Chronic Pelvic Pain

An Overview for Physical Therapists

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Disclosures

Proctor and lecturer for Intuitive Surgical
My Background

- Northwestern University Med School 1990
- Residency Bethesda Naval Hospital 1994
- Military Service Guam / Pensacola FL 1994-98
- Private Practice Kailua HI 1998-2006
  - Urogyn and general GYN
- Worked with Dr. David Redwine 2006-2008
  - Laparoscopic Endometriosis Excision
- Franciscan Medical Group 2008-2016
- Pacific Endometriosis and Pelvic Surgery starting 8/2016
  - Specializing in Endometriosis excision, pelvic pain, and Urogynecology
Pain Physiology
I am not a neurosurgeon

I am not a neurophysiologist

I am not a PT (although I try to think like one!)
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I am: A surgeon who is trying to understand what’s going on in my patients...
Pain Physiology

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Why do some have lots of pain with minimal disease?
Why do some have tons of disease and minimal pain?
Why do so many endo patients also have PFD and IC?
Why do some people get sensitized and some don’t?
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Why do some have lots of pain with minimal disease?
Why do some have tons of disease and minimal pain?
Why do so many endo patients also have PFD and IC?
Why do some people get sensitized and some don’t?

Why can I fix some people and not others????
My Background- Not the Academic One

Broken right Tib/Fib age 11
Sprained right ankle (lateral) numerous times age 16-40
Right shoulder dislocation age 22
Bulging discs C5-7 age 35-54
Left shoulder rotator cuff partial tear age 37
Right ankle surgery age 40
Labral tear and FAI right hip - arthroscopy age 50
Resprain right ankle (medial) with new onset plantar fasciitis age 52
My Background- Not the Academic One

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Resprain right ankle (medial) with new onset plantar fasciitis age 52

Why am I able to take only NSAIDS for pain, and can work and play sports given all these injuries when some people would be disabled, have multiple surgeries including fusions and joint replacements, and likely be on narcotics?
Pain Physiology
Pain Physiology

Pain hypersensitivity mechanisms at a glance

Vijayan Gangadharan and Rohini Kuner

Main receptors
- TRPV1
- TRPV2
- TRPV3
- TRPA1
- ASIC
- P2X3
- GPCR
- AMPA
- NMDA

Dorsal root ganglion

Spinal cord
- Main signals: Glutamate & GABA
- CGRP & Glu
- End & IP3

Peripheral sensitivities at nociceptors
- Nerve stimuli on mechanoreceptors, chemoreceptors, or proprioceptors.
- Primary afferent fibers.

Injured or inflamed tissue

Spinal dorsal horn
- Mechanoreceptors
- Afferents from nociceptors
- Pain transmission
- Antinociceptive mechanisms

Cortical sensitization of synaptic synapses
- Enhanced synaptic plasticity of nociceptors and coexpression of neuropeptides in the same afferent terminals.
- Re-expression of neuropeptides.

Recurrent nociception, excitatory postsynaptic potentials (EPSPs) in the spinal dorsal horn.
- Inflammatory mediators.
- Nociceptive neurotransmitters.
- Chemical transmitters.

Neural-glial interactions
- Activation of microglia and astrocytes in the spinal cord.
- Neuropeptides.
- Glutamate.
Pain Physiology

DMM Disease Models & Mechanisms

Main receptors

- TRPV1
- TRPV2
- TRPV3
- TRPV4
- TRPV5
- TRPV6
- TRPML1

Plasticity in nociceptor senses
- Sensitization of different types of primary afferent neurons
- Increased action potential firing
- Decreased threshold for sensory perception

Astrocyte

- ATP
- Mg
- P2X
- TRPV1
- NTX
- NMDAR
- AMPAR
- NMDA
- Kainate
- Sub P
- CGRP
- GLT-1
- GLAST

Dorsal root ganglion

- Microglial cell
- NMDA
- mGluR
- AMPA
- TRPV1
- PKA
- GABA, GABA, GABA

Plasticity of modulations
- Increased release of neuropeptides
- Increased firing of afferent nerve fibers

Injured or inflamed tissue

- Neurons stimulated or injured
- Release of neuropeptides
- Activation of inflammatory mediators
- Increased sensitivity to pain

Peripheral sensitization at nociceptors
- TRPV1, TRPV2, TRPV3
- PKA, PKC, PKD, PKC
- NMDA, AMPA, GluA
- mGluR, AMPA, GluA
- Inflammatory mediators
- Increase in sensitivity to pain

Pain projection neuron

- ERK, JNK
- NFκB
- Ins(1,4,5)P3
- PI3K
- Ca2+
- mGluR
- NMDAR
- AMPA, GluA

Cortical processing

- Pain processing in the cortex
- Involvement of different regions
- Integration of sensory information

