COMPLEX PAIN MANAGEMENT IN PALLIATIVE CARE

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Palliative Care Education Session

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- Disclosures - Nil

- 1. Create strategies to anticipate and plan for complex pain
- 2. Illustrate pain management strategies beyond opioids
- 3. Identify barriers to complex pain management in rural communities

INTRODUCTION

- ~80% cancer patients experience moderate to severe pain in advanced stages
- ~10-20% of those → opioid intractable with 5-25% requiring invasive modalities
- Overwhelming personal and socioeconomic burden associated with poorly controlled pain
  - ↓ QOL, ↑ use health care system, impairment interpersonal relationships, ↓function, ↑ rates of depression, anxiety and insomnia
- Chronic pain is the leading cause of disability in the world
- The Eastern Cooperative Oncology Group study found that nearly 50% of cancer pain patients did not receive adequate analgesic therapy
- Consistent with other research concluding pain is underdiagnosed and undertreated

INTRODUCTION

- Causal link to opioid crisis has magnified the fear of opioid prescribing
- COVID-19 pandemic has further strained system thus challenging clinicians with dual responsibility of limiting spread without impacting ability to treat pain
- Access and treatment of pain is deemed a fundamental human right
- For pts with longer-term palliative diagnosis, pain may be most important prognostic indicator
  - Pain as the fifth vital sign
- Intolerable side effects from systemic opioids/meds can result in severe sedation, immobility, decreased intake and an earlier EOL trajectory than that from disease alone

INTRODUCTION

- Concept of double effect may be acceptable when alternative is intractable pain and suffering
- Before pain declared intractable, every possible appropriate and accessible intervention should be exhausted
- Traditional "three tiered" WHO ladder often inadequate in complex patients
- Interventions have evolved beyond opioids and traditional "three tiered" approach and can offer dramatic effective pain relief for some patients
- Early identification of potential barriers and patients at high risk of poorly controlled pain can help mitigate challenges and improve delivery of optimal pain control

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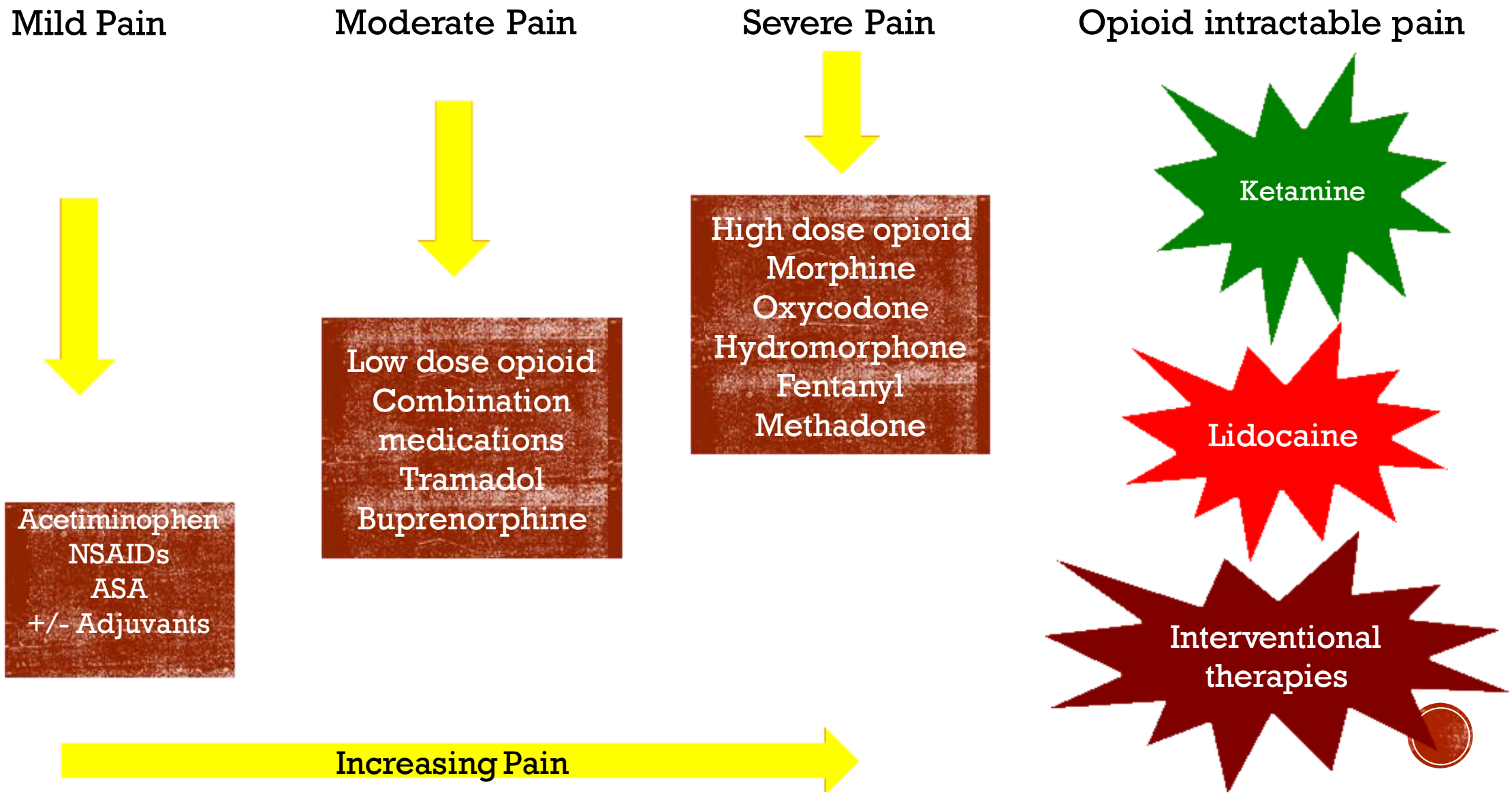
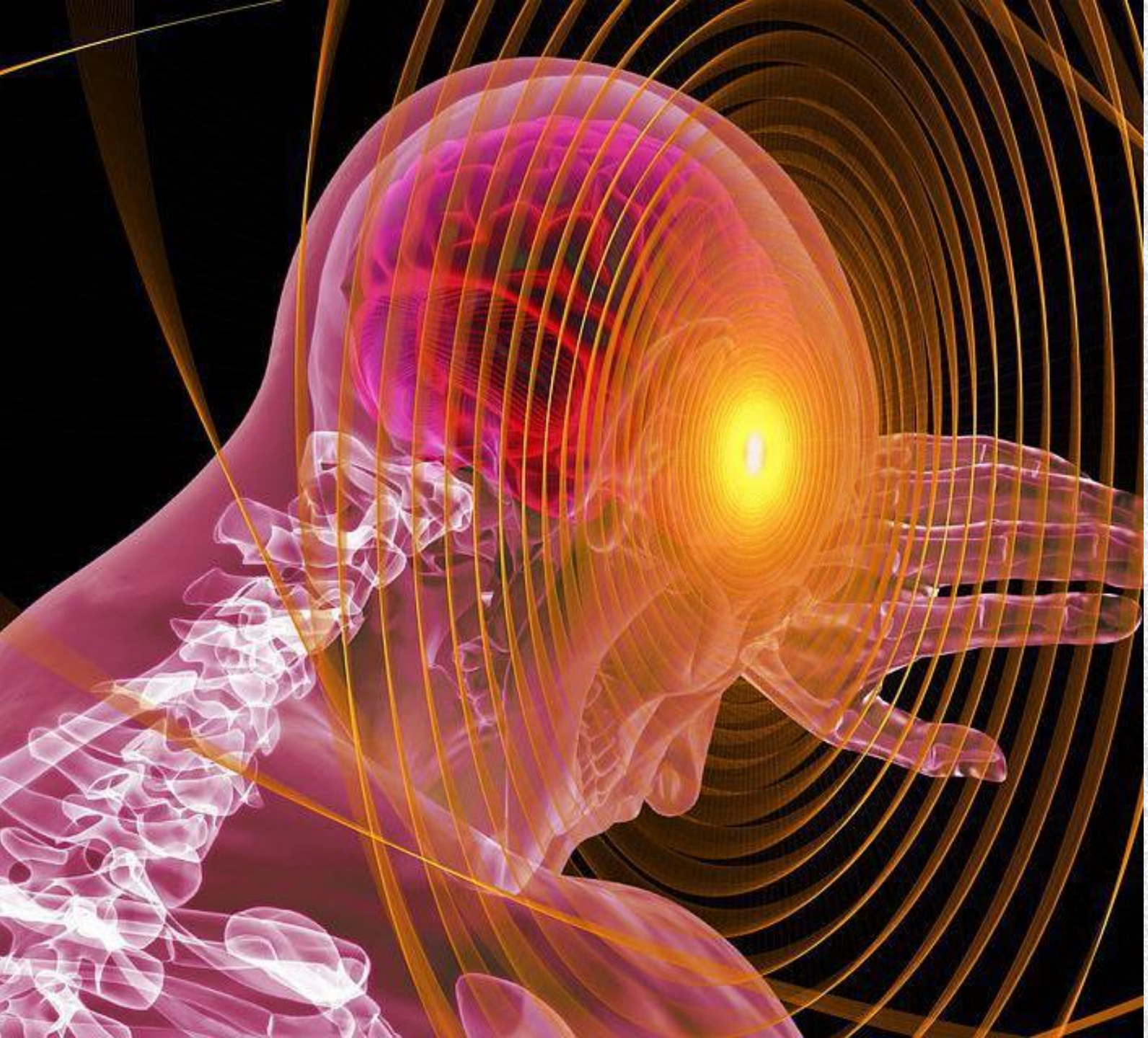




