MEDICAL TREATMENT UTILIZATION SCHEDULE (MTUS)
CHRONIC PAIN MEDICAL TREATMENT GUIDELINES

Part 1: Introduction

Part 2: ~~Official Disability~~ Chronic Pain Medical Treatment Guidelines ~~(ODG) Treatment in Workers' Compensation — Pain (Chronic)~~

Discussion

The proper title for Part 2 is "Chronic Pain Medical Treatment Guidelines" or "Chronic Pain Guidelines" as the Division is not adopting the ODG Guidelines, but rather a modified version of those Guidelines. Naming the guidelines "Official Disability Guidelines (ODG)" causes unnecessary confusion over whether the modified version adopted in 9792.24.2 and specified in 9792.21.1(a)(1) is being referenced/cited, or the most current version of the Official Disability Guidelines as defined in 9792.20(i) and specified in 9792.21.1(a)(2)(A).

July 2015

Table of Contents

~~Explanation of ODG Medical Literature Ratings~~

~~Ranking by Type of Evidence:~~
~~STUDIES:~~
~~1. Systematic Review/Meta-Analysis~~
~~2. Controlled Trial – Randomized (RCT) or Controlled~~
~~3. Cohort Study - Prospective or Retrospective~~
~~4. Case Control Series~~
~~5. Unstructured Review~~
~~OTHER:~~
~~6. Nationally Recognized Treatment Guidelines (from guideline.gov)~~
~~7. State Treatment Guidelines~~
~~8. Other Treatment Guidelines~~
~~9. Textbook~~
~~10. Conference Proceedings/Presentation Slides~~

~~Ranking by Quality within Type of Evidence:~~
~~a. High Quality~~
~~b. Medium Quality~~
~~c. Low Quality~~

Recommendation:
Re-rate the quality and strength of evidence supporting recommendations in accordance with the MTUS methodology for evaluating medical evidence in section 9792.25.1 and revise this explanatory list accordingly.

Discussion:
As proposed, in section 9792.24.2(a) the Chronic Pain Medical Treatment Guidelines are adopted and incorporated by reference into the MTUS. These ratings and rankings by quality within type of evidence are in conflict with, and do not conform to, the MTUS methodology for evaluating medical evidence in section 9792.25.1. All sections of the Medical Treatment Utilization Schedule regulations must be consistent with one another.

This ~~C~~chapter of the Medical Treatment Utilization Schedule, the Chronic Pain Medical Treatment Guidelines, was ~~is adopted~~ adapted with permission from *Official Disability Guidelines* (ODG)

Treatment in Workers' Comp~~,~~ that were published and copyrighted by the Work Loss Data Institute.

The proper title for Part 2 is "Chronic Pain Medical Treatment Guidelines" as the Division is not adopting the ODG Guidelines, but rather a modified version of those Guidelines. Naming the guidelines "Official Disability Guidelines (ODG)" causes unnecessary confusion over whether the modified version adopted in 9792.24.2 and specified in 9792.21.1(a)(1) is being referenced/cited, or the most current version of the Official Disability Guidelines as defined in 9792.20(i) and specified in 9792.21.1(a)(2)(A).

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Although this document was based on the Official Disability Guidelines (ODG), it is in fact not the Official Disability Guidelines (ODG) as it contains significant differences; therefore it is inappropriate for this section of the California regulations to be copyrighted by the Work Loss Data Institute (WLDI).

~~Any links to treatment guidelines external to this document refer to the Medical Treatment Utilization Schedule.~~

There appear to be multiple links to treatment guidelines external to this document that do not refer to the Medical Treatment Utilization Schedule.

PART 1: Introduction

These Chronic Pain Medical Treatment Guidelines apply when a patient has chronic pain that persists beyond the anticipated time of healing as determined by following the relevant sections of the Medical Treatment Utilization Schedule (MTUS). In following the clinical topics section, the physician begins by assessing the presenting complaint and determining whether there is a "red flag for a potentially serious condition" that would trigger an immediate intervention. Upon ruling out a potentially serious condition, the physician should provide conservative management. If the complaint persists, the physician needs to reconsider the diagnosis and decide whether a specialist evaluation is necessary. The Chronic Pain Medical Treatment Guidelines provide a framework to manage ~~all~~ treatment for chronic pain ~~conditions, even when the injury is not addressed in the clinical topics section of the MTUS~~.

Chronic Pain Medical Treatment Guidelines are intended to address treatment for chronic pain.

The Chronic Pain Medical Treatment Guidelines consist of an introduction (Part 1) and specific information on interventions and treatments for chronic pain (Part 2), a reformatted version of evidence-based treatment guidelines from the April 6, 2015 version of the Work Loss Data Institute's Official Disability Guidelines (ODG) Treatment in Workers' Compensation – Pain (Chronic), adapted with permission from the publisher. For specific guidance on opioid use, refer to the "MTUS Opioids Treatment Guidelines."

Definitions:

Pain: The International Association ~~for the Study~~ of Pain (IASP) defines pain as "an unpleasant sensory or emotional experience associated with actual or potential tissue damage, or described in terms of such damage." (Merskey, 1994) This definition describes pain as a subjective experience; therefore, unlike hypertension or diabetes, there is no objective measurement for pain intensity. Analysis of the objective data (history, psychosocial assessment, physical findings, imaging results, lab tests, etc.) is needed to evaluate the patient's subjective report of pain.

Types of Pain (Acute vs Chronic): Pain comes in many forms. Understanding which kind or kinds of pain a person is experiencing is a first step toward treatment. ~~Although acute and chronic pain is considered separately below, an individual can experience them simultaneously. Furthermore, current research shows that pain exists more on a continuum than in discrete categories of "acute" or "chronic" pain. This means that,~~ fFor some patients, the mechanisms responsible for pain persistence are engaged early in the injury process. Therefore, it is important to identify persons at risk for the development of chronic pain and to establish preventative measures to reduce the likelihood of pain persistence.

Under the definitions in the MTUS regulations, acute pain and chronic pain do not overlap. MTUS regulations, including the chapters of the Medical Treatment Utilization Schedule must be consistent with one another.

- *Acute pain* is pain lasting up to one month.~~, by definition, is~~ Typically the onset of acute pain is sudden ~~onset~~ and is expected to be of short duration. It can usually be linked to a specific event, injury, or illness—a muscle strain, a bone fracture, severe sunburn, or a kidney stone, for example. ~~People can self-manage many types of acute pain with over-the-counter medications or a short course of stronger analgesics and rest.~~ Acute pain usually subsides when the underlying cause resolves, such as when a fracture heals, or kidney stone or diseased tooth is removed. ~~In the "MTUS Opioids~~

~~Treatment Guidelines," acute pain is defined as pain lasting up to one month and~~

- *~~S~~subacute pain* ~~as~~ is pain lasting between one and three months.
- *Chronic pain* is ~~any pain that lasts more than three months following an injury and can be frustratingly difficult to treat. In the "MTUS Opioids Treatment Guidelines," chronic pain is defined as~~ pain lasting three or more ~~than three~~ months from the onset of pain. It can be frustratingly difficult to treat.

These modifications are necessary to conform to the definition of chronic pain in (b) of section 9792.20 in the MTUS regulations. The reference to management of acute pain is unnecessary.

Types of Pain (Mechanisms): Pain mechanisms can be broadly categorized as nociceptive, inflammatory, neuropathic, or unknown.

- *Nociceptive pain*: pain caused by activation of nociceptors, which are sensory neurons found throughout the body. A nociceptor is "a receptor preferentially sensitive to– a noxious stimulus or to a stimulus which would become noxious if prolonged." (Smith, 2009) Nociceptive pain is the type experienced with tissue damage such as contusion, burn, or injury to a body part.

- *Inflammatory pain:* pain which occurs in response to tissue injury, when inflammation develops and local nociceptors become highly sensitive even to normal stimuli, such as touch. This is another type of "warning" pain, indicating the need for a period of healing, and this pain generally disappears after the injury resolves. In conditions such as rheumatoid arthritis or gout, inflammatory pain persists as long as the inflammation does. (IOM, 2011)

- *Neuropathic Pain*: "pain initiated or caused by a primary lesion or dysfunction of the nervous system." **Neuropathic pain is caused by a malfunction of the peripheral or central nervous system due to an injury or an illness**. (Normal nociception would not be considered dysfunction of the nervous system). The cause may be an underlying disease process (as in diabetes) or injury (e.g., stroke, spinal cord damage), but neuropathic pain may not have an observable cause and can be considered maladaptive "in the sense that the pain neither protects nor supports healing and repair." (Costigan, 2009)

- *Unknown causes:* pain that arises without a defined cause or injury. Examples of such chronic pain conditions are fibromyalgia, irritable bowel syndrome, vulvodynia, chronic headaches, and temporomandibular disorders. Research points to impaired central pain sensitivity and responses in these conditions, but their complex mechanisms have not yet been unraveled. (Kindler, 2011)

Overview

~~Acute and c~~Chronic pain affects large numbers of Americans, with at least 100 million adults in the United States burdened by chronic pain ~~alone~~. The annual national economic cost associated with chronic pain is estimated to be $560–635 billion. Pain is a uniquely individual and subjective experience that depends on a variety of biological, psychological, and social factors, and different population groups experience pain differentially. (IOM, 2011)

The reference to acute pain is not necessary.

Chronic pain has a significant impact on the individual and on society as a whole, and it is the primary reason for delayed recovery and costs (medical and indemnity) in the workers' compensation system. Most chronic pain problems start with an acute nociceptive pain episode. As a result, effective early care is paramount in preventing chronic pain. Not surprisingly, pain has become the subject of intensive scientific research, and researchers are generating a growing evidence base regarding the diagnosis, treatment, and management of painful conditions.

The experience of pain is a complex phenomenon. Multiple models have evolved over time to explain it. Traditionally, the biomedical model explains pain through etiologic factors (e.g., injury) or disease whose pathophysiology results in pain. It is now understood that this classic biomedical approach (pursuit of a pathoanatomical diagnosis with the view of targeting and treating a specific "pain generator") is incomplete. Its exclusive application can result in unrealistic expectations on the part of the physician and patient, inadequate pain relief, and excessive disability in those with pain that persists well after the original injury has healed. A strictly biomedical approach to pain is simply too reductionist; rather, what is called for is an approach that recognizes the complexity of the pain experience. Similar to what has been learned about other chronic diseases, chronic pain ultimately affects (and is affected by) many intrinsic and extrinsic aspects of a person's life.

In general, the early theories of how pain works failed to address some key issues. (IOM, 2011)

- The relationship between injury and pain varies (that is, a minor injury may produce great pain, and a significant injury may produce minor pain), as does the relationship between the extent of injury and the resulting disability.
- Non-noxious stimuli can sometimes produce pain (allodynia), and minor amounts of noxious stimuli can produce large amounts of pain (hyperalgesia).
- The locations of pain and tissue damage are sometimes different (referred pain).
- Pain can persist long after tissue heals.
- The nature of pain and sometimes its location can change over time.
- Pain is a multidimensional experience, with strong psychosocial influences and impacts.
- Responses to a given therapy vary among individuals.
- Earlier theories have not led to adequate pain treatment.

