

MANAGING PAIN IN THE OLDER ADULT



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EDITOR


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This book is dedicated to the special men in my life, Mark, Brian,
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Preface

The older adult population is the fastest-growing segment of our society. It is estimated that by 2030, 22% of the population will be over 65 years of age. The majority of that population will have at least one chronic health condition. Over 70% will experience some form of persistent pain. The challenge for health care providers is to understand the mechanisms of acute and chronic pain as well as the physiologic changes associated with aging.

Although our knowledge of aging and pain continues to grow daily, both continue to challenge health care providers. The older population poses unique health care issues and challenges. Persistent pain is under-recognized and under-treated. Providers are often hesitant to treat pain in older adults due to adverse effects and interactions of many of the treatment options. A deeper understanding of common pain syndromes and treatments for older adults will provide a more comprehensive approach to their persistent pain.

This text is written as a reference for health care providers seeking to expand their knowledge of pain in the older population. The intent of the book is to provide a user-friendly approach to examine various treatment options as well as some of the cautions that need to be heeded in treating pain in the older population. The better the understanding of pain in older cognitive and cognitively impaired adults will result in better pain control for this population.

The book is organized in two sections. The first section explores background information on pain in the elderly and common pain syndromes. Special consideration for assessing pain and pain-related behaviors in the older population is included in this section. A chapter related to sleep and behavioral aspects of pain in older adults provides insight into environmental influences on pain and pain perception.

The second section of this book is dedicated to the management of pain in the elderly. A multimodality approach to pain management is emphasized. Most chronic conditions, pain being no exception, are best treated with

multiple therapies, as one method of treatment is limiting. The chapters on physical therapy, interventions, and complementary therapies provide alternatives for older adults in managing their pain. Special considerations for use of pharmacotherapeutic agents with older adults are provided in a separate chapter. Improving mobility and function in patients with persistent pain is the focus of another chapter.

Managing Pain in the Older Adult is unique in that the focus of the text centers specifically on the needs of the older person suffering from pain. Improving function and quality of life are priorities in treating this population. This text is targeted toward primary care providers, as they are the health professionals that older adults feel most comfortable with in discussing their health concerns. Early recognition of pain syndromes in the elderly can lead to earlier treatment and control of pain, thus decreasing the development of undesired pain-related behaviors or co-morbidities.

Michaelene P. Jansen

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SECTION I

Background Information

CHAPTER 1

Pain in Older Adults

Michaelene P. Jansen

Pain has been defined by the International Association for the Study of Pain as an unpleasant sensory and emotional experience associated with potential or actual tissue damage. This is the most widely used definition when describing the concept of pain. Pain accompanies many health conditions across all ages and presents a challenge for health care providers. Pain can be acute or chronic in nature. Pain is considered persistent or chronic if it occurs for three or more months. The American Geriatric Society (AGS) adopted the term *persistent pain* in lieu of chronic pain to promote a more positive perception of ongoing pain. Table 1.1 describes attributes associated with acute and persistent pain. The purpose of this text, and particularly this chapter, is to explore the concept of pain and its unique characteristics in the older population.

The older adult population is the fastest-growing segment in our society. Approximately 36 million Americans, or 12% of the population, are over 65 years of age (Federal Interagency Forum on Age-Related Statistics, 2004). Between 2010 and 2030, the older adult population is expected to grow by 75% to over 69 million. The greatest increase will be seen in the over-85-year-old population. It is estimated that the over-85-year-old population will increase to 18.2 million by 2050 (Administration on Aging, 2006).

The older population is likely to suffer from chronic illnesses such as arthritis, diabetes, joint disorders, and other conditions that elicit a pain response. Approximately 25%–50% of community-dwelling older adults experience persistent

TABLE 1.1 Differences Between Acute and Persistent Pain

Acute Pain	Persistent Pain
Imminent or actual tissue damage	No biological advantage
Damage to tissue limited	Functional capacity reduced
Reflex and behavioral response	Maladaptive responses common
Immobilization facilitates healing	Comorbidities such as depression, anxiety, sleep disturbances

pain (American Geriatric Society (AGS), 2002; Blyth, March, Barnabie, Jorm, Williamson, & Cousins, 2001; Mantyselka, Kumpusalo, Ahonen, Kumpusalo, Kauhanen, et al., 2001). Given the chronic and frail state of residents in long-term care facilities, it is not surprising that persistent pain occurs in 45%–80% of these residents (AGS, 2002; Ferrell, 1995). These statistics emphasize the need for understanding how pain manifests itself in older adults.

