



September 2023

Dear Attendee:

We are delighted to have your participation in the 37<sup>nd</sup> Annual Southern Pain Society Meeting. We hope you find the speakers enlightening and informative.

As you may know, SPS represents the 18 southern states including Puerto Rico. Our membership base is strong and we have many active and engaged professionals who serve on a number of committees.

If you have not already done so, we hope you will consider becoming a member of the Society and participate in the growth and development of the organization as well as the discipline of pain management. Membership applications are included in your packet and you will get 2 years membership for the price of one if you sign up during the meeting. We welcome your input, suggestions and contributions.

Best wishes for a great meeting!

A handwritten signature in black ink that reads "Thomas Davis, MD". The signature is fluid and cursive, with the letters "T", "D", and "M" being particularly prominent.

Thomas Davis, MD  
President

Slide 1

How to Develop a  
Multidisciplinary Pain Program  
in a Non-Academic Setting

Norman Harden  
Depts PM&R and PTHMS  
Northwestern University  
normanharden3@gmail.com

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Slide 2

Pain Management in Urban and  
non-academic Practice

There is no reason that high quality cannot  
be delivered in a non-academic and/or rural  
practice

Harden 2023

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Slide 3

- Conflicts: none
- Bias: An interdisciplinary approach is far  
better than a unidisciplinary.

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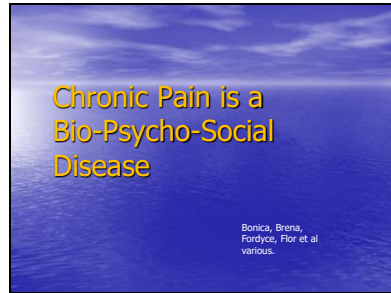
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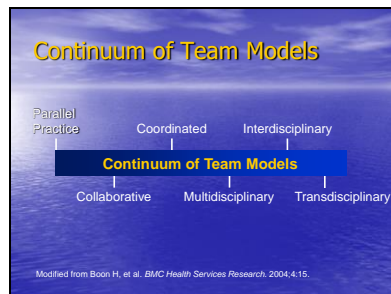
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Slide 5



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Slide 6



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Slide 7

### Interdisciplinary Team Model

- Patient Centered
- Team members work together toward a common goal
- Make collective therapeutic decisions
- Communicate and consult with other team members, facilitated by planned face-to-face meetings
- Teams possess a combination of skills that no single individual demonstrates alone
- Team is able to achieve more than the sum of the individuals involved

Boon H, et al. BMC Health Services Research. 2004;4:15. Cummings I. The interdisciplinary team. In: Doyle D, Hanks GW, MacDonald N, editors. Oxford textbook of palliative medicine. Oxford:Oxford University Press; 1998. Parker GM. Cross-functional teams, working with allies, enemies and other strangers. San Francisco:Jossey-Bass Publishers; 1994.7:31-40.

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Slide 8

### THE TRUTH AS WE KNOW IT.....

THE JOURNAL OF  
PAIN MANAGEMENT

ISSN 0895-5162

VOLUME 10  
NUMBER 1  
JANUARY 1992

Efficacy of multidisciplinary pain treatment centers:  
a meta-analytic review

Barry Pao<sup>1,2</sup>, Thomas Flynn<sup>3</sup> and Dennis C. Turk<sup>4</sup>

<sup>1</sup>Department of Clinical Neurophysiology, University of Western Australia, Perth, Australia; <sup>2</sup>Department of Neurology, University of Washington, Seattle, Washington; <sup>3</sup>Department of Psychology, University of Washington, Seattle, Washington; and <sup>4</sup>Department of Psychology, University of Washington, Seattle, Washington

Received 1 April 1991; revised accepted 9 May 1991; accepted 12 September 1991

**Abstract** Seven literature studies that evaluated the efficacy of multidisciplinary treatment for chronic pain were included in a meta-analysis. Within and between group effect sizes revealed that multidisciplinary treatment for chronic pain was superior to the treatment, waiting list, as well as single discipline treatment with a medical component or physical therapy treatment. The effect size was in the small to medium range. The behavioral effects of multidisciplinary treatment were not limited to improvements in pain, mood and performance but also extended to behavioral variables such as return to work or use of the health care system. These results have to consider the efficacy of multidisciplinary pain treatment. However, these results must be interpreted cautiously in the quality of the study design and study characteristics in general. Suggestions for improvements in research design as well as management aspects of chronic pain are provided.

**Key words:** Neuroscience, Chronic pain, Multidisciplinary treatment

1992

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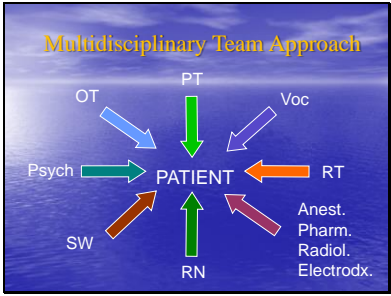
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Slide 10



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Slide 11

**Doctors Role:**  
**Diagnose first then Treat**

- Ideal is to match the mechanism of disease with the mechanism of action of the intervention.
- Problem: We know little about the mechanisms of our interventions, and less about the pathophysiology of pain.

Harden et al *Pain Medicine* 2022

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Slide 12

**Must use available diagnostic Criteria**

Table 4  
Proposed experimental revision of CRPS diagnostic criteria

(1) Continuing pain which is disproportionate to any inciting event

(2) Must report at least one symptom in each of the four following categories

Sensory symptoms of hyperalgesia  
Fluctuating reports of temperature asymmetry and/or skin color changes and/or skin color asymmetry  
Allodynia/itching reports of edema and/or swelling changes and/or swelling asymmetry

Motor symptoms: evidence of decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin)

(3) Must display at least one sign in two or more of the following categories

Sensory evidence of hyperalgesia (to pinprick) and/or allodynia (to light touch)

Vasomotor evidence of temperature asymmetry and/or skin color changes and/or asymmetry

Edema/itching evidence of edema and/or swelling changes and/or swelling asymmetry

Motor/trophic evidence of decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin)

Harden et al *Pain* 1999

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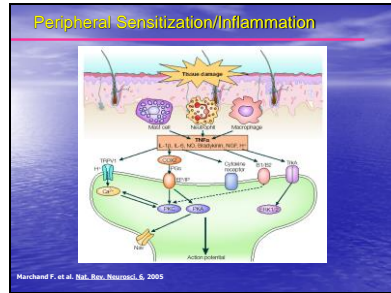
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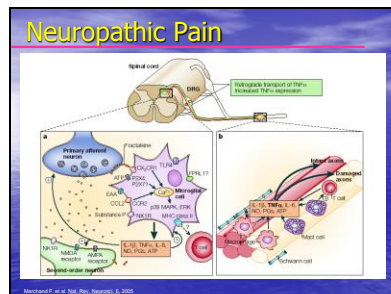
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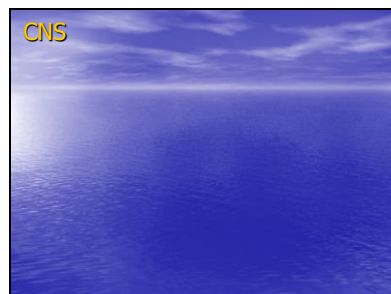
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Slide 14



