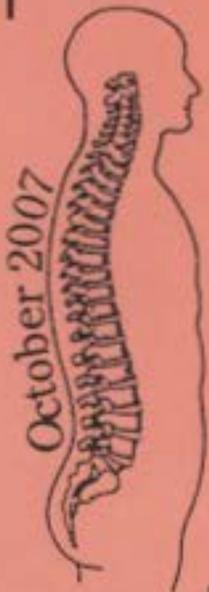


# PRIMER OF NECK AND BACK INJURIES FOR ATTORNEYS AND STAFF

*Third Edition*

by  
Gerald A. Schwartz, B.S., Pharmacy, J.D.



Virginia Trial  
Lawyers Association

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# **PRIMER OF NECK AND BACK INJURIES FOR ATTORNEYS AND STAFF**

THIRD EDITION, 2007

By:

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Alexandria, Virginia

## **PREFACE TO THE THIRD EDITION**

This third edition again is intended to be a primer on neck and back injuries for persons who have never handled a personal injury claim. Complex anatomy is described in simple, everyday language. Analogies have been used whenever possible to simplify medical concepts. After reading this outline, it is hoped that the reader will have a working knowledge of the anatomy of the neck and back in order to properly evaluate a neck and back injury case; to effectively interpret medical reports, x-ray, MRI and CT scan reports; and to effectively conduct direct and cross-examination of a medical expert.

Some parts of the first edition (November, 1992) were revised in the second edition in 1998. New material in the second edition included a discussion of "How to Get Favorable Medical Articles Into Evidence" (p. 118); Part Three, Fibromyalgia (pp. 120-126); and Part Five, Drugs Used to Treat Neck and Back Injuries (pp. 133-151). New material in the third edition (2007) includes an update on Drugs Used to Treat Neck and Back Injuries (p. 133-151) and Discography (p. 127).

## ACKNOWLEDGMENTS

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## CLASSIFICATION OF INJURIES

Injuries to the neck and back are classified in many ways. This outline will classify these injuries simply into three categories: PART ONE - Symptoms resulting from injuries to the spinal column and its contents; PART TWO - Hyperextension-Hyperflexion soft tissue injuries; PART THREE - Fibromyalgia.

### PART ONE

#### I. SPINAL COLUMN INJURIES

##### A. ANATOMICAL REGIONS OF THE SPINE.

The spine is divided into four regions: cervical (neck), thoracic (dorsal - upper back), lumbar (low back), and sacral (fused).

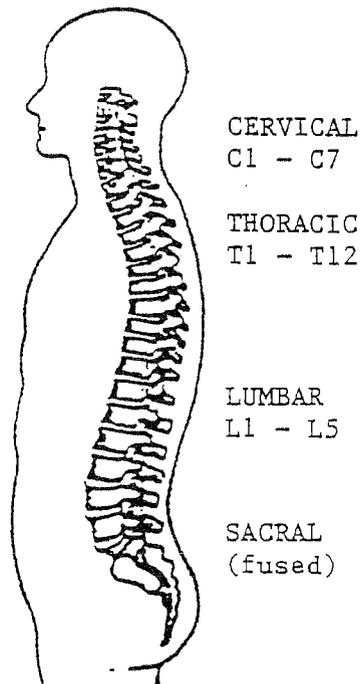


FIGURE 1

©See p.(ii)

## B. THE VERTEBRAL COLUMN.

The vertebral column (spine) is composed of individual bony vertebrae, hinged together by stacking one vertebrae on top of another. Each vertebrae is assigned to a region and labeled with a number starting from top to bottom. For example, the cervical vertebrae are numbered consecutively from top to bottom, from C1 through C7; the thoracic vertebrae are numbered consecutively from T1 through T12; and the lumbar vertebrae are similarly numbered from L1 through L5. The five sacral vertebrae (referred to as the sacrum) are fused together into one large bone. The junction between the last lumbar vertebrae (L5) and the first sacral vertebrae (the sacrum) is called the lumbosacral junction or lumbosacral joint and is abbreviated LS. Physicians often refer to the low back as the lumbosacral spine. Figure 1.

## C. THE VERTEBRAE.

A diagram of an individual vertebrae is found in Figure 2.

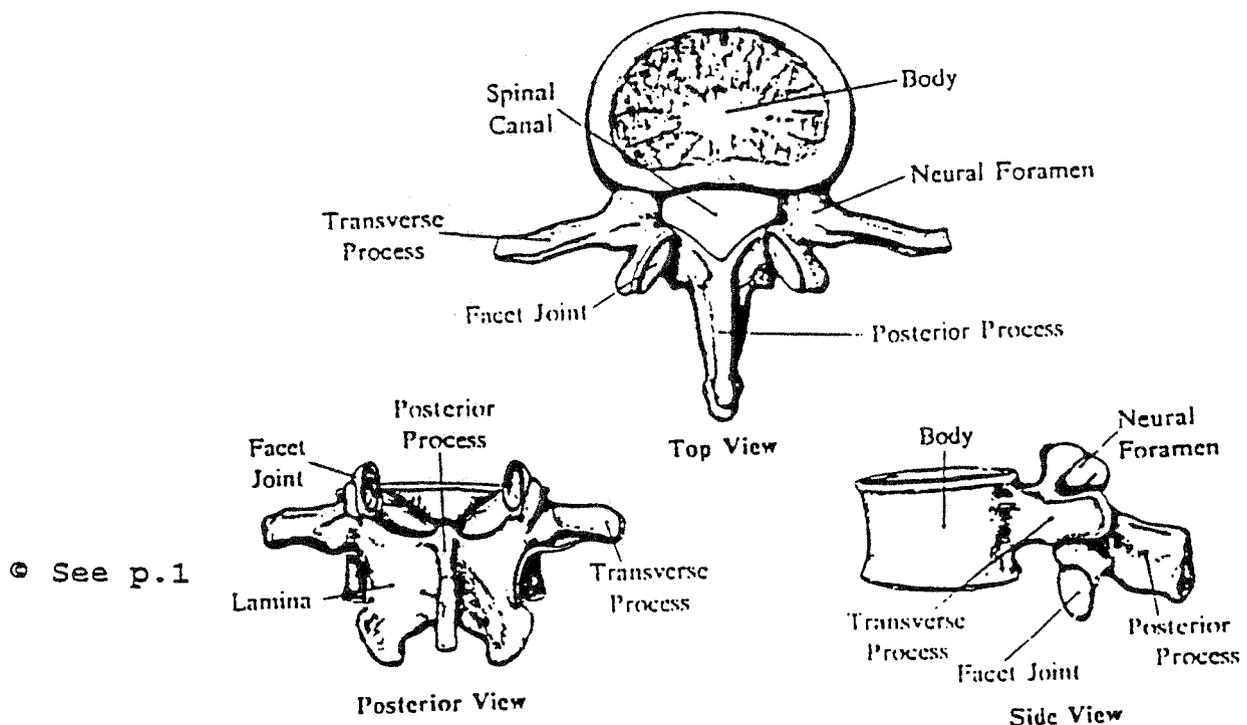


FIGURE 2

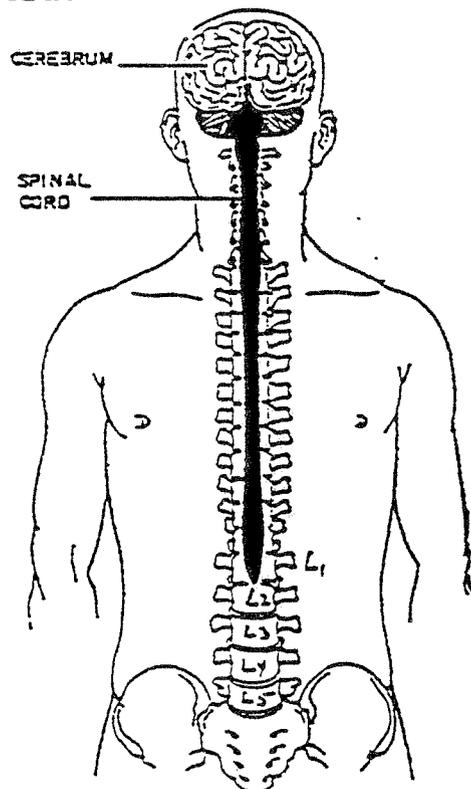
The spinal canal is formed by stacking one vertebrae on top of another around the cylindrical spinal cord. The piece of bone in the individual vertebrae, which surround the spinal cord, is referred to as the neural arch (neural referring to nerves and arch referring to an archway).

Lamina is a piece of bone found in the back of the vertebrae and forms the back of the neural arch, the "archway" to the cord. In performing an operation called a "laminectomy", the surgeon must first remove the bony lamina to operate on the spinal cord and the nerve roots.

#### D. THE VERTEBRAL COLUMN PROTECTS THE SPINAL CORD.

The spinal cord lies within the vertebral column and is surrounded and protected by the bony vertebrae. The spinal cord ends at the level of the first lumbar vertebrae and forms the cauda equina - a horses tail appearance of nerves which continue down the remainder of the spinal cord.

FIGURE 3



©See p.1

## E. THE SPINAL CORD:

### 1. LONG TRACTS:

The spinal cord is cylindrical measuring 26" long with an average width of 5/8". The spinal cord is an extension of the brain and is analogous to a telephone cable with ascending and descending long tracts of wires. The ascending tracts conduct impulses to the brain, and the descending tracts carry impulses away from the brain. The brain controls voluntary movements by sending and receiving nerve messages along the spinal cord through these long tracts called the pyramidal and extrapyramidal tracts.

Damage to the long tracts in the spinal cord is called myelopathy. Figure 4A and 4B is a cross-section of the spinal cord showing pyramidal and extrapyramidal ascending and descending long tracts inside the spinal cord.

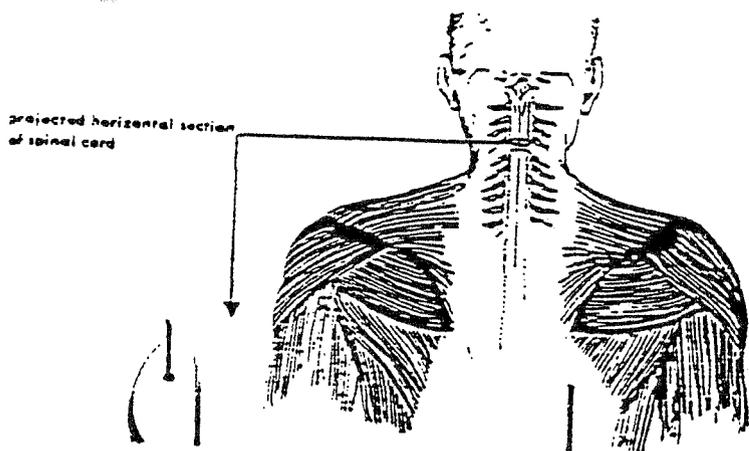


FIGURE 4A

©See p.1

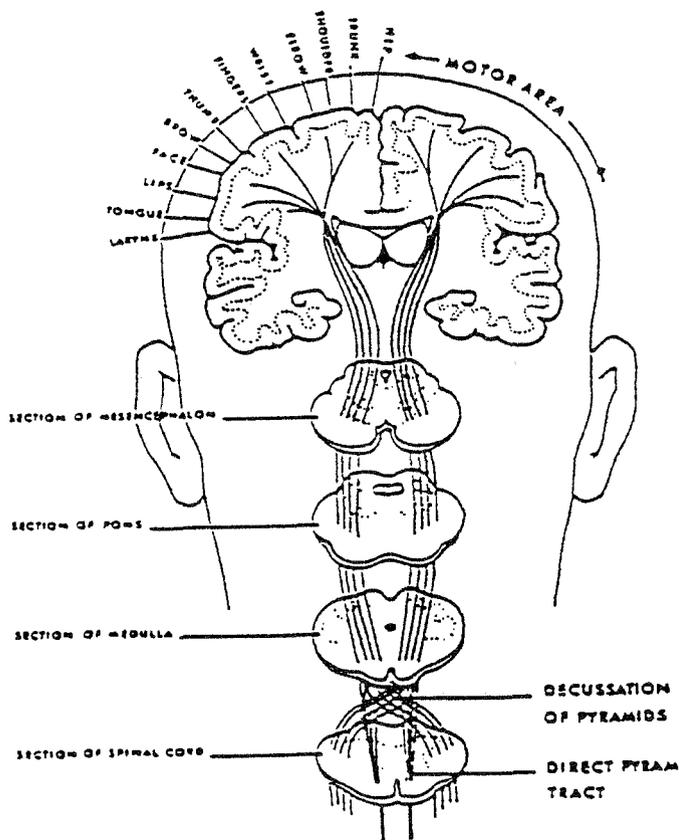


FIGURE 4B

## 2. MYELOPATHY - LONG TRACT INJURY:

Compression of the spinal cord produces a condition called myelopathy (myel = spinal cord; opathy = disease). Compression of the cord can be caused by many events, including a large central disc herniation pressing against the spinal cord; or a preexisting osteophyte present on the vertebrae striking the spinal cord during a rear-end collision with resulting hyperextension, flexion of the spine.

If the spinal cord is compressed, damage can be done to the "long tracts" present inside the spinal cord (pyramidal and extrapyramidal tracts)

If the spinal cord is compressed in the cervical area, the condition is known as cervical myelopathy. Symptoms include peculiar sensations in the hands with clumsiness and weakness, especially in the legs with hyperactive reflexes and some spasticity. The clinical picture often presents a patchy distribution of symptoms.

Patients with long tract damage to the spinal cord (myelopathy), will show a classic finding, on physical exam - a positive (+) Babinski test - described in the next section.

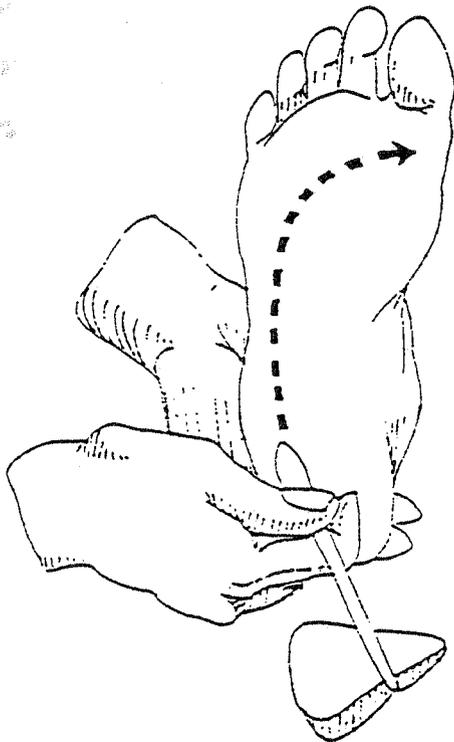
### (a) THE BABINSKI TEST:

This is a simple test developed over 100 years ago by a neurologist named Babinski. A positive test suggests an upper motor neuron injury in the pyramidal tract, one of the long tracts sending impulses to the voluntary muscles from the cerebral cortex of the brain down the spinal cord. A positive Babinski sign in an

infant (under 18 months of age) is insignificant since the infant's central nervous system is not fully developed.

The Babinski test is performed by stroking the bottom of the foot (the sole of the foot). This is called the plantar surface of the foot. The stroking motion is from the heel toward the toes. A Babinski test is positive if the big toe extends upward. The other toes may curl downward or fan out, while the big toe is extended upward, but this is not necessary for a positive test. In a normal person, a negative Babinski sign results when all the toes turn downward (in a plantar direction - toward the plantar surface of the foot - sole of the foot).

MEMORY AID: A good way to remember that the big toe in a (+) Babinski test goes upward is to associate Babinski with skiing from the upward slope of the mountain.



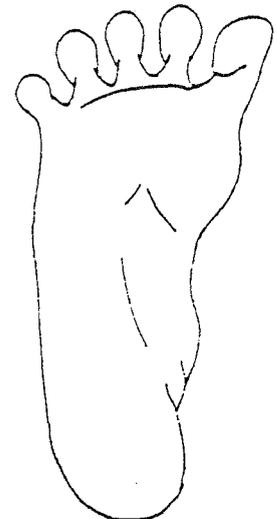
Babinski Test

FIGURE 5A



Negative Test

FIGURE 5B



Positive Test

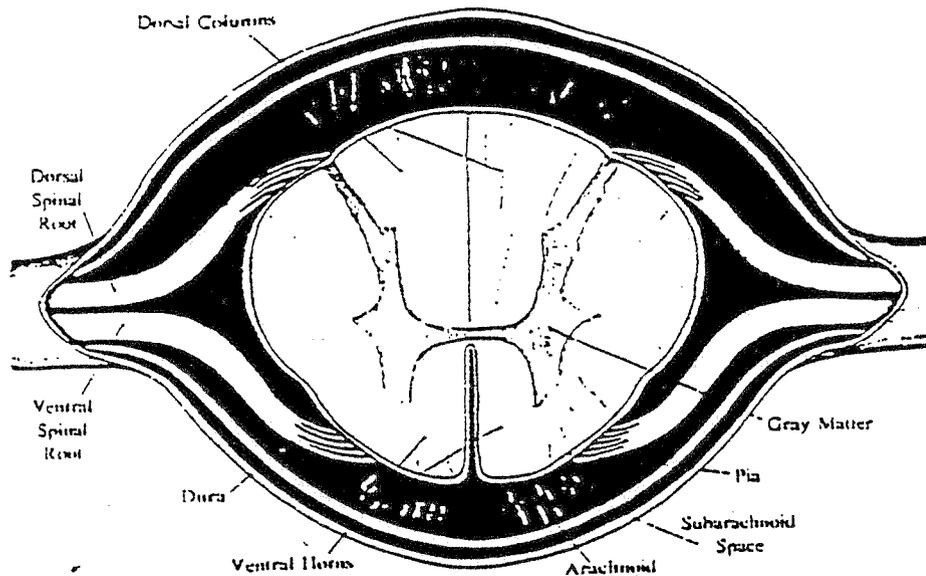
FIGURE 5C

### 3. THE DURA (THECAL SAC):

The spinal cord is covered and protected by the dura, also called the meninges, which consists of three layers from inside out as follows: (1) inside layer - pia mater (latin for soft mother since this layer is soft); (2) middle layer - arachnoid (arachnoid meaning spider since this layer looks like a spider web); and (3) outer layer - dura mater (latin for hard mother since this layer is tough and fibrous). The dura mater is also referred to as the thecal sac (not to be confused with fecal sac which is the bowel). Infection of the meninges is called meningitis.

The space between the first layer, pia mater, and the second layer, arachnoid, is called the subarachnoid space (under the arachnoid). The subarachnoid space contains the cerebrospinal fluid (CSF) that surrounds the brain and spinal cord. A spinal tap consists of removing cerebrospinal fluid (CSF) from the subarachnoid space; while a myelogram test consists of injecting a contrast dye in this space to outline the spinal cord and its nerve roots.

**PRACTICE NOTE - "INDENTING THE THECAL SAC":** The outside membrane covering the spinal cord and its nerve roots is the thecal sac, which is often referred to in MRI reports. For example, the radiologist often states "the lesion is indenting the thecal sac" - meaning that the space-occupying lesion such as a protruded disc, a protruding osteophyte, etc., is indenting the thecal sac of the spinal cord.



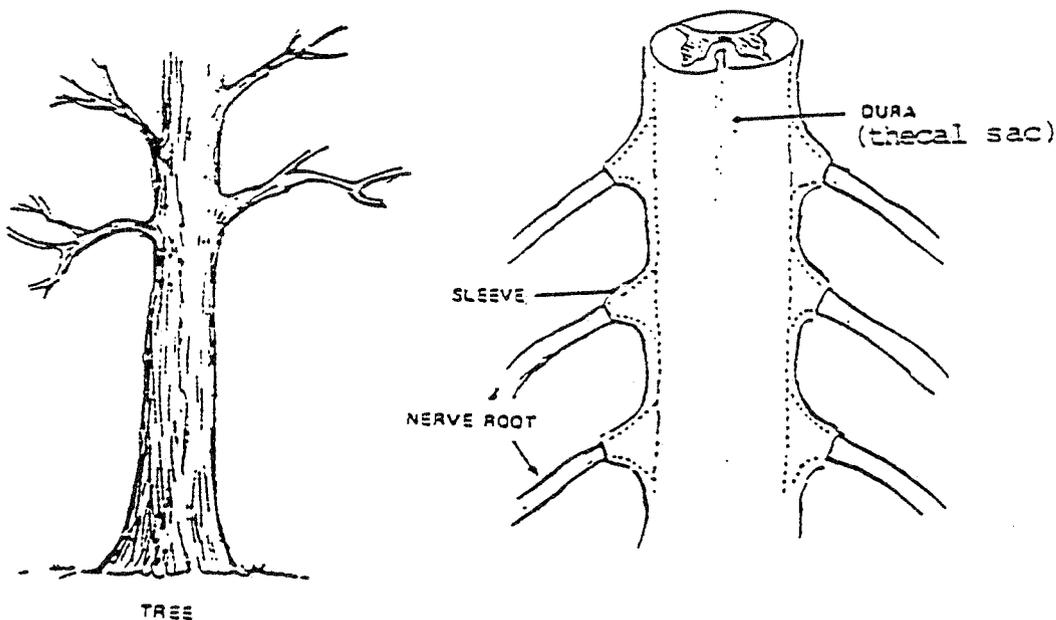
©See p.1

FIGURE 6

4. SPINAL NERVE ROOTS:

(a) Analogy To Tree Roots:

The spinal cord has roots that grow directly out of the cord like roots from a tree. These roots are called the spinal nerve roots.



©See p.1

FIGURE 7

(b) Motor and Sensory Nerve Roots:

Figure 6 above is a cross-section of the spinal cord, showing the nerve roots. Each level of the spinal cord has one pair of nerve roots with roots growing out of the right and left. Each nerve root, as it grows from the spinal cord, consists of two cables: (1) a motor root cable in the front (anterior); and (2) a sensory root cable in the back (posterior). The sensory root carries nerve impulses of sensation from the skin to the spinal cord. The motor root carries nerve impulses away from the spinal cord to a muscle, in our arms and legs. The term "motor" refers to motion - the movement of a muscle. It is analogous to the motor of a car which causes the car to move. Hence, motor nerves carry impulses which result in movement of a muscle.

These separate sensory and motor nerve roots travel less than 1/2" from their outgrowths in the spinal cord before exiting the spinal canal. Once they exit the spinal canal, through the vertebral foramen (open window), the separate sensory and motor nerve roots join together to become a spinal nerve - a mixed cable of both sensory and motor nerves. The spinal nerves innervate (supply nerves) to the muscles in the torso, arms and legs.

**PRACTICE NOTE:** According to Houts and Marmor, the separation of motor and sensory nerve roots inside the spinal canal is important to physicians performing a neurological exam. For example, if a disc compresses a nerve root in the spinal canal, the physician will find motor and sensory abnormalities if both the motor and sensory nerve roots are compressed. If only the motor root is compressed, only muscle weakness will be detected with no loss of sensation. On the other hand, if only the sensory nerve root is compressed, the physician will detect sensory abnormalities with no muscle (motor) weakness.

(c) Nerve Sleeves:

The nerve roots are 1/2" outgrowths from the spinal cord. They, like the spinal cord, are covered by the dura. The dura covering a nerve root is called the "nerve sleeve". The nerve root travels less than 1/2" from its outgrowth on the spinal cord to its exit point at the vertebral foramen, where it exits the spinal canal to become a spinal nerve. The nerve sleeves cover the nerve roots, like the sleeve of a short sleeve shirt covers the upper arm. The nerve sleeves protect the nerve roots within the bony spinal canal. (Remember, the dura is a tough, fibrous outer membrane). Figure 7 above illustrates the nerve sleeves.

**PRACTICE NOTE - THE NERVE SLEEVE PROVIDES THE ANATOMICAL BASIS FOR MYELOGRAPHIC TESTING:** Myelogram - a contrast dye is injected into the subarachnoid space of the spinal cord. If the nerve sleeve is pinched inside of the spinal canal, for example, by a herniated disc, the dye cannot fill the nerve sleeve. The outline of the nerve sleeve is absent on the myelogram x-ray. This is called a "filling defect" which suggests nerve root compression.

F. THE SPINAL CANAL:

Each vertebrae has a neural arch on its backside (Figure 2). The individual vertebrae is analogous to a domino with a hole in its back end (the neural arch), and the spinal cord is analogous to a round yellow magic marker. The spinal column is "formed" when individual vertebrae are stacked one on top of another around the

cylindrical spinal cord - like stacking individual dominoes with a hole in it around a magic marker. In most people, the vertebral neural arches form a "loose fit" around the spinal cord, like stacked horse shoes around a metal stake. A canal is formed around the spinal cord by the neural arch of each vertebrae. The spinal canal contains the neural (pertaining to nerve) elements which are the spinal cord and the spinal nerve roots, before they exit the canal. The outside column formed by stacking the bony vertebrae around the spinal cord is called the spinal column (or the spine) and is a protective bony casing for the spinal cord.

Figure 8A illustrates "formation" of the spinal canal by stacking individual vertebrae on top of each other, like horseshoes around the cylindrical spinal cord. Figure 8B shows a portion of the spinal canal housing the cord and the spinal nerve roots before they exit the vertebral foramen.

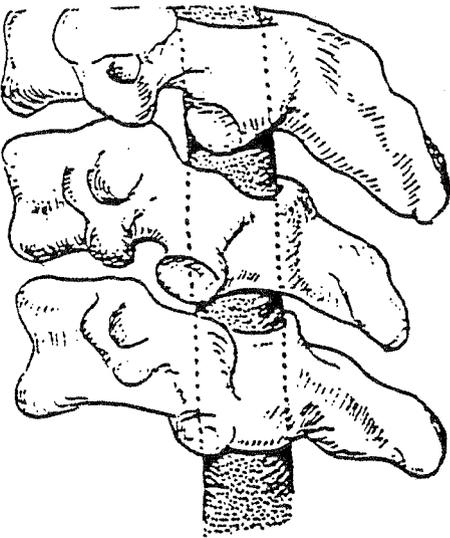


FIGURE 8A

©See p.1

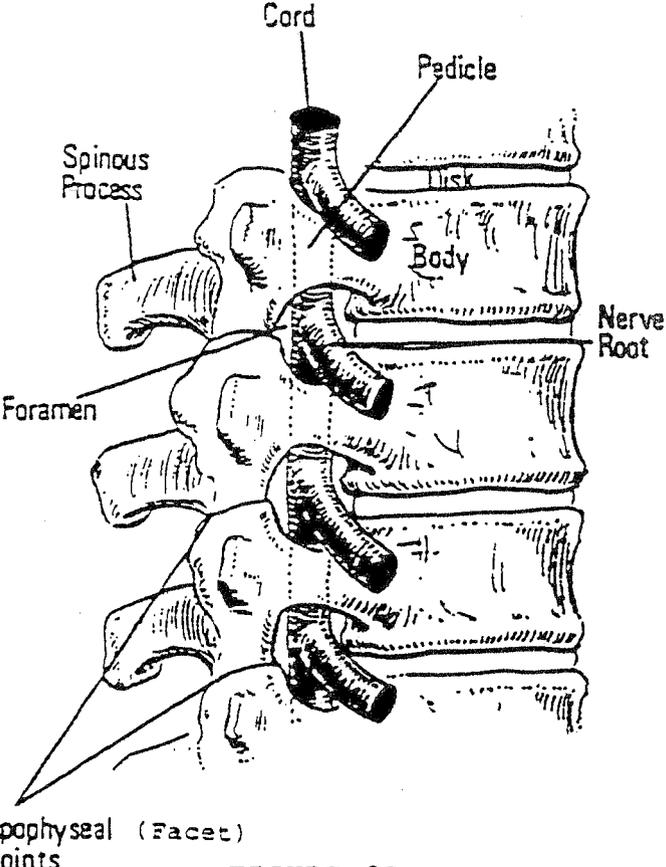


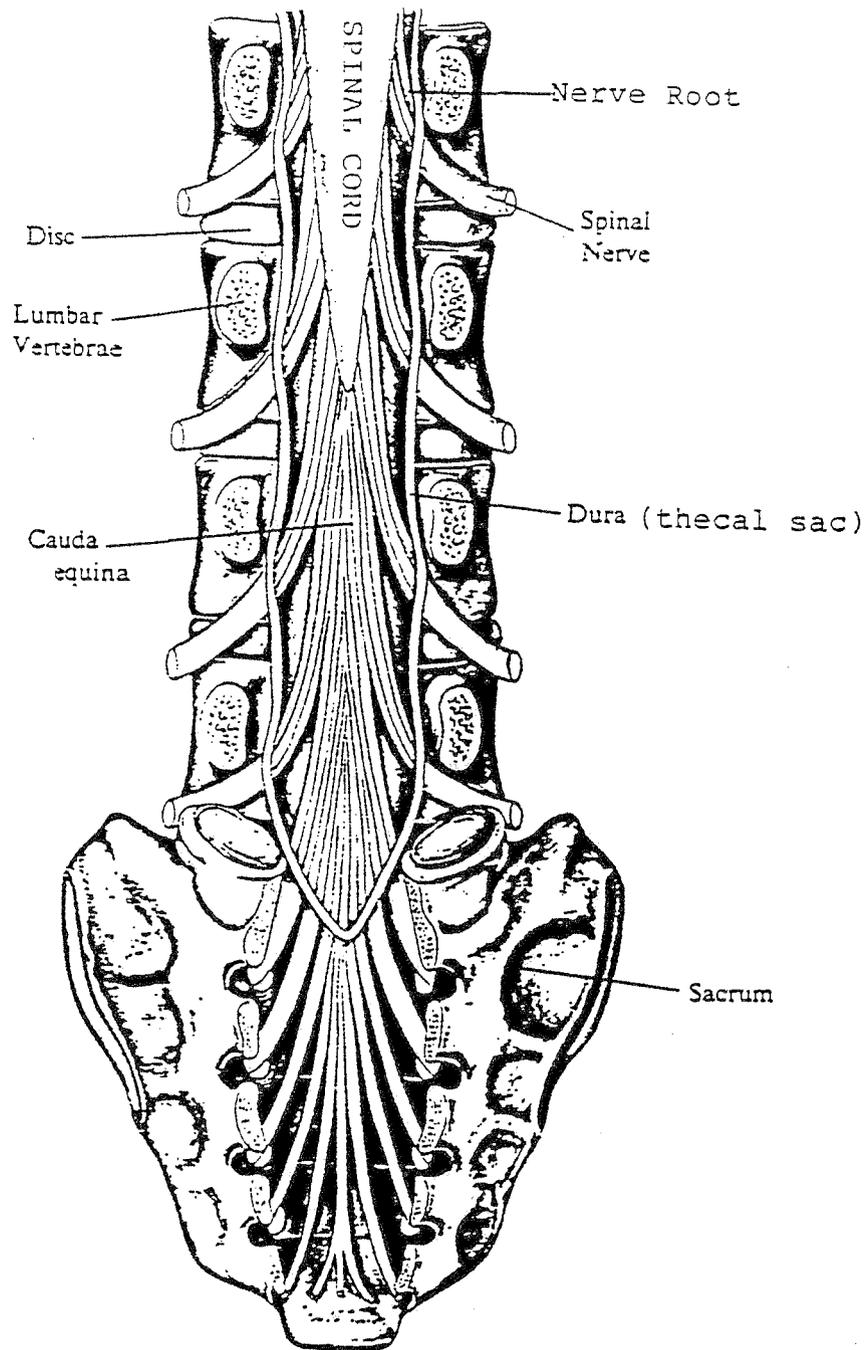
FIGURE 8B

PRACTICE NOTE: Mechanical compression and irritation of the spinal nerve root as it exits the spinal canal through the bony foramen will result in radiating pain down the course of the nerve and is called radiculopathy. This concept is the key to understanding nerve root injury.

## G. CAUDA EQUINA:

The vertebral column is longer than the spinal cord (Figure 3). The spinal cord ends at the level of the first lumbar vertebrae (L1). At this level, the spinal cord sends out nerve fibers which run down the remainder of the vertebral canal, all the way to the bottom of the sacrum. These nerve fibers look like a horse's tail when viewed from the back. Accordingly, this group of nerves is named cauda equina (horses tail). The thecal sac (outer dura membrane) of the cauda equina is firmly anchored to the bottom of the vertebral column. Figure 9 shows the cauda equina housed within a portion of the spinal column.

PRACTICE NOTE: A spinal tap and a myelogram are usually performed at a level below L1 to avoid damage to the spinal cord which ends at the L1 level in the average person. In some people, the spinal cord ends further down near the L2 level. The needle is inserted into the subarachnoid space which surrounds the cauda equina and is filled with cerebrospinal fluid.



©See p.1

FIGURE 9

1. CAUDA EQUINA SYNDROME:

Severe compression of the cauda equina results in rapidly progressing bowel, bladder and leg paralysis, requiring emergency surgery to remove the source of compression. Cauda equina syndrome (syndrome = pattern of symptoms) is extremely rare but when found, presents a medical emergency.

## H. PERIPHERAL SPINAL NERVES:

### 1. ORIGIN AND PATHWAY:

Peripheral spinal nerves are formed from the spinal nerve roots at the point where the nerve root exits the spinal canal through the vertebral foramen (open window) (Figure 8B above). The vertebral foramen is also referred to as the neural (pertaining to nerves) foramen. The spinal nerves innervate (supply nerves) to muscles in the arms and legs - in the periphery, hence the name peripheral nerves. They are also referred to as peripheral spinal nerves since they originate from the nerve roots in the spinal cord. Figure 9 illustrates the origin and the pathway of the peripheral nerves.

