

Psychophysiological Contributions to Phantom Limbs*

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Recent studies of amputees reveal a remarkable diversity in the qualities of experiences that define the phantom limb, whether painless or painful. This paper selectively reviews evidence of peripheral, central and psychological processes that trigger or modulate a variety of phantom limb experiences. The data show that pain experienced prior to amputation may persist in the form of a somatosensory memory in the phantom limb. It is suggested that the length and size of the phantom limb may be a perceptual marker of the extent to which sensory input from the amputation stump have re-occupied deprived cortical regions originally subserving the amputated limb. A peripheral mechanism involving a sympathetic-efferent somatic-afferent cycle is presented to explain fluctuations in the intensity of paresthesias referred to the phantom limb. While phantom pain and other sensations are frequently triggered by thoughts and feelings, there is no evidence that the painful or painless phantom limb is a symptom of a psychological disorder. 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 emotional processes.

There is something almost tragical [sic], something ghastly, in the notion of these thousands of spirit limbs haunting as many good soldiers, and every now and then tormenting them...when...the keen sense of the limb's presence betrays the man into some effort, the failure of which of a sudden reminds him of his loss (1).

Many patients awake from the anaesthetic after an amputation feeling certain that the operation has not been performed. They feel the lost limb so vividly that only when they reach out to touch it or peer under the bed sheets to see

it do they realize it has been cut off. This startling realization does nothing to subdue the reality of the limb they experience, and may even intensify the sensations. Weir Mitchell (1) coined the term "phantom limb" to describe the phenomenon and provided the first detailed study.

A distinction is usually made between painful and non painful phantom limbs (2). The most salient property of the non painful phantom is its tingling or "pins and needles" feeling, but other qualities of sensation include temperature, posture, length, volume and movement (3). A recent study estimated the incidence of non painful phantom limbs to be between 80% and 100% (3). For many amputees, however, a distressing problem is phantom limb pain (4). The pain may be an intensification of the paresthesias that define the non painful 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 causing excruciating pain. Many amputees describe the pain in the phantom limb as indistinguishable from the pain they experienced in the limb prior to amputation. For others, the phantom is the seat of an intense burning pain as if it were being held too close to an open flame. Amputees frequently suffer from several types of pain (3).

A recent survey based on several thousand amputees revealed that more than 70% continued to experience phantom limb pain of considerable intensity more than 25 years after the amputation (5). Equally striking is the low success rate of treatments for phantom limb pain: in the long term only seven percent of patients are helped by the more than 50 types of therapy used to treat phantom limb pain (4). This intractability reflects our ignorance about the mechanisms that contribute to phantom limb pain.

A controversy has arisen over the origin of phantom limb phenomena. In an attempt to find a single explanatory mechanism, theories have focused on only one aspect of phantom limbs and have ignored or discounted the others (2). The cause has been sought in the activity of primary afferent fibers, spinal cord cells and supra-spinal sensory nuclei (3,6). Another class of theory has attempted to explain the phantom limb solely on the basis of psychological and emotional processes (7). Melzack (6) recently concluded that it cannot be explained by a single mechanism. 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 and psychological factors that contribute to the development of a phantom limb.

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Pain Memories in Phantom Limbs

Recent research findings, both clinical and basic, show that the central nervous system is capable of functional or structural change in response to noxious somatosensory inputs. The data strongly suggest that these long term, injury-induced "plastic" changes contribute to the experience of pain long after the offending stimulus has been removed or the injury has healed (8-11).

Evidence also suggests that pain experienced prior to amputation may influence the course of phantom limb pain many months later. Pre-amputation pain of more than one month's duration is predictive of phantom limb pain as long as six months after the amputation, as is pain in the limb on the day before the amputation (12). Relief from pain by a continuous epidural block for three days prior to amputation decreases the incidence of phantom limb pain six months later (13).

Perhaps the most striking clinical evidence of injury-induced neuroplasticity comes from studies of amputees who report phantom limb pain resembling pain experienced in the limb before amputation (8). Amputees may experience the sensation of a painful ingrown toenail digging into the phantom toe, or the steady, gnawing pain of a diabetic foot ulcer that was present at the time of amputation. These "somatosensory pain memories" are described as having the same qualities of sensation as the pre-amputation pain and are experienced in the same location. The patients insist that they are suffering real pain and that the experience is not merely a cognitive recollection of earlier pain.

Between ten percent and 79% of amputees report experiencing pain after amputation that is similar to the pain they experienced before amputation (8). Among those reported are cutaneous lesions such as painful diabetic and decubitus ulcers, blisters, gangrene, corns, ingrown toe-nails, and cuts or gashes. Phantom limb pains may resemble pain resulting from deep tissue injuries or damage to bony structures that were present prior to amputation. The phantom limb may assume the same painful posture as the real limb prior to amputation, especially if the arm or leg had been immobilized for a long period.

For some amputees, pain memories consist of perceptually integrated experiences that incorporate visual, tactile or motor components of the original pain (8). These include graphic, affect-laden descriptions of pains being inflicted on the patient, sensations of blood-filled boots and of blood trickling down the phantom limb. Although most somatosensory memories correspond to painful pre-amputation lesions, others are innocuous and appear to represent common, everyday sensory experiences such as the awareness of a ring, a wristwatch, or bandages that once wrapped a wound.

Persistent pain memories are not limited to amputees (8). Patients with temporary or permanent forms of deafferentation, which do not involve amputation, sometimes report pains that are similar to those originally experienced prior to the interruption of afferent impulses. Phantom limbs associated with brachial plexus avulsions, spinal cord injuries, or

spinal anesthesia, may assume the same painful posture of the real limb at the time of the accident or anaesthetic block.

Painful and non painful sensations after the removal or deafferentation of body structures other than the limbs may also occur (8). Ulcer pain may persist after a vagotomy or subtotal gastrectomy. Some patients report the sensation of a full bladder and the feeling that they are urinating even though the bladder has been completely removed. Surgical excision of the rectum does not preclude vivid sensations of passing gas and feces.

