Physiology and Treatment of Pain

Jennifer E. Helms, RN, PhD
Claudia P. Barone, RN, EdD, LNC, CPC, CCNS-BC, APN

Pain often occurs in critical care patients and is one of the most clinically challenging problems for critical care nurses. Pain and discomfort in these patients can be due to surgical and posttraumatic wounds, invasive monitoring devices, prolonged immobilization, mechanical ventilation, and routine nursing procedures such as suctioning and dressing changes. In addition, patients may have a preexisting chronic pain condition, complicating the assessment and treatment of acute pain. Pain is a problem in critical care that has not been adequately addressed. Strategies for changing pain management practices include providing documentation, implementing pain guidelines, using algorithms, and increasing education in pain management for acute and critical care nurses. A review of pain physiology is essential to fully understand the principles of pain management.

The pain experience can be functionally divided into acute and chronic types. Acute and chronic pain are due to different physiological mechanisms and thus require different treatments. In addition, children, adults, and elderly persons have both subtle and sharp differences in the perception of pain. Much of the nursing literature on pain is focused on common interventions but does not explain the physiological mechanisms of pain and the vastly different types of pain that patients may have. Thus, in this article, we review theories of pain and examine the physiology of pain, with emphasis on the types of pain and their manifestations. To provide the best possible care for patients experiencing pain, nurses must understand the physiology of pain, the different types of pain and their varied manifestations, the diversity of patients’ responses, and the rationale for choices of pain control methods.

Evolution of Pain Theories

As early as 1644, Descartes proposed a theory of pain, that a straight-line channel of pain exists from skin to brain. During the 19th century, von Frey theorized that pain pathways move from specialized receptors in...
body tissues to a pain center in the brain. The focus of this theory, known as the specificity theory, is specialized peripheral receptors rather than a central mechanism of pain in the brain. However, although receptors are specialized, a focus on peripheral receptors does not explain how an amputee can feel pain in the amputated limb (a phenomenon known as phantom limb pain) when the peripheral receptors no longer exist.

According to the pattern theory of pain proposed in the late 19th century, pain is the result of stimulation of certain nerve impulses that form a pattern and are then combined and dumped into the spinal cord as a lump sum of pain, a process called “central summation.” This theory can better account for the phantom limb phenomenon, because the focus is on what occurs in the brain rather than on peripheral receptors. However, the theory does not account for other factors in pain perception, such as the effect of placebos on pain.

In 1965, Melzack and Wall published the well-known gate control theory of pain, the theory most familiar to nurses. According to this theory, a mechanism in the brain acts as a gate to increase or decrease the flow of nerve impulses from the peripheral fibers to the CNS. An “open” gate allows the flow of nerve impulses, and the brain can perceive pain. A “closed” gate does not allow flow of nerve impulses, decreasing the perception of pain (Figure 1). Although the gate control theory has been widely accepted since the 1970s, it leaves unanswered questions, including chronic pain issues, sex-based differences, stress effects, and the effects of previous pain experiences.

In 1999, Melzack and Wall presented a newer theory of pain, consistent with the idea of gate control, that addresses some of these unanswered questions. This “new and improved” theory, the neuromatrix theory, says that each person has a genetically built-in network of neurons called the

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**Authors**

Jennifer E. Helms is an associate professor of nursing at Arkansas Tech University, Russellville, Arkansas.

Claudia P. Barone is professor and dean, College of Nursing, University of Arkansas for Medical Sciences, and a registered nurse II at University Hospital, PRN, Little Rock, Arkansas.

Corresponding author: Jennifer E. Helms, RN, PhD, Associate Professor of Nursing, Arkansas Tech University, Dean Hall, 402 W "O" St, Russellville, AR 72801 (e-mail: jhelms@atu.edu).

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“body-self neuromatrix.” Just as each person is unique in physical appearance, each person’s matrix of neurons is unique and is affected by all facets of the person’s physical, psychological, and cognitive makeup, as well as his or her experience. Thus, the pain experience does not reflect a simple one-to-one relationship between tissue damage and pain.

Pathways of Pain

Nociceptors, or pain receptors, are free nerve endings that respond to painful stimuli. Nociceptors are found throughout all tissues except the brain, and they transmit information to the brain. They are stimulated by biological, electrical, thermal, mechanical, and chemical stimuli. Pain perception occurs when these stimuli are transmitted to the spinal cord and then to the central areas of the brain. Pain impulses travel to the dorsal horn of the spine, where they synapse with dorsal horn neurons in the substantia gelatinosa and then ascend to the brain. The basic sensation of pain occurs at the thalamus. It continues to the limbic system (emotional center) and the cerebral cortex, where pain is perceived and interpreted (Figure 2).

