RANCHO LOS AMIGOS
NATIONAL REHABILITATION CENTER



Spinal Shock & Acute Spinal Cord Injury

3/11/2023

Dr. Jeremy Lou, MD

Financial Disclosures

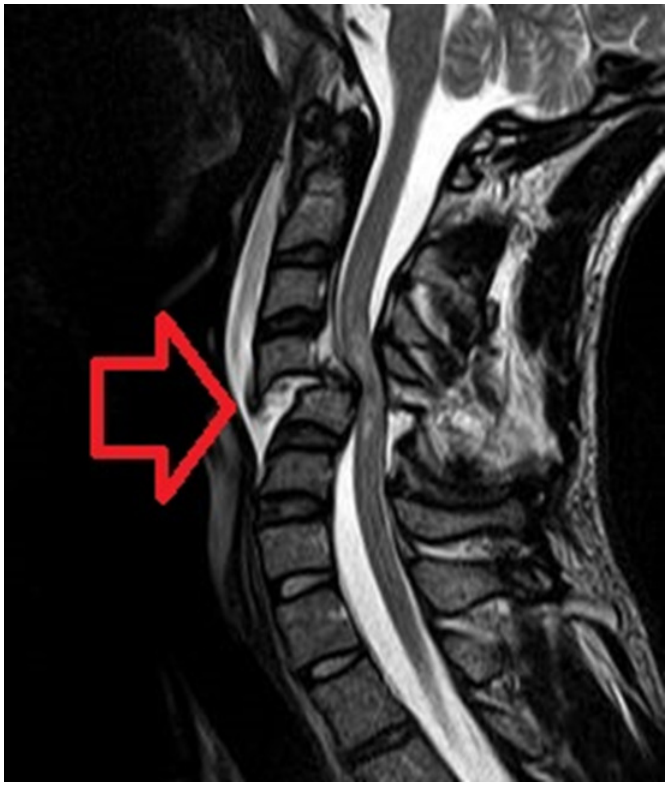
- None

Objectives

- Introduction to spinal cord injury (SCI) & epidemiology
- Define spinal shock
- Describe the etiology & pathophysiology of spinal shock
- Distinguish spinal shock from neurogenic shock
- Describe the clinical evaluation of acute traumatic SCI
  - ISNCSCI (ASIA) exam brief overview
- Medical management of spinal shock following acute SCI
- Recovery from spinal shock & ongoing debate

Introduction

- SCI is a common injury in the U.S.
- Incidence of 54 million persons / year
- Approximately 280,000 living survivors of traumatic SCI as of 2017 in U.S.
- Nontraumatic SCI prevalence is more unclear, but is likely even more common



Acute SCI Pathophysiology: A 2-Step Process

Primary Injury

- Immediate effect of traumatic forces
  - Compression
  - Contusion
  - Shearing
  - Penetration
  - Kinetic (i.e. energy from bullet)

Secondary Injury

- Occurs minutes to hours after primary injury
  - Ischemia
  - Hypoxia
  - Inflammation/edema
  - Excitotoxicity, free radicals
  - Disturbance of ion homeostasis
  - Apoptosis

What is “Spinal Shock?”