#### ORIGIN AND PATHWAY OF PERIPHERAL NERVES

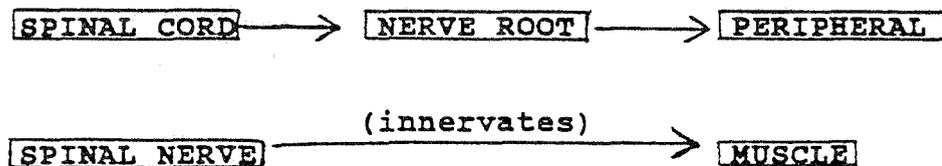


FIGURE 9A

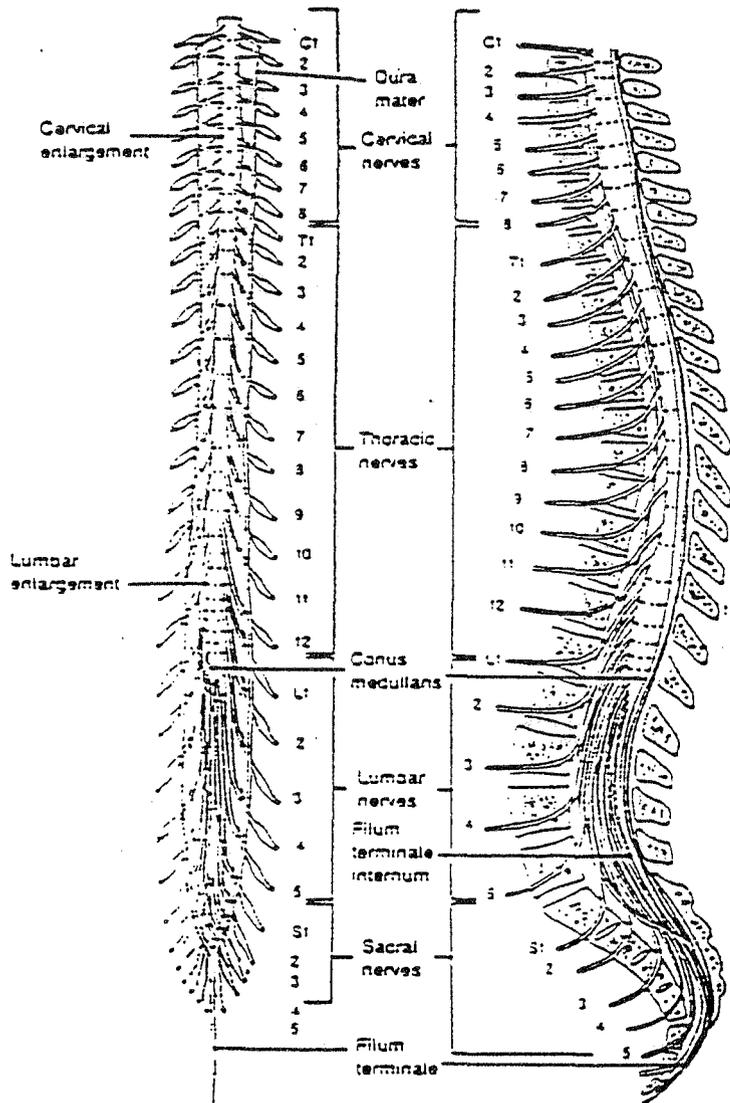
### 2. NUMBERING OF SPINAL NERVE ROOTS.

The nerve roots, which form the spinal nerves, exit the spinal canal between an upper and lower vertebrae (Figures 8A and 8B above).

Spinal nerves are numbered after their corresponding bottom vertebrae. For example, the spinal nerve root before it exits the spinal canal between the C5 - C6 vertebrae is called the C6 nerve

root. After the nerve root exits the foramen, it is called the C6 spinal nerve. There are 31 pairs of spinal nerves - one spinal nerve exits to the right innervating the right side, and the other exits to the left innervating the left side of the body. In the cervical region there are 7 vertebrae and 8 spinal nerves. The first cervical nerve goes toward the head.

Figure 10 shows the relationship of the spinal nerves to the vertebrae as they exit the spinal canal.



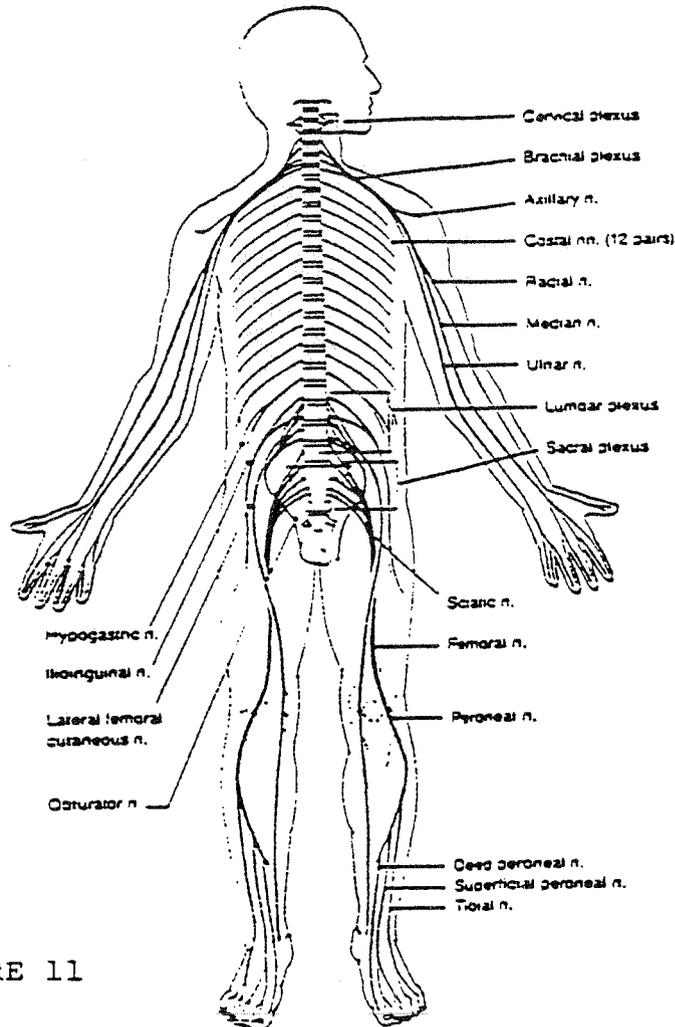
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FIGURE 10

3. NERVE PLEXUS:

Once the spinal nerves exit the vertebral foramen, they divide into nerve plexus (a network of nerves) such as the brachial (brachial = pertaining to the arm) plexus, in the neck, and the lumbar plexus, in the low back. The nerve plexus then divides into branches which innervate the muscles of the arms and legs. For example, the brachial plexus in the neck divides into the radial, median, and ulnar nerves which innervate the arm and fingers. The lumbar plexus divides into the sciatic and peroneal nerves which innervate muscles in the legs. Each spinal nerve is a mixed nerve with both sensory (sensation) and motor (motor = muscle movement) fibers, which carry impulses to and from the spinal cord, respectively.

Figure 11 diagrams the peripheral spinal nerves.

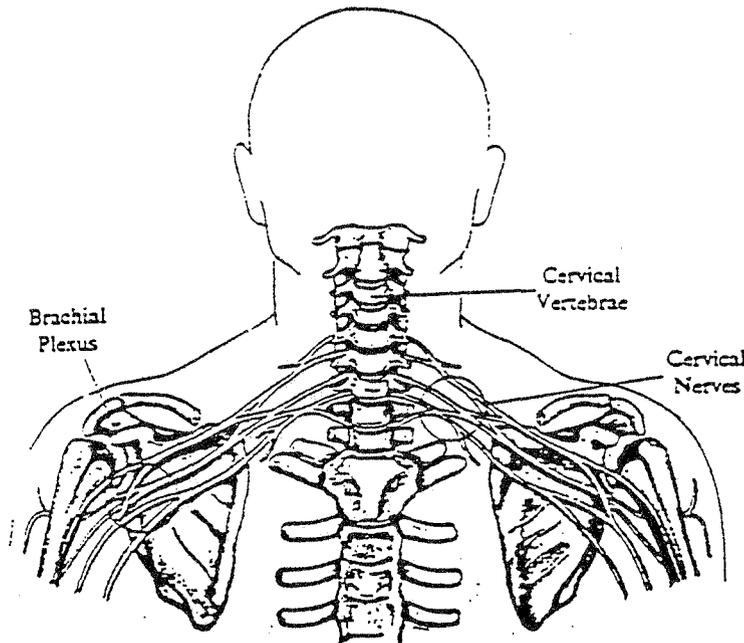


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FIGURE 11

(a) Brachial Plexus and Thoracic Outlet Syndrome:

(i) Anatomy - The brachial plexus is formed by the C5, C6, C7, C8 and T1 spinal nerves in the neck. This network of nerves (brachial plexus) leave the neck area by passing through an outlet between the first rib and the clavicle (collar bone), called the thoracic outlet, enroute to the arm. After passing through the thoracic outlet, the brachial plexus continues and divides into the radial, median and ulnar nerves, which innervate the arm and fingers. Figure 11A.



©See p.1

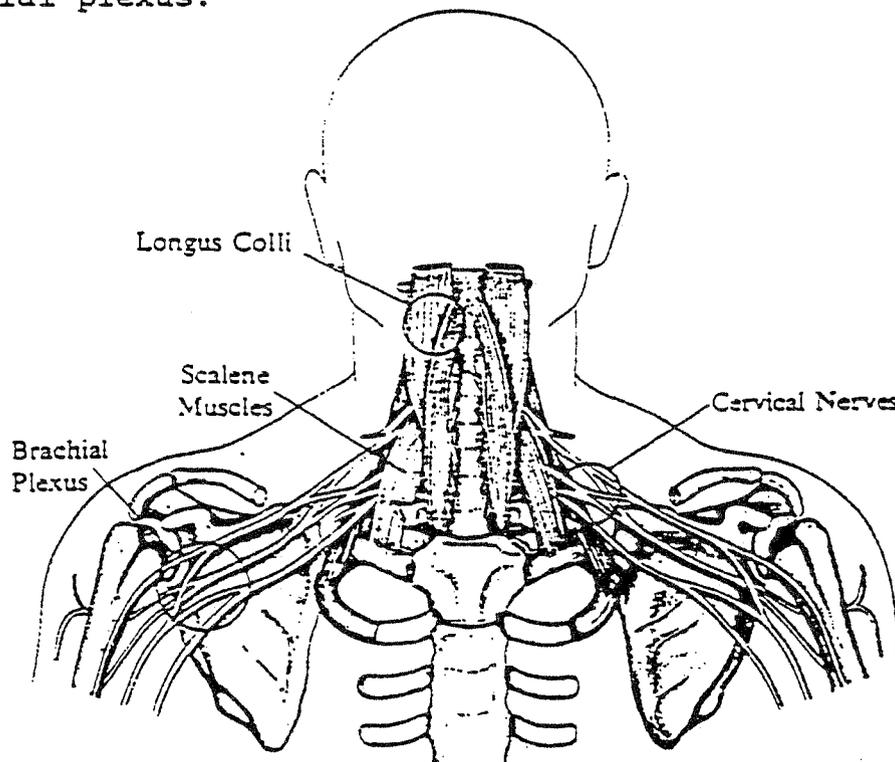
FIGURE 11A

(ii) Delayed Symptoms - If the brachial plexus is compressed while passing through the thoracic outlet, the resulting injury is called thoracic outlet syndrome (syndrome = a pattern of symptoms). Unless there is an acute (recent), direct injury to the thoracic outlet, such as a motorcycle driver being thrown to the

ground, symptoms may take weeks or months to develop. For example, if the long, thin scalene muscles which cover the cervical spine and attach to the first rib are torn (as in a cervical sprain), scar tissue formed by the injured muscle during the healing process, can compress the nerves that run between them, causing irritation of the brachial plexus.

In addition, if the collar bone or first rib are fractured, the slowly developing callus formation (overgrowth of bone at the fracture site), can also compress the nerves of the brachial plexus producing a delayed onset of symptoms.

Figure 11B shows the relationship of the scalene muscles to the brachial plexus.



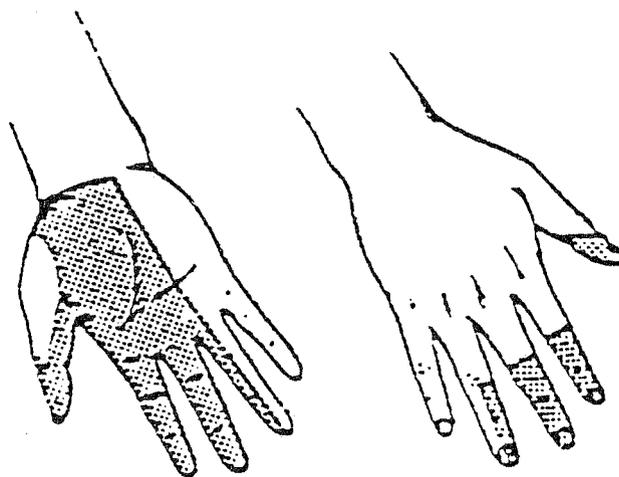
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FIGURE 11B

The thoracic outlet is in the costo-clavicular space (costo = rib; clavicular = clavical) - the space between the 1st rib and the clavicle (collar bone)

(b) Carpal Tunnel Syndrome:

(i) Anatomy - The median nerve originates from the C5-C8 nerve roots. The C5-C8 spinal nerves form part of the brachial plexus before dividing into the radial, median and ulnar nerves, which innervate the arm, hand and fingers. The median (middle) nerve is so named because it is in the middle, between the radial and ulnar nerves as they pass down the arm from the brachial plexus. (Figure 11 above). The nerve distribution of the median nerve in the hand is illustrated in Figure 11C.



©See p.1

FIGURE 11C

(ii) Compression of the Median Nerve in the Carpal

Tunnel - The median nerve travels from the arm into the hand by passing through a tunnel in the wrist called the carpal tunnel. (Analogous to a train passing through a tunnel). The top of the carpal tunnel consists of the carpal bones at the top of the wrist. The bottom of the tunnel consists of a broad ligament called the carpal ligament (ligaments hold bones

together). Localized swelling from trauma, such as by striking extended hands on the dashboard or by holding the steering wheel tightly during an auto collision, takes up space in the carpal tunnel, compressing the median nerve which is "just passing through" the tunnel, enroute to the hand and fingers.

(iii) Symptoms of Carpal Tunnel Syndrome - Symptoms resulting from carpal tunnel syndrome, such as pain, numbness, tingling and weakness, follow the nerve distribution of the median nerve into the hand and fingers, as noted in Figure 11C.

Patients with carpal tunnel syndrome often complain of being awakened at night from pain and often drop objects due to the weakness of the hand. Symptoms of carpal tunnel syndrome are often delayed in onset from weeks to months following trauma. Positive (+) EMG testing is often diagnostic for carpal tunnel syndrome; however, a patient may have a "subclinical" carpal tunnel syndrome, which is not picked-up on the EMG until a later date.

(iv) Double Crush Syndrome - Injury to Both Ends of the Median Nerve - A patient may injure both ends of the median nerve in the same auto collision. This is called "double crush syndrome" since the nerve was injured twice - once at each end. For example, he may injure the C6 nerve root in the neck (the origin of the median nerve) and at the same time sustain injury to the median nerve as it passes through the carpal tunnel, by striking his extended hands against the dashboard at the time of

impact. Since the C6 nerve root innervates the entire arm, down to the fingers, the treating physician must be careful to diagnose two separate injuries to the median nerve - at the C6 nerve root level in the neck, and at the carpal tunnel level in the wrist.

PRACTICE NOTE: During the initial interview, it is important to ask the client if he was either holding the steering wheel tightly or struck his extended hands against the dashboard at the time of impact. This initial history may later help prove that the carpal tunnel syndrome was post-traumatic, and a result of the auto collision, especially in delayed onset cases where causation, is vigorously contested.

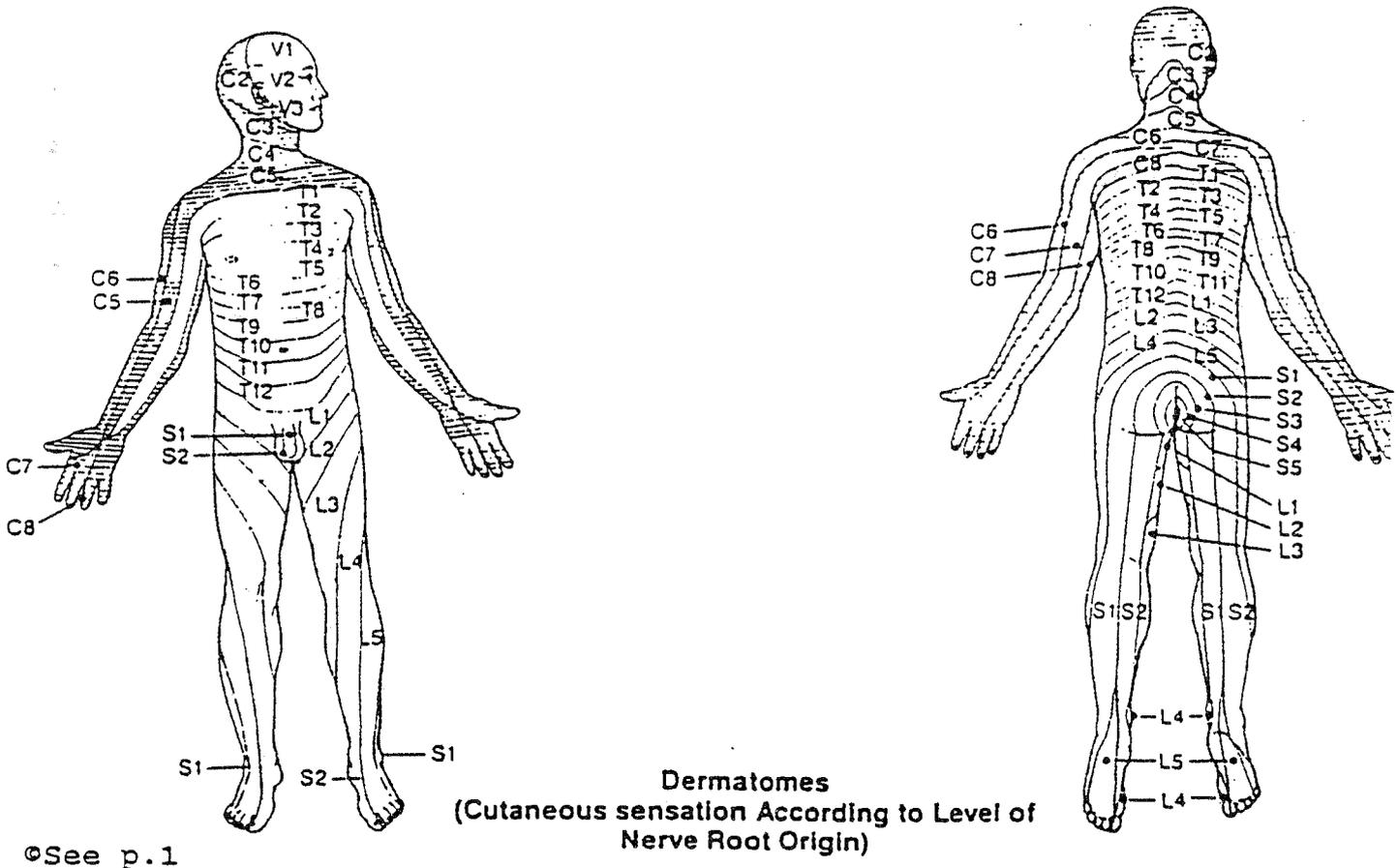
## I. CLINICAL DIAGNOSIS OF NERVE ROOT INJURY:

### 1. SEGMENTAL DIVISION OF THE SPINAL CORD:

The spinal cord is divided into segments (cervical, thoracic, lumbar and sacral). Nerve roots exit the spinal cord at specific subsegments (levels), such as the sixth cervical (C6) level; the fifth lumbar (L5) level; or the first sacral (S1) level. Each subsection of the spinal cord sends nerve impulses to a specific address - a specific muscle or muscle group called myotomes and supplies the feeling of sensation (touch, pain and temperature) to specific addresses in the skin called dermatomes. The peripheral spinal nerves are analogous to cables sending impulses along the length of the cable. Each nerve root is "programmed" to innervate a specific muscle(s) (myotome) and a specific area of the skin (dermatome). The nerve impulse from a specific subsection of the spinal cord always follows the same pathway to the same muscle and to the same area of the skin.

Therefore, an injury to a spinal nerve root produces a specific pattern of muscle weakness and a specific pattern of altered skin sensation based upon the level injured. This concept forms the basis for diagnosing the specific level of nerve root injury by examining the myotome and dermatome in the periphery (arms or legs). The nerve impulse flows "down stream". If there is a blockage high up the river, such as at the nerve root level, the effects will be felt "down stream" in the tributaries - the myotome and the dermatome.

(a) Dermatome Chart:



©See p.1

FIGURE 12

## 2. CLINICAL NEUROLOGICAL EXAMINATION:

A clinical examination refers to a physical exam performed by a physician observing the patient's responses to testing. The basis for the neurological exam consists of a muscle (motor) examination, a reflex examination, and a sensory examination. An easy way to remember these three parts of the neurological examination is to use the abbreviation "MRS" (Motor, Reflex, Sensory).

The clinical exam involving motor, reflex and sensory testing gives the physician "clues" to a specific diagnosis. Abnormal tests only indicate an injury somewhere along the course of the nerve impulse from the brain to the muscle. Abnormal test results can be caused by a tumor, by a spinal nerve root compressed by a herniated disc or a large osteophyte, or by an injury to the muscle being tested, to name a few.

Physicians often form an initial differential diagnosis (a laundry list of possible diagnoses). Further testing, hopefully, will rule out (abbrev. "r/o") each possibility on the list, resulting in a specific diagnosis.

(a) Motor Examination - Motor refers to a muscle or to a nerve whose function results in movement. For example, motor nerves innervate muscle resulting in movement. The term is analogous to a motor of a car - it is the motor that produces movement. In the human body, muscles produce movement; hence the term motor examination - refers to an examination of the peripheral muscles such as the calf, biceps, thigh muscles, etc.

Muscle testing is performed on individual muscles to test for muscle strength and weakness. The patient is asked to contract a specific muscle with all his might. The physician resists the patient's effort by applying a directly opposite force - pushing with his hands - in the opposite direction.

During physical examination, muscle strength is graded as either 5, 4, 3, 2, 1 or 0, with 5 being normal. Anything less than 5 is abnormal. For example, a notation in a medical report which states, "muscle testing of the biceps muscle was 5+ (or sometimes referred to as "++++")" indicates no muscle weakness - normal muscle strength.

The test for muscle weakness must be conducted by comparing the strength of the muscle in both the right and left extremity.

The results of muscle testing, at times, vary from examination to examination. On one day, the motor exam can show weakness, and on another day it can be normal. In addition, the time of day or the patient's activities can effect the exam. For example, the patient who has had a hard day of physical labor, who is examined in the late afternoon, may have slightly different findings than if he were examined the first thing in the morning. Therefore, it is important for muscle testing to be conducted over several examinations.

The physician, performing a motor exam, also observes and measures specific muscles for atrophy (decreases in size of the muscle due to non-use or weakness).

(b) Sensory Examination - Each spinal nerve contains motor fibers and sensory fibers - a mixed cable. Each dermatome in the skin receives sensations of pain, touch and temperature, and relays these sensations along sensory fibers backup to the specific level of the spinal cord which innervates the dermatome. By way of analogy, feelings of sensation are relayed back up-stream from the skin to the main river in the spinal cord. An injury to the spinal nerve root, in the spinal canal, will interfere with the sense of sensation - pain, touch and temperature - in the skin corresponding to the dermatome pattern of the injured spinal nerve root. Identification of the involved dermatome leads to a diagnosis of the specific level of nerve root injury in the spinal canal.

The sensation of pain is tested by pricking the patient's skin with a pin or by a pinwheel. In response, the patient will tell the doctor each time he feels a pinprick. The sensation of light touch is examined by touching the patient's skin with a cotton swab and recording the patient's response of what he feels.

The results of the sensory testing with a pinprick for pain and a cotton swab for touch are recorded as either normal, increased, decreased, altered or absent. Physicians use the following terminology:

1. **Increased sensitivity** - Hyperesthesia (hyper = increased, and esthesia from the Greek = sensation)
2. **Decreased sensitivity** - Hypesthesia (hyp from hypo = decreased, and esthesia from the Greek = sensation)
3. **Altered sensation** - Dysesthesia (dys = dysfunction, and esthesia from the Greek = sensation)

4. Absent (no sensation; numbness) - Anesthesia (an = absent, and esthesia from the Greek = sensation)

Paresthesia (para from the Greek = beyond, and esthesia from the Greek = sensation) describes a patient's complaints of abnormal sensations in the arm or leg, such as burning, tingling, and numbness. A history of paresthesia from the patient is consistent with nerve root irritation.

Like muscle testing, sensory testing is positive when there is a difference between one extremity compared to the other.

A patient who has consumed analgesics (pain relieving drugs) or alcohol may give a false negative test result since he may not feel the full extent of the pinprick because of these drugs.

- i. Stocking Glove Anesthesia - If a patient's sensory exam reveals sensory loss, such as numbness surrounding a body part as the arm, hand or leg - like a glove fitting over a hand or a stocking fitting over a leg - the patient's sensory loss does not follow the dermatome pattern and is called non-anatomic - stocking anesthesia or stocking glove anesthesia. In addition, a patient's sensory loss may be on one side of the body only. Medical terminology used to describe symptoms on only one-half of the body contain the prefix "hemi" (meaning half). For example, if a patient experienced abnormal sensations, such as numbness and tingling, only on the right side of his body, this is called "hemiparesthesia". Again, this is non-anatomic, and like stocking glove anesthesia is suggestive of a functional condition (an

emotional overlay) or conversion hysteria.

Physicians label symptoms resulting from an emotional (psychological) response as "functional" and symptoms resulting from actual physical injury, such as compression of a nerve root, as "organic" or "somatic" (soma from the Greek = body as distinguished from the mind).

Conversion hysteria (also called post-traumatic neurosis) is an involuntary response by the patient, which is often caused by trauma. The patient's psychological response to the trauma is so overwhelming that it cannot be controlled and the patient's mind which "converts" emotional symptoms to physical symptoms which mimic the symptoms of nerve root irritation, i.e., pain radiating down an extremity, sensory loss, muscle weakness. However, as noted, these symptoms do not follow the well recognized dermatome and myotome patterns and are termed non-anatomic.

**PRACTICE NOTE:** The terms "stocking glove anesthesia" and "non-anatomic pattern" are red flags for conversion hysteria or for an emotional overlay - the patient's emotional symptoms overlay and superimpose physical pain.

(c) **Reflex Testing** - Reflex testing is another procedure which helps identify the specific level of nerve root damage in the spinal cord. The test is performed by the physician striking a deep tendon with his reflex hammer (tendons attach bone to muscle).

Striking the tendon causes the muscle to stretch and contract. For example, if the nerve root which supplies motor nerves to the muscle involved in the test is injured, the reflex will be abnormal. An abnormal reflex pinpoints the location in the spinal cord of the nerve root injury. To be meaningful, the reflex should be absent in only one extremity, not both. (However, a very large central disc herniation, in appropriate circumstances, could possibly compress the nerve roots on the right and left sides, causing neurological symptoms down both legs which result in abnormal reflexes in both extremities - this is the exception, not the rule.)

Reflexes are graded as either 1+, 2+, 3+ or 4+. Sometimes, only plus signs are used. For example, 2+ would be ++.

**PRACTICE NOTE:** Defense examiners often try to detect malingering (faking) by striking bone and not the tendon. Striking a bone would give no response since only striking the appropriate tendon causes the muscle to contract. Therefore, if the patient contracts the muscle when only the bone is tapped with the reflex hammer, this is an inappropriate non-anatomic response suggesting malingering or possibly hysteria.

Hyperactive reflexes are suggestive of damage to the descending long tracts (pyramidal tract) in the spinal cord (myelopathy). The long tracts inside the spinal cord carry impulses from the brain down the spinal cord via an upper motor neuron to the spinal nerves.

Deep tendon reflex testing, such as the ankle jerk and the knee jerk tests, result in a finding of clonus in patients with long tract damage. The reflex arc is involuntarily repeated many

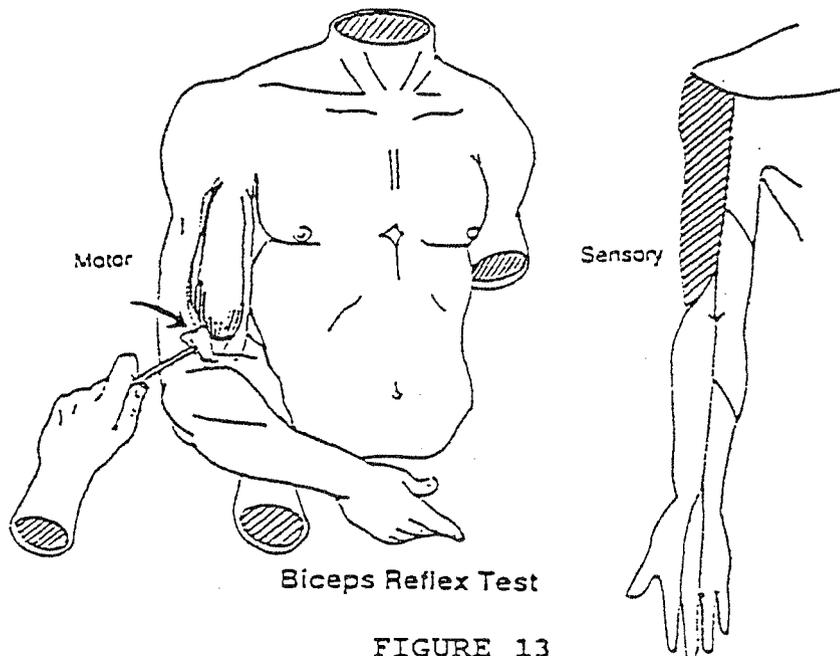
times producing jerking of the extremity at a rapid rate. Hyperactive reflexes are graded ++++ (4+).

5. EXAMPLES INVOLVING SPECIFIC NERVE ROOT LEVELS:

(a) The Fifth Cervical Nerve Root (C-5):

The fifth cervical nerve root (C5) innervates the biceps muscle and supplies sensation to the lateral side of the upper arm. Nerve root compression of the C5 nerve root, for example, by a herniated disc or a large osteophyte, will produce weakness in the biceps muscle (found during motor examination) and altered sensation such as numbness and decreased perception of pain (sensory loss found during sensory examination) in the lateral side of the upper arm. The biceps reflex, which measures the nerve impulse from the C5 nerve root to the biceps muscle, will be abnormal if the C5 nerve root were damaged.

Figure 13 illustrates the biceps reflex test and the area of diminished sensation (dermatome).



(b) The Fourth Lumbar Nerve Root (L4):

Everyone is familiar with the knee-jerk reflex where the physician strikes the patella (kneecap) tendon with a reflex hammer as the patient sits with his/her legs dangling over the examining table. As the physician strikes the patella tendon, the quadriceps muscle (the large muscle in the front of the thigh which straightens the knee) contracts, causing the leg to jerk upwards. This is a negative test. The muscle contracted properly indicating no deficiency with the nerve.

The L2, L3 and L4 nerve roots form the femoral nerve which runs down the front of the thigh, and innervates to the thigh muscle (quadriceps).

Although the patella Tendon reflex is innervated by the L2, L3 or L4 nerve roots, the L4 nerve root predominates. Accordingly, the patella reflex is clinically considered a test for the L4 nerve root. If the leg fails to jerk upward when tested, the test is positive (+) - a clinical indication of L4 nerve root compression.

Figure 14 shows the Patella Tendon Reflex Test.

Patella Reflex Test

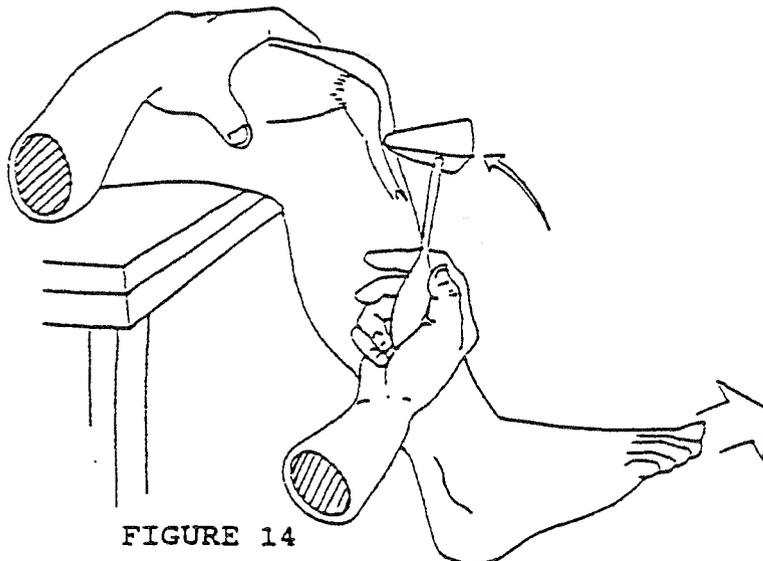


FIGURE 14

©See p.1

(c) The Fifth Lumbar Nerve Root (L5):

The L5 nerve root innervates the long sciatic nerve which runs down the back of the leg. The sciatic nerve "stretch" test (straight leg raising test) helps diagnose nerve root compression and is discussed in detail in the section labeled "Sciatica".

The L5 nerve root innervates a specific myotome - the anterior tibial muscle which raises the foot at the ankle, dorsiflexion (dorsi = dorsum - back of the foot; flexion = bending toes upward - hence backward bending of the foot at the heel). In addition, this muscle also controls the extensor digitorum longus muscle, which extends the toes upward.

Muscle strength of the L5 myotome is tested by having the patient walk on his heels then on his toes.

Figure 15 illustrates L4-L5 disc protrusion compressing the L5 nerve root resulting in ankle dorsiflexion weakness and toe extension weakness (the myotome innervated by the L5 nerve root).

