The Reality of Phantom Limbs[1]

Joel Katz[2]

This paper evaluates the joint influence of peripheral neurophysiological factors and higher-order cognitive and affective processes in triggering or modulating a variety of phantom limb experiences, including pain. Part 1 outlines one way in which the sympathetic nervous system may influence phantom limb pain. A model involving a sympathetic-efferent somatic-afferent cycle is presented to explain fluctuations in the intensity of sensations referred to the phantom limb. In part 2, the model is extended to explain the puzzling finding that only after *amputation are thoughts and feelings capable of evoking referred sensations to the (phantom) limb. While phantom pains and other sensations frequently are triggered by thoughts and feelings, there is no evidence that the painful or painless phantom limb is a symptom of a psychological disorder. In part 3, the concept of a pain "memory" is introduced and described with examples. The data show that pain experienced prior to amputation may persist in the form of a memory referred to the phantom limb causing continued suffering and distress. It is argued that two independent and potentially dissociable memory components underlie the unified experience of a pain memory. This conceptualization is evaluated in the context of the surgical arena, raising the possibility that under certain conditions postoperative pain may, in part, reflect the persistent central neural memory trace left by the surgical procedure. It is concluded that the experience of a phantom limb is determined by a complex interaction of inputs from the periphery and widespread regions of the brain subserving sensory, cognitive, and affective processes.*

[1]Preparation of this manuscript was supported by a Medical Research Council of Canada Research Fellowship.

[2]Address all correspondence concerning this article to Joel Katz, Ph.D., Department of Psychology, The Toronto Hospital, General Division, 200 Elizabeth Street, CW2-306, Toronto, Ontario, M5G 2C4, Canada.

Many patients awaken from the anaesthetic after an amputation believing that the operation has not been performed. Their perception of the lost limb is so real that not until they lift the bed sheets to see it do they realize it has been cut off. This startling realization has little effect on the reality of the limb they experience, and in some cases may even intensify the sensations that define it. Weir Mitchell (1871) coined the term *phantom limb* to describe the persisting sensory awareness of a limb after amputation.

A distinction is usually made between the painful and nonpainful phantom limb (Melzack & Wall, 1988). The most salient property of the nonpainful phantom is its tingling, "pins-and-needles" feeling (paresthesias), but other qualities of sensation include temperature, posture, length, volume, and movement (Jensen & Rasmussen, 1989). Recent studies estimate the incidence of the nonpainful phantom at approximately 80% to 100% (Jensen & Rasmussen, 1989). For many amputees, however, a distressing problem is phantom limb *pain* (Sherman, 1989). Dysesthesias are common; many patients report a painful intensification of the paresthesias or pins-and-needles sensation that defines the nonpainful phantom limb. Some sufferers describe bouts of paroxysmal shooting pains that travel up and down the limb. Others report the phantom to be in a cramped or otherwise unnatural posture that gives rise to excruciating pain. Many amputees describe the pain in the phantom limb as indistinguishable from the pain they experienced in the limb prior to amputation. In still others, the phantom is the seat of an intense burning pain as if the hand or foot were being held too close to an open flame. Frequently amputees suffer from several types of pain (Jensen & Rasmussen, 1989).

A recent survey based on several thousand amputees reveals that more than 70% continue to experience phantom limb pain of considerable intensity more than 25 years after amputation (Sherman, Sherman, & Parker, 1984). Equally striking is the low success rate of treatments for phantom limb pain: In the long term only 7% of patients are helped by the more than 50 types of therapy used to treat phantom limb pain (Sherman, 1989). This intractability reflects our ignorance about the mechanisms that contribute to phantom limb pain.

A controversy has arisen over the origin of the phantom limb. In an attempt to find a single explanatory mechanism, theories have focused on only one aspect of phantom limb experience and have ignored or discounted others (Melzack & Wall, 1988). The cause has been sought in the activity of primary afferent fibers, spinal cord cells, and supraspinal sensory nuclei (Jensen & Rasmussen, 1989; Melzack & Wall, 1988). Another class of theory has attempted to account for the phantom solely on the basis of psychological and emotional processes (Szasz, 1975). Melzack (1989) recently concluded that the phantom limb cannot be explained by a unitary mechanism

—whether peripheral, central, or psychological. He proposed a theory in which the simultaneous outputs of neural networks in widespread regions of the brain combine to produce the various qualities of human experience. This paper reviews some of the peripheral, central, psychological, and emotional factors that contribute to phantom limb experience.

SYMPATHETIC NERVOUS SYSTEM CONTRIBUTIONS TO PHANTOM LIMB EXPERIENCE

Phantom Limb Pain

Evidence that the sympathetic nervous system is involved in phantom limb and stump pain comes from studies of procedures that pharmacologically block (Livingston, 1943) or surgically interrupt (Kallio, 1950) the sympathetic supply to the involved limb with at least temporary alleviation of pain. Transient relief from phantom limb pain also has been reported with propranolol (Marsland, Weekes, Atkinson, & Leong, 1982). Electrical and mechanical stimulation of the lumbar sympathetic chain produces intense pain referred to the phantom limb whereas, in nonamputee pain patients, the sensations are referred to the abdomen or flank (Noordenbos, 1959). Regional sympathetic hyperactivity may also contribute to the development of phantom limb pain through excessive vasoconstriction and sweating at the stump and surrounding regions (Livingston, 1943). The condition may spread centrally from the stump to involve the phantom limb. Hyperalgesia (heightened pain) and allodynia (pain arising from gentle touch) may be referred to the phantom limb upon stimulation of the stump whether or not the stump is painful or shows signs of trophic or vascular changes (Doupe, Cullen, & Chance, 1944). The characteristic qualities of superficial burning pain and deep aching pain may provide additional evidence of sympathetic nervous system involvement (Doupe et al., 1944).

Despite frequent assertions that the sympathetic nervous system is involved in the production and maintenance of phantom limb pain, surprisingly few studies have compared correlates of peripheral sympathetic nervous system activity at the stump and contralateral limb. Sliosberg (1948) examined 141 amputees and found the stump to be cooler than the intact limb in 94 of them but he did not relate the temperature difference to the presence or absence of phantom limb pain. Kristen, Lukeschitsch, Plattner, Sigmund, and Resch (1984) 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 or without phantom limb pain. Nyström and Hagbarth (1981) made microneurographic recordings of activity from muscle nerve fascicles of the peroneal nerve in a patient with a below-knee amputation who suffered from intense cramping pain referred to the phantom foot. Although bursts of activity in sympathetic fibers were accentuated by the Valsalva maneuver, the phantom pain remained unchanged suggesting that, in this patient, the cramping pain was independent of peripheral sympathetic nervous system activity.

In contrast, Sherman and his colleagues (Sherman, 1984; Sherman & Bruno, 1987) recently observed a negative correlation between temperature at the stump and the presence of burning phantom limb and stump pain indicating that reduced blood flow to the stump is associated with increased levels of pain. Katz (1992) followed up this line of inquiry and compared skin conductance and surface skin temperature of the stump and contralateral limb in amputees reporting phantom limb pain (Group PLP), nonpainful phantom limb sensations (Group PLS), or no phantom limb at all (Group No PL). The results showed that mean skin temperature was significantly lower at the stump than the contralateral limb in Groups PLP and PLS, but not Group No PL.

These results suggest that the presence of a phantom limb, whether painful or painless, is related to the sympathetic-efferent outflow of cutaneous vasoconstrictor fibers in the stump and stump neuromas. The related finding that stump skin conductance responses over time correlated significantly with the intensity of phantom limb paresthesias, but not other qualities of sensation, supports the hypothesis (outlined below) of a sympathetic-efferent somatic-afferent mechanism involving both sudomotor and vasoconstrictor fibers. The most parsimonious explanation of these findings is that the paresthetic or dysesthetic component of the phantom limb may be triggered by sympathetic-efferent activity (Katz, 1992).

Phantom Limb Paresthesias

Although a normal phantom occurs whenever nerve impulses from the periphery are blocked or otherwise removed (Wall, 1981), it is also true that direct stimulation of the amputation stump frequently exaggerates the tingling or paresthetic quality of sensation typical of the painless phantom limb (Carlen, Wall, Nadvorna, & Steinbach, 1978). Careful questioning of amputees reveals that the nonpainful phantom limb is not perceived as a static phenomenon. The paresthetic quality of sensation, which defines

the phantom limb percept, is in a constant state of flux, with changes occurring in intensity, body part, or both. For example, Katz, France, and Melzack (1989) reported a subject whose phantom sensations consisted of a "numbness" that defined a region including the lateral three toes. Within this circumscribed area, he experienced rapid "waves of numbness" that increased and decreased the intensity of the involved phantom parts.

One mechanism that has been proposed to account for the paresthetic component of the phantom limb is a cycle of sympathetic-efferent somatic-afferent activity (Katz, 1992; Katz et al., 1989). As shown in Fig. 1, stump skin conductance levels correlate significantly over time with the intensity of phantom limb paresthesias. It is hypothesized that changes in the intensity of phantom limb paresthesias reflect the joint activity of cholinergic (sudomotor) and noradrenergic (vasomotor) postganglionic sympathetic fibers on primary afferents located in the stump and stump neuromas (Fig. 2). Release of acetylcholine and noradrenaline from postganglionic

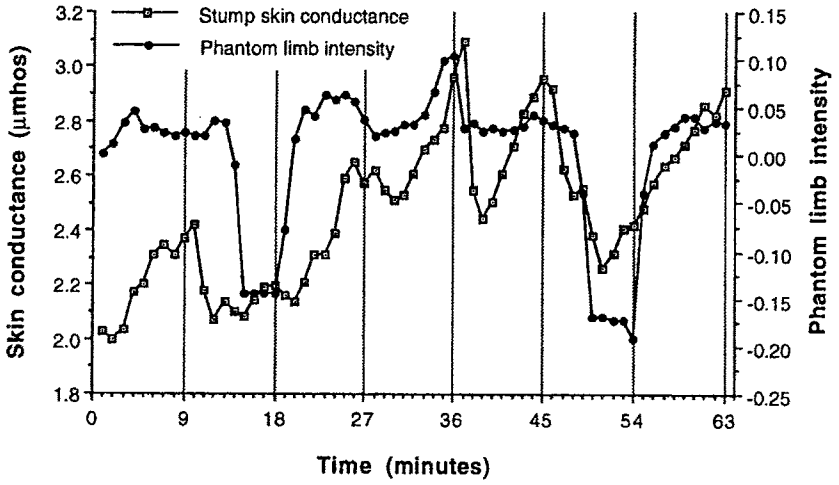

Fig. 1. A minute-by-minute plot of the relationship between stump skin conductance and the intensity of nonpainful phantom limb paresthesias for a subject with an amputation above the knee. Skin conductance was continuously measured at the stump over a 63-min period while the subject monitored the intensity of the phantom limb by turning a dial. Phantom limb intensity ratings have been transformed so that a value of 0.0 represents the intensity at the start of the session and deviations from zero correspond to increases and decreases in phantom limb intensity. Each data point represents a mean of 30 values consecutively sampled at 2-s intervals. Note that changes in the intensity of paresthesias (described by the subject as increases and decreases in "numb" sensations referred to the phantom toes) occur in concert with changes in stump skin conductance. (Adapted from Katz et al., 1989, with permission.)

