Welcome!
Pain Management

Pain is Inevitable.
Suffering is Optional.

CareOregon Pharmacy
Today’s Agenda

• Welcome and Introduction – 8:00
• Chronic Pain 101– 8:05
• Medication Review – 8:55
• Break – 9:30
• Difficult Conversations– 9:45
• New in 2016 – 10:45
• Questions – 11:00
• Closing – 11:20
Oregon Health Providers Make It Harder To Get Opioids

Objectives

1. Understand the pathophysiology of pain and neurobiologic pain mechanisms.
2. Describe the impact of environment and life experiences on the patient’s pain management.
3. Summarize non-pharmacologic, behavioral, and pharmacologic treatments for chronic pain.
4. Identify key medication problems with a patient’s pain management regimen.
5. Gain concrete tools to address difficult conversations with patients about pain management.
Chronic Pain 101

New in 2016

Difficult Conversations

Bridging the Gap

Treatment Options

Medication Management
Chronic Pain 101: Sensation, Emotion and Reward

Rachel Solotaroff, MD, MCR
Central City Concern
Chair, CareOregon Metro
Chronic Pain/Chemical Dependency Task Force
Objectives

• Understand neurobiology and underlying mechanisms of sensory aspects of pain
• Provide examples of pharmacologic, behavioral and activity-based therapies to address pain
• Present a new paradigm for understanding pain based on emotion, reward, and interaction with addiction
Disclosures

• I have no financial disclosures

• I believe in embracing the complexity of chronic pain
Understanding Sensory Pain
Understanding Pain in Less than 5 minutes!

https://www.youtube.com/watch?v=gy5yKbduGkc
What is “Brain Pain?”

- Pain **not** from a hip, a knee, an abdomen
- Evolution of terms:
  - “Spastic colitis” → Irritable Bowel Syndrome
  - “Fibrositis” → Fibromyalgia
  - “Interstitial Cystitis” → Painful Bladder Syndrome
- Different treatments for Brain Pain than acute pain or true peripheral pain
- Namely, opioids, injections, surgical procedures do not work in brain amplification syndromes
Which Person Has Pain?
## Mechanistic Characterization of Pain

**Any combination may be present in a given individual**

<table>
<thead>
<tr>
<th>Peripheral (nociceptive)</th>
<th>Peripheral Neuropathic</th>
<th>Central Neuropathic or Centralized Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inflammation or mechanical damage in tissues</td>
<td>Damage or dysfunction of peripheral nerves</td>
<td>Characterized by central disturbance in pain processing</td>
</tr>
<tr>
<td>NSAID, opioid-responsive</td>
<td>Responds to both peripheral and central drugs (TCA’s, neuroactive compounds)</td>
<td>Responsive to neuroactive compounds altering levels of neurotransmitters involved in pain transmission and rehab</td>
</tr>
<tr>
<td>Responds to procedures</td>
<td>May respond if pain is localized to specific nerve</td>
<td>No response</td>
</tr>
</tbody>
</table>
| Classic examples:  
- Acute pain due to injury  
- Osteoarthritis  
- Rheumatoid arthritis  
- Cancer pain | Classic examples:  
- Diabetic neuropathic pain  
- Post-herpetic neuralgia | Classic examples:  
- Fibromyalgia  
- IBS  
- TMJD  
- Tension headache |
How Do You Identify Brain Pain?

• Involves more areas of the body, “multi-focal”
• Flares are not predictable
• Use different descriptors, hard to localize, sound “nerve” like
• Higher current and lifetime history of pain
• Accompanied by:
  – Fatigue
  – Memory problems
  – Sleep disturbances
  – Mood disturbances
  – Same neurotransmitters as “volume control”
• Sensitive to multiple sensory stimuli
Other Clinical Characteristics of Central Pain

• 1.5 – 2x more common in women
• Strong genetic predisposition (take a family hx)
• Can be triggered or exacerbated by stressors:
  – Early life stressors
  – War (but not natural disasters)
  – Infections (Lyme, Epstein Barr)
  – Environmental (mold)
  – Major surgical procedures
  – Peripheral pain syndromes (eg. RA, SLE OA)
  – Psychological stress/distress

