ANATOMY OF THE NERVOUS SYSTEM

The Nerve and Nerve Root

Nerves comprise the central nervous system and peripheral nervous systems (1). A nerve is an enclosed, cable-like bundle of peripheral axons, which are long, slender projections of neurons. Nerves possess a three-layer fascial structure (Figure 15.1) that includes the innermost endoneurium that offers little mechanical support, a middle layer of perineurium, which is a thin but dense epithelium, and an outermost epineurium that acts as a cushion for tissue. The perineurium provides the most strength in tension and also maintains the integrity of the axon and prevents damage during trauma (2). Blood vessels to the nerves are located primarily in the epineurium. The blood vessels are slightly coiled to adapt to excursion of the nerves. All layers of the nerve are innervated and have a thin but potentially important plexus of nociceptors (2).

Figure 15.1: The Three-Layer Fascial Structure of a Nerve

Nerves are categorized into three groups based on the direction that signals are conducted. Afferent nerves conduct signals from sensory endings to the central nervous system (CNS). Efferent nerves conduct signals from the CNS along motor neurons to the muscles and reactionary aspects of the body. Mixed nerves involve both afferent and efferent axons within the same bundle. Any and all of these types can be injured during a number of processes.

Nerves are attached to the spinal cord at a region identified as the nerve root. The nerve root contains an anterior (ventral) and a posterior (dorsal) root, the dorsal containing the spinal
ganglion. Ventral roots house the motor, or efferent, fibers to the body, whereas dorsal roots house the afferent fibers to the CNS. In contrast, sympathetic fibers of the sympathetic nervous system, which is part of the autonomic nervous system that extends from the upper thoracic region (T1) to the lumbar region (L3), are both efferent and afferent.

The Peripheral Nerve

The point at which the nerve exits the spinal cord is called the nerve root and distal to the point where the ventral and dorsal roots connect is called the peripheral nerve. There are 31 pairs of spinal nerves that exit the spinal cord through openings between the vertebrae. Nerve roots in the cervical spine are identified by the caudal segment of the intervertebral foramen and exit above the correspondingly numbered vertebral body from C2 to C7 in regions identified as intervertebral foramen. For example, the C3 nerve root exits above the C3 vertebral body, as does the C5 nerve root above the C5 body. The single exception is the C8 nerve root exiting above the T1 body.

In the sagittal plane, the intervertebral foramen (Figure 15.2) is bordered superiorly and inferiorly by the pedicles, posteriorly by the facet joint, posterior-medially by the ligamentum flavum, anteriorly by the uncovertebral joint, and anterior-medially by the intervertebral disc and posterior longitudinal ligament (3). Within the thoracic and lumbar spines, each nerve root exits below the correspondingly numbered vertebral body; thus, L4 exits between L4 and L5. Similar structures border the thoracic and lumbar regions with the exception of an uncovertebral joint, which is unique to the cervical spine.
Cross-sectionally, the spinal cord lies within the spinal column and the dorsal and ventral nerve groups merge near the intervertebral foramen (Figure 15.3). In the cervical spine, the nerve roots exit in an anterior direction, whereas in the thoracic and lumbar spine, the nerves exit laterally or posteriorly.
Structural changes to the intervertebral discs, ligaments, and capsule lead to viscoelastic losses and movement abnormalities (4). In particular, flexion-extension movements may cause a variety of neurological symptoms in severe degenerated conditions (5). During extension, the spinal canal shortens and narrows because of infolding of ligaments. The ligamentous infolding causes dorsilateral encroachment in the canal. In addition, the disc may bulge posteriorly in selected situations, further reducing space dorsilaterally. These structural changes may lead to kinetic changes such as decreased movement, compression on dorsilateral nerve roots or nerve root ganglia, compression on the spinal cord, and pain (4).

