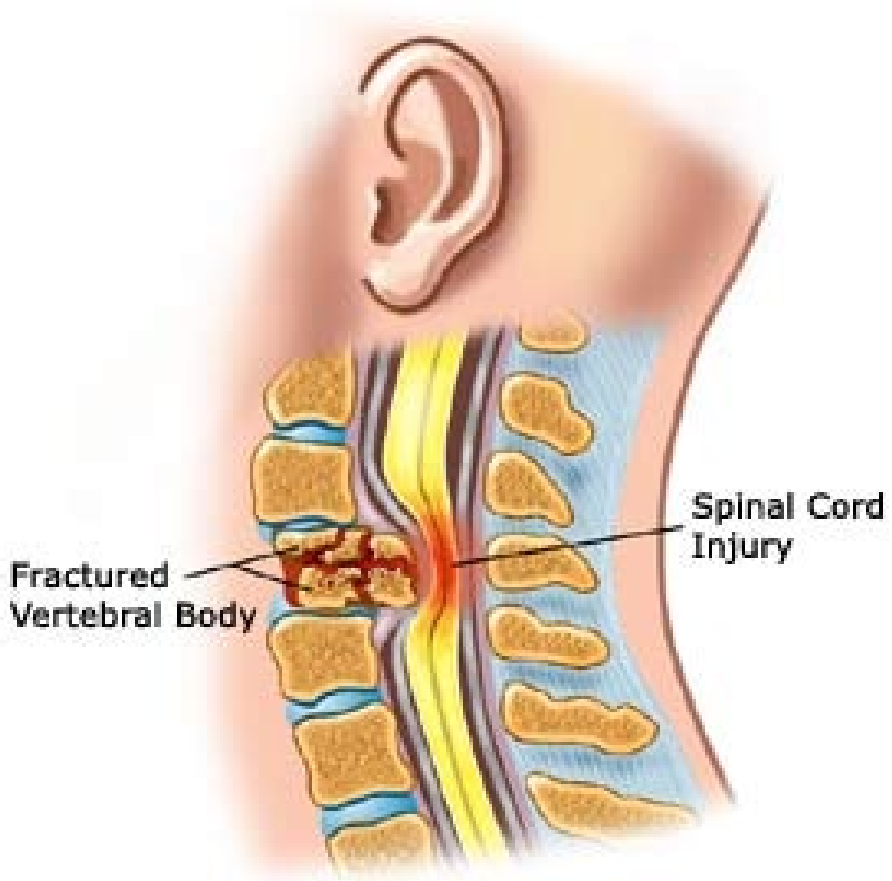
C13.4

Spinal Cord Trauma

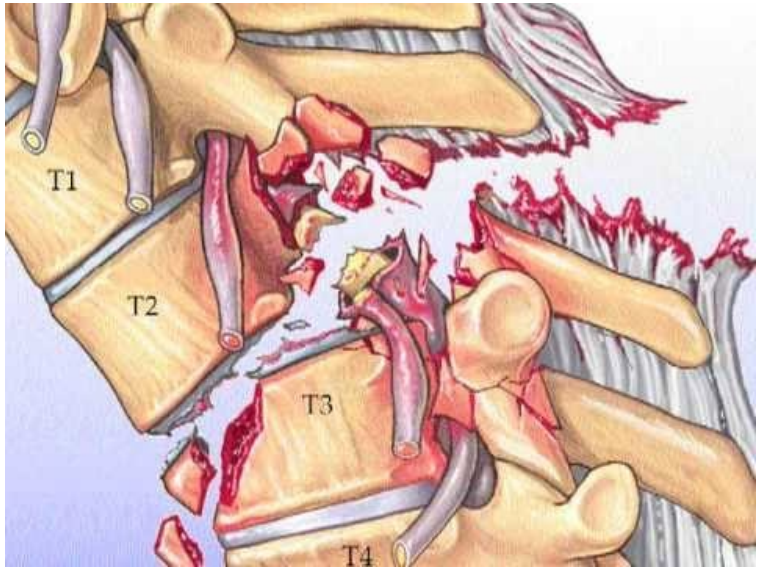
Complete Transection of Spinal Cord



* Taken from Anatomy and Physiology the Unity of Form and Function by Saladin

In United States 10 to 20 thousand people become paralyzed annually by spinal cord trauma (vertebral fractures)

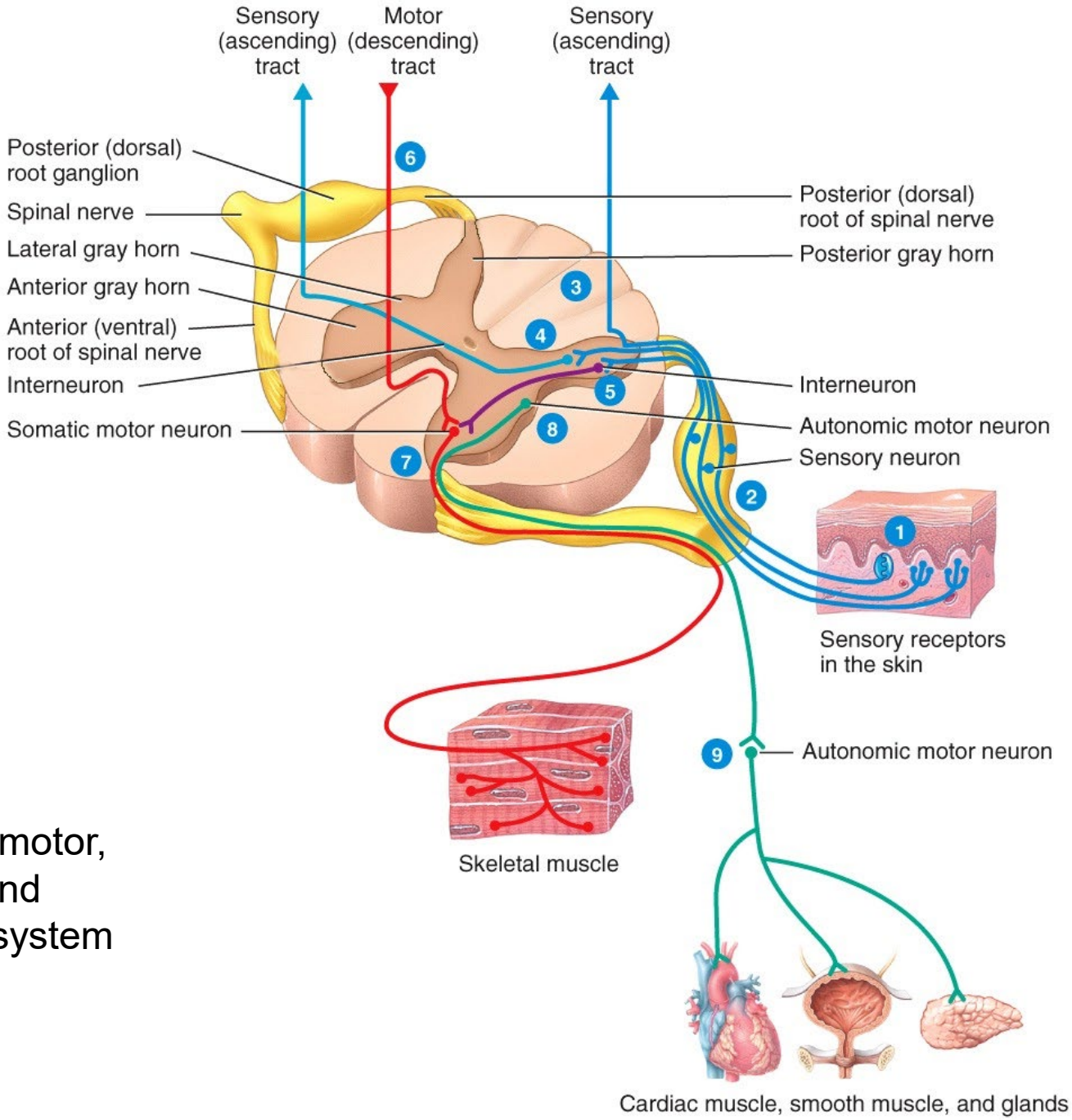
Males mostly between 16 to 30 yrs old /// associated with high risk behavior



50% of these accidents involve motorcycles and auto /// 18% sports

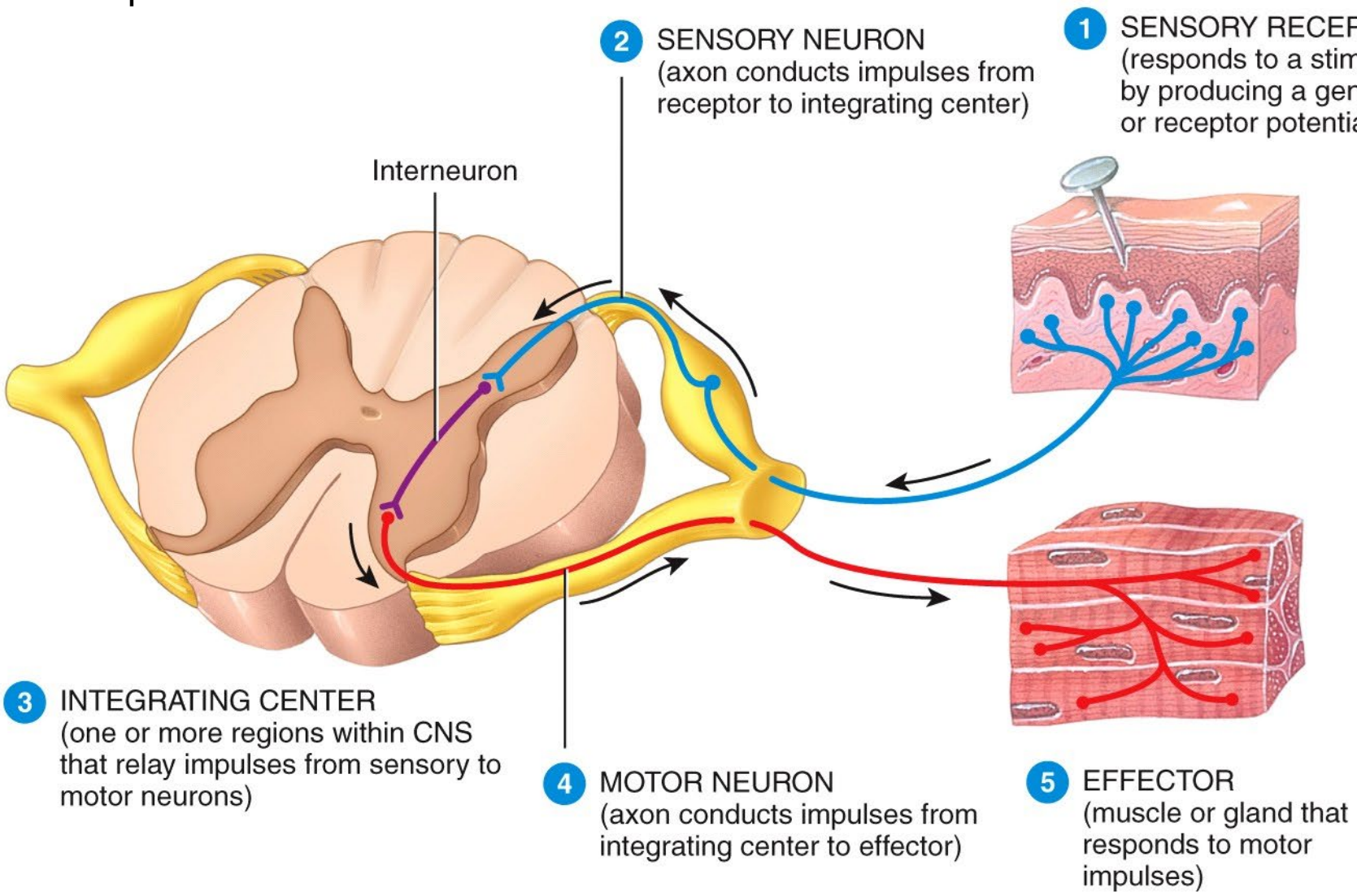
Elderly people above average because of falls

Nerve Fiber Projections from Spinal Cord



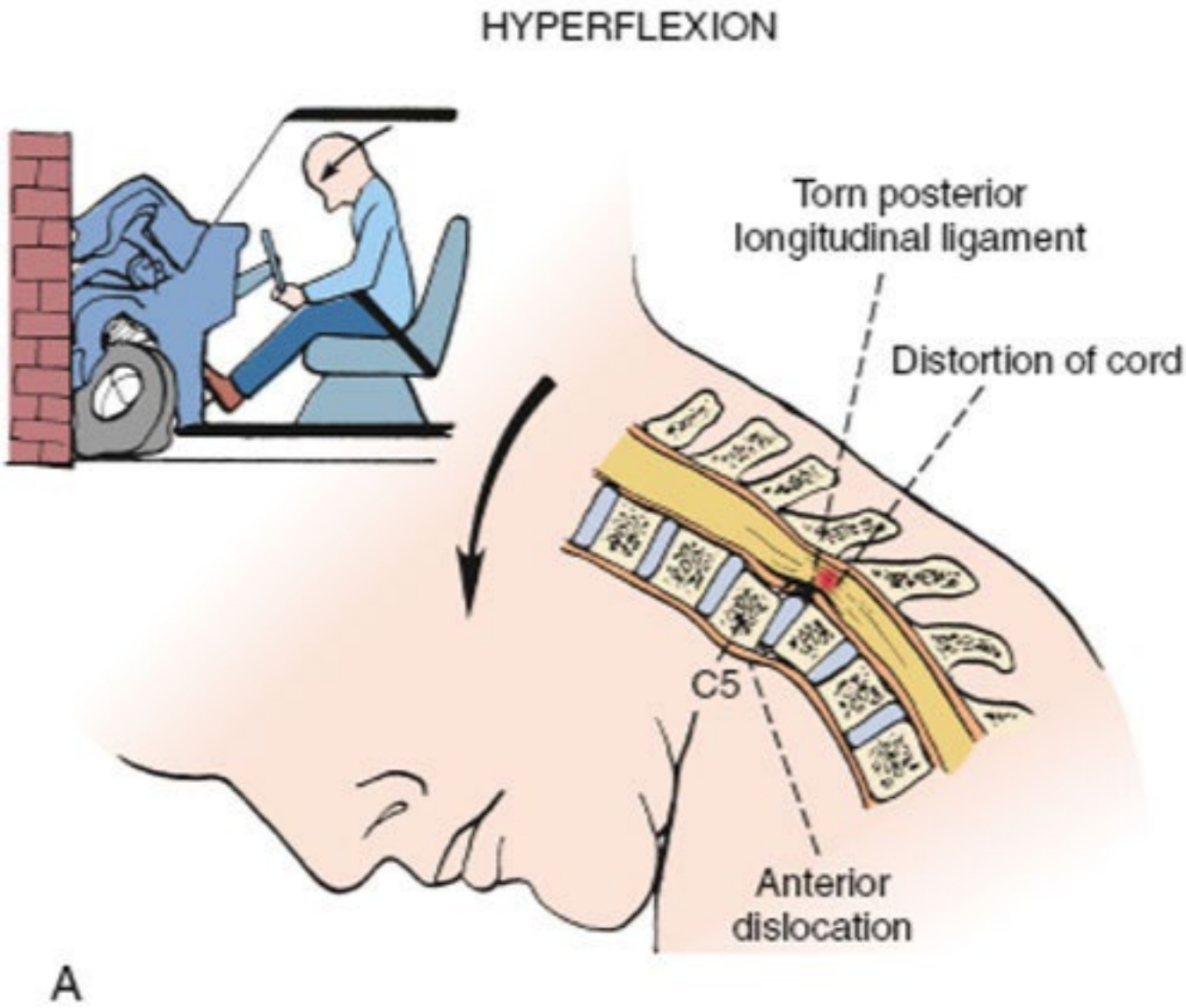
Review pathways for motor, somatosensory, and sympathetic nervous system fibers.

Spinal Cord Reflex



Note: this is a polysynaptic somatic reflex. Deep tendon reflexes are monosynaptic.