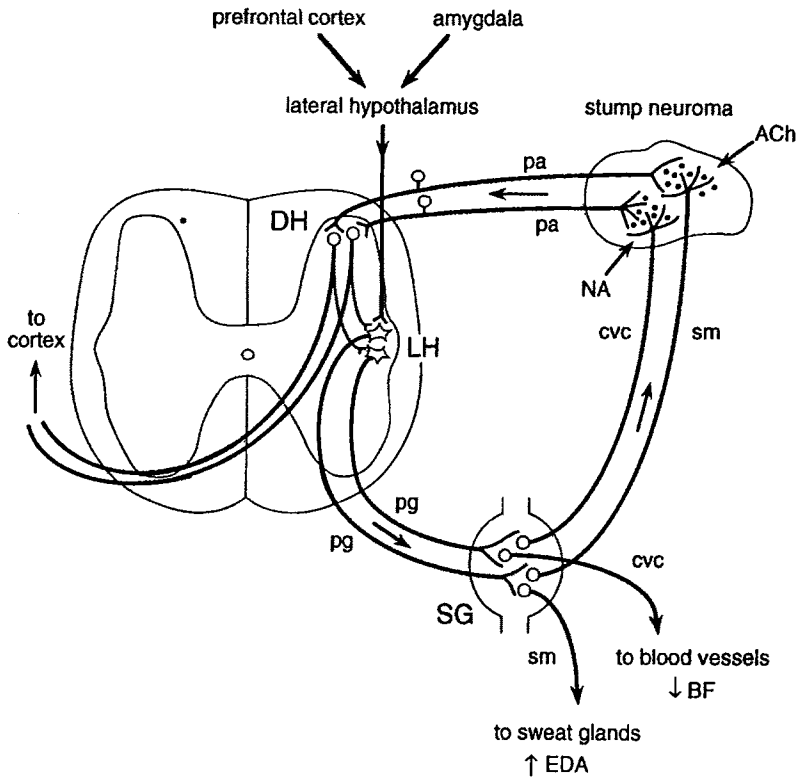

Fig. 2. Schematic diagram illustrating a mechanism of sympathetically-generated phantom limb paresthesias. Spontaneous activity or excitatory inputs descending from cortex (e.g., due to the perception of a salient event, loud noise, thought, feeling, etc.) increase the discharge rate of preganglionic (pg) sympathetic neurons with cell bodies in the lateral horn (LH) of the spinal cord and terminals in the sympathetic ganglion (SG). These neurons excite postganglionic noradrenergic (NA) cutaneous vasoconstrictor (cvc) and cholinergic (ACh) sudomotor (sm) fibers that impinge on effector organs (vascular smooth muscle and sweat glands) in the stump and on sprouts from large diameter primary afferent (pa) fibers that have been trapped in a neuroma. The release of ACh and NA on effector organs results in increased electrodermal activity (EDA) and decreased blood flow (BF) to the stump. Release of these chemicals in the neuroma activates primary afferents that project to spinal cord dorsal horn (DH) cells subserving the amputated parts of the limb. These neurons, in turn, feed back to the preganglionic sympathetic neurons and project rostrally where the impulses contribute to the perception of phantom limb paresthesias. If DH cells have been sensitized due to injury, or nociceptive primary afferents are activated, then the perception may be dysesthetic. (Reproduced with permission from Katz, 1992.)

sympathetic fibers produces transient vasoconstriction and heightened skin conductance responses. As well, neurotransmitter release onto apposing peripheral fibers trapped in stump neuromas increases primary afferent dis-

charge. This information is transmitted rostrally where it gives rise to referred phantom sensations upon reaching central structures subserving the amputated parts of the limb. The moment-to-moment fluctuations in the intensity of phantom limb paresthesias reported by many amputees may, in part, reflect a cycle of sympathetic-efferent somatic-afferent activity. Increases in the intensity of phantom limb paresthesias would follow bursts of sympathetic activity and decreases would correspond to periods of relative sympathetic inactivity.

The possibility that heightened electrodermal activity at the stump occurs as a *consequence* of the perception of a change in the intensity of paresthesias does not appear to be tenable, since shooting pains, somatosensory memories, and phantom limb movements do not also correlate with stump skin conductance (Katz, 1992). That is, changes in stump skin conductance are related only to the perception of paresthesias and not to other qualities of sensation. Thus, the paresthetic component of the phantom limb represents the perceptual correlate of a central autonomic mechanism that operates on peripheral structures. In the following section, this mechanism is elaborated to explain how psychological and emotional processes alter phantom limb sensations.

PSYCHOLOGICAL AND EMOTIONAL CONTRIBUTIONS TO PHANTOM LIMB EXPERIENCE

It is not surprising that amputees suffering with phantom limb pain exhibit higher than normal levels of psychological and emotional distress. Depression (Caplan & Hackett, 1963; Lindesay, 1985; Sherman, Sherman, & Bruno, 1987; Shukla, Sahu, Tripathi, & Gupta, 1982), anxiety (Parkes, 1973; Shukla et al., 1982), and other forms of psychopathology are common (Morgenstern, 1970; Parkes, 1973; Shukla et al., 1982; Steigerwald, Brass, & Krainick, 1981). Moreover, amputees with severe phantom limb pain score higher on psychological inventories measuring depression (Lindesay, 1985) and neuroticism (Morgenstern, 1970) than do amputees who have little or no pain.

The occurrence of phantom limb pain and psychological disturbance has led to three conclusions: (1) Pain is a symptom of a psychological disorder (Parkes, 1973; Szasz, 1975), (2) psychological disturbance is a consequence of pain (Sherman & Bruno, 1987), or (3) the two are causally unrelated (Caplan & Hackett, 1963). At present, the consensus is that there is no difference in the prevalence rates of pain of psychological origin among amputees and the general population. There is no evidence to suggest that surgical amputation predisposes an individual to develop pain of

psychological origin, nor that patients who undergo amputation are at greater risk for developing such pain. However, a prospective study has yet to be conducted in which preoperative measures of psychological and emotional functioning are obtained sufficiently prior to amputation so as to avoid the confounding effects of preamputation pain and hospitalization.

Psychodynamic Explanations

Psychodynamic explanations of phantom limb phenomena have been advanced as evidence of the amputee's difficulty in adapting to the mutilated state (Frazier & Kolb, 1970; Parkes, 1973; Parkes & Napier, 1975; Szasz, 1975). Denial (of the loss or the associated affect) and repression are the most common defense mechanisms proposed to explain the presence of a painless phantom (Szasz, 1975), painful phantom (Parkes, 1973; Parkes & Napier, 1975; Stengel, 1965; Szasz, 1975), and various alterations in the form of the phantom limb (Abramson & Feibel, 1981; Weiss, 1958).

Though often elegantly formulated, psychodynamic explanations are not consistent with the accumulation of physiological and psychological data. For example, many amputees become profoundly depressed after surgery, yet phantom pain and other sensations persist. The cooccurrence of depression and pain is inconsistent with the role of denial since the intense negative affect implies awareness, if not acceptance, of the loss (Caplan & Hackett, 1963). In fact, for many amputees, the affect associated with the loss is so overwhelming that it cannot be contained and seems to "spill over" into the phantom thereby increasing the intensity of paresthesias (Simmel, 1959).

There are other inconsistencies between psychodynamic theory and empirical evidence. Apparently healthy individuals who, by all objective measures, have adjusted to the amputation continue to report the presence of a phantom years after amputation (Simmel 1959). Phantoms that occur after injury to the central nervous system (CNS) (e.g., when sensory and motor nerve roots are torn from the spinal cord or the spinal cord is transected) are similar to amputation phantoms in quality of sensation even though the real limb(s) is still present but totally anaesthetic and paralyzed. One would not expect denial of the loss of function to produce a phantom defined by paresthesias (Weinstein, 1962). Phantoms do not develop if the process of sensory loss is gradual, as in leprosy (Price, 1976), yet there should be as great a need for denial in these cases. Finally, procedures that temporarily block the supply of afferent impulses from reaching the CNS (e.g., anaesthetic nerve blocks, blood pressure cuff occlusion) reliably result in the perception of a phantom limb which persists until the flow of

afferent input has been restored (Melzack & Bromage, 1973; Wall, 1981). Under these circumstances, it is difficult to see the need of a phantom limb to fulfill the putative ego-protective function of defending the individual from a loss.

Although denial is more commonly associated with diseases that have no visual evidence of infirmity (Caplan & Hackett, 1963), the foregoing does not imply that denial of the loss, affect, illness, or future implications plays no part in the overall adaptation to amputation (Rosen, 1950). Patients may demonstrate their denial of the importance of these realities in a variety of ways (Bradway, Malone, Racy, Leal, & Poole, 1984; Turgay & Sonuvar, 1983), but these do not include having a phantom. For the vast majority of amputees, the presence of a phantom limb — painful or painless — is not a symptom of a psychological disorder.

Characterological Disturbances

In addition to the role of specific defense mechanisms in the genesis of phantom limb pain, it is postulated that phantom limb pain may be psychologically determined by characterological disturbances such as "compulsive self-reliance" and "rigidity" (Parkes, 1973). With the exception of a recent review (Sherman et al., 1987), the idea that patients with persisting phantom limb pain are rigid and exhibit compulsively self-reliant personality characteristics has been uncritically accepted by researchers and clinicians working in the field of phantom limb pain (Dawson & Arnold, 1981; Dernham, 1986; Lundberg & Guggenheim, 1986; Shukla et al., 1982) despite the absence of empirical evidence to support this view.

The association between the presence of pain and psychological distress (e.g., depression and anxiety) or particular personality traits or styles (e.g., rigidity and compulsive self-reliance) may be influenced by biased sampling procedures so that the characteristics of a select group of patients (e.g., those referred to a pain center) come to define the population at large (Merskey, 1989; Sherman et al., 1987). Sherman et al. suggested 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. Long after less assertive patients have given up actively seeking help, these sufferers of phantom limb pain continue to search for relief despite repeated failures. According to Sherman et al., this self-selection bias explains 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.

