



Chapter 96 – Spinal Cord

Episode overview

1. Describe the arterial supply of the spinal cord
2. Label the spinal cord, and describe the major ascending and descending tracts
3. List the features of transverse cord syndrome.
4. Describe 3 common partial cord syndromes and list 3 most likely causes of each
5. Differentiate between Conus Medullaris and Cauda Equina Syndromes
6. List 10 causes of non-traumatic spinal cord dysfunction (6 intrinsic / 4 extrinsic)
7. Describe the clinical features + management of each:
 - a. Transverse Myelitis
 - b. Spinal SAH
 - c. Syringomyelia
8. What are 4 causes of spinal cord infarction? And what is the common cord syndrome?
9. List 5 RFs for spinal epidural abscess. And list 4 common bacteria implication in epidural abscess
10. List 4 causes of spinal epidural hematoma
11. List 4 possible investigations for spinal epidural abscess and diskitis. What are the expected findings?
12. How does the treatment of diskitis differ from epidural abscess?
13. Describe clinical presentation of spinal neoplasm, investigations and management.

Wisecracks

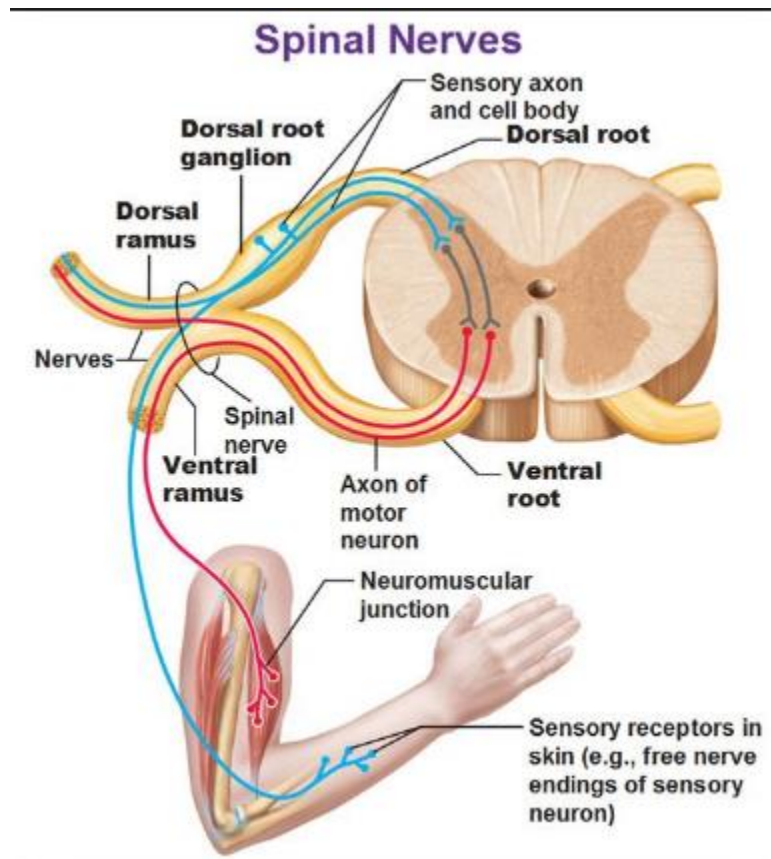
1. Spinal cord syndrome recap
2. Immunodeficiency myelopathy ddx

Rosen's in Perspective

“Like the brain, the spinal cord is covered by three meningeal layers: (1) the inner pial layer, (2) the arachnoid, and (3) the outer dural layer. At its lower end, the spinal cord tapers into the conus medullaris, then the lumbar and sacral nerve roots form the cauda equina as they descend caudally in the thecal sac before exiting the spinal canal at the respective foramina.

There is a cervical enlargement (cord level C5 to T1) gives rise to the brachial plexus and subsequently to the peripheral nerves of the upper extremity. The lumbar enlargement (L2 to S3) gives rise to the lumbosacral plexus and peripheral nerves of the lower extremity. The space surrounding the spinal cord within the spinal canal is reduced in the area of the enlargements, potentially leaving the cord more vulnerable to compression in these regions.

At each level, the anterior root conveys the outflow of the motor neurons in the anterior horn of the spinal cord, and the posterior root contains sensory neurons and fibers that convey sensory inflow.” - Rosen's 9th Edition, Chapter 96



Check out: <http://www.frca.co.uk/article.aspx?articleid=100360> for some awesome anatomy pics all in one spot!