The precise details of the experiences of pain described by the patients involve localization, discrimination, affect and evaluation — that is, all the dimensions of perceptual experience. These properties are a function of integrated brain activity. It is likely that the output of sensitized spinal cells activate the neural structures in the brain that subservise memories of earlier events. The data suggest that somatosensory input of sufficient intensity and duration can produce lasting changes in central neural structures subserving the sensory-discriminative dimension of pain. These are combined with cognitive-evaluative memories of the pre-amputation pain to give rise to the unified experience of past pain in the phantom limb. The separate somatosensory and cognitive memory components, which appear to underlie the unified experience of a pain memory, are consistent with recent evidence of multiple, dissociable memory systems that specialize in processing specific kinds of information (14).

Prospective studies are needed to assess the contribution of the type, location, duration, and intensity of pre-amputation pain to post-operative phantom limb and stump pain, as well as the temporal relation between pain and amputation and the role of deafferentation. In particular, it is unclear whether or not deafferentation is necessary for the formation and/or re-activation of somatosensory memories. Deafferentation may simply provide a condition in which the central source of the pain becomes obvious to the observer since the peripheral source is no longer present. Alternatively, the interruption of afferent input may facilitate the central neural changes that contribute to somatosensory memories by removing normal inhibitory control mechanisms. Since amputation also results in the loss of visual and haptic information from the limb, the central inhibition governing established pain "traces" may be further reduced by the absence of all sources of external information that might otherwise confirm or disconfirm the percept (for example, of a painful diabetic ulcer) arising from somatosensory channels alone.

Clinical Implications

The central changes in neural functioning, which are induced by injury prior to amputation, alter subsequent perception and set the stage for pathological pain. Noxious input may be perceived as more painful (hyperalgesia) than it would otherwise have been, and innocuous input may give rise to frank pain (allodynia) (10,11). Pain should be alleviated prior to amputation in order to prevent pathological pain from developing after the amputation (13).

The observation that brief, intense pain experienced shortly before amputation may contribute to pain after amputation (8) is consistent with the results of animal studies showing that brief noxious stimuli sensitize the central nervous system to subsequent input (15). The injury barrage produced by surgical incision and other noxious events (for example, cutting nerves and bone) may also lead to relatively long-lasting changes that later contribute to post-operative pain (8-11,16). The suggestion that separate memory systems are involved in the neural representation of pain memories implies that both somatosensory and cognitive systems must be blocked in order to interfere with the formation of a pain memory (8).

Amputation performed under general anesthesia alone interferes with the formation of the *cognitive* but not the *somatosensory* memory component, the development of which appears to be independent of the conscious awareness of pain (17). It is hypothesized that this results in greater post-operative phantom limb and stump pain due to the sensitization of cells in the dorsal horn and more rostral sensory structures. Alternatively, spinal local anesthetic administered prior to surgery blocks the formation of the *somatosensory* but not the *cognitive* memory component. This procedure would produce declarative memories (often traumatic) of the amputation procedure, but reduce post-operative phantom limb and stump pain due to the spinal blockade at the time of incision and subsequent amputation. The combined use of pre-operative spinal analgesia and general anesthesia during amputation would be expected to interfere with *both* the somatosensory and cognitive memory systems by blocking the transmission of nociceptive impulses (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 (18,19) have shown that analgesic agents administered prior to incision reduce both post-operative pain and analgesic requirements among patients undergoing a variety of surgical procedures. However, the efficacy of *pre-emptive analgesia* has yet to be evaluated for limb amputations.

Somatosensory Reorganization

Telescoping of the Phantom Limb

Immediately after amputation, the phantom limb usually feels perfectly normal in all respects, but with time the proximal portions of the limb begin to fade and soon disappear (Figure 1). In the case of an amputation performed at the shoulder, the phantom limb may consist of only the lower arm and hand, with a gap between the shoulder stump and the phantom elbow. In approximately one-third of amputees, a process known as "telescoping" begins (20,21). The phantom is felt to gradually approach the stump so that the hand is located in phenomenal space on a level with the elbow of the other arm (Figure 1). As this process continues, the amputee may find that the hand is protruding from the end of the stump (22). Later, the phantom hand may retract into the stump so that only the tips of the fingers jut out. In some cases, these

may eventually disappear completely and permanently into the stump (23).

When Guéniot (20) first introduced the phenomenon of telescoping, he also reported that it might be accompanied by a "shrinking" of the phantom limb to the dimensions of a child's limb. This occurs gradually in both upper and lower limbs (23-25), although the amount of shrinking is variable. Adult amputees have likened the size of their phantom hand or foot to that of a baby's (26), a silver dollar (27) and even a postage stamp (28).

Cortical Maps and Phantom Limbs

The neurophysiological correlates of telescoping and shrinking are not known. However, certain features of somatosensory cortical maps derived from microelectrode penetration studies with monkeys after amputation are consistent with certain behavioural and perceptual phenomena involving phantom limbs reported by human amputees. For example, it has been hypothesized that the perceptual changes in the length and size of the phantom limb (see Figure 1) parallel a process of cortical reorganization that has been documented in area 3b of somatosensory cortex after monkeys had undergone a digit amputation (29). The perceived length and size of the phantom limb may thus be a *perceptual marker* of the extent to which cutaneous input from the stump and surrounding tissue has "taken over" cortical regions originally driven by input from the amputated limb (29,30).

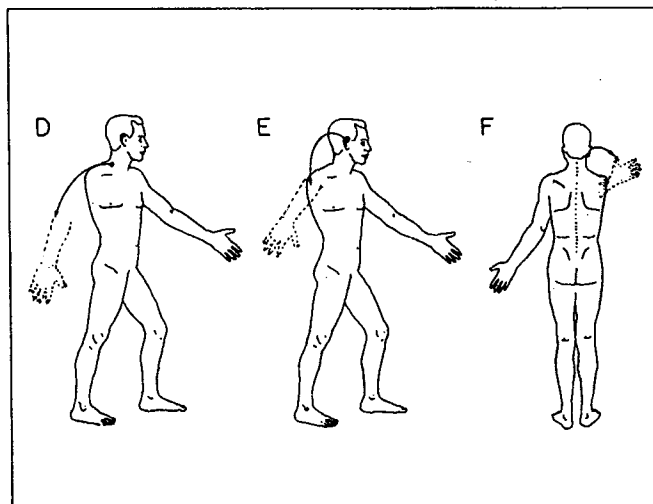


Figure 1. Patient who sustained a complete fore-quarter amputation of his right shoulder in a work accident and later developed phantom limb pain. The figurines depict referred sensations reported by the patient during transcutaneous electrical nerve stimulation. Dots represent points where stimulation was applied; arrows indicate where the sensations were referred. D: The patient reported a phantom limb of normal length (lower arm and hand only), with a gap between the shoulder stump and phantom elbow. During stimulation above the clavicle, the patient's phantom hand began to swell and become warm. E: During stimulation to the right ear, the phantom arm telescoped into the stump so that the elbow protruded from the stump. F: Later stimulation of the same point resulted in a further retraction of the phantom, leaving only the wrist and hand attached to the stump. (Reproduced from Katz and Melzack (22) with permission).

