MEDICAL COMPLICATIONS IN CHRONIC SCI

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Health Promotion in SCI

- Freedom from preventable disease
- Early detection of disease
- Limit or manage effects of disease or disability
- Goal: Improve HRQOL, participation, survival, rehospitalization

SCI HEALTH MAINTENANCE
Distinct Themes

- Missed/Delayed Diagnosis
- Treatment Considerations
- Unique conditions
- Functional impact
- Altered presentation
- Equipment needs

COVERED IN OTHER LECTURES:

- DVT and PE (Acute)
- CV: Hypotension/orthostatic hypotension
- Ventilatory failure
- Genitourinary
- Hypercalcemia
- GI/Abdominal
- Pressure Ulcers
REHOSPITALIZATION AFTER SCI

Tetraplegia
- Respiratory
- Model Systems
- 1995-2002
- Cardenas et al 2004

Paraplegia
- Pressure Ulcers

LEADING CAUSE = GENITOURINARY

PRIMARY CAUSE OF DEATH

General Population
- Heart disease
- Cancer
- Cerebrovascular disease

SCI (Model Systems)
- Pneumonia/respiratory 22%*
- Heart Disease 19.6%**
- Hypertensive & ischemic 7.8%
- Non-ischemic 11.8%
- Infection 10.4% (sepsis)

*#1 in tetraplegia
**#1 in paraplegia and AIS-D (all levels)

Preventive Care: Veterans with SCI
Weaver and LaVela

Hypertension 49% vs. 26%
Lipid Disorders
Diabetes 19% vs. 7%
Obesity
Infection Pneumonia, UTI, pressure sores
- Less likely to be vaccinated against flu
- More likely to have received pneumococcal vaccine
Cancer Bladder

OUTLINE:
Medical Complications of Chronic SCI

- Review by system:
  - Why these associated medical conditions occur
  - Key concepts marked with:  
  - Pitfalls in work-up and treatment
  - Extra slides marked “For Your Review/Reference” for self-study

- Cardiovascular
- Respiratory
- Musculoskeletal
- Pain
- Late neurologic complications
- Spasticity
SCI: CARDIOVASCULAR COMPLICATIONS

SCI AND AUTONOMIC DYSFUNCTION

- T1-T7: heart and blood vessels
- More common in more complex and higher cervical level injuries

CARDIOVASCULAR COMPLICATIONS
Themes

- Autonomic dysregulation
- Blunted CV exercise response
- Increased prevalence of CV disease risk factors

AUTONOMIC DYSREFLEXIA (AD):

1. Paroxysmal episodes of hypertension
2. Possibly associated with characteristic signs and symptoms
3. Occurring as a response to a noxious stimulus
4. In susceptible patients: T6 or above

Cause: disconnection between supraspinal (brain) control and preganglionic sympathetic neurons in thoracolumbar cord
RISK FOR AUTONOMIC DYSREFLEXIA

**Neurologic Level: T6 level or above**
- 48-85% with SCI above T6
- Rarely reported with lower level injuries

**ASIA Impairment**
- Any (A&B more common)

**Onset**
- Rarely sooner than 2 months post-injury
- 92% had first episode within the first year

AD CLINICAL PRESENTATION:
Variable

**Symptoms:**
- Pounding headache
- Flushing above LOI
- Nasal Congestion
- Anxiety
- Blurred/spotty vision

**Signs:**
- Elevated BP
- Bradycardia (tachycardia less frequently)
- Sweating above LOI
- Piloerection
- Cardiac arrhythmia

Sorting Out the Features of AD

**Above: Parasympathetic**
- Headache
- Flushing
- Nasal Congestion
- Bradycardia

**Injury Level**

**Below: Sympathetic**
- Pallor/Cool extremity
- Piloerection
- Bladder/Intestinal sphincter contraction
AD: Differential Diagnosis

- Cardiac ischemia: atypical presentation possible
- Pre-eclampsia
- Intrathecal Baclofen Withdrawal
- Pheochromocytoma
  - Pitfall: exceedingly rare. For entire US population with chronic SCI (296,000), expect only 1 per year

