Cervical Myelopathy

Pathology of the cervical spinal cord. Usually results from cord compression in the setting of spinal canal stenosis; ossification of ligaments, degenerative disc disease.

Prevalence: est. 605 people per million. More common with age. If you evaluate patients because of falls or imbalance, you will encounter myelopathy and should know (1) how to screen for it and (2) when to advocate for further workup.

Patients may report loss of dexterity of the upper extremities, and/or clumsy gait. They may report neck and/or shoulder pain.

Gait abnormality may be the first symptom observed. Look for:

- Abnormal (often narrowed, sometimes widened) Base of Support
- Apparent delayed initiation of swing phase
- Impaired foot clearance
- Frank staggering.

Examination should include focused testing of:
Sensation: light touch localization, proprioception
Strength: myotomal screening and serial MMT
Reflexes: including Achilles, Quadriceps, Brachioradialis
Coordination of the upper and lower extremities

Because the dorsal column is often affected, it is crucial to assess proprioception thoroughly. Use isolated joint testing, but also test Romberg, eyes closed finger-to-nose, and heel-to-shin tests.

Check for gastroc clonus. Ask about bowel/bladder changes. Look at shoe soles.

Grading Deep Tendon Reflexes	
0	No response; always abnormal
1+	Slight but definitely present response; may or may not be normal
2+	Brisk response; normal
3+	Very brisk response; may or may not be normal
4+	Tap elicits repeating reflex (clonus); always abnormal

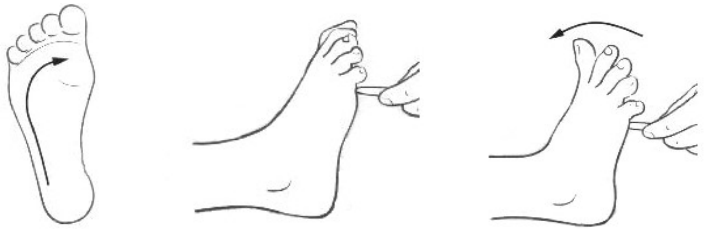
Cook's cluster can be used to help identify cervical myelopathy. A patient with 3 or more of the following findings is very likely (94% or greater) to have cervical myelopathy.

Cook's Myelopathy Cluster	
1	Gait deviation
2	Positive Hoffman's
3	Inverted Supinator sign
4	Positive Babinski
5	Age > 45 years

Hoffman's is performed by flicking the nail bed of the patient's middle finger downward and watching for thumb adduction.

Inverted supinator sign is tested by tapping the Brachialis' distal tendon with arm slightly pronated. A positive test looks like finger flexion and/or elbow extension when you test the reflex.

Positive Babinski looks like toe extension and "fanning":



Absence of abnormal reflexes alone does not rule out myelopathy. When this entire cluster is performed appropriately, patients with ≤ 1 positive finding are unlikely to have cervical myelopathy.

MRI is used to identify myelomalacia. Thoracic MRI may include lower c-spine and extend to conus (usually L1-L2). Degenerative changes are more common at C5-C6 and C6-C7 levels.

Naturally, consider presence of UE symptoms to help localize level of suspected lesion.

Often treated with surgical decompression / fusion, though myelopathy is not always caused by compression.

Mechanical Ventilation

Working with patients requiring mechanical ventilation (MV) can be intimidating at first. Mobilizing these patients is an established care standard, and PT intervention can produce enormous impact.

Ventilation can be invasive (e.g., the patient is endotracheally intubated), or non-invasive (e.g., BiPAP).

When patients require MV, often some sedation is used to facilitate ventilator tolerance while maintaining alertness within the desired range – typically**, RASS** between **0** and **-1**. Sedating agents affect much more than alertness, and **propofol**, a common first-line sedating drug, significantly reduces **systemic vascular resistance** and **stroke volume**.

Pressors may be used to maintain mean arterial pressure (MAP) within goal range. **Norepinephrine**, or **levophed**, is a frequently-used alpha-1 agonist which **produces peripheral vasoconstriction**, but also has **an inotropic effect** (it increases contractility of the myocardium). Determination of readiness for mobility is guided by trend in pressor requirement, not absolute values. Often, a little active mobility can improve BP in hypotensive patients with down-trending pressor requirements.

Two commonly-encountered ventilator modes for intubated patients are Volume Control/ Assist Control (**VC/AC**) and Pressure Support (**PSV**).

VC/AC ensures that patients receive the desired **tidal volume** (**Vt**) during each inspiratory phase. This is based on the patient's body size. **Respiratory rate** (**RR**) is also set, so a desired **Minute Ventilation** is achieved (Vt x RR**). PEEP**, or positive end expiratory pressure, is the pressure on the airway at the end of the expiratory phase which prevents atelectasis. **FiO2** is fraction of inspired Oxygen and this format (e.g. 0.50, rather than 50%) for this value is used because it enables easy calculation of the **P/F ratio**, which defines Acute Respiratory Distress Syndrome (**ARDS)** severity, guiding treatment approach.

Ventilator settings are documented like this: Mode/RR/Vt/PEEP/FiO2; e.g. VC/AC/24/440/10/0.50.

Pressure Support Ventilation assists the patient's inspiratory effort, meaning the patient initiates the inspiratory phase actively. The vent adds a set amount of pressure during the inspiratory phase but does not guarantee that a set tidal volume is attained. This mode requires inspiratory muscle activity and is often better tolerated by patients that are alert and mobilizing actively.

Typically for ventilator **weaning**, a setting of PSV 5/5 is trialed for 30 minutes, meaning that a patient breathes actively through the endotracheal tube (ETT) with 5 cm H20 of pressure support during inspiratory phase, and 5 cm H20 of PEEP. Patients often require greater than 5 PS to maintain Vt during active mobilization. With settings unchanged, Vt often improves significantly in upright sitting.