• Generally normal physical exam except for diffuse tenderness
Central Pain on a Continuum

• Not “yes” or “no” – occurs over a wide distribution on a bell-shaped curve

• Diagnostic labels largely historical and irrelevant
  – Fibromyalgia (FM)
  – Irritable Bowel Syndrome (IBS)

• “Fibromyalgia-ness” predicts pain intensity, symptoms and disability over a wide range of rheumatic disorders
  – RA, OA, regional musculoskeletal pain, FM
Central Pain on a Continuum

Proportion of individuals in chronic pain states that have centralized their pain

Peripheral

Acute Pain
Osteoarthritis
Rheumatoid Arthritis

Centralized

SC Disease
Ehler’s Danlos
Low Back Pain
Fibromyalgia
Tension HA
IBS
Treating Sensory Pain
Neurotransmitter Targets in Central Pain

Facilitation
- Substance P
- Glutamate and excitatory amino acids
- Serotonin (5HT$_{2a,3a}$)
- Nerve growth factor

Inhibition
- Descending antinociceptive pathways
  - Norepinephrine-serotonin (5HT$_{1a,b}$), dopamine
  - Opioids
    - GABA
    - Cannabinoids
Drugs to Treat Chronic Pain

“Rule of 30% and 50%”:
- Our best drugs work marginally well in 50% of people, fairly well in 30% of people
- 50% of people will fail to respond at all, or have side effects

<table>
<thead>
<tr>
<th>Strong Evidence</th>
<th>Moderate Evidence</th>
<th>Weak Evidence</th>
<th>No Evidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Dual reuptake inhibitors such as Tricyclic compounds (amitriptyline, cyclobenzaprine) and SNRI’s and NSRI’s (minacipran, duloxetine, venlafaxine)</td>
<td>• Tramadol</td>
<td>• Cannabinoids for non-neuropathic pain</td>
<td>• Opioids, corticosteroids, NSAIDS, benzodiazepine and non-benzodiazepine hypnotics</td>
</tr>
<tr>
<td></td>
<td>• Anticonvulsants (e.g. pregabalin, gabapentin)</td>
<td>• Other less selective SSRI’s or NRIs</td>
<td></td>
</tr>
</tbody>
</table>
Downstream Consequences of Pain

Symptoms of Pain, Fatigue, etc.
- Nociceptive processes (damage or inflammation of tissues)
- Disordered Sensory Processing

Functional Consequences:
- Increased distress
- Decreased activity
- Isolation
- Poor sleep
- Maladaptive Illness Behaviors

Dually Focused Treatment
- Pharmacologic therapies to improve symptoms
- Non-pharmacologic therapies to address dysfunction
Psychotherapeutic Approaches

• Cognitive Behavioral Therapy:
  – What we choose to think about has an impact on our behaviors
  – Thoughts, behaviors and mood interact
  – Can use skills like reality testing, examining thoughts to change behaviors
  – Challenges negative thoughts

• Acceptance and Commitment Therapy:
  – Doesn’t challenge negative thoughts, as much as produces awareness of present
  – Avoid thoughts of what has happened /what will happen to me
  – Become more centered on what we have right now
  – Aware of what you have without judgement
  – Strategies/commitments of what to do with the present
Behavioral Approaches

- Energy Conservation
- Pacing
- Pleasurable Activity Scheduling
- Activity → Exercise
- Sleep Hygiene
Understanding Emotion and Reward in Chronic Pain (and Addiction)
Understanding Reward and Emotion in Chronic Pain

Reward learning processes may contribute to persistence and amplification of pain.
Review of the Neural Circuits of Addiction

Koob, CSAM Addiction Medicine Review Course, 2014
Neural Circuits of the Binge/Intoxication Stage

- Prefrontal Cortex
- Dorsal Striatum
- Nucleus accumbens
- Orbitofrontal Cortex

**Incentive Salience**
- euphoria
- intoxication
- cue learning
- habits

**Binge/Intoxication**
- Withdrawal/Negative Affect
- Preoccupation/Anticipation

**Future targets**
- partial agonists (intoxication blockers)
- drug vaccines (intoxication blockers)

Koob GF, Volkow ND. *Neuropsychopharmacol REV*, 2010, 35:217-238
Converging Acute Actions of Drugs of Abuse on the Ventral Tegmental Area and Nucleus Accumbens