COMPLEX PAIN MANAGEMENT

1. Create strategies to anticipate and plan for complex pain

ANTICIPATING COMPLEX PAIN

- Several attempts to establish a standardized classification system to predict cancer related pain control for both clinical practice and research
- To date, no universally accepted pain classification measure that can accurately predict prognosis of pain in cancer patients
- Edmonton Classification System for cancer pain (Bruera et al) and The Cancer Pain Prognostic Scale are two examples
- Difficulties in definitions, interpretations and lack of evidence of predictive value have limited widespread use
- Despite lack of consensus, several common themes identified and often observed in clinical practice

ANTICIPATING COMPLEX PAIN

- Pain experience and response to treatment are influenced by:
  - 1) **Patient factors**
  - 2) **Disease factors**
  - 3) **Pain characteristics**

ANTICIPATING COMPLEX PAIN

2. PATIENT FACTORS

- Psychological distress
- Cognitive function
- Addiction history
- Current total daily opioid dose
- Tolerance (opioid escalation index)
- Age?
- Gender?

ANTICIPATING COMPLEX PAIN

2. DISEASE RELATED FACTORS

Primary site of cancer or sites of spread – 73%

- Tumor-related somatic pain (bone, soft tissue)
- Tumor-related visceral pain syndromes (bowel obstruction/perforation, liver capsule pain, tumor hemorrhage, midline retroperitoneal syndrome, perineal pain, peritoneal carcinomatosis, ureteric obstruction)
- Tumor-related neuropathic pain (leptomeningeal, cranial neuralgias, radiculopathies, peripheral mononeuropathies, paraneoplastic syndromes)
- Stage of cancer

ANTICIPATING COMPLEX PAIN

2. DISEASE RELATED FACTORS

Primary cancer sites at high risk of pain

- Head and neck (67 to 91%)
- Urogenital (40 to 60%)
- Gynecological (50 to 71%)
- Lung/Bronchus (44 to 67%)
- Breast (40 to 89%)
- Pancreas (72 to 85%)
- Esophagus (56 to 94%)
- Virtually all patients with multiple myeloma and sarcoma have pain

