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PAINFUL AND NON-PAINFUL PHANTOM LIMBS: THE INFLUENCE OF PERIPHERAL AND CENTRAL FACTORS

by

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A thesis submitted to the Faculty of
Graduate Studies and Research in
partial fulfillment of the requirements
for the degree of Doctor of Philosophy

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ABSTRACT

The four studies in this thesis deal with peripheral and central factors in phantom limb phenomena. Study 1 documents sensations referred to the phantom limb during transcutaneous electrical nerve stimulation (TENS) applied at the outer ears, face, and torso. The results suggest that some phantom sensations are maintained by activity of sympathetic fibers in the stump. Other sensations appear not to involve a peripheral mechanism. Studies 2 and 3 focus on sympathetic nervous system correlates of phantom sensations reported during TENS applied at the outer ears and contralateral leg. The results indicate that TENS significantly reduces the intensity of phantom limb paresthesias whereas a placebo control has no effect. Significant relationships between (a) phantom limb intensity and (b) stump skin conductance and temperature suggest that a cycle of sympathetic-efferent--somatic-afferent activity explains phantom limb paresthesias. Skin temperature was significantly lower at the stump than the intact limb in amputees with phantom limb pain (PLP), but not in those with a painless phantom, or no phantom limb at all, indicating that PLP is associated with reduced blood flow to the stump. Study 4 examines PLP that resembles pain experienced in the limb before amputation. These somatosensory memories constitute compelling evidence of a purely central component of PLP. Finally, amputees with and without PLP could not be differentiated by scores on personality, depression, or anxiety inventories. The results of the four studies indicate that the experience of phantom limb paresthesias involves a central mechanism that acts on peripheral structures whereas others qualities of experience depend exclusively on activation of central neural structures. Implications for treatment, research, and clinical practice are discussed.

RESUME

Les quatre études présentées portent sur la nature des facteurs périphériques et centraux reliés aux phénomènes du membre fantôme. La première étude documente les sensations perçues dans le membre fantôme lors de la stimulation électrique percutanée des nerfs (SEP) effectuée sur les points de l'oreille externe, du visage et du torse. Les résultats suggèrent que certaines sensations sont entretenues par l'activité de fibres sympathiques du moignon, tandis que certaines autres ne semblent pas impliquées de mécanisme périphérique. Les études 2 et 3 examinent les paramètres sympathiques des sensations fantômes rapportées lors de la SEP des oreilles externes et de la jambe controlatérale. Les résultats indiquent que la SEP réduit de façon significative l'intensité des paresthésies du membre fantôme tandis qu'un contrôle placebo n'a aucun effet. Des relations significatives entre (a) l'intensité du membre fantôme et (b) la conductivité et la température de la peau du moignon suggèrent qu'un circuit efférent-sympathique--afférent-somatique est responsable des paresthésies du membre fantôme. La température de la peau du moignon était considérablement plus basse que celle du membre intact chez les amputés souffrant de douleur au membre fantôme (DMF); par contre, ceci n'était pas le cas chez les amputés sans douleur fantôme ou sans membre fantôme. Ceci indique que la DMF est associée à une réduction de la circulation sanguine au moignon. L'étude 4 examine les cas de DMF semblables à la douleur ressentie dans le membre avant l'amputation. Ces mémoires somatosensorielles témoignent en faveur d'une composante centrale à la DMF. Finalement, les amputés avec et sans DMF n'ont pu être différenciés d'après leurs résultats sur des questionnaires de personnalité, dépression, et anxiété. Les résultats de ces quatre études indiquent que les paresthésies du membre fantôme impliquent un mécanisme central agissant sur les structures périphériques, alors que les autres sensations perçues dépendent exclusivement de l'activité de structures nerveuses centrales. L'implication de ces résultats pour les méthodes de traitement, la recherche, et la pratique clinique est examinée.

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INTRODUCTION

"There is something almost tragical, something ghastly, in the notion of these thousands of spirit limbs haunting as many good soldiers, and every now and then tormenting them with the disappointments which arise when ... the keen sense of the limb's presence betrays the man into some effort, the failure of which of a sudden reminds him of his loss" (Mitchell, 1871, p. 565-566).

Many patients awake from the anesthetic after an amputation feeling certain that the operation has not been performed. They feel the lost limb so vividly and clearly that only when they reach out to touch it do they realize it has been cut off. This startling realization does nothing to subdue the reality of the limb, and may even intensify the sensations which define it. This phenomenon was first reported in 1545 by the French surgeon Ambroise Paré (Keynes, 1959) and later by Descartes (Lott, 1986). But it was Weir Mitchell, in 1871, who coined the term "phantom limb" and one year later, as part of his seminal book on nerve injuries (Mitchell, 1872), included a chapter devoted to the study of painful and non-painful phantom limbs. The term "phantom limb" so accurately captures the nature of the phenomenon that it was adopted by virtually all subsequent writers.

Overview of the Thesis

This thesis consists of four studies which deal with various aspects of the phantom limb experience. As an introduction to this work, I will first describe the painless and painful phantom limb. Next, I will briefly present the conditions, other than amputation, which give rise to the experience of phantom phenomena. This is followed by a presentation of some of the many qualities of referred phantom sensations that have been reported. Causal mechanisms which have been proposed to explain the phantom are then briefly reviewed, followed by a presentation of the role of psychological and emotional factors, the autonomic nervous system and more central influences. It is clear that certain qualities of phantom limb sensation may be triggered by peripheral factors. Other experiences are entirely independent of peripheral

inputs. The diversity and variety of referred sensations indicate that they cannot be explained by a single mechanism.

The research presented in this thesis demonstrates the importance of both peripheral and central factors in the experience of painful and non-painful phantom limbs. Study 1 is a series of case presentations of patients with phantom limb pain which documents the many qualities of sensations referred to the phantom limb during transcutaneous electrical nerve stimulation (TENS) applied at the outer ears, face and torso. The results of Study 1 raise the possibility that referred phantom sensations may be governed by activity in post-ganglionic sympathetic fibers located in the stump. Studies 2 and 3 focus on peripheral sympathetic nervous system correlates of painful and non-painful phantom sensations reported during TENS applied at the outer ears. On the basis of significant relationships between (a) phantom limb intensity and (b) stump skin conductance and skin temperature, it is proposed that a normal level of activity in sympathetic efferents located in the stump is a sufficient condition for the perception of paresthesias and dysesthesias referred to the phantom limb. The sympathetic nervous system also is implicated in the development or maintenance of phantom limb pain by the finding that stump skin temperature is significantly lower than the intact limb in subjects with phantom limb pain, but not in subjects with a painless phantom or those with no phantom limb at all. Study 4 is a retrospective examination of phantom limb pain experiences that resemble the pain experienced in the limb prior to amputation. Pre-amputation pain is frequently reported to persist after the limb has been cut off, providing some of the strongest evidence to support a central component of phantom limb pain.

The Painless Phantom Limb

Description

In the days immediately after an amputation, when the phantom limb sensation is most vivid and striking, it is common for a patient to report feeling the presence of the entire limb. Typically, the predominant sensation is one of intense paresthesias -- an excessive,

occasionally painful, tingling throughout the absent limb. Initially, the paresthesias overshadow other more subtle qualities of phantom experience. These include a sense of temperature (Carlen, Wall, Nadvorna, & Steinbach, 1978; Haber, 1956; Jensen & Rasmussen, 1989; Sunderland. 1978), posture (Carlen et al., 1978; Jensen & Rasmussen, 1989; Riddoch, 1941), length (Cronholm, 1951; Guéniot, 1861; Haber, 1956; Weiss & Fishman, 1963), volume (Browder & Gallagher, 1948; Cronholm, 1951; Morgenstern, 1964), and movement (Henderson & Smyth, 1948; Friedmann, 1978; Katz & Melzack, 1987; Mitchell, 1871, 1872; Simmel, 1958).

Prevalence and course

The presence of a non-painful phantom after an amputation is so common that it has become standard practice to prepare the patient pre-operatively for its subsequent occurrence (Dernham, 1986; Solomon & Schmidt,1978). Estimates of its prevalence range from about 80% to 100% (Cronholm, 1951; Ewalt, Randall, & Morris, 1947; Feinstein, Luce, & Langton, 1954; Haber, 1956; Henderson & Smyth, 1948; James, 1973; Jensen, Krebs, Nielsen, & Rasmussen, 1983; 1984; 1985; Kegel, Carpenter, Burgess, 1977; Mitchell, 1872; Parkes, 1973; Pitres, 1897; Randall, Ewalt, & Blair, 1945; Roth & Sugarbaker, 1980; Sliosberg, 1948; Solonen, 1962; Varma, Lal, & Mukherjee, 1972). Much of the variability can be attributed to differences in the length of time between amputation and interview. The frequency of occurrence of a painless phantom does not appear to be related to the extremity, side, level of amputation, age, gender, or reason for amputation (Jensen et al., 1984; Jensen & Rasmussen, 1989).

Most patients report that they first became aware of the phantom limb on awakening from the anesthetic after the amputation (Feinstein et al., 1954; Jensen et al., 1984; Jensen & Rasmussen, 1989; Simmel, 1958; 1962). There is considerably more variability in the estimates of the duration of the non-painful phantom. In some amputees, the phantom may disappear within days or weeks of the amputation (James, 1887; Solonen, 1962). In others, it

remains unchanged for up to 60 years (James, 1887; Jensen & Rasmussen, 1989; Van Bogaert, 1934). However, in the majority of cases, the normal phantom undergoes a number of predictable changes within several years of amputation (Jensen & Rasmussen, 1989; Jensen et al., 1983, 1984, 1985). These include fading, telescoping, and shrinking.

Fading. With the passage of time, the proximal parts of the phantom begin to fade and soon disappear, leaving a gap between the stump and the remaining phantom parts. The phantom extremity and digits become the most salient features of the limb. At this stage, the phantom may continue to fade or begin a process of telescoping or shrinking. The extent of fading is variable so that, in the case of an above-elbow amputee several years after the amputation, all that may remain is the experience of the thumb and index finger extended in phenomenal space. Continued fading may eventually lead to complete disappearance. Once a phantom has faded or disappeared completely, it can usually be brought back by a wide range of exteroceptive and interoceptive stimuli.

Telescoping. After some fading has occurred, the phantom limb usually consists of only the hand or foot. These are felt at a normal distance from the body. The intervening parts have disappeared so that the phantom hand or foot feels as if it were hanging unattached in empty space. At this stage the process of telescoping may begin (Guéniot, 1861). The hand or foot gradually approaches the stump and the gap begins to close. Among lower extremity amputees, the phantom foot may be located in phenomenal space on a level with the calf of the other leg. As the telescoping process continues, the amputee may find that the foot is "attached" to the stump. Later, the phantom foot may retract into the stump so that only the tips of the toes jut out. Eventually even these may retreat further, disappearing completely and sometimes permanently into the stump.

There are times when a completely telescoped phantom will temporarily lengthen so that the hand or foot is once again felt at a normal distance from the stump (Bors, 1951; Cronholm, 1951; Gallinek, 1939; James, 1887; Jung, 1948a; Mitchell, 1872; Pitres, 1897;

Riddoch, 1941; Simmel, 1956). More rarely, the converse also occurs; a phantom which is already telescoped may temporarily retract completely into the stump, or one which is of normal, or shortened length, may become even shorter (Haber, 1958; Henderson & Smyth, 1948; Jalavisto, 1950; Simmel; 1956). The circumstances and stimuli which bring about these changes are extremely varied and range from peripheral stimulation of the stump and other regions of the body to more central influences.

Shrinking. When Guéniot (1861) first introduced the phenomenon of telescoping he also reported that it might be accompanied by a marked shrinking of the phantom to the dimensions of a child's limb. This gradual diminution in perceived size or volume has since been reported to occur both in upper and lower limb amputees (Bornstein, 1949; Browder & Gallagher, 1948; Cronholm, 1951; Gallinek, 1939; Haber, 1955, 1956, 1958; Henderson & Smyth, 1948; Jensen & Rasmussen, 1989; Livingston, 1938; Morgenstern, 1964; Pitres, 1887; Schilder, 1950; Simmel, 1956a, 1962; Solonen, 1962). The extent of shrinking that occurs is quite variable. Phantoms of adult amputees have been reported to be the size of a doll's or a baby's hand (Browder & Gallagher, 1948), a silver dollar (Livingston, 1943) and even as small as a postage stamp (Morgenstern, 1964).

Factors governing the fading, telescoping, and shrinking of the phantom are not well understood. Briefly, the gradual fading of the phantom is thought to represent a perceptual correlate of the re-establishment of control over hyperactive or spontaneously active cells which subserve the phantom (Carlen et al., 1978; Wall, 1981). The gradual telescoping and shrinking have been hypothesized to depend upon input from intact peripheral fibers located in the stump and appear to correspond to the known short- and long-term changes in the reorganization of central nervous system structures including somatosensory cortex (Jenkins, Merzenich, & Ochs, 1984; Katz, 1989a; Merzenich, Nelson, Stryker, Cynader, Schoppmann, & Zook, 1984).

The Painful Phantom Limb

Description

Pain is among the many sensations which define the phantom limb. As Melzack and Wall (1988) and Wall (1981) have pointed out, the distinction between a painful and painless phantom is not a clear cut one, but for purposes of description and classification (Merskey, 1986) it useful to separate them. Painful phantom experiences vary along a number of dimensions which include intensity, quality, location, frequency, and duration. The various qualities of phantom limb pain have been the subject of several studies and reviews (Bradley, 1955; Brown, 1968; Gillis, 1964; Jensen et al., 1983, 1985; Jensen & Rasmussen, 1989; Krebs, Jensen, Krøner, Nielsen, & Jørgensen, 1985; Sherman, Sherman, & Parker, 1984; Sunderland, 1978). Phantom limb pain may be an intensification of non-painful paresthesias (Jensen et al., 1984, 1985; Sherman et al., 1984) Some sufferers report paroxysmal shooting pains that travel up and down the limb (Appenzeller & Bicknell, 1969; Katz & Melzack, 1987; Sliosberg, 1948). In others the phantom limb is reported to be in a cramped or otherwise unnatural posture that gives rise to excruciating pain (Brown, 1968; Gessler & Struppler, 1983; Mitchell, 1872). In still others, the phantom pain may resemble a pain that existed in the limb prior to its removal (Jensen et al., 1985; Jensen & Rasmussen, 1989; Katz & Melzack, 1988; Leriche, 1947a, 1947b; L'Hermitte & Susic, 1938; Nathan, 1956, 1962, 1985). Frequently, amputees suffer from several types of phantom pain (Jensen et al., 1985; Russell & Spalding, 1950) such as sharp, lancinating pains referred to specific locations in the phantom superimposed on a continuous background of diffuse burning pain.

Prevalence and course

Early reports estimated the risk of developing phantom limb pain after amputation to be extremely low. Estimates ranged between 1% and 10% (Ewalt et al., 1947; Randall et al., 1945). Unfortunately, these studies drastically under-estimated the incidence of phantom limb pain. Recent surveys based on several thousand amputees reveal that between 78% (Sherman

et al., 1984) and 85% of patients (Sherman and Sherman, 1983) continue to experience significant amounts of phantom limb pain more than 25 years after amputation. Even the more conservative estimates far exceed the earlier figures. Jensen et al. (1985) found that two years after amputation, 59% of their sample still complained of painful phantom limbs. Seven years after amputation, the prevalence of phantom limb pain was 52 % (Krebs et al., 1985). Using a very conservative criterion, Sherman and Sherman (1983) estimated no less than 51% of their sample of 1,200 amputees continued to live in pain more than two and a half decades after losing their limbs. Phantom limb pain appears to be equally prevalent among men and women, and does not appear to be related to side, level, age, or cause of amputation (Jensen et al., 1985; Jensen & Rasmussen, 1989; Sherman & Sherman, 1980; Sherman et al., 1984).

The high frequency of pain after amputation is all the more striking when one considers the low success rate of treatments for phantom limb pain (Sherman, 1980; Sherman, Sherman, & Gall, 1980). Of the more than 68 currently available published treatments, not one is effective in treating a majority of patients. Furthermore, many studies designed to evaluate treatment efficacy use small numbers of patients and rarely control for placebo effects. Sherman et al. (1980) note that studies which use adequate sample sizes report success rates at the end of treatment of about 30 percent. This is no better than the placebo response rate for chronic pain patients (Sherman et al., 1980). In the long run, fewer than one percent of patients with phantom limb pain report complete and lasting relief (Sherman et al., 1984; Sherman and Sherman, 1985).

The different qualities of phantom limb pain, its high prevalence and low treatment success rate point to the complexity of the problem and suggest that there is not just one cause of phantom limb pain. The development and severity of this disorder is determined by the interaction of multiple inputs which vary across individuals and include one's past experience with pain and the immediate physiological, motivational and psychological states of the individual (Melzack, 1971, 1974; 1989a; Melzack & Casey, 1968; Noordenbos, 1959; Melzack and Wall, 1988). Clearly there is a need for clinical investigations of the

physiological mechanisms and psychological processes associated with phantom limb pain as well as for placebo-controlled clinical trials and outcome studies aimed at modifying these mechanisms and processes once they have been isolated.

Conditions which Give Rise to Phantoms

It is now well established that amputation of a limb is only one of the conditions which give rise to the experience of a phantom (Frederiks, 1963, 1969; Melzack & Loeser, 1978; Melzack, 1989a; Riddoch, 1941; Simmel, 1958). Any structure which has the capacity to elicit sensations in the intact body has the potential to become a phantom after it has been removed or deafferented. Phantom limbs occur after brachial plexus avulsions in which the spinal roots are torn from the spinal cord (Angelergues, Hécaen, & Guilly, 1958; Bruxelle, Travers, & Thiebaut, 1988; Jensen & Rasmussen, 1989; Kissel, Rousseaux, & de Ren, 1950; L'Hermitte & Sébillotte, 1938; Narakas, 1981; Riddoch, 1941; Reisner, 1981b; Scotti, 1969; Sweet, 1975; Wynn Parry, 1980, 1983). They occur very frequently after spinal cord lesions, injuries and complete transections (Berger & Gerstenbrand, 1981; Bors, 1951, 1963; Conomy, 1973; Ettlin, Seiler, & Kaeser, 1980; Evans, 1962; Mayeux & Benson, 1979; Melzack & Loeser, 1978; Pollock, Boshes, Arieff, Finkelman, Brown, Dobin, Kesert, Pyzik, Finkle, Tigay, & Zivin, 1957; Sweet, 1975; Weinstein, 1962). In addition, phantom limbs have been reported to occur after lesions of the somatosensory pathways and nuclei in the brain (Critchley, 1971; Frederiks, 1963; 1969; Nathan, Smith, & Cook, 1986; Riddoch, 1941).

It is important to realize that in these cases the actual limbs are still present but their input has been cut off from the brain. A stimulus applied to the deafferented region is not appreciated even though the patient feels the presence of a limb -- the phantom limb. These patients may not be aware that the limb they feel is not the limb they see until the two become dissociated and the phantom is felt to occupy a space or assume a posture that differs from the place or position of the real limb.

Removal of body parts other than limbs has also been reported to give rise to the perception of a phantom, including the eye (Cohn, 1971; Critchley, 1950; Hoffman, 1955), nose (Hoffman, 1955; Riddoch, 1941), breast (Bressler, Cohen, & Magnussen, 1955, 1956; Christensen, Blichert-Toft, Giersing, Richardt, & Beckmann, 1982; Cofer, 1981; Jarvis, 1967; Simmel, 1966; Staps, Hoogenhout, & Wobbes, 1985; Weinstein, Vetter, & Sersen, 1970), penis (Heusner, 1950), tooth (Marbach, 1978; Pöllmann, 1981; Reisner, 1981a), and tongue (Hanowell & Kennedy, 1979).

Painful and non-painful phantom sensations have also been reported to occur after the surgical removal of internal organs. Ulcer pain has been reported to persist after vagotomy (Szasz, 1949) or subtotal gastrectomy with removal of the ulcer (Gloyne, 1954), labour pain and menstrual cramps following total hysterectomy (Dorpat, 1971), the sharp, burning bladder pain of cystitis despite complete removal of the bladder (Brena and Sammons, 1979), pain referred to the anus and rectum after being removed (Baker, 1984; Boas, 1983), and the pain of a severely ulcerated cornea after enucleation of an eye (Minski, 1943). Non-painful sensations include micturation and a full bladder after its removal (Arcadi, 1977; Bors, 1951; Kane & Simes, 1977), and the passing of feces and gas following the removal of the rectum (Bors, 1951; Critchley, 1950; Farley & Smith, 1968). These phantoms sensations and pains are experienced as if the actual organs were still present. They are as real to the patient as were the original sensations which accompanied the normal functioning of the organs prior to their removal.

Even procedures which produce temporary and reversible forms of deafferentation have been reported to produce phantom limbs. These include routine local anesthetic blocks for dental treatment (Wall, 1981; Melzack & Wall, 1988), blocks of the brachial plexus (Melzack & Bromage, 1973; Tatlow & Oulton, 1955), and spinal roots and cord (Bromage & Melzack, 1974; Khurana, Singh, Chhabra, & Kamra, 1979; Miles, 1956; Prevoznik & Eckenhoff, 1964; Wallgren, 1954). Phantom limb phenomena also occur after occlusion of an arm by a blood-pressure cuff (Gross & Melzack, 1978; Wall, 1981).

Referred Pains and Other Sensations

Sensations are often referred to the phantom limb after applying stimuli to virtually any site on the body surface. The circumstances and stimuli which produce these changes are extremely varied and range from peripheral stimulation of the stump and other regions of the body to more central influences. Referred phantom sensations, including transient changes in the perception of the length of the limb, have been produced by surgery of the stump (Simmel, 1956a), bringing an object near the stump (Haber, 1958; Henderson & Smyth, 1948; Jalavisto, 1950; Shukla Sahu, Tripathi, & Gupta, 1982b; Simmel, 1956a), injections of local anesthetics into the stump or stellate ganglia ipsilateral or contralateral to the amputated limb (Jung, 1948a, 1948b; Livingston, 1938, 1943), attaching a weight to the stump or prosthesis (Jung, 1948a), placing the stump and contralateral limb in warm and cold water (Bors,1951, 1963) and putting on an artificial limb (Bors, 1951; Cronholm, 1951; James, 1887; Jung, 1948a; Mitchell, 1872; Pitres, 1897; Riddoch, 1941; Simmel, 1956a). A variety of cognitive and emotional stimuli, including hypnosis (Schilder, 1950), concentration (Morgenstern, 1964; Riddoch, 1941; Simmel, 1956a), fright (Henderson & Smyth, 1948), forceful reminders of the events which led to amputation, the sight of other amputees (Simmel, 1956a), and witnessing cruel and violent acts (Pilowsky & Kaufman, 1965; Stengel, 1965) are known to influence the quality of the phantom limb experience.

In addition, "spontaneously" occurring sensations and pains may be referred to the phantom from diseased viscera along typical and atypical pathways. Bouts of angina are felt as pain referred to the phantom left arm (Cohen, 1944, 1976; Cohen & Jones, 1943, Deenadayalan, 1976; Leriche, 1947c; L'Hermitte, de Robert, & Nemours-Auguste, 1940). Sciatica is referred down the back of the phantom leg (Bornstein, 1949; Finneson, Haft, Kreuger, 1957; King, 1956), and the characteristic cramping pain of intermittent claudication is felt in the phantom calf after walking a short distance (Franke & Gall, 1981). Appenzeller and Bicknell (1969) report a patient with tabes dorsalis who, following amputation of his leg,

continued to suffer lightening-like pains in the phantom which coincided with the pains in his remaining intact leg.

Painful and non-painful phantom sensations can be exacerbated by ill health and fever (Bonica, 1953; Solonen, 1962; Sliosberg, 1948; Sunderland, 1978), a duodenal ulcer (Leriche, 1947c), the recurrence of cancer (Sugarbaker, Weiss, Davidson, & Roth, 1984), an acute herpes zoster infection (Wilson, Person, Su, & Wang, 1978), and palpation of an inflamed prostate (Maloney & Darling, 1966). The typical march of Jacksonian epilepsy which begins in the fingers and moves up the limb persists in the phantom after amputation of the arm (Jefferson, 1935). Pre-amputation lesions and pains are frequently referred to the phantom limb and experienced as if the limb had never been removed (Danke, 1981; Jensen et al. 1985; Katz & Melzack, 1988; Leriche, 1947a, 1947b; L'Hermitte & Susic, 1938; Nathan, 1956, 1962, 1985). Drug-induced tremor and dyskinesia of the phantom limb have also been reported (Janovic & Glass, 1985; McCalley-Whitters & Nasrallah, 1983). Amputees with Parkinson's disease report tremors of the phantom hand (cited in Melzack, 1989a). The administration of a diuretic to a woman whose bladder had been removed reliably produced a "severe intense urge to void" (Arcadi, 1977). Going through the motion of scratching the point in space corresponding to the site of severe itching of a phantom foot produced relief which could not be obtained by any other method (Jacome, 1978). Perspiration of the stump of an upper extremity amputee is felt as droplets of sweat trickling down the phantom hand (Pitres, 1897). A breeze directed at the stump is felt as a breeze on the phantom foot (James, 1887). Walking through puddles on an artificial limb produces a wet feeling on the phantom foot (James, 1887; Pitres, 1897).

This brief review is not exhaustive, but it is representative of the hundreds of reports in the literature illustrating the kinds of experiences that arise following the removal, or deafferentation, of an internal organ or body part. These range from the perception of simple paresthesias referred to diffuse regions of the phantom limb after a stimulus is applied at the stump to more complex perceptions which, in many respects, appear no different from

experiences which arise from an intact limb or organ. However, the phantom does not simply mirror the form of the absent limb once it has been cut off. If the stump is capable of transmitting information from the peripheral fibers, there are (temporarily reversible) long-term, use-related changes in the perceived form (i.e., shape, length, and volume) of the phantom limb which appear to correlate with modifications in the organization of central neural structures subserving the phantom limb (Kallio, 1948, 1950a; Katz 1989a).

Theories of Phantom Limb Phenomena

Three classes of theory have been proposed to account for the phantom limb but the evidence indicates that each fails on different grounds (Melzack, 1971; 1989a; Melzack & Wall, 1988). Arguments have been presented for a *peripheral* origin of the phantom limb which include the increased firing rate of primary afferents located in stump neuromas, stump ischemia, adhesions, and the formation of bone spurs (see Jensen & Rasmussen, 1989; Sunderland, 1978). However, since phantom limb pain is not always relieved by procedures which are designed to remedy these conditions (Melzack, 1971; Livingston, 1943; Nyström & Hagbarth, 1981), the cause of pain has been sought in the central nervous system.

Central explanations hypothesize that the output of hyperactive cells in the spinal cord (Carlen et al., 1978, Livingston, 1943; Wall, 1981), somatosensory projection pathways (Melzack, 1971; Melzack & Loeser, 1978), or somatosensory cortex (Head & Holmes, 1911; Simmel, 1958; 1962) are sufficient to explain the phantom limb. However, here too, the evidence indicates that surgical procedures, which either block or destroy the structures in which the phantom limb pain is hypothesized to reside, fail to alter the pain or the phantom, or do so only temporarily (Melzack, 1971; Melzack & Loeser, 1978; White & Sweet, 1969).

Finally, there have been serious attempts to explain phantom limb phenomena by general *psychological* and *emotional* processes, and in particular, as the result of denial and repression (Kolb, 1954; Parkes, 1973, 1976; Szasz, 1975). This latter class also frequently has been used by proponents of the peripheral and central views to discount phantom limb

pains which could not be explained by the current physiological and anatomical knowledge. This practice of relegating certain inexplicable phenomena to the realm of the psychological or emotional may free the theorist from considering them further, but it implicitly blames the amputee for his or her phantom limb pain.

Part of the controversy over the origin of phantom limb phenomena has arisen from the attempt to find a single explanatory mechanism (Melzack & Wall, 1988). Theories have tended to focus on only one type of phantom experience and ignore or discount the rest. The complexity and diversity of these, and other, phenomena have led Melzack (1989a) to conclude that the phantom limb cannot be explained by a unitary mechanism--whether peripheral, central or psychological. It is clear from the reports and studies of amputees that the qualities of experience reported as phantom phenomenon are no different from those of any other experience, and that nothing short of an integrated theory of human perception, learning, motivation and behaviour can account adequately for these experiences.

Melzack (1989a) has recently proposed that the experience of phantom limb phenomena (among other experiences) involves *cyclical processing and synthesis* of neural impulses through functional neural networks (the neuromatrix) which are the neural substrate of all the various aspects of the body-self. These networks are genetically built-in and are modified as a consequence of repeated experiences so that their characteristic patterns (neurosignatures) can be generated autonomously in the absence of input. No single brain region or unitary mechanism is responsible for the experience of the phantom limb. Rather, the simultaneous outputs of the networks in widespread regions of the brain combine to produce the various qualities of experience.

Two of the four conclusions that Melzack (1989a) proposes have direct bearing on the research presented in this thesis. The first conclusion is that "*The experience of a phantom limb has the quality of reality because it is produced by the same brain processes that underlie the experience of the body when it is intact*" (p. 4). The second conclusion is that "*Neural networks in the brain generate all the qualities of experience that are felt to originate in the*

body; inputs from the body may trigger or modulate the output of the networks but are not essential for any of the qualities of experience" (p. 4).

In the remainder of the Introduction I will review the evidence for (a) psychological and emotional contributions to phantom limb pain, (b) autonomic nervous system inputs as triggers for painful and non-painful phantom sensations, and (c) the involvement of more central influences suggesting that noxious somatosensory inputs at the time of, or shortly before, deafferentation influences the subsequent experience of pain.

Psychological and Emotional Factors

It comes as no surprise to find that amputees suffering phantom limb pain exhibit higher than normal levels of psychological and emotional distress. Depression (Caplan and Hackett, 1966; Lindesay, 1985; Sherman, Sherman & Bruno, 1987; Shukla et al., 1982a), anxiety (Parkes, 1973; Shukla et al., 1982a) and other forms of psychopathology are common (Dawson & Arnold, 1981; Ewalt et al., 1947; Lindesay, 1986; Morgenstern, 1970; Parkes, 1973; Shukla et al., 1982a; Steigerwald, Brass, Krainick, 1981). The obvious question is whether the phantom limb pain is a *cause* or *consequence* of the psychopathology. Arguments have been advanced in favour of both hypotheses but here I will only deal with the putative role of emotional and psychological disturbance as a cause of phantom limb pain. At the outset, it should be noted that the definitive prospective study has yet to be conducted. Such a study would have to obtain pre-operative measures of psychological and emotional functioning sufficiently prior to amputation so as to avoid the confounding effects of pre-amputation pain and hospitalization.

The explanation of phantom limb pain in terms of psychological and emotional processes is an offshoot of the more general question of the psychogenesis of chronic pain syndromes. This is a complex issue with a long history and implications which are beyond the scope of the present review. It will be touched on only briefly here. One of the major problems is in finding common ground for the two main perspectives on pain; the biological,

which makes use of neurophysiological concepts tied to anatomical structures, and the psychological, which deals with abstract constructs at a different level of analysis. However, it remains doubtful whether a translation of concepts from one system of thought to the other will prove entirely fruitful, since this would not address the issue of responsibility which seems to lie at the heart of the difference between the approaches.

Traditionally, when a pain problem has defied explanation based on the current state of anatomical and physiological knowledge of the nervous system, the tendency has been to deal with it by relegating it to the psychological realm (e.g., Bailey and Moersch, 1948; Henderson and Smyth, 1948). The implication of this shift in the level of analysis from neurophysiological to psychological is that the mechanisms proposed to underlie and give rise to the pain are somehow different depending on the hypothesized origin. Usually this difference also extends to how the patient is viewed and treated[1].

Psychoanalytic theory has been the dominant force in generating explanations on the psychogenesis of pain (Engel 1959; Kolb, 1954; Schilder, 1950; Szasz, 1975). Psychodynamic theorists (e.g., Engel, 1959; Stengel, 1965; Szasz, 1975) rely on the concepts of unconscious drives and needs to explain the persistence of pain in the absence of objective evidence of physical pathology, and thus the presence of chronic pain in certain patients is proposed to be intimately bound up with psychopathology and emotional disturbance. One can discern two separate but related attempts to explain painful and non-painful phantom limb sensations according to psychodynamic principles. Both rely heavily on the defense mechanisms of denial and repression to explain the psychogenesis of phantom limb pain but the first is a more explicit account.

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[1]Bailey and Moersch (1948) provide an example of the shift in theoretical orientation and level of analysis from biological to psychological in their discussion of the mechanisms of phantom limb pain when confronted with the otherwise inexplicable phenomenon of a patient whose phantom included the sensation of a wood sliver which had been under the nail of his index finger at the time of amputation. In the course of their discussion, they equate "cerebral" with "psychical" and then "psychical" with "obsession neurosis" without offering an explanation for their shift in orientation and leap in logic.

The explicit view explains the normal phantom as the result of a grief reaction over the amputated part. It is a "wish-fulfilling hallucination resulting from the denial of a lost part" (Frazier & Kolb, 1970, p. 492). The painful phantom results from the "denial of the affect associated with the loss" (Frazier & Kolb, 1970, p. 492). According to this view both painful and non-painful phantom sensations are viewed as pathological psychological responses to amputation.

The less explicit view of the psychogenesis of phantom phenomena has been proposed by Parkes in his extensive work on the psychological and emotional consequences of amputation (Parkes, 1972, 1973, 1975, 1976; Parkes & Napier, 1970; 1975). In particular, Parkes (1973) examined the emotional reactions and personality characteristics of amputees immediately following the loss of a limb, and one year later, after they had returned home and attempted to resume their normal lifestyles. Amputees with phantom limb pain were found to be psychologically "rigid" and "compulsively self-reliant" but the questionnaires and scales used to measure these characteristics lack construct and predictive validity. Although it is implied that these personality characteristics are causally related to the presence of phantom limb pain, Parkes provides no explanation for how the pain develops. Denial and repression are invoked as the mechanisms responsible for producing phantom limb pain but Parkes is not specific about what is being denied and repressed. Furthermore, no data are given to support these claims.

Simmel (1959) and Weinstein (1962) have provided convincing evidence and arguments against the role of denial in the genesis of all but a minority of painful and non-painful phantom limbs. Apparently healthy individuals who adjusted to the amputation continue to report the presence of a phantom years after the amputation. Phantoms which result from deafferenting lesions are no different from amputation phantoms yet in the former case only the function has been lost since the real limb is still present. One would not expect denial of the loss of function to produce a phantom defined by paresthesias. Phantoms do not occur if the process of sensory loss is gradual, as in leprosy (Simmel, 1956b; but see Price,

1976a), yet there should be as great a need for denial in these cases. Phantoms are often reported to be completely telescoped, shortened, shrunken, or otherwise misshapen, but denial and repression fail to account for these short- and long-term changes in the length, volume, and shape of the phantom limb.

Caplan and Hackett (1963) conducted in-depth interviews with elderly patients who had recently undergone an amputation. They could find no evidence for denial of affect and suggest that the tendency to deny is less frequent when the disability or illness is accompanied by visual signs of the infirmity such as an amputation. In general, the consensus is that denial, whether of the loss of the limb or of the affect associated with it, does not account for the presence of painful or non-painful phantoms in the vast majority of cases. This does not imply that denial of the affect, illness, or the future implications of the loss plays no part in the overall adaptation to the amputation (Rosen, 1950), including the experience of phantom limb pain. Some patients deny these realities by spending their waking hours absorbed by the phantom or stump pain with which they are plagued.

In summary, patients with phantom limb pain are frequently emotionally and psychologically distraught but the nature of the link between the pain and psychopathology remains an empirical question. Psychodynamic theory has been the dominant force in generating hypotheses concerning psychogenic pain with denial and repression playing prominent roles in phantom limb pain. Among the psychological characteristics proposed to produce phantom limb pain is the presence of a "rigid" and "compulsively self-reliant" personality (Parkes, 1973). However, there are serious questions concerning the validity and reliability of these results. One purpose of Studies 2 and 4 is to examine the psychological and emotional characteristics of amputees with and without phantom limb pain. In particular, the question of psychological "rigidity" among amputees will be assessed.

Autonomic Nervous System Influences

The autonomic nervous system (ANS) has often been implicated in the production and maintenance of painful and non-painful phantom limbs (de Jong & Cullen, 1963; Doupe, Cullen, & Chance, 1944; Kristen, Lukeschitsch, Plattner, Sigmund, & Resch, 1984; Lawrence, 1980; Leriche, 1939, 1942, 1947c; Livingston, 1938, 1943; Melzack, 1971, 1974; Morgenstern, 1970; Nathan, 1947; Sherman, 1984; Sherman & Bruno, 1987; Sunderland, 1978; Wall, 1984). The exact role it plays is still unknown but in many cases the evidence for ANS involvement is clear.

Painful and non-painful phantom limb sensations are frequently triggered by autonomic functions such as urination (Appenzeller & Bicknell, 1969; Carlen et al., 1978; Dougherty, 1980; Friedmann, 1978; Henderson & Smyth, 1948; Jensen et al., 1983, 1984; Maloney & Darling, 1966; Mitchell, 1872; Noordenbos, 1959; Sliosberg, 1948; Solonen, 1962; Steinbach, Nadvorna, & Arazi, 1982; Sugarbaker et al., 1984; Sunderland, 1978), defecation (Appenzeller & Bicknell, 1969; Friedmann, 1978; Henderson & Smyth, 1948; Noordenbos, 1959; Sliosberg, 1948; Steinbach et al., 1982; Sunderland, 1978), and ejaculation or orgasm (Appenzeller & Bicknell, 1969; Friedmann, 1978; Noordenbos, 1959; Riding, 1976; Sliosberg, 1948; Steinbach et al., 1982; Sugarbaker et al., 1984). These, and other autonomic nervous system functions may, via visceral afferent fibres, provide inputs that converge and summate at higher CNS levels to produce sensations and pain referred to the phantom. Furthermore, emotion-laden discussions and topics, anxiety and stress, fright, and startle have also been found to increase painful and non-painful phantom limb sensations (Carlen et al., 1978; Doupe et al., 1944; Henderson & Smyth, 1948; James, 1973; Jensen et al., 1983, 1984; Krebs et al., 1984, 1985; Livingston, 1943; Parkes, 1973; Pilowsky & Kaufman, 1965; Pitres, 1897; Riddoch, 1937; Sherman & Sherman, 1985; Sherman et al., 1984; Sliosberg, 1949; Solonen, 1962; Steinbach et al., 1982; Sunderland, 1978; Varma et al., 1972).

Nathan (1980, 1983, 1988) has reviewed the evidence implicating the sympathetic nervous system in certain chronic pain states. There are two reasons to suppose that the sympathetic nervous system is involved in pain. The first is that chronic pain patients may show signs of abnormal peripheral sympathetic activity in the affected region. The second is that interventions aimed at blocking the sympathetic supply frequently produce relief of pain, especially if hyperpathia and allodynia are present (Loh & Nathan, 1978; Loh, Nathan, Schott, 1981; Loh, Nathan, Schott, & Wilson, 1980; Nathan, 1980).

Symapthetically-maintained pain states are hypothesized to come about by an escalating cycle of sympathetic-efferent and somatic-afferent activity (Blumberg & Jänig, 1981, 1983; 1985; Devor, 1983; Roberts, 1986; Roberts & Fogelsong, 1988a, 1988b; Wall, Scadding, & Tomkiewicz, 1979). Direct evidence that sympathetic activity produces increases in primary afferent discharge is available from studies using animal (Barasi & Lynn, 1983; Blumberg & Jänig, 1981; Devor & Jänig, 1981; Korenman & Devor, 1981; Pierce & Roberts, 1981; Roberts, Elardo, & King, 1985) and human (Hallin & Wiesenfeld-Hallin, 1983) subjects. Other evidence from animals indicates that regenerating sprouts which have formed a neuroma are abnormally sensitive to the effects of noradrenaline (Korenman & Devor, 1981; Wall & Gutnick, 1974), acetylcholine (Diamond, 1959; but see Korenman & Devor, 1981), and circulating levels of plasma adrenaline (Blumberg & Jänig, 1981; Devor, 1983; Devor & Jänig, 1981; Korenman & Devor, 1981; Wall & Gutnick, 1974; Scadding, 1981). The onset and severity of autotomy following peripheral nerve section in mice and rats is significantly reduced by injections of guanethidine, a drug which depletes the peripheral terminals of sympathetic fibres of noradrenaline and prevents further release (Coderre, Abbott, & Melzack, 1984; Wall, Scadding, & Tomkiewicz, 1979). In addition, autotomy in mice is prevented by β-blockers, and with prolonged blockade primary afferent discharge is reduced (Scadding, 1981).

Although Nathan (1988) does not specifically address the problem of phantom limb pain, it is apparent that the sympathetic nervous system is involved in many cases. Excessive

vasoconstriction and sweating at the stump and surrounding regions suggest that regional sympathetic hyperactivity may contribute to the development of phantom limb pain (Leriche, 1949; Livingston, 1938; 1943). Hyperpathia and allodynia may not always be present, especially if the pain is restricted to the phantom limb and has not spread to the stump (e.g., Doupe et al., 1944). However, the characteristic superficial burning pain and deep aching may provide another clue that the sympathetic nervous system is involved (Hannington-Kiff, 1989).

Sympathetic blocks (Blankenbaker, 1977; Leriche, 1942; Livingston, 1938; 1943) and sympathectomies (Kallio, 1950b cited in Sherman, 1980) have been found to be at least temporarily effective in alleviating phantom limb and stump pain. Relief from phantom limb pain has also been reported with propranalol, a beta adrenergic blocking agent (Ahmad, 1979, 1984; Marsland, Weekes, Atkinson, & Leong, 1982; Ollie, 1970; but see Scadding, Wall, Wynn Parry, & Brooks, 1982). Electrical stimulation of the sympathetic chain in an amputee produced pain referred to the phantom limb but in non-amputees the pain is referred to the abdomen (Noordenbos, 1959).

Two hypotheses have been advanced to account for the pain and sympathetic abnormalities that arise following peripheral nerve lesions. The first hypothesis is that the normal reflex pattern of discharge in postganglionic cutaneous vasoconstrictor fibres is altered after a peripheral nerve lesion (Blumberg & Jänig, 1981, 1983, 1985). Many of these fibers begin to discharge in a manner similar to muscle vasoconstrictor fibres which are tied to cardiac rhythmicity. This is hypothesized to lead to a disruption of the thermoregulatory function of the postganglionic cutaneous vasoconstrictor neurons resulting in trophic changes and pain.

The second hypothesis is that sympathetically-maintained pains arise from sympathetic activation of low-threshold mechanoreceptors which terminate on wide-dynamic-range spinal neurons (Roberts, 1986; Roberts & Fogelsong, 1988a, 1988b). In normal tissue such activity does not lead to pain, but in tissue which has previously sustained damage, spinal cord cells

become sensitized and subsequent mechanoreceptor activity, which normally is non-noxious, is perceived as pain. This hypothesis does not require dystrophic and vascular changes to be present and may explain phantom limb pain which persists, despite completely healed tissue, and in the absence of associated stump pain (e.g., see Doupe et al., 1944).

Together, these two hypotheses can account for a number of phantom limb phenomena. Microelectrode recordings of postganglionic sympathetic fibres from nerve fascicles in awake humans reveals a characteristic pattern of discharge (Bini, Hagbarth, Hynninen, & Wallin, 1980a, 1980b; Hallin & Wiesenfeld-Hallin, 1983). Bursts of sympathetic activity in vasomotor and sudomotor fibres are reliably followed by electrodermal responses and plethysmographic signs of vasoconstriction which can be modified by a variety of internal (e.g., deep breathing, Valsalva maneuver, mental arithmetic, distraction) as well as external (e.g, electric shocks, loud noise) stimuli (Delius, Hagbarth, Hongell, & Wallin, 1972; Hagbarth, Hallin, Hongell, Torebjörk, & Wallin, 1972; Hallin & Torebjörk, 1974; Jänig, 1988; Wallin, 1988; Wallin & Fagius, 1988).

Bursts of activity in sympathetic fibres located in the stump may be followed by increases in primary afferent discharge which are referred to the phantom limb as transient increases in non-painful paresthesias. In some cases, wide-dynamic-range neurons in the dorsal horns may become sensitized due to the trauma associated with amputation. In these cases the effect of noradrenaline released from peripheral sympathetic fibers onto primary afferents located in the stump would produce dysesthesia and pain instead of the usual non-painful paresthesias (Nathan, 1988; Roberts, 1986; Roberts & Fogelsong, 1988a, 1988b). Finally, the functioning of vasocontrictor and sudomotor fibres becomes abnormal after peripheral nerve transections. These changes may help to explain the vascular and trophic abnormalities found at the stump and surrounding tissues in some amputees (Blumberg & Jänig, 1981, 1983, 1985).

Surprisingly few studies have examined peripheral manifestations of sympathetic nervous system functioning at the stump in human amputees (Livingston, 1943; Kristen et al.,

1984; Sherman, 1984; Sherman & Bruno, 1987; Sliosberg, 1948). Sliosberg examined 141 amputees and found the stump to be cooler than the intact limb in 94 of them but did not relate the temperature difference to the presence or absence of phantom limb pain. Kristen et al. assessed phantom and stump pains using thermography and found that a "patchy asymmetrical temperature" distribution of the stump thermograms was significantly more frequent among stump pain sufferers than in patients who were free from stump pain. However, when examined for the presence of phantom limb pain, thermographic records taken of the stump were no different for patients with and without phantom limb pain.

In contrast, Sherman recently observed a negative correlation between temperature at the stump and the presence of burning phantom limb and stump pain, implying that reduced blood flow to the stump is responsible for increased levels of phantom limb pain (Sherman, 1984; Sherman & Bruno, 1987). Furthermore, repeated measurements of the same patients on different occasions revealed that lower temperatures at the stump relative to the intact limb were associated with greater intensities of phantom and stump pain. Unfortunately, in the majority of cases this relationship was confounded by co-existing stump pain, and so it is not possible to unambiguously attribute the phantom pain to altered blood flow at the stump. Since stump pain was a significant problem for most patients it would not be surprising to find that the blood flow was reduced at the stump when compared to the intact limb (e.g., Kristen et al., 1984). In order to make the claim that phantom limb pain is associated with abnormal sympathetic nervous system activity at the stump, it is necessary to compare the two limbs in patients suffering from phantom limb pain but not stump pain.

In summary, the sympathetic nervous system may play a role in the expression of painful and non-painful phantom sensations according to mechanisms proposed by two hypotheses. The first accounts for phantom limb sensations and pain by a neural mechanism. Normal post-ganglionic sympathetic efferent activity in sudomotor and vasomotor fibers located in the stump result in a phasic pattern of neurotransmitter release. This release increases the rate of firing in adjacent primary afferents projecting onto spinal cord cells

subserving the portion of the limb which is missing. This alteration in activity of spinal cord cells could be referred to the phantom limb and perceived as increases and decreases in the intensity of painful or non-painful paresthesias.

The second hypothesis explains sympathetically-maintained phantom limb pain by a vascular mechanism. Alterations in the thermoregulatory pattern of cutaneous sympathetic fibers following amputation may lead to abnormalities in the vascular and trophic functioning of stump tissues producing ischemia and eventually, pain. Studies 2 and 3 will compare the peripheral manifestations of sympathetic nervous system activity at the stump and intact limb in amputees with phantom limb pain, non-painful phantom limb sensations, and no phantom limb at all. In addition, these studies will examine the relationship between the intensity of phantom limb sensations (both painful and painless) and sympathetic nervous system activity at the stump.

Evidence of "Pain Traces" in the Central Nervous System

Perhaps the most convincing evidence that phantom limb pain involves a central component comes from amputees who report that the pain they experienced in their limb before amputation persists in the phantom after removal of the painful extremity. This type of phantom limb pain, characterized by the persistence or recurrence of a previous pain, is experienced with the same qualities and in the same location of the phantom limb as the pre-amputation pain. These *somatosensory memories* are frequently felt so vividly that the amputee finds it difficult to believe that the limb has been removed. In this section I will review evidence suggesting that the effects of injury sustained to a limb prior to its deafferentation may be retained in central nervous system structures and influence the subsequent development of pain. This evidence is part of a growing body of literature pointing to the existence of memory-like mechanisms for noxious somatosensory stimulation and argues strongly in favour of pre- and post-operative regional anesthesia as an effective

treatment for the management of pain in the surgical candidate (Bach, Noreng, & Tjéllden, 1988; Cousins, 1989; McQuay, Carroll, & Moore, 1988; Wall, 1988).

First, I will review the evidence from animal studies showing that noxious stimulation applied shortly before deafferentation leads to an increase in behaviours indicative of pain. Then, I will briefly suggest that somatosensory memories reflect the joint occurrence of two forms of memory corresponding to separate *somatosensory* and *cognitive* components. Finally, I will present evidence from case histories and larger group studies mainly of amputees who report phantom limb pain that resembles the pain experienced in the limb before amputation.

Animal studies

Peripheral neurectomy or dorsal rhizotomy in animals is followed by a form of self-mutilation of the deafferented limb, termed *autotomy*, in which the animal chews and scratches the distal portions of the insensitive foot or paw to the point of amputation (Wall, Devor, Inbal, Scadding, Schonfeld, Seltzer, & Tomkiewicz, 1979; Wall, Scadding, & Tomkiewicz, 1979). It is now well established that this form of self-mutilation is a response to painful or dysesthetic sensations referred to the denervated limb and represents a valid model of the deafferentation pain seen in humans (Coderre, Grimes and Melzack, 1986a).

Dennis and Melzack (1979) found that a painful, formalin-induced irritation of the rat forepaw one hour prior to cutting the dorsal roots led to an accelerated onset of autotomy and a shift in the site of chewing as compared to untreated control animals. In formalin-treated animals, post-rhizotomy chewing occurred earlier, and was confined to the more distal regions of the paw close to site of the formalin injection. In untreated controls, chewing was predominantly limited to proximal regions of the paw. This study provided the first experimental evidence in animals demonstrating that a prior injury can influence the course and pattern of the pain induced by posterior rhizotomy and pointed to the presence of a "pain memory" specific to the location of the formalin-induced irritation.

More recently, the results of a series of studies conducted by Coderre and his colleagues have confirmed that a brief noxious input prior to denervation is capable of producing long-term central changes (Coderre & Melzack, 1985, 1986, 1987; Coderre et al., 1986a, 1986b). These studies demonstrate that denervation-induced autotomy in rats is significantly enhanced (both in onset and severity) following a prior heat injury (Coderre & Melzack, 1985, 1986, 1987) or formalin injection (Coderre et al., 1986b). The referred hyperalgesia induced by a prior heat injury appears to depend upon increased spinal cord activity since autotomy in pre-injured animals was reduced by spinal anesthesia or a combination of C-fibre and sympathetic blockade. The results of these studies also raise the possibility that autotomy after nerve transection arises, in part, as a consequence of the injury barrage produced by the transection of peripheral nerves. The effect of this barrage may be to set up a "pain memory" in central neural structures.

Two forms of memory

Researchers working in the area of memory recently have presented convincing evidence for the existence of two types of memory systems. They are specialized for processing different kinds of information and lead to the formation of separate, dissociable neural representations which correspond to unique aspects of a common environmental event (Mishkin & Appenzeller, 1987; Mishkin, Malamut, & Bachevalier, 1984; O'Keefe & Nadel, 1978; Squire, 1982, 1987). One type of memory is specialized for the acquisition and storage of perceptual, motor, and cognitive skills (e.g., "habits") and thus is presumed to process information that is usually not accessible to conscious awareness. The second type of memory is specialized for processing information concerning the time, place, and content of an event, and thus consists of knowledge which can be declared (e.g., cognitions). Despite differences in terminology, level of analysis, and theoretical orientation, there is a general consensus that the subdivision of memory into separate systems captures the inherent organizational structure and function of the human nervous system (Mishkin et al., 1984; Squire, 1987).

