An Osteopathic Approach
To The
Chronic Low Back Pain Patient

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Objectives

• Review a definition of chronic pain
• Consider a multifactorial approach to chronic pain
• Discuss the evaluation of a patient with chronic pain
• Develop treatment strategies for such a patient
Chronic Pain

- “Pain is an unpleasant sensory and emotional experience usually associated with actual or potential tissue damage, or described in terms of such damage.” (IASP)

- “Chronic Pain is pain of >3months duration or past the normal time of tissue healing.” CDC Chronic Pain Guidelines 2016
Chronic Pain

• 2012 National Health Interview Study showed that 11.2% of adults report having daily pain (CDC Guidelines)

• 75-85% of population will have back pain

• 10% of these will have chronic back pain
  – Accounts for 90% of costs
    • $20-$50 Billion/annually

• 9.6–11.5 million adults, or approximately 3%–4% of the adult U.S. population, were prescribed long-term opioid therapy in 2005 (CDC)
Chronic Pain

• 1999-2014: Death rate associated with opioid pain medication has increased markedly
  – Increased in parallel with opioid-related overdose deaths

• Opioid use disorder
  – AKA: Abuse, Dependence, Addiction
  – Pattern of opioid use leading to clinically significant impairment or distress
  – Non-control of use and use resulting in social problems and a failure to fulfill major role obligations at work, school, or home
Chronic Pain

- CDC Guidelines
  - CDC Guideline for Prescribing Opioids for Chronic Pain — United States, 2016
  - CDC Website
    - http://www.cdc.gov/mmwr/volumes/65/rr/rr6501e1.htm
    - Recommendations for prescribing opioids for chronic pain in the primary care setting
    - Outside of cancer, end of life, palliative care pain relief
Neurophysiology

• Nociceptors virtually everywhere
  – Skin, fat pads, muscles, ligaments, fascia, joint capsules, periosteum, blood vessel walls.

• Three categories of chemical pain mediators
  – Local nociception inducers
    • Bradykinin, histamine, acetylcholine, …
  – Sensitize nociceptors
    • Prostaglandins, leukotrienes, interleukins, …
  – Produce extravasation of neuropeptides
    • Substance P, Calcitonin gene-related peptide
Neurophysiology

• Nociceptors transmit to **DORSAL HORN**
  – **A delta** – large (1-4 mu), fast (12-30 m/s)
    • sharp localized pain
  – **C fibers** - small (0.1-1.0 mu), slow unmyelinated (1-2 m/s)
    • Diffuse, burning, throbbing, aching
  – **A alpha** (12-20 mu, 70-120 m/s)
  – **A beta** (5-12 mu, 30-70 m/s)
    • Episodic, sharp, stabbing pain.

• All go to substania gelatinosa of the dorsal horn
Neurophysiology

• In the spinal cord
  – Nociception impulses ascend via contralateral spinothalamic tract
    • Some fibers end in medial and lateral thalamic nuclei
  – Others ascend via spinoreticular tracts to brain stem
Neurophysiology

- In the brain
  - A delta sends impulses to neothalamus, and somatosensory cortex
    - Allows for localization and discrimination of type of pain
  - C fibers sends impulses to a variety of locations: brainstem, midbrain nuclei, cortical limbic system.
    - Key locations for behavior modification: conditioned behavior, learned avoidance
    - Useful for survival, has other effects
Neurophysiology

• Central Nervous system
  – Sympathetic stimulation
    • Lowers pain threshold
  – Vasoconstriction
    • Lowers pain threshold
  – ? Norepinephrine may sensitize nociceptors
    • Amplifies pain response, (primarily to new trauma)

• Continued stimulation will “may induce changes in the spinal cord neurons that enhance responsiveness…”
Neurophysiology

• Pain inhibition
  – Pre and Post synaptic opioid receptors
    • Highly concentrated in 2\textsuperscript{nd} and 3\textsuperscript{rd} layers of the substantia gelatinosa, cerebral cortex, hypothalamus, medial thalamus, amygdala, extrapyramidal regions, sympathetic preganglionic neurons.
  – Opioids can be exogenous (morphine, others) or endogenous (beta endorphins, enkephalins).
Neurophysiology

