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Phantom Pain

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Preface

Phantom pain is an intriguing mystery that has captured the imagination of health care providers and the public alike. How is it possible to feel pain in a limb or some other body part that has been surgically removed? Phantom pain develops among people who have lost a limb or a breast or have had internal organs removed. It also occurs in people with totally transected spinal cords. Unfortunately, phantom pain is a medical nightmare. Many of the people reporting phantom pain make disproportionately heavy use of the medical system because their severe pains are usually not treated successfully. The effect on quality of life can be devastating.

Phantom pain has been reported at least since 1545 (Weir Mitchell as related by Nathanson, 1988) and/or experienced by such diverse people as Admiral Lord Nelson and Ambroise Paré (Melzack & Wall, 1982; Davis, 1993). The folklore surrounding phantom pain is fascinating and mirrors the concepts about how our bodies work that are in vogue at any particular time. Most of the stories relate to phantom limbs and date from the mid-1800s. The typical story goes like this: A man who had his leg amputated complained about terrible crawling, twitching feelings in his leg. His friends found out where the leg was buried, dug it up, and found maggots eating it. They burned it, and the pain stopped. Another man complained of a swollen feeling with frequent stinging or biting pains. He didn't know what had been done with his leg. When his friends investigated, they found out that it had been dumped into the water. Once he realized that he was feeling fish biting his leg, he was able to live with the pain, but it never went away. In another case, a man reported feeling terrible burning in his phantom that began just after he had an amputation. His doctor found out that the burning started when his assistant burned the limb to dispose of it. They poured water on the limb, and the burning stopped. Jensen *et al.* (1984) reported that similar stories were prevalent in Scandinavian folklore. Their stories included an itching hand that had been buried and subsequently cremated to stop the sensation. Another concerns a burning hand

that was cremated and had its ashes scattered in a lake to stop the pain. In view of the abundance of stories like these, it is no wonder that many people thought that phantom pain was all in the sufferers' heads.

Our ways of thinking about the body have changed since those times, as have our ways of trying to investigate the causes of pain problems. Any review of the current, modern "scientific" literature about phantom pain will produce a plethora of clinical articles with minimal supporting evidence that the treatments they espouse actually work. The review will also bring out many theoretical articles seeking to explain the various phenomena comprising phantom pains that are based nearly entirely on conjecture supported by only the thinnest threads of tangentially related experimental evidence. In other words, these clinical and theoretical publications are our current scientific folklore.

Amputees don't generally read the medical literature in detail. They do talk with each other and have their own general opinions and arguments about what causes phantom pain and which treatments work (and which don't). This body of fragmented, conflicting knowledge (folklore) has considerable credibility in the amputee community and guides many amputees in their choices of care to seek and to avoid. Amputees also tend to view the devastation of phantom pain far differently than medical practitioners do. Many clinicians do not feel that a problem is really clinically important (i.e., has a major clinical impact) if it occurs only a few times per year and does not make an obvious, direct contribution to the patient's physical dissolution. In turn, many amputees are afraid to discuss their phantom pain with their doctors for fear of losing their credibility or being thought of as wimps. They are also deeply concerned about being offered treatments still commonly used by the medical community that the amputee community rejected long ago as useless or, worse, harmful. This body of knowledge has not been commonly available to the medical community until the last few years, so there are really two bodies of folklore to consider when attempting to tease out the realities of phantom pain.

It is the intention of this book to present an integrated compilation of information about phantom pain and associated phenomena that has been elucidated through organized investigations in order to view much of our current folklore in relation to information more likely to withstand the test of time. In recent years, organized studies from areas such as epidemiology, psychology, physiology, and medicine have begun to alter phantom pain's reputation from that of a mysterious syndrome apparently experienced by a few mentally unbalanced amputees, for which dozens of supposedly effective treatments were available, to that of a poorly understood problem experienced by most amputees, for which no effective treatments were available, to a moderately well-understood problem, for which several

treatments have been proven effective. Thus, when reading this book, readers can expect to find our best evaluation of effective treatments, current clinical and physiological evidence upon which current therapeutic recommendations are based, and, of course, conjectures based on what we think we know, which, themselves, need to meet the tests of time and further research.

There have been previous attempts to synthesize what is known about phantoms into a coherent framework, or at least to gather much of the available knowledge into one volume. For example, Cronholm's work entitled "Phantom Limbs in Amputees" appeared in 1951, and Siegfried and Zimmerman's edited volume entitled *Phantom and Stump Pain* came out in 1981. Numerous other reviews in journal article, chapter, and booklet form have appeared both before and since these works. Thus, our review is only the latest in a series of attempts to elucidate the field. The authors of this volume have been among the principal authors of almost all of the more recent review articles and chapters dealing with phantom limb pain. They have also written a substantial number of the recent basic research and clinical articles in field. Thus, much of the material here has been published in one form or another in a variety of recent works (e.g., Sherman *et al.*, 1994a, b), and we appreciate the permission given by various publishers to use the material.

Many people were instrumental in performing the studies that led to this book. I specifically want to thank my wife, Crystal Sherman, who not only participated in the design and performance of many of our studies but also acted as a sounding board for my ideas throughout our years of trying to view the problem of phantom pain from every approach. She has the endless patience to listen to an unending barrage of incomplete, nebulous ideas and the rare ability to sort out the ones with a few shreds of hope for success, and then help us gradually work the survivors into rational studies.

A few words should be said about the way the contributions to this volume were interdigitated. Most "multiauthor" books recently published in the sciences are actually compendiums of independent contributions in which each author writes a chapter on his or her area of expertise. The explosive growth of knowledge along with subspecialization frequently makes this a necessity if a reasonably broad topic is to be covered authoritatively. Unfortunately, the contributions are frequently contradictory, repetitive, and rarely sufficiently well organized to produce a coherent whole that would be as well integrated as a single-author book on the same topic. The topic of "phantom pain" is no exception. The amounts of specialized knowledge available on this subject are such that no one "expert," however erudite, can do it all.

The authors wanted to avoid the typical problems of multiauthorship

without limiting each contributor to writing on just one narrow subtopic. Thus, although each subsection was written independently by one of us, the subsections were subsequently interdigitated throughout the text to produce a document that we hope you will find to be integrated and coherent with a logical flow of information. For example, three of the authors wrote about sensory characteristics of phantom pain, and four wrote about its treatment. It would have been confusing to have characteristics written in three different places in the book and treatment recommendations in four.

As might be expected, when authors with diverse areas of expertise join in one effort, different approaches are taken in exploring the same questions. Thus, some of the issues appear in more than one chapter, with a different "slant" in each location. Each author reviewed the entire text, so numerous comments were available for each subsection. Virtually all of the comments were negotiated into mutually acceptable compromises before being incorporated into the text. Thus, a subsection ascribed to one author may have been shaped with contributions from the other authors.

RICHARD A. SHERMAN

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Phantom Pain

CHAPTER 1

Locations, Characteristics, and Descriptions

Richard A. Sherman, Joel Katz, Joseph J. Marbach, and Kim Heermann-Do

Locations and Descriptions of Phantom Limb Pain

Many of the large surveys and reports on which this information is based were conducted on a population of amputees who were veterans of the United States and other military forces. This is a selected group, so the results could be biased. However, our work with people whose amputations were not related to the military provided results similar to those in the above surveys (Nyström & Hagbarth, 1981), and other workers have recently confirmed similar rates of occurrence in other populations (Sherman & Arena, 1992; Sherman, Arena, & Ernst, 1990). Thus, we feel that our results are representative of the general amputee population. Figure 1 illustrates typical phantom sensations.

Perhaps the most critical point is that the vast majority of amputees who experience phantom pain are very consistent in their descriptions of their pain and of its location. In other words, an amputee who reports burning phantom pain apparently emanating from the ankle will consistently report that description at that location even if intensity varies or he or she feels other descriptive types of phantom pain elsewhere in the phantom.

Many clinicians report that there is a natural progression in expression of the phantom over time. The typical scenario is that the phantom mimics the original limb in detail and size just after amputation and gradually telescopes into the distal end of the residual limb (stump) while progressively losing detail from proximal to distal. This process is illustrated in

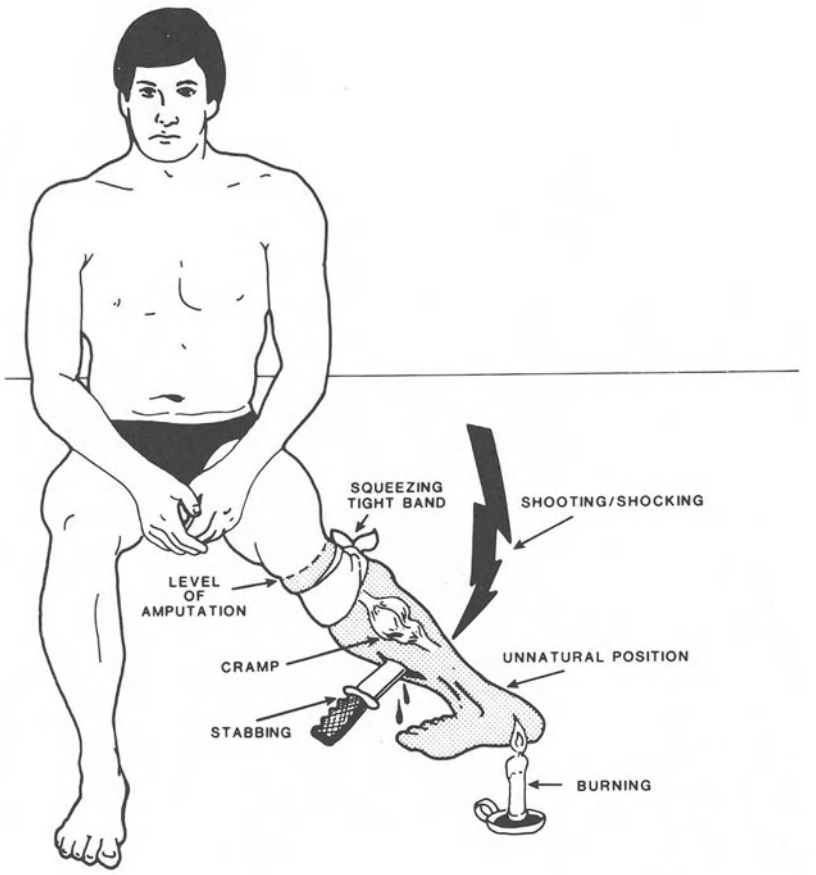

Figure 1. Typical descriptions of phantom limb pain (from Sherman, Sherman, & Grana, 1989).

Figure 2. Shukla, Sahu, Tripathy, and Gupta (1982a, 1982b) reported the presence of telescoping in nearly two-thirds of the 72 amputees they interviewed. The scenario typically includes the perceived ability to control much of the motion in the phantom and the presence of most of the normal, benign sensations that would be present in any intact limb at about their normal intensities. Weiss and Fishman (1963) interviewed 239 adult unilateral amputees and found that the longer the residual limb, the more telescoping occurred. Hrbek's (1976) review indicated that movements of the phantom could be spontaneous, automatic reflexive (as in motions relating to losing one's balance), in conjunction with movements of the

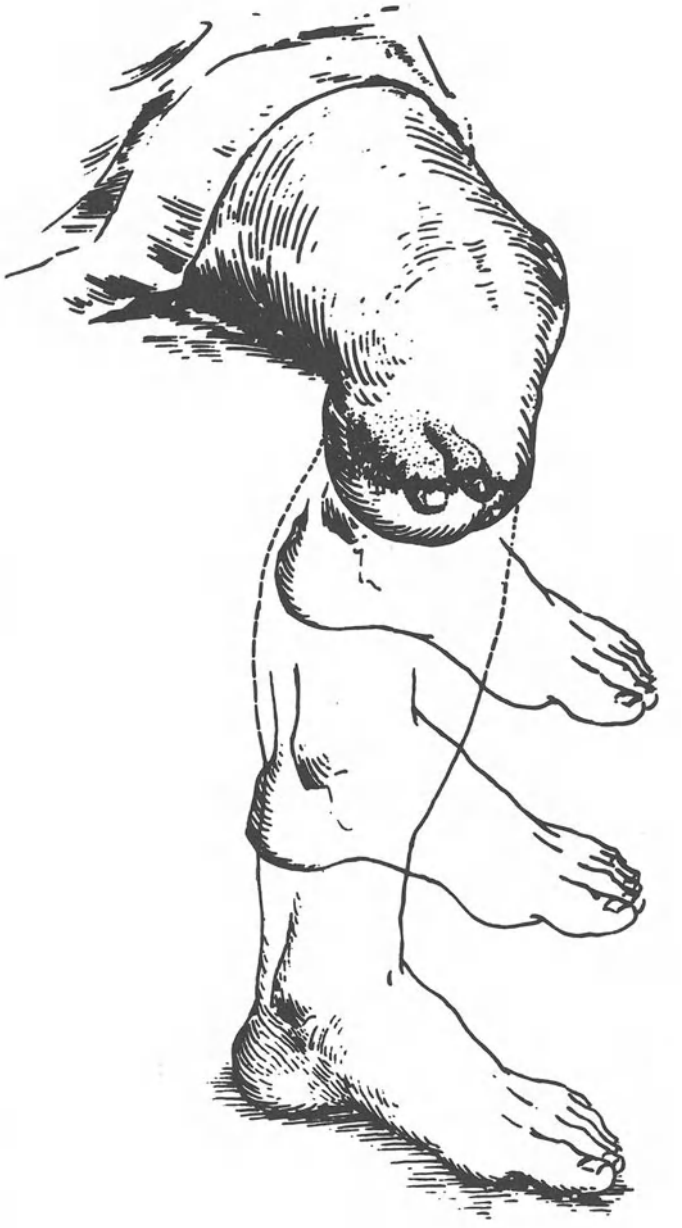

Figure 2. Progressive telescoping of the phantom over time after amputation (from Studies relating ..., 1952).

intact limb, or voluntary. Steinbach, Nadvorna, and Arazi (1982) did a 5-year follow-up of 42 posttraumatic amputees and found that the longer the phantom appeared to be, the greater was the perception of motion. Most reviewers indicate that the level of control and sensation gradually decreases in conjunction with the telescoping phenomenon. We have not found this to be the case when phantom pain is present. Rather, the limb remains detailed and distinct when chronic phantom pain is reported.

Verbal descriptions of painless and painful phantoms have been in the medical literature for hundreds of years, but they frequently lack critical details such as relationships with location of pain before amputation, description of the pain, and exact location on the phantom. In 1952, an unsigned progress report was sent to the Advisory Committee on Artificial Limbs of the National Research Council that reported work performed between 1946 and 1951. It included numerous drawings made by skilled medical illustrators working in conjunction with amputees to depict their pain. As far as we can determine, this work was never actually published in any source widely available to clinicians. The work included epidemiologic, electrophysiological, treatment, and other studies on about 236 amputees. We have included results from the report throughout this book (Studies Relating ..., 1952). Figures 2–8 are from this source. Cronholm (1951) began his extensive series of studies on 122 amputees in Sweden in 1947 and published them in 1951, so the two studies occurred over about the same period of time and probably used similar patients—amputees from World War Two.

Relationships between Phantom Limb Pain and Painless Phantom Sensations

Cronholm (1951) quotes Weir Mitchell, D. Katz, and numerous others who discussed the amputee's ability to move painless phantoms. Several of these observers stated that changes in muscle tension in the phantom that would have moved the missing portion of the limb now controlled corresponding motions in the phantom. Cronholm quotes other workers including Henderson and Smyth (1948) and Lunn (1948) as reporting phantoms to have similar sensations to those found in intact limbs including "pleasant" tingling. Lunn (1948, as quoted by Cronholm, 1951) reported that 56% of his 150 amputees noted temperature in the phantom ranging from slightly warmer than the intact limb to burning. Only about 27% felt that the limb was about the same temperature as the intact limb.

Respondents to three studies by Sherman *et al.* (Sherman & Sherman, 1983; Sherman, Sherman, & Parker, 1984; Sherman, Ernst, & Markowski, 1985) were asked to describe the location, feeling, and intensity of phantom sensations that were not painful. These descriptions were compared with

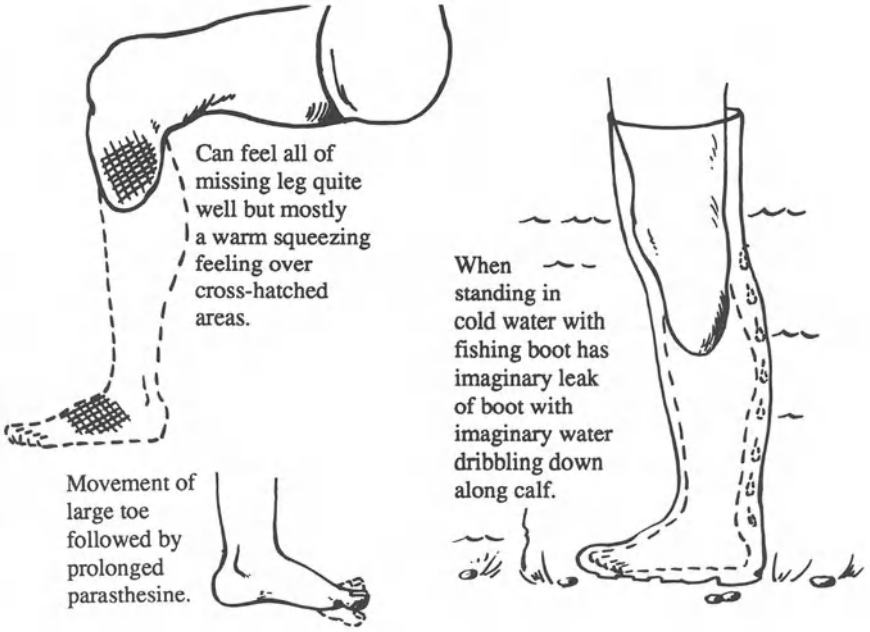

Figure 3. Painless phantom sensations including movement (from Studies relating ..., 1952).

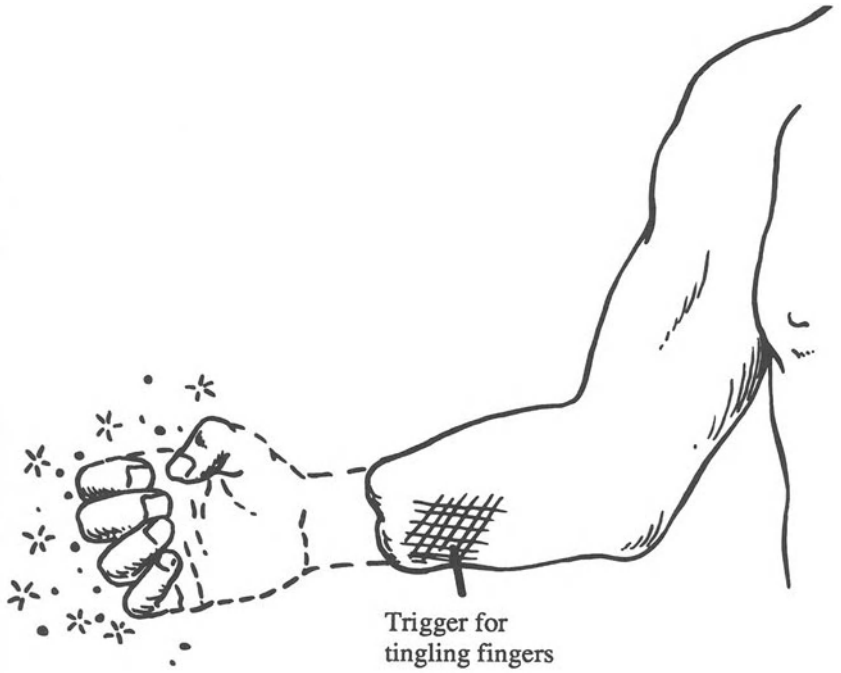

Figure 4. Telescoping of the phantom into the residual limb (stump) as well as depiction of a "trigger" area in the stump that initiates episodes of phantom pain (from Studies relating ..., 1952).

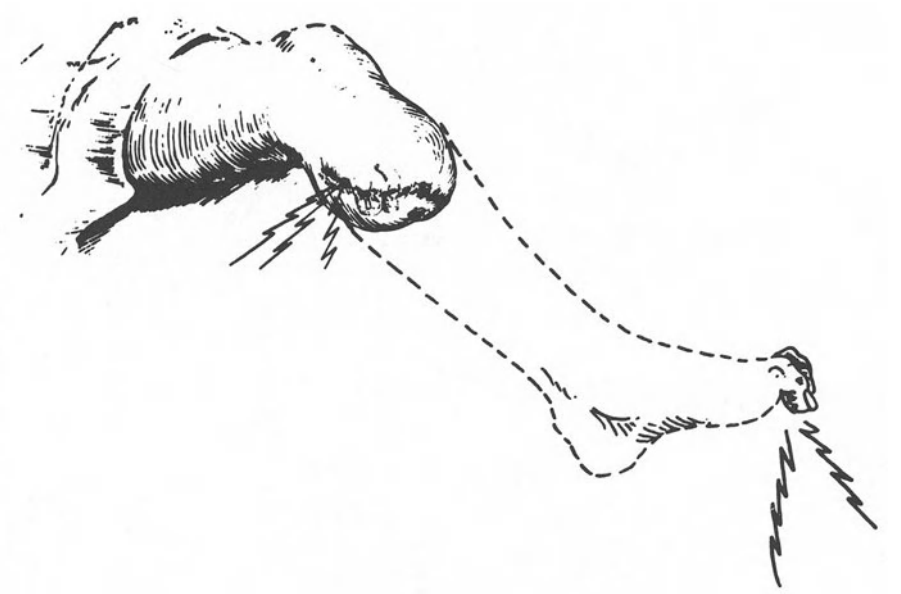

Figure 5. Depiction of a "trigger" area in the stump that initiates episodes of cramping phantom pain and flexion of the toes.

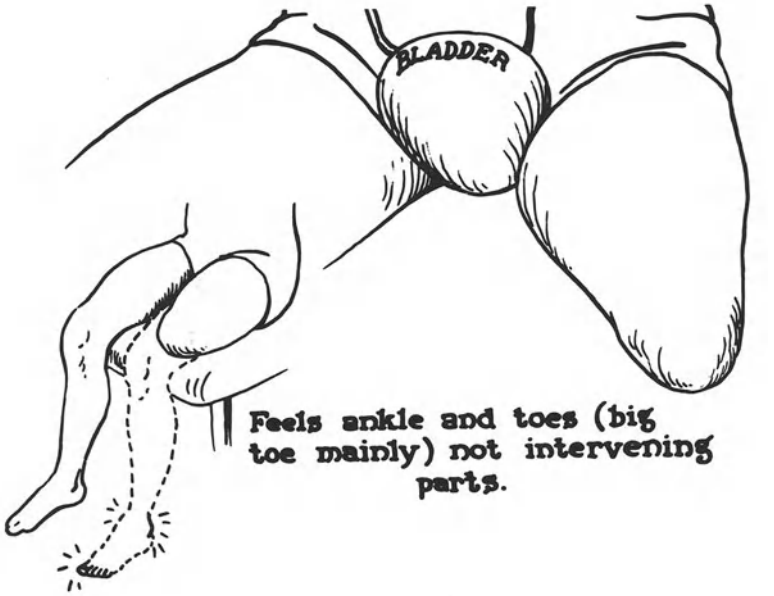

Figure 6. First of two illustrations depicting potential relationships between cortical representation and location of phantom sensations. Drawing of sensations from a distended bladder initiating discomfort in the phantom (from Studies relating ..., 1952).

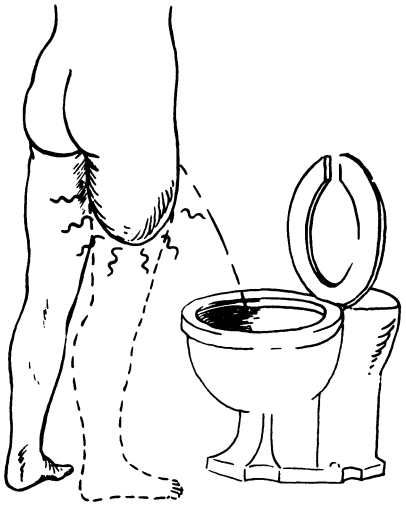

Figure 7. Second of two illustrations depicting potential relationships between cortical representation and location of phantom sensations. Drawing of urination initiating burning phantom pain near the stump (from Studies relating ..., 1952).

the same information requested for painful phantom sensations. The distribution of the locations of painful and benign phantom sensations were statistically similar ($\chi^2 = 11.3$ with 8 df, $p = 0.185$), as were the descriptions of what the benign and painful sensations felt like ($\chi^2 = 6.19$ with 8 df, $p = 0.626$). These parallels are illustrated in Table 1.

Sherman and Sherman (1983) requested 1200 survey respondents to rate the intensity of their painful and nonpainful (benign) phantom sensations on the same 0–100 scale. Those reporting intermittent phantom pain rated their nonpainful sensations as being of the same intensity as those not reporting any phantom pain ($\chi^2 = 45.67$ with 76 df, $p = 0.998$). They usually referred to the same sensations as being painful, so they did not have nonpainful ones to rate. There was a trend for all amputees with phantom pain to rate the intensity of their benign feelings slightly higher than did those without phantom pain (respective means of 57.4 ± 30.4 and 43.1 ± 36.4 with $t = 1.96$ and 578 df, $p = 0.051$). However, this is clearly a rating artifact, as those with continuous phantom pain were usually rating the intensity of their phantom pain rather than that of their benign sensations. The average rating for the worst episode of phantom pain was 68.7 ± 29.99, and for the least intense usual pain was 17.99 ± 22.81.

Benign sensations were usually continuous, but phantom pain was intermittent in many cases. Clearly, about half of those reporting phantom

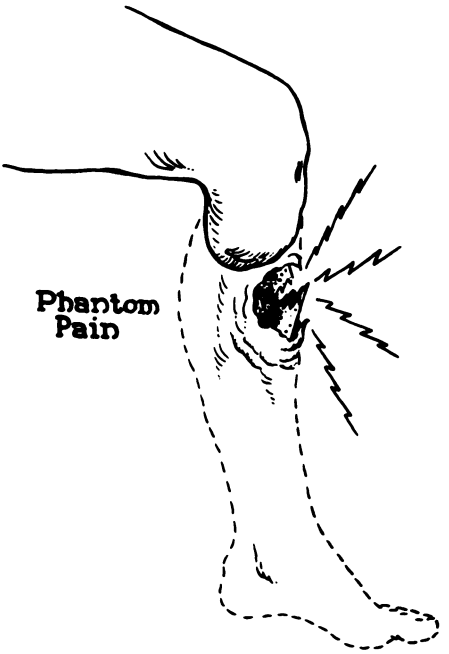

Figure 8. Illustration depicting potential central "pain memory" phenomena. Drawing is of phantom pain at the site of a lesion present before amputation (from Studies relating ..., 1952).

pain have continuous pain, and about half have intermittent pain. Episodes are most frequently reported to be once per day or once per week, with fewer reporting monthly, yearly, or rarer episodes. Episodic patterns were usually temporally consistent in length for each individual, but different people reported that episodes lasted from seconds to weeks. Durations of seconds, minutes, and hours were most commonly reported; reports of durations of days or weeks were rare. Sherman *et al.* (1984) requested similar information from a larger (5000) population of amputees surveyed and found that the comparative distributions of usual intensity of the phantom pains and painless phantom sensations were very similar. Respondents were asked to rate the intensity of painless sensations on the 0–10 scale as if they were painful. Those people who experienced both phantom pain and painless sensations were usually those with episodic phantom pain. They reported that their painless phantom sensations had an average intensity of 5.6 ($\pm$ 3.9). Those respondents who never reported any phantom pain gave their painless phantom sensations an average

Table 1. Locations and Descriptions of Phantom Sensations[a]

	Painful phantom sensations		Painless phantom sensations	
	Military	Civilian	Military	Civilian
Reported locations				
Toes/fingers	29	29	33	31
Foot/hand	41	39	39	39
Ankle/wrist	12	11	13	11
Lower leg/arm	9	14	8	13
Knee/elbow	5	5	5	4
Near but not at end of stump	4	1	2	1
Reported descriptions				
Warm	3	8	9	11
Hot	15	14	11	11
Squeeze	16	13	27	22
Unusual position	4	4	3	4
Broken	1	0.3	1	0.2
Tingle	14	13	19	19
Sharp shock/shooting	32	33	19	22
Cramp	15	14	11	11

[a]Percentages of subjects reporting each category.

intensity of 5.7 (± 3.5). The similarities in intensity, description, and distribution between painful and painless phantom sensations tend to confirm the impression formed as a result of our earlier work that continuous phantom pain may be an intensified version of painless sensations, although episodic pain has other origins.

Incidence of Phantom Limb Pain among Adults

It has been estimated that there are about 450,000 amputees in the United States (Stein & Warfield, 1982). Until just a few years ago, only about 0.5% of them were thought to experience phantom pain (Kolb, 1954). Many of these patients were thought to have psychological problems that led them to exaggerate or totally invent their pain problems. Our studies have shown that the majority of amputees suffer from significant amounts of phantom pain on a frequent basis.

We conducted a series of surveys of over 11,000 amputee veterans (Sherman & Sherman 1983; Sherman *et al.*, 1984) in which over 80 percent of

respondents reported significant phantom pain. Other workers have confirmed similar rates of occurrence in other populations (Carlen, Wall, Nadrorna, & Steinbach, 1978; Jensen, Krebs, Nielsen, & Rasmussen, 1985; Sternbach, 1982; Purry & Hannon, 1989). Our respondents reported that virtually all treatments have a reputation for being painful failures or embarrassing, useless referrals to psychiatrists. In one of our amputee surveys (Sherman *et al.*, 1985), 69% of the 2700 veteran amputees responding told us that their physicians had directly stated or had clearly implied that the pain was "just in their heads." The great majority of amputees responding to this survey were afraid to tell their physicians that they were suffering with phantom pain for fear that the physician would think them "insane." They were afraid to jeopardize the critically important relationship with their physician or to risk losing credibility in reporting stump problems at a stage when a verbal report is frequently the only evidence that problems exist.

Problems in the residual limb are frequently painful and can entirely prevent the use of a prosthesis for extended periods of time unless intervention is begun before development of obvious skin breakdown and other highly noticeable effects. This could account for differences in the reported rate of phantom limb pain such as Kolb's (1954) finding that 1/2% of amputees in a large clinic initially reported having phantom pain but 5% admitted having it when asked directly. Sternbach's analysis (Sternbach *et al.*, 1982) of the literature suggests that a 0.5–10% incidence for chronic phantom pain is the accepted norm. Our survey had no connection with health care providers and showed that 85% of respondents reported experiencing significant phantom pain. This survey had a 61% response rate. Because 85% of the respondents reported phantom pain, a minimum of 51% of the surveyed population had phantom pain even if all nonrespondents were free of phantom pain. We feel that our results are representative of the general amputee population. The discrepancy between more recent studies and those of earlier workers is probably a result of sampling bias. Recent samples were either randomly selected or included the entire population, whereas others were working with self-selected groups requesting treatment for other conditions requiring continuing physician support and acceptance.

It is especially unfortunate that the higher rates of occurrence of phantom pain were established by independent workers between 1947 and 1952, but the information was usually in unpublished documents or sources that were not readily available to the clinical community. For example, a progress report dated 1952 from the "Prosthetic Devices Research Project" (Studies relating ..., 1952) reported rates of at least 70% for persistent phantom pain from 208 amputees.

Intensity and Frequency of Occurrence of Phantom Limb Pain

As early as 1947 (Studies relating ..., 1952), it was known that, for a minority of amputees, phantom pain does go away a random number of years after the amputation but that most continue to suffer from phantom pain anywhere from a few isolated incidents lasting for moments per year to continuous pain. It was also known that amputees who were pain-free for years could suddenly and inexplicably begin to experience phantom pain years after their amputations. For example, the above study (Studies relating ..., 1952) of 218 subjects found that 18% had continuous phantom pain, 33% had daily episodes, 34% had episodes less than once per week, 35% had episodes less than once per month, and none had episodes less than once per year. Twenty-two percent reported that the pain was severe enough to interfere with their routines, and 81% reported that their pain was moderate or slight. Several subjects reported two levels of pain, so the total adds up to over 100%. The duration of painful episodes ranged from days for 11%, through hours for 13% and minutes for 48% to seconds for 28% for the 35 subjects for whom data were available.

Sherman *et al.* (1984) found similar results in their survey of 5000 veteran amputees, which had a 55% response rate. About half indicated that phantom pain decreased at least slightly with time, whereas the other half reported either no change or an increase. One quarter of those who reported no phantom pain indicated that they initially had some but that it had disappeared over time. Among those with current phantom pain, 27% felt it for at least 20 days per month, 10% for 11–20 days, 14% for 6–10 days, 35% for 2–5 days, and 14% for 1 day per month. Among amputees with daily episodes of phantom pain, the number of hours per episode ranged from over 15 hr by 17%, 11–15 hr by 7%, 6–10 hr by 14%, 2–5 hr by 32%, and 1 hr or less by 20%. A similar survey by Sherman and Sherman (1983) showed that amputees rated the duration of their episodes as seconds by 38%, hours by 37%, days by 11%, months by 2%, and continuous by 12%. The usual intensity of phantom pain is positively correlated with duration of episodes; this relationship is illustrated in Figure 9. The relative frequency and duration of phantom pain episodes between people whose amputations were related to military or civilian events are presented in Table 2. The relative intensities of episodes for these groups are shown in Table 3. Jensen *et al.* (1985) found that the rate of occurrence of phantom pain in 58 patients dropped from an initial rate of 72% 8 days after amputation to 65% 6 months after surgery and then to 59% 2 years after surgery.

Respondents were asked to rate the intensity of their pains on a scale of 0 through 10 in which 0 equaled no pain and 10 equaled so much pain

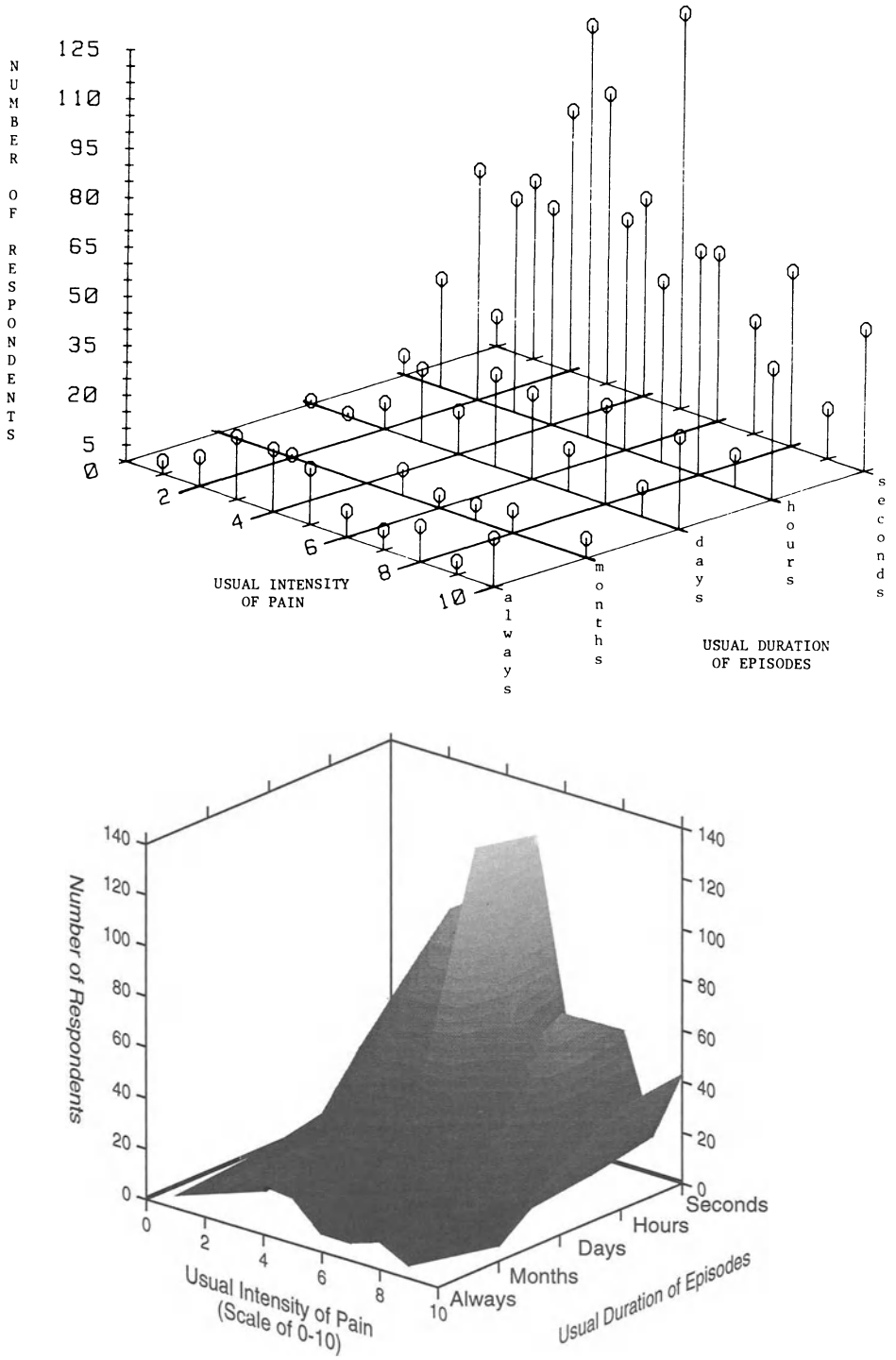

Figure 9. Usual intensity of phantom pain versus duration of episodes.

Table 2. Relative Frequency and Duration of Phantom Pain Episodes

Days/month of phantom pain			Hours/day of phantom pain			Duration of individual episodes		
Days/month	Military[a]	Civilian[a]	Hours/day	Military[a]	Civilian[a]	Time	Military[a]	Civilian[a]
Over 20	34	40	Over 15	27	35	Seconds	38	37
11–20	10	7	11–15	7	7	Hours	37	33
6–10	6	8	6–10	14	11	Days	11	8
2–5	35	31	2–5	32	29	Months	2	1
1	14	13	1 or less	20	18	Continuous	12	20
Below 1	0.3	1						

[a]Percentage of subjects reporting each category.

Table 3. Relative Intensity of Phantom Pain[a]

Rating	Military	Civilian
Average	5.3 (4.9)	5.0 (2.7)
Worst	7.7 (4.6)	7.4 (2.6)
Least	2.9 (5.1)	2.4 (2.4)

[a]On the 0–10 scale showing means, with standard deviation in parentheses.

that they would commit suicide if they had to bear it for one more second. On this scale (Table 3), the average intensity of phantom pain was rated as 5.3 (± 4.9), the worst was 7.7 (± 4.6), and the least was 2.9 (± 5.1). Some respondents rated their least pain 10 (suicide level). Most of those were people who had brief episodes that were always quite severe but did not last long enough to cause them to commit suicide.

Gerhards, Florin, and Knapp (1984) found that 30% of 178 male above-the-knee unilateral amputees (mostly from war injuries) suffered an average of 200–500 hr per year of phantom pain and 100–200 hr per year of severe stump pain. Gerhards found that 53% felt severely burdened by pain, and only 7% experienced neither stump nor phantom pain.

Phantom Limb Pain among Children

In contrast to the many published studies describing phantom limbs in adults, the problem of phantom limb pain among children and adolescents has not received the clinical and research attention it deserves. In fact, very little is known about the demographic characteristics of pediatric amputees. Krebs and Fishman (1984) attempted to get a rough idea of some 'haracteristics by sending a survey to all of the members of the Association of Children's Prosthetic–Orthotic Clinics in the United States and Canada. Forty-five of the 74 clinics replied and gave information on 4105 children under the age of 21. Congenital limb deficiencies outnumbered amputees by a ratio of two to one. However, more unilateral lower-limb amputees were reported than similar congenital deficiencies. Eighty percent of amputees were unilateral. Boys outnumbered girls by a ratio of three to two, and 54% of the amputees had the left limb removed.

Case studies of phantom limb pain in children (Bradley, 1955; Roger, 1989) and adolescents (Frazier & Kolb, 1970; McGrath & Hillier, 1992;

Riddoch, 1941; Solomon & Schmidt, 1978) with congenital absence of limbs (Sohn, 1914) are rare, but this rarity does not seem to accurately reflect the scope of the problem. For example, Boyle, Tebbe, Mindell, & Mettlin (1982) reported that approximately 7 years after amputation performed in childhood or adolescence, 70–75% of individuals continue to experience phantom limb pain, although none reported the pain to be severe. A recent retrospective survey of 24 individuals who underwent amputation in childhood or adolescence found a prevalence rate of 92% for phantom pain, with the pain persisting for months to years in all subjects. Although there was an overall trend for the number of episodes and intensity of phantom pain to decrease with time, 36% reported that the pain remained unchanged (Krane & Heller, 1995). The most common descriptors of the pain included "sharp," "tingling," "stabbing," and "uncomfortable."

Consistent with the literature from the adult amputee population (Jensen, Krebs, Nielsen & Rasmussen, 1983), Miser and Miser (1989) contend that phantom limb pain is more common if the child suffered preamputation pain, but they do not include data to support this claim. Lacroix, Melzack, Smith, and Mitchell (1992) report the case of a 16-year-old adolescent who underwent amputation of her right foot in infancy because of a congenital abnormality consisting of a flatfoot that was locked in a position incapable of movement. The patient described her phantom foot as flat and stuck in a forward position even though she had no awareness of the nature of the congenital abnormality. The study by Krane and Heller (1995) found that the majority of children with phantom limb pain also reported having had preamputation pain, but this relationship did not reach conventional levels of significance. More data are needed on the similarity of pains before and after amputation and not simply on the relationship between pain before and after amputation. We do not know the extent to which preoperative pain and/or noxious events during surgical amputation contribute to phantom limb pain in children.

Taken together, these data suggest that phantom limb pain in children is not unusual and even appears to be a significant problem for some children. It is unclear whether the paucity of data represents an undetected problem: a child's response to pain may differ from that of an adult depending, in part, on the child's age, cognitive level, and emotional maturity (McGrath, 1990). In support of this is the discrepancy between the incidence of phantom limb pain as reported by the child and the relative lack of physician/nurse documentation of the problem (Krane & Heller, 1995). It is also possible that the absence of information in the literature reflects the lower priority assigned to phantom limb pain when compared with other obstacles the child or adolescent must contend with in coping with life after amputation (Boyle *et al.*, 1982; Lasoff, 1985; Tebbi & Mallon,

1987; Tebbi, Petrilli, & Richards, 1989). Self-esteem, depression, social support, and family functioning have recently been assessed in child amputees (Varni, Rubenfeld, Talbot, & Setoguchi, 1989a, 1989b, 1989c; Varni, Setoguchi, Talbot, & Rubenfeld Rappaport, 1991) and strategies have been designed for them to cope directly with social and interpersonal situations that arise as a consequence of the amputation (Varni & Setoguchi, 1991). Nevertheless, there is a conspicuous absence of information on phantom limb pain among children and adolescents. We do not know the nature or magnitude of the problem. The incidence, various qualities, intensity, frequency, and duration of episodes, relationship to preamputation pain, and time course of phantom limb pain are virtually undocumented in children.

Considerably more research has focused on whether phantom limbs occur in children born without limbs, in child and adolescent amputees, or in adults who underwent amputation in childhood or adolescence. These studies are usually carried out to support one of two opposing theories concerning the origins of the body schema or body image. Until recently, a frequently cited paper (Riese & Bruck, 1950) which found no evidence for phantoms in children under the age of 6 years has been used to support the view that the body image is a use-dependent phenomenon requiring years of sensory and motor experience to develop (Simmel, 1956, 1962a).

More recent studies have presented conflicting data and point to the alternate view that the body image experience may result from processing within genetically determined, hard-wired neural structures (LaCroix *et al.*, 1992; Melzack, 1989). Phantom limbs do occur in young children as well as in older individuals who underwent amputation in infancy or early childhood (Easson, 1961; Lacroix *et al.*, 1992; Poeck, 1964; Sohn, 1914). The evidence that phantom limbs are reported by individuals with congenital absence of limbs (Vetter & Weinstein, 1967; Weinstein & Sersen, 1961; Weinstein, Sersen, & Vetter, 1964) provides some of the strongest data to support the suggestion that the phantom limb represents the perceptual correlate of an innate neural substrate of the body experience (Melzack, 1989). Notwithstanding the absence of an intact limb from birth, as well as years of subsequent sensory–motor experience with the residual appendage, which arguably might lead to a reorganization of brain regions subserving the absent limb (Skoyles, 1990), the phantom limb is perceived to be remarkably similar to an intact limb.

With the exception of the study by Krane and Heller (1995), there is virtually no information on the incidence, quality of sensation, and time course of the nonpainful phantom limb in children. Krane and Heller (1995) found that 100% of their subjects reported phantom sensations, which they described most commonly as "tingling," "uncomfortable," "pins/needles," "tickling," and "itching." As with phantom limb pain, there was a ten-

dency for the sensations to develop within days of amputation and to decrease with time in a majority of subjects.

Impact of Phantom Limb Pain on Amputees' Lives

It has been known for some time that amputation frequently has a long-term negative effect on employment that is largely unrelated to decreased mobility and dexterity or to concurrent medical problems such as diabetes (Durance & O'Shea, 1988; Buijk, 1988; Millstein, Heger, & Hunter, 1986; Kegel, Carpenter, & Burgess, 1978). Several studies have not found a significant decrement in employment among younger amputees (Thyregod *et al.*, 1988; Purry & Hannon, 1989; Millstein, Bain, & Hunter, 1985). However, most of these studies did not look at relationships between pain and employment problems. In a review of the records of over 1000 people whose amputations were related to industrial accidents, Millstein *et al.* (1985) found that the presence of phantom pain was negatively correlated with successful employment. Parkes (1973) examined 46 amputees and found that 13 months after an amputation, the rate of employment was inversely correlated with severity of phantom limb pain.

As part of a survey, Sherman, Sherman, and Parker (1984) asked 5000 veteran amputees about the impact phantom pain had on their lives. The survey was structured so that the responses were clearly related to phantom pain rather than to stump pain or other debilitating factors. Phantom pain prevents 18% of those respondents who want to work from working and interferes with the work of 33.5% of those who are employed. The level of interference with those who are employed is significant in that 15% lose between 1 and 4 work days per month, 18% find that quality slips when they hurt, and 36% find it hard to concentrate because of the pain. We found that phantom pain interferes with sleep for 82% of the respondents, with most losing the equivalent of an hour per night or 7 hr per week. It stops 43% from social activities and 45% from carrying out normal activities. For those affected, the overall time lost is usually between 1 hr and 1 day per week. Thus, phantom pain has an important impact on an amputee's vocational activities.

Predisposing Factors to Experiencing Phantom Limb Pain

Preamputation Predictors of Persistence of Phantom Pain

No significant differences between those reporting and those not reporting phantom pain were related to the original cause of the amputa-

tion ($\chi^2 = 4.36$ with 4 df, $p = 0.359$). Forty-two percent and 46%, respectively, of the amputations were necessitated by direct combat injuries; 33% and 39% were for combat-associated problems; 18% and 19% were for accidents not related to combat; 7% and 6% were caused by disease. There were also no differences for the presence or absence of pain before amputation or years of pain prior to amputation. Preamputation familiarity with amputees was not a predictive factor ($\chi^2 = 6.98$ with 4 df, $p = 0.14$). There were no significant differences between the pain and nonpain groups related to age at amputation ($\chi^2 = 59.58$ with 58 df, $p = 0.42$). The mean age at amputation of our previous group was 24.9 ($\pm$ 5.6) and 25.79 ($\pm$ 1) years for our present group, both of which are very different from the age at amputation for most other amputees. As far as could be determined, the particular armed conflict that respondents had participated in did not correlate with the presence or severity of phantom pain. There is considerable disagreement in the literature on the relationship between pain before amputation and presence and description of postamputation pain. Jensen *et al.* (1985) found a relationship for 36% of their 58 patients immediately after amputation but for only 10% when the group was reinterviewed 2 years later. This decreased relationship with time could account for the differences reported in the literature, as Sherman *et al.* (1984) studied veterans whose amputations had occurred many years before the study, whereas many other studies were done with amputees just following their amputations. These relationships are discussed in depth in the chapters on mechanisms.

Postamputation Predictors of Persistence of Phantom Pain

The average number of years since amputation was 26 ($\pm$ 12.57) for amputees reporting phantom pain and 30 ($\pm$ 12.01) for those not reporting it ($F = 1.10$, $p = 0.245$). Many respondents' amputations had occurred only a year or so before receipt of the survey. Of those respondents who experienced phantom pain, 14% reported that their phantom pain had gone away, and 42% reported that it had gradually decreased over time. The remainder (44%) reported no change. Thus, phantom pain cannot be assumed to decrease gradually to a nonproblematic point. Age when surveyed was also not a major contributing factor in reporting phantom pain. Those reporting phantom pains were an average of 51.6 ($\pm$ 12.9) years old, and those not reporting it were an average of 56.2 ($\pm$ 12.7) years old. The mean age among respondents in our trial study was 51.4 ($\pm$ 13.4) compared with a mean of 52.7 ($\pm$ 13.1) for this group. Those reporting and those not reporting phantom pain were similar in their use of prostheses ($\chi^2 = 12.64$ with 8 df, $p = 0.13$). This lack of difference held for above- and below-the-knee/elbow and upper versus lower extremity comparisons. The presence of

stump pain correlated highly with reports of phantom pain in the previous study. This was also the case in the present study, as 66% of those reporting phantom pain also reported stump pain, whereas only half of those not reporting phantom pain reported stump pain. This difference is statistically significant ($\chi^2 = 42.1$ with 10 df, $p = 0.001$) and probably of actual clinical importance as well. Those reporting phantom pain also reported more frequent stump pain ($\chi^2 = 11.4$ with 3 df, $p = 0.009$). We are not aware of any differences in monetary compensation dependent on report of phantom pain. The amputation alone is the usual basis for amount of compensation.

