

29. Pain After the Injury: When Neural Signaling Becomes Self-Sustaining

Absurd Health Institute | Medical Concept Papers, Volume Four | VI. From mechanism to disease

Publication type: Revised medical concept and hypothesis essay — not peer reviewed **Corrective edition:** 17 October 2026 **Substantive text:** 1,203 words

Abstract

Central and peripheral sensitization can amplify nociceptive signaling and persist after an initial injury improves. This paper distinguishes altered pain processing from claims that pain is imaginary and explores the importance of nervous-system plasticity alongside ongoing tissue pathology.

Central and peripheral sensitization can amplify nociceptive signaling and persist after an initial injury improves. This paper distinguishes altered pain processing from claims that pain is imaginary and explores the importance of nervous-system plasticity alongside ongoing tissue pathology.

Pain and injury are not identical variables

Nociceptive activity commonly signals tissue threat, but pain is a nervous-system experience shaped by peripheral input and central processing. After intense or repeated nociception, neurons in pain pathways can become more responsive. Central sensitization can produce amplified responses, pain from normally non-painful stimuli and spread of sensitivity. These responses are physiological, not evidence that the patient invented or chose the pain.

How a protective alarm remains activated

Early pain may protect an injured site from further damage. Persistent amplification can outlast much of the initial peripheral lesion. The nervous system can change synaptic efficacy, inhibitory balance and the significance assigned to sensory input. Peripheral inflammatory and immune signals may contribute in some conditions. Distinct syndromes involve different combinations of peripheral generators and central amplification, so a single 'brain pain' label is inadequate.

The causal ambiguity

A patient with persistent pain may have active tissue pathology that deserves treatment, altered neural gain, or both. Simply telling patients their pain is sensitization can miss fractures, tumors, neuropathy, infection or inflammatory disease. Conversely, repeated structural imaging may fail to explain amplified symptoms, and unnecessary procedures can bring their own harms. Mechanistic humility is essential.

Testing the proposition

Sensory testing, characteristic clinical findings and carefully interpreted research measures can support a sensitization hypothesis. No single common symptom conclusively establishes central sensitization in an individual. The critical prediction is that pain intensity and functional limitation can sometimes remain disproportionate to an identifiable current tissue lesion because processing has changed. Improved central or peripheral modulation should be judged by meaningful patient outcomes.

Treatment is not a proof of etiology

A patient's improvement with exercise adaptation, psychological therapy, medications or nervous-system-focused care does not establish that the original illness was psychological. Several approaches can affect common final pathways regardless of the initial cause. Similarly, failure of one intervention does not prove irreversibility.

A broader lesson

Pain is a particularly clear example of biological history altering present function. Neural pathways can retain a changed gain setting after the initiating injury has diminished, but that insight should expand compassionate clinical assessment rather than become an excuse to dismiss ongoing pathology.

Sensitization is measurable, but not a universal explanation

Allodynia, hyperalgesia and altered temporal summation can arise from changes in nociceptive processing. Such observations provide real physiological content to the sensitization concept. But many chronic pain disorders also have ongoing peripheral generators, and in some patients those remain dominant. A responsible account does not substitute neural amplification for a needed investigation of structural, inflammatory or malignant causes.

Memory without psychological blame

The nervous system changes with experience through plasticity. Persistent changes in gain or inhibitory control can outlast the original noxious event. Calling this a form of biological memory does not mean the patient chose to remember pain or failed to forgive an injury. Emotional context and attention can modulate pain while peripheral pathology remains genuine. The model should therefore improve compassion, not encourage diagnostic dismissal.

The clinical target is function as well as intensity

Pain treatment can aim to reduce symptom burden, improve mobility, restore sleep and increase participation in ordinary life. These outcomes may change at different rates. A patient who regains function while still experiencing some pain has made real progress; a patient whose pain score decreases while medication harms cognition has experienced a tradeoff. Such multidimensional evaluation fits the broader recovery framework of this volume.

