MANAGEMENT
Goals of Treatment
Goals in Pain Management

• Involve the patient in the decision-making process
• Agree on realistic treatment goals before starting a treatment plan

- Optimized pain relief
- Improved function
- Minimized adverse effects

# Pain Should Be Treated in a Timely Manner

<table>
<thead>
<tr>
<th>IASP Recommendations for Wait Times</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Wait time</strong></td>
</tr>
<tr>
<td>Treat immediately</td>
</tr>
</tbody>
</table>
| 1 week (most urgent) | • Painful severe condition with risk of deterioration or chronicity  
| | • Pain in children  
| | • Pain related to cancer or terminal or end-stage illness |
| 1 month (urgent or semi-urgent) | • Severe undiagnosed or progressive pain with risk of increasing functional impairment, generally of 6 months’ duration or less |
| 8 weeks (routine or regular) | • Persistent long-term pain without significant progression |

IASP = *International Association for the Study of Pain*


Deciding on the Best Course of Treatment for the Patient

Collaborative Care

- Patient as the ultimate manager of his/her illness
- General practitioner ± other health care professional(s)
- Family

Treatments for Pain

- Medications
- Regional anesthetic interventions
- Surgery
- Psychological therapies
- Rehabilitative/physical therapies
- Complementary and alternative medicine
Multimodal Treatment of Pain Based on Biopsychosocial Approach

- Pharmacotherapy
- Stress management
- Interventional pain management
- Biofeedback
- Complementary therapies
- Education
- Biofeedback
- Lifestyle management

- Physical therapy
- Occupational therapy
- Sleep hygiene

Non-pharmacological Treatment
Non-pharmacological Interventions

- Non-pharmacological interventions are commonly used in clinical practice
- Establishing reliable evidence of efficacy and effectiveness can be challenging in terms of design and interpretation of studies

<table>
<thead>
<tr>
<th>Type of therapy</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Psychological</td>
<td>• Hypnosis&lt;br&gt;• Relaxation&lt;br&gt;• Cognitive behavioral therapy</td>
</tr>
<tr>
<td>Physical</td>
<td>• Acupuncture&lt;br&gt;• Transcutaneous electrical nerve stimulation&lt;br&gt;• Healing touch and massage&lt;br&gt;• Occupational therapy</td>
</tr>
<tr>
<td>Clinical process</td>
<td>• Pain assessment&lt;br&gt;• Physician advice and communication&lt;br&gt;• Education</td>
</tr>
</tbody>
</table>

Psychological Therapies

- Individual and group counseling
- Biofeedback
- Relaxation techniques
- Self-hypnosis
- Visual imaging
- Learning or conditioning techniques
- Behavioral techniques
- Cognitive techniques
- Psychotherapy

Rehabilitative/Physical Therapies

- Heat
- Deep heat (ultrasound)
- Cryotherapy
- Aquatic therapy
- Transcutaneous electrical nerve stimulation
- Iontophoresis and phonophoresis
- Traction
- Exercise
- Manual therapy
- McKenzie method
- Core stabilization
What is complementary and alternative medicine?

A group of diverse medical and health care systems, practices, and products that are not generally considered part of conventional medicine.

– NCCAM definition
## Evidence of Potential Benefits of Complementary and Alternative Medicine

<table>
<thead>
<tr>
<th>Procedure/Medicine</th>
<th>Arthritis</th>
<th>Headache</th>
<th>Low back pain</th>
<th>Neck pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acupuncture</td>
<td>√</td>
<td>√</td>
<td>√</td>
<td>X</td>
</tr>
<tr>
<td>Balneotherapy (mineral baths)</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Feverfew</td>
<td></td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gamma linoleic acid</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glucosamine/chondroitin</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Herbal remedies</td>
<td>X</td>
<td></td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Massage</td>
<td></td>
<td></td>
<td>√</td>
<td></td>
</tr>
<tr>
<td>Spinal manipulation</td>
<td></td>
<td></td>
<td>√</td>
<td>X</td>
</tr>
<tr>
<td>Progressive relaxation</td>
<td></td>
<td></td>
<td>√</td>
<td></td>
</tr>
<tr>
<td>Prolotherapy</td>
<td></td>
<td></td>
<td></td>
<td>X</td>
</tr>
<tr>
<td>Tai chi</td>
<td></td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yoga</td>
<td></td>
<td></td>
<td></td>
<td>√</td>
</tr>
</tbody>
</table>

