

**Nursing 202: Advanced Concepts of Nursing**  
**Unit IV: Neurotrauma**  
**Spinal Cord Injuries (SCI)**

- **Spinal Cord**

About 18 inches long

The major bundle of nerves that carry nerve impulses from the brain to the rest of the body

Extends from the base of the brain to about the waist

- **Vertebra**

Function = protection & support

“Rings” of bone (33)

Named according to location:

Cervical (7)

Thoracic (12)

Lumbar (5)

Sacral (5) fused as 1

Coccygeal (4) fused as 1

- **Upper Motor Neurons**

Lie within the spinal cord (above L1)

Carry messages back and forth from the brain to the spinal nerves along the spinal tract

Lesions: see spasticity, hyperreflexia below the level of lesion

- **Lower Motor Neurons**

Spinal nerves that branch out from the SC to the body

Exit and enter at each vertebral level and communicate with specific areas of the body

**They activate the muscle they innervate**

Sensory and motor neurons

Lesions:

**Cause weakness, paralysis, flaccidity**

- **Vascular Supply**

Arterial

Anterior spinal and two posterior spinal arteries

Arise from Circle of Willis

feed the entire length of the spinal cord

- **History**

Prior to WWII, life expectancy was months-10yrs

Cause of death used to be renal failure

Now many advances in urology

Major cause of death is now compromised respiratory function

Pneumonia, PE or Septicemia

Christopher Reeves had a major impact on research

- Long-term issues remain

Disruption in growth and development

Altered family dynamics

Economic loss

High cost of rehabilitation and long-term care

- Trauma or damage to spinal cord

Temporary or permanent dysfunction

- 17,000 new SCIs each year in United States

- 282,000 Americans living with SCI
- Decreased life expectancy; increased mortality
- 30% rehospitalization rate
- Causes: Most related to trauma

38% motor vehicle collisions

30.5% falls

13.5% violence

9% sports injuries

2/3 from diving incidents

- **Sex**

Males 82%

More risk takers

Usually, a hx of multiple injuries prior to SCI

- **Risk Factors**

Age

Sex

ETOH/Drug Use

Time of year - summer

#### Classification of SCI

- SCI is classified by:

Mechanism of injury

Level of injury

Degree of injury

- **Knowing the type of collision/accident can aid in assessment of injury (MOI)**

Forces responsible for hyperextension and hyperflexion

Acceleration (hit in the rear when stopped)

Deceleration (stopped while in motion)

Deformity (various alterations in the spinal cord and supporting structures)

- Mechanisms of Injury

Flexion

Flexion-rotation

Most unstable, leading to severe neurologic deficits

Hyperextension

Vertical compression (axial loading)

Extension-rotation

Lateral flexion

Penetrating

- **Hyperextension (head snaps back)**

Usually fracture of posterior element of spinal column and disruption of anterior longitudinal ligaments.

- **Hyperflexion (head snaps forward)**

Head on collision

Deceleration from the front

Head and body continue moving forward until contact is made

The head is forced forward until it rests on the chest

- **Vertical Compression (axial loading)**

Vertical force on the spinal columns

Diving accidents, falls on buttock

- **Rotation**

Wedge fx

Extreme lateral flexion or twisting of the head and neck

May result in posterior ligaments tear, dislocation or fracture

- **Penetrating**

Missiles, bullets, knives

- Types of Injury/ Classification

- **Vertebral Column Injury without SCI**

Immobilize

Protect from SCI – bone fragment or unstable vertebrae can injure the cord

- **Vertebral Column Injury with SCI**

Prevent more damage!

