

Spinal Cord Injury

Pathogenesis · Morphology · Clinicopathologic localization

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Learning outcomes

- Explain how vertebral displacement produces focal cord injury and tract disconnection.
- Relate lesion level and tissue compartment to motor, sensory, and respiratory deficits.
- Recognize acute hemorrhagic–necrotic injury, later cystic gliosis, and Wallerian degeneration.
- Integrate neuropathology with neurological examination and imaging.

Mechanical insult

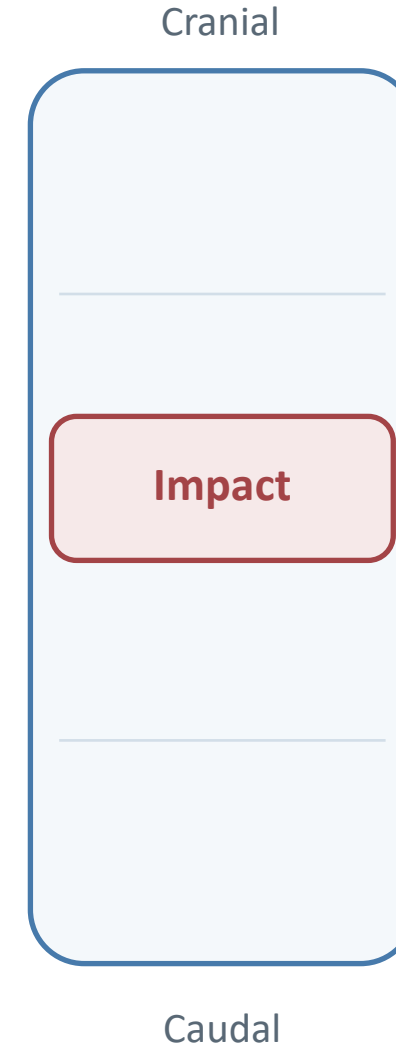
Local tissue loss

Tract disconnection

Functional deficit

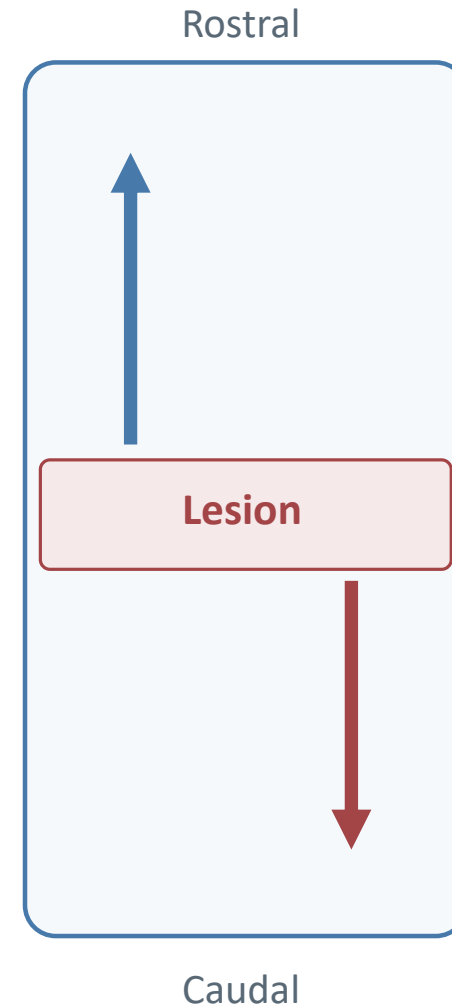
Spinal cord injury: Pathological definition

- Traumatic insult damages neurons, axons, supporting cells, and/or cord microvasculature.
- The neurological syndrome reflects both tissue destruction at impact and interruption of long pathways.
- Functional deficit may extend far beyond the small anatomic region that is directly injured.



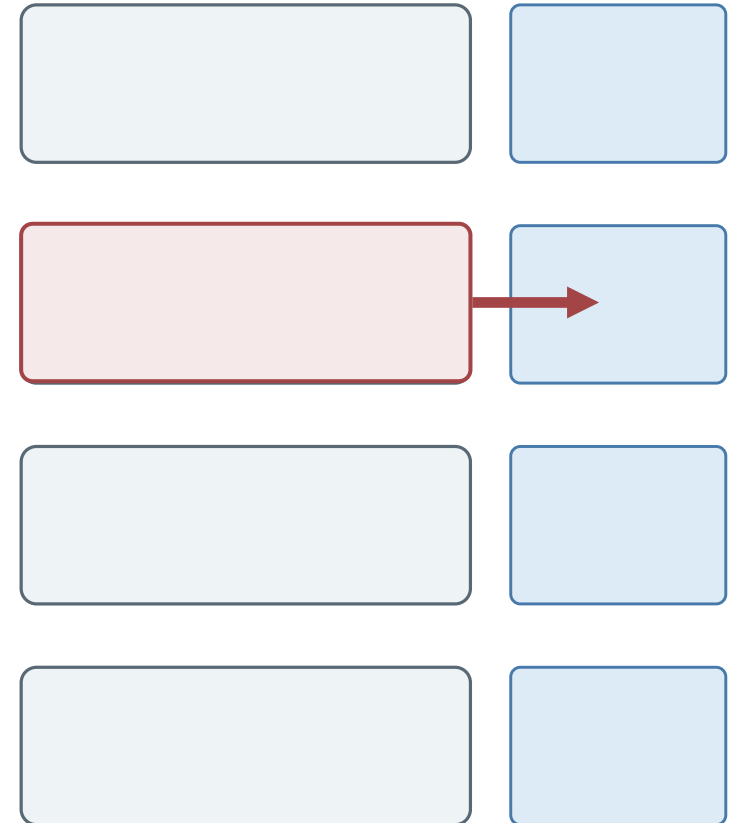
Why a small cord lesion can be devastating

- Many ascending and descending pathways are concentrated within a narrow cross-sectional area.
- Long tracts carry signals between the brain and multiple spinal segments.
- An interruption at one level can affect function throughout caudal regions.



Typical mechanical setting

- Most traumatic cord injuries accompany transient or permanent displacement of the vertebral column.
- Mechanical displacement can transmit deforming forces to the enclosed cord.
- Bone alignment alone does not fully characterize intramedullary tissue damage.

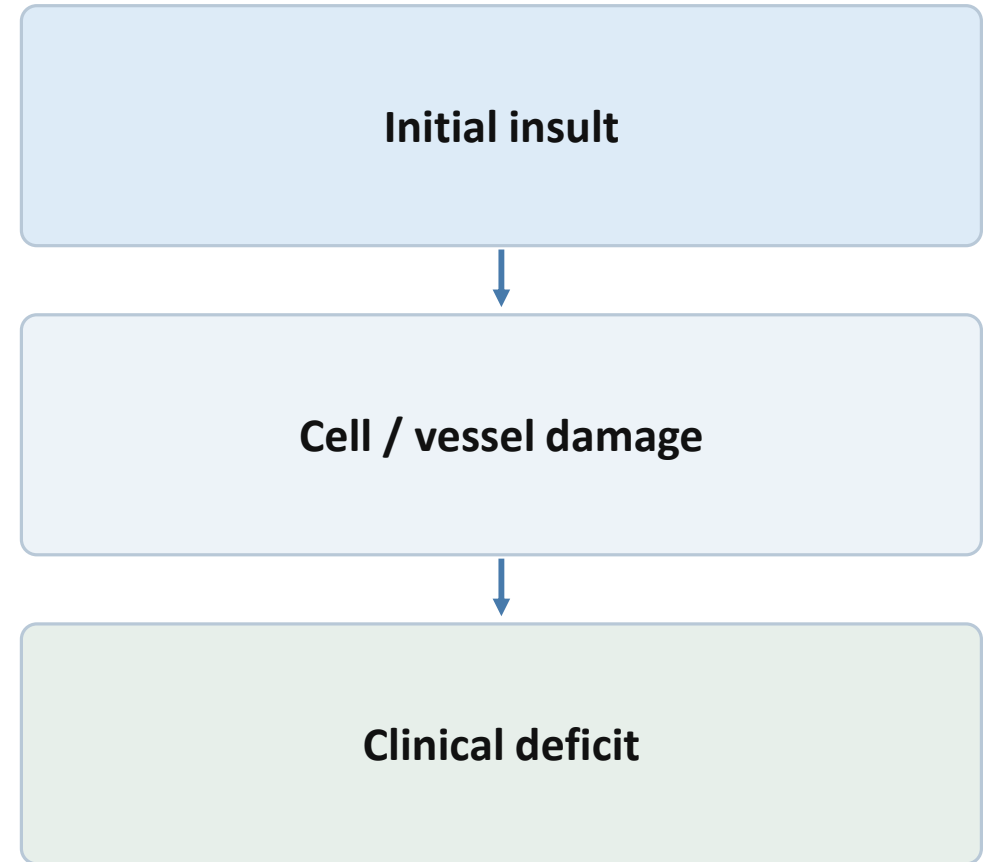


Vertebra

Cord

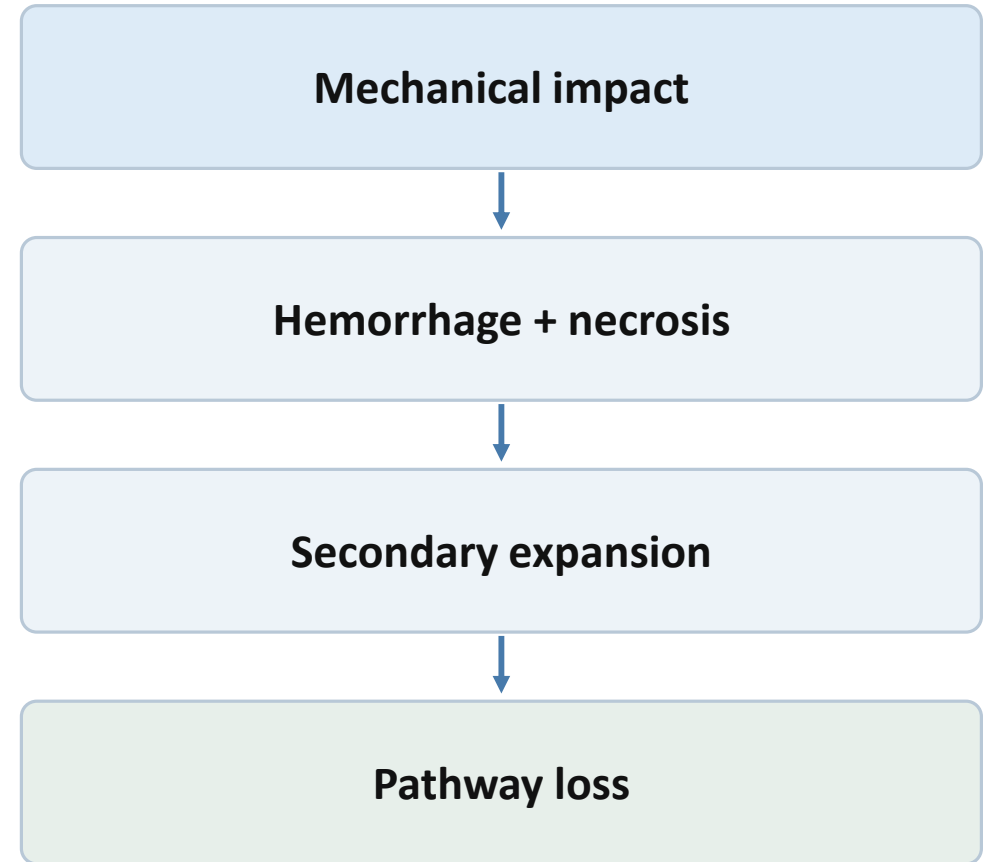
Spinal motion and injury severity

- A high-energy force can deform the cord during the event of impact.
- Compression, stretching, and shear may coexist in an individual injury.
- Final neurological damage reflects the spatial distribution of tissue injury, not only an external fracture.



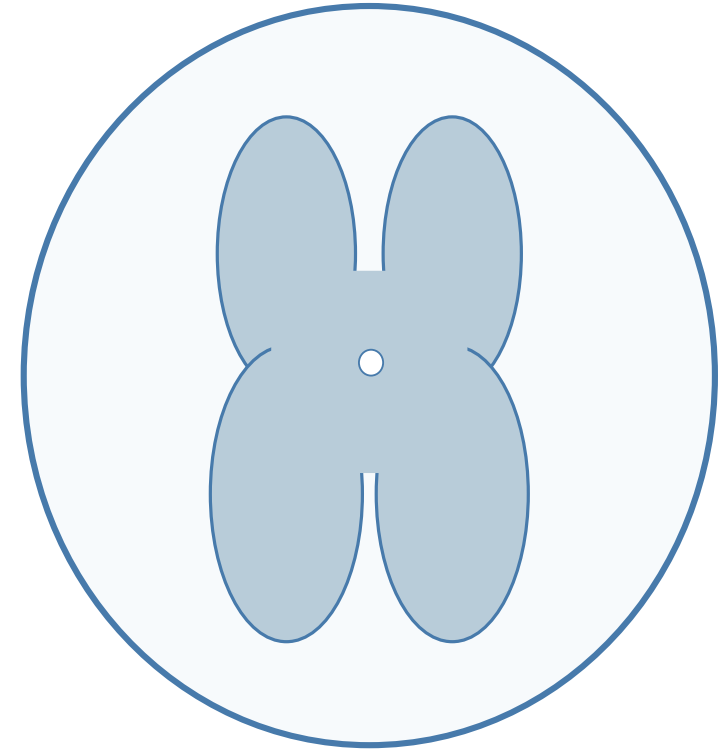
From trauma to neurological impairment

- Primary mechanical injury disrupts cellular elements and blood vessels.
- Secondary vascular and inflammatory injury can extend tissue damage.
- Axonal disruption and local gray-matter loss together account for functional deficits.



White matter and gray matter are injured differently

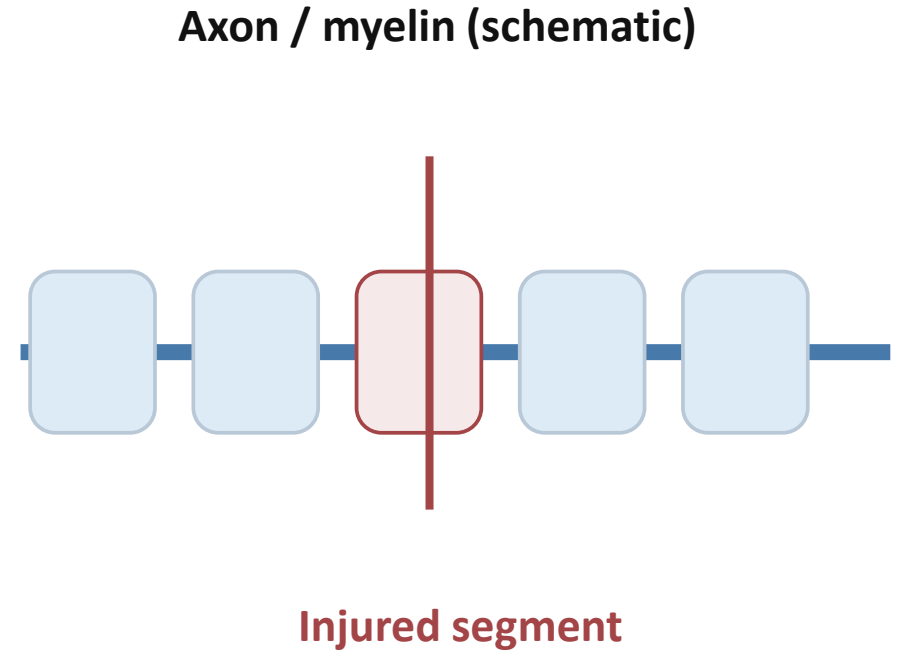
- White matter contains long myelinated axons that connect distant neural regions.
- Gray matter houses local neuronal cell bodies, synapses, and segmental circuits.
- White-matter disruption disconnects; Gray-matter necrosis removes segmental processing.



