

Pain & Symptom Management

Goals & Objectives

Course Description

“Pain & Symptom Management” is an online asynchronous continuing education course for occupational therapists and occupational therapy assistants. This course presents contemporary information about pain management including sections on pain mechanisms, clinical assessment, pharmacological options and therapeutic interventions.

Course Rationale

The information presented in this course is applicable for occupational therapy professionals in all settings. A greater understanding of pain and symptom management will enable occupational therapists and occupational therapy assistants to provide more effective and efficient rehabilitative care to individuals who are experiencing pain.

Course Goals and Objectives

Upon completion of this course, the participant will be able to:

1. Differentiate between acute and chronic pain.
2. List and differentiate the different types of pain mechanisms.
3. Identify and select appropriate pain assessment tools.
4. Define the steps of the WHO Pain Relief Ladder
5. Identify the non-opioid medications used to treat pain.
6. Identify the opioid medications used to treat pain
7. List the adverse effects of Opioid medications
8. Recognize the interventional approaches available to treat pain
9. Identify and differentiate various rehabilitative treatments for pain
10. Identify and differentiate mind-body therapies
11. Define patient self-management of pain

Course Provider – Innovative Educational Services

Course Instructor - Michael Niss, DPT

Target Audience – Occupational therapists and occupational therapy assistants

Level of Difficulty – Introductory

AOTA Classification Code for CE Activity – Category 1: Client Factors; Category 2: Intervention, Approaches to intervention, Outcomes

Course Prerequisites - None

Method of Instruction/Availability – Online text-based course available continuously.

Criteria for issuance of CE Credits - A score of 70% or greater on the course post-test.

Continuing Education Credits – 1 hour, .1 AOTA CEU, 1.25 NBCOT PDU

Fees - \$9.95

Conflict of Interest – No conflict of interest exists for the presenter or provider of this course.

Refund Policy - Unrestricted 100% refund upon request. The request for a refund by the learner shall be honored in full without penalty or other consideration of any kind. The request for a refund may be made by the learner at any time without limitations before, during, or after course participation.

Pain & Symptom Management

Course Outline

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Background and Definitions

Pain is a subjective individual experience encompassing sensory, cognitive, emotional, and social dimensions. One person may report a moderate level of pain following an injury while another person with a similar injury may report a much higher, or lower, pain level. This individual-by-individual experience of pain depends on numerous factors that include the person's unique genetic structure, cognitive abilities, motivation, emotional status, and psychological state. In addition, environmental factors, gender, past experiences, cultural / social influences, and general health condition also play a role.¹

A widely-accepted definition of pain was developed by the International Association for the Study of Pain. They defined pain as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.”² The IASP definition means that pain is a subjective experience; one that cannot be objectively measured and depends on the person's self-report. There can be a wide variability in how a person experiences pain to a given stimulus or injury. Pain may be experienced as a prick, tingle, sting, burn, or ache. Normally, acute pain is a protective response to tissue damage resulting from injury, disease, overuse, or environmental stressors. Pain in its most benign form warns individuals that something isn't quite right, that further investigation is warranted. At its worst, however, pain limits function and overall well-being. Pain is a complex perception that differs enormously among individual patients, even those who appear to have identical injuries or illnesses.³

Societal Impact

The socioeconomic consequences of chronic pain include reduced quality of life, negative impact on relationships, job loss or reduced job responsibilities, and increased rates of depression. In addition to the personal social and psychological “costs” for the person suffering with chronic pain, there are also considerable economic costs to the patient and to society as a whole. A recent Institute of Medicine (IOM) report places this cost at more than 500 billion dollars per year in the USA¹; this economic burden is higher than the healthcare costs for heart disease, cancer and diabetes combined, and stems from the costs of healthcare services, insurance, welfare benefits, lost productivity, and lost tax revenues, among others.¹ Pain symptoms cause major medical and socioeconomic problems and are the most common cause of long-term disability in middle aged people.⁴ The American Pain Society estimates that 9 percent of the U.S. adult population suffers from moderate to severe, noncancer related chronic pain.⁵ However, epidemiological research has suggested that the prevalence of chronic pain varies, depending on how the survey questions are asked and how chronic pain is defined. Researchers have estimated that from 10 to 20 percent of adults report having chronic pain when defined as persistent pain lasting at least 3 months.⁶ People who are 50 years of age and older are twice as likely to have been diagnosed with chronic pain when compared to people who are younger.^{2, 6}

Neurobiology of Pain

When nociceptors are excited, the stimulus is converted through transduction into action potentials that travel to the dorsal horn of the spinal cord. Signals then continue from the dorsal horn to the brain along multiple pathways in the cord: to the somatosensory cortex, where pain is evaluated; to the limbic system, where emotional reactions are mediated; to the autonomic centers that control such automatic functions as breathing, perspiration, and heart rate; and to other parts of the brain, where a behavioral response to the stimuli is determined. Nociceptive impulses are also transmitted to nearby terminals of the same nerve, where they may lead to diffuse pain and release of inflammatory substances that produce the flare and swelling that is a protective response to tissue injury.⁷

Nociceptive input triggers a pain-inhibiting response. Signals traveling the ascending pathways are met by descending signals that emerge at various points along the spinal cord and brain. This antinociceptive response involves many chemicals, including endorphins, enkephalins, gamma aminobutyric acid, norepinephrine, serotonin, oxytocin, and relaxin. Inhibitory signaling serves to attenuate nociceptive input, dampening the formation of pain sensation and providing pain relief.⁷

Neurotransmitters

There are many different neurotransmitters in the human body and they play a role in normal function as well as in disease. In the case of nociception and pain, they act in various combinations at all levels of the nervous system to transmit and modify signals generated by noxious stimuli.⁸ Some of the neurotransmitters that play a prominent role in pain transmission and suppression are glutamate, GABA, norepinephrine, serotonin, and opioids.

