Presented in a clear, systematic format, this clinically oriented book provides nurses and physicians with quick access to much-needed pain management guidelines. With a unique focus on treatment options for patients with chronic persistent pain, this guide provides critical guidance on managing difficult conditions such as fibromyalgia and neuropathic pain.

The text expertly assists practitioners in assessing pain in a variety of patient populations and provides professional insight on selecting patient-appropriate medications and interventions to achieve optimal pain management for adult patients.

KEY FEATURES:
• Contains the newest guidelines on how to use a combination of pain screening tools to accurately assess the nature, intensity, and occurrence of patient pain
• Provides information on new medications and combinations of medications to use for chronic pain
• Presents essential information on safe prescribing and screening tools such as the Opioid Risk Tool (ORT) and screening tools for aberrant behaviors
• Provides strategies for utilizing nontraditional treatment options such as acupuncture, energy therapies, and psychological and coping strategies
Pain Distress/Intensity Scale

None 0
Annoying 1
Uncomfortable 2
Dreadful 3
Horrible 4
Mild Pain 5
Severe Pain 6
Very Severe Pain 7
Worst Possible Pain 8
Agonizing 9
Worst Possible Pain 10

0 1 2 3 4 5 6 7 8 9 10
Yvonne D’ArCY, MS, CRNP, CNS, is an expert pain management and palliative care nurse practitioner who has received several awards for nursing excellence, and who possesses a remarkable publishing and presentation career history. Currently, Ms. D’ArCY is the nurse practitioner for Pain Management and Palliative Care at Suburban Hospital–Johns Hopkins Medicine, in Bethesda, Maryland. She has significant experience in pain management, having worked as Oncology Pain Service and Staff Education Coordinator at Johns Hopkins Oncology Center; Acute Pain Service Coordinator and Pain Clinic Supervisor at the Mayo Clinic in Jacksonville, Florida; and Pain Center Manager and Clinical Coordinator of the Acute Pain Service at the Heartland Health System in St. Joseph, Missouri. Her accomplishments include delivering more than 100 poster and oral presentations, publishing more than 100 journal articles on pain-related topics, authoring 3 books on pain, serving as a consultant for hospitals and health care providers, providing peer review for and serving on the editorial boards of Nursing, The Journal for Nurse Practitioners, and Pain Management News. Ms. D’ArCY has been an active member of the American Society of Pain Management Nurses, having served on the Board of Directors and as Chairman of the Clinical Practice Committee, is a member of the American Pain Society, and serves as a member of the Clinical Guidelines Committee and the American Academy of Nurse Practitioners. She is the recipient of the Nursing Spectrum Excellence Division Award for Advancing and Leading the Profession.
Compact Clinical Guide to

CHRONIC PAIN MANAGEMENT

An Evidence-Based Approach for Nurses

Yvonne D’Arcy, MS, CRNP, CNS
This book is dedicated to all the practitioners who work with patients who have chronic pain of all types. These patients are a group of special people with individual needs and hopes. For all the hard work, time, and dedication you have extended to these patients, I express my sincere appreciation.
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Combined Thermometer Scale  Inside Front Cover
Nurse practitioners (NPs) are the fastest growing group of practitioners in primary care practice. There are currently 125,000 NPs who are licensed to practice, with the majority working in primary care. In 2008, 8,000 NPs graduated from NP educational programs. Growth-wise, the current U.S. Government Accountability Office report on growth and trends in primary care indicates that primary care NPs are increasing at a rate of 9.44% per capita compared with 1.77% per capita for medical doctors in primary care.

Simultaneously, the number of aging Americans has steadily increased with the entry of the baby boomers into the ranks of those patients who are candidates for chronic pain complaints, such as osteoarthritis, low back pain, and other degenerative pain conditions. In primary care, pain is one of the biggest reasons that patients seek help from health care providers. About 26 million people in the United States suffer from severe pain at least monthly. In the working population, pain is responsible for one-fourth of all sick time lost from work, or 50 million lost workdays per year. Unfortunately, about 28% of Americans with pain do not feel that there is any real solution for their pain.

Research in pain management is trying to provide new solutions and answers to these difficult-to-treat patients who have complex pain needs. There are many new guidelines and position statements, both general and disease-specific, that make practice recommendations for using long-term opioid therapy, diagnosing and treating low back pain, and use of medication such as methadone. Genetic factors are being studied to determine which patients have the best
outcomes with certain pain medications. New technology is growing every day so that new delivery systems are available to deliver the available pain medications.