Pain that persists or recurs in the phantom limb after amputation also involves two separate, dissociable forms of memory (Katz, 1989b). One component of the unified experience is somatosensory. It is referred to the phantom limb in the same location and with the same qualities as the pre-amputation pain. The other memory component is cognitive. It contains declarative information about general and specific aspects of the pre-amputation pain, including its location, frequency and duration, how and when it started, factors which relieved and aggravated it, etc. It is this declarative information which enables the amputee to recognize the somatosensory component. Elsewhere I have provided arguments suggesting that under the appropriate conditions these two forms of memory may be dissociated (Katz, 1989b). A brief elaboration of the two forms of memory is presented below.

Somatosensory memory component. This is an anatomical representation of the somatosensory qualities of the pre-amputation pain. Its presence is signalled by the recurrence or persistence of pain referred to the phantom limb, and is defined in terms of the location in the body, quality of the sensation (e.g., burning, shooting, etc.), and intensity. It consists of the activation of the same, or a very similar, set of neural assemblies which coded the somatosensory event as it occurred prior to the amputation. It is a higher-order functional unit that corresponds to the temporal and spatial patterning of nerve impulses produced by the activation of receptors in the region of the limb where the original sensation occurred. The particular information that is generated by the activation of the somatosensory component is a coded pattern of nerve impulses that specifies the body part, quality and intensity of the somatosensory event.

Cognitive memory component. This component contains information related to when and in what places the pre-amputation sensation was felt as well as information about the body part, quality, and intensity of the experience. This information gives each somatosensory experience its unique and personal meaning and provides the basis of the identifying label and response (e.g. "my pain", a corn, diabetic ulcer, finger ring, etc.). The identification of a

phantom sensation as having been felt before is a form of recognition memory based on a judgement of familiarity and a sense of the past. One must know, or have access to knowledge about, what one has (and therefore has not) previously experienced in order to state whether two experiences separated in time are the same or different.

Clinical studies and case reports of somatosensory memories[2]

Clinical studies and case reports of phantom limb pain which resembles the pain experienced prior to amputation can be divided into two classes based on whether the information provided is predominantly qualitative or quantitative. Case reports are generally rich in qualitative and descriptive details, but provide little information concerning the frequency with which somatosensory memories occur after amputation. On the other hand, studies involving larger numbers of amputees occasionally provide these statistics but with few descriptions, so that it is not known whether somatosensory memories are more likely to occur with some kinds of pains than with others.

Table 1 contains a list of case and larger group studies which report somatosensory memories. Included is a description of the type of somatosensory memory reported, and whether the original experience was present at (or near) the time of deafferentation (if applicable) or whether it had occurred before. It can be seen that somatosensory memories occur after a variety of conditions, although most are associated with some form of loss of afferent input such as amputation.

The table shows the many types of pain that recur following amputation. Case studies of amputees include somatosensory memories of cutaneous lesions such as painful diabetic and decubitus ulcers (Bors, 1951; Danke, 1981; Katz & Melzack, 1987; Kolb, 1954; Leriche,

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[2]The term "somatosensory memory" will be used to refer to the *experience* of a phantom pain which resembles a pain that existed at some time before the amputation. This usage corresponds to the joint activation of the somatosensory and cognitive memory components. Use of the separate terms "somatosensory memory component" and "cognitive memory component" will be used only when it is necessary to distinguish between the two forms of memory.

Table 1. Summary of case reports and group studies in which somatosensory memories (SMs) have been reported, including the number of cases, a brief description of the type and location of the original experience which later recurred as a SM, whether the original experience occurred at/near or before the time of deafferentation (if applicable), and the condition of occurrence of the SM.

Author(s) and number of cases of SMs reported	Description of type and location of original experience and subsequent somatosensory memory	Original experience at/near or before time of deafferentation	Condition of occurrence of SM
Mitchell (1872) (n = 2)	- following gunshot wound "the thumb turned into the palm, and continued in this state of spasm, so that when the limb was removed six hours later, the nail of the thumb had cut into the palm" (p.355). Nine years later, the phantom thumb remained in this state of spasm, cutting into the palm.	at/near	amputation
	- sensation of phantom hand and fingers in rigid extension just as they had been for the 2 weeks prior to amputation.	at/near	amputation
James (1887) (n = 3)	- one case each of a blister on the heel, and chilblains on the toes, present at the time of accident, and later felt in the phantom after amputation	at/near	amputation
	- sensation of toenails of phantom foot needing cutting	not reported	amputation
Jackson (1889) (n = 2)	- hand blown off when a cylinder containing explosives discharged The phantom hand is in the same posture as if encircling a vessel	at/near	amputation
Charcot (1892) (n = 1)	- sensation of a tight engagement ring on phantom finger	at/near	amputation
Pitres (1897) (n = 1)	- pain and sensation of a corn on the dorsal surface of the small toe present at the time of amputation continued to cause the patient distress after the limb had been removed	at/near	amputation
Kogerer (1930) (n = 1)	- feels pain of the operation wounds on phantom middle finger	at/near	amputation
Van Bogaert (1934) (n = 2)	- one patient reported his phantom foot felt like a " wooden sole", his big toe "like a large thorn". These were the same descriptions he used prior to the amputation when his foot and toe were painful.	not reported	amputation
	- for 5 days following spinal anesthesia the patient reported his legs to be in the same position as they had been in during the operation	at/near	spinal anesthesia

Table 1 continued

Author(s) and number of cases of SMs reported	Description of type and location of original experience and subsequent somatosensory memory	Original experience at/near or before time of deafferentation	Condition of occurrence of SM
L'Hermitte & Susic (1938) (n = 3)	- experienced pain in phantom foot of the crush injury which led to amputation	at/near	amputation
	- burning pain of gangrene experienced in the small toe prior to its amputation persisted in the phantom toe	at/near	amputation
	- pain in knee due to tumour persisted in phantom after amputation	indeterminate	amputation
Leriche (1939, 1947a, 1947b) (n = 5)	- sensations of patient's hand flattened against the wall, tearing of skin, and pain from wounds incurred in a car accident persisted in the phantom 3 years after amputation	at/near	amputation
	- painful tingling experienced in the hand at the time of a crush injury to the elbow 3 days before amputation of the hand was still present in phantom 20 years later	at/near	amputation
	- prior to amputation, the region surrounding the Achilles tendon had become ulcerated and painful, but had healed completely by the time of the operation several months later. Six years after amputation, and following an injection of lipiodol into the stump, the pain of the ulcerated Achilles tendon instantly re-appeared and never left.	before	amputation
	- a post-traumatic pain which had been relieved 2 years earlier by cervical sympathetic ramisection was re-experienced following an injection of a local anesthetic into the maxillary nerve for routine dental work.	before	cervical sympathetic ramisection
	- a pain of traumatic origin, located in the hand and fingers, which had been relieved 6 years earlier by stellectomy, was re-experienced in an overworked labourer following heavy use of a hammer.	before	stellectomy
Bailey & Moersch (1941) (n = 1)	- pain from a wood sliver which had been under the nail of the index finger for one week before the amputation of arm was still present in the phantom 2 years later	at/near	amputation

Table 1 continued

Author(s) and number of cases of SMs reported	Description of type and location of original experience and subsequent somatosensory memory	Original experience at/near or before time of deafferentation	Condition of occurrence of SM
Riddoch (1941) (n = 2)	- patient's right arm blown off by an explosion of the bomb he was holding; phantom hand continues to be in same posture	at/near	amputation
	- left arm amputated after 3 years of pain; removal of the arm failed to alter the posture or pain in the phantom hand and fingers	at/near	amputation
Minski (1943)	- wound in knee re-experienced after amputation	not reported	amputation
(n = 3)	- pain in big toe from gangrene re-experienced in phantom toe after amputation	not reported	amputation
	- pain from severe corneal ulceration persisted following enucleation of the eye	not reported	enucleation of eye
Herrmann & Gibbs (1945) (n = 1)	- pain in phantom toes similar to pre-amputation pain but less severe	at/near	amputation
Hutchins & Reynolds (1947) Reynolds & Hutchins (1948) (n = 14)	- tooth pain from dental work and extractions performed without anesthesia or under nitrous oxide was referred to treated (or site of extracted) teeth following stimulation of nasal mucosa one week later	at/near	dental work, tooth extraction
Browder & Gallagher (1948) (n = 15)	- 12 patients with severe pain before amputation indicated that the subsequent phantom limb pain bore a distinct resemblance to the pre-operative pain	at/near	amputation
	- was holding a blasting cap between his index finger and thumb when it accidently exploded. Three years later the phantom fingers remained in the same posture.	at/near	amputation
	- severed flexor tendon in ring finger resulting in a stiff finger; 7 years later amputation of the arm was followed by a phantom in which all fingers of the hand could be "moved"except the ring finger which was still stiff	at/near	amputation
	- after a severe burn, the patient's leg gradually became acutely flexed as a result of a cicatrix; the phantom leg retains the same pre-amputation posture	at/near	amputation

Table 1 continued

Author(s) and number of cases of SMs reported	Description of type and location of original experience and subsequent somatosensory memory	Original experience at/near or before time of deaiferentation	Condition of occurrence of SM
Henderson & Smyth (1948) (records lost)	- at least one case of each of the following reported in the phantom limb following amputation: discomfort of ingrown toenail, corn, compression of toes in a tight boot, the impression of a split finger nail, a painful whitlow, the sensation of a finger ring;	before	amputation
	- wound from combat injury, sprained ankle;	at/near	amputation
	- the pain of suppurative arthritis, the sensation of a traction pin, pain in relation to pressure points and splints, the sensation of lice crawling under a plaster	at/near	amputation
Sliosberg (1948) (n = 1)	- feels the sensation of the handle of the cane, which he was holding at the time of the accident, pressing against the palm of the phantom hand	at/near	amputation
Bornstein (1949) (n = 5)	- 3 patients reported the distinct sensation of a watch strap around the phantom wrist, one of whom felt the buckle especially clearly	not reported	amputation
	- sensation of ring on middle finger of phantom	not reported	amputation
	- fourth and fifth fingers developed contractures due to infection. After a below-elbow amputation, the phantom fingers were felt in the same stiff posture. When the phantom became painful the fingers would swell just as they had prior to the amputation.	not reported	amputation
Szasz (1949) (n = 2)	- pain of a long-standing peptic ulcer persisted after vagotomy, and after healing of the lesion as shown by roentgen examination.	not reported	vagotomy
	- same as above case but ulcer symptoms present intermittently for 45 years	not reported	vagotomy
Stevenson (1950) (n = 3)	- one case each of a painful knee, a bunion on the foot, and the pain of gangrene which had affected the middle 2 toes, persisted in the phantom after amputation	not reported	amputation
Bors (1951) (n = 1)	- sensation of a decubitus ulcer on the elbow which had healed by time of amputation was later experienced in the phantom. This patient also had sensation that fingernails needed clipping.	before	amputation

Table 1 continued

Author(s) and number of cases of SMs reported	Description of type and location of original experience and subsequent somatosensory memory	Original experience at/near or before time of deafferentation	Condition of occurrence of SM
Cronholm (1951) (n = 3)	- feeling of concrete under the nails which was present at the time of the accident persisted in the phantom	at/near	amputation
	- received a compound fracture of his leg in an accident followed, 4 months later, by amputation. The phantom sensations consisted of (a) a painful compression of the fractures and (b) the pieces of bone grating against each other	not reported	amputation
	- pain of shell wound incurred 9 months before amputation was re-experienced in the phantom	not reported	amputation
Gloyne (1954) (n = 1)	- ulcer pain persisted after subtotal gastrectomy and the removal of the lesion	not reported	gastrectomy and removal of ulcer
Kolb (1954)	- shooting pains referred to the site of previous decubitus ulcers	not reported	amputation
Noble et al. (1954) (n = 1)	- patient's phantom leg was "felt curved and mashed as it had been ... following the accident" (p. 611) which led to amputation	not reported	amputation
Wallgren (1954) (n = 1)	- sensation of gentle pressure from the oscillograph cuff which had been around the thigh at time of lumbar puncture persisted once the cuff had been withdrawn	at/near	spinal anesthesia
Bressler et al. (1955) (n = 1)	- pain in phantom breasts was the same as she had had prior to both mastectomies	not reported	amputation
Blood (1954) (n = 1)	- sensation of a "sore corn" on the small toe persisted in the phantom after the limb was removed	not reported	amputation
Haber (1956, 1958) (n = 4)	- 3 subjects felt the sensation of rings on their phantom fingers; in one the sensation disappeared 2 months after amputation	not reported	amputation
	- sensation of a watch around phantom wrist	not reported	amputation

Table 1 continued

Author(s) and number of cases of SMs reported	Description of type and location of original experience and subsequent somatosensory memory	Original experience at/near or before time of deafferentation	Condition of occurrence of SM
Engel (1959) (n = 4)	- bouts of severe pain in the right side of the patient's forehead were identical in location and character to the original pain experienced when a home-made bomb he was preparing exploded prematurely	not applicable	psychogenic
	- ear pain from past otitis continued to recur after initial inflammation	not applicable	psychogenic
	- throat pain from past peritonsillar abscess recurred periodically	not applicable	psychogenic
	- pain from past cystitis recurred on occasion	not applicable	psychogenic
Nathan (1962) (n = 4)	- pain in the left lower limb of a cut from an ice-hockey skate was re-experienced in the phantom leg 5 years after accident	before	amputation
	- pain in the right knee of a severe comminuted fracture of the patella, which had been excised six years earlier, was re-experienced after bilateral cordotomy	before	bilateral cordotomy
	- painful ingrowing toe nail present at the time of a spinal cord injury persisted in phantom	at/near	spinal cord injury
	- "phantom consisted of a foot in a boot with his toes squelching in the blood which filled his boot" p (133).	at/near	amputation
Frederiks (1963) (n = 2)	- phantom arm flexed and immobile, pressed upon chest just as it had been carried for months before amputation	at/near	amputation
	- phantom foot inclined to the left, as it had been before amputation	at/near	amputation
Cook & Browder (1965) (n = 1)	- after amputation of two fingers the patient reported pain of a "sticking, pins and needles" quality in the phantom fingers similar to the original pre-amputation pain	not reported	amputation
Farley & Smith (1968) (n = 34)	- sensations of fullness, feces, flatus, tightness and dragging in the phantom rectum following excision. In one patient the sensation of tightness was distressing.	not reported	excision of rectum
Appenzeller & Bicknell (1969) (n = 15)	- in 15 patients the phantom pain was similar to that experienced before amputation	at/near	amputation
White & Sweet (1969) (n = 1)	- pain in ulcerated areas of the foot persisted in the phantom after it had been amputated	at/near	amputation

Table 1 continued

Author(s) and number of cases of SMs reported	Description of type and location of original experience and subsequent somatosensory memory	Original experience at/near or before time of deafferentation	Condition of occurrence of SM
Dorpat (1971) (n = 3)	- 3 cases of menstrual cramps or labor pains of the same duration, intensity, and frequency as those occurring before total hysterectomy. One patient was a 70 year old who, on the second day after hysterectomy, remarked "I haven't had cramps like this for 35 years".	before	total hysterectomy
Varma et al. (1972) (n = 1)	- one patient reported feeling the sensation of a wristwatch and a ring on the phantom wrist and finger	not reported	amputation
Conomy (1973) (n = 1)	- incurred a spinal cord injury when thrown from his motorcycle. At the accident scene he felt as though he was still seated upright on his his motorcycle although he was actually lying on his back. This sensation persisted for 15 hours and recurred intermittently over the ensuing weeks.	at/near	spinal cord injury
Nielson et al. (1975) (n = 1)	- amputation below-the-knee was followed by a phantom sensation "similar in character and intensity to the one he had [had] when the foot was crushed" (p. 303).	not reported	amputation
Parkes (1976, 1973) (n = 7)	- about half the patients with moderate or severe phantom limb pain likened it to the pain which they had experienced before the amputation.	at/near	amputation
Friedmann (1978) (n = 11)	- one case each of painful chilblains on the phantom foot, phantom "tennis elbow", a finger ring and a wrist watch represented in the phantom; 4 cases of phantom shoe; and 3 cases of phantom bandages, all experienced after amputation	not reported	amputation
Henry & Montuschi (1978) (n = 3)	- cardiac pain referred to the dorsal region over T4, the site of an old compression fracture sustained in an accident 20 years earlier	not applicable	cardiac pain
	- cardiac pain referred to the left thoracic region at the site of a herpes zoster inflammation incurred 30 months before	not applicable	cardiac pain
	- referred cardiac pain radiating to both arms in the distribution of a previous disc lesion at C4-5 from a head injury sustained 7 years before	not applicable	cardiac pain

Table 1 continued

Author(s) and number of cases of SMs reported	Description of type and location of original experience and subsequent somatosensory memory	Original experience at/near or before time of deafferentation	Condition of occurrence of SM
Marbach (1978) (n = 1 or more)	- in at least one patient who received endodontic treatment or tooth extraction to eliminate pain, the same pain persisted, referred to "phantom tooth"	at/near	dental work, tooth extraction
Brena & Sammons (1979) (n = 1)	- discomforting sensation of "having a full bladder" and episodes of sharp, burning, pain following the removal of the bladder for chronic cystitis.	not reported	removal of urinary bladder
Sherman et al. (1979) (n = 6)	- 37.5 % of patients reported phantom limb pain of the same description and in the same location as the pre-amputation pain	not reported	amputation
Roth & Sugarbaker (1980) (n = 11)	- 17.5 % of patients reported phantom limb pain or other sensations which had occurred some time before the amputation	not reported	amputation
Berger & Gerstenbrand (1981) (n = 6)	- 6 cases of spinal cord injury in which the position of the phantom body and limbs corresponded to the patients' position at the time of spinal cord injury	at/near	spinal cord injury
Danke (1981) (n = 10)	- 2 cases of corns on the small toe years before amputation reported a re-experience of the sensations in the phantom	indeterminate	amputation
	- injury to knee 15 years before R-A/K amputation recurred as phantom pain	before	amputation
	- sensation of sprained ankle 13 years before L-A/K amputation recurred as phantom pain	before	amputation
	- injuries to calf and toe 4 years before R-A/K amputation re-experienced in the phantom	before	amputation
	- injury to the knee and a corn on the small toe 1 year before R-A/K amputation were both re-experienced in the phantom	indeterminate	amputation
	- sensation of a plaster cast on the leg and of a corn 5 months before R-A/K amputation were both re-experienced as phantom phenomena	before	amputation
	- cutaneous ulcers on the great toe and ankle 3 months before L-A/K amputation re-experienced in the phantom	indeterminate	amputation
	- sensation of a plaster cast on leg 2 weeks before R-A/K amputation recurred as phantom phenomenon	before	amputation

Table 1 continued

Author(s) and number of cases of SMs reported	Description of type and location of original experience and subsequent somatosensory memory	Original experience at/near or before time of deafferentation	Condition of occurrence of SM
Reisner (1981a, 1981b) (n = 3)	- two patients with brachial plexus injuries following motorcycle accidents reported a third (phantom) arm and hand which was in the same posture as the arm and hand had been at the time of the accident	at/near	brachial plexus injury
	- persistent tooth pain following tooth extraction	at/near	tooth extraction
Wall et al. (1985) (n = 2)	- in 12.5% of cases the location of their phantom pain was identical before and after amputation	at/near	amputation
Jensen et al. (1985) (n = 21)	- phantom limb pain was similar to the pre-amputation pain in location and character in 36% of patients 8 days after amputation, and in 10% of patients six months, and 2 years later	at/near	amputation
Katz & Melzack (1987) (n = 6)	- after a dorsal rhizotomy, the patient developed a painful phantom hand and arm."Within hours of recovery, [her] index, middle and ring fingers [were] cramped, little finger sore" Several years later despite amputation of the arm she continued to feel the same cramping, sore pain.	at/near	amputation
	- pain from stepping on a nail that "tore a chunk" from her heel 2 weeks before amputation persisted in phantom for several weeks	at/near	amputation
	- pain from diabetic ulcers on the medial and lateral surfaces of the ankle persisted in the phantom	at/near	amputation
	- burning in the toes from gangrene which began two months prior to amputation continues to cause pain in the phantom toes 7 years later	at/near	amputation
	- "pleasant sensation of draining" in her arm and hand when having the arm drained of excess fluid on numerous occasions prior to amputation recurred in the phantom	before	amputation
	- carries phantom arm bent at elbow with the hand pressed against the chest just as it had been carried during the final year prior to amputation	at/near	amputation

Table 1 continued

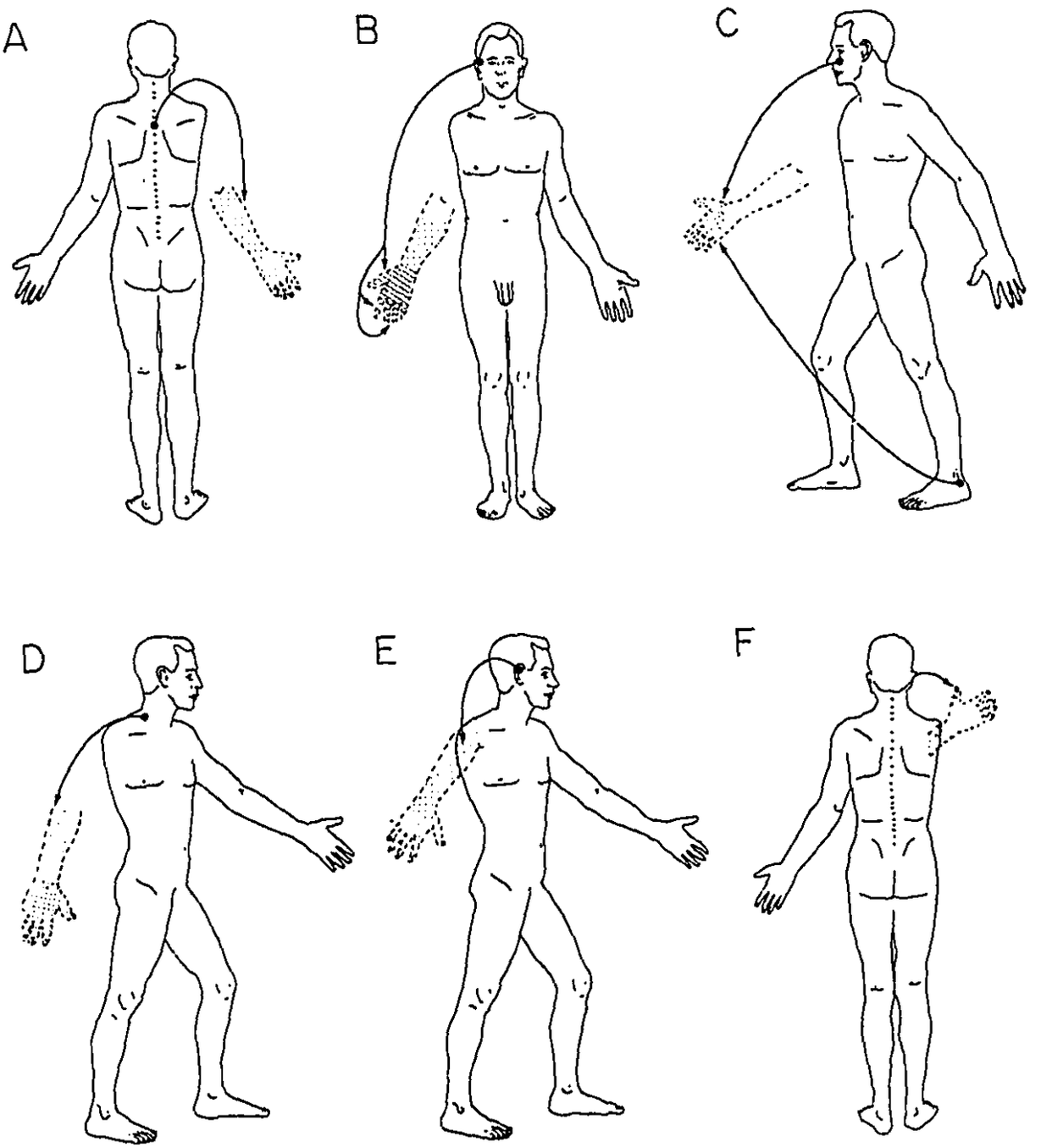
Author(s) and number of cases of SMs reported	Description of type and location of original experience and subsequent somatosensory memory	Original experience at/near or before time of deafferentation	Condition of occurrence of SM
Sacks (1987) (n = 1)	- discomfort of an ingrown toenail which had not been taken care of before amputation persisted in the phantom	at/near	amputation
Jensen & Rasmussen (1989) (n = 1)	- phantom arm and hand felt to be in the same position as at the time of a motorcycle accident with the hand as if clenching the handlebars despite total paralysis and sensory loss. The phantom hand persisted in the same posture after the limb was amputated 8 months later.	at/near	brachial plexus injury followed by amputation

1947a, 1947b; White & Sweet, 1969), gangrene (Katz & Melzack, 1987; L'Hermitte & Susic, 1938; Minski, 1943; Stevenson, 1950), corns (Blood, 1954; Danke, 1981; Henderson & Smyth, 1948; Pitres, 1897) blisters (James, 1887), and ingrown toenails (Henderson & Smyth, 1948; Sacks, 1987). Reports of deep tissue injuries (Nathan, 1956, 1962) and pain from damage to bony structures (Cronholm, 1951; Nathan, 1956, 1962) also are represented in the phantom. The phantom may assume the same painful posture as that of the real limb prior to amputation especially if the arm or leg had been immobilized for a long time.(Bornstein, 1949; Browder & Gallagher, 1948; Frederiks, 1963; Jackson, 1889; Katz & Melzack, 1987; Mitchell, 1872; Riddoch, 1941).

Although most of these somatosensory memories correspond to painful pre-amputation experiences, non-painful, "super-added" phantom sensations are also known to occur after amputation. Super-added phantom sensations include the experience of a finger ring (Bornstein, 1949; Charcot, 1892; Friedmann, 1978; Haber, 1956, 1958; Henderson & Smyth, 1948; Varma et al., 1972), wristwatch and strap (Bornstein, 1949; Friedmann, 1978; Haber, 1956, 1958; Varma et al., 1972), shoe (Friedmann, 1978; Henderson & Smyth, 1948), plaster cast (Danke, 1981; Solonen, 1962), and the handle of a cane which was being held when the hand was accidently amputated (Sliosberg, 1948). More complex phantom experiences include the sensation of blood-filled boots (Nathan, 1962) and blood trickling down the phantom limb (Bradley, 1955).

Despite such detailed descriptions, there is little information on the frequency with which the different types of pain and sensation occur in the general population of amputees. Global statistics occasionally are provided in studies of groups of amputees but descriptive details are scant. For example, Browder and Gallagher's (1948) figures indicate that 12 of 26 patients (46%) with severe pain before the extremity was removed reported that the subsequent phantom limb pain bore a distinct resemblance to the pre-operative pain but no qualitative information is provided. Similarly, Appenzeller and Bicknell (1969) found that phantom limb pain was similar to the pain experienced before amputation in 79% of patients.

Figure 1. Referred sensations reported by Case 57 during electrical stimulation. Dots represent points on the body where stimulation was applied. Arrows indicate where the sensations were referred. **A:** Stimulation applied at a point on the midline of the patient's upper back produced an increase in the paresthetic sensations that usually defined his phantom limb. **B:** During stimulation applied at a point on his right ear, the patient reported that the phantom hand became warm and the fingers began to move. **C:** Increased paresthesias in the phantom arm resulted from stimulation applied at points to either side of his nose and as far away as the left ankle. **D:** during stimulation to a point just above the clavicle, the patient reported that his phantom hand began to swell and feel warm. **E:** In response to stimulation applied at a point on his right ear, he reported that his phantom arm had telescoped into the stump so that the elbow protruded from the stump. **F:** Later, stimulation applied at the same point resulted in a further retraction of the phantom, leaving only the wrist and hand protruding from the stump.


A
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Parkes (1976) reported that about 50% of his patients who had moderate or severe phantom limb pain likened it to the pain which they had experienced before amputation. Roth and Sugarbaker (1980) reported that 17.5% of their amputees had phantom pains or sensations that mimicked pre-amputation experiences.

Sherman and his co-workers surveyed American veterans who sustained amputations related to military service, and civilian amputees whose amputations were primarily accident- and disease-related (Sherman, Gall, & Gormly, 1979; Sherman & Sherman, 1983; 1985; Sherman et al., 1984). In the earlier study, it was found that 37.5% of patients reported phantom limb pain of the same description, and in the same location, as the pre-amputation pain (Sherman et al., 1979). In the other studies, pain in the limb prior to amputation was not related to the subsequent development of phantom limb pain, but few details were given[3] (Sherman & Sherman, 1983; 1985; Sherman et al., 1984).

Wall, Novotny-Joseph, and Macnamara (1985) arrived at a similar conclusion following their retrospective study of 25 patients who underwent hemipelvectomies or hip disarticulations for cancer of the lower extremities. Fourteen of the 16 patients who had had pre-amputation pain subsequently developed phantom limb pain as did eight of the nine who did not have any pre-amputation pain. Only two patients (12.5%) reported that their phantom limb pain was identical in location to the pain they experienced prior to the amputation.

Jensen et al. (1985) conducted the only prospective, long-term study designed to examine the relationship between pre-amputation pain and the subsequent development and course of phantom limb pain in a series of 38 amputees. Information was obtained regarding the location and character of pain experienced the day before amputation, and again, with respect to phantom limb pain, 8 days, 6 months and 2 years after amputation. Pre-amputation

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[3]It should be noted that a statistical analysis which tests whether phantom limb pain occurs more frequently in patients who experienced pain in the limb before amputation than in patients who did not, does not necessarily address the issue of the similarity of pains before and after amputation. The conclusions that may be drawn from such data are limited, and unless backed up with additional information on the nature of the similarities (and differences), the observed relationship may merely indicate that certain individuals are more likely to suffer pain than others, both before and after amputation. Similarly, negative findings may reflect the fact that certain frequently occurring pre-amputation pains rarely occur as phantom phenomena after the limb has been cut off.

pain and phantom limb pain were similar in both location and quality in 36% of patients eight days after amputation and had decreased to 10% at the 6-month and 2-year follow-ups.

A re-examination of the clinical records of the chronic pain patients studied by Katz and Melzack (1987) revealed that six of the eight amputees (75%) reported that at least one type of phantom limb pain they suffered from was similar to that which they had experienced before amputation (see Table 1 for descriptive details).

These studies indicate that between 12.5% and 79% of amputees report that they suffer from phantom limb pain similar to the pain they experienced prior to amputation but the nature of the pains are rarely specified. The wide range in estimates may be explained by a variety of factors, including the criteria used to determine similarity, the type, severity and duration of the pre-amputation pain, and whether a description of the pain was also obtained prior to amputation. But since these studies do not report information of this nature, it is not known which factors are important.

To summarize, studies of groups of amputees report the incidence or prevalence of somatosensory memories after amputation but with little information about the types of pains which recur. Case reports provide these descriptive details but without information regarding the frequency of their occurrence. What is lacking is a systematic description and categorization of various types of pre-amputation pains which persist or recur following amputation, and an estimate of their relative frequencies of occurrence. Study 4 will examine, retrospectively, the prevalence of painful and non-painful somatosensory memories in a random sample of amputees.

Focus of Research

The thesis consists of four studies. The first is a series of case presentations of amputees with phantom limb pain. Study 1 documents the referred sensations and pains these patients report during a form of transcutaneous electrical nerve stimulation (TENS) applied at several regions of the body. These reports raise the questions of whether the referred phantom

sensations (a) are induced by TENS and (b) have peripheral sympathetic nervous system correlates which can be measured at the stump. Studies 2 and 3 are designed to address these questions by measuring skin conductance and skin temperature at the stump and intact limb in amputees reporting a painful phantom, non-painful phantom, and no phantom at all . Study 4 is a retrospective examination of the frequency with which various types of pre-amputation experiences are reported to recur in the phantom limb after amputation.

STUDY 1: REFERRED SENSATIONS

The first study consists of a series of case presentations of amputees with phantom limb pain and documents the referred sensations they report during transcutaneous electrical nerve stimulation (TENS) applied to the outer ear, torso, and face. This phenomenon was initially observed during a placebo-controlled trial of "auriculotherapy" which showed that although electrical stimulation applied to the outer ear failed to produce significant relief of chronic pain, it unexpectedly resulted in a large number of referred sensations (Melzack & Katz, 1984). This phenomenon was subsequently reviewed in detail by Katz and Melzack (1987) in a series of 98 chronic pain patients. Thirty-nine percent of patients reported a variety of sensations referred to different parts of the body, the majority being experienced in the painful region. Among the sensations were paresthesias, pain, temperature changes, pressure, and constriction. When examined on the basis of diagnosis, it was found that patients with phantom limb pain reported the most varied types of sensations.

The purpose of this first study is to describe the referred sensations experienced by the eight patients with phantom limb pain since their reports provide the rationale for the two following studies. The original clinical note (Katz & Melzack, 1987) presented case histories of only three of the patients. This section, therefore, contains the remaining five amputees as well as McGill Pain Questionnaire pain ratings obtained before and after electrical stimulation. TENS was delivered at a low frequency (4 to 10 Hz) with long and short pulse widths

(125 ms and approximately 100 μs, respectively). Stimulation intensity was increased until the patient reported a strong, but not uncomfortable sensation.

Case Descriptions

Case 29

Female: born 1917 *Left forequarter amputation*

This patient was seen at the Montreal General Hospital Pain Center in 1981. In 1958 a neoplasm was discovered in her left breast. She received a course of radiation therapy to the left upper quarter of her body. During the next 8 years she developed radiation necrosis of the chest wall with extensive decomposition of the sternum, upper thoracic ribs, and left clavicle. At the same time she became aware of excruciating pains in the left arm and hand. In 1964 a left radical mastectomy was performed. In 1973 a left dorsal rhizotomy was performed at C4-C8. The patient reported feeling phantom limb pain within hours of recovering from the rhizotomy. Several months later she underwent a right cordotomy at C1-C2. In 1979 a left forequarter amputation was performed in which the arm was removed at the shoulder joint.

The patient described the phantom pain as a tight, cramping sensation referred mainly to the phantom hand and fingers. She noted that at times the pain was "very similar" to the pain she had suffered prior to amputation, especially the cramping in her index, middle and ring fingers and the "soreness" in the small finger. She reported that when the pain was at low intensity she could "move" the phantom fingers and wrist at will, but at high intensity her ability to control these movements disappeared. The pain was usually worse at meal times and the end of the day when she was tired. It could be triggered by reading or watching violent material on the television. Noxious stimuli applied to the sound hand were felt locally, and in the phantom hand, as pain. A small region on her abdomen would become "full of pins and needles" when the phantom pain increased. The application of TENS to this region produced a "loosening" of the painful phantom posture.

The patient was seen on three occasions in which TENS was applied at a variety of points on her outer ears. On all sessions she reported sensations referred to the phantom during electrical stimulation. These included a "clutching" sensation between the phantom elbow and wrist, a sensation of heat which travelled down the forearm from the elbow to the wrist, changes in the posture of various phantom parts, movement, and increases and decreases in paresthesias and phantom pain. TENS had little effect on the intensity of the phantom limb pain (see Appendix A-1).

Case 51

Female: born 1927 *Bilateral above-knee amputations*

This patient was seen in 1981. She had been referred for phantom limb pain of 40 years' duration. She recounted that at the age of 14 she was run over by a tram car. She did not feel any pain until the car was moved and then lost consciousness. She remembers awakening in a hospital bed screaming for the doctors to relieve the excruciating pain in her heel. It was not until she looked under the bed covers that she realized that her legs had been amputated.

Two weeks prior to the accident, when running down a flight of stairs, she had "torn a chunk" of flesh from her heel on a nail, and felt pain that persisted up to the time of the accident. The pain she experienced in her phantom heel on awakening in the hospital after the amputation was the same as the pain due to the earlier injury. This pain disappeared shortly after the amputation.

At the time of the interview, the patient reported two types of phantom limb pain. One was a burning pain mostly in the toes of the phantoms but occasionally spreading to the backs of the ankles and the soles of the feet. The more troublesome pain was in the right phantom which she described as "seizures", like "hot poker stabs in the knee". With the passage of time, these pains became more frequent and intense. She did not have a painless phantom: when she was pain-free, she was also phantom-free. The phantoms were defined by the

presence of pain. Watching "unpleasant" scenes on television increased the pain and caused her legs to move about.

She was seen on two occasions. Stimulation applied to the ears was followed by an initial intensification of the burning pain in both phantom feet. However, in this case it is doubtful whether the TENS was responsible for the pain increase. The patient was extremely anxious and apprehensive about receiving "electrical" stimulation. The pain reaction appeared to be part of a generalized emotional response to the stimulation. Nevertheless MPQ pain ratings were reduced at the end of both sessions (see Appendix A-1).

Case 54

Female: born 1912 *Right above-knee amputation*

The patient was seen in 1981, four weeks after amputation. She reported a burning in the ankle, heel, sole and toes of the phantom foot. She was aware of her phantom shin only during bouts or "jabs" of pain. She reported that she continued to feel painful diabetic ulcers on the medial and lateral aspects of her phantom foot which had been present at the time of amputation. Occasionally there was a "swelling" feeling in her phantom ankle. On the first session she noticed a dramatic decrease in the intensity of the phantom pain during TENS applied to both ears (see Appendix A-1). During stimulation she stated that her phantom toes merged into an indistinct "lump". At the same time she experienced a heavy numb sensation spreading up her phantom leg. Pain relief lasted for several hours after stimulation.

Case 57

Male: born 1926 *Right forequarter amputation*

This patient was a 55-year-old manual labourer who sustained a complete fore-quarter amputation of his right shoulder as a result of a work accident two years prior to his referral to the pain center. He had suffered from continuous phantom limb pain ever since the accident. His phantom limb consisted solely of the lower arm and hand, with a gap between the stump and the beginning of the phantom elbow (Figure 1). He described the limb as suspended in

space. Whenever he walked, it behaved as a real limb would, swinging naturally back and forth in synchrony with his contralateral leg. Such automatic movements of the limb were common. He described how, while walking down a flight of stairs, he instinctively reached out to grab the bannister with his phantom arm to avoid a fall, but ended up lying on the landing below. In contrast, it required considerable effort for him to willfully move the phantom limb: he reported that he was able to partially open the hand, but this was a slow and frustrating process that required enormous concentration.

He described the pain as a throbbing across the dorsum of the phantom hand. The thumb and index finger were especially sensitive. On his first visit to the pain center, one of his primary complaints was that he had great trouble falling asleep at night, for if he lay on his right side he would experience a sharp increase in pain, and when he turned over, the phantom arm would rise upward like a helium-filled balloon until it was fully extended over his head. After several minutes in this posture, his arm would become heavy with fatigue, and an unbearable pain would ensue, forcing him to shift position once again.

On the first session, during electrical stimulation applied at a point on his left ear, he reported a warm sensation that traveled down his phantom arm and into the hand which then began to swell. Later, when the stimulus was applied to the same point on his his right ear, he remarked that his phantom limb had suddenly telescoped into the stump so that the once empty space was now occupied by the lower arm. On the following day, during stimulation of the same point, he reported a further abrupt retraction of the limb, leaving the wrist and hand protruding from the stump. The patient subsequently reported an improvement in his sleep and no longer experienced involuntary tendencies to use the phantom in moments of urgency. The shortened version of the phantom was maintained at a five-month follow-up visit, and more recently in a telephone interview three years later.

Case 60

Male: born 1921 *Bilateral above-knee amputation*

This patient was a 60-year-old man with bilateral lower extremity phantom pain. The pain consisted of intense, rhythmic throbbing in the big toes of both phantom feet. As the pain increased in severity, the throbbing became the focus of frequent, sharp, lightning-like bursts of pain which shot up the length of his phantom legs, causing an uncontrollable jerking of the stumps, leaving him visibly shaken.

This patient was seen on seven occasions. Without exception, within three minutes of stimulation applied at either ear, he reported that both phantom limbs would vanish. With the disappearance of his phantom legs, any pain he had previously been experiencing was also reportedly abolished. This state of "phantom-less" limbs persisted for several hours after stimulation until he would suddenly notice that his phantom limbs had returned. During the interval of stimulation that preceded the disappearance of his phantom legs, he remarked that he was experiencing sensations in new and unfamiliar parts of his limbs, including a tingling in both phantom soles and heels. These were parts that he had never felt after he lost his legs and were not features that ordinarily defined his phantom limbs.

Case 67

Male: born 1938 *Left above-knee amputation*

This 42-year-old man was seen in 1981 for a very brief time. In 1978 he was hit by a car while riding a bicycle leading to the amputation of his left leg above-the-knee. He had suffered from phantom limb pain ever since. The patient was seen on a single occasion only, at request of his physician, and McGill Pain Questionnaire pain ratings were not obtained. For about 5 minutes, a variety of points on his ears were electrically stimulated. During and after stimulation the patient stated emphatically that there had been absolutely no change in phantom limb sensations or pain.

Case 79

Female: born 1927 *Left forequarter amputation*

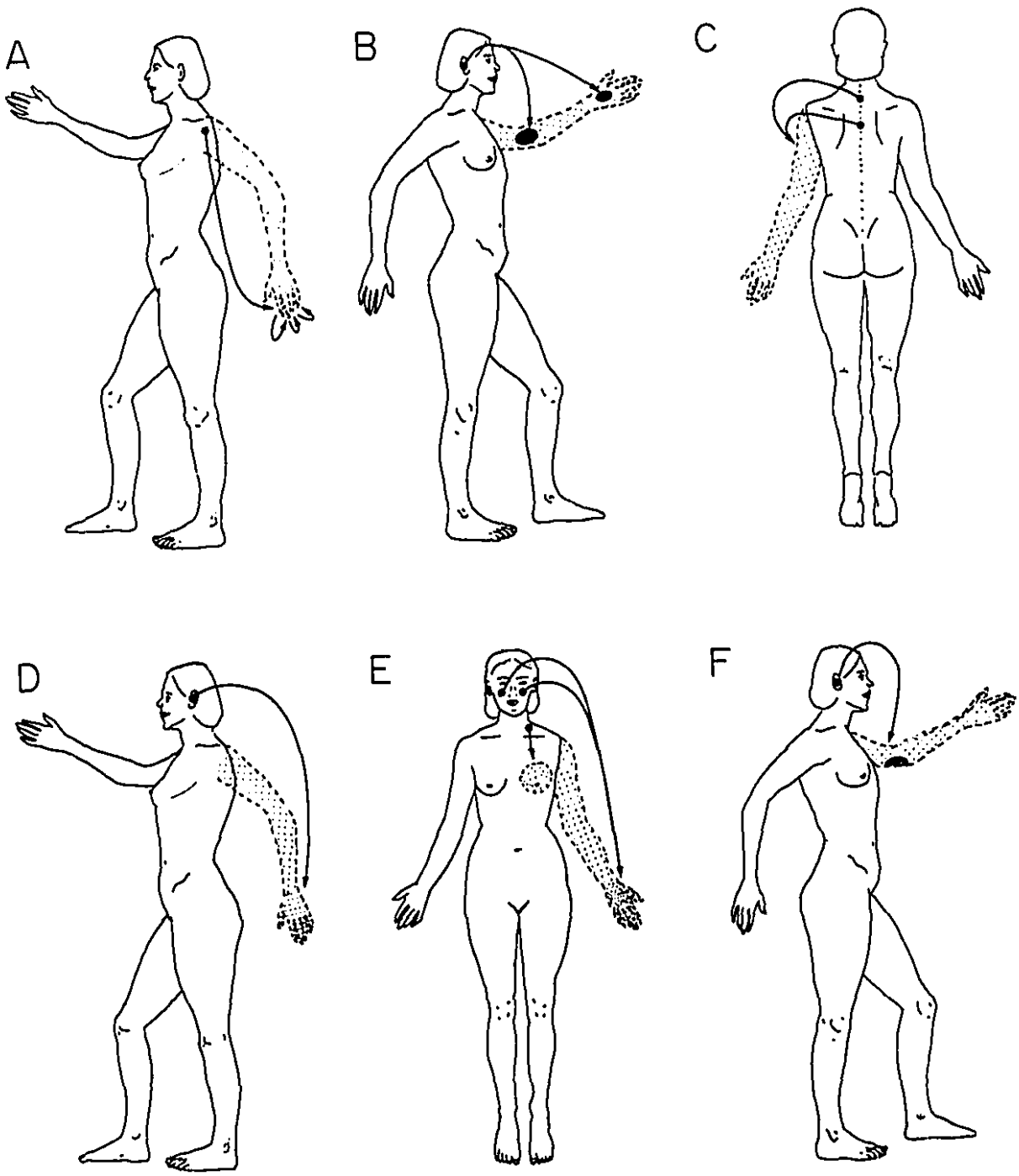
This patient was a 54-year-old woman who had been referred to the Pain Center, in 1981, because of severe and unrelenting phantom limb pain of three years' duration. In 1962 she had received an over-exposure of irradiation during a course of cobalt radiotherapy following a left radical mastectomy for breast cancer. During the next eight years she developed progressive numbness, atrophy, and paresis of the left arm with marked necrosis and ulcerations of the left chest wall and arm which required repeated debriding and drainage. By 1978 there was complete numbness and paralysis of the left arm. A left fore-quarter amputation was performed, and the area of ulceration on the chest was repaired by skin grafts using tissue from the amputated arm. Immediately after the amputation she reported a distinct and severely painful phantom arm.

She described the usual position of her phantom arm as one in which the upper arm rested at her side. The elbow was bent at an angle slightly less than 90 degrees and the hand lay below her right breast. The limb was always painful. The pain consisted of an intense, hot, generalized throbbing of the phantom arm. The fingers and hand would often swell out of proportion to the rest of the arm, producing an intense constricting pain. When the pain reached an intolerable level, she described how the entire limb would extend rigidly outward at shoulder height until the pain subsided.

She was seen on five occasions. On each of these, she reported a considerable increase in the intensity of phantom sensations during electrical stimulation applied at points on her ears, face, and body (Figure 2). On the first session, when stimulation was applied to a point on her left ear, she reported feeling tingling sensations referred to parts of her phantom arm that she had never experienced before. She described fleeting, yet distinct, sensations along the anterior aspect of her elbow, and an itching of the palm and fingers.

On another occasion when stimulation was applied to the ear she reported, with some surprise, a "pleasant sensation of draining" in her phantom arm, and remarked that it was very

Figure 2. Referred sensations reported by Case 79 during electrical stimulation. To simplify the illustration of the referred patterns, the phantom limb is depicted as being at the side rather than bent at the elbow and across the chest as the patient actually reported. Dots and arrows represent stimulation and referral sites, respectively. **A:** Stimulation applied at a point on the stump produced a sensation in the phantom arm that consisted of electric-shock-like activity that jumped from finger to finger. **B:** When stimulation was applied to her right ear, she remarked that the sensations were referred to the inside of her phantom elbow and parts of the palm, neither of which she had experienced before. **C:** An increase in the paresthetic sensations in her phantom arm resulted from stimulation applied at the two points on the midline of her upper back. **D:** Stimulation of a point on her left ear produced a sensation of heat "rushing" into the phantom hand. **E:** Stimulation applied on either side of the nose produced a cool sensation in the phantom hand; when applied to a point just above the clavicle on the left side, she reported a distinct sensation of a phantom breast. **F:** During stimulation applied to a point on the right ear the patient reported that her phantom elbow became warm and she described a pulsing sensation that traveled down the wrist and into the thumb.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much like the sensation she had experienced prior to the amputation when the ulcers on her arm had been drained. Stimulation applied just above the left clavicle produced a distinct awareness of a phantom breast, another sensation that she had not experienced before. Applying the stimulus approximately 1.5 cm to the left and right of her nose produced a rushing sensation of "coolness" in the phantom hand and a tingling in the palm. On the third session, she described a pulsing sensation that traveled down the wrist and into the phantom thumb. Later, stimulation applied to a point on the stump produced a spread of activity which she described as an "electric shock-like" sensation that jumped from one digit to the next, beginning at the index finger, and continuing until the entire hand was tingling. A telephone follow-up three years later indicated that she continued to apply electrical stimulation to her outer ears for 20 minutes each day with satisfactory pain relief for up to three hours.

Case 93

Female: born 1933 *Left forequarter amputation*

This patient was seen in 1981. Her left arm had been removed several years earlier as the result of a neoplasm. For a period of one year prior to the amputation she carried her left arm in a sling with the elbow at an angle of approximately 60 degrees. Ever since the amputation she has felt the phantom limb to be in the same pre-amputation posture. She was seen on 11 occasions. TENS was applied at various point on her outer ears and torso. Among the sensations she reported during TENS were itchiness, pressure and constriction, "lightness", "electricity", and "shooting". This patient reported a considerable cumulative reduction in the intensity of her phantom pain over the course of treatment (Appendix A-2).

Discussion

These case reports suggest that there is a convergence and summation of somato-sensory inputs from the outer ear, face and torso at neuron pools in the central nervous system and that their activity underlies the referred sensations these patients experience. All but one

patient reported an overall increase in the vividness of phantom sensations during stimulation. In addition, two of them experienced parts of their phantom limbs for the first time. Pressure and constriction, changes in perceived weight, paresthesias, temperature, and pain were among the more frequent phantom sensations reported.

The referred sensations can be explained by two general mechanisms. The first is a purely "central" account. Many of the sensations these patients reported during stimulation resemble those of patients with pain problems or other disorders undergoing electrical stimulation of various pathways and structures along the somatosensory projection system. The most common sensation reported by patients who receive electrical stimulation of the dorsal column-medial lemniscal pathway is one of paresthesias whereas electrical stimulation of the spinothalamic pathway invariably produces referred sensations of temperature change, and on rare occasions, pain (Krainick & Thoden, 1981, 1989; Krainick, Thoden, & Riechert; 1980; Mundinger & Neumüller, 1981; Nittner, 1982; Tasker, 1989; Tasker, Organ, & Hawrylyshyn, 1982; Young, 1989). In patients who suffer from chronic pain, electrical stimulation at almost any brain site reproduces their pain (Nathan, 1985; Obrador & Dierssen, 1966; Tasker, 1989). Thus, it is possible that inputs from the outer ear and torso converge at higher levels of the CNS to produce painful and non-painful sensations referred to the phantom.

A central explanation of referred sensations is also consistent with the remarkable sensory memory re-experienced by Case 79 who reported feeling "a pleasant sensation of draining" in her phantom limb that she had originally experienced years earlier prior to the amputation of her arm. Leriche (1947a, 1947b) described a patient who developed painful ulcerations of the Achilles tendon and surrounding region from an ill-fitting plaster cast. The ulcers had completely healed at the time of an (unrelated) amputation several months later. Although the patient subsequently developed stump pain, she did not suffer from pain in the phantom limb until six years later, when an injection of lipiodol into the stump instantly, and permanently, revived the pain of the ulcerated Achilles tendon. Nathan (1956, 1962) described

a similar phenomenon after applying noxious stimuli to the stump of an amputee, who later re-experienced the pain of a skating injury that he had sustained 5 years earlier when his leg was intact. More recently, Jensen et al. (1985) observed that as long as two years after the amputation, 10% of their sample of amputees described the localization and character of their phantom limb pain as similar to the pain they experienced before the amputation. Reports of this kind indicate that somatosensory memory traces are laid down at the time of injury, and that under the appropriate conditions they may be re-activated and the original sensations re-lived.