- **SCIWORA Syndrome**

SCI without radiographic abnormality

- **Vertebral**

Fracture

**Simple (singular break)**

Alignment usually intact

Usually no neural compression

**Compression (wedge)**

Often caused when vertebral column is flexed

Hard collar for 2 months

**Comminuted (burst)**

Vertebral column is straight with shattered vertebral body

Fragments can damage the cord

May need rodding and removal of fragments

Dislocation

One vertebrae overrides another

Supporting ligaments often injured

Tx = traction & immobilization

Stable vs. Unstable

Degree of Injury

- Complete

Total loss of sensory and motor function below level of injury

- Incomplete (partial)

Mixed loss of voluntary motor activity and sensation

Some tracts intact

Five major syndromes associated with incomplete injuries (to be discussed)

Special Type Cervical Fractures / Dislocation

**Jefferson**

Rare

Fx of C1

Burst Fracture  
Unstable  
From vertebral compression (axial loading)  
Special Type Cervical Fractures / Dislocation

---

**Atlanto - Occipital**

Dislocation  
Avulsion of C1 body from occipital bone  
Rare almost always immediately fatal

---

**Odontoid (Dens) Fx**

Need to R/O in all pts in MVC with neck pain  
Can be missed in X-ray  
Standard of care is now CT  
Special Type Cervical Fractures / Dislocation

---

**Hangman Fx**

Most unstable with neuro deficit  
Separation body of C2 from posterior elements  
Classified by the amount of displacement in body of C2  
Often fatal

- **Partial (incomplete) SCI**

---

**Brown-Sequard Syndrome**

Damage to ½ of cord  
Typically results from penetrating injury  
Ipsilateral paralysis or paresis  
Contralateral loss of pain and temperature  
Example

**Left** side of the cord has been transected

Paralysis of all voluntary muscles below the injury on the **left** side (ipsilateral)

Loss of the perception of touch, vibration and position on **left**

Loss of perception of pain and temp on the **right** side of the body (contralateral)

- **Partial (incomplete) SCI**

---

**Anterior Cord Syndrome**

Injury at the anterior spinal artery causes compromised blood flow  
Flexion injury  
Loss of pain, temp, motor function below the level of the injury  
Light touch, position, vibration intact

- **Partial (incomplete) SCI**

---

**Cauda Equina Syndrome**

“Horsetail” below the spinal cord  
Lumbosacral nerve roots  
Affects lower motor neurons (Flaccid paralysis of lower limbs)  
Bowel and bladder dysfunction  
Severe asymmetric pain

- **Partial (incomplete) SCI**

---

**Central Cord Syndrome**

Injury/edema of the central cord  
Commonly cervical region/ older adults  
Motor weakening or paralysis in upper extremities  
Lower intact

- **Partial (incomplete) SCI**

### Conus Medullaris Syndrome

Damage to lowest portion of spinal cord

Decrease in or loss of sensation in perianal area

Areflexic bladder/ bowel

Impotence

Pain is uncommon

- **Levels of Injury**

**Dermatome** identifies specific areas of the **skin** supplied by sensory fibers.

Touch, vibration, position, pain, temp

**Myotome** identifies **muscle** group innervated by nerves

Motor function

- Case Study

**EMS arrives on scene of a 23 y.o. male diving off the dock into the bay. The patient cannot move his fingers or hands.**

**What level is his injury?**

- Etiology and Pathophysiology
  - Primary Injury
- Direct physical trauma from blunt or penetrating trauma
- Spinal cord compression by

Bone displacement

Interruption of blood supply

Traction from pulling on cord

- Penetrating trauma, causing tearing and transection
- Predisposed areas= C5-6

Maximum movement of cervical spine takes place, cord largest at this level, multiple nerve fibers that feed the upper extremities

- **Pathophysiology**

Vertebral injury vs. spinal cord injury

Can have a fx without cord injury

R/F Compressing the spine

Initial Injury

Chain of Events

1 hour - petechial hemorrhage in gray matter

4 hours - infarction in gray matter

Hemorrhage, edema, metabolites act together to produce ischemia which progresses to necrotic destruction of cord

Hypoxia results - decreases O<sub>2</sub> supply below the level to meet the metabolic needs of the cord.

Increase in lactic acid and ↑ vasoactive substances = vasospasm = more hypoxia = more necrosis

24 hours - permanent damage because of the edema that developed secondary to inflammation - cord compressed!

When the cord is compressed more, then inflammation ↑ at the site- = ischemia.

Additional edema extends the level of the injury beyond the immediate level of the injury for 72 hours to 7 days.