Slide 15



Slide 16



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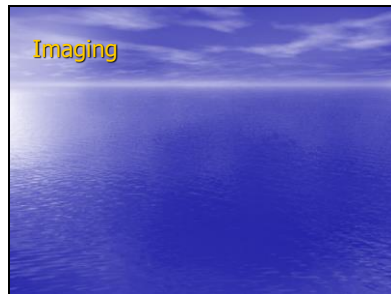
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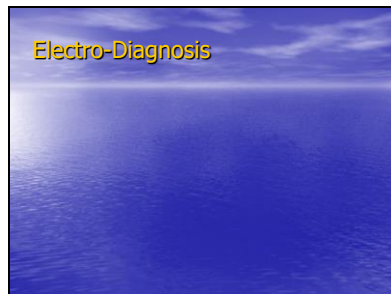
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Slide 18



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Slide 19

**Myopain:**

**Fibromyalgia** (Whole body)  
**Myofascial Pain Syndrome** (regional)

- Weakness
- Dystonia
- secondary ~ contracture
- kinesiophobia

Trigger Points, Tender Points

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Slide 20

**Psychopathology**

Fear  
Anxiety  
Anger  
Suffering  
Depression  
Failure to Cope



Raja SN et al. Anesthesiology. 2002;96:1254-1260.

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Slide 21

**Pain and Depression in Primary Care**

- 1/3 to 1/2 of patients with CP have co-morbid depression
- 3/4 of depressed people present with physical sxs, including pain
- Pain often best indicator of dep in elderly
- Depression increases impairment from CP
- Pain+depression=increased health care use/cost

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Slide 22

### Anxiety

- Pain Anxiety
  - Associated with depressed mood, high levels of pain, and low lifting and carrying capacity during functional assessment in patients with chronic pain.
  - Exposing patients to physical maneuvers that they expect to elicit increased pain reduces pain anxiety and increases physical activity.

Burns WJ et al. Pain 2000; 84: 247-252  
Mayer T et al. Clin Pain 2002; 12: 251-261

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Slide 23

### Axis II

prevalence of personality disorders in chronic pain populations is 31-64%

Weissberg, 2000

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Slide 24

### Operant Issues

(esp. Social Work, Voc Rehab)

Work Comp	54%
3rd Party Lawsuit	17%
Litigation Total	71%
IME'S	23%

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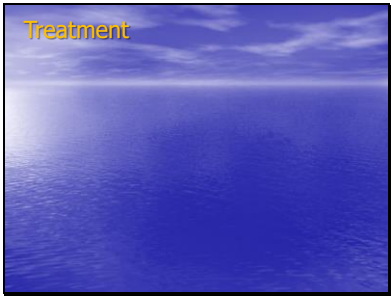
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Slide 27



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Slide 28

Pharmacotherapy (empirical, consensus)

Reason for inability to begin or progress	Action
Mild to moderate pain	Simple analgesics and/or blocks
Excruciating, intractable pain†	Opioids and/or blocks or later, more experimental interventions
Inflammation/swelling and edema	Steroids, systemic or targeted (locally) or NSAIDs (chronically); immunomodulators
Depression, anxiety, insomnia	Sedative, analgesic antidepressant/antidysrhythmic and/or psychotherapy
Significant allodynia/hyperalgesia	Anticonvulsants and/or other sodium channel blockers and/or NMDA-receptor antagonists
Significant osteopenia, immobility and trophic changes	Calcitonin or bisphosphonates
Profound vasomotor disturbance	Calcium channel blockers, sympatholytics and/or blocks

Harden et al 2022

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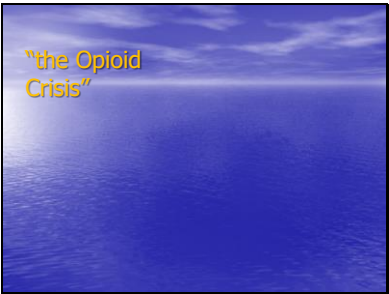
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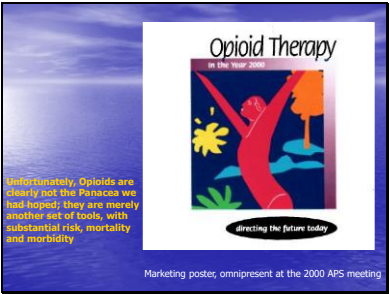
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Slide 30



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Slide 31

**Interventional Pain Therapy**

- Minimally Invasive Therapies
  - Sympathetic / Somatic nerve blocks
  - IV Regional nerve blocks
- More Invasive Therapies
  - Epidural / Plexus Catheter Blocks
  - Neurostimulation
  - Intrathecal Drug Infusion
- Surgical Therapies
  - Sympathectomy
  - Motor Cortex Stimulation

Rutten A. Interventional therapies. Complex Regional Pain Syndrome: Treatment Guidelines. RSDSA press. 2006;31-62. Velasco F. Pain. 2009; Volume 147, Issue 1, Pages 91-98.

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Slide 32

**Never underestimate the psychotherapeutic value of 'What the Doctor says'**

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Slide 33

**Physical Therapy (DPT, RPT)**

- De-emphasizing high tech, passive modalities
- Emphasizing low tech, self management and active modalities
- Reactivation "Reanimation"
- Stretch/strengthen
- Desensitization
- Mirror Therapy

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Slide 34

**Adage/Legend/Lore/Anecdote/  
Empirical 'Truth'**

Must counter 'kinesiophobia'  
Must reanimate the affected part  
Must normalize use

\*first thought to be stimulation of large fibers to 'shut' the pain gate  
(precedence over small fiber input), normalize and balance input to CNS

\*but evidence from mirror therapy suggests there is more to it than just  
afferent normalization

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Slide 35

**Reactivation, Aerobic conditioning**

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Slide 36

**Functional Movement**

- As often as possible, use function based exercises
  - For UE involvement: instead of working on supination/pronation, have the patient practice turning door knobs/opening doors
  - For LE involvement: exercise should promote gait and equal weight bearing (i.e. offset static standing, progressive step ups, weight shifting during leisure activity)

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Slide 37

### Occupational Therapy (OTR)

- Postural training
- Work/fun station analysis, correction
- Orthotics
- Work hardening

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Slide 38

### Functional Positioning

- Proper posture
- Decrease compensation
- Proper body mechanics
- Ergonomic principles

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Slide 39

### Desensitization

- Initiate at evaluation appointment, if possible
- Explain clearly how to perform and what to expect
  - Measure patient understands that desensitizing an area to a pressure/temperature does not mean that it will begin to feel "good"
- Be creative with desensitization media
  - Mrs. G. very fearful/denies/poor of treatment, poor prior experience with OT= started desensitization by petting her kitten
- Establish daily regimen for desensitization multiple times/day
  - Consistent demand is key

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Slide 40

### Edema Management

- Passive Edema Management
  - Use of Coban™ or Jobst®/Isotoner® garments for severe edema
  - Consider functional splinting
- Active Edema Management
  - Control of edema through activity and positioning

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Slide 41

### Mirror therapy for CRPS

- Daily exercise of cardinal movements of the affected limb while viewing an image of their unaffected limb in a mirror for 30 minutes daily has been shown to improve pain, motor function, and edema!
- Sensory discrimination training while looking toward the affected body part but seeing the opposite part of the body in the mirror also decreased pain and increased tactile acuity?



