In USA: 30–60 new injuries per million pop. /year
- Incidence (new cases): 10,000 new cases of SCI/year
- Prevalence (total # of existing cases): 200,000–250,000 cases
Gender: 82% male vs. 18% female
Age: Average age at injury: 31.7 years of age
- Patients injured after 1990 had an average age at time of injury of 34.8 years
  56% of SCIs occur among persons in the 16–30 year age group
  Children 15 years of age or younger account for only 4.5% of SCI cases
  Persons older than 60 years of age account for 10% of SCI cases
- Falls are the most common cause of SCI in the elderly
- Motor vehicle accidents (MVAs) are the second most common cause of SCIs in the elderly
Causes: MVAs: 44%
  Violence (most are gunshot): 24%
  Falls: 22%
  Sports (most are diving): 8%
  Other: 2%
Time of Injury: Season: Summer (highest incidence in July)
  Day: Weekends (usually Saturday)
  Time: Night
Characteristics of Injury: Tetraplegia: C5 is most common level of injury
  Paraplegia: T12 is most common level of injury
Type of injury: Tetraplegia: 51.9%
  Paraplegia: 46.27%
  Incomplete tetraplegia: 29.6%
  Complete paraplegia: 28.1%
  Incomplete paraplegia: 21.5%
  Complete tetraplegia: 18.5%
  Complete or substantial recovery by time of discharge: 0.7%
  Persons for whom this information is not available: 0.7%
Demographics:
There is a close association between risk of SCI and a number of indications of social class, all of which have profound implications for rehabilitation:
- SCI patients have fewer years of education than their uninjured counterparts
- SCI patients are more likely to be unemployed than non-SCI pts.
- SCI patients are more likely to be single (i.e. never married, separated, divorced)
   Note: Postinjury marriages (injured and then married) survive better than preinjury marriages (injured after marriage)

ANATOMY

The vertebral column (Figure 7–1) consists of:
7    cervical
12   thoracic
5    lumbar
5    sacral
4    coccyx

Spinal Cord:
Located in upper two-thirds of the vertebral column
The terminal portion of the cord is the conus medullaris, which becomes cauda equina (horse’s tail) at approximately the L2 vertebrae

The spinal cord has an inner core of gray matter, surrounded by white matter. The white matter consists of nerve fibers, neuroglia, and blood vessels. The nerve fibers form spinal tracts, which are divided into ascending, descending, and intersegmental tracts. The location and function of various tracts are shown below (Figure 7–2).

### LONG TRACTS IN THE SPINAL CORD

<table>
<thead>
<tr>
<th>Key</th>
<th>Tract</th>
<th>Location</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Fasciculus gracile: dorsal columns (posterior)</td>
<td>Medial dorsal column</td>
<td>Proprioception from the leg Light touch Vibration</td>
</tr>
<tr>
<td></td>
<td>Same as above</td>
<td>Lateral dorsal column</td>
<td>Proprioception from the arm Light touch Vibration</td>
</tr>
<tr>
<td></td>
<td>Spinocerebellar</td>
<td>Superficial lateral column</td>
<td>Muscular position and tone, unconscious proprioception</td>
</tr>
<tr>
<td></td>
<td>Lateral spinothalamic</td>
<td>Ventrolateral column</td>
<td>Pain and thermal sensation</td>
</tr>
<tr>
<td></td>
<td>Ventral spinothalamic</td>
<td>Ventral column</td>
<td>Tactile sensation of crude touch and pressure</td>
</tr>
<tr>
<td></td>
<td>Lateral corticospinal tract (pyramidal)</td>
<td>Deep lateral column</td>
<td>Motor: Medial (cervical)-Lateral (sacral) C S (motor neuron distribution)</td>
</tr>
<tr>
<td></td>
<td>Anterior corticospinal tract</td>
<td>Medial ventral column</td>
<td>Motor: Neck and trunk movements</td>
</tr>
</tbody>
</table>

![FIGURE 7–2. Transverse section of the spinal cord (use key above for long tracts location and function).](image-url)
MAJOR ASCENDING AND DESCENDING PATHWAYS IN THE SPINAL CORD
(A SCHEMATIC VIEW)

Note where tracts cross in relation to brain stem (Figure 7–3)
- Corticospinal tract crosses at brain stem to contralateral side, then descends
- Spinocerebellar tract does not cross; remains ipsilateral as it descends
- Spinothalamic tract crosses low to contralateral side, then ascends
- Dorsal columns ascends, crosses at brain stem to contralateral side

**Descending Pathways**
- The corticospinal tract (motor pathways) extends from the motor area of the cerebral cortex down through the brainstem, crossing over at the junction between the spinal cord and brainstem. The corticospinal pathway synapses in the anterior horn (motor grey matter) of the spinal cord just prior to leaving the cord. This is important for motor neurons above the level of this synapse [connecting anterior horn and anterior horn are termed upper motor neurons (UMN) whereas those below this level (peripheral neurons) are termed lower motor neurons (LMN)]. Cerebral lesions result in contralateral defects in general.
- The spinocerebellar tract (unconscious proprioception) remains ipsilateral. Cerebral lesions produce ipsilateral malfunctioning.

**Ascending Pathways**
- Spinothalamic tract (pain and temperature) enters the spinal cord, crosses over to the opposite half of the cord almost immediately (actually within 1–2 spinal cord vertebral segments), ascends to the thalamus on the opposite side, and then moves on the cerebral cortex. A lesion of the spinothalamic tract will result in loss of pain-temperature sensation contralaterally below the level of the lesion.

**FIGURE 7–3.** A Schematic View: The major long tracts in the spinal cord (ascending and descending arrows depict direction).
• Dorsal columns (proprioception vibration) initially remains on the same side of the spinal cord that it enters, crossing over at the junction between the spinal cord and brainstem. The synaptic areas just prior to this crossing are nucleus cuneatus and nucleus gracilis. Their corresponding spinal cord pathways are termed fasciculus gracilis and fasciculus cuneatus. Fasciculus gracilis and fasciculus cuneatus are collectively termed posterior (dorsal) columns. A lesion of the posterior columns results in the loss of proprioception and vibration ipsilaterally below the level of the lesion.

**Blood Supply of the Spinal Cord (Figure 7–4)**

- Posterior Spinal Arteries arise directly or indirectly from the vertebral arteries, run inferiorly along the sides of the spinal cord, and provide blood to the posterior third of the spinal cord.
- Anterior Spinal Arteries arise from the vertebral arteries, uniting to form a single artery, which runs within the anterior median fissure. They supply blood flow to the anterior two-thirds of the spinal cord.
- Radicular Arteries reinforce the posterior and anterior spinal arteries. These are branches of local arteries (deep cervical, intercostal, and lumbar arteries). They enter the vertebral canal through the intervertebral foramina.
- The artery of Adamkiewicz or the arteria radicularis magna is the name given to the lumbar radicular artery. It is larger and arises from an intersegmental branch of the descending aorta in the lower thoracic or upper lumbar vertebral levels (between T10 and L3) and anastomoses with the anterior spinal artery in the lower thoracic region. The lower thoracic region is referred to as the watershed area. It is the major source of blood to the lower anterior two-thirds of the spinal cord.
- The Veins of the Spinal Cord drain mainly into the internal venous plexus.

---

**FIGURE 7–4.** Arterial and venous supply to the spinal cord. (transverse section).
SPINAL PATHOLOGY

TYPES OF CERVICAL SPINAL CORD INJURY: PATHOLOGY

Compression Fractures—slight flexion of the neck with axial loading (Figure 7–5) (Bohlmann, 1979)

- C5 is the most common compression fracture of the cervical spine
- Force ruptures the plates of the vertebra, and shatters the body. Wedge shaped appearing vertebra on X-ray.
- May involve injury to the nerve root and/or cord itself
- Fragments may project into spinal canal
- Stable ligaments remain intact

Flexion-Rotation Injuries

Unilateral facet joint dislocations (Figure 7–6)

- Vertebral body < 50% displaced on X-ray
- Unstable (if the posterior ligament is disrupted)
- Narrowing of the spinal canal and neural foramen
- C5–C6 most common level
- Also note that flexion and rotation injuries may disrupt the intervertebral disc, facet joints, and interspinous ligaments with little or no fracture of the vertebrae
- Approximately 75% have no neurological involvement because the narrowing is not sufficient to affect the spinal cord
- If injury results, it is likely an incomplete injury


FIGURE 7–6. Unilateral facet joint dislocation. A: lateral view. Note: there is less than 50% anterior dislocation of the vertebral body. B: posterior view.
Flexion Injuries

Bilateral facet joint dislocations (Figure 7–7)

- Vertebral body > 50% displaced on X-ray
- Both facets dislocate
- Unstable; secondary to tearing of the ligaments
- Most common level is C5–C6 because of increased movement in this area
- More than 50% anterior dislocation of the vertebral body causes significant narrowing of the spinal canal
- Spinal cord is greatly compromised
- 85% suffer neurologic injuries
- Likely to be a complete injury

Hyperextension Injuries (Figure 7–8)

- Can be caused by acceleration-deceleration injuries such as MVA
- Soft tissue injury may not be seen in radiologic studies
- Stable; anterior longitudinal ligament is disrupted
- Spinal cord may be involved
- Can be seen in hyperextension of the C-spine and appear as Central Cord syndrome. This most commonly occurs in older persons with degenerative changes in the neck.
- Clinically: UE motor more involved than LE. Bowel, bladder, and sexual dysfunction occur to various degrees.
- C4–C5 is the most common level
SPINAL CORD INJURIES

TABLE 7-1. Spinal Cord and Pathology Associated with Mechanism of Injury

<table>
<thead>
<tr>
<th>Types of Spinal Injury</th>
<th>Stability</th>
<th>Possible Resultant Injury</th>
<th>Most Common Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Compression Axial loading (i.e., diving)</td>
<td>Stable Ligaments remain intact</td>
<td>Crush fracture w/ fragmentation of vertebral body and projection of bony spicules into canal</td>
<td>C5</td>
</tr>
<tr>
<td>Flexion Rotation Injury Unilateral dislocation</td>
<td>Unstable (if posterior ligament disrupted) Vertebral body &lt;50% displaced on Xray</td>
<td>Spinal cord not severely compromised; likely to be incomplete injury</td>
<td>C5–C6</td>
</tr>
<tr>
<td>Flexion Bilateral dislocation</td>
<td>Unstable (if post ligament disrupted) Vertebral body &lt;50% displaced on X-ray</td>
<td>Ant. dislocation of C-spine with compression of spinal cord; spinal cord greatly compromised; likely to be complete injury</td>
<td>C5–C6</td>
</tr>
<tr>
<td>Hyper Extension Injury Central Cord syndrome</td>
<td>Stable; Anterior longitudinal ligament may be disrupted</td>
<td>Hyperextension of C-spine clinically: UE weaker than LE; likely to be incomplete injury</td>
<td>C4 C5</td>
</tr>
</tbody>
</table>

Spinal Compression 2° to metastatic disease

Majority of tumors affecting the SC are metastatic in origin
95% are extradural in origin involving the vertebral bodies
Results in compression of the anterior aspect of the spinal cord
70% of spinal mets occur in the thoracic spine

CERVICAL BRACING (also see Prosthetics & Orthotics Chapter)

<table>
<thead>
<tr>
<th>Removable Cervical Orthoses:</th>
<th>Nonremovable Cervical Orthoses:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Least restrictive: Soft collar</td>
<td>Halo is the most restrictive cervical orthosis of all cervical orthoses.</td>
</tr>
<tr>
<td>Philadelphia collar</td>
<td></td>
</tr>
<tr>
<td>SOMI brace</td>
<td></td>
</tr>
<tr>
<td>Four poster</td>
<td></td>
</tr>
<tr>
<td>Most restrictive: Minerva brace</td>
<td></td>
</tr>
</tbody>
</table>

Cervical Bracing

The Minerva brace is the most restrictive removable brace, followed by the four poster, then SOMI.
Philly collar is less restrictive, and soft collar is the least restrictive of the listed braces.
Halo is the most restrictive, but not removable.
See P & O section for more in-depth discussion of spinal bracing.

COMPLETE vs. INCOMPLETE LESIONS

Complete lesions are most commonly secondary to the following
1. Bilateral cervical facet dislocations
2. Thoracolumbar flexion-rotation injuries
3. Transcanal gunshot wounds

Incomplete injuries are most commonly secondary to the following
1. Cervical spondylosis—falls
2. Unilateral facet joint dislocations
3. Noncanal penetrating gunshot/stab injuries
OTHER FRACTURES OF THE SPINE

Cervical Region:

Jefferson Fracture: (Figure 7–9)
- Burst fracture of the C1 ring
- Mechanism: axial loading causing fractures of anterior and posterior parts of the atlas
- If the patient survives, there are usually no neurologic findings with treatment

Hangman Fracture: (Figure 7–10)
- C2 burst fracture
- Body is separated from its posterior element, decompresses cord (No SCI)
- If the patient survives, there are only transient neurologic findings with appropriate Tx

FIGURE 7–9. Jefferson fracture (Superior view).