The biopsychosocial model of pain recognizes that pain is ultimately the result of the patient's pathophysiology and psychological state, cultural background/belief system, and relationship/interactions with the environment (workplace, home, disability system, and health care providers). Therefore, pain has become understood as a complex condition involving numerous areas of the brain. Multiple two-way communication pathways in the central nervous system (from the site of pain to the brain and back again) and emotional, cognitive, and environmental elements work together to form a complete, interconnected pain apparatus. Because it has numerous interacting and contributing causes and multiple effects, chronic pain resembles many other chronic diseases. (Gatchel, 2007; IOM, 2011)

Pain Mechanisms

Within the biomedical model, pain mechanisms are broadly categorized as nociceptive or neuropathic. Inflammatory mechanisms may also play a role. While there are similarities, each mechanism has unique features and characteristics. This mechanistic approach may provide insight into appropriate therapeutic strategies.

Several reviews have detailed the mechanisms and mediators of pain and the components of the ascending and descending pain pathways. In nociceptive pain, signal transduction in nociceptor somatosensory afferent terminals converts mechanical, electrical, thermal, or chemical energy into an action potential which is transmitted to the dorsal horn of the spinal cord by specialized nerve fibers. The signal is then transmitted through ascending spinal-cortical pathways to the brain. These signals evoke a response in multiple brain systems, a "distributed network," consistent with the variety of physical, cognitive, affective, and reflexive reactions to pain that people experience.

Since multiple areas of the brain interact with other areas of the brain, past memories, external environmental factors, and internal cognitive factors (i.e., psychosocial factors) influence or modulate the pain experience. How the brain integrates all the input is, in part, the basis for the biopsychosocial model for, and approach to, the management of pain.

In contrast to nociceptive pain, neuropathic pain is "pain initiated or caused by a primary lesion or dysfunction of the nervous system." (Turk, 2001) The altered modulation of the pain response in patients with neuropathic pain causes a state of hyperexcitability and continuous pain signal output in the absence of peripheral tissue damage. "'Neuropathic pain can result from injury or trauma (e.g., surgery), infection (e.g., post-herpetic neuralgia), endocrine (e.g., diabetes, hypothyroidism), demyelination (e.g., multiple sclerosis), errors in metabolism, neurodegenerative disorders (e.g., Parkinson's disease), or damage directly to the spinal cord or brain (e.g., thalamic stroke)." (Backonja, 2001; Martucci, 2014)

Neuropathic pain is characterized by symptoms such as lancinating, electric shock-like, paroxysmal, tingling, numbing, and burning sensations that are distinct from nociceptive pain.

Many neuropathic pain states have traditionally been thought of as having a primary peripheral etiology. Recent investigation, however, using functional neuroimaging— techniques, demonstrates that many neuropathic and other chronic pain conditions may have a large centralized component (central vs. peripheral model). These conditions include, but are not limited to, chronic low back pain (CLBP), fibromyalgia, irritable bowel syndrome, temporomandibular disorders, and Complex Regional Pain Syndrome (CRPS)/Reflex Sympathetic Dystrophy (RSD). (Barad, 2014; Mackey, 2004; Ung, 2012; Younger, 2010)

Inflammation can play a significant role in both nociceptive and neuropathic pain. Inflammation occurs when cells and tissue are damaged and release chemical mediators (commonly referred to as "the inflammatory soup") that not only induce an inflammatory response but also sensitize nociceptors and other somatosensory components of the nervous system. Peripheral sensitization occurs when inflammatory mediators cause a reduction in the threshold required for nociceptor activation. A similar short-term central sensitization can occur in which neuronal excitability and responsiveness in the dorsal horn increase. In central sensitization, chemical mediators for inflammation can also upregulate the expression of genes that alter synaptic transmission.

Current research indicates that because of neuronal plasticity, protracted central sensitization (neuronal hyperexcitability) can result in long-term changes that may be important in the transition from acute to chronic pain and the development of chronic pain syndromes. Patients with these syndromes generally have severe and persistent pain that is disproportionate to the tissue injury.

Models

Models are the conceptual framework for understanding pain. They serve to establish parameters for reasonable outcomes and acceptable standards of care, which are helpful for physicians, patients, families, healthcare providers, carriers, and compensation systems. Several different models of pain have developed over time, each with strengths and weaknesses.

Acute vs. Chronic Pain Model

In many situations, acute pain serves as a highly adaptive and beneficial experience. Fundamentally, it serves as a protective warning of actual or impending tissue damage. Acute musculoskeletal pain is a common example in the injured worker and is often a signal of real or impending tissue damage.

Most acute pain is self-limited and may respond to short-term administration of analgesics and conservative therapies. However, continued activation of nociceptors with less than adequate pain control can lead to peripheral and central sensitization, a risk factor for persistent pain with prolonged disability, delayed return to baseline function, and delayed return to work.

Chronic pain differs from acute pain in more than just the time course. Whereas acute pain serves as a protective warning signal, chronic pain has no known survival benefit. Evidence suggests that generation and subsequent maintenance of chronic pain, as opposed to acute pain, may involve changes in central pain processing mediated through mechanisms of neural plasticity and may ultimately lead to hyper-excitability of central structures in the spinal cord and brain. To complicate matters, unremitting pain may be associated with depression –and/or anxiety.

~~As a practical matter, it is noted that "the distinction between acute and chronic pain is somewhat arbitrary" and "chronicity may be reached from one to six months post injury." ACOEM recognizes that the most clinically useful definition might be that "chronic pain persists beyond the usual course of healing of an acute disease or beyond a reasonable time for an injury to heal." (ACOEM, 2014) The definition of chronic pain, "any pain that persists beyond the anticipated time of healing," is derived from Bonica's Management of Pain. (Turk, 2001) Therefore, it is a clinical decision to recognize chronicity or persistence of pain: (1) when the condition is not improving over time; (2) when there is a lack of improvement with treatments directed to the specific injured body part (see Clinical Topics section of the MTUS); or (3) in the absence of a specifically correctable anatomic lesion (refer to the relevant Clinical Topics section of the MTUS). It often takes a number of months for the clinician to recognize when pain has become chronic.~~

According to section 9792.20, as used in this article, chronic pain means pain lasting three or more months from the initial onset of pain. This paragraph is best deleted as it is not necessary and may cause confusion and dispute over whether or not the pain is chronic pain, and consequently which guidelines are applicable.

Illness Behavior Model

As previously stated pain is a subjective experience, influenced and modulated by cognitive, emotional, and environmental elements. Psychosocial factors can affect the perception and expression of pain. These might include, but are not limited to, a tendency toward anxiety,

depression, somatization, fear avoidance, emotional liability, catastrophizing, job dissatisfaction, perceived injustice, and embellishment. Further, while frank malingering is rare, secondary gain factors, such as disability income and avoidance of perceived unpleasant tasks can impact the overall clinical presentation. Taken together, psychosocial factors often play a larger role in eventual patient outcome than obvious somatic factors as determined by the nature and extent of the original injury. Efforts directed solely to the management of possible physical pain generators without addressing psychosocial factors may result in a suboptimal outcome.

Biomedical vs. Biopsychosocial Model

The traditional biomedical model "assumes disease to be fully accounted for by deviations from the norm of measurable biological (somatic) variables." (Engel, 1977) According to this model, there is always a direct causal relationship between a specific pathophysiologic process and the presence and extent of a particular symptom. While this model has served the medical community well in the treatment and cure of certain diseases (e.g., infectious diseases), it has generally failed in the treatment of chronic illness, including persistent pain. For example, for decades the prevailing approach has been to identify the "pain generator" and remove it by cutting it out or blocking it.

In 1977 Engel proposed an alternative, the biopsychosocial model, which focuses greater attention on the patient, rather than presumed pathophysiology. The biopsychosocial model approaches pain and disability as a complex interplay of biological, psychological, and social factors. These psychosocial factors can be easily assessed.

The following chart contrasts these two pain models (Hanson, 1993)

Pain Models

Biomedical model	Biopsychosocial model
Most appropriate for treating acute pain conditions	More useful for those with chronic pain conditions
Emphasizes peripheral nociception	Recognizes the role that central mechanisms play in modulating peripheral nociception or generating the experience of pain in the absence of nociception
Focuses on physical disease mechanisms	Recognizes the importance of illness behavior including cognitive and emotional responses to pain
Takes a reductionistic approach to understanding and treating pain	Takes a multidimensional systems approach to understanding and treating pain
Relies on medical management approaches	Uses self-management approaches in addition to medical management

Researchers have found evidence that psychosocial variables are strongly linked to the transition from acute to chronic pain disability and that psychosocial variables generally have more impact than biomedical or biomechanical factors on back pain disability. (Linton, 2000) Thus, when clinical progress is insufficient or protracted, the clinician should consider the possibility of delayed recovery and be prepared to address any confounding psychosocial variables.

Medical vs. Self-Management Model

Understandably, patients want their chronic pain "cured" or eliminated. Unfortunately, no definitive cures currently exist for the majority of persistent pain problems, such as axial spine pain, peripheral neuropathies, and fibromyalgia. As is the case with all chronic medical conditions, chronic pain must be managed, when it cannot be cured. In the medical model, responsibility resides primarily with the physician. However, emphasis is increasingly being placed on encouraging patients to accept some pain and to make self-management efforts that can improve function and quality of life, even if they don't eliminate all pain. An approach that emphasizes participation in daily activities despite pain as well as fostering a willingness to have pain present without responding to it may aid in reducing the "distressing and disabling influences of pain." (Institute of Medicine, 2011) The self-management approach places primary responsibility on the person with chronic pain. Self-management strategies can significantly improve a patient's function and quality of life, while reducing subjective experiences of pain. It is important to educate patients to avoid persistent and unrealistic expectations for an elusive cure when none exists. This unrealistic curative view, often unwittingly fostered by healthcare providers, predictably leads to repeated failures, delayed recovery, and unnecessary disability and costs.

Self-efficacy is a psychological construct related to that of control. Believing that one can perform a task or respond effectively to a situation predicts pain tolerance and improvements in physical and psychological functioning. Research suggests that "a primary aim of chronic low back pain rehabilitation should be to bring about changes in catastrophic thinking and self-efficacy," because greater self-efficacy improves pain, functional status, and psychological adjustment. (Keefe, 2004) Researchers posit several explanations for why self-efficacy works to control pain, including the theory that people who expect success are less likely to be stymied when confronting the challenge of pain.

The goals of self-management and self-efficacy reinforce the benefits that accrue when people take a more active role in managing their pain. While self-efficacy as a sole method may not be sufficient to achieve pain control in many situations, treatment should include efforts to help patients actively manage pain.

Risk Stratification

Importance of early identification

Patients not responding to initial or subacute management (see Clinical Topics Section MTUS) or those thought to be at risk for delayed recovery should be identified as early as possible. Simple screening questionnaires may be used early in the clinical course to identify those at risk for delayed recovery. Those at risk should be more aggressively managed to avoid ineffective treatment and needless disability. Factors that help identify at-risk patients include: (1) those unresponsive to conservative therapies demonstrated to be effective for specific diagnoses in others; (2) the presence of significant psychosocial factors negatively impacting recovery; (3) loss of employment or prolonged absence from work (which has a high predictive value); (4) previous history of delayed recovery or incomplete rehabilitation; (5) lack of employer support to accommodate patient needs; and (6) a history of childhood abuse (verbal, physical, sexual, etc.) abandonment, or neglect (adverse childhood experience, or ACE).

