Do No Harm: Putting Safer Pain Management Guidelines into Practice – Module 3

1.1 Introduction

Welcome to the Oklahoma Primary Healthcare Improvement Cooperative's online course for clinicians - Do No Harm: Putting Safer Pain Management Guidelines into Practice.

This Online Enduring Material educational program is designed for healthcare professionals. The contents of this program are taken from the National Academy's Institute of Medicine's white paper Pain Management and the Opioid Epidemic: Balancing Societal and Individual Benefits and Risks of Prescription Opioid Use and the 2016 CDC, 2017 VA-DOD, and 2017 Oklahoma State Department of Health Guidelines for Pain and Opioid management, and Oklahoma law.

The program was developed through a grant from the Oklahoma Department of Mental Health and Substance Abuse Services by the Oklahoma Primary Health Care Cooperative of The University of Oklahoma Health Sciences Center and the OU-TU School of Community Medicine, and released in August, 2019.

1.2 Pain

Hi, I am Steve Crawford, and I will be your guide through module 3: Pain.

Module 3 - Pain, provides an overview of what clinicians may not have learned in school about the most common symptom and disease in practice. By understanding the neurobiology, psychology, and sociology involved in the expression of pain, clinicians will be better prepared to correctly diagnosis painful conditions and make appropriate recommendations for treatment. This module takes about 15 minutes to complete and provides ¼ hour of CME credit.

1.3 Planning and review Committees

The panel of experts who reviewed this course represent primary care clinicians, pharmacists, educators, and specialists in pain, addiction, and palliative care, and a national expert in the epidemiology of the opioid crisis.

1.4 Relevant Disclosure and Resolution

None of the members of the CME Planning committee have a relevant financial relationship or affiliation with commercial interests to disclose.

1.5 Relevant Disclosure and Resolution for Expert Review Panel

None of the expert reviewers have a relevant financial relationship or affiliation with commercial interests to disclose.

1.6

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Accommodation Statement:

Accommodations are available by contacting Jan Quayle at 405-271-2350, ext. 8 or e-mail to: jan-quayle@ouhsc.edu.

1.7 Professional Practice Gap Being Addressed

The knowledge gap being addressed in this module is: Oklahoma healthcare providers may be unaware of the recent advances in the neurobiology, pathophysiology, and psychology of the experience of pain and its neural modulation particularly of chronic pain.

1.8 Objectives

At the completion of this module, you will be able to describe pain as being a complex neurophysiologic and social experience.

You will be able to differentiate acute from chronic pain, and nociceptive from neuropathic pain.

You will be able to describe the basic pain physiology, neural circuits and neurotransmitters involved in acute and chronic pain.

Lastly, you will be able to define the role of opioid treatment in chronic painful conditions by understanding their neurobiology of analgesia, tolerance, dependence, and development of opioid use disorder.

1.9 What is Pain?

Pain is a sensory, emotional, cognitive, and motor phenomenon that motivates action in response to actual or perceived tissue or bodily injury. Perception of pain results from a complex of neural, glial, and cellular connections with both ascending and descending components in the spinal cord that modulate transmission of pain signals.

Pain is experienced when neural signals from the peripheral and central nervous system integrate with thoughts, memories, and emotions. Although pain often begins peripherally (such as whacking your hand against a door) it is not recognized until sensory nerve impulses reach the thalamus, from which impulses stimulate many brain regions to create the experience of pain.

1.10 Peripheral, Spinal, & Brain Pain Pathways

This diagram displays the peripheral and spinal pain pathways.

· Products of inflammation activate nociceptors at the tip of A-delta and C sensory neurons.

· The stimuli are converted into electrical signals rapidly transmitted along A-delta fibers and more slowly along C fibers of peripheral nerve axons.

· The cell bodies of sensory nerves are located in the dorsal root ganglion.

· The distal axon of sensory nerves leaves the dorsal root ganglion and enters the spinal cord dorsal horn grey matter where dendrites release excitatory neurotransmitters and synapse with adjacent neurons to amplify and distribute the nociceptive signal.

· Some signals stimulate the lower motor neurons of the anterior horn causing reflex muscle contraction to stabilize injured tissue.