MV is used to facilitate gas exchange. Arterial Blood Gas draws examine **pH** (normal is 7.4; less is acidosis, more is alkalosis); **PaO2** and **PaCO2** (partial pressures of O2 and CO2, respectively), and HCO3-. Kidneys produce HCO3- to help regulate homeostasis, and when a metabolic derangement produces an acidotic state, HCO3- is used to buffer the rate of pH decrease.

MV affects more than just gas exchange. Positive pressure ventilation, invasive or not, increases **intrathoracic pressure** which reduces venous return. Because of this reduction in **preload** at the Right Atrium, **cardiac output** is reduced, which can affect all organ systems. AHRQ used to recommend deferment of mobilization until **PEEP < 10** is tolerated, and though patients are mobilized on higher PEEPs, this cardiopulmonary impact is important to keep in mind.

Patients who are endotracheally intubated often benefit from **airway clearance techniques** for mobilization of secretions. **Active mobilization** (e.g., walking, or at least standing to transfer to a chair) is encouraged.

Monitor vital sign response closely, particularly during position changes. FiO2 is sometimes increased temporarily, during mobility. Note the position of the ETT before and after activity - e.g., 23 cm at the lip.

Reference literature and other clinical resources at PTMeasures.com

Common Toxicities

in hospitalized elderly

Acutely abnormal mental status may be caused by structural problems (e.g., a lesion), metabolic problems (e.g., renal failure with electrolyte derangement), infectious problems (e.g., meningitis) and by toxic problems.

Older adults are at risk for polypharmacy, and those with some degree of chronic renal insufficiency, are particularly at risk for potential toxicities in the setting of drug interactions and impaired renal clearance.

A patient for example who is anticoagulated with warfarin can present with INR above goal range (and sometimes above detectable range) without dose change or med error – whether in the setting of reduced clearance of the drug or other physiologic change – and this will be identified with routine lab testing upon admission.

Some antibiotics cross the blood-brain barrier, including the cephalosporins ceftriaxone and cefepime.

Beta-lactam antibiotics: Mechanisms of action and resistance and adverse effects – UpToDate

Predicting risk of adverse drug reactions in older adults - PMC

Abnormal mental status may be a new deficit, or may occur with recrudescence of prior deficits, for example, caused by an old frontal stroke.

Delirium may under-appreciated among hospitalized elderly, but labeling acutely altered mental status as delirium means that a medically reversible cause is not present. Sometimes a reversible cause is present, but hasn't yet been identified.

Pancoast Syndrome

Pancoast lesions are tumors at the superior sulcus of the lung. Most are non-small cell lung cancer.

Incidence of Pancoast tumors specifically is 6,000 – 10,000 per year; lung cancer incidence is greater than 200,000 per year.

If the lesion has spread to involve the brachial plexus, patients may report pain in the areas of C8 – T1 distribution.

You may observe weakness and atrophy of _

As a hospital PT, you may encounter this syndrome in patients presenting to the ED with unrelenting shoulder pain, and/or LE weakness.

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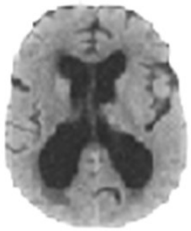
Normal Pressure Hydrocephalus

Intracranial pathology in which the brain doesn't absorb cerebrospinal fluid normally. Identified by clinical exam and confirmed by ventriculomegaly on Head CT. Can be idiopathic; may result from other conditions.



Incidence of idiopathic NPH: 2-20 people per million per year. Prevalence among 70-79 y/o is 0.2%, but among 80+ y/o, prevalence is 6%.

Characterized by clinical triad of:

1. Gait abnormality
2. Urinary incontinence
3. Abnormal cognition

Which is sometimes remembered as "wobbly, wet, and wacky."

Gait apraxia results from disruption of periventricular tracts involved with frontal lobe function, and looks **magnetic**, with:

- Apparent delay of initiation of swing phase producing appearance of feet "stuck" to the floor
- Shortened step length
- Impaired foot clearance
- Reduced gait speed

Because gait abnormality may be the clearest objective symptom of NPH, physical therapists will encounter cases of undiagnosed NPH and can help facilitate earlier, better care for patients that might need intervention.

NPH may be treated by ventriculoperitoneal shunt. To evaluate appropriateness of a VP as long-term treatment, patients undergo high-volume (30 – 50 mL) lumbar puncture.

Physical therapist examination and gait evaluation (1) before and (2) shortly after LP is instrumental in judging response to the intervention and guiding determination of long-term treatment.

Facility protocol or neurologist instruction may dictate when the post-LP gait evaluation should be performed. Usually, it is performed within one hour, since CSF is repleted at a rate of approx. 20 mL/ hour.

Carefully measure the following parameters as part of your pre- and post-LP gait assessment:

- Gait speed
- Distance ambulated in a given time interval
- Step length
- Step cadence
- Number of steps needed to turn in a circle
- Degree of assistance required

Of the 3 impairments which produce the clinical triad of NPH, gait abnormality is most responsive to treatment via VP shunt (and perhaps therefore most-responsive to the high-volume LP, though this has not been proven separately).

You may also evaluate apraxia specifically, for example using the Luria manual sequencing task or another test that can be easily compared pre- and post-intervention.

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Lambert-Eaton Myasthenic Syndrome

Paraneoplastic syndromes like Lambert-Eaton myasthenic syndrome (LEMS) produce deficits in the setting of malignancy through mechanisms other than metastasis and secondary complication.

In LEMS, autoimmune activity at the neuromuscular junction can cause weakness which may be apparent to the discerning clinician in even early small cell lung cancer (SCLC). LEMS can occur as an autoimmune problem without malignancy.

In LEMS, antibodies to voltage-gated Ca^{2+} channels limit acetylcholine release by the presynaptic neuron. This mechanism explains why repeated muscle contraction may paradoxically improve force production.

