What kinds of information do most of these small diameter, thinly myelinated or unmyelinated axons carry?

What components of the autonomic nervous system are present in the sacral levels of the spinal cord, and what structures do they innervate?

Is this the dorsal (posterior) or ventral (anterior) surface of the spinal cord? How did you decide?

About this Atlas
- Mouse-over comments or the structure outlines and arrows with attached note icons for additional information.
- Select categories of information to view IDENTIFY - CONNECTIONS/FUNCTIONS - CLINICAL
- Add your own drawings and notes

This is the S4 or S5 spinal cord segment. However this part of the cord is enclosed by the L1 vertebra. What's going on?
Afferent information concerning movements of the ipsilateral lower extremity is conveyed to the cerebellum by the dorsal spinocerebellar tract. Yet strangely this tract is not present in the spinal cord caudal to about the L2 segment. What's going on? Can you briefly describe how mechanosensory information concerning the ipsilateral foot and ankle reaches the cerebellum?

How would you test the integrity of this tract (and the other components of the system it is part of) in a patient? Name two tests. Where on the patient would you perform them?

Which of the deep tendon (muscle stretch) reflexes might be absent or difficult to elicit if this level of the cord were damaged?

What is the location of muscles that are innervated by motor neurons located laterally (STAR) in the ventral horn gray matter? What muscles are innervated by motor neurons located more medially (CIRCLE)?

What kinds of sensory information does this tract convey?
One way to identify this segmental level of the spinal cord is by the absence of a prominent lateral enlargement of the anterior horns and by the presence of a very large Clarke's nucleus. This nucleus (also called the nucleus dorsalis or column of Clarke) forms a bulge in the intermediate gray matter that pushes up into the posterior columns. Be sure that you identify Clarke's nucleus in this section (it is one of the circled structures). While Clarke's nucleus extends from T1 to L2, it is largest from about T10-L2.

What nucleus contains the cell bodies of axons that travel in this tract? Do they carry information concerning the same side of the body or the opposite side? About the ARM or the LEG?

If a meningioma compresses the anterior lateral spinal cord here, at this level, what SENSORY abnormality is the patient's neurologic exam most likely to reveal?
If this region is damaged at this level, and on this side, name 3 signs or symptoms that the patient is likely to show on neurologic exam.

Neurons whose cell bodies are located here send their axons outside the CNS to synapse with what specific cells? Do those axons cross the midline?

Where are the cell bodies of these axons located? On which side of the nervous system, and in what structures?

There is a ventral as well as a dorsal spinocerebellar tract, but we won't discuss it further in this Atlas because its functions and clinical importance remain unclear. **OPTIONAL INFORMATION**
Say a little about the connections and functions of this tract.

A small central lesion in the spinal cord like the one shown in **RED** would impair what kinds of sensation? On which side of the body, or on both sides? Given this level of the spinal cord, roughly where would you predict that sensation is lost? (You can consult a Dermatome Map for specific information.)
What space are these structures located in?

Many axons traveling in this tract will synapse in nucleus gracilis. In what part of the CNS is that nucleus located? What kinds of sensation are these axons essential for? Will these axons cross the midline before they synapse?

Several years after a spinal cord lesion indicated by the red filled area, would you expect the patient to show upper motor neuron or lower motor neuron signs/symptoms in the LEG and FOOT? Would they be present ipsilateral or contralateral to the lesion?

The central canal of the spinal cord, is enormously enlarged in this section. This is not normal, and we're not really sure of the cause.
Patients often report the pain produced by cardiac ischemia as coming from the chest wall or from one or both arms. These regions are innervated in part by upper thoracic spinal cord segments. This is an example of referred pain in which pain information from the heart (carried by visceral sensory fibers into the upper thoracic cord) is referred to somatic structures from which somatic sensory fibers enter the same levels of the spinal cord.

Can you think of a reasonable explanation?

Please don't be concerned with the details of identifying Mid-Thoracic vs this Upper Thoracic level of the spinal cord. What IS important is that you identify the major tracts, and think about what happens if there is disease or injury affecting the thoracic cord.

A Short Case
A 67-year-old man has a year-long history of progressive weakness of his right leg. He has also noticed numbness and tingling in his left leg. Neurologic exam demonstrates:
- Weakness, exaggerated deep tendon (muscle stretch) reflexes, and increased tone in the right leg and foot, and a dorsiflexor plantar response (up-going toe) on the right.
- Decreased joint position sense in the right leg and foot.
- Decreased pinprick sensation on the left side of the trunk from about 2.5 cm (1 in) below the level of the nipple on down, and including the entire left leg and foot.
Other parts of the exam were reported as normal.

Can you localize the lesion? In other words, can you say approximately WHERE in the spinal cord the damage is? WHAT STRUCTURES are damaged to produce each sign and symptom?