Sensory transmission
- Nociceptive signals
- Ascending pathways to the brain

Nature Reviews Neuroscience
Pain Physiology

**Pain processing pathways**

**a Ascending**
- Somatosory Cortex
- Anterior Cingulate Cortex
- Somatosory Cortex
- Thalamus

**b Descending**
- Amygdala
- PAG

**Midbrain**
- Dorsal column nuclei

**Medulla**
- Spinothalamic tract
- Aα or Aβ fibers

**Spinal Cord**
- Injury
- Primary afferent nociceptors
- C or Aδ fibers
- Dorsal Horn
- Dorsal Root Ganglion
- RVM
- Visceral pain, nociceptive pain, cognitive modulation
- Endogenous opioids, endorphins
- 

**Key Points**
- Pain processing involves multiple pathways.
- The ascending pathway includes somatosory and anterior cingulate cortex.
- The descending pathway modulates pain through endogenous opioids.

**References**
- For detailed information, refer to the cited sources.
Pain Physiology

So you can explain it to your patients!
Important Concepts in Pain Processing

- Fear-Avoidance behavior
- Pain Catastrophizing
- Central Sensitization
- Pelvic Crosstalk
- Viscero-Somatic and Viscero-Visceral Convergence
- Neurogenic Inflammation
“We believe pain arises in the nervous system but represents a complex and evolving interplay of biological, behavioral, environmental, and societal factors that go beyond simple explanation”

Edvard Munch

www.iom.edu/Reports.aspx
Basic Pain Processing - Lorimer Moseley

FIGURE 1: THE PAIN PATHWAY

The Pain Pathway

1. Site of injury
2. Spinal cord
3. Brainstem
4. Cerebrum

Basic Pain Processing

A) Reticular formation
   affects consciousness; mild pain increases concentration, severe pain causes unconsciousness

B) Medulla oblongata
   stimulates the respiratory and cardiovascular centre

C) Thalamus
   Relay station: distribution of signals to various areas of the brain, including transmission to the cerebral cortex

D) Hypothalamus and pituitary gland
   Endocrine or hormonal response (e.g. release of beta-endorphin)

E) Limbic system
   regulates the pain threshold and emotional reactions

F) Cerebral cortex
   perception of pain
Variability in Pain Perception

- Sources of variability in reporting pain
  - Sex
  - Age
  - Genetics
  - Psychological or psychosocial factors
  - Nervous system processing
Impact of Psychological Factors in the Experience of Pain

Acceptance and Commitment
Misdirected Problem Solving
Self Efficacy
Stress Diathesis
Fear Avoidance
# Impact of Psychological Factors in the Experience of Pain

<table>
<thead>
<tr>
<th>Theory</th>
<th>Description</th>
<th>Psychological Processes Featured</th>
<th>Mechanism</th>
<th>Examples of Treatment Intervention Strategies</th>
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| **Fear-avoidance model**   | A painful injury may result in catastrophizing and fear, which lead to avoidance of certain movements. This behavior, in turn, leads to more avoidance, dysfunction, depression, and ultimately more pain. | ● Cognitive interpretation featuring catastrophizing  
   ● Emotions: fear, worry, and depression  
   ● Attention: fear keys attention on internal stimuli (hypervigilance)  
   ● Behavior: avoidance of movement | Activity avoidance leads to physical degeneration and social isolation; vicious circle | Promote physical and social activation (e.g., with graded activity) |
| **Acceptance and commitment model** | Rigid beliefs (e.g., that the pain must be cured) may block the pursuit of long-term life goals. Reducing futile attempts to achieve unrealistic goals (acceptance) produces flexibility and engagement in pursuing important life goals (commitment). | ● Cognitive: flexibility in beliefs, life goals, and commitment  
   ● Emotions: anger and frustration  
   ● Behavior: commitment, pursuing goals | Repeated (futile) attempts to control or alleviate pain lead to frustration | Provide realistic treatment goals and encourage client participation in decision making |
| **Misdirected problem-solving model** | Normal worry about pain may tune the patient into certain ways of solving this problem (e.g., medical cures). When this does not actually solve the problem (e.g., with chronic pain or certain forms of musculoskeletal pain), it results in more worry and an even narrower view of the nature of the problem, making it less likely to actually solve the problem. | ● Emotions: worry as a driving force  
   ● Attention: pain demands attention  
   ● Cognitions: beliefs about cause of pain  
   ● Behavior: attempts to solve problem | Hypervigilance to pain symptoms contributes to rumination and failed attempts to escape pain; vicious circle | Redirect problem-solving efforts toward achievement of functional goals |
| **Self-efficacy model**     | The belief that a person is capable of coping with pain is directly related to self-management; low self-efficacy, with feelings that the pain is uncontrollable cause physical and psychological dysfunction. | ● Cognitive interpretation: beliefs concerning controllability of pain  
   ● Behavior: coping skills | Fluctuating pain reduces perceptions of control and mastery over pain | Encourage self-care and self-management strategies, reduce dependence |
| **Stress-diathesis model**  | Significant psychological stress and limited coping resources predispose a person to pain and being less prepared to deal with it. Thus, pain is more likely to result in functional difficulties and emotional distress. | ● Emotions: stress, depression, and anxiety  
   ● Behavior: coping strategies and skills | Protective psychosocial factors buffer the emotional impact of pain, whereas distress and emotional dysregulation predispose to pain | Improve stress management skills and social support |
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**Detrimental**
Fear Avoidance Model