ANTICIPATING COMPLEX PAIN

2. DISEASE RELATED FACTORS

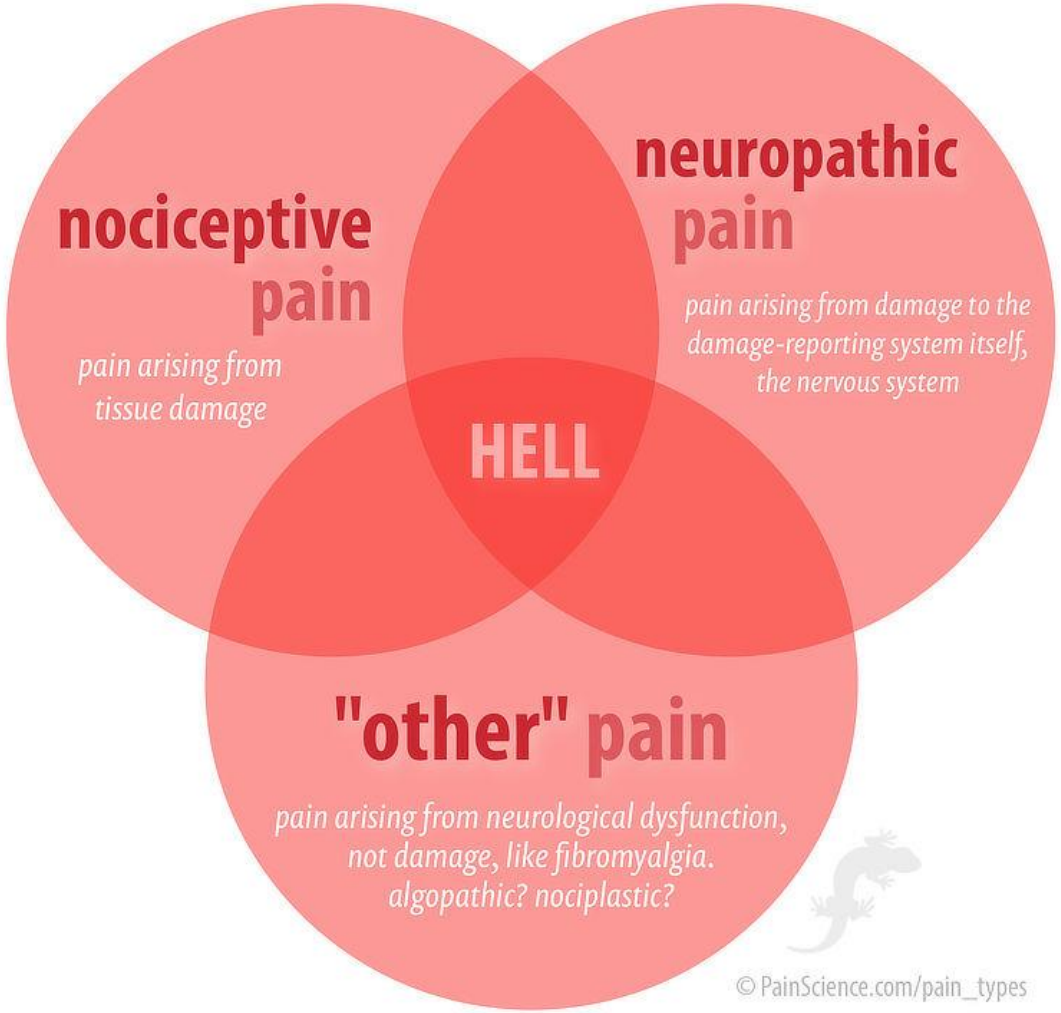
Antineoplastic treatment – 17%

- Oral mucositis
- Chemo-induced (headache, neuropathy, arthralgias/myalgias, hand-foot syndrome, etc.)
- Radiation-induced (bone pain, plexopathy, enteritis/proctitis, lymphedema, osteonecrosis)
- Immune therapy (joint and muscle pain)
- Hormone therapy (headaches, muscle and joint pain)
- Postsurgical pain syndromes (phantom limb, postmastectomy, lymphedema, postthoracotomy, etc.)

ANTICIPATING COMPLEX PAIN

2. **DISEASE RELATED FACTORS**

Conditions unrelated to cancer – 10%



ANTICIPATING COMPLEX PAIN

- 3. **PAIN CHARACTERISTICS**
  - Type of pain:
    - Nociceptive, neuropathic, mixed
    - Neuropathic associated with higher risk
  - Temporal characteristics and pattern of pain occurrence
    - Intermittent, constant, incidental
    - Incidental pain associated with higher risk
  - Pain intensity and time since onset
    - Higher intensity and chronic associated with higher risk
  - Number of pain sites
    - Multiple associated with higher risk

Edmonton Classification System for Cancer Pain

Patient Name: _____

Patient ID No: _____

For each of the following features, circle the response that is most appropriate, based on your clinical assessment of the patient.

1. Mechanism of Pain

No	No pain syndrome
Nc	Any nociceptive combination of visceral and/or bone or soft tissue pain
Ne	Neuropathic pain syndrome with or without any combination of nociceptive pain
Nx	Insufficient information to classify

2. Incident Pain

Io	No incident pain
Ii	Incident pain present
Ix	Insufficient information to classify

3. Psychological Distress

Po	No psychological distress
Pp	Psychological distress present
Px	Insufficient information to classify

4. Addictive Behavior

Ao	No addictive behavior
Aa	Addictive behavior present
Ax	Insufficient information to classify

5. Cognitive Function

Co	No impairment. Patient able to provide accurate present and past pain history unimpaired
Ci	Partial impairment. Sufficient impairment to affect patient's ability to provide accurate present and/or past pain history
Cu	Total impairment. Patient unresponsive, delirious or demented to the stage of being unable to provide any present and past pain history
Cx	Insufficient information to classify.

ECS-CP profile: N__ I__ P__ A__ C__ *(combination of the five responses, one for each category)*

Assessed by: _____ **Date:** _____

ANTICIPATING COMPLEX PAIN

- Case example:
  - 35 y.o male newly diagnosed stage 4 NSC lung cancer with diffuse bony metastasis
  - Initiated on chemotherapy + radiation to bony mets and primary lung
  - PMhx: Chronic low back pain, polysubstance abuse, anxiety/depression with previous suicide attempts, methadone maintenance therapy
  - Medications: MEDD >1000 mg, gabapentin, nortriptyline, duloxetine, baclofen, mirtazapine, acetaminophen
  - Pain:
    - Rated 15/10, exacerbated by recent radiation, multiple areas, often "all over"
    - Neuropathic and nociceptive components
    - Constant with severe, intermittent incident pain patterns
  - Social history: NFA, minimal social supports, children estranged/foster care, no phone

ANTICIPATING COMPLEX PAIN: PLANNING

- Early identification and management of pain
- Early palliative care referral
- Early identification and management psychological distress
- Early identification and management of cognitive impairment
- Early identification and management of addiction
- Close monitoring during periods of possible pain exacerbations
- Early collaboration and collateral from multidisciplinary teams

COMPLEX PAIN MANAGEMENT

- Illustrate pain management strategies beyond opioids

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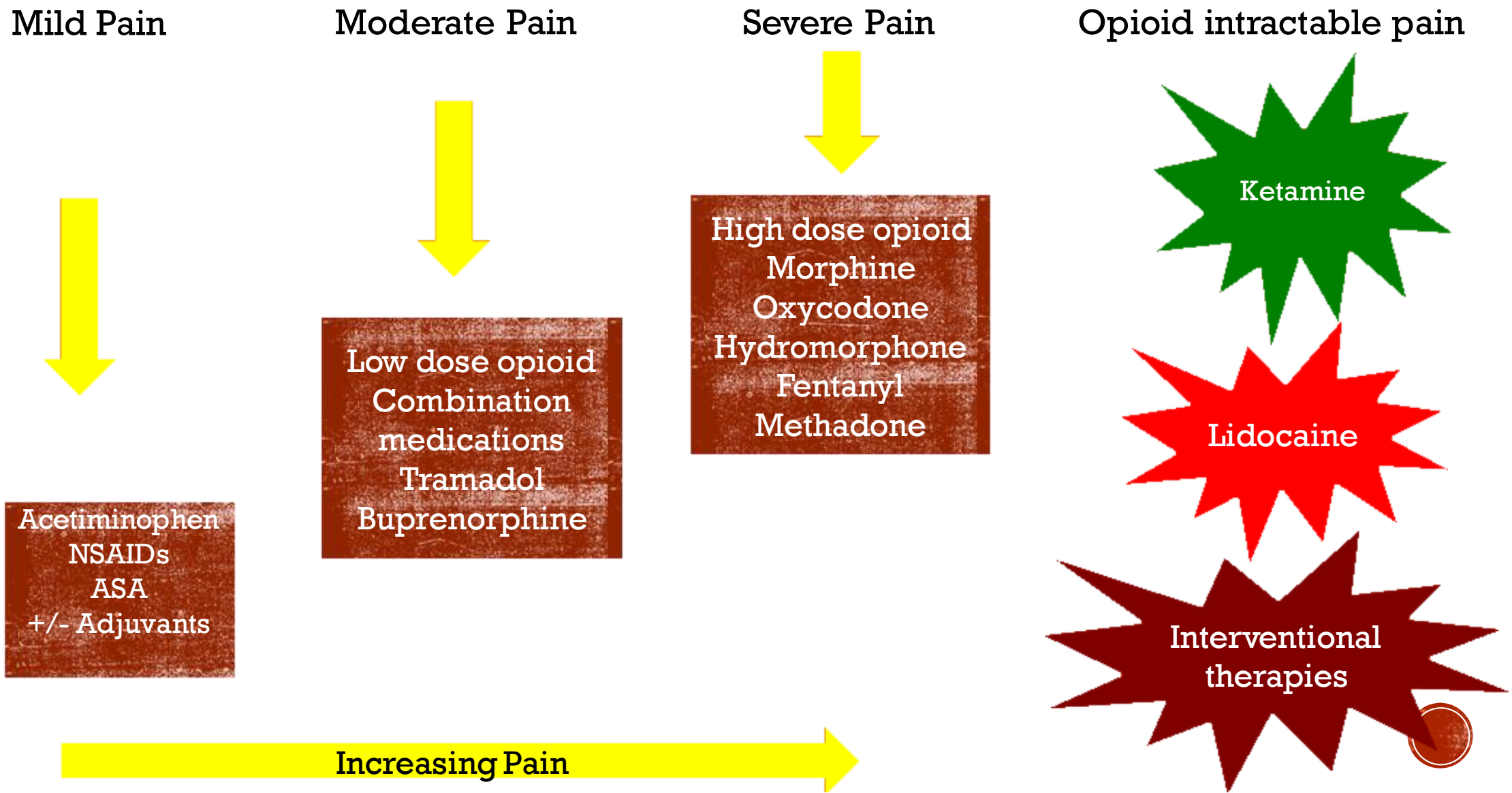