A second explanation of the referred sensations is that they reflect a central mechanism that acts on peripheral structures. Changes in the perceived temperature of various parts of the phantom limb raise the possibility that electrical stimulation affects the discharge of peripheral sympathetic fibers and thus alters the pattern of blood flow in stump tissues. Changes in the temperature of the stump are accompanied by corresponding temperature changes referred to the phantom (James, 1887; Livingston, 1938, 1943; Mitchell, 1872; Pitres, 1897; Sunderland 1978). Spinal cord stimulation results in increased peripheral blood flow and the subjective sensation of paresthesias and warmth in the extremities (Dooley, 1977; Groth, 1985; Meglio, Cioni, Dal Lago, De Santis, Pola, & Serricchio, 1981). It is possible that sensations of warmth, referred to the phantom, represent a vasodilation of stump blood vessels as the result of a reduction in peripheral sympathetic activity. Likewise, the increases and decreases in the intensity of paresthesias referred to the phantom may reflect a sympathetic-efferent--somatic-afferent cycle in which alterations in the amount of neurotransmitter release determine, in part, the rate of primary afferent discharge from peripheral fibers in the stump. Such a mechanism may account for alterations in the intensity of paresthesias referred to the phantom limb.

These two explanations of the referred sensations are not mutually exclusive and it may be that one, or both, are operative within a given subject. Thus some perceived changes in the phantom limb may reflect purely central mechanisms and others may represent a combination of peripheral and central influences. That the referred sensations are due simply

to suggestion is unlikely since no suggestions were given, yet there is a remarkable consistency in the reports of these patients.

Subjective reports of pain relief (Appendices A-1 and A-2) were common during and after TENS but in the absence of control data, the effectiveness of this form of stimulation cannot be addressed. Stanley and his colleagues found that volunteers undergoing TENS applied at point on the head (i.e., "trans-cranial electrical stimulation") report "warm and tingling sensations over their bodies" and also show a higher threshold to various forms of painful stimuli (Stanley, Cazalaa, Limoge, & Louville, 1982; Stanley, Cazalaa, Atinault, Coeytaux, Limoge, & Louville, 1982). However, they also did not control for the effects of non-specific factors. The question of whether TENS applied at the outer ear is more effective in reducing phantom limb pain than a placebo control will be one focus of Study 2.

These data on referred sensations suggest that deafferentation due to disease, injury or other lesions of the CNS leads to a hypersensitivity of the CNS and an increased probability of referred pain of long duration. Sensations referred to the phantom limb, brought about by electrical stimulation at a variety of sites distant from the stump, suggest that inputs from widespread regions of the body converge at excitatory neuron pools in the CNS. It is also possible that descending fibers tracts which terminate in the lateral horns of the spinal cord are responsible for inhibiting sympathetic nervous system activity in the stump and surrounding tissue.

Study 2, which follows, measures pain levels and sympathetic nervous system activity during electrical stimulation applied at the outer ears.

STUDY 2: SYMPATHETIC NERVOUS SYSTEM CORRELATES OF PAINFUL AND NON-PAINFUL PHANTOM LIMBS

The second study examines the hypothesis that painful and non-painful phantom limb sensations have peripheral sympathetic nervous system correlates that can be measured at the stump.

Objectives and Hypotheses

The four specific objectives of this investigation are:

1. To determine whether reports of sensations referred to the phantom limb during TENS applied at the ears reflect a central mechanism that acts on peripheral sympathetic fibers.

Measures will be taken of phantom limb intensity, skin conductance, and surface skin temperature at the stump and contralateral intact limb. Since the design calls for continuous monitoring of these variables it will be possible to determine whether changes in the intensity of phantom limb pain and other sensations are correlated with changes in sympathetic nervous system activity at the stump.

2. To determine whether TENS applied at the outer ears is more effective than a placebo treatment in reducing phantom limb pain.

Although Melzack and Katz (1984) failed to demonstrate that "auriculotherapy" was effective in decreasing chronic pain, their sample did not contain any cases of phantom limb pain. Reports from other subjects with phantom limb pain who received this form of electrical stimulation in the pilot observations described in Study 1 suggest that it may be effective in reducing phantom limb pain.

3. To ascertain whether the reports of referred sensations during TENS applied at the outer ears are due to the electrical stimulation or are "placebo"-induced, or possibly, whether they occur spontaneously, and are noticed especially when attention is focused on the phantom

limb. This study will alternate baseline periods of no stimulation with TENS or with a placebo control on two different days.

4. To compare the peripheral manifestations of sympathetic nervous system activity at the stump and intact limb in three groups of amputees: (a) Group PLP, comprising subjects with phantom limb pain; (b) Group PLS, comprising subjects with non-painful phantom limb sensations; and (c) Group No-PL, comprising subjects who reported not feeling a phantom limb at all.

Results of recent studies by Sherman suggest that phantom limb pain is associated with reduced blood flow at the stump compared to the intact limb (Sherman, 1984; Sherman & Bruno, 1987). However, many of the patients reported by Sherman and Bruno had significant amounts of stump pain as well as phantom limb pain. Thus, the presence of phantom limb pain was confounded with concurrent stump pain, making it unclear whether the decreased blood flow was a function of stump pain, phantom pain, or both. Furthermore, since Sherman did not include a group of amputees without phantom limb pain, it may be that decreased blood flow to the stump is a characteristic of all stumps regardless of the patient's status with respect to phantom limb pain. Based on the results of Sherman (1984), and Sherman and Bruno (1987), two hypotheses are proposed: (a) the stump will register higher skin conductance levels and lower skin temperature than the contralateral intact limb for subjects in Group PLP only, and (b) the stump will register higher skin conductance levels and lower skin temperature in Group PLP when compared to Groups PLS and No-PL.

Methods

Sample

Twenty-eight amputees (18 males and 10 females) with unilateral upper or lower extremity amputations comprised the sample for Study 2. The mean age of the sample at the time of testing was 52.8 years (range = 23 to 73). The mean time since the amputation was 5 years with a range of 36 days to 46 years. The site of amputation was below the knee in 9,

above the knee in 16, below the elbow in 1, and above the elbow in 2. One subject had lost an arm and a leg. In this case measurements were taken from only the upper extremity. The reason for amputation was peripheral vascular disease (including diabetes mellitus) in 12 subjects, accident in 9, arterial thrombosis in 3, tumour in 2, and one each for radiation damage and polio.

Subjects were assigned to one of three groups on the initial session based on the presence or absence of painful or non-painful phantom limb sensations. Group No-PL comprised 8 amputees who reported that they did not feel the presence of a phantom limb at the time of testing. Group PLS consisted of 9 amputees who reported feeling only non-painful phantom limb sensations at the time of testing. Group PLP consisted of 11 subjects who reported having phantom limb pain at the time of testing. Subjects were recruited by advertisements placed in local newspapers and newsletters, postings at orthopedic appliance shops, and from the Catherine Booth Hospital Center, Constance-Lethbridge Rehabilitation Center, Montreal Convalescent Hospital Center, Montreal General Hospital, and Royal Victoria Hospital.

Pain assessment and psychological measures

Subjects completed the following battery of questionnaires and personality inventories (see Appendices B-2 to B-8).

McGill Comprehensive Pain Assessment Schedule (MCPAS). The MCPAS is a standardized clinical assessment interview instrument devised by Monks and Taenzer (1983) and based on a form originally developed by Melzack (1975). It is designed to gather patient information from a number of interrelated systems, including the biological, psychological and social. The abridged version used in this study was modified for use with amputees.

McGill Pain Questionnaire (MPQ). The MPQ was developed by Melzack (1975) to obtain quantitative and qualitative measures of the experience of pain. The MPQ yields two global scores, the pain rating index (PRI) and the present pain intensity (PPI), which have been found to provide valid and reliable measures of pain (Hunter, Phillips, & Rachman, 1979; Melzack, 1983; Prieto & Geisinger, 1983; Reading, 1983). The PRI is the sum of the rank values of the words chosen from 20 sets of qualitative words, each set containing two to six adjectives that describe the sensory, affective and evaluative properties of pain. The lists of pain descriptors are read to the patients who are asked to choose the word in each category that best describes their pain at the moment. The PPI is rated on a scale of zero to five as follows: 0 = none, 1 = mild, 2 = discomforting, 3 = distressing, 4 = horrible, and 5 = excruciating. An alternate weighted-rank method for scoring the MPQ is also available (Melzack, Katz, & Jeans, 1985). This method has been demonstrated to be more sensitive to pain intensity than the rank scoring method described above, and has been advocated for use in research. The MPQ takes approximately 5 minutes to administer.

Eysenck Personality Inventory (EPI). The EPI is a widely used clinical and research instrument which provides two unidimensional orthogonal measures of personality each conceptualized along a continuum (Eysenck & Eysenck, 1968). These dimensions are extraversion-introversion (EPI-E) and neuroticism-stability (EPI-N). A lie scale (EPI-L) is included to screen out blatant attempts at falsification. The EPI consists of a 57 item inventory in forced choice format, and takes about 10 minutes to administer. Test-retest reliability coefficients range between .80 and .97 with an interval of several months between testings. Concurrent validity has been established through correlations of the EPI-E and EPI-N with other recognized personality inventories.

Beck Depression Inventory (BDI). The present study used the 21-item BDI (Beck, 1967; Beck, Ward, Mendelson, Mock, & Erbough, 1961). Each item is composed of several statements varying in the degree to which they reflect depressive symptomatology and attitudes. Each statement is assigned a rank value between zero and three (with higher ranks corresponding to higher levels of depression). The subjects choose one statement from each item that best reflects how they are feeling at the moment. The total BDI score is the sum of the rank values associated with each statement the subjects endorse. The BDI is one of the better standardized and validated self-rating scales available for measuring depression (Beck, Steer, & Garbin, 1988; Engelsmann, 1976). The BDI takes about 10 minutes to complete.

Spielberger State-Trait Anxiety Inventory (STAI). The STAI is composed of two forms, each of which measures separate dimensions of anxiety (Spielberger, Gorsuch, & Lushene, 1970). The STAI-S consists of 20 statements and measures "state anxiety". The subjects are required to respond on the basis of how they are feeling "right now" (i.e., at the moment when completing the form). The STAI-T measures anxiety as a more enduring personality trait and consists of another 20 statements that pertain to how the subjects "generally feel". The STAI-S has been shown to be sensitive to psychological manipulations that alter anxiety level. Test-retest reliability coefficients of the STAI-T have been reported to be relatively high, reaching approximately .70 after a 3 month interval, and increasing with decreasing time between testings. The STAI has also shown relatively high correlations with other well known measures of anxiety. The STAI takes about 10 minutes to administer.

Wesley Rigidity Questionnaire (WRQ). Wesley (1953) defines rigidity as the "tendency to persist in responses that may previously have been suitable in some situation or other but that no longer appear adequate to achieve current goals or to solve current problems." (p. 129). The version of the WRQ used in this study consists of a 50 item forced-choice questionnaire. The subject responds "true" or "false" to each item indicating whether or

not the item applies to him- or herself and is awarded one point for each "rigid" answer. The total rigidity score is the sum of points obtained across the 50 items. There are no data pertaining to the reliability of the WRQ except that five clinical psychologists all rated the 50 items as high on rigidity. Concurrent validity of the WRQ was established through its relationship with performance on the Wisconsin Card-Sorting Test. High scorers on the WRQ were found to take significantly more trials to reach criterion on the Wisconsin Card-Sorting Test and produced significantly more perseverative errors than subjects scoring below the median. The WRQ takes about twenty minutes to administer.

Mood Rating Scale (MRS). The MRS consists of a series of 18 visual analogue rating scales (Bond & Lader, 1974; Herbert, Johns, & Dore, 1976). Each visual analogue scale (VAS) consists of a 100 mm horizontal line with one word at either pole that defines the end-points of a bipolar continuum (e.g., Alert/Drowsy). The subject is to place a mark across the horizontal line at a point which best describes how he or she is feeling at the time in relation to the two polar adjectives. In a factor analytic study, the MRS yielded two factors ("Alertness" and "Tranquility") when administered to 38 subjects on over 700 occasions during experiments dealing with the quality of sleep (Herbert et al., 1976). Bond and Lader demonstrated the sensitivity of the MRS to mood changes following administration of an anti-anxiety agent. Only those VASs that revealed loadings on either factor of .75 or above were used in Study 2 . Using this criterion the total number of bipolar items was reduced from 18 to 12 (eight comprising the Alertness scale and four comprising the Tranquility scale). The MRS yields two scores corresponding to each factor. The total score for each factor is the sum of the distances (in mm) from the positive poles of the VASs. The MRS takes about five minutes to administer.

Sleepiness Rating Scale (SRS). The SRS consists of two 4-point scales that measure sleepiness and fatigue (Murray, Williams, & Lubin, 1958). Subjects choose one statement from each scale that captures how sleepy and tired they are feeling at the time. The SRS score

is obtained by adding the rank values associated with each statement endorsed (minimum score = 0; maximum score = 8). In a sleep deprivation study Murray et al. found the SRS to correlate negatively with body temperature as hours without sleep increased. This scale takes less than one minute to administer.

Experimental apparatus

Transcutaneous electrical nerve stimulation (TENS) was delivered to the outer ears using an Agar Electronics Neurogar III stimulator connected to two silver earrings which gently clasped the subject's earlobes. Skin conductance (SC) measurements taken at homologous points on the stump and contralateral intact limb were obtained using a portable Thought Technology biofeedback module with digital display (SC200T) and Ag/AgCl Beckman electrodes. The electrode paste consisted of a mixture of physiological saline and a neutral ointment cream having the recommended concentration of approximately 0.050 molar NaCl (Fowles, Christie, Edelberg, Grings, Lykken, & Venables, 1981). Surface skin temperature (ST) measurements at the stump and contralateral intact limb were obtained using a Yellow Springs Instruments (YSI) digital thermometer, Model 49TA, and a YSI Model 409A temperature probe. Skin conductance and skin temperature leads from each limb were connected to a two-channel electromechanical relay which switched channels every 10 seconds. The output from the relay fed into the SC and ST digital displays. Every 10 seconds the SC and ST displays alternated between providing information from the stump and intact limb.

The subject rated changes in the perceived intensity of phantom limb sensations including pain (PLI) by turning a dial which allowed 180 degrees of rotation. The 90 degree setting was labelled "USUAL", 0 degrees, "LESS", and 180 degrees, "MORE". The dial was connected to a 1.35 volt mercury battery via a 10,000 ohm potentiometer and the output fed into a digital volt meter which registered 0 through 0.675 to 1.35 volts corresponding to the 0,

90, and 180 degree settings, respectively. All measurements were obtained on a continuous basis. Digital displays, including a digital timer, were videotaped for later scoring.

Skin sensitivity to pressure was assessed using the Semmes-Weinstein pressure aesthesiometer (Shaw Laboratories, New York) which consists of a set of 20 individual nylon filaments of equal length (38 mm) ranging from 0.06 to 1.14 mm in diameter. Each filament has been assigned a value that represents the logarithm of the force required to bend it maximally when pressed against the skin. Pressure sensitivity thresholds were obtained from the stump first and then the intact limb.

Subjects were shown the filaments and told that each would be applied at a point on the skin of the stump and then at the mirror-image point on the intact limb. A point was chosen near the tip of the stump several cm from the skin conductance electrodes and temperature probe. The designated point and its mirror-image on the intact limb were marked with a felt-tipped pen so that they could be accurately localized from trial to trial. Strands of hair surrounding the selected points were carefully cut to ensure that the filaments came in contact with the skin only.

Subjects were told to close their eyes and indicate when they felt they had been touched. On each trial a filament was applied to the designated point on the skin for approximately one second. In order to reduce the likelihood of anticipatory responses, trials were separated by an interval ranging from five to fifteen seconds. Filaments were applied individually in ascending serial order until the subject had correctly detected the filaments on five consecutive trials. Pressure sensitivity threshold (PST) was defined by the value associated with the filament that had been used on the first of these five trials (i.e., the filament with the smallest diameter).

Design

Subjects received TENS on the first session and placebo "stimulation" on the second, or the reverse order. The procedure (see below) on both sessions was identical except that on the placebo session, non-conducting leads connected the electrical stimulator and ear electrodes so that the subject received no current. Each session was divided into three consecutive periods of 10 minutes each, including an initial resting baseline (B1), bilateral ear stimulation (BES), and a final resting baseline (B2). Throughout the 30 minute session, skin conductance and skin temperature were measured from homologous points on the stump and contralateral limb while the subject monitored changes in the intensity of painful and non-painful phantom limb sensations by turning the dial.

Procedure

The study was carried out by an experimenter who made the initial contact with the subjects, described the objectives and procedures of the study, and scheduled the sessions for the same time on two consecutive days or with as few days intervening between sessions as could be arranged. Subjects were requested to refrain from smoking and drinking coffee and alcohol on the days they were to be seen. Those with phantom limb pain were asked not to take any pain medication on the days they had been scheduled so that between group differences in skin conductance and temperature might be maximized and an accurate medication-free description of the pain could be obtained.

When the subjects arrived for the first session they were brought into a quiet room where the experimental procedures were explained to them and their informed consent was obtained (see Appendix B-1). Subjects were interviewed using the MCPAS as a structured interview guide and then given an envelope containing the EPI, BDI, STAI-T, and WRQ which they were to complete at home and return on the second session. The experimenter reviewed the format and instructions for the questionnaires with each subject until he was satisfied they were understood. Subjects were instructed to fill out the forms on their own,

without the help of family members or friends. If subjects needed help with certain questionnaire items they were told to bring them to the experimenter's attention at the second session.

The procedure for the remainder of the first session and for the second session was the same. Subjects were asked to remove their prostheses and expose the homologous region of the intact limb. Those with fore-quarter amputations and high above-the-knee amputations were offered a hospital gown into which they could change. For the next 20 to 30 minutes subjects sat quietly to allow the exposed stump and intact limb to adjust to the conditions in the room. During this period subjects completed the MRS, SRS, STAI-S and were read the MPQ.

At the same time, the experimenter cleaned the stump and intact limb with alcohol. Skin conductance electrodes and temperature probes were placed on the distal portion of the stump approximately 5 cm from its end and at mirror-image regions on the contralateral intact limb. On each limb the two SC electrodes were separated from each other by approximately 3 cm. Stimulating electrodes were attached to the subject's ears. Mirror-image points on the two limbs were marked with a felt-tipped pen and pressure sensitivity measures were obtained first from the stump, and then the intact limb, using the Semmes-Weinstein aesthesiometer.

Subjects were instructed in the use of the dial to monitor phantom limb sensations. They were told that the 90° setting labelled "USUAL" was to represent the intensity level of their phantom limb sensations or pain at the start of the 30 minute session. They were told to pay close attention to their phantom limb during the next 30 minutes and to turn the dial in the appropriate direction if they noticed any type of change in sensation or pain. They were to indicate the magnitude of a change by the size of the angle of arc produced by turning the dial. In addition, they were asked to describe the nature of the change they experienced. A short practice period followed in which subjects familiarized themselves with the use of the dial and its range. Physiological recording for the 10-minute initial resting baseline period was initiated when subjects understood how to use the dial and had begun monitoring their phantom limb.

At the beginning of the bilateral ear stimulation period (BES) on both sessions, the experimenter turned on the TENS stimulator which was within view of the subjects. They were told that, depending on certain stimulation parameters, they might or might not feel its effects. Stimulation intensity was increased until they reported a strong but tolerable sensation on their ears (TENS session) or until the experimenter announced that they were receiving the appropriate amount of current (placebo session). Subjects were instructed that if adjustments to the intensity of the ear stimulation were required, they were to inform the experimenter who would increase or decrease it accordingly. TENS was delivered at a rate of 4 Hz and pulse width of 100 μsec. Stimulation intensity ranged from 10 to 30 volts across a fixed resistance of 2000 ohms. At the end of BES the experimenter turned off the stimulator and told subjects that they were no longer receiving current. They were informed that the 10-minute final resting baseline period had begun and were reminded to continue to monitor their phantom limb.

Following the final resting baseline period, post-stimulation measures of the MRS, SRS, STAI-S, MPQ, and pressure sensitivity were obtained. The stimulating and recording equipment were removed and both limbs cleansed with alcohol. Throughout both 30-minute sessions the experimenter monitored the digital displays from behind an opaque curtain.

Psychophysiological data reduction

Psychophysiological data collected during each session were reduced in the following manner. The videotapes were reviewed and one value of SC, ST, and PLI was obtained every 10 seconds for both 30 minute sessions. Since SC and ST instruments alternated between displaying information from the stump and intact limb every 10 seconds, each session provided a total of 90 values of SC and ST from each limb and 180 values of PLI. Half of these latter values corresponded to the intensity of phantom limb sensations at the same time as the SC and ST read-outs displayed measurements from the stump, and the other half corresponded to measurements from the intact limb.

It was not possible to reduce the sampling rate of one measurement every 10 seconds without compromising the reliability of the data. Approximately five seconds were required for the output displays to stabilize after the relay had switched from one limb to the other. When reviewing the videotape, values of SC, ST, and PLI were sampled approximately eight seconds into the 10-second interval to ensure accurate measurements.

Mean values of PLI were transformed by subtracting a constant of 0.675 from each. This served to relocate PLI scores so that the 90 degree setting labelled "USUAL" took on a value of 0.0, and deviations from it, in the clockwise and counter-clockwise directions (corresponding to increases and decreases in PLI), had maximum values of ±0.675, respectively.

Results

The results will be presented in five sections. Section I examines demographic, clinical and personality variables for the three groups to determine their comparability prior to testing. Section II evaluates the effects of TENS on SCL, ST, PLI ratings and MPQ pain rating indexes. The relationships between (a) changes in the intensity of phantom limb sensations and (b) skin conductance and skin temperature measured at both limbs are also assessed in this section. Section III is concerned with evaluating the claim that PLP is associated with altered sympathetic nervous system activity at the stump. Specific hypotheses concerning anticipated between-group differences in skin conductance and skin temperature are tested. Also evaluated are limb differences within the three groups. Section IV examines the various qualities of phantom limb sensations and pain reported during the three test periods on the TENS and placebo sessions. Section V describes and examines different patterns of PLI responding for individual subjects and includes material on the phenomenology of the painful and non-painful phantom limb. This section was included because of the large variability in the present data set which suggests that group averages may not always provide representative descriptions of the variables under investigation. It was also intended to complement the more

quantitative analyses which do not address the phenomenological experience and descriptive aspects of the painful and non-painful phantom limb.

Major findings

The major findings of Study 2, to be elaborated in subsequent sections, are listed below:

1. TENS applied at the ears had no significant effect on skin conductance levels or skin temperature at either limb. Mean levels of SC and ST on the TENS session did not differ significantly from the placebo session.

2. Phantom limb intensity decreased significantly for Group PLS during Period BES on the TENS session but not on the placebo session. Mean MPQ pain ratings (PRI-S and PRI-T) for Group PLP were significantly lower after TENS but not after placebo.

3. Skin conductance at both limbs correlated significantly with phantom limb intensity in a significantly greater proportion of subjects in Group PLS than Group PLP.

4. Mean skin temperature was significantly lower at the stump than the intact limb for Group PLP but not Groups PLS or No-PL.

5. There was a significant between-group difference in the qualities of sensations that defined painful and painless phantom limbs. Overall, Group PLP reported more varied types of sensations that Group PLS.

I. Comparability of groups

Demographic and clinical variables. Demographic and clinical variables obtained from the McGill Comprehensive Pain Assessment Schedule (MCPAS) administered during the initial interview are presented in Table 2 for each of the three groups. Univariate one-way ANOVAs computed for each variable in the table revealed no significant between group differences. Thus, at the time of the interview the three groups were comparable in mean age,

Table 2. Means and standard deviations (SD) of demographic and clinical variables for the three groups of amputees. Also shown are F statistics and p-values for univariate one-way ANOVAs comparing the groups.

	Group							
	Phantom limb pain (PLP) (n = 11)		Phantom limb sensation (PLS) (n = 9)		No phantom limb (No-PL) (n = 8)			
Demographic/clinical variable	Mean	SD	Mean	SD	Mean	SD	F (2,25)	p-value
Age at time of interview	46.6	16.3	55.0	12.6	58.8	12.7	1.91	ns
Years of education	8.5	3.0	11.1	2.8	9.8	3.8	1.60	ns
Years since amputation*	2.9	2.8	3.2	6.4	9.9	15.7	1.22	ns
Number of surgical operations	3.3	3.8	5.2	4.7	4.1	5.1	0.47	ns
Number of current medical problems	1.0	1.1	1.2	0.7	1.5	1.2	0.49	ns

*ANOVA calculated on log-transformed values

education level, time since the amputation, number of surgical operations (excluding the amputation), and current medical problems.

Table 3 shows the frequency and percentage of the total number of subjects in each group on the remaining demographic and clinical variables. Chi-square tests of independence for two-way tables were calculated for each variable shown in the table. The three groups did not differ significantly in the number of English- and French-speaking subjects, marital status, living arrangements, cause or level of amputation, use of prosthesis, or site of testing.

Psychological and emotional variables. Table 4 contains the mean scores for the three groups on the three Eysenck Personality Inventory sub-scales (EPI-E, EPI-N, EPI-L), the Wesley Rigidity Questionnaire (WRQ), Spielberger Trait Anxiety Inventory (STAI-T) and the Beck Depression Inventory (BDI). Separate one-way univariate ANOVAs carried out on each measure did not reveal any significant between group differences indicating that at the time of testing the three groups were comparable in terms of their scores on the inventories and questionnaires presented in Table 3.

II. Effects of TENS on psychophysiological variables, pain scores, and mood ratings

Data transformation. The raw data values of phantom limb intensity (PLI), skin conductance (SC), and skin temperature (ST) from each session and limb were submitted to a non-linear smoothing procedure (S-Plus, Statistical Sciences Inc., 1987). This function uses running medians to calculate the smoothed values, computes and smooths the residuals, and then adds the two smoothed series. Figures 3, 4,and 5 show the mean smoothed and unsmoothed values of skin conductance and skin temperature from both limbs during the three periods of the TENS and placebo sessions for Groups PLP, PLS, and No-PL, respectively. Smoothed data values are represented by the solid lines and the original unsmoothed values are shown as points.

Table 3. Number and percentage of subjects in each of the three groups on demographic and clinical variables. Also shown are chi square (χ^2) statistics and p-values for tests of independence on each variable.

	Group							
	Phantom limb pain (PLP) (n = 11)		Phantom limb sensation (PLS) (n = 9)		No phantom limb (No-PL) (n = 8)			
Variable	Number	%	Number	%	Number	%	χ^2(df=2)	p-value
Mother tongue								
English	6	54.5	4	44.4	5	62.5	0.56	ns
French	5	45.5	5	55.6	3	37.5		
Marital status								
Married	5	45.5	3	33.3	3	37.5	0.32	ns
Single/Widowed/Divorced	6	54.5	6	66.7	5	62.5		
Living arrangements								
Alone	3	27.3	1	11.1	2	25.0	0.85	ns
With at least one other	8	72.7	8	88.9	6	75.0		
Cause of amputation								
PVD or PVD & DM	4	36.4	4	44.4	4	50.0	0.37	ns
Tumour/Trauma/Other	7	63.6	5	55.6	4	50.0		
Level of amputation								
Above knee or elbow	8	72.7	6	66.7	3	37.5	2.61	ns
Below knee or elbow	3	27.3	3	33.3	5	62.5		
Uses prosthesis daily								
Yes	7	63.6	8	88.9	8	100.0	4.59	ns
No	4	36.4	1	11.1	0	0.0		
Hospitalized at time of testing								
Yes	4	36.4	4	44.4	2	25.0	0.70	ns
No	7	63.6	5	55.6	6	75.0		

Table 4. Means and standard deviations (SD) of psychological and emotional variables for the three groups of amputees. Also shown are F statistics and p-values for uni-variate one-way ANOVAs comparing the groups.

	Group							
	Phantom limb pain (PLP) (n = 10*)		Phantom limb sensation (PLS) (n = 9)		No phantom limb (No-PL) (n = 8)			
Psychological variable	Mean	SD	Mean	SD	Mean	SD	F(2,24)	p-value
Eysenck Personality Inventory								
Extraversion	13.3	3.2	11.3	4.4	13.4	2.7	0.98	ns
Neuroticism	13.9	6.1	9.9	5.4	7.0	6.7	2.97	ns
Lie Scale	3.2	1.4	3.8	1.9	4.6	2.4	1.25	ns
Wesley Rigidity Questionnaire	24.6	7.8	28.0	5.4	29.5	4.7	1.49	ns
Spielberger Trait Anxiety Inventory	41.2	11.3	37.7	9.9	35.9	13.2	0.51	ns
Beck Depression Inventory	14.0	10.3	7.7	5.5	9.1	9.7	1.36	ns

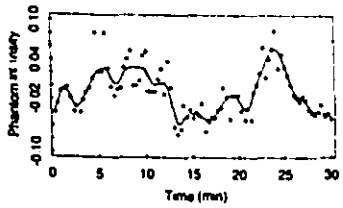
*Reduced sample size due to one subject who could not read sufficiently well to fill out the questionnaires.

Figure 3. Mean smoothed and unsmoothed data for Group PLP showing phantom limb pain intensity ratings (4 top panels), skin conductance (4 middle panels), and skin temperature (4 bottom panels) on the TENS and placebo sessions during Periods B1 (1 - 10 min), BES (11 - 20 min), and B2 (21 - 30 min). Original, unsmoothed values are represented by points (•), smoothed values by solid lines. The two upper panels shown under skin conductance and skin temperature correspond to readings taken at the stump (upper left) and intact limb (upper right) on the TENS session. The two lower panels show data from the stump (lower left) and intact limb (lower right) on the placebo session. Values of phantom limb intensity represented in the two left panels correspond to phantom intensity ratings taken at the same time skin conductance and skin temperature readings displayed information from the stump on the TENS and placebo sessions, respectively. Values shown in the two right panels correspond to phantom intensity ratings taken at the same time skin conductance and skin temperature readings displayed information from the intact limb on the TENS and placebo sessions, respectively. See "psychophysiological data reduction" in Methods section for details.

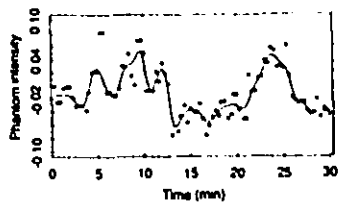
GROUP PLP

PHANTOM LIMB INTENSITY

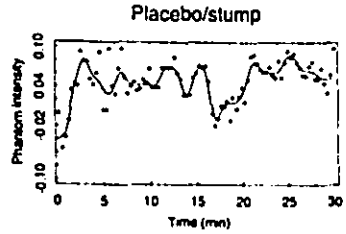
TENS/stump



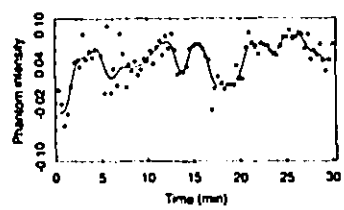
TENS/intact



Placebo/stump

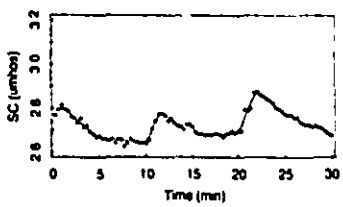


Placebo/intact

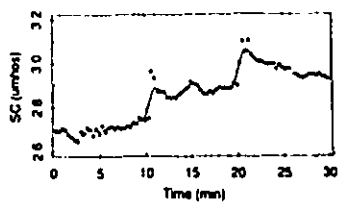


SKIN CONDUCTANCE

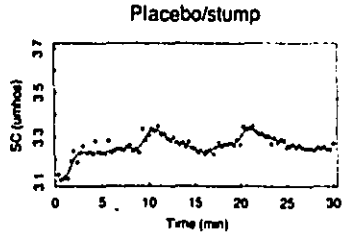
TENS/stump



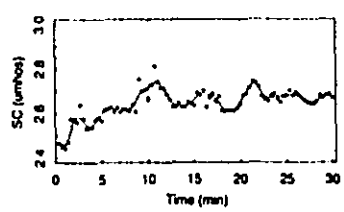
TENS/intact



Placebo/stump

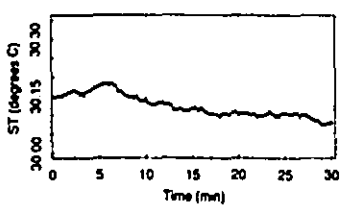


Placebo/intact

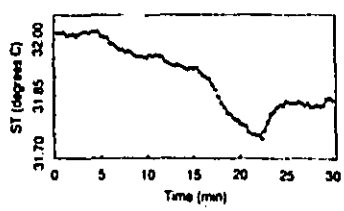


SKIN TEMPERATURE

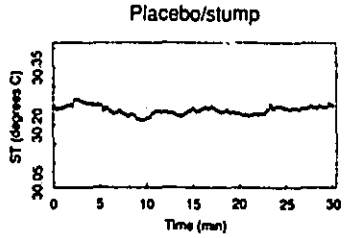
TENS/stump



TENS/intact



Placebo/stump



Placebo/intact

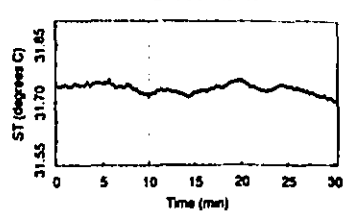
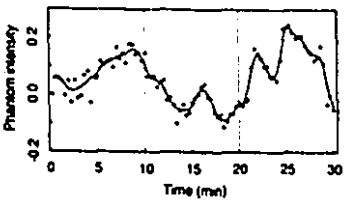


Figure 4. Mean smoothed and unsmoothed data for Group PLS showing non-painful phantom limb intensity ratings (4 top panels), skin conductance (4 middle panels), and skin temperature (4 bottom panels) on the TENS and placebo sessions during Periods B1 (1 - 10 min), BES (11 - 20 min), and B2 (21 - 30 min). Original, unsmoothed values are represented by points (•), smoothed values by solid lines. The two upper panels shown under skin conductance and skin temperature correspond to readings taken at the stump (upper left) and intact limb (upper right) on the TENS session. The two lower panels show data from the stump (lower left) and intact limb (lower right) on the placebo session. Values of phantom limb intensity represented in the two left panels correspond to phantom intensity ratings taken at the same time skin conductance and skin temperature readings displayed information from the stump on the TENS and placebo sessions, respectively. Values shown in the two right panels correspond to phantom intensity ratings taken at the same time skin conductance and skin temperature readings displayed information from the intact limb on the TENS and placebo sessions, respectively. See "psychophysiological data reduction" in Methods section for details.

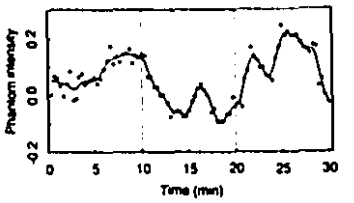
GROUP PLS

PHANTOM LIMB INTENSITY

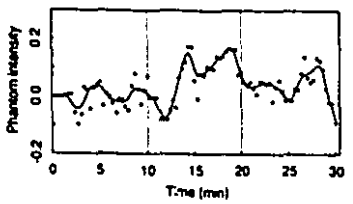
TENS/stump



TENS/intact



Placebo/stump



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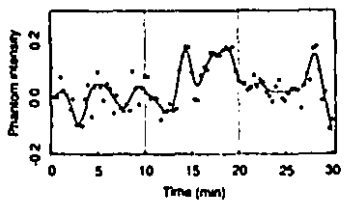
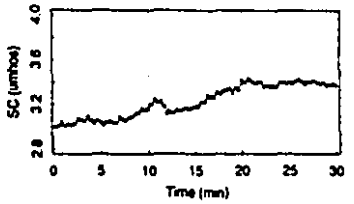
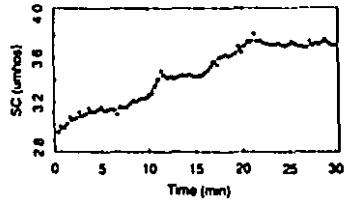


SKIN CONDUCTANCE

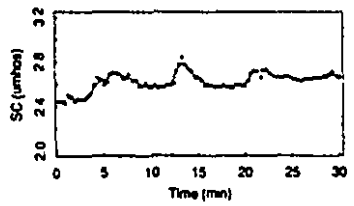
TENS/stump



TENS/intact



Placebo/stump



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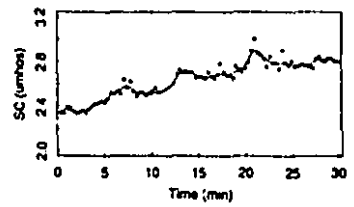
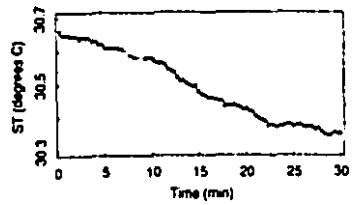
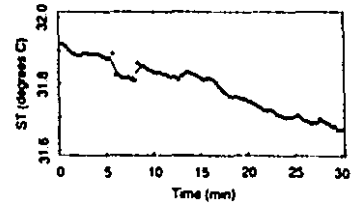


SKIN TEMPERATURE

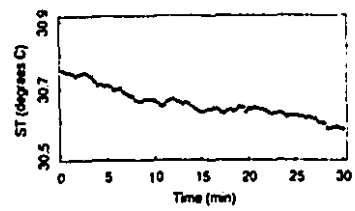
TENS/stump



TENS/intact



Placebo/stump



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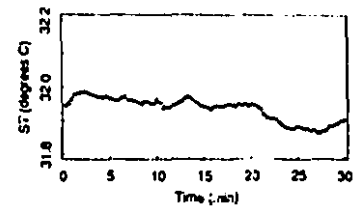
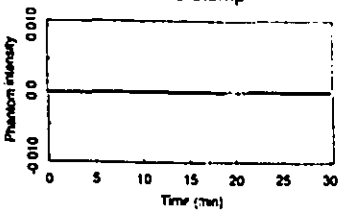


Figure 5. Mean smoothed and unsmoothed data for Group No-PL showing phantom limb intensity ratings (4 top panels), skin conductance (4 middle panels), and skin temperature (4 bottom panels) on the TENS and placebo sessions during Periods B1 (1 - 10 min), BES (11 - 20 min), and B2 (21 - 30 min). Original, unsmoothed values are represented by points (•), smoothed values by solid lines. The two upper panels shown under skin conductance and skin temperature correspond to readings taken at the stump (upper left) and intact limb (upper right) on the TENS session. The two lower panels show data from the stump (lower left) and intact limb (lower right) on the placebo session. Values of phantom limb intensity are equal to zero since Group No-PL comprised subjects who reported no phantom limb at all during any period on either session.

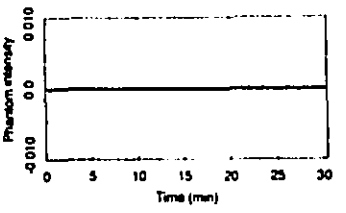
GROUP NO-PL

PHANTOM LIMB INTENSITY

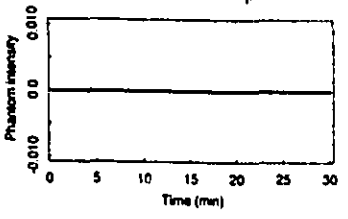
TENS/stump



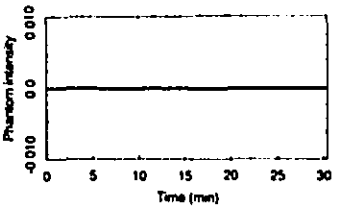
TENS/intact



Placebo/stump

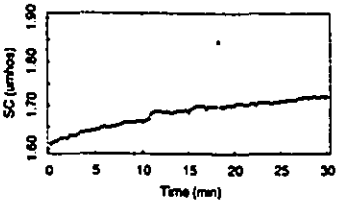


Placebo/intact

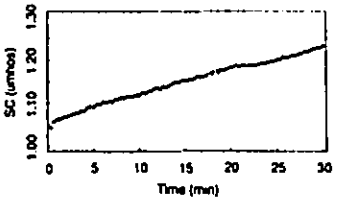


SKIN CONDUCTANCE

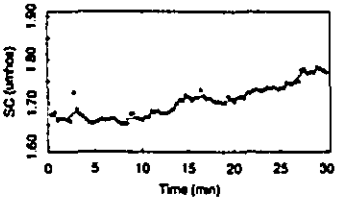
TENS/stump



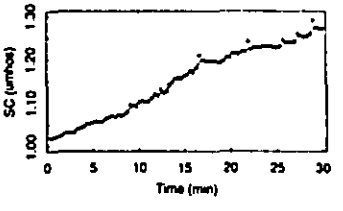
TENS/intact



Placebo/stump

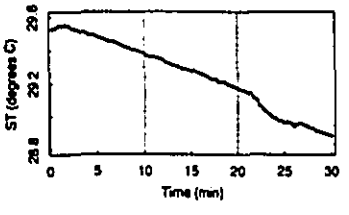


Placebo/intact

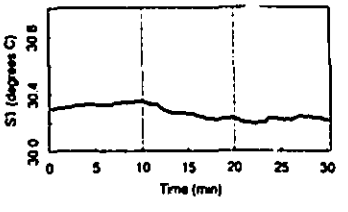


SKIN TEMPERATURE

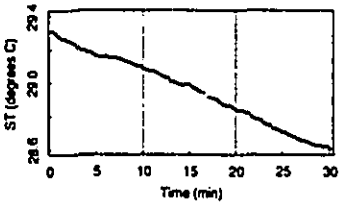
TENS/stump



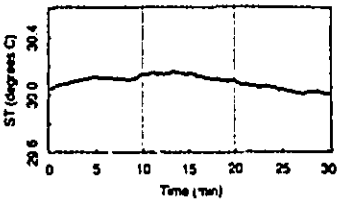
TENS/intact



Placebo/stump



Placebo/intact



Data analysis and criteria for judging statistical significance. In order to assess the global effects of TENS on skin conductance and skin temperature, smoothed values of skin conductance and skin temperature were averaged across each of the three 10-minute periods (B1, BES, B2) and the data were submitted to a 4-way between-within MANOVA using skin conductance levels (SCL) and skin temperature (ST) as dependent variables, Group (No-PL, PLS, PLP) as the independent samples factor, and Session (TENS, placebo), Period (B1, BES, B2), and Limb (stump, intact) as repeated measures factors. Multivariate effects were evaluated for statistical significance using the Pillai-Bartlett criterion as recommended by Olson (1976). Significant multivariate effects were then followed up with univariate ANOVAs computed on both dependent variables. Univariate effects involving the repeated measures factor, Period, were evaluated for significance using conservative degrees of freedom as recommended by Greenhouse and Geisser (1959). When testing interaction effects involving terms with different sources of error, a pooled error term and adjusted degrees of freedom were calculated using Satterthwaite's adjustment as recommended by Winer (1971).

The results of the MANOVA revealed significant main effects for the Period and Limb factors. In addition, significant interactions were found for the Session x Period and Period x Limb effects as well as the three-way interaction of Group x Period x Limb. Table 5 contains a summary of the significant multivariate and univariate effects along with the F ratios, degrees of freedom and levels of significance.

A chi-square test for two-way tables indicated that there was no significant difference with respect to the proportion of subjects receiving the two orders of TENS and placebo in the three groups (χ^2 (2) = 1.60, $p > .05$).

Skin conductance levels and skin temperature. Since this section deals with the effects of TENS vs placebo on skin conductance, skin temperature, and phantom limb intensity, only the Session x Period effect will be evaluated here. The three-way Group x Period x Limb

Table 5. Summary table of the 4-way MANOVA on skin conductance levels and skin temperature showing statistically significant multivariate and univariate effects. Multivariate F-ratios are approximate and based on the Pillai-Bartlett criterion. Univariate effects involving the repeated measurement factor, Period, are evaluated with conservative degrees of freedom as recommended by Greenhouse and Geisser (see text).

Multivariate effect	Dependent variable	F-ratio	df	p
Period		7.49	(4, 100)	.0009
	Skin conductance	11.6	(1, 25)	.002
	Skin temperature	10.9	(1, 25)	.003
Limb		9.7	(2, 24)	.001
	Skin temperature	18.5	(1, 25)	.0002
Session x Period		2.69	(4, 100)	.036
	Skin temperature	3.38	(1, 25)	.08
Period x Limb		5.62	(4, 100)	.0009
	Skin conductance	13.9	(1, 25)	.001
	Skin temperature	4.71	(1, 25)	.04
Group x Period x Limb		5.88	(8, 100)	.0009
	Skin temperature	8.32	(2, 25)	.002

interaction will be explored in Section III which deals with between-group differences in skin conductance and skin temperature.

Although the MANOVA revealed a significant Session x Period interaction (F (4, 100) = 2.69, $p < .05$), univariate ANOVAs were not significant for either skin conductance levels (SCL) or skin temperature (ST) using conservative degrees of freedom. Thus, there is no evidence that mean SCL or ST were differentially affected on the TENS and placebo sessions. Table 6 contains the Session x Period cell means and standard deviations for skin temperature (ST) and skin conductance levels (SCL) summed across the Group and Limb factors. It can be seen that there is little difference between mean values on the two sessions indicating that TENS did not lead to significant changes across periods when compared to the placebo.

Intensity ratings of painful and non-painful phantoms. Figure 6 shows mean intensity levels of phantom limb sensations (Group PLS) and phantom limb pain (Group PLP) across the three periods on the TENS and placebo sessions. Phantom limb intensity was reduced significantly for subjects in Group PLS during electrical stimulation on the TENS session. This was not observed during the placebo control or for subjects in Group PLP. A more detailed presentation of these results are presented below.

In order to statistically evaluate the effects of TENS on the intensity of phantom limb pain and non-painful phantom limb sensations, values of phantom limb intensity (PLI) were averaged across each of the three 10-minute periods and the means submitted to a 3-way between-within ANOVA using Groups (PLS and PLP) as the independent samples factor, and Session (TENS and placebo) and Period (B1, BES, and B2) as the repeated measures factors. Only Groups PLS and PLP are included in this analysis since Group No-PL comprised subjects who reported not having a phantom limb and whose ratings of PLI measured over time have a mean and variance equal to zero. It is important to remember that PLI ratings for the two groups represent different qualities of sensation. Group PLS was monitoring changes

Table 6. Means and standard deviations (SD) of skin temperature and skin conductance levels for the Session x Period interaction.

Skin temperature

	Period					
	B1		BES		B2	
Session	Mean	SD	Mean	SD	Mean	SD
TENS	30.79	1.79	30.70	1.82	30.60	1.84
Placebo	30.72	1.85	30.68	1.91	30.61	1.99

Skin conductance level

	Period					
	B1		BES		B2	
Session	Mean	SD	Mean	SD	Mean	SD
TENS	2.45	1.91	2.59	1.95	2.69	2.04
Placebo	2.34	2.48	2.42	2.37	2.48	2.30

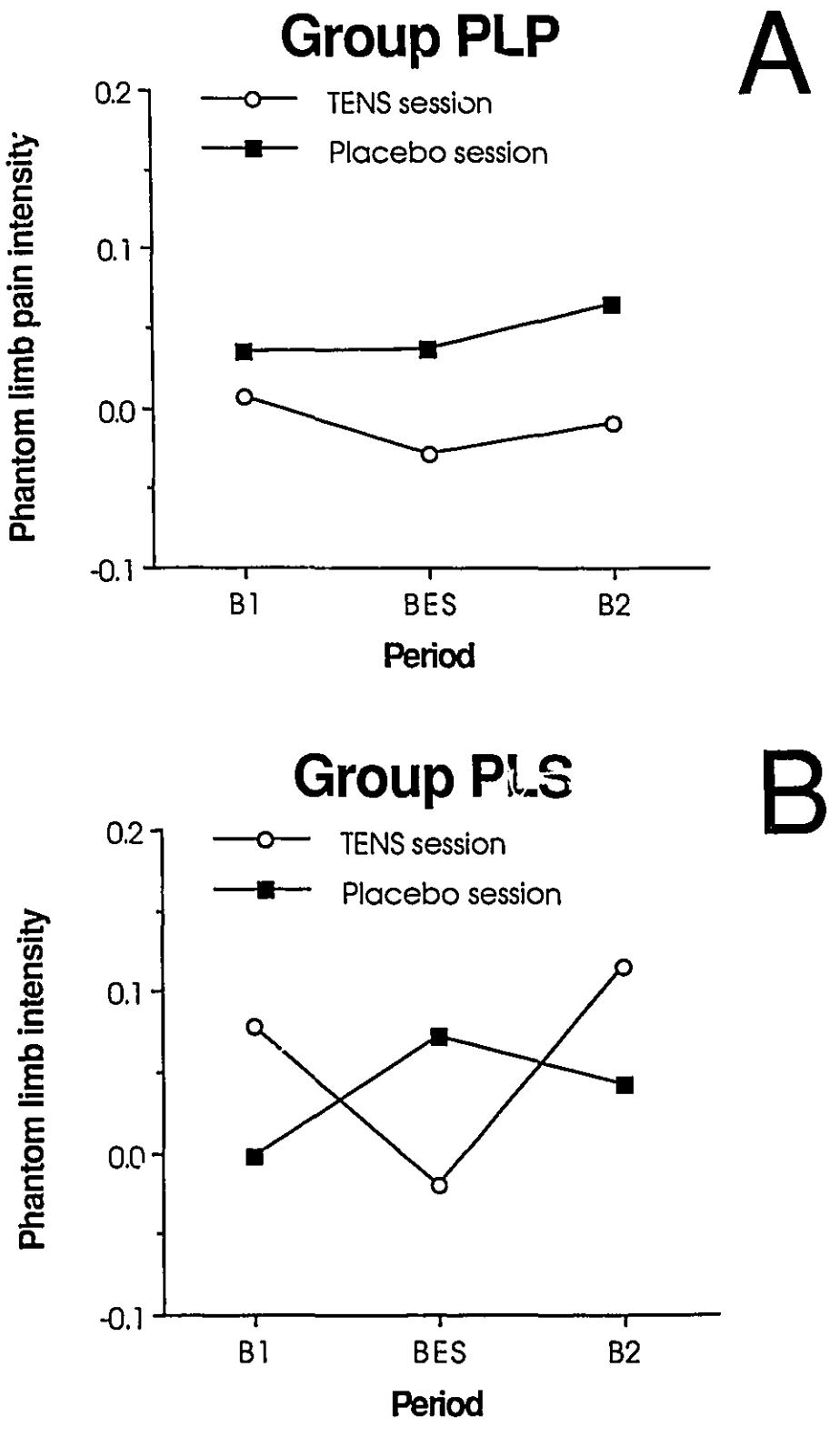


Figure 6. Mean intensity levels of phantom limb pain and non-painful sensations for Groups PLP (A) and PLS (B) respectively, during Periods B1, BES, and B2 on the TENS and placebo sessions.

in the intensity of non-painful paresthesias whereas Group PLP was monitoring pain intensity. As a result, between group comparisons in PLI are not meaningful.