Schematic transverse cord

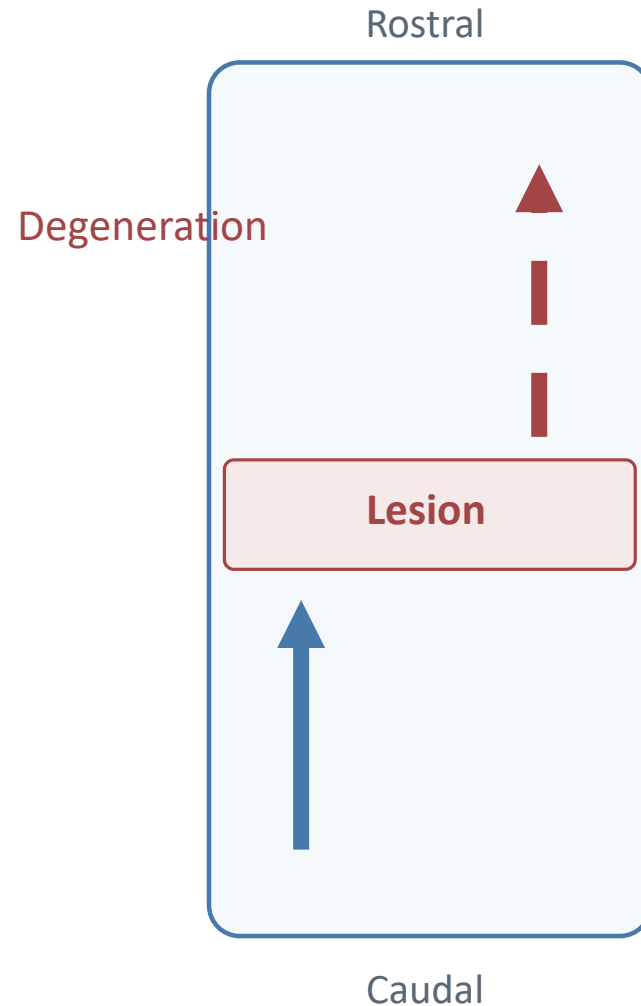
Primary axonal disruption

- Long tracts may be damaged at the level of impact.
- Signals from the brain cannot reliably cross the affected axonal segment.
- Even distant intact spinal segments can become functionally disconnected.



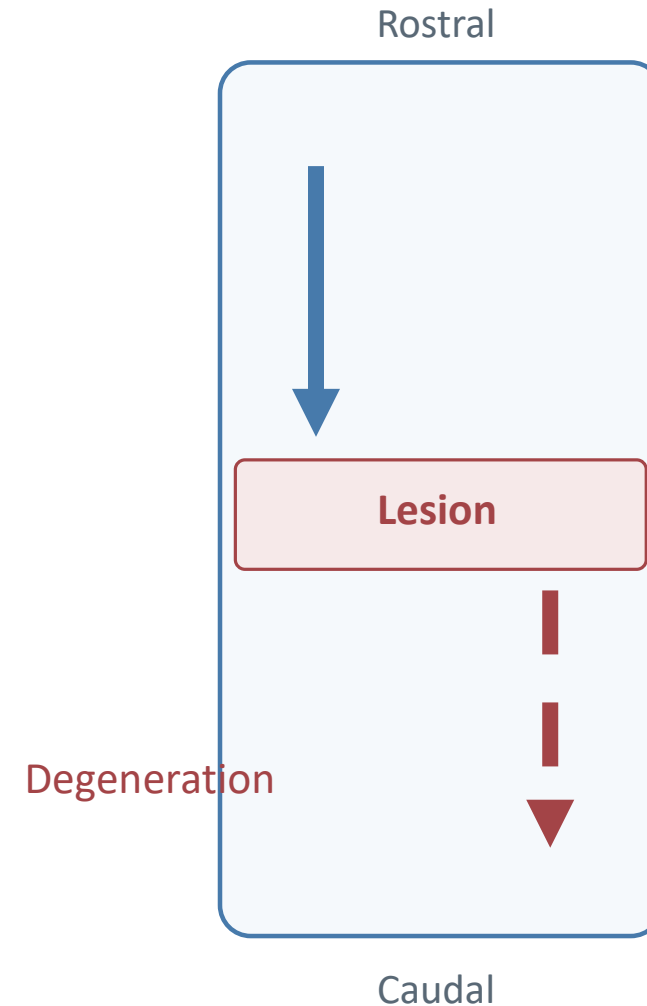
Ascending pathways: Sensory information

- Ascending tracts transmit peripheral sensory information toward higher centers.
- Cord injury can prevent sensory information arising caudal to the lesion from reaching the brain.
- A sensory level can therefore localize the functional interruption.



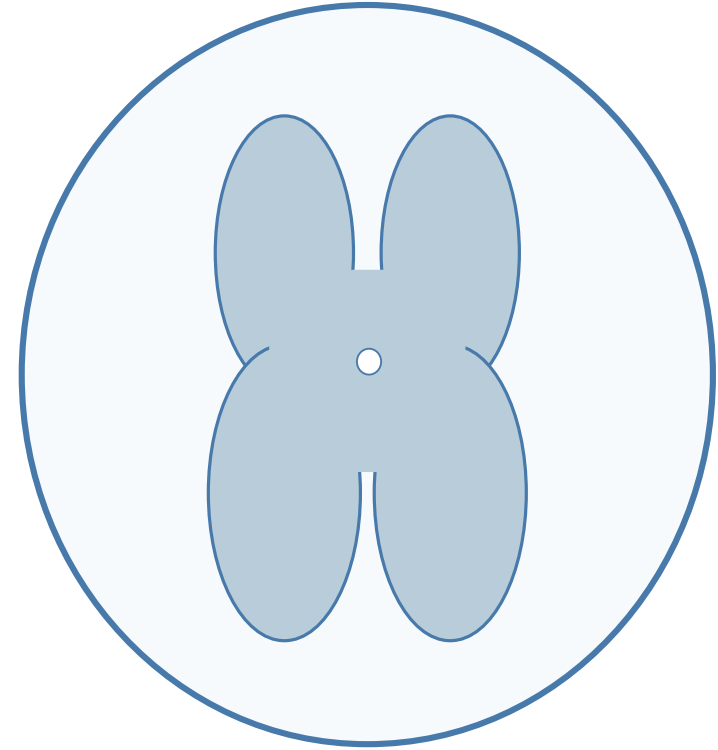
Descending pathways: Motor commands

- Descending tracts convey central motor signals to spinal motor networks.
- Interruption impairs voluntary motor output below the injury.
- Motor manifestations depend on tract involvement and the lesion level.



Segmental gray-matter destruction

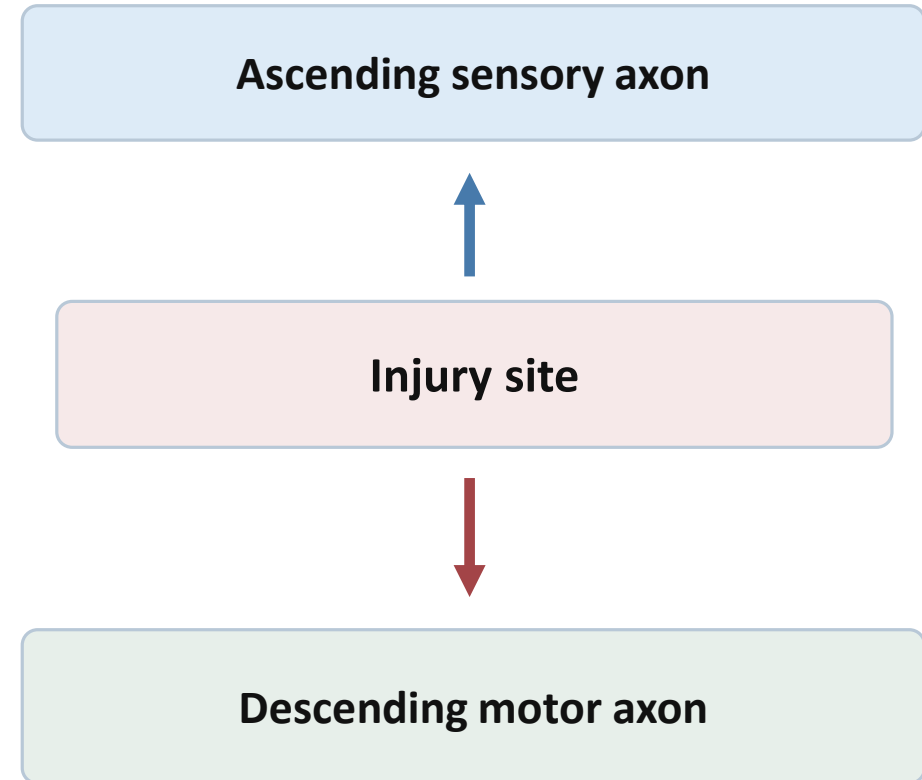
- Trauma at the lesion epicenter can directly destroy spinal neurons and interneurons.
- Damage to local motor circuits may produce segmental dysfunction.
- Segmental tissue loss coexists with long-tract disconnection in severe injuries.



Schematic transverse cord

A conceptual two-component lesion

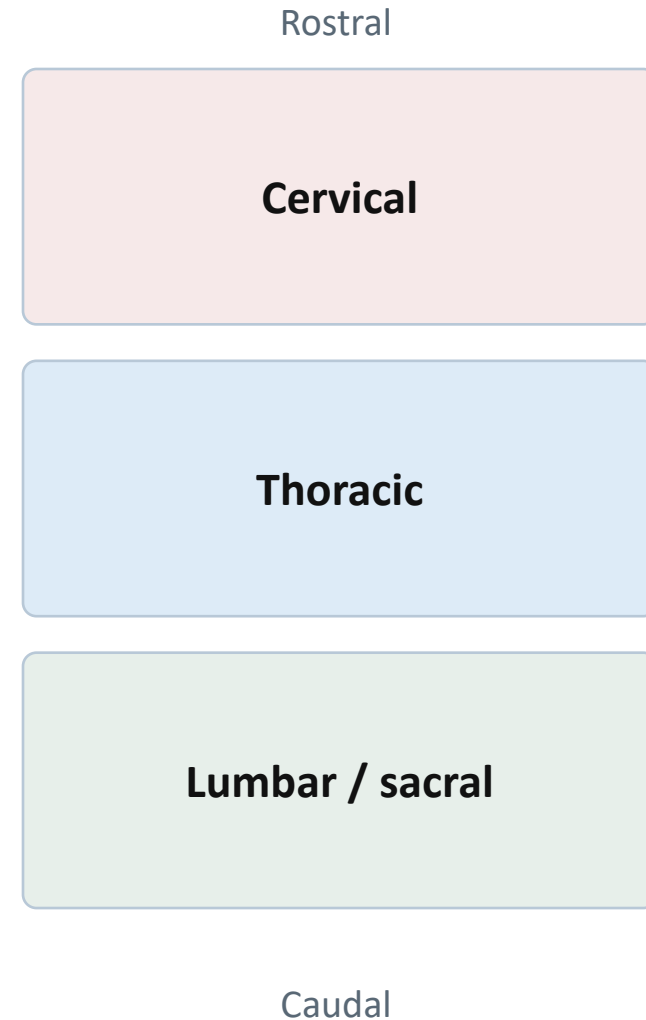
- Local lesion: Hemorrhage, necrosis, and loss of neurons at the impact site.
- Long-distance consequence: Interrupted axons impair connection with more caudal cord.
- The combination is more informative than describing only the visible lesion epicenter.



Distal = away from each neuron's soma

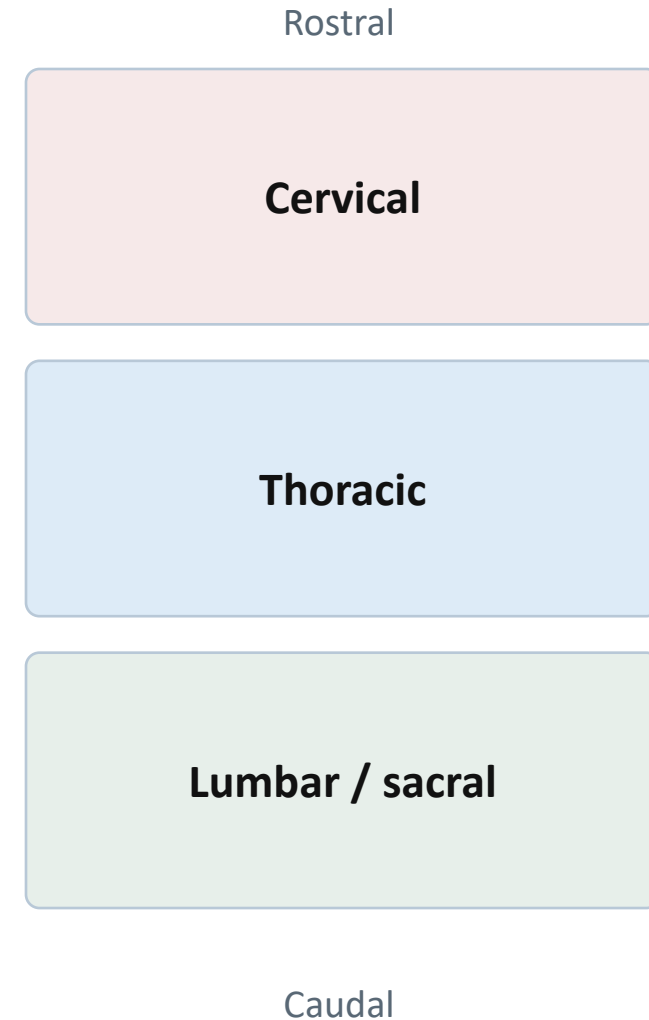
Lesion level determines distribution

- Neurological manifestations depend strongly on the rostrocaudal level of injury.
- Higher lesions place more descending/ascending pathways and segmental functions at risk.
- Interpret the affected vertebra separately from the neurological level of injury.



