AAGL 26

55TH GLOBAL CONGRESS ON MIGS

BOSTON, MASSACHUSETTS
NOVEMBER 13-16, 2026

Syllabus

PG-14003

Structured Management of Chronic Pelvic Pain and Its Mitigation

HONORARY CHAIR
Charles H. Koh, MD

SCIENTIFIC PROGRAM CHAIR
Nash S. Moawad, MD

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Michael Hibner, MD, PhD



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PG-14003: Structured Management of Chronic Pelvic Pain and Its Mitigation

Chair: Frank Tu
Co-Chair: Sawsan As-Sanie
Faculty: Sawsan As-Sanie, Ashley Gubbels, Georgine Lamvu, Frank Tu

Course Description

This course brings diverse perspectives from a panel of expert clinicians to achieve the goal of reducing the burden of chronic pelvic pain. We will focus on three areas of contemporary emphasis in covering state of the art management strategies: a) the underlying neurobiology of central sensitization shared across chronic overlapping pain conditions as a core mechanistic concept in treating CPP conditions that do not respond durably to conventional use of NSAIDs, hormonal suppression, or gynecological surgery; b) identification of regional/peripheral nerve dysregulation and when to consider targeted therapy; and c) novel opportunities emerging to reverse or potentially prevent the onset of CPP. Drawing on existing published literature and clinical perspectives, the panel will cover a blend of both procedural and non-procedural approaches to provide a comprehensive care philosophy.

Learning Objectives: *At the end of this course, the participant will be able to:* 1) Align evidence-based approaches to the history and physical exam in CPP alongside a globally endorsed classification framework (RU MOVVING SOME) for characterizing candidate mechanisms and modulators of chronic pain emergence and persistence; 2) Identify features suggestive peripheral nerve involvement in CPP and how to provide targeted treatment of such disorders in both the clinic and operating room; and 3) Recognize candidate high-profile targets for prevention of CPP following pelvic surgery or persistent episodic pelvic pain.

Course Outline

2:15 pm	Welcome, Introduction and Course Overview	F. Tu
2:20 pm	Efficient, Evidence Based History and Physical Exam Approach to Non-Endometriosis CPP	S. As-Sanie
2:45 pm	Neuropathic Pain - Evaluation and Management	A. Gubbels
3:10 pm	Real World Application of a CPP Classification System RU Movving Some – to Improve Clinician-Patient Communication, Validate Patient Experiences, and Enhance Both Clinical Care and Research Efforts	G. Lamvu
3:35 pm	Concepts Emerging to Prevent Development of CPP	F. Tu
4:10 pm	Discussion Questions & Answers	All faculty
4:15 pm	Adjourn	

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All individuals in a position to control educational content are required to disclose financial relationships with ACCME-defined ineligible companies within the past 24 months. AAGL identifies and mitigates all relevant financial relationships before individuals assume their respective roles. Educational content is also subject to independent peer review by individuals without relevant financial relationships.

Disclosure information and acknowledgment of any commercial support are provided to learners before the activity to promote transparency and maintain the independence and integrity of the education.

Asterisk (*) denotes no financial relationships with ACCME-defined ineligible companies to disclose.

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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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Linda D. Bradley, MD, Medical Director*
Linda Michels, Executive Director, AAGL*

COURSE CHAIR DISCLOSURE

Frank Tu, MD, MPH – Stockholder: Maipl Therapeutics

COURSE CO-CHAIR DISCLOSURE

Sawsan As-Sanie, MD, MPH – Consultant: Lilly, Gesynta, UCB Biopharma

FACULTY DISCLOSURES

The following faculty have agreed to support their presentations and clinical recommendations with the best available evidence from the medical literature (in alphabetical order by last name).

Sawsan As-Sanie, MD, MPH – Consultant: Lilly, Gesynta, UCB Biopharma
Ashley Gubbels, MD*
Georgine Lamvu, MD – Consultant: SoLa Pelvic Therapy
Frank Tu, MD, MPH – Stockholder: Maipl Therapeutics

MITIGATION OF RELEVANT FINANCIAL RELATIONSHIPS

All relevant financial relationships have been mitigated.

Drs. Lee, Lemos, Mikhail, and Chaudhari's planning decisions, as part of the Scientific Program Committee (SPC), were peer reviewed by other planners on the SPC who had disclosed no financial relationships with ineligible companies, and found to be fair, balanced, and free of commercial bias. Dr. Moawad's financial relationships ended 12/2024, thus mitigating them. For most of his AAGL Professional Education Committee service, Dr. Mourad had no financial relationships to disclose. He recently disclosed a new relationship as a Proctor for Intuitive Surgical. Since then, he has not participated in activities involving control or influence over accredited educational content; therefore, no additional mitigation was necessary.

Drs. As-Sanie and Tu's roles as Course Chair and Co-Chair were limited primarily to course oversight and moderation, rather than development of educational content. Any planning decisions or content under their influence were subject to independent peer review by individuals without relevant financial relationships.

The presentation content for course faculty, Drs. As-Sanie, Lamvu, and Tu, has been peer reviewed and found to be fair, balanced, and free from commercial bias.

Efficient, Evidence-Based History and Physical Exam Approach to Chronic Pelvic Pain.

Sawsan As-Sanie, MD, MPH
Robert K Ferguson and Virginia A Ferguson Professor in Obstetrics and Gynecology
Co-Chief, Gynecology
Director, Minimally Invasive Gynecologic Surgery
Director, Endometriosis and Chronic Pelvic Pain Consultative Clinic
University of Michigan

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Objectives

1. **Review** the mechanisms that lead to chronic pain, with particular emphasis on nociplastic pain disorders.
2. **Apply** an evidence-based, patient-centered approach to obtaining a comprehensive history in patients with chronic pelvic pain.
3. **Perform** a systematic, evidence-based physical examination to identify musculoskeletal, myofascial, neurologic, and visceral contributors to chronic pelvic pain.

3

Clinical scenario

Mary is a 43-year G2P1 female who presents with pelvic pain. She has a history of surgically treated endometriosis, but her pain recurred within 3 months of surgery

She reports daily lower pelvic cramping, and her pain i worse during her menstrual cycles and with intercourse. She also has fatigue, bloating, constipation, urinary urgency and frequency.

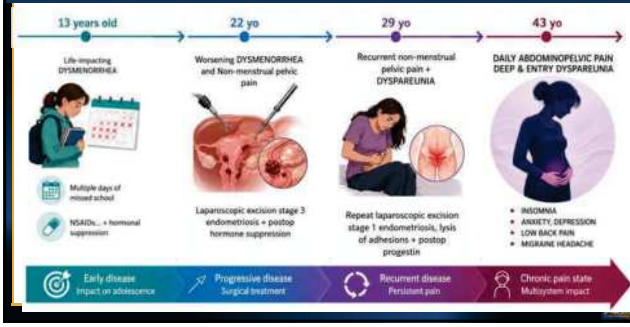
She is not sleeping well, has used most of her sick time at work, and her symptoms are affecting her ability to work, take care of and enjoy time with her children. She has had no improvement with birth control pills, NSAIDs



She tells you she cannot live like this and would like a hysterectomy

4

Clinical scenario



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Why is pelvic pain so difficult for providers to effectively manage?

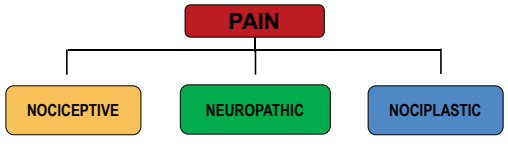
First must understand the mechanisms by which individuals experience chronic pain



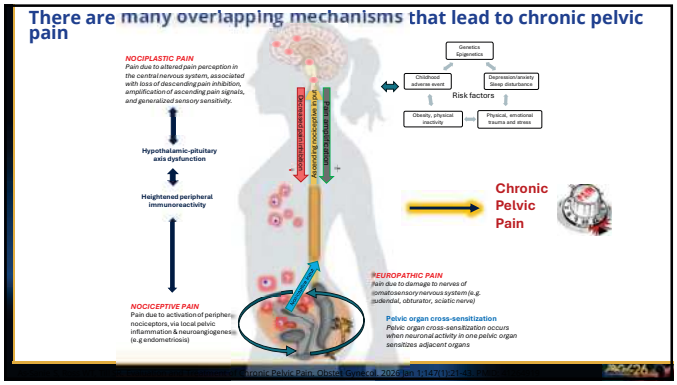
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IASP Mechanistic Classification of Pain

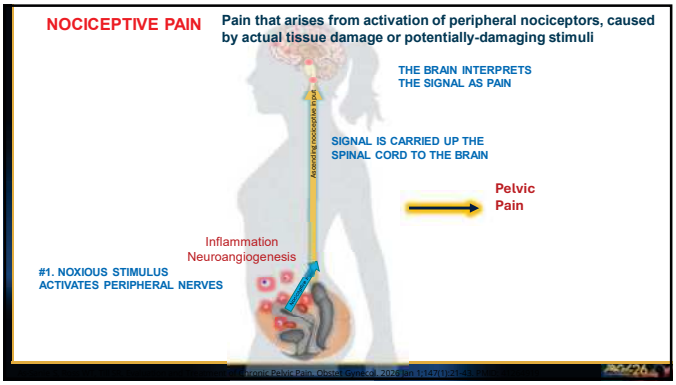
An unpleasant sensory and emotional experience associated with, or resembling that associated with, actual or potential tissue damage.