Recent studies indicate that among an unselected sample of amputees, those with phantom limb pain, painless phantom limb sensations, or no phantom limb at all cannot be distinguished by their scores on the Eysenck Personality Inventory, Beck Depression Inventory, Spielberger State or Trait Anxiety Inventory, or a mood rating scale (Katz & Melzack, 1990, 1991). Moreover, the results show that there are no significant intergroup differences in scores on a questionnaire designed to measure psychological rigidity as defined by a tendency to persist in behaviors that were effective at one time, or in a particular situation, but no longer are adequate to accomplish current goals.

Psychological and Emotional Processes Influence Phantom Limb Experience

As reviewed above, idea that emotional and psychological processes can cause pain traditionally has been tied to the notion of psychopathology. However, it is becoming increasingly clear that under certain circumstances pain may be triggered by these processes in psychologically healthy individuals as well. Although instances of psychologically or emotionally triggered pain and psychopathology may be present in the same amputee, their cooccurrence should not be taken as *prima facie* evidence of a causal link.

It is commonly accepted that anxiety or stress influences pain perception and subsequent behavior (Merskey, 1989). The aggravation or alleviation of pain referred to phantom body parts also may be mediated in part by psychological processes that alter anxiety levels (Kolb, 1954). Phantom breast pain after mastectomy is provoked by emotional distress in 6% of women 3 weeks after surgery and in 29% 1 year later (Krøner, Krebs, Skov, & Jørgensen, 1989). Fifty percent of lower-extremity amputees report that attacks of phantom limb pain are triggered by emotional distress (Jensen, Krebs, Nielsen, & Rasmussen, 1985) as long as 7 years after amputation (Krebs, Jensen, Krøner, Nielsen, & Jørgensen, 1985). A combination of progressive relaxation training and electromyographic biofeedback of stump and forehead muscles produces significant reductions of phantom limb pain and anxiety (Sherman, 1976) that are sustained for up to 3 years (Sherman, Gall, & Gormly, 1979). Finally, stress levels and pain intensity ratings sampled over a 180-day observation period correlate significantly for most amputees (Arena, Sherman, & Bruno, 1990).

There are also examples of psychological or emotional processes precipitating transient but profound alterations in the quality and intensity of phantom limb sensations. These processes include hypnosis (Schilder, 1950), concentration (Morgenstern, 1964; Riddoch, 1941), distraction (Parkes, 1973), relaxation (Sherman, 1976; Sherman et al., 1979), fright

(Henderson & Smyth, 1948), forceful reminders of the events that led to amputation (Simmel, 1956), the sight of other amputees (Simmel, 1956), and witnessing cruel and violent acts (Pilowsky & Kaufman, 1965; Stengel, 1965). One amputee, interviewed by the present writer, described his reaction to an accident involving his wife by reporting " . . . goose bumps and cold shivering down the phantom [leg]. It went through me. Everything emotional will get you that." Another amputee stated, "It's like everything I feel goes there — the good and the bad."

A Centrally Triggered Sympathetic-Efferent Somatic-Afferent Mechanism

The material presented above indicates that cognitive and affective processes reliably trigger transient pains or sensations referred to the phantom limb. The model schematically represented in Fig. 2 outlines a mechanism through which cognitive and affective processes associated with higher cortical and limbic centers may alter phantom limb sensations. The reciprocal connections between cortical, limbic and lateral hypothalamic structures are well documented (Brodal, 1981; Smith & DeVito, 1984). The lateral hypothalamus is involved in the control and integration of neural activity associated with affectively charged behavior (Brodal, 1981; Melzack & Casey, 1968; Smith & DeVito, 1984) and has direct projections to the lateral horn of the spinal cord. The intensity of phantom limb paresthesias and dysesthesias may thus be modulated by higher brain centers involved in cognitive and affective processes *via* a multisynaptic network of descending inputs that impinges on preganglionic sympathetic neurons producing diffuse peripheral autonomic discharge and activation of primary afferent fibers located in stump neuromas.

Occasionally, the effects of intense affect (e.g., fright, horror) are experienced diffusely over the entire body as *cutis anserina* associated with pilomotor contraction (i.e., "goose bumps"). Among amputees, however, a more frequent occurrence is that the perception of less salient events and emotions precipitates these sensations throughout only the phantom limb. The tendency for affectively charged and psychologically meaningful experiences to be referred to the phantom limb, but not to other parts of the body, is consistent with two lines of evidence suggesting that the threshold for impulse generation is lower both in regenerating primary afferents in the stump and in deafferented central cells subserving the phantom limb than it is in the intact nervous system.

First, regenerating sprouts, which are trapped in a neuroma, are exceedingly sensitive to the postganglionic sympathetic neurotransmitters noradrenaline (Wall & Gutnick, 1974) and acetylcholine (Diamond, 1959),

and they discharge rapidly when these substances are present. In contrast, intact peripheral fibers do not show this chemosensitivity, and thus have a higher threshold compared with regenerating sprouts. Second, the loss of afferent nerve impulses (deafferentation) resulting from amputation produces a disinhibition of cells in the dorsal horn and more rostral sensory structures giving rise to the perception of a phantom limb (Melzack & Loeser, 1978; Wall, 1981). This consequence of deafferentation implies that the threshold for detecting sympathetically triggered afferent impulses arising from stump neuromas should be lower than at other, intact body sites since stump impulses would be subject to less inhibition upon reaching the spinal cord. This fits well with the observation that the threshold for detecting sensations in the phantom limb during stimulation of the stump is lower than at the site of stimulation itself (Carlen et al., 1978).

Another possibility is that amputation leads to increased expression of alpha-1 adrenergic receptors located on mechanoreceptors or nociceptors (Campbell, Meyer, Davis, & Raja, 1992) in stump neuromas. This hypothesis would explain the perception of phantom limb paresthesias or dyesthesias in the absence of regional sympathetic hyperactivity. Taken together, these observations may explain the puzzling finding that only after amputation does the (phantom) limb become the site of affectively or cognitively triggered sensations.

The suggestion that the perception of phantom limb sensations may reflect the activity of postganglionic sympathetic fibers on stump primary afferents is obviously not meant to imply that paresthesias arise only from a peripheral source. Blocking the afferent supply to a body region is sufficient to produce the experience of a painless phantom defined by paresthesias (Melzack & Bromage, 1973; Wall, 1981) and electrical stimulation of the medial lemniscal pathway gives rise to the sensation of paresthesias referred to the territory subserved by the cells being stimulated (Tasker, Organ, & Hawrylyshyn, 1982). Moreover, it is likely that through repeated activation, neural circuitry is strengthened among brain regions subserving cognitive, affective, and sensory processes so that phantom limb sensations and pain may be triggered by thoughts and feelings in the absence of primary afferent feedback from peripheral structures (LeDoux, 1989; Leventhal, 1982).

Implications for Treatment of Phantom Limb Pain

Given that cognitive and affective processes may trigger or exacerbate phantom limb pain, it is of the utmost importance that patients be prepared prior to amputation for the presence of a phantom limb. Patient education programs and treatment of stress prior to and after amputation have be-

come standard practice (Butler, Turkal, & Seidl, 1992; McGrath & Hillier, 1992; Sherman, 1989). Patients who are ill prepared psychologically for amputation suffer needlessly with phantom limb pain and concern about their sanity (Solomon & Schmidt, 1978).

Finally, it is noteworthy that mental stress and anxiety not only provoke transient increases in the intensity of phantom limb sensations and pain (Arena et al., 1990; Sherman, 1976; Sherman et al., 1979), but they also induce reflex bursting activity in cutaneous sudomotor and vasomotor sympathetic fibers (Delius, Hagbarth, Hongell, & Wallin, 1972; Hagbarth, Hallin, Hongell, Torebjörk, & Wallin, 1972). Moreover, distraction or attention diversion (and intense concentration) which reduces phantom limb pain (Morgenstern, 1964; Parkes, 1973) also diminishes peripheral sympathetic nervous system activity (Hagbarth et al., 1972). These findings provide support for the model shown in Fig. 2 and suggest that relaxation training and other cognitive strategies directed at anxiety reduction and increasing self-control may be effective in reducing phantom limb pain in certain amputees.

PAIN MEMORIES IN PHANTOM LIMBS AND DEAFFERENTED STRUCTURES

A striking property of phantom limb pain is the presence of a pain that existed in a limb prior to its amputation (Melzack, 1971). This class of phantom limb pain is characterized by the persistence or recurrence of a previous pain, has the same qualities of sensation, and is experienced in the same region of the limb as the preamputation pain (Katz & Melzack, 1990). Cases studies of amputees have revealed pain "memories" of painful diabetic foot ulcers, bedsores, gangrene, corns, blisters, ingrown toenails, cuts and deep tissue injuries, and damage to joints and bony structures. As well, the phantom limb 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 prolonged period.

The proportion of amputees who report similar pain before and after amputation may be as high as 79% (Katz & Melzack, 1990). Pain memories in phantom limbs appear to be less common when there has been a discontinuity, or a pain-free interval, between the experience of pain and amputation. This is consistent with the observation that relief of limb pain by continuous epidural blockade for 3 days before amputation decreases the incidence of phantom limb pain 6 months later (Bach, Noreng, & Tjéllden, 1988). Furthermore, compared with pain that is temporally noncontiguous with amputation, pain experienced at or near the time of amputation has

a higher probability of persisting into the phantom limb (Jensen et al., 1985; Katz & Melzack, 1990).

Pain also persists in patients with deafferentation that does not involve amputation. In these conditions, the involved body part is still present but it is devoid of sensibility due to an interruption in the supply of sensory (afferent) information (i.e., deafferentation). Brachial plexus avulsions, in which the sensory nerve roots supplying the arm and hand are torn from the spinal cord, often produce pain that is felt in the deafferented and anesthetic region (Jensen & Rasmussen, 1989; Reisner, 1981). Similarly, patients with spinal cord injuries (Berger & Gerstenbrand, 1981; Conomy, 1973) may complain of pain referred to body parts below the level of the transection. For example, Nathan (1962) described a patient who continued to feel the pain of an ingrown toenail after a complete spinal cord break. As well, patients undergoing spinal anesthesia (Van Bogaert, 1934; Wallgren, 1954) and those with injuries of the brachial plexus or spinal cord sometimes report that a limb is in the same uncomfortable, often painful, posture it was in prior to the injury or block. These postural phantom sensations do not usually persist beyond several days and in most cases are at least temporarily reversed by competing visual inputs which reveal a dissociation between the real and felt limb(s).

