An Atlas of Investigation and Management

CHRONIC PAIN

Chronic pain is one of the most common symptoms resulting in medical consultation. The increasing focus on chronic pain presents difficulties for the busy practitioner: clinicians must efficiently condense widely varied symptomatic descriptions into characteristic patterns to permit accurate diagnosis and implement effective treatment. This atlas serves as a useful educational resource for the healthcare provider by providing ready access to characteristic descriptions of common pain syndromes – accompanied throughout by algorithms, useful illustrations, imaging studies and evidence-based data summaries from the latest research, all presented in an easy-to-understand visual format.

Chronic Pain: an Atlas of Investigation and Management offers a unique and broad-based perspective on the subject, drawing on the resources and extensive clinical experience of internal medicine, neurology, oncology, rheumatology and anaesthesia. Pain assessment and management is comprehensively addressed by including common syndromes from most body regions and inclusion of medication, non-medication, and interventional therapy options for both non-malignant and malignant chronic pain. A companion resource focused on pain management tools for patients provides charting documentation aids and educational patient handouts to facilitate the patient’s understanding of their individual pain syndrome and a variety of pain management techniques.

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CHRONIC PAIN

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Preface

Chronic pain affects nearly one in every four adults worldwide, with pain one of the most common symptoms resulting in medical consultation. Patients typically describe a complex pattern of discomfort, disability, and distress, with pain affecting physical, social, and psychological functioning. Clinicians must efficiently condense widely varied symptomatic descriptions into characteristic patterns to permit accurate diagnosis and implement effective treatment. *Chronic Pain: An Atlas of Investigation and Management* provides an up-to-date, comprehensive resource for the busy healthcare provider, offering ready access to characteristic descriptions of common pain syndromes, patient case histories, photographs, and imaging studies, and evidence-based data summaries from the latest research studies, all presented in easy-to-understand visual formats. Diagnostic and treatment algorithms included throughout provide practical tools for effectively assessing and managing patients in the busy clinical setting.

This Atlas offers a unique and broad-based perspective, drawing on the resources and extensive clinical experience of anesthesiology, internal medicine, neurology, oncology, and rheumatology. Each of the authors is a recognized authority in chronic pain management, accumulating a wealth of knowledge through direct patient care and pain research. Authors address both nonmalignant chronic pain syndromes and cancer pain, with an additional chapter focused on important end-of-life pain issues. Pain assessment and management is comprehensively addressed by including common syndromes from most body regions and inclusion of medication, nonmedication, and interventional therapy options for both nonmalignant and malignant chronic pain.

*Chronic Pain: An Atlas of Investigation and Management* draws on decades of medical practice with chronic pain patients from each of the contributing authors, ensuring that the recommendations and patient tools have proven value in actual clinical practice.

Dawn A. Marcus, MD

Abbreviations

ADL activities of daily living  EMG electromyography  PHN postherpetic neuralgia
AMPA α-amino-3-hydroxy-5- ESR erythrocyte sedimentation rate  PIP proximal interphalangeal
methylisoxazole-4-propionic acid  FDA Food and Drug Administration  RA rheumatoid arthritis
BBB blood–brain barrier  GABA gamma aminobutyric acid  ROM range of movement
BTP break-through pain  HIV human immunodeficiency virus  RSD reflex sympathetic dystrophy
CGRP calcitonin gene-related peptide  IBS irritable bowel syndrome  SNRI serotonin and norepinephrine
CMC carpometacarpal  MCP metacarpophalangeal  reuptake inhibitor
CNS central nervous system  MMP matrix metalloprotease  SSRI selective serotonin reuptake
CRP C-reactive protein  MRI magnetic resonance imaging  inhibitor
CRPS complex regional pain syndrome  MTX methotrexate  TCA tricyclic antidepressant
CT computed tomography  NK-1 neurokinin-1  UBO unidentified bright object
DHE dihydroergotamine  NMDA N-methyl D-aspartate  WBC white blood cell
DIP distal interphalangeal  OA osteoarthritis
DMARD disease-modifying  UBO unidentified bright object
  antirheumatic drug  WBC white blood cell
Chapter 1

Definition and classification of chronic pain

Introduction

Chronic pain is one of the most common conditions seen in primary care practices, accounting for 40% of office visits (1.1). Data from the World Health Organization identified the worldwide prevalence of significant, persistent pain at about 23% (1.2). The three most common pain locations were back pain (53%), headache (48%), and joint pain (46%). When re-evaluated after 12 months, pain complaints persisted in 49% of patients with baseline pain worldwide. In addition, 70% of persistent pain patients are managed by their primary care physician, while only 2% see a pain management specialist.

