Osteopathy and Mirror Therapy for Phantom Limb Pain

Joseph Tessler
Western University of Health Sciences

Abstract
Phantom limb pain (PLP) is a major complication of limb amputation surgery, affecting up to 75% of post-amputation patients. The exact mechanism of PLP remains unknown, but current research suggests several neurologic pathways may be involved. Treatment for PLP involves a combination of pharmacological and non-pharmacological modalities. Mirror therapy appears to be growing in popularity since it was first researched in 1996 and current therapies attempt to build on its promising results. The goals of this article are to review the current treatment options available as well as propose osteopathic manipulative techniques that may potentially be applied to mirror therapy in the treatment of PLP.

Introduction
Phantom limb pain (PLP) is a common complication of limb amputation. In 2005, there were approximately 1.6 million individuals in the United States living with an amputation; the number is expected to double by 2050. The vast majority of amputations involve the lower limb; of those, greater than 90% occur below the knee. The most common causes for lower extremity amputation are peripheral vascular disease (PVD) and diabetes mellitus (DM). Up to 75% of post-amputation patients reported PLP. The quality of life for post-amputation patients may be severely compromised, and PLP alters the patients’ abilities to perform daily tasks, sleep, and adhere to exercise and rehabilitation programs.

Neurological post-amputation complications can be broadly divided into stump pain, phantom limb sensation, and PLP. Stump pain is defined as pain localized to the portion of the remaining limb after amputation and is characterized as sharp, burning, and electrical-like. Phantom limb sensations are non-painful perceptions of the removed limb, as if the limb is still present. These feelings typically diminish over time. PLP is defined as painful sensations localized to the region of a removed body part and are
often associated with tingling, cramping, cold, or burning sensations.\textsuperscript{6}

**Mechanisms**

PLP has been a well-known complication of amputation patients since 1551, when it was first described by the French military surgeon, Ambroise Pare. However, the exact pathophysiology remains unknown.\textsuperscript{5, 7} PLP is neuropathic, meaning the pain results from primary damage to or lesion of the nerves.\textsuperscript{8} Neuropathic pain may result from trauma, compression (inflammation), metabolic disorders, infections, toxins, or masses.\textsuperscript{9}

Initially, investigators believed PLP was a psychological symptom, but current research suggests that PLP is caused by lesions to three separate areas of the nervous system – the peripheral nerves, spinal cord, and cerebral cortex.

Peripheral nerve transection during amputation results in disruption of normal afferent signaling to the spinal cord. Mechanisms involving pro-inflammatory cascades and microgliosis result in ion channel sensitization, thereby lowering nerve depolarization thresholds, ultimately leading to nociceptor hyper-excitation.\textsuperscript{8, 10} The deregulated peripheral excitation induces changes within the spinal cord as well, inducing axonal sprouting within the dorsal horn of the spinal cord and effectively increasing the receptive field responsible for pain transmission.\textsuperscript{7, 10, 11} Lastly, cortical reorganization may contribute to PLP. Cortical areas initially reserved for the amputated extremity are invaded by neighboring cortical areas.\textsuperscript{7} Stimulation of the cortical regions may reactivate downstream afferent pathways, eliciting pain and sensation. Initially, investigators believed PLP was a psychological symptom, but current research suggests that PLP presents due to input from three distinct levels of the nervous system - the peripheral nerves, spinal cord, and cerebral cortex.

**Management**

There is currently no specific treatment protocol for PLP, but a multi-disciplinary approach which includes pharmacological and non-pharmacological measures appears to be most effective.\textsuperscript{12} Pharmacological regimens are similar to the management of other neuropathic pain which target peripheral and central pain pathways. The common medications utilized in the management of PLP are listed in Table 1.\textsuperscript{7, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22}

Several non-pharmacological modalities have been effective in treating PLP.\textsuperscript{7, 11} A review of the current literature by the Italian Consensus Conference on Pain in Neurorehabilitation also recommended cognitive-behavioral therapy (CBT), hypnosis, and mirror therapy in the treatment of neuropathic pain including PLP.\textsuperscript{23} Traditionally, classic mirror therapy involves super-imposing the image of a residual limb at the location of the phantom limb with a mirror.\textsuperscript{24} Functional magnetic resonance imaging (fMRI) studies by Foell et al. revealed reversal of cortical reorganization in the primary somatosensory cortex and the inferior parietal cortex after 4 weeks of mirror therapy in patients with unilateral arm amputation.\textsuperscript{25} Hypnosis, along with other
Relaxation techniques such as mindfulness and breathing exercises have been shown to alleviate other neuropathic pathologies and may benefit PLP patients. Osteopathic manipulative treatment is also recommended for neuropathic pain patients including PLP. Transcutaneous electrical nerve stimulation (TENS) has shown therapeutic benefit as well. Mulvey et al. found TENS to potentially reduce PLP and stump pain when patients engage in movement and while at rest. Another study by Tilak and colleagues similarly reported short-term pain reduction with TENS, but found no difference between TENS and mirror therapy; furthermore, long-term benefits were not established.