Painful and nonpainful sensations also persist or recur after surgical removal or deafferentation of body structures other than the limbs, such as breasts (Krøner et al., 1989), teeth (Marbach, 1978; Sicuteri, Nicolodi, Fusco, & Orlando, 1991), and internal and special sense organs. Ulcer pain has been reported to persist after subtotal gastrectomy with removal of the ulcer (Gloyne, 1954). Patients have reported labor pain and menstrual cramps after total hysterectomy (Dorpat, 1971), rectal pain (Boas, Schug, & Acland, 1993) and hemorrhoids (Oveson, Krøner, Ørnsholt, & Bach, 1991) after removal of the rectum and anus, the burning pain of cystitis after complete removal of the bladder (Brena & Sammons, 1979), and the pain of a severely ulcerated cornea after enucleation of an eye (Minski, 1943).

As noted above, not all phantom limb memories are of painful experiences. Nonpainful preamputation sensations do recur but they are not as common and tend to include the sensation of objects that were once worn on the limb. These *superadded* sensations (Haber, 1956) vary in complexity from such simple somatosensory qualities as the sensation of bandages that once wrapped a wound (Friedmann, 1978; Katz & Melzack, 1990), a plaster cast (Danke, 1981), finger rings, and wristwatches (Friedmann, 1978; Haber, 1956) to the multimodal, perceptually integrated phenomenon of a phantom foot clad in a sock and a shoe of specific type and color (Katz & Melzack, 1990).

Superadded sensations in phantom limbs bear a striking resemblance to a type of tactile hallucination in patients with lesions of the parietal lobe (Allen, 1928; Critchley, 1971). With such "spontaneous stereognostic sensations . . . the patient has a feeling as if something were lying in the palm of one hand. The feeling may be so vivid that the patient can go on to describe the size, shape, texture and temperature of the object, and he may be astonished to find later that the hand is really empty" (Critchley, 1971, p. 91). Allen presents a detailed case study of a patient, who upon recovering from the anesthetic following removal of a large tumor from the left postrolandic sensory cortex, thought he was holding an object in his right hand. Over the next 2 days the shape and size of the objects he felt varied. "At one time he felt a smooth, round object which he described as 'like a ball which just fits into the palm of my hand.' Again, he felt 'something rough and jagged and hard like a piece of road granite.' Later he felt 'a flat round object — like a ladies' small mirror.' He also felt a long, round object 'like a long, round pencil case,' and an object 'like a matchbox'" (Allen, 1928, p. 138).

Taken together, these case reports and studies of amputees reveal that pain memories are not merely images or cognitive recollections; they are direct experiences of pain that resemble an earlier pain in location and quality. They are perceptually complex experiences that may even involve information from multiple sensory modalities including visual, olfactory, tactile, and motor components that had accompanied the original experience. The precise details of the experiences of pain involve localization, discrimination, affect, and evaluation — that is, all the dimensions of perceptual experience — and these properties are a function of integrated brain activity. It is likely that the outputs of sensitized spinal cells activate the neural structures in the brain that subserve memories of earlier events.

Separate Somatosensory and Cognitive Memory Components Underlie Pain Memories

A closer examination of the phenomenon suggests that the experience of a pain memory reflects the joint activity of two separate memory subsystems with properties and functions specialized for processing somatosensory and cognitive (declarative) information, respectively. The *somatosensory memory component* consists of the same, or very similar, neural circuitry that was activated by the peripheral input prior to amputation. It is a higher-order functional unit that codes the temporal and spatial patterning of nerve impulses specifying the body part, quality of sensation, and intensity of the somatosensory experience.

The *cognitive memory component* contains declarative information related to when and in what context the preamputation pain occurred as well as *meta*-information *about* the body part, quality of sensation, and intensity of the preamputation experience. The declarative information contained in the cognitive component provides the unique, personal meaning associated with the somatosensory component and provides a basis for the identifying label and response (e.g., "my pain", a corn, diabetic ulcer, etc.). The determination that a current sensory impression has occurred before involves a process of recognition: 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.

To clarify the distinction between the two forms of memory, consider an amputee who occasionally feels the painful "hole" on his phantom shin corresponding to a long-standing preamputation ulcer as well as the sensation of the bandages that once wrapped the wound. Stripped of the declarative information contained in the cognitive component, which serves to identify and give meaning to the somatosensory qualities of the phantom pain, the sensation of bandages wrapping the wound would probably be described nonspecifically in terms of a band of light pressure or tightness around the leg. That is, the somatosensory descriptions used to convey the sensation are the same regardless of whether or not the cognitive component is present. But when the cognitive component has been activated, the descriptive response includes a unique identifying label (e.g., "bandages"), the phantom limb experience is accompanied by a sense of familiarity, and the patient has access to declarative information that ties the somatosensory qualities of the sensation to the original event.

Evidence of a Double Dissociation Between Somatosensory and Cognitive Components

There is evidence that it is possible to demonstrate a double dissociation of these two memory components. Evidence of the cognitive component in the absence of the somatosensory component is common and occurs whenever amputees recall details about a preamputation pain (e.g., its duration, quality of sensation, location, intensity) without also reexperiencing the somatosensory qualities of that pain (Katz & Melzack, 1990). Dissociation of the opposite kind is not as common and is more difficult to demonstrate since without the knowledge (i.e., contents of the cognitive memory component) of what one has felt in the past, the reactivation of the somatosensory qualities of a past pain would be perceived as novel and

therefore would not be recognized as having occurred before. Moreover, it is rare to find a situation in which (1) an amputee demonstrates amnesia or forgetting (of the contents of the cognitive memory component) and (2) an independent source had verified the nature of the pain at the time of injury before amputation.

Nevertheless, there are several lines of evidence supporting dissociation of this kind. The first comes from experiments that model the phantom limb in animals (Katz, Vaccarino, Coderre, & Melzack, 1991). Sectioning the sciatic and saphenous nerves in the rat is followed by self-mutilation (autotomy) of the denervated hindpaw. It is well established that autotomy is a response to pain or dysesthesias (painful paresthesias or tingling) referred to the anesthetic limb and represents a model of the phantom limb. A brief thermal injury of a specific region of the hindpaw just prior to nerve sections changes the usual pattern of autotomy over the following days. Animals injured before, but not after, nerve sections direct autotomy to the site of prior injury. Since the nerve sections produce a deafferentation of the entire hindpaw, the central effects of the injury are sustained in the absence of further inputs from the hindpaw, implying that painful or dysesthetic sensations are referred specifically to the region of the denervated limb that had received the injury.

The correspondence between the sites of prior injury and subsequent autotomy parallels descriptions of human amputees who report the persistence of a preamputation pain or lesion referred to the same location of the phantom limb. In these experiments (Katz et al., 1991), the injury was always induced while the rats were under a general anesthetic, and they were maintained under the general anesthetic until well after the sciatic and saphenous nerve transections had been performed. Thus, although the rats never experienced the thermal injury in an awake state, their behavior in the days after the nerve sections revealed that the effects of the injury were still capable of influencing perception and behavior (in the absence of further inputs from the injured region). These findings provide empirical support for the hypothesis that the unified experience of a pain memory involves two potentially dissociable forms of memory, one of which (the somatosensory component) is independent of the conscious experience of pain.

Lacroix, Melzack, Smith, and Mitchell (1992) recently provided compelling clinical evidence of a dissociation between the cognitive and somatosensory memory components. They report the case of a 16-year-old girl who was born with a congenital deformity of the right foot which was amputated when she was just 6 years old. At the time of the interview 10 years after amputation, the patient reported a flat phantom foot that was stuck in a forward position. This description corresponded to information subsequently obtained from her medical records verifying a right flatfoot

which was locked in an equinovalgus position and incapable of movement. Interestingly, the patient was not aware that her foot had been deformed as a child, for she mistakenly described her foot as she "remembered" it prior to amputation as being normal and freely mobile. This case report demonstrates the remarkable capacity of the central nervous system to retain, for years after amputation, a complete representation of the cut-off part, including its somatosensory qualities, proprioceptive sensibility, and associated motor program. Moreover, the case demonstrates that the neural circuitry underlying the somatosensory component is capable of being activated and of influencing conscious awareness independent of the cognitive component.

Although separate representations of the somatosensory and cognitive components are formed during repeated occurrences of the preamputation pain, such frequent and temporally contiguous activity would result in a tendency for these representations to occur more often together than alone once the limb has been removed. There is evidence that the two memory systems may be reciprocally connected so that activation of either memory component can lead to activation of the other. The presence of the somatosensory component is sufficient to activate the contents of the cognitive component as implied by the process of recognition involved when a patient identifies the somatosensory qualities of the experience as having occurred before. The possibility also exists that the link is bidirectional. One subject in the study by Katz and Melzack (1990) reported that he could reproduce at will the sensation of the "hole" from a gangrenous ulcer he had on the medial aspect of his foot prior to amputation, but if he did not concentrate on it, the somatosensory component remained out of his awareness. It is important to note, however, that activation of the representation underlying the cognitive component is not to be equated with the conscious awareness of thoughts about the past pain, but when such thoughts occur, excitation of the corresponding neural assemblies must have been involved.

Implications of Separate Memory Components

There are important implications associated with the suggestion that separate somatosensory and cognitive memory systems underlie pain that persists after amputation. For one, conscious awareness of the contents of the cognitive memory component is not necessary for the *reactivation* of the somatosensory component (although it may facilitate the process when present). Second, it is clear that the conscious experience of pain is not a necessary condition for the *formation* of the somatosensory memory component. That is, the formation of the somatosensory component can occur

even when there is no conscious awareness of pain at the time of injury or trauma (Katz et al., 1992; Katz et al., 1991) or when the cognitive component is not accessible through introspection (Lacroix et al., 1992).

These findings raise the possibility that, just as brief, intense pain experienced in a limb shortly before its amputation persists as phantom limb pain memory (Katz & Melzack, 1990), the effects of the primary afferent "injury discharge" on spinal cord dorsal horn neurons produced by surgical incision (and subsequent cutting of muscle, nerve, and bone) may also produce lasting changes that later contribute to postoperative pain. This implies that both somatosensory and cognitive systems must be blocked in order to interfere with the formation of a pain memory arising from the surgical procedure (Fig. 3).

Patients who have sustained traumatic amputation either by accident, combat-related injury, or emergency surgical procedures carried out without anesthetics or analgesics (e.g., in war-ravaged parts of the world) are at highest risk for developing postamputation problems (Fig. 3a). Traumatic amputation would be expected to result in the formation of both the somatosensory and cognitive memory components. The expected outcome would include heightened stump pain (stump hyperalgesia), heightened phantom limb pain intensity, recognition of the somatosensory qualities of the pain, and a posttraumatic stress disorder arising from the traumatic events.

