Management Strategies for Phantom Limb Pain

by

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ABSTRACT

Phantom limb pain (PLP) is a common finding among individuals who have lost any part of their body through amputation. Approximately 80% of individuals with amputations report experiencing PLP. Risk factors for the development of PLP after amputation include uncontrolled pre-amputation pain, residual limb pain (RLP), passive coping skills, and poor adjustment to prosthesis. PLP has existed in the literature for over 500 years, yet the exact mechanism is not understood. Current understanding suggests both peripheral and central nervous system factors contribute to PLP.

Pharmacological treatment modalities for PLP include aggressive pain management before, during and after amputation surgery. Liberal use of opioids postoperatively has been shown effective in minimizing PLP and RLP. PLP is classified as neuropathic pain and use of anticonvulsants like gabapentin and antidepressants are recommended. N-methyl d-aspartate (NMDA) receptor antagonists, such as ketamine, memantine and dextromethorphan, are effective in preventing and treating PLP. Therapy with these medications should be initiated early in the postoperative period. Many individuals often report greater PLP control with non-pharmacological treatments than pharmacological. Non-pharmacological treatments include mirror therapy, virtual reality, trans-electric nerve stimulation (TENS), and relaxation therapy. A multidisciplinary approach to the treatment of PLP is required. Early preoperative assessment of coping skills, adequate preoperative pain management, and patient education about PLP is necessary. Postoperative pain management, social support and use of non-pharmacological treatment modalities should be initiated early for best outcomes.
CHAPTER ONE
INTRODUCTION

In the United States, roughly 158,000 individuals experience limb amputations annually (Darnall, 2008). Phantom limb pain (PLP) and phantom limb sensations (PLS) are commonly found among individuals who have experienced the loss of any body part through amputation. PLP is described as being more intense in the distal areas of the phantoms and have qualities such as throbbing, stabbing, burning, or cramping (Flor, 2002). Others have described the pain as crushing, twisting, or that the phantom limb feels malpositioned (Bloomquist, 2001). Also troubling for the amputee are PLS. Nearly all amputees experience PLS. These non-painful sensations are described as feelings of cold or warmth, tingling, itching or electric sensations (Flor, 2002). Nearly 30% of amputees experience “telescoping” which is retraction of the phantom limb toward the residual limb. In some cases, the sensation of phantom limb disappears altogether. Telescoping is associated with increased PLP (Flor, 2002).

PLP is a commonly experienced, often chronic, long-term, co-morbid phenomenon. The prevalence of PLP among amputees is estimated to range from 30% to 81%, with two-thirds of those patients reporting moderate to very strong suffering (Eichenberger et al., 2005). In patients with upper extremity amputation, 81% reported nonpainful limb sensations, 79% reported PLP, and 71% reported residual limb pain (RLP) (Hanley et al. 2009). Similar results were found in a survey conducted in 2005 revealed that of 914 persons with limb loss, 95% of respondents reported experiencing one or more types of amputation related pain. PLP was most common (79.9%) and RLP was reported in 67.7% of respondents (Ephraim, Wegener, Mackenzie, Dillingham, & Pezzin, 2005).
Many patients do not discuss PLP with their health care providers. Sherman and Sherman (1983) conducted a survey in 764 individuals with amputations and reported 648 experienced significant PLP. Of those, 61% of the patients reported having discussed the problem with their health care providers and only 17% were offered treatment. Further, 29% reported that their complaints were ignored, 5% were told it would go away soon, and the rest were told that nothing could be done. The average intensity of PLP before discussing it with providers was 74 on a scale of 100 (Sherman & Sherman, 1983). Even among war-related trauma amputees, lack of support from medical personnel was a major contributor to lack of reported PLP (Robbins, Vreeman, Sothmann, Wilson, & Oldridge, 2006).

**RISK FACTORS**

Individuals who have bilateral amputations as well as those with lower limb amputation are at a higher risk for developing PLP (Dijkstra, Geertzen, Stewart, & Van Der Schans, 2002). Pre-amputation pain is frequently a risk factor for PLP. Katz and Melzack (1990) demonstrated that patients who report pain at or near the time of amputation experienced similar pain quality and location in the phantom limb after surgery. Preamputation pain is a significant predictor of chronic PLP up to 24 months post surgery. Acute PLP is also a predictor of chronic PLP at 6 and 12 months after amputation (Hanley, Jensen, & Smith, 2007).