Odontoid Fracture (Figure 7–11, 7–12)

- C2 odontoid is fractured off at its base
- Commonly results from trauma
- Patient usually survives
- Usually only transient neurologic signs with appropriate Tx

Thoraco Lumbar Region

Chance Fracture (Figure 7–13)

- Most commonly seen in patients wearing lap seat belts
- Transverse fracture of lumbar spine through body and pedicles, posterior elements
- Chance fractures are seldom associated with neurologic compromise unless a significant amount of translation is noted on the lateral radiographs


**FIGURE 7–12.**

Type 1: Oblique fracture through upper part of the dens; treatment is with rigid cervical orthosis such as Philadelphia collar. Type 2: Fracture at the junction of the odontoid process and the vertebral body; if displacement is less than 5 mm and angulated less than 15 degrees, then halo is appropriate; otherwise operative treatment with C1 to C2 fusion or screw fixation. Type 3: Fracture extends down through vertebral body; treatment is with halo. (From Nesathurai S. The Rehabilitation of People With Spinal Cord Injury: A House Officer’s Guide. © Boston Medical Center for the New England Regional Spinal Cord Injury Center. Boston, MA: Arbuckle Academic Publishers, with permission).
Vertebral Body Compression Fracture (anterior wedge fracture) (Figure 7–14)

- Mechanism: most common injuries caused by axial compression with or without flexion: vertebrae body height is reduced—may cause thoracic kyphosis (Dowager hump)
- Spontaneous vertebral compression fractures are stable injuries—ligaments remain intact

SCIWORA – SPINAL CORD INJURY WITHOUT RADIOLOGIC ABNORMALITY

This condition is commonly seen in young children and older adults

Children

- Mechanism of injury in children include
  - Traction in a breech delivery
  - Violent hyperextension or flexion
- Predisposing factors in children include
  - Large head-to-neck size ratio
  - Elasticity of the fibrocartilaginous spine
  - Horizontal orientation of the planes of the cervical facet joints

Older Adults

- Mechanism of injury in the elderly includes
  - A fall forward and a blow on the head causing an acute central cord syndrome; the ligamentum flavum may bulge forward into the central canal and narrow the sagittal diameter as much as 50%
- Note: Delayed onset or paralysis may occur due to vascular mechanism or edema accumulation at the injury site, although this is uncommon
- Essential history in a person with head or neck pain includes identifying any neurological symptoms
- Flexion/Extension films should be done cautiously only after static neck films have been cleared by a radiologist and only if there are no neurologic symptoms or severe pain present
- Empiric use of a 24-hour cervical collar with repeat films at resolution of cervical spasm is warranted
IMPORTANT DEFINITIONS

Types of Injuries

Tetraplegia
- Replaces quadriplegia
- Impairment or loss of motor and/or sensory function in the cervical segments of SC due to damage of neural elements within spinal canal
- Results in impairment of function in arms, trunk, legs, pelvic organs
- Does not include brachial plexus lesions or injury to peripheral nerves outside neural canal

Paraplegia
- Impairment or loss of motor and/or sensory function in thoracic, lumbar, or sacral segments of SC
- Trunk, legs, pelvic organs may be involved, arm function spared
- Refers to cauda equina and conus medullaris injuries, but not to lumbosacral plexus lesions or injury to peripheral nerves outside the neural canal

Other Definitions

Dermatome
Area of skin innervated by the sensory axons within each segmental nerve (root)

Myotome
Collection of muscle fibers innervated by the motor axons within each segmental nerve (root)

UPPER MOTOR NEURON INJURY vs. LOWER MOTOR NEURON INJURY

<table>
<thead>
<tr>
<th>Upper Motor Neuron Injury</th>
<th>Lower Motor Neuron Injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>Supply:</td>
<td>Supply:</td>
</tr>
<tr>
<td>Begins in the prefrontal motor cortex, travels through the internal capsule and brainstem, and projects into the spinal cord</td>
<td>Begins with the anterior horn cells of the spinal cord and includes the peripheral nerves</td>
</tr>
<tr>
<td>Upper Motor Neuron Findings</td>
<td>Lower Motor Neuron Findings</td>
</tr>
<tr>
<td>Increased muscle stretch reflexes</td>
<td>Hyporeflexia</td>
</tr>
<tr>
<td>Babinski response</td>
<td>Flaccid weakness</td>
</tr>
<tr>
<td>Detrusor sphincter dyssynergia (depending on level of lesions)</td>
<td>Significant muscle wasting</td>
</tr>
</tbody>
</table>

Note: Lesions of the upper lumbar vertebral bodies can present with a mixture of upper and lower neuron findings

NEUROLOGIC LEVEL, SENSORY LEVEL, AND MOTOR LEVEL OF INJURY:
(Hoppenfeld, 1977)

Lesions are classified according to a neurologic, motor, and sensory level of injury. They are further divided into complete and incomplete lesions.
1. **Sensory level of injury**

- Most caudal segment of the SC with normal (2/2) sensory function on both sides of the body for pinprick, and light touch
- For the sensory examination there are 28 key sensory dermatomes, each tested separately for light touch (with a cotton tip applicator) and pinprick (with a safety pin)

<table>
<thead>
<tr>
<th>Scores</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Absent</td>
</tr>
<tr>
<td>1</td>
<td>Impaired</td>
</tr>
<tr>
<td>2</td>
<td>Normal</td>
</tr>
</tbody>
</table>

The face is used as the normal control point.

For pinprick testing: The patient must be able to differentiate the sharp and dull edge of a safety pin.

<table>
<thead>
<tr>
<th>Scores</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Not able to differentiate between the sharp and dull edge</td>
</tr>
<tr>
<td>1</td>
<td>The pin is not felt as sharp as on the face, but able to differentiate sharp from dull</td>
</tr>
<tr>
<td>2</td>
<td>Pin is felt as sharp as on the face</td>
</tr>
</tbody>
</table>

For light touch, a cotton tip applicator is compared to the face sensation

<table>
<thead>
<tr>
<th>Scores</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>Normal—same as on face</td>
</tr>
<tr>
<td>1</td>
<td>Impaired—less than on the face</td>
</tr>
<tr>
<td>0</td>
<td>Absent</td>
</tr>
</tbody>
</table>

It is very important to test the S4/S5 dermatome for light touch and pinprick

2. **Motor level of injury**

- Most caudal key muscle group that is graded three-fifths or greater with the segments above graded five-fifths in strength.
- A possible score of 100 can be obtained when adding the muscle scores of the key muscle groups (25 points per extremity).

There are 10 key myotomes on the left and right side of the body:

<table>
<thead>
<tr>
<th>Myotome</th>
<th>Index Muscle</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>C5</td>
<td>Biceps brachialis</td>
<td>Elbow flexors</td>
</tr>
<tr>
<td>C6</td>
<td>Extensor carpi radialis</td>
<td>Wrist extensors</td>
</tr>
<tr>
<td>C7</td>
<td>Triceps</td>
<td>Elbow extensors</td>
</tr>
<tr>
<td>C8</td>
<td>Flexor digitorum profundus</td>
<td>Finger flexors (FDP of middle finger)</td>
</tr>
<tr>
<td>T1</td>
<td>Abductor digitii minimi</td>
<td>Small finger abductor</td>
</tr>
<tr>
<td>L2</td>
<td>Iliopsoas</td>
<td>Hip flexors</td>
</tr>
<tr>
<td>L3</td>
<td>Quadriceps</td>
<td>Knee extensors</td>
</tr>
<tr>
<td>L4</td>
<td>Tibialis anterior</td>
<td>Ankle dorsiflexors</td>
</tr>
<tr>
<td>L5</td>
<td>Extensor hallucis longus</td>
<td>Long toe extensors</td>
</tr>
<tr>
<td>S1</td>
<td>Gastrocnemius</td>
<td>Ankle plantarflexors</td>
</tr>
</tbody>
</table>

**Manual Muscle Testing Grading System**

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No movement</td>
</tr>
<tr>
<td>1</td>
<td>Palpable movement or visible contraction</td>
</tr>
<tr>
<td>2</td>
<td>Active movement through full range of motion with gravity eliminated</td>
</tr>
<tr>
<td>3</td>
<td>Active movement through full range of motion against gravity</td>
</tr>
<tr>
<td>4</td>
<td>Active movement against moderate resistance through full range of motion</td>
</tr>
<tr>
<td>5</td>
<td>Normal strength based on age, sex, and body habitus</td>
</tr>
</tbody>
</table>
3. Neurologic level of injury

- Most caudal segment of the spinal cord with both normal sensory and motor function on both sides of the body, determined by the sensory and motor levels
- Since the level may be different from side to side, it is recommended to record each side separately

4. Skeletal level of injury

- Level where the greatest vertebral damage is noted by radiographic evaluation

COMPLETE VS. INCOMPLETE LESIONS

Complete injury (Waters 1991)

- Absence of sensory and motor function in the lowest sacral segment
- The term Zone of Partial Preservation is only used with complete lesions
- Refers to the dermatomes and myotomes caudal to the neurological level of injury that remain partially innervated

Incomplete injury

- Partial preservation of sensory and/or motor functions below the neurological level, which includes the lowest sacral segment. Sacral sensation and motor function are assessed.
- Sacral sparing —voluntary anal sphincter contraction or sensory function (light touch, pinprick at the S4–S5 dermatome, or anal sensation on rectal examination) in the lowest sacral segments.
- Due to preservation of the periphery of the SC
- Indicates incomplete injury
- Sacral sparing indicates the possibility of SC recovery, with possible partial or complete return of motor power
- There is also the possibility of return of bowel and bladder function
- The concept of sacral sparing in the incomplete SCI is important because it represents at least partial structural continuity of the white matter long tracts (i.e., corticospinal and spinothalamic tracts). Sacral sparing is evidenced by perianal sensations (S4–S5 dermatome), and rectal motor function. Sacral sparing represents continued function of the lower sacral motor neurons in the conus medullaris and their connections via the spinal cord to the cerebral cortex.

ASIA IMPAIRMENT SCALE: CLASSIFIES COMPLETE AND INCOMPLETE INJURIES:

A = Complete: No motor or sensory function is preserved in the sacral segments
B = Incomplete: Sensory but not motor function is preserved below the neurological level and includes sacral segments
C = Incomplete: Motor function preserved below the neurological level; more than half the key muscles below the neurological level have a muscle grade less than 3
D = Incomplete: Motor function preserved below the neurological level; at least half the key muscles below the neurological level have a muscle grade of 3 or more
E = Normal: Motor and sensory function

Assigning an ASIA Level (Figure 7–15)

1. Examine 10 index muscles bilaterally
2. Examine 28 dermatomes for pinprick and light touch
3. Complete rectal exam to assess sensation and volitional sphincteric contraction
4. Determine left and right motor levels
5. Determine left and right sensory levels
6. Assign final motor and sensory levels
7. Determine neurological level, which is the most caudal segment with normal motor and sensory function
8. Categorize injury as complete or incomplete by ASIA impairment scale (A,B,C,D,E)
9. Calculate motor and sensory score
10. Determine zone of partial preservation if complete injury (“A” on impairment scale)

**CLINICAL EFFECTS OF SCI: DIVIDED INTO TWO STAGES**

1. Spinal Shock–Areflexia
2. Heightened Reflex Activity

**1. Stage of Spinal Shock**
- Reflex arc is not functioning
- Loss of motor function is accompanied by atonic paralysis of the bladder, bowel, gastric atony
- All the muscles below the level of the lesion become flaccid and hyporeflexic
- Loss of sensation below the level of the lesion
- Temporary loss or depression of all spinal reflex activity below the level of the lesion
- Autonomic function below the level of the lesion is also impaired
- Temporary loss of piloerection, sweating, vasomotor tone in the lower parts of the body
- Believed to be due to a sudden and abrupt interruption of descending excitatory influences

**Duration:** Lasts from 24 hours to 3 months after injury. Average is 3 weeks.

Minimal reflex activity is noted usually with the return of the bulbocavernosus reflex and the anal wink reflex.

