The phantom limb pain

Generally, theories are based on neurological pathway and cortical re-organisation based on amputated people. Neurobiological theories proposed so far are divided into peripheral, spinal and central mechanism, which all overlap each other to a certain extent. Peripheral mechanism states that impulses created by neuromas at the stump are perceived by the brain as pain. On the other hand, the central mechanism explains phantom pain in terms of central sensitisation. Whilst, spinal mechanism describes phantom limb pain as found in the brain itself.

On the other hand, some researchers believe that there are psychological explanations to phantom limb pain. Researchers have proposed that phantom limb pain can be explained by studying the personality of the amputee. Yet researchers have also tried to verify that phantom limb pain is a result of amputated people using defence mechanisms to cope with their loss.

Peripheral mechanism:

According to this mechanism, pain is perceived in the brain as neuromas are formed in the tip of the stump, which create impulses that travel up the spinal cord.

After amputation, fibres of the nerve endings form neuromas that create abnormal impulses travel to the brain. There is good evidence to suggest that neuromas are able to spontaneously fire impulses, it is also known that neuromas increase the sensitivity of mechanical and other stimuli received.

Chabal et al (1989) in an experiment demonstrated that when gallamine, a potassium channels blocker was injected into the neuromas of amputees, their stump pain increased. He suggested that the permeability in ion channel found in neuromas contributed to phantom limb pain. This suggests that neuromas are a possible cause for phantom limb pain, this provides evidence for the peripheral mechanism. More experimental studies support this mechanism, as when neuromas are surgical removed, the pain is reduced at the phantom limb. Again, signifying neuromas are involved in phantom limb pain.

However, there are a lot of observational and experimental studies that do not support the peripheral mechanism. Physical stimulation of neuromas can increase C-fiber activity, therefore the level of phantom pain increase, however, once the neuromas have stopped firing, action potentials pain still persists.

As when peripheral nerves treated with conduction blocking agents, the pain is not completely eliminated, it is only reduced (2), and this suggests that neuromas are not the sole cause for phantom limb pain but there are other factors involved.

Furthermore, studies show that pain can occur in the absence of the stump (7), hence, absence of neuromas, which empathises the view that neuromas are not the sole reason.

In addition to this, pain can occur when limb is congenitally absent. Weinstein and Sersen (1961) described five children with congenital absence who experienced phantom limb pains on a limb that never existed (8). In a

follow-up study, Weinstein et al (1964) reported another 18 cases in which phantoms were experienced in congenitally absent limbs. This suggests that neuromas and the stump may not even be involved in phantom limb pain but may be a factor that increases the sensation felt by patients (9). Moreover, phantom limb pain is present immediately after amputation, this rules out the causal role of neuromas, as the amputated stump have not yet formed neuromas.

More recent research has confirmed these findings suggesting strong evidence for alternative explanations of phantom limb pain, as merely studying the peripheral system may not be sufficient to explaining phantom limb (10-11).

Central mechanism:

According to this mechanism, phantom pain is a result of central sensitisation. Central sensitisation is where sensory neurons in the dorsal horns of the spinal cord become sensitised by peripheral tissue damage or inflammation. This type of sensitisation has been suggested as a possible causal mechanism for chronic pain conditions. It is also suggested by researchers to explain phantom limb pain.

The loss of afferent input from periphery results in irritations in dorsal horn or in the central nervous system leading to permanent changes in synaptic structure and reduced inhibitory processes and increased excitability processes is seen in the dorsal horn.

Under normal circumstances, circuitry in the brain remains largely stable throughout life. However, functional MRI studies in patients with amputated limbs have shown that almost all patients have had a motor cortical reorganisation.

Cortical organization is described in terms of maps known as homunculus. For example, sensory information from the foot projects to one cortical site and the projections from the hand target to another site. As the result of this Organisation based on anatomical relation of sensory inputs to the cortex, cortical representation of the body resembles a homunculus. (Illustration of sensory homunculus).

The phantom limb sensation, which is thought to result from disorganisation in the brain homunculus and the inability to receive input from the targeted area. Studies have shown that there is a high correlation with the extent of physical re-organisation and the extent of phantom pain.

Major motor re-organisation occurs in a downward shift from the hand area of the cortex to the face area, especially the lips. Sometimes there is a side shift of the hand motor cortex to the ipsilateral cortex. In patients with phantom limb pain, the reorganisation was great enough to cause a change in cortical lip representation into the hand areas which occurred during lip movements (14).