This book is a comprehensive guide to treating chronic pain. NPs will find information on prescribing opioids safely, pain assessment, and interventional options for pain relief. Using opioid agreements, performing urine screens, and learning to screen patients for potential problems when opioids are initiated are all detailed, with examples of screening tools and agreements.

The author is an NP who sees many patients with chronic pain in her practice. She has many years of experience diagnosing and treating patients with chronic pain, and she is considered to be a leader in her field. She is sharing her expertise and experiences in this book so that other NPs can provide high-quality pain management to their patients and get positive outcomes. The book provides easy-to-use features and case studies, and will be a useful addition to any clinician’s references for clinical practice.

Bill McCarberg, MD
Founder, Chronic Pain Management Program
Kaiser Permanente
San Diego, California
Overview and Assessment of Chronic Pain
INTRODUCTION TO CHRONIC, PERSISTENT PAIN

Health care providers are seeing larger numbers of patients with chronic, persistent pain than ever before. The causes of the pain are varied, but they all still have the potential for disability, decreased functionality, and decreased quality of life. The number of patients with chronic pain has increased nationally to the point that many health care providers consider chronic pain to be a major national public health problem (Trescot et al., 2008).

What we do know about most nurse practitioners is that they feel that their basic nurse practitioner education did not prepare them to treat patients with chronic pain. In a survey of 400 nurse practitioners, 62% of the respondents felt they had been prepared to assess patients with chronic pain, whereas 38% indicated they did not feel prepared. When treatment of chronic pain was queried, only 44% of the respondents felt they had been prepared to treat chronic pain, whereas 56% felt they had not been prepared (D’Arcy, 2009a). When asked to choose which of the concerns were most important when prescribing opioids for chronic pain, the same survey group indicated the following concerns from most important to least important:

1. Cost
2. Fear of regulatory oversight
3. Addiction  
4. Not knowledgeable enough about medications  
5. Don’t want to be seen as different from other prescribers (D’Arcy, 2009a)

These fears of regulatory oversight and legal consequences are also colored by fears of addicting patients to opioids when long-term opioid therapy is used to treat chronic pain. When the survey respondents were asked how comfortable they were with prescribing opioids for 12 months or more when the patient had chronic pain, only one third of the respondents felt they had a good comfort level with the practice (D’Arcy, 2009a). These findings are consistent with those of other surveys on pain management knowledge and attitudes that have been used for several years.

Despite national efforts to teach patients and prescribers about chronic pain by declaring 2000 to 2010 as the Decade of Pain Control and Research, we have made little headway. Moreover, pain is still undertreated. Although only approximately 5% of patients in primary care develop a substance abuse disorder/addiction with long-term opioid use, including all of those patients who have been exposed to opioids and those patients who had never used opioids, these outdated perceptions still persist (Fishbain, Cole, Lewis, Rosomoff, & Rosomoff, 2008). Although there are other studies that categorize substance abuse disorders and opioid misuse differently and have different statistical findings, the endpoint is that true addiction is not as common as prescribers perceive it to be, and most patients with chronic pain can tolerate long-term opioid therapy successfully.

Who are these patients with chronic pain and what are their complaints? Because chronic, persistent pain is now considered a chronic illness, how can these patients cope with the long-term nature of the condition? What types of treatments are available? What is the best way to assess pain in these patients? The following chapters of this book will answer these questions and provide evidence-based information on many aspects of chronic, persistent pain.
PREVALENCE OF CHRONIC PAIN

A patient with chronic pain may be anyone you see or know. They may be young or old, wealthy or homeless. Chronic pain does not respect age, race, financial status, or gender. It can affect anyone at any time, and the effects of the pain can be life changing. The pain can be the result of surgery, an injury, disease, or treatments such as chemotherapy, or it may just start for no apparent reason. Once the pain occurs, it will affect every aspect of the person’s life. Every patient with chronic pain has a story to tell of how the pain has changed their lives and how they have learned to adapt and cope with it.