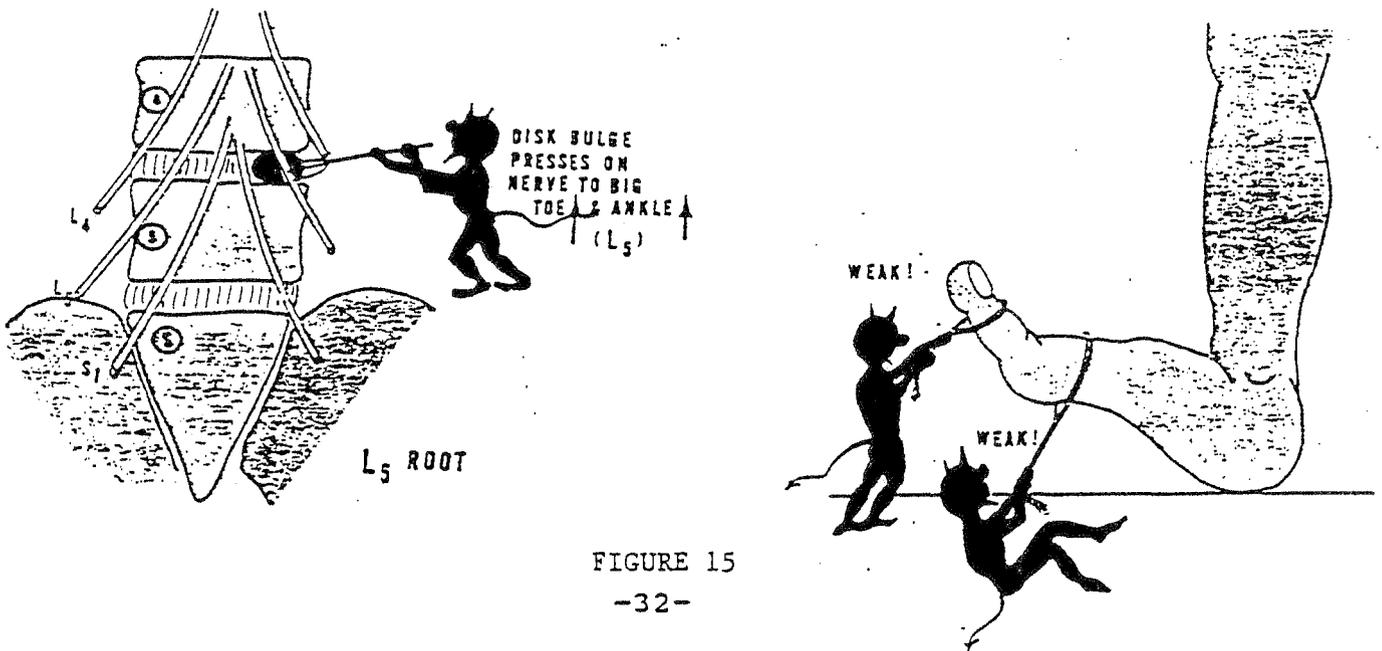


FIGURE 15

(d) The First Sacral Level (S-1):

The first two sacral levels (S1 and S2), innervate the calf muscle (gastrocnemius) and the soleus muscle beneath the calf (myotome) with S1 being most dominant. The S1 nerve root supplies sensation to the skin at the lateral side of the foot (dermatome). Damage to the S1 nerve root, for example, by a herniated disc or a large osteophyte compressing the nerve root, causes weakness in the calf and soleus muscles, which would be observed during motor testing of these muscles.

Sensory testing to the lateral side of the foot would reveal altered sensation, i.e., numbness and decreased feeling of pain.

Reflex testing which measures the nerve impulse from the nerve root to the calf and soleus muscles would be abnormal if the S1 nerve root were damaged. The Achilles tendon (named after the Greek hero Achilles) attaches the calf and soleus muscles to the ankle. Striking the Achilles tendon with a reflex hammer causes the calf muscle and the soleus muscle to involuntarily contract in a downward motion (plantar flexion of the foot - plantar = bottom of the foot; flexion = bending downward). An abnormal Achilles tendon reflex test is consistent with nerve root compression of the S1 root.

Figure 16A illustrates the Achilles tendon reflex test. Figure 16B shows S1 and S2 nerve roots innervating the gastrocnemius and the soleus muscle which is responsible for plantar flexion (bending the foot downward).

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Achilles Tendon Reflex Test

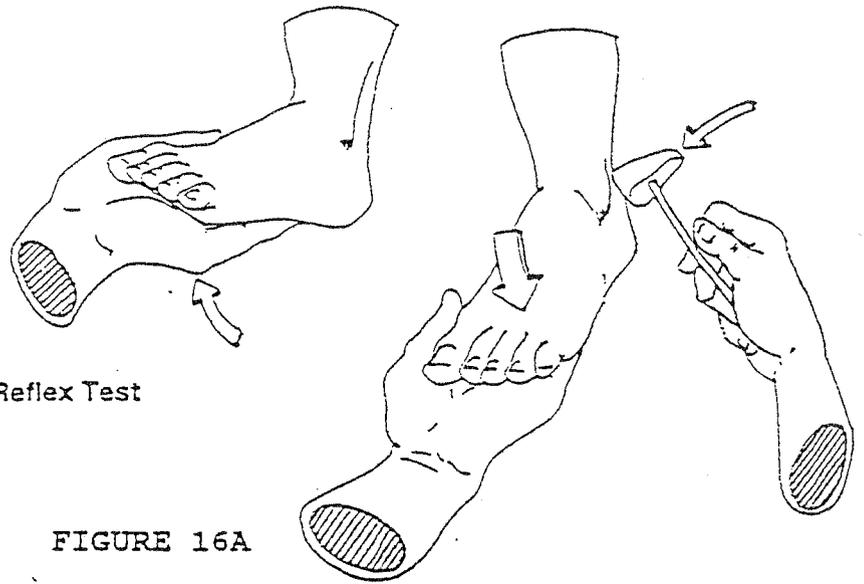
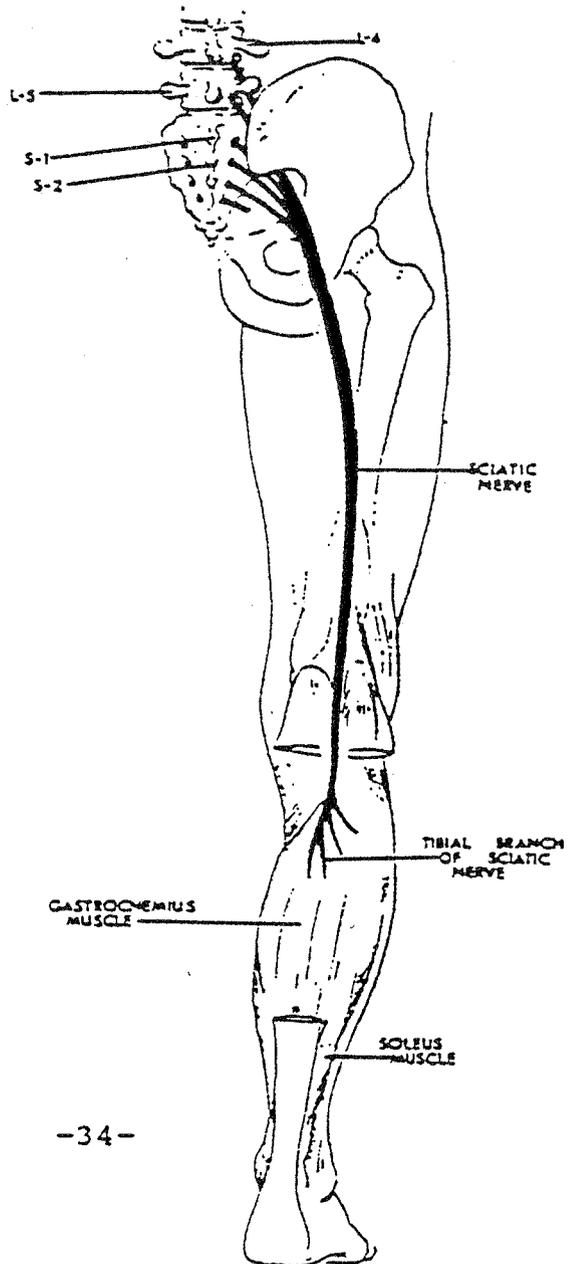


FIGURE 16A

FIGURE 16B



©See p.1

(e) Dermatomes of the Lumbosacral Nerve Roots:

Each nerve root innervates a specific area of skin (dermatome) with sensory nerves responsible for the perception of touch, pain and temperature.

Figure 17 shows the dermatomes of the lumbar and sacral nerve roots.

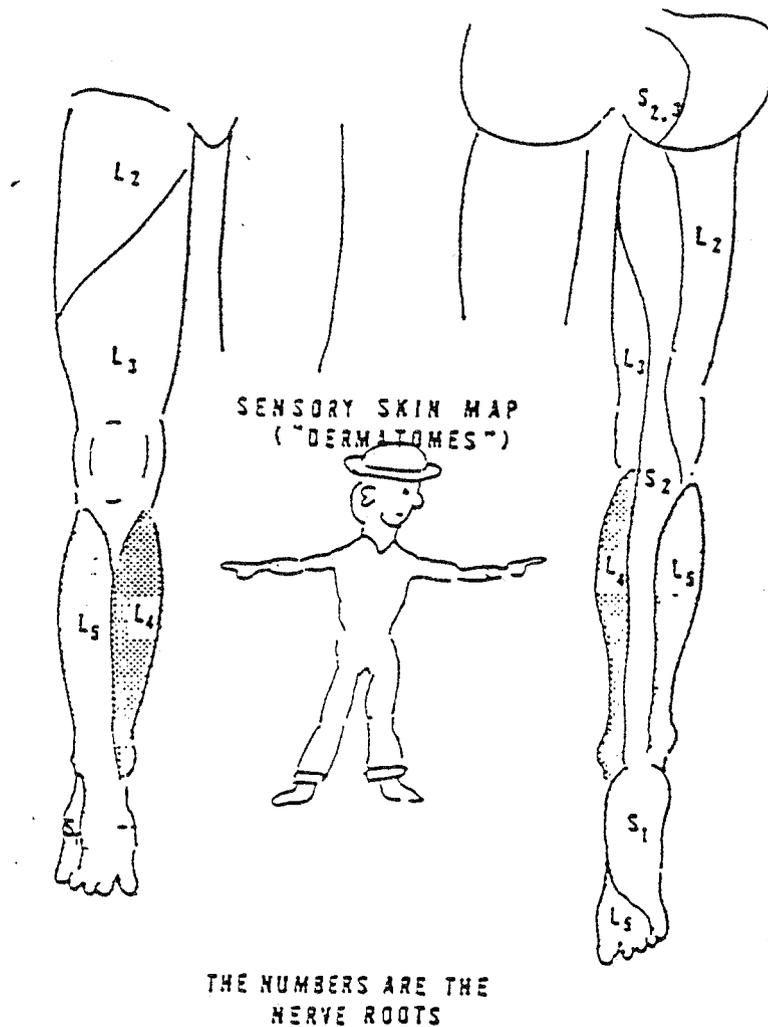


FIGURE 17

©See p.1

MEMORY AID: A good way to associate nerve root levels with the corresponding reflex test and myotome is as follows:

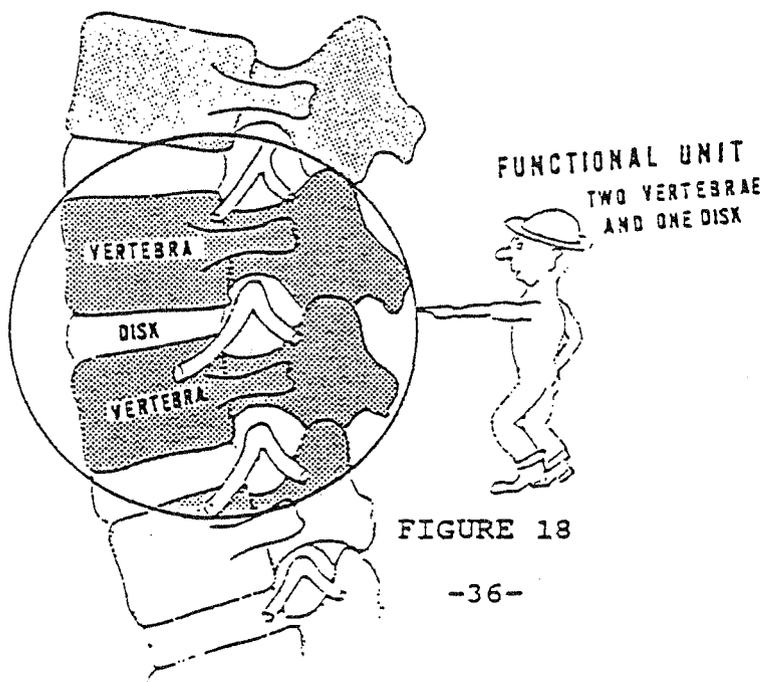
Nerve Root  
Level

Muscle/Reflex Test

C5	.....innervates.....	Biceps (to make a muscle, all <u>5</u> fingers form a fist)
L4	.....innervates.....	Knee Jerk Reflex (Quadri- ceps Muscle) <u>4</u> letters in "knee"
L5	.....innervates.....	Toes ( <u>5</u> toes)
S1	.....innervates.....	Archilles Tendon Reflex (calf) Archille <u>s</u> 1

**J. THE FUNCTIONAL UNIT OF THE SPINE.**

The functional unit of the spine consists of two vertebral bodies (bone) between a disc. Since the disc is between two vertebrae, it is also called an intervertebral disc. This functional unit can be considered a "vertebral-body sandwich" with the disc in the middle, analogous to a slice of bologna. The disc consists of mainly cartilage and acts as a shock-absorber between the bony vertebrae.



©See p.1

## II. ANATOMY OF AN INTERVERTEBRAL DISC

### A. NUMBERING:

The disc is numbered two ways; (1) to correspond to the top vertebrae in the "vertebral body sandwich"; and (2) to designate both the vertebrae in the sandwich. For example, the disc between the fourth vertebral body and the fifth vertebral body is called the L4 disc (corresponding to the top vertebrae) and also the L4 - L5 disc to designate the top and bottom discs in the sandwich.

There is no disc between the first and second cervical vertebrae nor is there a disc in the sacral region (tailbone) since all the vertebrae in the sacrum are fused together as one bone.

Many times, physicians refer to the low back as the lumbosacral spine (the vertebrae and discs of the lumbar and sacral region). The disc between the last lumbar vertebrae (L5) and the sacrum is referred to as the L5-S1 disc or the lumbosacral joint.

Figure 19 illustrates the "vertebral body sandwich, with the intervertebral disc in the middle.

**NOTE:** The most frequent level for a disc herniation in the neck (cervical spine) is at C5-C6 and C6-C7 and in the low back at the L4-L5 and the L5-S1 disc. These levels have the greatest load exerted upon them and exert the greatest motion. Therefore, there is a greater potential for herniation and osteoarthritis formation at these levels.

#### SUPERIOR VIEW

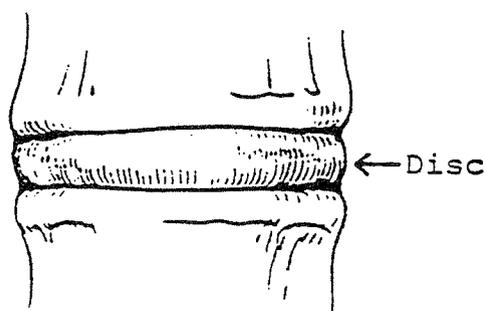


FIGURE 19

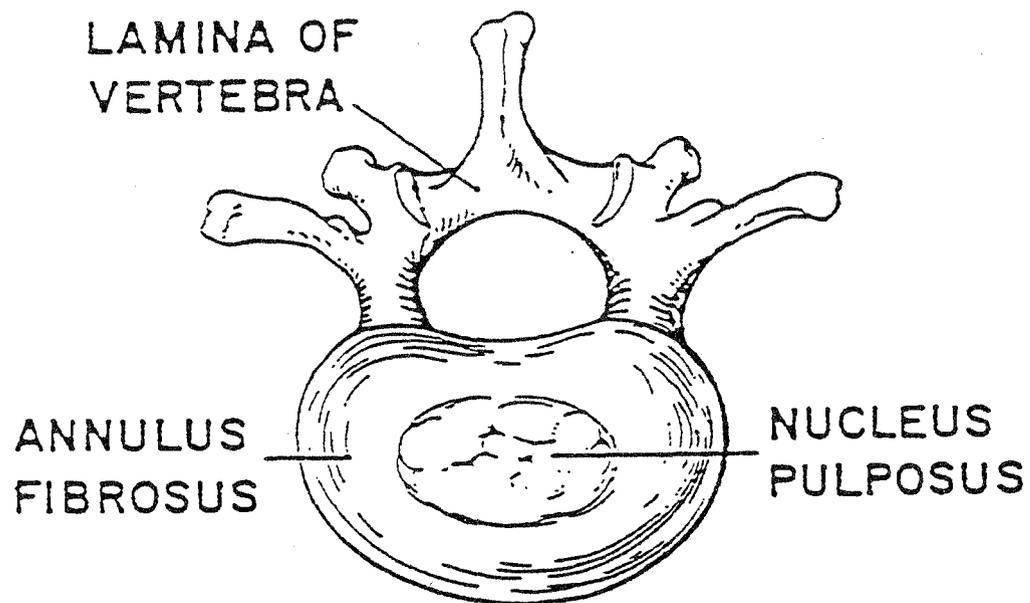
©See p.1

#### ANTERIOR VIEW

### III. ANNULUS FIBROSUS (OUTSIDE FIBROUS RING OF A DISC)

The outside of the disc is called the annulus fibrosus (annulus = ring, fibrosus = fibrous) and is merely a ring of fibrocartilage, mostly fibrous protein. The fibers of the annulus fibrosus are arranged in concentric layers. The main function of this outer ring is to contain the jelly-like material inside the disc.

The fibers of the annulus fibrosus are stronger in the front (anterior) than in the back (posterior). Accordingly, since the inside of the disc is under pressure, the nucleus pulposus (the jelly-like material inside) tends to herniate at the weakest point in the outer fibrous ring - posteriorly.



©See p.1

FIGURE 20

Damage to the annulus fibrosus (the outer ring of the disc) may cause rupture of the annulus fibrosus resulting in disc herniation by allowing the jelly-like center of the disc, which is under pressure, to push out the confines of its annular outside ring. In many cases, the patient sustains injury to the outside annular ring without immediate disc herniation. Over time, with normal wear and tear, the annulus fibrosus will deteriorate and lose its capacity to hold the jelly-like material inside the disc resulting in disc rupture (herniation) at a later date.

**PRACTICE NOTE:** To explain causation to the jury, an analogy to a tire may be used. The outside rim of a tire (equivalent to the annulus fibrosus) hits the curb causing unnoticeable damage to the outside of the tire. With normal road use of the tire, several weeks later, the tire will blow and the inside air will escape through the damaged outer ring, causing the tire to flatten. Similarly, initial damage to the annulus fibrosus, with time, can result in a deterioration of this outer ring. The inside jelly-like material which, like the tire is also under pressure, now breaks through the fibers of the annulus fibrosus causing disc herniation.

#### **IV. NUCLEUS PULPOSUS** **(JELLY-LIKE CENTER OF DISC)**

The inside center of the disc is called the nucleus pulposus which consists of collagen fibers meshed in a jelly-like substance with a high water content. The inside of the disc is semi-gelatinous. As we get older, the percentage of water in the center of the disc decreases and the disc is said to "dry out" which reduces the height of the disc space. The disc space is the space between the

upper and lower vertebrae of the "disc sandwich" and is occupied by the disc. As the disc loses water, the size of the disc decreases, causing the disc space to also narrow. The function of the disc is to act as a shock absorber. Loss of water decreases the ability of the nucleus pulposus to perform this function. The "drying out" of the inside of the disc, with advancing age, is the beginning stage of degenerative disc disease with the resulting formation of osteoarthritis, (bony overgrowths (spurs) on the vertebral bodies).

An intervertebral disc is like a jelly doughnut. The inside of the disc is similar to the inside of the doughnut. The annulus fibrosus, the circular (annular) ring which holds the jelly inside the disc, is similar to the outside tough layer of the jelly doughnut which holds the jelly inside. Rupture of the annulus fibrosus results in the inside jelly-like nucleus pulposus extruding out of the disc - just like the rupture of the outside layer of the jelly doughnut - with the inside jelly extruding out of the doughnut.

## V. BULGING, PROTRUDED AND HERNIATED DISCS

Since the jelly-like nucleus pulposus inside the center of the disc is under pressure, it will bulge out or protrude at the weakest spot in its outer fibrous ring, the annulus fibrosus.

A disc is herniated (ruptured) if the jelly-like nucleus pulposus breaks through fibers of the annulus fibrosus. The fibers of the annulus fibrosus have thus ruptured, allowing the semi-gelatinous nucleus pulposus out, like the jelly inside a doughnut

rupturing through the outside layer. A herniated disc is often called an extruded disc because, like a tube of toothpaste, the jelly is extruded outside its tube; hence the name extruded disc. A herniated disc is also referred to as a "prolapsed disc". (Prolapsed meaning out of position). Lastly, a herniated disc is called a "slipped disc" since the disc is said to slip out of place.

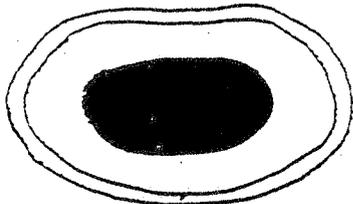
When a piece of the jelly-like nucleus pulposus, which has ruptured through the outer annulus fibrosus ring, breaks-off into the spinal canal, disc material is said to have sequestered. This type of disc is called a sequestered disc.

If the jelly-like nucleus pulposus does not break through the annulus fibrosus, the disc has not herniated. However, the jelly inside of the disc may bulge or protrude out at the weakest spot of the annulus fibrosus. Some physicians use these terms interchangeably. However, generally, a protruded disc is one where the nucleus pulposus not only bulges out, but actually pinches a nerve root. A bulging disc and protruded disc differ from a herniated disc in that the former have not ruptured through the outer annular ring; the inner jelly material of the nucleus pulposus is still contained within the annulus fibrosus even though it bulges out.

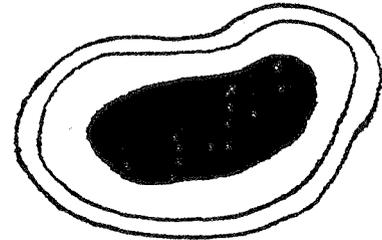
**PRACTICE NOTE:** A herniated disc is often abbreviated in medical records as "HNP" which stands for herniated nucleus pulposus since it is the nucleus pulposus which herniates through its outer annulus fibrosus ring.

## VI. PATHOLOGICAL CHANGES IN THE NUCLEUS PULPOSUS

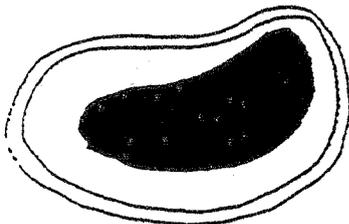
The various pathological changes in the jelly-like nucleus pulposus adapted from Weisel, et al, Low Back Pain, 2d Ed., p.89, are diagrammed below.



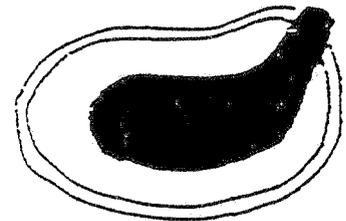
NORMAL



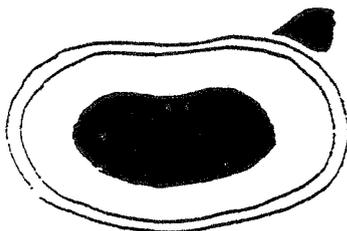
BULGING  
(bulges out but does not pinch nerve root)



PROTRUDED  
(protrudes out - pinching the nerve root, but does not break through the outer annular ring)



1. HERNIATED
2. EXTRUDED
3. PROLAPSED
4. SLIPPED



SEQUESTERED  
(breaks off into the spinal canal)

FIGURE 21

## VII. SITES OF DISC HERNIATION:

A disc may herniate in the front (anterior) or in the back (posterior). In addition, the disc may herniate at the midline (central disc herniation) or to the side (lateral disc herniation). The term lateral is used in medicine to refer to "the side" as opposed to the midline. An analogy can be made to a football pass - a pass to the side is a lateral pass.

The anatomy of the annulus fibrosus and the anterior and posterior longitudinal ligaments generally determine the site of disc herniation, i.e., anterior, posterior lateral, central.

### A. ANATOMY OF THE ANTERIOR AND POSTERIOR LONGITUDINAL LIGAMENTS:

The anterior and posterior longitudinal ligaments run up and down the spinal column covering the front and back of the vertebrae and discs, respectively. These long ligaments are fixed to the vertebrae and discs and hold them in place. The anterior longitudinal ligament covers the front end of the annulus fibrosus and the posterior longitudinal ligament covers the back.

For a disc to herniate, the inside jelly of the nucleus pulposus must break through two barriers: (1) the outer annular ring of the disc; and (2) the longitudinal ligament covering the outside of the disc.

Most discs herniate posteriorly (in the back) not anteriorly (front side). The reason for this is twofold: (1) the annulus

fibrosus is anatomically weaker in the back than in the front; and (2) the anterior longitudinal ligament is anatomically broader and stronger than the narrower and weaker posterior longitudinal ligament. Since the disc is under constant pressure, like the inside of a pressure cooker, inside disc material (nucleus pulposus) will herniate at the weakest point of its outer ring, and at the weakest point of the ligament covering its outer ring - posteriorly. The thinner and weaker ligament, the posterior longitudinal ligament, is rhomboid in shape. Therefore, the weakest spot in the posterior longitudinal ligament is not in the center, but to the side (laterally). The majority of disc herniations are found in the posterior lateral position. (In the back of the disc to the right or left side).

Figures 22A, 22B, 22C and 22D show the anterior and posterior longitudinal ligaments holding the vertebrae and discs in place. Figure 22D shows the rhomboid shape of the posterior longitudinal ligament and the dot (●) illustrates the "weak spots", posteriorly, where disc herniations are most likely to occur.

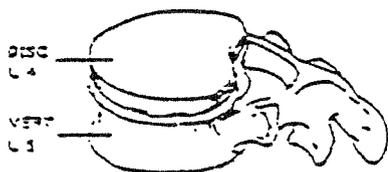


FIGURE 22A

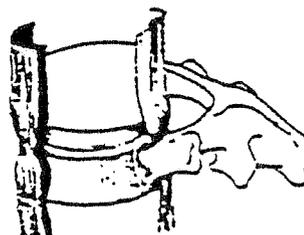


FIGURE 22B

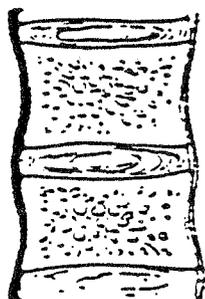


FIGURE 22C

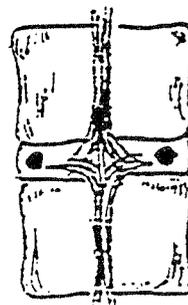


FIGURE 22D

©See p.1

**B. ILLUSTRATIONS OF DISC HERNIATIONS:**

Figure 23A shows a cross-section of a normal disc; Figure 23B shows central disc herniation; and Figure 23C shows posterior lateral disc herniation (left sided).

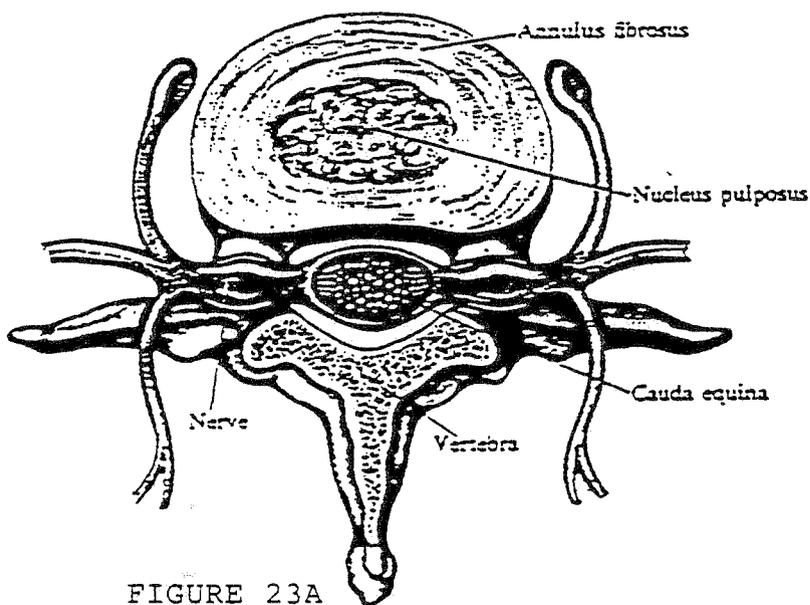


FIGURE 23A

Disc—Cross Section

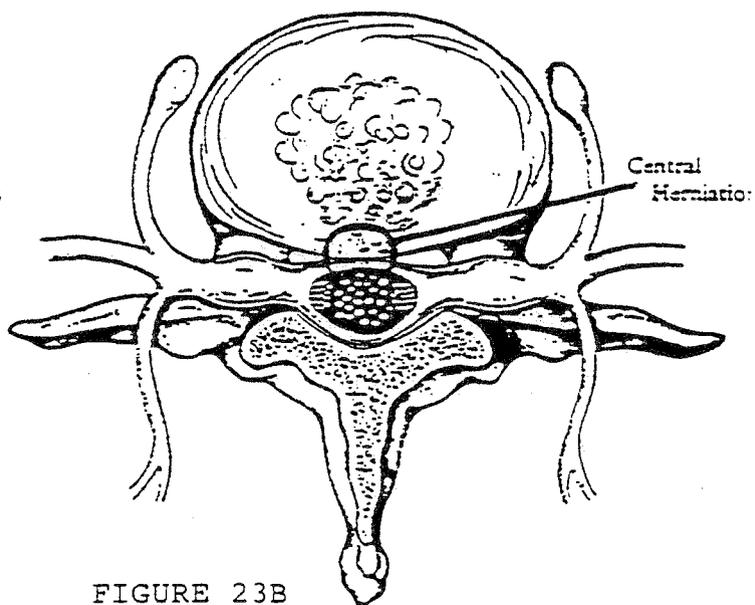


FIGURE 23B

Central Herniation

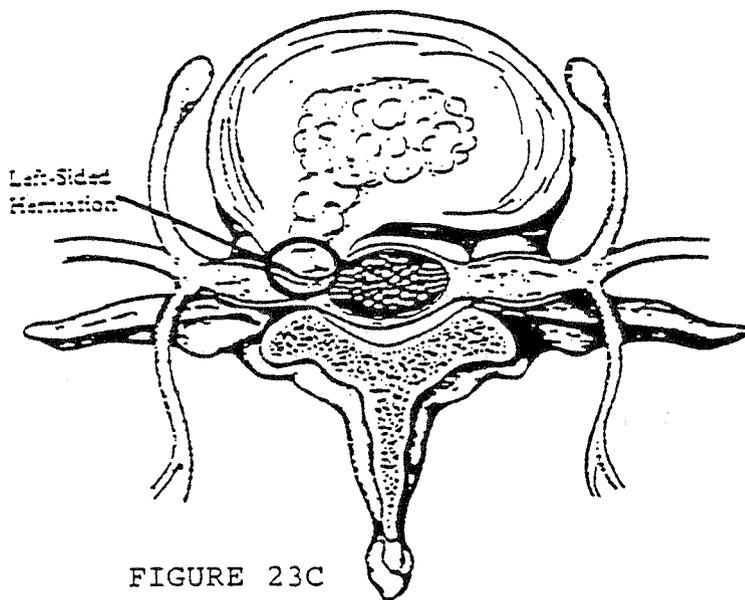


FIGURE 23C

Left-Sided Herniation

©See p.1

## C. DISC HERNIATION AND SYMPTOMS:

### 1. RADICULAR SYMPTOMS RESULT FROM COMPRESSION OF THE NERVE ROOT:

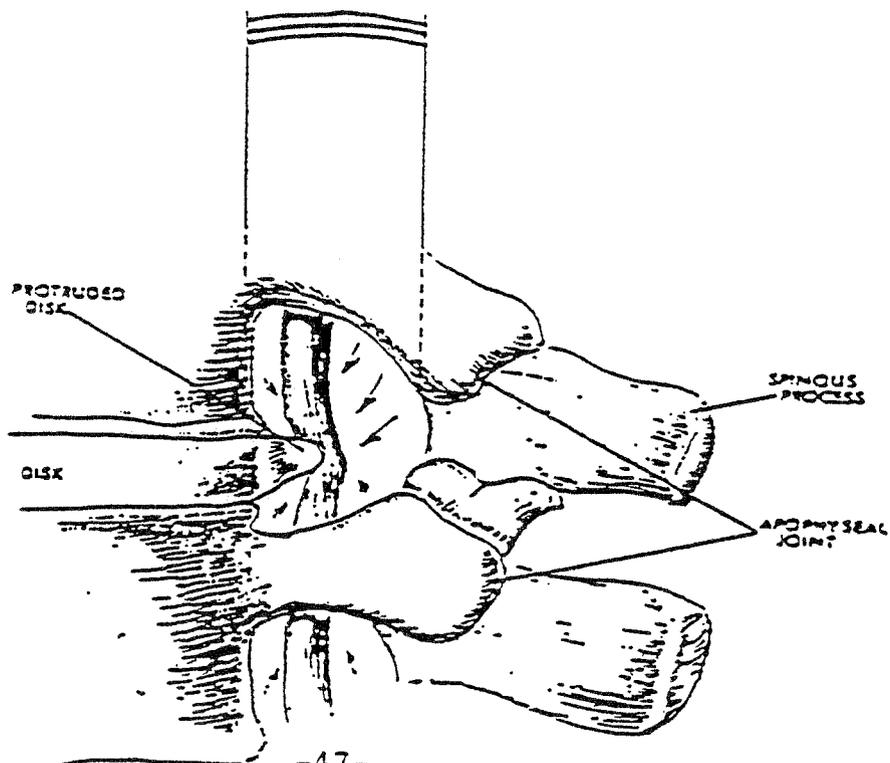
The symptoms of disc herniation vary depending upon the location and size of the disc herniation. Radicular (radiating pain) develops only when the nerve root is compressed by the herniated disc. Nerve roots grow out of the spinal cord and extend laterally to the right and left in the spinal canal before exiting on each side forming the right and left sided spinal nerves, which innervate the right and left arms and legs. Disc herniation at the midline, called central herniation, unless it is very large, will not generally compress the nerve roots to the left or to the right. Accordingly, a patient with a small central disc herniation generally will have only local symptoms at the level of the herniation, such as in the neck or back, without pain radiating down the course of the spinal nerve. A very large central disc herniation could compress both the right and left nerve roots, resulting in bilateral radiating pain on the right and left sides down the course of the spinal nerve. In this rare instance, motor, sensory and reflex examinations will be positive for both extremities. Lateral disc herniation, as shown in Figure 23C will result in compression of the nerve root on the side (pictured left side) with resulting pain radiating down the course of the peripheral nerve into the left extremity.