Types of Spinal Cord Injuries



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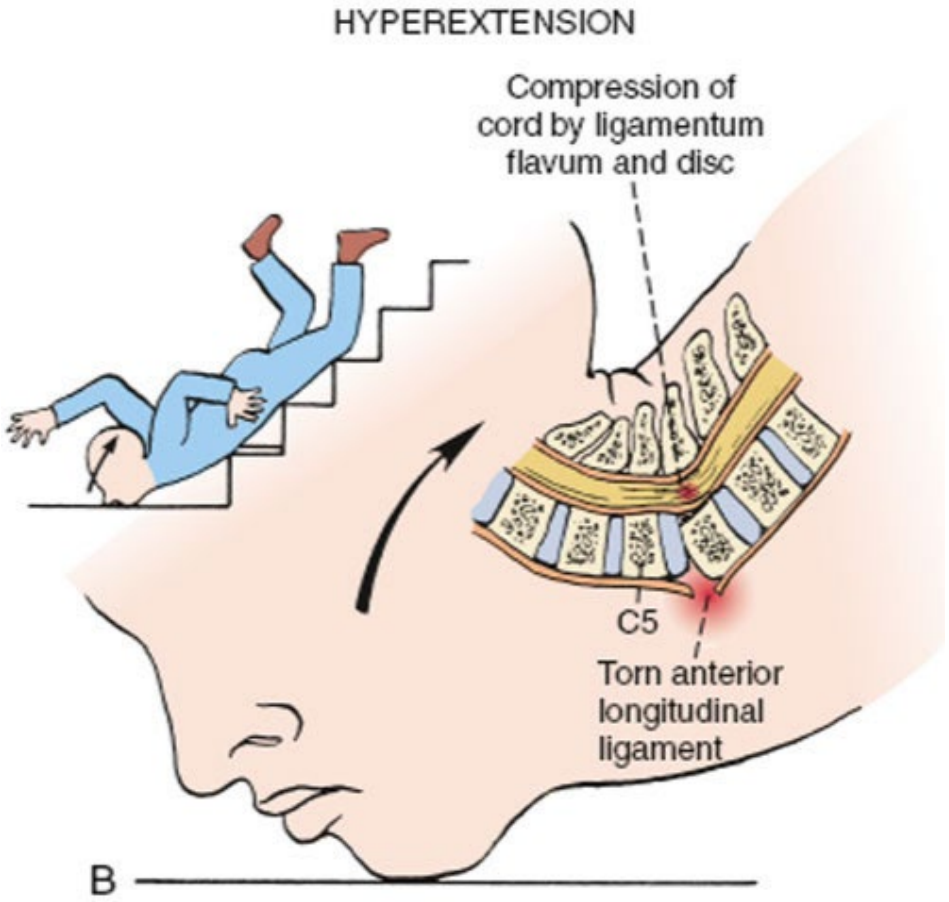
Types of Spinal Cord Injuries



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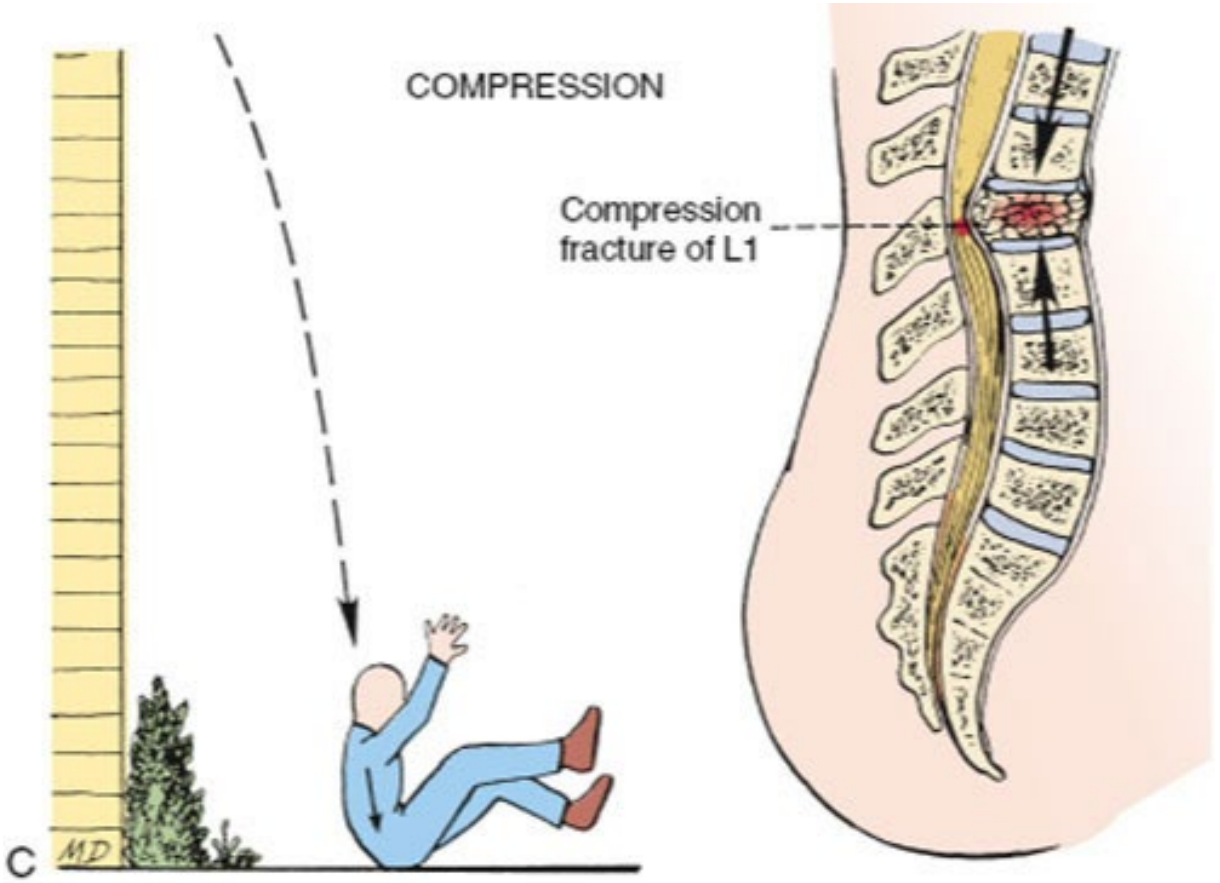
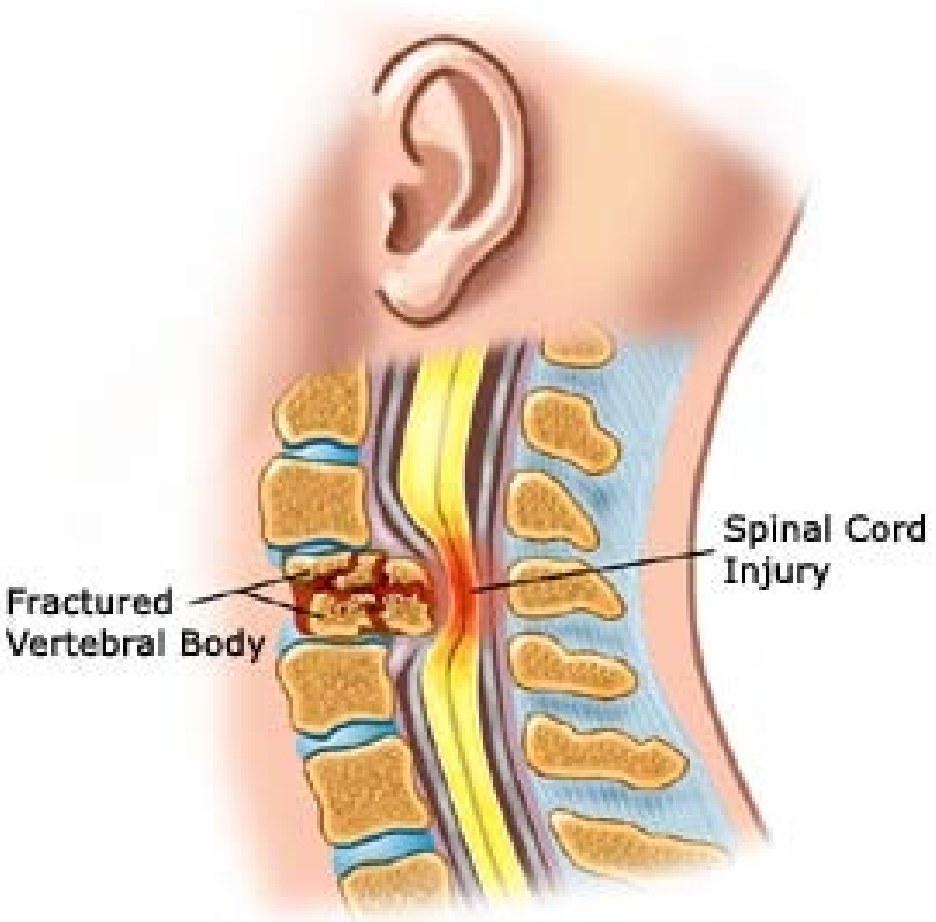
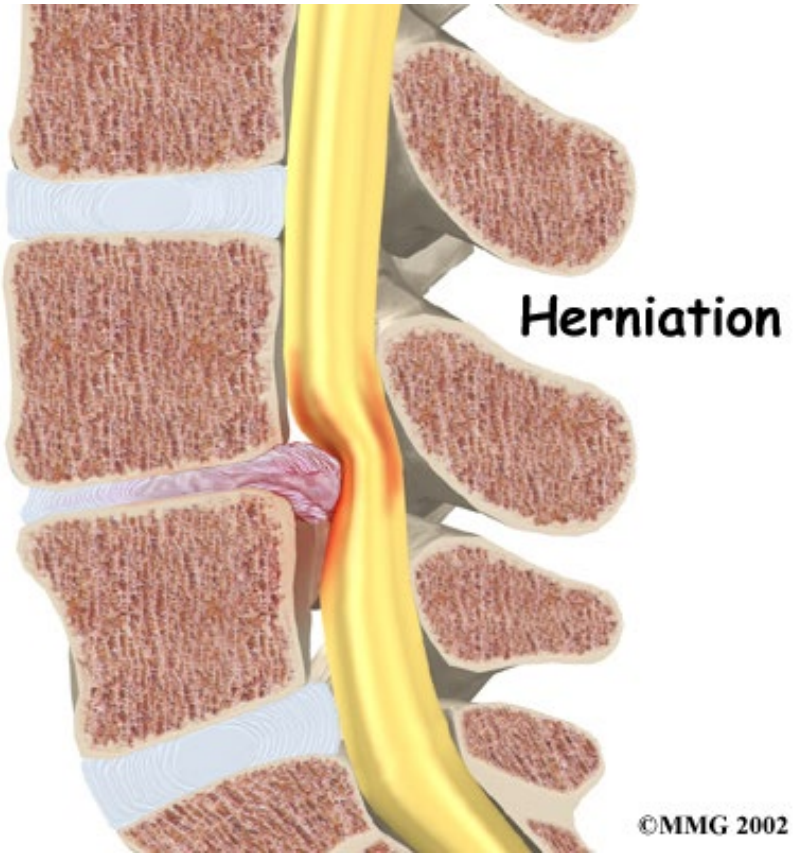
Types of Spinal Cord Injuries



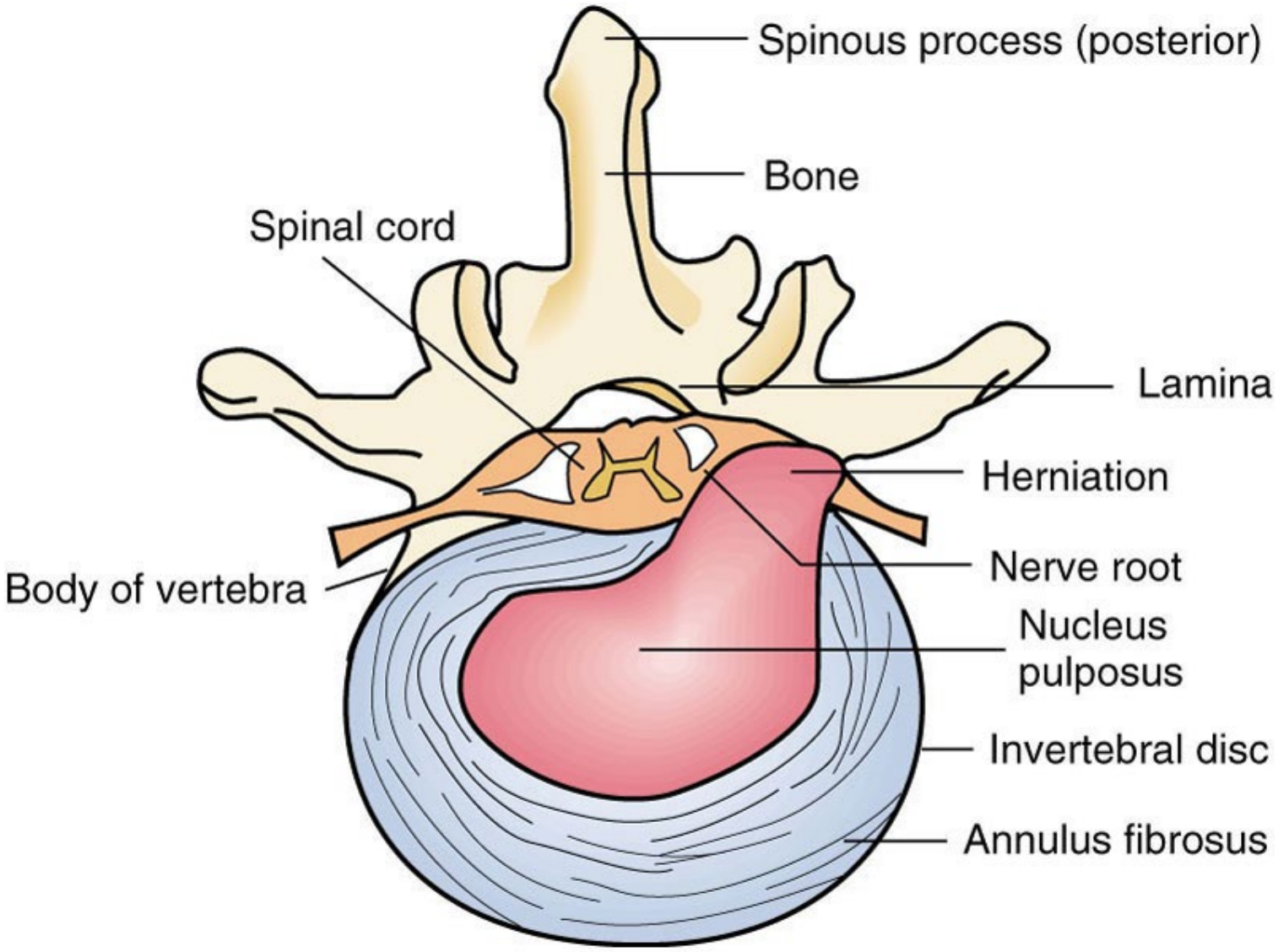
From Thibodeau GA: *Anatomy and Physiology*, St. Louis, 1987, Mosby.

Fractured Bone VS Disc Herniation





Herniated Intervertebral Disk



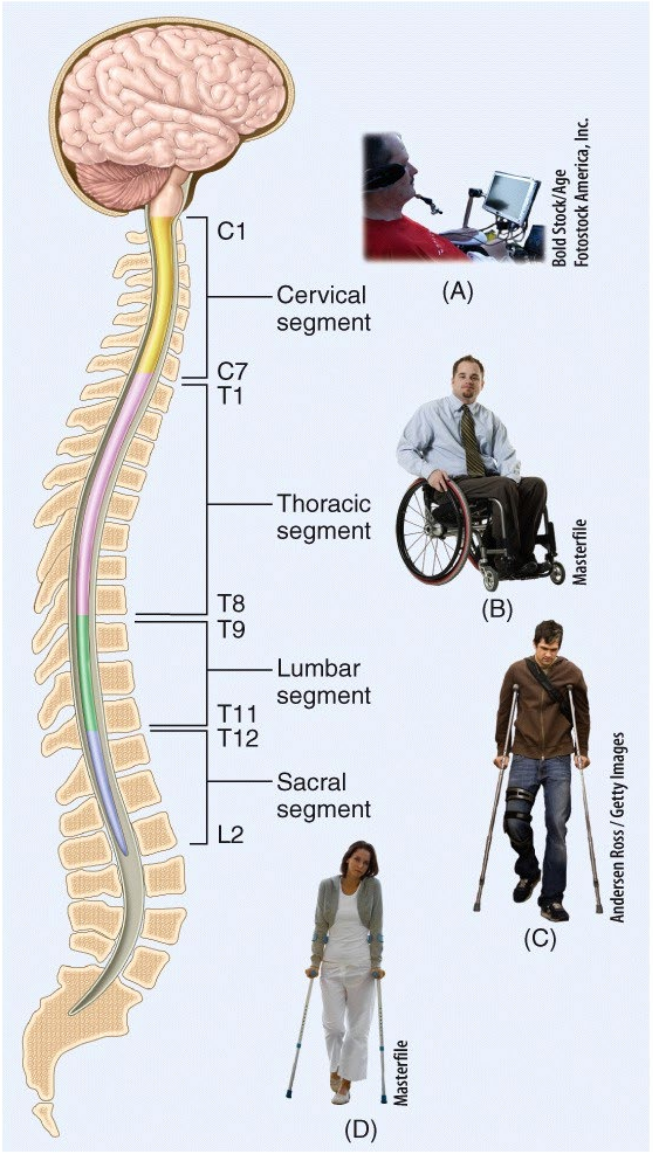
Effect of Injury

After complete transection there is complete loss of motor control at and below the level of injury

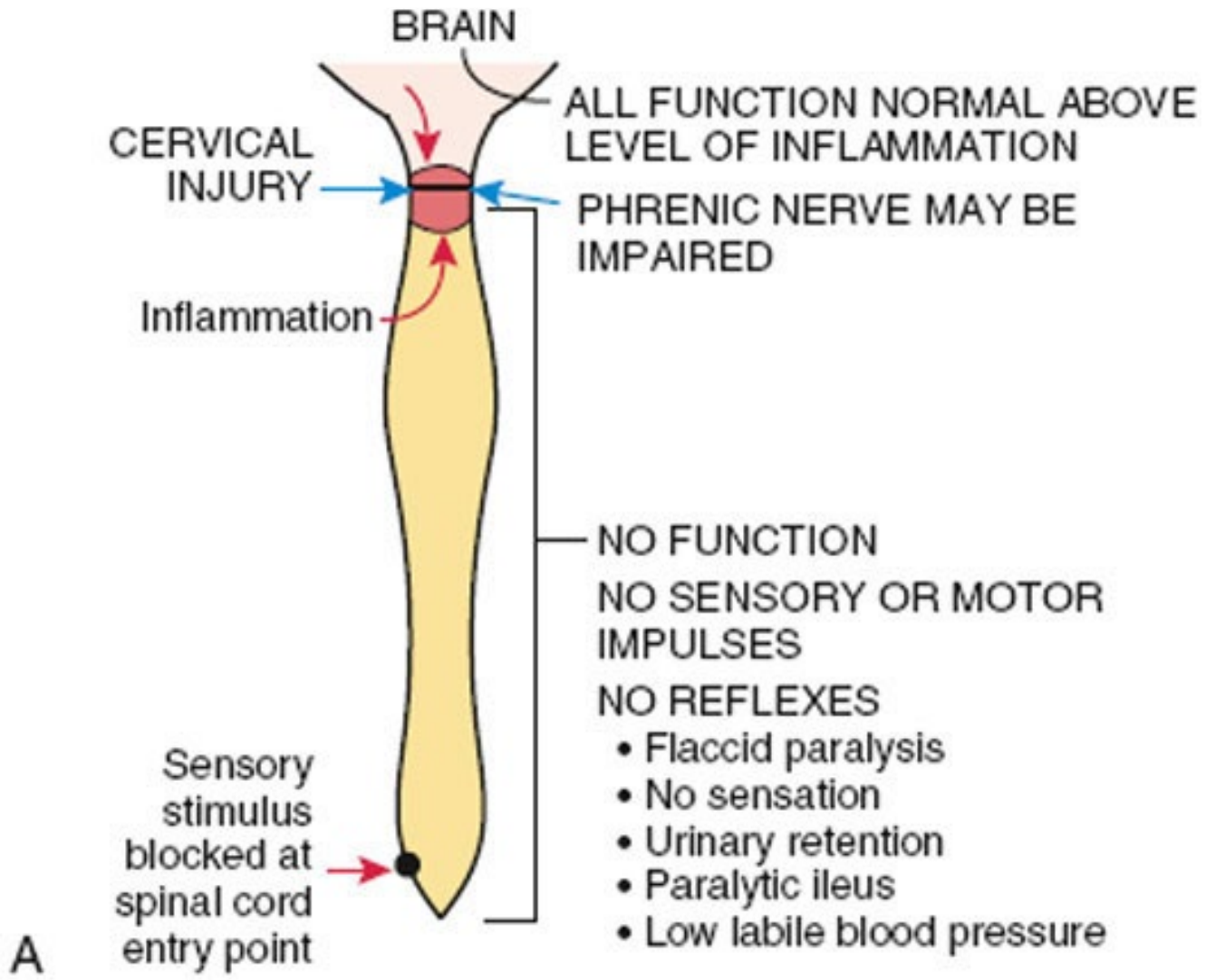
If injury is superior to C4 then there is a threat of respiratory failure