Amputation performed under general anesthesia alone (Fig. 3b) would interfere with the formation of the *cognitive* but not the *somatosensory* memory component. However, unlike a pain memory that resembles a long-standing preamputation lesion, the somatosensory qualities of postsurgical pain would not be recognized by a patient whose surgery was performed under a general anesthetic, since the patient would not have had any conscious experience of pain at the time of incision and amputation. Upon awakening from the general anesthetic, the patient's complaints of pain would reflect the persistent central neural memory trace left by the surgical procedure in addition to input from transected fibers in the amputation stump (Wall, 1989). This is hypothesized to result in enhanced postoperative phantom limb pain and heightened pain at the site of the incision (incisional hyperalgesia).

Administration of spinal local anesthesia alone (Fig. 3c) would block the formation of the *somatosensory* but not the *cognitive* memory component. The preincisional spinal blockade would prevent the injury barrage from reaching the CNS, resulting in less intense postoperative phantom limb pain and incisional pain. However, in the absence of a general anesthetic, awareness during amputation can produce vivid declarative memo-

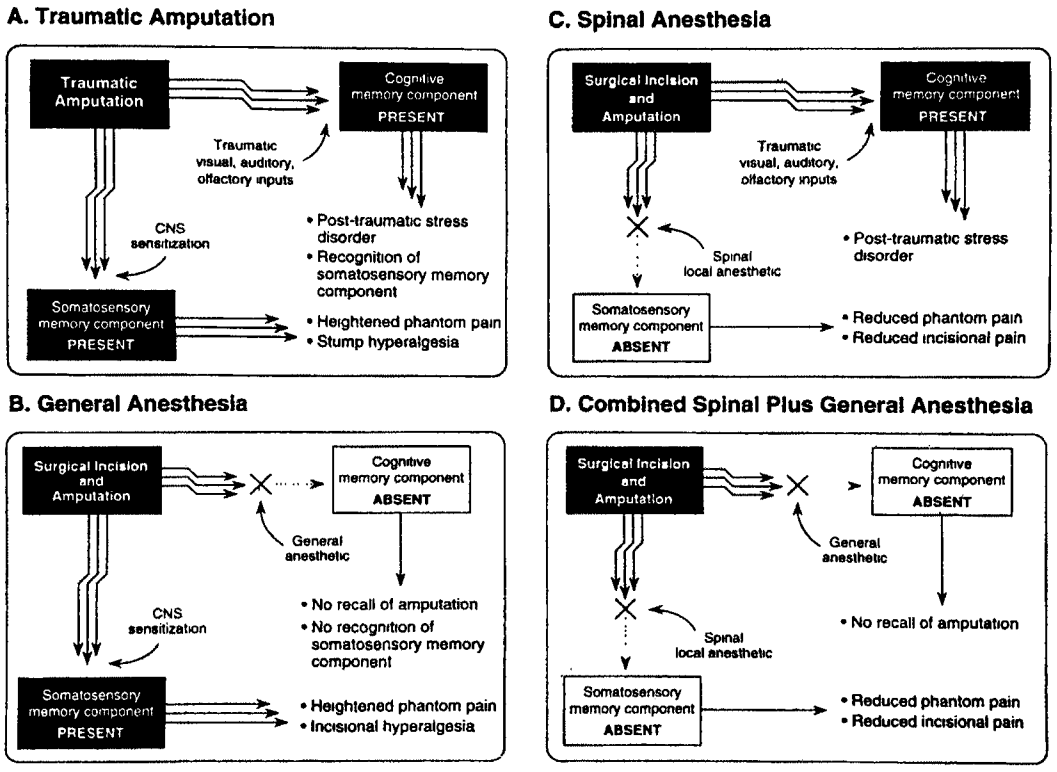

Fig. 3. Predicted postoperative pain status and psychological status following traumatic amputation or surgical amputation performed under general anesthesia, spinal local anesthesia, or combined spinal local anesthesia plus general anesthesia (see text for details). CNS = central nervous system.

ries of operating room events that develop into a posttraumatic stress disorder.

Combined use of spinal anesthesia and general anesthesia (Fig. 3d) would be expected to interfere with both *somatosensory* and *cognitive* memory systems by blocking the transmission of nociceptive impulses (arising from the cutting of tissue, nerve and bone) at the level of the spinal cord, and by ensuring that the patient is unconscious during the surgical procedure. Recent studies of patients undergoing surgery show that combined use of general anesthesia plus *preincisional* epidural administration of an opioid (Katz et al., 1992) or *preincisional* local anesthetic infiltration (Ejlersen, Bryde Anderson, Eliasen, & Mogensen, 1992) is more effective in reducing postoperative pain and/or analgesic requirements than combined use of general anesthesia plus *postincisional* (Katz et al., 1992) or *postsurgical* (Ejlersen et al., 1992) administration of the same agent by the same route. The efficacy of *preemptive analgesia* has yet to be evaluated for limb amputation.

Factors That Influence the Formation and Reactivation of Pain Memories

Amputation or Deafferentation. When a missing or completely anesthetic limb continues to be the source of pain that resembles an old injury, it is reasonable to assume that the pain is centrally represented. It is unclear, however, whether the interruption of normal sensory nerve impulses (deafferentation) or amputation is necessary for pain memories to develop. The interruption of afferent input associated with amputation or deafferentation may facilitate the central neural changes that contribute to the formation of pain memories by removing normal inhibitory control mechanisms (see Coderre, Katz, Vaccarino, & Melzack, 1993 for a review).

Alternatively, deafferentation may merely provide a condition under which persistent CNS activity becomes obvious to the observer since the peripheral source has been removed or its afferent supply interrupted. This may explain why pain memories are almost exclusively reported to occur in patients with deafferenting lesions and rarely under other circumstances. Examples of recurring pain in the absence of obvious deafferentation include cardiac pain referred to the site of a compression fracture in the upper back sustained 20 years earlier (Henry & Montuschi, 1978) and pain, in response to stimulation of the nasal mucosa, referred to teeth that had recently been filled (Hutchins & Reynolds, 1947; Reynolds & Hutchins, 1948). Noordenbos and Wall (1981) described seven patients with partial peripheral nerve injury and subsequent pain who underwent nerve resection and graft or ligation. Following a pain-free period all seven redeveloped

pain of the same quality and in the same location as they had experienced prior to nerve resection, although in some patients the recurrence of pain was restricted to a smaller area within the originally painful region.

If deafferentation or amputation is not a necessary condition for a pain memory to develop, then we must ask why they are reported so infrequently among patients in whom the flow of afferent impulses has not been interrupted. One possibility is that certain peripheral injuries do become represented centrally, but because the peripheral source of pain is so obvious (e.g., a surgical incision), the existence of a central somatosensory component is not even considered. For example, it has been shown that postoperative pain following thoracic surgery is less intense if patients received a general anesthetic plus an epidural opioid infused *before* incision vs. a general anesthetic plus an epidural opioid infused 15 min *after* incision (Katz et al., 1992). Nociceptive impulses during surgery reach the spinal cord and contribute to a state of persistent central sensitization that increases postoperative pain intensity after the patient awakens from the general anesthetic. Blocking nociceptive inputs before but not after incision attenuates the development of the central somatosensory component. The obvious source of ongoing pain after surgery (i.e., the incision) may blind the observer to the possibility that the very act of cutting may have set up a permanent representation that amplifies subsequent inputs from the wound.

The possibility that a central somatosensory component may be masked by the more obvious peripheral source of pain is further supported by studies of patients undergoing electrical brain stimulation during neurosurgical procedures. Pain is rarely elicited by test stimuli unless the patient suffers from a long-standing pain problem (Lenz, Kwan, Dostrovsky, & Tasker, 1989; Obrador & Dierssen, 1966). Electrical stimulation of a variety of subcortical structures in patients with chronic pain frequently evokes pain and in some instances may even reproduces the patient's pain. Although these studies involved patients with central or deafferentation pain, electrical brain stimulation may even elicit pain responses in patients with pain that is not long-standing and that does not involve extensive nerve injury or deafferentation. Nathan (1985) described a patient who underwent thalamic stimulation for a movement disorder. The patient had been suffering from a toothache for 10 days prior to the operation. Electrical stimulation of the thalamus reproduced the toothache.

Inputs from the Periphery. There is evidence that in some cases the reactivation of a pain memory requires a peripheral trigger. Leriche (1947) described a patient who did not experience phantom limb pain until 6 years after amputation, when an injection into the stump instantly and permanently, revived the pain of a former painful ulceration of the Achilles ten-

don. Nathan (1962, 1985) reported a similar phenomenon after applying noxious stimuli to the stump of an amputee who later reexperienced the pain of an ice-skating injury he had sustained 5 years earlier when his leg was intact. Katz and Melzack (1990) reported a patient with an amputation below the knee who discovered that when he maximally flexed his knee he could briefly elicit in the phantom limb, the sensation of "the dry, callused, tight skin" he used to feel on the sole of his foot. Another amputee who had suffered from intermittent claudication prior to amputation continued to experience the same pain referred to the phantom calf after walking a short distance. These reports indicate that past pains may be reexperienced months or even years after the original injury, in some cases by a peripheral trigger which provides the input required to activate the central neural structures subserving the memory trace.

Inputs from Modalities Other than Somesthesis. Pain memories sometimes comprise highly complex perceptual phenomena that include components from several modalities which were involved in the original experience. Many preamputation pains have corresponding visual elements such as a discolored and festering diabetic ulcer, or a raw, red open surgical wound. Some may even have associated olfactory cues including the foul stench of putrid diabetic ulcers and gangrene. These and other examples (Henderson & Smyth, 1948; Jacome, 1978; James, 1887; Wallgren, 1954) suggest that separate modality-specific sensory memories of the preamputation experience may be formed at the time of injury or during episodes of pain. The additional sensory modalities may contribute to the formation of a higher-order functional unit during the contiguous activation of modality-specific representations associated with bouts of preamputation pain (Bindra, 1978).

The role of vision is especially important since it dominates over other sensory modalities in circumstances involving exteroceptive sensibility. Lower-limb amputees frequently report that it was not until they looked under the bed sheets that they realized their limb had been cut off (Gallinek, 1939; Simmel, 1956). Patients undergoing brachial plexus or spinal blocks, those with complete brachial plexus avulsions, and spinal cord transections all report vivid phantom limbs which are felt to be coincident with the position of the real limb as determined by sight (Bors, 1951; Evans, 1962; Wynn Parry, 1980). This is demonstrated clearly when a patient's deafferented limb is moved from one position to another with his or her eyes closed. Under these circumstances, the felt position of the phantom corresponds to the last seen position of the real limb. However, when the patient's eyes are opened, the phantom is reported to "fuse" with the new position of the real limb as perceived by sight (Bromage & Melzack, 1974; Evans, 1962; Melzack & Bromage, 1973; Wallgren, 1954). It is also worth

noting that prolonged visual deprivation results in significant increases in cutaneous sensitivity among healthy volunteers who have all their limbs intact (Zubek, Flye, & Aftanas, 1964).

