Global Year Against Acute PAIN

OCTOBER 2010 – OCTOBER 2011

Mechanisms of Acute Pain

Introduction

Patients with surgery, injury, childbirth, and acute illness experience pain caused by damage to a variety of tissues. Commonly injured tissues include skin, muscle, bone, tendons, ligaments, and visceral organs. Symptoms vary depending upon the type of tissue injured and the extent of the injury. Sensory pathways for pain caused by tissue damage transmit information from the damaged tissue to the central nervous system (CNS).

Nociceptor Activation, Sensitization, and Hyperalgesia in Acute Pain

- Nociceptors are sensory receptors that respond to tissue damage. Nociceptors respond during and after acute events such as surgery, injury, childbirth, and acute illness [3].
- Nociceptors have unique response properties that depend on the organ that they innervate. These unique properties, in part, provide the basis for differences in clinical acute pain states after different organ injuries.
- Sensitization is a characteristic of nociceptors in which responses to stimuli are enhanced at the site of injury [3].
- Nociceptor sensitization produces primary hyperalgesia at the site of injury, which generates ongoing pain at rest and enhanced pain during and after surgery, injury, childbirth, and acute illness [2,4].

Mediators of Nociceptor Activation and Sensitization in Acutely Injured Tissue

- Substances that are released during acute injury and cause acute pain are not entirely known.
- Prostaglandins released by tissue trauma sensitize nociceptors [1].
- Other mediators of nociceptor activation and sensitization include nerve growth factor, which is increased in incisions and also sensitizes nociceptors [1].
- Additional factors thought to contribute to acute pain are acidity, interleukins, and cytokines.
- In some cases, nerves may be directly injured and become activated.

Central Sensitization and Acute Pain

- Nociceptive input during and after surgery, injury, childbirth, and acute illness can enhance the responses of pain-transmitting neurons in the CNS, amplifying clinical pain [5].
- Increased responsiveness of nociceptive neurons in the CNS to normal or subthreshold afferent input is termed “central sensitization” [3].
- The magnitude of central sensitization depends on many factors, including the type tissue and the extent of the injury.
- Central sensitization amplifies transmission of input from peripheral tissues and produces secondary hyperalgesia, an increased pain response evoked by stimuli applied to tissue outside the area of injury [2].
- Central sensitization is expressed in a variety of other forms that include both spinal and supraspinal mechanisms.

References

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