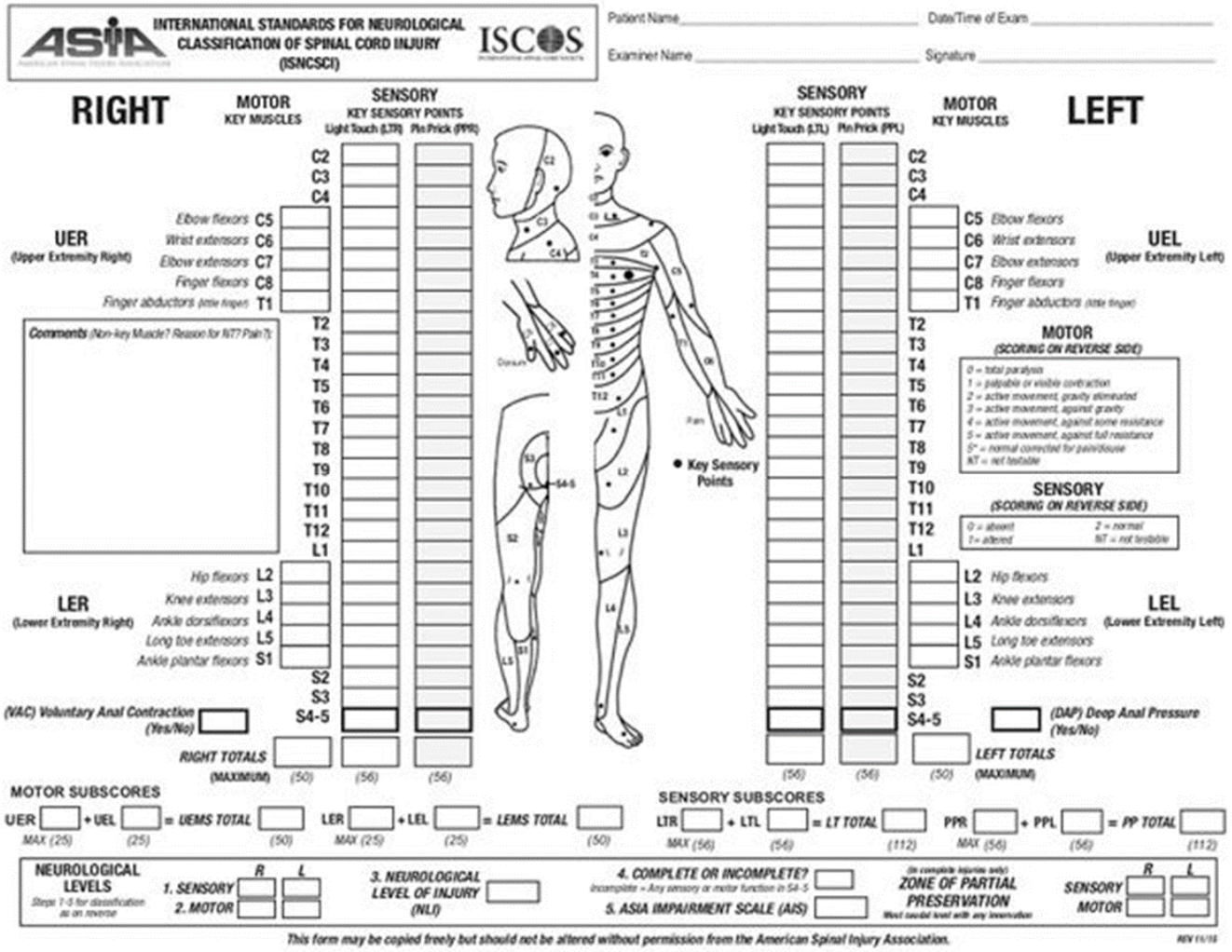
- Sudden, transient loss of spinal cord function below the neurologic level of injury after an acute SCI
- Flaccid paralysis, anesthesia, absent bowel/bladder control, areflexia
  - Definition is controversial
  - No overarching consensus over when spinal shock actually “ends”
- *Must be distinguished from neurogenic shock*

	Spinal Shock	Neurogenic Shock
Definition	Immediate transient loss of cord function (motor, sensory) and reflexes below injury level	Sudden loss of sympathetic nervous system signal pathway
Mechanism	Peripheral neurons unresponsive to brain stimuli 2/2 cord injury	Autonomic pathway disruption -> loss of sympathetic tone & vasodilation, hemodynamic instability due to sympathetic-parasympathetic imbalance
Motor	Flaccid paralysis	Variable
BP	Variable (typically hypotension)	Hypotension
Pulse	Variable (typically bradycardia)	Bradycardia
Bulbocavernosus reflex	Absent	Variable

Acute Evaluation

- Primary assessment
  - Airway, breathing, circulation, disability (neuro)
  - Immobilize spine (C-collar, backboard w/supportive blocks)
  - Ventilation support (mechanical vent vs intubation)
  - Fluids*
  - Urinary catheter placement
  - Neurologic exam

Acute Evaluation: ISNCSCI (ASIA) Exam



INTERNATIONAL STANDARDS FOR NEUROLOGICAL CLASSIFICATION OF SPINAL CORD INJURY (ISNCSCI)

Patient Name _____ Date/Time of Exam _____

Examiner Name _____ Signature _____

RIGHT | **LEFT**

MOTOR KEY MUSCLES | SENSORY KEY SENSORY POINTS — Light Touch (LT) / Pin Prick (PP)

UER (Upper Extremity Right): Elbow flexors C5; Wrist extensors C6; Elbow extensors C7; Finger flexors C8; Finger abductors (little finger) T1

UEL (Upper Extremity Left): C5 Elbow flexors; C6 Wrist extensors; C7 Elbow extensors; C8 Finger flexors; T1 Finger abductors (little finger)

Comments (Non-key Muscle? Reason for NT? Pain?):

LER (Lower Extremity Right): Hip flexors L2; Knee extensors L3; Ankle dorsiflexors L4; Long toe extensors L5; Ankle plantar flexors S1