• Pain inhibition
  – Serotonin,
    • Can increase dorsal horn thresholds
  – Descending inhibition via spinal cord
    • Modified by drugs
    • Modified by hypnosis
    • Modified by operant conditioning
    • Brain-Derived Neurotropic Factor (BDNF)
      – After nerve injury, affects microglial cells of cord by causing release of BDNF thus disrupting inhibition within the cord (Neuropathic pain)
      – University of Toronto 12/19/2005
Nociception: Etiology of Facilitation

The Theory of the Development of Somatic Dysfunction
Nociception Theory

• **Habituation**
  – The process of decreasing response of a neural pathway with continuous stimulation
  – Ubiquitous phenomenon

• **Sensitization**
  – The opposite idea of habituation

• These two processes exist together to help maintain a homeostasis between over-reaction and under-reaction to a stimulus
Nociception

- Current theory as to cause for the facilitation involves alteration of nociceptive stimuli
- Once a stimulus is strong enough to activate (depolarize) nociceptive pathways, impulses travel to the cord and then branch to multiple sites
- Results in release of peptides at the motoneuron level in the peripheral tissues
Nociception Theory

• The peptides are of the inflammatory cascade and initiate the release of prostaglandins, bradykinins, etc.
  – Chemical soup that spreads in the tissues

• This results in lowering nociceptor thresholds thus increasing input to the cord
Nociception Theory

• Inflammation disrupts the balance between habituation and sensitization
  – Results in larger than normal motor outputs to the autonomies and somatic systems
  – This then is thought to set up the low-threshold spinal reflexes Korr and Denslow talked about: **THE FACILITATED SEGMENT**
Allostasis

Immunoneural/Hormonal Response to Segmental Facilitation
Allostasis

- Begin with a stimulus (insult) applied to tissues
- Develop chemical soup of inflammation
- Causes Primary afferent sensitization
- Results in hyperalgesia
  - The exaggerated response to a noxious stimulus
- Allodynia – pain perceived from stimulus not normally known to cause pain
  - Descending inhibitory influences are decreased
Allostasis

• Secondary hyperalgesia develops
  – Central Sensitization (CNS)

• Dorsal Horn neurons
  – Lose inhibitory neuron function
  – All aid in maintaining facilitation
Allostasis

• **Ventral Horn Motor Effects**
  – Facilitation refers information to the CNS Brainstem, Hypothalamus, and Cortex
  – Facilitation outflows to autonomics
    • Affecting visceral function
  – Facilitation outflows to soma
Allostasis

• **Brainstem**
  – Facilitation decreases endogenous descending pathways
  – Arousal system (Neuroendocrine/Immune Axis)
    • Catecholamines/glucocorticoids
    • Long term facilitation damages this system
    • Leads to loss of control of protective mechanisms
      – *Increased Allostatic Load*
ALLOSTASIS

- PHYSICAL STRESS
  - Somatic
  - Visceral

- PSYCHOSOCIAL STRESS
  - Emotional

- NOREPI
- AROUSAL SYS.: NEUROENDOCRINE/ IMMUNE AXIS
- CORTISOL

- ALL SYSTEMS OF BODY INTEGRATED
Physiologic Effects

• Pain affecting the CNS
  – Segmental spinal reflexes
    • Muscle spasm, vasoconstriction
  – Suprasegmental reflex responses
    • Noxious input at several levels causes release of catecholamines, steroids, renin-angiotensin, others
  – Fatigue, sedation
  – Cortical response
    • What the patient thinks and believes about the pain.
Physiologic Effects

• Physical Effects
  – Cardiovascular
    • Tachycardia, HTN, inotropic effects
  – Pulmonary
    • Hypoxia, hypercarbia, atelectasis, decreased vital capacity, others
  – GI
    • Nausea, vomiting, ileus, inhibited intake
Physiologic Effects