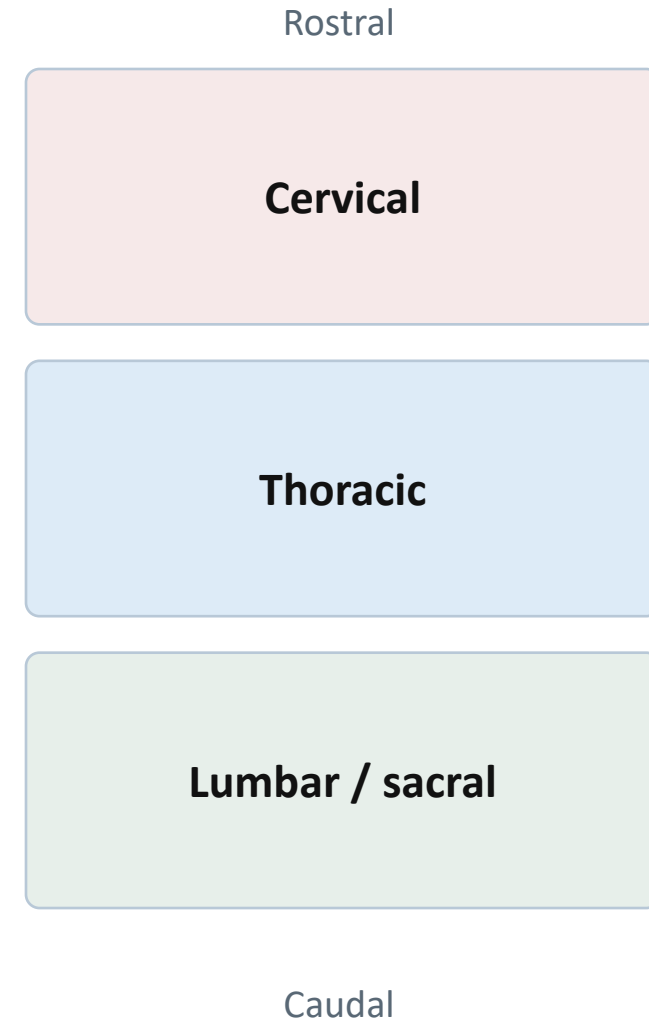
Cervical injury: Tetraplegic pattern

- Cervical cord injury can impair both upper and lower limbs.
- Degree of arm/hand involvement varies with the specific segment and spared pathways.
- High cervical injury may also compromise breathing.



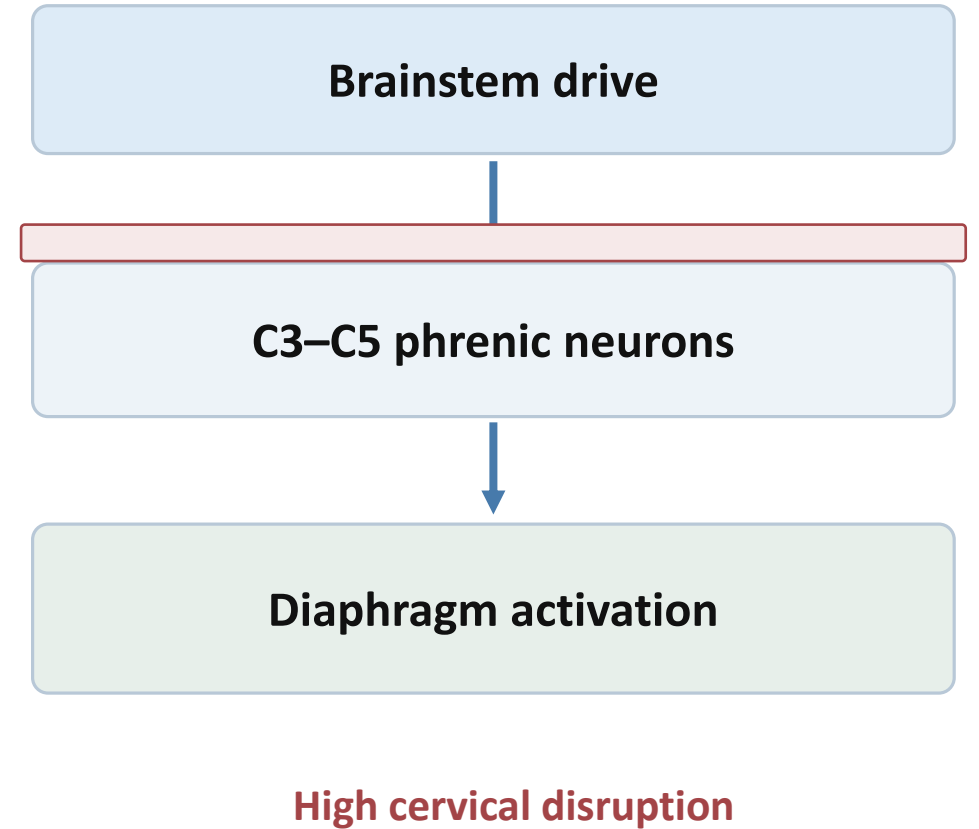
Thoracic injury: Paraplegic pattern

- Thoracic-region cord injury typically spares motor pathways serving the arms.
- Legs and trunk functions below the lesion may be impaired.
- Autonomic and sensory deficits may accompany paralysis.



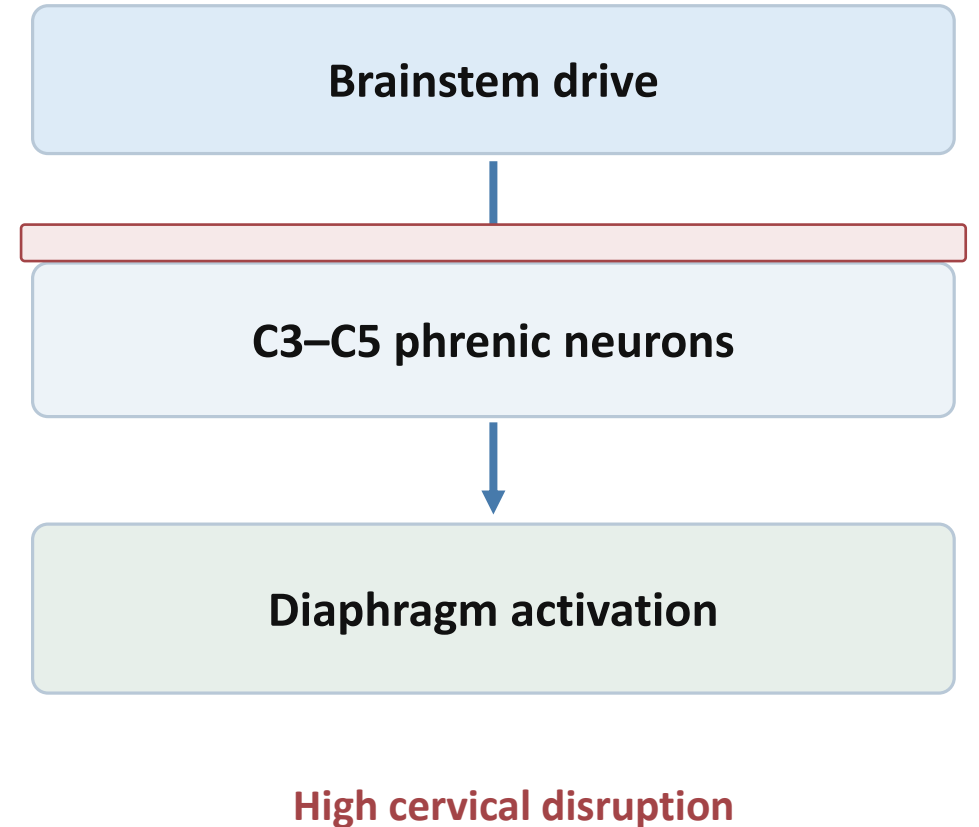
Above C4: The respiratory hazard

- The possibility of respiratory compromise when injury is above C4.
- Disrupted descending drive to the phrenic motor system can impair diaphragm function.
- Respiratory evaluation is therefore central to acute localization and safety.



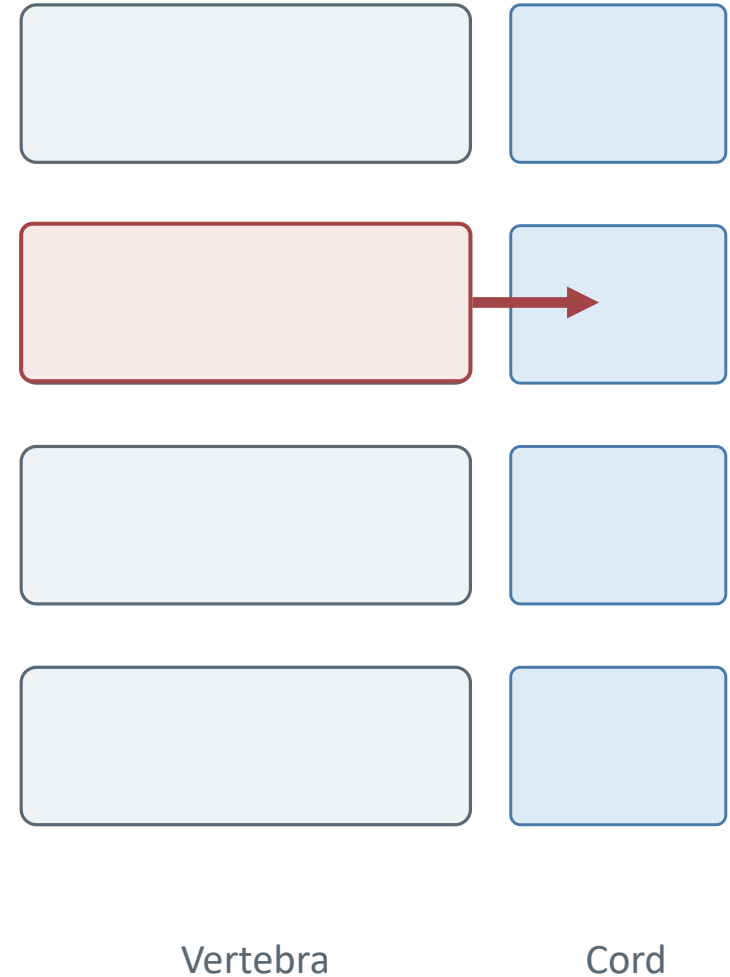
Phrenic motor output: C3–C5

- Phrenic motor neurons receive segmental contributions largely from C3–C5.
- Very high cervical lesions can interrupt descending control of diaphragm activation.
- The neurological deficit is determined by neural damage, not vertebral numbering alone.



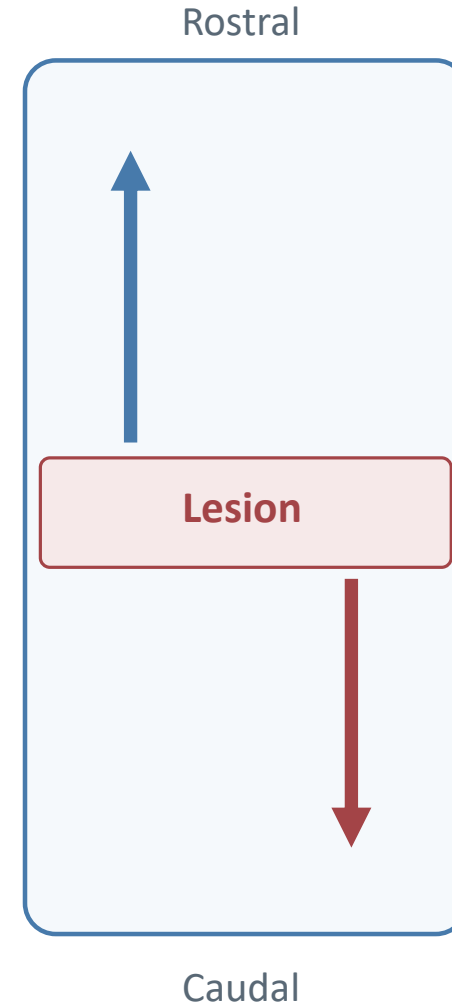
Vertebral level $\neq$ cord segment

- The bony vertebral level and the neurological spinal cord segment are not always identical.
- This distinction is particularly important in the lower spine.
- Report imaging anatomy and neurological examination as related but distinct findings.



Loss of function below the injury

- Descending motor output may fail to reach otherwise intact caudal motor neurons.
- Ascending sensory traffic may fail to cross the injured level.
- Below-level impairment need not mean uniform necrosis throughout the distal cord.



Complete and incomplete lesions

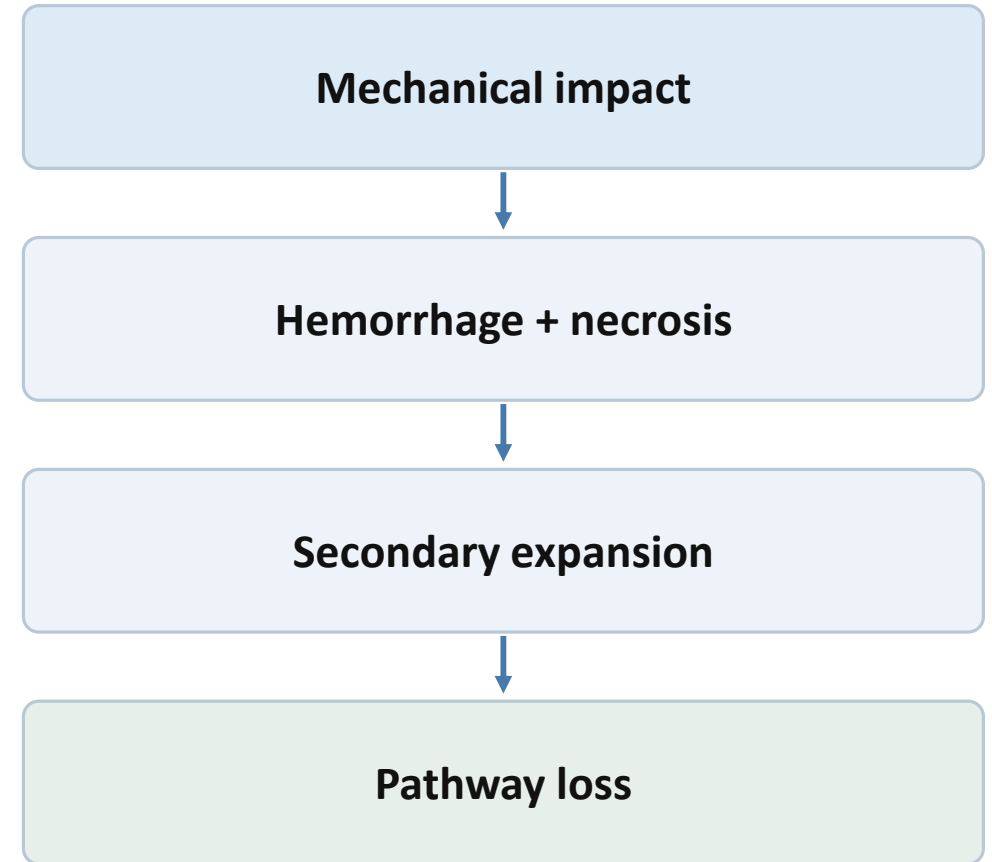
- A neurological injury can abolish all tested sacral sensory and motor function or preserve some pathways.
- Apparent severe weakness does not alone establish neurological completeness.
- Standardized classification requires sacral testing rather than inspection of a cord image.

Complete: No sacral sparing

Incomplete: Sacral sparing

Primary versus secondary tissue injury

- Primary injury is the direct mechanical destruction at impact.
- Secondary injury evolves through ischemia, edema, excitotoxicity, oxidative stress, and inflammation.
- Secondary events help explain why the pathologic field may exceed the initial mechanical focus.