Attention- fear keys attention to internal stimuli (hyper-vigilance)
Cognition- pain catastrophizing
Emotion- fear, depression, and anxiety
Behavior- activity avoidance is the hallmark of the model

Linton and Shaw 2011
Placebo and Nocebo Effects

**Placebo** - larger response in patients with a positive emotional state

Activated opioid and dopamine neurotransmission in brain regions associated with pain, reward, and affect
Enhanced activity in classical descending pain inhibitory pathways - i.e. 
  rACC-amygdala-PAG-rostroventral medulla-spinal cord connection,
Reduced neural activity in the spinal cord during placebo analgesia in nociceptive pain

**Nocebo** - Fear of pain predicted most significant hyperalgesic response
Biochemically opposite responses from placebo analgesia

Pain Catastrophizing

Totally Illogical Leap

Point A
- Man, I got C on my exam
- I could lose my scholarship...

Point B
- Then that would mean I'll have to take out more loans
- ZOMG!! The world is over!!

Semi-logical Links
Pain Catastrophizing
### Pain Catastrophizing Scale

<table>
<thead>
<tr>
<th>Statement</th>
<th>Not at all</th>
<th>To a slight degree</th>
<th>To a moderate degree</th>
<th>To a great degree</th>
<th>All the time</th>
</tr>
</thead>
<tbody>
<tr>
<td>I worry all the time about whether the pain will end</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>I feel I can’t go on</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>It’s terrible and I think it’s never going to get any better</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>It’s awful and I feel that it overwhelms me</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>I feel I can’t stand it anymore</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>I become afraid that the pain will get worse</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>I keep thinking of other painful events</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>I anxiously want the pain to go away</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>I can’t seem to keep it out of my mind</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>I keep thinking about how much it hurts</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>I keep thinking about how badly I want the pain to stop</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>There’s nothing I can do to reduce the intensity of the pain</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>I wonder whether something serious may happen</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
</tr>
</tbody>
</table>
Catastrophizing: a predictor of persistent pain among women with endometriosis at 1 year

115 women with pelvic pain and surgically diagnosed endo
McGill pain score, Pain catastrophizing scale, other psychometric and QOL tests
Followed over 1 year
Multiple interventions including medications, surgery, PT
— No stratification for excision vs ablation, hysterectomy, BSO, etc

Figure 1 — Differences between baseline and 1-year McGill total pelvic pain within catastrophizing groups (P-values are from paired Student's t-tests between baseline and 1-year McGill total pelvic pain scores within each catastrophizing group).

Catastrophizing - fMRI imaging

29 patients with fibromyalgia
Administered pain catastrophizing questionnaire
fMRI while blunt pressure stimulus applied
Controlled for depression

“Pain catastrophizing, independent of the influence of depression, is significantly associated with increased activity in brain areas related to anticipation of pain (medial frontal cortex, cerebellum), attention to pain (dorsal ACC, dorsolateral prefrontal cortex), emotional aspects of pain (claustrum, closely connected to amygdala) and motor control.”

“Catastrophizing influences pain perception through altering attention and anticipation, and heightening emotional responses to pain.”

Central Sensitization

**Definition:** increased responsiveness of the central nervous system to a variety of stimuli, e.g., pressure, temperature, light, and medication.

**Hyperalgesia and allodynia** — expressions of disturbed pain processing in the central nervous system or central sensitization (CS).

**Temporal summation or wind up** — dysfunctional descending inhibitory pathways, and increased facilitatory modulation are possible mechanisms contributing to sensitization of the central nervous system.

**Central hyperexcitability** — causes hyperalgesia, allodynia, and referred pain across multiple spinal segments, leading to chronic widespread pain.