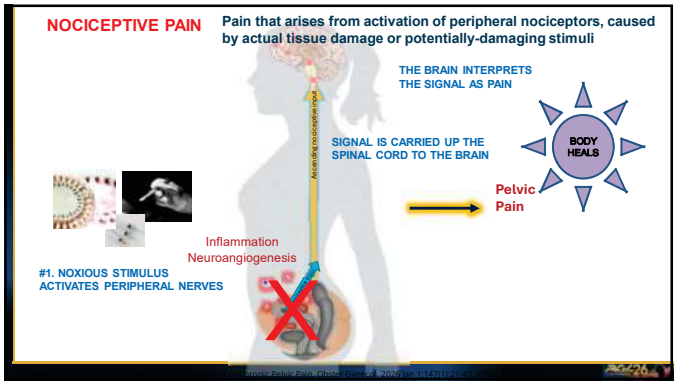
7


There are many overlapping mechanisms that lead to chronic pelvic pain
NOCIPLASTIC PAIN
Pain due to altered pain perception in the central nervous system, associated with loss of descending pain inhibition, amplification of ascending pain signals, and generalized sensory sensitivity.
Hypothalamic-pituitary axis dysfunction
Heightened peripheral immunoreactivity
NOCICEPTIVE PAIN
Pain due to activation of peripheral nociceptors, via local pelvic inflammation & neuroangiogenesis (e.g endometriosis)
Descending pain inhibition
Ascending nociceptive input
Genetics
Epigenetics
Childhood adverse event
Depression/anxiety
Sleep disturbance
Risk factors
Obesity, physical inactivity
Physical, emotional trauma and stress
Chronic Pelvic Pain
NEUROPATHIC PAIN
Pain due to damage to nerves of somatosensory nervous system (e.g. pudendal, obturator, sciatic nerve)
Pelvic organ cross-sensitization
Pelvic organ cross-sensitization occurs when neuronal activity in one pelvic organ sensitizes adjacent organs

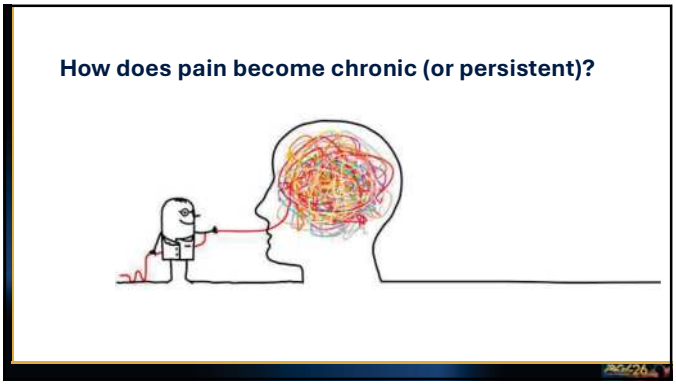
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NOCICEPTIVE PAIN
Pain that arises from activation of peripheral nociceptors, caused by actual tissue damage or potentially-damaging stimuli
THE BRAIN INTERPRETS THE SIGNAL AS PAIN
SIGNAL IS CARRIED UP THE SPINAL CORD TO THE BRAIN
Ascending nociceptive input
Pelvic Pain
Inflammation
Neuroangiogenesis
#1. NOXIOUS STIMULUS ACTIVATES PERIPHERAL NERVES

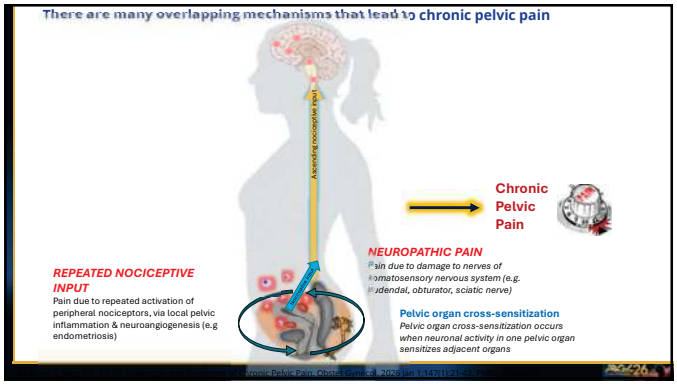
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NOCICEPTIVE PAIN
Pain that arises from activation of peripheral nociceptors, caused by actual tissue damage or potentially-damaging stimuli
THE BRAIN INTERPRETS THE SIGNAL AS PAIN
SIGNAL IS CARRIED UP THE SPINAL CORD TO THE BRAIN
Ascending nociceptive input
BODY HEALS
Pelvic Pain
Inflammation
Neuroangiogenesis
#1. NOXIOUS STIMULUS ACTIVATES PERIPHERAL NERVES

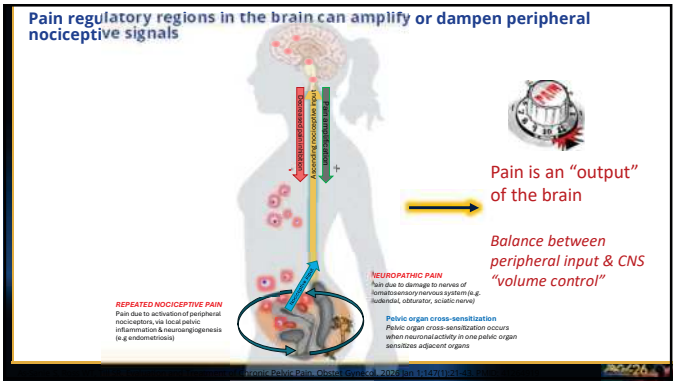
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How does pain become chronic (or persistent)?

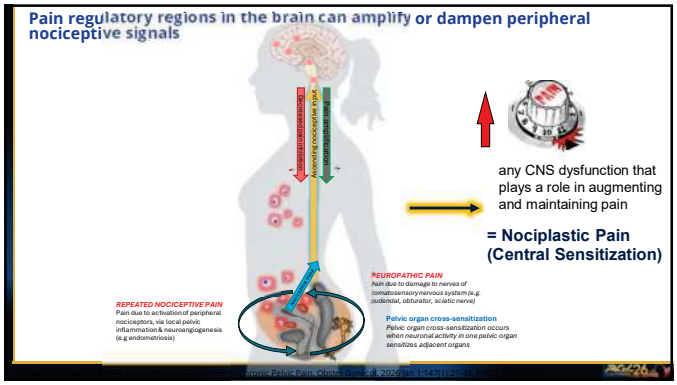
11


There are many overlapping mechanisms that lead to chronic pelvic pain
Ascending nociceptive input
Chronic Pelvic Pain
NEUROPATHIC PAIN
Pain due to damage to nerves of somatosensory nervous system (e.g. pudendal, obturator, sciatic nerve)
REPEATED NOCICEPTIVE INPUT
Pain due to repeated activation of peripheral nociceptors, via local pelvic inflammation & neuroangiogenesis (e.g endometriosis)
Pelvic organ cross-sensitization
Pelvic organ cross-sensitization occurs when neuronal activity in one pelvic organ sensitizes adjacent organs

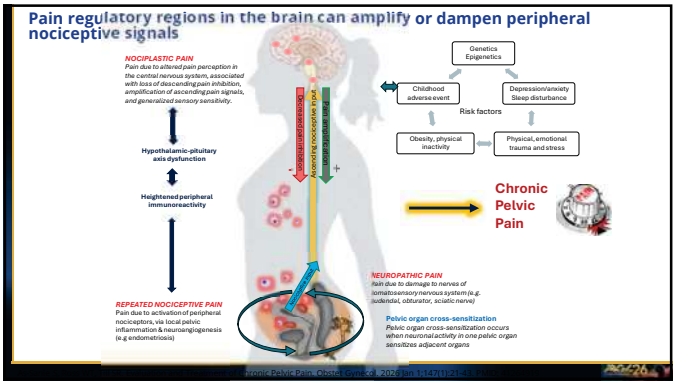
12


Pain regulatory regions in the brain can amplify or dampen peripheral nociceptive signals
Descending pain inhibition
Ascending nociceptive input
Pain is an "output" of the brain
Balance between peripheral input & CNS "volume control"
NEUROPATHIC PAIN
Pain due to damage to nerves of somatosensory nervous system (e.g. pudendal, obturator, sciatic nerve)
REPEATED NOCICEPTIVE PAIN
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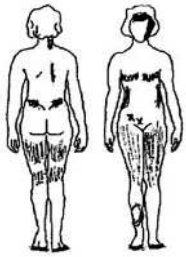
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Shared features of nociplastic pain syndromes

- Multifocal, widespread pain
- Higher lifetime history of pain throughout the body
- 1.5 – 2X more common in females
- Strong familial/genetic underpinnings
- Pain is often triggered or exacerbated by "stressors



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Not just a pain disorder.... Cardinal symptoms of nociplastic pain include



Greater sensitivity to internal sensations (somatic awareness)



Fatigue



Memory difficulties



Greater sensitivity to multiple sensory stimuli (sound, light)

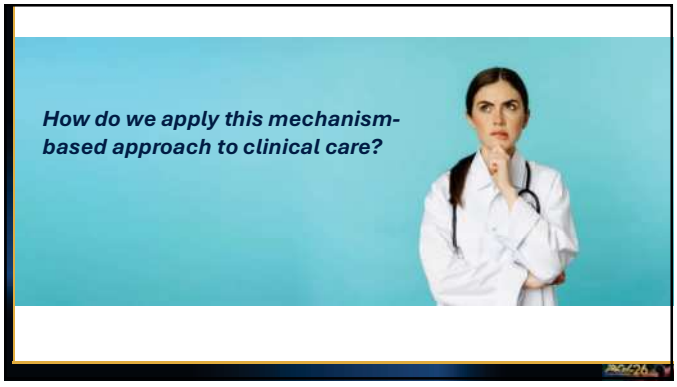


Poor, non-restorative sleep



Mood disorders (anxiety, depression)

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Chronic pelvic pain is a symptom, not a diagnosis

R U MOVVING SOMe *FIGO-IPPS Classification System for Female Chronic Pelvic Pain*

