Preface

My fellowship in Pain Management was filled with busy days of rounding on patients, performing countless procedures, and preparing myself for the dreaded American Board of Anesthesiologists (ABA) Oral Board Examination. I never really dedicated the time I should have to studying or thoroughly understanding the fine details I later found I needed to know in order to master the ABA Pain Boards Certification Exam. I also discovered that there wasn't a comprehensive system available that could help me prepare for the exam, and cover the multifaceted aspects of the board certification exam while remaining financially feasible for my salary.

What I found most challenging and frustrating at the same time was the multiple resources I needed to access in order to obtain the information I needed to properly prepare for the Pain Management Certification exam. Over the months that followed, I read books and articles from multiple sources and kept meticulous notes for review. At the conclusion of my studies I realized that I had produced over 200 pages of notes that could be used as a pain board review for future generations. Shortly thereafter, *Beyond Pain* was born. I used the knowledge gained and study materials accumulated to develop a study course that could be used by pain practitioners in all levels of training for a reasonable cost.

What you will find at BeyondtheBoards.com and specifically in *Beyond Pain* is a complete and thorough review of the material you need to know in order to achieve top notch knowledge in the field of Pain Management. My intention is to completely immerse you in the field and keep you *continuously updated* so that you can achieve superior knowledge that can be utilized during your clinical career. In addition, my goal is not only to help you prepare for what lays ahead, but to provide you with the knowledge needed to continue to succeed throughout your career and beyond.

It is clear that medicine is as much a business as it is an art. Yet, we receive no education in Asset Protection, Tax Reduction, and Wealth Preservation during our medical training. While my intention is to help you prepare for your board certification exam, I also intend on helping you learn more about business and finance as it relates to physicians. So, study hard, use the resources available to you here and once you've passed the boards as I'm sure you will with persistence and dedication, come back and visit us at BeyondtheBoards.com for further education.

Finally, if there are any comments or recommendations for improvement with respect to this board study material, if there are any mistakes that should be corrected, or if any changes have occurred of which I am not aware, please help me and your colleagues stay updated by contacting me through my website.

Table of Contents

How to Use This Book

This book was produced after countless hours of studying, note taking, cross referencing numerous sources, and multiple cups of coffee. This book truly contains all the essential information you *must* know. You may at times ask yourself if you really need to know some of the information contained in this book. The answer to that question is yes, without a doubt. Some of the information, anatomical references, and neurological pathways are quite complex and cannot be mastered easily. Hence, this is NOT a book you can read and regurgitate after one read. Repeated exposure to the study material within these pages is essential. I would not claim that this book contains absolutely everything you would see on the certification exam. But I will promise that if you truly know all the material in this book, not only will you guarantee your ability to pass the board examination, you will also become a better physician.

There are 38 chapters in this book. I would recommend that you review each chapter *at least* three times. Once you begin studying with this book you will realize that all the extra verbiage seen in most text books has been eliminated and only the essential core contents remain. If you study one chapter per day, and review the book in its entirety three times, you will require approximately 120 days (that's four months!) so do not procrastinate. Begin each day by reading a chapter and taking notes or making up practice questions based on each section of the chapter. If you resort to highlighting, you may find yourself highlighting entire pages so I would refrain from that specific study technique. At the end of your study sessions, review your notes or the study questions you've come up with, and then go back and review the contents of the chapter you read the day before.

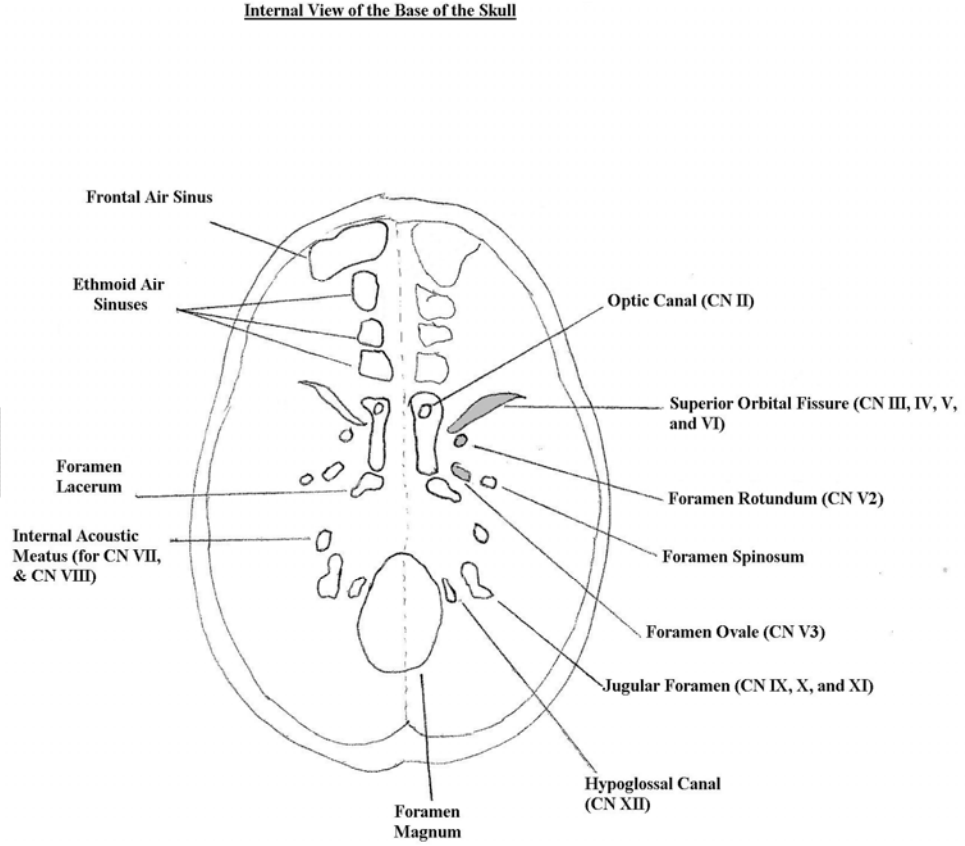
If you would rather obtain practice questions designed to assess your mastery of the material contained within these pages, I would recommend utilizing the study companion to this book (*Beyond Pain: The Study Companion*) which contains questions and answers with page references based on the thirty eight chapters in this book. I have found this study companion to be an extremely useful tool in promoting active learning and in driving the information home. In either case, true understanding of this material is essential to your ability to pass the Pain Boards Certification Exam. To that end, let us get started…

