SPINAL SHOCK

By

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- I would like to express my sincere condolences to all the Turkish population especially my Friends, Colleagues for the Mine disaster , May Allah will support the hearts of their families and the souls of the departed rest in peace and to the injured we wish them a quick recovery

Overview

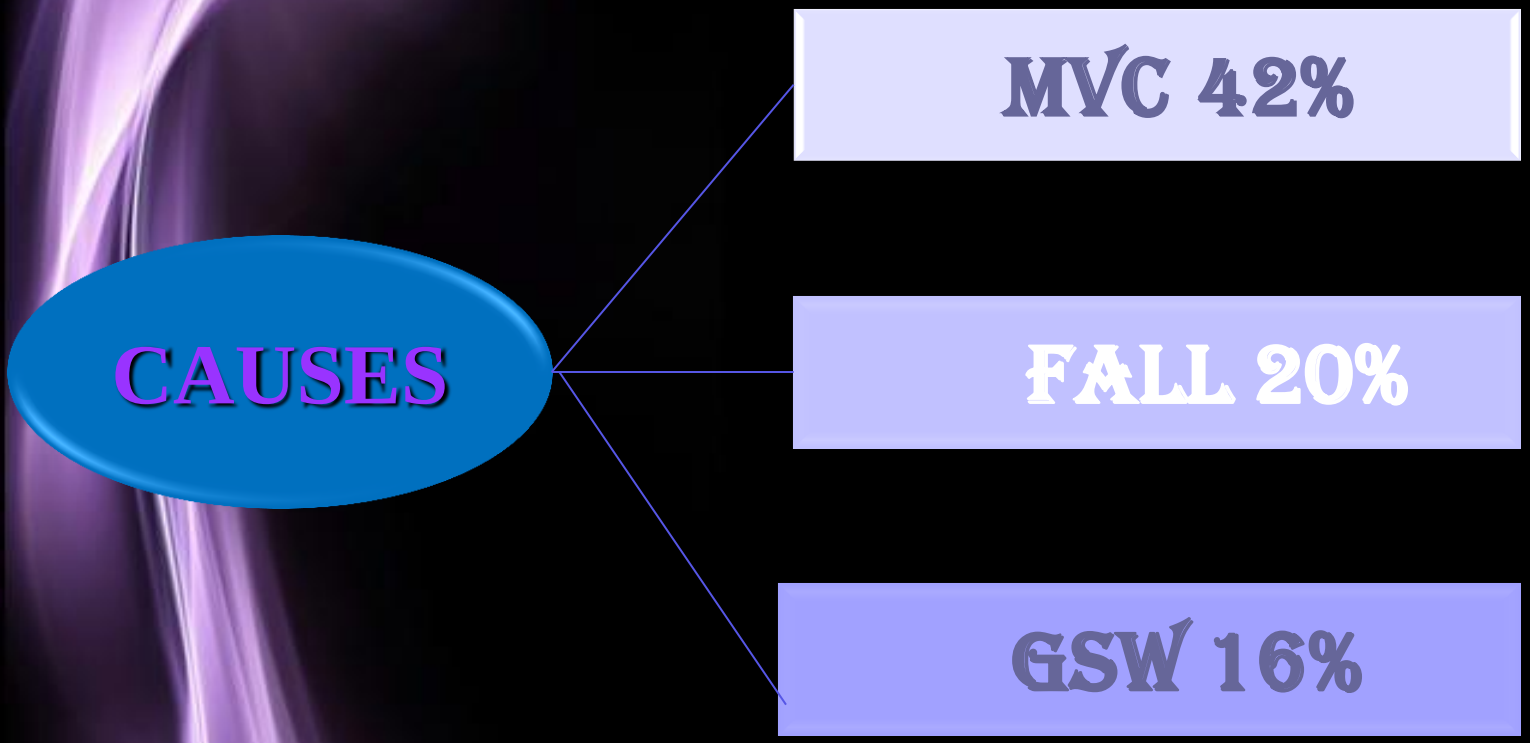
- Epidemiology
- Pathophysiology
- Classification of SCI
- Difference b/w neurogenic & spinal shock
- Treatment strategies

Spinal Cord Injury (SCI)

