AOMA 2020
Chronic Pain

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• Disclosures: None
Chronic Pain - Objectives

• Elucidate the epidemiology of chronic pain and its economic impact.
• Differentiate the difference between acute and chronic pain in:
  • Clinical Presentation
  • Autonomic Nervous System Activity
  • Neurologic Changes
  • Approach to Treatment
• Compare and contrast subjective and objective findings in acute and chronic pain.
• Integrate the physiology and pathology of nociception and chronic pain neural tracts.
• Discriminate between the various clinical pain syndromes reviewed.
• Describe influences on perception of pain, such as pain amplification.
• Describe neurologic changes involved in chronic pain, including those that interface with reward/addiction pathways.

Chronic Pain - Epidemiology

• 2016 – 20% of US adults had chronic pain (@50 million people) with an additional 8% (@20 million) with “high-impact” chronic pain (ie disability.)
Chronic Pain - CDC Pain Statistics

• 2010
  • One-fifth of adults 65 years and older said they had experienced pain in the past month that persisted for more than 24 hours.
  • Almost three-fifths of adults 65 and older with pain said it had lasted for 1 year or more.
  • More than one-quarter of adults interviewed said they had experienced low back pain in the past 3 months.

• 2016
  • An estimated 20.4% of U.S. adults (50.0 million) had chronic pain and 8.0% of U.S. adults (19.6 million) had high-impact chronic pain, with higher prevalence associated with advancing age.

https://www.cdc.gov/mmwr/volumes/67/wr/pdfs/mm6736a2-H.pdf

Chronic Pain: Historical/Social Perspective

• Descartes (1596-1650) described pain in three stages:
  • Onset of tissue damage;
  • Movement of a signal up a transmission line;
  • Conscious experience and behavioral response.

• Pain means different things to different people:
Chronic Pain - Types of Pain

• According to the International Association for the Study of Pain (IASP), pain can be classified, based on:
  1. the region of the body involved (e.g., head, visceral),
  2. pattern of occurrence’s duration (acute and chronic), or
  3. the system of which dysfunction that may cause the pain (e.g., gastrointestinal, nervous).

• Pain is also classified based on only three characteristics: symptoms, mechanisms and syndromes.

• Thus, internationally pain has been classified into three major classes—
  • nociceptive pain,
  • neuropathic pain and
  • inflammatory pain

Chronic Pain - Nociception

• The transmission and perception of pain is termed “nociception”
• Pain is produced by unpleasant noxious stimuli: heat, cold, pressure, and mechanical injury.
• Receptors throughout the body that respond to pain are nociceptors.
Chronic Pain – Areas of High Nociceptor Concentration

- Subcutaneous tissue
- Periosteum
- Fascia
- Ligaments
- Joint capsules
- Cornea of the eye

Chronic Pain – Areas of Low Nociceptor Concentration

- Bone
- Skeletal muscle
- Cartilage
Chronic Pain - Afferent Neurons

- A fibers: Largest and most rapid conducting.
- B fibers: Intermediate size and speed
- C fibers: Small and slow conducting
- A-delta subtype: Respond to strong stimuli very rapid and localized. A sharp pain such as a finger stick.

Chronic Pain – Nociception – Aδ- and C-fibers

- Aδ- and C-fibers are two primary afferent nociceptors types responding to noxious stimuli and have specialized free nerve endings that are widely located in the skin, muscle, joint capsule, bone and some major internal organs.
- Nociceptors are not stimulated directly by noxious stimuli
- Chemical mediators are released when injury occurs which activate nociceptors – will review in a moment
Chronic Pain –
Nociception – A Fibers

• Group A nerve fibers were classified by Erlanger and Gasser as fibers that are myelinated. It can be further subdivided into Aα, Aβ, Ay and Aδ with different sets of characteristics each.
• These fibers generally terminate in laminae I, III, IV and V of the DH of the spinal cord with some lamina II inner projection.
  • Type Aα: both Type Ia and Ib of the sensory fibers from muscle spindle endings and Golgi tendon are grouped into this type. It is mainly used to determine the proprioceptive function.
  • Type Aβ: it is a low-threshold, cutaneous, slow or fast adapting type of mechanoreceptors, and is a Type II afferent fiber from the stretch receptor. The Aβ-fibers belong to laminae III and IV.
  • Type Ay: Type II afferent fibers from the stretch receptors.
• Type Aδ: the thermal and mechanical nociceptors that terminate in the rexed laminae I and V; is a Type III afferent fiber. Aδ-fibers are also the smallest myelinated nerves and have a relatively fast conduction velocity of 30 m/s. The diameter of Aδ-fibers is about 2–5 μm, and is responsive towards short-lasting and pricking pain.


