

DIAGNOSIS OF PAIN AN ATLAS OF SIGNS AND SYMPTOMS

STEVEN D. WALDMAN

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PHYSICAL DIAGNOSIS OF PAIN AN ATLAS OF SIGNS AND SYMPTOMS

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Every Long Journey Begins with a First Step Confucius

This book is dedicated to my children— David Mayo, Corey, Jennifer, and Reid all of whom are sick of hearing me invoke the above quote ... but have nevertheless steadfastly followed its timeless wisdom in their daily lives!

Preface



"I knew it was too good to be true ... some things never change!" (From Kaplan EL, Mhoon D, Kaplan SP, Angelos P. Radiation-induced thyroid cancer: The Chicago experience. *Surgery* 146:979, 2009.)

While it's true that I hadn't quite finished medical school when Wilhelm Roentgen took an x-ray of his wife's hand, there is no doubt in my mind that this simple act forever changed the way medicine would be practiced. (Rumor has it he was actually trying to find a way to make her wedding ring disappear rather than diagnosis anything, as he was in love with his much younger and sexier x-ray tech!) Be that as it may, from that point on, physicians have constantly been looking for a way to make the diagnosis without actually examining the patient. X-ray gave way to fluoroscopy, which gave way to computerized tomography, which gave way to ultrasound, which gave way to magnetic resonance imaging, which has recently given way to PET scanning. Each modality's initial promise of an easier way to make the diagnosis always seemed to fall short of the mark. Yet hope springs eternal in the human breast, and many hope that rather than medical imaging, it will be the human genome that finally releases the medical profession from actually having to examine the patient!

In our perennial search for a less up close and personal way to come up with what's wrong with the patient, we must constantly be reminded that "some things never change" ... and one of those things is the amazing clinical utility of the properly taken history and properly performed physical examination. Yes, we actually have to touch the patient. Yes, we actually have to exert some effort. However, can you think of anything that has a higher yield for the patient and physician alike? I certainly can't.

In reviewing the Prefaces for the first and second editions of this text, I was struck by the spot-on accuracy of the musing of the great baseball player Yogi Berra when he said, "It's like déjà vu all over again!" Put another way, when all else fails ... EXAMINE THE PATIENT!

> Steven D. Waldman Summer 2015

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Functional Anatomy of the Bony Cervical Spine

THE VERTEBRAE OF THE CERVICAL SPINE

To fully understand the functional anatomy of the cervical spine and the role its unique characteristics play in the evolution of the myriad painful conditions that have the cervical spine as their nidus, one must first recognize that unlike the thoracic and lumbar spine, whose functional units are quite similar, the cervical spine must be thought of as being composed of two distinct and dissimilar functional units. The first type of functional unit consists of the atlanto-occipital unit and the atlantoaxial units (Figs. 1-1 and 1-2). While these units help to provide structural static support for the head, they are uniquely adapted to their

primary function of facilitating focused movement of the head to allow the optimal functioning of the eyes, ears, nose, and throat. The uppermost two functional units are susceptible to trauma and the inflammatory arthritides as well as to the degenerative changes that occur as a result of the aging process.

The second type of functional unit that makes up the cervical spine is very similar to the functional units of the thoracic and lumbar spine and serves primarily as a structural support for the head and secondarily functions to aid in the positioning of the sense organs located in the head (Fig. 1-3, *A*-*C*). Disruption of this second type of functional unit, which comprises the lower five cervical vertebrae and



• Figure 1-3 Functional units of the cervical spine in normal (A), flexed (B), and extended (C) positions.

their corresponding intervertebral discs, is responsible for the majority of painful conditions encountered in clinical practice (see Chapter 15).

THE MOBILITY OF THE CERVICAL SPINE

The cervical spine has the greatest range of motion of the entire spinal column and allows movement in all planes. Its greatest movement occurs from the atlanto-occipital joint to the third cervical vertebra. Movement of the cervical spine occurs as a synchronized effort of the entire cervical spine and its associated musculature, with the upper two cervical segments providing the greatest contribution to rotation, flexion, extension, and lateral bending. During flexion of the cervical spine, the spinal canal is lengthened, the intervertebral foramina become larger, and the anterior portion of the intervertebral disc becomes compressed (Fig. 1-3, B). During extension of the cervical spine, the spinal canal becomes shortened, the intervertebral foramina become smaller, and the posterior portion of the anterior disc becomes compressed (Fig. 1-3, C). With lateral bending or rotation, the contralateral intervertebral foramina become larger, while the ipsilateral intervertebral foramina become smaller. In health, none of these changes in size results in

functional disability or pain; however, in disease, these movements may result in nerve impingement with its attendant pain and functional disability.