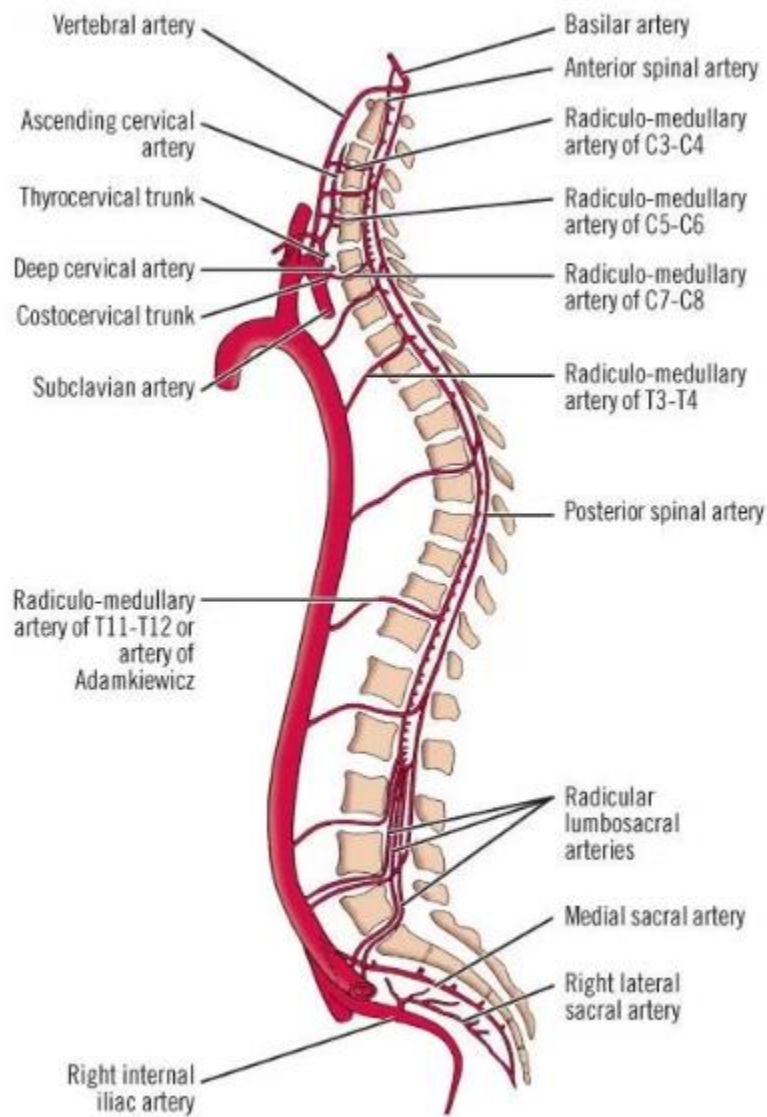
1) Describe the arterial supply of the spinal cord

See <http://www.frca.co.uk/images/spinal-cord4.jpg>

“The arterial supply of the spinal cord is derived primarily from two sources.

- *The single anterior spinal artery arises from the **paired vertebral arteries**. This anterior spinal artery runs the entire length of the cord in the midline anterior median sulcus and supplies roughly the anterior two thirds of the spinal cord.*
- *Blood supply to the posterior third of the spinal cord is derived from the smaller **paired posterior spinal arteries**.*

The anterior and the posterior spinal arteries receive segmental contributions from radicular arteries, the largest being the radicular artery of Adamkiewicz, which typically originates from the aorta between T8 and L4. The venous drainage of the cord largely parallels the arterial supply.” - Rosen’s 9th Edition, Chapter 96



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2) Label the spinal cord, and describe the major ascending and descending tracts

See Rosen's Figure 96.1 for the complete diagram.

- **Spinothalamic:** crossing of sensory information occurs near the level of entry of the spinal nerve. A cord lesion affecting one lateral spinothalamic tract will lead to decreased pain and temperature sensation on the **CONTRALATERAL** side of the body.
- **Posterior column:** crossing of sensory information occurs in the medulla
- **Corticospinal tracts:** decussation occurs in the medulla



3) List the features of transverse cord syndrome.

Aka: complete cord syndrome!

Usually in the setting of trauma (other causes: infarction, hemorrhage). If the complete syndrome lasts more than 24 hrs, recovery doesn't occur.