Possible sentinel of more insidious pathology in otherwise asymptomatic patient

AD: TRIGGERS

USUALLY A PAINFUL STIMULUS

- **Bladder (75-82%):** distension, infection, stones, procedure
- **Bowel (13-19%):** distension from fecal impaction
- Pressure ulcers
- Ingrown toenails
- Abdominal emergencies
- Fractures/soft tissue injury
- Body positioning
- Labor, Delivery
- Genital: Ejaculation, epididymitis, testicular torsion
- Anesthesia induction
- Urodynamics (60-85% of individuals w/tetraplegia)
- DVT, pneumonia, tight clothing

AD TREATMENT:

Consortium for Spinal Cord Medicine

[Consumer and Health Care Provider Guides](www.pva.org)

AUTONOMIC DYSREFLEXIA COMPLICATIONS

- Subarachnoid or intracerebral hemorrhage
- Retinal hemorrhage
- Cardiopulmonary: AFib, MI, pulmonary edema
- Seizure
- Death
AUTONOMIC DYSREFLEXIA
INITIAL TREATMENT

Elevate head  
Loosen constrictive clothing  
Monitor BP, HR frequently  
Systematic survey

Principle: Identify and eliminate offending stimulus

AD: SURVEY FOR CAUSATIVE FACTORS

BLADDER FIRST
• Confirm bladder is decompressed  
• Indwelling catheter: check for kinks; irrigate or change  
• No catheter: Ultrasound or instill 2% lidocaine jelly and insert catheter

BOWEL NEXT
• Rectal exam  
• 2% lidocaine  
• Manual evacuation

Other causes
Note: May need medications to control BP

AD: PHARMACOLOGIC TX

□ Indications:  
   □ Per CPG: should be considered if SBP>150  
   □ Relative HTN: SBP elevation > 20-40 mmHg over baseline  
   □ Ideally, rapid-onset and short duration  
□ Risks:  
   □ Rebound hypotension once noxious stimulus is removed

AD: PHARMACOLOGIC AGENTS

□ Calcium channel blockers  
□ Alpha blockers (e.g. prazosin)  
□ Beta blockers  
□ Direct arterial/venous dilators (e.g. nitroglycerin paste)  
□ Indirect arterial/venous dilators  
□ Ganglionic blockers  
□ Sympatholytic agents

Guidelines don’t make a recommendation which med to use

Typical protocol:  
□ First line: NTG ointment  
□ Second line: hydralazine
**CORONARY HEART DISEASE AND SCI**

Conflicting (or weak) evidence: increased prevalence of CHD in SCI

Cardiovascular mortality elevated

- Frankel 1998: especially if level above T5
- Age ≤ 30 years old: 6x general population
- Age 31-60: 30% higher
- > 60 yrs: no significant difference

Recent systematic review: SCI is not an independent risk factor for carbohydrate and lipid metabolism disorders or cardiovascular morbidity and mortality

*Wilt TJ et al January 2008*

**OTHER CARDIOVASCULAR RISKS IN SCI**

Many CV risk factors may be more prevalent with SCI:

↓ cardiac output

- HTN
- Risk for silent ischemia
- ↓ Physical activity, exercise tolerance
- Lipid disorders
- Diabetes/
  Glucose intolerance?
- Obesity/
  ↑ % body fat

May result in increased risk for premature CHD

**METABOLIC SYNDROME & SCI**

- Definition of Metabolic Syndrome
  - IDF vs NCEP ATPIII vs WHO
- Prevalence estimates: studies support increase in SCI
  - 34-44.8% in SCI
  - Nash 2007, Maruyama, Castillo 2007

**CORONARY HEART DISEASE AND SCI: Diagnosis**

May be more difficult to diagnose:

- Atypical or absent symptoms of myocardial ischemia due to sensory loss, esp. above T5
  - Jaw pain/toothache
  - AD
  - Nausea, SOB, spasticity increase, fatigue
- Diagnostic testing considerations
  - Arm ergometry for paraplegia
  - Pharmacologic stress testing most practical
CORONARY HEART DISEASE AND SCI: Management Considerations

- Similar treatment options as in general population
- May need retraining in energy conservation strategies
- Low resting BP
- Modified cardiac rehab may be feasible
  - Stiens et al., PMR Clin NA 1995

CHD PREVENTION: Dyslipidemia and SCI

HDL<35
- 24-40% of SCI vs 10% in general population
- LDL no different

Treatment
- Evidence does not suggest different treatment threshold
- Lifestyle changes: ? Possible to increase activity level

CHD PREVENTION: Obesity

- Definition in SCI: 2 of 3 individuals with SCI overweight
- Decrease in fat-free body mass → BMI may be misleading
  - 13% greater fat per unit BMI
- ? Overestimation of resting metabolic rate by 14-27%