1.1 Percentage of primary care practice visits for pain. (Based on Mäntyselkä P, et al., 2001.)

1.2 This survey of primary care patients in 14 countries identified the prevalence of persistent pain, defined as pain occurring on most days for at least 6 months. The pain also needed to be significant enough to result in presentation to a healthcare provider, use of medication, or significant interference with activities. (Based on Gureje O, et al., 2001.)
1.3 Impact of chronic pain. A community-based survey of people in 15 European countries and Israel identified at least moderately severe chronic pain lasting at least 6 months in 19%. Among those who were employed full- or part-time, nearly one in three changed their job or job duties due to pain, while one in five lost a job. Nearly half of all people with chronic pain reported disturbances in household and social activities. One in five reported developing depression and the majority had disturbed sleep. (Based on Breivik H, et al., 2006.)
1.4 Annual per-patient costs from chronic low back pain. Annual 2002 costs for chronic low back pain were estimated by evaluating patients in 14 centres in Sweden. The majority of pain-related costs were indirect, with work absence having the major impact. About 60% of employed patients with low back pain missed at least 1 day of work during the preceding 3 months, with an average work loss of 33 out of 60 possible work days. (Based on Ekman M, et al., 2005.)

<table>
<thead>
<tr>
<th></th>
<th>Total</th>
<th>Direct</th>
<th>Indirect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Euros</td>
<td>25,000</td>
<td>20,000</td>
<td>15,000</td>
</tr>
<tr>
<td>US dollars</td>
<td>10,000</td>
<td>0</td>
<td>5,000</td>
</tr>
</tbody>
</table>

1.5 Chronic pain (pain for >3 months) prevalence in children and adolescents. (Based on Perquin CW, et al., 2000.)

1.6 Definition of acute versus chronic pain

Daily life frequently results in experiences of mild pain – stubbing a toe, twisting an ankle, bumping the ‘funny bone’. These episodes of new-onset pain are termed acute pain. In most cases, acute pain resolves quickly. With more severe injuries, acute pain can persist for several months, resolving during the healing process. The duration of healing depends on the amount of blood flow to various tissues (Table 1.2). After an injury without an ongoing degenerative condition, healing is expected to be completed within 3 months. When pain persists beyond the time of healing or longer than 3 months, this is termed chronic pain (1.6). Chronic pain can continue without the occurrence of ongoing degenerative illness or additional injury.

Table 1.1 Percentage of patients reporting beliefs about doctors’ attitudes toward chronic pain

- My doctor does not think my pain is a problem – 20%
- My doctor never asks about my pain – 22%
- I don’t get enough time to talk to my doctor about pain – 23%
- No one believes my pain is as bad as it is – 29%
- My doctor would rather treat an illness than my pain – 43%

(Adapted from Breivik H, et al., 2006)

Table 1.2 Time to achieve normal healing

<table>
<thead>
<tr>
<th>Organ type</th>
<th>Time to complete healing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin</td>
<td>3–7 days</td>
</tr>
<tr>
<td>Bones</td>
<td>6 weeks</td>
</tr>
<tr>
<td>Tendons and ligaments</td>
<td>3 months</td>
</tr>
</tbody>
</table>
4 Definition and classification of chronic pain

1.6 Acute versus chronic pain. Pain often occurs with trauma or illness, generally decreasing during the period of healing. Acute pain usually resolves within 3 months. Chronic pain persists after healing is completed, due to continued activation of neural pain pathways and muscle spasm. (Based on Marcus DA, 2005.)

