



Chapter 10 – Weakness

NOTE: CONTENT CONTAINED IN THIS DOCUMENT IS TAKEN FROM ROSEN'S EMERGENCY MEDICINE 9th Ed.

Italicized text is quoted directly from Rosen's.

Key Concepts:

This chapter in Rosen's 9th Edition does not have any associated core concepts, but we have compiled a list of four key things you need to remember when approaching your next weak patient:

1. Cast a broad net when assessing and investigating your patient complaining of generalized weakness. These individuals can harbour serious pathology, so be thorough.
2. Bell's Palsy will typically cause complete or partial paralysis of the upper and lower halves of the face; if you see forehead sparing, think of a more sinister intracranial pathology causing their weakness.
3. If crossed features are present (e.g., arm, leg, hand weakness with contralateral facial involvement), consider a brainstem lesion and perform a detailed cranial nerve examination. Vertebrobasilar insufficiency and demyelinating diseases should be at the forefront of your differential diagnosis list in these patients.
4. In patients presenting with symptoms of ascending bilateral lower limb weakness, consider Guillain-Barre Syndrome early and monitor frequently for evidence of impending respiratory compromise. Tachypnea, shallow respirations, and complaints of dyspnea should prompt you to investigate for potential respiratory muscular fatigue. Pulmonary Function Testing, ICU admission, and mechanical ventilation may be necessary in these patients.

Core Questions:

1. What structures are affected by UMJ, LMJ, and NMJ lesions, and what are causes of weakness associated with each?
2. What are common signs of UMN, LMN, and NMJ dysfunction?
3. What are (7) pathophysiologic causes of non-neurologic weakness (Box 10.1)?
4. What is the DDX of neuromuscular weakness? (Table 10.1)
5. Describe an approach to general weakness in the ED.



Wisecracks:

1. Differentiate between “plegia” and “paresis”.
2. List (5) DDX’s for non-neurologic weakness (based on pathophysiologic processes).
3. List (5) non-emergent causes of peripheral neuropathy (Box 10.2)
4. Explain how you recognize an ED patient that may be approaching the end of life.

Rosen’s in Perspective

Weakness is a common ED presentation, with up to 10% of ED patients presenting with this chief complaint. More than 50% of patients will be diagnosed with a serious condition – causes range from cardiac, respiratory, metabolic, and infectious pathology. This often presents in our older patients, as co-morbid disease. In addition to organic illness, the complaint of “generalized weakness” can also be the indicator of major depressive disorder. When considering the population that typically presents with this complaint at triage, it is important not to forget psychiatric illnesses on your differential. Wide is key here.

The key to working up weakness in the ED is differentiating neurologic causes of weakness (UMJ, LMJ, NMJ) vs. non-neurologic causes of weakness (electrolyte disturbances, CHF, sepsis, malignancy, etc). You have to cast a wide net, perform a thorough history, review of systems, and physical examination. Additionally, you may need to get collateral information from your EMR and family members presenting with the patient. We love our geriatric patients. However, the sweet little old lady presenting with weakness is often a terribly vague historian, with multiple co-morbidities and a non-localizing physical exam. Getting to the bottom of their complaint may take more time than you think!

In this episode, we will discuss “need to know” content for weakness in the ED, and we will give you an efficient, safe approach to these patients. Specifically, we will be providing you all with a cursory overview of the differential diagnosis for generalized weakness, evaluate different neurologic and musculoskeletal causes of weakness, and give you some information as to dispossess your next 84-year-old retiree. More information on the specific causes of weakness are spoken to in later chapters of Rosen’s, so check them out!

Core Questions:

[1] What structures are affected by UMJ, LMJ, and NMJ lesions, and what are causes of weakness associated with each?

Upper motor neuron (UMN) lesion: A lesion in the cerebral cortex or corticospinal tract of the brainstem and spinal cord.

Examples of UMN Disorders: CVA, MS, Intracranial malignancy



Lower motor neuron (LMN) lesion: Lesion of anterior horn, nerve root, axon, or peripheral nerve.

Examples of LMN Disorders: Diabetic Neuropathy, Radiculopathy, GBS

Neuromuscular junction lesion: disruption of normal signalling from pre-synaptic motor neuron to the post-synaptic muscle fiber.

Examples of NMJ Disorders: Botulism, Polymyositis, Dermatomyositis, Lambert-Eaton Syndrome

[2] What are common signs of UMN, LMN, and NMJ dysfunction?

Signs of upper motor neuron (UMN) dysfunction include:

[INDICATE LESION IN THE CEREBRAL CORTEX OR CORTICOSPINAL TRACT]

- Spasticity to extension in the upper limbs
- Spasticity to flexion in the lower limbs
- Hyperreflexia
- Pronator Drift
- Hoffman's Sign
- Babinski's Sign
- Preserved muscle bulk

Signs of lower motor neuron (LMN) dysfunction include:

[INDICATE LESION IN THE ANTERIOR HORN, AXONAL EXTENSIONS AT NERVE ROOT, OR PERIPHERAL NERVES]

- Flaccidity
- Depressed or absent reflexes
- Fasciculations
- Muscle cramps
- Diminished muscle bulk

Signs of neuromuscular junction (NMJ) dysfunction include:

- Slowly progressive/fluctuating course
- Bulbar or ocular signs
- UMN/LMN/Mixed picture can occur
- Proximal mm weakness > distal
- Decreased muscle bulk



[3] What are (7) pathophysiologic causes of non-neurologic weakness (Box 10.1)?

