Pain Management as a Vital Component of Wound Care

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Loews Philadelphia Downtown
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The Opioid Abuse Epidemic- the Need To Build A Better Toolbox

A man who cannot work without his hypodermic needle is a poor doctor. The amount of narcotic you use is inversely proportional to your skill. — Martin H. Fischer


Wound Care Providers Encounter Patients with Multiple Types of Pain Requiring Interventions

Etiologies for Wound-Related Pain

**Background Pain** intermittent or continuous pain at the wound site, even at rest from underlying acute or chronic diseases.

**Incident Pain** wound site pain occurs throughout the day during simple activities such as sneezing, coughing, walking or changing position.

**Procedural Pain** is directly related to routine activities and office procedures like wound dressing changes, debridement or biopsies.

**Post-Operative or Post-Traumatic Pain** related to significant soft and/or hard tissue injury or manipulation, like a surgical intervention, crush, burn, fracture or amputation.

World Health Organization for the Management of Pain- WHO Ladder Staged Advanced Analgesics

World Health Organization for the Management of Pain

- **WHO Ladder Staged Advanced Analgesics**
  - Pain persisting or increasing
    - Opioid for moderate to severe pain
    - Non-Opioid
    - Adjunctive Therapies
  - Pain persisting or increasing
    - Opioid for mild to moderate pain
    - Non-Opioid
    - Adjunctive Therapies

Joint Commissions (JCAHO) Pain Management Standards Establish Wound Care Excellence Criteria

JCAHO Standards PC.02.01.07 Pain Assessment and Management with 2015 Clarification


- Recognize the right for appropriate assessment and management of pain- train providers
- Screen for pain during initial assessment and, when clinically required, during ongoing, periodic re-assessments
- Educate patients suffering from pain, and their families, about pain management
- Both pharmacologic and non-pharmacologic strategies have a role in the management of pain

In the past Dr. Smith has worked on the advisory boards and/or speakers panel for HealthPoint, KCI, Convatec, Diversified Clinical Services, Wake Pharma, and Neuro Biotech.

She has been an employee or consultant with Diversified Clinical Services (DCS), Day and Zimmermann, BioD LLC, and Derma Sciences.

Adrianne P. S. Smith MD Disclosure
Opioid Use for Chronic Non-Cancer Pain (CNCP) Has Been Steadily Increasing

10 Million Americans Use Opioids Annually [prior term narcotics](1) 2% use opioids 5 days per week  
~50% use opioids for ≥ 2 years  
~20% use opioids for ≥ 5 years

Long-acting Opioid Sales Double Every 3-4 yrs; (2)  
Correlates increased opioid use noted for  
- Chronic pain  
- Non-metastatic pain

Not necessarily related to an increase in abuse; related to an increased availability in the community

Prescription Drug Overdose Related Deaths Surpassed Accidents as Primary Cause of Death

Prescribed Opioid use is escalating with increased diagnosis and treatment of chronic pain syndromes. Opioids have an adverse effect on quality of life (QOL). Opioid Overuse/Abuse/Accidental Death are increasing. Opioid-Related Adverse Drug Events (ORADE) are the primary types of adverse drug events reported in most health systems and increase the total cost of care.

Opioid Pain Relief May Demonstrate Diminishing Effects Overtime and With Accelerations in Doses

Opioid May Adversely Impact Quality of Life (QOL) and Activities of Daily Living (ADL) Measures

Prescription Painkiller Sales and Deaths

http://www.cdc.gov/drugoverdose/data/

Brain Plasticity Leads to Heightened Perception of Pain: Hyperalgesia and Alloodynia with Routine, High Dosage and Abuse of Opioids

The Role of Plasticity in Chronic Pain Sensitization

Mechanisms:
- Chronic progression
- Neuraxial pain
- Enhanced opioid metabolism
- Phasic pain

Mechanisms:
- Sensitization of primary afferent neurons
- Activation of descending and central pain inhibitory systems

Mechanisms:
- Receptor down-regulation
- Supersensitivity of opioid receptors
- Opioid-induced hyperalgesia

Mechanisms:
- Increased pain threshold
- Endogenous opioid analgesia
- Pain relief

Medulla
Periaqueductal gray
Preganglionic sympathetic nervous system
Antalgic
Hyperalgesic
Allodynic
Chronic pain