Cervical spine myelopathy, which is a pathology associated with the spinal cord, is hallmarked by the stenotic encroachment of the cervical spinal cord and corresponding neurological changes (6). The encroachment may lead to structural and vascular changes and originates from sagittal narrowing of the spinal canal. The narrowing may cause compression of the spinal cord and often originates from 1) osteophytes secondary to degeneration of intervertebral joints, 2) stiffening of connective tissues such as the ligamentum flavum at the dorsal aspect of the spinal canal, which can impinge on the cord by “buckling” when the spine is extended, 3) degeneration of intervertebral discs together with subsequent bony changes and 4) other degenerative connective tissue changes (7).

Dynamic movements of the spinal cord are regulated by the spinal column and the anchoring elements of the spinal cord. The primary anchoring elements are the dentate ligaments and the filum terminale (8). Secondary stabilizers include Hoffmann’s ligaments, the dura mater, and the dural ligaments. In normal subjects, length changes of the spinal cord are from 4.5 to 7.5 cm, with flexion increasing tension in the spinal cord and extension decreasing tension (9). Spinal cord compression occurs from a number of mechanisms, most notably from the friction that is present from degenerative changes during movements of extension and flexion. Ventral osteophytes can prevent upward and downward movement of the spinal cord during physiological motions (10). Furthermore, thickening of tissues and bony changes called a spondylitic bar increase the friction placed upon the spinal cord during movements and cause permanent damage.
The dura mater is inherently strong and resistant to mechanical forces because it is made up of collagen fibers and contains approximately 7% elastin in the ventral region and up to 14% elastin in the dorsal region.

Nerve roots and peripheral nerves are also anchored by connective tissue structures. Nerve roots are anchored by the dura, which connects to the perineurium of the nerve root. In addition, the nerve roots are attached to the transverse processes in the lower cervical spine through a system of ligaments and soft tissue anchors, an extraforaminal process that is also present in the thoracic spine. Within the periphery, entrapment or compression of the nerve can occur within the series of tunnels that guide the course of the nerve or in regional ligamentous areas where ligaments serve to stabilize the nerve in a particular region.

Damage to the peripheral nerves and nerve roots may occur from a number of causes. With the exception of the dorsal root ganglia, nerve roots are normally mechanically insensitive. However, upon trauma or as a response to a chemical or mechanical change (which may manifest as a vascular change) within the environment of the structure, nerve fiber axons and nerve fiber terminals within peripheral nerve sheaths may demonstrate increased sensitivity to mechanical stress, a process known as mechanosensitivity.

Mechanically oriented space occupying material may be transient, (swelling or extruded disc material) or permanent (e.g., osteophytes). The most common compressive causes of radiculopathy are disc herniation and degenerative spine components such as osteophytes, facet joint hypertrophy, and ligament hypertrophy. Disc herniations occur when nuclear material from the acute soft disc herniation impinges on a nerve root either posterolaterally or intraforaminally. The degenerative causes are associated with a loss of disc height and a “hard disc” bulging with resultant compressive elements such as the ligaments and osteophytes. Location-wise, anterior causes (soft or hard disc herniation and osteophytes from the uncinate processes in the cervical spine) are the most common cause of radicular symptoms. Other causes include ischemia, trauma, neoplastic infiltration, spinal infections, postradiation, immune-mediated diseases, lipoma, and congenital disorders.
Ongoing compression may lead to ischemia and fibrosis of the nerve (21). Stresses such as pressure or a stretch on the nerve may be the catalyst that produces immediate symptoms (22). Clinically, this may translate into sharp bursts of pain that dissipate as soon as the offending force is removed or may lead to symptoms continuing on after the stimulus is removed (23). Worth noting is that sustained compression or tension of a nerve will compromise neural circulatory perfusion leading to ischemia (23). Ischemia can lead to increased adrenosensitivity, which may be the principle pathophysiological mechanism that underpins sympathetically maintained pain precipitated by nerve injury (24). It has also been hypothesized that any restriction of neural tissue excursion can contribute to peripheral nerve entrapment syndromes and that repetitive trauma from shearing could occur at the entrapment with normal functional movement patterns (25).