This is lifted straight from Box 10.1 in Rosen's 9th Edition.

Non-neurologic Weakness Box 10.1 Rosen's 9th Edition
<ul style="list-style-type: none"> ● Alterations in Plasma Volume ● Alterations in Plasma Composition ● Derangement in Circulating RBC's ● Decrease in Cardiac Pump Function ● Decrease in Systemic Vascular Resistance ● Increased Metabolic Demand ● Mitochondrial Dysfunction ● Global Depression of the Central Nervous System

[4] What is the DDX of neuromuscular weakness (Table 10.1)?

DIAGNOSIS	FEATURES
CRITICAL DIAGNOSES	
Cerebral cortex or subcortical	Ischemic or hemorrhagic cerebrovascular accident (CVA)
Brainstem	Ischemic or hemorrhagic CVA
Spinal cord	Ischemia, compression (disk, abscess, or hematoma)
Peripheral nerve	Acute demyelination (Guillain-Barré syndrome)
Neuromuscular junction	Myasthenic or cholinergic crisis Botulism Tick paralysis Organophosphate poisoning
Muscle	Rhabdomyolysis



EMERGENT DIAGNOSES	
Cerebral cortex or subcortical	Tumor, abscess, demyelination
Brainstem	Demyelination
Spinal cord	Demyelination (transverse myelitis) Compression (disk, spondylosis)
Peripheral nerve	Compressive plexopathy (hematoma, aneurysm) Paraneoplastic vasculitis uremia
Muscle	Inflammatory myositis

[5] Describe an approach to general weakness in the ED.

First - Evaluate clinically (H&P plus investigations)

Non-focal picture. Consider investigating the following differentials:

- Cardiac
- Metabolic
- Volume status and lytes.
- Infectious
- Endocrinopathy
- Functional Decline
- Etc.

In most patients - broad investigations. Start with labs, ECG and then imaging as directed by your findings. Dysfunction with basically any of the organ systems can cause this and your patient may have co-morbid disease to complicate things. **This is a very common ED presentation.**

For an approach to Focal Weakness - see Rosen's Figure 10.1



Wisecracks:

[1] Differentiate between “plegia” and “paresis”.

This one is for all of you keeners out there looking to impress the next pedantic Neurologist over the phone at 3 AM. We have been asked this a time or two, so here is your definitive answer:

- “-paresis” refers to moderate loss of power
- “-plegia” refers to complete loss of motion

It is as simple as that. Just a little weaaaakk with the pareeeeeesis.

[2] List a DDX for nonneurologic weakness .

Box 10.1

1. Alterations in plasma volume (dehydration)
2. Alterations in plasma composition (glucose, electrolytes)
3. Derangement in circulating red blood cells (anemia or polycythemia)
4. Decrease in cardiac pump function (myocardial ischemia)
5. Decrease in systemic vascular resistance (vasodilatory shock from any cause)
6. Increased metabolic demand (local or systemic infection, endocrinopathy, toxin)
7. Mitochondrial dysfunction (severe sepsis or toxin-mediated)
8. Global depression of the central nervous system (sedatives, stimulant withdrawal)

[3] List 5 non-emergent causes of peripheral neuropathy (box)

Box 10.2

1. Connective tissue disorder
2. External compression (entrapment syndrome, compressive plexopathy)
3. Endocrinopathy (diabetes)
4. Paraneoplastic syndromes
5. Toxins (alcohol)
6. Trauma
7. Vitamin deficiency

[4] Explain how you recognize an ED patient that may be approaching the end of life.

We are emerg docs and that means we like to fix problems! However, I wanted to include this section to get you to think about situations where it is appropriate to take a step back, initiate



some care-planning discussions, and help families understand the predicted trajectory of their loved one. **Elderly patients with ED visits for general weakness often have many of these indicators of decline present if you look for them... We are doing patients and families a service when we recognize signs that the patient may be nearing end of life and talk to them about it.**

As per GSF document, a UK-based resource. (Gold Standards Framework)

<https://www.goldstandardsframework.org.uk/content/uploads/files/General%20Files/Prognostic%20Indicator%20Guidance%20October%202011.pdf>

Step 1: Ask the “Surprise Question” in patients with advanced/multisystem disease – “Would you be surprised if the patient were to die in the next few months, weeks, days?”

Step 2: Does the patient have general indicators of decline and increasing needs? The following indicators are quoted from the GSF document.

Decreasing activity – functional performance status declining limited self-care, in bed or chair 50% of day and increasing dependence in most activities of daily living

- Co-morbidity is regarded as the biggest predictive indicator of mortality and morbidity
- General physical decline and increasing need for support
- Advanced disease - unstable, deteriorating complex symptom burden
- Decreasing response to treatments, decreasing reversibility
- Choice of no further active treatment
- Progressive weight loss (>10%) in past six months
- Repeated unplanned/crisis admissions
- Sentinel Event e.g. serious fall, bereavement, transfer to nursing home
- Serum albumen <25g/l

Step 3: Assess for indications of decline in specific clinical situations (see document)

For example

- in cancer patients who spend ≥50% of the time in bed, the prognosis is 3 months or less
- CHF patients with NHYA III or IV disease, severe sx despite optimal therapy
- COPD with recurrent hospitalizations in 3/12 mo, MRC 4 or 5 dyspnea
- etc

The goal is not to answer the question of “how long do I have.” Rather, getting patients involved in these kinds of discussions can help them plan to make the most of the time they have left, avoid crisis ED visits and admissions (potentially with early involvement of palliative care services), and ensure their symptoms are managed.