Lose all sensation from the level of injury and below

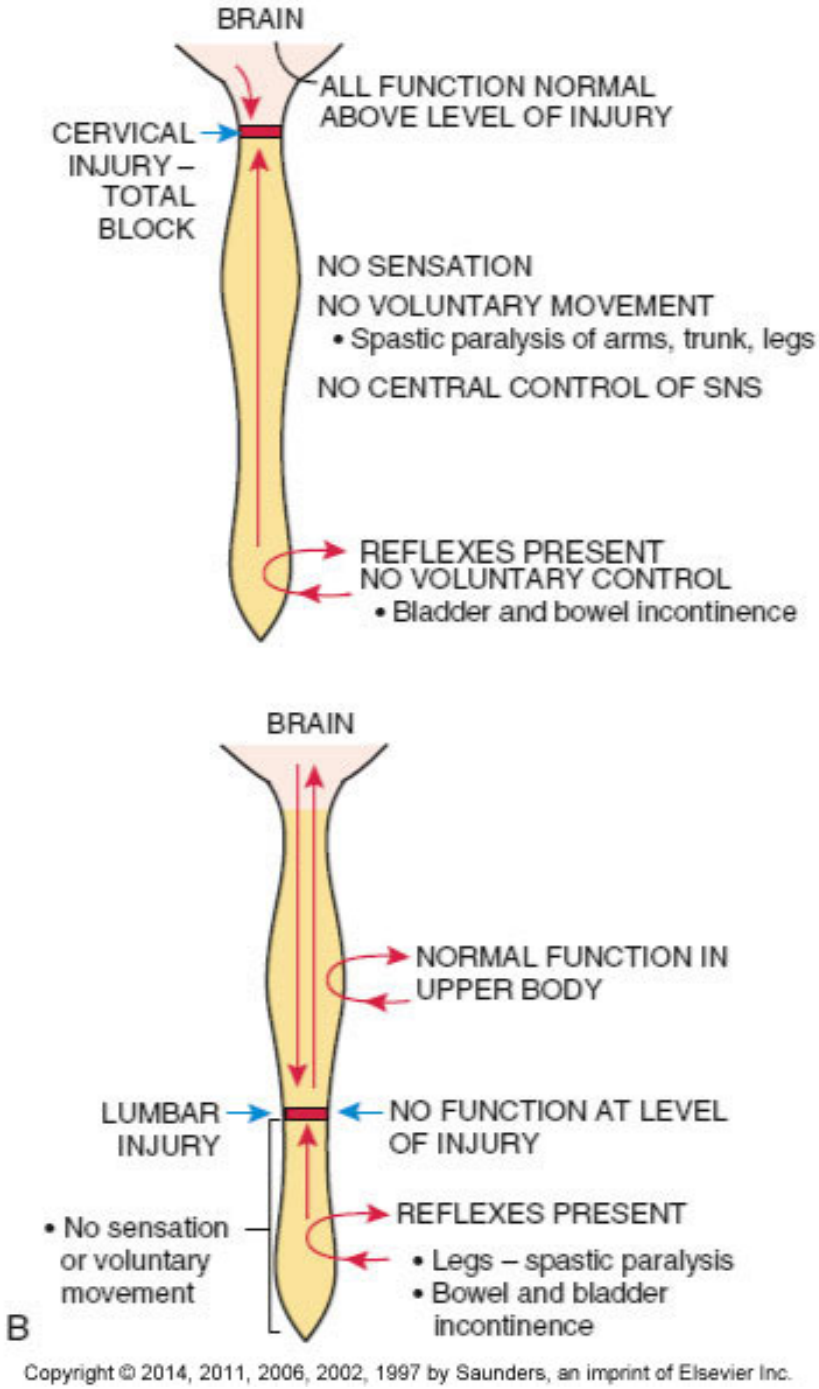
Some patients temporarily feel burning pain within one or two dermatomes of lesion level.



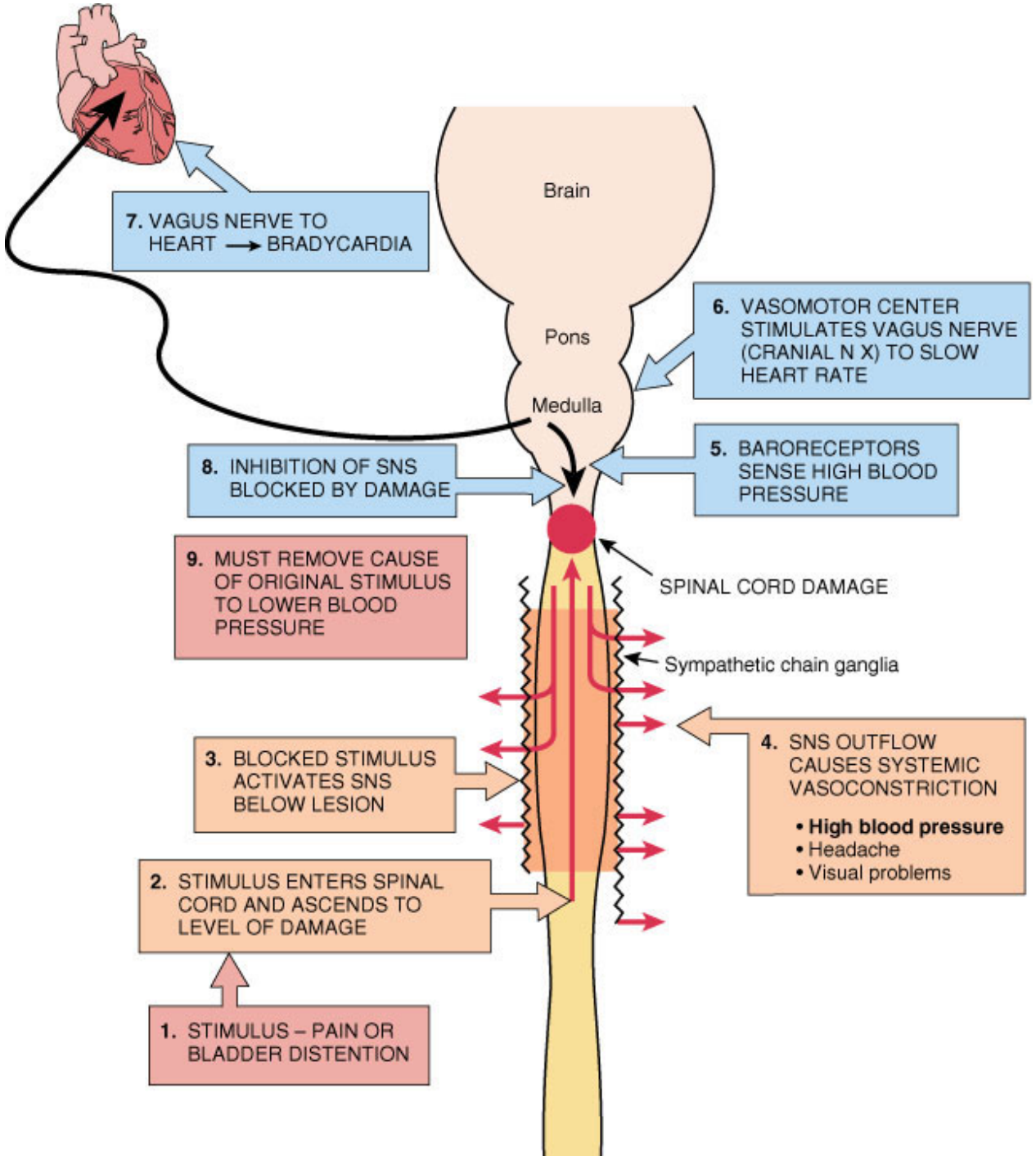
Events Immediately Following Spinal Cord Transection in Cervical Area



Effects of Spinal Cord Damage After Initial Paralysis and As Reflexes Return (Hyperreflexia)

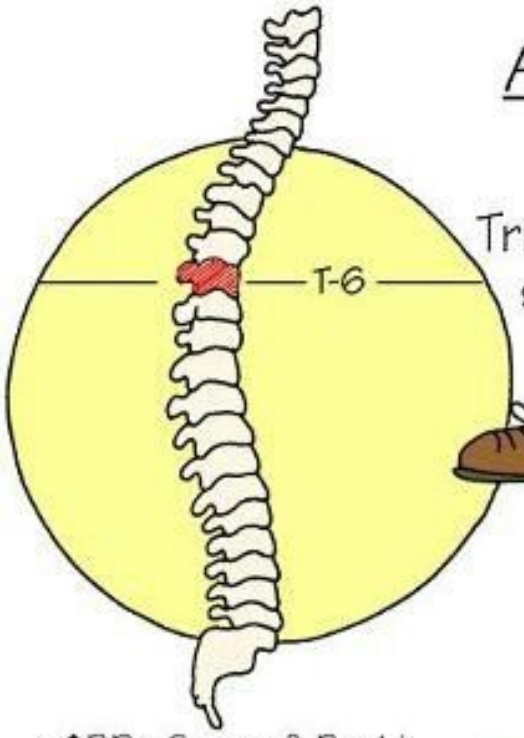


Autonomic Dysreflexia Following Spinal Cord Damage



AUTONOMIC DYSREFLEXIA...

(Spinal Cord Injury At T-6 Or Higher)



Triggered by sustained stimuli at T-6 or below from:

Restrictive Clothing

Full Bladder or UTI

Pressure Areas

Fecal Impaction

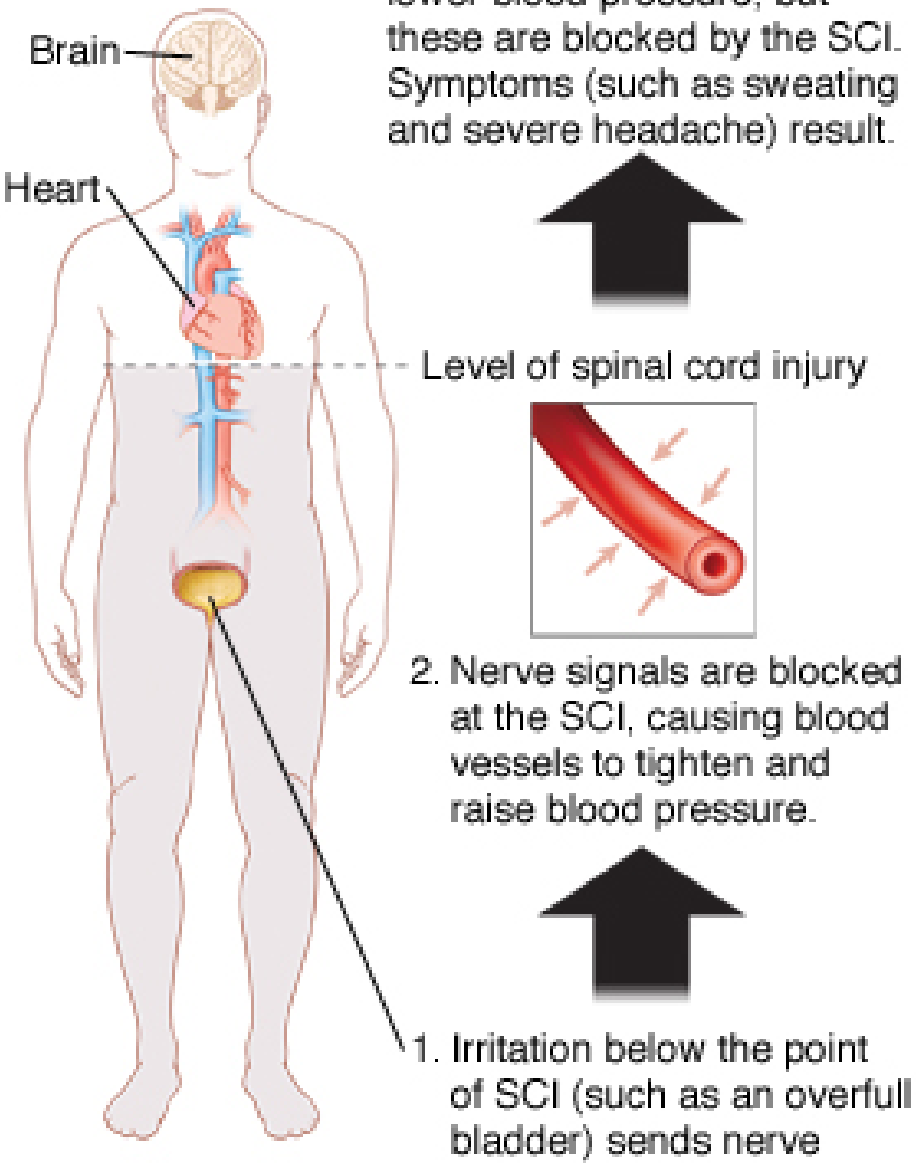
* ↑BP - Severe & Rapid
* Flushed Face
* Headache
* Distended Neck Veins
* ↓Heart Rate
* ↑Sweating