1.7 Physiology of acute and chronic pain. Acute pain is associated with inflammation that brings repair cells to the site of injury and activation of spinal pathways that send instructive pain messages to encourage future injury avoidance and provide protective muscle spasm, like a natural splint. During the subsequent weeks or months of healing, inflammation typically resolves and fewer impulses are sent from the spine to register as pain or trigger muscle spasm. When pain persists beyond the period of healing, the memory of the initial injury results in persistent neural messages for pain and muscle spasm. (Based on Marcus DA, 2005.)
Acute pain is an important experience, motivating the injured person to rest and recuperate during the critical healing period. Increased blood flow and protective muscle spasm assist in speeding recovery. Acute pain also provides learning opportunities so that future injury can be avoided (1.7). In some cases, pain persists after healing is completed. This persistent or chronic pain provides patients with false signals about injury and encourages excessive activity restriction.

Patients experiencing chronic pain typically notice fluctuations in their pain severity (1.8). In some cases, pain remains at a moderate level for weeks and then increases to severe pain or a pain flare lasting several days to weeks. In other patients, pain severity may fluctuate between mild, moderate, and severe several times daily. Flares in pain may occur in response to medication withdrawal, stress, increased activity, sleep disturbance, additional injury, or the natural physiology of the pain response. Short-lived pain flares are often well managed with nonpharmacological techniques, such as physical therapy modalities, stretching exercises, relaxation, and distraction techniques. Long-lasting flares, continuing for more than 1 day, often require medication or interventional therapies.

### Pathophysiology of pain

Nerves can be divided based on their size, speed of conduction (depending on degree of myelination), and type of messages transmitted (Table 1.3). Large, fast conducting A-β fibres send nonpainful sensory messages and may help block pain signals. For example, pain can be temporarily relieved by lightly stroking the painful area. The thinly myelinated A-δ and unmyelinated C-fibres send pain impulses and are called nociceptors. Based on conduction speed, sharp pain will be conducted more quickly than dull pain. Nociceptors differ from neurons sending nonpainful touch signals by having a slower speed of signal conduction and an inability to adapt to repeated activation. Although repetitive touch signals will result in reduced nerve firing or desensitization, persistent pain messages result in continued nerve discharges and sensitization.

Noxious signals are transmitted via free nerve endings to the spinal cord and subsequently to the brain (1.9). A variety of neurotransmitters are important for relaying pain messages, including substance P, glutamate, and calcitonin gene-related peptide. Descending pathways from the brain help modulate pain activity via serotonin, norepinephrine, and opioid release. Pain perception is also modulated by important cerebral influences on the limbic system (1.10). This may explain how psychological techniques, like cognitive restructuring and stress management, can effectively reduce chronic pain complaints.

<table>
<thead>
<tr>
<th>Neuron type</th>
<th>Size</th>
<th>Average conduction speed</th>
<th>Adaptability</th>
<th>Signal</th>
</tr>
</thead>
<tbody>
<tr>
<td>A-β</td>
<td>Large – 8 μm</td>
<td>Fast – 50 m/sec</td>
<td>Yes</td>
<td>Touch, proprioception, inhibit pain</td>
</tr>
<tr>
<td>A-δ</td>
<td>Small – 1 μm</td>
<td>Moderate – 10 m/sec</td>
<td>No</td>
<td>Sharp pain</td>
</tr>
<tr>
<td>C</td>
<td>Small – 1 μm</td>
<td>Slow – 1 m/sec</td>
<td>No</td>
<td>Dull pain</td>
</tr>
</tbody>
</table>
1.9 Peripheral pain messages activate central structures in the spinal dorsal horn. A-δ and C-fibres transmit peripheral pain signals to the dorsal spinal root, which sends messages to the superficial dorsal horn. Connections from lamina 1 convey pain signals to the substantia gelatinosa, where pain messages are modulated via intermediate neurons and signals from descending neural tracts travelling in the dorsolateral funiculus. Activation of descending pathways helps explain how pain can be lessened with distraction or on the battlefield. Second-order neurons then cross the spinal cord and travel to the thalamus via the lateral spinothalamic tract. Third-order neurons from the thalamus signal the somatosensory cortex, resulting in the conscious perception of pain.