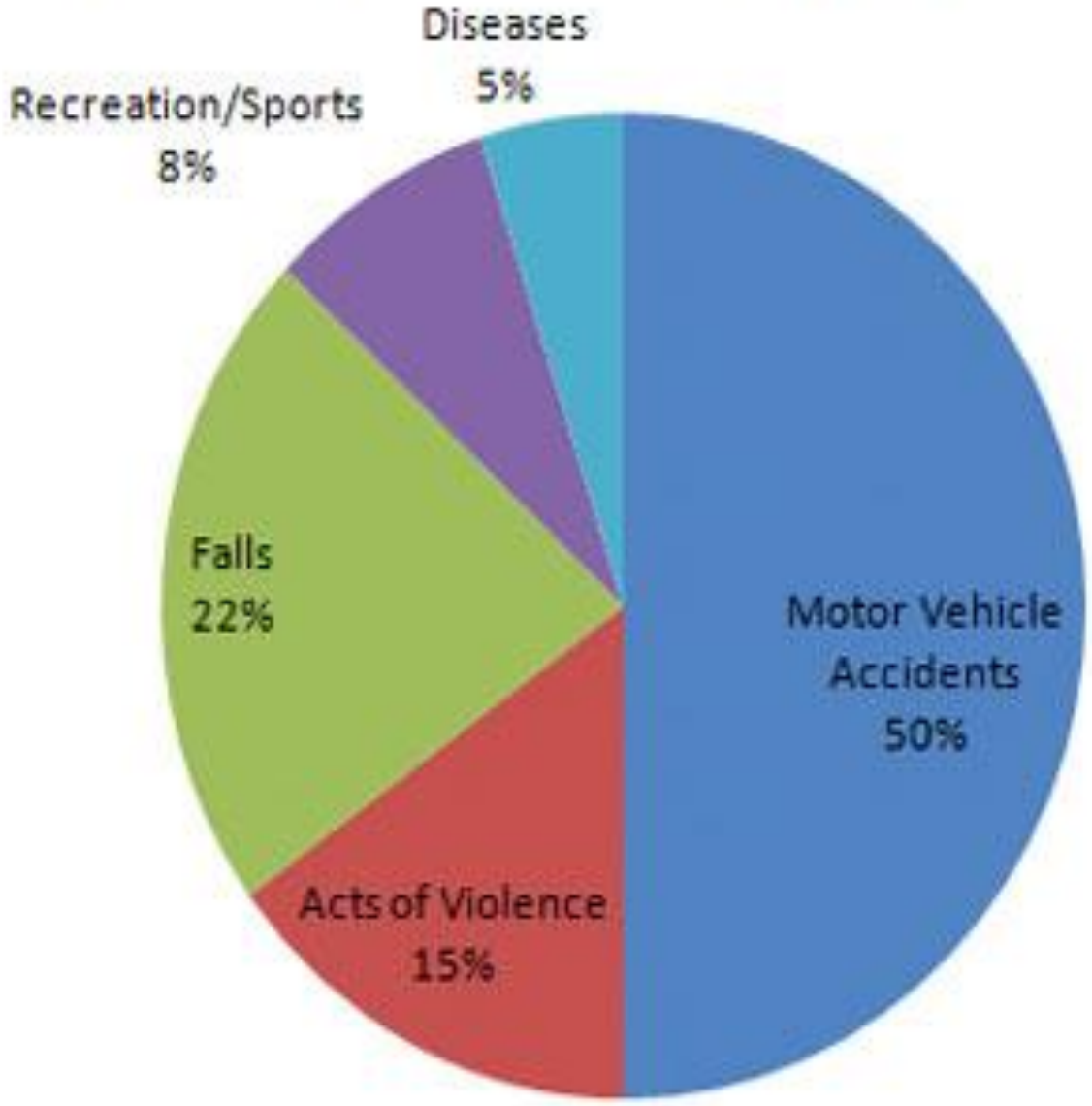
Epidemiology

- **Incidence: 10,000 – 12,000 / Year.**
- **80-85% males (usually 16-30 y/o), 15-20% female.**
- **50% of SCI's are complete.**
- **50-60% of SCI's are cervical.**
- **Immediate mortality for complete cervical SCI ~ 50%.**

Spinal Cord Injury (SCI)



Causes of Spinal Cord Injury in the U.S.



Pathophysiology

Primary

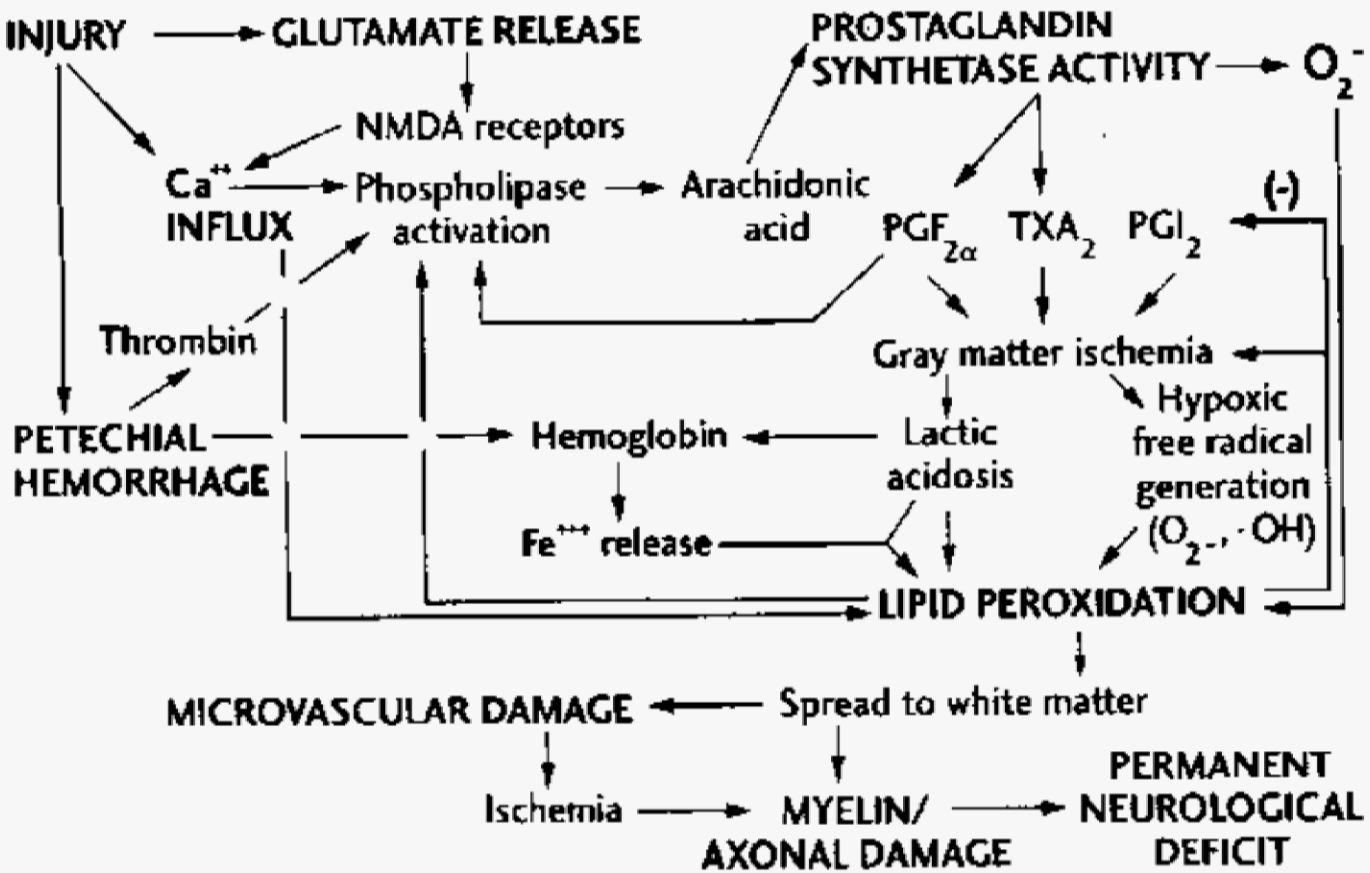
- Result of initial trauma.
- Injury usually permanent.