Neural Circuits of the Withdrawal/Negative Affect Stage

**Negative Affect**
- dysphoria
- anxiety
- irritability
- malaise

**Binge/Intoxication**
**Withdrawal/Negative Affect**
**Preoccupation/Anticipation**

**Neurobiological targets**
- GABA modulators (homeostatic resetters)
- CRF₁ antagonists (stress reducers)
- κ opioid antagonists (dysphoria reducer)

Koob, CSAM Addiction Medicine Review Course, 2014
Conceptual Model of Alcohol/Drug Dependence

# Reward Transmitters Implicated in the Motivational Effects of Drugs of Abuse

<table>
<thead>
<tr>
<th>Positive Hedonic Effects</th>
<th>Negative Hedonic Effects of Withdrawal</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Dopamine</strong></td>
<td>Dopamine – “dysphoria”</td>
</tr>
<tr>
<td><strong>Opioid Peptides</strong></td>
<td>Opioid Peptides – pain</td>
</tr>
<tr>
<td><strong>Serotonin</strong></td>
<td>Serotonin – “dysphoria”</td>
</tr>
<tr>
<td><strong>GABA</strong></td>
<td>GABA – anxiety, panic attacks</td>
</tr>
</tbody>
</table>

Koob, CSAM Addiction Medicine Review Course, 2012
Anti-Reward Transmitters Implicated in the Motivation Effects of Drugs of Abuse

<table>
<thead>
<tr>
<th>Positive Hedonic Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dynorphin – “dysphoria”</td>
</tr>
<tr>
<td>Corticotropin-Releasing Factor (CRF) – stress</td>
</tr>
<tr>
<td>Norepinephrine – stress</td>
</tr>
<tr>
<td>These are ACTIVATED in amygdale and ventral striatum during withdrawal</td>
</tr>
</tbody>
</table>

Koob, CSAM Addiction Medicine Review Course, 2012
The Reward System in Pain

What does the reward system do? A Quick Decision-Making Process:

- Dopamine neurons from Ventral Tegmental Area estimate value of reward/relief-seeking
- Nucleus Accumbens (NAc) neurons decide whether to initiate reward/relief-seeking behavior then initiate reward/relief-seeking habits based on decision
- Frontal cortex also receives information, can inhibit NAc, but slowly

Dopamine Pathways

Serotonin Pathways

Functions
- Reward (motivation)
- Pleasure, euphoria
- Motor function (fine tuning)
- Compulsion
- Perseveration

Functions
- Mood
- Memory processing
- Sleep
- Cognition
So What’s the Problem?

- Addictive drugs and search for pain relief can dump tons of dopamine into these circuits
- Addictive drugs increase activity in these neurons, or prolong actions of neurotransmitters they release
- New research show pain relief activates these neurons to drive habitual relief seeking
An Example:

“I’m going to lie down on the couch.”

- Back pain gets better, and your brain listens: “I got reward!”
- Your brain will refer that relief back to the laying down, and learn lying down as new context – as new opportunity to get relief.
- When pain relief was present, your reward learning circuit turned on.
What Happens Over Time?

**Accelerator:**
D1 receptor

**Brakes:**
D2 receptor

**D1 Receptors:**
Dopamine in the receptors tells Nucleus Accumbens to say “Yes!”

**D2 Receptors:**
Activation of these receptors slows decision-making; allows frontal cortex time to step in
Too Much Accelerator is a Bad Thing

- When DA neurons are chronically over-active, they activate D1 receptors to:
  - Activate anti-reward circuits that increase stress response and worsen mood – both amplify pain signals
  - Desensitize and down-regulate D2, eliminating the brakes
  - Sensitize D1 pathways, speeding up decisions to seek relief
  - Pain severity increases and relief-seeking behaviors become compulsive
What Happened to the Brakes?

