or spinal cord may prompt consultation. Severe generalized weakness, as well as any respiratory symptoms, suggests need for emergent referral to an emergency department.

Transient symptoms are not necessarily innocent. If weakness or numbness is in a half-body distribution, or if speech or vision in one eye is transiently altered, a transient ischemic attack (TIA) is possible and may represent the opportunity for intervention and stroke prevention.

Table 10-3 Time Frame for Evaluation and Referral: Weakness and Numbness

Evaluate and Refer	Acute	Subacute	Chronic

Immediate	**Stroke—possible hemisensory or hemiplegia** **Any respiratory symptoms** **Rapidly progressive weakness** **Paresthesias with incontinence** **Suspected TIAs resolved with motor, sensory, or language impairment**		
Within 72 Hours		**Ptosis** **Isolated diplopia** **Multiple sclerosis indolent symptoms**	
Within Two Weeks			**Distal sensory disturbance consistent with peripheral neuropathy** **Complicated migraines with sensory disturbances**

VII. CASE DISCUSSIONS

Case 10-1. Most patients presenting to the office or clinic with a chief complaint of weakness will not have a neurologic cause of their symptoms. However, the report of diplopia as well as the presence of ptosis does suggest a neurologic etiology of symptoms. Neurologic examination provided no clear clues. Key to this patient is the report of fatigability. Patients with myasthenia gravis are frequently suspected of having a psychiatric cause of their symptoms since the symptoms fluctuate over hours and days and the presence of clear neurologic symptoms is frequently lacking. **Video 10-5** gives a summary of clinical findings of Myasthenia Gravis and shows an examination of a patient with myasthenia gravis. This patient should be referred to a neurologist for further evaluation promptly—within days—to confirm the tentative diagnosis. The presence of significant generalized weakness or any respiratory symptoms should prompt immediate referral in an emergency department setting.

Case 10-2. The presence of a skin breakdown in the sacral area of this ambulatory patient suggests profound anesthesia. Spinal cord disease, specifically external compression of the cord or the terminal region of the cord (conus medullaris or cauda equina) should be at the top of the list of diagnostic possibilities, although other anatomic localization is possible as well. Cord compression is disabling with any neurologic deficits likely being permanent. Frequently neurologic signs of spasticity and abnormal reflexes take some time to develop, and their absence may be falsely reassuring to the examiner. Determination of postvoiding urine residual may suggest cord involvement with neurogenic bladder. Urgent neuroimaging with involvement of a neurologist should be pursued with the idea being to detect a potentially treatable cause of spinal cord compression. MRI is the test of choice, but local practice patterns may suggest a different imaging or referral strategy. Pursuing this strategy detected an epidural abscess with spinal cord compression. The classic presentation of spinal cord abscess is localized spine pain in a febrile patient, but at times epidural abscesses may present in a surprisingly indolent manner.

VIII. CME QUESTIONS

1. A 30-year-old woman reports two months of nightly pain in the right hand with the first three digits subjectively being numb. The numbness fluctuates throughout the day with some movements exacerbating the numbness. Grip is good. Tinel signs and Phalen maneuver are inconclusive. Reflexes are normal and symmetric. Which of the following is the most likely site of the lesion?

a. CNS—brain
b. CNS—spinal cord
c. Peripheral nervous system—mononeuropathy
d. Peripheral nervous system—radiculopathy

2. A 65-year-old patient with insulin dependent diabetes comes to your office with transient numbness in her left face, arm, and leg. The episodes have occurred three times in the past week, lasting about 20 minutes each. There is no associated weakness. Examination is unremarkable. Which of the following is the most likely cause of this syndrome?

a. Migraine
b. Peripheral neuropathy
c. Spinal cord disease
d. Transient ischemic attack

3. A patient with known myasthenia gravis presents to your office with progressive weakness and subjective dyspnea over two days. He has had a cold recently and has been prescribed antibiotics by another practitioner for suspected bronchitis. The patient has been stable on an anticholinesterase inhibitor for several months and reports no change in this medication. Which of the following actions would be prudent?
a. Increase anticholinesterase inhibitor
b. Perform office edrophonium (Tensilon) test
c. Refer to emergency department for further assessment
d.Stop antibiotic

4. A patient with a history of lumbar disc disease comes to the office with increasing low back pain and increased weakness in the left leg. Similar symptoms have been present since her lumbar decompression surgery two months ago, but the patient now reports urinary incontinence and some mild fecal soiling. Which of the following might be the best course of action?
a. Direct her to the emergency department for immediate evaluation
b. Initiate a neurology clinic visit
c. Obtain an outpatient MRI at earliest opportunity—approximately one week
d. Refer back to her surgeon at earliest appointment

5. A 25-year-old student presents to the office with two to three days of paresthesias in all extremities and a feeling that he is losing his strength in general. He walked to your clinic but states that he felt exhausted while doing so. Sensory examination is unremarkable. Overall strength is good. Reflexes are absent. Which of the following is a likely possibility?
a. Amyotrophic lateral sclerosis
b. Guillain-Barré syndrome

c. Multiple sclerosis
d. Spinal cord lesion