Impact of Marijuana Use on Patient Care: From Recreation to Reconciliation

CPFI 2018 Conference
Bonclarken Conference Center
Flat Rock, North Carolina (2)

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PLUG INTO THE POWER OF PRAYER
Disclaimer

- I have no financial interest or direct affiliation with any company or organization involved with medical or recreational marijuana or hemp products.

- This is an educational program. Please consult with your PCP for medical advice.
Objectives

- Discuss legal landscape and product quality-related issues regarding recreational marijuana
- Review significant marijuana side effects and common drug interactions
- Describe ‘at risk’ patient populations using recreational marijuana case scenarios
- Review the pharmacist-led hospital admission MedRec process & marijuana policy development for health systems
# International MJ Legislation

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<tr>
<th>Argentina</th>
<th>Canada</th>
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<td>Czech Republic</td>
<td>India</td>
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<td>Ecuador</td>
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<td>Jamaica</td>
<td>Spain</td>
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<td>Mexico</td>
<td>Uruguay</td>
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States/Territories Permitting Recreational Marijuana Use

- Colorado (2012)
- Washington (2012)
- Alaska (2014)
- District of Columbia (2014)
- Oregon (2014)
- California (2016)
- Massachusetts (2017)
- Nevada (2017)
- Maine (2017)
- Vermont (2018)
2. California

State Marijuana Laws

**Ballot Measure:** Proposition 64: *The Adult Use Marijuana Act* – Approved Nov. 9, 2016 by 57% of voters

**Law:** *Control, Regulate and Tax Adult Use of Marijuana Act*

**State Website:** [California Cannabis Portal](https://www.cannabis.ca.gov)

**Effective:** Nov. 9, 2016 (revised penalties); Jan. 1, 2018 (retail sales); 2023 (restrictions to be lifted on large-scale corporations)

**California Medical Marijuana Laws:**
Medical cannabis patients are not subject to the limits of the recreational law and may possess up to 8 oz usable marijuana, and 6 mature or 12 immature plants.

Possession and Cultivation Limits

**Age:** 21+

**Usable Marijuana:** up to 1 oz

**Plants:** up to 6 plants, including the harvest from those 6 plants

**Hash & Concentrates:** up to 8 g (more than 8 g is a misdemeanor)
Oregon Recreational Marijuana Retail Sale Limits

A retailer may not sell more than the following amounts to a recreational customer at any one time or within one day:

- 1 oz of usable marijuana
- 5 grams of cannabinoid extracts or concentrates
- 16 oz of a cannabinoid product in solid form
- 72 fluid oz of a cannabinoid product in liquid form
- 10 marijuana seeds
- 4 immature marijuana plants
Introduction to Cannabis

- *Cannabis indica* and *Cannabis sativa* are the best-known species.

- A product's chemical profile is more important than the strain of plant from which it originated.

- Products should be characterized by analytical chemistry - percentages of cannabinoids and terpenoids.
Common Modes of Administration and Formulations

- Inhalation by smoking or vaporization
  - (herbal cannabis, resin, concentrates)
- Oral
  - (prescription cannabinoids, edibles, tinctures)
- Oro-mucosal or sublingual
  - (lollipops, lozenges, nabiximols)
- Topical or Rectal
  - (herbal cannabis, resin, concentrates)
Δ-9-THC Detection

- **Serum** - Active THC (cannabinoids) (positive at 20 ng/mL)
  MedTox Immunochromatographic test

- **Urine** - Inactive THC-COOH-glucuronide (positive at 50 ng/mL). Answers, “has this person used cannabis over the last days or weeks?”