## 2. DIAMETER OF THE SPINAL CANAL:

Depending upon the size of the patient's spinal canal, a small posterior lateral disc herniation may not protrude far enough into the spinal canal to compress the nerve root. These patients may have no symptoms since the nerve root is not compressed. The diameter of the spinal canal (which houses the spinal cord) varies from individual to individual. A person with a small spinal canal ("not much room") is more likely to develop symptoms from a small disc herniation than a person with a large spinal canal since a person with a large spinal canal has "excess room" in the canal for a space-occupying lesion such as a herniated disc. The spinal canal narrows with age, and this condition is called spinal stenosis discussed later under the heading "Spinal Stenosis".

Figure 24 shows a portion of the spinal canal and illustrates the principle that a bigger disc protrusion or herniation is required to compress a nerve root in a person with a large spinal canal.

FIGURE 24



©See p.1

3. PATIENTS WITH ASYMPTOMATIC DISC HERNIATION MAY DEVELOP SYMPTOMS IN LATER YEARS:

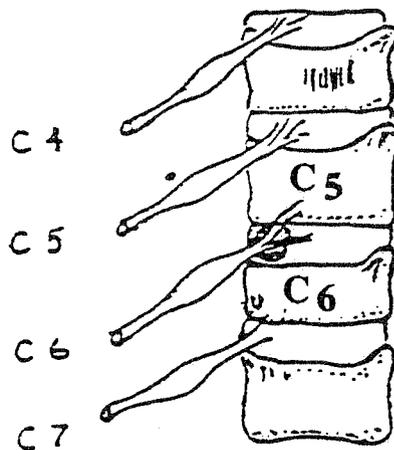
As discussed, disc herniation may not result in radicular symptoms unless the nerve root is compressed.

In later years, a patient with an asymptomatic disc herniation may develop radiating pain if the herniated disc material becomes larger and/or the diameter of the spinal canal narrows with resulting nerve root compression.

D. DISC HERNIATION COMPRESSES THE LOWER ROOT:

The nerve roots exit the spinal column at 45 degree angles and travel downward toward the extremities as the spinal nerves.

A disc herniation (slipped disc) will result in compression of the lower nerve root passing downward across the disc. For example, herniation of the disc between C5-C6 will result in compression of the C6 nerve root. Likewise, herniation of the L4-L5 disc will result in nerve root compression of the L5 nerve root.



©See p.1

FIGURE 24A

## VIII. RADICULOPATHY AND RADICULITIS

Whenever a protruded or herniated disc compresses (pinches) a nerve root, pain and paresthesia (abnormal sensations such as tingling or numbness) are felt by the patient "down stream" into the arm or leg along the course of the spinal nerve which is pinched.

For example, a disc compressing the seventh cervical nerve root (C-7) will produce radiating pain along the course of the spinal nerve into the arm. This radiating pain (radiating downward from the nerve root along the course of the nerve) is called radicular pain since it originates from the spinal nerve root. ("radicular" = spinal nerve root; hence radicular pain).

Physicians call the medical condition resulting from compression (pinching) of a spinal nerve root a radiculopathy (radicular = spinal nerve root; opathy = disease). Pain and paresthesia (numbness, burning and tingling) follow the course of the spinal nerve. Radiating pain down the arm from compression of a cervical nerve root is called cervical radiculopathy; and radiating pain down the leg from compression of a lumbar nerve root is called lumbar radiculopathy.

Radiculopathy may result from many "space occupying lesions" (conditions that take up space in the spinal canal), such as a protruded or herniated disc, a large osteophyte (bony spur), or a tumor.

Radiculitis (radicular = spinal nerve root; itis = inflammation) is a medical condition resulting from inflammation of a spinal nerve root with pain and paresthesia along the course of the spinal nerve. A nerve root may be inflamed without being pinched, such as from being stretched (neuropraxia) or from surrounding tissue swelling, both resulting from the trauma of an auto collision.

#### **A. COUGHING, SNEEZING AND BOWEL STRAINING:**

Increased pressure is placed on a "space occupying lesion" such as a herniated disc, in the vertebral canal, when the patient coughs, sneezes or strains his bowels. The increased pressure further compresses the spinal nerve root resulting in pain "down stream", along the course of the compressed spinal nerve.

Reproduction of symptoms of nerve root compression by sneezing, coughing or straining is the basis of the Valsalva test. This is a simple test where the patient is asked to bear down and exert pressure in a manner similar to a bowel movement while holding his breath. A positive test results in reproduction of nerve root symptoms either radiating down the arm (cervical radiculopathy) or down the leg (lumbar radiculopathy).

**PRACTICE NOTE:** In medical reports, physicians will often note, "the patient denies increased pain with sneezing or coughing." The physician, in reciting the patient's history, is describing a negative (-) Valsalva maneuver which is one piece of evidence ruling out nerve root irritation.

## IX. SCIATICA (IRRITATION OF THE SCIATIC NERVE)

### A. ANATOMY:

The sciatic nerve is the longest nerve in the body. It is formed from the L4, L5, S1, S2, S3 nerve roots. The sciatic nerve runs down the back and the side of the leg to the toes. Figure 25A shows the sciatic nerve from the front, and Figure 25B shows the sciatic nerve from the back view.

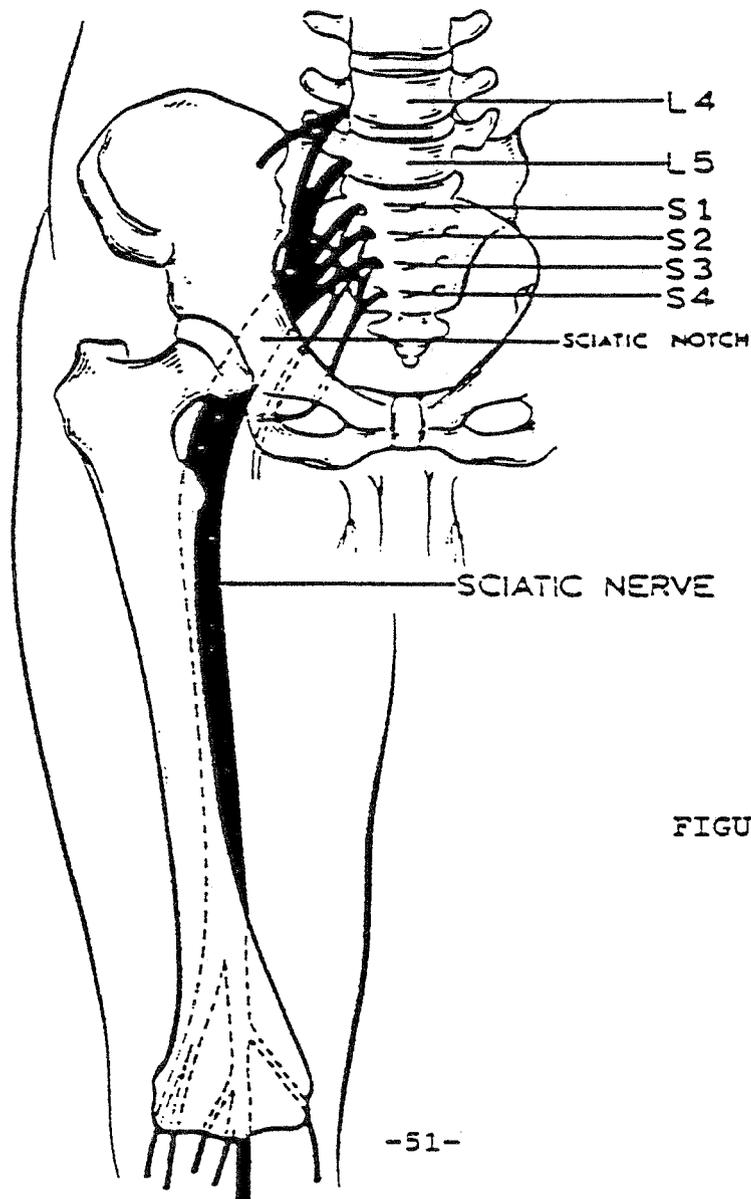


FIGURE 25A

©See p.1

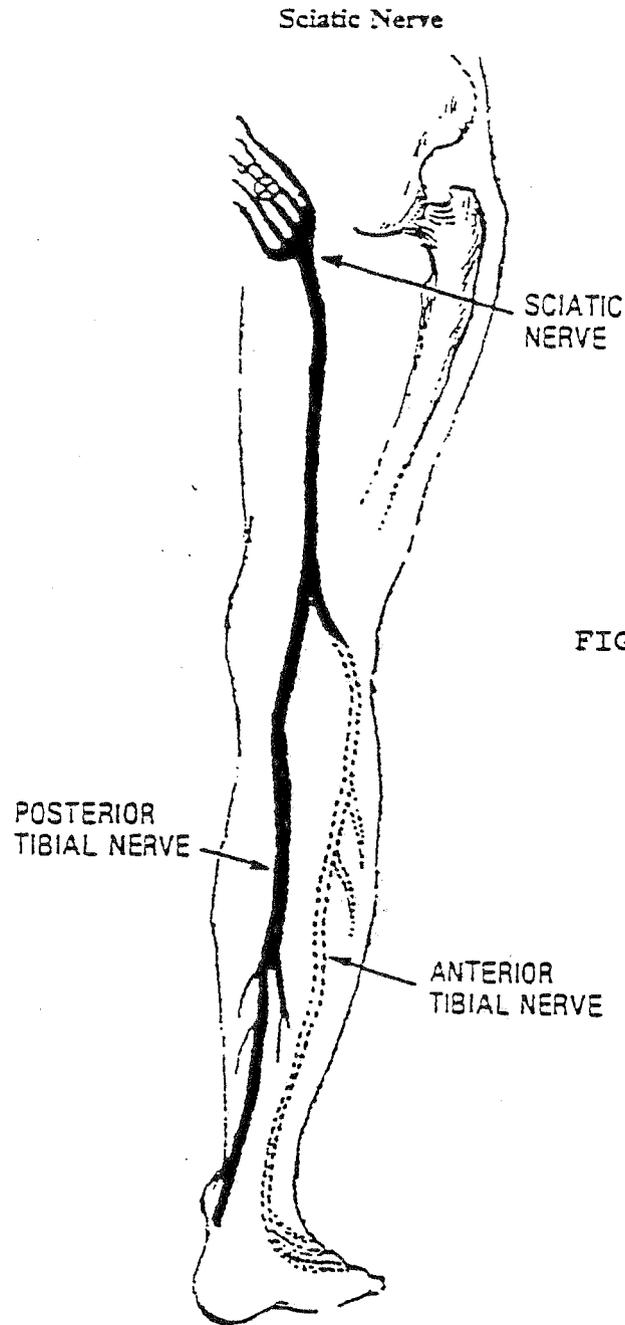


FIGURE 25B

©See p.1

## **B. SYMPTOMS OF SCIATICA:**

Compression of any of the nerve roots which form the sciatic nerve result in pain and paresthesia radiating down the course of the long sciatic nerve - radiculopathy. The classic complaint is

pain radiating down the buttock, back of the thigh, down the side of the leg and down the back of the leg, generally at least below the knee.

Radiculopathy involving the sciatic nerve is given a special name in honor of the body's longest nerve - sciatica. A detailed discussion of sciatica - its symptoms, examination findings, causes and treatment is found in Attachment No. 4 - Kahanovitz, "Sciatica: Verifying the Diagnosis, Offering Relief", J. Musculoskeletal Medicine, Jan. 1998, pages 51-58.

### **C. ANATOMICAL BASIS FOR SCIATIC STRETCH TESTS:**

Sciatic tension tests, such as the straight leg-raising test (SLR), the Lasegue's test, and the Milgram test, puts the sciatic nerve "on stretch", and are universally performed during physical examination of the low back as an aid in diagnosing sciatica.

The sciatic nerve is elastic. If any of the nerve roots, which form the sciatic nerve, is compressed, the distance the sciatic nerve must travel from the spinal canal down the leg to the toes is increased.

The tension tests put the sciatic nerve on "stretch" by taking up the normal "slack" in the long sciatic nerve which increases the compression and tension on an already compressed and irritated nerve root. The result is reproduction of radiating pain along the course of the sciatic nerve - a positive (+) test. A normal sciatic nerve (non-irritated) does not cause pain when put under stretch by these tests.

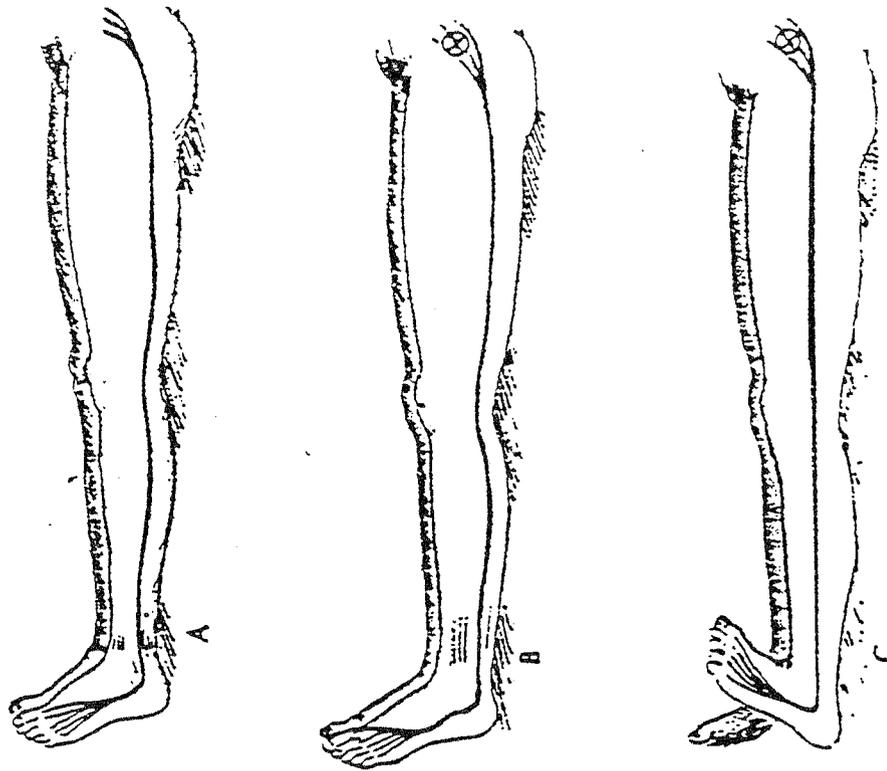


FIGURE 26

Diagrammatic representation of the concept of the sciatic nerve "on stretch" which produces "hyperirritability" and pain. (A) Normal nerve in relaxed position. (B) The space-occupying lesion (X) develops at the nerve's source increasing the distance the inelastic nerve must travel. (C) Dorsal extension (upward bending of the ankle joint) places the taut, "hyperirritable" nerve "on stretch," which causes pain. These physical phenomena are the physiological and anatomical base for many of the low back diagnostic maneuvers.

©See p.1

#### D. THE STRAIGHT LEG RAISING TEST:

##### 1. PERFORMING THE TEST:

The straight leg-raising test is performed with the patient lying on his back (supine) on the examining table. The physician simply lifts the patient's leg off of the table, flexing

the hip in the direction of the patient's head. The test is positive (+) if the patient reports radiating pain down the course of the sciatic nerve before a 90 degree angle is reached. The physician records the angle at which the sciatica is reproduced, e.g., 60 degrees. Generally, the more severe a patient's condition, the lower the angle is required to reproduce the sciatic pain. The opposite (normal) leg is also tested and will also result in a positive straight leg-raising test in many cases with sciatica reproduced in the involved leg - a contralateral straight leg-raising test.

## 2. POSITIVE TEST:

A positive sciatic stretch test, such as a straight leg-raising test is an indication of nerve root compression in the nerve roots which form the sciatic nerve. As previously noted, a disc can be herniated yet cause no nerve root compression. Symptoms of sciatica will not be experienced unless the nerve root is actually compressed by the herniated disc. A patient with a herniated disc which does not compress the nerve root will generally experience only localized low back pain (or be free from symptoms) and will have a negative straight leg-raising test. However, later in life, the inside disc material from the nucleus pulposus may become larger and actually press upon the nerve root producing sciatica with a positive straight leg-raising test.

The physician must distinguish a positive straight leg-raising test from a tight hamstring muscle (the thigh muscle in back of the

thigh when bends the knee). A tight hamstring muscle can restrict the angle the patient can raise his/her leg due to muscle spasm. The straight leg-raising test can be further defined to rule out pain from a tight hamstring muscle by additional maneuver of stretching the foot upward (dorsiflexion) toward the patient's head while the patient's leg is already off of the examining table and flexed. In addition, the patient's neck may be flexed forward by bending the chin toward the chest. Both maneuvers exert an additional "stretch" on the sciatic nerve confirming that a positive straight leg-raising test is the result of nerve root irritation and not a tight hamstring muscle.

The result of the straight leg-raising test usually vary from one office examination to the next depending on periods of decreased (remission) symptoms and periods of increased (exacerbation) symptoms. In addition, the results of the straight leg-raising test may be altered if a patient taking analgesics (pain relievers) since his threshold for experiencing sciatic pain may be increased, i.e., the test may be positive only by raising the leg at a higher angle in order to reproduce the sciatic pain.

### 3. THE VARIATION - SEATED STRAIGHT LEG-RAISING TEST:

A variation of the straight leg-raising test is done with the patient seated at the edge of the examining table with his legs dangling off the edge instead of lying supine.

The seated straight leg-raising test is performed by the physician who raises the patient's leg upward to the horizontal

position by straightening the patient's knee. The sciatic nerve is thus stretched.

**PRACTICE NOTE:** Defense examinations (IME) often have the patient perform both standard (supine) straight leg-raising test and the seated straight leg-raising test to observe discrepancies between both tests in an effort to rule-out malingering (faking).

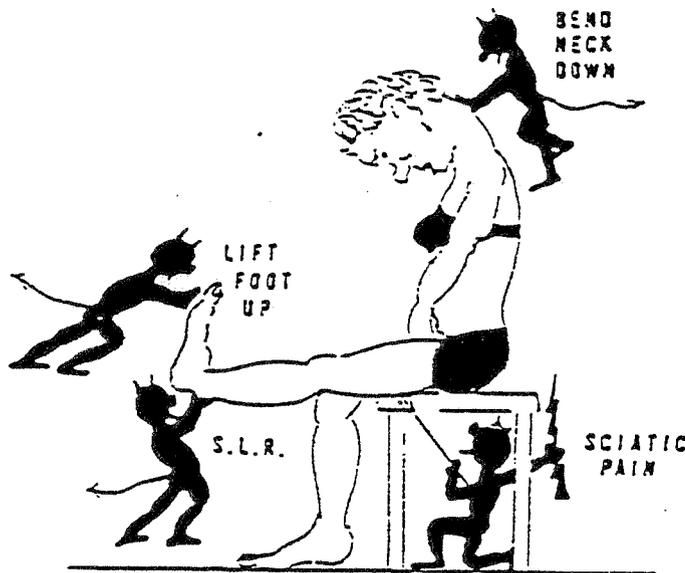
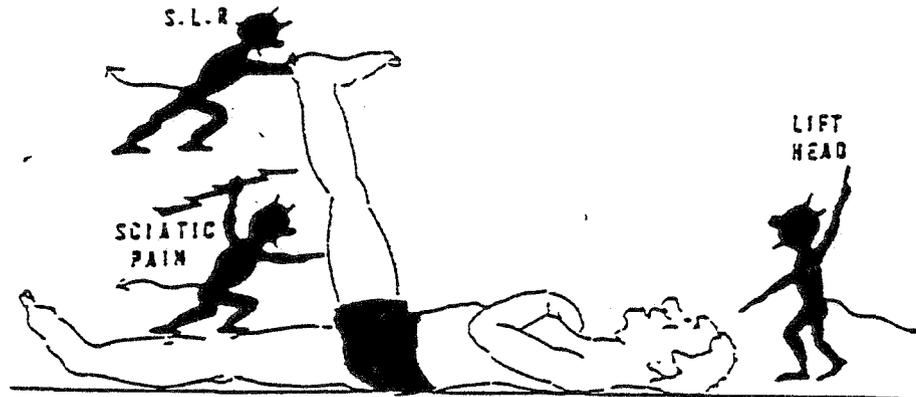


FIGURE 27

©See p.1

## X. OSTEOARTHRITIS OF THE SPINE

### A. HYPERTROPHIC DEGENERATIVE CHANGES:

Osteoarthritis (osteo = bone) of the spine involves "hypertrophic degenerative changes" and can occur at different places in the vertebral column. The term "hypertrophic degenerative changes" refers simply to an overgrowth of bone on the vertebrae, and is used synonymously with osteoarthritis.\* Osteophytes, spur like projections that look like the spurs of a cowboy boot, develop.

**NOTE:** Rheumatoid arthritis should not be confused with osteoarthritis. Rheumatoid arthritis of the spine involves systemic inflammatory conditions usually effecting several joints, including the knee joint, joints in the hand and the spine. Environmental factors trigger rheumatoid arthritic conditions in genetically predisposed persons. A patient with rheumatoid arthritis experiences classic symptoms of severe pain and stiffness in the morning which improves later in the day with activity. In contrast, osteoarthritis results from the overgrowth of bone, and is either wear and tear or traumatic arthritis. Osteoarthritis usually is localized to specific areas, and is not systemic (affecting the whole body).

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\*Strictly speaking, arthritis can only occur at a synovial joint. Although the facet joint is the only synovial joint in the vertebral column, the term osteoarthritis is loosely used by physicians to describe excess bony growth at any location in the vertebral column whether or not a true synovial joint.

1. DEGENERATIVE DISC DISEASE - THE AGING PROCESS:

As we age, our vertebrae and discs degenerate. An analogy can be made to a new car which slowly ages and degenerates with increased mileage. While we are young, our intervertebral discs have full height, the annulus fibrosus is firm and the nucleus pulposus is tense, and the outside annular ring is thick and functioning; our foramen are equivalent to big open windows; our spinal canal has sufficient "breathing room" for the spinal cord and the nerve roots; our facet joints are smooth with well oiled joint cartilages gliding freely one over the other; and our spinal ligaments are vibrant, elastic and strong.

The aging process begins with the intervertebral disc. The disc acts as a shock absorber, distributing stress between the vertebrae above and below it. The inside nucleus pulposus is able to act as a shock absorber because of its high water content. As we age, the nucleus pulposus slowly loses water and gradually "dries out". The outside annular ring (annulus fibrosus) slowly degenerates by losing its elasticity and slowly develops fissures. The intervertebral disc space and disc height (the space occupied by the disc sandwiched between the upper and lower vertebrae) becomes smaller. As the disc space gradually collapses, stress is placed on the posterior facet joints and ligaments which are not suited to handle the excessive stress. Degenerative, hypertrophic changes develop at the anterior (front) side of the vertebral body. Overgrowth of bone (hypertrophic degenerative changes) occur. Bony

spurs (which look like the spurs on a cowboy boot) develop close to the disc. Gradually, spurs develop at the facet joints and the spinal canal is rimmed with large osteophytes (spurs). As a result of additional stress, the spinal ligaments (especially the ligamentum flavum) thicken and encroach the spinal canal and the foramen. Spinal stenosis gradually develops with narrowing of the spinal canal. In advanced cases, the osteophytes (spurs) bridge across the disc space.

Degenerative disc disease in the cervical spine (neck) is a medical condition called cervical spondylosis.

Figure 28 illustrates the degenerative disease process (degenerative hypertrophic changes - osteoarthritis).

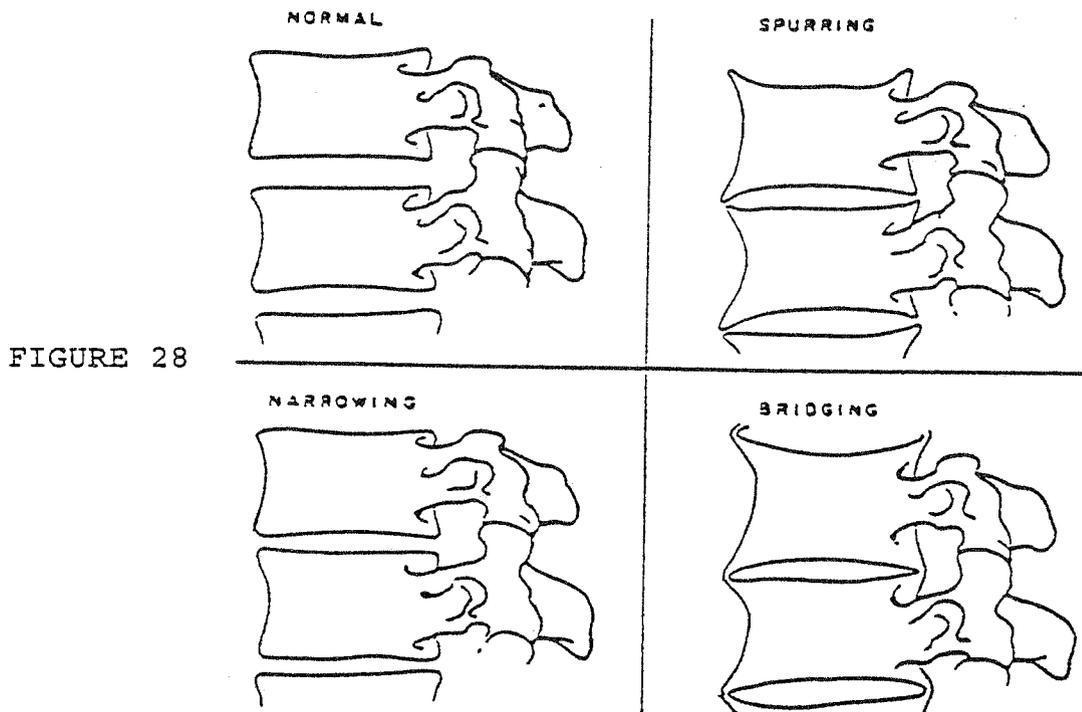


FIGURE 28

©See p.1

Diagrammatic representation of changes seen in X-ray views of disk injuries and osteoarthritis. When injured, the disk space tends to narrow due to collapse of the nucleus pulposus. Osteophytes or spurs develop at the edges of the vertebral body. In old cases, the osteophytes may actually bridge across the disk space.

2. PEOPLE WITH DEGENERATIVE DISC DISEASE OFTEN HAVE NO SYMPTOMS:

People with degenerative disc disease (osteoarthritis) often have no symptoms whatsoever. For example, almost all people over 55 have asymptomatic cervical spondylosis (degenerative disc disease of the cervical spine) which is only detected if plain x-rays are taken. People with cervical spondylosis can go throughout life with no symptoms. Wiesel, et al., in their text book, Neck Pain, 2d Ed., at page 50-51, states, "...In cadaver studies, almost everyone over the age of 55 will show evidence of cervical spondylosis. Obviously, not all of these patients are symptomatic. (Friedenberg, et al.\*), have emphasized the lack of correlation between radiographs of the cervical spine and symptoms. ...Radiographs (AP, lateral, oblique) of the cervical spine in cervical spondylosis show varying degrees of changes. These include disc space narrowing, osteophytosis, foraminal narrowing, degenerative changes of the facets, or instability. As previously discussed, these findings do not correlate with symptoms...". (emphasis added).

3. TRAUMA PRECIPITATES SYMPTOMS OF PREEXISTING, ASYMPTOMATIC OSTEOARTHRITIS:

Osteoarthritis of the spine is characterized by hypertrophic degenerative changes - the overgrowth of bone

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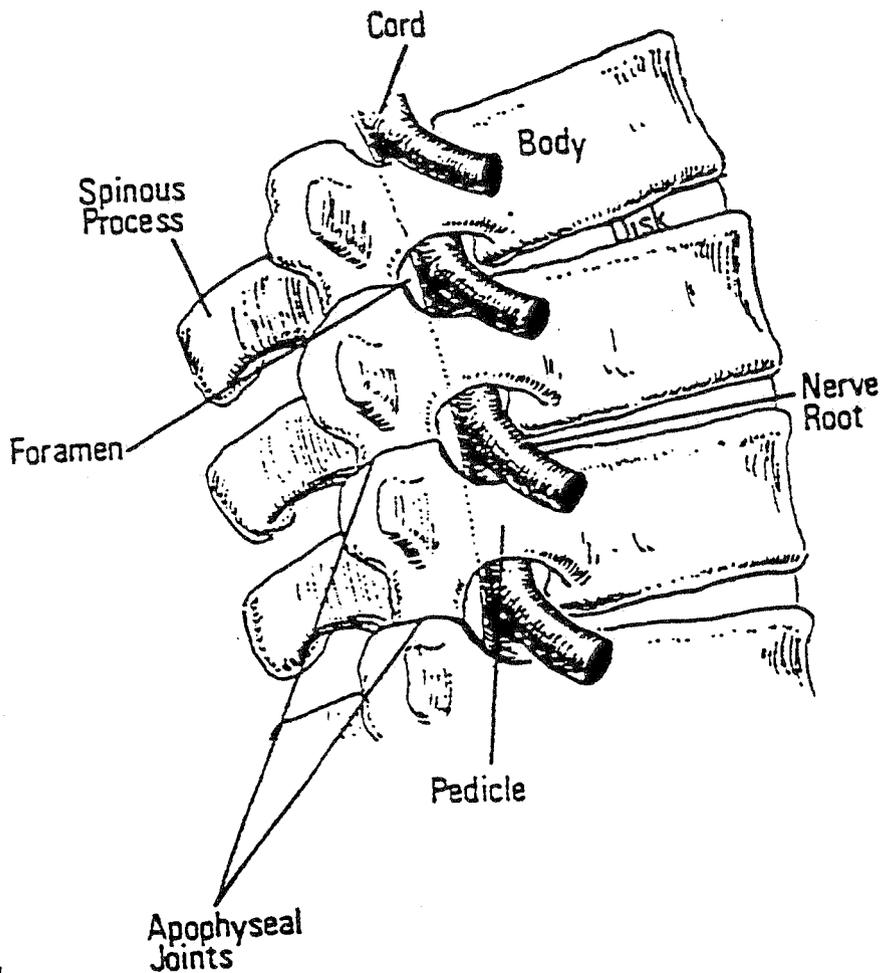
\*"Deneration - Disc Disease of the Cervical Spine", JBJS 45A, 1171-1178, 1963)

resulting in bony spur-like projections. The spur-like projections are sharp like the spur on a cowboy boot. These osteophytic spurs are located on the vertebrae near the vertebral foramen, the window where the nerve root exits from the spinal canal. As noted, many people have severe osteoarthritis of the spine with no symptoms whatsoever.

The hyperextension - hyperflexion motion (extending the neck backward and forward beyond its normal limits) as seen in auto collisions can cause a sharp pointed spur to strike a nerve root in the spine precipitating symptoms. In addition, the trauma can result in compression of a preexisting osteoarthritic spur against a nerve root. Trauma, as seen in hyperextension-hyperflexion injuries, causes stretching and tearing of the spinal ligaments. The surrounding microhemorrhage and tissue swelling can precipitate the symptoms of osteoarthritis in an asymptomatic person.

Professor Hoppenfeld, in his textbook Orthopedic Neurology, A Diagnostic Guide to Neurologic Levels, p. 8, (1977) describes the effects of an auto collision on the cervical spine as follows: "Patients frequently develop neck pain after automobile accidents which cause the cervical spine to whip back and forth (whiplash) or twist. The resulting injury may stretch an individual nerve root, cause a nerve root to impinge upon an osteoarthritic spur, or produce a herniated disc..." (emphasis added).

Figure 29 illustrates the result of extreme hyper-extension (forced bending of the neck backward as seen in auto collisions). The foramen narrow and constrict the nerve roots. If bony sharp spurs are present on the edges of the vertebrae, or in the foramen - anywhere in the vicinity of the nerve root - the nerve root will "take a direct hit" from the spur and the spur may even compress the nerve root resulting in symptoms in an otherwise asymptomatic patient with preexisting osteoarthritis of the spine.



©See p.1

FIGURE 29

4. ANALOGY OF OSTEOARTHRITIS OF THE SPINE TO A GRAY HAIR AND A MATCH:

The jury should understand that degenerative osteoarthritis, even in severe cases, can produce no symptoms. The aging process in the spine can be painless like the aging process involving our hair turning gray - with no symptoms. Trauma from an auto collision can precipitate symptoms like striking a match. The phosphorus is already present at the tip of the match. Trauma from the friction strike, lights up the match causing symptoms. Similarly, trauma from an auto collision lights up symptoms in asymptomatic, preexisting osteoarthritis of the spine. The defendant takes the plaintiff as he finds her and is responsible for precipitation and aggravation of the symptoms.

PRACTICE NOTE - CROSS-EXAMINATION OF THE DEFENSE "IME"

PHYSICIAN: The defense, many times, will allege that the patient's symptoms are solely the result of a preexisting osteoarthritis of the spine. The defense examiner must admit that patients, even with severe degenerative osteoarthritis are often asymptomatic. The following is a sample cross-examination of the defense doctor which illustrates this point:

Q: Doctor, in your own practice, you have at times seen patients with severe osteoarthritis of the spine, as shown on x-ray, who had no symptoms whatsoever just before a traumatic injury, such as an auto collision?

A: Yes.

Q: Doctor, isn't it well known in medicine that many patients who have severe degenerative osteoarthritis of the spine, often have no symptoms whatsoever?

A: Yes.

Q: Doctor, isn't it also well known in medicine that patients with severe degenerative osteoarthritis of the spine, as shown on x-rays, may go throughout their lifetime with no symptoms?

A: Yes.

Q: Doctor, before this auto wreck, Mr. Plaintiff had no history whatsoever of neck or back pain.

A: Yes, that is correct.

Q: Doctor, would you agree that the precipitating cause of the plaintiff's pain (severe osteoarthritis) was the auto collision of November 5, 1997?

A: Yes.