**Treatment Proposal**

Osteopathic Manipulative Treatment (OMT) techniques such as muscle energy (MET), myofascial release (MFR), balanced ligamentous tension (BLT), and counterstrain have been recommended in the treatment of neuropathic pain. However, there has been no research exploring the possible application of these techniques to the contralateral limb of PLP patients during mirror therapy.

**OMT**

OMT can treat neuropathic pain including PLP by reducing mechanical stresses to the body region, balancing neural inputs, and reducing pain by eliminating the nociceptive

---

**Table 1. Pharmacologic Options for Treating Phantom Limb Pain**

<table>
<thead>
<tr>
<th>Drug Class</th>
<th>Mechanism</th>
<th>Medication Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nonsteroidal anti-inflammatory drugs (NSAIDs)</td>
<td>Inhibit cyclooxygenase, and halt production of prostaglandins</td>
<td>Naproxen</td>
</tr>
<tr>
<td>Other analgesic</td>
<td>Cannabinoid receptor agonist and activates vanilloid subtype 1 receptor (TRPV1)</td>
<td>Acetaminophen</td>
</tr>
<tr>
<td>Tricyclic antidepressants (TCAs)</td>
<td>µ- and κ-opioid receptors inhibitors</td>
<td>Nortriptyline, amitriptyline</td>
</tr>
<tr>
<td>Anticonvulsants</td>
<td>Bind calcium channels to reduce Glutamate release</td>
<td>Gabapentin</td>
</tr>
<tr>
<td>Serotonin and norepinephrine receptor inhibitor (SNRIs)</td>
<td>µ- and κ-opioid receptors inhibitors</td>
<td>Venlafaxine, Duloxetine</td>
</tr>
<tr>
<td>Opioids</td>
<td>Bind to opioid receptors in cerebral cortex; may also inhibit GABA interneurons within the spinal cord</td>
<td>Morphine</td>
</tr>
<tr>
<td>NMDA receptor antagonists</td>
<td>Inhibit glutamatergic receptors in cerebral cortex and spinal cord</td>
<td>Ketamine, Calcitonin</td>
</tr>
<tr>
<td>Local anesthetic</td>
<td>Inhibits Na ion channels and halts initiation of nerve impulse and conduction</td>
<td>Bupivacaine</td>
</tr>
</tbody>
</table>
Osteopathic physicians consider the consequence of all innervations to a somatic region including spinal facilitation, proprioceptive function, and the autonomic nervous system. Providing beneficial palpatory techniques to the residual limb during mirror therapy may help reduce PLP by diminishing peripheral afferent firing by the affected limb. Ramachandran and Rogers-Ramachandran noted that afferent sensation of the phantom limb was produced when the residual limb was viewed as being touched in the mirror. Giummarra et al. found that manipulating and touching extremities viewed in the mirror elicited an embodiment response.

Additionally, a recent study by Schmalzl, Ragnö, and Ehrsson revealed that visuo-tactile therapy may be a more efficient therapy for PLP than visual therapy alone. They demonstrated that the combination of visual and tactile stimulation augmented therapy compared to mono-stimulation. If afferent sensation can be produced by palpating the residual limb, it may be possible to modulate the afferent signals with osteopathic manipulation. Likewise, Kawashima et al. reported that active motion during mirror therapy has the potential to increase phantom limb awareness and may increase sensations associated with PLP. They also noted that simulated violent maneuvers to mirrored limbs invoked painful stimuli to the phantom limb. OMT in conjunction with mirror therapy may activate proprioceptive sensations of the phantom limb. Therefore, OMT during mirror therapy may have the ability to elicit changes in the cortical pathway of PLP. Given the results from these studies, it is possible that providing OMT to the residual or unaffected limb in conjunction with mirror therapy may provide some relief and better overall management for PLP. Depending on the characteristic and location of PLP, the specific techniques listed may provide symptomatic relief.

**MET**

Applying MET during mirror therapy may decrease the perceived muscle tension and hypertonicity in phantom limb patients. MET focuses on treating somatic dysfunction by targeting the muscle fibers of a given region. The goal is to reset intrafusal and extrafusal muscle fiber lengths. Applying MET to the contralateral limb of PLP may be beneficial due to the properties of antagonist muscle inhibition and crossed extensor reflexes intrinsic to the neuromuscular system. Antagonist muscle inhibition refers to the fact that active muscle group contraction in a certain direction results in relaxation of the antagonist muscles. The crossed extensor reflex describes the relaxation of agonist muscles in the contralateral limb when a muscle group contracts. The restoration of physiological muscle fiber length reduces hypertonicity and reflex contractions that may be involved in limiting range of motion and muscle ache.

