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Pain is one of the most common reasons for patients to seek medical attention and one of the most prevalent medical complaints in the US;[1,2] 9 out of 10 Americans aged 18 or older suffer pain at least once a month, and 42% experience it every day.[3] Consequently, physicians and other practitioners need education to assist in developing the skills needed to evaluate and manage patients with pain.

Educational Objectives

- Describe the pathophysiology of pain and the underlying mechanisms.
- Utilize the critical elements of the pain history, and evaluate characteristics of pain relevant to diagnosis and management.
- Recognize the distinction between acute and persistent pain.
- Describe tools used to assist in the assessment of pain to guide management options.

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This continuing medical education program is intended for primary care physicians and those physicians who care for patients experiencing pain.

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CME Needs Assessment

Pain is one of the most common reasons for patients to seek medical attention and one of the most prevalent medical complaints in the US.[1-3] According to the 2006 National Center for Health Statistics Report, one in 10 Americans overall and three in five of those 65 years or older said that they experienced pain that lasted a year or more.[2] More than one-quarter of adults said they had experienced low back pain, and 15% of adults experienced migraine or severe headache in the past three months. Between the periods 1988-94 and 1999-2002, the percentage of adults who took a narcotic drug to alleviate pain in the past month rose from 3.2 percent to 4.2 percent.

For the the millions of Americans who experience persistent pain, the impact on function and quality of life can be profound.[2-4] Pain is associated with high utilization of health care[4] and the societal costs related to treatment are compounded by the loss in productivity associated with persistent pain. Lost productive time from common pain conditions among workers costs an estimated $61.2 billion per year and most of this is related to reduced performance while at work.[5] The total annual cost of poorly controlled persistent pain most likely exceeds $100 billion.

Physicians and other clinicians need current, state-of-the-art education to assist them in developing the necessary skills to evaluate and manage patients with persistent pain. This CME program reviews assessment and management of persistent pain syndromes that are frequently seen in primary care.

1. Watkins EA, Wollan PC, Melton LJ 3rd, Yawn BP. A population in pain: report from the Olmsted County health study. *Pain Med.* 2008;9(2):166-74.

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4. Von Korff M, Lin EH, Fenton JJ, Saunders K. Frequency and priority of pain patients' health care use. *Clin J Pain.* 2007 Jun;23(5):400-8.

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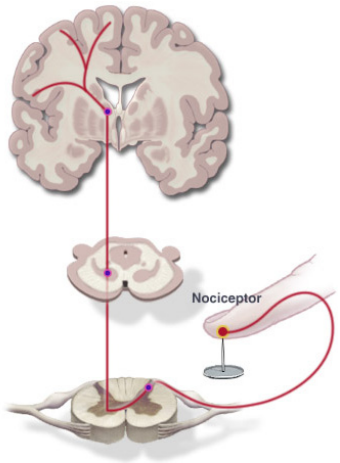
Introduction: What is Pain?

The International Association for the Study of Pain (IASP) defines pain as "an unpleasant sensory and emotional experience which we primarily associate with tissue damage or describe in terms of such damage, or both." This definition recognizes that pain is a perception and not a sensation. One influential model described pain in terms of three hierarchical levels: a sensory-discriminative component (*e.g.*, location, intensity, quality), a motivational–affective component (*e.g.*, depression, anxiety), and a cognitive-evaluative component (*e.g.*, thoughts concerning the cause and significance of the pain).[6]

Three Hierachical Levels of Pain

Sensory-Discriminative Component *location, intensity, quality*
Motivation-Affective Component *depression, anxiety*
Cognitive-Evaluation Component *thoughts concerning the cause and significance of the pain*



There is an important implication of both the IASP definition and the hierarchical model of pain: As a perception, pain may or may not correlate with an identifiable source of injury. The activity in the body's "nociceptive" system, which senses noxious stimuli and generates a physiological and behavioral response, can be initiated by injury and sustained by neuroplastic changes even after healing; activity in this system can occur in the absence of any discrete injury but in association with a recognizable disease. In some cases, pain can develop and be unrelated to any identifiable physical process. In all cases, the reality that pain is a perception indicates the potential for profound influence of psychological and emotional factors, cognitions, and varied external events.

There is another important implication of the concept of pain as perception: It is almost always best to believe that the patient is experiencing what is being reported. Because there is no objective indicator for pain, experts agree that the best clinical approach in most circumstances is to assume that the patient is reporting a true experience, even in the absence of a clear explanation. Importantly, accepting a patient's complaint of pain as valid does not require clinical identification of a physical cause, or demand the initiation of a specific treatment. Almost always, it is a sound foundation for assessment and an important beginning in developing an effective physician-patient dialogue. The risk that rare cases of malingering or factitious disorder may lead the credulous physician to initial error is more than balanced by the benefits associated with a stance of compassionate acceptance and concern.