Cells identified by penetrations within the deprived region of sensory cortex, which originally had receptive fields on the amputated digits, respond after amputation to new input on the adjacent digits, palmar pads and stump. Reoccupation involves an orderly topographic expansion of adjacent parts of the hand into the deprived region and appears to progress in a proximo-distal direction. This process parallels the perceptual experience of telescoping. Thus, the perceived distance between the phantom and the stump may be a function of the distance separating their respective representations in the cortical map (29).

Skin surfaces that expand into the region of cortex corresponding to the amputated digits have receptive fields that are considerably smaller than those in normal regions of the cortex. Skin surfaces located farther from the site of amputation are represented near the boundary of the reorganized zone and have relatively larger receptive fields (29). These observations are consistent with studies that have compared the sensory acuity of the stump and the contralateral intact limb of human amputees. Lowered thresholds at the stump are observed for light touch, two-point discrimination and point localization after amputation of the upper extremity (31) and for two-point discrimination after amputation of the lower extremity (30,32). Similar results have been obtained for pressure sensitivity and two-point discrimination thresholds among children with a congenital absence of a limb (33,34). Sensory thresholds at the distal end of the stump are significantly lower than those at more proximal regions of the stump but homologous sites on the contralateral intact limb do not differ from each other (31). These observations together suggest that the sensory acuity of the stump is enhanced as it progressively occupies more cortical territory originally subserving parts of the amputated or congenitally absent limb (29).

Territorial reoccupation is not always complete (29). Electrically "silent" zones, or islands, are found within the reorganized region of cortex that remain unresponsive to cutaneous stimulation two months after amputation. Six months later, there is a noticeable shrinking in the size of the silent zone so that the outer rim becomes responsive to new input from adjacent digits. This finding parallels reports that over a period of several months after amputation, the phantom hand or foot is perceived to shrink in size as it approaches the stump (20,23-28).

Evidence of a link between telescoping and cortical reorganization is suggested by the differential sensory acuity of stumps of amputees with "telescoped" and "extended" (i.e., normal length) phantom limbs (31). Point localization is significantly enhanced in above-elbow amputees whose phantom limbs are perceived to be inside the stump as compared with those who report phantom limbs of normal length. Light touch and two-point discrimination thresholds also show greater stump sensitivity among amputees with telescoped phantom limbs. These findings suggest that as a result of the central readjustment of cutaneous input after amputation, the distal region of the stump takes over the tactile and sensory functions of the amputated hand.

Referred Phantom Limb Sensations

The observation that cells in the re-occupied cortex respond to new input from skin surfaces on the stump, palmar pads and adjacent digits (29) raises the question of where (to which body part) a stimulus would be localized when applied to a novel receptive field. Reports of sensations referred to the phantom limb upon stimulation of the stump further support the hypothesis that central reorganization after amputation involves an expansion of stump input into brain regions originally representing the amputated limb. For example, Pitres (35) noted that when the stump is pricked or scratched lightly, a sensation with the appropriate quality of experience is felt at precise points of the phantom extremity. James (36) remarked how a breeze directed at the stump is felt as a breeze on the phantom foot. One of Haber's (37) subjects with a telescoped phantom limb remarked "It feels just like you are touching my hand." Similarly, Morgenstern (28) described a subject with bilateral lower extremity amputations and phantom limbs of shorter than normal length who reported that stimulation of the stump gave rise to dual percepts that "are very hard to describe, as there are separate sensations in the stump and in the foot which appear to come from the same point."

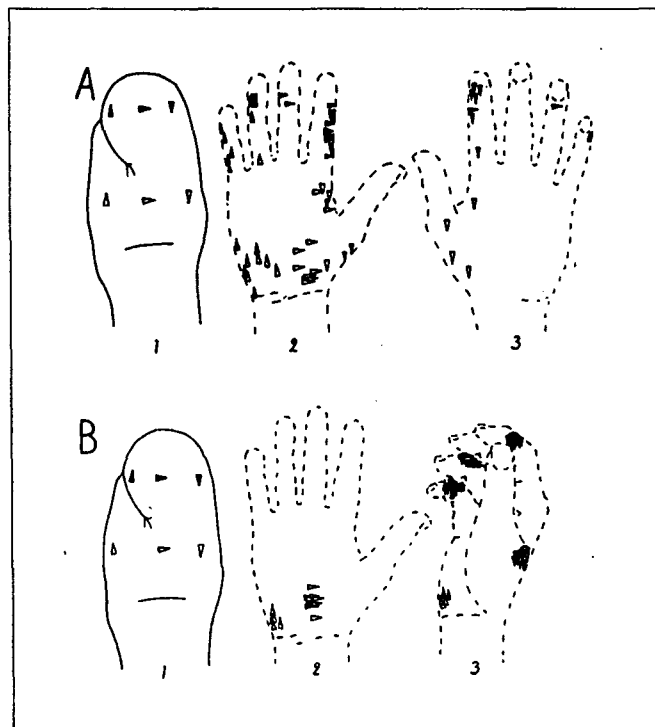


Figure 2. Pattern of referral sites on the phantom hand elicited by stimulation of the volar surface of the stump of a patient with a right below-elbow amputation. The phantom was "telescoped" so that the wrist was inside the stump with the hand protruding from it. Points on the stump (1), represented by the six symbols, were stimulated ten times each using light to firm pressure on two occasions (A and B) one month apart. The patient specified on a chart the location of the sensations referred to the phantom hand (2 and 3). (Reproduced from Cronholm (24) with permission).