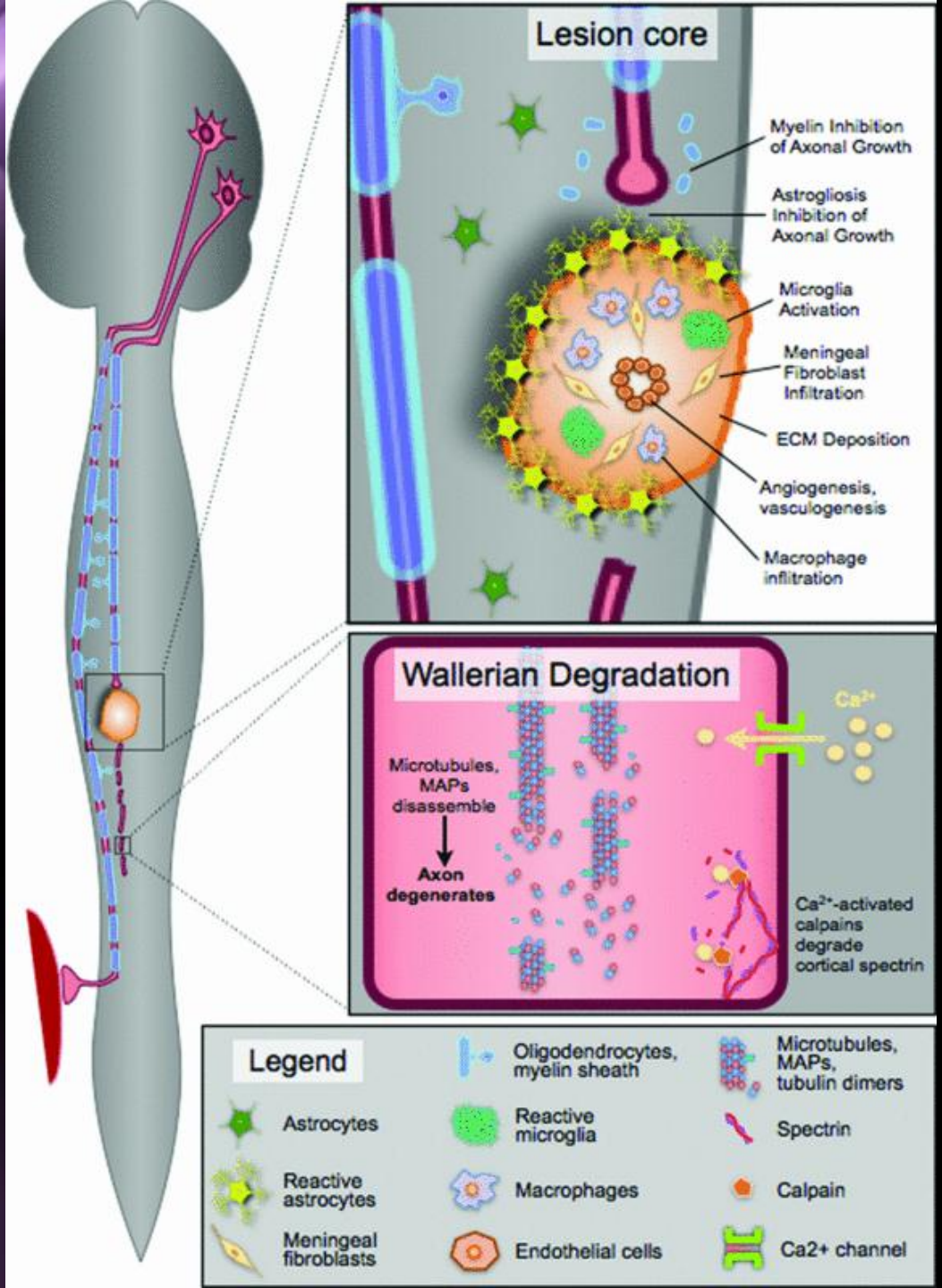
Secondary

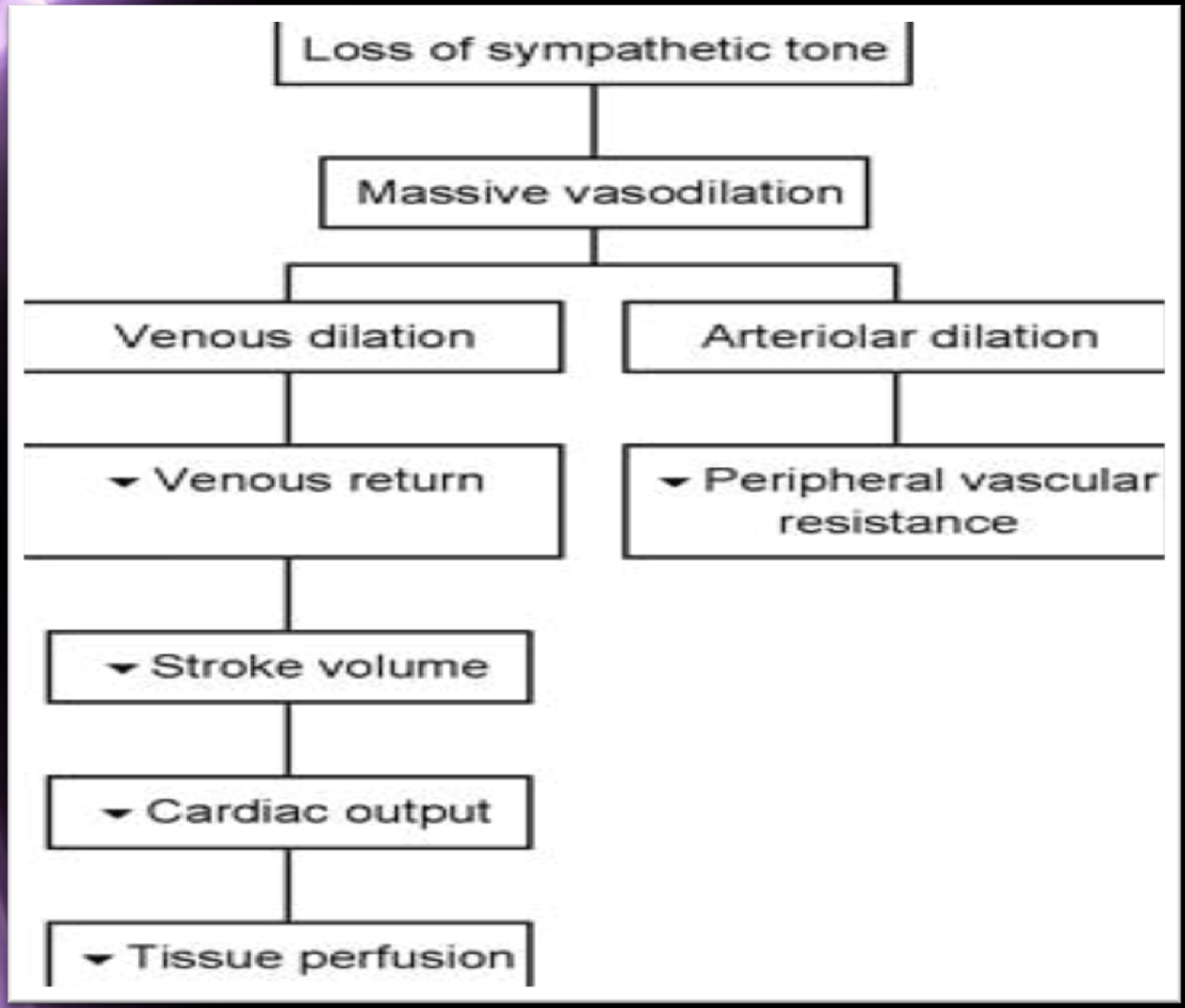
- Occurs after Spinal cord trauma
- Damage at cellular level
- Necrosis (Cells swell, burst and leak toxic substances to other cells)
- Apoptosis

Secondary Injury Cascade

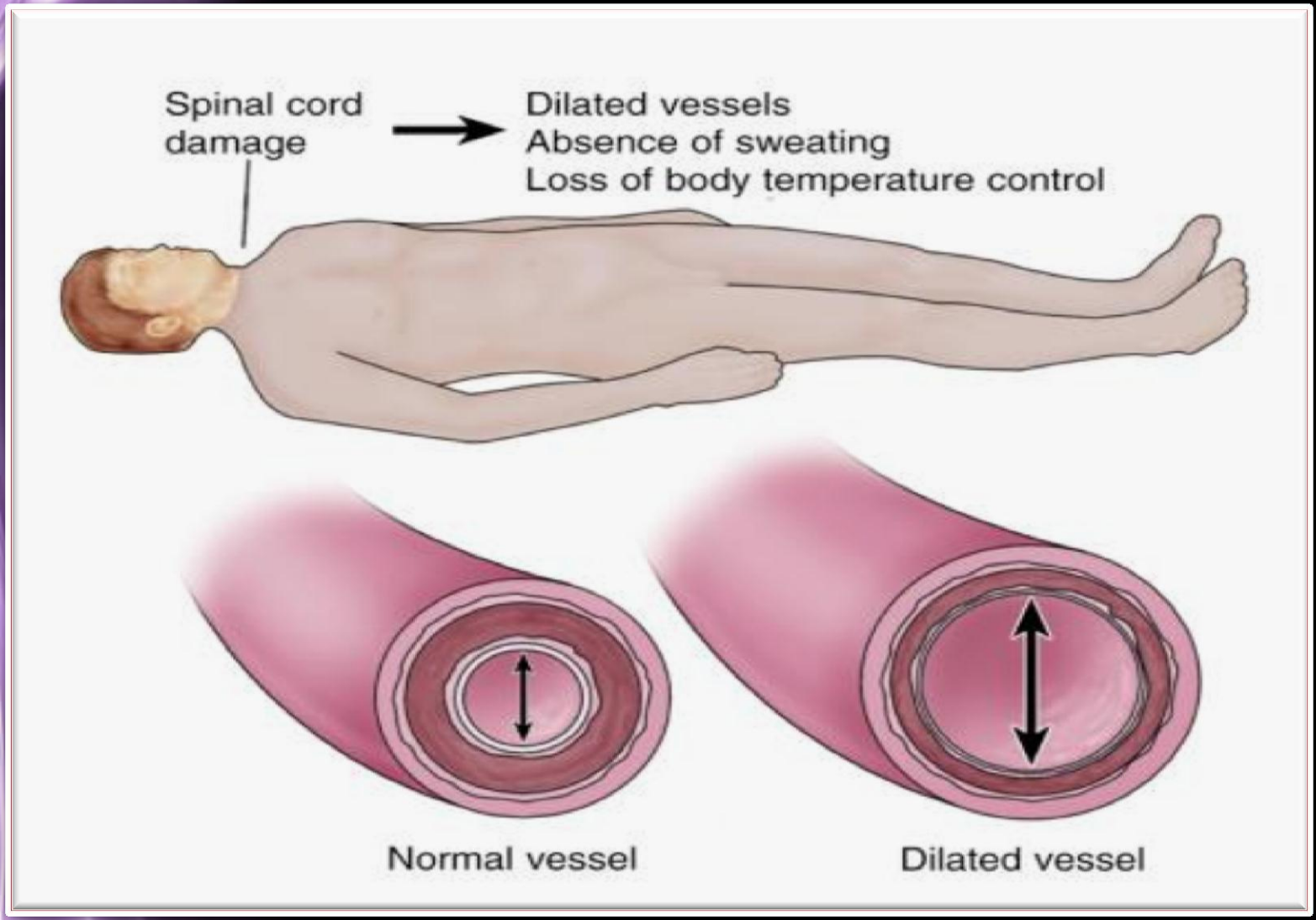
current understanding




Lesion core
Myelin Inhibition of Axonal Growth
Astrogliosis Inhibition of Axonal Growth
Microglia Activation
Meningeal Fibroblast Infiltration
ECM Deposition
Angiogenesis, vasculogenesis
Macrophage infiltration
Wallerian Degradation
Microtubules, MAPs disassemble
Axon degenerates
Ca2+
Ca2+-activated calpains degrade cortical spectrin
Legend
Astrocytes
Reactive astrocytes
Meningeal fibroblasts
Oligodendrocytes, myelin sheath
Reactive microglia
Macrophages
Endothelial cells
Microtubules, MAPs, tubulin dimers
Spectrin
Calpain
Ca2+ channel