1.10 Central modulating influences on pain perception (adapted from Robert Bennett, MD). After pain impulses travel to the thalamic nuclei, pain perception is influenced by signals from the limbic system and prefrontal cortex. These cortical influences help explain the modulating role that memory and learning have on pain perception.
Central sensitization model of chronic pain

When pain symptoms persist beyond the healing period, pain is often perpetuated by activation of the central nervous system (CNS) (Table 1.4). Under normal conditions, presynaptic neural input from one neuron produces a predictable and comparable signal output after postsynaptic activation (1.11). When nerves become sensitized in the dorsal horn in chronic pain patients, postsynaptic signal output exceeds presynaptic input from peripheral nociceptors, resulting in a reduced pain threshold and the spread of pain beyond the area of the original injury.

Several experimental models of chronic pain have been developed in animals subjected to peripheral nerve injury. These models effectively describe changes that occur in patients with neuropathic pain and may also explain changes occurring in other chronic pain conditions. Similar to humans with chronic pain, animals exposed to peripheral nerve injury likewise develop hyperalgesia, allodynia to cold and mechanical stimulation, and pain behaviours (favouring or gnawing at the injured leg) (1.12)11. This heightened pain response persists for about 2 months in rats experiencing chronic constriction injury with ligatures loosely tied about the proximal sciatic nerve and up to 7 months after partial sciatic nerve ligation with a tight ligature. These experimental models may be likened to humans who experience nerve trauma or compression (e.g. postoperative pain, carpal tunnel syndrome, or a herniated disc). Even after surgery to repair damage or relieve constriction, chronic pain, hyperalgesia, and allodynia often persist.

Neural sensitization has been studied most thoroughly in the dorsal horn of the spinal cord, using the rodent models described above. Changes occur in neurotransmitter activity at both pre- and postsynaptic sites in the dorsal horn where first-order peripheral nerve terminals activate second-order

<table>
<thead>
<tr>
<th>Table 1.4 Triad of sensitized pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Hyperalgesia: amplified response to painful stimulus</td>
</tr>
<tr>
<td>2 Allodynia: pain perception to nonpainful stimulus</td>
</tr>
<tr>
<td>3 Receptive field expansion: spread of pain beyond area of original injury</td>
</tr>
</tbody>
</table>

1.11 Neural processing under normal and chronic pain conditions (adapted from Robert Bennett, MD). This series of drawings depicts changes in nerve signalling with chronic pain exposure. The vertical bars represent action potentials. A: Normal processing, with the signals generated from pain input corresponding to the amount of pain generation output. With repeated pain signalling, the dorsal horn becomes sensitized, forming the basis of neural wind-up (B). Wind-up is mediated by glutamate-dependent N-methyl d-aspartate (NMDA) receptor activation. With sensitization, pain output signals exceed what would be predicted based on the number of input action potentials. Persistent dorsal horn sensitization leads to amplified pain sensitivity and the triad of chronic pain: hyperalgesia (C), allodynia (D), and pain expansion (E).
central neurons in the spinal cord (1.13). Alterations in the ability of glutamate to activate N-methyl d-aspartate (NMDA) receptors with repetitive peripheral nerve activation result in postsynaptic calcium influx and a cascade of events that results in excessive pain signalling from the second-order neuron. Changes in pre- and postsynaptic transmission provide possible sites for medication intervention with drugs designed to decrease excitatory activity by blocking NMDA receptors, calcium channels, or glutamate, or increasing GABA inhibitory drive.

1.12 Sensory dysfunction induced by peripheral nerve injury. Sensation was tested in rats before (Pre-: P) and every other day after sciatic nerve chronic constriction injury. Withdrawal from cold stimulation was tested on odd days and touch response was tested on even days. Rats exposed to peripheral nerve injury experienced an increase in pain response to placing their feet on an ice plate (cold allodynia, top) and a reduced threshold for perceiving light touch as pain (hyperalgesia, bottom) after 3–5 days. There were no changes in the uninjured rats. (* Denotes significant change from preinjury level, \( P < 0.05 \).) (Based on Keay KA, et al., 2004\textsuperscript{11}.)