Pathophysiology of Pain

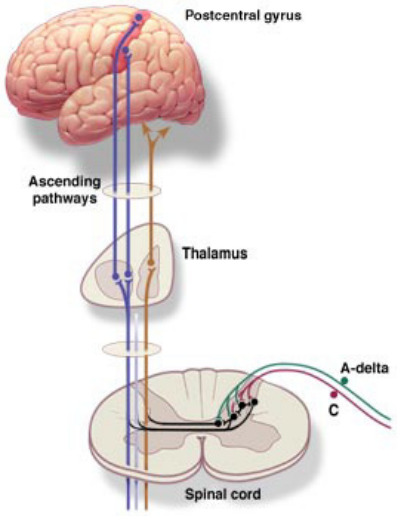
Enormous strides have been made in understanding the neurophysiology and neurochemistry of the systems that transmit and modulate information about noxious events.[7,8] Much also is known about acute inflammation, which commonly drives these neural processes. In contrast, relatively little is known about the pathophysiology underlying most persistent pain syndromes. Nonetheless, it is now widely accepted that persistent pain may be sustained by different types of mechanisms and experts agree that clinical characteristics can be used to broadly divide pain syndromes into nociceptive, neuropathic, psychogenic, mixed, or idiopathic. Although this classification is clearly an oversimplification, it has been found useful in assessment and therapeutic decision making.

Nociceptive Pain and Its Mechanisms

Clinically, pain can be labeled "nociceptive" if it is inferred that the pain is due to ongoing activation of the nociceptive system by tissue injury. Although neuroplastic changes (such as those underlying tissue sensitization) are clearly involved, nociceptive pain is presumed to occur as a result of the normal activation of the sensory system by noxious stimuli, a process that involves transduction, transmission, modulation and perception.

Tissue injury activates primary afferent neurons called nociceptors, which are small diameter afferent neurons (with A-delta and C-fibers) that respond to noxious stimuli and are found in skin, muscle, joints, and some visceral tissues.[7] These fibers have specific receptors that may be responsible for noxious mechanical, chemical or thermal stimuli. One class, called transient receptor potential (TRP) receptors, has been undergoing intensive investigation in the hope of ultimately yielding new therapies for pain.[9] The TRPV1 receptor, for example, has been found to be the specific site for reaction to capsaicin, a compound that activates C-fiber nociceptors. Presumably, nociceptive processes linked to noxious events involving somatic or visceral structures begin with activation of these specific receptors, which leads to transduction, the process by which exposure to a sufficient stimulus produces depolarization of the peripheral nerve.

Nociceptive primary afferent neurons are varied. Most are “silent”, active only when suprathreshold stimuli impinge. Some are specific to one type of stimulus, such as mechanical or thermal, but most are polymodal. The number and size of the receptive fields served by each fiber may be small or large, respectively. The meaning of this variability in terms of physiology or disease is not yet known, and research linking different types of nociceptors to disease states, or potential therapeutic targets, is still rudimentary.



Depolarization of the primary afferent involves a complex neurochemistry, in which substances produced by tissues, inflammatory cells and the neuron itself influence transduction. The role of prostaglandins, bradykinin, protons, nerve growth factor, and other compounds provide opportunities for the development of new analgesic drugs.

Once depolarization occurs, transmission of information proceeds proximally along the axon to the spinal cord and then on to higher centers. Complex systems that modulate this input occur at all levels of the neuraxis and are best characterized in the spinal cord. The neuroanatomy, neurophysiology and neurochemistry of these processes are very complex.[7,8,10] Transmission across the first central synapse may be influenced by activity in the primary afferent itself and modulatory neural pathways that originate segmentally or supraspinally; further modulation results from processes initated by glial cells.[7,8,11] The neurochemistry of these processes involves an extraordinary array of compounds, including endorphins, neurokinins, prostaglandins, biogenic amines, GABA, neurotensin, cannabinoids, purines, and many others.

The endorphinergic pain modulatory pathways are characterized by multiple endogenous ligands and different types of opioid receptors: mu, delta, and kappa. Endorphins are present in the periphery, on nerve endings, immune-related cells and other tissues, and are widely distributed in the central nervous system (CNS). They are involved in many neuroregulatory processes apart from pain control, including the stress response and motor control systems. Opioid drugs mimic the action of endogenous opioid ligands. Most of the drugs used for pain are full mu receptor agonists.

Other pain modulating systems, such as those that use monoamines (serotonin, norepinephrine and dopamine), histamine, acetylcholine, cannabinoids, growth factors and other compounds, are targets for nontraditional analgesics, such as specific antidepressants and anticonvulsants. It is likely that entirely novel analgesic compounds will become commercially available in the future as drug development programs target these systems.