• What happens to D2?
• If you hit them hard with a lot of dopamine at once, they are desensitized
• D2 Receptor can’t work again until it is recycled (takes an hour) or until a new receptor is synthesized
• You may ultimately wind up with a system that has no brakes
Inflammatory Injury Model

- All about elevated dopamine
- Patients with elevated dopamine are more likely to develop chronic pain
  - Acute injury $\rightarrow$ chronic pain
  - Drugs that increase dopamine $\rightarrow$ chronic pain
  - Smokers and people given opioids for an acute injury $\rightarrow$ chronic pain even after injury heals
How to Get off the Couch

• Get your brakes back on
• Need just enough DA to activate D2 receptors, get some inhibition but not knock them out
• Consistent low level DA input to build back inhibition
• Lots of tiny opportunities for little reward: make someone smile, enjoy sunshine
• The tiny things in life are what make life good, and allow D2 receptors give your brain time to make a choice.
Implications for Treatment

In both chronic pain and addiction, same healing process:

• Reduce exposure to huge dopamine signals:
  – Limit use of addictive drugs or medications, junk food, fast-acting analgesics, tobacco
  – Prevent desensitization of D2 pathway

• Increase exposure to small rewards:
  – Social reinforcement, problem-solving, effective emotional coping, small goal achievement
  – Increase activity of D2 pathway
Take Home Points

• Central pain:
  – Affects ~30% population
  – May be treated with behavioral and pharmacologic approaches
  – Opioids generally make this condition worse

• Treating behavioral health conditions in people with chronic pain:
  – Is important – keep treating them!
  – Won’t solve the underlying pain problem
Take Home Points (continued)

• Be thoughtful in assessment and diagnosis to identify conditions that are opioid-responsive

• Addiction and chronic pain have common neural circuitry → treatments are similar
Acknowledgements

• Daniel Clauw, MD
• Jodie Trafton, PhD
Please hold questions – thanks!
Pain Medications: Dr. Feelgood or Bad Medicine?

Tara Berkson, PharmD
MEDS Ed Coordinator
CareOregon
Dr. Feelgood or Bad Medicine?

The New Yorker

Letter from Wichita | May 5, 2014 Issue

Prescription for Disaster

The heartland’s pain-pills problem.
Take Home Points

• What are our patients learning about pain medications from the media?
• What evidence exists about the benefits of pain medications?
• What are the top risks of chronic pain medication use?
## Treatment Recommendations

<table>
<thead>
<tr>
<th></th>
<th>Osteoarthritis</th>
<th>Somatic Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1st line</strong></td>
<td>Acetaminophen (APAP) NSAIDs</td>
<td>APAP NSAIDs</td>
</tr>
<tr>
<td><strong>2nd line</strong></td>
<td>Topical analgesics Intra-articular analgesics</td>
<td>Anticonvulsants TCAs</td>
</tr>
<tr>
<td><strong>3rd line</strong></td>
<td>Opioids</td>
<td>Cyclobenzaprine Opioids: short-term</td>
</tr>
</tbody>
</table>
## Treatment Recommendations cont

<table>
<thead>
<tr>
<th></th>
<th>Neuropathic Pain</th>
<th>Fibromyalgia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1ˢᵗ line</strong></td>
<td>TCAs</td>
<td>APAP</td>
</tr>
<tr>
<td></td>
<td>SNRIs</td>
<td>TCAs</td>
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<tr>
<td></td>
<td>Gabapentin, pregabalin</td>
<td>SNRIs</td>
</tr>
<tr>
<td></td>
<td>Topical analgesics</td>
<td>Cyclobenzaprine</td>
</tr>
<tr>
<td><strong>2ⁿᵈ line</strong></td>
<td>Carbamazepine</td>
<td>Gabapentin, Pregabalin</td>
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<tr>
<td></td>
<td>Oxcarbazepine</td>
<td>Older SSRIs</td>
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<tr>
<td></td>
<td>Oxcarbazepine</td>
<td>Tramadol</td>
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<tr>
<td></td>
<td>Topiramate</td>
<td></td>
</tr>
<tr>
<td><strong>3ʳᵈ line</strong></td>
<td>Opioids</td>
<td></td>
</tr>
<tr>
<td><strong>Not Recommended</strong></td>
<td></td>
<td>Opioids NSAIDs</td>
</tr>
</tbody>
</table>

