

PSIWORLD 2014

Etiopathogenetic explanations and treatment options in phantom limb syndrome. A literature review

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Abstract

Phantom limb syndrome is a controversial subject when talking about its etiopathogenetic mechanisms and efficient treatment modalities. The article reviews different etiology mechanisms, both neurological and psychological, as there is no consensus among specialists in these fields, as well as different ways of dealing with reducing pain in people that suffer from phantom limb syndrome. It also highlights the gaps on the subject and the need for future research on certain aspects of the phenomenon.

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Peer-review under responsibility of the Scientific Committee of PSIWORLD 2014.

Keywords: phantom limb; etiology explanations; neuromatrix theory; treatment; mirror box; hypnosis

1. Introduction

Phantom limb syndrome is defined as the persistence of the perception of a limb that has been amputated and has been mentioned starting with Antiquity. The one who firstly mentioned it in medical literature and offered it a scientific character was the neurologist Silas Weir Mitchell in 1871. Since then, scientists in neurology and psychology have been stating different etiologies and treatment modalities, none of them being agreed upon as most efficient. The present study reviews theoretical articles and studies on etiology and treatment of this syndrome.

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2. Neurological theories regarding phantom limb syndrome etiology

The first explanatory theory is the peripheral theory, which underlines the importance of neuromas. According to this theory they send impulses through the spinal cord to the brain (cortex), impulses that may be felt as painful. Thus the neurons that were sectioned and that are located in the stump create, by unusual firing, sensitive messages in the somatosensory cortex. Even though they are not well organized and clearly defined, they entertain and maintain the perception of the missing part of the body, as well as the pain located in that corporal segment (Sherman & Sherman, 1989; Devor & Faulkner, 1999). This first theory of etiopathogenesis stands for some therapies based on the administration of analgesics, as well as for the use of massage and the electrical stimulation of the stump (Sherman & Sherman, 1989). Because “the phantom” did not disappear specialists have also considered surgical interventions, but they did not provide a significant amelioration. The peripheral theory has been contested frequently, for some arguments like the fact that the phantom limb syndrome appears in many cases immediately after amputation, while the neuroma formation is a process that requires some time post amputation, it is not an immediate process (Wall & Fitzgerald, 1981). Another argument against the peripheral theory would be the fact that the PLP phenomenon is also present in the case of spinal traumatism, and in this case the peripheral nervous system is not at all affected (Hotz-Boendermaker et al, 2008; Curt, Yengue, Hilti, Brugger, 2010).

In order to demonstrate the peripheral origin of phantom limb syndrome, it has been stated that it does not appear in the case of children who are congenitally missing a limb. But this hypothesis has been proved to be false, because some patients with congenital absence of a limb reported to have lived the sensation of the presence of the missing limb (Melzack, 1996). A study conducted by Wilkins, McGrath, Finley and Katz (1998) has reported the presence of this sensation in 42% of those who presented a congenital absence of a limb, 29% of them suffering also from phantom limb pain.

In contrast to peripheral theories, central theories claim that phantom limb syndrome is the result of the firing of central nervous system neurons. The loss of afferent input determines the firing of neurons situated in the central nervous system, and it provokes permanent changes in the synaptic structure. This results in an increase of nerves irritability and a reduction of the inhibitory process, this phenomenon being difficult to interrupt. Cortical reorganization is one of the most cited explanations in the phantom limb syndrome etiology. Literature suggests that the cortical area corresponding to the amputated part of the body is “taken in responsibility” by the representative zones next to that area, from the somatosensory cortex, but also from the motor cortex (Melzack, 1992; Melzack, 2005; Moseley, 2006).

Ronald Melzack (1992) introduced the term “the neuromatrix theory” to explain the sensations felt in the amputated limb, as phantom limb sensation and phantom limb pain. The neuromatrix theory follows naturally after peripheral and central theories, integrating elements from both of them. This theory sustains the idea of a neuronal matrix that integrates different types of inputs generated from different body parts, including somatosensory, limbic, visual, thalamic-cortical elements. The neuromatrix would contain three important pain dimensions: sensorial, cognitive and affective. The internal body conscience is created at the cerebral level, being activated by perceptual impulses. The term “neurosignature” was proposed by Melzack to refer to the activity pattern generated by the brain, which is actualized by the consciousness and by the body and self -perception. Melzack states that phantom pain is generated by deprivation of many inputs from limbs to the neuromatrix, causing the formation of abnormal signature. The central theory of phantom limb syndrome origin is sustained by an experiment made by Hindenmayer. He realized a study where participants that suffered from phantom limb sensation had to perform the same tasks with the intact limb, as well as with the “missing” limb. He examined EEG results and they showed “the same modifications for both body parts” (Hindenmayer, 1962).

3. Psychological theories regarding phantom limb syndrome origin

Parallel to neurological theories, phantom limb syndrome has been considered to be a phenomenon of psychological origin. Difficult psychological adaptation to a new medical situation and body change, the change in the quality of life, has been considered to stand for the origin of phantom sensations and their maintainence in time (Weil, 1991). This theory is based upon the presumption that the phantom limb constitutes the object of narcissistic

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