LEL (Lower Extremity Left): L2 Hip flexors; L3 Knee extensors; L4 Ankle dorsiflexors; L5 Long toe extensors; S1 Ankle plantar flexors

C2 C3 C4 T2 T3 T4 T5 T6 T7 T8 T9 T10 T11 T12 L1 S2 S3 S4-5

● Key Sensory Points

MOTOR (SCORING ON REVERSE SIDE)

0 = total paralysis
1 = palpable or visible contraction
2 = active movement, gravity eliminated
3 = active movement, against gravity
4 = active movement, against some resistance
5 = active movement, against full resistance
5* = normal corrected for pain/disuse
NT = not testable

SENSORY (SCORING ON REVERSE SIDE)

0 = absent; 1 = altered; 2 = normal; NT = not testable

(VAC) Voluntary Anal Contraction (Yes/No)

(DAP) Deep Anal Pressure (Yes/No)

RIGHT TOTALS (MAXIMUM) (50) (56) (56)

LEFT TOTALS (MAXIMUM) (56) (56) (50)

MOTOR SUBSCORES

UER + UEL = UEMS TOTAL (MAX 25 + 25 = 50)

LER + LEL = LEMS TOTAL (MAX 25 + 25 = 50)

SENSORY SUBSCORES

LTR + LTL = LT TOTAL (MAX 56 + 56 = 112)

PPR + PPL = PP TOTAL (MAX 56 + 56 = 112)

NEUROLOGICAL LEVELS (Steps 1-5 for classification on reverse)

1. SENSORY R L
2. MOTOR R L

3. NEUROLOGICAL LEVEL OF INJURY (NLI)

4. COMPLETE OR INCOMPLETE? (Incomplete = Any sensory or motor function in S4-5)

5. ASIA IMPAIRMENT SCALE (AIS)

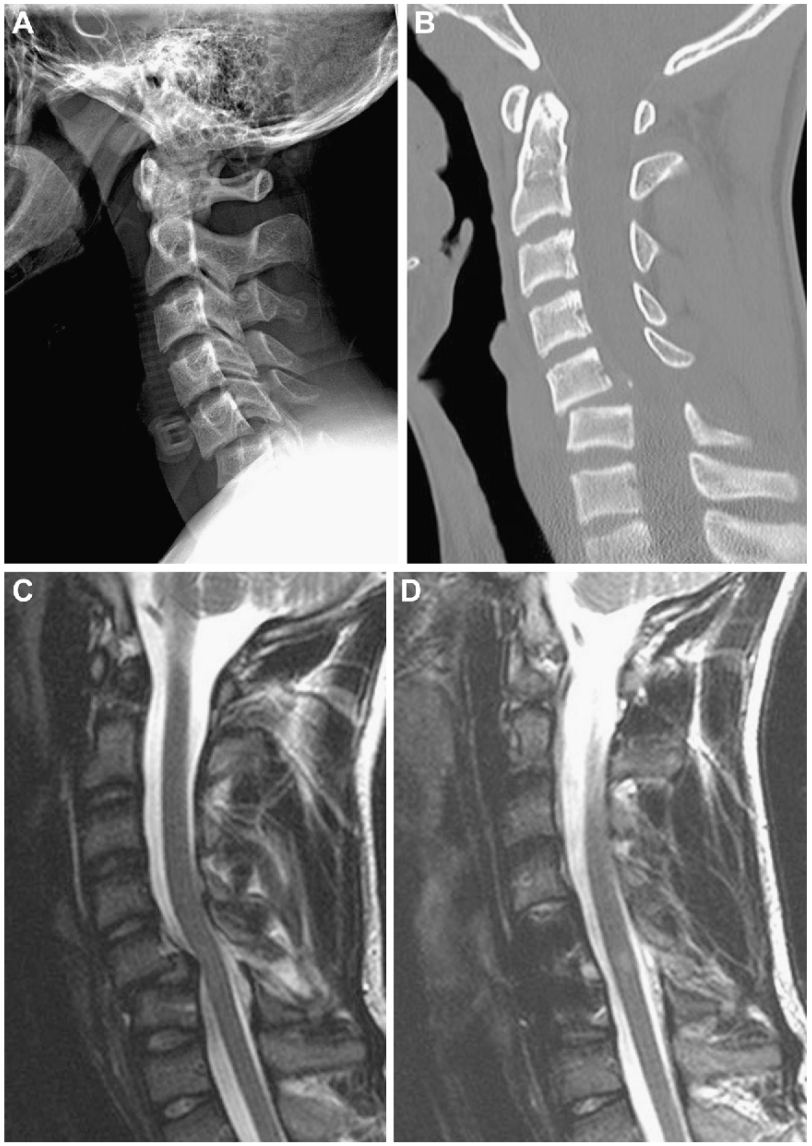
ZONE OF PARTIAL PRESERVATION (for complete injuries only; Most caudal level with any innervation) — SENSORY R L; MOTOR R L

