GLOBAL CONGRESS ON MIGS

AGL 2021

SYLLABUS

617-PELV:
Pelvic Pain - A Time to Heal
Professional Education Information

Target Audience
This educational activity is developed to meet the needs of surgical gynecologists in practice and in training, as well as other healthcare professionals in the field of gynecology.

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617-PELV: Pelvic Pain - A Time to Heal

Co-Chairs: Sawsan As-Sanie and Frank F. Tu

Faculty: Erin Carey, Michael Hibner, M. Jean Uy-Kroh, Juan Diego Villegas-Echeverri

This course brings diverse perspectives from a panel of expert clinicians to achieve the goal of healing the patient afflicted with chronic pelvic pain. Dialogue across the continuum of the course will reinforce key biological and clinical principles that have been found by these senior clinicians to effectively treat these conditions that go beyond a traditional gynecology-centric focus. Drawing on existing published literature and clinical perspectives, the panel will cover the rationale for approaching pelvic pain in an interdisciplinary and longitudinal fashion to optimize a woman's pelvic health trajectory. A blend of both procedural and non-procedural treatments are covered as part of this comprehensive care philosophy.

Learning Objectives: At the conclusion of this course, the participants will be able to: 1) Describe how to be aware of the core biological pathways responsible for emergence and persistence of pelvic pain; 2) describe an appropriate initial workup for patients with chronic pelvic pain; and 3) prescribe therapy for chronic pelvic pain that accounts for the multidisciplinary nature of this condition.

COURSE OUTLINE

2:30 pm Welcome, Introduction and Course Overview

2:35 pm An Island of One: Providing Comprehensive Multidisciplinary Pelvic Pain Therapy

M.J. Uy-Kroh

2:55 pm When Pain is in Your Head: CPP and Central Sensitization

J.D. Villegas-Echeverri

3:15 pm Pelvic Cross Organ Sensitization

F.F. Tu

3:35 pm Pins and Needles: Exploring the Science Behind Dry Needling, Acupuncture, Trigger Point Injections and Chemodeneration in Myofascial Pelvic Pain

E. Carey

3:55 pm Hysterectomy for Chronic Pelvic Pain: Is it Hype or the Best Hope?

S. As-Sanie

4:15 pm Pudendal Neuralgia and Nerve Entrapment

M. Hibner

4:35 pm Questions & Answers

5:00 pm Adjourn
PLANNER DISCLOSURE
The following members of AAGL have been involved in the educational planning of this workshop (listed in alphabetical order by last name).

Linda J. Bell, Admin Support, AAGL*
Linda D. Bradley, MD, Medical Director, AAGL*
Erin T. Carey, MD, MSCR
Honorarium: Teleflex Medical, MedIQ
Mark W. Dassel, MD
Contracted Research: Myovant Sciences
Linda Michels, Executive Director, AAGL*
Vadim Morozov, MD
Speaker: AbbVie
Consultant: Medtronic, Lumenis
Erinn M. Myers, MD
Speakers Bureau: Laborie Medical Technologies, Teleflex Medical
Other: Unrestricted educational grant to support NC FPMRS Fellow Cadaver Lab: Boston Scientific Corp. Inc.

Amy Park, MD
Speaker: Allergan

Nancy Williams, COO, CME Consultants*
Harold Y. Wu, MD*
Sawsan As-Sanie, MD, MPH
Consultant: AbbVie, Bayer HealthCare, Myovant Sciences, Eximis
Royalty: UpToDate
Frank F. Tu, MD, MPH
Consultant: AbbVie, Myovant Sciences, UroShape
Royalty: Wolters Kluwer

FACULTY DISCLOSURE
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).

Sawsan As-Sanie, MD, MPH
Consultant: AbbVie, Bayer HealthCare, Myovant Sciences, Eximis
Royalty: UpToDate
Erin Carey*
Michael Hibner, MD*
Frank F. Tu, MD, MPH
Consultant: AbbVie, Myovant Sciences, UroShape
Royalty: Wolters Kluwer
M. Jean Uy-Kroh, MD*
Juan Diego Villegas-Echeverri, MD*

Content Reviewers have nothing to disclose.

Asterisk (*) denotes no financial relationships to disclose.

All relevant financial relationships noted have been mitigated.
We have a problem

How can I fix it?

Every clinician's journey is unique, much like our patients

Questions I get asked the most
- Experience setting up 2 very different chronic pain programs

Framework to set yourself up to help & treat patients

Themes

You don’t know what you don’t know
- Knowledgeable colleagues
- Local & National Resources

A robust strategic plan

Imperative to know:
- what drives your clinical landscape
- what drives YOU professionally

What approach works best for you & your patients?

Starvation amidst a bounty
INERdisciplinary v. MULTIdisciplinary

The essence of multidisciplinary approach inherently involves frank, open discussion with both colleagues & patients.
- colleague selection is important but also a luxury

Know who is driving the bus

Comprehensive Recipe(s)

No singular fix.
"One pot recipe, 7 ingredients"
Elusive & misguided but here is a recipe... encourage you to break the mold

The role of standardization

* Most impactful *

Reset how YOU regard pain

Today's panel of speakers and their topics exemplify this

The context of pain influences our patients & our treatment selection
The Fix

**Self directed education**
- Reading
- Professional discussions
- Observations
- Roadtrips
- Trans disciplinary

PAIN has similar features and struggles

Caveats & frustrations

Poor correlation between extent of disease and severity of pain.

Medical therapies are non-specific & effectively treat other causes of CPP.

Just because a patient responds to Rx it does NOT confirm the patient has the disease

Reset how you think about pain

- Pain ≠ Pain perception

Pain perception is the result of an intricate processing system
We have a problem

How can I fix it?

Every clinician's journey is unique, much like our patients.

Questions I get asked the most:
- Experience setting up 2 very different chronic pain programs

**Framework** to set yourself up to help & treat patients—intentional v. allocated, self directed study not just referral

**BUILD a community of colleagues**

Don’t be afraid to say, “I don’t know,” to ask for help, to refer.