Reproductive	**Endometriosis,** adenomyosis, dysmenorrhea, fibroids, ovarian remnant
Urinary	Bladder pain syndrome/interstitial cystitis, urethral syndrome
Musculoskeletal	High tone pelvic floor dysfunction, piriformis syndrome, SI joint dysfunction, coccydynia
Other	not Otherwise specified (e.g. foreign body)
Vulvovaginal	Vulvodynia, vaginismus
Vascular	Pelvic vascular insufficiency, Nutcracker syndrome, May Thurner syndrome
Idiopathic	No pain contributor identified despite comprehensive clinical evaluation
Neurologic	Pudendal neuralgia, ilioinguinal neuralgia, iliohypogastric neuralgia, herpetic neuralgia
Gastrointestinal	Irritable bowel syndrome, inflammatory bowel disease, chronic diverticulitis

Sensitization
Overlapping Pain Conditions
Mental Health

FIGO PELVICPAIN

Lamvu G, Villegas-Echeverri JD, Allaire C, As-Sanie S, Carrillo I, Khalil S, Horne AW, Wang A, Munro MG. Developing the FIGO-IPPS "R U MOVVING SOMe" classification system for female chronic pelvic pain. Int J Gynaecol Obstet. 2025 Nov;171(2):550-565. doi: 10.1002/ijgo.70522. Epub 2025 Sep 8. PMID: 40922503

19

Chronic overlapping pain conditions frequently co-occur

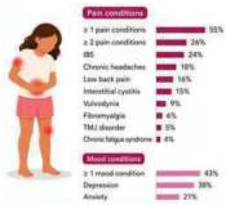
Adult patients with endometriosis:



- **19x** odds of urologic chronic pain syndromes
- **16x** odds of vulvodynia
- **5x** odds of irritable bowel syndrome
- **4x** odds of fibromyalgia
- **3x** odds of migraine headache

Schrepf A, Phan V, Clemens JQ, Maixner W, Hanauer D, Williams DA. ICD-10 Codes for the Study of Chronic Overlapping Pain Conditions in Administrative Databases. J Pain. 2020 Jan-Feb;21(1-2):59-70. PMID: 31154033

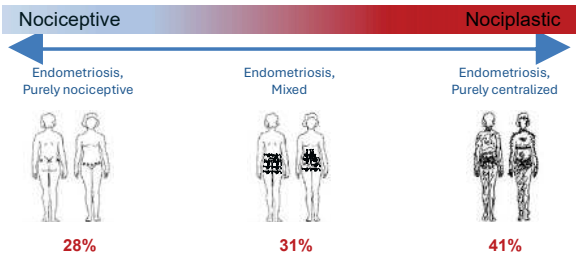
Adolescent patients with endometriosis:



Smorgick N, Marsh CA, As-Sanie S, Smith YR, Quint EH. Prevalence of pain syndromes, mood conditions, and asthma in adolescents and young women with endometriosis. J Pediatr Adolesc Gynecol. 2013 Jun;26(3):171-5. doi: 10.1016/j.jpag.2012.12.006. Epub 2013 Mar 16. PMID: 23507008.

20

2. Pain mechanisms occur along a continuum, with variable degrees of peripheral versus central mechanisms across the population of affected individuals



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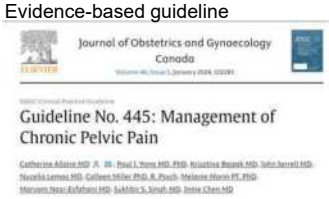
How do you evaluate a patient with CPP?



22

Newest CPP Guidelines

Evidence-based guideline



A summary of CPP Guidelines



JAMA. 2021; 325(23):2381-2391.

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Guideline Components

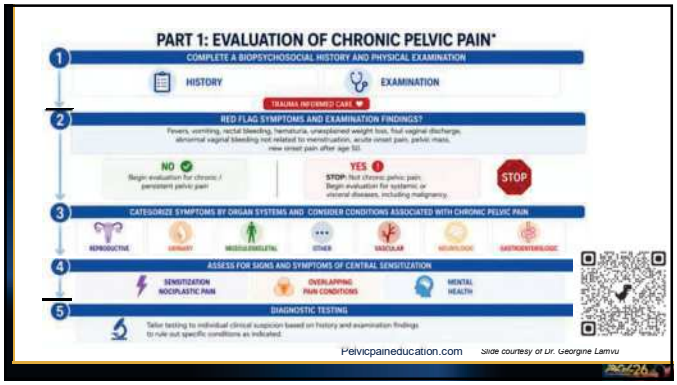
- **BIOPSYCHOSOCIAL HISTORY AND TRAUMA INFORMED CARE** Evaluation guide
- **EXPANDED PHYSICAL EXAMINATION**
- **MULTIMODAL THERAPY**

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BIOPSYCHOSOCIAL HISTORY AND TRAUMA INFORMED CARE

- The **biopsychosocial approach** tries to identify physical, mental, environmental, and traumatic factors that impact pain severity *beyond* peripheral nociceptors.
- **Trauma-informed** care focuses on patient-centered communication, building trust between the patient and the clinician, providing a safe space for the patient to disclose sensitive topics, giving the patient a sense of control, and taking measures to help reduce anxiety about the interaction with the clinician / medical team.

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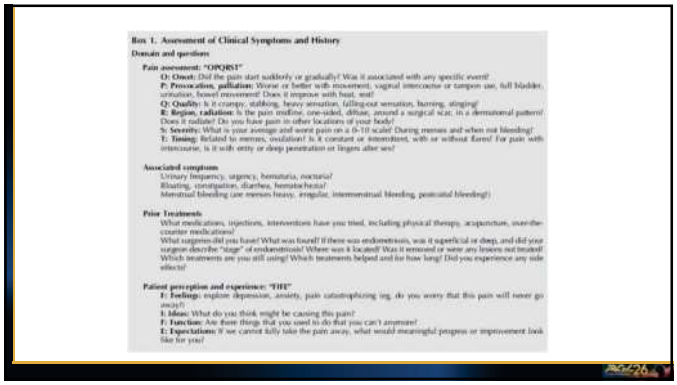
HISTORY

- *Character, duration, location of pain-*
  - dysmenorrhea, non-menstrual pain, dyspareunia
- *Exacerbating & relieving factors*
- *Associated GI or GU symptoms?*
- *Any other chronic pain syndromes?*
- *Symptoms of dysfunction… Mood, sleep, physical function*



http://pelvicpain.org/

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Box 1. Assessment of Clinical Symptoms and History

Domain and questions

Pain assessment: "OPQRST"

O: Onset: Did the pain start suddenly or gradually? Was it associated with any specific event?
P: Provocation, palliation: Worse or better with movement, vaginal intercourse or tampon use, full bladder, urination, bowel movement? Does it improve with heat, rest?
Q: Quality: Is it cramp, stabbing, heavy sensation, falling-out sensation, burning, stinging?
R: Region, radiation: Is the pain midline, one-sided, diffuse, around a surgical scar, in a dermatomal pattern? Does it radiate? Do you have pain in other locations of your body?
S: Severity: What is your average and worst pain on a 0–10 scale? During menses and when not bleeding?
T: Timing: Related to menses, ovulation? Is it constant or intermittent, with or without flares? Is pain with intercourse, is it with entry or deep penetration or fingers after sex?

Associated symptoms

Urinary frequency, urgency, hematuria, nocturia?
Bloating, constipation, diarrhea, hematochezia?
Menstrual bleeding (are menses heavy, irregular, intermenstrual bleeding, postcoital bleeding?)

Prior Treatments

What medications, injections, interventions have you tried, including physical therapy, acupuncture, over-the-counter medications?
What surgeries did you have? What was found? If there was endometriosis, was it superficial or deep, and did your surgeon describe "stage" of endometriosis? Where was it located? Was it removed or were any lesions not treated?
Which treatments are you still using? Which treatments helped and for how long? Did you experience any side effects?

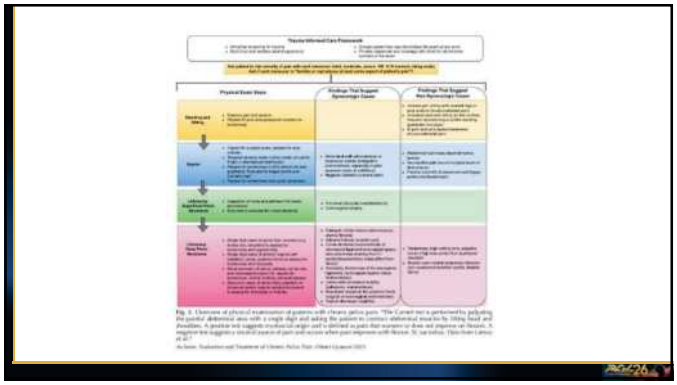
Patient perception and experience: "FIRE"

F: Feelings: explore depression, anxiety, pain catastrophizing (eg, do you worry that this pain will never go away?)
I: Ideas: What do you think might be causing this pain?
F: Function: Are there things that you used to do that you can't anymore?
E: Expectations: If we cannot fully take the pain away, what would meaningful progress or improvement look like for you?

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Step 2: Physical Exam

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Trigger Points

focal, hypersensitive, palpable nodules in the muscle

- *Associated with referred pain, motor dysfunction, autonomic symptoms*
- *Abnormal neuromuscular depolarization*
- *Carnett sign for diagnosis of myofascial pain*
  - Pain with palpation worse during abdominal wall contraction
  - Sensitivity 78%, Specificity 88%, Intraobserver



Srinivasan R, Greenbaum DS. Chronic abdominal wall pain: a

Abdominal/Pelvic Neuralgias

Ilioinguinal, iliohypogastric, pudendal, obturator, genitofemoral neuralgias can all cause pain in the lower abdominal, pelvic, perineal, labial, vaginal regions.