Glutamate

One excitatory neurotransmitter of special interest to pain researchers is glutamate, which plays a major role in nervous system function and in pain pathophysiology. The modulation of glutamate neurotransmission is complex, but it plays a key role in heightening the sensitivity to pain through increased responsiveness of excitatory receptors in the spinal cord dorsal horn and in the brain. This is part of a process called central sensitization and contributes to making pain persist.⁸

GABA

Unlike glutamate, GABA (or gamma-aminobutyric acid) is predominately an inhibitory neurotransmitter in that it generally decreases or blocks the activity of neurons. Most of what we know of its role in pain is related to its function in inhibiting spinal cord neurons from transmitting pain signals and therefore dampening pain.⁸

Norepinephrine and Serotonin

Norepinephrine and serotonin are neurotransmitters used by the descending pain pathways from the brain stem to dampen the incoming signals from painful stimuli from

the site of the injury or inflammation. Serotonin receptors also are present on the nerves that supply the surface of the brain involved in migraines.⁸

Opioids

The opioids are another important class of neurotransmitters that are involved in pain control, as well as pleasure and addiction. Their receptors are found throughout the body and can be activated by endogenous (produced by our bodies) opioid peptides that are released by neurons in the brain. The enkephalins, dynorphins, and endorphins are some of the body's own natural pain killers. They may be more familiar for the role of endorphins in the feeling of well-being during exercise—the runner's high.⁸

Acute Versus Chronic Pain

Pain does serve an important vital function as a warning signal of tissue damage, resulting from an accidental trauma, infection, or inflammation, for example. It usually disappears after the injured tissue has healed. In contrast to such acute pain episodes, chronic pain is usually considered as having no biological role but is associated with changes in the peripheral and central nervous systems that contribute to its persistence. Because of these changes that involve alterations in brain morphology, physiology, and neurochemistry, chronic pain is now often viewed as a neurological disorder akin to other chronic medical illnesses and conditions involving analogous alterations in the nervous system (e.g., epilepsy, Parkinson's disease); in other words, chronic pain is a disease or illness in its own right. Furthermore, chronic pain may be a pain disorder per se (e.g., fibromyalgia, trigeminal neuralgia, temporomandibular disorders, migraine) although affecting other functions (e.g., mobility, cognitive function), or be an accompaniment of many chronic diseases and disorders (e.g., arthritis, diabetes, cancer, HIV/AIDS).¹

All pain starts as acute pain; however, not all pain progresses to chronic pain. Approximately 20% of acute pain conditions can transition into chronic pain, especially if the acute pain is not appropriately managed.¹ There may be many causative factors that account for prolonged pain. Apart from the psychosocial influences on chronic pain development, physiologic factors that contribute to chronic pain include alterations in the spinal cord that occur when acute pain is inadequately treated. These changes lead to increased excitability, decreased inhibition, and reorganization of certain spinal tracts. The time frame for defining chronic pain varies from 3 to 6 months of ongoing pain. However, some would argue that chronic pain is any pain that persists longer than the reasonably expected healing time for the involved tissues.

Acute Pain

Acute pain is an unpleasant, though normal sensory experience in response to a noxious stimulus and plays an important protective role by alerting a person to actual or potential physical injury.⁹ Acute pain, for the most part, results from disease,

inflammation, or injury to tissues. This type of pain generally comes on suddenly, for example, after trauma or surgery, and may be accompanied by anxiety or emotional distress. The cause of acute pain can usually be diagnosed and treated. The pain is self-limiting, which means it is confined to a limited period of time and severity.⁸ Painful symptoms can often be self-managed while the underlying cause resolves and recovery occurs. Such instances generally require little or no professional intervention. Acute pain does not always resolve as expected however, especially if it is associated with a serious disease or condition, or begins with an injury that does not receive timely or appropriate medical care.⁹

When pain persists after the underlying cause is resolved, it may signal that pain-initiated changes in the central nervous system have occurred. If so, this chronic pain is no longer a symptom of another disorder and has become the disease itself.⁹ The persistence of pain creates a complex biopsychosocial phenomenon that may interfere with many aspects of a person's life—ability to work, social activities, and both physical and mental health.⁹

Chronic Pain

Chronic pain is now believed to be a chronic disease condition in the same manner as diabetes and asthma. Chronic pain can be made worse by environmental and psychological factors. By its nature, chronic pain persists over a long period of time and is resistant to many medical treatments. It can, and often does, cause severe problems.⁸

Pains such as migraine and fibromyalgia, in which there is no noxious stimulus and no apparent neurological lesion, are attributed to dysfunction of a structurally intact CNS. Chronic pain often results from a process of neural sensitization following injury or illness in which thresholds are lowered, responses are amplified (hyperalgesia), normally non-noxious stimulation becomes painful (allodynia), and spontaneous neural discharges occur. Increased signaling disconnected from nociceptive input can become autonomous, self-sustaining, and progressive, leading to the continuous perception of pain even in the absence of ongoing tissue damage. Thus, chronic pain is not equivalent to prolonged acute pain and for clinical purposes is best considered a distinct disorder.⁷

Pain Mechanisms

The three mechanisms underlying the pathophysiology of pain are nociceptive, neuropathic, and psychogenic.

Nociceptive

Nociceptive pain, which may be either somatic or visceral in nature, originates with a chemical, mechanical, or thermal injury to tissue that stimulates pain receptors that

transmit a signal to the central nervous system (CNS), causing the perception of pain. Pain receptors are found in somatic (e.g., cutaneous, bone) and visceral tissues. The amount of visceral sensory innervation and the diffusion of visceral pain signals within the brain explain the difficulty experienced by patients in describing or localizing visceral pain compared with somatic pain. A specific type of visceral pain is referred pain, which is explained by the commingling of nerve fibers from somatic and visceral nociceptors at the level of the spinal cord. Patients mistakenly interpret the pain as originating from the innervated somatic tissue. Visceral pain may be accompanied by autonomic signs such as sweating, pallor, or bradycardia. Somatic pain is more easily localized.¹⁰

Neuropathic

Neuropathic pain is pain caused by damage to the peripheral nervous system or the CNS (spinal cord or brain). Neuropathic pain can be caused by direct trauma, disease (such as diabetes mellitus, herpes zoster, multiple sclerosis), or secondary to treatment (i.e. radiotherapy, chemotherapy or surgery). The pain may be spontaneous or evoked by stimuli. Patients who experience pain from non-noxious stimuli are classified as having allodynia.¹⁰

Psychogenic

Emotional distress may also contribute to the pain experience. If the patient's pain appears to be disproportionate to the underlying stimulus, it is important to evaluate for psychological and existential distress which may be contributing to the pain complaint. Disproportionate pain is termed hyperalgesia.¹⁰ Psychosocial issues can either exacerbate or ameliorate the experience of pain. Depression and anxiety can have a large influence on the pain experience.¹⁰ These psychosocial issues cannot be easily treated through traditional somatic based approaches, and may require an additional referral to a psychological or psychiatric professional.¹⁰