1. Caschito C et al. New England Journal of Medicine 2009; 361:6, 634-636.  
2. Moseley GL et al. Pain, 2009; 144: 214-19

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Slide 42

### Flare Up Plan

- Decrease but DO NOT STOP activity
- Utilize all pain management strategies
  - Pacing of activities
  - Thermal modalities
  - Relaxation or biofeedback exercises
  - Medications
- Increase emphasis on scrubbing during flare and for several days after

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Slide 43

**Therapeutic Recreation**

- Leisure assessment
- Modifications

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Slide 44

**Psychotherapy**

- Cognitive behavioral therapy
- Stress management
- Coping skills
- Relaxation techniques
- Imagery
- Self hypnosis

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Slide 45

**Cognitive Factors That May Contribute to Treatment Outcomes**

- Readiness to Change
- Acceptance of Chronicity
- Cognition of situation
- Five stages that reflect readiness to make behavioral changes
  - Precontemplation
  - Contemplation
  - Preparation
  - Action
  - Maintenance

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Slide 46

**Biofeedback**

- EMG biofeedback
- Autogenic training
- Progressive muscle relaxation
- Meditation
- Sleep hygiene

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Slide 47

**Vocational Rehabilitation**

- Job description
- Site analysis
- Testing
- Return to work assessment
- Placement

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Slide 48

**Complementary-Alternative  
Medicine**

- 629 million visits to CAM practitioners 1997
  - Up from 427 million in 1990
  - Only 243 million visits to PCP's in 1997
- 42.1% of population used at least one of 16 CAM therapies 1997
  - 33.8% in 1990

Eisenberg, JAMA 1998.

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Slide 49



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Slide 1



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# Managing Chronic Pain in the Substance Use Disorder Patient: Theory & Practice

Sudheer Potru, DO, FASA, FASAM  
Medical Director, Complex Pain and High-Risk Opioid Clinic  
Atlanta VA Health Care System  
Assistant Professor, Department of Anesthesiology  
Emory University School of Medicine

 @SudheerPotru

Presentation to the Southern Pain Society Meeting  
September 29, 2023

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Slide 2



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# No Financial Disclosures

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Slide 3



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# Learning Objectives

- Differentiate addiction from chronic pain and physical dependence
- Identify appropriate medical and psychosocial treatment options for comorbid addiction along with pain
- Describe strategies for counseling patients regarding addiction and linkage to specialist treatment

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Slide 4

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About Me

- Triple-boarded in anesthesiology, pain medicine, and addiction medicine
- I run a super-specialty multidisciplinary pain clinic for veterans on high-dose opioids or with comorbid SUD issues
- This lecture is a combination of evidence-based practice, common sense, and good old-fashioned hard knocks of clinical practice

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Slide 5

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So how exactly does addiction happen?

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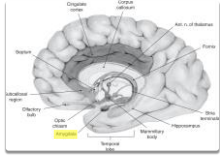
DEPARTMENT OF ANESTHESIOLOGY

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Neurobiology I: Limbic System

- Emotion
- Behavior
- Memory
- Long-term motivation



From Koob GF, et al. (2012) The ABCs of Addiction Medicine, Second Edition. New York, New York: LWW.

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Slide 7

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### NB II: Mesolimbic i.e. "Reward" Pathway

- Ventral tegmental area (VTA) → **\*\*dopaminergic**, GABAergic, glutamatergic
- VTA connects to:
  - Nucleus accumbens** (reinforcement and reward for motor learning)
  - Prefrontal cortex** (higher-order processing e.g. planning/executive functioning)
- Release of dopamine into NAcc regulates motivation and desire for stimuli and causes reinforcement and reward for motor learning
- Release of dopamine into PFC affects executive functioning (substances)

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Slide 8

FAHRY

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Slide 9

FAHRY

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### Dopamine: the "pleasure" neurotransmitter

- Released when we eat, drink, sleep, have sex, etc. (life-sustaining activities)
- Drugs of abuse cause release of dopamine in much higher concentrations**, so regular life-sustaining activities don't provide as much euphoria
- This results in corruption of the reward pathway

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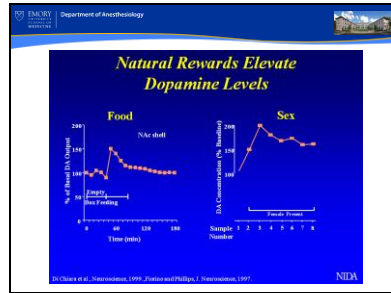
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graph TD; A[POU] --> B[Behaviors more suggestive of POU]; B --> C[Deterioration in function (work, social)]; C --> D[Illegal activities (selling medication, forging prescriptions, buying from non-medical sources)]; D --> E[Altering the route of administration (snorting, injecting)]; E --> F[Multiple episodes of "lost" or "stolen" prescriptions]; F --> G[Resistance to change therapy despite negative outcomes]; G --> H[Refusal to comply with toxicology testing]; H --> I[Concurrent, active abuse of alcohol, illegal drugs]; I --> J[Use of multiple physicians or pharmacists to obtain the prescription]; J --> K[Behaviors less suggestive of POU]; K --> L[Medication hoarding]; L --> M[Requesting specific pain medications]; M --> N[Openly acquiring multiple medications from other providers]; N --> O[Occasional unactioned dose escalation]; O --> P[Nonadherence to other recommendations for pain therapy];
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The flowchart illustrates the progression of behaviors from Prescription Opioid Use (POU) to more severe, non-medication-related actions. It begins with POU, which leads to behaviors more suggestive of POU. These behaviors include deterioration in function (work, social), illegal activities (selling medication, forging prescriptions, buying from non-medical sources), altering the route of administration (snorting, injecting), multiple episodes of "lost" or "stolen" prescriptions, resistance to change therapy despite negative outcomes, refusal to comply with toxicology testing, concurrent active abuse of alcohol and illegal drugs, and the use of multiple physicians or pharmacists to obtain prescriptions. These behaviors lead to behaviors less suggestive of POU, which include medication hoarding, requesting specific pain medications, openly acquiring multiple medications from other providers, occasional unactioned dose escalation, and nonadherence to other recommendations for pain therapy.

Slide 16


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So how do you treat addiction?

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Non-pharmacologic therapies for SUDs

Cognitive-behavioral therapy

Contingency management

Social and family support

Community reinforcement

Network therapy

12-step programs

Aversion therapy

Spirituality programs

Motivational interviewing

From Koob GF (2010) The Neurobiology of Addiction. Boston: Second Edition. New York: New York: 2010.