- Total loss of voluntary, distal to the injury:
 - Motor
 - **Sphincter dysfunction**
 - Sensory (i.e. NO sacral sparing or rectal tone)
 - **Sensory loss at a clear anatomic level**
 - Autonomic innervation

Reflexes still can (but not always) occur because they are mediated by the spinal levels; examples would be:

- DTR's
- Autonomic dysfunction:
 - Neurogenic shock
 - Priapism

If you have a bulbocavernosus reflex = spinal shock is over; if you don't have a BC reflex the spinal injury has likely led to spinal shock.

“Spinal shock refers to the loss of muscle tone and reflexes with complete cord syndrome during the acute phase of injury. Spinal shock typically lasts less than 24 hours but has been reported occasionally to last days to weeks. A marker of spinal shock is loss of the bulbocavernosus reflex, which is a normal cord-mediated reflex that may be preserved in complete cord lesions. The bulbo-cavernosus reflex involves involuntary reflex contraction of the anal sphincter in response to a squeeze of the glans penis or a tug on the Foley catheter. The termination of the spinal shock phase of injury is heralded by the return of the bulbocavernosus reflex; increased muscle tone and hyperreflexia follow later.” – Rosen's



4) Describe 3 common partial cord syndromes and list 3 most likely causes of each

Central Cord syndrome Central contusion to grey matter and CS+ST tracts from hyperextension falls	Brown-Sequard syndrome Hemisection of spinal cord penetrating trauma	Anterior cord syndrome hyperflexion/herniation/hypotension
<ul style="list-style-type: none"> • Most common • hyperExtension injury due to bulging lig. Flavum • Falls, MVCs in the elderly with OA and stenosis • Cerv. canal narrowing: disc protrusion / tumour 	<ul style="list-style-type: none"> • Penetrating injury • Tumours, epidural hematoma, AVMs, spondylolysis, DDD, herpes zoster myelitis, radiation or iatrogenic spinal injury 	<ul style="list-style-type: none"> • Post-op hypotension in aortic surgery (injury to vertebral art) • Infection, MI, vasospasm • Herniated bone frag/discs
<ul style="list-style-type: none"> • Bilateral MOTOR paresis • Upper > lower extremities • Distal muscle groups > proximal (anatomy!) • Sensory burning dysesthesias UE • +/- bladder dysfunction 	<ul style="list-style-type: none"> • Ipsilateral motor loss (CS) • Ipsilateral vib/proprioception loss (PC) • CONtralateral sensation of pain and temperature (ST) • MOST pts do not have the classic pattern (above); and have partial sensory/motor impairment 	<ul style="list-style-type: none"> • Loss of motor function, pinprick, light touch below the level • (preservation of posterior column: touch, position, proprioception) • Mixed motor/sensory findings
Bladder function, and ambulation may not return in the elderly patient	Good prognosis	Often poor functional improvement

5) Differentiate between Conus Medullaris and Cauda Equina Syndromes

This is a difficult differentiation in clinical practice! Lots of overlap! Can get a combined syndrome. They both can have saddle anesthesia, both can have variable sensory loss. Both can have impaired sphincter control.

Conus Medullaris ~L1 level	Cauda Equina Syndromes Dysfunction at the level of the NERVE ROOTS
Weakness may be of an upper motor neuron type (increased motor tone, abnormal reflexes) - Usually bilateral symptoms**	Weakness may be of a lower motor neuron type (flaccid, decreased reflexes) - Usually unilateral symptoms
Causes: <ul style="list-style-type: none"> • Central disc herniation • Neoplasm • Trauma • Vascular insufficiency 	Causes: <ul style="list-style-type: none"> • Midline rupture of an intervertebral disk (L4-L5) • Tumours
fecal/overflow urinary incontinence Impotence, distal motor weakness, sensory loss in a saddle distribution	fecal/overflow urinary incontinence Impotence, distal motor weakness, sensory loss in a saddle distribution. Urinary retention is the most consistent finding (>90%).



6) List 10 causes of non-traumatic spinal cord dysfunction (6 intrinsic / 4 extrinsic)

Ok, so you did your diagnostic sleuthing - brain vs. spinal cord vs. motor end-plate.