1.13 Pre- and postsynaptic changes after peripheral nerve stimulation. These diagrams represent the synapse between first-order (peripheral) neurons and second-order neurons in the dorsal horn. N-methyl d-aspartate (NMDA) receptors are located throughout the central nervous system and normally are involved with removing dying neurons. A: Under normal circumstances, NMDA receptors are blocked by magnesium, preventing activation by glutamate. The presynaptic release of glutamate normally activates nonpain \( \alpha \)-amino-3-hydroxy-5-methylisoxazole-4-propionic acid (AMPA) receptors. In addition, inhibitory gamma aminobutyric acid (GABA) normally prevents excessive release of glutamate and substance P.

B: After peripheral nerve injury, GABA activity decreases and excessive activation of AMPA receptors by glutamate and neurokinin-1 (NK-1) receptors by substance P results in loss of NMDA-receptor magnesium blockade, allowing glutamate to bind to NMDA receptors. This results in an influx of calcium into the postsynaptic neuron. After calcium enters the postsynaptic cell, there is further activation of NMDA receptors, more calcium influx, and increased presynaptic release of substance P, resulting in wind-up, signalling a stronger pain message than would have been sent by the original peripheral nerve impulse. Ca: calcium; Mg: magnesium.
The ability of pain exposures to change subsequent sensitivity to pain in humans has been demonstrated in two important studies investigating the role of perinatal circumcision pain on later sensitivity to routine vaccinations\textsuperscript{12,13}. Response to vaccination given at 4–6 months was compared in boys who had (N=30) and had not (N=12) been circumcised as newborns\textsuperscript{12}. Demographics were similar between groups. Pain reactions were significantly higher among boys who had been circumcised (1.14A). Previously circumcised babies were also less responsive to premedication before vaccination with local anesthetic (1.14B). In a second study, boys circumcised after pretreatment with 5% topical lidocaine–prilocaine had a significantly lower response to vaccination than boys circumcised with no anesthesia (P<0.05)\textsuperscript{13}. These data support the theory that painful peripheral stimulation can produce long-lasting changes in central pain processing mechanisms.

**Understanding myofascial pain**

Muscle spasm and hurting frequently accompany chronic pain and may be either the primary pain generator or a secondary pain contributor. The protective muscle spasm that occurs with an acute injury can persist as a nonproductive, persistent muscle spasm and tenderness. In addition, changes in normal movement patterns due to pain and deconditioning from excessively rested muscles can further aggravate muscle spasm. Patients often develop co-contraction of complementary muscles, e.g. muscle flexors and extensors. This co-contraction results in restricted active movement and increased pain.

Muscle or myofascial pain is characterized by areas of involuntarily contracted muscle (Table 1.5). These contracted areas result in focal tenderness and shortened muscles, with reduced range of motion and muscle strength (1.15). Palpation may also result in predictable pain referral patterns, which will be described for common myofascial pain conditions in subsequent chapters.

### Table 1.5 Hallmarks of myofascial pain

- **Taut muscle band**
  - Contracted cord of muscle fibres
- **Local twitch response**
  - Plucking band or inserting needle into band causes involuntary muscle contraction
- **Trigger points**
  - Palpating taut band results in local tenderness (latent trigger point) or referred pain (active trigger point)
Chronic pain assessment

All patients should be asked to complete a basic pain assessment, including both qualitative and quantitative pain assessment measures. The most efficient method for patients to describe their pain location and quality is through the use of a pain drawing (1.16). Familiarity with pain drawings allows the practitioner to assess patients with pain complaints rapidly. These drawings can be particularly helpful for patients with complicated pain syndromes. Patients often focus on their most severe or newest ache when verbally describing their pain. Understanding the full scope of their pain involvement allows the diagnosis of pain conditions that affect more widespread areas, such as fibromyalgia and rheumatoid arthritis. In addition, pain drawings can also help show common pain referral patterns from neuropathic or radicular pain, as well as myofascial pain. Completed pain drawings will accompany patient case reports in subsequent chapters.