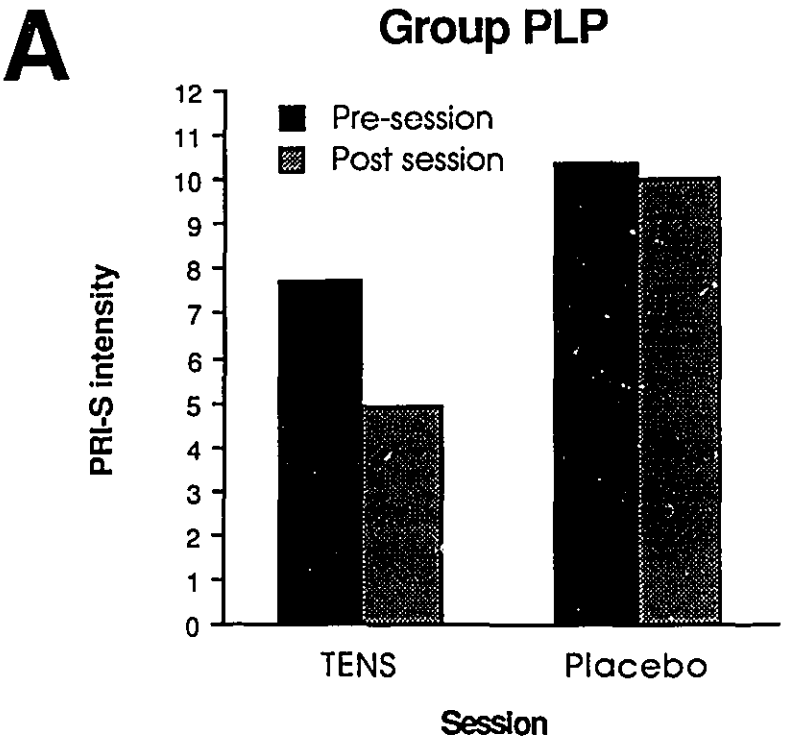
The ANOVA was followed by planned comparisons evaluating the mean change in PLI from B1 to BES and BES to B2 for each group on both sessions. Significant differences were found only for Group PLS on the TENS session. As shown in Panel b of Figure 6, the intensity of non-painful phantom limb sensations was reduced significantly during BES when compared to the initial and final resting baseline levels (F (1, 69) = 4.26, $p < .05$ and F (1, 69) = 7.91, $p < .01$, respectively).

Table 7 shows the various patterns of PLI responding that were observed on the TENS and placebo sessions for subjects in Groups PLP and PLS. Filled circles (•) indicate that at least one change in the intensity of the phantom was recorded by the subject within the period under consideration. Periods characterized by a total absence of reported changes have been left blank (-). It can be seen that the modal pattern for both groups is characterized by changes in phantom intensity in all periods on both sessions. Only three subjects (two in Group PLS and one in Group PLP) reported the presence of a phantom limb that did not change at any time during the course of the experimental procedures. A finer-grained examination of these patterns will be undertaken in Section V.

McGill Pain Questionnaire ratings. The McGill Pain Questionnaire (MPQ) was administered before and after each session in order to assess differential quantitative and qualitative changes in painful and non-painful phantom limb sensations brought about by TENS versus the placebo control. Figures 7 and 8 show that for Group PLP, post-stimulation ratings of the PRI-S and PRI-T were reduced significantly after receiving TENS but not the placebo control. On the other hand, Group PLS showed non-significant changes in the MPQ scores on both sessions. A more detailed presentation and examination of the McGill Pain Questionnaire data follows.

Table 7. Patterns of PLI responding observed during periods B1, BES, and B2 on the TENS and placebo sessions. Also shown are the total number of subjects, as well as the number within Groups PLP and PLS, exhibiting each response pattern. Filled circles (•) indicate that changes in the intensity of phantom limb pain or sensations were reported during the period under consideration. Dashes (-) indicate a period in which no changes were reported.

				Session					
				TENS			Placebo		
	PLP	PLS	Total	B1	BES	B2	B1	BES	B2
	7	3	10	•	•	•	•	•	•
	-	1	1	•	•	•	-	•	•
	1	-	1	•	•	•	•	-	-
	-	1	1	•	•	•	-	-	-
	-	1	1	-	•	•	-	•	•
	1	-	1	-	•	•	-	•	-
	-	1	1	-	•	•	-	-	-
	1	-	1	-	-	•	-	-	-
	1	2	3	-	-	-	-	-	-
Total	11	9	20						



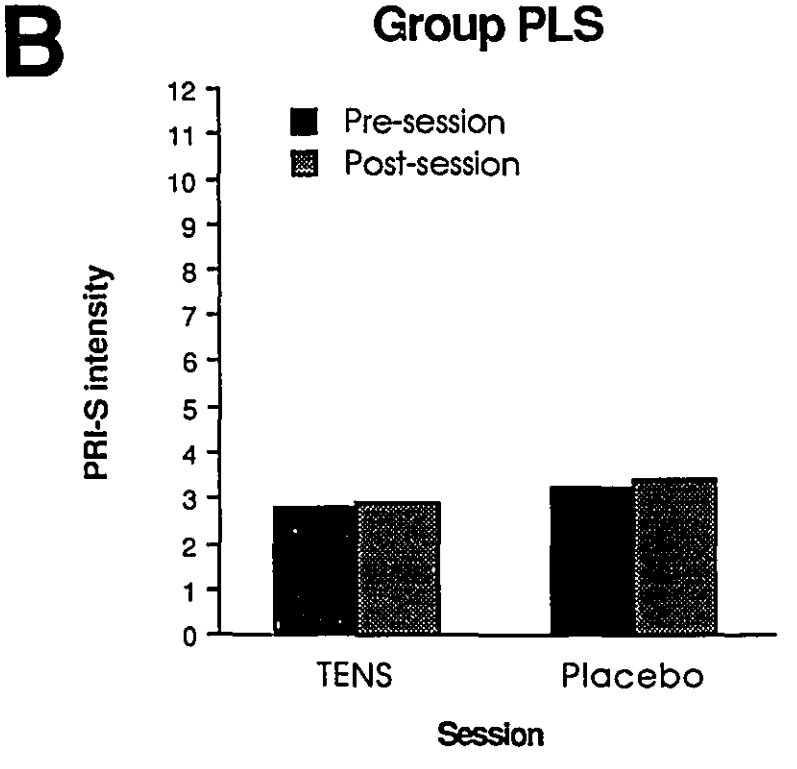
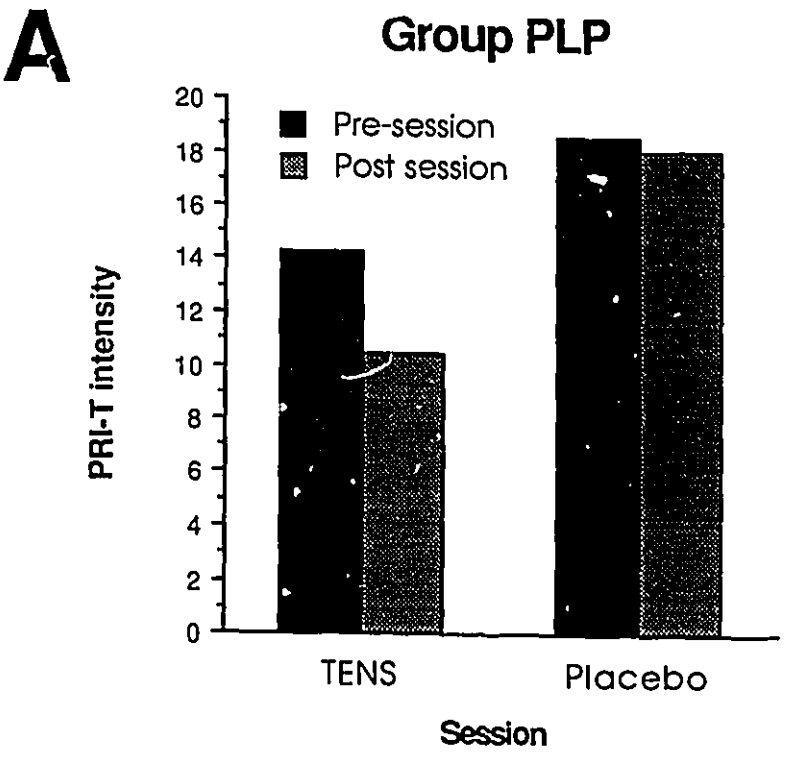


Figure 7. Mean ratings for the sensory class (PRI-S) of the McGill Pain Questionnaire before and after stimulation on the TENS and placebo sessions shown for Groups PLP (A) and PLS (B).



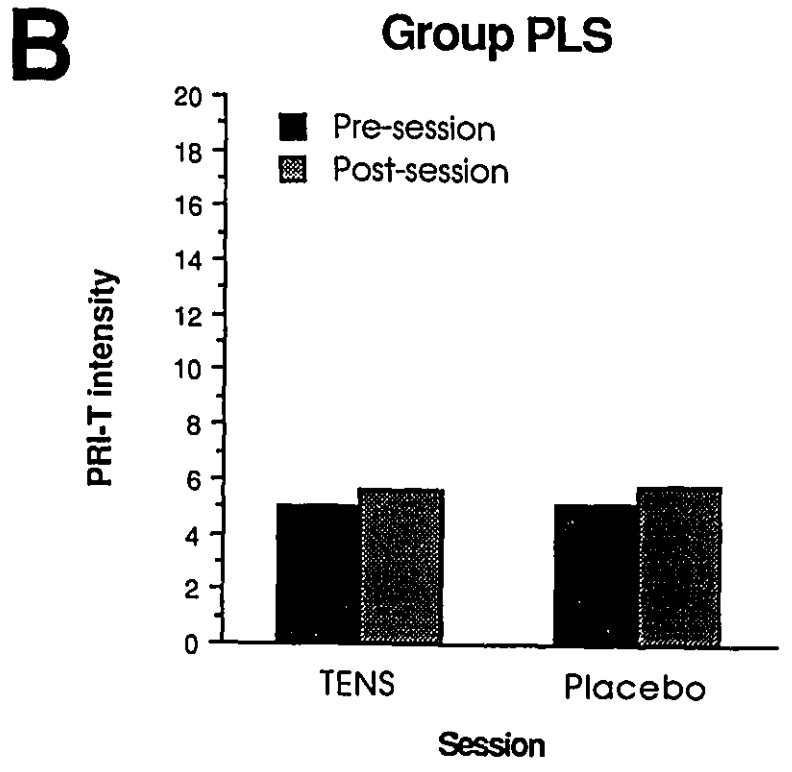


Figure 8. Mean ratings for the total class (PRI-T) of the McGill Pain Questionnaire before and after stimulation on the TENS and placebo sessions shown for Groups PLP (A) and PLS (B).

Pain rating indexes for the sensory (PRI-S), affective (PRI-A), evaluative (PRI-E), miscellaneous (PRI-M), and total (PRI-T) classes were calculated for each MPQ administration and are presented in Table 8 for Groups PLS and PLP. It is important to note that the PRIs reported by Group PLS are not ratings of *pain* intensity but of *non-painful* phantom limb sensations. The PRIs were analyzed by a 3-way between-within MANOVA using the four PRI classes (PRI-S, PRI-A, PRI-E, and PRI-M) as dependent variables, Group (PLS and PLP) as the independent samples factor and Session (TENS and placebo) and Occasion (pre-session and post-session) as repeated measures factors. A separate ANOVA was carried out on the total pain rating index (PRI-T) because of the linear dependence of the PRI-T on the other pain rating indexes (i.e., PRI-S + PRI-A + PRI-E + PRI-M = PRI-T). In addition the PPI scores from Group PLP were submitted to a 2-way repeated measures ANOVA using the same factors as outlined above. Group PLS was excluded from this analysis since it comprised subjects with non-painful phantom sensations whose scores on the PPI were zero. Planned comparisons indicated that post-session ratings of the PRI-S and PRI-T from Group PLP were significantly lower than pre-session scores following TENS (F (1, 34) = 7.48, $p < .01$ and F (1, 31) = 7.09, $p < .01$, respectively). These effects are displayed in Figures 7 and 8 which show the pre- and post-treatment ratings of the PRI-S and PRI-T respectively for Group PLP (panel A) and PLS (panel B) on the TENS and placebo sessions.

A more detailed examination of the MPQ data can be found in Table 9 which contains the descriptors chosen by 30 percent or more of subjects in Groups PLP and PLS at each administration. For ease of reading, table entries have been omitted for a given session when both pre- and post-intervention administrations yielded values below 30 percent. Several points are noteworthy. The most salient feature is the greater number of descriptors endorsed by more subjects in Group PLP on both sessions, consistent with the higher PRI-Ts for this group. Second, a major difference between the groups can be found in the class of words used to describe their phantom limbs. Not one descriptor from any of the affective categories

Table 8. McGill Pain Questionnaire present pain intensity (PPI) scores and pain rating indexes (PRI) on the TENS and placebo sessions showing pre- and post-session sensory (PRI-S), affective (PRI-A), evaluative (PRI-E), miscellaneous (PRI-M), and total (PRI-T) classes for Groups PLP and PLS. Note that unlike Group PLP, the PRIs from Group PLS do not represent pain intensity but instead, intensity of non-painful phantom limb sensations. For this reason all PPI ratings from Group PLS have a value of zero corresponding to the MPQ descriptor "no pain".

		Group							
		Phantom limb pain (PLP) (n = 11)				Phantom limb sensation (PLS) (n = 9)			
		TENS		Placebo		TENS		Placebo	
MPQ class	Test	Mean	SD	Mean	SD	Mean	SD	Mean	SD
PRI-S	Pre	7.7	5.7	10.4	7.2	2.8	2.7	3.2	4.7
	Post	4.9	3.4	10.0	7.6	2.9	2.6	3.4	4.4
PRI-A	Pre	1.8	1.8	2.6	3.4	0.1	0.3	0.0	0.0
	Post	1.2	1.6	2.3	2.5	0.0	0.0	0.1	0.3
PRI-E	Pre	1.2	0.8	1.7	1.3	0.4	0.7	0.6	0.5
	Post	1.1	1.0	1.7	1.3	0.2	0.4	0.7	0.5
PRI-M	Pre	3.5	2.9	3.8	3.1	1.7	1.5	1.3	1.6
	Post	3.2	1.8	4.1	2.5	2.4	2.4	1.6	1.3
PRI-T	Pre	14.3	9.0	18.5	12.9	5.0	4.0	5.1	6.0
	Post	10.5	6.1	18.1	13.3	5.6	4.6	5.8	5.6
PPI	Pre	2.0	1.0	2.1	0.8	0.0	0.0	0.0	0.0
	Post	1.5	0.9	1.9	0.9	0.0	0.0	0.0	0.0

Table 9. Descriptors of the McGill Pain Questionnaire chosen by 30% or more (bold type) of subjects in Groups PLP and PLS before and after receiving TENS and placebo. Note that unlike Group PLP, the MPQ adjectives chosen by subjects in Group PLS do not describe phantom limb pain but instead, non-painful phantom limb sensations.

		Group							
		Phantom limb pain (PLP) (n = 11)				Phantom limb sensation (PLS) (n = 9)			
		TENS		Placebo		TENS		Placebo	
MPQ Class	Descriptors	Pre	Post	Pre	Post	Pre	Post	Pre	Post
Sensory	beating	–	–	27.3	**36.4**	–	–	–	–
	pricking	**45.5**	27.3	**45.5**	**36.4**	**33.3**	**33.3**	–	–
	cramping	–	–	**36.4**	18.2	–	–	–	–
	hot	–	–	**36.4**	**36.4**	–	–	–	–
	tingling	**72.7**	**36.4**	**63.6**	**45.5**	**55.6**	**55.6**	**66.7**	**66.7**
Affective	tiring	**45.5**	**54.5**	**36.4**	**45.5**	–	–	–	–
	sickening	–	–	**36.4**	27.3	–	–	–	–
	punishing	–	–	**36.4**	9.1	–	–	–	–
	wretched	–	–	**36.4**	27.3	–	–	–	–
Evaluative	annoying	**72.7**	**72.7**	**54.5**	**45.5**	–	–	**66.7**	**66.7**
Miscellaneous	squeezing	**36.4**	18.2	9.1	**36.4**	–	–	–	–
	numb	27.3	**45.5**	**63.6**	27.3	**33.3**	**33.3**	**33.3**	**33.3**
	nagging	27.3	**36.4**	**45.5**	**45.5**	–	–	–	–
PPI	discomforting	**63.6**	**45.5**	**36.4**	**45.4**	–	–	–	–
	distressing	–	–	**36.4**	27.3	–	–	–	–

is endorsed by 30 percent or more of subjects in Group PLS whereas at least one third of Group PLP use some of these adjectives on both sessions. In addition, Group PLS shows remarkable consistency in their choice of descriptors within as well as between sessions, almost exclusively choosing descriptors from the class of sensory descriptors. Third, pre- versus post- stimulation changes for Group PLP on the TENS session are most evident for adjectives which are frequently used to describe the "normal" non-painful phantom (e.g., "pricking", "tingling", and "numb"). Fourth, there is a consistency in the choice of descriptors across groups. Every descriptor chosen by 30 percent or more of subjects in Group PLS was also chosen by 30 percent or more subjects in Group PLP, although there are other descriptors the latter group also endorses with greater frequency. This indicates that the phantom limb experiences of the two groups have in common a paresthetic quality although painful phantoms consist of more than this shared component.

Mood ratings. Ratings from the MRS-T, MRS-A, SRS, and STAI-S taken on each session before and after stimulation are shown in Table 10 for the three groups. These variables were entered as dependent variables into a 3-way MANOVA (Group x Session x Occasion) as described above for the MPQ classes. There were no significant main effects or interactions, indicating that mean mood ratings were comparable between groups and remained virtually unchanged across sessions and test occasions. Thus, the post-TENS decrease in the MPQ PRI-S and PRI-T observed in Group PLP can not be attributed to alterations in mood or mood-related states such as anxiety and sleepiness.

Pressure sensitivity thresholds. Table 11 contains the group means and standard deviations for the pressure sensitivity thresholds at both limbs taken before and after stimulation on the two sessions. These data were submitted to a 4-way between-within ANOVA (Group x Session x Limb x Occasion). There were no significant main effects or interactions.

Table 10. Group means and standard deviations (SD) for the Spielberger State Anxiety Inventory (STAI-S), Mood Rating Scale-Alertness (MRS-A), -Tranquility (MRS-T), and Sleepiness Rating Scale (SRS) obtained pre- and post-stimulation on the TENS and placebo sessions.

			Group					
			Phantom limb pain (PLP) (n = 11)		Phantom limb sensation (PLS) (n = 9)		No phantom limb (No-PL) (n = 8)	
Session	Variable	Test	Mean	SD	Mean	SD	Mean	SD
TENS	STAI-S	Pre	32.9	8.0	28.7	10.4	27.9	9.1
		Post	32.4	7.1	27.4	9.0	28.9	10.2
	MRS-A	Pre	7.9	1.4	8.7	2.0	8.7	1.5
		Post	7.0	2.0	8.4	2.0	8.9	1.5
	MRS-T	Pre	7.7	1.4	8.6	1.3	7.7	1.9
		Post	8.0	1.6	9.0	1.0	9.0	1.4
	SRS	Pre	2.5	0.6	2.4	0.7	2.3	0.5
		Post	3.5	1.8	3.0	1.7	2.4	0.7
Placebo	STAI-S	Pre	31.9	9.5	29.8	9.9	29.1	10.9
		Post	32.1	7.6	27.4	7.8	29.5	9.7
	MRS-A	Pre	8.0	1.5	8.0	2.4	8.8	1.8
		Post	6.8	1.9	8.4	2.1	8.9	1.7
	MRS-T	Pre	7.7	1.1	7.6	2.6	8.7	1.9
		Post	7.4	1.5	8.5	1.6	8.4	2.1
	SRS	Pre	2.6	0.8	2.4	0.9	2.5	0.8
		Post	3.5	0.9	2.6	0.9	2.4	0.7

Table 11. Group means and standard deviations (SD) for pressure sensitivity thresholds taken at the stump and intact limb before and after stimulation on TENS and placebo sessions. Table entries represent the logarithm of the force required to bend filaments of varying diameters when applied to the skin, hence the lower the threshold, the greater is the sensitivity of the skin.

			Group					
			Phantom limb pain (PLP) (n = 11)		Phantom limb sensation (PLS) (n = 8*)		No phantom limb (No-PL) (n = 8)	
Session	Limb	Test	Mean	SD	Mean	SD	Mean	SD
TENS	Stump	Pre	3.940	0.627	3.799	1.047	4.182	0.470
		Post	3.821	0.946	3.802	0.887	4.194	0.474
	Intact	Pre	3.852	0.489	4.087	0.858	4.102	0.468
		Post	3.647	0.685	3.712	0.766	4.161	0.273
Placebo	Stump	Pre	3.926	0.663	3.976	1.127	4.259	0.794
		Post	3.983	0.632	3.856	1.247	4.297	0.660
	Intact	Pre	3.403	0.703	4.077	0.814	4 237	0.279
		Post	3.638	0.584	3.727	1.089	4.106	0.196

* Pressure sensitivity thresholds are not available for one subject

Correlations between phantom intensity and psychophysiological variables. The relationship between (a) changes in skin conductance and skin temperature measured at both limbs and (b) the intensity of phantom pain/sensations (PLI) across periods was assessed for each subject. Pearson correlation coefficients for each subject are contained in Table 12 for skin conductance and Table 13 for skin temperature. Because skin temperature data was, in general, highly negatively correlated with skin conductance, it will not be included in the following presentation since it provides little in the way of unique information that is not also available from an examination of skin conductance. Furthermore, examinations of the raw plots of skin temperature against phantom limb intensity seemed to indicate that, for most subjects, skin temperature lacked the sensitivity apparent in the measure of skin conductance.

Significant correlations between changes in PLI and SC indicate that in general, increases and decreases in the intensity of phantom limb sensations/pain are accompanied by concomitant changes in skin conductance. Table 12 shows the correlation coefficients computed between phantom limb intensity and SC measured at the stump and intact limb on the two sessions for each subject in Group PLP and PLS. For ease of reading in the table all non-significant (i.e., $p > .05$) correlations have been replaced with empty cells.

The most salient feature of these data is that the majority of subjects in Group PLS show significant positive correlations between the intensity of phantom limb sensations and skin conductance measured on both limbs whereas this is not the case for Group PLP. A chi-square analysis comparing the frequencies of significant positive, negative, and non-significant correlations between the groups was significant for the stump (χ^2 (2) = 7.48, $p < .01$) and intact limb (χ^2 (2) = 9.39, $p < .01$). The main source of these effects was the preponderance of significant positive correlations in Group PLS as compared to Group PLP (χ^2 (1) = 7.49, for the stump and χ^2 (1) = 6.24, for the intact limb, both $p < .05$ using Yate's correction for continuity). This indicates that there is a significant positive relationship between SC on both limbs and PLI for proportionally more subjects in Group PLS than PLP. In fact, the table shows that every subject in Group PLS shows at least one

Table 12. Pearson correlation coefficients describing the relationship between phantom limb intensity (PLI) and skin conductance measured at the stump and intact limb during TENS and placebo sessions for each subject in Groups PLS and PLP. Note that for Group PLS, PLI refers to the intensity of non-painful phantom limb sensations, and for Group PLP, the intensity of phantom limb pain. All correlation coefficients are based on a sample size of n = 90 points and are significant at the .05 level or less. All non-significant correlations have been omitted and replaced with empty cells (--). N/A indicates the subject did not report any changes in the intensity of phantom sensations/pain on the session in question and hence the calculation of a correlation coefficient is not applicable.

	Group PLS			
	TENS session		Placebo session	
Case number	Stump	Intact limb	Stump	Intact limb
E02	.72	.54	.51	.58
E05	—	—	—	—
E08	.95	.92	n/a	n/a
E12	.84	.82	.38	.39
E23	.31	.29	-.42	—
E24	—	.64	n/a	n/a
E26	.72	.71	.28	.39

	Group PLP			
	TENS session		Placebo session	
Case number	Stump	Intact limb	Stump	Intact limb
E04	—	—	—	-.36
E06	—	—	.35	.44
E07	—	—	—	—
E10	.50	.49	.28	—
E11	-.38	—	-.32	—
E14	—	—	—	.29
E15	—	—	—	-.42
E16	—	-.58	n/a	n/a
E20	-.37	-.41	.56	.61
E28	—	—	—	—

Table 13. Pearson correlation coefficients describing the relationship between phantom limb intensity (PLI) and skin temperature measured at the stump and intact limb during TENS and placebo sessions for each subject in Group PLS and PLP. Note that for Group PLS, PLI refers to the intensity of non-painful phantom limb sensations and for Group PLP, to the intensity of phantom limb pain. All correlation coefficients are based on a sample size of n = 90 points and are significant at the .05 level or less. All non-significant correlations have been omitted and replaced with empty cells (---). N/A indicates the subject did not report any changes in the intensity of phantom sensations/pain on the session in question and hence the calculation of a correlation coefficient is not applicable.

	Group PLS			
	TENS session		Placebo session	
Case number	Stump	Intact limb	Stump	Intact limb
E02	-.38	.35	-.58	---
E05	---	---	---	---
E08	-.83	-.87	n/a	n/a
E12	-.81	---	---	.62
E23	.56	---	---	---
E24	---	.34	n/a	n/a
E26	-.61	-.75	---	-.34

	Group PLP			
	TENS session		Placebo session	
Case number	Stump	Intact limb	Stump	Intact limb
E04	---	-.32	.30	.48
E06	-.48	.35	---	.48
E07	---	---	-.33	---
E10	-.28	-.43	-.33	---
E11	.45	---	---	---
E14	-.36	---	-.30	-.45
E15	---	---	---	---
E16	.92	-.35	n/a	n/a
E20	.45	.42	-.37	---
E28	.47	---	.36	.82

significant correlation between these variables. Furthermore the relationship appears to be independent of whether TENS is delivered since skin conductance correlates with PLI on both TENS and placebo sessions although the relationship on the latter session is not as striking since fewer subjects reported changes in phantom limb intensity on the placebo session, thus precluding calculation of a correlation coefficient.

It was not possible to examine the temporal relationship between the two variables to determine whether changes in one preceded or followed changes in the other due to the low sampling rate of one measurement per limb every other 10 seconds. This constraint fixed the minimum possible time lag between consecutive measurements taken from the same limb at 20 seconds, clearly far too wide a time frame to capture the temporal relationship between the variables if one existed. An attempt was made to isolate segments or windows of time within which changes in PLI had occurred, and to examine the behaviour of stump skin conductance within this same time frame. Unfortunately the useable data from any given 10 sec period was limited to approximately 3-sec intervals which seriously reduced the feasibility of further data exploration along these lines. Thus, the issue of whether changes in PLI preceded or followed changes in stump skin conductance cannot be addressed.

Between-limb correlations of skin conductance and skin temperature. Table 14 shows the Pearson correlation coefficients, calculated for both dependent variables, between the stump and the intact limb for each subject in the three groups on the TENS and placebo sessions. As can be seen correlations between the two limbs are, in general, extremely high for skin conductance and skin temperature suggesting that the processes responsible for the fluctuations in both limbs over time are governed by a common mechanism and are not specific to the stump.

III. Between-group differences in stump skin conductance levels and skin temperature

This section is concerned with evaluating (a) the claim that stump skin conductance levels and skin temperature are altered relative to the intact limb for subjects with phantom

Table 14. Pearson correlation coefficients between stump and intact limb measurements of skin conductance and skin temperature on the TENS and placebo sessions displayed for each subject in the three groups. All correlation coefficients are based on a samle size of n = 90 points and are significant at the .01 level or less. All non-significant correlations have been omitted and replaced with empty cells (--).

	Group PLP					Group PLS					Group No-PL			
	Skin conductance		*Skin temperature*			*Skin conductance*		*Skin temperature*			*Skin conductance*		*Skin temperature*	
Case	TENS	Placebo	TENS	Placebo	Case	TENS	Placebo	TENS	Placebo	Case	TENS	Placebo	TENS	Placebo
E04	.77	.58	.52	.91	E01	.84	.97	.93	.36	E03	.97	.98	-.28	-
E06	.98	.96	-	.86	E02	.97	.83	.47	.64	E13	.99	.93	.80	.63
E07	-.80	-.47	.99	.95	E05	.28	.43	-.71	.48	E17	.98	1.00	.91	-.71
E10	.98	.96	-	.38	E08	.98	.96	.89	.97	E18	-.85	-.80	.98	.95
E11	.90	.99	.39	.88	E09	.90	.62	-.85	.96	E19	.99	.99	.83	-.52
E14	-	.98	.84	.84	E12	.98	.99	-	-	E21	.99	.94	.89	.91
E15	.98	.91	.78	1.00	E23	.89	.68	.64	.65	E22	.79	.99	.99	.44
E16	-.57	.22	-	.80	E24	.38	-	-	.91	E27	-.90	.72	-.54	-.96
E20	.78	.81	.88	.51	E26	.54	.98	.91	.54					
E25	.84	.98	-.50	-.49										
E28	.98	.91	.74	.38										

limb pain, (b) the relationship between McGill Pain Questionnaire scores and psychophysiological measures taken at the stump and intact limb, and (c) the significant 2-way Period x Limb interaction and the 3-way Group x Period x Limb interaction reported in Section II.

Tests of planned comparisons. Table 15 shows mean skin conductance levels and skin temperature at the stump and intact limb averaged across periods and sessions for the three groups. It was hypothesized that the stump would register higher skin conductance levels and lower skin temperature (a) than the contralateral intact limb for subjects in Group PLP only, and (b) in Group PLP compared to Groups PLS and No-PL combined.

The first hypothesis was supported for skin temperature but not skin conductance levels. Planned comparisons revealed that the stump was significantly lower in temperature than the intact limb for Group PLP only (F (1, 25) = 8.48, $p < .007$). Although the stump was also cooler than the intact limb for Group PLS and No-PL, the temperature difference was not enough to reach conventional levels of significance (F (1, 25) = 4.02, and F (1, 25) = 3.29, $.05 < p < .10$, for the two groups, respectively). Differences between skin conductance levels at the stump and intact limb were not significant for any group.

With regard to the second hypothesis, a comparison of the joint means of Groups PLS and No-PL with Group PLP did not reach significance for stump skin conductance (F (1, 33) = 0.73, $p > .05$) or stump skin temperature (F (1, 39) = 0.27, $p > .05$).

The suggestion that fluctuations in stump skin conductance and temperature are accompanied by changes in the perceived intensity of phantom limb sensations implies that subjects reporting the presence of a phantom limb should display greater variability in stump SC and ST compared to subjects who reported no phantom limb at all. This can be seen by examining and comparing the plots of mean skin conductance and skin temperature for each group shown in Figures 3, 4, and 5. The comparatively low variability for Group No-PL is most apparent for stump skin conductance shown in the two panels labelled TENS/stump and

Table 15. Group means and standard deviations (SD) for skin conductance and skin temperature at both limbs summed across Periods and Sessions.

	Group					
	Phantom limb pain (PLP) (n = 11)		Phantom limb sensation (PLS) (n = 9)		No phantom limb (No-PL) (n = 8)	
	Mean	SD	Mean	SD	Mean	SD
Skin conductance						
Stump	2.99	3.96	2.93	1.38	1.69	0.79
Intact limb	2.75	2.11	3.04	1.28	1.15	0.44
Skin temperature						
Stump	30.18	2.31	30.57	1.52	29.10	1.62
Intact limb	31.82	1.57	31.86	1.17	30.18	2.27

Placebo/stump. Compared to Groups PLS and PLP, Group No-PL shows little fluctuation in SC over time. This is especially evident when one considers that the scale (in μmhos) for Group No-PL is one-half that of Group PLP, and one quarter that of Group PLS.

The hypothesis that the variability in SC and ST is lower for subjects who do not report a phantom limb (Group No-PL) than those who do (Groups PLP and PLS) was tested by examining between group differences in within subject variability of SC and ST across sessions and periods. Within subject variances in SC and ST were calculated for the three periods (B1, BES, B2) at both levels of session and limb. These values were submitted to a 4-way between-within MANOVA (Group x Session x Period x Limb) with the expectation that the Group x Limb interaction would reach significance. Figure 9 shows the mean within subject variance in stump skin conductance and skin temperature for the three groups of subjects. Although the variability appears to be markedly lower for stump skin conductance in Group No-PL, the multivariate Group x Limb interaction term did not reach significance (F (4, 50) = 1.08, $p > .05$).

Relationship between pain ratings and psychophysiological variables -- Between-subject correlations. Table 16 shows the Pearson correlation coefficients describing the relationship between (a) measurements of ST, SCL, and PST taken at the stump, and the intact limb, and (b) the MPQ pain rating indexes and PPI for all subjects on the two sessions. Also shown is the relationship between the stump-intact limb difference scores and the MPQ data. It can be seen that only a few significant correlations were found between phantom limb intensity and peripheral measures of skin conductance and skin temperature. It does not appear that large differences between the limbs are associated with high MPQ ratings and small differences with low ratings. These results indicate that there is no simple linear relationship between the magnitude of stump-intact limb differences and phantom limb intensity. An elaboration of these results is presented below.

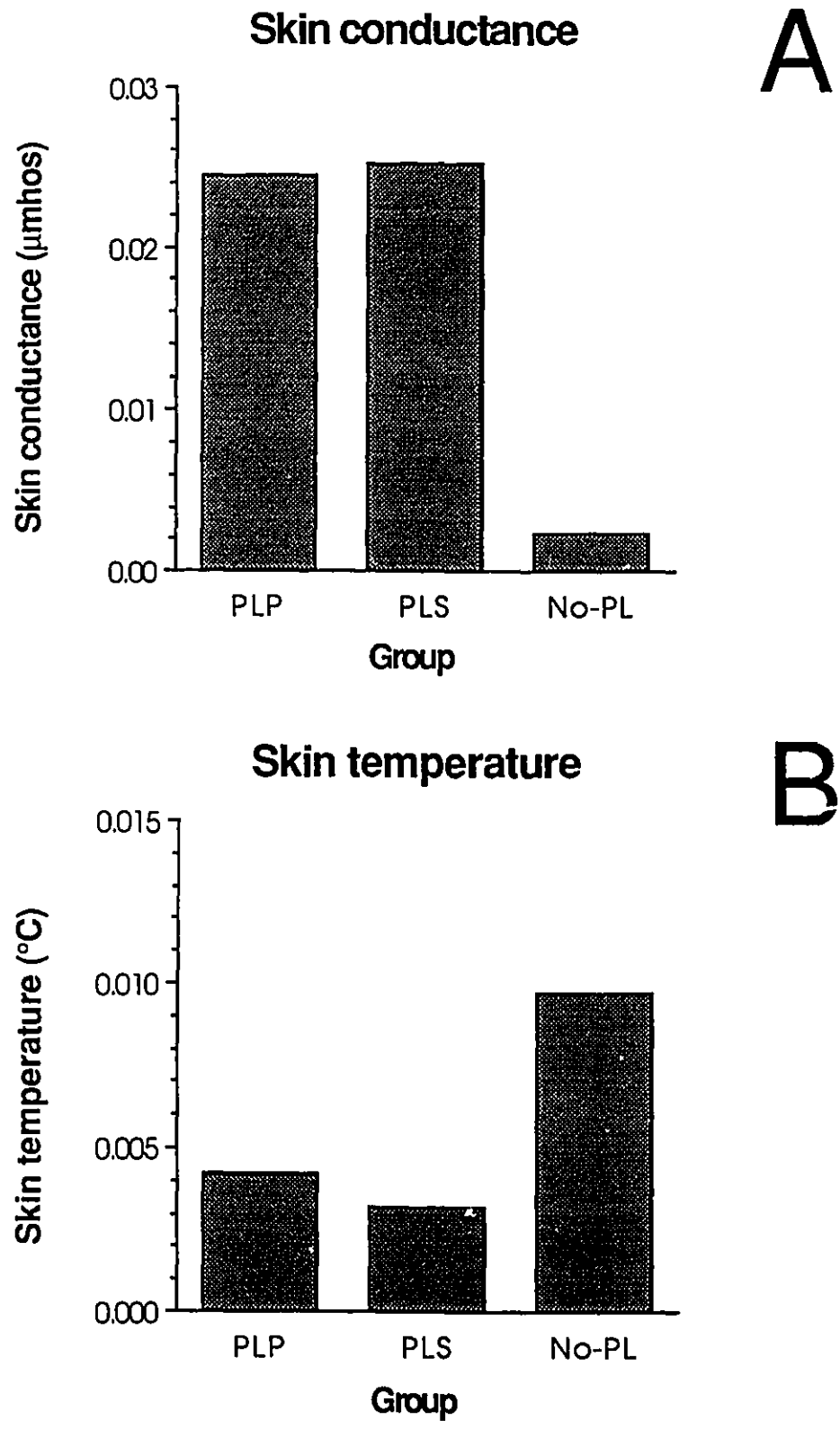


Figure 9. Mean within-subject variance of stump skin conductance (A) and stump skin temperature (B) for the three groups summed across Session and Period factors.

Table 16. Pearson correlation coefficients describing the relationship between between (a) MPQ pain rating indexes (PRI) and (b) skin temperature, skin conductance, and pressure sensitivity thresholds at the stump and intact limb on the TENS and placebo sessions. All correlation coefficients are based on the total number of subjects in the three groups (n = 28).

	TENS session					
	PPI	PRI-S	PRI-A	PRI-E	PRI-M	PRI-T
Skin temperature						
Stump	.16	-.24	-.01	.11	.03	-.12
Intact limb	.24	.14	.15	.27	.20	.19
Difference	-.06	-.38*	-.16	-.15	-.16	-.31
Skin conductance						
Stump	.19	.19	-.03	-.03	-.14	.06
Intact limb	.25	.27	.11	.13	.21	.25
Difference	-.01	-.04	-.17	-.19	-.42*	-.19
Skin sensitivity						
Stump	-.06	-.08	-.07	.07	-.18	-.10
Intact limb	-.17	-.30	-.20	-.04	-.13	-.25
Difference	.10	.21	.12	.14	-.09	.13

	Placebo session					
	PPI	PRI-S	PRI-A	PRI-E	PRI-M	PRI-T
Skin temperature						
Stump	.09	.06	-.12	-.02	.00	.01
Intact limb	.01	.16	.04	.04	.00	.11
Difference	.09	-.10	-.19	-.06	.00	-.10
Skin conductance						
Stump	.45**	.23	.30	-.01	.13	.22
Intact limb	.44**	.22	.20	.03	.17	.21
Difference	.38*	.20	.35	-.05	.06	.20
Skin sensitivity						
Stump	-.20	-.26	-.16	-.10	-.25	-.25
Intact limb	-.22	-.33	-.06	-.34	-.26	-.29
Difference	.01	.05	-.08	.19	.01	.03

** p<.01, *p<.05, both two-tailed

In order to examine the relationship between the psychophysiological variables and the presence or absence phantom limb pain, mean values of skin temperature and skin conductance were calculated for each subject based on the first ten-minutes (i.e., Period B1) from each session and correlated with the corresponding pre-session pain rating indexes (PRI) from the McGill Pain Questionnaire (MPQ). Examination of data was limited to the baseline period on each session in order to avoid the possible confounding effects of TENS (and placebo), and to ensure that the measurements taken from the two sets of variables to be correlated represented estimates from the same time frame. Stump-intact limb difference scores were obtained by subtracting measurements taken at the intact limb from those at the stump. Negative difference scores indicate that relative to the intact limb the stump is lower in temperature, lower in skin conductance, and more sensitive to applied pressure.

Table 16 shows that with the exception of four significant correlation coefficients, there does not appear to be a linear relationship between phantom limb intensity and skin temperature or skin conductance levels at either limb. Significant correlations involving the PPI and PRI-M should be interpreted cautiously since both variables are positively skewed and have extremely small ranges. The significant negative correlation (-.38) on the TENS session between the PRI-S and the stump-intact limb temperature difference is suggestive, and indicates that the cooler the stump is relative to the intact limb, the higher is the PRI-S. However, since this relationship was not also observed on the placebo session it is best to interpret the significance of the correlation coefficient cautiously.

Within-subject correlations. Six subjects reported PLP intensities (as measured by the PPI) that differed from Session 1 to Session 2. This permitted a within-subject examination of their stump-intact limb difference scores across sessions to determine whether higher PLP PPIs were associated with larger (absolute) difference scores. Table 17 contains these stump-intact difference scores for the three dependent variables on both sessions as well as the PPI score corresponding to the intensity of their phantom limb pain. Although there are only six

Table 17. Stump-intact limb difference scores for temperature, skin conductance and pressure sensitivity for the six subjects who reported different levels of phantom limb pain intensity on Sessions 1 and 2. Negative difference scores indicate that relative to the intact limb the stump is lower in temperature, lower in skin conductance, and more sensitive to applied pressure.

		Stump-Intact limb difference scores			
Subject		PLP PPI	Temperature (°C)	Skin conductance (μmhos)	Pressure sensitivity (log force)
E04	Session 1	3	-0.58	-1.67	3.81
	Session 2	2	-1.40	-1.02	1.57
E10	Session 1	0	-1.40	0.38	-0.47
	Session 2	1	-0.98	0.10	0.86
E11	Session 1	4	0.13	-0.30	-0.09
	Session 2	2	0.09	-0.83	0
E14	Session 1	3	0.39	0.15	0.62
	Session 2	2	-1.69	0.99	0
E16*	Session 1	2	-2.65	-2.68	0.25
	Session 2	3	-2.79	-0.25	0.62
E20	Session 1	2	0.23	0.86	-0.86
	Session 2	1	0.17	-0.20	0.62

*This subject also had stump pain of a burning quality on both sessions.

subjects, it is apparent that there is little relationship between pain intensity and the stump-intact limb difference scores.

Three subjects reported having stump pain and one stump tingling on both sessions. Table 18 shows the stump-intact limb differences score for these subjects as well as their PLP PPI scores. Once again, the small sample size suggests a cautious interpretation of these data, but for the three subjects shown in the table, there is not a strong relationship between the intensity of stump pain/sensations and the magnitude of the differences between the two limbs. These data, taken together, do not appear to support the hypothesis that the magnitude of the difference in sympathetic nervous system activity between the stump and intact limb is related to phantom limb or stump intensity.

Differences over time. The results in this section are concerned with evaluating differences in the pattern of mean skin temperature and skin conductance levels over time (i.e., across periods). Two statistically significant results emerged:

1. Overall, the pattern of skin conductance levels at the stump differed from the intact limb. Whereas stump SCL increased significantly from Period B1 to Period B2, for the intact limb mean levels were significantly greater during BES as compared to B1 and during B2 as compared to BES. This pattern would suggest that the rate of increase also differs for the two limbs.

2. Mean stump skin temperature decreased significantly across periods for Group No-PL but not for Groups PLP or PLS. Once again, this suggests that there is a difference in the slope or rate of temperature loss at the stump among the three groups. These results are presented in more detail below.

The 4-way MANOVA reported in Section II revealed a significant multivariate 2-way interaction of Period x Limb (see Table 5). This effect was explored at the univariate level for each dependent variable. The Period x Limb interaction was significant for the ANOVA on skin conductance levels (F (1, 25) = 13.9, $p < .001$) and skin temperature

Table 18. Stump-intact limb difference scores for skin temperature, skin conductance and pressure sensitivity thresholds for the four subjects who reported stump pain or stump tingling on Sessions 1 and 2. PPI scores corresponds to intensity of phantom limb pain. Subject E02 had a painless phantom on both sessions while subjects E18 and E22 had no phantom limb at all. Negative difference scores indicate that relative to the intact limb the stump is lower in temperature, lower in skin conductance, and more sensitive to applied pressure.

				Stump-Intact limb difference scores		
Subject		PLP PPI	Stump pain/ sensation	Temperature (°C)	Skin conductance (µmhos)	Pressure sensitivity (log force)
E02	Session 1	0	tingling	-3.21	-0.24	3.09
	Session 2	0	tingling	-3.63	-1.52	0.42
E16	Session 1	2	burning	-2.65	-2.68	0.25
	Session 2	3	burning	-2.79	-0.25	0.62
E18	Session 1	0	burning	0.31	0.79	0.23
	Session 2	0	burning	-1.06	1.63	-0.14
E22	Session 1	0	burning	-0.05	1.15	0.25
	Session 2	0	burning	-1.67	2.62	0.62

(F (1, 25) = 4.71, $p < .04$). An examination of the effect for skin temperature will be undertaken later, in the context of the higher-order interaction involving the Group factor.

Figure 10 shows the Period x Limb cell means for skin conductance levels. Although the stump displayed greater skin conductance levels than the intact limb at all periods, simple main effects indicated that these differences were not statistically significant (all $p > .05$). However, when examined across periods for each limb, simple main effects were significant for the stump and the intact limb (F (1, 25) = 4.59, $p < .04$ and F (1, 25) = 16.9, $p < .0004$, respectively). The pattern of differences across periods was evaluated using the Newman Keuls multiple comparison procedure. Skin conductance levels at the stump increased significantly ($p < .01$) from B1 to B2. For the intact limb all pairs of means were significantly different ($.05 < p < .01$) indicating that skin conductance was greater during Period BES when compared to B1, and during B2 when compared to BES.

Results from the 4-way MANOVA reported in Section II revealed a significant 3-way interaction for the Group x Period x Limb effect. Subsequent univariate ANOVAs indicated that the significance was largely due to differences among the means for skin temperature (F (2, 25) = 8.32, $p < .002$). Figure 11 shows a plot of the 3-way interaction. The simple interaction of the Group x Limb effect was not significant at any of the three levels of Period.

The simple Group x Period interaction effect was significant for the stump (F (2, 25) = 5.28, $p < .01$), but not the intact limb (F (2, 25) = 0.10, $p > .05$) indicating that the pattern of stump skin temperature over the three periods was different for the three groups. This interaction was explored by computing simple simple main effects across the Period cell means measured at the stump for each level of Group. Significant F ratios were found for Group No-PL (F (1, 25) = 20.4, $p < .0001$) but not Group PLS (F (1, 25) = 2.85, $p > .05$) or Group PLP (F (1, 25) = 0.99, $p > .05$). Using the Newman Keuls procedure for post hoc hypothesis testing all pairs of means differed significantly ($p < .01$) for Group No-PL. Together these results indicate that stump skin

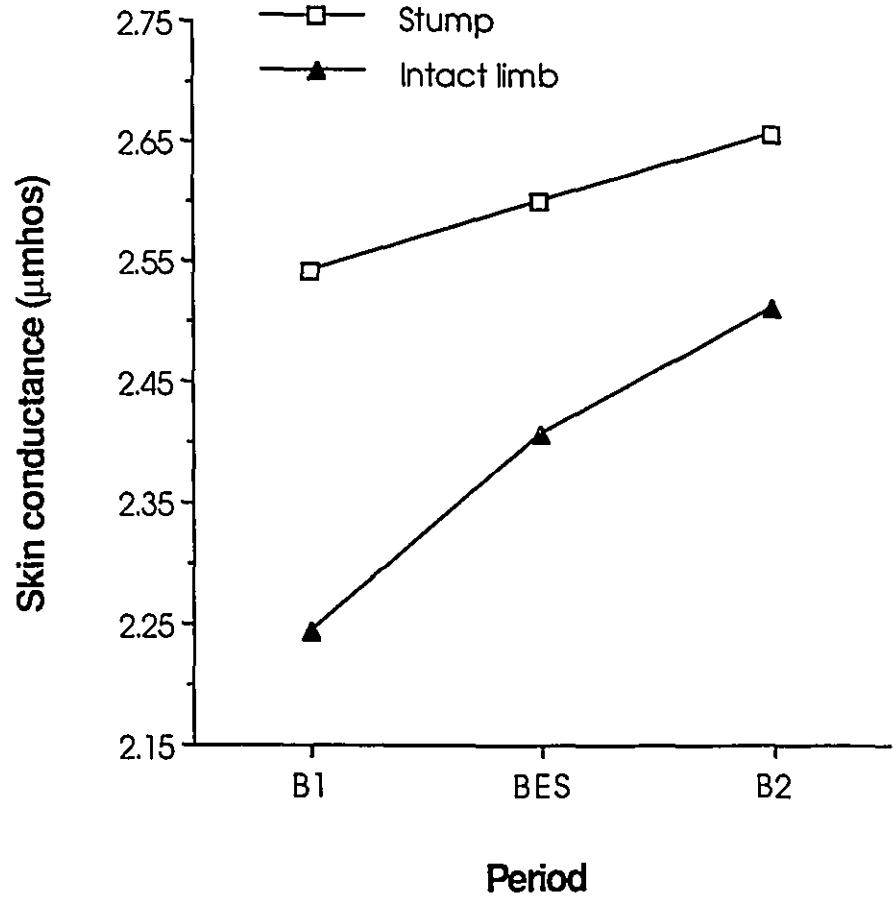


Figure 10. Mean skin conductance levels at the stump and intact limb shown for Periods B1, BES, and B2.

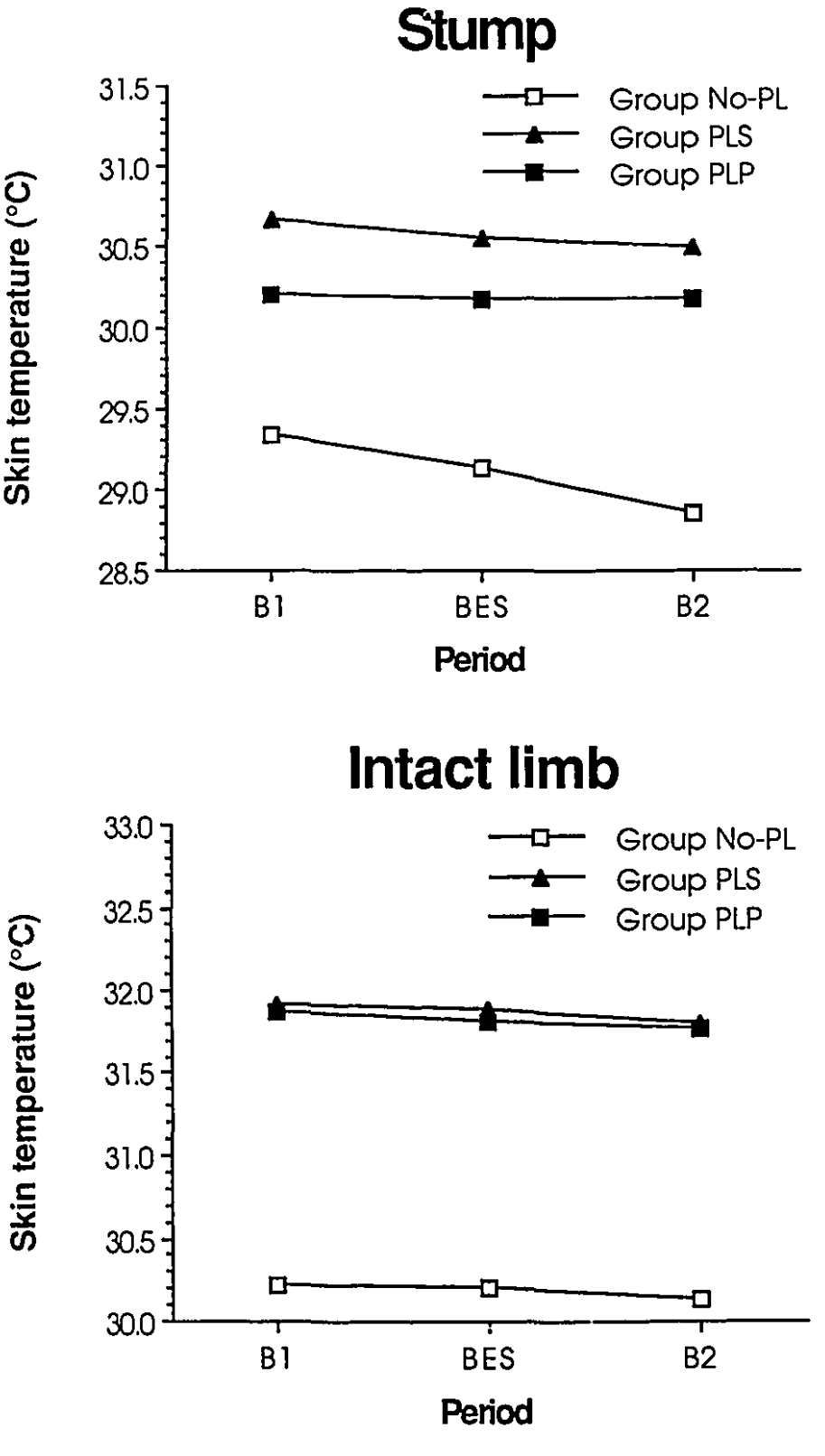


Figure 11. Plot of the three-way interaction cell means showing skin temperature at the stump (A) and intact limb (B) for each group across Periods B1, BES, and B2.

temperature decreased significantly from B1 to BES, and BES to B2 for subjects in Group No-PL but not for the two remaining groups.