The incidence of LEMS is about 3 cases per million person=years, which is lower than the incidence of myasthenia gravis (MG), but by a smaller difference than its much lower prevalence might suggest if life-expectancy were comparable. 60-75% of patients with LEMS are male.

When the right physical exam techniques are used, the pattern of weakness specific to LEMS can be differentiated from global strength loss in the context of compromised nutrition, chemotherapy, and other systemic problems.

Patients may report perceived weakness of gradual onset, particularly functional weakness disproportionate to observed strength on MMT – difficulty ascending stairs or standing from a chair. Ptosis and diplopia are common cranial-nerve symptoms. Dry mouth is a commonly-reported symptom.

Look for a pattern of weakness affecting proximal muscles more severely than distal muscle groups. Weakness is usually symmetric and often affects the legs to a greater degree than the arms. When serial manual muscle testing is performed, force production may **improve** with repetition, rather than fatigue,.

Reflexes are often diminished or absent, though this may change as well if re-tested after repeated muscle contraction.

Cranial nerve involvement is common. Patients may present with bilateral upper lid ptosis, dysarthria, and dysphagia, prompting myasthenia gravis workup.

Many patients with LEMS develop dysautonomia. Sensory loss is not an expected finding in LEMS.

Patients may not have a diagnosis at all when you identify these symptoms – they may present to emergency departments or be admitted to hospitals with non-specific failure of self-care at home.

The responsibilities of the PT are to recognize the cluster of exam findings, communicate their significance to the care team, facilitate safe discharge planning, and adapt therapeutic intervention understanding the pathophysiology.

LEMS is confirmed by electrodiagnostic testing. Chest imaging is recommended for patients with LEMS, ACh-esterase inhibitors (pyridostigmine) may be used.

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Parkinsonism

Parkinsonism is a clinical syndrome involving **bradykinesia** plus at least one of the following: **tremor**, **rigidity**, **gait abnormality**. Parkinson Disease (PD), or idiopathic Parkinsonism, is caused by loss of dopaminergic neurons in the substantia nigra. PD generally responds to drugs like Sinemet, while Parkinsonism of other causes typically does not.

In hospital, consider timing PT treatment around Sinemet dosing in patients diagnosed with PD. Screen for movement disorder symptoms in patients with unexplained falls, or when injury resulting from a fall overshadows identification of the fall's cause.

Patients may report balance deficits, sleep changes, depression, micrographia and loss of smell early in disease course. In PD, symptoms often begin asymmetrically; reduced arm swing or rest tremor may be observed unilaterally.

Test **Rapid Alternating Movements** allowing sufficient repetition to observe for decrease in movement speed and amplitude – at least 10 reps. Cue the patient to perform fast, large movements. Use arm pronation/supination, or open/close fist, or thumb to finger tapping to test UEs. Use toe tap to test LEs.

Rigidity (unlike spasticity) is not velocity or direction dependent.

The Froment maneuver can help activate subtle cogwheel rigidity. While supporting the patient's arm, test passive range in a circular motion at the wrist. Instruct the patient to tap their knee with their contralateral arm. This may uncover cogwheeling on the side you are testing.

Observe for a pill-rolling UE tremor while the arms are at rest, in a pronated position.

Check for postural tremor by instructing patient to maintain position of 90 deg shoulder flexion with arms held forward. Look for action tremor with finger-nose-finger, without moving visual target.

Postural reflexes may diminish. Patients may demonstrate a posterior bias with delayed correction upon standing from a chair, or frank retropulsion.

The **Pull Test** can be useful: stand behind the patient and provide a posterior-directed external perturbation (by pulling from the anterior shoulder bilaterally) and evaluate whether the patient can recover balance with a step or two.

Gait abnormalities are common:

- Reduced trunk rotation and arm swing
- Shortened step length
- Narrowed base of support
- Impaired foot clearance
- Festinating - progressive exaggerated anterior cente of gravity displacement, with reduction in step lengt

Most people with PD develop **dysautonomia**. Consider compression stockings and abdominal binders. Reduced facial expression is common. Supranuclear gaze palsies may occur: ocular-motor range of movement (particularly vertical) may be reduced; visual smooth pursuits may look "choppy" – an appearance caused by saccadic intrusion. Cognition is often affected, and hallucinations may occur in Lewy Body dementia.

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Rigidity (unlike spasticity) is not velocity or direction dependent. Check for **cogwheeling** – a ratchet-like resistance to passive movement.

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Initiation of movement and "freezing" may present unique challenges. Auditory cueing may be helpful, particularly for gait cadence.

Most people with PD develop **dysautonomia**. Check 3-position VS; consider compression stockings and abdominal binders.

Supranuclear gaze palsies may occur: ocular-motor range of movement (particularly vertical) may be reduced; visual smooth pursuits may look "choppy" – an appearance caused by saccadic intrusion.

Consider Hoehn and Yahr Scale staging.

Other clinical syndromes may appear similar:

Progressive Supranuclear Palsy is defined by frontal dysfunction – impaired abstract thought early; abnormal verbal fluency, motor perseveration, behavioral disturbances later. Hypometric saccades are common. Downgaze palsy is the most specific impairment for PSP.

Lewy Body Dementia involves progressive cognitive deficits and visual hallucinations, as well as physical features of Parkinsonism.

Multiple Systems Atrophy can include Parkinsonian features, but is defined by dysautonomia (sometimes isolated, earlier than other features), may predominantly look like cerebellar ataxia, and often features dysphagia. Look for anterocollis (dystonia producing neck flexion).

Corticobasilar Degeneration involves executive dysfunction, aphasia, apraxia; limb rigidity, dystonia, myoclonus (sometimes one limb, occasionally symmetric), hyperreflexia (30-50%); sometimes limb agnosia.

