Neuropathic pain: mechanisms and their clinical implications

BMJ 2014; 348 doi: http://dx.doi.org/10.1136/bmj.f7656 (Published 05 February 2014) Cite this as: BMJ 2014;348:f7656

Abstract

Neuropathic pain can develop after nerve injury, when deleterious changes occur in injured neurons and along nociceptive and descending modulatory pathways in the central nervous system. The myriad neurotransmitters and other substances involved in the development and maintenance of neuropathic pain also play a part in other neurobiological disorders. This might partly explain the high comorbidity rates for chronic pain, sleep disorders, and psychological conditions such as depression, and why drugs that are effective for one condition may benefit others. Neuropathic pain can be distinguished from non-neuropathic pain by two factors. Firstly, in neuropathic pain there is no transduction (conversion of a nociceptive stimulus into an electrical impulse). Secondly, the prognosis is worse: injury to major nerves is more likely than injury to non-nervous tissue to result in chronic pain. In addition, neuropathic pain tends to be more refractory than non-neuropathic pain to conventional analgesics, such as non-steroidal anti-inflammatory drugs and opioids. However, because of the considerable overlap between neuropathic and nociceptive pain in terms of mechanisms and treatment modalities, it might be more constructive to view these entities as different points on the same continuum. This review focuses on the mechanisms of neuropathic pain, with special emphasis on clinical implications.