IV. Qualitative changes in phantom limb sensations

The audio portion of the videotapes from both sessions were transcribed verbatim for each subject. The transcripts were reviewed and each report of a referred sensation or pain was coded in terms of its quality and location in the body. The results are presented below.

Sensations referred to regions other than the phantom limb. Two subjects, both in Group No-PL, reported feeling unusual sensations referred to regions other than the phantom limb. One (Case E25) noticed a "slight electric current inside the stump" on two occasions during BES on the TENS session. These sensations lasted about 15 seconds each and were not painful. The subject claimed that these were sensations which she had never experienced before. No other sensations were reported at any other time. The second subject (Case E03) reported feeling a sharp pain under her left breast on three occasions during BES on the TENS session.

Sensations referred to the phantom limb. The reports of sensations during Period B1 (on both sessions) provide an indication of the rate at which different phantom phenomena occur spontaneously when subjects are requested to focus attention on the phantom limb, since subjects are not expecting to receive TENS in this period. It is clear, from the patterns of PLI responding shown in Table 7, that the painful and non-painful phantom limb is not perceived as a static and fixed entity, but, as shall be seen, a dynamic and frequently changing phenomenon characterized by changes in tactile, kinesthetic, and proprioceptive sensibility.

For purposes of simplification and description, the reported sensations have been grouped into eight categories as follows: (a) *paresthesias* (i.e., reports of numb, tingling, prickling sensations, "pins and needles", "buzzing", "electric current" and "electric shocks"), changes in (b) *heat intensity* (including burning, hot, warm, and "steaming"), (c) *cold*

intensity ("ice", "freezing", cold, and cool), (d) *pressure and constriction* (tight, squeezing, "swollen", "full", "expanding"), (e) *weight*, (f) *posture* (immobility, "paralyzed", "stiff", "clutching" and "grabbing"), (g) *somatosensory memories* , and (h) *other* sensations (throbbing, pulsating, "pumping", twitching, sore, aching,"shocks" and "spasms").

Figure 12 shows the percentage of subjects in the two groups reporting at least one occurrence of each of the eight types of sensation summed across period and session. The figure does not depict the *frequency* with which each sensation was reported. Furthermore, these data represent a subset of the sensations registered by the subjects; not all sensations which were indicated using the dial were also verbally reported.

Chi-square analyses using Yate's correction for continuity were computed to determine whether the two groups differed in the proportion of subjects reporting each type of sensation. The analyses for the categories describing sensations of pressure and "other" sensations were significant (χ^2 (1) = 4.13, $p < .04$, and χ^2 (1) = 6.87, $p < .008$, respectively) indicating that the proportion of subjects reporting these types of sensation during the two sessions was greater in Group PLP than Group PLS. More specifically, these results demonstrate that there was a significant difference between groups in terms of the qualities of sensations that they were monitoring when using the dial to indicate changes in phantom limb intensity throughout the experimental procedures. These results are further evidence in support of the suggestion that the painful phantom limb embodies the same basic qualities of sensation as the painless phantom (i.e., paresthesias) and more (e.g., sensations of pressure as well as throbbing, pulsating, etc.).

V. The phenomenology of the phantom limb

As previously mentioned the subjects displayed a number of different patterns of responding when monitoring the intensity of phantom limb pain/sensations (Table 7). These were classified according to whether or not the subject reported a change in phantom intensity in each of the three periods. A more detailed examination of PLI as it changed over each 30

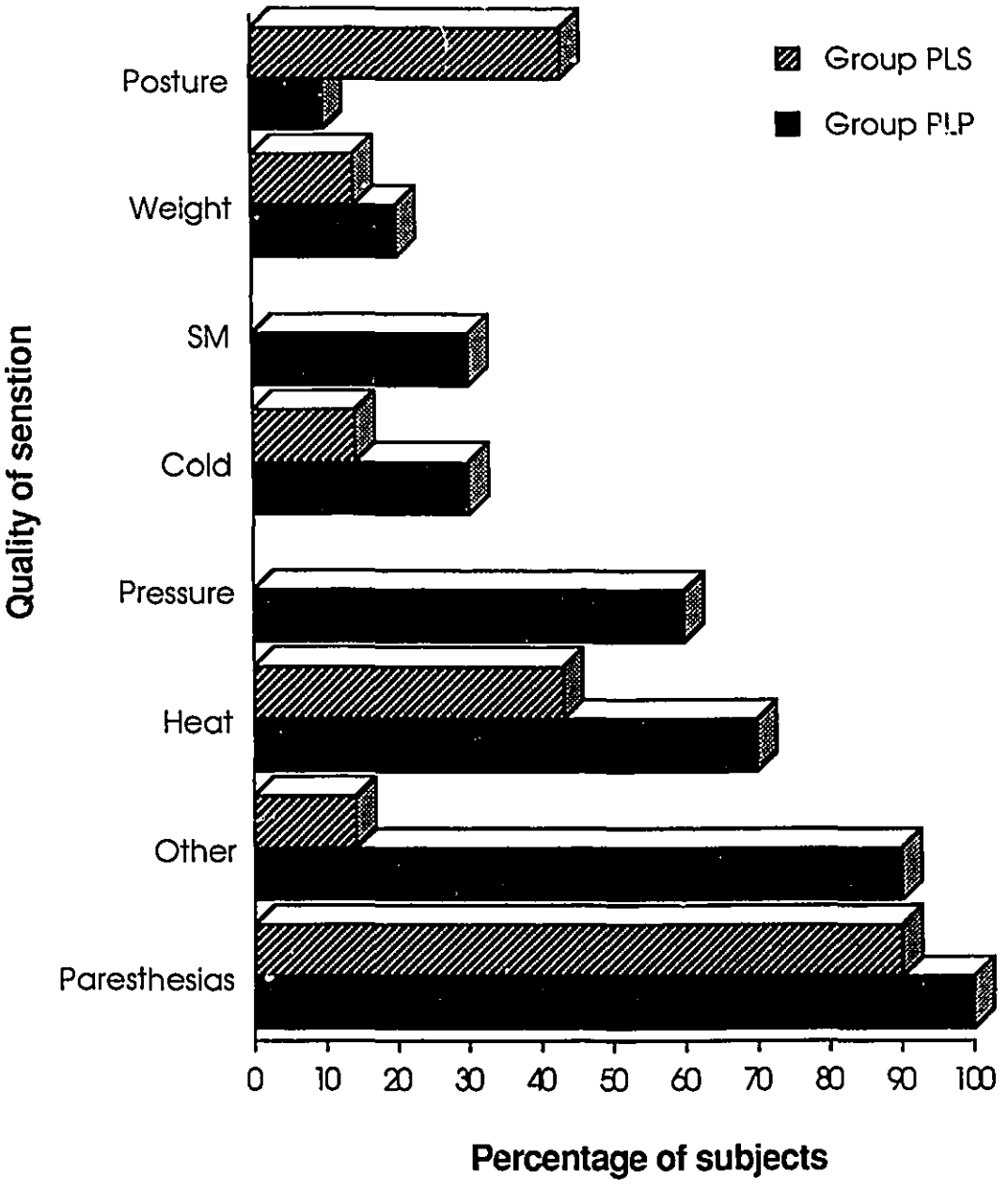


Figure 12. Percentage of subjects in Groups PLP and PLS reporting various qualities of sensation referred to the phantom limb on both sessions. SM = Somatosensory memory. "Other" sensations include throbbing, pulsating, "electric shocks", etc. (see text for details).

minute sessions revealed that there were three general categories of responding which seem to reflect different types of phantom limb experiences. The most common pattern is characterized by rapid fluctuations in phantom limb intensity which correspond to increases and decreases in paresthesias. The second category is described by relatively long periods (e.g., 5 minutes) in which the phantom limb remains stable and unchanged, followed by an abrupt increase or decrease, and a re-stabilization at the new level. The third pattern was found only in one subject with PLP but is probably not an uncommon occurrence. It is characterized by extremely brief increases in phantom pain that correspond to "jabs" or "bolts" which occur without warning and frequently are reported to travel the length of the phantom limb.

The graphs in Figures 13 and 14 show some typical examples of PLI responses produced by subjects in both groups. Also included are plots of stump skin conductance data, thus enabling a visual inspection of the relationship between changes in PLI and SC corresponding to the correlation coefficients presented in Table 12. The two graphs in Figure 13 show the relationship between phantom limb intensity and stump skin conductance on the TENS session for a subject (Case E23) with non-painful phantom limb sensations characterized by paresthesias. The lower plot (Figure 13b) contains only the data from the first resting baseline period (B1) when most of the changes in PLI and SC occurred. This period is characterized by rapid and almost simultaneous fluctuations in stump skin conductance and the intensity of non-painful phantom limb paresthesias. That is, increases and decreases in stump conductance parallel the increases and decreases in the intensity of paresthesias referred to the phantom limb.

Figure 14 shows the relationship between the same variables for Case E02 (Panel a) with non-painful paresthesias and Case E07 (Panel b) with phantom limb pain of a burning quality. Note the lack of a relationship between stump skin conductance and phantom limb pain intensity for this subject. On the other hand, Case E02 (Panel A) reveals a remarkably strong relationship with the increase in skin conductance preceding the perception of intense

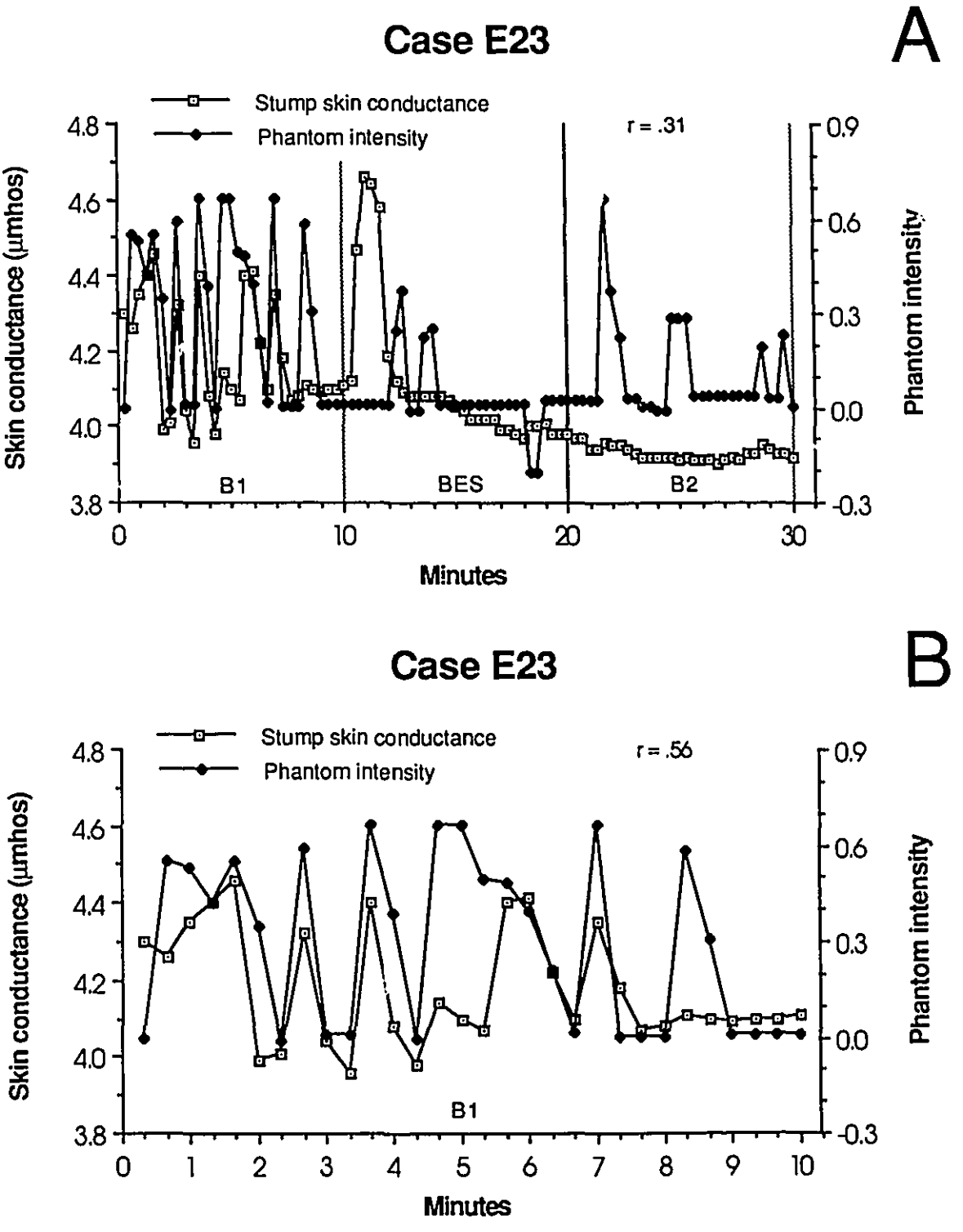


Figure 13. A: Plot of unsmoothed data from Case E23 showing the relationship (r = .31) between stump skin conductance (SC) and the intensity of non-painful phantom limb paresthesias (PLI) during Periods B1, BES, and B2 on the TENS session. B: Data from the same subject showing only Period B1. Note the tendency for SC and PLI to increase and decrease in tandem (r = .56).

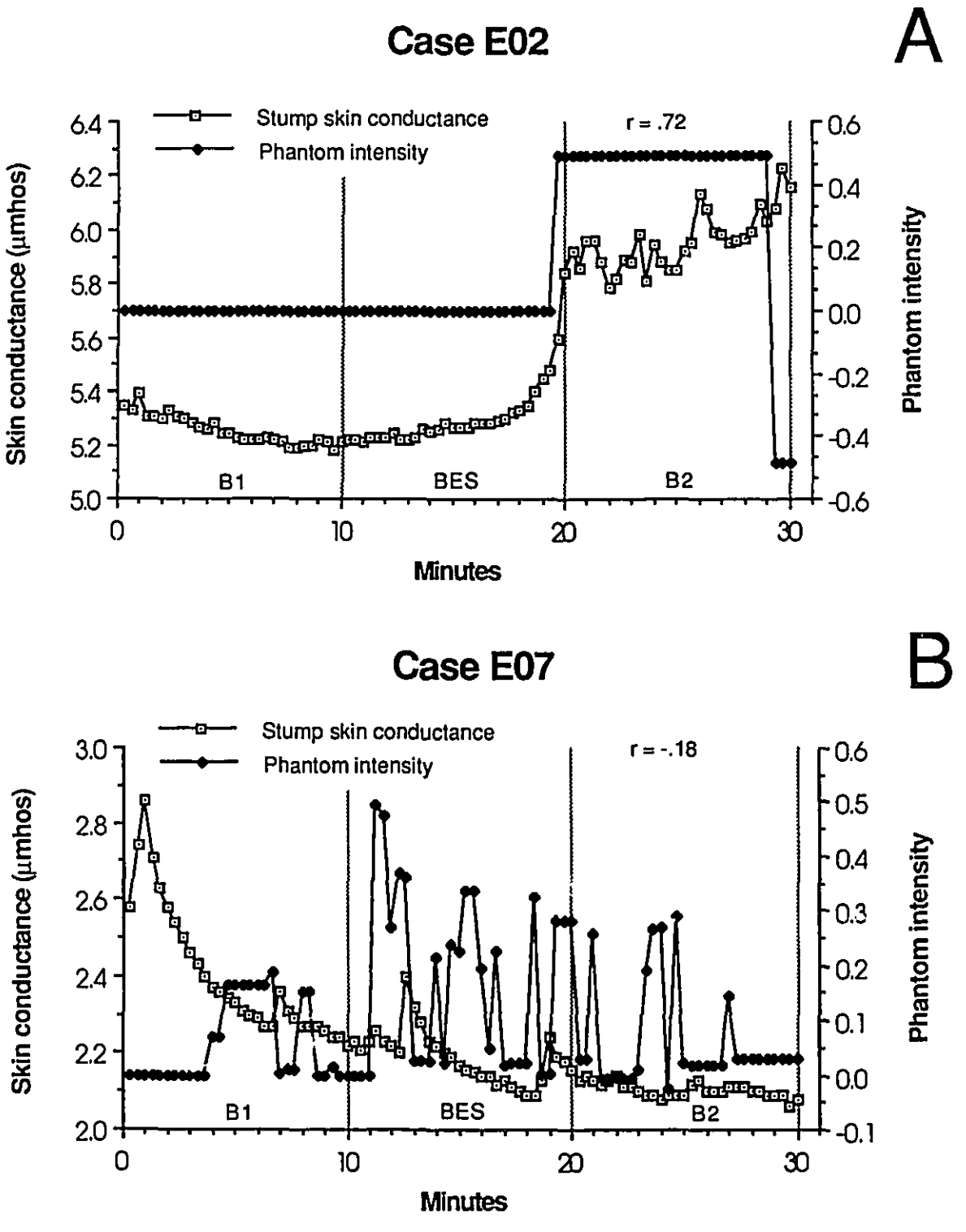


Figure 14. A: Plot of unsmoothed data from Case E02 showing the relationship (r = .72) between stump skin conductance (SC) and the intensity of non-painful phantom limb paresthesias (PLI) during Periods B1, BES, and B2 on the TENS session. B: Data from Case E07 who reported phantom limb pain of a burning quality. Note the absence of a significant relationship (r = -.18) between stump skin conductance and phantom pain for this subject.

phantom paresthesias by at least two minutes (see below for a description of the sensations reported by this subject).

In the remainder of this section I will present excerpts from the transcripts of certain subjects describing the phantom sensations and pain they experience. The object is to try to convey to the reader the phenomenological experience of a phantom limb as expressed by the subjects in their own words. The approach I have taken is purely descriptive and is intended to complement the more quantitative data provided in the previous sections and to elaborate on the information presented in Figure 12. The following material is presented according to the type of sensation reported so that excerpts from different subjects appear under sub-headings corresponding to the eight categories outlined above. In this way, reports of the same type of sensation may be compared, across subjects, for their similarities and differences. Excerpts are preceded by the subject's case number as well as the period and session in which they were reported.

Paresthesias and numbness.

Case E02

BES-TENS "It's getting more. ... Right down to the toes. ... It's just there, that's all. Just seems to have stopped, but I can feel it, you know what I mean? It's not ... paresthesias. It's like kind of a ... let's call it numb."

B2-TENS "Now it's just static. ... It's just like it's numb. ... it's not tingling as much. ... It's just like it was dead ... or numb."

Case E05

Interview "I feel right now like there's something. It's not a foot, it's ... it's just the end, you know, before the foot starts, but there's no pain. ... it's a mild, mild, prickly sensation but as a whole ... needles everywhere, you know, not individual, as a whole. It's hard to explain ... you know sometimes you feel pinpricks everywhere ... where what I feel now is the bottom, but not the full foot, like the back ... It feels like the beginning of a numbness or some kind of activity, like pinpricks, but altogether, not separated ... and it's mild. I'm not uncomfortable with it."

Case E08

BES-TENS "It seems to become more frozen or numb again, a little more. Like it's getting ... like if it was there and heavier... If I can compare it, it would be about the same feeling as when ... they freeze your teeth. It doesn't feel cold, but it feels like it's getting thick, clumsy, that's it, thick."

BES-TENS "It's getting more frozen--if I may use this expression. It's not cold, but it's getting like heavier, numb completely. It's going above the ankle a little in the front only. I don't feel anything in the back. The heel and the back of the ankle, I don't feel, but the front there, where the arch of the foot is, I feel it ... on the left side only."

Changes in temperature

Case E04

B2-Placebo "That left hand is getting cool ... I don't think it's *cool*, it's almost as though there were ice in the veins; *cool*, *cool*. Not quite cold enough to get frostbite, but cool."

BES-Placebo "I can feel the fingertips sort of pulsing but cool, and then there's the ... the heel of my hand. The elbow's there, but not doing much of anything ... Now it's warm in my upper arm, or just above the elbow. Just warm *there*, not overall warm ... and expanding, pulsing ... my pinky finger is throbbing as though in sympathy to where it's expanding."

Case E05

BES-Placebo "Now I feel like I could feel my knee. ... Without pain, yah, but it's cold. I never felt that before. I felt my knee because it was cold at the end... not the stump ... it seemed like it was further than my stump and it felt *cold*, but felt like a knee, like I would touch my knee and it would feel cold. Now I start to feel my foot again ... heavy. ... Heavier and uh ... I feel my knee, now. Boy that's a new sensation!"

Case E20

BES-TENS "The pain ... the pain, it's different. It's not *pain*. It's like if you froze your hands outside and then you go in. It's hot, it burns, but it's very, very low. It's hot, I feel my foot like, like it was frozen ... as if you had frozen it outside in the snow and it's coming back, softly. Like it's thawing. It seems a little like that."

Sensations of pressure and constriction

Case E04

B1-Placebo "Um ..... I don't know how to explain that to you: If you have a knot and it loosens, you know, like a muscle spasm type thing, I just felt that in my elbow ... and now my hand is tingling."

Case E07

B2-Placebo "Um, in the back of my ankle, I feel it. A squeezing ... and also a tingle, like freezing cold."

Somatosensory memories

Case E04

B1-TENS "... the hand is tingling now and I'm well aware of the elbow, somewhere between the elbow and the hand. I feel as though it's full of edema, extra fluid where it doesn't belong. ... When I say edema, I remember the feeling I had when I wore the sleeve. That's what it brings to mind. This was long before the amputation when I used to wear a sleeve to keep the swelling in the arm down, you know."

B1-Placebo "OK now, my hand seems to be starting to drain. ... It's like draining it from edema. It's as though it were trickling out, and it's a little cooler. I guess I explain it that way because it reminds me of how my hand and arm felt when I was in that machine at the hospital having the edema pumped out. I can't really say... it's as though my arm were up and the fluid was leaving it. Now, it still feels swollen. ... There's the draining feeling, leaving the finger tips, going down the finger into the palm of the hand, through the palm, into this part [the heel of her hand], then it just doesn't happen. I mean there's nothing there: The wrist is there, but it isn't part of the draining ... well, it's not draining anymore, now it's throbbing."

B2-Placebo "My thumb feels the way it did when I first was losing the sensation ... the feeling of being asleep, but more ... fat and being asleep. ... That strange tingling feeling."

Case E06

B1-TENS "... and that's where I had the pain. I'm just thinking of that now. You know when I had the bypasses? The side of my leg, that's where it was so sore, where I'm getting those there now. That's how I knew that I had a blockage ... it was sore down the side of my leg."

B2-TENS "It's down the side of the leg again. When I went in there, [referring to the hospital] I had the pain on the side of my leg which told me that something was wrong, you know. That's when I had no circulation. And it's the same kind of pain I got, like down the side of my leg on the outside."

Case E20

BES-Placebo "I just had a pain in my leg. Bone pain. When I was a kid, I had a pain like that in my leg. Well, like eight, nine, ten years ago. I had pain in my leg. You know in my bone. Like my bone hurt. You know, some kids get that - when they grow up - It's the same thing I got."

Sensations of weight

Case E04

B1-Placebo "I'd say that there was a heaviness almost as though I had a breast again. Not quite a phantom pain, but ... as though I'm supporting weight."

B2-Placebo "A tight feeling in the upper arm, heavy, ... a load .... almost the way it would be if you were lifting weights I imagine, and the muscles are all tense."

Case E05

BES-TENS "I start to feel my foot, but without pain, without ... a weight. ... the foot is starting to form completely. ... I don't feel the weight. I don't feel the form of the foot."

Case E06

B2-Placebo "The heaviness ... It isn't *so* heavy, so I can feel the leg from the knee down. [When the leg feels heavy] it just feels heavy and I can't distinguish ... the knee from the foot."

Postural sensibility

Case E04

BES-TENS "It's cool and comfy and quiet at my side. Instead of the hand being out here [arm outstretched] it's sort of against the body like this [hanging by her side].

B2-TENS "...but my hand, instead of being against my side is back out again ... now this is with my arm straight down, you understand. It's not waving in front of me."

Case E12

B1 TENS "Now I can't even bend my toes. It's like they're paralyzed. ... usually I *think* I can move them."

Other sensations

Case E04

B1-Placebo "There's a feeling in the elbow, as though I've been batting a tennis ball around and it's tired ... and it's cool again. The fingers don't seem to be so spread eagle ... I feel the whole palm like a ... sort of a wave or something going over it, and it comes back and goes over it ... the hand is cool ... the elbow feels just ... full."

B1-TENS "This is really a dichotomy. My normal reaction is to ignore the pain and I have the feeling that you'd like me to concentrate on it ... My arm's just there. It's not cool but it's not hot and invading my mind ...It's not as cool as it was before ... but it's not drawing attention to itself. ... I know that when I start thinking about my arm it feels it has licence to hurt, so I guess one of the things I try to do is *not* think about it. It's sort of like keeping the lid on Pandora's Box".

Case E05

BES-Placebo "It's funny, I felt just then, I felt my knee again, but not good ... not a pain, but not cold like a little while ago. Like nerves was working inside, inside the knee. ... I didn't feel the coldness that I did a while ago. That coldness felt good."

Case E11

Interview "The only way I can describe it now, is like looking out the window ... but the window is frosted. You see what is there, but you don't see the sharp edges of everything. Or, if you touch somebody but there's something on the skin so you don't touch the skin, but you feel the skin, you feel ... Oh, I don't know, something like that ... but it can be very intense even if it is like that".

"It's painful because the feeling is excessive, but it's not a pain like if you cut yourself or banged yourself. It's not that kind of pain. I can't call it a pain. It's not a pain. It's not a *physical* pain, I would say, because that [motioning to the space below the stump] is not physical ... and it is. So, how can I describe something that is physical and is not? It's physical in the way that I feel it, but it's not there. So how do you describe something like that? So it's painful but ... it's not a pain. I don't know what else to say".

Case E15

BES-TENS "... it's more like a "dull ache", like a toothache. It's more like an arthritis, like a rheumatism maybe, you know, it's just ... not actually to touch it. Not like a sore spot, just inside. ... Now you know when I was doing that before [lifting the stump and moving it around in circles], that was aching ... now you see it's a lot easier to move, and without aching. I can still just barely feel it. I can feel it a little bit but not like before. ... It's taken most of it away. Damn near all of it away."

B2-TENS "This has a tendency to ache down here [referring to the stump] ... especially like after I work out ... Actually it's aching right now ... After I've been doing my exercises and walking around. ... it has a tendency to feel very heavy, the stump ... When the stimulation [TENS applied at the ears] was on it feels very light, and no pain."

Discussion

Referred sensations

The results of Study 2 demonstrate the remarkable diversity in quality and intensity of painful and non-painful sensations referred to the phantom limb. Subjects reported sensations that ranged from simple diffuse paresthesias to complex experiences of past somatosensory events that were initially felt in the limb prior to the amputation. The range of sensations underscores the complexity of the neural mechanisms responsible for the phantom limb percept whether painful or painless (Melzack, 1989a).

Reports of referred sensations were evident in all periods on both sessions. They were reported during baseline periods, in which subjects were not expecting to receive TENS, during Period BES on the placebo control session when they believed they were, and during BES on the TENS session when electrical stimulation was delivered. This lack of specificity made it extremely difficult to determine the source of these sensations. There are at least three possibilities. First, referred phantom sensations may be spontaneously occurring and noticed especially when attention is focused on the phantom limb. One can only speculate on whether the process of directing attention to the phantom changes its perceived quality and intensity. This possibility will be addressed below. Second, they may be induced by the expectation of

receiving TENS, and third, they may be due to the effects of electrical stimulation. There is evidence to support all three possibilities and it is likely that each is operative within a single subject as shown by an examination of the transcripts from each session.

However, for subjects who reported changes only during the BES period on the TENS session it was apparent that the sensations induced by TENS were primarily changes in the intensity of paresthesias, and in particular, numbness. Most impressive was the significant *decrease* in the intensity of non-painful paresthesias for subjects in Group PLS during electrical stimulation on the TENS session.

TENS-induced decrease in phantom limb intensity

Mean levels of PLI were reduced significantly during BES on the TENS session for Group PLS but not Group PLP. On the other hand McGill Pain Questionnaire PRI-S and PRI-T scores were significantly lower following TENS for Group PLP but not Group PLS. Similar changes were not apparent for either group on the placebo session. Furthermore, ratings of mood state, sleepiness, and anxiety remained virtually unchanged across sessions and test occasions, thus ruling out the possibility that the decrease in pain was mediated by emotional factors.

At first glance it may seem inconsistent that phantom limb intensity was reduced during but not after TENS in Group PLS, and after but not during TENS in Group PLP. However, this apparent inconsistency can be explained by the basic difference between painful and non-painful phantom sensations. First, consider the results from Group PLS. The "normal" non-painful phantom is defined by its paresthetic quality. It is a fairly uniform and universal phenomenon and is frequently described as a "tingling", "prickling", "electric current", or "numb" sensation (Henderson & Smyth, 1948; James, 1973; Jensen et al., 1984; Simmel, 1962; Wall, 1981). Consistent with these reports, the majority of subjects endorsed MPQ descriptors from the sensory class which confirms the basic paresthetic quality of the painless phantom (see Table 9). There was very little variation in the quality of non-painful

phantom sensations experienced by different subjects. The observed reduction in PLI during period BES on the TENS session indicates that TENS reduced the paresthetic quality of the normal, non-painful phantom. The reduction was evident only for the duration of electrical stimulation. By the end of the final resting baseline period (i.e., B2) PLI ratings had returned to the pre-stimulation baseline level. Post-stimulation MPQ ratings were taken more than 10 minutes after BES offset and thus any changes that had occurred were no longer evident. Thus the lack of a significant change in PRIs is not inconsistent with the pattern of PLI responses observed across the 30 minute sessions.

The "painful" phantom is a less homogeneous entity than the non-painful phantom, and while many sufferers describe a painful or non-painful paresthetic component, most are beset by other types of pains as well (Bradley, 1955; Brown, 1968; Gillis, 1964; Jensen et al., 1983, 1985; Livingston, 1943; Sherman et al., 1980; Sliosberg, 1948; Sunderland, 1978). Evidence for this diversity in Study 2 comes from Table 9 showing that subjects in Group PLP endorsed more MPQ descriptors from a wider range of classes. In addition, when monitoring levels of PLI, Group PLP reported more varied qualities of sensation than did Group PLS. Subjects in Group PLP reported significantly more sensations of pressure as well as "jabs", throbbing, pulsating, and others (Figure 12).

These basic differences between painful and painless phantom limbs may explain why subjects in Group PLP did not show a decrease in PLI during TENS, but did report a significant reduction in phantom limb pain as measured by the PRI-S and PRI-T 10 minutes later. Post-TENS changes in the phantom, as measured by the MPQ, were limited to those descriptors used to describe the paresthetic or dysesthetic component of the phantom. It is reasonable to assume, then, that TENS reduced the same aspect of the phantom in both groups. For subjects in Group PLP, the reduction in paresthesias during TENS may have been overshadowed by other, more troublesome and painful, sensations they were also monitoring. The proportion of the total pain that was reduced by TENS may have been minimal in relation that which remained so that in the context of the other phantom limb

sensations, these subjects reported relatively small changes in PLI. This may account for the lack of a significant reduction in PLI during Period BES on the TENS session.

It is difficult to account for the difference in the duration of the effect of TENS for the two groups. For Group PLS the reduction in paresthesias lasted for the duration of the electrical stimulation only, whereas it was apparent more than 10 minutes after TENS offset for Group PLP.

Results of a previous controlled trial of "auriculotherapy" (Melzack & Katz, 1984) showed that electrical stimulation applied at the outer ear was no more effective than a tactile placebo control condition similar to that used in Study 2. There are two major differences between these studies. The first has to do with the nature of the pain states treated. The sample in the Melzack and Katz study comprised patients with chronic pain syndromes of diverse etiology but did not contain any amputees suffering from phantom limb pain. The second difference concerns the pulse duration of the electrical stimulation, it being long (125 msec) in the study by Melzack and Katz, and short (100 μsec) in Study 2. This latter difference will be part of the focus of Study 3.

The reduction in painful and non-painful paresthesias produced by TENS applied at the outer ear can be explained by a diffuse noxious inhibitory control (DNIC; LeBars, Dickenson, & Besson, 1979; 1983) mechanism activated by a form of "hyperstimulation" (Melzack, 1971; 1975, 1989b; Melzack & Wall, 1988). Moderate to intense stimulation, of various kinds, applied at sites distant from the region of pain is effective in relieving chronic pain (Carabelli & Kellerman, 1985; Melzack & Wall, 1988). Such stimulation activates brainstem structures that exert an inhibitory control over nociceptive neurons in the spinal cord dorsal horns (LeBars et al., 1979, 1983; Melzack, 1971, 1975, 1989b; Melzack & Wall, 1988). The outer ear is richly innervated by somatic afferents, including five cranial and two cervical spinal nerves (Bossy, Golewski, Maurel, & Seoane, 1977; Brodal, 1972). It is reasonable to assume that electrical stimulation applied to the outer ears produces its effects by activating this descending pain control system. Since TENS is a non-invasive treatment with

few contraindications (Tyler, Caldwell, & Ghia, 1982) it could be applied at the outer ear routinely in cases of painful phantom limb paresthesias.

The relationship between phantom limb intensity and psychophysiological variables

Changes in stump skin conductance and skin temperature correlated significantly with changes in phantom intensity on both TENS and placebo sessions for many subjects, suggesting a relationship between sympathetic activity at the stump and the intensity of phantom limb sensations. The nature of the relationship is not entirely clear but two hypotheses are proposed.

Sympathetic activity results in phantom limb paresthesias. The first hypothesis explains the relationship between sympathetic activity at the stump and the intensity of phantom limb sensations by assuming that neurotransmitter release from sympathetic fibers provides a sufficient condition for primary afferent impulse generation and, as a consequence, paresthesias referred to the phantom limb. Sympathetic activity in the form of skin conductance responses and changes in skin temperature reflect the activity of post-ganglionic sudomotor and vasomotor fibers (Bini et al., 1980a, 1980b; Delius et al., 1972; Hagbarth et al., 1972; Hallin & Torebjörk, 1974; Jänig, 1988; Jänig, Sundlöf, & Wallin, 1983; Wallin, 1983, 1988). Multiunit sympathetic activity recorded from skin nerve fascicles in awake humans shows a strong relationship to effector organ responses including vasoconstriction and sweat gland activity (Bini et al., 1980a, 1980b; Delius et al., 1972; Hallin & Torebjörk, 1974). These studies show that bursts of activity in sudomotor and vasomotor fibers are reliably followed by transient electrodermal responses and plethysmographic signs of vasoconstriction within the region of skin subserved by the sympathetic fibers under study.

It is proposed that increases and decreases in the intensity of phantom limb paresthesias are in part sympathetically-governed. According to this hypothesis, changes in the intensity of phantom sensations reflect the activity of cholinergic (sudomotor) and

noradrenergic (vasomotor) postganglionic sympathetic fibers on primary afferents located in the stump and stump neuromas. Bursts of activity in sympathetic fibers produce transient vasoconstriction and heightened skin conductance levels. Shortly after, afferent fibers in stump neuromas would increase their rate of firing due to the liberation of acetylcholine and noradrenaline. These impulses are referred to certain regions of the phantom as increases in paresthesias. Thus, the moment to moment fluctuations in the intensity of phantom limb sensations characteristic of the subjects' response patterns, may, in part, represent a cycle of sympathetic-efferent and somatic-afferent activity. Increases in the intensity of phantom limb sensations would follow bursts of sympathetic activity due to neurotransmitter release and decreases would correspond to periods of relative sympathetic inactivity.

Bursts of sympathetic activity may be spontaneous, or induced by orienting stimuli, including environmental events, emotions, and cognitions (Bini et al., 1980a, 1980b; Delius et al., 1972; Hagbarth et al., 1972). Loud noises, fright, violent scenes on television have all been reported to increase the intensity of phantom limb sensations, including pain (Henderson & Smyth, 1948; Livingston, 1943; Pilowsky & Kaufman, 1965; Pitres, 1897; Simmel, 1956; Stengel, 1965). The mechanism by which these stimuli accomplish this may be via the peripheral actions of sympathetic efferents on primary afferents located in the stump.

The question was raised earlier of whether actively monitoring and attending to the phantom limb in some way alters the phantom sensations. At least one subject indicated that attending to the phantom seemed to worsen the pain. Distraction was an effective coping strategy which reduced the phantom limb pain. Diversion of attention has been shown to alter peripheral sympathetic activity (Hagbarth et., 1972). Reflex sympathetic discharges in response to a repetitive stimulus applied to a subject's leg disappeared when his attention was focused on an unrelated event. This suggests that distraction may be effective in reducing phantom limb pain by activating a descending control mechanism which inhibits pre-ganglionic sympathetic outflow, thereby decreasing peripheral sympathetic discharge and the

rate of firing in primary afferent fibers in the stump which project to central structures subserving the phantom limb.

Phantom limb paresthesias result in sympathetic activity. The second hypothesis explains the relationship between sympathetic activity at the stump and the intensity of phantom limb sensations by assuming that peripheral sympathetic activity, in the form of electrodermal and vasoconstrictive responses, is a *consequence* of a perceived change in the intensity of the phantom limb. According to this hypothesis, the subject's detection of a change in phantom limb pain or other sensations produces vasoconstriction and heightened electrodermal activity much as any anticipated and salient signal during a vigilance task results in a generalized orienting response (Delius et al, 1972; Hagbarth et al, 1972; Voronin, Bonfitto, & Vasileva, 1975).

A closer examination of these two hypotheses indicates that they may not be mutually exclusive. The major distinction between them concerns the temporal order in which the sympathetic efferent and somatic afferent activity is hypothesized to occur. Such a distinction represents an arbitrary punctuation of events in a repetitive cycle. Once underway, it makes little difference which occurs first.

Sympathetic activity *has* been found to modify afferent inflow at the receptor level in animals (Barasi & Lynn, 1983; Pierce & Roberts, 1981; Roberts et al., 1985) and in the awake human (Hallin & Wiesenfeld-Hallin, 1983; see Nathan, 1988 for a review). Thus even if, as the second hypothesis, assumes, the initial SC response occurs as a consequence of the perception of a change in phantom limb intensity , the resulting sympathetic discharge which led to the sudomotor and/or vasomotor response would be expected to increase afferent discharge as outlined above.

Although these two hypotheses are not mutually exclusive, the evidence from Study 2 favours the first hypothesis. The first hypothesis assumes that the paresthetic component of the painful and non-painful phantom is, in part, sympathetically-governed. Other qualities of

pains, however, are obviously unrelated to this peripheral mechanism. Thus, cramping pains, shooting pains, somatosensory memory pains, and phantom limb movements probably are generated by involvement of more central structures. It has already been established that the painful phantom consisted of more than the paresthetic component for subjects in Group PLP (Figure 12). These subjects reported changes involving more varied types of sensations when monitoring their phantom limb during the experimental procedures.

If sympathetic activity represented a response to a phantom sensation, as the second hypothesis assumes, then the detection of *any* phantom sensation, whether painless or painful, would be expected to produce increases in electrodermal activity. According to this hypothesis, skin conductance should correlate with both non-painful paresthesias and phantom limb pain. However, the results showed that significant correlations between skin conductance and PLI occurred significantly more frequently among subjects in Group PLS than PLP. This indicates that many of the phantom limb pains which were monitored with the dial had no peripheral correlate (see e.g., Figure 14b). These results are inconsistent with the second hypothesis which does not distinguish between the effects of different qualities of phantom limb sensation.

Between-limb correlations of psychophysiological variables. Subjects in Group PLS did not display a significant difference between mean levels of skin temperature or skin conductance at the two limbs. For these subjects there was no evidence of excessive sympathetic nervous system activity at the stump. Furthermore, between-limb correlations were extremely high for skin conductance and skin temperature indicating that fluctuations in sympathetic activity in the stump were accompanied by similar changes in the intact limb. Since these changes occur bilaterally, and without evidence of hyper-activity, it is of interest to ask why bursts of sympathetic activity do not also produce a sensation of paresthesias in the intact limb. That is, why do normal levels of sympathetic discharge in the stump lead to fluctuations in paresthesias for subjects who report the presence of a phantom, whereas

equivalent sympathetic output from fibers in the intact limb produces no such changes? One answer to this question can be found in the hyper-sensitivity of regenerating sprouts to the effects of sympathetic discharge (Wall & Gutnick, 1974). But not all amputees who showed a significant relationship between stump SC and PLI were recent amputees.

There is another explanation for the finding that normal levels of sympathetic activity in both limbs result in referred phantom paresthesias but no perceptible sensation in the intact limb. The explanation has to do with the hyper-sensitivity of spinal cord dorsal horn cells arising from the loss of inhibitory control brought about by amputation. Dorsal horn cells which normally subserve the limb are released from inhibitory control and give rise to the perception of a phantom limb (Carlen et al., 1978; Melzack & Wall, 1988; Wall 1981). This reduction in inhibition suggests that thresholds for detecting non-noxious stimuli will be reduced. Carlen et al. (1978) found that the effects of electrical stimulation applied at low intensities to the surface of the stump was frequently felt in the phantom but not at the actual site of stimulation until the intensity had been increased. That is, the threshold for detecting sensations in the phantom limb during stump stimulation was lower than at the site of stimulation itself.

Sympathetically-governed afferent discharge is occurring at low levels at all times (Bini et al., 1980a, 1980b). In the intact limb these impulses are inhibited at the level of the spinal cord. But impulses from fibers in the stump are unopposed when they reach cells in the spinal cord and more rostral structures where they give rise to the experience of paresthesias originating in the phantom limb. Thus, one need not postulate hyper-active sympathetic activity or an increased rate of primary afferent discharge to account for the perception of sympathetically-induced phantom limb paresthesias.

That the stump and intact limb did not differ significantly in pressure sensitivity thresholds is not necessarily evidence against the possibility that spinal cord cells subserving the phantom limb are in a state of hyper-sensitivity. The stump may show a lower threshold for detecting phantom sensations than local sensations even though between limb differences

in pressure sensitivity thresholds are negligible. Subjects were to indicate when they detected a sensation at the *stump*, not in the phantom limb.

However, the lack of a significant difference between pressure sensitivity thresholds at the stump and intact limb is surprising since amputation stumps have consistently been found to display greater skin sensitivity than the corresponding region of the contralateral intact limb. Lowered thresholds at the stump have been demonstrated in adults for light touch, two-point discrimination and point localization after amputations of the upper extremity (Katz, 1920 cited in Haber, 1955) and for two-point discrimination following lower extremity amputations (Teuber, Krieger, & Bender, 1949; Varma et al.,1972). Similar results have been found for pressure sensitivity and two-point discrimination thresholds in children with congenital absence of limbs (Wilson, Wilson, & Swinyard, 1962; Weinstein, Sersen, & Vetter, 1964). This increased acuity has been attributed to a process of central reorganization in which the stump is hypothesized to take over the vacated cortical regions (Merzenich et al., 1984) Most of these studies obtained tactile acuity measurements from several regions of the stump and intact limb whereas in Study 2 only one point was selected. It is possible that the lack of a significant between-limb difference in sensitivity thresholds in Study 2 stems from the restricted sampling of regions on the two limbs.

The suggestion that sympathetic activity is responsible for changes in the intensity of the paresthetic component of the phantom limb is not meant to imply that all paresthesias are due to primary afferent discharge. Local anesthetic blocks of the stump sometimes fail to abolish the phantom (Nyström & Hagbarth, 1981). Spontaneous activity in the cells of the dorsal root ganglion (DeSantis & Duckworth, 1982; Wall & Devor, 1981) and spinal cord (Wall, 1981) may be sufficient to produce these sensations as is electrical stimulation of the medial lemniscal pathway and more rostral brain structures (Tasker et al., 1982; Young, 1989). However, the results of Study 2 strongly suggest that an important contribution to the perception of the normal, non-painful phantom limb comes from

sympathetically-governed primary afferent discharge of fibers located in the stump and stump neuromas.

Relationship between phantom limb pain and stump temperature

The stump was significantly lower in temperature than the intact limb for subjects with phantom limb pain, but not for subjects with a non-painful phantom or those with no phantom limb at all. These results confirm Livingston's (1938, 1943), and more recently Sherman's (Sherman, 1984; Sherman and Bruno, 1987) assertion that phantom limb pain is associated with reduced blood flow at the stump relative to the intact limb.

However, the results of Study 2 do not support the claim that phantom limb pain intensity is positively related to the magnitude of the temperature difference between the stump and intact limb (Sherman, 1984; Sherman & Bruno, 1987). The six subjects who reported different levels of PPI on the two sessions showed no tendency for the temperature difference between the stump and intact limb to increase with increasing phantom pain intensity. Perhaps the lack of a significant relationship between pain intensity and temperature difference stems from the relatively low overall pain intensities reported by the subjects in Group PLP compared to those reported by Sherman (personal communication, Nov. 20, 1988).

The results of Study 2 point to the importance of a peripheral vascular component as one of the contributing factors to the development or maintenance of phantom limb pain. Excessive vasoconstriction may, in part, be responsible for the pain in the present series of subjects. Interventions aimed at restoring normal sympathetic functioning have proved effective for some sufferers of phantom limb pain (Livingston, 1938, 1943). Sympathetic blocks should be tried in cases of PLP in which the stump is significantly lower in temperature than the intact limb. On the other hand, the presence of a peripheral abnormality does not imply that the primary disorder is a peripheral one, or, that interventions aimed at the periphery will necessarily eliminate the pain (Sunderland, 1978; Melzack, 1971). That the relationship between peripheral vascular factors and phantom limb pain is not a simple one is

further reinforced by the finding that skin temperature (on both limbs) decreased significantly over time on both sessions, yet MPQ ratings after the TENS session were significantly reduced for Group PLP.

Summary and Conclusions

The results of Study 2 strongly suggest that peripheral factors play a role in the normal, non-painful phantom as well as the painful phantom limb. The stump was significantly lower in temperature than the intact limb for subjects complaining of phantom limb pain but not in subjects with painless phantom sensations or those with no phantom limb at all. The between-limb temperature difference in Group PLP is evidence of an abnormal peripheral vascular component associated with phantom limb pain and suggests that reducing the sympathetic vasoconstrictive tone may lead to a decrease in pain.

Significant relationships were found between stump skin conductance, skin temperature and the intensity of phantom limb paresthesias. These results support the hypothesis that increases and decreases in peripheral sympathetic nervous system activity lead to corresponding changes in the firing rate of primary afferents located in the stump which are then referred to the phantom limb as paresthesias or dysesthesias. Other phantom limb experiences are independent of this peripheral mechanism.

Finally, TENS applied to the outer ears was significantly more effective than a placebo in reducing the intensity of non-painful and painful phantom limb paresthesias. Given, the low success rate of current treatments for phantom limb pain (Sherman, 1980; Sherman et al., 1980), electrical stimulation applied at the outer ear could be employed as a method of relieving the frequently annoying, and painful paresthesias reported by most sufferers of phantom limb pain.

STUDY 3: A CASE STUDY OF TENS APPLIED AT THE EARS AND CONTRALATERAL LEG

The third study is a placebo-controlled trial of TENS applied to the contralateral lower leg and outer ears of an amputee with non-painful phantom sensations. A number of case reports have recently shown that TENS or local anesthetic agents applied to the contralateral limb can be effective in reducing phantom limb pain (Carabelli & Kellerman, 1985; Flöter; Gessler, Struppler, & Oettinger, 1981; Gross, 1981, 1982; Winnem & Amundsen, 1982).

Objective

The purpose of this study is to compare the effects of electrical stimulation applied at the ears and contralateral lower leg on phantom limb intensity, stump skin conductance, and skin temperature. The subject had participated in Study 2 in which a marked reduction in the intensity of his PLP was noted during TENS applied to his ears. No reduction was noted during the placebo session. In Study 2, TENS was delivered to the ears using a short pulse duration (i.e., 80 μsec at 4 Hz). In Study 3, TENS is applied to the outer ears using a longer pulse duration (i.e., .125 msec at 4 Hz). These latter parameters match those used by Melzack and Katz (1984) who failed to find a significant reduction in chronic pain. Thus, the next study may provide preliminary results which bear on the role of pulse duration.

Methods

Case E20

The subject was a 23 year-old man who had lost his right leg above the knee as the result of a motorcycle accident in early September 1987. He was seen, initially, 61 days after the amputation while a patient at a Montreal area hospital, where he had been transferred for prosthetic fitting and training. At the time of the initial interview he reported several distinctly different types of phantom sensation which occurred with varying regularity.

Phantom limb pain. Immediately following the amputation he felt painful paresthesias in the phantom foot. Several days post-operatively he began to develop paroxysmal "spasms" in the phantom calf, knee, and thigh as though suddenly "stabbed with a sharp pencil". These pains occurred without warning and at times repeatedly, with successive "stabs" separated by several seconds.

Somatosensory memories. On a number of occasions the subject had noticed painful and non-painful phantom sensations that were replicas of experiences felt in the right leg prior to amputation. He reported feeling what he termed "growing pains" on the anterior aspect of his phantom shin. These were in all respects the same pains which he had originally experienced as an adolescent approximately 8 years earlier but had not felt in the intervening years until the amputation.

Two sensations which he originally experienced at the accident scene have recurred as phantom phenomena. He described the first as the beginning of a muscle cramp in his right calf that threatened repeatedly to develop into a full-blown spasm but never did. The second occurred a short time later when the ambulance attendants arrived and were required to cut off his right boot in order to release his foot. He felt a sensation of "cool fresh air" on his foot and a considerable release of constrictive pressure as it was freed from the boot. The phantom sensation, which has recurred a number of times, consists of the latter sensation and does not include the "cool fresh air" component. This phenomenon of past somatosensory events re-experienced in the phantom is a frequent sequela of amputation and has been recently described elsewhere (Katz & Melzack, 1988).

At the time of the initial interview. he reported that the "spasms" had decreased in frequency, occurring approximately once a day for several seconds. The painful paresthesias defined a region of his phantom foot that included the instep, toes, and occasionally the ankle. They had decreased in intensity but were constantly present and still painful. He felt that his phantom leg had "telescoped" slightly, estimating it to be 1-2 inches (2.5-5 cm) shorter than

his left leg. It was flexed at the knee in a fixed position whenever he was not wearing his prosthesis and otherwise assumed the postures of the artificial limb. He frequently experienced the illusion that the phantom leg penetrated solid objects such as the mattress when he lay supine in bed or on tabletops where he often rested his stump.