TCAs: Tricyclic Antidepressants
SNRIs: Serotonin-Norepinephrine Reuptake Inhibitors
NSAIDs: Non-Steroidal Anti-Inflammatory Drugs
### Acetaminophen (Tylenol)

<table>
<thead>
<tr>
<th>The Good</th>
<th>The Bad</th>
<th>The Ugly</th>
</tr>
</thead>
<tbody>
<tr>
<td>• OTC pain relief</td>
<td>• Not an anti-inflammatory</td>
<td>• Only 2-3 extra tablets per day may cause liver injury</td>
</tr>
<tr>
<td>• Fever reduction</td>
<td>• Hard to keep track of doses - in multiple combination products</td>
<td>• Boxed warning</td>
</tr>
<tr>
<td>• Appropriate for children</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Few drug interactions</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Minimal GI upset</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Helps opioid effectiveness</td>
<td></td>
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</tr>
</tbody>
</table>

Maximum dose: 4g per day or 12 regular strength tablets
Nonsteroidal Anti-Inflammatory Drugs

First line for arthritis, inflammation

- Side Effects: GI upset, dizziness
- Caution: renal dysfunction, CV disease, heart failure

<table>
<thead>
<tr>
<th>Generic</th>
<th>Brand</th>
</tr>
</thead>
<tbody>
<tr>
<td>Celecoxib</td>
<td>Celebrex</td>
</tr>
<tr>
<td>Diclofenac</td>
<td>Cataflam, Voltaren (topical)</td>
</tr>
<tr>
<td>Etodolac</td>
<td>Lodine</td>
</tr>
<tr>
<td>Ibuprofen</td>
<td>Motrin, Advil</td>
</tr>
<tr>
<td>Indomethacin</td>
<td>Indocin</td>
</tr>
<tr>
<td>Ketorolac</td>
<td>Toradol</td>
</tr>
<tr>
<td>Meloxicam</td>
<td>Mobic</td>
</tr>
<tr>
<td>Naproxen</td>
<td>Naprosyn</td>
</tr>
</tbody>
</table>
NSAID Selectivity

Physiological stimuli

- Prostaglandin$_1$
- Prostaglandin$_2$

- Stomach mucosa
- Platelet stickiness

Injury, infection, inflammatory stimuli

- Prostaglandin$_3$
- Prostaglandin$_4$

- Pain
- Inflammation

COX1

- COX1 inhibitor

COX2

- COX2 inhibitor

- COX inhibitor
Are We Still Talking About CV Risk?
Antidepressants

Drugs that affect serotonin (5-HT) and norepinephrine (NE) receptors

<table>
<thead>
<tr>
<th>Older SSRIs</th>
<th>SNRIs</th>
<th>TCAs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluoxetine (Prozac)</td>
<td>Duloxetine (Cymbalta)</td>
<td>Amitriptyline (Elavil)</td>
</tr>
<tr>
<td>Sertraline (Zoloft)</td>
<td>Milnacipran (Savella)</td>
<td>Desipramine</td>
</tr>
<tr>
<td>Paroxetine (Paxil)</td>
<td>Venlafaxine (Effexor XR)</td>
<td>Nortriptyline (Pamelor)</td>
</tr>
<tr>
<td></td>
<td>Desvenlafaxine (Pristiq)</td>
<td></td>
</tr>
</tbody>
</table>
FDA Indications

• Duloxetine: diabetic neuropathy, fibromyalgia, chronic pain
• Milnacipran: fibromyalgia
• Venlafaxine: diabetic neuropathy, cancer pain, migraine/HA prophylaxis
Side Effects

- SSRIs: nausea, vomiting, diarrhea, sexual dysfunction, headache
- SNRIs: nausea, sexual dysfunction, increase in diastolic blood pressure, sweating
- TCAs: sedation, dry mouth, weight gain, constipation, blurred vision
<table>
<thead>
<tr>
<th>Dr. Feelgood</th>
<th>Bad Medicine</th>
</tr>
</thead>
<tbody>
<tr>
<td>FDA indications = more evidence</td>
<td>Placebo-controlled, Short term trials</td>
</tr>
<tr>
<td>May increase dose of older SSRIs</td>
<td>SNRIs/TCAs: higher doses = more side effects instead of switching drugs</td>
</tr>
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</tbody>
</table>
Brain Break