Another risk factor for PLP is uncontrolled, residual limb pain (RLP). Amputees who report feelings of being able to move the phantom limb tended to have experience more PLP (Richardson, Glenn, Horgan, & Nurmikko, 2007). Lack of adjustment to prosthetic device was also associated with greater PLP. Individuals reported artificial limb as feeling like a foreign body rather than fusing with the body had significantly more PLP (Kern, Busch, Rockland, Kohl,
& Birklein, 2009). Compounding risk factors for PLP include being divorced, poverty, and having other co-morbid conditions (Darnall et al. 2005). Depression and anxiety tend to decrease after 2 years after the amputation, but social discomfort and body-image anxiety often continue (Horgan & Maclachlan, 2004). Situational stress can significantly increase PLP (Arena, Sherman, Bruno, & Smith, 1990). Passive coping strategies before amputation, especially catastrophizing, were associated with the development of PLP.

HISTORY

PLP has existed in the literature since 1551 when a French military physician, Ambriose Pare, referred to it in his writings on his work with soldiers (Roullet, Nouette-Gaulain, Brochet, & Sztark, 2009). Dr. Silas Mitchell, a civil war neurologist, named the condition PLP in 1871 (Herman, 1998). The PLP phenomenon has puzzled many in health care over the years and its pathophysiology continues to be studied. While PLP and PLS have been referenced in the literature for nearly 500 years, the exact mechanism of PLP remains largely unknown (Wilkes, Ganceres, Solanki, & Hayes, 2008).
CHAPTER 2
MECHANISM OF PHANTOM PAIN

The mechanism of PLP is poorly understood at this time. Unraveling neurologic changes in PLP will aid in its prevention and treatment (Nikolajsen & Jensen, 2001). Several explanations for PLP have been proposed. Earlier, the work was focused on peripheral nerves at the site of amputation. The current understanding suggests influences from both peripheral and central nervous systems contribute to PLP (Table 1).

One mechanism of PLP occurs at the peripheral site of amputation is the formation of neuromas. After amputation, primarily severed myelated afferent nerves endings shrink in the periphery. Consequently, regenerative sprouting of injured nerve endings occurs and these develop into neuromas, which are tangled masses of axons (Flor, Nikolajsen, and Troels, 2006). Ectopic neurological discharges from these stump neuromas send abnormal messages to the spinal cord evoking pain sensations. This is why alodynia, a heightened sensitivity to touch, occurs from a light touch to the site of a neuroma. It has been established that by tapping residual limb neuromas sensations can be felt in the phantom limb (Sugarbaker, Weiss, Davidson, & Roth, 1984). Sometimes, however, PLP occurs before the formation of neuromas. Therefore, other areas of the nervous system have been explored.

Two opposite phenomena occur when a peripheral nerve is severed - a dramatic drop of normal inputs from the periphery and a surge of abnormal activity from the spinal nerves (Casale, Alaa, Mallick, & Ring, 2009). After nerve damage, a cascade effect of abnormal signals and chemical processes occur and cause a state of sensitization, or increased excitability of neurons, within the spinal cord (Flor, 2002). After nerve injury, there is an increase in general
excitability of neurons within the spinal cord (Nikolajsen & Jensen, 2001). Random ectopic nerve firings from damaged peripheral nerves are transmitted to the dorsal horn of the spinal cord. As a result of the increased peripheral nerve activity, excess glutamate (an excitatory neurotransmitter) activates the NMDA receptors. NMDA receptor activation can lead to permanent changes in the synaptic structure of the dorsal horn. At the same time, inhibitory neurochemicals such as gamma-aminobutyric acid (GABA) are destroyed as a result of increased ectopic firings. The two phenomena together cause hyperexcitability of the spinal cord (Flor, 2002). As a result, painful messages that are normally blocked at the dorsal horn level of the spinal cord are transmitted up the spinal cord to the brain. Transmission of noxious input caused by inflammation, irritation or pressure on the severed peripheral nerve induces a prolonged state of hyperexcitability, which leads to increased postoperative pain and PLP (Prantl et al., 2005). This process can lead to neuron death and is thought to contribute to chronic and phantom pain syndrome.

