

Figure 5-1. Dorsal view of the cervical enlargement of the spinal cord and the corresponding roots of spinal nerves.

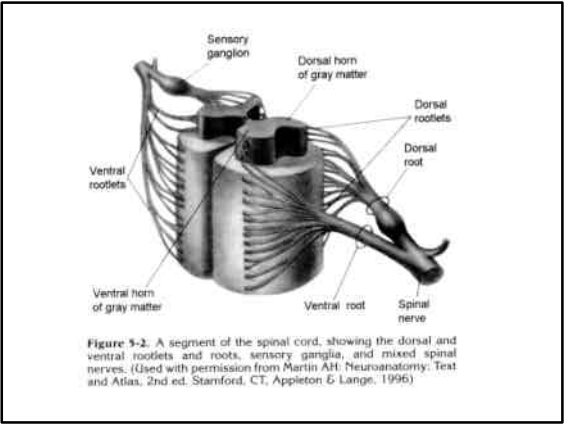


Figure 5-2. A segment of the spinal cord, showing the dorsal and ventral rootlets and roots, sensory ganglia, and mixed spinal nerves. (Used with permission from Martin AH: Neuroanatomy, Text and Atlas, 2nd ed. Stamford, CT, Appleton & Lange, 1996)

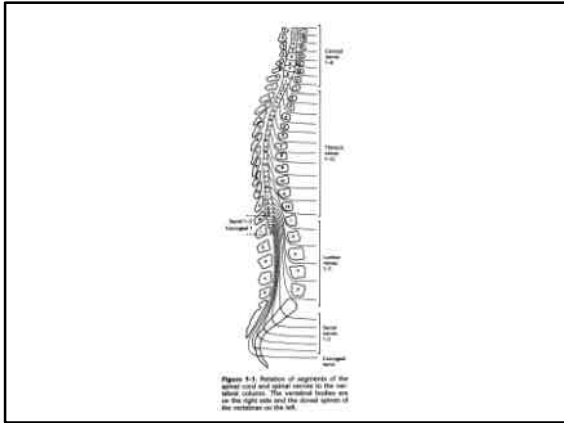


Figure 5-3. Section of segments of the spinal cord and spinal nerves to the vertebral column. The ventral bodies are on the right side and the dorsal spine of the vertebrae on the left.

