Phantom pain: a ghost in the machine, or a biological basis?  

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Introduction

Phantom pain is pain coming from a body part that is no longer there, it is a ‘persistent image or memory of that part of the body’ that has been lost. It is categorized as a type of chronic, neuropathic pain, suggesting that it is caused by damage to the somatosensory nervous system that may last for years to come. It is very prevalent - found most commonly in 60-80% of people whose limbs have been amputated, but phantom pain also arises when other organs, such as breasts and eyes, are removed. Despite the prevalence of this phenomenon and the fact that amputations are one of the oldest surgical procedures, phantom pain was shrouded in secrecy and not mentioned in literature for a long time, perhaps due to the fear of insanity. It was only first named by Silas Weir Mitchell after he worked with many amputees in a military hospital during the Civil War. But what is the actual basis of phantom pain? It may be a ghost in the machine: something unknown and obscure, such as our consciousness or imagination, operating within the known system of our body, or it may have physical biological causes. To what extent can phantom pain be explained by the physiological workings of our body (as opposed to previously conceived psychological causes)? This essay will explore this question through analyzing different models of pain, the biological mechanisms of phantom pain, psychological misconceptions and factors affecting phantom pain.

Nature of phantom pain

Phantom pain occurs in 80% of amputees, and appear within a few days of amputation. It has been described as burning, shooting, stabbing, or cramping pain. The pain experienced in the missing body part is often the similar to the pain felt before amputation, giving it some memory-like qualities. They are perceived while not actually present, making them hallucinations, but they differ from other hallucinations as they can be controlled and moved by the person to a certain extent. Other than amputees, 20% of congenitally limb-deficient people experience phantom pain, suggesting that this sensation is perhaps something innate. In the case of phantom limbs, they often become extensions of the body image, and are needed to merge with the prosthesis in order for the prosthesis to be used.

Social and psychological factors affecting phantom pain

In the past, phantom pain was believed to be the imagination of the amputee under the influence of certain psychological issues, such as grief, change in body image, and depression. It was generalized as psychosomatic pain - in a 1983 survey, 69% of 2700 veteran amputees reported that their physicians ‘had clearly implied that the pain was just in their heads’.

Given the post-war social context in which phantom pain was first widely discussed, the cause phantom pain was often linked back to the trauma under which the loss of limbs occurred. The doctor-patient relationship was also of importance: there was a bias in the referral of patients that doctors did not get along with to mental health professionals more frequently than patients with similar conditions

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who met the doctors’ expectations. The patients referred by doctors tend to be the patients who report their phantom pain and obstinately demand treatment, leading to the misrepresentation of the majority of phantom pain patients as those with mental health or psychological issues. In a study investigating the effect of different wars (WWII vs. Korean War), the preparation allowed for an amputation (sudden/traumatic vs. chronic), and the cause of the amputation (war vs. civilian related issues) on phantom pain, researchers found that there was little contrast between these groups. This suggests that phantom pain has a primarily biological basis, and that the psychosomatic component of phantom pain is minimal.

Psychoanalyst Franz Alexander provided a model involving four factors affecting susceptibility to psychosomatic phantom pain: personality type, emotional stress, the combination of psychological stress and conflict, and hereditary qualities. While such factors affect the intensity of the pain, they are supported by a variety of physiological causes, and are not the only causes of phantom pain. Stress and exhaustion are now proven to trigger physiological responses in the body, such as the release of adrenaline and cortisol, which shows that these psychological factors themselves have biological bases. Hence, while patients with abnormally high psychological risk factors tend to report more severe phantom pain, it is concluded that phantom pain is not more affected by psychological factors than other types of chronic pain.

Physiological models of pain

When faced with the overwhelming statistics of phantom pain presenting the prevalence of pain stemming from something that is no longer present, we are no longer able to dismiss phantom pain as rare cases of insanity, but we must ask: how do we feel pain, and what makes pain real? The Cartesian model of pain is a relatively straightforward, logical one. It is based on nociception, which is our body’s response to painful or potentially painful stimuli through the nervous system. When peripheral tissue damage occurs, signals are sent through the spinal cord to the brain, which receives the signal indicating pain. Hence, physical damage processed by the peripheral nervous system is what triggers the nociceptive pathways, and it is what makes pain real.