Chronic, persistent pain accounts for 40 million patient visits annually and is the most common reason that patients seek help from health care professionals. On average, chronic pain patient has:

- Had pain for 7 years
- Had three major surgeries
- Incurred medical bills of $50,000 to $100,000

There are many different types of chronic pain. Responses to a survey by Research America indicate that the most common types of chronic pain include the following:

- Back pain (28%)
- Arthritis and joint pain (19%)
- Headaches/migraine (17%)
- Knee pain (17%)
- Shoulder pain (7%)

Low back pain, the most common type of chronic pain, has become a common complaint in the American health care system. The normal aging process causes the spinal discs to desiccate and flatten. By age 20, the vascularity of the spinal discs decreases and by age 30, the desiccation of the disc can cause fissures to develop in the endplates of the vertebral bodies (D’Arcy, 2009). Because of these spinal changes that occur as we age, it is estimated that 95% of the population will have the beginning of degenerative disc disease by age 50 (D’Arcy, 2009b). Patients
who are at risk for low back pain, the most common type of chronic pain, include the following:

- Those who are older than 55 years
- Obese patients
- Those with poor physical condition and who do not engage in regular exercise
- Lower socioeconomic groups who have fewer opportunities to access health care
- Workers who have engaged in heavy labor over time
- Those with reduced spinal canal dimensions (spinal stenosis) (D’Arcy, 2009b)

Unfortunately, the picture of chronic pain for the older patients is even more grim. The American Geriatrics Society estimates that 80% of patients in long-term care facilities experience chronic daily pain. Assessing and treating pain in these patients is difficult because of the high incidence of dementia and nonverbal patients. The story for community-dwelling elders is a little better, with 25% to 50% of them reporting chronic pain that affects their ability to function. No matter what the age of the patient with chronic pain is, there is an impact on the way these patients lead their lives.

**COSTS OF CHRONIC PAIN**

The cost of chronic, persistent pain cannot be fully measured, because it includes not only lost work time, or increased health care utilization, but also personal and quality-of-life issues, which have costs that cannot be calculated. Patients with chronic pain are often misunderstood and undertreated for the pain. As they seek relief for their pain, the health care system may view them as drug seeking rather than relief seeking. This may cause the chronic pain patient to present a social mask to the public that hides the full extent of the pain. Assessing pain in a patient who is trying to hide the effects of chronic pain is much more difficult and requires a comprehensive set of questions to obtain the needed information.
Most patients with chronic pain report pain that exists at some level throughout the day. The patients with low back pain may have episodes of pain in which the pain increases and then returns to a lower, more tolerable level. Along with the pain, patients can become anxious or depressed as the pain appears to be untreatable or as pain intensity increases. The uncertainty of the pain experience can lead patients to feel helpless and hopeless.

**Monetary Costs**

It is difficult to measure the cost of chronic pain. The best evaluation is that chronic pain costs are estimated to be $100 billion per year and are related to the following:

- Health care
- Welfare and disability costs
- Losses in tax revenue
- Lost productivity through both absenteeism and presentism (at work, but in pain)

Presentism alone is estimated to cost $61.2 billion per year (Stewart, Ricci, Chee, Morganstein, & Lipton, 2003). Most patients with chronic pain want to work but are limited by pain. Because these patients try to work but are less productive, it is a hidden cost that is hard to evaluate.

**Personal Costs**

Pain that is long term can take a toll on money and employment, but it can also rob the patient of quality of life, disrupt sleep, and cause significant depression. Depression is a common occurrence in patients with chronic pain. The depression is more of a situational depression than a deep-seated clinical effect. If depression is a part of a patient’s chronic pain, treatment with antidepressants is indicated. If the depression is allowed to go untreated, the patient may develop suicidal ideation. Unfortunately, the rate for suicide in chronic pain patients is twice the rate for the similar patient demographic without chronic pain (Tang & Crane, 2006).
Chronic pain can also result in sleep disturbances, which can rob the patients of needed rest and restorative sleep they need to help them cope with the stress of daily life with pain. Sleep disturbances are common and occurred in about 55% of patients in one study who reported restless/or light sleep after the onset of pain (Marin, Cyhan, & Miklos, 2006). The most common adverse effects of sleep disturbances that are reported by patients include the following:

- Delayed onset of sleep
- Daytime fatigue
- Nonrestorative sleep

The personal costs for the older patient with chronic pain are very significant. Because prescribers are reluctant to provide high-level opioid medications to many older patients, for fear of unwanted side effects such as oversedation, constipation, and confusion, the pain will be untreated or undertreated (Bruckenthal & D’Arcy, 2007). Undertreated pain can lead to the following:

- Depression, anxiety
- Decreased socialization
- Sleep disturbances
- Impaired ambulation and functioning
- Increased health care utilization and costs (Bruckenthal & D’Arcy, 2007)

Chronic pain can lead to poor self-esteem, financial ruin, and diminished quality of life. It affects the health status of the patient by suppressing natural killer cells, decreasing the body’s ability to defend itself against tumor and virus-infected cells. Chronic pain not only reduces the quality of life but also can have an impact on life itself.