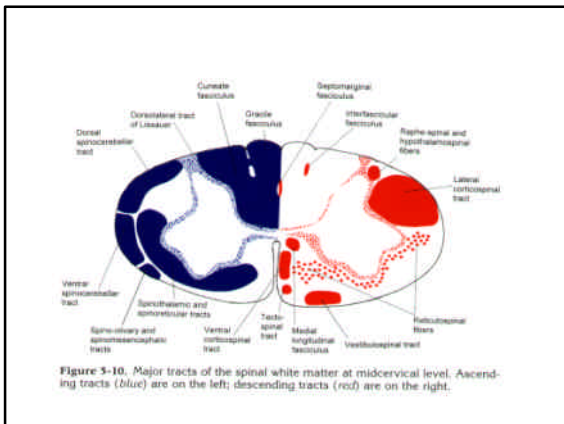
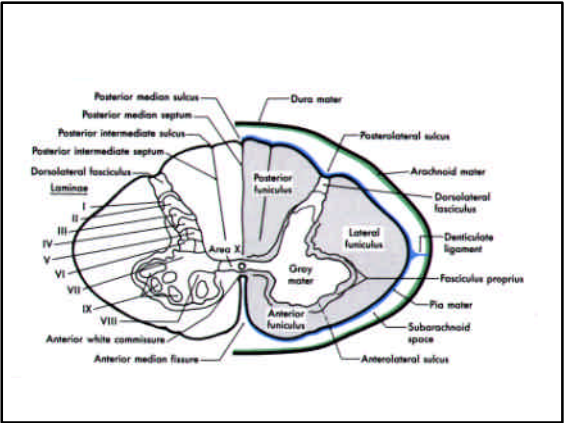
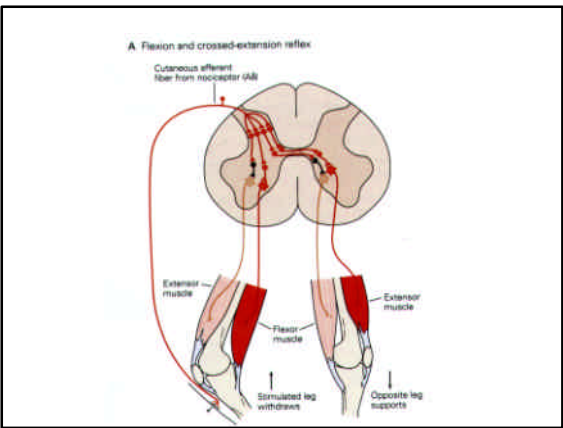
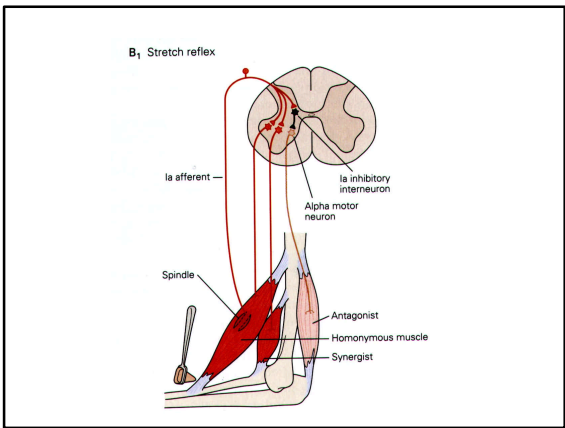
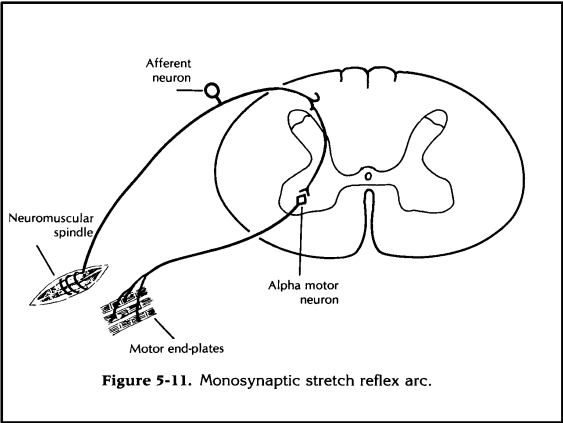
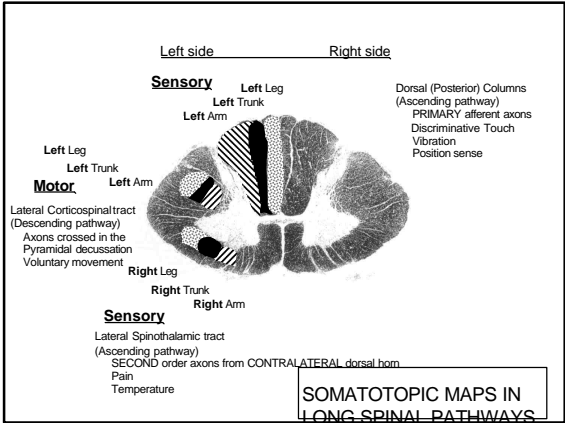
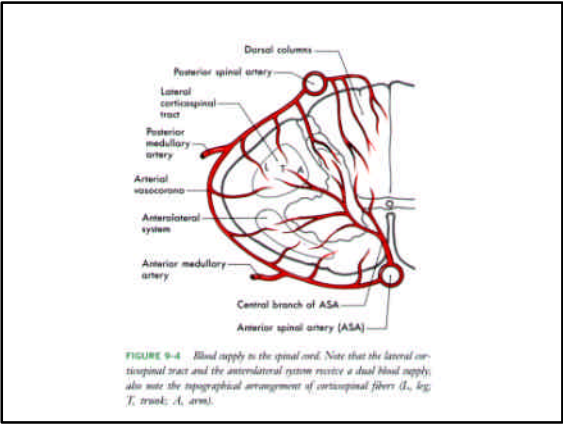
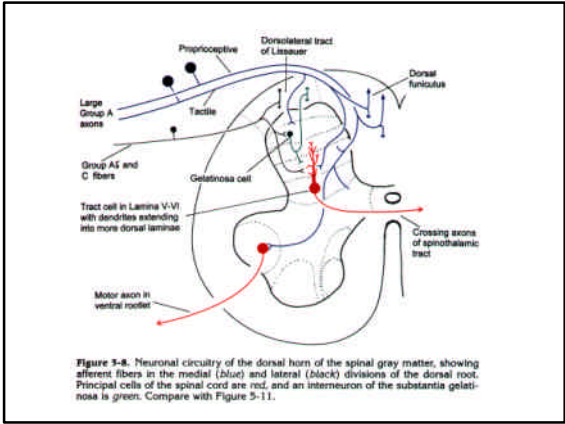