Chronic Pain –
Nociception: C Fibers

• Mainly nociceptive in function, carrying the sensory information and assembling around 70% of the afferent nociceptive information, which then enters the spinal cord.
• Unmyelinated with less than 2 μm in diameter.
• Relatively slow conduction velocity of approximately 2 μm/s.
• Nerve fibers at the dorsal roots (Type IV afferent fibers) and postganglionic fibers in the ANS can be categorized in this group.
• Terminate in laminae I and II in the grey matter of the spinal cord. In terms of nociception, C-fibers nociceptors are
• Polymodal: activated by thermal, mechanical and chemical stimuli.
• Poorly localized, such as burning sensation of the skin.
• In terms of neurochemistry, C-fibers can be classified as either peptidergic or non-peptidergic, and about 50% of these fibers express neuropeptides inclusive of calcitonin gene-related peptide (CGRP), neurokinins and substance P (SP).
Chronic Pain – Nociception: What about B fibers?

• B Fibers: nerve fibers are moderately myelinated with conduction velocities of 3–14 m/s.

• Preganglionic nerve fibers of the autonomous nervous system (ANS) and general visceral afferent fibers belong to this group.

Chronic Pain - Nociception: Pain Pathway

• Pain mechanism undergoes three events—
  1. transduction,
  2. transmission and
  3. modulation when there is a presence of noxious stimuli.

• Transduction occurs along the nociceptive pathway:
  1. (1) stimulus events are converted to chemical tissue events
  2. (2) chemical tissue and synaptic cleft events are then changed into electrical events in the neurons; and
  3. (3) electrical events in the neurons are transduced as chemical events at the synapses.

• Transmission of the electrical events along the neuronal pathways, while neurotransmitters in the synaptic cleft transmit information from a post-synaptic terminal of one cell to a pre-synaptic terminal of another.

• Modulation takes place at all level of nociceptive pathways through the primary afferent neuron, dorsal horn and higher brain center by up- or down-regulation.
Chronic Pain - Pain Pathway

- Nociceptor activation is transduced along the axons of peripheral nerves which terminate in the dorsal horn of the spine. (upper image)
- Then travel via spinal cord and through the spinothalamic tract to output on the thalamus. (lower image)
- Thalamus is a “relay station” for sensory information to the cerebral cortex.
- Nociceptive pathways terminate in discrete subdivisions of thalamic nuclei known as the ventral posterior lateral nucleus and the ventromedial nucleus.


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Chronic Pain - Pain Pathways

- From these nuclei, nociceptive information is relayed to various cortical and subcortical regions, including the amygdala, hypothalamus, periaqueductal grey, basal ganglia, and regions of cerebral cortex.
- Most notably, the insula and anterior cingulate cortex are consistently activated when nociceptors are stimulated by noxious stimuli, and activation in these brain regions is associated with the subjective experience of pain.
- In turn, these integrated thalamocortical and corticolimbic structures, which collectively have been termed the pain “neuromatrix,” process somatosensory input and output neural impulses which influence nociception and pain perception.

Chronic Pain – Nociceptive Neurotransmitters

- **Inflammatory mediators:** PGE2, PGI2, LTB4, NGF, proton, BK, ATP, adenosine, SP, NKA, NKB, 5-HT, histamine, glutamate, NE and NO

- **Non-inflammatory mediators:** CGRP, GABA, opioid peptides, glycine and cannabinoids


Chronic Pain - Neuropathic Pain

- **Due to nerve injury or nerve impairment and is often associated with allodynia:** central pain sensitization that is a result of repetitive non-painful stimulation of the receptors. Pain response arises from a stimulus that is normally non-painful, due to sensitization process from repetitive stimulation.

- **Often secondary to inflammation or metabolic diseases, such as diabetes, trauma, toxins, tumors, primary neurological diseases and herpes zoster infection.**

  *Ramsay Hunt syndrome is peripheral facial nerve palsy accompanied by an erythematous vesicular rash on the ear (zoster oticus) or in the mouth.*
Chronic Pain - Neuropathic Pain

• What’s This?