Sensitivity and Reactivity to Pain Unrelated to the Amputation

If people reporting phantom pains are more sensitive or reactive to pain than those not reporting it, they might report as pain what the other group reports as painless sensations. We evaluated this possibility by requesting information on responses to common pains not related to the amputation. There was no difference in intensity of stomachache requiring medication ($\chi^2 = 13.4$ with 14 df, $p = 0.49$) or in hours waited before using medication for the stomachache ($\chi^2 = 0.59$ with 3 df, $p = 0.99$). The same lack of significant difference occurred for headache intensity requiring medication ($\chi^2 = 4.9$ with 15 df, $p = 0.11$) and for the time interval before taking medication for it ($\chi^2 = 1.87$ with 3 df, $p = 0.60$). Less well understood shoulder pain of longer duration was rated for pain intensity and for duration before going to a doctor and produced a similar lack of difference ($\chi^2 = 4.84$ with 7 df, $p = 0.68$). These measures of pain reactivity and sensitivity do not predict reports of phantom pain.

Phantom Body Pain following Spinal Cord Injury

Very little is known about the phantom body sensations experienced by people with spinal cord injuries. Bors (1951) interviewed 52 spinal-cord-injured patients about their phantom sensations. Seven of these were amputees. The lesions were diagnosed as complete in 43 of the patients. All of the patients reported experiencing phantoms with sensations described as burning, tingling, pins and needles (as though asleep), and tightness. A phantom penis was present in 19 of 49 patients, and phantom erections were reported by 14 of them. Twenty of the 38 patients questioned felt that they had voluntary control of the phantom's movement. The sensations appeared to emanate from virtually all parts of the body. Bors reported one patient who had phantom pain following an amputation 5 years before

his spinal cord injury. The phantom pain stopped after the spinal cord injury, but that the phantom remained similar if less distinct. The other six amputees in Bors's study had their amputations following their spinal cord injuries. All six reported that they could distinguish their amputation phantoms from their spinal cord injury phantoms in various ways. Pollock *et al.* (1951) reviewed 246 patients with "complete" transections and found that most experienced burning phantom pain. Nepomuceno *et al.* (1979) surveyed 356 spinal-cord-injured patients who had been hospitalized at their facility and received responses from 56% of them. Of these, 80% reported phantom sensations, with half of them reporting that the sensations were painful. Of these respondents, 25% rated the pain as extreme, and 44% said that it interfered with daily activities. Sweet (1975) reviewed the research on nonpainful phantom body sensations among spinal-cord-interrupted patients and found them to be similar to nonpainful phantom limb sensations among amputees.

Berger and Gerstenbrand (1981) had 37 spinal-cord-injured patients describe and sketch their phantoms. Thirty-five of the 37 reported phantoms of some kind, with 16 reporting nonpainful phantoms and eight reporting phantom pain. They reported that there might be a relationship between the site of the spinal cord lesion and the occurrence of phantom pain. Sherman, Ernst, and Markowski (1986; unpublished data) studied 35 patients with clinical diagnoses of complete transverse spinal cord tissue destruction who were interviewed about any sensations they felt below the level at which "normal" feelings were evident. All reported experiencing various feelings most of the time, and 33 reported that some of those sensations were usually quite painful. The descriptions usually centered on burning and tingling terms rather than cramping or shooting. The sensations were usually altered by sitting up for too long or other postural, compressive, or obstructive changes or environmental factors likely to affect blood flow patterns in the lower extremities. Typical descriptions and locations from this series of diagnostically complete spinal cord injured patients are summarized in Table 4.

The subjects in this series were not selected for reports of phantom body pain nor for any other criteria other than being the first available on the participating unit who met the study's criteria of clinically complete spinal injury. Thus, the results are likely to be typical of patients with spinal cord transections.

As noted in Table 4, very little is known about the frequency with which phantom body sensations among spinal-cord-injured patients are seriously painful. There have been no major objective studies of pain experienced by spinal-cord-injured patients below the level of "normal sensations," so the only information available comes from clinical experi-

Table 4. Descriptions of Phantom Body Pain Given by Men with Diagnostically Complete Spinal Cord Injuries

Subject	Age	Years since injury	Injury level	Description and location of pain
1	62	13	C5–6	Mild to moderate tingling in his inner left thigh and moderate spasmodic pain in the left lower quadrant of his abdomen
2	37	3	C6–7 and T11–12	Burning along the back of the left thigh and in three dime-sized irregular ovoids along the lower spine
3	41	11	C6	Mild bilateral tingling from his hips through his feet along all surfaces of the extremities and burning in his right palm
4	27	3 months	T7	Tingling in both hips and thighs and a "tight pants" sensation around his waist and hips
5	41	19	T10–11	Tingling along the inner surfaces of both upper thighs and an intense stabbing along a thin line starting at about 2 cm below the end of normal sensations at the left hip that extended down the front of the leg to his big toe
6	37	7	C7	Burning in the right lower leg and all surfaces of the right foot
7	51	11	L3 and T11–12	Burning pain running along a clearly demarcated strip that ran down the central anterior aspect of the left leg to the middle of the left foot
8	62	39	T10	Burning pain in the left buttock that was worst in a central circular area with pain gradually decreasing in a circular pattern as distance from the center increased
9	63	23	T10	Severe burning centered over the anterior iliac crest of the left hip with the highest level of the painful area starting several centimeters below the bottom of the zone where normal sensations faded away
10	43	10	T11	Burning pain in the top of the lateral superior aspect of the right outer thigh just below the hip

ence and a few clinical case studies. Pollock *et al.* (1951) reviewed 246 patients with "complete" transections and found that most experienced burning phantom pain. Many other workers reporting on their experiences with large numbers of spinal-cord-injured patients found that phantom body pain to be common and almost impossible to treat successfully on

follow-up (Bors, 1951; Freeman & Heimburger, 1947; Botterell, Callaghan, & Jousse, 1954; Davis & Martin, 1947). Sweet (1975) reviewed the research on nonpainful phantom body sensations among spinal-cord-interrupted patients and found them to be similar to nonpainful phantom limb sensations among amputees. Burke and Woodward (1976) reviewed the research on painful phantom body sensations and reported that significant phantom pain was found in 3–70% of patients, depending on the study. Sherman, Ernst, and Markowski (1986; unpublished data) studied 35 patients with clinical diagnoses of complete transverse spinal cord tissue destruction. All patients reported experiencing various feelings most of the time, and 33 reported that some of those sensations were usually quite painful.

Phantom Breast: Postmastectomy Pain

Bressler, Cohen, and Magnussen (1955a, 1955b) comment that although phantom breast sensations were among the first of the phantom phenomena reported in the medical literature (Ambroise Paré reported them in 1550) and were commented on again during the American Civil War (Weir Mitchell noted them in 1872), very little is written about phantom breast pain. Their work and review of the literature showed that about half the postmastectomy women interviewed reported phantom breasts but that none had spontaneously mentioned them to a doctor. An unsigned editorial (Editorial, 1979) in *The Lancet* reported that although the rate of phantom breast phenomena was between 10% and 64%, a mastectomy counselor in a British center stated that no patient had spontaneously complained of the symptom. Thus, phantom breast phenomena are probably similar to phantom pain phenomena in that patients are not likely to report them to their health care providers.

According to estimates from the National Hospital Discharge Survey for 1991, there were 216,000 women in the United States who were discharged from short-stay nonfederal hospitals with a diagnosis of malignant neoplasm of the breast (Graves, 1994). Postmastectomy pain occurrence is estimated at 4–6% (Foley, 1987). One etiological model posits that surgical injury to the intercostobrachial nerve during axillary node dissection is a potential mechanism for the pain. It is not known how many women suffering from "postmastectomy pain syndrome" (Stevens, Dibble, & Miaskowski, 1995) meet criteria for phantom breast sensations (PBS) in contrast to, for example, cicatrix pain. Others suffering more advanced disease may confuse the symptoms of PBS with those of other diffuse visceral and somatic pains. Nevertheless, some individuals experience

chronic pain affecting the anterior thorax, axilla, and/or medial upper arm beginning after mastectomy or removal of a lump. The pain is often described as burning and hyperesthetic. Sufferers may be unable to tolerate light touch, clothing, or a prosthesis. The pain is constant, may last for years, and is often unresponsive to conventional analgesics. Movements, particularly those of the arm on the affected side, often exacerbate the pain.

Besides pain, PBS is characterized by a variety of nonpainful sensations, which include itching, heaviness, and the perception that the removed breast is present. Wilentz (1991) reports that her review of the literature shows that "phantom breast pain was clearly distinguishable from, and unrelated to, pain or discomfort associated with the surgical scar. Most women described the phantom pain as: knife-like, pricking, shooting, sticking, squeezing, throbbing, burning, pressing, or crushing." Although a few women described the pain as constant, most described episodes lasting seconds, minutes, or hours. Pain was elicited by emotional distress, exercise, clothes, and weather, with relief provided by rest, touch, and heat. Thus, phantom breast pain is very similar in characteristics to phantom limb pain. There are, however, a number of potentially important differences as well. For one, the incidence of PBS tends to be considerably lower than that of phantom limb pain. Second, PBS and phantom breast pain are reported only in women; there have been no reports of the phenomena in men. Third, phantom breast pain tends to become more diffuse over time (Krøner, Knudsen, Lundby, & Hvid, 1992), whereas phantom limb pain becomes more localized to the distal portions of the limb. Finally, PBS occurs more frequently in younger women (Jarvis, 1967; Staps, Heogenhout, & Wobbes, 1985; Weinstein, Vetter, & Sersen, 1970), but age does not influence the development or experience of phantom limbs.

Physical examination of PBS and postmastectomy pain syndrome patients is negative. In a study of 95 women after mastectomy, 20% experienced chronic pain that was characteristic of a deafferentation syndrome. Age, years of education, ethnicity, marital status, employment status, and presence of a metastasis did not predict pain versus no-pain status (Stevens *et al.*, 1995).

The incidence of PBS, between a third and a half of postmastectomy patients who were sampled (Table 5), is lower than that reported for phantom limbs (Jensen & Rasmussen, 1986). However, there are several potential explanations for these rates. Underreporting may relate to fears of pejorative labeling. Some sufferers express apprehension that PBS will be construed as a psychogenic disorder. Simmel (1966) suggests that the absence of kinesthetic experience associated with bony joints accounts for the lower incidence. From the perspective of Melzack's (1993) genetically

Table 5. Phantom Breast Sensations

Authors	Year	Number of patients	Incidence of phantom breast sensation (%)	Incidence of painful phantom breast sensation (%)	Nonpainful sensations
Crone-Munzebrock	1950	49	53	54	Hanging, heavy weight, swelling, hardening
Ackerly, Lhamon, and Fitts	1955	50	22	18	Itching, tingling, heaviness, moving
Critchley	1955	30	10	—	—
Bressler *et al.*	1955b	25	64	31	Mild burning, itching
Simmel	1966	77	40	—	—
Jarvis	1967	104	23	33	Itching (62%)
Weinstein *et al.*	1970	203	34	24	Tingling
Jamison, Wellischi, Katz, and Pasnam	1979	41	53.7	41	Itching, soreness
Christensen, Blichert-Toff, Giersing, Richardt, and Beckmann	1982	31	35.5	—	—
Straps *et al.*	1985	89	33	15	Itching
Kroner, Krebs, Skov, and Jørgensen	1989	120	25.8	13.3	—
Stevens *et al.*	1995	95	—	20	Tingling, itching

built-in neuromatrix, physical maturation may be a factor. As with the adult dentition, the adult female breast emerges during adolescence, well after the establishment of the putative neuromatrix. More "ancient" structures may be more thoroughly represented than are the structures that emerge later in the history of the individual.

A review of the literature suggests that PBS and other postmastectomy pain conditions have received little attention. Research is needed to identify subgroups of patients, response to drugs, and improvement in surgical technique such as the role of the intercostobrachial nerve (Paredes, Puente, & Potel, 1990).

Orofacial Phantom Pain and Phantom Sensations

The face is a common site of tissue injury following trauma or infection. In addition, the face and mouth are the sites of many medical and dental surgical procedures that can alter the condition of tissue. Injured tissues sometimes exhibit allodynia (a pain response to a normally nonpainful stimulus) and hyperalgesia (an increased response to a stimulus that is normally painful, i.e., sunburn). Injured tissue may also display spontaneous pain. In addition, "normal," nondamaged tissue adjacent to the site of injury may display behavior that Dubner (1991) terms secondary hyperalgesia. Recent advances detailed in succeeding chapters provide evidence that the pain associated with peripheral tissue injury may result from such neural mechanisms as nociceptor sensitization, neuroma formation, and altered central nervous system (CNS) processing. These new findings have radically changed our understanding of the diagnosis and pathogenesis of orofacial phantom pain, stump pain, and phantom sensations. This section reviews the impact of current research on three orofacial conditions: phantom tooth pain, pain of edentate tissue, i.e., intraoral stump pain, and phantom sensations often manifested as a "bad bite" or malocclusion.

Orofacial pain is difficult to diagnose and treat, in part because of the multiplicity of pain-sensitive structures in the face (Fromm, 1991). Patients are often confused about care seeking. Dentists, otolaryngologists, and neurologists are all consulted for the diagnosis and treatment of oral and facial pain (Marbach & Lipton, 1978). The vast majority of orofacial pain conditions are conventional affairs. However, there are patients whose symptoms of orofacial pain are neither readily diagnosed nor routinely dispatched. Some patients who have elusive tooth and face pain suffer from orofacial phantom pain and allied conditions. This phenomenon presents an unusual challenge to clinicians.

Phantom Tooth Pain

Phantom tooth pain (PTP) is a syndrome of persistent pain in the face, teeth, and other oral tissues that may follow dental or surgical procedures such as pulp extirpations (root canal therapy), apicoectomy (root tip amputation), or tooth extraction. Surgery involving tissue adjacent to the teeth, such as exenteration of the contents of the maxillary antrum, also can result in phantom tooth pain. Interest in tooth pain of obscure origin is recent (Harris, 1974). The term phantom tooth pain was first used by Marbach (1978a) and has since been validated by others as a clinical entity (Rees & Harris, 1978; Reik, 1984; Brooke, 1980; Schnurr & Brooke, 1992; Pollmann, 1990; Marbach, 1993). A variety of other terms for phantom tooth pain such as atypical odontalgia, idiopathic odontalgia, and atypical facial pain are in use (Bates & Stewart, 1991).

Phantom tooth pain is characterized by persistent pain in endodontically treated teeth for which there is no evidence of pathology on physical and radiographic examination. Furthermore, neither repeated endodontic treatment, apicoectomy, nor tooth extraction render the affected area free of pain. Dental procedures and other surgical interventions such as trigeminal rhizotomy and microvascular decompression frequently exacerbate pain severity and may increase its distribution. Phantom tooth pain resembles other phantom pain syndromes that arise following amputation and injury to those peripheral nerves whose somatosensory pathways transmit pain sensation. Typical locations of phantom tooth pain are illustrated in Table 6, which presents the criteria and salient clinical characteristics of phantom tooth pain. Since it was first described (Marbach, 1978a), other researchers have corroborated many but not all of these features (Schnurr and Brooke, 1992; Pollmann, 1992).

Nonpainful Oral Phantom Sensations

Orofacial phantom sensations have been viewed traditionally as a psychiatric disorder (Marbach, 1976, 1978b). However, this position has been reinterpreted in light of recent research (Sherman, 1989a, 1989b; Jensen, Krebs, Nielson, & Rasmussen, 1984; Melzack, 1993). Brief experiences with sensations akin to phantom bite syndrome are common. Most people who have undergone dental treatment are familiar with the perceptual assessments associated with the final adjustments of even a single dental filling. The onset of the phantom bite syndrome is typically associated with extensive dental prostheses and orthodontic treatment. Others report that the initial onset was a single filling. They complain of continuous discomfort and are frequently distressed by their unfamiliar bite. The

Table 6. Criteria and Clinical Characteristics of Phantom Tooth Pain

1. The onset of pain is usually associated with an injury to a peripheral nerve. The injury often occurs in the course of routine dental and medical surgical procedures. Injuries also occur as the result of physical trauma to the face.
2. The onset of pain does not necessarily coincide with deafferentation at the tooth site. Pain may be delayed for days, weeks, months, or perhaps years.
3. The pain may endure long after healing of the injured tissues and spread to adjacent healthy tissue. Spreading can follow synaptic reorganization of an injured afferent nerve with resulting structural and functional changes in associated areas.
4. Phantom tooth pain is more likely to develop in patients who have sufferent pain in the tooth or face before the peripheral nerve section or endodontic treatment.
5. The pain is described as a constant, dull, deep ache with occasional spontaneous sharp pains. There are no refractory periods.
6. Sleep is undisturbed by pain. Many cases report a brief pain-free period on awakening. This period can last from seconds to about 1 hr.
7. Peripheral stimuli can momentarily exacerbate the pain but appear to have no prolonged influence. Percussion over the site of the injured nerve may result in Tinel's sign.
8. These stimuli can be of a type normally not nociceptive. There appears to be a lowered pain threshold (allodynia).
9. The pain is often worse at the site of the original trauma; although in chronic cases, patients have difficulty in localizing the pain, in part as the result of pain spreading to adjacent issues (secondary hyperalgesia). Additionally, precise localization of tooth pain is difficult. The treatment of neighboring teeth obscures the original condition. Nonpainful phantom phenomena also confound accurate perception and localization of the pain site.
10. Radiographic and laboratory tests are negative.
11. Without early intervention, the pain, once established, is often permanent.
12. Phantom tooth pain occurs in both sexes.
13. Phantom tooth pain has been reported in adults but not in children.
14. There is no evidence currently that phantom tooth pain is characterized by a premorbid personality. Whether affective states such as major depression are a cause or consequence of chronic pain remains to be determined.

care-seeking behavior of phantom bite syndrome sufferers often turns into a frustrating, expensive, and extended effort to restore the original bite (Marbach, 1978b).

Nonpainful nonocclusal orofacial phantom sensations are also sources of complaint (Marbach, 1978a; Pollmann, 1992). These sensations include nonspecific sticking sensations, a sensation similar to a wooden toothpick fragment splinter stuck in the gingiva. Others report an impression that certain teeth and oral soft tissues feel enlarged and also misaligned. It may be that those who suffer from both painful and nonpainful phantom phenomena are more likely to emphasize their pain symptom, so that other sensations go underreported.

Epidemiologic Considerations of Phantom Tooth Pain

Only one study could be found that gathered epidemiologic data on phantom tooth pain (Marbach, Hulbrook, Hohn, & Segal, 1982). The study yielded a rate of eight cases (3%) per 256 women who developed phantom tooth pain following endodontic therapy. Other cases met subthreshold levels (three of four criteria) for a diagnosis of phantom tooth pain. However, providing evidence of a genetic predisposition to deafferentation pain (Devor & Raber, 1990) trasferred the focus of attention from the individual tooth to the individual himself or herself as the unit at risk. It appears that a vulnerable person is at risk each time endodontic therapy is performed. Per capita endodontic treatment in the United States will likely increase with an aging population eager and able to afford the services. The rates of phantom tooth pain will also rise.

Phantom Sensations and Pain from Other Organs

Very little is known about phantom sensations and phantom pain from organs other than those discussed above. R. Davis' (1993) review of phantom sensations noted reports of pain in the tongue, bladder, rectum, and genitals as well as the areas discussed above. Dorpat (1971) theorized that only internal organs that normally generate "mental representations" are capable of giving rise to phantom phenomena. He proposed this theory to account for why phantom phenomena are reported from some organs and not others.

Uterus

Dorpat (1971) presented a case study of a 38-year-old married mother who, on the third day after a total hysterectomy, experienced menstrual cramps and labor pains. The pain was very similar in intensity and duration to menstrual cramps and labor pains she had previously experienced. In another case reported by Dorpat (1971), a 48-year-old single woman developed phantom uterine contraction pains following a total hysterectomy. She described the cramps as similar in quality, intensity, and duration to menstrual cramping pains.

Genitals

Davis (1993) noted that phantom sensations from the genitalia may include erections, pleasure, orgasm, or pain. Patients lacking organs will

often perceive the organ as present and attempt to urinate or ejaculate. Heusner (1950) reports the case of a 70-year-old man who had his penis amputated. Two to 4 years following the amputation, he felt an erect penis and described a penile ghost. These descriptions of a penile ghost abruptly ended when the patient suffered a gunshot wound to the spine and paraparesis. In another case report, a 52-year-old man who had his penis amputated felt a burning pain at the tip of the phantom penis. The rest of the organ felt as it had prior to the amputation.

Rectum

Ovesen, Krøner, Ørnsholt, and Bach (1991) interviewed all 22 surviving patients who had undergone complete excision of the rectum. Sixty-eight percent reported phantom sensations, and 27% reported phantom pain. Farley and Smith (1968) report that phantom rectal sensations are common after complete rectal excision. In a study of 50 patients, 34 reported having phantom pain. Twenty of the patients reported phantom rectal sensations, which included sensation of either flatus or feces. A study of 286 patients completed by Boas, Schug, and Acland (1993) found an 11.5% incidence of persisting perineal pain after rectal amputation. The overall incidence of pain in Boas's study is lower than that in some other studies because other studies looked for any phantom sensations, whereas pain was the specific criteria.

Stomach

Wangenstein and Carlson (1931) reported the case of a patient who continued to have periodic hunger sensations after a gastrectomy in which all the stomach and part of the duodenum were removed. The feeling of hunger was in the same location as when his stomach had been intact.

Bladder

Bors (1951) reported in a study with 50 spinal cord injury patients that in seven patients phantom bladder sensations existed and consisted of distension or micturition. Brena and Sammons (1979) reported one case that they felt was clearly a phantom pain problem. The case was a 38-year-old woman who had her bladder surgically removed for chronic cystitis. The patient likened the pain to "having a full bladder." The patient responded well to relaxation training. Most phantom urinary symptoms are not painful but resemble the nonpainful sensations typically associated with full bladders and micturation.

Relationships between Recognition of Phantom Pain's Actual Incidence and Development of Treatment Strategies

Correct recognition of the prevalence of a chronic pain disorder is a basic key to understanding its relationship to the patient's overall condition. If a disorder is thought to be very rare, considerable time will be spent looking for differences between the patient reporting the disorder and the mass of similar patients not reporting it. For example, as detailed above, until recently significant phantom limb pain was felt to occur among only 0.5–5% of amputees. Reports of the lower occurrence rates were based on the number of amputees who reported phantom pain without being asked, and the higher rates were based on studies in which the physician asked patients whether they had phantom pain.

Thus, when clinicians were approached by amputees requesting treatment for phantom pain, the obvious thing to do was to look for differences between other amputees and those reporting phantom pain. No obvious physical differences were ever located in clinical evaluations, and no comparative studies were preformed. However, as is frequently the case in chronic pain syndromes, if one looks hard enough one can find something in the body that at least appears to be out of the ordinary. For phantom pain, the answer was abnormalities in the stump such as formation of neuromas. The logic is that if the patient reports pain, there must be something physically wrong with the stump, with nerves or blood vessels related to the stump, or with the peripheral or central nervous system. If objective physical findings can not be demonstrated, the patient "must" be experiencing a psychological problem.

It is likely that most physicians confronted with rare cases of phantom pain have done their best to decide on a possible mechanism and then have tried any treatments that might ameliorate it rather than having to tell their patients either that they just did not know what to do or that it was "all in their head." The unproven existence of rare physiological abnormalities causing phantom pain leads directly to treatments based on correcting these differences between normal amputees and those reporting phantom pain. When no differences can be identified, or a long series of treatments do not work, psychological problems tend to be cited among those patients persistent enough to continue complaining of pain after experiencing numerous hunting expeditions for treatments.

Thus, a very skewed population of chronic pain patients tends to reach the behavioral clinician. They consist largely of those with whom the clinician does not get along and those who persist in complaining about their pain through numerous trials (Glickman, 1980; Hackett, 1978; Sher-

man, Ernst, Barja, & Bruno, 1986; Usdin & Lewis, 1979). These patients are characterized by persistence, rigidity, and unwillingness to tell the clinician what the clinician wants to hear (Parks, 1973; Sherman, Gall, & Gormly, 1979). In other words, they are the rare patients who tell the clinician when the treatment did not work instead of just trying another clinician or giving up and living with the pain.

Thus, their failure to understand that most amputees experience phantom pain causes clinicians to look for gross differences between "typical" pain-free amputees and "atypical" amputees, when the actual differences could have been found only by comparing amputees while they were in pain and not in pain.

Chapter 2

Phantom Pain as an Expression of Referred and Neuropathic Pain

M. Devor

The Potential Roles of the Different Portions of the Nervous System in Expressing Phantom Pain

The neural mechanisms that permit perception of phantom limbs have been investigated over many years (Melzack, 1989a; Sherman, 1989a, 1989b; Sherman, Arena *et al.*, 1990). A basic explanation of the underlying concepts is included in the attached amputee guide (Appendix II). A huge body of research has demonstrated that sensations reaching the brain are identified as to location on the skin by the homunculi in the sensory parts of the brain, including the somatosensory cortex, which contains several representations of the entire body surface. Thus, a pinch of the left index finger tip stimulates neurons in a location on the homunculi representing the left index finger tip. If the finger has been amputated, and the same signal is started by stimuli anywhere along the remaining nerve paths between the finger's stump and the homunculi, the resulting sensation seems to emanate from the finger tip. The real question is how these misleading impulses form and why the resulting sensation is frequently, but not always, painful. The relationships among phantom pain, phantom sensations, and possible changes in the homunculi are discussed in this and subsequent chapters.

In discussing the origins of phantom limb sensation, including phantom limb pain, most authors express one of three convictions: (1) that phantoms arise in the periphery, especially in the amputation stump, (2) that they arise in the central nervous system (CNS), particularly the spinal cord, and (3) that they are akin to hallucinations and arise in the

psyche. By evaluating clinical phenomenology in the light of recent neurophysiological data obtained from experimental nerve injury preparations, it becomes evident that each of these three plays a role.

The thesis developed in this chapter is the following. The neural activity underlying phantom limb sensation appears to originate primarily at ectopic sources in the periphery and, to a certain extent, in the CNS. These signals are amplified by central sensitizing mechanisms triggered by the nerve injury and by the ectopic discharge itself. By "ectopic discharge," we mean neural discharge that originates at an abnormal location, say along the length of a nerve, rather than at the normal location at the sensory nerve ending in the skin. The composite signal drives a high-order cell assembly whose activity defines conscious sensation. It is the properties of this central representation that determine the shape of the phantom percept, while the lower-level drive determines its intensity and sensory quality. Although all three levels play a role, the primacy and ready accessibility of peripheral nervous system (PNS) processes recommend them as the best targets for therapeutic intervention. One previously ignored PNS structure, the dorsal root ganglion, appears to make a particularly important contribution to phantom limb pain and ought to be exploited in the design of future therapeutic trials.

Where Is Sensation?

Perception of a limb, the "somatosensory psyche," is a consequence of the activity of neurons in one or more CNS representations of the body (homunculi). We refer to this assembly of cells as the "neural matrix of conscious sensation" (Melzack, 1989, 1993). Where are these neurons?

Direct electrical stimulation of the arm representation in the primary somatosensory cortex evokes a sensation felt in the arm, not one felt in the head (Penfield & Rasmussen, 1955). Likewise, in amputees, such stimulation evokes phantom limb sensation (Woolsey, Theodore, Erickson, & Gilson, 1979). However, the fact that primary cortical stimulation evokes recognizable sensation does not mean that the neural matrix of conscious sensation resides there. Sensation could reside in a subsequent, higher-order neural map, or it could be distributed in several cortical and/or subcortical regions that function in parallel. The same can be said of brain regions that drive the primary somatosensory cortex. Local stimulation of the ventrobasal thalamus also evokes sensation of the limb, as does stimulation of the dorsal column nuclei and the dorsal horn of the spinal cord (K.D. Davis, Tasker, Kiss, Hutchinson, & Dostrovsky, 1995). Moving into the periphery does not change the analysis. Stimulation of sensory endings

in the skin evokes sensation not because consciousness resides in the skin but because it ultimately evokes conscious sensory experience. The necessary conclusion is that understanding phantom sensation amounts to identifying those sources of neural excitation, active in amputees, that are most directly responsible for shaping the activity of the neural matrix of conscious sensation.

The perceptual body schema unquestionably resides in the brain (Melzack, 1989). If we can set off on a flight of fancy, let us imagine a disembodied brain soaking in a nutrient bath on a laboratory bench, still alive and awake. Without a retina, the individual inside probably experiences the room lights as out. That, however, shouldn't prevent his conjuring up detailed and colorful visual images or "experiencing" light when an electrical probe is applied to his optic nerve or visual cortex.

How would such an individual experience his body? One possibility would be the "absence" of body, the somatosensory equivalent of visual blackness. Alternatively, he may experience a "phantom" body, equivalent to the visual dream. In either event, electrical stimulation of the spinal cord or the somatosensory thalamus or cortex should trigger an overriding sensory experience, the somatosensory equivalent of stimulation-elicited light. The neural matrix of conscious sensation may have some intrinsic activity, but normally it is filled with content (activated) by afferent input originating from below. "Peripheral stimuli are the blood the sensory ghost must drink in order to be awakened to its phantom experience" (Gallinek, 1939). In an intact limb, "from below" generally means sensory receptors of peripheral nerve endings. In the case of amputation, the skin and deep limb tissues are gone. However, it cannot be concluded that the residual phantom sensation must be generated autonomously at the highest levels of perceiving. One needs first to consider all of the potential ectopic sources that lie between the missing limb and the neural matrix of conscious sensation.

Peripheral Nervous System Sources of Ectopic Neural Activity Underlying Phantom Limb Sensations

Laboratory investigations over the past decade or two (reviewed in Devor, 1994) have provided a wealth of information on potential ectopic sources of impulse initiation between the skin and the cortex. The cellular mechanisms responsible for this activity are also coming to be understood, information that will be essential in the development of more effective therapies (Devor, Lamazov, *et al.*, 1994b). Much of this information has come from animal models of nerve injury. As is to be expected, the data

derived from such models are more useful for investigating the parameters and mechanisms of the ectopic firing process than for asking questions about resultant sensation. However, the correlation between ectopic activity (ectopia) in animal preparations and sensations experienced by human amputees is so striking as to imply strongly that ectopia indeed underlies phantom limb sensation in man. In the few instances in which neural ectopia has been studied experimentally in amputees, the conclusions from the animal work have been largely confirmed (see below).

Ectopia Originating in Nerve-End Neuromas

Mechanosensitivity of Neuromas

The cell body of primary sensory neurons resides in a dorsal root ganglion, near the spinal column, and not in the limb itself. Therefore, limb amputation leaves a proximal nerve stump still connected to the dorsal root ganglion, the spinal roots, and the spinal cord. When the cut end of the nerve attempts to regenerate but cannot because its target tissue (the amputated limb) is gone, a nerve-end neuroma forms. When nerves are cut, neuromas always form. Despite many attempts, nobody has found a way to stop the abortive effort of the axon stump to regenerate. The question is only whether or not the neuroma that forms will be a source of paresthesias and pain.

It has been obvious since ancient times that pressing on a nerve-end neuroma often evokes paraesthesia and pain (Tinel sign). Direct recordings from neuromas in experimental animals have confirmed that ectopic discharge is indeed generated in neuromas during the application of mechanical force (Fig. 1; Wall & Gutnick, 1974a, 1974b; Devor, 1994). This has been confirmed in human amputees with phantom limb pain (Nyström & Hagbarth, 1981). The relationship of this evoked discharge to phantom sensation is straightforward. Pressure on the ends of stump nerves generates stump pain. Pressure on neuromas of nerves that used to serve the missing limb triggers or exacerbates phantom limb pain (Sunderland, 1978; Souques-Poisot, 1905; Henderson & Smyth, 1948; Kugelberg, 1946). Each burst of evoked ectopic discharge generated by percussion of the neuroma triggers a corresponding burst of phantom pain (Nyström & Hagbarth, 1981).

The additional sensation evoked by pressing on neuromas is, of course, distinguishable as a superimposed sensation (Henderson & Smyth, 1948). The preexisting background phantom derives from ongoing ectopic activity originating at various sources. The mechanosensitivity of neuroma endings, however, may contribute to this background. Potential causes of pressure internal to the stump include adhesions, edema, and muscle

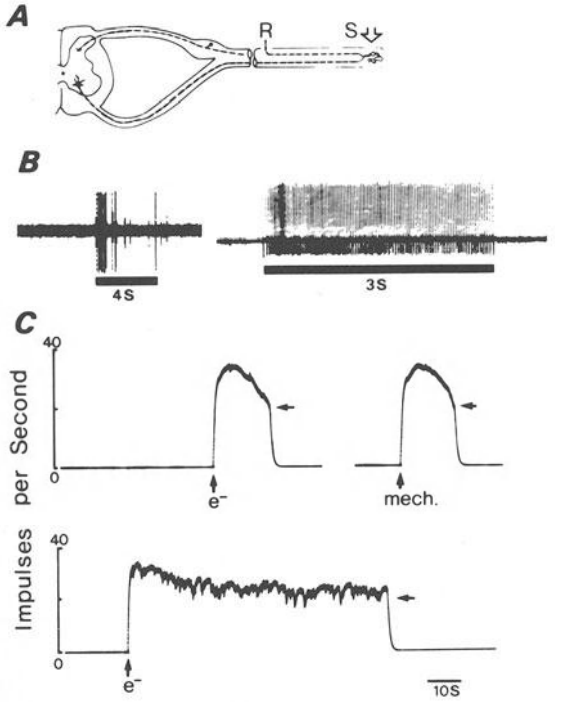

Figure 1. Abnormal mechanosensitivity of injured peripheral nerve axons. (A) Recordings were made from sensory axons (R) in chronically injured rat sciatic nerve (see Devor, 1994). (B) Many fibers responded to sustained displacement at the injury site (S) with a rapidly (left) or slowly adapting (right) spike discharge. (C) Some fibers responded with a prolonged discharge burst that long outlasted the momentary stimulus applied [electrical (e^-) or mechanical] (from Devor, 1994).

spasm. It is presumably on this basis that the reduction of stump muscle spasm reduces phantom limb pain (Sherman & Arena, 1992). External to the stump, of course, are obvious factors such as pressure from a poorly fitting prosthesis.

Ectopic mechanosensitivity is a property of the individual injured axon. It does not require axonal aggregates. When large numbers of axons become trapped within the bulk nerve end, an easily palpable neuroma is detectable. However, there may also be profuse, chaotic sprouting into the surrounding tissue. The consequent formation of disseminated micro-neuromas may present as general tissue sensitivity and not be recognized as a neuroma at all.

The development of ectopic mechanosensitivity is not a trivial process.

Modest pressure on an intact nerve trunk does not evoke a Tinel sign. Normal midnerve sensory axons are not mechanosensitive. The emergence of mechanosensitivity at ectopic midnerve neuroma sites requires a fairly complex alteration of the local electrical membrane properties of the injured nerve fibers. The specifics of synthesis, transport, and membrane incorporation of the proteins required to generate mechanosensitivity have only begun to be investigated (Devor, 1994).

Spontaneous Ectopia in Neuromas

Sensory fibers in nerve-end neuromas and disseminated microneuromas often have spontaneous impulse discharge unrelated to any discernible stimulus (Fig. 2; Wall & Gutnick, 1974b; Devor, 1994). The underlying physiology is closely related to mechanosensitivity but is not identical to it. Thus, not all spontaneously active axons are mechanosensitive, and not all mechanosensitive axons fire spontaneously. Spontaneous firing is expected to evoke an ongoing phantom sensation. The quality of the phantom, tingling or cramping, stabbing or burning, tonic or paroxysmal, must be related to the particular population of afferent fibers that happen to be firing spontaneously (e.g., low-threshold mechanoreceptors versus noci-

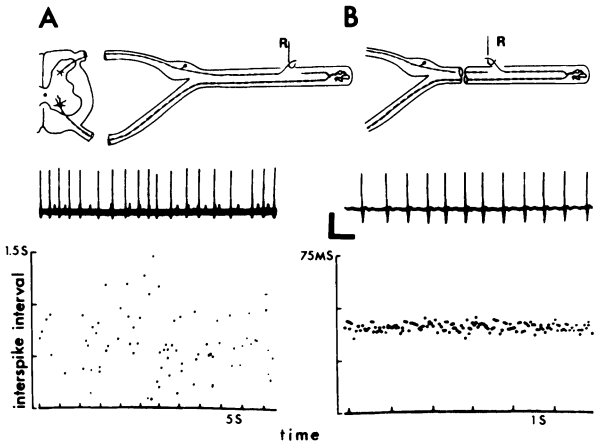

Figure 2. Spontaneous ectopic discharge is generated in chronically injured sensory neurons (see Devor, 1994). Alternative sources are the dorsal root ganglia (A) and the nerve injury site (B). The dot displays below the sample spike trains illustrate two of the most common firing patterns: slow, irregular (left, the most common pattern in dorsal root ganglia); and rapid, rhythmic, with highly regular intervals between consecutive impulses (right, most common pattern in neuromas).

ceptors) and to their dynamics. Electric shocks activate all afferent types simultaneously. Such activity occurring as a spontaneous paroxysm of ectopia is expected to feel like an electric shock.

A point frequently raised to undermine the role of neuroma pathophysiology in phantom pain is that neuromas take a long time to develop, where phantoms often appear "immediately" (hours or days). This argument reveals a fundamental misunderstanding of the functional properties of injured afferents. It is true that the massive tangle of sprouts described by pathologists takes weeks or months to form. However, spontaneous firing begins immediately on axonal division ("injury discharge") and, in some axons at least, never fades. Massive spontaneous firing is present within 3 days (Devor, 1994; Devor & Bernstein, 1982; Baik-Han, Kim, & Chung, 1990). Mechanosensitivity emerges within hours (Koschorke, Meyer, Tillman, & Campbell, 1991). The biological process responsible for neuroma ectopia appears to be related to axonal endbulb formation, which is rapid, rather than to the slower formation of a swollen bulk nerve-end neuroma (Fried, Govrin-Lippman, Rosenthal, Ellisman, & Devor, 1991).

Other Sensitivities

Phantom sensation is variable from individual to individual and often changes over time. So too are the degree and pattern of spontaneous and evoked neuroma firing. Moreover, both are influenced by a range of similar factors. For example, neuroma firing is often accelerated by sympathetic stimulation (specifically norepinephrine released from postganglionic sympathetic endings in the neuroma) and by circulating epinephrine. In both cases, the adrenergic agonist appears to act on adrenoreceptors in afferent endings in the neuroma (Devor, 1994). The expected sensory correlates—exacerbation of phantom pain during sympathetic activation caused by emotional stress, increased abdominal pressure (e.g., coughing), autonomic function, etc.—are well recognized (Sherman *et al.*, 1984; Jensen & Rasmussen, 1994). Interestingly, urination, defecation, and ejaculation, which involve activation of sympathetic efferents in the lumbosacral region exclusively, exacerbate phantom leg pain but not phantom arm pain (Haber, 1956). As expected, direct injection of adrenergic agonists into neuromas evokes intense phantom pain in human amputees (Chabal, Jacobson, Russell, & Burchiel, 1992). The relationship of phantom limb pain to activity in the sympathetic nervous system is discussed in greater detail in Chapter 4.

Neuroma endings may also develop sensitivity to a variety of other internal and external stimuli. For example, some classes of neuroma afferents (especially nociceptors) are sensitive to inflammatory mediators

(e.g., prostaglandins) that may be present in the stump (Devor, White, Goetzl, & Levine, 1992), and many are sensitive to ischemia and anoxia (Korenman & Devor, 1981). This is presumably the reason that decreased blood flow in the residual limb triggers burning phantom pain (Sherman & Arena, 1992; Chapter 6, this volume).

One of the unexpected ectopic sensitivities of unmyelinated neuroma afferents is to cold (Fig. 3; Matzner & Devor, 1987). This accounts for pain exacerbation during cold weather in patients living in northern climates and for the soothing effect of stump socks and other methods of warming the stump (Engkvist, Wahren, Wallin, Torebjork, & Nyström, 1985; Sherman *et al.*, 1984). The list of sensitivities of neuroma endings identified to date is already long (Devor, 1994), but it is unlikely to be complete. In effect, any depolarizing stimulus probably activates neuroma endings.

Interindividual Variability

The experience of phantom limb sensation and pain is unique to the individual amputee. This includes the quality and intensity of ongoing pain and peculiarities in the specific stimuli that exacerbate it. At least three factors associated with abnormal neural discharge probably contribute to interindividual variability.

(1) The amount of ectopia generated in neuromas depends on which nerve is involved, where it was cut, and how it interacts with surrounding tissue in the stump. There are also intrinsic differences in the likelihood that a given functional class of afferent will develop ectopic sensitivity and ectopic spontaneous firing. These are excitability variables associated with the injured sensory neuron itself (Devor, 1994).

(2) The degree of neuroma activity also depends on the presence of

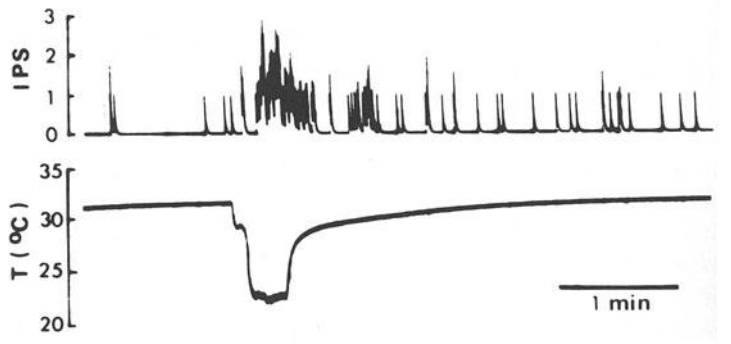

Figure 3. Cooling excites afferent C-fibers trapped at sites of experimental nerve injury. IPS, impulses per second. (From Matzner & Devor, 1987, with permission.)

exacerbating stimuli. Is there local inflammation? Is there good tissue oxygenation? What is the temperature outside?

(3) Finally, there is good evidence from animal preparations for a constitutional, genetically inherited predisposition for painful versus quiescent nerve injuries (Devor, 1994). Specifically, by selective breeding, it has proved possible to generate strains of animals that consistently show high or, alternatively, low levels of neuropathic symptomatology following a uniform nerve injury (Devor & Raber, 1990). Genetic predisposition may account for the elevated probability that, if one neuroma is a source of pain, others in the same individual will also be painful (Henderson & Smyth, 1948; White & Sweet, 1969). *A priori*, it is not unreasonable to expect that a particularly painful neuroma is "the luck of the draw" and that reamputation will deal a new hand of cards. Unfortunately, this logic does not usually apply in practice (White & Sweet 1969; Sunderland, 1978). The implication is that there is an intrinsic, individual predisposition to neuropathic pain in man as there is in animals (Devor & Raber, 1990; Mailis & Wade, 1994).

Failure of Treatments Aimed at Particular Exacerbating Factors

There are a multiplicity of factors that determine spontaneous neuroma firing, and a large variety of exacerbating conditions. Each of these vary from individual to individual and from time to time. Illegal parking will often elicit a traffic fine, but consistently feeding the meter will not protect you from a speeding citation. Likewise, removal of a particular type of exacerbating stimulus, e.g., by sympatholysis, may affect the annoyance of phantom pain during micturition, but it is unlikely to have a decisive effect on the overall level of pain. The multiplicity of independent exacerbating factors is a likely explanation of the failure of so many treatment modalities (Sherman, Sherman, & Gall, 1980; also see Chapter 8).

Failure of Neuroma Excision and Reamputation

As noted, neuroma ectopia develops rapidly. Therefore, excision of neuromas, or reamputation at a higher level, is not expected to provide more than temporary relief except in those selected cases in which a particular exacerbating stimulus, usually mechanical, was dominant. The original, constitutionally determined pathophysiology responsible for spontaneous neuroma firing simply reestablishes itself. Indeed, pain is often more severe after reamputation because the original level of nerve injury is closer to the dorsal root ganglia and because additional, proximal nerve tributaries are now involved.

Is ectopic discharge originating in neuromas in fact a major source of the neural activity that generates phantom limb sensation and phantom limb pain? This question ought to be easily answered. As noted above, mechanical, chemical, and electrical stimulation of neuromas evokes sensation referred into the phantom. However, to establish the role of neuromas in creating the baseline phantom, one needs to know whether silencing the relevant nerves with a diagnostic block well central to the stump (e.g., brachial or lumbar plexus block) causes the phantom and its pain to vanish. Unfortunately, the published literature provides distressingly contradictory opinions on this issue. There are three common sources of confusion.

(1) One needs assurance that the diagnostic block was complete. Specifically, one needs to know whether the Tinel sign evoked from the major stump neuromas is gone. There can be no doubt that pain resulting from percussion of a neuroma indeed reflects ectopic impulses originating in the neuroma. The question mark is only with respect to spontaneous phantom pain. Unfortunately, this information is rarely provided.

(2) If the phantom "persists," one needs to know if this is the original phantom or a new, qualitatively different one. Nerve block in intact limbs consistently yields a phantom sensation if the block is complete (Simmel, 1962b; Melzack & Bromage, 1973). This is most commonly experienced with dental anesthesia. The lip may be totally insensitive, but one does not feel a "hole" in one's face. Rather, there is a "numb" sensation, usually of a swollen lip. This is a phantom. The source of neural activity underlying such so-called "normal phantoms" is presumably central to the block, perhaps the dorsal root ganglia (see below). Alternatively, they may result from the release of spinal neurons from ongoing inhibition by normal low-threshold afferent input (Wall, 1981). Indeed, such disinhibition could contribute to true amputation phantoms, although probably not to phantom pain, as "normal phantoms" are never painful. The diagnosing physician needs to determine whether, in the presence of nerve block, the patient is feeling a "normal phantom" or his original idiosyncratic phantom.

(3) Local anesthetics cannot be relied on to block the propagation of nerve impulses for more than some tens of minutes. Recovery from the block is signaled by return of the Tinel sign. As a practical matter, pain relief sometimes long outlasts the expected duration of the block. A possible reason is movement of the local anesthetic to the source of ectopic firing. The process of impulse generation is far more sensitive to block than that of impulse propagation (Devor *et al.*, 1992; Devor, Lomazov, *et al.*, 1994b).

Nerve blocks, even if repeated many times, cannot be expected to produce long-lasting relief. Nonetheless, many authors register as "fail-

ures" trials in which one or a few blocks failed to cure pain. Their conclusion that the neuroma cannot be a prime source of pain is patently absurd. If the pain stopped even for a few minutes during the block, this is good evidence of a peripheral source. The provision of lasting relief requires development of ways to eliminate ectopia on a long-term basis.

In the experience of many physicians, phantom limb sensation and pain are temporarily stopped by nerve block, or at least substantially reduced, in at least 90% of amputees. Part of the residual 10% remain in question because of uncertainties as to the completeness of the block. Nonetheless, it is also widely believed that occasionally, phantom pain persists despite satisfactory block. Unfortunately, controlled, quantitative data on this point do not appear to be available. In their absence the figure <10% serves as a tentative starting point in considering more central sources of ectopia.

Ectopia Originating in the Dorsal Root Ganglion

Animal Studies

Animal experimentation has shown that dorsal root ganglia associated with an injured nerve are a second major source of spontaneous ectopic discharge (Fig. 4A; Wall & Devor, 1983; Burchiel, 1984; Kajander, Wakisaka, & Bennett, 1992). Indeed, even in the absence of nerve injury, a

Figure 4. Hypertonic (6%) saline solution excites ectopic impulse discharge in dorsal root ganglia associated with an injured nerve. (A) Baseline spontaneous firing originating in a lower lumbar dorsal root ganglion (L4 and/or 5) in a rat whose sciatic nerve was subject to a constriction injury 7 days earlier. (B) Topical application of 6% saline accelerated the firing and recruited several previously silent afferents. The record was taken about 20 min after application of the 6% saline. All active units were C-fibers.

low level of ongoing activity is generated in the dorsal root ganglia. This may form the basis for the "normal phantom" experienced during nerve block in intact limbs. In the presence of nerve injury, recordings of ectopic firing from dorsal root axons central to the dorsal root ganglion show that both the neuroma and the dorsal root ganglion make a significant contribution. Individual axons may show a dual source (Kirk, 1974; Wall & Devor, 1983).

Just as in the neuroma, ectopic activity originating within dorsal root ganglia is exacerbated by mechanical, physical (e.g., temperature), chemical, and metabolic variables, particularly when the associated peripheral nerves have been injured (Devor, Lomazov, *et al.*, 1994). Sympathetic efferent activity and circulating epinephrine, for example, affect dorsal root ganglion ectopia (Burchiel, 1984; Devor, Jänig, & Michaelis, 1994).

