Pain Management in Individuals with Developmental Disabilities

Irene Zamora, MSN, RN, CNS
Certified Pain Management Nurse
RN Educator at UNMH
Online A/GNP Student at UMass/Boston
Objectives

- Discuss the best approach for assessment of pain in individuals with intellectual disabilities
- Differentiate between acute and chronic pain
- Analyze the characteristics of opioids vs. nonopioids
- Clarify terminology surrounding drug abuse and addiction
- Identify at least two strategies for optimum pain management in this patient population
Prevalence of pain in Individuals with ID

- Valkenburg, et al. state that up to 50% of children and adults with Intellectual Disabilities experience gastroesophageal reflux disease
  - Significant pain and discomfort
- Assessment in this special population can be challenging when there is no self report
  - Then what?
- Things to consider....
During Assessment

- Consider biases
  - Age
  - Gender
  - Attractiveness
  - Intellectual and physical abilities
Assessment in the Cognitively Impaired

- Direct observation or history from caregivers
  - Assessment by proxy—nursing assistants or family members or regular caregivers
- Observe during movement (walking, morning care, transfers)
- Unusual behavior should trigger assessment of pain
Pain Indicator for Communicatively Impaired Children (PICIC)

Most common cues identified by 67 parents:

- Screwed up or distressed looking face
- Crying with or without tears
- Screaming, yelling, groaning, moaning
- Stiff or tense body
- Difficult to comfort or console
- Flinches or moves away if touched

Common Pain Behaviors in Cognitively Impaired Elderly Persons

- Facial expressions
- Verbalizations, vocalizations
- Body movements
- Changes in interpersonal interactions
- Changes in activity patterns or routines
- Mental status changes
Vocal complaints: Non-verbal (Expression of pain, not in words, moans, groans, grunts, cries, gasps, sighs)
Facial Grimaces/Winces (Furrowed brow, narrowed eyes, tightened lip jaw drop, clenched teeth, distorted expressions).
Bracing (Clutching or holding onto side rails, bed, tray table, or affected area during movement)
Restlessness (Constant or intermittent shifting of position, rocking, intermittent or constant hand motions, inability to keep still)
Rubbing: (Massaging affected area)
(In addition, record Verbal complaints).
Vocal complaints: Verbal (Words expressing discomfort or pain, "ouch" "that hurts"; cursing during movement, or exclamations of protest “stop”; “that’s enough”)
Other Assessment Scales for Cognitively Impaired

- Pain Indicator for Communicatively Impaired Children
  - 10–49 yrs; non-verbal; indicators—facial, activity, vocal, consolability, physiological, individual indicators (0–24 point scale)

- Non-communicating Children’s Pain Checklist—postoperative version
  - 3–19 years; postop pain; indicators—facial, activity, vocal, social, physiological (27 items on a 0–81 point scale—11/81: mod to severe pain)

- Paediatric Pain Profile—postoperative pain
  - 1–18 years; indicators—facial, activity, vocal, social, consolability, physiological (20 items with 0–60 point scale—14/60: mod or worse pain)
More scales

- Checklist Pain Behavior—postoperative pain
  - 3–19 years; indicators—facial, activity, vocal, physiological; (10 point scale)

- FLACC (Revised Face, Legs, Activity, Cry, Consolability)—postoperative pain
  - 4–19 years; indicators—facial, activity, vocal, social, consolability, physiological and individual indicators; (5 items with 0–10 point scale—4/10: mod pain
Pain can impact
- communication
- Socialization
- Cognitive function
- Combined with fear cognitive function may even be effected more

Greater pain caused greater dysfunction across domains

Pain had a greater impact on individuals with more severe ID

Less-obvious Pain Indicators

- May be attributed to psychosis or dementia
  - Aggressive behavior
  - Fidgeting
  - Noisy breathing
  - Rapid blinking
  - Rigid, tense body posture
- Untreated pain can increase confusion
  - Patients on opioids at risk for dose being cut
Assume Pain is Present

- Assume Pain is Present
- Is there a painful stimulus
  - Surgical incision
  - Fracture
  - Painful procedure
  - Any tissue damage
- If so, treat
  - Observe
Types of Pain