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Generalized Weakness

Hospital PT evaluations include several core responsibilities:

1. Describe functional limitations
2. Identify the body systems impairments producing those limitations
3. Understand whether those impairments can be adequately explained by the known medical status and course
4. Select interventions and prognosticate functional recovery based on knowledge of body systems and disease processes

We're more effective contributors to the diagnostic process (and in our interventions) when we can recognize the patterns that impairments may form – we know what else to look for when the features of a clinical syndrome take shape, and we can effectively advocate for further diagnostic attention when we provide lots of clear, objective information.

Skillful PT exam can help differentiate between problems of the CNS, PNS, neuromuscular junction (and even between pre- and post-synaptic areas of the NMJ), and muscle itself.

When a patient presents with generalized weakness, or diffuse strength deficits, evaluate **time course** and **physical pattern**.

Time Course: are symptoms constant, or fluctuating? Did they progress gradually over a longer period? Are they recent, but progressive? Are they completely acute?

Physical Pattern:

- Are deficits symmetric?
- Are all extremities affected?
- Is the trunk affected?
- Are there cranial nerve symptoms?
- Is weakness worse proximally or distally?
- Is there a level at which deficits begin?
- Is strength fatiguing – does it worsen with serial repetitions of muscle testing, or does force production improve with repetition?
- What other abnormalities are there? – Consider tone and reflexes, sensory impairment, pain, mental status, atrophy, fasciculations, cardiopulm endurance, motor planning, movement speed, isolation of intended muscle groups. Integumentary problems can provide valuable clues.

Specificity of weakness pattern ranges greatly – from very low to nearly pathognomonic. But even nonspecific findings can yield good utility when paired with time course.

Ascending (distal > proximal) weakness is often seen acutely in Guillain-Barre, or with chronic, gradual progression in cases of other peripheral nerve pathologies.

Proximal > distal weakness is seen in many problems ranging from myopathies to Myasthenia Gravis to LEMS. Serial MMT is crucial – does strength improve or worsen?

Is strength worse functionally than on MMT? Is subjective severity worse than MMT strength?

Spinal cord pathologies may or may not feature patterns, e.g. Central Cord Syndrome generally looks like bilateral UE weakness that spares the LEs somewhat.

Careful screening for cranial nerve deficits, including for diaphragmatic weakness, can contribute greatly to team-based differential diagnosis.

If the problem is deconditioning, or residual strength impairment as part of an illness episode (either being treated medically or that should primarily be treated with therapy), then strengthening intervention can be effective. Closed-chain, weight-bearing activities are often best. Don't forget about inspiratory muscle training.

For some problems (e.g. unresolved severe electrolyte abnormality, medication-induced myopathy, complete spinal cord injury), working to directly strengthen the muscle may not be productive. There can be benefits to physical intervention – airway clearance, adaptive strategies training, prevention of secondary complication – but it's important (both for prognostication and therapist resource allocation) to understand whether the source of observed weakness can be directly treated through PT intervention.

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Orthodeoxia

Orthodeoxia describes positional hypoxemia in which SpO2 is lower in upright sitting than in supine or recumbent position. It may be accompanied by *platypnea* – shortness of breath in upright position.

Orthodeoxia can result from intracardiac or intrapulmonary shunt, causing deoxygenated blood to be limited in gas exchange, or deoxygenated blood to mix with oxygenated blood. The result of either is circulation of blood carrying less oxygen than normal. Orthodeoxia can also be caused by ventilation-perfusion V/Q) mismatch.

There are many causes of (and contributors to) abnormal shunting, including patent foramen ovale or atrial-septal defect, and *hepatopulmonary syndrome*, in which a cascade of changes arising from portal hypertension (with or without liver cirrhosis) indirectly leads to dilation of pulmonary vasculature. V/Q mismatch is often caused by pulmonary embolism (PE).

If perfusion to the lung bases is good but ventilation is impaired, for example because of pneumonia or atelectasis, this can create a V/Q mismatch.

If gas exchange is normal at the lower lung zones, but perfusion is impaired (e.g., from pulmonary hypertension, or pulmonary embolism which affects the lower zones somewhat more frequently), this also can produce a V/Q mismatch.

Atelectasis can occur as a result of immobility – direct mechanisms include mechanical obstruction (e.g. mucus plug), external pressure (e.g. abdominal ascites, or other space-occupying problem); as well as surface-tension factors (as in ARDS).

If a patient is positioned for a prolonged period in recumbent supine, the lower lobes may be at greater risk for becoming atelectatic due to dependent position. Oxygenation would be expected to improve with mobility, repositioning, incentive spirometry and PEP therapy, and other airway clearance techniques.

If oxygenation frankly worsens in the upright position, continues to be worsened as that position is maintained, and is improved or resolved upon returning to the supine position, factors that could produce orthodeoxia might be considered. Other factors like exaggerated HR response are important to note, particularly in patients at risk for PE.

The finding of worsened oxygenation in the upright position, which is sustained and then improved upon return to supine, is an important observations which PTs may be the first to make, and an important piece of information that can help the care team further identify the pathophysiologic causes of a patient's symptoms.

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Acute Dizziness

Subjective label not meaningfully correlated w/ symptom source – don't let ***room spinning*** vs. ***lightheaded*** vs. ***unsteady***, etc. guide exam.

Timing: Are symptoms ***persistent*** or ***episodic***?

Trigger: Is there a movement/ position ***trigger***, or are symptoms ***spontaneous***? Ask clearly whether symptoms are present at rest.

If patient has spontaneous, persistent dizziness AND nystagmus, this is **Acute Vestibular Syndrome (AVS)** – use **HINTS** cluster to differentiate peripheral from central source:

	Peripheral	*Central*
Head Impulse	**Abnormal**	**Normal**
Nystagmus	**Unidirectional Horizontal**	**Direction-changing / vertical/ torsional**
Test of Skew	**No Skew**	**Vertical Skew**

In a patient with persistent dizziness and nystagmus, unless **every** item of this cluster is consistent with the **Peripheral** pattern noted, a **Central cause** must be suspected.

Acute unilateral hearing loss is benign in 50-60% of cases, but can be caused by AICA stroke. Treat as a **red flag**.

