AGL 2022

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SYLLABUS

PELVIC-605: Understanding Chronic Overlapping Pain Conditions

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The following members of AAGL have been involved in the educational planning and/or review of this course (listed in alphabetical order by last name).

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The following have agreed to provide verbal disclosure of their relationships prior to their presentations. They have also agreed to support their presentations and clinical recommendations with the "best available evidence" from medical literature (in alphabetical order by last name). Tim A. Deimling, MD, MS
Anna E. Reinert, MD
Ashley Gubbles, MD – Consultant: AbbVie
Sara R. Till, MD, MPH
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PELV-605: Advanced Robotics Course for Laparoscopic Surgeons

Chair: *M. Jean Uy-Kroh, MD, Ashley Gubbles, MD*

Faculty: *Tim A. Deimling, MD, MS, Anna E. Reinert, MD, Sara R. Till, MD, MPH*

Course Description

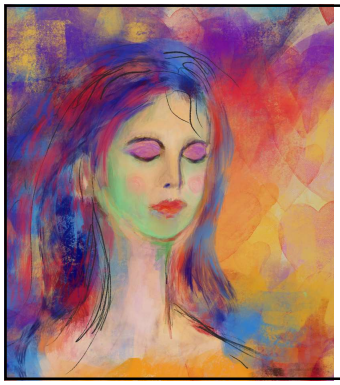
Pelvic pain is an enigma for many. When it isn't endometriosis, what do you do next? This course is designed for clinicians looking to understand more about chronic pelvic pain conditions, focusing on evaluation, diagnosis, and initial management along with expert pearls for those already managing these complex syndromes. Beginning with an overview of the biopsychosocial pathophysiology underlying chronic pelvic pain, the panel will lead participants through common syndromes focusing on diagnosis, initial management, and when to refer to a specialist. Evidence-based management will be discussed allowing attendees to feel confident in their management yet also providing guidance should pain persist.

Learning Objectives

At the conclusion of this course, the participant will be able to: 1) Evaluate and diagnose common chronic overlapping pelvic pain conditions; 2) Employ initial management of these conditions, both in isolation and conjunction with one another; and 3) Determine when referral to a sub-specialist is indicated for persistent pain.

Course Outline

7:00 am	Welcome, Introduction and Course Overview	M.J. Uy-Kroh/A. Gubbels
7:05 am	Understanding the Biopsychosocial Nature of Chronic Pelvic Pain	A. Gubbles
7:30 am	Myofascial Pain	S.R. Till
7:55 am	Interstitial Cystitis and IBS	A.E. Reinert
8:20 am	Vulvodynia	M.J. Uy-Kroh
8:45 am	The Role of Central Sensitization in Persistent Pain	T.A. Deimling
9:10 am	Questions & Answers	All Faculty
9:30 am	Adjourn	



Understanding the Biopsychosocial Nature of Chronic Pelvic Pain

Ashley Gubbels MD FACOG

Assistant Professor, Creighton University School of Medicine-Phoenix

Department of OBGYN

St. Joseph's Hospital and Medical Center, Phoenix

Objectives

- Understand the terminology of pain
- Demonstrate understanding of the complex changes in the CNS leading to CPP
- Acknowledge psychologic and social contributions to the pain experience

Terminology: Pain

Terminology: Chronic Pain

International Association for the Study of Pain (IASP 2020)

Pain lasting >3 months

Pain is always a personal experience that is influenced to varying degrees by **biological, psychological, and social factors.**

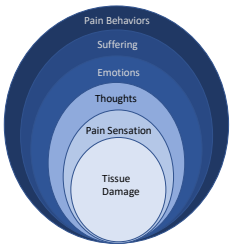
Pain and nociception are different phenomena. Pain cannot be inferred solely from activity in sensory neurons.

Through their life experiences, individuals **learn the concept of pain**.

A person's report of an experience as pain should be respected.

Although pain usually serves an adaptive role, it **may have adverse effects on function and social and psychological well-being**.

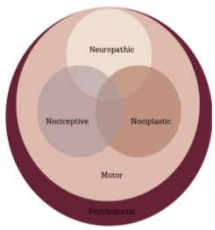
Verbal description is only one of several behaviors to express pain; inability to communicate does not negate the possibility that a human or a nonhuman animal experiences pain.



Terminology: Types of pain/mechanisms

Nociceptive	• arises from actual or threatened damage to non-neural tissue and is due to the activation of nociceptors
Neuropathic	• caused by a lesion or disease of the somatosensory nervous system • Clinical description, not a diagnosis
Nociplastic	• arises from altered nociception in the absence of actual or threatened tissue damage • Sensitization is the primary underlying mechanisms for nociplastic pain

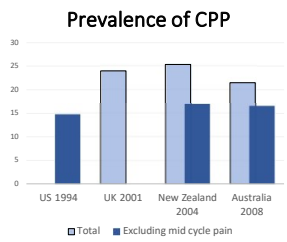
Types are not exclusive



Terminology: Chronic Pelvic Pain (CPP)

- Cyclical or non-cyclical lower abdominal pain
  - at least 6 months duration
  - Unrelated to pregnancy
  - Not exclusively due to dysmenorrhea or dyspareunia
- Disproportionately affects females but can affect men and individuals across the gender continuum
- Can be caused by all 3 types of pain mechanisms

Epidemiology



Condition	General Prevalence
Irritable Bowel Syndrome	12%
Vulvodynia	10-16%
Endometriosis	10%
Interstitial Cystitis/Painful Bladder Syndrome	3-7%
Myofascial pelvic pain	unknown

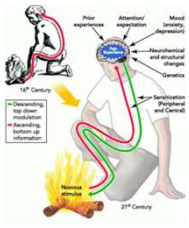
Epidemiology

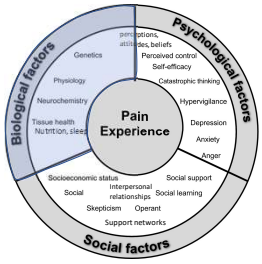
- Considerable impact on well being of women
- Associated with:
  - Physical dysfunction
  - Depression/anxiety
  - Personal economic losses
  - Poor quality of life
- CPP patients compared to women without CPP[1]
  - use three times more medications
  - four times more likely to undergo gynecologic surgery
  - five times more likely to undergo hysterectomy

Till SR, As-Sanie S, Schrepf A. Psychology of Chronic Pelvic Pain: Prevalence, Neurobiological Vulnerabilities, and Treatment. Clin Obstet Gynecol. 2019 Mar;62(1):22-36

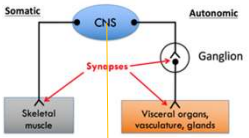
Biopsychosocial Model

Biology of pain





Physiology of Pain

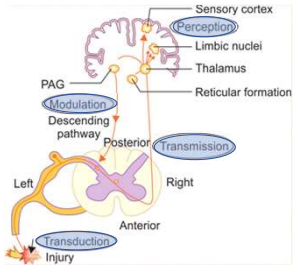


Somatic
- Innervate skin, skeletal muscle, joints
- Generally travels along single nerve distributions
- Results in specific localization to site of injury

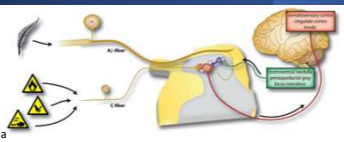
Visceral/Autonomic
- Innervate smooth muscle of hollow organs and peritoneum
- Mediated through sympathetic and parasympathetic divisions
- Results in poorly localized pain, non-specific/tough to describe
- May be associated with other autonomic symptoms (sweating, nausea, heart rate changes)