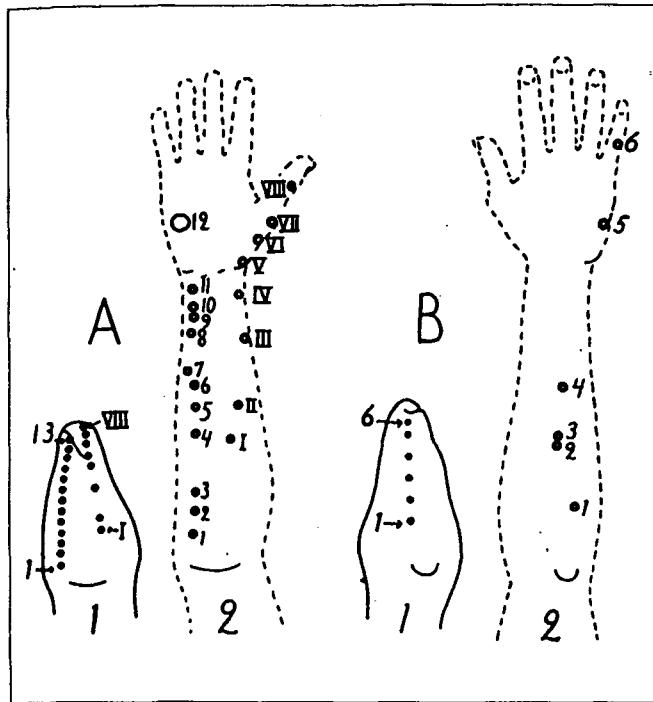


Figure 3. Pattern of referred sensations in the phantom limb in response to stimulation of points on the volar (A) and dorsal (B) aspects of the stump. There was no phantom limb when the stump was fully relaxed but when the patient tightened the muscles of the stump, he reported a completely telescoped phantom which was smaller than normal and localized entirely within the stump. For illustrative purposes, the phantom is drawn to scale in relation to the stump. Sensations were referred to specific points on the phantom limb (open circles in 2) in response to deep pressure applied at the stump (closed circles in 1) only when the stump muscles were tensed. Note the correspondence between sites of stimulation and referred sensation. In response to stimulation applied at point 13 on the stump, the subject reported feeling a sensation "in space," distal to the tips of the phantom fingers (Reproduced from Cronholm (24) with permission).

Cronholm (24) systematically studied the pattern of referred phantom limb sensations elicited by applying a variety of stimuli to the stump. Figures 2 and 3 show the results of these experiments with two patients with amputations below the elbow and telescoped phantom limbs. A light touch on the stump produced a distinct sensation that specific points on the phantom hand had been touched. Stimulation with objects of different temperatures elicited appropriate sensations of heat or cold in the phantom limb. The data reveal a remarkable isomorphism and consistency between the site and quality of the stimulus applied to the stump and the referred sensations experienced in the phantom limb.

Extensive Use of the Stump Alters the Form of the Phantom Limb

The plasticity of cortical reorganization after digit amputation in monkeys appears to be dependent on the use of the digit (29). The changes in somatosensory cortex of intact adult monkeys (i.e., without peripheral nerve injury) exposed to prolonged non noxious stimulation of the finger are very

similar to those experienced after the amputation of a digit (38) and cannot be explained exclusively by the pattern of peripheral nerve regeneration (39). These use-dependent changes in the monkeys' somatosensory cortex parallel findings that the length and shape of the phantom limb change dramatically after extensive use of the stump (40).

There is evidence that telescoping is also use-dependent. Kallio (40) surgically cleaved below-elbow stumps so that the two branches could function as a "forcepslike gripping organ" without the need of a prosthesis. After extensive post-operative training, the branches of the stump were capable of pronation, supination, grasping, and of opening and closing at a rate which approximated that of normal fingers. Two to six years later, 36% of the patients reported a cleft phantom hand in which certain fingers had fused or others had disappeared to accommodate the shape of the stump. The phantom hand had shortened so that it coincided with the tips of the branches or was completely within the stump. Opening and closing the branches of the stump were accompanied by corresponding movements in the phantom hand.

Further evidence comes from observations that phantoms associated with complete spinal cord transection (41,42) or brachial plexus avulsion (in which all sensory roots have been torn from the cord) do not shorten (43,44). There is a fundamental difference between the deafferentation produced by amputation and that resulting from a complete spinal cord transection or root avulsion. Sensory loss after surgical amputation is always distal to the dorsal root ganglion. Peripheral fibers in the stump regenerate and continue to transmit information to the spinal cord and brain. In contrast, in cases of complete spinal cord transection or root avulsion, the interruption is proximal to the dorsal root ganglion resulting in complete anesthesia caudal (or distal) to the injury. Although subsequent amputation results in the regeneration of peripheral fibers, nerve impulses are not transmitted beyond the level of the prior lesion.

The bulk of evidence indicates that telescoping does not occur when somatosensory input is prevented from reaching the central nervous system. Patients with spinal cord injuries who subsequently undergo amputations report telescoping only if the lesion is incomplete (41,42). If all pathways to the brain are severed, the phantom from a subsequent amputation does not become telescoped (41,42,45,46). Similarly, in patients with brachial plexus injuries, avulsion of all roots does not result in telescoping even after amputation of the involved extremity (43). The shortening experienced by some patients (3,47,48) with brachial plexus injuries (whose real arms are still present) may be explained by intact primary afferents or input from regenerating peripheral fibers that ruptured distal to the dorsal root ganglion.

Phantom Limb Pain and Telescoping

A remarkably consistent observation is that complete telescoping does not occur if the patient suffers from chronic phantom limb pain. It is generally held that: 1. shortening or telescoping occurs if the phantom limb is painless (3); 2. pain prevents or retards telescoping (23,49); 3. during bouts of

pain a normally telescoped or shortened phantom limb will temporarily become longer until the pain has subsided (3,21,49); and 4. relief from phantom limb pain is frequently accompanied by a rapid telescoping of the previously painful phantom (22,50,51).

