Compact Clinical Guide to Acute Pain Management
An Evidence-Based Approach for Nurses
Yvonne D’Arcy, MS, CRNP, CNS

This book provides much-needed guidelines that are presented in an easy-to-use, systematic format for quick access to core concepts on acute pain management. It is designed to help busy practitioners accurately assess pain in a variety of patient populations, and select patient-appropriate medications and interventions to achieve optimal pain management for adult patients.

Intended for use in primary care, internal medicine, and acute- and long-term care settings, this book covers the topics of acute pain assessment, both pharmacologic and nonpharmacologic treatment options, current information from national guidelines, along with regional anesthesia techniques, patient-controlled analgesia, and epidural pain management.

KEY FEATURES:
• Offers important new perspective on combination use of pain scales to accurately predict individual pain management needs for more customized and effective management
• Delivers information on how to treat acute pain in hospitalized patients who also suffer from chronic pain and substance abuse
• Offers new information on opioid polymorphisms and their surprising effect on pain medication effectiveness
• Includes a special chapter on managing pain in difficult-to-treat patient populations

This is an essential reference for primary care providers in clinics, hospitals, specialty care, and critical care to assess pain in general populations and provide tips for performing pain assessment on patients with acute pain.

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I dedicate this book to my children, Rob, Lauren, and Leslee, who were an early part of my becoming a nurse and creating my practice as a pain management specialist.
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*Combined Thermometer Scale Inside Front Cover*
I was a nontraditional student when I returned to school for my BSN. I often wondered if I was making the right choice, but after finding myself after graduation on a large medical-surgical unit, I knew that I was always meant to be a nurse. I loved absolutely everything about being a nurse.

After an orthopedic trauma of my own for which I had multiple surgeries and many difficulties with pain management, I went looking for answers to questions about why my pain control had been so poor. As a med-surg nurse, I always felt I did a good job of treating pain in my patients. After my own experiences, I realized that I had missed a lot in treating my patients’ pain. I had a new mission: to improve pain management for hospitalized patients, not only for those patients I cared for but for all patients who had pain. I realized that perhaps I was meant to help teach nurses how to better manage pain in their patients by drawing on my own experiences.

As part of my master’s program, I developed an acute pain resource nurse program in 1995. The curriculum covered all aspects of acute pain. It was a start to educating nurses in acute pain management, but the program reached only a select few nurses who attended the program.

This book in the Compact Clinical Guide series is aimed at reaching large numbers of practitioners who see many patients who are having all types of acute pain. It provides some basic concepts such as pain transmission and pain medications, and then some specific pain types such as sickle cell and abdominal pain. There are short case studies at the end of each chapter that help to illustrate the
concepts of the chapter. All of the information in the book relies heavily on national guidelines and evidence-based materials.

I have worked on several acute pain services over the last 15 years. I hope that by sharing my experience with patients who have acute pain, you will be better able to meet the needs of your own patients with acute pain. I have tried to provide the type of clinical guide that I would have liked to have when I first started my practice. I wish you great success with managing pain in your patients and I hope you will find this guide a useful resource.

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Overview of Acute Pain
OVERVIEW OF ACUTE PAIN

The problem of pain is universal. There are relatively few people who have never experienced pain. Those who lack the ability to feel pain because of genetic defects are rare. Most people identify pain as a negative experience and look for a quick way to relieve the pain. In the United States, medicine chests are filled with home remedies and over-the-counter pain relievers. Advertisers supply the American public with examples of products to treat minor sprains, strains, headaches, and muscle aches on television and over the Internet.

Many patients will try to self-treat acute pain that is minor. For more severe unrelieved acute pain needs, patients seek help from health care providers at some type of clinic, emergency room, or hospital.

For all types of health care providers (e.g., nurses, physicians, physical therapists, and pharmacists), the need to know how to assess acute pain, choose an effective treatment, and provide information on effective pain management is critical. Usually, the treatment of mild level acute pain involves some type of medication to relieve pain combined with cold packs, wraps, casts, or slings. For more severe level acute pain from trauma or surgery, more complex techniques are needed, such as intravenous pain medications, patient-controlled analgesia (PCA) or epidural analgesia, or regional analgesic methods, such
as nerve blocks using local anesthetics. No matter what type of acute pain a patient has, effective treatment will help ease the pain, decrease the patient’s anxiety about getting good pain relief, and decrease the potential for having the pain develop into a chronic or persistent pain condition that is harder to treat.