81% 79% 67% 65% 65% 59% 54% 49% 41% 31% 20%

Exercise Sleep Meals Chores Socialize Sex Concentrate Relationship Work Childcare

Chronic Pain Patients Quality of Life % Unable to Perform ADL

Source: American Pain Society Chronic Pain Patient Survey http://www.ampainsoc.org/links/roadblocks/

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http://www.cdc.gov/drugoverdose/data/
Opioid Induced Hyperalgesia (OIH) and Opioid Tolerance May Influence Wound Pain and Healing

Common Characteristics of Opioid-induced Hyperalgesia (OIH)

- Worsening pain over time in spite of, and because of, opioid dose increases
- Nociceptive sensitization
- Area of pain more diffuse
- Pain and lesser quality and harder to pinpoint

http://www.uspharmacist.com/content/d/feature/34014/

Heroin Use is Increasing as Opioid Use Decreases and National Abuse Increases

Evidence indicates that individuals who are unable to obtain prescription drugs may begin to use heroin, which is more readily available and less expensive

- Of those who report heroin use, 80.5% report having engaged in non-medical use of prescription drugs
- Of youth and young adults who report non-medical use of prescription drugs, 14.9% also report using heroin

Evaluating Pain in the Patient with a Painful Wound

A doctor who cannot take a good history and a patient who cannot give one are in danger of giving and receiving bad treatment. — Anonymous


Definition and Categorization of Pain

Dictionary Definition:

“Physical suffering or discomfort caused by illness or injury”.

International Association for the Study of Pain

“Unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage”.

Categorization of Pain: Acute and Chronic

<table>
<thead>
<tr>
<th>Acute Pain (&lt;3 months)</th>
<th>Chronic Pain (≥ 3 months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Relatively brief duration ≤ 3 months</td>
<td>Longer in duration &gt; 3 months</td>
</tr>
<tr>
<td>Often develops in association with a short lived stimuli that initiates the pain response but is not persistent</td>
<td>Often accompanies a disease process or injury with a persistent or unremitting underlying effect; Inflammation, recurrent injury</td>
</tr>
<tr>
<td>Example: Postop pain, Perioperative pain, Post-traumatic pain, Select disease processes</td>
<td>Example: Rheumatoid Arthritis (RA), Osteoarthritis (OA), Low Back Pain (LBP), Malignancy associated pain Fibromyalgia, Chronic Regional Pain Syndrome (CRPS)</td>
</tr>
</tbody>
</table>

Grading Pain Severity Typically Measures Sensory Component of Pain Assessment

<table>
<thead>
<tr>
<th>Level</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>0</td>
</tr>
<tr>
<td>Mild</td>
<td>1-3</td>
</tr>
<tr>
<td>Mod</td>
<td>4-6</td>
</tr>
<tr>
<td>Severe</td>
<td>7-10</td>
</tr>
</tbody>
</table>

The Painful Experience Has Emotional, Cognitive and Psychosocial Components

Assessing Anxiety, Excitement and Worry Help Assess Catastrophizing Behavior in Chronic Pain

Pain Assessments Should Be Interpreted with an Understanding of Patient's Emotional State

Complete Pain Assessments Also Evaluate Energy, Sleep, Activity and Mood

Pain Should Be Correlated With Functional Outcomes Minimizing Subjective Assessment
Nociceptive versus Neuropathic Pain: Distinguishing the Anatomic Origin of Pain

Nociceptive pain: Nociceptors in tissues send pain signals to the CNS.

Neuropathic pain: Damage to the nerve itself causes typical pain symptoms.