Cagnie, et al. Seminars in Arthritis and Rheumatism 44 (2014) 68-75
Mechanisms of Central Sensitization

1. Increased synaptic excitation - Inflammatory mediators lower nociceptor activation thresholds
2. Temporal Summation
   Identical stimuli become increasingly painful
3. Expansion of receptive fields
   Within dorsal horn of spinal cord substance P sensitizes additional neurons surrounding the original stimulus
4. Crosstalk
   Viscero-Visceral, Viscero-Somatic, and Somato-Visceral convergence
5. Impaired Inhibition of nociceptive stimuli
   DNIC absent in patients with TMJ and IBS

Central and Peripheral sensitization

### Table

<table>
<thead>
<tr>
<th>External stimulus</th>
<th>Peripheral sensory pathways</th>
<th>Central sensory pathways</th>
<th>Sensation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Innocuous stimulus</td>
<td>Sensitized terminal</td>
<td></td>
<td>Primary alldynia</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Primary hyperalgesia</td>
</tr>
<tr>
<td>Noxious stimulus</td>
<td>Nociceptor</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
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### Diagram

- **Peripherally Sensitized Nociceptor**: When an innocuous or noxious stimulus is applied, the nociceptor is sensitized, leading to primary alldynia or primary hyperalgesia.
- **Central Sensitization**: Following sensitization of the nociceptor, a low-threshold neuron is activated leading to secondary alldynia or secondary hyperalgesia.
- **Ectopic Activity**: In the absence of a peripheral stimulus, ectopic activity in the low-threshold neuron can lead to paresthesia or dysesthesia.
- **Spontaneous Pain**: Without a peripheral stimulus, spontaneous pain can be experienced due to sensitization of the nociceptor.

Can We Reverse Central Sensitization?

Possibly....

Study from Shanghai
100 patients with endometriosis surgically treated with excision and 3 months of postop GnRHa
70 controls with no pain and no history of endometriosis
Measured Central Sensitization with ischemic and electrical pain tests, as well as dysmenorrhea preop and at 3 and 6 months postop

Dysmenorrhea, IPT, and generalized hyperalgesia decreased in cases
Pain Threshold normalized postop

He, et al Reproductive Sciences 17(12) 1099-1111
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Pathway of Pelvic Crosstalk

or

Viscero-Visceral / Viscero-Somatic convergence

Can occur in DRG, Spinal cord, Brainstem and Thalamus

Multiple studies have shown increased excitability and sensitization of spinal neurons in response to induced inflammation of the colon which subsequently results in hyperalgesia in the bladder

Mechanism of crosstalk thought to be from neuronal cell bodies sprouting multiple and branching axons

3-10% of DRG neurons have multiple axons
15% of DRG neurons from distal colon and bladder have multiple axons, mostly in T12-L2 and L5-S2 segments

Convergent DRG neurons found in the following structures:
- Colon, bladder, and reproductive organs
- Lumbar muscle and knee
- Lumbar disc and groin skin
- Lumbar facet joint and sciatic nerve
- Intercostal and splanchnic nerves
- Heart and left arm

Most Valuable Reference Papers on Pain Processing

Steven J. Linton, and William S. Shaw  PHYS THER 2011;91:700-711

Central and Peripheral Pain Generators in Women with Chronic Pelvic Pain: Patient Centered Assessment and Treatment
Hoffman, D. Current Rheumatology Reviews, 2015, 11, 146-166

Neural Mechanisms of Pelvic Organ Cross-sensitization
Malykhina (2009) Neuroscience 149:660-672

A Life of Pelvic Pain
Common Causes of Pelvic Pain

- Endometriosis
- Interstitial Cystitis
- Levator Myalgia
- Adenomyosis
- Pudendal Neuralgia
- Vulvodynia
- Orthopedic (Labral Tears, FAI, etc)
Pelvic Pain - what is it?

- OB/GYN — Ruptured Cyst, PID
- Urologist — UTI, IC
- Gastroenterologist — IBS
Pelvic Pain— what is it?

Pelvic Peritoneum
Adhesions (intraperitoneal and fascial)
Pelvic Floor Muscles
External Pelvic Muscles
Nerves
Fascia
Ligaments and tendons

Physical Therapist—
Endometriosis
Endometriosis

**Definition:**
Presence of endometrial glands and stroma outside the uterine cavity

**Most common locations:**
- Uterosacral ligaments, culdesac, ovarian fossae, bladder flap
- Intestinal endometriosis: rectosigmoid, appendix, terminal ileum.
  - Frequently seen with endometriomas.

**Classic Symptoms:**
- Sharp stabbing or burning pain - may be lateral or central
- Initially premenstrual, progresses to all month
- +/- Dyspareunia
- Nausea
- Dyschezia
Endometriosis

**Diagnosis:**

**History:**
- Typically sharp pain, starts days to weeks before menses, improves or resolves mid-late menses
- Often dysmenorrhea as teenager
- Dyspareunia
- Many women can differentiate between sharper endo pain and uterine pain

**Physical Exam:**
- Abdomen- often normal. Tenderness RLQ, LLQ, SP
- Pelvic- Use 1 finger! Palpate levators, bladder, culdesac, uterosacralis, uterus
  - Use small Pederson speculum atraumatically