However, another model was proposed by psychologist Robert Melzack in 1990. The neuromatrix model suggests that different parts of the nervous system work together to form a matrix. In this model, the central nervous system (CNS) is not the recipient of signals of pain, but rather works in response with external stimuli to generate sensations of pain. As the CNS is responsible for many different things, such as emotion, cognition, and behaviours, this model takes into the account of other psychological factors up- and down-regulating the degree of which pain is felt. As it suggests that it is not only physical tissue damage that causes pain, it provides a possible explanation for phantom pain and other types of chronic pain (such as non-specific lower back pain) by changing the way we understand pain.

Mechanisms of phantom pain

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Both of the mentioned models of pain are considered in the understanding of phantom pain. The Cartesian model offers some explanation to phantom pain in peripheral nerve damage. Even before the amputation of limbs or surgical removal of organs, the patient is already likely to have suffered a certain degree of nerve damage due to a lack of blood flow to tissues. As surgery is a type of trauma in itself, further nerve damage is caused. This causes a non-specific immune response, where inflammation occurs and nociceptive pathways are activated with the release of cytokines, sending signals to the brain indicating painful or potentially damaging stimuli. This results in the peripheral nervous system becoming very sensitized, producing exaggerated responses to non-painful stimulus, and is the likely cause for acute phantom pain.

The overwhelming nociceptive stimulus also leads to changes in the pain pathways of CNS, namely in the spinal cord. This leads to dorsal horn cells becoming hyperexcitable and firing random signals. At the receptor level, the NMDA (N-methyl-D-aspartate) receptors also modify nociceptive signals, causing non-painful stimuli to be received and interpreted as painful, demonstrating the concept of Melzack’s neuromatrix model, with the CNS being a source of the pain. The threshold for pain is lowered due to the hypersensitivity of the spinal cord, leading to central sensitization, with chronic pain occurring as a result.

Amputation also results in the formation of a neuroma. This is created when a nerve is cut, and the axon of the damaged nerve regenerates and sprouts. These neuromas show spontaneous and abnormal responses to mechanical or chemical stimulation (due to the increased expression of sodium channels), hence they have a role in inducing phantom pain later on in the course.

As mentioned previously, phantom pain can be regarded as an innate response to the removal of a limb or organ, where the nervous system adapts to this loss, producing phantom sensations in the process. In the dorsal horn of the spinal cord, reorganization of nerves occur. The large dorsal horn nerves spread their nerve endings to the small peripheral nerves that have died because of the amputation, thereby connecting, perhaps in a way reviving, the nerves of a body part no longer present. Hence, signals from the amputated area still reach the brain, and the brain interprets these signals to create the sensation that the body part is still there, hence allowing pain to still be felt there. These changes in the peripheral and central nervous system provide part of the biological basis for the cause of phantom pain.

**Phantom pain as a memory**

The reorganization of nerves also occurs in the somatosensory cortex of the brain. Again, leading back to before the body part is removed, either intense or continuous pain was likely to have been experienced. The area in the somatosensory complex responsible for processing the pain increases as a

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result of the increased representation of such pain. After the amputation, these expanded areas that process the neuronal input still remain. This phenomenon works in tandem with the observation that in many amputees, stimulation of other locations (e.g., the mouth) can lead to changes in the sensations of the phantom limb. Neuroscientist Ramachandran described an experiment with an amputee, where touching certain areas of his ipsilateral face resulted in correlating sensations in his phantom arm in precise locations (see Appendix 1).\footnote{Ramachandran, et al. “Use of Visual Feedback, in Particular Mirror Visual Feedback, in Restoring Brain Function | Brain | Oxford Academic.” OUP Academic, Oxford University Press, 8 June 2009, academic.oup.com/brain/article/132/7/1693/328686.}

In a study involving adult owl monkeys with amputated digits, MRI scans for blood flow showed that the area of the somatosensory cortex representing the mouth shifted into the area representing the amputated limb in the primary somatosensory cortex (S1).\footnote{Flor, Herta. “Phantom-Limb Pain: Characteristics, Causes, and Treatment.” 2017, doi:10.18411/a-2017-023.} Hence, we can understand this pain as a sort of memory of previously felt pain: these areas for the phantom limb are still stimulated by neuronal input in other locations, so that the brain interprets the signals from the mouth as signals for the phantom limb, which is likely to lead to pain. This phenomenon provides a possible explanation for why phantom pain is often similar to the pain felt before the amputation, and demonstrates our neuroplasticity - the brain’s ability to change and adapt.