Risk Factors: surgical retractors, low transverse incisions, laparoscopy trocars, mesh implantation, obstetric trauma, spinal injury.



www.aneskey.com

Rehn DR, et al. Am J Obstet Gynecol. 2003

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Scars ….A perfect environment for

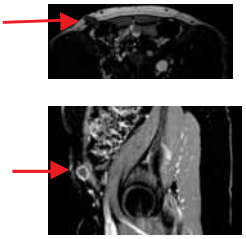
Neuropathies

Endometriomas

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Abdominal wall endometriosis

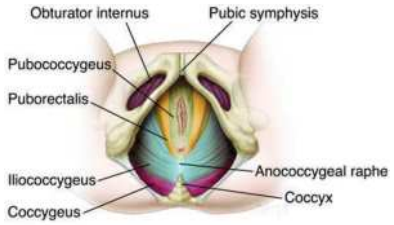




Abdominal wall endometriosis:
- Cyclic abdominal pain
- Tender nodule
- Most common after cesarean section

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Single digit pelvic floor exam



Slide courtesy of Georgine Lamvu, MD MPH

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Pubococcygeus



Coccygeus



Iliococcygeus



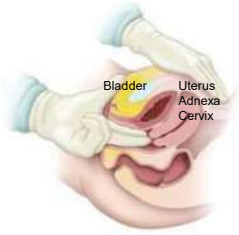
Obturator Internus



- **Single well-lubricated digit, slow insertion**
- **Voluntary contraction, strength and tone**

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Vaginal examination, bi-manual



- Adnexal masses
- Uterine masses
- Rectovaginal nodularity or pain
- Uterosacral nodularity

Slide courtesy of Georgine Lamvu, MD MPH

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Red flag

- Blood in stool
- New bowel symptoms over 50
- Irregular vaginal bleeding over 40
- Post coital bleeding
- New pain after menopause
- Excessive weight loss
- Pelvic mass
- Suicidal ideation
- Change in character or severity of pain in a patient with a history of chronic pain

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Unlocking the Unseen: Understanding the Neurology of Pelvic Pain

Ashley Gubbels, MD FACOG
Cleveland Clinic

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Disclosure

I have no financial relationships to disclose.

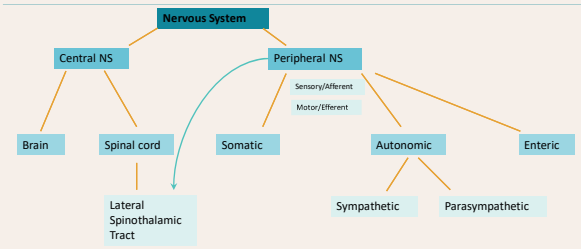
2

Objectives

- Review the types of pain and how to identify neuropathic pain
- Discuss the difference between somatic and visceral pain
- Understand the diagnostic process to determine neurologic pathways involved

3

Nervous system



4

Function of pelvic nerves



Relay sensory information	Regulate pelvic organ function	Coordinate motor activity
Bladder and bowel	Urinary: filling, storage, and voiding	Internal and external urethral and anal sphincters
Genital and perianal skin	Bowel: continence and defecation	Pelvic floor, gluteal, and lower-extremities
Pelvic floor	Sexual: arousal, erection, and orgasm	
Buttocks		
Lower extremities		

5

Types of pain

Multiple ways to classify
- Acute - Chronic
- Nociceptive – Neuropathic – Nociplastic
- Visceral - Somatic



All are incomplete



6

Nociceptive – Neuropathic – Nociplastic



7

Visceral - Somatic

	Visceral Pain	Somatic Pain
Quality	-Vague, poorly localized, dull -Involves entire abdomen/pelvis -Radiation to low back	-Specific/precisely localized -Allodynia -Electrical
Vegetative symptoms	Present -Malaise/Fatigue/Irritability -Pupillary dilation -Inhibited salivation -Tachycardia (autonomic dysfunction) -Nausea/Vomiting -Pallor/diaphoresis -Anxiety	Absent
Radiation	Into low back via hypogastric plexus	Caudally to corresponding dermatome(s)
Motor dysfunction	Pelvic organ dysfunction (hypersensitivity/OAB, IBS)	Pelvic organ dysfunction Sexual dysfunction Ambulatory dysfunction

8

What is neuropathic pain?



- Pain arising as a consequence of a lesion or disease affecting the somatosensory system.

This involves the peripheral nerves, spinal cord, or brain that process pain signals.

- Lesion may be peripheral or central
- May be caused by a disease process, injury, infection, or loss of limb.
- Clinical description, not a diagnosis

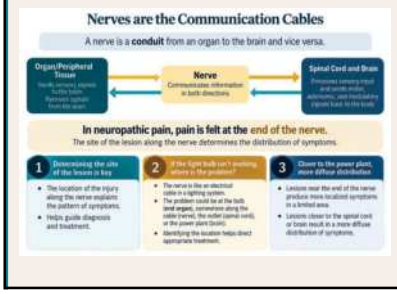
Often Coexists With...

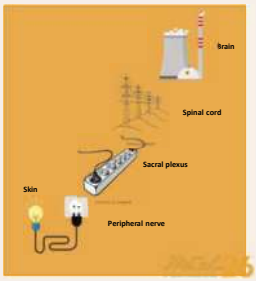
Neuropathic pain frequently coexists with myofascial dysfunction and central sensitization — a multifactorial, biopsychosocial problem.

Psychological factors
Social factors
Lifestyle factors
A BIOPSYCHOSOCIAL PROBLEM

9

Neuropathic pain simplified





Analogy credit to Marc Possover MD

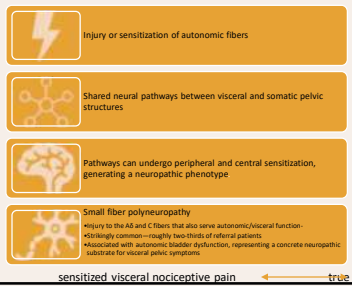
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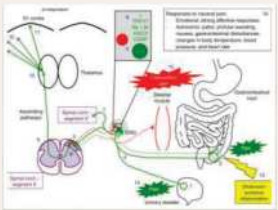
Somatic Neuropathic Pain



11

Visceral "Neuropathic" Pain





sensitized visceral nociceptive pain ⟷ true visceral neuropathic pain

12

Why this matters to surgeons?



- Musculoskeletal/neuropathic contributors
  - found in 50–90% of women with CPP at specialized centers
  - frequently overlooked
- Focusing only on visceral/organic causes leads to prolonged pain, delayed treatment, and **unnecessary surgery**
  - Pelvic/abdominal surgery and mesh are themselves risk factors for iatrogenic neuralgia
  - Surgery is both a cause and a solution

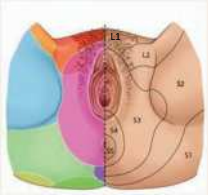
Origin of CPP is not gynecologic in 70-80% of patients.

13

When to suspect a neuralgia

- Pain follows a **dermatome** or specific nerve distribution
- **Allodynia** or **hyperalgesia** on exam
- Descriptors: *burning, pins and needles, razor blades, cutting, hot, raw skin*
- Onset after **trauma, surgery, childbirth, or repetitive activity** (prolonged sitting, long-distance cycling), even remote history

- **Risk factors:**
  - prior pelvic/abdominal surgery, mesh, high-intensity sports/cycling, sedentary/prolonged-sitting occupations, diabetes, herpetic infection



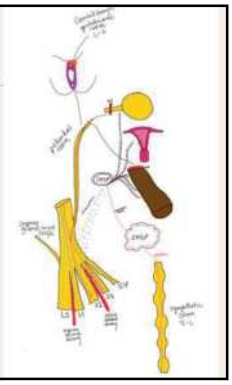
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Neuropelveology: A change in how to think

15

Neuropelveological Algorithm

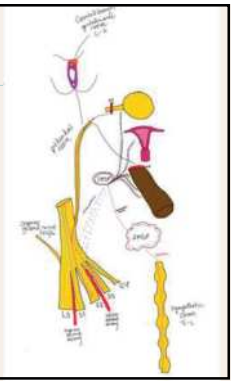
1. Visceral or somatic
2. Nerve pathway(s)
3. Level
4. Etiology
5. Confirmation



16

Neuropelveological Algorithm

1. Visceral or somatic
2. Nerve pathway(s)
3. Level
4. Etiology
5. Confirmation



17

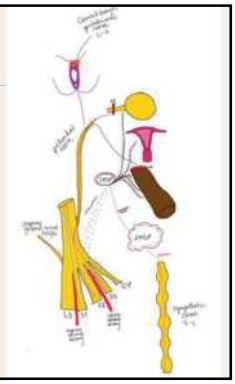
Visceral - Somatic

	Visceral Pain	Somatic Pain
Quality	-Vague, poorly localized, dull -Involves entire abdomen/pelvis -Radiation to low back	-Specific/precisely localized -Allodynia -Electrical
Vegetative symptoms	Present -Malaise/Fatigue/Irritability -Pupillary dilation -Inhibited salivation -Tachycardia (autonomic dysfunction) -Nausea/Vomiting -Pallor/diaphoresis -Anxiety	Absent
Radiation	Into low back via hypogastric plexus	Caudally to corresponding dermatome(s)
Motor dysfunction	Pelvic organ dysfunction (hypersensitivity/OAB, IBS)	Pelvic organ dysfunction Sexual dysfunction Ambulatory dysfunction

18

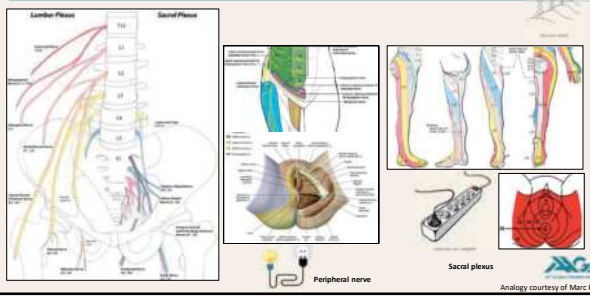
Neuropelveological Algorithm

1. Visceral or somatic
2. Nerve pathway(s)
3. Level
4. Potential etiology
5. Confirmation



19

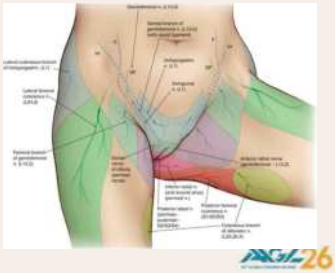
Nerve pathway → Dermatomes



20

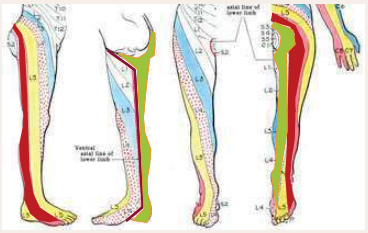
Relevant peripheral nerves

- Iliohypogastric T12-L1
- Ilioinguinal L1
- Genitofemoral L1-2
- Obturator L2-4
- Pudendal S2-4 (Dorsal clitoral, Perineal, Inferior anal)
- Posterior femoral cutaneous S1-3



