NEUROMUSCULAR 1

FINAL EXAM STUDY GUIDE

Spinal Cord Injury

↳ **American Spinal Injury Association (ASIA) Classifications:**

- **Standardized outcome measure:** ICF + neurological level of injury
  - ↳ **neurological level of injury:** most caudal (distal) level of the spinal cord w/ normal motor & sensory function on both left & right side of the body
    - **motor level:** most caudal segment with normal motor function bilaterally
      - ↳ tested through key myotomes
      - ↳ 3/5 = normal
    - **sensory level:** most caudal segment with normal sensory function bilaterally
      - ↳ tested through light touch & pinprick via key dermatomes
      - ↳ 2/2 = normal

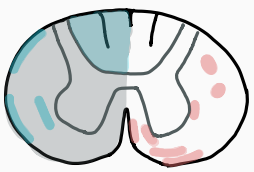
no sacral sparing →

- **complete injury:** NO sensory or motor function in the lowest sacral segments S4-5 ASIA = A
  - ↳ **zone of partial preservation:** areas of intact motor/sensory function below the neurological level in the absence of segments S4-5

sacral sparing = good prognosis →

- **incomplete injury:** has motor and/or sensory function below the neurological level, including sensory and/or motor function at S4-5
  - ↳ sporadic areas of function

↳ **Brown Seaward Syndrome:** hemisection injury



- only half of the spinal cord is affected
- ipsilateral loss of proprioception, vibration, & motor function at & below lesion level → damage to dorsal column & lateral corticospinal tract
- contralateral loss of pain & temperature → damage to spinothalamic tracts
  - ↳ loss begins several dermatome segments below lesion level
- usually caused by penetration wounds

↳ **Autonomic dysreflexia:** life threatening dysfunction of the nervous system triggered by noxious stimuli below the level of the lesion

- afferent input from these stimuli reach the lower spinal cord and initiate a mass reflex response
  - ↳ results in elevation of blood pressure
- observed in injuries above T6
- most common in complete injuries

* common cause is bladder & bowel distention/irritation

- **presentation of symptoms:**
  - hypertension → can cause seizures, cardiac arrest, subarachnoid hemorrhage, stroke, & even death
  - bradycardia
  - headache
  - profuse sweating
  - increased spasticity
  - restlessness
  - vasoconstriction below lesion level
  - vasodilation above lesion level
  - constricted pupils
  - nasal congestion
  - piloerection (goosebumps)
  - blurred vision

↳ **cardiovascular impairment:**

- orthostatic hypotension ← usually only significant in people w/ SCI above T6

↳ to minimize effects:
- the cardiovascular system should be allowed to adapt gradually by a slow progression to the vertical position
- start by elevating the head of the bed
- progress to a reclining wheelchair with elevating leg rests & a tilt table
- monitor vital signs carefully
- patient should always be moved very slowly
- use compressive stockings & an abdominal binder
- can use meds

★ sacral sparing: maintained sensation around S4/S5 anal region → indicates incomplete SCI

↳ common complications:
- Neurological complications:
  - ↳ Spinal shock: an immediate period of areflexia post spinal cord trauma
    - absence of all reflex activity, impairment of autonomic regulation resulting in hypotension and loss of control of sweating & piloerection
    - loss of bulbocavernosus reflex, cremasteric reflex, Babinski response, & delayed plantar response
    - 24 hours & evolves over time
    - resolution within 1-3 days
    - primary neurological complication
  - ↳ loss/ dysfunction of motor, sensory, and autonomic systems
- Primary Impairments:
  - ↳ motor & sensory impairments are usually primary impairments
- possible impairments:
  - ↳ autonomic dysreflexia
  - ↳ spastic hypertonia
  - ↳ orthostatic hypotension
  - ↳ impaired temperature control
  - ↳ pulmonary issues
  - very common → ↳ bowel / bladder dysfunction: UTI, spastic / flaccid bladder, spastic / flaccid bowel
  - ↳ sexual dysfunction

lesion above sacral segments; lesion below sacral segments; lesion above S2; lesion of S2-4 or cauda equina

- secondary medical complications:
  - ↳ pressure sores
  - ↳ pneumonia
  - ↳ DVT
  - ↳ pain
  - ↳ contractures
  - ↳ heterotopic ossification
  - ↳ osteoporosis & fracture