Subacute Delayed Recovery

Complaints of pain are the most common obstacles to return to work. Undertreatment of pain and/or unrealistic expectations may play a role in delayed recovery. However, the subacute phase is a critical time for the injured worker, as additional time away from work may result in adverse medical (e.g., overtreatment), familial, economic, and psychological consequences (e.g., depression and anxiety), which can exacerbate pain complaints. When the physician recognizes that the problem is persisting beyond the anticipated time of tissue healing, the working diagnosis and treatment plan should be reconsidered, and psychosocial risk factors should be identified and addressed. If necessary, patients should be directed to resources capable of addressing psychosocial barriers to recovery.

Increasingly, time-limited Cognitive Behavioral Therapy (CBT) is being used successfully to do just that. Literature review meta-analysis has shown the CBT model of intervention to be more effective than wait list controls and alternative active treatment. (Morely,1999) Both the cognitive and behavioral intervention components of CBT have been found effective.

The behavioral component of CBT focuses on physiologic self-management techniques such as reinforcement for participation in functional activities, progressive relaxation, and autogenic/self-hypnosis. These techniques decrease the stress arousal response system associated with chronic pain. CBT techniques may be especially effective for patients with high stress arousal response, guarding behavior and history of ACE.

Patients with Intractable Pain

Studies have shown that the longer a patient remains out of work the less likely he or she is to return. Similarly, the longer a patient suffers from chronic pain the less likely treatment, including functional restoration efforts, will be effective. Nevertheless, if a patient is highly motivated and prepared to make the effort, a multidisciplinary evaluation for admission for treatment in a functional restoration program, (consistent with California Health and Safety Code section 124960) should be considered.

Assessment Approaches

History and Physical Examination

The treating physician has limited sources of objective information. Therefore, it is important for the physician to take a thorough history in clinical assessment and treatment planning for the patient with chronic pain. Whenever possible, this history should include a review of medical records. Clinical recovery may be dependent upon identifying and addressing previously unknown or undocumented medical and/or psychosocial conditions. A thorough physical examination is also important to establish or confirm diagnoses and to observe and better understand pain behaviors. The history and physical examination also serves to establish reassurance and patient confidence. Diagnostic studies should be ordered in this context and not simply for screening purposes.

If a –diagnostic workup is indicated and it does not reveal any clinically significant contraindications, the physician should encourage the patient to engage in an active rehabilitation and self-management program. Effective treatment of the chronic pain patient

requires familiarity with patient-specific past diagnoses, treatment outcomes, persistent complaints and psychosocial variables.

Evaluation of Psychosocial Factors

Psychosocial factors have proven better predictors of chronicity than clinical findings. Such variables/factors can and should be assessed; they include a history of abuse, anxiety, depression, fear-based avoidance of activity, catastrophizing, self-medication with alcohol or other drugs, patient/family expectations, medical-legal claims management issues, and employer/supervisor/worksite factors.

Childhood trauma may contribute significantly to pain chronicity. A 2010 CDC Study of 26,000 Americans adults revealed that 60% reported childhood familial problems, 15% experienced physical abuse, more than 12% had been sexually abused, and nearly 9% had at least five ACE episodes, (CDC, 2010). Such events (per the ongoing ACE study) correlate with delayed recovery and poor outcomes from injury. Clearly, assessment of psychosocial factors is a critical element of patient evaluation.

Functional Restoration Approach to Chronic Pain Management

Many injured workers require little treatment, and their pain will be self-limited. Others will have persistent pain that can be managed with straightforward interventions and do not require multi-disciplinary treatment. However, for patients with more refractory problems and sufficient motivation, a multidisciplinary, functionally oriented (not pain-oriented) treatment approach with a goal of independent self-management may be a more effective treatment approach. (Flor, 1992; Guzman, 2001)

Functional restoration is an established treatment approach that aims to minimize the residual complaints and disability resulting from acute and chronic medical conditions. Functional restoration may be considered if there is a delay in return to work or a prolonged period of inactivity. Functional restoration is the process by which the individual acquires the skills, knowledge, and behavioral changes necessary to avoid preventable complications and assume or re-assume primary responsibility ("locus of control") for his or her physical and emotional well-being post injury. The focus is on increasing activities of daily living (ADL), including returning to work. The individual thereby maximizes functional independence and the pursuit of vocational and avocational goals, as measured by functional improvement (see 8 CCR § 9792.20 (~~f~~e)).

This definition was renumbered from (f) to (e) in the recent regulatory revisions.

Independent self-management is the long-term goal of all forms of functional restoration. The process and principles of functional restoration can be applied ~~apply~~ to ~~a wide range of conditions, including acute injuries (e.g., sports, occupational), catastrophic injuries (e.g., brain and spinal cord injury), and chronic conditions (e.g.,~~ chronic pain ~~and multiple sclerosis)~~.

This section is specific to chronic pain.

It should be emphasized that functional restoration is not necessarily a full-time, multi-week treatment program, but rather an approach that emphasizes patient empowerment and personal responsibility.

A coordinated, goal-oriented, functional restoration approach can incorporate pharmacologic treatment, therapeutic interventions, CBT, and physical rehabilitation.

Using medications in the treatment of pain requires a thorough understanding of the mechanism underlying the pain as well as the identification of comorbidities that might predict an adverse outcome (refer to the "MTUS Opioids Treatment Guidelines"). Choice of pharmacotherapy must be based on the type of pain to be treated, though more than one pain mechanism may be involved. The physician should tailor medications and dosages to the individual, taking into consideration patient-specific variables such as comorbidities, other medications, and allergies. The physician should be knowledgeable regarding prescribing information and adjust the dosing to the individual patient. If the physician prescribes a medication for an indication not in the approved FDA labeling, he or she has the responsibility to be well informed about the medication and ~~confident~~ if it is not specifically recommended in the MTUS Chronic Pain Guidelines, to provide medical evidence that its use is scientific and evidence based. When effective, medications should provide a degree of analgesia that allows the patients to engage in rehabilitation, improvement of basic activities of daily living, and~~/or possibly~~ return to work. No drugs have been proven to reverse, cure, or "heal" chronic pain. In addition, periodic review of the ongoing chronic pain treatment plan for the injured worker is essential.

If off-label use of a drug is not specifically "recommended" in the MTUS Chronic Pain Guidelines, providing medical evidence that its off-label use is nonetheless reasonably required to cure or relieve the injured worker from the effects of his or her injury, is reasonable necessary to protect the injured worker from unnecessary and deleterious care.

Return to work is a primary goal.

When choosing an invasive procedure to treat a specific chronic pain problem, the provider must make a complex judgment in order to ensure that the desired and expected outcome is worth the risk involved.

Please refer to Part 2 of the Chronic Pain Treatment Guidelines to find specific guidelines on chronic pain treatments that include pharmacotherapy, invasive pain procedures, psychological and behavioral therapies, physical and occupational therapies, and other approaches. The treatment must be tailored to the individual case. Regardless of who is providing the treatment, be it an individual provider, a multidisciplinary group of providers, integrated interdisciplinary pain program, or a functional restoration program, it is important to design a treatment plan that explains the purpose of each component of the treatment. Furthermore, demonstration of functional improvement is necessary at various milestones in the functional restoration program to justify continued treatment.

Pain Outcomes and Endpoints

Because pain is a subjective experience, it cannot be readily validated or objectively measured. (AMA, 2001) ~~(~~Therefore, unlike many other chronic diseases, which may have objective measurements that can be used to assess the extent of the problem and treatment outcomes, chronic pain has no objective measurement. Measuring a patient's pain requires correlating objective data with the patient's subjective reporting to arrive at a comprehensive outcome representing the state of pain.

Complicating the measurement of pain is that there is often a wide variability in how much pain a given stimulus or injury will cause. This variability is influenced by genetics, mood, beliefs, sex, ethnicity, and other factors such as early-life pain experiences with pain. (Kim, 2004)

Chronic pain is often associated with an overall reduction in the patient's quality of life ~~which~~ and may l~~ead to~~ be associated with depression, anxiety, impaired social and physical function, and sleep disturbance. Moreover, there appears to be relative independence between pain and these co-existing stressors. Therefore, to capture the pain experience, it is necessary to also define and characterize these related domains. (Malhotra, 2012) In addition, it is essential to understand the extent to which pain impedes function. (AMA, 2001)

It is more accurate to describe these factors as associated.

The physician treating patients in the workers' compensation system must be aware that just because an injured worker has reached a permanent and stationary status or maximal medical improvement does not mean that the patient is no longer entitled to future medical care. The physician should periodically review the patient's course of treatment and any new information about the etiology of the pain or the patient's state of health. Continuation or modification of pain management depends on the physician's evaluation of the patient's progress toward treatment objectives. If it is unsatisfactory, the physician should assess how appropriate it is to continue the current treatment plan and whether to consider other therapeutic modalities. If the patient taking controlled substances to treat chronic pain experiences decreased pain and can demonstrate increas~~ed~~ing levels of function ~~or~~ and improved quality of life, then the treatment ~~has had~~ is having a satisfactory outcome.

The language should not inadvertently suggest permanent dependency on controlled substances.

Additionally, fluctuations are likely to occur in the natural history of patients with chronic pain. If exacerbations and "breakthrough" pain occur during the chronic clinical course, adjustments to the treatment will be necessary.

Conclusion

Chronic pain affects approximately 100 million adults in the U.S., with a national economic cost exceeding half a trillion dollars per year. Pain is a uniquely individual and subjective experience. Further, while pain can be a symptom of another condition, when it becomes persistent, it can become a disease in its own right, one that is associated with structural and functional changes of the peripheral and central nervous system. These changes can lead to the generation and maintenance of chronic pain conditions, with associated disability. While biologic mechanisms play a role in the perception of pain, it is important to recognize that psychological and environmental factors play an important role as well. Recognition of these factors will allow the physician to better (1) treat the recently injured patient, (2) identify the "at risk" patient, and (3) refer the patient with intractable chronic pain to the appropriate resources. A full assessment of the patient is required to determine the best approach in each case.

Therapy for chronic pain ranges from single modality approaches for the straightforward case to comprehensive interdisciplinary functional restoration care for the more challenging case. Therapeutic components such as pharmacologic, interventional, psychological, and physical approaches have been found to be most effective when performed in an integrated manner. All therapies should aim to restore function rather than merely eliminate pain, and demonstrated functional improvement is essential in assessing treatment efficacy. Typically, with increased function comes a perceived reduction in pain and increased perception of its control. These changes ultimately lead to an improvement in the patient's quality of life.