Chapter

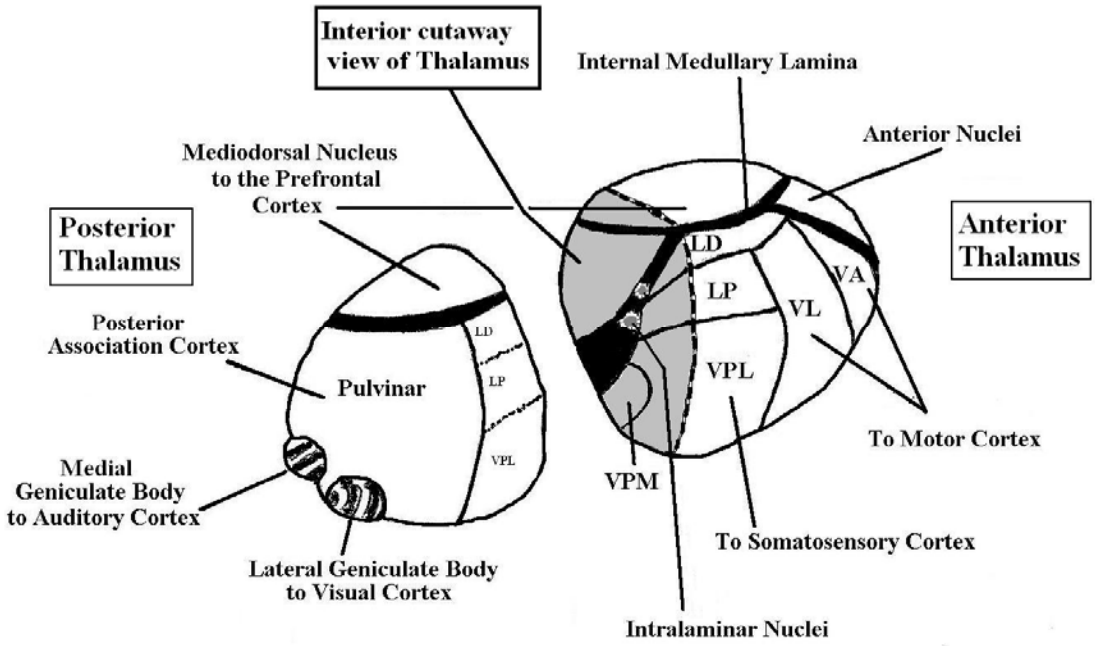
1

Anatomy of the Brain and the Cranial Nerves

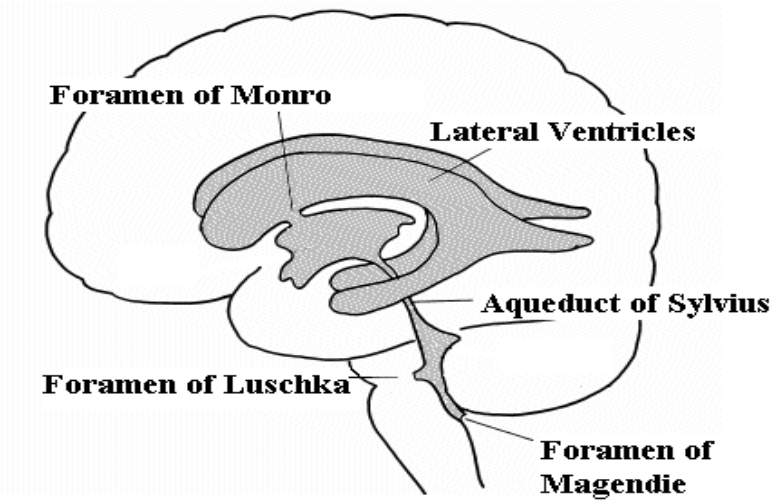
1. Review bony structures of the skull specifically the base anatomy and CN routes (Interior aspect of base of skull):