Human Studies

Dorsal root ganglion ectopia has not been sought specifically in neurographic recordings in humans. However, while recording from nerves central to a neuroma in patients with phantoms, Nystöm and Hagbarth (1981) noted that anesthetic block of the neuroma eliminated the Tinel response but failed to eliminate much of the ongoing nerve activity. It is likely that this persistent activity originated in the dorsal root ganglia and propagated outward to the recording electrode.

Another specific indication of dorsal root ganglion involvement in phantom limb sensation comes from studies by Feinstein, Luce, and Langton (1954), who injected hypertonic saline (6%) into the interspinous tissue in normal volunteers. This stimulus evoked transient pain in the corresponding dermatome. Identical stimulation in amputees rapidly (within seconds) evoked a natural painful phantom limb sensation and "filled out" phantoms that had faded with time postamputation and become incomplete. In animal preparations, axons do not fire on topical application of 6% saline. Dorsal root ganglion neurons, on the other hand, do (Fig. 4). Thus, Feinstein *et al.* (1954) were probably activating the dorsal root ganglia nearest to their injection needle. The exacerbation of the phantom was often followed by its disappearance for a time, an effect expected from the postactivation refractoriness of dorsal root ganglion neurons (perhaps from prolonged activity-dependent afterhyperpolarization; Amir & Devor, 1995). Intraspinous injection of procaine caused phantom pain and paresthesias to decrease in intensity, although usually not to disappear completely. Combined suppression of ectopia from several neighboring dorsal root ganglia and from associated neuromas is probably necessary to completely silence the phantom.

Amplification and Cross-Excitation in Dorsal Root Ganglia

Ectopia in the dorsal root ganglia can amplify afferent signals that originate in stump neuromas and in normal sensory endings in the stump. One such amplification process is evoked dorsal root ganglion afterdischarge. A dorsal root ganglion neuron that is silent but on the threshold of firing might be nudged into a firing mode by spike activity arising in the stump (Devor, 1994). A second such amplification process is dorsal root ganglion cross-excitation. It has recently been established that activity in one population of dorsal root ganglion neurons tends to depolarize and excite neighboring neurons that share the same ganglion (Devor & Wall, 1990). Exacerbation of dorsal root ganglion ectopia by sympathetic efferent activity was noted above (Devor, Jänig *et al.*, 1994). The net effect of these amplification processes is to augment the impulse barrage flooding the CNS.

If phantom limb sensation were generated within dorsal root ganglia or within neuromas and dorsal root ganglia, nerve block would not stop it. Thus, among the phantoms that are legitimately spared by nerve block, some, and perhaps all, probably have a dorsal root ganglion component. To date, the dorsal root ganglion has rarely if ever been excluded as a possible source of the ectopic discharge underlying phantom limb sensation. For this reason it is impossible to estimate the proportion of cases in which the neuroma is not essential and a CNS source needs to be invoked.

Central Nervous System Sources of Ectopic Neural Activity Underlying Phantom Limb Sensation: Why Doesn't Dorsal Rhizotomy or Ganglionectomy Work?

There have been many attempts to eliminate neuropathic pain by surgical interruption of the dorsal roots (dorsal rhizotomy) or by excision of the dorsal root ganglia. Both types of surgery almost always provide relief for a short time, but pain returns after weeks or months despite maintained, total anesthesia of the stump and elimination of the Tinel sign (White & Sweet, 1969). Thus, as a practical matter, rhizotomy is ineffective except when the expected lifetime of the patient is short. Investigators who favor CNS models of phantom limb pain point out that if abnormal impulse activity associated with neuromas, dorsal root ganglia, or any other PNS source were responsible for the pain, then rhizotomy or ganglionectomy should provide definitive relief.

Compelling as this argument appears at first glance, it is misleading. Indeed, the mere fact that rhizotomy usually does relieve phantom pain for a time constitutes prima facie evidence for a peripheral source of the

underlying ectopia. The question is why pain returns. In intact limbs, even when it is beyond doubt that a painful source is in peripheral tissue, deafferentation rarely provides lasting relief. Rhizotomy does eliminate pain derived from the peripheral tissue. However, this original pain is replaced by a new pain, "deafferentation pain," triggered by the rhizotomy itself. It is the eventual emergence of deafferentation pain that renders rhizotomy and ganglionectomy an ineffective clinical strategy. Deafferentation pain is a separate phenomenon whose emergence cannot be taken as evidence that the original pain source lay within the CNS.

If phantom limb pain were generated primarily in the CNS, then rhizotomy should not relieve it even temporarily. To stretch credulity somewhat, it could be argued that the central generator of phantom pain is somehow suppressed by rhizotomy and that it emerges once again when this suppression fades (diaschisis). In principle, it should be possible to detect whether a postrhizotomy phantom is or is not a novel sensory event. Specifically, if the phantom derived from ectopia in the neuroma and dorsal root ganglion, its sensory details (burning, shooting etc.) should change following rhizotomy. If the postrhizotomy phantom were qualitatively identical to the original phantom, this would hint at a CNS source. Unfortunately, such detailed sensory analysis is rarely reported. Moreover, even if it were, it would not necessarily be conclusive. At least some aspects of sensory quality are determined high in the CNS, within the neural matrix of conscious sensation (see below). If so, this latent percept might be kindled equally well by afferent drive from the neuroma or dorsal root ganglia and from the spinal cord.

Abnormal Discharge Originating in the CNS— The Dorsal Horn of the Spinal Cord

Dorsal rhizotomy may trigger elevated spontaneous firing in the dorsal horn, including activity with an unusual bursting pattern. Neural activity of this sort, which has been documented both in animals and in man, is thought to contribute to deafferentation pain (Loeser & Ward, 1967; Loeser, Ward, & White, 1968). Similar activity also occurs following nerve injury, and it may thus contribute to postamputation phantom sensation. In the later case, however, it is necessary to exclude the possibility that the increased central activity is not simply secondary to peripheral activation (Sotgiu, Biella, & Riva, 1994).

Traumatic avulsion of dorsal roots in the absence of amputation triggers some of the most severe cases of phantom limb pain (Wynn-Parry, 1980). An often effective remedy is destruction of the dorsal horn by means of dorsal root entry zone surgery (Nashold & Ostdahl, 1979). This implies

that ectopic firing in the deafferented dorsal horn is a primary source of pain in these patients. The dorsal root entry zone operation may also be useful for postamputation pain (Saris, Iacono, & Nashold, 1988; but see the discussion of the reliability of this approach in Chapter 8). This, result, however, is consistent with a PNS origin of the underlying ectopia as well as an intrinsic spinal origin.

Supraspinal Representations: Phantom Body Pain in Paraplegics

An often quoted piece of evidence in favor of ectopic sources in supraspinal CNS structures is the existence of "phantom body" sensation in patients with (clinically) complete spinal cord transection (Melzack, 1989). Although the reality of this phenomenon is not in question, its relevance to postamputation phantoms remains tenuous. The reason is the same as that concerning dorsal rhizotomy. Even in those rare cases in which an amputation phantom preceded the spinal cord injury (Bors, 1951), it is generally impossible to know whether one is dealing with persistence of the original phantom or its replacement by a new one. In general, phantom sensation in paraplegics lacks the clarity and immediacy of amputation phantoms (Weinstein, 1969). As discussed later in this volume, phantom pain in patients with "clinically complete" injuries of the spinal cord may still react to some degree to stimuli in the periphery (Sherman, Ernst, & Markowski, 1986). This may mean that the spinal cut was not, in fact, complete. Alternatively, some other residual sensory conduction pathway may have been present. One such possibility is pain transmission along the sympathetic ganglion chain, with entry into the spinal cord along dorsal roots above the level of the spinal injury (Sherman, Ernst, & Markowski, 1986).

Anterolateral cordotomy (transection of the anterolateral white matter of the spinal cord) usually eliminates phantom pain in amputees for a time. Pain returns within 6 months in about 50% of cases, however, and within 3 years in 80% (Siegfried & Cetinalp, 1981; White & Sweet, 1969). As for cordotomy in the treatment of pain in general, the return of phantom pain reflects either the emergence of a new phantom of central origin or the uncovering of an alternative spinal conduction pathway.

Presumably abnormal bursting neural activity has been reported in the somatosensory thalamus (ventrobasal complex) and cortex following nerve injury, dorsal rhizotomy, and spinal cord injury, in both animals and man (Lenz, *et al.*, 1987; Lombard, Nashold, & Pelissier, 1983; Albe-Fessard & Lombard, 1983; Guilbaud, 1991; Dougherty & Lenz, 1994). Such discharge might underlie phantoms of supraspinal origin. Infrequently, supraspinal lesions along the central somatosensory conduction pathways have been

shown to trigger phantom limb pain. For example, Baron and Maier (1995) reported recently on a patient with a traumatic medullary infarction located along the spinobulbothalamic tract on the left side. In addition, the right leg was amputated. Phantom limb pain was present, and it was exacerbated by stimuli applied to the stump. Epidural block eliminated the stump sensitivity, but the phantom itself persisted. Perhaps, its origin was at the site of infarction.

Electrical stimulation of the cortical limb representation in amputees can evoke phantom sensation, including pain (Woolsey *et al.*, 1979). Likewise, there have been several reports of sudden relief from phantom pain following surgical lesions of the somatosensory cortex or spontaneous infarction (Woolsey *et al.*, 1979; Yarnitzky, Barron, & Bental, 1988; Appenzeller & Bicknell, 1969). This observation suggests either a subcortical ectopic source or one intrinsic to the affected cortex. In principle, abnormal firing subserving phantom limb sensation might arise anywhere along the somatosensory projection pathway, including within the neural matrix of conscious sensation itself.

Interaction among the Sources of Neural Activity Underlying Phantom Limb Sensation

Central Sensitization and the Dual Effect of PNS Ectopia

Ectopic discharge from neuromas and dorsal root ganglia contributes to phantom limb pain in two different ways. Most obviously, it directly drives central transmission neurons and hence evokes sensation. There is also a second, indirect mode. Specifically, it is now known that C-fiber input from peripheral tissue can trigger a unique spinal hyperreactive state called "central sensitization" (Devor *et al.*, 1991; Woolf, 1992). In the presence of central sensitization, a touch input is felt as pain (secondary hyperalgesia; Hardy, Wolf, & Godell, 1952; Campbell, Raja, Meyer, & MacKinnon, 1988; Torebjork, Lundberg, & LaMotte, 1992; LaMotte, Shain, Simone, & Tsai, 1991). Ectopic nociceptive input from injured nerve branches can likewise trigger central sensitization (Gracely, Lynch, & Bennett, 1992).

Fortunately, central sensitization has only a short half-life, fading within one or a few hours. For this reason, the secondary hyperalgesia triggered by acute injuries only briefly outlasts the instigating peripheral noxious drive. Unfortunately, in the presence of a continued nociceptive input such as from neuroma or dorsal root ganglion ectopia, central sensitization can apparently be maintained (refreshed) indefinitely. In an intact limb, this occasions an extended zone of ongoing pain and (secondary)

hyperalgesia (Gracely *et al.*, 1992). After amputation, the expected outcome is pain amplification, with phantom limb pain aroused by ectopic A-fiber activity, as well as by C-fiber ectopia. In addition, central sensitization could yield (secondary) hyperalgesia on the stump (Jensen & Rasmussen, 1994).

The synaptic mechanism of central sensitization is currently under detailed scrutiny. It apparently involves the transient engagement of a particular class of glutamate-sensitive membrane channels that have a significant Ca^{2+} conductance (NMDA receptors; Fig. 5; Woolf, 1992). On these grounds, there is hope that NMDA receptor antagonists might form a new class of analgesic drugs. Theoretically, central sensitization could play a major role in the generation of phantom limb pain and stump hyperalgesia, at least in some patients. As in intact individuals, its role can be evaluated by determining to what extent activation of A-afferents in nerves that used to serve the extremity in fact exacerbates phantom pain.

Long-Term CNS Changes following Nerve Injury

Peripheral nerve injury triggers persistent CNS amplifications over and above the NMDA receptor-mediated central sensitization noted above. The mechanism and clinical relevance of these effects, however, are uncertain. It is now well established that nerve injury induces substantial metabolic changes in the axotomized primary afferent neuron in the dorsal root ganglia and also in postsynaptic neurons in the CNS. For example, in dorsal root ganglion neurons, the synthesis of some proteins is up-regulated, and that of others is down-regulated (Hokfelt, Zhang, & Wiesenfeld-Hallin, 1994). Similar if less dramatic changes in gene expression occur in the spinal cord (Dubner & Ruda, 1992). There are a number of correlated, and perhaps consequential, functional (synaptic) changes in the CNS. These include the collapse of presynaptic inhibition (Wall & Devor, 1981) and the rewiring (including expansion) of somatosensory receptive fields (Devor & Wall, 1981).

It is widely assumed that these functional changes underlie some aspects of chronic neuropathic pain conditions, probably including phantom limb pain, but the precise links remain tenuous and speculative. For example, it has been proposed that in the course of synaptic rewiring, low-threshold afferents come to be functionally connected to ascending spinal projection neurons that signal pain (Devor, 1988). Another speculation is that the rapid neural discharge generated by the acutely injured peripheral tissue brings about the excitotoxic death of inhibitory interneurons in the spinal cord (Wilcox, 1991; Dubner & Ruda, 1992) and, hence, a hyperexcitable, disinhibited spinal cord. If so, then preemptively blocking the acute

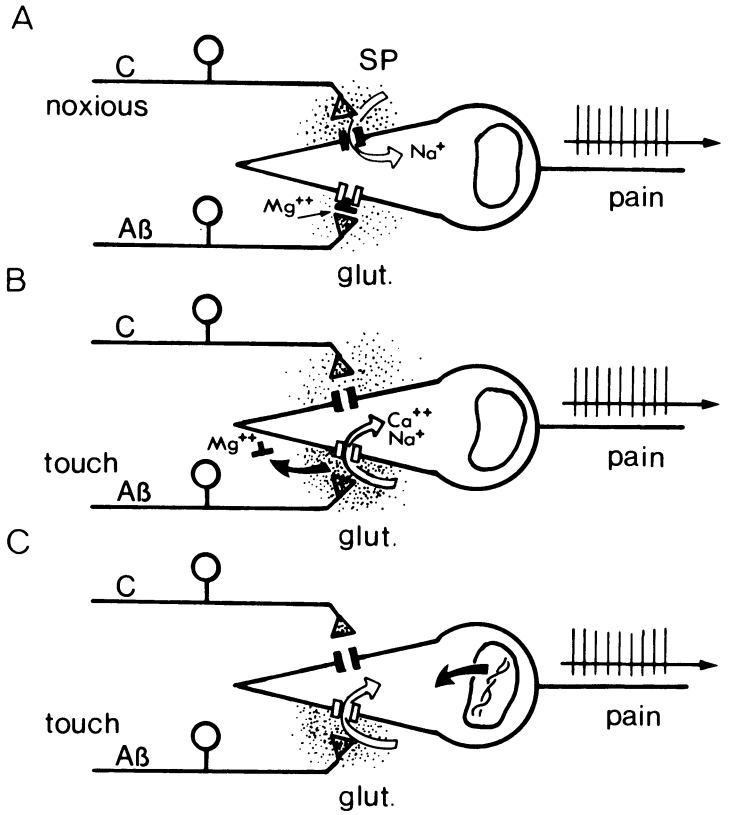

Figure 5. Proposed "central sensitization" mechanism for triggering touch-evoked pain. (A) Normally, activity in peripheral C-nociceptors activates spinal multireceptive neurons by means of excitatory amino acid and peptide neurotransmitter(s), probably including substance P(SP). This triggers an ascending pain signal. Touch input, carried along low-threshold Aβ afferents, evokes release of the neurotransmitter glutamate (glut.). However, this drives the spinal neurons only minimally because the NMDA-type glutamate receptors on the postsynaptic dendrites are blocked at normal membrane potentials by Mg^{2+} ions. (B) Intense noxious C input produces prolonged (tens of seconds) SP-evoked depolarization. This displaces the Mg^{2+} block, enabling the NMDA receptors. Subsequently, glutamate released from Aβ touch afferents strongly activates the multireceptive neurons and hence evokes pain (allodynia and hyperalgesia). Calcium entering the spinal neurons through the enabled NMDA receptor channels may trigger phosphorylation of channels (through activation of a Ca^{2+}-dependent protein kinase), sustaining the touch-evoked pain state for hours (Woolf, 1992). (C) More speculatively, a change in gene expression triggered by tissue or nerve injury could prolong the central sensitization state indefinitely. (Reprinted from Devor, 1996, with permission.)

"injury discharge" associated with the amputation surgery might prevent the later development of chronic pain. There is some suggestive evidence to this effect in animal preparations (Dennis & Melzack, 1979; Coderre & Melzack, 1987; Katz, Vaccorino, Corderre, & Melzack, 1991; Coderre, Katz, Vaccarino, & Melzack, 1993; Seltzer *et al.*, 1991; Gonzales-Darder, Barbera, & Abellan, 1986) but only two relevant studies concerning human amputees (Bach, Noreng, & Tjéllden, 1988; Jahangiri, Bradley, Jayatunga, & Dare, 1994). Extensive epidural analgesia prior to, and for a time following, amputation led to a lower than expected incidence of phantom pain on follow-up 6 and 12 months later. Unfortunately, however, both studies suffer from too small a sample size, too short a follow-up time and not enough explanation of how the presence of phantom limb pain was assessed. For the present, these studies must be viewed with caution. The subject of measures that might be taken at the time of amputation to prevent the later development of phantom limb pain is discussed in Chapters 5 and 8 of this volume.

"Centralization" of Phantom Limb Pain

There is a long and firmly held belief in the clinical literature that chronic neuropathic pain, including phantom limb pain, may begin in the periphery but that it eventually "burns its way" into the central nervous system and becomes independent of peripheral sources. This is sometimes called pain "centralization." The fact that peripheral injury and injury-related sensory signals may alter central gene expression has given new impetus to this idea. The belief in centralization has several clinical underpinnings, but their interpretation is a subject of controversy. For example, it is often pointed out that pains not resolved early tend to be refractory to later treatment. On the face of it, this observation suggests that long-standing pains do get "burned" into the brain. However, a moment of reflection shows this argument to be circular. After all, those pains that are intrinsically refractory are the very ones most likely to persist despite multiple treatments and the passage of time.

Other often-quoted evidence of centralization is the reported sensation in phantom limbs of preamputation details such as rings, bunions, focal pains, etc. (Cronholm, 1951; Parkes, 1973; Henderson & Smyth, 1948; Katz & Melzack, 1990; Haber, 1956). Similarly, the position of the phantom is thought often to reflect the last position of the limb before amputation. These are examples of "phantom pain memories."

In this volume we consider two alternative approaches to the issue of phantom limb memories. In the present chapter, we take the position of the skeptic, arguing that phantom memories may be largely wishful thinking,

or, if they are real, that they resemble conventional memories conjured up by pain originating in peripheral ectopic sources. In Chapter 5, on the other hand, we take the opposite stand. There, we argue that phantom limb memories reflect true sensations generated in a portion of the brain that was imprinted by pain of the limb before it was amputated. The reader is invited to chose his preferred side or to remain agnostic on the matter.

When an amputee reports, say, that he feels on his phantom finger the ring that he used to wear, should his report necessarily be taken at face value? Such anecdotes might reflect specific sensations centralized from years of wearing the ring, but they might also result from memory of the way the ring felt, aroused, say, by new tingling sensations associated with stump neuromas or ectopic dorsal root ganglion activity. These actual sensations might "anchor" the belief that it is the ring that is felt, a belief that might also be held by the attending physician/researcher, who might provide subtle or not-so-subtle prompting. The same problem holds for phantoms that "persist" despite dorsal rhizotomy or spinal transection. As noted above, these events trigger (new) deafferentation and central pains that could be rationalized by the patient and his physician as continuation of an old pain that is in fact gone. Carefully controlled prospective studies are needed to resolve this issue.

Jensen *et al.* (1985) made the first effort in this direction. In their cohort, virtually all of the patients had limb pain prior to amputation. Immediately after amputation, phantom pain resembled the preamputation pain in location and character in only a third, and after 2 years in only 10%. Wall, Novotny-Joseph, and MacNamara (1985) found no relationship between the quality and location of phantom pain and pain preceding amputation among 25 lower extremity amputees who had their surgeries because of cancer. Sudden traumatic amputation of previously healthy limbs does not appear to yield results very different from amputation after extended periods of pain (Sherman & Sherman, 1985; Carlen *et al.*, 1978), but this matter has not been studied in a sufficiently systematic manner.

The mere fact that in most amputees nerve or spinal block transiently suppresses phantom pain indicates that the generator does not become independent of the periphery even when the pain has lasted for years. A similar conclusion can be drawn from studies of chronic nonphantom pain in patients and in animals. For example, a patient described by Gracely *et al.* (1992) had a localized scar that proved to be the primary source of a long-standing neuropathic pain. This source triggered A-fiber touch-mediated hyperalgesia over a large part of the limb, presumably as a result of central sensitization. Within seconds or minutes of local anesthetic block of the primary ectopic source, the widespread tenderness disappeared, and it returned immediately as the block wore off. Likewise, despite years of

prior arthritic pain, total hip replacement surgery generally provides rapid relief, with no sign of pain having been centralized. Sherman *et al.* (1992a, 1992b) have reported numerous cases of long-standing cramping phantom pain being entirely cured within minutes of patients learning to associate cramping sensations in their phantoms with spikes in the EMGs of the residual limb and subsequently learning to control the spikes.

We are not aware of any reliable animal studies in which a prolonged acute pain was shown to become centralized. On the other hand, several studies have demonstrated an effect of prior acute pain on the later development of chronic neuropathic pain (reviewed by Coderre *et al.*, 1993; also see Chapter 5). In a typical example, Dennis and Melzack (1979) induced acute pain with intradermal formalin and soon afterward deafferented the limb by dorsal rhizotomy. The resulting autotomy, a behavioral indicator of ongoing neuopathic pain, was more severe than when formalin was applied after the deafferentation. Autonomy behavior may even be directed at the specific part of the foot that was subjected to the prior pain stiumulus (Katz *et al.*, 1991).

In another set of animal experiments of possible relevance to the issue of phantom pain memories, local anesthetics were used to block hindlimb nerves before cutting them. This attenuated the later development of autotomy (Gonzalez-Darder *et al.*, 1986; Seltzer, Beilin, Ginzburg, Paran, & Shimko, 1991). The interpretation offered by the authors was that blocking the "injury discharge" generated when the nerves were cut avoided persistent central sensitization, or excitotoxic disinhibition and hence prevented the emergence of abnormal CNS amplification.

Although these studies are thought provoking, it is risky to draw conclusions about centralization from them. In the animal studies, only a few tens of minutes of moderate pain was enough to generate the proposed centralization effect. If this were true in man, then every pain we ever felt would stay with us forever. In fact, even very severe and/or prolonged pains rarely, if ever, leave such an imprint when the peripheral source of pain is finally relieved. Consider the passing of a kidney stone, surgery for total hip replacement, or childbirth.

Amputation Distorts Central Somatosensory Representations: Possible Basis for Telescoping of Phantom Limbs

The shape and position of phantoms usually change with time, often in an orderly sequence. First, the proximal limb (arm, thigh) tends to fade. Later, the perceived location of the distal limb (hand, foot) "telescopes" inward toward the stump (Henderson & Smyth, 1948; Haber, 1956; Jensen

& Rasmussen, 1994). This process is usually complete within the first postamputation year. Such alterations have their counterpart in late changes in PNS ectopia (Devor, 1994), retrograde death of axotomized and deafferented neurons, and observed changes in the shape of central somatosensory maps over time.

Retrograde Degeneration of Neurons following Nerve Injury

In neonates, transection of the peripheral branch of a sensory neuron rapidly leads to the death of the neuron. This retrograde cell death undoubtedly contributes to the relative scarcity and indistinct nature of phantom limbs in individuals with congenitally absent limbs or juvenile amputations (Simmel, 1962b; Vetter & Weinstein, 1967). Sensory neurons in adult dorsal root ganglia are far less sensitive to axotomy. However, in time, a proportion of chronically axotomized dorsal root ganglion neurons do die (e.g., Devor, Govrin-Lippmann, & Raber, 1985). Their gradual attrition may well play a role in the late fading of phantoms. Neurons that have died cannot contribute to the ectopic discharge generated in stump neuromas and dorsal root ganglia. Interestingly, retrograde cell death can sweep transsynaptically, back into the CNS. Thus, beginning many months after the injury, there may be substantial atrophy in the dorsal horn as a result of retrograde transsynaptic loss of deafferented spinal neurons. Indeed, there are several reports of atrophy in the cortical representation of the missing limb in long-term amputees (Campbell, 1905; Woolsey *et al.*, 1979; Dougherty & Lenz, 1994). This reflects retrograde atrophy across several synapses.

Reference of Sensation into Phantom Limbs and Improved Two-Point Discrimination

The shape of central somatosensory maps (homunculi) reflects the density of sensory innervation of the corresponding tissues and the accuracy (resolution) of sensory discrimination, not the actual or perceived shape of the limb itself. The fingers and the lips, for example, are overrepresented in proportion to their contribution to body surface area, while the abdomen and thorax are underrepresented (Penfield & Rasmussen, 1955). This principle, which is also seen in CNS visual and auditory maps, has long been presumed to reflect genetic preprogramming coupled with fine tuning by the environment during a critical period of early life.

Recent evidence indicates that in the somatosensory system, in contrast to other sensory systems, central maps may remain labile into adulthood. For example, when nerves of the hindlimb are severed acutely, cells

in the corresponding part of the spinal map cease responding to stimulation of the skin. However, some time later, they regain response, now to stimulation of neighboring, still-innervated skin of the thigh and lower back (Fig. 6; Devor & Wall, 1981). In effect, the thigh–back representation has spread into the former foot area. Corresponding adjustments in somatosensory maps are expressed at the level of the dorsal column nuclei, the thalamus, and the somatosensory cortex in animals and in man (Kaas, Merzenich, & Killackey, 1982; Devor, 1988; Yang *et al.*, 1994).

There are two expected sensory consequences of such map reorganization. First, in amputees, stimulation of the thigh–back should provoke sensation felt in the foot, and stimulation of the chest wall or chin should provoke sensation referred to the phantom arm. Indeed, these patterns of reference have been well described in human amputees (Cronholm, 1951; Howe, 1983; Ramachandran, Stewart, & Rogers-Ramachandran, 1992). The second expected sensory consequence is that spatial resolution on the stump, e.g., as measured by two-point discrimination, should improve.

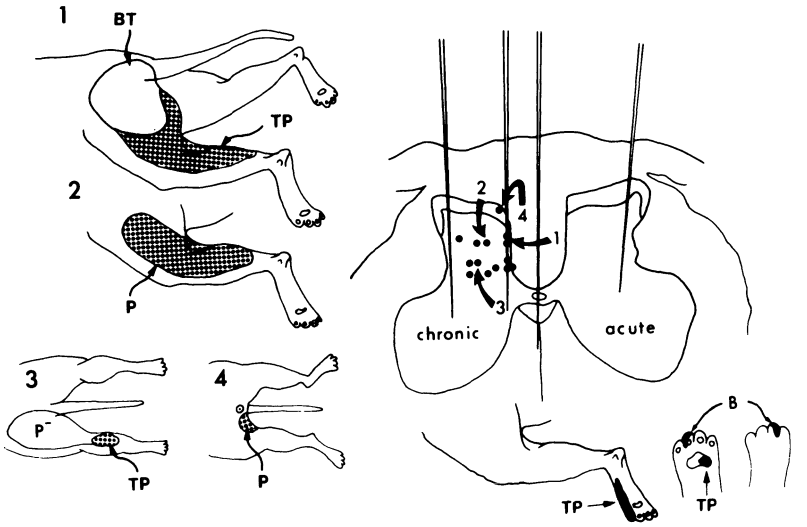

Figure 6. Reorganization of a CNS somatosensory map following nerve injury. Neurons in the medial part of the lower lumbar dorsal horn normally map the distal part of the foot. That is, cells in this part of the spinal body representation respond to stimulation of a toe or a small patch of skin on the foot (sketches on lower right: B, response to light brushing of skin; T, touch; P, pressure). All such responses are silenced immediately on acute transection of the nerves serving the foot (sciatic and saphenous nerves). However, days to weeks later, these same cells begin to respond to the nearest adjacent skin, i.e., the calf, thigh, and lower back (sketches on the left). This functional rewiring and map reorganization occurs in the CNS, not in the periphery. (From Devor & Wall, 1981, with permission.)

This is because a much larger proportion of the central maps is now devoted to the (proximal) skin of the stump. This phenomenon is also well known in human amputees (Haber, 1955; Teuber, Krieger, & Bender, 1949; Weinstein, 1969).

The evidence of map lability following nerve section and limb amputation is complemented by corresponding observations where sensory input is exaggerated. Thus, when an excess of cutaneous stimulation was delivered to two fingers in a monkey for a period of several months, the representation of these fingers in the primary sensory cortex was shown to expand at the expense of neighboring, less extensively stimulated fingers (Jenkins *et al.*, 1990; Clark, Allard, Jenkins, & Merzenich, 1988). This effect could form the basis for persistence of special features of the limb before amputation in the postamputation phantom, i.e., phantom limb memories (Chapter 5). Interestingly, Flor *et al.* (1995), using magnetoencephalography (MEG) reported recently that the degree of remapping observed in different amputees correlated well with the intensity of their phantom limb pain. There was little or no reorganization among amputees who did not have any phantom pain. They interpreted this finding as indicating that the cortical plasticity caused the pain. Although this may be so, Flor *et al.* (1995) may be confusing cause and effect. Specifically, an equally likely explanation of their observations is that phantom pain, which originates in the stump and dorsal root ganglia, exaggerates cortical plasticity.

Distortion of the Body Schema

Imagine that the somatosensory homunculus in the dorsal horn, or one in the primary somatosensory cortex, in fact constituted the neural matrix for conscious sensation. In that case, its internal layout would reflect the way the body was felt. As the thigh/back expanded onto the foot area, the foot would fade from the perceived body schema, and the thigh–foot would expand accordingly. Likewise, in upper limb amputees, the phantom hand would fade, and the chest–chin would inflate. These changes should take weeks to months at most. In fact, distortions of these sorts in the body schema are not described. Rather, as noted, stimulation of the thigh–back comes to be referred into the phantom foot in this time frame, and stimulation of the chest–chin comes to be referred into the phantom hand. Both foot and hand retain their natural shape, size, and salience in the body schema, although the proximal limb fades. This is direct evidence that the neural matrix of conscious sensation is not coterminal with the dorsal horn body map or with the primary cortical body map.

On the other hand, within the time frame of many months, distortions of the perceived body schema do commonly occur. Inward telescoping of

the distal extremity is the prime example. Likewise, when loss of a limb occurred prenatally or at a young age, the body schema usually does not include the missing limb (Simmel, 1962b; Vetter & Weinstein, 1967). Such alterations in the body schema imply that in time, the internal layout of the neural matrix for conscious sensation itself becomes distorted. Indeed, this lability may prove to be the handle with which the seat of (somatosensory) consciousness might ultimately be located. Specifically, in amputees whose phantom hand has telescoped, one might search for a central map in which the hand representation has drifted toward the representation of the stump. Modern imaging techniques such as functional MRI or MEG might already have sufficient spatial resolution to make such a search feasible.

Conclusion

An outlook that views peripheral nerves as bundles of inanimate telephone wires leads to grossly erroneous expectations about the sensory outcome of limb amputation and of surgical approaches to treatment of phantom limb pain. Amputation does not silence afferent signals from the limb. Indeed, it may augment such signals. We now know that the proximal stump of severed nerves and the associated dorsal root ganglia generate ectopic spontaneous and evoked discharge that gives rise to sensation referred to the missing limb (phantom limb). Amplification processes within the CNS may augment this phantom signal and distort it.

(Re)cutting peripheral nerves is no solution. The same cellular processes that evoked the ectopia in the first place almost always reestablish themselves, often quite rapidly. The fact that no palpable neuroma is present is irrelevant. Severed axon endings fire impulses long before a swollen neuroma forms, as do axotomized sensory neurons within the dorsal root ganglia. Dorsal rhizotomy and cordotomy are no solution for somewhat different reasons. These manipulations do eliminate abnormal sensory input from the periphery; however, they replace it with deafferentation pain, probably originating from within the CNS. We are now in a position to ask for the individual amputee, what are the relevant neural sources of phantom limb pain (rational diagnosis), and in some instances at least, to bring them under control (rational management). Rational diagnosis requires systematic stepwise application of local anesthetic blocks at appropriate target sites. Rational treatment requires silencing ectopic sources by means that do not simply replace them with alternative sources. Accomplishing this will require a better understanding of the pathophysiological processes that bring about the ectopia in the first place (Devor, Lamazov, *et al.*, 1994).

CHAPTER 3

Potential Mechanisms of Phantom Tooth Pain

J. Marbach

Introduction

Phantom tooth pain could potentially be a manifestation of an unusual neuropathic pain disorder. Examples of neuropathic pain disorders include reflex sympathetic dystrophy, neuroma, and nerve compression pain. Some suggest that those neuropathic pains that have a predominantly central "generator" comprise the so-called deafferentation pain syndromes (Portenoy, 1991). This theory does not exclude the possibility that a peripheral lesion is required to sustain the pain resulting from the central generator (Devor *et al.*, 1991) (Fig. 1).

Evidence exists that dental pulp amputation not only results in a lesion at the apex or tip of the tooth root but, consistent with other nerve injuries, also influences the CNS (Sessle, 1987; Zimmerman, 1991; Bullitt, 1991). Therefore, extirpation of the nerve and other contents within the tooth collectively known as the pulp may serve not only in its clinical function of abolishing acute pain but also as the initiating factor for chronic pain. It is yet to be determined if phantom tooth pain follows root therapy of nonvital painless teeth.

Intraoral Stump Pain

The site of intraoral stump pain is usually in the alveolar process, in contrast to phantom tooth pain, whose locus is the missing tooth itself. Davis (1993) considers stump pain an important cause of prosthetic limb rejection among amputees. Soreness following tooth extraction is common,

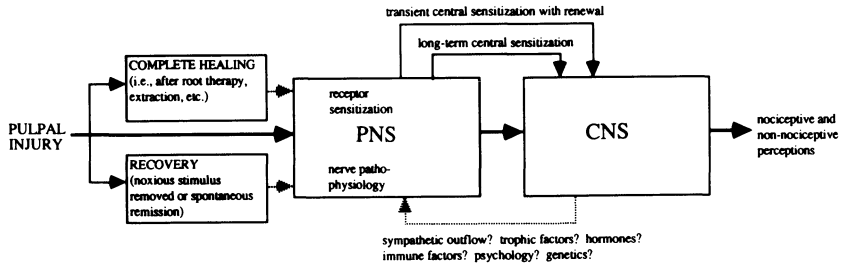

Figure 1. Hypothetical diagram of how a tooth pulp injury may culminate in phantom tooth pain or a nonpainful phantom sensation. Solid arrows have more research support, dotted arrows less (adapted from Devor *et al.*, 1991).

as is pain thought to be linked to ill-fitting dentures and other prostheses. These types of painful conditions occasionally do not disappear with time or with adjustments or replacements of the prostheses. Some of these individuals may suffer from a form of stump pain.

Those who undergo multiple tooth extractions and wear dentures are primarily the elderly. Age and disease are thought to reduce the thickness of the tissue sufficiently to permit a prosthesis to impinge (Sherman, 1989a) on the underlying jaw bone. Others suggest that pain produced by innocuous mechanical stimuli, called mechanical allodynia, results from activation of large-diameter, low-threshold mechanoreceptive afferent fibers (Dubner, 1991). Mechanical allodynia has also been implicated in neuroma pain (Gracely *et al.*, 1992). These studies offer an explanation of how light touch or pressure, as occurs under a denture, could produce intense pain. Recognition of this potential source of pain could result in considerable financial savings for sufferers.

Pathophysiological Mechanisms of Phantom Tooth Pain

Phantom tooth pain is persistent, allodynic, and frequently but not always delayed in the onset. These characteristics, combined with the fact that the dental pulp has been entirely amputated, argue for the role of a central mechanism (Devor *et al.*, 1991; Marbach, 1993). Evidence for the central mechanism is strengthened by the fact that in only a small percentage of cases does chronic pain follow pulp amputation. Recent evidence shows that injury to a peripheral nerve in the rat results in a pain syndrome similar to that of neuropathic nerve pain states in humans (Bennett, Kajander, Sahara, Iadorala, & Sugimoto, 1989). Electron microscopic analyses of

nerves exposed to the same type of injury demonstrate a near complete loss of large myelinated fibers distal to the injury. There was also damage to small myelinated fibers, suggesting that changes in all calibers of peripheral nervous system (PNS) axons may contribute to neuropathic pain in certain types of injury (Basbaum, Gautron, Jazat, Mayes, & Guilband, 1991). These findings, combined with evidence that allodynia associated with neuropathic pain conditions involves altered CNS processing (Dubner, 1991), argue for a PNS/CNS etiology for phantom tooth pain.

Devor and others (1991) have proposed a working model of neuropathic pain. Figure 2 is adapted from this model. It posits changes in both PNS and CNS. In the first hypothesis, "central sensitization" persists following nerve injury. No further triggering signal is necessary. Central "sensitization" is a term used to signify changes in the CNS only some of which have been described. The second hypothesis requires a repeat or renewal of the triggering mechanism because the period of "central sensitization" following peripheral injury fades. In the third hypothesis, painful neuropathic signals are generated exclusively in the PNS. Devor and

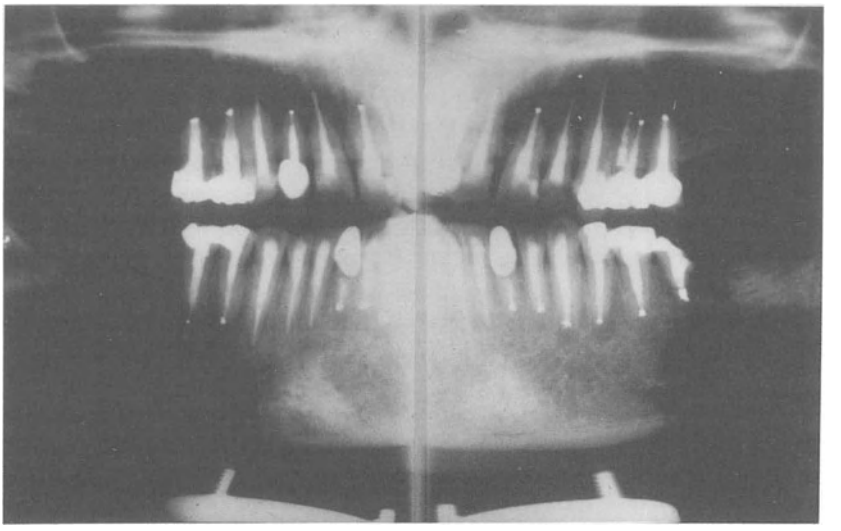

Figure 2. Radiograph of a 22-year-old woman with phantom tooth pain of 4 years' duration. The 12 molar teeth were extracted after endodontic therapy of the first and second molars. All other teeth have been treated endodontically, followed by apicoectomy. Severe pain persists in all of the teeth and edentate areas as well as in the face and regions of the neck. A total of 38 separate root canal treatments, 22 apicoectomies, and 12 extractions were performed for relief of pain (Reprinted from Marbach *et al.*, 1982).

several colleagues (1991) object to the term "deafferentation pain" because, in humans, rarely is the source of pain demonstrably not an afferent input entering the CNS. As evidence, they cite the persistence of afferent activity in several peripheral nerves even after complete nerve transection. With dental pulp amputation, the focus of abnormal impulse generation could be neuroma formation at the root apex. Tasker (1984, 1989) and others (Portenoy, 1991), in contrast, suggest that deafferentation pain is a useful term and emphasize the clinical similarities between various central and deafferentation syndromes.

Melzack's (1993) theory of "a genetically built-in neuromatrix for the whole body" postulates a continuous exchange of information from the environment and PNS to the brain. The theory should predict a genetic program governing the neuromatrix code for the loss of deciduous but not permanent teeth. In a study of kittens, Hu and Sessle (1989) suggested that "natural tooth deafferentation" associated with exfoliation of the primary teeth may differ considerably from the "sudden insult of deafferentation imposed by the simple endodontic procedure in the adult cats." The anatomic and electrophysiological differences between these two processes could account for the lack of observed or reported phantom tooth pain among human children.

Chapter 4

The Role of the Sympathetic Nervous System in Phantom Pain

Joel Katz

Introduction

Recently, Sherman and Arena (1992) have argued that phantom limb pain is not a unitary syndrome but a symptom class, with each class subserved by different etiological mechanisms. For example, one class of phantom limb pain that is characterized by a cramping quality is associated with electromyographic (EMG) spike activity in muscles of the stump, whereas burning phantom limb pain shows no such association (Sherman & Arena, 1992). Katz and Melzack (1990) have identified a class of phantom limb pain that resembles in quality and location a pain experienced in the limb before amputation. Although the precise physiological mechanisms that underlie these somatosensory pain memories are unknown, the presence of preamputation pain clearly is a necessary condition for these pain memories to develop.

Another class of phantom limb pain may come about through involvement of the sympathetic nervous system (SNS). This chapter reviews the theoretical and empirical work that implicates a role for the SNS in contributing to phantom limbs. A brief description of sympathetcially maintained pain is presented, followed by a selective review of evidence for a sympathetic–efferent, somatic–afferent coupling mechanism based on experimental literature. Involvement of the SNS in an animal model of phantom limb pain is then presented, followed by a review of literature suggesting that the SNS contributes to both nonpainful and painful phantom limbs. A model of phantom limb pain is developed that involves a sympathetic–efferent somatic–afferent cycle of activity initiated by higher

brain centers involved in cognitive and affective processes. Finally, results of treatments that block the sympathetic supply are briefly reviewed.

Sympathically Maintained Pain

The role of the sympathetic nervous system in triggering or maintaining pathological pain has been a source of considerable confusion and debate (Campbell, Raja, Selig, Belzberg, & Meyer, 1994; Jänig, 1990b; Nathan, 1989; Ochoa, Torebjörk, Marchettini, & Sivak, 1985; Schott, 1993). Sympathetic nervous system involvement in pain has been attributed to a cycle of sympathetic–efferent somatic–afferent activity in which neural and/or vascular mechanisms participate. Pain is hypothesized to arise, alternatively, from sympathetically triggered ephaptic transmission (Jänig, 1985), sympathetic activation of nociceptors (Campbell, Meyer, Davis, & Raja, 1992; Devor, 1983), or sympathetic activation of low-threshold mechanoreceptors that terminate on sensitized spinal cord cells (Roberts, 1986). A quite different approach is the suggestion of injury-induced alteration in the activity pattern of postganglionic cutaneous vasoconstrictor neurons, which lose their normal thermoregulatory function leading to trophic changes and ischemia (Jänig, 1985).

Systems for classifying the role of the SNS in pain place emphasis on different aspects of the disorder. A clearly important distinction is between sympathetically maintained pain (SMP) and sympathetically independent pain (SIP) (Campbell *et al.*, 1992). Sympathetically maintained pain is defined as pain arising from the action of the sympathetic efferents on afferent fibers in injured peripheral tissue: by definition, it is abolished when the sympathetic supply to the painful region is blocked (Campbell *et al.*, 1994). In contrast, SIP is not dependent on the sympathetic efferents so that maneuvers that are directed at blocking peripheral sympathetic activity do not affect the pain.

One of the major advances achieved by this classification is to dissociate the presence of pain from signs of sympathetic dysregulation in the affected region (e.g., altered temperature, excessive sweating, trophic changes). Evidence of abnormal sympathetic nervous system activity need not accompany SMP (Treede, Davis, Campbell, & Raja, 1992). A model for SMP has been proposed that involves injury-induced up-regulation of α-adrenoreceptors on nociceptors and ongoing sensitization of central pain-signaling neurons following adrenergic activation of nociceptors by norepinephrine released from peripheral sympathetic terminals (Fig. 1). Under these conditions, touch-evoked pain or allodynia (if present) is hypothesized to develop through central modulation from ongoing nociceptor activity. Local anesthetic blockade of the sympathetic supply to the

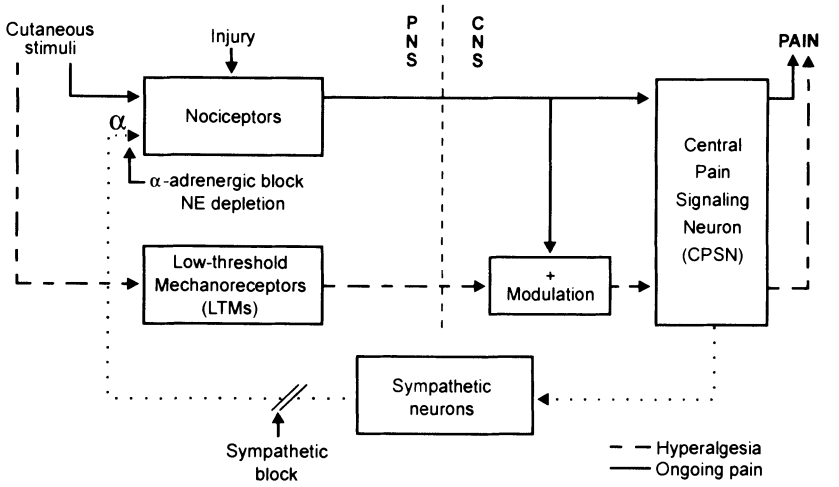

Figure 1. A model for sympathetically maintained pain. Following injury, spontaneous pain results from a sympathetic–sensory coupling mechanism in which nociceptors up-regulate α-adrenergic receptors and respond to norepinephrine (NE) released from sympathetic terminals in the affected region. Sympathetically generated nociceptor activity produces a dynamically maintained state of central sensitization so that activity in low-threshold mechanoreceptors, which normally is not painful, now evokes allodynia in response to light touch. Blocking the sympathetic supply to the region interrupts the sympathetic–sensory coupling mechanism and restores the central neurons to a desensitized state, thus relieving both ongoing pain and allodynia. Reproduced with permission from Campbell *et al.* (1994).

involved region temporarily prevents the release of norepinephrine and reverses the state of central sensitization so that both touch-evoked and ongoing pain are relieved.

According to another system of classification (Jänig, 1993), SMP is thought to represent one of two disorders involving the sympathetic nervous system. *Sympathetic algodystrophy* (reflex sympathetic dystrophy) is characterized by ongoing pain, touch-evoked pain, abnormal regulation of blood flow and sweating, and trophic changes. *Sympathetic dystrophy* is distinguished by the absence of spontaneous pain. In this system, the signs of abnormal sympathetic nervous system activity take diagnostic precedence over the response to treatments that block the sympathetic supply to the affected region. Thus, in contrast to the SMP–SIP classification, pain that persists following sympathetic blockade in a patient with clear signs of regional sympathetic dysregulation and ongoing pain would not suggest a diagnosis of SIP but one of sympathetic algodystrophy, implying that the sympathetic nervous system is somehow involved in maintaining the pain.

Evidence of Sympathetic–Sensory Coupling following Peripheral Injury

Substantial evidence exists for a sympathetic–efferent somatic–afferent coupling mechanism both in the normal, noninjured state (Hallin & Wiesenfeld-Hallin, 1983) and after tissue damage or peripheral nerve injury. However, it is only in the presence of injury-induced pathophysiology that such sympathetic-sensory coupling contributes to pathological pain. Jänig (1993) has outlined some of the possible modes of coupling between the sympathetic efferents and somatic afferents in injured tissue. These include chemical coupling (e.g., α_1-adrenergic), ephaptic coupling (e.g., direct electrical crosstalk), microenvironmental coupling (e.g., changes in the micromilieu of the primary afferent fibers), and indirect coupling in which norepinephrine is postulated to have a presynaptic effect on α_2-adrenergic receptors leading to prostaglandin release and a lowering of the primary afferent threshold (Levine, Taiwo, Collins, & Tam, 1986). Empirical support for coupling other than that of a chemical nature (i.e., through release of norepinephrine from postganglionic sympathetic fibers in close proximity to primary afferent fibers) is scant (Jänig, 1990b). In the present context, ephaptic coupling, which is more likely to occur after partial nerve injury (e.g., after high-velocity gunshot injury), is probably an unlikely mechanism for phantom limb pain following amputation, although it may be more likely to contribute to phantom limb pain following incomplete ruptures or traction injuries of the brachial plexus (as frequently occurs in a motorcycle accident).

Regenerating afferent fibers that are trapped in a neuroma develop a hypersensitivity to intravenous or intraarterial injection of adrenergic agonists and to stimulation of the sympathetic supply of the neuroma (Fig. 2) (Blumberg & Jänig, 1981; Devor & Jänig, 1981; Korenman & Devor, 1981; Scadding, 1981; Wall & Gutnick, 1974b). In addition, chemical coupling is abolished following administration of the α-adrenergic receptor antagonist phentolamine but usually not after β-adrenergic blockade (Blumberg & Jänig, 1981; Devor & Jänig, 1981; Korenman & Devor, 1981; Wall & Gutnick, 1974a, 1974b). These findings form the basis of the hypothesis that paresthesias, dyesthesias, and pain may arise from sympathetic–sensory chemical coupling in damaged tissue (Roberts, 1986).