References for Introduction

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PART 2: ~~Official Disability~~ Chronic Pain Medical Treatment Guidelines ~~(ODG) Treatment in Workers' Compensation — Pain (Chronic)~~

~~Procedure Summary — Guidelines for~~ Chronic Pain ~~Treatment~~		
Procedure/Topic	**Recommendation**	**~~Summary of~~ Supporting medical evidence**
Click to go ahead: A | B | C | D | E | F | G | H | I | K | L | M | N | O | P | Q | R | S | T | U | V | W | Y | Z		
Acetaminophen (APAP)	Recommended for treatment of ~~acute pain,~~ chronic pain & acute exacerbations of chronic pain. With new information questioning the use of NSAIDs, acetaminophen should be recommended on a case-by-case basis. The side effect profile of NSAIDs may have been minimized in systematic reviews due to the short duration of trials. On the other hand, it now appears that acetaminophen may produce hypertension, a risk similar to that found for NSAIDs. ~~*Acute pain:* Recommended as an initial choice for treatment of acute pain. See Medications for acute pain (analgesics).~~ ~~*Osteoarthritis:*~~ Recommended as an initial treatment for mild to moderate pain, in particular, for those with gastrointestinal, cardiovascular and renovascular risk factors. (Laine, 2008) If pain is inadequately treated or there is evidence of inflammation, alternate pharmacologic treatment should be considered. In patients with moderate to severe disease, initial treatment with an NSAID may be warranted. The decision to use either class of drugs should be made on a case-by-case basis, incorporating factors including side effect profile and patient preferences. Current guidelines note that evidence is limited to make an initial recommendation with acetaminophen, and that NSAIDs may be more efficacious for treatment. In terms of treatment of the hand it should be noted that there are no placebo trials of efficacy and recommendations have been extrapolated from other joints. (Zhang, 2007) The selection of acetaminophen as a first-line treatment appears to be made primarily based on side effect profile in osteoarthritis guidelines. (Zhang, 2008) The most recent Cochrane review on this subject suggests that non-steroidal anti-inflammatory drugs (NSAIDs) are more efficacious for osteoarthritis than acetaminophen in terms of pain reduction, global assessments and improvement of functional status. No significant difference was found between overall safety, although patients taking NSAIDs were more likely to experience an adverse GI event. It is important to note that the median trial duration was only 6 weeks. (Towheed, 2006) See NSAIDs; NSAIDs, GI symptoms & cardiovascular risk; & NSAIDs, hypertension and renal function. Also see specific body-part chapters in the MTUS. *Adverse effects: Hepatotoxicity:* Acetaminophen overdose is a well-known cause of acute liver failure. Hepatotoxicity from therapeutic doses is unusual. (Hunt, 2007) A warning is given on all acetaminophen products that patients that consume ≥ 3 alcoholic drinks a day should discuss use with their physician, although a systematic review of acetaminophen use in alcoholic subjects concluded that there was little credible evidence to implicate therapeutic doses as a cause of fulminant hepatotoxicity in alcoholics. (Dart, 2007) Recent RCTs found that short-term treatment (3-5 days) of acetaminophen in newly abstinent alcoholic patients did not cause hepatic injury. (Kuffner, 2007) (Bartels, 2008) Acetaminophen, when used	

Procedure Summary — Pain	
Procedure/Topic	**Summary of medical evidence**
	at recommended maximum doses, may induce ALT elevations >3× ULN in up to nearly 40% of subjects. *Renal toxicity:* Renal insufficiency occurs in 1 to 2% of patients with overdose. (Mazer, 2008) *Hypertension and cardiovascular risk:* Cohort analysis reveals that acetaminophen use is associated with hypertension but evidence from randomized controlled trials is limited. This risk is similar to that found for NSAIDs. (Forman, 2007) (Montgomery, 2008) An increased cardiovascular risk was found in the Nurse's Health Study. (Chan, 2006) (Laine, 2007) (Laine, 2008) Acetaminophen may have more risks than originally thought, particularly when it is taken at the higher end of standard therapeutic doses. (Wise, 2015) *Dose:* Acetaminophen has been shown definitively to work synergistically with opioids, enhancing pain relief in a way that is opioid-sparing. (FDA, 2008) Despite acetaminophen's synergistic effect with opioids, fixed combination products are problematic because it is not possible to titrate the opioid dose relative to the acetaminophen dose when the fixed combination is used. Furthermore, in order for acetaminophen to produce an effective analgesic effect, it needs to be used on a regular basis. Consequently, it is best to administer acetaminophen as a single drug and on a routine basis and then, if necessary, to add an opioid as a single entity that may be titrated to effect. (Ray, 2013) ~~*Dose:*~~ The recommended dose for mild to moderate pain is 650 to 1000 mg orally every 6 hours with a FDA-approved maximum of 4 g/day. In calculating the maximum daily dose, it is necessary to combine all sources of acetaminophen in, including many OTC preparations as well as many common opioid combinations that include acetaminophen. An FDA advisory committee has recommended new restrictions on acetaminophen, voting that the single adult acetaminophen dose should be no more than 650 mg with a maximum total dose for 24 hours, decreased to no more than 3,250 mg. (FDA, 2009) The FDA asked drug manufacturers to limit the strength of acetaminophen in prescription drug products, predominantly combinations of acetaminophen and opioids, to 325 mg per pill, to reduce the risk of severe liver injury and allergic reactions. A Boxed Warning has been added to the label of all prescription drug products that contain acetaminophen. (FDA, 2011) To help encourage appropriate acetaminophen use, the newly implemented dosing instructions of Extra Strength Tylenol® (acetaminophen) have lowered the maximum daily dose from 8 pills per day (4,000 mg) to 6 pills per day (3,000 mg). The dosing interval has changed from 2 pills every 4–6 hours to 2 pills every 6 hours. (McNeil, 2014) Acetaminophen is best administered independently and on a routine basis with, if necessary, an opioid added as a single entity that may be titrated to effect.
Actiq® (oral transmucosal fentanyl lollipop)	Not recommended for chronic non-cancer pain. Actiq® (oral transmucosal fentanyl citrate), a fast-acting highly potent "lollipop" painkiller produced by Cephalon, is indicated only for the management of breakthrough cancer pain in patients with malignancies who are already receiving and who are tolerant to opioid therapy for their underlying persistent cancer pain. Actiq

Procedure Summary — Pain	
Procedure/Topic	**Summary of medical evidence**
	is contraindicated in acute pain; is not for use in chronic pain; and has a Black Box warning for abuse potential. See also Fentanyl.
Acupuncture	See the MTUS Acupuncture Treatment Guidelines for recommendations.
A-delta fiber electrodiagnostic testing	Not recommended. See Quantitative sensory threshold testing (QST) testing.
Alendronate (Fosamax®)	See Bisphosphonates. Bisphosphonates are a class of drugs that inhibit osteoclast action and the resorption of bone. Alendronate (Fosamax®) is in this class.
Alexander technique	See Education.
Alprazolam (Xanax®)	Not recommended for treatment of chronic pain or for ~~long-~~term use. See Benzodiazepines. Alprazolam, also known under the trade name Xanax and available generically, is a short-acting drug of the benzodiazepine class used to treat moderate to severe anxiety disorders, panic attacks, and as an adjunctive treatment for anxiety associated with major depression.
Ambien® (zolpidem tartrate)	Ambien® is a brand name for zolpidem tartrate produced by Sanofi-Aventis. See Zolpidem (Ambien®).
Amitriptyline	Recommended. Amitriptyline is a tricyclic antidepressant. Tricyclics are generally considered a first-line agent unless they are ineffective, poorly tolerated, or contraindicated. See ~~Antidepressants for chronic pain~~ for general guidelines, as well as specific Tricyclics ~~listing fo~~r more information and references.
Antianxiety drugs	See Anxiety medications in chronic pain.
Anticonvulsants	See Anti-epilepsy drugs (AEDs).
Antidepressants for chronic pain	Recommended as a first-line option for neuropathic pain, and as a possibility for non-neuropathic pain. *(Specify conditions for non-neuropathic pain)* (Feuerstein, 1997) (Perrot, 2006) Tricyclics are generally considered a first-line agent unless they are ineffective, poorly tolerated, or contraindicated. Analgesia generally occurs within a few days to a week, whereas antidepressant ~~effect takes lon~~ger to occur. (Saarto-*Cochrane*, 2005) Assessment of treatment efficacy should include not only pain outcomes, but also an evaluation of function, changes in use of other analgesic medication, sleep quality and duration, and psychological assessment. Side effects, including excessive sedation (especially that which would affect work performance) should be assessed. (Additional side effects are listed below for each specific drug.) It is recommended that these outcome measurements should be initiated at one week of treatment with a recommended trial of at least 4 weeks. The optimal duration of treatment is not known because most double-blind trials have been of short duration (6-12 weeks). It has been suggested that if pain is in remission for 3-6 months, a gradual tapering of anti- depressants may be undertaken. (Perrot, 2006) (Schnitzer, 2004) (Lin-*JAMA*, 2003) (Salerno, 2002) (Moulin, 2001) (Fishbain, 2000) (Taylor, 2004) (Gijsman, 2004) (Jick-*JAMA*, 2004) (Barbui, 2004) (Asnis, 2004) (Stein, 2003) (Pollack, 2003) (Ticknor, 2004) (Staiger, 2003) Long-term effectiveness of anti-depressants has not been established. (Wong, 2007) The effect of this class of medication in combination with other classes of

Procedu	Summary of medical evidence
	drugs has not been well researched. (Finnerup, 2005) The "number needed to treat" (NNT) methodology has been used to calculate efficacy of the different classes of antidepressants. (Sindrup, 2005) ***Specifically studied underlying pain etiologies:*** (also see below for specific drugs) *Neuropathic pain:* Tricyclic antidepressants are recommended as a first- line option, especially if pain is accompanied by insomnia, anxiety, or depression. (Saarto-Cochrane, 2007) (ICSI, 2007). ~~Other recent reviews recommend both tricyclic antidepressants and SNRIs (i.e., duloxetine and venlafaxine) as first-line options. (Dworkin, 2007) (Finnerup, 2007).~~ *Delete or clarify if the Chronic Pain Medical Treatment Guideline is recommending the use of both tricyclic antidepressants and SNRIs for chronic pain, otherwise this will foster confusion and disputes over whether this is an MTUS-recommended treatment.* *Non-neuropathic pain:* Recommended as an option in depressed patients, but effectiveness is limited *(Specify conditions)*. Non-neuropathic pain is generally treated with analgesics and anti-inflammatories. ~~In guidelines for painful rheumatic conditions recommended by Perrot, it was suggested that antidepressants may be prescribed as analgesics in non-depressed patients, with the first- line choice being tricyclics initiated at a low dose, increasing to a maximally tolerated dose. (Perrot, 2006)~~ *Delete or clarify the Chronic Pain Medical Treatment Guideline is recommending this treatment for chronic pain, otherwise this will foster confusion and disputes over whether or not this treatment is recommended in the MTUS.* ***Specific studied disease states*** *Fibromyalgia: Delete this segment unless there is a recommendation for the MTUS Chronic Pain Guidelines that is supported by the studies that follow.* There have been 25 controlled trials that have studied the use of antidepressants for fibromyalgia, including 3 meta-analyses. Good results were found with duloxetine in treating fibromyalgia (Arnold, 2007). Several studies evaluated tricyclics. (Perrot, 2006) (Moulin, 2001) A review of two double blind, placebo controlled trials concluded that duloxetine was safe and effective in women with fibromyalgia for up to 12 weeks (with long-term studies needed). (Arnold, 2007) Duloxetine is approved by the FDA for treatment of fibromyalgia. (FDA 2010) Another review indicated that there is strong evidence that amitriptyline is effective for fibromyalgia and suggested that more information is needed regarding the role of SNRIs and SSRIs. (Goldenberg, 2007) Compared with placebo, the SNRIs duloxetine (Cymbalta) and milnacipran (Savella) are slightly more likely to reduce pain in patients with fibromyalgia, according to a new Cochrane meta-analysis, but they are not superior in terms of reducing fatigue and sleep problems or in improving quality of life, and they appear to cause more adverse effects. (Häuser, 2013) ~~Refer to MTUS Low Back Complaints.~~ *Delete or clarify.* *Osteoarthritis:* No studies have specifically studied the use of antidepressants to treat pain from osteoarthritis. (Perrot, 2006) In depressed patients with osteoarthritis, improving depression symptoms was found to decrease pain and improve functional status. (Lin-JAMA, 2003) ***Antidepressant discontinuation****: Specify recommendations.* Nearly all classes of antidepressants have been linked to discontinuation reactions that are distinct from recurrence or relapse of underlying psychiatric pathology. It does appear that discontinuation reactions can occur regardless of the particular indication for use. The most common research involves discontinuation of serotonin-reuptake inhibitors (Serotonin-discontinuation syndrome). *Symptoms:* Symptoms of discontinuation vary between classes of