2. Review the structures of the Thalamus:



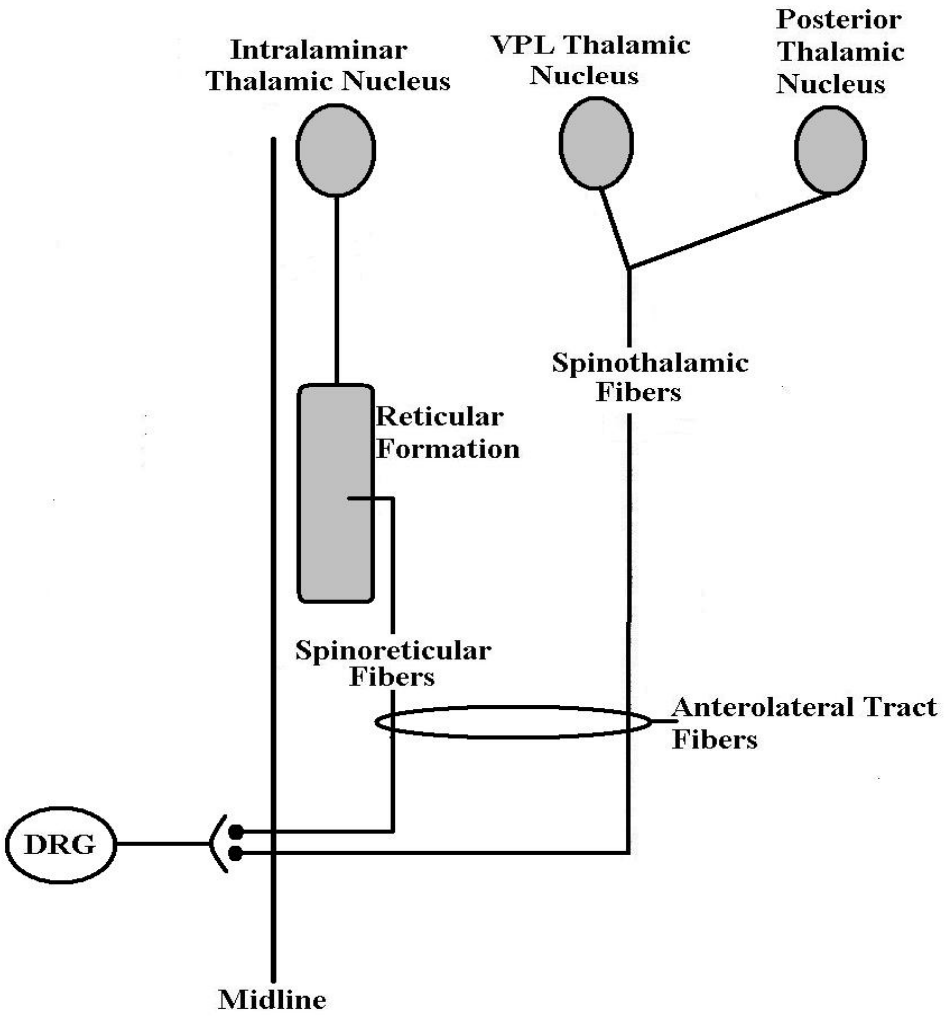
3. CSF is produced by the Choroid plexus of the lateral ventricles; it courses through the **Interventricular foramen of Monro** to the third ventricle. From the third ventricle it passes through the cerebral **aqueduct of Sylvius** into the fourth ventricle where it exits **L**aterally via the foramen of **Luschka** or **M**edially via the foramen of **Megendie**. CSF then circulates around the spinal cord and the brain and is reabsorbed by arachnoid granulations into the superior sagittal sinus.



4. There are two components to our perception of pain. These include the motivational-affective component which relays information from the dorsal column to the limbic system via multiple contributory pathways from the reticular formation, to the hypothalamus, to the medial thalamus and finally to the limbic forebrain. The other is the sensory pathway which relies on information exchange from the lateral thalamus to the somatosensory cortex.
5. The ascending spinal nociceptive pathways rely on neurotransmitters such as Substance P, Glutamate agonists, Calcitonin G related protein (CGRP), and TNF alpha. These pathways include:
    a. Spinothalamic tract: This is the most prominent ascending nociceptive pathway from the spinal cord. **It is composed of axons from nociceptive specific to Wide Dynamic Range (WDR) neurons in Laminae I and V of the dorsal horn.** It projects to the **contralateral** side of the spinal cord and ascends in the anterolateral white matter and terminates in the lateral (VPL and Central Lateral nucleus) thalamus. Most of the cells project to the contralateral thalamus but a small fraction project to the ipsilateral thalamus. This pathway is illustrated below.

i. The majority of the axons project laterally to the **VPL**, VPM, VPI, VL, and LP nuclei of the Thalamus, but there are also some projections to the CM and CL nuclei.

ii. This tract helps to mediate the sensations of pain, cold, warmth and touch.