Pain Assessment

Comprehensive assessment of pain involves both clinician observation and patient report. The goal of the initial pain assessment is to characterize the pathophysiology of the pain and to determine the intensity of the pain and its impact on the patient's ability to function. Ideally, comprehensive pain assessment includes a discussion about the patient's goals and expectations for pain management. This conversation may lead to a fruitful discussion about balancing pain levels and other patient goals, such as mental alertness. Comprehensive pain assessment also includes pain history, pain intensity, quality of pain, and location of pain. For each pain location, the pattern of pain radiation is assessed. Also important is provider awareness of the patient's current pain management treatment plan and how the patient has responded to treatment; this includes how adequately the current treatment plan addresses any breakthrough or episodic pain.¹⁰

A full assessment also reviews previously attempted pain therapies and reasons for discontinuation; other associated symptoms such as sleep difficulties, fatigue, depression, and anxiety; functional impairment; and any relevant laboratory data and diagnostic imaging. A focused physical examination includes clinical observation of pain behaviors, pain location, and functional limitations.¹⁰ Effective pain treatment begins with screening at every visit and a thorough assessment if pain is present. Patient self-report is the standard of care for evaluating pain. Many tools have been developed to quantify the intensity of pain.¹⁰

Assessment Tools

Standardized instruments provide ways to assess and track patient pain levels, function, substance use, and other factors important to managing pain. Standardized tools provide supplemental information for treatment planning and assessment of risk and outcomes. If used well, tools can reduce clinician bias during patient assessment. The sensitivity and specificity of screening instruments vary, and all can yield false-positive or false-negative results. In addition, no single instrument has been shown to be appropriate for use with all patient populations. Because of their limitations, standardized tools should not be the absolute determinants of treatments offered or withheld.⁷

When using standardized tools, clinicians should¹¹:

- Understand the strengths and weaknesses of each tool.
- Select a tool appropriate for the patient, considering memory problems, cognitive impairments, eyesight, literacy level, cultural background, gender, ethnicity, and other factors.
- Teach patients how to use self-administered tools, even “self-explanatory” tools; otherwise, the information they provide may be invalid.

The assessment of pain should include documentation of the following:

- Pain onset, quality, and severity; mitigating and exacerbating factors; and the results of investigations into etiology
- Pain-related functional impairment
- Emotional changes (e.g., anxiety, depression, anger) and sleep disturbances
- Cognitive changes (e.g., attentional capacity, memory)
- Family response to pain (i.e., supportive, enabling, rejecting)
- Environmental consequences (e.g., disability income, loss of desired activities, absence from desirable or feared work)

Several factors may complicate an assessment of pain levels in any pain patient:

- Some patients may report not only their level of pain intensity, but their suffering, which may be greater than their pain intensity.
- Clinicians tend to believe that a patient’s pain level is actually lower than the patient reports, except when the patient reports low pain¹²

- Clinicians are especially likely to underestimate—and, therefore, to undertreat—pain and disability in women, the elderly, minorities, and people of low economic status.¹³

Tools to Assess Pain Level⁷

Faces Pain Scale

(wongbakerfaces.org)

Strength:

- Easy to use
- Usable with people who have mild to moderate cognitive impairment
- Translates across cultures and languages

Weakness

- Visual impairment may affect accuracy or completion
- May measure pain affect, not only pain intensity

Numeric Rating Scale

(www.va.gov/PAINMANAGEMENT/docs/Pain_Numeric_Rating_Scale.pdf)

Strength

- Easy to use if patient can translate pain into numbers
- Easy to administer and score
- Can measure small changes in pain intensity
- Oral or written administration
- Sensitive to changes in chronic pain
- Translates across cultures and languages

Weakness

- Difficult to administer to patients with cognitive impairments because of difficulty translating pain into numbers

Verbal Rating Scale / Graphic Rating Scale

(www.scielo.br/img/revistas/reeusp/v49n5//0080-6234-reeusp-49-05-0804-gf02.jpg)

Strength

- Easy to use
- Oral or written administration
- High completion rate with patients with cognitive impairments
- Sensitive to change and validated for use with chronic pain
- Correlates strongly with other tools

Weakness

- Not as sensitive as NRS or Visual Analog Scale

Visual Analog Scale

(www.physio-pedia.com/images/6/63/Visual_analog_scale.gif)

Strength

- Easy to use, but must be presented carefully
- Precise
- Sensitive to ethnic differences

- Easily translated across cultures and languages
- Some evidence that a horizontal line may be better than a vertical (“thermometer”) orientation

Weakness

- Visual impairment may affect accuracy
- Can be time consuming to score, unless mechanical or computerized VAS tools are used
- Low completion rate in patients with cognitive impairments
- Difficult to administer to patients with cognitive impairments
- Cannot be administered by phone or email
- Subject to measurement error

Tools to Assess Several Dimensions of Pain⁷

Brief Pain Inventory

(https://openi.nlm.nih.gov/imgs/512/367/3973876/PMC3973876_JMedLife-06-383-g008.png)

Strength

- Short form better for clinical practice
- Fairly easy to use
- Useful in different cultures
- Translated into and validated in several languages

Weakness

- Not easily used with patients with cognitive impairments

McGill Pain Questionnaire

(<http://anesthesiology.pubs.asahq.org/data/Journals/JASA/931183/28FF1.png>)

Strength

- Short form easier to administer
- Extensively studied

Weakness

- Measures pain affect
- Not appropriate for patients with cognitive impairments
- Translation complicated
- Meaning of pain descriptors may vary across racial and ethnic groups

Special Considerations

Self-report is accepted as the gold standard of pain assessment; however, for certain vulnerable populations, such as children, those with learning disabilities, and those who are cognitively impaired, self-report may not be feasible or reliable. While adults and children older than 7 years can effectively utilize the numerical rating scale, young children and those with cognitive impairment may benefit from using a pictorial scale such as the Faces Pain Scale. Cognitive impairment may impede a person’s ability to describe pain, recall pain events, or understand the tools used to assess pain, leading such patients to receive more or less analgesia. Pain assessment can be evaluated via direct observation, family/caregiver report, and evaluation of response to pain relief

interventions. For patients with advanced dementia, tools relying on professional caregiver assessment of pain through the observation of patient behaviors have been developed.¹⁰ Cognitive impairment extends beyond patients with a diagnosis of dementia, such as those with brain tumors and delirium. In such patients, the Faces Pain Scale and the Colored Analogue Scale as well as vertical instead of horizontal orientation of scales may be preferable to the numeric rating scales.¹⁰

Prognostic Indicators

Several pain-related factors and patient-related factors predict response to pain treatment. Specifically, a high baseline pain intensity, neuropathic pain, and incident pain are often more difficult to manage. Furthermore, several patient characteristics such as a personal or family history of illicit drug use, alcoholism, smoking, somatization, mental health issues such as depression or anxiety, and cognitive dysfunction are associated with higher pain expression, higher opioid doses, and longer time to achieve pain control.¹⁰