Recommendation: 
- Basal energy requirement by 10% for low paraplegia, 25% high tetraplegia

RESPIRATORY DISEASE IN CHRONIC SCI
Importance of Respiratory Disorders in SCI Medicine

- Leading cause of death in 1st year and chronic SCI
  - Pneumonia accounts for 67.4%
  - 20% of deaths during first 15 years post-SCI
- NSCISC data: increased mortality in tetraplegia (9-18x) vs age-matched general population
- Contributor to high cost of care:
  - Skilled care is required outside of hospital
  - VA: double rate of PNA OP visits vs. non-SCI (Smith 2007)
  - Re-hospitalizations

Chronic SCI: Respiratory Disorders

- Pneumonia, (Secretion Management, Atelectasis)
- Ventilatory Failure
- Sleep-Disordered Breathing
- Venous Thromboembolic Disease

SCI: LUNG VOLUMES

<table>
<thead>
<tr>
<th>Lung Volume</th>
<th>SCI Effect vs. non-SCI</th>
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<tbody>
<tr>
<td>TLC</td>
<td>↓</td>
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<tr>
<td>VC</td>
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<td>ERV</td>
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<td>FEV1</td>
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<tr>
<td>RV</td>
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- Restrictive disease

Chronic SCI:
- FVC = 60-70% of normal

RESPIRATORY COMPLICATIONS: PNEUMONIA

- Aspiration: Dysphagia
- Ventilator-associated
- Community-acquired or viral
  - Streptococcal PNA most common
  - Yearly influenza vaccination recommended, unless contraindications
  - Consider pneumococcal vaccination
Late-Onset Respiratory Insufficiency After SCI

**Symptoms**
- Tachypnea, dyspnea
- Daytime somnolence, fluctuating mental status, increased positional influence on breathing
- Erythrocytosis

**Treatment**
- CPAP/BiPAP
- O2
- Mechanical ventilation/tracheostomy

**Associated Factors**
- Ventilator use with SCI
- Recurrent atelectasis/pneumonia
- VC < 2 L
- Nocturnal hypercapnia
- Mean SaO2 < 95%

SLEEP-DISORDERED BREATHING & SCI

**SCI:** 26-65%
- Acute tetraplegia: 83%
- Chronic tetraplegia: 25-40%

**Primarily obstructive sleep apnea**

**Associated factors?**
- Increased BMI and neck circumference
- But common in non-obese
- High motor level
- Age
- Baclofen or other antispasticity medication

CONSEQUENCES OF SLEEP APNEA

**Presumed similar to general population**
- Excessive daytime sleepiness; MVC's
- Cardiovascular: HTN; arrhythmias

**Possible consequences in SCI:**
- Poor participation in rehabilitation
- Impaired wound healing due to hypoxia or sleeping in chair
- Unexplained non-ischemic cardiac deaths

SLEEP-DISORDERED BREATHING & SCI

**Ask about symptoms**
- Daytime somnolence, morning headache
- Restorative sleep
- Snoring/gasping

Positive response should prompt evaluation
- Complete polysomnography

**Dilemma:** majority with SCI don’t complain of daytime somnolence

**Treatment:** same as general population
- CPAP, BIPAP, O2
- Reduce weight, ETOH
Reinstitution of VTE Prophylaxis in Chronic SCI

- Risk of DVT drops within 3 months of injury
- No unbiased estimate of how commonly DVT occurs in chronic SCI
- Kim (1994): 229 admissions with SCI>6 months duration
  - 43 patients underwent radionuclide venogram
  - 22 "clinically DVT-free": 1/22 with chronic DVT
  - 21 "DVT-suspected": 2/21 with chronic DVT and 4/21 with acute DVT
  - 9.3% incidence of acute DVT

- Frisbie and Sharma (2012)
  - Systematic review spanning 1956-2009
  - 0 - 18.7% using autopsy, imaging, clinical, ECG
  - Possible subclinical presentations with pulmonary HTN on Echo

VTE PROPHYLAXIS FOR CHRONIC SCI

- Rimler 2011:
  - 260 Plastic Surgery cases without VTE prophylaxis
  - No cases of VTE in 2 months post-operative time
  - 5 cases with VTE 2.5-37 months post-operative time
    - 4/5 had history of VTE

- Unclear: pharmacologic agent and dose; IVC filters?
- Awaiting new CPG

Possible Indications (CSCM CPG)

- Lower extremity fracture or lower limb surgery
- Other surgeries?
- Hospitalization due to acute medical illness?
- Anticipated prolonged bedrest (e.g. pressure ulcer)??