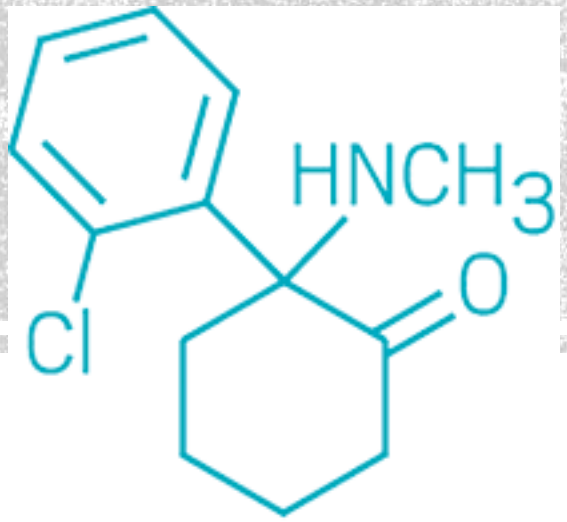
CASE EXAMPLE

- 45 yo M with multiple myeloma
- Presents with severe lancinating pain to upper C-spine radiating upwards to skull and shoulders bilaterally
- Has completed CyBorD induction, stem cell transplant, palliative radiation to spine, pelvis, ribs, and vertebropasty T6 – L3
- MRI +ve pathological # C2 and C7, C-spine precautions immediately initiated
- Initiate opioid infusion and steroids but titration ineffective with signs of opioid neurotoxicity with increasing doses
- Subjective and objective pain reports are severe/excrutiating
- Awaiting neurosurgical consult but closest center COVID outbreak and transfer delayed
- What do you do?

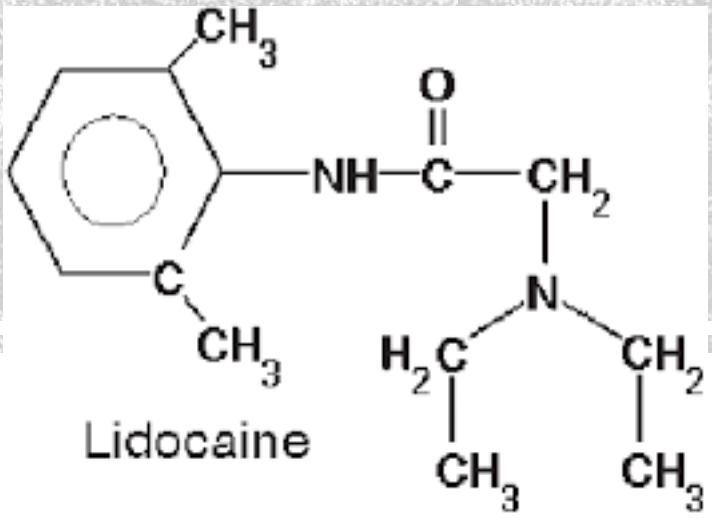
CASE EXAMPLE

- Is patient in pain crisis?
- Is current medication effective? Is dosing adequate?
- Is current formulation and route appropriate?
- Are side effects tolerable?
- What is patient's prognosis/clinical condition?
- Is pain likely short term or long term?
  - ?Other definitive managements/interventions
- What resources are available
- Are staff educated on proposed treatment?
  - Drug protocol available, nursing guidelines established, any ward restrictions?
- What are pt's goals of care
  - Are proposed interventions reasonable to pt

KETAMINE AND LIDOCAINE



Ketamine



Lidocaine

KETAMINE

- A dissociative anesthetic agent and N-methyl-D-aspartate (NMDA) receptor antagonist
- Also interacts with Ca and Na channels, cholinergic transmission, noradrenergic and serotonergic reuptake inhibition, and opioid-like receptors
- Derivative of PCP (angel dust) first synthesized in 1962
- Initially introduced as dissociative anesthetic for which still widely used
- Following FDA approval 1970, gained momentum for pain initially in treatment of burn injuries, dressing changes and transporting acutely injured patients during Vietnam war

KETAMINE

- Now used in wide range of acute and chronic pain conditions especially neuropathic and opioid refractory/tolerant states
- NMDA receptor major contributor to the "wind-up" phenomenon implicated in central sensitization syndrome, hyperalgesia, allodynia
- May serve to "reset" opioid receptors thus protecting against/treatment of opioid tolerance

KETAMINE

- When used for pain, experience suggest most effective as LOW dose continuous infusion
- May allow significant reduction in systemic opioid dose → improving function/QOL
- Most often for short term use while more definitely treatment strategies implemented or EOL
- For patients with short prognosis and for whom stopping infusion is not tolerated can remain on infusion indefinitely or consider rotation to PO

KETAMINE

- ***Indications***
  - Neuropathic pain
  - Incident pain
  - Opioid intractable pain
  - Topical wound management
  - Mucositis
  - Hyperalgesia, allodynia
  - Terminal depression
  - Post-operative

- ***Contraindications***
  - Raised ICP
  - Raised intraocular pressure
  - Severe cardiac disease
  - Severe hypertension
  - Epilepsy
  - Psychosis