Levels of THC or metabolites do not correlate with efficacy or toxicity.
## Detected in the Urine

<table>
<thead>
<tr>
<th>THC-COOH-Glucuronide</th>
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<tbody>
<tr>
<td>Single Use</td>
<td>3 Days</td>
</tr>
<tr>
<td>Moderate Use (4x/Week)</td>
<td>5 – 7 Days</td>
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<tr>
<td>Daily Use</td>
<td>10 – 15 Days</td>
</tr>
<tr>
<td>Long-Term Heavy Smoker</td>
<td>&gt;30 Days</td>
</tr>
<tr>
<td>THC - T1/2</td>
<td>2-7 Days</td>
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Recreational Marijuana Issues

- Quality control/product safety (legal vs street)
- Lingering contaminants in marijuana sold on the street
- Dealers typically sell cannabis by weight; some use sand or glass beads to make their products heavier
- Breathing these particles over years may inflame and scar the lungs
- Higher THC content than medical marijuana limits?
- Not detectable with Breathalyzer test
- Risk of accidents for drivers with THC levels higher than 5 ng/mL blood (similar to blood alcohol concentration of 0.08%)
Dabbing

- Flash vaporizing butane hash oil based concentrate
- More intoxicating than smoking or vaping
Butane Hash Oil Burns

- Names: dabs, wax, earwax, honey, honey oil, shatter
- Contain up to 97% THC
- Products commercially manufactured; some users make them at home
- 20 yo man presented to ED after explosion with burns to face, hands, trunk
- He had been manufacturing hash oil using butane extraction (highly flammable solvent)
- Treated with surgical debridement, pain meds, standard burn care
- Am J Health Syst Pharm 2017;74:1907
Compare Illegal Chemical Structures for JWH-018 and THC - Marijuana Alternatives

- K2, Spice, AK47 belong to a group of blends that contain a mixture of inert plant matter plus chemical grade synthetic cannabinoids sprayed on it.

- Street drug symptoms similar to marijuana PLUS sympathomimetic SXS: agitation, anxiety, tachycardia, tremors, seizures, HEPATOTOXICITY. Plus in April 2018 IL & other states incident with rat poison in 94 cases including 2 deaths (brodifacoum).

- Agonists at CB1 and CB2 receptors.

- May be NDMA glutamatergic antagonists (like ketamine - euphoria, analgesic).
### Synthetic Cannabinoids Family

<table>
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<tr>
<th>Synthetic Cannabinoids Family</th>
<th>Principal Compounds</th>
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</thead>
<tbody>
<tr>
<td>Benzoylindole</td>
<td>AM-694, AM-2233, AM-679, RCS-4, RCS-8</td>
</tr>
<tr>
<td>Naphthoylindole</td>
<td>JWH-018, JWH-022, JWH-073, JWH-081, JWH-122, JWH-210, AM-2201, AM-2232, MAM-2201</td>
</tr>
<tr>
<td>Phenylacetylindole</td>
<td>JWH-167, JWH-250, JWH-316</td>
</tr>
<tr>
<td>Indazolecarboxamide</td>
<td>ADB-PINACA, ADB-FUBINACA, AB-FUBINACA, AB-PINACA, 5F-APINACA, AKB48 (APINACA), MAB-CHMINACA</td>
</tr>
<tr>
<td>Cyclohexylphenyl</td>
<td>CP-55, 940, CP-47, 497, 497-C8 homologue</td>
</tr>
<tr>
<td>Naphthylmethylinindole</td>
<td>JWH-175</td>
</tr>
<tr>
<td>Naphthylpyrrole</td>
<td>JWH-145, JWH-307, JWH-370</td>
</tr>
<tr>
<td>Naphthylmethyldiene</td>
<td>JWH-176, JWH-220</td>
</tr>
<tr>
<td>Aminoalkylindole</td>
<td>WIN-55, 212-2</td>
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<tr>
<td>Adamantoylindoles</td>
<td>AB-001</td>
</tr>
<tr>
<td>Tetramethylcyclopropylketone indole</td>
<td>UR-144, XLR-11</td>
</tr>
<tr>
<td>Quinolinyl ester indole</td>
<td>5F-PB-22, PB-22</td>
</tr>
<tr>
<td>Ibenzopyran</td>
<td>HU-210, JWH-133</td>
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- Kaiser Permanente Northern California review. Questionnaire and tox test within two weeks of questionnaire
- From 2002 to 2014 prevalence of self-reported, past month marijuana use among US adult pregnant women increased from 2.4% to 3.9%
- In aggregated 2002-2012 data, 14.6% of US pregnant adolescents reported past month use
- From 2009 to 2016 adjusted prevalence of prenatal marijuana use based on self report or tox increased from 4.2% (95% CI, 4%-4.5%) to 7.1% (95% I, 6.7%-7.5%)
- Prenatal marijuana use may impair fetal growth and neurodevelopment despite women’s perception of little to no harm in prenatal use
ENDOCANNABINOID SYSTEM

Concentrations of CB receptors

The Endocannabinoid System

Brain cells (neurons) communicate with each other by sending chemical messages. The chemicals (neurotransmitters) cross a gap between neighboring neurons before attaching to their specific receptors.