b. <u>Cervicothalamic tract:</u> Arises from neurons in the lateral cervical nucleus (located in the white matter of the upper two cervical spinal cord segments). The cervical nucleus receives input from **Laminae III and IV**. The axons travel to the **<u>contralateral</u>** side and ascend in the medial

lemniscus of the brain stem to nuclei in the midbrain and to the VPL and VPM nuclei of the thalamus. Some axons from Laminae II and IV project through the dorsal columns of the spinal cord (along with large A-beta fibers) to end in the cuneate and gracilis nuclei of the medulla (it is debatable whether or not this tract exists).

c. Spinomesencephalic tract: Most axons of this tract originate from **Laminae IV-VI as well as Laminae X and the ventral horn**. Some of these neurons give off collaterals that end in the lateral thalamus. The axons project in the **contralateral** anterolateral quadrant of the spinal cord to the Mesencephalic (Pons-midbrain) reticular formation and *periaqueductal gray matter (PAG)* and via the Spinobrachial tract to the Parabrachial nuclei. In turn, the Parabrachial nuclei project to the *Amygdala* (which is the fear center of the brain), a major component of the limbic system concerned with emotion. Many axons in the pathway project in the dorsal part of the lateral funiculus rather than the anterior so they *may be spared in an anterior cordotomy* and hence may give rise to recurrent pain.
    i. Recall that the PAG is also a synaptic location for descending inhibitory fibers.

d. Spinoreticular tract: This tract is composed of axons from **Laminae VII and VIII**. These axons ascend in the ipsilateral anterolateral quadrant of the spinal cord and terminate in the reticular formation of the Medulla (in the midbrain there are projections to the nucleus Coeruleus, Parabrachial nucleus, and the Kollker-Fusner nucleus as well as nucleus Gigantocellularis). From the reticular formation there are further projections to the hypothalamus, medial thalamus, and the Cingulate

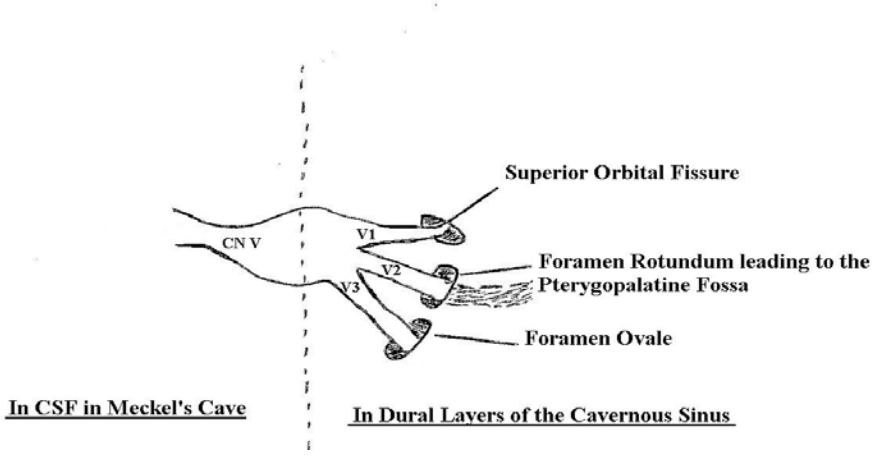
Gyrus. This is a slowly conducting pathway as compared to the Spinothalamic tract.

e. Spinohypothlamic tract: Comprises of axons from **Laminae I, V, and VIII**. It projects directly to the supraspinal autonomic control centers (including the Amygdala) and is thought to *activate complex Neuroendocrine and cardiovascular responses* (this is where fight or flight comes from!).

f. Postsynaptic Dorsal Column tract: Arises from cells distributed medial to lateral in **Laminae III as well as from a few cells lateral to Laminae X**. These axons are innervated by serotonin only fibers. There is a possible role for this pathway in relaying of visceral pain, but it is generally thought to serve as a pathway involved in two point discrimination and position sense.

6. There are a few descending inhibitory pathways which originate from the periventricular grey matter. These pathways rely on neurotransmitters such as Serotonin, NE, Opiates, and GABA. The *Periaqueductal Gray* (PAG) sends excitatory signals to the *Locus Coeruleus* (LC) in the Pons, Subceruleus, Parabrachial and the Nucleus Raphe Magnus in the Medulla (Brainstem raphe nuclei) which in turn send inhibitory signals via the dorsal longitudinal fasciculus which synapses on Laminae I, II, and V in the dorsal columns.
7. The cranial nerves include:
    a. **I- Olfactory**: special sensory smell.
    b. **II- Optic**: special sensory vision.
    c. **III- Oculomotor**: motor to all extraocular muscles except superior oblique and lateral rectus (SO4-LR6), parasympathetic to ciliary and pupillary constrictor muscles. Visceral motor sympathetic mediated for pupillary dilation.