REV 11/15

ASIA Impairment Scale		
A	Complete	No motor, no sensory, no sacral sparing
B	Incomplete	No motor, sensory only
C	Incomplete	50% of muscles LESS than grade 3 (can't raise arms or legs off bed)
D	Incomplete	50% of muscles MORE than grade 3 (can raise arms or legs off bed)
E	Normal	Motor and sensory function are normal

Acute Evaluation: Imaging

Plain radiographs (XRAY)	**Rapid assessment of alignment, fractures, & soft tissue swelling** **- AP, lateral, open-mouth ondontoid view (C spine), swimmer's view** **- Typically not sufficient for initial evaluation** **- Can sometimes miss fractures**
Computed tomography (CT)	- Higher sensitivity than plain films for fracture detection - Can be done without moving patient out of supine - Rule out head injury - Often prioritized over XRAYs - Lower sensitivity for spinal cord pathology than MRI
Magnetic resonance imaging (MRI)	- Superior to CT for soft tissue & intra-cord pathology (i.e. hemorrhage) - Indicated if suspecting SCI with negative CT scan - More technically difficult to perform - Contraindicated for pacemaker, metallic foreign bodies, etc. - No radiation


A
B
C
D

Medical Management of Spinal Shock

- Hypotension, Bradycardia
- Respiratory complications
- Bowel & Bladder Dysfunction
- Venous thromboembolism, PE
- Pressure injury prevention & other prophylaxis

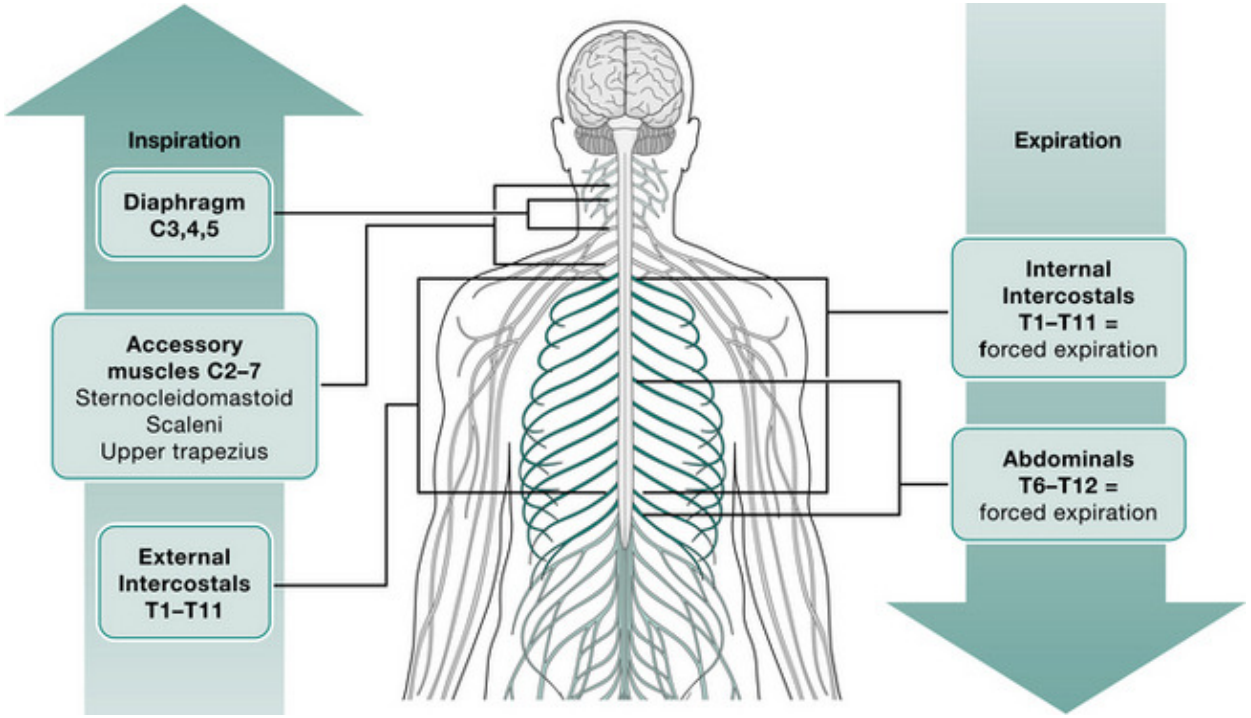
Hypotension, Bradycardia

- Due to autonomic dysregulation & imbalance
- MABP goal: 85mmHg – 90mmHg fo first 7 days after SCI
- Fluids (avoid overload!)
- Inotropic therapies
  - Norepinephrine
  - Dopamine
  - Phenylephrine
  - Midodrine, Desmopressin
- Atropine & pacing for bradycardia (C1-C5)