**PREVALENCE OF ACUTE PAIN**

It is difficult to determine just what the prevalence of acute pain is because it can be treated in many different places, and it can be the result of many different conditions. From the data that we do have, we know that the prevalence of acute pain is high and that pain can come from various different sources. Whether acute pain is the result of trauma, surgery, or injury, the patient will need to cope with the pain until it resolves after healing takes place. During that time, the patient can experience effects on his or her lifestyle that may limit activity, require analgesic use, and require a period of rehabilitation. Emotional responses to undertreated pain, such as anxiety or fear of pain, can also complicate pain relief and cause the pain to be perceived as more intense, as having a negative impact on function, and as being more invasive into personal lives.

One estimation of the prevalence of acute postoperative pain is based on the finding that out of the 73 million surgeries that took place in 2003, approximately 80% of patients experienced postoperative pain and that 86% of them reported experiencing pain that was either moderate, severe, or extreme (Apfelbaum, Chen, Mehta, & Tong, 2003). Prior to surgery, 59% of these patients indicated that postoperative pain was a concern (Apfelbaum et al., 2003). Consider that 70% of all current surgeries are performed in ambulatory care centers, where quick and effective pain management is a necessity (Apfelbaum et al., 2003). The use of new techniques, adequate medications, and appropriate assessment of medication efficacy for each patient is essential to providing the type of pain relief that patients seek following surgery.

In emergency departments, acute pain is a common complaint. In a midwestern hospital emergency department, a chart review for 1,665 visits during a 7-day period revealed that 61% of patient charts
had pain documented in some area of the chart (Cordell et al., 2002). In 52% of the pain charts, pain was documented as the chief complaint (Cordell et al., 2002). In these cases where the patient is seen for a very brief period of time, efficient and effective pain management is a must. Emergency department (ED) care providers of all types need to understand medications for pain, pain assessment, and patient education about pain management. Making effective use of the short time period during which patients are seen in the ED will help provide effective pain relief and prevent readmission.

Good pain management is needed for acute care patients because of the short length of stay most patients with acute pain experience. This is the basis for using the best pain regimen prior to discharge. Ensuring that patients understand how to take medication for pain can help prevent readmission for pain. Readmissions for uncontrolled pain are the most common when compared with all other non pain reasons for readmission in the first week after surgery (Polomano, Dunwoody, Krenzischek, & Rathmell, 2008). The cost of these readmissions totals $1,869 ± $4.553 using mean charges (Coley, Williams, DaPos, Chen, & Smith, 2002). From same-day surgery centers, pain accounts for 36% of all unanticipated admissions and readmissions, with 33% of the patients having had an orthopedic procedure (Coley et al., 2002).

From hospital settings to ED and surgery centers, unrelieved pain is fairly common. Undertreatment can be costly, resulting in readmission for pain treatment or repeated office visits. Nurse practitioners and other health care providers can play an important role in reversing this process by becoming more fully acquainted with appropriate pain assessment tools, learning to use pain medications more effectively, and selecting the appropriate medication for the pain complaint to provide the best opportunity for adequate pain relief.

DIFFERENCES BETWEEN ACUTE AND CHRONIC PAIN

There are considerable differences between acute pain and chronic pain. Differences occur in the time frame for the pain complaint, but perhaps more importantly, in the pathophysiology of the pain...
and the appropriate choice of treatments that can deliver the most effective relief for that pain. Learning the differences between the types of pain can help the health care provider choose the best methods for treating pain.

**Acute Pain**

Acute pain is very different from chronic, persistent pain. Acute pain usually has a short duration and has an identifiable cause, such as trauma, injury, or surgery (American Pain Society [APS], 2008; American Society for Pain Management Nursing [ASPMN], 2010). The source of acute pain is tissue damage, and the sensation of pain warns the body that it has been injured. The duration of acute pain is expected by the patient to be short-term and to resolve as the injury heals.

Anxiety is common with acute pain. There may also be some sympathetic nervous system activity, such as increases in heart rate or respiratory rate. Acute pain can also cause diaphoresis, nausea, and vomiting related to the pain, and/or pallor. Once the pain is controlled, the patient can relax and additional signs and symptoms may lessen or resolve.

**Clinical Pearl**

Do not base your assumption on the presence of pain by observing changes in blood pressure or increased heart rate. These may be caused by other responses, such as anxiety or fear, and may not be a result of the pain. Patients with chronic pain accommodate the continued pain and do not typically experience these physiological changes.

**Chronic Pain**

Chronic pain, on the other hand, is pain that lasts beyond the normal healing period, which is usually longer than 3 to 6 months (APS, 2008). Chronic pain has no purpose and can exist without visible injury. Some patients with chronic pain can relate a specific incident, such as surgery, a fall, or injury, to the onset of the pain. Other
patients just wake up with pain that continues despite all the best efforts at treatment. Chronic pain can also be the result of cancer, related to the spread of the disease, tumor growth, or side effects of chemotherapy. Chronic pain does not need tissue injury to exist, and physical damage may not be evident on X-ray, magnetic resonance imaging (MRI), or computed tomography (CT); nonetheless, the pain persists.