10-25% of AVS is caused by stroke. Most cases are neuritis.

Head Impulse testing can be challenging. Look for patient's eyes to move with head on impulse, followed by corrective saccade back to target. Ease in to the test by first having patient actively turn head while maintaining gaze fixation.

Triggered, Episodic Dizziness: symptoms are not present at rest, but provoked by movement or specific position.

Causes include **BPPV** and **Orthostatic Hypotension**.

Most BPPV involves the posterior semicircular canal. The provocation test for PC BPPV is the **Dix-Hallpike**. The treatment maneuver is the **Epley**.

In the ED, horizontal canal BPPV is almost as common as PC BPPV. The test is the **Supine Roll**. The simplest treatment is the **Gufoni**.

HINTS testing is not used in Triggered, Episodic dizziness syndromes.

Spontaneous, Episodic Dizziness is defined by symptoms that occur without movement or position trigger.

Spontaneous, transient symptoms can occur in setting of arrhythmia and hypoperfusion. **TIA** is a more common cause.

Meniere's syndrome is a rare cause, and dizziness and nystagmus are accompanied by fluctuating hearing loss. **Vestibular migraine** is a more common diagnosis of exclusion.

HINTS is more reliable than MRI within 48 hrs. of onset. Use HINTS only in Acute Vestibular Syndrome.

Absence of focal neuro deficits does not rule out stroke **– only 20% of AVS caused by stroke features a focal deficit**.

If **Head Impulse is equivocal** and cluster is otherwise c/w peripheral pattern – treat as CVA if patient can't stand + walk unsupported.

Vertebral artery dissection preceded by trauma in only half of cases.

Nystagmus of peripheral source beats toward more active side: **Right** horizontal nystagmus caused by a peripheral hypofunction will feature abnormal **Left** Head Impulse.

Peripheral hypofunction / neuritis can still be dangerous (e.g. bacterial otitis media).

Abnormal Head Impulse is occasionally caused by **AICA stroke**; use whole HINTS cluster in AVS.

Gaze fixation may aid exam. Peripheral nystagmus should dampen a little with focus on visual target.

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Visit PTMeasures.com for more clinical resources, or use this QR code:

Acute Dizziness

Subjective label for symptoms **not** meaningfully correlated w/ symptom source – **don't** let *room spinning* vs. *lightheaded* vs. *unsteady*, etc. guide exam.

Instead, consider objective parameters:

1. Timing: Are symptoms ***persistent*** or ***episodic***?

2. Trigger: Is there a movement/ position ***trigger***, or are symptoms ***spontaneous***? Ask clearly whether symptoms are present at rest. *"So are you dizzy right at this moment?"*

If patient has spontaneous, persistent dizziness AND nystagmus, this is **Acute Vestibular Syndrome (AVS).**

In AVS, use **HINTS** cluster to differentiate peripheral from central source:

	Peripheral	***Central***
Head Impulse	**Abnormal**	**Normal**
Nystagmus	**Unidirectional Horizontal**	**Direction-changing / vertical/ torsional**
Test of Skew	**No Skew**	**Vertical Skew**

⚠ In a patient with persistent dizziness and nystagmus, unless **every** item of this cluster is consistent with the **Peripheral** pattern noted, a **Central cause** must be suspected.

Acute unilateral hearing loss is benign in 50-60% of cases, but can be caused by AICA stroke. Treat as a **red flag**.

10-25% of AVS is caused by stroke. Most cases are vestibular neuritis.

Head Impulse testing can be challenging. Look for patient's eyes to move with head on impulse, followed by corrective saccade back to target. Prepare patient to allow quality testing by first having patient actively turn head while maintaining gaze fixation on target (e.g. examiner's nose).

References, resources, exam technique videos →

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Triggered, Episodic Dizziness: symptoms are not present at rest, but provoked by movement or specific position.

Causes include **BPPV** and **Orthostatic Hypotension**.

Most BPPV involves the posterior semicircular canal. The provocation test for PC BPPV is the **Dix-Hallpike**. The treatment maneuver is the **Epley**.

In the ED, horizontal canal BPPV is almost as common as PC BPPV. The test is the **Supine Roll**. The simplest treatment is the **Gufoni**.

HINTS testing is not used in Triggered, Episodic dizziness syndromes – only in AVS.

Spontaneous, Episodic Dizziness is defined by symptoms that occur without movement or position trigger.

Spontaneous, transient symptoms can occur in setting of arrhythmia and hypoperfusion. **TIA** is a more common cause.

Meniere's syndrome is a rare cause, and dizziness and nystagmus are accompanied by fluctuating hearing loss.

Vestibular migraine is a more common diagnosis of exclusion.

HINTS is more reliable than MRI within 48 hrs. of onset. Use HINTS only in Acute Vestibular Syndrome.

Absence of focal neuro deficits does not rule out stroke **– only 20% of AVS caused by stroke features a focal deficit**.

If **Head Impulse is equivocal** and cluster is otherwise c/w peripheral pattern – treat as CVA if patient can't stand + walk unsupported.

Vertebral artery dissection preceded by trauma in only half of cases.

Nystagmus of peripheral source beats toward more active side: **Right** horizontal nystagmus caused by a peripheral hypofunction will feature abnormal **Left** Head Impulse.

Peripheral hypofunction / neuritis can still be dangerous (e.g. bacterial otitis media).

Abnormal Head Impulse is occasionally caused by **AICA stroke**; use whole HINTS cluster in AVS.

Gaze fixation may aid exam. Peripheral nystagmus should dampen a little with focus on visual target.

Evaluation Approach

Hospital physical therapists are responsible for much more than patient mobility and discharge planning. We work sequentially to answer:

1. What is the patient's functional limitation?
2. What are the specific impairments that produce this limitation?
3. Are these impairments explained by the known medical context?