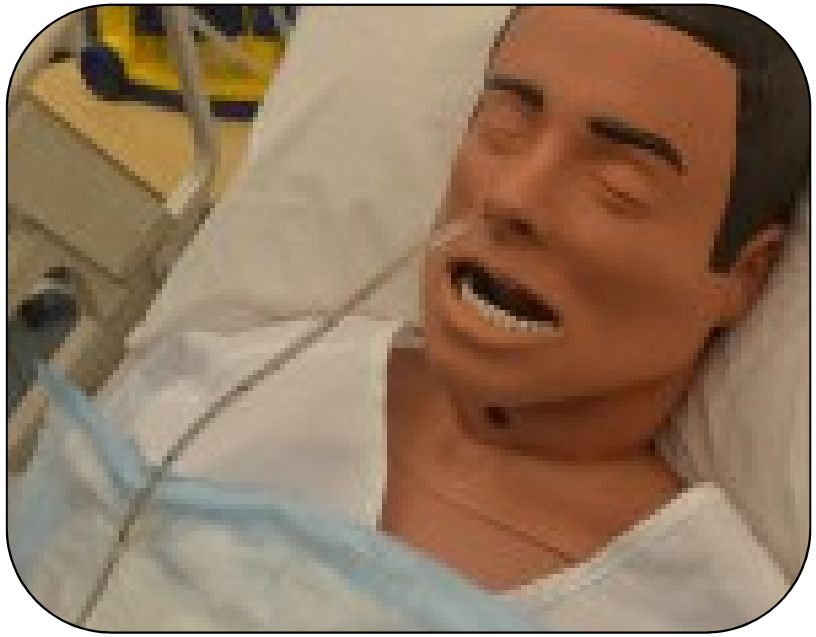
Respiratory Complications

Common pulmonary complications include:

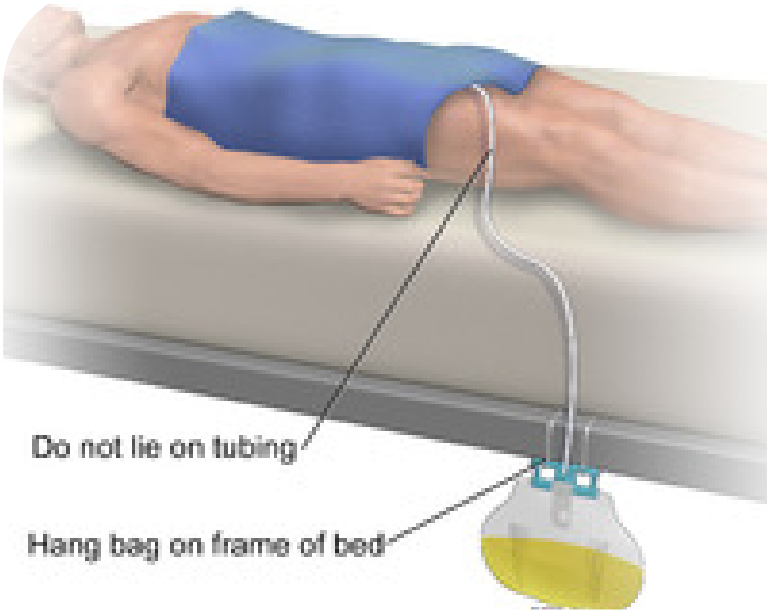
- Respiratory failure
  - Increased RR, declining forced vital capacity, rising pCO2, falling pO2
- Pulmonary edema
- Pneumonia
- Pulmonary embolism
- Tracheostomy
  - Done within 7-10 days unless able to extubate
  - ASIA A SCI more likely to require tracheostomy



Bowel & Bladder Dysfunction



Paralytic ileus: Decompression with NGT. Once resolved, begin enteral nutrition.