A morphological time sequence

- Acute: Hemorrhage, necrosis, and axonal swelling at the lesion site.
- Evolution: Inflammatory clearance and loss of destroyed parenchyma.
- Chronic: Central cystic cavitation, reactive gliosis, and distant tract degeneration.

Acute
Hemorrhage / necrosis



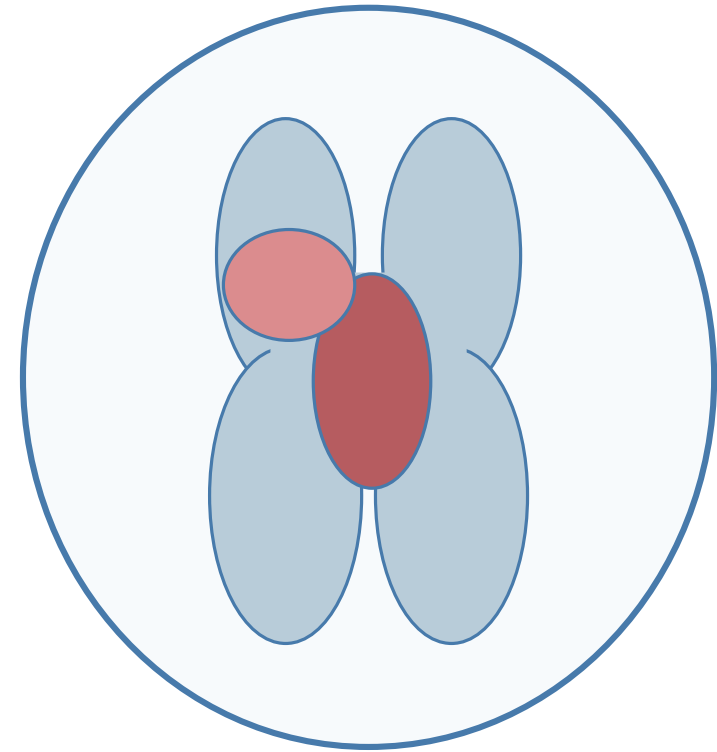
Evolving
Debris clearance



Chronic
Cavity + gliosis

Acute hemorrhage within the cord

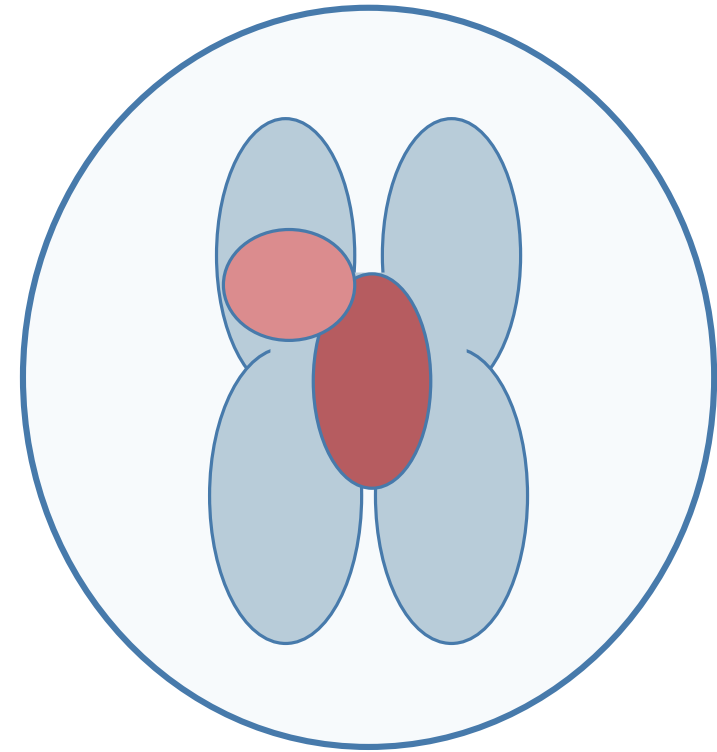
- Vessel disruption introduces blood into injured spinal parenchyma.
- Hemorrhage marks damage near the lesion epicenter and may coexist with edema.
- Blood extravasation is a genuine morphologic finding—not equivalent to complete transection.



Schematic transverse cord

Acute necrosis at impact

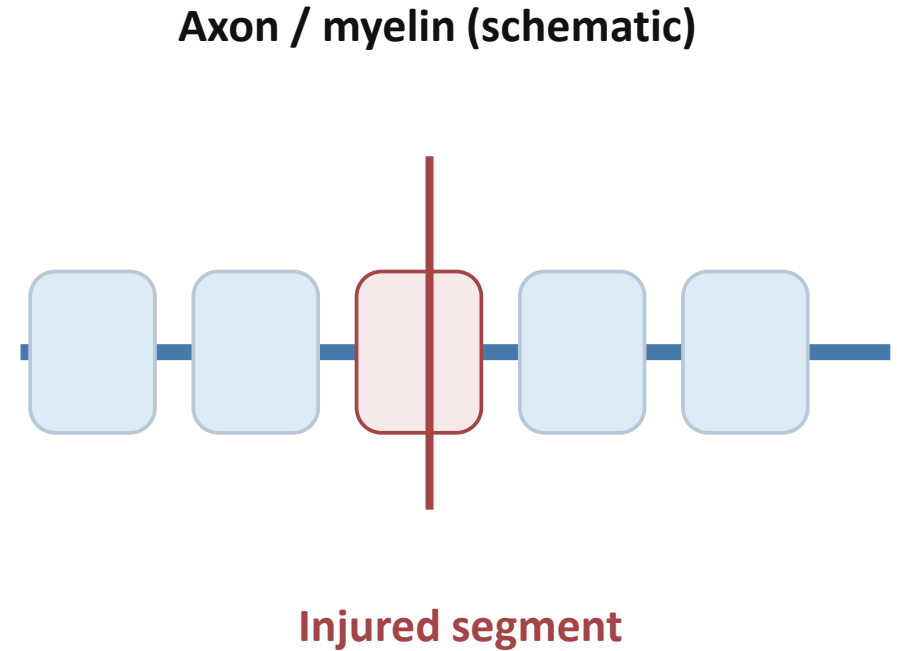
- Direct mechanical and vascular injury can destroy neurons and glia.
- Necrosis eliminates functional tissue rather than merely suppressing impulse conduction.
- The acute lesion can involve both central gray matter and nearby white matter.



Schematic transverse cord

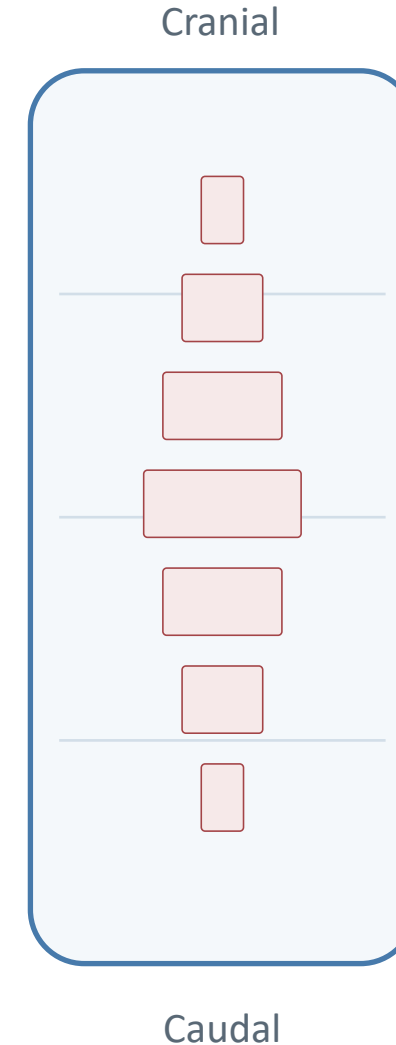
Axonal swelling in surrounding white matter

- Swollen axons in white matter around the acute injury.
- Axonal swelling signals disrupted axonal integrity and transport.
- Surrounding tract injury may be wider than the focus of frank hemorrhage.



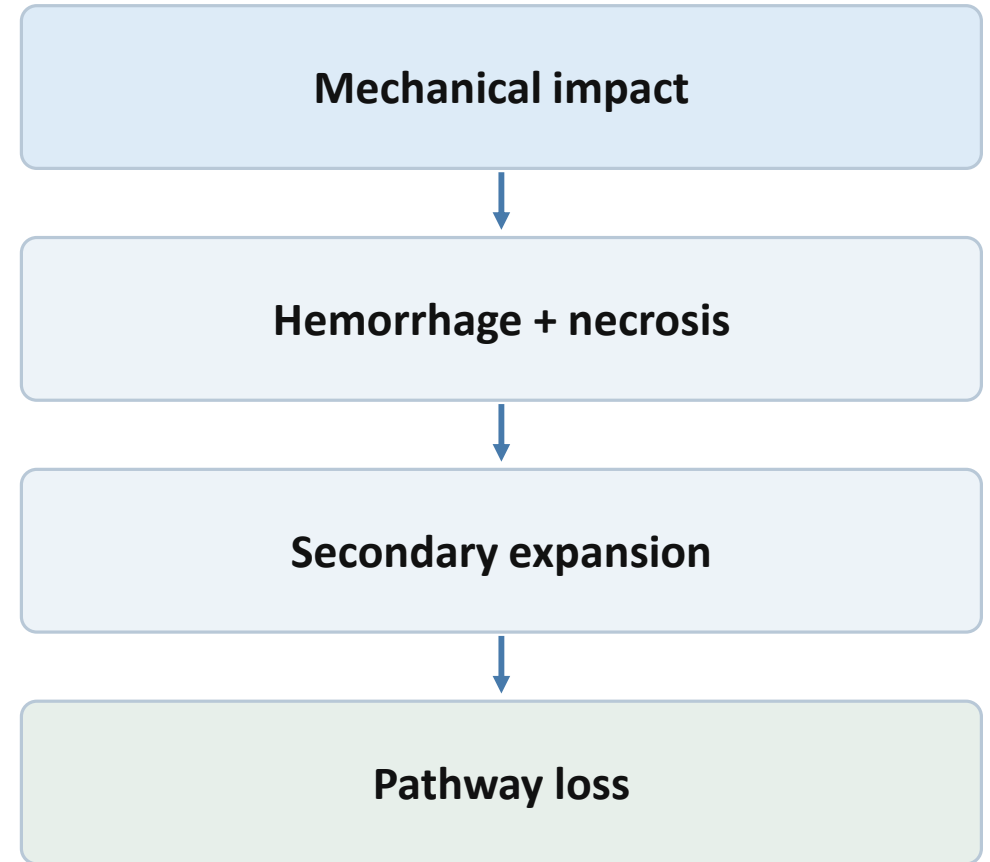
Spatial tapering away from the epicenter

- The greatest structural destruction is typically centered on the impact zone.
- Histological severity decreases in cord sections farther above or below the focus.
- Distant degeneration follows tract direction and should be distinguished from the local injury field.



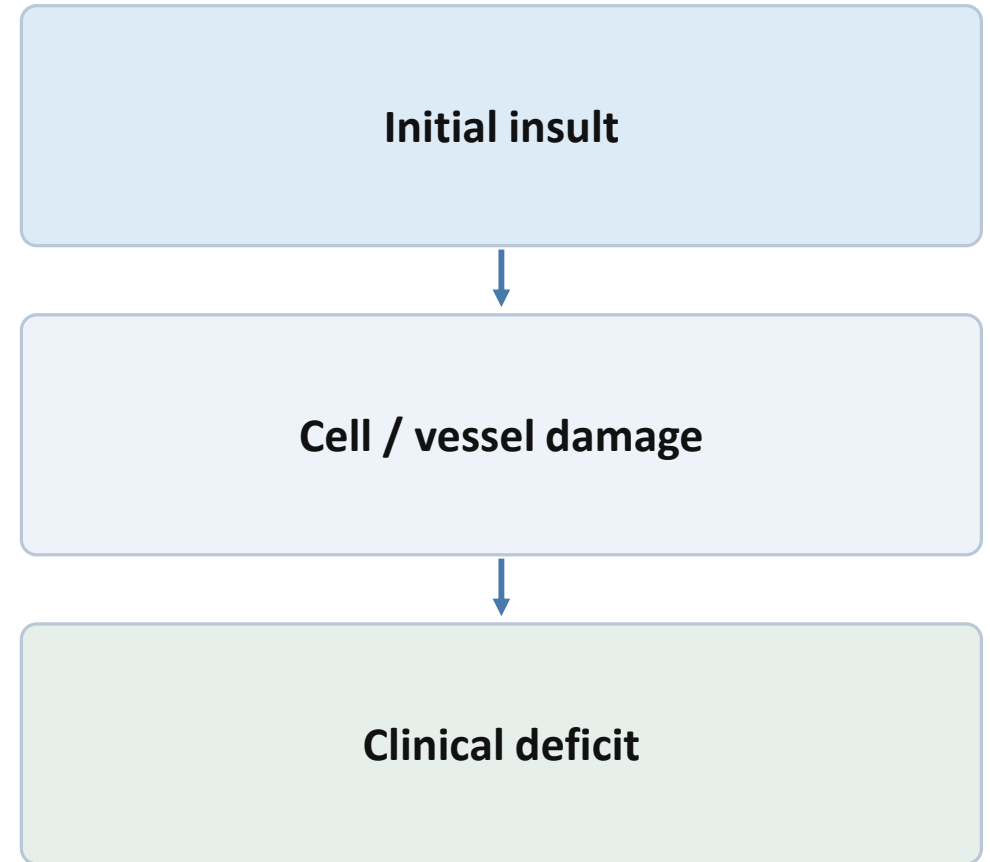
Secondary hypoperfusion and ischemia

- Microvascular injury and tissue swelling can worsen delivery of oxygen and substrates.
- Ischemic stress promotes further neuronal and glial dysfunction.
- A mechanically small lesion can have a biologically amplified injury zone.