KETAMINE

- Onset
  - IV 5-10 min, subq 15-20 min, oral 30-60 min
- Duration
  - Parenteral 30 min – 2 hrs
  - Oral 4-6 hrs
- Metabolism
  - Liver with extensive first pass to norketamine
- Excretion
  - 90% renal, feces

KETAMINE

- ***Dosing***
  - Great variety in practice/literature regarding dose, route, and frequency
  - Lack of good quality evidence to guide practice
  - Most clinical experience supports low dose (subanesthetic) minimizing intolerable SE
  - Most effective when used short term
  - Important to consider long term plan

KETAMINE

- ***Infusion***
  - Typical starting dose 5 mg/hr subq or IV
    - Often following trial dose 5-10 mg x 1 push
  - Increase by 5 mg/hr every 24 hr to max 20 mg/hr
  - Weight based protocols 0.1-0.4 mg/kg/hr
  - Usual dose range 50 – 600 mg/24 hr
  - If pain is controlled infusion weaned/stopped after several days and pain reassessed
  - If pain reemerges consider:
    - Addition/rotation neuropathic agents
    - Interventional options
    - Oral ketamine

KETAMINE

- Oral ketamine
  - More potent than parenteral
  - Metabolism to norketamine which is equianalgesic and produced at higher levels than when given parenterally
  - No official guidelines for conversion to oral
    - Most suggest 25-50% dose reduction (but as high as 1:1)
  - Typically divided bid-qid
  - Titrate by 5-10 mg increments
  - Typical starting dose 5-10 mg PO qid if naive
  - Usual tolerated dose range 60-100 mg/d
    - (up to max 900 mg/day reported)

KETAMINE

- "Burst" ketamine protocol
  - Pulse course offered over max 5 d
  - Offers adequate trial over short period and minimizes logistics of long-term use
- Starting dose 100mg/24h via continuous infusion
  - If effective continue for 3 days then cease
- If ineffective, increase to 300mg/24h
  - If effective, continue for 3 days then cease
- If ineffective, increase to 500mg/24h
- Cease at day 5 whether effective or not

KETAMINE

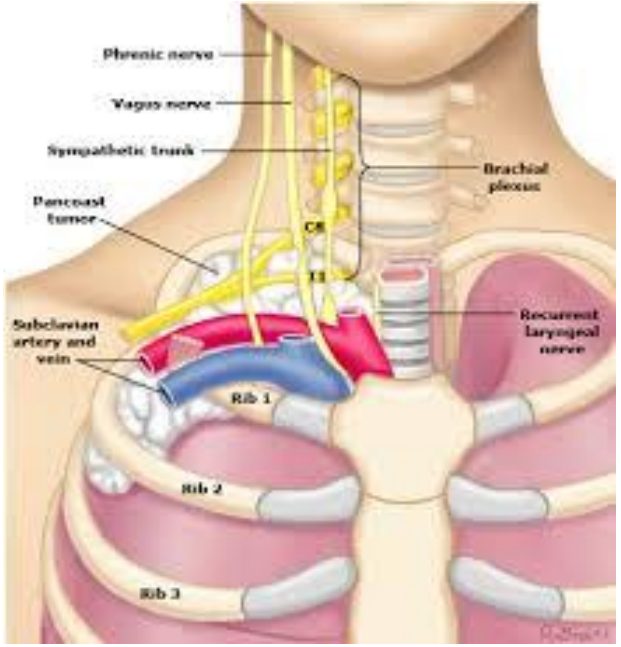
- Side effects
  - Tachycardia, arrhythmias
  - Dysphoria
  - Hypertension
  - Salivation
  - Nausea
  - Urinary symptoms: LUTS, papillary necrosis, ulcerative cystitis(chronic use)
  - Hepatobiliary toxicity (chronic use)
  - Emergence phenomena: dysphoria, vivid dreams, excitement/agitation, irrational behavior, confusion, hallucinations
- When short course, low dose (<0.2mg/kg/hr) SEs are infrequent
- Co-administration of protective agent (benzo, haloperidol) minimizes/prevents side-effects

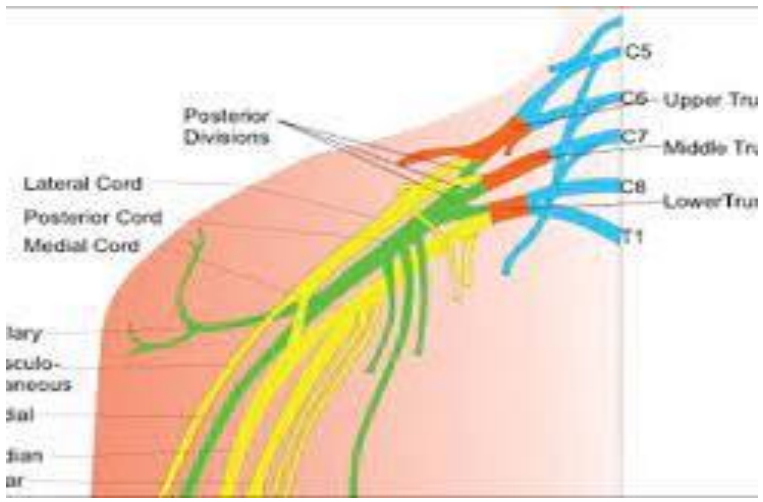
KETAMINE

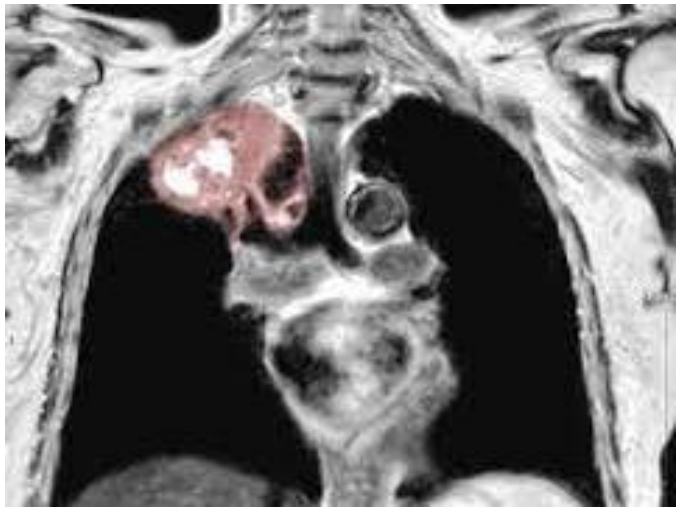
- Evidence remains poor and conflicting
- Clinical experience demonstrates very effective for some patients
- Likely best when short term, low dose, intractable syndromes
- More studies to better determine:
  - ?which patients
  - ?which route
  - ?which dose

CASE EXAMPLE

- 56 y.o F with NSC Pancoast tumor right lung
- Severe neuropathic pain radiating to right neck, shoulder, upper arm from brachial plexus infiltration
- PPS 70%
- Status post palliative XRT and on 1st line systemic therapy
- Admitted with pain crisis despite:
  - Methadone 30 mg PO tid +10 mg PO q3hrs max 3 doses/day
  - Gabapentin 900 mg PO tid
  - Nortriptyline 25 mg qHS
  - Baclofen 10 mg qid PRN
  - Hydromorphone PRN via CADD 10-20 mg subq q15 min PRN


Phrenic nerve
Vagus nerve
Sympathetic trunk
Brachial plexus
Pancoast tumor
C8
T1
Subclavian artery and vein
Recurrent laryngeal nerve
Rib 1
Rib 2
Rib 3


C5
C6
Upper Tru
C7
Middle Tr
C8
Lower Tru
T1
Posterior Divisions
Lateral Cord
Posterior Cord
Medial Cord



THOUGHTS?