Chronic Pain - Neuropathic Pain

• Herpes Zoster Ophthalmicus
  • Prodrome of HZO consists of fever, malaise, and chills.
  • Initial symptom is hyperesthesia or paresthesia, which may be severe, along the affected dermatome.
  • Followed by a maculopapular rash that becomes vesicular and finally pustular before crusting
  • Tx: Oral acyclovir within 72 hours of onset; Gabapentin/Pregabalin; steroids?

https://www.aao.org/focalpointsnippetdetail.aspx?id=4387b620-2451-4e4f-b9a7-6c51b8351a03
Chronic Pain - Inflammatory Pain

• Inflammation may lead to three major responses:
  • Hyperalgesia: tenderness or lowered threshold to the thermal or mechanical stimulation-induced pain (primary hyperalgesia)
  • Allodynia: central sensitization that leads to the triggering of pain response that normally does not provoke pain, such as a light touch
  • Sympathetic maintained pain

Chronic Pain – Acute (Not Chronic) Pain

• Pain that is associated with a well-defined cause with rapid onset.
• This type of pain follows an injury and often resolves with the healing process.
• Treatment often consists of rest, OMT, pain medication, and allowing the body to heal where injury has occurred.
Chronic Pain –
Objective Signs of Acute Pain

• Sympathetic Nervous System Activation:
  • Increased heart rate
  • Increased systolic and diastolic blood pressure
  • Pupillary dilation
  • Increased muscle tension
  • Increased bronchiole diameter
  • Conversion of Glycogen to glucose
  • Increased release of adrenaline: epinephrine, norepinephrine, dopamine.
  • Cortisol secretion.

Chronic Pain –
Physical Effects of Acute Pain

• The presence of acute pain can be determined by evaluating several physiologic effects of pain, most notably how pain activates the sympathetic nervous system. As widely understood,
  • Pain stimulates the sympathetic nervous system, which in turn increases heart rate (HR) and causes peripheral vasoconstriction... Theoretically, pain could be detected within an organism based on the effect of activating the sympathetic nervous system in response to pain. By measuring that effect, the pain level may be deduced. For instance,... [p]hotoplethysmography (pulse oximetry) can be used to assess vasomotor tone (vasoconstriction) and HR and, thus, could potentially be used as a surrogate to assess perioperative pain.

K. Hamunen, V. Kontinen, E. Hakala, P. Talke, M. Paloheimo, E. Kalso; Effect of pain on autonomic nervous system indices derived from photoplethysmography in healthy volunteers, BJA: British Journal of Anaesthesia, Volume 108, Issue 5, 1 May 2012, Pages 838–844,
Chronic Pain – Pain... and all that comes with it

• That “pain”, ie nociception, is only one element of chronic pain has a long history in human thought.
• The science to support this is developing....

Chronic Pain – Not So Simple

• Common Symptoms expressed by patients with chronic pain:
  • A dull ache
  • Throbbing
  • Burning
  • Shooting
  • Squeezing
  • Stinging
  • Soreness
  • Stiffness

• Sometimes pain is just one of many symptoms, which can also include:
  • Feeling very tired or wiped out
  • Not feeling hungry
  • Trouble sleeping
  • Mood changes – depressed, stressed
  • Weakness
  • A lack of energy

https://www.webmd.com/pain-management/guide/understanding-pain-management-chronic-pain#1
Chronic Pain - Influencers

• What Can Influence Pain?
  • Awareness level (Johnson and Rice 2007)
  • Anxiety and Fear of the Unknown (Mitchell and Loustau 2008)
  • Patient’s Age (Walker, et al 2003, Sorenson and Luckman 2011)
  • Education (Sorenson and Luckman 2011)
  • Depression (The presence and influence of mild depressive symptoms on post-operative pain perception following a)
  • Social Support (or lack thereof) (Che X, et al 2018)
  • State of Functioning
  • Sense of Control in Life
  • Emotional Stress
  • Diet
  • Genetics
  • Chronicity

Chronic Pain - Influencers - Diet

• Diet

  • “Anti-inflammatory diets” are intended to reduce food-based sources of inflammation and thus reduce pain.
  • Diets rich in vegetables, fruits, legumes, seeds and nuts shown to reduce morbidity and increase lifespan.

Chronic Pain - Influencers – Cognitive Processes

• What else can affect Perception of pain?
• Cognitive Processes
  • “...perception is critically determined by expectations and their modification through learning. Research on pain has just begun to embrace this view. Insights into these processes promise to open up new avenues to pain prevention and treatment by harnessing the power of the mind.”