**PAIN TRANSMISSION**

**Pain Theories**

The mechanisms of pain transmission are different for acute pain and chronic pain. The onset of acute pain is sudden and can
Pain Transmission

Provoke a fight or flight type of response, with adrenaline release that will subside rapidly. Chronic pain, on the other hand, is long term, and over time, more complex and advanced pain-facilitating responses, such as the activation of N-methyl-D-aspartate (NMDA) receptors, take place. Many pharmaceutical companies aim the action of their medications at a specific sites in the pain transmission process. As one example, retaining serotonin at the synaptic junction can help reduce the amount of pain-facilitating substances available to create or continue the pain stimulus.

There are theories that have been advanced over the years about how pain is transmitted and what physiologic mechanisms are involved. One of the earliest theorists was René Descartes who felt that pain was a stimulus response mechanism. This concept was also called the labeled line theory. In this theory, pain was seen as a painful stimulus that traveled up to the brain, resulting in the body recognizing the sensation as pain. An example would be a stimulus, such as a burn or trauma, that would travel up to the brain, and the brain would recognize it as pain. The resulting response would be for the body to withdraw from the pain, such as removing the hand from a fire. This theory focuses primarily on the physical aspect of pain rather than including the emotional or psychological aspects of the pain experience.

Especially for patients with chronic pain, the psychological and emotional component of pain is an important aspect of the condition. Older theories, such as those espoused by Pavlov, considered pain to be a learned response that was affected by cultural and learned behaviors that could be offset by operant conditioning. Turk described pain as a multidimensional experience and proposes that the patient, not the health care practitioner, is really the specialist on the pain. This theory empowers the patient to become active participants in pain treatment and helps the patient diminish negative behaviors and increase positive reinforcing behaviors (American Society for Pain Management Nursing [ASPMN], 2010).
Perhaps the most well known theory of pain transmission is the Gate Control Theory developed by Melzack and Wall in 1965 (ASPMN, 2010). In this theory, the psychological and physiological aspects of pain transmission are combined. Simplistically, the Gate Control Theory states that a pain stimulus can be significant enough in intensity to “open” a neuronal gate that will allow the pain stimulus to proceed up the nervous system to create a sensation that can be identified as pain by the brain.

The actual steps in pain transmission according to the Gate Control Theory include the following:

- A pain stimulus from the body periphery is carried by A-delta and C nerve fibers to the dorsal horn of the spinal cord.
- The gate is located in the substantia gelatinosa in the dorsal horn of the spinal cord, and it can facilitate or inhibit, either promote or stop, the progression of the nerve impulse through the central nervous system.
- If the painful stimulus is of sufficient intensity or persists, the pain is transmitted up through the limbic system to the cerebral cortex.
- In the cerebral cortex, the stimulus is recognized as pain and the efferent neural path is activated to provide a response to the pain. (Adapted from ASPMN, 2010)

As science has investigated and furthered the knowledge of this pain transmission theory, several other concepts have emerged:

- The central control processes and central intensity process located in the brain and limbic system help to translate the understanding of the sensation and can modulate the section of the descending pain pathways.
- When pain stimuli entering the nervous system reach critical levels, the T-cell system is activated, which creates a link between the brain and body that connects the subjective and objective experience of pain.
- By increasing the sensation of pain, peripheral nerve sensitization can be caused through continued nerve stimulation producing a state of hyperexcitability because of alternation in the sodium ion channels. Continued pain stimulation can be increased as inflammatory response persists.
Wind-up and neuroplasticity can also occur. Wind-up is a phenomenon that develops when, as the result of continued moderate to severe pain, the NMDA receptors are activated. These receptors serve to process the pain faster and with more intensity, creating a pain intensity that is greater than expected for the stimulus. The pain response is greatly enhanced when wind-up has occurred. Central sensitization can occur as a result of wind-up, which allows normal tissue to become extremely sensitive to pressure in areas that are not identified as painful.

Neuroplasticity is the result of moderate to severe pain that lasts for more than 24 hours and occurs in the spinal area of the nervous system. With neuroplasticity, pain fiber growth is stimulated and the pain inhibition system is damaged, resulting in more intense pain that is widespread, lessening the ability of the body to stop the pain.