Loss of sympathetic tone
Massive vasodilation
Venous dilation
Arteriolar dilation
Venous return
Peripheral vascular resistance
Stroke volume
Cardiac output
Tissue perfusion

Perfusion & Neurogenic Shock



Classifications of SCI

According to

- **Level.**
- **Severity of Neurologic Deficit.**
- **Spinal Cord Syndromes.**
- **Morphology.**

Classifications of SCI

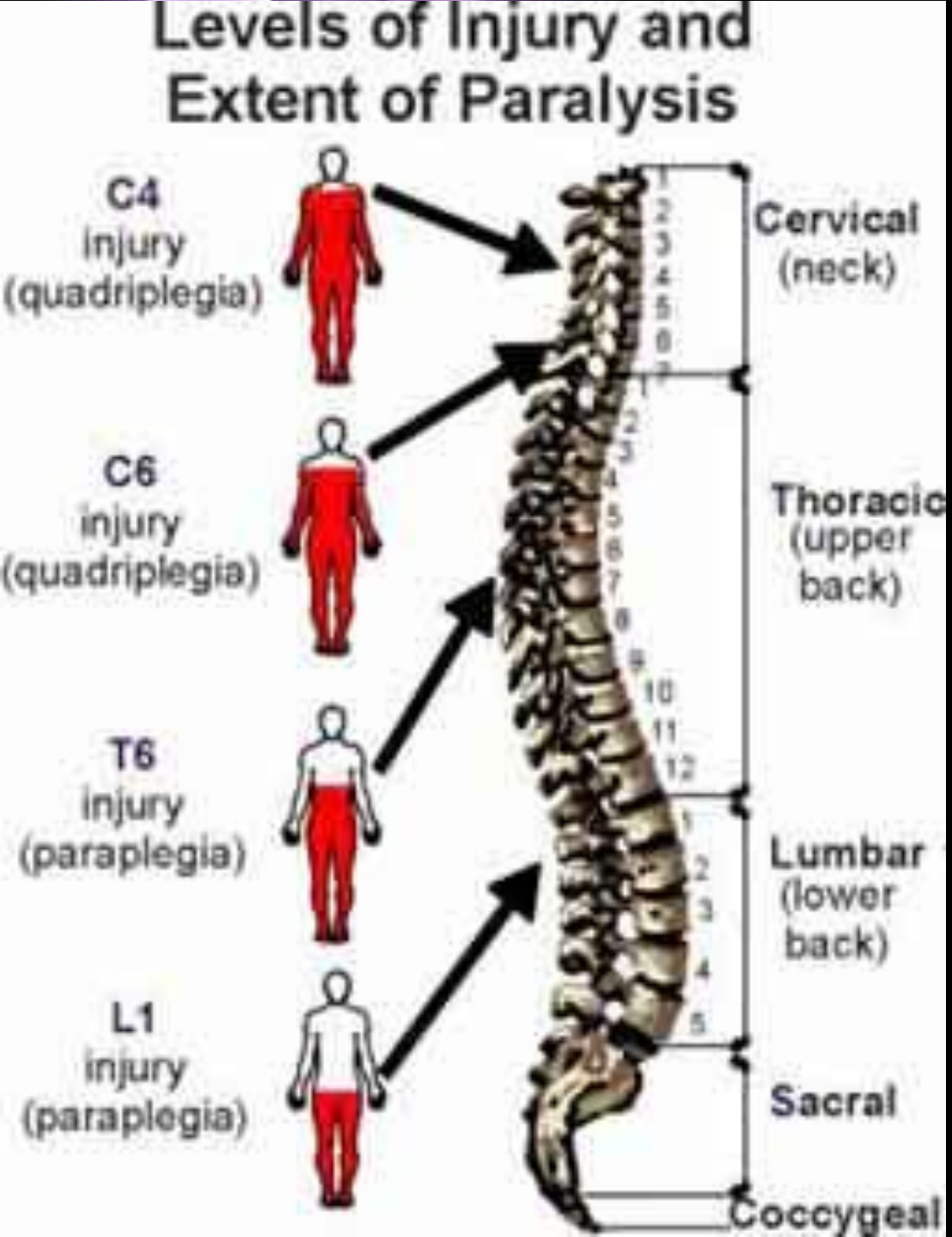
Complete

absence of sensory & motor function in lowest sacral segment after resolution of spinal shock