Nociceptive pain can be acute (short-lived, remitting) or persistent (long-lived, chronic), and may primarily involve injury to somatic or visceral tissues. Pain that is inferred to be related to ongoing activation of nociceptors that innervate somatic structures, such as bone, joint, muscle and connective tissues, is termed “somatic pain”. This pain is recognized by identification of a lesion and characterisitics that typically include a well localized site and an experience described as aching, squeezing, stabbing, or throbbing. Arthritis and metastatic bone pain are examples of somatic pain. Pain arising from stimulation of afferent receptors in the viscera is referred to as visceral pain. Visceral pain caused by obstruction of hollow viscus is poorly localized and is often described as cramping and gnawing, with a daily pattern of varying intensity. When organ capsules or other structures such as myocardium, are involved, however, the pain usually is well localized and described as sharp, stabbing or throbbing, descriptors similar to those associated with somatic pain.

Nociceptive pain of any type can be referred and some referral patterns are clinically relevant. For example, injury to the hip joint may be referred to the knee and bile duct blockage may produce pain near the right shoulder blade.

Nociceptive pain may involve acute or chronic inflammation. The physiology of inflammation is complex. In addition to an immune component, retrograde release of substances from C polymodal nociceptors also may be involved. This “neurogenic inflammation” involves the release from nerve endings of compounds such as substance P, serotonin, histamine, acetylcholine, and bradykinin. These substances activate and sensitize other nociceptors. Prostaglandins produced by injured tissues also may enhance the nociceptive response to inflammation by lowering the threshold to noxious stimulation.

Neuropathic Pain and Its Mechanisms

Neuropathic pain is the label applied to pain syndromes inferred to result from direct injury or dysfunction of the peripheral or central nervous system. These changes may be caused by injury to either neural or non-neural tissues. Although neuropathic pain may be strongly influenced by ongoing tissue injury, or other stimuli that activate the sensory system, there is an assumption that the fundamental mechanisms sustaining the pain have become independent of any ongoing tissue injury.[12]

Neuropathic pain has varied characteristics. It may mimic the quality of somatic pain, but also is frequently described in terms that warrant the descriptor "dysesthetic:" an uncomfortable, unfamiliar sensation such as burning, shock-like or tingling. Neuropathic pain syndromes may be associated with referred pain, allodynia (pain induced by non-noxious stimuli, *e.g.* light touch), hyperalgesia (increased response to a noxious stimuli), or hyperpathia (exaggerated pain responses following a stimulus, often with aftersensation and intense emotional reaction).

Although again representing a gross oversimplication of very complex processes, it may be valuable to subclassify neuropathic pain syndromes based on additional inferences of the primary location of the sustaining mechanisms.[13] Some neuropathic pain syndromes are presumed to involve a predominating peripheral generator (*e.g.*, compressive or entrapment neuropathies, plexopathies, radiculopathies and polyneuropathies). Other syndromes appear to depend on processes that predominantly reside in the spinal cord, brain or both (*e.g.*, pain due to spinal cord injury or post-stroke pain). The clinical relevance of this distinction primarily resides in decisions about invasive peripheral interventions. If there is a relatively high level of certainty that the pain is related to a peripheral process, then an intervention to ameliorate this (*e.g.*, release of entrapment, or injection or resection of a neuroma) should be considered; if there is high certainty that the generator is central, further peripheral intervention should be avoided.

Some of the neurophysiologic and neuroanatomic changes that may occur in peripherally-generated neuropathic pain are understood.[12,14] Injury to a peripheral nerve axon can result in abnormal nerve morphology. The damaged axon may grow multiple nerve sprouts, some of which form neuromas. These nerve sprouts, including those forming neuromas, can generate spontaneous activity, which peaks in intensity several weeks after injury. These areas of increased sensitivity are associated with a change in sodium receptor concentration, and other molecular processes, and also can occur at sites of demyelination or nerve fiber injury not associated with the severing of axons. Unlike normal nerve, these injured regions are more sensitive to physical stimuli, which is clinically associated with tenderness and the appearance of Tinel's sign (*i.e.*, pain or tingling when the area over a nerve is tapped). After a period of time, atypical connections may develop between nerve sprouts or demyelinated axons in the region of the nerve damage, permitting "cross-talk" between somatic or sympathetic efferent nerves and nociceptors. Dorsal root fibers may also sprout following injury to peripheral nerves.