Chronic Pain - Influencers – Emotional Activity

• Emotional Activity
  • Activation of the dorsolateral prefrontal cortex (DLPFC) area results in diminished pain.
  • Activation of the DLPFC also correlated with:
    • Positive emotional states

• ACC: Anterior Cingulate Cortex
• ANS: Autonomic Nervous System
• MBS: Mind-Body Syndrome

Rakel D. Integrative medicine, 4th Ed. 2018. Chapter 102
Chronic Pain - Influencers - Genetics

• **Genetics**

  • Currently there are 100 million people in the US with chronic pain.


  • Several studies indicate while genetic predisposition exists for a wide number of influencing factors on pain, “life events” are often required to trigger their expression (ie depression, migraines, etc. might have a environmental genetic trigger – ie epigenetics)

  • A study by Taylor indicated that:
    • A specific genotype for depression can be turned on by a stressful childhood and turned off by nurturing and emotional support.

    Taylor S.E.: Mechanisms linking early life stress to adult health outcomes. Proc Natl Acad Sci USA 2010; 107: pp. 8507-8512

Chronic Pain - Influencers - Genes

• **Genetics**

  • Individuals were identified with an inherited pain insensitivity and rapid healing – patients who did not need anesthesia, pain medications and also experienced faster than normal healing.

Habib AM, et al. Microdeletion in a FAAH pseudogene identified in a patient with high anandamide concentrations and pain insensitivity. British journal anaesthesia. 28 March 2019
Chronic Pain – Pain and Emotions

• In a 2013 study published in Brain magazine:

• Using fMRI, people with acute back pain were followed and distinct changes in neural function/structure were identified if the pain became chronic.

“We observed that brain activity for back pain in the early, acute/subacute back pain group is limited to regions involved in acute pain, whereas in the chronic back pain group, activity is confined to emotion-related circuitry... [my emphasis]


Chronic Pain – Pain and Emotions

• In a 2013 study published in Brain magazine....

The results demonstrate that brain representation for a constant percept, back pain, can undergo large-scale shifts in brain activity with the transition to chronic pain. These observations challenge long-standing theoretical concepts regarding brain and mind relationships, as well as provide important novel insights regarding definitions and mechanisms of chronic pain.”

Chronic Pain – Autonomic Changes

• The chronicity of pain appears to complicate a direct correlation in objective measures between sympathetic response and pain.

• As noted in a recent study,
  ....chronic stress responses can be expressed by decreased... sympathetic hyporeactivity to a stressful stimulus.”


Not Caring About Pain?
Textbook sympathetic dysregulation

Chronic Pain – Blunted Sympathetic Response

• In other words, the sympathetic response to chronic pain may be different than acute pain, perhaps even typified by a blunted response.

• This apparent effect of chronic pain upon the autonomic system has been noted:
  • ...that the sympathetic nervous system is affected in CLBP [chronic low back pain] and FBSS [failed back surgery syndrome] patients with abnormalities in SSR [sympathetic skin response] and that the dysfunction of sympathetic nervous system may contribute to the intensity and chronicity of pain in these groups of patients.

Chronic Pain - Cycles

• Generally Speaking, Acute Pain in a Healthy Person Triggers a Cycle of Inflammatory, Neural, Genetic and Other Changes – becomes ongoing if the pain becomes chronic.
• These Neural Changes tend to involve other non-nociceptive areas in pain signal as it travels through the CNS which can influence (and change) the nociceptive signal’s affect.
• Therefore comprehensive treatment of Pain Expands Beyond Treating the Nociceptive Trigger – especially chronic pain
• Implications on Opiate Crisis? Will get to in a minute.

Chronic Pain - Cycles - Fear Avoidance Model

• Reaction to initial injury, or “Pain Experience” determines either confrontation and recovery from the event, or the cycle on the left:
  • Fear ➔
  • Hypervigilance ➔
  • Depression ➔
  • Return to Pain Experience

The FAM of pain. From: Vlaeyen and Linton
Chronic Pain – So Many Variables

• Why Is Pain Influenced By So Many Variables?
• How Many Variables Should a Physician Treat?
  • i.e. What Should Be The Standard of Care For Physicians?
  • Neurology v Psychiatry?
    • Ie Should treatment of chronic pain involve emotion-based treatments? (yes)
    • Screening for who with acute pain will likely transition to chronic pain should be standard – ie how many factors influencing pain are active in the patient’s life
    • Part of managing acute pain should be the awareness that if it becomes chronic the patient’s brain CHANGES.
    • Maybe managing acute pain needs to involve treating all pain influencers...?