Makes it difficult to assess the extent of the injury.

Secondary Injury

- Ongoing, progressive damage that occurs after primary injury

Causes further permanent damage

Begins a few minutes after injury and lasts for months

- Events Leading to Secondary Injury
- Spinal Shock
- Occurs shortly after injury and lasts days to weeks

Temporary syndrome

- Characterized by

Loss of deep tendon reflexes and sphincter reflexes

Loss of sensation

Flaccid paralysis below level of injury

- **Often masks post-injury neurologic function**
- Can last 1-6 weeks, on average
- Ends when spastic paralysis replaces flaccid paralysis
- Prognosis:

A person who gradually loses neurological functioning after an accident has some chance of recovering at least partial function when spinal shock dissipates.

Return of function is not due to regeneration

Spinal shock masks the function which may still be intact

Patient and family will ask about recovery but **there is no way to know at first!!**

- Neurogenic Shock
- Occurs in cervical or high thoracic (at or above T6) injury; can last 1 to 3 weeks
- Loss of SNS (fight or flight) innervation causing unopposed parasympathetic response

Peripheral vasodilation

Venous pooling

Decreased cardiac output

- Characterized by

Hypotension (less than 90 mmHg)

Bradycardia

Temperature dysregulation

- Diagnostics

CT scan

Preferred study for location and degree of injury and degree of spinal canal compromise

Cervical x-rays

MRI

Soft tissue injury and neurologic conditions

Guide decisions about surgery

Comprehensive neurologic examination

CT angiogram

- Emergency Management
- Pre-hospital
- Immediate goals

Patent airway

Adequate ventilation/breathing

Adequate circulating blood volume

Prevent extension of spinal cord damage

- Suspect SCI in any patients with:

Wounds of the face, neck, head, & shoulders

Any unconscious patient

Head injuries

Neck pain

Any motor or sensory losses

Signs of spinal shock

Initial

Patent airway and adequate respirations

Administer O<sub>2</sub>; keep >90%

Maintain SBP above 90 mmHg

IV access; infuse NSS or LR

Immobilize and stabilize cervical spine

Assess for other injuries

Control external bleeding

- Immobilization of cervical spine

Jaw Thrust

No head tilt or chin lift

No flexion or extension of neck

Rigid cervical collar

Backboard with straps

Keep supine; log-roll for transfers

Chemical or physical restraints if needed to protect from further injury

- Intubation to secure airway if in respiratory distress

- IV fluids and vasopressors

Treat neurogenic shock

Keep SBP above 90 mmHg

- Stabilize at scene, then transfer to nearest medical facility that ideally specializes in SCI

- Thorough assessment

Determine degree of deficit

Establish level and degree of injury

- Acute Care

- Initial assessment in ED

Managing ABCs and vital signs

Secure airway, intubate if indicated

Maintain: HR, mean arterial pressure above 85 mmHg, SBP above 90 mmHg

Medical interventions and diagnostics for hemodynamic stability

Neurologic assessment

Test muscle groups bilaterally

Record strength, symmetry and spontaneous movement

Sensory assessment

Pinprick

Position sense and vibration, as time permits

Rectal tone and priapism

- Review Priorities of Care...

- Emergency Management

- Primary Survey

Airway, Breathing, Circulation, Disability

- Prevent further cord shock

- Always assume cervical spine injury

Maintain neutral alignment

No head tilt, No flexion of neck

### **Airway**

C1-4 phrenic nerve does not function = loss of ability to cough & deep breath

Jaw thrust

No head tilt or chin lift

No flexion or extension of the neck

### **Breathing**

Above C4 = loss of respiratory muscle

Below C4 = diaphragmatic breathing if phrenic nerve functioning

Hypoventilation r/t decrease vital capacity

### **Circulation**

Hypovolemia

External bleed – can see and control

Internal bleed – more difficult

Remember neurogenic shock!!

### **Disability**

Baseline neuro status

Estimate cord involvement

Need to avoid flexion, extension, rotation of the spine.

### **Exposure**

Remove clothing

Log Roll

Spine must be kept in neutral alignment

Patient must be placed on a longboard with cervical collar- WE move the pt!

