Spinal Cord Injury

- American Spinal Injury Association (ASIA) Classifications:
  - Standardized outcome measure: ICE + 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
      - 3/4 = normal
    - Sensory level: most caudal segment with normal sensory function bilaterally
      - Tested through light touch & pinprick via key dermatomes
      - 2/4 = normal
  - 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
  - Incomplete injury: Has motor and/or sensory function below the neurological level, including sensory and/or motor function at S4-5
  - Spared areas of function
    - Brown Seaward Syndrome: Hemisection injury
      - Only half of the spinal cord is affected
      - Ipsilateral loss of proprioception, vibration, & motor function at 3 levels above lesion level → damage to dorsal column (internal arcuate fasciculus) & 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
    - Presentation of symptoms:
      - Hypertension
      - Headache
      - Profuse sweating
      - Increased spasticity
      - Restlessness
      - Vasoconstriction below lesion level
      - Vasodilation above lesion level
      - Constricted pupils
      - Nasal congestion
      - Piloerection (goosebumps)
      - Blurred vision
    - Common cause is bladder & bowel distention/irritation
  - Cardiovascular impairment:
    - Orthostatic hypotension

No sacral sparing

Sacral sparing: Good prognosis

Heart attack

Hypertension

Cardiac arrest

Subarachnoid hemorrhage

Stroke

Death

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 medic

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 bulboanureous reflex, cremasteric reflex, Babinski response, or delayed plantar response
    - 24 hours & evolves over time
    - resolution within 1-3 days
  - primary neurological complication
  - loss of dysfunction of motor, sensory, and autonomic systems
  - primary impairments:
    - motor & sensory impairments are usually primary impairments
  - possible impairments:
    - autonomic dysreflexia
    - spastic hypertension
    - oromandibular hypotension
    - impaired temperature control
    - pulmonary issues
    - urinary issues
  - secondary medical complications:
    - bowel/bladder dysfunction: UTI, spastic/paracal bladder, spastic/paracal bowel
    - sexual dysfunction

best predictor of motor recovery
- ASIA level A → motor recovery 1 level below the initial neurological level
- ASIA levels B,C,D → ? prognosis
- pinprick sensation in B/E → ? prognosis in one year if present & most post injury

outcome measures:
- SCIM: Spinal cord injury independence measure
  - specifically for SCI patients
  - 19 items w/ 3 subcategories:
    - self-care
    - respiration
    - sphincter management
    - mobility
  - score 0-100 → higher score = more independent

sacral sparing: maintained sensation around S4/S5 anal region → indicates incomplete SCI
Guillain-Barré 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 (VAFS): 10-item questionnaire asking about the subjective experience of fatigue.
- Function specific:
  -钡elin Index: Measures level of assistance required by pt on 10 items of mobility & self-care ADLs.
  - Modified Hughes Scale of GB Disability: Assesses functional status of GB & patients.
- Participation Related:
  - Short Form 36: 34 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 visual feedback.
- Extrinsic causes of shoulder subluxation:
  - Positioning.
  - Handling.
  - Assistive devices.
- Goals & Interventions for Subluxation:
  - Maintain alignment.
  - Patient & caregiver education.
  - Early intervention & stopping.
- Shoulder dysfunction post stroke:
  - Shoulder pain during flaccid stage can lead to subluxation, pain, & impingement.
  - Shoulder pain during spastic stage can lead to subluxation, pain, & impingement.

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.
Cognitive & perceptual Dysfunction

- Cognition: act or process of knowing, including awareness, reasoning, judgment, intuition, & memory
- Perception: integration of sensory impressions into information psychologically meaningful → ability to select stimuli & interpret them

Clinical indicators of deficits:

<table>
<thead>
<tr>
<th>Difficulty or inability to</th>
<th>Patients may:</th>
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<tbody>
<tr>
<td>perform simple tasks</td>
<td>hesitate many times</td>
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<tr>
<td>independently/safely</td>
<td>appear distracted and frustrated</td>
</tr>
<tr>
<td>initiating/ completing a task</td>
<td>exhibit poor planning</td>
</tr>
<tr>
<td>switching from one task to another</td>
<td>be inattentive to one side of the body</td>
</tr>
</tbody>
</table>