Two types of fibers are involved in pain transmission. The large A delta fibers produce sharp well-defined pain, called “fast pain” or “first pain,” typically stimulated by a cut, an electrical shock, or a physical blow. Transmission through the A fibers is so fast that the body’s reflexes can actually respond faster than the pain stimulus, resulting in retraction of the affected body part even before the person perceives the pain. After this first pain, the smaller C fibers transmit dull burning or aching sensations, known as “second pain.” The C fibers transmit pain more slowly than the A fibers do because the C fibers are smaller and lack a myelin sheath. The C fibers are the ones that produce constant pain.

Regulators of Pain

Chemical substances that modulate the transmission of pain are released into the extracellular tissue when tissue damage occurs. They activate the pain receptors by irritating nerve endings. These chemical mediators include histamine, substance P, bradykinin, acetylcholine, leukotrienes, and prostaglandins. The mediators can produce other reactions at the site of injury, such as vasoconstriction, vasodilatation, or altered capillary permeability. For example, prostaglandins induce inflammation and potentiate other inflammatory mediators. Aspirin, nonsteroidal anti-inflammatory medications, and the new COX-2
inhibitors block cyclooxygenase 2, the enzyme needed for prostaglandin synthesis, thus reducing pain. Consequently, these medications are often prescribed for painful conditions due to inflammation.

The body also has a built-in chemical mechanism to manage pain. Fibers in the dorsal horn, brain stem, and peripheral tissues release neuro-modulators, known as endogenous opioids, that inhibit the action of neurons that transmit pain impulses. β-Endorphins and dynorphins are the types of natural opioid-like substances released, and they are responsible for pain relief. Endorphins are the modulators that allow an athlete to continue an athletic event after sustaining an injury. Endorphin levels vary from person to person, so different persons experience different levels of pain.

This endogenous opioid mechanism may play an important role in the placebo effect. A placebo is an inactive substance or treatment used for comparison with "real" treatment in controlled studies to determine the efficacy of the treatment under study. Despite the lack of any intrinsic value, placebos can and do produce an analgesic response in many persons. Placebo analgesia can affect nociceptive mechanisms in the cortex of the brain and descending pathways of the spinal cord. Matre et al found that expectations about pain and analgesia can modify pain perception by altering pain mechanisms in the spinal cord. For example, psychological factors such as the threat of pain and expectations about analgesia modify spinal pain transmission, thereby modifying pain.

**Acute and Chronic Pain**

**Acute Pain**

Acute pain serves a biologic purpose by providing a warning that illness or injury has occurred. The pain is usually confined to the affected area and is limited over time. Acute pain stimulates the sympathetic nervous system, resulting in "fight or flight" response symptoms, including increased heart and respiratory rates, sweating, dilated pupils, restlessness, and apprehension.

Types of acute pain include somatic, visceral, and referred (see Table). Somatic pain is superficial, coming from the skin or subcutaneous tissues; visceral pain originates in the internal organs and the linings of the body cavities. Referred pain is felt in an area distant from the site of the stimulus; it occurs because the area of referred pain is supplied by the same spinal segment as the site of the stimulus (Figure 3). Referred pain often occurs with visceral pain. Examples include shoulder pain from myocardial infarction, back pain from pancreatitis, and right shoulder pain from gallbladder disease.

**Chronic Pain**

Chronic pain is prolonged pain, persisting beyond the expected normal healing time. This characterization was previously the official definition of chronic pain according to the International Association for the Study of Pain. The term chronic is still widely
used, although many pain experts now think that all forms of chronic pain are variations of the same phenomenon and should be labeled specifically, such as neuropathic pain.23 Chronic pain can be continuous (eg, arthritis) or intermittent (eg, migraines). Chronic pain is poorly understood and is more complex and difficult to manage than is acute pain. Understanding chronic pain requires recognizing that the nervous system is not hardwired. If it were hardwired, each noxious stimulus, such as a needle stick, would elicit exactly the same nervous system response at the same intensity every time. But pain is much more complex, involving affective and cognitive traits of the person who experiences it. Melzack and Wall8 showed that repeated stimulation of C fibers results in progressive buildup of electrical response in the CNS, a phenomenon called windup, somewhat analogous to the effect of winding up a child’s windup toy. The more the toy is wound up, the faster and longer the toy will run. This persistent stimulation of peripheral nerves winds up the CNS, leading to intensified stimulation of nerve fibers that is referred to as nonnociceptive pain. The concept of windup is crucial to understanding chronic pain.24 Windup is the reason pain can continue long after the expected recovery time for an injury or a pain-initiating event.