Have compassion. Validate. Be honest. Reach out. If need be, restart.
When the pain is in your head: Central Sensitization and Chronic Pelvic Pain

Juan Diego Villegas-Echeverri, MD
Unidad de Laparoscopia Ginecológica Avanzada y Dolor Pélvico
ALGIA
Clínica Comfamiliar
Pereira- Colombia
jvillegas@algia.com.co
www.algia.com.co

I have no financial relationships to disclose

Learning objectives

• At the conclusion of this activity, participants will be better able to:
  • Understand the differences between acute and chronic pain
  • Recognize the neurobiological and pathophysiological characteristics of Central Sensitization (CS)
  • Identify the role of CS in patients with persistent Chronic Pelvic Pain
  • Discuss the concepts of Chronic Overlapping Pain Conditions
  • Understand the clinical implications of CS and the role of intra-inter and multi disciplinary approach in patients with Chronic Pelvic Pain (CPP)

Outline

• Some definitions and general issues
  • Evolution of our understanding of pain. How has understanding pain processing evolved?
• When the pain is in your head: What is CS?
• How to diagnose CS?
• Clinical implications
• Conclusions

Some definitions and general issues

CS and CPP

Chronic Pelvic pain

• Non cancer
• Lasting more than 3 months *
• Pain symptoms perceived to originate from
  • Pelvic organs
  • Anatomical pelvis
  • Anterior abdominal wall below the umbilicus
  • Lumbo-sacral region
  • Hip – buttock
• Associated with negative cognitive, behavioral, sexual and emotional consequences
• Cyclical pelvic pain is considered a form of chronic pelvic pain if it has significant impact
How bad is it?

- 20 - 30% of all ObGyn Office visits
- 15 - 20% of all ER ObGyn Visits
- 20 - 40% of all Gyn Laparoscopies
- 41% never seek medical help
- 80% of patients are underserved (unsatisfactorily managed, undiagnosed, untreated)
- Only 15% of physicians surveyed said they “enjoyed” caring for CPP patients


QOL

- Post-coital pain
- General health
- Mood
- Deep dyspareunia
- Feeling depressed

How understanding (chronic) pain processing has evolved?

- Greek and Roman philosophers debated the origins of pain
- Pain emanated from the heart
  - Imbalance of humors or fluids in the body
- Early civilizations, religion and pain
  - Pain was sent by God to punish or test a person
  - It was often purposely not treated for fear of interfering with God’s will
- Sín, penance and repent
- Descartes
  - First to suggest pain came from the brain
  - Linear process from the periphery to the CNS
- Specificity, intensity, pattern, and gate control theories
- Biopsychosocial model and chronic pain

“Prior to the discovery of central sensitization, the prevailing view on pain processing in the central nervous system was of a largely passive neural relay that conveyed by encoded action potentials, information on the onset, duration, intensity, location and quality of peripheral noxious stimuli, much like a telephone wire, from one site to another.”

3 basic concepts

• Acute and chronic pain are not the same
• Nociception is not pain perception
• Anatomy of emotions overlaps Anatomy of pain

Chronic pain is completely different from acute pain

It is important to know the difference before you decide a treatment!

Pain and emotions: More than a feeling

• The anatomy of pain overlaps the anatomy of emotions
• Pain helps individuals to avoid future harm
• A spectrum of physical and psychological events
  • Tissue damage, visceral discomfort, shifts in attention, arousal, negative affect, and a desire to avoid repeating the experience
  • Although it also impairs wellbeing
• Pain Empathy
  • Empathic experiences activate somatosensory cortex and facilitate motor programs associated with nociceptive pain

Brain Regions Involved in Pain Processing:
Anatomy of pain overlaps the anatomy of emotions

1. Anterior cingulate cortex (ACC)
2. Prefrontal cortex
3. Insular cortex
4. Somatosensory cortex
5. Thalamus
6. Periaqueductal gray
7. Amygdala

Nociception vs. Pain Perception

No Brain, No pain!

Nociception
• Physiologic process of neural pathway activation by a potentially tissue damaging mechanical, thermal or chemical stimulus

Pain Perception
• Conscious perception of pain experience related to nociception influenced by somatosensory processing and psychosocial factors
Visceral and somatic structures in the pelvis share neural pathways
- Similar symptoms and making it difficult to differentiate visceral vs somatic
- Both visceral and somatic structures receive signals from and send input into the central nervous system
- Viscero-visceral cross-sensitization and viscerosomatic convergence
- Augmented or repeated input can enhance central nervous system responsiveness and decrease pain inhibition

COPC
- Chronic pelvic pain is associated with many visceral, neurological, musculoskeletal, and psychological symptoms
- Multiple pelvic pain syndromes often coexist
  - 48% of women with IC have endometriosis
  - 30% to 75% of individuals with IC have IBS

Why do we see patients with CPP who also have
- Are they crazy?
- Are they only depressed and anxious?
- Do they need a new surgery?
- They just want to bother us?
- Or that these painful disorders tend to cluster indicates something about these patients?

Finding many overlapping pain conditions suggests a common pathophysiology (and possibly a common treatment)

Definition
- CS has been operationally defined as an amplification of neural processing within the central nervous system to painful input
- It is responsible for the pain arising spontaneously
- It spreads beyond the site of injury (secondary hyperalgesia)
- It is exaggerated and prolonged in response to noxious stimuli
- It is associated with the development and maintenance of chronic pain
- It is responsible for the pain arising spontaneously
- It is associated with the development and maintenance of chronic pain
- It is exaggerated and prolonged in response to noxious stimuli
- It is associated with the development and maintenance of chronic pain
Enhanced pain response to noxious stimulus in somatosensory pathways

Bidirectional increase in synaptic efficacy

Reduction in inhibition

Amplification of the nociceptive signal

Augmented extension of pain in the somatosensory cortex

Temporal summation (a repetitive, consistent level of noxious stimulus to peripheral C-fibers)

Progressive increase in the perceived intensity of the stimulus

Decreased sensory threshold

Strengthening of normally ineffective synapses

Low threshold sensory inputs can activate the pain circuit

Initial peripheral insult

Peripheral excitation

Central sensitization

Descending modulation dysfunction

Top down or Bottom up?