21

Relevant dermatomes

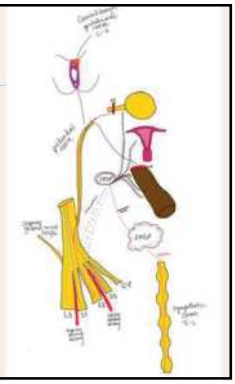




22

Neuropelveological Algorithm

1. Visceral or somatic
2. Nerve pathway(s)
3. Level
4. Etiology
5. Confirmation



23

Level

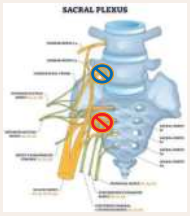
Location of pain	Peripheral Nerve	Plexus
Vulva	Genitofemoral (from anterior) Pudendal (from posterior)	S3-4
Anal	Pudendal	S2-3
Perineal	Pudendal	S2-4
Coccygeal	Coccygeal	S2-4
Gluteal	Cluneal	S1-2
Dorsal sciatica	PFCN (partial)	S1/2
Ventral sciatica		L4-5 (spinal)

- Keys
  - History
  - Palpation
- Confirm
  - via guided nerve block
  - >50% improvement after LA injection

24

Level

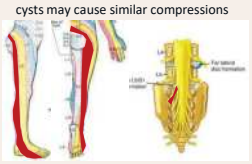
- Level of the problem
  - Bulb vs cord vs surge protector
- Spinal vs pelvic origin
  - Pelvic irritation of sciatic → allodynia of sacral dermatomes only
  - Spinal irritation → combines sciatica with dorsal and ventral leg symptoms L4-5/S1

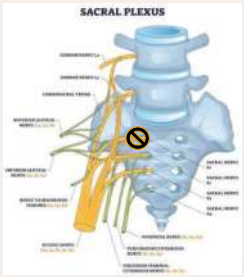


25

Level

- Spinal vs pelvic origin
  - L5/S1 disc herniation
    = S1 radiculopathy
  - No discs in the sacrum, however Tarlov cysts may cause similar compressions



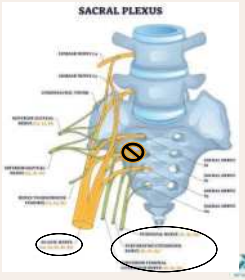


26

Level

- Sacral plexus
- S2 causes buttock, dorsal/medial leg symptoms + genital pain (via pudendal)

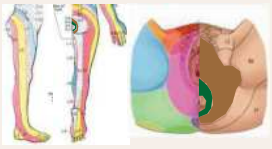


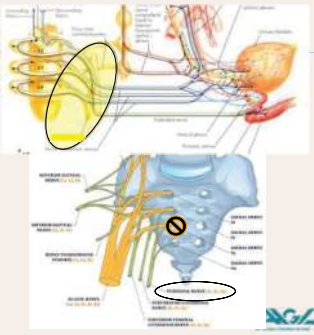


27

Level

- Sacral plexus
- S3/4 causes genital pain only (mostly perineal/vulvar)
- Problems with bladder function
  - Neurogenic vs non-neurogenic



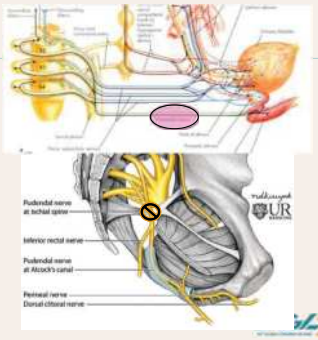


28

Level

- Pudendal neuralgia (Alcock's canal)
- Clitoris, labia minora, perineum, perianal skin without leg symptoms
- Bladder/rectal dysfunction
  - Neurogenic vs non-neurogenic

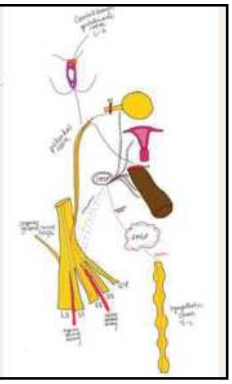




29

Neuropelveological Algorithm

1. Visceral or somatic
2. Nerve pathway(s)
3. Level
4. Etiology
5. Confirmation



30

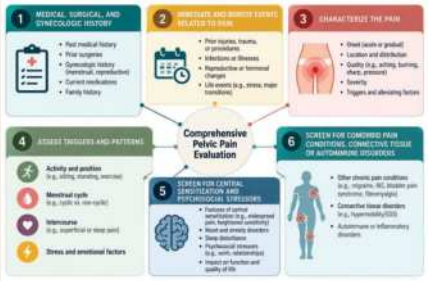
Determine potential etiology

- Detailed history
- Neuropelveologic exam
- Pelvic MR neurogram vs tractography (experimental), evolving use of ultrasound
- Neurogenic vs non-neurogenic

	Nerve functions	Symptoms
Neurogenic	Decreased	Hypo or anesthesia Urinary retention Sexual dysfunction Constipation
Non-neurogenic	Increased	Skin hypersensitivity Bladder hypersensitivity/OAB Parasthesias Phantom pain

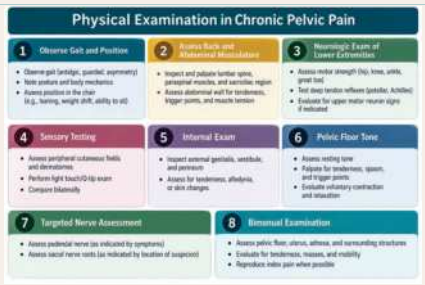
31

History



32

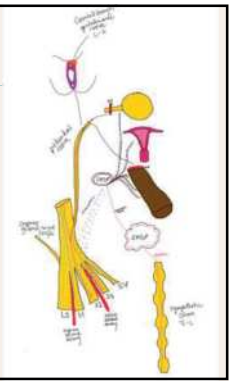
Examination



33

Neuropelveological Algorithm

1. Visceral or somatic
2. Nerve pathway(s)
3. Level
4. Potential etiology
5. Confirmation



34

Confirm and treat

- Accessible nerves
  - Local anesthetic blocks
  - +/- Botox



- Inaccessible nerves
  - Laparoscopic evaluation-nerve release/neurolysis
  - Neuromodulation





35

Objectives

- Review the types of pain and how to identify neuropathic pain
- Discuss the difference between somatic and visceral pain
- Understand the neuropelveologic diagnostic process

36

Acknowledgments

- Drs. Michael Hibner and Mario Castellanos
- Dr. Marc Possover

37

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38

Integrating the **R U MOVVING SOMe** classification into clinical practice to simplify the chronic pelvic pain evaluation.

Prof. **Georgine Lamvu**, MD, MPH
University of Central Florida College of Medicine
Orlando VA Healthcare System

1

Disclosures

- Past President and Past Chair of the Board-International Pelvic Pain Society
- Director of the VA-UCF Certificate Program in Clinical Research
- Director of the National Coordinating Center- VA Fellowship in Minimally Invasive Gynecologic Surgery (MIGS)
- NIH, DoW research funding
- The opinions and statements expressed in this lecture are my own and not the opinion or position of the VA, DoW, or NIH.
- Consultant, SoLa Pelvic Therapy

2

Objectives

1. Apply a trauma-informed, biopsychosocial approach to evaluate patients with chronic pelvic pain and identify red-flag findings requiring evaluation for systemic or visceral disease.
2. Use the FIGO-IPPS **R U MOVVING SOMe** classification system to identify peripheral pain contributors, sensitization/nociplastic pain, chronic overlapping pain conditions, and mental health comorbidities.
3. Develop patient-centered, phenotype-directed multimodal treatment plans using shared decision-making, interdisciplinary care, and scheduled longitudinal follow-up.



3

It is 4:00 PM on a Friday…

The nurse informs you that patient Dolores Smith is here for pelvic pain, and she seems angry and anxious. The nurse also reminds you that the clinic closes in 30 minutes and that the staff will leave at that time.

4



Virtual case

- 32-year-old G3P2, dysmenorrhea progressed to CPP in 5 years, **referred to GYN for CPP.**
- Dyspareunia, urgency, frequency, constipation, pain worsened by activity and exercise
- Fibromyalgia, migraines, low back pain, anxiety, PTSD, magnification
- Cannot sleep, work, or do usual activities
- Excised stage III endometriosis, no additional therapy, improved temporarily after surgery then worsened
- Worried about divorce due to pain
- Frustrated and wants doctors to 'fix' her pain
- Exam: hyperalgesia, TP, myalgia, negative UA and normal ultrasound

5



Questions

- How many of you have seen a similar in your practice?
- How long does it take you to evaluate and select therapies for a patient with similar presentation?
- How do you feel when you realize the Veteran's mental health is affected by chronic pain?

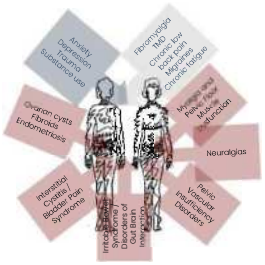
6

High prevalence and negative impact, yet healthcare systems fail CPP patients

Lack of Agreed Definition
There is no agreed upon definition for CPP, with Various societies defining CPP in different ways.

Multifactorial condition
Several conditions involving somatic and visceral structures are associated with CPP, making evaluation and management challenging.