Peripheral sensitization can occur as a result of neuroplasticity. This creates a condition in which nonpainful touch and pressure become painful (ASPMN, 2010).

As we study and begin to understand the process and theory of pain transmission, more information about the process is discovered. As science expands its understanding of the pathophysiology of pain, more information will lead to a better understanding of the transmission process.

The Concept of Nociception

How is pain really felt? The concept of nociception can help us determine just how pain moves through the nervous system, and it can also provide us with ideas about how we can interfere with pain facilitation and about pain inhibition. Nociception is defined as the perception of pain by sensory pain receptors called nociceptors located in the periphery. In the theory of nociception, there are four stages, or levels, of pain transmission.

1. **Transduction.** A noxious stimuli converts energy into a nerve impulse, which is detected by sensory receptors called nociceptors.
2. **Transmission.** The neural pain signal moves from the periphery to the spinal cord and brain.
3. *Perception.* The pain impulse is transmitted to the higher areas of the brain, where it is identified as pain.

4. *Modulation-facilitating and inhibitory input.* Input from the brain either inhibits or facilitates the sensory transmission at the level of the spinal cord. Forms the brain modulates or influences the sensory transmission at the level of the spinal cord. (Berry, Covington, Dahl, Katz, & Miaskowski, 2006; D’Arcy, 2007)

The transmission of pain is basically the passing along of a pain stimulus from the peripheral nervous system into the central nervous system, where it is translated and recognized as pain. The afferent nerve fibers are the means of moving the stimulus along the neuronal pathways.

Nociception can come from *visceral organs,* where pain is identified as “crampy” or “gnawing,” or it can be *somatic,* from skin, muscles, bones, and joints, where pain is identified as “sharp.” There are several different types of receptors that can trigger a pain response:

- **Mechanoreceptors**—activated by pressure
- **Thermal receptors**—activated by heat or cold
- **Chemoreceptors**—activated by chemicals, such as inflammatory substances (ASPMN, 2010)

### Peripheral Pain Transmission

Pain can first be experienced by free nerve endings or nociceptors located in the periphery of the body. As a person cuts a hand or fractures an extremity, the pain stimulus is first perceived in the nerves closest to the injury. In order for a pain stimulus to be created, the sodium ions on the nerve fiber must depolarize, and this causes the pain stimulus to be initiated and passed along the neural circuitry. There are two main types of nerves that transmit pain impulses or stimuli:

- **A-delta fibers** are large nerve fibers covered in *myelin* that can transmit a nerve impulse rapidly. The pain transmitted on an A-delta fiber is easily localized and the patients may describe the pain as sharp or stabbing.
- **C fibers** are smaller and *unmyelinated,* and the pain impulse is conducted at a much slower rate. Pain that is produced by C fibers is identified by patients as achy or burning in nature (ASPMN, 2010).
Two primary substances can help facilitate the transmission of pain from the periphery. *Substance P* is a neurotransmitter secreted by the free nerve endings of C fibers whose function is to speed the transmission of the pain impulse. *Bradykinin* is a second type of neurotransmitter whose function is to participate in the inflammatory response and hyperalgesia (ASPMN, 2010). Nociception can stimulate both A-delta and C fibers for pain transmission.

Other substances that participate in the facilitation of pain include the following:

- **Histamine** is a substance released from mast cells produced in response to tissue trauma.
- **Serotonin** can be released from platelets and is produced in response to tissue trauma.
- **COX products (prostaglandin E\textsubscript{2} and thromboxane E\textsubscript{2})** act to sensitize and excite C fibers causing hyperexcitability.
- **Cytokines (interleukins and tumor necrosis factor)** can sensitize C fiber terminals and participate in the inflammatory and infection process involving mast cells.
- **Calcitonin gene-related peptide** (CGRP) is located at C fiber nerve endings and produces local cutaneous vasodilatation, plasma extravasation, and skin sensitization in collaboration with substance P production (ASPMN, 2010).

Once transduction takes place, the nerve impulse is passed through a synaptic junction from the peripheral nervous system to the central nervous system. This synaptic junction has various functions and secretes various substances. Some medications, for example *pregabalin*, act at the synaptic junction by blocking calcium channels. This in turn can reduce the amount of neuronal firing and decrease the passage of pain stimuli. The synapse is between the peripheral neuron into the central nervous system via the dorsal root ganglion.