The American Geriatric Society has recognized this need and has developed guidelines related to persistent pain in older persons (AGS, 2002). Persistent pain is unfortunately under-treated in the older population for a wide variety of reasons. Consequences of untreated persistent pain include depression, anxiety, social isolation, impaired sleep, and impaired mobility. Untreated pain can lead to widespread consequences in the elderly, increasing health care utilization and costs (Ferrell, 1991). Muscle deconditioning, abnormalities in gait, falls, slow recovery, cognitive impairments, and malnutrition either contribute to or become worse with pain. Health care providers need to understand the comprehensive nature of pain in older adults (Gloth, 2001). This chapter begins this process of understanding pain in older adults by discussing the physiological mechanisms of pain in the elderly.

AGE-RELATED CHANGES IN PERCEPTION OF PAIN

Older persons often have more than one source of pain. Given the multiple causes of pain, it is often difficult to localize or target the pain source. The high prevalence of dementia, sensory impairments, compounds the problem. There is no consensus on whether perception and sensitivity of pain change with aging (Gibson & Helme, 2001). The American Geriatric Society panel concluded that no age-related changes are clinically significant in terms of perception of pain (AGS, 2002). There is a lack of studies however, in older adults, especially over age 75, examining sensitivities to pain and pain treatments. Some evidence supports

the theory that good coping strategies in older adults lead to less psychological distress associated with pain (AGS, 2002). One animal study suggests that age-related changes in perception of nociceptive pain are curvilinear, and age does need to be considered in treating pain (Finkel, Besch, Hergen, Kakarenka, Pohida, et al., 2006). Another animal study speculates that neuroimmune responses at the site of injury are developmentally regulated and less likely to produce persistent pain when injury occurs at a young age (Ririe & Eisenach, 2006).

There are multiple variables that contribute to the perception of pain in the elderly, making it difficult to determine what influences are most important. One small study examined acute and persistent pain in older adults and the influence of cognition on pain perception (Schuler, Njoo, Hetseramann, Osler, & Hauer, 2004). They found that perception of pain was independent of cognition, although cognitive impairment affected the ability to localize pain. Older individuals with chronic pain tend to use more pain descriptors, use more analgesics, and have more disability than older patients with acute pain (Schuler et al., 2004). Physiological adaptation to persistent pain is compromised because other coexisting health conditions decrease resiliency and functionality (Ebener, 1999). Those with persistent pain often have more difficulty falling asleep and exhibit more depression and anxiety.

PHYSIOLOGY OF PAIN

The physiology of pain pathways has been widely studied, and ongoing clinical and laboratory studies continue to expand our knowledge of pain. One of the most cited theories of pain was proposed by Melzack and Wall in 1965 and is widely known as the Gate Control Theory of Pain. Their original hypothesis and framework proposed that T cells (now known as wide dynamic range neurons) carry impulses to cortical centers. These wide dynamic range neurons (WDR) could be excited by large-diameter fibers and inhibited by interneurons in the dorsal horn of the spinal cord. Although the theory explains some of the mechanisms associated with the pain process, it does not explain all of them. Pain is a complex process that involves memory, expectations, and emotions (Ferrell, 1991). Although the biopsychosocial model of pain is widely accepted, Goldberg (2007) proposes that the old nociceptive or Cartesian concept of persistent pain developed during the eighteenth century be reexamined.

The following discussion will review the general physiology of pain transmission. The reader is referred to physiology textbooks if a more in-depth and detailed physiological analysis of pain transmission is desired. At the present time there is not sufficient evidence that demonstrates that pain transmission

changes significantly with aging. The main physiological change related to aging that has been documented is that A-delta fiber function decreases with aging. C fiber function remains stable, although there is some belief that older adults preferentially rely on C-fiber input.