Acute (nociceptive) pain

- Noxious stimuli induce the physiologic processes meant to signal a problem and protect us from further injury
- Multiple steps of pain processing:
  - Transduction
  - Transmission
  - Perception
  - Modulation



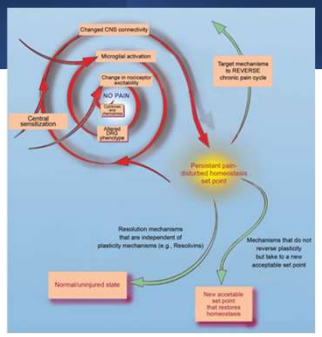
Acute Pain



- Primary hyperalgesia
  - Tissue damage decrease in threshold for C-fiber activation and pain perception
  - Occurs by upregulation of existing receptor sensitivity resulting in increased response to same stimulus along with increased number of receptors
- Peripheral sensitization
  - Uninjured area surrounding injury also becomes increasingly sensitive to both mechanical stimuli (secondary hyperalgesia) and innocuous stimuli (allodynia)
  - Process is meant as a protective mechanism to minimize use of the injured area and allow healing
- Once healing occurs, the system reverts to its pre-injured state

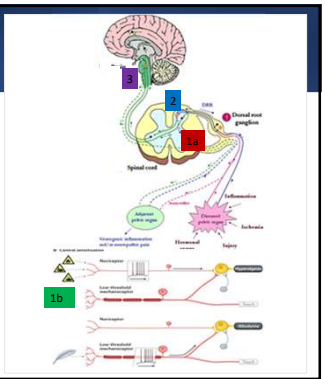
Chronic pain

- Maladaptation of acute system
- Pain itself modifies the function of the peripheral and central nervous systems
- Chronic pain is NOT long-lasting acute pain
  - Lasts longer than 3 months
  - Occurs in addition to pain from the original condition, often after condition has resolved
- Central sensitization is a key component of development



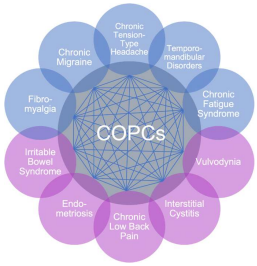
Central sensitization

- Defined as increased responsiveness of nociceptive neurons in CNS to their normal or subthreshold afferent input
- 3 phases
  1. Acute-increased response to activation and strengthening of connection between synapses
     - 1a. Wind up
     - 1b. Long-term potentiation
  2. Late-increased responsiveness to interneurons within spinal cord
  3. Disinhibition-loss of inhibitory neurotransmitters and interneurons



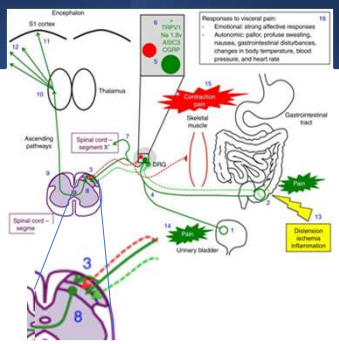
Why is pelvic pain different?

- Increased frequency of chronic overlapping pain conditions
- Effect on visceral systems
  - Bowel
  - Bladder
  - Reproductive/Sexual
- Recurrent nature of pain
- Psychological impact may be greater due to the effect on these intimate functions



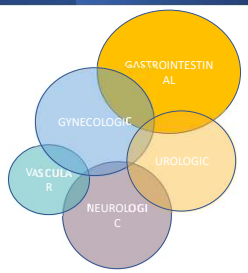
COPC's & Neural Convergence

- Visceral organs are typically nearly insensate
- Hypersensitivity arises from increased perception from stimulation of these structures
- Organ cross sensitization (cross talk)
  - Viscerovisceral convergency
    - Colon to bladder sensitization
      IBS patients often develop bladder irritability symptoms
    - Bladder to colon sensitization
      much less frequent
  - Viscerosomatic convergence
    - Abdominal myofascial pain or pelvic floor myofascial tension/pain



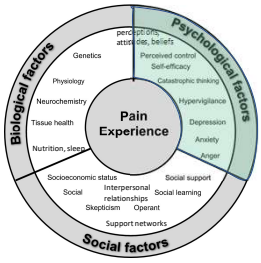
Etiologies of CPP

- CPP is an end symptom of various insults
- Results from multiple overlapping pain conditions with each contributing to the pain state
  - More than 1 pain condition is common
  - Those with >1 condition tend to have more pain
  - Pain severity is not consistently associated with degree of anatomic pathology or number of pain conditions
- Ultimately common neuropathology rather than anatomic changes drive symptoms



Howard F. Chronic pelvic pain. Obstetrics & Gynecology 2003; 101:594.

Psychology of pain



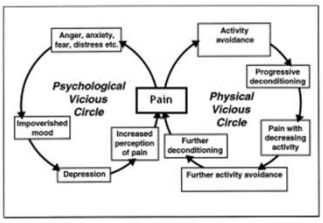
People with chronic pain are **three times more likely** to develop depression and anxiety than those without chronic pain.

Source: Harvard Medical School, 2017

Psychologic impact of pain

CPP patients commonly report:

- Stress-related symptom onset or increase in intensity
- Difficulty coping
- Depression, anxiety, and panic disorders
- Tertiary outpatient CPP clinic:
  - more than 50% of women had moderate to severe anxiety
  - more than 25% moderate to severe depression
- Increasing evidence that pain may be a stronger risk factor for developing depression or anxiety than the inverse



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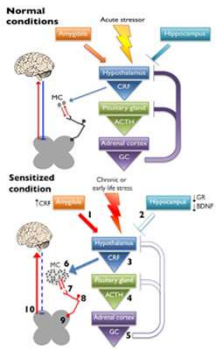
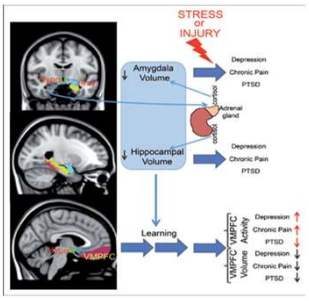
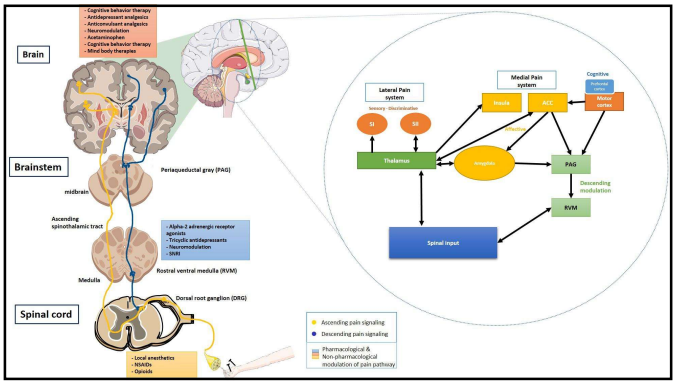
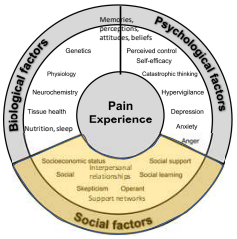






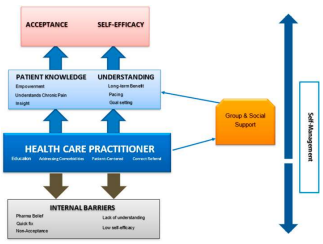
Social factors and pain



Social factors influencing pain

Contributors to function/dysfunction

- Social relationships/support
- Socioeconomic status
- Education level
- Access to care
- Cultural attitudes/beliefs