**Central Nervous System Pain Transmission**

As the pain stimulus is passed from the peripheral nervous system into the central nervous system, the signal passes through the dorsal
root ganglion to a synaptic junction in the substantia gelatinosa located in the dorsal horn of the spinal cord. As the stimulus pushing the pain impulse forward overcomes any opposing or inhibiting forces, the “gate” is opened, allowing the pain impulse to proceed up the spinal cord to the limbic system and brain.

The opening of the gate is controlled by a summing of all the forces involved in the conduction of the pain impulse. If the facilitating forces, neural excitability, and pain-facilitating substances, such as substance P, predominate, the pain impulse is passed on. If pain inhibiting forces predominate, the signal is blocked and the gate does not open. If by chance the pain impulse is perceived as potentially life threatening, a reflex arc across the spinal cord will fire, causing an immediate response to protect the affected area (e.g., touching a hot surface causes the body to react by removing the hand from the hot surface). This event can take place before any central processing of the neural signal.

Centrally active pain-facilitating and inhibitory substances include the following:

**Facilitating**

- Substance P
- Glutamate—responsible for communication between the peripheral and central nervous systems. Also plays a role in activating the NMDA receptors
- Aspartate
- Cholecystokinin
- CGRP
- Nitric oxide

**Inhibitory**

- Dynorphin—an endogenous opioid
- Enkephalin
- Norepinephrine
- Serotonin
- Beta-endorphin—an endogenous opioid
- Gamma-aminobutyric acid (GABA) (ASPMN, 2010)
Also performing an inhibitory role are the opioid receptors located both presynaptically and postsynaptically that are available for binding opioid substances, such as morphine, and producing analgesia. Although there are opioid receptors located at other sites in the body, those that are located inside the spinal cord have the most information available about how they function.

As the pain impulse passes through the dorsal horn, it passes across the spine to the lateral spinothalamic tracts, which then allow the pain impulse to proceed up to the thalamus and limbic system, activating the emotions and memories associated with pain, and then to the cerebral cortex, where the pain impulse or stimulus is recognized as pain. Although this process seems complicated, the body can conduct a pain impulse in only milliseconds.

Within the limbic system, two pain substances, norepinephrine and serotonin, are active. Current drug therapies, such as tricyclic antidepressants and selective serotonin reuptake inhibitors (SSRIs), are aimed at this process and use the substances to reduce the amount of serotonin available to activate neuronal firing at synaptic junctions. The synaptic junctions have such varied functions that they not only are important for producing pain but are also critical sites for reducing pain by controlling the production of pain-facilitating substances and actions.

Once the pain stimulus reaches the cerebral cortex, the afferent pathway is completed. At that time, the efferent nerve fibers are used to pass the neuronal response identified as pain back to the periphery or affected area. Descending nerve fibers from the locus ceruleus and periaqueductal gray matter are activated, and the pain stimulus is passed back down the efferent pathway, where a response to the pain stimulus, such as moving the affected area away from the pain, is produced.

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**Clinical Pearl**

All patients with chronic pain should be assessed for depression and sleep disturbances when they are seen by their regular health care provider. Treating these conditions will help decrease the effects of pain that the patient is experiencing.
1. The Problem of Chronic Pain

## TYPES OF CHRONIC PAIN AND DIFFERENCES BETWEEN ACUTE AND CHRONIC PAIN

A patient who has chronic pain is very different from a patient who has acute pain. *Acute pain is pain that is the result of tissue injury, such as injury from trauma or surgery* (American Pain Society [APS], 2008). Patients expect that when their injury heals, the pain will resolve. As the pain decreases, the patient is able to resume their normal everyday activities and level of functioning. Acute pain serves the purpose of warning the person that an injury has occurred and appropriate action is needed (e.g., treatment or moving away from the source of the pain).

Chronic pain is a different life experience. *Chronic pain is pain that lasts beyond the normal healing period of 3 to 6 months* (APS, 2008). *It is the result of injury or potential tissue damage* (APS, 2008). Chronic, persistent pain has many different sources, and the pain that the patient complains of may be in several different areas of the body. Chronic, persistent pain may exist even though there is no detectable physical source for the pain (Marcus, 2000).