You think it's the spinal cord, now you have to figure out which process is behind the spinal cord dysfunction. It's generally broken down into:

- Primary (intrinsic cord disease - demyelination, infection, infarction)
- Secondary (compression of the cord, **originating outside the dura**)

Another term to know: Myelitis = spinal cord inflammation with dysfunction. The causes of myelitis are LEGION - think of it like saying a joint has "arthritis"

So here's the list: (See Box 96.1 in Rosen's for original table)

Nontraumatic Causes of Spinal Cord Dysfunction

- Intrinsic: inflammatory - infectious - infarction
- **PROCESSES AFFECTING THE SPINAL CORD OR BLOOD SUPPLY DIRECTLY**
 - MS
 - Transverse myelitis
 - Spinal arteriovenous malformation, subarachnoid hemorrhage
 - Syringomyelia
 - HIV myelopathy
 - Other myelopathies
 - Spinal cord infarction
- Extrinsic: infection - cancer (infiltration) - vascular
- **COMPRESSIVE LESIONS AFFECTING THE SPINAL CORD**
 - Spinal epidural hematoma
 - SEA
 - Diskitis
 - Neoplasm
 - Metastatic
 - Primary CNS

Workup:

- CT for trauma, or bone involvement
- MRI is crucial for inflammation, edema, infection, hemorrhage.
 - Gadolinium enhancement increases sensitivity
- LP is the next step (after imaging has ruled out compressive/mass lesions)
 - To look for inflammatory or demyelinating disorders



7) Describe the clinical features + management of each:

Spinal assessment:

- Key general history taking to figure out onset (rapid = likely vascular vs. slow = infectious); trauma history, cancer history, etc.
- Physical exam:
 - Motor: bulk, tone, strength, BC reflex, post-void residual
 - Sensory:
 - Light touch and pinprick - contralateral spinothalamic tract
 - proprioception/vibration - ipsilateral posterior column
 - Sacral dermatomal testing
 - Reflexes:
 - 0 - 4+ for DTR's
 - Can be absent in an UMN if sensory function is lost or spinal shock exists.
 - Clonus
 - Babinski's sign
- Things that may mimic spinal cord dysfunction but are easily treatable!
 - Hypoglycemia
 - hypokalemia

a) Transverse Myelitis

- Heterogenous group of inflammatory disorders that disturb ascending/descending pathways in the spinal cord (compressive lesions, trauma, infection, malignant infiltration). *Commonly (30%) post-viral infectious myelitis. May also be infectious, autoimmune (lupus, sjogren's, MCD).*
- Usually the thoracic cord affected.
- **Clinical features:**
 - Rapid progressive neuro deficits - maximally in 24 hrs or may take days/weeks.
 - Back pain; low grade fever (SEA).
 - Paraplegia, transverse level of sensory impairment, sphincter disturbance
 - May have paresis, hypertonia, hyperreflexia, clonus, positive Babinski's
 - ANS dysfunction
- **Diagnosis:** MRI with gadolinium enhancement; CSF often normal.
- **Management:**
 - Treat the underlying cause
 - No role for steroids
 - Hospitalization - consult neurosurgery/neurology

b) Spinal SAH

- See diagram at: <http://www.frca.co.uk/images/spinal-cord3.gif>
- Rare disease. May occur in the epidural, subdural, subarachnoid, intramedullary layers.



- USUALLY **caused by an AV malformation**. Can also occur from tumours, cavernous angiomas, spontaneously in anticoagulated pts.
- **Clinical features:**
 - Thunderclap, excruciating back pain. May extending into flank/radiate.
 - May have h/a and neck stiffness.
 - Neuro deficits vary:
 - Numbness, weakness, sphincter dysfunction.
- **Diagnosis:** MRI without contrast; LP can confirm the dx.
- **Management:**
 - Reverse any anticoagulation
 - Treat the underlying cause
 - Consult neurosurgery

c) Syringomyelia (aka syrinx)

- Cavitory lesion in the substance of the spinal cord. 90% of these patients have a Chiari I malformation. May also occur post spinal trauma or after meningitis.
- **Clinical features:**
 - h/a, neck pain, sensory changes (cape like), gait disorder, low cranial nerve dysfunction
 - Symptoms worsen with increasing ICP
 - Lower limb hyperreflexia, weakness/wasting of hands/arms, dissociated sensory loss (Post Column preservation of proprioception and light touch), ataxia
- **Diagnosis:** MRI
- **Management:** Follow-up evaluation usually adequate; NO ER treatment usually needed.

8) What are 4 causes of spinal cord infarction? And what is the common cord syndrome?

- Aortic dissection
- Post spinal cord/aortic arch surgery
- Global ischemic states
- Systemic lupus
- Vasculitis
- Cryptogenic

Presents with an anterior spinal cord syndrome. Proprioception, vibration and light touch usually preserved (posterior column).