d. **IV- Trochlear**: motor to superior oblique muscle.
e. **V- Trigeminal**: sensory from surface of head and neck to sinuses, meninges, and external tympanic membranes. Motor to muscles of mastication. (Review anatomy at end of section especially regarding → Trigeminal (Gasserion) ganglion in Meckel's Cave which contains CSF and which divides into an Ophthalmic branch [exiting via the Superior Orbital Fissure], Maxillary branch [Foramen Rotundum], and the Mandibular branch [Foramen Ovale].
    i. Recall that the Sphenopalatine foramen leads to the Pterygopalatine fossa where the Pterygopalatine ganglion resides. The contents of the Pterygopalatine fossa include the terminal branches of the *Maxillary artery*, the Maxillary nerve (V2) with its branches to the upper teeth, floor of orbit, and the face and skin, and the Pterygopalatine ganglion for distribution of parasympathetics to the nose and palate.
    ii. An illustration of the Trigeminal nerve and the appropriate foramen is shown below:



f. **VI- Abducens**: motor to lateral rectus.

g. **VII- Facial**: *Motor* to muscles of facial expression and stapedius, visceral motor, general *sensory* from small area around the external ear and external tympanic membrane, *parasympathetic* supply to all glands of head except parotid (innervated by CN IX) and skin glands, *taste* to anterior 2/3 of the tongue.

h. **VIII- Vestibulocochlear**: special sensory for balance, hearing.

i. **IX- Glossopharyngeal**: *motor* to stylopharyngeus muscle, *parasympathetic* to parotid gland, visceral s*ensation* from the carotid body, general sensation from posterior 1/3 of the tongue, and sensation from internal surface of tympanic membrane, visceral motor and sensory.

j. **X- Vagus**: general motor and sensory, visceral motor and sensory. Motor to pharynx and larynx, parasympathetic to all thoracic and abdominal viscera as far caudal as the splenic flexure, visceral sensory from pharynx, larynx, thorax, and abdomen. General sensation from small area around the external ear.

k. **XI- Accessory**: motor to sternocleidomastoid and trapezius muscles.

l. **XII- Hypoglossal**: motor to intrinsic and extrinsic muscles of tongue except the palatoglossus.

8. A summary of the cranial nerves that carry parasympathetic fibers is shown in the table below:

Cranial Nerve	**Effect**
CN X (Vagus)	Heart, GI system
CN IX (Glossopharyngeal)	Parotid gland
CN VII (Facial)	Lacrimal gland, Mucosal glands of nose and palate, Submandibular and Sublingual glands
CN III (Oculomotor)	Pupil constriction, accommodation

Chapter

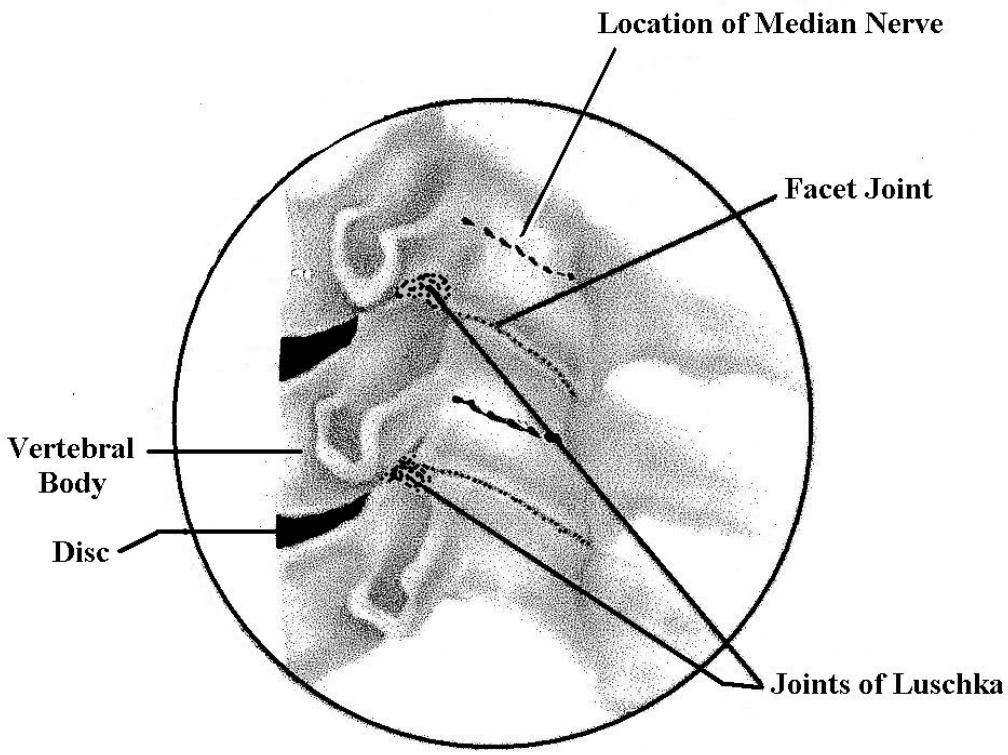
2

Anatomy of the Spine

1. Imaging considerations of the spine:
    a. MRI is very good for screening of the spine because you can see sagittal and axial views. MRI is good at looking at soft tissue, while CT scans are good at looking at bone. In addition, CT scans are reconstructed axial images only and that is their limitation.
    b. MRI images come in two patterns: T1 and T2 weighted images. **T1** is better for visualizing **fat** and fat appears white on T1 weighted images. T2 is better at visualizing water and water appears white on T2 weighted images. (remember T-t**w**o visualizes **w**ater)
    c. The disks and CSF should appear bright in a T2 weighted image. T1 is good at looking at the structure of the vertebrae (bones).
    d. When reading the MRI, start with the T2 weighted image and look for "dark" disks as well as the posterior longitudinal ligament. Scan the sagittal view and look at the corresponding axial view for further detail. Also, examine the Psoas muscle for abnormalities that may explain the patient's pain.
    e. When and why do you use contrast? To determine if there is an **infection** or a **neoplasm,** and to distinguish a **sequestered disk** from something that is malignant or infected.