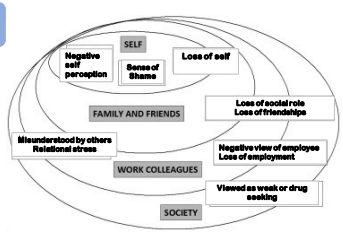


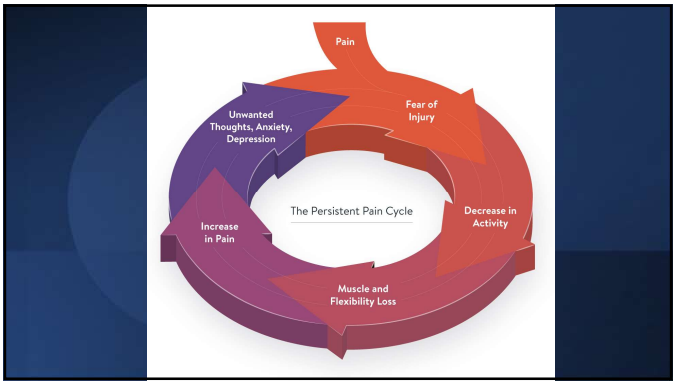
Armour, M., Sinclair, J., Ng, C.H.M. *et al.* Endometriosis and chronic pelvic pain have similar impact on women, but time to diagnosis is decreasing: an Australian survey. *Sci Rep* **10**, 16253 (2020)

Social impact of chronic pain

Impact

- Loss of self
- Relational isolation
- Increased educational/work absenteeism
- Financial stress
- Negative societal image
- Overall decreased quality of life





Pelvic myofascial pain: The missing puzzle piece?

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Disclosures

- No financial relationships
- Supported by NICHD 1K23HD09928301 (not directly related to this presentation)
- Many of the therapies discussed are off-label

Objective

- To review prevalence and pathophysiology of pelvic myofascial pain in chronic pelvic pain
- To review relevant anatomy and evaluation techniques
- To provide an overview of therapeutic techniques that can be considered to treat pelvic myofascial pain

CPP is not a diagnosis

- Endometriosis
- Myofascial muscle pain
- Vulvodynia
- Irritable bowel syndrome
- Interstitial cystitis/bladder pain syndrome
- Abdominal wall nerve entrapment
- Abdominal wall endometriosis
- Pelvic venous disorders
- Pudendal neuralgia
- Chronic SI joint pain
- Coccydynia

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Why should I care?

- How is this relevant to my surgical practice?
  - Unrealistic expectations = unhappy patient
- Significant symptom overlap with surgical conditions
  - Endometriosis
  - Fibroids
  - Pelvic organ prolapse
- Patients with untreated pelvic myofascial pain report worse pain and use more opioids in postop period
- Identifying all pain contributors allows
  - Better counseling about surgical outcomes
  - Higher likelihood of successful treatment of pelvic pain and meeting patient's goals

Abu-Alnadi et al, 2021

What is myofascial pain?

- Pain that originates from dysfunctional, hypertonic, hypercontractile, or hypersensitive muscles or fascia
- Often associated with trigger points
  - Hyperirritable nodule within skeletal muscle or associated fascia
  - Tenderness on palpation
  - Can cause referred pain or even autonomic symptoms
  - Can be latent (pain only when stimulated) or active (spontaneously painful)
- Can occur throughout the body
  - In CPP, very common in pelvic floor, abdominal wall, back

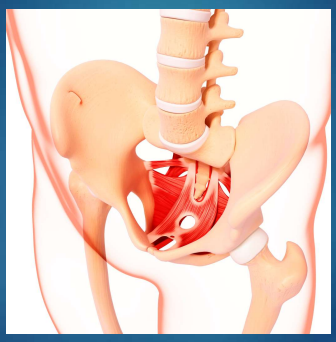
Prendergast & Weiss, 2003
FitzGerald & Kotarinos, 2003
Lavelle et al, 2007

Prevalence in CPP

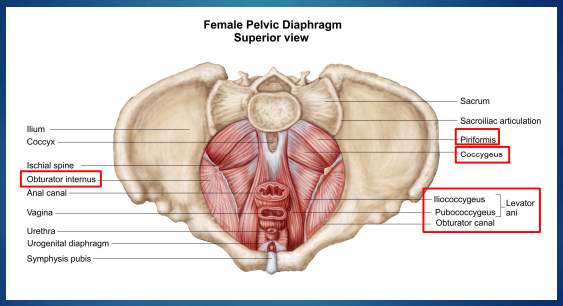
- Prevalence varies across studies, between 40-75%
- Often present in addition to (maybe in response to?) other CPP conditions
  - Endometriosis (22-65% prevalence)
    - Persistent pain s/p endometriosis surgery – **100% prevalence**
  - Vulvodynia (90%)
  - Interstitial cystitis/Bladder pain syndrome (55-80%)
  - Irritable bowel syndrome (? around 40%)
- Reminder – CPP contributing conditions are NOT mutually exclusive
- Unlikely to spontaneously resolve after treating other CPP contributors

Yong et al, 2014; Loving et al, 2014
Orr et al, 2020; Shafrir et al, 2021; Phan et al, 2020
Reissing et al, 2005
Yang et al, 2018; Gupta et al, 2022
Chitkara et al, 2004; Mulak & Paradowski, 2010

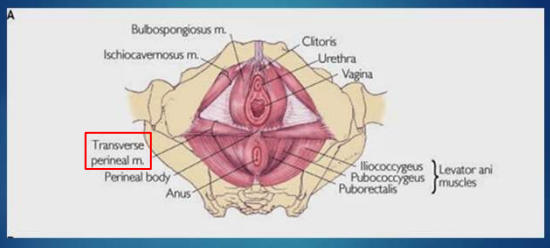
Pelvic myofascial pain



Pelvic myofascial pain

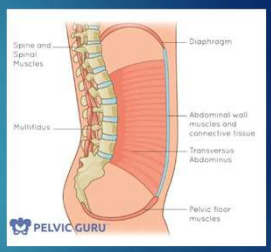


Pelvic myofascial pain



Pelvic myofascial pain

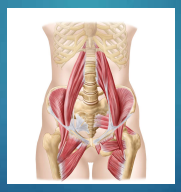
- Pelvic floor muscles have many functions
  - Extremely active as part of abdominal core
  - Function is intrinsically connected to abdominal wall, back, hips, lower extremities
  - Support bladder, bowel, and sexual functions

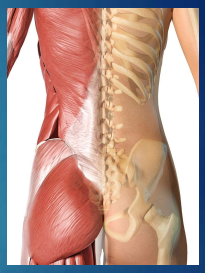


https://pelvicguru.com/

Pelvic myofascial pain

- The pelvis is not an island
- Consider adjacent systems
  - Abdominal wall
  - Low back
  - SI joints
  - Coccyx
  - Hips





Pathophysiology

- ???
- Central sensitization/nociplastic pain
  - Chicken or egg?
- Metabolic or neurochemical imbalance within trigger point
- Repetitive neuromuscular microtrauma
- Response to nearby dysfunction, inflammation, or pain
  - Dysfunction in one muscle tends to spread to nearby groups
  - Muscles are impacted by physiologic and psychologic stress

Shafrir et al, 2021; Vandyken et al, 2021; Kuner 2010
Gerwin et al, 2004; Shah & Gilliams, 2008
Tuttle et al, 2014; Jafri, 2014