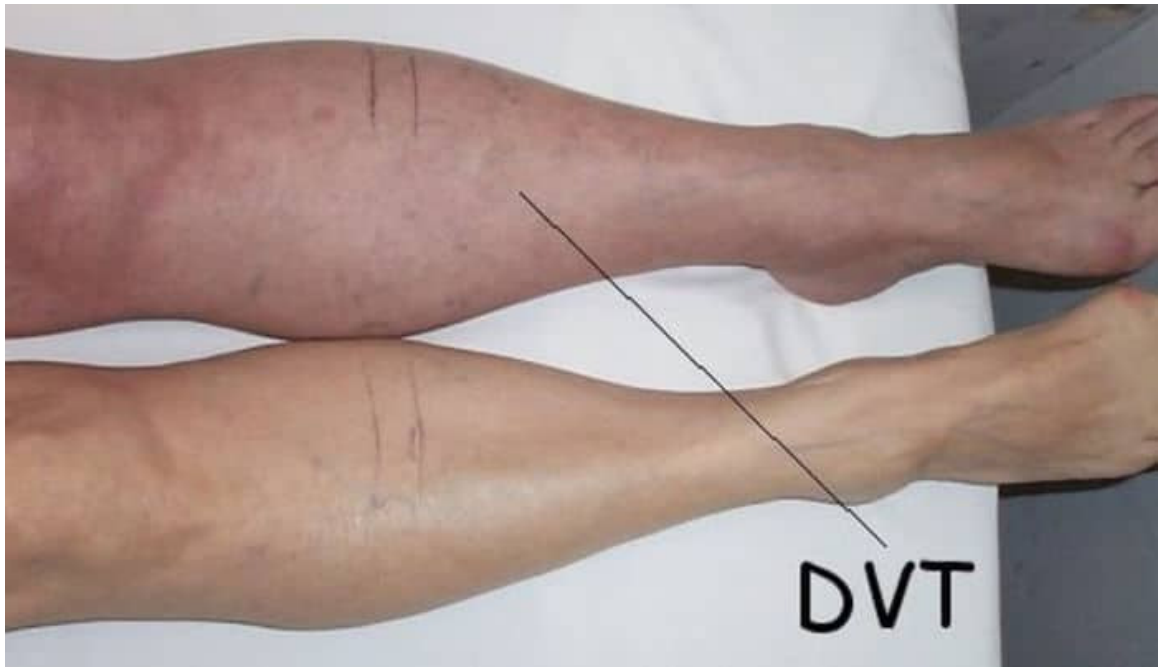


Urinary catheter placement (i.e. foley)

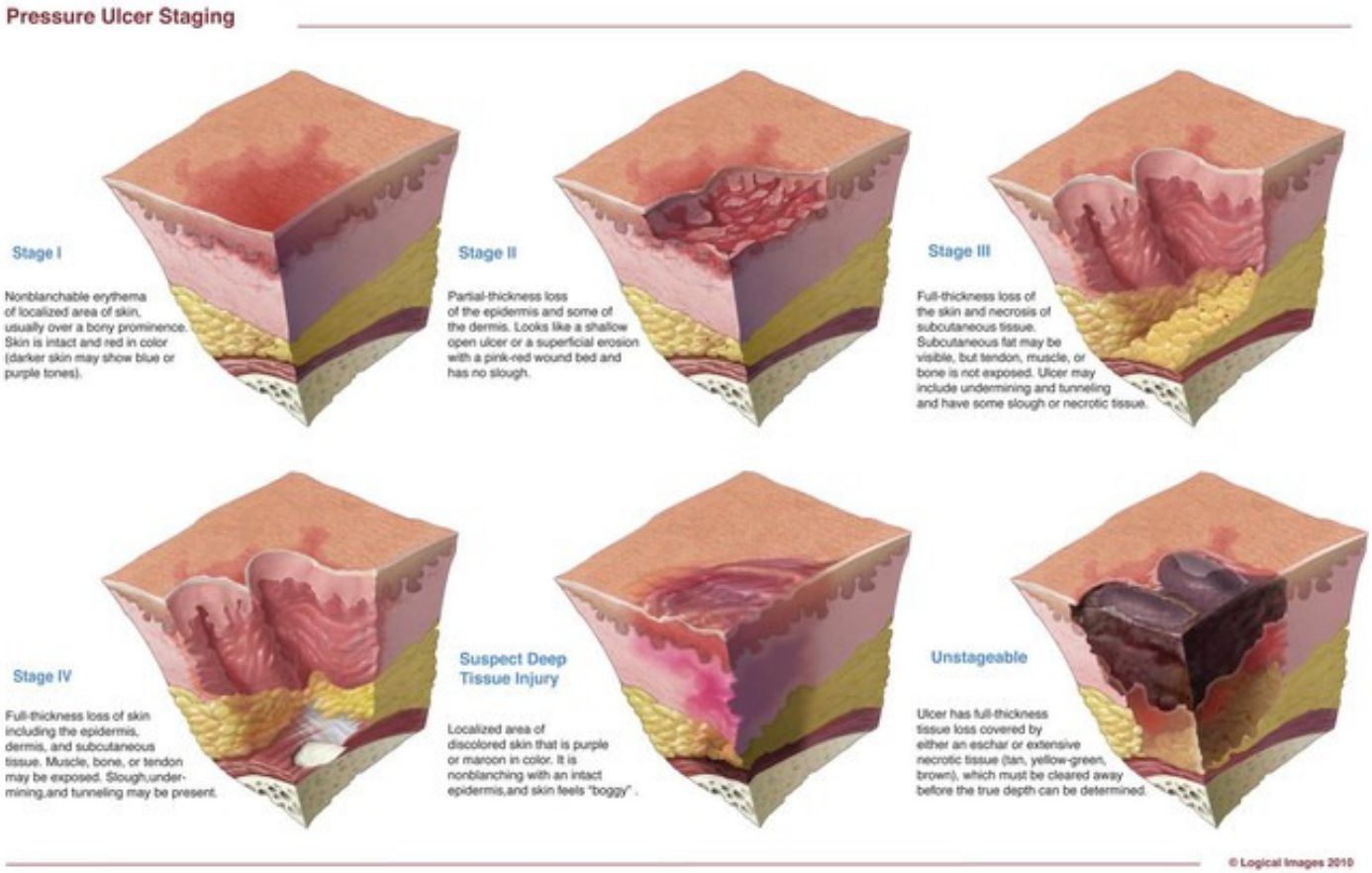
Transition to intermittent catheterization if able (usually after transfer to rehab)