↳ best predictor of motor recovery → preserved motor function
- ASIA level A → motor recovery 1 level below the initial neurological level
- ASIA levels B, C, D → ↑ prognosis
- pinprick sensation in B LE → ↑ prognosis in one year if present 4 most post injury

↳ Outcome measures:
- SCIM: Spinal Cord Injury Independence Measure ← ICF: activity
  - ↳ specifically for SCI patients
  - ↳ 19 items w/ 3 subcategories:
    - self-care
    - respiration
    - sphincter management
    - mobility
  - ↳ score 0-100 → higher score = more independent

↳ valid & reliable
↳ may be more responsive than the FIM

Guillain-Barre Syndrome

↳ **Outcome Measures:**
- **Fatigue:**
  - ↳ **Fatigue Severity Scale (FSS):** self-administered 9-item rating scale, emphasizes functional impact of fatigue
  - ↳ **Fatigue Impact Scale (FIS):** assesses quality of life problems related to fatigue
  - ↳ **Visual Analog for Fatigue Scale (VAS-F):** 10 item questionnaire asking about the subjective experience of fatigue
- **Function Specific:**
  - ↳ **Barmel Index:** measures level of assistance required by pt on 10 items of mobility & self-care ADLs.
  - ↳ **Modified Hughes Scale of GBS disability:** assess functional status of GBS patients
- **Participation Related:**
  - ↳ **Short Form 36:** 36 items that help to determine health status & physical functioning
  - ↳ **Nottingham Health Profile:** questionnaire to assess social & personal effects of illness

Upper Extremity Function

↳ **Visual feedback for reaching:**
- **visual feedback:** primary function that relates to attainment of final accuracy in reaching
- disruption of CNS & PNS disrupts timing & accuracy of task performance
- performance is improved when using vision

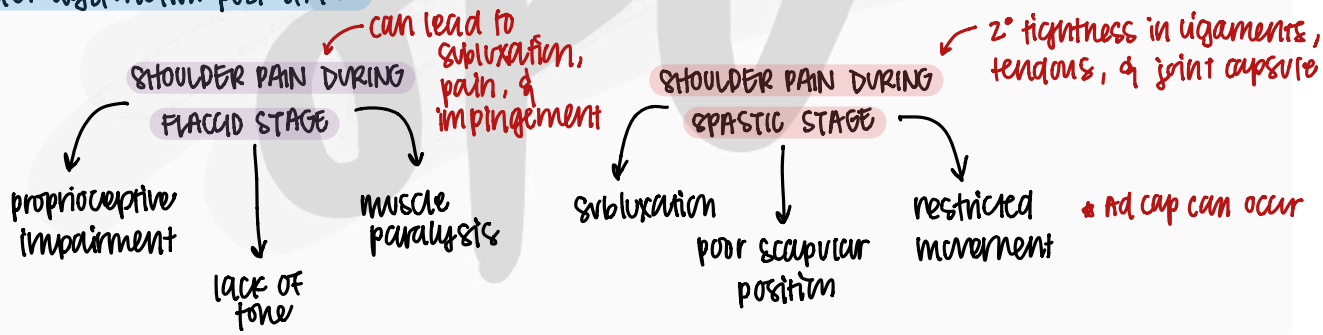
* Target location problems: - head, eye, trunk control altered
- gaze stabilization: oculomotor pathology, vestibular damage, cerebellar damage

* primary function of visual feedback in reaching is related to the attainment of final accuracy
↳ constancy of thumb position w/ relation to the wrist may be a strategy of providing clear visual feedback info regarding the end point of the limb

↳ **extrinsic causes of shoulder subluxation:**
- positioning
- handling
- assistive devices

↳ **goals & interventions for subluxation:**
- maintain alignment
- patient & caregiver education
- early intervention & stopping 2° & indirect impairments → pt education is key

↳ **shoulder dysfunction post stroke:**



Psychosocial Disorders

↳ **Psychosocial factors:**
- mental health predictor of physical health
  - ↳ patients w/ physical disabilities may not respond well to PT because of psychosocial issues
- high engagement & participation positively influences recovery
- mind-body connection
- increased presence of psychosocial illness after physical illness/disability & vice versa