Further, changes in spinal cord excitability result from the cascade effect that takes place after nerve damage. The injury-triggered expression of substance P, a neurotransmitter that is involved in transmission of pain information, causes low threshold afferents to appear more like nociceptors. It is thought that A-delta fiber input from periphery, ectopic input combined with input from residual low-threshold afferents contributes to phantom pain sensations. “A remarkable effect of the spinal changes evoked by nerve injury is that low-threshold afferents can become functionally connected to ascending spinal projection neurons that carry nociceptive information” (Flor et al., 2006, p. 877). The hyperexcitability of the both the peripheral nervous
system and spinal cord is thought to trigger PLP (Flor, 2002).

Cortical changes in the brain take place as a result of amputation (Flor, 2008). According to Melzack (2001), there is a prewired synaptic architecture of neurons within the brain known as the neuromatrix. Melzack (2006) argues that PLP is caused when an area of the neuromatrix is deprived from input from its respective limb and the neuromatrix produces its own abnormal firings in substitution for the lack of input from the periphery. These abnormal firings are stored as pain memory (Woodhouse, 2005). Also, since the brain no longer receives input from the amputated limb, areas adjacent to the somatosensory zone of the cortex can begin providing input to those areas. As a result of this reorganization, the brain senses pain from the body part that is not providing input. With upper limb amputations, magnetic resonance imaging studies have demonstrated shifts in the somatosensory cortex (Flor 2008). It has been proposed that greater cortical reorganization leads to greater PLP.

Ramachandran, Stewart, and Rogers-Ramachandran (1992) suggested changes within the brain map take place in PLP. PLP arises because tactile and proprioceptive inputs from the amputated limb have ceased and sensory information from tissues surrounding the amputated limb encroaches on the brain map area of the corresponding limb. As those surrounding tissues spontaneously release nerve impulses, the brain misinterprets the signals as arising from the missing limb and is felt as PLP. Ramachandran and associates (1995) further theorized that PLP was a disconnection between visual feedback and the proprioceptive representations of an amputated limb. PLP resulted from disruption of normal intention to move the limb and the absence of the appropriate sensory feedback of visualizing the limb movement (Ramachandran & Hirstein, 1998).
The mechanism of PLP is not completely understood, but both peripheral and central factors contribute to the syndrome. After amputation, overactive neurochemicals produce hypersensitivity and perceived pain. Changes in the brain and processing of pain memories also contribute to the phenomena.

Table 1. Mechanisms of PLP

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<td>Glutamate down GABA</td>
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PLP – Phantom Limb Pain

GABA - gamma-aminobutyric acid

NMDA - N-methyl d-aspartate
CHAPTER THREE

MANAGEMENT

Treatment of PLP after limb amputation is difficult and often unsuccessful. Most management strategies are based on expert opinion or anecdotal evidence. Randomized controlled trials for management of PLP are limited. Numerous pharmacological and non-pharmacological treatments have been studied. It is suggested that a multidisciplinary approach to management of PLP is needed. A survey conducted in 1980 identified 68 different treatment modalities for PLP (Sherman & Sherman, 1980). Since then, many treatment modalities have been developed with mixed results.

Factors associated with positive adjustment to loss of limb include greater elapsed time since amputation, greater social support, greater satisfaction with prosthesis, active coping skills, optimistic personality, lower level of amputation at the lower limbs, lower levels of PLP and RLP (Horgan & Maclachlan, 2004). Pre-emptive treatment of PLP should include psychosocial and physical interventions as the relationship between coping styles, pre-surgical pain and pain memories contribute to the development of PLP (Richardson et al., 2007). Other factors that contribute to the development of PLP are noxious intraoperative inputs and acute post operative pain (Lambert et al. 2001).