- Multi-infarct dementia: from multiple small infarcts of the brain, abrupt onset, 7-10 days, stepwise & punctiform deterioration of intellectual function may coexist w/ Alzheimer's disease
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 & use regulation to achieve goal
  - Ability to initiate & maintain action & switch, stop action
  - Effectiveness performance: quality control of self-correlation of behaviors

- Prosopagnosia: inability to recognize the faces of familiar people
- Stereognosia: inability to recognize forms by handling/touching them
- Disorder: astereognosia

- 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 contralateral side
  - Sensory loss is common (a recovery rate too)

- 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
  - W can't locate items in the drawer

- Anognosia: severe perceptual impairment
  - Severe form of neglect
  - Denies body part as their own or denies the paresis/paralysis
  - Spontaneously resolves within the 1st 3 months after CVA
  - More common in patients w/ @ hemispheric lesions
  - Safety is a concern

- Sensoagnosia: 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

Dementia is associated w/ mortality rules

Old Material

- ICF framework: international classification of functioning, disability, & health
  - Body functions & structure, activity, & participation
**Motor Function & Spasticity:**
- **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 (0-6)

**IV STEP goals: 1) Participation, prediction, plasticity, prevention:**
- Explore PT's role in preventing disabling conditions
- Evaluate new ways to classify movement disorders
- Estimate 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
  - Confrontational weakness, sensory loss, cognitive confusion
  - LE involvement
- **Middle Cerebral A:** affects frontal, temporal, parietal, occipital lobe, internal capsule & structures
  - Most common stroke
  - Contralateral weakness, sensory loss, vision problems, aphasia
  - LE involvement
- **Posterior Cerebral A:** affects occipital lobe & part of thalamus
  - Less common stroke
  - Contralateral weakness, sensory loss, vision problems
  - **Verteobasilar 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 lean & shortened step width
    - Circumduction, hip hiking
  - **Quadriceps →** knee hyperextension during loading response
    - Trunk leans forward & moves the cord
  - **Hamstrings →** knee flexion at initial contact, knee buckling at stance phase
sumened
stop
length
d
Hanae
time
quad
demand
crown
gait
Adductors
causes
ccontralateral
pevals
to
d
medial
displacement
of
leg
at
stance
phase
scissoring
gait

Weakness/
paralysis:

PF
excessive
knee
flexion
during
stance
phase

decreased
heel
rise
during
terminal
stance

DF
flat
foot/
forefoot
contact
at
initial
contact

Trot
Clearance
during
Swing
phase

Quadiceps
poor
knee
control
during
loading
response

destabilizes
knee
during
midstance

Hip
Flexors
problems
of
limb
advancement
during
Swing
phase

Hip
Extensors
causes
forward
trunk
lean
during
stance
phase

Hip
Abductors
pended
Engberg
Gait
during
stance
phase

Synergies:

VE
Flexion:

Scapular
retraction
/relaxation

Shoulder
abduction,
ER

Elbow
flexion

Wrist
flexion

Fingers
flexion

LE
Flexion:

Hip
Ext
Add
IR

Knee
Ext

Ankle
PF,
Inv

TIP

Compensatory
Motor
Strategies:

Ante
Strategy:

Small
perturbations
with
firm
ground

MMS
proximal

Foreward
Sway:
Gastrocnemius,
Hamstrings,
Paraspinals

Backward
Sway:
Hip
Ant
Quads
Abs

Hip
Strategy:

Big
&
Fast
perturbations
with
soft,
compliant,
&
Harrow
BoS

MMS
distal

Foreward
Sway:
Abs,
Quads

Backward
Sway:
Paraspinals,
Hamstrings

Stepping
Strategy:

Very
Large
&
Fast
perturbations
or
ankle/hip
impairments

Foreward
Sway:
Step
Forward

Backward
Sway:
Step
Backward

GHB
Eaa
a.m
<table>
<thead>
<tr>
<th><strong>Epidemiology</strong></th>
<th><strong>Stroke</strong></th>
<th><strong>Parkinson’s Disease</strong></th>
<th><strong>Traumatic Brain Injury</strong></th>
<th><strong>mTBI/Concussion/PCS</strong></th>
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<tbody>
<tr>
<td></td>
<td>#5 leading cause of death in US, F&gt;M, majority &gt;65 y/o, African-Americans most impacted, increased risk with previous hx of stroke</td>
<td>1 mil people living with PD, risk increases with age, average age of onset = 50-60 y/o, M&gt;F</td>
<td>Vary by age, MVA, falls, highest risk = 0-4 and 15-19 y/o, M&gt;F</td>
<td>Any age, falls, MVA, violence, playground injuries, sports, F&gt;M, ~150k concussions every year PCS: &gt;10 days of symptoms, &gt;3 wks of symptoms for HS athletes</td>
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| **Etiology**       | **Ischemic:** lack of blood flow due to narrowing opening for oxygen to go through; cerebral thrombus, embolism, atherosclerosis | **Idiopathic:** 10% genetic, 3 different onsets possible | **Open head injury:** skull is fractured, usually associated with intracranial hemorrhage | **Direct blow to the head, neck, face, or body causing brain injury** |
|                   | Hemorrhagic: uncontrollable bleeding due to compression of brain; intracerebral or subarachnoid hemorrhage | **Parkinson-like diseases:** Secondary parkinsonism, Parkinsonism-plus syndromes | **Closed head injury:** injury with skull intact |  |

| **Pathophysiology** | **Ischemic:** complete occlusion of blood flow leads to a core area of neuronal cell death | **Degeneration of dopamine producing neurons in the basal ganglia:** neuronal death of substantia nigra causing less DA on the striatum | **Primary brain injury:** damage occurs at the moment of impact; focal brain injury, blast injury, diffuse axonal injury | **Blunt force:** ion channel dysfunction, metabolic energy crisis, physiologic axonal stretching |
|                    | - Release of glutamate | - Lewy body inclusion bodies develop with disease progression: protein accumulation | **Secondary brain damage:** occurs within min-hours after injury; intracranial hematomas, herniation, hypoxic-ischemic injury, epilepsy/seizures, intracranial infections | **Blast-related:** shock waves disrupts brain tissue, penetrating injury and blunt trauma |
|                    | - Altered Ca2+ ion channels causes influx of Ca2+ into neuron | | | |
|                    | - Activation of destructive Ca sensitive enzymes | | | |
|                    | - Further neuronal cell death (prenumbra) | | | |

| **Expected & Unique Neurological Impairments** | **Muscle weakness, sensory loss, cognitive confusion, vision problems, aphasia, paralysis, impulsive behavior, poor judgement, flat affect, depression** | **TRAP:** tremor, rigidity, bradykinesia/akinesia/hyperkinesia, postural instability | **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** |
|                                                  | 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 | | | **Blast-related:** dizziness, vertigo, hearing loss, cog. Deficits, HA, disequilibrium |
|                                                  | | Start hesitation, freezing gait, depression, anxiety, psychosis, dementia, sleep disturbances, lack of smell, excessive sweating, saliva production, oily skin, dysarthria | | PCS: depression/anxiety, panic, sleep alterations |