True or False

Gabapentin and pregabalin work on the GABA receptors in the brain.
Anticonvulsants for Pain

- Gabapentin (Neurontin, Gralise) & pregabalin (Lyrica)

- FDA indications:
  - Gabapentin: postherpetic neuralgia
  - Pregabalin: neuropathic pain, fibromyalgia, postherpetic neuralgia

- Side Effects: nausea, vomiting, fatigue, dizziness, dry mouth

- Titrate slowly

- Do not stop suddenly
Other Anticonvulsants

• Carbamazepine, oxcarbazepine: trigeminal neuralgia
  – Side Effects: nausea, constipation, dry mouth, rash

• Topiramate: migraine prophylaxis
  – Side Effects: weight loss, dizziness, memory impairment (“Dope-amax”)

<table>
<thead>
<tr>
<th>Generic</th>
<th>Brand</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbamazepine</td>
<td>Tegretol</td>
</tr>
<tr>
<td>Oxcarbazepine</td>
<td>Trileptal</td>
</tr>
<tr>
<td>Topiramate</td>
<td>Topamax</td>
</tr>
</tbody>
</table>
“I Don’t Have Seizures”

- Patient education is key for drugs with multiple indications
- “What did your doctor tell you this was for?”
OTC Topicals

• Capsaicin: desensitizes sensory nerves over time
  – Caution when applying- wear gloves, wash hands
  – Use 3-4 times per day on affected area
  – May cause burning sensation which fades over time

• Menthol/methyl salicylate (Bengay cream, Salonpas patches)
  – Don’t apply heat to the area
  – Menthol causes cooling sensation
Prescription Topicals

• Lidocaine patch:
  – Wear for 12 hours, remove for 12 hours
  – May cut patches

• Diclofenac (Voltaren):
  – Apply up to 4 times daily
  – Do not apply heat or cover area with bandages
  – Carries same Black Box warning as oral NSAIDs
Skeletal Muscle Relaxants

- Work in CNS to relax muscles
- Side Effects: dizziness, drowsiness, dry mouth, confusion, GI
- Cyclobenzaprine: evidence in fibromyalgia

<table>
<thead>
<tr>
<th>Generic</th>
<th>Brand</th>
</tr>
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<tbody>
<tr>
<td>Baclofen</td>
<td>Lioresal</td>
</tr>
<tr>
<td>Carisoprodol</td>
<td>Soma</td>
</tr>
<tr>
<td>Cyclobenzaprine</td>
<td>Flexeril</td>
</tr>
<tr>
<td>Methocarbamol</td>
<td>Robaxin</td>
</tr>
<tr>
<td>Tizanidine</td>
<td>Zanaflex</td>
</tr>
</tbody>
</table>
Opioids, Opiates, Oh My!

“A group of middle-aged whites in the U.S. is dying at a startling rate”

-Washington Post, 11/2/15
## Opioid Receptor Subtypes

<table>
<thead>
<tr>
<th>Receptor Subtype</th>
<th>Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mu1</td>
<td>Euphoria, supraspinal analgesia, confusion, dizziness, nausea</td>
</tr>
<tr>
<td>Mu2</td>
<td>Respiratory depression, CV effects, GI effects, miosis, urinary retention</td>
</tr>
<tr>
<td>Delta</td>
<td>Spinal analgesia, CV depression, decreased brain and myocardial oxygen demand</td>
</tr>
<tr>
<td>Kappa</td>
<td>Spinal analgesia, dysphoria, psychomimetic effects, feedback inhibition of endorphin system</td>
</tr>
</tbody>
</table>
Opioid Analgesics

- All Schedule II except:
  - Tramadol (Sched IV)
  - < 90mg codeine
- Limit 30 day supply with no refills

