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Pathophysiology and Mechanisms of Neuropathic Pain

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Introduction

The importance of the sympathetic nervous system in neuropathic pain has been demonstrated by analgesia following sympathectomise in animals and humans, and by pain exacerbation through activation of the sympathetic nervous system [1]. Sympathetically maintained pain may be explained by sprouting of sympathetic neurons into dorsal root ganglia of injured sensory neurons and post-injury sprouting of sympathetic fibres into the dermis. The blockade of nerve conduction in neuropathic conditions causes nerve dysfunction, which can result in numbness, weakness and loss of deep tendon reflexes in the affected nerve area. Neuropathic conditions also cause aberrant symptoms of spontaneous and stimulus-evoked pain. Spontaneous pain is commonly described as burning, shooting or shock-like. Stimulus evoked pain includes allodynia and hypoalgesia. Allodynia can be caused by the lightest stimulation, such as skin contact with clothing or a light breeze. These sensory abnormalities may extend beyond nerve distributions, which may lead to the inappropriate diagnosis of a functional or psychosomatic disorder [2]. The diagnosis of neuropathic pain is based primarily on history and findings on physical examination. Assessment of the patient with suspected neuropathic pain should focus on ruling out treatable conditions, confirming the diagnosis of neuropathic pain and identifying clinical features that might help individualize treatment. Box 1 lists principal details of the clinical evaluation, including history, physical examination and special tests. Neuropathic pain, caused by a lesion of the nervous system, is especially problematic because it is often experienced in parts of the body that other-wise appear normal, it is generally chronic, severe and resistant to over-the-counter analgesics, and it is further aggravated by allodynia. It may result from various causes that affect the brain, spinal cord and peripheral nerves, including cervical or lumbar radiculopathy, diabetic neuropathy, cancer-related neuropathic pain, post-therapeutic neuralgia, HIV-related neuropathy, spinal cord injury, trigeminal neuralgia and complex regional pain syndrome type II, among others [3]. Complex regional pain syndrome type I is not considered a cause, because there is no definable nerve lesion. The epidemiology of neuropathic pain has not been adequately studied, partly because of the diversity of the associated conditions. Current pooled estimates suggest that neuropathic pain may affect as much as 3% of the population. The personal impact of neuropathic pain is most vividly appreciated by people who experience this devastating condition [4]. Those affected have described their pain using the McGill Pain Questionnaire with descriptors such as punishing cruel and tiring–exhausting. Ample evidence indicates that neuropathic pain impairs patients' mood, quality of life, activities of daily living and performance at work. People with the condition have been found to generate 3-fold higher health care costs compared with matched controls [5]. In the United States, health care, disability and related costs associated with chronic pain have been estimated at $150 billion annually, of which almost $40 billion is attributable to neuropathic pain. Regeneration after nerve injury results in the formation of neuromas and sprouting of new nerve projections among uninjured neighbouring neurons. Collateral sprouting then leads to altered sensory properties that may be realized as expanded receptive fields. Uncontrolled neuronal firing after experimental nerve injury is largely attributed to increased expression of sodium channels. This mechanism is supported by several lines of evidence, including blockade of neuropathic pain with sodium-channel–blocking local anaesthetics. Demyelination of diseased nerves may be another cause of increased neuronal excitability [6]. In addition to sodium channels, expression of voltage-gated calcium channels is also increased following nerve injury. Calcium entry through voltage-gated calcium channels is necessary for the release of substance P58 as well as glutamate from injured peripheral nerves [7]. Within the dorsal root ganglion, increased expression of the α-2-delta subunit of voltage-gated calcium channels correlates with onset and duration of allodynia [8]. Clinical support of the role of this protein in neuropathic pain arises from the analgesic efficacy of α-2-delta voltage-gated calcium-channel antagonists, gabapentin and pregabalin [9]. Activation of descending pathways has been shown to reduce pain transmission in animals and humans and is thought to contribute to the analgesic effect of opioids and antidepressants. Paradoxically, this system can also facilitate pain transmission and may contribute to some chronic pain states [10].

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Conflict of Interest

None

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