LIDOCAINE

- An amide local anesthetic agent and nonselective Na-channel blocker
- Used for local anesthesia and systemically as anti-arrhythmic drug
- Use in pain first described in WWII for acute post-operative states
- Now used in management of various pain states:
  - Neuropathic: Diabetic neuropathy, post-herpetic neuralgia, centrally mediated pain, malignant nerve infiltration (e.g. brachial plexus), ischemic pain
  - Opioid refractory pain

LIDOCAINE

- Injured nerves develop abnormal, spontaneously active Na-channels at the site of injury and nerve pathway
- Conduction of pain mediated by changes in Na-ion concentrations across neuron membranes
- Lidocaine produces analgesia by several mechanisms but likely primarily by suppressing abnormal firing/blocking Na-gated ion channels peripherally, centrally
- Pain relief may be exceptional for some
- Effect may last far beyond therapeutic blood levels (average ~10 days)

LIDOCAINE

- Inexpensive
- Side-effect profile is predictable and with a wide safety margin
- Given as intermittent single dose or continuous infusion (center dependent)
- Symptoms of toxicity are transient and easily reversible given it's short ½ life
  - within < 2 hrs

LIDOCAINE

- Contraindications
  - Cognitive impairment (must be able to report pain and side effects)
  - Prior allergy to local "amide" type anesthetics (likely known from previous dental, rare)
  - Liver failure
  - Renal failure
  - Severe cardiac failure, bradycardia, heart block
  - Uncontrolled seizures
  - Poorly controlled hypertension
  - Hypokalemia

LIDOCAINE

- Pre-procedure
  - EKG within 14 days IF:
    - Male > 65 yrs, female >55 yrs and/or cardiac history
  - Baseline: Vitals, K, Cr, BUN, LFT
- Monitoring
  - As clinically indicated
  - Repeat ECG, labs
  - Lidocaine levels (IV: 8-12 hrs, SUBQ: 24-72)

LIDOCAINE

- Dosing
  - ***Lidocaine Challenge***
    - Loading dose: 1-5 mg/kg (typically 2mg/kg or 100 mg) IV or subq over 15-60 minutes
    - 30 minutes post infusion assess pain (time to analgesia anywhere between 1-45 minutes)
    - If pain improved and no SE start continuous infusion
  - ***Continuous infusion***
    - Initiated at 0.5-3 mg/kg/hr, lowest possible dose to control pain
    - Typical range 1-2mg/kg/hr

LIDOCAINE

- Dosing
  - ***Intermittent Dosing***
    - 5-15 mg/kg (maximum 900 mg)
    - Infused over 60 – 120 min
    - Repeat as per patient's need (average every ~10-30d)
    - Subsequent doses based on previous effect and toxicities
    - Discontinue if no response or side effect

LIDOCAINE

- Occasionally patients will report dramatic stepwise "erasing of pain"
- May need rapid decrease in pain meds/opioids
  - Consider 20-30% decrease especially if longer acting
- Therapeutic range for pain relief between serum lidocaine levels 5-20 mmol/L
  - Well below threshold of severe toxicity
  - No cardiac risks at clinically appropriate levels
- If blood levels increase, side effects are sequential, predictable and easily reversed by stopping infusion

LIDOCAINE: TOXICITY

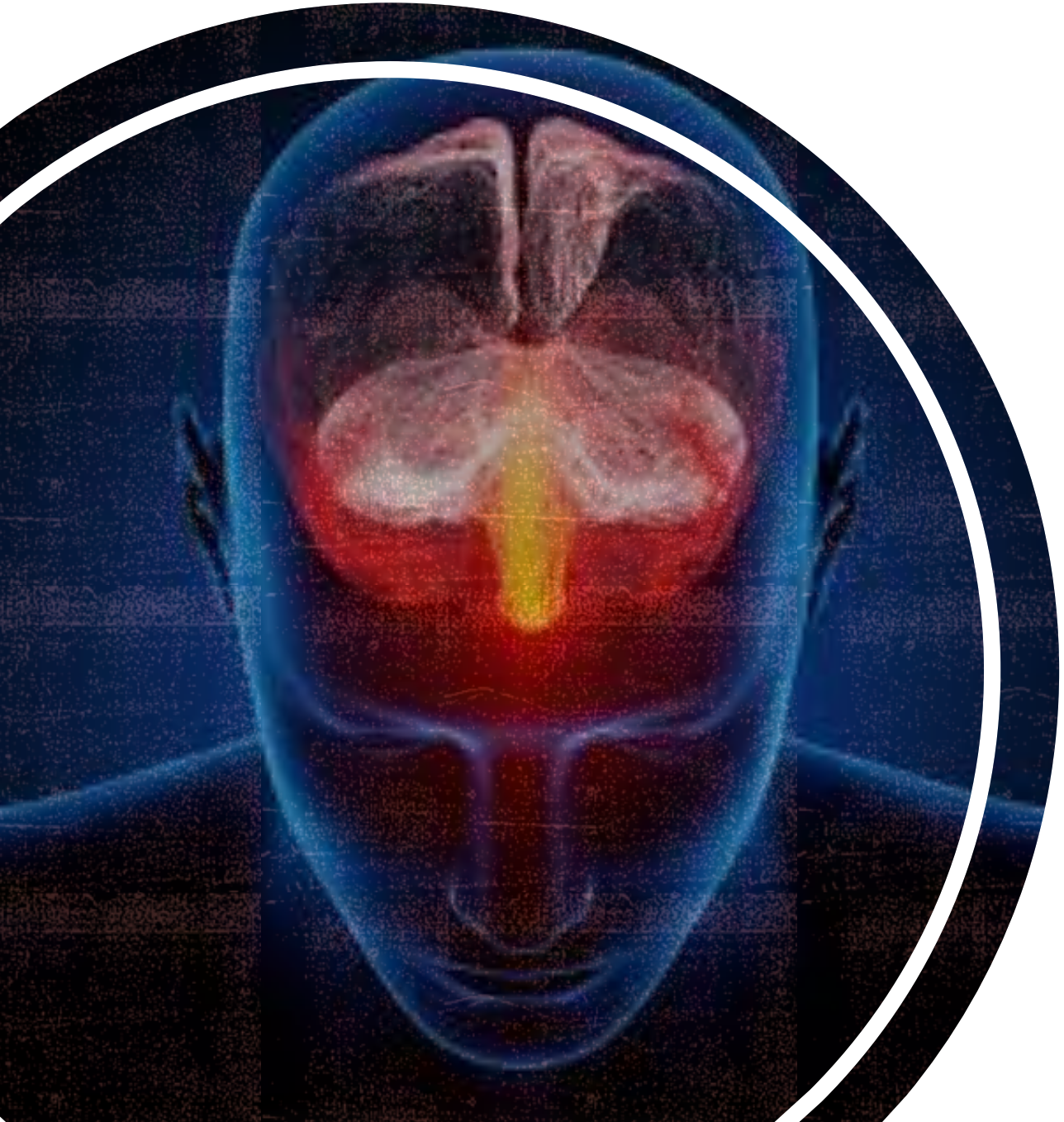
- Progressive, predictable toxicity occurs with increasing dose and serum levels
  - 8-13 mmol/L: Perioral numbness, lightheadedness, nausea at upper end of therapeutic range
  - 17-25 mmol/L: Metallic taste, elevated BP, drowsiness, paraethesia
  - >21 mmol/L: Visual/auditory disturbances, muscle twitching, confusion, agitation, psychosis and dysarthria
  - > 34 mmol/L: Severe muscle twitching/convulsions, coma
  - > 42 mmol/L: Seizures, AV block, hypotension and subsequent cardiovascular collapse