Regardless of mechanism, both theories support changes in the way the central nervous system processes noxious input and how pain is ultimately perceived

From peripheral to central sensitization

Peripheral sensitization

- Decrease in threshold
- Increased receptive fields
- Spontaneous activity of peripheral ends of nociceptors
- It occurs after tissue damage and inflammation
- Enhanced propagation of nerve endings
- Two process have been implicated in this increased sensitivity
- Early posttranslational changes in the peripheral terminals of nociceptors
- Altered gene expression
- Primary hyperalgesia

Central sensitization

- Central sensitization contrast typically transfers in tactile allodynia and secondary hyperalgesia
- Pain is generated as a consequence of changes within the CNS that lead to alterations of how to interpret sensory inputs, rather than reflecting the presence of peripheral noxious stimuli
- Enhanced synaptic transfer
- Shorter lasting, activity triggered mechanisms
- Wind-up and heterosynaptic potentiation
- Long lasting effects
- Alterations in microglia, astrocytes, gap junctions, membrane excitability, and gene transcription
- Maintenance / persistence

Bottom up or Top down?

The "bottom up" theory supports an increase in pain perception due to excess noxious peripheral input that eventually sensitizes the central nervous system to the point of perceiving pain even when there is no peripheral drive

The "top down" theory suggests that changes already present within the central nervous system drive the perception of pain, regardless of peripheral noxious input

Regardless of mechanism, both theories support changes in the way the central nervous system processes noxious input and how pain is ultimately perceived
How to diagnose CS

Central Sensitivity Inventory

- Severity ranges have been recommended:
  - Subclinical = 0–29
  - Mild = 30–39
  - Moderate = 40–49
  - Severe = 50–59
  - Extreme = 60–100
  - $>40$ = high index of symptoms being consistent with a CSS

Clinical Criteria of Central Sensitization in Chronic Pelvic and Perineal Pain (Convergences PP Criteria)

- This clinical tool is applicable to patients with chronic pelvic and perineal pain lasting more than three months, reporting symptoms that appear to be disproportionate to the findings of physical examination and complementary investigations
- The presence of five or more items is suggestive of sensitization of pelvic pain

Other ways to test alterations in sensory perception

- Tactile Stimuli
  - Response to tactile stimuli (von Frey filaments)
- Vibration Stimuli
- Thermal Stimuli
- Electrical Stimuli
- Injection of Specific Pain Mediators
  - Capsaicin or hypertonic saline
- Ischemic Stimuli
- Distention Stimuli

Physical Exam

- Look for allodynia / Hyperalgesia
- Pressure pain sensitivity
- Pain sensitivity was assessed by applying discrete pressure stimuli to the thumbnail using a previously validated protocol
- Cotton-tipped applicator (CTA) test
  - A CTA was gently passed down across the abdomen in the region of the T10-L1 dermatomes

Additional references:

All central pain disorders share these clinical features

- Spread pain
- Verbal description of neuropathic pain
- More often they have current and lifelong pain
- Somatic symptoms
  - Fatigue, memory, sleep disorders
- Increased sensitivity to multiple sensory stimuli
  - Sound or light
- Higher incidence of co-morbidities (other related syndromes)
- Opioids are not effective and/or consistent in reducing pain
- More frequent in women (1.5 - 2X)
- Genetics - family
Neuroimaging of pain mechanisms: Is chronic pain a neurodegenerative disorder?

Brain morphology
- Voxel-based morphometry
  - Changes in regional gray matter (GM) density/volume
  - Changes in regional brain morphology
  - Decreases in GM volume
    - Thalamus, cingulate cortex, putamen, precuneus, secondary somatosensory cortex, superior temporal gyrus, cerebellum, and insular cortex

Brain function
- Using fluoro-deoxyglucose PET
  - Increased activity in prefrontal/orbitofrontal regions and left ventral posterior thalamus
  - Decreased activity in somatosensory regions of left hemisphere
- Functional MRI
  - Deactivation of brain regions in response to noxious thermal stimulation
  - Higher activity in the left entorhinal cortex and inferior/middle temporal gyrus
  - Higher activation in the left entorhinal cortex during non-painful tactile vestibular stimulation
  - Higher activation in the insular and frontal cortical regions

Clinical implications
(Medical management of CS is in its infancy)

There is enough published evidence to show that CPP is a Central Sensitization Syndrome

Regarding treatment, what does it mean that a patient with CPP has CS?
- Patients with CS respond differently to standard therapy
  - May respond less well to "peripherally" targeted therapies
    - AO
    - Surgical treatment
  - They may experience more acute and chronic post surgical pain
  - Early treatment to avoid transition from acute to chronic pain
  - Cure vs reduction of disability
Before
- Endometriosis era
- Negative laparoscopy
- Scattered management
- Surgery and surgery only. And if there is no improvement, more surgeries

Now
- General theories of chronic pain
- COPC and multiple pain generators
- Non-surgical management (single surgery)
- Central sensitization as a pain perpetuator
- Multi, inter and interdisciplinary approach
- Biopsychosocial model

When CPP is a Disease:
Treatment Goals - Never Promise a CURE
- Offer treatment for identifiable symptoms and concurrent psychological morbidity
- Restore normal function
- Improve quality of life by controlling symptoms
- Minimize disability
- Prevent recurrence of chronic symptoms and disability
- (Don’t promise a cure)

