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.

🕮 SHEA CLASSIFICATION I–IV

I	Superficial epidermis and dermal layers
II	Extends to adipose tissue
III	Full thickness skin defect down to and including muscle
IV	Destruction down to bone and or joint structures

GRADE-DANIEL CLASSIFICATION (FIGURE 7–36)

Less commonly used classification:

Levels of ulceration	**1**	**2**	**3**	**4**	**5**
	Skin erythema or induration	Superficial ulceration advances into dermis	Extends into subcutaneous fat	Extends through muscle down to bone	Ulcer extends into bone/jt capsule, or body cavity

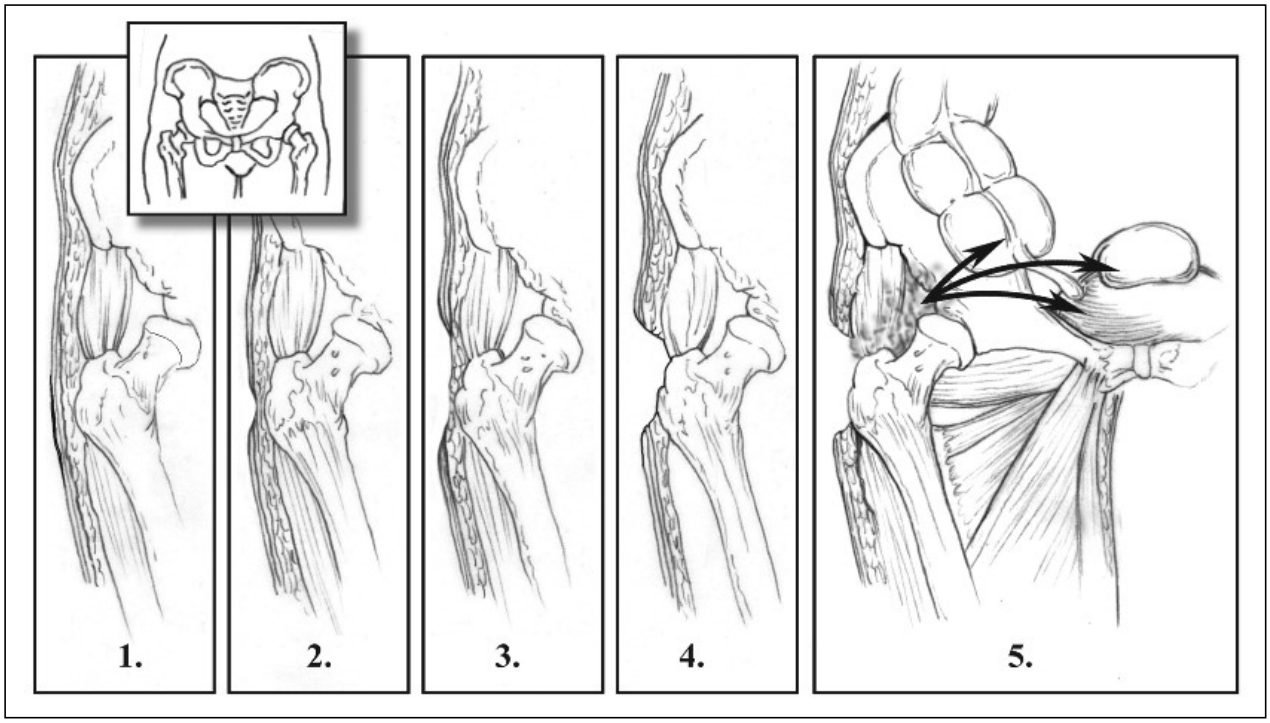

FIGURE 7–36. Levels of ulceration graded according to depth of tissue involvement