**Bulbocavernosus reflex (male):**

**Figure 7–16**
- The bulbocavernosus reflex arc is a simple sensory-motor pathway that can function without using ascending or descending white-matter, long-tract axons.
- Usually the first reflex to return after spinal shock is over. If the level of the reflex arc is both physiologically and anatomically intact, the reflex will function in spite of complete spinal cord disruption at a higher level.
- Indicates that reflex innervation of bowel and bladder is intact
- Performed by squeezing the penis and noting stimulation of anal sphincter contraction
- At this time the bladder can be expected to contract on a reflex basis (although clinically this rarely occurs)
- Bowel will empty as a result of reflex induced by fecal bulb or rectal suppository stimulation
Perianal Sphincter Reflex (anal wink)
- Perianal stimulation causes contraction of the anal sphincter
- Indicates that reflex innervation of the bowel and bladder is intact

2. Stage of Increased Reflex Activity
- As the spine recovers from shock, the reflex arc functions without inhibitory or regulatory impulses from the brain, creating local spasticity and clonus
- Reflexes become stronger, and come to include additional and more proximal muscles
- Pattern of higher flexion is noted
- Dorsiflexion of the big toe (Babinski sign)
- Fanning of the toes
- Achilles reflex returns, then patellar
- Bladder starts to present contractions at irregular intervals with release of urine
- Reflex defecation

ASIA NEUROLOGICAL EXAMINATION TWO COMPONENTS—SENSORY AND MOTOR

Localizing Level of Injury and Asia Classification (Asia, 1996), (Mayard and Bracken, 1997)

ASIA Key Sensory Levels (Figure 7–17)
C2: Occipital protuberance
C3: Supraclavicular fossa
C4: Superior AC Joint
C5: Lateral side of the antecubital fossa
C6: Thumb (and index finger)
C7: Middle finger
C8: Little finger
T1: Medial ulnar side of antecubital epicondyle
T2: Apex of axilla
T3: Third intercostal space (IS)
T4: Nipple line – fourth IS
T5: Fifth intercostal space - fifth IS
T6: Xiphoid – sixth IS
T7: Seventh intercostal space – seventh IS
T8: Eighth intercostal space – eighth IS
T9: Midway between T8 and T10 – ninth IS
T10: Umbilicus – tenth IS
T11: Eleventh intercostal space – eleventh IS
T12: Ingual ligament at midpoint
L1: Half the distance between T12 and L2
L2: Midanterior thigh
L3: Medial fem condyle
L4: Medial malleolus
L5: Dorsum of foot at third MTP joint
S1: Lateral heel
S2: Popliteal fossa in the midline
S3: Ischial tuberosity
S4 and S5: Perianal area (taken as one level)

ASIA Key Motor Levels
C1–C4: Use sensory level and diaphragm to localize lowest neurological level
C5: Elbow flexors
C6: Wrist extensors
C7: Elbow extensors
C8: Finger flexors (FDP of middle finger)
T1: ABD digiti minimi (small finger abductor)
T2–L1: Use sensory level
L2: Hip flexors
L3: Knee extensors
L4: DF ankle dorsiflexors
L5: Long toe extensors
S1: Plantar flexors

Reflexes
S1S2: Gastrocnemius (ankle jerk)
L3L4: Quadriceps (knee jerk)
C5C6: Biceps, brachioradialis
C7C8: Triceps, finger flexors
L5: Medial hamstring
INCOMPLETE SPINAL CORD INJURY SYNDROMES

**Central cord syndrome** (Figure 7–18) This is the most common syndrome.

- Results from an injury involving the center of the spinal cord
- It is predominantly a white matter peripheral injury
- Intramedullary hemorrhage is not common
- It may occur at any age, but is more common in older patients
- Produces sacral sensory sparing, greater motor weakness in the upper limbs than the lower limbs. Anatomy of the corticospinal tracts is such that the cervical distribution is medial and sacral distribution is more lateral. Since the center of the SC is injured, upper extremities are more affected than lower extremities.
- Patients may also have bladder dysfunction, most commonly urinary retention
- Variations in sensory loss below the level of the lesion
Recovery: Lower extremities recover first and to a greater extent. This is followed by improvement in bladder function, then proximal upper extremity, and finally intrinsic hand function. (Roth et al., 1990)

Brown-Sequard Syndrome: (Figure 7–19, 7–20) Constitutes 2%-4% of all traumatic SCI

- Results from a lesion that causes spinal hemisection
- (Ipsilateral) focal injury to the spinal cord causes deficits distal to the site of the lesion. Because tracts cross at different locations, deficits affect different sides, i.e.
- Ipsilateral—motor and proprioception deficits
- Contralateral—pain and temperature deficits
- Associated with stabbing and gunshot wounds
- Patients have ipsilateral motor and proprioceptive loss, and contralateral loss of pain and temperature

Result

Ipsilateral:
Motor and proprioceptive deficits (right sided)

Contralateral:
Pain and temperature deficits (left sided)
Anterior Cord Syndrome: (Figure 7–21)

Caused by:
A lesion involving the anterior two thirds of the spinal cord preserving the posterior columns, such as:
- Anterior spinal artery lesions, direct injury to the anterior spinal cord, bone fragments or a retropulsed disc
- Polyarteritis nodosa, angioplasty, aortic and cardiac surgery, and embolism, can result in injury to the anterior two-thirds of the spinal cord (Ditunno, 1992)

FIGURE 7–20. Brown-Sequard syndrome lesion: depicts point of injury, i.e. right-sided gunshot or knife wound. Follow tracts distal from the point of injury. Result is ipsilateral motor and proprioceptive deficits (right-sided) contralateral pain and temperature deficits (left-sided).

FIGURE 7–21. Anterior cord syndrome. (transverse section of the spinal cord—refer to Figure 7–2 for anatomic landmarks).
Result:
Variable loss of motor function (corticospinal tract) and sensitivity to pain and temperature, pinprick sensation, (spinothalamic tract) with preservation of proprioception and light touch

Recovery:
There is only 10%–20% chance of muscle recovery in most cases. (Kirshblum, 1998)
Of those who recover, coordination and muscle power is poor.

Posterior Cord Syndrome (Figure 7–22)
- Least frequent syndrome
- Injury to the posterior columns results in proprioceptive loss (dorsal columns)
- Pain, temperature, touch are preserved. Motor function is preserved to varying degrees.

Conus Medullaris Syndrome
- Injury to the sacral cord (conus) and lumbar nerve roots within the spinal canal, usually results in areflexic bladder and bowel, and lower limbs (in low-level lesions) i.e., lesions at B in Figure 7–23.
- If it is a high conus lesion, bulbocavernous reflex and micturition may be present, i.e., lesions at A in Figure 7–23.

Cauda Equina Syndrome:
- Injury to the lumbosacral nerve roots within the neural canal, results in areflexic bladder, bowel, lower limbs, i.e., lesions at C in Figure 7–23.
- Bulbocavernous reflex absent

FIGURE 7–22. Posterior cord syndrome. (transverse section of the spinal cord—refer to Figure 7–2 for anatomical landmarks).

## SPINAL CORD INJURIES

### Table 7-2  Conus Medularis vs. Cauda Equina Syndrome

<table>
<thead>
<tr>
<th>CONUS MEDULLARIS</th>
<th>CAUDA EQUINA SYNDROME</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Location:</strong></td>
<td><strong>Location:</strong></td>
</tr>
<tr>
<td>L1–L2 vertebral</td>
<td>L2–sacrum vertebral</td>
</tr>
<tr>
<td>level injury of</td>
<td>level</td>
</tr>
<tr>
<td>sacral cord (S1–S5) and lumbar roots</td>
<td>Injury to lumbosacral nerve roots</td>
</tr>
<tr>
<td><strong>Causes:</strong></td>
<td><strong>Causes:</strong></td>
</tr>
<tr>
<td>• L1 fracture</td>
<td>• L2 or below fracture</td>
</tr>
<tr>
<td>• Tumors, gliomas</td>
<td>• Sacral fractures</td>
</tr>
<tr>
<td>• Vascular injury</td>
<td>• Fracture of pelvic ring</td>
</tr>
<tr>
<td>• Spina bifida, tethering of the cord</td>
<td>• Can be associated with spondylosis</td>
</tr>
<tr>
<td><strong>Resultant Signs and Symptoms:</strong></td>
<td><strong>Resultant Signs and Symptoms:</strong></td>
</tr>
<tr>
<td>1. Normal motor function of lower extremities unless S1–S2 motor involvement (since only involves S1–S5)</td>
<td>1. Flaccid paralysis of lower extremities of involved Lumbosacral nerve roots</td>
</tr>
<tr>
<td>Areflexic lower extremities If lumbar root involvement results in a lower motor neuron lesion (LMN)</td>
<td>Areflexic LE—results in a LMN Lesion</td>
</tr>
<tr>
<td>2. Saddle distribution sensory loss (touch is spared)</td>
<td>2. Sensory loss in root distribution</td>
</tr>
<tr>
<td>3. No pain</td>
<td>3. Pain</td>
</tr>
<tr>
<td>4. Symmetric abnormalities</td>
<td>4. Abnormalities predominate on one side (asymmetric)</td>
</tr>
<tr>
<td>5. Severe bowel, bladder, sexual dysfunction</td>
<td>5. High cauda equina lesions (lumbar roots) Spare bowel and bladder</td>
</tr>
<tr>
<td>Areflexic bowel</td>
<td>Lower lesions (S3–S5) can involve bowel and bladder and sexual dysfunction</td>
</tr>
<tr>
<td>Areflexic bladder</td>
<td>6. Bulbocavernosus reflex is absent (in low cauda equina [sacral] lesions)</td>
</tr>
<tr>
<td>6. If it is a high conus lesion, bulbocavernosus reflex may be present</td>
<td></td>
</tr>
<tr>
<td><strong>EMG:</strong> Normal EMG (except for external sphincter or S1, S2 involvement)</td>
<td><strong>EMG:</strong> Findings show multiple root level involvement Px: Good</td>
</tr>
</tbody>
</table>
TABLE 7-3. Functional Potential Outcomes for Cervical SCI (Complete) Patients (Kirshblum, 1998)

<table>
<thead>
<tr>
<th></th>
<th>C3–C4</th>
<th>C5</th>
<th>C6</th>
<th>C7</th>
<th>C8–T1</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Feeding</strong></td>
<td>May be able with adapted equipment</td>
<td>Independent with equipment after set up</td>
<td>Independent with equipment</td>
<td>Independent</td>
<td>Independent</td>
</tr>
<tr>
<td><strong>Grooming</strong></td>
<td>Dependent</td>
<td>Independent with equipment after set up</td>
<td>Independent with equipment</td>
<td>Independent</td>
<td>Independent</td>
</tr>
<tr>
<td><strong>UE Dressing</strong></td>
<td>Dependent</td>
<td>Requires assistance</td>
<td>Independent</td>
<td>Independent</td>
<td>Independent</td>
</tr>
<tr>
<td><strong>LE Dressing</strong></td>
<td>Dependent</td>
<td>Dependent</td>
<td>Requires assistance</td>
<td>May be independent with equipment</td>
<td>Independent</td>
</tr>
<tr>
<td><strong>Bathing</strong></td>
<td>Dependent</td>
<td>Dependent</td>
<td>Independent with equipment</td>
<td>Independent</td>
<td>Independent</td>
</tr>
<tr>
<td><strong>Bed Mobility</strong></td>
<td>Dependent</td>
<td>Requires assistance</td>
<td>Independent with equipment</td>
<td>Independent</td>
<td>Independent</td>
</tr>
<tr>
<td><strong>Weight Shifts</strong></td>
<td>Independent with power Dependent in manual</td>
<td>Requires assistance</td>
<td>Independent</td>
<td>Independent</td>
<td>Independent</td>
</tr>
<tr>
<td><strong>Transfers</strong></td>
<td>Dependent</td>
<td>Requires assistance</td>
<td>Possible independent with transfer board</td>
<td>Independent with or without board except floor transfer</td>
<td>Independent</td>
</tr>
<tr>
<td><strong>W/C Propulsion</strong></td>
<td>Independent with power Dependent in manual</td>
<td>Independent with power Short distances in manual with lugs or plastic rims on level surfaces</td>
<td>Independent manual with plastic rims on level surfaces</td>
<td>Independent except curbs</td>
<td>Independent</td>
</tr>
<tr>
<td><strong>Driving</strong></td>
<td>Unable</td>
<td>Unable</td>
<td>Specially adapted van</td>
<td>Car with hand controls or adapted van</td>
<td>Car with hand controls or adapted van</td>
</tr>
<tr>
<td><strong>Bowel and Bladder</strong></td>
<td>Dependent</td>
<td>Dependent</td>
<td>Independent—bowel assists—bladder</td>
<td>Independent</td>
<td>Independent</td>
</tr>
</tbody>
</table>
THE HIGHEST COMPLETE SCI LEVEL THAT CAN LIVE INDEPENDENTLY WITHOUT THE AID OF AN ATTENDANT IS A C6 COMPLETE TETRAPLEGIA.

- This patient would have to be extremely motivated
- Feeding is accomplished with a universal cuff for utensils
- Transfers require stabilization of elbow extension with forces transmitted from shoulder musculature through a closed kinetic chain
- Bowel care is performed using a suppository insertion wand or other apparatus for digital stimulation
- Outcome studies of a subset of patients with motor and sensory complete C6 SCI revealed the following percentage of patients were independent for key self-care tasks:
  - Feeding—16%
  - Upper body dressing—13%
  - Lower body dressing—3%
  - Grooming—19%
  - Bathing—9%
  - Bowel Care—3%
  - Transfers—6%
  - Wheelchair propulsion—88%

C7 level is the usual level for achieving independence.

MEDICAL COMPLICATIONS OF SCI

Important Levels to Remember:
T6 and above: Individuals with SCI are considered to be at risk for
  1. Autonomic Dysreflexia
  2. Orthostatic Hypotension
T8: If lesion above T8, patient cannot regulate and maintain normal body temperature
(Note: an easy way to remember this level is to spell the word temp eight ture.)
Central temperature regulation in the brain is located in the hypothalamus.