↳ the longer someone suffers from mental illness, there is a greater risk of developing a physical illness

↳ **psychosocial adaptation:**
- how patients work towards attaining an optimal state of function within their environment
- ongoing, dynamic
- adjustment is the final phase
- **congenital vs. adventitious illness:**
  - ↳ **congenital:** born w/ illness
    - only know life w/ impairment
    - development mirrors kids w/o disabilities
  - ↳ **adventitious:** accidental
    - experience an acute sence of loss & grief

* they experience very different psychological issues

- **phase model:**

time ↓
SHOCK
ANXIETY
DENIAL
DEPRESSION
INTERNALIZED ANGER
EXTERNALIZED HOSTILITY
ACKNOWLEDGEMENT
ADJUSTMENT

↳ **grief, mourning, & sorrow:**
- **grief:** state of distress that results from a significant loss
  - ↳ preoccupation w/ loss & feelings of worthlessness & helplessness
  - ↳ **symptoms:**
    - tightness in throat
    - muscular weakness
    - emptiness in abdomen
    - SOB
    - choking
    - painful anxiety
    - forgetfulness
    - poor concentration
    - insomnia
    - disorganized cognitive function
    - loss of appetite
    - impulsive
  - ↳ period is unpredictable & variable → too long = can compromise immune system
  - ↳ grief is a natural process & is necessary for the development of a new self-concept

Cognitive & perceptual Dysfunction

↳ **cognition:** act or process of knowing, including awareness, reasoning, judgment, intuition, & memory

↳ **perception:** integration of sensory impressions into info that is psychologically meaningful → ability to select stimuli & interpret them

↳ clinical indicators of deficits

DIFFICULTY OR INABILITY TO:	PATIENTS MAY:
perform simple tasks independently / safely	→ hesitate many times
initiating / completing a task	→ appear distracted and frustrated
switching from 1 task to another	→ exhibit poor planning
visually locate or identify objects that are necessary for task completion	→ be inattentive to 1 side of the body

* multi-infarct dementia: from multiple small infarcts of the brain, abrupt onset, >60 y/o, stepwise & paroxysmal deterioration of intellectual function
↳ may coexist w/ Alzheimer's Disease

- following 1 step commands → complete tasks impulsively
- complete tasks in a timely manner → deny presence or extent of disability

* dementia is associated w/ ↑ mortality rates

↳ **cognitive & perceptual deficits:**

- **executive function:** the capabilities that enable a person to engage in independent, purposeful, self-serving behavior
  - ↳ **volition:** capacity to determine what one needs & wants to do
    - awareness of self, environment, & society
  - ↳ **planning:** organization of steps to accomplish task, weighing alternatives, decision making
  - ↳ **purposive action:** productivity & self regulation to achieve goal
    - ability to initiate & maintain action & switch/stop action
  - ↳ **effective performance:** quality control & self correlation of behaviors

patients can have deficits in any/all of these

- **prosopagnosia:** inability to recognize the faces of familiar people
- **stereognosis:** ability to recognize forms by handling/touching them
  - ↳ disorder = astereognosis
- **ideational apraxia:** lost of the idea of what to do
  - ↳ unable to conceptualize a task & cannot perform a purposeful motor act on command / automatically
  - ↳ unable to verbally describe process of performing the task
- **ideomotor apraxia:** understands what to do but can't perform task when commanded
  - ↳ but habitual tasks can be done automatically
- **unilateral neglect:** inability to register & integrate stimuli & perceptions from 1 side of the body (body neglect) or environment (spatial neglect)
  - ↳ damage to either hemisphere but Ⓡ more common
  - ↳ can't register stimuli on the contralesional side
  - ↳ sensory loss is common (↓ recovery rate tho)
- **figure-ground discrimination:** inability to visually distinguish an object from the background
  - ↳ difficulty locating objects
  - ↳ can't ignore irrelevant visual stimuli
  - ↳ have trouble selecting the appropriate cues
  - ↳ ex) can't locate items in the drawer
- **anosognosia:** severe perceptual impairment
  - ↳ severe form of neglect
  - ↳ denies body part is their own OR denies the paresis/paralysis → "my arm has a mind of its own"
  - ↳ spontaneously resolves within the 1st 3 months after CVA → "I left it at home"
  - ↳ more common in patients w/ Ⓡ hemispheric lesions → "I left it in the closet"
  - ↳ safety is a concern
- **somatoagnosia:** impairment in body scheme
  - ↳ difficulty following instructions that require distinguishing body parts
  - ↳ may be unable to initiate movements
  - ↳ can also make exercising more difficult
  - ↳ providing sensory cues & input to the affected limb can really help these pts
- **Right-left discrimination:** inability to identify R & L sides of one's body or of examiner
  - ↳ unable to follow commands that include terms left & right
  - ↳ unable to initiate commands

old Material

↳ **ICF framework:** International Classification of Functioning, Disability, & Health

