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Perspective

Short Note on Neuropathic Pain

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DESCRIPTION

Neuropathic pain is a prevalent clinical condition that has a negative impact on patients' quality of life. Neuropathic pain is caused by a somatosensory system injury, according to a growing body of evidence from animal and human investigations. Injured peripheral nerve fibres cause a strong and long-lasting ectopic input to the CNS, as well as subsequent changes in dorsal horn neuronal excitability in some situations. There is now compelling evidence that defining neuropathic pain using a mechanism-based approach rather than an etiology-based approach may aid in tailoring therapy to the particular patient and may be effective in drug testing. This article outlines our current knowledge of the peripheral and central pathophysiological mechanisms that underpin neuropathic pain, with a focus on how symptoms are translated into mechanisms.

'Pain emerging as a direct result of a lesion or disease affecting the somatosensory system,' according to a commonly accepted definition of neuropathic pain.

Neuropathic pain is a common symptom of many peripheral and central nervous system illnesses. Distal symmetric peripheral neuropathies (e.g., diabetic neuropathy) and focal neuropathies associated to trauma (e.g., traumatic brachial plexus injuries), as well as surgical procedures, are the most common causes of neuropathic pain (e.g., breast surgery). Multiple sclerosis, spinal cord damage, and stroke are examples of CNS illnesses that cause neuropathic pain. The large range of etiologies suggests that neuropathic pain is common in the general population. In large population samples, postal surveys aimed to explore chronic pain with neuropathic characteristics found a 7–8% prevalence of neuropathic pain in the general population.

Neuropathic pain is caused by a variety of pathophysiological processes. Convincing evidence on the relationship between underlying pathophysiological mechanisms and neuropathic pain symptoms now suggests that classifying neuropathic pain according to a mechanism-based approach rather than an etiology-based approach may help in targeting therapy to the individual patient and in testing new drugs. We outline our present knowledge of the peripheral and central pathophysiological mechanisms underlying neuropathic pain in this article, with a focus on how symptoms are translated into mechanisms.

Neuropathic pain animal models

Animal models of peripheral nerve injuries, which are mostly designed to mimic human disorders, provide the majority of our current understanding on the complicated pathophysiological processes that cause neuropathic pain. Although these models have the essential benefit of advancing our understanding of the mechanisms underlying neuropathic pain, they frequently underestimate the role of certain targets or processes in human neuropathic pain. To imitate the clinical settings of amputation, some investigations have used complete nerve transection and ligation. To imitate the clinical situation of partial peripheral nerve injury, partial nerve ligation and sparing nerve injury were used. The closure of the spinal nerves successfully simulates spinal root injury caused by a lumbar disc herniation. Demyelination caused by an immune response or a toxin mimics demyelinating neuropathy.

In animal models, vincristine, paclitaxel, and cisplatin were utilised to imitate polyneuropathy caused by tumour chemotherapy. Finally, damage to pancreatic insulin-producing cells caused by streptozocin in rats provides an experimental model of diabetic neuropathy.

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