ORTHOSTATIC HYPOTENSION (see Table 7-4) (Corbett, 1971)
State of transient reflex depression

Cause: Lack of sympathetic outflow, triggered by tilt of patient > 60 degrees
- Lesion T6 or above
- T1–L2 responsible for:
  - Tachycardia, vasoconstriction and increased arterial pressure
  - Heart and blood vessels supplied by T1–T7

Mechanism
- Upright position causes decrease in blood pressure (BP)
- Carotid body baroreceptors sense decrease in BP, which would usually increase sympathetic outflow.
However brainstem is unable to send a message through the SC to cause sympathetic outflow and allow vasoconstriction of splanchnic bed to increase BP

**Resultant Symptoms**

1. Hypotension—loss of sympathetic tone (decreased systemic venous resistance, dilation of venous vessels) (decreased preload to the heart)
2. Tachycardia—Carotid body responds to hypotension, no increase in sympathetic outflow, however, they can still inhibit parasympathetics, but the increase in heart rate is not sufficient enough to counterbalance decrease BP
3. Patient can lose consciousness

**Treatment**

1. Reposition—Trendelenburg/daily tilt table/recliner wheelchair
2. Elastic Stocking/Abdominal Binder/Ace wrap LE
3. Add Salt/Meds:
   - Salt Tablets 1 gram QID
   - Florinef® (mineralocorticoid): 0.05–0.1 mg QD
   - Ephedrine (alpha agonist): 20–30 mg QD–QID
   - Use caution: The same patient is at risk for autonomic dysreflexia
4. Fluid resuscitation: monitor for neurogenic pulmonary edema
5. Orthostasis lessens with time due to the development of spinal postural reflexes. This causes vasoconstriction due to improved autoregulation of cerebrovascular circulation in the presence of perfusion pressure

**AUTONOMIC DYSREFLEXIA** (see Table 7-4) (Braddom, 1991) (Lindan, 1980)

**Onset:** After spinal shock, usually within first 6 months–1 year

**Incidence:** 48%–85%

**Cause:** Noxious stimulus below the level of the lesion causing massive imbalanced sympathetic discharge, i.e., too much sympathetic outflow

Most commonly caused by distended, full bladder

**Lesion:** SCI patients with lesions T6 or above (complete lesions)

**Mechanism:** Syndrome of massive imbalanced reflex sympathetic discharge in patients with SCI above the splanchnic outflow

This is secondary to the loss of descending sympathetic control and hypersensitivity of receptors below the level of the lesion

**Potential Symptoms:**

- Noxious stimuli—Increases sympathetic reflex spinal release
  - Regional vasoconstriction (especially GI tract)
  - Increases peripheral vascular resistance—increases cardiac output, increases BP
  - Carotid body responds to HTN causing reflex bradycardia by the dorsal motor nucleus of the vagus nerve

**Symptoms:**

- Headache, Flushing
- Piloerection
- Sweating above level of SCI
- Blurry vision (pupillary dilation)
- Nasal Congestion

**Note:** The brainstem is unable to send message through SCI to decrease sympathetic outflow and allow vasodilation of splanchnic bed to decrease BP
Most common causes:
- Bladder—blocked catheter
- Bowel—fecal impaction
- Pressure ulcers
- Ingrown toenails
- Urinary tract infections
- Bladder stones
- Gastric ulcers

- Labor
- Abdominal emergency
- Fractures
- Orgasm
- Epididymitis
- Cholecystitis

Treatment:
- Sit patient up
- Remove TEDS/Abdominal binder
- Identify and remove noxious stimulus
- Nitroglycerine—to control BP—1/150 sublingual or topical paste, which can be removed once noxious stimulus corrected
- Procardia®: 10 mg chew and swallow
- Hydralazine: 10–20 mg IM/IV
- Clonidine: 0.3–0.4 mg
- ICU - Nipride

Prevent Recurrence:
- Dibenzyline: 20–40 mg/day alpha blocker
- Minipress®: 0.5–1 TID alpha blocker
- Clonidine: 0.2 mg BID

Potential Complications of Autonomic Dysreflexia: If hypertensive episodes are not treated, complications can lead to: Retinal Hemorrhage, CVA, SAH, Seizure, Death
Autonomic Dysreflexia predisposes patient to atrial fibrillation by altering normal pattern of repolarization of the atria, making the heart susceptible to reentrant-type arrhythmias.
### TABLE 7-4  Orthostatic Hypertension vs. Autonomic Dysreflexia

<table>
<thead>
<tr>
<th><strong>Orthostatic Hypotension</strong></th>
<th><strong>Autonomic Dysreflexia (AD)</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Trigger:</strong></td>
<td><strong>Trigger:</strong> Noxious stimulus: especially full bladder below level of lesion</td>
</tr>
<tr>
<td>Tilt patient &gt; 60 degrees</td>
<td></td>
</tr>
<tr>
<td><strong>Due to:</strong></td>
<td><strong>Due to:</strong> Too much sympathetic outflow, loss of descending control, hypersensitivity</td>
</tr>
<tr>
<td>Lack of sympathetic outflow</td>
<td>status post spinal shock usually within first six months</td>
</tr>
<tr>
<td><strong>Lesion:</strong></td>
<td><strong>Lesion:</strong> T6 or above</td>
</tr>
<tr>
<td>T6 or above</td>
<td></td>
</tr>
<tr>
<td><strong>Symptoms:</strong></td>
<td><strong>Symptoms:</strong> Hypertension due to noxious stimulus.</td>
</tr>
<tr>
<td>Hypotension due to being positioned in the upright position</td>
<td>Bradycardia: carotid body responds to hypertension</td>
</tr>
<tr>
<td>Tachycardia: carotid body responds to hypotension</td>
<td>HA flushing</td>
</tr>
<tr>
<td>Patient loses consciousness</td>
<td>Piloerection</td>
</tr>
<tr>
<td></td>
<td>Sweating above level SCI</td>
</tr>
<tr>
<td></td>
<td>Blurred vision, pupillary dilation</td>
</tr>
<tr>
<td></td>
<td>Nasal congestion</td>
</tr>
<tr>
<td><strong>Note:</strong></td>
<td><strong>Note:</strong> Noxious stimulus causes massive sympathetic output</td>
</tr>
<tr>
<td>Upright position causes decrease in BP, carotid body Baroreceptors sense decrease BP, but brainstem is unable to send message through SC to cause sympathetic outflow and cause vasoconstriction of splanchnic bed to increase BP</td>
<td>Carotid body senses increased BP, but brainstem is unable to send message through SC to cause decreased sympathetic outflow and allow for vasodilation of splanchnic bed to bring BP down</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Tx:</strong></th>
<th><strong>Tx:</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Reposition: Trendelenburg</td>
<td>1. Sit patient up</td>
</tr>
<tr>
<td>2. Elastic stockings</td>
<td>2. Remove noxious stimulus (look for bladder distension, fecal impaction, etc.)</td>
</tr>
<tr>
<td>3. Abdominal binders</td>
<td>3. Treat hypertension</td>
</tr>
<tr>
<td>4. Increase salt</td>
<td>• Consider temporary treatment with nitrates (transderm), hydralazine (parenteral), morphine (parenteral), captopril (oral), labetalol (oral or IV)</td>
</tr>
<tr>
<td>5. Fluid resuscitation: monitor neurogenic pulmonary edema</td>
<td>• Decide need for intensive care and IV agents such as nitroglycerine, nitroprusside, spinal anesthesia</td>
</tr>
<tr>
<td><strong>Meds:</strong></td>
<td>It is estimated that 48%–85% of patients with high level SCI have symptoms of autonomic dysreflexia.</td>
</tr>
<tr>
<td>Florinef® (Mineralocorticoid)</td>
<td>Can lead to:</td>
</tr>
<tr>
<td>Salt Tablets</td>
<td>1. Retinal Hemorrhage</td>
</tr>
<tr>
<td>Ephedrine (Alpha Agonist)</td>
<td>2. CVA</td>
</tr>
<tr>
<td></td>
<td>3. SAH, seizure, death</td>
</tr>
<tr>
<td></td>
<td>AD may predispose patient to A. fib. by altering the normal pattern of repolarization of the atria, making the heart susceptible to reentrant-type arrhythmias.</td>
</tr>
</tbody>
</table>
BLADDER DYSFUNCTION

Neuroanatomy and Neurophysiology of Voiding

Central Pathways
- **Corticopontine Mesencephalic Nuclei—Frontal Lobe**
  Inhibits parasympathetic sacral micturition center
  Allows bladder storage
- **Pontine Mesencephalic**
  Coordinates bladder contraction and opening of sphincter
- **Pelvic and Pudendal Nuclei—Sacral Micturition**
  Integrates stimuli from cephalic centers
  Mediates the parasympathetic S2–S4 sacral micturition reflex
- **Motor Cortex to Pudendal Nucleus**
  Voluntary control (contraction/inhibition) of the external urethral sphincter

Peripheral Pathways (Figure 7–25)
- **Parasympathetic Efferents—S2–S4**
  Travel through the pelvic nerve to parasympathetic receptors
  Allows contraction of the bladder and emptying
- **Sympathetic Efferents—T11–L2**
  Travel through hypogastric plexi to sympathetic receptors
  (Alpha 1 + Beta 2 adrenergics)
  Urine Storage
- **Somatic Efferents—S2–S4**
  Travel through pudendal nerve to innervate striated muscle of external urethral sphincter
  Prevents urine leakage or emptying
- **Afferent Fiber**
  Travel through pudendal and pelvic nerve through the hypogastric plexi to thoraco-lumbar SC
  Origin — detrusor muscle stretch receptors
  external anal and urethral sphincter
  perineum and genitalia
  When bladder becomes distended, afferent nerve becomes activated for parasympathetic stimulation, resulting in emptying of bladder

* Neurologic Innervation of the Bladder (Bladder Receptors) (Figure 7–24)
  - **Cholinergic Muscarinic–M2**
    Located in the bladder wall, trigone, bladder neck, urethra
  - **Beta 2 Adrenergic**
    Concentrated in the body of the bladder, neck
  - **Alpha adrenergic**
    Located on the base of the bladder (neck and proximal urethra)
    (Note: Bladder wall does not have baroreceptors)

*Note:
Alpha Adrenergic receptors respond to the appearance of norepinephrine with contraction

Beta adrenergic receptors respond to the appearance of norepinephrine with relaxation
Urethral Sphincter

Internal Sphincter:
- Innervated by T11–T12 sympathetic nerve
- Contracts sphincter for storage
- Smooth muscle

External Sphincter
- Innervated by S2–S4 pudendal nerve
- Prevents leakage or emptying
- Skeletal muscle, voluntary control

Storage

Sympathetic (Figure 7–25)
encouraged during fight, flight

T11–L2 sympathetic efferents
- Travel through the hypogastric nerve
- Causes the sphincter to contract and body to relax
- Urine is stored

Alpha1 Receptors Adrenergic
- NE causes contraction of neck of bladder and prevents leakage
- Closes internal urethral sphincter and detrusor outlet, promoting storage

B2 Receptors Adrenergic
- Located in body of bladder
- Activation causes relaxation of body of bladder to allow expansion
- Inhibitory when activated

Emptying

Parasympathetic (Figure 7–25)
encouraged during relaxation

Muscarinic (M2) cholinergic receptors are located in:
- The bladder wall
- Trigone
- Bladder Neck
- Urethra

Stimulation of pelvic nerve (parasympathetic)
- Allows contraction of bladder + therefore, emptying!

B2 Receptors Adrenergic
- Relaxation of the bladder neck on the initiation of voiding

FIGURE 7–24. Bladder and proximal urethra distribution of autonomic receptors.
FIGURE 7–25. Neurologic innervation of the bladder.
Evaluation of Urinary Function: Cystometrogram and Pelvic Floor EMG

During cystometry: sensation, capacity, and the presence of involuntary detrusor activity are evaluated. A typical urodynamic study is depicted in Figure 7–26

- Sensations evaluated include:
  First sensation of bladder filling—occurs at approximately 50% of bladder capacity
  First urge to void—proprioceptive sensation
  Strong urge to void—proprioceptive sensation
- Accepted normal bladder capacity is 300–600ml
  Functional bladder capacity = voided volume + residual urine volume

FIGURE 7–26. Instrumentation for urodynamic studies is not standardized. The illustration above uses radio-opaque fluid. Some physicians, however, prefer to use carbon dioxide. Normal bladder function can be divided into storage and voiding phases. The first sensation of bladder filling is between 100 cc and 200 cc. The patient experiences bladder fullness between 300 cc and 400 cc and the sense of urgency between 400 cc and 500 cc. Intravesical pressure does not increase significantly during the storage phase due to the vascoelasticity of the vesical wall. During the voiding phase, sphincter activity stops and the bladder contracts. During normal voiding, the EMG signal will be silent, intravesical pressure will increase, and urethral pressure will decrease. Fluoroscopy will qualitatively assess bladder contraction and document any potential vesoureteral reflux. (From Nesathurai S. The Rehabilitation of People With Spinal Cord Injury: A House Officers Guide. © Boston Medical Center for the New England Regional Spinal Cord Injury Center. Boston, MA: Arbuckle Academic Publishers, with permission).
Normal Detrusor Contraction
Detrusor Pressure cm H2O and EMG Pelvic Floor (Fig. 7-27)

**FIGURE 7–27.** Normal Cystometrogram/Pelvic Floor EMG. 1. Bulbocavernous reflex. 2. Contraction of pelvic floor muscles during later phase of filling (progressively increasing electrical activity) 3. Functional bladder capacity. 4. Detrusor contraction that occurs during voiding. 5. Electrical silence (abrupt) which occurs during voiding. 6. Electrical activity of pelvic floor muscles that occurs during voluntary inhibition.

*Genitourinary Function and Management*

During the acute period after injury, the bladder usually presents *areflexic*, i.e., spinal shock phase.