5. MINOR IMPACT CASE - USE PREEXISTING OSTEOARTHRITIS AS AN OFFENSE:

Jurors have a difficult time believing that a substantial injury can result from a minor impact collision. In a patient with preexisting, asymptomatic osteoarthritis, the defense will argue that the patient's injury is a result of this preexisting condition and not the minor impact collision. This defense should be used as an offense on behalf of the plaintiff. An elderly person with asymptomatic osteoarthritis is more likely to sustain a significant neck injury from a minor impact than would a healthy football player, 19 years of age. In addition, the soft tissue injury to the plaintiff's neck, ligaments and muscles will take much longer to heal, and may even be permanent because of the activation of the plaintiff's preexisting asymptomatic osteoarthritis. (Remember: The defendant takes the plaintiff as he finds him.)

(a) Closing Argument - Analogy to Eggs and Golf Balls:

The "eggshell skull" doctrine can be illustrated to the jury, during closing argument, by asking a rhetorical question using an analogy to "eggs and golf balls".

CLOSING ARGUMENT: Assume, this case were about a farmer taking eggs to the market. Enroute, he is rear-ended, and all the eggs fall out of the truck and break as they hit the ground. No one would have any trouble awarding the farmer damages for the cost of his eggs. Is it a defense to say "the farmer should have been carrying golf balls"? Similarly, is it a defense to say that Mr. Plaintiff should have had the spine of a 19 year old boy?

6. TRAUMA ITSELF MAY CAUSE OSTEOARTHRITIS:

Trauma from an auto collision can itself cause hypertrophic degenerative changes (osteoarthritis) in the spine. The trauma from an auto collision results in hyperextension, hyperflexion of the spine with resulting tearing and stretching of ligaments. Microhemorrhaging and swelling in the tissues surrounding the vertebrae develop - causing the bony vertebrae to react by overgrowing bone - hypertrophic spur formation. It is important to obtain plain x-ray films taken on the day of the accident in the emergency room and compare these with x-ray films taken at a later date. It normally takes at least 4 - 6 months for degenerative changes to develop in the spine as a result of trauma. In addition, trauma can accelerate an already existing degenerative condition. A comparison of the x-rays, CT scans and MRI films at different time intervals is essential to document the development of osteoarthritis where none previously existed and to document the acceleration of a preexisting osteoarthritic condition. For

example, in one case the author tried, the initial emergency room x-ray report found "minimal degenerative changes"; whereas six months later, the x-ray report noted "moderate degenerative changes" in the same area of the cervical spine. The trauma of the auto collision had accelerated the preexisting osteoarthritis of the spine.

## **B. FACET JOINT - OSTEOARTHRITIS:**

In the back of each vertebrae are two sets of facets (a smooth flat surface of bone) which "hook-up" with the facets of vertebral bodies directly above and below when the vertebrae are stacked together, one on top of the other, to form the vertebral column. When the facets hook-up, they form facet joints (also called apophyseal joints). A joint is defined as a place in the body where two bone surfaces meet. This "meeting place" is also called an articulation and the joint surfaces are called articular surfaces. The surfaces of the facets are smooth and flat. Facet joints are true synovial joints (the only synovial joints in the vertebral column), since it is lined by a thin synovial membrane which produces a "gliding oil" called synovial fluid. Synovial fluid is present in all synovial joints, such as the knee, and helps the bones of the joint glide over each other. Synovial fluid also lubricates the joint surfaces preventing wear and tear breakdown. The facet joints glide over each other and permit motion of the spine, especially bending forward (flexion) and backward (extension).

Chiropractic manipulation involves the facet joints. If those joints experience subluxation (misalignment) they are put back in place by manipulation.

As we grow older, our facet joints experience wear and tear at the smooth joint surfaces. With time, some people develop osteoarthritis of the facet joint which is characterized by excess bone growth at the joint surfaces. The condition is degenerative, and will gradually deteriorate slowly. In addition, bony, sharp, pointed spurs develop at the facet joints as part of the process of degenerative joint disease.

Figure 30 shows the facet joints.

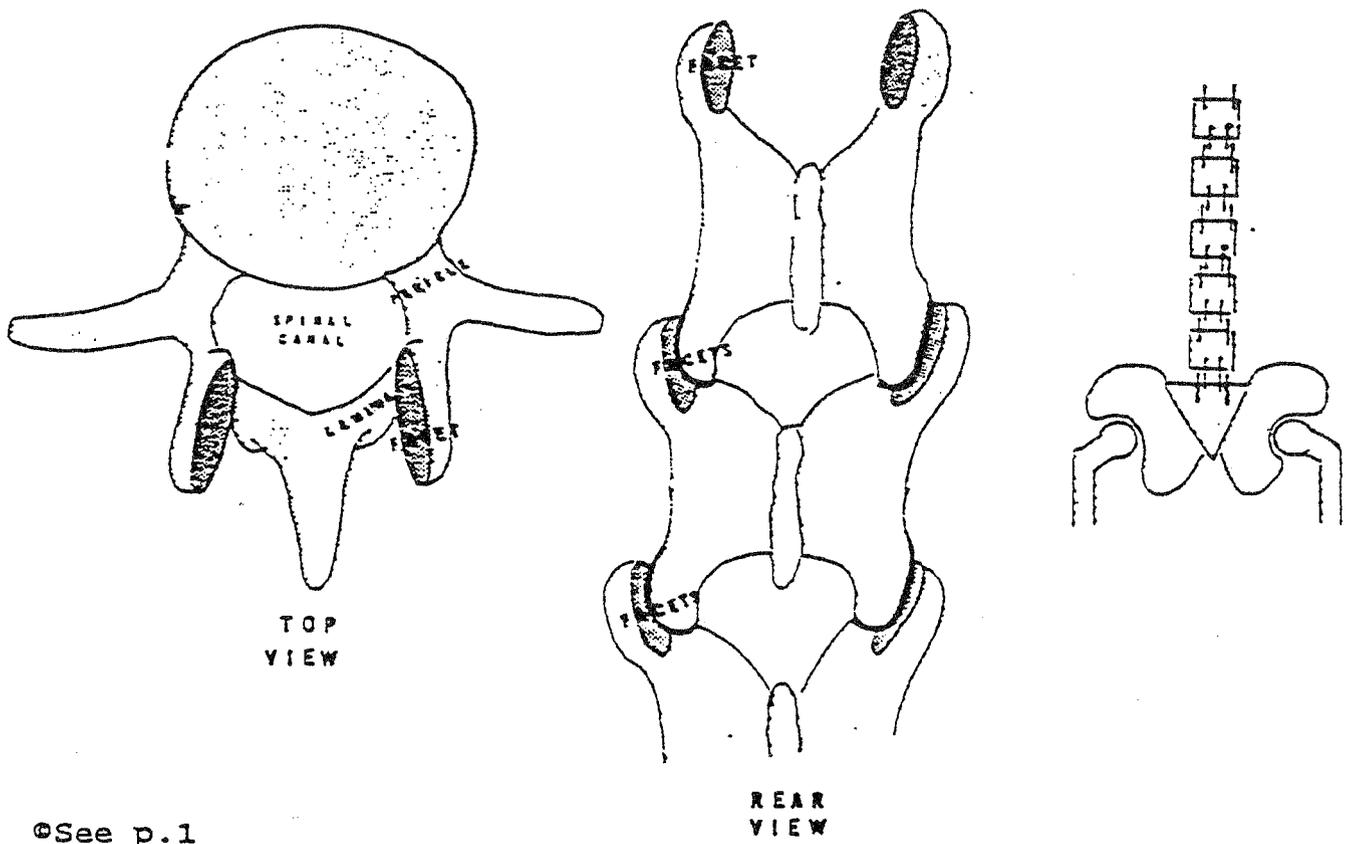


FIGURE 30

### C. SPINAL STENOSIS:

Spinal stenosis (narrowing) is a condition resulting from narrowing of the spinal canal. By analogy, the spinal canal is formed by stacking horseshoes around a metal stake (the spinal cord). If the neural arches of the vertebrae (equivalent to our horseshoe) are narrowed, there is less room around the spinal cord - hence, the spinal canal is said to be narrowed.

If the window of the foramen, where the nerve root exits the spinal canal, is also narrowed, the nerve root is given less room as it exits the spinal canal through the open foramen window. The window begins to close on the nerve root.

The size of the spinal canal and the diameter of the foramen are important in considering nerve root compression. Generally, the smaller the spinal canal and the smaller the diameter of the foramen, the greater the probability that nerve root compression, from, for example, a herniated disc, will result. In people with a large spinal canal and large foramen, the nerve root has enough "breathing room" to escape pressure from a source of compression, such as a herniated disc, which takes up space. With disc herniation in a small spinal canal, there is just not enough "breathing room" available for the nerve root to be free from compression.

Spinal stenosis is a relative condition and symptoms, if any, will vary from person to person, depending upon the degree and site of narrowing. As we age, our spinal canal becomes narrower. Some people will have no symptoms; while others will have localized

back pain or nerve root compression along the course of the spinal nerve if the narrowing pinches the nerve root. Usually several levels of the spinal cord are involved, not merely one root level since the entire canal is smaller.

Spinal stenosis is used by physicians to describe the relative narrowing of the spinal canal and the narrowing of the diameter of the foramen.

Spinal stenosis in the cervical (neck) region of the spine is often referred to as cervical spondylosis which is degenerative disc disease of the cervical spine.

The term "lumbar spinal stenosis" is a recognized medical condition. Symptoms can be either localized to the area of spinal stenosis, or radiate down the course of the involved spinal nerve.

Figure 31A shows a cross-section of a vertebrae with a normal spinal canal. Figure 31B shows a spinal canal with degenerative stenosis. Note the narrowing of the neural arch (the horseshoe around the spinal cord).

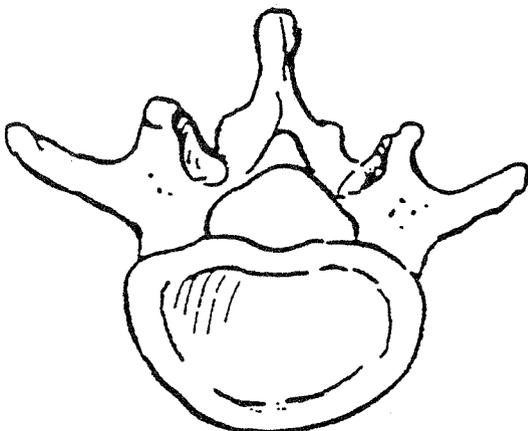


FIGURE 31A

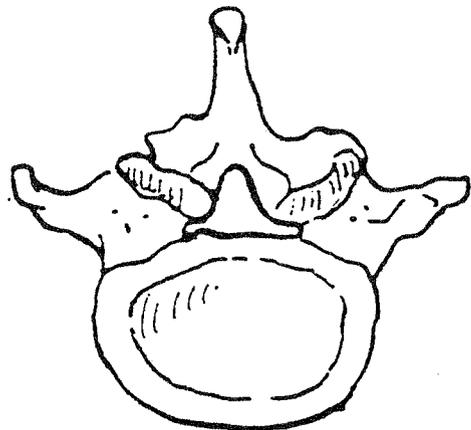


FIGURE 31B

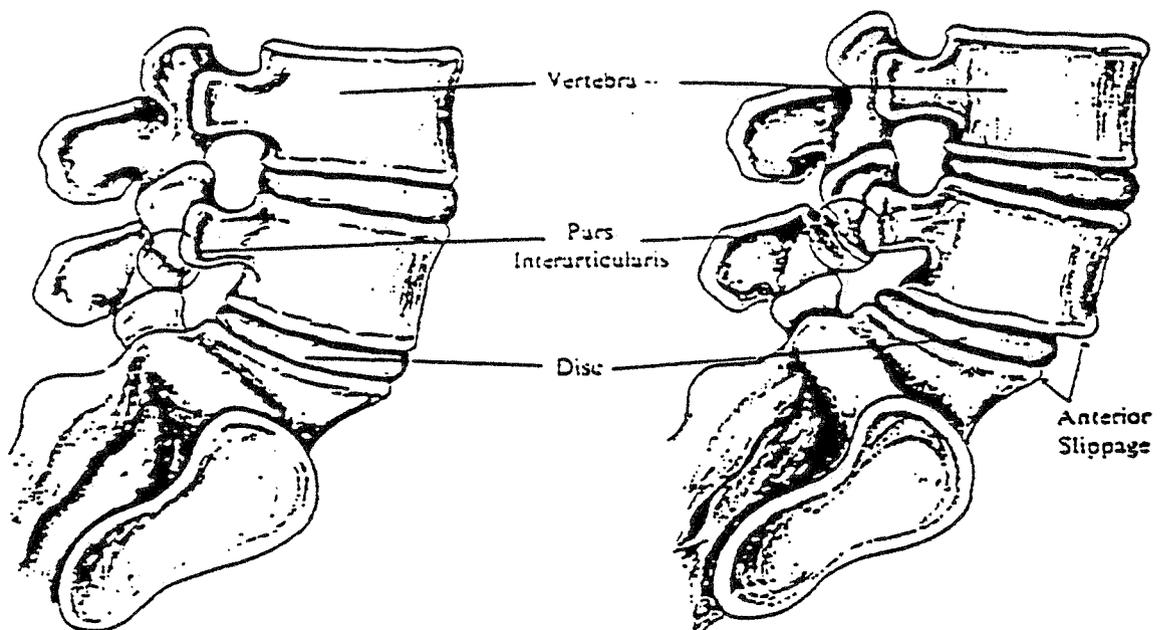
## XI. SPONDYLOLYSIS AND SPONDYLOLISTHESIS (THE SPONDY BROTHERS)

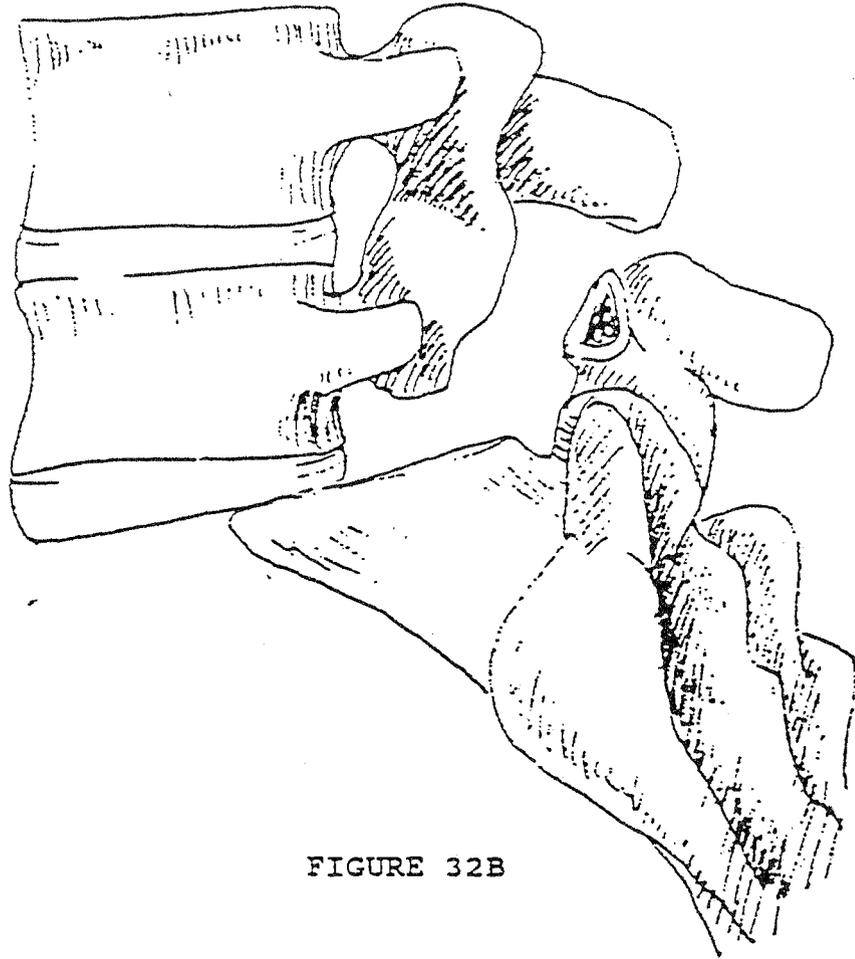
Spondylolysis is derived from the Greek (spondylos = vertebrae and lysis - dissolution) since this condition represents a flattening of a vertebral body without any forward displacement. Spondylolisthesis, on the other hand, involves forward displacement - slippage of one vertebrae on another. The bottom vertebrae is a shelf for the top vertebrae. Spondylolisthesis is derived from the Greek (spondylosis = vertebrae, olisthanein = to slip).

Spondylolisthesis is found mainly at the lumbosacral joint where the L-5 vertebrae slips forward (like the Leaning Tower of Pisa) on the S-1 vertebral body.

**MEMORY AID:** An easy way to remember that spondylolisthesis involves slippage of a vertebrae is to think of a graduate student failing his thesis exam - "slipped up on his thesis"!

Figure 32A compares spondylolysis and spondylolisthesis. Note the slippage of the L5 vertebrae on the first sacral vertebrae which is the classic finding in spondylolisthesis. Figure 32B shows Grade 4 spondylolisthesis.





©See p.1

FIGURE 32B

#### A. DEFECT IN THE PARS INTERARTICULARIS:

Both spondylolysis and spondylolisthesis result from a congenital or developmental defect in the back portion of the vertebral body near the neural arch, called the pars interarticularis. Note Figure 32A. A fracture of the pars interarticularis occurs either at birth or in early life. The fragments of the fracture are united by fibrous tissue and do not have the strength of a normal bony union. Patients with this condition normally do not have symptoms until the mid 30s, or they may go through life with no symptoms whatsoever. A sudden twisting, lifting motion or trauma from an accident will precipitate back symptoms. Once the back symptoms first begin,

they will continue on a recurring basis with periods of remission. The symptoms resulting from these conditions are normally localized to the low back unless the slipped vertebrae of spondylolisthesis pushes on the nerve root causing radiating pain along the course of the spinal nerve.

## **B. PREEXISTING CONDITION OFTEN WITH NO SYMPTOMS:**

Many people with spondylolisthesis, even past middle age, have no symptoms. Weisel, et al., in their text Low Back Pain, 2d Ed., p.71, state, "At this point, it should be reemphasized that in some people, even severe displacement is asymptomatic and gives rise to no disability. It is not uncommon to pick-up a previously unrecognized spondylolisthesis on a routine gastrointestinal radiological study of a 50 year old patient."

## **C. THE ROLE OF TRAUMA IN SPONDYLOLYSIS AND SPONDYLOLISTHESIS:**

A patient with spondylolysis or spondylolisthesis is an "eggshell skull" plaintiff with an already preexisting unstable back from the condition itself. A traumatic event, such as an auto collision, can trigger or precipitate the symptoms of spondylolysis or spondylolisthesis in an asymptomatic patient. (The symptoms can also develop without any noticeable acute trauma).

A patient with preexisting spondylolisthesis with only localized low back pain may develop nerve root irritation following trauma if the "slipped forward" vertebrae is pushed into

a nerve root. Spondylolisthesis is measured by grades 1-4, depending upon the degree of slippage of the fifth lumbar vertebrae. Trauma from an auto collision can cause the vertebrae to slip forward even more. For example, trauma may push a Grade 1 condition to Grade 2.

In addition, trauma can convert a spondylolysis (which involves a defect of the pars interarticularis, but with no slippage of one vertebrae on another) into a spondylolisthesis by causing a slippage of the top vertebrae.

It is possible to sustain an acute fracture of the pars interarticularis as a result of trauma but this is rare. The defense will claim that the fracture of the pars interarticularis is a preexisting condition. If there are no prior x-rays, the only way to prove the recent fracture is with a bone scan within three months of the acute injury, which will light-up at the site of the fracture if the fracture is recent. (A "hot spot" resulting from the accumulation of the radioactive bone scan dye).

# PART TWO

## I. SOFT TISSUE INJURIES

Soft tissue injuries result from forced hyperextension-hyperflexion motion caused by auto collisions. Injuries to the following soft tissues are discussed in this section\*:

- Ligaments, muscle and fascia -- strain and sprain injuries and myofascial pain syndrome (MPS).
- Blood vessels (vertebral and carotid artery) -- vasoconstriction with decreased blood flow to the brain.
- Sympathetic nerves -- nerve stimulation and irritation.
- Brain -- post-concussion syndrome.

### A. MECHANISM OF INJURY - FORCED HYPEREXTENSION-HYPERFLEXION MOTION.

In all auto collisions, the head and neck are forced in the direction of the striking car.

#### 1. REAR-END COLLISIONS:

Rear-end collisions result in a sudden acceleration of the body forward, while the head and neck are forcefully hyperextended (backward) followed by a recoil of forced hyperflexion (forward).

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\*Fibromyalgia is discussed in Part III

2. HEAD-ON COLLISIONS:

Head-on collisions result in the head and neck being forced forward in hyperextension followed by a recoil of hyperflexion (backward).

3. SIDE COLLISIONS:

In side collisions, the head and neck are thrown to the side towards the striking car and then recoils in the opposite direction.

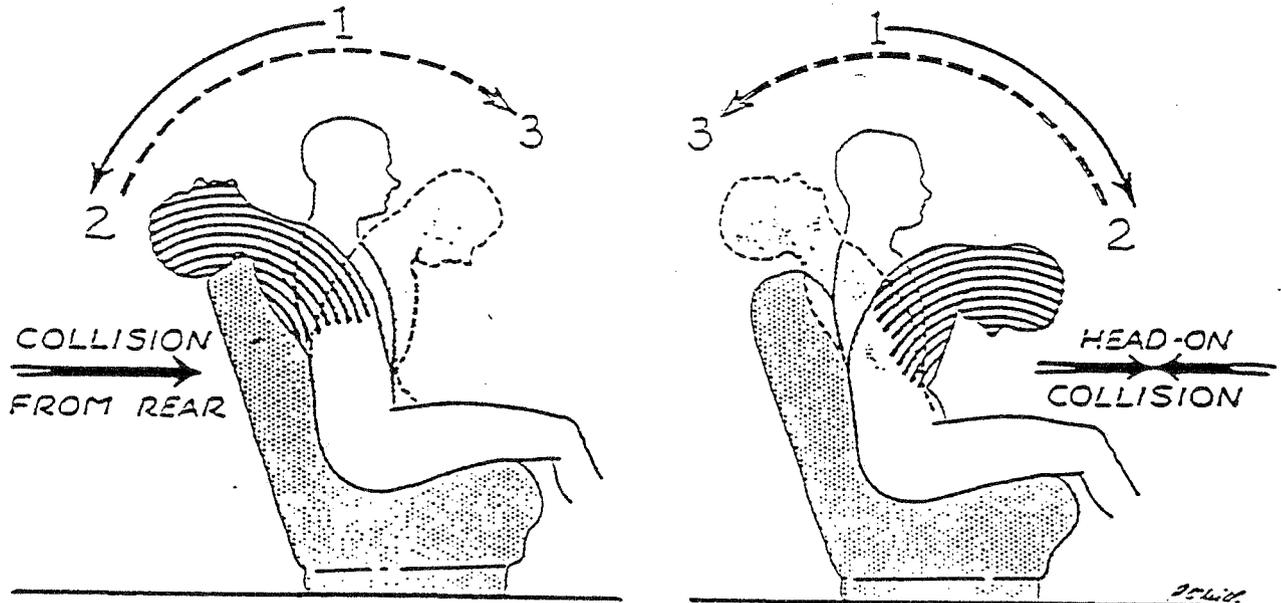
4. THE SUDDEN STOP:

In sudden stops, the body is accelerated forward.

In a rear-end collision, the defense attorney will attempt to prove that a sudden stop caused the plaintiff's neck injury rather than the rear-end collision. If the plaintiff testifies that his head and neck were "pushed forward causing my head to strike the windshield", the laws of physics support the defense attorney's contention.

If the cause of the plaintiff's injury were solely from the rear-end collision, the plaintiff's head and neck would first be thrown backward (hyperextension) in the direction of the striking car, followed by recoil (hyperflexion) forward. Since the plaintiff testified that his head and neck were pushed forward (without first being thrown backward), the plaintiff's own testimony establishes the cause of his striking the windshield to be the sudden stop.

Figure 33 illustrates the mechanism of forced hyperextension-hyperflexion motion resulting in soft tissue injury from rear-end and head-on collisions.



Schematic Drawing of Mechanism of Injury in Rear-End and Head-on Collision

FIGURE 33

©See p.1

**PRACTICE NOTE:** A victim of a rear-end collision has his head and neck whipped backward and forward very quickly. To the victim, which motion occurs first is often blurred in his mind. Therefore, during the initial client interview, it is important to discuss the mechanism of injury with the client.

## **B. FACTORS AFFECTING THE SEVERITY OF INJURY:**

Many variables determine the extent of soft tissue injury from an auto collision. These include:

- The position of the head at impact;
- The acceleration speed of the car being struck;

- Vehicle parameters, such as:
    - Being built to collapse (energy absorbing);
    - Head restraints;
    - Seatbacks;
    - Seatbelts and shoulder harnesses.
  - Human factors, such as the condition of the plaintiff prior to impact.
1. EQUATING BODILY INJURY WITH THE EXTENT OF PROPERTY DAMAGE CONDEMNED:

The practice of equating bodily injury with the extent of property damage to the autos has been condemned by physicians and engineers. For example, Drs. Foreman and Croft, in their textbook, Whiplash Injuries (1988), p. 62, state:

"The notion that one can calculate or predict the type or extent of soft tissue damage sustained by occupants of a vehicle merely by calculating the G forces produced in the vehicle by the resultant collision should clearly be laid to rest. The practice of calculating bodily injury to the victim by estimating the cost of auto damage is naive at best and should be condemned..."(emphasis added).

Similarly, Drs. Carroll et al., "J. Musculoskeletal Medicine" (June 1992) p.100 state:

"The amount of damages to the automobile bears little relation to the force applied to the cervical spine of the occupants..."

2. THE POSITION OF THE HEAD AT IMPACT:

It is well known that a victim whose head is partially rotated and caught off balance will incur a severe injury even from a minor impact. More severe injuries result from oblique angle

collisions, where a driver is hit from the left rear corner while turning left. (The driver's head is rotated at impact).

Drs. Foreman and Croft, "Whiplash Injuries", (1988) p.68, explain the physiological basis for the increased severity of injury:

"When the head is rotated 45°, the amount of extension that the spine is capable of is decreased by 50 percent. This results in increased compressive load at the facet joint....

The intervertebral foramen is also small on the side of rotation and lateral flexion, and its contents are more vulnerable to injury..."

See also, Carroll et al., J. Musculoskeletal Medicine (June 1992) 97, 109, Attachment No. 1.

### 3. ACCELERATION OF THE CAR BEING STRUCK:

The acceleration of the plaintiff's car upon being struck is a factor in determining the extent of soft tissue injury since the rate of acceleration forward determines the rate of hyperextension of the neck following impact. Professors Wiesel, et. al., in their text, Neck Pain, 2d Edition (1992), p. 292, discuss acceleration as a factor in causing injury:

"....it becomes apparent that the degree of injury depends on the force applied and inertia rather than the speed of the striking vehicle. The faster the struck vehicle is accelerated forward, the more rapid the hyperextension of the neck and the greater the injury. Therefore, a car struck by a large truck or loaded bus travelling 5-7 mph will be accelerated forward as much as if struck by a small car travelling in a much greater velocity. Also, how rapidly the struck car is accelerated forward will depend on road and traffic conditions. If the struck car is essentially bumper to bumper with the car in front, it cannot be catapulted forward, then a strike from the rear might result in

significant damage to the struck car, from both forward and back collisions but the struck car might not be accelerated forward making the resulting injury to the driver slight. In this case, the damage to the car might be severe. If, however, the struck car were on an icy street with no cars in front, a rear end collision would accelerate the struck car at a high rate causing very little demonstrable damage to the car but a severe hyperextension cervical injury to the driver..." (emphasis added).

Other factors affecting the acceleration of the plaintiff's car at impact and hence the rate of hyperextension are whether the brakes were on at the time of impact and the speed of the plaintiff's vehicle at the time it was struck.

The faster the plaintiff is travelling at time of impact, the greater will be his acceleration. A greater acceleration results in a greater rate of hyperextension with increased soft tissue injury.

4. SURPRISE COLLISIONS AND BRACING:

"Surprise" rear-end collisions result in greater soft tissue injury than collisions where the victim is aware of an impending crash and has braced himself. Bracing for the collision decreases the extent of the forced hyperextension and recoil hyperflexion forces which cause injury to the soft tissues.

5. PARAMETERS OF THE PLAINTIFF'S VEHICLE:

(a) Energy Absorbing - Built to Collapse Vehicles:

The force (energy) from an auto collision is either absorbed by the car or by its occupants. In older cars which are "built like a tank," little property damage is done to the car with most of the force of impact absorbed by its occupants. Modern

safety engineering has now designed cars that are "built to collapse,". The car absorbs the energy of impact rather than the occupants. For example, Volvo has advertised the safety of its cars by "collapsing on impact" - absorbing the forces which would otherwise have been transmitted to the occupant - thereby providing a safer car.

Drs. Foreman and Croft, in "Whiplash Injuries", p. 67, describe the secret of safety engineering as follows:

"...The driver climbs out of the wreckage with only minor injuries. The secret of this incredible crashworthiness is built into the car - this intentional piece-meal disintegration absorbs nearly all the energy in the crash. Thus, a car that is built to collapse with impact (within safe limits) is safer than one that is not...."

(b) Head Restraints:

Head restraints are designed to limit injury resulting from the forceful hyperextension (backward) motion of the head and neck at the time of impact. If the force of the backward hyperextension phase is reduced, so will the force of the hyperflexion - with less injury to the muscles and ligaments of the neck.

In 1969, head restraints became mandatory. Unfortunately, head restraints have become "headrests" with the initial purpose of safety becoming second place to comfort. If the head restraint is not adjusted properly, increased neck injury can result. For example, if the head restraint is adjusted downward, it acts as a fulcrum, especially with tall drivers, causing greater injury to the ligaments and muscles of the neck.

(c) Seatbacks:

Seatbacks, like head restraints, if properly designed and used, decrease the rate of forced hyperextension and its recoil hyperflexion during impact. Proper seat adjustment and the degree of "spring" in the seatback directly influence the extent of injury. For example, a seatback which is designed like a "steel spring" would increase the intensity of the recoil hyperflexion in a rear-end collision.

Engineering studies have shown that controlled seatback collapse at the time of impact reduces the severity of soft tissue injuries. Accordingly, cars having this type of seatback design result in less severe neck injuries.

(d) Seatbelts:

The severity of soft tissue injury is affected by the use of seatbelts, head restraints, and shoulder straps. Wiesel, et al., Neck Pain, 2d Edition, pg. 13, describes this relationship.

"The influence of the seatbelts on acute neck injuries is dependent on other factors - the most important of which is the presence or absence of seat head restraints. If head restraints are present, the use of seatbelts alone result in fewer neck injuries. Without head restraints, the use of seatbelts alone appear to slightly increase the incidence of neck injuries. Combining seat belts with a shoulder strap reduces the incidence of acute neck injuries even without head restraints. The combined use of seatbelt, shoulder strap and head restraint significantly reduces the number of acute neck injuries."

Some vehicles contain shoulder harnesses which lock at a distance from the occupant's body for extra comfort. During impact, the slack in this type of shoulder harness allows the

occupant's body to travel a greater distance, with potentiation of forward angular rotation, resulting in more severe neck injury had no slack been present.

(e) Human Factors:

People, whose neck structure is less resilient will suffer greater soft tissue injury than others with more resilient, elastic muscles and ligaments. Drs. Foreman and Croft, in their text Whiplash Injuries (1988), p. 67, describe this physiological variance affecting the extent of soft tissue injury:

"Certain physiological variances in people will significantly affect the amount of damage they sustain in a collision. This is why, as is often seen in practice, an occupant in a crash may be injured, while another may escape with no apparent injury. With an increase in age, the elasticity of the tissues decreases. Range of motion in the cervical spine also decreases. In both cases, the potential for injury is increased because the neck is less resilient..."

In addition, a plaintiff, who has asymptomatic degenerative osteoarthritis of the spine, is more likely to sustain injury in a minor impact collision than a younger person in his twenties with no osteoarthritis of the spine.

C. CATEGORIES OF SOFT TISSUE INJURIES:

1. INJURIES TO LIGAMENTS, MUSCLES AND FASCIA:

(a) Spinal Ligaments:

(i) Ligaments Hold the Spine in Place - The spine is essentially a stacked "bag of bones." Without a binder (glue), the vertebral column would topple like a high stack of dominos.

The spinal ligaments are the glue holding each vertebrae in the vertebral column in place. The ligaments essentially prevent the vertebrae from "slipping around." (Remember that the ligaments hold bone to bone. In the spine, the ligaments hold vertebrae to vertebrae).

(ii) Interspinal Ligament - Each vertebrae has projections on its back side called a "process." We can feel these projections by placing our fingers in the middle of our back. These projections are also called the spinous process (spinous = spine). The interspinous ligament (between the spine), also called the spinal ligament, hold the spinous processes together.

This interspinal ligament is illustrated in Figures 34 and 35.

(iii) Ligamentum Flavum (The Yellow Ligament) - Ligamentum Flavum (flavum = yellow) bind the lamina (the back of neural arch) of one vertebrae to another. (Remember: In the aging process of the disc, the ligamentum flavum thickens and begins to encroach the spinal canal and the foramen.

(iv) The Anterior and Posterior Longitudinal Ligaments -- (The Long Ligaments) - The anterior and posterior longitudinal ligaments run the entire length of the vertebral column in the front (anterior) and in the back (posterior). As noted, these ligaments are attached to the vertebrae and discs and actually cover the outside of the annulus

fibrosus of the disc. The posterior longitudinal ligament is the weaker of the two and is rhomboidal in shape. Figure 22 above and Figures 34 and 35 below, illustrate these ligaments.

(v) Diagram of the Spinal Ligaments - Figures 34 and 35 show the major spinal ligaments.