Pain Management

Effective pain management can generally be accomplished by paying attention to the following steps:¹⁰

1. Regular screening to ensure that the patient's pain is recognized early.
2. Proper characterization of the pain to identify underlying pathophysiology, which could significantly influence treatment options.
3. Determining whether the pain requires pharmacologic and/or other modalities of treatment. Pain is often multifactorial in nature, so factors that may modulate pain expression, such as psychological distress and substance use, should be assessed.
4. Identifying the optimal pharmacologic and nonpharmacologic treatment options, including referrals to specialists, if needed. Complex pain often requires multidimensional interdisciplinary evaluation and intervention. There are many issues to consider when determining the most appropriate treatment, such as the following:
 - Previous pain treatments.
 - Patient prognosis.
 - Predictive factors for pain control (e.g., psychological distress).
 - Impact on function.
 - Comorbidities (e.g., renal or hepatic failure).
 - Risk of misuse of or addiction to pain medications.
 - Patient preference.
5. Providing proper education about treatment, including medication administration, expected side effects and associated treatments, and when patients can expect improvement. If opioids are considered, opioid phobia and the risks of opioid use

and misuse should be addressed. Patients and family caregivers should be educated about the safe storage, use, and disposal of opioids.

6. Monitoring the patient longitudinally with return visits.

Pain Management Goals¹⁴

All successful and effective pain management programs focus on achieving these essential goals:

- Maximize function while minimizing pain.
- Provide measurable improvement in physical/functional capabilities.
- Return to work/recreation (when applicable).
- Assist patient in assuming self-management responsibilities.
- Maintenance of functional gains upon discharge with appropriate provision for after-discharge planning/follow-up.
- Decrease medication utilization.
- Reduce health care utilization (decrease focus on medical procedures).
- Independent self-management is the long-term goal of all forms of functional restoration.

Pharmacological Management

WHO Pain Relief Ladder¹⁰

A commonly used approach to pain management employs the World Health Organization (WHO) pain relief ladder, which categorizes pain intensity according to severity and recommends analgesic agents based on their strength.

Step 1 on the WHO pain relief ladder treats mild pain. Patients in this category receive nonopioid analgesics such as acetaminophen, nonsteroidal anti-inflammatory drugs, or an adjuvant analgesic, if necessary.

Step 2 treats patients experiencing mild to moderate pain who are already taking a nonopioid analgesic, with or without an adjuvant analgesic, but who are still experiencing poor analgesia. Step 2 agents include tramadol and acetaminophen products containing hydrocodone, oxycodone, and codeine.

Step 3 treats moderate to severe pain with strong analgesics. Step 3 opioids include morphine, hydromorphone, fentanyl, levorphanol, methadone, oxycodone, and oxycodone.

Familiarity with opioid pharmacokinetics, equianalgesic dosing, and adverse effects is necessary for their safe and effective use. The appropriate use of adjuvant pharmacological and nonpharmacological interventions is needed to optimize pain management.

Non-Opioid Medications

Acetaminophen

Acetaminophen is an effective analgesic and antipyretic without anti-inflammatory activity. Acetaminophen is generally well tolerated, causes little or no gastrointestinal irritation, and is not associated with ulcer formation. Acetaminophen has been associated with liver toxicity when the recommended daily dose is exceeded or in patients who chronically use alcohol. Patients may not realize that many over-the-counter preparations may contain acetaminophen. In general, the total daily dose of acetaminophen should not exceed 3 grams per 24-hour period from all sources, including narcotic-acetaminophen combination preparations. Patients who consume three or more alcoholic drinks per day are at greater risk for liver toxicity, and consideration should be given to the use of other analgesics or limiting the acetaminophen dose to 2 grams per 24-hour period from all sources. Liver function monitoring is recommended as clinically indicated.¹⁴

Nonsteroidal Anti-inflammatory Drugs (NSAIDs)

Often initiated when an individual has mild pain, NSAIDs are useful in managing moderate and severe pain as adjunct agents to opioids. No single NSAID is preferred over others, and all are better than placebo for analgesia. As opioid adjuncts, acetaminophen and NSAIDs have shown benefit both in improved analgesia and in decreased opioid use. These agents are used with care or perhaps avoided in patients who are elderly or have renal, hepatic, or cardiac disease. Prominent side effects are gastrointestinal irritation, ulcer formation, and dyspepsia, with other side effects of concern being cardiotoxicity, nephrotoxicity, hepatotoxicity, and hematologic effects.¹⁰

Muscle Relaxants

Muscle relaxants (e.g., baclofen, cyclobenzaprine, carisoprodol, metaxalone, tizanidine) are most useful for acute musculoskeletal injury or exacerbation of injury. Chronic use of benzodiazepines or any muscle relaxant is not recommended due to their habit-forming potential, severe sedation, seizure risk following abrupt withdrawal, and documented contribution to deaths of patients on chronic opioids due to respiratory depression.¹⁴

Anticonvulsants (Carbamazepine, Gabapentin, Pregabalin)

These medications are not considered first-line medications in the treatment of non-acute pain. All patients on these medications should be monitored for suicidal ideation, hepatic and renal functioning as well as consideration of the potential for medication interaction. Anticonvulsants are not recommended for axial spine pain (neck or back pain without documented radiation) unless there is evidence of a related neuropathic component. These agents can also be considered in the setting of post-traumatic migraine headache.¹⁴

Antidepressants (Tricyclics, SSRIs, SSNRIs)

Classified into several categories based on their chemical structure and their effects on neurotransmitter systems. Pain responses may occur at lower doses with shorter response times than observed when these agents are used in the treatment of mood disorders. Neuropathic pain, diabetic neuropathy, post-herpetic neuralgia, and cancer-related pain may respond to antidepressant doses low enough to avoid adverse effects that often complicate the treatment of depression. All patients being considered for antidepressant therapy should be evaluated and continually monitored for suicidal ideation and mood swings. Many antidepressant medications have the potential to lower seizure threshold. Compliance and functional recovery may be compromised by secondary weight-gain and fatigue. In general, side effects can be mitigated if a low dose is initiated and slowly increased as tolerated. When discontinuing antidepressant medication, attention is required for the potential for withdrawal reactions, especially in the case of venlafaxine and certain tricyclics. Antidepressant medications may be helpful when there is nocturnal sleep disruption.¹⁴