May initially manage the bladder with indwelling catheter, while intravenous body fluids are administered. An intermittent catheterization program should be established soon after, with fluid restriction of approx. 100 cc/hr.

Volumes should always be monitored and maintained below 400–500 cc to avoid:
- Vesicoureteral reflux—caused by bladder hypertrophy and loss of the vesicoureteral angle (see previous page). This is normally prevented by the anatomy of the ureter, which penetrates the bladder obliquely through the trigone and courses several centimeters into the bladder epithelium.
- Overflow incontinence
- Hydro-ureter

Urodynamic studies should be performed to assess:
The bladder neck, the external sphincter, and the detrusor

Note: Bladder dysfunction is closely related to the level of injury, i.e., lower motor neuron vs. upper motor neuron.
**TABLE 7-5  Lower Motor Neuron Bladder vs. Upper Motor Neuron Bladder**

<table>
<thead>
<tr>
<th>LMN Bladder Failure to Empty (Fig. 7-28)</th>
<th>UMN Bladder Failure to Store (Fig. 7-29)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Causes:</strong></td>
<td><strong>Causes:</strong></td>
</tr>
<tr>
<td>• Spinal Shock: when reflex arc is not functioning due to initial trauma</td>
<td>• SCI: when reflex arc returns after initial trauma to cord passes</td>
</tr>
<tr>
<td>• Conus Medullaris Syndrome</td>
<td>• CVA</td>
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<tr>
<td>• Cauda Equina Syndrome</td>
<td>• Multiple Sclerosis</td>
</tr>
<tr>
<td>• Tabes Dorsalis, Pernicious Anemia, Syringomyelia</td>
<td></td>
</tr>
<tr>
<td>• Multiple Sclerosis</td>
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<tr>
<td><strong>Lesion:</strong></td>
<td><strong>Lesion:</strong></td>
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<tr>
<td>Complete destruction of Sacral Micturition Center (S2–S4) at S2 or below Lesion involving exclusively the peripheral innervation of the bladder</td>
<td>Above Sacral Micturition Center (above S2)</td>
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<tr>
<td><strong>Can Result in:</strong></td>
<td><strong>Can Result in:</strong></td>
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<tr>
<td>Big Hypotonic Bladder (flaccid, areflexic bladder), Tight Competent Sphincter</td>
<td>Small Hyperreflexic, Overactive, Little Bladder</td>
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<tr>
<td>Results in:</td>
<td>Results in:</td>
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<tr>
<td><strong>Failure to Empty</strong></td>
<td><strong>Failure to Store</strong> (Incontinence)</td>
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<tr>
<td>TX:</td>
<td>TX:</td>
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<tr>
<td>• Intermittent Catheter</td>
<td>• Ditropan: direct smooth muscle relaxer</td>
</tr>
<tr>
<td>• Crede maneuver (suprapubic pressure)</td>
<td>• Pro-Banthine®, Detrol®, anticholinergic</td>
</tr>
<tr>
<td>• Valsalva maneuver</td>
<td>• Tofranil®, ephedrine: stimulates alpha, beta receptors to allow storage</td>
</tr>
<tr>
<td>• Drugs to induce urination</td>
<td></td>
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<tr>
<td>Urecholine: stimulate cholinergic receptors</td>
<td></td>
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<tr>
<td>Minipress®, block alpha adrenergic receptors</td>
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<tr>
<td>Dibenzyline: block alpha adrenergic receptors</td>
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<tr>
<td>Hytrin®, block alpha adrenergic receptors</td>
<td></td>
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<tr>
<td>Cardura®, block alpha adrenergic receptors</td>
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</tbody>
</table>
LMN Bladder (Figure 7–28)
Failure To Empty
Cystometrogram and EMG

Note:
• Internal sphincter may have increased tension and prevent voiding
  These patients usually only void by:
• Overflow voiding when bladder can no longer expand

UMN Bladder (Figure 7–29)
Failure to Store
Cystometrogram and EMG

Due to upper motor neuron lesion, there is no suppression of micturition center, therefore, patient voids prematurely


Combination Type Bladder (Figures 7–30, 7-31)

Many patients (as many as 85%) with SCI develop Detrusor Sphincter Dyssynergia (DSD)

Causes:
- Central Cord Syndrome
- MS
- Progression of SCI (UMN Lesion)

Lesion:
Neurologic injuries between the sacral (S2–S4) and pontine micturition center

Resultant Scenario:
Tight little bladder (Detrusor hyperreflexia)
Tight sphincter (Sphincter hyperactivity)

Result:
Failure to void

Risk if Not Treated:
- Infected urine travels up towards kidneys (Figure 7–30)
- Note: These patients frequently have frequency and urgency, but lack of coordination between bladder and sphincter. This prevents complete bladder emptying.
- Result: Increased residual volumes, urine becomes infected, patient then tries high-pressure voiding against closed sphincter, this sends infected urine up to kidneys

Treatment:
1. Anticholinergic Meds – to expand the detrusor to prevent infected urine from going up to the kidneys (intact sphincter is good for continence)
2. Intermittent catheterization
3. Antimuscarinic drugs (i.e., anticholinergic) to cause bladder relaxation
4. Alpha blocker—to open bladder neck
5. Sphincterotomy

FIGURE 7–30. Reflux of infected urine backs up towards kidneys.

FIGURE 7–31. Cystometrogram and EMG in a patient with complete upper motor neuron bladder dysfunction shows:
1. Brisk bulbocavernous reflex. 2. Bladder capacity is reduced. 3. High intravesical pressure during detrusor contraction. 4. Detrusor/external urethral sphincter dyssynergia with marked electrical activity of the pelvic floor muscles during detrusor contraction.
The normal bladder mechanism that prevents vesicoureteral reflux is described below:

**Normal Anatomy (Figure 7–32A)**
- The one-way valve mechanism can remain competent only as long as the oblique course of the ureter within the bladder wall is maintained
- During relaxation of the bladder, when urine is being stored, the ureter pumps urine into the bladder
- During bladder contraction the valve shuts closed. As a result urine cannot reenter the ureter and, therefore, the bladder is emptied with no reflux of urine into the ureters. (Figure A)

**Bladder Wall Hypertrophy (Figure 7–32B)**
- When bladder hypertrophy causes the course of the distal ureter to become progressively perpendicular to the inner surface of the bladder, the vesicoureteral function becomes incompetent, permitting vesicoureteral reflux
- During relaxation of the bladder, the ureter pumps urine into the bladder
- During bladder contraction, because the distal ureter becomes perpendicular to the inner surface of the bladder, the valve cannot close. Urine is forced up the ureter to the kidney and hydronephrosis can result. (Figure B)
- Reflux is further complicated by acute or chronic pyelonephritis with progressive renal failure.

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**FIGURE 7–32.** Vesicoureteral Junction. **A(1):** Normal Open Valve: The muscle of the ureter “milks” urine through the valve into the bladder. **A(2):** Normal Closed Valve: When the bladder contracts, the valve is pressed shut. The normal valve prevents urine from flowing back into the ureter.

**B(1):** Abnormal Open Valve: The abnormal valve still allows urine to pass through the ureter into the bladder. **B(2):** Abnormal Valve: The abnormal valve is unable to close. When the bladder contracts, urine is pushed back into the ureter and kidney.
**Intermittent Catheterization (IC)**

IC has reduced many of the associated complications of the indwelling catheter, which include epididymitis, penoscrotal abscess, fistula formation, renal and bladder calculi, and malignancy. IC has been shown to reduce the incidence of urinary tract infection.

Pathophysiology of Urinary Tract Infections (UTI):

UTIs are generally caused by the endogenous flora of the host overcoming the competing normal flora and host defense mechanism. The presence of the UTI is affected by:

- The virulence of the invading microorganism
- The condition of the urine as the culture medium
- The host defense mechanisms

**In general:**

An acidic concentrated urine inhibits microbial growth. UTIs are prevented by the washout effort of large volumes of urine. The large flow of fluid impedes the adherence of microorganisms and dilutes the concentration of microorganisms.

**Management of UTIs**

**Asymptomatic UTIs**

In SCI patients on an intermittent catheterization (IC) program recurrent asymptomatic bacteriuria (with less than 50 WBC per high power field) is generally not treated to avoid the development of resistant organisms.

(The exception to this is evidence of vesicoureteral reflux, hydronephrosis, or growth of urea splitting organisms.)

In general, asymptomatic UTI in individuals with indwelling catheters should not be treated.

**Symptomatic UTIs**

Symptomatic bacteriuria with fever leukocytosis or increased spasticity is treated and the catheterization is increased to reduce bacterial concentration and remove the urine that serves as a culture medium for bacterial growth. (A Foley may be necessary if volumes are too large for an IC program)

**Most Common Urinary Tract Complications in Neurogenic Bladder**

- Irregular, thickened bladder wall and small diverticuli—earliest changes
- Vesicoureteral reflux: 10%–30% of poorly managed bladders, leads to pyelonephritis, renal stones
- Hydronephrosis and hydroureters caused by outlet obstruction
- Overdistended areflexic bladder
- Bladder infections can lead to marked reduction in compliance of bladder

**Prevention**

All of these complications can be prevented by adequately draining the bladder at a pressure below 40 cm H<sub>2</sub>O, either by intermittent catheterization along with the use of anticholinergic drugs or by timely surgical relief of the outflow obstruction

**Prophylactic Treatment of UTIs in SCI patients**

- Prophylactic antibiotic role is not fully established, but it is still used
  (i.e., Macrodantin<sup>®</sup> (nitrofurantoin))
- Vitamin C supplementation, cranberry juice, methenamine salts—overall used as acidifying agents
SEXUAL DYSFUNCTION

Physiology of Normal Sexual Act

Male Sexual Act (Bors, 1960)
Male erectile and ejaculatory function are complex physiologic activities that require interaction between vascular, nervous, and endocrine systems.
Erections are controlled by parasympathetic nervous system
Ejaculations are controlled by sympathetic nervous system

Erection:
Controlled by a reflex arc that is mediated in the sacral spinal cord
A reflex involves an afferent and an efferent limb

The Afferent Limb:
• Consists of somatic afferent fibers from the genital region that travel through the pudendal nerve into the sacral spinal cord

The Efferent Limb:
• Involves parasympathetic fibers that originate in the sacral spinal cord. These fibers travel through the cauda equina and exit via S2–S4 nerve roots.
• Postganglionic parasympathetic fibers secrete nitric oxide, which causes:
  – Relaxation of smooth muscle of the corpus cavernosum
  – Increases blood flow to the penile arteries—vascular sinusoids of the penis become engorged with blood
  – The result is an erection
  – This reflex is modulated by higher brainstem, subcortical, and cortical centers.

Ejaculation:
• Signals the culmination of the male sexual act, and is primarily controlled by the sympathetic nervous system
• Similar to sympathetic innervation to the bladder—these fibers originate in the thoracolumbar spinal cord (T11–L2), then travel through the hypogastric plexus to supply the vas deferens, seminal vesicle, and ejaculatory ducts

Female Sexual Act
• Sexual excitement is the result of psychogenic and physical stimulation
• Stimulation of the genital region, including clitoris, labia majora, and labia minora, causes afferent signals to travel via the pudendal nerve into the S2–S4 segment of the spinal cord
• These fibers interact with the efferent parasympathetic fibers that project through the pelvic nerve
• The result is:
  – Dilation of arteries to perineal muscles and tightening of the introitus
  – Bartholin’s glands secrete mucus, which aids in vaginal lubrication
• Female orgasm is characterized by the rhythmic contraction of the pelvic structures.
  Female orgasm also results in cervical dilation, which may aid in sperm transport and fertility.
Erectile Dysfunction

Men with SCI may obtain reflexogenic or psychogenic erections.

**Reflexogenic Erections**

- Can occur independently of conscious awareness and supraspinal input (mediated by paraspinal division of ANS through sacral roots S2–S4).
- Are secondary to manual stimulation of the genital region (however, once stimulation has been removed, the erection may no longer be sustained).

**Psychogenic Erections**

- Involve supraspinal (above SC) effects, that are the result of erotic stimuli that result in cortical modulation of the sacral reflex arc.
- Erection is mediated by central origin and psychological activated center.

In general, erections are more likely with incomplete lesions (both UMN and LMN) than complete lesions. Many times, men with SCI can only maintain an erection while the penis is stimulated and the quality of the erection is insufficient for sexual satisfaction. As such, the erection must be augmented or induced.

**Methods to Induce Erections**

- Oral Therapy – Sildenafil, i.e., Viagra®
- Intracavernosal injection therapy – i.e. papaverine, alprostadil, phentolamine
- Penile vacuum device
- Transurethral devices – i.e., alprostadil
- Penile implants

Ejaculatory Dysfunction

In men with SCI, the ability to ejaculate is less than the ability to obtain an erection.

The rate of ejaculation varies depending on the location and nature of the neurologic injury:

- Complete UMN lesions: ejaculation rate is estimated at 2%
- Incomplete UMN lesions: ejaculation rate is estimated at 32%
- Complete LMN lesions: ejaculation rate is estimated at 18%
- Incomplete LMN lesions: ejaculation rate is estimated at 70%

Most SCI males are unable to ejaculate. If they do, they are usually incomplete LMN lesions.