Figure 5-10. Major tracts of the spinal white matter at midcervical level. Ascending tracts (blue) are on the left; descending tracts (red) are on the right.

Segmental Variation of Spinal Gray Matter

- Great expansion of the ventral horns in the cervical and lumbar enlargements reflects the number of motor neurons innervating the limbs
- Intermediolateral cell column (preganglionic sympathetic neurons) present in lateral lamina VII in segments T1 through L2 or L3
- Sacral autonomic nucleus (preganglionic parasympathetic neurons) present in lateral lamina VII in segments S2 through S4
- Nucleus dorsalis of Clarke (origin of the dorsal spinocerebellar tract) present in medial lamina VII in segments T1 through L2 or L4
- Spinal border cells (contribute to ventral spinocerebellar tract) present at the lateral edge of the ventral horn in lumbar segments
- Spinal accessory nucleus present in lateral regions of the ventral horn in segments C1 through C5
- Phrenic nucleus in ventromedial portions of segments C3 through C5
- Onuf's nucleus in most ventral part of ventral horn of segment S2



Spinal Injury

Functional losses that result from spinal injuries depend on

! the specific gray and white matter structures damaged AND
! the side and level of the cord at which the lesion occurs

Damaged structure → associated functional loss

Long ASCENDING (sensory) pathways → loss of associated function from the body BELOW the level of the lesion

- a. **Dorsal Columns** → loss of touch, vibration and position sense IPSILATERAL to the lesion
- b. **Spinothalamic tract** → loss of pain and temperature sense CONTRALATERAL to the lesion, from about 2 segments BELOW the level of the lesion
- c. **Spinocerebellar** tracts → disruption of coordinated movement (especially gait) and muscle tone; bilateral limb dystaxia

Long DESCENDING (motor) pathways → loss of function in muscles BELOW the level of the lesion IPSILATERAL to the lesion (N.B. which may be CONTRALATERAL to the cells of origin of the axons)

- a. **Corticospinal tract** → distal weakness (paresis)/paralysis of voluntary movement of distal muscles, hyperreflexia, spasticity = "upper motor neuron" signs
- b. **Bulbospinal tracts** (reticulospinal, vestibulospinal) → disruption of muscle tone; abnormal reflexes
- c. **Hypothalamospinal tract** (* above T1) → Homer's Syndrome IPSILATERAL to the lesion (ptosis (dropping eyelid), mydriosis (pupillary constriction), anhidrosis (lack of sweating), enophthalmos (eyeball retraction))

Additional SEGMENTAL motor and sensory losses at the level of the lesion

- a. **ventral horn** motor neurons → flaccid paralysis and atrophy of skeletal muscles innervated by that spinal level ("lower motor neuron" signs)
 - b. damage to crossing axons forming the lateral spinothalamic tracts (**anterior white commissure** below the central canal) → bilateral "loss" of pain and temperature sense from a region of the body slightly below the lesion
 - c. damage to **dorsal roots**, **dorsal horn**, **ventral horn**, **ventral roots** → loss of segmental reflexes at the level of the lesion
- 2 After recovery from a period of "spinal shock," segmental reflexes BELOW the level of lesion return but are **hyperactive**