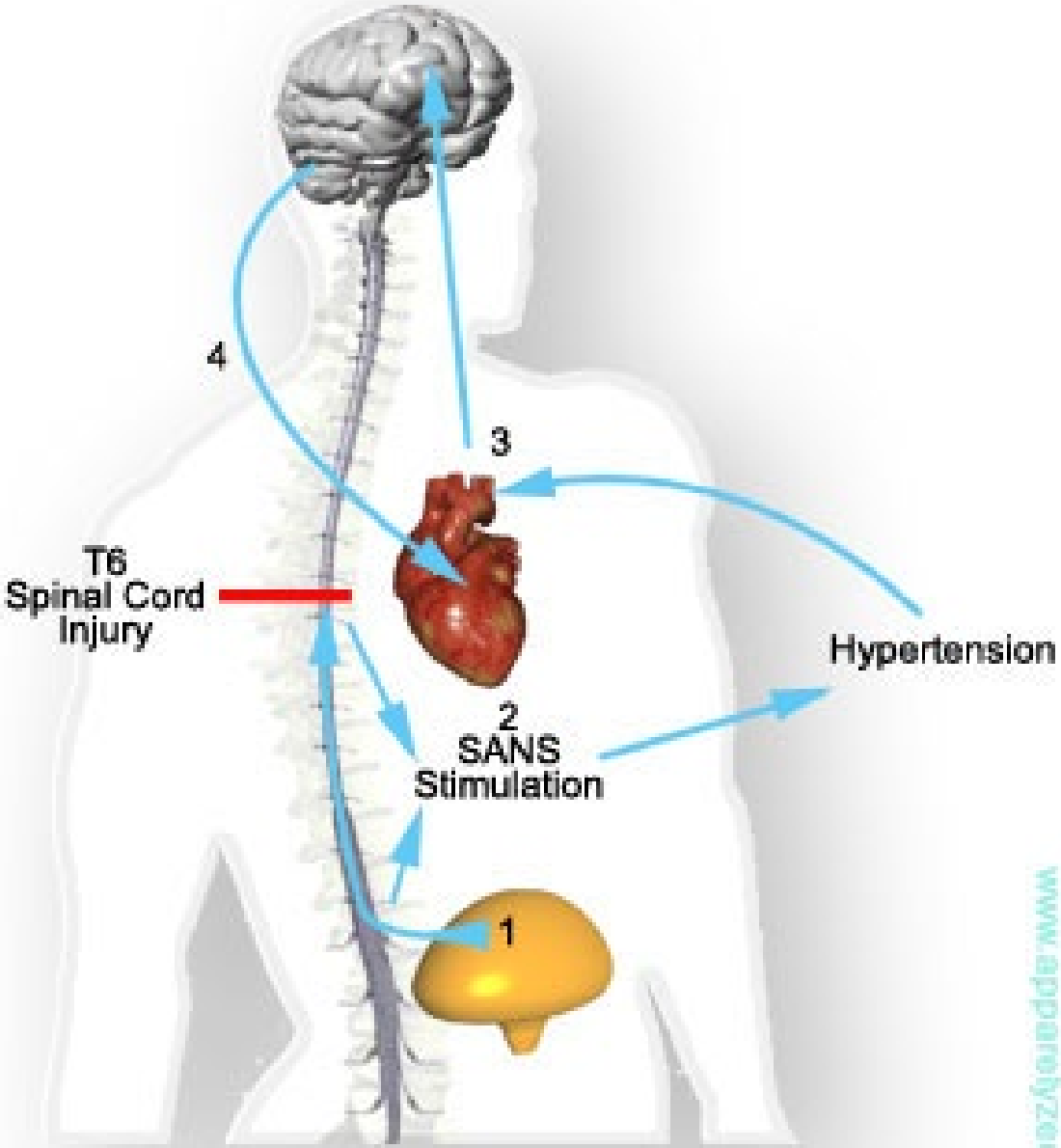
Vasodilation Above

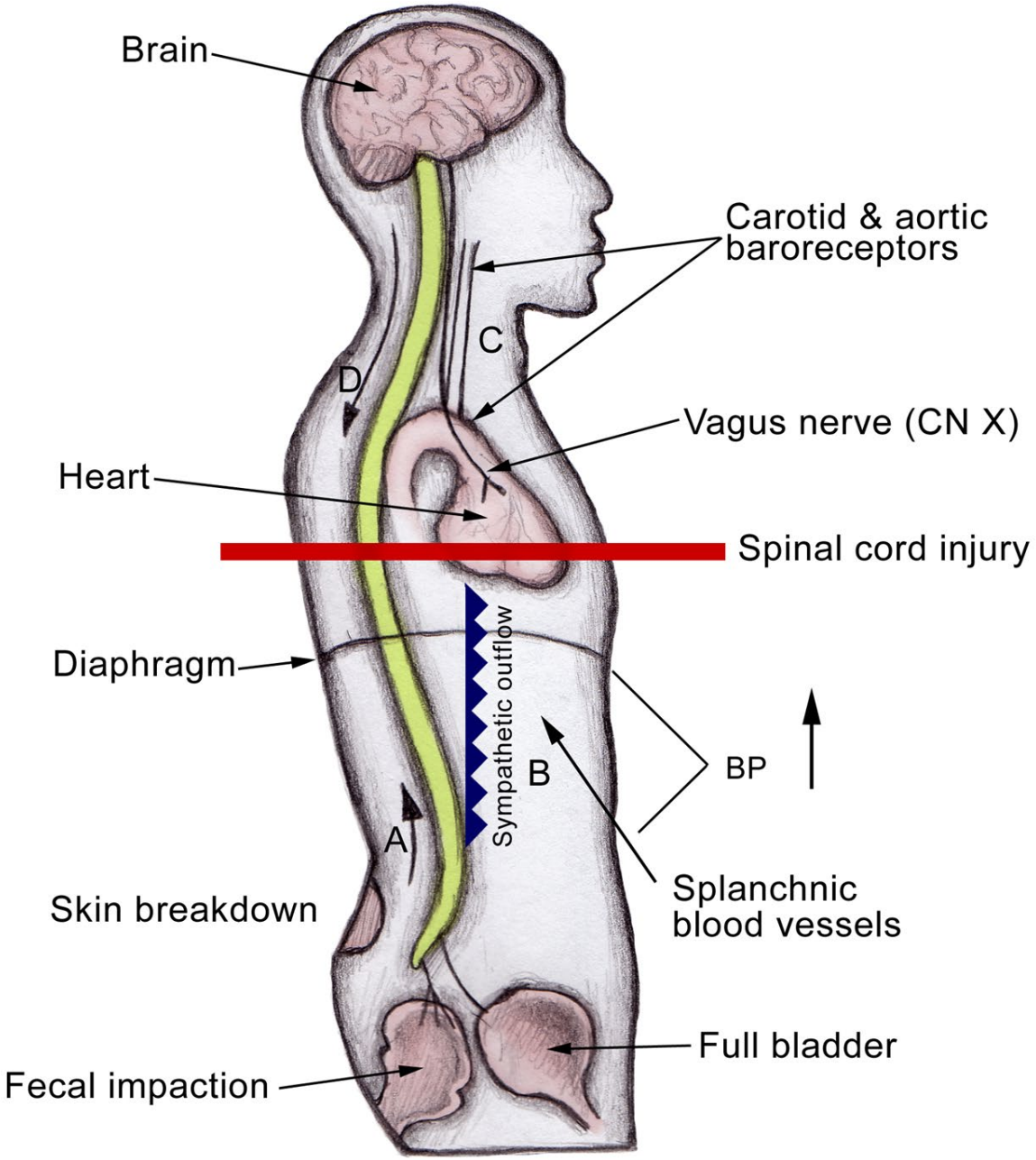
- - Level of Injury - -

Vasoconstriction Below Level of Injury

* Pale
* Cool
* No Sweating


3. The brain sends signals to lower blood pressure, but these are blocked by the SCI. Symptoms (such as sweating and severe headache) result.
Brain
Heart
Level of spinal cord injury
2. Nerve signals are blocked at the SCI, causing blood vessels to tighten and raise blood pressure.
1. Irritation below the point of SCI (such as an overfull bladder) sends nerve signals up the spine.

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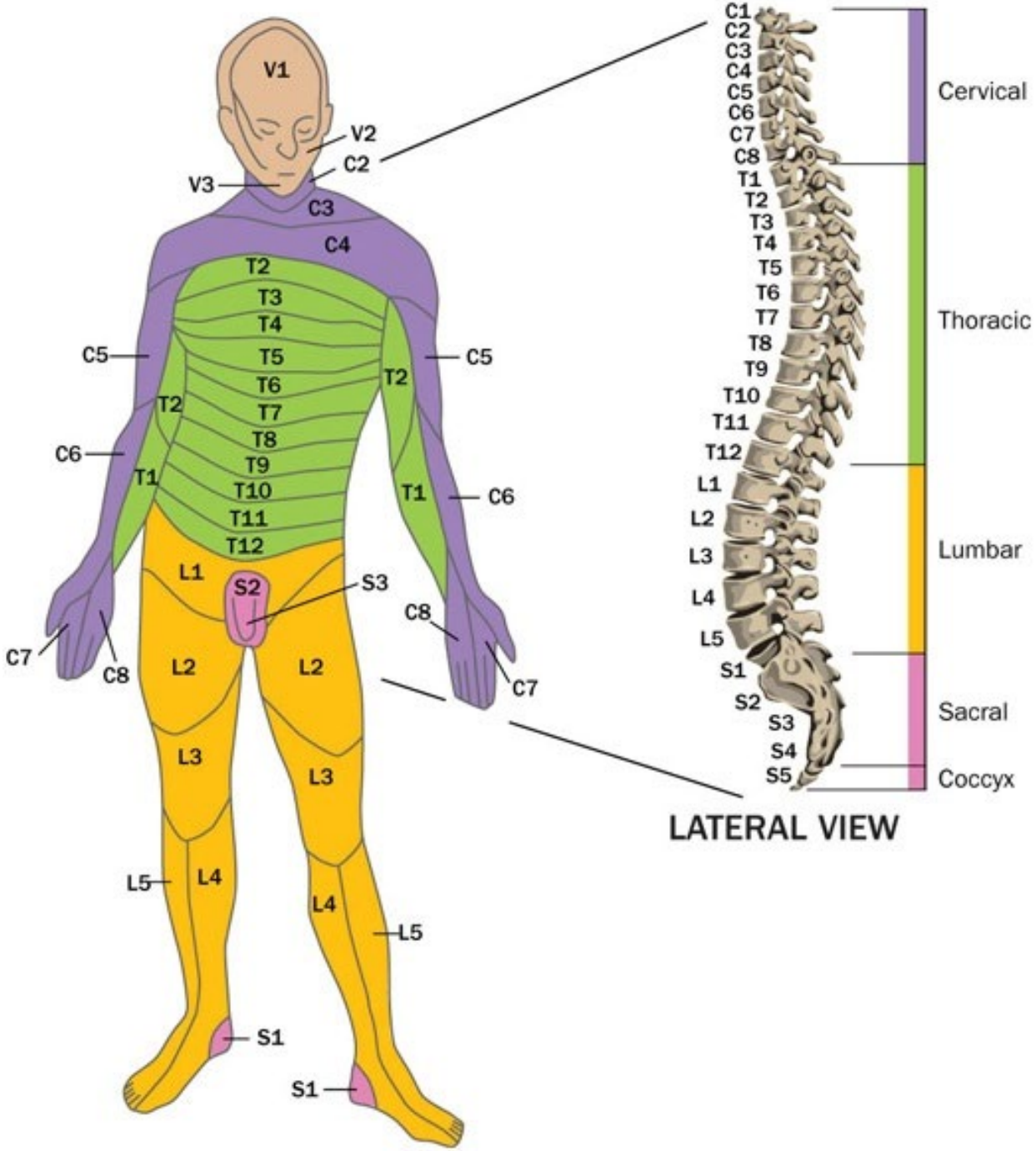
Carotid & aortic baroreceptors
C
D
Vagus nerve (CN X)
Heart
Spinal cord injury
Diaphragm
Sympathetic outflow
B
BP
A
Skin breakdown
Splanchnic blood vessels
Full bladder
Fecal impaction


V1
V2
V3
C2
C3
C4
T2
T3
T4
T5
T6
T7
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T9
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T11
T12
C5
C5
T2
T2
C6
T1
T1
C6
L1
S2
S3
C7
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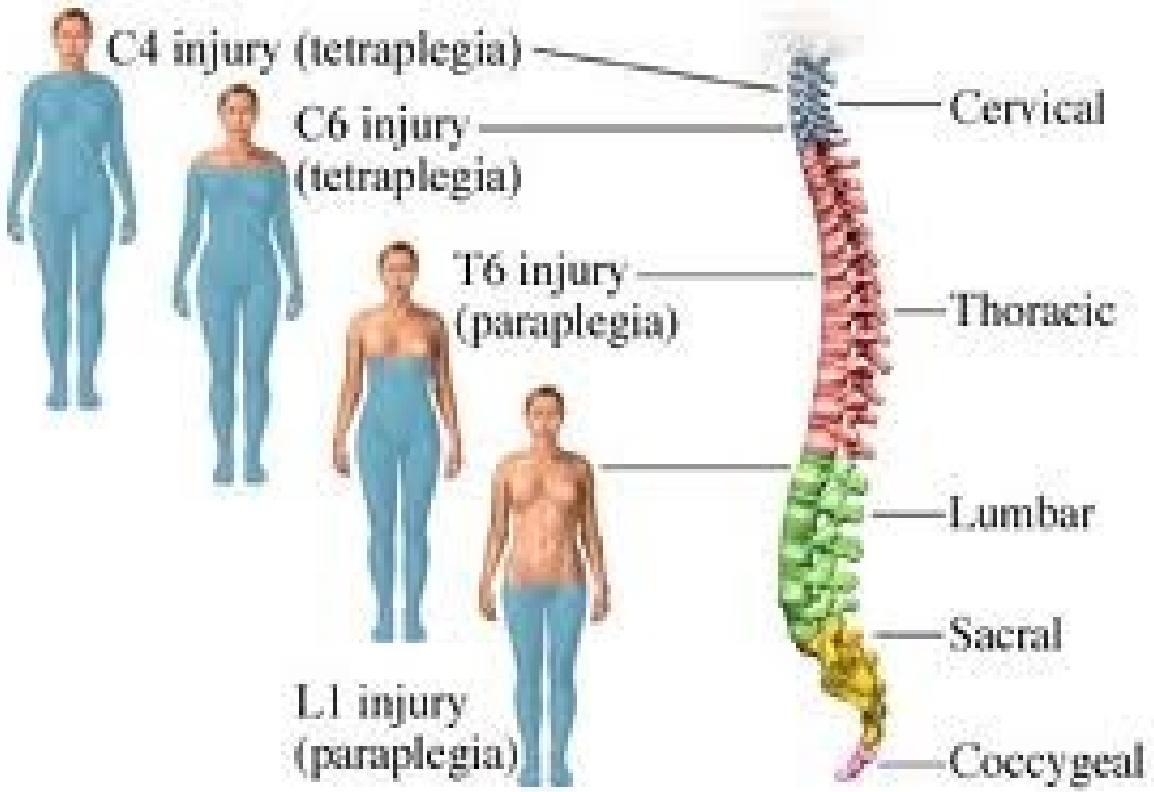
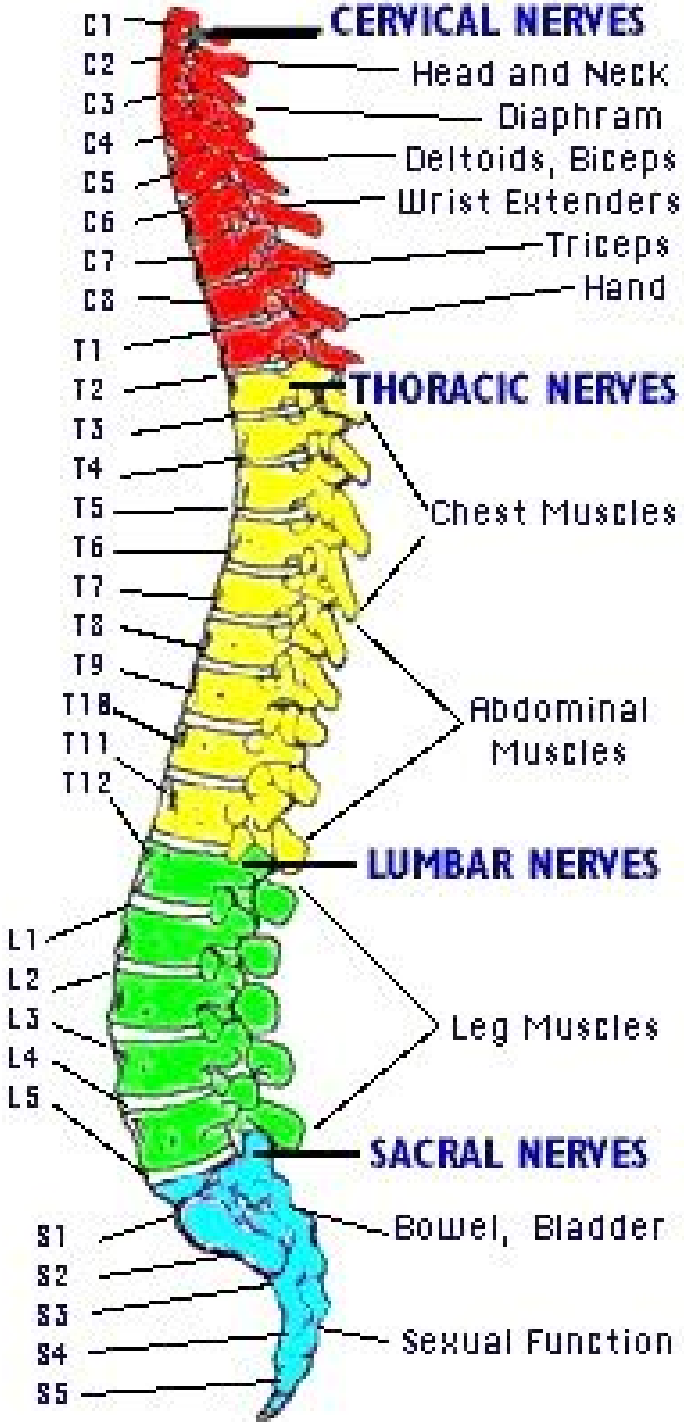
S4
S5
Cervical
Thoracic
Lumbar
Sacral
Coccyx
LATERAL VIEW


C4 injury (tetraplegia)
C6 injury (tetraplegia)
T6 injury (paraplegia)
L1 injury (paraplegia)
Cervical
Thoracic
Lumbar
Sacral
Coccygeal


C1
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C7
C8
T1
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T8
T9
T10
T11
T12
L1
L2
L3
L4
L5
S1
S2
S3
S4
S5
CERVICAL NERVES
Head and Neck
Diaphram
Deltoids, Biceps
Wrist Extenders
Triceps
Hand
THORACIC NERVES
Chest Muscles
Abdominal Muscles
LUMBAR NERVES
Leg Muscles
SACRAL NERVES
Bowel, Bladder
Sexual Function

Early Effect of Injury

Patient exhibit a syndrome called "spinal shock" // the entire spinal cord is traumatized

Spinal shock is loss of "spinal cord function"

Spinal shock is not the same as cardiovascular shock or neurogenic shock that causes the collapse of the circulatory system

Skeletal muscles below level of injury exhibit flaccid paralysis

Absence of reflexes below injury because of the lack of stimulation from higher levels of the CNS

Lacks bladder and bowel reflexes (retains urine and feces) for period of 8 days to 8 weeks (average 20 days following spinal shock).

Early Effect of Injury

Due to injury the sympathetic nervous system innervation to blood vessels below injury stops

Blood vessels normally under sympathetic tone

Following transection - Exhibit neurogenic shock as vessels dilate and blood pressure drops dangerously low

Under normal brain function brain sends low level tonic excitatory signal to skeletal muscles which moves resting membrane potential closer to threshold. Below injury these signals are stopped so now these skeletal muscles are “hyperpolarized” or harder to contract!

Fever may also occur because hypothalamus can not induce sweating to cool body

Spinal shock can last from a few days to several weeks

What Happens As Spinal Shock Subsides?

Somatic reflexes (i.e. skeletal muscles) begin to reappear /// reversing flaccid paralysis with spastic paralysis

Toes to feet to legs // more acurate understanding is monosynaptic reflexes reappear before polysynapatic reflexes

During spinal shock the postsynaptic membrane on skeletal muslces upregulate acetylcholine receptors in absence of neurotransmitter // when acetlycholine again secreted greater response //

Autonomic reflexes (i.e. smooth muscle) begin to reappear (reversing retention) /// incontinent urinary bladder and rectum // empty reflexively in response to stretch /// no control over external sphincter muscles

What Happens As Spinal Shock Subsides?

