BrJP. 2026, v.9:e202683 • https://doi.org/10.63231/2595-0118.e202683-en SBED

BrJP
Brazilian Journal of Pain

EDITORIAL

We understand phantom pain, so why do we still struggle to treat it? The paradox of phantom pain

Entendemos a dor fantasma, então por que ainda temos dificuldade em tratá-la? O paradoxo da dor fantasma

Célia Maria de Oliveira[1], Juliana Barcellos de Souza[2]

[1.] Universidade Federal de Minas Gerais (UFMG), Belo Horizonte, MG, Brasil.
[2.] Universidade do Estado de Santa Catarina (UDESC), Florianópolis, SC, Brasil.

Correspondence to:
Célia Maria de Oliveira
cmariol@terra.com.br

Phantom limb pain can be investigated through reports of medical observation, in which pain assessment is associated with the difficulty of identifying and understanding pain in an absent segment, such as pain in the right hand in patients with a right upper limb amputation. To understand this phenomenon, researchers and clinicians have proposed theoretical models.

In 1999, Melzack expanded the Gate Control Theory (published by him and Patrick Wall in 1965[1], updating the model to account for the presence of pain even in the absence of the limb segment[2]. Understanding pain through the Cortical Neuromatrix Theory[3] distinguishes nociception from pain and considers that each individual possesses a neurosignature. Interactions within the cortical matrix vary according to biological and genetic characteristics, experiences and life events, as well as the way these are interpreted and stored in the cortical matrix, in addition to individual expectations and planning. This model personalizes pain perception and incorporates individuality into the interpretation of symptoms, sensations, memories, emotions, and expectations. In the specific case of phantom limb pain (PLP), recognized for more than a century as a unique phenomenon, there is an integration of body memory, neural plasticity, and central and peripheral neuropathic mechanisms.

Pain assessment remains a challenge in both clinical practice and research. Despite efforts to measure it objectively, pain is a subjective, dynamic, and multifactorial experience. Validated instruments assist in quantifying and monitoring pain, promoting greater uniformity in clinical follow-up and contributing to clinical decision-making and to the understanding of mixed or complex pain[4].

In recent decades, advances in neuroscience have demonstrated that the brain maintains somatosensory maps organized and resistant to peripheral loss, supporting the hypothesis that painful perception in the absent limb results from the persistence of these representations[5]. Neuromatrix theory[3] reinforced this understanding by showing that pain is produced by an integrated neural network and does not depend exclusively on peripheral input. This concept explains the occurrence of intense pain even in the total absence of the limb and supports the development of interventions such as mirror therapy[6] and imagery techniques[7], which aim to stimulate activity in the somatosensory cortex and associated neural networks. Despite advances in central models, the relative contribution of peripheral and central mechanisms in PLP remains under debate, reflecting the unresolved complexity of this condition.

Although studies have refined theoretical and conceptual models, clinical practice still faces significant challenges, such as treating pain in an absent limb. Pharmacological interventions remain frequent and generally constitute the first line of treatment; however, their effects on PLP are limited, reinforcing the need for combined approaches. Strategies based on neuroplasticity, sensorimotor rehabilitation, and modulation of central circuits, such as visual therapies, noninvasive stimulation, and cortical reorganization, have gained increasing support in the literature[8-10]. Widely disseminated interventions, such as mirror therapy, present heterogeneous results, highlighting the gap between neurobiological plausibility and consistent clinical effectiveness. These approaches analyze characteristics of pain, including location, somatosensory descriptors, affective and cognitive factors, and the relationship with previous body maps, demonstrating the persistence of active neural circuits even after amputation[6,11].

In clinical practice, this complexity translates into concrete challenges. Patients with PLP frequently receive priority treatment through pharmacological or peripheral approaches, even when pain maintenance is strongly associated with central and representational mechanisms. The absence of systematic evaluation of aspects such as body perception, somatosensory organization, and functional impact may limit the effectiveness of interventions. Incorporating these dimensions into clinical reasoning is crucial for the quality of care.

Despite the relevance of central mechanisms, PLP cannot be understood exclusively as a cortical phenomenon. Peripheral changes, such as neuromas, ectopic activity, and alterations in spinal excitability, also contribute to the painful experience[12]. The interaction between these factors makes PLP one of the most complex neuropathic conditions to manage, requiring an integrated clinical approach.