LIDOCAINE VS KETAMINE

- No comparative studies
- Most often prescriber preference/familiarity/patient preference
- May depend on developed drug protocols, nursing education and established monitoring guidelines
- What is the long term strategy?
  - If short term ?ketamine
  - Longer term ?lidocaine
- Capacity within hospital/community
  - Can infusion be maintained outside of hospital?

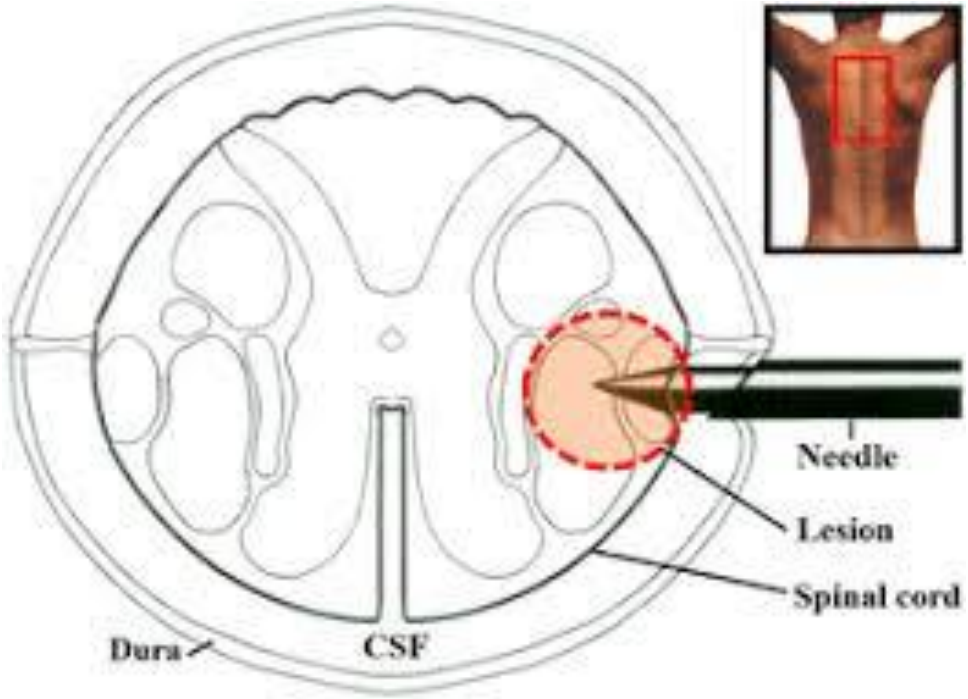
KETAMINE AND LIDOCAINE

- Initially introduced as "last resort" for patients closest to death and willing to accept "risky and unconventional" therapy
- Many years of clinical experience to support improved pain and quality of life with little associated risk/toxicity at recommended doses
- Doses used for pain control are generally low and safe
- Pain relief can last from a few hours to a few weeks
- Has gone from "unconventional" to "conventional" with numerous protocols for administration in most all acute care settings



COMPLEX PAIN MANAGEMENT

- What strategies exist beyond pharmacologic options?



INTERVENTIONAL THERAPIES

INTERVENTIONAL THERAPIES

- Extremely variable across centers/geographical location/clinical expertise
- Largely dependent on functional status, prognosis, accessibility, ability to travel
- Talk with your local/regional anesthetists, orthopods, IR, gastroenterologists, neurosurgeons, other?
- Evidence base for all limited

VERTEBRAL AUGMENTATION

VERTEBRAL AUGMENTATION

- ***Vertebroplasty***
  - Injection of bone cement under fluoroscopic guidance
- ***Kyphoplasty***
  - Introduction of inflatable balloon to restore height and create cavity for cement
- Choice of vertebroplasty vs kyphoplasty based on technical considerations of performer
- Same procedure from referee's perspective
- Indications
  - Symptomatic pathological vertebral compression # without epidural disease/retropulsion of bony fragments
  - Pain refractory to noninvasive therapies

VERTEBRAL AUGMENTATION

- Mechanism for pain relief not entirely understood
- Presumably, cement stabilizes pathological microfractures reducing mechanical forces that irritate CNS
- All spinal levels theoretically possible
- C-spine very rarely attempted
  - Technically more challenging
  - Complications more catastrophic
  - Less frequent (vs T and L spine) site of metastasis

VERTEBRAL AUGMENTATION

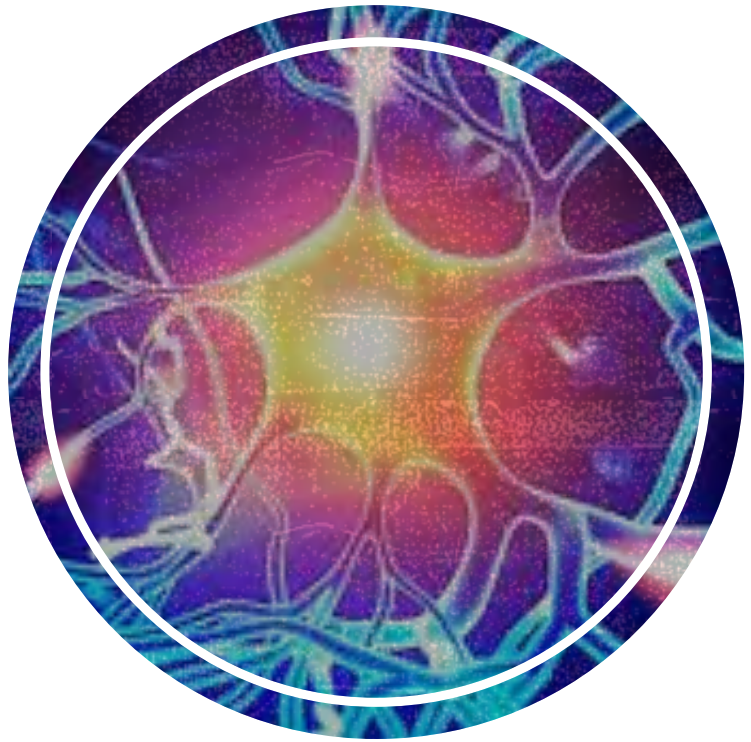
- Performed by: IR (esp higher levels), orthopedics, neurosurgery
- Contraindications
  - Presence of epidural disease, neurological compromise/unstable #, systemic or local infection, uncorrected hypercoagulable state, severe cardiopulmonary disease
- Complications
  - Minor complications < 3%
    - Small asymptomatic local cement leaks, fracture of adjacent vertebrae
  - Major < 1%
    - Extravasation of cement (PE, stroke, damage to SC), direct injury from instrumentation, infection, bleeding