Treatment recommendations
- Several visits, long-term follow-up, and interaction with multiple clinicians
- Teams of clinicians from various specialties
- Pain management should focus on all biopsychosocial factors known to affect pain severity and recovery
- Patient education
  - Multiple pain contributors
  - Pain mechanisms
  - The role the central nervous system in pain modulation
  - Significant lifestyle changes
  - Self-management
- Combination mind-body and interdisciplinary interventions are recommended over single-agent pharmacotherapy or surgery
- Condition-specific therapies to be combined with adjuvant therapies

Start with the gold-standard in managing associated factors
- Hormone treatment for cyclical pain (or chronic pain with cyclical exacerbation)
- Surgical treatment should be reserved for specific conditions (excision or ablation of endometriosis)

Consider adding central action medication:
Antidepressants and antiepileptics for pain management*
- When conventional gynecological treatment has failed
- As first line therapy:
  - CPP with diffuse abdominal pain
  - CCCM and persistent pain
  - Previous negative diagnostic laparoscopy
  - Peripheral neuropathy
  - Potential suicide
  - Exist before
Nerve blocks
- Nerve blocks for diagnostic, prognostic, therapeutic, and prophylactic purposes
  - Somatic nerve blocks
  - Facet blocks
  - Lumbar Facet, Median Branch Blocks
  - Cervical Facet, Median Branch Blocks
  - Sacroiliac Joint Injection
  - Epidural Steroid Injection
  - Selective Nerve Root Block
  - Sympathetic Nerve Blocks
  - Blockade of the Stellate Ganglion
  - Lumbar Sympathetic Blockade
  - Celiac Plexus Block
  - Superior Hypogastric Plexus Block
  - Myofacial blocks / Trigger points

Cognitive-behavioral therapy
- Positive effect (40-60%) on function and QOL
- Better than pharmacological treatment alone
- Teaches self-control and management techniques
- Improves pain coping
- Reduces disability
- Why is it not more frequently used?
  - Although there is consensus on its usefulness
  - Doctors do not recommend it
  - Has no industry support
  - Difficult to bill
  - Not enough trained therapists

Exercise and CPP
- Improves short, medium and long-term pain scores
  - Aerobic exercise improves
    - Global Wellness (SMD -49)
    - Function (SMD -58)
    - Pain (SMD -65)
  - Muscle-building and strengthening exercise can also be effective
  - Improvement in pain and function is greater with
    - Guided training
    - Aerobic exercise
    - Swimming
    - Walking
    - Yoga

Sleep and pain
- Sleep deprivation
  - Causes symptoms similar to FMG
  - Impairs descending pathways of pain inhibition
  - Impairs the coping mechanisms
  - Why is it not more frequently used?
  - Hygiene instructions
  - Prescribe specific medications
  - Take advantage of adverse effects from other medications (TCAs)
  - Limit medications that can affect restful sleep
    - Long-acting opioids
    - Beta blockers - clonidine
    - SSRI

Physical therapy
- Improves daily function and pain
- PFTM
- Trained and experienced physiotherapists
- Individualized therapy
  - Pain location
  - Severity of disability
Before referring your patients for physical therapy

- Identify an experienced professional in your area
- Patients should be aware of vaginal and/or rectal manipulation
- Exercises at home
- At first there may be exacerbation of pain
- Multiple sessions

Alternative / complementary therapies: Acupuncture

- Slight-to-moderate improvement in pain and functioning
- No serious harms
- Superior to placebo in dysmenorrhea
- Limited results in non-cyclical pain

Functional Medicine: Diet and Chronic Pain

- "Functional nutrition"
- Genetics / Epi genetics
- Autoimmune / Inflammatory
- Emotional stress and cortisol levels
- Hormonal imbalance
- Insulin resistance
- Cortisol dysregulation
  - Plenty published data on diet and pain improvement in:
    - IBF
    - IBS
    - IC
    - Painful conditions related to stress
    - Endometrosis

Nutritional support can reduce chronic pain

- Increase omega-3 fatty acids
- Decreases intensity of dysmenorrhea
- Reduces serum levels of stress hormones
- Cut back on sugar
- Pro-inflammatory
- Less dysmenorrhea, urgency, nocturia and bladder pain
- Include dietary supplements such as selenium and zinc
  - Improves the integrity of the intestinal mucosa
  - Significant reduction in inflammation criteria
  - Improved pain scores.
- Optimize the composition of the gut microbiota
  - Prebiotics, probiotic supplements *, homemade cured meals
- Antioxidants
  - Melatonin reduces daily pain scores and dysmenorrhea

The presence of psycho-pathology must be identified

- Your ability to function
- The interpersonal relationships
- How has your pain affected your mood?
- Sexual function
  - How much stress do you have in your life?
  - Have you ever been a victim of physical, emotional or sexual abuse?
  - Describe your support system

Smartphone technology

- It allows to carry out interventions and psycho education in addition to the treatment
- Social networking: social support
- Reduces daily pain levels and pain interference with daily activity
  - Self hypnosis
  - Pain diaries
  - Follow-up to medical prescriptions and taking medications
  - Meditation
Some other considerations

- Acupuncture
- Biofeedback
- Manipulative/manual Medicine
- Hydrotherapy/spa treatments
- Hypnotherapy
- Meditation
- Mindfulness
- Guided imagery

En resumen

Cuando todo falla

• Chronic pelvic pain is a diagnosis
• Chronic pelvic pain is a chronic disease
• Multi, intra and inter disciplinary approach
• Therapies considered "standard" (hormone therapy or gynecological surgery) are not always indicated or useful
  - Opioids, NSAIDs, locks and surgical procedures are overused
  - Centrally acting analgesics and non-pharmacological therapies (exercise, yoga and CBT) are effective but rarely used
Always individualize the treatment!