Symptoms

- Feels like:
  - Aching, cramping
  - Spasm, sharp, shooting
  - Fullness, bloating
  - Heavy, falling out
  - Difficult to differentiate from visceral sensations
- Pain is located:
  - Pelvis (global or focal)
  - Vulva
  - Abdomen
  - Hips, buttocks
  - Low back
  - Location may shift

FitzGerald & Kotarinos, 2003

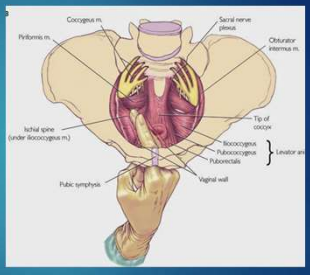
Symptoms

- Pattern:
  - Continuous or intermittent
  - May be exacerbated by
    - Intercourse
    - Full bladder
    - Urinary tract infection
    - Bowel movement
    - Standing or driving
    - Menstrual period
    - Physical/psychological stress
  - May be worse at end of day
  - May be improved by lying down, heat, stretching

Yong et al, 2014
Gupta et al, 2022
Meister et al, 2019

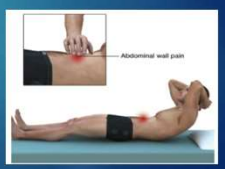
Diagnosis

- Clinical symptoms
- Exam
  - Palpation of muscle reproduces pain
  - Muscles may feel hypercontractile



Exam technique

- Palpate using 1-2kg pressure
- Ask patient
  - Pain intensity (0-10)
  - Reproduce symptoms
- Bilateral SI joints
- Bilateral lower quadrants of abdomen (medial to ASIS)
  - Insertion of iliacus
- Suprapubic
  - Insertion of rectus abdominis
- Carnett sign – abdominal wall
  - Myofascial or visceral?

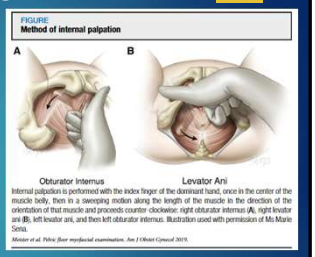


Meister et al, 2019

https://aneskey.com/abdominal-pain-12/

Exam technique

- Pelvic floor (single digit!)
  - Levator ani
    - Pubococcygeus
    - Iliococcygeus
  - Obturator internus
  - Coccygeus/Piriformis
  - Transverse perineal
  - Urethra, bladder
- Tips
  - Trauma-informed approach
  - Quick pause if pelvic floor reproduces symptoms
  - Pelvic floor first, then uterus/adnexa, speculum only if needed



Treatment

- Education!
- Physical therapy
- Muscle relaxants
- Injections
  - Botox injection
  - Anesthetic trigger point injection
- Acupuncture/Acupressure
- Exercise

Physical Therapy

- Pelvic floor, abdominal wall, hips, back
- Muscle control, mobilization, biofeedback
- Evidence
  - Chronic pelvic pain
    - Improved pain and dyspareunia
    - Decreased use of pain medication
  - Interstitial cystitis/bladder pain syndrome
    - Improved pain, urgency, frequency
    - Improved sexual function
  - Vestibulodynia
    - Improved pain and dyspareunia

Zoorab et al, 2015; Anderson et al, 2015
FitzGerald et al, 2009; FitzGerald et al, 2016
Goldfinger et al, 2016; Gentilcore-Saulnier et al, 2010

Physical Therapy

- Best practices
  - PT with pelvic floor expertise
  - Indication for referral
  - Clarify expectations
    - Temporary increase in pain
    - Internal/vaginal therapy
    - Home exercise program
- Treat co-existing pain conditions
  - IC/BPS
  - Vulvodynia
  - Dysmenorrhea/Endometriosis
- Consider vaginal estrogen before PT if indicated

Physical Therapy

- If inadequate response to PT or rapid recurrence of myofascial symptoms, reevaluate for:
  - Pudendal neuralgia
  - SI joint dysfunction
  - Hip joint dysfunction
  - Fibromyalgia

Cyclobenzaprine

- Muscle relaxant, TCA-like properties
  - ⇧ norepinephrine and serotonin
- Evidence
  - Fibromyalgia – improved pain, sleep, fatigue and tenderness
  - Acute myofascial pain (neck/back) – improved pain and function
  - No evidence in CPP (off-label use)
- Hits the chronic pain triad (myofascial pain, sleep disturbances, nociplastic/centralized pain)

Tofferi et al, 2004
Moldofsky et al, 2011
Childers et al, 2005
Landy et al, 2011

Other muscle relaxants

- Methocarbamol
  - Centrally acting, unclear mechanism of action
  - Hypothesized to decrease nerve transmission in spinal polysynaptic pathways and prolong refractory period in muscle cells
  - No evidence in CPP (off-label use)
- Tizanidine
  - Centrally acting alpha-2 receptor agonist
  - Decreases release of glutamate and aspartate (excitatory amino acids)
  - No evidence in CPP (off-label use)

Anxiolytics

- Vaginal diazepam
  - Local analgesic affect, GABA
- Evidence
  - Controversial…. (very much off label)
  - Small observational studies show improved pain or dyspareunia
  - Placebo-controlled RCTs studies show no benefit
  - Vaginal absorption
    - Serum diazepam levels consistent with oral use
    - Lower bioavailability but longer time to peak concentration
    - Long half-life
    - So, serum levels can accumulate with daily use!

Rogalski et al, 2010; Carrico & Peters, 2011
Crisp et al, 2013; Holland et al, 2019
Larish et al, 2019

Trigger point injections

- Trigger point injections
  - Pelvic floor or abdominal wall
  - Off-label
  - Mechanism unclear
    - Functional changes to primary afferent nerves
    - Dry needling
    - Hydrodissection
    - Substance P, histamine, neuropeptides

Scott et al, 2009
Hameroff et al, 1981

Trigger point injections

- Evidence
  - CPP – improved pain
  - Dysmenorrhea + myofascial pain – Improved pain
  - Fibromyalgia – improved pain and decreased NSAID use
- Techniques vary widely across studies
  - Bupivacaine or lidocaine
  - May add sodium bicarbonate to neutral pH
  - No evidence that steroids improve efficacy in RCT, may cause harm
  - Amount of anesthetic injected per site varies between <1ml to 10ml

Zoorab et al, 2015; Montenegro et al, 2015; Kim et al, 2013; Langford et al, 2007
Huang et al, 2014; Affaitati et al, 2009; Affaitati et al, 2011; Giamberardino et al, 2011
Labat et al, 2017

Trigger point injections

- "Best" practices
  - Consider coupling with PT
  - Special attention to incisions
  - Abdominal wall - may need spinal needle to reach fascia
  - Pelvic floor – pudendal nerve block kit
  - If improvement, repeat in 2-4 weeks
  - Goal is to graduate

Botulinum injection

- Off-label
- Mechanism
  - Decreases release of acetylcholine at presynaptic plate on neuromuscular junction -> flaccid paralysis of muscle in about 48-72 hours
  - New presynaptic plates grow in about 6-12 weeks and muscle contractility returns to baseline
  - Secondary mechanism – decrease afferent nerve activity by reducing available substance P and glutamate -> decrease sensation

Botulinum injection

- Evidence in pelvic myofascial pain - Mixed
  - Small studies, often observational, control group is difficult
  - Improved pelvic pain, dyspareunia, sexual function
    - Results varied widely – some with minimal effect, some with dramatic effect
    - Probably not as helpful for dysmenorrhea

Abbott et al, 2006; Adelowo et al, 2013; Mooney et al, 2021; Jha et al, 2021
Haraldson et al, 2020; Levesque et al, 2021