There have been many theories proposed to explain this cortical re-mapping but none have been supported. This suggests that the concept of central mechanism cannot be accepted as the sole causal factor in contributing to phantom limb due to its lack of experimental evidence.

Spinal mechanism:

According to this mechanism, phantom limb pain is found in the brain itself. Davies et al (1998) reported that thalamic stimulation evokes phantom pain (15). Holmes (1911) described a case where a patient reported the total disappearance of left leg phantom pain following a lesion in the patient's right hemisphere, (39) suggesting that the pain is found in the brain. Merzenich et al (1984) amputated fingers in monkeys, which lead to a 1-2mm invasion into cortical representation of amputated finger in primary somatosensory cortex suggesting that pain didn't involve the amputated limb but was a lot to do with the organisation of the brain (16). Pons et al (1991) reported a similar finding, but a greater invasion in adult macaques (18). Ramachandran (2000) confirmed this using MEG, where he looked at the re-organisation in the upper limb of amputees, and he found that the sensory input from face activated the hand area in the brain, a 2-3cm invasion (19-21). This provided strong evidence for the spinal mechanism.

Livingston (1943) carried out a study on thirty six amputated people who suffered from phantom limb pain. They were given local anaesthetic into spinal cord, and it was found that nine out of the thirty-six patients said their pain was permanently gone and over two-thirds said they felt temporarily reduced pain. He suggested that ' closed, self-sustaining, reverberating circuits' are set up by chronic peripheral irritations or by release of spinal cord cells from inhibitory control through the loss of afferent input (31). Furthermore, once these circuits are established, surgical removal of the peripheral source has no effect on them and, therefore, will not abolish the pain, suggesting more evidence for the spinal mechanism.

The mechanism of dis-inhibition in the spinal cord resulting in phantom limb pain was examined by Wall (1981). He proposed that the sudden lack of afferent input following amputation results in a number of changes at both the peripheral and spinal level.

Furthermore, Wall proposed that there are both immediate and chronic changes. Such processes described by Wall suggest that the effects of peripheral nerve lesions spread beyond the damaged cells into the spinal cord itself. Therefore, it addresses some of the observations on phantom phenomena not explained by peripheral theories (41). Thus suggesting a more valid theory of explanation of phantom limb pain.

Neuromatrix theory:

The neuromatrix theory was proposed by Melzack (1990), which followed the spinal mechanism. He argues that we have a built-in matrix of neurons which is spread around the brain. When the active matrix is deprived of input from the limbs, phantom pain arises as the neuromatrix produces abnormal firing as a substitute (17). The theory suggests that the conscious awareness and perception of self is created in the brain, which can be modified by different perceptual inputs (22). Melzack describes the basic output from the neuromatrix as a “ neurosignature” which is particular to the individual. The matrix is genetically determined but is modified throughout life, to create a neurosignature. It is this neurosignature of a body part that determines how it is consciously perceived (4).

In the case of amputated patients, abnormal input to the matrix after amputation results in pain. This abnormal input is a result of lack of normal input or high levels of input caused by excessive firing of damaged nerves.

Melzack also suggested that neuromatrix extends to three major neural circuits. The input systems contributing to the neurosignature are primarily the somatosensory, limbic, and thalamocortical systems. Studies carried out on brain-damaged patients have shown that the ones that refused to accept their limbs as part of their body have shown to have damage in their somatosensory systems (25). Output is transferred into conscious awareness by integration of all these system. It is suggested that the pain aspect of output occurs for a number of reasons. For example, cramping may occur because limb movement is the pre-wired part of neuromatrix. Thus, after amputation, neuromatrix receives no signal from periphery that the limb is moving, so neuromatrix output will include the basic neurosignature. This will send strong messages for the limb to move so patients report cramping (23, 26).

Somatosensory pain memory illustrates how experiences can shape the pattern produced by neuromatrix (24). This suggests that neural representation of pre-amputation pain is formed subsequent to one very intense pain experience, since pain memory is experienced as both sensory and affective events. We assume that both experiences are encoded in neuromatrix (27).

This neuromatrix theory is consistent with the spinal, central and peripheral mechanism. The theory also suggests that psychological factors may also

produce input that activates the matrix and results in pain by fear, insomnia, stress, and fatigue (28-29).