🕮 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

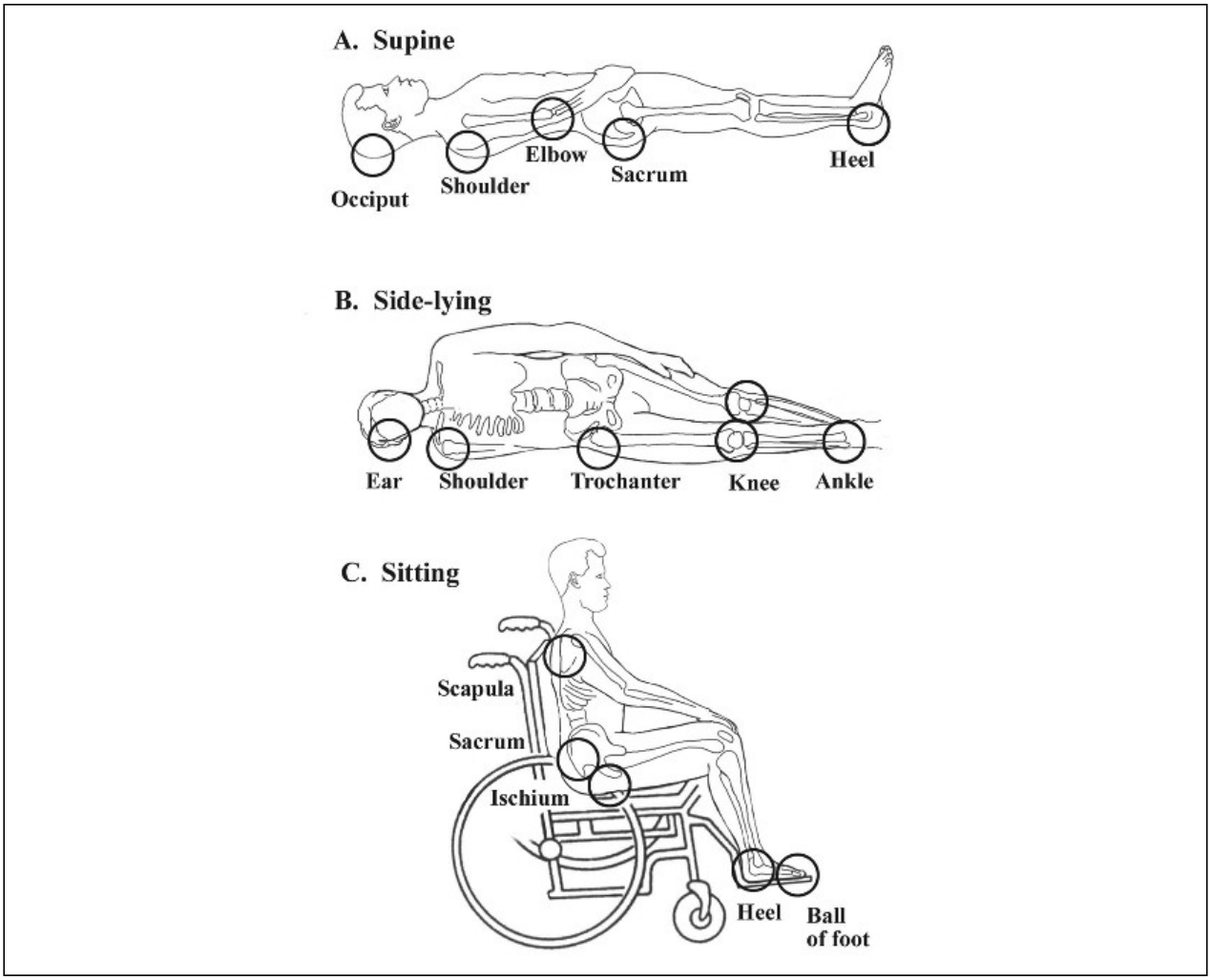

FIGURE 7–37. Common locations of pressure ulcers

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 7-6. Treatment: According to Shea Classification

Grade	I	II	III	IV
Depth of Ulcer	**Superficial epidermis** and dermal layers	**Extends to adipose** tissue	**Full thickness defect** to and including muscle	**Destruction down** to bone and/or joint structures
Treatment	Alteration of mattress Wet to dry dressing	Sharp/enzymatic debridement of ulcer	Sharp/enzymatic debridement of ulcer	Surgery/surgical consultation
Alternative Treatment		Possible surgical consultation	Surgical consultation	

🕮 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

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8

Physical Modalities, Therapeutic Exercise, Extended Bedrest, and Aging Effects

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PHYSICAL MODALITIES

Modalities that use physical energy for their therapeutic effect.
Includes:
- Pressure
- Thermotherapy—Heat and cold
- Hydrotherapy
- Light therapy—ultraviolet radiation, laser
- Electrotherapy
- Manipulation, mobilization, traction, massage, acupuncture

These are used as adjuncts to a therapy program including exercise and patient education

THERMOTHERAPY

- The amount of energy a tissue gains or loses depends on several factors:
  - Nature of the tissue
  - Agent used
  - Duration of exposure
- Temperature has an effect on:
  - **Viscosity**
  - **Nerve conduction**—heat increases nerve conduction velocity; cold decreases it
  - **Blood flow**—heat increases arterial and capillary blood flow; cold decreases blood flow
  - **Collagen extensibility**—heat increases tendon extensibility, collagenase activity is increased; cold decreases enzyme activity
- Temperatures > 45–50°C (113–122 °F) or < 0°C (32°F) can injure tissue

A. Heat

Therapeutic uses for heat are based on:
- Hyperemia
- Analgesia
- Hyperthermia
- Decreased muscle tone
- Increase in collagen elasticity