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Peripheral input and its importance for central sensitization

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Abstract

Many pain states begin with damage to tissue and/or nerves in the periphery, leading to enhanced transmitter release within the spinal cord and central sensitization. Manifestations of this central sensitization are windup and long-term potentiation. Hyperexcitable spinal neurons show reduced thresholds, greater evoked responses, increased receptive field sizes, and ongoing stimulus-independent activity; these changes probably underlie the allodynia, hyperalgesia, and spontaneous pain seen in patients. Central sensitization is maintained by continuing input from the periphery, but also modulated by descending controls, both inhibitory and facilitatory, from the midbrain and brainstem. The projections of sensitized spinal neurons to the brain, in turn, alter the processing of painful messages by higher centers. Several mechanisms contribute to central sensitization. Repetitive activation of primary afferent C fibers leads to a synaptic strengthening of nociceptive transmission. It may also induce facilitation of non-nociceptive Aβ fibers and nociceptive Aδ fibers, giving rise to dynamic mechanical allodynia and mechanical hyperalgesia. In postherpetic neuralgia and complex regional pain syndrome, for example, these symptoms are maintained and modulated by peripheral nociceptive input. Diagnosing central sensitization can be particularly difficult. In addition to the medical history, quantitative sensory testing and functional magnetic resonance imaging may be useful, but diagnostic criteria that include both subjective and objective measures of central augmentation are needed. Mounting evidence indicates that treatment strategies that desensitize the peripheral and central nervous systems are required. These should generally involve a multimodal approach, so that therapies may target the peripheral drivers of central sensitization and/or the central consequences.

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