- Body functions & structure, activity, & participation

↳ **Motor Function & Spasticity:**
- **Motor function:** motor control & learning
- **spasticity:** increase in resistance to passive elongation that is elicited during a fast, passive stretch
  ↳ **Modified Ashworth Scale:**
  0 → no increase in tone
  1 → slight increase in tone, end of range (may catch & release)
  1+ → slight increase in tone through less than ½ range
  2 → marked increase through most of the range (still moves easily)
  3 → passive movement difficult
  4 → rigid (no movement)
  ↳ can also use Tardieu scale (V1-V3, 0-5)

* Direct impairment: b/c of condition
Indirect impairment: complications from other
Composite impairment: combined effects of both

↳ **IV STEP goals:** 4P's: participation, prediction, plasticity, prevention
- Explore PT's role in preventing disabling conditions
- Evaluate new ways to classify movement disorders
- Summarize critical periods of emergence of neuroplasticity & strategies for maximizing experience
- Analyze & apply emerging measures & interventions to optimize pt participation

↳ **Expressive aphasia:** damage to Broca's area
- has intact auditory comprehension but hard time expressing what it is that they want to say
- can become frustrated

↳ **Receptive aphasia:** damage to Wernicke's area
- impaired auditory comprehension so they can't understand what you're saying
- don't get frustrated

↳ **vascular stroke syndromes:**
- **Anterior cerebral A:** affects frontal & parietal lobes, basal ganglia
  ↳ contralateral weakness, sensory loss, cognitive confusion
  ↳ LE involvement
- **Middle Cerebral A:** affects frontal, temporal, parietal, occipital lobes, internal capsule & structures
  ↳ most common stroke
  ↳ contralateral weakness, sensory loss, vision problems, aphasia
  ↳ UE involvement
- **posterior cerebral A:** affects occipital lobe & part of thalamus
  ↳ less common stroke
  ↳ contralateral weakness, sensory loss, vision problems
- **vertebrobasilar A:** affects brain stem & cerebellum
  ↳ causes locked in syndrome

↳ **Brunnstrom Stages:**
STAGE 1 ——— Flaccidity
STAGE 2 ——— spasticity **begins**, no voluntary movement
STAGE 3 ——— spasticity **worsens**, voluntary movement occurs in synergy
STAGE 4 ——— spasticity **declines**, some voluntary movement out of synergy
STAGE 5 ——— spasticity **continues to decline**, relative independence from synergy
STAGE 6 ——— spasticity **disappears**, isolated joint movement, ~normal coordination/speed

↳ **Gait:**
- **Abnormal tone (spasticity):**
  ↳ PF → prevents heel strike (causes flat foot), toe drag during swing phase
  → forward trunk lean & shortened step width
  → circumduction, hip hiking
  ↳ Quadriceps → knee hyperextension during loading response
  → trunk leans forward & moves the COM
  ↳ Hamstrings → knee flexion at initial contact, knee buckling at stance phase

→ shortened step length = ↓ stance time & quad demand
→ crouch gait

↳ Adductors → causes contralateral pelvis to drop & medial displacement of leg at stance phase
→ scissoring gait

↳ Hip flexors → reduction of hip extension during mid & terminal stance phase
(stiffness) → knees flex to bring pelvis into alignment