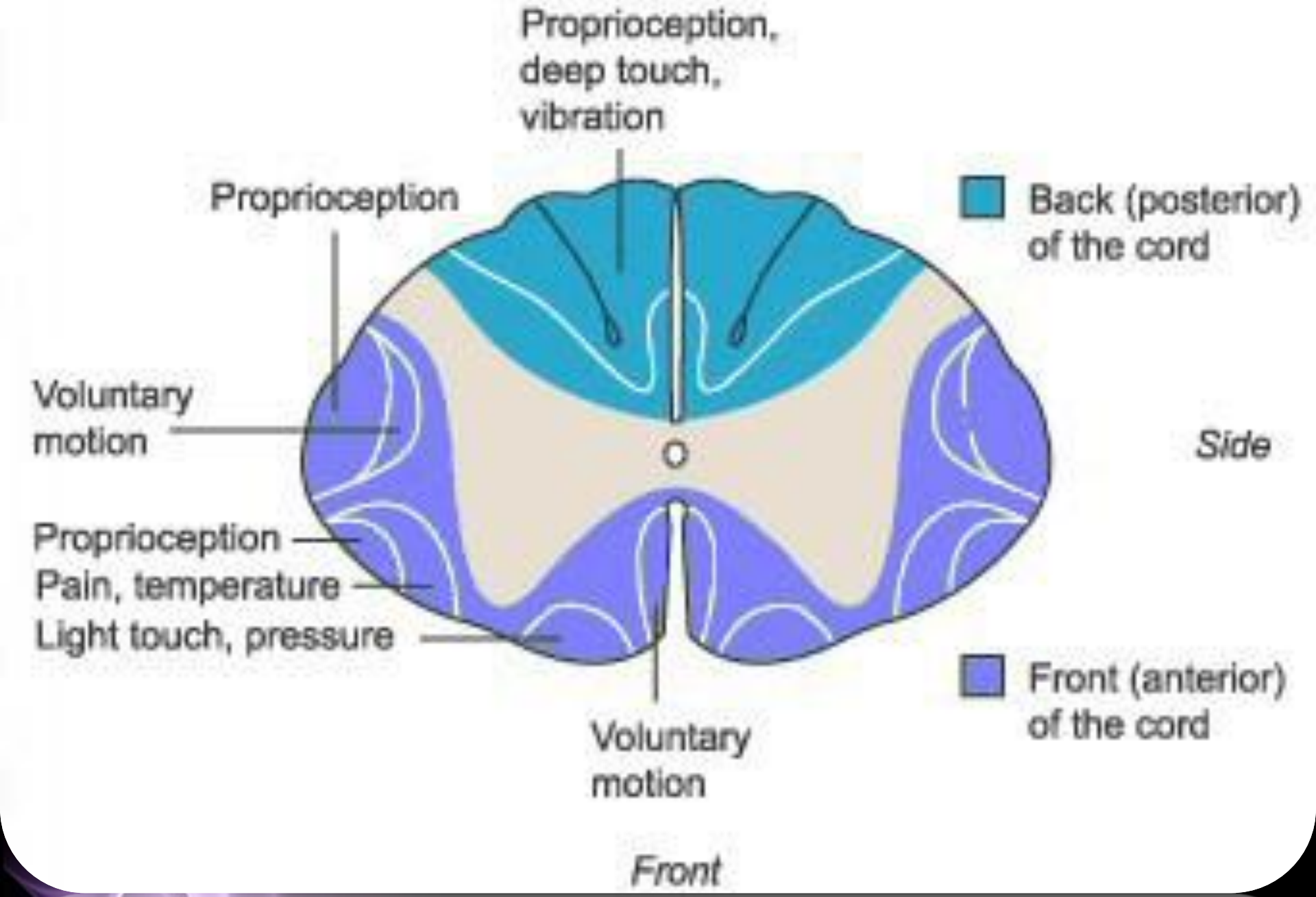
Incomplete

presence of sensory & motor function in lowest sacral segment (indicates preserved function below the defined neurological level)

Levels of Injury and Extent of Paralysis



Spinal Cord - Horizontal View



Incomplete / Partial SCI

Posterior

Central

Anterior

Lateral

Brown-Sequard Syndrome

Spinal shock

Definition

- **Spinal shock** was first defined by Whytt in 1750 as a loss of sensation accompanied by motor paralysis with initial loss but gradual recovery of reflexes, following a spinal cord injury (SCI)– most often a complete transection.

SPINAL SHOCK

- Transient or temporary physiologic rather than anatomical complete loss of all neurological function .
- It is not really shock.
- Of unknown mechanism
- The etiology &significance remain controversial.

SPINAL SHOCK

- SC usually traumatic in origin
- Present in 50% of Pt. w SCI.
- Started within few minutes usually recovers within 24 hours , but may last longer.
- If it is defined by the initial recovery of any reflex, then it is probably last no longer then 20 minutes~ 1 hour

SPINAL SHOCK

- May be as a result of the migration of potassium ions from the intracellular to extracellular spaces
- Neurons become hyperpolarized & unresponsive to stimuli.
- Important no one can evaluate neuralgic deficit until SC end.

Phases of spinal shock

Phase 1	0-1 days – A-reflexia / Hyporeflexia, loss of descending facilitation.
Phase 2	1-3 days – Initial reflex return, Denervation super sensitivity.
Phase 3	1-4 weeks – Hyper-reflexia (initial), Axon–supported synapse growth.
Phase 4	1-12 months – Hyper-reflexia and spasticity. Soma-supported synapse growth.

Bulbo-cavernosus reflex S_{1-3}

- Anal sphincter tone when squeezing glans Penis& Foley's catheter tagging
- It is absent in SC&in lesion below level of T12-L4
- Indicates when present end of SC
- Further improvement for complete injury will be minimal
- If level of reflex arc is physiologically and anatomically intact then it will function even if the cord above is severed.

Neurogenic shock

Definition

- It is a distributive type of shock resulting in hypotension, occasionally with bradycardia, that is attributed to the disruption of autonomic pathway with the spinal cord.

- Usually with SCI above T6.

Neurogenic shock

- 2/3 of the pt with cervicle injury and SBP < 90 have neurogenic shock.
- It is potentially fetal.
- It is diagnosed by excluding.

	SPINAL SHOCK	NEUROGENIC SHOCK
	Due to acute SCI (above T1)	Hemodynamic phenomenon (AboveT6)
Mechanism	Neurons become temporarily unresponsive to brain stimuli	Disruption of autonomic pathway (decrease SVR)
Time	~48-72 hours immediately after SCI	~48-72hours immediately after SCI
BP	Hypotension	Hypotension
Pulse	Bradycardia	Bradycardia
Reflexes/BCR	Absent	Variable
Motor	Flaccid Paralysis	Variable

Autonomic Dysreflexia

- Autonomic dysreflexia is permanent.
- Occurs from Phase 4 onward.
- SCI usually above T5.
- Usually caused by obstructed urinary catheter or fecal impaction. It is characterized by unchecked sympathetic stimulation below the sci (from a loss of cranial regulation).
- Occurs anywhere from 6mths to 2 yrs after the injury.