Patients with chronic pain who are experiencing acute pain may have no visible response to the new pain. Because chronic pain is a daily phenomenon, most patients accommodate to the pain experiences, do not expect to be pain free, and will tolerate high levels of pain on a daily basis. Patients with chronic pain, such as from cancer, will not often demonstrate signs of sympathetic nervous system (SNS) activation, such as increased pulse or heart rate (APS, 2008).

The lack of response to acute pain in patients with chronic, persistent pain can be confusing for health care providers. Many providers expect to see high levels of pain behaviors, such as guarding, moaning, crying, or withdrawing, from patients when the pain is rated as severe. However, for the patient with chronic pain who is experiencing acute pain, there may be few, if any, indications of the pain, let alone any outward signs consistent with high levels of pain. For patients with chronic pain who are experiencing acute pain, the same type of pain assessment is performed in which the patient is asked to rate the pain using the Numeric Rating Scale (NRS)—the 11-point Likert scale from 0 to 10—where 0 is no pain and 10 is the worst pain imaginable. The number is then used to choose an intervention to treat the pain complaint. More information on assessing pain and on acute pain in patients with chronic pain will be provided in later chapters of the book.

One very important aspect of treating acute pain is the concept that untreated or undertreated acute pain can progress to a chronic pain condition that is much more difficult to treat (APS, 2008; Macintyre, Scott, Schug, Visser, & Walker, 2005). One perfect example of this is complex regional pain syndrome (CRPS), formerly called reflex sympathetic dystrophy syndrome (RSDS).
Patients who are candidates for the development of CRPS include the following:

- Patients with surgical nerve injury or trauma.
- Patients with crush-type injuries with prolonged, persistent pain.
- Patients who report high levels of unrelieved pain after acute injury or surgery, despite changes in treatment.

As pain stimuli continuously present to peripheral neurons in a constant feedback loop, the pain experienced can be converted to a centrally controlled pain condition, such as CRPS, that is difficult to treat and is less responsive to ordinary pain medications. There are two types of CRPS: Type I, which corresponds to the older RSDS category and occurs without a nerve lesion; and Type II, which was formerly called causalgia, and has a detectable nerve lesion (Rowbotham, 1998).

For a pain condition to be diagnosed as CRPS, some of the following criteria must be present:

- Regional pain—usually in the upper or lower extremities
- Sensory motor changes—sudomotor abnormalities
- Changes to the skin
- Thickening of the nails
- Hair loss in the affected area

Although CRPS is considered to be a chronic condition, it can develop in acute care patients as they undergo repeated surgeries, frequent débridements, or other types of procedures that cause continuous injury to tissue or nerves at the same site. Some of the telltale complaints of patients with developing CRPS include the sensation of burning pain; painful numbness; sensitivity to temperature changes, cold especially; and pain out of proportion to the injury. The recommendation from pain management specialists is to treat acute pain aggressively to limit the effect on the individual and to decrease the potential for the development of CRPS (D’Arcy, 2007; Macintyre et al., 2005).
The acute care nurse practitioner learning to identify the signs and symptoms of developing neuropathic pain such as CRPS can make an immeasurable difference to the final outcome for the patient. If the condition is identified and aggressive pain management occurs quickly, the chance that the pain can be minimized and treated effectively is dramatically increased. Use of medications designed for treating neuropathic pain, such as antiepileptic medications or antidepressants, make the difference in being able to return to a normal lifestyle after the injury or surgery as opposed to dealing with a chronic pain condition for the remainder of the patient’s life.

**Neuropathic Pain**

CRPS is just one type of pain that is called neuropathic pain—pain that is caused by an injury to a peripheral nerve or the central nervous system, or CNS (ASPMN, 2010; Staats et al., 2004). Neuropathic pain is a type of chronic pain, but it may have its source in an acute pain condition. Some of the causes of neuropathic pain include the following:

- Nerve damage from surgery, such as an entrapment syndrome, post-thoracotomy pain, postmastectomy pain syndrome, phantom limb or breast pain, or posthysterectomy pain
- Neuropathy from a disease or injury, such as postherpetic neuralgia, diabetes, or CRPS
- Neuropathic pain as the result of treatments, such as chemotherapy, especially when using such agents as vinca alkaloid or other chemotherapeutic agents
- Centrally originating pain syndromes, such as poststroke pain, fibromyalgia, and spinal cord injury pain (List taken from Berry, Covington, Dahl, Katz, & Miaskowski, 2006; D’Arcy, 2007)

Although some acute pain conditions may lead to chronic pain syndrome, if the condition has a neuropathic component, it is by far more difficult to treat, and the suffering the patient endures is much more destructive to a patient’s quality of life, is more intense, and is long term. The key to differentiating neuropathic pain from a less
complex chronic pain is the use of descriptors such as burning, painful tingling, or numbness. More information on neuropathic pain descriptors will be provided in the chapter on pain assessment.

