BEST PRACTICES IN THE ASSESSMENT AND MANAGEMENT OF CHRONIC PAIN

A CME/CE-CERTIFIED RESOURCE COMPRODENDIUM

Date of Release: June 5, 2012
Date of Credit Expiration: June 4, 2013

Jointly sponsored by Albert Einstein College of Medicine of Yeshiva University, Montefiore Learning Network, and Asante Communications, LLC.

This activity is supported by an independent educational grant from Lilly USA, LLC. For more information regarding Lilly grant funding visit www.lillygrantoffice.com.
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Preamble

Activity Goal
The goal of this educational activity is to enhance the diagnostic and clinical skills of healthcare professionals who treat patients with chronic pain.

Intended Audiences
The educational activity is intended for physicians, physician assistants, nurse practitioners, and other healthcare professionals interested in chronic pain management.

Statement of Need and Learner’s Gap
At least 116 million adult Americans suffer from chronic pain, which remains the leading reason for patient visits to primary care.¹ The socioeconomic burden is significant. Medical expenses and lost productivity associated with chronic pain conditions reach an estimated $635 billion in the United States each year.¹ Effectively treating this patient population is particularly challenging because pain can be caused and maintained by numerous biologic factors—including genetic predisposition, tissue injury, aging, joint and disc degeneration, previous medical interventions, and such chronic diseases as osteoarthritis, diabetes, and cancer, to name a few.²,³ Further, the patient-specific pain experience is not solely dependent on painful sensory inputs; emotional status, psychological responses, and social circumstances markedly contribute to pain levels and impairments in physical, affective, cognitive, and occupational functioning.²,³ The heterogeneity of chronic pain—in etiologies, clinical presentations, and outcomes—confounds diagnosis and treatment. Mechanism-based pain classification, comprehensive patient histories, and targeted approaches to physical exams can help clinicians better identify the causes of chronic pain and select appropriate treatments.⁴ For example, whenever possible, clinicians should match the analgesic mechanisms of action of prescribed medications with the likely underlying pain etiology. Further, combinations of pharmacologic, nonpharmacologic, and interventional treatment approaches may be required for some individuals.⁵,⁶ Primary care clinicians are on the frontline of chronic pain management. Long-term relationships with patients facilitate comprehensive and longitudinal assessment of evolving symptoms, allowing for tailored therapeutic regimens that help patients reach functional goals.

References


**Learning Objectives**
At the completion of this initiative, participants should be better prepared to:

- Discuss the neurobiology of pain processing and how dysfunction in nociceptive pathways contributes to various chronic pain disorders
- Comprehensively assess patients with chronic pain based on biopsychosocial factors that shape the severity, chronicity, and adverse functional consequences of the painful conditions
- Evaluate the clinical profiles of available pharmacologic agents based in part on mechanisms of action, analgesic effectiveness, and treatment-related risks and side effects
- Employ mechanism-based pharmacotherapy for patients with common chronic pain syndromes, such as low back pain, osteoarthritis, fibromyalgia, and painful neuropathic disorders
- Combine pharmacologic and nonpharmacologic therapies for patients with chronic pain with the objectives of reducing pain, meeting predefined functional goals, and minimizing potential treatment-related harm

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Steven Jay Feld of Albert Einstein College of Medicine, or a member of his household, owns securities in Bioheart, Inc.; Chelsea Therapeutics, Inc.; and Pharmacopeia, Inc.

Jim Kappler, PhD, of Asante Communications, LLC, helped draft the chapters and has no relevant financial relationships to disclose.

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Chapter 1: Neurobiology of Chronic Pain: Implications for Mechanism-Based Diagnosis and Treatment

Perry G. Fine, MD

Key Points

Central mechanisms, including remodeling of neural circuits and affective processing, markedly contribute to pain and related disability in a range of chronic conditions.

Clinicians should attempt to identify likely pain mechanisms to guide treatment decisions.

Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage.1 Normally, pain is adaptive, signaling harm or potential harm to the body and helping promote healing and recovery. In chronic pain, however, the nociceptive system becomes persistently activated and pain continues past the usual period of healing. Moreover, the perception of chronic pain does not result solely from tissue damage or injury. Instead, each patient’s subjective experience reflects nociceptive signaling filtered through genetic background, prior experiences, current psychological status, and sociocultural influences.2,3 The result is a complex disease requiring multidimensional assessment and, for some patients, multimodal treatment.
Nociception

The nociceptive signaling pathway can be simplified into 4 discrete stages. During transduction, noxious thermal, mechanical, or chemical stimuli are converted into neural activity by receptors on sensory neurons called nociceptors. The resulting action potentials are transmitted to the dorsal horn of the spinal cord and up central ascending tracts, entering the brain through the thalamus. In the brain, incoming activity is routed to regions involved in sensation, emotion, stress, and motor responses, resulting in the overall perception of pain and associated behavioral responses. Neural circuits in the peripheral and central nervous systems also modulate nociceptive signals by inhibiting or facilitating the activity as it moves to the brain. For example, descending tracts from the brain and spinal cord function to dampen incoming nociceptive signaling in the spinal cord via such neurotransmitters as serotonin, norepinephrine, and endogenous opioid peptides.

Central and Peripheral Sensitization

Scientific and clinical studies during the last few decades have uncovered remarkable plasticity in the nociceptive system. Regardless of initial pain type, ongoing nociceptive activity—for example, in response to persistent inflammation, nerve injury, or inherently overactive central neural pathways—can increase ascending signaling to the brain, reduce descending inhibitory signals, and precipitate spontaneous and widespread pain. The result is hypersensitivity in both peripheral and central circuits long after an injury has healed or even in the absence of an injury.

Peripheral nervous system pathologies in chronic pain states may include preferential loss of nociceptor free nerve endings, which can cause excessive branching and increased depolarization in remaining nociceptors, and enhanced spontaneous activity in neural subpopulations of the dorsal horn. Ongoing activity in nociceptors also increases the strength of synaptic connections all along the nociceptive neuraxis, resulting in hyperalgesia or increased sensitivity to normally noxious inputs. Additionally, pain thresholds are lowered, and allostynia—pain in response to normally non-noxious stimuli—develops as low-intensity stimuli normally carried by other peripheral nerve fibers begin to activate central pain-related circuits. The precise mechanisms and timing of these transitions—collectively termed peripheral and central sensitization—vary considerably among individuals based on physiologic,
genetic, psychological, and sociocultural factors. However, once established, these sensitization processes can perpetuate pain and related disability independent of peripheral inputs.

**Mechanism-Based Pain Classification**

Identifying the likely etiologies contributing to a patient’s chronic pain can help shape assessment strategies and aid in the selection of targeted treatment approaches. One proposed approach involves classifying pain into 4 categories: nociceptive, inflammatory, neuropathic, and dysfunctional. As discussed above, nociceptive pain is an adaptive and protective sensation that is essential to detect and minimize contact with potentially noxious stimuli. Inflammatory pain is caused by injury or infection, activation of the immune system, and the resulting production of proinflammatory mediators near or at some distance from the site of damage to promote healing. In the acute setting, inflammatory pain may be adaptive, although ongoing inflammation—as occurs in patients with rheumatoid arthritis, for example—should be addressed to minimize tissue damage and reduce the potential for neuroplastic remodeling in nociceptive circuits. The other two pain types are consistently maladaptive and do not serve any protective function. Neuropathic pain results from a lesion or damage to the peripheral or central nervous system; respective examples include painful diabetic neuropathy and pain associated with multiple sclerosis. Dysfunctional pain conditions, such as irritable bowel syndrome and fibromyalgia, are thought to result from heightened sensitivity in the nociceptive system even in the absence of a detectable stimulus. Biochemical and neuroimaging studies suggest that these patients may generally have increased activity in sensory neural pathways and dysfunctional descending inhibition from supraspinal centers, leading to widespread pain.

When selecting analgesic treatment for various chronic conditions, clinicians should consider which of these different mechanisms may be contributing to the patient’s pain. For instance, a patient with predominantly inflammatory pain is likely to benefit from different treatment approaches than a patient whose pain is due to a peripheral neuropathy. It is also important to note that a patient’s overall pain may result from more than one of these mechanisms. While widespread pain and central sensitization are hallmarks of dysfunctional pain syndromes, recent evidence suggests that if left untreated, relatively localized pain conditions may develop into larger areas of pain and increased pain referral.
In one study, investigators observed reduced pressure pain thresholds in patients with painful knee osteoarthritis both at the affected joint and in remote areas not directly affected by osteoarthritis, suggesting a central allodynic state of pain processing.\textsuperscript{11} Other studies have yielded similar results in patients with chronic low back pain.\textsuperscript{12,13}

One group recently identified factors in the clinical presentation of chronic low back pain with or without leg symptoms that can be used to categorize pain as nociceptive (inflammatory), peripheral neuropathic, or dysfunctional (central sensitization).\textsuperscript{14} A review of 38 potential criteria found 14 symptoms that were positively or negatively linked to one of the 3 pain types (see associated video and resource).\textsuperscript{14} For example, the strongest predictive factor for peripheral neuropathic pain was a dermatomal or cutaneous distribution of pain referral, whereas dysfunctional pain was strongly associated with diffuse, nonanatomic areas of pain and tenderness in response to palpation.\textsuperscript{14} When incorporated into the patient history and physical exam, these criteria provide a quick approach to identifying which pain mechanism(s) may be contributing to the chronic pain condition.