**Adjuncts:** Ultrasound can detect endometriomas, rectal nodules
- CT not useful, MRI can detect bowel nodules but **Very** dependent on rads
Endometriosis

Staging:
Devised by ASRM for infertility
- Stage I - superficial peritoneal disease
- Stage II - deeply infiltrating but no adhesions
- Stage III - Endometrioma
- Stage IV - Obliterated CDS or bowel involvement

Pain does not correlate with stage -
Pain and Disease run in parallel
Endometriosis

Peritoneal
Less correlation with pain location
Sometimes asymptomatic
Tends to occur in younger patients—especially those with significant central sensitization and Catastrophizing

Deeply Infiltrating
Good correlation between location of pain and disease.
Amount of pain depends on individuals pain sensitivity.
Endometriomas indicate higher likelihood of bowel disease
Endometriosis

**Treatment:**

**Hormonal Suppression**
- OCPs, Cyclic Progestins - Successful 80%
- Mirena IUD
- GnRH agonists - No better efficacy than progestins, but significant sometimes permanent side effects, costly
- Aromatase inhibitors - Similar to GnRH agonists but need ovarian suppression

**Surgery**
- **Excision** - Removes disease
- **Ablation** - Burns top layer with cautery or laser
Normal Pelvis
Endometriosis Stage 2
Post Excision
Endometriosis
Endometriosis Stage 4
Post Excision Stage 4
2 Years Post Excision
Endometriosis / Adenomyosis

- Uterosacral Ligament
- Evil Uterus
Adenomyosis

Endometriosis of the myometrium

Classic Symptoms:
- Central crampy pain - not sharp
- Often fills whole pelvis
- Pain at the end of orgasm

Treatment:
- OCPs
- Presacral Neurectomy
- Hysterectomy
Endometriosis - Theories of Origin

Reflux Menstruation / Sampson’s Theory

- Happens in 90% of women, only 10% have endo
- Should result in random distribution of lesions and increasing number of locations with increasing age
- Should result in endometriosis genetically identical to endometrium (autotransplant)
- No evidence of early attachment / implantation
- Cannot explain locations of endo outside the pelvis
  - Brain
  - Lung
  - Prostate

Means that endometriosis can never be cured!
Endometriosis - Theories of Origin

Peritoneal Metaplasia

“Secondary Mullerian system”  
Lauchlan, 1972

Innate ability of peritoneal cells to change into cells resembling all parts of the female reproductive tract

Endosalpingiosis

Endocervicosis

Leiomyomatosis peritonealis disseminata

Primary Peritoneal Carcinoma

Endometriosis - Theories of Origin

Peritoneal Metaplasia

Integrates with undisputed facts:

- Ability of peritoneum to “morph” into mullerian type tissues
- Location of endometriosis lesions (uterosacrals, CDS, ovarian fossae most common)
- Static nature of disease (grows deeper, not wider)
- Occurrence outside abdomen (brain, lung, prostate)
- Initial transformation has been visualized in ovaries
- Endo found in infant, premenarchal girls
- Genes found in peritoneum of women with endo, absent in normal patients
Endometriosis - Theories of Origin

Peritoneal Metaplasia

- Normal appearing peritoneal cells in women with endometriosis contain activated genes required for the formation of the uterus and endometrium. (WNT7A, PAX8)

- These genes are absent in women without endometriosis

- Enables development of endometriosis and other mullerian type tissue from peritoneum

Gaetje, et al, Fertility and Sterility 2007
Endometriosis - Theories of Origin

Peritoneal Metaplasia

- Initial transformation from normal ovarian tissue to endometriosis has been visualized.

- 110 cases of ovarian endometriosis, 34 showed IE

Zheng, et al Int J Gynecol Pathol 2005
Endometriosis - Theories of Origin

Peritoneal Metaplasia

- Endometriosis has been identified in human fetuses
  - Autopsy study of 36 female fetuses from 14 weeks to term
  - Stained for CA-125 and estrogen receptors
  - 4 of 36 (11%) had glands and stroma outside the uterine cavity
    - Culdesac, rectovaginal septum, rectum, posterior myometrium

Surgical Approaches to Endo

• **Ablative techniques** (laser, cautery)
  - Burns top 1-2mm of tissue
  - Can’t reach deeply infiltrating disease
  - Doesn’t work for bowel disease, endo over ureters or vessels

• **Excision**
  - Removes all visible disease without removing reproductive organs
  - Allows for removal of endo over ureter, on bowel, diaphragm, etc.
Ablation

- Very few studies

- Short term studies (6 mo f/u) show equivalent results with excision for MILD disease!  
  Wright, et al Fertil Steril 2005

- 1 study with 6 year f/u of laser ablation in stage 1 and 2 disease – 74% chance of pain recurrence at an avg of 20 mo  
  Jones, et al JSLS 2001