Procedure/Topic	Summary of medical evidence
	antidepressants, and between different drugs in the classes. These may include changes in mental/psychological status (confusion, restlessness, agitation, anxiety, worsening of mood, panic attacks, dysphoria, manic symptoms, and decreased level of consciousness), neurological changes (tremor, rigidity, clonus, myoclonus, hyperreflexia, ataxia, and rigidity), autonomic changes (diaphoresis, shivering, mydriasis, nausea and diarrhea), and changes in vital signs (tachycardia, hypertension, hyperthermia, and tachypnea). Commonly patients describe both psychological and somatic symptoms (the latter described as flu-like, with or without gastrointestinal physical symptoms). Symptoms are thought to occur in at least 20% to 25% of patients upon discontinuing of serotonin-reuptake inhibitors (with reports of at least 50% with drugs with shorter half-lives such as paroxetine or venlafaxine). Symptoms tend to emerge within 2 to 5 days with a usual duration of 1 to 2 weeks. The primary risk factors for this reaction include use of antidepressants with shorter half-lives, longer duration of treatment, and abrupt discontinuation. *Specify recommendation*. *Differentiation from depression relapse or recurrence:* Differentiating factors include looking for symptoms that are more likely to occur with discontinuation reaction (dizziness, electric shock-like sensations, "rushing" sensations, headache and nausea) as well as observing for rapid reversal of symptoms (complete resolution within 1 to 2 weeks of the taper/discontinuation is less likely to be due to depression). Later onset of symptoms (after at least two to three weeks of discontinuation/tapering) or prolonged symptoms (3 weeks or greater) are more commonly associated with a relapse of psychiatric pathology or another intercurrent disease. *Consider deleting since here anti-depressant was prescribed for chronic pain relief rather than psych treatment.* **SPECIFIC ANTIDEPRESSANTS:** ***Tricyclic antidepressants*** are recommended over selective serotonin reuptake inhibitors (SSRIs), unless adverse reactions are a problem. Caution is required because tricyclics have a low threshold for toxicity, and tricyclic antidepressant overdose is a significant cause of fatal drug poisoning due to their cardiovascular and neurological effects. Tricyclic antidepressants have been shown in both a meta-analysis (McQuay, 1996) and a systematic review (Collins, 2000) to be effective, and are considered a first-line treatment for neuropathic pain. (Namaka, 2004) (Dworkin, 2003) (Gilron, 2006) (Wolfe, 2004) (Dworkin, 2007) (Saarto- Cochrane, 2007) This class of medications works in both patients with normal mood and patients with depressed mood when used in treatment for neuropathic pain. (Sindrup, 2005) Indications in controlled trials have shown effectiveness in treating central post-stroke pain, post-herpetic neuralgia (Argoff, 2004), painful diabetic and non-diabetic polyneuropathy, and post-mastectomy pain. Negative results were found for spinal cord pain and phantom-limb pain, but this may have been due to study design. (Finnerup, 2005) Tricyclics have not demonstrated significance in randomized-control trials in treating HIV neuropathy, spinal cord injury, cisplatinum neuropathy, neuropathic cancer pain, phantom limb pain or chronic lumbar root pain. (Dworkin, 2007) One review reported the NNT for at least moderate neuropathic pain relief with tricyclics is 3.6 (3-4.5), with

Procedure Summary — Pain	
Procedure/Topic	**Summary of medical evidence**
	the NNT for amitriptyline being 3.1 (2.5-4.2). The NNT for venlafaxine, calculated using 3 studies, was reported to be 3.1 (2.2-5.1). (Saarto-*Cochrane*, 2007) Another review reported that the NNT for 50% improvement in neuropathic pain was 2 to 3 for tricyclic antidepressants, 4 for venlafaxine, and 7 for SSRIs (Perrot, 2008). *Side-effect profile:* Tricyclics are ~~contraindicated~~ not recommended in patients with cardiac conduction disturbances and/or decompensation (they can produce heart block and arrhythmias) as well as for those patients with epilepsy. For patients > 40 years old, a screening ECG is recommended prior to initiation of therapy. (Dworkin, 2007) (ICSI, 2007) They can create anticholinergic side effects of dry mouth, sweating, dizziness, orthostatic hypotension, fatigue, constipation, and urinary retention. (Finnerup, 2005) To minimize side effects, it is ~~suggested~~ recommended that titration ~~should~~ be slow and based on the patient's response. (Namaka, 2004) An alternative choice may be a SNRI*(conditions?)*. (Finnerup, 2005) (Sindrup, 2005) (Dworkin, 2007) The muscle relaxant cyclobenzaprine is closely related to the tricyclic antidepressants so caution is advised when using cyclobenzaprine. (FDA, 2011) *Dosing Information:* *Amitriptyline*: *Neuropathic pain*: *Specify recommendations.* The starting dose may be as low as 10-25 mg at night, with increases of 10-25 mg once or twice a week up to 100 mg/day. (ICSI, 2007) The lowest effective dose should be used (Dworkin, 2007). *Fibromyalgia*: One review recommended the following dosing regimen: Start with low doses, such as 5-10 mg 1-3 hours before bedtime. Dose may be increased by 5 mg at two-week intervals; final dose is dependent upon efficacy and patient tolerability to side effects. Doses that have been studied range from 25 to 50 mg at bedtime. (Goldenberg, 2007) ***Selective serotonin and norepinephrine reuptake inhibitors (SNRIs):*** *Duloxetine (Cymbalta®)*: FDA-approved for anxiety, depression, diabetic neuropathy, fibromyalgia and chronic musculoskeletal pain. (FDA, 2010) Used off-label for neuropathic pain and radiculopathy. Duloxetine is recommended as a first-line option for diabetic neuropathy. (Dworkin, 2007) No high-quality evidence is reported to support the use of duloxetine for lumbar radiculopathy *(specify not recommended)*. (Dworkin, 2007) More studies are needed to determine the efficacy of duloxetine for other types of neuropathic pain. *Side effects:* CNS: dizziness, fatigue, somnolence, drowsiness, anxiety (3% vs.2% for placebo), insomnia (8-13% vs. 6-7% for placebo). GI: nausea and vomiting (5-30%), weight loss (2%). Duloxetine can worsen diabetic control in some patients. It also causes sexual dysfunction. (Maizels, 2005) *Dosing:* 60 mg once a day as an off-label option for chronic pain syndromes. Dosage adjustment may be required in patients with renal insufficiency. *(Specify as recommendations with citations)* *Venlafaxine (Effexor®):* FDA-approved for anxiety, depression, panic disorder and social phobias. Off-label use for fibromyalgia, neuropathic pain, and diabetic neuropathy. *(Delete or recommend with citations)* *Side-effect profile:* CNS: (≥ 5%) drowsiness, weakness, dizziness, dry

Procedure Summary — Pain	
Procedure/Topic	**Summary of medical evidence**
	mouth, insomnia, nervousness/anxiety (13/6% vs. 6/3%), tremor, headache, seizures. GI: N&V, constipation, weight loss (2-18%). Pre-existing hypertension should be controlled. Cholesterol may be increased (5%). Sexual dysfunction has also been noted. (Maizels, 2005) (ICSI, 2007) *Dosing: Neuropathic pain (off-label indication):* 37.5 mg once daily, increase by 37.5 mg per week up to 300 mg daily. (Maizels, 2005) (ICSI, 2007) *Trial period:* Some relief may occur in first two weeks; full benefit may not occur until six weeks. Withdrawal effects can be severe. Abrupt discontinuation should be avoided and tapering is recommended before Discontinuation *(Add specific recommendations)*. ***Bupropion (Wellbutrin®),*** a second-generation non-tricyclic antidepressant (a noradrenaline and dopamine reuptake inhibitor) has been shown to be effective in relieving neuropathic pain of different etiologies in a small trial (41 patients). (Finnerup, 2005) While bupropion has shown some efficacy in neuropathic pain there is no evidence of efficacy in patients with non-neuropathic chronic low back pain. (Katz, 2005) Furthermore, a recent review suggested that bupropion is generally a third-line medication for diabetic neuropathy and may be considered when patients have not had a response to a tricyclic or SNRI. (Dworkin, 2007) *Add specific recommendations.* *Side-effect profile:* Headache, agitation, insomnia, anorexia, weight loss *Dosing Information: Neuropathic pain (off-label indication):* 100 mg once daily, increase by 100 mg per week up to 200 mg twice daily. (Maizels, 2005) ***Selective serotonin reuptake inhibitors (SSRIs),*** a class of antidepressants that inhibit serotonin reuptake without action on noradrenaline, are controversial based on controlled trials. (Finnerup, 2005) (Saarto-*Cochrane*, 2005) It has been suggested that the main role of SSRIs may be in addressing psychological symptoms associated with chronic pain. (Namaka, 2004) More information is needed regarding the role of SSRIs and pain. *Needs clarification and clean-up.* *Side Effects: Bleeding:* An association has been found between the use of SSRI antidepressants and gastrointestinal bleeding. This risk is increased with the concomitant use of ASA or NSAIDs. It is ~~suggested~~ recommended increased risk for GI bleeding be discussed with patients that have other risks for GI bleeding. An association with increased intraoperative blood loss has also been found with SSRI use. (Movig, 2003) A treatment option for those at risk for bleeding includes switching to an antidepressant with a lower degree of inhibition of serotonin reuptake (Intermediate reuptake: venlafaxine, amitriptyline, imipramine, citalopram; Low reuptake: desipramine, doxepin, trazodone, bupropion, mirtazapine). SSRIs with the highest degree of inhibition of serotonin reuptake include paroxetine, sertraline, and fluoxetine. (Looper, 2007) *Specify recommendations.*
Antiemetics (for opioid nausea)	Not recommended for nausea and vomiting secondary to chronic opioid use. ~~Recommended for acute use as noted below per FDA-approved indications.~~ Nausea and vomiting is common with use of opioids. These