**Conclusions**

Chronic pain is a disease state characterized by overactive nociceptors, increased peripheral receptive fields, enhanced synaptic efficacy, and dysfunctional inhibitory modulation of nociceptive signaling.\textsuperscript{8} As pain persists, affective and emotional processing also begins to play prominent roles in the pain experience.\textsuperscript{2} Because many factors interact to produce chronic pain and associated functional deficits, effective management requires comprehensive assessment and patient-specific treatment plans that rationally intervene at appropriate points, potentially including initial nociceptive input, central amplificatory processes, and emotional and behavioral responses to pain.

**References**

Chapter 1: Neurobiology of Chronic Pain: 
Implications for Mechanism-Based Diagnosis and Treatment


Mechanism-Based Diagnoses of Musculoskeletal Pain

**Nociceptive**
- Is pain usually intermittent and sharp with movement or mechanical provocation? Is it described as a constant dull ache or throb at rest?
- Is pain localized to the area of injury or dysfunction with or without some somatic referral?
- Is the pain clearly and proportionately mechanical and anatomic relative to aggravating and easing factors?
- Is there an absence of pain variously described as burning, shooting, or electric-shock like?
- Is pain not associated with other dysesthesias (eg, “pins and needles,” heaviness)?
- Is there an absence of night pain or disturbed sleep?
- Does the patient report an absence of antalgic (ie, pain relieving) postures or movement patterns?

**Peripheral Neuropathic**
- Is there a history of nerve injury, pathology, or mechanical compromise?
- Is the pain referred in a dermatomal or cutaneous distribution?
- Is there pain or symptom provocation with mechanical or movement tests that move, load, or compress neural tissue?

**Central Sensitization**
- Is the pain disproportionate to the nature and extent of injury or pathology?
- Is there a disproportionate, nonmechanical, unpredictable pattern of pain provocation in response to multiple/nonspecific aggravating/easing factors?
- Is there a strong association with maladaptive psychosocial factors (eg, negative emotions, poor self-efficacy, maladaptive beliefs, and pain behaviors)?
- Are there diffuse or nonanatomic areas of pain or tenderness on palpation?

Chapter 2: Comprehensive Chronic Pain Assessment

April Hazard Vallerand, PhD, RN, FAAN

**Key Points**

Comprehensive assessment of chronic pain requires examining biopsychosocial contributions and consequences.

Clinicians should attempt to identify likely pathologic mechanisms and specific diagnoses contributing to the pain symptoms.

Based on an in-depth clinical interview, medical history, physical exam, and appropriate ancillary testing, clinicians and patients should identify realistic functional goals to serve as measures of the effectiveness of any prescribed treatment regimen.

A complex, multidimensional experience, chronic pain is a biopsychosocial condition associated with significant disability, affective symptoms, cognitive impairment, deconditioning, and disturbed sleep. Comprehensive patient evaluations should seek to identify functional deficits and underlying pain mechanisms. Clinicians can then attempt
to obtain specific diagnoses, set attainable treatment goals, and select appropriate therapies. A structured framework for chronic pain assessment includes in-depth clinical interview, medical history, physical exam, and appropriate ancillary testing. This chapter describes some general clinical approaches to chronic pain assessment, while the following chapter provides more detailed recommendations for specific chronic pain conditions, including low back pain, osteoarthritis, neuropathic pain, and fibromyalgia.

**Patient History: Characterizing Pain and Patient Function**

Each patient’s pain profile should be detailed. Importantly, as a subjective sensory and emotional experience, there is no diagnostic test for pain, and clinicians must rely on patient self-report. Specific inquiries should be made into pain characteristics, including location, referral pattern, intensity, qualities, temporal profile, and palliating and precipitating factors. Unidimensional scales, such as the numeric rating scale (NRS), are commonly used and easy to administer to most patients. Yet studies have suggested that these tools are only modestly accurate in identifying patients with clinically important pain and do not by themselves improve chronic pain treatment in primary care. This outcome reflects, in part, the multifactorial and dynamic nature of chronic pain, the effects of which are unlikely to be captured with single parameter tools. Multidimensional scales evaluate various aspects of pain and its consequences. The Brief Pain Inventory (BPI), for example, asks patients to rate current and recent levels of pain, responses to treatment, and effects on function and quality of life. Available in a short form as well, the BPI has been translated and validated in multiple languages and can be completed by the patient in the waiting room or prior to the office visit (see associated tool and video). Other multidimensional options include the McGill Pain Questionnaire and the recently developed PEG, a 3-item scale that helps primary care clinicians assess Pain intensity, Enjoyment of life, and General activity interference.

Nonetheless, self-report tools may be challenging for some older adults or cognitively impaired individuals. Nonverbal cues, such as guarding, grimacing, and restricted movement, should be noted in patients who have trouble with verbal self-report. When individuals are unable to articulate their pain experience, clinicians can consider obtaining information from other sources, such as family members or daily caregivers. Changes
in interpersonal interactions, activity patterns, and mental status should also be noted; in some cases, an analgesic trial may be required to determine whether behaviors are a result of pain. One expert panel recently recommended 2 tools—the Pain Assessment in Advanced Dementia (PAINAD) and Pain Assessment Checklist for Seniors with Limited Ability to Communicate (PACSLAC)—for assessing pain in nonverbal residents of nursing homes. These questionnaires were selected by the panel as the most scientifically and clinically relevant, applicable to everyday practice, and aligned with regulatory guidelines.

In addition to obtaining self-reports of pain, inquiring about baseline function can produce insights into areas of significant disability, employment issues, and problems with activities of daily living. Moreover, identifying pain-related functional impairment helps the clinician and patient set realistic treatment goals and provides objective outcome parameters to evaluate disease progression and treatment responses at follow-up visits. Although functional assessment tools are available, they are not commonly used in primary care, in part, because they have been developed for specific disease states, and functional deficits and treatment goals are inherently patient-specific. On the other hand, clinicians can simply ask the patient “What does this pain keep you from doing?”; these types of questions can help identify important and meaningful activities for the patient, give the patient a sense that the clinician truly cares about him or her as an individual, and reveal pain management goals to work towards.

The clinical interview and medical history should focus on conditions that may cause or exacerbate pain. Psychopathologies, such as depression or anxiety, may affect pain perception. Other psychosocial factors that increase the risk of chronic pain, disability, and poor treatment outcomes include job dissatisfaction, reduced physical activity, and a history of emotional, sexual, or physical abuse. Acute life stressors will also markedly affect current pain levels and functional deficits. Chronic pain is commonly comorbid with sleep disturbances, necessitating at least brief evaluations of sleep patterns. Of note, the relationship between sleep and pain is complex and bidirectional: disturbed sleep can decrease pain thresholds, whereas pain at night reduces sleep quantity and quality. Clinicians should record previous responses to analgesic interventions and inquire about personal and family history of substance abuse, particularly if opioids or other medications with high potential for abuse are being considered for the treatment plan.
Physical Exam and Ancillary Testing

Although chronic pain in many ways represents a disease unto itself, it is also usually a symptom of one or more underlying conditions. Whenever possible, the etiology and pathophysiology of the pain condition should be identified and classified according to presumed mechanisms. Identifying characteristics of the pain presentation that are associated with inflammation, damage to the nervous system, or central sensitization may allow targeted treatment of pain generators and/or inform appropriate selection of symptomatic therapy. This process can be facilitated by the physical exam, including musculoskeletal and neurologic evaluations.

In general, the physical exam should be based on the patient’s history and used to guide subsequent imaging and laboratory testing. Pain behaviors, range of motion, and other movements can help identify pain sources and support reports of pain severity. General palpation can identify widespread pain or areas of particular tenderness. Specific provocative maneuvers are used to stress ligaments and joints in an effort to reproduce pain symptoms. The neurologic exam is performed to identify sensory or motor abnormalities. Clinicians can use warm and cold objects to test for temperature sensation, a brush or cotton swab to detect allodynia, and a pin for hyperalgesia.

Combinations of laboratory and diagnostic testing can also provide clues to underlying pathologies. More detailed neurologic assessments may include quantitative sensory testing, which is recommended for diagnosis of small fiber neuropathies and allows more acute identification of sensory dysfunction. Imaging evaluations are used to confirm or rule out causes of chronic pain, particularly when fracture or neoplasm is suspected. In many cases, however, conventional radiography, computerized tomography, or magnetic resonance imaging will not identify causes of chronic pain. Therefore, imaging and other tests may be reserved only when the outcomes are going to affect treatment choices. For example, the American College of Physicians and the American Pain Society guidelines recommend diagnostic imaging and testing for patients with chronic low back pain only when severe or progressive neurologic deficits are present or when serious underlying conditions are suspected.
Conclusions

Multidimensional assessment of patients with chronic pain is crucial to identifying treatment goals and selecting appropriate therapy. Patient self-report is essential. A thorough history and physical exam are cornerstones of an accurate differential diagnosis. For many individuals, a full understanding of the pain problem will occur over multiple visits, providing familiarity with the patient, revealing relative contributions from identified injuries and central nervous system dysfunction, and determining responses to palliative approaches.

