INTRODUCTION TO CASE MANAGEMENT
Patient Interview / Case History
Subjection Portion
Styles of Interview

DOCTOR CENTERED
7 Cardinal features of Symptoms
1. Location and Radiation
2. Quality
3. Quantity
4. Chronology
5. Setting
6. Modifying Factors
7. Associated Symptoms

PATIENT CENTERED
NURS – Emotion Handling
1. Naming
2. Understanding
3. Respect
4. Support

Different Types of Histories
1. Problem Focused
2. Comprehensive
Beyond "OPQRST". Skeleton for the History
Additional Areas to Include: social history, occupational history, ADLs, past medical history, review of systems.

Social History
1. Alcohol Consumption
2. Tobacco Use
3. Recreational Drug Use
4. How do these affect the Chief Complaint?

Occupational History
1. Workforce stressors

2. Mechanical Stress
3. Prior Work Related Injuries

ADL (Activities of Daily Living)
1. Dressing, Grooming, Eating etc
2. Extended ADL's
3. Golf, biking, playing with grandkids etc

Past Medical History
1. Childhood Illnesses
2. Trauma
3. MVA, Slips, Falls etc
4. Work Related Injuries (if not covered in Occ Hx)
5. Hospitalizations/Surgeries
6. Medications: current, past

Review of Systems: often uncover other area of complaint

EXAMINATION
1. History
2. Age of patient, sex of patient
3. Chief complaint – OPQRST
4. Social, Occupational, ADL, Past medical, Review of systems etc..
5. Physical Examination
6. Inspection
7. Palpation
8. Percussion
9. Instrumentation
10. ROM
11. Orthopedic
12. Neurologic
13. Exam of related areas
14. Lab and X-Ray

Knowing how to complete an accurate assessment – from taking the health history to performing the physical exam – will help you

to uncover significant problems. This will lead to correct differential diagnoses as well as make appropriate case management decisions. The history alone allows a correct diagnosis in approximately 80% of cases.

Purpose of the Patient Interview

1. Doctor patient rapport
2. Reason for care
3. Determine if your patient will benefit from Chiropractic care
4. Determine if a referral is needed – red flags
5. Determine appropriate case management

Physical Examination

1. Objective Portion of Case Management
2. Orthopedic Testing
3. Neurologic Testing
4. Visceral Examination

HIPPARONI: Mnemonic to remember step in Physical Exam

History
Inspection
Palpation
Percussion
Auscultation – viscerogenic causes
Range of Motion – active & passive
Orthopedic
Neurologic
Instrumentation

Clinical Impressions

1. Doctor's Assessment of Condition
2. Diagnosis

a. What you think is going to date
b. Will have a list of differentials

All Clinical findings MUST be covered in the clinical impression. No generic terms are allowed. i.e. lumbalgia, cervicalgia etc..

VINDICATE: Mnemonic
Universal Differential Diagnosis Tool. Helps to determine working diagnosis. See VINDICATE Powerpoint

Plan
What are you going to do to help the patient. How are we going to reach this.
Goals
a. Short Term Goal
b. Long Terms Goals
See Goal Writing for Chiropractors Presentation

Care Plans
1. Frequency
2. Duration
3. Adjunct Procedures
4. Patient Education
5. Home Exercise Plans

Life University College of Chiropractic
COC Mission Statement
Mission
The mission of the Life University's College of Chiropractic is to educate, mentor and graduate skilled and compassionate Doctors of Chiropractic to be primary care clinicians, physicians, teachers, and professionals, using the University's core life proficiencies as their foundation.
Goal #1: Students graduating from the College of Chiropractic will demonstrate a depth of knowledge, skills and behaviors required of competent Chiropractic primary health care clinicians and will be capable of providing safe and effective care.
Goal #2: Students will learn to fulfill the role and responsibilities of chiropractic primary care clinicians by demonstrating competence in eliciting patient history, in performing examination procedures (physical, neuromusculoskeletal, radiographic, laboratory, and chiropractic) and integrating those findings to create a diagnosis and assess the need for chiropractic care and/or

further diagnostic studies in the development of effective and appropriate case management plans.

MUSCULOSKELETAL COMPLAINTS

The approach to the patient's musculoskeletal complaint is a standardized, often sequential search for what can and cannot be managed by the examining doctor.

Involved Tissue

Must be able to determine the pain generator. (ie What tissue is involved?). Is it: Muscle, Tendon, Ligament / Joint capsule, Bursae, Joint, Disc, Nerve, Visceral Referred Pain

Pain

1. Acute traumatic pain – rule out: Fracture, dislocation, gross instability

2 .Non-traumatic pain – rule out: Tumors, inflammatory arthritides, infections or visceral referred pain (See illustration "Ascending pain pathway")

Types of Pain: (See illustration- "Regions of the somites")

1. Scleratogenous
2. Myogenous
3. Dermatognous
4. Visceral
5. Bone Pain

Quality of Pain

Aching	Muscle, tendon, ligament
Bounding	Vascular, circulation
Burning	Nerve root, arterial, visceral
Cramp	Muscle
Boring deep pain	Cancer, bone pain
Unrelenting deep pain	Cancer, visceral
Dull	Musculoskeletal, visceral
Gnawing	Ulcer

Radiating (well delineated)	Nerve (causalgia – burning pain from periph.nerve)
Radiating (diffuse)	Sclerotogenous or myogenous
Sharp/localized	Facet, visceral referred
Stabbing	Visceral, nerve root
Shooting	Nerve root, entrapment
Tearing	Anuerysm or severe sprain
Throbbing	Vascular, circulation
Tingling (segmental)	Nerve root, peripheral nerve
Tingling (non segmental)	If bilateral: cord compression, M.S., Diabetes.
	If unilateral: plexus

Visceral Referred Pain

Site of Referral	Organ
Right Shoulder inferior scapula	Right lung, Liver, Gallbladder
Left shoulder	Left lung, stomach, spleen
Jaw, neck, left shoulder and arm	Heart
Either shoulder	Diaphragm
Flank & Upper abdomen	Kidney
Substernal	Esophagus, heart, lung
Spine at T10	Pancreas
Suprapubic	Bladder
Occiput, forehead	Eye
Temporal HA	TMJ

If It crosses the knee or shoulder it's usually disk or neurologic

Musculoskeletal Complaints

All joints function differently due to bony configuration but all joints must be evaluated in relation to their kinetic chain. Such as in, Shoulder injury and TMJ.

All joints are structurally composed generally of the same tissues:

* Bone * Cartilage and synovium * Ligaments * Tendons/muscles

* Capsule * Bursa * Blood vessels, nerves, fat and skin
All of the structures that compose a joint can be injured by compression or stretch. **Compression** – fracture of bone, neural dysfunction in nerves ((alters axoplasmic flow). **Stretch** – tendon/muscle, ligament/capsule, neural/vascular, or bone/epiphyseal damage
Weight bearing joints are more susceptible to chronic degeneration and DJD. Non-weight bearing joints may be transformed into weight bearing joints to do mechanical changes.
Non-mechanical processes – infection, cancer, immunological processes

Conditions Most Commonly Encountered:
btumor, fracture, infection, bone loss
2. Muscle: strain/rupture (Concentric injury as muscle is contracted, Eccentric as muscle is lengthened). *Trigger points* (associated with MPS – see external links "Managing Myofascial Pain Syndrome" on Blackboard). Atrophy, myoscitis ossificans, muscular dystrophy
3. Tendon:
a. Paratenonitis (old term of tenosynovitis)= inflammation of the tendon
Clinical Signs are: swelling, pain, crepetation along the tendon, local tenderness, warmth
b. Tendonitis: old term was strain or tear of tendon. Symptomatic Degeneration of a tendon Including Vascular Disruption as well as Inflammatory repair response.
c. Tendinosis: new term. Intratendonious degeneration. Atrophy. Microtrauma. Non Inflammatory
4. Ligament (excessive force on opposite side). Sprain or Rupture.Valgus/varus force. Chronic stresses.
5. Bursa: bursitis
6. Fascia: Myofascitis (An inflammation of the thin layer of fibrous tissue (fascia) that surrounds a muscle and attaches it to the bone.
7. Joints: arthritis, subluxation (Chiropractic), synovitis, infection, medical dislocation/subluxation.