These observations imply that qualitatively different processes of somatosensory reorganization are associated with painful and non painful phantom limbs. The nature of the central neuroplastic changes may be correlated not only with the presence of pain, but also with other perceptual phenomena such as the length and size of the phantom limb. For example, in the absence of pain, profound perceptual changes in the length and form of the phantom accompany extensive post-operative use of the stump (40). With practice and time, there appears to be an adaptive restructuring of the phantom as it conforms to the combined visual and kinesthetic percept of the stump. This clearly is not the case when chronic pain is present, since the painful phantom limb not only retains its original length and size but also may be a repository of past pain and lesions (8).

Clinical Implications

The foregoing discussion presents a strong case for peripheral input from the stump as a major source of the perceptual changes that occur in the length and size of the phantom limb after amputation. Coupled with Haber's (31) finding that stumps with telescoped phantom limbs have greater cutaneous sensitivity than those with extended phantom limbs, it is reasonable to suppose that extensive sensory retraining of the stump and surrounding tissues after amputation would facilitate the related processes of cortical reorganization and telescoping. Given the tendency for painful phantom limbs to be of normal length and telescoped phantom limbs to be painless, it may be possible to both facilitate telescoping and to reduce pain by implementing sensory retraining geared toward enhancing afferent input from the stump.

Contributions of the Sympathetic Nervous System

Phantom Limb Pain

It has been hypothesized that sympathetically maintained pain results from a cycle of sympathetic-efferent somatic-afferent activity involving neural (52,53) and vascular (54) changes. Pain has been attributed to sympathetically triggered ephaptic transmission (55), sympathetic activation of sensitized nociceptors (56) or low threshold mechanoreceptors that terminate on sensitized spinal cord cells (52,53), and injury-induced alteration in the pattern of post-ganglionic cutaneous vasoconstrictor neurons, which lose their normal thermoregulatory function, leading to trophic changes and ischemia (54,55).

Evidence that the sympathetic nervous system is involved specifically in phantom limb and stump pain comes from studies in which the sympathetic supply to the involved limb was pharmacologically blocked (27,50) or surgically interrupted (57), resulting in at least a temporary alleviation of pain. Transient relief from phantom limb pain has also been

reported with propranolol (58). Conversely, electrical and mechanical stimulation of the lumbar sympathetic chain produces intense pain in the phantom limb, whereas in non amputee patients suffering from pain, the sensations are referred to the abdomen or flank (59). Regional sympathetic hyperactivity may also contribute to the development of phantom limb pain through excessive vasoconstriction and sweating at the stump and surrounding regions (27,50). The condition may spread centrally from the stump to involve the phantom limb. Hyperalgesia and allodynia 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 (60). The characteristic qualities of superficial burning pain and deep aching pain may be additional evidence of the involvement of the sympathetic nervous system (60).

Despite frequent assertions that the sympathetic nervous system is involved in causing and maintaining phantom limb pain, surprisingly few studies have compared correlates of peripheral sympathetic nervous system activity at the stump and contralateral limb. Sliosberg (61) examined 141 amputees and found the stump to be cooler than the intact limb in 94 of them. However, Sliosberg did not relate the temperature difference to the presence or absence of phantom limb pain. Kristen et al (62) assessed phantom limb and stump pain using thermography and found that a "patchy asymmetrical temperature" distribution of the stump thermogram was significantly more frequent among stump pain sufferers than among patients who were free from stump pain. However, thermographic records taken of the stump were no different for patients with or without phantom limb pain. Nystrom and Hagbarth (63) made microneurographic recordings of activity from muscle nerve fascicles of the peroneal nerve of a patient with a below-knee amputation who suffered from intense cramping pain in the phantom foot. Although bursts of activity in sympathetic fibres were accentuated by the Valsalva manoeuvre, the phantom pain remained unchanged suggesting that this patient's cramping pain was independent of peripheral sympathetic nervous system activity.

In contrast, Sherman and colleagues (64,65) recently found a negative correlation between temperature at the stump and burning phantom limb and stump pain indicating that reduced blood flow to the stump is associated with more pain. However, the relationship between phantom pain and limb temperature was confounded by co-existing stump pain in the majority of patients. Since abnormal blood flow and sweating are common features of sympathetically maintained pain (55,56), there is no reason to assume that patients with burning stump pain might not also have a lower stump temperature.

To evaluate the claim that phantom limb pain (in the absence of concomitant stump pain) is associated with abnormal sympathetic nervous system activity at the stump relative to the intact limb, Katz (66) compared skin conductance and surface skin temperature of the stump and contralateral limb in amputees reporting phantom limb pain, non painful phantom limb sensations, or no phantom limb at all. The mean skin

temperature was significantly lower at the stump than the contralateral limb among those with and without phantom limb pain, but not those without a phantom limb. These results suggest that a phantom limb, whether painful or not, is related to the sympathetic-efferent outflow of cutaneous vasoconstrictor fibers in the stump and stump neuromas. Stump skin conductance responses over time were significantly correlated with the intensity of phantom limb paresthesias, but not other qualities of sensation. This finding supports the hypothesis (outlined below) of a sympathetic-efferent somatic-afferent mechanism involving both sudomotor and vasoconstrictor fibers. The simplest explanation of these findings is that the paresthetic or dysesthetic component of the phantom limb may be triggered by sympathetic-efferent activity (66).

Phantom Limb Paresthesias

Although a normal sensation of a phantom limb occurs whenever nerve impulses from the periphery are blocked or otherwise removed (67), direct stimulation of the stump also frequently exaggerates the paresthetic sensations of the painless phantom limb (68). A cycle of sympathetic-efferent somatic-afferent activity is one mechanism that may alter the intensity of phantom limb paresthesias (66,69). As shown in Figure 4, stump skin conductance correlates significantly over time with the intensity of phantom limb paresthesias. 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 also are not correlated with stump skin conductance (66). That is, changes in stump skin conductance appear to be related only to the perception of paresthesias and not to other qualities of sensation.