The average patient with chronic pain may rate his pain level at high intensity, yet be able to function at some level. This is confusing for health care providers who expect a patient who rates his/her pain intensity at 7 out of 10 to be showing signs of severe pain, such as grimacing, moaning, or guarding the painful area. The patient with chronic pain has learned to adapt both consciously and unconsciously. Functionality is a better measure of pain relief in patients with chronic pain (ASPMN, 2010; Marcus, 2000). Physiologically, the patient with chronic pain may not have increases in blood pressure or heart rate when they are experiencing their normal daily chronic pain. Discussing the different types of chronic pain will provide insight into the causative factors for the pain.

There are several different types of chronic pain. Exhibit 1.1 identifies the two main categories, nociceptive and neuropathic pain. Figure 1.1 further classifies chronic pain into mixed and visceral pain.
Exhibit 1.1

Differences Between Nociceptive and Neuropathic Pain

**Nociceptive**
—Produced by peripheral mechanoreceptors, thermoreceptors, and chemoreceptors.
—Serves to warn the body that injury has occurred.
—Pain is proportionate to receptor stimulus.

**Neuropathic**
—Caused by damage to the peripheral or central nervous system.
—May involve an inflammatory process that perpetuates the pain stimulus.
—Nociceptive input not required for pain to occur.
—Pain is of higher intensity and disproportionate to pain stimulus.

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**Figure 1.1** Pain classification. IBS = irritable bowel syndrome.
Nociceptive Pain

Nociceptive pain is the result of tissue damage, surgery, or injury. When patients have surgery or sprain an ankle, nociceptive pain is the result. Almost everybody has experienced some type of nociceptive pain.

The stimulus for nociceptive pain is generated from various sources and specialized sites located throughout the body. Activation of thermoreceptors (heat), mechanoreceptors (tissue injury, pressure), and chemoreceptors (chemical irritants) can all create nociceptive pain. This means that when a patient burns a hand, has a crush injury, or has an infection, a nociceptive pain stimulus is produced. The pain stimulus is then passed along the peripheral nervous system by a voltage-gated sodium channel that allows an influx of sodium ions into the neural cell, also creating an action potential that allows the stimulus to pass to the central nervous system. Once the pain stimulus reaches the spine, if the pain stimulus is sustained, the pain signal is transmitted past the dorsal horn of the spine, through the limbic system, and into the cerebral cortex. There the stimulus is recognized as pain and translated, and an appropriate response is provided (see Figure 1.2).

Nociceptive pain is often acute and the pain will diminish as healing occurs. This type of pain serves as a warning signal to the body, which can identify the injury and protect itself. Exceptions to the short-term nature of the pain are chronic degenerative conditions such as osteoarthritis or rheumatoid arthritis, which can produce long-term pain. Outside of chronic, incurable degenerative conditions such as arthritis, it is reasonable to expect that most acute, nociceptive pain will resolve once healing takes place.

For treatment, analgesics typically are quite effective in reducing the intensity of nociceptive pain, with a 2-point reduction on the Numeric Rating Scale (NRS) or 30% reduction in intensity considered a good response. Nonopioid analgesics, such as ibuprofen or acetaminophen, work well for mild and moderate pain, especially that which is aching in nature or related to an inflammatory process.
Brain processes the message and alerts the body of pain.

Nerves pick up the injury and send the message to the brain.

- Dashed line shows message flow from pain site to brain.
- Dotted line shows message flow from brain to pain site.

Injury occurs in the body.

**Figure 1.2** Pain stimulus process. *Source:* Used by permission of Anatomical Charts, Park Ridge, IL.
Opioids that work well for all types of pain are generally reserved for moderately severe and the most severe forms of pain. Examples of descriptors that patients use to report nociceptive pain include the following:

- Dull
- Gnawing
- Aching
- Throbbing

**Neuropathic Pain**

Neuropathic pain is defined as: “Pain initiated or caused by a primary lesion or dysfunction in the nervous system (peripheral or central) that disrupts impulse transmission and modulation of sensory input” (Treede et al., 2008). A neuropathic pain condition can occur alone, in combination with another neuropathic condition, or with nociceptive pain. Because neuropathic pain is the result of neural damage and not tissue injury, clinical examinations, radiographic studies, and blood studies may be normal, but the patient still has pain.

Sometimes, this pain is created by a sustained inflammatory response within the nerves. Inflammation activates many irritating chemicals, such as bradykinin, substance P, hydrogen ions, prostaglandins, histamine, and tumor necrosis factor (TNF), all known to worsen pain while changing the structure and function of affected and adjacent nerves. As a result, nerve cells become more permeable to calcium and sodium channels, resulting in neuronal hyperexcitability and leading to an exaggerated response to pain stimuli (Benarroch, 2007).