Absence of Classification System
Until recently, there was no classification system to help clinicians, educators, researchers, and patients navigate the challenges of CPP and its contributors.



7

Traditional medical models approach CPP using an organ-based, siloed framework.

However, patients do not experience pain in isolation, and their symptoms rarely conform to these artificial boundaries.

We all need to communicate (and speak the same language) to effectively take care of CPP!

8

A call to action from FIGO and IPPS



Our communities must address 3 problems first

1. Barriers to health-related care
2. Gaps in healthcare education
3. Patient support and advocacy



9

Need for a New Framework

A new classification system was needed to:

- **Empower patients** by providing a framework for understanding their condition.
- **Aid healthcare professionals** in diagnosis and management.
- **Facilitate education** for trainees and patients.
- **Standardize research** and terminology.
- **Inform policymakers** for resource allocation.

10



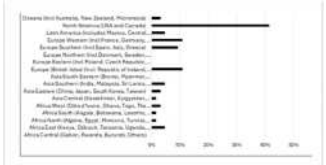
A new definition and classification system for conditions associated with CPP.

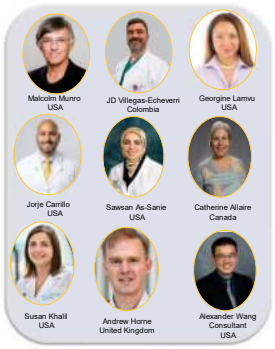
The system is comprised of **12** categories of conditions associated with CPP, arranged in the acronym **R U MOVVING SOME**.

11

FIGO-IPPS CPP Consensus Group

- Approval for the project was obtained from FIGO ECAC and IPPS Executive Board.
- 114 countries invited, 77 stakeholders representing 47 societies, including advocacy groups, persons who live with pain, clinicians, researchers, educators, publishers.
- Delphi methodology to achieve consensus, 84-44% response rate.



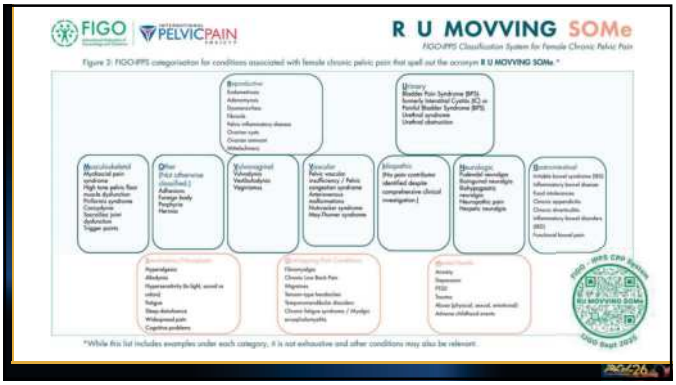


12

FIGO-IPPS consensus definition of CPP

- Pain localized to the pelvis that may be cyclical or non-cyclical and ***typically* lasts 3 months or longer after failed initial therapies.**
- The pain can significantly impact **societies and individuals;** however, on an individual level, the pain may be associated with disability, distress, and negative cognitive, behavioral, sexual, emotional, and **economic consequences.**
- The pain may present with symptoms of musculoskeletal, urinary, bowel, reproductive, **vascular, and neurologic dysfunction, such as** urgency, frequency, abnormal bowel movements, **dysmenorrhea, allodynia, hyperalgesia, vascular insufficiency, and abnormal uterine bleeding.**

13



14

Why the emphasis on SOMe?
The biopsychosocial model of CPP

Chronic pelvic pain involves **interaction between**:

- Peripheral **nociceptive** generators (e.g., endometriosis, bladder pain syndrome, pelvic floor myalgia)
- Central **sensitization**
- **Psychological** amplification (catastrophizing, anxiety, trauma exposure)
- **Behavioral** adaptations (avoidance, guarding, sleep disruption)
- **Social context** (stigma, relationship strain, work disability)

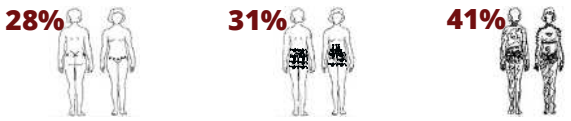
Psychological and behavioral therapies target **cognitive, emotional, and behavioral drivers** of pain-related disability and suffering

15

All three patients depicted below have CPP, yet they are different. Pain mechanisms occur along a continuum, with variable degrees of peripheral versus central mechanisms

Peripheral ⟷ **Nociplastic**

Endometriosis, Purely peripheral	Endometriosis, Mixed	Endometriosis, Purely centralized
28%	31%	41%



Slide courtesy of Dr. Sawsan As-Sanie

16

Key Point #1

If you take care of patients with chronic pain (including pelvic pain) you must become comfortable with phenotyping and adapting patient care to each phenotype (precision medicine).

17

Key Point #2

BPS assessment is essential… even if it takes more time! The BPS model can help determine the phenotype, and the phenotype should be used to guide management.

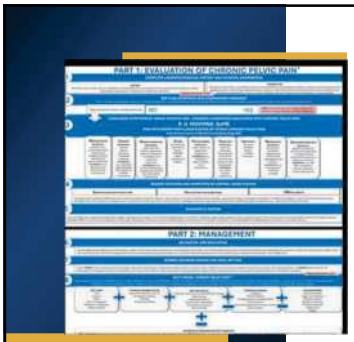
Evidence: Integrated, multidisciplinary approaches improve outcomes in CPP.

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The 30-Second Screening Protocol

- Incorporate these questions into your health history or clinical intake:
  - *"Do you have chronic pain in other areas, like your lower back or pelvis?"*
  - *"Do you have a history of very painful periods, or painful bowel, or painful bladder issues that affect your daily life?"*

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Additional resource to be used with R U MOVVING SOMe

www.pelvicpaineducation.com

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Let's apply these resources to our virtual clinical case.

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Dolores Smith

Virtual Case

- 32-year-old G3P2, dysmenorrhea progressed to CPP in 5 years, **referred to GYN for CPP.**
- Dyspareunia, urgency, frequency, constipation, pain worsened by activity and exercise
- Fibromyalgia, migraines, low back pain, anxiety, PTSD, magnification
- Cannot sleep, work, or do usual activities
- Excised stage III endometriosis, no additional therapy, improved temporarily after surgery then worsened
- Worried about divorce due to pain
- Frustrated and wants doctors to 'fix' her pain
- Exam: hyperalgesia, TP, myalgia, negative UA and normal ultrasound

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1. Phenotype based on history and examination

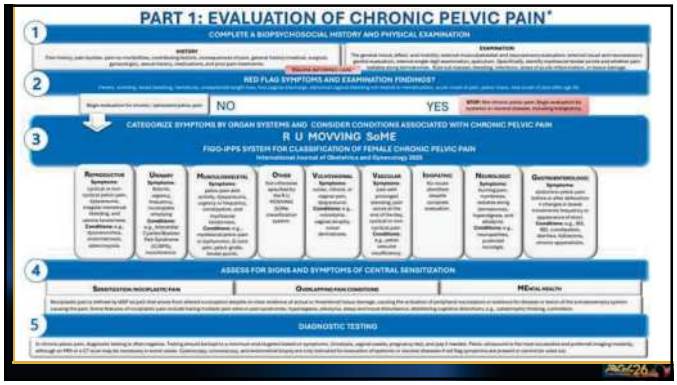
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Pelvic Pain E&M in 5 Efficient Steps

- **Step 1:** Download your CPP Evaluation and Management Guide and R U MOVVING SOMe
- **Step 2:** Take a biopsychosocial history and perform a brief physical exam (use the guide to help you)
- **Step 3:** Categorize the patient's symptoms / conditions/findings using the R U MOVVING SOMe Classification System
- **Step 4:** Educate the participant, establish goals, and select therapies
- **Step 5:** Close the referral loop and schedule follow ups



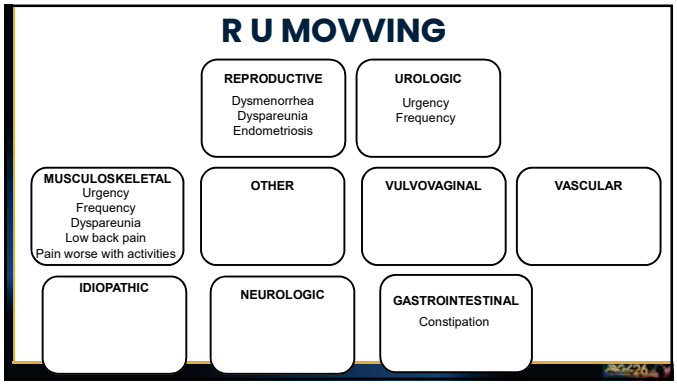
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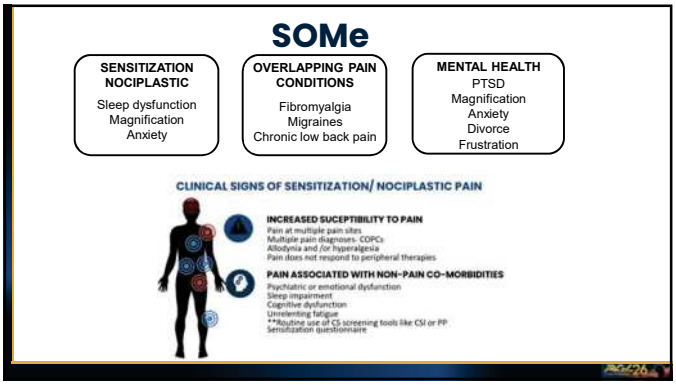
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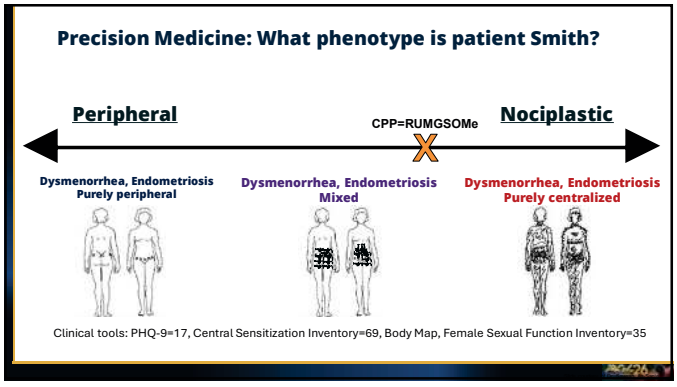
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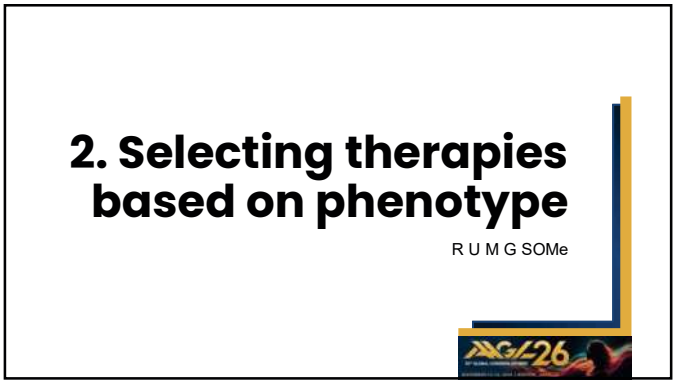
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R U MOVVING SOMe: Conditions to consider based on phenotype R U M G SOMe