**Legend:** √ = promising evidence of potential benefit; X = limited, mixed or no evidence to support use

Treating Pain: 
Use a Mind-Body Approach

- Biopsychosocial approach to assessing and treating chronic pain offers a uniquely valuable clinical perspective
- Mind-body perspective now generally accepted by pain researchers
- Found to be useful by clinicians in various disciplines, such as osteopathic medicine, rheumatology, and physiotherapy

Pharmacological Treatment
Analgesics Affect Different Parts of the Pain Pathway

Ascending input

Descending modulation

Dorsal horn

Dorsal root ganglion

Spinothalamic tract

Peripheral nerve

Peripheral nociceptors

Local anesthetics

Antidepressants

nsNSAIDs/coxibs

Opioids

Local anesthetics

α₂δ ligands

Antidepressants

nsNSAIDs/coxibs

Opioids

Coxib = COX-2 inhibitor; nsNSAID = non-specific non-steroidal anti-inflammatory drug

Mechanism-Based Pharmacological Treatment of Nociceptive/Inflammatory Pain

Noxious stimuli

Transduction

Peripheral sensitization

Inflammation

nsNSAIDs/coxibs, opioids

nsNSAIDs/coxibs

Local anesthetics

\( \alpha_2\delta \) ligands

Acetaminophen

Antidepressants

Opioids

Descending modulation

Ascending input

Spinal cord

Brain

Perception

Opioids

Coxib = COX-2 inhibitor; nsNSAID = non-specific non-steroidal anti-inflammatory drug

Acetaminophen

• Action at molecular level is unclear

• Potential mechanisms include:
  – Inhibition of COX enzymes (COX-2 and/or COX-3)
  – Interaction with opioid pathway
  – Activation of serotoninergic bulbospinal pathway
  – Involvement of nitric oxide pathway
  – Increase in cannabinoid-vanilloid tone

What are NSAIDs (nsNSAIDs/coxibs)?

**NSAID = Non-Steroidal Anti-Inflammatory Drug**

- Analgesic effect via inhibition of prostaglandin production
- Broad class incorporating many different medications:

**Examples of nsNSAIDs:**
- Diclofenac
- Ibuprofen
- Naproxen

**Examples of Coxibs:**
- Celecoxib
- Etoricoxib
- Parecoxib

ASA = acetylsalicylic acid; coxib = COX-2-specific inhibitor; nsNSAID = non-specific non-steroidal anti-inflammatory drug

How do nsNSAIDs/coxibs work?

Coxib = COX-2-specific inhibitor; NSAID = non-steroidal anti-inflammatory drug
nsNSAID = non-specific non-steroidal anti-inflammatory drug

COX-2 Is Expressed in the CNS

• Prostaglandins in the CNS are important in central sensitization and hyperalgesia\(^1\)

• Peripheral inflammation leads to central induction of COX-2\(^2\)
  – Occurs even with complete sensory nerve block\(^3\)
  – Humoral signal (IL-6?) may play a role in signal transduction across blood-brain barrier\(^3\)
  – IL-1beta plays an important role centrally\(^3\)
  – Elevation of prostaglandins in CSF lead to hyperalgesia\(^3\)
  – Inhibition of IL-1beta synthesis or receptors reduce CSF levels of COX-2, prostaglandin and hyperalgesia\(^3\)
  – Inhibition of COX-2 centrally has similar effects\(^3,4\)

CNS = central nervous system; CSF = cerebrospinal fluid; IL = interleukin
COX-2 Results in Sensitization to Pain

• Peripheral Sensitization
  – COX-2 is expressed following tissue injury
  – Prostaglandins produced increase nociceptor sensitivity to pain

• Central Sensitization
  – Peripheral inflammation leads to induction of COX-2 in CNS
  – Occurs even with complete sensory nerve block, possibly due to a humoral signal
  – Prostaglandins produced by COX-2 in CNS cause further sensitization to pain

• Result: hyperalgesia and allodynia

CNS = central nervous system
COX-2 Is Involved in Central Sensitization

- Central induction of COX-2 result in increased prostaglandin production
- PGE2 stimulation of EP receptors in the dorsal horn will:
  - Activate PKC, phosphorylating and further enhancing NMDA channel opening
  - Directly activate certain dorsal horn neurons by opening EP2 receptor linked ion channels
  - Reduced inhibitory transmission of glycinenergic inter-neurons
  - Increased depolarization and excitability of dorsal horn neurons