Terms

There are several terms that need to be defined when discussing pain physiology. These terms are used frequently when describing mechanisms of pain pathways and may be helpful to review. A summary of these terms is listed in Table 1.2. Nociception and pain are often used interchangeably, but their distinction should be noted. Nociception is the reception of input into the central nervous system by sensory receptors known as nociceptors. Nociceptors provide information about injury to tissues. Not all information delivered through this system will be perceived as painful. Pain is the perception of an adverse sensation arising from a specific area of the body. It is the cognitive and subjective nature of pain that makes it difficult to manage clinically.

There are three main types of afferent nerves that are involved in pain transmission based on structure, degree of myelination, and function. *A-beta fibers* transmit sensory information regarding touch, vibration, and hair deflection. These fibers are large-diameter fibers that are myelinated, meaning that transmission along these nerves is rapid. *A-delta fibers* respond to noxious mechanical stimulation. These fibers are small-diametered, myelinated fibers and have slower conduction velocity compared to the A-beta fibers. *C polymodal nociceptors* originate at deep receptors such as ligaments, muscles, and connective tissues. These fibers are the smallest and slowest of the fibers and require greater stimulation to transmit pain impulses.

There are two major types of pain sensations, nociceptive pain and neuropathic pain. Nociceptive pain occurs from activation of free nerve endings in the skin or deeper tissues. These pain receptors, known as nociceptors, are activated by mechanical, thermal, or chemical stimuli. Nociceptive pain can be somatic or visceral in nature. Neuropathic pain arises from abnormal function of the nervous system. Partial or complete damage to nerve fibers can produce subtle abnormalities such as altered temperature or unpleasant sensation. The physiology of these two pain mechanisms will be discussed separately.

Neuropathic Pain

Neuropathic pain is very common in older adults (Ahmad & Goucke, 2002). There are many causes of neuropathic pain, often related to degeneration or

TABLE 1.2 Pain-Related Terms

Term	Definition	Comments
Nociceptive Pain	Stimulation of pain receptors (nociceptors) from visceral or somatic areas. Often described as a sharp, lancinating pain.	Arises from inflammation, mechanical deformation or ongoing injury. Responds well to analgesics and nonpharmacologic strategies.
Nociceptor	Pain receptor that is stimulated by an injury.	
Neuropathic Pain	Involves the peripheral or central nervous system. Often described as a burning, aching type pain.	Not relieved by common analgesics, but neuroleptic and antidepressant medications are often helpful.
Allodynia	Painful response to nonpainful stimuli. Activated by sensitized mechanoreceptors.	Light touch or clothing can trigger this response.
Hyperalgesia	Increased response to painful stimuli. Activated by sensitized polymodal nociceptors.	Appears to an observer as an overreaction to a painful stimulus.
Neuroplasticity	Organizational changes that develop in the brain as a result of experience.	Some experts contribute the concept of neuroplasticity to perception of neuropathic pain.
Interneuron	Neuron that communicates only with other neurons.	Interneurons are defined in terms of activity in peripheral and central nervous system.
Afferent nerve	Nerve that carries impulses toward the central nervous system.	Pain is transmitted to the dorsal horn of the spinal cord via afferent nerves.
Efferent nerve	Nerve that transmits impulses from the central nervous system toward the peripheral nervous system.	A motor neuron is an example of an efferent nerve.
Axon	Extension of a nerve cell that transmits impulses away from the cell body and branches as it terminates.	Axons synapse at its termination.
Lamina	Refers to a thin layer in the dorsal horn of the spinal column.	First order neurons terminate in the lamina I–X of the dorsal horn.

TABLE 1.3 Common Neuropathic Pain Syndromes in Older Adults

Central post-stroke
Diabetic neuropathy
Postherpetic neuralgia
Phantom limb pain
Intracostal neuralgia following thoracotomy
Ilio-inguinal neuralgia following hernia repair
Radicular spine pain
Trigeminal neuralgia

age-related changes. Table 1.3 provides examples of neuropathic conditions found in older adults. The list is not inclusive. The physiology underlying neuropathic pain involves injury to the nerve that results initially in damage or death of the axon but then develops axonal sprouting forming a neuroma (Yaksh, 2005). Neuromas are believed to cause spontaneous afferent activity. Injury to the nerve can cause increased dorsal horn excitability, altered inhibitory control, and reorganization of nonneuronal cells. An individual suffering from neuropathic pain may experience hyperalgesia or allodynia. Hyperalgesia is increased pain sensation, whereas allodynia is pain sensation with non-noxious stimuli. The four most common types of neuropathic pain are direct stimulation of pain-sensitive neurons, automatic firing of damaged nerves, deafferentation, and sympathetically mediated pain (Belgrade, 1999).