f. CT scans are composed of a soft tissue field and a bone field.
g. Myelography is used to inject contrast into the intrathecal space. It is used to examine nerve root obstruction, or CSF obstruction. It is used to demonstrate normal anatomy or the lack thereof. CT can be combined with myelography and is often used in patients that have metal hardware since MRI would result in a lot of artifact.
h. Discography has multiple components including volumetric, manometric, radiographic, and pain provocation (concordant or discordant).
    i. Volumetric exam involves injecting contrast into a disk. The maximum volume that can be placed in a lumbar disk is 2 cc, while a cervical disk can take 5 cc.
    ii. Manometric evaluation involves transduction of pressures with increased disk volume and can be fairly subjective.
    iii. Radiographic examination involves x-ray or CT examination of the contrast pattern produced with the injection.
    iv. Pain provocation is the "meat of the matter". If the patient's pain is reproduced with volume injection into the specific disk, then the source of the pain and the diagnosis is made. This is concordant pain. Discordant pain is where the patient has no pain on injection or has a new pain on injection.
        1. Discography complications include discitis, meningitis, epidural abscess, post dural puncture headache, intrathecal hemorrhage, and Arachnoiditis. Intravenous antibiotics must be administered with this procedure.

2. Anatomy of the *5 joint complex (C-spine):*
    a. The lower cervical segments from C3-C7 consist of a 5 joint complex. This complex is composed of one <u>intervertebral disc</u>, two <u>facet joints</u>, and two <u>uncovertebral joints of Luschka</u> (these are not really joints but are rather articulations of the uncinate process which when arthritic can cause nerve impingement in the neck).



    b. For the cervical segment the nerve roots exit ***<u>above</u>*** the given pedicle. Hence the C5 nerve root exits above the C5 pedicle (the exception to this rule is C8 which exits above T1).

c. To view cervical facets on x-ray, a lateral view should be obtained. If one wishes to view the foramen, an oblique view should be obtained. In the lumbar region, the oblique view is used to identify the facets and the lateral view is used for the foramen.

d. In an AP view of the cervical region, a "half moon" shape is noted on either end of the spine, the median nerve innervating the given facet is located in the center of this half moon. In the lateral view, the cervical median branch nerve lies at the center of the trapezoid-shaped articular process.

e. In the cervical region as in the lumbar region, each particular facet is innervated by the median nerve from the current level, and the level above.

3. Anatomy of the *3 joint complex (L-spine):*

a. In the lumbar region there are no uncovertebral joints of Luschka. The lumbar region consists of a 3 joint complex. This complex is composed of one intervertebral disc, and two facet joints.

b. For the lumbar and thoracic segments the nerve roots exit ***below*** the given pedicle. Hence, the L4 nerve root exits below the L4 pedicle. If there is a disk herniation at L4-L5, the L4 nerve root has already exited and the L5 nerve root is the one that is affected.

c. The *anterior longitudinal ligament is thicker* than the posterior longitudinal ligament. Hence, posterior herniations are more common than anterior herniations.

4. Abnormal anatomy of the spine:

a. Intervertebral disk

i. The disk is made up of two components: the Annulus fibrosis and the Nucleus pulposus. The Annulus fibrosis consists of concentric

lamellar rings around the disk, the outer 1/3 of which is innervated. The Nucleus pulposus is made up of 70-90% water and becomes desiccated with age. The intervertebral disks are avascular structures and receive nutrients via cartilaginous end plates. These cartilaginous vertebral endplates are susceptible to fractures which can disrupt the nutrient supply and cause damage to the intervertebral disks. Once the disk is damaged it will degenerate and either undergo *resorption* or it will undergo *internal disk disruption*.

1. Isolated disk resorption is accompanied by degradation of the nucleus, disk height narrowing, subchondral marrow degeneration, and osteophyte formation (spondylosis).
    a. Subchondral marrow changes are referred to as Modic changes of which there are three types: I, II, and III. Modic changes result due to fractures of the cartilage and the disk endplates and indicate advanced DDD.
2. Disk dehydration leads to loss of disk height, foraminal narrowing, and osteophyte formation in an attempt to stabilize the spine. "Traction and Claw" osteophytes form and attach the annular fibers to the vertebral body to stabilize the spinal segment. These osteophytes are horizontally oriented and indicate DDD and instability.
3. If the nucleus and the annulus are affected then there is internal disk disruption and this can result in annular fissures, disk bulge, protrusion, herniation, and sequestration.