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Pelvic Cross-Organ Sensitization

Frank Tu, MD, MPH
Dept. of Obstetrics and Gynecology
NorthShore Health
Pritzker School of Medicine

Disclosures
• Funding research NICHD, NIDDK, Dot Laboratories, Eximis
• Consultant – AbbVie, Myovant, UroShape
• Royalties – Wolters Kluwer

Learning objectives
• Review proposed peripheral mechanisms underlying pelvic visceral organ sensitization
• Examine comorbid uterine bladder symptomatology as a human model for studying cross-organ sensitization
• Consider strategies to reduce pelvic pain exploiting this risk pathway

Visceral pain hypersensitivity is characteristic of pelvic pain syndromes

Convergence Evidence
• Concomitant chronic diseases increased for patients with visceral pain
• Urinary Bladder/Colon
  – Anatomically and functionally related
  – Patients with IBS often show signs of urinary bladder hypersensitivity
• Reproductive tract /ureter
  – Both existing (dysmenorrhea) and latent (endometriosis, ovarian cysts) conditions enhance referred pain due to ureteral calculi

the urinary bladder may be more vulnerable to cross-modulation than other pelvic organs

*Extravasation of Evans Blue dye anti-inflammatory processes in the bladder appear less effective in inducing cross-sensitization in colon or uterus
**Underlying central contributions are complex**

- Psychological problems high among pelvic pain sufferers
  - Both community and tertiary
- Experimental evidence shows acute stress and anxiety alter GI function
- Animal studies show stress (maternal separation in childhood) can initiate visceral hypersensitivity in adult life (without histological changes)

**Early inflammatory or noxious events induce long-lasting pain sensitivity**

- D8-21 irritation in rats induces bowel pain sensitivity
- May include impaired development of opioid system

**Cortical contributions**

- Visceral pain stems from imbalanced CNS processing of sensory information
  - Thalamus to the sensorimotor cortex
  - Subsequent descending modulation of pain
- ??? why consistently seen changes in salience network do NOT result in widespread visceral pain sensitivity

**Factors in visceral sensitization**

**Complex changes promote organ sensitization**

- Following insult, inflammatory mediators released to reduce threshold
  - ↑ nociceptive channels
  - ↑ macrophage/mast cell cytokine release
- Spinal neuron DRG/DH transcription is altered by nociceptive input
- May be dependent on hormonal phase (proestrus)
- Antidromically produced DRR’s are generated in microcircuits between DH interneurons

**Regional mechanisms**

- Antidromic activity
- Dichotomizing fibers
- Intraganglionic, intra-axonal influence
- Local autonomic ganglia
Dysmenorrhea sufferers can exhibit silent bladder pain

Tu FF et al, Clin J Pain. 2013

Cross-organ sensitivity suggests broad dysfunction early on

Tu FF et al 2020 AJOG

Potential therapeutic implication/ prevention strategy

Women previously successfully treated for dysmenorrhea have equivalent pain with nephrolithiasis to DYS(-) women

• persistent afferent input to the spinal cord sustains altered central processing and leads to spontaneous motor abnormalities, hyperalgesia, pain and allodynia.

Potential therapeutic implication/ prevention strategy


Women previously successfully treated for dysmenorrhea have equivalent pain with nephrolithiasis to DYS(-) women

• persistent afferent input to the spinal cord sustains altered central processing and leads to spontaneous motor abnormalities, hyperalgesia, pain and allodynia.

Cross-organ treatment outcomes

• Giamberardino et al 2010
  • Prospective cohort study (with initial 6 mo OBS only comparator)
    - DYS treatment (OCP 6 mth) reduces IBS pain NRS 75-> 50 (n=8, 3 mth post OCP)
    - IBS dietary treatment(6 mth) improves improves total # of painful periods (n=13)
    - Asymptomatic ENDO treatment (laser LSC for fertility) reduces # kidney stone attacks (n=18, 6 mth postop)

Implications for surgical management of EAPP

• 3 yr followup of 981 eoe surgeries (2004-2012, CF, France)
  • 45% stage III-IV
  • <7% urological and GI procedures
  • 31% postop hormonal suppression

Conclusions

• Both peripheral and central mechanisms are involved in the generation and maintenance of pelvic cross-organ sensitization
• Need validated screening methods for asymptomatic sources of pelvic nociceptive input.
• Aggressively treat dysmenorrhea, EAPP, and silent sources of pelvic visceral pain, if we can identify them, EARLY.
References


Pins And Needles:
Exploring The Science Behind Dry Needling, Acupuncture, Trigger Point Injections And Chemodenervation In Myofascial Pelvic Pain

Erin T. Carey, MD MSCR
Associate Professor
Division/Fellowship Director MIGS
University of North Carolina

I have no relevant financial relationships to disclose

Objectives

• Demonstrate knowledge regarding the prevalence and etiology of myofascial pain
• Recognize differences in interventional myofascial treatments
• Interpret treatment data from available medical literature
• Employ unique injection techniques to improve interventional treatment of myofascial pelvic pain

Disclosures

Pelvic floor anatomy

Trigger point

Myofascial pain distribution
Prevalence in US Women

- 35%
- 30%
- 25%
- 20%
- 15%
- 10%
- 5%
- 0%

SOURCES

Prevalence of hypertonic pelvic floor disorders in U.S. adult females is ~10%

United States Adult Population (2020): 255 M
United States Adult Females: 130 M
Prevalence of Hyper PFD in U.S. Adult Women: 10%
Number of US Adult Females with Hyper PFD: 13M

High tone pelvic floor muscles
9%
7.4%

Pelvic tension myalgia (levator ani syndrome)

Myofascial Pelvic Pain within Chronic Pelvic Pain
Chronic Pelvic Pain: 15%
This includes pelvic floor myofascial pain, dyspareunia, vaginismus, and vulvodynia
5.7 – 26.6%

Chronic Pelvic Pain Prevalence: 15%
Within the CPP Patient Population:
- 58.3% have tender pelvic muscles (Montenegro, 2010)
8.7% of US Adult Females
- 30-70% cases involve musculoskeletal issues (Dani, 2017)
4.5 – 10.5% of US Adult Females