Botulinum injection

- Significant variability in practice patterns
  - Eligibility (first line treatment or failed PT)
  - Sedation
  - Dose (50-400u)
  - Muscle groups injected
  - Concurrent pudendal nerve block
  - Repeat PT after injection

Botulinum injection

- Best practices (meta-analysis)
  - At least 100u
  - Identify involved muscles by palpation just prior injection
  - Concomitant physical therapy
- Most experts recommend use in conjunction with PT
  - Not a replacement for PT, rather helps you move beyond plateaus in PT

Gari et al, 2022
Karp et al, 2019
Meister et al, 2021

Acupuncture

- Targets specific points along twenty "meridians" (pathways) that run through the body, modify chi
  - Acupuncture – thin needles
  - Acupressure – manual application of pressure
- Controversial, but low risk
  - Many studies are low quality, high risk for bias
  - Difficult to discern effect from placebo
  - Systematic reviews are inconsistent
  - BUT
    - Anecdotal success
    - Low risk of side effects when done by trained providers

Ernst, 2009

Acupuncture

- Endometriosis
- Intervention:
  - RCT, Sham/placebo (non-endometriosis sites)
  - 2x/wk for 5-10 weeks
- Results
  - Decreased pelvic pain
  - Decreased dyspareunia
  - Improved ability to perform daily activities
  - Improved quality of life

Wayne et al, 2008
De Sousa et al, 2016
Rubi-Klein et al, 2010

Acupuncture

- Evidence in CPP
  - Endometriosis – decreased pain and dyspareunia, improved quality of life
  - Abdominal wall myofascial pain – decreased pain
- Evidence in other myofascial conditions
  - Fibromyalgia – decreased pain and fatigue
  - Low back pain – decreased pain, improved functional status
  - Chronic tension headache – decreased frequency

Wayne et al, 2008; De Sousa et al, 2016; Rubi-Klein et al, 2010
Mitidieri et al, 2017
Mist et al, 2018; Hsieh et al, 2006; Lam et al, 2013; Linde et al 2013

Acupressure

- Dysmenorrhea
  - Decreased pelvic pain
  - Decreased # days with pelvic pain
  - Decreased need for pain medication
  - Improved physical function and social function

Bazarganipour et al, 2017
Blodt et al, 2018

Acupuncture

- Best practices
  - Adjunct therapy
  - Provider with expertise in endometriosis or pelvic pain – partner with reputable integrative medicine experts in your area
  - Likely need at least 5-8 sessions to know if they will be responder

Summary

- Pelvic myofascial pain is extremely common among patients with chronic pelvic pain
  - Pelvic pain conditions are not mutually exclusive
- Pelvic physical therapy is first line treatment
- Consider adjunct treatments in select patients
  - Muscle relaxants
  - Trigger point injections
  - Botulinum injections
  - Acupuncture/Acupressure

Interstitial Cystitis and Irritable Bowel Syndrome

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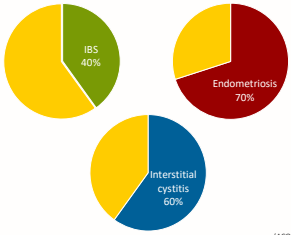
Disclosures

I have no financial relationships to disclose

Objectives

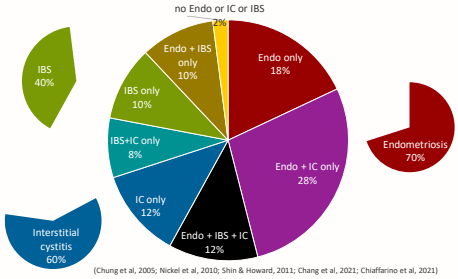
- Examine the role of Interstitial cystitis (IC) and Irritable Bowel Syndrome (IBS) in Chronic Pelvic Pain
- Define diagnostic criteria for IC and construct a differential diagnosis
- Diagram evidence-based management of IC
- Define diagnostic criteria for IBS and construct a differential diagnosis
- Diagram evidence-based management of IBS
- Explore multidisciplinary management of IC and IBS

Overlapping Pelvic Pain Conditions

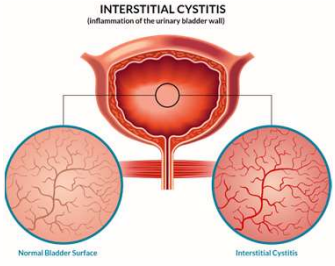


(ACOG, 2020; Hibner, 2021)

Overlapping Pelvic Pain Conditions



(Chung et al, 2005; Nickel et al, 2010; Shin & Howard, 2011; Chang et al, 2021; Chiaffarino et al, 2021)



(Image courtesy of Glycologix, Inc.)

Interstitial Cystitis/Painful Bladder Syndrome/Bladder Pain Syndrome

- Specific etiology unknown
- Hypotheses:
  - Repetitive physical bladder insults leading to chronic injury of the bladder epithelium
  - Disruption of Glycosaminoglycan (GAG) layer, leading to mucosal injury
  - Excess mast-cell activation
  - Visceral hypersensitivity of the bladder due to viscerovisceral convergence, viserosomatic convergence, and central sensitization
  - Neuroinflammatory upregulation, increased bladder neuronal density
  - Genetic or autoimmune factors
  - Painful subset of overactive bladder syndrome

(Castellanos et al, 2012; Hanno et al, 2015)

Diagnosis of Interstitial Cystitis

- "An unpleasant sensation (pain, pressure, discomfort) perceived to be related to the urinary bladder, associated with lower urinary tract symptoms of more than six weeks duration, in the absence of infection or other identifiable causes."
  - The Society for Urodynamics and Female Urology
- Pain that worsens with specific food or drinks
- Pain that worsens with bladder filling and improves with urination
- Presence of Hunner's lesions/glomerulations on cystoscopy not needed for diagnosis
  - American Urologic Association

(Hanno and Dmochowski, 2009; Hanno et al, 2015)

Differential Diagnosis of Interstitial Cystitis

Overactive bladder Hypertonic pelvic floor Infection (TB, CT, HSV, BV, yeast vaginitis) Recurrent UTI	Bladder stones Urethral diverticulum Bladder cancer Radiation cystitis	IBS Endometriosis Pelvic prolapse Pudendal neuralgia Vulvodynia

Red Flags:
- Microscopic or gross hematuria
- Sterile pyuria

"Complicated" IC/PBS/BSP:
- Incontinence / OAB
- GI signs/symptoms
- Gynecologic signs/symptoms

(Castellanos et al, 2012; Hanno et al, 2015)

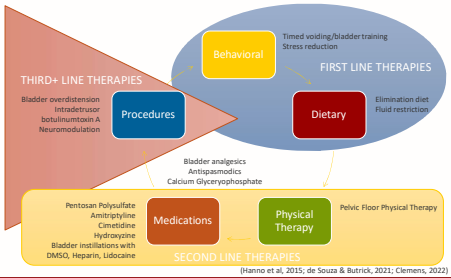
Evaluation of Interstitial Cystitis

- Validated Questionnaires
  - Pelvic Pain, Urgency, and Frequency Scale (PUF)
  - O'Leary-Sant Symptom and Problem Indexes
- Urinalysis and culture
- STD testing/urethral culture

Additional work-up considerations:
- Urine cytology (if hematuria or history of tobacco use)
- Post-void residual
- Urethral culture (if urethral pain)
- Potassium Chloride Stress Test (PST)
  - No longer recommended by AUA
  - Sensitivity 80%, specificity 97%
- Cystoscopy

(Castellanos et al, 2012; Hanno et al, 2015)