- No studies of ablation for stage IV endo
Ablation

Surgical treatment of endometriosis: a prospective randomized double-blind trial comparing excision and ablation


Their conclusion
- no significant difference in postoperative pain at 12 months

• 54 pts in excision group, 49 ablation
• 21 and 25 patients lost to followup - not enough statistical power
• 53% of excision group and 22% of ablation group had deeply infiltrating disease
• No stage IV patients in ablation group, 4 in excision group
• Residents performed the surgeries
# Outcomes

**Reduction in VAS score by 12 months after operation.**

<table>
<thead>
<tr>
<th>Factor</th>
<th>Excision group mean (SD)</th>
<th>Ablation group mean (SD)</th>
<th>T-test P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall pain</td>
<td>2.9 (3.4)</td>
<td>2.9 (2.9)</td>
<td>.93</td>
</tr>
<tr>
<td>Pelvic pain</td>
<td>2.6 (3.5)</td>
<td>2.7 (2.7)</td>
<td>.94</td>
</tr>
<tr>
<td>Period pain</td>
<td>2.4 (3.9)</td>
<td>2.0 (3.9)</td>
<td>.69</td>
</tr>
<tr>
<td>Back pain</td>
<td>1.6 (3.9)</td>
<td>1.1 (2.9)</td>
<td>.49&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Rectal pain</td>
<td>1.4 (3.7)</td>
<td>0.5 (2.7)</td>
<td>.22</td>
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<tr>
<td>Thigh pain</td>
<td>0.9 (2.9)</td>
<td>0.4 (3.0)</td>
<td>.46</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>2.4 (3.1)</td>
<td>2.0 (3.7)</td>
<td>.60</td>
</tr>
<tr>
<td>Defecation pain</td>
<td>1.8 (3.5)</td>
<td>0.7 (3.1)</td>
<td>.16</td>
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<tr>
<td>Voiding pain</td>
<td>0.4 (2.3)</td>
<td>0.6 (2.7)</td>
<td>.70</td>
</tr>
<tr>
<td>Nausea</td>
<td>1.7 (2.7)</td>
<td>0.6 (3.6)</td>
<td>.13</td>
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<tr>
<td>Abdominal bloating</td>
<td>2.4 (3.4)</td>
<td>1.5 (2.8)</td>
<td>.21</td>
</tr>
<tr>
<td>Vomiting</td>
<td>1.1 (2.4)</td>
<td>0.9 (2.3)</td>
<td>.60</td>
</tr>
<tr>
<td>Dyspareunia</td>
<td>3.1 (4.1)</td>
<td>1.8 (4.1)</td>
<td>.17</td>
</tr>
</tbody>
</table>
More problems with laser and cautery...

“Superficial” endometriosis may actually be deep disease
Conservative Excision of Endometriosis

- Surgical removal of all disease, leaving normal reproductive organs in situ
- No need to castrate women in their 20s, 30s, or early 40s
- Allows for treatment of deeply infiltrating endometriosis, as well as lesions overlying the ureter, on the bowel or in the wall of the bladder
- Fertility is preserved, often enhanced even after bowel resections
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- Fertility is preserved, often enhanced even after bowel resections
Published CURE rates following excision

As judged among reoperated patients:

66% cured by laparotomy excision

57% cured by laparoscopy excision*
Redwine DB. Fertil Steril 1991;56:628-34

57% cured by laparoscopy excision
Varol et al. JAAGL 2003:10;182-9

56% cured by laparoscopy excision
Abbott et al. Fertil Steril 2004;82:878 - 84

(No post op medical therapy)

* DISEASE REDUCTION IN MOST OF THE OTHERS
Recurrence after excision


<table>
<thead>
<tr>
<th>Quarters post op</th>
<th>Cumulative persistence/recurrence rate</th>
<th>Wheeler, Malinak</th>
<th>Redwine</th>
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<tr>
<td>0.0</td>
<td>0.000</td>
<td>0.000</td>
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<td>0.537</td>
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</tr>
<tr>
<td>0.6</td>
<td>0.992</td>
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</table>

**Note:** The table shows the cumulative persistence/recurrence rates over different quarters post-op. The graph compares the results with those of Wheeler and Malinak.
## Recurrence after excision

### Recurrence: Amount of new disease after excision, quarterly

<table>
<thead>
<tr>
<th>Quarters post op</th>
<th># of pelvic areas involved</th>
<th>Mean number of pelvic areas involved at reoperation</th>
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<tr>
<td>35</td>
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<td>0.008</td>
</tr>
</tbody>
</table>

Laparoscopic Excision: Response of "moderate" to "debilitating" symptoms

Pelvic pain away from menses
Pelvic pain with menses
Cramps with menses
Dyspareunia
Painful bowel movements

Colored numbers = number of patients responding to question

With moderate To debilitating level of symptoms

YEARS AFTER SURGERY

"...recurrence is not frequent, and cure... by conservative surgery is usual."