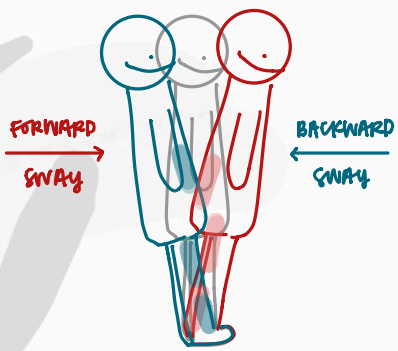
- Weakness / paresis:
  - ↳ PF → excessive knee flexion during stance phase
    → decreased heel rise during terminal stance
  - ↳ DF → flat foot / forefoot contact at initial contact
    → ↓ foot clearance during swing phase
  - ↳ Quadriceps → poor knee control during loading response
    → destabilizes knee during midstance
  - ↳ Hip flexors → problems w/ limb advancement during swing phase
  - ↳ Hip extensors → causes forward trunk lean during stance phase
  - ↳ Hip abductors → Trendelenberg gait (during stance phase)

* normal gait only requires a 2+ strength grade for hip flexors

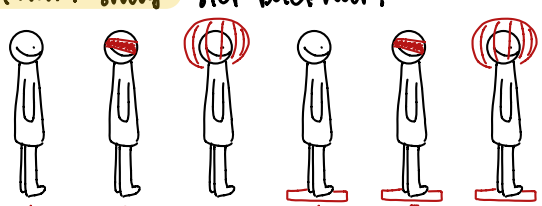
- Synergies:
  - ↳ UE flexion:
    - scapular retraction / elevation
    - shoulder abd, ER
    - elbow flex
    - wrist flex
    - fingers flex
  - ↳ LE flexion:
    - hip ext, add, IR
    - knee ext
    - ankle PF, inv
    - toe PF

↳ Compensatory Motor Strategies:

- Ankle strategy:
  - ↳ small perturbations w/ firm surface
  - ↳ mms fire distal → proximal
  - ↳ Forward sway: gastroc, hamstrings, paraspinals
  - ↳ Backward sway: tib ant, quads, abs
- Hip strategy:
  - ↳ big & fast perturbations w/ soft, compliant, & narrow BOS
  - ↳ mms fire proximal → distal
  - ↳ Forward sway: abs, quads * named for trunk position
  - ↳ Backward sway: paraspinals, hamstrings
- Stepping strategy:
  - ↳ very large & fast perturbations or ankle / hip impairments
  - ↳ Forward sway: step forward
  - ↳ Backward sway: step backward



↳ CTSIB:



	1	2	3	4	5	6
Accurate	vest, vision, somato	vest, somato	vest, somato	vest, vision	vest	vest
Inaccurate	—	—	vision	somato	somato	vision, somato