Direct Stimulation of Pain-Sensitive Neurons

Neuropathic pain can be caused by direct stimulation of C-nociceptors. Stimulation can be in response to mechanical stretching, compression, or chemical mediators. A tumor or mass compressing a nerve complex or spinal cord will be perceived in the area of nerve distribution for those fibers. Release of chemical mediators secondary to the inflammatory response in disc herniation, for example, can irritate adjacent nerve roots.

Automatic Firing of Damaged Nerves

Nerves that are damaged due to disease or injury can result in spontaneous impulse firing from the site of injury or along the damaged nerve. This type of nerve pain is seen in patients with large-fiber diabetic neuropathy, chemotherapy,

pesticides, or any type of unrelenting neuropathic pain. Neuropathic pain resulting from automatic firing of damaged nerves can be described as lancinating, stabbing, or shooting pain. When multiple nerve fibers are affected and fire asynchronously, the pain will be described as a continuous burning-type pain (Belgrade, 1999).

Deafferentation

Pain transmission follows an upward route from the area of injury or damage to the spinal cord, brain stem, and cortex, relaying the impulse from level-ordered neurons. Interruption at any level of the ordered neurons can result in irritability and aberrant firing of nerves. Examples of this type of neuropathic pain include phantom limb pain, diabetic neuropathy, post-herpetic neuropathy, or peripheral nerve trauma. Central post-stroke syndrome is an example of ongoing neuropathic pain that is experienced at one site but is generated at the infarct site further along the pain transmission route.

Sympathetic Mediated Pain

Sympathetic mediated pain refers to autonomic nervous system activity that occurs with pain stimulation. Sympathetic response may be triggered initially by the inflammatory response. The sympathetic activity can continue in a regional versus a dermatomal response area. The sympathetic nerves release norepinephrine, which can further stimulate C polymodal nociceptors and subsequently further stimulate sympathetic mediated pain. The person experiences diaphoresis, skin temperature changes, and altered peripheral circulation along with persistent neuropathic pain. As the sympathetic cycle persists, chronic regional pain syndrome (CRPS), previously known as reflex sympathetic dystrophy (RSD), can develop.

Other Mechanisms

Neuropathic pain involves changes in the peripheral and central nervous systems (Charlton, 2005). There is some evidence that changes in the sodium channels within the peripheral nervous system promote the ectopic activity pain transmission (Waxman, 1999). Blocking of these sodium channels with the use of local anesthetics and anticonvulsant medications has shown effectiveness in reducing neuropathic pain. Sodium channels have also been found in C-fibers, which holds potential for future pharmacologic intervention. Other observations and potential explanations for neuropathic pain include presence

of nerve growth factor that may influence responsiveness and regrowth of sensory neurons during inflammation and nerve injury (Charlton, 2005).

Central nervous system involvement in neuropathic pain centers on the concept of neuroplasticity. Neuroplasticity refers to organizational changes in the brain in response to experience. Injury to the nerve and injury-related areas undergo long-term neuroplastic changes, enhancing pain sensation such as hyperalgesia or allodynia (Zhou, 2007). The anterior cingulate cortex (ACC) in the forebrain is believed to be a major area for pain-related perception, in that it may serve as a key area for pain interacting with other cognitive functions. The ACC contains many pyramidal cells, interneurons, and non-pyramidal cells. Pyramidal cells exhibit excitatory activity whereas nonpyramidal cells are inhibitory neurons containing gamma aminobutyric acid. The pyramidal cells project to the periaqueductal gray and hypothalamus and may contribute to descending modulation (Zhou, 2007). Pain modulation and the role of neurotransmitters will be discussed later in this chapter.