Orthopedic Tests: reproduce Patient complaint(s). Reveal Laxity. Demonstrate weakness. Demonstrate restriction. Remember **Intent of test**, biomechanics and anatomy being affected. Any pain or response (whether expected or not) provides insight

Vertebral Motor Unit consists of: vertebral Bodies, intervertebral disc (IVD), facet joints, spinal Nerve, and ligaments
Intervertebral Foramen-

DISC DISEASE
Intervertebral Disc Disorders
Forms a symphsis or ampiarthrosis between 2 adjacent vertebral bodies. Largest avascular structure in the body. All the of IVDs combined together comprise 20-33% of the total length of the spinal column.

All IVDs Are Composed Of: annulus fibrosis, verterbral end plate, nucleus pulposis. This is present in all levels of the spine. This disc is incapable of independent motion, but does move with the 6 cardinal motions of the spine.

Major Stresses A Disc Goes Through: axial compression, shearing, bending, twisting. This can occur independently or with coupled motion.

Unique Role of the Disc: osmotic system, holds neighboring vertebra together and simultaneously pushing them apart. Dynamic structure that responds to stresses applied from vertebral movement or from static loading.

Annulus Fibrosis
10-12 concentric sheet of connective tissue. Though can be as many as 15-25. Number of layers decrease with age, but the remaining layers gradually thicken to compensate. Fibers are orientated at 65º from vertical in alternating layers. 50% of fibers are under stress with rotation at any given time. Lamella are

thicker anterior than posterior. Resulting the disc being thicker anterior than posterior. The IVD maybe thinner but it is more densely packed, therefore stronger tissue posterior than anterior. Outermost layer of the annulus attached to the vertebral body via Sharpey's Fibers.

Nucleus Pulposis (NP)
Semi fluid mass. At birth the water content is 80% and 68% in the elderly. Fully contained by the Annulus Fibrosis (AF) in the healthy adult. There is no clearly defined area between the NP and AF except in early youth.

Innervation
Sinuvertebral Nerve: a branch of the ventral ramus and the sympathetic trunk, innervates the outer half of the IVD, posterior longitudinal ligament (PLL), dura mater, and the spinal canal vessels.

Disc Degradation: 3 Types (See blackboard for illustrations)
1. Protrusion / Bulge (Contained)
Bulges outward through a tear in the AF, but **does not escape** from the outer AF or the PLL. Bulges against the PLL and dura generally producing dull, poorly localized pain in the lower back and SI region. Pain is worse in the morning due to imbihition. Generally no leg symptoms. Pain is worse with sitting, because the noiceptors within the AF are irritated by the protrusion.
2. Extrusion
Nuclear material remains attached but **escapes** the AF or PLL. Extrusion is generally posterolateral in nature into the IVF. Patient will have the same presentation as with a protrusion, though leg pain will generally be present and the pain levels maybe greater.
3. Sequestration
The migrating disc material escapes the disc all together and becomes a **free floating fragment.** This fragment has potential to migrate up and down the central canal.

Intervertebral Foramen (see blackboard for illustration)

Contents: spinal nerve, lymphatic's, vascular supply, and adipose tissue.
Borders: inferior articular process, pedicle, superior endplate, disc, inferior endplate, pedicle, superior articular process.

Medial vs. Lateral Protusion: protrusion is named in reference to the nerve root.
1. Patient's with a **LATERAL protrusion** will have increased PAIN when they lean INTO the side of the lesion and have antalgia away from the side of pain.
2. Patient's with a **MEDIAL protrusion** will have increased PAIN when they lean AWAY from the side of the lesion and have antalgia toward the side of pain

Cervical Discs
5 Cervical disc levels are present. NP consists of no more than 25% of the disc as compared to 50% in the lumbar spine. Does not have successive lamella with alternating pattern as in the lumbar spine. Protected by uncoverterbal joints, which reinforce the posterolateral aspect of the disc. Significant cervical disc herniations can lead to lower extremity involvement (myleopathy). As much as 10-15% of the asymptomatic patient has a cervical disc protrusion, depending on age. Compare that to 30-50% of asymptomatic patients have a lumbar disc protrusion.

ARM OR LEG PAIN
Common Causes of Extremity Pain
1. Local Lesion: contusion, spasm, strain
2. Referred Pain: myotagenous, dermatongenous, scleratagenous
3.**Claudication:** Intermittent claudication usually refers to cramplike pains in the legs (usually the calf muscles, but may be in the thigh muscles) caused by poor circulation of the blood to the leg muscles.
4. Peripheral Nerve Lesions
5. Plexopathies
a. Lumbar
b. Cervical: Thoracic Outlet Syndrome (TOS)

Claudication
Reproducible ischemic muscle pain. Cramplike feeling in the leg
1. Vascular Claudication
Peripheral Artery Occlusive Disease
Atherosclerosis is major cause. Thrombus formation. Occurs during physical activity. Intermittent in nature. Relived with rest. Pain develops due to inadequate blood flow. Generally bilateral in nature. Peripheral color changes noted. Decreased or absent pulses in the lower extremity
2. Neurogenic Claudication
Pain is generally unilateral in nature. Occurs in the lower extremity. Pain is decreased when laying down. Burning sensation. Normal pulses. Produced from space occupying lesion generally (disc, tumor, osteophyte)

Peripheral Nerve Lesions
Impaired Peripheral Nerve Integrity and Muscle Performance. Can mimic disc lesions
Pattern: decreased muscle strength, impaired proprioception and sensory integrity, difficulty with manipulation skills. Patient will describe the sensation as-numbness, tingling, pins and needles.

Common Pathologies
1. Carpal Tunnel Syndrome (ulnar nerve)
2. Cubital Tunnel Syndrome (ulnar nerve)
3. Radial Tunnel Syndrome (radial nerve)
4. Tarsal Tunnel Syndrome

Nerves
Upper Extremity Peripheral Nerve Commonly Entrapped
1. Radial nerve
2. Ulnar nerve
3. Median nerve
Lower Extremity Peripheral Nerves
1. Sciatic
2. Deep Peroneal

3. Tibial Nerve

BRACHIAL PLEXUS

1. Radial Nerve (aka Musculospinal nerve)

C5 - T1 (continuation of posterior cord). Largest nerve of Brachial Plexus. Most commonly injured nerve

Innervates:

a. Triceps: C6, C7, C8. Extension of forearm; long head steadies head of abducted humerus

b. Anconeus: C7, C8, T1. Assists triceps in extending forearm; stabilizes elbow joint; abducts ulna during pronation

c. Brachioradialis

d. Extensor Carpi Radialis longus

e. Abductor Pollicus Longus: C7, C8. Abducts thumb and extends it at carpometacarpal joint

Lesion of Radial Nerve:

Wrist Drop inability to: straighten (extend) wrist, inability to abduct thumb therefore see adduction of thumb, loss of sensation especially dorsum web of hand dorsum of hand (not including fingernails), loss (decrease) triceps and brachioradialis reflex

2. Ulnar Nerve

C8 – T1 (from Medial Cord). Sensory to little finger and ½ of ring finger.

Innervates:

a. Abductor digiti minimi: abducts little finger

b. Adductor Pollicus: adducts thumb

c. Flexor Digiti Minimi Brevis: flexes proximal phalanx of little (5th) finger

d. Flexor Carpi Ulnaris: flexes and adducts hand (at wrist)

e. Flexor Digitorum Profundus:

-medial part: ulnar nerve (C8 and T1)

-lateral part: anterior interosseous branch of median nerve (**C8** and T1)

Flexes distal phalanges at DIP joints of medial four digits; assists with flexion of hand

Ulnar nerve sensory distribution: 5th and half of the 4th digits on both, the palm and dorsum of the hand.

