



## An Overview of Neuropathic Pain

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### Introduction

Neuropathic pain is caused by a lesion or disease of the somesthesia, including peripheral fibres (A $\beta$ , A $\delta$  and C fibres) and central neurons, and affects 7–10% of the overall population. Multiple causes of neuropathic pain are described and its incidence is probably going to extend due to the ageing global population, increased incidence of DM and improved survival from cancer after chemotherapy. Indeed, imbalances between excitatory and inhibitory somatosensory signalling, alterations in ion channels and variability within the way that pain messages are modulated within the central system all are implicated in neuropathic pain. The burden of chronic neuropathic pain seems to be associated with the complexity of neuropathic symptoms, poor outcomes and difficult treatment decisions. Importantly, quality of life is impaired in patients with neuropathic pain due to increased drug prescriptions and visits to health care providers, also because the morbidity from the pain itself and therefore the inciting disease. Despite challenges, progress within the understanding of the pathophysiology of neuropathic pain is spurring the event of latest diagnostic procedures and personalized interventions, which emphasize the necessity for a multidisciplinary approach to the management of neuropathic pain.

Although distinct definitions of neuropathic pain are used over the years, its most up-to-date (2011) and widely accepted definition is pain caused by a lesion or disease of the somesthesia. The somesthesia allows for the perception of touch, pressure, pain, temperature, position, movement and vibration. The somatosensory nerves arise within the skin, muscles, joints and fascia and include thermoreceptors, mechanoreceptors, chemoreceptors, pruriceptors and nociceptors that send signals to the medulla spinalis and eventually to the brain for further processing (BOX 1); most sensory processes involve a thalamic nucleus receiving a sensory signal that's then directed to the cerebral mantle. Lesions or diseases of the somatosensory system can cause altered and disordered transmission of sensory signals into the medulla spinalis and therefore the brain; common conditions related to neuropathic pain include postherpetic neuralgia, tic douloureux, painful radiculopathy, diabetic neuropathy, HIV infection, leprosy, amputation, peripheral nerve injury pain and stroke (in the shape of central post-stroke pain). Not all patients with peripheral neuropathy or central nervous injury develop neuropathic pain; for instance, an outsized cohort study of patients with DM indicated that the general prevalence of neuropathic pain symptoms was 21% in patients with clinical neuropathy. However, the prevalence of neuropathic pain increased to 60% in those with severe clinical neuropathy. Importantly, neuropathic pain is mechanistically dissimilar to other chronic pain conditions like inflammatory pain that happens, for instance, in atrophic arthritis, during which the first

cause is inflammation with altered chemical events at the location of inflammation; such pain is diagnosed and treated differently.

Neuropathic pain is related to increased drug prescriptions and visits to health care providers. Patients typically experience a definite set of symptoms, like burning and electrical-like sensations, and pain resulting from non-painful stimulations (such as light touching); the symptoms persist and have a bent to become chronic and respond less to pain medications. Sleep disturbances, anxiety and depression are frequent and severe in patients with neuropathic pain, and quality of life is more impaired in patients with chronic neuropathic pain than in those with chronic non-neuropathic pain that doesn't come from damaged or irritated nerves.

Despite the increases of placebo responses that end in the failure of multiple new drugs in clinical trials, recent progress in our understanding of the pathophysiology of neuropathic pain provides optimism for the event of latest diagnostic procedures and personalized interventions. This Primer presents the present descriptions of the presentation, causes, diagnosis and treatment of neuropathic pain, with attention on peripheral neuropathic pain as long as our knowledge is bigger than that of central neuropathic pain.

### Epidemiology

The estimation of the incidence and prevalence of neuropathic pain has been difficult due to the shortage of straightforward diagnostic criteria for giant epidemiological surveys within the general population. Thus, the prevalence of neuropathic pain within the chronic pain population has mainly been estimated on the idea of studies conducted by specialized centres with attention on specific conditions, like postherpetic neuralgia, painful diabetic polyneuropathy, postsurgery neuropathic pain, MS, medulla spinalis injury, stroke and cancer.

### Conclusion

Characteristics has been estimated to be within the range of 7–10%.

Chronic neuropathic pain is more frequent in women (8% versus 5.7% in men) and in patients >50 years aged (8.9% versus 5.6% in those 12,000 patients with chronic pain with both nociceptive and neuropathic pain types, mentioned pain specialists in Germany, revealed that 40% of all patients experience a minimum of some characteristics of neuropathic pain (such as burning sensations, numbness and tingling); patients with chronic back pain and radiculopathy were particularly affected.

Genetic stratification is tougher in common acquired neuropathic pain states, like painful diabetic neuropathy, because such conditions are polygenic and subject to considerable environmental interaction. Thus, the relevance of a private target like Nav1.7 in these conditions is a smaller amount clear. Despite these limitations, the prospect of personalized medicine may be a breakthrough towards promising pain management strategies.

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