THE CERVICAL VERTEBRAL CANAL

The bony cervical vertebral canal serves as a protective conduit for the spinal cord and as an exit point of the cervical nerve roots. Owing to the bulging of the cervical neuromeres as well as the other fibers that must traverse the cervical vertebral canal to reach the lower portions of the body, the cervical spinal cord occupies a significantly greater proportion of the space available in the spinal canal relative to the space occupied by the thoracic and lumbar spinal cord. This decreased space results in less shock-absorbing effect of the spinal fluid during trauma and also results in compression of the cervical spinal cord with attendant myelopathy when bone or intervertebral disc compromises the spinal canal (Fig. 1-4). Such encroachment of the cervical cord by degenerative changes or disc herniation can occur over a period of time, and the resultant loss of neurologic function due to myelopathy can be subtle; as a result, a delay in diagnosis is not uncommon.



• Figure 1-4 Cervical spondylosis. Sagittal T1-weighted spin echo (A) and sagittal T2-weighted fast spin echo (B) magnetic resonance images of the cervical spine demonstrate disc degeneration at essentially every cervical level, in addition to loss of disc space height and, in B, diminished signal intensity. Severe central canal stenosis is related to both anterior disc herniation with osteophytes and posterior ligamentous hypertrophy at most of the cervical levels. A focal area of high signal intensity within the cord at the C5-C6 level reflects posttraumatic myelomalacia. (From Resnick D, Kransdorf MJ, editors: *Bone and joint imaging*, ed 3, Philadelphia, 2005, Saunders, p 147.)

The cervical vertebral canal is funnel-shaped, with its largest diameter at the atlantoaxial space and progressing to its narrowest point at the C5-C6 interspace. It is not surprising that this narrow point serves as the nidus of many painful conditions of the cervical spine. The shape of the cervical vertebral canal in humans is triangular but is subject to much anatomic variability among patients. Those patients with a more trifoil shape generally are more susceptible to cervical radiculopathy in the face of any pathologic process that narrows the cervical vertebral canal or negatively affects the normal range of motion of the cervical spine.

THE CERVICAL NERVES AND THEIR RELATION TO THE CERVICAL VERTEBRAE

The cervical nerve roots are each composed of fibers from a dorsal root that carries primarily sensory information and a ventral root that carries primarily motor information. As the dorsal and ventral contributions to the cervical nerve roots move away from the cervical spinal cord, they coalesce into a single anatomic structure that becomes the individual cervical nerve roots. As these coalescing nerve fibers pass through the intervertebral foramen, they give off small branches, with the anterior portion of the nerve providing innervation to the anterior pseudo-joint of Luschka and the annulus of the disc and the posterior portion of the nerve providing innervation to the zygapophyseal joints of each adjacent vertebra between which the nerve root is exiting. These nerve fibers are thought to carry pain impulses from these anatomic structures, and this notion of the intervertebral disc and zygapophyseal joint as distinct pain generators diverges from the more conventional view of the compressed spinal nerve root as the sole source of pain emanating from the cervical spine. As the nerve fibers exit the intervertebral foramen, they fully coalesce into a single nerve root and travel forward and downward into the protective gutter made up of the transverse process of the vertebral body to provide innervation to the head, neck, and upper extremities (Fig. 1-5).



• Figure 1-5 Position of cervical nerves relative to cervical vertebrae.

IMPLICATIONS FOR THE CLINICIAN

The bony cervical spine is a truly amazing anatomic structure in terms of both its structure and its function. The two uppermost segments of the cervical spine are vitally important to a human's day-to-day safety and survival, but with the exception of cervicogenic and tension-type headaches, they are not the source of the majority of painful conditions involving the cervical spine that are commonly encountered in clinical practice. However, the lower five segments provide ample opportunity for the evolution of myriad common painful complaints, most notably cervical radiculopathy and cervicalgia, including cervical facet syndrome.

2 Functional Anatomy of the Cervical Intervertebral Disc

The cervical intervertebral disc has two major functions: (1) to serve as the major shock-absorbing structure of the cervical spine and (2) to facilitate the synchronized movement of the cervical spine while at the same time helping to prevent impingement of the neural structures and vasculature that traverse the cervical spine. Both the shockabsorbing function and the movement and protective function of the cervical intervertebral disc are a function of the disc's structure as well as the laws of physics that affect it.

To understand how the cervical intervertebral disc functions in health and becomes dysfunctional in disease, it is useful to think of the disc as a closed, fluid-filled container. The outside of the container is made up of a top and a bottom called the endplates, which are composed of relatively inflexible hyaline cartilage. The sides of the cervical intervertebral disc are made up of a woven crisscrossing matrix of fibroelastic fibers that tightly attaches to the top and bottom endplates. This woven matrix of fibers is called the annulus, and it completely surrounds the sides of the disc (Fig. 2-1). The interlaced structure of the annulus results in an enclosing mesh that is extremely strong yet at the same time very flexible, which facilitates the compression of the disc during the wide range of motion of the cervical spine (Fig. 2-2).