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Pharmacologic treatment of SUDs

• Alcohol

- disulfiram
- Acamprosate
- Naltrexone/gabapentin
- Topiramate/gabapentin may help

• Opioids

- Buprenorphine
- Methadone
- Naltrexone

• Benzodiazepines

- Carbamazepine for w/d
- Flumazenil for intoxication

• Stimulants

- No FDA-approved agents
- TCAs may help

• Tobacco

- NRT
- Varenicline
- Bupropion
- Nortriptyline

• Cannabinoids

- No FDA-approved agents
- Rimonabant (CB1 antagonist)

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Complex persistent opioid dependence (CPOD)

- Somewhere between physiologic opioid dependence and OUD, coined by Manchhara and Ballantyne (no DSM or ICD code)
- CPOD is characterized by:
  - Poor pain control
  - Aberrant behaviors
  - Declining function
  - Medical/psychiatric instability
  - Difficulty tolerating opioid tapers (severe loss of function)
- "Although OUD commonly develops through the hedonic use of opioids, illicitly and/or via prescriptive pain treatment, **CPOD distinctly starts and persists within a therapeutic context of pain treatment** where LOT is initiated and continued as a therapeutic strategy through shared decisions by the patient/provider dyad."
- Some patients benefit with buprenorphine

Montgomery et al. J Gen Intern Med 2022;37(1): 566-571

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LETTER TO THE EDITOR

BUPRENORPHINE: NOT A SILVER BULLET, AND STILL CONTROVERSIAL

Utilizing buprenorphine in treating CPOD has saved lives. While buprenorphine may have an improved side effect profile compared to other opioids, and while the liability related to prescribing it may be somewhat lower versus other opioids, it is our firm belief that **buprenorphine should not replace all full opioid agonist medications in the pain management arsenal**. As with any other drug, excellent treatment modality for appropriate clinical situation, the risks and benefits of all therapies patients and clinical scenarios, is ultimately **patient** individualized and a plan discussed collaboratively with the patient and interdisciplinary team.

We applaud the pain community for its increasing awareness of substance use disorders within our patient population and commitment to address the issue more effectively. We must also exercise caution and remember that buprenorphine, while an excellent treatment modality for appropriate clinical situation, the risks and benefits of all therapies patients and clinical scenarios, is ultimately **patient** individualized and a plan discussed collaboratively with the patient and interdisciplinary team.

Potru et al. Journal of Opioid Management (2021)

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Slide 21

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Opioid Risk Tool (ORT)

Mark each box that applies	Female	Male
Family/History of substance abuse		
Alcohol	3	3
Illegal Drugs	2	3
No drugs	4	4
Personal History of substance abuse		
Alcohol	3	5
Illegal Drugs	4	4
No drugs	5	5
Age between 18-45 years	1	1
History of simultaneous sexual abuse	3	0
Psychological disease		
ADHD, PTSD, bipolar, schizophrenia	2	2
Anxiety disorder	1	1
Scoring totals		

Woolson JA, Woolson K. Predicting aberrant behaviors in opioid-treated patients: preliminary validation of the Opioid Risk Tool. Arch Intern Med 2003; 163: 1292-1299.

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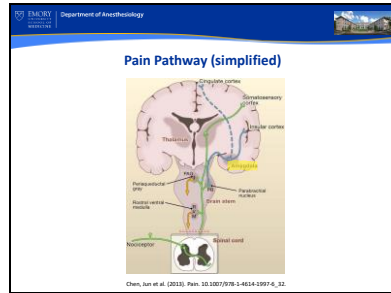
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Slide 22



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Slide 23

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### Mental Health, Pain, and Addiction

- My clinical experience is that, often, the highest opioid doses end up going to those with history of physical or emotional trauma (stated or unstated) → **CRITICAL**
- > 50% of the opioids in the U.S. go to those with mental health disorders (16%)
- Treating physical pain, emotional suffering, or both?
- Manifestation of trauma as centralized pain syndromes (fibromyalgia, IBS, migraine, etc)
- Possible mechanisms:
  - Amygdala?
  - Glut cell activation?

Green MA, Laska LS, Clark SD. Prescription Opioid Use among Adults with Mental Health Disorders in the United States. J Am Board Fam Med. 2017 Jun-Aug;30(6):657-667. doi: 10.1007/s12068-017-9263-3. PMID: 28570624.

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Slide 24

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### So how do I approach the addicted patient clinically?

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Slide 25



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## Big Question: do you want this patient in your clinic or not?

Everything starts from that!

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### American Society of Addiction Medicine: Definition of Addiction (2011)

- "Addiction is a **primary, \*\*\*chronic disease\*\*\* of brain reward, motivation, memory and related circuitry**. Dysfunction in these circuits leads to characteristic biological, psychological, social and spiritual manifestations. This is reflected in an individual pathologically pursuing reward and/or relief by substance use and other behaviors.
- Addiction is **characterized by** inability to consistently abstain, **impairment in behavioral control, craving, diminished recognition of significant problems with one's behaviors and interpersonal relationships, and a dysfunctional emotional response**. Like other chronic diseases, addiction often involves **cycles of relapse and remission**. Without treatment or engagement in recovery activities, addiction is progressive and can result in disability or premature death."

From Koob GF et al. (2011). The ASAM Criteria of Addiction Medicine. Second Edition. New York, New York: ASAM.

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Slide 27



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### Clinical Evaluation

- If long-term sobriety:
  - Will admit to issues in the past: "I don't want medications"
  - Will repeatedly ask if certain treatments are habit-forming
  - Will ask if treatments are likely to affect their mood
- If undiagnosed:
  - None of the above
  - Will persevere on getting psychoactive medications
- If short-term sobriety, who knows
- Just remember that there are permanent brain changes that take years to change in some instances

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### Clinical Evaluation (cont)

- Disguise your addiction evaluation as a pain evaluation
  - Start by asking how long ago they started using pain medications
  - Ask in detail how they use their medications
  - Do they run out early often? Do they have withdrawal?
  - Some OUD patients will complain of compulsions to be "out of pain"
  - "Pseudoaddiction" – *o*y, don't get me started on Purdue Pharma
- Generate genuine empathy by imagining a close family member sitting in the exam room with you, dealing with both pain and addiction
- Frame everything in the context of safety, e.g. "I don't want anything bad to happen to you"
- Chronic pain patients with substance use disorders will have pain flares/exacerbations
  - Treat them appropriately
  - Uncontrolled pain is a factor for relapse!!**
  - SUD patients resemble opioid-TOLERANT → they need MORE, not less**

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### Clinical Evaluation (continued) – Red Flags

- With an unexpected test result, it's time to (gently) ask more questions
- If you're unsatisfied with the answer or if it's inconsistent with objective findings (POMP, UDS, random pill count, etc.), it's time to ask more questions
- When someone says "the medication relaxes me" or "I take it and then I can finally go to sleep", it's time to ask more questions
- You may discover SUD, CPD, or some underlying mental health issue
  - If so, it's time to refer to a specialist or start buprenorphine if OUD/CPD

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### Treatment Considerations

- Think about a buprenorphine patch to bridge these patients if you don't want to give C-II medications
  - Virtually impossible to overdose, except with significant amounts of CNS depressants etc also
  - Maybe somewhat protective, although receptor occupancy likely too low
- Assume fentanyl products are in all pills from non-medical sources
  - This is a big problem with stimulants (pts opioid-naïve)

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Let's talk a little bit more about buprenorphine...