Meyer-Cervical Orthosis – provides constant cervical spine traction & reduces the risk of injury

during resuscitation and transport

Temperature instability may occur, keep covered & warm

Also remember other injuries – usually have multiple injuries (internal and head)

- Emergency Department Management
- Assessment

### **Airway**

Cervical injury- can identify if they are unable to move their fingers and hands

Clear the c-spine

CT is gold standard

Only medical provider clears

Vital Signs

- Obtain history of accident

Mechanism of injury

Immediate neuro status

Treatment at the scene

Mode of transport

All vital data

- PMH

Pre-existing diseases

Medications

Allergies

- Foley Catheter

Atonic bladder

Prevent urinary distension and retention

- NGT

Decrease gastric distention

Prevent aspiration of vomitus

Consider PPI/ H2 Blocker for stress ulcer prevention

- How It Affects Each System

- Respiratory

- Respiratory complications closely correspond to level of injury

- Above C3

Total loss of respiratory muscle function; will arrest within minutes if not intubated

- C3 to C5

Respiratory insufficiency; loss of phrenic nerve innervation to diaphragm

Decreased strength in chest and abdominal wall

Require intubation

- Incomplete SCI

Respiratory function variable

- Cervical and thoracic injuries

Paralysis of abdominal muscles and often intercostal muscles causing ineffective cough and leading to risk for aspiration, atelectasis, pneumonia

Hypoventilation and impaired intercostal muscles lead to decreased vital capacity and tidal volume

Prevent hypoxemia that can worsen secondary injury

Maintain oxygen saturation >92%

- Assessment:

Dyspnea

pCO<sub>2</sub> greater than 20 mmHg above baseline (retaining)

Change in rate, rhythm, or quality of respirations

Note anxiety, cyanosis, altered mental status

ABG's

- Interventions:

Intubation → tracheostomy, if needed

Turning Q 2 hours to promote postural drainage, if possible

- Cardiovascular

- Injury above T<sub>6</sub> leads to dysfunction of sympathetic nervous system (SNS)

- Leads to neurogenic shock

Bradycardia

Hypotension

Peripheral vasodilation

Relative hypovolemia because of increased capacity of dilated veins

Reduced venous return decreases cardiac output, causing hypotension

- Identify cause of hypotension

Hemorrhagic from traumatic injuries?

Control any external bleeding

- Chronic Low Blood Pressure:

Improves when spinal shock dissipates

Spasticity can help keep BP increased

Postural hypotension

- DVT

Prevention techniques

- Urinary
- Urinary dysfunction occurs in most patients with SCI
- Neurogenic bladder

Bladder dysfunction related to abnormal or absent bladder innervation

No reflex detrusor contractions (flaccid, hypotonic)

Hyperactive reflex detrusor contractions (spastic)

Lack of coordination between detrusor contraction and urethral relaxation (dyssynergia)

- Urinary retention is common during spinal shock
- Bladder is atonic and over distended causing hydro-ureter and hydronephrosis, infections, & kidney damage
- Need foley cath or self-catherizing program to prevent distention & kidney damage

Scheduled intermittent self-caths (Q 4 hours)

- Complications:

Kidney stones:

Pt is not weight bearing → loses Calcium from bones → excreted via urinary tract

Prevention:

Increased fluids

Cranberry juice to keep urine acidic

Weight bearing ASAP

UTI

Can lead to renal damage, one of the leading causes of death

Prevention:

Aseptic technique during caths

Bladder training program

Increased fluid

Secure catheter properly

- Gastrointestinal
- Decreased GI motor activity leading to gastric distention and development of paralytic ileus

Gastric emptying may be delayed

Excessive release of HCl causing stress ulcers

Dysphagia may be present

- Intraabdominal bleeding

Difficult to diagnose; no pain or tenderness

Monitor BP, Hgb & Hct, and abdominal girth

- Neurogenic bowel

Impaired peristalsis; slow movement of stool

Defecation reflex damaged

Anal sphincter tone relaxed

Results in constipation, incontinence, and possible impaction, ileus, or megacolon