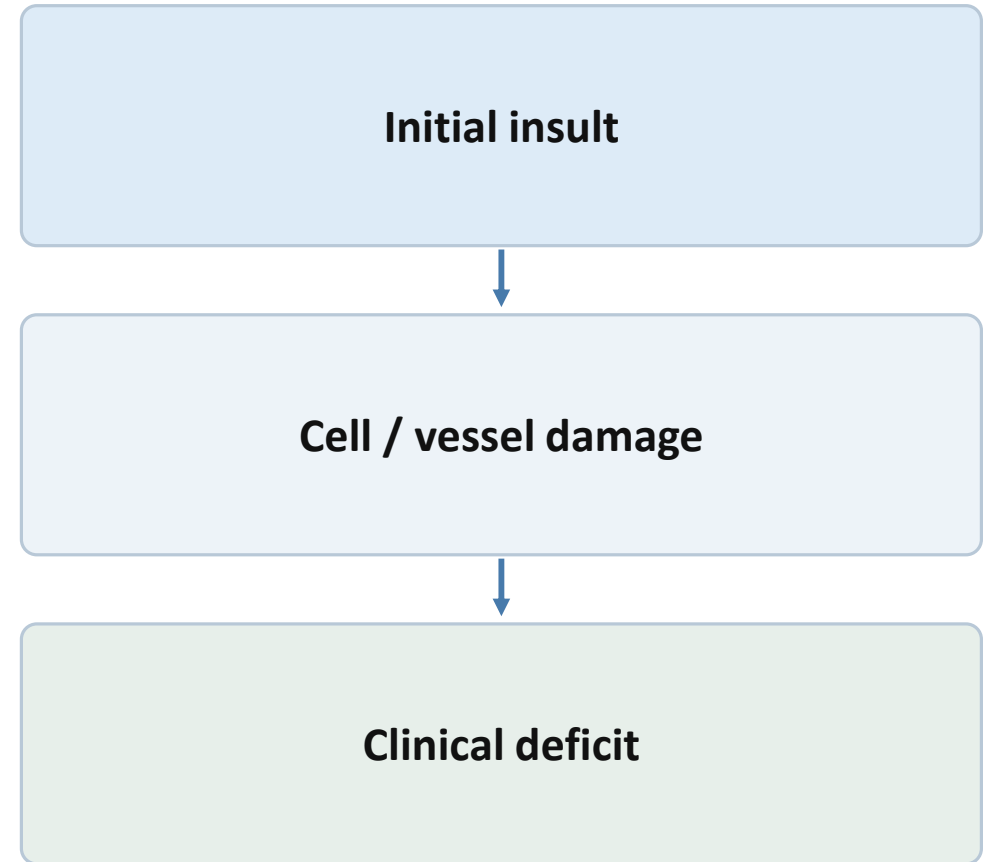
Excitotoxic signaling

- Injured neural tissue may release excess excitatory neurotransmitters.
- Ionic dysregulation and calcium overload can intensify cellular damage.
- This is a secondary-injury mechanism, not a directly visible histologic diagnosis.



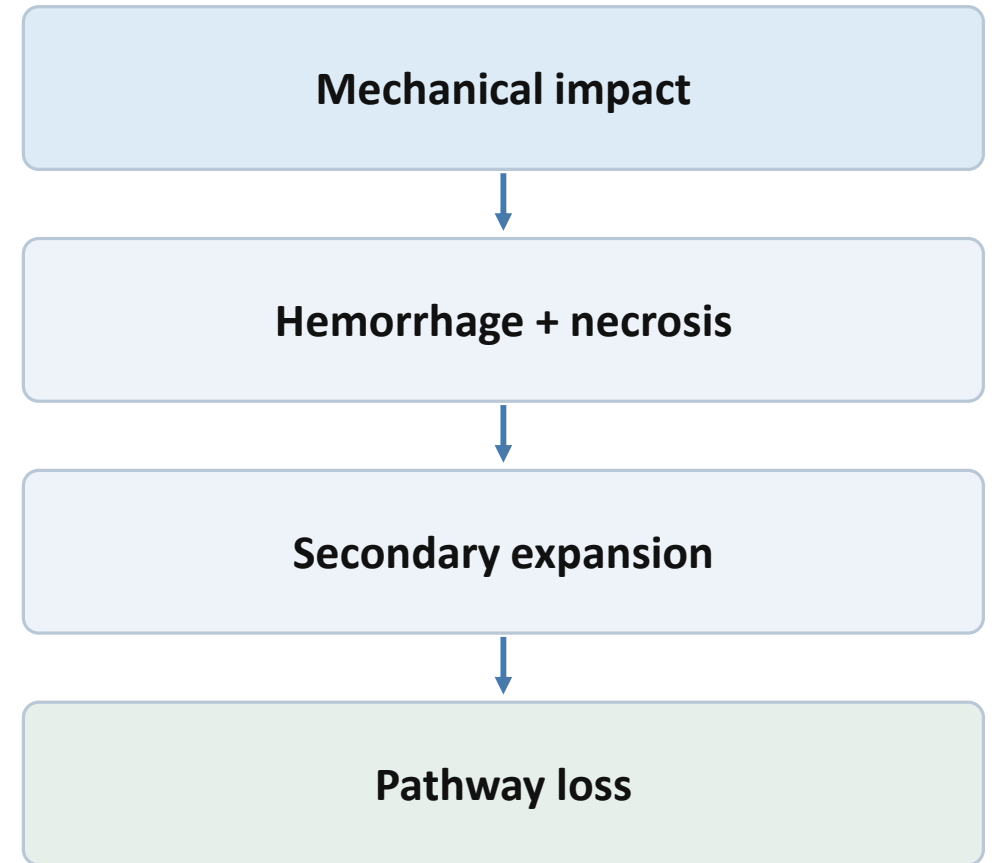
Oxidative injury

- Secondary inflammatory and ischemic processes generate reactive oxygen species.
- Oxidative stress can injure membranes, proteins, and neural cells.
- It contributes to secondary injury but is not identified by routine H&E alone.



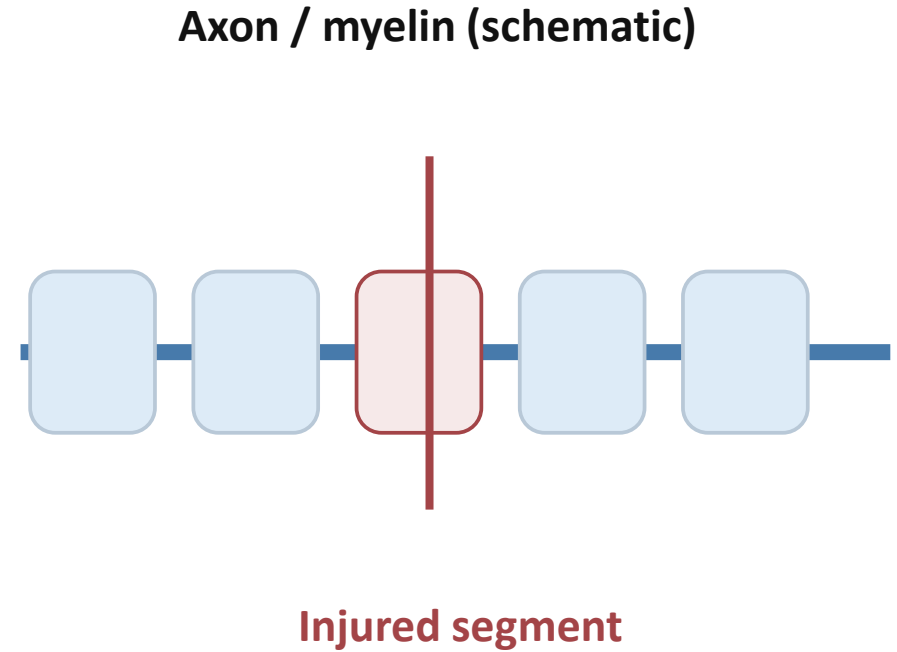
Inflammation after traumatic necrosis

- Resident microglia and infiltrating phagocytes respond to damaged tissue.
- Inflammation removes debris but can also amplify local tissue injury.
- The response evolves as acute necrosis transitions toward gliosis and cavitation.



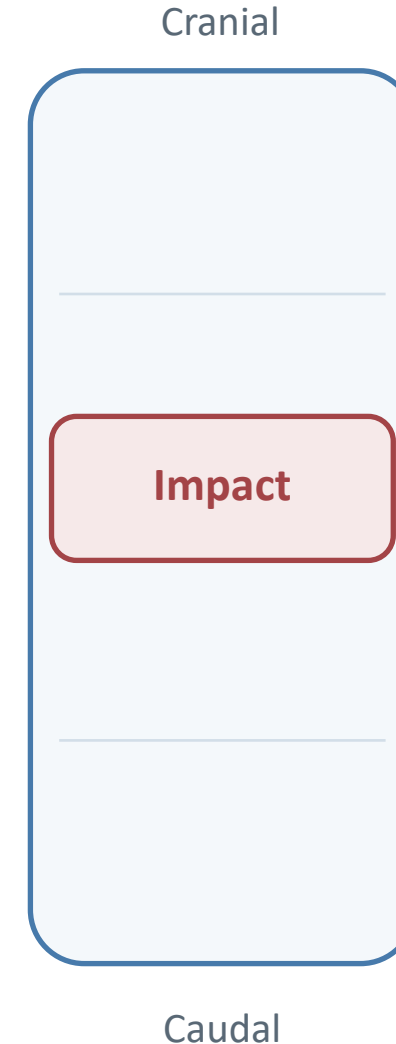
Oligodendrocyte loss and demyelination

- Oligodendrocytes maintain myelin around multiple CNS axons.
- Secondary cell injury can impair surviving axons by loss of myelin.
- Axonal continuity and myelin integrity are related but distinct determinants of conduction.



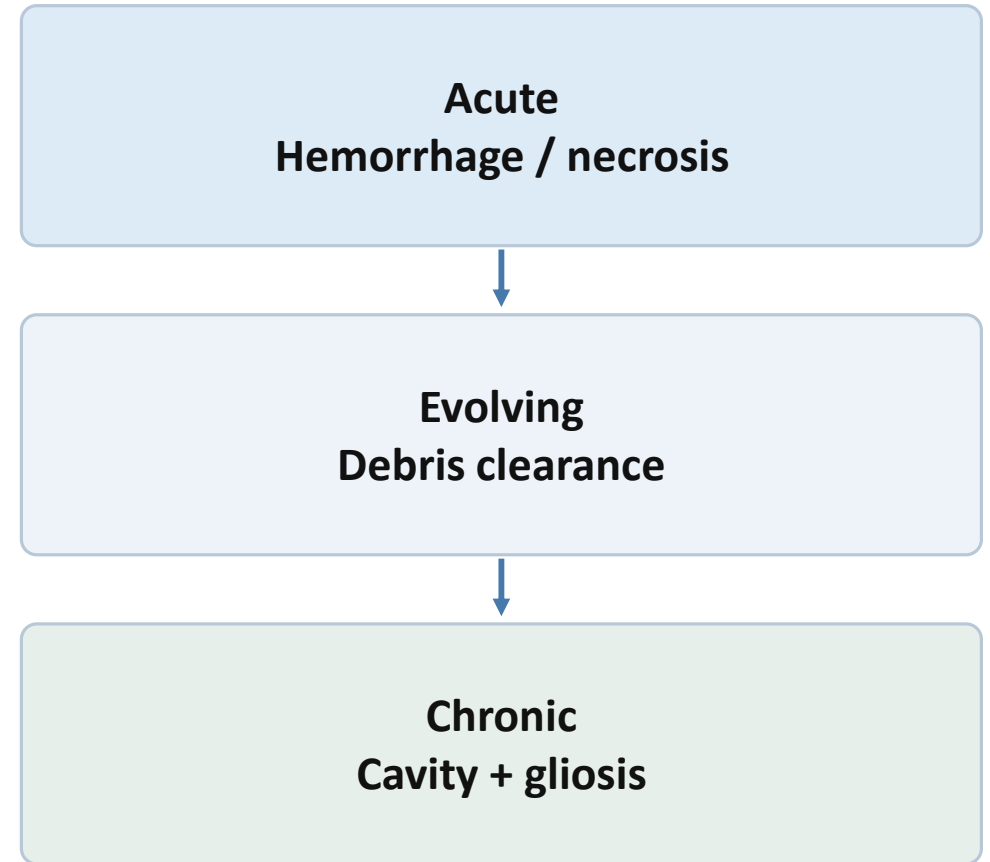
Edema expands functional injury

- Swelling increases tissue pressure in an anatomically constrained compartment.
- Edema may compromise microvascular flow and impulse conduction.
- Imaging-visible edema cannot on its own establish irreversible neuronal loss.



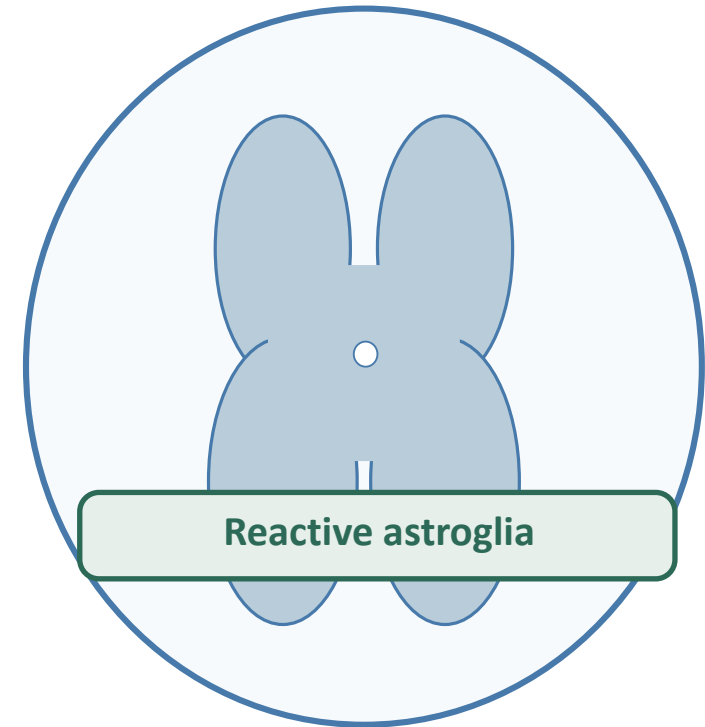
Phagocytic removal of necrotic tissue

- Microglia and blood-derived macrophages clear fragmented cell and myelin debris.
- Macrophage-rich tissue is part of injury evolution, not an independent disease.
- The removed necrotic center may later appear as a cystic cavity.



Astrocytic reactivity and gliosis

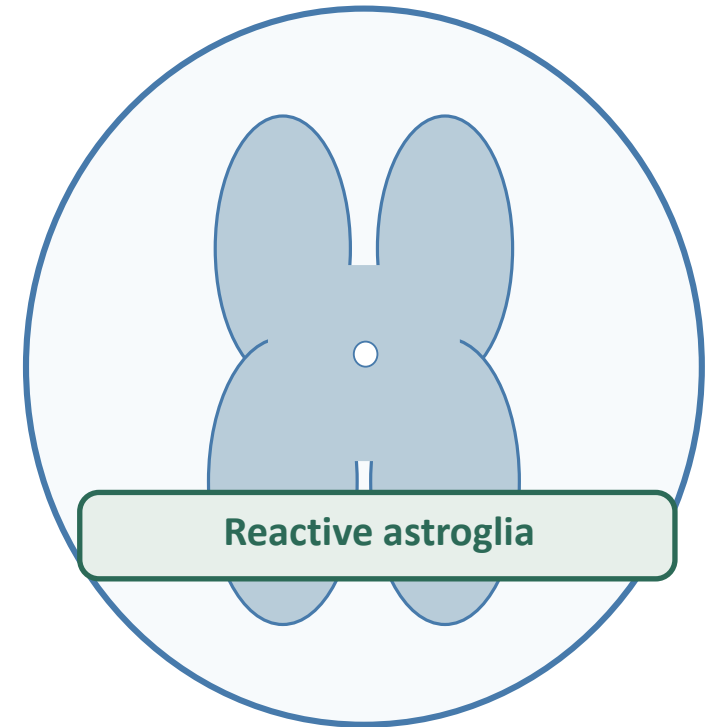
- Reactive astrocytes hypertrophy and proliferate around injured tissue.
- Gliosis is a major histopathologic marker of CNS injury.
- A gliotic rim marks a mature tissue response rather than restored neuronal circuitry.