The subject had participated in Study 2 in which he had received electrical stimulation applied at his outer ears on one session and placebo on a control session. The results from both sessions showed statistically significant correlations between skin conductance at the stump, at the contralateral limb, and phantom pain intensity. The subject had also received several treatments of TENS applied to the contralateral leg when he was in the hospital, which had led to a reduction in the painful paresthesias. Based upon these outcomes, the effects of TENS applied to the contralateral leg and outer ears on stump skin conductance, temperature and PLI was examined. The present set of results was obtained 138 days post-amputation. By then the painful spasms had become less frequent. The paresthesias persisted at a less intense level which he considered annoying but no longer painful. They defined a smaller region of the phantom limb which included the outer three toes.

Experimental apparatus

Transcutaneous electrical nerve stimulation (TENS) was applied to the contralateral leg and outer ears using a two-channel Grass model S88 stimulator. Two Grass model SIU5 stimulus isolation units were used to isolate the subject from the stimulator. Bilateral ear stimulation (BES) was delivered through two silver earrings which gently clasped the subject's earlobes. Contralateral leg stimulation was delivered using two standard rubber TENS electrodes (Agar Electronics).

Stump skin conductance measurements were obtained using a portable Thought Technology biofeedback module with digital display (SC200T) and Ag/AgCl Beckman electrodes. The electrode paste consisted of a mixture of physiological saline and a neutral ointment cream having the recommended concentration of approximately 0.05 molar NaCl

(Fowles et al., 1981). Surface skin temperature (ST) measurements at the stump were obtained using a Yellow Springs Instruments (YSI) digital thermometer, Model 49TA, and a YSI Model 409A temperature probe.

The subject rated changes in perceived phantom limb intensity (PLI) by turning a dial which allowed 180 degrees of rotation. The 90 degree setting was labelled "USUAL", 0 degrees, "LESS", and 180 degrees "MORE". The dial was connected to a 1.35 volt mercury battery via a 10,000 ohm potentiometer and the output fed into a digital volt meter which registered 0 through 0.675 to 1.35 volts corresponding to the 0, 90, and 180 degree settings, respectively. Measurements were obtained on a continuous basis and digital displays, including a digital timer, were videotaped for later scoring.

Procedure

The subject was tested on two consecutive evenings. After he arrived at the laboratory, he was seated in a quiet room where the experimental procedures were explained to him and his informed consent was obtained. He removed his prosthesis and sat quietly for 20 minutes to allow the stump to adjust to the laboratory environment. Skin conductance electrodes and temperature probe were then placed on the distal portion of the stump approximately 5 cm from its end. Earring electrodes were placed in position and TENS electrodes were placed on the subject's lower left leg (over the soleus and gastrocnemius muscles). The subject was instructed in the use of the dial to monitor phantom limb intensity. He was told to pay close attention to his phantom limb and to turn the dial in the appropriate direction if he noticed any type of change in sensation. He was to indicate the magnitude of a change by the size of the angle of arc produced by turning the dial. Recording of physiological measures was initiated when the subject understood how to use the dial and had begun monitoring his phantom limb.

Design

Each session was divided into seven periods of nine minutes each: an initial resting baseline (B1), contralateral leg stimulation (CLS), a recovery baseline (B2), bilateral ear

stimulation (BES), a second recovery baseline (B3), combined CLS and BES (CLS/BES), and a final resting baseline (B4).

The subject received "placebo" on the first session and TENS on the second. The procedure on both sessions was identical except that on the placebo session, non-conducting leads connected the stimulator and isolation units so that the subject received no current. At the beginning of each stimulation period on both sessions the experimenter turned on the Grass stimulator which was within view of the subject. He was told that he might or might not feel the effects of stimulation and, when he did, he was to inform the experimenter. During stimulation periods of the TENS session, stimulation intensity was increased until the subject reported a strong but not uncomfortable sensation. Small muscle contractions at the stimulation sites on the left lower leg were visible during CLS and CLS/BES. During "stimulation" periods of the placebo session the experimenter slowly turned the dial that controlled the intensity, asking the subject to report if and when he felt the stimulation. After some time the experimenter stated that the current was at the appropriate level. On both sessions the subject was periodically asked if he could feel something and to indicate if it became too strong.

TENS at the outer ears was delivered, using the same procedure, at 4 Hz, 125 ms pulse duration and an intensity ranging between 20 and 24 volts. For comparative purposes, the same parameters for BES were the same as those in a previous study (Melzack & Katz, 1984). CLS was delivered at 100 Hz, 80μs pulse duration and an intensity ranging between 100 and 120 volts. These were the stimulation parameters which the subject had received during his previous course of contralateral TENS.

Psychophysiological data reduction

Psychophysiological data collected during each session were reduced in the following manner. The videotapes were reviewed and recorded values of SC, ST, and PLI were obtained at 2-second intervals for each 63 minute session. These values were averaged to

obtain mean levels for each minute. Mean values of PLI were transformed by subtracting a constant of 0.675 from each. This served to relocate PLI scores so that the 90 degree setting labelled "USUAL" took on a value of 0.0, and deviations from it, in the clockwise and counter-clockwise directions (corresponding to increases and decreases in PLI), had maximum values of ±0.675, respectively.

Results

Changes in phantom limb intensity

Figure 15 shows a minute-by-minute plot of mean PLI within the 7 periods on the TENS and placebo sessions. The results indicate that mean intensity of phantom limb paresthesias were significantly lower when TENS was applied to the contralateral leg (Periods CLS and CLS/BES) than when the subject received placebo stimulation. Furthermore, levels of PLI were significantly lower during CLS and CLS/BES when compared to the three baseline periods on the TENS session but not the placebo session. A detailed presentation of the statistical procedures and results of this analysis follows.

The data shown in Figure 15 were submitted to a 2 X 7 repeated measures ANOVA using Session (TENS and placebo) as the repeated measures factors and Period (B1 through B4) as the independent samples factor. The analysis revealed significant effects for Period (F (6, 56) = 10.55, $p < .0001$) and for the Period x Session interaction (F (6, 56) = 6.06, $p < .0001$). Simple main effects of the two-way interaction for Period during TENS and placebo were significant (F (6, 56) = 8.02, $p < .0001$ and F (6, 56)= 14.7, $p < .0001$, respectively).

In order to explore the nature of the differences in PLI within the TENS and placebo sessions the following set of 6 mutually-orthogonal contrasts were evaluated across the 7 periods in each session: Contrast one compares phantom activity during the four baseline periods with that during the three stimulation periods. Contrasts two, three and four compare B1 with B2, B3 with B4, and the joint effects of B1 and B2 with that of B3 and B4,

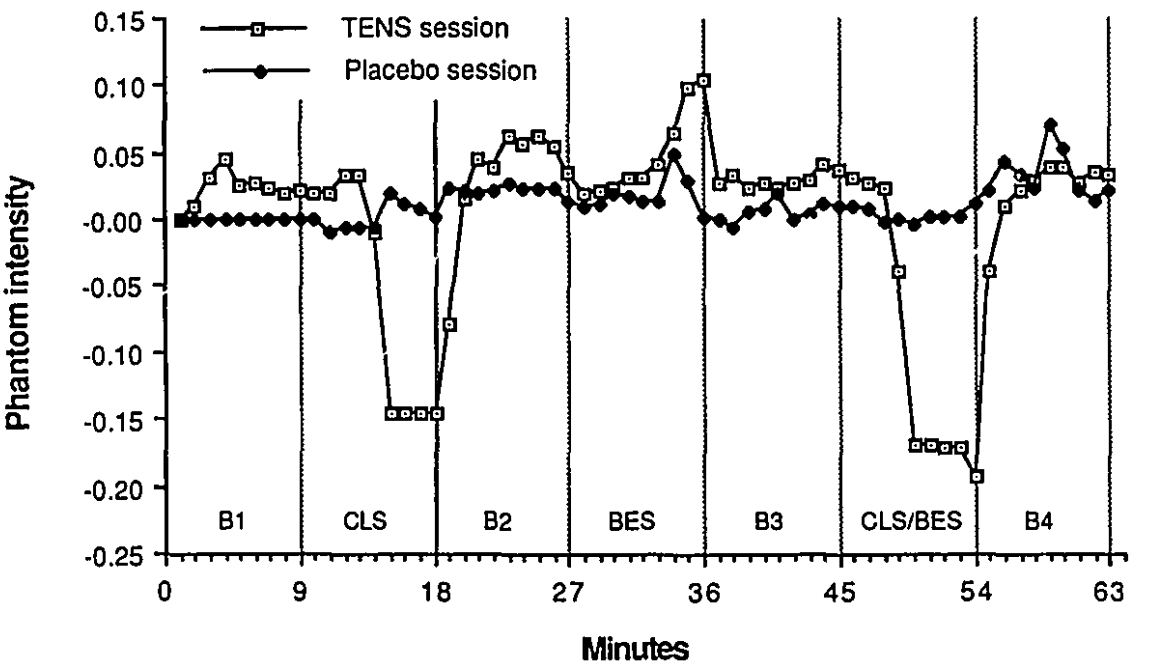


Figure 15. Mean subjective intensity of non-painful phantom limb sensations across the seven periods on the TENS and placebo sessions. Intensity scores have been transformed so that a value of 0.0 represents the subject's normal level of phantom paresthesias. Initial resting baseline (B1), contralateral leg stimulation (CLS), first recovery baseline (B2), bilateral outer ear stimulation (BES), second recovery baseline (B3), combined CLS and BES (CLS/BES), final resting baseline (B4).

respectively. Contrast five compares PLI during CLS/BES with the joint effects of CLS and BES, and the sixth compares CLS and BES. Using Scheffé's (1953) method for post hoc hypothesis testing, the first, fifth and sixth contrasts reached conventional levels of significance ($p < .05$) for the TENS session. These results indicate that 1) during electrical stimulation PLI were significantly reduced when compared with the baseline periods, 2) mean levels of PLI did not differ significantly across the baseline periods, 3) CLS/BES resulted in significantly lower levels of PLI than did the combined effects of CLS alone and BES alone, and 4) levels of PLI were significantly higher during BES than CLS. For the placebo session, quite a different pattern of results emerged. Contrasts two, three, and six reached significance indicating differences in PLI during the two first and two last baseline periods and significantly different levels during placebo BES and CLS.

Analyses of the simple main effect of Session at each of the 7 periods revealed that mean levels of PLI were significantly lower in the TENS session than the placebo session for CLS alone and CLS/BES , (F (1, 56) = 8.39, $p < .01$ and F (1, 56) = 24.7, $p < .01$ respectively), but did not differ significantly during the baseline or BES periods.

Changes in stump skin temperature

Mean skin temperature of the stump across the two sessions are displayed in Figure 16. The most striking aspect is the anomalous increase (of 3° C) from initial to final baseline periods during the placebo session. This contrasts with the more typical fluctuating pattern shown during the TENS session. Because of the obvious differences in ST during the initial baseline periods, and the anomalous increase during the placebo session, further analyses of these data would be misleading. Nevertheless, they are presented for subsequent reference.

Changes in stump skin conductance levels

Figure 17 shows stump skin conductance levels averaged every minute across the seven periods for the two sessions. Visual inspection of the data from the TENS session reveals a fairly consistent pattern of stump electrodermal activity across the baseline periods

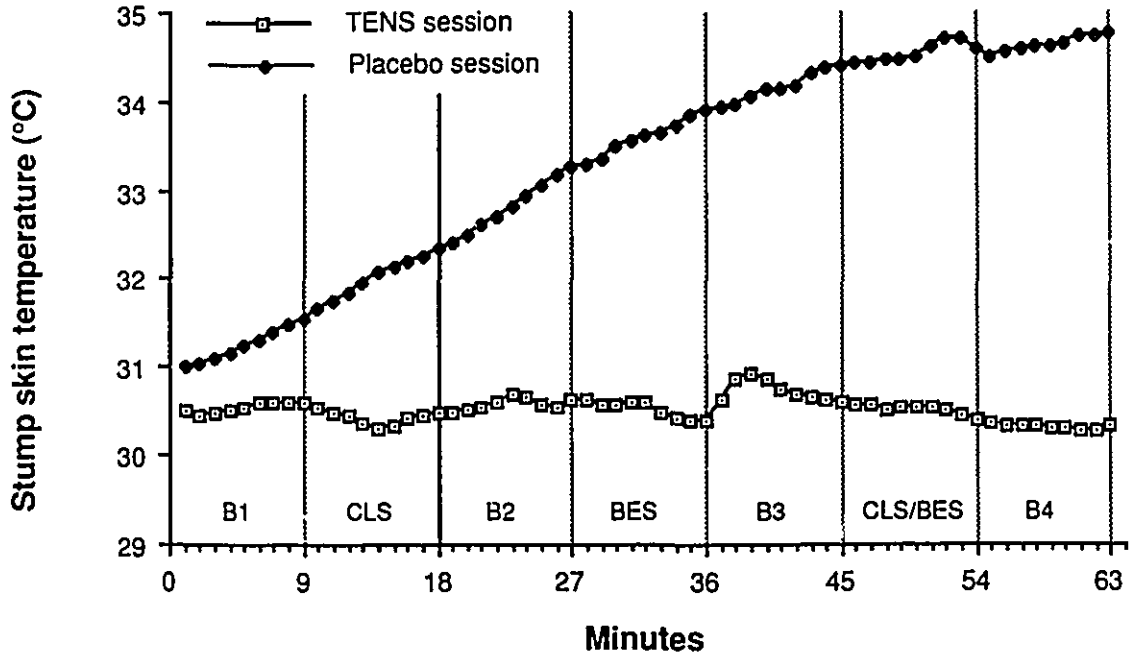


Figure 16. Mean stump skin temperature (°C) across the seven periods on the TENS and placebo sessions. Abbreviations as in Figure 15.

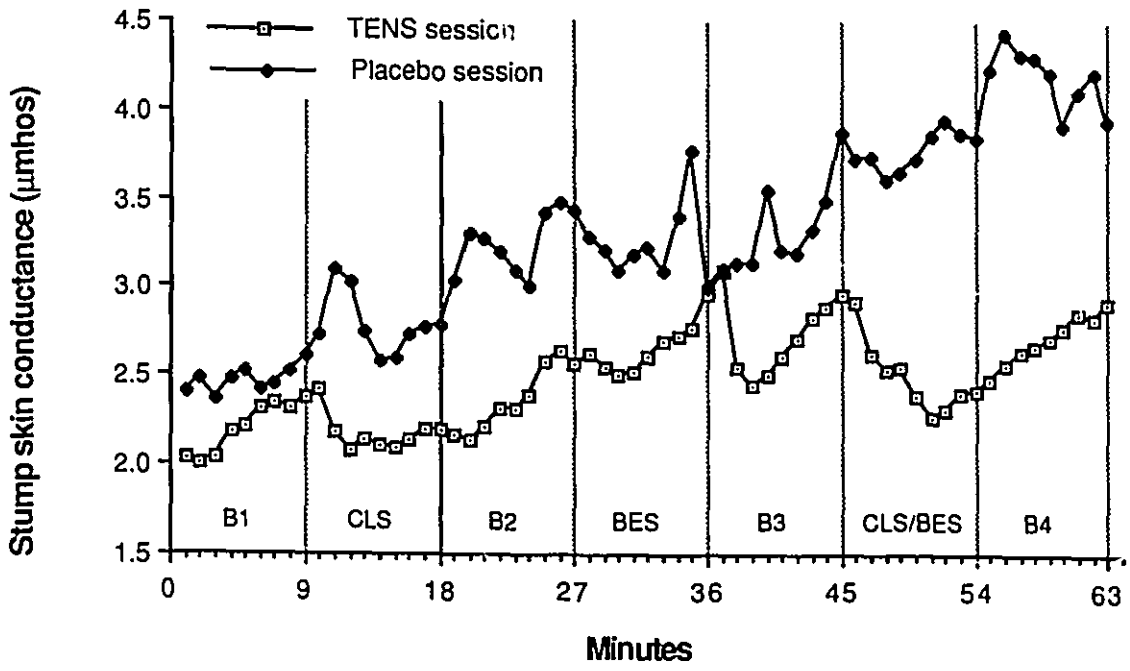


Figure 17. Mean stump skin conductance levels (µmhos) across the seven periods on the TENS and placebo sessions. Abbreviations as in Figure 15.

and a different but likewise consistent pattern during periods of stimulation. Stump SCL appear linear during the baseline periods and curvilinear during stimulation. This pattern is not exhibited during the placebo session.

In order to statistically describe and evaluate the pattern of electrodermal activity across periods and sessions, a separate stepwise regression analysis of SCL was performed for each period forcing the linear and quadratic components of time into the equation as predictors. Table 19 contains the coefficients of determination (r^2) for the linear and quadratic equations obtained from each analysis. Table entries represent the proportion of the total variance in stump SCL accounted for by the regression equation at that step. To simplify reading in the table, values of r^2 have been omitted when the unique contribution of the quadratic component to the overall regression (over and above the linear) is not significant (i.e., $p > .05$). Consequently, when reading in the table, a statistically significant linear regression indicates that a significant proportion of the total variance in SCL within the period under evaluation can be explained by a straight-line relationship with time and a statistically significant quadratic regression indicates that the relationship between SCL and time is well described by a one-bend curve.

It can be seen that during the TENS session, the best description of the relationship between stump SCL and time is a straight line for the baseline periods and a positively accelerated one-bend curve (see Figure 17) for the stimulation periods. The sole exception is Period B3, but Figure 17 shows that the main contribution to the significant quadratic regression for this period derives from the first minute which immediately followed bilateral ear stimulation (BES), and probably reflects a carry-over effect of BES into the following period (i.e., B3); when the first minute is excluded from the analysis the coefficient of determination increases significantly from .09 to .90 for the linear regression. Taken together these data indicate that the onset of electrical stimulation in periods CLS, BES, and CLS/BES is associated with an initial decrease in stump SCL followed several minutes later by an

Table 19. Coefficients of determination (r^2) for the stepwise regression analyses of stump skin conductance levels using linear and quadratic components of time as predictors.

		B1	CLS	B2	BES	B3	CLS/BES	B4
TENS session	Step 1[a] Linear	.90**	.11	.91**	.70**	.09	.60*	.97**
	Step 2[b] Linear + Quadratic	--	.74**	--	.95**	.63*	.91**	--
Placebo session	Step 1[a] Linear	.43*	.15	.33	.05	.55*	.50*	.49*
	Step 2[b] Linear + Quadratic	--	--	--	--	--	--	--

[a] df = (1, 8)
[b] df = (2, 8)
** $p < .001$, * $p < .05$

increase. On the other hand during the baseline periods SCL increase as a linear function of time and with what appears to be remarkably consistent slopes.

An examination of Figure 17 in conjunction with the coefficients of determination in Table 19 indicate that during the placebo session, the behavior of stump SCL is not tied to the onset and offset of CLS, BES, and CLS/BES as it is for the TENS session. The relationship between stump SCL and time is linear for several of the placebo session periods (e.g., B1, B3, CLS/BES, B4), although none of the stimulation periods shows a significant quadratic relationship.

Relationship between stump skin conductance levels and phantom limb intensity

Figure 18 shows the relationship between stump SCL and PLI on both sessions (Note that the skin conductance scale is different for Figures 17 and 18). The Pearson correlation coefficient calculated between these variables was significantly different from zero for both the TENS (r (61) = .41, $p < .001$) and placebo (r (61) = .52, $p < .0001$) sessions. These significant correlations indicate that in general, changes in stump SCL and PLI tend to occur in tandem, regardless of whether or not TENS is delivered. That is, changes in skin conductance correlate significantly with changes in phantom sensations both in TENS and in placebo sessions.

In an effort to determine the temporal relationship between these variables time lag correlations were computed using the raw (2-second) data. For both sessions, values of skin conductance were lagged 2, 4, and 6 seconds behind values of PLI and correlation coefficients were evaluated for each lag. This procedure was repeated, lagging values of PLI behind those of SC. None of the correlations improved (even minimally) the originally significant correlation. Thus, it is not known whether skin conductance responses preceded or followed changes in PLI.

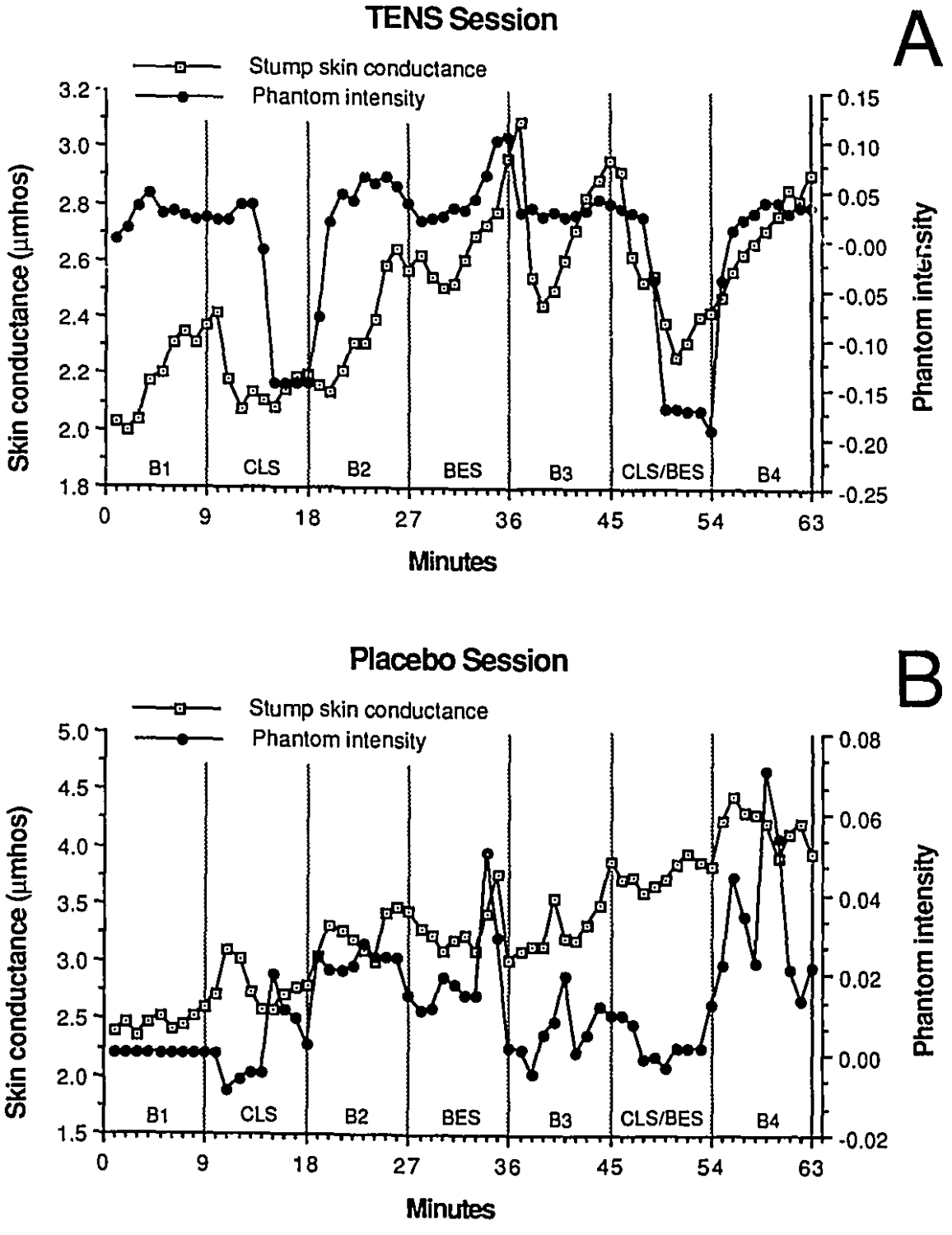


Figure 18. Mean stump skin conductance levels and subjective intensity of non-painful phantom limb sensations across the seven periods on the TENS (A) and placebo (B) sessions. Note the scale differences in skin conductance and phantom intensity between the two graphs. Abbreviations as in Figure 15.

Subjective reports

At the start of each session the subject was asked to describe what he felt in the phantom. Phantom sensations consisted of a "numbness" that defined the region of his outer three toes. Within this relatively small area sensations were not static, but continuously changing. He described these normal changes as rapid "waves" of numbness that increase and decrease the intensity of the involved phantom parts. Some of these "waves" were extremely subtle while others were much more noticeable.

At the beginning of the fifth minute of contralateral leg stimulation (CLS) on the TENS session the subject spontaneously remarked "I feel practically no phantom pain"[4] and explained that the numbness that defined his phantom toes was practically gone. Near the end of the fourth minute of CLS/BES on the same session he once again reported that the numbness in his phantom toes had practically gone and added that the phantom calf muscle was "tired" as if he had "walked a lot or done too much exercise". It was not unpleasant. According to the subject this phantom sensation was not in the same location as, or similar in quality to, the TENS electrodes stimulating his left calf. This "tired" sensation was not apparent during the final resting baseline period that followed. At the end of the TENS session the sensation of tiredness was reproduced with electrical stimulation applied to the contralateral leg alone (i.e., without concomitant ear stimulation).

Discussion

The results of this case study indicate that TENS applied to the contralateral extremity was significantly more effective than a placebo in decreasing the intensity of non-painful phantom limb paresthesias whereas electrical stimulation applied to the outer ears led to a non-significant increase. This latter outcome is consistent with an earlier study (Melzack & Katz,

[4]This subject, as well as other amputees we have encountered, referred indiscriminately to *all* phantom sensations as "phantom pain" even when not in pain, as subsequent verification revealed. Paradoxically, this is more often true of the patients who have little or no phantom pain at all.

1984) in which electrical stimulation applied to the outer ears failed to produce significant relief of chronic pain when compared to a control condition but led to reports of referred sensations in a large percentage of chronic pain patients. In that study, as in this one, electrical stimulation applied at the ears was delivered at a rate of 4 Hz and a pulse width of .125 msec. However, in Study 2, this subject had received TENS applied at the ears using the same frequency but a shorter pulse width of 100 μsec, and had shown a decrease in the intensity of his phantom limb pain not evident on the placebo session. These differences in the effects of electrical stimulation suggest that pulse duration may be the critical factor responsible the lack of significant pain relief in the Melzack and Katz (1984) study.

It is clear that TENS applied to the contralateral leg significantly reduced the intensity of phantom paresthesias well below the baseline levels. Whether these stimulation-induced changes in the intensity of phantom paresthesias reflect supraspinal (LeBars et al., 1979; 1983), segmental (Fitzgerald, 1982), or both types of mechanisms cannot be answered but the results of Fitzgerald's study seem particularly relevant. Fitzgerald demonstrated the existence of segmental contralateral control over dorsal horn cells in lamina 5 of the spinal rat. Electrical stimulation of the contralateral sural nerve, which contains cutaneous afferents only, produced excitatory effects in cells with inhibitory receptive fields on the contralateral lower leg or foot. These effects were obtained using A-fiber but not C-fiber stimulation. Inhibitory effects of contralateral A-fiber stimulation have also been observed (Brown, Kirk, & Martin, 1973; Mendell, 1966). Together , these studies may shed some light on the subject's reports of referred phantom sensations during TENS. On two occasions while receiving TENS at the contralateral calf, the subject stated that he became aware of a sensation in his phantom calf at the same time as he indicated a decrease in the intensity of the paresthesias in his phantom toes. It is assumed that under normal circumstances, the subject's perception of his phantom toes results from the lack of inhibitory control exerted over spinal cord cells subserving these regions, and the absence of his phantom calf is evidence of the redevelopment of this control. The effect of contralateral leg stimulation on spinal cord cells subserving the phantom might be

to facilitate inhibitory control in regions lacking it and disinhibit those for which it had already re-developed. This would explain the simultaneous but opposite effects the subject reported in the intensity of his phantom toes and calf.

The significant direct relationship between stump skin conductance levels and phantom limb intensity confirms the results from Study 2 which revealed correlations between peripheral sympathetic activity and paresthesias referred to the phantom. In the absence of evidence on the temporal relation between these two variables the nature of their association remains obscure.

The results of Study 3 implicate the cholinergic postganglionic sympathetic fibers as a mediator of the increases and decreases in the paresthesias reported by the subject. Bini et al. (1980a, 1980b) recorded multiunit sympathetic activity from human nerve fascicles and found that spontaneous activity in postganglionic sympathetic fibers to the smooth muscle of blood vessels and sweat glands is frequently followed by vasomotor and sudomotor activity in the form of vasoconstriction and electrodermal responses. Although sympathetic activity in one type of fiber (e.g., vasomotor) was often accompanied by activity in the other (i.e., sudomotor) this relation is not a necessary one and both complementary and opposite responses were recorded.

The results of Study 3 seem to reflect this potential for independent sympathetic vasomotor and sudomotor activity as indicated by the apparent dissociation of electrodermal activity and skin temperature on the placebo session. Phantom limb intensity and skin conductance levels correlated significantly on both sessions whereas skin temperature appeared to covary with phantom limb intensity (and inversely with skin conductance) only on the session in which TENS was given. It is possible that a more sensitive measure of stump blood flow might have shown the latter relationship on the placebo session as well.

Nevertheless, the significant correlations between stump skin conductance and phantom intensity implicates the cholinergic component of the postganglionic sympathetic outflow as a possible mediator of the relationship between the two variables. Very little has

been done in the field of pain and cholinergic chemosensitivity (see Nathan, 1980, and Ochoa et al., 1985) but a study by Diamond (1959) showed that regenerating nerves are abnormally sensitive to the effects of acetylcholine and discharge rapidly in its presence.

According to this hypothesis, spontaneous bursts of activity in sudomotor sympathetic fibers located in the stump result in sweat gland activity and heightened skin conductance levels. At the same time, or shortly after, afferent fibers in stump neuromas would increase their rate of firing due to the liberation of acetylcholine. These impulses are referred to certain regions of the phantom as increases in paresthesias. Thus, the moment to moment fluctuations in phantom limb intensity (described as "waves" by the subject) may in part represent a sympathetic efferent-somatic afferent cycle. Increases in phantom limb intensity would follow bursts of sympathetic activity due to neurotransmitter release and decreases would correspond to periods of relative sympathetic inactivity.

It is important to note that skin conductance levels at the stump correlated significantly with phantom limb intensity even on the placebo session. This correlation implies that the relationship between skin conductance and phantom intensity cannot be due to TENS since TENS was not delivered on the placebo session, but that when TENS is applied contralaterally, both stump skin conductance and phantom intensity decrease. Although stump skin conductance began to drop by the first minute after CLS and CLS/BES, it was several minutes before the subject reported a change in phantom limb intensity (Figure 18a). These lags did not appear to be the result of a lapse in the subject's concentration due to the distracting influence of TENS onset, for similar lags were not evident during the same periods on the placebo session when the subject was equally distracted (Figure 18b). Furthermore, this subject, and others tested in a similar manner (unpublished data) showed that they are quite capable of continued monitoring of phantom sensations during TENS (and placebo) onset by shifting their attention from the phantom to the site of the TENS electrodes, and back again.

It is difficult to account for the concomitant changes in electrodermal activity and phantom sensations from TENS to baseline periods. The pattern of skin conductance was consistently linear during the baseline periods indicating a progressive increase in sympathetic sudomotor activity. In contrast, it was consistently curvilinear during periods of electrical stimulation indicating an initial decrease followed by an increase in sudomotor responses. Correlates of peripheral autonomic activity during TENS have been reported but the results are inconsistent (Abram, 1976; Abram, Asiddao, & Reynolds, 1980; Dooley & Kasprak, 1976; Ebersold, Laws, & Albers, 1977). Spinal cord stimulation in patients with peripheral vascular disease has been found to produce pain relief as well as a profound increase in peripheral blood flow and skin temperature (Groth, 1985; Meglio et al., 1981). The application of TENS to the contralateral limb may lead to an initial reduction in sympathetic sudomotor activity in the stump, followed by decreased primary afferent discharge, and a reduction in phantom limb paresthesias.

STUDY 4: SOMATOSENSORY MEMORY FOR PAIN AFTER AMPUTATION

The final study of the thesis is a retrospective examination of the similarity between pre-amputation pain and phantom limb pain. As reviewed in the Introduction, studies of groups of amputees report the incidence or prevalence of somatosensory memories after amputation but with little information about the types of pains which recur. Case reports provide these descriptive details but without information regarding the frequency of their occurrence.

Objective

The purpose of this study is to determine, in a random sample of amputees, the frequency with which different types of pre-amputation pains are reported to be re-experienced as phantom pains after amputation.

Methods

Sample

Sixty-eight amputees (45 male, 23 female), ranging in age from 23-79 years (mean 58.7 years) volunteered to participate. The 28 subjects from the previous study are included among the 68. There were 57 unilateral, one double ipsilateral, and 10 bilateral amputees for a total of 79 amputated limbs. The major reason for amputation was peripheral vascular disease (PVD) in 43 subjects (52 limbs) 24 of whom (28 limbs) also had diabetes mellitus (DM). The next most frequent cause of amputation was accident, which accounted for 13 subjects (15 limbs), followed by tumour in 5, radiation damage in 2, osteomyelitis in 1, and polio in 1. The mean number of years since amputation was 4.9 (range 20 days to 46 years). Subjects were recruited from the same sources reported in Study 2 and from the Ste. Anne's Veterans Hospital "Liaison" Center. Informed consent (see Appendix C-1) to participate was obtained from all subjects.

Procedure

Subjects were interviewed as part of a larger project studying painful and non-painful phantom limbs. One portion of the interview examined the relationship between pain experienced in the limb before amputation and the subsequent development of phantom limb pain. Subjects were asked whether or not they had experienced pain in the limb before it was amputated. They were asked to specify the type(s) of pain and its duration. Subjects with PLP were asked whether the PLP was similar in quality, location, or intensity to the pain they experienced *prior* to the amputation. The term *somatosensory memory of a prior pain* (SMp) will be used when referring to these types of phantom phenomena.

Subjects were also asked whether the PLP was similar to any other pain or lesion they may *formerly* have had, but that was not present or had healed before the amputation was performed. The term *somatosensory memory of a former pain* (SMf) will be used when referring to these phantom phenomena. The essential difference between the two types of SM is one of perceived continuity: SMps are described as the persistence of the pre-amputation pain and resemble sensations (and/or pains) that were present in the limb up to the time the amputation was performed. Subjects with SMps often report that when first recovering from the anesthesia they believed (and felt) that the amputation had not been done because they continued to feel their limb as it was prior to the anesthesia. SMfs are defined by a discontinuity or a pain-free interval between the time the pain (or sensation) was last experienced and the amputation.

Following the interview subjects completed the following personality inventories and questionnaires: Eysenck Personality Inventory (EPI), Wesley Rigidity Questionnaire (WRQ), Beck Depression Inventory (BDI), and the Spielberger Trait Anxiety Inventory (STAI-T).

Inclusion criteria for somatosensory memories

Retrospective reports such as these pose certain problems with regard to their reliability and verifiability. These problems include the (a) reliance on the subject's memory to

determine the relationship between the pre-amputation pain and phantom limb pain without an independent source of evidence to confirm or disconfirm the retrospective report, (b) possibility that some subjects with phantom limb pain will try to explain the pain by likening it to a past pain, and (c) perceived demand characteristics to respond to the investigator's questions in a favorable way.

The following procedures were implemented to minimize the influence of these factors. Reports from subjects who were vague or appeared not to remember but "thought" that there was a similarity were excluded. Only those subjects who claimed that they were certain that the phantom pain was the same as, or very similar to, the pre-amputation pain were questioned further. After this initial indication that a similarity existed, two conditions had to be met in order for phantom phenomena to qualify as somatosensory memories. Subjects had to indicate that the (a) quality and (b) location of the pre-amputation and phantom sensations were the same. This was readily established for exteroceptive pains associated with a pre-amputation lesion which had had a visual component, for these were frequently described in one statement (e.g., "I can feel my ingrown toenail", "I feel the hole where the ulcer was", etc.). However, even these reports were followed up with questions to ascertain the nature of the similarity in pain.

If only the quality *or* the location was reported to be the same, the PLP was not included as a SM. Subjects were also asked to rate the similarity in intensity of the pre-amputation and phantom pains but this information did not serve as a criterion for inclusion. Since the intensity of a phantom sensation appears to be analogous to the strength or vividness of a "psychological" memory it was reasoned that intensity ratings should not enter into the process of determining whether a phantom pain qualified as a SM. Memories tend to fade with time elapsed since the original event yet they still represent that event.

In order to minimize the demand characteristics of the situation, subjects had been informed that the study was investigating non-painful and painful phantom limbs. Equal attention was paid to reports of pre-amputation pains which did not recur in the phantom limb

as those which did. The investigator avoided asking leading questions and although subjects were encouraged to elaborate their responses they were not prompted. Inconsistencies which occasionally arose over the course of one or more interviews with a subject were followed up with further questions to clarify the nature of the similarity in pains.

Results

Table 20 contains a case-by-case description of some of the relevant clinical and demographic variables related to amputation for the 68 subjects. Also included is specific information about the type and location of the pre-amputation pain reported and the pains which were later experienced in the phantom limb as SMs (both SMp and SMf). As well, the table contains descriptions of non-painful pre-amputation experiences which were reported to have recurred as phantom phenomena following amputation. Finally, the table includes reports of SMs which had been present for some time after amputation but had disappeared by the time the interview took place.

Forty-one of the 68 subjects (60.3%) reported that they had experienced at least one type of pain in their limb at the time of the amputation.

Twenty-nine subjects reported SMs which resembled painful and non-painful pre-amputation sensations. Twenty-five of the 29 reported SMps, three reported only SMfs and one reported a SM of a non-painful pre-amputation sensation. Together, the twenty-nine subjects produced a total of 55 SMs. Thus, 42.6% (29/68) of the total sample reported at least one somatosensory memory (SMf or SMp) which resembled, in quality and location, a painful or non-painful pre-amputation sensation experienced at some time before the limb was removed. Sixty-one percent (25/41) of those who reported having had pre-amputation pain at, or near, the time of amputation indicated that at least one of these pains continued to persist or recur in the phantom as SMps.

In addition, 21 subjects (all with PVD or PVD/DM) had a history of intermittent claudication characterized by severe leg pain brought on by walking, and one of them claimed

Table 20. Demographic and clinical variables related to amputation listed for each the 68 subjects, including status with respect to any type of phantom limb pain (PLP), age, sex, type and level of amputation, reason for amputation, and the number of days elapsed since amputation. A short description is provided of the type, location, and duration of pre-amputation pains experienced and those which were subsequently reported as somatosensory memories referred to the phantom limb after amputation. Also included is a description of non-painful sensations which were experienced as phantom phenomena as well as instances of past somatosensory memories which had been present after amputation but had disappeared by the time of the interview.

Case	PLP	Age	Sex	Type and level of amputation*	Reason for amputation+	Days since amputation	Type and location of pre-amputation pain	Duration of pre-amputation pain	Somatosensory memory referred to phantom limb following amputation
101	yes	65	M	R-A/K	tumour	518	none	—	no
							shotgun wound to (R) calf at age 15	—	no
102	yes	50	M	R-B/K	osteomyelitis	2400	osteomyelitis wound on medial surface of shin just above ankle	since age 7 but worse for last 2 years since being kicked	Continues to feel the same pain about once every 7 weeks. Also feels the "hole" or ulcer just above the ankle which never healed and the sensation of the bandages which wrapped the wound. When the pain comes on he still reaches down and "cups" the spot where the bandages and wound used to be.
103	yes	65	M	L-A/K	PVD	4380	1. intermittent claudication in (L) calf, swollen foot	2 months	no
	yes			R-A/K	PVD	2555	2. burning, throbbing pain in toes up to instep of (R) foot	2 months	"There's something very similar in the right (phantom foot). The burning and throbbing is excruciating".
104	yes	66	F	R-FQ	radiation damage	1460	1. Arm swollen, large and heavy	—	Years after receiving radiation therapy for breast cancer her arm became swollen, heavy and painful. When sitting, she would rest her arm in her lap. This is the position which her phantom arm now assumes whenever she sits down. She also feels the same painful swelling and heaviness in her phantom arm and hand.
							2. fractured wrist at age 14	—	no
105	yes	60	M	L-Symes	work accident	548	none	—	
106	yes	61	M	R-FQ	work accident	2920	none	—	
107	no	63	M	L-B/K	PVD/DM	1520	gangrene in toes	2 weeks	no

Table 20 continued

Case	PLP	Age	Sex	Type and level of amputation	Reason for amputation	Days since amputation	Type and location of pre-amputation pain	Duration of pre-amputation pain	Somatosensory memory referred to phantom limb following amputation
108	yes	62	F	L-A/K	PVD/DM	2190	1. cramping, burning, stabbing pain in toes of (L) foot	2 years	The phantom pain in "the three toes on the left that went black with gangrene is the same".
							2. fractured (L) great toe 15 years prior to amputation	—	Tripped on railway tracks and fractured great toe on two occasions. She reports feeling the same throbbing pain in the phantom toe as she had when it was broken. She never gets this pain in the (R) phantom toe.
							3. Arthritis in (L) knee	—	She reports feeling an "arthritic pain" in the (L) phantom knee during damp weather which is the same as the pre-amputation pain.
	yes			R-A/K	PVD/DM	1460	4. Pain in toes and back of (R) leg	2 years	The pain in the phantom is the same but not as intense as it was originally.
109	no	65	F	R-B/K	PVD	70	pain in foot	2.5 years	no
110	yes	74	M	R-A/K	PVD	1700	intermittent claudication	—	no
111	yes	59	F	L-A/K	tumour	285	1. burning pain in leg	15 years	no
							2. site of surgical excision on lateral surface of (L) lower leg	—	Once every two weeks or so she feels the pain and the wound as they were before the amputation. The phantom pain not as intense and does not last as long as the original pain.
							3. bedsore on foot	2 months	From time to time she is aware of the sensation of the bedsore.
112	yes	71	M	R-B/K	PVD/DM	180	missing	6 months	no
113	yes	76	F	L-A/K	PVD/DM	2615	1. intermittent claudication	1 year	no
							2. hot, burning at site of surgery on lateral surface of (L) lower leg	2 months	Occasionally feels the "hole"on her phantom lower leg and the same hot burning pain she used to feel when the bandages were changed.
114	no	60	M	R-A/K	PVD/DM	1580	1. none in (R) leg	—	no
	yes			L-A/K	PVD/DM	1215	2. intermittent claudication and rest pain in calf	6 months	no
115	yes	52	M	L-B/K	PVD/DM	1004	diabetic ulcers and gangrene on foot	3 weeks	no

Table 20 continued

Case	PLP	Age	Sex	Type and level of amputation	Reason for amputation	Days since amputation	Type and location of pre-amputation pain	Duration of pre-amputation pain	Somatosensory memory referred to phantom limb following amputation
116	yes	72	M	L-A/K	PVD	1551	intermittent claudication	1 year	Continues to feel the same pain in his phantom calf when walking.
117	yes	44	F	L-A/K	PVD/DM	577	intermittent claudication	5 years	no
118	no	69	F	R-B/K	PVD/DM	1337	none	—	no
119	no	56	F	L-A/K	PVD	1004	1. intermittent claudication	5 years	no
							2. gangrene in toes	—	No longer by the time of the interview but she reports that after the amputation, and over the following 18 months, she felt the same pre-amputation pain in the toes, "especially the big one, like if you held a match under it".
120	yes	73	M	R-HQ	tumour	335	none	—	no
121	no	56	F	R-B/K	PVD/DM	335	pain in region around toe. Developed dry gangrene	1 week	no
122	no	58	M	L-A/K	PVD	942	intermittent claudication in calf	1 month	no
123	yes	70	M	R-A/K	PVD/DM	2920	1. diabetic ulcer on (R) instep	4-5 months	no
	yes			L-A/K	PVD/DM	1065	2. diabetic ulcer on (L) great toe	2 months	no
124	no	60	M	R-A/K	PVD	1460	intermittent claudication	3 months	no
	no			L-A/K	PVD	1098	intermittent claudication	3 months	no
125	yes	72	M	L-B/K	PVD/DM	121	none	—	no
126	yes	75	M	L-A/K	PVD	5931	1. lesion from bypass on (L) thigh	1 year	no
	yes			R-A/K	PVD	5475	2. pain in (R) leg	2 months	no
							3. frequently had calf cramps in both legs prior to the amputations	—	He reports getting calf cramps in both phantom limbs once every 6 weeks. The cramps are just as intense as the original pains used to be.

Table 20 continued

Case	PLP	Age	Sex	Type and level of amputation	Reason for amputation	Days since amputation	Type and location of pre-amputation pain	Duration of pre-amputation pain	Somatosensory memory referred to phantom limb following amputation
127	yes	71	F	L-A/K	car accident	1186	1. cannot remember	—	no
	yes			R-A/K	car accident	1186	2. cannot remember	—	no
128	no	55	M	L-A/K	PVD	5475	1. intermittent claudication entire leg painful	2 weeks	no
	no			R-A/K	PVD	3924	2. none in (R)	—	no
129	yes	54	F	L-B/K	PVD/DM	219	1. intermittent claudication in calf and toes	2 years	no
							2. ingrown toenail (inner margin of great toe)	1 month	Approximately twice a week she feels a mild burning pain accompanied by the sensation of her toenail digging into the skin. This lasts from 30 seconds to 1 minute and is not as intense as the original pain "It's not really a pain but an aggravation".
							3. tight, vice-like pain around ankle	6 months constantly	At least once a day lasting 2 or 3 minutes she feels the same sensation in the phantom ankle but it is less intense. Changes in weather are associated with increases in this pain just as they were prior to the amputation.
							4. corn on small toe 1 year prior to, but not at the time of, amputation	—	Periodically continues to feel the corn for short durations. As with the ingrown toenail the sensation is more an aggravation than a pain.
130	no	60	M	R-B/K	PVD/DM	91	1. diabetic ulcer on second toe but not painful	—	no
							2. numbness in lower leg following accident	35 years	The sensation that he has now in the phantom foot and leg is the same as before.
131	yes	79	F	R-B/K	PVD	20	1. burning in toes and ball of foot	1 month	no
							2. painful ulcers on lateral surface of foot	—	no
							3. frequently suffered from foot and calf cramps prior to the amputation	—	no
							4. pain on instep	1 month	no

Table 20 continued

Case	PLP	Age	Sex	Type and level of amputation	Reason for amputation	Days since amputation	Type and location of pre-amputation pain	Duration of pre-amputation pain	Somatosensory memory referred to phantom limb following amputation
132	yes	62	M	R-A/K	PVD/DM	91	1. intermittent claudication	3 years	no
							2. burning under toes, ball of foot and heel	3 years but "excruciating" 2 months prior	At least 3 times a day for variable periods he feels the same pain only considerably less intense.
							3. stump pain from initial B/K amputation	48 days	Once a day or more often he feels the pain from the "first cut". This pain lasts for up to several hours and feels as the stump did after the first amputation with pain primarily in the region of the incision.
							4. stump pain from subsequent high B/K revision	37 days	He also feels phantom stump pain from the "second cut". Neither phantom pain is as intense as the original pain. He reports having experienced the stump pains from both amputations and the phantom foot pain (described above) simultaneously. At the time of the interview he reported that his phantom foot felt like it was "comfortably in a boot or a shoe".
133	no	55	M	L-B/K	Buerger's disease	2454	intermittent claudication	4 years	no
	no			R-B/K	Buerger's disease	2067	intermittent claudication	4 years	no
134	yes	63	M	L-B/E	motorcycle accident (brachial plexus injury)	15695	cannot remember	—	no Until 10 years ago he experienced his phantom arm in a rigidly fixed position that resembled the position it had been in at the time of the motorcycle accident with his hand clenched as if it were still gripping the handlebars. He also reported the frequent sensation of "blood dripping down the phantom hand". Both sensations have subsequently disappeared.
135	yes	63	M	R-B/K	PVD/DM	44	1. Developed friction blister that ulcerated and became infected leading to amputation. Because of diabetic neuropathy this was not painful.	13 weeks	no
							2. Ingrown toenail subjected to periodic "home" surgical remedies over the years.	42 years	Frequently feels the toenail digging into the toe .This sensation lasts several minutes and is "more of an annoyance rather than a pain".

Table 20 continued

Case	PLP	Age	Sex	Type and level of amputation	Reason for amputation	Days since amputation	Type and location of pre-amputation pain	Duration of pre-amputation pain	Somatosensory memory referred to phantom limb following amputation
135 continued							3. Callus on sole of foot	35 years	Feels the "area of dry skin that sort of tightens. That's the callus. It's not really a pain, more of an annoyance."
136	yes	68	M	R-B/K	PVD/DM	476	1. intermittent claudication	12 years	no
							2. gangrene in great toe	2.5 months	Approximately once a week for several minutes at at time he feels the same (but less intense) burning pain in the phantom toe.
	yes			L-2 toes	PVD/DM	64	3. gangrene in great toe	10 days	About once a day he feels the same pain in his phantom toe. "It is very painful, like a kind of burning in my big toe". He also reports that when it hurts he has a mental image of the toe, discolored and black, just as it was prior to the amputation.
137	yes	41	F	R-B/K	PVD	57	1. ulcer on great toe	1 year	She feels the phantom pain is in every respect the same as the original pain. It was "the worst pain of my life. I'm feeling the hole is there and it's paining me. It feels like he (the doctor) is cleaning out the hole and packing in medication and cotton".
							2. "blood clot" on lower calf	—	Feels the same swelling and bulging sensation accompanied by periodic jabs of pain similar to the pre-amputation pain.
							3. pain in ankle	1 .5 years	Feels this pain 2-3 times per day in phantom ankle.
							4. Unable to move great toe	—	Reports being unable to move phantom great toe but is able to move the other ones.
138	yes	65	M	L-A/K	PVD	21	1. intermittent claudication	5 years	no
							2. several extremely painful ulcers on medial and lateral surfaces of ankle	1 year	no
139	yes	53	M	R-A/K	PVD	2590	1. intermittent claudication	2 years	No longer by the time of interview but for a period of six months following amputation he periodically re-experienced the pain of intermittent claudication in his phantom calf when sitting or resting but not when walking.