Consider additional risk factors: cancer, prior DVT, CHF, age>70, obesity

MUSCULOSKELETAL CONDITIONS IN CHRONIC SCI
MUSCULOSKELETAL COMPLICATIONS IN SCI

Bone Health
- Osteopenia, osteoporosis, fractures
- Heterotopic ossification
- Charcot joint or spine

Neck and shoulder pain
- Rotator cuff tear: 71% of individuals with paraplegia with shoulder pain
- Arthropathy

Wrist pain
- Carpal tunnel syndrome/nerve entrapment
- Overuse injuries

SCI AND BONE LOSS

Time Course
- Rapid onset
- Peak: 10-16 weeks
- Continues after 1 year
- New homeostasis later

Knee BMD loss at 1.5-2 years post-injury
- M: 33%
- F: 50% or more

Lazo 2001: DXA
- Osteoporosis 60%
- Osteopenia 19.5%

SCI AND BONE LOSS

Patterns of Loss

Exclusively below level of injury in 1st 24 months
- Pelvis and lower limbs, usually preserves spine (F)
- Investigate any unusual osteopenia of spine or distal radius

Trabecular > Cortical bone

Pathophysiology
- Acute: Resorption > Formation
- Multifactorial: mechanical, hormonal, neurogenic

BONE LOSS AND SCI

- Manifests as increased incidence of fractures
  - MSCIS: 15 yrs = 39%
  - Lazo 2001 = 34%

- Distal femur > proximal tibia

- Fracture threshold – possibly 50% reduction in BMD at knee
  - Garland, Top Spinal Cord Inj Rehabil 2005
Prevention of Osteoporosis Following SCI

**No consensus on prevention**

Correct nutritional deficiencies
- Calcium, vitamin D (limited evidence in SCI)
- Ca may be beneficial in women w/ chronic SCI
- Ca 1000 mg/d likely beneficial if U Ca <250 mg/d and mid-normal PTH (North Am Menopause Society, Ott 2001)

Correct endocrine abnormalities: hypogonadism
- Low testosterone: earlier, higher prevalence by decade of life, greater rate of age-related decline (Bauman 2014)

Bone Loss Prevention Following SCI

With ACUTE, motor-complete SCI, current research shows no definite long-term benefit from:

- Standing frame
- Electrical stimulation of lower limb muscles
- Bisphosphonates

? Benefit from early intervention, longer duration, higher intensity/frequency

FRACTURE MANAGEMENT

CHRONIC SCI

**Cause: can be minor injury**

Fracture rate up to 40% in chronic SCI

- Paraplegics > tetraplegics
- 10x greater complete > incomplete injuries

**Symptoms**
- Soft tissue swelling, warmth, increased spasticity, AD
- High index of suspicion necessary
- Counsel patient on risk and symptoms

FRACTURES IN CHRONIC SCI: PITFALLS

- Missing a fracture after minor trauma
- Not considering DVT prophylaxis
- Skin breakdown under circumferential cast
- Unnecessary operative treatment of fractures best managed conservatively
- Failure to maximize patient’s function while wearing an immobilizer to treat fracture
MUSCULOSKELETAL COMPLICATIONS: Upper Limb

- High prevalence in all neurological categories
- Shoulder: approximately 50% (paraplegia)
- Contributors: MWC propulsion, transfers, assistive devices
- Shoulder protection (See CPG)
  - Limit overuse/impact
  - Minimize sustained overhead activities, transfers
  - Wheelchair seating
- Aggressive treatment of carpal tunnel syndrome may be indicated early
- Post-op limitations impact mobility

HETEROTOPIC OSSIFICATION

Neurogenic Heterotopic Ossification: Definitions

Pathologic ectopic ossification
- occurring within soft tissue planes surrounding neurologically affected joints
- occurs in individuals with CNS injury, burns

Etiology: Poorly understood. Role of trauma?

