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Pain Perception

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Pain serves a protective role that alerts an individual to injury from the environment or from within. Based on current knowledge, all vertebrates, and some invertebrates, experience pain in response to actual or potential tissue damage. Many types of pain are encountered, with the most common being acute, chronic, cancer, and neuropathic. Acute pain is sudden in onset and can be severe. However, it disappears when the stimulus is removed or in a short period of time, and it tends to be self-limiting. Acute pain has a biologic function, because it serves as a warning that something is wrong while leading to protective behavioral changes. Acute pain is a symptom of disease, whereas chronic pain in and of itself, is a disease of altered neuroprocessing. Chronic pain lasts for several weeks to months and persists beyond the expected healing time. Chronic pain does not serve a biologic function and imposes severe detrimental stresses. Cancer pain is the result of primary tumor growth, metastatic disease, or the toxic effects of chemotherapy and radiation. Cancer pain can be acute, chronic, or intermittent and is related to the disease itself or the treatment. Neuropathic pain originates from injury or after injury of the peripheral or central nervous systems, such as trauma (eg, amputation and crushing injury), vascular injury (eg, thromboembolic disease), endocrinopathy (eg, diabetes mellitus), or infection (eg, post-herpetic neuralgia), possibly associated with motor, sensory, or autonomic deficits.

For an animal to experience pain, nociceptive information must be sent to higher centers in the CNS to be integrated, modulated, and interpreted into the conscious perception of pain. Noxious stimuli (heat, cold, mechanical, chemical) activate free sensory nerve endings known as nociceptors. A-δ and C-fibers transmit sensory information from nociceptors to the dorsal horn of the spinal cord, which directs and modulates input from the periphery and higher centers. Nociceptive information arriving in the dorsal horn of the spinal cord may activate motor neurons responsible for the reflex responses to noxious stimuli (such as withdrawing a limb). Importantly, nociceptive sensory input may be amplified or inhibited by spinal interneurons and glial cells.

Sensory information is relayed to higher centers in the CNS along a variety of pathways that differ according to species. In general, nociceptive information ascends the spinal cord along superficial and deep pathways to the brain stem with connections to the thalamus, reticular formation (responsible for level of arousal), and limbic system (responsible for emotions). From these areas of the brain, nociceptive information is relayed to the cortex, where it is perceived as pain. Activity in spinal nociceptive pathways is strongly influenced by descending antinociceptive systems that originate in the brain stem. Endogenous antinociceptive neurotransmitters (eg, endorphin, enkephalin, dynorphin, serotonin, and norepinephrine) inhibit the transmission of nociceptive information in the spinal cord and brain.

The neuroanatomic components of the nociceptive/pain pathways and pain-suppressing systems can change in response to sustained sensory input. Peripheral sensitization of nociceptors and central sensitization of nociceptive pathways in the dorsal horn, spinal cord, and brain can develop as a result of extensive tissue trauma or nerve injury. The process of peripheral and central sensitization has been termed “wind-up” and refers to the neuroanatomic changes (plasticity) that result in heightened or exaggerated pain states. Additionally, these exaggerated pain states often do not respond to conventional analgesic therapy. The use of opioids is especially limited, likely because of a down regulation of opioid receptors, a phenomenon that has been reported in the dorsal root ganglion and the dorsal horn. Thus, changes in the CNS in response to repeated and sustained nociceptive input (ie, pain) complicate the clinical management of pain.