**Methods to Induce Ejaculation**

- Intrathecal neostigmine
- Subcutaneous physostigmine
- Direct aspiration of sperm from vas deferens
- Vibratory stimulation – can be used at home
  - Increased incidence of autonomic dysreflexia
  - Males inseminate females with syringe
- Electroejaculation: most popular in USA
  - In incomplete lesion: very painful
  - If sensation is intact, patient cannot tolerate pain leading to heart rate and BP increase, and *autonomic dysreflexia* is a problem.
Equipment used has been modified and it is possible to obtain the ejaculate through low-intensity constant repeatable current. Patients need medical supervision. Hospitals or office-based procedure: to evaluate through anoscopy before and after procedure to assess for injury to rectal mucosa. Pretreat with Nifedipine (has lowered risk of autonomic dysreflexia).

Direct Stimulation of Hypogastric Nerve
- Ejaculate is obtained through the use of an implanted hypogastric nerve stimulator.
- Surgical procedure—not appropriate for patients with intact pelvic pain sensitivity, since hypogastric nerve stimulation causes severe pain.

Sexual function might not return for 6–24 months. 80% experience return within 1 year of injury, 5% in 2 years.

Infertility in Males With SCI (Linsenmeyer and Perkash, 1991)
Fertility in many paraplegic and tetraplegic men after SCI is severely impaired. Two major causes are (already discussed), ejaculatory dysfunction and poor semen quality.

Poor semen quality is secondary to
- Stasis of prostatic fluid
- Testicular hyperthermia
- Recurrent UTI
- Abnormal testicular histology
- Changes in hypothalamic-pituitary-testicular axis
- Possible sperm antibodies
- Type of bladder management
- Chronic—Long-term use of various medications

Stasis of prostatic fluid
- Decreases sperm motility
- Studies have shown that in patients who did not have spontaneous ejaculations, there was an improvement in semen quality after 2–4 electroejaculations

Testicular Hyperthermia
Studies have shown higher deep scrotal temperatures (average = 0.9°C higher) in men with paraplegia who were seated when compared to noninjured control subjects who were seated. Men with SCI often sit with their legs close together, in contrast to nonimpaired men.

Sperm Counts and Motility Indices
- Sperm counts are lower in men who were having prostatic inflammation compared to those who were not
- Leukocytes (WBC > 10^6) in the spermatic fluid reduced total sperm count 41%, sperm velocity 12%, and total motile sperm 66%
- The single worst predictive factor for immobility to penetrate an ovum was leukocyte concentration in the semen
- Postinfective changes may affect fertility, such as atrophy of the testicles or obstruction of epididymal ducts.

Most Common Finding Noted on Biopsy is Atrophy of the Seminiferous Tubules
No investigations have found a significant correlation among biopsy finding, level of injury, length of injury, hormonal changes, or number of UTIs.
Testosterone
Appears to remain normal or slightly above or below normal

Antisperm Antibodies
- Inhibit cervical mucous penetration
- Despite studies, immunologic-mediated infertility remains controversial
- Infertility due to antibodies is often not an absolute condition; additional time may be required but pregnancy can occur
- Two factors associated with antibody formation include: obstruction of the genital tract and UTIs

Female Infertility
Immediately postinjury 44%–58% of women suffer from temporary amenorrhea. Menstruation returns within six months post injury. Most women with SCI are fertile.

Birth Control
Can be problematic for SCI women:
- Condoms—provide protection
- Diaphragm—need adequate hand dexterity
- Oral Contraceptives—associated with increased risk of thromboembolism
- IUD—can increase risk of pelvic inflammatory disease, which may lead to autonomic dysreflexia

Pregnancy
The likelihood of pregnancy after spinal cord injury is unchanged, since fertility is unimpaired. Pregnant women with SCI have an increased risk of:
- UTIs
- Leg edema
- Autonomic dysreflexia
- Constipation
- Thromboembolism
- Premature birth
Uterine innervation arises from T10–T12 level
Patients with lesions above T10 may not be able to perceive uterine contractions. Pre-eclampsia may be difficult to differentiate from autonomic dysreflexia. Autonomic dysreflexia may be the only clinical manifestation of labor.
GASTROINTESTINAL COMPLICATIONS AND BOWEL MANAGEMENT

Innervation of Bowel—Review of Anatomy and Neuroregulatory Control (Figure 7–33)

- The colon is a closed tube bound proximally by the ileocecal valve and distally by the anal sphincter
- The colon is composed of smooth muscle oriented in an inner circular and outer longitudinal layer
- The lower colon and anorectal region receive innervation by the sympathetic, parasympathetic, and somatic pathways
- In addition, the intrinsic enteric nervous system (ENS), composed of the Auerbach’s (myenteric) plexus and the Meissner’s (submucosal) plexus, coordinates the function of each segment of the bowel
- Auerbach’s plexus is primarily motor; Meissner’s plexus is primarily sensory. Both these plexi lie between the walls of smooth muscle mentioned earlier.
- The parasympathetic and sympathetic nervous systems modulate the activity of the ENS, which in turn inhibits the inherent automaticity of the bowel’s smooth muscle

The Parasympathetic Nervous System
- Increases upper GI tract motility
- Enhances colonic motility
- Stimulation is provided by the action of the vagus nerve, which innervates proximal to mid. transverse colon, and by the splanchnic nerves (pelvic nerve), which originate from the S2–S4 region, which innervate the descending colon and rectal region

The Sympathetic Nervous System
- Stimulation inhibits colonic contractions, and relaxes the internal anal sphincter favoring the function of storage
- Innervation projects through the hypogastric nerve via superior mesenteric, inferior mesenteric, and celiac ganglia

The Somatic Nervous System
- Increases external anal sphincter tone to promote continence
- The external sphincter (EAS) consists of a circular band of striated muscle that is part of the pelvic floor

Anal Region

The Internal Anal Sphincter
- Composed of smooth muscle under the influence of the sympathetic system (T11–L2)
- Surrounds the anus proximally.
- In patients without SCI the sphincter normally relaxes with filling of the rectum.

The External Anal Sphincter
- Composed of skeletal muscle
- Helps to maintain continence by increasing its tone
- It acts under volitional control, learned by maturation, and reflex activity.
- It is innervated by the pudendal nerve (roots S2–S4). Higher cortical centers and the pontine defecation center send stimulus for EAS relaxation, allowing defecation.
Storage and Defecation in the Neurologically Intact Individual

Storage
- The internal anal sphincter, is sympathetically activated (T11–L2) allowing for relaxation. This occurs with filling of the rectum in patients without SCI.
- External anal sphincter (EAS) tone increases, secondary to spinal cord reflexes and modulated action of higher cortical regions, maintaining continence.

Defecation
- Rectosigmoid distention causes reflex internal anal sphincter relaxation.
- Volitional cortical activity sends signal to pontine defecation center. Volitional contraction of the levator ani muscle occurs, opening the proximal canal, relaxing the external anal sphincter and puborectalis muscles.
- Reflexive rectal propulsive contractions take place resulting in expulsion of the stool bolus.

Defecation in the Spinal Cord Injured Patient

Upper Motor Neuron Lesions (Hyperreflexic Bowel)
- Cortical control is disrupted, with decreased ability to sense the urge to defecate.
- EAS cannot be voluntarily relaxed and pelvic floor muscles become spastic. However, nerve connections between the spinal cord and colon, as well as the myenteric (Auerbach's) plexus remain intact and the stool can be propelled by reflex activity.

Lower Motor Neuron Lesions (Areflexic Bowel)
- Spinal shock or lesion below conus medullaris
- Reflex defecation is absent.
• Myenteric (Auerbach’s) plexus coordinates the movement of stool, but movement is slow
• Overall, constipation may result (most common result)

Note: Attenuated or absent external anal sphincter contractions may result in fecal smearing or fecal incontinence

In SCI the GI system can be affected by loss of sympathetic and parasympathetic input at the transverse and descending colon, resulting in decreased fecal movement. In SCI, fecal impaction and constipation is the most common complication during recovery. To help with defecation, the physician may take advantage of two reflexes:

The Gastrocolic Reflex
Increased colonic activity occurs in the first 30 to 60 minutes after a meal (usually within 15 minutes). Therefore, place the SCI patient on the commode within one hour subsequent to a meal.

The Anorectal Reflex (Rectocolic Reflex)
Occurs when the rectal contents stretch the bowel wall reflexively, relaxing the internal anal sphincter. Suppositories and digestive stimulation cause the bowel wall to stretch and take advantage of this reflex.

Note this reflex
Can be manipulated by digital stimulation of the rectum. Digital stimulation is accomplished by gently inserting a lubricated finger into the rectum, and slowly moving the digit in a clockwise manner.

Management of Bowel Dysfunction in SCI

Acute Phase

Gastric Atony and Ileus
• After a significant SCI, the patient is at high risk for the development of gastric Atony and Ileus, which may cause vomiting and aspiration
• Stomach decompression by nasogastric tube should be considered in all acutely SCI patients.
• The ileus onset can be delayed for 24–48 hrs.
• The ileus usually lasts from 3–4 days up to 7 days.

Gastric Atony and Ileus (or Adynamic Ileus) occurs in 63% of patients with SCI
• Results from spinal shock and reflex depression.

Management
– NG suction to prevent GI dilation and respiratory compromise
– IV fluids
– Abdominal Massage—TENS to stimulate peristalsis of gut
– Injections of neostigmine methylsulfate (Prostigmine) 3–5 hrs.
– As soon as bowel sounds appear, start clear liquid diet
– If persists—may use Reglan

Chronic Phase

• Colonic distention: problems with small bowel motility and gastric emptying
• Pseudo obstruction: no evidence of obstruction on radiographic studies.
• Abdominal distention, nausea, vomiting, constipation.
• Secondary causes: electrolyte imbalance and medications (narcotics, anticholinergics)
Management
- NG suction
- Remove offending agent
- If cecum is dilated >12 cm., surgical decompression or colonoscopy

Constipation
Long-Term Management
- Defecation done using bedside commode (sitting facilitates emptying)
- Maintain adequate fluid intake, medications that decrease bowel motility such as narcotics, tricyclic agents, and anticholinergics should be minimized

Diet: high fiber

Meds
Bulk Cathartics: promotes evacuation by retaining or pulling H2O into colon
- Metamucil®: dietary fiber increase

Irritants
- Castor oil, irritates bowel—AVOID castor oil

Fecal Softener
- Colace®: increases fluid accumulation in GI tract, AVOID Peri-Colace®—causes cramping

Oral Stimulants
- Senokot®: stimulates peristalsis by acting on Auerbach’s plexus

Suppositories: placed high against rectal wall
- Glycerine: draws water into stool/stretches rectal wall
- Dulcolax®: stimulates peristalsis. Stimulates sensory nerve endings

Bowel Program
Initially Aims for Bowel Movement Daily
Surgical Intervention—Bowel diversion done when incontinence becomes a problem

Bowel Program consists of:
- Glycerin supp. (or Dulcolax®)
- Encourage patient to have BM at same time QD
- Use Gastrocolic Reflex—Q1hr post breakfast or dinner

If meds. are used, start with:
1. Dulcolax® Q daily after meal - dinner or breakfast
2. Stool softener (Colace®): 100 mg. TID
3. Senokot®: PO q daily at noon

(Spas ticity of external anal sphincter may signify interference with bowel care) complications of neurogenic bowel:
Fecal Incontinence: Skin breakdown, ulcerations, UTI
Fecal Impaction: Autonomic hyperreflexia
Must use Lidocaine® Gel during digital extraction

Anticholinergic meds used for failure-to-store bladder can cause severe constipation
Bowel dysfunction affects the patient’s community integration—socially, vocationally and psychologically

Other Gastrointestinal Complications
Gastroesophageal reflux
- Avoid prolonged recumbency; elevate the head of the bed
- Avoid smoking
- Avoid medications: Ca+ channel blockers, Valium®, nitrates, anticholinergics
**Treatment:**
Provide antacids for mild to moderate symptoms:
- H₂ antagonists, Metoclopramide 10 mg tid, Omeprazole 20 mg QD

**Gastroesophageal bleeding**
- Most frequently secondary to perforating and bleeding ulcers
- Stress ulcers secondary to interruption of sympathetic vasoconstrictors (vasodilatation and mucosal hemorrhage)
- Steroid use
- Increased gastric secretion

**Treatment:**
Provide prophylaxis with:
- Antacids
- H₂ blockers—Cimetidine, Ranitidine, Famotidine
- Sucralfate—stimulates local prostaglandin synthesis

*Endoscopy* is the diagnostic method of choice
*With active GI bleeding*—maintain BP, correct coagulation deficits, consult GI/Surgical service

**Cholecystitis**
- Most common cause of emergency abdominal surgery in SCI patients
- Increased risk: 3x > in SCI
- Possible causes: abnormal gallbladder motility in lesions above T10, abnormal biliary secretion, abnormal enterohepatic circulation

**Treatment:**
Observe, May opt for surgical removal or dissolution

**Pancreatitis**
- Most common in the first month post injury.
- May be related to steroid use—increased viscosity of pancreatic secretions
- May suspect when adynamic ileus doesn’t improve.