American Academy of Orthopedic Surgeons
Cubital Tunnel Syndrome: compression of Ulnar nerve within cubital tunnel. Tendinous arch connecting the humeral and ulnar heads of the flexor carpi ulnaris muscle. Numbness and tingling little finger and ulnar half of 4th digit. This discomfort often is accompanied by weakness of grip (ulnar side of hand). 2nd most common compressive neuropathy. Affects men 3-8 times > women
Causes
Frequent bending of the elbow--pulling levers, reaching, or lifting. Constant direct pressure on the elbow over time may lead to cubital tunnel syndrome. Irritation from leaning on the elbow while you sit at a desk or from letting the elbow rest during a long drive or while running machinery. Damage from a blow to the cubital tunnel.

Pisiform / Hamate Syndrome (aka Guyon's Canal Syndrome)
Ulnar nerve compression w/in tunnel of Guyon (formed by the pisiform and hamate bones). Pins and needles in ½ of the 4th and the 5th digits →burning pain in the wrist and hand and decreased sensation. Weakness in muscles innervated by ulnar nerve → difficulty abducting/adducting fingers, adducting thumb thus affecting ability to pinch. Can see problems with power grip.

Froment's Sign
Paralysis of adductor Pollicis → **Froment's sign.** Grasping a piece of paper between the tip of the thumb (with the IP joint extended) and the radial side of the second digit. Froment's sign is considered positive if the person flexes the thumb's IP joint (contraction of Flexor Pollicis longus). When gripping paper between thumb and index finger in order to maintain hold on the paper when examiner tries to pull the paper from the person's fingers.

Lesion of Ulnar Nerve

Claw Hand deformity (Hand of Benediction). Paralysis and atrophy of: flexor carpi ulnaris, part of flexor digitorum profundus, hypothenar muscles and interosseous muscles except first two lumbricals. Paresthesia medial forearm fingers 4 and 5 atrophy hypothenar eminence and finger 5

3. Median Nerve
C5 - T1 (union of lateral and medial cords)
Innervates:
a. Pronator Teres: pronates and flexes forearm (at elbow). Entrapment site for the Median Nerve. Can mimic Carpal Tunnel Syndrome
b. Flexor Carpi Radialis
c. Palmaris longus: flexes hand (at wrist) and tightens palmar aponeurosis
d. Flexor Digitorum Superficialis: flexes middle phalanges at PIP joints of medial four digits; acting more strongly, it also flexes proximal phalanges at MCP joints and hand
e. Abductor Pollicus Brevis: abducts thumb and helps oppose it.

Median nerve sensory distribution: 1^{st}, 2^{nd}, 3^{rd} and lateral 4^{th} digit on the palmar surface of the hand. 1^{st}, 2^{nd}, 3^{rd}, and lateral 4^{th} to the DIP joints on the dorsum surface.

Median Nerve Dysfunction
Weak finger and hand flexion. Weak pronation. Inability to oppose, flex or abduct the thumb. Weak abduction and pronation of wrist, wrist will deviate to ulnar side. Sensory loss fingers 1-3, 1/2 of 4, tips of fingers (including the nails)

Ape Hand Deformity
Is the result of median nerve palsy leading to wasting of the thenar eminence. The thumb falls back in line with the fingers as a result of the pull of the extensor muscles and the individual is unable to oppose or flex the thumb.

Carpal Tunnel Syndrome

Compression of the median nerve within the carpal tunnel. Flexor tendons. Repetitive flexion and extension.
Borders of Carpal Tunnel: scaphoid, trapezium, pisiform and hook of the hammate, flexor reticaculum.

Signs/Symptoms of Carpal Tunnel Syndrome

Pain, numbness, tingling, itching → palm of hand, thumb, index , middle finger, and ½ ring finger. Night pain (flexed wrists) eased by shaking the hand, changing hand position. Numbness when bringing fingers together in a pinching motion. Clumsiness with precision grip. Feeling of swelling. When chronic will begin to demonstrate: thenar atrophy. Difficulty differentiating between hot and cold by touch.

Carpal Tunnel Syndrome

Causes:
1. Anterior lunate
2. Swelling, fluid retention during pregnancy or menopause, rheumatoid, trauma, injury, sprain, fracture etc.
3. Separation of radius and ulna affecting pronator teres
4. Repetitive stress
5. Repeated use of vibrating hand tools
6. Cyst or tumor in the tunnel.

Evaluation of Carpal Tunnel Syndrome

1. Inspection
2. Palpation
3. Senory Evaluation
4. Tests: Tinel's, Phalen's, Prayer's sign, reverse Phalen's, Opponens pollicus muscle test

Pronator Teres Syndrome

If there is edema or hypertrophy of the pronator teres muscle the Median nerve can become entrapped as it passes between the superficial and deep portions of this muscle.
Repetitive pronation/ supination, achiness and fatigability of muscle with use, rest decreases symptoms. Tenderness of Pronator

teres muscle. Paresthesia fingers 1 – 3, weakness of opposition, finger flexion and making a fist. Pain with resisted pronation, weakness of pronator teres **if** entrapment occurs at or above the elbow before Median nerve passes through muscle (pronates/flexes elbow). If compression of Median nerve is within the muscle, then muscle itself will be fine and neuropathy will show distal. Positive Phalen's.

LUMBAR PLEXUS

Piriformis Syndrome (Sciatic Nerve)

Entrapment of the Sciatic Nerve as it passes under the Piriformis Muscle. *Etiologies:* 1)Direct or indirect trauma to the gluteal or SI region. 2)Flexion contracture of the Hip producing pelvic obliquity. Gender: females 6:1 ratio to men. Spasm or inflammation of the Piriformis muscle.

Deep Peroneal Nerve Entrapment (aka - Anterior Tarsal Tunnel Syndrome)

Etiology: 1)Contusion of the terminal branch of the deep peroneal nerve below the posterior tibialis and extensor hallicus brevis tendons. 2)Compression from tight footwear, ganglion cyst, pes cavus or direct trauma to the region.

Tarsal Tunnel Syndrome

Tibial nerve entrapment through the tarsal tunnel (located between the flexor retinaculum and medial malleolus). *Etiology:* post-traumatic, neoplastic, inflammatory, secondary to fluid retention or valgus foot derformity.
Physical examination for tarsal tunnel: positive Tinnel's Foot Sign, pain with passive dorsiflexion or eversion, heel deformity, valgus or varus in nature, weakness of the foot intrinsics. Normal neurologic examination

Plexopathies

1. Cervical Plexopathy
a. Brachial Plexus Involvement
b. Thoracic Outlet Syndrome is a prime example

2. Lumbosacral Plexopathy
a. Direct Compression
b. Tumor or Pelvic mass: diabetes, pelvic surgery

Thoracic Outlet Syndrome
Collection of signs and symptoms to include pain, tingling, numbness and or coldness in the hand. Most noted on the Ulnar side of the hand

1. True Neurologic TOS
The disorder is rare and is caused by congenital anomalies (unusual anatomic features present at birth). It generally occurs in middle-aged women and almost always on one side of the body. Symptoms include weakness and wasting of hand muscles, and numbness in the hand.

2. Arterial TOS
Occurs on one side of the body. It affects patients of both genders and at any age but often occurs in young people. Like true neurologic TOS, arterial TOS is rare and is caused by a congenital anomaly. Symptoms can include sensitivity to cold in the hands and fingers, numbness or pain in the fingers, and finger ulcers (sores) or severe limb ischemia (inadequate blood circulation).

3. Venous TOS
Is also a rare disorder that affects men and women equally. The exact cause of this type of TOS is unknown. It often develops suddenly, frequently following unusual, prolonged limb exertion.