Table 20 continued

Case	PLP	Age	Sex	Type and level of amputation	Reason for amputation	Days since amputation	Type and location of pre-amputation pain	Duration of pre-amputation pain	Somatosensory memory referred to phantom limb following amputation
139 continued							2. had catheter for drainage inserted in posterior (R) calf before amputation	missing	No longer by the time of interview but over a two month period following amputation he continued to feel the sensation of the incision from which the catheter protruded.
140	yes	70	M	L-A/K	PVD	3102	1. intermittent claudication	8 years	no
							2. ulcer in web between 4th and 5th toe	2 months	Approximately every two months feels the same "steady, gnawing pain" as before, but not quite as intense.
	yes			R-A/K	PVD	1945	1. intermittent claudication	10 years	no
							2. ulcer in web between first two toes	2 months	Same as (L) phantom pain.
E01	no	49	M	L-A/K	PVD	242	1. ulcer on knee that wouldn't heal	—	no
							2. calf and foot in pain, foot swollen	2-3 weeks	no
E02	no	62	M	R-A/K	PVD	1095	none	—	no
E03	no	58	F	L-B/K	PVD/DM	544	ulcer under great toe	3 months	no
E04	yes	59	F	R-FQ	radiation damage	2920	arm swollen, tight	2 months	The sensation in phantom arm is identical: "A feeling of being overstuffed, tight, of oedema and swelling". Occasionally she has a "pleasant feeling of draining" which she used to feel when her arm arm was drained on numerous occasions prior to the amputation.
E05	yes	61	M	R-A/K	PVD/DM	882	1. intermittent claudication in calf and rest pain	6 years	no
							2. diabetic ulcer on foot	—	no
							3. gangrene from toes to calf	3 hours	His phantom toes "sting and burn" as before but the pre-amputation pain was worse.
E06	yes	60	F	R-A/K	tumour	1977	1. burning from lower leg to great toe	1 year	Frequently she feels the same burning pain down the phantom leg to the great toe. The

Table 20 continued

Case	PLP	Age	Sex	Type and level of amputation	Reason for amputation	Days since amputation	Type and location of pre-amputation pain	Duration of pre-amputation pain	Somatosensory memory referred to phantom limb following amputation
E06 continued							2. pain from wound on medial surface of thigh following surgical operation	2 months	At least once a week she feels the same pain. "It was the most sickening pain I 've ever had. It would affect my stomach, the pain was so bad ... The phantom pain is the same, but not as bad". In addition to the pain she also feels the "hole" or wound in her thigh as it felt after the surgical excision but prior to the amputation.
E07	yes	31	F	R-B/K	car accident	2099	none		no
E08	no	53	M	L-B/K	arterial thrombosis	121	stabbing pain on lateral surface of foot and small toe	15 days	He constantly feels a less intense version of the painful stabbing which he describes as a non-painful "gentle pressing feeling" in the same locations of the phantom.
E09	yes	40	M	R-B/K	motorcycle accident	91	1. ingrown toenail 2. throbbing around ankle	— 9 months	no The throbbing pain in the phantom ankle feels exactly as it did before the leg was removed, but "nowhere around the level" of intensity. This sensation has occurred approximately 10 times since the amputation.
E10	no	66	M	L-A/K	PVD	153	1. missing 2. ingrown toenail	2 months —	no no
E11	yes	54	F	R-A/K	PVD/DM	730	1. intermittent claudication 2. gangrene on 2nd and 3rd toes	4 years —	no no She feels that her "whole (phantom) leg is wrapped in cotton and very, very uncomfortable". On one occasion she had the feeling that the phantom foot was wearing a "white sock and a black patent leather shoe with straps".
E12	yes	73	F	L-A/K	PVD	669	missing	3 months	no

Table 20 continued

Case	PLP	Age	Sex	Type and level of amputation	Reason for amputation	Days since amputation	Type and location of pre-amputation pain	Duration of pre-amputation pain	Somatosensory memory referred to phantom limb following amputation
E13	no	68	M	L-B/K	PVD/DM	1430	diabetic ulcer on medial surface of foot near great	1 year of steady pain	For approximately 1 year after the amputation he continued to feel the same painful ulcer, "that was bigger than a silver dollar". At the time of the interview, he could reproduce the sensation of the ulcer by concentrating on his phantom limb but unless he did so, it remained out of his awareness.
E14	yes	31	F	L-B/K	motorcycle accident	41	cannot remember but was told most of the skin and flesh on the sole of her foot had been torn away in the accident	—	No, although she "thinks" that the cramp-like pain she feels in the phantom arch is related to the state of her foot at the time of amputation.
E15	yes	65	M	L-A/K	PVD/DM	137	severe pain down length of leg to great toe	1.5 years	no
E16	yes	54	M	L-A/K	PVD	1065	1. sole of foot was red and tender 2. great toe extremely painful and twisted on top of second toe 3. knee bent at slightly less than 90 degrees which he was unable to straighten	1-1.5 years 3 months 11 days 11 days	no Ever since the amputation he has felt both the phantom toe and knee in their pre-amputation postures. The phantom knee is fixed in the bent posture regardless of his overall body position. His attempts to straighten out the phantom lead to a burning pain in the toes. Neither phantom pain is as intense as the original pre-amputation pain.
E17	no	55	M	R-A/K	car accident	1035	none	—	no
E18	no	54	M	L-B/K	work accident	2008	missing	2 years	no
E19	yes	74	M	L-A/K	polio	16790	none	—	no

Table 20 continued

Case	PLP	Age	Sex	Type and level of amputation	Reason for amputation	Days since amputation	Type and location of pre-amputation pain	Duration of pre-amputation pain	Somatosensory memory referred to phantom limb following amputation
E20	yes	23	M	R-A/K	motorcycle accident	61	1. At the accident scene the calf of his injured leg began to cramp.	minutes	Feels the same sensation in phantom calf
							2. At the accident scene his boot was cut to free his foot. The sensation was of a removal of constrictive pressure and a cool feeling on his foot.	minutes	On several occasions he has noticed the same sensation of a removal of constrictive pressure from his foot as if it had just been freed from a tight boot. The cool sensation is not part of the phantom experience. He also experienced "growing pains" on the lateral surface of his phantom lower leg which he originally felt on numerous occasions as an adolescent 8 years ago.
E21	no	68	M	L-B/K	PVD/DM	91	none	—	no
E22	yes	62	M	L-B/K	PVD/DM	143	1. intermittent claudication	2-3 years	no
							2. burning pain in calf, shin, sole of foot and toes	6 months	Occasionally has the same burning sensation only not as strong as the pre-amputation pain.
E23	yes	33	F	R-A/K	arterial thrombosis	36	1. burning pain in toes that spread to foot and lower leg as time progressed	10 days	Continues to feel the burning pain in the same location about 10 times a day. The phantom pain is the same intensity as the original pain.
							2. following thrombosis she required emergency surgery of calf involving a 6" incision performed without anesthetic. She felt a pinching sensation as the skin on her calf split open.	10 days	Two weeks after amputation the patient first felt the same pinching or "splitting" sensation referred to the skin of the phantom calf where the incision was made. Since then this has occurred 5-6 times a day each lasting up to 10 seconds. Lately, the sensation has begun to change and is now followed by a "numbness" in the same region of the phantom calf.
E24	yes	64	F	R-A/K	tumour	7355	had a discomforting pain in her calf when she walked. The calf would feel tight and swollen "as if the skin would be pulled".	8 years	Periodically feels the same tight, swollen pain in the phantom calf. The phantom sensation is not as intense as the original pain and occurs when she is sitting or standing, but not while she walks.
E25	yes	25	M	R-B/E	electrical burn	1966	had throbbing, cramping pain in his hand and wrist	1 day	The phantom pain is the same but not as intense as the original pain.
	yes			R-A/K	electrical burn	1973	had throbbing, cramping pain in his leg from the knee down	7 days	The phantom pain is the same but not as intense as the original pain

Table 20 continued

Case	PLP	Age	Sex	Type and level of amputation	Reason for amputation	Days since amputation	Type and location of pre-amputation pain	Duration of pre-amputation pain	Somatosensory memory referred to phantom limb following amputation
							in his leg from the knee down		intense as the original pain
E26	yes	60	M	L-A/E	arterial thrombosis	89	Ischemic pain in hand. Fingers were cold, fingernails blue, and his hand was weak.	4 days	no
E27	no	32	F	L-A/K	car accident	6731	none	—	no
E28	yes	45	M	L-A/K	motorcycle accident	470	1. Beginning the day of the accident and becoming more intense with time he developed a burning causalgic pain in his toes and foot. The slightest touch from the bed sheets would set off an excruciating pain.	18 months	He continues to feel the same "steady burning" confined to the toe region "like someone's taking a pair of pliers and pulling off the nails"
							2. Required emergency surgery on lateral lower leg performed without anesthetic. "When he (the doctor) opened it up, it burned so much ... I fainted away." The wound was initially 7.5 " long and 2.5" wide.	6 months	About once a day he feels the wound as it felt prior to the amputation but the phantom pain is usually not as intense. However, occasionally he is awakened at night in a state of panic, drenched in sweat, with excruciating phantom pain from the old wound. At these times he also feels that "the doctors and nurses are there working on it". While in hospital approximately three months prior to the amputation, he remarked to his doctors that he felt his leg to be 12"-15" shorter than where it lay on the bed. The surgical wound persisted in this "telescoped" representation of his limb which has remained unchanged even after the amputation.

*Type and level of amputation is presented according to the side, left (L) or right (R), followed by the level, above (A) or below (B) the major limb joint of the lower or upper extremity, either the knee (K) or elbow (E). Thus a listing of R-B/K refers to a right-sided, below-the-knee amputation. FQ and HQ refer to forequarter and hindquarter amputations, respectively.

+PVD/DM and PVD refer to peripheral vascular disease with and without associated diabetes mellitus, respectively.

that after walking a short distance (with a prosthetic leg) he still suffers from the same pain referred to his phantom calf. Of these 21 subjects 10 also had had other pains at, or near, the time of amputation; the remaining 11 suffered only the pain of intermittent claudication. If one includes the pain of intermittent claudication as a pre-amputation pain, then 50% (i.e., 26 of 52) of patients reported phantom limb pain which resembled, in quality and location, a pain experienced before the amputation.

Three subjects reported having had SMps that had since disappeared. One of these three also reported having experienced the pain of intermittent claudication referred to his phantom calf periodically for two months after amputation, but in this case the onset was unrelated to physical exertion.

Sixteen subjects reported having had pain at, or near, the time of the amputation, but had never experienced these pains in the phantom limb. In addition, 4 subjects who reported SMps for some pre-amputation pains indicated that other pains which they had had at the time of amputation had never been represented in the phantom limb. Together these 20 subjects reported having had a total of 25 pre-amputation pains.

Qualities and locations of somatosensory memories

Figure 19 shows the distribution of the 55 SMs according to the type or quality of pre-amputation experience. With the exception of the five reports of non-painful "super-added" sensations and one case of painless paresthesias, the SMs are predominantly replicas of distressing pre-amputation lesions and pains. These have been grouped into 10 categories and are displayed, in Figure 20, along with the distribution of pre-amputation pains that were not experienced as SMs. Figure 21 shows the distribution of SMs displayed according to the region or location of the limb in which the pains were reported.

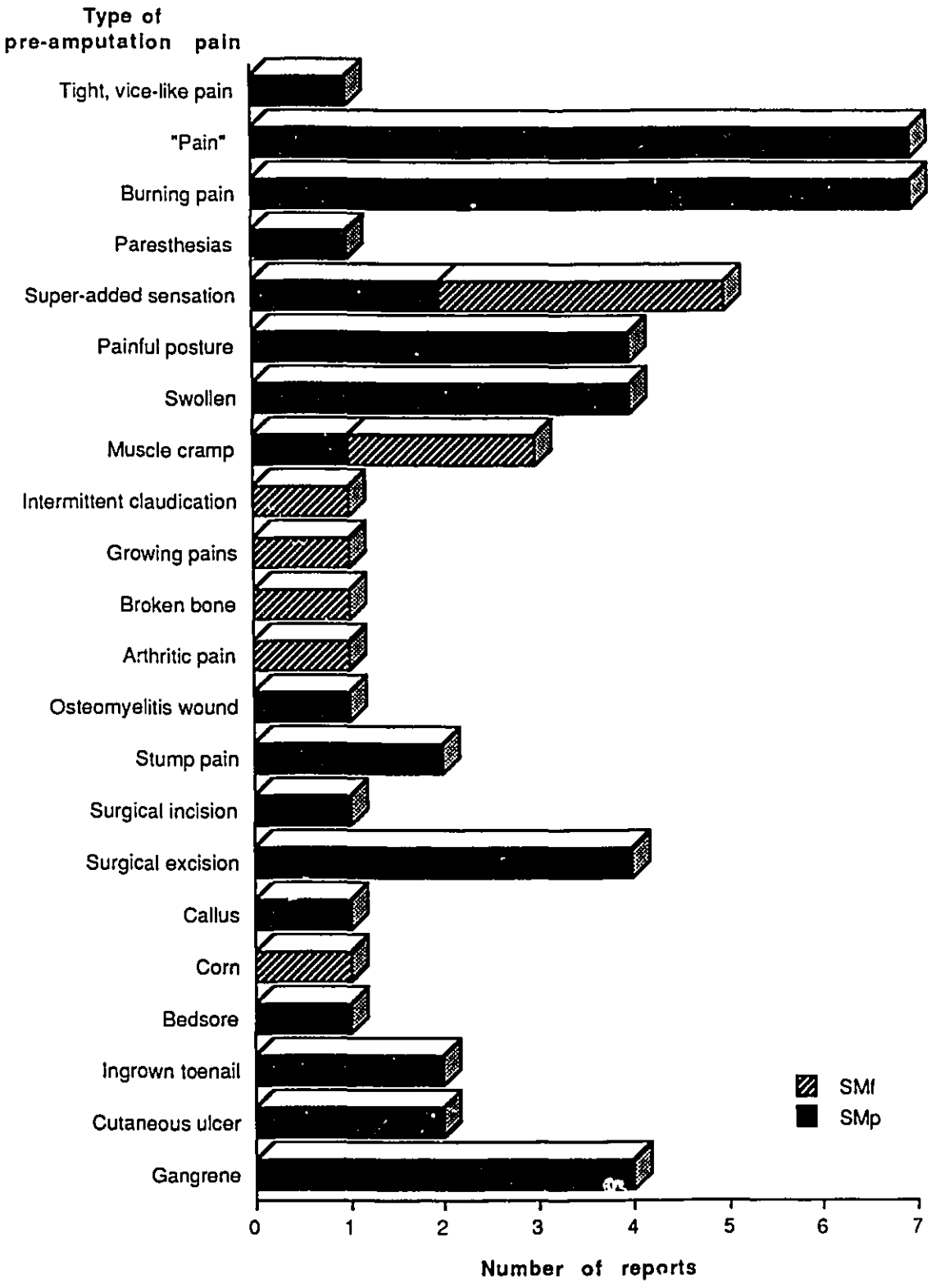


Figure 19. Histogram showing the number of reported somatosensory memories of various types of pre-amputation pain referred to the phantom limb after amputation. SMp refers to somatosensory memories of prior pains that were present immediately prior to amputation.SMf refers to somatosensory memories of former pains which were separated from the amputation by a pain-free interval.

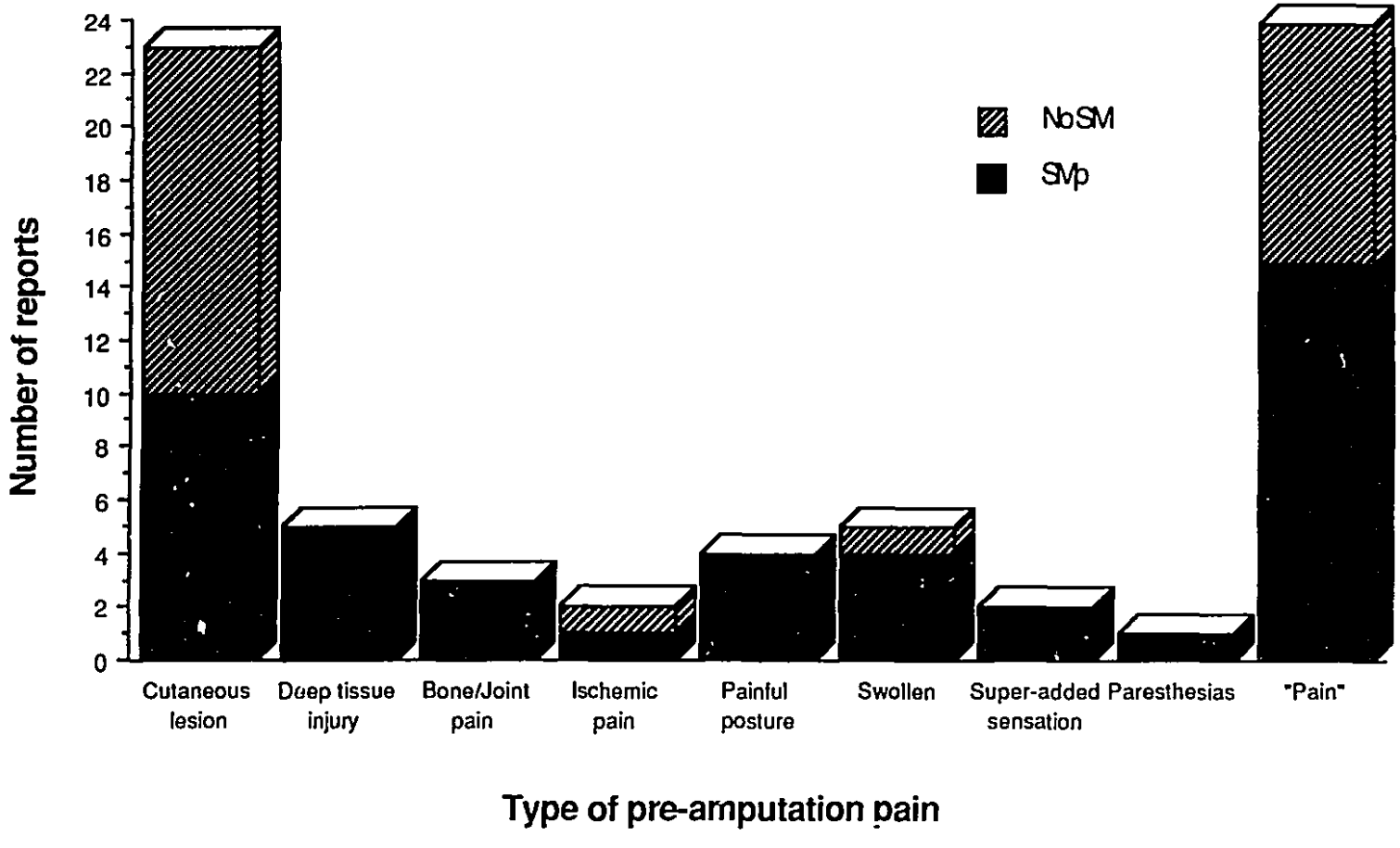


Figure 20. Histogram showing the type and number of pains that were reported to have been present at the time of amputation and were subsequently experienced in the phantom limb as somatosensory memories (SMp). Also shown are the types and numbers of pre-amputation pains that were reported to have present at the time of amputation but were not experienced as somatosensory memories (No SM).

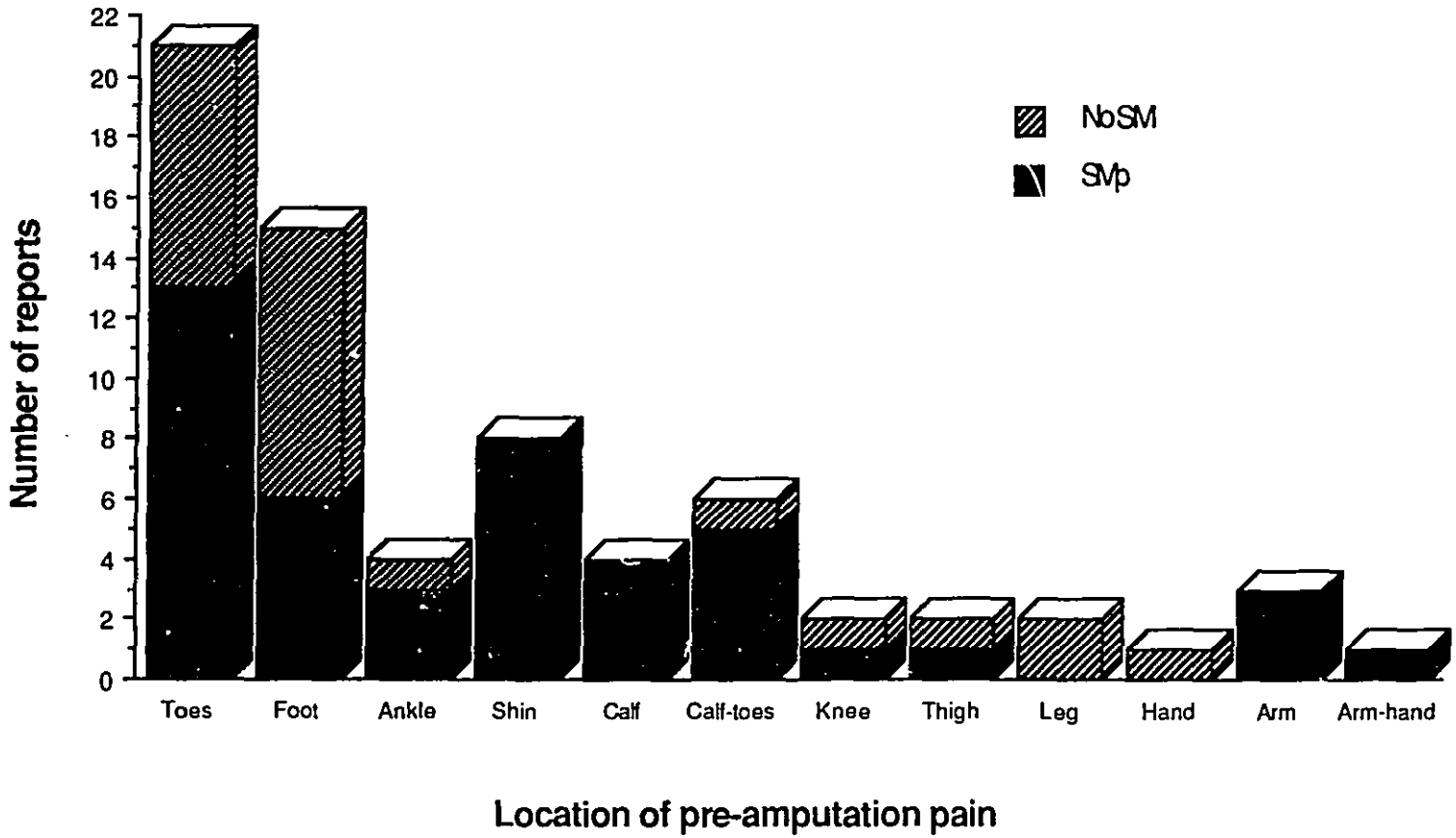


Figure 21. Histogram showing the region of the limb that was reported to have been painful at the time of amputation, and the number of pains that were subsequently experienced in the same region of the phantom limb as somatosensory memories (SMp). Also shown is the number and region of pre-amputation pains that were not experienced as somatosensory memories (No SM).

Intensity ratings of somatosensory memories

Nineteen of 48 SMs were rated as just as intense as the original pre-amputation pain; the remaining 29 were reported as less intense. An examination of the distribution of the type of pre-amputation pain according to the intensity of the SM revealed that, in general, pre-amputation pains which are frequently extremely painful (e.g., gangrene, stump pain, surgical excisions) tended to be rated less intense when they occurred as SMs. On the other hand, the less painful pre-amputation pains (e.g., corn, callus, broken toe) tended to retain their level of intensity when they were experienced in the phantom limb.

Comparison of subjects with and without somatosensory memories

The sample was divided into three groups based on their status with respect to phantom limb pain. Group PLP/SM consisted of 25 subjects who reported PLP similar in quality and location to their pre-amputation pain (n = 23 with SMps and n = 2 with SMfs). Group PLP/No-SM consisted of 19 subjects who suffered from phantom limb pain but did not have pain prior to amputation (n = 13), or if they did (n = 6), it bore no resemblance to the subsequent phantom limb pain. Group No-PLP consisted of 17 subjects who did not suffer from phantom limb pain. Four subjects who reported non-painful SMs were excluded as were the three subjects who reported having had SMps that had since disappeared.

Table 21 contains demographic and clinical characteristics of the three groups. They did not differ significantly in mean age (F (2, 58) = 1.33, $p > .05$) or time since the amputation (F (2, 58) = 0.42, $p > .05$). Furthermore, there were no significant differences with respect to gender (χ^2 (2) = 0.60, $p > .05$), prosthetic use (χ^2 (2) = 3.41, $p > .05$), level of amputation (χ^2 (2) = 1.61, $p > .05$), number of limbs amputated (χ^2 (2) = 0.13, $p > .05$), or whether the amputation followed an accident or illness (χ^2 (2) = 0.44, $p > .05$).

Table 21. A comparison of the mean age, time since amputation and other clinical characteristics of the three groups of amputees. Standard deviations are provided in parentheses.

	Group		
Clinical characteristics	PLP/SM (n = 25)	PLP/No-SM (n = 19)	No-PLP (n = 17)
Age at interview	56.4 (14.5)	62.7 (14.0)	57.9 (8.6)
Years since amputation	4.6 (5.3)	4.8 (10.3)	4.1 (5.2)
Number of males	15 (60%)	13 (68%)	12 (71%)
Uses prosthesis regularly (n)	12 (48%)	11 (58%)	13 (76%)
Amputation:			
- above major joint (n)	18 (72%)	12 (63%)	9 (53%)
- due to illness (n)	20 (80%)	14 (74%)	14 (82%)
- unilateral (n)	20 (80%)	16 (84%)	14 (82%)

Table 22 shows the mean scores on the personality, depression, and anxiety inventories for the three groups. A series of univariate one-way ANOVAs comparing the three groups did not reveal any significant differences on the EPI-E, EPI-N, or EPI-L subscales (F (2, 57) = 2.25, $p > .05$; F (2, 57) = 0.29, $p > .05$; F (2, 57) = 1.11, $p > .05$, respectively) the WRQ (F (2, 57) = 1.92, $p > .05$), BDI (F (2, 57) = 0.42, $p > .05$), or STAI-T (F (2, 57) = 0.19, $p > .05$)

The mean duration of pre-amputation pains that were later re-experienced in the phantom limb as SMps was 2.8 years (sd = 8.7 years) as compared to 1.1 years (sd = 3.0 years) for those pre-amputation pains that were not. The extreme negative skewness in the distribution of the duration of pains which developed into SMps was, in large part, due to one subject who reported two SMps that had had pre-amputation durations of 42 and 35 years. A log transformation was performed on these data and an analysis of variance was carried out on the transformed scores. There was no significant difference between the mean duration of these two categories of pre-amputation pains (F (1, 60) = 0.86, $p > .05$).

"Super-added" sensations of non-painful pre-amputation experiences

Five subjects described phantom limb sensations that resembled non-painful sensations experienced in the intact limb before it was amputated. Two reported the experience of a shoe-clad phantom foot. A third subject reported feeling the sensation like that of the bandages which once wrapped his wound. The fourth subject was a 23 year-old young man who had lost his right leg above the knee as the result of a motorcycle accident. He was interviewed approximately two months after the amputation. One sensation which he originally experienced at the accident scene occurred when the paramedics arrived and were required to cut off his right boot in order to release his foot. He felt a sensation of a considerable release of constrictive pressure as his foot was freed from the boot. This sensation has recurred several times since the amputation.

Table 22. A comparison of the mean scores on the personality, depression, and anxiety inventories for the three groups of amputees. Standard deviations are provided in parentheses.

	Group		
Variable	PLP/SM (n = 24*)	PLP/No-SM (n = 19)	No-PLP (n = 17)
EPI-Extraversion	13.2 (4.0)	11.2 (3.6)	11.1 (4.0)
EPI-Neuroticism	9.9 (5.9)	10.8 (6.2)	9.3 (6.5)
EPI-Lie	3.8 (1.6)	4.1 (1.8)	4.7 (2.7)
WRQ-Rigidity	26.0 (5.4)	29.1 (7.6)	29.4 (5.7)
BDI-Depression	9.8 (10.2)	11.2 (7.7)	8.5 (7.1)
STAI-T-Anxiety	35.8 (12.6)	36.9 (10.4)	38.1 (12.0)

*n=24 instead of 25 due to one subject who could not read well enough to fill out the questionnaires.

Another subject, described in detail in Study 1 (pp. 37-38), was re-interviewed six years later, for Study 4. She described a "draining" in her phantom hand and arm that resembled a pre-amputation sensation she had experienced on numerous occasions when the ulcers on her arm had been drained. She reported the phantom sensation as it occurred during the interview:

> "Now my hand seems to be starting to drain ... It's like draining it from edema. It's as though it were trickling out, and it's a little cooler ... It reminds me of how my hand and arm felt when I was in that machine at the hospital having the edema pumped out ... It's as though my arm were up and the fluid was leaving it ... there's the draining feeling leaving the fingertips, going down the fingers into the palm of the hand, through the palm into this part [the heel of the hand], then it just doesn't happen. ... The wrist is there, but it isn't part of the draining."

Multi-modal somatosensory memories

In five cases the SMps were described as more than just somatosensory in nature but were perceptually complex experiences that included descriptions of associated visual, tactile, and motor components that had accompanied the original experience. The painful somatosensory memories frequently were accompanied by high levels of anxiety and stress.

One woman, reported above, had a visual image of her phantom foot "wearing a white sock and a black patent leather shoe". Another subject continued to reach down to cup the wound which he felt on his phantom shin whenever the pain came on, just as he had done for years prior to the amputation. The third subject who had developed gangrene in his big toe prior to the amputation reported that when he feels the same burning in his phantom toes he also has a mental image of the big toe, discoloured and black, as it was prior to the amputation.

The fourth subject had undergone a right below-knee amputation for PVD. She reported having had an extremely painful ulcer in her big toe which was frequently cleaned

and treated by her physician in an effort to avoid amputation. During the interview she gave the following description of the pain in her phantom toe: "It was the worst pain of my life. I'm feeling the hole is there and it's paining me! It feels like he [the doctor] is cleaning out the hole and packing in medication and cotton."

The fifth subject was seen 15 months after a left above-knee amputation. He had spent 18 months in hospital following a motorcycle accident in which his left leg was severely damaged. He had had continuous pain in his leg ever since the day of the accident. He reported that during his hospital stay he had undergone numerous surgical operations on his leg, including one emergency procedure performed without an anesthetic. He described this procedure and his reaction as follows: "When he [the surgeon] opened it up, it burned so much, like taking a match and letting it burn ... and I fainted away." He had brought to the interview photographs of his leg, taken at the hospital, showing the wound (7" long and 2" wide) on his shin that he continued to feel in the phantom limb. He stated that he is frequently awakened at night feeling weak, covered in sweat and in a state of panic. He feels the wound and the same excruciating pain in the phantom limb as he had suffered before the amputation, and added that at those moments, it feels as if "the doctors and nurses are working on it."

Eliciting stimuli

With few exceptions, these subjects were unable to specify the eliciting stimuli or conditions that gave rise to the perception of the SMs. Five subjects noticed a relationship between the onset of the SM and certain behaviours or environmental events. Two of them reported that changes in the weather would still bring on the same pain they used to feel prior to the amputation when their limbs were intact. One felt an increase in the tight, vice-like pain around her phantom ankle. The other claimed that she was still capable of predicting when the weather was about to change by the onset of an arthritic ache in her phantom knee.

The third, with a right below-knee amputation, discovered that if he lay on his back and maximally flexed his right knee, he could briefly elicit the sensation of "the dry, callused,

tight skin" that he used to feel on the sole of his foot. If he continued to flex and extend his knee for a minute or so, he found that the sensation persisted continuously until he stopped.

A fourth subject reported that for about one year following the amputation he felt the same pain as well as the "hole" from a gangrenous pre-amputation ulcer on the medial aspect of his foot that had been "bigger than a silver dollar". At the time of the interview he could reproduce the SM at will by concentrating on his phantom limb, but unless he did so the SM remained out of his awareness. The fifth subject continued to experience the pain of intermittent claudication in his phantom calf after walking a short distance.

Case descriptions

Case 108

Female: born 1925 *Bilateral above-knee amputation*

This 62 year old retired nurse had undergone a left above-knee amputation in 1981, and two years later, a right above-knee amputation as a result of peripheral vascular disease and associated diabetes mellitus. She is an extremely insightful and thoughtful woman who, despite her physical limitations, maintains an active and independent lifestyle.

At the time of the interview she reported having experienced four phantom pains that resembled pains she had felt in her limbs at some time before the amputations. She reported having had pre-amputation pain of approximately 2 years duration in both limbs which became progressively more intense with time. She had had pain in the lateral "three toes on the left that went black with gangrene" which persisted after the amputation. She continues to suffer weekly bouts of this phantom limb pain which last for several hours. The pre-amputation pain on the right started in the toes and spread to the back of the leg. The pain she feels in the right phantom is the same, although the original pre-amputation pain was more intense.

This patient also reported two SMfs. She recounted that about 10 years before the amputation of her left leg, she had twice broken her left big toe. Since the amputation she has experienced the same throbbing pain in her left phantom big toe. She has never felt a

throbbing pain in her right phantom big toe. The second SMf was an arthritic ache in her left phantom knee which has been described above.

Case 119

Female: born 1931 *Left above-knee amputation*

This subject was a 56-year-old woman with PVD who was interviewed 2 years, 9 months after her left leg was amputated above-knee subsequent to the development of gangrene in the toes. Immediately after the amputation and intermittently over the following 18 months she describes having felt the same burning pain in her phantom toes, especially the big toe, "like if you held a match under it". At the time of interview, she reported having last felt this pain approximately one year and a half earlier.

Case 132

Male: born 1925 *Right above-knee amputation*

This 62-year-old retired businessman with peripheral vascular disease underwent a right below-knee amputation followed 48 days later by a second amputation at a higher level (below the knee), and 37 days after that, a third amputation above-knee. Following each of the three amputations, he suffered intense stump pain located at the site of the incision. He reported that prior to the initial amputation he had suffered two types of pain. The first was a "shooting, burning pain like a hot iron was driven into the toes." The second was a burning pain under the heel and ball of the foot. Both pre-amputation pains were felt immediately after the initial amputation and have persisted ever since. At the time of the interview, 3 months after the first amputation, he reported feeling these pains at least three times a day for variable periods of time, although the intensity was considerably reduced.

In addition to these pains, he reported that at least once a day he feels the pain of the "first cut" which he described as the same pain he had felt in his stump after the first amputation. This pain can last up to several hours and feels as the limb did after the first amputation with pain primarily in the region of the phantom stump and incision. At these

times, the phantom limb is reported to be the same length as it had been after the first amputation and lacks all the parts below the level of the phantom stump. Similarly, he frequently feels the pain from the "second cut" which is identical to the stump pain he had following the second below-knee amputation. Although he usually experiences these three pains separately, he reports that he has felt the pain of the "first cut," the "second cut," and the original pre-amputation pains simultaneously. At the time of the interview he reported that his phantom foot felt like it was resting "comfortably in a boot or a shoe". This experience was more than just the somatosensory qualities of slight pressure at certain points on the foot. So long as he did not look at the empty space below his stump, he was certain that his foot was clad in some sort of footwear.

Case 134

Male: born 1925 *Left below-elbow amputation*

This 63-year-old man sustained a combat injury as a young soldier serving overseas in the second world war. He was shot in the left shoulder region, thrown from the motorcycle he had been driving, and set off a land mine when he hit the ground. He sustained multiple injuries to the left side of his body including a brachial plexus avulsion. Thirty days later his left arm was amputated below the elbow. His memory for the events between the accident and amputation is poor. He reported that, as soon after the amputation as he can remember, his phantom hand was "sore and tightly clenched as if still holding the handlebar" of the motorcycle. He experienced the sensation "that blood was dripping from the phantom hand."

He reports having three other types of pain: a constant burning pain in the stump and hand; a pain slightly more intense, that can last for days; and successive "jabs" of pain referred to the stump that can each last up to 30 seconds and continue for several hours. Over the years he has had brachial plexus blocks, TENS, intensive physiotherapy, and numerous medications. Approximately 10 years ago he sought the help of a psychiatrist with whom he worked for about one year. The painful posture and dripping sensation "started to ease off" at

about that time although he is not certain whether to attribute the change to the psychotherapeutic intervention. At the time of the interview he reported, "Now the pain is like a little ball of fire in the stump."

Discussion

Pain experienced in a limb at the time of, or shortly before, amputation frequently persists in the form of a somatosensory memory referred to the phantom limb. Sixty-one percent of subjects who had pain at, or near, the time of amputation reported that the phantom limb pain they experienced resembled the pre-amputation pain in quality and location. This figure compares well with the results of Study 1 in which 6 of the 8 amputees reported somatosensory memories. It is also consistent with the study by Appenzeller and Bicknell (1969) who found that phantom limb pain was similar to the pain experienced before amputation in 79% of patients. It is higher than that reported by Parkes (1976) who found that about 50% of his patients who had moderate or severe phantom limb pain likened it to the pain which they had experienced before amputation. It is also higher than that in Browder and Gallagher's (1948) study whose numbers indicate that 12 of 26 patients (46%) with severe pain before the extremity was removed indicated that the subsequent phantom limb pain bore a distinct resemblance to the pre-operative pain. However, in both the Parkes and Browder and Gallagher studies the percentage reporting SMs was based on patients who had fairly intense pre-amputation or phantom pains. In Study 4, *all* instances of phantom limb pain that were reported to resemble the pain before amputation were recorded--regardless of intensity. Thus, the inclusion of pre-amputation pains which had been considered merely annoyances or minor discomforts (e.g., corn, callus, ingrown toenail), might explain the higher prevalence of somatosensory memories reported in Study 4 as compared to Parkes (1973), and Browder and Gallagher (1948).

There are four studies in which the rate of occurrence of SMs is considerably below the 61% found in Study 4 (Jensen et al., 1985; Roth & Sugarbaker, 1980; Sherman et al.,

1979; Wall et al., 1985). Roth and Sugarbaker (1980) and Sherman et al. (1979) provide few details about the type of pre-amputation pain experienced, making comparison of these studies difficult.

Wall et al. (1985) found that only 12.5% of their patients reported the location of their pain to be identical before and after amputation. The main difference between the study conducted by Wall et al. and most others (including Study 4) lies in the reason for amputation. Wall et al. report that all patients had undergone amputation for neo-plastic disease whereas occlusive vascular disease and trauma account for the majority of amputations in most other studies. In Study 4 , there were too few subjects in most diagnostic categories to carry out a detailed statistical analysis of the relationship between cause of amputation and the development of SMs.

The prospective design used by Jensen et al. (1985) gives their results more weight than those of Study 4. Jensen et al. found that pre-amputation pain and phantom limb pain were similar in both location and quality in 36% of patients eight days after amputation, and had decreased to 10% at the 6-month and 2-year follow-ups. However, Jensen et al. give few details concerning (a) their procedure of pain assessment before and after amputation, and (b) the types of pre-amputation pains experienced. Thus, it is difficult to determine the reason(s) for the discrepancy between their results and those of Study 4. Nevertheless, a number of suggestions are offered below.

First, Jensen et al. (1985) report that 35% of their sample suffered from "intermittent daily pain" prior to amputation. Since the vast majority of their subjects were amputated due to occlusive vascular disease it is likely that the "intermittent daily pain" they suffered was the pain of intermittent claudication. The presence of intermittent claudication prior to the amputation was not included as a pre-amputation pain in Study 4 although these data were collected. The pain of intermittent claudication occurs extremely infrequently as a somatosensory memory after amputation. In fact, of the 21 subjects in Study 4 who suffered from intermittent claudication prior to the amputation, only two reported its recurrence as a

phantom pain. It is reasonable to assume that the inclusion in the Jensen et al. study, and the exclusion in Study 4, of a pain which occurs very frequently prior to amputation but rarely as a phantom pain, may in part have led to different estimates of the percentage of amputees reporting similar pains before and after amputation.

Second, Jensen et al. (1985) indicate that 36% of patients with pre-amputation pain reported pain similar in quality and location to the phantom limb pain experienced 8 days after amputation. However, these authors also state that "the day before operation, 17 patients (29%) had no pain, while 41 patients (71%) still had some pain" (p. 270). Thus, the definition of "pre-amputation pain" used in the study by Jensen et al. encompassed a wider time frame than that of Study 4 which was limited to the period within (approximately) a day of amputation. If one calculates the percentage of patients in the Jensen study who reported similar pains before and after amputation based only on patients who were in pain the day before the amputation, then the figure increases to 51% (21 out of 41 patients).

Finally, many patients who undergo amputation have several types of pre-amputation pain (e.g., pain arising from corns, cutaneous ulcers, surgical bypass incisions, gangrene, diabetes mellitus, intermittent claudication). An accurate assessment of each pain is difficult to obtain, but unless they are all described and rated prior to amputation, it is possible that an SM which develops is not one of those previously rated. Furthermore, given that SMs tend to be transient and of relatively short duration, it is likely that some patients will not be experiencing that particular pain at the time of interview. These considerations may, in part, help to explain the discrepancy between the percentage of subjects with similar pains before and after amputation reported in the Jensen et al. (1985) study and in Study 4.

Considerations of a retrospective study

The results from Study 4 indicate that the most patients who report having had pain prior to the amputation continue to feel the same pain referred to the phantom limb after amputation. However, the similarity between pains was assessed in a retrospective manner,

based on a the patient's comparison of the phantom limb pain with his or her memory of the pre-amputation pain. The lack of an objective pre-amputation description of the pain, to which the subsequent phantom limb pain could be compared, raises the valid criticism that the patients' cognitive memory of pain may not accurately reflect the original pain experience. Due to the distorting effects of memory and the passage of time, the remembered pain might not have the same properties as the original pain. This is a problem common to all retrospective studies and one which can only be indirectly assessed.

Nevertheless, a number of studies have addressed the issue of the accuracy and reliability with which a variety of past pains are remembered (Eich, Reeves, Jaeger, & Graff-Radford, 1985; Hunter et al., 1979; Kent, 1985; Kwilosz, Gracely, & Torgerson, 1984; Linton & Götestam 1983; Linton & Melin, 1982, Roche & Gijsbers, 1986). On the whole, the results indicate that patients are surprisingly good at remembering past pains, and that when distortions occur, they involve mainly the *intensity* of the remembered pains (Eich et al.1985; Linton & Götestam, 1983; Linton & Melin, 1982). Severe pains tend to be overestimated and remembered as worse; mild or moderate pains are underestimated (Kwilosz et al, 1984; Roche & Gijsbers, 1986). Memory for the qualitative sensory dimension of pain, assessed with verbal descriptors instead of numerical values or visual analogue scales, appears to be less vulnerable to distortion (Hunter et al, 1979; Linton & Götestam, 1983; Roche & Gijsbers, 1986).

The results of these studies on pain and memory suggest that the subjects Study 4 were not affected by the distorting effects of memory or time. This suggestion is supported by two lines of evidence. First, the focus was on the somatosensory aspect of the phantom limb pain, the very dimension which appears to be least affected in studies of pain and memory. Intensity was not used as to determine whether a phantom pain qualified as a somatosensory memory. Second, it must be reiterated that these somatosensory memories are not cognitive memories, but actual sensations experienced in the phantom limb. Thus, for patients who were experiencing SM pains at the time of the interview, it was not a matter of *recalling* how a

pain felt in its absence, but of *recognizing* one that persisted (i.e., identifying a current sensory impression as familiar). Even for patients who were not experiencing the SM pain at the time of the interview, their task involved remembering whether, subsequent to the amputation, they had recognized any phantom pain as having occurred before. It is well established that recognition produces more accurate results than recall in standard tests of memory (Glass & Holyoak, 1986).

As a safeguard against the potential distortions of memory, only reports of phantom limb pain which the patient judged to be of the same quality and referred to the same location as the pre-amputation pain were included as somatosensory memories. If there was any doubt about either of these criteria the patient's report was excluded. According to many of the patients, the pre-amputation pains had been so well-localized and of so distinct a quality that they had no difficulty identifying them as the same pain when they recurred in the phantom following amputation. This is clearly illustrated by the details contained in Table 20 and from the cases described above. For example, somatosensory memories of corns, ulcerated skin, ingrown toenails were re-experienced *exactly* as they had been prior to the amputation. It was not a matter of having to separately assess their location and quality, and then decide whether the pain was familiar. The sensation of these phantom pains, in most cases, seemed to automatically evoke responses and behaviours which had accompanied the pre-amputation pains. These phantom pains were *re*-experienced, complete with the sense of familiarity, the label, and emotional response (Nathan, 1962).

The vast majority of patients had noticed the similarity between the pre-amputation and phantom pains on their own. Very few became aware of a similarity, for the first time, as a result of the examiner's questions posed during the interview. However, for all patients, in order to qualify as a somatosensory memory, both the quality and intensity of the pre-amputation and phantom pains had to be the same.

Factors influencing the development and expression of somatosensory memories

The results of Study 4 suggest that there are two factors, other than the amputation proper, which stand out as important in the development of somatosensory memories. The first is the experience of a *painful*, aversive event frequently accompanied by high levels of stress and anxiety. The second concerns the timing of the painful event relative to the amputation. The role of these, and other, factors are discussed below.

Pre-amputation pain. Pain, or some associated aversive aspect of it, possibly stress, appears to be crucial for the development of somatosensory memories since all but a few were originally experienced as painful prior to the amputation. The importance of pain in establishing this type of phantom phenomenon was also noted by Nathan (1962). Reports of somatosensory memories of non-painful pre-amputation sensations and experiences were rare. Such "super-added" sensations included the sensation of bandages which once wrapped a wound and the perception that the phantom foot was wearing a shoe. The occasional case report can be found in which non-painful sensations subsequently recur as phantom phenomena following amputation (Danke, 1981; Friedmann, 1978; Haber, 1956, 1958; Henderson and Smyth, 1948; Sliosberg, 1948; Solonen, 1[illegible]2; Varma et al., 1972) or spinal anesthesia (Wallgren, 1954), but these are exceptions. In general, the development and expression of somatosensory memories are intimately tied to the experience of pain. But the aspect(s) of pain which are crucial to this relationship remain a mystery.

Temporal relation between pain and amputation. When pain is experienced in a limb at, or near, the time of amputation there is a high probability that it will persist into the phantom limb and continue to cause the patient distress and suffering. In contrast, pains and painful lesions which were experienced some time before the amputation but had healed, and were no longer painful when the limb was removed, were less likely to recur in the phantom. When there is a discontinuity or a pain-free interval between the experience of pain and amputation,

the likelihood of that pain becoming incorporated into the phantom limb is diminished. Since every pain a subject remembered having had was not recorded, this conclusion may seem unwarranted. However, the fact that only seven of the 55 reports of SMs were of former pains makes it unlikely that these seven were the only other pains suffered over the lifetime of the subjects. Furthermore, over the course of the interview, many patients remarked that they had sustained injuries, undergone surgeries, and suffered from other pains as well, yet these had never been represented in the phantom limb. This conclusion is well-supported by examining the ratio of SMps to SMfs reported in the literature (Table 1). With the exception of a very few reports (i.e., Bors, 1951; Danke, 1981; Henderson & Smyth, 1948; Leriche, 1947a, 1947b; Nathan, 1962) the vast majority of reports and studies consist of pains which were present when the limb was removed.

Another explanation can be offered to explain why SMps far outnumbered SMfs. Pain which is discontinuous with amputation may not be remembered as well as pain which is present at the time of amputation. Thus, the relative rarity of SMfs may reflect forgetting (or inaccessibility) of the cognitive component of former pre-amputation pains. According to this hypothesis, the temporal proximity of the original pain to the amputation is important only in that it differentially favours the subsequent retrieval process for the cognitive memory of recent pains over past ones with little or no change in the strength or accessibility of the somatosensory memory component. After amputation, subjects may demonstrate poor (or no) cognitive memory for former pains and better memory for recent ones even though the somatosensory memory is active in both instances. Activation of the same (or a very similar) constellation of neural elements that represents the somatosensory qualities of a pre-amputation pain would not be recognized as having occurred before if it corresponded to a former pain that had long since healed, and would, if it corresponded to a pain that was present when the limb was amputated. In the latter case subjects would remember the sensation and report that their pain was the same before and after amputation. In the former

case the declarative information tied to the sensation would be forgotten and subjects would not report a resemblance even though one existed.

Duration of pre-amputation pain. The reported length of time a patient's limb was painful before the amputation does not appear to be related to the persistence or recurrence of that pain following amputation. The duration of pre-amputation pains which were later experienced in the phantom limb was not significantly different from that of pains which were not. This is not to say that there is not a critical period of time beyond which pre-amputation pains will be experienced as somatosensory memories and within which they will not: some pre-amputation pains may be brief enough to lack the properties required to establish a permanent central representation. However, if this is so, then on the average, subjects with pre-amputation pain exceeded this limit. Since reported length of time in pain does not differentiate those who develop SMs from those who do not, clearly there are factors other than duration of pre-amputation pain which govern the development and expression of somatosensory memories. Although Jensen et al. (1985) found that the prevalence of phantom limb pain six months after amputation was significantly greater in amputees who had had long- as opposed to short-lasting pre-amputation, this relationship does not necessarily address the phenomenon of somatosensory memories since it relates to phantom limb pain in general. Since certain individuals suffer more pain than others it would not be surprising to find that this is true both before and after amputation.

Type and location of pre-amputation experience. Judging from the wide variety of pre-amputation pains experienced and their reported location in the limb, it is concluded that the factors that govern whether or not a pain will be re-experienced following amputation do not include its type and location. Because there were too few observations relative to the rather large number of categories of pains and body locations, it was not possible to carry out statistical tests of whether the frequency of pre-amputation pains that were later experienced in the phantom limb differed in type and location from those that were not. However, from the

data presented in Figures 21 and 22, it appears that any type of pain or location has the potential to be represented as a phantom pain following amputation including annoying ingrown toenails, painful diabetic and gangrenous foot ulcers, the throb of a broken toe, surgical wounds on the shin and thigh, calf cramps, and remarkably, stump pain from a previous amputation. There is an obvious bias involved in being able to recall, after amputation, the type and location of a pre-amputation pain if that pain continues to be an ongoing source of distress in the phantom limb. Because of this bias, subjects with SMs would be expected to report having had a greater number, and more varied types, of pre-amputation pain than subjects whose pains did not recur as phantom phenomena. Thus, the histogram in Figure 20, showing that every deep tissue injury, bone and joint pain, and painful posture that was reported to have been present prior to amputation was also experienced after, may reflect this bias in recall rather than a predisposition for these types of pains to be represented in phantom limb.

On the other hand, recent evidence provided by Wall indicates that increases in the excitability of spinal cord cells receiving input from transected peripheral nerves last many times longer following activation of nociceptors in muscle as opposed to cutaneous tissue (Wall, 1988; Wall & Woolf, 1984, 1986). The trend for all reports of pre-amputation pains involving deep tissue injuries to be subsequently reported as a phantom pain (Figure 20) could reflect a mechanism similar to that reported by Wall and Woolf. Deep tissue injuries may be more likely than cutaneous lesions to persist in the form of a somatosensory memory referred to the phantom limb following amputation.

Intensity of pre-amputation pain. Pre-amputation pain intensity was not recorded in Study 4 but it is clear from the descriptions of the subjects that somatosensory memories develop from pre-amputation pains that are merely "annoying" as well as "excruciating". Intensity, alone, does not determine whether a pain will persist or recur after amputation but from the data shown in Figure 20 it seems that there is a trend for severe pains (e.g.,

gangrene, burning pain, cutaneous ulcers, surgical wounds) to be represented with a greater frequency than mild pains (e.g., ingrown toenail, corn, callus, broken toe). Although Jensen et al. (1985) also did not record pre-amputation pain intensity, they reach a similar conclusion based on patient narcotic requirements: severe pre-amputation pain resulted in persistent phantom pain more frequently than did the less severe pains.

It is possible that the development of SMs depends on a mechanism whose threshold is sensitive to a combination of intensity and duration (e.g., a multiplicative function of the two parameters) so that short, but intense pains (e.g., gangrene) and long-lasting mild or innocuous sensations (e.g., a bandage or a wedding band) produce sufficient excitation to produce long-term central changes. This would explain why duration and intensity of pre-amputation pain alone each fail to differentiate patients who develop SMs from those who do not. A prospective study examining pre-amputation pain intensity and duration may shed some light on this issue.