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Buprenorphine Formulations

Formulation	Indication	Dose (mg)	Frequency	Notes
Sublingual tablet (generic)	Opioid dependence	2 mg, 8 mg	Once daily	N
Sublingual tablet, film (generic, Suboxone)	Opioid dependence	2 mg/0.5 mg, 4 mg/1 mg, 8 mg/2 mg, 12 mg/3 mg	Once daily	Y
Sublingual tablet (Suboxone)	Opioid dependence	0.7 mg/0.16 mg, 1.4 mg/0.32 mg, 2.9 mg/0.71 mg, 5.7 mg/1.4 mg, 8.6 mg/2.1 mg, 11.4 mg/2.9 mg	Once daily	Y
Buccal film (Bunavail)	Opioid dependence	2.1 mg/0.5 mg, 4.2 mg/1 mg, 6.3 mg/1 mg	Once daily	Y
Buccal film (Belbuca)	Chronic pain	75 mg, 150 mg, 300 mg, 450 mg, 600 mg, 750 mg, 900 mg	Every 12 hours	N
Intravenous (Buprenex)	Acute pain	0.3 mg/mL	Every 6 hours as needed	N
Subcutaneous extended release injection (Sublocade)	Moderate to severe opioid use disorder	100 mg/0.5 mL, 300 mg/1.5 mL	Monthly	N
Transdermal patch (Butrans)	Chronic pain	5 mcg/hr, 7.5 mcg/hr, 10 mcg/hr, 15 mcg/hr, 20 mcg/hr	Every 7 days	N

Warner M, Warner M, Cunningham R, et al. A Practical Approach for the Management of the Mixed Opioid-Addicted Patient Requiring Buprenorphine During Acute Pain and for Opioid Withdrawal. 2019;10(1):1-10.

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Slide 36

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Is precipitated withdrawal a real concern?

Not if you give clear instructions.

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## Conversion to Buprenorphine (Webster)

For patients taking doses below the following amounts ( $< 200 \mu\text{g/day}$ ):

- Fentanyl transdermal: 25  $\mu\text{g/h}$
- Oxycodone: 400 mg/day
- Hydromorphone or morphine: 300 mg/day
- Hydrobromide: 240 mg/day
- Oxycodone: 40 mg/day
- Tapentadol: 400 mg/day

1. Discontinue after the last night-time dose.

2. Consider initiating an alternative  $\mu$ -agonist (e.g., buprenorphine, or a combination of buprenorphine and naloxone) on an equal weight basis.

3. Consider increasing the following amount by the preceding value, until the patient is comfortable with the prescribed alternative.

**Caution:** Do not stop  $\mu$ -agonist or combination in the preceding statement.

In patients transitioning to buprenorphine from higher doses of other  $\mu$ -agonists:

- Buprenorphine transdermal:  $> 25 \mu\text{g/h}$
- Oxycodone:  $> 400 \text{ mg/day}$
- Hydromorphone or morphine:  $> 300 \text{ mg/day}$
- Hydrobromide:  $> 240 \text{ mg/day}$
- Oxycodone:  $> 40 \text{ mg/day}$

4. Discontinue after the last night-time dose.

5. Consider initiating an alternative  $\mu$ -agonist (e.g., buprenorphine, or a combination of buprenorphine and naloxone) on an equal weight basis.

6. Consider increasing the following amount by the preceding value, until the patient is comfortable with the prescribed alternative.

**Caution:** Do not stop  $\mu$ -agonist or combination in the preceding statement.

7. Consider increasing the dose of the buprenorphine by the preceding value, until the patient is comfortable with the prescribed alternative.

**Pain Medicine, 21(4), 2020, 714-723**

doi: 10.1093/pm/pnz356

**Legal Considerations**

**Drug Addiction Treatment Act (DATA) 2000**

- Permitted qualified physicians to treat opioid addiction with Schedule III, IV, and V medications (i.e. only buprenorphine)
- Required 8 hours of training as prescribers, 24 hours for mid-level providers
- Apply to Substance Abuse and Mental Health Services Administration (SAMHSA) for "X-waiver" once a doctor's first letter becomes X
- The practitioner [must have] the capacity to provide diversion referral, [or in another manner] appropriate counseling and other appropriate ancillary services."

Source: Regulatory and Compliance Management | www.samhsa.gov

**Identifying Barriers to Buprenorphine Treatment for Patients with Opioid Use Disorder Among Anesthesiologists and Pain Practitioners: A Survey Study**

Samuel John, BS, MPH, NREMT, David R. Bowman, MS, and Sallie Perin, MD, FRCPC

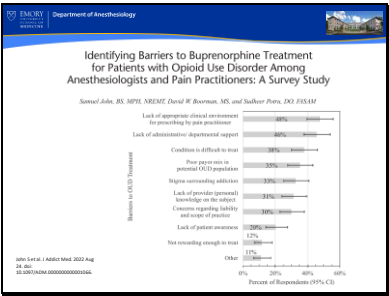
Barrier	Anesthesiologists (n=100)	Pain Practitioners (n=100)
Lack of appropriate clinical environment (percepting the patient population)	48%	40%
Lack of administrative departmental support	34%	30%
Confusion as to what is difficult to treat	28%	20%
Poor patient or potential OUD population	25%	20%
Stigma surrounding addiction	22%	20%
Lack of provider knowledge or knowledge on the subject	21%	20%
Concerns regarding liability and scope of practice	19%	20%
Lack of patient insurance	17%	20%
Not considering enough to treat	17%	20%

Legend: Anesth (Blue), Pain (Orange)

X-axis: Percent of Responders (0%, 20%, 40%, 60%, 80%, 100%)

Y-axis: Barriers to OUD Treatment

Slide 40



Slide 41

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You are called for an inpatient pain consult for a 23-year-old woman w/ a prior episode of overdose secondary to intravenous drug use with heroin, but used 3 days ago. She is in the ICU and having mild pain, but reports experiencing severe opioid cravings and withdrawal symptoms. Clinician has helped only slightly. The hospital's addictionologist is on vacation and will return next week. Would you be willing to initiate buprenorphine treatment in the hospital for this patient?

A 55-year-old man with OUD and treated with buprenorphine in post-operative day 0. He reports no shoulder arthralgia is about to go home. If discharge with buprenorphine is part of the treatment plan, would you as an anesthesia provider be willing to prescribe it for a few days?

**"Overall, most practitioners agreed to prescribe buprenorphine in both cases (63% acute pain, 78% chronic pain, respectively)."**

**Work Environment as a Barrier**

Three of the top 4 barriers suggest that future improvements to OUD-related care should alter the work environment. These barriers are: "lack of appropriate clinical environment for prescribing by pain practitioners," "lack of administrative/departamental support," and particularly for chronic pain practitioners, "poor access to potential OUD population." The underlying theme is that policies are needed to enact systemic changes to the OUD treatment clinical environment, rather than solely focusing on individual-level, practitioner training. For example, several re-

John S et al. J Addict Med. 2022 Aug; 24(4):453-458. doi: 10.1093/ajam/0000000000000086.