It is hypothesized (66,69) that changes in the intensity of phantom limb paresthesias reflect the joint activity of cholinergic (sudomotor) and noradrenergic (vasomotor) post-ganglionic sympathetic fibers on primary afferents located in the stump and stump neuromas (Figure 5). The release of acetylcholine and noradrenaline from post-ganglionic 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 discharge. This information is transmitted rostrally, where it gives rise to referred phantom sensations upon reaching central structures subserving the amputated parts of the limb. Therefore, 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 follow bursts of sympathetic activity and decreases correspond to periods of relative sympathetic inactivity.

Direct support for this hypothesis requires that changes in the intensity of phantom limb paresthesias be correlated with simultaneous microneurographic recordings from post-gan-

glionic sympathetic and primary afferent fibers in amputation stump neuromas.

Psychological and Emotional Contributions

It is not surprising that amputees suffering from phantom limb pain exhibit higher than normal levels of psychological and emotional distress. Depression (71-74), anxiety (74,75) and other forms of psychopathology are common (74-77). Moreover, amputees with severe phantom limb pain score higher on psychological inventories measuring depression (72) and neuroticism (76) than amputees who have little or no pain.

The co-occurrence of phantom limb pain and psychological disturbance has led to three possible conclusions: 1. pain is a symptom of a psychological disorder (7,75); 2. psychological disturbance is a consequence of pain (73); or 3. the two are causally unrelated (71). At present, the consensus is that there is no difference in the prevalence rates of pain of psychological origin among amputees and in 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 of developing such pain. However, prospective studies have yet to be conducted in which pre-operative measures of psychological and emotional functioning are obtained long enough before the amputation to avoid the confounding effects of pre-amputation 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 (7,75,78,79). Denial (of the loss or the associated affect) and repression are the most common defense mechanisms proposed to explain a painless (7) or painful phantom limb (7, 75,79,80) and various changes in the form of the phantom limb (81,82).

Psychodynamic explanations are not consistent with the accumulation of physiological and psychological data. For example, many amputees become profoundly depressed after surgery, yet phantom limb pain and other sensations persist. The co-occurrence of depression and pain is inconsistent with the role of denial since the intense negative affect implies awareness, if not acceptance, of the loss (71). 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 limb, thereby increasing the intensity of paresthesias (83).

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 a phantom limb years after amputation (83). Phantoms that occur after CNS lesions (for example, root avulsions or spinal cord transection) are similar to amputation phantom limbs in the quality of the sensation even though the real limb is still present but totally anesthetic

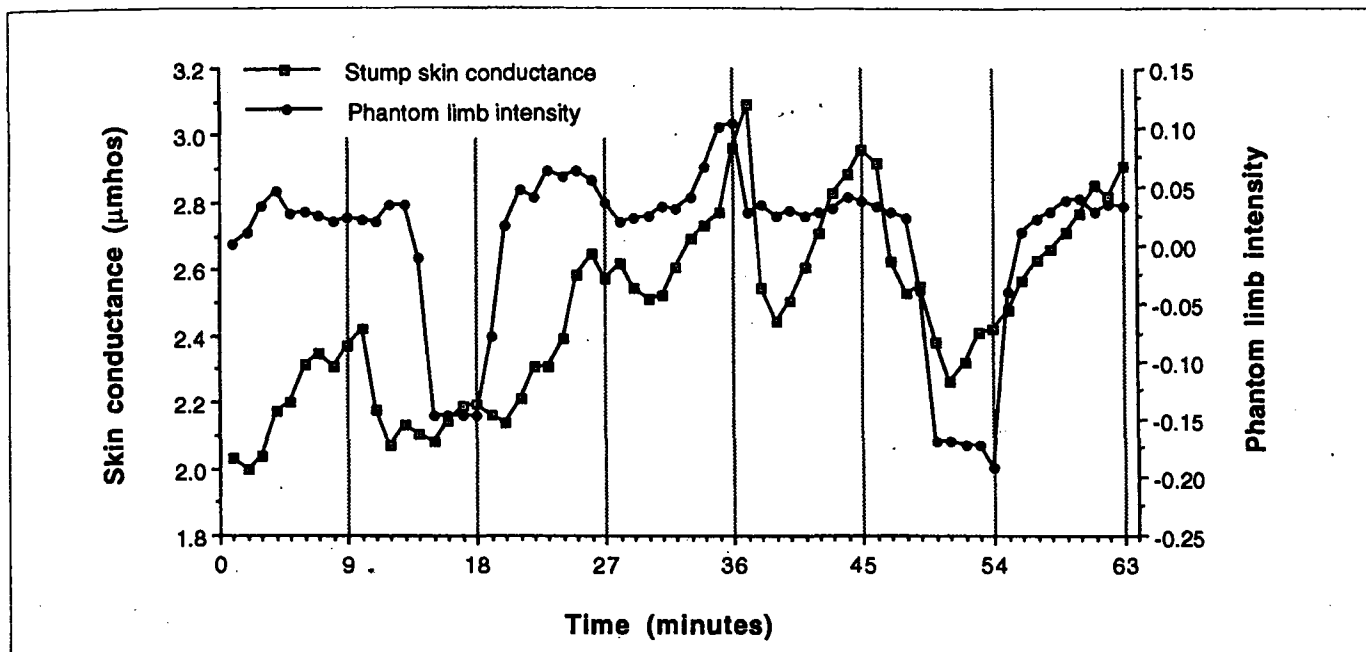


Figure 4. A minute-by-minute plot of the relationship between stump skin conductance and the intensity of non painful phantom limb paresthesias for one subject with an amputation above the knee. Skin conductance was continuously measured at the stump over a 63 minute period; the subject monitored the intensity of the phantom limb by turning a dial. Phantom limb intensity ratings were 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 two second intervals. Changes in the intensity of paresthesias (increases and decreases in "numb" sensations in the phantom toes) occur with changes in stump skin conductance. (Adapted from Katz et al (69) with permission).