In addition to neurobiological mechanisms, PLP imposes significant psychosocial challenges. Persistent pain hinders the use of prostheses, compromises functional rehabilitation, and profoundly impacts emotional well-being. Patients frequently report bodily strangeness, anxiety, and difficulty adapting to their new condition[7]. The therapeutic management of PLP goes beyond the physical dimension, requiring interdisciplinary and multidisciplinary approaches that consider the multiple dimensions of pain experience and patient functionality.

Although contemporary models recognize the complexity of PLP, their incorporation into clinical practice remains limited, revealing a persistent gap between knowledge and care.

In the current context, in which chronic pain constitutes one of the major global public health challenges, PLP clearly demonstrates the limitations of fragmented models of understanding. More than expanding theories, the challenge lies in articulating them meaningfully in clinical practice. This requires professionals not only to possess technical knowledge, but also sensitivity to interpret patterns, recognize the uniqueness of each patient's experience, and adapt therapeutic strategies to complex and often invisible realities.

In recent decades, advances in understanding PLP have been remarkable, but the consistent transfer of knowledge into care and rehabilitation remains modest. On the one hand, neuroscience has provided increasingly sophisticated models to explain pain; on the other hand, clinical practice still lacks equally robust strategies to effectively treat PLP. Heterogeneous therapeutic outcomes and the difficulty of integrating central, peripheral, and psychosocial mechanisms reflect not only the complexity of PLP, but also the limitations of still-fragmented multiprofessional approaches.

Overcoming this scenario will require more than new research findings; it will require a paradigm shift in which knowledge is not only produced but systematically integrated into care. In PLP, the contemporary challenge is not merely to understand why pain persists, but also to develop clinical responses that support the follow-up and care of patients living with this complex pain condition.

REFERENCES

1. Melzack R, Wall PD. Pain mechanisms: a new theory. Science. 1965;150(3699):971-9. https://doi.org/10.1126/science.150.3699.971. PMid:5320816.
2. Melzack R. Phantom limbs and the concept of neuromatrix. Trends Neurosci. 1990;13(3):88-92. https://doi.org/10.1016/0166-2236(90)90179-E. PMid:1691874.
3. Melzack R. From the gate to the neuromatrix. Pain. 1999;82(Suppl 1):S121-6. https://doi.org/10.1016/S0304-3959(99)00145-1. PMid:10491980.
4. Flor H. Phantom limb pain: characteristics, causes, and treatment. Lancet Neurol. 2002;1(3):182-9. https://doi.org/10.1016/S1474-4422(02)00074-1. PMid:12849487.
5. Ramachandran VS, Hirstein W. The perception of phantom limbs. Brain. 1998;121(9):1603-30. https://doi.org/10.1093/brain/121.9.1603. PMid:9762952.
6. Subedi B, Grossberg GT. Phantom limb pain: mechanisms and treatment approaches. Pain Res Treat. 2011;2011:864605. https://doi.org/10.1155/2011/864605. PMid:22110933.
7. Piazza AA, Luz SCT, Souza JB, Vieira AC, Medeiros AB. Telerehabilitation for the treatment of phantom limb pain: a quasi-experimental study. BrJP. 2025;8:e20250032. https://doi.org/10.63231/2595-0118.20250032-en.
8. Kuner R, Flor H. Structural plasticity and reorganization in chronic pain. Nat Rev Neurosci. 2016;18(1):20-30. https://doi.org/10.1038/nrn.2016.162. PMid:27974843.
9. Flor H, Nikolajsen L, Jensen TS. Phantom limb pain: a case of maladaptive CNS plasticity? Nat Rev Neurosci. 2006;7(11):873-81. https://doi.org/10.1038/nrn1991. PMid:17053811.
10. Collins KL, Russell HG, Schumacher PJ, Robinson-Freeman KE, O'Conor EC, Gibney KD, Yambem O, Dykes RW, Waters RS, Tsao JW. A review of current theories and treatments for phantom limb pain. J Clin Invest. 2018;128(6):2168-76. https://doi.org/10.1172/JCI94003. PMid:29856366.
11. Nikolajsen L, Jensen TS. Phantom limb pain. Br J Anaesth. 2001;87(1):107-16. https://doi.org/10.1093/bja/87.1.107. PMid:11460799.
12. Jensen TS, Finnerup NB. Neuropathic pain: mechanisms and treatment. Eur J Neurol. 2014;21(2):217-27.