**MFR/BLT/Counterstain**

Visualization of phantom limb treatment with MFR, BLT, and counterstrain during mirror therapy may help reduce inappropriate nociceptive feedback from the affected limb. MFR attempts to free or loosen the fascia surrounding muscle tissue, effectively
reducing restriction around the region and allowing free movement of the tissue. Fascia is connective tissue that surrounds other structures of the body such as muscles and organs.\textsuperscript{30} There are numerous functions of fascia, but those regarding musculoskeletal limbs include muscle movement and stability, proprioceptive communication and feedback, and myofascial tension.\textsuperscript{36} After an injury, fascial layers become thickened and distorted, resulting in chemotaxis that promote fibroblast proliferation and congregation.\textsuperscript{30, 37} Fibroblasts deposit collagen linearly along paths of muscle tension, guided by piezoelectricity – a current produced from mechanical stress. Immobilization of the injured region results in the formation of dense connective tissue that may entrap neuromuscular and vasculature structures, which can lead to stiffness, pain, and edema.\textsuperscript{30, 38} MFR is utilized to reduce myofascial tension and distortion in order to restore neuroreflexive activity, improve circulatory and lymphatic function to an area, and reduce pain. MFR may alter afferent proprioceptive information to change muscle tension.\textsuperscript{35} This is accomplished by reducing excitation of the gamma motor system and restoring the normal length and motion of the muscle spindle.\textsuperscript{30}

BLT can be applied to rebalance the tension across articular joints. It is theorized that ligaments may provide proprioceptive information to help determine muscle response to motion.\textsuperscript{35} The total tension within a given joint is distributed between the stabilizing ligaments. The individual ligaments transmit proprioceptive information to affect muscle tonicity.\textsuperscript{39} Although individual ligaments may undergo changes in tension, the total tension does not change. The total tension is altered when extrinsic factors such as injury, inflammation, or other mechanical forces act upon the joint. When trauma occurs at an articular joint, the ligaments are initially strained and moved into an asymmetrical force distribution and cannot return to their physiologic tension balance.\textsuperscript{30} Asymmetrical tension surrounding articular joints may manifest as increased temperature or hypertonicity of the region during palpation.\textsuperscript{35} Prolonged hypertonicity and inflammation may be interpreted as cramping, stiff, and painful stimulus at the joint region. BLT is accomplished in three components – disengagement, exaggeration, and balance. In disengagement, the joint is either compressed or decompressed in order to allow more articular motion. Secondly, the joint is placed in its original position of injury until a point of balance is found. This position is held, and with the body’s inherent respiratory cooperation, a release occurs that signals the return of the ligaments to their normal physiologic state of tension.\textsuperscript{30}

Counterstrain points are non-radiating tender regions of the body that can be treated by moving the body into a position in which the muscles are shortened.\textsuperscript{35} It is hypothesized that positioning the body reduces nociceptive input to the spinal cord.\textsuperscript{40} Counterstrain tenderpoints are believed to develop from inappropriate hyperactive nociceptive signaling from gamma motor neurons.\textsuperscript{41} Unwarranted myofascial lengthening creates afferent signaling to the cortex which subsequently interprets the signal as potential for myofascial damage. An
The efferent signal is produced which causes rapid contraction of the myofascial tissue. The contraction of the myofascial tissue results in rapid lengthening of the antagonist myofascial tissue produces nociceptive feedback resulting in hypertonic myofascial tissue that manifests as a tenderpoint. Counterstrain technique manipulates the tenderpoint region into a position of minimal tenderness. This decreases nociceptive activity and reduces the afferent feedback signaling tissue damage as well as decrease hypertonicity in the surrounding myofascial tissue.

Conclusion
Treatment for phantom limb pain is best when utilizing a combination of medication, cognitive therapy, and manual therapy. With the growing popularity of mirror therapy, more research is necessary to establish the maximum potential for illusory therapy through supplementary modifications to the classic treatment. Because of their documented benefits for neuropathic patients, OMT techniques such as MFR, soft tissue, BLT, and counterstrain should be considered in conjunction with mirror therapy. To optimize the management of PLP, it is necessary to explore the potential therapeutic benefits of these modalities to the contralateral limb during mirror therapy as well as to the residual stump.

Acknowledgements
The author would like to thank Dr. Calvin J. Okey, DO and Dr. Wanda Shok Yin Chang, MD for their valuable insight and help in researching this article.

Work Cited
11. Hsu E, Cohen SP. Postamputation pain: epidemiology, mechanisms, and


27. Clayton K. The use of manual therapy by amputees with phantom limb pain; a UK based questionnaire study. British School of Osteopathy.