References

Chapter 2: Comprehensive Chronic Pain Assessment

Brief Pain Inventory—Short Form

First Name ___________________________ Date ______________
Last Name ___________________________ Time ______________

1. Throughout our lives, most of us have had pain from time to time (such as minor headaches, sprains, and toothaches). Have you had pain other than these everyday kinds of pain today?
   ❑ Yes ❑ No

2. On the diagram, shade in the areas where you feel pain. Put an X on the area that hurts the most.

3. Please rate your pain by circling the one number that best describes your pain at its worst in the last 24 hours.

   No pain | 0 1 2 3 4 5 6 7 8 9 10 | Worst pain imaginable

4. Please rate your pain by circling the one number that best describes your pain at its least in the last 24 hours.

   No pain | 0 1 2 3 4 5 6 7 8 9 10 | Worst pain imaginable

5. Please rate your pain by circling the one number that best describes your pain on the average.

   No pain | 0 1 2 3 4 5 6 7 8 9 10 | Worst pain imaginable

6. Please rate your pain by circling the one number that tells how much pain you have right now.

   No pain | 0 1 2 3 4 5 6 7 8 9 10 | Worst pain imaginable

Cont’d ➤
**Brief Pain Inventory—Short Form (cont’d)**

7. What treatments or medications are you receiving for your pain?

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8. In the last 24 hours, how much relief have pain treatments or medications provided? Please circle the one percentage that shows how much **relief** you have received.

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9. Circle the one number that describes how, during the past 24 hours, pain has interfered with your:

**A. General activity**

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**B. Mood**

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**C. Walking ability**

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**D. Normal work** (includes both work outside the home and housework)

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**E. Relations with other people**

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**F. Sleep**

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**G. Enjoyment of life**

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Chapter 3: Assessment Protocols for Chronic Pain Conditions: Neuropathic Pain, Low Back Pain, Osteoarthritis, and Fibromyalgia

Susan Cochella, MD, MPH

**Key Points**

Assessing a patient with chronic pain requires clinical evaluations of all underlying diagnoses and potential contributory inflammatory and neurologic mechanisms.

Assessment protocols should reflect the growing body of evidence supporting central nervous system dysfunction in a range of chronic pain disorders.

Patients should be screened for nonphysical types of pain, such as emotional and spiritual pain, which, if present, should be addressed with appropriate measures.

Structured, evidence-based assessment protocols for common chronic pain disorders can improve diagnoses and provide efficiencies in busy clinical practices. Identifying potential causes, classifying pain based on inferred underlying pathophysiologic mechanisms, and addressing medical emergencies are critical.\(^1\,2\) The role of dysfunction in the central nervous system should be considered in all patients with chronic pain.\(^1\) Aberrant

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**Accompanying Video Vignette**

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central nociceptive processing is characterized by hyperexcitability in pain-related neural pathways and altered endogenous pain inhibition, manifesting clinically as lower pain thresholds, multifocal or widespread pain, and, at times, other somatic symptoms, including cognitive problems and sleep disturbances. All chronic pain patients should also be assessed for emotional and spiritual pain, as physical pain can be more difficult to treat if such needs are not addressed. This chapter provides brief clinical recommendations that can help guide comprehensive assessments of some of the most common chronically painful conditions.

Neuropathic Pain

Neuropathic pain is a manifestation of a lesion or disease affecting the nervous system and is generally characterized by both negative and positive phenomena. Certain underlying disorders, such as diabetes mellitus or multiple sclerosis, indicate neuropathic pain. Moreover, features of the clinical presentation can support various neuropathic pain diagnoses, including stimulus-independent pain and reports of “burning” or “electric shock–like” pain. During the clinical exam, special attention should be paid to stimulus-evoked hypersensitivities, including allodynia (eg, pain in response to light touch) and hyperalgesia (eg, increased pain in response to pinprick). Affected areas are focal in mononeuropathies, but typically distal and symmetric in polyneuropathies. Concomitant loss of sensation is also common; small-fiber neuropathies are linked to abnormal thermal, pain, and pressure sensations, whereas neuropathies involving damage to small and large fibers produce these symptoms plus additional problems with muscle strength, reflexes, and perception of vibration. Some conditions—for example, complex regional pain syndrome—are associated with autonomic nervous system symptoms, such as abnormal sweating or skin temperature. Of note, patients with such disorders as trigeminal or postherpetic neuralgia may appear normal on clinical exam.

Although assessment of neuropathic pain lacks validated diagnostic criteria, self-report questionnaires and ancillary testing can be helpful. For instance, the Leeds Assessment of Neuropathic Symptoms and Signs is used to distinguish neuropathic and nociceptive pain by combining sensory descriptors with the results of a bedside exam of sensory dysfunction. A targeted neurologic exam is appropriate for most primary care patients. Evaluations that include quantitative sensory testing (QST) can help in select cases by identifying neuropathies, defining affected
areas, and characterizing disease progression and treatment responses over time. A recently proposed bedside QST protocol recommends a neuropathic screening tool, pain diagrams, and sensory mapping to delineate areas affected by deficits, allodynia, and hyperalgesia in response to vibratory, thermal, tactile, and painful stimuli. Importantly, however, results from neuropathic screening tools and ancillary testing should only be interpreted in the context of a more global clinical assessment. Currently, neuropathic screening tools do little to guide therapeutic decisions for neuropathic pain.

**Low Back Pain**

Most primary care patients with low back pain do not have an identifiable causal disease or spinal problem. Evaluations should include a medical and psychosocial history, physical exam, and neurologic testing to determine the presence and degree of any nerve root involvement. The review of systems should specifically probe for and document red flags that indicate or rule out a serious underlying pathology, such as malignancy, infection, osteoporosis, or cauda equina syndrome. Clinical practice guidelines suggest a diagnostic triage process that classifies patients into one of the following categories: nonspecific low back pain, low back pain associated with a specific spinal cause, or a radicular syndrome. The exam should also include assessment of psychosocial risk factors, such as depression and poor coping skills, that increase the likelihood of significant disability. If opioids are being considered, a history of childhood abuse or substance abuse should be collected; positive histories increase the patient’s risk of medication abuse and addiction.

The physical and neurologic exams of patients with low back pain are based on a working knowledge of lumbosacral spine anatomy (see associated resource and video). Deformities suggestive of degenerative disease or osteoporotic fractures should be noted. Neurologic evaluations include testing of motor strength and sensory perception by examining myotomes, dermatomes, and tendon reflexes associated with specific spinal nerve roots; L4, L5, and S1 deserve particular attention as those most likely to be affected by a herniated lumbar disc. All tests should be performed on both sides of the patient to compare responses. Bilateral findings consistent with sacral root compromise, together with urinary retention or fecal incontinence, suggest cauda equina syndrome, which requires immediate surgical attention. Clinicians can also use physical maneuvers, such as the straight leg raise or seated slump test
to reproduce the patient’s pain symptoms and identify pain generators by stressing specific anatomic structures. These maneuvers can help sort pain that requires surgical attention from that which does not.

Although imaging is commonly ordered for patients reporting back pain, studies have shown that radiographic abnormalities correlate poorly with symptoms. Additionally, disc degeneration and annular disruption are common in asymptomatic subjects. Therefore, evidence-based practice guidelines appropriately recommend judicious use of diagnostic imaging in low back pain, reserving these tests for patients with severe or progressive neurologic deficits or when red flags for a serious, underlying condition are present. When imaging is used to determine whether more invasive treatment is appropriate (eg, injection therapy), magnetic resonance imaging is preferred owing to better visualization of soft tissue and other anatomic structures.

**Osteoarthritis**

Osteoarthritis is a degenerative disorder of articular cartilage. Diagnosis is primarily based on patient history and physical exam. Osteoarthritis pain classically involves stiffness with movement after periods of rest, brief episodes of morning stiffness, and stiffness with cold or weather change. Other potential causes should also be considered. The differential diagnosis includes rheumatoid and other autoimmune arthritis, bursitis, referred pain from the spine, connective tissue disease, and gout, among other conditions. Neuromuscular and biomechanical factors, such as aberrant joint alignment after sports injury, increase the risk of osteoarthritis and should be identified in the patient history.

Pain symptoms in osteoarthritis are complex and influenced by local factors (eg, joint inflammation) and sensitization of central pain-processing pathways. In fact, pain and functional impairment associated with osteoarthritis do not correlate strongly with disease severity on radiographs; some individuals with obvious cartilage loss report no pain, whereas others with joint pain show only limited tissue degeneration. Therefore, plain radiographs are usually not required unless pain continues at night or is not activity-related, or if the patient is referred for joint replacement surgery. Blood tests can help rule out rheumatoid arthritis and other forms of inflammatory arthritis in select cases. Like any chronically painful disorder, contributory psychosocial factors (eg, depression, weight gain) should be identified and addressed in the treatment plan.
Fibromyalgia

Fibromyalgia is the prototypical widespread chronic pain disorder. It is thought to result from aberrant amplification of pain-related signals alongside reduced endogenous pain inhibition in the central nervous system. Fibromyalgia often occurs without any identifiable tissue injury. Clinical manifestations of fibromyalgia include widespread pain, muscle fatigue, disturbed sleep, cognitive deficits, and affective symptoms. Although the disorder is no longer considered solely a diagnosis of exclusion, conditions that mimic fibromyalgia should be considered in patients presenting with widespread pain. Clinicians can perform detailed joint and neurologic exams, as well as a complete blood count and tests that determine the erythrocyte sedimentation rate, levels of muscle enzymes, and liver and thyroid function to rule out endocrine and autoimmune disorders.

The 1990 American College of Rheumatology (ACR) classification criteria define fibromyalgia as a history of widespread pain for at least 3 months and the presence of pain on palpation of at least 11 of 18 tender points. These criteria, however, were developed for research purposes and some clinicians have argued that they do not account for the prominence of symptoms other than pain. Moreover, the tender point criterion has been criticized for inter-examiner differences and associations with the patient’s current state of distress rather than being a reliable indicator of persistent widespread tenderness. To address these issues, the ACR developed a new diagnostic methodology for fibromyalgia that relies on 2 clinical measures: a widespread pain index (WPI) and a composite symptom severity (SS) scale. The WPI (scored 0-19) assesses pain in 19 discrete body regions, whereas the SS scale (scored 0-12) examines fatigue, waking unrefreshed, cognitive issues, and additional somatic symptoms. Patients meet the diagnostic criteria for fibromyalgia if the following 3 conditions are met: i) WPI value 7 or greater and SS scale score 5 or greater or WPI value between 3 and 6, and SS scale score 9 or greater; ii) symptoms have been present at a similar level for at least 3 months; and iii) the patient does not have a disorder that would otherwise explain the pain.