Patients with chronic pain may not have the behaviors associated with acute pain.12 Additionally, autonomic nervous system responses (eg, nausea, vomiting, pallor, sweating) decrease with prolonged pain. The body’s fight-or-flight reaction, which normally occurs with acute pain, does not occur because the sympathetic nervous system has adapted to persistent pain impulses. Understanding chronic pain, therefore, requires listening to the person’s description of it, because expected physical symptoms may not be present. Unfortunately, because of the lack of objective evidence of pain, many patients who report chronic pain are viewed as hypochondriacs and malingerers by health care professionals.

Some evidence indicates that chronic pain and depression share the same physiological pathway.25,26 Tricyclic antidepressants and selective serotonin reuptake inhibitors have been used successfully for relief of many chronic pain syndromes such as neuropathic pain, low back pain, and fibromyalgia. These medications block the reuptake of neurotransmitters such as epinephrine and norepinephrine, thereby altering neurotransmission along pain pathways.27 Patients should be educated that the onset of analgesia with these medications differs from the onset of the antidepressant effect; analgesia will occur sooner than the expected antidepressant effect will. Some patients prescribed an antidepressant as therapy for pain may misunderstand the purpose of the drug and assume that their complaints of pain are viewed as an indication of depression or hypochondria. Health care professionals therefore should explain to patients that antidepressants, in lay terms, help block pain impulses.

Special Types of Pain
Neuropathic Pain

Chronic, often intractable pain due to injury to the peripheral nerves is known as neuropathic pain. According to Devor and Seltzer,28 this pain is a paradox. Injury to peripheral nerves should deaden sensation, much as cutting a telephone wire leaves the phone line dead, but the opposite occurs in neuropathic pain. Injury to the peripheral nerves can cause spontaneous paresthesias, numbness, pain with movement, tenderness of a partly denervated body part, and
pain that is electric shock–like, burning, shooting, or tingling.

Abnormally amplified signals in the CNS due to windup result in central sensitization, which is an increased sensitivity of spinal neurons. Central sensitization causes allodynia (pain from a stimulus that does not normally produce pain, such as touch) and hyperalgesia (a heightened pain response to a stimulus that is painful). Transcutaneous electrical nerve stimulation is used as an adjuvant therapy for some patients with neuropathic pain. With this technique, stimulating the large-diameter nerve fibers closes the gate in the spinal cord dorsal horns. The mainstay of treatment for neuropathic pain, however, is pharmacotherapy with antiepileptics and antidepressants. Antiepileptic drugs inhibit discharges on damaged nerves, and antidepressants enhance dorsal horn inhibition (ie, they help close the gate). The tricyclic antidepressants, despite their poor side effect profile, are more effective in treating neuropathic pain than are the newer serotonin selective reuptake inhibitors.

**Phantom Pain**

After amputation of a limb, a patient may experience painful sensations in the missing limb. As many as 70% of amputees report this phantom limb pain, usually within the first week after amputation. Painful sensations, which are typically intermittent, are described as shooting, stabbing, pricking, squeezing, throbbing, and burning. The missing limb may feel twisted or cramped. Often preamputation pain and phantom pain are similar. Most patients report a decrease in the degree and incidence of phantom pain in months to years after the amputation. Although several theories have been proposed to explain the pathophysiology of phantom pain, the exact etiology remains unknown. The origin of phantom pain is thought to be in the CNS and may be a somatosensory “memory” that involves complex neural interactions in the brain.

Treatment for phantom pain is challenging and often unsuccessful. No medications are specifically indicated for phantom pain, but anticonvulsants (carbamazapine), antidepressants (doxepin), β-blockers, and opioids have been used successfully to relieve phantom pain in some patients. Transcutaneous electrical nerve stimulation and sympathetic blocks have had limited success.