4. Traumatic TOS
May be caused by traumatic or repetitive activities such as a motor vehicle accident or hyperextension injury (for example, after a person overextends an arm during exercise or while reaching for an object). Pain is the most common symptom of this type of TOS, and often occurs with tenderness. Paresthesias, sensory loss, and weakness also occur. Certain body postures may exacerbate symptoms of the disorder. The lower 2 nerve roots of the brachial plexus, C8 and T1, are most commonly (90%) involved, producing pain and paresthesias in the ulnar nerve distribution. The second most common anatomic pattern involves the upper 3 nerve roots of the brachial plexus, C5, C6, and C7, with symptoms referred to the

neck, ear, upper chest, upper back, and outer arm in the radial nerve distribution

Neurologic
Pain, particularly in the medial aspect of the arm, forearm, and the ring and small digit. Paresthesias, often nocturnal, awakening the patient with pain or numbness. Loss of dexterity. Cold intolerance. Headache

Venous
Pain, often in younger men and often associated with strenuous work

Arterial
Pain. Claudication. Often in young adults with a history of vigorous arm activity

Physical Examination
In most cases, the physical examination findings are completely normal. The sensory examination is often unreliable. Orthopedic tests such as the Adson, costoclavicular, and hyperabduction maneuvers, are unreliable, although Roo's test (EAST - elevated arm stress test) is the most reliable test available. It evaluates all 3 types of thoracic outlet syndrome (TOS).

Lumbosacral Plexopathy
1. Direct Compression: tumor or pelvic mass.
2. Diabetes
3. Pelvic Surgery

MUSCLE WEAKNESS

General Weakness
1. Depression: tiredness, fatigue, sense of worthlessness, lack of other organic symptoms
2. Infection: fever, lymph nodes
3. Hormonal
4. Metabolic: tiredness, fatigue upon exertion – anemia
5. Chronic Fatigue syndrome see criteria in Souza

Specific Weakness
1. Neurologic: check distal extremities
2. Muscular: check larger more proximal muscles (Gluteals, shoulder girdle)
3. Myoneural: evaluate tiredness, cranial nerves

Terminology
Myopathy: disease of skeletal muscle not caused by nerve disorders. The skeletal or voluntary muscles become weak or wasted. Can be inherited, inflammatory, endocrine related, chemical poisoning. Usually degenerative and progressive (muscular dystrophy)
Myelopathy: disturbance or disease of the spinal cord. Results in loss of sensation and mobility. Canal stenosis can lead to myelopathy. Cervical spondylotic myelopathy is the most common cause of spinal cord dysfunction in the elderly.
"Spondylosis" is a general term encompassing degenerative disease affecting the vertebra, disc, and surrounding ligaments.
Signs and Symptoms: neck pain, problems with balance, numbness in the hands and fingertips, and difficulty with fine movements like buttoning a shirt. Possibly exaggerated reflexes.

Hierarchical system of neurons that control voluntary (somatic) muscle activity.
Lower motor neurons → cell bodies in the anterior column of spinal cord → synapse directly with muscle fibers. (LMN's innervating the head have cell bodies in the respective cranial nerve motor nuclei in the brain stem.)
Upper motor neurons → cell bodies in cerebral cortex and other supraspinal nerve centers → influence lower motor neurons either with direct synaptic contact or through interneurons. Neurons of the cerebellum and the basal ganglia influence the upper motor neurons.

Lower Motor Neuron

Originates from axons of the anterior horn out to the periphery and includes the myoneural junction. Motor neurons that innervate voluntary muscles and cranial nerves located in the brainstem. Known as the Final Common Pathway , the final link between the CNS and voluntary muscles.
Examples of LMNL's:
IVF encroachment, TOS, Disc herniation, Canal Stenosis, Piriformis Syndrome, Subluxation, Bell's Palsy, ALS, Traumam, Myasthenia Gravis, Polio, Neuroma, Diabetic Neuropathy

Upper Motor Neuron
The neurons of the corticospinal tract project directly from the motor cortex, and are topographically represented on the precentral gyrus of the cerebral cortex. Cerebral cortex through brainstem down to **BUT NOT** including the anterior horn of the spinal cord. Cell bodies (of Betz Cells) of the Precentral gyrus initiate motor action. Axons then travel through the internal capsule as the *Corticobulbar tract* (to Cranial nerve nuclei) and as the *Corticospinal tract* (Pyramidal tract).
The Corticospinal tract eventually: decussates (80% of it) in the medullary pyramids to form the Lateral Corticospinal tract (movements of the limbs – execution of skilled movements). The rest continues ipsilateral as the Anterior Corticospinal and decussates in the anterior white commisure. The Corticospinal Tract is known as the *Pyramidal System* because of its decussation in the pyramids.
Examples of UMNL's
1. Bruising/trauma to spinal cord, Cerebral Palsy, Multiple Sclerosis, Stroke (CVA)
2. Head trauma, Syringomyelia, Tumor (brain, cord), Brain infection (encephalitis),
3. Hematoma, subdural, Cerebral Aneurysm, ALS

The Motor Homonculus
The areas assigned to various body parts on the cortex are proportional not to their size, but rather to the complexity of the movements that they can perform. Example: the areas for the hand

and face are especially large compared with those for the rest of the body. The speed and dexterity of human hand and mouth movements → ability to use tools and the ability to speak.

Clinical Findings LMNL versus UMNL

LMNL		**UMNL**
Flaccid*****	**Paralysis**	*****Spastic (weak muscles)
↓, absent	**DTR**	↑, hyper
Altered, absent	**Superficial Reflexes**	altered, absent
Absent	**Pathological Reflexes**	*****Present
None	**Clonus**	*****Present
Hypotonic	**Tonicity**	Hypertonic
Yes*****	**Atrophy**	None (or disuse)
Yes*****	**Fasciculations**	None
Present*****	**Trophic Changes**	None

Fasciculations = visible spontaneous contraction of groups of muscle fibers (when anterior horn itself is involved)

CEREBELLUM

The cerebellum is concerned with coordination (of motor activity), fine control of motion and postural reflexes. The cerebellum stores learned sequences of movements, it participates in fine tuning and co-ordination of movements produced elsewhere in the brain, and it integrates all of these things to produce movements so fluid and harmonious that we are not even aware of them.

1. Coordination of motor activity and fine movements. Evaluates how well motor movements from cerebral cortex are being carried out. If there is a problem the cerebellum detects it, and sends feedback information to motor areas so that corrections can be made. Receives input from pons (relaying information from cerebral cortex) specifying goals of intended movement, the cerebellum then calculates specific sequence of muscle contractions required to achieve movement goals. Range, velocity and strength of muscle contraction. Procedural memory - route of puzzle, riding a bike

2. Maintenance of posture and balance

Via information from CN 8 (Vestibular portion – where body is in space), visual information from the superior colliculus (mid brain)

Signs and Symptoms: Cerebellar dysfunction

Hypotonia: loss of resistance offered by muscles to palpation or passive range of motion. "floppy, loose-jointed, rag doll appearance. Patient appears inebriated.

Disequilibrium: loss of balance → trunk and gait ataxia (motor ataxia).

Ataxia = Lack of coordination or clumsiness of voluntary movement (not the result of muscular weakness) due to error in the speed, force and direction of movement.

Cerebellar Ataxia a.k.a. **Motor Ataxia:**

Patient is unstable on feet whether eyes are open or closed. Cannot stand with feet together. Worse when lying down. Gait is wide based, reeling, staggering and drunken in appearance. The patient will tend to lean or stagger towards the side of the cerebellar lesion.

Dyssynergia - loss of coordinated muscle activity. **Rebound Phenomenon of Holmes** (Stewart - Holmes Rebound). The inability to "check" a motion (by antagonist muscle as compensatory muscular relaxation does not occur) when resistance is removed. Lose ability to adjust to changes in muscle tension.