Inside this container of the top and bottom endplates and surrounding annulus is water that contains a mucopolysaccharide gel-like substance called the nucleus pulposus (see Fig. 2-1). The nucleus is incompressible and transmits any pressure placed on one portion of the disc to the surrounding nucleus. In health, the water-filled gel creates a positive intradiscal pressure that forces the adjacent vertebrae apart and helps to protect the spinal cord and exiting nerve roots. When the cervical spine moves, the incompressible nature of the nucleus propulsus maintains a constant intradiscal pressure, while some fibers of the disc relax and others contract.

As the cervical intervertebral disc ages, it becomes less vascular and loses its ability to absorb water into the disc. This results in a degradation of the disc's shock-absorbing and motion-facilitating functions. This problem is made worse by degeneration of the annulus, which allows portions of the disc wall to bulge, distorting the ability of the nucleus pulposus to evenly distribute the forces placed on it through the entire disc. This exacerbates the disc dysfunction and can contribute to further disc deterioration, which can ultimately lead to actual complete disruption of the annulus and extrusion of the nucleus as well as render the disc more susceptible to damage from even minor trauma (Fig. 2-3; also see Chapter 3). The deterioration of the disc is responsible for many of the painful conditions that emanate from the cervical spine that are encountered in clinical practice (see Chapter 15).



• Figure 2-1 The cervical intervertebral disc can be thought of as a closed, fluid-filled container.



• Figure 2-2 The cervical intervertebral disc is a strong yet flexible structure, shown here in the range of motion of the cervical spine.



• Figure 2-3 Posttraumatic discovertebral injury: lucent annular cleft sign. A, Hyperextension injury. Lateral radiograph shows a linear collection of gas within the annular fibers of the intervertebral disc adjacent to the vertebral endplate. The lucent cleft sign (*arrow*), often seen after hyperextension injuries, is believed to represent traumatic avulsion of the annulus fibrosus from its attachment to the anterior cartilaginous endplate. **B**, Hyperflexion injury. Observe the gas density within the posterior portion of the C4-C5 disc (*arrow*) on this lateral radiograph obtained in flexion. This patient was recently involved in a rear-end impact motor vehicle collision and had severe neck pain. (From Taylor JAM, Hughes TH, Resnick D: *Skeletal imaging: atlas of the spine and extremities*, ed 2, St. Louis, 2010, WB Saunders.)

3 Nomenclature of the Diseased Cervical Disc

Much confusion surrounds the nomenclature that is used to describe the diseased cervical disc. Such confusion exists in part because of the use of a system of nomenclature that was devised before the advent of computed tomography and magnetic resonance imaging and in part because of the focus by radiologists and clinicians alike on the impingement of the intervertebral disc on neural structures as the sole source of pain emanating from the spine. This second viewpoint ignores the disc and facet joint as an independent source of spine pain and leads to misdiagnosis, treatment plans with little chance of success, and needless suffering for the patient. By standardizing the nomenclature of the diseased cervical disc, the radiologist and clinician can do much to avoid these pitfalls when caring for the patient with spinal pain. The following classification system will allow the radiologist and clinician to communicate with each other in the same language. It also takes into account the fact that the intervertebral disc may be the sole source of spinal pain and that certain findings on magnetic resonance imaging should point the clinician toward a discogenic source of pain and an early consideration of discography as a diagnostic maneuver prior to surgical interventions. More than 90% of clinically significant disc abnormalities of the cervical spine occur at C5-C6 or C6-C7.

THE NORMAL DISC

As was discussed in Chapter 2, the normal disc consists of the central gel-like nucleus pulposus, which is surrounded concentrically by a dense fibroelastic ring called the annulus. The top and bottom of the disc are made up of cartilaginous endplates that are adjacent to the vertebral body. The laws of physics (primarily Pascal's law) allow the disc to maintain an adequate intradiscal pressure to push the adjacent vertebrae apart. On magnetic resonance imaging, the normal cervical disc appears symmetrical with low signal intensity on T1-weighted images and high signal intensity throughout the disc on T2-weighted images. In health, the margins of the cervical disc do not extend beyond the margins of the adjacent vertebral bodies (Fig. 3-1).

THE DEGENERATED DISC

As the disc ages, both the nucleus and the annulus undergo structural and biochemical changes that affect both the disc's appearance on magnetic resonance imaging and the disc's ability to function properly. Although this degenerative process is a normal part of aging, it can be accelerated by trauma to the cervical spine, infection, and smoking. If the degenerative process is severe enough, many but not all patients will experience clinical symptoms.