Topical Pain Medications

Topical pain medications (e.g., capsaicin, lidocaine, topical NSAIDs and topical salicylates and non-salicylates) may be an acceptable form of treatment in selected patients. A topical agent should be prescribed with strict instructions for application and maximum number of applications per day to obtain the desired benefit and avoid potential toxicity. For most patients, the effects of long-term use are unknown and thus may be better used episodically. These agents may be used in those patients who prefer topical treatments over oral medications. Localized skin reactions may occur, depending on the medication agent used.¹⁴

Capsaicin - offers a safe and effective alternative to systemic NSAIDs, although its use is limited by local stinging or burning sensation that typically disappears with regular use. Patients should be advised to apply the cream on the affected area with a plastic glove or cotton applicator to avoid inadvertent contact with eyes and mucous membranes. Long-term use of capsaicin is not recommended.¹⁴

Topical lidocaine - is only indicated when there is documentation of a diagnosis of neuropathic pain. In this instance, a trial for a period of not greater than four weeks can be considered, with the need for documentation of functional gains as criteria for additional use.¹⁴

Topical NSAIDs - may achieve tissue levels that are potentially therapeutic. Overall the low level of systemic absorption can be advantageous, allowing the topical use of these medications when systemic administration is relatively contraindicated (such as patients with hypertension, cardiac failure, peptic ulcer disease or renal insufficiency).¹⁴

Topical salicylates or nonsalicylates - overall do not appear to be more effective than topical NSAIDs. May be used for a short-term course especially in patients with chronic

conditions in whom systemic medication is relatively contraindicated or as an adjuvant to systemic medication.¹⁴

Opioids

The use of opioids for the relief of moderate to severe pain is considered necessary for most patients. For moderate pain, weak opioids (e.g., codeine or tramadol) or lower doses of strong opioids (e.g., morphine, oxycodone, or hydromorphone) are often administered and frequently combined with nonopioid analgesics. For severe pain, strong opioids are routinely used; although no agent has shown itself to be more effective than another, morphine is often considered the opioid of choice because of provider familiarity, broad availability, and lower cost.¹⁰

Fentanyl, a synthetic opioid 50 to 100 times more potent than morphine, is available in a variety of delivery methods to offer additional options for management of breakthrough pain. All rapid-acting fentanyl products are intended for use only in patients already tolerant to opioids. The risk of addiction with these rapid-onset agents has not been elucidated.

Methadone can be given via multiple routes (oral, intravenous, subcutaneous, and rectal); has a long half-life (13 to 58 hours) and rapid onset of action; and is inexpensive. Methadone may be particularly useful for the management of opioid-induced neurotoxicity, hyperalgesia, and neuropathic pain. Methadone is safer for patients with renal failure, and is preferred for those with known opioid allergies because it is a synthetic opioid. However, methadone also has several distinct disadvantages, including drug interactions. Given the complexities related to methadone administration, it is important that this opioid be prescribed by clinicians with experience who can provide careful monitoring.¹⁰

Adverse Effects of Opioids

Adverse effects from opioids are common and may interfere with achieving adequate pain control.

Central Nervous System (CNS)

Adverse effects on the CNS may be attributed to opioids' anticholinergic activity or direct effect on neurons. Sedation and drowsiness are common but typically transient adverse effects. The effects of opioids on cognitive or psychomotor functioning are not well established. Given the incidence of sedation, caution is exercised when an opioid is initiated or when dose escalation is required. Delirium is associated with opioids but is typically multifactorial in origin.^{10,15}

Respiratory Depression

Opioid-induced respiratory depression may be caused by a blunting of the chemoreceptive response to carbon dioxide and oxygen levels and altered mechanical

function of the lung necessary for efficient ventilation and gas exchange. Opioid-induced respiratory depression may manifest through decreased respiratory rate, hypoxemia, or increases in total exhaled carbon dioxide.^{10,16}

Nausea and Vomiting

Opioid-induced nausea occurs in up to two-thirds of patients receiving opioids, and half of those patients will experience vomiting.¹⁷ Opioids cause nausea and vomiting via enhanced vestibular sensitivity, via direct effects on the chemoreceptor trigger zone, and by causing delayed gastric emptying.¹⁸ Tolerance to opioid-induced nausea and vomiting (OINV) may develop, and symptoms typically resolve within 1 week.¹⁰

Constipation

Constipation is the most common adverse effect of opioid treatment, occurring in 40% to 95% of patients.¹⁹ It can develop after a single dose of morphine, and patients generally do not develop tolerance to opioid-induced constipation. Opioids cause constipation by decreasing peristalsis, which occurs by reducing gastric secretions and relaxing longitudinal muscle contractions, and results in dry, hardened stool.¹⁹

Constipation is exacerbated by dehydration, inactivity, and comorbid conditions such as spinal cord compression. Patients are encouraged to maintain adequate hydration, fiber intake, and regular exercise, in addition to taking laxatives.¹⁰

Opioid Endocrinopathy

Opioid endocrinopathy (OE) is the effect of opioids on the hypothalamic-pituitary-adrenal axis and the hypothalamic-pituitary-gonadal axis over the long term. Opioids act on opioid receptors in the hypothalamus, decreasing the release of gonadotropin-releasing hormone.²⁰ This results in a decreased release of luteinizing hormone and follicle-stimulating hormone, and finally a reduction of testosterone and estradiol released from the gonads. These effects occur in both men and women. Patients may present with symptoms of hypogonadism such as decreased libido, erectile dysfunction, amenorrhea or irregular menses, galactorrhea, depression, and hot flashes.¹⁰

Opioids and Addiction

Drug overdose deaths and opioid-involved deaths continue to increase in the United States. The majority of drug overdose deaths (more than six out of ten) involve an opioid.²¹ Deaths from prescription opioids—drugs like oxycodone, hydrocodone, and methadone—have more than quadrupled since 1999.²¹ Currently, 91 Americans die every day from an opioid overdose.²¹

Addiction is defined as continued, compulsive use of a drug despite harm. The following aberrant behaviors may suggest addiction or abuse; further assessment is required to make the diagnosis¹⁰:

- Aggressive complaining about the need for more drugs.
- Drug hoarding during periods of reduced symptoms.
- Acquiring similar drugs from other medical sources.
- Requesting specific drugs.
- Reporting psychic effects not intended by the physician.

- Resistance to a change in therapy associated with tolerable adverse effects accompanied by expressions of anxiety related to the return of severe symptoms.
- Resistance to referral to a mental health professional.
- Unapproved use of the drug to treat another symptom or use of the drug for a minor symptom (e.g., use of fentanyl for mild headache pain).
- Unsanctioned dose escalation or other nonadherence to therapy on one or two occasions.
- Unconfirmed multiple allergies to multiple opioids.