Intention Tremors: involuntary tremors which begin upon initiation of voluntary movement, may be intensified at termination of the movement

Dysdiadochokinesia: the inability to perform rapidly alternating movements**.**

Dysmetria = Past Pointing Phenomenon . The patient is unable to estimate distances accurately, overshoots intended goal. Inability to stop muscular movement at desired point. Intention tremor is considered a type of dysmetria. Lack of smooth voluntary motions. The breaking down of a smooth muscle act into a number of jerky awkward components.

Nystagmus: involuntarily rhythmical (either vertical, horizontal or circular) motion of the eye. Often gets worse when the patient looks to the side of the lesion.
Dysarthria (slurred or scanning speech)**:** lack of coordination of the muscles used in speaking. Hesitant, slurred, and explosive speech with pauses in the wrong places.

BASAL GANGLIA

The basal ganglia is composed of paired nuclei and is part of the Extrapyramidal System that serves to modulate and adjust the tone of motions initiated by the pyramidal system.
Basal Ganglia (extrapyramidal) **Disorders** are characterized by:
Dyskinesia: involuntary movements. Tremors, choreiform movements, ballism, athetoid movements and dystonia. Alterations in muscle tone. Disturbances in postural stability. Bradykinesia
Resting Tremors (the ONLY Basal Ganglia Tremor). Seen in Parkinsons - degeneration of the Substantia Nigra of the Basal Ganglia. Present at rest usually disappear or diminish with action. Seen in hands (pill-rolling), and feet
Chorieform Movements: rapid, abrupt, highly complex jerky movements that appear to be well coordinated but are performed involuntarily. Affecting fingers, hands, arms, face, tongue, head
Athetoid Movements: involuntary, ceaseless,irregular, slow continuous writhing wormlike motions. Seen more commonly in the hands and fingers - wriggly and crawly
Rigidity: increased muscle tone, increased resistance to passive movement.
a. Cogwheel Rigidity - has underlying tremor related. Seen in Parkinsonsb.
b. Lead pipe rigidity smooth, no underlying tremor
Hypotonia: decreased muscle tone
Disturbances in Postural Reflexes: inability to recover balance quickly - patient falls when attempting to walk or stand. Lack of fluidity in movement

Huntington's Chorea

Hereditary disease, autosomal dominant characterized by abnormal facial and limb movements. Gradual onset between 25 - 55 years of age, male or female. Degeneration of neurons in the cerebral cortex and basal ganglia which leads to: chronic progressive chorea, mental retardation, emotional disturbances, dementia.
Symptoms include: fidgety, clumsiness, restlessness, irritability and moodiness
Progresses to: choreiform movements, abnormal gait, postural changes, personality changes (paranoia, hostility, agitation, hallucinations, apathy, depression), impairment of recent memory and intellectual function. Death usually results 5 - 10 years after onset due to pulmonary, cardiac or trauma related incidence. At end of illness patient is bedridden, mute,immobilized. Genetic counseling is recommended before starting a family

Parkinson's a.k.a. Paralysis Agitans, Shaking Paralysis
Degeneration of the substania nigra with subsequent decrease in Dopamine production. Idiopathic disorder, onset 50 – 60 y.o.a.
Characterized by: tremor, hypokinesia (bradykinesia), rigidity, abnormal gait and posture. Resting Tremors. Muscular Rigidity: increased muscle tone, increased resistance to passive movement. Cogwheel Rigidity and Lead pipe rigidity

Slowness of Movement (Hypokinesia or Bradykinesia)
Difficulty initiating voluntary movements. Mask like facial expression, decreased blinking, staring. Slow speech, soft voice **(hypophonia)**. Impaired handwriting reverting to small print – **micrographia.** Difficulty with rapid repetitive movements. Difficulty rising from a chair, turning over in bed
Abnormal Gait and Posture. **Muscle rigidity** leads to postural deformities: head held in flexion, stooped trunk, arms held in towards body with elbows in flexion. **Personality changes:** passive, dependent, fearful, indecisive depressed, dementia

POSTERIOR (DORSAL) COLUMNS
Mostly ascending tracts of sensory information: vibration, position sense (proprioception), pressure, discriminatory touch. Evaluate

Topognosis, two-point discrimination, Pallesthesia (vibration), Romberg's, Position Sense.

Tabes Dorsalis

Tertiary Neurosyphilis. Damages dorsal roots and posterior columns. Loss of proprioception and vibratory sensation. Sensory ataxia. (+) Romberg's

Syringomyelia

An idiopathic disease of the brain stem and spinal cord associated with cavitation (filled with thick yellow fluid). Common in the lower cervical / upper thoracic spine. Onset 30 - 50 years of age. Headache/ shoulder pain. Early loss of pain and temperature (Lateral Spinothalamic tract) in a shawl like distribution with preservation of light touch and proprioception. Preservation of touch and deep pressure. Horner's syndrome may develop. Other forms of Ataxia

FORMS OF ATAXIA

1. Vestibular Ataxia

Stems from problems with the labyrinth of the inner ear, vestibular portion of cranial nerve VIII, or brain stem vestibular nuclei. Is gravity – dependent. Lack of coordination of limb movements is not seen in supine position, become apparent when the person attempts to stand or walk. Unilateral nystagmus. Vertigo
Other forms of Ataxia

2. Sensory Ataxia

Caused by lesions in the proprioceptive pathways in peripheral sensory nerves, sensory roots, posterior column of spinal cord or medial lemnisci. Impaired sensation of joint position and movement of affected limb, diminished vibration sense. Ataxia associated with numbness and tingling. Wide based gait otherwise would be very unsteady on feet, cane or holding on makes it easier to walk Slappage – gait. (+) Romberg's Sign = when stand with feet together eyes closed, Unsteady in dark, worse when eyes are closed

SENSORY SYSTEM: Numbness, Tingling and Pain

Sensory Alterations
Paresthesia:
Dysesthesia:

Examination Process
1. History
Define what the patient is feeling. Is it organic or manifestation of a biopsychosocial issue? Attempt to localize the sensation. Distal vs. proximal. Local vs widespread. Are there associated symptoms? Pain, or Weakness
What was the onset? Traumtic vs slow or insidous onset
2. Review of Systems: Diabetes, Thyroid Disease, Alcoholism, Kidney or Liver Disease, Anxiety or Depression, Drug History
3. Evaluation
a. Inspection: Gait analysis, Posture analysis
b. Palpation: Muscle tone (Upper motor vs lower motor neuron lesion). Abnormal responses to touch
c. Range of Motion: Looking for abormal muscle response. Cogwheel or Leadpipe Rigidity
4. Sensory Examination
5. Motor Examination
Signs of Motor Neuron Disease. Nerve Root vs Peripheral nerve issues

Sensory System
Consists of: clinically testable sensations. Pain and temperature. Proprioception and vibration. Touch and deep Pressure
Progressive Myleopathy: lateral lesion will show evidence of ascending loss. Central lesion will have sparing of vibration and joint position sense. What is the etiology of this? **Paresthesias and Dysesthesia:** Damage to Large Fiber Axons. Structural damage to: compressive forces and demylenation (Multiple Sclerosis is prime example)

Numbness

1. True Numbness: clinical findings are present. Result of damage to the sensory fibers: Loss or damage. Affects pain and temperature
2. Perceived Numbness: no neurologic findings are present

Peripheral Neuropathies
1. Axonal Degeneration: normal conduction. Amplitude is decreased
2. Paranodal or Segmental demylenation: velocity is decreased. Amplitude is normal

Painful Neuropathies
1. Small Fiber
Only A δ and C fibers are involved. Most Common in patients > 50 years of age. Generally feet to knees, but not above the knee. Touch is affected. Sparing of proprioception and vibration. Underlying cause rarely determined
2. Large and Small Fiber
Damage to the Small nerve fibers. A β, α as well as δ and C.
Affects: vibration, proprioception, muscle strength, DTR's.
Examples: Diabetic Neuropathy, HIV Related Neuropathy