NMDA = N-methyl-D-aspartate; PGE2 = prostaglandin E2; PKC = protein kinase C
COX-2 Inhibition Minimizes Sensitization

- Signal for COX-2 induction likely to persist with peripheral inflammation
- To minimize sensitization, COX-2 should be inhibited centrally and in the periphery
  - As early as possible
  - Continued until peripheral inflammation resolved
- Ideal COX-2 inhibitor should be able to act in periphery as well as centrally
  - Should readily cross blood-brain barrier

Adverse Effects of nsNSAIDs/Coxibs

All NSAIDs:
• Gastroenteropathy
  – Gastritis, bleeding, ulceration, perforation
• Cardiovascular thrombotic events
• Renovascular effects
  – Decreased renal blood flow
  – Fluid retention/edema
  – Hypertension
• Hypersensitivity

Cox-1-mediated NSAIDs (nsNSAIDs):
• Decreased platelet aggregation

Coxib = COX-2-specific inhibitor; NSAID = non-steroidal anti-inflammatory drug; nsNSAID = non-specific non-steroidal anti-inflammatory drug
Composite includes non-fatal myocardial infarction, non-fatal stroke, or cardiovascular death compared with placebo; chart based on network meta-analysis involving 30 trials and over 100,000 patients.

Coxib = COX-2 inhibitor; nsNSAID = non-specific non-steroidal anti-inflammatory drug

Gastrointestinal Risk with nsNSAIDs/Coxibs

Pooled Relative Risks and 95% CIs of Upper Gastrointestinal Complications

CI = confidence interval; coxib = COX-2 inhibitor; NSAID = non-steroidal anti-inflammatory drug; nsNSAID = non-specific non-steroidal anti-inflammatory drug

### Risk Factors for Gastrointestinal Complications Associated with nsNSAIDs/Coxibs

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Odds Ratio/Relative Risk for Ulcer Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of GI bleeding/perforation</td>
<td>13.5</td>
</tr>
<tr>
<td>Concomitant use of anticoagulants</td>
<td>6.4</td>
</tr>
<tr>
<td>History of peptic ulcer</td>
<td>6.1</td>
</tr>
<tr>
<td>Age ≥60 years</td>
<td>5.5</td>
</tr>
<tr>
<td>Single or multiple use of NSAID</td>
<td>4.7</td>
</tr>
<tr>
<td>Helicobacter pylori infection</td>
<td>4.3</td>
</tr>
<tr>
<td>Use of low-dose ASA within 30 days</td>
<td>4.1</td>
</tr>
<tr>
<td>Alcohol abuse</td>
<td>2.4</td>
</tr>
<tr>
<td>Concomitant use of glucocorticoids</td>
<td>2.2</td>
</tr>
<tr>
<td>Smoking</td>
<td>2.0</td>
</tr>
</tbody>
</table>

ASA = acetylsalicylic acid; coxib = COX-2-specific inhibitor; GI = gastrointestinal; NSAID = non-steroidal anti-inflammatory drug; nsNSAID = non-specific non-steroidal anti-inflammatory drug; SSRI = selective serotonin reuptake inhibitor

# Guidelines for nsNSAIDs/Coxibs Use Based on Gastrointestinal Risk and ASA Use

<table>
<thead>
<tr>
<th>Gastrointestinal risk</th>
<th>Not elevated</th>
<th>Elevated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Not on ASA</td>
<td>nsNSAID alone</td>
<td>Coxib nsNSAID + PPI</td>
</tr>
<tr>
<td>On ASA</td>
<td>Coxib + PPI nsNSAID + PPI</td>
<td>Coxib + PPI nsNSAID + PPI</td>
</tr>
</tbody>
</table>

ASA = acetylsalicylic acid; coxib = COX-2-specific inhibitor; nsNSAID = non-selective non-steroidal anti-inflammatory drug; PPI = proton pump inhibitor

How Opioids Affect Pain

Modify perception, modulate transmission and affect transduction by:

- Altering limbic system activity; modify sensory and affective pain aspects
- Activating descending pathways that modulate transmission in spinal cord
- Affecting transduction of pain stimuli to nerve impulses