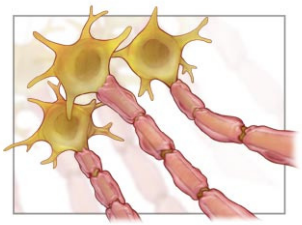


figure 1: Neural Axon

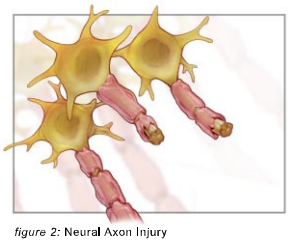


figure 2: Neural Axon Injury

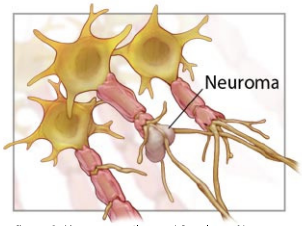


figure 3: Nerve sprouting and forming a Neuroma

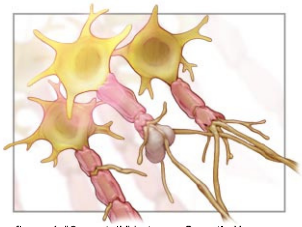


figure 4: "Cross-talk" between Somatic Nerves

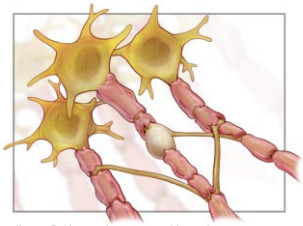


figure 5: Neural Axon Injury Healed

Other changes occur in peripheral nerve that are related to pain and yet poorly characterized. Anterograde and retrograde transport of compounds may shift and messages that are received in cell bodies may turn on specific genes. More proximally, there are identifiable trans-synaptic changes. Some of these alterations in morphology and function result in peripheral sensitization, which may be related to a lower threshold for signaling or an expansion in receptive fields.

In contrast to the still rudimentary understanding of the mechanisms of peripherally-generated neuropathic pain, there is almost no information about the processes that induce or sustain centrally-generated pain syndromes. Function neuro-imaging has demonstrated the extraordinary neuroplasticity of the brain in the setting of a neuropathic pain, such as phantom pain, but the mechanisms responsible are unknown.[15]

Psychological and "Idiopathic" Pain Mechanisms

There is an exceedingly complex relationship between the psyche and pain perception.[16] In some patients, the experience of persistent pain appears to induce disturbances in mood (reactive depression or anxiety), impaired coping (often with catastrophization), and other processes, which in turn, appear to worsen pain and pain-related distress. Other patients have premorbid or comorbid psychosocial concerns or psychiatric disorders that are best understood as evolving in parallel to the pain. These disturbances also can contribute to the pain experience and driver pain-related distress. Patients with personality disorders, substance use disorders, or mood disorders often are best served by primary treatment for the psychiatric problem at the same time that pain-related interventions are offered. This array of premorbid, comorbid and reactive psychosocial disturbances is individual, complex and may occur in a shifting mix of primary and secondary concerns.

This complexity highlights the importance of psychosocial and psychiatric evaluation as a fundamental aspect of the pain assessment. All patient with persistent pain and all patients with acute pain that has been challenging to control should be evaluated for mood, status of coping and adaptation, family and social support, and a range of psychiatric disorders that may influence the experience of pain or pose targets for therapy.

On occasion, the psychological evaluation yields evidence that the pain itself is predominantly sustained by psychological factors. This phenomenon is known generically as "psychogenic" pain and is subject to the specific diagnoses codified under the Somatoform Disorders in the Diagnostic and Statistical Manual of the American Psychiatric Association.[17] The evidence for a somatoform disorder must be more than the mere lack of an identifiable physical etiology for the pain. It is very important that patients who have acute or persistent pain without a known physical source not be inappropriately labeled. This may lead to inadequate assessment in the future and therapeutic decisions that are inappropriately skewed; unfortunately, in many quarters, it also leads to stigmatization of the patient and the potential for greater suffering on this basis. When reasonable inferences about the sustaining pathophysiology of a pain syndrome cannot be made, and there is no positive evidence that the etiology is psychiatric, it is best to label the pain as "idiopathic."

Assessing the Patient in Pain

Initial Pain Assessment

Pain management depends on a comprehensive assessment. This is especially true for the patient with persistent pain. Pain assessment should be ongoing (occurring at regular intervals), individualized, and documented so that all involved in the patient's care have a clear understanding of the pain problem. As a result of the pain assessment, the clinician should understand the nature of the pain in terms of its etiology, pathophysiology and syndrome; its impact on many domains of life; and relevant premorbid conditions and comorbidities that will influence treatment decisions. This understanding requires detailed questions about the pain characteristics, an assessment of the impact of the pain in multiple domains, and an evaluation of related concerns and comorbidities. Based on this information, the findings on a physical examination and review of records and existing laboratory and imaging data, a working diagnosis can be developed that includes an understanding of the pain's etiology, pathophysiology and syndrome. From this formulation, a plan of care can be developed that may include the need for additional evaluation and an initial set of therapies to address the pain and other concerns.