Histology
- Osseous, not just calcification
- Histologically and biochemically indistinguishable from normal bone or callous

Mechanism of Heterotopic Ossification Formation
Patterns of HO in SCI

Sites of ossification:

- Extra-articular: between connective tissue/muscle layers
- Most common:
  - Hip (anteromedial): 90% between ASIS and lesser trochanter
  - 2nd: Knee (medial epicondyle)
  - Other: shoulder, elbow

Located below neurologic level (unless patient also has TBI or other causative factor)

Patterns of HO in SCI

Time course

- Onset 1-6 months
- Peak 2 months
- May occur years later

Maturation is centripetal

Risk/associated factors

- Male
- Older age
- Complete injury
- DVT
- Spasticity
- Pressure ulcer

HO Incidence in SCI

- Overall: 10-53%
- Most cases: incidental radiographic finding only
  - Occasionally worked up as a sarcoma
- 10% of cases severe
  - Large amount of ossification
  - Significant ROM and functional limitation
  - 5-8% ankylosis

HO: Clinical Presentation & Differential Dx

Clinical Presentation

- Acute
  - Warmth, swelling, erythema
  - +/- Fever (esp nocturnal); pain

- Indolent
  - Decreased joint ROM (most common sign)

Differential

- HO
- DVT
- Trauma (e.g. fracture or ligament disruption)
- Hemarthrosis
- Cellulitis
- Septic joint
- Contracture
HO: ONSET OF DX ABNORMALITIES

TIME POST-INJURY

2-3 WEEKS:
Lab +/- bone scan abnormalities

1-4 MONTHS:
Clinical presentation (peak 2 months)

AT CLINICAL PRESENTATION:
Alk phos, bone scan, and x-rays are typically all abnormal

↑ ESR
? Early alk phos
1st, 2nd bone scan phases

3.5-6 weeks: alkaline phosphatase
4-5 wks: Fever, swelling
5-6 wks: ultrasound, bone scan 3rd phase
7 weeks: plain radiographs

Alkaline Phosphatase and HO

- Elevated acutely in all patients with clinically significant HO
- ↓ specificity
- Peak value at about 10 weeks
- Level may not correlate with severity or maturity of HO
- Other markers: CRP, ESR, CPK

Diagnosis of HO in SCI

- Triple Phase Bone Scan = gold standard
- Radiographs
  - No visible calcification for 7-10 days after bone scan becomes positive
  - Earliest findings can be seen 1 month post-injury
  - May have no visible calcifications at onset of symptom
- Other
  - Ultrasound: “zone phenomena”
  - MRI: hyperintense T2 early; contrast enhancement and muscle swelling
Triple-Phase Bone Scan

- Increased vascularity at 17 days
- Bone uptake at 24 days
- Best available indicator of maturity

Potential Complications: HO

- ROM Loss
  - Prevents sitting
  - Unequal seated pressure distribution
  - Impact on self-care ability, UE use
- Pressure ulcer
- Peripheral nerve entrapment
- DVT

Treatment of HO in SCI

- NSAIDs: For early osteoid stage
- ROM
- Etidronate
  - Chemiabsorbs to Ca hydroxyapatite crystals
  - NO effect on osteoid development, inhibits matrix mineralization
- XRT: not typically primary treatment but effective

Surgical Treatment for HO

<table>
<thead>
<tr>
<th>Timing</th>
<th>Outcomes</th>
<th>Considerations</th>
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<tbody>
<tr>
<td>• HO should be &quot;mature&quot; (usually 6-18 months) • Severe functional limitation (especially wheelchair seating)</td>
<td>• Most patients gain enough range to sit • Most have some recurrence</td>
<td>• ??XRT • ?? Adjuvant etidronate + NSAIDs • High complication rate! • excess bleeding • Infection</td>
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</table>
**PAIN & SCI**

Prevalence: 63-91% (Donnelly 2005)

- Higher rate: GSW or lower level injury
- Moderate-Severe: 25-60%
- Severe, disabling in 20-30% (Bryce et al 2001)
- MSCIS data (Cardenas 2005):
  - % with pain: 81% at 1 year post-SCI; 83% at 25 years
  - Pain interfering with activities: 70% year 1; 51% at 25 years

Most common sources:

- Neuropathic: central, radicular
- Musculoskeletal: carpal tunnel, shoulder
- Less common: visceral

**PAIN & SCI**

- Taxonomy
  - ISCI-BPDS
  - IASP
    - Nociceptive: MSK, visceral, skin
    - Neuropathic: Above-, at-, below-level pain
  - Cardenas (Neurologic vs. MSK)
    - Neuro: SCI pain, transition zone, radicular, visceral
    - MSK: mechanical spine, overuse
  - Bryce-Ragnarsson classification system