**Central Pain**

Central pain is a form of chronic pain caused by a lesion or dysfunction in the CNS. Causative lesions include infarction, hemorrhage, abscess, degeneration, tumors, and traumatic injury in the brain or spinal cord. For example, stroke, multiple sclerosis, and spinal cord injury can all result in central pain. The term thalamic pain is often used synonymously with central pain, although thalamic pain is specifically caused by lesions in the thalamus. The intensity of the pain ranges from mild to excruciating, but the pain is constant and irritating, causing the patient much suffering. Patients with central pain often report burning, aching, lancing (“cutting”), pricking, lacerating, and pressing sensations. The location of the pain depends on the lesion involved; the pain may occur in an entire half of the body or in only a small area, such as a hand.

The specific mechanisms of central pain are poorly understood, and no treatment is universally effective. Usually combinations of various treatments have the best results. However, treatments typically only reduce the pain, rather than eliminating it, so patients should be warned that relief probably will not be complete. Treatment includes pharmacotherapy, transcutaneous electrical nerve stimulation, and neurosurgery. Antidepressants, antiepileptics, antiarrhythmics, local anesthetics, analgesics, and a wide variety of other medications have been used for central pain, all with varying success. Neurosurgical procedures, such as cordotomy (interrupting the pathways that carry pain through the spinal cord) and thalamotomy (destroying cells in the thalamus), may be tried in patients with central pain that do not respond to other treatments, but even these measures have had only limited success.

**Differences in Populations**

**Sex-Based Differences**

An abundance of research has indicated sex-based differences in the experience of pain. Women report pain with greater frequency than men and have lower thresholds and tolerance to painful stimuli. Differences also exist in the types of painful conditions that are prevalent in women and men. For example, headaches occur in both sexes, but women experience more tension headaches and migraines with aura, whereas men report more cluster headaches and migraines without aura. Musculoskeletal conditions (eg, fibromyalgia) and autoimmune diseases are reported much more often by women than by men.

The reason for these sex-based differences is a matter of debate.
Potential mechanisms in pain include sex hormones, differences in the brain and spinal cord in men and women, genetics, sociocultural roles, stress, and neuroactive agents. Interestingly, researchers have found that brain activity in men and women differs during a pain experience. Silverman et al used positron emission tomography to examine brain activation patterns in healthy men and women who were not in pain and compared the patterns with those of men and women experiencing a painful condition. The brain patterns of the men and women experiencing pain differed significantly, but no sex-based differences were detected in the control group. This finding suggests that pain is processed differently depending on sex.

**Pain in Children**

Until the 1970s, pain in children was ignored in health care research. The common assumption was that children did not experience pain to the extent that adults do, because of the immature nervous system, or that children would not remember the pain. Consequently, children were often undermedicated or not medicated at all for pain. This practice continued until the late 1980s, when changes began to occur in pain management in infants and children as a result of research, consumer demands, and legislation to promote development of drugs for these patients. Substantial evidence now indicates not only that children experience pain but that the pain experience may have long-term adverse consequences.

The misperception that infants have immature nervous systems and therefore do not feel pain is still common. All nerve pathways necessary for pain transmission and perception are present and functioning by 24 weeks’ gestation. Research in both animal models and human newborns confirms that a lack of analgesia for pain causes “rewiring” in the nerve pathways involved in the transmission of pain. Consequently, an infant or child who experiences pain once will have greater pain perception during later painful experiences. For example, Taddio et al found that babies who did not receive analgesia or anesthesia during circumcision later had greater behavioral and physiological disturbances during immunization. Furthermore, a lack of adequate postoperative analgesia in children can increase morbidity. In a study by Anand and Hickey, compared with postoperative infants who received high-dose opioid analgesia, postoperative infants who did not had a significantly higher risk for death.

Another common myth is that children do not experience chronic pain. Indeed, children do experience chronic pain syndromes, such as complex regional pain syndrome, as well as acute forms of pain related to chronic conditions such as sickle cell anemia. They also experience various forms of recurrent pain, most commonly headache, abdominal pain, back pain, chest pain, and limb pain.

**Pain in the Elderly**

The effects of aging on pain sensation, perception, and behavior are not well established. Findings from studies on pain in human aging are conflicting, partly because of inconsistent research methods and ambiguous research definitions of pain. Some notable consistencies have been found, however. Compared with younger adults, elderly persons rely more on second pain (C fiber) than on first pain (A fiber). This difference means that older adults are more likely to describe a painful injury or stimulus as burning (slower C fiber second pain) rather than as sharp or pricking (faster A fiber first pain). Another well-documented finding in the elderly is a slower response time to pain.