Nociceptive Pain

Nociceptors located in somatic tissues are dense and can easily localize pain. For example, when someone cuts their finger, the person is fully aware of where the laceration occurred. Nociceptors located in visceral organs are less dense, and pain experienced from these areas is more diffuse or referred. There are several causes of visceral pain including ischemia, chemical damage, spasm, distention, or stretching of connective tissue (Table 1.4). When visceral pain is referred to the body's surface, the individual localizes the pain in the dermatomal segment from embryonic development, not where the organ is currently located. A classic example is left shoulder pain with laceration of the spleen.

Impulses from nociceptors located in free nerve endings are transmitted to the dorsal horn of the spinal cord via myelinated fibers (A-delta) or

TABLE 1.4 Causes of Visceral Pain

Cause	Example
Ischemia of visceral tissue	Chest pain
Chemical damage to viscera	Peritonitis
Spasm of smooth muscle	Intestinal spasms
Visceral distention	Distended bladder
Stretching of connective tissue	Abdominal pain

slower unmyelinated fibers known as C polymodal nociceptors. The A-delta fibers appear responsible for sending signals to the brain that interpret pain as sharp and lancinating. The slower C fibers transmit the secondary pain sensation, that of a burning, aching type pain that persists after the initial injury (Serpell, 2005). These enter the spinal cord via the dorsal horn and synapse in lamina I, V, and X. The wide dynamic range neurons (WDR) in lamina V are thought to be “sensory-discriminative” and project into the ventrobasal thalamus, where another synapse occurs before projecting to the somatosensory cortex (Yaksh, 2005). Marginal, nociceptive specific neurons ascend from lamina I in the dorsal horn and project contralaterally to ventrobasal and medial thalamus. Input from these neurons appears to have a role in the “affective-motivational” component of the pain pathway (Yaksh, 2005).

There are three major ascending pathways that transmit nociceptive information, the spinothalamic, spinoreticular, and spinomesencephalic tracts. The *spinothalamic* tract is the main nociceptive tract that originates from neurons found in laminae I–VII and X and ascend contralaterally to the ventral thalamus. The neuron entering the dorsal horn is referred to as the first ordered neuron. The neuron projecting to the thalamus is known as the second order neuron, and the neuron projecting to the somatosensory cortex is the third order neuron. Table 1.5 identifies the ordered neurons and their projections. Interneuron activity and neurotransmitter release occurs at these synapses. The *spinoreticular* tract is made up of axons from laminae VII and VIII. Some axons cross the midline, while others project upward uncrossed. These axons terminate in both the reticular formation and the thalamus. Neurons in laminae I, VI–VIII, and X ascend contralaterally in the *spinomesencephalic* tract to the mesencephalic reticular formation, the lateral periaqueductal gray and some areas in the midbrain. Other ascending transmission pathways include the *dorsal column post-synaptic spinomedullar tract* and the *propriospinal multi-synaptic ascending system*. Pain transmission via multiple tracts provides support for use of multiple therapies in managing pain. Many C fibers terminate in reticular areas of brain-stem and intralaminar nuclei of the thalamus. These

TABLE 1.5 Afferent Pain Transmission

Neuron	Synapse
First order neuron	Dorsal horn of spinal cord
Second order neuron	Thalamus
Third order neuron	Somatosensory cortex

areas have a strong arousal effect on the nervous system and may help explain why patients have sleep disturbances with severe pain.

Neurotransmitters

The nociceptive signals from the dorsal horn neurons are transmitted by chemical means. Several amino acids and peptides have been identified as excitatory or inhibitory neurotransmitters. Table 1.6 identifies some of the afferent neurotransmitters. Some peptides that have been identified include cholecystokinin, dynorphin, somatostatin, bombesin, vasoactive intestinal peptides, and substance P. Glutamate, an amino acid, has a role in excitation. Glutamate is most likely the neurotransmitter associated with A-delta pain, and substance P with C fiber pain (Guyton & Hall, 2006). Other excitatory neurotransmitters include bradykinin, serotonin, histamine, potassium ions, acids, ACTH, and proteolytic enzymes. Prostaglandins and substance P enhance the sensitivity of pain endings but do not directly excite them (Guyton & Hall, 2006). The nonadapting nature of pain receptors leads to hypersensitivity.