# Opioids and Pain Management

<table>
<thead>
<tr>
<th>Opioid Receptor</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mu</strong></td>
<td>Supraspinal analgesia, respiratory depression, sedation, miosis, euphoria, cardiovascular effects, pruritis, nausea/vomiting, decreased gastrointestinal motility, dependence, tolerance</td>
</tr>
<tr>
<td><strong>Delta</strong></td>
<td>Analgesia, euphoria, dysphoria, psychotomimetic effects</td>
</tr>
<tr>
<td><strong>Kappa</strong></td>
<td>Spinal analgesia, dysphoria, psychotomimetic effects, miosis, respiratory depression, sedation</td>
</tr>
</tbody>
</table>
Opioids Modulate Control of “ON” and “OFF” Cells

- Opioid stimulation of mu-receptors on “ON” cells
  - Reduced “ON” cell activity
  - Reduced facilitation of pain transmission at dorsal horn
  - Less pain

- Opioid stimulation of mu-receptors on GABA-ergic interneurons innervating “OFF” cells
  - Reduced GABA-ergic interneuron activity
  - Reduced inhibition of “OFF” cells
  - Increased “OFF” cell inhibition of pain transmission at dorsal horn
  - Less pain

GABA = γ-aminobutyric acid

Opioids Can Induce Hyperalgesia

- **Primary hyperalgesia**
  - Sensitization of primary neurons → decrease threshold to noxious stimuli within site of injury
  - May include response to innocuous stimuli
  - Increase pain from suprathreshold stimuli
  - Spontaneous pain

- **Secondary hyperalgesia**
  - Sensitization of primary neurons in surrounding uninjured areas
  - May involve peripheral and central sensitization

Opioids Can Induce Allodynia

- Pain evoked by innocuous stimuli
- Central sensitization → pain produced by Aβ fibers
- Possibly mediated by spinal NMDA receptors

NMDA = N-methyl-D-aspartate
# Adverse Effects of Opioids

<table>
<thead>
<tr>
<th>System</th>
<th>Adverse effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrointestinal</td>
<td>Nausea, vomiting, constipation</td>
</tr>
<tr>
<td>CNS</td>
<td>Cognitive impairment, sedation, lightheadedness, dizziness</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Respiratory depression</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>Orthostatic hypotension, fainting</td>
</tr>
<tr>
<td>Other</td>
<td>Urticaria, miosis, sweating, urinary retention</td>
</tr>
</tbody>
</table>

*CNS = central nervous system*

Mechanism-Based Pharmacological Treatment of Neuropathic Pain

Medications affecting peripheral sensitization:
- Capsaicin
- Local anesthetics
- TCAs

Medications affecting descending modulation:
- SNRIs
- TCAs
- Tramadol, opioids

Medications affecting central sensitization:
- $\alpha_2\delta$ ligands
- TCAs
- Tramadol, opioids

SNRI = serotonin-norepinephrine reuptake inhibitor; TCA = tricyclic antidepressant

Role of $\alpha_2\delta$-Linked Calcium Channels in Neuropathic Pain

- Increased numbers of calcium channels
- Increased calcium influx
- Increased neuronal excitability

INCREASED PAIN SENSITIVITY

Binding of $\alpha_2\delta$ ligands to $\alpha_2\delta$ inhibits calcium channel transport

Calcium channels transported to nerve terminals in dorsal horn

Injury stimulates production of calcium channel

Nerve injury

Note: gabapentin and pregabalin are $\alpha_2\delta$ ligands
$\alpha_2\delta$ Ligands Bind to $\alpha_2\delta$ Subunit of Voltage-Gated Calcium Channels

Note: gabapentin and pregabalin are $\alpha_2\delta$ ligands
**α₂δ Ligands Reduce Calcium Influx in Depolarized Human Neocortex Synaptosomes**

<table>
<thead>
<tr>
<th>Concentration (μM)</th>
<th>Ca²⁺ fluorescence (% of control)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vehicle</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>*</td>
</tr>
<tr>
<td>100</td>
<td><strong>p &lt;0.05 vs. vehicle</strong></td>
</tr>
<tr>
<td>1,000</td>
<td></td>
</tr>
</tbody>
</table>

*Fink K et al. Neuropharmacology 2002; 42(2):229-36.*
$\alpha_2\delta$ Ligands Modulate Calcium Channel Trafficking