Myofascial pain prevalence

Acupuncture vs Dry needling

- Acupuncture:
  - Auricular and distal points allowed
  - Needle retention allowed
  - Electrical stimulation/heat/cold
  - Uses a meridian map to place needles
  - 885 hours of training + 250 supervised patients

- Dry needling:
  - No auricular or distal points
  - No needle retention
  - No electrical stim/heat/cold
  - Identification of clinical trigger points
  - PT + 50 hours of training + 200 supervised patients

Acupuncture

- Variation in main pain level
  - Analysis of the pooled data using a fixed-effects model showed that acupuncture had a positive effect on the primary pain level
    - MD = 1.35, 95% CI = 1.01–1.72, P < 0.0001

- Variation in peripheral blood CA-125 levels
  - Analysis of the pooled data using a fixed-effects model showed that acupuncture had a positive effect on the peripheral blood CA-125 level
    - MD = 5.9, 95% CI = 1.56–10.25, P = 0.008

- Clinical effective rate
  - Analysis of the pooled data using a fixed-effect model acupuncture had a positive effect on the clinical effective rate, vs controls
    - OR = 2.07, 95% CI = 1.24–3.44, P = 0.005

**Acupuncture**

### Systematic Review and Meta-Analysis

**The efficacy and safety of acupuncture in women with primary dysmenorrhea**

A systematic review and meta-analysis

Hye Lim You, KMD, Hee R. J., KMD, Yoon Kee Young Pak, KMD, Heung Lee, Su Jeong Heo, KMD, PhD, Jin Moa Lee, KMD, PhD, Kyung Sun Park, KMD, PhD

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**Acupuncture**

### Dry Needling

- Prospective studies using dry needling have not been performed in the pelvic region.
- Dry needling in other body regions has been shown to reduce pain and is non-inferior to wet needling.

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### Trigger Point Injections

- A prospective study using dry needling has not been performed in the pelvic region.
- Dry needling in other body regions has been shown to reduce pain and is non-inferior to wet needling.
Trigger point injections

- Trigger point injections (TPI) aka ‘wet needling’
  1. Depolarization of the associated nerve fibers due to the released potassium
  2. Washout of the nerve-sensitizing agents by the injection solution
  3. Interruption of the central feedback mechanism
  4. Focal necrosis of problem tissues due to the local anesthetics
- Minimal data to support trigger point injections for levator ani spasm in women with CPP

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- Distal
  1. 1 o'clock (yellow)
  2. 3 o'clock (black)
  3. 5 o'clock (orange)

- Proximal
  1. 1 o'clock (blue)
  2. 3 o'clock (red)
  3. 5 o'clock (green)

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Retrospective chart review on 101 women with a total of 257 visits for TPIs between 2012-2015

- Pain was measured using numerical rating scale (NRS)
- Significant improvements in pain noted at visits

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Ashi Acupuncture versus Local Anesthetic Trigger Point Injections for the Treatment of Abdominal Myofascial Pain Syndrome: A Randomized Clinical Trial


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Transvaginal Trigger Point Injections Improve Pain Scores in Women with Pelvic Floor Hypertonicity and Pelvic Pain Conditions

Trigger point injections

• Overall pain on NRS improved in both groups but faster in LTPI (4 weeks vs 7 weeks PT)

• Both interventions improved FSFI, but greater in women with PT

Onabotulinumtoxin A trigger point injections

• Onabotulinum toxin mechanism of action:
  1. Primary efferent effect of inhibiting the calcium-dependent release of acetylcholine at the presynaptic plate of the neuromuscular junction in humans
  2. Secondary afferent effect on sensation
     - may have a central action, explaining why its effects on sensation and pain may be of rapid onset

Onabotulinumtoxin A trigger point injections

• In retrospective studies, the effect of botox on myofascial pelvic pain +/- in combination with pelvic floor PT is promising

A randomized, double-blind, placebo-controlled trial of onabotulinumtoxin A trigger point injections for myofascial pelvic pain

• Overall pain on NRS improved in both groups but faster in LTPI (4 weeks vs 7 weeks PT)

• Both interventions improved FSFI, but greater in women with PT
Conclusions

- Acupuncture seems to alleviate pain by increasing pain thresholds by activating analgesic brain mechanisms through the release of neurohumoral factors.
- Limited but promising evidence in dry needling and trigger point injections in myofascial pain.
- In women with severe pain refractory for PT, Onabotulinumtoxin A trigger point injections may be a reasonable next step.
- Additional evidence for the interventional treatment of myofascial pain is needed.

References

Hysterectomy for chronic pelvic pain: Is it Hype or the Best Hope?

Disclosures

1. Consultant for Abbvie, Myovant Sciences, Bayer, Eximis
2. UpToDate author royalties

“But I know something else, too, and I know it as intensely as I know I want a baby: that something is wrong with my uterus. I can feel it, deeply specific yet unverified, despite so many tests and so much medical dialogue. I just sense that the uterus I have been given is defective.”

Vogue, February 14, 2018, By Lena Dunham

She tells you she cannot live like this and wants it all out, including her ovaries... What do you recommend?

A. She was 26 years old?
B. She had a prior negative laparoscopy?
C. She also had history of Interstitial Cystitis and Fibromyalgia?
Objectives

1. Define the incidence of & risk factors for persistent pelvic pain following hysterectomy
2. Discuss the utility of bilateral salpingo-oophorectomy at time of hysterectomy for CPP
3. Identify indications and cautions when prescribing postoperative hormone replacement therapy after BSO in women with endometriosis

Hysterectomy is often done for multiple symptoms in each patient

No Pain

Mild Pain

Severe Pain

Hystereotomy

Abnormal uterine bleeding

NO bleeding

Most women are satisfied

- 78-86% of all women undergoing hysterectomy report improvement in mental, physical or social function
- 60% report improvement in dyspareunia

Most women are satisfied, but there are risks

- Potential for serious morbidity
- Regret over loss of fertility
- Significant risk of persistent pain

Cardiovascular and metabolic morbidity after hysterectomy with ovarian conservation: a cohort study


Prevalence of persistent pain after hysterectomy


Presurgical FM score was predictive of persistent pain 6 months after surgery for every 1 point increase in FM score. 27% risk of failure to relieve pelvic pain.