No evidence exists that pain intensity lessens with age. Pain as a sensory process does not mimic other senses, such as hearing and sight, which gradually diminish with normal aging. Altered reactions to painful events may be due to loss of communications skills, cognitive abilities, or the failure of basic reflexes due to aging. Additionally, pain in the elderly may be manifested as something other than pain, such as delirium. Referred pain may be atypical in the elderly, as in silent (painless) myocardial infarction. Although this lack of referred pain is a clinical problem, no definitive evidence exists of the relationship between age and silent myocardial infarction.

**Assessment and Treatment of Pain**

Pain management in critically ill patients can be challenging. For a variety of reasons, critically ill patients may be unable to verbalize, or they may not fully communicate the nature of their pain. Patients and health care providers may assume that treatment with opioid analgesics can lead to addiction. Despite efforts to relieve pain, harmful physiological effects can ensue, including inadequate sleep, exhaustion, disorientation, anxiety, tachycardia, increased myocardial oxygen demand, immunosuppression, and increased catabolism. Recognition of pain...
in critically ill patients is crucial. Painful stimuli can be triggered by a variety of conditions or treatments, such as incisions, drains, ischemia, inflammation, edema, and indwelling invasive and noninvasive catheters, and by a patient’s previous experiences with painful stimuli.

Assessment
Pain is an important problem in critical care, and the assessment of pain should be a priority. The Joint Commission developed a pain assessment and management program that hospitals must implement to fulfill accreditation requirements. Unfortunately, even with pain assessment guidelines and mandates in place, clinicians often underrate pain. Critically ill patients’ self-reports of pain can be inaccurate or inconclusive because of endotracheal intubation, use of sedatives, or an unconscious state. As a result, health care providers must rely on sensory, physiological, and behavioral parameters to assess the presence of painful stimuli. These parameters include increases in heart rate and blood pressure, anxiety, and difficulty in providing mechanical ventilation.

Pain scales can be used to determine the degree of pain a patient is experiencing. Two commonly used scales are the numeric rating scale and the FACES scale. Scores on the numeric rating scale range from 0 to 10, with 10 being the worst pain ever experienced and 0 being no pain sensation at all. The difficulty with using this scale is that critically ill patients often cannot speak because of endotracheal intubation. The second pain scale is the FACES pain-rating scale, which may be more useful in critical care. This scale includes 6 faces with indications of increasing pain intensity (Figure 4); a patient points to the appropriate face to indicate the patient’s pain level.

Some patients cannot provide a self-report because they are sedated or have cognitive impairment. Assessment of these patients requires use of a tool that relies on evidence of pain behaviors. The Behavioral Pain Scale was developed for use in critically ill patients and is used to evaluate behaviors that may indicate pain, including facial expression, upper limb movement, and ventilator compliance. Use of the scale is limited to patients who can demonstrate the behaviors being assessed.

Treatment
Once pain has been assessed, interventions directed toward pain relief must be implemented. Pain management can be divided into pharmacological and nonpharmacological interventions. In a study by Gelinas et al, nonpharmacological interventions were used in 22% of the pain episodes evaluated. A variety of comfort-producing measures were implemented, including endotracheal tube suctioning, repositioning in bed, massage, oral care, and reassurance. Other nonpharmacological measures for critically ill patients include...
application of heat or cold, massage, therapeutic touch, guided imagery, and relaxation techniques.

Principles of pharmacological management begin with preemptive analgesia (before the pain begins) or as soon as possible after the pain begins. Preemptive analgesia not only reduces the pain response but also can reduce the chance of long-term sequelae. As previously described, painful experiences can imprint themselves on the nervous system. Preemptive analgesia can prevent noxious signals from reaching the CNS, thereby reducing the chance that spinal neurons will become sensitized and lead to heightened pain responses (hyperalgesia) or pain experiences from typically painless sensations (allodynia). Continuous pain management is essential. Recent evidence suggests that duration of action of pain medications should be administered intravenously for the quickest onset of action. Importantly, not only the onset of action but also the expected duration of action of pain medications must be understood so that the medication schedule can maximize pain control efforts. Around-the-clock dosing is appropriate for critically ill patients. Such dosing helps prevent pain and maintain a pain rating that is satisfactory or tolerable to the patient and helps prevent the physiological changes due to poor pain management. Despite efforts to relieve pain, harmful physiological effects can ensue, including inadequate sleep, exhaustion, disorientation, anxiety, tachycardia, increased myocardial oxygen demand, immunosuppression, and increased catabolism. Expectations of patients regarding pain can radically change the strength of pain responses in the spine. Research has shown that pain relief, to a large degree, depends on what the patient expects from a pain relief intervention. More recent research confirmed the converse of this phenomenon, that antianalgesic expectations can dramatically reduce the effect of analgesic treatments by blocking the action of the drugs. In other words, if a patient expects little or no pain relief from an analgesic or a pain relief measure, then the action of that drug or measure can be blocked in the spine, and the drug or measure will be ineffective.