This process of assessment can be straightforward and brief in the setting of acute pain related to trauma or surgery. It increases in complexity and the time required as the pain becomes persistent, fails to respond to conventional therapy, or is observed to be occurring in a biomedical or psychosocial context that complicates the understanding of the pain or poses challenges in management.

Initial Pain Assessment Guidelines

- Obtain a detailed history, including an assessment of the pain characteristics, impact of the pain on multipledomains (physical, psychosocial, role functioning, work, etc.), related concerns and comorbidities (other symptoms, psychiatric disorders including substance use disorder, etc.), prior work-up and working diagnosis, and prior therapies
- Conduct a physical examination, emphasizing the neurological and musculoskeletal examination
- Obtain and review past medical records and diagnostic studies
- Develop a formulation including 1) working diagnoses for the pain etiology, pain syndrome and inferred pathophysiology, and 2) plan of care including need for additional diagnostic studies and initial treatments for the pain and related concerns

Patient History

Pain should be viewed like any other medically relevant state: The patient's presenting signs and symptoms, combined with ancillary data, yields a working differential diagnosis, which in the case of pain, is best understood in terms of etiology, inferred pathophysiology and syndrome. Among key ancillary data are details from the past medical history, which may reveal problems relating to the patient's pain (*e.g.*, history of diabetes, toxic exposures, or alcoholism pointing towards a diagnosis of neuropathy) or disorders that would influence therapeutic decision making. A medication history is essential and should include current and recent prescription and nonprescription medications, the patient's perception of the efficacy of those drugs, medications stopped in the recent past and the reasons for that, dietary supplements, assessment of medication adherence (compliance), determination of all prescribers and pharmacies used, and any problems patients have in obtaining, paying for, and taking their medications.

Critical Elements of the Pain History [18-20]

- Characteristics of the pain
- Prior evaluation of the pain
- Prior treatments for the pain
- Patient's perception of impact of the pain on multiple domains
    - Physical functioning
    - Mood and psychological well being
    - Social, familial, and marital well being
    - Role functioning, including work, social, family
    - Sleep, energy level
- Premorbid and comorbid medical and psychiatric conditions
- Comprehensive medication history

Substance Use History

Given the importance of controlled prescription drugs, such as the opioids, in pain management, it also is essential to obtain a substance use history. This typically starts by asking the patient about current and past smoking and use of alcohol ("How do you use alcohol?" "Has it always been this way, or was your pattern of drinking different in the past?"). These queries should be followed by specific questions about other drugs, which should be asked in a dispassionate and non-judgmental way ("What about other drugs?" "Any marijuana now or in the past?" "Any use of prescription or non-prescription pills, now or in the past?" "Cocaine, PCP, methamphetamine, ketamine, heroin?"). Any positive response should engender some additional detail about the use pattern, the degree to which is interfered with normal life, and any previous efforts to obtain help in stopping. Finally, every patient should be asked about a family history of alcohol or drug abuse. Given the importance of these family factors as a risk for problematic drug-related behavior by the patient, it is best to know at the time of the initial evaluation and use this information to stratify the risk of problems should the decision be made at any point to offer a controlled prescription drug such as a opioid.

Day-to-Day Activities and Pain Diary

Understanding the patient's day-to-day activities, including sleep patterns, impact of the pain on work and personal relationships, and present level of physical activity can help the clinician focus on the physical and psychological rehabilitation process. If certain stressful activities exacerbate persistent pain, this will need to be considered in developing psychological, as well as physical rehabilitation approaches to pain management.

Given the importance of comprehensive evaluation and documentation during the course of pain treatment, information about function, mood, and other domains of quality of life is essential over time. Having patients keep a written record of their pains — sometimes called a pain diary — can provide the documentation and allow the clinician to target recommendations. One approach to the diary has patients write down what they are doing three to four times per day at relatively fixed intervals. This information is supplemented with a pain intensity score and the medication that has been taken during the prior period. Alternatively, a diary focused on a specific activity, *e.g.* walking, can be kept. On this, the patient records each episode of walking for exercise, along with the pain and any drugs they are taking. Pain diaries can be individualized in many other ways and should be reviewed together by the clinician and patient as a way of establishing goals, offering support and suggestions, and encouraging a therapeutic alliance in the service of health and quality of life.

Physical Examination and Diagnostic Evaluation

Other data also are critical to the comprehensive pain assessment. A physical examination should be done at the time of the initial pain assessment and then repeated over time as required by the clinical situation. The examination should include assessment of mental status, inspection (posture, guarding, splinting, signs of sympathetic dysfunction), vital signs, and neurological assessment, with emphasis on sensory dysfunction and musculoskeletal status.