9) List 5 RFs for spinal epidural abscess. And list 4 common bacteria implication in epidural abscess

Risk factors:

- Diabetes



- Injection drug use
- Chronic renal failure
- Alcoholism
- Immunosuppression
- Recent infection

Usually the infection is spread from a hematogenous source → either epidural space or vertebra with extension to the epidural space. Other common sources: SSTI's

Common bacteria:

- Staphylococcus aureus (>50%)
- Aerobic and anaerobic streptococci
- E. Coli
- Pseudomonas aeruginosa

Clinical features:

- Backache with tenderness to percussion
 - Usually days to weeks of pain
- Fever, sweats, rigors
- Progressive neuro deficits including bowel/bladder disturbance.
- Paraplegia and delirium are late findings

10) List 4 causes of spinal epidural hematoma

- Post trauma
 - LP
 - Epidural anesthesia
 - Spinal surgery
- Coagulopathic
 - Anticoagulated
 - Thrombocytopenia
 - Liver dz or alcoholism
- Spontaneous
 - Spinal or dural AVM
 - Vertebral hemangioma

Almost everyone needs a decompressive laminectomy.

11) List 4 possible investigations for spinal epidural abscess and diskitis. What are the expected findings?

- **MRI with IV contrast:** Enhancement
- **CBC:** WBC is insensitive and nonspecific. May be elevated @ 13-16000 / uL
- **ESR/CRP:** Not specific, but 100% sensitive if elevated.
- **Lumbar spine xray:**



- Destruction of the lumbar disc space (diskitis) usually after 2-4 weeks of disease
- Disc space narrowing
- Loss of vertebral body endplates

Notice these are NOT in the list:

- CT scan (bone artifact!)
- LP (relatively contraindicated: but would show elevated protein and inflammation)

12) How does the treatment of diskitis differ from epidural abscess (SEA)?

SEA:

*“Management: Urgent surgical consultation for decompression usually is required. Antibiotics effective against the most common pathogens (particularly *S. aureus*) should be started empirically. One possible regimen that covers gram-positive and gram-negative organisms consists of a third-generation cephalosporin (ceftriaxone 2000 mg every 24 hours) plus vancomycin (15 to 20 mg/kg IV every 8 to 12 hours), both given intravenously, plus rifampin (10 mg/kg by mouth or IV once a day).” - Rosen’s 9th Edition, Chapter 96*

Diskitis:

- Surgery is usually NOT needed
- Broad spectrum abx:
 - Vancomycin 10-15 mg/kg IV
 - Ceftriaxone 2 g IV

13) Describe clinical presentation of spinal neoplasm, investigations and management.

- Usually metastatic tumours from breast, lung, lymphoma
 - Thoracic sites most common
- Clinical features:
 - Back pain
 - Worse with recumbency
 - WORSE at night
 - Worse with valsalva, sneezing
 - Neuro deficits depending on the site.
- Investigations:
 - Normal plain radiograph and ESR <20 = very unlikely cancer
 - MRI with IV contrast
 - CT myelography
- Management:
 - This is an area where steroids may recommended for cases of acute spinal cord compression syndromes.
 - Radiation treatment is recommended for cord compression by tumours.
 - Surgery



Wisecracks

1) Quick recap of spinal cord syndromes

Please see Rosen's Figure 96.1 to see the full diagram

SYNDROME	SENSORY	MOTOR	SPHINCTER INVOLVEMENT
CENTRAL CORD	Variable	Upper extremity weakness, distal > proximal	Variable
BROWN-SÉQUARD	Ipsilateral position and vibration sense loss Contralateral pain and temperature sensation loss	Motor loss ipsilateral to cord lesion	Variable
ANTERIOR CORD	Loss of pin and touch sensations Vibration, position sense preserved	Motor loss or weakness below cord level	Variable
TRANSVERSE CORD (COMPLETE)	Loss of sensation below level of cord injury	Loss of voluntary motor function below cord level	Sphincter control lost
CONUS MEDULARIS	Saddle anaesthesia may be present, or sensory loss may range from patchy to complete transverse pattern	Weakness may be of upper motor neuron type	Sphincter control impaired
CAUDA EQUINA	Saddle anaesthesia may be present, or sensory loss may range from patchy to complete transverse pattern	Weakness may be of lower motor neuron type	Sphincter control impaired

2) Immunocompromised Myelopathy DDX:

- HIV myelopathy
- Toxoplasmosis
- Lymphoma
- VZV infection
- CMV infection