Unit 3 Assignment

	Stroke	Parkinson's Disease	Traumatic Brain Injury	mTBI/Concussion/PCS
Epidemiology	#5 leading cause of death in US, F>M, majority >65 y/o, African-Americans most impacted, increased risk with previous hx of stroke	1 mil people living with PD, risk increases with age, average age of onset = 50-60 y/o, M>F	Vary by age, MVA, falls, highest risk = 0-4 and 15 -19 y/o, M>F	Any age, falls, MVA, violence, playground injuries, sports, F>M, ~150k concussions every year PCS: >10 days of symptoms, >3 wks of symptoms for HS athletes
Etiology	Ischemic: lack of blood flow due to narrow opening for oxygen to go through; cerebral thrombus, embolism, atherosclerosis Hemorrhagic: uncontrollable bleeding due to compression of brain; intracerebral or subarachnoid hemorrhage	Idiopathic, 10% genetic, 3 different onsets possible Parkinson-like diseases: Secondary parkinsonism, Parkinsonism-plus syndromes	Open head injury: skull is fractured, usually associated with intracranial hemorrhage Closed head injury: injury with skull intact	Direct blow to the head, neck, face, or body causing brain injury
Pathophysio-logy	Ischemic: complete occlusion of blood flow leads to a core area of neuronal cell death - Release of glutamate - Altered Ca2+ ion channels causes influx of Ca2+ into neuron - Activation of destructive Ca sensitive enzymes - Further neuronal cell death (prenumbra)	Degeneration of dopamine producing neurons in the basal ganglia: neuronal death of substantia nigra causing less DA on the striatum Lewy body inclusion bodies develop with disease progression: protein accumulation	Primary brain injury: damage occurs at the moment of impact; focal brain injury, blast injury, diffuse axonal injury Secondary brain damage: occurs within min-hours after injury; intracranial hematomas, herniation, hypoxic-ischemic injury, epilepsy/seizures, intracranial infections	Blunt force: ion channel dysfunction, metabolic energy crisis, physiologic axonal stretching Blast-related: shock waves disrupts brain tissue, penetrating injury and blunt trauma
Expected & Unique Neurological Impairments	Muscle weakness, sensory loss, cognitive confusion, vision problems, aphasia, paralysis, impulsive behavior, poor judgement, flat affect, depression FAST (face, arms, speech, time): Sudden numbness/weakness of face, arm, or leg, sudden confusion, trouble speaking or understanding, trouble seeing, trouble walking, dizziness, LOB, severe headache	TRAP: tremor, rigidity, bradykinesia/akinesia/hypokinesia, postural instability Start hesitation, freezing gait, depression, anxiety, psychosis, dementia, sleep disturbances, lack of smell, excessive sweating, saliva production, oily skin, dysarthria	Paresis, abnormal tone, impaired motor function, impaired postural control, impaired cognition and executive functions, aggression/agitation, disinhibition, communication and swallowing impairments, coma/vegetative state/MCS	Headache, fogginess, emotional liability, LOC, amnesia, irritability, slowed reaction times, sleep impairments, decreased attention, disorientation, memory impairment, dizziness, impaired speech Blast-related: dizziness, vertigo, hearing loss, cog. Deficits, HA, disequilibrium PCS: depression/anxiety, panic, sleep alterations
Outcome Measures	Stroke specific: Fugl-Meyer, STREAM, SIS Postural control/balance: Berg, PASS, Tinetti, TUG, CTSIB	Body functions: MDS-UPDRS, MOCA, H &Y, PD fatigue scale, FOG questionnaire Activity: 10 min walk, 6 min, FGA, Mini BESTest, 5xSTS, 9hole peg, MDS-UPDRS, TUG, ABC scale, pull test Participation: PDQ-8, PDQ-39	GCS, CRS-R, LOCF. Berg, HiMAT, 10m walk, 6min walk	GCS, SAC, SCAT3, MACE