Devor and his colleagues (Devor & Jänig, 1981; Korenman & Devor, 1981) have shown not only that sympathetic-sensory coupling occurs in the periphery within experimental neuromas but that activity in dorsal root ganglion (DRG) neurons can also be modulated by sympathetic activation after transection of the sciatic nerve (Devor *et al.*, 1994). Responses to both electrical stimulation of preganglionic sympathetic efferents and system-

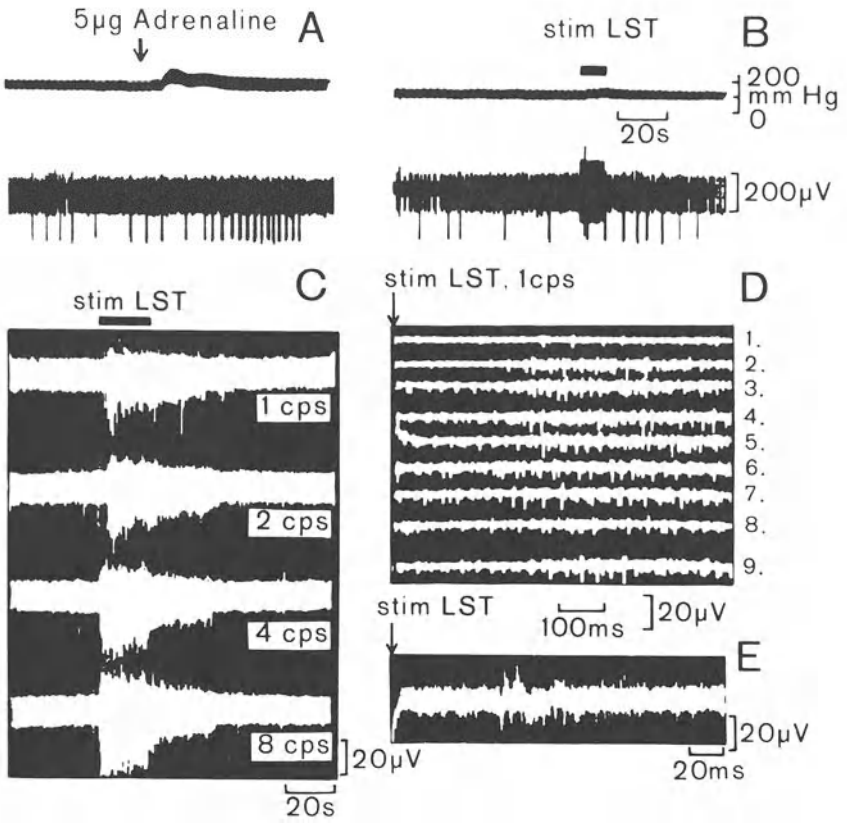

Figure 2. Responses of myelinated (A, B) and unmyelinated (C–E) afferent fibers in cat (A, B) and chronic rat (C–E) neuromas to intravenous injection of 5 μg epinephrine and electrical stimulation of the lumbar sympathetic trunk (LST). C: Stimulation of the LST at frequencies that mimic the physiological discharge rate of sympathetic efferents (i.e., 1–4 Hz) produced activation of unmyelinated afferents in a neuroma of a rat's sciatic nerve 8.5 months after sciatic and saphenous nerve transections. D: Evidence of "wind-up" (increased responsiveness) following repetitive LST stimulation at 1 Hz. E: Activity in postganglionic axons in a branch of the posterior biceps nerve by electrical stimulation of the central cut end of the nerve. Reproduced with permission from Blumberg and Jänig (1981).

ically administered epinephrine were blocked by phentolamine. The various modes of sympathetic–sensory coupling (Jänig, 1993) may also develop in the DRG. The recent finding that injury to the sciatic nerve is followed by sprouting of sympathetic efferents around large-diameter cell bodies in the DRG (McLachlan *et al.*, 1994) increases the potential for sympathetic–sensory coupling and makes the DRG a likely and previously

unsuspected source of sympathetically triggered pain and dysesthesias (Devor *et al.*, 1994).

The Autotomy Model of Phantom Limb Pain in Animals

Wall and colleagues (Wall, Devor *et al.*, 1979; Wall, Scadding, & Tomkiewicz, 1979) developed a rodent model of anesthesia dolorosa in which peripheral neurectomy is followed by self-mutilation behavior termed *autotomy*. In the autotomy model, the sciatic and saphenous nerves of the rat are transected at midthigh level, resulting in complete anesthesia and loss of motor function in the peripheral territories subserved by these nerves. Within 1–3 weeks of denervation, the rats begin to bite and scratch the distal portions of the insensitive paw to the point of amputation. Although there is a controversy over the interpretation of the self-mutilative behavior (Kruger, 1992; Rodin & Kruger, 1984), most researchers do not doubt that autotomy is a response to pain or dysesthesias referred to the anesthetic limb and therefore represents an animal model of phantom limb pain (Coderre, Grimes, & Melzack, 1986b; Devor, 1991). Because the hindpaw is still present, the autotomy model more closely resembles conditions in humans that arise after complete brachial plexus ruptures or dorsal root avulsions. Nevertheless, because the nerve sections produce a deafferentation of the entire hindpaw, it is inferred that any pain or dysesthesias experienced in the denervated territory must be phantom pain. The nature of the autotomy behavior in rats parallels reports of phantom limb pain in human amputees. As noted above, it is not uncommon for amputees to report brief bouts of paroxysmal pain that is experienced as arising from the phantom limb. Observation of rats with denervated hindpaws reveals similar bouts of self-mutilative behavior, presumably in response to pain attacks and/or dysesthesias referred to the denervated paw.

Evidence of Sympathetic–Sensory Coupling in the Autotomy Model

Not only do procedures that enhance or mimic sympathetic outflow increase autotomy levels, but those that reduce or block sympathetic activity decrease the degree of autotomy. Thus, autotomy is enhanced by the monoamine oxidase inhibitor pargyline (Yasumo, Nishigori, & Yamaguchi, 1984) which increases norepinephrine storage and release from peripheral sympathetic terminals. Administration of the antisympathetic agent guanethidine, which in adult rodents acts by preventing the release of norepinephrine from sympathetic nerve terminals, has been shown to reduce

autotomy (Wall *et al.*, 1979; Coderre & Melzack, 1987; Coderre, Abbott, & Melzack, 1984). Likewise, the incidence of autotomy is significantly reduced among rats treated with guanethidine for 10 days beginning 2 days after birth (Colado, Del Rio, & Peralta, 1994). Moreover, neonatal guanethidine sympathectomy not only reduced the self-mutilative behavior relative to controls but also suppressed the changes in spinal norepinephrine normally observed among untreated animals 15 and 60 days after sciatic and saphenous nerve sections.

It has been pointed out (Blumberg & Jänig, 1981; Jänig, 1990b) that the excitation of afferents within an acute experimental neuroma by relatively high-frequency electrical stimulation (10–25 Hz) of the lumbar sympathetic trunk may not have clinical relevance. This is because of the nonphysiological rates required to elicit afferent activity. Jänig (1990a) reported that low-frequency electrical stimulation (1–8 Hz) of the sympathetic supply, but not intravenous epinephrine, elicits activity in C fibers 8.5 months after sciatic and saphenous nerve transections (Fig. 2C). Afferent fibers, which were activated by rates of lumbar sympathetic trunk stimulation within the physiological range of sympathetic efferent fibers (i.e., 1–4 Hz), displayed characteristics suggestive of wind-up (Fig. 2D). That is, repeated lumbar sympathetic trunk stimulation resulted in increasingly greater responsiveness. Note, however, that by 1 to 2 hr after the onset of stimulation, all units had stopped responding.

The failure of epinephrine to elicit afferent activity within these very old neuromas raises the question of the nature of the postganglionic sympathetic–efferent somatic–afferent coupling mechanism. Low-frequency electrical stimulation of the lumbar sympathetic trunk was capable of evoking afferent activity, which provides indirect evidence for the possibility that physiological levels of sympathetic activity may contribute to autotomy behavior in certain animals. Furthermore, the finding that such stimulation was effective in a chronic neuroma, 8.5 months after denervation, provides a mechanism whereby normal levels of sympathetic activity might evoke chronic phantom limb pain and dysesthesias long after amputation.

Effects of Increasing or Decreasing Sympathetic Activity on Autotomy Levels among Animals Injured Prior to Sciatic and Saphenous Nerve Sections

A growing body of clinical and laboratory data shows that injury produces prolonged changes in central nervous system function that influence responses to subsequent somatosensory inputs. The data strongly suggest that this injury-induced neuroplasticity may contribute to the

experience of pain long after the offending stimulus has been removed or the injury has healed (Coderre *et al.*, 1993).

The autotomy model described above has also been used to explore the effects of a prior injury on the subsequent development of pain referred to the anesthetic limb in an attempt to model the observations among human amputees that preamputation pain persists as phantom limb pain following amputation. Studies have shown that chemical or thermal injury of the paw prior to deafferentation increases the severity of autotomy or leads to a shift in the site of self-mutilation (Coderre & Melzack, 1986; Katz *et al.*, 1991). Because all sensory input from the injured paw is eliminated as a consequence of deafferentation, the enhanced autotomy has been attributed to increased pain resulting from sensitization of central cells by the earlier injury, thus reflecting a change in central neural function that long outlasts the duration of injury.

Coderre, Grimes, and Melzack (1986a) and Coderre and Melzack (1987) examined the effects of altering sympathetic activity or central monoaminergic activity on autotomy levels among animals with or without hindpaw injuries induced prior to sciatic and saphenous nerve sections. In one study (Coderre *et al.*, 1986a), rats received bilateral electrolytic lesions or sham lesions of central noradrenergic neurons in the locus coeruleus that are known to exert a tonic inhibitory influence over dorsal horn neurons. Autotomy progressed more rapidly among lesioned rats that received an injury prior to denervation, although the degree of autotomy did not differ from that in lesioned animals that did not receive a prior injury. In a second study (Coderre & Melzack, 1987), the enhancement of autotomy that typically develops when a paw is injured before denervation was decreased by a combination of intrathecal capsaicin and guanethidine but not by guanethidine or capsaicin alone, suggesting that both C-fiber activity and sympathetic outflow are critical to the heightened autotomy. In contrast, intrathecal guanethidine alone, but not the combination of capsaicin and guanethidine, was effective in reducing autotomy among uninjured rats. One implication of these findings is that, in the presence of CNS sensitization by a prior injury, procedures designed to treat phantom limb pain by reducing the afferent *or* efferent limb of a sympathetic-sensory cycle of activity may not be effective until both C-fiber activity *and* sympathetic efferent activity are abolished.

Heritability of Neuropathic Pain

One of the more exciting lines of recent research raises the issue of the heritability of neuropathic pain. The work was done in rats using the

autotomy model: Devor and Raber (1990) developed two lines of rats by interbreeding those that exhibited high levels of autotomy and interbreeding those that showed low levels of autotomy. Offspring had their sciatic and saphenous nerves transected, and rats that showed high levels of autotomy were interbred, as were those that showed low levels of autotomy. Interbreeding by selecting for high or low autotomy behavior was carried out for 13–15 generations. From the third generation onward, high- and low-autotomy rats could be distinguished by level of autotomy. In addition, there was a significant decrease over the generations in the variability of autotomy within lines. By the 11th generation, the incidence of autotomy approached 90% in the high-autotomy line and was approximately 10% in the low-autotomy line. Moreover, the kinetics of autotomy behavior were altered with a shift to a much earlier onset after denervation among high-autotomy animals as the interbreeding continued. Rather than beginning approximately 3 weeks after nerve section, successive generations showed autotomy onset as early as the first week. Twelfth-generation high-autotomy rats showed significantly greater sensitivity on sensory and thermal testing than did low-autotomy rats.

Based on the pattern of autotomy among hybrid rats and backcrossed hybrids, the authors inferred that the mode of inheritance of the autotomy trait is through a single autosomal recessive gene. Whether the two lines of rats differ in their relative sensitivity to sympathetically generated afferent activity or background level of sympathetic outflow has not yet been established. However, in another study autotomy levels were found to differ as a joint function of the strain of rat and the level of environmental stress (Wiesenfeld-Hallin & Hallin, 1983), suggesting that genetic differences in sympathetic outflow may account for the pain-related behavior under stressful conditions. The strong genetic component associated with the autotomy trait raises the possibility that among patients, some may inherit a predisposition to develop chronic neuropathic pain after amputation (Devor & Raber, 1990).

Phantom Limb Pain

The detailed and highly technical work carried out with experimental neuromas stands in stark contrast to the dearth of information on the role of the sympathetic nervous system in phantom limb pain among human amputees. Generally, reports are poorly controlled and are based on small sample sizes, making generalization questionable. Furthermore, with the exception of more recent studies, phantom limb pain (and pain relief, if a treatment is involved) is not assessed with sufficient attention to important

parameters such as quality, frequency, intensity, and duration of pain. This criticism is especially relevant in the light of the multiple mechanisms and levels of the PNS and CNS that have been proposed to contribute to phantom limb pain. For example, the findings (Sherman & Arena, 1992) that "cramping" phantom limb pain correlates with EMG measurements but not blood flow at the stump and that "burning" stump and phantom limb pain correlate with stump blood flow but not EMG recordings underscore the importance of assessing the quality of the pain reported by patients with phantom limb pain.

Evidence that the sympathetic nervous system is involved in phantom limb pain comes from studies using pharmacological block (Livingston, 1938, 1943) or surgical interruption (Bailey & Moersch, 1941; Kallio, 1950) of the sympathetic supply to the involved limb. This was reported to produce at least temporary alleviation of pain. Long-term relief of phantom limb pain has been reported with propranolol, a β-adrenergic blocking agent. Unfortunately, these reports are uncontrolled and unblinded (Ahmad, 1984; Marsland, Weekes, Atkinson, & Leong, 1982; Oille, 1970). An open trial of propranolol in six (nonamputee) patients with pain from peripheral nerve injuries showed very little benefit (Scadding, Wall, Wynn Parry, & Brooks, 1982). Electrical or mechanical stimulation of the lumbar sympathetic chain produces intense pain referred to the phantom limb (Echlin, 1949; Noordenbos, 1959), whereas sensations are referred to the abdomen or flank in pain patients without amputation (Noordenbos, 1959).

Regional sympathetic hyperactivity has also been hypothesized to 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 on stimulation of the stump whether or not the stump is painful or shows signs of trophic or vascular changes (Doupe, Cullen, & Chance, 1944; Livingston, 1938). The characteristic qualities of superficial burning pain and deep aching pain may provide additional evidence of sympathetic nervous system involvement (Doupe *et al.*, 1944). However, just as some sympathetically maintained pains occur in the absence of regional sympathetic abnormalities (Campbell, Meyer, & Raja, 1992), not all patients with phantom limb pain resulting from sympathetic nervous system involvement would be expected to show signs of abnormal sympathetic nervous system activity at the stump (e.g., trophic changes, abnormal sympathetic reflexes and sweating, alterations in stump blood flow). This possibility suggests that the abnormality associated with sympathetically maintained pains of this type does not reside in the sympathetic nervous system but in

the afferent supply of the involved extremity (Schott, 1993; Treede *et al.*, 1992). The possibility that there may be no signs of sympathetic nervous system abnormality underscores the importance of using diagnostic sympathetic blocks, the phentolamine test, or regional infusions of guanethidine to ascertain the presence of SMP.

Even when sympathetic nervous system abnormalities are present, their relationship to pain in the stump and pain in the phantom is not always clear-cut (Sunderland, 1968). For example, Livingston (1938) reports cases of amputees with phantom limb pain who had abnormalities in sweating and large temperature differences between the stump and contralateral intact limb but who did not complain of stump pain. Local anesthetic infiltration of the sympathetic ganglia was followed by relief of phantom limb pain, a sense of warmth and relaxation in the phantom, and a reversal of the vasomotor, sudomotor, and trophic changes at the stump—all of which often extended well beyond the duration of action of the local anesthetic. Despite the correlation between the restoration of normal sympathetic functioning and the relief of phantom limb pain, it remains unclear whether the sympathetic abnormalities were responsible for the pain or whether both were caused by a common third factor (e.g., reduced sympathetic transmitter release).

Nyström and Hagbarth (1981) carried out microneurographic recordings of activity from skin and muscle nerve fascicles in two amputees with phantom limb pain. One patient had sustained a below-knee amputation and suffered from intense cramping pain referred to the phantom foot. Recordings from muscle nerve fascicles in the peroneal nerve showed that although bursts of activity in sympathetic fibers were accentuated by the Valsalva maneuver, the phantom pain remained unchanged, suggesting that the pain was not dependent on sympathetic activity. The second patient had undergone amputation of his left hand at the wrist secondary to extensive lacerations following an agricultural accident. Microneurographic recordings were taken from a skin nerve fascicle in the left median nerve at the wrist. In both patients, tapping the neuroma at the stump evoked marked neural activity, afterdischarge, and an intensification of the phantom limb pain. Interestingly, although local anesthetic infiltration into the tissue of the stump surrounding the neuroma abolished (or reduced in one patient) the tap-induced increase in neural activity and phantom limb pain, in neither patient were the spontaneous or background neural activity and phantom limb pain changed. In the light of Devor and Wall's (1990) recent work, the ongoing neural activity that persisted after lidocaine infiltration may well have originated in the DRG and propagated antidromically to reach the recording electrode in the stump (Devor, 1994).

Further evidence of a possible connection between the sympathetic

nervous system and pain after amputation comes from a single-blind study (Chabal *et al.*, 1992) of nine amputees with stump pain ($n = 9$) and concomitant phantom limb pain ($n = 3$) who received successive perineuromal injections of normal saline (0.5 ml), epinephrine (5 μg in 0.5 ml normal saline), and lidocaine (1 ml 1%). Within 1–2 sec of injection of epinephrine, all patients reported an increase in the intensity of local stump pain, although only one of the three patients with phantom limb pain noted an increase in phantom limb pain (Fig. 3).

The quality of the pain following injection of epinephrine was described as "poorly localized shooting or electric shock-like" while the area of discomfort increased from baseline. Four patients remarked that the limb was "on fire." Lidocaine injection significantly decreased but did not abolish the pain. Five patients who also received a control injection of subcutaneous epinephrine (5 μg in 0.5 ml normal saline) in a region distant from the neuroma reported a localized, minor stinging of approximately 1–2 sec duration that was described as distinctly different from the pain experienced in response to perineuromal injection of epinephrine. Unlike the results of Wallin, Torebjörk, and Hallin (1976) in which hyperalgesia took 30 min to develop after iontophoretic application of epinephrine in a previously sympathectomized (non-amputee) patient, the immediate response of these patients to perineuromal injection of epinephrine suggests a direct α-adrenergic coupling mechanism. The possibility of indirect chemical or microenvironmental coupling cannot be excluded.

The Relationship between Phantom Limbs and Correlates of Sympathetic Nervous System Activity at the Amputation Stump

Despite frequent assertions that the sympathetic nervous system is involved in the production and maintenance of phantom limb pain, surprisingly few studies have actually examined peripheral sympathetic nervous system activity at the stump and contralateral limb. Sliosberg (1948) studied 141 amputees and found that the stump was cooler than the intact limb in 94 patients, but he did not relate the temperature difference to the presence or absence of phantom limb pain. Kristen, Lukeschitsdi, Plattner, Sigmund, and Resck (1984) reported that a "patchy asymmetrical temperature" distribution of stump thermograms was significantly more frequent among stump pain sufferers than in patients who were free from stump pain. However, thermograms were no different for patients with or without phantom limb pain. In contrast, Sherman and colleagues (Sherman, 1984; Sherman & Bruno, 1987) observed a negative correlation between temperature at the stump and the presence of burning, tingling, or throbbing phantom limb and stump pain. This indicates that reduced

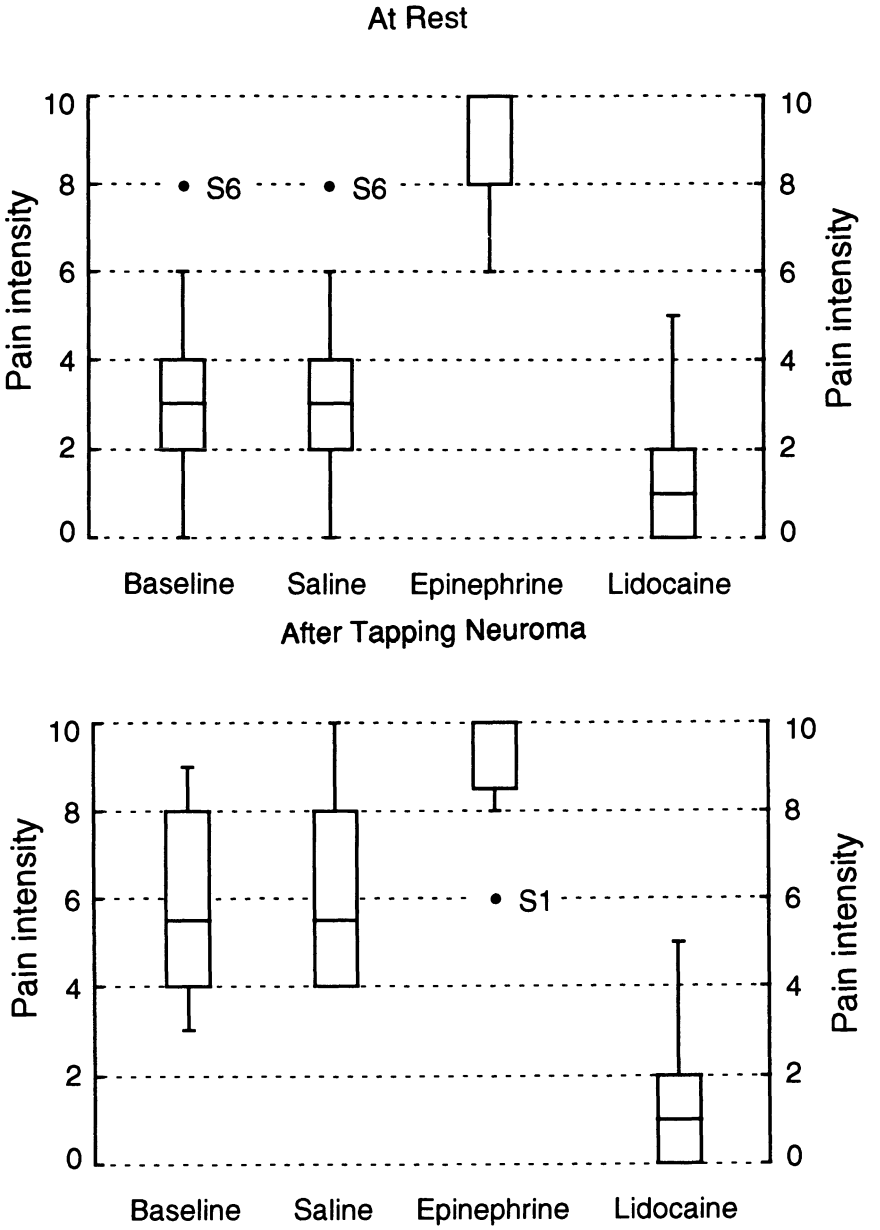

Figure 3. Box-and-whisker plots depicting pain intensity reported by amputees before (baseline) and after perineuromal injection of saline, epinephrine, and lidocaine, shown for conditions of rest and after tapping the neuroma. Median values correspond to the horizontal line partitioning each box; the first and third quartiles are represented by the ends of the boxes. Individually identified patients (e.g., S1, S6) represent relatively unusual values. Note the significant increase in pain following injection of epinephrine and the reduction following lidocaine injection. Box plots were generated from data presented by Chabal *et al.* (1992).

blood flow to the stump is associated with increased levels of pain. Repeated measurements of the same patients on different occasions revealed that lower temperatures at the stump relative to the contralateral limb were associated with greater intensities of phantom limb and stump pain, suggesting that the reduced blood flow was in some way causally tied to the pain. However, in the majority of cases, the relationship between phantom pain and limb temperature was confounded by coexisting stump pain so that it was not possible to attribute the presence of phantom limb pain unambiguously to altered blood flow at the stump.

Katz (1992b) followed up this line of inquiry by comparing skin conductance and surface skin temperature of the stump and contralateral limb in amputees reporting phantom limb pain, nonpainful phantom limb sensations, or no phantom limb at all. The results showed that although mean skin temperature was lower at the stump than on the contralateral limb in all groups, the difference was significant only for the two groups that had a phantom (Fig. 4). Differences between stump and intact limb temperature in excess of $-1°C$ were associated with the presence of a phantom limb with no concomitant stump pain (Table 1).

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 with other qualities of sensation supports the hypothesis (outlined below) of a sympathetic–sensory coupling 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.

Psychophysical Correlates of 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 *et al.*, 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 on 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.

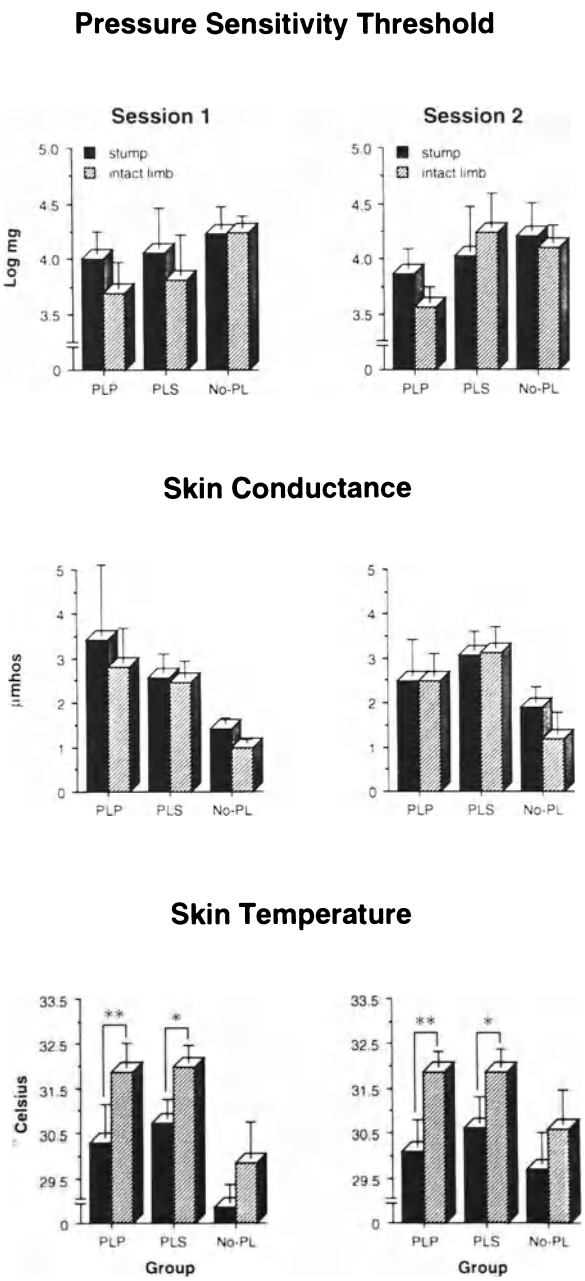

Figure 4. Mean pressure sensitivity thresholds, skin conductance, and skin temperature of the stump and contralateral limb for three groups of amputees on two sessions separated by at least 24 hr. PLP refers to phantom limb pain; PLS, nonpainful phantom limb sensations; No PL, no phantom limb at all. Mean stump skin temperature was significantly lower than that of the contralateral limb for groups PLP ($p < 0.005$) and PLS ($p < 0.05$) but not Group No PL. Reproduced from Katz (1992b), with permission.

Table 1. Mean Stump–Intact Limb Difference Scores for Pressure Sensitivity Thresholds, Skin Conductance, and Skin Temperature of Three Groups of Amputees on Two Sessions[a]

	Group PLP (n = 11)		Group PLS (n = 9)		Group No PL (n = 8)	
Session 1						
PST (log mg)	0.31	(1.3)	0.25	(1.2)	−0.004	(0.5)
SC (μmho)	0.61	(3.0)	0.11	(0.7)	0.43	(0.4)
ST (Celsius)	−1.59*	(1.8)	−1.26*	(1.3)	−0.88	(1.9)
Session 2						
PST (log mg)	0.30	(0.6)	−0.21	(0.6)	0.11	(0.7)
SC (μmho)	0.02	(1.7)	−0.06	(1.5)	0.74	(0.9)
ST (Celsius)	−1.75*	(1.8)	−1.25*	(1.5)	0.85	(2.0)

[a]Sessions were separated by at least 24 hr. Standard deviations are shown in parentheses. Stump-intact limb difference scores were obtained by subtracting measurements taken at the intact limb from those at the stump. Negative difference scores indicate that, relative to the intact limb, the stump is lower in skin temperature, lower in skin conductance, and more sensitive to applied pressure. *Significantly different ($p < 0.05$) from Group No PL. Reproduced from Katz (1992b) with permission.

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, 1992b; Katz *et al.*, 1989). As shown in Figures 5–7, 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) post-ganglionic sympathetic fibers on primary afferents located in the stump and stump neuromas. Release of acetylcholine and norepinephrine from postganglionic sympathetic fibers produces transient vasoconstriction and heightened skin conductance responses. In addition, 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 on 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 (Katz, 1992b; Katz *et al.*, 1989). If central sensitization has also developed

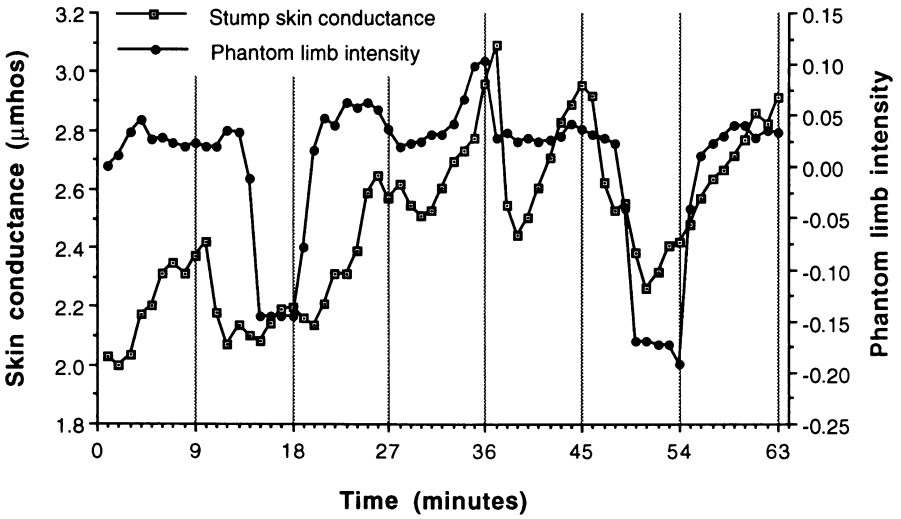

Figure 5. A minute-by-minute plot of the relationship between stump skin conductance and the intensity of nonpainful phantom limb paresthesias for a subject with an amputation above the knee and paresthesias referred to the phantom foot and toes. Skin conductance was continuously measured at the stump over a 63-min period while the subject indicated 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-sec 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.

through either prior injury, trauma during amputation, or peripheral inflammation, or, if the sympathetic–sensory coupling involves nociceptors (Roberts, 1986), the sensation may be one of dysesthesia.

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, as shooting pains, somatosensory memories, and phantom limb movements do not also correlate with stump skin conductance (Fig. 8) (Katz, 1992b). That is, changes in stump skin conductance are related only to the perception of paresthesias (Figs. 5–7) and not to other qualities of sensation (Table 2).

The precise role of postganglionic sudomotor fibers in generating phantom limb paresthesias is not known. The possibility exists that the relationship between stump skin conductance levels and phantom limb

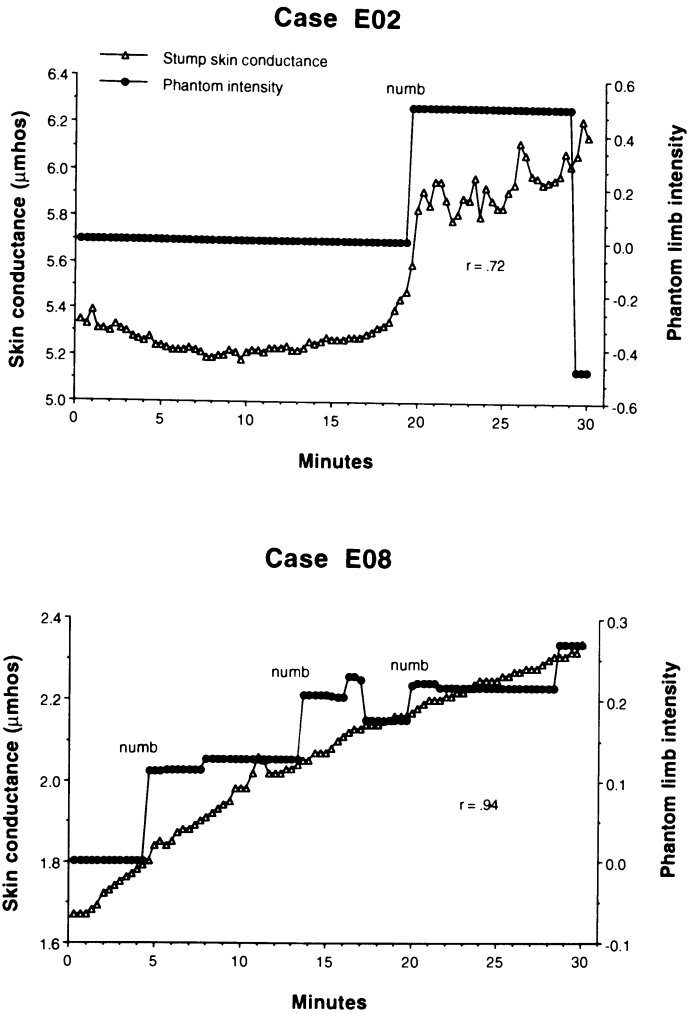

Figure 6. Plots of the relationship between stump skin conductance and the intensity of phantom limb paresthesias for two subjects with nonpainful phantom limb paresthesias. Skin conductance was measured at the stump over a 30-min period while the subjects indicated the intensity of the phantom limb by turning a dial. Each data point represents a mean of three values consecutively sampled at 10-sec intervals. Changes in the intensity of paresthesias (described as increases and decreases in "numb" sensations referred to the phantom limb) occur in concert with changes in stump skin conductance. Also shown is the correlation coefficient describing the strength of the relationship between the two variables and the subjects' descriptions of the quality of the phantom sensation. Reproduced from Katz (1992b), with permission.

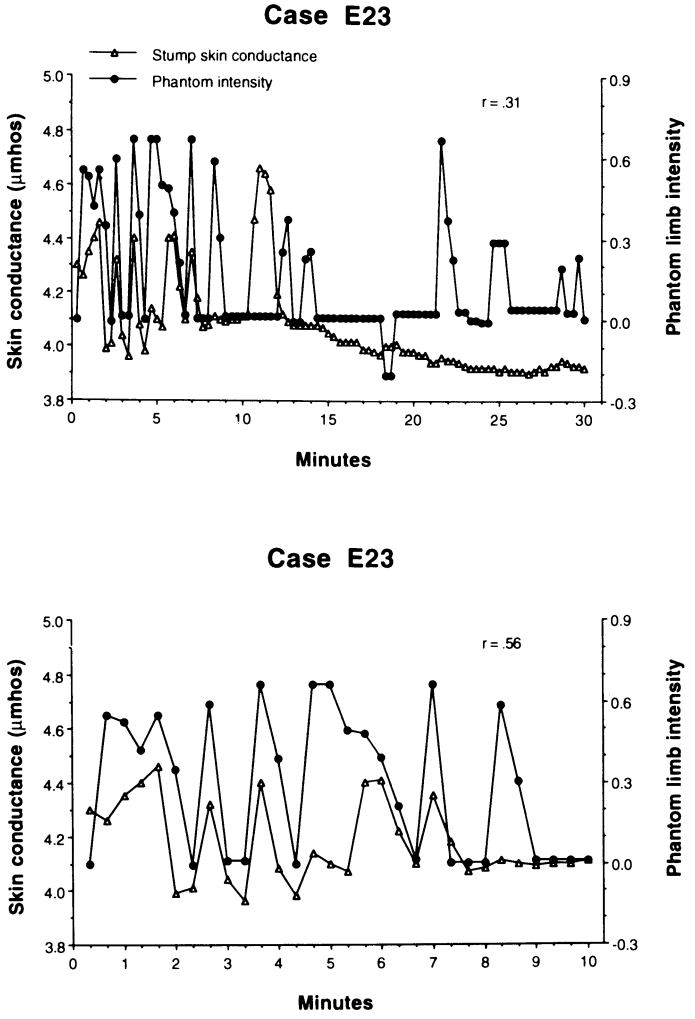

Figure 7. The relationship between stump skin conductance and phantom limb intensity for another amputee with phantom limb paresthesias shown for a 30-min session (top panel). For ease of viewing, the bottom panel shows only the first 10 min of the same session when the two measures showed a prominent tendency to covary. All changes in phantom limb intensity were described by the subject as increases and decreases in "numbness" experienced in the phantom foot. Reproduced from Katz (1992b), with permission.

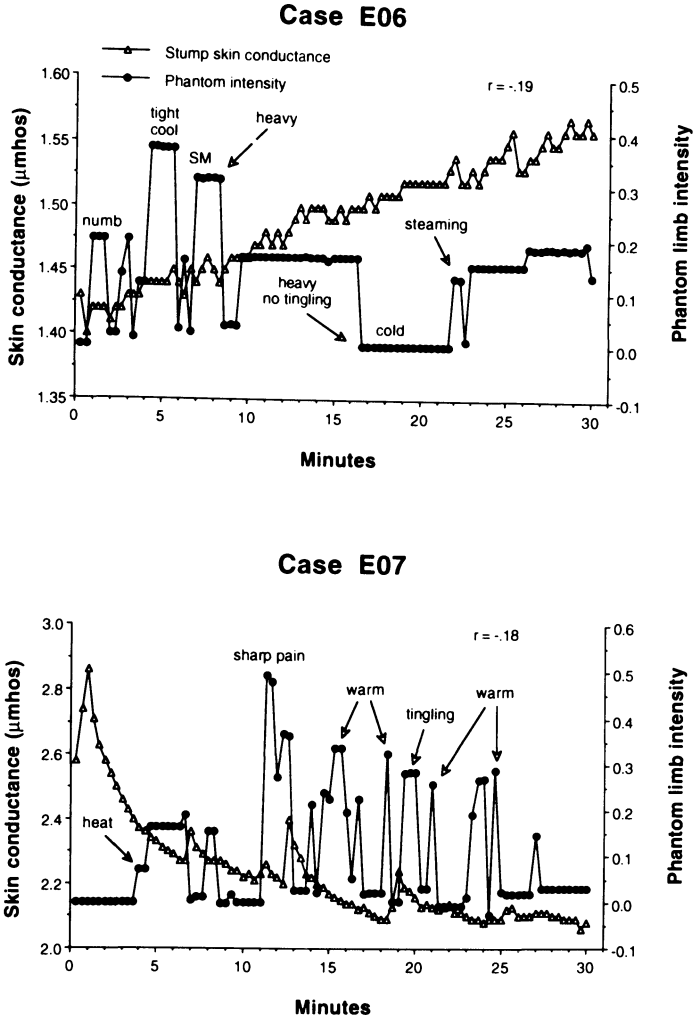

Figure 8. Plots of the relationship between stump skin conductance and various qualities of phantom limb pain for two subjects in Group PLP. Note that, unlike the plots in earlier figures, there is no relationship between stump skin conductance and the intensity of phantom limb pain. Reproduced from Katz (1992b), with permission.

Table 2. Mean Pearson Correlation Coefficients for Patients with Phantom Limb Pain (Group PLP) and Nonpainful Phantom Limb Paresthesias (Group PLS)[a]

	Group PLP (n = 11)		Group PLS (n = 9)		p
Correlation coefficient (r)					
PLI and stump SC	−0.02		0.29*		0.01
PLI and stump ST	0.06		−0.17		ns
Number of significant rs					
PLI and stump SC	6/19	(32%)	8/12	(67%)	0.06
PLI and stump ST	8/19	(42%)	5/12	(42%)	ns

[a]PLI, phantom limb intensity; SC, stump skin conductance; ST, stump skin temperature; ns, not significant ($p > 0.05$); * significantly ($p < 0.05$) different from zero. Shown for each group is the number of significant ($p < 0.002$) correlations between PLI and stump SC and between PLI and stump ST. Values of p are from χ^2 and ANOVA F test for between-group comparisons of frequencies and means, respectively. Reproduced from Katz (1992b), with permission.

paresthesias reflects a direct cholinergic–afferent coupling mechanism (Diamond, 1959). However, Devor and Bernstein (1982) failed to reproduce evidence of such coupling. Moreover, peripheral sudomotor blockade with atropine in patients with sympathetically-maintained pain failed to have an immediate analgesic effect, suggesting that the cholinergic limb of the sympathetic nervous system does not contribute to SMP (Glynn, Stannard, Collins, & Casale, 1993). Another possibility is based on the premise that stump skin conductance provides a more accurate indication of postganglionic discharge than surface skin temperature. Because sudomotor and vasomotor fibers tend to discharge in tandem (Bini, Hagbarth, Hynninen, & Wallin, 1980), skin conductance responses may merely be a marker for an adrenergic–afferent coupling mechanism generated by epinephrine release following activity in postganglionic vasomotor fibers.

There are several lines of indirect evidence to support the hypothesis that moment-to-moment fluctuations in the intensity of phantom limb paresthesias reflect sympathetic-sensory coupling. First, sympathetic activity measured by skin conductance responses and changes in skin temperature reflects the activity of postganglionic sudomotor and vasomotor fibers, respectively (Bini *et al.*, 1980; Hagbarth, Hallin, Hongell, Torebjörk, & Wallin, 1972). Multiunit sympathetic activity recorded from skin nerve fascicles in awake humans shows a strong relationship to effector organ responses including vasoconstriction and sweat gland activity (Bini *et al.*, 1980; Hagbarth *et al.*, 1972). These studies demonstrate that bursts of activity in sudomotor and vasomotor fibers are reliably followed by tran-

sient electrodermal responses and plethysmographic signs of vasoconstriction within the region of skin subserved by the sympathetic fibers under study.

Second, intraneural recordings from sensory nerve fascicles in conscious humans reveals a remarkably strong relationship between the perception of nonpainful paresthesias and spontaneous bursting activity in afferent fibers (Nordin, Nyström, Wallin, & Hagbarth, 1984; Ochoa & Torebjörk, 1980). Finally, nonnoxious percutaneous electrical stimulation of afferent nerves located in the stump of forearm amputees produces paresthesias referred to a localized region of the phantom hand but not the stump. Subsequent alterations in the amplitude of electrical stimulation are paralleled by corresponding perceptual changes in the intensity of phantom limb paresthesias (Anani & Körner, 1979).

Taken together, these studies suggest that the paresthetic component of the phantom limb may in part represent 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 might alter phantom limb sensations through their actions on the SNS. Direct support for this hypothesis is not available and would require that changes in the intensity of phantom limb paresthesias (or dysesthesias) be correlated with microneurographic recordings from postganglionic sympathetic and primary afferent fibers in amputation stump neuromas.

The idea that emotional and psychological processes can cause pain has traditionally 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. 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 *et al.*, 1989). Fifty percent of lower extremity amputees report that attacks of phantom limb pain are triggered by emotional distress (Jensen *et al.*, 1985) as long as 7 years after amputation (Krebs, Jensen, Krøner, Nielsen, & Jørgensen, 1985). A combination of progressive relaxation training and EMG 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 *et al.*, 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 pre-

cipitating transient but profound alterations in the quality and intensity of phantom limb sensations. These processes include concentration (Morgenstern, 1964), 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), and witnessing cruel and violent acts (Pilowsky & Kaufman, 1965; Stengel, 1965). One amputee, in an interview 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" (Katz, unpublished observations).

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 Figure 9 outlines a mechanism through which cognitive and affective processes associated with higher cortical and limbic centers may alter phantom limb sensations. The reciprocal connections among 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 precipitate 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 that are trapped in a neuroma are exceed-

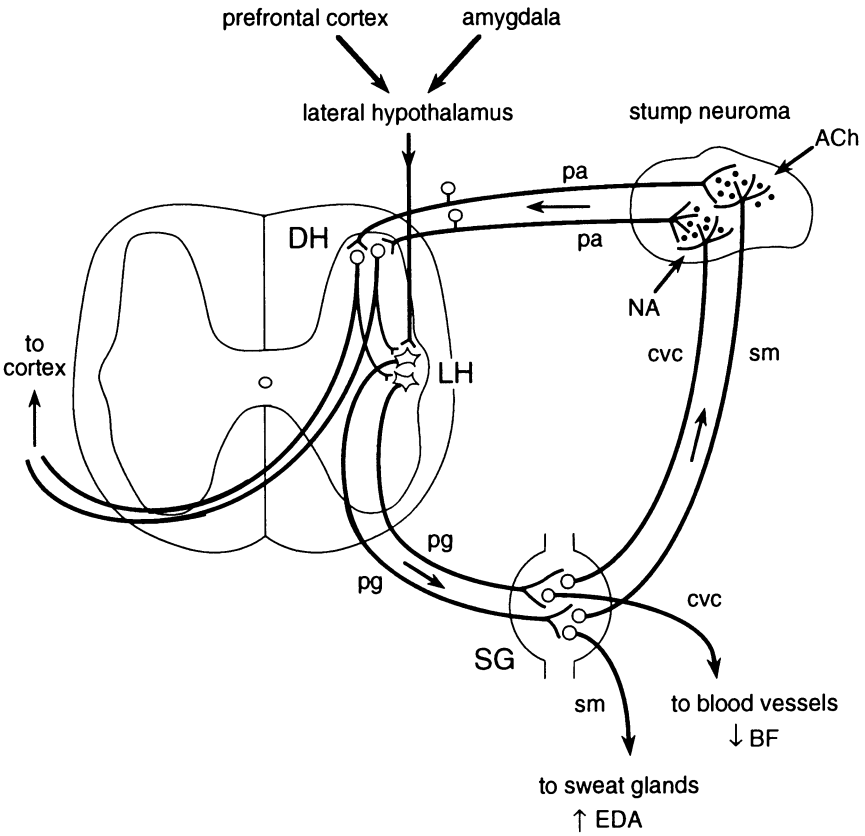

Figure 9. Schematic diagram illustrating a mechanism of sympathetically generated phantom limb paresthesias. Spontaneous sympathetic activity or excitatory inputs descending from cortex (e.g., from the perception of a salient event, loud noise, thought, or feeling) increase the discharge rate of preganglionic (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 norepinephrine 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 by injury, or nociceptive primary afferents are activated, then the perception may be one of dysesthesias. Adapted from Fields (1987), with permission.

ingly sensitive to the postganglionic sympathetic neurotransmitters norepinephrine (Wall & Gutnick, 1974b) 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 because stump impulses would be subject to less inhibition on reaching the spinal cord. This is consistent with the observation that the threshold for detecting sensations in the phantom limb during stimulation of the stump is lower than that at the site of stimulation itself (Carlen *et al.*, 1978).

Another possibility (Campbell *et al.*, 1992) is that amputation leads to increased expression of α-adrenergic receptors located on mechanoreceptors or nociceptors in stump neuromas. This hypothesis would explain the perception of phantom limb paresthesias or dysesthesias in the absence of regional sympathetic hyperactivity or trophic changes at the stump. 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.

Treatment Implications Involving the SNS

As noted above, the majority of studies of phantom limb pain lack the rigorous control conditions and adequate sample sizes to conclude with certainty that specific treatments are more effective than no treatment or placebo treatment. Chabal's (Chabal *et al.*, 1992) findings involving intraneuromal injection of epinephrine provide the strongest evidence in support of an adrenergic sympathetic–sensory coupling mechanism underlying stump pain and possibly phantom limb pain as well. The results of early studies showing that local anesthetic infiltration into the sympathetic chain (Livingston, 1938, 1943) and sympathectomy (Bailey & Moersch, 1941; Kallio, 1950) at least temporarily relieve phantom limb pain also suggest that sympathetic ganglion blocks or surgical sympathectomies are effective because they block the release of norepinephrine from the peripheral sympathetic terminals.

It should be noted, however, that pain relief in response to a local anesthetic sympathetic block may be related to factors other than sympa-

thetic blockade. Limitations that reduce the specificity of diagnostic sympathetic blocks include a systemic action of the local anesthetic and diffusion of the agent to the dorsal roots, resulting in small fiber block (Raja, 1993). The lack of permanence of sympathectomy for phantom limb pain (Kallio, 1950) may result from a variety of factors including inadequacy of diagnosis, extent of sympathectomy, surgical skill, and confusion about anatomy (Campbell *et al.*, 1994). The finding that β-adrenergic receptor blockade does not seem to be effective in relieving phantom limb pain (Scadding *et al.*, 1982) is consistent with the negative results of propranolol for treatment of SMP in nonamputees (Campbell, Raja, & Meyer, 1993).

Phantom limb pain and stump pain respond well, but temporarily, to epidural or spinal administration of local anesthetics or opioids (Jacobson & Chabal, 1989; Jacobson, Chabal, & Brody, 1989; Jacobson, Chabal, Brody, Mariano, & Chaney, 1990). Although the relevant assessments to determine the presence of SMP were not done in these studies, the possibility remains that the continuous sympathetic blockade achieved by epidural infusions of local anesthetic agents may prove effective in the management of patients with SMP (Campbell *et al.*, 1994). To date, neither the phentolamine test (Raja, Treede, Davis, & Campbell, 1991) nor regional infusions of guanethidine have been tried for phantom limb pain. Raja (1993) has published guidelines for evaluating patients suspected of having SMP.