The extent to which an underlying etiology for the pain should be sought depends on the context of the patient's illness. Laboratory and radiographic evaluation are usually appropriate in the cases of acute nonsurgical pain, and in cases of persistent pain that has not previously been adequately evaluated, or that has recently changed or is now occurring in association with an evolving disease (*e.g.*, cancer). However, most experts believe that repeated evaluation of the same pain in a patient with long-standing persistent pain rarely yields useful results and may divert attention from symptom control and functional restoration. The guiding principle is to perform a diagnostic evaluation when information needed to establish or confirm a diagnosis is lacking, and when there is a meaningful chance that the test will both yield information and be actionable, *i.e.*, will allow a change of therapy.

Evaluation of Pain Characteristics & Intensity

Pain is inherently subjective and the patient self-report is the gold standard in assessment. Ideally, the description of the pain should characterize its temporal relations, intensity, location, quality and factors that exacerbate or relieve it. Factors that either exacerbate or relieve pain can suggest an underlying cause that can contribute to diagnosis. For example, relief of back pain upon lying down may suggest disc disease, while allodynia in a region of normal looking skin may indicate a neuropathic mechanism. Identification of these factors may also assist in the treatment of pain.

Table: Pain Characteristics

Characteristics	Potential Elements
Temporal	Acute, recurrent, or persistent Onset and duration Course and daily variation, including breakthrough pain
Intensity (verbal rating or 0-10 numeric scale)	Pain “on average” last day or week Pain “at its worst” last day or week Pain “at its least” last day or week Pain “right now”
Topography	Focal or multifocal Focal or referred, and specific radiation Superficial or deep
Quality	Any descriptor (*e.g.*, aching, throbbing, stabbing or burning) Familiar or unfamiliar
Exacerbating / relieving factors	Volitional (“incident pain’) or non-volitional

Source: Portenoy RK and Kanner RM, Definition and assessment of pain. In Portenoy RK and Kanner RM, eds. *Pain Management: Theory and Practice,* Philadelphia: F A Davis; 1996; 7.

Acute vs. Persistent Pain

The distinction between acute and persistent pain is particularly relevant. Acute pain characteristically is of recent onset and is anticipated to have a relatively short duration of no more than days or weeks. Pain is usually considered persistent if it continues more than 3 to 6 months or if it meets one of the following criteria: 1) persisting for at least one month beyond the usual course of an acute illness or the time required for an injury to heal, 2) associated with a chronic pathologic process, or 3) recurring at relatively short intervals (days, weeks, or several months). (Table: Differences Between Acute and Persistent Pain)

Recurrent acute pain is highly prevalent and is the hallmark of some diseases, such as sick cell disease, hemophilia, some inflammatory arthropathies, and some subsets of headache. Nearly all patients with progressive diseases, such as cancer and AIDS, also experience repeated episodes of acute pain which may be related to the disease, therapeutic interventions, or unrelated processes.

Patients with persistent pain commonly experience intermittent episodes of acute pain, which may occur spontaneously or in association with a particular activity. When acute severe pains occur in the setting of persistent “background” pain

Table: Differences Between Acute and Persistent Pain

Characteristics	Acute Pain	Persistent Pain
Temporal features	Recent onset and expected to last no longer than days or weeks	Remote, often ill-defined onset; duration unknown
Intensity	Variable	Variable
Associated affect	Anxiety may be prominent when pain is severe or cause is unknown; sometimes irritability	Irritability or depression
Associated pain- related behaviors	Pain behaviors (*e.g.*, moaning, rubbing, splinting) may be prominent when pain is severe	May or may not give any indication of pain; specific behaviors (*e.g.*, assuming a comfortable position) may occur
Associated features	May have signs of sympathetic hyperactivity when pain is severe (*e.g.*, tachycardia, hypertension, sweating, mydriasis)	May or may not have vegetative signs such as: lassitude, anorexia, weight loss, insomia, loss of libido; these signs may be difficult to distinguish from other disease-related effects.

Source: Portenoy RK and Kanner RM, Definition and assessment of pain. In Portenoy RK and Kanner RM, eds. Pain Management: Theory and Practice, Philadelphia: F A Davis; 1996; 7.

that is otherwise well controlled with an opioid regimen, they are called “breakthrough pains.” Almost two-thirds of cancer patients with persistent pain report breakthrough pain. Given their frequency, breakthrough pain and other types of acute pain should be specifically assessed during the evaluation of persistent pain. A daily pattern of varying pain intensity may exist and diurnal variations are found in some painful conditions such as rheumatoid arthritis (*i.e.*, pain worse in the morning).

