# SPINAL CORD LESIONS

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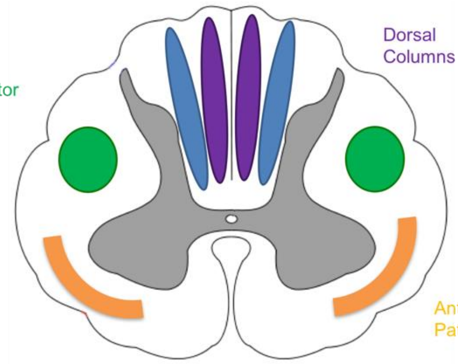
## BACKGROUND ANATOMY




The three most clinically important tracts within the spinal cord are:

1. The dorsal columns, which run dorsally
2. The lateral corticospinal tracts, which run laterally as part of the lateral motor systems
3. The spinothalamic tracts, which run anteriorly in the anterolateral pathways

## ANATOMICAL SUMMARY OF TRACTS

| NAME OF TRACT | FUNCTION | ROUTE OVERVIEW |
|---|---|---|
| Dorsal Columns | Carrying information on fine touch, vibration, and proprioception from the periphery towards the brain (particularly to the primary somatosensory cortex) | Information runs in the ipsilateral (same side as stimulus) dorsal column, up to the caudal medulla. Here, fibres in the more lateral cuneate fasciculus (representing the upper body) synapse in the ipsilateral nucleus cuneatus, and fibres from more medial gracile fasciculus (lower body) synapse on the ipsilateral nucleus gracilis. The post-synaptic neurons then decussate as the internal arcuate fibres, and continue towards the thalamus that is contralateral to the original sensory stimulus. |
| Lateral Corticospinal Tracts | Carrying efferent impulses from the motor cortices to the peripheral musculature, to control fine, voluntary movement | Motor neurons arising in the motor cortices descend through the ipsilateral cerebral hemisphere, condensing down in the corona radiata to form the posterior limb of the internal capsule. This dense motor bundle then continues through the cerebral penduncles and ventral pons until it reaches the cervico-medullary junction. At this point, 85% of descending motor fibres decussate in the medullary pyramids, and descend contralaterally to their hemisphere-of-origin as the lateral corticospinal tract. |
| Spinothalamic Tracts | Carrying information on pain and temperature from the periphery towards the brain | First order sensory neurons enter the cord from the periphery and synapse immediately in the dorsal horn's marginal zone. The second order neurons can then ascend/descend a few spinal levels in Tract of Lissauer before decussating via the anterior commissure, which takes 2-3 spinal levels. The second order axons then travel via the spinothalamic tract contralateral to original stimulus, towards the thalamus. |

# CLINICAL SYNDROMES RESULTING FROM CORD LESIONS

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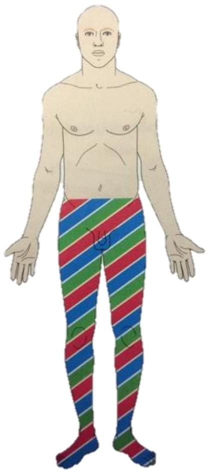
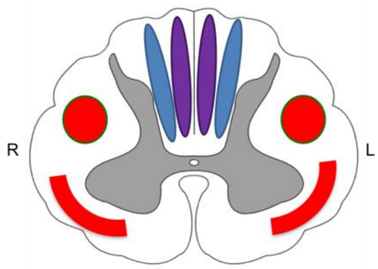
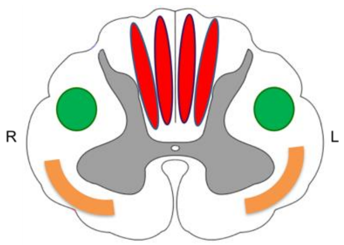
|---|---|
| MOTOR LOSS | |
| FINE TOUCH/ VIBRATION LOSS | |
| TEMPERATURE/PAIN LOSS | |