Chronic Pain - Clinical Case

• A 40 year old female with a past history of irritable bowel and depression presents with a chief compliant of diffuse muscle aches and stiffness for 4 months duration. The pain is 5/10 and interferes with activity of daily living.
• Pt admits to constant fatigue, waking up unrefreshed, headache, and occasional hand and feet swelling.
Chronic Pain - Clinical Case

- Physical exam reveals multiple areas on the body very tender to palpation. There is no joint swelling or deformity noted.
- Lab work including CBC, CMP, ESR, Rheumatoid factor, ANA, TSH, are normal.
- Diagnosis of exclusion: Fibromyalgia Syndrome

Chronic Pain - Fibromyalgia

- Most common rheumatologic syndrome in ambulatory medicine, 3-10% population.
- Over 75% are women between 20-50 years of age.
- Characterized by diffuse aches, stiffness, and fatigue.
- Considered a diagnosis of exclusion.
- Rule out thyroid disease, lupus, rheumatoid arthritis, malignancy, infectious disease, etc.
- Pain and fatigue are worse with stress, cold, and physical activity.

Fibromyalgia Mimics

- Lupus
- Multiple sclerosis
- Rheumatoid arthritis
- Polymyalgia rheumatica
- Axial spondyloarthritis
- Thyroid disease
- Type 2 diabetes
- Anemia
- Chronic fatigue syndrome

Large Differential: Rule Them Out!
Chronic Pain – 1990 ACR Fibromyalgia Diagnostic Criterion

- Commonly referred to for past studies.
- Will often see reference to this as a result.
- 11/18 tenderpoints present for greater than 3 months duration.
- Replaced by 2010 criterion since it did not account for fatigue and cognitive dysfunction.
Chronic Pain –
2010 ACR Fibromyalgia Diagnostic Criterion

A patient satisfies diagnostic criteria for fibromyalgia if the following 3 conditions are met:

• Widespread pain index (WPI) ≥7 and symptom severity (SS) scale score ≥5 or WPI 3 - 6 and SS scale score ≥9.
• Symptoms have been present at a similar level for at least 3 months.
• The patient does not have a disorder that would otherwise explain the pain.

Chronic Pain –
Fibromyalgia - Widespread Pain Index

• WPI: note the number areas in which the patient has had pain over the last week. In how many areas has the patient had pain? Score will be between 0 and 19.

Shoulder girdle, left
Shoulder girdle, right
Upper arm, left
Upper arm, right
Lower arm, left
Lower arm, right
Hip (buttock, trochanter), left
Hip (buttock, trochanter), right
Upper leg, left
Upper leg, right
Lower leg, left
Lower leg, right
Jaw, left
Jaw, right
Chest
Abdomen
Upper back
Lower back
Neck
Chronic Pain -
2016 Update to 2010 ACR Fibromyalgia Diagnostic Criterion

• Fibromyalgia may now be diagnosed in adults when all of the following criteria are met:
  1. Widespread pain index (WPI) ≥7 and symptom severity scale (SSS) score ≥5 OR WPI 4–6 and SSS score ≥9.
  2. Generalized pain, defined as pain in at least 4 of 5 regions, is present.
  3. Symptoms have been present at a similar level for at least 3 months.
  4. A diagnosis of fibromyalgia is valid irrespective of other diagnoses. A diagnosis of fibromyalgia does not exclude the presence of other clinically important illnesses.

ABSTRACT NUMBER: 997, 2016 Revisions to the 2010/2011 Fibromyalgia Diagnostic Criteria;

Chronic Pain –
2016 Update to 2010 ACR Fibromyalgia Diagnostic Criterion cont.

• The revision makes the following changes:
  1. Changes criterion 1 to “Widespread pain index (WPI) ≥7 and Symptom Severity Scale (SSS) score ≥5 OR WPI 4–6 and SSS score ≥9.” (WPI minimum must be ≥4 instead of previous ≥3)
  2. Adds a generalized pain criterion (Criterion 2) that is defined as pain in at least 4 of 5 regions (left upper, right upper, left lower, right lower, axial). In this definition, jaw, chest and abdominal pain are not evaluated as part of the generalized pain definition.
  3. Standardizes and makes 2010 and 2011 criterion (criterion 3) wording the same: “Symptoms have been generally present for at least 3 months.”
  4. Removes the exclusion regarding disorders that could (sufficiently) explain the pain (criterion 4) and adds the following text: “A diagnosis of fibromyalgia is valid irrespective of other diagnoses. A diagnosis of fibromyalgia does not exclude the presence of other clinically important illnesses.”
  5. Adds the Fibromyalgia Symptom (FS) [or polysymptomatic distress (PSD)] scale as a full component of the fibromyalgia criteria.