These findings demonstrate the powerful influence of vision in determining the phantom limb percept. When there is a discrepancy or contradiction between incoming information from different modalities, or when a state of uncertainty exists based upon somatosensory input alone, additional information is sought *via* the visual sense which usually determines the perceptual experience. Since amputation also results in the loss of visual and tactile information related to the limb, the central influences that normally inhibit established pain traces may be further reduced by the absence of information from these external sources that could otherwise confirm or disconfirm the percept (e.g., of a painful diabetic ulcer) arising from the periphery. Following amputation, the likelihood of reactivation of a pain memory that had a visual component (e.g., a diabetic ulcer) is increased since the potential inhibitory effect of vision has also been removed. In general, as the number of modalities involved in the preamputation pain experience increase (and thus the more sources of potential feedback are removed), the greater is the probability of reactivating a past pain once the limb has been removed since there are fewer senses available to provide a reality-based check (i.e., exert an inhibitory influence) on the perceptual processes generating the phantom.

Leventhal (1982) has proposed a similar conceptualization in which a *schematic-emotional mechanism* generates a concrete (nonsemantic, nonpropositional) multicomponent code of sensory and affective events. This multimodal representation is formed through integration of information from a variety of senses during repeated or multiple events that evoke similar emotional states and may be (re-) activated even in the absence of many of the stimulus configurations that were present during its formation. The present conceptualization of the somatosensory memory component differs somewhat from Leventhal's schematic-emotional mechanism. Whereas an affectively charged experience may facilitate the formation of the somatosensory memory component, the affective state that accompanies the unified experience of a pain memory after amputation is not thought to be a reactivation (i.e., a memory) but is believed to be generated on a moment-by-moment basis, determined, in part, by current sensory input and cognitive-evaluative processes (see section on cognitive and affective processes).

The Use of Language. Language may play an important role in the development and reactivation of pain memories since it appears to facilitate integration of information from various sense modalities (Bindra, 1978; Marks, 1978). In particular, the analogic aspect of the verbal message conveys meaning by likening certain qualities (e.g., sensory) of the pain expe-

rience to some other experience — whether fancied or real — and is aided by using such figures of speech as simile, metaphor and hyperbole, or more subtly through allegory. Thus consider one woman's bittersweet description of the pins-and-needles sensation so characteristic of phantoms, as "champagne bubbles and blisters" after a left shoulder amputation simultaneously marked the end of a prolonged period of suffering and the beginning of life without an arm (Janovic & Glass, 1985). Or consider the patient with diabetes mellitus who describes the burning pain of a putrefying and discolored gangrenous ulcer on his toe as "hellfire and brimstone."

The formation of a higher-order polymodal representation of the pain is facilitated by the unifying verbal response which captures the entire experience signaled by the contiguous activation of modality-specific representations arising from separate sensory channels (e.g., visual, olfactory, somatosensory). The foregoing implies that, after repeated bouts of pain, information signaling the presence of an injury in one modality would activate corresponding representations in other modalities (e.g., in the absence of input from the painful part after amputation). In this context, language functions to simultaneously access multimodal representations, strengthen their interconnections, and, through convergence of input to neocortical association areas, facilitate both the formation of a pain memory as well as its reactivation after amputation.

Psychopathology and Emotional Disturbance. It is not uncommon for proponents of theories of phantom limb pain to discount as psychological in origin pain that could not be explained on the basis of current physiological and anatomical knowledge (Bailey & Moersch, 1941; Henderson & Smyth, 1948; Lakoff, 1990). The practice of relegating certain inexplicable phenomena to the psychological or emotional realm may free the theorist from considering them further, but it changes how the amputee is viewed and treated, and implicitly blames him or her for the pain. It is crucial to differentiate legitimate attempts to explain how psychopathology influences phantom limb experience from attempts to use the label as an explanation.

It has been argued that the similarity of pain before and after amputation represents a psychopathological response to amputation in which the psychological or emotional importance of the preamputation pain determines the likelihood of its reexperience in the phantom limb. Henderson and Smyth (1948) described the case of a soldier who sprained his ankle jumping from a truck and therefore could not keep up with his companions. Shortly after he was wounded in the same leg above the ankle and was taken prisoner. The leg was amputated a few days later but he continued to experience only the pain of the ankle sprain. The soldier remarked that had it not been for the sprain, he would not have been captured. Bailey

and Moersh (1941) described a patient whose phantom included the sensation of a wood sliver that had been under the nail of his index finger at the time of amputation. They discussed the importance of "both psychical and physical trauma" at or near the time of amputation and concluded that the persistence of preamputation pain represents an "obsession neurosis."

These case reports raise the possibility that emotional and psychological disturbances contribute to pain that persists after amputation but their conclusions should be viewed as hypotheses to be tested in a prospective study of patients scheduled for amputation. In seeking rational explanations for phantom limb pain, patients, clinicians, and researchers may conclude that the significance of the preamputation pain was instrumental in its representation in the phantom limb. Furthermore, any psychological theory must take into account the literature documenting the recurrence of corns, ingrown toenails, calluses, etc., which, prior to amputation, are rarely considered psychologically important to the patient.

The only study to compare subjects reporting pain memories with those who did not have phantom limb pain or those who had phantom limb pain that bore no resemblance to their preamputation failed to find any significant inter-group differences in depression, anxiety, or personality (Katz & Melzack, 1990). Thus, at the time of interview, approximately 5 years after amputation, there was no evidence to suggest that levels of psychopathology or emotional disturbance were different for subjects who reported phantom limb pain of any type compared to pain-free subjects. However, as noted above, the relationship between emotional disturbance and psychopathology at the time of injury (or the significance of the injury) and the subsequent development of a phantom limb pain memory has yet to be addressed in a prospective study.

Pain memories also occur in certain psychiatric patients in the absence of deafferentation and without positive physical signs of peripheral injury (Bressler, Cohen, & Magnussen, 1955; Engel, 1959; Szasz, 1949). Patients presenting with this clinical picture may obtain a diagnosis of conversion hysteria or embark on a fruitless course of treatment focused at the periphery. In his seminal paper on the "pain-prone patient," Engel (1959) introduced the concept of a pain memory to explain his observation that, during emotionally stressful circumstances, certain psychiatric patients reported repeated bouts of pain similar in quality and location to a past pain. The circumstances under which the pains recurred were believed to be symbolic of the traumatic event in which the pain was first experienced. According to Engel, "the capacity to experience pain in the first place develops from numerous peripherally induced experiences but thereafter pain experience, like visual or auditory experience, may occur without the cor-

responding stimulation of the end organ . . . . The term 'pain memories' refers to the ideational complexes, conscious and unconscious, associated with past pain experiences, stimulation of which may later give rise to pain. This pain is not the 'old' pain anymore than the joy evoked by certain memories is the same joy that was felt on the occasion of the original joyous experience" (Engel, 1959, pp. 900-901). Engel was careful to leave open the possibility that not all patients suffering from the recurrence of a past pain have pain of psychological origin in the sense that warrants the psychiatric diagnosis of the "pain-prone patient." We do not know the factors responsible for the development and maintenance of pain memories in these patients nor do we know how they differ from the pain memories reported by amputees.

Cognitive and Affective Processes. A separate but related issue concerns the role played by nonpathological cognitive and affective processes in the development or subsequent expression of pain memories after amputation. Recent work in the field of mood and memory has demonstrated that material with high affective loading is learned best (Singer & Salovey, 1988). Moreover, mood state during recall may enhance retrieval of specific information that is compatible in content with that feeling state (see Isen, 1987, for a review). These findings suggest that the role of affect in pain memories might be twofold: (1) to facilitate the formation of the somatosensory and cognitive memory components, perhaps through the peripheral and central release of neuroendocrine products into the general circulation, and (2) to facilitate the reactivation of both memory components by creating a central emotional state similar in affective tone to that experienced prior to amputation, biasing attention, information processing, and memory functioning in favor of pain-related material.

For example, traumatic injuries incurred as a result of an accident or an emergency surgical procedure performed without anesthetic form the basis of highly specific and vivid declarative memories (Katz & Melzack, 1990) much like "flashbulb memories" that occur after extremely stressful events (Squire, 1987). The events surrounding these traumatic preamputation injuries may be reexperienced accompanied by high levels of anxiety. 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, posttraumatic chronic pain syndrome precipitated by the initial trauma (Engel, 1959; Muse, 1985, 1986). In these cases, the stress response associated with the initial trauma may be instrumental in the formation of a pain memory. In addition, specific cognitive or affective domains related to the traumatic event may become sensitized so that they develop the capacity to serve as central triggers for the reactivation of the pain after amputation.

Finally, the nature and origin of the emotional response that accompanies a pain memory requires comment. We have proposed that the unified experience of a pain memory involves information from separate somatosensory and cognitive memory components. However, it is clear that in many cases, pain memories, like most painful experiences, are also accompanied by an aversive emotional state and a desire to be free of the pain (Melzack & Casey, 1968; Melzack & Wall, 1988). The affective or emotional tone, unlike the somatosensory and cognitive components, does not appear to be a reactivation of a previously stored representation. It is generated on a moment-by-moment basis and is determined by the combined information present in the two memory components. Thus patients modulate their affective response as a joint function of (1) the intensity, quality, and location of the sensory-discriminative aspects of the experience and (2) a cognitive appraisal of the somatosensory component including its meaning, expectations about its duration, the patients' ability to cope, and other declarative information.

CONCLUSIONS

The material presented in this paper reveals that the phantom limb is not perceived as a static entity but as a frequently changing perceptual experience. Phantom limb phenomena range from simple, diffuse sensations of tingling to perceptually complex experiences of pains and lesions that originally were felt in the limb prior to amputation. While phantom pains and other sensations frequently are triggered by the perception of salient events, thoughts, and feelings, there is no evidence that the painful or painless phantom limb is a symptom of a psychological disorder. The sympathetic nervous system may provide an important link between higher brain centers involved in cognitive and affective processes and phantom limb sensations through its peripheral actions on primary afferents located in stump neuromas. Pharmacologic and nonpharmacologic treatments geared toward reducing sympathetic outflow may prove effective in managing phantom limb pain for some amputees. Taken together, the data are consistent with Melzack's (1989) concept of a neuromatrix which postulates (1) the various qualities of experience of the limb before and after amputation are virtually indistinguishable because they both reflect coordinated activity among the same neural networks in the brain, and (2) inputs from the periphery may trigger or modulate phantom limb sensations and pains but are not necessary for any of the qualities of experience.

REFERENCES

Abramson, A. S., & Feibel A. F. (1981). The phantom phenomenon: its use and disuse. *Bulletin of the New York Academy of Medicine, 57,* 99-112.