Venous Thromboembolism (VTE), PE

- DVT occurs in 50%-100% of untreated patients after TSCI
- Greatest incidence between 72 hours & 14 days
- Risk is not affected by level or severity of SCI
- All SCI patients should have DVT prophylaxis if not contraindicated



Pressure Injury Prevention

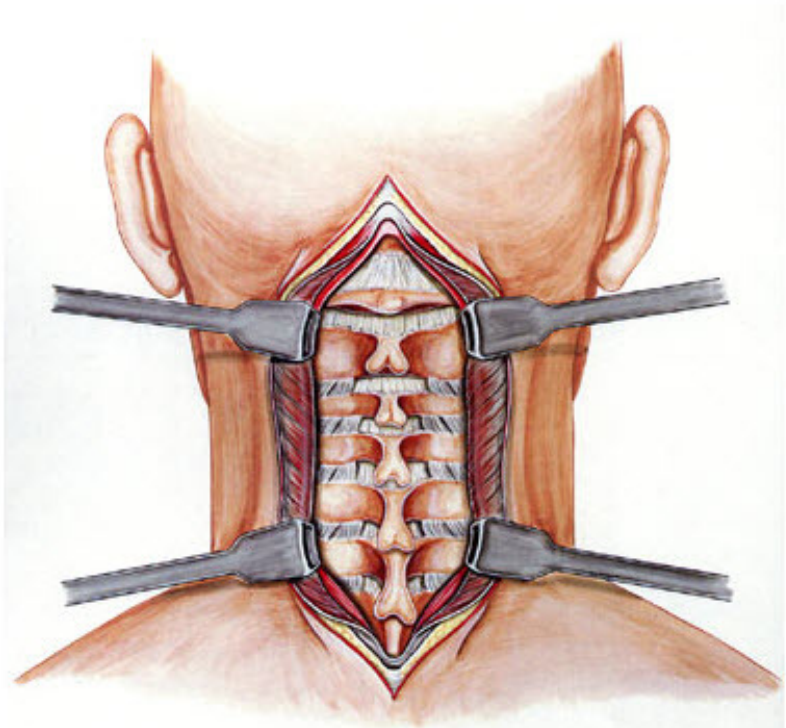


Other Prophylaxis & Management

- Gastric ulcer prophylaxis
  - Proton pump inhibitors x 4 weeks
- Pain Management
  - Multimodal
  - Usually require medications & non-pharmacologic approaches
  - Post-operative use of opiates may be indicated
    - IV pain medications considered NOT compatible with most ARU settings
- Temperature control (esp. in cervical SCI)
  - Poikilothermia
  - Absence of vasomotor control, sweating below lesion

Additional Considerations

- No specific pharmacologic therapy is available to treat SCI or spinal shock.
  - Glucocorticoids are generally NOT recommended in acute SCI
- Surgery is indicated for significant cord compression & deficits, but optimal timing is not clear
- Autonomic dysreflexia
- Orthostatic hypotension
- Nutrition & weight
- Mental health needs