Schematic transverse cord

GFAP as an astroglial marker

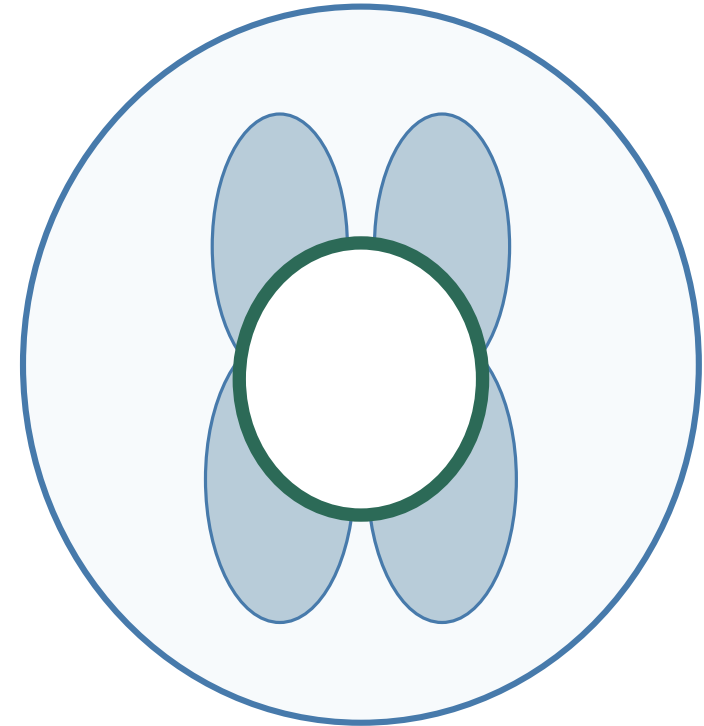
- Astrocytic processes express glial fibrillary acidic protein (GFAP).
- GFAP immunostaining can highlight the reactive astroglial border of chronic lesions.
- Positive astroglial staining supports gliosis; It is not specific for traumatic etiology.



Schematic transverse cord

Central cavitation in chronic lesions

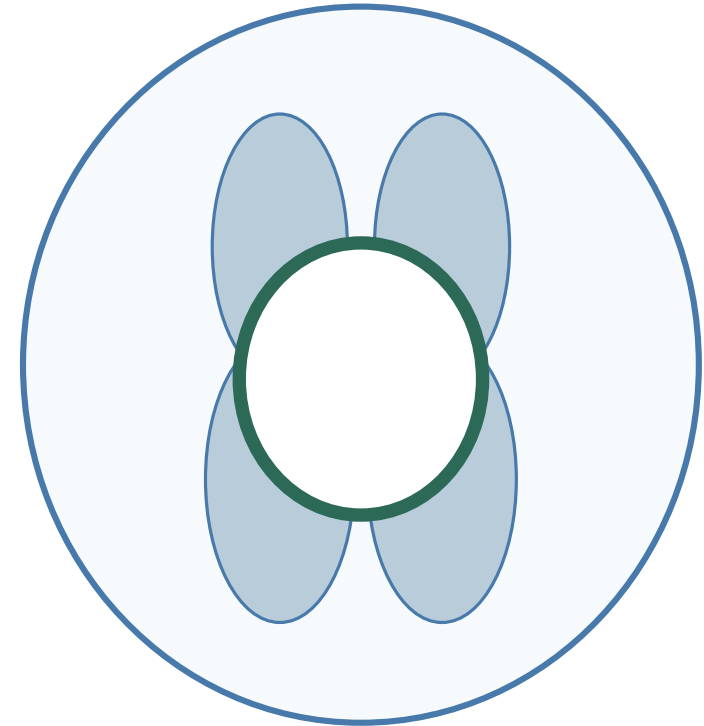
- Areas of destroyed spinal parenchyma may become cystic over time.
- The cystic center reflects tissue loss, rather than formation of normal cord tissue.
- Cavitation commonly coexists with surrounding gliosis.



Schematic transverse cord

Cystic cavity versus gliotic rim

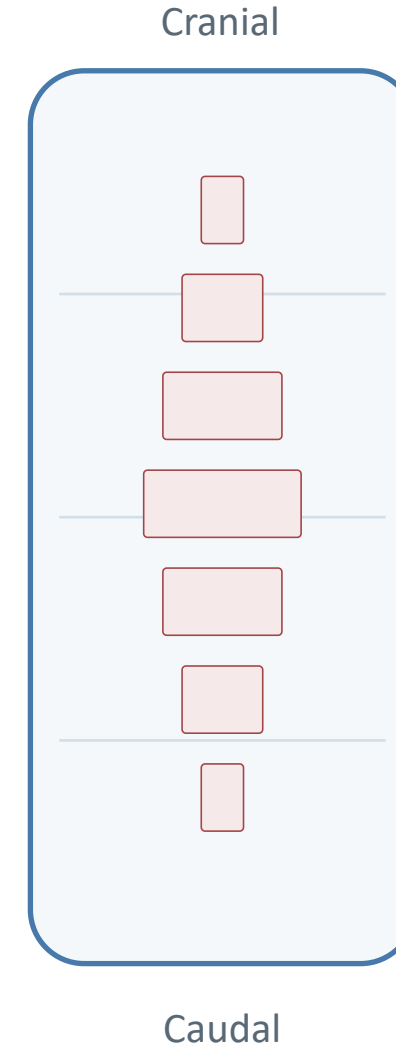
- Central compartment: Absence of previously viable neural tissue.
- Peripheral response: Astrocytic gliosis at the lesion margin.
- A mature spinal lesion therefore contains both a negative space and a reactive cellular border.



Schematic transverse cord

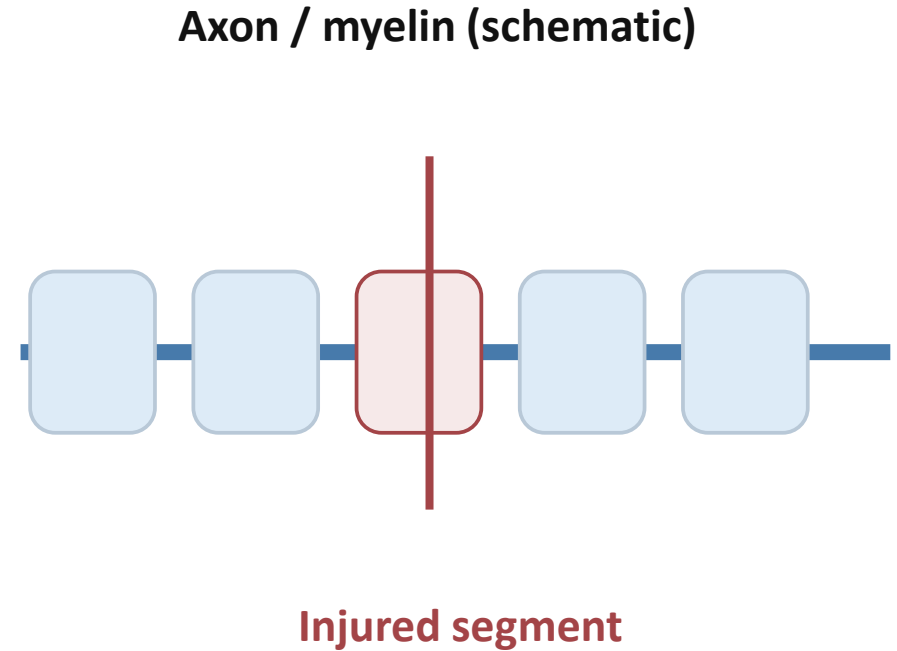
Late changes are not uniform along the cord

- The local lesion remains centered near the injury site.
- Distal degeneration tracks along specific affected white-matter bundles.
- Serial sections are more informative than a single cross-section.



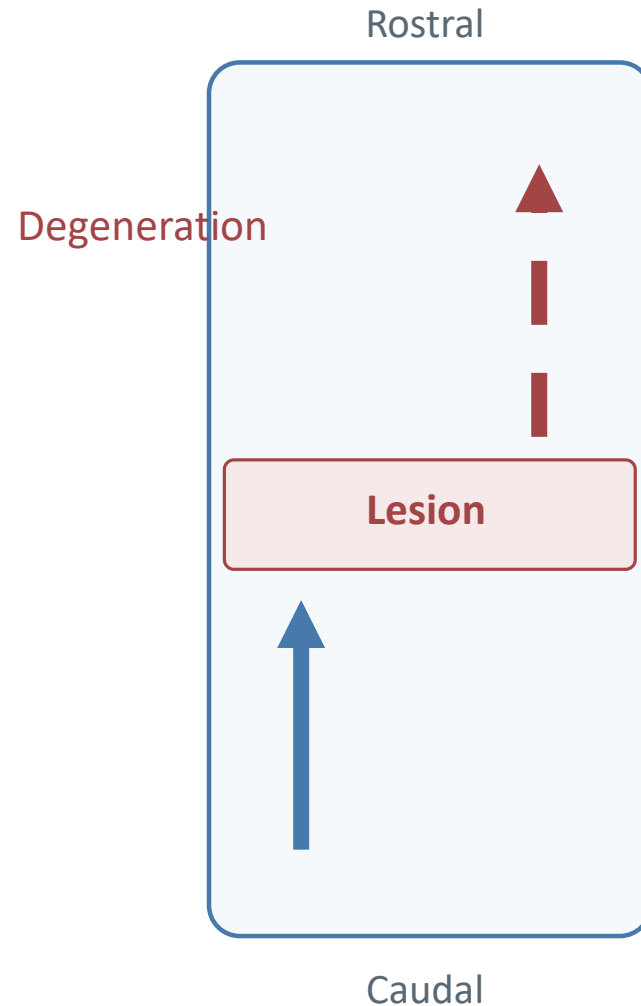
Wallerian degeneration: Definition

- An axon segment disconnected from its neuronal cell body undergoes degeneration.
- The degenerative process can extend over a long distance from the primary lesion.
- Wallerian degeneration is a secondary axonal consequence, not a second traumatic impact.



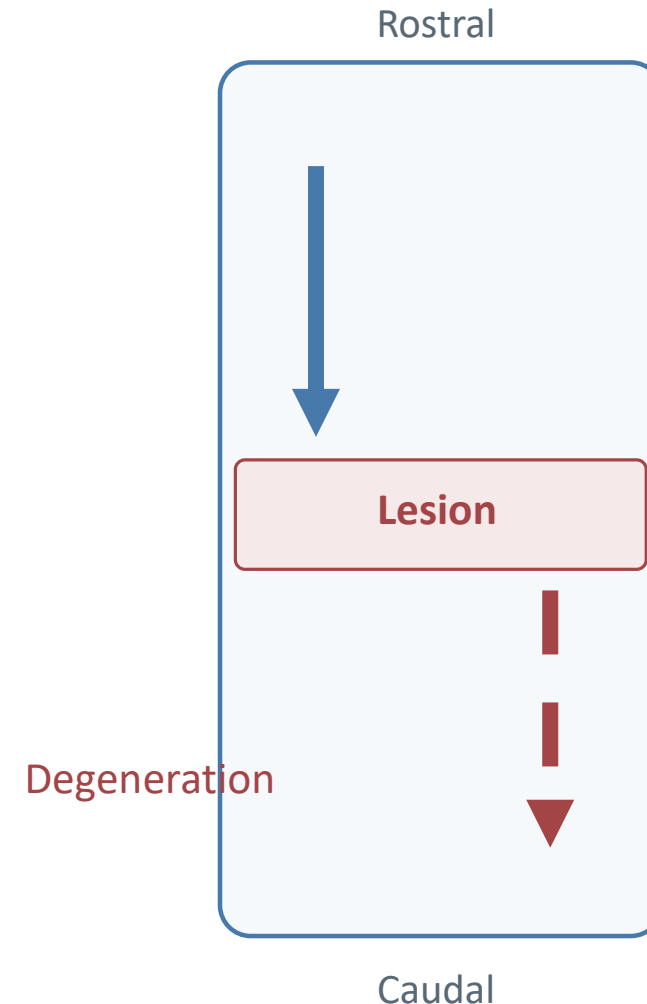
Ascending tract degeneration above injury

- Sensory pathways originate in neurons located caudally and project rostrally.
- When such axons are severed, their rostral distal segments degenerate above the lesion.
- Robbins specifically describes secondary ascending Wallerian degeneration above the focus.



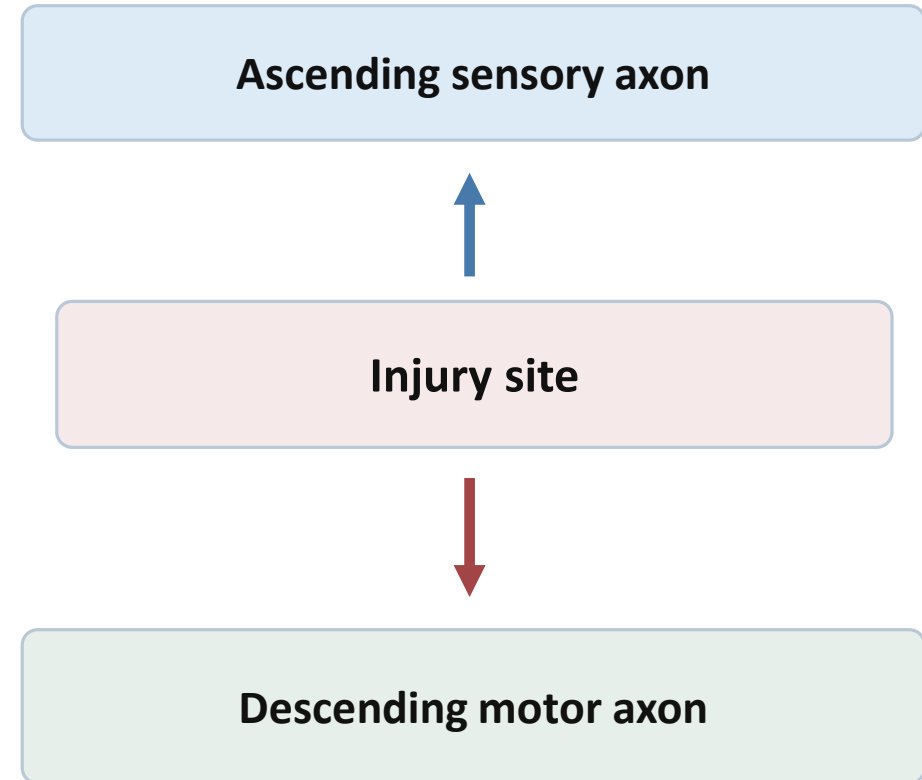
Descending tract degeneration below injury

- Many motor pathways arise rostrally and descend toward spinal levels.
- Their distal disconnected segments lie caudal to the injury.
- Descending Wallerian degeneration below the lesion.