Physical signs

- **HTN –**

- Bradycardia - 2º to HTN acting on the carotid sinus
- Ventricular arrythmias

- Profuse sweating and flushing (vasodilation) above the lesion

Clinical signs

Severe headache, dyspnea, nausea, shivering, blurred vision, loss of bladder/bowel control and sweating

Management strategies

Current & Future

Reduce the effects of damage

A- Maintain circulation
B- Maintain oxygenation
C- Reduce neurotoxins and free radicals (Meth)
D- Reduce inflammation (IL 10,Cytok,TNF blocker)
E- Reduce apoptosis (NO inhibitors)
F- Cooling (induction of hypothermia)

Management strategies

Current & Future

Encourage correct neuron function and connection

- Cells (Schwann cells, macrophages, others).
- Matrix (Netrins,Neural glues)
- Nerve graft (PN Implants)

Enhance regeneration and axon growth

Replace lost nerve cell

Inhibit scar and gliosis formation


RFDS

Airway consideration

In obtunded / unconscious patients

Surgical airway less preferred, unless necessary.

pressure) RSI use bougie or styled

to maximize first attempt success.

Airway consideration

- RD can occur after SCI either due to thoracoabdominal injury or SCI.
- With severe thoracic or cervical SCI, chest can move paradoxically.
- Neurogenic pulmonary edema can occur.

Breathing considerations

Airway consideration

Breathing considerations

- Maintain spinal cord perfusion
- Hypotenstension must be prevented and treated
- hypotension is very deleterious (cord hypoperfusion,and increasing secondary injury)..

Circulation Consideration

and CHF.

Circulation Consideration

- IF SVR decreased, but CO&HR are adequate ,then Norepinephrine, or Phenylephrine can be used.

- If SVR decreased with impaired CO&HR,then

Inotrpic agent(dopamine) may be more useful.

Circulation Considerati

- atropin (0.4-0.6mg/4h) is used frist line for symptomatic bradycardia (should be kept ready).

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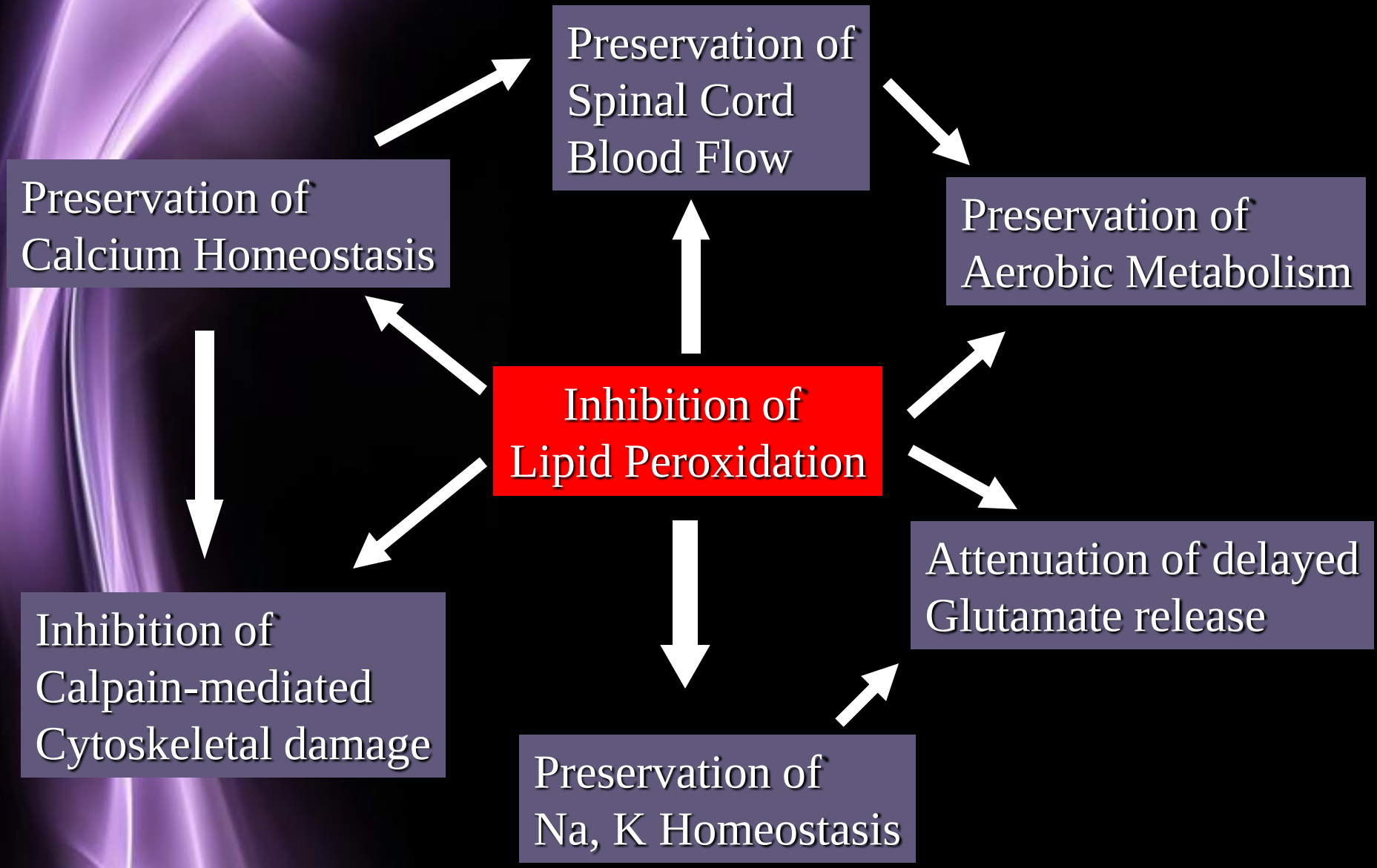
- Dopamine (2-10 mag/kg/min) or epinephrine may be helpful.

- Methylaxanthine (theo,aminophyline) have been used effectively for refractory symptomtic bradycardia

Circulation Considerati

- Psudoephadrine is an effective adjunctive therapy (facilitate the discontinuation of vasopressors and or atropin .
- pacing if necessary.