The neuromatrix theory is similar to the gate control theory and can be applied to chronic pain not just phantom pain. Gate control theory proposes that pain is the perception whose quality and intensity is influenced by the individual’s history and their state of mind.

However, the neuromatrix theory fails to explain how the pain can spontaneously end and how some amputees do not suffer from phantom limb pain. This is a major criticism, which subsequently reduces the theory’s strength. The theory also does not explain how these factors influence pain. The theory is also very difficult to test, although animal models have been informative, they do not provide great support. However, the neuromatrix does provide a mechanism to explain perception of pain and also psychological management of pain. (30). Overall, it is clear that further research is required in this area.

Psychological explanations:

There are a number of psychological explanations that have been proposed. Some researchers have proposed that phantom limb pain can be explained by considering the personality of the amputee. For example, Parkes (1973) found that those who have a persistent phantom pain scored high on personality measure of ‘ compulsive self-reliance’ and ‘ rigidity’. This suggests that there is something about these personality traits that cause amputated people to have a higher chance of experiencing phantom pain than those who scored lower on the questionnaire test ‘ compulsive self-

reliance’ and ‘ rigidity’. Parkes also suggested that those who are rigid dislike and resist change, so experience phantom pain as they find it difficult to accept the loss of a limb. Those who are compulsive self-reliance also get pain because they feel helpless. Patients become distressed having to rely on someone else (32). However, Sherman said that psychological explanations of phantom pain have less to do with personality but more to do with after amputation experiences. He said that treatment success rate is low so this will cause stress in all but the most persistent, self-reliant individuals to insist treatment (28, 33). Contradicting this Shukla et al (1982) found no difference in personality in amputated people who experience phantom limb pain and those that do not experiences phantom limb (40).

Another psychological explanation is that pain is a result of the use of defence mechanisms such as denial or repression (34-37). Weiss and fishman (1963) suggested that patients may deny limb loss therefore pain is a reinforcement of the existence of the limb (36). Kolb further states that, the more important the body part is to the individual emotionally, the more the patient will be in denial of the loss (34). However, Simmel (1958) argues that phantom pain occurs as individuals are flooded with emotions and anxiety associated with the loss of the body part (35). However, this concept is difficult to test, as the causal link between denial, depression and phantom limb pain cannot be established.

Further difficulty in establishing the role of psychological distress comes from symptoms overlapping, as it is difficult to establish which symptom is reflected by which disorder. For example, difficulty in getting to sleep is regarded as a symptom of depression. Yet, it is also a feature of chronic

pain. Similarly, endorsement of items relating to fatigue is indicative of depression, but is also associated with chronic pain in the absence of depression.

Relationship between phantom limb and psychological distress is confounded by a variety of post-amputation factors. Also, research in this field failed to differentiate between acute adjustment reaction and chronic problems (28), for example it is common to feel reactions of shock, grief and denial, for people who just underwent amputation, (38) and this is expected as part of the post-amputation phase.

Conclusion:

Overall both the neurobiological and psycho explanations are key to explaining phantom limb pain. Yet, the neurological basis and mechanisms for phantom limb pain given in this essay are all based from experimental theories and observations. Our knowledge in this field is little. There is limited empirical evidence to support studies carried on amputated patients. Also, we have a limited knowledge about the true mechanism causing phantom pains which suggests that it is difficult to establish an explanation for phantom limb pain. However, a number of these have been proposed and many theories highly overlap, making it difficult to reach a conclusion.

One of the major weaknesses to the peripheral mechanism is that it has failed to explain how it is possible for the stump to be manipulated and the result sensation felt are pain and not any other sensation such as touch, pressure or itchiness (5-6). Using the information provided, I do not believe

that phantom limb pain can be explained by peripheral mechanism. Yet, I do believe that it does have a role in increasing phantom limb pain.

Spinal mechanism does provide a better explanation for phantom limb pain. However, it failed to explain why phantom limb pain has also been reported in cases where there is no nerve damage and in cases where there has been a complete transaction of the spinal cord (23).

On the other hand, strong evidence supports the psychological explanation for phantom limb pain but recent study suggests that psychological factors do not play causal role in phantom pain but do increase levels of pain sensation (28). It is also interesting to note that this research on “ personality types” are associated with increased phantom limb pain, whereas none discuss personality types that are less likely to experience phantom limb pain. Yet, it could be argued that the neurobiological mechanism has stronger evidence to support phantom limb pain, suggesting a better explanation.