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Therapies for phenotype R U MG SOMe



Pharmacotherapy

- Local or peripheral anesthetics (e.g., suzetrigine, blocks), NSAIDS, Alpha-2 adrenergic receptor agonists (e.g., clonidine), Anticonvulsants (not effective in CPP, but effective for neuropathies), Tricyclic antidepressants, SNRIs, Muscle relaxants, Steroids
- Opioids are generally avoided in CPP because they can worsen pain, constipation, PFM dysfunction, sexual dysfunction, and mood instability.

Avoid prn use, wean on/off to manage side effects
Treat constipation
Suppress menstruation if pain is exacerbated by menstruation (even if the pain does not have a gyn etiology).

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The 30-Second Explanation

1. **Acknowledge & Validate:** Living with pain can be exhausting and debilitating, and there are millions of women who suffer from this type of pain.
2. **Explain pain mechanisms:** Often, chronic pain is felt in more than one area and may be due to several conditions because these conditions are actually connected through your central nervous system—the brain, spinal cord, and nerves that process pain signals. The brain is the main organ that controls all bodily functions, including how much pain you feel.
3. **Introduce the expectation for a whole-body therapeutic approach:** When pain becomes persistent and/or it affects multiple areas in the body, we recommend 'multi-modal' treatment, which involves a combination of therapies to care for your entire body, not just a single organ(not just your [ovaries]).
4. **Provide hope for healing:** While we do not yet have a cure, with the right individualized care, persons with chronic pain can get better and get back to living fully. With your input, we will create a therapy plan tailored to your specific needs and goals.

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Key Point #3

CPP is multifactorial, it is best managed by an interdisciplinary biopsychosocial model. Behavioral and mental health are not adjunctive- they are foundational and essential in chronic pain care.

ACOG recommends referral for cognitive behavioral therapy (CBT), pelvic floor physical therapy, and sex therapy, alone or in combination, for myofascial and psychosocial contributors to CPP.



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Follow up



- Establish regular visits until the patient is stable.
- Failing to establish follow up makes patients feel abandoned and unsure.
- Change your language!



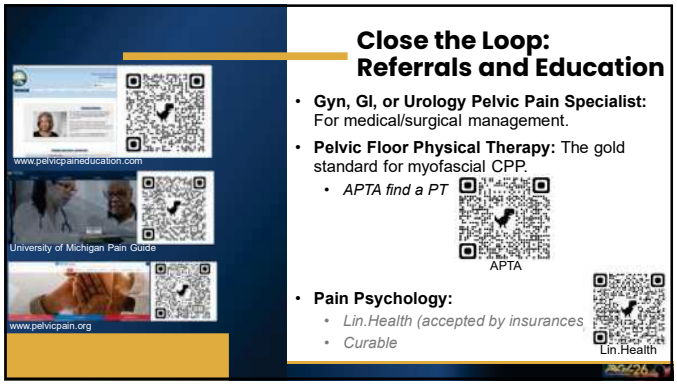
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Key Point #4

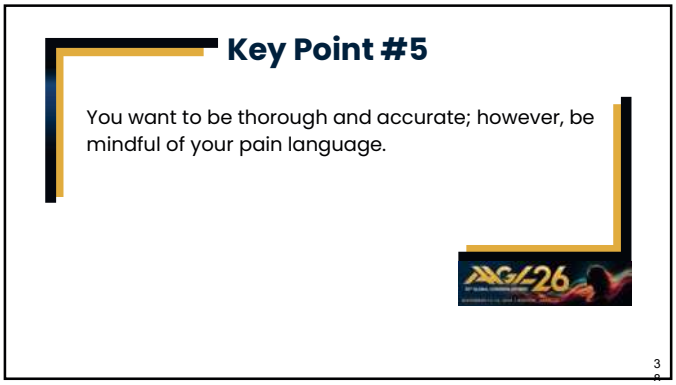
CPP patients should have established long-term care.

Regularly scheduled visits help overcome access issues, reduces anxiety and reassures the patient that they will not be abandoned/dismissed.

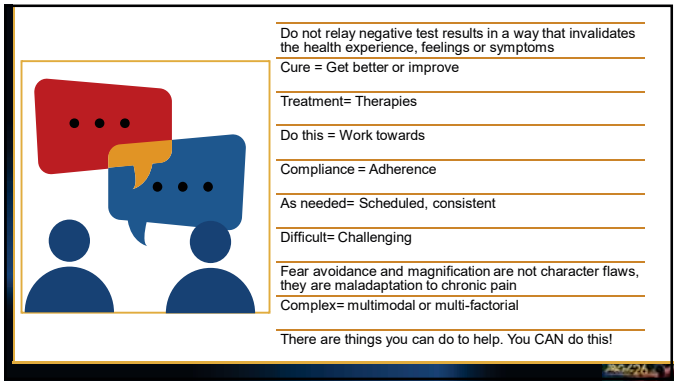
36


Close the Loop:
Referrals and Education
Gyn, GI, or Urology Pelvic Pain Specialist: For medical/surgical management.
Pelvic Floor Physical Therapy: The gold standard for myofascial CPP.
APTA find a PT
APTA
Pain Psychology:
Lin.Health (accepted by insurances
Curable
Lin.Health
www.pelvicpaineducation.com
University of Michigan Pain Guide
www.pelvicpain.org

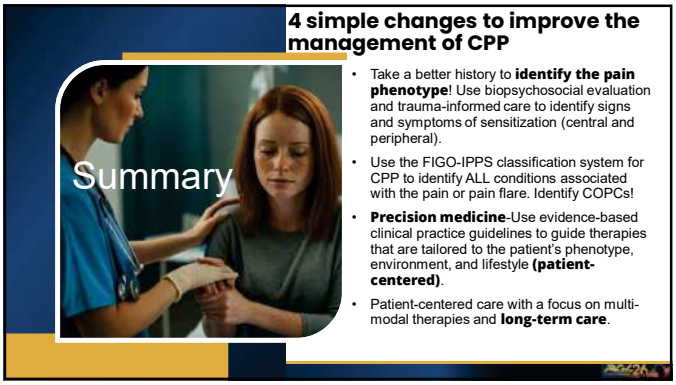
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Key Point #5
You want to be thorough and accurate; however, be mindful of your pain language.

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Do not relay negative test results in a way that invalidates the health experience, feelings or symptoms
Cure = Get better or improve
Treatment= Therapies
Do this = Work towards
Compliance = Adherence
As needed= Scheduled, consistent
Difficult= Challenging
Fear avoidance and magnification are not character flaws, they are maladaptation to chronic pain
Complex= multimodal or multi-factorial
There are things you can do to help. You CAN do this!

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4 simple changes to improve the management of CPP
Summary
Take a better history to identify the pain phenotype! Use biopsychosocial evaluation and trauma-informed care to identify signs and symptoms of sensitization (central and peripheral).
Use the FIGO-IPPS classification system for CPP to identify ALL conditions associated with the pain or pain flare. Identify COPCs!
Precision medicine-Use evidence-based clinical practice guidelines to guide therapies that are tailored to the patient's phenotype, environment, and lifestyle (patient-centered).
Patient-centered care with a focus on multi-modal therapies and long-term care.

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THANK YOU
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Canada
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USA
Andrew Horne
United Kingdom
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USA

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Promising solutions to improving female pelvic pain – envisioning a prevention framework

Frank Tu, MD, MPH · Endeavor Health, Pritzker School of Medicine, University of Chicago

NOVEMBER 13-16, 2026 · BOSTON · #AAGL26

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Disclosures

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- Stock options – Maipl Therapeutics

2

Objectives

- Review risk factors for CPP and CPSP
- Translate the current conceptual framework for A2CPS - a model postsurgical pain study
- Recognize viscero-visceral convergence as a framework for identifying CPP vulnerability and preventing CPP emergence

3

Developmental model of chronic pain

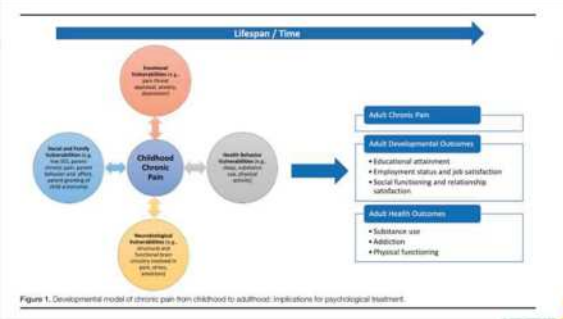


Figure 1. Developmental model of chronic pain from childhood to adulthood: implications for psychological treatment.