<p>| <strong>Outcome Measures</strong> | <strong>Stroke specific:</strong> Fugl-Meyer, STREAM, SIS | <strong>Body functions:</strong> MDS-UPDRS, MOCA, H &amp; Y, PD fatigue scale, FOG questionnaire | <strong>GCS, CRS-R, LOCF. Berg, HiMAT, 10m walk, 6min walk</strong> | <strong>GCS, SAC, SCAT3, MACE</strong> |
|                     | Postural control/balance: Berg, PASS, Tinetti, TUG, CTSIB | Activity: 10 min walk, 6 min, FGA, Mini BESTest, 5xSTS, 9hole peg, MDS-UPDRS, TUG, ABC scale, pull test | Participation: PDQ-8, PDQ-39 | |
|                     | | Participation: PDQ-8, PDQ-39 | | |</p>
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<th><strong>Epidemiology</strong></th>
<th><strong>SCI</strong></th>
<th><strong>GB</strong></th>
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<tbody>
<tr>
<td>17,000 new cases/year in US</td>
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<td>1-4 cases per 100,000</td>
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<tr>
<td>243,000-347,000 currently living in US</td>
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<td>3000-6000/year in the US</td>
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<td>Avg age: 42 y/o</td>
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<td>Males &gt; females</td>
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<td>Majority non-Hispanic white males</td>
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<tr>
<td>Types of Injury</td>
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<tr>
<td>Traumatic: MVA (most common), falls, violence, sports</td>
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<tr>
<td>Non-Traumatic: vascular dysfunction, vertebral subluxation due to RA/DJD, spinal neoplasm, syringomyelia, abscess, infection (syphilis, transverse myelitis), MS or ALS</td>
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<tr>
<td>Traumatic more common of the two</td>
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<tr>
<td>Common MOI</td>
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<td>C-spine: concomitant rotation, lateral flexion, shearing forces</td>
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<tr>
<td>Whiplash, falling backwards and landing with chin on tub</td>
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<tr>
<td>Forces of: flexion, axial loading, distraction, extension,</td>
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<tr>
<td>T-spine: GSW, MVA, falls</td>
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<tr>
<td>Flexion, axial loading</td>
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<td></td>
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<tr>
<td>Less common than cervical, more likely to be complete</td>
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<tr>
<td>Most common site: T12-L1 junction (transition from UMN to LMN)</td>
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<tr>
<td>L-spine: falls, MVA, GSW, direct load onto spine</td>
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<tr>
<td>Forces of: flexion, axial loading, flexion combined with distraction or rotation</td>
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<thead>
<tr>
<th><strong>Pathophysiology</strong></th>
<th><strong>SCI</strong></th>
<th><strong>GB</strong></th>
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<tbody>
<tr>
<td>Damage due to impingement and/or compression of cord: bony/soft tissue, penetrating/non-penetrating</td>
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<tr>
<td>Blunt trauma: primary neuronal damage to cell bodies/axons</td>
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<tr>
<td>Secondary injury causes the most damage: ischemia, demyelination, edema, necrosis</td>
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<tr>
<td>Initial trauma → apoptotic cell death, interruption of blood flow, inflammation, increased amino acids → ischemia, disruption of homeostasis → calcium accumulation and demyelination → cell death</td>
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<thead>
<tr>
<th><strong>Expected and Unique Neurological Impairments</strong></th>
<th><strong>SCI</strong></th>
<th><strong>GB</strong></th>
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<tbody>
<tr>
<td>Complete Impairment</td>
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</tr>
<tr>
<td>NO motor/sensory function is preserved in S4-5</td>
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<tr>
<td>Incomplete Impairment</td>
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<tr>
<td>Sensory but not motor function is preserved below the neurological level and includes S4-5</td>
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<tr>
<td>Motor function is preserved below the neurological level, and &gt; 1/2 of key muscles below level have a muscle grade &lt;3</td>
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<td></td>
</tr>
<tr>
<td>Motor function is preserved below the neurological level, and &gt; 1/2 of key muscles below level have a muscle grade &gt;3</td>
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<tr>
<td>Presentation</td>
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<tr>
<td>Tetraplegia vs. Paraplegia (incomplete tetraplegia most common)</td>
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<tr>