- Treatment:

Bowel Regimen and retraining

After spinal shock dissipates, the patient begins having reflex emptying of the bowel following a stimulus

Meal or abdominal effleurage

Daily rectal stimulant

Suppository, small-volume enema

Digital stimulation or manual evacuation

Establish routine patterns

Plan diet to avoid diarrhea/ constipation

Have patient sit on toilet or commode

Maintain privacy

*Goal: BM every day or every other day and will not be incontinent at other times!*

Integumentary

- Potential for skin breakdown

Decreased or absent sensation, leading to pressure injuries over bony prominences

Develop quickly

Risk of infection and sepsis

- Integumentary

- Lifelong risk

- Causes:

Inability to move

Sensory losses prevent sensations of discomfort

Cannot recognize need to be moved

Decreased circulation to areas below level of injury

- Decubitus Ulcers

Prevention: skin care measures are imperative!

Turn frequently!- Q 2 hours

Mobilize ASAP

Keep Dry

Careful and ongoing skin assessment

Prophylactic dressings to sacrum/ heels

Aquacel

- Poikilothermia: inability to maintain a constant core temperature

Interruption of SNS prevent peripheral temperature sensations from reaching hypothalamus

Decreased ability to sweat or shiver below the level of injury

More common with high cervical injury

- Musculoskeletal

- Lack of weight bearing causes loss of Ca<sup>+</sup>

Increased risk for fractures

Progressively, causes skeletal deformities, generalized osteoporosis, joint stiffness, muscle

weakness, contractures

- After spinal shock:

Lack of control from brain

Flaccid → spastic phase

Reflexes may be inappropriate and excessive

- After Spinal Shock:

Spinal Automatism: spinal reflex activities that automatically occur following severance of the cord

Persistent abnormal erection of the penis without sexual desire

Stimulation of skin of the lower abdomen or thigh may precipitate

Autonomic Bladder: stimulation of the lower abdomen causing reflex emptying of the bladder

Mass flexion

- Muscle Spasms

Can occur spontaneously or in response to stimuli and often continue until the stimulus is identified and removed

Mild spasticity can benefit: prevents atrophy & osteoporosis

Severe spasticity: detrimental effects exceed benefits

Difficult to stay in wheelchair

Improper positioning

Affects ADL's

Treatment:

Relaxation

PT

Medications: Dantrolene, Baclofen (pump), Robaxin, Valium, Botox injections

Drastic: Rhizotomy (cut abnormally firing sensory nerve)

- Contractures

Affect ADL's

- Neurogenic Heterotopic Ossification

Osteogenesis in a part of the body which does not normally form bone, such as soft tissue

Cause is unknown

30% severe SCI patients develop

Found below level of injury

- Signs and Symptoms:

Redness, warmth, swelling, decreased ROM

Elevated alkaline phosphatase

X-ray: shows osseous formation

- Treatment:

No definitive

Position change, splinting, ROM,

Indocin (NSAID)

Didronel: regulates bone metabolism, slows new bone formation

- Pain

Often have pain present at the level of the injury, which radiates along the spinal nerves originating in that area

I.E.: CP may occur following thoracic injuries, leg pain may follow lumbar injuries

Treatment:

Analgesics

Opiates

Sedatives

Antispasmodics

Pregabalin

Tricyclic antidepressants

- Metabolic Needs

- SCI leads to increased metabolism and increased protein breakdown

Decreased lean body mass, muscle atrophy, and weight loss

- Nutritional support

Start early; enteral or parenteral diet to address caloric, protein, and micronutrient needs

Adequate nutrition

Prevents skin breakdown

Reduces infection

Decreases muscle atrophy

- Psychological

Consider the age group

Loss of body image

Dependency

- Shock/ Denial

Disaster has struck

Reaction is disbelief

Denial is main defense

Other may go along with denial

- Anger

Reality hits hard

Lashes out, pushes blame

Verbal assaults

Be prepared:

Accept the patient, but not their behavior

Try to get the patient involved in their own care

Family needs support

Staff will suffer during this phase

- Factors that affect progress:

Previous personality difficulties, alcohol/ drug use, importance of appearance, livelihood (required mobility, athlete?)