The terms acute, persistent, and recurrent pain can be used to depict a wide variety of pain syndromes. Acute monophasic pain is usually of brief duration and is expected to resolve as the underlying cause resolves over hours, days, weeks, or months. This type of pain follows surgery and trauma. When the acute pain is recurrent at relatively brief intervals, the impact can mirror persistent pain. Recurrent pains of this type may be reported by patients with headaches, dysmenorrhea, sickle cell anemia, inflammatory bowel disease, arthritis or musculoskeletal disorders. Pain that is present most of the time, typically fluctuating but seldom absent, comprises a broad clinical group, including persistent pain associated with cancer, many noncancer progressive diseases (*e.g.*, AIDS, some patients with sickle cell anemia, some patients with autoimmune diseases, some patients with neurological syndromes, such as small fiber neuropathies), nonprogressive or slowly progressive diseases (*e.g.*, severe osteoporosis, many types of neuropathic pain, such as postherpetic neuralgia and painful polyneuropathy), and idiopathic syndromes (*e.g.*, fibromyalgia, atypical facial pain, chronic pelvic pain of unknown etiology).

Assessing Pain Intensity

Quantifying the intensity of pain is an essential part of initial and ongoing pain assessment. A variety of validated pain scales are available to assist in the measurement of pain. The clinician should select a method of assessing pain intensity and incorporate it into routine clinical use, obtaining the pain measurement in the same way each time. Whichever method is chosen, it should be systematically applied.[21] Pain measurement tools include simple unidimensional scales or multidimensional questionnaires.

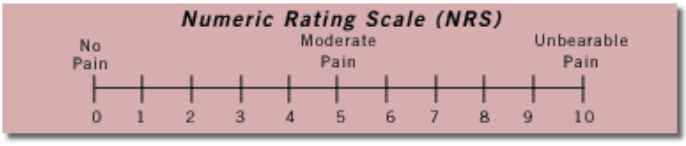
Pain measurement should include both the time-frame and the clinical context of the pain. Patients with acute pain are usually asked to describe their pain “right now” and may be asked about the average intensity over a fixed period of time in order to provide information on the course of the pain. With persistent pain, experts often find it useful to inquire about pain over the previous week and obtain separate measures for pain “on average”, pain “at its worst”, and pain “at its least”.

Commonly used unidimensional scales include the Verbal Rating Scale (VRS), the Numeric Rating Scale (NRS), a Visual Analog Scale (VAS), and a Pictorial Scale. The choice of pain scale may depend on the patient’s age, ability to communicate, or other specific circumstances. While the VRS (*i.e.*, “none”, “mild”, “moderate”, and “severe”) is the simplest measure, other scales can provide additional information.

Unidimensional Pain Scales

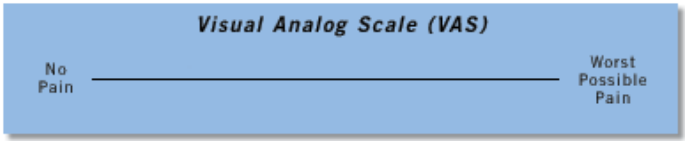
Numeric rating scale

In the clinical setting, the NRS is simple to use and is one of the most common approaches for quantifying pain. Patients indicate their pain intensity on a scale of 0 to 10, with 0 indicating no pain and 10 the worst pain imaginable. In the research setting, this scale is more sensitive to treatment- induced changes than the VRS. The NRS can be used at the bedside by the clinician or at home by the patient as part of a pain diary that serves as a record of pain intensity at fixed times throughout the day. Empirical data suggest that three daily assessments can provide detailed information about the actual pain experience of patients with persistent pain.[22] The NRS can be a helpful technique for clarifying the relationship between pain and activity, the effectiveness of pain treatments, and the pattern of the patient’s pain.



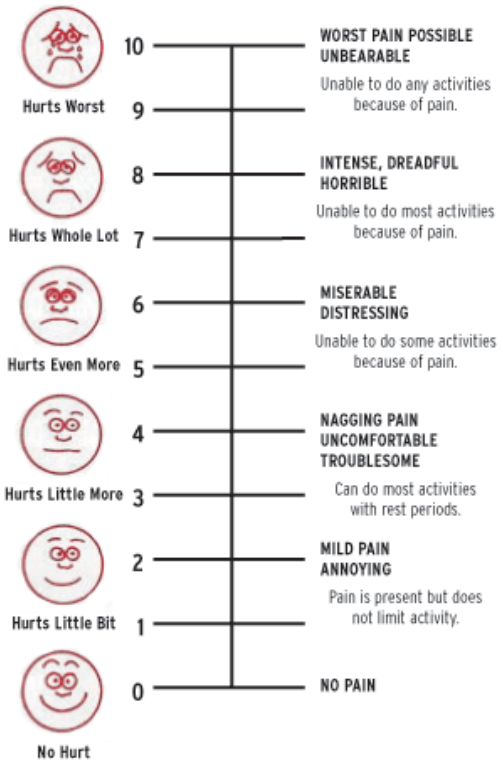
Visual analog scale

The VAS is another validated approach to pain measurement and is conceptually similar to an NRS. The most common VAS consists of a 10-cm line with one end labeled “no pain” and the other end labeled “worst pain imaginable.” The patient marks the line at the point that best describes the pain intensity. The length of the line to the patient’s mark is measured and recorded in millimeters. The main theoretical advantage of the VAS is that it does not limit pain to 10 discrete levels of intensity, permitting a more detailed rating of pain.