Presynaptic: The neuron sending a message by releasing a chemical when signaled to do so.

Postsynaptic: The neuron receiving the message when its receptors are activated by specific chemicals (neurotransmitters).

Neurotransmitters: The chemical messengers that travel from one brain cell to another.

Receptors: Activated by neurotransmitters, receptors trigger a set of events allowing a message to be passed along to other neurons.

Endocannabinoids: Natural chemicals (anandamide and 2-AG) that bind to cannabinoid receptors in the brain and the body.

THC: The main active ingredient in marijuana; THC, also a cannabinoid, interferes with the normal functioning of the endocannabinoid system.
Endocannabinoids

- Anandamide and 2-AG

- Neural and nonneural cells in injured tissues produce arachidonic acid derivatives called endocannabinoids.

- They modulate neural conduction of pain signals by mitigating sensitization and inflammation through the activation of cannabinoid receptors that are also targeted by delta-9-THC.
Compounds in Cannabis

- Cannabis, like all herbs, is a polypharmaceutical substance.
- 108 cannabinoids have been isolated (Hanuš 2008).
- The cannabis-derived cannabinoids of most therapeutic interest are THC and cannabidiol (CBD).
  - Minor cannabinoids include cannabigerol, cannabichromene, and tetrahydrocannabivarin (a short-chain C19 homolog of THC).
- Terpenoids are common, often aromatic, organic compounds found in many plants. Terpenoids found in cannabis include β-caryophyllene, myrcene, limonene, and pinene.
- As many as 420 other constituents occur in the plant (Turner et al 1980). -phencyclidine (Angel Dust); -no standardization
Main Phytocannabinoids

- **Psychoactive: THC** ($\Delta$-9-THC, $\Delta$-8-THC, 11-hydroxy-THC [active metabolite]). Binds to CB1 & CB2 receptors as a partial agonist.

- **Not Psychoactive: THCV** (tetrahydrocannabivarin): an analogue of THC
Main Phytocannabinoids

Not Psychoactive:

- CBD (cannabidiol)
- CBN (cannabinot) - degradation product of THC
- CBC (cannabichromene) - sedative and analgesic
- CBG (cannabigerol) - precursor of other cannabinoids
Cannabinoid CB1 Receptors

- Mostly in **brain (cerebellum, cerebral cortex, basal ganglia)**, spine, GI tract, liver, pancreas, skeletal muscle combined with **GABAergic & dopaminergic & serotonergic receptors**, to affect appetite, pain sensation, memory, mood

- In the **hippocampus** and **amygdala**, areas associated with partial seizures. CB1 receptors are also present in nociceptive and non-nociceptive sensory neurons of dorsal root ganglion and trigeminal ganglion as well as in defense cells such as macrophages, mast cells, and epidermal keratinocytes.
Cannabinoid CB2 Receptors

- Activation causes inhibition of proinflammatory cytokine production, cytokine, and chemokine release, and blockade of neutrophil and macrophage migration (anti-inflammatory)

- In peripheral immune system T-cells, B cells, spleen, macrophages (immunosuppression), kidneys, lungs

- In peripheral nerve terminals with a role in anti-nociception
CANNABIS

Side effects
Marijuana Use May Raise Risk of Dying from Hypertension

- European Journal of Preventive Cardiology August 8, 2017

- Three fold risk increase with each additional year of use (NHANES survey); adjusted hazard ratio for death due to hypertension of 3.42 (CI 1.2 - 9.79)

- HR greater than that for current cigarette smokers (HR 1.06; 95% CI 0.4 - 2.77), former smokers (1.33; 95% CI 0.57 - 3.1), alcohol users (HR 0.95; 95% CI 0.37 - 2.45), and those with a prior diagnosis of hypertension (HR 0.81; 95% CI 0.32 - 2.06) or CVD (HR 1.94; 95% CI 0.42 - 8.97)