- Depression:

May exhibit regression or severe crying

Suicidal but unable to complete

Allow to vent feelings

Family support

- False Hopes of Cure

Builds unrealistic hopes for new discovery

- Acceptance:

Comes with mature feelings of interdependence

Functions within limits of disability

- Rehabilitation:

Most therapeutic setting is an environment that assesses, treats, and empowers the patient and family

Recovery and limitation depends on the level of injury and motivation

- Sexuality

- Extent of injury depends on level of function:

Affected:

Erections (psychogenic)

Ejaculation

Orgasm

Fertility status

Males: Testicular atrophy → decreased spermatogenesis

Females: precipitous delivery risk, autonomic dysreflexia

Counseling

Wait until person is ready

Involve the family (as appropriate)

Encourage open communication with the partner

- Treatment Measures
- Nonoperative
- Stabilization of injured spinal segment and decompression

Traction or realignment

Eliminates damaging motion

Prevent secondary damage

- Early realignment of unstable fracture-dislocation

Closed reduction through craniocervical traction

- High cervical SCI (most complex)
- Immobilization

Maintain neck in neutral position

Keep body in correct alignment

Logroll to prevent movement of spine

Closed reduction with skeletal traction

Crutchfield or Gardner-Wells tongs or halo

Maintain traction at all times

Disadvantage: pin displacement

Hold head in neutral position and get help

- Cervical Traction

Closed reduction with skeletal traction

Goal: spinal reduction

Monitor with x-ray, neurologic and pain assessments if alert; serial x-rays if comatose

Surgery: cervical fusion or other stabilization procedure

Postoperative: hard cervical collar or sternal-occipital-mandibular immobilizer brace

Halo vest

Spinal fractures with or without acute SCI; no surgery but immobilization needed

Allows movement and ambulation while bones fuse

Contraindicated with:

Ligament instability, severe cervical deformity, morbid obesity, older age, cachexia, or

noncompliance

Patient and caregiver teaching

Used following acute SCI to manage instability and decompress the spinal cord

Reduces secondary injury and improves outcomes

- Pharmacology
- Use of methylprednisolone to treat acute SCI—mixed evidence

Not recommended by American Association of Neurological Surgeons and Congress of Neurological

Surgeons

Not approved by FDA

AOSPine Guidelines (2017) suggest high-dose within 8 hours of SCI

- VTE prophylaxis

Low-molecular-weight heparin or low-dose heparin (unless contraindicated)

- Vasopressor agents

Maintain mean arterial pressure above 85 to 90 mmHg (e.g., phenylephrine, norepinephrine)

Improve spinal cord perfusion

Significant risk of complications

Ventricular tachycardia,  $\uparrow$ troponin, metabolic acidosis, and atrial fibrillation

Consider level of injury, age, and co-morbidities

- Phenomenas of SCI
- Autonomic Dysreflexia

Hyper-reflexia

Occurs in persons with SC lesions above most of the sympathetic nerve outflow of the cord (T6)

Occurs after spinal shock and return of reflex activity

Occurs in response to visceral stimulation

- **Pathology**

Stimulation of the sensory receptors below the level of the cord lesion

The intact autonomic system below the level of the lesion responds to stimulation with reflex

arterial vasoconstriction =  $\uparrow$  BP

High BP is sensed by the baroreceptors in the carotid sinus and the aorta response is  $\downarrow$  HR

Visceral and peripheral vessels do not dilate because the efferent (motor) impulses cannot pass through the cord lesion.

Most common cause is **distension of the bladder** or rectum; other causes can be stimulation of the skin or pain receptors.

- **S & S**

Life Threatening!!  $\Rightarrow$  requires immediate intervention

**Headache!**

Flushing & diaphoresis

**Skin becomes pale below the level of the lesion!**

Bradycardia

Piloerection (Goose Bumps)

Nasal congestion

Vision changes

Anxiety

o **Treatment:**

Elevate HOB 45 degrees

Monitor BP every 3-5 minutes

Remove the cause:

TEDS

Bladder distention  $\rightarrow$  straight cath, catheter kinked?