• Creates one set of criteria (2016) instead of having separate physician (2010) and patient (2011) criteria by replacing the physician estimate of somatic symptom burden with ascertainment of the presence of headaches, pain or cramps in lower abdomen, and depression during the previous 6 months.

Chronic Pain – Fibromyalgia: Symptom Severity Scale Score

<table>
<thead>
<tr>
<th>Symptoms Evaluated</th>
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<tbody>
<tr>
<td>Fatigue</td>
</tr>
<tr>
<td>Waking unrefreshed</td>
</tr>
<tr>
<td>Cognitive symptoms</td>
</tr>
</tbody>
</table>

For the each of the 3 symptoms above, indicate the level of severity over the past week using the following scale:

- 0 = no problem
- 1 = slight or mild problems, generally mild or intermittent
- 2 = moderate, considerable problems, often present and/or at a moderate level
- 3 = severe: pervasive, continuous, life-disturbing problems

Considering somatic symptoms in general, indicate whether the patient has:

- 0 = no symptoms
- 1 = few symptoms
- 2 = a moderate number of symptoms
- 3 = a great deal of symptoms

- The SS scale score is the sum of the severity of the 3 symptoms (fatigue, waking unrefreshed, cognitive symptoms) plus the extent (severity) of somatic symptoms in general. The final score is between 0 and 12.
- Somatic symptoms that might be considered:
  - muscle pain, irritable bowel syndrome, fatigue/tiredness, thinking or remembering problem, muscle weakness, headache, pain/cramps in the abdomen, numbness/tingling, dizziness, insomnia, depression, constipation, pain in the upper abdomen, nausea, nervousness, chest pain, blurred vision, fever, diarrhea, dry mouth, itching, wheezing, Raynaud's phenomenon, hives/welts, ringing in ears, vomiting, heartburn, oral ulcers, loss of/change in taste, seizures, dry eyes, shortness of breath, loss of appetite, rash, sun sensitivity, hearing difficulties, easy bruising, hair loss, frequent urination, painful urination, and bladder spasms.

Chronic Pain - Fibromyalgia

- Cause of fibromyalgia: Multifactorial?
- Sleep Disturbance: Etiology or Symptom?
  - Greater then 70% of patients have alpha wave intrusion into non-REM delta wave sleep.
- Considered Central Sensitivity Syndrome
- Neurosensitivity syndrome associated with abnormal CNS Pain processing.
- Excess levels of Substance P & Glutamate.
- Low levels of Serotonin & Norepinephrine.
Chronic Pain –
Fibromyalgia Associated Conditions & Syndromes

- Migraine Cephalgia
- Chronic fatigue syndrome
- Irritable bowel syndrome
- Depression
- Restless Leg Syndrome
- TMJ syndrome
- Myofascial pain syndrome
- Interstitial Cystitis

- Look for Connections!

Chronic Pain –
Fibromyalgia - Treatment/Medications

- Treatment of fibromyalgia consists of reassurance/education, gradual exercise, tricyclic antidepressants, SSRI’s.
- Muscle relaxers such as cyclobenzaprine help with sleep and spasm.
- Gabapentin, pregabaline and SNRI’s currently FDA indicated for treatment. These agents help with pain, depression and energy levels.
- Narcotics should be avoided if possible. Can be used in refractory cases.
Chronic Pain – Fibromyalgia - OMT

- OMT is very useful with fibromyalgia patients.
- Avoid direct techniques, HVLA.
- Indirect techniques, myofascial release and counterstrain are very effective.

Chronic Pain – Sympathetically Maintained Pain

- Complex regional pain syndrome (CRPS) is another type of neuropathic pain.
- Also know as reflex sympathetic dystrophy (RSD), causalgia, sympathetically maintained pain, as well as several other names.
- May result from a major injury, but it also can be caused by a relatively minor trauma.
Chronic Pain – Complex Regional Pain Syndrome

- Also known as Reflex Sympathetic Dystrophy (RSD) or Causalgia.
- Can also occur after stroke or MI.
- Associated with nerve dysfunction and increased sympathetic output to the injured region on the body.
- Autonomic homeostasis enters a disruptive cycle with nociception.