	SCI	GB
Epidemiology	• 17,000 new cases/year in US • 243,000-347,000 currently living in US • Avg age: 42 y/o • Majority non-Hispanic white males	• 1-4 cases per 100,000 • 3000-6000/year in the US • Males > females
Etiology	Types of Injury • Traumatic: MVA (most common), falls, violence, sports • Non-Traumatic: vascular dysfunction, vertebral subluxation due to RA/DJD, spinal neoplasm, syringomyelia, abscess, infection (syphilis, transverse myelitis), MS or ALS • Traumatic more common of the two Common MOI C-spine: concomitant rotation, lateral flexion, shearing forces • Whiplash, falling backwards and landing with chin on tub • Forces of: flexion, axial loading, distraction, extension, T-spine: GSW, MVA, falls flexion, axial loading, flex+rotate • Less common than cervical, more likely to be complete • Most common site: T12-L1 junction (transition from UMN to LMN) L-spine: falls, MVA, GSW, direct load onto spine • Forces of: flexion, axial loading, flexion combined with distraction or rotation	• Acute inflammatory demyelinating immune-mediated polyneuropathy • Nerve roots and peripheral nerves affected • Leading to flaccid paralysis, sensory impairment and autonomic nervous system impairment • Idiopathic 27% • Infection 66% → campylobacter jejuni, mycoplasma pneumoniae, cytomegalovirus, Epstein Barr virus, flu Secondary Risks/Complications • Infections • Organ system failure • Cardiac specific symptoms (dysrhythmias, low CO, marked BP fluctuations) • DVT and pulmonary embolus risk is higher • Death
Pathophysiology	• Damage due to impingement and/or compression of cord: bony/soft tissue, penetrating/non-penetrating • Blunt trauma: primary neuronal damage to cell bodies/axons • Secondary injury causes the most damage: ischemia, demyelination, edema, necrosis Initial trauma → apoptotic cell death, interruption of blood flow, inflammation, increased amino acids → ischemia, disruption of homeostasis → calcium accumulation and demyelination → cell death	• Spinal roots and peripheral nerves attached by microphages and T lymphocytes in response to virus • Damage occurs directly to the myelin sheath along axon • Severity depends on axonal damage • Mild: axon intact, rapid re-myelination • Severe: axonal damage or loss, axonal regeneration required • Myelin sheath damage causes decreased action potentials (slowed conduction, desynchrony, disturbed conduction)
Expected and Unique Neurological Impairments	Complete Impairment • NO motor/sensory function is preserved in S4-5 Incomplete Impairment • Sensory but not motor function is preserved below the neurological level and includes S4-5 • Motor function is preserved below the neurological level, and > 1/2 of key muscles below level have a muscle grade <3 • Motor function is preserved below the neurological level, and > 1/2 of key muscles below level have a muscle grade >3 Presentation • Tetraplegia vs. Paraplegia (incomplete tetraplegia most common) Clinical Syndromes • Brown Sequard Syndrome: hemisection injury, only half of SC affected, ipsilateral loss of proprioception, vibration, and motor function at and below level of lesion, contralateral loss of pain and temp • Anterior Cord Syndrome: flexion injury of C-spine, bilateral loss of motor function, pain, and temp sensitivity at and below injury level, intact light touch and proprioception • Central Cord Syndrome: most common due to hyperextension injury, paralysis and sensory loss in UE, varying involvement in trunk and LE's • Cauda Equina Injury: injury to the lumbosacral nerve roots of the cauda equina, LMN signs, flaccid paralysis of LE's, areflexic bowl and bladder (difficult for self-care) Primary/Secondary Impairments • Motor output, sensory input, autonomic dysreflexia, spastic hypertonia, cardiovascular, temp, pulmonary, bowel and bladder, sexual dysfunction	Medical Diagnostic Criteria • Motor weakness (rapid progression, symmetrical, distal to proximal, areflexive distal tendons (LMN)) • Mild sensory symptoms, parasthesias/hypesthesias • Autonomic dysfunction: tachycardia, arrhythmia • Absence of fever at onset, history of recent flu-like illness • Lab results not conclusive: elevated protein in CSF • Nerve conduction study: abnormal velocity • Recovery occurs 2-4wks after plateau of disease process Common Clinical Presentation • Rapid evolution of bilateral, symmetrical flaccid paralysis • Fatigue: severity is correlated with older age • DTR diminished or absent • Sensory symptoms: tingling, numbness, decreased vibratory senses, stocking glove distribution • Pain: muscle aching, large muscle groups, progressive worsening as disease progresses Less Common Clinical Presentation • Ventilator dependent (concomitant dysautonomia) • CN involvement • ANS symptoms (pooling of blood, urinary retention, ileus, poor venous return Prognosis • Max paralysis within 1-2 days of onset and greatest severity reached after 3 weeks • Once plateaued, motor recovery occurs within 2-4 wks • Ambulatory within 6 months, persistent residual impairment , mild neurological deficits
Outcome Measures	• ASIA's International Standards for Neurological Classification of Spinal Cord (ISNCSCI)	• System specific → FSS, FIS, VAS-F • Function Specific → Barthel Index, Modified Hughes Scale of GBS disability, FIM • Participation → Short form 36, Nottingham Health Profile
Practice Settings	• SCI patients can be seen in all PT settings due to the variety of presentations along with the level of injury and age.	• GB patients can be seen in all PT settings depending on the progression of the diagnosis and ambulatory ability.