Why “distal” changes anatomical direction

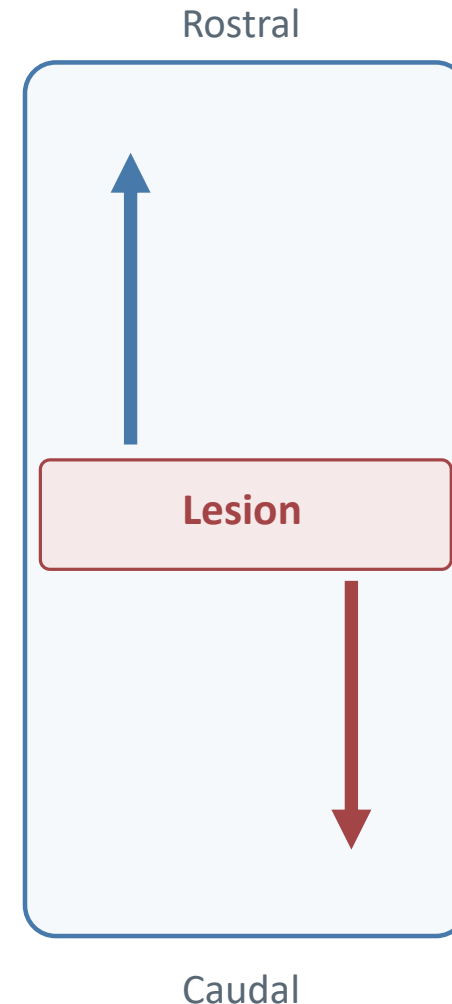
- Distal is defined relative to the neuron’s cell body, not simply “below the lesion.”
- Ascending sensory axons: Distal disconnected segment can be rostral.
- Descending motor axons: Distal disconnected segment is usually caudal.



Distal = away from each neuron’s soma

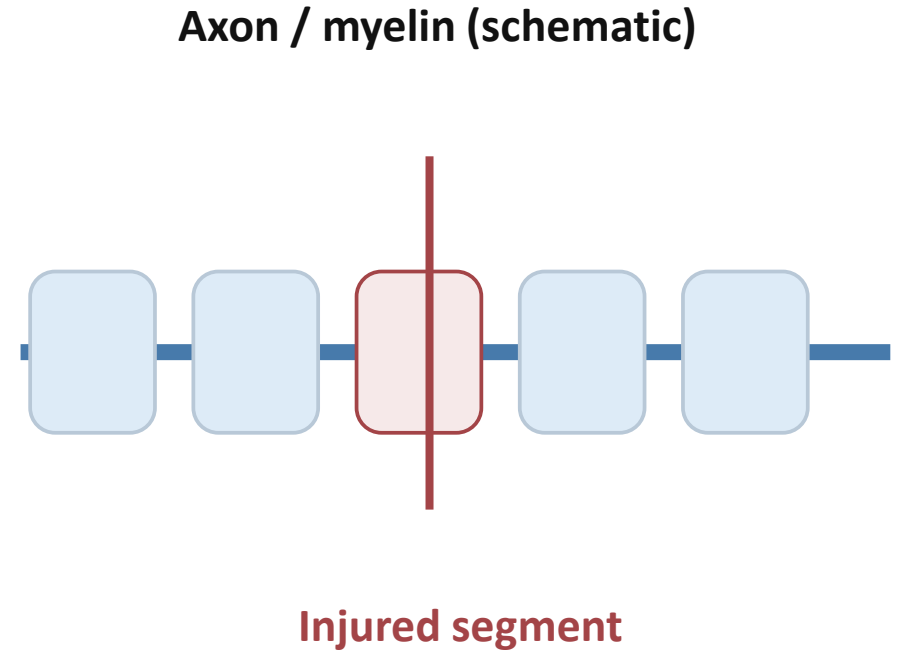
Tract-specific pattern is a localization clue

- Degeneration respects anatomic tracts rather than extending indiscriminately.
- The side, tract position, and direction of degeneration may help reconstruct injured pathways.
- Interpret degenerating tracts in relation to known axonal projection direction.



White-matter pallor and myelin breakdown

- Degenerating axons are accompanied by degeneration of their myelin sheaths.
- Chronic affected bundles may show reduced myelin staining and tract atrophy.
- Histologic tract changes complement, rather than replace, neurological localization.



CNS versus peripheral nerve repair

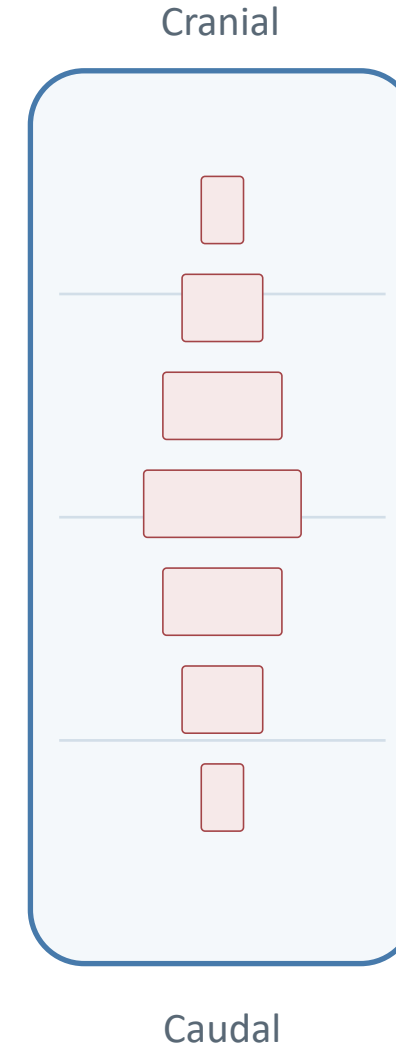
- Peripheral nerve axonal regeneration can be supported by Schwann cell pathways.
- Spinal cord long tracts do not routinely recover by equivalent regeneration.
- Gliosis, tissue cavitation, and disrupted long connections complicate restoration of function.

CNS: Oligodendroglia

PNS: Schwann cells

Microscopic sampling strategy

- Examine a section through the lesion epicenter for hemorrhage and necrosis.
- Compare serial sections immediately rostral and caudal to assess injury taper.
- Examine distant white-matter tracts for patterned secondary degeneration.



Interpreting tissue stains in context

- H&E: Cellular injury, hemorrhage, necrotic debris, and evolving cavity.
- Myelin stains: Affected tract pallor or myelin loss when used appropriately.
- GFAP: Reactive astroglial response in chronic margins.
- Interpret morphology with anatomical level and time after trauma.

H&E: Tissue architecture

Myelin stain: Tracts

GFAP: Astroglia

Acute and chronic morphology: Comparison

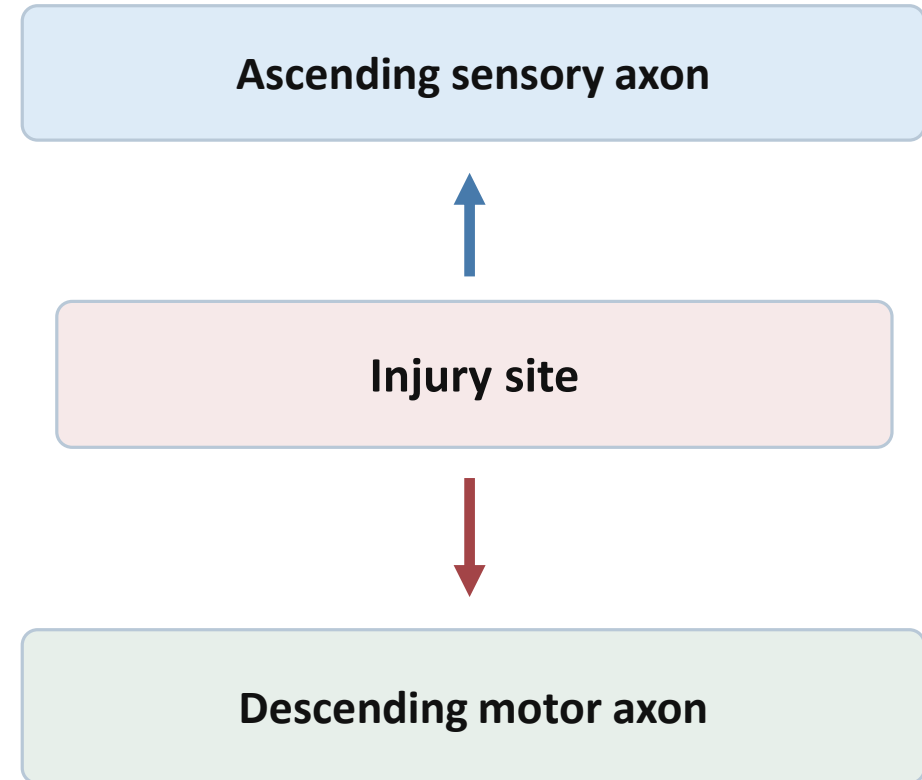
- Acute injury emphasizes hemorrhage, necrosis, and axonal swelling.
- Chronic injury emphasizes cavitation, gliosis, and long-tract degeneration.
- The two patterns may overlap in evolving lesions.

Acute: Hemorrhage / necrosis

Chronic: Gliosis / cavity

Loss of conduction versus structural loss

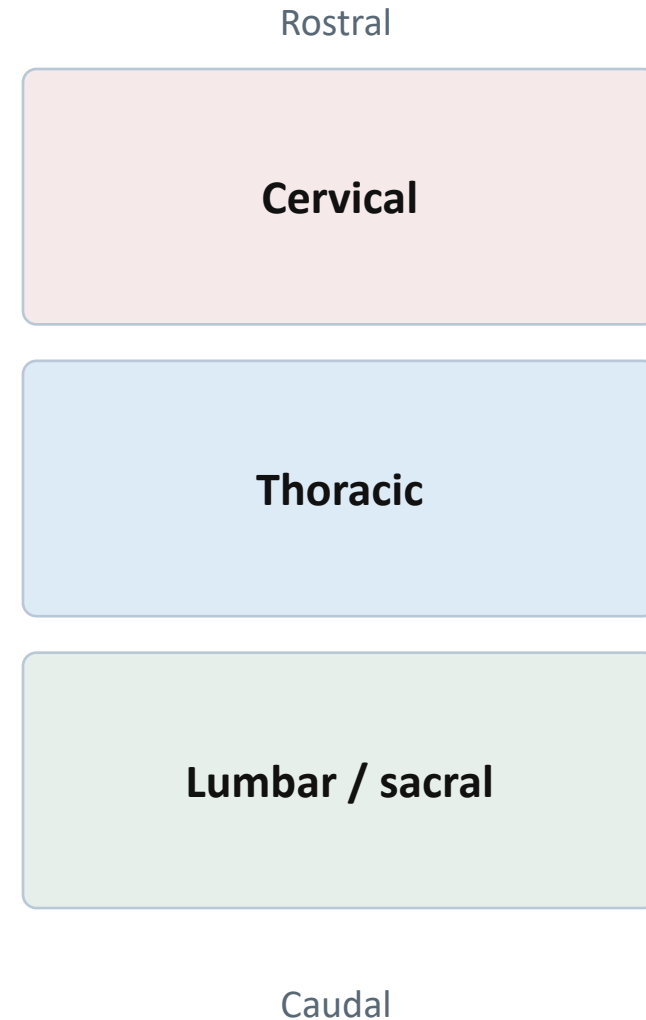
- Neurologic dysfunction can arise from interrupted conduction in injured pathways.
- Histologic necrosis demonstrates irreversible tissue destruction in the sampled area.
- Do not infer the degree of viable axonal sparing from a single imaging sequence or thin section.



Distal = away from each neuron's soma

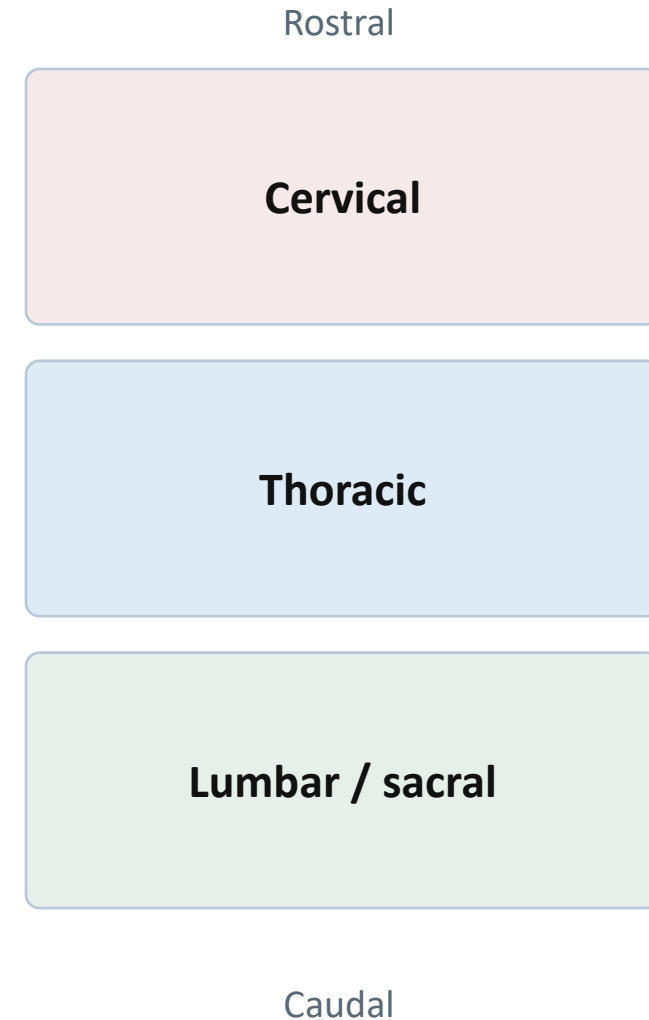
Paraplegia: Clinicopathologic mapping

- A thoracic cord lesion may interrupt descending pathways serving the legs.
- Upper-extremity motor circuits remain rostral to the injury.
- Sensory and autonomic deficits below the level provide additional localization.



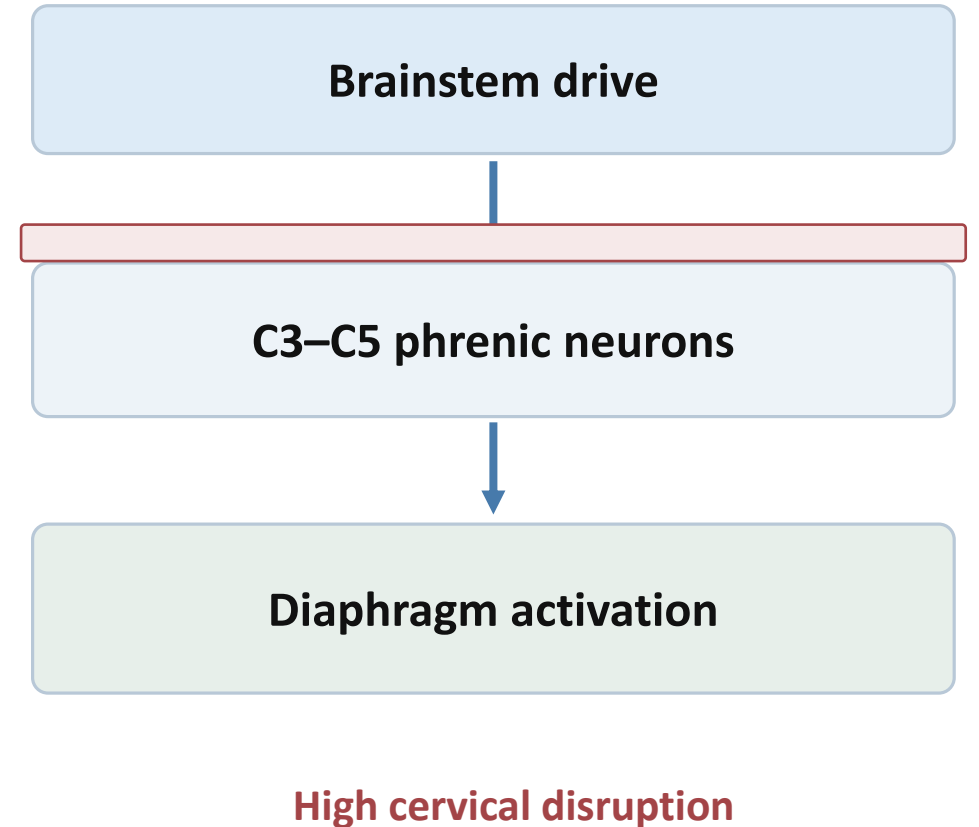
Tetraplegia: Clinicopathologic mapping

- Cervical lesions can disconnect spinal circuitry supplying all four limbs.
- Segmental cervical gray-matter damage can add focal upper-limb weakness.
- Functional outcome depends on level, severity, and preserved axonal pathways.