Procedure Summary — Pain	
Procedure/Topic	**Summary of medical evidence**
	side effects tend to diminish over days to weeks of continued exposure. Studies of opioid adverse effects including nausea and vomiting are limited to short-term duration (less than four weeks) and have limited application to long-term use. If nausea and vomiting remains prolonged, other etiologies of these symptoms should be evaluated for. The differential diagnosis includes gastroparesis (primarily due to diabetes). Current research for treatment of nausea and vomiting as related to opioid use primarily addresses the use of antiemetics in patients with cancer pain or those utilizing opioids for acute/postoperative therapy. Recommendations based on these studies cannot be extrapolated to chronic non-malignant pain patients. There is no high-quality literature to support any one treatment for opioid-induced nausea in chronic non-malignant pain patients. (Moore 2005) *Make specific recommendations.* *Promethazine (Phenergan®):* This drug is a phenothiazine. It is recommended as a sedative and antiemetic in pre-operative and post-operative situations. Multiple central nervous system effects are noted with use including somnolence, confusion and sedation. Tardive dsykensia is also associated with use. This is characterized by involuntary movements of the tongue, mouth, jaw, and/or face. Choreoathetoid movements of the extremities can also occur. Development appears to be associated with prolonged treatment and in some cases can be irreversible. Anticholinergic effects can occur (dry mouth, dry eyes, urinary retention and ileus). *Ondansetron (Zofran®)*: This drug is a serotonin 5-HT3 receptor antagonist. It is FDA-approved for nausea and vomiting secondary to chemotherapy and radiation treatment. It is also FDA-approved for postoperative use. Acute use is FDA-approved for gastroenteritis. See also Nabilone (Cesamet®), for chemotherapy-induced nausea, but not pain. *Make specific recommendations*
Anti-epilepsy drugs (AEDs) for pain	*Anti-epilepsy drugs (AEDs) are also referred to as anti-convulsants.* Recommended for some neuropathic pain (pain due to nerve damage) *Specify conditions.*, but not for acute nociceptive pain (including somatic pain). (Gilron, 2006) (Wolfe, 2004) (Washington, 2005) (ICSI, 2005) (Wiffen-*Cochrane*, 2005) (Attal, 2006) (Wiffen-*Cochrane*, 2007) (Gilron, 2007) (ICSI, 2007) (Finnerup, 2007) (Wiffen-Cochrane, 2013) There is a lack of expert consensus on the treatment of neuropathic pain in general due to heterogeneous etiologies, symptoms, physical signs and mechanisms. Most randomized controlled trials (RCTs) for the use of this class of medication for neuropathic pain have been directed at postherpetic neuralgia and painful polyneuropathy (with diabetic polyneuropathy being the most common example). There are few RCTs directed at central pain and none for painful radiculopathy. (Attal, 2006) The choice of specific agents reviewed below will depend on the balance between effectiveness and adverse reactions. See also specific drug listings below: Gabapentin (Neurontin®); Pregabalin (Lyrica®); Lamotrigine (Lamictal®); Carbamazepine (Tegretol®); Oxcarbazepine (Trileptal®); Phenytoin (Dilantin®); Topiramate

Procedure Summary — Pain	
Procedure/Topic	**Summary of medical evidence**
	& Tiagabine (Gabitril®) *Outcomes:* A "good" response to the use of AEDs has been defined as a 50% reduction in pain and a "moderate" response as a 30% reduction. It has been reported that a 30% reduction in pain is clinically important to patients and a lack of response of this magnitude may be the "trigger" for the following: (1) a switch to a different first-line agent (TCA, SNRI or AED are considered first-line treatment); or (2) combination therapy if treatment with a single drug agent fails. (Eisenberg, 2007) (Jensen, 2006) After initiation of treatment there should be documentation of pain relief and improvement in function as well as documentation of side effects incurred with use. The continued use of AEDs depends on improved outcomes versus tolerability of adverse effects. AEDs are associated with teratogenicity, so they must be used with caution in woman of childbearing age. Preconception counseling is recommended for anticonvulsants (due to reductions in the efficacy of birth control pills). (Clinical Pharmacology, 2008) Manufacturers of antiepileptic drugs will need to add a warning to their labeling indicating that use of the drugs increases risk for suicidal thoughts and behaviors, according to an FDA Alert issued December 16. (FDA MedWatch, 2008) *Specifically studied disease states:* (also see below for specific drugs) *Painful polyneuropathy:* AEDs are recommended on a trial basis (gabapentin/pregabalin) as a first-line therapy for painful polyneuropathy (with diabetic polyneuropathy being the most common example). The other first-line options are a tri-cyclic antidepressant (if tolerated by the patient), or a SNRI antidepressant (such as duloxetine) *Specify conditions for tri-cyclic antidepressants and SNRI antidepressants*. (Attal, 2006) (Jensen, 2006) *Postherpetic neuralgia:* Gabapentin and pregabalin are recommended. (Attal, 2006) (Backonja, 2004) ~~*Central pain:* There are so few trials (with such small sample size) that treatment is generally based on that recommended for peripheral neuropathy, with gabapentin and pregabalin recommended. Lamotrigine has been found to be effective for central post-stroke pain (see below for specific drugs), and gabapentin has also been found to be effective. (Backonja, 2004)~~ *Delete or clarify that these MTUS Chronic Pain Guidelines recommend gabapentin and pregablin, and/or lamotrigine.* ~~*Acute pain:* Not indicated due to lack of evidence.~~ *Treatment of pain associated with osteoarthritis of the hip:* Not ~~indicated~~ recommended *Spinal cord injury:* Gabapentin is recommended for chronic neuropathic pain. (Levendoglu, 2004) ~~*CRPS:* Gabapentin has been recommended (Serpell, 2002)~~ *Delete or clarify that these MTUS Chronic Pain Guidelines recommend gabapentin for chronic pain associated with CRPS.*~~*Fibromyalgia:* Gabapentin and pregabalin have been found to be safe and efficacious to treat pain and other symptoms. (Arnold, 2007) (Crofford, 2005) Pregabalin is FDA approved for fibromyalgia.~~ *Delete or clarify that these MTUS Chronic Pain Guidelines recommend gabapentin and pregabalin for fibromyalgia pain* *Lumbar spinal stenosis:* Gabapentin produced statistically significant improvement in walking distance, decrease in pain with movement and sensory deficit in a pilot study. (Yaksi, 2007) *Myofascial pain:* Not recommended. There is a lack of evidence to demonstrate that AEDs significantly reduce the level of myofascial or acute

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	musculoskeletal pain, or other sources of somatic pain. (Wiffen-*Cochrane*, 2005) (Washington, 2005 *Postop pain:* AEDs may also be an option for postoperative pain, resulting in decreased opioid consumption. (Peng, 2007) (Buvanendran, 2007) **SPECIFIC ANTI-EPILEPSY DRUGS:** ***Gabapentin (Neurontin®, Gabarone™, generic available)*** has been shown to be effective for treatment of diabetic painful neuropathy and postherpetic neuralgia and has been considered as a first-line treatment for neuropathic pain. (Backonja, 2002) (ICSI, 2007) (Knotkova, 2007) (Eisenberg, 2007) (Attal, 2006) (Wiffen-*Cochrane*, 2013) This RCT concluded that gabapentin monotherapy appears to be efficacious for the treatment of pain and sleep interference associated with diabetic peripheral neuropathy and exhibits positive effects on mood and quality of life. (Backonja, 1998) It has been given FDA approval for treatment of post-herpetic neuralgia. The number needed to treat (NNT) for overall neuropathic pain is 4. It has a more favorable side-effect profile than Carbamazepine, with a number needed to harm of 2.5. (Wiffen2-Cochrane, 2005) (Zaremba, 2006) Gabapentin in combination with morphine has been studied for treatment of diabetic neuropathy and postherpetic neuralgia. When used in combination the maximum tolerated dosage of both drugs was lower than when each was used as a single agent and better analgesia occurred at lower doses of each. (Gilron-NEJM, 2005) Recommendations involving combination therapy require further study. *Mechanism of action:* This medication appears to be effective in reducing abnormal hypersensitivity (allodynia and hyperalgesia), to have anti-anxiety effects, and may be beneficial as a sleep aid. (Arnold, 2007) *Specific pain states:* *Acute pain*: There is limited evidence to show that this medication is effective for acute pain, and for postoperative pain, where there is fairly good evidence that the use of gabapentin and gabapentin-like compounds results in decreased opioid consumption. This beneficial effect, which may be related to an anti-anxiety effect, is accompanied by increased sedation and dizziness. (Peng, 2007) (Buvanendran, 2007) (Menigaux, 2005) (Pandey, 2005) *Spinal cord injury:* Recommended as a trial for chronic neuropathic pain that is associated with this condition. (Levendoglu, 2004) *CRPS:* Recommended as a trial. (Serpell, 2002) *Fibromyalgia:* Recommended as a trial. (Arnold, 2007) *Lumbar spinal stenosis:* Recommended as a trial, with statistically significant improvement found in walking distance, pain with movement, and sensory deficit found in a pilot study. (Yaksi, 2007) *Side-Effect Profile*: Gabapentin has a favorable side-effect profile, few clinically significant drug-drug interactions and is generally well tolerated; however, common side effects include dizziness, somnolence, confusion, ataxia, peripheral edema, and dry mouth. (Eisenberg, 2007) (Attal, 2006) Weight gain is also an adverse effect.