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 also induce reflex bursting activity in cutaneous sudomotor and vasomotor sympathetic fibers (Delius, Hagbarth, Hongell, & Wallin, 1972; Hagbarth *et al.*, 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 indirect support for the model shown in Figure 9 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.

Chapter 5

Central Nervous System Correlates and Mechanisms of Phantom Pain

Joel Katz

Persistence of Preamputation Pain (Pain Memories)

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). Case 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. Appendix I provides an annotated bibliography of the literature, in chronological order, of reports of pain memories.

The following descriptions reported by patients in the study by Katz and Melzack (1990) illustrate some of many qualities of pain that persist or recur after amputation. They also attest to the reality of the phantom limb: the pain memories reported by these patients are indistinguishable from the sensations and pains they experienced in the limb before amputation. In this chapter, in contrast to Chapter 3, we take the position expressed by many patients—that pain memories are precise sensations indistinguishable from the pains experienced before amputation.

Case 108: Female, 62 Years Old, Bilateral Above-Knee Amputation

This 62-year-old retired nurse had undergone a left above-knee amputation in 1981 and, 2 years later, a right above-knee amputation as a result of peripheral vascular disease and associated diabetes mellitus. She was an extremely insightful and thoughtful woman who, despite her physical limitations, maintained an active, independent lifestyle. At the time of the interview, she reported having experienced several phantom pains that resembled pains she had felt in her limbs at some time before the amputations. She reported having had preamputation pain of approximately 2 year's duration in both limbs that became progressively more intense with time. She had had pain in the lateral "three toes on the left that went black with gangrene" that persisted after the amputation. She continued to suffer weekly bouts of this phantom limb pain. The preamputation pain on the right leg started in the toes and spread to the back of the leg. The pain she reported in the right phantom is the same, although the original preamputation pain was more intense. She also claimed that she was still capable of predicting weather changes by the onset of an arthritic ache in her phantom knee.

Case 132: Male, 62 Years Old, Right Above-Knee Amputation

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

In addition to these pains, he reported that at least once a day he feels the pain of the"first cut" that is, the same incisional pain he had felt in his stump after the first amputation. This pain can last up to several hours and feels as the limb did after the first amputation, with pain primarily in the region of the phantom stump and incision. At these times, the phantom limb is reported to be the same length it had been after the first amputation and lacks all the parts below the level of the phantom stump. Similarly, he

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

Case 134: Male, 63 Years Old, Left Below-Elbow Amputation

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

He reports having three other types of pain: a constant burning pain in the stump and hand; an additional superimposed pain that is more intense and that can last for days; and successive "jabs" of pain referred to the stump that can each last up to 30 sec and continue for several hours. Over the years he has received brachial plexus blocks, transcutaneous electrical nerve stimulation (TENS), intensive physiotherapy, and numerous medications. Approximately 10 years ago he sought the help of a psychiatrist with whom he worked for about 1 year. The painful posture and dripping sensation "started to ease off" at about that time, although he is not certain whether to attribute the change to the psychotherapeutic intervention. At the time of the interview he reported, "Now the pain is like a little ball of fire in the stump."

The proportion of amputees who report similar pain before and after amputation may be as high as 79% (Katz & Melzack, 1990), although according to the only prospective study carried out, the incidence is 36% 8 days after amputation and 10% 6 months later. 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 preamputation pain by continuous epidural blockade between 1 and 3 days before amputation decreases the incidence of phantom limb pain 6 months to 1 year later (Bach *et al.*, 1988; Jahangiri *et al.*, 1994).

Compared with pain that is temporally non-contiguous 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). Other studies have not found a relationship between pain prior to amputation and the development of phantom pain (e.g., Sherman *et al.*, 1984). It is our clinical impression that only occasionally are these pain memories as intense as the original painful experience. They are experienced with greater frequency immediately after amputation and tend to fade with time. Although amputees may report the recurrence of a preamputation pain years after amputation, these pain memories do not usually interfere with the patient's everyday life and are readily distinguished from other qualities of phantom pain. It should be noted, however, that there are exceptions to this statement; some patients suffer terribly with persisting pain memories.

Pain memories also occur 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 as a result of 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, 1994; Reisner, 1981a). 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 limbs.

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, 1978b; 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 *et al.*, 1993) and hemorrhoids (Oveson *et al.*, 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). Critchley (1971) stated that 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" (p. 91). Allen (1928) presents a detailed case study of a patient who, on 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' " (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. We do not know what triggers the re-activation of the neural

structures in the brain that subserve these memories, but likely possibilities include output from neuromas, dorsal root ganglia and spinal cord cells.

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 metainformation 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). 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. This unified experience of a pain memory involving activation of both memory components is called the neural matrix of conscious (somatosensory) sensation in Chapter 2.

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 because, 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 *et al.*, 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. Because 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 dissociable forms of memory, one of which (the somatosensory component) is independent of the conscious experience of pain.

Lacroix *et al.* (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 which 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.*, 1991, 1992) 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 may persist as a phantom limb pain memory (Katz and 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. If this is true, then it should be possible to interfere with the formation of a pain memory arising from surgical amputation by blocking both somatosensory and cognitive systems prior to the operation (Fig. 1).

Patients who have sustained traumatic amputation by either 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. 1A). Traumatic amputation would be expected to result in the formation of both the somatosensory and cognitive memory components. The expected outcome would include heightenend 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. However, this has not yet been substantiated.

Amputation performed under general anesthesia alone (Fig. 1B)

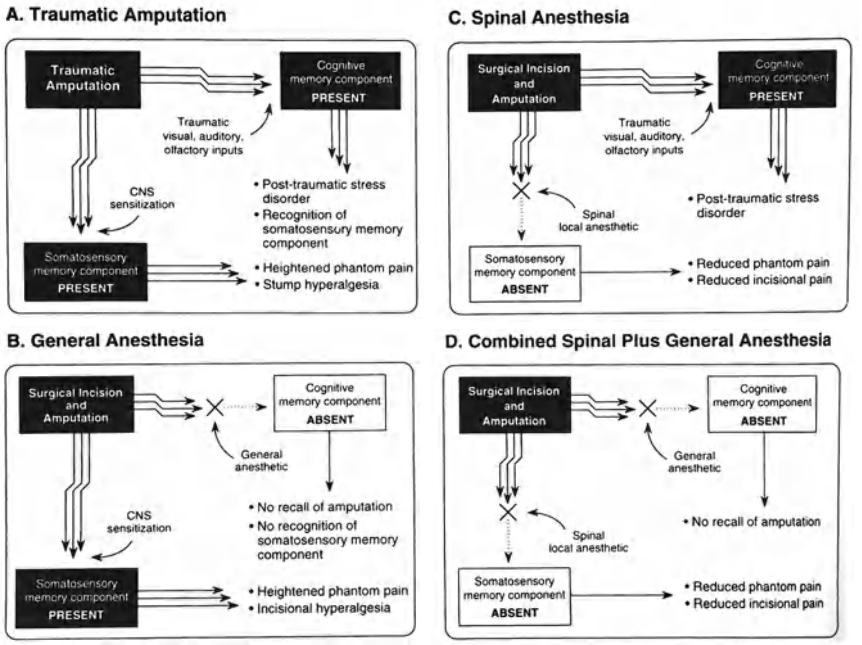

Figure 1. Predicted postoperative pain status and psychological status following traumatic amputation (A) or surgical amputation performed under general anesthesia (B), spinal local anesthesia (C), or combined spinal local anesthesia plus general anesthesia (D). Reproduced from Katz (1993), with permission

would be expected to 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 the patient. Because surgery was performed under a general anesthetic, the patient would not have had any conscious experience of pain at the time of incision and amputation. On 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. 1C) would be expected to 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 memories of operating room events that could develop into a posttraumatic stress disorder.

Combined use of spinal anesthesia and general anesthesia (Fig. 1D) 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 thoracic surgery and lower abdominal surgery show that it is possible to at least partially preempt postoperative pain by interrupting the transmission of nerve impulses between the periphery and the spinal cord. Combined use of general anesthesia plus preincisional epidural administration of an opioid (Katz *et al.*, 1992) or a local anesthetic agent (Katz *et al.*, 1994) is more effective in reducing postoperative pain and analgesic requirements than combined use of general anesthesia plus postincisional administration of the same agent by the same route (see Tables 1 and 2; Fig. 2). These studies support

Table 1. McGill Pain Questionnaire Scores 24, 48, and 72 hr after Surgery for Patients Who Underwent Lower Abdominal Surgery under General Anesthesia plus Lumbar Epidural Bupivacaine before or after Incision[a]

	Preincision		Postincision		
	Mean	(SD)	Mean	(SD)	*p*
24-hr assessment					
n	16		20		
PRI-T	9.6	(13.0)	8.3	(5.6)	NS
PPI	0.7	(0.6)	1.2	(0.7)	0.05
48-hr assessment					
n	14		18		
PRI-T	4.4	(3.4)	7.2	(5.3)	NS
PPI	0.7	(0.8)	1.0	(0.8)	NS
72-hr assessment					
n	10		18		
PRI-T	2.5	(2.4)	7.9	(6.9)	0.003
PPI	0.3	(0.5)	1.1	(0.9)	0.03

[a]Pre- and postincision refer to time of epidural bupivacaine administration. PRI-T refers to the total pain rating index of the MPQ, and PPI refers to the Present Pain Intensity. Adapted from Katz *et al.* (1994) with permission.

Table 2. McGill Pain Questionnaire Descriptors Chosen by 33% or More of Patients Who Underwent Lower Abdominal Surgery under General Anesthesia plus Lumbar Epidural Bupivacaine before or after Incision[a]

Time	MPQ class	MPQ descriptor	Preincision	Postincision
Day 1	PRI-Sensory	Dull	—	40
		Sore	—	40
		Tender	50	55
	PRI-Affective	Tiring	—	35
	PPI	Mild	57	50
		Discomforting	—	35
Day 2	PRI-Sensory	Tender	43	50
		Dull	—	39
	PPI	Discomforting	—	33
Day 3	PRI-Sensory	Dull	—	39
		Sore	—	44
		Tender	—	39
	PRI-Evaluative	Annoying	—	33
	PPI	Mild	33	40

[a]Adapted from Katz *et al.* (1994), with permission.

the idea that surgical incision and subsequent noxious intraoperative events sensitize neurons in the dorsal horn of the spinal cord, i.e., "central sensitization." In the hours and days following surgery, inputs from the wound impinge on a sensitized nervous system, which amplifies the peripheral signal, and leads to enhanced postoperative pain and a greater need for postoperative analgesia. By this interpretation, blockade of the noxious input associated with surgery prevents the development of the somatosensory memory component and results in less intense pain.

In the study by Bach *et al.* (1988) noted above, it was reported that 3 days of continuous epidural morphine plus bupivacaine prior to amputation reduced the incidence of phantom limb pain 6 months later compared with a control group that did not receive an epidural before amputation. These results have been confirmed by Jahangiri *et al.* (1994), who compared a standard general anesthetic plus on-demand opioids for postoperative analgesia with a continuous infusion of epidural diamorphine, clonidine, and bupivacaine beginning 24–48 hr before amputation and continuing for a period of 3 days after, amputation. The study by Bach *et al.* evaluated the long-term effect of eliminating preamputation pain on the subsequent development of phantom limb pain. Their results suggest that pain before

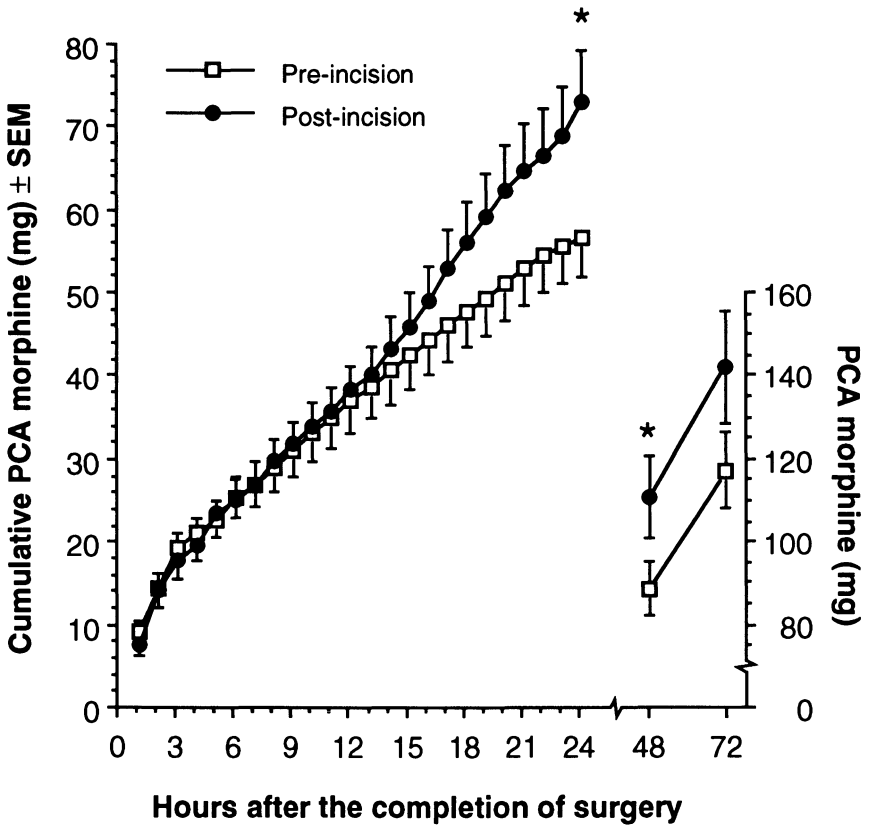

Figure 2. Cumulative postoperative, patient-controlled consumption of intravenous morphine shown for patients who undrwent lower abdominal surgery under combined general anesthesia plus lumbar epidural bupivacaine before (pre) or after (post) incision; $*p < 0.04$. Adapted from Katz *et al.* (1994), with permission.

amputation is, in some way, associated with the development of long-term phantom limb pain.

However, because all patients underwent amputation under epidural analgesia, it does not address the separate but related question of whether blocking the noxious inputs during surgery reduces postoperative phantom limb pain and stump pain. Similarly, the study by Jahangiri *et al.* was geared toward eliminating noxious inputs associated with preamputation pain, perioperative trauma, and postoperative inflammation. Thus, although these studies demonstrate a beneficial effect of blocking noxious inputs pre-, intra-, and/or postoperatively, they do not specifically address

the question of which factor(s) (e.g., pre-, intra-, or, postoperative) are responsible for the development of enhanced phantom limb pain. Further research is required to isolate the relevant factors and their roles in determining pain at various points in time after amputation. In addition, interpretation of these results should be tempered by a number of methodological problems (e.g., small sample sizes, nonrandom assignment of patients to treatment group, insufficient details about pain assessment).

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. We do not know, 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 (Coderre *et al.*, 1993).

Alternatively, deafferentation may merely provide a condition under which persistent CNS activity becomes obvious to the observer because the peripheral source has been removed or its afferent pathway interrupted. This may explain why pain memories are almost exclusively reported to occur in patients with deafferenting lesions and infrequently under other circumstances. Rare examples of recurring pain in the absence of obvious deafferentation include cardiac pain referred not to the chest and left arm as is common but to the site of a compression fracture in the upper back sustained 20 years earlier (Henry & Montuschi, 1978). Another example is pain in response to stimulation of the nasal mucosa, referred to teeth that had recently been filled (Hutchins & Reynolds, 1947; Reynolds & Hutchins, 1948).

If deafferentation or amputation is not a necessary condition for a pain memory to develop, then we must ask why such memories 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 wound), the existence of a central somatosensory component is not even considered. For example, it has been shown that postoperative pain following major surgery is less intense if patients received a general anesthetic plus an epidural opiate or local anesthetic before incision versus a general anesthetic plus an epidural opioid or local

anesthetic after incision (Katz *et al.*, 1992, 1994). These studies suggest that 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 awakes from the general anesthetic. Blocking nociceptive inputs before but not after incision appears to attenuate the development of the central somatosensory component. The obvious source of ongoing pain after surgery (i.e., the wound) 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. It is important to note however, that many studies do not support the hypothesis that preincisional administration of analgesic or anesthetic agents is more effective in reducing postoperative pain than postincisional administration of the same agent by the same route (Niv & Devor, 1993; Dahl, 1994).

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 reproduce the patient's own familiar pain. Although these studies involve 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 (1947a) 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 tendon. Nathan (1962, 1985) reported a similar occurrence 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 discov-

ered 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 a peripheral trigger 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 that 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 because 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 shortly after surgery that they became certain for the first time that their limb had indeed been cut off (Gallinek, 1939; Simmel, 1956). Patients undergoing brachial plexus or spinal blocks, those with complete brachial plexus avulsions, and those with spinal cord transections report vivid phantom limbs that 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 on somatosensory input alone, additional information is sought via the visual sense, which usually determines the perceptual experience. Because 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 because the potential inhibitory effect of vision has also been removed. In general, as the number of modalities involved in the preamputation pain experience increases (and thus, more sources of potential feedback are removed), the greater is the probability of reactivating a past pain once the limb has been removed because 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). Rather, it is believed to be generated on a moment-by-moment basis, determined, in part, by current sensory input and cognitive–evaluative processes (see *Cognitive and Affective Processes*, below).

The Use of Language

Language may play an important role in the development and reactivation of pain memories because 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 experience 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."

It is hypothesized that the formation of a higher-order polymodal representation of the pain is facilitated by the unifying verbal response that 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 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 pain that can not be explained on the basis of current physiological and anatomic knowledge as psychological in origin (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 the 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) describe the case of a soldier who sprained his ankle jumping from a truck and therefore could not keep up with his companions. Shortly afterwards, 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 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) describe 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 discuss the importance of "both psychical and physical trauma" at or near the time of amputation and conclude that the persistence of preamputation pain represents an "obsession neurosis."

These case reports raise the possibility that emotional and psychological disturbance 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. that, prior to amputation, are rarely considered psychologically important to the patient.

The only study to compare amputees 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 pain failed to find any significant intergroup differences in depression, anxiety, or personality characteristics (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 amputees who reported phantom limb pain of any type compared to their pain-free peers. 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, 1955a, 1955b; 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 pa-

tients 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 corresponding 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 any more 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 and that memory is enhanced when mood state during recall matches that during acquisition (Singer & Salovey, 1988). 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, thus biasing attention, information processing, and memory functioning in favor 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 require 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. Rather, 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 ability to cope, and other declarative information.

Chapter 6

Physiological Correlates

Richard A. Sherman

Physiological Correlates and Precursors of Phantom Limb Pain

If phantom limb pain is a referred pain syndrome similar to sciatica, specific changes in physiology such as blood flow, nerve conduction, and muscle tension that influence neural activity in the periphery ought to affect phantom pain. On the other hand, if the impulses responsible for phantom pain originate exclusively in the CNS, such correlates might not be found or might be only very indirectly associated with changes in the pain.

Considerable research has been performed that demonstrates that a variety of central and peripheral physiological markers differ when phantom pain is present or absent. Some of the studies have compared pain-free amputees with those who normally report phantom pain, and others have compared amputees during pain-free and painful periods. A few of the studies have been able to establish physiological precursors specific to a variety of descriptions of phantom limb pain that change before phantom pain changes in intensity. Several studies have purposely changed the activity levels of the physiological precursors and found corresponding changes in the levels of phantom pain. These studies provide direct support for physiological mechanisms underlying phantom pain.

Burning–Tingling–Throbbing Phantom Limb Pain

Reduced near-surface blood flow to a limb has been implicated as a predictive physiological correlate (first cousin to a cause) in many pain conditions including causalgia and reflex sympathetic dystrophy (Karstetter & Sherman, 1991). Return of blood flow to normal patterns through any intervention, including time alone, usually results in either the complete

cessation or a significant decrease of the pain (Sherman, Arena, Searle, & Ginther, 1991). If phantom limb pain is a referred pain syndrome, anything affecting the nerve endings in the residual limb is likely to affect phantom pain as well. In Chapter 3, we summarized a large number of studies that demonstrated that the ends of the nerves that used to serve the amputated limb are still sensitive to stimuli. Of particular importance to the topic of the present chapter are the observations that (1) cooling the nerve ends causes increased firing rates in C-fibers, and (2) reducing blood flow to the extremity results in exaggerated ectopic firing both because of ischemia and because of cooling (Campbell, 1987; Jänig, 1987; Haber, 1955; Korenman & Devor 1981; Koschorke, Meyer, & Campbell, 1987; Matzner & Devor, 1987; Sherman & Arena, 1992).

Cronholm (1951) quotes Pitres (1897) as having stated that the perceived temperature of the phantom is related to the temperature of the stump. Measurements of skin temperature in amputees have been made since at least 1952 (Studies relating ..., 1952). This study found that amputees' stumps were cooler at the distal end than corresponding points on the intact extremity. It also found that the cooler areas on the stump did not warm up when attempts were made to increase cutaneous blood flow through such approaches as giving the subjects whiskey to drink. Wahren (1990) reviewed the propensity of finger amputees to be very sensitive to the cold and for their pain to be aggravated by cold environments. They found that the residual portions of the fingers were cooler than corresponding areas on the intact hand and were more sensitive to pain in response to cooling. Kristen *et al.* (1984) reported using videothermographic recordings of temperature in the residual limbs of 50 amputees to detect phantom pain. They found that most amputees having phantom pain showed different patterns of temperature than those who did not.

A consistent inverse relationship between intensity of phantom limb pain and temperature in the residual limb relative to the intact limb has been demonstrated for burning, throbbing, and tingling descriptions of phantom pain but not for any other descriptions (Sherman & Bruno, 1987).

> It has also been established that (a) for these descriptors of phantom pain there is a day-to-day relationship between the relative amount of blood flow in the stump and pain intensity and that (b) there is an immediate change in pain when blood flow changes. However, this does not mean that the changes in blood flow cause the change in pain. It is possible that a change in pain intensity causes a physiological chain reaction [that] eventually causes a decrease in blood flow to the stump. This is improbable for several reasons. Although videothermographs normally record only near-surface blood flow patterns, hands are thin enough so that thermographs can record blood flow patterns throughout the hand. In four cases of burning or tingling phantom pain following a finger amputation, blood flow changed only in the area just proximal to

the amputation site. The rest of the hand was essentially unchanged, and there were no changes in the paired area of the intact hand. If the blood flow change were a result of a spinal reflex response, we might have expected a bilateral change. Alternatively, if unilateral, the entire dermatome containing the stump ought to have cooled off. This was not the case. Therefore, we conclude that a reflex reaction is not likely. The subjects were taught to increase blood flow in the stump by using temperature feedback to relax and thus dilate the peripheral blood vessels. Increasing peripheral blood flow to the cool area of the stump resulted in a decrease in the pain intensity. If the decrease in blood flow was [in response] to an increase in pain, blood flow would decrease for all descriptors of phantom pain, not just a consistent few (Sherman & Arena, 1992).

The relationship of burning phantom pain to blood flow in a bilateral amputee is illustrated in Figure 1. The subject reported that phantom pain was present in the relatively cool residual limb but not in the warmer limb.

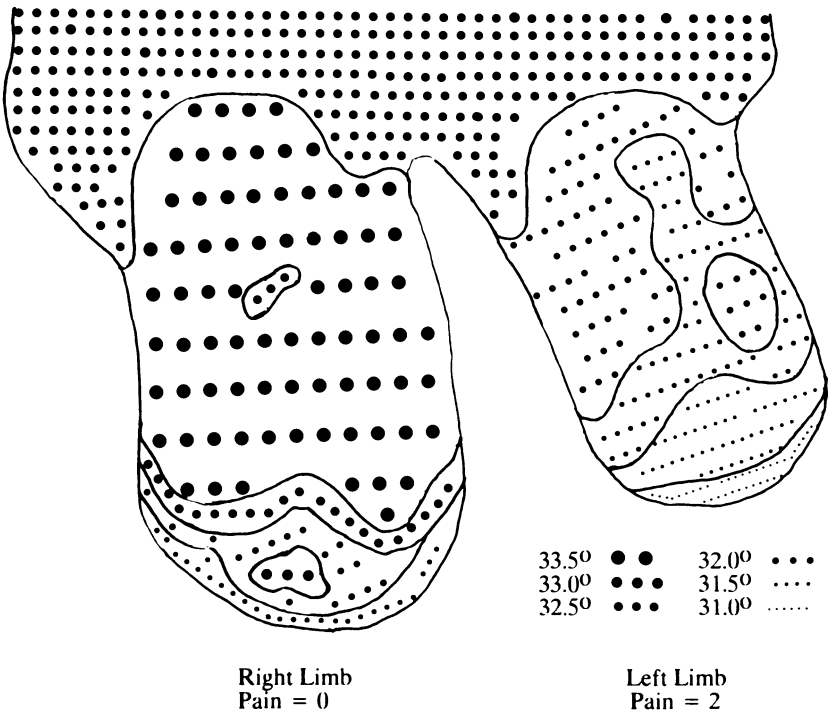

Figure 1. Redrawn color videothermogram of an above-knee bilateral amputee's residual limbs. The right residual limb had a pain level of 0 while the left residual limb had burning phantom pain rated at an intensity of 2 on a scale of 0 to 10. The size of each dot represents the temperature in degrees Celsius recorded on the skin's surface. Differences of less than 1°C are within normal limits.

This and numerous thermograms like it form the key evidence that the decreased blood flow associated with burning phantom pain is not a consequence of general sympathetic hyperactivity. Only the painful residual limb shows decreased blood flow; the other residual limb maintains its temperature. Figure 2 illustrates the increase in intensity of burning phantom pain with progressive decreases in surface blood flow in the residual limb. This tight relationship has been observed numerous times (Sherman & Bruno, 1987) and indicates that there is more than a casual relationship between the two.

> The existence of a vascular related mechanism for burning phantom pain is also supported by the short-term effectiveness of invasive procedures, such as sympathetic blocks and sympathectomies, which increase blood flow to the limb. These reduce the intensity of burning phantom and stump pain but do not change other descriptors (Sherman, 1980; Wall, 1981). It is indirectly supported by the virtual ineffectiveness of every surgical procedure involving severing nerves either in the spinal cord or running between the amputation site and the spinal cord (Sherman & Sherman, 1985; Wall, 1981). Beta blockers such as propranolol cause dilation of peripheral blood vessels and have been reported to be successful in ameliorating phantom pain at least in the short term (Marsland *et al.*, 1982; Sherman & Arena, 1992).

Relationships between muscle tension and burning phantom limb pain (Sherman & Bruno, 1987) have been shown to be largely a result of the change in near-surface blood flow that accompanies increased muscle tension (Laughlin & Armstrong, 1985; Richardson Schmitz, & Burchers, 1986).

Cramping–Squeezing Phantom Limb Pain

Cronholm (1951) quotes Amyot, Livingston, and others as having noted increased muscle tension and spasms in the stumps of amputees. He found that 51 of 99 amputees questioned about stump muscular activity reported spontaneous hyperactivity.

Onset and intensity of cramping and squeezing descriptions of phantom pain are related to muscle tension in the residual limb. A variety of studies have demonstrated that intensity of cramping phantom pain and amount of muscle tension in the residual limb change together both from day to day (Sherman and Arena, 1992) and from moment to moment (Sherman et al., 1992a).

Changes in surface electromyographic (EMG) representations of muscle tension in the residual limb precede changes in cramping and squeezing phantom pain by up to several seconds. This relationship does not hold for any other descriptions of phantom pain. The method for establishing this relationship is illustrated in Figure 3, and a typical recording from a bilateral amputee is illustrated in Figure 4. The actual statistical relation-

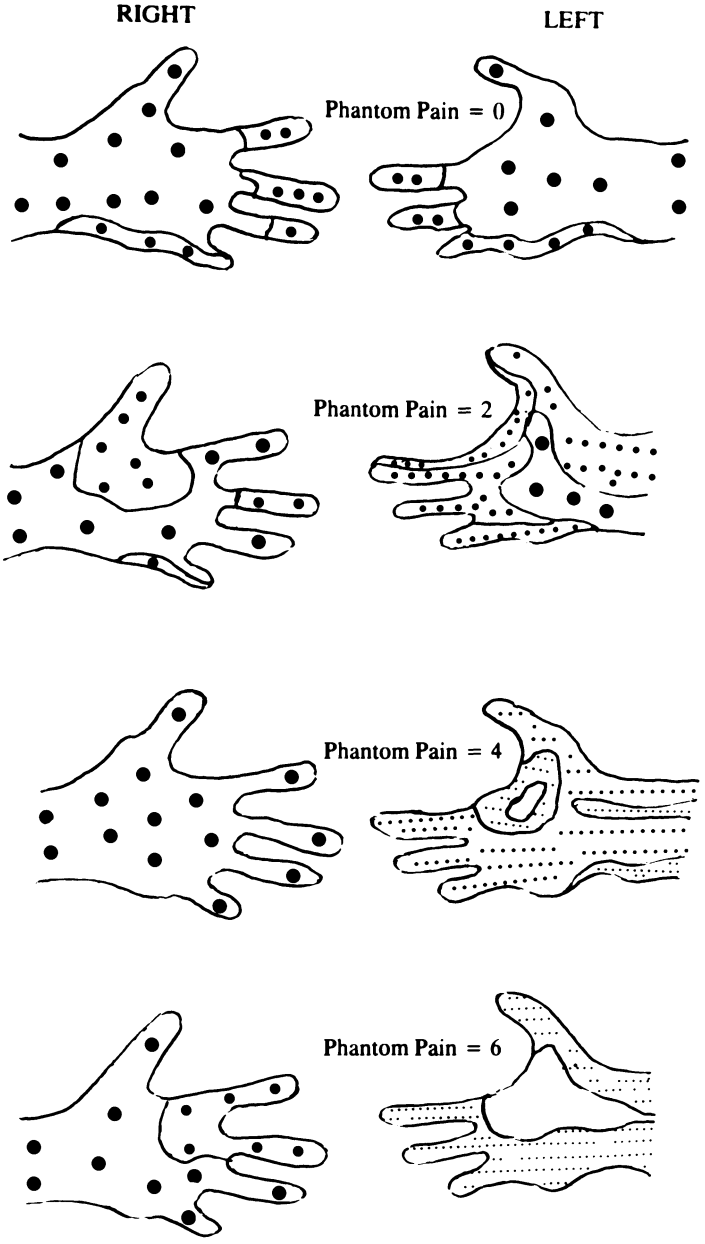

Figure 2. Redrawn color videothermograms of an amputee missing the index finger on the left hand. Size of each dot represents relative warmth at the skin's surface, with the largest dots showing the most warmth and blank areas being coolest. Blank areas are essentially the same temperature as the surrounding room. Burning phantom pain intensity is rated on a scale of 0 to 10.

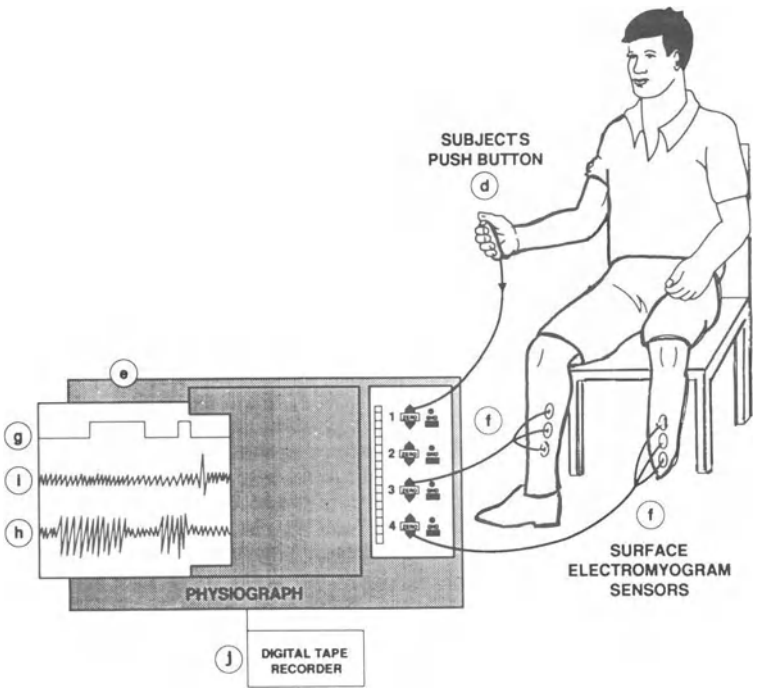

Figure 3. Methodology used to establish temporal relationships between change in phantom pain intensity and change in surface EMG of the residual limb. Both of the subject's limbs are instrumented for recording of surface electromyographic (EMG) representations of muscle tension (f). The signals go to a chart recorder (e), where they are displayed with line i showing the signal from the intact limb and line h showing the signal from the residual limb (stump). The subject presses a button (d) when the intensity of phantom pain increases and releases the button when the intensity returns to its prior level. The button press signal is displayed on line g. The entire session is recorded both on paper and by a digital tape recorder (j). This figure is a revision of one from Sherman and Sherman (1991). Reprinted from *APS Bulletin*, *1*(4), with permission of the American Pain Society, Skokie, IL 60077-1057. Copyright 1995, American Pain Society.

ships for each patient are presented in Table 1. The critical point illustrated is that the amputee shows changes in muscle tension only in the painful residual limb. Surface EMG in the pain-free residual limb does not change significantly. If the change in EMG was simply a reaction to the change in pain, the change in EMG would have followed, rather than preceded, the change in pain, and at least some change in muscle tension in the pain-free limb should have been observed, as would be expected from a generalized withdrawal reflex from pain.

Relationships between overall muscle tension in the residual limb and cramping phantom pain have also been shown to hold throughout the day

Figure 4. Samples from a chart recording showing surface EMG from the residual limb and button-press responses to changes in cramping phantom pain intensity made by a bilateral amputee. Reprinted from Sherman *et al.* (1992a) with permission of Elsevier Publishers.

Table 1. Temporal Relationships between Phantom Limb Pain and Muscle Tension in the Residual Limb[a]

Phantom pain description	Most common relationship between change in EMG and change in phantom pain	Number of times pain changed	Number of times change was in most common direction	Time (sec; mean/S.D.) between EMG change and pain change	99% confidence limits
Cramping					
1	EMG changed before phantom pain changed	22	21	2.0/2.2	0.8–3.2
2	EMG changed before phantom pain changed	24	20	0.8/0.9	0.3–1.2
3	EMG changed before phantom pain changed	6	6	1.7/0.8	0.9–2.6
4	EMG changed before phantom pain changed	5	5	1.2/0.5	0.6–1.9
5	EMG changed before phantom pain changed	19	19	1.8/1.1	1.1–2.4
6	EMG changed before phantom pain changed	12	12	1.5/1.0	0.7–2.3
7	EMG changed before phantom pain changed	16	16	0.7/0.5	0.4–0.9
Burning					
1	Close to same time				
2	EMG did not change				
3	EMG did not change				
Shocking–shooting–stabbing					
1	EMG did not change				
2	EMG did not change				
3	Minor changes in EMG				
4	Minor changes in EMG				
5	EMG occasionally changed before pain changed	27	7	0.3/0.9	−0.2–0.8
6	No changed in EMG				
Combinations of descriptions					
Shock/burn/stab	Close to same time				
Cramp/shock	EMG change before pain change	10	10	2.9/0.4	2.6–3.2
Cramp/burn/shock	EMG change before pain change	4	3	7.3/7.1	−3.3–17.8
Cramp/burn/shock	EMG change before pain change	36	16	4.5/9.7	0.3–8.8

[a]Data abstracted from Sherman *et al.* (1992a). Only subjects who showed changes in pain during the recording session are included in this table.

in subjects' normal environments (Sherman, Evans, & Arena, 1993). Figure 5 illustrates these relationships in two amputees with cramping phantom limb pain who wore an ambulatory recording device for 2 days. The device was capable of recording surface EMG from the residual limb and button press representations of pain intensity (Sherman, Arena, *et al.*, 1991). The relationship between cramping phantom pain and muscle tension in the residual limb is supported by the consistent success of treatments resulting in reduction of residual limb muscle tension for cramping phantom pain but not for other descriptions (Sherman 1976; Sherman, Arena, *et al.*, 1991, 1992a,c).

Numerous amputees report that cramping phantom pain decreases with any activity that tends to decrease muscle contraction levels in the residual limb and increases with activities increasing overall levels of contraction. Thus, such activities as phantom exercises that result in changes in muscle tension in the residual limb can result in temporary changes in intensity of phantom pain. Gessler (1984) reported that when the muscles of the residual limbs of ten amputees with chronic cramping phantom pain were relaxed, the phantoms felt as though they were opening.

Shocking–Shooting Phantom Limb Pain

This family of descriptions is the least common of those usually encountered in our clinical and research experience. With three exceptions, neither we nor other writers have found any relationships between changes in any physiological variables and either onset or change in these descriptions of phantom pain. One exception is based on a 3-year log kept by an amputee who experienced shocking phantom pain. He noted the date, time, and intensity of each episode and sent logs to us each month. These relationships are depicted over time in Figure 6. A major decrease in both intensity and frequency of episodes occurred after he began a combination of (1) preventing the limb from cooling off rapidly and (2) performing limb-warming exercises (Sherman, Evans, Caminer, Sherman, & Wolf, 1993). This is especially interesting because none of our clinical patients has received substantial benefit from any therapeutic intervention for this description of phantom pain. This has been the case even among those amputees with several descriptions of phantom pain. Pains having other descriptions were usually relieved without a corresponding decrease in the shocking–shooting description. Our clinical results parallel the results we have deduced from the literature (e.g., Sherman *et al.*, 1984).

The other potential physiological correlates are related to (1) compression neuropathy and (2) mechanical stimulation of the neuroma. They are discussed below.

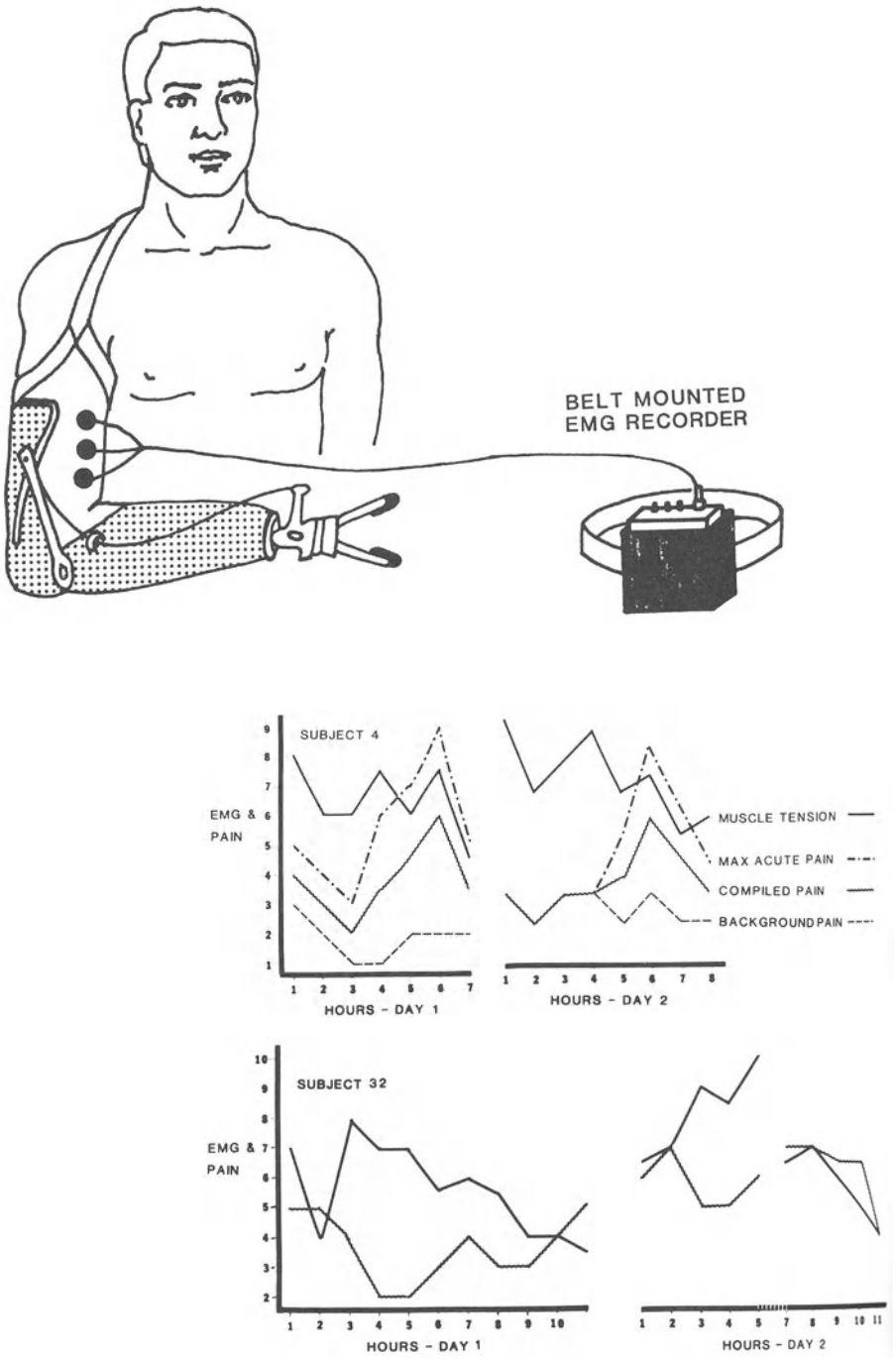

Figure 5. Relationships between cramping phantom pain and muscle tension in the residual limb recorded in subjects' normal environments.

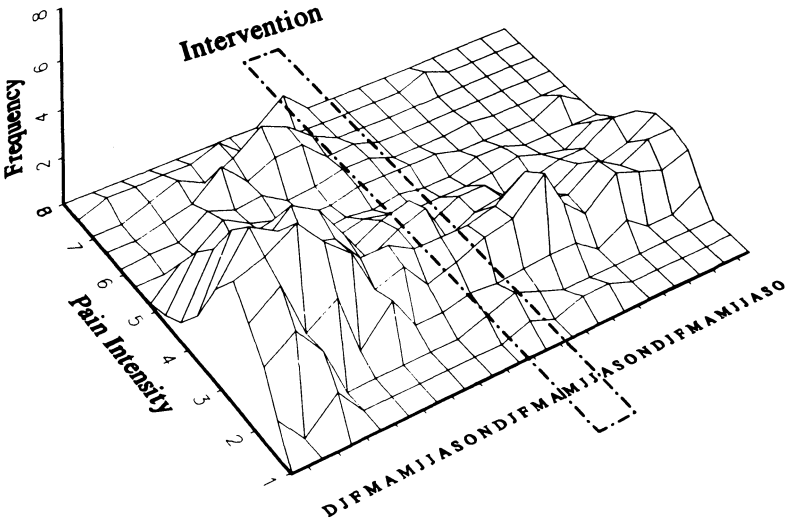

Figure 6. Relationships among frequency of episodes, intensity of shocking phantom pain, month of the year, and intervention.

Potential Sites of Referred or Neurogenic Pain

Tactile sensitivity in the residual limb is much greater than in corresponding areas of the intact limb. This holds for touch–pressure threshold, two-point discrimination distance, and ability to localize a point on the limb (Haber, 1955; Teuber *et al.*, 1949). Cronholm's (1951) careful study of relationships between pressure point stimulation of various parts of the body—especially the residual limb—and corresponding changes in shape and perception of the phantom leave little doubt that stimulation of the skin can result in changes in the phantom.

Numerous workers have asked amputees what sorts of changes in themselves and their environments alter the intensity of phantom limb pain or initiate episodes. For example, the 1952 report discussed in Chapter 2 (Studies relating ..., 1952) found 11 factors that various amputees reported as affecting their pain. These are summarized in Table 2. This group also performed segmental injections with 0.5 to 1.0 cc of 6% sodium chloride into the interspinous tissue. The point of injection was located by correlating the injection point (as marked by a lead pellet on the skin) with x-rays of the spine. The needle was inserted into tissues between two vertebrae. When the injection was into the L4–5 segment, there was a rapid onset of pain close to the site of injection and then radiation into the buttocks and posterolateral aspect of the thigh. This included a rapid "filling" of the

Table 2. Precipitating Factors from the 1952 Manuscript Studies[a]

	Phantom pain	Stump pain	Both
Irritation of stump	15	9	6
Activity	4	4	1
Fatigue	12	8	4
Rest	5	2	1
Alcohol	6	5	1
Weather and temperature	24	9	5
Prosthesis	7	8	2
Posture	6	4	2
Movement of phantom	4	0	0
Sphincters	1	1	0
Worry	4	3	1

[a]Number of amputees reporting each factor.

absent areas of the phantom limb. They give an example of an above-knee (AK) amputee who could usually sense his phantom foot but not the rest of the missing limb. The entire limb shape filled in and became clear during the injection. The phantom pain in his foot also spread into the newly "sensed" area. They also tried to decrease phantom pain by injecting between 2 and 10 cc of a 1% solution of procaine into the same spots that caused the pain to intensify. They state that no major nerve trunks were blocked during the procedure and that pain but not shape of the phantom could be reduced by the injection. They reported that cold phantoms appeared to warm up while cramped phantoms appeared to relax.

Sherman *et al.* (1985) found that 26% of veterans and 48% of civilians had no idea what influenced their pain. The rest reported influence by various aspects of the weather (48% and 14% of veterans and civilians, respectively), chronic problems with their prostheses (8% and 7%), mental stress (6% and 7%), fatigue (4% and 12%), intestinal and back problems (2% and 1%), and problems in the residual limb (26% and 48%). Jensen *et al.* (1983) found that phantom pain could be initiated by emotional distress, stump pressure, urination, cold, and coughing. It is important to note that problems initiating pain in one amputee do not do so in another, so there are many potential sources of referral to sort out.

Problems in the Residual Limb

The only consistent relationship between problems in the body and phantom pain demonstrated by our large amputee surveys was a concur-

rent occurrence, and frequently a concurrent change in intensity, of phantom and stump pain (Sherman *et al.*, 1984; Sherman & Sherman, 1985). Increased use of, irritation of, and pain in the residual limb correlated with greater frequency of episodes and increased intensity of phantom pain. We have observed this phenomenon among many of our patients who report both problems. An unusual example of this phenomenon was reported by Sugarbaker, Weiss, Davidson, and Roth (1984), who found that phantom pain became worse in two of their patients when cancers recurred in their residual limbs. It is likely that neuroma afferents and spinal neurons that originally had receptor fields in the amputated area become sensitive to changes in the residual limb such as mechanical compression by a tumor. The evidence for mechanosensitivity of severed nerve endings in the stump was discussed in Chapter 3. Thus, when the residual limb is stimulated, signals will reach the brain's homonculus at the site corresponding to the stimulated site on the residual limb as well as at the site that corresponds to the amputated area. In Chapter 2, we also reviewed the evidence that nerve ends in the residual limb are abnormally sensitive to a range of other stimuli. This could mean that nerves that used to serve the amputated area respond to chemicals normally released into the extracellular fluid by minor trauma and irritation occurring during relatively heavy use of a well-fitting prosthesis. It could also spell agony when the stump is irritated by a poorly fitting prosthesis or when sores develop.

> Campbell (1987) has indicated that nerve injuries sometimes cause shocking–shooting descriptions of pain among patients and that they are not altered by sympathetic blocks (which would increase blood flow to areas near the neuroma). Thus, any factors [that] change the temperature environment around the neuroma or result in a change in its mechanical stimulation, should alter phantom sensations. Changes in blood flow would tend to alter burning sensations, while mechanical stimulation from excessive use of the prosthesis (or an improperly placed prosthesis) with changed pressure should result in changes in several types of phantom pain including shocking–shooting pain. Jänig (1987) showed that there is considerable cross-talk in neuromas between efferent sympathetic nerves and afferent nocioceptors in the same fiber. Thus, many different stimuli in the body or environment (such as stress) that increase sympathetic tone should also increase phantom pain. (Sherman & Arena, 1992)

Compression Neuropathy

Nyström and Hagbarth (1981) found that distorting the neuroma formed near the end of the residual limb caused "sharp" phantom pain. The most recent 17 amputees to report shocking–shooting phantom pain in our clinic reported that their pain changed with (1) position, (2) sitting in one position for too long (lower limb amputees only), (3) bowel move-

ments (for seven of the amputees), (4) fatigue (for five), (5) extended use of the prosthesis (for five), and (6) with changes in weather (especially changes in barometric pressure and before storms) for five. Two of those sensitive to position reported that pressure on the buttock triggered the pain. These amputees were diagnosed by both orthopedics and rehabilitation medicine as having compression neuropathies. None of our other amputees usually report similar effects.

Effects of the External Environment Mediated through the Body

Twenty-five amputees kept a daily log of their phantom pain and factors affecting it (work, standing time, fatigue, stress, various aspects of the weather, etc.) for a year, and 117 kept logs for 6 months. There was a predictive relationship between changes in barometric pressure and changes in phantom pain intensity for many of the amputees (Arena, Sherman, & Bruno, 1989). Similar relationships between pain and barometric pressure have been well demonstrated for conditions such as arthritis (Sherman & Arena, 1992).