Chronic Pain - CRPS

- CRPS most often affects one of the extremities (arms, legs, hands, or feet) and is also often accompanied by:
  - "Burning" pain.
  - Increased skin sensitivity: Allodynia.
  - Changes in skin temperature: warmer or cooler compared to the opposite extremity.
  - Changes in skin color: often blotchy, purple, pale, or red.
  - Changes in skin texture: shiny and thin, and sometimes excessively sweaty.
Chronic Pain - CRPS

- Changes in nail and hair growth patterns.
- Swelling and stiffness in affected joints.
- Motor disability, with decreased ability to move the affected body part.

Osteopathic Algorithm For Management of Patients with Chronic or Recurrent Pain

Five Models of Osteopathic Care:
1. Biomechanical–Structural model
2. Respiratory–Circulatory model
3. Neurological model
4. Metabolic–Nutritional model
5. Behavioral–Biopsychosocial model

Foundations of Osteopathic Medicine: Philosophy, Science, Clinical Applications and Research, 4th E.
• Treatment of chronic pain requires an accurate diagnosis – First Step!
• Includes
  • Co-morbid medical diagnoses
  • Psychiatric diagnoses especially depression – screen (PHQ-9)
  • Social Factors
  • Behavioral Factors
• Evaluate and counsel patient on:
  • Proper nutrition
  • Levels of activity and exercise
  • Sleep and rest
  • The importance of creating and maintaining thoughts of wellness
Osteopathic Management of Chronic Pain

• Good evidence for:
  • Spinal manipulation
  • Exercise
  • Cognitive–behavioral therapy
  • Interdisciplinary rehabilitation

Osteopathic Management of Chronic Pain

• The inherent vitality and capacity for wellness of the patient is the focus for therapy.
• From the molecular, cellular, systemic, and regional perspectives, therapeutic strategies are conceived.
  • They are often stratified:
    • First, the physician will try treatment A and, if not successful, will change to treatment B.
  • They are typically multifaceted.
  • Sometimes, they are additive and even synergistic.
  • In this way, they represent a microcosmic, multidisciplinary, therapeutic plan.
• Of course, in some situations, multiple different health care providers are involved in delivering and coordinating the plan. This represents the interdisciplinary therapeutic team.
Osteopathic Management of Chronic Pain

• Osteopathic thinking has long considered pain as a symptom closely linked to somatic dysfunction.
• In addition to neuromuscular factors triggering nociception and the initial report of pain, biopsychosocial factors often worsen and perpetuate pain.
• Osteopathic physicians need to approach patients in chronic pain with support and empathy while drawing heavily from the expertise of their pain specialist colleagues.
• Caring for chronic pain often warrants an interdisciplinary approach, blending osteopathic thinking, and the best evidence-based knowledge about biopsychosocial treatments.
• The goal is to assist the patients internal healing and their ability to mount a homeostatic response. Each therapeutic prescription needs to be individualized.

Osteopathic Management of Chronic Pain
FOM 4th Ed., Chpt 14

<table>
<thead>
<tr>
<th>Model</th>
<th>Anatomical Correlates</th>
<th>Physiological Functions</th>
</tr>
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<tbody>
<tr>
<td>Biomechanical</td>
<td>Postural muscles, spine, and extremities</td>
<td>Posture and motion</td>
</tr>
<tr>
<td>Respiratory–Circulatory</td>
<td>Thoracic inlet, thoracic and pelvic diaphragms, tentorium cerebelli, costal cage</td>
<td>Respiration, circulation, venous, and lymphatic drainage</td>
</tr>
<tr>
<td>Metabolic–Energy</td>
<td>Internal organs, endocrine glands</td>
<td>Metabolic processes, homeostasis, energy balance, regulatory processes; immunological activities and inflammation and repair; digestion, absorption of nutrients, removal of waste; reproduction</td>
</tr>
<tr>
<td>Neurological</td>
<td>Head (organs of special senses), brain, spinal cord, autonomic nervous system, peripheral nerves</td>
<td>Control, coordination, and integration of body functions; protective mechanisms; sensation</td>
</tr>
<tr>
<td>Behavioral</td>
<td>Brain</td>
<td>Psychological and social activities, e.g., anxiety, stress, work, family; habits, e.g., sleep, drug abuse, sexual activities, exercise; values, attitudes, beliefs</td>
</tr>
</tbody>
</table>
Osteopathic Management of Chronic Pain