- Nociceptive vs Neuropathic
- Physiologic vs pathophysiologic
- Acute vs chronic
- Malignant vs nonmalignant
- Pain syndromes
Pain resulting from activation of primary afferent nociceptors by mechanical, thermal or chemical stimuli
Pain Mechanisms: The “Pain Process”

- The neural mechanisms by which pain is perceived involve a process that involves four major steps:

1. Transduction
2. Transmission
3. Modulation
4. Perception
Neuropathic pain
Pathophysiologic Pain

- Pain resulting from damage to peripheral nervous or central nervous system tissue or from altered processing of pain in the central nervous system
Neuropathic—Pathophysiologic Pain

- Results in cellular changes that occur in peripheral and central nervous systems
  - Results in sensitization to the transmission of pain signals
- Neuroplasticity—ability of neurons to change their structure and function
- Peripheral and central sensitization—response to stimuli is increased
Result of Central and Peripheral Changes

- Hyperalgesia
  - Primary hyperalgesia
  - Secondary hyperalgesia

- Allodynia

- 'wind-up' of C fibers (a phenomenon of progressively increased neural response to repeated noxious stimuli)
Chronic Pain—Subtypes

- Inflammatory
  - OA (27 million) and RA (1.5 million)
- Neuropathic
  - DN; PHN
- LBP—59 million
- Non-inflammatory, non-neuropathic pain
  - Fibromyalgia—5 million
  - CRPS
RSD/CRPS

- Reflex Sympathetic Disorder/Complex Regional Pain Syndrome
- An extreme example of chronic severe pain
- Can occur after any type of injury—small or large; surgery; burn
- Pain is as severe as the initial injury
- Effects the nervous system and can have swelling, discoloration, sweating to the effected area
- Allodynia is a major symptom
CRPS

http://www.abc.net.au/catalyst/stories/2621515.htm
The ABCs of Pain

**A**ffective Dimension

**B**ehavioral Dimension

**C**ognitive Dimension

**P**hysiological-Sensory Dimension
Definition of Pain

“Unpleasant sensory and emotional experience arising from actual or potential tissue damage or described in terms of such damage (IASP)
Medication Management--Analgesics

Analgesics
Three Types

- Nonopioids (acetaminophen, NSAIDS)
- Opioids (mu agonist, agonist-antagonist)
- Adjuvants (multiple examples) & Anesthetics
Acetaminophen

- Mechanism of action is not certain
- Probably centrally acting—\(?\text{cox}-3\) inhibitor

Acetaminophen toxicity

- Hepatotoxicity
  - Toxic metabolite (NAPQI)
  - Several other mechanisms lead to hepatotoxicity
  - Mechanism not completely understood
- Nephrotoxicity \(>4\text{g/day for long periods}\)
  - Uncertain cause
  - May be caused by activity of NAPQI in renal microsomes
  - Increase frequency to 6–8 hrs in renal failure
NSAIDS

- NSAIDS—Antiinflammatory, antipyretic, analgesic
- Mechanism of action—prostaglandin inhibition by way of COX–1
  - Prostaglandins
    - important in maintaining integrity of GI and duodenal mucosa
    - Important in modulating renal plasma flow
- NSAIDs inhibit formation of thromboxane—effecting platelet aggregation
- Use with caution in pts. with history of asthma
  - Inhibits prostaglandin E—responsible for bronchodialation
Transduction: Nociceptive Chemical Stimuli