The Department of Veterans Affairs (VA) and the Department of Defense (DOD) collaborated and developed a comprehensive set of guidelines for rehabilitation of lower limb amputation (Department Of Veteran Affairs & Department Of Defense, 2007). For prevention of PLP, the VA/DOD recommends developing an interdisciplinary, patient-centered post-
surgical treatment plan. Key disciplines should include vascular surgery, plastic surgery, internal medicine, pain management, prosthetics, social work services, case management, nursing, nutrition and occupational therapy. Early use of pharmacological and non-pharmacological means for pain control should be initiated in the preoperative phase and continues throughout rehabilitation and prosthetic training. Recommendations include aggressive post-surgical pain management including appropriate edema control and liberal use of narcotics. Early use of anticonvulsant medications, such as pregabalin or gabapentin, antidepressants, and the use of epidural or regional anesthesia are also recommended. Residual limb management is critical throughout the lifespan of the individual and pain should be managed adequately. Protective measures should be instituted to protect the residual limb through the use of rigid dressings and immobilizers early. Careful prosthetic training is essential to the adjustment of the loss of a limb and in minimizing PLP. Mental health services and support groups for amputees are also highly recommended.

PRE-OPERATIVE MANAGEMENT STRATEGIES

Katz and Melzack (1990) from their work with pain memories theory posit that an obvious strategy in prevention of PLP is to keep patients free from pain as long as possible before surgery to prevent the pain memories. Using both general and epidural anesthesia during amputation surgery may reduce the incidence of PLP rather than general anesthesia alone. Hanley and associates (2007) confirmed in their study of 104 participants that the greatest indicator of chronic PLP and RLP is preoperative pain intensity. Individuals at highest risk for the development of PLP should be provided intensive pain interventions before surgery to prevent or minimize chronic, long-term pain.
Because psychological distress is significantly related to PLP (Hill, 1993), preemptive coping strategies training may reduce PLP. Useful tools for helping amputees include relaxation techniques, coping strategies and stress management. Peer and psychosocial support groups are important components of a multidisciplinary pain management team.

Hanling and associates (2010) described preoperative mirror therapy to prevent PLP in 4 patients undergoing lower limb amputation. The majority of patients reported little or no PLP, no decrease in quality of life, and continued physical therapy treatment postoperatively.

OPERATIVE MANAGEMENT STRATEGIES

PLP can develop immediately after amputation or may not appear for several years. Schley and associates (2008) found that stump pain predominate the postoperative experience of patients undergoing traumatic amputations. Peaks of PLP and PLS occur at one month and 12 months after amputation (Schley et al., 2008). These peaks appear to be predictable and treatment aimed at these predicted times are needed. Lambert and coworkers (2001) investigated whether perioperative epidural block 24 hours before amputation was superior in preventing PLP than infusion of local anesthetic via perineural catheter. They studied 30 patients undergoing amputation and found that epidural blocks were not better at preventing PLP, but gave better relief of residual limb pain immediately postoperatively (Lambert et al. 2001).

Continuous infusion of local anesthetic agents through nerve sheath catheters provide adequate pain control after amputation and lower incidence of PLP (Morey, Giannoni, Duncan, Scarborough, & Enneking, 2002). Brorgi and associates (2009) reported a case with PLP immediately post amputation. A perineural sciatic catheter was surgically inserted and propivacaine was continuous infused. Within 6 hours of treatment, pain score decreased by 90%
and PLP disappeared. When the patient was free of PLP at 28 days, the infusion was stopped. No PLP sensations were reported at 6, 12, 24, and 36 months follow-ups (Borghi, Bugamelli, Stagni, Genco, & Colizza, 2009).

Prantl and associates (2005) described a novel surgical technique of joining dissected ends of severed nerves in order to treat patients with long-standing PLS and PLP. The hypothesis was by joining the ends of these severed nerves together, the technique would block the processes that cause hyperexcitability of the spinal cord resulting in increased post-operative pain and PLP. After surgery, patients received 5 days of sciatic nerve block through a microcatheter with local anesthesia. Of the 15 patients studied, 14 were free of PLP at 1 week, 3 months, 6 months, and 1 year after the procedure.