<td><strong>Clinical Syndromes</strong></td>
<td></td>
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<tr>
<td>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</td>
<td></td>
<td></td>
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<tr>
<td>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</td>
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<tr>
<td>Central Cord Syndrome: most common due to hyperextension injury, paralysis and sensory loss in UE, varying involvement in trunk and LE's</td>
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<tr>
<td>Cauda Equina Injury: injury to the lumbosacral nerve roots of the cauda equina, LMN signs, flaccid paralysis of LE's, areflexic bowel and bladder (difficult for self-care)</td>
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<tr>
<td><strong>Primary/Secondary Impairments</strong></td>
<td></td>
<td></td>
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<tr>
<td>Motor output, sensory input, autonomic dysreflexia, spastic hypertonia, cardiovascular, temp, pulmonary, bowel and bladder, sexual dysfunction</td>
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<table>
<thead>
<tr>
<th><strong>Outcome Measures</strong></th>
<th><strong>SCI</strong></th>
<th><strong>GB</strong></th>
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<tbody>
<tr>
<td>ASIA’s International Standards for Neurological Classification of Spinal Cord (ISNCSCI)</td>
<td></td>
<td>System specific for FSS, FIS, VAS-F</td>
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<tr>
<td>Function Specific → Barthel Index, Modified Hughes Scale of GBS disability, FIM</td>
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<tr>
<td>Participation → Short form 36, Nottingham Health Profile</td>
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<thead>
<tr>
<th><strong>Practice Settings</strong></th>
<th><strong>SCI</strong></th>
<th><strong>GB</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>SCI patients can be seen in all PT settings due to the variety of presentations along with the level of injury and age.</td>
<td></td>
<td>GB patients can be seen in all PT settings depending on the progression of the diagnosis and ambulatory ability.</td>
</tr>
<tr>
<td>Epidemiology</td>
<td>MS</td>
<td>ALS</td>
</tr>
<tr>
<td>--------------</td>
<td>----</td>
<td>-----</td>
</tr>
<tr>
<td>• 400,000 people</td>
<td><strong>most common</strong></td>
<td>30,000 people</td>
</tr>
<tr>
<td>• Onset typically between 20-40 years of age (30 y/o is average)</td>
<td></td>
<td>• High incidence (Guam and Japan - Western Pacific form)</td>
</tr>
<tr>
<td>• Women &gt; men</td>
<td></td>
<td>• Onset usually mid-to-late 50s, Men &gt; women</td>
</tr>
<tr>
<td>• Increased risk with those living above 40 latitude (up north)</td>
<td></td>
<td>• Sporadic ALS &gt; familial ALS</td>
</tr>
<tr>
<td>• <strong>Risks:</strong> genetics, race, geography</td>
<td></td>
<td>• 70-80% limb onset, 20-30% bulbar onset</td>
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</tbody>
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<table>
<thead>
<tr>
<th>Etiology</th>
<th>MS</th>
<th>ALS</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Unknown cause</td>
<td></td>
<td><strong>Known Risk Factors</strong></td>
</tr>
<tr>
<td>• Chronic – stays with them for the rest of their life</td>
<td></td>
<td>• Disease-causing mutations (SOD1, alsin), clusters (Western Pacific ALS/PDC), male &gt; female, mid to late 50’s, familial</td>
</tr>
<tr>
<td>• Demyelinating disease of the CNS and can have lesions anywhere in the CNS, which makes presentation extremely variable</td>
<td></td>
<td>Possible Risk Factors</td>
</tr>
<tr>
<td>• <strong>Autoimmune disorder</strong></td>
<td></td>
<td>• Neurotoxicant exposures: lead, mercury, pesticides</td>
</tr>
<tr>
<td>• Viral infection triggers the immune response but virus is unknown</td>
<td></td>
<td>• Lifestyle factors: smoking, alcohol</td>
</tr>
<tr>
<td>• Genetic susceptibility to immune system dysfunction</td>
<td></td>
<td>• Certain occupations: electrical/industrial workers, farmers</td>
</tr>
<tr>
<td>• 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</td>
<td></td>
<td>• Trauma: skeletal trauma, fractures, electric shock w LOC</td>
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<table>
<thead>
<tr>
<th>Pathophysiology</th>
<th>MS</th>
<th>ALS</th>
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<tbody>
<tr>
<td>• 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.</td>
<td></td>
<td><strong>Affects</strong></td>
</tr>
<tr>
<td>• <strong>Inflammation</strong> also occurs because of the presence of damage, which causes further swelling, impairs nerve conduction, and exacerbates the impairments</td>
<td></td>
<td>• UMN in cortex and corticospinal tracts</td>
</tr>
<tr>
<td>• BOTH cause slower nerve conduction rates and impairments</td>
<td></td>
<td>• Brainstem nuclei for CN V, VII, IX, X, XII</td>
</tr>
<tr>
<td>• Lesions can be anywhere in the CNS, but typically in the white matter (myelinated axons, tracts, etc.)</td>
<td></td>
<td>• Anterior horn cells in spinal cord</td>
</tr>
<tr>
<td>• <strong>Optic pathway, corticospinal tracts, dorsal column of the SC and cerebellar pederuncules are particularly susceptible</strong></td>