**Summary**

- Nerves are located in the central nervous system, within the spinal cord, and within the peripheral nervous system.
- The point at which the nerve exits the spinal cord is called the nerve root and where the ventral and dorsal root connect is called the peripheral nerve. A nerve is an enclosed, cable-like bundle of peripheral axons, which are long, slender projections of neurons.
- Ongoing compression may lead to ischemia and fibrosis of the nerve.

**ARTHROLOGY OF THE NERVOUS SYSTEM**

**Pliability of the nerve**

The flexibility of the nerve itself (endoneurium, perineurium, and epineurium) is pliable enough to avoid damage during most end-range activities. The internal anatomy of nerves permits the nerve to stretch approximately 10–20% before structural damage occurs (26). Nonetheless, selected mechanisms can cause nerve injuries including mechanical trauma from an acute compression injury, crush and percussion (such as compression from a cast or with compartment syndrome), a laceration from blunt trauma, penetrating trauma (stab wound), or a stretch injury.

Stretch injuries can be induced by traction or from a surgical or clinical procedure that extends the nerve beyond its tolerable limits of mobility (27). In animal studies, lengthening nervous tissue to greater than 6–15% of its total length may be detrimental to nerve function (28). Although
there is no single classification system reflecting nerve damage that is universally accepted, the 1943 classification proposed by Seddon is commonly quoted (29). The classification is based on three main types of nerve fiber injury and whether there is continuity of the nerve: axonotmesis, neuropraxia, and neurotmesis. Continued stress on a nerve can lead to diminished function of afferent conduction (30).

### Table 15.1: Seddon’s Classification for Anatomical Nerve Damage (29).

<table>
<thead>
<tr>
<th>Pathological Anatomical Continuity</th>
<th>Neurotmesis</th>
<th>Axonotmesis</th>
<th>Neuropraxia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Essential damage</td>
<td>May be lost</td>
<td>Preserved</td>
<td>Preserved</td>
</tr>
<tr>
<td></td>
<td>Complete disorganization</td>
<td>Nerve fibers interrupted</td>
<td>Selective demyelination</td>
</tr>
<tr>
<td>Clinical</td>
<td>Complete</td>
<td>Complete</td>
<td>Complete</td>
</tr>
<tr>
<td>Motor paralysis</td>
<td>Progressive</td>
<td>Progressive</td>
<td>Complete</td>
</tr>
<tr>
<td>Muscle atrophy</td>
<td>Complete</td>
<td>Complete</td>
<td>Very little</td>
</tr>
<tr>
<td>Sensory paralysis</td>
<td>Complete</td>
<td>Complete</td>
<td>Usually much sparing</td>
</tr>
<tr>
<td>Autonomic paralysis</td>
<td>Complete</td>
<td>Complete</td>
<td>Usually much sparing</td>
</tr>
<tr>
<td>Electrical phenomena</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td>Reaction of degeneration distal</td>
<td>Absent</td>
<td>Absent</td>
<td>Preserved</td>
</tr>
<tr>
<td>Nerve conduction potentials</td>
<td>Absent</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>Fibrillation</td>
<td>Present</td>
<td>Present</td>
<td>Occasionally detectable</td>
</tr>
<tr>
<td>Recovery</td>
<td>Essential</td>
<td>Not necessary</td>
<td>Not necessary</td>
</tr>
<tr>
<td>Surgical repair</td>
<td>1–2 mm/day after repair</td>
<td>1–2 mm/day</td>
<td>Rapid: days or weeks</td>
</tr>
<tr>
<td>Rate of recovery</td>
<td>According to order of innervation</td>
<td>According to order of innervation</td>
<td>No order</td>
</tr>
<tr>
<td>March of recovery</td>
<td>Always imperfect</td>
<td>Perfect</td>
<td>Perfect</td>
</tr>
<tr>
<td>Quality of recovery</td>
<td></td>
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</tbody>
</table>