Slide 42

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**Is buprenorphine effective for chronic pain?**



# Webster et al – buprenorphine provides good analgesia

In some clinical settings, buprenorphine had similar or greater analgesic efficacy and antipruritic effects as full-potential opioid agonists [14,29–32]. Intravenous (IV) buprenorphine was as efficacious as IV morphine for pain relief in IV morphine or morphine-controlled patients [14,29–32]. However, the analgesic effect of buprenorphine was shorter-acting than morphine, with a half-life between two variables, but change in pain intensity observed in clinical trials of randomized and fixed buprenorphine to placebo outperformed that of several scheduled II opioids across trials of chronic, acute pain, suggesting similar efficacy [29]. In addition, sublingual buprenorphine was as effective as IV morphine in managing acute mild-to-moderate pain [33]. Therefore, the characteristics of buprenorphine as an analgesic agent are unique, and it may be a useful analgesic agent that does not clinically require a general anesthetic.

*Pain Medicine*, 21(4), 2020, 714–723  
doi: 10.1093/pm/pnz356

[illegible][illegible]

Slide 46

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**Treatment of Chronic Pain With Various Buprenorphine Formulations: A Systematic Review of Clinical Studies**

Rohit Agre, MD,\* Anish Gulati, MD,† Senthil Gungor, MD,‡ Anuj Bharti, MD,§ and Neel Mehta, MD§

**Table 1. Characteristics of Included Studies That Evaluated Buprenorphine/Naloxone Formulations for Chronic Pain**

Reference	Year	Patients (n)	Study Design	Intervention	Comparator	Scale	Study Results	Adverse and Results
Agre et al <sup>1</sup>	2012	100	Randomized controlled trial	Transdermal buprenorphine/naloxone patch (5 µg/0.5 µg)	Transdermal fentanyl patch (25 µg)	100	Significant reduction in pain scores	Significant reduction in pain scores
Gulati et al <sup>2</sup>	2013	100	Randomized controlled trial	Transdermal buprenorphine/naloxone patch (5 µg/0.5 µg)	Transdermal fentanyl patch (25 µg)	100	Significant reduction in pain scores	Significant reduction in pain scores
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Mehta et al <sup>5</sup>	2016	100	Randomized controlled trial	Transdermal buprenorphine/naloxone patch (5 µg/0.5 µg)	Transdermal fentanyl patch (25 µg)	100	Significant reduction in pain scores	Significant reduction in pain scores

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Slide 47

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Slide 48

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**Treatment of Chronic Pain With Various Buprenorphine Formulations: A Systematic Review of Clinical Studies**

Rohit Agre, MD,\* Anish Gulati, MD,† Senthil Gungor, MD,‡ Anuj Bharti, MD,§ and Neel Mehta, MD§

Of the 25 studies reviewed, a total of 14 studies demonstrated clinically significant benefit with buprenorphine in the management of chronic pain:

- 1 study out of 6 sublingual and intravenous buprenorphine,
- the only sublingual buprenorphine/naloxone study,
- 2 out of 3 studies of buccal buprenorphine, and
- 10 out of 15 studies for transdermal buprenorphine showed significant reduction in pain against a comparator.

No serious adverse effects were reported in any of the studies. We conclude that a transdermal buprenorphine formulation is an effective analgesic in patients with chronic pain, while buccal buprenorphine is also a promising formulation based on the limited number of studies.




Slide 52

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Questions?

[sudheer.potru@emory.edu](mailto:sudheer.potru@emory.edu)

 [@SPotruDO](https://twitter.com/SPotruDO)

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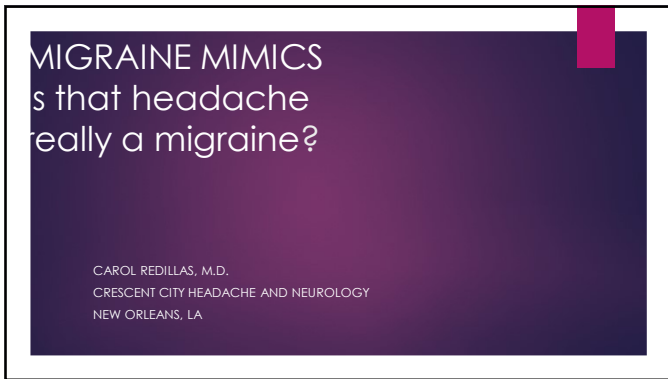
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**MIGRAINE MIMICS**  
Is that headache really a migraine?

CAROL REDILLAS, M.D.  
CRESCENT CITY HEADACHE AND NEUROLOGY  
NEW ORLEANS, LA

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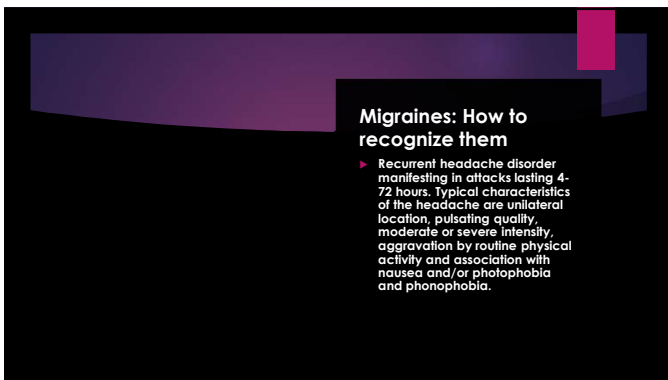
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**Migraines: How to recognize them**

- ▶ Recurrent headache disorder manifesting in attacks lasting 4-72 hours. Typical characteristics of the headache are unilateral location, pulsating quality, moderate or severe intensity, aggravation by routine physical activity and association with nausea and/or photophobia and phonophobia.

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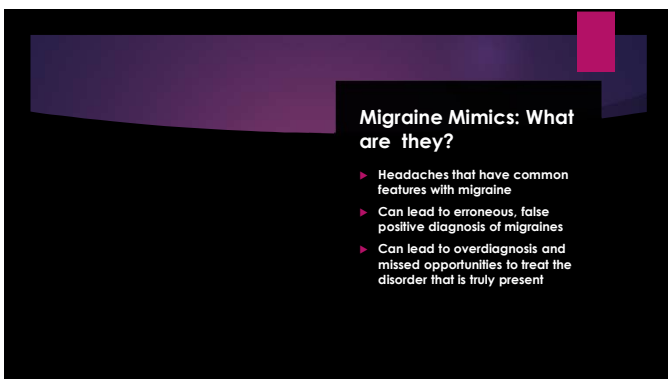
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**Migraine Mimics: What are they?**

- ▶ Headaches that have common features with migraine
- ▶ Can lead to erroneous, false positive diagnosis of migraines
- ▶ Can lead to overdiagnosis and missed opportunities to treat the disorder that is truly present

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### Migraine Mimics: When to consider