	inserted central catheter (PICC line), a form of intravenous access that can be used for a prolonged period of time for extended antibiotic therapy, may be required. Urgent consultation with a surgeon should be sought in cases of crepitus, circumferential cellulitis, necrotic-appearing skin, rapidly evolving cellulitis, pain disproportional to physical examination findings, severe pain on passive movement, or other clinical indications of necrotizing fasciitis. (Stevens, 2005) (Liu, 2011)
Cesamet®	See Nabilone.
Chi machine	Not recommended for chronic pain. May be used for lymphedema, but not recommended for other conditions, including chronic pain, since there is no evidence of its effectiveness.
Chiropractic treatment	See Manual therapy & manipulation.
Chlordiazepoxide	Not recommended. See Benzodiazepines.
Cholecalciferol	See Vitamin D.
Chondroitin sulfate	See Glucosamine (and Chondroitin Sulfate).
Chronic pain programs / (Functional restoration programs [FRPs])	Recommended where there is access to programs with proven successful outcomes (i.e., decreased pain and medication use, improved function and return to work, decreased utilization of the health care system), for patients with conditions that have resulted in "Delayed recovery." Also see Introduction to the MTUS Chronic Pain Guidelines. There should be evidence that a complete diagnostic assessment has been made, with a detailed treatment plan of how to address physiologic, psychological and sociologic components that are considered components of the patient's pain. Patients should show evidence of motivation to improve and return to work, and meet the patient selection criteria outlined below. While these programs are recommended (see criteria below), the research remains ongoing as to (1) what is considered the "gold-standard" content for treatment; (2) the group of patients that benefit most from this treatment; (3) the ideal timing of when to initiate treatment; (4) the intensity necessary for effective treatment; and (5) cost-effectiveness. It has been suggested that interdisciplinary/multidisciplinary care models for treatment of chronic pain may be the most effective way to treat this condition. (Flor, 1992) (Gallagher, 1999) (Guzman, 2001) (Gross, 2005) (Sullivan, 2005) (Dysvik, 2005) (Airaksinen, 2006) (Schonstein, 2003) (Sanders, 2005) (Patrick, 2004) (Buchner, 2006) These treatment modalities are based on the biopsychosocial model, one that views pain and disability in terms of the interaction between physiological, psychological and social factors. (Gatchel, 2005) See Biopsychosocial model of chronic pain. ***Types of programs:*** There is no one universal definition of what comprises interdisciplinary/multidisciplinary treatment. These pain rehabilitation programs (as described below) combine multiple treatments, and at the least, include psychological care along with physical and/or occupational therapy (including an active exercise component as opposed to passive modalities). The most commonly referenced programs have been defined in the following general ways (Stanos, 2006): (1) Multidisciplinary programs: Involves one or two specialists directing the services of a number of team members, with these specialists often having independent goals. These programs can be further subdivided into four

	levels of pain programs: (a) Multidisciplinary pain centers (generally associated with academic centers and include research as part of their focus) (b) Multidisciplinary pain clinics (c) Pain clinics (d) Modality-oriented clinics (2) Interdisciplinary pain programs: Involves a team approach that is outcome focused and coordinated and offers goal-oriented interdisciplinary services. Communication on a minimum of a weekly basis is emphasized. The most intensive of these programs is referred to as a Functional Restoration Program, with a major emphasis on maximizing function versus minimizing pain. See Functional restoration programs. ***Types of treatment:*** Components suggested for interdisciplinary care include the following services delivered in an integrated fashion: (a) physical treatment; (b) medical care and supervision; (c) psychological and behavioral care; (d) psychosocial care;and (e) education. ***Outcomes measured:*** Studies have generally evaluated variables such as pain relief, function and return to work. More recent research has begun to investigate the role of comorbid psychiatric and substance abuse problems in relation to treatment with pain programs. Recent literature has begun to suggest that an outcome of chronic pain programs may be to "demedicalize" treatment of a patient, and encourage them to take a more active role in their recovery. These studies use outcomes such as use of the medical care system post-treatment. The role of the increasing use of opioids and other medications (using data collected over the past decade) on outcomes of functional restoration is in the early stages, and it is not clear how changes in medication management have affected outcomes, if at all. See MTUS Opioids Treatment Guidelines for recommendations on the use of opioids. Also see specific body-part chapters in the MTUS. *Multidisciplinary back training*: (involvement of psychologists, physiotherapists, occupational therapists, and/or medical specialists). The training program is partly based on physical training and partly on behavioral cognitive training. Physical training is performed according to the "graded activity" principle. The main goal is to restore daily function. A recent review of randomized controlled studies of at least a year's duration found that this treatment modality produced a positive effect on work participation and possibly on quality of life. There was no long-term effect on experienced pain or functional status (this result may be secondary to the instrument used for outcome measure). Intensity of training had no substantial influence on the effectiveness of the treatment. (van Geen, 2007) (Bendix, 1997) (Bendix, 1998) (Bendix2, 1998) (Bendix, 2000) (Frost, 1998) (Harkapaa, 1990) (Skouen, 2002) (Mellin, 1990) (Haldorsen, 2002) *Intensive multidisciplinary rehabilitation of chronic low back pain:* The most recent Cochrane study was withdrawn from the Cochrane (3/06) as the last literature search was performed in 1998. Studies selected included a physical dimension treatment and at least one other treatment. ***Role of opioid use:*** See MTUS Opioids Treatment Guidelines for recommendations on the use of opioids.

Role of comorbid psychiatric illness: Comorbid conditions, including psychopathology, should be recognized as they can affect the course of chronic pain treatment. In a recent analysis, patients with panic disorder, antisocial personality disorder and dependent personality disorder were > 2 times more likely to not complete an interdisciplinary program. Personality disorders in particular appear to hamper the ability to successfully complete treatment. Patients diagnosed with post-traumatic stress disorder were 4.2 times more likely to have additional surgeries to the original site of injury. (Dersh, 2007) The prevalence of depression and anxiety in patients with chronic pain is similar. Cohort studies indicate that the added morbidity of depression and anxiety with chronic pain is more strongly associated with severe pain and greater disability. (Poleshuck, 2009) (Bair, 2008)

Predictors of success and failure: As noted, one of the criticisms of interdisciplinary/multidisciplinary rehabilitation programs is the lack of an appropriate screening tool to help to determine who will most benefit from this treatment. Retrospective research has examined decreased rates of completion of functional restoration programs, and there is ongoing research to evaluate screening tools prior to entry. (Gatchel, 2006) There is need for research in terms of necessity and/or effectiveness of counseling for patients considered to be "at-risk" for post-discharge problems. (Proctor, 2004) The following variables have been found to be negative predictors of efficacy of treatment with the programs as well as negative predictors of completion of the programs: (1) a negative relationship with the employer/supervisor; (2) poor work adjustment and satisfaction; (3) a negative outlook about future employment; (4) high levels of psychosocial distress (higher pretreatment levels of depression, pain and disability); (5) involvement in financial disability disputes; (6) greater rates of smoking; (7) increased duration of pre-referral disability time; (8) higher prevalence of opioid use; and (9) elevated pre-treatment levels of pain. (Linton, 2001) (Bendix, 1998) (McGeary, 2006) (McGeary, 2004) (Gatchel2, 2005) (Dersh, 2007)

Role of duration of disability: There is little research as to the success of return to work with functional restoration programs in long-term disabled patients (> 24 months).

Studies supporting programs for patients with long-term disability: Long-term disabled patients (at least 18 months) vs. short-term disabled (4 to 8 months) were evaluated using Pride data (1990-1993). No control was given for patients that did not undergo a program. During the time studied program dropouts averaged 8% to 12%. (It does appear that at the time of this study, participants in the program were detoxified from opioids prior to beginning.) The long-term disabled group was more likely to have undergone spinal surgery, with this likelihood increasing with time. Return to work was statistically different between the short-term disabled (93%) and the long-term disabled-18 months (80%). The long-term disabled-24 months group had a 75% return to work. Long-term disabled-18 month patients were statistically more likely to visit new health providers than short-term disabled patients (34% and 25% respectively). Work retention at one year in groups up to 24 months duration of disability was 80%. This dropped to 66% in the group that had been disabled for > 24 months. The

	percentage of recurrent lost time injury claims increased from around 1% in the groups disabled for < 35 months to 8.3% in the groups disabled for > 36 months. A main criterion for success appeared to be the decision of the patient to actively participate in the program rehabilitation goals. (Jordan, 1998) *Studies suggesting limited results in patients with long-term disability:* While early studies have suggested that time out-of-work is a predictor of success for occupational outcomes, these studies have flaws when an attempt is made to apply them to chronic pain programs. (Gallagher, 1989) (Beals, 1972) (Krause, 1994) Washington State studied the role of duration of work injury on outcome using a statistical model that allowed for a comparison of patients that participated in a multidisciplinary pain program (using data from 1991-1993) vs. those that were evaluated and not treated. This was not an actual study of time of disability, but of duration of injury (mean years from injury to evaluation of 2.6 years for the treated group and 4.0 years for the evaluated only group). The original statistical analysis allowed for a patient to be included in a "treated group" for those individuals that both completed and did not complete the program. Data was collected from 10 sites. Each of the centers was CARF approved and included Pysch/behavioral treatment, vocation counseling and physical therapy. A sub-study evaluated a comparison of patients that were treatment completers vs. those that did not participate (78.6%, N-=963). No information was given in terms of surgical procedures or medications. The primary outcome was time loss status of subjects 2 years after they had undergone the index pain center evaluation. In the 2001 study, if chronicity of duration of injury was controlled for, there was no significant benefit produced in terms of patients that were receiving time-loss benefits at 2-years post treatment between the two groups. Approximately 60% of both groups were not receiving benefits at the two-year period. As noted, the "treated patient" was only guaranteed to have started a program. A repeat analysis of only the patients who completed the study did not significantly change the results of the study. In a 2004 survey follow-up no significant difference was found between treated and untreated groups, although the treated group had better response. The survey response was 50%, and the treatment responders were more likely to be disabled at the time of the survey. The authors suggest that the results indicated early intervention was a key to response of the programs, and that modest goals (improvement, not cure) be introduced. (Robinson, 2004) (Robinson, 2001) [The authors also concluded that there was no evidence that pain center treatment affects either disability status or clinical status of injured workers.] ***Timing of use**:* Intervention as early as 3 to 6 months post-injury may be recommended depending on identification of patients that may benefit from a multidisciplinary approach (from programs with documented positive outcomes). See Chronic pain programs, early intervention. ***Role of post-treatment care (as an outcome):*** Three variables are usually examined; (1) New surgery at the involved anatomic site or area; (2) Percentage of patients seeking care from a new provider; (3) Number of visits to the new provider over and above visits with the health-care professional overseeing treatment. It is suggested that a "new provider" is

more likely to reorder diagnostic tests, provide invasive procedures, and start long-term analgesics. In a study to determine the relationship between post-treatment healthcare-seeking behaviors and poorer outcomes (using prospectively analyzed PRIDE data on patients with work-related musculoskeletal injuries), patients were compared that accessed healthcare with a new provider following functional restoration program completion (approximately 25%) to those that did not. The former group was significantly more likely to have an attorney involved with their case (22.7% vs. 17.1%, respectively), and to have had pre-rehabilitation surgery (20.7% vs. 12.1%, respectively). Return to work was higher in the group that did not access a new provider (90% vs. 77.6% in the group that did access). The group that did not access new providers also was more likely to be working at one year (88% vs. 62.2% in the group that accessed new providers). It should be noted that 18% of the patients that entered the program dropped out or were asked to leave. The authors suggested monitoring of additional access of healthcare over and above that suggested at the end of the program, with intervention if needed. (Proctor, 2004) The latest AHRQ Comparative Effectiveness Research supports the ODG recommendations. (AHRQ, 2011)

See also Chronic pain programs, intensity; Chronic pain programs, opioids; Functional restoration programs; Chronic pain programs, early intervention; Progressive goal attainment program (PGAP™).