Persistent Nociceptive Pain May Precede or Co-Exist With Neuropathic Pain

<table>
<thead>
<tr>
<th>Neuropathic Pain</th>
<th>Mixed Pain</th>
<th>Nociceptive Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Peripheral neuropathies (diabetes, HIV)</td>
<td>• Migraine and chronic daily headache</td>
<td>• Mechanical low back pain</td>
</tr>
<tr>
<td>• Post-herpetic neuralgia</td>
<td>• Fibromyalgia</td>
<td>• Rheumatoid arthritis</td>
</tr>
<tr>
<td>• Central post-stroke pain</td>
<td>• Phantom limb pain</td>
<td>• Osteoarthritis</td>
</tr>
<tr>
<td>• Spinal cord injury</td>
<td>• Complex regional pain syndrome</td>
<td>• Chronic inflammatory conditions</td>
</tr>
<tr>
<td>• Neuropathic low back pain</td>
<td>• Multiple sclerosis</td>
<td>• Somatoform pain disorder</td>
</tr>
<tr>
<td></td>
<td>• Low back pain</td>
<td>• Postoperative pain</td>
</tr>
<tr>
<td></td>
<td>• Myofascial pain syndrome</td>
<td>• Sickle cell crisis</td>
</tr>
<tr>
<td></td>
<td>• Skeletal muscle pain</td>
<td>• Sports/exercise injury</td>
</tr>
</tbody>
</table>

Different Types of Pain Descriptions Will Point to the Underlying Etiology and Origin of the Pain

- **Somatic**
  - Aching
  - Sharp
  - Knife-like
  - Throbbing
  - Spasm
  - Pounding
  - Stiffness
  - Sore
  - Bruising

- **Visceral**
  - Pulling
  - Hurts
  - Twisting
  - Like being Hit
  - Tense
  - Hard
  - Friction
  - Irritating
  - Grabbing

- **Neuropathic**
  - Burning
  - Tingling
  - Shooting
  - Stabbing
  - Jabbing
  - Shock-like
  - Piercing
  - Radiating
  - Gnawing
  - Pinching
  - Touchy
  - Sensitive

Components of Proper Pain Documentation

- **Onset**: At what point did the patient first notice the pain?
- **Location**: Where is the pain? (the patient can be asked to point to the location of the pain)
- **Duration**: What is the duration of the pain? How long does the episode of pain last? Does it return?
- **Characteristics**: How does the patient describe the pain? (i.e. Sharp, stinging, burning, etc.)
- **Aggravates/Alleviates**: Does anything aggravate the pain? Does anything relieve the pain?
- **Radiate**: Does the pain radiate to any other location or is it localized?
- **Treatments**: What treatments has the patient used in the past and present? Were they successful?

Universal Pain Assessment Tool: “Likert Scale” with Descriptions Improve Pain Assessments

- **Verbal descriptor scale**
  - No pain
  - Mild pain
  - Moderate pain
  - Severe pain
  - Worst pain possible

- **Wong-Baker Facial Pain Scale**
  - Pain intensity
  - Faces from happy to sad

PQRST-U Mnemonic

- **P**: Precipitating factors
  - What makes your pain better? Worse? Movement? Hygiene care?
- **Q**: Quality
  - Describe your pain for me?
- **R**: Radiation or pattern
  - Does the pain move from one place to another or does it stay in one place? Where?
- **S**: Severity or site
  - On a scale of 0-10 with 0=no pain and 10=worse pain, possible, where is your pain now? At its worst? At its best? After you take pain medication?
- **T**: Temporal nature
  - Is your pain constant or intermittent? How long have you had this pain?
- **U**: You!
  - What are your pain management goals including intensity, QOL and activity level? What does your pain mean to you?
Assessment Tools Can Describe Pain and Etiology
McGill Pain Questionnaire Short Form

<table>
<thead>
<tr>
<th>Item</th>
<th>Score</th>
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<tbody>
<tr>
<td>1.</td>
<td></td>
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<tr>
<td>2.</td>
<td></td>
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<tr>
<td>3.</td>
<td></td>
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<tr>
<td>4.</td>
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<td>5.</td>
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<tr>
<td>6.</td>
<td></td>
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<td>7.</td>
<td></td>
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<tr>
<td>8.</td>
<td></td>
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<tr>
<td>9.</td>
<td></td>
</tr>
</tbody>
</table>

1. Pain Rating Scale (PRS): Place a check mark (X) in the column that represents the degree to which you feel that type of pain. More than one check mark indicates the degree of the pain in your physician report.
2. Etiology of the painful wound: The information is based on the details of the pain as reported by the patient.

Etiology of the Painful Wound
The Clinical Perspective

A physician is obligated to consider more than a diseased organ, more than even the whole man—he must view the man in his world. — Harvey Cushing
Attributed by Rene Dubos, Man Adapting (1965, 1980), Chap. 12, 342. Dubos introduces the quote with “is reported to have taught” and no other citation.