- Risk may be greater than the risk established for cigarette smoking

- Adults aged 20 and older in survey; N = 1213 (mean age 37.7 years) in cohort
Marijuana/Hashish Bi-Phasic DOSE Effect on Autonomic Nervous System

- LOW DOSES: sympathetic activity is increased while parasympathetic activity is depressed, resulting in mild increases in heart rate and blood pressure

- HIGH DOSES: parasympathetic activity is increased and sympathetic activity is inhibited resulting in the potential for hypotension and bradycardia
Cardiovascular Disorders Associated with ACUTE vs CHRONIC Cannabis Use

- Arrhythmias precipitated by excessive physical activity especially during the first few hours of consumption
- Heterogenous effects on central and peripheral circulation
- Acute cannabis consumption shown to cause increase in BP (SBP) and orthostatic hypotension
- ECS is involved in regulation of heart rate and blood pressure
- THC can cause vasodilation by activating TRPA-1 channel, then reflex tachycardia
- Chronic use associated with decrease in HR and disappearance of orthostatic hypotension
- CB2 receptors are expressed in cardiomyocytes, coronary endothelial cells and smooth muscle cells
Cardiovascular Complications

_Cannabis use may be associated with:_

- Development of atrial fibrillation
- Reversible cerebral vasoconstriction syndrome (strong headaches, neurological focal deficit with reversible vasoconstriction)
- Stroke among youth - significantly underestimated
- Synthetic cannabinoids (Spice) can cause tachycardia & other sympathomimetic symptoms
CANNABIS & STROKES

- MOA: reversible vasoconstriction syndrome associated with subarachnoid hemorrhage, intracerebral hemorrhage, acute ischemic stroke with MJ use
- MOA Stroke: hypotension, cerebral vasospasm, arrhythmia associated cardioembolism
- Retrospective cohort analysis, recreational MJ associated with 17% increased likelihood of AIS hospitalization
- Likelihood increased when MJ combined with tobacco use (31%) and with cocaine use (42%)
- Incidence of AIS greater among MJ users compared to non users (RR: 1.13, 95% CI: 1.11-1.15, p < 0.0001) and had greatest difference in the 24-34 age group (RR: 2.26, 95% CI: 2.13-2.38, p < 0.0001)
Cannabis can affect cerebral auto-regulation and vascular tone leading to vasoconstriction and acute ischemic stroke.

51 yo female

PMHx: HTN, asthma, heavy cannabis use

CC: left upper and lower extremity weakness (2 hours); BP 256/112 mm Hg

Code stroke called, emergent CT scan of her head without contrast revealed acute right cerebral infarct

Urine drug screen positive for cannabis

Treatment: IV labetalol, rtPA. Marked confusion, slurred speech, repeat CT showed new hemorrhage in left pons, death

THC decreases platelet aggregation via activating 2-AG, increased cardiac oxygen demand, vasoconstriction

CB1 & CB2 receptors on platelets

rtPA is 80% cleared after 10 minutes but effects on coagulation cascade may last up to 24 hours (prolongs PT and aPTT)

- N = 108 patients, 26% CB+. Delayed cerebral ischemia diagnosed in 50% of CB+ and 24% of urine drug screen negative patients (p = 0.01).
- CB+ independently associated with development of delayed cerebral ischemia (OR, 2.68; 95% CI, 1.03-6.99; p = 0.01).
- Significantly higher number of CB+ than urine drug screen negative patients had poor outcomes (35.7% vs 13.8%; p = 0.01).
- Univariate analysis, CB+ associated with composite end point of hospital mortality/severe disability (OR, 2.93; 95% CI, 1.07-8.01; p=0.04).
- After adjustment for other predictors, this effect was no longer significant.
- Preliminary Conclusion: CB+ is independently associated with delayed cerebral ischemia and possibly poor outcome in patients with aSAH.
Drug Interactions

Cytochrome P450 Enzymes

- THC and CBD are metabolized by CYP3A4 and CYP2C9 (Yamaori et al 2012, Watanabe et al 2007).
  - CYP3A4 inhibitors slightly increase THC levels.
  - CYP3A4 inducers slightly decrease THC and CBD levels.