FOM 4th Ed., Chpt 14

Cognitive Behavioral Therapy: Based on RCT were shown superior to no treatment, placebo and wait list controls

<table>
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<tr>
<th>Strategy</th>
<th>Focus of Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cognitive restructuring</td>
<td>Self-statement reappraisal to change pain and distressing thoughts and decrease anxiety</td>
</tr>
<tr>
<td>Challenging irrational beliefs</td>
<td>Catastrophizing, all or none thinking, negative predicting, selectively focusing on pain, overgeneralizations, jumping to conclusions, fatalistic viewpoints</td>
</tr>
<tr>
<td>Motivating patients</td>
<td>Reduce social isolation, improve communication and problem-solving skills and outcome expectancy</td>
</tr>
<tr>
<td>Desensitize patients</td>
<td>Pacing activities, goal setting, muscular relaxation training</td>
</tr>
<tr>
<td>Reinforce activity</td>
<td>Operant and respondent conditioning</td>
</tr>
<tr>
<td>Teach self-management</td>
<td>Breathing techniques, biofeedback, imagery training, distraction techniques</td>
</tr>
<tr>
<td>Educate on mind/body/spirit</td>
<td>To understand body unity and our inherent capacity for self-regulation, self-learning, and the importance of healthy lifestyle choices</td>
</tr>
<tr>
<td>Relapse prevention</td>
<td>Pain coping skills, support groups, family interventions, caregiver support/education, breakthrough pain rescue planning</td>
</tr>
</tbody>
</table>

Osteopathic Management of Chronic Pain

• Exercise!
  • Meta-regression analysis found the best outcomes for exercise, incorporating the following elements:
    • Individualized regimens
    • Supervision
    • Stretching
    • Strengthening
  • When exercise included these features, compared to no treatment, pain scores went down nearly 20% and functions scores improved by 60%! The standard exercise course includes:
    • Educating the patient
    • Clearly defined goals
    • A program of 30 minutes performed twice daily and beginning with low-stress activities (walking, aquatic exercise, stationary bike with rest breaks)
    • Encouraging self-management

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Osteopathic Management of Chronic Pain

• OMT
  • Osteopathic medicine is at its most essential level a mechanism for changing pain-related behaviors.
  • From the OMT effects on segmental, regional, and global neuromusculoskeletal activity, to its balancing limbic and autonomic effects, it is attempting to influence neurologic and homeostatic cycles.
  • At the whole-person level, OMT affords the patient an opportunity to function and feel better.
  • Even if only transient, OMT permits the patient a glimpse of his or her capacity for having less pain.

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• Patients who have to “live with the pain” are taught to hold the following ideas as true:
  • My pain is real. It occurs in my body, and I have a reaction to it in my head.
  • I accept that I may need outside help to control my pain, and I refuse to quit or give in to the pain and the deterioration it causes.
  • At times, my pain has had an overwhelming influence on my life, but I believe that I can choose how I react to it.
  • My best efforts and those of the medical community have not stopped my pain. This is not necessarily a fault of mine or a shortcoming of medicine. I will no longer fight with myself about this or blame medicine. No fight, no blame.
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- Cont.
  - I will recognize some aspects of my coping with the pain that I am doing well and will also admit to myself mistakes that I have made.
  - I will forgive myself unconditionally for my past mistakes and forgive others whom I perceive are responsible for my pain and troubles.
  - I will fix any mistakes I've made struggling to live with the pain.
  - I will go forward with hope that the pain will someday pass or be relieved and, at the same time, recognize that I have to cope with the pain that I have.

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- cont
  - I will strive for higher goals, making pain a side issue, to be managed as necessary.
  - I now recognize there is more to my life than struggling with pain.
  - With this knowledge, I will separate myself from my pain management program or doctor, with the complete understanding that I may return at any time.
  - I understand that I have more important goals in my life than coping with the pain and I will strive toward them as my mission.
Chronic Pain - Summary

• Our understanding of nociception and the transition to chronic pain continues to expand.
• Acute pain is neurologically different from chronic pain, thus requiring appropriate treatment.
• Chronic pain becomes expressed neurologically as emotion; another example of neuroplasticity.
• Potential for chronic pain can depend on a variety of factors: environment, genetics, adverse childhood experiences, and others.
• Treatment options for chronic pain abound, including OMT, CBT, exercise, counseling, herbs and botanicals, and medications.
• This is a large and growing challenge for medicine and our society in general.

Chronic Pain

• Thank You!

• Any Questions/Comments?