References


Physical Exam Overview for Chronic Low Back Pain

Pain Behaviors
- Observe patients for guarding, grimacing, and bracing behaviors indicative of movements that cause pain
- Observe for depressed affect, compliance issues, or lack of response to treatments, indicative of emotional or spiritual needs

Static Stance, Posture, and Gait
- Assess balance, base of support, arm swing, and trunk and shoulder rotation
- Note circumduction of either leg when walking

Neurologic Function
- Identify areas of pain radiation
- Identify areas of sensory loss using pinprick or a cold stimulus
- Areas of aberrant sensation can reveal pathology at specific spinal nerve roots

Motor Strength Testing Against Resistance
- Knee extension (predominantly L4)
- Dorsiflexion of great toe and foot (predominantly L4 and L5)
- Plantar flexion of great toe and foot (predominantly S1)
- Responses are graded from 0 (no activity) to 5 (normal)

Muscle Stretch Reflexes
- Knee jerk (L4)
- Ankle jerk (S1)
- Responses are scored from 0 (no response) to 4+ (hyperactive with clonus); 2+ indicates a normal stretch reflex

Dural Tension Testing for Nerve Root Compression
- **Straight leg raise test**: Patient is supine with both legs straight. Examiner lifts straight the affected leg by flexing at the hip. Test is positive when radiculopathy is reproduced with leg raised to a hip angle of at least 30°.
Physical Exam Overview for Chronic Low Back Pain (cont’d)

- **Seated slump test:** In a seated position with the spine flexed forward, the knee of the affected leg is passively extended and the foot dorsiflexed. A positive test occurs when radiculopathy is reproduced.

**Provocative Physical Tests for Sacroiliac Joint Pain**

- **Sacroiliac compression test:** Patient lies supine with hips and knees straight. Examiner applies outward pressure on the medial side of both anterior superior iliac spine positions, creating compression at the sacroiliac joint. A positive test involves reproduction of pain and suggests sacroiliac pathology.

- **Sacroiliac distraction test:** Patient lies with affected side up and hips and knees flexed. Examiner applies downward compression to the affected anterior iliac spine, opening the sacroiliac joint. A positive test involves reproduction of pain and suggests sacroiliac pathology.

- **Patrick’s sign (flexion abduction external rotation or “Faber” test):** Patient lies supine and places foot of affected side on the opposite thigh, bending affected leg at the knee and externally rotating the hip. Examiner applies downward pressure to the affected knee and unaffected anterior superior iliac spine. A positive test can occur in 3 ways: reproduction of pain at the sacroiliac joint suggests sacroiliac pathology, inguinal or groin pain suggests hip pathology, and inability to press the affected knee to the level of the leg suggests iliopsoas tightness.

- **Gaenslen test:** Patient lies supine with affected side on edge of exam table. The unaffected leg is bent at the hip and knee, maximally flexing the leg against the abdomen and chest. Examiner brings the affected hip into hyperextension applying light downward pressure to the knee. A positive test involves reproduction of pain and suggests sacroiliac pathology.

**Myofascial Trigger Points**

- Palpate target areas for tender nodules in skeletal muscle fibers
Chapter 4: Mechanism-Based Pharmacotherapy for Chronic Pain

Susan Cochella, MD, MPH

Key Points

Selection of pharmacotherapy for chronic pain should reflect each patient’s likely underlying biochemical and neural pain mechanisms, patient characteristics including functional status and comorbidities, and the relative benefit-risk profiles of available agents.

Chronic pain conditions are often associated with central nervous system dysfunction, which can be targeted with analgesic agents that reduce neural excitability or augment descending inhibition of pain-related signals.

Patients with chronic pain should receive pharmacotherapy alongside appropriate nonpharmacologic treatment options, including interventions for emotional and spiritual pain.

Choosing a pharmacologic approach for a patient with chronic pain requires individualized treatment based on numerous patient-specific factors and benefit-risk profiles of therapeutic options. These considerations are not reflected in contemporary analgesic stepladders.

Accompanying Video Vignette

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which should not be used as the sole guide for therapeutic decisions. Medication regimens can and should be constructed to address identified and presumed molecular and cellular processes contributing to the pain experience. In particular, the duration, severity, and presentation of pain symptoms—in the context of the underlying diagnosis or source of pain—provide important clues about medications that are more likely to be effective. For instance, pain due to peripheral inflammation can be addressed with nonsteroidal anti-inflammatory drugs (NSAIDs), which decrease the production and release of prostaglandins and thereby reduce inflammatory pain. On the other hand, NSAIDs are not effective for neuropathic or other centrally mediated pain. Long-standing and/or severe pain complaints—for example, from untreated acute pain following serious injury, persistent inflammation, or damage or dysfunction in the nervous system—can result in neuroplastic reorganization of both peripheral and central circuits that remains long after an injury has healed, or develops even in the absence of identifiable tissue damage.

Even seemingly localized conditions, such as osteoarthritis, can cause widespread tenderness. For these patients, it is important to consider approaches that not only address localized disease mechanisms and joint inflammation but also drugs that target the central allodynic state of pain processing. Options include agents that are thought to dampen pain-related neural activity, such as \( \alpha_2 \delta \) ligands, and drugs that enhance signals in central inhibitory pathways that descend from the brain to the spinal cord, including tricyclic antidepressants (TCAs) and serotonin-norepinephrine reuptake inhibitors (SNRIs).

Patient-specific parameters identify certain analgesics as more or less appropriate. A patient’s prior response to a medication or drug class provides insights into expected outcomes. The comprehensive medical status of the patient should be considered as well. When comorbid conditions exist, opportunities to accomplish 2 treatment goals with one medication should be considered. Examples include considering an SNRI for patients with comorbid depression or gabapentin for patients with comorbid insomnia. The presence of liver or kidney dysfunction may affect drug metabolism or clearance, impacting medication selection and dosing. Full medication regimens must be considered to avoid potentially additive side effects and interactions at the site of drug metabolism, such as the cytochrome P450 system. Patient preferences and the likelihood of treatment adherence should also be addressed. In general, prevention of pain can be more effectively accomplished than addressing pain after it has manifest; therefore, many experts suggest that most patients with
persistent, functionally impairing pain should be treated with scheduled dosing rather than medication dosed in an as-needed fashion. A careful patient history of addiction and any childhood abuse should be explored as these determinants increase the patient’s risk of eventual abuse of prescription medications. Patients with high abuse potential should be followed more intensively if potentially addictive treatment options are used, such as opioids or tramadol.

Older patients require a unique and careful approach, as they are at higher risk for adverse drug reactions. Age-associated differences in gastrointestinal function may prolong the effects of drugs or enhance bowel issues related to opioids. Renal filtration rates are usually lower, slowing drug excretion. Anticholinergic side effects of such drug classes as the TCAs may increase confusion, constipation, and incontinence. Thus, older patients are often more sensitive to the analgesic and nonanalgesic effects of medications, requiring initiation at low doses, careful upward titration, and frequent reassessment. In addition, the American Geriatric Society has stated that, owing to gastrointestinal, cardiac, and renal risks, NSAIDs should only be used rarely and with extreme caution in highly selected older individuals with persistent pain. While opioids may be appropriate for carefully screened older patients, recent data suggest an increased risk of falls and fractures in older patients taking scheduled opioids, although a direct causative relationship has not been established.

Therapy for pain should be aimed at achieving preplanned and agreed upon goals, with assessment of various parameters, including function and pain, against these goals at each visit. While pain scores are part of this process, they are not the only outcome metric, as pain is multidimensional and subjective. To capture the full effects of chronic pain treatment, goals should focus on restoring—or in some cases, maintaining—patient function. Achieving an appropriate balance between pain relief, functional abilities, and tolerability requires appropriate patient expectations. This step can be accomplished by advising patients up front that medications are only one component of effective treatment plans, and that complete eradication of pain is rare.

Nonphysical types of pain must also be identified and managed while developing initial treatment plans and during ongoing care. Patients with chronic physical pain often suffer from emotional or spiritual types of pain as well. These other components of pain can exacerbate the subjective experience of physical pain and respond differently to various treatments. For example, emotional pain therapies include counseling,
behavioral changes, and antidepressant or mood stabilizing medications. Spiritual interventions—unique to the patient and his or her religion or culture—can help address those needs. When emotional and spiritual pain is appropriately addressed, a patient’s physical pain often becomes more manageable.

As stated above, initial treatments should be selected using a personalized risk-benefit approach, with consideration to types of pain and medication side effects. The accompanying Table highlights recent clinical insights that can inform this process. Patients across the chronic pain spectrum will benefit differentially from each medication option. Of note, chronic pain results from multiple neurobiologic mechanisms interceding at peripheral and central levels of pain-processing neural pathways; therefore, multimodal treatment approaches may be needed to optimally address the complex pathophysiology associated with chronic pain. In fact, well-designed studies in chronic neuropathic pain have shown that combining agents with different mechanisms of action and nonoverlapping adverse event profiles can provide additive or synergistic analgesia with reduced side effects compared with monotherapy.\textsuperscript{13,14}

While clinical practice guidelines can aid drug selection, they are influenced by the opinions and composition of the specific guideline group, leading to biased interpretations of the evidence.\textsuperscript{15-18} In addition, data consolidation and publication processes often preclude guidelines from incorporating the most recently published studies. Therefore, clinicians need to stay abreast of recommendations from various sources, integrating recent data not included in the guidelines. They must also remain alert to the medical and psychosocial needs of their patient populations. Such an approach will facilitate appropriate prescription of analgesics that maximize pain relief, improve functional outcomes, ensure patient safety, and minimize treatment-related side effects.