Focal impairments require an explanation. As a hospital-based therapist evaluating patients admitted with weakness, after a fall, or with other non-specific complaints, you will throughout your career be the first to identify the symptoms of an undiagnosed movement disorder, or spinal cord pathology, or stroke, or of many other problems in need of diagnosis and treatment.

Other providers may test strength, coordination, sensation, and reflexes; may screen cognition, look at images, and synthesize medical histories. But in a system focused on passive modes of examination and intervention, we can lead the way toward better diagnostic accuracy through rigorous understanding of how constellations of exam findings fit together to localize a lesion. We, practically alone, examine a patient's gait, which can at times provide a fuller story than all other physical findings together. We can make clear our unique value by elevating practice to the high level we are trained for.

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Principles of localization

Developing a problem list is a crucial first step, but knowledge of clinical syndromes will tell you what other signs to look for once you start to uncover focal abnormalities. Consider basics:

- Mental status
  - Alertness
  - Command following
- Motor planning
- Attention
  - Hemi-body
  - Hemi-environment
  - Neglect
- Speech
  - Dysarthria
  - Expressive impairment
  - Receptive impairment
- Ocular-motor fxn
- Range of Motion
- Force Production
- Movement Speed
- Tone
- Reflexes
- Coordination
- Sensation
- Edema
- Vascular signs
- Tremor
  - Action
  - Intention
  - Postural

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Guillain-Barre Syndrome

GBS is an immune-mediated problem that affects the peripheral nervous system. A viral trigger can provoke GBS (40-80% of cases).

Antibody activity is thought to mediate the disease process, though not all patients with GBS test positive for expected antibodies, and clinical syndrome doesn't necessarily correlate with antibody type. Acute Inflammatory Demyelinating Polyneuropathy (AIDP) is the most common GBS type in the US; reported frequency of other variants ranges significantly.

CSF may show albuminocytologic dissociation; electrodiagnostic testing may be used if available, particularly in cases of atypical presentation. GBS is generally treated with IVIG, sometimes plasmapheresis; supportive care needs may be substantial.

Symmetric, progressive weakness of all extremities, more severe initially among distal muscle groups.

Hyporeflexia: DTRs must be tested, may be absent.

Patients may report paresthesias and pain. Proprioception is often affected: test eyes closed finger-to-nose, and sitting trunk control with eyes closed. Sensory loss is distal to proximal/ ascending "stocking/ glove" pattern – not loss below a spinal cord level.

Tone may be normal early; often some degree of hypotonia is present.

No upper motor neuron signs are typically expected in GBS – this is generally a peripheral nervous system diagnosis, though variants may involve CNS as well.

Autonomic function can be affected. Bowel and bladder function not usually affected early in course.

Contrast with critical illness polyneuropathy/myopathy, in which weakness pattern is generally proximal > distal.

Cranial nerves may be affected; look for external ocular-motor limitations, dysphagia, sometimes facial weakness.

When bulbar weakness is observed, serial monitoring of Forced Vital Capacity is often used to closely track disease progression and to inform supportive care needs.

Miller-Fisher (MF): variant defined by ophthalmoplegia, areflexia, and (sensory) ataxia.

Cognition not directly affected, except in Bickerstaff encephalitis (MF sub-variant).

Other variants may feature predominantly sensory loss or purely motor symptoms, but primarily bulbar weakness without extremity weakness is not typical of GBS.

A focus of PT intervention during acute phase is reducing risk of secondary problems like pneumonia, DVT, worsened dysautonomia resulting from immobility, pressure injuries, loss of Range of Motion, and further deconditioning. All are possible, and made less likely by PT intervention even during symptom progression phase.

17-30% of patients with acute GBS require mechanical ventilation.

Symptom severity generally peaks by 4 weeks from onset. Course typically monophasic. Recovery may not begin immediately upon progression halt.

Age, cranial nerve involvement, and need for mechanical ventilation are factors associated with greater residual impairment.

About half of patients ambulate independently 8 weeks after IVIG and plasma exchange. 20% have some degree of lasting morbidity.

As patients approach chronic phase, LE orthoses (AFO, KAFO) may be indicated.

PT intervention is crucial for management of GBS, but attentive clinicians in hospital environments may also be the first providers to recognize the constellation of ascending weakness, hyporeflexia, paresthesias and/or proprioception impairments.

Focal impairments need an explanation. Awareness of clinical syndromes will help you know what to look for in terms of body structure/function items you may not always test.

PRES

Posterior Reversible Leukoencephalopathy Syndrome, also called Hyperperfusion Encephalopathy, is usually (but not always) caused by hypertension.

The clinical presentation of this neurologic disorder may include abnormal mental status, vision changes, headache, seizure.

There are a number of proposed mechanisms revolving around endothelial dysfunction and disordered cerebral auto-regulation.

Causes or triggers may include kidney disease, autoimmune problems, and immunosuppressants. When systemic blood pressure increases, small vessels vasoconstrict to maintain appropriate circulation. Immunosuppressants can cause tissues changes which alter the effectiveness of that autoregulation.

MRI demonstrates abnormalities usually in the posterior cortex which are usually symmetric.

Physical therapists should understand the likely course of the syndrome – i.e. that symptoms should improve when PRES is treated early. Therapists should also screen for deficits of abnormal mental status and visual changes, including visual field deficits.

Focal Task-Specific Dystonia

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Inattention and Neglect

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Abnormal Mental Status

Hospital PTs will evaluate and treat patients with abnormal mental status frequently. Though not a PT's responsibility to identify the cause of abnormal mental status, or to assess it in detail, it is important to quickly but effectively screen cognition as part of our evaluations.

Many medical problems can produce abnormal mental status, directly or indirectly. Toxic

Level of alertness (talk about RASS); attention, neglect

Aphasia

PRES / hypertensive encephalopathy

Hepatic

Wernicke's

Talk about GCS?

DoC – talk about stimulants (amantadine, ?)