Use of the Lower Limb and Phantom Pain

Data from the above logs showed that, among lower limb amputees, phantom and stump pain are frequently directly related to length of time standing. The stump frequently has relatively poor venous return and tends to swell when the amputee stands relatively still for prolonged periods. The data also indicate that phantom pain increases when the stump is subjected to sustained mechanical distortion resulting from heavy work and extensive walking over rough terrain (Arena, Sherman, Bruno, & Smith, 1988). Increased swelling probably causes an increase in pressure on small blood vessels, which, in turn, decreases blood flow and, thus, cools the nerves. Increased swelling probably causes an increase in pressure both directly on nerve ends and on the small blood vessels. Decreased blood flow, in turn, cools the nerve ends and produces a local ischemia. All three effects (mechanical pressure, cooling, and ischemia) are known to increase ectopic neural firing (see Chapter 3). Sixty-three percent of the amputees who kept their logs for a year had a relationship between phantom pain and physical activity (Sherman & Arena, 1992). Thus, it is likely that decreased blood flow, burning phantom pain, and swelling caused by changes in barometric pressure, position, etc. are all interrelated.

In a pilot survey of 42 healthy people who had traumatic amputations within the last 10 years, all reported highly significant problems using their prostheses for work. Virtually all had phantom and stump pain directly

related to the use of the prosthesis. The pain was clearly related to physical problems caused by the inability of the prostheses' sockets to support the body during activity. The junctions between even the best fitting sockets and the residual limbs simply are not able to withstand the pressures associated with sustained motion.

Chapter 7

Psychological Factors Influencing Phantom Pain

Richard A. Sherman

Until just a few years ago, the body of literature related to phantom limb pain presented a misleading picture of the psychological status of amputees with chronic phantom limb pain. This situation probably arose because (1) many of the authors based their work and conclusions on unintentionally biased samples drawn from those few amputees requesting treatment and on unsubstantiated assumptions about the general population of amputees, and (2) the frequent failure to differentiate clearly between acute adjustment reactions following amputation and chronic problems.

The behavioral science literature suggested that significant phantom limb pain was rare (Kolb, 1954; Parkes, 1973; Sternbach, 1968) and usually characterized it as being a manifestation of either some mental–emotional problem or as one of basic personality structure. The mental and emotional problems were usually said to include unresolved grief (Parkes, 1975), depression (Lindesay, 1985), psychosomatic manifestation of an unstable personality (Gillis, 1969, as quoted by Dawson & Arnold, 1981; Sherman & Sherman, 1983), and psychopathological misinterpretation of ordinary phantom sensations (Schilder, 1935, as quoted by Abt, 1954). The etiologies based on personality structure usually included great rigidity with compulsive self-reliance (Parkes, 1973) and psychosomatic style (Morgenstern, 1964). These characteristics are still accepted in many areas. However, the rigidity observed among some amputees with phantom pain is probably the result of a selection bias because patients with phantom limb pain who are not actively seeking treatment cannot be distinguished from those without pain on a measure of psychological rigidity (Katz & Melzack, 1990).

As detailed in the proceeding chapter, there is clear evidence that phantom pain is related to physiological phenomena in that (1) certain descriptions of phantom pain are related to specific physiological precursors, (2) phantom pain changes in intensity after the specific physiological parameter related to that description changes but not when other physiological phenomena not related to that description change, (3) phantom pain does not change when several physiological parameters sensitive to anxiety and depression change, and (4) changing the physiological precursors of phantom pain results in its immediate resolution. None of these events, especially immediate resolution, would be anticipated if phantom pain were a manifestation of some aberrant psychological process.

As has been demonstrated for virtually all other chronic pain states, phantom pain appears to be influenced by stress, depression, and anxiety. For example, Arena, Sherman, and Bruno (1990) had 27 amputees reporting frequent episodes of phantom limb pain keep daily logs for 6 months. They found that 37% showed a consistent precursor relationship between changes in situational stress and changes in perceived intensity of phantom limb pain. Dawson and Arnold (1981) interviewed ten amputees with painful phantoms and found that the perceived intensity of phantom pain increased with situational stress but was not related to preamputation pain. Hill (1993) recently found similar relationships among 60 amputees evaluated for relationships between coping strategies and perceived intensity of phantom pain. Thus, an understanding of the actual role of psychological factors in maintenance and perceived intensity of phantom pain for the average, psychologically intact amputee is critical to assisting amputees to adjust to their amputation and to the chronic pain that is a frequent and unfortunate sequela. Weiss, Fishman, and Krause (1971) concluded that the amount of disability from an amputation (e.g., above- versus below-knee) is related to subsequent psychological health.

Why Chronic Phantom Pain Would Be Viewed as a Psychological Problem

Phantom pain appears to be a typical example of those chronic pain states in which health care providers are pushed to the limit by predictably human reactions to the frustrations resulting from lack of success (Hendler, 1982). Frustration on the part of both health care providers and their patients influences the doctor–patient relationship and referral patterns. Most people have difficulty understanding how pain from a severed limb could have a very real physiological basis. For this reason, the patient is suspected of having a psychological problem manifested either consciously or unconsciously through the pain. Skepticism is increased by

such well-publicized folklore as amputees who suffered from phantom limb pain until a buried, itching hand was cremated or until the ashes of a cremated hand, which felt as though it was burning, were scattered in a cold lake. Prior to the demonstration of mechanisms for referred pain, such as sciatic leg pains arising from lumbar nerve root compression, health care providers had no reason to believe that phantom pain was not a creation of the amputee's imagination.

Many health care providers probably view the origin of phantom pain as at lease partially emotional or psychological not only for the reasons noted above. Other reasons include accurate findings such as Kolb's (1954) that episodes of pain can be intensified or started by stress, and statements such as Riddoch's (1941) that fear, fatigue, and insomnia precipitate episodes of phantom pain. However, these factors are recognized as initiating and intensifying episodes of most types of chronic pain with known physiological bases (Weisenberg, 1975). The finding that phantom pain responds in a manner similar to other chronic pains with recognized organic bases should have decreased the perception that phantom pain is largely psychological or emotional in origin. This does not appear to have happened.

In a survey of veteran amputees (Sherman & Sherman, 1983), 69% of the 2700 people responding stated that their physicians had directly stated or had clearly implied that the pain was just in their heads. The great majority of amputees responding to this survey were afraid to tell their physicians that they had phantom pain for fear that the physician would think them insane. They were afraid to jeopardize the critically important relationship with their physician or to risk losing credibility in reporting stump problems at a stage when verbal report is frequently the only evidence that problems exist. Stump problems are frequently painful and can entirely prevent the use of a prosthesis for extended periods of time unless intervention is begun prior to development of obvious skin breakdown and other highly noticeable effects. This would account for differences in the reported rate of phantom limb pain such as Kolb's (1954) finding that 0.5% of amputees in a large clinic initially reported having phantom pain but that 5% admitted having it when asked directly.

Sternbach's (1982) analysis of the literature suggested that a 0.5–10% incidence for chronic phantom pain was the accepted norm at that time. The above survey (Sherman *et al.*, 1984) was carried out independent of health care provision and showed that 85% of respondents reported experiencing significant phantom pain. This survey had a 61% response rate. Because 85% of the respondents reported phantom pain, a minimum of 51% of the surveyed population had phantom pain even if all nonrespondents were free of phantom pain.

Sherman and Arena published the following analysis of the major

theories relating characterological personality characteristics to phantom pain in 1992:

> Franz Alexander (1950) has summarized the traditional theory of psychophysiological disorders. It is a contribution of psychoanalytic thought to psychosomatic illness. Four factors entered into Alexander's model. The first factor was a specific personality type or style which leads one to express emotional stress physiologically, generally a neurotic personality. "Corresponding to every emotional situation there is a specific syndrome of physical change, psychosomatic responses ... [in] neurotic patients ... under the influences of prolonged emotional disturbances, chronic disorders of the body may develop" (pp. 39–40). The second component was a psychoanalytic psychosexual stage that the individual was functioning at when the emotional stress occurs. The third factor is the particular kind of psychological stress or conflict involved. For example, headaches and other "cardiovascular disorders" are due to unexpressed, unconscious rage, whereas gastrointestinal disorders are due to fears [that] come about as a threat to dependency needs, and phantom limb pain comes about as castration fears. The fourth factor was one of heredity, which "prewired" the individual to experience emotional stress in a particular organ system ("organ weakness"). To Alexander, disease was a function of (a) birth injuries, (b) nature of infant care (e.g., toilet training), (c) hereditary contributions, (d) organic diseases of infancy (these heighten the vulnerability of certain organs), (e) accidental physical traumatic experiences of infancy and childhood, (f) accidental emotional traumatic experiences of infancy and childhood, (g) emotional climate of family and specific personality traits of parents and siblings, (h) later physical injuries, and (i) later emotional experiences in intimate personal and occupational relations (Alexander, 1950, p. 52). These factors, according to Alexander, contribute to *all* disease, not just those traditionally thought of as psychosomatic/psychophysiologic. One difficulty with this account, however, is that it is based on Cannon's (1920) theory of emotional specificity, which states that patterns of physiological responses are specific to particular emotional states. Although Ax's (1953) classic demonstration of distinctive patterns of somatovisceral reaction during intense emotional states supported Cannon's theory of emotional specificity, a large number of recent studies do not support this hypothesis. They conclude that there is no specific physical response to a specific emotion [see Greenfield and Sternbach (1972) for an excellent overview of the research].
>
> The personality theory of phantom limb pain postulates that individuals who possess certain personality traits will develop specific psychosomatic/psychophysiological disorders. This theory is really a variant of Franz Alexander's (1950) emotional specificity hypothesis of psychosomatic disorders with some major changes (e.g., no hereditary "organ weakness" is required). The personality theory also postulates that amputees with phantom limb pain either (1) have had a previous close association with an amputee [that] arouses fantasies of personal mutilation, which are controlled by repression [and] reemerge when they themselves become amputees (e.g., fear of castration) or (2) hostile dependency on one or both parents: The role of identification with another amputee in the maintenance of the painful symptom led to the consideration of the possible fantasy life of our patients in regard to their own body and body parts. In a number of instances, patients have disclosed to us super-

stitious, yet terrifying, rationalizations to explain the existence of the phantom limb. "... [For example, one] frightened boy overacted emotionally to the existence of the phantom illusion by the superstitious rationalization that the sensations perceived by him were due to fantasized burning of the amputated leg. In another patient, a butcher by trade, the loss of the limb led to the revivification of a horrifying experience which aroused cannibalistic thoughts when he was asked by an alcoholic to supply a fresh human ham. In association with such fantasies, the patient attempts to master his grief and mourning over the loss of the amputated part" (Kolb, 1954, p. 37).

The major problem with the emotional specificity and personality theories of phantom limb pain is that they are based on anecdotal case reports or uncontrolled clinical interviews. This situation is similar to theories of other disorders such as headache that attempt to explain the pain as primarily of psychological or emotional origin (Blanchard, Andrasik, & Arena, 1984). The few studies that have assessed amputees with phantom limb pain using well-validated, objective psychological measures do not support the idea that the pain is psychological in origin (Katz & Melzack, 1990). Important methodological flaws in the phantom pain literature include (1) selection bias of the patients sent to mental health professionals and (2) confounding acute adjustment reactions to amputation with basic personality structure. On the basis of the available literature, it is impossible to rule out personality factors as being involved in the etiology and/or maintenance of phantom limb pain. However, because of the methodological problems outlined above and the absence of a causal relationship between psychopathology and pain for other chronic pains, it seems likely that characterological factors and personality traits do not cause phantom limb pain.

To adequately evaluate the personality theories of phantom limb pain, one would need to (1) have sample sizes of at least 25 per group, (2) include a control group of amputees without significant phantom limb pain, (3) examine acute and chronic phantom limb pain subjects, (4) have comparison groups of other chronic pain disorders, (5) use objective and well-validated psychological tests such as the MMPI, and (6) examine the various types, based on pain descriptors, of phantom limb pain patients (e.g., burning versus cramping versus shooting). We are currently in the process of conducting such a study.

As described above, the literature has failed to support the hypothesis that specific personality characteristics of amputees with phantom limb pain lead those individuals to experience phantom limb phenomena. There is another, more viable, hypothesis regarding the relationship between psychological factors and pain. This is that day-to-day variations in situational stress have an impact on phantom limb pain. This hypothesis is more likely because we have shown that in the headache literature there is a relationship, albeit weak, between changes in affect and changes in head pain (Arena, Blanchard, & Andrasik, 1984).

Our review of the literature (Sherman, Sherman, & Bruno, 1987) and of our interviews with our own patients showed that onset and intensity of phantom and stump pain are affected by stress, exhaustion, and other factors in much the same way other chronic pain syndromes are (Arena, Sherman, Bruno, & Smith, 1990; Sherman & Sherman, 1985; Sherman,

Sherman, *et al.*, 1987; Riddoch, 1941; see Chapters 4 and 5 for a mechanism for stress/anxiety-induced pain).

We recently reported the results of the first investigation into the relationship between situational stress and phantom limb pain (Arena, Sherman, Bruno, & Smith, 1990). Twenty-seven male amputees were asked to record their pain and overall stress levels for 180 days using a 1–10 rating scale. Four possible relationships into the etiology and or maintenance of phantom limb pain were examined using cross-logged correlational techniques: (1) there is no relationship between the two factors, (2) an isomorphic relationship exists in which same-time changes in pain led to same-time changes in stress, (3) there is a precursor relationship, in which changes in stress precede changes in pain, and (4) there is a consequence relationship, in which changes in pain precede changes in stress. The data were analyzed both within the day (subjects recorded their pain and stress four times a day) and between days, and using both the Pearson product moment and Spearman rank order correlations. Essentially the same results were obtained, and, therefore, only the between-days and Pearson product moment results are presented here.

Results indicated that 20 individuals (74.1%) demonstrated some significant pain–stress relationship, with the majority (17; 63.0%) demonstrating a significant isomorphic relationship. Twelve amputees (44.4%) demonstrated a significant pain-precedes-stress relationship, whereas 10 subjects (37%) demonstrated a significant stress-precedes-pain relationship. Seven individuals (25.9%) displayed no stress–pain relationship. In no instance was there a significant isomorphic or precursor relationship in the negative direction, and in only one instance was there a significant negative consequence relationship.

These results are meaningful not only because they clearly demonstrate that psychological factors can affect chronic phantom limb pain but because phantom limb pain is increasingly being treated through behavioral and psychophysiological interventions (Sherman, 1980; Sherman, Arena *et al.*, 1990). Such interventions are based on two hypotheses. The first is a predominantly psychophysiological theory that states that there are either (1) blood flow changes as a function of the amputation, or (2) there are increased muscle tension levels or spasms in the residual limb. The alternative hypothesis is predominantly psychological and postulates that there is a relationship between either (1) personality traits or (2) situational stress in amputees with phantom limb pain. As we have discussed above, there is empirical support for the first hypothesis but not the second. These data, however, which demonstrate a relationship between situational stress and phantom limb pain, provide a theoretical rationale for the use of behavioral and psychophysiological interventions such as biofeedback and relaxation therapy in amputees with phantom limb pain.

Personality Characteristics of Amputees Reporting Phantom Limb Pain: Unintentional Selection Bias of the Patients Sent to Mental Health Professionals

It is difficult to use the behavioral literature as a guide for making a valid characterization of the usual personality characteristics and problems of amputees because much of it is based on work done with subjects referred to mental health professionals rather than on random samples of amputees. Most workers, including Sherman *et al.* (1979), who have experience working with amputees coming to a health care provider solely to request treatment for phantom pain report that these patients tend to be very rigid and frequently appear to be compulsively self-reliant (Parkes, 1973). The surveys discussed above did not evaluate these traits. However, because the respondents requesting treatment were very similar to nonrequesters in other characteristics, it is likely that other factors may lead to selection for personality style among patients requesting treatment. Two critical factors to consider are that (1) it is common knowledge in the amputee community that most treatments for chronic phantom pain do not work at all (Sherman & Tippens, 1982) and (2) that a number of studies using postsurgical pain patients have shown that compliant people are less likely to report failure of treatments or to ask for further treatment than rigid, self-reliant people. For example, Lasagna, Mosteller, Van Fiesinger, and Beecher (1954) found that 50% of surgical pain patients reported decreased pain when given a medication placebo. Of the half who did not report decreased pain, only half of these (one quarter of the study group) reported a decrease in pain when given morphine as opposed to virtually all of those reporting reduced pain from the placebo. The nonresponders were significantly more rigid and controlled than the much more dependent responders were.

There is no more objective way to measure success of a pain treatment other than through the patient's report. Thus, the results of the Lasagna *et al.* (1954) study and the multitude of similar pain placebo response studies are actually a measure of the patient's willingness to tell his physician that a relatively minor part of his overall surgical care program was not successful. Because non-dependent people are less likely to be concerned about keeping the doctor's good will or about offending or "bothering" the doctor by saying that a minor part of the treatment does not work, they are more likely to give the physician an accurate report of treatment success. They are also more likely to resist the fear of being thought insane by their physicians. The above studies indicate they are less shy about letting a physician know when a treatment did not work. Therefore, they might also be less shy about endangering the relationship with their physician by reporting the existence of phantom pain. This would lead to self-selection

of patients toward those who would appear to be rigid and self-reliant. It should be noted, however, that among an unselected sample of amputees who were not seeking treatment, those with phantom limb pain could not be distinguished from their pain-free peers by their 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 (Katz & Melzack, 1990).

Studies of patients with chronic jaw pain (Sherman, 1985a) and chronic low back pain (Sherman, 1985b) produced subjective and some objective evidence that physicians based their referrals to psychiatry more on a feeling that the patient was disturbed than on a need for treatment of chronic pain. Interviews by Sherman, Sherman, *et al.* (1987) with orthopedists and psychiatrists referring dozens of amputees for psychophysiological evaluation support this hypothesis. They reported that their small sample of MMPIs lent support to this idea, as 60% of patients referred by psychiatrists showed psychotic elevations, 80% were clinically depressed, and all showed elevations on the psychosomatic scales. By comparison, of patients referred by orthopedists, 40% were within normal limits, 60% showed elevations on the psychosomatic scales, and none showed evidence of psychotic problems.

The clinical consensus seems to be that health care providers tend to refer to mental health professionals those pain patients with whom they do not get along or who do not meet the provider's needs more frequently than they refer similar patients who do meet their needs and expectations. They also seem to refer more of those pain patients lacking objective evidence of their complaints and treatment failure (Hackett, 1978; Usdin & Lewis, 1979). The typically rigid, persistent amputee who requests treatment for phantom pain would fit this profile perfectly because the treatments are not likely to work and the patient is likely to tell this to the health care provider.

This unrecognized bias in selection has led to the inappropriate categorization of the general population of amputees with phantom pain. When this bias is combined with the equally unsubstantiated assumption that phantom pain is largely a psychological problem, the result is that a high percentage of people reporting phantom pain are referred to mental health professionals for help. This does not mean that many amputees never have psychological problems. The relatively normal rate of occurrence of psychological problems in this population is discussed below. For example, when random or consecutive samples of amputees from surgical or rehabilitation clinics are evaluated, 20–60% are accessed as being clinically depressed (Riddoch, 1941; Shukla *et al.*, 1982a, 1982b) and/or having psychosomatic problems (Morgenstern, 1964).

Confounding Acute Adjustment Reactions to Amputation with Basic Personality Structure

The selection bias discussed above could lead mental health professionals to overestimate the degree of psychopathology among amputees with phantom pain. Many of the nonamputee chronic pain patients seen by psychiatrists have personality problems that cause reported pain intensity to be disproportionate to the degree of physical illness (Hendler, 1982). However, the expectation that chronic pain patients are likely to have personality problems can be confused with underlying causes of the chronic pain. Parks (1975) has written about relationships between grieving for a lost limb and for a deceased spouse. He hypothesized that a disordered grief process could lead to the grief being expressed as pain in the phantom—a psychic anguish turned to pain in the missing limb.

Sherman, Sherman, *et al.* (1987) reported that they had not encountered any patients with this problem but that they had treated many amputees who had difficult adjustment problems and others who were psychotic prior to their amputations. They seriously questioned the suggestion that amputations frequently cause psychotic reactions, although it leads people who are already unbalanced to have such reactions. Acute anxiety reactions following traumatic amputations should magnify phantom pain sensations. Sherman, Sherman *et al.* (1987) reported having worked with over a dozen people within a day of their having amputations for chronic vascular disease. These people were not warned prior to amputation that they would experience phantom sensations after amputation and were not aware of them from other sources. All had considerable anxiety and reported severe phantom pain when questioned. Several had acute anxiety reactions and believed that they had gone insane because they experienced a painful phantom limb despite the fact that the real limb had been amputated. When told that the sensations were normal and real, the reactions dropped off to a reasonable level. The phantom pain was reported to decrease to a manageable level as well.

The incidence of severe emotional problems should be about the same among amputees as among the general population. People with major psychological problems are likely to have more difficulty adjusting and may show the same kinds of hysterical reactions and pains to amputation as they would for other operations. These people may require help from a mental health professional. The same logic holds that psychological intervention is likely to be required to help a small minority of patients over acute reactions. Treatment of chronic problems is not likely to be directly helpful unless a chronic anxiety state is already part of the patient's personality and plays a role in magnifying the phantom pain just as it would any other problem. The respondents to the survey discussed above

reported that psychotherapy, major antidepressants, and other psychoactive drugs were frequently tried unsuccessfully for alleviating their pain. Thus, it is likely that the few patients who were chronically unstable may be helped by psychological interventions. However, the vast majority of psychologically normal amputees will not benefit from these interventions when they are delivered in an attempt to treat chronic phantom pain.

The Chronic Phantom Pain Patient as a Depressed Somatizer

Many studies have indicated that between 20% and 40% of amputees evaluated appear to be depressed (Morgenstern, 1970; Randall *et al.*, 1945; Shukla *et al.*, 1982a, 1982b). Depression can reach psychotic levels among some amputees (Shukla *et al.*, 1982a, 1982b). However, both Siller and Silverman (1958), as quoted by Friedman (1978), and Melzack (1971) felt that amputees were no different from nonamputees. Lindesay (1985) found amputees being treated for pain to be more depressed than other amputees. These findings open the door to the possibility that at least some instances of phantom pain are expressions of underlying depression or that the expression of phantom pain is more influenced by depression than would be expected by the usual magnification of painful sensations by depression. Turk and Salovey (1984) performed a detailed review of the evidence supporting the theory that chronic pain is a physical manifestation of an underlying depressive disorder. They largely debunked the theory for chronic pain in general. The following evidence supports this assertion.

The most frequently used and best validated objective psychological test for evaluating depression, somatization, and other personality variables is the Minnesota Multiphasic Personality Inventory (MMPI). Neither this test nor any similar ones are used as the sole basis for a diagnosis of depression. However, the MMPI is frequently presented in the literature as the way the diagnosis was substantiated or as the way a large group of people were screened. Many of the publications reporting the presence of psychosomatic or hypochondriacal characteristics among chronic pain patients, including amputees, based their assessments on the presence of a moderate "conversion V" configuration in the hysteria, hypochondriasis, and depression scales of the MMPI.

Moderate levels of this configuration have now been clearly demonstrated to be an artifact of the content of the questions contained in the scales and have minimal relationship to any psychological factors (Sherman, Camfield, & Arena, 1995). Many of the questions concern physical sensations that are experienced by virtually all patients with chronic pain.

It is these questions that produce the moderate conversion V configuration. The scales rise above the moderate elevations usually reported for chronic pain patients when psychologically oriented questions are endorsed. The MMPI consists of 566 true–false questions that were chosen on the basis of their ability to distinguish people with various types of mental disorders from psychologically normal people. It was never intended to differentiate psychologically normal chronic pain patients from ones with psychological problems that might lead them to report their symptoms, including pain, as being more intense than would be expected from their physical condition. Basic structural problems inherent in using the standard MMPI scales with chronic pain patients were discussed by Naliboff, Cohen, and Yellin (1982).

Chronic pain patients frequently answer many of the questions on the "psychosomatic indicator" scales very differently than do pain-free people but answer them similarly to the way known somatizers do because of the nature of the questions. These scales include such questions as "not being able to sit still," "being able to work as well as ever," "being in as good health as friends," "never felt better," "muscle weakness," "numbness in areas of the body," and "disturbed sleep." Smythe (1984) published an editorial about this problem in 1984. Merskey (1985) clarified the problem further with regard to headache patients in 1985. We have been collecting data since 1983 to standardize the MMPI for use with psychologically normal chronic low back pain patients and can confirm the clear existence of this problem with the MMPI in that population (Sherman *et al.*, 1995).

Many studies have shown that chronic pain changes people's personalities and their awareness of other bodily problems (Sternbach, 1968). These people are more conscious of their bodies than are most people (Weisenberg, 1975) and would therefore be expected to score higher than "normals" on scales concentrating on this element.

Melzack (1971) reported that amputees are no more neurotic than normals, and Shukla *et al.* (1982a, 1982b) reported that 1% of their amputee group showed psychotic symptoms within days of amputation. Only Lindesay (1985) divided his population into those who requested treatment for phantom pain and those who did not. As might be expected, those requesting treatment for phantom pain were significantly more depressed. Sherman, Sherman *et al.* (1987) gave MMPIs to 21 phantom pain patients. Ten of them showed clinical levels of depression, 15 showed psychosomatic patterns, four showed elevated psychotic scales, and four were within normal limits. Eight patients showed elevations on both psychosomatic and depression scales. All four patients showing elevations on the psychotic scales were among the eight with elevations on both psychosomatic and depressive scales. Sherman, Sherman *et al.* (1987) reported interviewing

dozens of amputees and having had contact with over 10,000 others through surveys and 6-month- and 1-year-long home logs. They reported that the overall consensus from these contacts was that amputees have no more, but certainly no less, severe psychiatric problems than the general population. Arena and Sherman (in press) recently analyzed the MMPIs of 24 consecutive amputees requesting treatment for phantom limb pain and compared them with age- and sex-matched subjects without a history of chronic pain. They found that the amputees were similar to the pain-free controls and within accepted limits of "normal" for all scales other than moderate elevations of the HS and HY scales, which produced the expected low conversion "V" as discussed above.

Sherman and Arena attempted to evaluate psychosomatic aspects of phantom pain through several questions on their surveys. They (Sherman & Sherman, 1983; 1985; Sherman *et al.*, 1984) asked amputees about their use of medical facilities, physicians, and medications for headache and shoulder pain. There were no differences for these factors between those amputees who reported phantom pain and those who did not. There were also no differences between the groups regarding prior knowledge of amputees or pain prior to an amputation; neither they nor Dawson and Arnold (1981) found a correlation between intensity of phantom pain and pain prior to amputation. They analyzed their data for differences in intensity, characteristics, and occurrence of phantom limb pain for three contrasting conditions:

1. The war in which the respondents lost their limbs (e.g., popular WW II versus unpopular Vietnam).
2. Whether the amputations were caused by war as opposed to civilian-related problems.
3. Unexpected traumatic amputation (war and accident) versus expected chronic amputation, e.g., required as a result of disease (diabetes).

They anticipated a difference between those contrasting groups if phantom limb pain had a major psychosomatic component. However, there were no differences at all. They concluded that psychosomatic factors were important bases for chronic phantom pain in the average amputee.

Living with apparently untreatable chronic pain is very depressing for most of the amputee, low back pain, and jaw pain patients interviewed by Sherman, Sherman *et al.* (1987). Thus, it is not surprising that they appear depressed on tests such as the MMPI and when talking to interviewers about their problems. Many studies have shown that there is a high correlation between depth of depression or intensity of anxiety and magnification of pain intensity (Melzack & Wall, 1982; Usdin & Lewis, 1979;

Weisenberg, 1975). Thus, any amputee who becomes depressed or anxious would be expected to report an increase in phantom limb pain. Sherman had amputee veterans keep daily home logs of their phantom pain intensity, stress, anxiety, depression, and several other factors (Arena, Sherman, Bruno, & Smith, 1990). In one study, 27 male amputees kept daily logs for 6 months. Of these, 52% reported that increased in depression, psychological stress, and anxiety intensified their phantom pain. The daily logs corroborate this assessment. Forty-one percent showed a consistent positive correlation between the presence and intensity of the above factors and the intensity of phantom pain; 11% showed a moderately consistent relationship; and 22% showed no relationship although the symptoms were reported. A further 26% did not record any of the above as factors affecting intensity of phantom pain. Thus, about half of the participants probably are not affected enough by these factors for them to notice it. These preliminary data provide objective support for Riddoch's (1941) and Kolb's (1954) assertions that anxiety, fear, and other stresses precipitate episodes of phantom limb pain. One of our most successful methods for reducing the intensity of phantom pain has emphasized helping patients reduce the anxiety related to the experience of phantom limb pain (Sherman *et al.*, 1979).

Rigid Personalities, Locus of Control, and Chronic Phantom Pain

If there are significant relationships between the occurrence of phantom pain and personality characteristics, it is plausible that amputees who believe they can tell what alters or controls their phantom pain might have more powerful needs to feel control over what happens to them in general. In other words, they might not actually know what caused the pain, but they might have such a great need to feel they have control that they confabulate relationships between the pain and various factors. It is also plausible that compulsively self-reliant, rigid people might have such a need for control that they would be unwilling to give up control of their sensations or of their body parts, even after removal.

The need to feel control over what happens is essentially the same concept as internal–external locus of control. A scale measuring this variable was developed by Rotter (1996) and has been used and validated in hundreds of studies. The form is short (40 questions) and does not ask the types of personal psychological questions that tend to induce recipients to discard surveys. The questions are about attitudes toward life in general rather than about health matters. Sherman and Arena (1992) reported that they used the scale because they felt that people would be less sensitive

about answering such questions and so would be more likely to answer a mail response survey. They also felt that the information obtained would be of more value than a health-related questionnaire because they were interested in evaluating a general personality characteristic rather than an attitude toward health.

They mailed surveys to 1200 of the 1321 members of the National Amputation Foundation. Of the 1200 letters sent out, 33 were returned as not being deliverable or appropriate, and 477 usable responses were received. The response rate of 41% is far lower than the 61% returned by the same group during the previous survey of this population by Sherman *et al.* (1984) and limits the usefulness of the data. Because of the previous survey, they were familiar with the group's characteristics and so had expected a very low response rate because of the predictable reaction to being asked "psychological" types of questions. Daniel's formula for estimating minimum sample size (Daniel, 1983) shows that a 40% response rate is sufficient to produce confidence limits of less than 0.95 for both the organization and the general population of veteran amputees if the responses were truly randomly drawn from the population. There is probably an unknown selection bias in responding that requires a very conservative interpretation of the results.

There was no significant relationship between locus of control and report of phantom limb pain. Nor was there any difference between the scores of those respondents who reported phantom pain and those who did not. There was also no significant relationship between locus of control and either usual intensity or frequency and duration of episodes of phantom pain.

We felt that certain factors might be more obviously connected with changes in particular descriptive types of phantom limb pain than with others. However, there was no visual or statistical relationship between description of phantom pain (e.g., burning, stabbing, cramping) and awareness that factors affected phantom pain ($\chi^2 = 16.26$ with 18 df, $p = 0.575$). Multiple regression analysis indicates that the descriptive type of phantom pain was not related to locus-of-control score. Among those reporting phantom pain, there was no significant relationship between locus of control and whether or not the respondent was aware of factors that affected his phantom pain.

Multiple regression analysis showed that, with one exception, there was no relationship between locus of control score and any one factor or any combination of factors that were reported to affect phantom pain. There was a 0.02 probability that people reporting fatigue as a factor affecting their phantom pain would produce a high score on the locus-of-control scale. Because 75 factors were considered, it is not surprising that one factor would happen to correlate with locus-of-control score just by

chance. We chose 0.01 for the level of significance that had to be achieved before we considered it likely that groups probably really were different, specifically to avoid this problem. Visual inspection of frequency graphs did not show any pattern of relationship between locus-of-control score and factors affecting phantom pain, which tends to confirm the lack of a relationship suggested by the statistical tests.

When possible, those factors identified by respondents as affecting their phantom pain were sorted into two groups: those felt to be clearly either internal to the responder (such as fatigue) or those external to him (such as weather). This was frequently a somewhat arbitrary decision, but it was felt that if any trends differentiating between the groups emerged, the part played by each factor could be statistically evaluated to permit resorting as necessary. The internal factors were: rest/lie down, change position, fatigue, exercise, stand/walk too long, work, back pain, other pain, excretion, illness, anxiety, stress, relaxation, depression, frustration, thinking about pain, and sleep. The external factors were: humidity, temperature variables, change in weather, dry stump sock, insert stump into water of various temperatures, and squeeze stump. There was no relationship between locus-of-control score and factors identified as being either internal or external.

In order to be certain that the limited sample of respondents was similar to the larger group who responded to the previous survey of the same population and to the very large groups of amputees responding to other surveys by the same team, Sherman and Arena (1992) checked the relationships between several key variables. The significant relationship between report of phantom and stump pain remained at about the same level ($\chi^2 = 6.696$ with 2 df, $p = 0.035$) as did the lack of relationship between cause of amputation and report of phantom pain ($\chi^2 = 5.92$ with 5 df, $p = 0.31$). Twenty-six percent reported that their phantom pain changed when their stump pain did. As we found in previous studies, there was no relationship between report of phantom pain and whether or not the amputation resulted from military problems ($\chi^2 = 0.76$ with 1 df, $p = 0.38$). There was no relationship between intensity of phantom pain and frequency of episodes (worst phantom pain intensity $\chi^2 = 53.54$ with 60 df, $p = 0.71$; average intensity $\chi^2 = 76.38$ with 70 df, $p = 0.28$; least intensity $\chi^2 = 66.34$ with 54 df, $p = 0.12$).

Conclusion

Based on (1) the lack of a relationship between locus of control and phantom pain, (2) analysis of the literature, and (3) the results of our other work with amputees discussed above, we conclude that chronic phantom

pain is affected by stress, anxiety, and depression much as are other chronic pain syndromes. There is no convincing evidence that a greater proportion of amputees reporting chronic phantom pains are psychologically abnormal or have abnormal personalities than the proportion of psychologically abnormal people found in the general population after adjustment is made for the effects of intractable chronic pain patients, their clinicians, and test instruments. Thus, it is very unlikely that characterological personality characteristics play a critical role in causing phantom pain.

Chapter 8

History of Treatment Attempts

Richard A. Sherman

The literature on treatment of phantom pain is highly contradictory, as it is largely based on short-term studies of small groups of patients. The lack of a year-long follow-up as a part of routine clinical practice tends to prevent providers from realizing that treatments have not been effective. Numerous medical, psychiatric, and surgical treatments have been randomly applied to those amputees requesting treatment for phantom limb pain. An update of an analysis of the literature originally done by Sherman (1980) combined with a survey of practitioners treating phantom pain (Sherman *et al.*, 1980) showed that 68 unrelated treatments for phantom pain were in recent or current use. Both practitioners and the published literature universally reported them to be successful. They ranged from highly invasive interventions such as lobotomies through spinal surgery and reamputation to more innocuous treatments such as "phantom exercises" (in which a sufferer is told to reach into himself and control the movements of his phantom, or even just recognize its existence), injection of the stump with local anesthetics, and relaxation training. Almost all of the research consisted of clinical, single-group studies with follow-ups of less than 6 months.

Reviews by senior clinicians based on their extensive clinical experience have stated that none of the treatments in the authors' areas of expertise were successful (Sherman, Ernst, Barja, & Bruno, 1988). The survey of practitioners showed that all felt that they could successfully treat phantom pain using one or several of 50 treatments reported. Each "successful" treatment was included among the unsuccessful prior attempts by numerous other respondents. This wide variety of treatments is illustrated in Figure 1. The most promising of the recent treatments was transcutaneous electrical stimulation (TENS). Finsen *et al.* (1988) carried

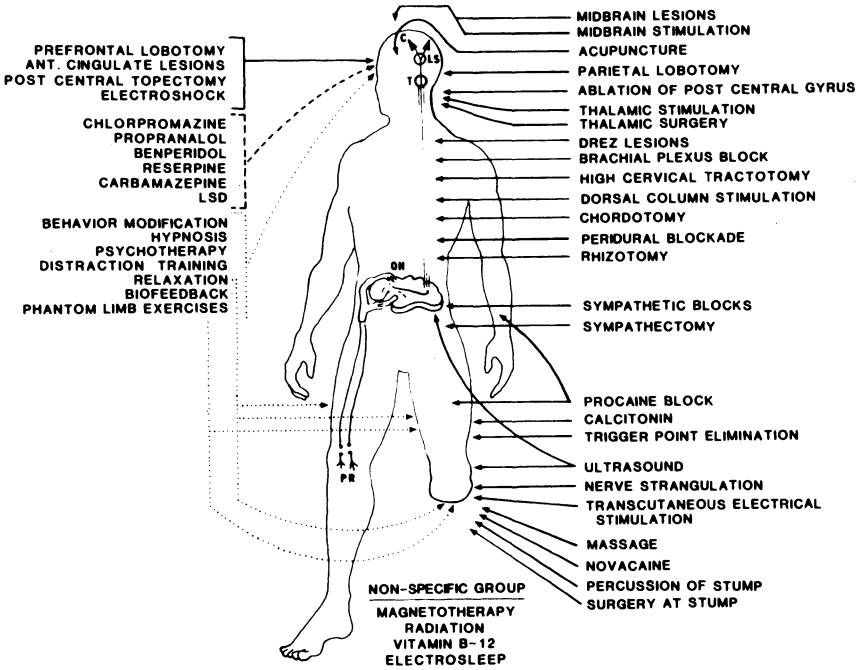

Figure 1. Treatments for phantom pain reported as being successful. Locations shown are only approximate. ———, physical Rx; - - -, chemical Rx; · · ·, psychological Rx; C, cortex; LS, limbic system; T, thalamus; DH, dorsal horn; PR, pain "receptor." From Sherman (1989b), reprinted with permission of W.B. Saunders Co.

out a controlled study that showed that TENS is frequently effective initially. However, there was no difference between placebo and treatment groups by the end of the first year. The study by Finsen *et al.* is one of very few placebo-controlled, long-term follow-up studies in the field. Its negative result was published only after years of positive reports from short-term, single-group clinical trials. These trials were interpreted by many clinicians as indicating that an effective treatment was available and that phantom pain was no longer a major treatment problem. This typifies the failure of the literature on management of phantom limb pain and emphasizes the need for a change in editorial policy so that short-term, small-group reports are not misinterpreted as having more than very limited clinical applicability.

Surveys of 10,000 randomly selected veteran amputees (Sherman *et al.*, 1984), all 1200 members of a national amputee veterans group (Sherman & Sherman, 1983), and over 500 self-selected amputees whose amputations were of civilian origin (Sherman & Sherman, 1985) showed that only 1% of

the respondents reported significant benefits from the host of treatments attempted (0.7% major permanent reduction in pain and 0.4% cure). The data from the original survey of veterans are summarized in Table 1. It must be emphasized that the data in this table have been confirmed subsequently, so the actual numbers of patients reporting failure of these treatments is far larger than indicated in the table. The above surveys of both clinicians and patients indicated that there was no relationship between the treatment applied and the symptoms of phantom pain reported.

The combined results of the studies by Sherman *et al.* show that the overall success rate for treatment was dismal. When treatments provided by the medical community are considered, only 1.1% of the respondents received lasting important benefits (0.7% large permanent change and 0.4% cure); 8.9% reported minor permanent improvements; 7.3% reported major temporary help from their treatments; 5.5% reported some very minor help; and 27.4% reported no change at all. Thus, at most, 8.4% of the respondents treated could be said to have been helped to any real extent. As it is probable that most patients do not list nonprescription pain medications and home remedies when filling out forms about treatments, it is likely that all respondents reporting pain had tried mild analgesics, heat, rubbing the stump, and other simple home remedies without positive effects or they would have mentioned them. The data reported are likely to err on the side of a lack of reporting of ineffective treatments rather than on leaving out effective ones.

Many popular treatments such as acupuncture are not yet widely available in U.S. military and Veterans Affairs hospitals, so Sherman respondents are less likely to have tried these novel treatments than the civilian amputees. Clinicians trying to use the information in this chapter will find themselves attempting to juxtapose the potential validity of the reports from a few veterans reporting failure of a rarely reported technique, such as hypnosis, with the generalizability of single case studies such as Siegel's (1979) report of short-term success (1 month follow-up) using hypnosis. They will also need to decide if the lack of recent reports of success of a technique mean that earlier reports of success have not been confirmed. For example, Nashold, Ostdahl, Bullitt, Friedman, and Brophy (1983) reported success with five patients treated with DREZ lesions, and this is quoted in the literature but has rarely been replicated. Other groups have been unable to replicate studies such as the Nielson, Adams, and Hosobuchi (1975) report of success using dorsal column stimulation with five of six amputees. Follow-up periods ranged from 7 to 25 months, so one might conclude that the technique had proved to be effective. However, numerous others are emphatic that the technique's effectiveness for phantom pain is lost after several years.

It is vital to note that there is no evidence that any surgical procedure

Table 1. Amputees' Reports of the Effectiveness of Treatments that Health Care Providers Have Reported to Be Effective

	Success (number of reports at each level of success)					
Type of treatment	No effect	Minor temporary	Minor permanent	Large temporary	Large permanent	Cure
Acupuncture	0	3	0	1	0	0
Alcohol (drinking)	28	86	0	3	0	0
Analgesics	195	421	116	52	2	2
Anterior cingual lesion	0	1	0	0	0	0
Anticonvulsants	3	4	0	0	0	0
Antidepressants	2	0	0	0	0	0
Biofeedback	0	1	0	0	0	0
Cordotomy	1	0	0	0	0	0
Electrical stim. of stump	6	7	1	2	1	0
Electroshock	2	3	0	0	0	0
Explanation/ reassurance	1	0	0	0	0	0
Increased use of prosthesis	0	5	0	2	0	2
Heat on stump end	26	54	0	5	0	0
Hypnosis	2	1	0	1	0	0
Injection (unspecified)	7	10	0	1	0	1
Local anesthetics	6	8	0	9	1	0
Massage of stump end	10	31	2	2	1	0
Narcotics (unspecified)	6	10	0	5	0	0
Nerve block (unspecified)	5	3	0	0	1	0
Nerve strangulation	2	2	0	2	0	1
Neurectomy	4	1	0	0	0	0
Novacaine blocks	9	6	0	0	0	0
Peripheral nerve stim.	2	1	0	0	0	0
Phantom limb exercises	2	0	0	0	0	0
Phenthiazine	1	0	0	0	0	0
Physical therapy	12	11	1	1	0	0
Pills (unspecified)	9	24	0	3	0	0
Press end of stump	1	2	0	0	0	0
Psychotherapy	10	0	0	1	0	0
Quinine	1	3	0	0	0	0

(*continued*)

Table 1. (Continued)

Type of treatment	Success (number of reports at each level of success)					
	No effect	Minor temporary	Minor permanent	Large temporary	Large permanent	Cure
Raise stump	0	1	0	1	0	0
Refit prosthesis	0	0	1	0	0	0
Relaxation training	3	0	0	1	0	0
Sedative hypnotics	13	56	0	6	0	0
Stump desensitization	2	3	1	2	1	0
Stump revision (surgical)	13	11	1	2	0	0
Sympathetic block	1	1	0	0	0	0
Sympathectomy	2	3	0	0	0	0
Thalamic stimulation	0	1	0	0	0	0
Ultrasound at stump	2	7	0	3	2	0

performed on the residual limb or spinal cord has any lasting positive effect when done solely for relief of phantom pain. There is also no evidence that psychotherapy or major tranquilizers will cure the pain (Melzack & Wall, 1982). Thus, most widely used treatments do not work, and heroic efforts are doomed to failure. It is also important to note that these treatments have not stopped being used. They continue to resurface as practitioners are confronted with amputees requesting treatment for phantom limb pain. Some practitioners attempt to use treatments that normally form part of their treatment plan without checking the literature to ascertain that what sounds like a potential solution has, in fact, been tried and failed. This would not be so critical if such treatments were in the realm of the harmless but ineffective or briefly effective "phantom exercises" that keep being rediscovered and, thus, keep reappearing in newspaper stories (e.g., *New York Times*, 1995). Unfortunately, many of the highly invasive treatments causing permanent damage and disability continue to be used even though they are known to have no lasting effect.

Chapter 9

Mechanism-Based Assessment and Management

Richard A. Sherman, D. E. Casey Jones, and Joseph J. Marbach

Phantom Limb Pain

Development

The first hints that there might be a rational clinical approach to relieving phantom limb pain came from attempts at relating treatment effectiveness to descriptions of the pain. Surveys and clinical experiences have demonstrated that virtually all amputees give consistent descriptions of their phantom pain. The most common descriptive groups are burning (including tingling, pins and needles, etc.), cramping (including tightness, squeezing, etc.), and shocking–shooting. Careful examination of survey responses and review articles showed that sympathectomies could be moderately successful in reducing burning phantom pain, but not other descriptors, for up to a year (Sherman, 1984). Interventions causing reduced muscle tension in the residual limb resulted in lower levels of cramping/squeezing descriptors of phantom pain but not of others (Sherman, 1976). These early findings led to elucidation of physiological correlates of phantom pain and, eventually, to several effective management techniques based on them (Sherman, 1989a, 1989b).

Prevention

Prevention of Initial Occurrence of Phantom Pain

Reports of attempts to alter surgical procedures so as to reduce the initial occurrence of phantom pain include tying off the major nerve trunks in a variety of ways, avoiding placing the residual limb in abnormal or stressful positions during amputation, and placing the residual limb in a rigid dressing as soon as is practical. We have not been able to find objective evidence that these techniques are particularly effective. The practice of preempting phantom limb pain by administration of epidural agents prior to amputation has met with some success (see Chapter 5). Bach *et al.* (1988) carried out the first controlled study in which lumbar epidural morphine and bupivacaine was administered prior to amputations in 11 of 25 elderly patients. One year later, three of the controls had phantom pain while none of the treated patients did. More recently, these findings have been confirmed by Jahangiri *et al.* (1994) using a similar design. These studies suggest that the practice of blocking noxious inputs from reaching the spinal cord before, during, and after amputation may prevent the development of long-term phantom limb pain. Elizaga, Smith, Sharar, Edwards, and Hanson (1994) followed nine amputees treated during the amputation process and 12 amputees who were not pretreated for 6 months and found no differences in occurrence of phantom pain (77% versus 50%, respectively). Thus, until more studies are conducted, it is uncertain whether this technique will be shown to be effective on follow-up. We urge readers to keep up with new studies and to begin using this technique if the mass of evidence points in the direction of effectiveness.

Prevention of Magnifiction of Normal Phantom Sensations in the Acute Postoperative Period

Virtually all amputees experience phantom sensations just after an amputation. As discussed above, this can be a real shock to patients who have not been prepared. When the anxiety engendered by these sensations is combined with the other stresses inherent in the operative and rehabilitative processes, sensations can be magnified tremendously. Stress and the attendant magnification of pain can be significantly reduced by properly educating patients about phantom sensations and the surgical and rehabilitative processes before or as soon after amputation as possible. Whenever possible, every patient about to have an amputation should be educated as throughly as possible about the upcoming process (hospitalization, surgery, recovery, etc.) and rehabilitation. This reduces the enormous stress associated with the situation and permits the patient to use for the recovery

process the great amount of energy that would otherwise be expended on responding to stress.

Gerhards *et al.* (1984) found a high correlation between poor rehabilitation outcome and lack of understanding of the amputation and rehabilitation processes. We strongly recommend that every patient about to have an amputation be provided with a copy of the *Amputee Guide* which forms Appendix II to this book. When preamputation education is impossible, it should be provided as soon after the amputation as practicable.

Amputees are no more or less psychologically healthy than any group of individuals (Sherman, Sherman, *et al.*, 1987). A proportionate number of people with preexisting psychological problems also have amputations. The stress of amputation and the phantom pain that follows can exacerbate these problems and produce acute psychological and emotional distress (including hysterical reactions). These acute reactions to the stresses of surgery and pain should be treated by the appropriate professionals as quickly as possible.

Prevention of the Pain from Becoming Chronic

Pain is exceedingly difficult to treat effectively once it has persisted for more than a year (Melzack & Wall, 1982; see Chapter 3). On this basis, every effort must be made to prevent phantom and stump pain from becoming chronic. In order to minimize stress, our approach has been to educate preamputees and recent amputees about the entire process—from surgery to rehabilitation—they are going through in order to minimize stress. We ensure that they understand that phantom sensations are normal and illustrate the mechanisms of referred sensations so that they can picture how it is possible to feel sensations from a body part that has been surgically removed. When appropriate, we use examples of referred pain from everyday life such as "the pain that runs down the forearm when the elbow is struck."

Changes in patterns of blood flow in the residual limbs of amputees reporting chronic burning phantom pain look exactly like the changes we have recorded among subjects with chronic reflex sympathetic dystrophy (Sherman & Arena, 1992; Karstetter & Sherman, 1991). The problems may have similar underlying mechanisms. Reflex sympathetic dystrophy is nearly impossible to ameliorate significantly, let alone cure, once it has become chronic.

Treatment of Phantom Limb Pain Immediately after Amputation

The authors have had mixed experiences with the effectiveness of medications for control of postsurgical phantom pain. Sherman and Jones

have found that traditional pain medications usually do not affect phantom pain significantly. Katz has found that morphine delivered by a patient-controlled pump (PCA) is effective in controlling postsurgery phantom and stump pain. Epidural analgesia and anesthesia also may be effective and render patients almost pain-free after amputation (Bach *et al.*, 1988; Jahangiris *et al.*, 1994). The optimal treatment for acute postamputation phantom pain appears to be a combination of attempts at control with medication and patience, stress control, and relaxation training. These latter treatments are normally provided by the psychologists on the health care team. As the residual limb begins to recover, the phantom pain almost always decreases to very manageable intensities. However, it is misleading to tell patients that their phantom pain is likely to go away entirely because at least 80% of amputees have functionally significant episodes of phantom or stump pain every year, and many have almost constant, very low-level stump and phantom pain, which they define as being just over the threshold of nonpainful sensations. These occurrences were discussed in Chapter 1.