References


Chapter 4: Mechanism-Based Pharmacotherapy for Chronic Pain


10. Rolita L. Unintended consequences; increased prescription of narcotic analgesics for OA in the elderly is associated with increased falls and fractures in the post-Vioxx era. Paper presented at: ACR/ARHP Annual Scientific Meeting 2011; Chicago, Illinois.


### Pharmacologic Agents for Chronic Pain

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<td>Acetaminophen</td>
<td>• FDA advisory committee has recommended decreasing maximum daily dose from 4000 mg to 3250 mg owing to liver toxicity risk</td>
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<tr>
<td>NSAIDs (eg, ibuprofen, celecoxib)</td>
<td>• Inhibit prostaglandin production and release and reduce inflammation and associated pain</td>
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<td>• Risk of gastrointestinal and renal complications (COX-2–selective agents reduce gastrointestinal risks)</td>
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<td>• Risk of serious cardiovascular events</td>
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<td>• In older adults with persistent pain, use rarely and with extreme caution in highly selected individuals</td>
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<td>TCAs (eg, amitriptyline, imipramine)</td>
<td>• Inhibit serotonin and norepinephrine reuptake in CNS, augmenting descending inhibition of nociceptive pathways</td>
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<tr>
<td></td>
<td>• Side effects include dry mouth, urinary retention, constipation, sedation, sexual dysfunction, arrhythmias, postural hypotension, and heart block</td>
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<tr>
<td>SNRIs (eg, duloxetine, milnacipran)</td>
<td>• Inhibit serotonin and norepinephrine reuptake in CNS, augmenting descending inhibition of nociceptive pathways</td>
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<td></td>
<td>• Superior tolerability profiles compared with TCAs</td>
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<td></td>
<td>• Side effects include nausea, dizziness, and restlessness</td>
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<tr>
<td>$\alpha_2\delta$ Ligands (eg, gabapentin, pregabalin)</td>
<td>• Bind to $\alpha_2\delta$ subunits of calcium channels to reduce release of pain-related neurotransmitters</td>
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<td>• Side effects include somnolence, dizziness, peripheral edema, and angioedema</td>
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<td>• Side effects include burning pain on contact with warm/hot water or in hot weather</td>
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<td>• Initial burning sensation may persist</td>
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<td>Lidocaine Patch</td>
<td>• Topical administration avoids systemic side effects</td>
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<td>• Side effects include skin irritation</td>
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<td>Opioids (eg, morphine, oxycodone)</td>
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<td>• Side effects include respiratory depression, nausea, constipation, somnolence, and pruritus</td>
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<td></td>
<td>• Abuse potential requires initial and ongoing assessment for misuse, abuse, and addiction</td>
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<tr>
<td>Tramadol, Tapentadol</td>
<td>• Activate opioid and nonopioid analgesic systems</td>
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<tr>
<td></td>
<td>• Opioid side effects</td>
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<tr>
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<td>• Abuse potential requires initial and ongoing assessment for misuse, abuse, and addiction</td>
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CNS, central nervous system; COX-2, cyclooxygenase-2; FDA, US Food and Drug Administration; NSAID, nonsteroidal anti-inflammatory drug; SNRI, serotonin-norepinephrine reuptake inhibitor; TCA, tricyclic antidepressant.

Chapter 5: Principles of Multidrug Therapy for Chronic Pain

April Hazard Vallerand, PhD, RN, FAAN

Key Points

Multidrug therapy for chronic pain can provide additive or synergistic analgesia with reduced side effects compared with monotherapy.

Multidrug regimens should be constructed using drugs with different mechanisms or sites of action, with slow dose titration of one analgesic agent at a time.

Chronic pain results from a complex set of disease processes and neuroplastic mechanisms. Not surprisingly, many patients will require combinations of pharmacologic, nonpharmacologic, and interventional approaches to optimize therapeutic outcomes. Indeed, advances in understanding the neuropathophysiologic underpinnings of various chronic pain types have led to the recommendation that, to the fullest extent possible, drug therapy should be matched to underlying mechanisms. Because chronic pain states often result from dysregulation at multiple sites along pain-related neural circuits, combining drugs with different mechanisms or sites of action may lead to improved pain control compared with monotherapy.

Accompanying Video Vignette

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**Rationale for Combining Analgesics**

Several fundamental processes in pain-related neural pathways can be targeted using pharmacologic agents. For instance, nonsteroidal anti-inflammatory drugs (NSAIDs), opioids, and topical agents, such as capsaicin, help interrupt the transduction of noxious stimuli into neural activity in nociceptors. Other medications, such as \( \alpha_2\delta \) ligands, are thought to impede the transmission of action potentials from the periphery to the spinal cord and brain. Modulatory processes in the peripheral and central nervous systems can be altered as well; for example, tricyclic antidepressants, serotonin-norepinephrine reuptake inhibitors (SNRIs), and opioid analgesics enhance signaling in descending tracts from the brain and spinal cord to dampen incoming nociceptive neural activity. Finally, pain is perceived at numerous cortical locations as a compilation of sensory and emotional cognitive processes, many of which are affected by certain centrally acting agents.

Combining medications with different sites or mechanisms of action has several theoretical benefits. As discussed throughout this compendium, multiple cellular processes contribute to amplified nociceptive signals in chronic pain states, providing numerous potential therapeutic targets. Multidrug regimens may achieve additive or synergistic analgesia, particularly if complementary pain mechanisms are addressed. Two medications may also result in a broader analgesic spectrum for mixed pain conditions—for example, an NSAID to reduce inflammatory pain and an SNRI to counteract central sensitization. Moreover, optimal pain relief may be achieved at lower doses of the individual agents than if the medications were prescribed alone; this approach can reduce the incidence and/or severity of adverse events if the side effects of the medications are not more additive than the analgesic effects.

Alternatively, although this chapter primarily discusses combinations of pain medications, some patients will benefit from pharmacologic approaches that specifically antagonize the side effects of the prescribed analgesic, improving tolerability at therapeutic doses.

**Evidence for Multidrug Therapy in Chronic Pain**

Multidrug therapy for pain was first applied clinically through fixed-dose combination formulations, such as acetaminophen and hydrocodone, and with intrathecally or epidurally delivered medications in the perioperative setting. Despite a vast amount of clinical anecdotal experience with multidrug therapy for various chronic pain conditions, most direct
comparisons of combination analgesic therapy with single-drug treatment have been conducted in patients with chronic neuropathic pain. The number of studies is small, yet several important observations have emerged. For example, combining gabapentin with morphine or nortriptyline in patients with painful diabetic neuropathy or postherpetic neuralgia resulted in greater pain relief than any of the individual agents. Patients treated with morphine and gabapentin also required lower doses of the opioid, leading to reduced incidence of constipation. Other studies have demonstrated the benefits of add-on therapy, during which a selected medication is added to an existing, stable treatment regimen. There is little and, at times, conflicting clinical trial data on combination therapy in chronic pain disorders other than neuropathic pain. Clearly, more studies are needed to establish a better evidence-based framework for combination drug therapy.

Clinical Considerations for Multidrug Therapy
Backonja and colleagues have proposed general principles that can help guide the prescription of medication combinations for chronic pain (see associated tool and video). Agents with a rigorous evidence base and defined mechanism of action should be prioritized for initial therapy, while considering the patient’s responses to previous drug treatments. If pain relief or functional outcomes from a single titrated medication are inadequate, the clinician should consider adding another analgesic agent with a different mechanism of action. When selecting a second analgesic medication, thought should be given to potential drug-drug interactions and the possibility of additive side effects if the adverse event profiles of the analgesics or other prescribed medications overlap. In general, only one drug should be initiated at a time. To allow the development of tolerance to adverse effects, dose titration should be performed slowly, especially in older patients who are generally more susceptible to adverse drug reactions. Pain relief, functional gains, and adverse events should be continually assessed and evaluated. Of note, clinically meaningful relief is often defined as a greater than 30% improvement or a reduction in pain intensity of 2 or more points on a 0 to 10 scale.

Conclusions
Scientific and clinical insights into chronic pain increasingly support multidrug pharmacotherapy for appropriately selected patients. Yet a number of unsolved issues remain. Most chronic pain conditions
and drug combinations have not been formally examined in well-designed clinical trials. Moreover, a number of practical issues have not been addressed, including optimal dosing schedules for specific combinations, appropriate therapy for symptoms comorbid with pain, and whether adherence suffers with multidrug regimens. Although current recommendations focus on sequential add-on therapy, some authors have suggested that concurrent titration of medications may help identify better dosing ratios. Finally, efforts should be undertaken to explore the potential functional benefits of combination treatment that includes nonpharmacologic modalities, such as physical rehabilitation and cognitive behavioral therapy. Nevertheless, given the multiple neurologic and molecular pathways that can contribute to various chronic pain conditions, some patients are unlikely to achieve adequate analgesia or meet functional goals with a single analgesic agent, even if it is titrated optimally. These individuals may benefit from combinations of analgesic agents selected and dose administered to maximize analgesia and ensure patient safety.