Pain severity can be quantified using a numeric rating scale, visual analogue scale, or descriptive scale (1.17). In clinical practice, the numeric rating scale is usually the easiest for patients to understand and doctors to interpret quickly. Numeric pain ratings of 5 or higher correlate with substantial pain-related interference and disability14. In patients with nonmalignant chronic pain, a score of ≥5 correlates with moderate pain and interference, while scores of ≥7–8 denote severe pain and interference. Among patients with cancer-related pain, a score of >4 similarly corresponds to moderate pain and >7 severe pain15.

Pain severity and interference can also be quantified at initial screening and in post-treatment assessments using validated screening tools, such as the Profile of Chronic Pain: Screen, a 15-item questionnaire that patients can complete in about 5 minutes (1.18). This tool characterizes pain severity, functional impact, and emotional distress.

Patients reporting chronic pain require a comprehensive evaluation of both their pain complaint and general medical condition. Reviewing a pain drawing, pain severity assessment using the numeric rating scale, and the Profile of Chronic Pain: Screen helps clarify pain complaints. A detailed history and physical examination help differentiate among possible diagnoses and the need for additional laboratory or radiographic testing (1.19).
1.16 Pain drawing recording sheet. Patients are instructed to shade all painful areas. Patients are instructed to shade all painful areas using the following key: /// for pain; :::: for numbness; **** for burning. (Based on Marcus DA, 2005.)

<table>
<thead>
<tr>
<th>Pain</th>
<th>Numbness</th>
<th>Burning or hypersensitivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>///</td>
<td>::::</td>
<td>****</td>
</tr>
</tbody>
</table>

1.17 Pain severity assessment measures. 

**A**
Patients rate pain severity from 0 (no pain) to 10 (unbearable pain).

Circle the number that indicates your pain severity:

<table>
<thead>
<tr>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>No pain</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>Unbearable pain</td>
</tr>
</tbody>
</table>

**B**
Patients mark their pain severity on a 100 mm line. Pain severity is measured in mm from zero (no pain) to 100 mm (worst pain imaginable).

Mark your pain severity along this line:

No pain —————————————————— Worst pain imaginable

**C**
Patients choose descriptors about pain from: none, mild, moderate, severe.

Circle the term that indicates your pain severity:

None    Mild    Moderate    Severe
12 Definition and classification of chronic pain

<table>
<thead>
<tr>
<th>Patient name:</th>
<th>Date:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Circle an answer for each question.</td>
<td></td>
</tr>
</tbody>
</table>

**A Pain severity**

1 **How often do you get pain?**
   - 0 – Never
   - 1 – Less than once per month
   - 2 – Once per month
   - 3 – Twice per month
   - 4 – Once per week
   - 5 – Several times weekly
   - 6 – Daily

2 **How severe is your average level of pain? (0 = very little pain, 9 = unbearable pain)**

<table>
<thead>
<tr>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
</tr>
</thead>
</table>

3 **How often do you get 1 hour of severe pain?**
   - 0 – Never
   - 1 – Less than once per month
   - 2 – Once per month
   - 3 – Twice per month
   - 4 – Once per week
   - 5 – Several times weekly
   - 6 – Daily

4 **How severe is your most severe level of pain? (0 = very little pain, 9 = unbearable pain)**

<table>
<thead>
<tr>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
</tr>
</thead>
</table>

**B Pain interference: rate how often your pain interferes with each of the following:**

1 **Enjoyable activities**
   - 0 – Never
   - 1 – Less than once per month
   - 2 – Once per month
   - 3 – Twice per month
   - 4 – Once per week
   - 5 – Several times weekly
   - 6 – Daily

2 **Home responsibilities**
   - 0 – Never
   - 1 – Less than once per month
   - 2 – Once per month
   - 3 – Twice per month
   - 4 – Once per week
   - 5 – Several times weekly
   - 6 – Daily

3 **Relationships**
   - 0 – Never
   - 1 – Less than once per month
   - 2 – Once per month
   - 3 – Twice per month
   - 4 – Once per week
   - 5 – Several times weekly
   - 6 – Daily

4 **Personal goals**
   - 0 – Never
   - 1 – Less than once per month
   - 2 – Once per month
   - 3 – Twice per month
   - 4 – Once per week
   - 5 – Several times weekly
   - 6 – Daily