- Renal
  - Oliguria, urinary retention
- Endocrine
  - Excessive adrenergic activity, vagal inhibition, increased O2 demands, hyperglycemia
Physiologic Effects

- **Vascular**
  - Thromboembolism
- **CNS**
  - Fatigue, sedation
- **Immunologic**
  - Inhibition of cellular immunity, impaired wound healing, risk of infection
Psychophysiologic Effects

- General arousal (sympathetic response)
- Sleep disturbance
- Reduced activity
- Impaired behavioral responses
  - Concentration
  - Problem solving
  - Short term memory
  - Decision making capacity
Psychophysiologic Effects

• Limbic system connected via spinoreticular tract connections
• Emotional lability plays a role in the perpetuation of the pain and the chronicity of the somatic dysfunction
• Depression causes a decrease of the function of the descending inhibitory tracts
  – Pain and TART findings may change with mental state
Psychophysiological Effects

• Negative emotional reactions
  – Anger
  – Fear
  – Depression
  – Frustration

• Negative family and social responses
  – Helplessness or overprotectiveness
Psychophysiologic Effects

• “Vicious Pain Cycle”
  – Inactivity, fatigue lead to muscle weakness (1% per day after first 10 days)
  – Leads to increased pain
  – Leads to increased inactivity, fatigue, loss of muscle tone
  – Leads to further inactivity…
Physiologic Effects

• Musculoskeletal
  – Spasm
  – Connections to postural control center
    • Maintenance of myofascial strain pattern in chronic pain patients
  – Limited mobility
Muscular Imbalance
Muscle Fiber Types

• Slow-twitch muscle
  – Oxidative metabolism, high capillary density
  – Slow twitch speed
  – Function of these muscles more **tonic** or postural
  – React to functional disturbance by shortening and tightening
Muscular Imbalance

Muscle Fiber Types

• Fast-twitch muscle
  – Glycolytic metabolism
  – Fatigue rapidly
  – Low capillary density
  – **Phasic** in function and react to disturbance by weakening

• All muscles have a mixture of both
  – Some more one than the other
Muscular Imbalance
Muscle Fiber Types

• Postural/tonic function
  – Pelvis hip region
    • Hamstrings
    • Iliopsoas
    • Rectus femoris
    • Tensor fascia lata
    • Hip adductors
    • Piriformis
  – Trunk
    • Erector spinae group (lumbar & cervical)
    • Quadratus lumborum
    • Scalenes
Muscular Imbalance

Muscle Fiber Types

• Postural/tonic function
  – Shoulder girdle
    • Pectoralis major
    • Levator scapulae
    • Upper trapezius
    • Biceps brachii
  – Lower Extremity
    • Gastrocnemius
    • Soleus
Muscular Imbalance
Muscle Fiber Types

• Phasic
  – Pelvic hip region
    • Vastus medialis and lateralis
    • Gluteus medius, maximus and minimus
  – Trunk
    • Erector spinae muscles (mid-thoracic)
  – Shoulder girdle
    • Rhomboids
    • Lower trapezius
    • Triceps brachii
  – Lower extremity
    • Tibialis anterior
    • Fibularis (perineal longus & brevis)
Concept of Muscular Imbalance

• Definition
  – Shortening and tightening of tonic muscle groups with weakness in phasic muscles with associate loss of control on integrated muscle function

• Constant adaptation of muscle balance to gravity
  – Faulty posture changes center of gravity
  – Results in mechanoreceptor responses requiring muscle responses
  – Change in mechanical behavior of joints
  – Over time may produce a change in cord memory and maladaptive pattern of tension and weakness begins
  – Result is changes in available motion within patterns of motions
  – This is a multifactorial problem and highly complex but simply stated you have muscles tightening, other weakening, and asymmetric control from a neurological stand point maintaining the maladaptive somatic dysfunction
The Key Dysfunction

• Biotensegrity (Levin MD)
  – Icosahedral design applied to the body
  – Tension trusses = myofascial tissues
  – Compression trusses = bones
  – Body is a layer on top of layer of tensegrity systems
  – The presenting posture is the balancing of these forces
    • Normal or abnormal
The Key Dysfunction