Criteria for the general use of multidisciplinary pain management programs:

Outpatient pain rehabilitation programs are recommended if all ~~may be considered medically necessary in~~ the following circumstances apply:

these changes are necessary to clarify when the programs are necessary

(1) The patient has a chronic pain syndrome, with evidence of significant loss of function that persists beyond three months and has evidence of three or more of the following *too often patients are inappropriately referred when the loss of function is insignificant*: (a) Excessive dependence on health-care providers, spouse, or family; (b) Secondary physical deconditioning due to disuse and/or fear-avoidance of physical activity due to pain; (c) Withdrawal from social activities or normal contact with others, including work, recreation, or other social contacts; (d) Failure to restore preinjury function after a period of disability such that the physical capacity is insufficient to pursue work, family, or recreational needs; (e) Development of psychosocial sequelae that limits function or recovery after the initial incident, including anxiety, fear-avoidance, depression, sleep disorders, or nonorganic illness behaviors (with a reasonable probability to respond to treatment intervention); (f) The diagnosis is not primarily a personality disorder or psychological condition without a physical component; (g) There is evidence of continued use of prescription pain medications (particularly those that may result in tolerance, dependence or abuse) without evidence of improvement in pain or function.

(2) Previous methods of treating chronic pain have been unsuccessful and there is an absence of other options likely to result in significant clinical improvement.

(3) An adequate and thorough multidisciplinary evaluation has been made. This should include pertinent validated diagnostic testing that addresses the following: (a) A physical exam that rules out conditions that require treatment prior to initiating the program. All diagnostic procedures

	necessary to rule out treatable pathology, including imaging studies and invasive injections (used for diagnosis), should be completed prior to considering a patient a candidate for a program. The exception is diagnostic procedures that were repeatedly requested and not authorized. Although the primary emphasis is on the work-related injury, underlying non-work related pathology that contributes to pain and decreased function may need to be addressed and treated by a primary care physician prior to or coincident to starting treatment; (b) Evidence of a screening evaluation should be provided when addiction is present or strongly suspected; (c) Psychological testing using a validated instrument to identify pertinent areas that need to be addressed in the program (including but not limited to mood disorder, sleep disorder, relationship dysfunction, distorted beliefs about pain and disability, coping skills and/or locus of control regarding pain and medical care) or diagnoses that would better be addressed using other treatment should be performed; (d) An evaluation of social and vocational issues that require assessment. (4) If a goal of treatment is to prevent or avoid controversial or optional surgery, a trial of 10 visits (80 hours) may be implemented to assess whether surgery may be avoided. (5) If a primary reason for treatment in the program is addressing possible substance use issues, an evaluation with an addiction clinician may be indicated upon entering the program to establish the most appropriate treatment approach (pain program vs. substance dependence program). This must address evaluation of drug abuse or diversion (and prescribing drugs in a non-therapeutic manner). In this particular case, once drug abuse or diversion issues are addressed, a 10-day trial may help to establish a diagnosis, and determine if the patient is not better suited for treatment in a substance dependence program. Addiction consultation can be incorporated into a pain program. If there is indication that substance dependence may be a problem, there should be evidence that the program has the capability to address this type of pathology prior to approval. (6) Once the evaluation is completed, a treatment plan should be presented with specifics for treatment of identified problems, and outcomes that will be followed. (7) There should be documentation that the patient has motivation to change, and is willing to change their medication regimen (including decreasing or actually weaning substances known for dependence). There should also be some documentation that the patient is aware that successful treatment may change compensation and/or other secondary gains. In questionable cases, an opportunity for a brief treatment trial may improve assessment of patient motivation and/or willingness to decrease habituating medications. (8) Negative predictors of success (as outlined above) should be identified, and if present, the pre-program goals should indicate how these will be addressed. (9) If a program is planned for a patient that has been continuously disabled for greater than 24 months, the outcomes for the necessity of use should be clearly identified, as there is conflicting evidence that chronic pain programs provide return-to-work beyond this period. These other desirable types of outcomes include decreasing post-treatment care

	including medications, injections and surgery. This cautionary statement should not preclude patients off work for over two years from being admitted to a multidisciplinary pain management program with demonstrated positive outcomes in this population. (10) Treatment is not suggested for longer than 2 weeks without evidence of compliance and significant demonstrated efficacy as documented by subjective and objective gains. (Note: Patients may get worse before they get better. For example, objective gains may be moving joints that are stiff from lack of use, resulting in increased subjective pain.) However, it is also not suggested that a continuous course of treatment be interrupted at two weeks solely to document these gains, if there are preliminary indications that they are being made on a concurrent basis. (11) Integrative summary reports that include treatment goals, compliance, progress assessment with objective measures and stage of treatment, must be made available upon request at least on a bi-weekly basis during the course of the treatment program. (12) Total treatment duration should generally not exceed 4 weeks (20 full-days or 160 hours), (or the equivalent in part-day sessions if required by part-time work, transportation, childcare, or comorbidities). (Sanders, 2005) If treatment in excess of 4 weeks is required, a clear rationale for the specified extension and reasonable goals to be achieved should be provided. Longer durations require individualized care plans explaining why improvements cannot be achieved without an extension as well as evidence of documented improved outcomes from the facility (particularly in terms of the specific outcomes that are to be addressed). (13) At the conclusion and subsequently, neither re-enrollment in repetition of the same or similar rehabilitation program (e.g. work hardening, work conditioning, out-patient medical rehabilitation) is medically warranted for the same condition or injury (with possible exception for a medically necessary organized detox program). Prior to entry into a program the evaluation should clearly indicate the necessity for the type of program required, and providers should determine upfront which program their patients would benefit more from. A chronic pain program should not be considered a "stepping stone" after less intensive programs, but prior participation in a work conditioning or work hardening program does not preclude an opportunity for entering a chronic pain program if otherwise indicated. (14) Suggestions for treatment post-program should be well documented and provided to the referral physician. The patient may require time- limited, less intensive post-treatment with the program itself. Defined goals for these interventions and planned duration should be specified. (15) Post-treatment medication management is particularly important. Patients that have been identified as having substance abuse issues generally require some sort of continued addiction follow-up to avoid relapse. Inpatient pain rehabilitation programs: These programs typically consist of more intensive functional rehabilitation and medical care than their outpatient counterparts. They ~~may be appropriate~~ are recommended for patients who meet one or more of the following criteria *clarification needed*: (1) don't have the minimal functional capacity to participate effectively in an outpatient program; (2) have medical conditions that require more

	intensive oversight; (3) are receiving large amounts of medications necessitating medication weaning or detoxification; or (4) have complex medical or psychological diagnosis that benefit from more intensive observation and/or additional consultation during the rehabilitation process. (Keel, 1998) (Kool, 2005) (Buchner, 2006) (Kool, 2007) As with outpatient pain rehabilitation programs, the most effective programs combine intensive, daily biopsychosocial rehabilitation with a functional restoration approach. If a primary focus is drug treatment, the initial evaluation should attempt to identify the most appropriate treatment plan (a drug treatment /detoxification approach vs. a multidisciplinary/interdisciplinary treatment program). See Chronic pain programs, opioids; Functional restoration programs. Also, see MTUS Opioids Treatment Guidelines" for recommendations on the use of multidisciplinary pain programs related to opioids.
Chronic pain programs, early intervention	Recommended, based on identification of patients that may benefit from early intervention via a multidisciplinary approach, as indicated below. The likelihood of return to work diminishes significantly after approximately 3 months of sick leave. It is now being suggested that there is a place for interdisciplinary programs at a stage in treatment prior to the development of permanent disability, and this may be at a period of no later than 3 to 6 months after a disabling injury. (Robinson, 2004) (Gatchel, 2003) (Jordan, 1998) Some early intervention programs have been referred to as "secondary treatment," and differ from the more traditional, palliative care pain programs by not only the earlier onset of treatment, but by treatment intensity and level of medical supervision. (Mayer, 2003) *Recommendations for identification of patients that may benefit from early intervention via a multidisciplinary approach:* (a) The patient's response to treatment falls outside of the established norms for their specific diagnosis without a physical explanation to explain symptom severity. (b) The patient exhibits excessive pain behavior and/or complaints compared to that expected from the diagnosis. (c) Risk factors are identified with available screening tools or there is a previous medical history of delayed recovery. (d) The patient is not a candidate where surgery or other treatments would clearly be warranted. (e) Inadequate employer support or evidence of work organizational factors limiting return to work without interventions. (f) Evidence of psychosocial barriers that make return to work unlikely. (g) Loss of employment or evidence of partial disability involving ability to perform only "part-time" work or work with "light-duty" restrictions for greater than 4 months. (Mayer, 2003) (Gatchel, 2003) For general information see Chronic pain programs.
Chronic pain programs, intensity	Recommend adjustment according to patient variables, as indicated below. Research is ongoing as to what treatments are most necessary as part of interdisciplinary treatment for patients with subacute and chronic pain, and how intense such delivery of care should be. The more traditional models of interdisciplinary pain management often provide what has been referred to as tertiary care; a more intensive, and often, more

	palliative treatment for chronic pain. Research as to the intensity of treatment that is required for earlier intervention remains ongoing ("secondary intervention" see Chronic pain programs, early intervention). Several examples show the difference in results based on intensity of treatment that occur based, in part, on variables such as gender, age, prognosis, diagnosis, and duration of pain. A recent study showed that for men with low back pain that had been "sick-listed" for an average of 3 months, there was no difference between extensive multidisciplinary treatment and usual care in terms of return to work. Significantly better results were found for men who received a "light treatment program" compared to usual care, and these results remained significant at 12, 18 and 24 months. (Skouen, 2002) On the other hand, an extensive program has been shown to be the most effective treatment modality for patients considered to be in categories of poor health, and poor prognosis who were "sick-listed" for the same period, although the effect tapers after one to two years. (Haldorsen, 2002) For general information see Chronic pain programs.
Chronic pain programs, opioids	Recommend assessing the effects of interdisciplinary pain programs on patients who remain on opioids throughout treatment, and to determine whether opioid use should be a screening factor for admission to or continuation in a program. Also see MTUS Opioids Treatment Guidelinesfor recommendations on the use of multidisciplinary pain programs. The limited research that is available indicates that daily opioid use, in low doses, does not decrease effectiveness of chronic pain programs, although outcomes may be less optimal for patients who continue to use opioids. (Dersh, 2008) Current research indicates that simultaneous dependency/addiction programs with pain programs are a viable option. Some patients will require treatment of addictive disease before pain management can be effectively addressed. Patients with opioid dependence may require additional, long-term follow-up after the rehabilitation program. Criteria for this follow-up are still under research. ***Programs that include detoxification as part of their protocol*** *PRIDE Program:* In 2008 the PRIDE program (Progressive Rehabilitation Institute of Dallas for Ergonomics) (Dersh 2008) evaluated the role of post-injury opioid-dependence disorder (ODD) to assess if prescription opioid dependence (assessed at the beginning of rehabilitation) affected treatment outcome in patients with chronic disabling occupational spinal disorders. All patients with opioid dependence exhibited a lack of improvement or worsening in psychological well-being and social and vocational functioning despite the clinician's best attempts at pain control. As noted, patients were required to taper off of all opioids early in treatment. Patients who had the following identified during initial treatment were referred to a facility psychiatrist (who had board certification in addiction): 1) evidence of use of high-dose/potency opioids or multiple opioids; 2) patients with a known history of current or lifetime substance-use disorders; 3) patients with known or easily apparent psychiatric disturbance; 4) patients that did not progress well in their detoxification under care of the attending physician. A diagnosis of substance dependence was made, in part, using the structured clinical interview for