- CBD, but not THC, is metabolized by CYP2C19 (Stout and Cimino 2014).
CANNABIS

Drug Interactions
Cytochrome P450 Enzymes

- THC is a CYP1A2 inducer.
  - Theoretically, THC can decrease serum concentrations of clozapine, duloxetine, naproxen, cyclobenzaprine, olanzapine, haloperidol, and chlorpromazine (Flockhart 2007, Watanabe et al 2007).

- CBD is a potent inhibitor of CYP3A4 and CYP2D6.
  - As CYP3A4 metabolizes about a quarter of all drugs, CBD may increase serum concentrations of macrolides, calcium channel blockers, benzodiazepines, cyclosporine, sildenafil (and other PDE5 inhibitors), antihistamines, haloperidol, antiretrovirals, and some statins (atorvastatin and simvastatin, but not pravastatin or rosuvastatin).
  - CYP2D6 metabolizes many antidepressants, so CBD may increase serum concentrations of SSRIs, tricyclic antidepressants, antipsychotics, beta blockers and opioids (including codeine and oxycodone).
Cannabis/Drug Interactions & Effects

- May interact with warfarin (THC and CBD increase warfarin levels) (Yamaori et al 2012)

- Increases bleeding when used with anticoagulants (warfarin, Xarelto, Eliquis), antiplatelet agents (Plavix, Brilinta), NSAIDs (Celebrex, Motrin, Aleve, ASA)
Psychiatric and Medical Management of Marijuana Intoxication in the EMRDept

Patient #1

- 34yof who recently gave birth and is lactating
- CC: racing thoughts, insomnia, euphoria x 1 week
- Disruptive behavior psychotic symptoms after recreational marijuana edible cannabis (THC). Auditory hallucinations. “Broke into neighbor’s home requesting to go to heaven. Feared people were stealing from her and that something bad was going to happen.”
- Social History: Adopted
- Illicit Drug History: recreational cannabis lip balm, cannabis chocolate bars, cannabis dabbing daily over past week
Patient #1

Labs/Diagnostic Tests:

K+ = 3.2 mg/dL
12-Lead EKG: QTcB interval = 508 msec
Temp = 97.5F; HR = 96
BP = 148/111; Resp Rate = 11
Random BS = 196
9-carboxy-THC Blood Level - over 500 ng/mL
Unremarkable CT head
Unremarkable CBC
Patient #1

**PTA OTC/Meds:**
- Energy drinks (+ coffee)
- Propranolol 20 mg po BID for hypertension
- Sumatriptan 50 mg po PRN migraines
- Feverfew 100 mg po daily migraine prevention
- Benadryl 25 mg po HS PRN sleep
- Imodium (loperamide) po at higher than package recommended dose of 8 mg/day (euphoria)

**Diagnosis:** Marijuana-induced psychotic disorder, Marijuana use disorder
Patient #1

- Chronic marijuana users have lower serum sodium and potassium than non users
- Heavy consumption of carbs while intoxicated leads to increase in serum insulin levels driving potassium into cells and causing serum hypokalemia
- Hypokalemia produces reentrant arrhythmias by decreasing conductivity and increasing resting membrane potential, duration of action potential, and duration of the refractory period
- May see periodic hypokalemic paralysis
- EKG changes include decrease in T wave amplitude, presence of U waves, and prolonged QTcB (THC or Imodium)
Patient #1 Workup

- Check co-ingestion of other medications (positive urine tox screen for opioids)

- Check coffee consumption - via mesolimbic dopamine activity, caffeine may precipitate psychosis or worsen affective lability and mood states

- EKG - tele monitor (checking DI with cannabis/propranolol)
Patient #1 Treatment

- Treat hypokalemia and blood sugar excursions

- Risperidone 0.5 mg q 6 h and lorazepam 1 mg q6 h for psychosis and anxiety, respectively

- DC coffee & energy drinks (caffeine)

- Opioid Detox Program - 72 hours in hospital. Warm referral to addiction management center for MAT therapy; Lactation consultant for alternatives