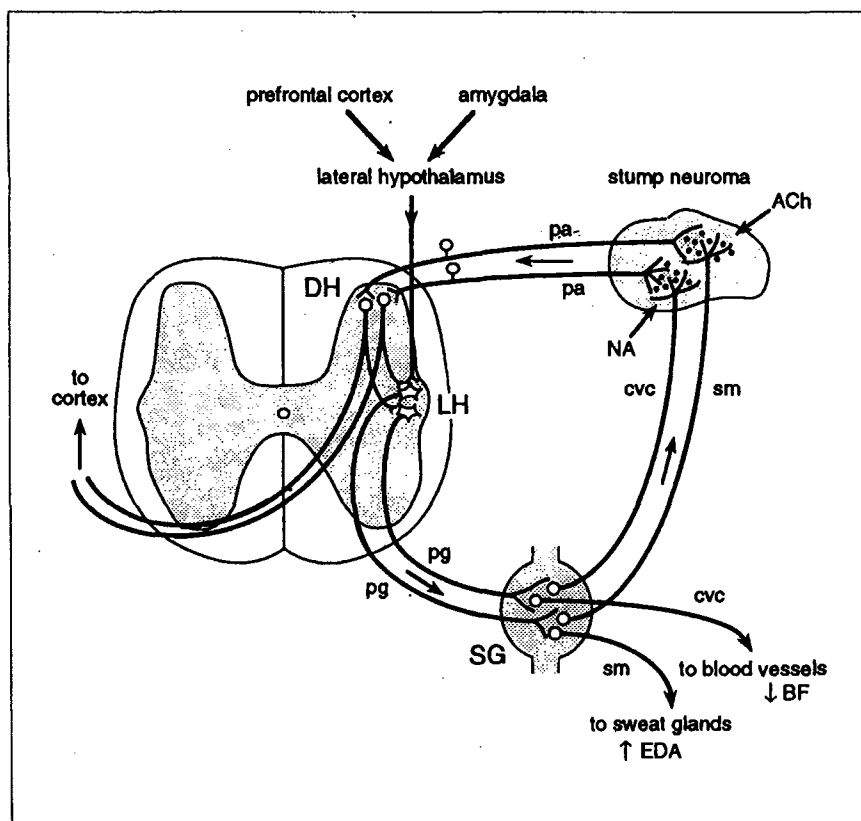


Figure 5. Illustration of a mechanism of sympathetically generated phantom limb paresthesias. Spontaneous activity or excitatory input descending from cortex increases the discharge rate of pre-ganglionic (pg) sympathetic neurons with cell bodies in the lateral horn (LH) of the spinal cord and terminals in the sympathetic ganglion (SG). These neurons excite post-ganglionic 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 pre-ganglionic 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. (Adapted from Fields (70) with permission).

and paralyzed. One would not expect denial of the loss of functioning to produce a phantom defined by paresthesias (84). Phantoms do not develop if the process of sensory loss is gradual, as in the case of leprosy (85), 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 (for example, anesthetic 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 (67,86). Under these circumstances, it is difficult to see the need for 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 (71), the foregoing does not imply that denial of the loss, affect, illness or future implications plays no part in the overall adaptation to the amputation (87). Patients may demonstrate their denial of the importance of these realities in a variety of ways (87), but these do not include a phantom limb. For the vast majority of amputees, a phantom limb — with or without pain — is not a symptom of a psychological disorder.

Characterological Disturbances

In addition to the role of specific defence mechanisms in the development 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" (75). Parkes and Napier (79) describe the "denier" or "defiant type" of amputee by the "obstinate refusal to admit defeat even against better advice...who never accepts that he has lost anything at all. He appears to have a compulsive need to do everything at least as well as he could before operation and if possible, better, as if to convince himself and everyone else that he is not incapacitated at all." With the exception of a recent review (73), 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 (74,88-90), despite the absence of empirical evidence.

In many instances an association between pain and psychological distress (for example, depression and anxiety) or particular personality traits or styles (for example, rigidity and compulsive self-reliance) is influenced by biased sampling procedures so that the characteristics of a select group of patients (for example, those referred to a pain centre) come to define the population at large (73,91). Sherman et al (73) suggest that the low success rate of most treatments for phantom limb pain deters all but the most persistent or self-reliant patients. Long after less assertive patients have given up actively seeking help, these sufferers continue to search for relief despite repeated failures. According to Sherman (73), 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 (8,92) indicate that among an unselected sample of amputees, those with phantom limb pain, painless phantom limb sensations or those with 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. The results also show that there are no significant between-group differences in scores on a questionnaire designed to measure psychological "rigidity", defined by a tendency to persist in behaviours that were effective at one time, or in a particular situation, but no longer are adequate to accomplish current goals.

Pain Memories in Phantom Limbs

Proponents of peripheral or central theories of phantom limb pain have tended to discount pain that could not be explained on the basis of current physiological and anatomical knowledge as psychological in origin (23,93). Arguments have been advanced to explain the similarity of pain before and after amputation as a pathological response to amputation in which the psychological importance of the pre-amputation pain determines whether or not it will be experienced in the phantom limb. These arguments are untenable in light of two lines of recent evidence. First, amputees who reported that their phantom limb pain was the same before and after amputation could not be differentiated using personality, depression, or anxiety inventories from those who did not have phantom limb pain or from subjects who had phantom limb pain which bore no resemblance to their pre-amputation pain (8).

Second, sectioning the sciatic and saphenous nerves of rats is followed by self-mutilation (autotomy) of the denervated hindpaw (94). It is well established that autotomy is a response to painful or dysesthetic sensations referred to the anesthetic limb and represents a model of the phantom limb in the rat. 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 input from the hindpaw, implying that painful or dysesthetic sensations are referred specifically to the region of the injured denervated limb. The correspondence between the sites of prior injury and subsequent autotomy parallels descriptions of human amputees who report the persistence of a pre-amputation pain or lesion referred to the same location of the phantom limb.

In these experiments (94), the injury was always induced while the rats were under a general anesthetic; the rats were maintained under the general anesthetic until well after the sciatic and saphenous nerve transections had been performed. Although the rats never experienced the thermal injury in an awake state, their behaviour in the days after the nerve sections revealed that the effects of the injury were still capable of influencing perception and behaviour (in the absence of further input from the injured region). These findings

imply that somatosensory pain memories reported by human amputees need not represent psychologically important pain and support 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 (8).