Posterior Cervical Spine Surgery

Therapy Interventions: PT, OT, SLP, Nursing, Psychology

Phases Of Recovery from Spinal Shock

Phases	Features/Characteristics
Phase 1 (0-24 hours)	Areflexia -> Delayed plantar reflex (S1) -> bulbocavernosus reflex (S3-S5) -> abdominal wall -> cremasteric reflex (L2) Sympathetic dysfunction -> bradyarrhythmia, AV block, hypotension
Phase 2 (1 – 3 days)	Cutaneous reflexes emerge more Deep tendon reflexes (DTRs) remain absent
Phase 3 (4 days – 1 month)	Short axon & synaptic growth Deep tendon reflexes usually return Babinksi sign may appear
Phase 4 (1 – 12 months)	Long axon & synaptic growth Reflexes become hyperactive Malignant hypertension & AD may appear

When does spinal shock actually "end?"

- Usually lasts for days or weeks after SCI
- Average duration: 4-12 weeks, or longer
- Definition & pattern of reflex recovery is debatable & controversial
  - Lack of consensus defining cessation
  - Not all reflexes are uniformly depressed in each patient
  - Reflex recovery is individualized
- Resolution over days to months -> slow transition to spasticity & return of UMN signs

Patient Education?

- Currently there is no actual consensus on when spinal shock ends
- Consider spinal shock & recovery on an individualized basis
- Spinal shock may last 4-12 weeks, or longer
- Distinguish from neurogenic shock!

References

- 1. Endrit Ziu; Fassil B. Mesfin. “Spinal Shock”. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. URI: Spinal Shock - StatPearls - NCBI Bookshelf (nih.gov)
- Ko HY. Revisit Spinal Shock: Pattern of Reflex Evolution during Spinal Shock. Korean J Neurotrauma. 2018 Oct;14(2):47-54.
- Chemical characteristics of traumatic spinal cord edema in cats. Effects of steroids on potassium depletion. Lewin MG, Hansebout RR, Pappius HM J Neurosurg. 1974;40(1):65.
- Antevil JL, Sise MJ, Sack DI, Kidder B, Hopper A, Brown CV. Spiral computed tomography for the initial evaluation of spine trauma: A new standard of care? J Trauma. 2006 Aug;61(2):382-7.
- Jia X, Kowalski RG, Sciubba DM, Geocadin RG. Critical care of traumatic spinal cord injury. J Intensive Care Med. 2013 Jan-Feb;28(1):12-23.
- Cheung V, Hoshide R, Bansal V, Kasper E, Chen CC. Methylprednisolone in the management of spinal cord injuries: Lessons from randomized, controlled trials. Surg Neurol Int. 2015;6:142.
- Ellingson, Benjamin M. et al. “Imaging techniques in spinal cord injury.” World neurosurgery 82 6 (2014): 1351-8 .
- Hurlbert RJ, Hadley MN, Walters BC, Aarabi B, Dhall SS, Gelb DE, Rozzelle CJ, Ryken TC, Theodore N. Pharmacological therapy for acute spinal cord injury. Neurosurgery. 2013 Mar;72 Suppl 2:93-105.
- Ko HY, Ditunno JF, Graziani V, Little JW. The pattern of reflex recovery during spinal shock. Spinal Cord. 1999 Jun;37(6):402-9.
- Kirshblum S, Millis S, McKinley W, Tulsky D. Late neurologic recovery after traumatic spinal cord injury. Arch Phys Med Rehabil. 2004 Nov;85(11):1811-7. doi: 10.1016/j.apmr.2004.03.015. PMID: 15520976.
- Ko HY. Revisit Spinal Shock: Pattern of Reflex Evolution during Spinal Shock. Korean J Neurotrauma. 2018 Oct;14(2):47-54. doi: 10.13004/kjnt.2018.14.2.47. Epub 2018 Oct 31. PMID: 30402418; PMCID: PMC6218357.
- Ditunno JF, Little JW, Tessler A, Burns AS. Spinal shock revisited: a four-phase model. Spinal Cord. 2004;42:383–395.


תודה
Dankie
Gracias
Спасибо
شكراً
Merci
Takk
Köszönjük
Terima kasih
Grazie
Dziękujemy
Dėkojame
Ďakujeme
Vielen Dank
Paldies
Kiitos
Täname teid
谢谢
Thank You
Tak
感謝您
Obrigado
Teşekkür Ederiz
감사합니다
Σας ευχαριστούμε
ขอบคุณ
Bedankt
Děkujeme vám
ありがとうございます
Tack