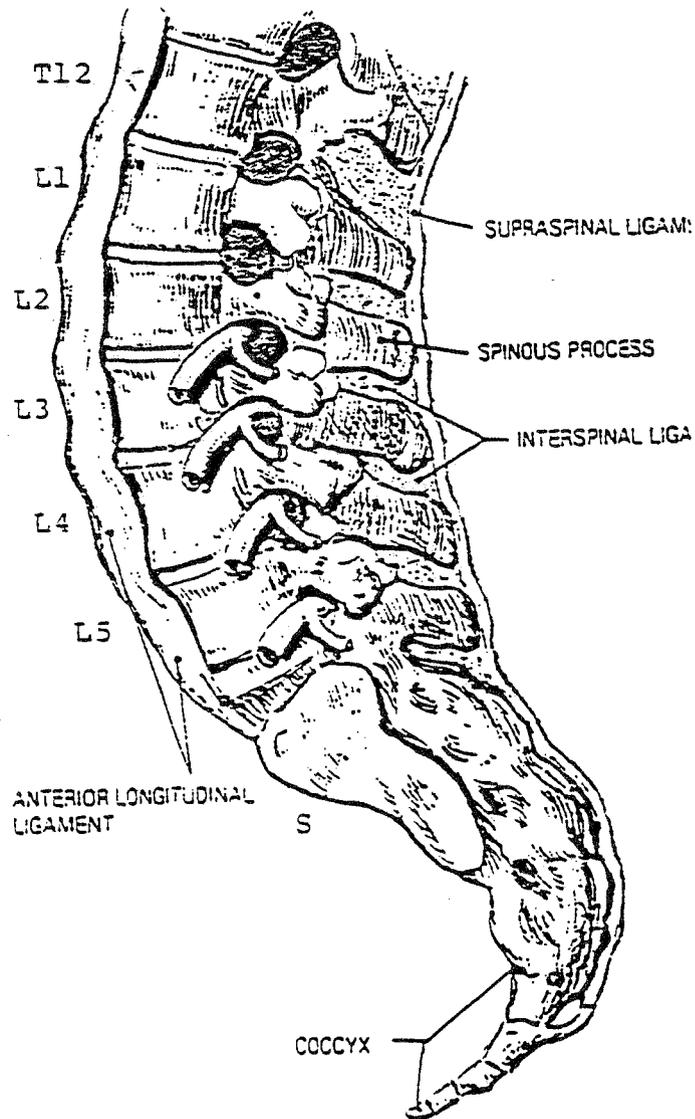
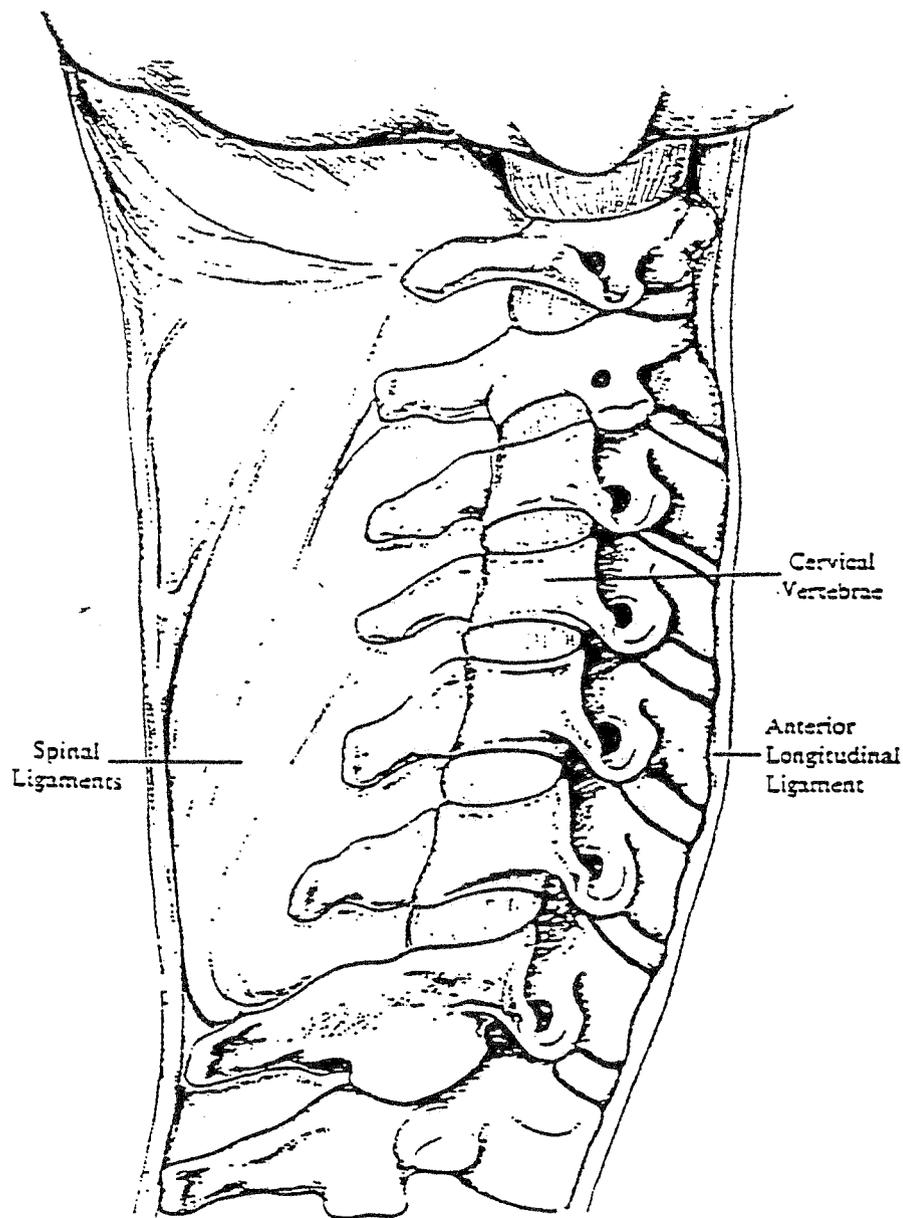


FIGURE 34



©See p.1

Cervical Ligaments and Cervical Vertebrae

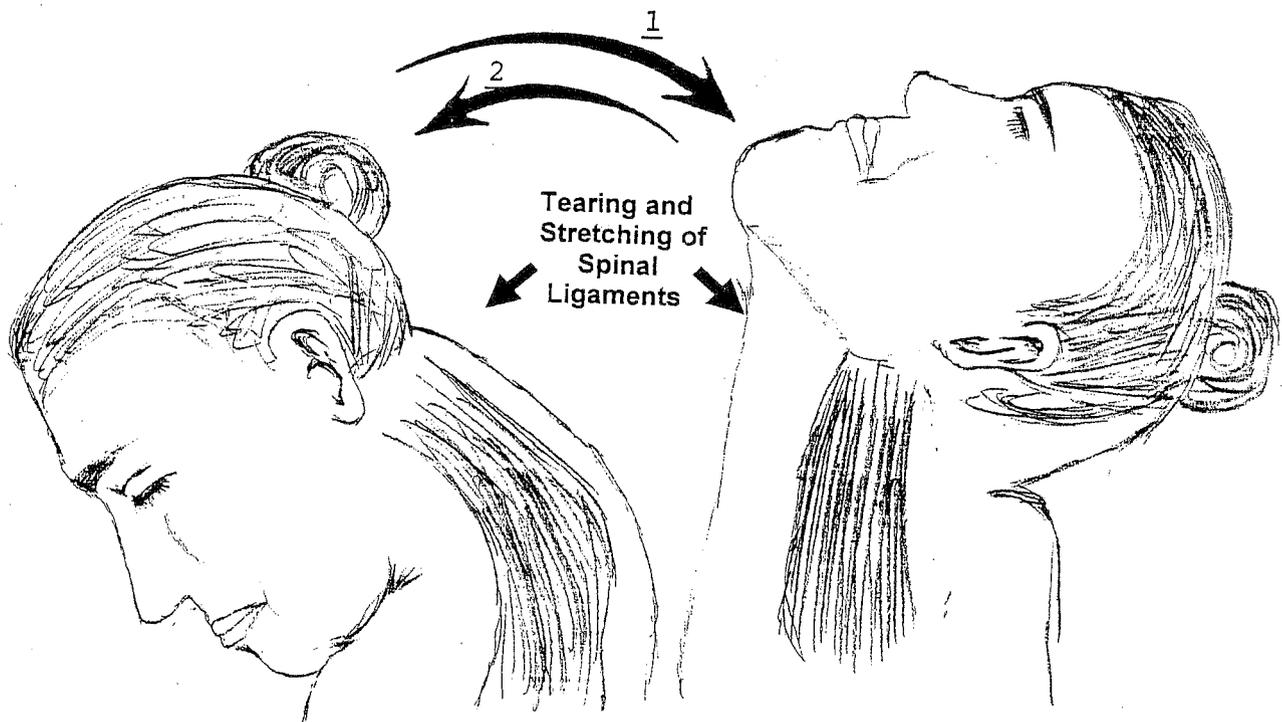
FIGURE 35

(vi) Injury to the Spinal Ligaments - Hyperextension-hyperflexion motion forcibly stretch and tear the spinal ligaments.

The tearing of spinal ligaments results in microhemorrhage and swelling deep within the surrounding tissues which cannot be seen

from the outside of the body nor with conventional radiological techniques such as x-rays, MRIs and CAT scans. The tearing of spinal ligaments may result in spinal nerve irritation and even herniation of the disc. For example, Wiesel, Neck Pain, (2nd Ed. 1992. p.291) states: "... tears of the anterior longitudinal ligament may cause hematoma formation with resultant cervical radiculitis (arm pain) and injury to the intervertebral disc..."

Figure 36 illustrates the tearing of spinal ligaments from a sudden hyperextension-flexion motion caused by a rear-end auto collision.



RECOIL FLEXION

HYPEREXTENSION

FROM REAR-END AUTO COLLISION

FIGURE 36

## B. MUSCLES AND FASCIA:

### 1. FASCIA:

Muscles and fascia lay on top of the spinal ligaments. Muscles are enclosed in a band of tough, fibrous tissue called fascia. (Fascia from the Latin meaning band). In essence, the muscle is "in the bag" - held together in a bag of fascia.

### 2. PARASPINAL MUSCLES:

The paraspinal muscles (para = beside) - also called the paraspinous muscles - lay on top of the spinal ligaments. These muscles protect the spine, and assist with movement. Figure 39B shows the paraspinal muscles. On top of the paraspinal muscles lay fascia and the exterior muscles of the neck and back, such as scalene muscles, shown in Figure 11B above. The paraspinal muscles in the neck are termed the cervical paraspinal muscles, and in the low back the lumbar paraspinal muscles.

### 3. TRAPEZIUS MUSCLES:

The trapezius muscle is named after its shape as a trapezoid (four-sided). The trapezius muscle is a large muscle arising from the back of the head and the cervical and thoracic vertebrae. The trapezius extends to the collar bone and the shoulder blades. It helps raise and straighten the shoulder. Since the trapezius is a large muscle, some physicians label various portions as T1, T2, T3, and T4.

#### 4. STERNOCLEIDOMASTOID MUSCLE:

The sternocleidomastoid muscle is named from its bony connections. (Sterno = chest; cleido = collar bone; and mastoid = the mastoid process below the ear). This long, thin muscle is injured in most rear-end auto collisions. Since its name is long, it is abbreviated "SCM." This muscle serves to rotate the neck and flex the head forward.

#### 5. SPLenius CAPITIS MUSCLE:

The splenius capitis (head) is a major muscle in the back of the head which originates from the neck area and goes all the way up the back of the head. This muscle is often injured in rear-end auto collisions. The occipital (back of the head) nerve passes through the splenius capitis muscle. If the splenius capitis muscle is in spasm, the occipital nerve will be pinched causing the nerve to go into a spasm. The result -- muscle tension headaches in the back of the head.

#### 6. INJURY TO MUSCLE AND FASCIA:

Like ligaments, muscles and fascia are stretched and torn by forceful hyperextension-hyperflexion motion.

Figure 37 illustrates hyperextension-hyperflexion injury to the trapezius and sternocleidomastoid muscles following an auto collision.

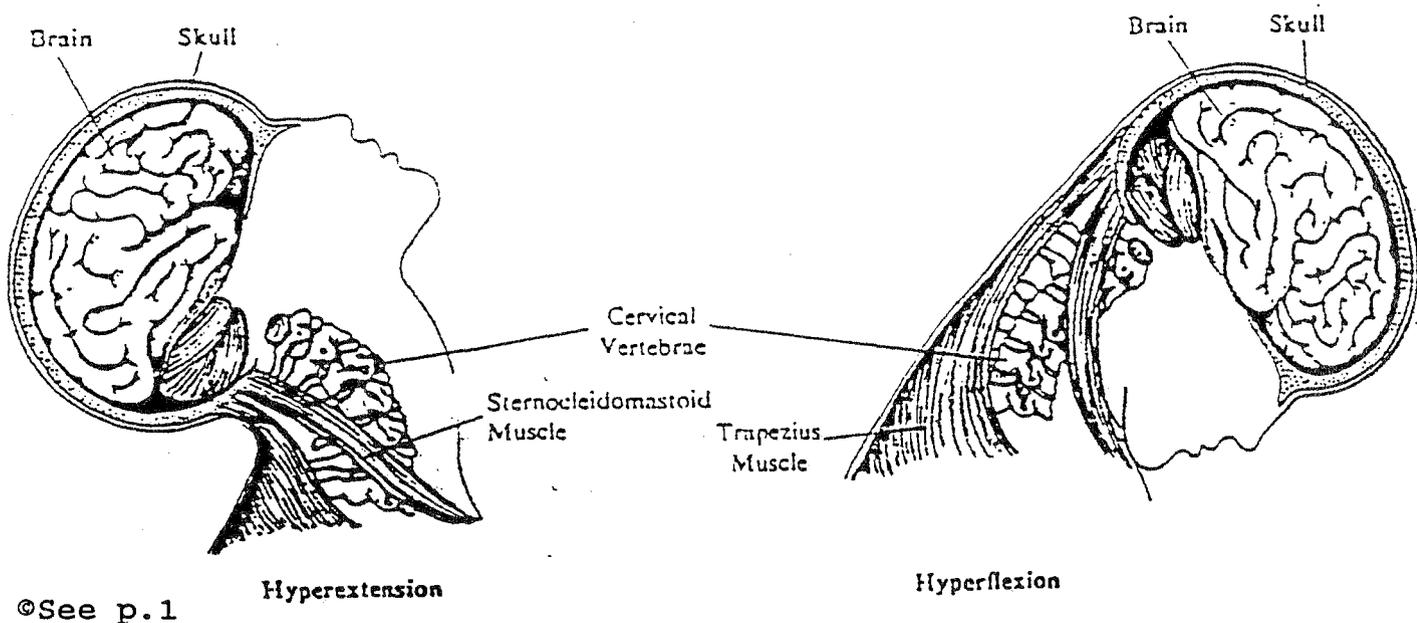


FIGURE 37

#### 7. MYOFASCIAL PAIN SYNDROME (MPS):

Myofascial pain syndrome (MPS) results from an injury to muscle and its surrounding band ("bag") of fascia. Trigger points (hyperirritable, hard nodules, like the tip of a pencil) develop within the injured muscle. A physician can feel the trigger point by pressing on it with his fingers reproducing classic pain patterns.

A trigger point itself creates pain and muscle spasm, in certain bands, not the entire muscle. Pain generated by a trigger point refers pain to neighboring areas. For example, a trigger point in a neck muscle can refer pain into the arm or up the neck. A trigger point in the low back can refer pain down the back or into the leg. Trigger points have been "mapped", and have specific

patterns of referred pain. This concept is the key to understanding the symptoms of myofascial pain syndrome.

Trigger points are treated with injections of a local anesthetic alone or mixed with an anti-inflammatory steroid, followed with vigorous stretching during physical therapy to break up the trigger point. The International Rehabilitation Medicine Association's Monograph on Myofascial Pain Syndrome is attached as Exhibit No. 6.

## **C. SPRAIN AND STRAIN INJURIES:**

### **1. DEFINITION:**

Hyperextension-hyperflexion injuries to ligaments and muscles are called strains and sprains. Professor Ruth Jackson, in her textbook, The Cervical Syndrome, 4th Ed. (1978) pp. 86-87 defines these terms as follows\*:

- Strain - the stretching of ligaments and muscles
- Sprain - the tearing of ligaments and muscles.

These injuries are also referred to as "myoligamentous strain or sprain." (Myo = muscle, Ligamentous = ligament).

---

\*Other physicians define a "strain" as a stretch or tear injury to a ligament; and a "sprain" as a stretch or tear injury to a muscle.

MEMORY AID: A good way to remember that strain injuries result in stretching is to associate the first two letters of Strain with the first two letters of Stretching.

(a) Reference to Levels of the Spine and Specific

Muscles:

Strain and sprain injuries are subdivided according to the four regions of the spine. For example, if ligaments and/or muscles of the neck are strained the injury is called a cervical strain. If ligaments and/or muscles of the low back are torn the injury is called a lumbar sprain or a lumbosacral (LS) sprain.

If specific muscles are injured, some physicians will specify the muscle in the diagnosis. For example a strain of the trapezius.

(b) Strain and Sprain are Distinct Injuries:

Strain and sprain injuries are distinct injuries resulting from forced hyperextension-hyperflexion motion. Although these terms have been used interchangeably, Dr. Jackson in her textbook, The Cervical Syndrome, 4th Ed. (1978) p. 86 states:

"Injuries of ligamentous, capsular and muscular structures are often referred to as strains and sprains. These two words are used interchangeably but erroneously, to indicate the type of injury."

2. HEALING: "ONCE A SCAR, ALWAYS A SCAR":

Torn ligaments and muscles heal by developing scar tissue. Scar tissue is permanent. Houts and Marmor use the term "Once a scar always a scar" to describe this permanent change from healthy tissue to scar tissue.

Healthy tissue is elastic while scar tissue is inelastic. Scar tissue shrinks during the healing process and looks knotted. Scar tissue is abnormal and can place stress on muscles and nerves causing further injury.

The scarring process varies from person to person; some people develop heavy thick scars; others develop thinner, finer scars. This concept explains why some patients develop permanent residuals from sprain injuries while others do not.

The formation of permanent scar tissue from sprain injuries is well recognized in medicine. For example, Professor Jackson, in The Cervical Syndrome, 4th Ed. (1978) p. 87 states:

"... sprains of the ligaments and capsules heal within 6-8 weeks by formation of scar tissue, which is less elastic and less functional than normal tissue. Varying degrees of residual alteration of their functional capacity is inevitable."

### 3. SYMPTOMS OF STRAIN AND SPRAIN INJURIES:

Common symptoms of strain and sprain injuries include:

1. Pain;
2. Muscle spasm;
3. Stiffness;
4. Decreased range of motion; and
5. Headaches.

#### (a) Onset:

Symptoms of strain and sprain injuries usually develop 12-24 hours after trauma. For example, Wiesel et al., Neck Pain, 2nd Ed. (1992) p. 293 state: "usually, the driver is often unaware

that he has been injured. He suffers little discomfort at the scene of the accident and often does not wish to go to the hospital. Later that evening or the next day, 12 to 14 hours after the accident, the patient begins to feel stiffness in his neck. Pain at the base of the neck increases and is made worse by head and neck movements. Soon, any movement of the head or neck causes excruciating pain ...."

PRACTICE NOTE: Defense attorneys minimize a plaintiff's injury by pointing out that the plaintiff did not report an injury at the scene nor went to an emergency room. In addition, emergency room records may not reflect all developing soft tissue injuries since symptoms in all areas may not be present. For example, a plaintiff may present at the emergency room only with neck pain. Back pain may not begin until the next morning. The treating physician should explain to the jury that delayed onset of symptoms is expected in strain and sprain injuries.

**(b) Referred Pain:**

Pain associated with neck and back strains and sprains is often referred to nearby areas. For example, pain is often referred from the neck to the following areas: the shoulder blade (scapula); to the back of the shoulder; to the chest (often referred to cervical angina pectoris); and to the back of the head (the occipital region). (From the Latin pertaining to the occiput -- back of the head).

Referred pain is distinguished from radicular (radiating pain) since referred pain results from a direct injury to ligaments and muscles whereas radicular pain results from compression of a spinal nerve root, with pain radiating down the course of the nerve into the arms or the legs.

Patients with strains and sprains may also experience radicular pain since forced hyperextension-hyperflexion motion can (1) stretch nerve roots (neuropraxia); (2) stretch and tear ligaments with resulting bleeding and swelling deep within the surrounding tissues which can irritate spinal nerves; (3) scar tissue from healing spinal ligaments or muscles can further irritate spinal nerves; and (4) cause a sharp pre-existing osteoarthritic spur to either strike or compress a nerve root.

(c) Muscle Spasm, Stiffness and Decreased Range of Motion:

Muscle spasm is an involuntary contraction of muscle to prevent further pain by "splinting" the injured area. Involuntary muscle spasm attempts to prevent movement which would increase pain. The result of muscle spasm is stiffness and decreased range of motion.

As discussed, ligaments bind and protect the spinal column. Stretching or tearing of these ligaments results in involuntary, reflex spasm of the paraspinal muscles, which cover the spinal ligaments. The result is immobilization of the spinal column and its surrounding ligaments. Muscle spasm has in effect placed a "protective splint" around the spinal column to prevent increased pain and further injury.

Severe, continuous muscle spasm of the neck muscles cause the head to tilt to one side. This condition is called torticollis, also known as "wryneck." (Latin for tortus = twisted; collum = neck).

(d) Headache:

The most common types of headache from cervical strain and sprain injuries, according to Houts and Marmor, are muscle tension (spasm headache); vascular headache and psychogenic headache (originated by the mind).

(i) Muscle Tension Headache - Muscle tension headaches are the result of muscle spasms. For example, as noted, fibers of the occipital nerve pass through the splenius capitis muscle, which runs from the neck all the way up the back of the head. Neck (cervical) strains and sprains often cause the splenius capitis muscle to go into spasm and become tense. Spasm of the splenius capitis muscle "pinches and tightens the occipital nerve" running between its muscle fibers, causing a muscle tension headache.

This condition is often referred to as occipital neuralgia (occipital = occipital nerve in the back of the head; neuralgia = pain along the course of the nerve).

A muscle tension headache is often called a basilar headache since it occurs at the base of the head (the occipital region).

(ii) Vascular Headaches - Vascular (blood vessels) headaches results from disrupted blood flow to the brain. Forced hyperextension-hyperflexion motion can reduce blood flow of the vertebral artery as it "snakes" it way through the cervical vertebrae to the brain. Figure 38.

A classic symptom of vascular headache is a "throbbing headache." The throbbing follows the beating of the heart.

(iii) Psychogenic (Emotional) Headache - Neck strain and sprain injuries produce physical pain. A person's life may be interrupted by the trauma of an auto collision resulting in emotional stress. The emotional stress can either cause muscle tension headache or aggravate one by generating additional muscle spasm creating a vicious cycle: muscle tension headache produces emotional stress which produces muscle spasm which causes additional emotional distress which produces additional muscle spasm.

#### 4. BLOOD VESSEL INJURY:

The vertebral artery supplies the back half of the brain with its blood supply. The vertebral artery "snakes" through and around the cervical vertebrae in route to the brain. The front half of the brain receives its blood supply from the common carotid and internal carotid arteries. Blood vessels are considered soft tissues because they are "soft" compared to hard tissue, such as bone and cartilage. Injury to the vertebral artery is also called a "vertebrobasilar vascular" injury (vertebro = vertebrae; basilar = base of the head; vascular = blood vessel).

The vertebral artery is particularly vulnerable to being stretched or compressed during forced hyperextension-hyperflexion motion causing the artery to go into spasm with resulting vasoconstriction.

(a) COMMON SYMPTOMS RESULTING FROM INJURY TO THE VERTEBRAL ARTERY:

Common symptoms resulting from hyperextension-hyperflexion injury to the vertebral artery are:

- (1) Loss of equilibrium and vertigo;
- (2) Nausea and vomiting;
- (3) Tinnitus (ringing in the ears);
- (4) Temporary deafness; and
- (5) Blurred vision and diplopia (perception of two images).

Figure 38 shows the vulnerable vertebral artery as it "snakes up" between the vertebrae enroute to the brain. Also shown is the carotid artery.

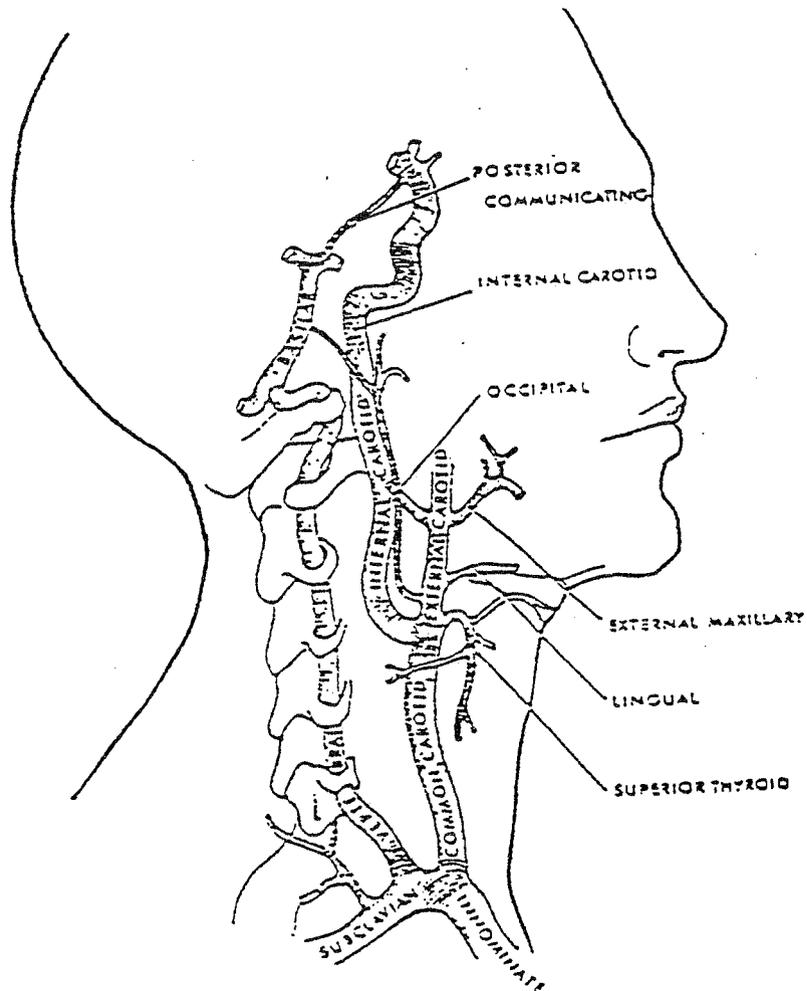


FIGURE 38

©See p.1

5. SYMPATHETIC NERVES:

(a) Anatomy of Sympathetic Nerves:

Sympathetic nerves are part of the autonomic nervous system. The central nervous system (CNS) consists of the brain, spinal cord, and spinal nerves (the cranial nerves are not discussed in this outline) which innervate skeletal muscles resulting in movement. Unlike the CNS, the autonomic nervous system regulates the body's internal environment by innervating involuntary muscle. The autonomic nervous system innervates the smooth muscle and glands in the following areas:

- |                       |                  |
|-----------------------|------------------|
| 1. Eye                | 5. Blood vessels |
| 2. Ear                | 6. Skin          |
| 3. Larynx (voice box) | 7. GI tract      |
| 4. Lungs              |                  |

These sympathetic nerves originate from the spinal cord below the level of C5 where they form chains of nerves. The sympathetic nerves are vulnerable to injury from forced hyperextension-hyperflexion motion.

(b) Function of Sympathetic Nerves:

The autonomic nervous system is divided into (1) sympathetic nerves and (2) parasympathetic nerves (para = opposite), each providing equal and opposite responses to smooth muscle and glands thereby providing the body with balance. For example, sympathetic nerves to the iris of the eye cause the pupil to enlarge (dilate) while parasympathetic nerves cause the pupil to

become smaller (constrict). Paralysis of the sympathetic nerve to the iris causes the pupil to constrict. (Horner's Syndrome, named after it's discoverer).

The sympathetic nervous system aids the body in an emergency -- prepares us for "Fight" and "Flight". Thus, stimulation of sympathetic nerves dilate the eye; increases the heart rate; constricts arteries where blood is not needed in an emergency, such as the skin and the intestines, while dilating the coronary arteries which supplies blood to the heart where it is needed; the skin is contracted and the sweat glands are stimulated. The parasympathetic nerve stimulation has the opposite effect -- preparing the body for relaxation such as constricting the eye; decreasing the heart rate; dilating arteries which supply blood to the skin and intestines.

(i) Blue Arms and Cold Hands - Victims of hyperextension-hyperflexion injury may report their extremities turn blue and cold. The basis for this change is injury to the sympathetic nervous system causing stimulation of the nerve.

Arteries which carry blood into the skin receive sympathetic and parasympathetic nerve supply. Sympathetic nerves cause these arteries to constrict (vasoconstriction) (vaso = blood vessels); while the corresponding parasympathetic nerve causes the artery to dilate with increased blood flow to the skin.

Coldness in an extremity, with the skin turning blue, is caused by the vasoconstriction of the sympathetic nerve with decreased blood flow to the skin. Since the blood flow to the skin

is decreased, the skin will turn blue and skin temperature will become colder. Blue skin is called "cyanotic." (The term cyanotic is derived from the effects of cyanide poisoning -- a lack of oxygenated blood to the tissues).

(ii) Symptoms Resulting From Injuries to the Sympathetic Nerves - Forced hyperextension-hyperflexion injuries to the sympathetic nerves cause either stimulation or irritation of the nerve resulting in the following recognized symptoms:

A. The Eye.

1. Dilation of the pupil.
2. Constriction of the pupil resulting from paralysis of the sympathetic nerve (Horner's Syndrome).
3. Blurred vision.
4. Pain behind the eye.
5. Excessive tearing.

B. The Ear.

1. Loss of equilibrium and balance.
2. Vertigo, dizziness and nausea.
3. Tinnitus (ringing in the ears).
4. Temporary deafness.

C. The Larynx (Voice Box).

1. Hoarseness.

D. The Skin.

1. Cold and blue.

6. BRAIN CONCUSSION (POST-CONCUSSION SYNDROME):

The brain consists of very soft tissue having the consistency of Jell-O. Imagine a 10 inch diameter Jell-O mold surrounded by two bowls -- one on top and one on the bottom. You have just pictured the brain housed in the skull! What effect will a sudden, violent, forced hyperextension-hyperflexion motion have on the Jell-O mold or brain? At a minimum, it will be "shaken up" which is the definition of a concussion -- a "shaking of the brain." Physical impact with a fixed object, such as the head striking the windshield is not necessary. The shaking of the brain has already occurred by the very force generated by the hyperextension-hyperflexion motion.

Post-concussion syndrome is a medical condition often seen following forced hyperextension-hyperflexion motion to the neck and head with a well recognized set of symptoms (syndrome).

Wiesel, et al., Neck Pain, 2nd Ed. (1992) p. 293 describes post-concussion syndrome resulting from an auto collision: "Patients receiving a whiplash injury of the neck can also suffer from a concussion. Mechanical deformation of the brain occurs during the acceleration-deceleration phase of the injury and concussion can occur without the head striking anything. This can account for transient loss of consciousness, as well as post-concussion syndrome or headache, photophobia, mild transient

confusion, tinnitus (ringing in the ears), fatigue, and transient difficulty with concentration."

**7. INJURY TO THE ESOPHAGUS AND TMJ JOINT.**

Forced hyperextension-hyperflexion injury may injure the esophagus (food pipe) resulting in difficulty swallowing, dysphagia (dys = dysfunction, phagia = to swallow).

Forced hyperextension-hyperflexion also causes injury to larynx (voice box) causing hoarseness.

During the hyperextension phase, the driver's head is forcibly thrown backwards, with the mouth opening, which may damage the temporomandibular joint (TMJ). The TMJ joint refers to the joint formed where the temporal bone meets the mandible in the jaw.

**D. NAMES OF HYPEREXTENSION-HYPERFLEXION INJURIES:**

Different names have been used interchangeably over the years to describe soft tissue injuries resulting from forced hyperextension-hyperflexion motion. These include:

- (1) Hyperextension-hyperflexion injuries;
- (2) Hyperextension-flexion injuries;
- (3) Acceleration-Deceleration injuries
- (4) Strain and sprain injuries;
- (5) Myoligamentous injuries;
- (6) Soft tissue injuries;
- (7) Myofascial pain syndrome (MPS);
- (8) Cervical syndrome; and
- (9) Whiplash injuries.

The terms hyperextension-hyperflexion, hyperextension-flexion, acceleration-deceleration, and whiplash injury refer to the mechanism of injury when the neck is forcibly thrown backward and forward, for example, during an auto collision.

"Whiplash" can be used offensively by equating the forces of hyperextension-hyperflexion motion with the force generated by the cracking of a large, long "bullwhip".

1. DIFFUSING THE WORD "WHIPLASH":

Jurors have negative feelings toward a plaintiff when the defense attorney calls his injury a "whiplash". The negative connotation of a "whiplash injury" must be diffused beginning with voir dire and ending with the closing argument. The following is a sample method of taking the sting out of a "whiplash injury."

VOIR DIRE:

Q: Has anyone injured his/her neck before?

A: Mrs Jones: I have.

Q: Did anyone call your injury a "whiplash?"

A: Yes.

Q: Were you offended?

A: Yes, because my neck really hurt.

CLOSING ARGUMENT (Plaintiff's Rebuttal):

"Whiplash!. . . .Whiplash!. The defense attorney has called this injury a "whiplash" -- not once -- but seven times during his closing argument. He is trying to plant the "seeds of prejudice" in your mind. Is this fair to Mr. Plaintiff? . . . ."

E. EXAMINATION FINDINGS OF HYPEREXTENSION-  
HYPERFLEXION INJURIES:

1. PLAIN X-RAY FINDINGS:

(a) Loss (Straightening) of the Normal Lordotic Curve:

(i) The Three Curves of the Spine - The vertebrae are not "stacked" in a straight line, one on top of the other. If they were, motion would be painful and limited. As a result, our spine (vertebral column) is curved in three places:

- (1) The Cervical Spine (Neck): The cervical curve is convex.
- (2) The Thoracic Spine (Mid-Back): The curve in the thoracic spine is with the convexity backwards.
- (3) The Lumbar Spine (Low Back): The curve in the lumbar spine is convex. The convexing curve of the low back balances the convexity curve of the neck.