Complex Regional Pain Syndromes
1. Type I (aka Reflex Sympathetic Dystrophy RSD)
Etiology is severe trauma: fracture, CVA, Myocardial Infarction, Peripheral Nerve Injury
Characterized by: pain and swelling in distal extremities. Unilateral in nature. Vasomotor Instability (Raynaud's Phenomenon). Trophic Skin Changes. Bone Demineralization (Sudek's Atrophy). Aberrant Sympathetic Activity is the etiology
Phase I: pain and swelling. Increase hair growth
Phase II: 3-6 months later. Skin is shinny and cool to touch
Phase III: atrophic skin. Flexion contracture. If this occurs in the shoulder it is called Shoulder- Hand Syndrome

CRPS II aka Causalgia

Similar to CRPS I. Peripheral nerve damage is present. Significant Pain is Present

Clinical Entities
Thoracic Outlet Syndrome
Postural Syndromes

Upper Crossed Syndrome: elevated and protracted scapula.
Winging of the scapula
Head protraction. Produces overstress of cervicocranial junction. C4-5. T4. Can produce chest pain of pseudo angina pectoris.
Glenohumeral joint complex.
You will see increased activity in the upper trap and levator scapula muscles to stabilize the head of the humerus. This produces rotation and abduction of the scapula. This will cause the supraspinatus to be overactive producing degeneration of this muscle.
Tightening and Shortening will be seen with: pectoralis major and minor, upper trapezius, levator Scapula, and SCM
Weakness is seen with: lower and middle trapezius fibers, serratus anterior, rhomboid major and minor .

Lower Crossed Syndrome
Tightened and Shortened Muscles: iliopsoas, rectus femoris, TFL, errector spinae, and
Quadratus Lumborum
Weakened Muscles: abdominal, gluteal muscles

Nerve Root
Numbness on sensory testing. Pattern is discrete and dermatonal . Muscle weakness in myotonal patterns. Reflex changes are seen. Compressive and Distractive testing is positive

Peripheral Nerve
Numbness will follow peripheral nerve on physical examination. Motor less is seen within the specific peripheral nerve. History

reveals some trauma or overuse. Reflex loss can occur if the pressure to the peripheral nerve is significant

Central Nervous System
Bilateral presentation is usually seen. May affect patients perception of temperature. If spotty think MS. Will see tract signs

Metabolic
Numbness is diffuse. Does not follow dermatonal patterns. Bilateral and distal . No motor findings are present. Constitutional Symptoms are present

GOAL WRITING FOR CHIROPRACTORS

Goals Must Be
Clearly defined. Have a definite time limit. Observable and measurable. Explained to the patient . Updated after a change is determined in the file: new condition, re-assessment, re-physical

Goal Structure
Goals should be patient-centered functional goals when at all possible. Reason: patients make the greatest gains when the care and the goals focus on an activity that are meaningful to them.

Goals consist of 2 types:
1.Short term
2.Long term
All goals should follow a specific recipe

Functional Goal Recipe
Goal =A+B+C+D+E
A= Actor
B= Behavior
C= Condition
D= Degree
E= Expected Time

Actor
This is the WHO of the goal. Who will carry out the activity. In outpatient care this generally the patient. " The patient will …"

Behavior
The WHAT of the goal. The description of the activity in understandable terms.
"… will be able to sit …." "..will be able to walk …"

Condition
Circumstances under which the behavior is carried out. Includes all essential elements of the performance. "…at her desk …" "... in the car…"

Degree
Quantitative specifications of performance. Expressed in measurable terms.
" … 4/10 to 2/10 …" " … from 56% to 30 % "

Expected Time
The How of the Goal. Need to have specific time frame. How long it will take to reach the goal. Stated in days, weeks, months or number of visits. "… by 1st reassessment …"
" …. within 2 weeks…" " … by rephysical…"

Short Term Goals
Interim steps for achieving long term goals. Must contain a specific time span. Basis for care planning. Specific care regime is designed to achieve care plan goals. Specific time span is set to meet these goals.
<u>Short Term Goal Examples:</u>
1. Patient should be able to sit at her desk for 2 hours with pain no greater than 2/10 by 1st reassessment or within one month, whichever comes first.
2. Patient will demonstrate proper lifting technique and work ergonomics by 1st reassessment or 1 month, whichever comes first.

3. Decrease patient's pain level when grocery shopping from 4/10 to 2/10 by rephysical or 3 months, whichever comes first.

Long Term Goals

The long term outcome of the care → The Final Product.
Established at Initial Visit
Based on the problem list. Each problem should have a goal associated with it. The basis for setting your short term goals.
Usually described in functional terms.
Long Term Goal Examples:
1. Patient should be able to lift 50 pounds and perform his job duties without assistance by re-physical or 3 months, whichever comes first.
2. Patient will demonstrate balanced posture in seated and standing position by 2nd re-physical or 6 months, whichever comes first.
3. Patient will demonstrate bicep muscle strength rated at 5/5 by 1st re-physical.
4. Patient will be able walk around the grocery store for 45 minutes with no pain by 2nd re-physical or 6 months, whichever comes first.

Goal Exceptions

It is best to write a functional goals for your patient, though there will be times that there is no functional loss. In this case, the following elements are still maintained: Actor, Degree, and Expected time
For Example:
The patient will decrease their Neck Disability Index scores from 56% to 22% by 2nd re-assessment or 6o days, whichever comes first.

Goals are primarily written for the Acute Intensive and Reconstructive part of the Chiropractic Lifestyle as defined by Life University. When a patient has NO subjective or objective findings nor functional loss, goal writing can be more difficult. In this case we are truly working to maintain the patient's current level of function.

In this case a goal could read: Maintain the patient's current level of function, as well as monitor patient's progress via Outcome Assessment Tools, Subjective Intake, Palpatory findings and Re-Assessment data.

RED FLAG SIGNS

Presentation age
1. Less than 20 years of age
2. Children have difficulty perceiving pain. When pain is present this is a significant finding that needs to be investigated especially if noted in children under the age of 11
3. Onset greater than 55 years of age

Previous history of: carcinoma, systemic steroids use (prolonged), drug abuse, HIV

Widespread Neurology
Neurologic symptoms overlapping more than one dermatone.

Abnormal Pain
Constant progressive non-mechanical pain. Pain that is not altered by position or medication. Severe pain with no history of trauma. Pain that wakes a patient from a sound sleep

Constitutional Symptoms
1. Weight loss: (10-15 pounds in 2 weeks or less) or 10 % Body weight in 30 day period
2. Fever
3. Chills
4. Night sweats

Cauda Equina Syndrome
Inability to empty bladder. Loss of the anal sphincter tone. Fecal incontinence. Saddle anesthesia around the anus, perineum or genitals. Widespread (>one nerve root)or progressive motor

weakness in the legs or gait disturbance. Unilateral leg pain worse than low back pain

References
•The Back Pain Revolution: ,2nd ed, Gordon Wadell: Churchill Livingston
•Orthopedic Physical Assessment, 4th ed, David J. Magee: Saunders
•APHCR Guidelines: Acute Low Back Pain Problems in Adults: An Assessment and Quick Treatment Reference Guide for Clinicians: Clinical Practice Guideline #14

REFERRED PAIN

Sources of Referred Pain
1. Myotomal
2. Dermatonal
3. Scleratomal
4. Visceral
5. Osseous

MYOTOMAL PAIN
Pain referred from muscle source. Referred to as myofascial pain

Symptoms terms commonly used in the literature:
Muscular Rheumatism
Myalgia / Myalgic Spots
Myofascitis
Myofascial Pain Syndrome

Definition: a hyperirritable locus within a taunt band of skeletal muscle, located in the muscular tissue or the surrounding fascia. The spot is painful on compression and can evoke characteristic referred pain and autonomic phenomena. Myofascial trigger point (MTP) is either active or latent. Both active and latent MTP cause dysfunction, only active produces pain.