As the degenerative process occurs, the nucleus pulposus begins to lose its ability to maintain an adequate level of hydration as well as its ability to maintain a proper mixture of proteoglycans necessary to keep the gel-like consistency of the nuclear material. Degenerative clefts develop within the nuclear material. Degenerative clefts develop within the nuclear materix, and portions of the nucleus become replaced with collagen, which leads to a further degradation of the shock-absorbing abilities and flexibility of the disc. As this process continues, the disc's ability to maintain an adequate intradiscal pressure to push the adjacent vertebrae apart begins to break down, leading to a further deterioration of function with the onset of clinical symptoms.

In addition to degenerative changes affecting the nucleus pulposus, the degenerative process affects the annulus as well (Fig. 3-2). As the annulus ages, the complex interwoven mesh of fibroelastic fibers begins to break down, with small tears within the mesh occurring. As these tears occur, the exposed collagen fibers stimulate the ingrowth of richly innervated granulation tissue that can account for discogenic pain. These tears can be easily demonstrated in magnetic resonance imaging as linear structures of high signal intensity on T2-weighted images that correlate with positive results when provocative discography is performed on the affected disc. When identified as the source of pain on discography, these annular tears can be treated with intradiscal electrothermal annuloplasty with good results (Fig. 3-3).

THE DIFFUSELY BULGING DISC

As the degenerative process continues, further breakdown and tearing of the annular fibers and continued loss of hydration of the nucleus propulsus lead to a loss of intradiscal pressure with resultant disc space narrowing, which can



• Figure 3-2 Contrast within the epidural space suggesting complete disruption of the disc annulus. *R*, Right. (From Waldman SD: *Atlas of interventional pain management*, ed 2, Philadelphia, 2004, Saunders, p 554.)



• Figure 3-3 Intradiscal electrothermal annuloplasty: schematic view. (From Waldman SD: *Atlas of interventional pain management*, ed 2, Philadelphia, 2004, Saunders, p 554.)

lead to an exacerbation of clinical symptoms. As the disc space gradually narrows owing to decreased intradiscal pressure, the anterior and posterior longitudinal ligaments grow less taut and allow the discs to bulge beyond the margins of the vertebral body (Fig. 3-4, *A*, *B*). This causes impingement of bone or disc on nerve and spinal cord, adding impingement-induced pain to the pain emanating from the disc annulus itself (Fig. 3-5). These findings are clearly demonstrated on magnetic resonance imaging and should alert the clinician to the possibility of multifactorial sources of the patient's pain and functional disability.

THE FOCAL DISC PROTRUSION

As the disc annulus and nucleus propulsus continue to degenerate, the ability of the annulus to completely contain and compress the nucleus propulsus is lost and with it the incompressible nature of the nucleus propulsus. This leads to focal areas of annular wall weakness, which allow the nucleus propulsus to protrude into the spinal canal or against pain-sensitive structures (Fig. 3-4, *C*). Such protrusions are focal in nature and are easily seen on both T1- and T2-weighted magnetic resonance images (Fig. 3-6). These focal disc protrusions may be either relatively asymptomatic if the focal bulge does not impinge on any pain-sensitive structures or highly symptomatic, presenting clinically as pure discogenic pain or as radicular pain if the focal protrusion extends into a neural foramen or the spinal canal.

THE FOCAL DISC EXTRUSION

Focal disc extrusion is frequently symptomatic because the disc material often migrates cranially or caudally, resulting in impingement of exiting nerve roots and the creation of an intense inflammatory reaction as the nuclear material irritates the nerve root. This chemical irritation is thought to be responsible for the intense pain that is experienced by many patients with focal disc extrusion and may be seen on magnetic resonance imaging as high-intensity signals on T2-weighted images (Fig. 3-7). Although more pronounced than a focal disc protrusion, focal disc extrusion is similar in that the extruded disc material remains contiguous with the parent disc material (Fig. 3-4, *D*).

THE SEQUESTERED DISC

When a portion of the nuclear material detaches itself from its parent disc material and migrates, the disc fragment is called a sequestered disc (Fig. 3-4, E). Sequestered disc fragments frequently migrate in a cranial or caudal direction and become impacted beneath a nerve root or between the posterior longitudinal ligament and the bony spine. Sequestered disc fragments can cause significant clinical symptoms and pain and often require surgical intervention. Sequestered disc fragments will often enhance on contrastenhanced T1-weighted images and demonstrate a peripheral rim of high-intensity signal due to the inflammatory reaction the nuclear material elicits on T2-weighted images. Failure to identify and remove sequestered disc fragments often leads to a poor surgical result. Magnetic resonance imaging of the cervical spine, cervical myelography with contrast-enhanced computed tomography, and discography will help the clinician to further delineate the type of disc herniation the patient is suffering from and aid in formulation of a treatment plan (Fig. 3-8).

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