<table>
<thead>
<tr>
<th>Generic</th>
<th>Brand</th>
</tr>
</thead>
<tbody>
<tr>
<td>Codeine</td>
<td></td>
</tr>
<tr>
<td>Fentanyl</td>
<td>Duragesic</td>
</tr>
<tr>
<td>Hydrocodone</td>
<td>Vicodin, Norco, Lortab</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>Dilaudid</td>
</tr>
<tr>
<td>Meperidine</td>
<td>Demerol</td>
</tr>
<tr>
<td>Methadone</td>
<td>Dolophine</td>
</tr>
<tr>
<td>Morphine</td>
<td>MS Contin, Kadian, Oramorph</td>
</tr>
<tr>
<td>Oxycodone</td>
<td>Oxycontin, Percocet</td>
</tr>
<tr>
<td>Oxymorphone</td>
<td>Opana</td>
</tr>
<tr>
<td>Tramadol</td>
<td>Ultram</td>
</tr>
</tbody>
</table>
Don’t Be “Naïve”

- Opioid-naïve:
  - < 60mg daily morphine equivalent dose (MED)
  - < 1 week therapy
- Start low and go slow
- Reserve long-acting opioids for around the clock pain management
  - Oxycodone CR, MS Contin, Methadone, Fentanyl Patch
- Watch total APAP daily dose
  - Vicodin, Percocet: 325mg APAP
Onset and Duration of Action

• Short-acting or immediate release
  – Hydrocodone, hydromorphone, morphine, oxycodone, oxymorphone
  – Onset: ~15 to 60 min
  – Duration: 4 to 6 hours

• Long-acting or extended/controlled release
  – Oxycodone CR, MS Contin, Methadone, Fentanyl Patch
  – Onset: ~30 to 90 min
  – Duration: 8 to 12 hours (oral), 72 hours (Fentanyl)
Side Effects
How to Manage Constipation

• Most common side effect
• Does not fade over time
• “Push and Mush” treatment
  – Laxative (senna) + stool softener (docusate)
• Naloxegol (Movantik): new Mu receptor antagonist
  – Does not cross blood-brain barrier
  – Placebo-controlled trials
  – No evidence with conventional treatment

"It takes Mu to tango."
Tolerance to Side Effects

• Nausea/vomiting
  – Most patients develop tolerance
  – Treat with ondansetron or prochlorperazine PRN

• Itching
  – Most patients develop tolerance
  – Cautiously consider diphenhydramine PRN

• Sedation
  – Most patients develop tolerance
  – Decrease opioid dose or change opioids
Pop Quiz

What is the source of the following information?

“Prescriptions for [opioid] drugs have climbed 300 percent in the last decade or so. In fact, Vicodin and other hydrocodone-combination painkillers are the most commonly prescribed drugs in the U.S.”
Patient Education

• Don’t drive or operate heavy machinery
• Don’t drink alcohol
• Difficulty breathing → call doctor or go to ER
• Fentanyl patch
  – Remove old patch before applying new one
  – Apply patch intact (do not cut)
  – May use tape around the edges of patch to hold in place
  – Do not apply heat
  – To discard, fold in half and flush down the toilet

Take Control of Your Pain
Become Educated and Empowered
Higher Risk Patients

- Sleep apnea
- Kidney dysfunction
- Use of CNS depressants → benzodiazepines, sedatives
- History of alcohol or drug abuse
Opioid Conversion

• General method
  1. Calculate 24-hr opioid requirement (ER + IR)
  2. Convert to equianalgesic oral dose using opioid conversion chart or calculator
  3. Consider 50% dose decrease for cross-tolerance

• Methadone and Fentanyl have separate conversion tools
New Formulations

• Zohydro ER: long-acting hydrocodone
  – No abuse deterrent
• Hysingla ER: long-acting hydrocodone
  – Forms thick gel if crushed
• Targiniq ER: oxycodone + naloxone
Dependence vs Addiction

- **Dependence**: stopping the drug will cause physical withdrawal; body has adapted to having drug around
  - Physiologic response
  - Anxiety, hypertension, intense pain, diarrhea

- **Addiction**: cravings or compulsive drug use despite harm or negative effect on life
PDMP

- Prescription Drug Monitoring Program
- **Oregon** pharmacies submit prescription data for controlled substances within 72 hours
- Healthcare providers may apply for access limited to patients under their care
Patient Case

SR is a 27-year old female who recently underwent ORIF surgery for a fractured left ankle during a roller derby bout.

