Acute pain management in dermatology

Mechanisms and pathways

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Learning objectives
Define acute pain and distinguish acute pain from chronic pain; define the three categories of acute pain: preoperative pain, operative pain, and postoperative pain; delineate best practices for clinical assessment of the three categories of pain; and discuss the strengths and weaknesses of the visual analog scale as applied to acute pain in dermatology.

Disclosures
Editors
The editors involved with this CME activity and all content validation/peer reviewers of the journal-based CME activity have reported no relevant financial relationships with commercial interest(s).

Authors
The authors involved with this journal-based CME activity have reported no relevant financial relationships with commercial interest(s).

Planners
The planners involved with this journal-based CME activity have reported no relevant financial relationships with commercial interest(s). The editorial and education staff involved with this journal-based CME activity have reported no relevant financial relationships with commercial interest(s).

The number of dermatologic surgical procedures performed is increasing each year. The pain associated with these procedures is a major concern for patients and its treatment is part of the increasing emphasis on outcomes and quality of clinical care. Better understanding of pain signaling and how commonly used analgesics function can help improve our surgical pain management. This is part I of a 2-part review that will highlight the anatomy of acute pain signaling from the skin to the central nervous system and the factors that influence the plasticity of the pathway. Having this foundation of knowledge is needed to enhance the clinical treatment of pain. Part II will provide an updated review of available treatments, with an emphasis on their appropriate use for postsurgical pain management. (J Am Acad Dermatol 2015;73:533-40.)

Key words: analgesic; anesthetic; chronic pain; hyperalgesia; pain; surgery; transduction.

INTRODUCTION

Key point

- **As the number of dermatology surgical procedures increases, adequate perioperative pain control should be a goal of care**

It is estimated that 75% of patients undergoing any surgery in the United States experience inadequate pain control.[1] In response, national, state and local health care guidelines support perioperative pain control measures as a part of patient safety initiatives. As the number of dermatology procedures in the United States increases year after year,[2] dermatologists should be aware of the mechanisms of acute pain and the interventions to control patient's perioperative pain.

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Funding sources: None.
Conflicts of interest: None declared.

The views expressed in this article are those of the author(s) and do not necessarily reflect the official policy or position of the Department of the Navy, Department of Defense, or the United States Government.

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Accepted for publication March 13, 2015.

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0190-9622/$36.00

http://dx.doi.org/10.1016/j.jaad.2015.03.061
Date of release: October 2015
Expiration date: October 2018

Abbreviations used:

CNS:	central nervous system
COX:	cyclooxygenase
GABA:	gamma-aminobutyric acid
NSAID:	nonsteroidal antiinflammatory drug
PAG:	periaqueductal grey
RVM:	rostral ventromedial medulla
STT:	spinothalamic tract
TRP:	transient receptor potential

In part I of this continuing medical education article we review the anatomy and mechanisms of pain signaling from the skin to the central nervous system. We will break down the acute pain pathway into 4 main categories: cutaneous transduction, spinal cord processing, cortical perception, and descending regulation from the cortex. We will then discuss the plasticity of pain signaling via various regulatory factors and how they influence acute and chronic pain. This will allow better understanding for pain management, which will be discussed in Part II.

OVERVIEW OF ACUTE PAIN

Key points

- **Pain sensation is evolutionarily advantageous, serving as a survival mechanism**
- **The pathway of acute pain can be divided into transduction, transmission, and perception**

Evolutionarily, acute pain is a protective mechanism.[3] It is a noxious sensation of short duration resulting from the sudden onset of traumatic injury, internal illness, or surgical procedure.[4] Existentially, acute pain means "do that again at your own risk." Pain sensation is the result of an exquisitely complex and dynamic peripheral and central neurologic system. The perception of pain is a subjective entity influenced by setting, past experiences, affect, sex, and cultural and cognitive factors.[5] Disruption of the intricate balance of biologic and psychosocial aspects of pain can result in chronic pain.[6] Acute pain and chronic pain are distinct, and a better understanding of their basic mechanisms can help guide more successful clinical treatment.

Pain can be simplified into 3 main processes: transduction, transmission, and perception. Transduction at the skin is the process of transferring a painful stimulus into electrical neuronal activity. Transmission through the spinal cord is the propagation of that electrical activity to the central nervous system (CNS). Perception within the brain is the final part of the pathway that leads to the subjective nature of pain that patients experience (Fig 1).[7]

CUTANEOUS TRANSDUCTION

Key points

- **There are 3 main primary sensory afferent fibers: Aβ, Aδ, and C**
- **Transducers are membrane receptors on primary afferents that are responsible for the sensation of sensory stimuli**
- **Nonneuronal skin cells have transducers and influence pain sensation**
- **Local anesthetics bind transducers to induce anesthesia**

The skin is more densely innervated by, and contains more varied types of, sensory afferents compared to other bodily tissues.[5] Cutaneous nerve fibers are highly specialized structures that work collectively with other tissues to transmit sensory information to the CNS.[8] They have cell bodies in the dorsal root ganglion, peripheral terminals at target tissues, and central terminals in the spinal cord.[6] This design allows rapid transmission from the periphery to the spinal cord.[9]

There are 3 broad categories of afferent sensory fibers: Aβ, Aδ, and C fibers (Table I).[8] Aβ fibers respond to innocuous stimuli and detect texture, vibration, and light pressure.[6,10] They are fast-conducting, large diameter, myelinated afferents that have nerve endings that often associate with nonneuronal cells (eg, Merkel cells, Pacinian corpuscles, and hair follicles) in their target tissues.[6,11] Aδ and C fibers respond to potentially injurious or noxious stimuli and are referred to as nociceptors.[8] Nociceptors are free nerve endings because they do not directly associate with other cells or tissues at their peripheral terminals.[11] Aδ fibers are medium diameter, lightly myelinated, and represent initial, sharp, localized pain.[6] C fibers are small diameter, unmyelinated nociceptors that represent more diffuse, dull, aching pain.[6]

Transducers are membrane receptors on nociceptors that respond to specific stimuli (ie, thermal, mechanical, and/or chemical), allowing transduction of a stimulus into electrical activity. Transducers are either associated with a membrane ion channel (ionotropic receptors) or associated with a second messenger-signaling cascade (metabotropic receptors). Ionotropic receptors rapidly transmit sensory information because of their association with ion channels that generate action potentials. Metabotropic receptors are slower responders, but their second messengers can have profound effects on ionotropic receptor function.[11] Knowledge about transducer families, such as transient receptor potential channels (TRPs) and acid-sensing ion channels, has grown exponentially in recent years. There