4. Internal disk disruption is a painful condition, and is the earliest form of DDD marked by disk dehydration, loss of disk height, subchondral marrow changes, and an annular fissure. It is diagnosed by CT discography. It is an annular fissure that extends to the outer 1/3 of the annulus (hence the reason for painful symptoms).

b. Annular fissures or Tears
    i. This is due to discontinuity of the annulus fibrosis. It is also called a High Intensity Zone and is not always symptomatic.

c. Disk bulge
    i. These are abnormalities of disk contour. If the disk bulge extends greater than 50% of the disk circumference and *greater than 3 mm beyond the vertebral body* it is called a generalized disk bulge. If it extends less than 50% of the circumference of the disk then it is a focal herniation.
    ii. Disk protrusion is the same as a herniations and an extrusion. The difference is the extent to which the disk bulges into the epidural space. In an axial view, a disk protrusion has a larger x-axis span than a y-axis extension. A herniation on the other hand has a small extension on the x-axis and a lot more on the y-axis (its extending into the epidural space more). A disk extrusion is simply a herniation that extends cranially and caudally. A disk extrusion is in continuity with the disk. A disk sequestration is an extrusion where the segment is no longer in continuity with the disk. Extrusions and sequestrations will not enhance with contrast which is another reason to obtain a contrast study when there is a mass in the lateral recess. If the mass "lights up" then it is not disk

sequestration. (Hence the progression is: protrusion → herniation → extrusion → sequestration)

iii. A central protrusion or herniation may not produce symptoms if it does not compress a nerve root whereas a lateral protrusion onto a nerve root can cause positive symptoms.

d. Spinal Stenosis, Lateral Recess Stenosis

i. Lumbar spinal stenosis is the most common cause of lumbar back pain in patients over the age of 50. The pathogenesis is due to degenerative disk disease and biomechanical stresses. It can also be due to facet and ligamentum hypertrophy causing cord compression.

ii. The pathogenesis of the stenotic pain can be due to arterial insufficiency, venous congestion, or compression.

1. Arterial insufficiency leads to large myelinated fiber loss, dorsal column disease and wide based gait due to loss of proprioception (+ Romberg test).
2. Venous congestion leads to neurologic claudication. Pain can be present in the buttocks, thighs, and legs. Pain increases with walking, standing, and sitting. It decreases with rest, and flexion of the spine.
3. Compression leads to impingement signs and decreased reflexes (lower motor neuron signs). There can be numbness, paresthesias, and weakness of the LE as well.

iii. Cervical spinal stenosis is associated with myelopathy whereas lumbar stenosis is not usually associated with myelopathy because the spinal cord ends at L2. If lower extremity hyperreflexia exists (upper motor neuron sign) one must consider cervical stenosis and

also check the upper extremity for hyperreflexia. If there is no UE hyperreflexia, then consider thoracic spinal stenosis.

iv. Upper motor neuron and lower motor neuron signs can be differentiated as follows:

Upper Motor Neuron	**Lower Motor Neuron**
Weakness	Atrophy
Spasticity	Flaccidity
Hyperreflexia	Hyporeflexia
Up-going plantar response	Fasciculation

5. Pathologic conditions of the spine:
    a. Vertebral bodies
        i. Tumors and metabolic bone disease
            1. Can affect the integrity of the vertebral bodies and cause painful, destructive lesions.
        ii. Infections
            1. It may be difficult to differentiate between DDD and infectious spondylitis. Infectious spondylitis can show degenerative lytic changes in the endplates. The classic findings of infectious spondylitis on MRI can be found on T1 weighed images of the *subchondral marrow*. Sometimes it is difficult to distinguish between Modic changes (extensive DDD) and infection. Contrast can be helpful in these circumstances which can help delineate an infectious process. T2 contrast images also will help enhance the disk, subchondral marrow, and the epidural space.

iii. DISH syndrome (diffuse idiopathic skeletal hyperostosis)
    1. This syndrome is associated with pain and stiffness. It is non-radiating in nature. The primary complaint is in the thoracic or lumbar region. Cervical pain can develop later. It occurs due to calcification and its etiology is unknown. The disease is diagnosed radiographically via "flowing" calcifications along the *anterolateral border of 4 contiguous vertebrae* with no loss of disk height or facet ankylosis. This is different from ankylosing spondylitis where there *are* disk height and facet changes, as well as sacro-iliac joint changes and calcifications.

iv. Ossification of the Posterior Longitudinal Ligament (PLL)
    1. The etiology of this disorder is unknown, but it is common in the cervical spine and occurs in 50-60 year old individuals (commonly of Japanese descent). Onset is usually after a minor trauma and the condition can coexist with DISH.
    2. Initial symptoms are neck pain, but as the spinal canal becomes more stenotic UE pain, dysesthesia, and weakness ensues. This can lead to LE symptoms and eventual bladder dysfunction.

v. Arthropathies (rheumatoid arthritis [spondylosis], and ankylosing spondylitis)
    1. Ankylosing spondylitis is a spine calcification disorder that is HLA-B27 positive. Calcification of the spine affects the sacro-iliac joints (which are affected first), the facets, the

intervertebral disks, and the costovertebral joints. There is also calcification of the annulus and the ligaments which gives rise to the term "Bamboo spine" which lacks flexibility and is susceptible to fractures. This is what sets this disease apart from DISH.