<td></td>
<td>• Sensory system and spinocerebellar tract</td>
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<table>
<thead>
<tr>
<th>Expected and Unique Neurological Impairments</th>
<th>MS</th>
<th>ALS</th>
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</thead>
<tbody>
<tr>
<td><strong>Common impairments</strong></td>
<td></td>
<td><strong>During Stages of Disease</strong></td>
</tr>
<tr>
<td>• Visual impairments: blurred vision, altered acuity, optic neuritis, diplopia</td>
<td></td>
<td>• Sprouting: early on, healthy intact axons sprout and innervate synaptic sites that were previously activated by the damaged neurons</td>
</tr>
<tr>
<td>• Damage to corticospinal tracts: paresis/plegia, spasticity, hyperreflexia (+ Babinski, + clonus)</td>
<td></td>
<td>• Contiguous progression: locally spread before moving to another area</td>
</tr>
<tr>
<td>• Damage to dorsal columns: impaired proprioception, paresthesia, dysesthesias</td>
<td></td>
<td>• Rostral/caudal spread: late stage</td>
</tr>
<tr>
<td>• Damage to cerebellar pederuncles: balance, coordination, tremor, ataxia, hypotonia, vestibular impairments</td>
<td></td>
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<thead>
<tr>
<th>Symptoms</th>
<th>MS</th>
<th>ALS</th>
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<tbody>
<tr>
<td>• Sensory, pain, visual, motor, fatigue, coordination, balance, gait, mobility, speech and swallowing, depression, emotional, cognitive, bladder, bowel, sexual</td>
<td></td>
<td><strong>Clinical Manifestations</strong></td>
</tr>
<tr>
<td>• Can suffer from bulbar paralysis</td>
<td></td>
<td>• Highly variable</td>
</tr>
<tr>
<td>• Fatigue is a primary problem in patients and can be disabling</td>
<td></td>
<td>• 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</td>
</tr>
<tr>
<td>• Patients with affective symptoms (emotional) are at higher risk of intellectual and cognitive impairments</td>
<td></td>
<td>• Symptoms are focal and asymmetrical at onset</td>
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<thead>
<tr>
<th>Subtypes</th>
<th>MS</th>
<th>ALS</th>
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<tbody>
<tr>
<td>• Relapsing-Remitting MS (RRMS): most common, acute attacks followed by remission until loss of oligodendrocytes causes inability to return to baseline, has best prognosis</td>
<td></td>
<td><strong>LMN Pathology Impairments</strong></td>
</tr>
<tr>
<td>• Secondary Progressive MS (SPMS): steady and irreversable decline with/without acute attacks</td>
<td></td>
<td>• Muscle weakness: LMN weakness worse than UMN weakness <strong>(cardinal sign)</strong></td>
</tr>
<tr>
<td>• 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</td>
<td></td>
<td>• Hyporeflexia and hypotonicity</td>
</tr>
<tr>
<td>• Progressive Relapsing MS (PRMS): least common, most severe, steady deterioration from onset with occasional acute attacks</td>
<td></td>
<td>• Fatigue: as motor neurons die, remaining ones become burnt out and work harder for the same result</td>
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<thead>
<tr>
<th>Outcome Measures</th>
<th>MS</th>
<th>ALS</th>
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<tbody>
<tr>
<td>• Expanded Disability Status Scale (EDSS)</td>
<td></td>
<td><strong>Atrophy</strong></td>
</tr>
<tr>
<td>• MS Functional Composite (MSFC)</td>
<td></td>
<td>• Muscle cramps: unknown cause</td>
</tr>
<tr>
<td>• Multiple Sclerosis Quality of Life-54 (MSQOL-54)</td>
<td></td>
<td><strong>Fasciculations:</strong> random twitches of muscle fibers</td>
</tr>
<tr>
<td>• Multiple Sclerosis Impact Scale (MSIS-29)</td>
<td></td>
<td><strong>UMN Pathology</strong></td>
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<thead>
<tr>
<th>Practice Settings</th>
<th>MS</th>
<th>ALS</th>
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<tbody>
<tr>
<td>• MS patients can be seen in all PT settings due to the variety of symptoms and presentations along with the age of onset.</td>
<td></td>
<td>• ALS Functional Rating Scale (ALSFRS)</td>
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<tr>
<td></td>
<td></td>
<td>• ALS Assessment Questionnaire (ALSAQ-40)</td>
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<tr>
<td></td>
<td></td>
<td><strong>Other impairments</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Respiratory: may be put on the ventilator</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Cognitive: mild deficits to dementia</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Bulbar Pathology Impairments</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• <strong>Bulbar muscle weakness</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• <strong>Spastic/flaccid bulbar palsy</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Dysarthria and dysphagia</td>
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<tr>
<td></td>
<td></td>
<td>• Sialorrhea</td>
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<tr>
<td></td>
<td></td>
<td>• Pseudobulbar affect</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>Other Impairments</strong></td>
</tr>
<tr>
<td></td>
<td></td>
<td>• ALS patients are also seen in all PT settings – a multidisciplinary approach is most advantageous</td>
</tr>
</tbody>
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