**PAIN TRANSMISSION**

**Pain Theories**

Some of the mechanisms of pain transmission are different for acute pain and chronic pain (Figure 1.1). The onset of acute pain is sudden and can provoke a fight or flight type of response, with adrenaline release that will subside rapidly. On the other hand, chronic pain is long term and can become more complex over time, and advanced pain facilitating responses such as the activation of \( N \)-methyl-d-aspartic acid (NMDA) receptors takes place. Many pharmaceutical companies aim the action of their medications at a specific site in the pain transmission process. For example, interfering with serotonin at the synaptic junction can help reduce the amount of pain-facilitating substances available to create or continue the pain stimulus.

There are multiple theories 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 (Cervero, 2005). Using 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 is a pain stimulus caused by a burn or trauma that travels from the site of the burn or trauma to the brain, where the brain recognizes the stimulus as pain. The resulting response would be for the body to withdraw from the source of pain (e.g., removing the hand from a fire or otherwise pulling the affected area away from the source of the pain). 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 psychologic and emotional component of pain is an important aspect of the condition. Older theories, such as those espoused by Pavlov, considered
3 Brain processes the message and alerts the body of pain.

2 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 going from brain to pain site.

Injury occurs in the body.

Figure 1.1 Pain transmission—Exemplar. Source: Used by permission of Anatomical Charts, Park Ridge, IL.
pain to be a learned response that was affected by cultural and learned behaviors that could be offset by operant conditioning (FORDYCE, 1976; PAVLOV, 1927). A contemporary theorist, TURK, described pain as a multidimensional experience and proposed that the patient, not the health care practitioner, is the specialist on his or her pain (TURK, 2003). This theory empowers patients to become an active participant in pain treatment and helps patients to diminish negative behaviors and increase positive reinforcing behaviors (ASPMN, 2010).

Perhaps the most well-known theory of pain transmission is the gate control theory developed by MEZACK and WALL in 1965 (ASPMN, 2010). In this theory, the psychological and physiologic aspects of pain transmission are combined. Quite simply, the gate control theory states that a pain stimulus can be of significant intensity to “open” a neuronal gate, allowing the pain stimulus to proceed through the nervous system to the brain to create a sensation that can be identified as pain.

The actual steps in pain transmission using 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 CNS.
- 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. These include the following:

- The central control processes and central intensity processes 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 reaches critical levels, the T-cell system is activated, which creates a link between the brain and the body to link 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 the 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 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 a 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 and lessening the ability of the body to stop pain. Peripheral sensitization can occur as a result of neuroplasticity. This condition creates another condition in which nonpainful touch and pressure become painful (ASPMN, 2010; Berry et al., 2006; Yaksh, 2005).

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 to determine just how pain moves through the nervous system, and it can also provide us with ideas about interference with pain facilitation
and inhibition. *Nociception* is defined as the perception of pain by sensory pain receptors called *nociceptors* located in the periphery (Sorkin, 2005). The theory of nociception suggests that there are four stages, or levels, of pain transmission.

**Stage 1. Transduction**—A noxious stimuli converts energy into a nerve impulse, which is detected by sensory receptors called nociceptors.

**Stage 2. Transmission**—The neural pain signal moves from the periphery to the spinal cord and brain.

**Stage 3. Perception**—The pain impulse is transmitted to the higher areas of the brain, where it is identified as pain.

**Stage 4. Modulation**—Facilitation and inhibitory input form the brain modulates or influences the sensory transmission at the level of the spinal cord (Berry et al., 2006; D’Arcy, 2007).

The transmission of pain is the passing along of a pain stimulus from the peripheral nervous system (PNS) into the CNS, where it is translated and recognized as pain. The afferent nerve fibers move the stimulus along the neuronal pathways.

Nociception can come from various locations: *visceral*, where pain is identified as crampy or gnawing pain; or *somatic*, pain from skin, muscles, bones, and joints identified by patients as sharp pain (Berry et al., 2006). The following 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; for example, 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. For a pain stimulus to be created, the sodium ions on the nerve fiber must depolarize, which causes the pain stimulus to be produced and passed along the neural circuitry.
The following 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 (ASPMN, 2010; Sorkin, 2005).