**N.B. LESIONED TRACTS ARE REPRESENTED IN RED IN THE PATHOLOGY PICTURE**

| TYPE | PATHOLOGY PICTURE | CLINICAL PICTURE | DESCRIPTION |
|---|---|---|---|
| TRANSVERSE LESION |   |  | Complete transection of the cord results in loss of fine touch/ vibration/ proprioception, as well as pain/temperature, and motor function bilaterally below the level of the lesion (i.e. the spinal level). Paralysis is typically upper motor neuron (spasticity, hyperreflexia, clonus, positive Babinski…) though there may be a lower motor neuron picture acutely due to spinal shock. |
| ANTERIOR LESION |   |  | Bilateral upper motor neuron signs and loss of pain/temperature sensation below the spinal level. Lesion spares dorsal columns, therefore fine touch is preserved throughout. |
| POSTERIOR LESION |   |  | Bilateral loss of fine touch, proprioception and vibration sense below the spinal level. As only the dorsal aspect of the cord is affected, motor function and pain/temp sensation are spared throughout. |

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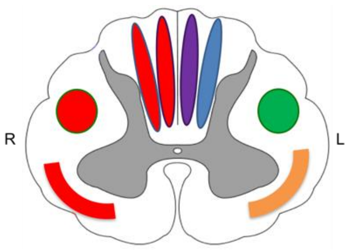
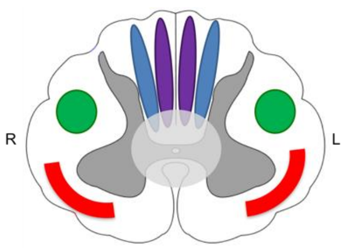
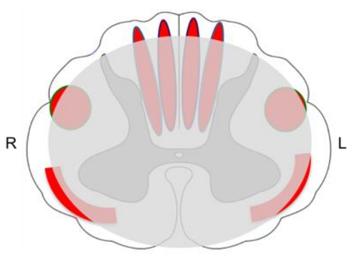
|---|---|---|---|
| HEMI-CORD LESION (BROWN-SEQUARD SYNDROME) |   |  | Loss of fine touch/ proprioception and vibration sense, as well as upper motor neuron signs ipsilateral to the lesion, with loss of pain/temp sensation contralateral to it. Discrepancy is due to immediate decussation of spinothalamic fibres, whereas dorsal columns/ lateral corticospinal tracts run ipsilateral to the side of the body they supply until the medulla. A hemi-cord lesion may damage spintothalamic fibres pre-decussation, sometimes leading to an ipsilateral band of pain/temp loss around the level of the lesion. |
| SMALL CENTRAL CORD LESION |   |  | A small central cord lesion disrupts only the spinothalamic fibres at the level of the lesion, as they decussate in the anterior commissure. Thus, a cervical central cord lesion results in a cape of pain/temp loss over the arms. |
| LARGE CENTRAL CORD LESION |   |  | A large central cord lesion disrupts all but the outer perimeter of the cord, where the sacral region is represented. Thus it causes dysfunction in all modalities below the level of the lesion, leaving a sacral island of neurological normality. |

## IDENTIFYING THE CAUSE OF A LESION

When trying to formulate a broad differential diagnosis for the cause of a spinal cord lesion, it can be useful to use a surgical sieve, such as INVITED MD:

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|---|---|
| **I**nfectious | Epidural abscess, tertiary syphilis (tabes dorsalis) |
| **N**eoplastic | Intrinsic/ extrinsic cord tumour |
| **V**ascular | Spinal stroke, AVM, cavernoma |
| **I**nflammatory | Transverse myelitis, MS, neuromyelitis optica |
| **T**raumatic | Stab wound, spinal fracture |
| **E**ndocrine | |
| **D**egenerative | Intervertebral disc prolapse, spondylosis |
| **M**etabolic | Vit B12 deficiency (combined subacute degeneration of the cord) |
| **D**rugs | |

Once the possible causes of a spinal lesion have been identified, return to the patient in question; review his/her particular risk factors (vasculopath/ malnourished/ malignancy) as well as the history of the presenting complaint (speed of onset, associated symptoms…). This additional information should help to narrow down a broad differential to the most likely underlying pathology.

## SUMMARY

When assessing a patient with a suspected spinal cord lesion, use clinical examination and history to first work out where in the cord (or elsewhere) the lesion is likely to be, and then, based on location and the clinical picture, think about what the lesion is likely to be, perhaps using a surgical sieve.

## FURTHER READING

*Neuroanatomy through clinical cases* by Hal Blumenfeld (from which the above 'clinical picture' figure diagrams were taken)