CDC 12 Recommendations for Using Opioids to Treat Chronic Pain²²

The CDC has developed the new Guideline for Prescribing Opioids for Chronic Pain to help primary care providers make informed prescribing decisions and improve patient care for those who suffer from chronic pain (pain lasting more than 3 months) in outpatient settings.

1. Nonpharmacologic therapy and nonopioid pharmacologic therapy are preferred for chronic pain. Clinicians should consider opioid therapy only if expected benefits for both pain and function are anticipated to outweigh risks to the patient. If opioids are used, they should be combined with nonpharmacologic therapy and nonopioid pharmacologic therapy, as appropriate.
2. Before starting opioid therapy for chronic pain, clinicians should establish treatment goals with all patients, including realistic goals for pain and function, and should consider how opioid therapy will be discontinued if benefits do not outweigh risks. Clinicians should continue opioid therapy only if there is clinically meaningful improvement in pain and function that outweighs risks to patient safety.
3. Before starting and periodically during opioid therapy, clinicians should discuss with patients known risks and realistic benefits of opioid therapy and patient and clinician responsibilities for managing therapy.
4. When starting opioid therapy for chronic pain, clinicians should prescribe immediate-release opioids instead of extended-release/long-acting (ER/LA) opioids.
5. When opioids are started, clinicians should prescribe the lowest effective dosage. Clinicians should use caution when prescribing opioids at any dosage, should carefully reassess evidence of individual benefits and risks when considering increasing dosage to ≥ 50 morphine milligram equivalents (MME)/day, and should avoid increasing dosage to ≥ 90 MME/day or carefully justify a decision to titrate dosage to ≥ 90 MME/day.
6. Long-term opioid use often begins with treatment of acute pain. When opioids are used for acute pain, clinicians should prescribe the lowest effective dose of immediate-release opioids and should prescribe no greater quantity than needed for the expected duration of pain severe enough to require opioids. Three days or less will often be sufficient; more than seven days will rarely be needed.
7. Clinicians should evaluate benefits and harms with patients within 1 to 4 weeks of starting opioid therapy for chronic pain or of dose escalation. Clinicians should evaluate benefits and harms of continued therapy with patients every 3 months or more

frequently. If benefits do not outweigh harms of continued opioid therapy, clinicians should optimize other therapies and work with patients to taper opioids to lower dosages or to taper and discontinue opioids.

8. Before starting and periodically during continuation of opioid therapy, clinicians should evaluate risk factors for opioid-related harms. Clinicians should incorporate into the management plan strategies to mitigate risk, including considering offering naloxone when factors that increase risk for opioid overdose, such as history of overdose, history of substance use disorder, higher opioid dosages.

9. Clinicians should review the patient's history of controlled substance prescriptions using state prescription drug monitoring program (PDMP) data to determine whether the patient is receiving opioid dosages or dangerous combinations that put him or her at high risk for overdose. Clinicians should review PDMP data when starting opioid therapy for chronic pain and periodically during opioid therapy for chronic pain, ranging from every prescription to every 3 months.

10. When prescribing opioids for chronic pain, clinicians should use urine drug testing before starting opioid therapy and consider urine drug testing at least annually to assess for prescribed medications as well as other controlled prescription drugs and illicit drugs.

11. Clinicians should avoid prescribing opioid pain medication and benzodiazepines concurrently whenever possible.

12. Clinicians should offer or arrange evidence-based treatment (usually medication-assisted treatment with buprenorphine or methadone in combination with behavioral therapies) for patients with opioid use disorder.

Other Interventional Approaches

While pharmacologic therapy using the World Health Organization (WHO) guidelines effectively manages most pain, approximately 10% to 20% of patients will have refractory pain or excessive side effects.¹⁰ For patients with refractory pain or specific regional pain syndromes, an interventional approach to treating pain has been proposed as the fourth step on the WHO pain relief ladder.¹⁰

Nerve Blocks

The celiac plexus block, used primarily for patients with upper abdominal pain, is the most commonly employed neurolytic blockade of the sympathetic axis, followed by the superior hypogastric plexus block and the ganglion of impar block for patients with lower abdominal or pelvic pain. Traditionally, the autonomic neural blockade was reserved for patients with inadequate response to oral opioids, but some researchers have suggested that the intervention—which is associated with decreased pain, reduced opioid consumption, improved performance status, and few complications—should be considered a first-line approach.¹⁰ For patients with regional pain, a peripheral nerve block infusing a local anesthetic can achieve local pain control. This approach can be

applied to any peripheral nerve, including the femoral, sciatic, paravertebral, brachial plexus, and interpleural nerves.¹⁰

Neuroaxial Delivery of Analgesia

When patients have pain that persists despite high doses of opioids and other analgesics or have intolerable side effects to oral opioids—such as delirium, sedation, or nausea—an alternative route of delivery may be considered. Compared with intravenous administration of opioids, epidural and intrathecal routes of delivery are 10 and 100 times more potent, respectively. Such routes of delivery allow high doses of analgesics to be administered with less systemic absorption and fewer side effects.¹⁰

Cordotomy

Cordotomy is reserved for pain refractory to other approaches and is done less commonly today. It is most effective in treating unilateral somatic pain from the torso to the lower extremities. The available literature suggests a high rate of efficacy, with 60% to 80% complete pain relief immediately after the procedure, falling to 50% at 12 months. Cordotomy is generally reserved for patients considered to be in the last 2 years of life, with pain refractory to other approaches, and may be done via the open route or the percutaneous route.¹⁰

Implanted Neurostimulation

Neurostimulation has been used for several decades and become increasingly popular for treatment of chronic intractable pain. A neurostimulator is a therapy with an implanted device consisting of leads and an implantable pulse generator (IPG). The leads are percutaneously inserted into epidural space or near to the peripheral nerve, where leads deliver electrical impulses. The IPG can be implanted in several different anatomic regions based on the patient or surgeon preference. The pain reduction from SCS may be associated with inhibition of neurotransmitters. Spinal cord stimulation and peripheral nerve stimulation have been widely used for various pain syndromes. The common device-related complications are depletion of pulse generator and lead migration. Other complications include pain at the IPG site, pain at the anchoring site and infection. Some of these complications are preventable in the well-trained surgeons.³

Physical Medicine and Rehabilitation

Passive Modalities

Passive treatment modalities focused solely on temporarily decreasing pain symptoms (e.g., heat treatments, cryotherapy, transcutaneous electrical nerve stimulation [TENS]) should be used sparingly as part of the physical therapy intervention. These modalities should be a means to an end, the end being decreasing pain to a sufficient extent to

allow patients to participate in subsequent active treatments aimed at positively affecting functional abilities.²³