· Activated post-synaptic spinal neurons cross the central spinal grey matter transmitting the nociceptive signal up the contralateral spinothalamic tracks.

· These fibers split, divide, and project into and through multiple brain nuclei within the pons, midbrain, and thalamic regions.

· The nociceptive impulse is sensed ~~as pain~~ in the thalamus where the signal is further amplified and distributed throughout the central nervous system to add feeling, meaning, thoughts, and reflexes or deliberate actions. The sensation of pain is thus experienced throughout the nervous system.

Returning to the mid-brain, signals stimulate neurons which release inhibitory neurotransmitters on the dorsal horn cells to dampen the incoming noxious stimuli.

1.11 Dynamic Regulation of Nociception

Nociceptive input from peripheral sensory nerves is dynamically regulated by neurons synapsing with descending spinal neurons to either amplify or dampen the neural signals and bringing nociceptive impulses under the "direction" the cortex. Please select the buttons to learn more.

· *Endogenous Opioids* - Neurons from the periaqueductal grey (PAG) release endogenous opioids (encephalin, endorphin, and dynorphin) throughout the spinal cord and in particular on the dorsal horn synapses.

· *Dampen Transmission* - Endogenous opioids dampen incoming sensory impulses in the dorsal horn grey matter. Opioids medications also work by dampening transmission from these dorsal horn cells.

· *Dopamine Surges* - Other neurons from the PGA terminate in the Ventral Tegmental Area (VTA) where surges of dopamine (DA) release to add salience and motivation to the pain experience. The action of opioid medications through this pathway also produce surges of dopamine playing an important role in how opioids used for pain control promote addiction to opioids. 2016 - In 2016 there were 97.9 prescriptions per 100 persons.

Serotonin and Noradrenalin - Serotonin and noradrenalin, arising from lower brain stem centers also inhibit nociceptive transmission and are responsible for the attenuation of chronic pain produced by antidepressants. The clinical effect on pain reduction usually begins only after days or weeks of treatment.

1.12 Expression of Pain

How patients sense and communicate their pain varies across individuals, cultures, and contexts. For example, two people with equivalent tissue injury may experience and report different pain levels and use different emotional messages to communicate suffering. This is commonly seen in degenerative diseases such as osteoarthritis in two patients with identical radiologic findings. One may have no pain while the other may have severe pain, suffering and disability.

Expression of pain may be a manifestation of depression, rather than a call for relief from tissue injury. Depression presenting as pain is particularly common in older adults and pain may resolve with treatment for depression.

Some patients may express pain to obtain attention or sympathy or to exercise control in relationships. Patients suffering from opioid use disorder may engage in desperate behavior to avoid withdrawal. This may include falsely claiming to have severe pain. Some may intentionally injure themselves to obtain opioids.

1.13 Clinical Types of Pain

Acute pain is usually a sudden, progressive, and predictable sensory response to tissue injury. As shown in the diagram, acute pain is influenced mostly by tissue injury with thoughts and emotions modestly affecting pain expression. Most people experiencing acute pain recognize that their symptoms will go away in a few days or weeks, which often prevents 'catastrophizing' the experience.

Chronic pain may begin as a symptom of inflammation or injury that does not resolve as expected. The pain lasts for more than 3 months. As shown in the diagram, chronic pain has a strong component of thought and emotional activation with tissue injury.

A CDC analysis of 2016 data estimated that chronic pain is common with 20.4% of U.S. adults having chronic pain and 8% have high impact pain that interferes with daily function and uses increased healthcare resources.

1.14 Approach to Chronic Pain Management

Let's turn now to recommendations for Chronic Pain. The first recommendation is to use a holistic approach to pain management.

Recommend lifestyle changes to improve overall health through better diet, weight loss, aerobic and strengthening exercises, sleep hygiene and stopping smoking.

Treat chronic pain using modalities that work throughout the nervous system. The multidisciplinary pain plan includes mind-body therapy such as cognitive-behavioral therapy,

mindfulness meditation training, massage therapy, acupuncture, chiropractic, and osteopathic manipulation. Physical medicine and rehabilitation treatments are also effective. Interventional therapy should be reserved for pain management of refractory conditions. Reserve opioid medications as a last resort, and only if expected benefits for both pain and function are anticipated to outweigh risks to the patient.