**Evaluate**
- Radiographs
- CT
- Ultrasonogram
- Labs: amylase, lipase

**Superior Mesenteric Artery (SMA) Syndrome**
Condition in which the third portion of the duodenum is intermittently compressed by overlying SMA resulting in GI obstruction (Figure 7–34) (Roth, 1991)

**Predisposing factors include:**
- Rapid weight loss (decrease in protective fatty layer)
- Prolonged supine position
- Spinal orthosis
- Flaccid abdominal wall causes hyperextension of the back
Exacerbated by:
- Supine positioning
- Tetraplegic patient with abdominal and cervical orthosis

Symptoms:
- Postprandial nausea and vomiting
- Bloating
- Abdominal pain

Diagnosis:
UGI Series: demonstrates abrupt duodenal obstruction to barium flow

Treatment:
Conservative
- Eat small, frequent meals in an upright position
- Lie in the left lateral decubitus position after eating
- Metoclopramide (Reglan®): stimulates motility of UGI tract

Rarely requires surgery
- If conservative treatment fails, surgical duodenojejunostomy (DJ ostomy)

Remember:
Any condition that decreases the normal distance between the SMA and aorta (weight loss, supine position, halo, flaccid abdominal wall) may result in compression of the duodenum described as the nutcracker effect

FIGURE 7–34. Lateral view through duodenum and left renal vein.
METABOLIC COMPLICATIONS

Hypercalciuria

Immobilization, decreased weight bearing promotes bone resorption. Patients become hypercalciuric—this may continue for 18 months. Vitamin D, parathyroid hormone are not involved in the process.

Hypercalcemia

Patients with hypercalciuria can, in rare cases, develop hypercalcemia. Symptoms: nausea, vomiting, dehydration, decreased renal function, decreased mental status, abdominal discomfort, anorexia, malaise. Symptoms of hypercalcemia can be remembered with the mnemonic stones, bones, and abdominal groans. Patients are most commonly young tetraplegic males.

Treatment: (Merli, 1984)
- Hydration—IV normal saline solution, furosemide
- Mobilize the patient: tilt table, weight bearing activities
- Decrease GI absorption of calcium: give steroids, decrease vitamin D
- Didronel®, Calcitonin

Osteoporosis

Secondary to disuse—localizes below the level of the lesion. Calcium excretion also increased (increased bone resorption). Approx. 22% bone loss 3 months post injury (Claus-Walker, 1975). Increased risk of fracture.

Treatment
- Weight bearing
- FES cycling—demonstrated to decrease the rate of bone resorption in acute SCI, effect remains after discontinuation
- Pharmacologic agents have not been proven to reduce osteoporosis in SCI.

Hyperglycemia

Up to 70% of the patients show insulin resistance, with abnormal response to glucose load. Rarely require treatment (Duckworth, 1983)
MUSCULOSKELETAL COMPLICATIONS

Upper Extremity Problems

Shoulder is the most commonly affected joint. Frequently secondary to weight bearing and overuse.

Causes
- Impingement syndrome
- Overuse
- DJD
- Rotator cuff tear/bicipital tendinitis
- Subacromial bursitis
- Capsulitis
- Myofascial pain
- Disuse
- Cervical radiculopathy

Diagnosis
- Consider possible causes such as heterotopic ossification, syrinx
- Perform complete physical exam, including functional assessment, ROM, flexibility, and sensation

Treatment
- Rest, pharmacotherapy for pain treatment
- Compensatory techniques to be used for daily function
- Treat the condition
- Educate the patient regarding posture, weight bearing

Compression neuropathies
Have been noted to increase with the length of time from injury.
- 27% incidence of carpal tunnel syndrome in SCI patients seen 1–10 years post-injury
- 54% incidence of carpal tunnel syndrome in SCI patients seen 11–30 years post-injury
- 90% incidence of carpal tunnel syndrome in SCI patients seen more than 31 years post-injury
  (Ditunno, 1992)

PULMONARY COMPLICATIONS OF SCI AND MANAGEMENT

Incidence

Respiratory complications occur in 50% of patients. During the first month post injury, if the initial rehabilitation period is included, the incidence increased to 67% of SCI patients.

Atelectasis/pneumonia have an onset within the first 24 days post injury

Pulmonary complications are more common in high cervical injuries (C1—C4)
The most frequent complications are pneumonia, atelectasis, and ventilatory failure.

In low cervical (C5—C8) and thoracic (T11—T12) complications are equally frequent.

Thoracic injuries present with: pleural effusion, atelectasis, pneumothorax, hemothorax, or both.
  (Fishburn, 1990; Jackson, 1994; Langis, 1992)
Pulmonary Dysfunction: Occurs for several reasons following SCI
1. Paralysis of some or all respiratory muscles to varying degrees
2. Loss of ability to cough secondary to varying levels of abdominal muscle paralysis
3. Injury to chest—e.g. rib fracture
4. Pulmonary injury—e.g. lung contusion

Predisposing Factors for Pulmonary Complications Include
Older Age
Obesity—restrictive respiratory deficits
Hx—COPD
Hx—Smoking

Pneumonia is the leading cause of death among long-term SCI patients. Patients tend to retain secretions in the lower lung fields due to:
- Difficulty in achieving postural drainage positions during the acute SCI period
- Altered ventilatory pattern after SCI (reduced airflow to lower lobes leads to atelectasis)
- Decreased ability to clear secretions independently, decreased effective cough

Left-sided respiratory complications are more common among hospitalized SCI patients. This is due to the following:
- The left mainstem bronchus takes off as a 40°–50° angle from vertical, making routine suctioning more difficult.
- In addition to this, there is a tendency to retain secretions in the lower fields.

Respiratory function may be affected to different degrees depending on the level of injury.

Pulmonary Compromise Related to Level of Injury
- Head trauma: May knock out respiratory drive
- Lesions above C3 (and incomplete lesions initially):
  - Initially they require ventilatory support
  - Later they will fall into two groups
    1. No damage to phrenic nucleus
    2. Damage to phrenic nucleus

No Damage to Phrenic Nucleus
- C3,4,5
- Determined by EMG of phrenic nerve
- Can stimulate phrenic nerve nucleus therefore, the patient will benefit from phrenic pacing (i.e., C1,2, and incomplete lesions)

Damage to Phrenic Nucleus
- C3,4,5
- Determined by EMG of phrenic nerve
- Cannot stimulate phrenic nerve nucleus, therefore, will not benefit from phrenic pacing (i.e., lesions of phrenic nucleus causing irreparable damage*)

Continue to require ventilatory support

*Note: Intercostal nerve grafts are being attempted

EMG of diaphragm is necessary to rule out damage to phrenic nerve nucleus
• C3: Respiratory failure secondary to disruption of diaphragmatic innervation, requiring mechanical ventilation
• C4: Generally the highest level of injury at which spontaneous ventilation can be sustained
• Injuries above C8: Loss of all abdominal and intercostal muscles, impairment of inspiration and expiration
• T1 through T5: Intercostal volitional function is lost
• T5 through T12: Progressive loss of abdominal motor function, impairing forceful expiration or cough
• Injuries below T12: Few complications if there is lung injury (e.g. trauma) otherwise, no respiratory dysfunction

Phrenic Pacing (Lee, 1989)
• Phrenic pacing has reduced the need for mechanical ventilation in tetraplegic patients with respiratory failure since its introduction in 1972
• The technique involves the electrical stimulation of intact phrenic nerves via surgically implanted electrodes to contract the diaphragm
• Induces artificial ventilation through electric stimulation of the phrenic nerve, which causes the diaphragm to contract
• Used successfully in patients with COPD, central hypoventilation, and high tetraplegia
• Treatment option in patients with respiratory paralysis after cervical injury above the origin of the phrenic neurons
• Option in patients who do not have significant impairment of the phrenic nerves, lungs, or diaphragm

Contraindications to Phrenic Pacing
• Denervated diaphragm (determined in EMG)
• Denervated—nonviable anterior horn cells C3, C4, C5
• Placement of phrenic pacer prior to 6 months post injury is contraindicated
  From 0–6 months—the chest is too FLAIL and flaccid
  You need some rigidity of chest wall to allow pacer to work
• Significant lung impairment

Major Complications of Phrenic Pacemaker
□ Signs of Failure of Pacemaker
  1. Sharp chest pain
  2. SOB—Shortness of Breath
  3. Absence of breath
  4. Erratic pacing
  5. Must maintain adequate ventilation via manual resuscitation bag

□ Causes of Phrenic Pacing Failure
  1. Diaphragmatic failure—due to overly aggressive pacing schedule
  2. Infection of lung and/or phrenic nerve
  3. Meds: including sedatives, tranquilizers, and narcotics
  4. Upper airway obstruction—tracheal aspiration
  5. Phrenic nerve damage from overstimulation or surgery

□ Benefits of Phrenic Pacing
• Increased arterial oxygenation despite decreased alveolar ventilation
• Longer survival in patients with SCI
• Increased daily function secondary to conditioning of the diaphragm from nocturnal pacing
Physiology of Lung:

**Inspiratory Muscles**
- Accessory muscles
- Diaphragm—main respiratory muscle, main muscle during quiet breathing (75% of volume change) Contracts at inspiration, relaxes at expiration
- External intercostals

**Expiratory Muscles**
- Abdominal muscles—primarily active during forceful expiration and in producing cough (contract); push the relaxed diaphragm toward the chest cavity
- Internal intercostals

---

**FIGURE 7–35.** Inspiration in the Normal Lung and in a Lung with Insult to the Phrenic Nerve

LATERAL VIEW OF THE LUNG
- Abdomen Moves Out
- Chest Wall Moves Out
- Diaphragm Moves Down

Note: Thoracic Cavity gets larger

LATERAL VIEW OF THE LUNG
- Chest Wall Moves Out
- Diaphragm Moves Up
- Abdomen Moves In
Pulmonary Function In SCI—Restrictive Respiratory Changes (Langis, 1992)

- *Forced Vital Capacity* (FVC) during the acute phase of cervical injury is noted to decrease 24%–31% when compared to the normal values secondary to paradoxical respirations.
- With development of intercostal and abdominal spasticity FVC *can improve to* 50–60% of predicted normal value.
- Tetraplegics develop *restrictive lung patterns*.
  - All volumes shrink (except residual volume).
  - If VC < 1 liter: consider ventilation mechanically.
  - If VC < 600: must have ventilation mechanically.
  - If VC > 1.5 get off ventilator.
- Signs of impending respiratory failure:
  - Increased respiratory rate with decreased tidal volume.
  - Decreased FVC < 15 cc/Kg body weight.
  - Decreased inspiratory force <20 mm H₂O.
  - Neurological level C3 or higher.
  - Patient can’t count to 15 slowly.

*Mechanically ventilate when:*

- VC < 1 liter.
- ABG show increasing PCO₂ or decreasing PO₂ levels.
  - PO₂ < 50.
  - PCO₂ > 50.
- Severe atelectasis.

**Prevention of Respiratory Complications**

- Use of incentive spirometer.
- Monitor CO₂ levels with ABGs.
- Monitor vital capacity.
- Cough assist—placing the hands on each side of the pts. upper abdomen and applying intermittent pressure, coordinated with the initiation of cough by the patient—helps produce forceful cough.
- Suctioning—remember—tracheal suctioning may cause increased vagal tone with SA node suppression and brady-arrhythmia, leading to cardiac arrest (only suction as you withdraw catheter).
- Chest physical therapy—(see pulmonary chapter).
- Strengthening of pectoralis major muscle, clavicular portion, in tetraplegic patients.
- Glossopharyngeal breathing—stroking maneuver to force air into the lungs by the use of the lips, soft palate, mouth, tongue, pharynx, and larynx, followed by passive exhalation.
- Pneumobelt—helps with exhalation. Inflatable, compresses the abdominal wall, diaphragm rises, and active respiration is produced.
HETERO TOPIC OSSIFICATION

Definition
Formation of mature lamellar bone indistinguishable from normal bone in soft tissues, most frequently deposited around a joint.
As bone matures it becomes encapsulated, not connected to periosteum.

Causes
- Possibly due to alteration in neuronal control over the differentiation of mesenchymal cells into osteoblasts which form new bone.
- A decrease in tissue oxygenation or induces changes in multipotential connective tissue cells in which new bone forms in planes between connective tissue layers.
- No definitive explanation established.

Incidence: Heterotopic Ossification (HO) has been reported to occur in 16%–53% of patients following SCI.
Clinically significant HO: (resulting in significant limitation of joint range) affects 10%–20% of SCI patients.
Occurs below the level of neurological injury (only in the area of paralysis, unless other factors are present such as TBI or burn).

Onset
1–4 months status post injury most common, but can present after first 6 months.

Symptoms: Early clinical findings include heat and soft tissue swelling.
Swelling progresses to more localized and firm area over several days, may present as ROM in joint decreases.
- Heat.
- Localized soft tissue swelling—may look like DVT.
- Decreased ROM of a joint.
- Joint erythema/joint effusion.
- Low grade fever.

Risk Factors
- Spasticity.
- Completeness of injuries.
- Trauma or prior surgery to joint.
- Age.
- Pressure ulcer in proximity of joint.

Diagnosis: Can be seen one week from onset in static bone scan or/triple phase bone scan precedes X-ray by at least 7–10 days.
Plain film detects HO in 7–10 days after clinical signs are observed.
Bone Scan returns to normal as HO matures in 6–18 months post injury.
Serum Alkaline Phosphatase: Increases at 2 weeks—exceeds normal levels at 3 weeks—peaks at 10 weeks—returns to normal after HO matures.
Not specific for HO.