Psychopathology and emotional disturbance. Subjects who reported that their pain was the same before and after amputation could not be differentiated on personality, depression, and anxiety inventories from those who did not have phantom limb pain or those who had PLP which bore no resemblance to their pre-amputation pain. Thus, at the time of the interview, there was no evidence to suggest that levels of psychopathology and emotional disturbance were different for subjects reporting phantom limb pain of any type as compared to pain-free subjects. However, it must be remembered that these data were obtained at the time of interview, an average of approximately 5 years after amputation, when most of the (acute) psychologically and emotionally traumatic events associated with amputation had past. It is possible that the psychological and emotional states of the individual at, or near, the time of amputation play a part in the development and expression of SMs.

Input from modalities other than somesthesis. In a number of cases the SMs consisted of highly complex perceptually-integrated phenomena including associated visual, tactual, and motor components which had accompanied the original experience. Many of the pre-amputation pains had had corresponding visual elements such as a discoloured and festering diabetic ulcer, or a raw red open surgical wound. Some even had associated olfactory cues including the foul smell of putrid diabetic ulcers and gangrene. Such multi-modal input reported by subjects in Study 4, and similar reports by others (Henderson and Smyth, 1948, James, 1887; Jacome, 1978; Parkes, 1976; Reny, 1889 cited in Souques & Poisot, 1905; Riese, 1928 cited in Price, 1976b; Souques & Poisot, 1905; Wallgren, 1954), suggest that the involvement of the visual and olfactory senses prior to the amputation is much like that of the somatosensory projection system in that separate modality-specific representations of the pre-amputation experience are formed at the time of injury or during painful episodes.

Occasionally a subject was exposed to a single, intensely painful and traumatic injury such as an accident or an emergency surgical procedure performed without anesthetic. For some, these brief, discrete episodes formed the basis of vivid cognitive memories of specific events very similar to "flashbulb memories" (Squire, 1987) which occur after extremely stressful events. Following amputation, the circumstances surrounding these traumatic injuries, accompanied by the same pains and vivid memories, were re-experienced together with high levels of anxiety. These experiences were more than somatosensory in nature and consisted of additional associated components from other modalities. For these few subjects, the nature and severity of the initial traumatic injury, the similarity of pain before and after amputation, and the subsequent disability and suffering suggest a stress-related, post-traumatic chronic pain syndrome precipitated by the initial trauma (Engel, 1959; Muse, 1985, 1986).

Time course of somatosensory memories

The time course of the SMs was not recorded in detail for all subjects in Study 4 but it appeared that for the majority who reported having had pain at the time of amputation, their first post-amputation impression was that the limb had not been removed since they continued to feel the same pain. That is, for this type of phantom limb pain, there does not appear to be a pain-free period after amputation in which the old pain is not experienced (Leriche, 1947c; Riddoch, 1941). When pre-amputation pain is present, it persists after the amputation with the same parameters as before. With the passage of time, these temporal characteristics change. Somatosensory memory pains tend to become intermittent and transient so that, for example, one that is constant immediately after amputation occurs progressively less frequently, for shorter durations, and with longer intervals between occurrences. This raises the question of what conditions or stimuli bring about the activation of a somatosensory memory that has been inactive for some time. With the exception of several patients who had noticed a relationship between the onset of a somatosensory memory and certain internal bodily or external environmental events, the patients were unable to specify the conditions which give rise to the perception of these pains.

A tentative model

Based on the results of Study 4 and similar reports in the literature it is possible to provide a tentative account of how a painful pre-amputation lesion becomes centrally represented and subsequently re-experienced in the phantom limb following amputation.

It is proposed that separate somatosensory and cognitive representations of painful events are formed and strengthened during single or multiple occurrences of the pre-amputation experience. The cognitive component consists of declarative information about the pain that has accrued over repeated painful episodes, and gives the pain its unique personal meaning and a sense of familiarity. The somatosensory component consists of a higher-order

functional unit that represents the particular spatial and temporal pattern of nerve impulses corresponding to the intensity, quality and body location of the pain or lesion. The development of the functional connections between elements that comprise this higher-order unit is hypothesized to depend on a mechanism which is sensitive to the product of the intensity and the temporal characteristics of the lesion. Thus, the somatosensory component associated with brief but intense events as well as mild pains of long duration are established when a critical threshold is exceeded. Once this higher-order unit has been formed, it can be activated even when only some of its elements are present in the sensory input (Bindra, 1976, 1978; Melzack, 1989a). It is proposed that similar functional units corresponding to the same event are simultaneously formed in other modalities (e.g., visual, tactual). Through reciprocal inter-modal connections developed during repeated instances of pain prior to the amputation, these modality-specific representations may come to activate the somatosensory memory component in the absence of input once the limb has been removed (Bindra, 1976, 1978; Melzack, 1989a).

The patient's use of language also enhances cross-modal integration by simultaneously accessing modality specific representations during a painful episode (Marks, 1978). Highly emotional states which frequently accompany intense pains may facilitate the formation of the somatosensory and cognitive memory components through the peripheral and central release of hormones and neurotransmitters.

Amputation creates a state of central hyper-excitability due to the loss of normal afferent input. This partially, or completely, releases the tonic inhibition governing the previously established somatosensory representation, or, allows a pre-existing pain to persist unchecked. Coupled with this release phenomenon, impulses arising from associated modality-specific representations of the pain via inter-modal connections and neo-temporal regions underlying the cognitive component of the memory converge and summate to facilitate activation of the somatosensory memory.

The possibility of reciprocal connections between the two memory representations of the pain suggests that psychological factors may contribute to the activation of the somatosensory component, but it unlikely that such factors contribute to its formation. The recurrence of a past pain referred to the phantom limb may be enhanced by a process of state-dependent recall (Singer & Salovey, 1988) based on a similarity of affect before and after amputation. This biases perception and memory in favour of pain-related material thus optimizing the conditions for the recurrence of a pre-amputation pain once the limb has been removed.

Finally, the affective or emotional tone that accompanies the experience of a somatosensory memory is not a re-activation of a stored representation, but is generated on a moment-by-moment basis, determined by the combined information present in the somatosensory and cognitive memory components. Thus affective response is modulated as a joint function of the intensity, quality and location (in the phantom limb) of the somatosensory component, the personal meaning of the pain, and other declarative information (Price, 1988). Together these events give rise to the unified experience of a familiar pre-amputation pain which is referred to the phantom limb.

Summary and conclusions

The results of Study 4, taken together, indicate that brief or prolonged intense somatosensory inputs can lead to long-term changes in the activity of central neural structures associated with the somatosensory projection system. These changes are experienced after amputation as a type of phantom limb pain characterized by the persistence or recurrence of a pre-amputation pain. This phenomenon is a frequent sequela of amputation, occurring in 61% of cases. The wide variety of pains reported to persist or recur following amputation suggests that any type of pain has the potential to be represented in the phantom limb although some are reported more frequently than others. For example, phantom limb pain characterized by intermittent claudication is rarely reported even though it occurs very frequently before

amputation in patients with peripheral vascular disease. There is the suggestion that patients with pre-amputation pain arising from neo-plastic disease are less likely to develop painful SMs than are accident victims and patients with occlusive vascular disease, but this possibility needs to be verified in future comparative studies.

A number of implications for treatment are suggested by the results of Study 4. First and foremost is the obvious strategy of maintaining all patients free from pain for as long as possible prior to the amputation so as to avoid the development of a permanent central representation of the somatosensory qualities of the pain. Second, if as hypothesized, a phenomenon similar to state-dependent recall occurs in which the similarity in intense affective state before and after amputation facilitates the re-activation of SMs, then attempts at reducing the affective dimension of the pre-amputation experience may interfere with both the development and subsequent experience of these pains. This can be accomplished with medication, psychotherapy and through adequate preparation of the patient for amputation. Similar interventions can be implemented during the acute and convalescent stages after amputation. Finally, use of combined general and spinal anesthesia during amputation would be expected to interfere with the formation of both the somatosensory and cognitive memory components of pain, thereby reducing the incidence of phantom limb pain arising from the amputation itself.

GENERAL DISCUSSION

The major findings and conclusions from the four studies are summarized below, followed by a general discussion of their implications for future research and clinical practice in the field of phantom limb phenomena.

Major Findings and Conclusions

1. The results of the four studies reported in this thesis demonstrate the extraordinary diversity of sensations experienced in the phantom limb. The data presented indicate that interacting peripheral and central factors are involved in the production of some of the qualities of experience, but others are wholly independent of the periphery and depend exclusively upon the activation of central structures.

2. Sympathetic nervous system involvement in phantom limb phenomena is suggested by two findings from Studies 2 and 3. The first is that paresthesias and dysesthesias referred to the phantom limb correlated significantly with stump skin conductance and skin temperature. The mechanism mediating this relationship may be a neural one in which certain phantom limb paresthesias result from changes in the rate of firing in primary afferents located in the stump due to sympathetic efferent activity.

The second finding implicates the sympathetic nervous system in cases of phantom limb pain. Study 2 showed that amputees with phantom limb pain had significantly lower skin temperature at the stump compared to the intact limb. The between-limb skin temperature difference was not significant for amputees with a non-painful phantom limb or those with no phantom limb at all. The mechanism mediating this effect may be a vascular one in which an abnormal pattern of activity in postganglionic cutaneous vasoconstrictor fibers in the stump sets the stage for subsequent phantom limb pain.

3. The combined results of Studies 2 and 3 demonstrated that transcutaneous electrical nerve stimulation (TENS) applied at the outer ears and contralateral limb decreased painful and non-painful paresthesias whereas a placebo control condition had no significant effect. A diffuse noxious inhibitory control (DNIC) mechanism (LeBars et al., 1979,1983) is proposed to mediate the reduction in paresthesias. The possibility also exists that descending controls act on peripheral sympathetic fibers to inhibit neurotransmitter release and thus reduce sympathetically-governed primary afferent discharge.

4. Study 4 showed that phantom limb pain characterized by a recurrence or persistence of the pain experienced prior to amputation is frequently reported to occur after amputation. These somatosensory memories provide convincing evidence of a purely central component to some phantom limb pains. The similarity in pain before and after amputation argues for adequate pre-operative anesthesia aimed at abolishing all traces of pain prior to the amputation.

5. The relationship between phantom limb pain and psychopathology was assessed in Studies 2 and 4. Amputees with or without phantom limb pain could not be differentiated by their scores on personality inventories and questionnaires measuring affect and mood. In particular, amputees with phantom limb pain did not differ from those with a painless phantom or no phantom limb at all in terms of "psychological rigidity". These results indicate, for the majority of amputees, that phantom limb pain is not related to psychopathology.

Referred sensations

The facility and accuracy with which any sensation is described depends on several inter-related factors. In the case of sensations referred to the phantom limb, it is important to consider the individual's proficiency with the language, level of general intelligence, willingness to share these experiences with the examiner, emotional significance of the amputation, and intensity of the phantom limb sensations and pain. Despite obvious between-

subject differences in these and other factors, the reports have much in common. First, the painful and non-painful phantom limb has an attention-riveting quality whether perceived for the first or hundredth time. The vividness and clarity of the sensations which define the phantom are striking. This point has been made by clinicians and scientists (e.g., Mitchell, 1866, 1871, 1872; Wall, 1980) ever since phantom limbs were first studied, but the running commentary offered by the subjects in Study 2, as they described the sensations they experienced captures the phenomenological experience and documents the remarkable variety of sensations which define the phantom limb.

Second, most phantom limb sensations are comprised of a mosaic of ever-changing sensations; some subtle, others striking in their clarity. Furthermore, it is not unusual for phantom limb pains to be reported as comprised of several types; for example, a squeezing, cold sensation, or a recurrent "stabbing" pain super-imposed on a background of burning pain. Few people report a static, unchanging phantom limb. These latter phantoms are usually described by individuals who are made anxious by the phenomenon and actively avoid attending to it. However, when attention is focused on the phantom, its fluidity and dynamic nature become apparent.

It became clear that many subjects had difficulty describing the sensations which were referred to the phantom despite the clarity of the sensations and pain they felt on a daily basis. Some treated the phantom as a foreign object to be avoided. Anxiety surrounding the strange sensations was also apparent. The possibility of anxiety-induced increases in the paresthetic or dysesthetic component of the phantom is highlighted by the results of Studies 2 and 3 which implicate the sympathetic nervous system as a trigger for certain phantom sensations.

In the course of obtaining detailed descriptions of these experiences, subjects were required to focus attention on their phantom and the interoceptive sensations which defined it. For some this served as a form of desensitization in which unfamiliar and frightening experiences became less foreign. These observations suggest that getting patients to attend to and describe the sensations they are experiencing may, in itself, prove helpful in the short and

long run. The short-term benefit of attending to and describing the phantom is a potential reduction in anxiety, decrease in sympathetic output and primary afferent discharge, and a reduction in the intensity of phantom limb paresthesias. The long-term benefit is that through descriptions of pains and other sensations our knowledge of the different qualities of phantom limb experience is enhanced. It is primarily through such descriptions that new ideas for treatment emerge.

Sympathetic nervous system involvement

Two hypotheses were presented to explain the role of the sympathetic nervous system in the development and maintenance of painful and non-painful phantom limbs. The results from Studies 2 and 3 provide evidence in support of both hypotheses.

The first hypothesis is that sympathetically-maintained pains are mediated by input from primary afferents located in stump neuromas and surrounding tissue (Roberts, 1986; Roberts & Fogelsong, 1988a, 1988b). Sympathetic efferent activity increases the rate of primary afferent discharge by the liberation of noradrenaline and acetylcholine from vasomotor and sudomotor fibers, respectively. The afferent impulses impinge on spinal cord cells subserving the phantom limb and give rise to the experience of paresthesias and dysesthesias. The results from Studies 2 and 3 showed that changes in the intensity of phantom limb paresthesias were significantly correlated with stump skin conductance and skin temperature. Other qualities of phantom limb experience were not correlated with these peripheral measures, suggesting that they may be independent of the sympathetic nervous system and generated by activity in spinal cord cells or more rostral structures.

The second hypothesis is that abnormal vascular and trophic changes which have been demonstrated after peripheral nerve injuries set the stage for chronic pain (Blumberg & Jänig, 1981, 1983, 1985). Discharge patterns of post-ganglionic cutaneous vasoconstrictor neurons located in the stump take on the characteristics of muscle vasoconstrictor neurons. They

exhibit a high degree of cardiac rhythmicity not present in intact cutaneous vasocontrictor fibers. This alteration in sympathetic output to skin fibers has been proposed to result in pain due to vascular and trophic changes which subsequently develop. Study 2 found evidence of abnormal vascular functioning at the stump in amputees with phantom limb pain. In these subjects, the stump was significantly lower in skin temperature than the intact limb. On the other hand, between-limb differences were not significant for amputees with a non-painful phantom limb or for those with no phantom limb at all. Blocking the sympathetic supply to the affected region may, at least temporarily, reduce the vasoconstriction and relieve the pain.

The efficacy of TENS

In the Introduction section of this thesis, it was noted that the failure rate of treatments for phantom limb pain is exceptionally high (Sherman, 1980). No one treatment is effective for a majority of patients. The results from Studies 2 and 3 suggest that part of the reason for the overall ineffectiveness of treatments is that the major locus of abnormal neural activity responsible for triggering the pain may be different in different patients. Thus, treatments aimed at modifying a putative peripheral mechanism would only be effective for certain patients. The results from Study 2 indicate that TENS applied at the outer ears was more effective than a placebo control in reducing the intensity of painful and non-painful paresthesias. Other qualities of phantom limb pain did not appear to be affected. This emphasizes the importance of deriving a classification of various types of phantom limb pain based on patients' descriptions of the quality of the pain and other parameters (e.g., Merskey, 1986). The results from Study 3 are suggestive and indicate that a larger-scale placebo-controlled group-study be undertaken to evaluate the efficacy of TENS applied at the contralateral limb in patients suffering from phantom limb pain. Whether this form of treatment would be effective for pains other than dysesthesias remains to be tested.

The practical importance of TENS applied at the outer ears should not be overlooked. Most patients with upper extremity amputation are dependent on others to help them

administer TENS in the traditional manner: only the most adept patients are able to apply the conductive gel and then position and affix the electrodes with only one hand. The practical advantage of TENS applied at the outer ear is that simple clip on earring electrodes are used, which can be easily attached to the earlobe by the patients themselves. The benefit of such a self-treatment program is that it would enable the patient to become less dependent on the health professional or family members from whom he or she would otherwise have to seek help.

Somatosensory memories

Sixty-one percent of subjects who reported having had pre-amputation pain indicated that they continued to experience the same pain referred to the phantom limb. The vast majority of somatosensory memories were of pains that were reported to have been present at the time of, or shortly before, amputation. Somatosensory memories of past pains that had occurred before the amputation were rare implying that if a pain-free interval preceded the amputation, the pain was less likely to recur. These results argue strongly for maintaining patients free from pain for as long as possible prior to amputation.

Furthermore, results from animal studies and clinical research involving amputees and other surgical candidates indicate that part of the pain which develops after peripheral nerve transection can be attributed to the injury barrage sustained when the nerve is cut (Bach et al., 1988; Bonica, 1953; Cousins, 1989; McQuay et al 1988; Wall, 1988). Animal studies indicate that the autotomy which develops after deafferentation can be significantly enhanced by an injury inflicted prior to deafferentation (Coderre et al., 1986a, 1986b; Dennis & Melzack, 1979) and that anesthetizing the primary afferents before they are cut leads to a significant reduction in the severity of autotomy (González-Darder et al., 1986).

These results have important implications for the development of phantom limb pain arising directly from the trauma produced by the surgical amputation of a limb (Bach et al., 1988; Bonica, 1953; Cousins, 1989; McQuay et al., 1988; Wall, 1988). Some component of

phantom limb pain undoubtedly represents the persistence of activity in hyperactive deafferented cells whose inputs were dramatically altered by the cutting of tissue, nerve, and bone during amputation. Amputation performed under only general anesthesia would not lead to the formation of a cognitive memory of the amputation, but the development of the somatosensory memory component would be unaffected. Thus, there would be no cognitive memory of having felt pain. The somatosensory quality of the phantom limb pain which subsequently develops obviously could not be recognized as having occurred before. This kind of dissociation between the cognitive and somatosensory memory components is supported by a number of studies which have shown that conscious awareness of pain is not necessary for the development of a somatosensory pain memory (Hutchins & Reynolds, 1947, Reynolds & Hutchins, 1948).

The effects of combined general anesthesia and spinal blockade for amputation should be more effective than general anesthesia alone in reducing the incidence of phantom limb pain. The combined anesthesia would be expected to interfere with the development of both the somatosensory and cognitive components by blocking the transmission of nociceptive impulses at the level of the spinal cord, and by ensuring that the patient is unconscious. On the other hand, general anesthesia alone would interfere with the formation of the cognitive memory component only. This suggestion is indirectly supported by studies using animals and humans undergoing amputation and other surgical procedures (Bach et al., 1988; González-Darder, 1986; McQuay et al., 1988).

Psychopathology and phantom limb pain

The results of Studies 2 and 4 indicate that amputees with phantom limb pain, painless phantom limb sensations and those with no phantoms at all could not be distinguished on the basis of their scores on the Eysenck Personality Inventory subscales (EPI-E, EPI-N, EPI-L), the Beck Depression Inventory, Spielberger State or Trait Anxiety Inventory, Wesley Rigidity

Questionnaire, or the Mood Rating Scale. These results demonstrate, at least for subjects in Studies 2 and 4, that phantom limb pain can exist in the absence of psychopathology.

More importantly, they should serve as a reminder to researchers and clinicians that not all patients with phantom limb pain show signs of concurrent psychopathology. Quite apart from the issue of whether or not phantom limb pain is caused by, or causes, psychopathology, conclusions such as those presented by Parkes (1973), and Parkes and Napier (1970), should be tempered by the lack of any significant differences among the groups of subjects in Studies 2 and 4. Parkes and Napier (1970) describe the "denier" or "defiant type" by the "obstinate refusal to admit defeat even against better advice, ... who never accepts that he has lost anything at all. He appears to have a compulsive need to do everything at least as well as he could before operation and if possible, better, as if to convince himself and everyone else that he is not incapacitated at all" (p. 442). Sufferers of phantom limb pain were described by Parkes (1973) as "rigid" and "compulsively self-reliant" individuals. Sherman, Gall, and Gormly (1979) also have encountered patients beset by unrelenting phantom pains who appear to be psychologically "rigid".

With the exception of a recent review paper by Sherman, Sherman, and Bruno (1987), the idea that patients with phantom limb pain have "rigid" and "compulsively self-reliant" personality characteristics has been uncritically accepted by researchers working in the field of phantom limb pain (e.g., Dawson & Arnold, 1981; Dernham, 1986; Lundberg & Guggenheim, 1986; Shukla et al., 1982a). Sherman et al. (1987) offer an interesting hypothesis to explain the prevailing view that has been spawned by the conclusions of Parkes' (1973) study. They suggest that the low success rate of most treatments for phantom limb pain serves as a deterrent to all but the most persistent or self-reliant individuals. These sufferers of phantom limb pain continue to search for relief despite repeated failures. Other, less assertive patients, continue to suffer as well, but they have given up actively seeking help. This self-selection bias would explain the tendency for individuals with "compulsively self-reliant" personality characteristics *and* phantom limb pain to dominate the clinical picture of the typical

patient with phantom limb pain. As appealing as this reasoning is, Parkes (1973) did not select patients seeking treatment for their pain problems. Furthermore, Parkes hypothesized that the "compulsive self-reliant" patient does *not* seek help.

Because there are serious questions regarding the validity and interpretation of Parkes' (1973) results, it is best to view them with caution. In particular, the use of an non-validated, single-item scale to measure "psychological rigidity" strongly suggests that Parkes' conclusions were premature -- especially in the light of the potential damage such negative attributions can have in a field beset with treatment failures. Furthermore, the results of Studies 2 and 4 failed to confirm Parkes' (1973) and Parkes and Napier's (1970) conclusion that amputees with phantom limb pain are more "psychologically rigid" than amputees not in pain.

One danger in attributing such negative traits to patients suffering from pain is that there is a tendency for researchers and clinicians who are less careful to over-generalize and ascribe these same traits to all patients with phantom limb pain (e.g., Dawson & Arnold, 1981; Dernham, 1986; Lundberg & Guggenheim, 1986; Shukla et al., 1982a). A second danger is that clinicians and researchers may begin to believe that these patients actually are "rigid" and "compulsively self-reliant" and forget that they merely endorsed items on a questionnaire which may have been correlated with an objective measure of rigidity. Worse yet, these patients may be damned from the start by their pain.

Some health professionals, much like some of the patients they try to help, tend to be egocentric. They adopt working definitions of terms such as "compulsively self-reliant" and "rigid" which are consistent with their role as helper. Patients who do not heed their advice, who try to rely on themselves, who attempt to make the best of a bad situation, who persist in trying to overcome the adversity they are faced with every day, all too often, may acquire these harmful labels because these patients do not behave in a way that conforms to the helper's expectations. Similar patients have been described as "oppositional individuals" (Ascher & Turner, 1980), "therapist killers" and "therapy addicts" (Weeks & L'Abate, 1982),

and "help-rejecting complainers" (Tennan, Rohrbaugh, Press, & White, 1981). They resist most direct attempts at change. They fight the therapist's moves to help (Katz, 1984). It is true that these individuals are exceedingly difficult to treat, but clinicians and researchers must be be mindful of their choice of words and intentions when attempting to "help." These terms have connotations which cast the behaviours and attitudes they describe in a negative light. They impede the already difficult processes of psychological and physical rehabilitation that confront the patient after the amputation of a limb. It is not too difficult to also see the positive traits possessed by these highly motivated and independent individuals.

Perhaps it would be more fruitful to investigate the phenomenon of *psychological reactance* (J.W. Brehm, 1966, 1972; S.S. Brehm, 1976; West & Wicklund, 1980) in amputees. Briefly, *reactance* is a psychological state which may be aroused when an existing freedom is threatened or lost. The extent of reactance experienced is determined by the interaction of three variables: the (a) strength of the threat, (b) number of freedoms threatened and, (c) importance of the freedom(s). The effects of reactance are behaviours aimed at restoring or re-asserting the threatened or lost freedom.

In the case of amputation, the loss of one (or two) limbs would be expected create a state of reactance since the freedoms lost and threatened are many, and usually exceedingly important to the individual. The theory predicts that one would expect to see amputees attempting to restore their lost freedoms by engaging in, or attempting to engage in, behaviours which they used to be free to do. The predictions made by reactance theory can account for behaviours which have traditionally been attributed to the "denier" or "defiant type" and interpreted as "rigid" and "compulsively self-reliant" (Parkes, 1973; Parkes & Napier, 1970; Sherman et al., 1979) but without the negative connotations. Depending upon the extent of reactance experienced, one would expect to find amputees to continue trying to accomplish what they used to do before the operation. The presence of phantom limb pain would be expected to further narrow the patient's repertoire of "free" behaviours, leading to a greater state of psychological reactance and more vigorous attempts to re-assert the lost or

threatened freedoms. Thus, one might expect individuals with phantom limb pain to show greater levels of psychological reactance than amputees not in pain. Effective approaches to helping are frequently counter-intuitive and involve empowering the client and avoiding therapist-client power struggles (Katz, 1984; Tennan et al., 1980; Weeks & L'Abate, 1982).

Concluding Comment

Research on the phenomenon of phantom limb pain has reached a turning point. Global descriptive statistics indicate that phantom limb pain is considerably more prevalent than the early figures suggested. It is clear that up to 80% of amputees suffer from one or more types of phantom limb pain. It is time to sub-divide and classify phantom limb pain into homogeneous groupings on the basis of their descriptive characteristics. Sherman has suggested that burning stump and phantom limb pain have a peripheral vascular component (Sherman, 1984; Sherman & Bruno, 1987). The results from the present thesis have shown that at least some of the paresthesias referred to the phantom limb have a peripheral basis, triggered by the effects of sympathetic efferent activity on primary afferents located in the stump. Other phantom pains are clearly independent of peripheral input but nonetheless may be circumvented by proper pre-operative anesthesia. The importance of obtaining a comprehensive description of the various phantom limb sensations cannot be overemphasized since it is on the basis of this information that potential mechanisms are identified and new ideas for treatment originate.

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APPENDICES

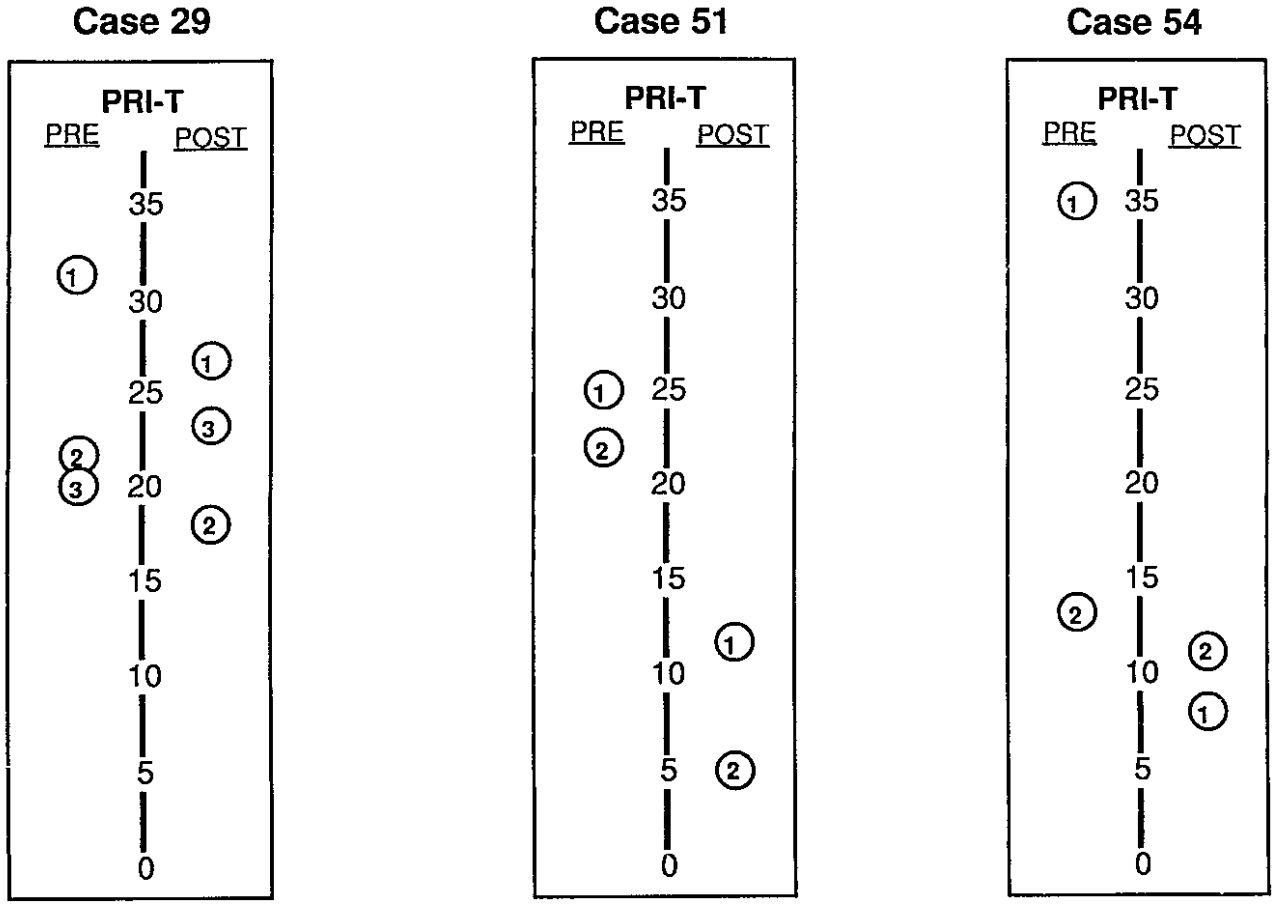


Fig. 1. Plots of McGill Pain Questionnaire scores for the total pain rating index (PRI-T) obtained before (PRE) and after (POST) TENS for Cases 29, 51, and 54. Numbers within circles correspond to session number.

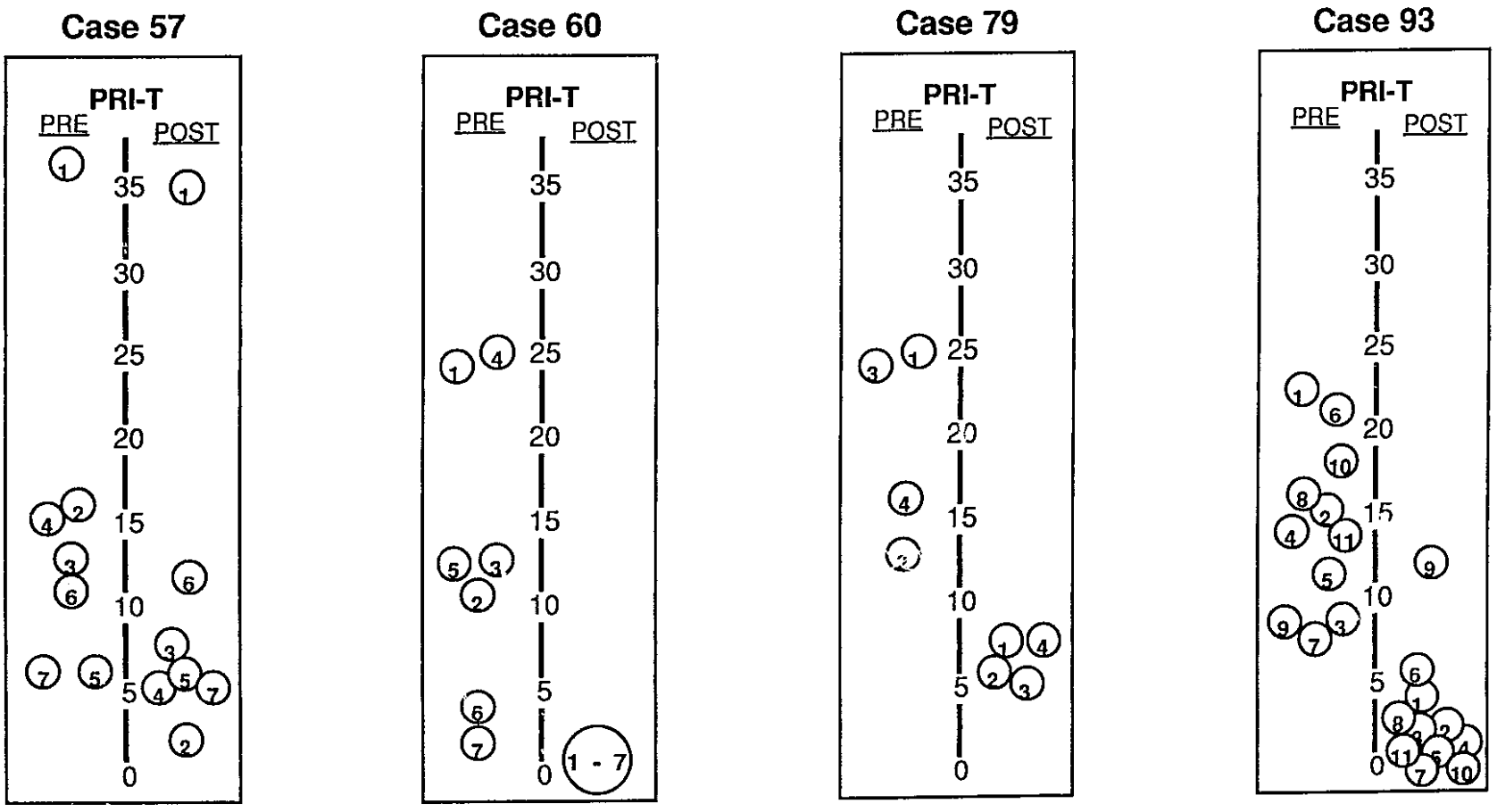


Fig. 2. Plots of McGill Pain Questionnaire scores for the total pain rating index (PRI-T) obtained before (PRE) and after (POST) TENS for Cases 57, 60, 79, and 93. Numbers within circles correspond to session number.

APPENDIX B-1

Consent Form

After having an arm or a leg amputated most people, at some time, feel that the cut off part is still there. This phenomenon has been called a **phantom limb**. Some individuals also report that the phantom limb can be very painful. The reason we are conducting this research project is to find out more about painful and non-painful phantom limbs so that more effective treatments can be found to help those who suffer from phantom limb pain.

If you decide to participate in the study, you will be seen on two occasions. Mr. Joel Katz will ask you some questions about your amputation, your phantom limb, and about any phantom pain you may have felt or still feel. He also has a number of questionnaires for you to fill out that have to do with your overall mood, how you generally feel, and about your attitudes and personality characteristics. We are particularly interested in hearing about any strategies or activities that you feel have been helpful to you in coping with your phantom limb pain so that we may pass this information along to others who suffer from phantom limb pain. This procedure takes about one hour.

We have found that when transcutaneous electrical nerve stimulation (TENS) is applied to points on the outer ears some individuals felt unusual sensations in the phantom limb. Others reported that their phantom limb pain was reduced after TENS. We are currently studying these effects. You may have noticed that on some days your stump is cold and clammy. At other times it may be drier and warmer. These kind of changes reflect the activity of the autonomic nervous system, and may be altered during TENS.

On both sessions we will measure the temperature and the amount of sweating at your stump and at your other limb while TENS applied to your outer ears. First, Mr. Katz will measure the sensitivity of the skin on your stump and your healthy limb. This is not a painful procedure. Then you will receive TENS applied to the outer ear for 10 minutes. At the same time we will measure the temperature and the amount of sweating at both limbs. We are also interested in any sensations that you may experience in your phantom limb, and we will provide you with a dial to turn to monitor them. Each of these sessions will last about one hour.

TENS is a non-invasive and well-recognized treatment for chronic pain problems. This form of stimulation produces a mild pulsing sensation on the ears that is not painful or dangerous. It is not known to produce any harmful effects, but is not recommended for patients who have a history of skin problems, pregnant women, or people with pacemakers. Please inform us if you fall into any of these categories.

If you decide to participate, the information that you provide will remain strictly confidential and will be used anonymously for purposes of research and publication. Only the researchers will have access to this information. Your participation would be greatly appreciated as the results will help us to improve the treatments for those who suffer from phantom limb pain.

I have read and understood the above information, and agree to participate in this research project as described above. I also understand that I am free to withdraw from the study at any time I choose.

_____ _____

Date Participant's signature

_____

Witness' signature

_____

Investigator's signature

APPENDIX B-1

Formulaire de Consentement

Suite à l'amputation d'un bras ou d'une jambe, la plupart des gens sentent, à un moment ou à un autre, que la partie amputée est toujours présente. Ce phénomène porte le nom de **membre fantôme**. Certaines personnes rapportent aussi que leur membre fantôme peut être très douloureux. Nous effectuons donc une étude sur les membres fantôme douloureux et non-douloureux afin de mieux comprendre ce phénomène et d'améliorer l'efficacité des méthodes de traitement pour ce type de douleur.

Si vous décider de participer à cette étude, nous devrons vous rencontrer à deux reprises. La première fois, M. Katz vous demandera de répondre à une série de questions concernant votre amputation, votre membre fantôme et toute douleur que vous avez pu ressentir ou que vous ressentez toujours dans votre membre fantôme. Il vous invitera également à compléter un certain nombre de questionnaires portant sur votre humeur, vos attitudes, vos caractéristiques de personnalité et comment vous vous sentez en général. Nous sommes particulièrement intéressés à savoir quelles stratégies ou activités vous utilisez pour vous aider à la douleur de membre fantôme afin que l'on puisse faire benificier d'autres personnes souffrant du même type de problème de l'information que vous nous fournirez. Cette première rencontre durera environ une heure.

Nous avons découvert que lorsque de la stimulation électrique percutanée des nerfs (SEP) est effectuée sur des points de l'oreille externe, certains individus ressentent des sensations inhabituelles dans leur membre fantôme. D'autres rapportent que la SEP réduit la douleur dans le membre fantôme. Nous effectuons présentement une étude sur ces phénomènes. Vous avez pu déjà noté que certains jours votre moignon est froid et moite; à d'autres moments, il peut être plus sec et chaud. Ces types de changements réflètent l'activité du système nerveux autonome et peuvent être affectés par la SEP.

Lors de la première et deuxième rencontre, nous mesurerons donc la température et le degré de transpiration de votre moignon et du membre correspondant lorsque de la SEP est effectuée sur des points sur votre oreilles externes. M. Katz prendra tout d'abord des mesures de sensibilité de la peau à votre moignon et au membre correspondant. Cette procédure ne vous causera aucune douleur. Par la suite, nous appliquerons de la SEP sur vos oreiles externes durant une période de 10 minutes. Au même moment, nous mesurerons la température et le degré de transpiration dans vos deux membres.

Nous nous intéressons également à tous types de sensations que vous pouvez avoir dans votre membre fantôme durant la SEP; pour enregistrer ces sensations, nous vous fournirons une sorte de cadran que vous aurez à actionner. Toute la procédure devrait durer environ deux heures chaque fois.

La SEP est un traitement bien reconnu pour les problèmes de douleur chronique. Cette forme de stimulation produit une sensations de légere battements sur les oreilles et n'est pas acunement douloureuse ou dangereuse. On ne connaît pas d'effet nuisible à ce type de traitement mais on ne le recommande pas chez les patients ayant des problèmes de peau, chez les femmes enceintes ou chez les gens qui ont un pacemaker. Veillez nous informer, s'il-vous-plaît, si vous appartenez à l'une ou l'autre de ces groupes.

Si vous décider de participer à cette recherche, nous tenons à vous assurer que toute l'information que vous fournirez restera strictement confidentielle et sera utilisée de façon anonyme pour des fins de recherche et de publication. Votre participation serait grandement appréciée puisqu'elle nous aidera à améliorer les traitements pour les personnes qui souffrent de douleur reliée à un membre fantôme.

J'ai lu et je comprends parfaitement l'information présentée ci-haut. J'accepte de participer à ce projet de recherche tel qu'il est décrit ci-haut. Il est entendu que je peux me retirer de cette étude à n'importe quel moment.

_____ _____

Date Signature du Participant(e)

_____

Signature du Témoin

_____

Signature du Responsable de la Recherche

APPENDIX B-2

McGill Comprehensive Pain Assessment Schedule
and
McGill Pain Questionnaire

Date of interview: _____ **Time:** _____

Name: _____ **Age:** _____

Date of birth: _____

Address: _____

_____

Phone number: _____ **Group:** _____

Hospital: _____ **Amputation:**

Unit number: _____ L☐ A☐ E☐ R☐ B☐ K☐

1. Pain History

(a) When was your limb amputated? Year: _____ Month: _____

(b) What was the cause of the amputation? Please check (✓) the appropriate box:

- ☐ peripheral vascular disease
- ☐ diabetes: If diabetic for how long? _____
- ☐ tumour Regimen: _____
- ☐ work accident
- ☐ car accident
- ☐ motorcycle accident
- ☐ combat injury
- ☐ other: _____

(c) Have you ever felt pain in the part of the limb that is no longer present (i.e., in the phantom limb)? ☐ yes ☐ no

If yes, how long after the amputation did you first feel pain in the phantom limb? _____

(d) Since the time you first felt pain in the phantom limb, the pain has:

- ☐ increased in ..... ☐ intensity
- ☐ decreased in ..... ☐ frequency
- ☐ gone away ☐ duration
- ☐ gone away and returned
- ☐ remained the same

How long have you had phantom limb pain? _____

(e) Did you have pain in your limb **before** it was amputated? ☐ yes ☐ no

If yes, for how long before the amputation? _____

Name: _____

Is the phantom limb pain that you now have the same as or similar to the pain that you had in your limb **before** it was amputated? ☐ yes ☐ no

If yes, please describe the similarity. _____

_____

_____

Similarity of:
☐ Location
☐ Quality
☐ Intensity

If no, is the phantom pain that you now have the same as or similar to **any** pain you had in your limb **before** it was amputated? ☐ yes ☐ no

If yes, please describe. _____

_____

Similarity of:
☐ Location
☐ Quality
☐ Intensity

(f) Is your stump painful?

☐ Yes ☐ No

If yes, describe the pain:

_____

_____

_____

_____

_____

(g) How often do you feel pain in the:

PL	STUMP	
☐	☐	constantly
☐	☐	once a day or more frequently
☐	☐	once a week or more frequently
☐	☐	once a month or more frequently
☐	☐	less than once a month

(h) When the pains begin how long do they usually last?

PL	STUMP	
☐	☐	seconds
☐	☐	minutes
☐	☐	hours
☐	☐	days

Types of PLP:
☐ Burning
☐ Paresthesias, pins & needles
☐ Jabs, bursts, shots, attacks
☐ Twisted posture
☐ Cramping
☐ Memory-like

(i) What parts of the phantom limb do they come from? _____

_____

2. Pain Modifiers

For each of the following: please mark with a **+** if it increases the pain;
please mark with a **-** if it decreases the pain;
please mark with a **0** if it has no effect on the pain

☐ cold weather
☐ warm weather
☐ exercise
☐ wearing a prosthesis
☐ massaging the stump
☐ touching the stump
☐ objects approaching the stump
☐ loud noises
☐ urination
☐ defecation
☐ sexual excitement/orgasm
☐ coughing or sneezing
☐ illness or colds
☐ smoking tobacco
☐ drinking alcohol
☐ bright lights

- ☐ anxiety or worry
- ☐ certain thoughts (e.g. _____)
- ☐ certain feelings (e.g. _____)
- ☐ certain memories (e.g. _____)
- ☐ certain TV or movie scenes (e.g. _____)
- ☐ reading certain material (e.g. _____)
- ☐ certain topics of conversation (e.g. _____)
- ☐ the presence of certain people (e.g. _____)
- ☐ social situations (e.g. _____)

Please list any other things that can affect the pain:

1. _____
2. _____
3. _____

3. Phantom Limb Description

(a) Please check (✓) the appropriate box or boxes that best describe what parts of your phantom limb you feel **right now.** For example, if your phantom consists of all 5 toes and the foot you would check the box marked "all 5 toes" and the box marked "foot".

Upper limb

- ☐ 4 fingers and thumb
- ☐ some fingers (specify _____)
- ☐ hand
- ☐ wrist
- ☐ forearm
- ☐ elbow
- ☐ upper arm
- ☐ shoulder

Lower limb

- ☐ all 5 toes
- ☐ some toes (specify _____)
- ☐ foot
- ☐ ankle
- ☐ lower leg
- ☐ knee
- ☐ upper leg
- ☐ hip

Compared to your other foot (hand) is the phantom foot (hand):
- ☐ longer
- ☐ the same length
- ☐ shorter (describe: e.g., telescoped into stump)

Posture:
- ☐ Relaxed
- ☐ Fixed
- ☐ Distorted

(b) In what position is your phantom limb **right now?**

Upper limb
- ☐ straight at your side
- ☐ bent at the elbow
- ☐ finger clenched in a fist
- ☐ other _____

Lower limb
- ☐ leg straight
- ☐ bent at the knee
- ☐ toes flexed
- ☐ other _____

(c) Is your phantom limb always in this position? Yes ☐ No ☐

If no, please indicate how it changes and when. _____

4. Habits

(a) Do you smoke? Yes ☐ No ☐ If yes, what/how much? _____

When did you last smoke? _____

(b) Please indicate the number of cups/bottles you drink of the following each day:

coffee _____ tea _____ cola _____

When was your last cup of coffee? _____

(c) Do you drink alcohol? Yes ☐ No ☐ If yes what type of alcoholic beverages?

When did you last have a drink? _____

5. Past and present medical history

(a) Please list any illnesses you have had at any age. Also include allergies, hospitalizations, operations, anesthetic procedures and psychological illnesses.

Name: _____

	Year	Problem	Treatment
1.	_____	_____	_____
2.	_____	_____	_____
3.	_____	_____	_____
4.	_____	_____	_____
5.	_____	_____	_____
6	_____	_____	_____

(b) Please list any illness or health problems other than the phantom limb pain that you may have now (i.e., high blood pressure, ulcer, etc.)

	Problem	Treatment
1.	_____	_____
2.	_____	_____
3.	_____	_____
4.	_____	_____
5.	_____	_____
6.	_____	_____

(c) Do you take medication for the pain? yes ☐ no ☐ If yes, please list all medication you are currently taking for the pain.

1. _____
2. _____
3. _____

(d) When did you last take medication for the pain? _____

Treatments

- ☐ TENS
- ☐ Acupuncture
- ☐ CTM, Acupressure
- ☐ Local injections
- ☐ Medications
- ☐ Surgery
- ☐ Psychotherapy
- ☐ Relaxation
- ☐ Hypnosis

Other treatments:

1. _____
2. _____
3. _____

	Were any effective?	For how long?
1.	_____	_____
2.	_____	_____
3.	_____	_____
4.	_____	_____

6. Personal Information

☐ Married For how long? _____
☐ Single
☐ Widowed Children: Sons ____ Daughters ____
☐ Separated
☐ Divorced

Has prosthesis: Yes No
Uses Prosthesis: Yes No

Living with ____ Others

If no, why not: _____

Education: _____

What type of work do you or did you last have (include housewife)?

_____

For how long? _____

Are there any hobbies, sports, recreational and social activities that you no longer do because of the pain (and/or the amputation)?

1. _____
2. _____
3. _____

What hobbies, sports, etc. do you still do?

1. _____
2. _____
3. _____

Do any of them take your mind off the pain? yes ☐ no ☐

If yes, which of the above?

1.
2.
3.

What is the most difficult aspect about having an amputation? _____

_____

How much does the phantom limb pain interfere with your daily life?

☐ Not at all
☐ Somewhat
☐ Moderately
☐ Very much
☐ Completely

McGill - Melzack Pain Questionnaire

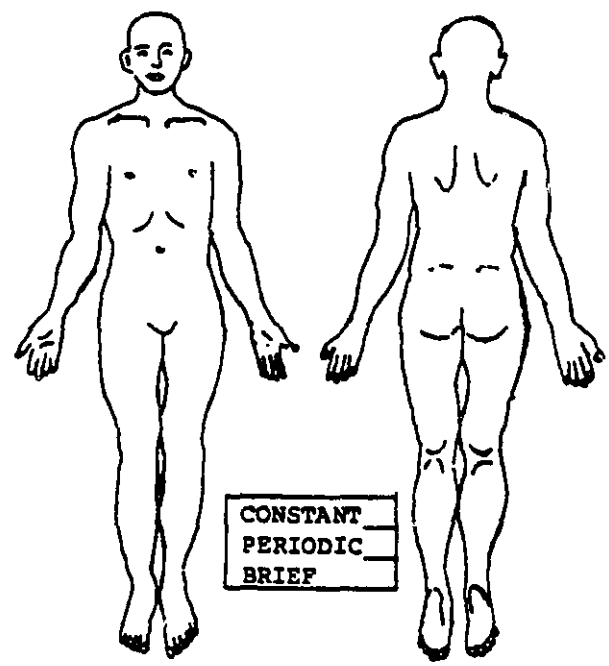
Patient's Name_____ Date_____ Time_____ am/pm
Analgesic(s) _____ Dosage_____ Time Given _____ am/pm
_____ Dosage_____ Time Given_____ am/pm

Analgesic Time Difference (hours): +4 +1 +2 +3

PRI: S_____ A_____ E_____ M(S)_____ M(AE)_____ M(T)_____ PRI(T)____
(1-10) (11-15) (16) (17-19) (20) (17-20) (1-20)

1 FLICKERING	11 TIRING	
QUIVERING	EXHAUSTING	
PULSING	12 SICKENING	
THROBBING	SUFFOCATING	
BEATING	13 FEARFUL	
POUNDING	FRIGHTFUL	
2 JUMPING	TERRIFYING	
FLASHING	14 PUNISHING	
SHOOTING	GRUELLING	
3 PRICKING	CRUEL	
BORING	VICIOUS	
DRILLING	KILLING	
STABBING	15 WRETCHED	
LANCINATING	BLINDING	
4 SHARP	16 ANNOYING	
CUTTING	TROUBLESOME	
LACERATING	MISERABLE	
5 PINCHING	INTENSE	
PRESSING	UNBEARABLE	
GNAWING	17 SPREADING	
CRAMPING	RADIATING	
CRUSHING	PENETRATING	
6 TUGGING	PIERCING	
PULLING	18 TIGHT	
WRENCHING	NUMB	
7 HOT	DRAWING	
BURNING	SQUEEZING	
SCALDING	TEARING	
SEARING	19 COOL	
8 TINGLING	COLD	
ITCHY	FREEZING	
SMARTING	20 NAGGING	
STINGING	NAUSEATING	
9 DULL	AGONIZING	
SORE	DREADFUL	
HURTING	TORTURING	
ACHING	PPI	
HEAVY	0 No pain	
10 TENDER	1 MILD	
TAUT	2 DISCOMFORTING	
RASPING	3 DISTRESSING	
SPLITTING	4 HORRIBLE	
	5 EXCRUCIATING	

PPI_____ COMMENTS:



CONSTANT
PERIODIC
BRIEF

ACCOMPANYING SYMPTOMS:	SLEEP:	FOOD INTAKE:
NAUSEA	GOOD	GOOD
HEADACHE	FITFUL	SOME
DIZZINESS	CAN'T SLEEP	LITTLE
DROWSINESS	COMMENTS:	NONE
CONSTIPATION		COMMENTS:
DIARRHEA		
COMMENTS:	ACTIVITY:	COMMENTS:
	GOOD	
	SOME	
	LITTLE	
	NONE	