Movement within the spinal canal involves up to 7 cm with extension to flexion (9,31). Cadaver studies have shown that the spinal cord slackens and tenses with flexion and extension movements (32), and when the cervical spine flexes, the cord moves cranially (33,34), and when the lumbar spine flexes, the cord position has conflicting results. Historically, cord movement has been purported to move cranially with lumbar flexion, but recent research has shown in one case series that in some subjects it moved cranially, some caudally, and in others it remained unchanged (35). Another cadaveric study found the cord remained unchanged, but the nerve roots became tense and...
moved medially (36). In addition to nerve displacement in the spinal canal, each nerve must be able to move within the natural nerve bed throughout the peripheral tissues (37). As the extremity assumes different functional positions, so too does each nerve relative to other tissues in the region. Movement of the peripheral nerves can also be facilitated by respiration; the median nerve was drawn 8mm toward the shoulder during a full inspiration (38). The capacity of each nerve to move relative to surrounding tissues cannot be overstated (37). For example, Wright et al. found that when the wrist was moved through 125° of movement from extension to flexion, the median nerve moved roughly 20 mm (39). In another cadaveric study, Wright et al. also found that the radial nerve required an average of 4.3 mm of excursion to accommodate 45° of wrist frontal plane motion and 8.8 mm of radial nerve excursion for 80° of elbow flexion from 10 to 90°. The same elbow motion caused 28% strain at the radial nerve. A combination of all available movement at the fingers, wrist, elbow, and shoulder created 9.4 mm of radial nerve motion at the wrist and 14.2 mm of motion at the elbow (40). The same 125° of sagittal wrist movement previously cited generated 13.6 mm of ulnar nerve movement at the wrist, and when hand, wrist, elbow, and shoulder movement were combined, 23.2 mm of ulnar nerve motion occurred at the wrist and 21.9 mm of motion occurred at the elbow. An in vivo study revealed approximately 1.5 cm of total movement in both the ulnar and median nerves at the wrist with full movement from flexion to extension (38). Elbow flexion caused ≥15% strain of the ulnar nerve, and wrist extension with radial deviation created similar strain at the wrist (41).

Each fascicle within a nerve must be able to move relative to other fascicles within the nerve. This capacity is permitted by the interfascicular epinerium. The lumbosacral nerve roots (L4-S1) move less with lower extremity movement during the straight leg raise maneuver and may require hip flexion >60° in order to generate significant displacement of the sciatic nerve in the lateral recess (42). Interestingly enough, the straight leg raise (SLR) maneuver without prepositioning the ankle resulted in greater distal displacement of the L5 and S1 nerve roots than when the ankle was prepositioned in dorsiflexion (43).
It has also been hypothesized that cervical dysfunction may provoke nerve tissue contributing to neurogenic pain condition (44). A central irritation as described accompanied by a peripheral irritation secondary to a host of pathologic entities such as a lack of normal excursion creates a condition where the nerve is pathophysiologically altered in more than one location. This condition has been referred to as a double-crush injury (45). This compound pathophysiologic condition acknowledges that any restriction in the normal excursion of nerve within the nerve bed may contribute to the pathology of the nervous system (25,46).

Other pathophysiologic processes that contribute to peripheral nerve pathology may include fibrous tissue buildup that limits extensibility of the neural tissue by making the nerve bed less pliable. A cadaver study found that a high percentage of specimens examined demonstrated fibrous bands around the arcade of Frohse, while two-thirds of the specimens showed a buildup of fibrous tissue at the distal end of the supinator muscle (47). It has been previously suggested that repeated movements around the elbow may cause the development of fibrous tissue, and this tissue may be a cause of peripheral nerve entrapment (48).

**Summary**

- The internal anatomy of nerves permits the nerve to stretch approximately 10–20% before structural damage occurs.
- It has also been hypothesized that cervical dysfunction may provoke nerve tissue contributing to neurogenic pain condition.
- Movement within the spinal canal involves up to 7 cm with extension to flexion.

**Online References**


33. Reid JD. Effects of flexion-extension movements of the head and spine upon the spinal cord and nerve roots. J Neurol Neurosurg Psychiatry. 1960;23:214–221.