- DC Imodium (prolonged QTCB) on discharge MedRec
Peri-Op Implications of Cannabis Use

- Important to obtain complete illicit drug use history and confirmatory tests if suspected before surgical intervention
- Significant respiratory symptoms and changes in spirometry
- Avoid CNS depressants like barbiturates, opioids, benzos, phenothiazines?
- Avoid drugs that increase HR like ketamine, atropine, epinephrine?
- Intra-op and immediate post-op need of opiates for analgesia in patients with history of recent or chronic cannabis consumption may be significantly increased
Propofol Induction

- Prospective, randomized, single blind study
- N = 30 males using cannabis > once/week; N = 30 nonusers
- Primary outcomes: Propofol ED50 and successful induction determined by loss of consciousness with bispectral index (BIS) value < 60 and insertion of laryngeal mask
- Results: Propofol dose needed to achieve target BIS value not significantly higher in user group, but this group needed significantly higher propofol dose to insert laryngeal mask (314.9 mg ± 109.3 mg vs 263.2 mg ± 69.5 mg, p < 0.04)
- Limitation: no blood level of cannabinoids measured for users
- Cannabis use increases propofol dose required to insert laryngeal mask
Prospective, randomized study

N = 42 cannabis users (based only on history)

N = 31 non-users

All: elective ortho surgery, received Demerol

Primary Outcome: Mean pain intensity difference at the first postop hour (MPID1) and sum of pain intensity differences (SPIID1)

Results: Users had significantly higher supplemental Demerol requirements (82.7 mg, SD = 3.4 vs 51.6 mg, SD = 42.7, p = 0.003) and significantly greater MPID1 scores (1.88, SD = 1.09 vs 1.35, SD = 1.12, p = 0.001) compared to non users

Female users required significantly more analgesic than males (93.3 mg, SD = 45.8 mg vs 78.3 mg, SD = 44.3, p = 0.025)

Conclusions: Greater demand of rescue opioid analgesia within first 6 hours after surgery
Types of Pain

- Visceral, neuropathic, somatic, bone

- INPATIENT MULTI MODAL PAIN TREATMENT: APAP, topical NSAIDs, gabapentin, pregabalin, antidepressants, aromatherapy, acupuncture, narcotics, ginger cream, lidocaine patch

Pain Origins (Objective vs Subjective Responses)

- **Nociceptive pain** - damage to body tissue (sharp, aching, throbbing). Invading immune cells secrete histamine, serotonin, bradykinin, prostaglandin, tumor necrosis factor alpha, interleukin 1 beta, interleukin 6, interleukin 17. Signals carried by C and A gamma peripheral nerves to dorsal root ganglia to thalamus to cortical area.

- **Neuropathic pain** - damage to sensory or spinal nerves sending inaccurate pain messages to higher centers. Diabetic neuropathy. SUBJECTIVE

- **Centralized pain** results from amped peripheral signals. Pain persists despite lack of clear peripheral cause. Fibromyalgia. SUBJECTIVE
Marijuana and Pain Management

- Subjective?

- There is no way to calculate an equi-analgesic dose of opioid to supplant any marijuana used prior to surgery even though there is a cannabis conversion table for different dose forms.

- Surgical anesthesia may be more complex in recent cannabis users with reports of more difficulty with sedation and induction of anesthesia.
Patient #2 - 70 yof 5’3” 223#

- **Social Hx:** Recreational marijuana, 2 joints smoked daily (neuropathic pain)

- **FSBS** = 124 (70-99 mg/dL)
- **Na** = 138 (135-145 mEq/L)
- **K** = 4.6 (3.6 - 5.1 mEq/L)
- **Cl** = 99 (101-111 mEq/L)

- **BUN** = 21 (8-26 mg/dL)
- **Cr** = 6.6 (0.4 - 1 mg/dL)
- Calcium = 4.2 (4.6 - 5.4 mg/dL) ionized

- **CC:** Foot ulcer (debride)

- **Allergies:** Morphine (confusion) Percocet (itch), Vicodan (itch)