Phospholipids released

Arachidonic cascade

5-Lipoxygenase
Prostaglandins

Cyclo-oxygenase

Thromboxane A2 platelet aggregation

PGI2

Vasodilation, Antiaggregation

PGE2

Fever, Pain

PGF2

Vasodilation, Uterine contraction

pain receptor

Trauma

Leukotrienes

Vasodilation

Final: pain receptor
<table>
<thead>
<tr>
<th>Class</th>
<th>Generic name</th>
<th>UAD</th>
<th>Brand name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proprionic acids</td>
<td>Naproxen</td>
<td>500 mg initially-followed by 250mg q6-8h</td>
<td>Naprosyn, Anaprox, Alleve Ansaid</td>
</tr>
<tr>
<td></td>
<td>Flurbiprofen</td>
<td>400-800mgQ6-8h</td>
<td>Daypro Motrin Orudis, Oruvail Toradol</td>
</tr>
<tr>
<td></td>
<td>Oxaprozin</td>
<td>25-75 mg Q6-8h</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ibuprofen</td>
<td>Max 120mg/d (parenteral)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ketoprofen</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Ketorolac</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indoleacetic acids</td>
<td>Sulindac</td>
<td>200mg Q12h</td>
<td>Clinoril Indocin Lodine</td>
</tr>
<tr>
<td></td>
<td>Indomethacin</td>
<td>25-50mg q8h</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Etodolac</td>
<td>200-40mg q6-8h</td>
<td></td>
</tr>
<tr>
<td>Class</td>
<td>Generic name</td>
<td>UAD</td>
<td>Brand name</td>
</tr>
<tr>
<td>------------------------------</td>
<td>-------------------------------------</td>
<td>--------------</td>
<td>-----------------------</td>
</tr>
<tr>
<td>Phenylacetic acids</td>
<td>Diclofenac</td>
<td>50 mg/q8h</td>
<td>Cataflam, Voltaren</td>
</tr>
<tr>
<td>Salicylic acids (nanacetylated)</td>
<td>Salsalate</td>
<td>1000-1500 mg/q12h</td>
<td>Disalcid Trilisate</td>
</tr>
<tr>
<td></td>
<td>Choline magnesium trisalicylate</td>
<td>1000-1500 mg/q12h</td>
<td></td>
</tr>
<tr>
<td>Naphthylalkanone</td>
<td>Nabumetone</td>
<td>1000-2000 mg/day</td>
<td>Relafen</td>
</tr>
<tr>
<td>oxicam</td>
<td>Piroxicam</td>
<td></td>
<td>Feldene</td>
</tr>
</tbody>
</table>
COX–2 Inhibitors

- May have fewer GI effects than COX–1 inhibitors
- Should be avoided in patients with creatinine clearance <30ml/min
  - Carry same risk as traditional NSAIDs
- Celecoxib—Celebrex
  - UAD=100–200 mg q12h max=400 mg/d
CLARIFYING TERMINOLOGY
ADDITION—WHAT IS IT??

- Is it a moral deficit in which the person freely chooses...
- a criminal offense...
- or is it a physiologic disease?
6–10% of the general population has an addiction to illicit drugs, prescribed opioids and alcohol.

In chronic pain populations—6–10%:
- Those with a history of previous addiction.

Chronic pain alone does not add to the risk of addiction.

Rate of addiction in patients without a previous history of addiction when taking opioids for pain remains to be ~1%.
Clarifying Definitions (AAPM, APS, ASAM 2001)

- Physical Dependence: Adaptation that is manifested by a drug class specific withdrawal syndrome that can be produced by abrupt cessation, rapid dose reduction, decreasing blood level of drug and/or administration of an antagonist
- “Dependence” is used by addiction specialists referring to addiction
Definitions

- Tolerance: A state of adaptation in which exposure to a drug induces changes that result in a diminution of one or more of the drug’s effects over time.

- Physical dependence, tolerance and addiction are separate phenomena but may co-exist.
Addiction is defined as a chronic, relapsing brain disease that is characterized by compulsive drug seeking and use, despite harmful consequences.

- National Institute of Health 2010
Behaviors that mimic drug abuse
- Drug seeking
- Clock watching
- Anticipate the next dose
- Demand more pain meds

Due to the undertreatment of pain
- Patients may become deceptive
- May even resort to the purchase of illicit drugs

Distinguished from addiction – analgesia demonstrates improved function and use the med as prescribed not for sedation or euphoria
Clarification of Terms

- Substance misuse
  - Use other than intended purpose
- Substance abuse
  - Use that is unlawful or detrimental
- Diversion
  - Given, sold, or traded to someone other than the patient for whom it was intended
- Nonmedical use
  - Taking the drug for the feeling it gives
“Opiophobia”