Abdominal Binder

Notify Provider

Medicate prn

Teach the patient and family to recognize signs and symptoms

- Nursing Prioritization

Your SCI patient develops sudden onset of diaphoresis, vision changes and a "pounding" headache.

Upon your arrival, what are the priority interventions?

1. Check patient's blood pressure

2. Elevate HOB to 45 degrees
3. Remove the stimulation
  1. i.e. catheter kinked, distended bladder/ rectum, TEDS
4. Notify Provider
  1. May need to administer antihypertensives
5. Continue to reassess blood pressure Q 3-5 minutes until symptoms resolved

- Horner's Syndrome
  - Paralysis of cervical sympathetic nerve trunk
- Signs & Symptoms:
  - Ptosis of eyelid
  - Loss of sweating over the affected side of face
  - Recession of eyeball into orbit
- Rehabilitation
- Overall goals
  - Maintain optimal level of neurologic functioning
  - Have minimal to no complications of immobility
  - Learn new skills, gain new knowledge, and acquire new behaviors to care for self or direct others to do so
  - Return to home at optimum level of functioning
- Interprofessional team effort
  - Rehabilitation nurses, HCPs, PT, OT, speech therapists, vocational counselors, psychologists, therapeutic recreation specialists, prosthetists, orthotists, case managers, social workers, and dieticians
- Patient expected to be involved in therapies and learn self-care
- Progress can be slow
- Can be very stressful
- Nurses provide frequent encouragement, specialized care, patient and caregiver education; and help coordinate efforts of team
- Spinal Cord Tumors, Abscesses, & Vascular Disorders
- Spinal Cord Tumors
- Cause significant spinal compression and neurologic dysfunction
- Classified as:
  - Primary: arise from some part of the spinal cord, dura, nerves, or vessels
  - Secondary: primary growths from other places in the body that have metastasized to the spinal cord
- Etiology and pathophysiology
- Further classification
  - Extradural—outside the dura
  - Intradural-extramedullary—between the spinal cord and dura
  - Intramedullary—within the substance of spinal cord
- Most tumors are slow growing
- Symptoms arise from
  - Compression and irritation of nerve roots
  - Displacement of the spinal cord
  - Gradual obstruction of blood supply
- Removal of tumor may lead to possible complete restoration of function
- Clinical manifestations

Location and extent of tumor determine the severity and extent of motor and sensory problems

Most common (early): back pain or pain radiating along compressed nerve root

May worsen with activity, coughing, straining, and/or lying down

Increasing clumsiness, weakness, and spasticity

Paralysis

Coldness, numbness, and tingling in one or more extremities

Incontinence, constipation, and urgency

- Diagnostic studies

Spinal x-rays

MRI

CT scan

CT myelogram

CSF analysis

- Nursing and interprofessional management

Spinal cord compression is an emergency to relieve ischemia

Corticosteroids to relieve edema

Surgical indications

Spinal cord compression

Pathology

Determine appropriate treatment

- Nursing and interprofessional management

Primary tumor removal; goal is cure

Metastatic tumors; palliative

Goal: restore or preserve function, stabilize the spine, alleviate pain

Chemotherapy and/or radiation may also be used

- Nursing and interprofessional management

Treatment goals

Relieve pain with analgesia

Maximize neurologic status

Prognosis varies

- Spinal Cord Abscesses

- Rare

- Cause

Staph and Strep organisms most often cause from open trauma of spinal cord or osteomyelitis

- S&S

S&S of infection – fever, chills, erythema, warmth over SC region

- Dx:

Myelogram, CT Spine, MRI Spine

- Tx:

Goal: halt the growth of the organisms and restore neuro function

IV Antibiotics

Steroids

Decompressive laminectomy

Possible reoccurrence, need follow-up!

- Vascular Disorders

- Possible to have rupture, thrombosis, or embolism of SC vessels- usually secondary to meningitis or compression
- Treatment and prognosis vary
- Consult neurosurgeon