Signs and symptoms that occur together and characterize a particular disease, condition, or structural lesion collectively constitute a SYNDROME. The signs/symptoms of the 67-year-old patient whose neurologic exam is described in the Short Case above constitute the Brown-Sequard syndrome. Review the key features of the syndrome and the lesion that produces it.
Injury to the T1 - T2 spinal cord can cause **Horner's syndrome**. List the key signs and symptoms of Horner's syndrome. Explain how a lesion at this level could produce it.

Which side of the patient would be affected - the same or the opposite side? Where else in the nervous system could a lesion produce Horner's syndrome - make a list.

Patient with unilateral lesions of the thoracic spinal cord below T1 routinely present with significant paralysis of the ipsilateral lower leg and foot, less weakness of hip muscles, and no apparent weakness of intercostal or abdominal muscles. How might we explain this "gradient of weakness"? (Note that the anterior corticospinal tract doesn't extend far enough caudal in the spinal cord to be the answer). By the way, why isn't the hand affected by lesions below T1?
Describe how you could test for the integrity of this tract (and the large structure in which its axons synapse). On actual exam, you discover that the patient has signs and symptoms of upper motor neuron paralysis because the corticospinal tract has been interrupted. Will you be able to detect deficits produced damage to the indicated tract? Why or why not?

BEWARE: This huge lateral expansion of the ventral horn gray matter is not the intermediolateral column. Compare its size with the slender, “pointy” intermediolateral column in upper thoracic segments (it is labeled in A-6).

If you looked at a neighboring section prepared with a cell stain, you’d discover this area contains the cell bodies of some very large MOTOR NEURONS. Note their position, way out lateral. Can you predict where the skeletal muscle that they innervate is located? What about the motor neurons in the region indicated by the STAR?

Axons in the spinothalamic tract are arranged according to the body region represented. Axons carrying information about the leg and foot are located more lateral than axons carrying information about the trunk. Axons concerned with the upper extremity are added medially.

QUESTION: Do these axons carry information about the ipsilateral or contralateral side. If they have crossed, where did this occur?
What signs and symptoms is a patient likely to show if she has a large demyelinated area (an MS plaque) in the region that is filled-in here?

If a patient has a recent lesion that has damaged motor neurons at the C5,6 segmental level of the spinal cord but spared the corticospinal tracts, what specific muscles do you expect will have reduced strength?

What about the tone of these muscles? Do you anticipate that it will be increased or decreased?

Which of the following reflexes would likely be abnormal? Describe...
- Biceps, Brachioradialis, Patellar tendon, Plantar response

Would you anticipate observing: 
- Clonus? Fasciculations? 
- Denervation atrophy 4 months later?

Clinically, the lateral corticospinal tract is the single most important descending motor pathway in the CNS. However, you should be aware of other descending pathways -- originating not from the cerebral cortex but from brainstem nuclei -- that also influence the activity of motor neurons. The best-understood of these pathways in humans are the reticulospinal and vestibulospinal tracts. These tracts are located in the ventral, medial white matter of the spinal cord. They are involved primarily in posture, gait, and balance, and they influence motor neurons located in medial parts of the ventral horns that innervate trunk and proximal muscles of the limbs.

This patient has signs and symptoms of a Lower Motor Neuron lesion. Compare them with what you would predict in an Upper Motor Neuron lesion.
(1) Have the axons of ganglion cells in these two tracts crossed the midline yet? Will they cross the midline before they synapse in nucleus gracilis and cuneatus?

(2) Branches of axons in *fasciculus cuneatus* also will synapse in the lateral cuneate nucleus. What is the significance of these connections?

This is the approximate location of the anterior corticospinal tract. Unlike axons in the much larger lateral corticospinal tract, these axons did not cross the midline in the pyramidal decussation. The tract is indicated in dashes because it is variable in size (1 in 6 of us don’t seem to have one), and because it decreases in size as it runs caudal in the cervical cord, and apparently ends completely in upper thoracic segments.

**MORE INFORMATION**

This section is rostral to the cervical enlargement. Why do you think the dorsal horns are so skinny at this level?

Motor neurons present in the cord at C3,4 innervate the diaphragm. This is one reason that high cervical cord transection can be fatal (either immediately or later). You may have heard about actor Christopher Reeve, who had massive hemorrhaging in his upper cervical cord following a horseback riding accident. He was quadriplegic and had to be placed on a ventilator because he couldn’t breathe on his own. He remained quadriplegic for the remainder of his life and could breathe without a respirator only after an electrical stimulation device was implanted in his diaphragm.

There’s also a group of motor neurons present at this level whose axons travel rostral and join the 11th cranial nerve. What muscles do they innervate?

Injury to the cord at this level can produce a Horner's syndrome as well as a number of other autonomic problems. Why is this the case? After all, there are no preganglionic autonomic neurons present in the cervical spinal cord.