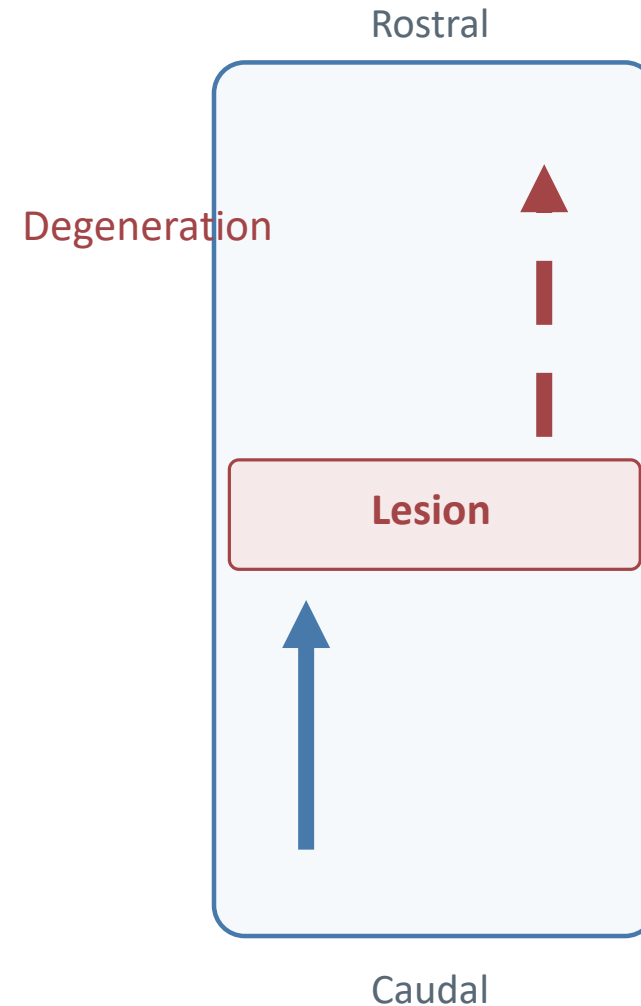
Respiratory compromise: Mechanism

- High cervical injury may interrupt brainstem drive to phrenic motor neurons.
- Diaphragmatic paralysis can threaten ventilation.
- A neurological deficit above C4 deserves particular attention to breathing.



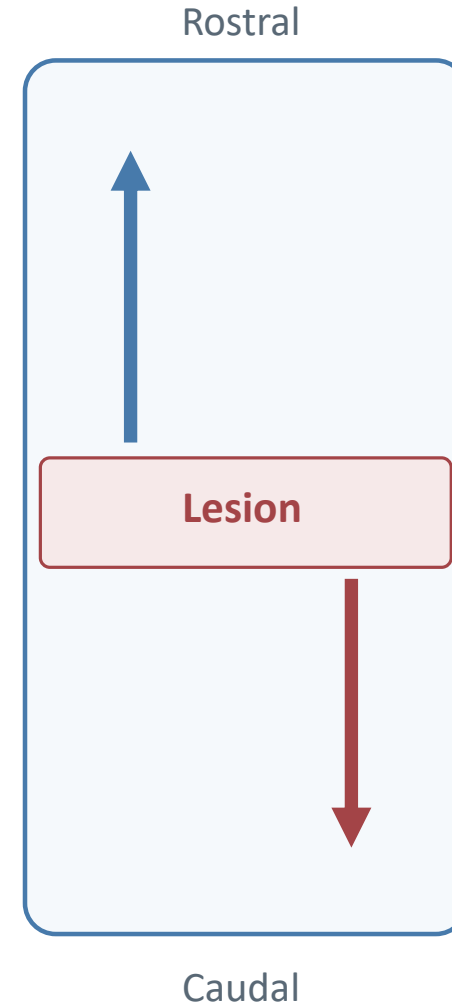
Sensory level and tract interruption

- A reproducible sensory level can suggest the segment where ascending input fails.
- Different sensory modalities sample different ascending pathways.
- Clinical sensory mapping and imaging must be interpreted together.



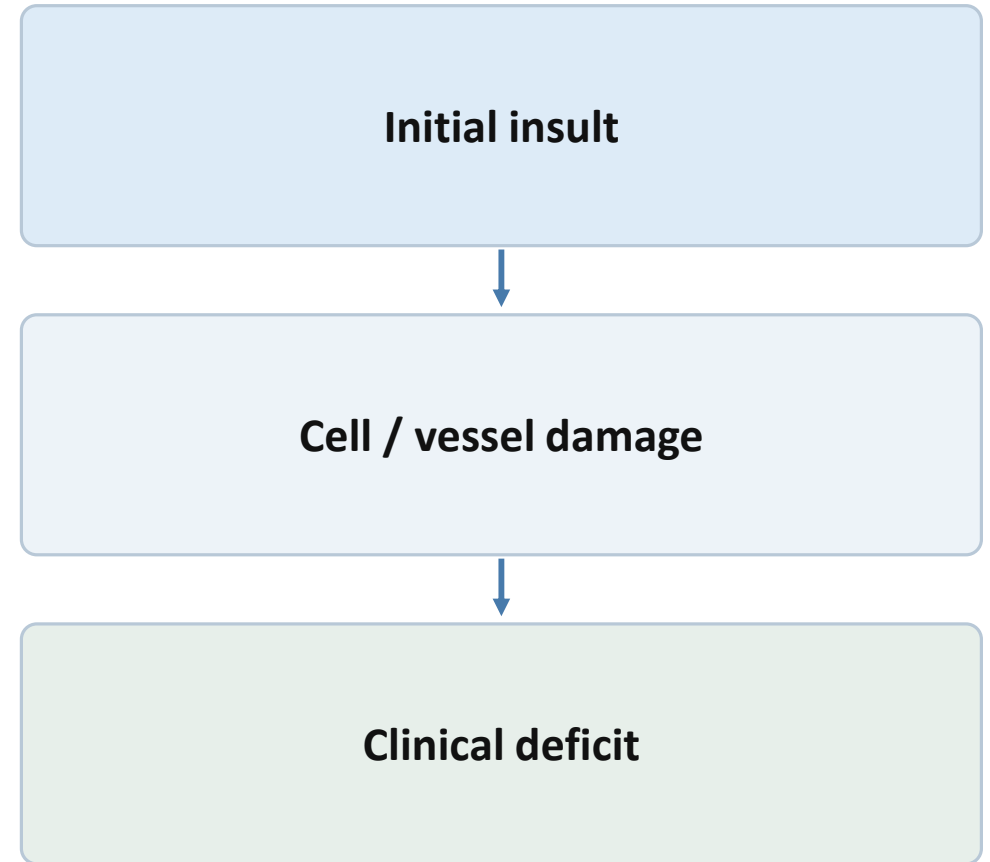
Bladder and bowel dysfunction

- Spinal cord lesions may disrupt descending regulation of autonomic and somatic circuits.
- Dysfunction may persist even when the directly damaged segment is rostral to pelvic organs.
- These consequences reflect pathway disconnection, not direct visceral trauma.



Autonomic dysfunction after cord injury

- Descending autonomic connections can be disrupted by injury.
- Severe cord injury can affect blood-pressure regulation, sweating, and other autonomic functions.
- Autonomic findings add clinical context but are not a substitute for motor/sensory examination.



Spinal shock versus structural completeness

- Early loss of reflex activity may obscure the extent of preserved pathways.
- Spinal shock is a physiological syndrome; Anatomical injury is a separate question.
- Classify neurological completeness with standardized examination, including sacral assessment.

Physiological suppression

Structural tissue loss

Clinical examination: What it establishes

- Determine segmental motor function, sensory function, and sacral preservation.
- Document right–left asymmetry and neurological level systematically.
- Repeat examinations can characterize clinical evolution after acute injury.

Mechanical insult

Local tissue loss

Tract disconnection

Functional deficit

Neuroimaging: Complementary questions

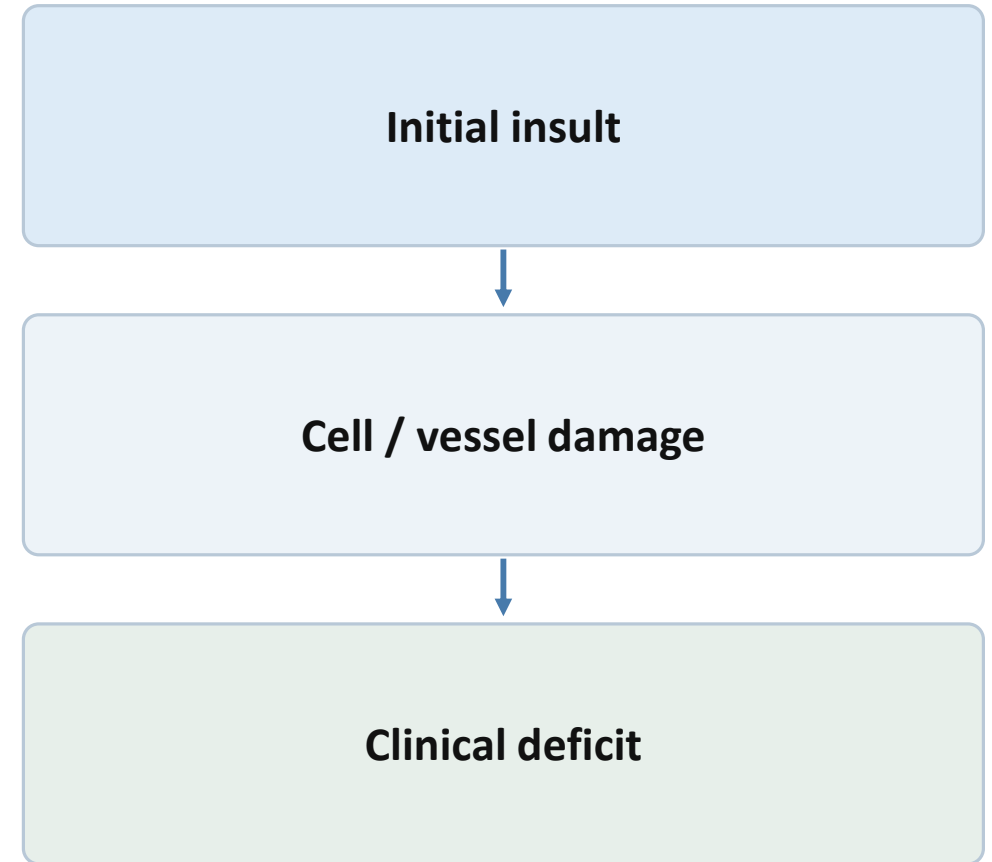
- CT helps evaluate bony injury and alignment after trauma.
- MRI is better suited to depicting cord edema, hemorrhage, compression, and soft tissues.
- Imaging morphology complements, but does not replace, neurological examination.

CT: Vertebral anatomy

MRI: Cord / soft tissues

ISNCSCI: Current classification framework

- The International Standards formalize sensory and motor examination after SCI.
- They define neurological level and completeness using standardized criteria.
- The current cited edition is the 9th edition (2026); Use its official worksheet for scoring.



Sacral sparing and neurological completeness

- Preserved function in S4–S5 indicates neurological incompleteness.
- Absent sacral sensory and motor function is central to the definition of complete SCI.
- Structural continuity on MRI is not itself the definition of neurological completeness.

Complete: No sacral sparing

Incomplete: Sacral sparing

Structural mimics and diagnostic boundaries

- Traumatic vertebral injury does not automatically establish spinal cord parenchymal damage.
- Spinal nerve-root injury and compressive lesions can produce overlapping deficits.
- The lesion must be localized using clinical examination and anatomically appropriate imaging.

Cord / central injury

Root / extra-axial injury

Clinical–imaging correlation

Case 1: Thoracic impact with leg paralysis

- Scenario: Vertebral displacement near the thoracic spine followed by marked bilateral leg weakness.
- What is the most likely mechanism of paralysis?
- Teaching interpretation: Interruption of descending motor pathways with local spinal damage.
- Do not assume the precise completeness grade from weakness alone.

Clinical clue

Likely lesion

Mechanism

Important limit

Case 2: High cervical injury and ventilation

- Scenario: High cervical trauma, severe limb weakness, and inadequate diaphragmatic movement.
- Which pathway is most immediately consequential?
- Teaching interpretation: Impaired descending drive to phrenic circuitry.
- Respiratory risk can coexist with other cord and vertebral injuries.

Clinical clue

Likely lesion

Mechanism

Important limit

Case 3: Long-standing cord lesion at autopsy

- Scenario: Central cystic tissue loss and gliosis at an old cord impact site.
- Above the injury: Degeneration of ascending white-matter pathways.
- Below the injury: Degeneration of descending white-matter pathways.
- The pattern supports both focal trauma and remote Wallerian degeneration.

Clinical clue

Likely lesion

Mechanism

Important limit

Putting pathology and localization together

- Site of impact predicts focal hemorrhagic–necrotic destruction.
- Interrupted tract orientation predicts which remote fibers degenerate.
- Neurological level and spared pathways predict the distribution of deficits.
- Time since injury predicts whether acute changes or chronic cavitation/gliosis dominate.

Mechanical insult

Local tissue loss

Tract disconnection

Functional deficit

Core pathology synthesis

- Acute spinal trauma: Hemorrhage + necrosis + swollen axons.
- Topographic pattern: A maximal impact lesion tapering rostrally and caudally.
- Late lesion: Central tissue loss with cystic transformation and gliosis.
- Long-tract consequence: Ascending degeneration above and descending degeneration below.

**Acute
Hemorrhage / necrosis**



**Evolving
Debris clearance**



**Chronic
Cavity + gliosis**

Discussion questions

- Why can a tiny cervical cord lesion cause more disability than a larger injury elsewhere in the CNS?
- Why does ascending Wallerian degeneration appear above the traumatic lesion?
- Which chronic changes distinguish tissue loss from functional disconnection?
- Why does neurological completeness require sacral examination?

Mechanical insult

Local tissue loss

Tract disconnection

Functional deficit

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