Hyper-reflexia or the Mass Reflex Reaction // occurs to somatic reflexes and ANS reflexes

stimuli from a full bladder or cutaneous touch can trigger extreme cardiovascular reaction

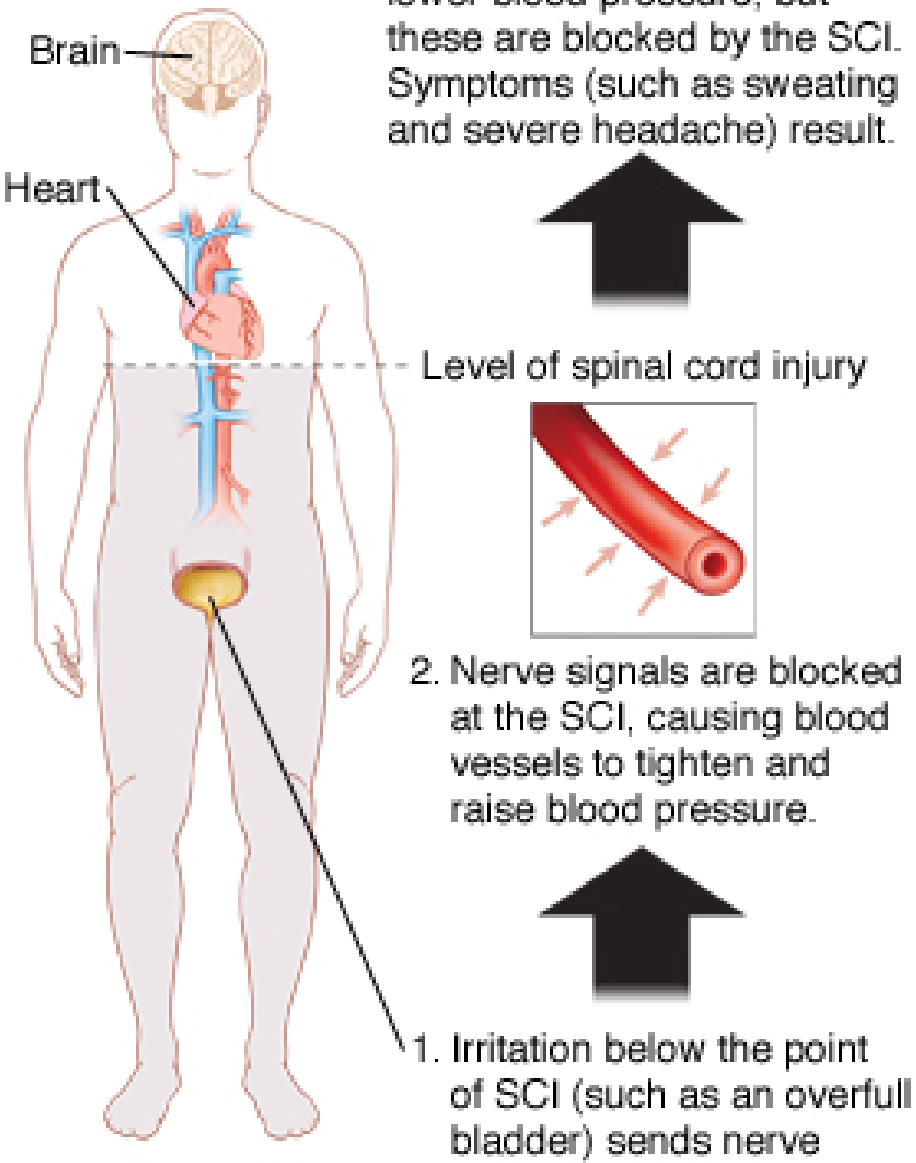
systolic blood pressure (normal systolic BP of 120 mmHg jump to 300 mmHg)

intense headaches, blurred vision, profuse sweating // homeostatic reflex slows heart to 30 to 40 bpm = bradycardia

What Happens As Spinal Shock Subsides?

At first men lose capacity for erection and ejaculation // may recover functions later with ability to ejaculate but without sensation

Women's menstruation may become irregular or cease.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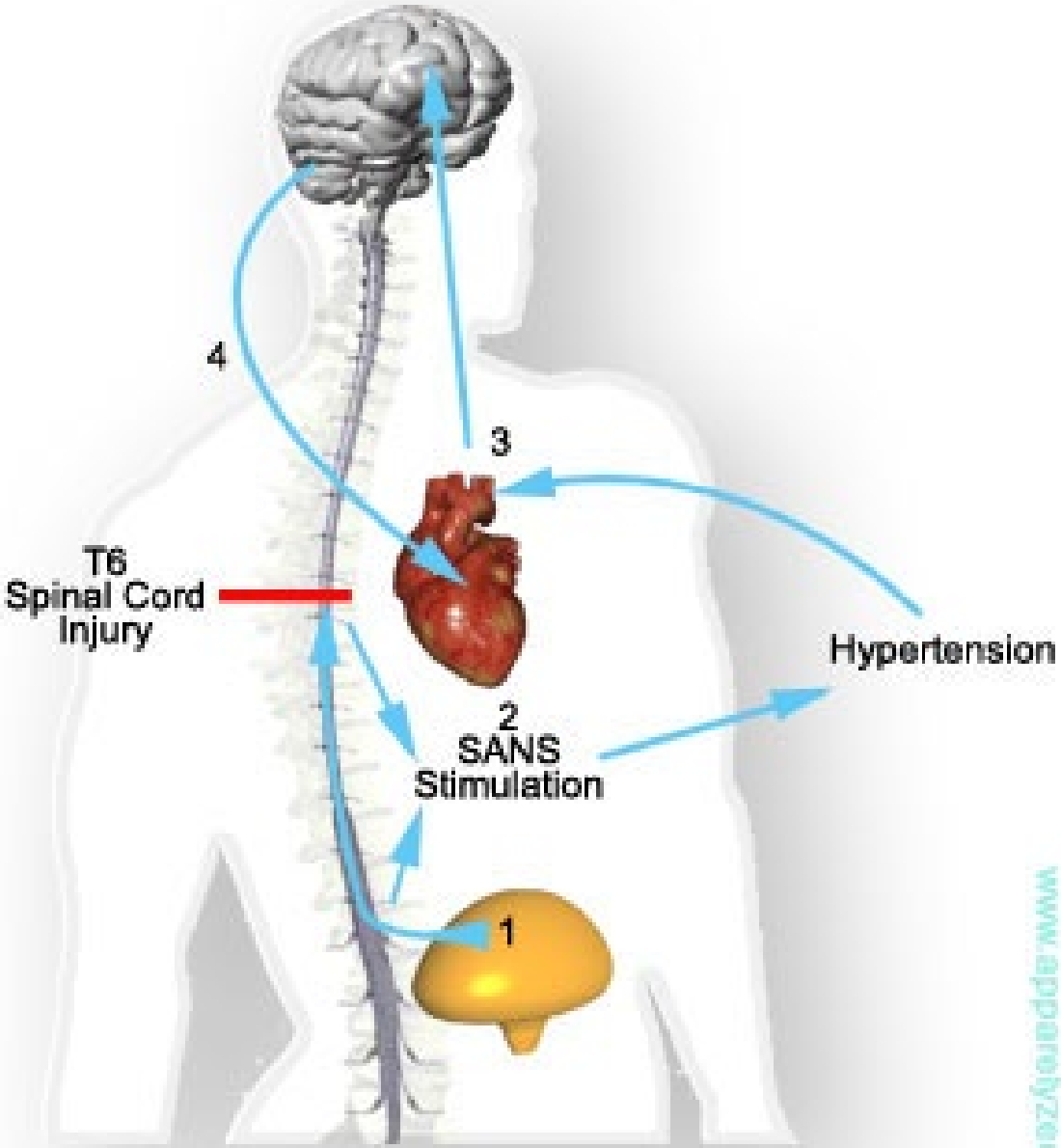

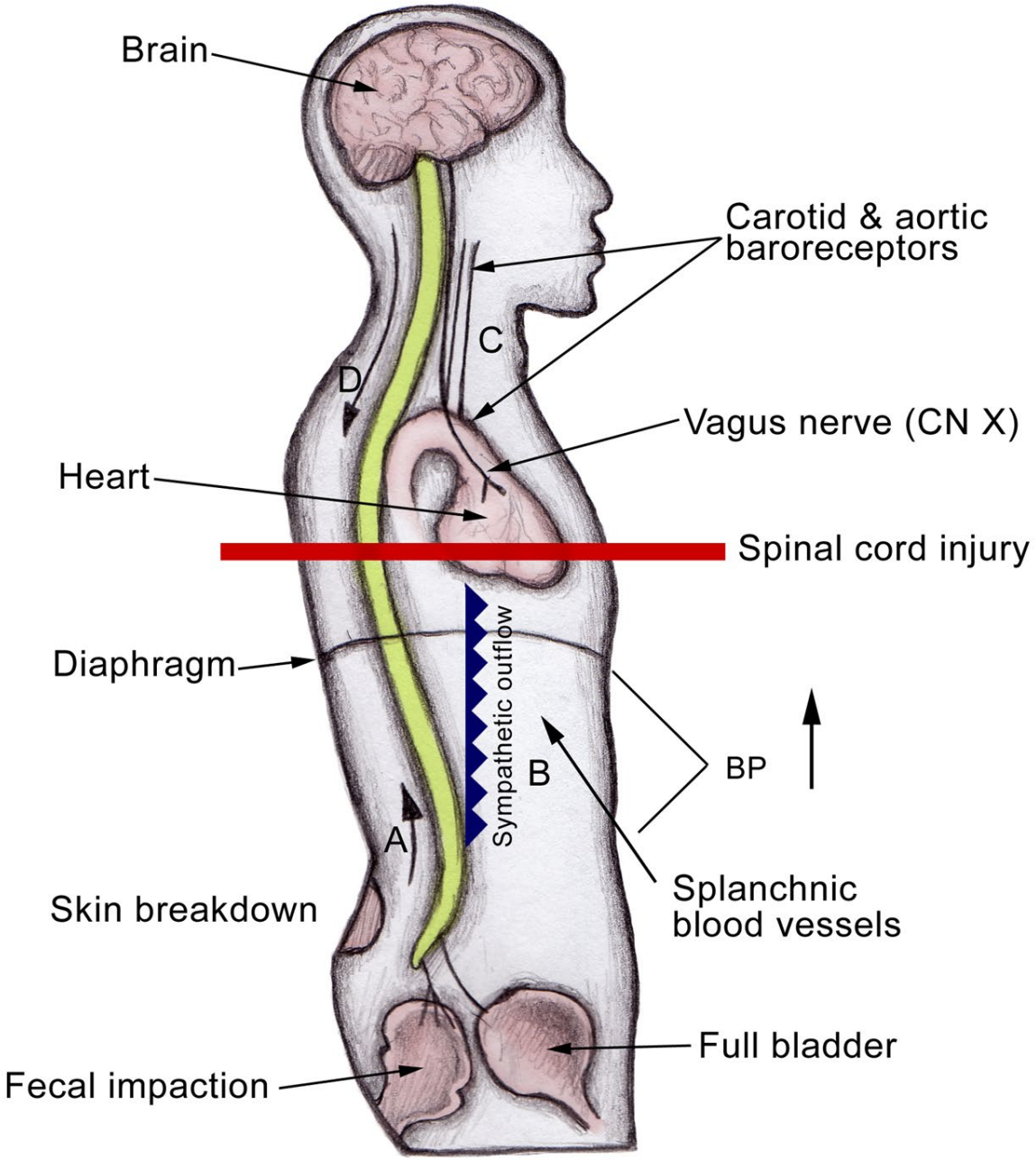
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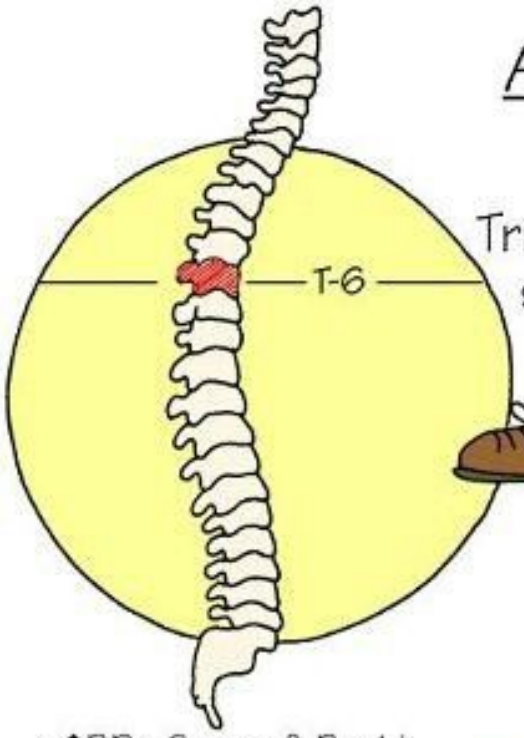

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- - Level of Injury - -

Vasoconstriction Below Level of Injury

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Most Serious Permanent Effects of Spinal Cord Trauma

Paralysis

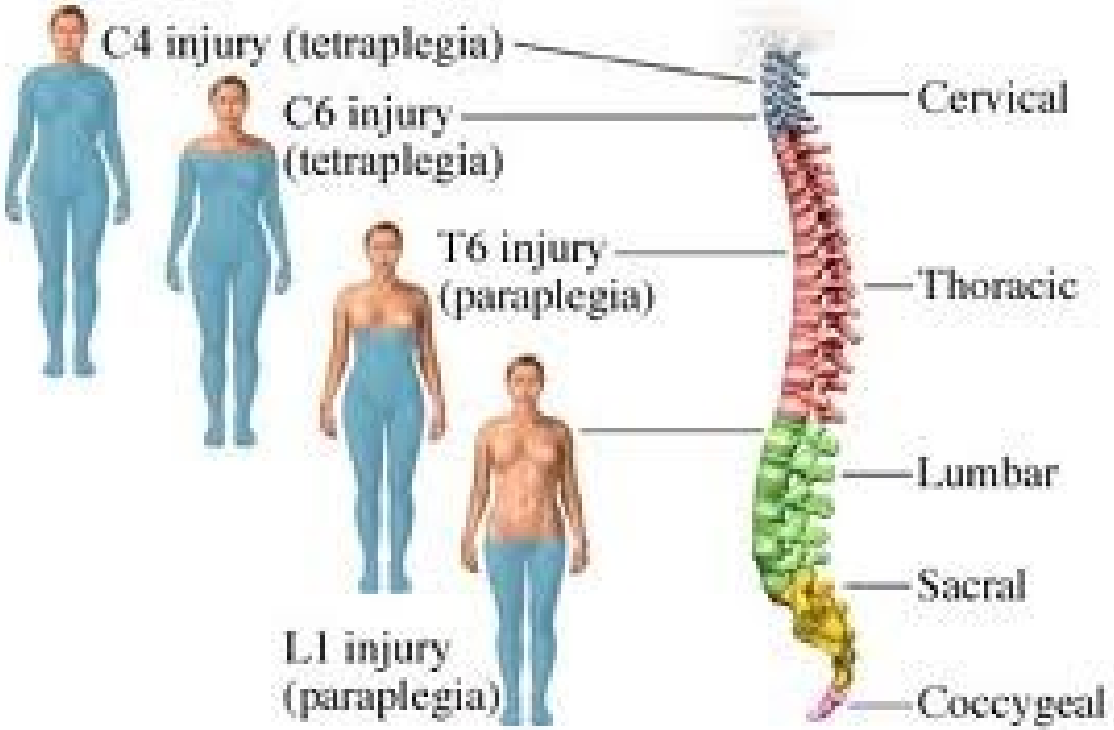
Initial flaccid paralysis is later changed to spastic paralysis as spinal reflexes are regained but without the CNS inhibitory signals which occurred before the trauma

Starts with chronic flexion of the hips and knees (flexor spasms) // later limbs become straight and ridgid (extensor spasm)

Most Serious Permanent Effects of Spinal Cord Trauma

Three type of paralysis:

* paraplegia (T1 to L1 lesion)
* quadriplegia (lesion above C5)
* hemiplegic (paralysis of one side of the body / associated with stroke and not cord lesion)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C6 injury (tetraplegia)
T6 injury (paraplegia)
L1 injury (paraplegia)
Cervical
Thoracic
Lumbar
Sacral
Coccygeal

Pathogenesis of Spinal Cord Trauma / Two Stages

First Stage // Instantaneous / destruction of spinal cord cells at site of trauma

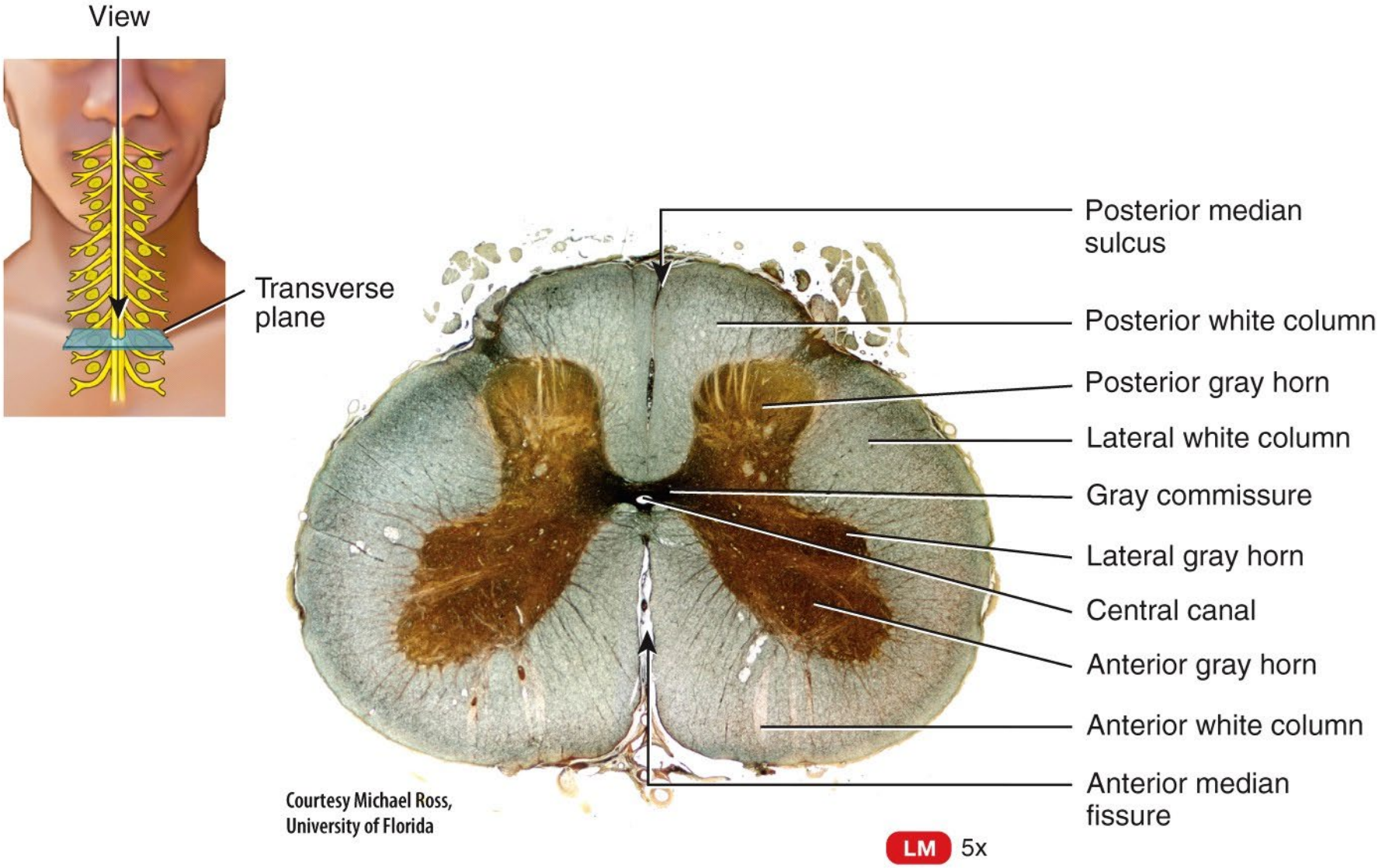
Second Stage // tissue death by necrosis and apoptosis which begins in minutes and last for days // far more destructive

Second stage converts a lesion in one spinal cord segment into a lesion that spans four or five segments (two above and two below)

Microscopic hemorrhages appear in the gray matter and pia mater within minutes of injury and grows during first two hours post injury

White matter becomes swollen

If lesion is in cervical region may be fatal if it effects respiration or brainstem function



(b) Transverse section of lumbar spinal cord

Second Stage Pathogenesis of Spinal Cord Trauma (cont.)