- ▶ At the time of the initial consultation
- ▶ Those with a diagnosis of migraine but has not responded adequately to treatment
- ▶ Those whose headache features change with time

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### Migraine Mimics: when to consider

Note: Sometimes, there is more than a single diagnosis present (i.e. migraine PLUS another condition)

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### Evaluating a patient with headaches

- ▶ Identify and exclude secondary headache disorders
- ▶ History and examination to search for "red flags"

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Headache "red flags"

- ▶ Rapid onset of symptoms
- ▶ Thunderclap headaches
- ▶ Presence of neurologic symptoms and signs
- ▶ Prominent neck pain with and without fever
- ▶ Age of onset > 50 y/o
- ▶ Worsening with position or Valsalva's

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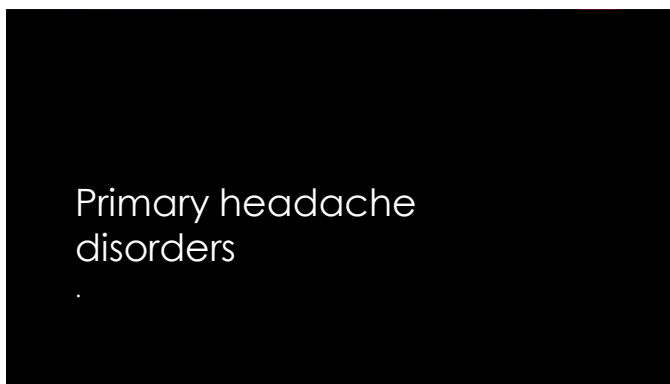
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Primary headache disorders

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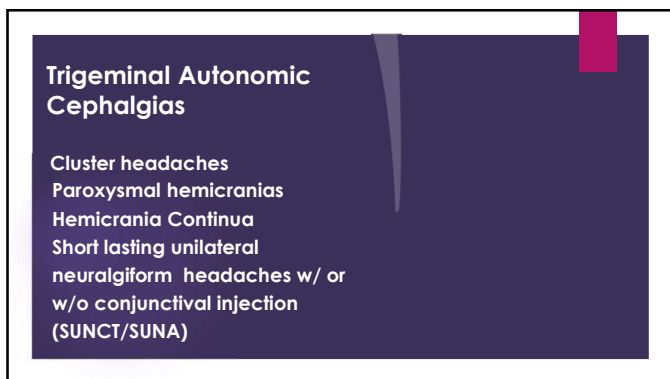
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Trigeminal Autonomic Cephalgias

Cluster headaches  
Paroxysmal hemicranias  
Hemicrania Continua  
Short lasting unilateral  
neuralgiform headaches w/ or  
w/o conjunctival injection  
(SUNCT/SUNA)

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### Trigeminal Autonomic Cephalgias

- ▶ Distinguished by attack frequency, attack duration and patterns of response to treatment
- ▶ Can be confused with migraines due to unilateral pain, overlap of associated symptoms and treatment response

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### TACs- Cluster headaches

- ▶ Characterized by severe unilateral pain associated with ipsilateral autonomic features and restlessness
- ▶ Attacks last 15 to 180 minutes
- ▶ Multiple attacks in a 24 hour period

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### TACs- Cluster Headaches

- ▶ Differentiating features from migraines
  - Shorter attack duration
  - Circannual and/or circadian pattern
  - Restlessness during an attack

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### TACs-- Hemicrania Continua

- ▶ As the name suggests, it is characterized by continuous headaches with exacerbations of pain

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### TACs-- Hemicrania Continua

- ▶ Can be confused with migraines especially if the history focuses on the painful exacerbations
- ▶ Differentiating features from migraines
  - Absence of pain free intervals
  - Absolute response to indomethacin

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## TACs– Paroxysmal Hemicrania

- ▶ Similar to cluster headache, it is a severe, episodic, unilateral headache that affects the periorbital and retroorbital regions.
- ▶ In contrast to cluster headache, which occurs 10 times more commonly in men, paroxysmal hemicrania occurs primarily in women
- ▶ Shorter duration and higher frequency of headaches compared to cluster
- ▶ Absolute response to indomethacin

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## TACs– Paroxysmal Hemicrania

- ▶ Differentiating features from migraines
  - Shorter attack duration
  - Multiple attacks in a 24 hour period
  - Absolute response to indomethacin

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## TACs– SUNCT/SUNA

- ▶ Characterized by moderate to severe single or multiple stabs of pain lasting from 1 to 600 seconds
- ▶ Can be differentiated from migraines by the very high frequency and very short duration of attack

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## Trigeminal Autonomic Cephalgias

	Cluster	Paroxysmal Hemicrania	SUNCT/SUNA	Hemicrania Continua
Sex Ratio F:M	1:3	2:1	1: 1.2	?
Quality of pain	Stabbing/sharp	Throbbing, piercing, stabbing	Burning, stabbing, cutting	Pressing, stabbing
Intensity of pain	Very severe	Very severe	Very severe	Moderate to severe
Location of pain	Orbital, temporal	Orbital, temporal	Periorbital	Nuchal to frontal
Frequency	Every 1-2 days or up to 8x a day	1-40/day	3-200/day	Continuous
Duration	15-180 min	2-30 min	5-240 sec	Daily, continuous

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## Trigeminal Autonomic Cephalgias

	Cluster Headache	Paroxysmal Hemicrania	SUNASUNCT	Hemicrania Continua
Autonomic symptoms	Present	Present	Present	Present but mild
Trigger: ETOH	Yes	Sometimes	No	No
Trigger: cutaneous stimuli	No	No	Yes	No
Response to indomethacin	No	Yes	No	Yes

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## New daily persistent headaches

- ▶ Persistent headache clearly remembered from onset
- ▶ Present for at least 3 months
- ▶ Notoriously difficult to treat
- ▶ Can exhibit migraine features
- ▶ Differentiated from migraines by absence of pain free periods from onset

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### Hypnic headaches

- ▶ Rare, occurs exclusively during sleep
- ▶ Dubbed as the "alarm clock headache" as it usually occurs at the same time at night
- ▶ Usually starts after the age of 50 years,

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### Hypnic headaches

- ▶ Unlike migraines, it never occurs during the daytime
- ▶ Not associated with any migrainous or autonomic symptoms

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### Secondary headache disorders

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## Arterial Dissection

- ▶ May be present with unilateral severe headaches with nausea and phobias
- ▶ Unlike migraines, pain is more rapid in onset and commonly associated with signs of cerebral ischemia and/or sympathetic compromise

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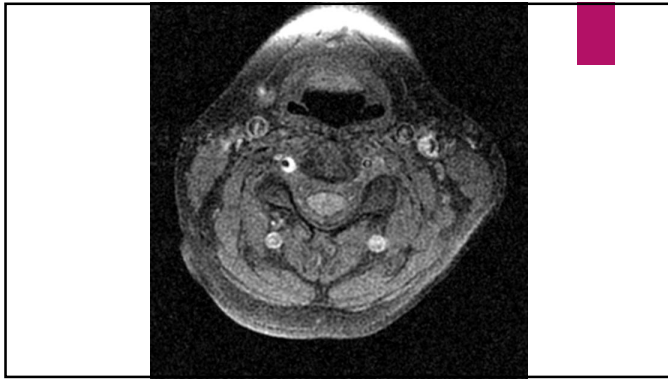
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## Giant cell arteritis (GCA)