Faces pain scale

This scale presents pictures of 6 to 8 different facial expressions depicting a range of emotions. This scale may be useful in young children, in patients who have mild to moderate cognitive impairment, or patients with other language barriers.[23]



Multidimensional Pain Scales

Aside from brief or predictable procedure-related pain, more comprehensive pain assessment requires the determination of other characteristics of the pain, such as location and quality, and its effect on mood and function. Multidimensional pain assessment tools have been developed to quantitate these aspects of pain. They take longer to administer than the unidimensional scales and some patients who are cognitively impaired or poorly educated may find them difficult to complete. They are generally used in pain research, but can be adapted for clinical use, if appropriate and valuable.

The McGill Pain Questionnaire (MPQ)

The MPQ is a validated multidimensional clinical tool that assesses pain in 3 dimensions—sensory, affective, and evaluative—based on 20 sets of words that patients select to describe their pain.[24] The words selected by the patient can be used to describe the quality of their pain, such as burning, shooting, electric, or pins and needles, and as throbbing, aching, or heavy. The description of these types of pain can suggest underlying nociceptive or neuropathic mechanisms. The MPQ takes between 5 and 15 minutes to complete, and thus has been used in pain research rather than clinical practice.

The Brief Pain Inventory (BPI)

The BPI is a well validated multidimensional pain measurement tool with demonstrated reliability and validity in patients with cancer, AIDS, and arthritis. Taking 5 to 15 minutes to administer, it includes 4 pain intensity scales ("right now", "on average", "at its worst", and "at its least"), as well as 7 scales assessing the impact of pain on general activity, mood, ability to walk, work, relationships, sleep, and enjoyment of life.[25] Each of these items is rated on a 0-10 numeric scale. The BPI is widely used in pain research and has been translated into a large number of languages.

The BPI operationalizes the evaluation of pain-related impact on function, mood and quality of life in its 7-item subscale. In this way, it reflects the clinical imperative to assess function and other domains as part of the overall pain assessment. These domains, and others (activities of daily living, other symptoms, social and intimate relationships, etc) also can be measured using any of a large number of validated measures focused specifically on the area in question.

In recent years, a number of multidimensional tools have been developed to assess specific types of pain. For example, there are now a number of instruments that screen for neuropathic pain, or assess it more fully once it is identified. An example of the latter instrument is the the Neuropathic Pain Scale,[26] which like other multidimensional questionnaires is generally used in the research setting. Pain assessment instruments also have been developed for a variety of other disorders, such as fibromyalgia, low back pain or arthritis, and will be addressed as relevant in subsequent modules.

The Treatment Outcomes of Pain Survey

Other instruments have been created in recent years to assess persistent pain and provide a means to track potentially large numbers of patients in terms of key functional domains. An example is the Treatment Outcomes of Pain Survey (TOPS), which is a pain-enhanced version of the health-related quality of life (HRQoL) instrument know as the Medical Outcomes Study Short Form 36 (MOS SF-36 or SF-36). The SF-36 is a useful research tool to determine group change, but lacks the sensitivity to determine individual patient changes. After patients complete the TOPS, the data can be entered into an electronic database which generates a 14 scale TOPS report for the individual patient, TOPS patient group, and group SF-36 reports. [27] A tool like the TOPS may be useful in developing large patient registries for research purposes, and potentially could be adopted for use in individual clinical practices.

Summary

As many as one-fifth of adult Americans, approximately 70 million people, report persistent pain annually. As a public health problem, pain ranks among the most serious, with its prevalence compounded by enormous cost, both in terms of direct health care utilization and in terms of lost productivity and disability. Each clinician must gain the competencies to address the challenges of pain assessment and management. One key competency is assessment based on detailed evaluation of the patient's self-report, combined with other information. Based on this assessment, a diagnosis of the pain in terms of etiology, pathophysiology and syndrome may be possible, and supplemented by an understanding of the impact of the pain in multiple domains and relevant premorbid and comorbid biomedical and psychosocial factors. This understanding is the foundation for the development of an effective plan of care.

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