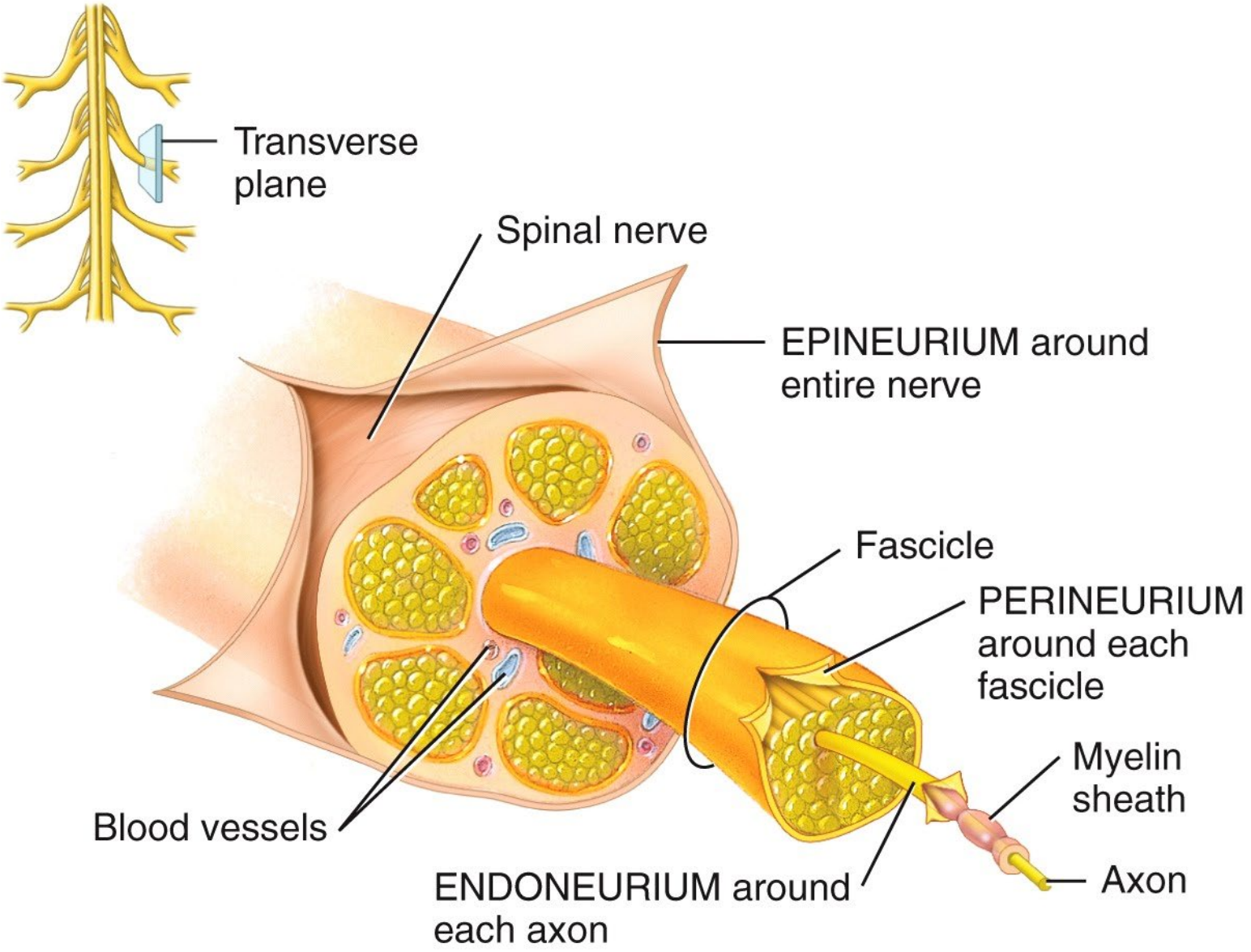
Ischemia leads to tissue necrosis

Within 24 hours white matter regains circulation but grey matter remains ischemic

Blood circulation bring into lesion leukocytes and macrophage // macrophage start to clean up cell debris but leukocytes (neutrophils) release free radicals and proteases which further contribute to the damage

Necrosis continues to worsen due to the leukocyte inflammatory response

Apoptosis of the oligodendrocytes (myelinating glial cells of the CNS) results in demyelinatin of spinal nerve fibers and death of the neurons



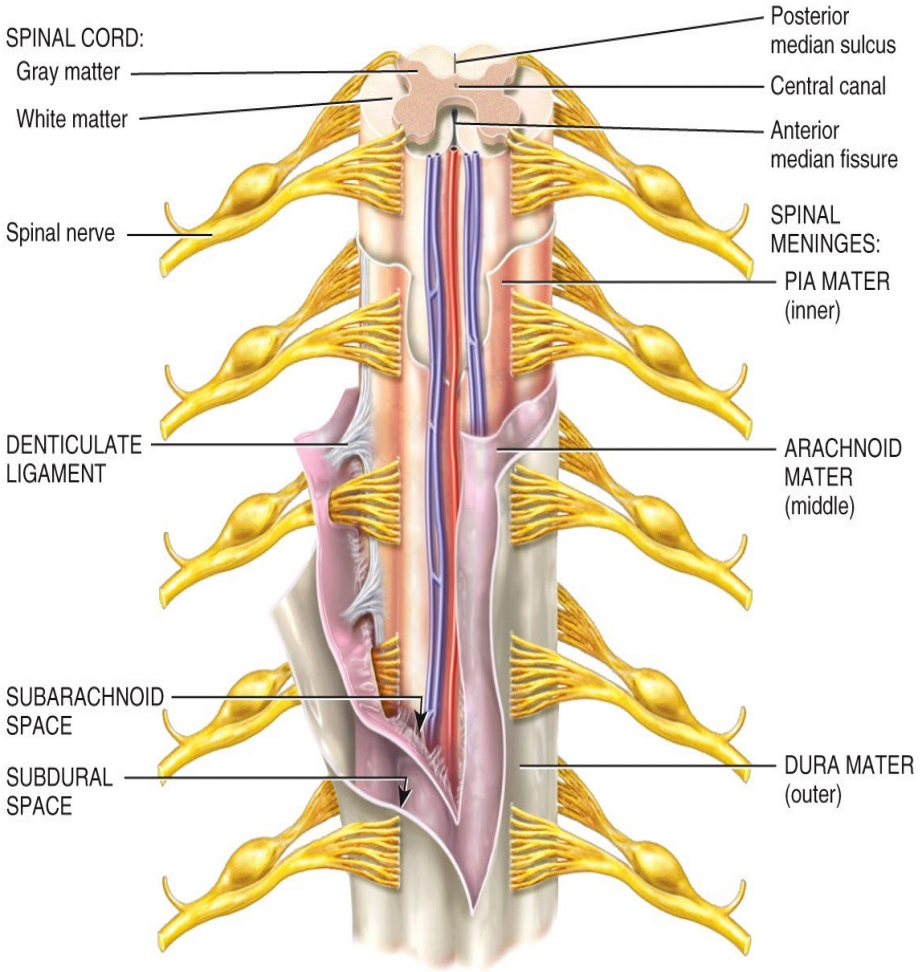
(a) Transverse section showing the coverings of a spinal nerve

Second Stage Pathogenesis Rate of Progression

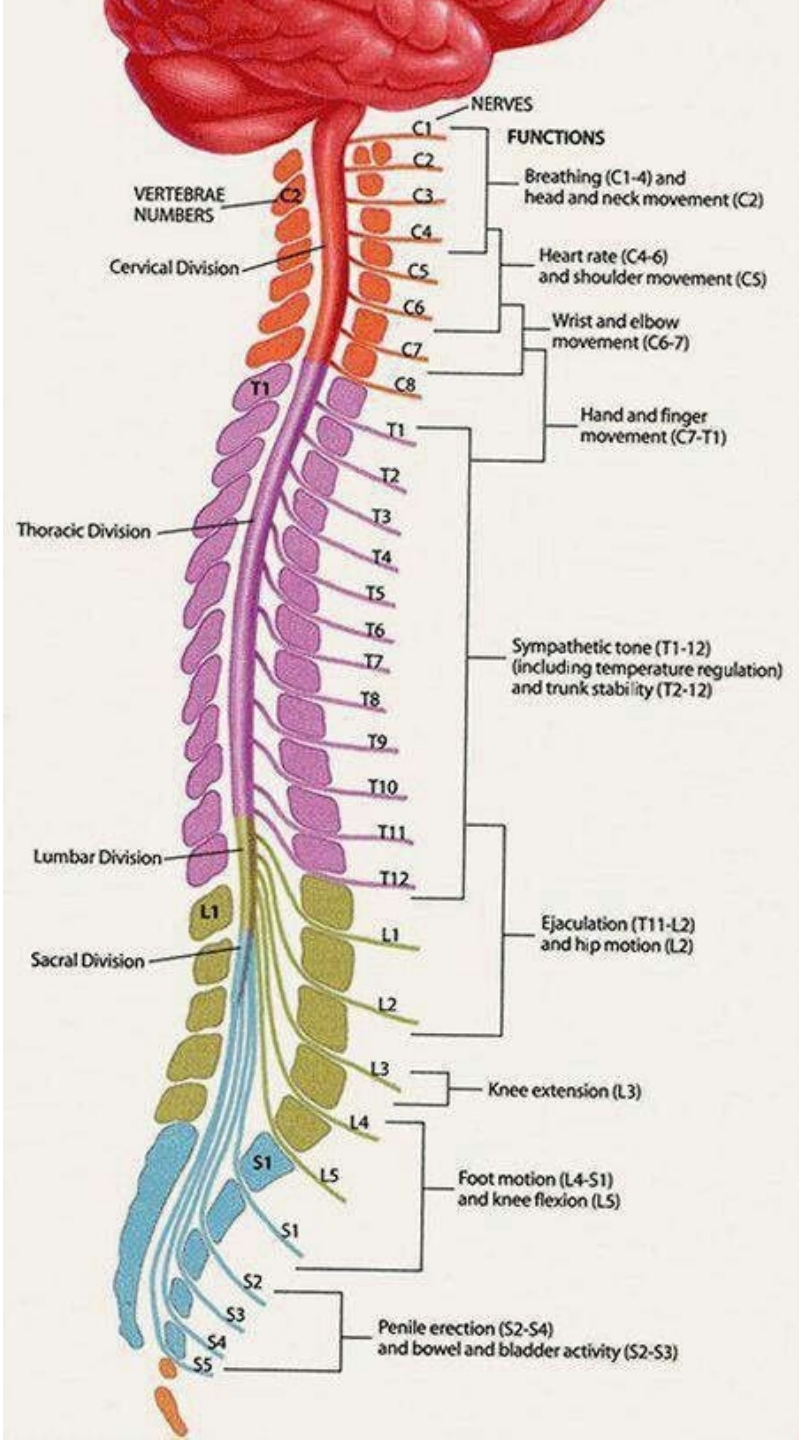
Within four hours the second stage of destruction may consume 40% of the cross sectional area

Within 24 hours the second stage of destruction may consume 70% of the cross sectional area.

Transforms up to five segments of the cord into a fluid-filled cavity // replaced with collagenous scar tissue over the next 3 to 4 weeks



(a) Anterior view and transverse section through spinal cord


NERVES
FUNCTIONS
C1
C2
C3
C4
C5
C6
C7
C8
T1
T2
T3
T4
T5
T6
T7
T8
T9
T10
T11
T12
L1
L2
L3
L4
L5
S1
S2
S3
S4
S5
VERTEBRAE NUMBERS
Cervical Division
Thoracic Division
Lumbar Division
Sacral Division
Breathing (C1-4) and head and neck movement (C2)
Heart rate (C4-6) and shoulder movement (C5)
Wrist and elbow movement (C6-7)
Hand and finger movement (C7-T1)
Sympathetic tone (T1-12) (including temperature regulation) and trunk stability (T2-12)
Ejaculation (T11-L2) and hip motion (L2)
Knee extension (L3)
Foot motion (L4-S1) and knee flexion (L5)
Penile erection (S2-S4) and bowel and bladder activity (S2-S3)

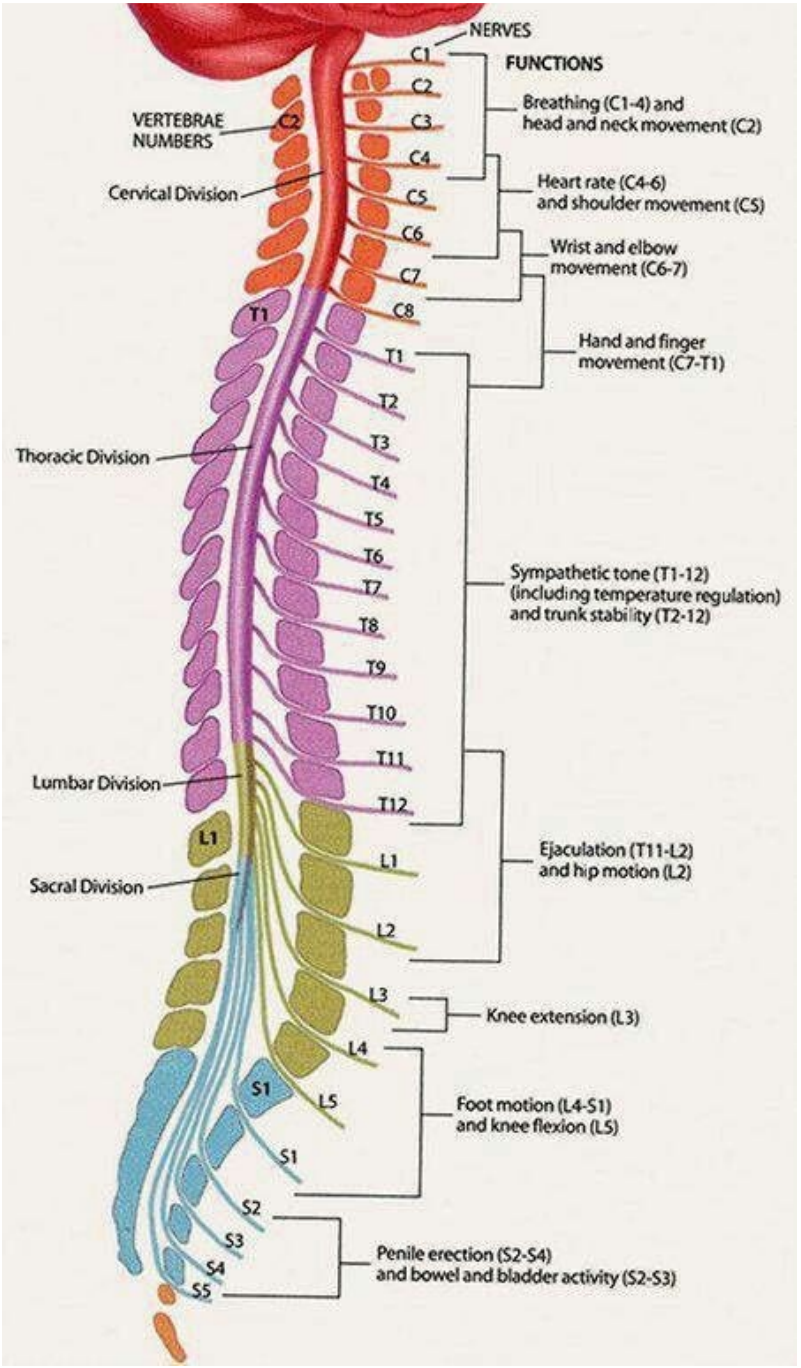
Treatment

First concern, immobilize spine to prevent further injury

Respiratory and life support may also be required depending on site of lesion

Methylprdnisolone (steroid) dramatically improves recover if administered within three hours

How do steroids reduce tissue damage in spinal cord transection? Reduce inflammation (see next slide)



Role of Steroids in Regulating Inflammation

Anti-inflammatory benefits of glucocorticoids:

decreasing capillary permeability

enhance hormones epinephrine and norepinephrine in the system to stabilize the vascular system

reduce the number of lymphocytes and mast cells in the area which decrease the amount of histamine and prostaglandins released.