b. Impingement upon spinal nerve roots
    i. Definition: *Radiculopathy* is motor, sensory, reflex, and paresthetic changes that follow a dermatomal distribution and can be traced back to a specific nerve root. Pain simply in a dermatomal distribution is called *Radicular* pain. *Radiculitis* refers to symptoms (pain) in a dermatomal distribution but with a normal neurologic exam. There might be a radiologic finding at the spinal level corresponding to the symptom. This is thought to be due to a chemical insult from a "leaky" disk.
    ii. It is thought that the mechanism behind symptoms associated with disk herniations is due to either nerve compression or inflammation from a leaking disk releasing PLA-2, Nitrous Oxide, Cytokines, TNFα, and Glutamate.
c. Disorder of alignment: Spondylolisthesis
    i. Spondylolisthesis is forward slippage of a superior on an inferior vertebra. There are five types of Spondylolisthesis:
        1. **Dysplastic** (congenital): affecting upper S1 or inferior L5
        2. **Spondylo-lytic**: a fracture of the pars interarticularis which has an unclear etiology. There is an association with sacral Spina Bifida. L5-S1 is the most common and occurs in caucasian young males, and female athletes (ex: gymnasts).

3. **Degenerative:** due to intersegmental instability and aging. L4-L5 is the most common, seen in African-American females over 40 years old. Sacralization occurs in 22% of the population.
4. Traumatic
5. Pathologic

ii. Spondylolisthesis patients complain of low back pain and leg pain. It can occur in patients as young as 5-7 years old. Symptoms start with twisting and lifting. If there is an acute fracture that is less than 3 months old, it can best be detected by a bone scan. *The degree of slippage does not equal the degree of pain.* Pain typically increases with flexion and activity, and is reduced with extension and rest. Depending on the degree of slippage spinal stenosis can result.

iii. Diagnosis is made by flexion and extension films. *If slippage is greater than 2 mm the spine is considered unstable and the treatment is surgical correction.*

d. Disorder of posture: Dowager's Hump
   i. This is an Osteoporotic spine kyphosis with loss of vertebral height most notable in the thoracic spine. The neck can be placed in a position of flexion and a low grade chronic back ache can exist.

6. Spinal anatomy: Compartmental Model
   a. The spine can be conceptually divided into three compartments; Anterior, Middle, and Posterior.
   b. Anterior compartments contains the anterior longitudinal ligament, anterior 2/3 of the disk, and the sympathetic chain and ventral ramus.

i. The anterior compartment is innervated by gray rami communicans, and the ventral ramus.

c. Middle compartment contains the meninges, spinal nerves, posterior longitudinal ligament, and posterior 1/3 of the disk, and *sinuvertebral nerves*.

i. The middle compartment is innervated by the sinuvertebral nerves which make multiple connections at multiple levels of the spine, they do not innervate just one level.

d. Posterior compartment contains the facets, muscles, SI, and medial branch of the dorsal ramus.

i. The posterior compartment is innervated by the medial branch of the dorsal ramus.

7. Treatment Paradigm:

a. Consider the etiology, pathogenesis, and the treatments outlined below:

Etiology	**Pathogenesis**	**Rx**
Annular tear	Leak of mediators, altered mechanism	IDET, Annuloplasty
Protrusion, Herniation	Leak of mediators, compression	Epidural steroids, IDET, surgery
Facet and SI	Biomechanical	RF
Failed Back Sx	Multiple	SCS, IDDS

b. Intradiscal Electrothermal Coagulation (IDET) can be used to cause *contraction of collagen tissue*, or to cause *thermal denervation* (irreversible at 45C) and granulation tissue formation. It is ideal for idiopathic disc disruption, contained HNP without radicular symptoms, annular tears, DDD with <u>less than 50% disc height loss</u>, and stabilization of a disc one level above a fusion.

c. Annuloplasty is similar to IDET. The difference is that with IDET the probe is inserted at the nuclear-annulus junction, whereas in Annuloplasty

the probe is inserted into the annulus itself. This procedure results in a monopolar RF lesioning that destroys the nociceptors in the outer 1/3 of the disc and causes collagen tissue contraction.

d. Nucleoplasty is used for contained disk herniations. This is really a selective percutaneous discectomy. A wand is inserted into nuclear tissue and Coblated via bipolar RF coagulation. This is a non-heat driven process that causes molecular disintegration. The bipolar RF creates a plasma layer around two electrodes from instilled NS. The highly ionized plasma field breaks molecular bonds. The byproducts are elementary particles and low MW gases. Hence, there is minimal collateral tissue necrosis.
e. Myeloscopy is basically spinal endoscopy via a caudal approach and is rarely used. It can be used for diagnosis of Arachnoiditis, for lysis of adhesions, aspiration of facet cysts, and visually guided ESI.
f. Epidural steroid injections can be performed via an interlaminar or transforaminal approach. The interlaminar approach is more of a "shot gun" maneuver while the transforaminal approach can be useful in applying medication onto a specific nerve root or to the anterior epidural space where a herniated disk might exist.