Active MTP
Causes the patient active pain. Pain maybe far from the site of the MTP. Restriction of movement and weakness of the affected muscle will be present.

Latent MTP
Clinically silent with respect to pain. May cause restriction of movement and weakness of the affected muscle. May persist for years after injury to an area.

Etiology
MTPs are thought to be brought on after:
1. Microtrauma
2. Macrotrauma
3. Sustained muscular contraction due to postural dysfunction

One recent theory being investigated is that they are intrafusal muscle fibers that are sensitive to adrenaline production and sympathetic stimulation. Remember that skeletal muscle is extrafusal muscle fibers.
MTPs become a site for sensitized nerves with altered metabolism.
Stimulation of these receptors can cause:
1. Localized ischemia to muscles and nerves. Results in bombardment of the nervous system with abnormal impulses creating hyrperalgesia in the segmentally related muscles and referral zone
2. Edema
3. Fibrosis
4. Temperature Changes
5. Focal or regional autonomic dysfunction
6. Vasoconstriction
7. Persistent hyperemia after palpation

Examination findings
Active and Passive stretch of a muscle with a MTP present will be painful to the patient within the muscle. The stretch ROM is reduced. Maximum contractile force is decreased. Deep tenderness

and dysesthesia are commonly referred by active MTPs to the zone of referred pain. Muscles in the immediate vicinity of a MTP will feel ropy or taunt. Moderate sustained pressure on a sufficiently irritable trigger point causes or intensifies pain in the reference zone for that given trigger point. In some patients dermographia or panniculosis (broad, flat thickening of the subcutaneous tissue) in the area overlying the active MTP is present . " Jump Sign" is present. Local pain is out of proportion of the pressure applied. This can be so intense that it causes the patient to " jump" and note significant pain. Local Twitch Response. Rolling palpation over a MTP causes the muscle in question to twitch.
Understanding key referral patterns for common MTP essential. Many patients will present with myofascial complaints and these need to be differentiated from plexopathies, neuropathies and radiculopathies. No neurologic findings will be present with MTP.

Fibromyalgia

Fibromyalgia is a clinical syndrome defined by chronic widespread muscular pain, fatigue and tenderness. Many people with fibromyalgia also experience additional symptoms such as fatigue, headaches, irritable bowel syndrome, irritable bladder, cognitive and memory problems (often called "fibro fog"), temporomandibular joint disorder, pelvic pain, restless leg syndrome, sensitivity to noise and temperature, and anxiety and depression. These symptoms can vary in intensity and, like the pain of fibromyalgia, wax and wane over time.

What causes Fibromyalgia?

There is no know cause. It is known however that people with fibromyalgia can have abnormal levels of Substance P in their spinal fluid. Genetics also appear to play a role leading to a familial tendency to develop fibromyalgia. It is not a disease, but rather a syndrome with a common set of characteristic symptoms including widespread pain and the presence of a defined number of tender points.

Criteria for Fibromyalgia

1. Patients must exhibit tender points in 11 or more out of 18 standardized sites
2. A tender point is defined as a point that becomes PAINFUL and not just tender when approximately 4 kg of pressure is applied to the spot
3. Prevalence is 10-20 times greater in women than in men. The reason for this is unknown.

DERMATONAL

Arises from Nerve Root. Follows given dermatonal pattern. Sharp, shooting, tingling, numbness. Very localized in nature

SCLERATOMAL

Non Dermatomal pain pattern. Crosses Dermatomal patterns. No hard neurologic findings. Normal muscle strength. Normal reflex evaluation. May have abnormal sensory findings.
Generally referred from:
1. Facet
2. Disc referred pain without nerve root involvement
Pain is non descript in nature. Patients will describe the pain a " deep dull ache"

VISCERAL PAIN

Characteristically visceral pain is different from that of somatic pain. There maybe overlapping symptoms. Visceral afferent receptor serve a function different from that of somatic receptors. Somatic receptors signal the body about external threats and therefore need to have a high degree of localization. Visceral receptors are designed more to provide information about homeostasis.
Often Central in nature. Reflects embryologic arrangement of most thoracic and abdominal organs. As organs migrate the brain does not get their forwarding address, therefore it is represented as central or midline pain. Overlap of sensory innervation to organs that are geographically related. The main stimuli in visceral conditions is distention, inflammation or ischemia.
Example:

1. The upper 2/3 of the esophagus, lungs, trachea, bronchi and the heart is innervated by spinal cord segments T1-T4 or T5.
2. Overlap continues with the lower 1/3 of the esophagus, stomach, duodenum, liver, gallbladder, pancreas and part of the small intestine all innervated by spinal segments T5-T10.
3. Pathology in one structure is difficult to distinguish from another structure by the brain.

OSSEOUS PAIN

Pain that originates from bone

1. Tumor
2. Infection
3. Fracture

Pain is very deep. Commonly worse at night. If stress fracture is present there is a history of overuse.

CHEST PAIN

Common Causes of Chest Pain

1. Musculoskeletal
2. Esophageal
3. Cardiac / Pulmonary
4. Infectious
5. Referred

Normal Anatomy

Right lung: has 3 lobes
Left lung: has 2 lobes

Auscultation

Normal breath sounds. Made by flow of air through respiratory tree

1. Vesicular	Most of lung fields: healthy tissue, low pitched
2. Bronchovesicular	Main bronchi, medium pitched
3. Bronchial	Trachea, high pitched

Affect of Pathology
Air, pus, effusion or shallow breathing secondary to pain will cause decreased sounds.
Consolidation/pneumomia→ ↑ transmission of sound→ inc. sound, bronchophony, whispered pectroliloquy, egophony

Normal Sounds :
1. Tympany→ gastric air bubble
2. Resonant→ lung
3. Hyperressonance→ air filled lungs
4. Dull→ organs
5. Flat→ muscle, bone

Percussion
Produces vibration and sound waves, comparison of sound. The sound is determined by the density of the tissue. From loud to soft: air, fluid, solid

Pleural effusion and Consolidation- dullness
Chronic Emphysema and Acute Pneumothorax – hyperresonance

Pleural Effusion
Fluid collects in the potential space that exists between the lung and the chest wall, displacing the lung upwards.
<u>In the presence of effusion:</u> fremitus over an effusion will be decreased. Percussion typically will elicit dullness. Auscultation will have decreased sounds (muffled or absent)

Tactile Fremitus
Normal lung transmits a palpable vibratory sensation to the chest wall. This is referred to as fremitus and can be detected by placing the ulnar aspects of both hands firmly against either side of the chest while the patient says the words "Ninety-Nine." This maneuver is repeated until the entire posterior thorax is covered. The bony aspects of the hands are used as they are particularly sensitive for detecting these vibrations

Lung consolidation

Normally air filled lung becomes filled with fluid or tissue, ex. - pneumonia. If a large enough segment of the lung is involved, it can alter the transmission of air and sound.

In the presence of consolidation: fremitus becomes more pronounced. Percussion will be dull. Auscultation will be clearer

Pneumonia

Inflammatory response→exudates (fluids, cells, or other cellular substances that are slowly discharged from blood vessels usually from inflamed tissues)→lung consolidation→ dyspnea, crackles, altered breath sounds, dullness with percussion

Symptoms: tachypnea, dyspnea, shallow breathing, flaring alae, muscle guarding and decreased movement, possible fever and malaise, possible chest pain at level of the pneumonia.

Exam: increased fremitus (consolidation), dullness upon percussion, crackles, bronchial sounds(↑ breath sounds, clarity), bronchophony etc.

Rib Fracture

Difficulty with inspiration. ↑ pain in supine position. (+) compression. Tuning fork increases pain. X-ray. Pathology osteoporosis.