- Survey for treatable cause
- Awaiting a clinical practice guideline

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**Treatment of Neuropathic Pain in SCI: Pharmacologic**

- Anticonvulsants: Gabapentin, pregabalin (more on this shortly)
- Antidepressants: Amitriptyline — mixed efficacy in SCI pain, side-effects
- Opioids: IT MS and clonidine: combo effective?
- IV MS reduced evoked allodynia but not spontaneous pain
- Alfentanil (µ receptor): decreased spontaneous AND evoked pain
- NMDA receptor antagonists: Ketamine
- Local anesthetics (Na channel blockers): TR or IV lidocaine; but mexiletine not effective

**Treatment of Neuropathic Pain in SCI: Non-pharmacologic**

- Modalities
- biofeedback/relaxation
- CBT
- Exercise
- Hypnosis
- PT
- TENS
- Spinal Cord Stimulation, DREZ ablation, electrical stimulation
Pain & SCI: Evidence-Based Treatment

Siddall 2006: proposed treatment algorithm

Teasell 2010: Meta-Analysis

- Strongest evidence:
  - gabapentin and pregabalin
  - Subarachnoid lidocaine, IV ketamine, IV morphine (short-term benefit, not practical for home use)
- Limited evidence or lack of evidence
  - TCAD – except for neuropathic pain in setting of depression
  - Tramadol
  - Opioids – conflicting conclusions re: efficacy for neuropathic pain

Guy 2014: Anticonvulsants for neuropathic pain post-SCI

- Gabapentinoids – large effect in 4 of 6 studies
- Lamotrigine – effective with incomplete SCI
- Valproate and Levetiracetam – ineffective
- Carbamezepine – effective for moderate to intense pain (?)SCI studies

Late Neurologic Complications of SCI

Post-traumatic cyst (39-59%): focal intramedullary cystic degeneration

Post-traumatic syringomyelia (3-4%): > 2 segments; rare early dx

- Signs/symptoms – clinical surveillance recommended
  - Loss of temperature/pain sensation, hyperhidrosis, change in tone, new weakness or change in bowel/bladder control or respiratory function, increased pain, new AD, Charcot joint
- Diagnosis: MRI
- Treatment:
  - Monitoring for expansion – clinical, radiographic (distinguish from myelomalacia)
  - Minimize valsala and Trendelenberg position
  - When to consider surgery? Long-term outcomes of

Myelomalacia

Motor Control Loop
Neuroscience: Exploring the Brain, 2nd ed.

Spasticity Overview
Treatment in SCI
Spasticity

Definition (Lance)

- A motor disorder characterized by velocity-dependent increase in tonic stretch reflexes/muscle tone with exaggerated deep tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the UMN syndrome.

Presentation

- Resistance to stretch: velocity (speed)-dependent
- Abnormal movements
- Exaggerated reflexes
- Intermittent or sustained involuntary muscle movements (e.g., clonus)
- Flexor or extensor spams or posturing

Prevalence in SCI:

- 65%-78% of persons with SCI
- Problematic in 28%-43%
- Medications needed in 43%-49%

UMN SYNDROME:

Positive and Negative Components

<table>
<thead>
<tr>
<th>POSITIVE</th>
<th>NEGATIVE</th>
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<tbody>
<tr>
<td>Overactivity or presence of co-contraction</td>
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<tr>
<td>Spasticity</td>
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<tr>
<td>Athetosis</td>
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<tr>
<td>Hyperreflexia (DTR, autonomic, cutaneous)</td>
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<tr>
<td>Release of primitive reflexes</td>
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<tr>
<td>Dystonia</td>
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<tr>
<td>Underactivity/absence of motor function</td>
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<tr>
<td>Weakness</td>
<td></td>
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<tr>
<td>Paralysis</td>
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<tr>
<td>Fine motor loss</td>
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<tr>
<td>Fatigue</td>
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<tr>
<td>More important factors affecting patient function</td>
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Spasticity: Ways to Evaluate

For Your Reference/Review

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<tr>
<th>Subjective assessment</th>
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<tr>
<td>Surveys</td>
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<tr>
<td>Penn Spasm Frequency Score</td>
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“Objective” assessment