Thermal Modalities

Superficial heating agents (e.g. hot packs, warm hydrotherapy, paraffin, fluidotherapy and infrared) or deep heating agents (e.g. short-wave and microwave diathermy, and ultrasound) can be used to increase blood flow, membrane permeability, tissue extensibility and joint range of motion in ways that can contribute to decreasing pain. Heat and cold alter both peripheral and central nervous system excitability, and can thus serve as a means of modulating pain.²³

Electric Current Modalities

Possible action mechanisms of electrotherapy have been suggested to be local release of neurotransmitters such as serotonin, raised levels of ATP, release of endorphin and pain reduction via gate theory. Dorsal column activation is another possible mechanism of electrotherapy. It has also been postulated that low frequency currents may increase microcirculation and endoneural blood flow.²⁴ Some of the most common electric current modalities used to treat pain are:

- Transcutaneous electrical neural stimulation (TENS)
- Electronic muscle stimulator (EMS)
- Interferential current stimulator
- High voltage pulsed galvanic stimulation (HVPGS), and
- Microcurrent electrical neuromuscular stimulation (MENS)

Exercise

Various forms of exercise can modulate pain either directly or indirectly. Passive or active exercise has a direct effect on pain through increasing input from joint mechanoreceptors. Indirect effects of exercise on pain may be related to increased blood flow, decreased edema, inhibition of muscle spasm, enhanced ROM, flexibility, strength and weight loss which may improve biomechanical factors and decrease joint stress. Improved sleep, enhanced mood, relaxation, reduction in anxiety and general well-being following regular exercise also can alter pain sensitivity positively in same way. An exercise program should address primary functional problems and impairments (pain, limited joint range of motion, muscle weakness) for functional independence. Flexibility, strength and aerobic endurance are the basic components for exercise programs aiming to control pain.²⁵ Improving the flexibility of the muscles, tendons and ligaments increases the range of motion and assists with the patient's functional movement. Increasing muscular strength provides stabilization and improved biomechanics of the head, neck, trunk, and limbs. Aerobic exercise increases the blood flow and nutrients to the soft tissues, improving the healing process and reducing stiffness.

Manual Therapies

Massage Therapy

Massage therapy (MT) is defined as a therapeutic manipulation of soft tissue using the hands or a mechanical device. It may be the earliest and most primitive tool to improve pain. Some of the most common types of MT include Swedish massage, Shiatsu, Rolfing, reflexology, myofascial release, and craniosacral therapy. MT delivered to soft and connective tissues may induce local biochemical changes that modulate local blood circulation, improve muscle flexibility, intensify the movement of lymph, and loosen adherent connective tissue, which may alternately improve reuptake of local nociceptive and inflammatory mediators. These local effects may subsequently influence neural activity at the spinal cord segmental level, thereby modulating the activities of subcortical nuclei that influence pain perception.²⁶

Spinal Manipulation

Spinal manipulation—sometimes called “spinal manipulative therapy”—is practiced by many health care professionals including chiropractors, osteopathic physicians, naturopathic physicians, physical therapists, and some medical doctors. The amount of force applied depends on the form of manipulation used. The goal of the treatment is to relieve pain and improve physical functioning.²⁷ A review of scientific evidence on spinal manipulation for a range of conditions concluded that spinal manipulation/mobilization may be helpful for several conditions in addition to back pain, including migraine and cervicogenic (neck-related) headaches, neck pain, upper- and lower-extremity joint conditions, and whiplash-associated disorders.²⁸

Dry Needling

Muscle trigger points (MTrPs) are found to be involved in the pain process whether the pain is in the trunk, neck, arm, or leg. Monofilament needles are used with dry needling and are inserted into MTrPs by touching, tapping or pricking the tip of the needle into the skin. MTrPs are defined as taut bands that are hypersensitive areas in the body and are painful upon palpation. MTrPs are classified as either active or latent. When palpated, active MTrPs may elicit referred pain, reproducing the patient’s symptoms; whereas, latent MTrPs do not cause a pain that will reproduce the patient’s symptoms but could be responsible for tightness of the muscle. Dry needling is used for myofascial pain, lower and upper extremity pain, neck pain, back pain, headaches, jaw pain, and migraines, among other conditions.²⁹ Dry needling is used as an adjunct for the management of pain and dysfunction in numerous neuromusculoskeletal conditions. Needling provides a hyperstimulation with an analgesic effect that is similar to heat and cold compress causing changes of the physiological responses within skin. It has been proposed that with needle insertion there is a disruption of nociceptors which will elicit a local twitch response within the MTrPs causing a disruption of the pain cycle with notable pain relief. Using dry needling in combination with various manual therapy techniques is considered when treating both MTrPs and non-trigger point conditions.²⁹

Mind-Body Therapies

Mind-body medicine emphasize in engaging both in mind and body to promote stress reduction and well-being by changing the manner in which patients respond to their stressors. Any intervention that changes a patient's mental or emotional state will produce corresponding changes in the body and could therefore be called a "mind-body" intervention.³⁰ Thus, mind-body therapies can also be used to treat and/or prevent a variety of conditions, including chronic pain disorders.³⁰ The National Center for Complementary and Alternative Medicine defines mind–body medicine in the following way: Mind-body medicine focuses on the interactions among the brain, mind, body, and behavior, and the powerful ways in which emotional, mental, social, spiritual, and behavioral factors can directly affect health. It presents as a fundamental approach that respects and enhances each person's capacity for self-knowledge and self-care, and it emphasizes techniques that are grounded in this approach. Some of the most common mind-body therapies are: meditation, guided imagery, hypnosis, tai chi (TC), qi gong, and yoga.²³

Other Pain Therapies

Acupuncture

Acupuncture, among the oldest healing practices in the world, is part of traditional Chinese medicine. Acupuncture practitioners stimulate specific points on the body—most often by inserting thin needles through the skin. In traditional Chinese medicine theory, this regulates the flow of qi (vital energy) along pathways known as meridians.³¹ It is believed that acupuncture stimulation activates larger A beta nerve fiber which then "gates" the nociceptive signals from A delta and C nerve fibers in substantia gelatinosa (SG) in spinal dorsal horn. However, gate control theory can only explain transient and local analgesia within the same and adjacent dermatomes where painful areas are. Gating control cannot produce distal and prolonged analgesia. Therefore, widespread regulation elicited by acupuncture at spinal and supraspinal levels has been the central question to be addressed for decades.³²

Cognitive Behavioral Therapy (CBT)