11% reported <50% improvement in pain @ 6 months. Not predictive of persistent pain.

Adenomysis
Uterine weight
Surgical route

11% reported <50% improvement in pain @ 6 months.

Factors associated with persistent pain

• Pain elsewhere (Brandsborg 2007, VanDenKerkhof 2012)
• Younger age (Shakiba 2008, MacDonald 1999, Hills 1995)
• Lack of pelvic pathology (Hills 1995)
• Lack of private insurance (Hills 1995)
• Depression (Kjerulff 2000, Hartmann 2004)
• Pain catastrophizing (Martin 2011, Casey 2014)

Factors NOT associated with persistent pain

• Route of hysterectomy (Brandsborg 2009)
• Preoperative dysmenorrhea (Stovall 1990)
• Preoperative uterine tenderness (Stovall 1990)
• Uterine fibroid symptom score (Brandsborg 2009)
• Uterine weight (Stovall 1990, Brandsborg 2009)
• Adenomysis (Stovall 1990)

...i.e. clinical factors that often guide physicians to offer hysterectomy

BSO should not be taken lightly

PROS
• Prevent ovarian cancer
• Relief of pelvic pain
• Prevent recurrent ovarian cysts
• Prevent recurrent endometriosis

CONS
• Hot flashes
• Vaginal dryness
• Osteoporosis
• Cardiovascular disease
• All cause mortality

What about the ovaries?

Preserve ovaries
Bilateral oophorectomy

Level II-2
Large, prospective observation study

Does BSO prevent recurrent pelvic pain?

1. Evidence of urgent recurrence after hysterectomy
   1990: Single site (n=99), retrospective, any CPP
   No benefit of BSO

2. Effect of hysterectomy on pain in women with endometriosis: a population-based registry study
   2015: National (n=137), prospective, any age
   No benefit of BSO in any age

…”If BSO is deemed necessary, what are the recommendations for postoperative hormone replacement therapy?

Suggested approach to surgical menopause in women with history of endometriosis

1. Be aware that endometriosis can rarely occur in menopause and can rarely transform to malignancy
2. Benefits of HRT likely outweigh risk, in select populations
3. No reason to delay HRT after surgery, can start immediately
4. Consider selecting methods or alternative (tibolone, E-bazedoxifene), especially those with history of advanced disease or incomplete resection
She tells you she cannot live like this and wants ‘it all out’.  

What do you recommend?

#2. Consider hysterectomy when:
1. Conservative options have failed
2. You have maximally treated overlapping pain conditions & centralized pain
3. Anticipated benefits outweigh risks

...and you have done the following 2 things....

#2. Be prepared!
Detailed exam and imaging should identify most deeply infiltrative endo & obliterated culdesac

Must excise all endometriosis and normalize anatomy, but preserve at least one ovary when feasible
Better to refer or call for assistance than perform incomplete surgery

References
References

Pudendal neuralgia

Michael Hibner, MD, PhD, FACOG, FACS
Arizona Center for Chronic Pelvic Pain

Pelvic floor muscle spasm
• Physical therapy + Botox / SoLà

Mechanical nerve compression
• Surgical decompression

Disease of the nerve (HSV, DM)
• Treatment of underlying disease

Pudendal neuralgia is a rare problem with the pudendal nerve that can affect both men and women. The pudendal nerve runs through your pelvic region, including your genitals, urethra, anus, and perineum. Your perineum is the area between your anus and genitals. The condition is also known as pudendal neuropathy, pudendal nerve entrapment, cyclist's syndrome, pudendal canal syndrome, or Alcock's.

Pudendal neuralgia can cause pain, especially when you sit. Men with pudendal neuralgia may have pain in the buttocks, scrotum, penis, and perineum. Women with pudendal neuralgia may have pain in their buttocks, vulva, urethra, and perineum.
Pudendal neuralgia
(hernia)

≠

Pudendal nerve entrapment
(migraine)

Pudendal neuralgia

- Painful neuropathic condition in the area of innervation of the pudendal nerve
- Pudendal nerve entrapment (PNE) is compression of the pudendal nerve by scar, ligaments or surgical material

causing pudendal neuralgia

- pudendal n.
- b- inferior cluneal n.
- c- obturator n.
- d- genitofemoral n.
Pain in the area of innervation of the pudendal nerve
- Pain is neuropathic in nature
  - Paresthesia – burning, tingling, prickling, numbness sensation
  - Allodynia – pain in response to non-painful stimulus
  - Hyperalgesia – pain out of proportion to the stimulus
- Pain is more severe with sitting
  - Pain absent or significantly less when lying down
  - Pain less when sitting on the toilet vs. chair
  - Sensation of foreign body in the rectum or vagina (allosthesia)*

Urinary symptoms – frequency, urgency, hesitancy
- Dyschesia
- Dyspareunia
- Pain with orgasm
- Pain with sexual arousal
- Persistent genital arousal (PGAD)

Incidence 1/100,000 (tipna.org)
- 4% of patients with pelvic pain (orpha.net)
- 70/30 women/men ratio
- 70% unilateral

Pudendal nerve entrapment (not pudendal neuralgia) is always related to some traumatic event to the pelvis
Pudendal neuralgia is often mistaken with:
- Vulvodynia
- Pelvic floor tension myalgia (levator ani syndrome, vaginismus)
- Interstitial cystitis

Significant tenderness to palpation along the course of Alcock’s canal (vaginal)
Palpation of the course of the nerve reproduces symptoms (Tinel’s sign)
My protocol