• Biotensegrity (Levin MD)
  – The motor response to the increased allostatic load drives the tension within the system
  – As disease processes and injury patterns occur in the body a change in the distribution of force would be expected thus changing tension within the MS system in order to establish a new and/or stable (although abnormal) posture.
    • Strain pattern
  – Change in force at one site will be compensated by instantaneous change throughout whole system
The Key Dysfunction

• The strain pattern will result in restriction within motion patterns
• Some patterns may have increased mobility, non-uniformly, to compensate for loss of motion in other areas of the body
• The increase mobility often is the site of overuse and thus the site of symptomatic expression
• The tighter patterns are the more significant ones to concentrate on
The Key Dysfunction

- McConnel
- Lack of external rotation and extension of the hip
  - Compensated by increased lumbar spine rotation (gait cycle)
- Annulus fibrosis layers have nociceptors
- Over time overuse injury increasing back pain
The Key Dysfunction

• With preexisting disease this can cause exacerbation of the patient’s pain
• Root problem not in the lumbar but in hip motion
• Treatment needs to be applied to the area of greatest restriction
The Key Dysfunction

• Treatment efficiency may be increased by treating the key dysfunction first
• Treatment that fails to address the key dysfunction will often fail to alleviate the patient’s symptoms for much time
Comprehensive Evaluation

• Each motion graded:
  – Starting Position
  – -4
  – -3
  – -2
  – -1
  – Reference Range
  – +1 or above as needed
Motion Grading
LINKAGE
Comprehensive Evaluation

• Evaluate
  – Standing, seated, supine, and prone

• Seated
  – SUE flexion, extension, abduction, adduction
  – BUE internal and external rotation
  – Scapular gaping
Comprehensive Evaluation

• Supine
  – SLE flexion, abduction, adduction
  – BLE flexion, internal and external rotation, adduction, abduction
  – BUE external and internal rotation
Comprehensive Evaluation

• Prone
  – BLE flexion at knee, internal and external rotation, extension at hip
  – SUE horizontal extension(abduction)
System Mobility - Normal

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## System Mobility – Acute

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## System Mobility - Chronic

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## System Mobility - Loose

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The chart illustrates the distribution of mobility across different categories, with a significant increase in the FROM category.
Treatment goals

- 100% cure seldom achieved
- Restore mobility to maximize patient’s function
- Don’t promise to relieve pain
- Education of the patient is the primary goal
- Peer group support very valuable
Treatment Steps

• First - pain relief
• Second – restoration of normal muscle length (ROM) and flexibility exercises
  – OMM applied here
• Third – gradual muscle strengthening and recovery of aerobic capacity
Chronic Low Back Pain

OMM Suggestions

• Functional Pathology vs Structural Pathology
  – Guide you as to what modalities applied

• Address multifidus, abdominal muscles, and gluteus medius muscles
  – Endurance exercises
  – Aid in muscle recruitment order in desired motion pattern
Chronic Low Back Pain
OMM Suggestions

• Let your grading evaluation guide you.
  – Break down the tightest patterns of motion for more specific myofascial and arthroidal dysfunctions
    • Look for the greatest restrictions within these areas as well
  – Apply treatment to these areas first then work on compensatory regions
Chronic Low Back Pain
OMM Suggestions

• Consider upper extremity dysfunction in low back pain
  – Latissimus dorsi fascial tissue is contiguous through the gluteus medius, into the posterior sacroiliac capsule, into the sacrotuberous ligament and into the origin of the posterior thigh muscles off of the ischial tuberosity.
Chronic Low Back Pain

OMM Suggestions

• Evaluate the patient’s gait
  – Dysfunction within the knee (hyperextension) or subtalar joint
    • Compensated by increased lumbopelvic rotation in transverse plane
    • Increased strain in lumbar spine
  – Dysfunction within the foot during swing phase
    • Shock absorption loss
    • Forces transferred to extremity and pelvis
  – Dysfunction within the foot during stance phase
    • Subtalar motion important
Patient Education