Assessment of Subchronic and Chronic Phantom Limb Pain

Good treatment usually begins with good assessment. Phantom pain is no exception to this dictum.

Search for Sources of Referred Pain

We have found that a quick evaluation is of limited value. In order to optimize the chances that the initial treatment will work, we need to (1) listen to the patient's description of the pain and the factors that affect it and (2) get a home log of pain and related factors. The 1-week home log is needed to permit changes in phantom pain to be correlated with changes in diet items (e.g., onions), excretion, changes in weather, physical or mental stress, and use of the prosthesis. Patients frequently miss relationships because time lags may be hours to days.

Phantom pain is almost always exacerbated by episodes of stump pain. Evaluate and treat the stump pain as appropriate before proceeding very much further with treatments aimed only at phantom pain. If the log indicates that phantom pain is related to use of the prosthesis, especially to the duration of its use, carefully evaluate its fit and effect on gait. Dr. Daniel Shapiro is a physiatrist whose specialty is amputee care. He (personal communication, 1995) states that

> An ill-fitting prosthetic device is one of the most easily treatable causes of phantom pain, yet inexplicably, it is often overlooked. Pain, numbness, or

paresthesia related to prosthetic use is a red flag. The symptoms are usually not as unpleasant as other phantoms and are in a dermatomal distribution. The clinician should obtain a history of phantom phenomena related to sitting, standing, or ambulating in the prosthesis or starting shortly after the prosthesis is removed. The dermatomal distribution suggests a local nerve root compression. Palpation of the suspected nerve reproduces the symptoms. Percussion sign is usually positive (Christopher & Koepke, 1963). Palpation of the nerve with the prosthesis donned provides further confirmation. Prosthetic pressure and shearing usually cause redness, hyperpigmentation, blistering, abscess, ulceration, or callosities over the involved area. Xeroradiography will further demonstrate abnormal compression by the prosthesis or migration of the distal fibula–tibia articulation. Prosthetic adjustment is curative. Compression of the common peroneal nerve during ambulation can be corrected by fusion of the fibula–tibia at the distal end to prevent migration. In the unilateral upper extremity amputee, the prosthetic harness may cause phantom pain on the amputated side or referred pain on the contralateral side. In the bilateral upper extremity amputee, in addition, the prosthetic harness may cause phantom pain in the contralateral side by compressing the brachial plexus. Compression by the prosthetic socket is analogous to that of the lower extremity prosthesis. Table 1 provides a summary of relationships between prosthetic defects and which nerves are entrapped. Part of the table is based on work by Christopher and Koepke (1963).

Normal referral patterns are still operative even if the limb to which pain is referred has been amputated, so look for referral of pain into the phantom from the back, bladder, etc. When relationships between activity or stump swelling and phantom pain are evident, a radical change in the way the stump is wrapped may be helpful. Interestingly, one of the only double-blind, crossover studies done in the treatment of phantom pain was performed by Conin, Hershler, Alexander, and Crisp (1993) with 34 ampu-

Table 1. Prosthetic Problems Causing Phantom Pain

Prosthetic defect	Type	Nerve entrapped
Posterior brim	AK	Sciatic
Pressure fibula	BK	Common peroneal
Popliteal bulge	BK	Posterior tibial
Too many socks	BK	Posterior tibial
High lateral wall	AK	Lateral femoral cutaneous
High medial wall	AK	Ilioinguinal
High medial wall	CATCAM	Ilioinguinal
Tight medial	Canadian	Ilioinguinal
Tight socket	BE	Ulnar or median
Tight suspension	BE	Radial
Tight socket	AE	Brachial plexus
Tight harness	AE	Contralateral plexus

tees who wore a special metal stranded fabric when episodes of phantom pain began. Twenty-one of their subjects reported the greatest decrease in pain when wearing the experimental garment as opposed to the placebo. Only two showed nearly complete pain relief, and average differences in pain intensity between the groups were very small, so the clinical importance of the device is still open to question.

Environmental factors may also be important in initiating and intensifying episodes of phantom pain. If changes in weather are related to changes in phantom pain, a trial of antiinflamatory drugs and/or other medications, which one might use when attempting to ameliorate the effects of changes in the weather on pain from to arthritis, are appropriate. If phantom pain changes with physical stress, have the patient reduce physical activity levels, check the prosthesis, and check for changes in blood flow in the residual limb with activity.

Be Aware of Psychological Factors that May Exacerbate the Pain

Ensure that the subject understands (1) that phantom pain is not likely to be of psychological origin and (2) how it is possible to continue to feel body parts that have been amputated. A psychological screen (including the MMPI) is not always necessary but is appropriate for identifying masked depression, situational anxiety, and hysterical reaction to loss of a limb. Both anxiety and depression magnify pain. If the log shows a change in phantom pain with changes in stress and anxiety, treat these latter symptoms appropriately (reduction through increased understanding, relaxation training, etc.). Major psychological disorders can exacerbate the pain. If the patient is isolated from other amputees, referral to a local amputee support group can be helpful. If the patient has an overwhelming need for pain (pain games within the family, somatization of problems, etc.), treat them appropriately before attempting other interventions aimed solely at relieving phantom pain.

Recommended Procedure for Evaluating and Treating Patients with Chronic Phantom Pain[1]

Rationale and Background

As early as 1979, it became apparent that different descriptions of phantom pain responded to different treatments (Sherman *et al.*, 1979). Our initial attempts to treat phantom limb pain with a combination of biofeed-

[1]Modified from Sherman and Arena (1992).

back and relaxation techniques showed excellent success up to 6-month to 3-year follow-up with 14 of 16 successive phantom pain patients. The major difference between those patients who succeeded in learning to control their pain and those who did not was the ability to relax in any measurable way. Our two failures never (1) demonstrated the ability to relax nor (2) reported subjective feelings that would be associated with learning to relax or to control muscle tension.

For the last few years we have been attempting to align our behavioral and medical treatments of phantom pain with underlying physiological correlates. Thus, when amputees demonstrated that decreased blood flow in the stump was related to increased burning phantom limb pain, peripheral vasodilators and temperature biofeedback were used to decrease the phantom pain. When increased muscle tension and spasms in the stump were related to episodes of cramping phantom pain, muscle relaxants and muscle tension biofeedback were used to control the pain.

Among our most recent cases, EMG biofeedback was effective for 13 of 14 trials for cramping phantom pain. The EMG biofeedback had minimal success with two and no success with 10 of 12 trials for burning phantom pain. It had no success with eight trials of shocking phantom pain. Temperature biofeedback was ineffective for four trials of cramping phantom pain, was effective for six of seven trials with burning phantom pain, and had no success with three trials for shocking phantom pain. Nitroglycerin ointment (a topical vasodilator) was ineffective for one trial of cramping phantom pain and one of shocking phantom pain but successful for two trials of burning phantom pain. Trental (a blood viscosity enhancer) was ineffective for two trials of cramping phantom pain and one of shocking phantom pain. Nifedipine (a systemic vasodilator) was effective for three trials of burning phantom pain but ineffective for one trial of cramping and two trials of shocking phantom pain. Cyclobenzaprine (a muscle relaxant) was effective for two trials of cramping phantom pain but ineffective for one of shocking phantom pain. Indomethacin (an antiinflammatory agent) was ineffective for two trials of cramping phantom pain. These medications have potential side effects and can not be used with many patients having a variety of medical problems. Thus, we prefer using self-control-oriented strategies to avoid these limitations.

It is clear that burning phantom pain responds to interventions that increase blood flow to the residual limb whereas cramping phantom pain responds to interventions that decrease tension and spasms in major muscles of the residual limb. Shocking–shooting phantom pain does not respond well or consistently to either type of intervention. The physiological bases for these changes were documented in Chapter 6.

We strongly recommend that biofeedback of appropriate parameters

be used in conjunction with other self-control-training strategies to treat cramping/squeezing and burning/tingling phantom limb pain. It is important for clinicians to recognize that biofeedback as utilized for control of phantom limb pain is not some kind of black-box psychomagic. Rather, it is simply the process of recording the physiological parameters (such as muscle tension in the residual limb) that precede changes in phantom pain and showing the signals to patients. The patient uses the information to change the signal. The patient also learns to associate sensations related to onset of phantom pain with tension in the muscle, decreased blood flow, etc. and to use the learned ability to control the parameter to prevent the onset of pain or to stop it if it has already begun. The concept of biofeedback is illustrated in Figure 1.

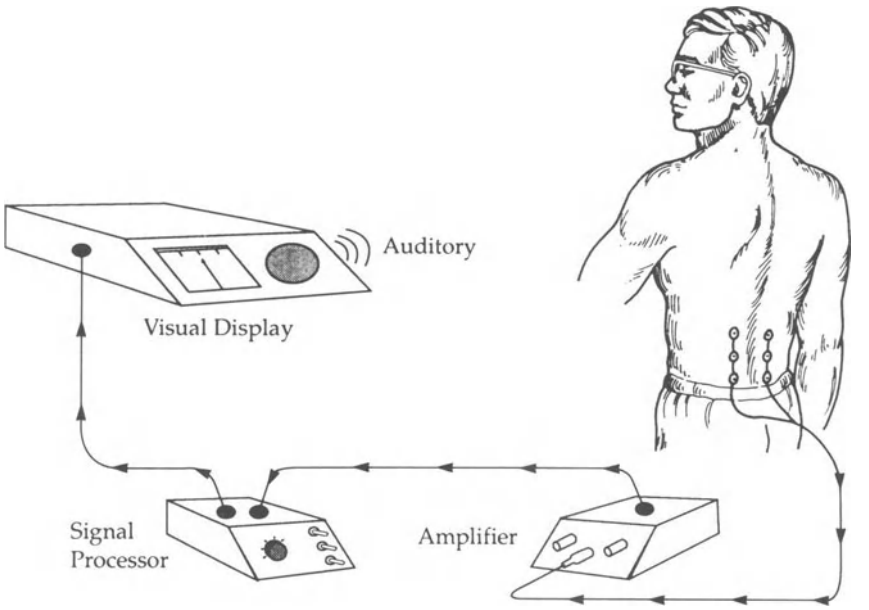

Figure 1. Concept of biofeedback. The subject is shown watching a display in which the position of the meter and the pitch of the tone are approximately proportional to the overall tension in the paraspinal muscles. The muscles' tensions are being recorded from surface electromyographic sensors taped over the bellies of the paraspinal muscles at L4, then amplified and processed for display. Most displays are on computer monitors, where the "raw" signal and any versions processed from it can be readily viewed. The patient and therapist use the information to help the patient relate sensations in the body to actual muscle tension levels on a moment-to-moment basis as well as to learn to control the muscles appropriately.

Break with Tradition

As detailed above, it has been clearly demonstrated that many traditionally accepted treatments simply do not work for more than a few percent of amputees and need not be tried.

Burning Phantom Pain

If the patient reports burning phantom pain (including tingling and similar descriptions), increased phantom pain with decreased atmospheric temperature, or decreased stump temperature before increased phantom pain, first give a trial of temperature biofeedback from the residual limb in conjunction with relaxation training containing warming exercises. If this is not effective, try peripheral vasodilators (such as nitroglycerin paste applied to the distal end of the residual limb) and, if necessary, multiple sympathetic blocks (single blocks tend to be of short duration and ineffective as a treatment, but may be a useful diagnostic tool).

Cramping Phantom Pain

If the patient reports cramping phantom pain (including twisting, gripping, etc.), or the stump shows spikes in the EMG and/or spasms during phantom pain, give a trial of muscle tension biofeedback from the residual limb in conjunction with muscle tension awareness and control training. If this is not effective, give a long trial of muscle relaxants.

Other Descriptions of Phantom Pain

Luckily, cramping and burning descriptors and their close relatives are the most commonly encountered. The others, such as shocking, shooting, and twisting are relatively rare. Their mechanisms are not known, and no treatments have consistently been shown to provide significant relief for more than a few months for the vast majority of amputees. Thus, there is no way to predict which treatments, if any, will provide lasting benefits. The guideline below can be followed for the best chance of relieving the pain.

Bartusch, Sanders, D'Alessio, and Jernigan (in press) recently reported that two patients with chronic shocking–shooting phantom limb pain who were unresponsive to other treatment modalities responded very well to clonazepam and that the effects were maintained for at least 6 months. Sherman, Evans, Caminer, *et al.* (1993) reported one patient who successfully treated himself for shocking–shooting phantom pain by not permitting the limb to change temperature quickly (as would happen

when going outside on a cold winter day or getting out of a warm bath). Tsushima (1982) reported one patient who had at least 2 months of relief from shooting phantom pain after temperature biofeedback but not after muscle tension biofeedback. McKechnie (1975) reported one case of sharp phantom pain (which Sherman *et al.* have included with their reports of shocking–shooting) being responsive to progressive muscle relaxation training for at least 6 months. The training included relaxing the muscles of the residual limb and imagining relaxing the muscles of the phantom. A comment at the end of the article indicates that distraction training did not help this patient. Although not commented on by the author, readers should be aware that progressive muscle relaxation exercises have been shown to increase blood flow to the extremities. Thus, the observed changes could be linked to many factors ranging from changes in muscle tension and blood flow in the residual limb to exercising the phantom.

What to Do if Initial Attempts Are Unsuccessful or if the Mechanism for the Description Given Is Unknown

The following treatments have shown fair success with short-term follow-ups and have not been reported as useless in amputee surveys. They should be tried for cases of cramping and burning phantom pain that do not respond to the treatments recommended above as well as for other descriptions of phantom pain. A 6- to 8-week trial of relaxation training for pain control can be followed by TENS. Try many locations on the residual limb with many intensities and waveforms. It must be emphasized that a long-term, controlled study showed that TENS is ineffective on follow-up. However, other studies have indicated at least temporary relief, which could be used to disrupt the pain cycle long enough for healing to take place on its own or for other treatments to work. For example, Katz and Melzack (1991) performed a controlled study that showed that auricular TENS can make small but statistically significant reductions in phantom pain for a brief period. We are not aware of long-term follow-ups for any of these studies. If these trials are ineffective, active range-of-motion exercises along with phantom exercises sometimes help. Next, try ultrasound at the stump. If it is initially successful, add steroids. The last attempt would be a trial of sedative hypnotics followed by a mixture of amitriptyline and fluphenazine.

Reports of several treatments having sufficient information and follow-up to permit evaluation have appeared in the clinical literature in the years since the surveys on which these recommendations are based were conducted. Davis (1993a) found mexiletine to give good to excellent results in reducing chronic phantom pain in 18 of 31 patients (see Chapter 3

for the rationale of using Na^+ channel-blocking agents like mexiletine.) Eleven of the 31 showed a "favorable response" when Clondidine was added. Only two did not respond at all. For the combined group, 12 were defined as having excellent results, 16 had a good result, and 13 did not show a clinically significant change. Unfortunately, the descriptions of phantom pain are not included in the report. All of the patients were followed for 1 year and maintained their decreases in pain. As far as can be determined from the report, none were actually cured but, according to their visual-analog pain scales, most were significantly helped.

Jaeger and Maiger (1992) used calcitonin with 21 patients having acute, just postamputation phantom pain. They gave each patient the drug or placebo infusions and found that only the drug reduced reports of phantom pain. Pain was reduced from a median of 7 to 4 on a visual-analog scale regardless of whether placebo or the real drug was given first. One week after treatment, 19 of the 21 had more than 50% relief of pain. Of these, 16 were pain-free. After 1 year, 8 of the 13 surviving patients had more than 75% pain relief. There was a second year of follow-up but the results are difficult to understand: the authors state they were the same as after 1 year. The value of this treatment is difficult to determine because many amputees show a similar decrease in phantom pain intensity and frequency during the years following amputation as reported in this study.

Both of these studies meet the criteria for reasonable studies with reasonable follow-up periods. They need to be replicated with larger numbers of subjects and better descriptions of the phantom pain before they can be accepted into the recommended armamentarium for phantom pain, but they are certainly worth a try if the mechanism-based treatments do not work.

Gross (1982) reported cases in which local anesthesia to hyperalgesic points on the intact limb reduced or abolished phantom pain in four amputees. Patients were followed for between 6 months and 2 years with continuing relief. Monga and Jaksic (1981) report using acupuncture with one patient who had received a number of previously unsuccessful treatment attempts including the use of β blockers and tranquillizers. Hypnotherapy did help this patient for a half-hour or so after each session. Acupuncture was by both needle and electrical stimulation to the residual limb and reduced the pain sufficiently for the patient to use a prosthesis. No long-term follow-up is reported. This case report is included here because one of the authors of this book (Sherman) has received about a dozen phone reports of successful use of acupuncture for relief of phantom pain. None have follow-ups on more than one or two cases. Thus, there is no way to tell whether this is another currently popular treatment that will fall by the wayside after failing to stand the test of time or whether it really

works. The authors can only encourage further testing and long-term follow-up of the above and numerous other techniques being tried on a few patients. Of course, any treatment that relieves otherwise intractable pain for a given period may be worth using if only to provide temporary relief. It may be more useful if it is sufficiently free of side effects that it can be repeated as needed.

If the above treatments do not work, other interventions (such as major tranquilizers, revision of a normal stump, sympathectomy, and surgical invasion of the spinal cord and brain) are even less likely to produce lasting results. It is probably best to tell the patient that current treatments are not likely to succeed. The best alternative is referral to a pain clinic for distraction training, optimal adjustment to living with pain, etc.

Treatment of Phantom Tooth Pain

Differential Diagnosis

Phantom tooth pain should be differentiated from two better known categories of facial pain disorders. The first category includes the typical neuralgias, the most common of which is trigeminal neuralgia (TN) (Fromm, 1991). Its paroxysmal sharp sudden recurrent electrical stabbing pain in the distribution of one or more branches of the trigeminal nerve is unlike the dull constant pain of phantom tooth pain. The age of onset of trigenminal neuralgia usually follows the fourth decade with a peak onset in the fifth and sixth decades. Other typical neuralgias are associated with acute herpes zoster, postherpetic neuralgia, geniculate neuralgia, and, of course, toothache from pulpitis. In addition, musculoskeletal disorders such as the temporomandibular pain and dysfunction syndrome (TMPDS) (IASP, 1994), arthritis of the temporomandibular joints, and maxillary sinusitis are sometimes confused with phantom tooth pain.

The second diagnostic category, atypical facial pain or atypical facial neuralgia (AFP), is in common use clinically. Frequently, phantom tooth pain patients are relegated to the imprecise diagnosis of atypical facial pain, a term deliberately rejected for its vagueness by the International Association for the Study of Pain (IASP, 1994). Atypical facial pain is a diagnosis made, traditionally, after excluding other possibilities for which the clinician has a physical explanation. The etiology of atypical facial pain is frequently attributed to psychological factors (Harris, 1974; Rees & Harris, 1978). Atypical facial pain is traditionally associated with female gender, depression, anxiety, and hysteria, although evidence is lacking (Adams & Victor, 1989). This should come as no surprise. The symptoms of

phantom phenomena do not follow patterns associated with the biomedical model of disease (Sherman, Sherman, *et al.*, 1987). The symptoms are recalcitrant to treatment, and examination reveals high rates of psychiatric comorbidity particularly depression (Lascelles, 1966; Lesse, 1987; Feinmann, Harris, & Crawley, 1984; Ford, 1983). Examination of the atypical facial pain literature suggests that many atypical facial pain patients meet criteria for phantom tooth pain.

Approaches to Treatment of Phantom Tooth Pain

Treatment is based on two approaches. The first, using oral medications, attempts to influence afferent impulses that culminate in central synaptic excitability. The second, nerve block injections, are directed at the changes in the chemistry of transported substances in the PNS. Another focus involves professional education that targets prevention and early diagnosis. This approach limits unnecessary deafferentation and promotes early treatment. Treatments such as dental and neurosurgical procedures should be avoided, as they are associated with high morbidity.

Centrally Acting Agents

Drug therapy is probably the most widely used clinical treatment for neuropathic pain. A variety of mechanisms that include increased brain serotonin and enhanced GABA binding has been investigated to explain analgesic effects of oral medications in a variety of neuropathic pains (DeMarinis, Fraioli, Esposito, Gagliardi, & Argnoli, 1992). Unfortunately, case reports and uncontrolled drug trials provide limited information to date. When positive results are obtained, it is rarely clear whether the improvement represents a placebo effect, a spontaneous remission, an impact on the causative factor(s), or a psychopharmacological effect concurrent with or independent of the pathophysiology of the pain. Despite these problems, the widespread use of the large variety of oral drugs requires careful examination for clues to etiology and differential diagnosis.

Narcotic and Non-narcotic Analgesics. Opiates are only moderately effective in reducing neuropathic pain (Arner & Meyerson, 1988). This is also true for phantom tooth pain (Marbach, 1978a). However, some experimental evidence suggests the contrary (Lombard, Besse, & Besson, 1992). Most nonnarcotic analgesics and nonsteroidal antiinflammatory drugs (NSAID) do not affect phantom tooth pain. Narcotics, nevertheless, may have a role in the treatment of chronic phantom tooth pain. As described in

detail by Portenoy and Foley (1986), a subset of patients suffering from chronic benign pain may benefit from a fixed daily dose of oral narcotic analgesic such as oxycodone (Percodan), meperidine (Demerol), or morphine. Patients selected for this program acknowledge that the drug is addictive and are screened for drug abuse proneness and medical suitability.

In addition, intranasal application of cocaine has been shown temporarily to abolish phantom tooth pain (Marbach & Wallenstein, 1988). Like the chronic use of opiates, cocaine possesses complex management issues. Besides its long recognized local anesthetic effects, cocaine has been reported to show central analgesic effects. Cocaine, unlike opiates, apparently does not show affinity for specific receptor sites (Reith, Sershen, & Lajtha, 1985). A dramatic loss of opioid binding sites has been shown to occur after deafferentation, perhaps helping to explain why cocaine is more effective than opiates in the treatment of phantom tooth pain.

Anticonvulsants. Carbamazepine (Tegretol) and phenytoin (Dilantin) have been widely used and studied in the treatment of orofacial neuropathies. Their mode of action appears to be blockade of Na^+ channels (see Chapter 3). This limits the ability of neurons to fire repetitively, especially at high frequencies. In an adequate dose, carbamazepine is effective for about 80% of trigeminal neuralgia patients within 24 hr. If a moderate dose of carbamazepine (600–1200 mg daily) relieves pain in a putative phantom pain patient, one should consider that the patient suffers from trigeminal neuralgia. Carbamazepine and phenytoin do not produce analgesia for phantom tooth pain. Phenytoin influences both axonal conduction and neural impulse initiation. Its site of action may be at the abnormal peripheral nerve or dorsal root ganglion (see Chapter 3), or within the central nervous system. Baclofen (Lioresal) is effective occasionally in phantom tooth pain as an adjunct to other medications.

With one exception, barbiturates, nonbarbituate hypnotics, and minor tranquilizers have little therapeutic efficacy in phantom tooth pain. Clonazepam (Klonopin), a benzodiazepine derivative, in doses of 1–3 mg daily may produce pain reduction of phantom tooth pain.

Tricyclics and Tranquilizers. Tricyclic iminodibenzyl derivatives have been used for many years, and with increasing frequency, in the treatment of many deafferentation pain syndromes (Maciewicz, Bouckom, & Martin, 1985). The precise site of action of these drugs is unknown. However, the pharmacological properties may be related to inhibition of synaptic serotonin and norepinephrine reuptake. Questions have been raised whether tricyclics act as analgesics or work by altering mood. Recent evidence supports an analgesic effect (Davar & Maciewicz, 1989).

The pharmacological properties of tricyclics may shed light, additionally, on the complex relationship of pain to depressed moods. For example, tricyclics act as analgesics in lower doses (amitriptyline, 10–40 mg) and with greater rapidity (1–3 days) than is the case for mood changes, which frequently require higher doses (amitriptyline, 75–150 mg) for longer periods (3–6 weeks). Phenothiazines potentiate the analgesic effects of tricyclics in phantom tooth pain. In severe cases, the combination of tricyclics and phenothiazines (i.e., perphenazine and amitriptyline, Triavil) can be effective (Marbach, Varoscak, & Cloidt, 1986). Monoamine oxidase inhibitors have been recommended for tricyclic nonresponders (Schnurr & Brooke, 1992; Lesse, 1987). The newer generation of antidepressant drugs have not displayed analgesic properties for the treatment of phantom tooth pain.

Peripherally Acting Agents

Local anesthetic injections are used routinely for the temporary relief of pain and to facilitate operative procedures. They are also effective in reducing and in certain cases eliminating phantom tooth pain alone or when combined with steroids. Injection of nerves using dexamethasone in combination with local anesthetics is clinically effective. In rats, local steroid injection to the site of nerve compression facilitated recovery of nerve conduction blockade when compared with saline-injected and non-injected control groups (Hono, Chena, Liu, & Yu, 1990). Corticosteroids silence ectopic impulse firing originating at nerve injury sites (Devor, Gourin-Lippman, & Raber, 1985).

Rates of success of steroid injection appear dependent on two factors. First, the proper site of injection must be determined because low doses of steroids are necessary when repeated injections are contemplated to avoid side effects. This is especially important because phantom tooth pain patients may appear initially vague regarding the site of pain. In fact, the pain can often be localized at specific sites aided by a careful history and physical examination. Some of these sites are at the teeth; others are at the terminal points of the divisions of the trigeminal nerve, and still others are at sites associated with other neuralgias, e.g., trigeminal, occipital (Fig. 2). Repeated clinical trials help to establish the correct injection sites for each patient.

The second factor in success is early treatment (Bach *et al.*, 1988). Steroids apparently promote peripheral nerve recovery most effectively when the injury is recent. Following injury of a peripheral nerve and a brief shutdown of neural activity, the injured axon forms endbulbs and sprouts. These sprouts differ from the parent nerve in an essential way. They readily generate action potentials either spontaneously or following mechanical,

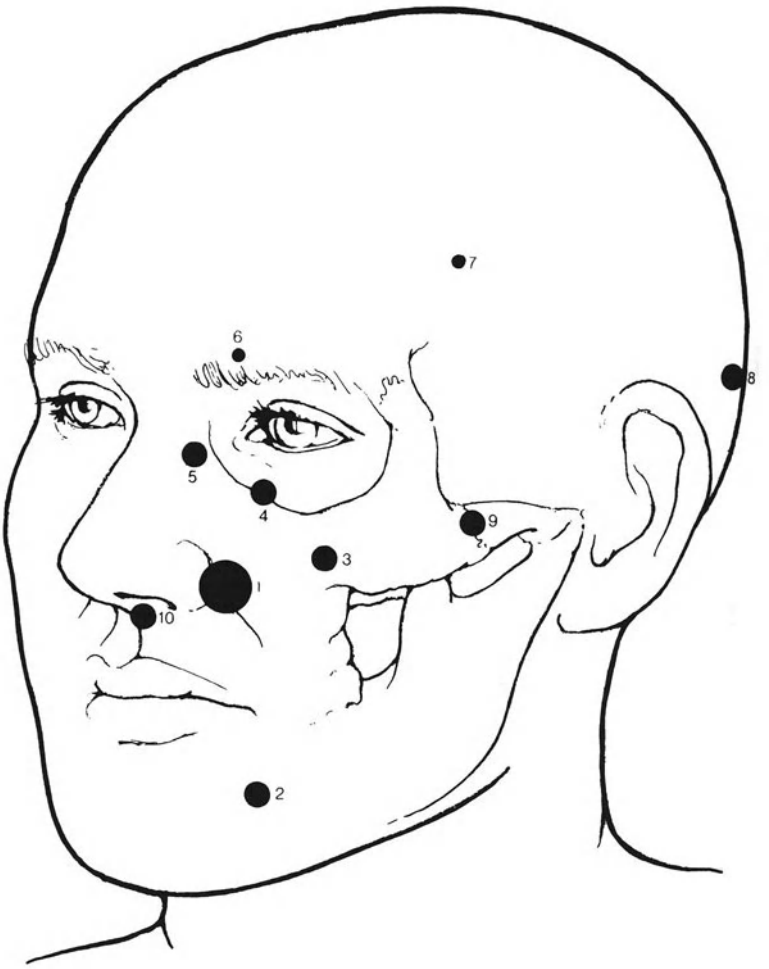

Figure 2. Dots indicates areas most commonly identified as painful by phantom tooth pain cases. Circle size indicates frequency of complaint of these most common specific sites of pain: 1, nasolabial fold; 2, mental nerve; 3, maxillary sinus; 4, infraorbital nerve; 5, palpebral nerve; 6, supraorbital nerve; 7, temporalis muscle area; 8, occipital nerve; 9, external pterygoid muscle area; 10, infranares.

chemical, thermal, or metabolic (i.e., ischemia) stimulation. If these sprouts reconnect with the skin or tooth pulp, more stable electrical characteristics are likely to be established, and the hyperexcitability state recedes. Fortunately, as Wall (1992) emphasizes, not all neuropathies result in pain.

General Surgery

Most neurosurgical and dental approaches have been tried on phantom tooth pain, mostly with extremely poor results. Recently reviewed data on neurosurgical treatment of pain syndromes of the trigeminal system were not encouraging (Rawlings & Wilkins, 1991). Their findings indicate that postneurosurgical patients make up the most recalcitrant of facial pain patients.

Dental

The most common treatment for phantom tooth pain is further endodontic therapy followed by apicoectomy and tooth extraction. There are two patterns to the clinical histories of phantom tooth pain cases. In the first case, a dentist or dental specialist is called on to examine a patient already suffering deafferentation pain, the result of an injury, illness, or surgery (e.g., Cladwell-Luc, rhinoplasty, silicon injection in the face). If a "suspicious" tooth is found in the area of pain distribution, endodontic treatment may be performed. If no tooth is found, the patient may urge treatment of a sound tooth in the belief that the pain might be of dental origin. In the second case, routine endodontic treatment is performed properly, but pain persists or is more severe than it was preoperatively. An assumption is made that either additional endodontic treatment is necessary or the wrong tooth was treated. Apicoectomy and tooth extraction are also logical sequelae of this approach to pain management.

Psychological Considerations

The symptoms of phantom tooth pain are often considered to be of psychological origin by those unfamiliar with its clinical physical characteristics. Part of the problem is that phantom tooth pain is often confused with atypical facial pain and thought to have a psychological etiology. Many studies of atypical facial pain, however, suffer from methodological problems. In an extensive study, 115 phantom tooth pain cases were compared with a contrast group of 151 myofascial facial pain cases and 137 nonpain controls on a variety of personality characteristics (Marbach, 1993). Only one trait personality factor, locus of control, statistically differ-

entiates the three groups. The chief psychological difference of the phantom tooth pain sample compared with the control groups were higher scores on a measure of demoralization. Demoralization can be interpreted as a consequence as well as an antecedent of chronic pain. This study did not show that phantom tooth pain cases are characterized by a specific premorbid personality.

Social Factors

Besides psychological morbidity, chronic pain patients experience strained social interactions that result from unsolved problems related to their clinical management. In a series of studies, we have found that facial pain patients identify dentists and physicians as the chief source of their perceived estrangement and feeling of being psychologically flawed (Marbach, Lennon, Link, & Dohrenwend, 1990). These feelings of social stigma are not necessarily the result of the patient's personality problems. Estrangement results from the pejorative labeling of the facial pain symptom as a psychological disorder by the clinicians whom these patients consult. Lennon, Link, Marbach, and Dohrenwend (1989) found that facial pain patients reported estrangement and dissatisfaction with intimate relationships, particularly with spouses. Together these two studies show that many facial pain patients, in a search for a meaningful explanation of their chronic pain, encounter negative reactions from clinicians, family, and friends. Clinicians, in turn, have been shown to resent chronically ill patients, for whom they play a caretaking role. They tend to depersonalize those who do not improve over a long period.

Treatment of Phantom Pain from Other Areas of the Body[2]

There have not been any formal, controlled trials of treatments for phantom pain from the body, organs, etc. In view of the poor history of interventions found in clinical reports standing the test of time, it is difficult to know how much emphasis to place on reports of rarely noted phenomena having even rarer treatments. For example, Brena and Sammons (1979) reported that the one case of phantom bladder pain they had experience with responded well to relaxation training ("about a 75% reduction in painful phantom perceptions").

[2]This section is written by R. A. Sherman and D. E. Casey Jones.

Chapter 10

Into the Future

Richard A. Sherman

Although our knowledge of phantom pain has come a long way in the last few years, it still has a long way to go. It is probably more productive to think of the current status of the field as being a work in flux rather than a mature, stable area with the solutions neatly laid out.

Putting Our Knowledge to Use

What to Stop Doing

Ineffective Treatment

Possibly the most critical path to the future leads through recognizing past misconceptions and mistakes and then putting them behind us. We need to let go of the idea that some interventions are worth doing because we know how to do them and they sound as though they might work even through they have been discredited. These range from the highly invasive procedures described in Chapter 8 to equally ineffective but harmless treatments such as phantom exercises. Many treatments have unintentional side effects or minor components that alter the physiological concomitants of phantom pain for a while so they seem to be effective if no follow-up is done. We need to learn to take these temporary changes into account when we evaluate the potential usefulness of a treatment.

We know that heroic measures such as major surgery on an apparently healthy residual limb or on the spinal cord are not effective. Psychiatric intervention with psychologically normal people reporting phantom pain is equally ineffective. Thus, we are aware of the strengths and limitations of the state of the art for now and can use that knowledge to avoid giving

harmful treatments and to anticipate which patients we are unlikely to cure.

Unrealistic Publishing

We know that most of the literature on treatment of phantom pain is wrong. The treatments they confidently espouse simply have not stood the test of time. Thus, editors need to be encouraged not to accept manuscripts based on tiny group studies with follow-ups less than 1 year that purport to demonstrate effective treatments for phantom limb pain. The plethora of these articles only continues to confuse practitioners without helping amputees.

What We Can Do Now with Current Levels of Imperfect Knowledge

Patient Education

We know that much of the anxiety causing magnification of relatively minor pain and phantom sensations just after amputation results from patients' lack of knowledge of phantom sensations as well as the surgical and rehabilitation processes. Updated handouts such as the one in Appendix II that explain these factors need to be given out to every patient before the amputation or as soon after it as possible.

Fitting Treatment to Pain

We know that the physiological precursors for the two most common descriptions of phantom pain (burning/tingling and cramping/twisting) have been identified and effective treatments are available. Thus, rational choices based on description of the pain can be made. We also know that physiological correlates for a few common descriptions of phantom pain, such as shocking and shooting, are unknown, and no treatments are consistently effective for them.

Some of the Explorations that May Bear Fruit

Changing Prosthetic Holders to Decrease Phantom Limb Pain

Because phantom pain is frequently caused directly or indirectly by problems with current methods for mounting the prosthesis, further ef-

forts to develop a percutaneous prosthesis holder mounted directly in weight-bearing bones should be encouraged. All published attempts to develop such a device that we are aware of failed because of infections along the rod passing through the skin into the bone. Although there are rumors of Scandinavians using such a device with humans, we are not aware of any formal studies currently under way. Long-term tests of a percutaneous prosthetic holder are under way in goats (R. Sherman, E. Lisceki, and P. Deffer, unpublished data). After 6 months, all three goats in which the device has been inserted have shown no signs of infection and are ambulating normally. Unfortunately, the study currently has not been able to attract funding, so it is likely to stop within a year of this book being published.

Prevention of Initial Occurrence

More studies of preemptive analgesia are required. Specifically, the relative contributions to phantom limb pain of preamputation pain, noxious surgical inputs, and postoperative inputs from the wound need to be determined. Tests of pre- and during-surgery methods for preventing the initial occurrence of phantom pain are crucial. The studies are very doable and simply need to be repeated with larger groups with longer follow-ups.

Use of Basic Knowledge of Neuromas and Damaged Nerves to Detect Physiological Precursors of Shocking–Shooting Phantom Pain

The information reviewed about neuromas and damaged nerves points toward there being relationships between these phenomena and shocking–shooting phantom pain. Investigations using stepwise series of nerve blocks should be performed to determine for the individual amputee which of these sites is crucial.

Relationships between Changes in the CNS and Phantom Pain

Of especial interest are the current studies showing that the homunculus changes proportionately more with greater intensities of phantom pain and shows virtually no change in amputees who do not have phantom pain. A study planned for the near future will compare brain scans of amputees having phantom pain before and after treatment so that differences within one person can be ascertained.

Long-Term Follow-up Studies

There are still a dearth of studies following amputees for at least 6 months after treatment. Until these studies are performed, the efficacy of any treatments will remain in doubt. These studies have to be performed at many institutions at once in order to get sufficient numbers of subjects. Sad experience has shown that practitioners will not participate in these kinds of studies for free (Sherman & Goeken, in press), and it is nearly impossible to get funding for them.

Appendix I

Literature Review

Joel Katz

This Appendix presents, in tabular form, a literature review in chronological order showing case reports and group studies in which somatosensory memories (SMs) have been reported. Information presented includes the number of cases, a brief description of the type and location of the original experience that later recurred as a SM, the temporal proximity of the original pain to the time of deafferentation (SMp or SMf), and the nature of the deafferenting lesion that brought the SM to the attention of the author(s). SMp designates a somatosensory memory of a *prior* pain present at or near the time of amputation; SMf indicates a somatosensory memory of a *former* pain in which a pain-free interval was experienced at the time of amputation [see Katz and Melzack (1990) for more precise definitions of SMp and SMf].

Author(s), year, and number of cases reported	Description of type and location of original experience and subsequent somatosensory memory	Type of SM	Nature of deafferentation
Mitchell (1872) (n = 2)	Following a gunshot wound, "the thumb turned into the palm and continued in this state of spasm, so that when the limb was removed six hours later, the nail of the thumb had cut into the palm" (p. 355). Nine years later, the phantom thumb continued to feel as if it were cutting into the palm.	SMp	Amputation
	Sensation of phantom hand and fingers in rigid extension just as they had been during the 2 weeks preceding amputation.	SMp	Amputation
James (1887) (n = 3)	One case each of a blister on the heel, and chilblains on the toes, present at the time of amputation and later felt in the phantom limb.	SMp	Amputation
	Sensation of toenails of phantom foot needing to be cut	Not reported	Amputation
Jackson (1889) (n = 2)	Hand blown off when a cylinder containing explosives discharged. The phantom hand is in the same posture, as if encircling a vessel.	SMp	Amputation
Charcot (1892) (n = 1)	Sensation of a tight wedding ring on phantom finger (10 years after amputation) and, from time to time, the pain that accompanied the removal of the ring before amputation.	SMf	Amputation
Pitres (1897) (n = 1)	Pain and sensation of a corn on the dorsal surface of the small toe, present at the time of amputation, continued to cause the patient distress after the limb had been removed.	SMp	Amputation
Kogerer (1930) (n = 1)	7–8 years after a forequarter amputation, the patient continued to feel the pain of the incisions that had been made on her thumb and index finger during multiple surgeries before amputation.	SMp	Amputation
Van Bogaert (1934) (n = 2)	One patient reported his phantom foot felt like a "wooden sole," his big toe "like a large thorn" 7 years after amputation. These were the same descriptions he used prior to the amputation, when his foot and toe were painful.	Not reported	Amputation
	For 5 days following spinal anesthesia, the patient felt his legs to have assumed the same position they had been in during the operation.	SMp	Spinal anesthesia

Lhermitte & Susic (1938) (n = 3)	Experienced pain in phantom foot of the crush injury that led to amputation.	SMp	Amputation
	Burning pain of gangrene, experienced in the small toe prior to its amputation, persisted in the phantom toe.	SMp	Amputation
	Knee pain from a tumor persisted in the phantom 23 years after amputation.	Indeterminate	Amputation
Leriche (1939, 1947a, 1947b) (n = 5)	Sensations of patient's hand flattened against the wall, tearing of skin, and pain from wounds incurred in a car accident persisted in the phantom 3 years after amputation. Duration of preamputation pain, 1 hr.	SMp	Amputation
	Painful tingling experienced in the hand at the time of a crush injury to the elbow 3 days before amputation of the hand was still present in phantom 20 years later.	SMp	Amputation
	Prior to amputation, the region surrounding the Achilles tendon had become ulcerated and painful but had healed completely by the time of lower limb amputation several months later. Six years after amputation, and following an injection of lipiodol into the stump, the pain of the ulcerated Achilles tendon instantly reappeared and never left.	SMf	Amputation
	A posttraumatic pain that had been relieved 2 years earlier by cervical sympathetic ramisection was reexperienced following an injection of a local anesthetic into the maxillary nerve for routine dental work.	Not applicable	Not applicable
	A pain of traumatic origin, located in the hand and fingers, that had been relieved 6 years earlier by stellectomy, was re-experienced in an overworked labourer following heavy use of a hammer.	Not applicable	Not applicable
Bailey & Moersch (1941) (n = 1)	Pain from a wood sliver that had been under the nail of the index finger for 1 week before amputation of the arm was still present in the phantom 2 years later.	SMp	Amputation
Riddoch (1941) (n = 2)	Patient's right arm blown off by an explosion of the bomb he was holding. The phantom hand continues to be in the same posture.	SMp	Amputation
	Left arm amputated after 3 years of pain. Removal of the arm failed to alter the posture or pain, which persisted in the phantom hand and fingers.	SMp	Amputation
Minski (1943) (n = 3)	Wound in the knee was reexperienced in the phantom knee after amputation.	Not reported	Amputation
	Pain in big toe from gangrene was reexperienced in phantom toe after amputation.	Not reported	Amputation
	Pain from severe corneal ulceration persisted following enucleation of the eye.	Not reported	Enucleation of eye

Author(s), year, and number of cases reported	Description of type and location of original experience and subsequent somatosensory memory	Type of SM	Nature of deafferentation
Hutchins & Reynolds (1947) Reynolds & Hutchins (1948) (n = 14)	Tooth pain from dental work and extractions performed without anesthesia, or under nitrous oxide, was referred to treated (or site of extracted) teeth following stimulation of nasal mucosa up to 2 months later.	SMp	Dental work, tooth extraction
Browder & Gallagher (1948) (n = 15)	12 patients with severe pain before amputation indicated that the subsequent phantom limb pain bore a distinct resemblance to the preoperative pain.	SMp	Amputation
	Was holding a blasting cap between his index finger and thumb when it accidently exploded. Three years later, the phantom fingers remained in the same posture.	SMp	Amputation
	Severed flexor tendon in ring finger resulting in a stiff finger; 7 years later, amputation of the arm was followed by a phantom in which all fingers of the hand could be "moved" except the ring finger, which continued to feel stiff.	SMp	Amputation
	After a severe burn, the patient's leg gradually became acutely flexed as a result of a cicatrix. The phantom leg retains the same preamputation posture.	SMp	Amputation
Henderson & Smyth (1948) (records lost)	At least one case of each of the following reported in the phantom limb after amputation: discomfort of ingrown toenail, corn, compression of toes in a tight boot, the impression of a split fingernail, a painful whitlow, the sensation of a finger ring.	SMf	Amputation
	Wound from combat injury, sprained ankle.	SMp	Amputation
	The pain of suppurative arthritis, the sensation of a traction pin, pain in relation to pressure points and splints, the sensation of lice crawling under a plaster cast.	SMp	Amputation
Sliosberg (1948) (n = 1)	Feels the sensation of the handle of the cane he was holding at the time of the accident pressing against the palm of the phantom hand.	SMp	Amputation
Bornstein (1949) (n = 5)	Three patients reported the distinct sensation of a watch strap around the phantom wrist, one of whom felt the buckle especially clearly.	Not reported	Amputation
	Sensation of a ring on the middle finger of the phantom.	Not reported	Amputation

	Fourth and fifth fingers developed contractures from infection. After a below-elbow amputation, the phantom fingers were felt in the same stiff posture. When the phantom became painful, the fingers would swell just as they had prior to the amputation.	Not reported	Amputation
Szasz (1949) (n = 2)	Pain of a long-standing peptic ulcer persisted after vagotomy and after healing of the lesion as shown by roentgen examination.	Not reported	Vagotomy
	Same as above case, but ulcer symptoms were present intermittently for 45 years.	Not reported	Vagotomy
Stevenson (1950) (n = 10)	One case each of a painful knee, a bunion on the foot, and the pain of gangrene that had affected the middle two toes, reported these sensations to have persisted in the phantom after amputation.	Not reported	Amputation
Bors (1951) (n = 1)	Sensation of a decubitus ulcer on the elbow that had healed by the time of amputation was later experienced in the phantom. This patient also had the sensation that his fingernails needed to be clipped.	SMf	Amputation
	Among the spinal-cord-injured patients, seven felt a distended rectum, one a feeling of continuous defecation, and one of rectal tightness. Also reported were distended phantom bladders (n = 7), the sensation of continuous micturation (n = 2), and phantom erection (n = 3) in the absence of actual erection.	SMf	Spinal cord injury
Cronholm (1951) (n = 3)	Sensation of concrete, reported to have been under the fingernails of a patient at the time of the accident, persisted in the phantom.	SMp	Amputation
	Received a compound fracture of his leg in an accident; 4 months later the limb was amputated. The phantom sensations consisted of (1) a painful compression of the fractures and (2) the pieces of bone grating against each other. Phantom pains continued to cause distress 30 years after amputation.	Not reported	Amputation
	Pain of shell wound incurred 9 months before amputation was reexperienced in the phantom.	Not reported	Amputation
Gloyne (1954) (n = 1)	Ulcer pain persisted after subtotal gastrectomy and the removal of the lesion.	Not reported	Gastrectomy and removal of ulcer
Wallgren (1954) (n = 1)	Sensation of gentle pressure from the oscillograph cuff that had been around the thigh at time of lumbar puncture persisted once the cuff had been withdrawn.	SMp	Spinal anesthesia

Author(s), year, and number of cases reported	Description of type and location of original experience and subsequent somatosensory memory	Type of SM	Nature of deafferentation
Bressler *et al.* (1955a, 1955b) (n = 1)	Pain in phantom breasts was the same as she had had prior to both mastectomies.	Not reported	Mastectomy
Blood (1956) (n = 1)	Sensation of a "sore corn" on the small toe persisted in the phantom after the limb was removed.	Not reported	Amputation
Haber (1956, 1958) (n = 4)	Three subjects felt the sensation of rings on their phantom fingers; in one, the sensation disappeared 2 months after amputation.	Not reported	Amputation
	Sensation of a watch around the phantom wrist was present after amputation.	Not reported	Amputation
Engel (1959) (n = 4)	Bouts of severe pain in the right side of the patient's forehead were identical in location and character to the original pain experienced when a home-made bomb he was preparing exploded prematurely.	Not applicable	Not applicable
	Ear pain from past otitis continued to recur after initial inflammation.	Not applicable	Not applicable
	Throat pain from past peritonsillar abscess recurred periodically.	Not applicable	Not applicable
	Pain from past cystitis recurred on occasion.	Not applicable	Not applicable
Nathan (1956, 1962, 1985) (n = 4)	Pain in the left lower limb of a cut from an ice-hockey skate was reexperienced in the phantom leg 5 years after accident.	SMf	Amputation
	Pain in the right knee of a severe comminuted fracture of the patella, which had been excised 6 years earlier, was reexperienced after bilateral cordotomy.	SMf	Bilateral cordotomy
	Painful ingrowing toenail present at the time of a spinal cord injury persisted in the phantom.	SMp	Spinal cord injury
	The "phantom consisted of a foot in a boot with his toes squelching in the blood which filled his boot" (1962, p. 133).	SMp	Amputation
Frederiks (1963) (n = 2)	The phantom arm was flexed and immobile, pressed on the chest, just as it had been carried for months before the amputation.	SMp	Amputation
	Phantom foot inclined to the left, as had the real foot before amputation.	SMp	Amputation

Farley & Smith (1968) (n = 34)	Sensations of fullness, feces, flatus, tightness and dragging in the phantom rectum following excision of the rectum. In one patient the sensation of tightness was distressing.	Not reported	Excision of rectum
Appenzeller & Bicknell (1969) (n = 15)	In 15 patients the phantom pain was similar to that experienced before amputation.	Not reported	Amputation
White & Sweet (1969) (n = 1)	Pain in ulcerated areas of the foot persisted in the phantom after it had been amputated.	SMp	Amputation
Dorpat (1971) (n = 3)	Three cases of menstrual cramps or labor pains of the same duration, intensity, and frequency as those occurring before total hysterectomy. One patient was a 70-year-old who, on the second day after hysterectomy, remarked, "I haven't had cramps like this for 35 years" (p. 28).	SMf	Total hysterectomy
Varma *et al.* (1972) (n = 1)	One patient reported feeling the sensation of a wristwatch and a ring on the phantom wrist and finger.	Not reported	Amputation
Conomy (1973) (n = 1)	Incurred a spinal cord injury when thrown from his motorcycle. At the accident scene, he felt as though he was still seated upright on his motorcycle, although he was actually lying on his back. This sensation persisted for 15 hr and recurred intermittently over the ensuing weeks.	SMp	Spinal cord injury
Parkes (1973, 1976) (n = 7)	About half the patients with moderate or severe phantom limb pain likened it to the pain that they had experienced before the amputation.	SMp	Amputation
Friedmann (1978) (n = 11)	One case each of painful chilblains on the phantom foot, phantom "tennis elbow," a finger ring, and a wrist watch represented in the phantom. Four cases reported the sensation of a phantom shoe; three reported phantom bandages that once wrapped a wound.	Not reported	Amputation
Marbach (1978a, 1978b) (n = 1 or more)	In at least one patient who received endodontic treatment or tooth extraction to eliminate pain, the same pain persisted, referred to "phantom tooth."	SMp	Dental work, tooth extraction
Brena & Sammons (1979) (n = 1)	Discomforting sensation of "having a full bladder" and episodes of sharp, burning, pain recurred following the removal of the bladder for chronic cystitis.	Not reported	Removal of urinary bladder

Author(s), year, and number of cases reported	Description of type and location of original experience and subsequent somatosensory memory	Type of SM	Nature of deafferentation
Sherman *et al.* (1979) (n = 6)	37.5% of patients reported phantom limb pain of the same description and in the same location as the preamputation pain.	Not reported	Amputation
Roth & Sugarbaker (1980) (n = 11)	17.5% of patients reported phantom limb pain or other sensations that had occurred some time before the amputation.	Not reported	Amputation
Berger & Gerstenbrand (1981) (n = 6)	Six cases of spinal cord injury reported the position of the phantom body and limbs corresponded to the patient's position at the time of spinal cord injury.	SMp	Spinal cord injury
Danke (1981) (n = 10)	Two cases of corns on the small toe years before amputation reported a reexperience of these sensations in the phantom.	Indeterminate	Amputation
	Injury to the knee 15 years before R-A/K amputation recurred as phantom pain. Sensation of sprained ankle 13 years before L-A/K amputation recurred as phantom pain.	SMf	Amputation
	Injuries to the calf and toe 4 years before R-A/K amputation were reexperienced in the phantom.	SMf	Amputation
	Injury to the knee and a corn on the small toe 1 year before R-A/K amputation were both reexperienced in the phantom.	SMf	Amputation
	Sensation of a plaster cast on the leg and of a corn 5 months before R-A/K amputation were both reexperienced as phantom phenomena.	SMf	Amputation
	Cutaneous ulcers on the great toe and ankle 3 months before L-A/K amputation reexperienced in the phantom.	Indeterminate	Amputation
	Sensation of a plaster cast on leg 2 weeks before R-A/K amputation recurred as a phantom phenomenon.	SMf	Amputation

Reisner (1981a, 1981b) (n = 3)	Two patients with brachial plexus injuries following motorcycle accidents reported a third (phantom) arm and hand, which was in the same posture as the arm and hand had been at the time of the accident.	SMp	Brachial plexus injury
	Tooth pain persisted following extraction of the offending tooth.	SMp	Tooth extraction
Wall *et al.* (1985) (n = 2)	In 12.5% of cases, the location of their phantom pain was identical before and after amputation.	Indeterminate	Amputation
Jensen *et al.* (1983, 1985) (n = 21)	Phantom limb pain was similar to the preamputation pain in location and character in 36% of patients 8 days after amputation and in 10% of patients 6 months and 2 years later.	SMp	Amputation
Katz & Melzack (1987) (n = 6)	After a dorsal rhizotomy, the patient developed a painful phantom hand and arm. "Within hours of recovery, [her] index, middle, and ring fingers [were] cramped, little finger sore." Several years later, despite amputation of the arm, she continued to feel the same cramping, sore pain.	SMp	Amputation
	Pain from stepping on a nail that "tore a chunk" from her heel 2 weeks before amputation persisted in phantom for several weeks.	SMp	Amputation
	Pain from diabetic ulcers on the medial and lateral surfaces of the ankle persisted in the phantom.	SMp	Amputation
	Burning in the toes from gangrene that began 2 months prior to amputation continues to cause pain in the phantom toes 7 years later.	SMp	Amputation
	A "pleasant sensation of draining" in her arm and hand when having the arm drained of excess fluid on numerous occasions prior to amputation recurred in the phantom.	SMf	Amputation

Author(s), year, and number of cases reported	Description of type and location of original experience and subsequent somatosensory memory	Type of SM	Nature of deafferentation
Katz & Melzack (1990) (n = 29)	Cutaneous lesions, deep tissue injury, bone/joint pain, ischemic pain, painful posture/position, swelling, superadded sensations reported to persist or recur after amputation.	SMp (n = 25) SMf (n = 7)	Amputation
Lacroix *et al.* (1992) (n = 1)	Sixteen-year-old girl reported phantom foot was "flat and stuck in a forward position." This description corresponded to medical records at time of amputation (at 6 years of age), documenting a right flat foot locked in an equinovalgus position.	SMp	Amputation
Jensen & Rasmussen (1994) (n = 1)	Phantom arm and hand reported to be in the same position as at the time of a motorcycle accident despite total paralysis and sensory loss in the real hand. The phantom hand persisted in the same posture after the limb was amputated 8 months later.	SMp	Brachial plexus injury followed by amputation

APPENDIX II

The Amputee's Guide to the Amputation and Recovery Process

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Introduction

If you are about to have an amputation, you are about to experience a major change in your life. You will be in a very novel environment—the hospital—and will have to deal with new people, ideas, and language. You probably don't really know what to expect. You aren't likely to have a good idea of the sequence and types of events that will take place, of what you are going to feel, or of what is normal and what is not. You are probably worried about what can go wrong, how well you will recover, how long it will take, and lots of other things.