The physicians call the normal curvature of the spine lordosis. Abnormal curvature of the spine is termed "loss of lordosis." For example, if the normal cervical and lumbar curves are abnormal, physicians refer to this condition as "loss of cervical lordosis" and "loss of lumbar lordosis", respectively.

Figure 39a shows the normal curves of the spine.

(ii) Paraspinal Muscle Spasm Causes Loss

(Straightening) of the Lordotic Curve - The

paraspinal (paraspinous) muscles are attached to the spine on top of the spinal ligaments. The paraspinal muscles fit "snugly" in the three curves of the spine, supporting the spine and assisting with movement.

Strain and sprain injuries result in stretching and tearing of ligaments and muscles. Paraspinal muscle spasms results - providing protective splinting to the injured area.

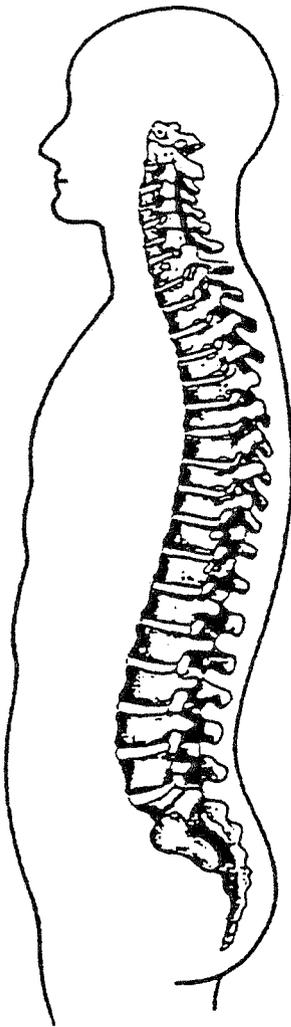
The posterior (back) paraspinal muscles in the neck and low back are stronger than the anterior (front) paraspinal muscles. Spasm of the posterior paraspinal muscles pulls the injured area of the spine straightening the normal lordotic curves in the neck and low back. A physician can demonstrate straightening of the normal lordotic curve by holding a ruler to the front and back margins of the vertebrae showing "straightening of the lordotic curve." An effective presentation to the jury can be made if the physician compares the abnormal x-rays with x-rays showing the normal lordotic curve. (Presumably from another patient or from the same patient when the acute phase of muscle spasm subsides).

The straightening of the lordotic curve is a positive (+) finding resulting from paraspinal muscle spasm.

(iii) Comparison of Normal and Straightened

Lordotic Curve - Figure 39A shows the normal curves of the spine. Figure 39B shows normal lumbar paraspinal (paraspinous) muscles with a normal lumbar lordotic curve (lumbar

lordosis). Figure 39C shows the posterior lumbar paraspinal muscles in spasm causing straightening of the lumbar lordotic curve (loss of lumbar lordosis).



Normal Curvature

FIGURE 39A

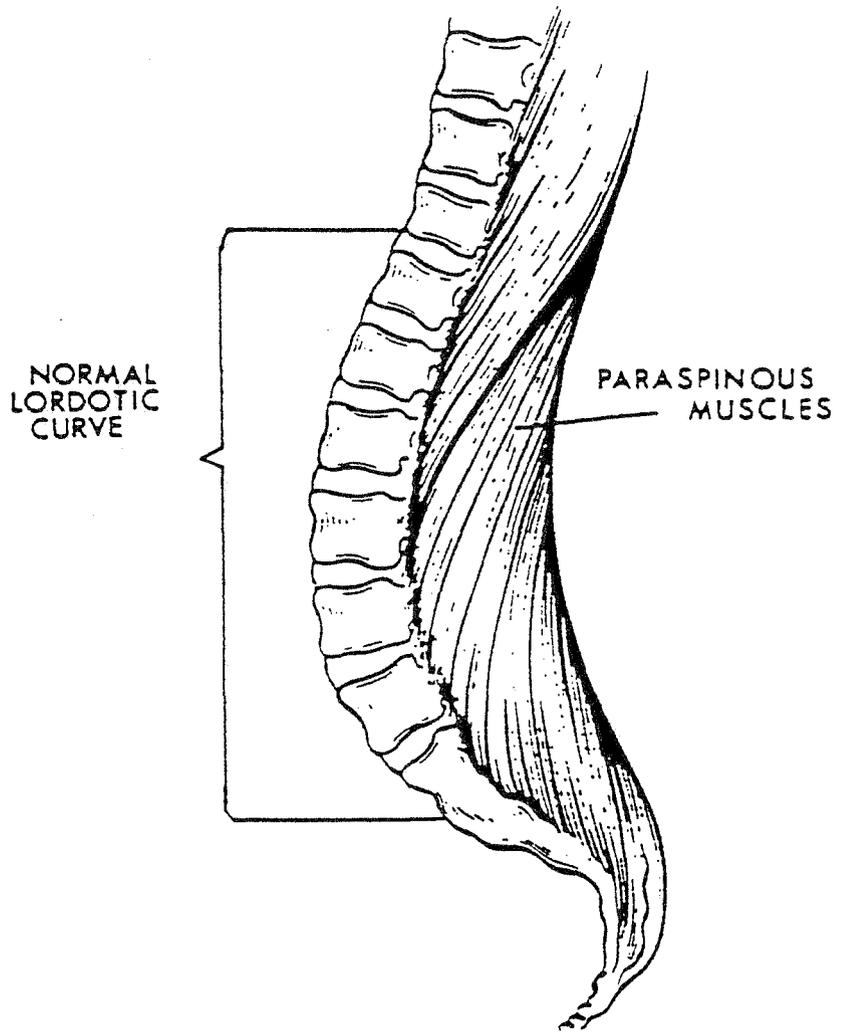
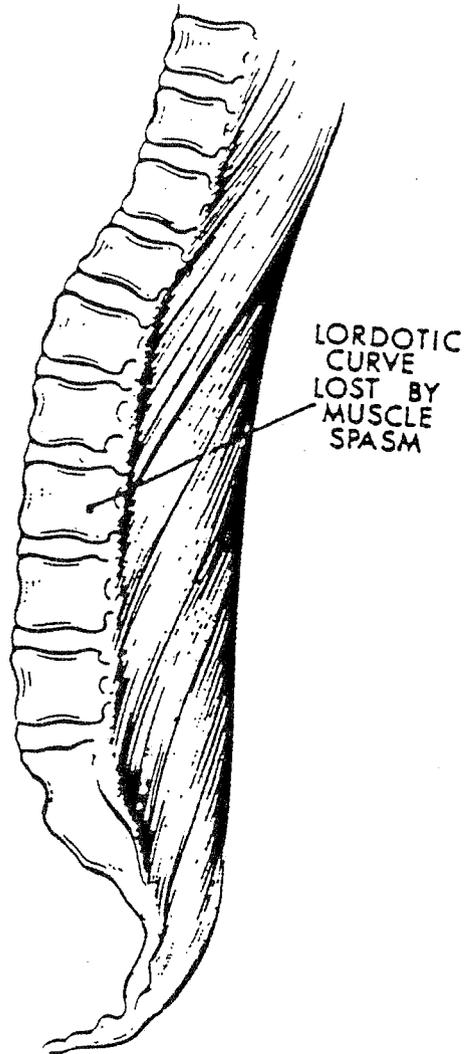


FIGURE 39B

©See p.1



©See p.1

FIGURE 39C

(b) Reversal of the Lordotic Curve:

Severe paraspinal muscle spasm not only straightens the lordotic curves, but goes a step further -- reverses the curve in the opposite direction. The convex curve in the neck and back is reversed.

(c) False Positive (+) Results:

(i) Routine Spinal X-ray Series - The routine spinal x-ray series consists of (1) A-P (anterior - posterior) views (front to back view); (2) Right and left oblique views; and (3) Lateral views (side views).

Each of the three standard "views" can be taken from three positions: (1) the neutral position; (2) the flexion position with the head and neck flexed toward the chest; and (3) the extension position -- the head and neck are extended backward.

(ii) Positioning of the Neck Causes False Positive - Lateral views of spine show straightening of the lordotic curve. As noted above, the lateral views are taken in the neutral, flexion and extension positions.

In the flexion position (with the head and neck flexed toward the chest), the normal lordotic curve in the neck is reduced, but does not completely straighten. Exception: Positioning, a patient with a long slender neck, for a lateral view of the spine, in the flexion position can itself result in loss of normal lordotic curvature. However, lateral x-rays taken in the neutral and extension position (head backwards) will not result in straightening of the lordotic curve by merely positioning the patient on the x-ray table.

Practice Note: The plaintiff should obtain all three lateral views of cervical spine x-rays. Loss of the normal lordotic curve in the flexion position could be the result of positioning a patient with a slender long neck. However, loss of the lordotic curve in the other

two views (neutral and extension) will prove that the loss of lordosis is as a result of muscle spasm, not merely positioning the patient on the x-ray table as may be the case with the lateral x-ray in the flexion position only.

(d) False Negative (-) Results:

Loss (straightening) of the normal lordotic curve of the spine is the result of paraspinal muscle spasm. If a patient is taking muscle relaxant medication, a false negative (-) result may occur.

(e) The Negative X-Ray:

Except for straightening of the lordotic curve, plain x-rays are generally negative in hyperextension-hyperflexion soft tissue injury cases. X-rays are designed to show "bone", not "soft tissues". The spinal ligaments, paraspinal muscles, intervertebral discs, nerve roots, spinal nerves, blood vessels, and sympathetic nerves -- all types of soft tissue -- will not show up on plain x-rays.

**Practice Note:** The positive use of the negative x-ray. The plaintiff's attorney should vividly demonstrate to the jury that plain x-rays do not show damage to soft tissues by having the medical expert draw on the x-ray itself the soft tissues, such as the spinal ligaments, paraspinal muscles, the intervertebral discs, the blood vessels, including the vertebral artery, the nerve roots and spinal nerves, as well as the sympathetic nerves. The jury must be shown, on the x-ray, the vulnerability of the these soft tissues to injury, with a forced hyperextension-hyperflexion motion. The negative x-ray thus becomes a positive plaintiff's exhibit. An excellent example of this courtroom technique is found in Houts and Marmor, Proving Medical Diagnosis and Prognosis, Vol. 3, Section 61.14 entitled, "Redirect Examination of Plaintiff's Medical Witness Who Reported 'Negative X-rays' of Plaintiff".

2. PHYSICAL EXAMINATION:

The physical examination includes motor reflex and sensory testing as discussed previously. In addition, the physician is looking for muscle spasm and decreased range of motion, particularly with soft tissue injuries.

(a) Muscle Spasm:

The physician palpates the patient's muscles for evidence of spasm. (Palpation = feeling with the hands to make a diagnosis). The finding of muscle spasm is the classic sign for acute (recent) strain and sprain injuries to the neck and back. As noted, a spasm is the body's involuntary response to protect an injured area from further pain. Since movement will result in increased pain, the purpose of spasm is immobilization -- splinting of the injured area. A person with muscle spasm is stiff and has difficulty moving the injured area.

Figure 40 illustrates palpation (feeling with the hands) for muscle spasm.

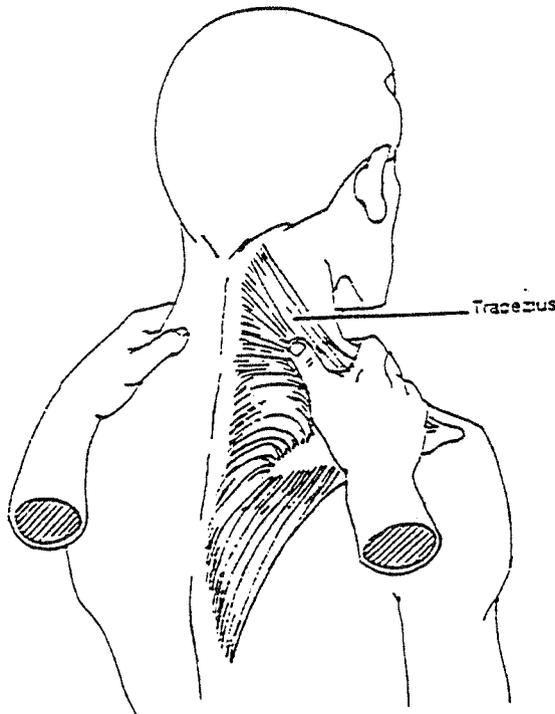


FIGURE 40

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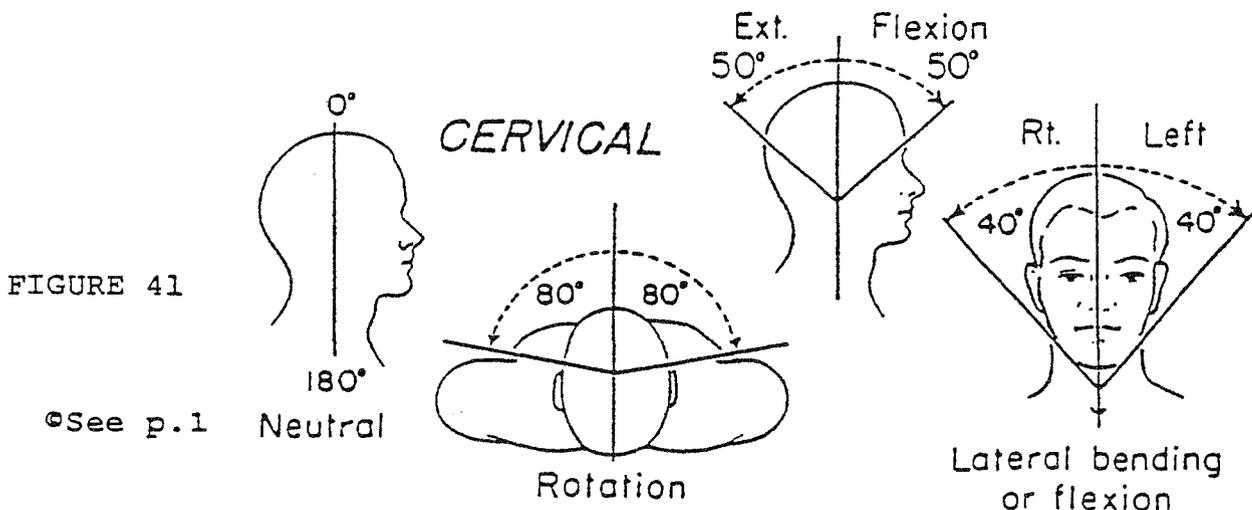
(i) Range of Motion (ROM) Examination - The result of muscle spasm is stiffness and decreased movement. For example, spasm of the cervical muscles will limit motion of the head and neck.

During physical examination, the physician observes and records the patient's limited range of motion when asked to move in certain directions. The patient often experiences pain while performing the motion testing, especially "at the extremes of motion."

With active range of motion the patient is the "active mover" -- performing the motions himself through the request of the physician. With passive range of motion the patient is the "passive mover" -- the physician is using his hands to perform all movements. Passive range of motion confirms the patient's limitation of range of motion.

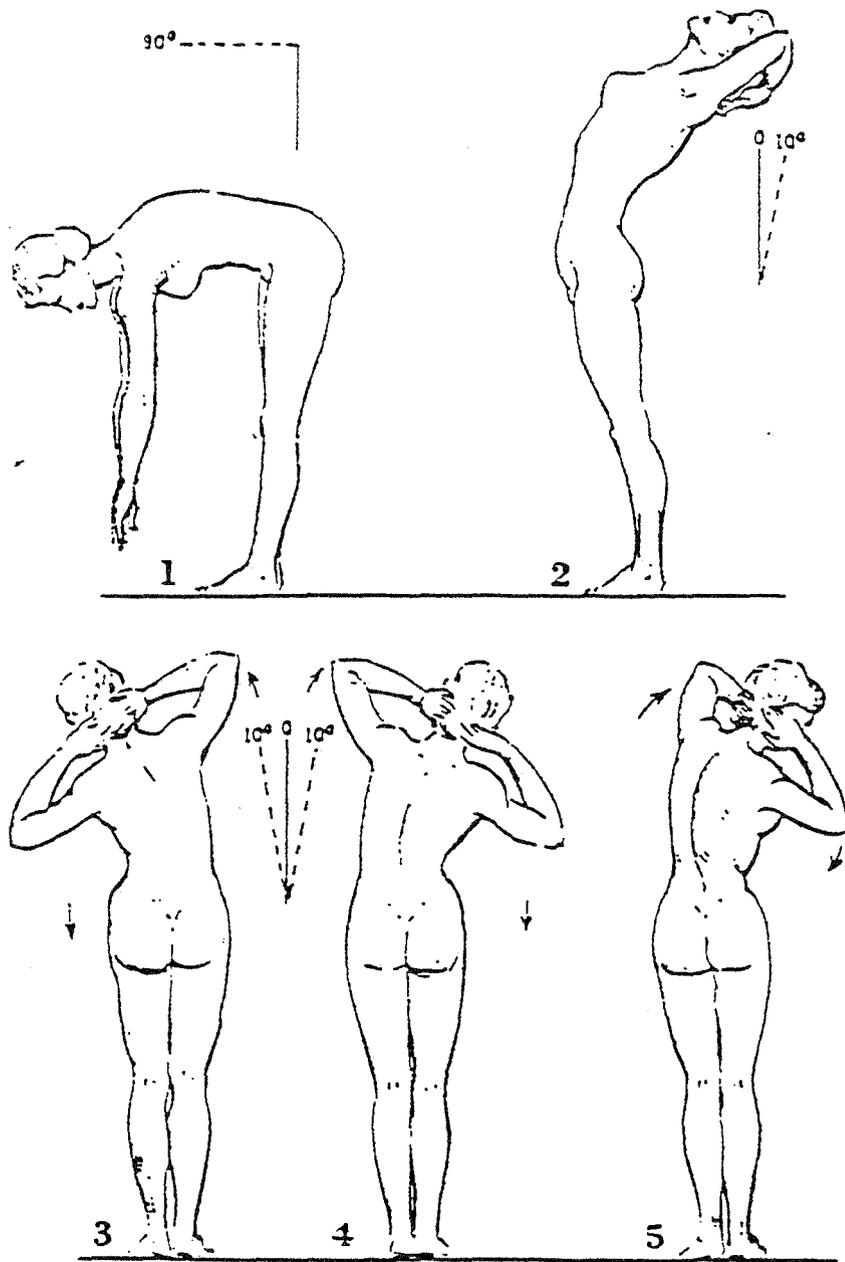
The patient's range of motion is recorded in degrees, and is compared with the normal range of motion for each movement.

Figure 41 shows the normal range of motion for neck movements and Figure 42 shows the normal range of motion for low back movements. A range of motion test is positive (+) if the angle of movement is less than the average range.



Average range of joint motions in the cervical spine (modified from Cave and Roberts).

## Range of Motion



1. Flexion, or forward, bending of the lumbar spine. The average normal flexion is about 90 degrees. 2. Extension, or backward bending, of the lumbar spine is also recorded in the range of back motion. The average extension is about 10 degrees. 3 & 4: Lateral, or side bending is measured in the lumbar spine. The average lateral bend is about 10 degrees. 5. Rotation, or twisting the spine, is measured and recorded as the degree of shoulder motion. The average rotation averages about 90 degrees.

©See p.1

FIGURE 42

### 3. SUBJECTIVE COMPLAINTS vs. OBJECTIVE FINDINGS:

The first part of every medical examination consists of the "history" where the patient describes to the doctor his complaints of pain. Based upon the patient's symptoms, findings on physical examination and results of appropriate tests, the physician makes a diagnosis. The history is important in making the diagnosis -- it is so important that medical students are taught the maxim: "listen to the patient, he is telling you the diagnosis."

The plaintiff's complaints of pain, provided in the history, are termed subjective complaints. Objective findings are those findings actually observed by the physician, such as feeling muscle spasm with his own hands.

Usually, after the initial acute phase, the objective findings will be minimal.

#### (a) Cross-examination of the IME Doctor:

The cornerstone of the defense of a soft tissue injury case is to use the "IME doctor" to point out to the jury the difference between subjective complaints and objective findings.

The following is a sample cross-examination "debunking" an IME doctor on subjective complaints vs. objective findings:

Q: Doctor, a patient's history is important in arriving at a diagnosis. Isn't that true?

A: Yes.

Q: Doctor, a history consists of the patient telling you his subjective complaints?

A: Yes.

Q: In your own practice do you rely upon the patient's history in forming your diagnosis?

A: Yes.

Q: Doctor, assume that a patient gave you a history of severe crushing chest pain radiating into the arm after shoveling snow. This patient could be suffering from an acute heart attack. Isn't that true?

A: Yes.

Q: Doctor, isn't this a medical emergency?

A: Yes.

Q: Doctor, assume that I just cut my finger on a piece of paper -- a paper cut. This would be an objective finding wouldn't it?

A: Yes.

Q: But the chest pain - that would be a subjective complaint?

A: Yes.

Q: Requiring immediate admission to the emergency room?

A: Yes.

Q: Thank you doctor, I have no further questions.

4. SOFT TISSUE INJURIES RESULT IN PERMANENT RESIDUALS:

The medical literature verifies that soft tissue injuries leave permanent residuals. For example, Drs. Gargan and Bannister at the University of Bristol followed 43 patients with soft tissue neck injuries for 8 to 12 years after the initial injury. Eighty-eight percent of the patient's had been in rear-end auto collisions. The results verifying permanent residuals are reported in Gargan and Bannister, "Long Term Prognosis of Soft Tissue Injuries of the Neck," J. Bone and Joint Surgery, Vol. 72-B, No.

5 Sept. 1990:

The Severity of Symptoms: Patients were subdivided into four groups on the basis of their symptoms. Group A (12%) were free of any discomfort and considered that they had made a complete recovery from their accident. (88% had not fully recovered). Group B (48%) were left with mild residuals which did not interfere with their work or leisure activities. Group C (28% complained of intrusive symptoms which handicapped work and leisure activities and caused them to seek relief by frequent intermittent use of analgesic, orthoses or physiotherapy. Group D (12%) suffered from severe problems and had lost their jobs, relied continually on orthoses or analgesics, and had undergone repeated medical consultations.

Dr. Gargan's and Dr. Bannister's long term study showed 28% of the patients had residual symptoms which were intrusive, and 12% of the patients had severe residual symptoms 10.8 years after the initial auto collision.

In an NIH sponsored study, Dr. Mason Hohl followed 146 patients, 5 years after their auto collision. No patient had pre-existing osteoarthritis. Symptomatic recovery (no residual problems) occurred only in 57% of patients (43% still had residual symptoms 5 years after the collision). Dr. Hohl's article appeared in the December 1974 issue of The Journal of Bone and Joint Surgery.

Medical school professors, Drs. Carroll, et al., "J. Musculoskeletal Medicine", June 1992, p. 97-114, reported similar findings of long-term permanent residuals in patients involved in auto collisions:

"...Long-term studies show that aches and pains with no evident physical cause persist in 20% - 45% of patients with significant whiplash injuries. Few of these patients have radicular pain. Roentgenographic (x-ray) studies show that degenerative problems develop after injury in 39% of patients. By comparison, degenerative changes develop over a comparable time in only 6% of the general population over age 30. Thus, it would seem apparent that whiplash injuries predispose patients to cervical degenerative osteoarthritis.

Hohl found that the following early symptoms or signs were associated with long-term disability: persistent upper extremity pain and numbness; interscapular pain; a sharp cervical curve reversal; and one level of localized restricted cervical motion. Men had a higher incidence of symptomatic recovery. Degenerative changes were more common in patients who had lost consciousness. A most important finding was that whether or not litigation proceeded did not appear to influence recovery among the patients studied." (emphasis added).

5. **PERMANENT RESIDUALS FROM SOFT TISSUE INJURIES REMAIN AFTER SETTLEMENT:**

Studies, in the medical literature, point out that patients with soft tissue injuries continue to experience permanent symptoms even after an injury case settles.

For example, Drs. Gargan and Bannister, in their article cited above, reviewed the prior medical literature and their own studies of patients 8 to 12 years after the date of injury and concluded that permanent residuals of soft tissues remain even after settlement, stating:

"McNab (1964) and Norris and Watt (1983) recorded that symptoms remained unchanged in those whose claims had been settled. The fact that symptoms do not resolve even after 10 years supports the conclusion of these authors."

Dr. Hohl, in his article in The Journal of Bone and Joint Surgery, December, 1974, found that 50% of his patients who made no claim, were still experiencing residual symptoms five years after their auto collision, stating: "44 of the 146 patients in the series had no legal claim.... Only 50% of these 44 patients became asymptomatic."

In addition, Drs. Carroll, et. al., "J. Musculoskeletal Medicine", June 1992, p. 97-114, reported similar results stating:

"... Research indicates that neither litigation nor personality disorder has any great bearing on the duration or intensity of a whiplash victims' symptoms."

6. HOW TO GET FAVORABLE MEDICAL ARTICLES INTO EVIDENCE:

Federal Rule of Evidence 803(18) entitled, "Learned Treatises" and Virginia Code §8.01-401.1, third paragraph\*, (1994 amendment) provide the answer:

"To the extent called to the attention of an expert witness upon cross-examination or relied upon by the expert witness in direct examination, statements contained in published treatises, periodicals or pamphlets on a subject of history, medicine or other science or art, established as a reliable authority by testimony or by stipulation shall not be excluded as hearsay. If admitted, the statements may be read into evidence but may not be received as exhibits. If the statements are to be introduced through an expert witness upon direct examination, copies of the statements shall be provided to opposing parties thirty days prior to trial unless otherwise ordered by the court." (emphasis added).

The last sentence requiring 30 days notice is not present in Federal Rule of Evidence 803(18). In addition, the Federal Rule uses slightly different wording to achieve the same result as Code §8.01-401.1, at line 8 above:

"...the testimony or admission of the witness or by other expert testimony or by judicial notice..."

The Virginia Supreme Court, in Weinberg v. Given, 252 Va. 221, 476 S.E.2d 502 (1996), held Virginia Code §401.1, third paragraph, "permits the hearsay content of such articles to be read into the record as substantive evidence..."

Following Weinberg v. Given, an expert witness can read favorable statements from medical articles directly into evidence

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\*A Guide to Evidence in Virginia, Rule 707(a) entitled, "Use of Learned Treatises with Experts"

provided the expert states (1) the article is reliable; and (2) the article is relied upon by the expert (the expert agrees with the statements in the article).

The Supreme Court, in Weinberg v. Given, 252 Va. 221, at page 223, approved the following foundation for the admissibility of statements from medical articles:

"Dr. Partridge also identified several articles that he believed were reliable and authoritative. For example, Dr. Partridge gave the following testimony:

"Q: And Doctor, are you familiar with the article entitled 'Oral Contraception: Past, Present and Future Perspectives' by Daniel Mishell? An again, this would be in the International Journal of Fertility, 1991. Are you familiar with that, sir?

A: Yes.

Q. Is that a reliable authority in the field as of 1991 on this issue?

A. Yes.

Q. And Doctor, going right to the last page, to the conclusion, 'Today 85 percent of prescriptions of [oral contraception] written in the United States specify formulations containing 30 to 35 micrograms of ethinyl estradiol, the lowest possible dose required to achieve optimal contraceptive [efficacy] with minimal risk of adverse effects. These new compounds do not appear to have any adverse cardiovascular effects and can be safely used by healthy, nonsmoking, premenopausal women until age 45 or older.' Did I read that correctly, sir?

A: You Did.

Q. Did you agree with that?

A. Yes."

# PART THREE

## FIBROMYALGIA

### A. INTRODUCTION

Fibromyalgia, (also called fibromyalgia syndrome - FMS, FS or FM), is a medically recognized soft tissue rheumatic condition involving pain in fibrous tissue and muscle. Fibrous tissue is tissue that contains fibers such as (1) fascia, (2) tough tendons that connect muscle to bone, and (3) ligaments which connect bone to bone. Fascia is a sheath that encloses and holds muscle together - like a strong "baggie".

The term fibromyalgia literally means pain from fibrous tissue and muscle ("fibro" referring to fibrous tissue; "my" referring to muscle; and "algia" meaning pain).

Myalgia is different from fibromyalgia since myalgia involves only simple muscle pain. Myositis is also different from fibromyalgia since it involves inflammation of muscle ("myo" meaning muscle; "itis" meaning inflammation).

The term "fibrositis" had been used interchangeably with "fibromyalgia" by physicians. In recent years, the term "fibrositis" has lost favor since studies have shown that patients suffering from fibromyalgia do not exhibit muscle inflammation.

A related condition is "fibromyositis" which does involve inflammation of the fibrous tissue and muscle. Fibromyositis is characterized by large hard nodules in the muscle, which can be felt when the physician palpates a patient's muscle with his hands. Patients with fibromyalgia do not have hard nodules but tender points.

Another condition related to fibromyalgia is myofascial pain syndrome (MPS), which is characterized by trigger points in muscle which refer pain in classic directions to adjacent areas. In contrast, fibromyalgia is characterized, not by trigger points, but by localized tender points.

## **B. SYMPTOMS OF FIBROMYALGIA**

The symptoms of fibromyalgia include:

1. Pain -- Widespread deep aching muscular pain with 11 out of 18 characteristic tender points. Pain and stiffness are worse in the morning.
2. Fatigue -- Widespread fatigue equivalent to the severe fatigue of "chronic fatigue syndrome".
3. Sleep Disorder-- Interruption of deep sleep (level 4 sleep). Patients awoken unrested and fatigued.

## **C. THE AMERICAN COLLEGE OF RHEUMATOLOGY'S CRITERIA FOR DIAGNOSING FIBROMYALGIA**

In 1990, the American College of Rheumatology developed criteria for the diagnosis of fibromyalgia. This criteria was

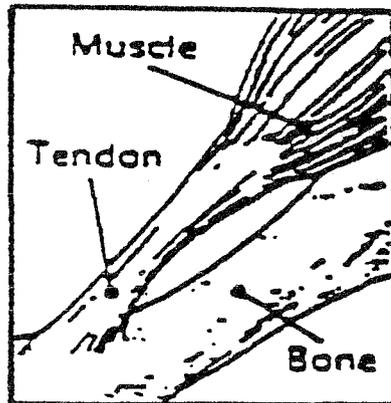
reported by Wolfe, et al., The American College of Rheumatology, 1990 Criteria for the Classification of Fibromyalgia, 33 Arth. & Rheum. pp. 160-172 (Feb. 1990).

The American College of Rheumatology gives its stamp of approval to a diagnosis of fibromyalgia if the following criteria are found:

1. Widespread pain in all four quadrants of the body for at least three months. Four-quadrant pain is defined as (a) pain in the left side of the body and in the right side of the body; and (b) pain above the waist and below the waist; and (c) axial skeletal pain (cervical spine, anterior chest thoracic spine or lumbosacral spine). Shoulder and buttock pain is considered pain for each involved side and low back pain is considered lower segment pain; and
2. Pain in 11 of 18 designated tender point sites.

#### D. PHYSICAL EXAMINATION (THE 11 TENDER POINTS)

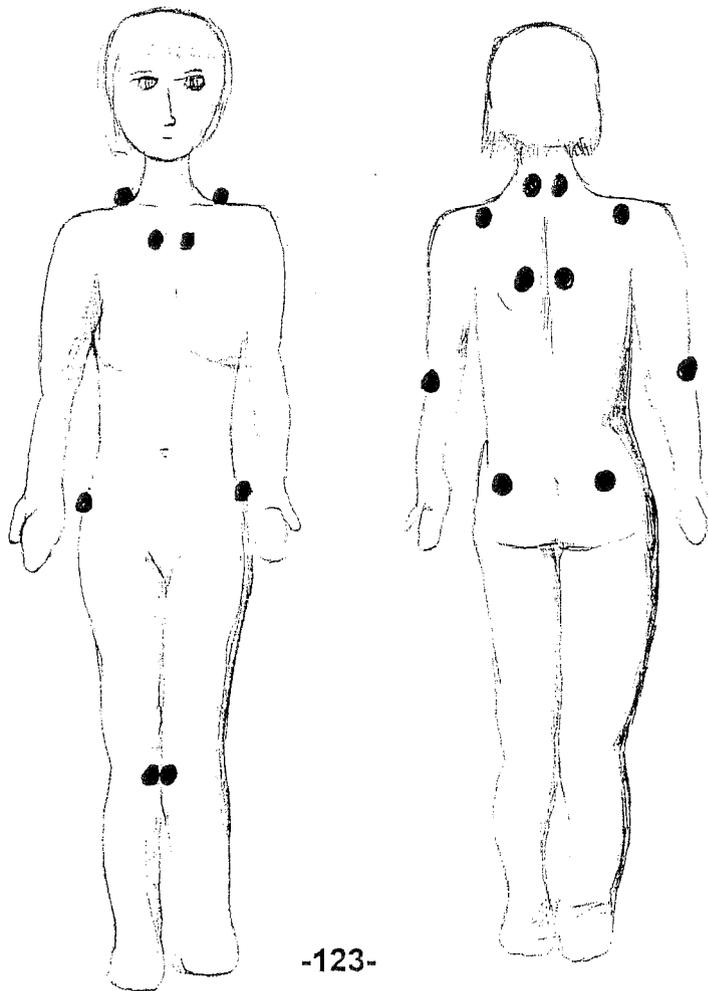
The key to diagnosing fibromyalgia, on physical examination, is finding 11 out of 18 classic tender points. Tender points are usually found in areas where tendons connect muscle to bone as illustrated below:



When a physician presses his thumb on a tender point, the patient usually will "flinch" or "jump" due to the pain. The 18 tender points is the only finding on physical exam confirming the diagnosis of fibromyalgia.

### E. LOCATION OF THE 18 TENDER POINTS

The following illustration shows the anatomic location of the 18 tender points according to The American College of Rheumatology's 1990 classification for fibromyalgia.



## F. LAB TESTS

There is no laboratory test to aid in the diagnosis of fibromyalgia. All lab tests are normal. However, normal lab tests can distinguish fibromyalgia from other conditions with similar symptoms. For example, rheumatoid arthritic conditions yield positive results from simple blood tests, such as a (+) erythrocyte sedimentation rate (ESR).