J.V. Meigs

*Obstet Gynecol 2:46, 1953*
Persistent Pain after Endo Excision

If we can successfully excise endo 80% of the time, why can’t we cure the pain that often?

Phantom Limb Pain
Persistent pain after amputation (usually traumatic)

CRPS
Mostly used to define persistent pain in a limb after traumatic injury
Mechanisms thought to be neurogenic inflammation, central sensitization, and increased sympathetic tone
With multi-modal therapy can be improved over several years

Interstitial Cystitis/PBS

ICS Definition of Painful Bladder

• The complaint of suprapubic pain related to bladder filling and is accompanied by other symptoms such as increased daytime and night-time frequency, in the absence of proven urinary tract infections or other obvious pathology

Bladder epithelial permeability and GAG layer:

1. – Proposed by Parsons in 1990

2. – Concept was that IC may be result of a defect in epithelial permeability layer GAG’s
   • Hyaluronic acid, Heparin, Chondroitin and keratan sulfate

3. – GAG layer functions as a permeability and anti-adherence layer

4. • Mast Cell Involvement:
   
   Produce histamine

   Histamine release in tissues causes pain and hyperemia and fibrosis

   Mast cell infiltrates found in IC (not specific for IC though)
Interstitial Cystitis/PBS

So what causes the mast cells to degranulate?

Bladder & Levators

Endometriosis
Adenomyosis
UC
Interstitial Cystitis/PBS

Diagnosis:

**Potassium Sensitivity Test**
- Mucosal leak could allow bladder to be more sensitive to stimulus (K+).
- Parsons et al.: instilled H20 or 0.4M K+ solution into normal and IC bladders
  - H20 did not provoke pain in either group
  - 4.5% of normal and 70% of IC patients had pain

**Patients will hate you!**

NIDDK criteria not necessary anymore
Tender bladder accompanied by pain with a full bladder and frequency
Cystoscopy not necessary unless persistent hematuria
Lidocaine/Heparin rescue

Interstitial Cystitis/PBS

Treatment:

Correct the underlying pain generators

Diet - avoid acidic, spicy foods, and alcohol

Medications

- Antihistamine (hydroxyzine or zyrtec)
- Elmiron (Pentosan polysulfate)
- Local anesthetic for bladder (pyridium, hyoscyamine, etc)
- Tricyclic Antidepressants - have anticholinergic properties as well as pain relief

Bladder instillations

- Various recipes/cocktails - we use 8cc 2% lido, 8cc .5% marcaine, 40,000u Heparin, and 4cc bicarb. Can also add steroid

Physical Therapy

- Helpful to relax levators, teach techniques to improve bladder emptying
Vulvodynia

Also called Vestibulodynia - 3-18% incidence

45% of women with vulvodynia also have at least one of the following: IBS, Fibromyalgia, or PBS

84% have PBS/IC

80-90% have pelvic floor spasm

>50% improved with biofeedback PT

Mechanism likely similar to IC-

Neurogenic Inflammation, Peripheral and Central Sensitization
Levator Myalgia
going other myofascial pain

**Symptoms:**
Achy pressure, worse with sitting
Often entrance dyspareunia

80% of women with endo have levator spasm
Caused by viscero-somatic convergence / guarding reflex

**Treatment:**
PT effective about 60-80%
Botox effective 80% in those who have failed PT
Muscle relaxants - systemic vs topical
    Compounded creams -
        Lidocaine, amitriptylene or gabapentin, Baclofen
Levator Ani Anatomy

The Pelvic Diaphragm = the deepest muscle layer

Anterior
- Symphysis pubis
- Urogenital diaphragm
- Urethra
- Vagina
- Anal canal

Posterior

Levator ani
- Pubococcygeus
- Iliococcygeus

(Obscurator internus)
- Coccyx

(Piriformis)

Superior View of Female Pelvis
Levator Myalgia
and other myofascial pain

Myofascial Pain is multifactorial

PTs are your best friends

Patterns of muscle spasm and history of how pain started will give clues to underlying cause

Triad of psoas, piriformis, obturator spasm is often from hip pathology
Pain at attachment of ribs and abdominal wall muscles often referred from pelvis
Viscero-somatic convergence can cause myofascial pain at spinal cord levels associated with various viscera
Pudendal Neuralgia

Searing, burning pain in vagina, vulva, clitoris

Branches in women: clitoral, perineal, rectal

Nantes Criteria:

(1) Pain in the anatomical territory of the pudendal nerve.
(2) Worsened by sitting. (not on a toilet seat)
(3) The patient is not woken at night by the pain.
(4) No objective sensory loss on clinical examination.
(5) Positive anesthetic pudendal nerve block.
Practical Takeaways from Preceding Info

What I have learned from my experiences treating pelvic pain for 10 years....