Pain is a protective signal that can become an illness

Acute nociceptive signaling helps organisms respond to potential tissue damage. After an injury, peripheral nociceptors can become sensitized by inflammatory mediators, while central neural circuits may alter how sensory input is amplified and interpreted. In neuropathic pain, damage or disease affecting the somatosensory nervous system can generate abnormal sensory activity. Some persistent pain states also involve altered sleep, autonomic regulation, movement patterns and psychological responses. These interactions are biologically real, but the existence of central sensitization does not prove that pain is imagined or unrelated to tissue pathology.

Timing matters because an initiating injury and a maintaining mechanism can diverge. A peripheral lesion may heal structurally while abnormal nerve firing persists. Conversely, continued mechanical compression or inflammatory disease may maintain pain through an active peripheral source. A clinical assessment should not choose a nervous-system explanation simply because routine imaging is normal, nor assume ongoing structural injury merely because pain is severe.

A model must distinguish specific phenotypes

Burning pain with allodynia after a defined nerve injury differs from inflammatory arthritis and from widespread pain without demonstrable peripheral nerve damage. Altered descending pain modulation might contribute to more than one condition, but the contributions and available treatments vary. Quantitative sensory testing, neurological examination, relevant imaging and disease-specific investigations provide different pieces of evidence. No one test confirms every proposed form of sensory amplification.

The model also needs to avoid a false opposition between biological and psychological factors. Threat expectations, attention, mood and previous experiences can influence neural pain processing without implying that the underlying suffering is voluntary. Those influences are part of nervous-system physiology. A compassionate explanation remains compatible with investigating structural disease, medication effects and functional limitations rigorously.

What would establish a self-sustaining loop

A longitudinal study might begin soon after a defined injury, assess peripheral nerve function and sensory processing repeatedly, and examine which findings predict chronic pain after the tissue heals. The central hypothesis is that some neural changes become predictors of later disability independently of ongoing peripheral damage. Demonstrating that a targeted intervention changes those neural processes and improves functional outcomes would be stronger evidence than a cross-sectional association.

The practical goal is meaningful relief and restored participation in daily life, not simply normalized imaging or a single pain score. Drug therapy, rehabilitation and psychological approaches can have evidence-based roles depending on diagnosis; no one strategy should be elevated to a universal cure. The proposed terrain framing is that local tissue injury and the nervous system can reshape one another over time, sometimes creating a problem that outlasts the original insult.

The clinical meaning of persistence

It would be a mistake to conclude that ongoing pain after healing proves the nervous system has learned pain in the absence of injury. A recurrent lesion, entrapment, inflammatory disease or other structural problem may remain active but hard to identify. Equally, normal imaging does not prove the pain is nonbiological. The diagnostic task is to determine the probable contribution of ongoing nociception, nerve pathology and altered central processing without collapsing all unexplained symptoms into one label.

This has practical consequences for research endpoints. A reduction in pain intensity is important, but participants may value sleep, return to activity, reduced medication burden and the ability to tolerate ordinary sensory experiences just as much. A treatment changing one neural excitability marker should not be called restorative unless it improves relevant function and safety. The most informative causal studies will also examine whether modification of a hypothesized neural feedback pathway precedes improvement, rather than merely correlating with it after successful rehabilitation.

Selected scientific reading

1. Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. *Pain*. 2011;152(Suppl):S2–S15.
2. Netea MG et al. Defining trained immunity and its role in health and disease. *Nat Rev Immunol*. 2020;20:375–388.

Editorial and evidentiary note

This is an expanded conceptual synthesis, not original clinical research or a clinical treatment protocol. It preserves selected references from the original edition, whose bibliographic metadata have not all been independently reverified. Claims about specific diagnoses and treatments require appropriate primary evidence and professional assessment. Mechanistic plausibility does not establish therapeutic efficacy.