- **PMHx:** CHF, non STEMI, CAD, PCI-stent, S/P CABG x 2, high cholesterol, DM Type I, **ESRD on dialysis MWF**, HTN, PAD, Diabetic Foot Ulcer/ Osteomyelitis, Peripheral Neuropathy, Anxiety
PTA Meds:
- Coreg 6.25 mg BID
- Lipitor 80 mg q HS
- Neurontin 100 mg TID
- Xanax 0.25 mg BID PRN anxiety after HD
- Levmir, Novolog daily
- Epogen 3200 Units daily MWF only (anemia)
- Sevelamer 2400 mg TID with meals (phosphate binder)
- Miralax PRN

New Meds:
- Vancomycin 1 gram IV x 1
- Cefepime 2 grams IV daily MWF only
- APAP 650 mg po q 4h PRN mild pain (1-3) or fever
- Tramadol 50 mg po q 4h PRN moderate pain (4-6)
- Dilaudid 0.5 - 1 mg IV q 6h PRN severe pain (7-10)
- Dilaudid 4 mg po q 4h PRN severe pain (7-10)
- Propofol, Versed, mepivacaine 2% to debride foot wound
Morphine, Vicodan, Percocet ‘intolerances/allergies’

Classes of Opioid Medications

<table>
<thead>
<tr>
<th>Phenanthrenes</th>
<th>Diphenyleptanes</th>
<th>Phenylpiperidine</th>
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<tbody>
<tr>
<td>Codeine</td>
<td>Methadone</td>
<td>Fentanyl</td>
</tr>
<tr>
<td>Hydrocodone</td>
<td>Propoxyphene</td>
<td>Meperidine</td>
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<tr>
<td>Hydromorphone</td>
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<tr>
<td>Levorphanol</td>
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<td>Other</td>
</tr>
<tr>
<td>Morphine</td>
<td></td>
<td>Tramadol</td>
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<tr>
<td>Oxycodone</td>
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Meperidine is not recommended for chronic pain because of the potential for accumulation of the neurotoxic metabolite, normeperidine, and a potentially fatal drug interaction with monoamine oxidase inhibitors (MAOIs).
Patient #2 - Cannabis/Drug Interactions

- Cardiovascular - CBD and Coreg increased [ ]. Monitor EKG, BP? THC cardiovascular side effects of long term use?

- Pain/Anxiety - CBD and increased [ ] Opioids, increased Xanax [ ]. Monitor pain med dosing?
Patient #2 (Treatment)

- Pain management (debridement):
  - Propofol dosing increase?
  - Address benzos and opioids together
  - DC Dilaudid, keep tramadol (acute)
  - Other modalities for chronic pain?

- Hemodialysis effects on THC removal from the blood:
  - None as THC metabolites are lipid soluble, not water soluble
Patient #2 Discharge MedRec

- Marijuana for diabetic foot ulcer/peripheral neuropathy pain
- Anxiety about hemodialysis and the future
Admission MedRec Process
**Medication Reconciliation**

*Medication discrepancy:* A difference between medication regimens derived from various sources (e.g., medical records and patients’ home medication lists).

- **Intentional discrepancy:** A medication prescribed differently from the patient’s original medication order due to the patient’s current health status (e.g., the reduced dosage of the antihypertensive agent due to patient’s hypotensive state).

- **Unintentional discrepancy:** A medication prescribed differently from the original medication order without a documented reason.
Motivational Interviewing (Miller & Rollnich)

- Person-centered, goal-oriented methods of communication for eliciting and strengthening intrinsic motivation for change
- Provider patient relationships characterized by PACE: partnership, acceptance, **compassion**, and evocation (drawing out of patients their own internal reasons for changes)
- Open-ended questions, affirmations, reflections, summaries
- SEEK FIRST TO UNDERSTAND
- [www.motivationalinterviewing.org](http://www.motivationalinterviewing.org)
Medical Marijuana Policy Development

- Hospital Inpatient Policy: Pharmacist-Led Medication Reconciliation
- Computer Documentation: ‘social history’ versus ‘medication’
- Storage, chain of evidence/log, employee FMLA usage, random drug screens, education (interactions), management of outpatient MD certification requests
- SOAP notes
Impact of Marijuana Use on Patient Care: From Recreation to Reconciliation

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Flat Rock, North Carolina

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