References


Ten Principles of Multidrug Therapy for Chronic Pain

1. **Prioritize** treatment approaches based on assessment and diagnosis of all medical and psychological morbidities as well as postulated pain-generating mechanisms.

2. **Review** doses, effects, and adverse events of past treatments, including over-the-counter and herbal/natural preparations, to help guide medication choices.

3. **Select** initial drugs with known mechanisms of action that have proved to be effective and safe for the identified chronic pain disorder(s).

4. **Dose** at low end of the recommended range and titrate slowly, particularly in older patients. Effective doses may be higher than those stated in the dosing guidelines.

5. **Combine** medications with differing mechanisms or sites of action.

6. **Use** one drug at a time and adjust dose by starting low, titrating slowly, and monitoring effects and adverse events until maximum benefit has occurred, an intolerable side effect has developed, or a usual therapeutic dose has been reached. Discuss choices and rationale with patients, allowing them some control over their care.

7. **Reassess** patient responses over time and continue to prescribe only those therapies that provide clinically meaningful relief (eg, >30% improvement or 2 points on 0-10 pain rating scale) and documented improvement in function.

8. **Monitor** for adverse effects and discontinue analgesics that prove intolerable at effective doses.

9. **Consider** changing to a drug with a similar mechanism of action if good efficacy is documented but side effects predominate with the current drug. If no similar drug is available, consider adding a medication to control side effects.

10. **Re-evaluate** all of these issues and any new potential drug-drug interactions when adding another drug.

Chapter 6: Multimodal Chronic Pain Management: Psychosocial Approaches
Patricia Bruckenthal, PhD, APRN-BC, ANP

Key Points

Treating chronic pain often requires approaches that address patient-specific emotional and behavioral responses to pain.

Patients should be encouraged to actively engage in their own management plans to improve coping skills and increase self-efficacy.

Cognitive-behavioral therapy (CBT) is an effective, evidence-based treatment modality for chronic pain management.

Chronic pain does not merely reflect overt tissue damage, and the correlation between the severity of physical pain and associated patient responses is moderate at best. In fact, both the perception and perpetuation of pain are dependent on complex interactions among behavioral, emotional, and cognitive factors. It is not surprising then that pharmacologic agents usually result in only 25% to 40% reductions in pain levels, even when disease and drug mechanisms are correctly matched. Moreover, patients who report significant analgesia from drug therapy or interventional procedures may not demonstrate...
accompanying improvements in physical or emotional functioning. Clearly, the multidimensional nature of chronic pain requires individualized care: some patients will benefit from single treatment approaches while others will need multimodal regimens.4

**Psychological and Behavioral Contributions to Chronic Pain**

A number of behavioral and psychosocial patient responses will affect pain levels and associated functional deficits. Patients with chronic pain may display maladaptive thought patterns, including magnification of pain, rumination about pain, and feelings of hopelessness or anxiety about how to manage pain.7 This type of catastrophizing can markedly contribute to perceptions of pain severity.7 Significant emotional distress (e.g., depression or anxiety) is common across various chronic pain populations, often contributing to reduced physical functioning and hindering treatment responses.8,9 For example, psychological stressors can induce activity in the hypothalamic–pituitary–adrenocortical hormone axis and autonomic neural circuits, which when persistently activated can increase fatigue, reduce muscle mass, and elevate pain levels.4,10 Patients may also engage in activity avoidance, either as a result of nociceptive signals or anticipatory fear of pain.11 Activity avoidance is strongly associated with pain-related disability, in part, via physical deconditioning and augmented pain-related neural signaling.12

Understanding how patients respond emotionally and behaviorally to chronic pain is critical to optimizing care. To complement the clinical interview and structure assessments, clinicians may want to employ tools that are specifically designed to examine cognitive, behavioral, and affective distress. For instance, the Pain Anxiety Symptom Scale Short Form 20 (PASS 20) is a 20-item questionnaire that uses 5-point scales to assess 4 domains related to pain anxiety: cognitive thoughts, pain-associated fear, physiologic arousal, and escape or avoidance behaviors (see accompanying tool and video).13 Scores on the PASS 20 can identify maladaptive thoughts or unhelpful behaviors. Patients can then be taught to specifically replace problematic responses with more positive coping approaches through CBT.

**Psychological and Behavioral Therapies**

In general, psychological and behavioral therapies have roots in the operant model of pain, in which dysfunctional learning processes play a
significant role in the development and maintenance of chronically painful conditions. In this model, pain behaviors and thought patterns are reinforced and learned as they are repeated. Chronic pain treatment based on the operant model does not necessarily focus on pathologic sources of nociceptive signaling, but rather seeks to identify and modify dysfunctional or irrational activities that either contribute to disability or prevent patients from appropriately adjusting to their condition. Concurrently, positive behaviors and thought processes are reinforced to improve coping skills, adaptation, and self-management. For example, instead of stopping all activities when pain levels begin to increase, patients can be instructed on appropriate pacing and distraction techniques to help them complete activities of daily living.

CBT is a well-studied and widely used psychosocial approach to reduce disability associated with chronic pain. This treatment approach attempts to modify maladaptive behaviors, emotions, and thoughts through a systematic, goal-oriented approach. Key components of CBT include education and coaching to teach patients about the roles of thoughts and emotions in maintaining stress and physical symptoms. Patients may be asked to record feelings and responses during situations that increase distress or limit function. Identified dysfunctional processes can be specifically targeted through stress management techniques, problem solving, goal setting, and activity pacing. CBT can be performed in a group setting to reduce costs and allow the practicing therapist to highlight successful examples of alternative thought and behavioral patterns from group members. Acceptance and commitment therapy (ACT), in contrast, encourages individuals to change their expectations from pain elimination to living as well as possible with chronic pain. In combination with usual care, both ACT and CBT have been shown to reduce pain interference, depression, and pain-related anxiety in individuals with chronic pain.

Many patients will look to clinicians to “solve” their pain problem with appropriate therapy, potentially failing to recognize the benefits of self-management. Motivational interviewing provides a framework to actively engage patients in their treatment as pain persists. The approach is built on expressing empathy, supporting self-efficacy, providing positive feedback, and handling resistance. To start, clinicians help patients realize that passivity and inactivity present significant risks to treatment outcomes. When the patient is ready to take an active role in the treatment plan, healthcare providers facilitate brief action planning to identify desired
behavioral changes, evaluate the patient’s confidence in the goals, and schedule follow-up visits. The patient is then encouraged to take steps toward those goals, with ongoing care fostering continued motivation and commitment. Overall, motivational interviewing is one method to encourage patients to assume control of their chronic pain conditions.

Other behavioral techniques for pain control include graded activity, in which the patient receives positive reinforcement for doing a predetermined quota of exercises. Exercise goals are adjusted if the patient fails to meet them, thereby removing negative reinforcement of pain behaviors. Progressive muscle relaxation training teaches patients to systemically tense and relax major muscle groups from head to toe, first in the clinical setting and then at home. These and other techniques can be combined with pharmacologic and interventional modalities to optimize reductions in pain and improvements in patient function and quality of life.

Conclusions

Elimination of chronic pain is unlikely for many patients, which can be demoralizing and emotionally distressing. Pain management experts and clinical practice guidelines have recommended that treatment should address the role of psychological and behavioral factors in pain-related disability. As a whole, published evidence supports psychosocial therapies to reduce pain and improve patient function. This chapter presents just a few of the numerous psychological and behavioral interventions for chronic pain. Like many treatment approaches, however, evidence of long-term benefits in chronic pain is limited, and one modality cannot be recommended over another in different patients or various pain conditions. Also, the outcomes for many of these techniques are highly dependent on the practitioner. Therefore, if referrals are required, clinicians should be familiar with the backgrounds and approaches of available healthcare providers to ensure best practice recommendations are being followed.

References


Chapter 6: Multimodal Chronic Pain Management:
Psychosocial Approaches


Chapter 6: Multimodal Chronic Pain Management: 
Psychosocial Approaches

**Pain Anxiety Symptom Scale Short Form 20**

*Please rate each item in terms of frequency, from 0 (Never) to 5 (Always).*

<table>
<thead>
<tr>
<th>Item Numbers</th>
<th>Never</th>
<th>Always</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. I can’t think straight when in pain</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>2. During painful episodes it is difficult for me to think of anything besides the pain</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>3. When I hurt I think about pain constantly</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>4. I find it hard to concentrate when I hurt</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>5. I worry when I am in pain</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>6. I go immediately to bed when I feel severe pain</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>7. I will stop any activity as soon as I sense pain coming on</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>8. As soon as pain comes on I take medication to reduce it</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>9. I avoid important activities when I hurt</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>10. I try to avoid activities that cause pain</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>11. I think that if my pain gets too severe it will never decrease</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>12. When I feel pain I am afraid that something terrible will happen</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>13. When I feel pain I think I might be seriously ill</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>14. Pain sensations are terrifying</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>15. When pain comes on strong I think that I might become paralyzed or more disabled</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>16. I begin trembling when engaged in activity that increases pain</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>17. Pain seems to cause my heart to pound or race</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>18. When I sense pain I feel dizzy or faint</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>19. Pain makes me nauseous</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
<tr>
<td>20. I find it difficult to calm my body down after periods of pain</td>
<td>0 1 2 3 4 5</td>
<td></td>
</tr>
</tbody>
</table>

**Total Score**

Cont’d
## Pain Anxiety Symptom Scale Short Form 20 (cont’d)

Means and standard deviations for the revised, shortened Pain Anxiety Symptoms Scale subscales and the total score (N=282 patients with chronic pain)

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cognitive (items 1 to 5)</td>
<td>12.27</td>
<td>6.73</td>
</tr>
<tr>
<td>Escape/avoidance (items 6 to 10)</td>
<td>12.84</td>
<td>6.11</td>
</tr>
<tr>
<td>Fear (items 11 to 15)</td>
<td>7.37</td>
<td>6.38</td>
</tr>
<tr>
<td>Physiological anxiety (items 16 to 20)</td>
<td>6.15</td>
<td>5.69</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>38.62</strong></td>
<td><strong>20.38</strong></td>
</tr>
</tbody>
</table>

Chapter 7: Multimodal Management: Interventional Therapies in Low Back Pain

Perry G. Fine, MD

Key Points

Patients with chronic low back pain that has failed to respond to conservative management can be referred for evidence-based diagnostic interventional procedures if the outcomes are likely to affect therapeutic decisions.