Certain physical features of limbs are also transferred onto their phantom counterparts, such as the shape and size, and the accessories worn, presenting the visual aspect of phantom pain. Alongside vision, proprioception - the ability to sense stimuli arising within the body regarding position, motion, and equilibrium, or the perception of body and self - is heavily involved in this aspect. This idea is seen in Ramachandran’s proposal of learned paralysis in phantom pain, and his mirror box treatment.

While phantom limbs differ from other hallucinations in their ability to be moved and controlled, they are sometimes able to contort themselves into painful positions and become paralyzed, such as in a clenched fist with the nails digging into the palms. Ramachandran hypothesized that the ability to move this phantom limb in the first place is because of the ‘brain being able to monitor its own motor commands to the phantom’. However, due to the lack of visual and proprioceptive confirmation of the movement of the limb, the limb is ‘abandoned’ by the brain - it is a learned process of the brain gradually forgetting the movement of the limb as there is no visual feedback. He devised the simple method of using a mirror box to provide mirror visual feedback: by inserting the phantom limb into one side behind the mirror with the remaining limb on the other side of the mirror, the illusion of both limbs being there is created. When the existing limb moves, the phantom limb is able to be released from its paralysis, echo the motor commands for the other limb and mirror the its motions. This provides the visual feedback necessary for the brain to unlearn the paralysis, and remember the movement. The visual appearance of the phantom limb triggers the proprioceptive feedback to immediately create the feeling of the limb, regaining control or ownership of the limb, alleviating the phantom pain.\footnote{“Phantoms, Shadows, and Sensory Ghosts.” Hallucinations, by Oliver W. Sacks, Vintage Books, 2013, p. 283.}

While the concept of phantom pain arising and fading as a memory appears to be rather abstract and psychological, physical reorganization of nerves and the brain’s processing of visual and proprioceptive feedback suggest that, though the exact mechanism is unclear, there is a biological basis in the brain for phantom pain.
Differentiating between reality and illusion

Ramachandran’s mirror visual feedback shows how the brain can be tricked into mistaking illusions for reality. There is further research into the ability of the primary somatosensory cortex (S1), which is responsible for proprioception and nociception, to differentiate between real touches and illusions. In an experiment with electrodes attached to along the arm, participants reported that real sensation of tingles travelling up the arm (electrodes activated consecutively up the arm) was equally as strong as the fake sensation of tingles travelling up the arm (electrodes activated at wrist then at elbow). While S1 activity did not change between the real sensation and illusion, other higher level brain structures did, suggesting that S1 cannot differentiate between the two, and that it is only the recipient of these structures’ interpretation of the signals. As mentioned previously, S1 plays a significant role in the interpretation of signals from other locations to create phantom pain, hence it opens up more paths to researching the interactions between brain structures that lead to illusions being interpreted as real. This change in our understanding of the brain provides insight into the physical location of the source of phantom pain.

Conclusion

With the gradual increase in our understanding of the human body and the human consciousness, we are now approaching the concept of pain differently. This is aided by new models explaining how we experience pain in relation to the nervous system, and new research demonstrating the abilities and plasticity of the brain in the reorganization of nerves. While certain aspects of phantom pain are not completely understood, for example how the ‘learned paralysis’ actually works in our body, and how our brain differs between real and imaginary, it is acknowledged that pain encompasses much more than physical tissue damage. Therefore, phantom pain cannot be regarded as a ghost in the machine, because as we continue to discover more about the mechanical functioning of our body, it is revealed that physiological responses do occur in relation to more subject concepts of emotion, memory, and body image. Phantom pain has indeed a strong biological basis, and the currently unexplained features of phantom pain will likely be elucidated with further research into the physiological workings of our body.

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