Nociceptor Nerve Endings are Located in Cutaneous Tissues, Around Muscles, Periosteal Sheath and Organs

Nociceptive Activation Process Converts Noxious Stimuli From Biochemical Into Electrical Signals

<table>
<thead>
<tr>
<th>Conversion of noxious stimuli into electrical current</th>
<th>Action potential from periphery to spinal column dorsal horn</th>
<th>Nociceptors axons from spinal column to the brain</th>
<th>Cerebral Cortex after thalamic and amygdala influence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bradykinin Substance P</td>
<td>Na+ Channels K+ Channels Calcitonin Gene Related Peptide</td>
<td>Ca++ Channels CGRP Glutamate Nitric Oxide (NO)</td>
<td>Ca++ Channels CGRP Glutamate Nitric Oxide (NO)</td>
</tr>
<tr>
<td>Histamine Serotonin</td>
<td>Lidocaine, Nerve Blocks, Anesthetics</td>
<td>Opioids, GABA, Serotonin, Clonidine</td>
<td>Antidepressants, tricyclics, Selective Serotonin, Clonidine</td>
</tr>
<tr>
<td>NSAID, ASA Serotonin inhibitor</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Review: Pain Signaling Pathways

Conversion of Noxious Stimuli Into Electrical Current is the First Step of the Nociceptive Process

<table>
<thead>
<tr>
<th>Substance</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Substance P</td>
<td>Pain Transmission Peptide</td>
</tr>
<tr>
<td>CGRP</td>
<td>Calcitonin Gene-Related Peptide (vasodilate/immune)</td>
</tr>
<tr>
<td>Pressure</td>
<td>Pressure—extreme changes trigger nociception</td>
</tr>
<tr>
<td>Heat</td>
<td>Temperature—extreme changes trigger nociception</td>
</tr>
<tr>
<td>Lipids</td>
<td>Lipids—acts as a pain trigger</td>
</tr>
<tr>
<td>ACDB (Na+)</td>
<td>Acids—acts as a pain trigger</td>
</tr>
<tr>
<td>ATP</td>
<td>Adenosine Triphosphate (2nd Messenger)</td>
</tr>
<tr>
<td>ATR (K+)</td>
<td>Adenosine Triphosphate (2nd Messenger)</td>
</tr>
<tr>
<td>TPH (5HT)</td>
<td>5 Hydroxytryptamine (5-HT) monoamine neurotransmitter</td>
</tr>
<tr>
<td>Serotonin</td>
<td>A-hydroxytryptamine (5-HT) monoamine neurotransmitter</td>
</tr>
<tr>
<td>Serotonin</td>
<td>Inflammatory Mediator causing RV vasodilation; immune cells released</td>
</tr>
<tr>
<td>Bradykinin</td>
<td></td>
</tr>
<tr>
<td>Substance P</td>
<td>Inflammatory Mediator causing RV vasodilation; immune cells released</td>
</tr>
</tbody>
</table>

NGF | Nerve Growth Factor
---|---
Bradykinin | Inflammatory Mediator causing RV vasodilation; immune cells released
Serotonin | A-hydroxytryptamine (5-HT) monoamine neurotransmitter
ATP | Adenosine Triphosphate (2nd Messenger)
Acids (Na+) | Acids—acts as a pain trigger
Lipids | Lipids—acts as a pain trigger
Heat | Temperature—extreme changes trigger nociception
Pressure | Pressure—extreme changes trigger nociception
CGRP | Calcitonin Gene-Related Peptide (vasodilate/immune)
Substance P | Pain Transmission Peptide

Nociceptors from spinal column to the brain.
Nociceptive Fiber Speed Differs Based Upon Level of Myelination

<table>
<thead>
<tr>
<th>Fiber</th>
<th>Myelination</th>
<th>Firing Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>C-Fiber</td>
<td>None</td>
<td>1.0 m/s</td>
</tr>
<tr>
<td>A-Delta Fiber</td>
<td>Intermediate</td>
<td>2-10 m/s</td>
</tr>
<tr>
<td>A-Beta Fiber</td>
<td>Complete</td>
<td>30-70 m/s</td>
</tr>
</tbody>
</table>

Pain Sensation signaling connects to both the Sympathetic and Parasympathetic autonomic system. Fibers may travel up or down the spine multiple segments to innervate organs required for the appropriate response.