Fear that opioids will cause addiction
- Up to 90% of the US population above age 12 has experimented with illicit drugs or alcohol
- Very small percentage go on to develop substance abuse
- Treating pts with a hx of addiction will cause relapse
  - In reality, pain is more likely to cause relapse
DSM-IV describes “Substance Dependence” as

- A maladaptive pattern of substance use manifested by at least 3 of the following occurring any time over a 12-month period
  - Tolerance
  - Withdrawal
  - Taking larger amounts over a longer period than was intended
  - Unsuccessful efforts to cut down
  - A lot of time spent in efforts to obtain the substance
  - Important activities given up because of use
  - Continuation of substance despite knowing that is causing problems
Characteristics of Opioids

- No ceiling effect
- Usually no end organ damage with chronic use
- Metabolized by the liver
  - Metabolite toxicity
    - Avoid using meperidine and propoxyphene
- Excreted by the kidney
- Cause tolerance and physical dependence
- Reversible with an antagonist
- Bind to opiate receptors—μ, κ, δ
- Tolerance to side effects except constipation
Pharmacokinetics

- Absorption
  - Drug solubility—lipophilic vs hydrophilic
- Bioavailability
- First pass Effect
- Solubility
- Metabolism ➔ metabolites, active or inactive
  - Prodrugs, e.g. codeine metabolized by CYP450 enzyme CYP2D6
- Half-life, clearance, steady state and accumulation
Pharmacodynamics

- Opioid responsiveness
  - Efficacy—extent to which a drug “works” (as compared to others)
  - Potency—the dose of a drug required to produce a specified effect, e.g. hydromorphone > potency than morphine
  - Opioid responsiveness—affected by age, organ dysfunction

- Tolerance—rule out disease progression; compliance to tx
  - OIH—rare
  - Incomplete Cross-tolerance—due to receptor subtypes—reduce new opioid 25% – 50% calculated equianalgesic dose (methadone dec. by as much as 90% then titrate as needed)

- Physical dependence
PRN

- What does “PRN” mean?
- If pain is ongoing give opioids ATC
- Half-life
- Steady state
- Time to peak effect
Opioids
Mu–agonists

Bind to mu opiate receptors blocking transmission of pain

- Morphine
- Fentanyl
- hydromorphone
- oxycodone
- hydrocodone
- Codeine
- *Methadone
- *meperedine
- *tramadol
Morphine

- Hydrophilic—delayed onset and longer duration
- Two metabolites but only one active at opioid receptor—morphine-6-glucuronide (M6G)—analgesic
  - Patients with renal impairment should start at $\frac{1}{4}$ dose and titrate as needed
    - Accumulation results in neurologic side effects as well
    - Removed with dialysis
Hydromorphone (Dilaudid)

- Hydrophyllic—similar to morphine
  - IV—1.5 mg:10 mg morphine/PO—7.5 mg:30 mg morphine
    - Onset 5 min; peak in 8–20 min. duration ~ 4 hrs
  - Oral
    - 60% bioavailable; onset 30 min. duration 3–4 hrs
  - Metabolized in the liver
  - Several metabolites

- Use decreased amounts in renal impairment due to possible sensitivity to hydromorphone–3-glucuronide possible neuroexcitation
  - there is no 6–glucuronide so may have fewer SEs

- May be safer than morphine in renal insufficiency
Fentanyl

- Lipophillic → Short half-life, short duration of action
  - UNLESS given regularly—then half-life is extended
- No active metabolites
- Safer in renal and liver failure
- Fewer side effects
- Half-life extends with continuous use
- Multiple formulations
  - transdermal, oral transmucosal, buccal
oxycodone

- Available in combination or single-entity
  - Short and Long-acting
- More potent than morphine
- Metabolized in the liver by cytochrome CYP2D6
  - Multiple metabolites
- Binds at $\mu$ and $\kappa$ receptors—may be better in chronic pain states
  - Half-life and bioavailability slightly longer than MS
  - One active metabolite—oxymorphone
  - Women may have a greater effect
  - Excretion impaired in uremic patients and
    - Elimination half-life is severely impaired in these patients
  - May cause CNS toxicity and sedation in renal failure
Hydrocodone (Vicodin)