Allen, I. M. (1928). Unusual sensory phenomena following removal of a tumour of the sensory cortex. *Journal of Neurology and Psychopathology, 9,* 133-145.

Arena, J. G., Sherman, R. H., & Bruno, G. M. (1990). The relationship between situational stress and phantom limb pain: Cross-lagged correlational data from six month pain logs. *Journal of Psychosomatic Research, 34,* 71-77.

Bach, S., Noreng, M. F., & Tjéllden, N. U. (1988). Phantom limb pain in amputees during the first 12 months following limb amputation, after preoperative lumbar epidural blockade. *Pain, 33,* 297-301.

Bailey, A. A., & Moersch, F. P. (1941). Phantom limb. *Canadian Medical Association Journal, 45,* 37-42.

Berger, M., & Gerstenbrand, F. (1981). Phantom illusions in spinal cord lesions. In J. Siegfried & M. Zimmermann (Eds.), *Phantom and stump pain* (pp. 66-73). New York: Springer-Verlag.

Bindra, D. (1978). *A theory of intelligent behavior.* New York: Wiley.

Boas, R. A., Schug, S. A., & Acland, R. H. (1993). Perineal pain after rectal amputation: a 5-year follow-up. *Pain, 52,* 67-70.

Bors, E. (1951). Phantom limbs of patients with spinal cord injury. *Archives of Neurology and Psychiatry, 66,* 610-631.

Bradway, J. K., Malone, J. M., Racy, J., Leal, J. M., & Poole, J. (1984). Psychological adaptation to amputation: An overview. *Orthotics and Prosthetics, 38,* 46-50.

Brena, S. F., & Sammons, E. E. (1979). Phantom urinary bladder pain — Case report. *Pain, 7,* 197-201.

Bressler, B., Cohen, S. I., & Magnussen, F. (1955). Bilateral breast phantom and breast phantom pain. *Journal of Nervous and Mental Disease, 122,* 315-320.

Brodal, A. (1981). *Neurological anatomy in relation to clinical medicine* (3rd ed.). New York: Oxford University Press.

Bromage, P. R., & Melzack, R. (1974). Phantom limbs and the body schema. *Canadian Anaesthetists' Society Journal, 21,* 267-274.

Butler, D. J., Turkal, N. W., & Seidl, J. J. (1992). Amputation: Preoperative psychological preparation. *Journal of the American Board of Family Practice, 5,* 69-73.

Campbell, J. N., Meyer, R. A., Davis, K. D., & Raja, S. N. (1992). Sympathetically maintained pain: A unifying hypothesis. In W. D. Willis Jr (Ed.), *Hyperalgesia and alodynia.* New York: Raven Press.

Caplan, L. M., & Hackett, T. P. (1963). Emotional effects of lower-limb amputation in the aged. *New England Journal of Medicine, 269,* 1166-1171.

Carlen, P. L., Wall, P. D., Nadvorna, H., & Steinbach, T. (1978). Phantom limbs and related phenomena in recent traumatic amputations. *Neurology, 28,* 211-217.

Coderre, T. J., Katz, J., Vaccarino, A. L., & Melzack, R. (1993). Contribution of central neuroplasticity to pathological pain: Review of clinical and experimental evidence. *Pain, 52,* 259-285.

Conomy, J. P. (1973). Disorders of body image after spinal cord injury. *Neurology, 23,* 842-850.

Critchley, M. (1971). *The parietal lobes.* New York: Hafner.

Danke, F. (1981). Phantom sensations after amputation: The importance of localization and prognosis. In J. Siegfried & M. Zimmermann (Eds.), *Phantom and stump pain* (pp. 56-61). New York: Springer-Verlag.

Dawson, L., & Arnold, P. (1981). Persistent phantom limb pain. *Perceptual and Motor Skills, 53,* 135-138.

Delius, W., Hagbarth, K. E., Hongell, A., & Wallin, B. G. (1972). Manoeuvres affecting sympathetic outflow in human skin nerves. *Acta Physiologica Scandinavica, 84,* 177-186.

Dernham, P. (1986). Phantom limb pain. *Geriatric Nursing, 7,* 34-37.

Diamond, J. (1959). The effect of injecting acetylcholine into normal and regenerating nerves. *Journal of Physiology (London), 145,* 611-629.

Dorpat, T. L. (1971). Phantom sensations of internal organs. *Comprehensive Psychiatry, 12,* 27-35.

Doupe, J., Cullen, C. H., & Chance, G. Q. (1944). Posttraumatic pain and the causalgic syndrome. *Journal of Neurology, Neurosurgery and Psychiatry, 7,* 33-48.

Ejlersen, E., Bryde Anderson, H., Eliasen, K., & Mogensen, T. (1992). A comparison between preincisional and postincisional lidocaine infiltration and postoperative pain. *Anesthesia and Analgesia, 74,* 495-498.

Engel G. L. (1959). 'Psychogenic' pain and the pain-prone patient. *American Journal of Medicine, 26,* 899-918.

Evans, J. H. (1962). On disturbance of the body image in paraplegia. *Brain, 85,* 687-700.

Frazier, S. H., & Kolb, L. C. (1970). Psychiatric aspects of pain and the phantom limb. *Orthopedic Clinics of North America, 1,* 481-495.

Friedmann, L. W. (1978). *The psychological rehabilitation of the amputee.* Springfield: C. C. Thomas.

Gallinek, A. (1939). The phantom limb: Its origin and its relationship to the hallucinations of psychotic states. *American Journal of Psychiatry, 96,* 413-422.

Gloyne, H. F. (1954). Psychosomatic aspects of pain. *Psychoanalytic Review, 41,* 135-139.

Haber, W. B. (1956). Observations on phantom-limb phenomena. *Archives of Neurology and Psychiatry, 75,* 624-636.

Hagbarth, K. E., Hallin, R. G., Hongell, A., Torebjörk, H. E., & Wallin, B. G. (1972). General characteristics of sympathetic activity in human skin nerves. *Acta Physiologica Scandinavica, 84,* 164-176.

Henderson, W. R., & Smyth, G. E. (1948). Phantom limbs. *Journal of Neurology, Neurosurgery and Psychiatry, 2,* 88-112.

Henry, J. A., & Montuschi, E. (1978). Cardiac pain referred to site of previously experienced somatic pain. *British Medical Journal, 2,* 1605-1606.

Hutchins, H. C., & Reynolds, O. E. (1947). Experimental investigation of the referred pain of aerodontalgia. *Journal of Dental Research, 26,* 3-8.

Isen, A. M. (1987). Positive affect, cognitive processes, and social behavior. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol. 20, pp. 203-253). New York: Academic.

Jacome, D. (1978). Phantom itching relieved by scratching phantom feet. *Journal of the American Medical Association, 240,* 2432.

James, W. (1887). The consciousness of lost limbs. *Proceedings of the American Society for Psychical Research, 1,* 249-258.

Janovic, J., & Glass, J. P. (1985). Metoclopramine-induced phantom dyskinesia. *Neurology, 35,* 432-435.

Jensen, T. S., Krebs, B., Nielsen, J., & Rasmussen, P. (1985). Immediate and long-term phantom pain in amputees: incidence, clinical characteristics and relationship to preamputation pain. *Pain, 21,* 268-278.

Jensen, T. S., & Rasmussen, P. (1989). Phantom pain and related phenomena after amputation. In P. D. Wall & R. Melzack (Eds.), *Textbook of pain* (pp. 508-521). Edinburgh: Livingstone Churchill.

Kallio, K. E. (1950). Permanency of results obtained by sympathetic surgery in the treatment of phantom pain. *Acta Orthopaedica Scandinavica, 19,* 391-397.

Katz, J. (1992). Psychophysical correlates of phantom limb experience. *Journal of Neurology, Neurosurgery and Psychiatry, 55,* 811-821.

Katz, J., France, C., & Melzack, R. (1989). An association between phantom limb sensations and stump skin conductance during transcutaneous electrical nerve stimulation (TENS) applied to the contralateral leg: A case study. *Pain, 36,* 367-377.

Katz, J., Kavanagh, B. P., Sandler, A. N., Nierenberg, H., Boylan, J. F., Friedlander, M., & Shaw, B. F. (1992). Preemptive analgesia: Clinical evidence of neuroplasticity contributing to postoperative pain. *Anesthesiology, 77,* 439-446.

Katz, J., & Melzack, R. (1990). Pain "memories" in phantom limbs: Review and clinical observations. *Pain, 43,* 319-336.

Katz, J., & Melzack, R. (1991). Auricular TENS reduces phantom limb pain. *Journal of Pain and Symptom Management, 6,* 73-83.

Katz, J., Vaccarino, A. L., Coderre, T. J., & Melzack, R. (1991). Injury prior to neurectomy alters the pattern of autotomy in rats. *Anesthesiology, 75,* 876-883.

Kolb, L. C. (1954). *The painful phantom: Psychology, physiology and treatment.* Springfield: C. C. Thomas.

Krebs, B., Jensen, T. S., Krøner, K., Nielsen, J., & Jørgensen, H. S. (1985). Phantom limb phenomena in amputees 7 years after limb amputation. In H. L. Fields, R. Dubner, & F. Cervero (Eds.), *Advances in pain research and therapy* (pp. 425-429). New York: Raven Press.

Kristen, H., Lukeschitsch, G., Plattner, F., Sigmund, R., & Resch, P. (1984). Thermography as a means for quantitative assessment of stump and phantom pains. *Prosthetics and Orthotics International, 8,* 76-81.

Krøner, K., Krebs, B., Skov, J., & Jørgensen, H. S. (1989). Immediate and long-term phantom breast syndrome after mastectomy: Incidence, clinical characteristics and relationship to pre-mastectomy breast pain. *Pain, 36,* 327-334.

Lacroix, R., Melzack, R., Smith, D., & Mitchell, N. (1992). Multiple phantom limbs in a child. *Cortex, 28,* 503-507.

Lakoff, R. (1990). The psychology and psychotherapy of the chronic pain patient. In T. W. Miller (Ed.), *Chronic pain* (pp. 499-524). Madison, CT: International Universities Press.

LeDoux, J. E. (1989). Cognitive-emotional interactions in the brain. *Cognition and Emotion, 3,* 267-289.

Lenz, F. A., Kwan, H. C., Dostrovsky, J. O., & Tasker, R. R. (1989). Characteristics of the bursting pattern of action potential that occurs in the thalamus of patients with central pain. *Brain Research, 496,* 357-360.

Leriche, R. (1947). De la douleur comme objet de connaissance. *Le Progrès Médical, 5,* 115-129.

Leventhal, H. (1982). The integration of emotion and cognition: a view from the perceptual-motor theory of emotion. In M. Clark & S. Fiske (Eds.), *Affect and cognition: The 17th annual Carnegie Symposium on Cognition.* Hillsdale, NJ: Erlbaum.