- Rule out all the other causes (PFTM)
- Treat PFTM – Botox injections, SoLa therapy
- CT guided PN blocks
- Amiofix/Epifix injection
- Pudendal nerve decompression
- Cryoablation/pRF ablation of the nerve
- Pudendal nerve stimulator (Simwave/Freedom, Interstim)
**Pudendal nerve injury**

| Proximity of Prolift®
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**Postoperative care**

- Avoid activities causing pain
  - No prolonged sitting
  - No squatting
- Continue physical therapy
- Continue medications
- Some patients will benefit from additional injections of Botulinum toxin/SoLa or nerve blocks

| Proximity of Prolift®
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<th>Right (mm)</th>
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<td>Pudendal n.</td>
<td>15.6 (± 2.5)</td>
<td>18.0 (± 2.9)</td>
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<td>Inferior rectal n.</td>
<td>11.0 (± 1.5)</td>
<td>8.3 (± 2.6)</td>
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**Removal of mesh from the nerve**
Outcomes

- Acute pain (VAS)
- Quality of life (SF-36)
- Sexual functioning (FSFI)
- Sitting time (CST)
- Patient expectations of improvement

Outcomes (Nantes)

- First improvement in pain 4 months
- Maximum improvement in pain 24 months
- Results worse if neuralgia > 10 years

Outcomes (Nantes)

- In my practice 75% of patients have improvement of at least 20% after pudendal neurolysis

Outcomes of repeat surgery

- 10 patients, 1 lost to f/u
- Mean follow-up 23 months
- 8.7% global improvement
- 2 patients pain free
- VAS decrease from 7.2 to 4.0 (p=0.02)
- Improvement in sitting time in 8 patients from 5 minutes to 45 minutes (p=0.08)
- No worsening of symptoms
- Correlation between global improvement and comfortable sitting time R=0.86

If surgery fails

- Muscle spasm/pain
- Continued physical therapy + Botulinum toxin A injections/Solà
- Central pain
  - Ketamine infusion
  - Nerve blocks
- Continued nerve compression
  - Repeat surgery
Why does surgery fail?

- Wrong diagnosis
- Incomplete decompression
- Nerve too damaged to recover
- Different pain after surgery
  - Muscle spasm
  - Central pain
- Re-scarring of the nerve

The End

mhibner@azccpp.com
**Cultural and Linguistic Competency**

Assembly Bill 1195 was signed into law on July 1, 2006 requiring local CME providers, such as the AAGL, to assist in enhancing the cultural and linguistic competency of California’s physicians (researchers and doctors without patient contact are exempt). This mandate follows the federal Civil Rights Act of 1964, Executive Order 13166 (2000) and the Dymally-Alatorre Bilingual Services Act (1973), all of which recognize, as confirmed by the US Census Bureau, that substantial numbers of patients possess limited English proficiency (LEP). It is the intent of the Legislature to encourage physicians and surgeons, continuing medical education providers located in California, and the Accreditation Council for Continuing Medical Education to meet the cultural and linguistic concerns of a diverse patient population through appropriate professional development.

**Linguistic Competence**: Providing readily available, culturally appropriate oral and written language services to limited English proficiency (LEP) members through such means as bilingual/bicultural staff, trained medical interpreters, and qualified translators.

**Cultural Competence**: A set of congruent behaviors, attitudes, and policies that come together in a system or agency or among professionals that enables effective interactions in a cross-cultural framework.  

Cultural and Linguistic Competence: The ability of health care providers and health care organizations to understand and respond effectively to the cultural and linguistic needs brought by the patient to the health care encounter.

Cultural competence requires organizations and their personnel to:

- Value diversity.
- Assess themselves.
- Manage the dynamics of difference.
- Acquire and institutionalize cultural knowledge.
- Adapt to diversity and the cultural contexts of individuals and communities served.

California Business & Professions Code §2190.1(c)(3) states that associations that accredit continuing medical education courses shall develop standards before July 1, 2006, for compliance with the cultural competency requirements. The associations may update these standards, as needed, in conjunction with an advisory group that has expertise in cultural and linguistic competency issues. Cultural competency means a set of integrated attitudes, knowledge, and skills that enables a health care professional or organization to care effectively for patients from diverse cultures, groups, and communities. At a minimum, cultural competency is recommended to include the following: (A) Applying linguistic skills to communicate effectively with the target population. (B) Utilizing cultural information to establish therapeutic relationships. (C) Eliciting and incorporating pertinent cultural data in diagnosis and treatment. (D) Understanding and applying cultural and ethnic data to the process of clinical care, including, as appropriate, information pertinent to the appropriate treatment of, and provision of care to, the lesbian, gay, bisexual, transgender, and intersex communities.

Title VI of the Civil Rights Act of 1964 prohibits recipients of federal financial assistance from discriminating against or otherwise excluding individuals on the basis of race, color, or national origin in any of their activities. In 1974, the US Supreme Court recognized LEP individuals as potential victims of national origin discrimination. In all situations, federal agencies are required to assess the number or proportion of LEP individuals in the eligible service population, the frequency with which they come into contact with the program, the importance of the services, and the resources available to the recipient, including the mix of oral and written language services. Additional details may be found in the Department of Justice Policy Guidance Document: Enforcement of Title VI of the Civil Rights Act of 1964 [http://www.usdoj.gov/crt/cor/pubs.htm](http://www.usdoj.gov/crt/cor/pubs.htm).

Executive Order 13166, “Improving Access to Services for Persons with Limited English Proficiency”, signed by the President on August 11, 2000 [http://www.usdoj.gov/crt/cor/13166.htm](http://www.usdoj.gov/crt/cor/13166.htm) was the genesis of the Guidance Document mentioned above. The Executive Order requires all federal agencies, including those which provide federal financial assistance, to examine the services they provide, identify any need for services to LEP individuals, and develop and implement a system to provide those services so LEP persons can have meaningful access.

Dymally-Alatorre Bilingual Services Act (Assembly Bill 305) requires that state agencies that serve a substantial number of non-English-speaking people employ a sufficient amount of bilingual persons in order to provide certain information and render certain services in a language other than English.