5 **Self-care**
   - 0 – Never
   - 1 – Less than once per month
   - 2 – Once per month
   - 3 – Twice per month
   - 4 – Once per week
   - 5 – Several times weekly
   - 6 – Daily

5 **Thinking clearly, problem-solving, concentrating, or remembering**
   - 0 – Never
   - 1 – Less than once per month
   - 2 – Once per month
   - 3 – Twice per month
   - 4 – Once per week
   - 5 – Several times weekly
   - 6 – Daily

**C Emotional burden (0 = never, 5 = extremely often)**

1 **How often does your pain make you sad or depressed?**
   - 0 – Never
   - 1 – Less than once per month
   - 2 – Once per month
   - 3 – Twice per month
   - 4 – Once per week
   - 5 – Several times weekly
   - 6 – Daily

2 **How often does your pain make you tense, anxious, or jittery?**
   - 0 – Never
   - 1 – Less than once per month
   - 2 – Once per month
   - 3 – Twice per month
   - 4 – Once per week
   - 5 – Several times weekly
   - 6 – Daily

3 **How often does your pain make you angry?**
   - 0 – Never
   - 1 – Less than once per month
   - 2 – Once per month
   - 3 – Twice per month
   - 4 – Once per week
   - 5 – Several times weekly
   - 6 – Daily

4 **How often does your pain make you isolated or lonely?**
   - 0 – Never
   - 1 – Less than once per month
   - 2 – Once per month
   - 3 – Twice per month
   - 4 – Once per week
   - 5 – Several times weekly
   - 6 – Daily

5 **How often does your pain reduce your ability to enjoy your life?**
   - 0 – Never
   - 1 – Less than once per month
   - 2 – Once per month
   - 3 – Twice per month
   - 4 – Once per week
   - 5 – Several times weekly
   - 6 – Daily
### Definition and classification of chronic pain

#### 1.18 (opposite) Profile of Chronic Pain: Screen questionnaire. This validated pain assessment questionnaire addresses pain severity, interference, and emotional burden. Scores are calculated for each category by adding response scores. Possible score ranges are: 0–30 for severity, 0–36 for interference, and 0–25 for emotional burden. Based on a United States national nonpain sample, norms for males are about 11 for pain severity, 3.5 for interference, and 3 for emotional burden. Norms for females are about 13 for pain severity, 5 for interference, and 5 for emotional burden. (Based on Ruehlman LS, et al., 200516.)

#### 1.19 Chronic pain assessment. EMG/NCV: Electromyography/nerve conduction velocity; MRI: magnetic resonance imaging; ROM: range of motion. Active ROM is performed by asking the patient to move joints through full range. Passive ROM is performed by asking the patient to relax; the examiner then moves the relaxed joint through the full range.
Differentiating among pain diagnoses

In general, chronic pain can be divided into several categories, including myofascial, mechanical, and neuropathic pain. Pain characteristics and physical examination findings can distinguish among pain categories. In some cases, patients may have contributions from several pain categories, such as migraine plus myofascial pain or neuropathic plus mechanical pain. Pain descriptions, like burning, cold, and numb, are often associated with neuropathic pain. Pain location along typical nerve distribution patterns can also help identify neuropathic pain. Myofascial pain is often quite severe and typically associated with restricted active motion. Characteristic muscle tenderness and pain referral patterns help identify myofascial pain. Mechanical pain is characterized by restrictions in joint movement when isolated from muscle contraction. In some cases, a physical therapy assessment is necessary to identify mechanical dysfunction in patients who are unable to relax muscles successfully for passive range of motion testing. Typically, patients with mechanical pain will experience pain reproduction or aggravation with movement of involved joints.

1.20 Pain assessment algorithm. AROM: active range of motion; EMG/NCV: electromyography/nerve conduction velocity; MRI: magnetic resonance imaging; PROM: passive range of motion. In general, patients with myofascial pain have limited AROM due to muscle cocontraction. Think muscular dysfunction when PROM exceeds AROM. If patients successfully reduce muscle contraction and relax joints for PROM testing, restrictions usually represent a mechanical dysfunction.
References