Transient Changes Triggered by Psychological and Emotional Processes

As noted above, the idea that emotional and psychological processes can cause pain traditionally has been tied to the notion of psychopathology. However, it is increasingly clear that under certain circumstances pain may also be triggered by these processes in psychologically healthy individuals. Although an amputee may suffer from both psychologically or emotionally triggered pain and psychopathology, their co-occurrence should not be taken as *prima facie* evidence of a causal link.

It is commonly accepted that anxiety or stress influences the perception of pain and subsequent behaviour (91). The aggravation or alleviation of pain in phantom body parts also may be mediated in part by psychological processes that alter anxiety levels (95). Phantom breast pain after a mastectomy was found to be provoked by emotional distress in six percent of women three weeks after surgery and in 29% one year later (96). One-half of all lower limb amputees report that attacks of phantom limb pain can be brought on by emotional distress (12) as long as seven years after the amputation (97). A combination of progressive relaxation and EMG biofeedback of stump and forehead muscles significantly reduces phantom limb pain and anxiety, and the improvements are sustained for up to three years (98,99). Finally, stress levels and pain intensity ratings sampled over a 180 day observation period correlate significantly for most amputees (100).

There are many examples of psychological or emotional processes precipitating transient but profound alterations in the quality and intensity of phantom limb sensations, including hypnosis (101), concentration (28,49), distraction (75), relaxation (98,99), fright (23), forceful reminders of the events that led to the amputation (102), the sight of other amputees (102), and witnessing cruel or violent acts (80,103). One amputee, interviewed by the author (J.K.), described his reaction to an accident involving his wife as "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."

Given that psychological and emotional processes may trigger or exacerbate phantom limb pain, it is essential that patients be prepared prior to amputation for the experience of a phantom limb. Patient education programs and treatment of stress before and after amputation have become standard practice in some institutions (4). Patients who are ill-prepared psychologically for amputation suffer needlessly from phantom limb pain and concern about their sanity (104).

Centrally Triggered Sympathetic-Efferent Somatic-Afferent Mechanism

The material presented above indicates that specific psychological and emotional processes reliably trigger transient pain or sensations referred to the phantom limb. The hypothesis schematically represented in Figure 5 outlines a mechanism by which cognitive and affective processes associated with higher cortical and limbic centres alter phantom limb sensations. The reciprocal connections between cortical, limbic and lateral hypothalamic structures are well documented (105,106). The lateral hypothalamus is involved in the control and integration of neural activity associated with emotionally charged behaviour (105-107) and has direct projections to the lateral horn of the spinal cord (105,106).

The intensity of phantom limb paresthesias and dysesthesias may therefore be modulated by higher brain centres involved in cognitive and affective processes through a multi-synaptic network of descending input that impinges on pre-ganglionic sympathetic neurons producing diffuse peripheral autonomic discharge and activation of primary afferent fibers located in stump neuromas. Mental stress and anxiety not only provoke transient increases in the intensity of phantom limb sensations and pain (95,99,100), but also induce reflex bursting activity in cutaneous sudomotor and vasomotor sympathetic fibers (108,109). Moreover, distraction or attention diversion (and intense concentration) which reduces phantom limb pain (28,75) also diminishes peripheral sympathetic nervous system activity (109).

Occasionally, the effects of intense emotions (for example, fright, horror) are experienced diffusely over the entire body as cutis anserina associated with pilomotor contraction (i.e., "goose bumps" or a tingling sensation). Among amputees, however, a more frequent occurrence is that less salient emotions precipitate these sensations only throughout the phantom limb. The tendency for emotionally 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. First, regenerating sprouts, which are trapped in a neuroma, are exceedingly sensitive to the post-ganglionic sympathetic neurotransmitters noradrenaline (110) and acetylcholine (111) and discharge rapidly when these substances are present. In contrast, intact peripheral fibers do not show this chemosensitivity and thus have a higher threshold than regenerating sprouts. Second, deafferentation results in a loss of inhibitory control over cells in the dorsal horn and more rostral sensory structures (67,112), giving rise to the perception of a phantom limb (59,67,68). 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 (68). These two observations may explain the propensity for the phantom

limb to be the site of emotionally generated, sympathetically triggered sensations and pain.

Conclusions

The material presented in this paper reveals that the phantom limb is not perceived as a static entity, but as a fluid, frequently changing perceptual experience characterized by fluctuations in tactile, thermal, kinesthetic and proprioceptive sensibility. Phantom limb phenomena range from simple diffuse paresthesias to perceptually complex experiences of pain and lesions that originally were felt in the limb before the amputation. Pain should be alleviated before the amputation to prevent pathological pain from developing afterward. Changes in the length and size of the phantom limb appear to vary with the use of the stump and indicate the importance of peripheral input in modifying the body image after amputation. Finally, the sympathetic nervous system may provide an important link between higher cortical activity and phantom limb sensations through its peripheral actions on primary afferents located in stump neuromas. The data are consistent with Melzack's (6) theory that: 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. input from the periphery may trigger or modulate phantom limb sensations and pains but are not necessary for any of the qualities of experience.

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Résumé

Des études récentes sur les amputés révèlent la remarquable diversité des expériences relatives au membre disparu, à savoir une expérience indolore ou douloureuse. L'auteur procède à une analyse sélective des observations sur les mécanismes périphériques, centraux et psychologiques qui actionnent ou modulent diverses expériences concernant le membre fantôme. Les données indiquent que la douleur vécue avant l'amputation peut persister en forme de «mémoire» somatosensorielle dans le membre fantôme. On suggère que la longueur et la grandeur du membre fantôme peuvent servir d'indicateurs perceptuels du fait que les données sensorielles du moignon occupent à nouveau les régions du cortex dépourvues qui servaient le membre amputé. On présente un mécanisme périphérique, qui comprend un cycle sympathique-efférent somatique-afférent, pour expliquer les variations de l'intensité des parasthésies référées au membre amputé. L'auteur conclut en indiquant que l'expérience relative au membre fantôme dépend d'une interaction complexe de données issues de la périphérie et de régions plus générales du cerveau qui dirigent les mécanismes sensoriels, intellectuels et affectifs.