Many of the abnormal processes, including inflammation, that underlie neuropathic pain operate on cyclic mechanisms that continue to spread and intensify the pain over time. Because few medications currently exist to halt the cycles that cause and worsen this type of pain, treatment is often difficult. It is very important to rule out treatable causes of the neuropathy before a final diagnosis is made of neuropathic pain.
There are two types of neuropathic pain, depending on the source of the lesion or nerve damage. **Central neuropathic pain** arises from central nervous system syndromes, such as pain that follows a stroke, multiple sclerosis, or a spinal cord injury. **Peripheral neuropathic pain** is caused by disorders affecting peripheral nerves, including postherpetic neuralgia (PHN), painful diabetic neuropathy (PDN), postthoracotomy pain, HIV-related neuropathies, and chemotherapy-induced neuropathies. Some forms of neuropathic pain have both peripheral and central nerve malfunctions generating the pain, such as complex regional pain syndrome (CRPS) and phantom limb pain.

As part of the evaluation of the patient, the painful area of the body is examined for abnormal sensations. Some patients will have diminished sensations of touch in affected areas, whereas others will have a heightened sensitivity to light touch or to hot/cold temperature changes. If stimulation that is not usually painful hurts, such as a wisp of a cotton swab or alcohol wipe, the patient is said to have **allodynia**. If there is an exaggerated response to a normally mild pain, such as a pinprick, the patient has **hyperalgesia**. These findings should verify for the clinician that the nervous system is the source of the pain.

Patients with neuropathic pain will often describe their pain as:

- Burning
- Shooting
- Painful tingling
- Painful numbness
- Strange, but clearly uncomfortable, sensations termed “dysesthesias”

**SUMMARY**

Although pain is a very common patient complaint, there are distinct differences between acute pain and chronic, persistent pain. Health care providers treating patients with pain should be able to recognize the differences between pain types and treat the type of pain accordingly. Acute pain may respond to typical analgesics, whereas chronic pain conditions may require a combined medication and complementary techniques plan of care. Treating acute
pain effectively can help prevent the development of a chronic pain condition, such as CRPS, that can be much more difficult to treat.

Understanding how pain is produced in the body can help health care providers learn to identify the way it is being expressed physically by the patients. Patients will often describe pain as achy or sharp and knowing that different mechanisms and nerve fibers are producing this presentation can help the provider better identify the source of the pain.

The production of pain is a very sophisticated, complex process that can be difficult to understand. Learning the mechanism of acute pain production and the facilitators and inhibitory substances can help a health care provider understand why medications such as antidepressants and antiepileptic drugs can decrease pain. More information on medication mechanisms of action will be provided in the medication chapter.
Peter Allen is a 75-year-old friend of yours who lives in an assisted living facility. He is really pretty active and has been walking every night except for the last 6 weeks, when his knees began to really bother him. He fell last week while trying to get to the bathroom. He hit his elbow, fractured his wrist, and bruised his ribs and knees. When you stop in to see Peter, he tells you this is not the first time he has fallen, but it is the first time he has really injured himself.

Overall, Peter is pretty healthy with only hypertension, mild diabetes controlled with oral medications, and some osteoarthritis in both knees. Peter talks to you about the pain he is experiencing. He says,

This is horrible pain. Nothing I take seems to help. Every time I move my arm I could just scream—it hurts that bad. They ask me to rate my pain intensity, and I always have to say 8. Sometimes I think they really don’t believe me. When I’m not moving, it feels just like a toothache. I wonder why I fell? Recently my knees have been acting up, and I have this odd numbness in my feet that seems worse at night. It seems like I’m stumbling at times. The doctor gave me some pain pills, but I’m afraid to take them because I don’t want to sleep all the time, and I hear about all those people getting addicted to them. I can’t sleep either. There’s no position that’s comfortable for my arm. Is there any way I can manage this pain better?
Questions to Consider

1. What type of pain does Peter have? Does he have more than one?
2. Why is Peter’s new pain so severe? Is there anything in his current condition that would give an indication of why the pain intensity is so high?
3. How have Peter’s comorbidities affected his current pain complaint?
4. How has the chronic pain changed Peter’s ability to function, his mood, and potentially his lifestyle?

REFERENCES


**ADDITIONAL RESOURCES**