Treatment of Interstitial Cystitis



(Hanno et al, 2015; de Souza & Butrick, 2021; Clemens, 2022)

Interstitial Cystitis Diet

Elimination diet for 2-6 weeks followed by followed by gradual reintroduction of foods to identify individual triggers

Medications for IC - Projected 10 and 30 day cost

AUA-Recommended

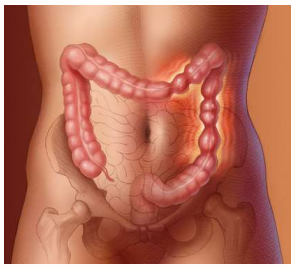
Medication	Frequency of Use	10 Days Cost	30 Days Cost
Pentosan polysulfate 100mg Tablet (Elmiron®)	TID	$289.20	$867.60
Cimetidine 400mg	Daily	$5.60	$16.80
Amitriptyline 25mg	Nightly	$0.70	$2.10
Hydroxyzine 25mg	Nightly	$0.70	$2.10
Phenazopyridine 200mg Tablet (Pyridium®)	TID (for up to 2 days)	$10.50	$31.50
Methenamine 1g Tablet	QID PRN	$33.56	$100.69
Hyoscyamine 0.125mg ODT (Levsin®/NuLev®/Levbid®)	Q4hr PRN	$10.80	$32.40
Montelukast 10mg Tablet (Singulair®)	Daily	$77.02	$231.05
Calcium glycerophosphate (Prelief®)	*2 Tablet QAC*	*$5.00*	*$15.00*

Italicized = OTC

(https://data.medicaid.gov/dataset/dfa2ab14-06c2-457a-9e36-5cb6d80f8d9)

Multidisciplinary Care for Interstitial Cystitis

- Refer to Pelvic Floor Physical Therapist
- Refer to Dietician to discuss IC Elimination Diet
- Refer to Urology/Urogynecology for further evaluation/management:
  - Urodynamic testing
  - Bladder instillations
  - Cystoscopy with hydrodistension
  - Cystoscopic intradetrusor botulinum toxin A
  - Neuromodulation:
    - Percutaneous tibial nerve neuromodulation (PTNS)
    - InterStim



Irritable Bowel Syndrome

- Specific etiology unknown
- Hypotheses:
  - Gastrointestinal motor dysfunction
  - Post-infectious alterations in GI tract neuromuscular function
  - Malabsorption / food intolerance
  - Visceral hypersensitivity of the colon due to viscerovisceral convergence, viscerosomatic convergence, and central sensitization
  - Genetic or autoimmune factors
  - Altered gut microbiome
  - Neuroinflammatory upregulation, increased neuronal density
  - Purely psychologic

(Aaronson et al, 2005; Viera et al 2002)

Diagnosis of Irritable Bowel Syndrome

- At least 12 weeks within the past 12 months of abdominal pain or discomfort which has 2 of 3 features:
  - Relieved with defection
  - Onset associated with a change in frequency of stool
  - Onset associated with a change in form (appearance) of stool

- Rome II Criteria

Additional symptoms:

- Abnormal stool frequency (greater than 3 bowel movements per day, or less than 3 bowel movements per week)
- Abnormal stool form (lumpy/hard or watery/loose)
- Abnormal stool passage (straining, urgency, or feeling incomplete evacuation)
- Passage of mucus
- Bloating or abdominal distension

(Aaronson et al, 2005)

Differential Diagnosis of IBS

SIBO	Gastrointestinal infection	Diverticulitis
Celiac's Disease	Inflammatory bowel disease	Diverticulosis
Lactose intolerance		Ovarian cancer
Colon cancer	Thyroid dysfunction	

ALARM SIGNS FOR POTENTIALLY MORE SERIOUS CAUSES OF IRRITABLE BOWEL SYNDROME SYMPTOMS

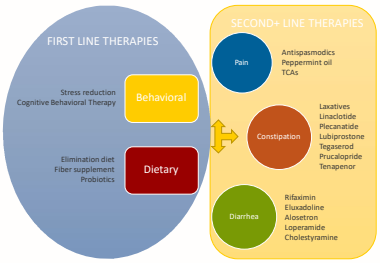
- Fever
- Unintended weight loss (> 10 lbs)
- Blood in stools
- Anemia
- Chronic severe diarrhea
- Abnormal physical findings
- Family history of inflammatory bowel disease or cancer
- Age > 50 years

Work-up considerations:

- Celiac's testing: - Antiendomysial antibody
  - Tissue transglutaminase
- TSH, free T4
- Giardia antigen
- Stool white blood cell count
- Stool ova and parasites
- Hydrogen breath test
- Colonoscopy
- Abdominal CT scan
- Pelvic ultrasound

(Aaronson et al, 2005; Viera et al 2002)

Treatment of Irritable Bowel Syndrome



(Brenner, 2021; Ford et al 2014)

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Dietary Interventions for IBS

Elimination diet for 2-6 weeks followed by followed by gradual reintroduction of foods to identify individual triggers

Traditional IBS diet

- regular meal pattern
- avoidance of large meal
- reduced intake of fat, insoluble fibers, caffeine, and gas-producing foods such as beans, cabbage, and onions

Characteristics and sources of common FODMAPs

	Word that corresponds to letter in acronym	Compounds in this category	Foods that contain these compounds
F	Fermentable		
O	Oligosaccharides	Fructans, galacto-oligosaccharides	Wheat, barley, rye, onion, leek, white part of spring onion, garlic, shallots, artichokes, beetroot, fennel, peas, chicory, pistachio, cashews, legumes, lentils, and chickpeas
D	Disaccharides	Lactose	Milk, custard, ice cream, and yogurt
M	Monosaccharides	"Free fructose" (fructose in excess of glucose)	Apples, pears, mangoes, cherries, watermelon, asparagus, sugar snap peas, honey, high-fructose corn syrup
A	And		
P	Polyols	Sorbitol, mannitol, maltitol, and xylitol	Apples, pears, apricots, cherries, nectarines, peaches, plums, watermelon, mushrooms, cauliflower, artificial sweetener containing polyols, and confectionery

FODMAPs: fermentable oligosaccharides, disaccharides, monosaccharides, and polyols.

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Medications for IBS Pain - Projected 10 and 30 day cost

ACG or AGA-Recommended

Medication	Frequency of Use	10 Days Cost	30 Days Cost
Amitriptyline 25mg capsule (Elavil®)	Nightly	$0.70	$2.10
Nortriptyline 25mg capsule (Pamelor®)	Nightly	$1.10	$3.30
Imipramine 25mg capsule (Tofranil®)	Nightly	$1.03	$3.09
Desipramine 25mg capsule (Norpramin®)	Nightly	$3.32	$9.97
Peppermint oil OTC (IBgard®, Pepogest®)	*TID PRN 30-60 minutes before meal*	*$4.16 (Pepogest) $27.46 (IBgard)*	*$12.48 (Pepogest) $82.43 (IBgard)*
Dicyclomine 20mg capsule (Bentyl®)	QID PRN 30-60 minutes before meal	$6.80	$20.40
Hyoscyamine 0.125mg ODT (Levsin®/NuLev®/Levbid®)	Q4hr PRN 30-60 minutes before meal	$10.80	$32.40
Simethicone 125mg Tab Chewable (Gas-X®)	*QID PRN*	*$4.28*	*$12.83*

Italicized = OTC

(Viera et al, 2002;Ford et al, 2014; Brenner, 2021; Wald, 2022)
(https://data.medicaid.gov/dataset/dfa2ab14-06c2-457a-9e36-5cb6d80f8d9)