Blocks the immune response / common cause of inflammation

Adverse effects of glucocorticoids

* atrophy of lymphoid tissue / less WBC increases likely infections

* catabolic effect / increase tissue breakdown and decreases protein synthesis which delays tissue regeneration

* contributes to osteoporosis / muscle wasting / lesions in skin

* delayed healing

* retention of salt with resulting hypertension and edema

* gluconeogensis causing rise in blood sugar

Spinal Shock VS Neurogenic Shock

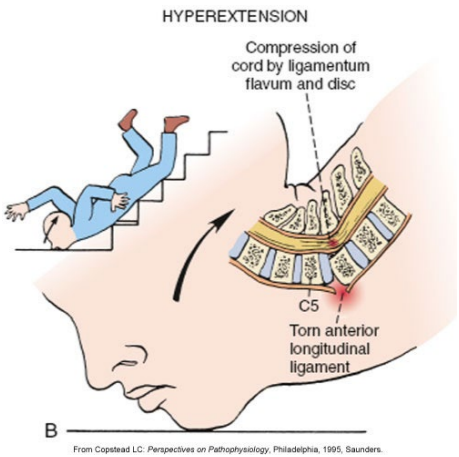
Spinal Shock

- Due to acute spinal cord injury
- Absence all volumtary motor control below level of injury
- Absence all reflex neurologic (somatic and ANS) below level of injury
- Characteristics: decreased reflexes, loss of sensations, flaccid paralysis below injury
- Last days to months
- Spinal shock and neurogenic shock can occur in same patient but they are not same disorder

Neurogenic Shock

- Hemodynamic phenomenon
- Loss of vasomotor tone with loss of sympathetic nervous system tone // inpaired cellular metabolism follows.
- Critical features: hypotension due to massive vasodilation // Bradycardia due to unopposed parasympathetic stimulation // Poikilothermia a condition where you are unable to regulate termerature
- Management: airway support // fluids as needed // atropine to reverse bradycardia // vasopressors for blood pressure support

Spinal Cord Transsection and Spinal Shock



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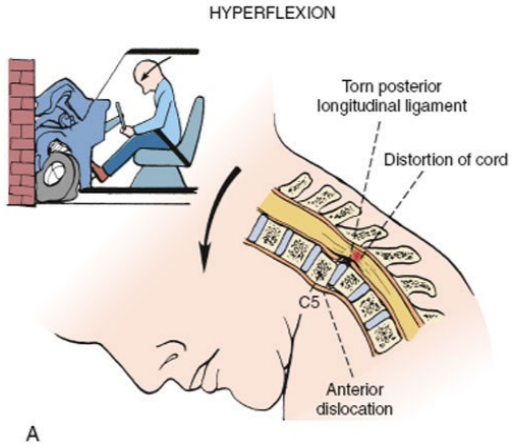

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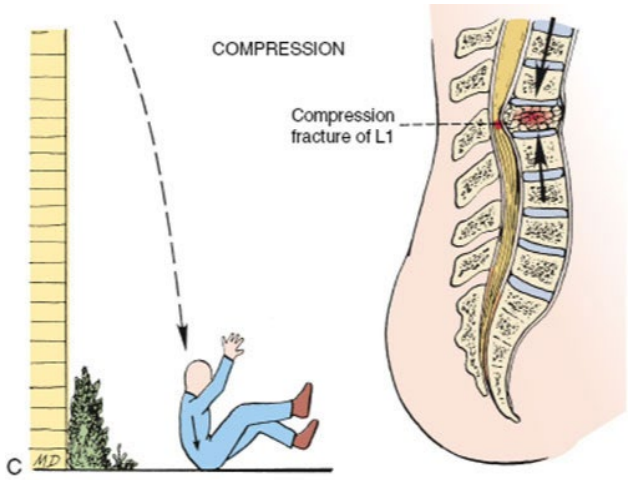


From Thibodeau GA: *Anatomy and Physiology*, St. Louis, 1987, Mosby.

Spinal Cord Injury

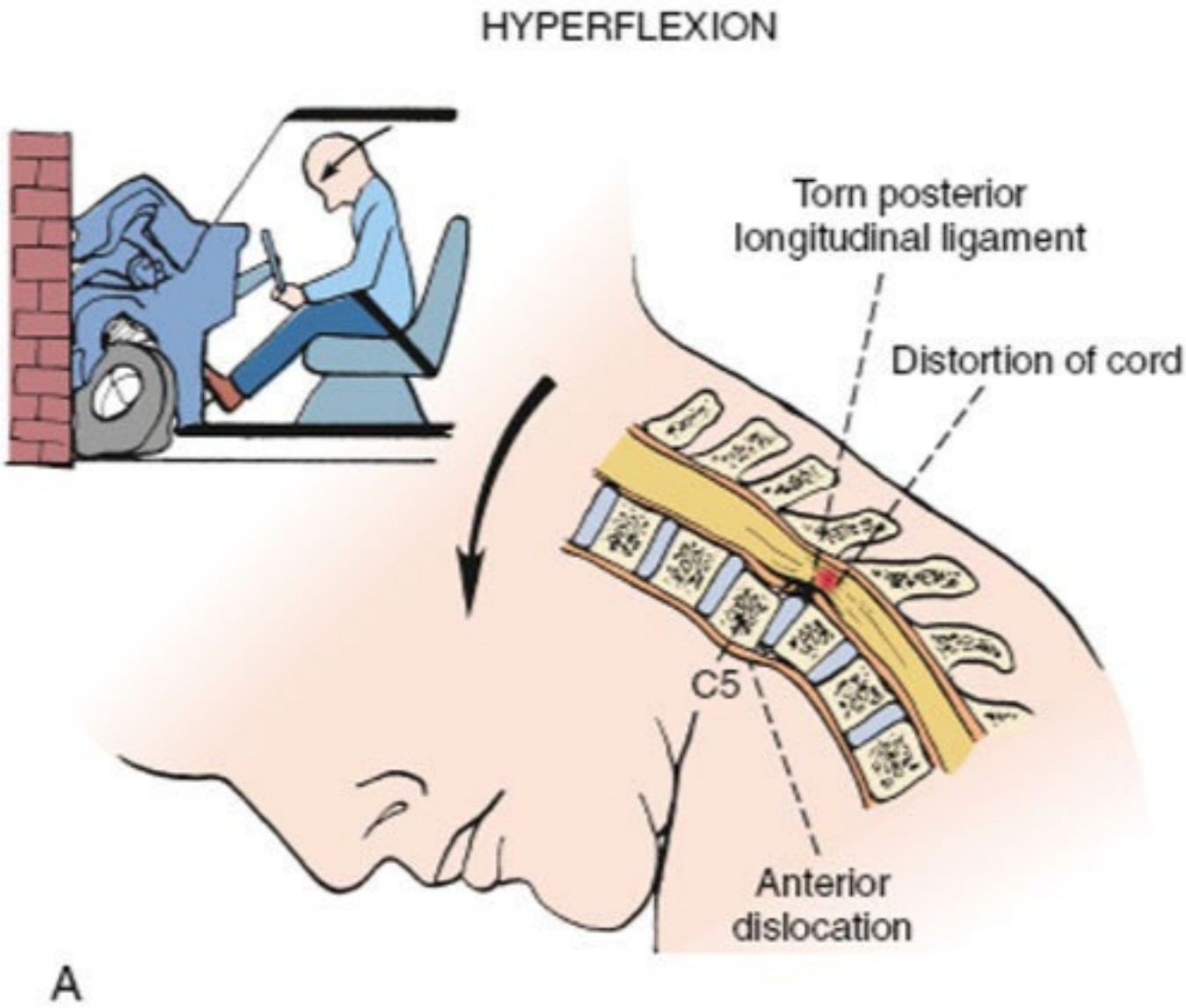
•Results from fracture, dislocation of vertebrae // Compresses, stretches, or tears spinal cord

•Cervical spine injuries // May result from hyperextension or hyperflexion of neck with possible fracture

•Dislocation of vertebra // May crush or compress spinal cord

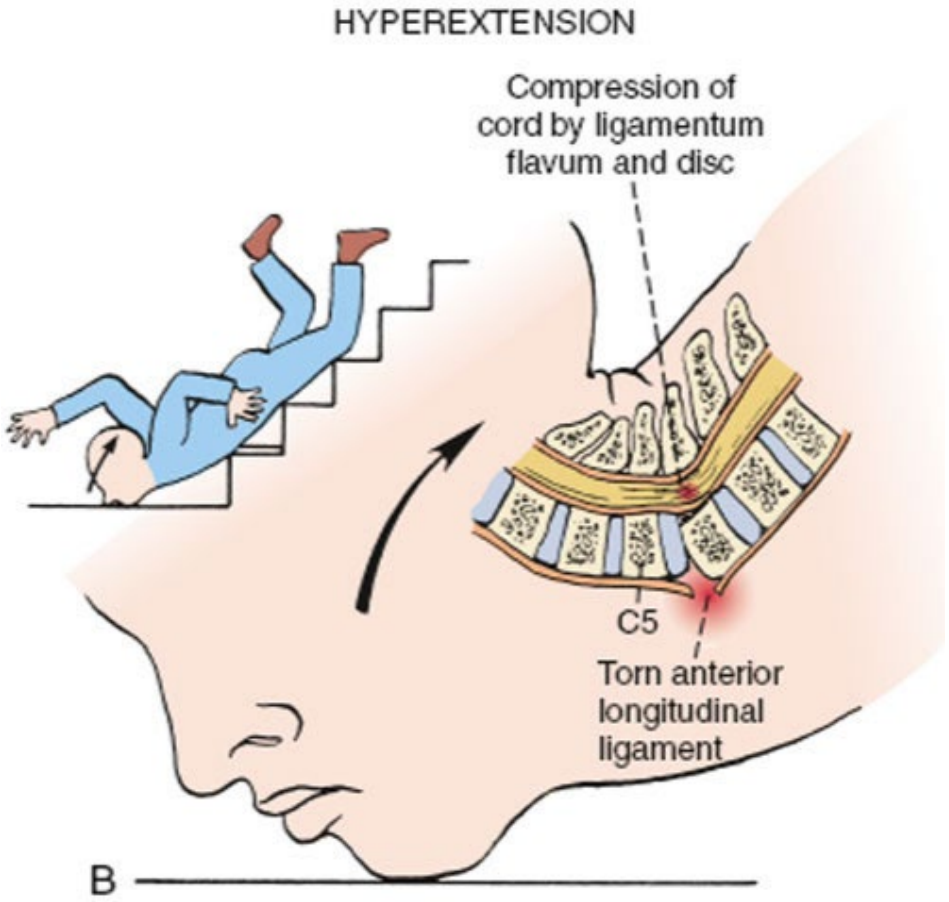
•Compression // Causes injury to spinal cord when great force is applied to top of the skull or to the feet

Types of Spinal Cord Injuries



From Copstead LC: *Perspectives on Pathophysiology*, Philadelphia, 1995, Saunders.

Types of Spinal Cord Injuries



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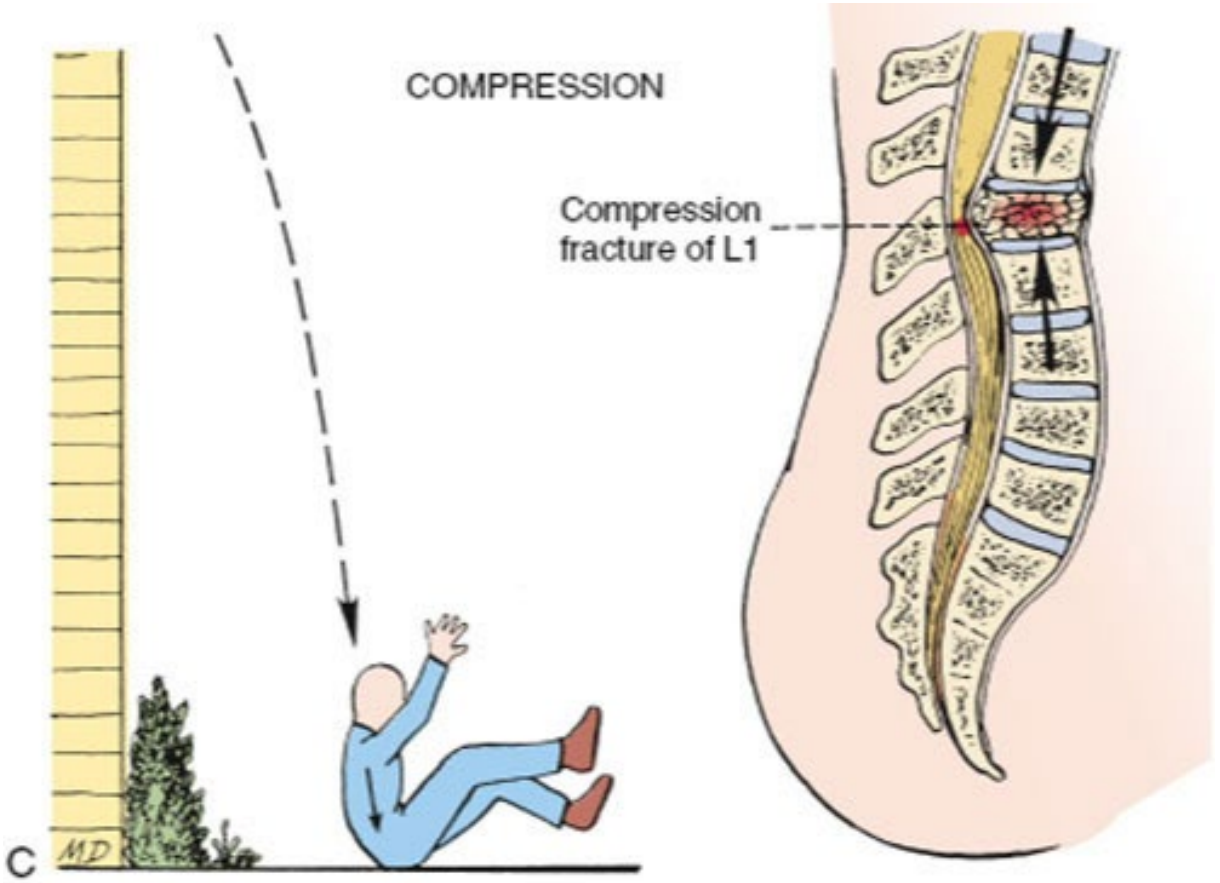
Types of Spinal Cord Injuries



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Spinal Cord Injury

•Classification of vertebral fractures

–Simple // Single line break

–Compression // Crushed or shattered bone in multiple fragments

–Wedge // Displaced angular section of bone

–Dislocation // Vertebra forced out of its normal position

Spinal Cord Injury

•Damage may be temporary or permanent. // Axonal regrowth may occur.

•Laceration of nerve tissue by bone fragments // Usually permanent loss of conduction in affected tracts

•Complete transsection or crushing of cord // irreversible loss of all sensory and motor function at and below the level of injury

•Partial transection or crushing // May allow recovery of some function

Spinal Cord Injury

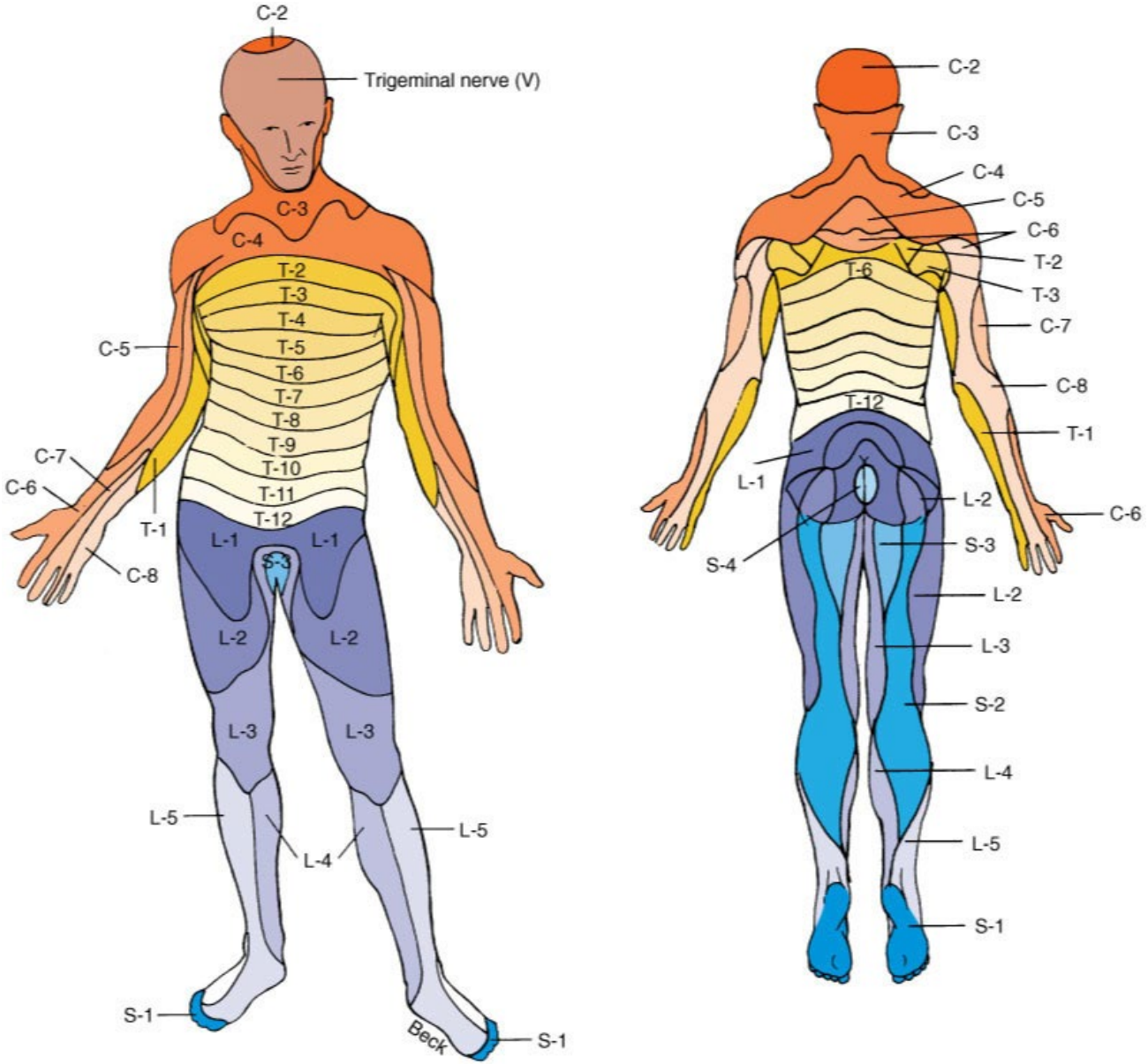
- Bruising // Reversible damage

- Prolonged ischemia and necrosis // Lead to permanent damage

- Release of norepinephrine, serotonin, histamine // Released by damaged tissue – causes vasoconstriction

- Assessment using dermatome map // Assessment of movement and sensory responses // Can determine the degree of damage or recovery

Dermatomes



From Thibodeau GA: Anatomy and Physiology, St. Louis, 1987, Mosby.

Spinal Cord Injury // Spinal Shock

–Initial period after injury – characterized by ANS reflexes absent

–Conduction of impulses ceases // Recovery dependent on amount of bleeding and surgical intervention

–Inflammation gradually subsides.

•Damaged tissue removed by phagocytes

•Scar tissue formation

–Reflex activity resumes below level of injury.