## G. CAUSATION

H.A. Smythe, a leading authority on fibromyalgia, in his textbook, Arthritis and Allied Conditions, p. 1249 (11th Ed. 1989) states that trauma may cause fibromyalgia:

"Trauma may initiate a chronic fibrositis (an old synonym for fibromyalgia) syndrome, with a frequency of 24% in one study of 95 patients. In addition, "injury" to the neck was described in 40% before the onset of symptoms and to the low back in 31%, significantly more often than in community controls (14 and 19%, respectively). This sequence is of significant medicolegal importance. In this series, the diagnosis was based on objective criteria (tender points) rather than subjective criteria. The special association with neck injury is notable because of the interaction between the cervical spine, nocturnal pain, and sleep disturbance. The chronicity of complaints after seemingly minor injury may be explained by re-injury of the affected site during sleep. Saskin, et al. "compared 11 fibrositis patients and 11 with "post-accident pain syndrome" and found identifying diffuse pain, exhaustion, point tenderness, personality scores, and sleep EEG disturbance."

Greenfield, et al., in their article "Reactive Fibromyalgia Syndrome", 35 Arth. & Rheum., pp. 678-681 (June 1992), found that patients who developed reactive fibromyalgia (as a reaction to a prior precipitating event, such as trauma from an auto collision, elective surgery, or a non-traumatic medical illness) had greater disability than patients who developed primary fibromyalgia (fibromyalgia caused without a prior precipitating event).

Post-Traumatic Fibromyalgia Syndrome (PTFS) is well recognized in the medical literature. For example, Thomas J. Romano, M.D., Ph.D., a Rheumatologist, teaching at the W. Virginia University School of Medicine, in his article, "Clinical Experiences With Post-Traumatic Fibromyalgia Syndrome", W. Va. Medical Journal, Vol. 86, No. 5, pp. 198-202, (May 1990) defines PTFS as, "It occurs in the setting of an acute injury, after which signs and symptoms of fibromyalgia syndrome (FS) appear. ...Often, the diagnosis is delayed. In this series the mean length of time from the date of injury to date of diagnosis of PTFS was 17.3 months..."

In addition, G.W. Waylonis et al., from the Department of Physical Medicine and Rehabilitation at Ohio State University, in their article, "Post-Traumatic Fibromyalgia", Am. J. Phys. Med. Rehabil., Vol. 73 (No. 6) pp. 403-412 (Nov.-Dec. 1994) described a 10 year study of 176 patients diagnosed with post-traumatic fibromyalgia, 60.7% noting symptoms after a motor vehicle accident. 85% of patients continued to have significant symptoms.

The often reported association between neck injuries caused by auto collisions and the onset of post-traumatic fibromyalgia

syndrome (PTFS) was validated by Buskila et al., in their article, "Increased Rates of Fibromyalgia Following Cervical Spine Injury: A Controlled Study of Traumatic Injury", Arthritis and Rheumatism, Vol. 39, pp. 446-452 (1997). They reported that 21.6% of patients with soft-tissue neck injuries (90% having classic whiplash) developed fibromyalgia versus 1.7% of patients with leg fractures.

Fibromyalgia can be caused without trauma, such as by a viral or bacterial infection or develop as secondary fibromyalgia - "on top of" another condition, such as rheumatoid arthritis or hypothyroidism. This latter type of fibromyalgia is called secondary fibromyalgia since it develops secondarily after the patient is already weakened by the first condition.

## **H. TREATMENT**

Fibromyalgia is a chronic, often debilitating, condition with no specific cure. Treatment consists of improving the sleep disorder and relieving pain. Low doses of tricyclic anti-depressants, such as Elavil, have improved the quality of sleep in fibromyalgia patients. Similarly, the muscle relaxant Flexeril has been found to improve the quality of sleep and reduce pain. In addition, nonsteroidal anti-inflammatory drugs (NSAIDs), have been prescribed to help alleviate the pain of fibromyalgia. Physical therapy and "tender pain injections" with lidocaine, an anesthetic, have also been employed to help relieve pain, but only provides temporary relief. Gentle, daily, low-impact aerobic exercise has also proved helpful.

## **PART FOUR**

### **SUMMARY OF COMMONLY USED DIAGNOSTIC TESTS**

#### **A. PLAIN X-RAYS:**

X-rays are also called roentgenograms named after Dr. Roentgen who discovered them. X-rays were previously discussed at pp. 105-110. X-rays only show injury to bone; however, straightening of the normal lordotic curve in the spine can be seen.

Plain x-rays help diagnose spondylolisthesis (slippage of one vertebrae on top of another); spinal stenosis (narrowing of the spinal canal); and degenerative disc disease, including osteophytic spurs and disc space narrowing (the decreased disc space between the vertebrae resulting from "drying out" of the disc).

#### **B. DISCOGRAPHY:**

A discogram is a procedure where a contrast dye is injected into the center of the disc. The contrast dye allows the disc to be seen on an x-ray. The x-ray procedure is called "fluoroscopy". Often a CT scan is obtained while the contrast dye is still present in the disc for greater resolution. X-ray and/or CT films are produced which visualize the disc.

If the injection temporarily reproduces the patient's pain, the discogram is said to be (+) positive. The discogram allows a physician to pinpoint which disc is the cause of the patient's pain.

A risk of the discogram is infection within the disc from the injection itself.

### C. NEEDLE EMG AND NCV TESTING:

There are two types of electromyographic testing: (1) needle EMG; and (2) surface EMG. Electromyography is derived from the words: electro meaning electricity; myo meaning muscle; graphy meaning writing.

Needle EMG testing measures the electrical activity of muscles (myotome) supplied by a specific spinal nerve root. It is an indirect method of diagnosing injury to a spinal nerve by measuring the electrical activity of the muscle innervated by the nerve root. If the nerve is damaged "upstream" at the nerve root level, the muscle will give off an abnormal pattern of electrical potential called denervation fibrillation. Denervation fibrillation occurs three weeks after injury to the nerve root. Accordingly, the test should not be performed within three weeks of injury.

A needle electrode is inserted through the skin into the muscle and connected to the electromyograph machine which picks-up electrical potential in the muscle. (The needle is long, and the procedure is painful).

The EMG produces a false negative in 20% of patients who have nerve root injury.

Nerve Conduction Velocity (NCV) testing is done at the same time as EMG testing. Nerve conduction velocity measures the speed of the nerve impulse. An injured spinal nerve will produce a nerve impulse at a slower speed, compared to known values.

#### **D. SURFACE EMG TESTING:**

Surface EMG is a test measuring the tension of a muscle. Surface electrodes, (instead of needles) are placed on top of the skin over the muscle to be tested. Electrical signals from the muscle produce a digital read-out. The examiner compares the digital read-out from the test with known normal values. A positive test indicates a state of muscle tension and contraction.

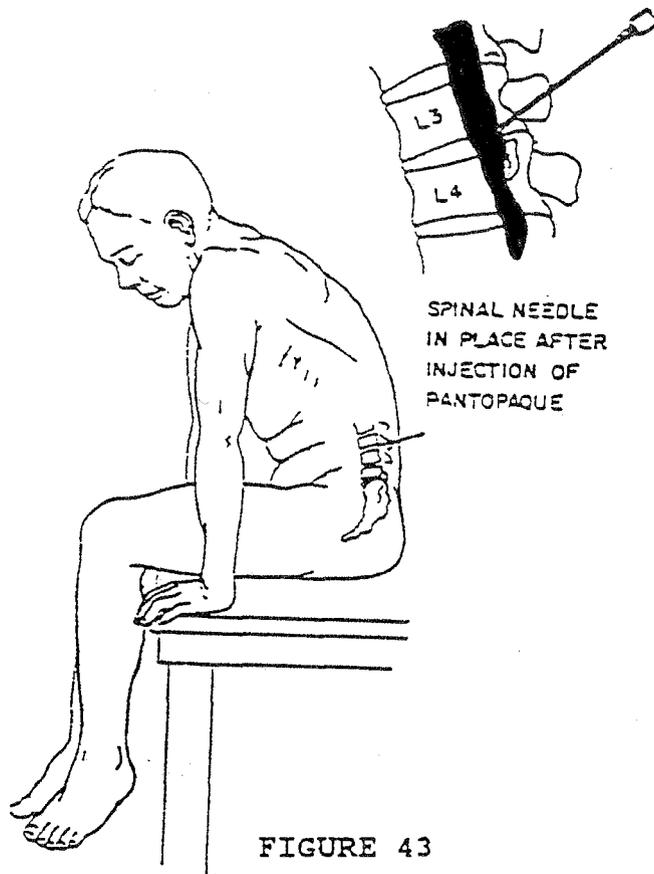
#### **E. MYELOGRAM:**

Myelography (myelo = spinal cord; graphy = writing) involves injecting a contrast dye into the subarachnoid space of the spinal cord. The dye mixes with spinal fluid and an outline of the spinal cord is seen on x-ray. If the nerve sleeve surrounding a spinal nerve root is compressed, for example, by a herniated disc or a large osteophyte, the nerve sleeve will be absent on a myelogram x-ray. This is called a "filling defect".

A patient is usually admitted into the hospital overnight for a myelogram. Side effects include severe headache, nausea, and vomiting.

Two types of contrast dyes have been used in the myelogram procedure: (1) an oil-base dye - Pantopaque (rarely used today), which is withdrawn following the procedure; and (2) a water-soluble dye - Metrizamide which is absorbed by the body following the procedure.

Figure 43 shows placement of the myelogram needle and Figure 44 shows the results of myelogram testing.



©See p.1

FIGURE 43

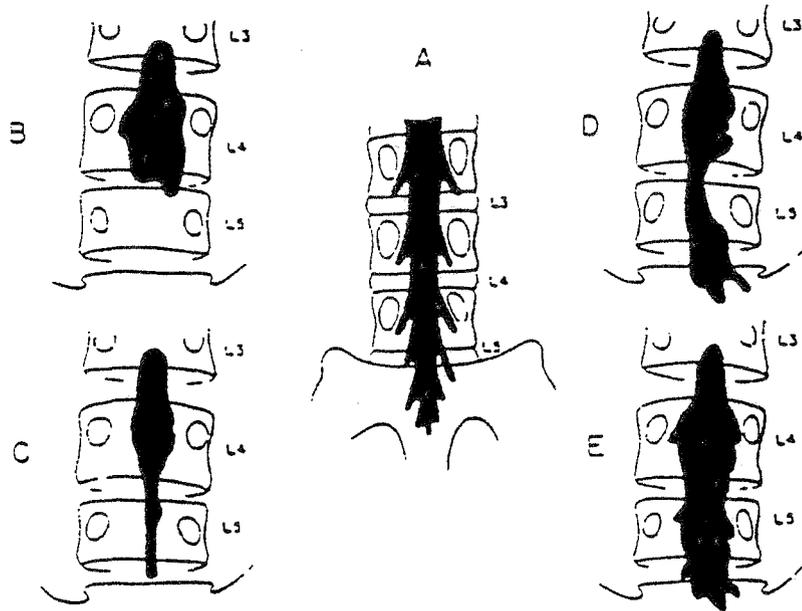


FIGURE 44

The myelogram is performed by injecting radiopaque (dense) dye through a needle into the spinal fluid contained in the subarachnoid space. An X-ray of this dense column of dye shows the outline of the cord and nerve sleeves as they come off the dura. An indentation of the column of dye may be caused by a disk or tumor. (A) The normal myelogram showing the outline of the dural sac and the nerve root sleeves leaving the canal. (B) A complete block of the spinal canal at L4-L5. (C) Narrowing of the disk space at L4-L5. (D) A large defect at L4-L5. (E) Blunting and depression of the nerve root at L5-S1.

©See p.1

## **F. CAT SCAN:**

A CAT (also abbrev. CT scan) is computerized axial tomography. An x-ray beam is directed through the body and the information is processed through a computer producing pictures of the internal structure of the body.

CT Myelogram is a procedure where a CAT scan is performed following a myelogram. The contrast myelogram dye is already present in the patient's blood stream, giving enhanced images of the body's internal structure.

The CAT scan does not show stretching and tearing of soft tissues from forced hyperflexion - hyperextension injuries.

## **G. MRI:**

MRI is an abbreviation of Magnetic Resonance Imaging which uses magnetic fields instead of x-rays to produce multi-planar images of internal body structures. The image shown on the MRI film is obtained by measuring differences in the water content (hydrogen ion) density in various tissues.

Gadolinium, a paramagnetic contrast dye is often injected into the patient to provide a better MRI picture.

The MRI has been found to visualize herniated discs better than CAT scan testing; whereas CAT scan testing shows better detail of bone.

An MRI should not be performed on patients who wear pacemakers or who have metal plates in their body since this would adversely effect the magnetic field.

Like the CAT scan, the MRI does not show stretching and tearing of soft tissues from forced hyperflexion - hyperextension injuries.

# PART FIVE

## DRUGS USED TO TREAT NECK AND BACK INJURIES

### INTRODUCTION

To treat neck and back injuries, physicians often prescribe pain killers (analgesics); anti-inflammatory drugs to decrease inflammation; muscle relaxants to decrease muscle spasm; anti-anxiety drugs to treat anxiety; anti-depressants to treat pain and depression resulting from the injury; and sleeping pills (hypnotics) to help clients awakened by pain to fall asleep.

### I. CLASSIFICATION OF DRUGS

#### A. BASED UPON ACTION

Drugs are classified based upon what they do -- their action in the body. Some major classifications are: Analgesics, Antibiotics, Antidiabetic Agents, Anti-Inflammatories, Anti-Depressants, Muscle Relaxants, to name a few.

#### B. BASED UPON GROUP FORMULA

Drugs are further classified based upon their group chemical formula. A group chemical formula is the basic chemical nucleus shared by all drugs within the group which is responsible for drug activity. Although all drugs within the group

have the same nucleus, each differs slightly by having a different substituent on the nucleus which "fine tunes" the drugs effects. For example, the "tricyclic anti-depressants" have three cyclic rings of carbon atoms as part of their basic nucleus hence the name, "tricyclic anti-depressants". Two well known "tricyclic anti-depressants" (abbreviated TCA's) include amitriptyline (Elavil) and doxepin (Sinequan). A new generation of cyclic anti-depressants having four rings has been recently used to treat depression. This class of anti-depressants is called "tetracyclic." A well known "tetracyclic anti-depressant" is maprotiline (Ludiomil).

#### 1. DRUG NAMES

Each drug has four names:

1. A group name;
2. A chemical name;
3. A generic name; and
4. A trade name (brand name).

For example, the anti-depressant drug Elavil (a) has the group name "tricyclic anti-depressant" (TCA) since it contains the tricyclic chemical nucleus which is responsible for its anti-depressant properties; (b) has a long chemical name; (c) has a generic name, amitriptyline; and (d) has the trade name, Elavil. Similarly, the anti-depressant drug Ludiomil (a) has the group name "tetracyclic anti-depressant"; (b) a long chemical name; (c) the generic name maprotiline and (d) the trade name Ludiomil.

The manufacturer gives the drug its generic and trade names when the drug is first marketed. U.S. Patent protection lasts seventeen years. Once the patent expires other drug companies also market their brand of amitriptyline. Today, there are several trade named products of amitriptyline including Elavil (the first); Amitril; Emitrip; Endep. In addition, amitriptyline is also manufactured and sold by generic drug companies under its generic name, Amitriptyline. Generic drugs are usually less costly than the trade named version of the same drug. Pharmacists are allowed to substitute a cheaper generic brand for a trade named prescription drug, unless the physician writes on the prescription, "Dispense as written".

(a) How Drugs are Named

Manufacturers often choose a generic name (which "sticks" with the drug forever) based upon the drug's chemical formula. For example, the generic name for the anti-anxiety drug, Librium, is Chlor diaz epoxide, named after three substituent chemical groups in the molecule -- chlorine, diazo, and expoide. The trade name Librium was chosen to convey its anti-anxiety effect -- bringing the patient back to a state of equilibrium.

Another method for naming drugs is based upon the source of the drug itself. For example, Premarin, is the trade name for an estrogen replacement drug used to control menopausal symptoms. Its source is from pregnant mares urine, hence the name Premarin.

Lastly, some drugs are named as the manufacturer's "in house joke". For example, the trade name anti-depressant, Trilafon, is shorthand for "try laughing"!

## **II. ANALGESICS**

### **A. INTRODUCTION**

The term analgesic refers to a drug that relieves pain and is derived from the word "analgia" meaning absence of pain (from the Greek "an" meaning negative and "algos" meaning pain). Some analgesics use "algos" (pain) as part of its trade name, i.e. "Synalgos-DC" the trade name for the narcotic pain killer Dihydrocodeine.

Analgesics are classified as either narcotic or non-narcotic.

### **B. NARCOTIC ANALGESICS**

#### **1. SISTER MORPHINE, BROTHER CODEINE AND A LITTLE VINEGAR - ENTER THE MORPHINE NARCOTICS**

Opium is the oldest drug in history. Opium is the air dried juice from the opium poppy plant. The name Opium is derived from the Greek word opion - meaning "juice". The opium "juice" is analogous to "chemical soup" and contains the drugs Morphine and Codeine, in addition to two dozen other alkaloids. (Alkaloid is a generic term referring to alkaline organic chemicals derived from plants).

Codeine is methylmorphine - the chemical brother of Morphine (almost a molecular twin). Morphine is a stronger pain killer than Codeine, but Codeine is less addicting. Morphine, one of the strongest pain killers, is also a powerful sedative. Indeed, the name Morphine is derived from the Greek word for sleep.

Over seventy-five years ago organic chemists tried to synthesize a derivative of Morphine hoping to retain its pain killing properties, with less addictive qualities. They added acetic acid (common vinegar) to Morphine and came up with diacetylmorphine, which they expected to become a "heroic drug" in the treatment of pain, and named it Heroin. Cutting's Handbook of Pharmacology, 7th Ed. (1984), page 625, describes the properties of Heroin as, "More potent than Morphine and would be highly satisfactory if it were not also addicting; not permitted in the United States."

Codeine is found in prescription cough medicines and is also classified as an anti-tussive. (From the Latin "tussis", meaning cough).

(a) The Morphine Group

Today, Morphine and Codeine are synthesized in the laboratory rather than extracting the chemicals from the opium poppy. Morphine is prescribed in the form of its sulphate, salt. Codeine is widely prescribed in the prescription drug Tylenol with Codeine. The prescription product is sold as Tylenol #1; Tylenol #2; Tylenol #3;

and Tylenol #4. The higher number indicates a higher dosage of Codeine. Oxycodone, found in Percodan, Percocet, and Tylox, is a powerful pain reliever, ten times more potent, and more addicting than Codeine. The generic and trade names for the Morphine Narcotic Analgesics are set forth below.

(i) The Morphine Narcotic Analgesics

<u>GENERIC NAME</u>	<u>TRADE NAME</u>
Morphine Sulfate (M.S.)	----
Codeine	Tylenol with Codeine
HydroMorphone	Dilaudid
HydroCodone	Vicodin, Lorcet, Anexsia, Hycodan and Dicodid
Oxymorphone	Numorphan
Oxycodone	Percodan, Percocet and Tylox, Oxycontin
Dihydrocodeine	Synalgos-D.C., Compal
Levorphanol	Levodromoran
Butorphanol	Studol
Nalbuphine	Nubain
Pentazocine	Talwin

PRACTICE NOTE 1 - USING NARCOTIC PAIN-KILLERS TO MAKE THE JURY FEEL YOUR CLIENT'S PAIN ON VOIR DIRE: People associate narcotic pain-killers with extreme pain. Make the jury feel your client's pain with the following sample voir dire:

Question: "Bob's doctor prescribed Percodan, a narcotic pain-killer to help lessen Bob's pain. Have you or any members of your family ever been prescribed a narcotic, pain-killer?"

Answer: Juror 1. - "My son was given Percodan when he broke his arm".

**PRACTICE NOTE 2 - THE DOCTOR'S DIRECT EXAMINATION - OXYCODONE (PERCODAN, PERCOCET, TYLOX):**

Question: "Dr. Smith, what medicine did you prescribe to help lessen Bob's pain?"

Answer: I perscribed Percodan, a narcotic pain-killer.

Question: Dr. Smith, how strong a narcotic pain-killer is Percodan?

Answer: Percodan is ten times stronger than Codeine.

Question: Dr. Smith, is Codeine also a narcotic pain-killer?

Answer: Yes.

Question: Dr. Smith, why did you prescribe Percodan instead of Codeine?

Answer: Because Bob was in so much pain he needed a stronger narcotic pain-killer. Percodan is ten times stronger than Codeine.

**(b) The Methadone and Meperidine (Demerol) Group**

Methadone produces strong pain relief similar to Morphine but its withdrawal symptoms are more easily tolerated for those addicted to the drug. Meperidine (Demerol) is between Morphine and Codeine in its pain relieving strength with a milder sedative effect. Although classified as a Methadone derivative, based upon its chemical formula, Propoxyphene (Darvon), is not classified as a narcotic under Federal Law governing the dispensing of prescription drugs.

**(i) Methadone and Meperidine (Demerol) Narcotic Analgesics**

<u>GENERIC NAME</u>	<u>TRADE NAME</u>
Methadone	Dolophine
Meperidine	Demerol and Isonipecaine
Propoxyphene (Non-Narcotic)	Darvon

## C. NON-NARCOTIC ANALGESICS

### 1. THE SALICYLATE GROUP

The Salicylates are derivatives of aspirin. Aspirin's chemical name is acetylsalicylic acid and is abbreviated "ASA". A relative of aspirin is Methylsalicylate, known as "oil of wintergreen" which is rubbed externally into "aching muscles". Some salicylates are found in over the counter external medications such as "Ben Gay" and "Deep Heat". They are used to treat muscle aches, since when applied they produce a feeling of warmth.

In addition to its analgesic properties (pain killing) the salicylates are also antipyretics (reducing fever). In higher doses the salicylates reduce inflammation. The side effects of the salicylates are gastrointestinal (GI) bleeding and tinnitus (ringing in the ears from the Latin word meaning "a ringing").

**PRACTICE NOTE:** A client who presents with a history of ringing in the ears may not be experiencing the residuals of a head or an inner ear injury, but merely an overdose of aspirin.

#### (a) The Salicylate Analgesics

<u>GENERIC NAME</u>	<u>TRADE NAME</u>
Aspirin	Bayer, Ecotrin, Bufferin
Diflunisal	Dolobid
Salsalate	Disalcid

## 2. ACETAMINOPHEN (TYLENOL)

Acetaminophen relieves pain and reduces fever, but does not relieve inflammation like aspirin. Acetaminophen does not have the gastrointestinal side effects of aspirin but may be toxic to the liver (where is it metabolized) with chronic use. Frequent alcohol intake (such as a few glasses of wine with dinner) while taking acetaminophen presents an increased risk to the patient for developing liver toxicity.

## 3. NSAIDs

Non-Steroidal Anti-inflammatory Drugs are abbreviated NSAIDs. These drugs such as Motrin and Naprosyn, in addition to relieving inflammation, also relieve pain. A new NSAID, ketorolac (Toradol) recently has been used to treat pain. Because of its gastrointestinal irritating side effects, Toradol's use is limited to 5-14 days duration. NSAIDs are discussed in detail below.

## 4. ANTI-DEPRESSANTS

Anti-depressants such as amitriptyline (Elavil), fluoxetine (Prozac), sertraline (Zoloft) and Venlafaxine (Effexor) have recently been used to treat chronic pain.

### III. ANTI-INFLAMMATORY DRUGS

#### A. STEROIDAL ANTI-INFLAMMATORY DRUGS

##### 1. DISCOVERY OF THE STEROIDS

Medicine exploded in the 1940's with the discovery of the steroids leading to several Nobel Prizes and the development of new wonder drugs to treat arthritis. The name steroid was derived from an old compound called sterol. In the 1940's when organic chemists first developed the formula for steroids they noticed that it was similar to the formula for an old compound named sterol. The suffix "oid" (from the Latin meaning "similar to") was added to sterol giving the name steroids. Steroids have a characteristic 4-ring chemical nucleus; the same nucleus found in sterol, in cholesterol, and in the sex hormones.

##### 2. THE HORMONES OF THE ADRENAL CORTEX

Our body produces three types of steroid hormones in the adrenal gland located just above the kidney. The outer layer of the adrenal gland, called the cortex, produces these hormones. These steroids are referred to as the adrenal cortical hormones (from the adrenal cortex). In addition, physiologically active steroids are called "corticosteroids" (referring again to the adrenal cortex).

##### 3. THE THREE CLASSES OF STEROIDS

Steroids are divided into three classes based upon their effect on the body:

- (i) Female (Estrogen) and Male (Androgen) Sex Hormones;
- (ii) The Mineralcorticoids;
- (iii) The Glucocorticoids.

All three classes of steroid hormones are produced by the adrenal cortex. (1) Estrogen and Testosterone, the sex hormones; (2) Aldosterone, a mineralcorticoid; and (3) Cortisone and Hydrocortisone, glucocorticoids.

The Mineralcorticoids regulate the body's electrolytes by controlling sodium retention and the excretion of potassium -- important body minerals -- hence the name Mineralcorticoids.

The Glucocorticoids increase the body's blood glucose -- hence the name Glucocorticoids. In addition, Glucocorticoids decrease inflammation. Synthesis of more potent derivatives of the body's own glucocorticoids (Cortisone and Hydrocortisone), lead to the development of today's potent "steroidal anti-inflammatory drugs" such as dexamethasone (Decadron), and triamcinolone (Aristocort).

Synthesis of derivatives of the body's sex hormones, Estrogen and Testosterone, have resulted in female "birth control pills" and "Anabolic Steroids", the latter promote nitrogen retention and weight gain. (The term "anabolic" is derived from the word "anabolism" -- retention of nitrogen).

**PRACTICE NOTE:** Anabolic Steroids, misused by athletes, have given the generic term "steroid" a "bad name". As a result, many lay people associate "steroid" with drug misuse. If the plaintiff were prescribed an anti-inflammatory steroid, the jury should be informed by the treating doctor, that this class of steroids (glucocorticoids) is vastly different from the anabolic steroids misused by athletes.

4. SIDE-EFFECTS OF ANTI-INFLAMMATORY STEROIDS

Chronic use of anti-inflammatory steroids (glucocorticoids) can result in serious side effects such as:

- (i) Osteoporosis (brittle and soft bones);
- (ii) Cushing's Syndrome with characteristic "moon face" and "buffalo hump" in the shoulder;
- (iii) Salt Retention and Increased Blood Pressure (Residual Mineralcorticoid effects);
- (iv) Decreased Immune System Response resulting in greater susceptibility to infection;
- (v) Suppression of Growth in Children;
- (vi) Behavioral Problems;
- (vii) Reproductive Problems.

5. SYNTHETIC STEROIDAL ANTI-INFLAMMATORY DRUGS

<u>GENERIC NAME</u>	<u>TRADE NAME</u>
Prednisone	Deltasone, Meticorten
Prednisolone	Delta Cortef, Sterane
Methyl Prednisolone	Medrol
Triamcinolone	Aristocort, Kenalog
Paramethasone	Haldrone, Stemex
Betamethasone	Celestone, Mexibate, Betarex and Diprolene
Dexamethasone	Decadron, Hexadrol

## B. NSAIDs

### 1. GENERAL

Non-Steroidal Anti-Inflammatory Drugs are abbreviated NSAIDs. Included within this classification are all drugs which reduce inflammation which are not steroids. The NSAIDs have side-effects, but they are not as serious as the steroidal anti-inflammatory drugs.

The NSAIDs have two physiological effects on the body -- reducing inflammation and relieving pain. Some NSAIDs have greater effectiveness at reducing inflammation, while others have greater effectiveness at reducing pain, such as ketorolac (Toradol).

All NSAIDs produce gastrointestinal side-effects such as nausea, vomiting and ulcers. Approximately one-quarter of all people who take NSAIDs develop gastrointestinal side-effects. The NSAIDs with the highest toxicity are Indocin, Tolectin, Meclomen, and Orudis.

**PRACTICE NOTE - Increased Specials - Blood Tests:**  
Clients prescribed NSAIDs for long term use to treat chronic pain should have blood tests taken 1-2 times per year, i.e., a chem. profile, to monitor for possible kidney and liver damage caused by long term use of NSAIDs.

## 2. NON-STEROIDAL ANTI-INFLAMMATORY DRUGS

<u>GENERIC NAME</u>	<u>TRADE NAME</u>
Aspirin	Bayer, Bufferin, Ecotrin
Diflunisal	Dolobid
Salsalate	Disalcid
Ibuprofen	Motrin, Advil, Nuprin
Naproxen	Naprosyn, Anaprox, Aleve
Fenoprofen	Nalfon
Ketoprofen	Orudis, Oruvail, Actron
Flurbiprofen	Ansaid
Oxaprozin	Daypro
Indomethacin	Indocin
Tolmetin	Tolectin
Sulindac	Clinoril
Diclofenac	Voltaren
Etodolac	Lodine
Meclofenamate	Meclomen
Mefenamic acid	Ponstel
Piroxicam	Feldene
Nabumetone	Relafen
Ketorolac (analgesic)	Toradol
Celecoxib	Celebrex
Meloxicam	Mobic

## IV. MUSCLE RELAXANTS

Most muscle relaxants are central nervous system (CNS) depressants and relieve muscle spasm by inhibiting spinal reflexes. Most muscle relaxants have side effects of drowsiness and dizziness.

### 1. MUSCLE RELAXANT DRUGS

<u>GENERIC NAME</u>	<u>TRADE NAME</u>
Meprobamate	Equanil, Miltown
Carisoprodol	Soma, Soprodol, Rela
Methocarbamol	Robaxin
Chlorphenesin Carbamate	Maolate
Diazepam	Valium
Chlorzoxazone	Paraflex; in Parafon Forte
Metaxalone	Skelaxin
Chlormezanone	Trancopal
Cyclobenzaprine	Flexeril
Orphenadrine	Norflex, Disipal
Baclofen	Lioresal

## V. ANTI-DEPRESSANTS

Tricyclic anti-depressants are abbreviated "TCAs." Their chemical nucleus consists of a tricyclic ring -- hence the name "tricyclic." The tricyclic anti-depressants have a delayed onset of 2-3 weeks. TCAs have a long half life in the body, and therefore may be administered only once a day, usually at bedtime, to minimize the side-effect of sedation and drowsiness. Other side effects of TCAs include lowering seizure threshold. Patients with seizure disorder may experience a recurrence of seizures while on TCAs. In addition, tricyclic anti-depressants exhibit cardiac toxicity and should be used with caution in patients who have had a heart attack. TCAs are CNS depressants. Patients taking TCA's should use caution while drinking alcohol, which is also a CNS depressant.

Selective Serotonin Reuptake Inhibitors (abbrev. "SSRI") are a new class of anti-depressants. The most well known "SSRI" is Prozac.

Antidepressants, such as Amitriptyline (Elavil), Prozac, Zoloft and Effexor have been found to have pain-lowering effects. They are now used to treat chronic pain in addition to being used to treat depression.

The most commonly prescribed anti-depressants are set forth below:

1. **ANTI-DEPRESSANT DRUGS**

<b><u>GENERIC NAME</u></b>	<b><u>TRADE NAME</u></b>
Amitriptyline (TCA)	Elavil, Amitril, Emitrip, Endep
Amoxapine (TCA)	Asendin
Clomipramine (TCA)	Anafranil
Desimipramine (TCA)	Norpramin, Pertofrane
Doxepin (TCA)	Sinequan, Adapin
Imipramine (TCA)	Tofranil, Pesamine, Dramine, Janimine
Nortriptyline (TCA)	Aventyl, Pamelor, Allegron
Protriptyline (TCA)	Vivactil
Trazodone (TCA Related)	Desyrel, Molipaxia
Trimipramine (TCA)	Surmontil
Citalopram (SSRI)	Celexa
Escitalopram (SSRI)	Lexapro
Fluoxetine (SSRI)	Prozac
Fluvoxamine (SSRI)	Luvox
Paroxetine (SSRI)	Paxil, Seroxat
Sertraline (SSRI)	Zoloft
Duloxetine (SNRI)	Cymbalta
Nefazodone (SNRI)	Serzone
Reboxetine (SNRI)	Edronax
Venlafaxine (SNRI)	Effexor
Mirtazapine (NaSSA)	Remeron, Zispin
Bupropion (AK)	Wellbutrin
Maprotiline (NRI)	Ludiomil

TCA = Tricyclic Antidepressant

SSRI = Selective Serotonin Reuptake Inhibitor

SNRI = Serotonin and Noradrenaline Reuptake Inhibitor

NaSSA = Noradrenergic and Specific Serotonergic Antidepressant

AK = Aminoketone

NRI = Noradrenaline Reuptake Inhibitor

## VI. ANTI-ANXIETY DRUGS

Drugs used to treat anxiety are called Anxiolytics (dissolve anxiety) or tranquilizers. Anxiety conditions include general anxiety, phobias and panic states.

The benzodiazepines were the first class of drugs to treat anxiety without major sedation. The group name, "benzodiazepine," is derived from the benzodiazepine chemical nucleus, which is responsible for anti-anxiety properties. Buspirone (BuSpar) introduced recently to treat anxiety with fewer side effects, is not a member of the benzodiazepine family.

Long-term use of the benzodiazepines may result in withdrawal symptoms such as anxiety, fatigue and insomnia when the drug is stopped. Low doses of benzodiazepines produce anti-anxiety relief; higher doses produce sedation.

### 1. ANTI-ANXIETY DRUGS

<u>GENERIC NAME</u>	<u>TRADE NAME</u>
Lorazepam	Ativan
Buspirone	BuSpar
Prazepam	Centrax
Chlordiazepoxide	Librium
Halazepam	Paxipam
Oxazepam	Serax
Clorazepate	Tranxene
Diazepam	Valium
Alprazolam	Xanax

## VII. SLEEPING PILLS

Many clients with neck and back injuries have trouble sleeping due to pain.

Drugs which induce sleep are called hypnotics.

The most popular sleeping pills today are the short acting (hypnotic) benzodiazepines. These drugs have been approved as "sleeping pills" for short-term use -- no longer than 4-6 weeks.

### 1. BENZODIAZEPINE "SLEEPING PILLS"

<u>GENERIC NAME</u>	<u>TRADE NAME</u>
Flurazepam	Dalmane
Temazepam	Restoril
Triazolam	Halcion
Zolpidem	Ambien
Quazepam	Doral
Estazolam	Prosom

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