	MS	ALS
Epidemiology	• 400,000 people • Onset typically between 20-40 years of age (30 y/o is average) • Women > men, Caucasians > other races • Increased risk with those living above 40 latitude (up north) • Risks: genetics, race, geography	• 30,000 people • High incidence (Guam and Japan → Western Pacific form) • Onset usually mid-to-late 50s, Men > women • Sporadic ALS > familial ALS • 70-80% limb onset, 20-30% bulbar onset
Etiology	• Unknown cause • Chronic – stays with them for the rest of their life • Demyelinating disease of the CNS and can have lesions anywhere in the CNS, which makes presentation extremely variable • Autoimmune disorder • Viral infection triggers the immune response but virus is unknown • Genetic susceptibility to immune system dysfunction • Increased risk with vitamin D deficiency and smoking, which is way people who live far from the equator and are less exposed to the sun have a higher risk of having MS	Known Risk Factors • Disease-causing mutations (SOD1, alsin), clusters (Western Pacific ALS/PDC), male > female, mid to late 50's, familial Possible Risk Factors • Neurotoxicant exposures: lead, mercury, pesticides • Lifestyle factors: smoking, alcohol • Certain occupations: electrical/industrial workers, farmers • Trauma: skeletal trauma, fractures, electric shock w LOC • Diet: high fat or glutamate, low fiber and antioxidants • Vigorous physical activity: heavy labor, athletics
Pathophysiology	• Virus appears and sets off an immune response, which then attacks the myelin and creates holes. Nerve conduction then slows down or gets completely blocked. Immune response also attacks oligodendrocytes, but some manage to survive. Those that survived can remyelinate the axons and decrease patient impairments. • Inflammation also occurs because of the presence of damage, which causes further swelling, impairs nerve conduction, and exacerbates the impairments • BOTH cause slower nerve conduction rates and impairments • Lesions can be anywhere in the CNS, but typically in the white matter (myelinated axons, tracts, etc.) • Optic pathway, corticospinal tracts, dorsal column of the SC and cerebellar peduncles are particularly susceptible	• Progressive degeneration and loss of motor neuron in the sc , brainstem, motor cortex Affects • UMN in cortex and corticospinal tracts • Brainstem nuclei for CN V, VII, IX, X, XII • Anterior horn cells in spinal cord • Sensory system and spinocerebellar tract During Stages of Disease • Sprouting: early on, healthy intact axons sprout and innervate synaptic sites that were previously activated by the damaged neurons • Contiguous progression: locally spread before moving to another area • Rostral/caudal spread: late stage
Expected and Unique Neurological Impairments	Common impairments • Visual impairments: blurred vision, altered acuity, optic neuritis, diplopia • Damage to corticospinal tracts: paresis/plegia, spasticity, hyperreflexia (+ Babinski, + clonus) • Damage to dorsal columns: impaired proprioception, paresthesia, dysesthesias • Damage to cerebellar peduncles: balance, coordination, tremor, ataxia, hypotonia, vestibular impairments Symptoms • Sensory, pain, visual, motor, fatigue, coordination, balance, gait, mobility, speech and swallowing, depression, emotional, cognitive, bladder, bowel, sexual • Can suffer from bulbar paralysis • Fatigue is a primary problem in patients and can be disabling • Patients with affective symptoms (emotional) are at higher risk of intellectual and cognitive impairments Subtypes • Relapsing-Remitting MS (RRMS): most common, acute attacks followed by remission until loss of oligodendrocytes causes inability to return to baseline, has best prognosis • Secondary Progressive MS (SPMS): steady and irreversible decline with/without acute attacks • Primary Progressive MS (PPMS): steady functional decline from onset without acute attacks and no periods of remission, has plateau periods but symptoms get worse over time • Progressive Relapsing MS (PRMS): least common, most severe, steady deterioration from onset with occasional acute attacks	Clinical Manifestations • Highly variable • Depends on extent of motor neuron loss, degree and combination of UMN and LMN loss, pattern of onset and progression, body regions affected stage of disease • Symptoms are focal and asymmetrical at onset LMN Pathology Impairments • Muscle weakness: LMN weakness worse than UMN weakness (cardinal sign!) • Hyporeflexia and hypotonicity • Fatigue: as motor neurons die, remaining ones become burnt out and work harder for the same result • Atrophy • Muscle cramps: unknown cause • Fasciculations: random twitches of muscle fibers UMN Pathology • Spasticity and Hyperreflexia • Pathological reflexes and Clonus • Weakness Bulbar Pathology Impairments • Bulbar muscle weakness • Spastic/flaccid bulbar palsy • Dysarthria and dysphagia • Sialorrhea • Pseudobulbar affect Other Impairments • Respiratory: may be put on the ventilator • Cognitive: mild deficits to dementia
Outcome Measures	• Expanded Disability Status Scale (EDSS) • MS Functional Composite (MSFC) • Multiple Sclerosis Quality of Life-54 (MSQOL-54) • Multiple Sclerosis Impact Scale (MSIS-29)	• ALS Functional Rating Scale (ALSFRS) • ALS Assessment Questionnaire (ALSAQ-40)
Practice Settings	• MS patients can be seen in all PT settings due to the variety of symptoms and presentations along with the age of onset.	• ALS patients are also seen in all PT settings – a multidisciplinary approach is most advantageous