- Clinical/Qualitative
- Modified Ashworth Scale
- Tendon Tap
- SCATS, SCI-SET
- Pendulum Test
- Electrophysiologic/Quantitative

Functional assessment

- Goal (walking) analysis
- Transfers

PENN SPASM FREQUENCY SCORE (PSFS)

- 0 No spasms
- 1 Mild spasms induced by stimulation
- 2 Infrequent spasms occurring 1x/hour
- 3 Spasms occurring >1x/hour
- 4 Spasms occurring >10X/hour

MODIFIED PSFS

- 1 Mild
- 2 Moderate
- 3 Severe

SPASTICITY EVALUATION: MODIFIED ASHWORTH SCORE

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
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<tbody>
<tr>
<td>0</td>
<td>No increase in muscle tone</td>
</tr>
<tr>
<td>1</td>
<td>Slight increase in muscle tone, followed by minimal resistance throughout the remainder of the range of motion</td>
</tr>
<tr>
<td>1+</td>
<td>Slight increase in muscle tone, followed by resistance throughout the remainder of the range of motion</td>
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<tr>
<td>2</td>
<td>More marked increase in tone, through most of the range of motion but joint ends moved</td>
</tr>
<tr>
<td>3</td>
<td>Considerable increase in muscle tone, passive movement is difficult</td>
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<tr>
<td>4</td>
<td>Affected part is rigid in flexion or extension</td>
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Spasticity Management Model

- Ablative surgery (non-reversible)
- Intrathecal medications (reversible)
- Oral medications – systemic tx
- Blocks – focal pharmacotherapy
- Modalities
- Stretching, casting, bracing
- Eliminate nociception

More likely: simultaneously using multiple approaches

FOCAL TREATMENT OF SPASTICITY

- Effectively creating a LMN syndrome to treat UMN symptoms
- Preferred to control regional spasticity and avoid systemic adverse drug effects
- Short duration (< ½ day):
  - Local anesthetic: lidocaine and bupivicaine
- Longer duration (2-5 months):
  - Chemical neurolysis: destruction of nerve
    - phenol 3.7%
    - ETOH 45-100%
  - Motor point block: effort to block nerve trunk after sensory branches or to block a particular muscle
  - Neurotoxin chemodenervation
    - Botulinum neurotoxin (BoNT)

BOTULINUM NEUROTOXIN DENERSVATION

- What is Botulinum Toxin?
  - Proteins synthesized by Clostridium botulinum, responsible for Botulism
  - At least 7 antigenically distinct serotypes with similar mechanisms of action; different receptors
  - Inhibits Ach release from presynaptic terminal of neuromuscular junction
- Goal: reduce force of contraction of overactive muscle
- Onset 2-6 days, peak 1-4 weeks, duration approximately 3 months (2-6 months)

BOTULINUM TOXIN MECHANISM OF ACTION

For Your Reference/Review
**BOTULINUM TOXIN INJECTIONS**

**Considerations – For Your Review/Reference**

- **Concerns**
  - excessive weakness
  - spread of medication: 2009 FDA mandated new label warning and “risk mitigation strategy” for all BoNT:
    - weakness, hoarseness, dysarthria, loss of bladder control, breathing difficulties, dysphagia, double/blurred vision, drooping eyelids
  - dosage limitations: prioritize use!
  - drug cost and insurance coverage issues
  - Antigenicity: avoid boosters
  - Note that there is no clear dose equivalence formulas when converting from one serotype to the other

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**BOTULINUM TOXIN FORMULATIONS**

- FDA-approved indications
  - Note: doses are NOT equivalent across preparations

**Botulinum Toxin A:**
- onabotulinumtoxinA – upper limb spasticity in adults, cervical dystonia, strabismus and blepharospasm, wrinkled face, spasmodic torticollis, hemifacial spasm, preventive tx of chronic migraines
- abobotulinumtoxinA – glabellar frown lines, cervical dystonia
- incobotulinumtoxinA – cervical dystonia, blepharospasm

**Botulinum Toxin B**
- rimabotulinumtoxinB - Cervical dystonia

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**ORAL MEDICATIONS**

**Baclofen**

- **Mechanism and site of action:**
  - pre- and postsynaptic GABA B receptors
  - dorsal spinal cord and brainstem
  - depresses monosynaptic and polysynaptic reflexes that facilitate spasticity

- **Pharmacokinetics:**
  - Peak: within 2 hours
  - t1/2 = 3.5 hours (2-6 hours)