- Only available in combination with acetaminophen, ibuprofen, aspirin
  - Onset 20 min. peak by 60 min; half-life 3.8 hrs
- Metabolized in the liver
  - Several metabolites
- Significant renal excretion of active forms
- Should be avoided in patients with renal failure
- Adverse effect – hearing loss
Demerol (meperidine)

- Half-life is 2–3 hrs (parenterally)
- Bioavailability from p.o. is ¼ that of parenteral
- Onset 10 minutes; peak effect 30 min. duration up to 4 hrs
- More likely than other opioid drugs to cause delirium in postop pts of all ages
  - More nausea and vomiting
- Limit use to 600 mg/d and no more than 48 hours due to metabolite—normeperidine
- Observe for signs of neuroexcitation—restlessness, shakiness, tremors, twitching and jerking
- Misconception—produces less biliary spasm than other opioids—all opioids can produce this
Normeperidine—only active metabolite of meperidine
- toxic metabolite
- half-life 15–20 hrs
- causes neuroexcitation—hyperreflexia, myoclonus, agitation and grand mal seizures
- half analgesic potency but twice the toxicity
- Is not reversed with naloxone and may increase risk of seizures if naloxone given

Use extreme caution in patients with seizure disorder

Use caution in patients with renal insufficiency

Contraindicated with MAOI (monoamine oxidase inhibitors)—can cause serotonin syndrome or death
Codeine

- 60mg = 600 mg of aspirin
- Not appropriate for moderate to severe pain
- Usually more constipating
- Has more psychotomimetic effects
- Metabolized in the liver to morphine
  - Several metabolites
  - Metabolism is necessary for analgesia
  - Poor metabolizers may show absence of analgesia
- Reduced renal clearance in advanced renal failure
  - Reports of serious adverse effects in renal failure
Inexpensive
Adverse effects similar to other opioids
Rapid onset—30–60 minutes; duration 4–6 hrs; peak effect 2.5 hrs
~ 80% bioavailability
No active metabolites
Long duration with continued use
No ceiling dose other than side effects
Has some SSRI and NMDA antagonist activity
For opioid naïve patients ➔ start at 2.5mg Q8H
Excreted in feces—considered safe in renal insufficiency
Methadone—not so good news

- Long half-life—15–60 hours—
  - Unpredictable
  - difficult to titrate
  - Difficult to convert from other opioids to methadone
- Duration initially is 3–6 hrs → 8–12 hr with repeated dosing
- Varied inter-individual effects
- Efficacy is greater with repeated dosing
- Multiple drug–drug interactions that can induce or inhibit effect by other drug or be effected
  - Close observation is required
Propoxyphene (Darvocet)

- REMOVED FROM THE MARKET IN 2011—YAY!
  - Was removed from the market in the UK many years ago
Dual-mechanism Analgesics

- Tramadol—for mod to moderately severe pain
  - Weak mu-agonist and norepinephrine and serotonin reuptake inhibitory activity similar to TCAs
  - Peak effect in ~ 2 hrs of 100mg dose
  - Potency equivalent to codeine and five times less potent than morphine
  - Ceiling effect
  - Max dose is 400mg/24h
  - Use with caution in pts w seizures or on SSRIs

- Tapentadol – Nucynta
  - Agonist at mu and blocks reuptake of norepinephrine
  - Schedule II drug
  - Indicated for mod–severe pain
  - Avoid combining with SSRIs
Titration of Opioids

- Based on effect
  - Increase dose 25%-100%
    - Ask patient how much pain was relieved by last dose
- Estimate 24 hr total and change to long-acting formula...for example
  - 2 tabs 5/325 Percocet Q4H → 20 mg OxyContin Q8H
Equianalgesic Dosing Guidelines