- $\alpha_2\delta$ ligands reduce trafficking of voltage-gated calcium channel complexes to cell surface \textit{in vitro}
- $\alpha_2\delta$ ligands prevent nerve-injury induced up-regulation of $\alpha_2\delta$ in the dorsal horn

BCH = 2-(-)-endoamino-bicycloheptene-2-carboxylic acid; ER = endoplasmic reticulum; GBP = gabapentin
### Adverse Effects of $\alpha_2\delta$ Ligands

<table>
<thead>
<tr>
<th>System</th>
<th>Adverse effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Digestive system</td>
<td>Dry mouth</td>
</tr>
<tr>
<td>CNS</td>
<td>Dizziness, somnolence</td>
</tr>
<tr>
<td>Other</td>
<td>Asthenia, headache, peripheral edema, weight gain</td>
</tr>
</tbody>
</table>

$\alpha_2\delta$ ligands include gabapentin and pregabalin

CNS = central nervous system

Inhibiting reuptake of serotonin and norepinephrine enhances descending modulation.
# Suggested Mechanisms of Analgesic Action of Antidepressants

<table>
<thead>
<tr>
<th>Mechanism of Action</th>
<th>Site of Action</th>
<th>TCA</th>
<th>SNRI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reuptake inhibition</td>
<td>Serotonin, Noradrenaline</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(+) milnicipran</td>
</tr>
<tr>
<td>Receptor antagonism</td>
<td>α-adrenergic, NMDA</td>
<td>+</td>
<td>-</td>
</tr>
</tbody>
</table>
| Blocking or activation of ion channels | Sodium channel blocker                       | +   | (+) venlafaxine/-duloxetine?
|                                      | Calcium channel blocker                       |     |                          |
|                                      | Potassium channel activator                   | +   | ?                        |
| Increasing receptor function         | GABA<sub>B</sub> receptor                    | + amitripline/desipramine | ?                         |
| Opioid receptor binding/mediated effect | Mu- and delta-opioid receptor                  | (+) | (+) venlafaxine          |
| Decreasing inflammation              | Decrease of PGE2 production, decrease of TNFα production |     |                          |

GABA = γ-aminobutyric acid; NDMA = N-methyl-D-aspartate; PGE = prostaglandin E; SNRI = serotonin-norepinephrine reuptake inhibitor; TCA = tricyclic antidepressant; TNF = tumor necrosis factor

## Adverse Effects of Antidepressants

<table>
<thead>
<tr>
<th>System</th>
<th>TCAs</th>
<th>SNRIss</th>
</tr>
</thead>
<tbody>
<tr>
<td>Digestive system</td>
<td>Constipation, dry mouth, urinary retention</td>
<td>Constipation, diarrhea, dry mouth, nausea, reduced appetite</td>
</tr>
<tr>
<td>CNS</td>
<td>Cognitive disorders, dizziness, drowsiness, sedation</td>
<td>Dizziness, somnolence</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>Orthostatic hypotension, palpitations</td>
<td>Hypertension</td>
</tr>
<tr>
<td>Other</td>
<td>Blurred vision, falls, gait disturbance, sweating</td>
<td>Elevated liver enzymes, elevated plasma glucose, sweating</td>
</tr>
</tbody>
</table>

*CNS = central nervous system; TCA = tricyclic antidepressant; SNRI = serotonin-norepinephrine reuptake inhibitor*

Assessment of Pain Pathophysiology Can Help Guide Appropriate Medication Therapy

- Nociceptive pain
- Neuropathic and central sensitization/dysfunctional pain

Opioids
For management of moderate to severe pain in appropriate patients

- Acetaminophen
- nsNSAIDs/coxibs

- α₂δ ligands
- Antidepressants

Most opioid treatment guidelines for chronic pain recommend use for patients after inadequate response to non-opioid therapy*

*Selected on the basis of the pathophysiology of patient’s pain, provided there are no contraindications for its use
Coxib = COX-2-specific inhibitor;
nsNSAID = non-specific non-steroidal anti-inflammatory drug
But... Patients with Chronic Pain of Just One Type of Pain Pathophysiology May be Rare

- Patients may have different pathophysiological mechanisms contributing to their pain
  - e.g., complex regional pain syndrome has multiple potential mechanisms, including nerve injury and inflammation – “mixed pain state”

- Therapies that will work better for a particular patient are likely to depend on the mechanisms contributing to the patient’s pain

- Patients with mixed pain may benefit from combination therapy
Adherence
Non-adherence to chronic pain medication is common...