Nitric Oxide Synthesis Supports Nociceptive Neurotransmission as a 1st and 2nd Messenger

Nociception Pathway Ultimately Terminates in the Central Nervous System (CNS) Where Pain Perception Occurs

"Pain-brain neurologic process where stimuli is interpreted through a system of parallel processes, and determined to be noxious or undesirable."

Thermo-Receptors
- Heat- warm, hot, burn
- Cold- cool, cold, ice

Mechano-Receptors
- Touch- crude, light, firm
- Pressure- dull, sharp
- Vibration- slow, moderate, fast
- Tension- tissue, periosteum, joint

Chemo-Receptors
- pH changes
- Chemical (eg. Capsaicin)
Centralization - inflammation induced CNS hypersensitivity to noxious and non-noxious stimuli resulting from physiological, functional and anatomical change.

1. Peripheral Sensitivity - altered high-threshold nociceptors
2. Phenotypic Switch - altered chemical makeup affecting CNS neuron properties
3. Central Sensitivity - increased excitability and CNS neuronal responsiveness
4. Perception Pain Alteration

Pain Centralization Occurs as a Response to Inflammatory Sensitization and Modification

Acute and Chronic Components Influence the Type and Intensity of Wound Pain

<table>
<thead>
<tr>
<th>Categorization Scheme</th>
<th>Description and/or Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inflammation</td>
<td>Underlying concurrent diseases</td>
</tr>
<tr>
<td>Infection</td>
<td>Bacterial, viral, fungal, infestations</td>
</tr>
<tr>
<td>Hypoxia-Ischemia</td>
<td>Hypoxia leads to acidosis</td>
</tr>
<tr>
<td>Bleeding</td>
<td>Heme-Induced Inflammation</td>
</tr>
<tr>
<td>Recurrent Injury</td>
<td>Activation of pressure sensors</td>
</tr>
<tr>
<td>Allergic Reaction</td>
<td>Mast Cell related triggers</td>
</tr>
<tr>
<td>Edema</td>
<td>Inflammation, allergic reaction or consequence of underlying disease</td>
</tr>
<tr>
<td>Centralization</td>
<td>Neuronal damage that changes somatosensory system</td>
</tr>
</tbody>
</table>

Factors Influencing Postoperative Pain

Post-operative Pain (>7wks)

Key Concepts in Inflammation Progression

MMPs/TIMPs
Endogenous Opioids
Cytokines/Resolvins
Growth Factors

Expression Level
MMPs/TIMPs
Cytokines/Resolvins
Growth Factors
Endogenous Opioids
Anti-Inflammatory
Cytokines
Resolvins
Heme Oxygenase
Aquaporins
Collagens
Angiogenesis

Conscientious and careful physicians allocate causes of disease to natural laws, while the ablest scientists go back to medicine for their first principles.
— Aristotle (Attributed.)
Progression and Resolution of Inflammation After Painful Stimuli

Key Concepts in Pain: Nociceptive vs Neuropathic

Conscientious and careful physicians allocate causes of disease to natural laws, while the ablest scientists go back to medicine for their first principles. — Aristotle (Attributed.)

Nociception Activation Includes a Protective Initial Pro-Inflammatory Response

Nociceptor Pain Receptors Embedded the Skin, Joints, Periosteum and Organs

1. Injury initiates the release of mediators that activate toll-like receptors (TLRs) on keratinocytes and mast cells (MC) close to the nerve terminal.
2. Vasodilators are also released, promoting adhesion and transmigration of immune cells including T cells (T), neutrophils (N) and monocytes (MN), and recruitment of macrophages (Mφ).
3. Released Inflammatory Mediators—bind receptors on nociceptor terminals—causes peripheral nociceptor sensitization.
4. Targets Include Cytokine Receptors (CytR), G protein–coupled receptors (GPCR), ligand-gated channels (LGC) and tyrosine kinase receptor type 1 (TrkA).

Nociceptor Pain Receptors Embedded the Skin, Joints, Periosteum and Organs

Interactions between immune cells and nerve terminals
2. Macrophage Recruitment—peripheral nerves and Schwann cells release TNF-α and IL-1β, which recruits the macrophages.
3. Nociceptive Nerve Terminal—secretes Substance P (SP) and CGRP through antidromic activation of neighboring nerve terminal branches. Substance P and CGRP promote vasodilation and extravasation of immune cells. Neutral endopeptidase (NEP) restrains neuroinflammation by degrading substance P and CGRP.