VERTEBRAL AUGMENTATION

- If ineffective or not possible consider radiofrequency ablation, facet joint injection, epidural steroid injection
- Conflicting literature but recent reviews support following claims (low grade evidence):
  - Significant and rapid reduction in pain (48-97% pain relief to some degree)
  - Decreased need for systemic opioids/side effects
  - Improved functional abilities
  - Low complication rates

NERVE BLOCKS AND NEUROLYSIS

- ***Nerve block***:
  - Injection of local anesthetic for diagnostic, prognostic, therapeutic purposes
    - **Sympathetic nerve block**:
      - e.g. Celiac plexus, superior hypogastric plexus
    - **Somatic nerve block**:
      - e.g. Intercostal nerve, brachial plexus, femoral nerve, epidural



NERVE BLOCKS AND NEUROLYSIS

- **Therapeutic block**
  - No randomized studies but many case series and expert opinions
  - Non-neurolytic or neurolytic
    - **Non-neurolytic**
      - i) **Bolus**: Local anesthetic bolus injection
      - ii) **Continuous**: Infusion via epidural, intrathecal or perineural catheter for short term or rarely longer term (tunneled) use
      - Eg. Continuous brachial plexus block, femoral nerve block, intrathecal catheter

NERVE BLOCKS AND NEUROLYSIS

- **Therapeutic block**
  - **Neurolytic**
    - Produces analgesia by destroying afferent neural input or sympathetic structures
    - Destruction via surgery, cold (cryotherapy), heat (radiofrequency ablation), or material (phenol, alcohol)
    - Extent of degeneration and extent and time of regeneration variable (3-6 months)
    - Risk: Deafferentation pain syndrome, inadequate relief, nerve injury, bleeding, paralysis

NERVE BLOCKS AND NEUROLYSIS

- **Neurolytic**
  - Often considered "last resort" except for:
  - i) **Celiac plexus neurolysis**
    - Upper abdominal malignancy (esp pancreatic cancer)
    - Safe and effective
    - Performed intraoperatively, endoscopically, fluoroscopy
    - Recommended early in course if pain significant (some experts even support first line strategy)

NERVE BLOCKS AND NEUROLYSIS

- **Neurolytic**
  - ii) **Superior hypogastric plexus neurolysis**
    - Gynecologic, colorectal, genitourinary cancers with pelvic pain
  - iii) **Others**
    - More rare
    - Must balance loss of permanent sensory and/or motor function (e.g. brachial plexus) with that of pain
    - Trigeminal nerve, medial branch N, brachial plexus, etc.

ADVANCED INTERVENTIONAL THERAPIES

- **Percutaneous Cervical Cordotomy**
  - Percutaneous destruction of spinothalamic tract relieving pain (temperature, deep touch) to one side of body
  - No numbness, strength and proprioception preserved
  - Indications: Unilateral pain below 4th cervical dermatome + prognosis <1 year
  - Sarcoma, mesothelioma (costopleural syndrome), brachial plexus infiltration
  - No follow-up required
  - Inexpensive
  - Dramatic reduction in systemic medications

ADVANCED INTERVENTIONAL THERAPIES

- **Neuraxial infusions**
  - Continuous infusion into epidural or intrathecal space
  - Allows for much less opioid (10x epidural, 100x intrathecal), with direct delivery to CNS
  - Estimated 2% palliative patient population
  - Epidurals may be limited to specific in-patient units
  - Access to permanent intrathecal catheters variable and limited across Canada
    - ?Any centers in Ontario
    - Vancouver IT program with partnership through BCCA since 2002
    - Tunneled catheters for long-term prognosis (>3months), must live within reasonable proximity to provider

COMPLEX PAIN MANAGEMENT

- Identify barriers to complex pain management in rural communities

BARRIERS TO EFFECTIVE PAIN MANAGEMENT

- ACCESS ACCESS ACCESS
  - Proximity to closest center/specialist
    - Ability to access service
    - Ability for local community to manage complications
  - Number of trained specialists
    - Waitlists, willingness as sole provider
  - Patient mobility/frailty
  - Weather and road conditions
  - Mode of transportation and lack of reliable driver
  - Costs of travel
  - Pandemic effect

BARRIERS TO EFFECTIVE PAIN MANAGEMENT

- Opioid crisis
- Lack of adequate training
- Patient and prescriber fear
- Costs
  - Coverage and drug availability issues
  - Non-drug therapies are expensive
    - CBT, acupuncture, mindfulness, physical and rehabilitation services

BARRIERS TO EFFECTIVE PAIN MANAGEMENT

- Pandemic impact
  - Increase in chronic pain consults?
  - Fear of in person visits
  - Fear of travel
  - Fear of hospitalization
  - Loss of access to "non-essential" services or out of town resources
  - Drug shortages?

BARRIERS TO EFFECTIVE PAIN MANAGEMENT: STRATEGIES

- How have you mitigated/solved these barriers?
  - Virtual and phone assessments
  - Development of drug protocols
    - Our local experience
  - Collaboration with other health professionals
    - Our local experience
  - Blurring of scope of practice?
  - Pain specific education (CMEs, conferences, LEAP, enhanced skills programs)
  - Addiction and withdrawal management services
  - Others?

BARRIERS TO EFFECTIVE PAIN MANAGEMENT: STRATEGIES

- Collaborative care:
  - Multidisciplinary approach is key
  - Social work
  - Psychiatry
  - Counselling services
  - Spiritual care
  - Addiction medicine
  - Pharmacy – drug protocols
  - Nursing – constant in-service of less common interventions
  - Interventional radiology
  - Anaesthesiology
  - Surgical (GI, general surgery, Ortho, Neurosurgery, OB-Gyne, plastics)
    - Anyone with an interest especially when rural

BARRIERS TO EFFECTIVE PAIN MANAGEMENT

What barriers have you encountered?

How has the pandemic impacted your ability to provide optimal pain management?

QUESTIONS?

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