Interneurons, neurons that communicate only with other neurons, play an important role in releasing excitatory or inhibitory neurotransmitters (Chao & Hart, 2003). There are actually two different definitions for interneurons, depending on whether one is referring to the peripheral or central nervous system. In the peripheral nervous system, an interneuron refers exclusively to neurons that only communicate to other neurons and have no central pathways or processes. These interneurons signal release of excitatory or inhibitory neurotransmitters pre- or post-synaptically. In the central nervous system, interneurons can also refer to a group of locally projecting neurons that release neurotransmitters. One group of locally projecting neurons releases the inhibitory neurotransmitter gamma aminobutyric acid and plays a role in pain modulation.

TABLE 1.6 Examples of Afferent Neurotransmitters

Neurotransmitter	Type
Substance P	Peptide
Adenosine triphosphate (ATP)	Purine
Aspartate	Excitatory amino acid
Glutamate	Excitatory amino acid

Pain Modulation

Pain transmission not only involves ascending nerve transmission but also descending pathways that allow the individual to react to the pain sensation. These descending pathways can be manipulated or enhanced to modulate the pain response. For example, stimulation of A-beta fibers from peripheral tactile receptors can depress pain transmission. Examples of this type of modulation include rubbing a painful area, applying liniment ointments, or use of acupuncture.

These descending pathways were discovered by stimulating areas in the brain, particularly the paraventricular gray area and ventrobasal area of thalamus. It was found that stimulation of these areas inhibited the nociceptive neurons in the dorsal horn of the spinal column.

The main descending modulating pathway in the midbrain has three basic components. First, there are neurons in the midbrain, in particular the periventricular and periaqueductal gray matter that have excitatory connections in the medulla that is serotonergic. This excitation releases serotonin, a neurotransmitter with inhibitory properties. Second, neurons in the medulla make inhibitory connections in the dorsal horn, particularly laminae I, II, and V, where the afferent nociceptors terminate. These connections include spinothalamic tract neurons that respond to painful stimuli. The third component involves local circuits in the dorsal horn that mediate the modulatory actions of the descending pathways.

A second major descending pathway occurs in the pons and involves norepinephrine. The descending pathways from the midbrain and pons are crucial links in the supraspinal modulation of nociceptive transmission. The descending serotonergic and noradrenergic axons contact the dendrites of the spinothalamic tract.

Another central mechanism of pain modulation involves endogenous opiate peptides. These peptides play a role in modulating nociceptive transmission at the level of the primary afferent synapse in the dorsal horn. Many peptides have been identified as endogenous opiates and fall within three classes: enkephalins, beta endorphins, and dynorphins. The superficial dorsal horn has a high density of enkephalin and dynorphin interneurons close to the terminals of nociceptive afferents and dendrites. The opiate peptides bind to specific membrane receptors. Three major classes of opiate receptors, mu, kappa, and delta, have been identified. The beta endorphins are active at mu receptors, whereas enkephalins are active at mu and delta receptors and dynorphin endogenous opiates are active at kappa receptors. Mu receptors are more prevalent at the terminals of nociceptive afferents and dendrites of post-synaptic

neurons. The mu receptors inhibit the excitatory neurotransmitter glutamate. Morphine is a potent agonist of mu receptors. Morphine inhibits interneuron that releases gamma aminobutyric acid (GABA). Descending inhibition of the spinothalamic tract is mediated by activation of the enkephalin interneurons in the dorsal horn. Discovery of these endogenous opioids led to the development of epidural and intrathecal delivery systems. Modulation of nociceptive transmission at the level of the primary afferent occurs with the combination of pre-synaptic and post-synaptic actions.

SUMMARY

Pain is a common concern of older adults. Pain can be acute or persistent. Physiological adaptation to persistent pain is compromised because exacerbations of other coexisting health conditions decrease resiliency and functionality (Ebener, 1999). Persistent pain can result in deconditioning, sleep disturbances, and poor nutrition. Understanding the physiological mechanisms of pain transmission and modulation helps the health care professional to develop an appropriate pain management plan.

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