GCS

As always, you can be more effective as a clinician if you look for associated focal impairments and understand the patterns that they form. For example – wobbly, wet and wacky, or neck rigidity and +ve Lhermitte's, or …. Nystagmus, asterixis, jaundice

Motor planning/ apraxia

Skin can be a clue

Seizure

Hypoglycemia

Hallucinations

Delirium specifically – association with falls

CAM-ICU

Sundowning

Neglect, inattention

Always – time course, episodic or constant?

CN abnormality + AMS = brainstem

Reflexes

Vitals – hypotensive hypoactive patient different than HTNsive, hyperactive …

Pumps or patches? Narcotics?

Trauma?

Abnormal Mental Status

Hospital PTs evaluate and treat patients with abnormal mental status often. Though not a PT responsibility to identify the cause of abnormal mental status or assess it in detail, a thorough evaluation must at least screen for it.

Many medical problems can produce acutely abnormal mental status, directly or indirectly:

Nearly any metabolic problem; ranging from blood glucose, to endocrine function, to blood gases, to electrolytes, and others. Toxic encephalopathies can arise from exposure to (or withdrawal from) substances. Infectious causes may or may not directly affect the CNS, (and can affect mental status without doing so). CNS lesions can affect mental status in isolation or with other symptoms.

Labels range widely. Abnormal mental status is a clear descriptor which avoids unintended connotation. Supplement it by providing objective detail about what you observe.

Begin with level of **alertness**. Plan for objective comparison between assessments; avoid non-specific descriptors like *obtunded.*

Patients with **hypoactive** mental status are sometimes considered not ready for mobility intervention, but often that readiness can be changed with different intervention (for rousing). Clear, objective assessment can track smaller progress particularly for patients with depressed level of consciousness.

Is the patient **responding** to your actions? Are their responses **delayed**? What specifically do their responses look like – does the patient **open their eyes**, **track the examiner**, make **eye contact**? Do they reach for (**localize**) a physical stimulus? What **stimulus** is required to elicit a response –e.g. **auditory**, **tactile, visual**, **noxious**, and how specifically is it delivered?

Does the patient follow commands **instructed**? Commands visually **demonstrated**? Do they demonstrate only **automatic movements** – e.g. response to presentation of handshake gesture? Is there **functional object use**?

Standardized tools to be aware of include RASS, CAM-ICU, and Glasgow Coma Scale.

Hospital delirium is strongly associated with **fall risk**, and thus important to address proactively.

Is the patient **oriented**? Instead of A&O x 3, 4, or 7, consider charting *oriented to person, place, time, and circumstances,* for better clarity.

There are many quick screens for judgment, calculation, recall. If a patient manages their own medicines at baseline, can they name 3 of them?

As with any focal impairment, effective examination of a patient with abnormal mental status involves looking for associated problems based on pattern recognition. Always, it's helpful to know how acute symptoms are and whether they are constant or episodic.

There are too many clinical syndromes which might include abnormal mental status to list, and too many potential associated findings to screen for everything. A PT evaluating a patient for non=specific failure to thrive should be ready to test for certain impairments in the right context.

- Does a patient present with abnormal cognition and urinary incontinence? A reason to look specifically for gait apraxia.
- Is there some jaundice in a patient with altered mental status, which prompts you to check for asterixis? Nystagmus?
- Confusion in the setting of hypertension should prompt a look for different impairments than confusion with hypotension. The posterior circulation territories are more susceptible to hypoperfusion (Posterior Reversible Encephalopathy Syndrome) – check visual fields.
- Any cranial nerve abnormality?
- Is there a history of seizure?
- Is it possible that a language impairment has been misunderstood as abnormal mental status?
- Is there neglect of part of the visual environment, or part of the patient's own body?
- Are there motor planning deficits? Can the patient sequence a multi-step task? (Consider Luria sequencing task). Left-Right confusion?
- Is the patient hallucinating? Are there extra-pyramidal symptoms, such as uncontrolled oral-motor movement?

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Vascular Assessment

As a hospital PT you won't be the first provider screening circulatory function, but you can add important pieces to the diagnostic puzzle with a few quick physical tests.

Hypoperfusion can be multifactorial – in a patient with aortic insufficiency and peripheral atherosclerosis, an episode of hypotension can produce symptoms (sometimes transient) easily mistaken for other problems, including dizziness and limb pain.

In a patient at risk for peripheral vascular disease, therapists should check pulses for symmetry (radial and posterior tibial are a start).

Consider checking for asymmetry in BP at either upper extremity. 15 mmHg difference between arms is considered a meaningful predictor.

Because of anatomy, typically venous insufficiency ulcers present on the medial surface of the lower extremity, and wounds caused by arterial insufficiency on the anterolateral shin. Hyperpigmentation and swelling are more commonly associated with venous insufficiency, while pallor and sometimes loss of hair (and "shiny" quality of skin) may present in arterial insufficiency.

Assess limb girth, and take a measurement if extremities aren't clearly symmetric. Note temperature to touch and overall appearance of skin.

Rubor, erythema, and clubbing can be important observations. Is rubor worsened in dependent position, and relieved with elevation? Is erythema symmetric? Is it blanchable? Clubbing is protuberant enlargement of fingertips (or distal toes), and indicates inadequate oxygenation. Check capillary refill by providing pressure at nailbeds and noting time for color to return to normal (< 2-3 sec is normal).

Look for JVD, which can provide a clue about Right heart pressure

Vascular claudication sometimes follows a pattern of pain with exertion, relieved by rest. Sometimes paresthesias are reported.

Ankle-Brachial Index is tested in supine by checking both DP and PT pulses (by doppler) in either foot against brachial BP at either arm, and reported by side.

For example: Left ABI is BP at L DP or PT (whichever is higher) / BP at LUE or RUE (whichever is higher).

Usually ABI > 1.4 or < 0.8 warrants vascular surgery evaluation, though urgency depends on values and other factors.

