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Human brain mechanisms of pain perception and regulation in health and disease.
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Source

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Abstract

CONTEXT:

The perception of pain due to an acute injury or in clinical pain states undergoes substantial processing at supraspinal levels. Supraspinal, brain mechanisms are increasingly recognized as playing a major role in the representation and modulation of pain experience. These neural mechanisms may then contribute to interindividual variations and disabilities associated with chronic pain conditions.

OBJECTIVE:

To systematically review the literature regarding how activity in diverse brain regions creates and modulates the experience of acute and chronic pain states, emphasizing the contribution of various imaging techniques to emerging concepts.

DATA SOURCES:

MEDLINE and PRE-MEDLINE searches were performed to identify all English-language articles that examine human brain activity during pain, using hemodynamic (PET, fMRI), neuroelectrical (EEG, MEG) and neurochemical methods (MRS, receptor binding and neurotransmitter modulation), from January 1, 1988 to March 1, 2003. Additional studies were identified through bibliographies.

STUDY SELECTION:

Studies were selected based on consensus across all four authors. The criteria included well-designed experimental procedures, as well as landmark studies that have significantly advanced the field.

DATA SYNTHESIS:

Sixty-eight hemodynamic studies of experimental pain in normal subjects, 30 in clinical pain conditions, and 30 using neuroelectrical methods met selection criteria and were used in a meta-analysis. Another 24 articles were identified where brain neurochemistry of pain was examined. Technical issues that may explain differences between studies across laboratories are expounded. The evidence for and the respective incidences of brain areas constituting the brain network for acute pain are presented. The main components of this network are: primary and secondary somatosensory, insular, anterior cingulate, and prefrontal cortices (S1, S2, IC, ACC, PFC) and thalamus (Th). Evidence for somatotopic organization, based on 10 studies, and psychological modulation, based on 20 studies, is discussed, as well as the temporal sequence of the afferent volley to the cortex, based on neuroelectrical studies. A meta-analysis highlights important methodological differences in identifying the brain network underlying acute pain perception. It also shows that the brain network for acute pain perception in normal subjects is at least partially distinct from that seen in chronic clinical pain conditions and that chronic pain engages brain regions critical for cognitive/emotional assessments, implying that this component of pain may be a distinctive feature between chronic and acute pain. The neurochemical studies highlight the role of opiate and catecholamine transmitters and receptors in pain states, and in the modulation of pain with environmental and genetic influences.

CONCLUSIONS:

The nociceptive system is now recognized as a sensory system in its own right, from primary afferents to multiple brain areas. Pain experience is strongly modulated by interactions of ascending and descending pathways. Understanding these modulatory mechanisms in health and in disease is critical for developing fully effective therapies for the treatment of clinical pain conditions.

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