The American Psychological Association recognizes cognitive-behavioral therapy (CBT) as an empirically supported intervention in management of chronic musculoskeletal pain; including rheumatoid arthritis, osteoarthritis, fibromyalgia, and low back pain. Its foundation is the gate control theory integrating the sensory, affective, and cognitive components of pain. Cognitive processes are thoughts, self-statements, or evaluations about the pain and beliefs, interpretations, or attributions regarding this condition. Commonly used CBT interventions included activity pacing and pleasurable activity scheduling.²³ Cognitive Behavioral Therapy (CBT) can be done in groups or individually. This treatment is often provided at an interdisciplinary pain program. Treatment with CBT does not imply that there is a concurrent psychiatric diagnosis; the treatment can

be considered to address psychosocial issues often associated with non-acute pain irrespective of any other documented or presumed psychopathology.¹⁴

Bio-feedback

Biofeedback is a form of behavioral medicine that helps patients learn self-awareness and self-regulation skills for the purpose of gaining greater control of their physiology. Electronic instrumentation is used to monitor the targeted physiology and then displayed or fed back to the patient through visual, auditory or tactile means, with coaching by a biofeedback specialist. Treatment is individualized to the patient's diagnosis and needs. The goal of biofeedback treatment is the transfer of learned skills to daily life. Candidates for biofeedback therapy or training must be motivated to learn and practice biofeedback and self-regulation techniques. Biofeedback is not appropriate for individuals suffering from acute pain or acute injury. It may be appropriate for non-acute pain when combined with a program including functional restoration.¹⁴

Self-Management

Self-Management (SM) is defined as actions taken by the patient to manage or minimize the impact of a chronic condition on everyday life.³³ The term management is key; by skillfully handling the disease condition, the individual can get back to life even while in pain. The basic tenets of SM include³³:

- active participation by the patient
- treatment of the whole person, not just the disease
- empowerment of the patient

Each therapy uses a combination of skills to allow patients to successfully manage their disease, including³³:

- pain medication management
- coping skills
- relaxation techniques
- problem solving
- enhancement of the patient's social network

The skills needed are determined by the physical and psychological status of the participants. Because of the chronic nature of pain and other conditions, SM is considered not only a strategy, but also an outcome. Success in chronic pain therapy is measured not as pain elimination, but as functioning in spite of pain. By focusing on empowerment, the person experiencing pain can maintain wellness and continue with life and work. Increasing the use of SM in the treatment of chronic pain will help the individual transition from disabled patient to functioning person. SM involves treatment for the chronic condition and its physical and emotional consequences while maintaining meaningful life roles. Education in SM helps individuals to optimally manage their condition, minimize long-term consequences, and achieve the best possible quality of life. The core skills of SM are³³:

- Problem solving—skills to solve everyday problems arising because of illness
- Decision-making—the ability to make sound decisions regarding chronic disease management
- Resource utilization —knowledge about available resources
- Partnership formation—skills to build and maintain partnerships to best address illness
- Taking action—the ability to set and achieve goals via action plans.

Palliative Care Referral

Palliative care, which is specialized medical care for people with serious illnesses with the goal to maximize quality of life for both patients and families, can provide expert assessment and management of pain and other non-pain symptoms. Palliative care providers work in interdisciplinary teams that include physicians, nurses, mental health specialists, social workers, chaplains, therapists and sometimes pharmacists and dietitians. For patients with refractory pain, prominent non-pain symptoms, or intense psychosocial distress, a referral to palliative care may be appropriate, where available. Palliative care specialists may also help manage patients with multiple comorbidities, those requiring higher doses of opioids, and those with a history of substance abuse or complex psychosocial dynamics that can complicate the management of pain and adherence to recommended medications.¹⁰

Improving Pain Management Knowledge

Several steps are needed to improve the understanding of pain and its management by clinicians, for the benefit of the patient in pain. They include:¹

1. increase pain curricular time in all health professional programs,
2. utilize current pain curricula developed by national and international pain-related organizations,
3. ensure pain is taught within a biopsychosocial framework and in an integrated interdisciplinary manner that reflects its multidimensional nature,
4. ensure there is sufficient coverage of pain in accreditation requirements of health professional programmes and in practice standards for healthcare professionals, hospitals, and other healthcare facilities,
5. synthesize new pain-related information for widespread readership by healthcare professionals, and
6. ensure effective knowledge transfer and application about pain and its management.

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Pain & Symptom Management

Post-Test

1. Which of the following is an opioid neurotransmitter? (p. 4-5)
 - A. Glutamate
 - B. GABA
 - C. Norepinephrine
 - D. Endorphin
2. Which of the following is NOT a pathophysiologic mechanism of pain? (p. 6-7)
 - A. Nociceptive
 - B. Chemolytic
 - C. Neuropathic
 - D. Psychogenic
3. Which pain assessment tool would be most appropriate for an individual with mild – moderate cognitive impairment? (p. 9-10)
 - A. Faces Pain Scale
 - B. Numeric Rating Scale
 - C. Visual Analog Scale
 - D. Brief Pain Inventory
4. A person has effective pain relief with Acetaminophen or NSAIDS. They would be on Step ____ of the WHO Pain Relief Ladder. (p. 12)
 - A. 1
 - B. 2
 - C. 3
 - D. 4
5. Which of the following is the most potent form of opioid medication? (p. 15)
 - A. Tramadol
 - B. Fentanyl
 - C. Morphine
 - D. Oxycodone
6. Which of the following is NOT a common adverse effect of opioid medication? (p. 15-17)
 - A. Respiratory depression
 - B. Cardiac arrhythmias
 - C. Nausea
 - D. Constipation

7. According to the CDC's guidelines, opioids used to treat acute pain are typically needed for ___ days or less; and are rarely needed for more than ___ days.
(p. 17-18)
- A. 3; 7
 - B. 5; 10
 - C. 7; 10
 - D. 7; 14
8. Diathermy, ultrasound, and HVPGS are all considered _____ modalities and should be used _____ to decrease pain and facilitate functional abilities.
(p. 19-20)
- A. thermal; frequently
 - B. passive; sparingly
 - C. electric current; progressively
 - D. active; simultaneously
9. Activity pacing and pleasurable activity scheduling are interventions used in which type of therapy? (p.22-23)
- A. Mind-body therapy
 - B. Guided imagery
 - C. Cognitive behavioral therapy
 - D. Biofeedback
10. Which of the following is NOT one of the core skills of self-management? (p. 23-24)
- A. Problem-solving
 - B. Resource utilization
 - C. Somatic acceptance
 - D. Partnership formation

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