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**ORAL MEDICATIONS:**

**Alpha-2 Adrenergic Agonist**

**Tizanidine**

- Structurally similar to clonidine, but only 1/10th to 1/50th cardiovascular potency
- Site of action: Spinal and supraspinal → spasticity of spinal and cerebral origin
- Mechanism: Reduces excitatory AA (glu, asp), substance P release from presynaptic spinal interneurons; facilitates inhibitory AA (glycine)
- Depresses polysynaptic reflexes (not monosynaptic)
- **Pharmacokinetics**
  - Peak: 1 hour
  - t1/2 = 2-4 hours
ORAL MEDICATIONS: Dantrolene

- Hydantoin derivative; only FDA-approved peripherally acting antispasticity agent
- Mechanism:
  - Blocks Ca release from sarcoplasmic reticulum
  - Decreases strength of contraction
- Pharmacokinetics:
  - t1/2 = 15 hours po, 12 hours IV
  - Peak 3-6 hours
  - Active metabolite peaks 4-8 hour

ORAL MEDICATIONS BENZODIAZEPINES

- DIAZEPAM
  - Mechanism: centrally-acting, do not directly bind to GABA receptors, but induce release of GABA from GABA A neurons
  - Antiepileptic, hypnotic, anxiolytic, antispasticity properties
  - Central site of action: brainstem reticular formation > spinal polysynaptic pathway
  - Increases presynaptic inhibition of afferents at spinal cord level
  - Depresses monosynaptic and polysynaptic transmission

INTRATHECAL BACLOFEN PUMP

- Indication: Approved by FDA to manage severe spasticity resulting from spinal cord or cerebral (brain) disease or injury
- Delivers precise, programmable dose of liquid baclofen directly into the intrathecal space
- Intrathecal delivery 100x more potent than po (Dralle 1985); can minimize systemic adverse side-effects

Medical Complications in SCI: Summary

- Not well recognized or understood by physicians outside of PM&R
- PM&R physicians need to provide expertise
- Identifying correct diagnosis
- Recommending treatments that take into account the physiologic alterations after SCI

- Bladder Management for Adults with SCI
- Preservation of the Upper Limb following SCI
- Respiratory Management Following SCI
- Depression Following SCI*
- Neurogenic Bowel Management in Adults with SCI*
- Outcomes Following Traumatic SCI*
- Acute Management of Autonomic Dysreflexia, 2nd Edition*
- Pressure Ulcer Prevention and Treatment Following SCI*
- Prevention of Thromboembolism in SCI, 2nd Edition
- Early Acute Management in Adults with Spinal Cord Injury
- Sexuality and Reproductive Health in Adults with SCI
* Consumer Guide available

Practice Question #1
- Which of the following conditions comprises the leading cause of death for individuals with paraplegia?
  - A) cancer
  - B) heart disease
  - C) respiratory disease
  - D) sepsis

Practice Question #2
- During an episode of autonomic dysreflexia, which of the following is expected to be present below the level of SCI?
  - A) piloerection
  - B) sphincter relaxation
  - C) skin flushing
  - D) extremity warming
Practice Question #2

- During an episode of autonomic dysreflexia, which of the following is expected to be present below the level of SCI?
- A) piloerection
- B) sphincter relaxation
- C) skin flushing
- D) extremity warming

Practice Question #3

- Characteristic features of bone loss after SCI include which of the following?
- A) Predilection for cortical rather than trabecular bone
- B) Onset is 12 months after injury
- C) Occurs almost exclusively below the level of SCI
- D) The greatest loss and highest fracture risk is at the hips

Practice Question #4

- Which of the following lung volumes is unchanged or increased after SCI?
- A) Residual volume
- B) Total lung capacity
- C) Vital capacity
- D) FEV₁
Practice Question #4

- Which of the following lung volumes is unchanged or increased after SCI?
  - A) Residual volume
  - B) Total lung capacity
  - C) Vital capacity
  - D) FEV₁

Practice Question #5

- What would you advise a 50 y/o M with CS AIS A tetraplegia regarding his risk for heart disease as a result of his SCI versus the general population?
  - A) His SCI is an independent risk factor leading to greater cardiovascular morbidity.
  - B) Arm ergometry is the most appropriate exercise tolerance test.
  - C) He is likely to experience typical cardiac pain.
  - D) His HDL levels are more likely to be <35.