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Medications for IBS-C - Projected 10 and 30 day cost

ACG or AGA-Recommended

Medication	Frequency of Use	10 Days Cost	30 Days Cost
Soluble fiber (psyllium/ispaghula) 15ml (i.e. MetaMucil®)	*Daily PRN*	*$3.06*	*$9.18*
PEG 3350 Electrolyte 17mg (Miralax®)	*Daily PRN*	*$0.45*	*$1.34*
Linaclotide 290mcg Capsule (Linzess®)	Daily	$156.90	$470.70
Plecanatide 3mg Tablet (Trulance®)	Daily	$162.70	$488.10
Lubiprostone 8mg Tablet (Amitiza®)	BID	$90.20	$270.60
Tegaserod 6mg Tablet (Zelnorm®)	BID	$49.33	$148
Prucalopride 2mg Tablet (Motegrity®)	Daily	$155.80	$467.40
Tenapenor 50mg Tablet (Ibsrela®)	BID	$499.07	$1497.20
Senna 8.6mg Tablet	*Daily PRN*	*$0.23*	*$0.68*
Bisacodyl 10mg Suppository (Dulcolax®)	*Daily PRN*	*$2.69*	*$8.07*
Milk of magnesia 30mL oral suspension	*Daily PRN*	*$1.57*	*$4.70*

Italicized = OTC

(Viera et al, 2002;Ford et al, 2014; Brenner, 2021; Wald, 2022)
(https://data.medicaid.gov/dataset/dfa2ab14-06c2-457a-9e36-5cb6d80f8d9)

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Medications for IBS-D - Projected 10 and 30 day cost

ACG or AGA-Recommended

Medication	Frequency of Use	10 Days Cost	30 Days Cost
Rifaximin 550mg Tablet (Xifaxan®)	BID x 2 weeks	$1237.29 (14 day cost)	
Eluxadoline 100mg Tablet (Viberzi®)	BID	$465.28	$1,395.84
Alosetron 1mg Tablet (Lotronex®)	BID	$171.64	$514.91
Loperamide 2mg (Imodium®)	*QID PRN*	*$11.54*	*$34.63*
Cholestyramine 4mg (Questran®)	TID	$25.87	$77.62

Italicized = OTC

(Viera et al, 2002;Ford et al, 2014; Brenner, 2021; Wald, 2022)
(https://data.medicaid.gov/dataset/dfa2ab14-06c2-457a-9e36-5cb6d80f8d9)

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Multidisciplinary Care for Irritable Bowel Syndrome

- Refer to Dietician to discuss FODMAPs Elimination Diet
- Refer to Psychotherapist for Cognitive Behavioral Therapy
- Refer to Gastroenterologist for further evaluation/management:
  - Colonoscopy
  - Infectious work-up
  - Autoimmune work-up
  - Hydrogen breath testing
  - Gastric motility testing
  - Advanced medical management

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Conclusions

- IC and IBS are common among patients with pelvic pain, often overlapping with conditions such as endometriosis
- Behavioral and dietary interventions are recommended first-line management for both IC and IBS
- A variety of medications can be used for second-line medical management of IC and IBS
- Multidisciplinary care can assist with management of IC and IBS

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Keck Medicine of USC

The Role of Central Sensitization in Persistent Pain

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Disclosures

I have no related conflict of interest

Objectives

1. Discuss the theory and mechanism of central sensitization
2. Review the challenges of specific to central sensitization in chronic pain
3. Explain strategies of multi-modal pain management with central sensitization

Central Nervous System

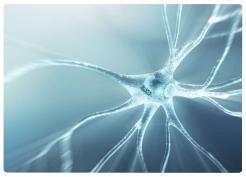
- Normal Adaptation of CNS
  - Enhanced protective function (Pain is Protective)
    - Repeated or Intense stimuli
      - Increased sensitization
      - Decreased activation threshold
      - Amplified response to subsequent input
  - Decreased/absent stimulation
    - Return to baseline

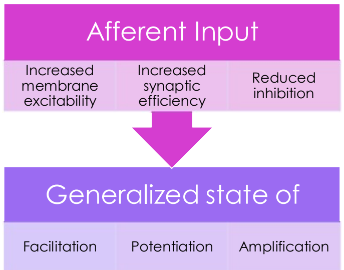
Central Sensitization

- Enhancement in neuronal function and circuits in nociceptive pathways
  - Increased membrane excitability
  - Increased synaptic efficacy
  - Reduced inhibition
  - Responds to activity, inflammation, neural injury

Central Sensitization

- Key Features
  - Allodynia
    - Pain from normally innocuous stimuli
  - Hyperalgesia
    - Exaggerated or prolonged response to noxious stimuli
  - Secondary Hyperalgesia
    - Pain spread beyond the site of noxious stimuli
- Central disassociation of presence, intensity and duration of stimuli and pain

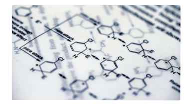




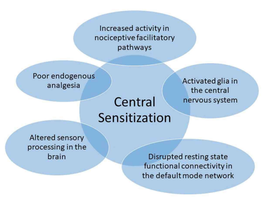
Mechanisms

Mechanism

- Altered Endogenous Analgesia
  - Down-regulation of efferent inhibition
    - GABA and Glycine
  - Pain interpreted as non-threatening typically suppressed by CNS
- Microglia Activation
    - Increase neuronal excitability
    - Increase TNF-alpha
    - Long-term potentiation and enhanced synaptic efficiency
    - Astrocyte activation



Central Sensitization



Treatment of central sensitization in patients with chronic pain: time for change? Jo Nijs, Laurence Leysen, Johan Vanlauwe, Tine Logghe, Kelly Ickmans, Andrea Polli, Anneleen Malfliet, Iris Coppieters & Eva Huysmans
https://doi.org/10.1080/14656566.2019.1647166
PUBLISHED ONLINE:

Central vs Peripheral Sensitization

Central Sensitization

- Threshold reduction in central nerves
- Responsive to non-noxious stimuli
  - Pain independent of insult
  - Not limited to location of insult
- Increased heat and Mechanical sensitivity

Peripheral Sensitization

- Threshold reduction in peripheral nerves
- Responsive to inflammation/injury
  - Intensity requirements decreased
  - Response amplified
- Typically, still requires insult
  - Symptoms limited to location of insult
- Associated heat sensitivity without mechanical sensitivity

Pathological Settings of Central Sensitization

- Peripheral Inflammation
  - Nerve terminals exposed to inflammation and nerve growth factors (NGF)
  - Activation of myelinated fibers
    - Low-intensity innocuous stimuli
    - Increased spinal neuropeptides
  - COX2 activation dorsal horn
    - Increased PGE2
  - Chronic inflammation
    - Inflammatory hyperalgesia
    - Heat hyperalgesia

Pathologic Settings of Central Sensitization

- Neuropathic Pain, Nerve Damage (including surgical)
  - Increased change relative to inflammation
  - Regenerative Changes
    - Altered synaptic function
    - Potential for myelinated fibers to develop new connections with nociceptive fibers
  - Seems to interrupt inflammatory pathway
    - Mechanism independent of inflammatory pathway
    - Potential mechanism of temp symptomatic relief in endometriosis excision

Cognitive-Emotional Factors

Cognitive-Emotional Sensitization			
Catastrophic beliefs	Stress	Perceived Injustice	Anger

Impacts		
Descending anti-nociceptive mechanisms	Expectations of Treatment	Adherence to Treatment

Central Sensitization Management

- Top-Down vs Bottom-Up approach
- Role of PAIN in treatment assessment
- Pharmacotherapy
- Psychosocial intervention