Lindesay, J. (1985). Multiple pain complaints in amputees. *Journal of the Royal Society of Medicine, 78,* 452-455.

Livingston, W. K. (1943). *Pain mechanisms.* New York: Macmillan.

Lundberg, S. G., & Guggenheim, F. G. (1986). Sequelae of limb amputation. *Advances in Psychosomatic Medicine, 15,* 199-210.

Marbach, J. J. (1978). Phantom tooth pain. *Journal of Endodontics, 4,* 362-372.

Marks, L. E. (1978). *The unity of the senses: Interrelations among the modalities.* New York: Academic Books.

Marsland, A. R., Weekes, J. W. N., Atkinson, R., & Leong, M. G. (1982). Phantom limb pain: A case for beta blockers? *Pain, 12,* 295-297.

McGrath, P. A., & Hillier, L. M. (1992). Phantom limb sensations in adolescents: A case study to illustrate the utility of sensation and pain logs in pediatric clinical practice. *Journal of Pain and Symptom Management, 7,* 46-53.

Melzack, R. (1971). Phantom limb pain: Implications for treatment of pathologic pain. *Anesthesiology, 35,* 409-419.

Melzack, R. (1989). Phantom limbs, the self, and the brain (The D.O. Hebb Memorial Lecture). *Canadian Psychology, 30,* 1-16.

Melzack, R., & Bromage, P. R. (1973). Experimental phantom limbs. *Experimental Neurology, 39,* 261-269.

Melzack, R., & Casey, K. L. (1968). Sensory, motivational and central control determinants of pain. In D. Kenshalo (Ed.), *The skin senses* (pp. 423-439). Springfield: C. C. Thomas.

Melzack, R., & Loeser, J. D. (1978). Phantom body pain in paraplegics: Evidence for a central 'pattern generating mechanism' for pain. *Pain, 4,* 195-210.

Melzack, R., & Wall, P. D. (1988). *The challenge of pain* (2nd ed.). New York: Basic Books.

Merskey, H. (1989). Psychiatry and chronic pain. *Canadian Journal of Psychiatry, 34,* 329-336.
Minski, L. (1943). Psychological reactions to injury. In W. B. Doherty & D. D. Runes (Eds.), *Rehabilitation of the war injured* (pp. 115-122). New York: Philosophical Library.
Mitchell, S. W. (1871). Phantom limbs. *Lippincott's Magazine of Popular Literature and Science, 8,* 563-569.
Morgenstern, F. S. (1964). The effects of sensory input and concentration on postamputation phantom limb pain. *Journal of Neurology, Neurosurgery and Psychiatry, 27,* 58-65.
Morgenstern, F. S. (1970). Chronic pain: A study of some general features which play a role in maintaining a state of chronic pain after amputation. In O. W. Hill (Ed.), *Modern trends in psychosomatic medicine* (pp. 225-245). London: Butterworth.
Muse, M. (1985). Stress-related, posttraumatic chronic pain syndrome: Criteria for diagnosis, preliminary report on prevalence. *Pain, 23,* 295-300.
Muse, M. (1986). Stress-related posttraumatic chronic pain syndrome: Behavioral treatment approach. *Pain, 25,* 389-394.
Nathan, P. W. (1962). Pain traces left in the central nervous system. In C. A. Keele & R. Smith (Eds.), *The assessment of pain in man and animals* (pp. 129-134). Edinburgh: Livingstone.
Nathan, P. W. (1985). Pain and nociception in the clinical context. *Philosophical Transactions of the Royal Society of London, 308,* 219-226.
Noordenbos, W. (1959). *Pain.* Amsterdam: Elsevier.
Noordenbos, W., & Wall, P. D. (1981). Implications of the failure of nerve resection and graft to cure chronic pain produced by nerve lesions. *Journal of Neurology, Neurosurgery and Psychiatry, 44,* 1068-1073.
Nyström, B., & Hagbarth, K. E. (1981). Microelectrode recordings from transected nerves in amputees with phantom limb pain. *Neuroscience Letters, 27,* 211-216.
Obrador, S., & Dierssen, G. (1966). Sensory responses to subcortical stimulation and management of pain disorders by stereotaxic methods. *Confinia Neurologica, 27,* 45-51.
Oveson, P., Krøner, K., Ørnsholt, J., & Bach, K. (1991). Phantom-related phenomena after rectal amputation: prevalence and clinical characteristics. *Pain, 44,* 289-291.
Parkes, C. M. (1973). Factors determining the persistence of phantom pain in the amputee. *Journal of Psychosomatic Research, 17,* 97-108.
Parkes, C. M., & Napier, M. M. (1975). Psychiatric sequelae of amputation [Special publication]. *British Journal of Psychiatry, 9,* 440-446.
Pilowsky, I., & Kaufman, A. (1965). An experimental study of atypical phantom pain. *British Journal of Psychiatry, 111,* 1185-1187.
Price, D. B. (1976). Phantom limb phenomenon in patients with leprosy. *Journal of Nervous and Mental Disease, 163,* 108-116.
Reisner, H. (1981). Phantom sensations (phantom arm) in plexus paralysis. In J. Siegfried & M. Zimmermann (Eds.), *Phantom and stump pain* (pp. 62-65). New York: Springer-Verlag.
Reynolds, O. E., & Hutchins, H. C. (1948). Reduction of central hyper-irritability following block anesthesia of peripheral nerve. *American Journal of Physiology, 152,* 658-662.
Riddoch, G. (1941). Phantom limbs and body shape. *Brain, 64,* 197-222.
Rosen, V. H. (1950). The role of denial in acute postoperative affective reactions following removal of body parts. *Psychosomatic Medicine, 12,* 356-361.
Schilder, P. (1950). *The image and appearance of the human body: Studies in the constructive energies of the psyche.* New York: International Universities Press.
Sherman, R. A. (1976). Case reports of treatment of phantom limb pain with a combination of electromyographic biofeedback and verbal relaxation techniques. *Biofeedback and Self Regulation, 1,* 353.
Sherman, R. A. (1984). Direct evidence of a link between burning phantom pain and stump blood circulation: a case report. *Orthopedics, 7,* 1319-1320.
Sherman, R. A. (1989). Stump and phantom limb pain. *Neurologic Clinics, 7,* 249-264.
Sherman, R. A., & Bruno, G. M. (1987). Concurrent variation of burning phantom limb and stump pain with near surface blood flow in the stump. *Orthopedics, 10,* 1395-1402.

Sherman, R. A., Gall, N., & Gormly, J. (1979). Treatment of phantom limb pain with muscular relaxation training to disrupt the pain-anxiety-tension cycle. *Pain, 6,* 47-55.
Sherman, R. A., Sherman, C. J., & Bruno, G. M. (1987). Psychological factors influencing chronic phantom limb pain: An analysis of the literature. *Pain, 28,* 285-295.
Sherman, R. A., Sherman, C. J., & Parker, L. (1984). Chronic phantom and stump pain among American veterans: Results of a study. *Pain, 18,* 83-95.
Shukla, G. D., Sahu, S. C., Tripathi, R. P., & Gupta, D. K. (1982). A psychiatric study of amputees. *British Journal of Psychiatry, 141,* 50-53.
Sicuteri, F., Nicolodi M., Fusco, B. M., & Orlando, S. (1991). Idiopathic headache as a possible risk factor for phantom tooth pain. *Headache, 31,* 577-581.
Simmel, M. L. (1956). On phantom limbs. *Archives of Neurology and Psychiatry, 75,* 637-647.
Simmel, M. L. (1959). Phantoms, phantom pain and 'denial.' *American Journal of Psychotherapy, 13,* 603-613.
Singer, J. A., & Salovey, P. (1988). Mood and memory: Evaluating the network theory of affect. *Clinical Psychology Review, 8,* 211-251.
Sliosberg, A. (1948). *Les Algies des Amputés.* Paris: Masson.
Smith, O. A., & DeVito, J. L. (1984). Central neural integration for the control of autonomic responses associated with emotion. *Annual Review of Neuroscience, 7,* 43-65.
Solomon, G. F., & Schmidt, K. M. (1978). A burning issue: Phantom limb pain and psychological preparation of the patient for amputation. *Archives of Surgery, 113,* 185-186.
Squire, L. R. (1987). *Memory and brain.* New York: Oxford University Press.
Steigerwald, F., Brass, J., & Krainick, J. U. (1981). The analysis of personality factors in the prediction of phantom limb pain. In J. Siegfried & M. Zimmermann (Eds.), *Phantom and stump pain* (pp. 84-88). New York: Springer-Verlag.
Stengel, E. (1965). Pain and the psychiatrist. *British Journal of Psychiatry, 111,* 795-802.
Szasz, T. S. (1949). Psychiatric aspects of vagotomy: IV. Phantom ulcer pain. *Archives of Neurology and Psychiatry, 62,* 728-733.
Szasz, T. S. (1975). *Pain and Pleasure* (2nd ed.). New York: Basic Books.
Tasker, R. R., Organ, L. W., & Hawrylyshyn, P. A. (1982). *The thalamus and midbrain of man.* Springfield: C. C. Thomas.
Turgay, A., & Sonuvar, B. (1983). Emotional aspects of arm or leg amputation in children. *Canadian Journal of Psychiatry, 28,* 294-297.
Van Bogaert, L. (1934). Sur la pathologie de l'image de soi (Études anatomo-cliniques). *Annales Médico Psychologiques (Paris), 14,* 519-555, 714-759.
Wall, P. D. (1981). On the origin of pain associated with amputation. In J. Siegfried & M. Zimmermann (Eds.), *Phantom and stump pain* (pp. 2-14). New York: Springer-Verlag.
Wall, P. D. (1989). To what would Gaston Labat be attending today? *Regional Anesthesia, 14,* 261-264.
Wall, P. D., & Gutnick, M. (1974). Properties of afferent nerve impulses originating from a neuroma. *Nature, 248,* 740-743.
Wallgren, G. R. (1954). Phantom experience at spinal anaesthesia. *Annales Chirurgicae et Gynaecologicae Fenniae, 43 (Suppl.),* 486-500.
Weinstein, S. (1962). Phantoms in paraplegia. In: *Proceedings of Eleventh Annual Clinical Spinal Cord Injury Conference, 11,* 138-152.
Weiss, S. A. (1958). The body image as related to phantom sensations: A hypothetical conceptualization of seemingly isolated findings. *Annals of the New York Academy of Science, 74,* 25-29.
Wynn Parry, C. B. (1980). Pain in avulsion lesions of the brachial plexus. *Pain, 9,* 40-53.
Zubek, J. P., Flye, J., & Aftanas, M. (1964). Cutaneous sensitivity after prolonged visual deprivation. *Science, 144,* 1591-1593.