Treating the Painful Wound with Pharmacologic and Non-Pharmacologic Interventions

A certain author defines a doctor to be a man who writes prescriptions till the patient either dies or is cured by nature. — Peter Shaw

The Reflector: Representing Human Affairs As They Are (1750). In The Pocket Lacon (1839), Vol. 1, 59.
Pro-Nociception and Anti-Nociception Processes and Interventions

<table>
<thead>
<tr>
<th>Pro-Nociception</th>
<th>Anti-Nociception</th>
</tr>
</thead>
<tbody>
<tr>
<td>Substance P (neu)</td>
<td>Descending anti-nociceptive pathway (monoamine, serotonin)</td>
</tr>
<tr>
<td>Excitatory AA (neu)</td>
<td>Opioids</td>
</tr>
<tr>
<td>Prostaglandins</td>
<td>GABA</td>
</tr>
<tr>
<td>Kines</td>
<td>Cytokines</td>
</tr>
<tr>
<td>Nerve growth factor</td>
<td>CRH</td>
</tr>
</tbody>
</table>

Spinal Cord

Equip Your Toolbox of Potential Therapeutic Adjunctive Interventions to Mitigate Pain

- Pain Mitigating Non-Opioid Dressings
- Physical Therapy Interventions
- Cognitive Interventions
- Non-Narcotic Medications

Pain Mitigating Non-Opioid Dressings Contain Components that Reduce Inflammatory Mediators

- Thermo-Receptors
  - Cooling
  - Buffers
  - Moisture
  - Oxygenated
- Mechano-Receptors
  - Silicone
  - Padding
  - Securing
  - Off-Loading
- Nociceptor
  - Buffers
  - Silvers
  - Collagens
  - ECM

Pain Mitigating Non-Opioid Dressings Contain Non-Opioid Medications of the following types:

- Single
- Silent
- Polymodal

Physical Therapy Interventions Applied Before, During and After the Onset of Pain to Best Relief

**Standard Physical Therapeutic Interventions**
- Heat and/or cold therapy
- Ultrasound
- Massage therapy
- Manual manipulation

**Complimentary Interventions**
- Electrical stimulation (e.g. TENS units)
- Electromagnetic Therapy
- Dance/Yoga
- Acupuncture

Cognitive Interventions Significantly Improve Pain Reduction and Prove to Be Vital Considerations

- COGNITIVE RESTRUCTURING
- RELAXATION

**SUPPORTIVE AND GROUP THERAPY**
- • Guided Imagery
- • Progressive Muscular Relaxation
- • Meditation
- • Music Therapy
- • Biofeedback

**COPING SKILLS TRAINING**
- • Family Support
- • Physical Therapy
- • Activity Pacing
- • Pain Diaries

**STRESS-MANAGEMENT INTERVENTIONS**
- • Sharing Feelings and Problems
- • Humor and Recreation
- • Regular Exercise

Cognitive Interventions Significantly Improve Pain Reduction and Prove To Be Vital Considerations

BARRIERS TO INTEGRATION OF COGNITIVE BEHAVIORAL THERAPIES

- Overemphasis on the biomedical model, both in clinical care and in medical education
- Lack of standardization of cognitive-behavioral techniques
- Lack of patient compliance in practicing these methods
- Physician reluctance to prescribe for psychological methods due to:
  - Lack of awareness of the benefits of psychological techniques
  - Concern that the patient will see these methods as treatment for mental illness
- Inconsistent and poor reimbursement by third party payers
- Ill-defined credentialing criteria for providers of these treatments, and subsequently unreliable execution of these methods
- Time intensive nature of psychosocial interventions


Key Caveats for Managing the Painful Wound

Assessment, Evaluation and Treatment Plans must include an understanding of your patients neurosensory, emotional and psychosocial status.

Inflammation Is The Underlying Etiology Of Pain, regardless of the clinical etiology, and should be treated.

Centralized Pain and Chronic Pain States may influence treatment related pain and should be addressed.

Functional and Psychosocial Improvements can be helpful in assessing pain reduction in some chronically painful wounds.

Opioids Are Not The First-line Treatment for most chronic non-cancer pain.