Allergies: NKDA
PMH: not significant
Medications: APAP 650mg PRN for pain after roller derby practices

Which of the following is an appropriate post-op pain management regimen for SR?

A. Fentanyl patch 50mcg every 72 hours
B. Oxycontin 30mg every 12 hours + Oxycodone IR 15mg every 4 hours PRN
C. Hydrocodone/APAP 5mg/325mg every 4 hours PRN
D. None of the above
Tapering Opioids

• Slower taper for long-acting opioids & stable patient
• Faster taper for short-acting opioids & less stable patient
• Short-acting opioids $\rightarrow$ 10% every 3 days
• Long-acting opioids:
  – 10% of initial total dose until at 30% of initial dose
  – Then 10% of current dose
• Manage symptoms with antidepressants, NSAIDs, clonidine, anti-nausea, anti-diarrhea
Take Home Points

• Opioids are last resort options for chronic, non-cancer pain
• Low doses, short courses
• “Don’t be naïve about opioid-naïve patients”
• Ensure side effects are appropriately managed
Please hold questions – thanks!
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Break
Chronic Pain: Bridging the Gap

- Difficult Conversations
- New in 2016
- Chronic Pain 101
- Treatment Options
- Medication Management
Bridging the Gap: How Values and Validation Support Change

Nadejda Razi-Robertson, LCSW
Behavioral Health Consultant
BRIDGING THE GAP: HOW VALUES AND VALIDATION SUPPORT CHANGE
CareOregon Metro’s Approach to Pain Management and Substance Use Disorders

Rachel Solotaroff, MD, MCR
Central City Concern
Chair, CareOregon Metro Chronic Pain/Chemical Dependency Task Force
Overview

• Goal of CPCD Task Force is to formulate interventions to support populations with chronic pain, substance use disorders, or both.
• Theme: no single intervention could be called upon as “the solution”; instead, focus on engaging patients at their own stage of change and readiness.
• Focus on patient- and population-centered care.
• Spirit of the recommendations is that positive patient experiences and improved clinical outcomes will be accomplished by providing low barrier, compassionate, relationship-based interventions that exist in a seamless care continuum.
4 Components of Recommendations

1. Design and implement chronic pain care management initiative for primary care clinics
2. Expand access to Medication Assisted Treatment (buprenorphine and Naltrexone XR) in primary care and specialty addictions settings.
3. Expand access to chronic pain supportive and specialty services, including acupuncture
4. Partner with Health Share of Oregon to continue and enhance CME offerings for chronic pain and addiction.
Chronic Pain Care Management Model

Referral Criteria:
- High BH complexity
- Unmet functional goals
- Illicit Drug Use

Phase I: Foundation
- Patient Registry
- Clinical Toolbox
- CME/Collaborative

Phase II: Develop Care Management Capacity
- Care Plan Template & Pain Pathway
- Identify & train Care Manager
- Guidelines & workflows for inter-disciplinary care
- Quality plan & measures
- Framework for Opioid Oversight Committee

Phase III: Deliver Quality
- Implement chronic pain care management
- Ongoing learning & development
- Expand target population

Provide Usual Primary Care
Team Members

- **Care Manager (1 FTE)** – focuses on identifying patients via registry, outreach and engagement with patient, co-development of care plan in partnership with patient and team, and ongoing coaching/support to patient. Strong emphasis on building patient’s motivation to engage in program.

- **Primary Care Champion (0.1 FTE)** – expands skill level in knowledge of chronic pain neurobiology, treatments, evidence of opioid harms. Serves as champion and thought leader of these concepts in primary care clinic. Staffs inter-disciplinary care team meetings, communicating with other providers regarding patients and care plans.

- **Behavioral health Clinician (0.25 FTE)** – provides evidence-based therapies to support patient in psychosocial management of pain, shift in focus from pain to function, readiness for other treatments (such as physical therapy or acupuncture), and crisis/anxiety management regarding issues such as pain flares and fear of decreased opioid dosages.

- **Pharmacist (0.1 FTE)** – assists care team with difficult questions surrounding medication tapers, conversions, and interactions.
Next Session

February 25th, 2016
Questions?
Summary

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Thank you!