Pain can be a severe and frequent symptom in intensive care unit patients. Many patients report dissatisfaction with inconsistent pain relief and the inability to acquire restful sleep as a result of painful stimuli. Pain control in intensive care unit patients should encompass use of a variety of pain-relieving approaches. These may include options such as traditional opioids and nonopioid analgesics as well as narcotics and synthetic narcotics. In addition, the use of nonsteroidal anti-inflammatory drugs in certain circumstances may augment a patient’s response to pain and provide relief. Personal beliefs of each patient and the patient’s family should also be considered useful when deemed appropriate. These beliefs might include prayer, meditation, relaxation techniques, and acupuncture.

**Conclusion**

The experience of pain is unique for each person and encompasses a person’s physical, psychological, cognitive, and emotional network. The first step in effectively dealing with pain is determining the specific type of pain a patient is experiencing. Acute pain and chronic pain have different manifestations. A patient with chronic pain will not have objective physiological indications expected of patients with pain, such as pallor, sweating, tachycardia, and facial grimacing. Additionally, differences in sex and age may play a role in a patient’s pain experience. Women and men can experience pain differently because of mechanisms not yet understood. Children do experience pain and should be treated accordingly even though they may not be able to verbally express their pain experience. Elderly patients may describe their pain differently than younger persons do and often respond more slowly to pain. This difference in description of pain does not mean that elderly patients experience less pain. Pain should be treated promptly and adequately in all patients. To provide the best care for patients in pain, nurses must be alert to the different types and manifestations of pain and the differences of each patient that contribute to the pain experience.
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1. Which of the following statements is correct about acute and chronic pain?
   a. Acute and chronic pain have different physiological mechanisms.
   b. Treatments for acute and chronic pain are the same.
   c. Children and adults share the same perceptions of acute and chronic pain.
   d. Physiologic indicators of acute and chronic pain are identical.

2. Which of the following pain theories includes the process of “central summation”?
   a. Gate control theory
   b. Specificity theory
   c. Neuromatrix theory
   d. Pattern theory

4. Where is pain interpreted?
   a. Substantia gelatiosa
   b. Noctceptors
   c. Cerebral cortex
   d. Limbic forebrain

5. Compared with A fibers, which one of the following is correct about C fibers?
   a. C fibers are larger
   b. C fibers have a myelin sheath
   c. C fibers produce “fast pain.”
   d. C fibers produce constant pain

6. What modulator allows an athlete to continue an athletic event after sustaining an injury?
   a. Leukotrienes
   b. Prostaglandins
   c. Endorphins
   d. Bradykinin

7. Which of the following statements is correct about visceral pain?
   a. Visceral pain is superficial
   b. Visceral pain is described as sharp and burning
   c. Visceral pain is poorly localized
   d. Visceral pain is described as painful numbness.

8. Which of the following is a source of neuropathic pain?
   a. Bladder distention
   b. Incisional pain
   c. Phantom limb pain
   d. Skeletal muscle spasms

9. What is the reason pain can continue long after the expected recovery time for an injury?
   a. Windup
   b. Open gate
   c. Body-self neuromatrix
   d. Somatosensory “memory”

10. Compared with men, which is correct about the pain experience in women?
    a. Women experience more migraines with aura
    b. Women have a higher pain threshold.
    c. Women report pain less frequently
    d. Women have a higher pain tolerance

11. Which of the following statements is correct about pain in children?
    a. Children do not experience chronic pain
    b. Inadequate postoperative analgesia in children can increase morbidity
    c. Children do not experience pain to the same extent as adults
    d. Children do not feel pain because of an immature nervous system

12. Which of the following statements is correct about pain in older adults?
    a. Older adults rely more on first pain than second pain
    b. Pain intensity lessens with aging
    c. Older adults have a faster response time to pain
    d. Pain may be manifested as delirium

13. Which of the following terms describes pain from a stimulus that does not normally produce pain?
    a. Hypesthesia
    b. Allodynia
    c. Hyperesthesia
    d. Hyperalgesia
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Crit Care Nurse 2008;28 38-49
American Association of Critical-Care Nurses
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