Evidence for epidural steroid injections is moderate for short-term benefits and poor for long-term benefits in patients with persistent radiculopathy due to herniated lumbar disc.

Shared decision making about surgical approaches requires a discussion of intensive interdisciplinary rehabilitation as a similarly effective option for nonspecific low back pain, or the moderate average benefits of surgery, which may decrease over time, for radiculopathy due to herniated lumbar disc or leg pain due to spinal stenosis.

Despite numerous pharmacologic options and non-interventional approaches to individualize care, managing persistent and disabling pain can be challenging. Interventional approaches may benefit some patients by

Accompanying Video Vignette

Perry G. Fine, MD

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specifically addressing anatomic structures or abnormalities that are serving as pain generators. These methods are most frequently used when the pain is thought to originate from a spinal source, thus this chapter will focus on select interventional techniques in patients with low back pain, including considerations for the referring primary care clinician. In general, interventional modalities can be used to diagnose and locate pain etiologies and as therapy for pain originating from soft tissues, facet or sacroiliac joints, spinal canal stenosis, or degenerated or herniated intervertebral discs.

Diagnostic Interventional Approaches

Diagnostic interventional techniques are used to evaluate potential anatomic sources of pain, which may allow better selection of patients for various therapeutic interventions. Blocking activity in nerves carrying nociceptive signals from a given structure theoretically should demonstrate whether the target is generating pain. Diagnostic blocks may be helpful in chronic spinal pain, especially in cases where patient self-report of symptoms, physical exam results, and imaging evaluations often fail to uncover a clear pathologic cause.

Appropriately referring a patient for diagnostic neural blockade is based on various assumptions. The underlying source of pain should be localized in a peripheral structure, and nociceptive neural activity should be relayed to the central nervous system via a single defined route. Moreover, local injections should dampen activity specifically in the targeted sensory nerves, and responses should reflect only the blocked afferent neural pathway. False-positive responses should also be considered and can be addressed by comparing responses to saline versus local anesthetics or by the durability of therapeutic effects over time.

Commonly used diagnostic approaches for low back pain include facet joint blocks, wherein intra-articular injections of anesthetic or injections targeting the medial branches of the dorsal rami are used to interrupt sensory signals. Patients can be referred for diagnostic transforaminal epidural injections to examine specific spinal nerve roots when the patient history, symptom profile, physical exam, and imaging tests do not clearly identify the irritated nerve root and the results are likely to affect treatment choices. Pain from the sacroiliac joint is difficult to definitively diagnose based on patient history, imaging, and provocative maneuvers; positive responses to intra-articular blocks with anesthetic may support a putative diagnosis of pain being caused by degenerative or arthritic changes in the sacroiliac joint.
Nevertheless, data from well-designed studies of diagnostic interventional techniques are variable and clinical practice guidelines do not always agree on the strength of the evidence. For example, a recent review from the American Pain Society (APS) concluded that provocative discography, which involves injecting radiographic contrast material into the nucleus of an intervertebral disc to reproduce the patient’s back pain, is not helpful for diagnosing discogenic sources of low back pain. Moreover, data supporting the accuracy of diagnostic sacroiliac joint, facet joint, or selective nerve root blocks are unreliable. In contrast, the American Society of Interventional Pain Physicians (ASIPP) identified stronger evidence for the utility of some of these diagnostic interventions—particularly, lumbar facet joint nerve blocks.

Therapeutic Interventional Approaches

Therapeutic interventional techniques for chronic back pain include injection therapy, neurolytic procedures, surgical interventions, and implantable devices (see associated resource). Injections targeting nerves with various anesthetics, anti-inflammatory agents, or neurotoxic compounds can inhibit pain-related neural signaling, reduce inflammation, and/or ablate neurons at the source of pain. The most common interventional procedure in the United States is epidural steroid injection, which involves placing corticosteroids into the epidural space between the dura and spine near specific spinal nerve roots (see associated video). Evidence for long-term efficacy of epidural steroid injections in low back pain is poor despite their widespread use. APS guidelines report that epidural steroid injections have short-term pain relief when used in patients with radiculopathy, but there is no evidence of benefit for spinal stenosis or low back pain without radiculopathy. Similarly, APS and ASIPP guidelines agree that the evidence for lumbar intra-articular facet joint injections—the second most commonly performed interventional technique—to treat presumed facet pain is poor, although more recent evidence supports lumbar facet joint nerve blocks for some patients.

Neurolytic procedures ablate specific neural structures using extreme cold (cryoablation), high temperature radiofrequency, or chemicals. For example, radiofrequency ablation involves precise destruction of nervous system elements using heat generated by a radiofrequency current. Heat generated by an electromagnetic field around the tip of an electrode forms a lesion in the neuronal tissue. Lesion size is dependent upon several factors, including electrode size, temperature, duration of
radiofrequency, local tissue characteristics, and configuration. A scar then forms over the course of the next 3 weeks. An increasingly utilized therapy, most evidence for this approach comes from studies that have targeted lumbar facet nerves. Before proceeding with radiofrequency ablation, diagnosis of facet joint pain should be established with 50% to 80% pain relief from controlled local anesthetic blocks.

Surgical interventions for chronic low back pain with or without radicular complaints are usually reserved for pain that persists despite conservative therapeutic management. Lumbar fusion, discectomy, and decompressive surgery are common surgical options for low back pain. Studies have shown that surgery for low back pain with radicular symptoms may have short-term benefits compared with conservative treatment, although improvements typically diminish over the long term. Importantly, the rate of spinal surgery has increased rapidly in the United States with surgical interventions too often performed in the absence of a clear indication. Significant complications from surgery are relatively common and include deep-vein thrombosis and neurologic injury. Further, pain often persists following spinal surgery and subsequent operations do not guarantee pain resolution. In fact, failed back surgery syndrome may occur in up to 40% of patients undergoing lumbar spinal surgery. Because of the high rate of complications associated with spinal surgery, it would be desirable to have well-identified patient characteristics highly predictive of therapeutic benefit. Unfortunately, patient characteristics of those most likely to benefit from surgery are not clear. Therefore, in the absence of an identified anatomic lesion that correlates strongly with the patient’s pain pattern or reproducible sensory/motor deficits, an extremely conservative approach to surgical intervention is recommended.

Implantable devices such as spinal cord stimulators and epidural or intrathecal drug delivery systems are used when pain persists despite systemic pharmacologic treatment, surgery, and injection procedures. Spinal cord stimulation involves placing epidural electrodes in proximity to the dorsal column of the spinal cord or at the corresponding dorsal root in patients with segmental pain. Published evidence supports a role for spinal cord stimulation in carefully selected individuals with chronic and disabling pain following surgery for a herniated disc and no evidence of a persistently compressed nerve root. Rates of complications following electrode placement are high, however.
Chapter 7: Multimodal Management: Interventional Therapies in Low Back Pain

Conclusions

All interventional approaches for chronic pain require a careful benefit-risk analysis. Clinicians should ensure that treatment options are stratified based on published evidence and that procedures are performed by appropriately trained, credentialed, and experienced healthcare providers under optimally controlled conditions. Additionally, because benefits can be modest, therapeutic decision making requires communication between the patient and all members of the healthcare team to balance treatment goals with potential benefits, harms, costs, and potential burdens of alternative therapies.²

References

## Select Nonsurgical Interventional Modalities for Chronic Back Pain

<table>
<thead>
<tr>
<th>Technique</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Injection therapy</strong></td>
<td></td>
</tr>
</tbody>
</table>
| Epidural corticosteroid injection | • Primary indication for radicular back pain, particularly subacute pain or acute exacerbations of chronic pain  
• Benefits are less likely for pain of long duration, nonradicular symptoms, or patients who smoke  
• Should be performed by experienced physician, under fluoroscopic guidance using contrast to detect vascular uptake and visualize spread of injectate  
• Evidence is better for transforaminal and interlaminar approaches to epidural space than for caudal approach |
| Facet joint injection     | • Facet joints are common sources of pain in cervical and lumbar regions  
• Depot corticosteroid administered using fluoroscopically guided joint injection may provide short-term relief for subset of patients |
| Sacroiliac joint injection | • Sacroiliac joint may result in low back and buttock pain  
• Injection of sacroiliac joint is technically challenging |
### Select Nonsurgical Interventional Modalities for Chronic Back Pain (cont’d)

<table>
<thead>
<tr>
<th>Technique</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Percutaneous radiofrequency neurotomy</strong></td>
<td>• Treatment for neck or back pain originating in facet joints</td>
</tr>
<tr>
<td></td>
<td>• Should be performed only after positive response to controlled local anesthetic blocks</td>
</tr>
<tr>
<td></td>
<td>• Performed by placing insulated needle electrode adjacent and parallel to nerves that supply joint</td>
</tr>
<tr>
<td></td>
<td>• Heat from radiofrequency current ablates nerve supply</td>
</tr>
<tr>
<td></td>
<td>• Multiple lesions must be performed at each nerve location</td>
</tr>
<tr>
<td></td>
<td>• Nerves regenerate over time so treatment outcomes are not permanent</td>
</tr>
<tr>
<td><strong>Spinal cord stimulation</strong></td>
<td>• Stimulating metal contacts placed in dorsal epidural space generate electrical field, alter the local neurochemical milieu in the dorsal horn, and suppress neuronal hyperexcitability</td>
</tr>
<tr>
<td></td>
<td>• Most commonly treated syndromes are characterized by chronic radicular pain, such as failed back surgery syndrome</td>
</tr>
</tbody>
</table>

Chapter 8: Chronic Pain and Comorbid Psychiatric or Sleep Disorders

Patricia Bruckenthal, PhD, APRN-BC, ANP

Key Points

Patients with chronic pain and comorbid psychiatric problems can benefit from a multidisciplinary approach to care, including appropriate medication regimens and psychological counseling.