Palermo TPAIN 2020

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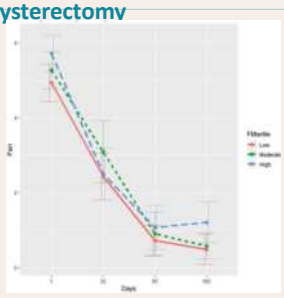
General chronic pain risk factors

modifiable	nonmodifable
Pain	Age
Mental Health	Sex
Smoking	Cultural background
Alcohol	Socioeconomic background
High BMI	History of trauma/injury/interpersonal violence
Physical activity/exercise	Heritable factors (including genetic)
Sleep (anti-inflammatory)	
Nutrition	
Employment status/occupational factors	
Mental Health	

Blyth FM et al. PAIN 2007

5

Centralized pain profile and Persistent post-surgical pain/ hysterectomy



- 176 participants with preop pelvic pain
- 15/126 persistent (>50% of baseline pelvic pain at 6 mths)

- Centralized pain score (p=0.01), endometriosis (p=0.05) & fibroids (p=0.03) associated with risk
- Unrelated – surgical route, adhesions, weight, adenomyosis, depression anxiety
- **Multivariate logistic regression ACR FM score - OR 1.27, 95%CI 1.03, 1.57) 6-months after surgery.**

As-Sanie S et al AJOG 2021

6

Dysmenorrhea/CPP related risks

- Genetic expression/regulation (ZMIZ1, NGF)[1]
- Menstrual pain foci[2]
  - viscero-visceral & viscero-somatic convergence
- Prior PID (I)
- Prior pelvic surgery (I)
- Pelvic sensory processing issues, Visceral hypersensitivity (II)
- Autoimmune disorder (II)

[1]Jones AV et al. Pain. 2016; Li Z et al. *Nat Commun* 2017;
[2]Giamberardino M et al. Pain 2010

7

1 yr f/u incident widespread pain

Table 4. Predictors of new chronic widespread pain at follow-up*

Parameter, score range	n	No. with new CWP	Univariate model OR†	95% CI	Multivariate model OR†	95% CI
Illness Behavior						
0–4	450	6	1	Referent	1	Referent
5–7	546	30	4.2	1.7–10.1	4.3	1.8–10.7
8–24	408	45	8.7	3.6–20.7	9.0	3.7–22.2
Somatic Symptoms						
0–2	1,353	72	1	Referent	1	Referent
3–5	51	9	3.8	1.7–8.2	3.3	1.5–7.4
GHQ						
0	712	39	1	Referent	1	Referent
1	158	13	1.6	0.8–3.0	1.0	0.5–2.1
2–5	264	11	0.8	0.4–1.5	0.5	0.2–1.0
6–12	109	18	2.0	1.1–3.7	0.9	0.4–2.0
Fatigue						
0	847	39	1	Referent	1	Referent
1–2	188	16	1.8	1.0–3.2	1.7	0.9–3.3
3–5	156	15	2.1	1.1–3.9	1.7	0.8–3.4
6–11	130	11	1.8	0.9–3.6	1.1	0.5–2.5
Health Anxiety						
0–5	520	25	1	Referent	1	Referent
6–11	469	26	1.1	0.6–2.0	0.8	0.5–1.5
12–44	415	30	1.5	0.8–2.5	0.9	0.5–1.7

* 95% CI = 95% confidence interval; GHQ = General Health Questionnaire.
† Odds ratio (OR) of having new chronic widespread pain (CWP) by scale score, age and sex adjusted.

Mail survey –

- Scotland (75% response rate)
- N= 1658 (no CWP baseline)
- (1404 returned)
- 18 -65 yr olds
- 5.8% new onset

McBeth J et al Arth Rheum 2001

8

CPSP major problem

- ~40 million people undergo surgery annually.
- 10% develop chronic postsurgical pain (CPSP)
- 1% severe CPSP QOL impacting
- Neuropathic component in 35 - 57% of the patients enduring CPSP

Accessed 10/9/26 https://www.iasp-pain.org/resources/fact-sheets/prevention-of-chronic-post-surgical-pain/

9

CPSP risk factors

Multiple biopsychosocial domains

- multiple patient reported outcome measures
  - pain intensity, sleep dysfunction, and disability
- psychosocial factors
  - anxiety, depression, resilience, social support, childhood and adult trauma
- neuroimaging signals
- QST
- molecular changes in genes, proteins, extracellular RNA, lipids, and metabolites

Berardi G, et al. Front. Med 2022

10

A2CPS Profile

- Examine whether putative biomarkers across multiple domains individually predict susceptibility or resilience to development of chronic pain 6 mths after surgery
- Develop biosignatures using the candidate biomarkers to determine if combinations of biomarkers improve prediction from acute to chronic pain
- Define novel putative biomarkers and biosignatures across multiple domains that predict susceptibility and resilience to development of chronic pain at 6 mths

2285 participants followed 0 – 12 mths post procedure
200 investigators!

A) Total knee arthroplasty
B) Thoracic surgery

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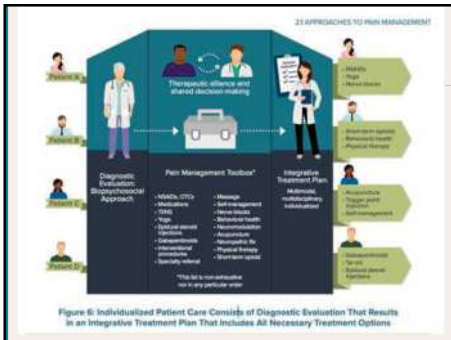


Figure 6: Individualized Patient Care Consists of Diagnostic Evaluation That Results in an Integrative Treatment Plan That Includes All Necessary Treatment Options

U.S. Department of HHS (2019, May). Pain Management Best Practices Inter-Agency Task Force Report: Updates, Gaps, Inconsistencies, and Recommendations. Retrieved from U. S. Department of Health and Human Services website: https://www.hhs.gov/ash/advisory-committees/pain/reports/index.html

- Reducing preoperative pain
- Limiting opioids (Nav1.8 receptor blockers)
- Positive psychology/Pain reprocessing therapy
- HRV (parasympathetic) coherence training
- Vagal nerve stimulators
- Prehab for sleep, exercise, catastrophizing risks
- Transitional pain service philosophy

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Slide poll on dysmenorrhea definitions

13

Pain prevention requires a risk phenotype

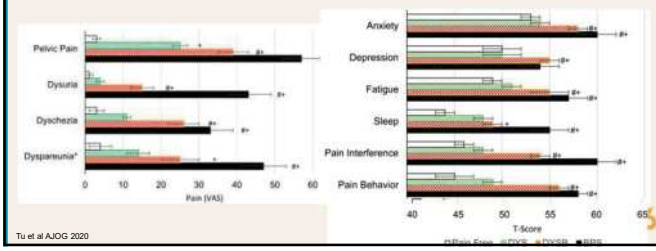
CRAMPP (R01 DK100368): goals

- Confirm presence of silent bladder hypersensitivity (DYSB)
- Does DYSB exhibit neurophysiological features similar to established CPP?
- Determine how much repeated pain experience, vs. circulating sex hormone levels influence descending modulation, and bladder pain sensitivity

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Aim 1: Worse symptoms for DYS-B -> MMH

	HC	DYS	DYSB	BPS	P value
n	29	40	37	18	
Menstrual Pain 0-100 VAS	10.0 [4.0, 22.0]	73.0 [65.0, 81.2]*	70.0 [64.5, 85.0]*	72.0 [60.0, 98.0]*	<0.001

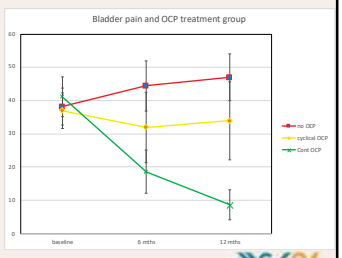


Tu et al AJOG 2020

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Aim 2: OCP use reduces bladder hypersensitivity

	baseline	6 mths	12 mths
no OCP (n=6)	38.2 (13.5)	44.5 (18.5)	47.0 (17.3)
cyclical OCP (n=4)	37.0 (10.7)	32.0 (21.2)	34.0 (23.5)
cont OCP (n=8->5)	41.2 (16.9)	18.6 (14.7)	8.6 (10.0)

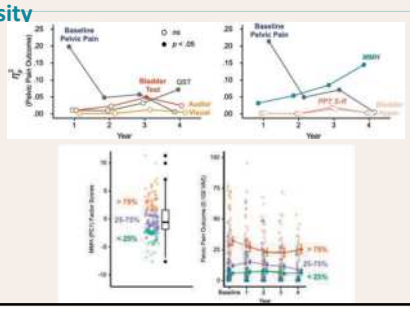


mixed model p = 0.02

Unpublished data

16

Composite sensory sensitivity and future pelvic pain intensity



Kmiecik M et al PAIN 2023

17

Future directions, limitations

- Only subgroups of dysmenorrhea exhibit central sensitization
- Cross organ sensitization – implications?
- Relative single measure vs. composite measure (MMH) improve on modest explanatory power for prediction?
- Underlying MMH cortical mechanisms? Modifiability?
- How to integrate all these central and peripheral contributors?

18

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- Lynn Walker - Vanderbilt
- Amy Bohnert (Loyola Univ - Chicago)
- See Wan Tham (U Washington)



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CULTURAL AND LINGUISTIC COMPETENCY & IMPLICIT BIAS

The California Medical Association (CMA) outlines 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 which began on 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)
Implicit Bias, Microaggressions, and Stereotypes Resources | NEA
Act, Communicating, Implicit Bias (racialequitytools.org)
https://www.contemporaryobgyn.net/view/overcoming-racism-and-unconscious-bias-in-ob-gyn
https://pubmed.ncbi.nlm.nih.gov/34016820/
https://hr.ucdavis.edu/departments/learning/certificate/implicit-bias
https://pmc.ncbi.nlm.nih.gov/articles/PMC7011228/