Overall non-adherence: 36-81%

Overuse: 3-75%
Underuse: 2-51%
Misuse: 13-32%

But rates vary substantially from study to study

Demographic and Medication-Related Factors Can Help Predict Non-adherence

- Younger age
- Health insurance compensation
- Cigarette smoking
- Self-medication
- Greater number of prescribed analgesics
- Greater number of pills to be taken

Non-adherence Is Also Related to Patient Concerns

<table>
<thead>
<tr>
<th>Type of non-adherence</th>
<th>Level of pain</th>
<th>Perceived need</th>
<th>Mistrust in doctor</th>
<th>Side effects</th>
<th>Concern over withdrawal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-adherence</td>
<td>NS</td>
<td>NS</td>
<td>$p &lt; 0.01$</td>
<td>$p &lt; 0.01$</td>
<td>$p &lt; 0.001$</td>
</tr>
<tr>
<td>Overuse</td>
<td>NS</td>
<td>$p &lt; 0.001$</td>
<td>NS</td>
<td>$p &lt; 0.05$</td>
<td>NS</td>
</tr>
<tr>
<td>Underuse</td>
<td>$p &lt; 0.05$</td>
<td>NS</td>
<td>$p &lt; 0.01$</td>
<td>NS</td>
<td>$p &lt; 0.01$</td>
</tr>
</tbody>
</table>

NS = non-significant
Strategies to Improve Adherence

- **S**implify regimen
- **I**mpart knowledge
- **M**odify patient beliefs and human behavior
- **P**rovide communication and trust
- **L**eave the bias
- **E**valuate adherence

Simplifying Medication Regimen

• If possible, adjust regimen to minimize:
  – Number of pills taken
  – Number of doses per day
  – Special requirements (e.g., bedtime dosing, avoiding taking medication with food, etc.)

• Recommend all medications be taken at the same time of day (if possible)

• Link taking medication to daily activities, such as brushing teeth or eating

• Encourage use of adherence aids such as medication organizers and alarms

Imparting Knowledge

- Provide clear, concise instructions (written and verbal) for each prescription
- Be sure to provide information at a level the patient can understand
- Involve family members if possible
- Provide handouts and/or reliable websites for patients to access information on their condition
- Provide concrete advice on how to cope with medication costs

## Modifying Patient Beliefs and Behaviors: Motivational Interviewing Technique

<table>
<thead>
<tr>
<th>Techniques</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Express empathy</td>
<td>• “It’s normal to worry about medication side effects”</td>
</tr>
<tr>
<td>• Develop discrepancy</td>
<td>• “You obviously care about your health; how do you think not taking your pills is affecting it?”</td>
</tr>
<tr>
<td>• Roll with resistance</td>
<td>• “I understand that you have a lot of other things besides taking pills to worry about”</td>
</tr>
<tr>
<td>• Support self efficacy</td>
<td>• “It sounds like you have made impressive efforts to work your new medication into your daily routine”</td>
</tr>
</tbody>
</table>

Providing Communication and Trust: Communication Tips

• Be an active listener
  – Focus on the patient
  – Nod and smile to show you understand
• Make eye contact

• Be aware of your own body language
  – Face the patient
  – Keep arms uncrossed
  – Remove hands from pockets
• Recognize and interpret non-verbal cues

McDonough RP, Bennett MS. *Am J Pharm Educ* 2006; 70(3):58;
Leaving the Bias

Learn more about how low health literacy can affect patient outcomes.

Specifically ask about attitudes, beliefs and cultural norms with regards to medication.

Tailor communication to patient’s beliefs and level of understanding.

Acknowledge biases.

Evaluating Adherence: 4-Step Strategy for Detecting Non-adherence

1. Ask an open-ended question about taking medicine

2. Normalize and universalize non-adherence to reverse the judgmental environment

3. Make the role of accurate information about adherence in medical decision-making explicit

4. Don’t ask about “forgetting” or “missed” doses until the first 3 steps have set the stage

Summary
Management: Summary

- It can be challenging to choose the best treatment for chronic and acute pain
- An approach combining physical and psychosocial interventions is recommended
- Choice of pharmacotherapy may be guided in part by the type(s) of pain
- Adherence to therapy is necessary for optimal patient outcomes