1.15 Chronic Pain Syndrome

Chronic pain syndrome differs considerably from acute pain although it may evolve following an episode of acute disease or injury. Chronic pain syndrome may have no demonstrable tissue injury, but persists as a psycho-sensory experience of either continuous or intermittent pain for months or years.

Chronic pain is associated with reduced physical functioning and poor quality of life. It is a major cause of work absenteeism, poor health, and increased medical encounters. Chronic pain is associated with comorbidities including impaired memory, cognition, attention, sleep disturbances, as well as psychiatric and behavioral disorders such as depression, anxiety, personality disorders, and somatization. The negative impact of chronic pain disproportionally affects disadvantaged populations with barriers to access to quality pain treatment.

1.16 Common Chronic Painful Conditions

Primary care physicians treat many conditions associated with chronic pain. Each of these respond best to a multi-modal pain plan that improves overall health, function, and wellbeing. From 2013 to 2015, 54.4 million people or 23% of the US population were diagnosed with arthritis according to the CDC. Back pain is one of the main reasons people visit a primary care physician and it is often associated with psychological and behavioral difficulties that benefit from multi-modal therapy. Fibromyalgia, with widespread waxing and waning pain and tenderness, affects approximately 2% of the population, mostly women. Patients are fatigued and usually have sleep disturbances. Analgesics play only a small role in this condition. Inflammatory diseases, complex regional pain syndrome, neuropathy, and spinal cord or brain disease usually benefit from specialized multimodal therapy to improve function.

Patients and physicians often focus on physical symptoms, especially somatic pain, associated with psychiatric disease leading to over use of analgesics and inadequate management of stress, depression, anxiety, or other chronic mental health disorders.

1.17 Social Pain Determinates of Health

The chronic pain associated with mental or social distress in the face of poverty, unemployment, lack of opportunity, and hopelessness brings patients to primary care practices that are ill-equipped to meet these mental health and social service needs. People living in rural communities have been disproportionally affected. Treating the "pain" of these conditions with medications rather addressing root solutions, distorts the use of medical care and reduces the community's overall health.

1.18 Pain and Toxic Stress

Every child and adult experiences stress. Positive stress, such as studying for a test, and tolerable stress, such as the death of a loved one while surrounded by supportive family, do not change the neurobiology of the brain, while chronic toxic stress, such as long periods of not having enough to eat, daily physical or emotional abuse, living with domestic violence, sexual abuse and subsequent fear of future events, can result in changes to the brain of a developing child. Brain connections are weakened as is the ability to use the prefrontal cortex to calm fears. Adverse childhood experiences (ACEs) can create a state of constant arousal and an inability to modulate, tolerate, or recover from stress, fear, or other emotional experiences or states. Studies have demonstrated that an ACE score of 4 or greater predicts an enhanced pain response and risk of substance use disorder later in life.

1.19 Summary

In summary, pain is a psycho-sensory experience of tissue injury, it may be nociceptive or neuropathic which in turn may be due to damage of peripheral nerves or damage to the central nervous system. Acute pain is usually the result of tissue injury while chronic pain is a more poorly understood total neurological system condition. The experience of pain is under complex brain neurotransmitter control, and is intimately tied to memories, past experiences, and emotions. Pain associated with mental illness, psychological or emotional distress, social isolation, and poverty may confuse treatment decisions by focusing on treating the symptom of pain rather than the root cause of suffering.

1.20 Instructions

Please answer the following self-assessment question by selecting the single best answer. You will receive immediate feedback and if you selected an incorrect response you may answer the question again.

1.21

Question 1

1.22

Question 2

1.23

Question 3

1.24

Question 4

1.25 Resources

The references on this slide provide additional resources for making changes in practice systems needed to implement practice guidelines.

1.25 Closing Instructions

The University of Oklahoma Office of Professional Development is providing CE credits. MDs are eligible for AMA PRA Category 1 Credit, Physician Assistants for AAPA Category 1 Credit, and Nurse Practitioners for AANC contact hours and Oklahoma pharmacology hours.

Until March 1, 2020, the University of Oklahoma will waive the $25 fee.

Click on the web link which will take you to the Office of Professional Development web site where you may register, take a test of knowledge, evaluate your learning experience, and print your CE certificate.