Patients with chronic pain should undergo an informed assessment of sleep function, potentially including self-report questionnaires, sleep diaries or logs, patient/partner interviews, and/or referral to specialists for sleep studies.

Mental and physical health issues commonly coexist in patients with chronic pain.\(^1\) Medical and psychiatric pathologies are particularly intertwined in the biopsychosocial illness of chronic pain, which manifests as a subjective experience affected by and impinging on a range of physical, behavioral, emotional, and economic domains in the patient’s life.\(^2,3\) One study found that 87% of adults in the United States with chronic spinal pain report at least one other chronic pain, physical, or mental condition.\(^3\) Osteoarthritis, for example, is associated with high rates of depression, hypertension, dyslipidemia,

Accompanying Video Vignette

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and obesity, among other medical issues. New diagnostic criteria for fibromyalgia specifically address the common occurrence of disturbed sleep, affective symptoms, and other somatic problems. Further, the prevalence of chronic pain is high among older patients, who are more likely to have other chronic conditions. Primary care management of chronic pain alongside other diagnoses can be challenging, requiring assessment of multiple symptoms, prescription of multimodal regimens, consideration of potential drug-drug interactions, and evaluation of multifaceted treatment outcomes. Clearly, addressing these parameters comprehensively is difficult in the typical primary care practice. A longitudinal approach is often necessary, potentially including consultations and/or referrals to other healthcare providers.

Chronic Pain and Psychological Comorbidities

Not surprisingly, major depression, anxiety disorders, and other psychiatric conditions are common among patients with chronic pain. For many patients, chronic pain persists over years or decades, resulting in sustained levels of emotional distress. Moreover, pain serves as a chronic stressor, potentially creating a feedback loop via dysfunction in the hypothalamo-pituitary-adrenal axis. Reduced levels of serotonin may concurrently contribute to poor mood and increased pain. Chronic pain–related cognitive impairment, employment problems, financial concerns, and sexual response issues are just a few of the multitude of psychosocial stressors that may drive affective distress in patients.

Although diagnoses may be challenging, it is important to identify patients with comorbid psychiatric disorders to help prevent magnification of symptoms and select appropriate therapies. One study found that more than half of patients with chronic back pain treated at a pain clinic had at least 1 current psychiatric diagnosis. Patients may exhibit chronic adjustment disorder, characterized by depressed mood, anxiety, and/or disturbed conduct in response to a triggering event or stressor, lasting more than 6 months. Depressive states ranging from sadness to major affective disorders are often present. Risk factors for comorbid pain and depression include older age, female gender, and underemployment. Screening tools may be helpful in identifying depression in patients with chronic pain (see associated tool and video). Clinicians should also be aware of somatic symptoms, such as decreased appetite, lack of energy, and insomnia. The most reliable diagnostic indicators of depression in patients with chronic pain
include feelings of hopelessness, worthlessness, excessive guilt, loss of self-esteem, and suicidal thoughts. \(^7\) Anxiety manifests as multiple cognitive, behavioral, and physiologic responses to pain. Patients with pain-related anxiety may complain of many somatic symptoms, including restlessness, vigilance, and insomnia, which can overshadow psychological manifestations. \(^12\) Of note, the patient’s medication history should be considered to ensure that withdrawal from certain agents, such as benzodiazepines, does not explain psychological symptoms.

**Chronic Pain and Sleep Disorders**

Disturbed sleep and sleep disorders are highly prevalent in many chronic pain populations. \(^13\) Osteoarthritis, for instance, commonly contributes to poor sleep, particularly in older individuals \(^14\); one study revealed that more than 1 of 3 older adults with osteoarthritis suffered from clinically elevated levels of pain and insomnia. \(^14\) The relationship between sleep and pain is complex and bidirectional: studies have shown that sleep is a significant predictor of pain on the following day and pain levels can be used to predict the duration of subsequent sleep. \(^15\) Lack of sleep decreases levels of endogenous endorphins, which normally dampen pain signaling. \(^9\) Thus, disturbed sleep can lower pain thresholds. Additionally, certain medications, including opioid analgesics and benzodiazepines, can contribute to sleep-disordered breathing in a dose-dependent manner. \(^16\)

To assess sleep in patients with chronic pain, clinicians can consider patient questionnaires (eg, Epworth Sleepiness Scale), sleep/pain diaries or logs, and patient/partner interviews to characterize a patient’s sleep patterns. \(^17,18\) A full sleep history helps identify practices that contribute to insomnia or poor-quality sleep. \(^19\) For those suspected of having obstructive sleep apnea, referral for a sleep study with polysomnography is usually required. \(^18\)

**Treating Chronic Pain and Comorbid Conditions**

Whether comorbid symptoms are causes or effects, the success of chronic pain treatment depends on helping patients adapt to their conditions and engage in self-management to complement medical interventions. When pain and associated conditions are poorly managed, a deleterious cycle of worsening symptoms can result. Treatment regimens, therefore, should provide analgesia, address comorbid problems, and, above all, improve patient function. \(^7\) At
times, medications may be available with published evidence of efficacy in both presenting diagnoses (eg, tricyclic antidepressants or serotonin-norepinephrine reuptake inhibitors for depression and certain chronic pain conditions).20,21 Other patients with multiple medical and psychological conditions may require more than one medication, necessitating particular caution about overlapping side effect profiles or interactions at sites of drug metabolism. For example, commonly prescribed analgesics, such as acetaminophen, nonsteroidal anti-inflammatory drugs, and opioids, can interact with agents used to treat depression.22 Certain psychiatric pathologies also increase the risk of substance abuse, which should be examined when considering medications with abuse potential, such as opioids.22

Managing chronic pain and comorbid conditions certainly requires a multimodal approach, potentially including interdisciplinary consultations with specialists. Sleep may improve with simple adjustments to sleep habits, including maintaining a regular sleep/wake schedule, minimizing noise and light in bed, and avoiding caffeine, alcohol, and nicotine before bedtime.19 Psychosocial therapies for chronic pain—discussed in Chapter 6 of this compendium—can also be used to address sleep or affective symptoms. For example, goal setting, journal keeping, and self-monitoring can increase motivation and adaptation in patients with pain and depression. Of note, some evidence suggests that pain-oriented cognitive behavioral therapy does not always improve sleep problems and that targeted interventions may be required.23 Supportive psychotherapy, behavioral interventions, relaxation techniques, meditation, guided imagery, and hypnosis may also be useful.7

**Conclusions**

Comorbidity of chronic pain and other medical or psychiatric conditions is associated with significantly poorer self-rated health, lower functional status, and lower ratings of overall quality of care. A management approach that considers the whole patient is more likely to provide analgesia, improve psychosocial functioning and quality of life, and reduce the full range of presenting symptoms. Sleep and affective disorders are just a few of the common conditions that can contribute to and result from the complex biopsychosocial illness of chronic pain. Clinicians should look to multimodal therapy that combines pharmacologic and nonpharmacologic treatment, and encourages the patient to take an active role in the management plan.
References

Patient Health Questionnaire-9 (PHQ-9)

Over the **LAST 2 WEEKS**, how often have you been bothered by any of the following problems?

<table>
<thead>
<tr>
<th></th>
<th>Not at all</th>
<th>Several days</th>
<th>More than half the days</th>
<th>Nearly every day</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Little interest or pleasure in doing things</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>2. Feeling down, depressed, or hopeless</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>3. Trouble falling or staying asleep, or sleeping too much</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>4. Feeling tired or having little energy</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>5. Poor appetite or overeating</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>6. Feeling bad about yourself — or that you are a failure or have let yourself or your family down</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>7. Trouble concentrating on things, such as reading the newspaper or watching television</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>8. Moving or speaking so slowly that other people could have noticed; Or the opposite — being so fidgety or restless that you have been moving around a lot more than usual</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>9. Thoughts that you would be better off dead or of hurting yourself in some way</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
</tbody>
</table>

**FOR OFFICE CODING**

0 + + +

**TOTAL SCORE:** _____
Patient Health Questionnaire-9 (PHQ-9) (cont’d)

If you checked off ANY problems, how DIFFICULT have these problems made it for you to do your work, take care of things at home, or get along with other people?

- Not difficult at all
- Somewhat difficult
- Very difficult
- Extremely difficult

Total scores of 5, 10, 15, and 20 for the first 9 items represent threshold cutpoints for mild, moderate, moderately severe, and severe depression, respectively.

Online participation is available at PAINClinician.com/CPCompendium or PrimaryCAREClinician.com/CPCompendium