• Direct instruction, repetition and reinforcement
• Encourage note taking, homework
• Pts clarify instructions, repeat back
• Patient set short term goals
• Patients maintain checklists (exercises, etc)
• Written contract – duration, frequency, quality of behaviors required
• Provide written materials
• INCLUDE FAMILY / CAREGIVERS
Drugs

- Pain control – NSAIDS
- Opioids – chronic pain
  - New Guidelines March 2016 on CDC Website
    - http://www.cdc.gov/drugoverdose/prescribing/guideline.html
  - Non–cancer related pain
  - Often combined with adjuvant drugs
Drugs

- **Tricyclic antidepressants**
  - Amitriptyline (Elavil)
  - Analgesic response 40-77%
  - Others
    - Desiprimine
    - Nortriptyline
Drugs

• Anticonvulsants
  – Carbamazapine (Tegretol)
    • Stabilizes neural membranes
      – Peripheral and central
    • Neuropathic pain
    • Mononeuropathies
Drugs

• Anticonvulsants
  – Gabapentin (Neurontin)
    • Stabilizes neural membranes
      – Peripheral and central
    • Neuropathic pain
    • Mononeuropathies
Drugs

• Muscle Relaxants
  – Cyclobenzaprine (Flexeril)
  – Metaxalone (Skelexin)
  – Tizandine (Zanaflex)
  • Efficacy in neuropathic pain
  • Fibromyalgia effective
Behavioral training

- Support groups
- Coping skills training
  - Positive affirmations “self talk”
  - Relaxation training
  - Systematic desensitization
  - Biofeedback
Invasive procedures

• Specialist Involvement
  – Injections – joints, soft tissue
  – Nerve blocks
  – Surgical decompression
  – Surgical ablation
    • Peripheral nerves, sympathectomy
    • Spinal cord tracts
    • Thalamus, other areas in the brain
  – Radiation therapy.
Conclusion

• Chronic pain patient has learned maladaptive ways of functioning, thinking, feeling, and behaving.

• Goal is to replace those ways with functional and productive ways of thinking, feeling and behaving. Will also lessen depression, restore self esteem, and often reduce dependency.
Conclusion

• Osteopathic manipulative treatments
  – Provide a physical, tangible change in the tissue function
  – Can reduce the facilitation
  – Can enhance function
  – Can provide lasting changes
Conclusion

• In general move the patient from
  – “victim of injury” to
  – “manager of recovery”
  – Move through stages of grief / grieving:
    • Shock – denial – anger – bargaining – mourning - acceptance
  – Improve functional capacity of the musculoskeletal system
Resources


Resources
Resources


Three pain scales

Simple Descriptive Pain Intensity Scale

No pain  Mild pain  Moderate pain  Severe pain  Very severe pain  Worst possible pain

0–10 Numeric Pain Intensity Scale

No pain  1  2  3  4  5  6  7  8  9  10  Worst possible pain

Visual Analog Scale (VAS)

No pain   Pain as bad as it could possibly be
Mark the areas on your body where you now feel your pain. Include all affected areas. Use the appropriate symbols indicated below:

- ACHE
- NUMBNESS
- PINES & NEEDLES
- BURNING
- STABLING

— Symbols for pain intensity:
- Left: LEFT
- Right: RIGHT
Mark the areas on your body where you now feel your pain. Include all affected areas. Use the appropriate symbols indicated below:

- ACHE: >>>
- NUMBNESS: ===
- PINS & NEEDLES: oooo
- BURNING: xxx
- STABBING: ////

![Body diagram with marked pain areas on the right shoulder]
Mark the areas on your body where you now feel your pain. Include all affected areas. Use the appropriate symbols indicated below:

- ACHE: 
- NUMBNES: 
- PINS & NEEDLES: 
- BURNING: 
- STABBING:

[Diagram showing areas marked with crosses on various parts of the body, indicating pain areas]