POST-OPERTIVE MANAGEMENT STRATEGIES

Medications

Mainstay pharmacological therapies for PLP included opioids, anticonvulsants and antidepressants (Morey, Giannoni, Duncan, Scarborough, & Enneking, 2002). Tricyclic antidepressants (TCAs) have been proven effective in treating different types of neuropathic pain. Tramadol is recommended for stable PLP and residual limb pain during the first month. Opioids are commonly effective for other neuropathic pain and can be considered for PLP. Gabapentin has demonstrated efficacy in other neuropathic pain but its efficacy for PLP has been mixed. Sodium channel blockers, lidocaine and mexiletine, have also been used to treat neuropathic pain with mixed results.
The World Health Organization (WHO) recognizes PLP as a significant problem and recommends treatment guidelines. The organization classifies PLP as a neuropathic pain and treatment recommendations are geared toward that classification. The WHO recommends a three step analgesic ladder. The first step is treatment with non-opioid analgesic. The second step for moderate to severe pain is to add a weak opioid to current regimen. The third step of the WHO ladder is adding a strong opioid like immediate release morphine (Mishra, Bhatnagar, Gupta, & Diwedi, 2008).

In a systematic review showed that in early PLP, less than two weeks post-operatively, no treatments were more effective than opioids. Therefore, clinicians need to administer adequate opioid analgesics that offer adequate pain relief to reduce risk of subsequent development of PLP (Halbert, Crotty, & Cameron, 2005). PLP persisting longer than six months has a poor prognosis for improvement and treatment is difficult. However, Wilder-Smith and associates (2006) showed tramadol and amitriptyline were effective in treating long-standing PLP in treatment naïve patients.

N-methyl d-aspartate (NMDA) receptor antagonists have been shown effective in treatment of PLP. This group of medications includes ketamine, dextromethorphan, and memantine. NMDA receptors play a crucial role in neural excitation and causing ionic channels to be permeable to calcium (Petrenko, Yamakura, Baba, & Shimoji, 2003). NMDA antagonists slow those processes by blocking the hyperexcitability of the nervous system caused by the influx of calcium caused by ectopic chemical processes after nerve damage. However, over-antagonism may occur and may be problematic as calcium is necessary for normal nerve function.
Ketamine reduces central hyperexcitability by its antagonistic activity at the NMDA receptor sites (Nikolajsen et al. 1995). Hocking and Cousins (2003) reviewed several studies that used ketamine for the treatment and/or prevention of PLP and found ketamine to be effective in reducing both surgical residual limb pain and phantom pain. In subjects who suffered from chronic PLP, ketamine has been shown to reduce PLP 48 hours after administration (Eichenberger et al., 2008). Ketamine has an elimination half-life of 2.5 hours, therefore, it may lead to spikes in serum concentration resulting in significant psychotomimetic effects, such as restlessness, hallucinations and anxiety disturbances (Hackworth et al., 2008). It has been reported that a majority of subjects received ketamine experienced psychotomimetic side effects (Hocking & Cousins, 2003). Because these side effects are severe, long-term use of ketamine is not ideal.

Memantine is another NMDA antagonist that binds to open NMDA receptors. Memantine has an elimination half-life of 60-80 hours compared to 2.5 hours for ketamine (Hackworth et al., 2008) so it is better tolerated and has fewer hallucinogenic side effects as ketamine. Successful treatment of PLP with memantine is related to early initiation of treatment at the earliest signs of PLP (Hackworth, Takarz, Fowler, Wallace, & Stedje-Larsen, 2008).

Dextromethorphan is another NMDA-receptor antagonist. Abraham and associates (2002) conducted a small, randomized controlled study describing the efficacy of oral dextromorphan in the treatment of PLP. This medication was found to effectively reduce PLP without significant side effects (Abraham, Marouani, Kellender, Meller, & Weinbroum, 2002).
Wu and associates (2008) studied the use of mexiletine, an oral sodium channel blocker, and sustained release morphine in treating PLP. Results showed that morphine treatment provided lower pain scores compared with placebo and mexiletine. The study results indicate that morphine, not mexiletine, resulted in a decrease intensity of post amputation RLP. The discussion also showed a higher level of side effects with the use of morphine.