Neuroprotection w/ MPSS



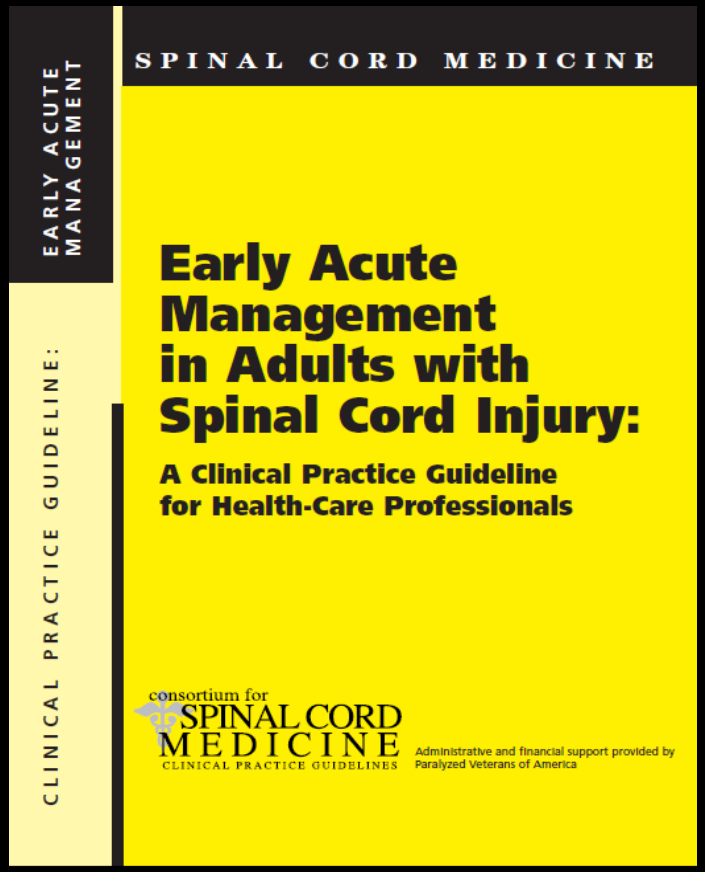
Methylprednisolone Therapy

- **The most recent guidelines published by the Consortium for Spinal** Cord Medicine Spinal Cord Society that "no clinical evidence exists to definitively recommending steroids in the treatment of acute SCI to improve functional recovery
- It is use can be considered only, at best ,a treatment option rather than treatment standard
- Case –by –case risk- benefit assessment should be performed
- Recommended by NASCIS III in non penetrating SCI within 8 Hrs of injury

Pharmacologic Neuroprotection in Patients with SCI

No clinical evidence exists to definitively recommend

the use of any neuroprotective pharmacologic agent, including steroids, in the treatment of acute SCI to improve functional recovery.

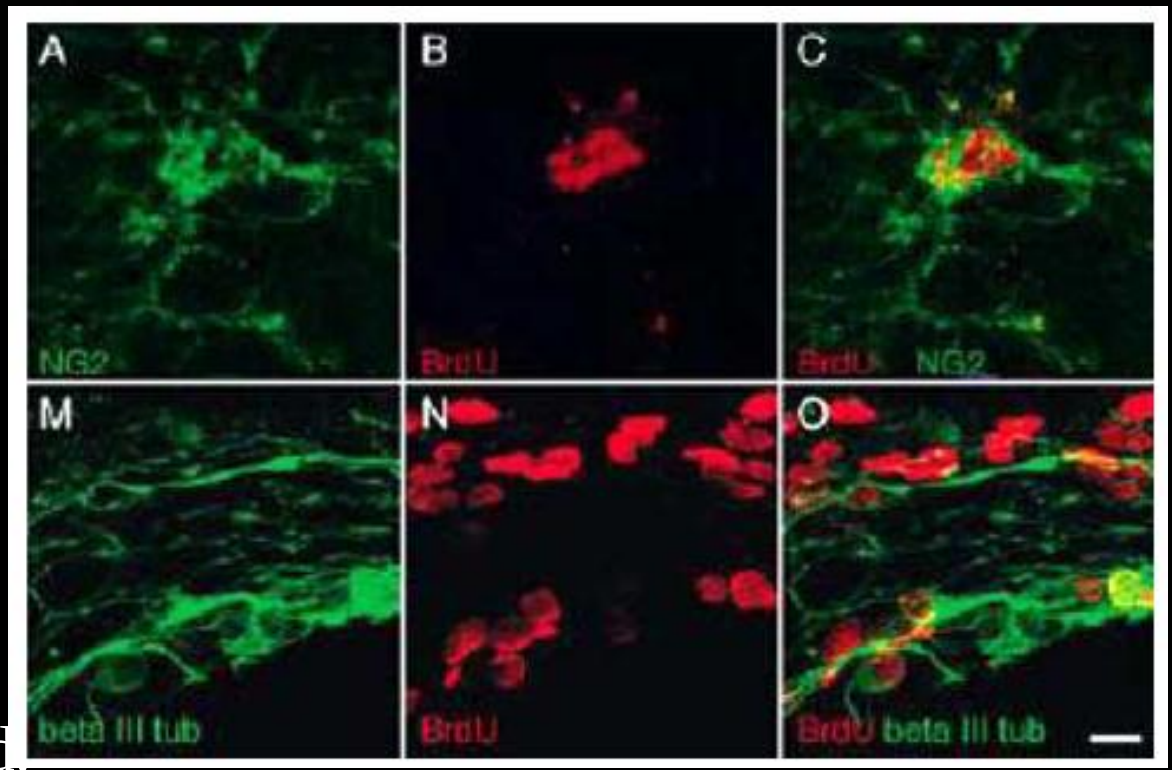


Stem Cell Therapy

- Ongoing studies of adult mesenchymal SCT

The overall future for SCT look promising

More researche on SCT for SCI is needed.



Timing of surgery

- Variable opinions, poor evidence.

The role of immediate surgical intervention in the management of spinal injuries is limited .

Summary

- Spinal cord injury manily caused by MVA
- There is no way to reverse sc damage
- Spinal shock & neurogenic shock can in same pt but not same disorder.
- SCI goals of care is prevention of further inury.

Questions

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THANK YOU