Treatment:
- Didronel® (etidronate disodium): 20 mg/Kg/day for 2 weeks then 10 mg/Kg/day for 10 weeks.
  Does not change overall incidence, but less HO is laid down overall.
- Indocin®—Not commonly used in acute SCI.
• ROM—Maintain function while HO matures—The goal is to maintain functional range. The affected joint should be gently moved through functional range—vigorous force should not be used as this may lead to further ectopic bone formation.
• Surgery—Used when HO severely limits ROM impairing function—should only be planned after bone is mature: 12–18 months post injury. Bone scan must be back to baseline and alkaline phosphatase should be back to normal.

Complications
Peripheral nerve entrapment
Decreased ROM/loss of function/ankylosing
HO overlying a bony prominence will directly predispose to pressure ulcer/skin breakdown secondary to poor positioning

DEEP VENOUS THROMBOEMBOLISM (DVT)/ PULMONARY EMBOLISM (PE) IN SCI

Deep Venous Thrombosis (DVT)

Predisposing Factors
Virchow’s Triad: Venous stasis/intimal injury/hypercoagulability
LE fractures
Obesity
Hx of previous DVT
DM
Arterial vascular disease
Immobility
Malignancy

Incidence
Ranges 47%–100%
– Varies Widely depending on the method of detection and number of cases evaluated in the study
More common in neurologically complete patients
More common in tetraplegic patients
10 times more frequent in plegic leg
20% of calf vein thrombi extend proximally

Onset
Most common during first 2 weeks after SCI
Greatest incidence decreases after 8–12 weeks post SCI

Diagnosis
Venogram is the Gold Standard
Venous Doppler is used as a screening test for lower extremity DVTs
Impedance Plethysmography—accurate in assessment of DVTs above the calf
Sensitivity—95%, Specificity—98%
Used to look for occlusions in the thigh, place cuff around the thigh and listen for flow

Complications: □ Pulmonary Embolism (PE) leading cause of death in acute LCI

Pulmonary Embolism (Fluter, 1993; Goldhaber, 1998)

Symptoms
Pleuritic chest pain
Dyspnea
Fever, hemoptysis
Tachycardia
Hypoxemia

Physical Examination
1. Increased S2 sound: severe pulmonary HTN → cor pulmonale. → Right heart failure
2. Dullness at bases of lungs

Incidence
1%–7% of SCI patients (Hull)

Diagnosis (PE)
EKG: R. Axis Deviation
Right Bundle Branch Block (RBBB):—if massive PE
ABG:—decreased PO₂ (PO₂ drops severely)
Chest X-ray:
  — wedge shaped opacity
  — fluid
  — vascularity
Perfusion lung scan: VQ mismatched

Gold Standard: Pulmonary arteriogram

Treatment (PE)
O₂
Heparin
Vasopressor to treat shock

Surgical Treatment
Embolectomy

Other Complications of DVT
• Postphlebitic syndrome (late complication of DVT)
  — distal venous hypertension (residual obstruction of outflow—incompetent valve)
  — swelling
  — exercise induced pain
  — pigmentation
  — ulceration
  — long standing autonomic dysreflexia

Prophylaxis Treatment and Prevention of DVT

Prophylaxis for DVT (Merli, 1988)
• External intermittent pneumatic compression devices
  — increases LE venous return
  — decreases stasis and stimulates fibrinolysis
  — if delays > 72 hours—need to do duplex prior to initiation
• Enoxaparin—low molecular weight heparin (LMWH)
  — Dose 30 mg SQ BID
  — Best intervention to prevent DVT if no contraindications
  — Not used in patients with active bleeding, TBI or coagulopathy
• Thigh-high graded compression stockings (TEDS)—alone, not prophylaxis
• Coumadin®
• Minidose subcutaneous unfractionated Heparin
• Greenfield filter (may be indicated in selected cases, high risk, or failed prophylaxis)
Treatment DVT:
• Heparin—if not contraindicated
  – standard: 5,000 units IV bolus; followed by a constant infusion of 1,000 units/ (25,000 units in 250 cc D5W at 10 cc/hr)
  – maintain PTT 1.5-2 times normal
  – at least 5–10 days of anticoagulant prior to mobilization
• Warfarin started once PTT therapeutic (approximately three days after Heparin started); takes 5 days to load; target INR 3.0
  Coumadin for 3 months in case with DVT
  Coumadin for 3–6 months in case w/PE
  (Note: Heparin can be discontinued once coumadin is 1/2 times normal for 48 hrs.)
• No ROM in involved extremity. With small popliteal clots, patients may transfer to bedside chair in 1–2 days. If clot is in proximal veins or with PE, immobilization 5–10 days.
• If anticoagulation is contraindicated, then an IVC filter is necessary

Prevention:
• Recommended that patients receive both a method of mechanical prophylaxis as well as anticoagulant prophylaxis
• Pneumatic compression stockings or device should be applied to the legs of all patients during the first two weeks following injury
  If this is delayed for more than 72 hours after injury, test to exclude the presence of clots should be performed
• Anticoagulant prophylaxis—LMW heparin or adjusted unfractionated heparin should be initiated within 72 hours after injury if there is no hemorrhage or risk of bleeding
  LMWH: 30 SQ BID

Functional Electrical Stimulation (FES) in SCI has two general uses
• As exercise to avoid complications of muscle inactivity
• As a means of producing extremity motion for functional activities
  FES can be used to
  – Provide a cardiovascular conditioning program
  – Increase muscle bulk strength and endurance
  – Attempt to decrease risk of DVT
  – Produce extremity motion for standing and ambulation

PAIN IN THE SCI PATIENT

Incidence of chronic pain in SCI population is estimated between 20%–50%
Pain may be musculoskeletal, neuropathic, or visceral

MUSCULOSKELETAL PAIN

Upper Extremity Pain: common in the SCI patient

Patients with SCI load joints that do not normally bear weight (shoulder, elbow, wrist)
This predisposes them to painful UE conditions
These conditions include
• Carpal tunnel syndrome (which is present in up to 90% of SCI pts. at 31 years post injury)
• Rotator cuff tendonitis
• Rotator cuff tears
• Subacromial bursitis
Cervical radiculopathy
Lateral epicondylitis
Medial epicondylitis
Myofascial pain

Less common causes of UE discomfort include
Syringomyelia
Heterotopic ossification
Angina
Aortic dissection
Pancoast tumor

**Syringomyelia: posttraumatic cystic myelopathy (Dworkin, 1985; Umbach and Halpern, 1991; Williams, 1992)**
- The pathogenesis of posttraumatic syringomyelia is not entirely understood. Cavitation of the spinal cord usually occurs at the level of the initial injury. Cavity formation may be secondary to liquefaction of the spinal cord or from central hematoma present at the initial injury. The lesion usually progresses in a cephalad direction. As the lesion progresses and compromises more nerve fibers, symptoms may become more apparent.
- Occurs in .3%–3.2% of the SCI population and is the most common cause of progressive myelopathy after SCI.
- It can occur 2–34 months post injury, and even much later
- It may present as pain and numbness; motor weakness is often associated with sensory loss
- It occurs more frequently with thoracic and lumbar regions
- Extension of the cavity can be upward or downward (normally cephalad)
- MRI is the most accurate diagnostic technique
- Treatment is surgical and drainage can be accomplished with a shunt to the subarachnoid space or peritoneum. Motor weakness and pain have a good prognosis with surgical treatments.

**Charcot Spine**

Charcot Joints: A destructive arthropathy of joints, with impaired pain perception or position sense. Loss of sensation of deep pain or of proprioception affects the joints normal protective reflexes, often allowing trauma (especially repeated minor episodes) and small periarticular fractures to pass unrecognized.

Charcot Spine: Spinal trauma and analgesia below the level of injury makes SCI patients particularly prone to insensate joint destruction. Joints themselves can be a source of pain that triggers autonomic dysreflexia or a nidus of infection after hematogenous spread.

**NEUROPATHIC PAIN**

Neuropathic pain may be of central or peripheral origin. Patients will complain of a burning or shooting pain. The discomfort may involve the abdomen, rectum, or lower extremity. It may exacerbated by other noxious stimuli, including urinary tract infections, renal stone, HO, etc. Neuropathic pain is more common with incomplete lesions. Neuropathic pain requires complete assessment.

**VISCERAL PAIN**

Evaluation of acute abdominal pathology in SCI patients with potentially impaired sensation can be very difficult. The typical clinical features may be absent. Pain, when present, may be atypical in quality and location. Increased spasticity and a general feeling of unwellness may be the only manifestations of a surgical emergency.
Spasticity

Spasticity presents as an abnormality of muscle tone and is common in SCI individuals. It becomes clinically apparent as spinal shock resolves. (See chapter on Spasticity.)

PRESSURE ULCERS

25%–40% of SCI patients develop pressure ulcers at some time during their life. Pressure ulcers are classified according to the extent of tissue damage.

<table>
<thead>
<tr>
<th>SHEA CLASSIFICATION I–IV</th>
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</thead>
<tbody>
<tr>
<td>I</td>
</tr>
<tr>
<td>II</td>
</tr>
<tr>
<td>III</td>
</tr>
<tr>
<td>IV</td>
</tr>
</tbody>
</table>

GRADE-DANIEL CLASSIFICATION (FIGURE 7–36)

Less commonly used classification:

<table>
<thead>
<tr>
<th>Levels of ulceration</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin erythema or induration</td>
<td>Superficial ulceration advances into dermis</td>
<td>Extends into subcutaneous fat</td>
<td>Extends through muscle down to bone</td>
<td>Ulcer extends into bone/jt capsule, or body cavity</td>
<td></td>
</tr>
</tbody>
</table>

FIGURE 7–36. Levels of ulceration graded according to depth of tissue involvement
MECHANISM OF DEVELOPING A PRESSURE ULCER

Local soft tissue ischemia results due to prolonged pressure over bony prominences, that exceed supra capillary pressure (70mm Hg)

1. Ischemia: lack of blood supply to the tissue
   - Frequently associated with hyperemia in the surrounding tissue
   - Increased local O2 consumption occurs

2. Pressure
   - Prolonged pressure over bony prominences, exceeding supracapillary pressure (70 mm Hg pressure continuously for 2 hours) results in occlusion of the microvessels of the dermis
   - Occlusion of the microvessels occurs when the force exerted on the vessel wall is greater than the intraarterial pressure
   - This results in immediate epidermal ischemia
   - Ischemia causes hyperemia of the surrounding tissue

Tissues vary with regard to their sensitivity to pressure
   Muscle is more sensitive to pressure, skin is more resistant to pressure

Important Facts

Note: 70 mm Hg pressure continuously × 2 hour: results in tissue damage

Muscle is more susceptible to pressure ischemia than skin

3. Friction (shearing force):
   - Removes corpus striatum (stratum corneum) of the skin
   - Friction mechanically separates the epidermis immediately above the basal cells
   - Friction is a factor in the pathogenesis since it applies mechanical forces to the epidermis

Common Locations of Pressure Ulcers (Figure 7–37)

During the acute period after SCI the most common locations of ulcers are due to the patient lying supine:
#1 Sacrum
#2 Heels

In chronic SCI patients the locations of ulcers are as follows:
Ischial decubitus (30 %)
Greater trochanter (20%)
Sacrum (15%)
Heels (10%)

Risk Factors

• Immobility
• Incontinence
• Lack of sensation
• Altered level on consciousness

Prevention of Pressure Ulcers

• Minimize extrinsic factors—pressure, maceration, and friction
• Decrease pressure forces, the patient should be turned and positioned every 2 hours
• Pressure relief every 30 minutes when sitting
• Proper cushioning and wheelchair seating (see wheelchairs)
• WC pushups
Treatment

- Prevention of pressure ulcers should always be the first line of defense
- Once a lesion has developed, however, rational treatment should be prescribed to reduce the progression of the ulcer; the extrinsic factors that contributed to the formation of the ulcer should be identified and treated
- In general, healing will be promoted if the wound remains clean, moist, and debrided—a noninfected wound will also promote healing.

<table>
<thead>
<tr>
<th>Grade</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depth of Ulcer</td>
<td>Superficial epidermis and dermal layers</td>
<td>Extends to adipose tissue</td>
<td>Full thickness defect to and including muscle</td>
<td>Destruction down to bone and/or joint structures</td>
</tr>
<tr>
<td>Treatment</td>
<td>Alteration of mattress/Wet to dry dressing</td>
<td>Sharp/enzymatic debridement of ulcer</td>
<td>Sharp/enzymatic debridement of ulcer</td>
<td>Surgery/surgical consultation</td>
</tr>
<tr>
<td>Alternative Treatment</td>
<td>Possible surgical consultation</td>
<td>Surgical consultation</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
POST OP MANAGEMENT OF SACRAL DECUBITUS GRAFTING

- Positioning—Patient should be prone for 2–4 weeks
  If this is not tolerated, pressure relief bed should be prescribed to prevent iatrogenic pressure.
- Control the patient’s spasticity.
- Antibiotic treatment—Used to address issues of infection
- Bowel and bladder management—To avoid contamination of the wound

Pressure Ulcer Complications

- Osteomyelitis
- Dehydration

REFERENCES


RECOMMENDED READING