- ▶ May be associated with visual loss (amaurosis fugax) that can be confused with migraine aura
- ▶ Unlike migraine, GCA is usually associated with systemic symptoms such as fever, weight loss, jaw claudication, temporal artery induration/tenderness

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## Giant cell arteritis (GCA)

- ▶ Usually affects people above 50 y.o
- ▶ Associated with elevated inflammatory markers i.e. ESR, CRP
- ▶ A headache NOT to miss! Can lead to irreversible visual loss and stroke

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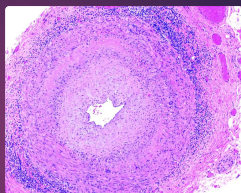
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Giant cell arteritis (GCA)

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## Stroke and vasculopathies

- ▶ Stroke, intracranial hemorrhage, venous thrombosis, reversible cerebral vasoconstriction syndrome may be associated with migraine features
- ▶ More commonly associated with focal neurological deficits and decrease in awareness compared to migraines

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## Hypertension

- ▶ Can present with migraine like headaches
- ▶ Typically associated with SBP > 1892 mm Hg and DBP > 110 mm Hg

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## Acute closed angle glaucoma

Can be differentiated from migraines by the presence of severe headache around one eye, associated with hardness and tenderness of the eye, blurred vision or visual loss, halos around objects and/or redness of the eye

- ▶ This is a medical emergency!

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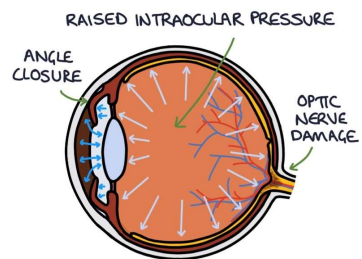
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## Acute closed angle glaucoma

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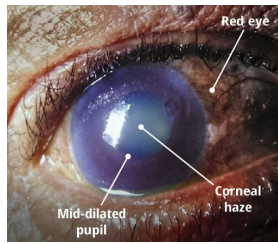
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## Acute angle closure glaucoma

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## Thunderclap headaches

- ▶ Warrants vigorous evaluation for a secondary cause such as subarachnoid hemorrhage, intracerebral hemorrhage, pituitary apoplexy, stroke, RCVS
- ▶ Develops to peak intensity in < 1 minute of onset
- ▶ After all life threatening conditions are ruled out, consider primary thunderclap headaches as a diagnosis

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## Transient ischemic attack

- ▶ Often confused for migraine aura and vice versa
- ▶ Inappropriate diagnosis of TIA in a patient with migraine can lead to extensive work-up and unnecessary use of antiplatelets
- ▶ Usually associated with vascular risk factors (HTN, HPL, CAD, etc) and lasts < 1 hour

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## CSF pressure related headaches

- ▶ Demographics for people with either migraine or intracranial hypertension (IIH) overlap significantly and these two conditions may be comorbid

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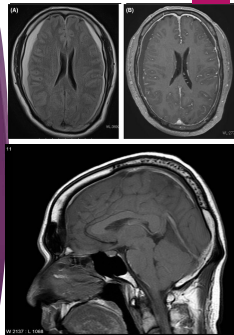
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## CSF pressure related headaches—intracranial hypotension

- ▶ Postural headaches or end of the day headaches may indicate CSF leak
- ▶ Opening pressure  $\leq 60$  mm H<sub>2</sub>O or less
- ▶ Sustained improvement after blood patch
- ▶ Cranial MRI changes i.e. brain sagging, pachymeningeal enhancement,



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## CSF pressure related headaches—intracranial hypertension

Presence of pulsatile tinnitus, visual obscurations, CN VI palsy and papilledema suggests IIH

Opening pressure  $> 25$  cm H<sub>2</sub>O



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## Headaches associated with neoplasms

- ▶ Migraine, nausea and vomiting can be a presenting feature
- ▶ Commonly precipitated by bending or Valsalva's
- ▶ Remain vigilant in patients with migraine whose symptoms worsen for unclear reasons

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## Headaches secondary to infections

- ▶ Meningitis-- frequently associated with headaches but commonly associated with nuchal rigidity, fever and altered mental status
- ▶ Sinusitis- nasal symptoms and facial pain are common in migraine but consider sinusitis in patients with nasal discharge, fever and halitosis

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## Sinus headaches

- ▶ Self-diagnosed sinus headache is nearly always migraine (90% of the time, American Migraine Study II)
- ▶ Migraine is commonly associated with forehead and facial pressure over the sinuses, nasal congestion and runny nose.
- ▶ Presence of fever, purulent nasal discharge, halitosis, alteration in smell are common

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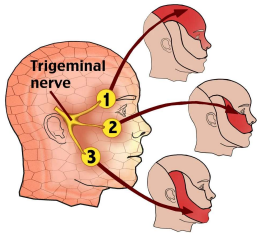
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## Sinus headache

► The first two branches of the trigeminal nerve—ophthalmic and maxillary—inervate the upper half of the face, including areas overlying the sinuses



The diagram shows a lateral view of a human head with the trigeminal nerve (CN V) highlighted in yellow. The nerve is labeled 'Trigeminal nerve'. Three branches are numbered: 1 (ophthalmic), 2 (maxillary), and 3 (mandibular). Red arrows indicate the distribution of these branches to the forehead, nose, cheek, and jaw areas, which are associated with the sinuses.

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## Cases

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## CASE # 1

- 48/F with daily constant headache, daily since onset 3 months ago, L sided pressure, 1/10 to 10/10 on the VAS associated with light and noise sensitivity
- No triggers
- Neurological exam, routine blood work and imaging studies of the brain are normal

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## CASE # 1

- ▶ What is the diagnosis? Any further work-up? How will you treat this patient

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## CASE # 2

- ▶ 53/F with new onset unilateral headaches in the last week, associated with fever, blurry vision and nausea. No precipitating factors or triggers identified.
- ▶ Neurological examination significant for tenderness on the left temple

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## CASE # 2

- ▶ What is the diagnosis?
- ▶ What tests will you order, if any?

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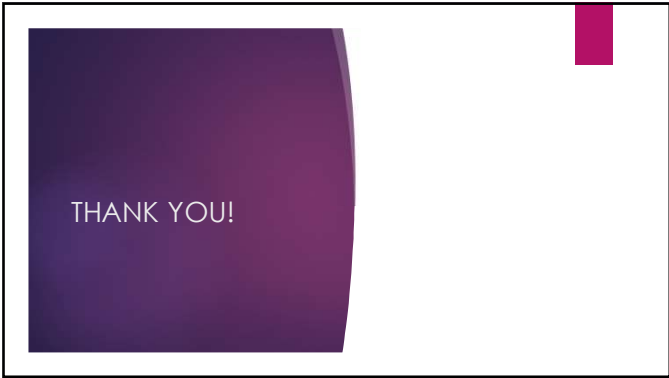
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