- Equianalgesic means approximately the same pain relief.
- The chart is a guideline. Titrate meds according to pt’s response.
- Chart is helpful when switching from one drug to another or when switching to another route.
- Dosages are not necessarily starting doses.
- Consider incomplete cross-tolerance.
<table>
<thead>
<tr>
<th>Drug</th>
<th>Oral Dose</th>
<th>IV Dose</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Morphine</td>
<td>30 mg</td>
<td>10 mg</td>
<td>3-5 hours</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>Breakthrough only (OTFC)</td>
<td>100mcg (0.1mg) 100 mcg/h TD ≈ 4 mg/h IV MS; 1mcg/h TD ≈ 2 mg/24 h oral MS</td>
<td>0.5-1 hour</td>
</tr>
<tr>
<td>Hydromorphone</td>
<td>7.5 mg</td>
<td>1.5 mg</td>
<td>2-4 hours</td>
</tr>
<tr>
<td>Meperidine</td>
<td>300 mg NR</td>
<td>75-100 mg</td>
<td>2-4 hours</td>
</tr>
<tr>
<td>Codeine</td>
<td>200 mg NR</td>
<td>130 mg</td>
<td>3-4 hours</td>
</tr>
<tr>
<td>Methadone</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Oxycodone</td>
<td>20-30 mg</td>
<td>----------</td>
<td>3-4 hours</td>
</tr>
<tr>
<td>Hydrocodone</td>
<td>30 mg</td>
<td>----------</td>
<td>3-4 hours</td>
</tr>
<tr>
<td>Nalbuphine</td>
<td>----------</td>
<td>10 mg</td>
<td>3-6 hours</td>
</tr>
</tbody>
</table>
Opioid Side Effects—Are all self-limiting except....

- Constipation
- Nausea and vomiting
- Pruritus
- Urinary retention
- Mental status changes
- Sedation
- Respiratory depression
PREDISPOSING FACTORS TO RESPIRATORY DEPRESSION

- Sedation
- Large doses of opioids
- Concomitant use of opioids and CNS depressants
- High-risk patients
  - obese
  - hx of pulmonary disease
  - hx of sleep apnea
  - advanced age (>65 years)
- Rarely seen in chronic pain management
Adjuvant Analgesics

- Medications that are typically used for another purpose
- Two classes
  - Multipurpose – acute and chronic pain
  - For specific types of pain
First-line Drugs in Chronic Pain

- gabapentin (Neurontin)—start w/ 100–300 mg/day Usual Effective Dose (UED) 300–3600 q8h
- pregabalin (Lyrica)—start with 100–150 mg/day; UED 150–600 q12h
- SNRI
  - Duloxetine (Cymbalta)—start w/ 30 mg/day; UED 60 mg q12h
ADJUVANT ANALGESICS: MAJOR CLASSES

- Anticonvulsants
- Antidepressants
- Psychostimulants
- Muscle relaxants
- Sedatives
Opioid Side Effects

- Nausea and vomiting
- Pruritus
- Urinary retention
- Mental status changes
- Sedation
- Respiratory depression
Opioid Induced Constipation

- The hand that writes the prescription for an opioid and
- Fails to write the order for a laxative should be
- The hand that removes the impaction
Adjuvant Examples

- Antidepressants
  - SSRIs, SSNRIIs, TCAs
- Anti-convulsants
- Corticosteroids
- Alpha-2 adrenoceptor agonists
- Anti-histamines

- Anti-spastics
- Muscle Relaxants
- NMDA receptor antagonists
Balanced Analgesia

- Inter-disciplinary approach
  - Medication management
  - Physical activity
  - Maximize nutritional contributions
  - Mental health
  - Support group
  - Spirituality
Non-Pharmacologic Interventions

- Increase activity
- Individualize interventions
  - music
  - artwork
  - humor
- Address constipating effects of opioids
Goal Setting

- Once assessment complete, discuss pain level and related goals with patient & family
- Should be based on functionality
- Decrease suffering
- Be realistic
- Be patient
- Patient and family education—why pain management
  - Minimize risk of complications
  - Myths about addiction
Documentation

- Analgesia
- Adverse effects
- ADLs
- Aberrhant behavior
In Summary

- Treat pain initially aggressively—
  - ***Titrate to Effect
- Adequate analgesia results in:
  - Early participation in activity
  - Prevention of complications
  - Decrease risk of chronic pain
  - Early return to individual level of functioning
- Use assessment tool specific to population
- Always combine modalities— opioids with nonopioids and pharmacologic with non-pharmacologic