Herpes Zoster

Unilateral pain, in band following a dermatomal pattern. Varicella virus. Fluid filled vesicles within 3 –5 days onset of pain

Costochondritis

Younger patient. Chest pain that is bilateral in nature. Middle ribs (2-5) close to sternum. Tenderness, (-) swelling. (+) Compression increases pain

Teitze's Syndrome

Women > 50 years of age. Moderate to severe pain. Upper chest (2nd/3rd ICS). Unilateral in nature. Overexertion, coughing→ inflammation. Tenderness and swelling is noted

Pulmonary Embolism
Middle Aged Male. Sudden Chest pain after pain in the calf. Low grade fever is present. Pleuritic Pain with out dyspnea. Pain is severe and similar to a Myocardial Infarction. Very High Mortality Rate. 600,000 case each year in the US. 1/3 end in death

Pleurisy
Sharp pain in the chest related to coughing , sneezing and positional in nature. Most noted with side bending to the same side or lying on the involved sided. Will often have a history of coexisting or recent history of respiratory infection.

Chest Pain Cardiac Origin

Angina Pectoris
Squeezing or pressure sensation in the Chest. Most noted after exertion. Pain will last up to 30 minutes in general. Rest will decrease the symptoms. Secondary to atherosclerosis.
More likely to see Angina in ambulatory care than MI

Myocardial Infarction
Majority of cases present to the Emergency Room and not seen in ambulatory care. nless the event occurs in your office. Severe substernal pain. Patient may be diaphoretic and in acute distress. Generally lasting over 30 minutes. Secondary to coronary thrombus or vasospasm

Intercostal Neuritis
Unilateral chest pain. Extends around the chest wall.
Causes: Diabetes (because of Neuropathy), Osteophytes, Rib Subluxation

Esophageal

Substernal pain noted with recumbent positions, dysphagia or heartburn. Afferent convergence of nerves from heart and esophagus explain why esophageal pain mimics cardiac pain. 50-60% of chest pain that is of unknown origin is thought to be esophageal in nature.

CAUDA EQUINA SYNDROME & CONUS MEDULLARIS SYNDROME

The spinal cord tapers and ends at the level between the first and second lumbar vertebrae in an average adult. The most distal bulbous part of the spinal cord is called the **conus medullaris**, and its tapering end continues as the filum terminale. The upper border of the conus medullaris is often not well defined. Distal to this end of the spinal cord is a collection of nerve roots, which are horsetail-like in appearance and hence called the **cauda equina** (Latin for horse's tail). These nerve roots constitute the anatomic connection between the central nervous system (CNS) and the peripheral nervous system (PNS).

The conus medullaris part of the spinal cord obtains its blood supply primarily from 3 spinal arterial vessels—the anterior median longitudinal arterial trunk and 2 posterolateral trunks. Less prominent sources of blood supply include radicular arterial branches from the aorta, lateral sacral arteries, and the fifth lumbar, iliolumbar, and middle sacral arteries. The latter contribute more to the vascular supply of the cauda equina, although not in a segmental fashion, unlike the blood supply to the peripheral nerves. The nerve roots may also be supplied by diffusion from the surrounding CSF. Moreover, a proximal area of the nerve roots may have a zone of relative hypovascularity.

injuries to this area often yield a combination of upper motor neuron (UMN) and lower motor neuron (LMN) symptoms and signs in the dermatomes and myotomes of the affected segments. On the other hand, a cauda equina lesion is a LMN lesion because the nerve roots are part of the PNS. Cauda equina and conus medullaris syndromes are classified as clinical syndromes of the spinal cord.

Table 1. Symptoms and Signs of Conus Medullaris and Cauda Equina Syndromes

	Conus Medullaris Syndrome	**Cauda Equina Syndrome**
Presentation	Sudden and bilateral	Gradual and unilateral
Reflexes	Knee jerks preserved but ankle jerks affected	Both ankle and knee jerks affected
Radicular pain	Less severe	More severe
Low back pain	More	Less
Sensory symptoms and signs	Numbness tends to be more localized to perianal area; symmetrical and bilateral; sensory dissociation occurs	Numbness tends to be more localized to saddle area; asymmetrical, may be unilateral; no sensory dissociation; loss of sensation in specific dermatomes in lower extremities with numbness and paresthesia; possible numbness in pubic area, including glans penis or clitoris
Motor strength	Typically symmetric, hyperreflexic distal paresis of lower limbs that is less marked; fasciculations may be present	Asymmetric areflexic paraplegia that is more marked; fasciculations rare; atrophy more common

Impotence	Frequent	Less frequent; erectile dysfunction that includes inability to have erection, inability to maintain erection, lack of sensation in pubic area (including glans penis or clitoris), and inability to ejaculate
Sphincter dysfunction	Urinary retention and atonic anal sphincter cause overflow urinary incontinence and fecal incontinence; tend to present early in course of disease	Urinary retention; tends to present late in course of disease

Signs of Cauda Equina Syndrome

1. Muscle strength in the lower extremities is diminished. leading to diminished strength in the glutei muscles, hamstring muscles (ie, semimembranosus, semitendinosus, biceps femoris), and the gastrocnemius and soleus muscles.
2. Sensation is decreased to pinprick and light touch in a dermatomal pattern corresponding to the affected nerve roots. This includes saddle anesthesia (sometimes including the glans penis or clitoris) and decreased sensation in the lower extremities in the distribution of lumbar and sacral nerves. Vibration sense may also be affected. Sensation of the glans penis or clitoris should be examined.
3. Muscle stretch reflexes may be absent or diminished in the corresponding nerve roots. Babinski reflex is diminished or absent.
4. Bulbocavernosus reflexes may be absent or diminished. This should always be tested.
5. Anal sphincter tone is patulous and should always be tested since it can define the completeness of the injury (with

bulbocavernosus reflex); it is also useful in monitoring recovery from the injury.
6. Urinary incontinence could also occur secondary to loss of urinary sphincter tone; this m ay also present initially as urinary retention secondary to a flaccid bladder.
7. Muscle tone in the lower extremities is decreased, which is consistent with an LMN lesion.

Signs of Conus Medullaris Syndrome

1. Patients may exhibit hypertonicity, especially if the lesion is isolated and primarily UMN.
2. Signs are almost identical to those of the cauda equina syndrome, except that in conus medullaris syndrome signs are more likely to be bilateral; sacral segments occasionally show preserved bulbocavernosus reflexes and normal or increased anal sphincter tone; the muscle stretch reflex may be hyperreflexic, especially if the conus medullaris syndrome (ie, UMN lesion) is isolated; Babinski reflex may affect the extensors; and m muscle tone might be increased (ie, spasticity).
3. Other signs include papilledema (rare, occurs in lower spinal cord tumors), cutaneous abnormalities (eg, cutaneous angioma, pilonidal sinus that may be present in dermoid or epidermoid tumors), distended bladder due to areflexia, and other spinal abnormalities (noted on lower back examination) predisposing the patient to the syndrome.

Causes: The most common causes of cauda equina and conus medullaris syndromes are the following:

1. Lumbar stenosis (multilevel)
2. Spinal trauma including fractures
3. Herniated nucleus pulposus (cause of 2-6% of cases of cauda equina syndrome)
4. Neoplasm, including metastases, astrocytoma, neurofibroma, and meningioma: Twenty percent of all spinal tumors affect this area.
5. Spinal infection/abscess, such as tuberculosis, herpes simplex virus, meningitis, meningovascular syphilis, cytomegalovirus, or schistosomiasis

6. Idiopathic, eg, spinal anesthesia: These syndromes may occur as complications of the procedure or of the anesthetic agent (eg, hyperbaric lidocaine, tetracaine).
7. Spina bifida and tethered cord syndrome
8. Other, rare causes
a. Spinal hemorrhage, especially subdural and epidural hemorrhage causing compression within the spinal canal
b. Intravascular lymphomatosis
c. Congenital anomalies of the spine/filum terminale including tethered cord syndrome
d. Conus medullaris lipomas
e. Multiple sclerosis
f. Spinal arteriovenous malformations
g. Late-stage ankylosing spondylitis
h. Neurosarcoidosis
i. Deep venous thrombosis of the spinal veins (propagated)