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, the function of which is to speed the transmission of the pain impulse. **Bradykinin** is a second type of neurotransmitter, the function of which 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 \(E_2\) and thromboxane \(E_2\)) act to sensitize and excite C fibers, causing hyperexcitability.

- **Cytokines** (interleukins and tumor necrosis factor [TNF]) 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 sanitization in collaboration with substance P production. (ASPMN, 2010; Berry et al., 2006; Sorkin, 2005)

Once transduction takes place, the nerve impulse is passed through a synaptic junction from the PNS to the CNS. This synaptic junction has various functions and secretes substances. Some medications (e.g., 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 CNS via the dorsal root ganglion.

Central Nervous System Pain Transmission

As the pain stimulus is passed from the PNS into the CNS, 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 retract and remove the hand from the hot surface). This event can take place before any central processing of the neural signal begins (Cervero, 2005).

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

Facilitating:
- Substance P
- Glutamate—responsible for the communication between the peripheral and central nervous system (Rowbotham, Kidd, & Porecca, 2006); also plays a role in activating the NMDA receptors (Mersky, Loeser, & Dubner, 2005)
- Aspartate
- Cholecystokinin
- CGRP
- Nitric oxide
Inhibitory:

- Dynorphin—an endogenous opioid
- Enkephalin
- Norepinephrine
- Serotonin
- B-endorphin—an endogenous opioid
- Gamma-aminobutyric acid (GABA) (ASPMN, 2010; Sorkin, 2005)

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 are the most well understood in how they function.

As the pain impulse passes through the dorsal horn of the spinal cord, it passes across the spine to the lateral spinothalamic tracts. The pain impulse proceeds up the spinal cord to the thalamus and limbic system. This activates the emotions and memories associated with pain, which are then passed to the cerebral cortex where the pain impulse or stimulus is recognized as pain. Although this process seems complicated, the body can transmit a pain impulse in milliseconds.

Two pain substances are active within the limbic system: norepinephrine and serotonin. Current drug therapies, such as tricyclic antidepressants and selective serotonin reuptake inhibitors (SSRIs), are aimed at reducing the amount of serotonin available to activate neuronal firing at synaptic junctions. The synaptic junctions have various functions. They not only produce pain impulses but also can influence pain by reducing the amount of pain-facilitating substances.

Once the pain stimulus reaches the cerebral cortex, the ascending pathway is completed. Once the pain is identified, the response is passed down along the descending neural pathways to the periphery. The descending impulse uses nerve fibers from the locus coeruleus and periaqueductal gray matter to pass the pain impulse downward so that the response to move the hand away from a hot surface, for example, occurs. The overall pain transmission relies on both the neural pathways and the neurotransmitters.
18 1. The Problem of Acute Pain

SUMMARY

Although pain is a very common patient complaint, there are distinct differences between acute and chronic, or 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 the typical analgesic, 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 expressed physically by 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 facilitating and inhibitory substances can help a health care provider understand why medications such as antidepressants and antiepileptic medication can decrease pain. More information on medication mechanisms of action will be provided in the medication chapter.
This case study indicates the type of patient scenarios that will be included in each chapter. Some of the information needed to answer the questions in this presentation can be found in the assessment and medication chapters.

Peggy Smith is a 65-year-old patient who was hanging Christmas lights on the front of her home when she fell from the ladder she was on. She landed on her left hip and now has significant pain with an external rotation to her left lower extremity. She was taken to the hospital by an ambulance, where she is diagnosed with a hip fracture and prepared for surgery to correct the deformity.

In the ED, Peggy is complaining of significant pain at 8 out of 10 level. When the triage nurse takes her history, Peggy relates a history of chronic, persistent pain in her low back from a motor vehicle accident that occurred 6 years ago. She takes four oxycodone with acetaminophen tablets every day for her back pain and occasionally receives an epidural steroid injection for a radicular pain that goes down her left leg. She drinks a glass of wine every day at dinner and occasionally will have alcohol when she is out with friends. How will you treat Peggy’s pain?
1. Does the fact that Peggy has chronic pain affect her new acute pain?
2. Because Peggy takes opioid medication every day, will her pain medication needs be different? If so, how will you adjust the doses of medication to accommodate the acute pain?
3. Is Peggy addicted to her usual pain medications?
4. What role does Peggy’s alcohol use play in her management needs?
5. What is the best type of pain management for Peggy in the postoperative time period? Does her age affect your decision?

REFERENCES