Aggressive pain control with opioids is crucial post-surgically to aid in prevention of PLP. NMDA receptor antagonists are effective in prevention and treatment of PLP. Patients need to be monitored closely for psychotomimetic side effects. Antidepressants and anti-convulsants have been shown helpful for neuropathic pain and are recommended as mainstay therapy for PLP.
CHAPTER FOUR
NON-PHARMACOLOGICAL STRATEGIES

Ketz (2008) conducted a retrospective descriptive study to determine the prevalence and characteristics of PLP as well as effectiveness of self- and standard medical treatment methods in combat-related traumatic amputees. Sixty-eight percent of patients received treatment from their health care providers and most were treated with gabapentin. However, greater pain relief was achieved with non-pharmacologic treatments. The conclusion of this study indicated the need for more effective interventions for PLP.

Ramachandran and associates (1998) developed a mirror therapy based on the theory that PLP was a result of a visual disconnects in brain. A mirror angled to allow the reflection of the intact limb gives the illusion that the patient has two intact limbs. Patients with chronic phantom limb spasms using Ramachandran’s mirror therapy found immediate relief after visualizing movement of “both” limbs.

Darnall (2008) reported a case study regarding a veteran with a recent traumatic lower limb amputation. Multiple pharmacological agents were prescribed, but did not improve the patient’s PLP. The patient reported a correlation between frequency of mirror therapy and decrease in pain intensity. Mirror therapy for 20 minutes a day for 1 month resulted in resolution of PLP. This case presentation showed that mirror therapy can be an effective therapy which is inexpensive and can be done by patients at home.

Cole and associates (2009) replicated the mirror therapy through the creation of a virtual reality environment that allowed participants to move their phantom limbs by moving the
residual limb. Participants, who reported having the ability to control of movement of the phantom limb within the virtual environment, reported a 22-100% reduction in pain.

Other non-pharmacologic strategies include using trans-electric nerve stimulation (TENS). Individuals with active PLP given home TENS stimulation to the contralateral limb reported improvement in their PLP after one year of therapy (Giuffrida, Simpson, & Halligan, 2010). Wilkes and associates (2008) reported successful treatment of chronic lower limb PLP with pulsed radiofrequency treatment of the sciatic nerve. These researchers reported that this technique allowed a patient who had experienced chronic, extreme PLP for 4 years post-amputation to be weaned from all oral analgesics.

Distraction techniques like immersive virtual reality (VR) have helped patients learn to modulate their pain (Gromala, Shaw, & Song, 2009). These immersive techniques mimic real environments. The VR system combines meditation and biofeedback. This allows the users to exert control and, as a result, pain is decreased.

A multidisciplinary approach to post-operative management of amputation is necessary in preventing and treating PLP. Pharmacological strategies in the management of PLP include medications for acute post-operative pain and neurolopathic pain. Liberal use of opioids and NMDA receptor antagonists should be initiated early for adequate PLP and RLP control. Careful post-operative management of the RLP is beneficial in preventing PLP. Non-pharmacologic strategies may be more helpful for some patients, so patients should be offered these treatment modalities. Support groups and teaching effective coping skills have been shown to decrease incidence and severity of PLP. (Figure 1)
Figure 1. PLP Strategies
CHAPTER 5

CONCLUSION

PLP is complex, yet common after amputation. It is a phenomenon experienced by most amputees. Patients need to know PLP and PLS are “normal” and should be treated as an expected outcome of surgery. It is important for providers to have clear, open discussions with the patient about PLP. No single treatment has been shown to be effective in eliminating PLP, however, effective treatment strategies should involve multiple modalities. Effective pain management should be initiated before, during and after amputation surgery can decrease incidence of pain memories and prevent PLP. Evaluation of coping skills before amputation surgery is essential. Nurses can aid patients in developing active coping strategies. Many pharmacological treatments are available, but non-pharmacological treatments may be more effective. Further research is needed to study the causes, prevention and effective treatment modalities for PLP.
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