Subclavian Steal Syndrome is posterior circulation hypoperfusion caused by diversion of blood (via retrograde flow) from the vertebral artery to the ipsilateral arm.

This can produce transient posterior-circulation symptoms (dizziness, ataxia, tinnitus, syncope) and/or UE symptoms (pain, weakness).

In a patient with episodic dizziness, check BP between arms, pulses on either side, and ask screening questions (though they may be low-yield).

Pulses are commonly graded using this scale:

0: absent
1+: faint, but detectable
2+: slightly diminished
3+: normal pulse
4+: bounding

Homan's test is commonly taught to PTs, though the sensitivity is low.

Wells' DVT criteria should be considered:

- Active cancer (+1)
- Bedridden > 3 days or major surgery within 12 wks (+1)
- Calf swelling > 3 cm relative to other leg (+1)
- Collateral superficial veins present (+1)
- Entire LE swollen (+1)
- Localized tenderness along deep venous system (+1)
- Pitting edema (symptomatic leg only) (+1)
- Paralysis/paresis or recent plater immobilization of LE (+1)
- Previous DVT (+1)
- Alternative diagnosis as likely or more likely (-2)

1-2 points considered moderate DVT risk; 3+ points considered high DVT risk

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Consider Thoracic Outlet Syndrome. Use Allen's Test.

Extrapyramidal Symptoms

are drug-induced abnormal motor features caused by blockade of dopamine receptors in the basal ganglia. They're often associated with antipsychotic medications. Sometimes these can be treated medically (by discontinuing the responsible drug and/or additional treatments), sometimes they are irreversible, and often Physical Therapy serial re-evaluation and treatment are warranted in the acute care setting.

Tardive Dyskinesia looks like repetitive, involuntary movement, usually of the face, mouth, and tongue, but can involve any muscle groups. It is more common with longer term use of antipsychotics.

Patients can develop **Parkinsonism** as a side-effect of psychiatric medications, including part or all of the triad of typical symptoms (cogwheel rigidity, pill-rolling rest tremor, bradykinesia), plus or minus other symptoms. ** Dysautonomia less common?

Focal or diffuse **dystonias** can develop – sustained, non-volitional contraction of one or more muscles. This is more common after medication changes.

Whole body movement, like continuous rocking or weight-shifting, is associated with **akathisia**, defined as subjective experience of restlessness, resulting from psychiatric medication use.

Serious, emergent conditions can develop, including **Serotonin Syndrome** and **Neuroleptic Malignant Syndrome**.

PT management techniques (hands-on techniques for tone), stretching, gait and balance, education, monitoring, contribution to differential diagnosis process,

Gait Analysis

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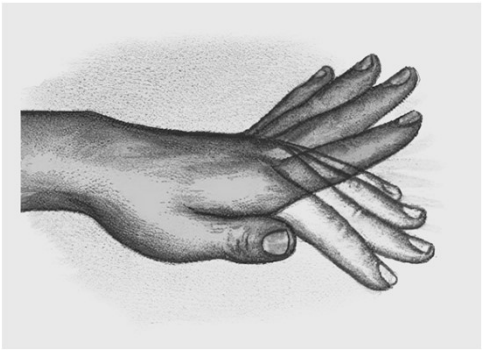
Myoclonus

Myoclonus – which can look like brief shocks of non-voluntary **activation** or **inhibition** of muscle groups – can present challenges to physical therapy intervention. Though PTs are not responsible for diagnosing its cause (which may be an emergency), it is essential to clearly identify and describe this and other pathologic movement accurately because we may be the first healthcare providers to observe it. Helping care teams identify clinical syndromes earlier during illness episodes through judicious physical exam is a core responsibility of effective practice.

In the hospital setting, new myoclonus more commonly results from metabolic or drug-related causes (toxicity or withdrawal), though in a general population, it is more likely a symptom of epileptic activity. It may be seen in post-hypoxic states.

Positive myoclonus looks like non-volitional bursts of muscle activity, while **negative myoclonus** looks like bursts of muscle inhibition. A single muscle group can be affected, or movements can be diffuse. **Asterixis**, the inability to maintain a static position as is sometimes observed in patients with severe hepatic insufficiency, is a form of negative myoclonus.



Bilateral "flapping tremor" (oscillating wrist flexion/ extension) when a patient is asked to maintain a position of shoulder flexion and elbow extension is commonly described as asterixis.

Myoclonus can be a symptom of neurodegenerative disease. It may even be the only symptom: cases have been described in which progressive asymmetric myoclonus, primarily associated with active movement, presents as an isolated symptom, though is not task-specific.

Myoclonic jerks can be rhythmic or non-rhythmic, and importantly **may worsen** with intention and active movement, depending upon the underlying cause or point of origin. The diaphragm may be affected (hiccups) as well as axial and appendicular muscles. Non-volitional jerking movements can be seen in patients at any level of consciousness.

Several parameters can be described, and describing pathologic movements in this way may be helpful even if you aren't sure whether what you're seeing is myoclonus.

- Are abnormal movements localized to one muscle group? Are they unilateral? Which muscle groups are involved, specifically?
- Are the observed movements rhythmic?
- Are non-volitional movements episodic, or continuous? How long do episodes last?
- Are movements elicited or exacerbated by active movement? Is there a difference between movement instructed by examiner, and self-initiated movement of the patient? Are abnormal movements present at rest? Does the patient continue to use the affected extremity during an episode of non-volitional movement?
- Do jerking movements occur with greater frequency or amplitude during weight-bearing? Do other positions or stimuli elicit or exacerbate movements?
- Does the patient's alertness change at all during episodes of non-volitional movement?

Even severe, diffuse myoclonus does not exclude patients from PT intervention in the hospital, and benefits of mobilization in critically ill patients are well-established.

To maximize safety, remove any possibility for a fall in patients with myoclonus: stand-step transfer with no space between bed and chair, for example, and use a chair follow and additional support for ambulation.

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