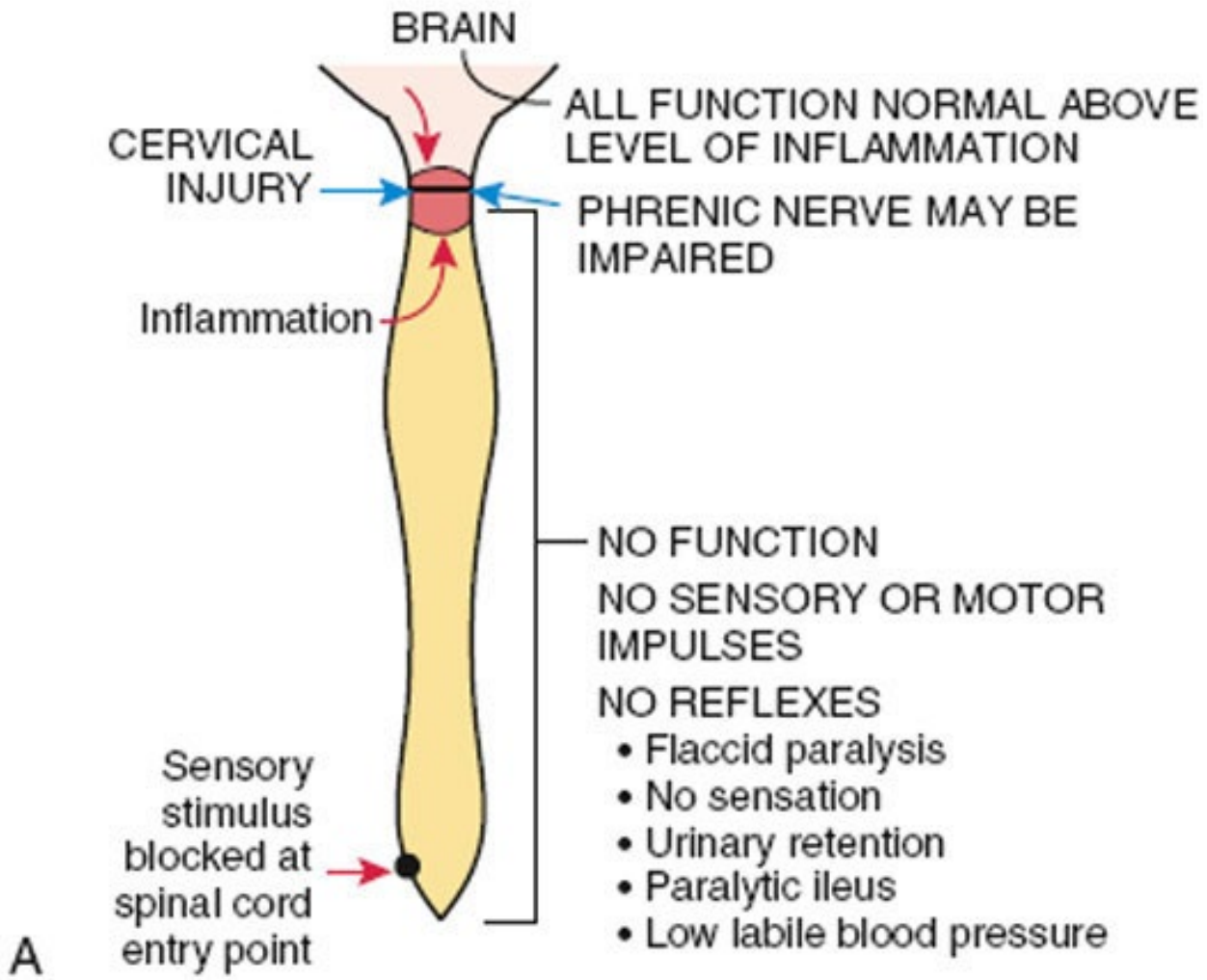
–Below injury no communication with higher levels of brain

–Control of reflexes below the level of damage is lost.

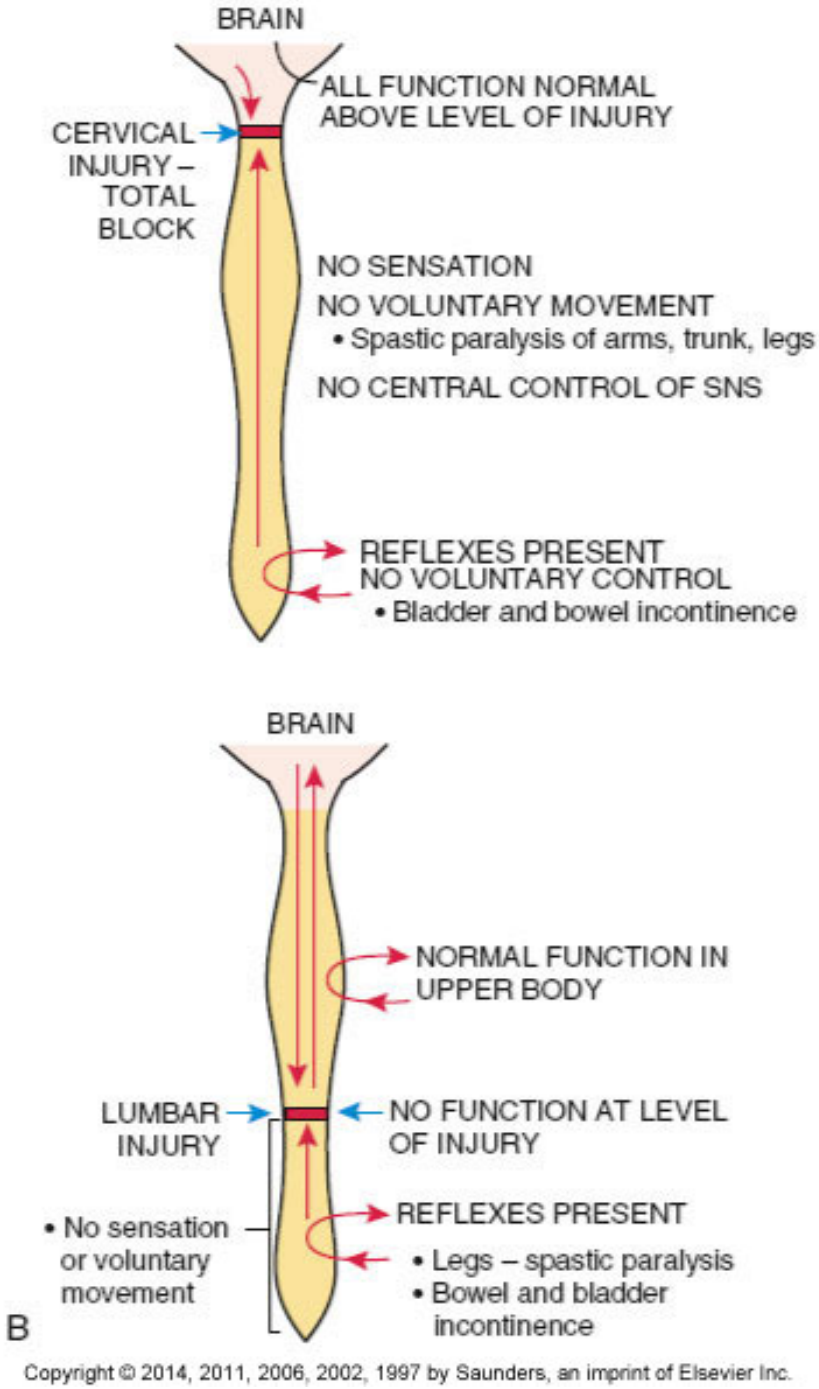
Stages of Damage

- Immediate

Events Immediately Following Spinal Cord Transection



Effects of Spinal Cord Damage After Initial Paralysis and As Reflexes Return (Hyperreflexia)



Spinal Cord Injury // Spinal Shock

•Two stages in post-traumatic period /// Spinal shock // Recovery and recognition of extent of functional loss

•Spinal shock /// Initially, all neurological activity ceases below and slightly above the level of injury.

–No reflexes present

–Condition may persist for days or weeks

- Flaccid paralysis
- Sensory loss at and below injured area
- Absence of all reflexes
- Loss of central control of autonomic

Spinal Cord Injury // Spinal Shock

•Recovery

–Gradual return of reflex activity below level of injury

–No impulses through specific area of damage

–Hyperreflexia may develop.

–Gradually, extent of damage will be revealed.

–Voluntary motor activity and sensory impulses are blocked at and below the level of damage.

–Many injuries are incomplete, and permanent damage varies among individuals.

Spinal Cord Injury // Spinal Shock

- Tetraplegia (quadriplegia) // Paralysis of all four extremities

- Paraplegia // Paralysis of the lower part of the trunk and legs

- Ipsilateral paralysis and contralateral loss of pain and temperature sensation // Depends on the point of decussation and location

Autonomic Dysreflexia

•Massive sympathetic reflex response that cannot be controlled from the brain

•Often initiated by infection, genital stimulation, or other stimuli

•Leads to:

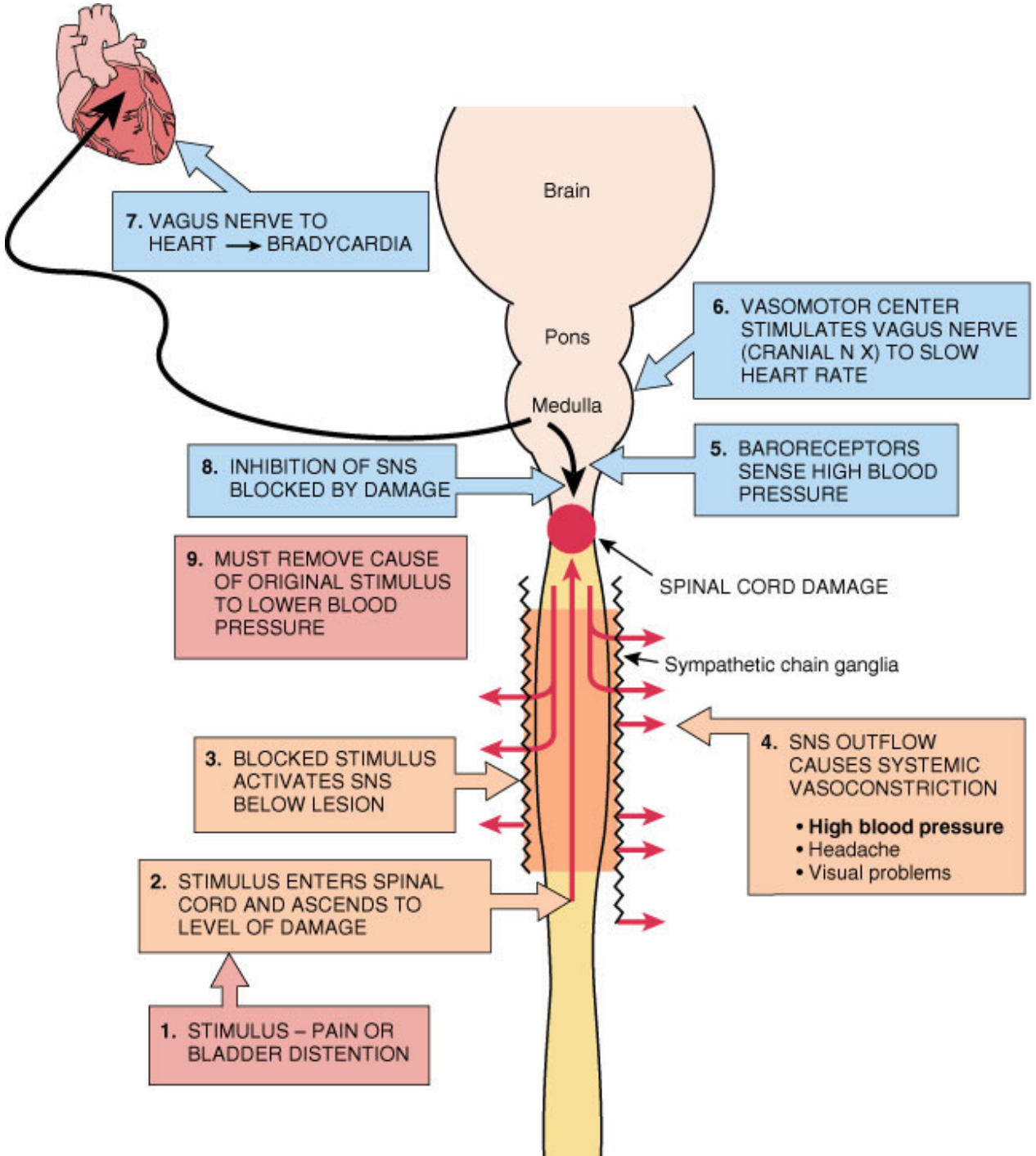
–Increased blood pressure

–Vasoconstriction below the injury

–Vasodilation above the injury

–Tachycardia

Autonomic Dysreflexia Following Spinal Cord Damage



Complications of Spinal Cord Injury

- Urinary tract infections
- Pneumonia
- Skin breakdown
- Spasm and pain
- Depression

Treatment of Spinal Cord Injury

–Treatment and rehabilitation begin at the time of injury.

–Immobilize spine.

–Maintain breathing and prevent shock.

–Hospital traction or surgery // Relieve pressure and repair tissues

–Glucocorticoids // Reduce edema and stabilize vascular system

–Ongoing care to prevent complications related to immobility

Another Spinal Cord Problem

•Herniated intervertebral disk

–Involves protrusion of the nucleus pulposus

–Tear in capsule may occur suddenly or develop gradually with aging or obesity.

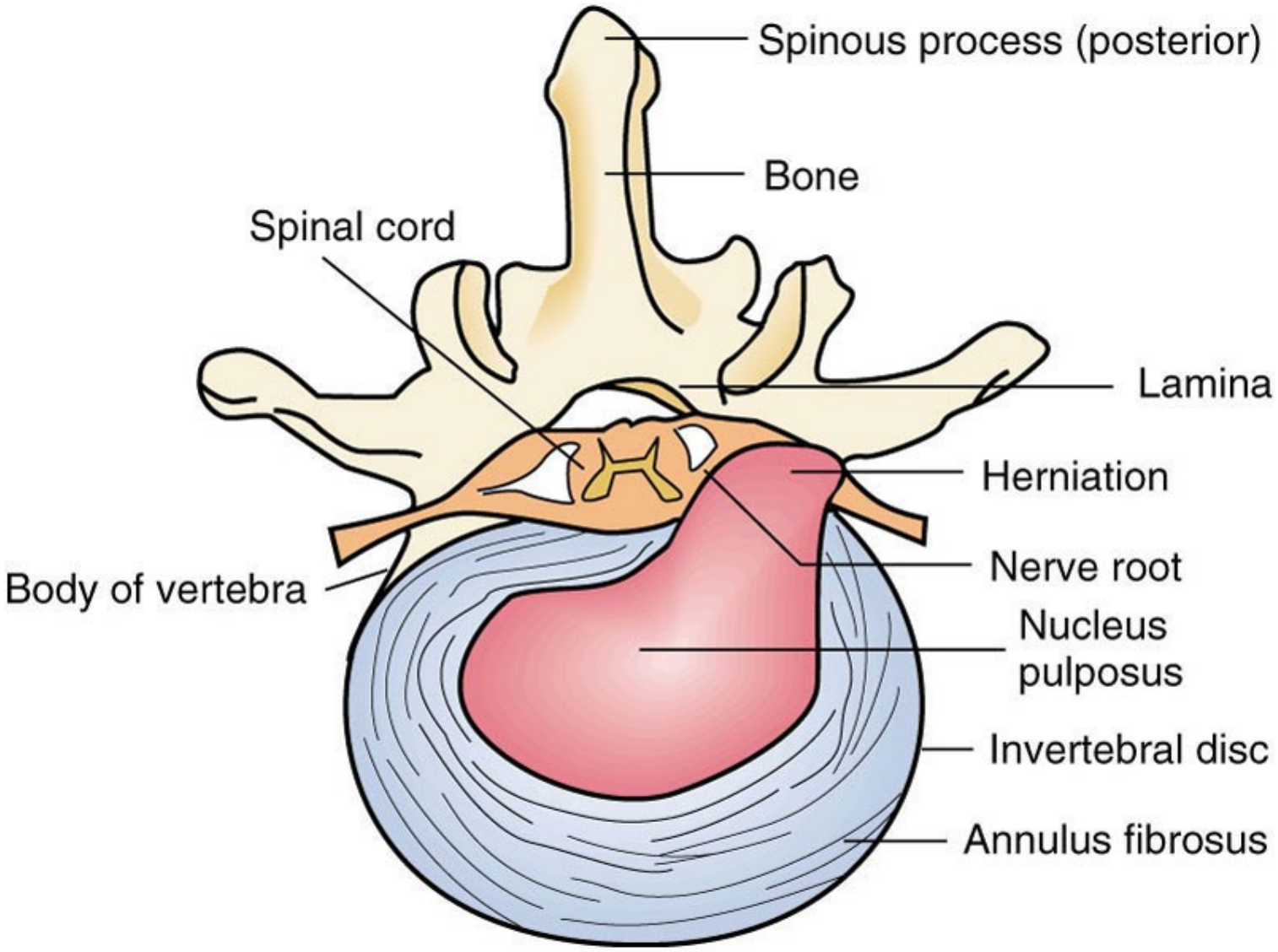
–Sensory, motor, or autonomic function may be impaired.

–Most common location—lumbosacral disks

–Some herniations involve cervical disks.

–If pressure is prolonged, severe permanent damage may occur.

Herniated Intervertebral Disk



Herniated Intervertebral Disk

•Predisposition to herniated disks

–Degenerative changes in the intervertebral disk

•Age
•Metabolic changes
•Obesity

–Herniation usually caused by trauma or poor body mechanics

•Signs depend on location and extent of protrusion.

–Most effects are unilateral.

–Large protrusions may cause bilateral effects.

Herniated Intervertebral Disk

•Diagnostic tests

–Myelography with contrast dye, CT, MRI

•Treatment

–Reduced weight-bearing, rest as needed

–Application of heat, ice, traction

–Drugs to relieve muscle spasm and pain

–Physiotherapy and occupational therapy

–Surgery in severe cases // Laminectomy or diskectomy, spinal fusion