Treat all primary (peripheral) pain generators aggressively and promptly

Bladder, pelvic floor, and myofascial pain are usually secondary or tertiary
Usually occur via viscero-visceral or viscero-somatic convergence
Often will improve spontaneously after addressing primary source
PT and other interventions more successful after removing primary source

Simultaneously treat central pain with medications for those severely sensitized

Educate patients about pain processing, fear avoidance concepts and role that anxiety and depression play
Patients with severe psychological issues benefit from Cognitive Behavioral Therapy and stress reduction techniques
Putting it all together

Mindfulness - the ability to respond to an experience without judgement - essentially the opposite of pain catastrophizing

CBT - teaches emotional control identifying and restructuring negative automatic thoughts

Acceptance and Commitment Therapy version of CBT

Counteracts avoidance
Putting it all together

Mindfulness - the ability to respond to an experience without judgement - essentially the opposite of pain catastrophizing. CBT teaches emotional control and restructuring automatic thoughts. Acceptance and Commitment Therapy (ACT) counteracts avoidance.
Cases
Chronic Pelvic Pain from Mesh Anchors used in Pelvic Floor Repairs

Case History, Surgical Video, and Discussion

Cindy M Mosbrucker MD
Franciscan Womens Health and Pelvic Surgery
Gig Harbor, WA
Chronic Pelvic Pain from Mesh Anchors used in Pelvic Floor Repairs

Case History, Surgical Video, and Discussion

Cindy M Mosbrucker MD
Franciscan Womens Health and Pelvic Surgery
Gig Harbor, WA
Case 1

- 61 yo G6P5 female who underwent ASC and sling in 2007
- Immediately postop she developed bladder pain, mixed urinary incontinence, recurrent UTIs, and dyspareunia
- Cystoscopy 4 months postop revealed blue sutures in the bladder muscularis, which were not removed
- Presented to me in 2011 with the same complaints as above
  - Exam revealed exquisitely tender bladder and levators, with grade 2 cystocele
  - Urodynamics revealed decreased compliance, uninhibited detrusor contractions at 300cc, complete emptying with normal flow rates
Robotic revision of sacrocolpopexy with removal of prolene sutures
Robotic revision of sacrocolpopexy with removal of prolene sutures
Case 1 postop

- She had nearly immediate relief of bladder pain
- Transurethral foley removed POD #7 after cystogram showed no leakage
- By 2 weeks postop she had improvement of her urge incontinence and her stream was stronger
- By 6 weeks postop the levators were relaxed and minimally tender
Discussion

- Medline search using terms “bladder pain, detrusor spasm, bladder spasm” and “foreign body” revealed 1 pertinent article:
  - “Unrecognized bladder perforations with mid-urethral slings”
    Foley et al. BJU Int. 2010 Nov;106(10):1514-8

- Retrospective review of 9 women with urinary symptoms after slings
  - Presented between 8 weeks and 18 mos postop
  - 8 had retropubic tension free slings, one had l-stop transobturator sling
  - 6 had dysuria, 4 had recurrent UTIs, 4 had urgency and frequency, 2 had pelvic pain
  - All had full-thickness perforations, 3 bilateral, and 3 had urethral mesh exposure
  - Cystoscopic resection resolved symptoms in all but 2 who required open surgery to remove the mesh from the detrusor muscle
Conclusions

- Foreign objects within the wall of the bladder can cause irritatative symptoms without perforation of the urothelium.

- Symptoms can include urgency, frequency, recurrent UTIs, and pain from the bladder itself as well as levator spasm.

- These symptoms can readily resolve after complete removal of the offending material from the detrusor.
58 yo female referred for urinary urgency and frequency.
History reveals dx of endometriosis in her 30s. Told it would go away after menopause
Exam reveals extremely tender left piriformis, ileococcygeus, and uterosacral
Laparoscopy shows endometriosis of left uterosacral and culdesac
Post Excision her pelvic floor relaxed quickly with PT and urinary symptoms resolved
Cases

42 yo female referred for acute urinary retention
Began about a week after an endometrial ablation
Exam reveals exquisite uterine tenderness and pelvic floor tight and tender
Hysterectomy done due to pain, path revealed adenomyosis and endometritis
Began voiding q30 minutes - her typical pattern
42 yo female referred for acute urinary retention
  Began about a week after an endometrial ablation
  Exam reveals exquisite uterine tenderness and pelvic floor tight and tender
  Hysterectomy done due to pain, path revealed adenomyosis and endometritis
  Began voiding q30 minutes - her typical pattern

Further history reveals sexual abuse for several years in her childhood
  Began intravaginal valium, pelvic PT
  Interstim placed for persistent urinary frequency- eventually voiding q2 hours
Thank you for your attention!