Top-Down Approach

- Treatment should be CNS focused
  - Individually tailored treatment
  - Pharmacologic agents focused on CNS pathways
  - Bio-Psycho-Social interventions
  - Address repeated peripheral insults as needed
    - NSAIDs
    - Physical Therapy

Pain Neuroscience Education

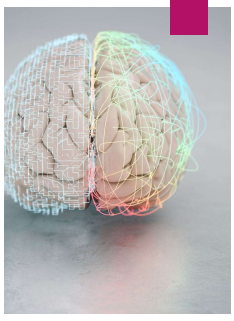
PAIN IS NO LONGER A RELIABLE MESSAGE

NATURAL FLUCTUATIONS IN THE NERVOUS SYSTEM SENSITIVITY IS GOING TO OCCUR

FIXED TREATMENT SCHEMES INDEPENDENT OF SYMPTOM FLUCTUATION

Pain Neuroscience Education

- Improves Maladaptive Pain Beliefs
- Decreased Pain and Disability
- Better Understand Their underlying condition
- Improved pain beliefs and coping strategies
- Improved therapeutic alliance
- Improved adherence to treatment



Pharmacologic Intervention

Overall Poor Long-term Success	Tricyclic Antidepressant	Serotonin-Norepinephrine Reuptake inhibitors	Alpha2-Delta ligands
• Particularly as solo treatment	• Amitriptyline, Nortriptyline	• Duloxetine	• Pregabalin, gabapentin

All Facilitate Descending Inhibition of Nociception

Pharmacologic Intervention

- Opioids
  - Absolutely Avoid for Long-Term treatment
  - Small, Short Effects
  - Opioid Induced hyperalgesia
  - Selective Pain Sensitization
  - Reward circuitry impacts can lead to chronic use even without abuse or misuse.

Psycho-Social

Psycho-Social

- Separation of short-term changes in pain and assessment of treatment effect
  - Persistent modification of treatment based on abnormal response to stimuli
- Focus on long-term improvements
  - Expectation management

Sleep Modification

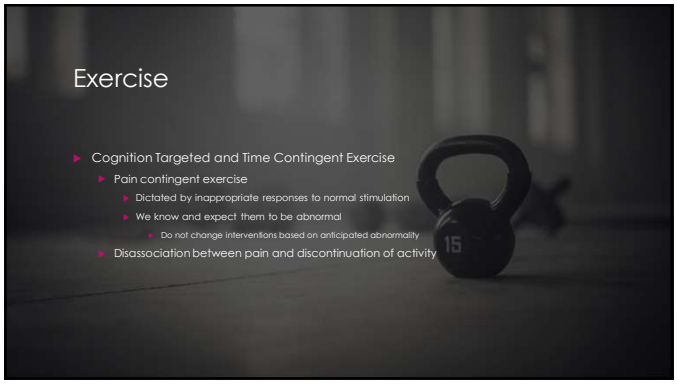
Sleep deprivation

hyperalgesia

Consistent and Adequate sleep

Improved descending anti-nociception

Exercise



- Cognition Targeted and Time Contingent Exercise
  - Pain contingent exercise
    - Dictated by inappropriate responses to normal stimulation
    - We know and expect them to be abnormal
      - Do not change interventions based on anticipated abnormality
  - Disassociation between pain and discontinuation of activity

Stress Management

Mindfulness Meditation

Cognitive Behavioral therapy

Cognitive Behavioral Therapy

- Maladaptive pain cognition
  - Targeted Therapy reduce CNS hyperexcitability
  - Increased prefrontal cortical volume

Conclusions

CENTRAL SENSITIZATION IS A COMPLEX MULTIFACTORIAL HYPERSENSITIZATION OF THE CNS THAT LEADS TO INCREASED INTERPRETATION OF PAIN IN THE ABSENCE OF NOXIOUS STIMULI OR TISSUE DAMAGE

CENTRAL SENSITIZATION IS DIFFERENT THAN PERIPHERAL SENSITIZATION AND TREATMENTS SHOULD BE FOCUSED ON CENTRAL MECHANISMS OF ACTION

PHARMACOTHERAPY ALONE PROVIDES POOR LONG-TERM SYMPTOMATIC RELIEF

TREATMENT SHOULD BE MULTI-FACTORIAL AND INDIVIDUALIZED

References

CULTURAL AND LINGUISTIC COMPETENCY & IMPLICIT BIAS

The California Medical Association (CMA) announced new standards for Cultural Linguistic Competency and Implicit Bias in CME. The goal of the standards is to support the role of accredited CME in advancing diversity, health equity, and inclusion in healthcare. These standards are relevant to ACCME-accredited, CMA-accredited, and jointly accredited providers located in California. AAGL is ACCME-accredited and headquartered in California.

CMA developed the standards in response to California legislation (Business and Professions (B&P) Code Section 2190.1), which directs CMA to draft a set of standards for the inclusion of cultural and linguistic competency (CLC) and implicit bias (IB) in accredited CME.

The standards are intended to support CME providers in meeting the expectations of the legislation. CME provider organizations physically located in California and accredited by CMA CME or ACCME, as well as jointly accredited providers whose target audience includes physicians, are expected to meet these expectations beginning January 1, 2022. AAGL has been proactively adopting processes that meet and often exceed the required expectations of the legislation.

CMA CME offers a variety of resources and tools to help providers meet the standards and successfully incorporate CLC & IB into their CME activities, including FAQ, definitions, a planning worksheet, and best practices. These resources are available on the CLC and IB standards page on the CMA website.

Important Definitions:

Cultural and Linguistic Competency (CLC) – The ability and readiness of health care providers and organizations to humbly and respectfully demonstrate, effectively communicate, and tailor delivery of care to patients with diverse values, beliefs, identities and behaviors, in order to meet social, cultural and linguistic needs as they relate to patient health.

Implicit Bias (IB) – The attitudes, stereotypes and feelings, either positive or negative, that affect our understanding, actions and decisions without conscious knowledge or control. Implicit bias is a universal phenomenon. When negative, implicit bias often contributes to unequal treatment and disparities in diagnosis, treatment decisions, levels of care and health care outcomes of people based on race, ethnicity, gender identity, sexual orientation, age, disability and other characteristics.

Diversity – Having many different forms, types or ideas; showing variety. Demographic diversity can mean a group composed of people of different genders, races/ethnicities, cultures, religions, physical abilities, sexual orientations or preferences, ages, etc.

Direct links to AB1195 (CLC), AB241 (IB), and the B&P Code 2190.1:

Bill Text – AB-1195 Continuing education: cultural and linguistic competency.
Bill Text – AB-241 Implicit bias: continuing education: requirements.
Business and Professions (B&P) Code Section 2190.1

CLC & IB Online Resources:

Diversity-Wheel-as-used-at-Johns-Hopkins-University-12.png (850×839) (researchgate.net)
Cultural Competence In Health and Human Services | NPIN (cdc.gov)
Cultural Competency – The Office of Minority Health (hhs.gov)
Implicit Bias, Microaggressions, and Stereotypes Resources | NEA
Unconscious Bias Resources | diversity.ucsf.edu
Act, Communicating, Implicit Bias (racialequitytools.org)
https://kirwaninstitute.osu.edu/implicit-bias-training
https://www.uptodate.com/contents/racial-and-ethnic-disparities-in-obstetric-and-gynecologic-care-and-role-of-implicitbiases
https://www.contemporaryobgyn.net/view/overcoming-racism-and-unconscious-bias-in-ob-gyn
https://pubmed.ncbi.nlm.nih.gov/34016820/