This guide has been prepared so you and your family will have a good idea of what to expect both before and after your amputation, so you can participate in your own care and recovery. You will be living with your amputation for the rest of your life, so you need to know what you are likely to experience, how to get along with one less limb, and what groups and literature are available to help you do it.

We don't expect most people to read every word of the guide. Look through it—pick and choose what you need to know now. Then hang onto it and read other parts as you need them. Share it with your family so they know what you are going through and what to expect.

The vast majority of people who have amputations do not know what to expect when they actually come to the hospital for an amputation. They know almost nothing about the surgery itself, what to expect in the way of length or difficulty in recovery, or what they are likely to feel—either physically or psychologically. This can be a very upsetting and frightening experience. You probably won't know much of the vocabulary used by your health care team and other patients. A brief look through the "translation of terms" section will give you a hint of what to expect.

Most people who have amputations can and do make excellent recoveries. However, recovery is a slow and physically exhausting process that requires considerable patience and understanding. If expectations are unrealistic, considerable anger and frustration can be added to an already emotionally draining situation. If you have a good idea of what to expect

and an idea of some of the concepts and words that will be used around you, you will be far more at ease. You will also have a much more realistic idea of how much recovery you can expect and how long it may take. This is invaluable in planning your future and guiding your family and friends.

It is vital to understand that your recovery can be prevented or slowed as much by a poor attitude as by your physical condition. Therefore, you should make every effort to understand what is likely to happen. It is especially important to read the sections on the psychological effects of an amputation and on phantom sensations. If you are not aware of normal reactions and feelings, you may upset yourself needlessly by thinking that something is wrong with you when you are actually reacting normally and are experiencing very normal feelings coming from the cut nerves.

Amputees are not rare. There are at least 26,000 living veterans who have had amputations as a direct result of military service and 10 to 15 times that number of civilian amputees. About twice as many men as women have amputations. Two major groups of people have amputations: relatively young, healthy people who are hurt in accidents or who develop tumors and older people with chronic diseases that eventually cause such severe problems that a limb must be amputated to save the patient's life. The most common reasons for a limb to be amputated among adults are poor circulation and when medical or surgical treatment is unable to restore, reconstruct, or salvage a damaged or diseased limb. Amputation is a reconstructive operation, part of a process to replace an irrecoverably diseased or damaged limb with a more useful artificial one. The older group has more experience with hospitals, usually expects the amputation long in advance, and frequently knows other people in their situation who have had amputations. In other words, they know pretty much what to expect and have a chance to prepare emotionally, financially, and socially for the event. The younger group almost never expects and amputation. Many are seriously hurt in motorcycle crashes, sports mishaps, or automobile crashes. Thus, many are from the group of relatively young people who, until the injury, believed they were immune to substantial harm.

Relatively young, healthy people can recuperate physically much faster than older, chronically debilitated people who have to overcome the remaining aspects of their chronic disease as well as the effects of amputation. However, the younger group is almost never as psychologically prepared as is the older group, so differences between them can equal out quickly. All in all, the better shape you were in prior to your amputation, and the more will power and effort you put into your rehabilitation, the quicker and more fully you will recover and return to your usual activities.

A few words about your mental state just before and after an amputation: You are under incredible stress during this time. Nobody thinks

clearly when under stress. Many of the anesthetics used during the surgery leave your mind somewhat "cloudy" for days to weeks afterwards. Unfortunately, you can not recognize the change in your ability to think clearly. Many prospects may appear bleaker to you than they would if you weren't under stress. Just before and after your amputation are not the times to make critical decisions! This guide can give you knowledge about what help is available and what you are likely to be able to do, but you may not make the best decisions for yourself based on this information. You ought to avoid major decision making for at least several weeks after your amputation and perhaps until you are well into your rehabilitation.

A few hints about working with your health care team: You are going to meet over a dozen professionals who are very important to you. Because you will meet so many so fast, because you are not really at your mental best just before and after the surgery, and because their roles are unfamiliar to you, it will be next to impossible to remember who is who. It really helps to write down the names and roles of the people you meet. You are very likely to be out of sorts because of the normal stress and exhaustion common following any major surgical procedure. This will be made worse by having to deal with people prodding you to do unfamiliar, somewhat scary activities that make you hurt. They may assume you understand why and how they want you to do something when you don't. Please have patience with everybody and ask questions. Try to keep as good a mood as possible. Your health care team understands that you are in pain, are going to be a bit grumpy, and may not be thinking too clearly for a while, so they are ready to meet you halfway. Remember that you may have to work with these people for months. A good, friendly working relationship will speed your recovery and make your life in the hospital and rehabilitation center far easier.

This booklet is a general guide to what will happen, but the specific details of your own amputation and rehabilitation may differ somewhat from what you find in this book, depending on your individual needs and the health care setting.

Chapter 1

Life and Events on the Ward before and after Amputation

Overview

While you are in the hospital, your closest contact with health care providers will be with nurses. They play a key role in the coordination of your care. Registered nurses (RNs) have at least several years of training and frequently have additional training and experience in their field of specialization such as orthopedic nursing. Nurses usually work in rotating shifts, so you will not always have the same nurse. However, most of the same team will probably be present throughout your stay, so you will have an opportunity to get to know most of them well. They are interested in all aspects of your life at the hospital—not just problems with your amputation. They know that all of your feelings affect your progress toward recovery. When you have questions, do not hesitate to ask. You are not expected to arrive on your ward knowing its routines or understanding all of your medical problems. If you do not ask questions, you could cause yourself unnecessary anxiety and discomfort as well as slowing down your recovery. Nurses are very busy. They may not be able to come just after you call and sometimes may not be able to stay and chat as long as both you and they would like. The evening and night shifts are generally quieter. A more leisurely atmosphere exists during those shifts, but they are more lightly staffed than the day shifts, so each nurse has to cover more patients.

Before Surgery

You are likely to have blood tests and x-rays before your surgery. These may be done before or after your admission to the hospital. If you have questions about any of the tests, don't hesitate to ask. You have a right to know. You may be admitted to the hospital on the day of surgery, or, if

you have medical problems that require evaluation in a hospital setting, you may be admitted one or more days before surgery.

Prior to surgery you will meet with a member of the anesthesia team. He or she will explain the anesthesia options available to you (general anesthetic, spinal block, etc.). With this person's help you will choose the type of anesthesia best for you.

You may also be visited, before surgery, by a physical therapist, an occupational therapist, a prosthetist (someone trained to make and fit artificial limbs), or all of these. These health professionals coordinated your rehabilitation and will gladly answer your questions and concerns. They may not cover everything you want to know. The major cause of fear and worry is the unknown. If you have questions or don't understand what you've been told, *ask*!

Just after being admitted to the ward, you will be asked questions about your medical history to help in planning your care and in preventing problems. For instance, information about medications you are taking or allergies you may have is extremely important. Any special concerns or needs you might have are important to mention so the health care team can help you go to surgery with as few worries as possible. The nurses and doctors need to know about any pain you have been having and what you have been doing to relieve it. Each person experiences pain differently and responds to it and to pain medications individually.

Surgical pain is very different from chronic pain. The best part about it is that it decreases each day and soon goes away completely. Many patients who have had severe chronic pain eliminated by surgery do not complain about postsurgical pain at all.

The registered nurses (RNs) on each shift normally try to talk to each patient about his or her progress. If you have any special concerns or requests, they will handle the problem on the spot, or if necessary, contact your doctor. The nursing staff tries to give each patient as much independence as possible. The idea is that the sooner you can get around on your own, the better you will feel, and the sooner you will be able to go home. The nurses know that it is important to talk with you frequently about your progress because the best way to be sure the entire team, including you, is trying to reach the same goals the same way is to keep communicating.

Family and friends are especially important to anyone's recovery. Sometimes both the patient and staff forget that families are going through almost as much stress as the patient and need to talk things out too. When family members visit you, introduce them to the nurses and let them know whom they will be talking with if they call the unit.

There are many diseases and germs in hospitals to which children have not developed sufficient resistance. For their own safety, they are

usually not allowed on hospital patient units. Additionally, children may not understand other patient's needs for rest and quiet. Unless approved by your doctor and the nursing staff, it's probably best to have children visit you after you have recovered sufficiently.

Hospital wards are not always the quiet places we perceive them to be. If may be difficult to get a complete night's sleep because of people passing in the hall or entering your room to give you medicine or monitor your "vital signs" (heart rate–pulse, blood pressure, and temperature). You will probably not be restricted to your room but, rather, will be encouraged to go to the ward's lounge and possibly to eat in the hospital's main cafeteria or central patient eating area. The idea is to help you meet other patients, to help you learn your way around, and to begin to feel "at home" as quickly as possible.

After Surgery

The most common general complication associated with amputation is infection. Even with modern sterile techniques and antibiotics, infections can occur, so good wound care is critical to successful healing. You play an important role in this vital aspect of the process as you do with all other aspects of your recovery.

After recovering from anesthesia, you will return to your "unit" (ward). You will be checked for possible problems such as bleeding from the wound site and will be monitored to make sure that complications are avoided. For the first 12 to 24 hours, you will have your blood pressure taken frequently and will be asked to cough frequently. Coughing clears mucus from your lungs and keeps airways open, helping to prevent fever and pneumonia. Depending on your type of anesthetic, you may be encouraged to drink fluids. If you have an intravenous infusion (IV), this will probably be stopped when the nurses are sure you can take fluids well or when IV medications have been stopped.

Ice packs are sometimes applied to the operated area over your bandage to reduce swelling and pain. You will probably not get any pain medication until most of the anesthesia is out of your system. You may be having pain when you are first settled back into your bed, but once you are made comfortable, it will probably subside. The nurses will watch you and give you medication as soon as they feel it is safe.

One of the big surprises for patients who have never had surgery before is how soon you are encouraged to get out of bed and move around. If you have had a leg removed, you may be in a wheel chair for a while. The odds are good that if there are no medical complications, you will be up by

the day after surgery. The reality of recovery is that the longer and more you stay in bed, the longer it will take to recover, and the more debilitated you will be. Thus, it is vital that you get up and start moving around as soon as possible.

The length of hospital stay following an amputation varies widely with the type of amputation, the age and general medical condition of the patient, the medical care setting, and other factors. If good outpatient services are available, you could be discharged after only a few days. Your rehabilitation will begin in the hospital but will extend long beyond your hospital stay. This rehabilitation may be undertaken on an outpatient basis with you staying in your own home and going to the places you must to receive your rehabilitative help, or you may go from the hospital to a rehabilitation center, where you will stay until you are able to undertake more of your own care.

Patients and staff get to know each other well. The patients also get to know each other quite well and develop many friendships. Talking with other amputees can be very helpful. You can share experiences and get a good idea of what to expect. There are many people in the hospital to work with and talk to. Being open about any problems—either physical or those you feel may be "in your mind"—will help you achieve the good attitude toward you recovery that is vital to progress. Support is available through psychologists, social workers, ministers, and other health care specialists as necessary. It is important to know that you are never alone in what you are going through and that the health care team wants to help you as much as possible.

CHAPTER 2

Surgery

History and Safety of Amputations

Amputations have been common throughout the known history of humanity. The earliest "recorded" amputations took place about 36,000 years ago in Spain and France. One such record is an imprint of a hand with finger amputations found on a cave wall in Gargas, Spain. In Egypt, artificial arms have been found buried with 2000-year-old mummies. Military reasons for amputations have been common throughout recorded history. Thus, health care providers have more experience with how to perform amputations and how to rehabilitate amputees than with most other surgical problems.

Modern anesthetics are very effective and permit surgeons to carry out the amputation painlessly, carefully, and gently with minimal harm to tissues of the stump.

Preparation for Surgery

The most frightening part of an amputation is usually the surgery itself. In fact, with modern anesthetics, surgical techniques, and antibiotics, the surgery is not a life-threatening procedure unless a very rare reaction to the anesthetic takes place or the patient is seriously weakened by other injuries or severe chronic disease. Pain during the surgery is virtually never a problem and can be well controlled afterwards.

Anesthesia

Anesthesia is provided by anesthesiologists or anesthetists. These health care providers are trained to keep you comfortable and "unaware" during your surgery. As mentioned earlier, you will probably be visited by a member of the anesthesia team prior to surgery. You will be asked questions about your medical history, allergies, previous surgery, and

other things knowledge of which will allow your anesthesia to be as safe as possible. During this time, you should ask questions about the different types of anesthesia that may be suitable for you as well as their advantages and disadvantages. There are numerous anesthetic techniques, and a member of the anesthesia team is well qualified to discuss these with you. Some types of anesthetic wear off very quickly. Some allow you to remain awake but not feel pain in the limb undergoing surgery. Your options may be limited by your medical condition. In any case, your anesthesiologist or anesthetist can advise and guide you as the two of you determine the best and safest anesthetic technique for you.

It takes a while for the effects of anesthetics to wear off. The effects and time to wear off depend on how long you were "under," the type and amount used, and your basic physical condition. Just after surgery, various members of your health care team will keep insisting that you cough and keep asking you about it. They are not just trying to annoy a sick person. They are insuring that you can take in enough air to speed your recovery and avoid complications.

Level of Amputation

The location of the amputation along a limb is referred to as its "level." Many amputees are surprised at the distance above the apparent site of the problem at which the limb is amputated. Many factors go into the surgeon's decision about where along the limb to cut. The four most important are:

1. The seriousness of the problem itself. In chronic illness conditions, the problem causing the amputation may be fare more extensive than it looks from the surface. In some cases, bones can be "soft" a foot or more above the site of an apparent skin breakdown. In accident cases, the bones and tissues may be destroyed well above the level where problems show at the surface. So, if the limb was amputated just above the level at which the problem showed on the surface, the limb would never heal properly, and no weight could ever be put on the residual limb stump, thus preventing reasonable rehabilitation. Enough blood must reach the end of the stump to permit healing and continued health. So the point along the limb where blood flow drops below adequate may determine the level of amputation.
2. The level at which the skin and near surface tissues can survive as part of a flap. The "flap" is the part of your skin and near-surface tissues that are used to cover the end of the amputation site. There

must be sufficient blood supply in nearby tissue and skin so that the flap gets enough blood to live. If parts of the flap die, you will have to have further surgery to correct the problem. This delays your recovery and could cause very severe illness.

3. Artificial limb type. The newer types of artificial limbs work best with certain lengths of limbs. A residual limb (stump) that is too long or short will interfere with good control. The best length of limb for a prothesis that will provide you the maximum movement and control with the minimum of discomfort will help determine the level of amputation. As you will learn, the wearing and use of a prosthesis is a real skill, so it is important to include your future use of a prosthesis in the decision. The residual limb (stump) must be appropriately padded to minimize rubbing of bones against the end of the limb and maximize your ability to control the prosthesis. There are many types of prostheses that allow a variety of levels of control and sustained movement. The type you are most likely to need in your daily life will play a role in determining the type of amputation done.
4. Knees and elbows. The surgeon will do everything possible to save the knees and elbows because the remaining limb is far more useful, and rehabilitation is quicker, with these joints functioning.

The Operation Itself

Because amputations are usually very straightforward, the operation usually takes only a few hours. An amputation is a careful procedure in which a flap is carefully designed (if one is to be used), and the limb is gradually cut away. As blood vessels are exposed, they are either tied off, or a special heater "melts" the ends of the vessels. Thus, there is almost no actual blood loss. The end(s) of the bone(s) are carefully shaped and padded with muscle, subcutaneous fat, and skin to optimize the fit and comfort of your ultimate prothesis. Then the wound is closed as much as appropriate and wrapped up. Sometimes, especially when the blood supply to the "flaps" is less than ideal, or in the case of preexisting infection, it is safest to leave the stump "wound" partially or even completely open to avoid complications. In those cases in which this technique is used, the wound may be closed at a second surgery or may be allowed to close on its own "by secondary intention." This is the safest technique to use in certain cases. Even amputation wounds left completely open will close and heal. Remember mother nature has been healing people far longer than medical science and in many cases does it better than doctors and nurses.

This guide is not intended to make you an expert in all of the varieties of amputations. Most are very similar and differ only in technical aspects. The figures on the following pages (Figs. 1 and 2) illustrate typical lower extremity amputations are provided to give you an idea of what the stump and underlying bones will look like. You amputation may well look similar to one of these, though the appearance and length of your stump may be somewhat different.

Remember that the muscles in your stump once went to the lower part of your leg. When you make some simple leg movement, you may have a considerable spasm (short series of cramps, which can be painful) in the

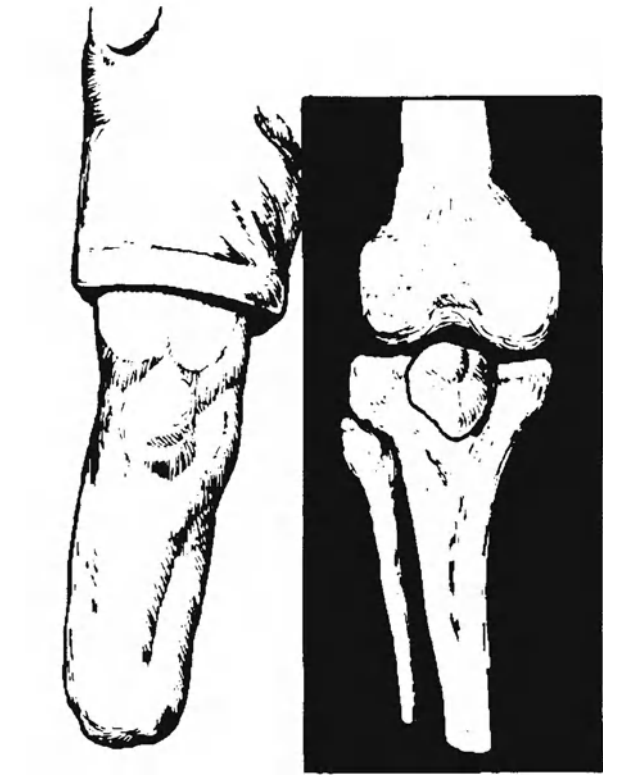

Figure 1. Below-the-knee (BK) amputation.

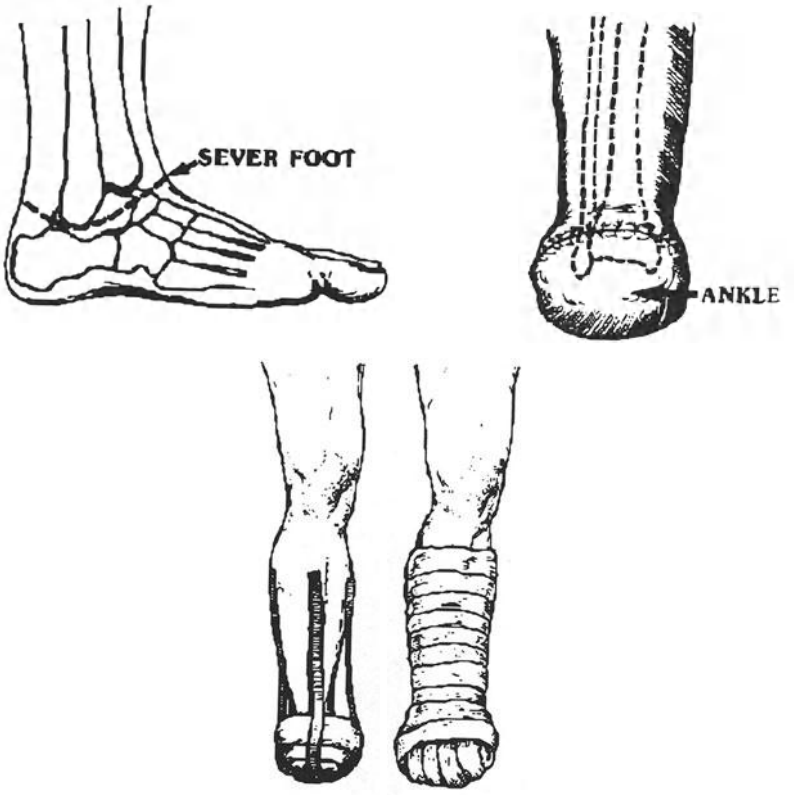

Figure 2. Syme's amputation at the ankle. Temporary bandage for the Syme's amputation.

remaining muscles because they no longer bear the weight or have the attachments they used to have.

"Leakage" of the Wound

All large cuts tend to drain fluid. You will be told what the normal leakage looks like and when to call the nurse if it is not right. A tube leading to a collecting bag may be placed into the amputation site to help drain off excess fluids so they don't collect beneath the skin. The tube will probably be removed in the first several days following your surgery.

CHAPTER 3

Healing and Pain in the Residual Limb

Healing

Healing takes longer the older you get. Older patients have relatively more difficulty fighting off infections, and the general level of strength and condition is relatively lower for older people than younger. For example, it takes longer to heal from a tooth extraction when you are 35 than when you are 25 years old. Thus, if you are relatively elderly and in poor physical condition because of long-standing medical problems and lack of exercise, you can expect a relatively long healing period. A young adult in excellent shape, both physically and mentally, who was not otherwise seriously injured during the accident that caused the need for an amputation might expect to heal in weeks to months.

Pain in the Residual Limb

As we understand it, the purpose of pain is to act as a warning to protect us and make us aware of disease and damage to our bodies. Pain is also important because it triggers both the brain and body (which really act as one) to mobilize the very complex system of healing, which includes the use of tissue proteins, hormones, vitamins, etc. Thus, when you are urged to eat well, it is not just to keep you happy and healthy but also to promote quick healing. Most pain sensors are near the skin's surface, but some are in muscles and other tissues. Pain is also a protection for ongoing healing. This is probably the largest cut you have every had. In spite of its size, it will heal similarly to small cuts you have had in the past. When you press on, or irritate a small cut, it hurts. The same thing happens with your stump until it heals. Once the stump heals, the mission of the pain is completed, so it should stop until the stump is harmed either from the inside or outside. You can expect the stump pain to decrease gradually after the operation

until it eventually goes away. Unfortunately, this is not always the case. For a very few people, it remains moderately severe, and for many it comes and goes as internal and external problems occur.

Until they heal, the nerves from the skin and muscles that were either cut or disturbed during the amputation are especially sensitive to chemicals released by bruised or cut tissues. If the stump is bruised significantly, these nerves can become incredibly sensitive to pressure and changes in blood flow within the stump, so considerable pain can be produced by even a minor bump or some changes in position. As healing progresses, the nerves become less sensitive. The stump may remain more sensitive than the rest of the body indefinitely.

Two common problems related to pain and healing in the stump are (1) the formation of neuromas and (2) poor stump pad position. Occasionally, when cut nerve ends heal, the ends grow into a convoluted ball called a neuroma. This can act as a short circuit in an electrical system and send pain messages to the brain that are not actually related to any damaging event in the stump. If the stump is not formed well or is not properly wrapped and shrunken, it will hurt when pressure is put on it. You and your health care team will keep very careful track of problems as you heal so they can be taken care of as needed. It is occasionally necessary to perform repeat surgery on some part of the stump or to remove a neuroma, so do not be surprised if you have to make a brief trip to the operating room some time after your initial healing period.

As you get older, you tend to get cramps at night both in your intact limbs and in the stump as well. These can be successfully treated with a variety of medications including calcium and muscle relaxants, depending on the underlying cause.

Cramps or apparently spontaneous severe jerks of the stump can occur at any age. They are frequently related to fatigue and overuse of the residual limb. When they aren't, you should ask your health care provider about the problem. The prosthesis may not be fitting properly, or you may need special training or muscle relaxants to ameliorate the problem.

Infections arising from skin irritation on the residual limb or from an infection that has spread from an area elsewhere in the body can cause severe stump pain. Because the stump may not have normal circulation, it can be more difficult for the body to fight off infections near the end of the stump, and medications take longer to work or are not as effective as they might otherwise be. Thus, don't allow an irritation to become infected. Take it seriously, and take care of it!

If you do not use your residual limb, it will get progressively weaker from lack of exercise, and your bones will get softer and easier to damage. This leads to easy bruising as well as pain from the muscles and bones. This

can be a very real problem, and there is no quick cure for pain resulting from damage to weakened bones. Prevention is the best approach. Keep your limb in good physical shape, and you will avoid most of the pain problems and disabilities that come with disuse.

Occasionally the end of the bone in the stump develops tiny spurs, which may cause irritation of adjacent muscles and severe pain with movement. They may have to be removed surgically.

We have surveyed over 7000 amputees. Over half of them report that they are bothered by stump pain at least a few times a year. It is incapacitating for a few but is simply an intermittent, endurable annoyance for most. The pain is frequently affected by the weather and physical exhaustion. Common pain medications and surgery have a mixed record of helping to relieve stump pain unless a specific problem can be identified and corrected. The underlying cause of the pain can be exceedingly difficult or impossible to determine, so the "trial-and-error" method of selecting a treatment is sometimes required to find the best remedy. Frequently, nothing helps, or the drug side effects are worse than the stump pain. The best thing to do in these cases seems to be to "grin and bear it." Some amputees slide slowly into alcoholism or become drug addicts as they attempt to self-medicate their pain. There is no need for this to happen!!! Your physician can refer you to pain specialists who can decrease the intensity of most cases of stump pain and help you learn to live with the remainder. Another common type of pain related to amputation seems to come from the part of the limb that has been removed. It is called "phantom limb pain" and is discussed in the next chapter.

Chapter 4

Sensations from the Part of the Limb that Was Removed (Phantom Limb Sensations)

Overview

One of the biggest surprises after an amputation can be discovering that sensations still seem to come from the missing limb and that sometimes its movement can apparently be controlled. Just after amputation, the phantom can feel so real that fresh (very recent, new) leg amputees occasionally try to stand up and walk away. Occasionally, the missing limb feels as though it is in a very uncomfortable position. Nearly all amputees report that they can sense the shape of the amputated limb for at least a few months after amputation. This "shadow limb" is called the phantom, and the feelings coming from it are called phantom sensations. Many continue to sense it all of their lives. In addition to the sense of shape, virtually all amputees report various feelings such as itching, warmth, twisting, etc. that seem to come from the limb. These feelings may change with time of day, fatigue, weather, and other factors. The great majority of amputees report that these feelings are painful at least occasionally.

Phantom Sensations

Feelings that appear to come from a limb that is no longer attached can be quite upsetting. It is important to understand that phantom limb sensations occur among virtually all amputees and that phantom limb pain is well recognized by the medical community. Your doctor and other health care providers will not think you're crazy if you tell them about a problem you are having with a portion of a limb that is no longer there. In order to understand how it is possible to feel something that isn't there, it is necessary to understand how the body is "wired" to feel things. Figure 3

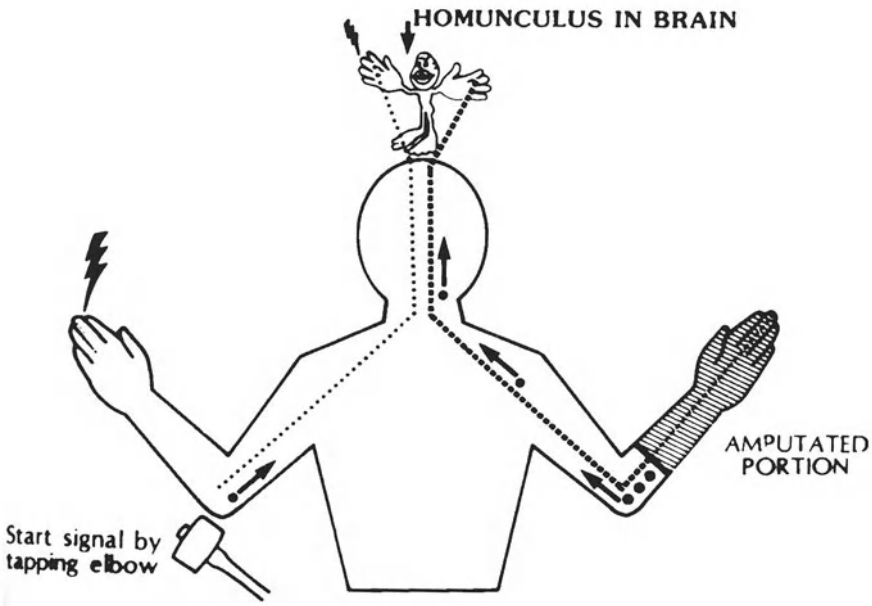

Figure 3. How pain can be felt in a part of the body different from where the pain "signal" started. When your finger is touched (lightning bolt), a signal travels along nerves past your elbow, through your spine, to your brain. The signal goes to a part of your brain (the homunculus) corresponding to your finger. You can send a signal to the same part of your brain by bumping your elbow (hammer) because the brain can't tell where it began. This is why your fingers tingle when you bump your "funny bone." The nerves and brain don't change much after an amputation, so you still feel your hand when you start a signal in the stump.

shows a diagram of how the brain and body are wired together to pick up feelings from your body. The upper rear portion of the brain contains a structure similar to a hard-wired telephone switchboard, which is called the sensory (for feeling) homunculus (for "little person"). It is shaped very much like a person and has a place for each nerve coming from your body surface. Thus, if you tap your right little finger, a signal that starts in the nerve endings in the finger runs through your hand, up your forearm, past your elbow, along your upper arm, into your neck, and eventually winds up at the corresponding right little finger in the homunculus.

The homunculus is not part of your conscious brain. It is essentially a switchboard. We do not think it can learn or change much after early childhood. It has no way of knowing where the signals reaching it actually started from. When you bump your elbow, you very often feel pain in your

fingers and hand. This is because the nerve that carries signals from your hands and fingers passes just under your elbow and is shocked when you bump your funny bone nerve. The homunculus does not know the signal actually started in your elbow, so it tells the conscious portion of your brain that the feelings came from the fingers and hand. Because it cannot learn, the pain in your hand continues although you consciously know that only the elbow was hit. This pathway is illustrated on the left side of Figure 6. Feelings that seem to come from one part of the body but are actually from another part are called "referred feelings."

Another way the homunculus becomes confused is illustrated by the feelings of cold and pain in the forehead that occur when you eat very cold ice cream. The nerve "wires" from the roof of the mouth run close to those from the forehead. This kind of cross-talk between nerves is similar to what happens when you are on the phone and hear another phone conversation in the background. Signals from one nerve are passed to another. The homunculus has no way of knowing that the transfer has occurred, so, even though you know you haven't been eating ice cream through your forehead, the homunculus still reports the forehead as being cold and painful.

A third way the homunculus is fooled about the starting place of signals occurs when a nerve is cut during surgery or by an accident. The raw end of the nerve is very sensitive to any kind of stimulus. Chemicals from cut or bruised skin can make it much easier for the nerve end to fire off a signal. Any minor pressure or other stimulus is enough to send a powerful signal to the homunculus. The homunculus has no way to know that the nerve has been cut and no longer runs from the area it used to serve, so it reports the feelings as coming from that area instead of the spot where the nerve was cut. Thus, the stimulus causing the feeling may not be where you feel it. If a limb is amputated, the three ways to confuse the homunculus discussed above still work. The homunculus still has no way of knowing that the limb is missing, even though you know perfectly well that it is gone. Anything that causes the nerves that once served the amputated limb to start a signal will cause a sensation that seems to come from the phantom limb. The "lightning bolt" hitting the left middle finger shows that when the end of the finger is stimulated, a signal travels through a series of nerves to a part of the brain (called the homunculus) that has a part for receiving signals from each part of the body. If the nerve serving the finger is hit as it traverses the elbow (instead of the finger being shocked), the signal follows the same path to the same place in the brain. So, the brain still thinks the finger has been hit and "feels" it there.

If the signal had started at the right elbow—in the stump of an amputated limb—the signal would still travel to the right middle finger

part of the brain, so the body would feel pain in that finger even though it is not there. You can not consciously convince the brain that the finger is not there, and the wiring does not change much as the years go by.

Phantom Pain

Pain that seems to come from one part of the body is frequently caused by problems in another part of the body. A common example is a person with a nerve pinched in the low back who feels pain running down the leg instead of in the low back. The signal starts in the back, but since the nerve is supposed to come from the leg, the homunculus reports it (refers it) as coming from the leg. If that leg was amputated, the pain would still seem to come from the same location on the "phantom" because little corresponding change occurs in the homunculus when the leg is amputated, and it cannot learn. This system was illustrated on the right side of Figure 3.

Painful feelings that appear to come from the amputated portion of the limb are called phantom pains. Some typical ones are illustrated in Figure 4. They may be burning, stinging, cramping, shooting, twisting, or other unpleasant sensations. They are always stronger versions of the painless phantom sensations. There is no reason to think that those amputees who report phantom pain are either exaggerating normal phantom sensations or have anything wrong with their minds. We have received over 7000 responses to questionnaires sent to amputees inquiring about problems with phantom pain. Over 80% of the respondents said that they had enough phantom pain to cause them real problems for at least a week every year. Most have episodes of pain that last anywhere from a few seconds per year to several weeks at a time, with several to many episodes per year. Some people have continuous pain that varies in amount from almost none to excruciating over the course of the year.

About half of the amputees who report phantom pain seem to be able to associate changes or onset of their pain with some change in themselves (such as stump irritation, exhaustion, back pain, or stress) or outside themselves (such as changes in humidity). It is important to note that two amputees who describe their phantom pain as being identical in frequency, severity, and type of feelings may report entirely different events that change the pain. For most amputees, phantom pain is worst just after amputation, while the stump is healing. However, it is not likely to go away permanently. A few amputees report that the severity does not decrease after stump healing but, rather, persists throughout life. Almost none of the respondents to our surveys reported that their phantom pain went away completely with the years after amputation. Thus, you will probably have

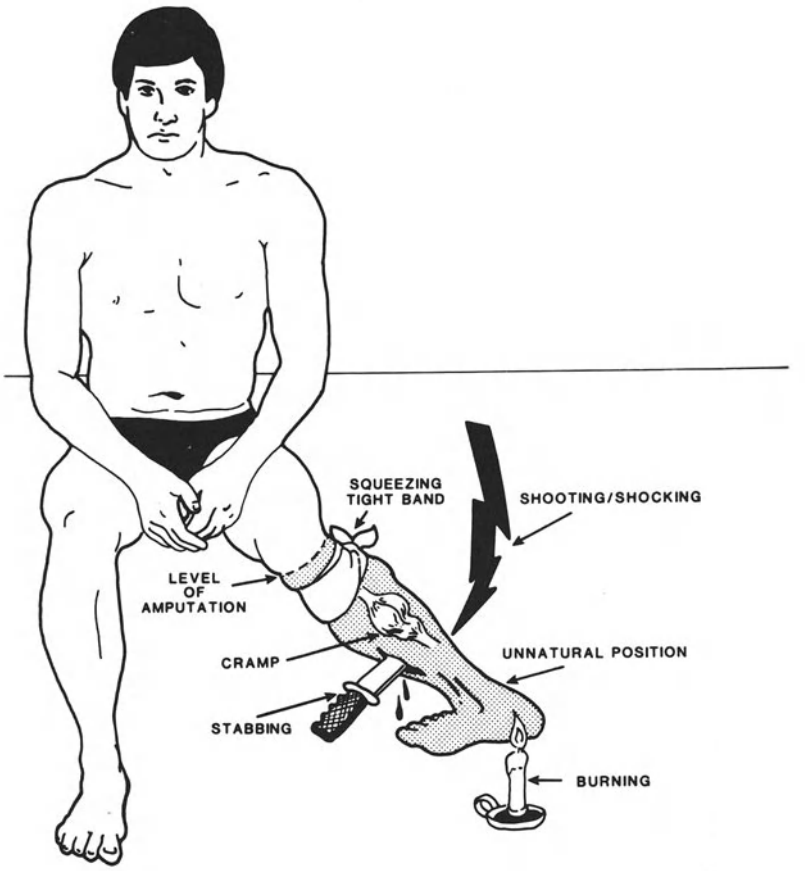

Figure 4. Phantom limb pain. Some of the typical painful feelings that seem to come from the missing limb.

some phantom pain. It may be enough to trouble you from a few times per year to almost all of the time. The amount of pain is likely to vary from almost negligible most of the time to severe once in a great while. You may be able to predict what causes your phantom pain to become worse and take measures to avoid the worst of it. Many amputees are afraid to talk about their phantom pain with their health care providers for fear of being thought to be crazy. Some reported that their health care providers either told them outright or strongly indicated that anyone who felt pain in a limb no longer present had mental problems and should see a psychiatrist.

There is *no* evidence or indication that amputees are any crazier (or more sane) than people who have not had amputations. (See the next

chapter for further discussion.) Most health care providers have learned that referred pain is a very common problem and that phantom pain is one example of it.

We now know what causes several descriptive types of phantom pain. For example, burning and tingling phantom pain is caused by decreased blood flow in the end of the stump, and cramping–squeezing phantom pain is caused by spasms in the stump. Unfortunately, we do not know what causes shocking–shooting phantom pain yet. Unless the treatment is related to the cause, it doesn't usually work. Most of the commonly used treatments for phantom pain do not have good long-term success rates because they are not related to the underlying causes. In addition to asking the 7000 amputees discussed above about their treatment experiences, we have surveyed many health care providers and have carefully reviewed the literature for rates of success of phantom pain treatments on 1-year follow-up. All three sources give the same answer. Many treatments offer temporary help, but (with the exception of those discussed below) even the best usually last only a few months to a year. A few of the thousands of respondents were helped significantly for an extended period of time, but each was helped by a different treatment. Surgery solely for treatment of phantom pain was not successful in any case. Do not become a victim by permitting an unknowledgeable physician to operate on you! Burning–tingling phantom pain is usually successfully treated by increasing blood flow to the residual limb. Cramping–squeezing phantom pain is usually successfully treated by decreasing muscle tension and spasms in the residual limb. Specific ways of accomplishing these changes include training you to control your own blood flow or muscle tension, use of muscle-relaxing drugs, and electrical stimulation.

There is a substantial incidence of alcoholism among amputees as a direct cause of attempts at covert self-treatment of phantom pain. Drinking alcohol does appear to temporarily reduce awareness of phantom pain, as it does for other types of pain. However, it is no more effective than other drugs, which are probably safer and are definitely easier to control.

CHAPTER 5

Psychological Reactions to Loss of a Limb

There can be no denying that the loss of a limb is a major event in anyone's life. The limb has been with you throughout your life, and you need it for normal functioning. As the fact of an amputation became clear, you probably thought about just what the limb does for you and your way of living. You know that you are going to have to learn to get along without it and are probably not sure just how well you will do. You are probably not sure about how much you will have to change your life style (job, recreation, etc.) or what the loss of the limb will mean to your relationships with people who are important to you. You may be worried about the pain from the amputation itself at the time of surgery and after recovery. All of these worries are natural and produce normal, predictable reactions.

Everyone reacts in his or her own way. Some are bothered by one aspect of an amputation more than others. Some people don't worry much at all. Some are terrified by the prospect of losing a limb or of having to face the future without it. You have your own unique blend of worries and uncertainties and your own way of handling problems. The important thing is that everyone reacts! Some people feel that they must hide their feelings and are better at putting on a "happy face" than others, but inside, everyone reacts to his or her worries and fears.

It is very important for you to be aware that you are likely to experience some of the very normal reactions to the loss of a limb that other amputees have reported. If you think you are going "crazy," the stress and anxiety can inhibit or prevent a recovery.

Some of the common reactions to amputation are discussed below. They are certainly not the only normal ones, and you may not have all or even some of them. For your own health, if you feel you may be reacting more than you should or that your reaction is abnormal, *ask*!!! Your health care team has worked with many amputees, but you have only your own experience to guide you. They are used to handling emotionally related

questions and can guide you to further sources of help in the cases where it is required.

When a person is unexpectedly told that a limb is going to have to be amputated or wakes up after an accident without a limb, a common first reaction is shock. People frequently either totally deny the need for amputation or have such feelings as "this can't happen to me" or "I won't let you do this to me." You may feel quite calm and believe that you are taking the entire thing very well. In time, many people start experiencing anger. It may be directed at themselves for getting into a state that requires an amputation or may be directed at others, including God and loved ones. It is normal to feel anguish and feel the need to cry out, "Why me?" This is a very normal "grief" reaction, and the physical and emotional steps in the process are well known. They will be recognized by your health care team but may not be obvious to you. The reaction is similar to what a spouse goes through when the husband or wife dies. If you do not believe that the process is powerful, consider all of the spouses who get very sick or die just after the death of their loved one.

You need to recognize the anger, understand that it comes from frustration and from losing control of your life for a while, and try to let it out of your system. It's too late now to go back and change anything, so you may as well get on with your life. If you don't let yourself get over the anger, you can use up so much energy that you will have far less available for recovery.

Before the amputation, there is usually a "bargaining" stage in which the patient tries to get God, the surgeon, or both to stop the amputation or remove the need for it. For most amputees, the physical need for the amputation is well understood and unhappily accepted as being vital. This generally does not stop the emotional reaction that leads to the bargaining process. Patients and their relatives may tell themselves that the problem is not as bad as it seems in spite of overwhelming, obvious evidence to the contrary. After "bargaining" does not work, a deep sadness and depression may set in. However, this normal emotional reaction makes everything look really bleak. Without realizing you are doing it, you may exaggerate the problems the amputation is likely to cause you in your daily life and emotional relationships. Depression drains your energy. You need all the energy you can get to recover physically and get on with your life. Your health care team recognizes the signs of depression and can help you get over it if necessary. The fact is that most people go through these stages of feelings but manage to get through them on their own. Almost everyone eventually snaps out of the sadness and reaches the acceptance stage of grief in which they face the facts, get the diseased limb out of the way, and get on with their lives.

All of the worries, concerns, uncertainties, and reactions discussed above, as well as the normal physical discomfort from an amputation, cause stress and anxiety. Everyone will suffer a different amount of stress and will express it in his or her own way. People in this situation are frequently short-tempered and very grouchy. You should be aware that prolonged anxiety and stress cause very well-known physical and emotional reactions. When you are under stress and are anxious, your body and mind try to get ready to defend themselves from an upcoming attack as best as possible. The body spends less energy digesting food and on curing infection because it is directing the energy so the muscles can fight off an attack. Blood pressure tends to go up so extra blood can get to the muscles. The muscles themselves tense up more than normal in preparation for fighting or running away. This costs you vital energy, which you need for recovery. You may get headaches and body aches from the sustained muscle tension. You cannot fight off infections as well as you should or digest food as easily as you could. You can expect to feel some anxiety. If you feel that you are more anxious than you should be, talk with your health care team about it. If there is a problem, they will help you deal with it.

At some point during your rehabilitation, you may get "really down," feel "blue," or become depressed by the amputation and the changes it may mean to your life. It is helpful to remember that depression is a normal reaction if it doesn't grow so big it overwhelms you or prevents you from seeing that life can still go on and has a bright side. It will help a lot for you to talk with your rehabilitation specialists and other amputees so you develop a realistic idea of just what your limitations are likely to be. The actual number of activities important to you that you will not be able to do or will have to substantially modify may be far smaller than you think. It may be hard to believe, but things usually do not look quite so bad after some time has passed, so give yourself a chance to adjust. The more active you are in rehabilitation efforts and in thinking about and planning for the many alternatives available to you in the future, the faster the "blues" will disappear.

How we feel about ourselves, our "self-concept" or "self-image," continues to develop and change throughout our lives. It is certainly obvious that a serious event, such as an amputation, can disrupt and make some changes in self-concept. These changes can be either positive or negative depending on how the person deals with the loss. The self-concept that emerges after an amputation may include a greater feeling of strength developed in the effort to overcome the losses that have occurred. On the other hand, some individuals may turn to a dependent life style, using amputation as an excuse.

Another aspect of your "self-image" is what the image of your body means to you emotionally. Very "macho" people as well as those who are highly concerned with the attractiveness of their bodies to the opposite sex tend to have more problems adjusting if they believe that their image will suffer in the "eyes" of the people they feel a need to impress. Many people are afraid that they will not be as well respected by their peers and business associates because they are "crippled." In fact, a large proportion of relatively young amputees much such good recoveries that you can't tell that they have a limb missing. Many are as active in sports as they were before the amputation. However, the reality is that many people will react to you differently! Almost everyone does adjust to the new body image. For some the adjustment is slower and more painful than for others. You and those close to you will have to have the patience, strength, and understanding to adjust to your new body. An important part of developing a positive self-concept after amputation rests on your ability to adjust your value system. In other words, try to avoid comparing your current situation with what "used to be." You will probably make a mistake in your comparison. Following a lower extremity amputation, it requires a good deal more skill to ascend a set of stairs than it did to run a mile with two normal legs. Recognize the effort you put into your rehabilitation and take pride in it. The most successfully adjusted people seem to be those who can view their changed capabilities in proper perspective with more positive events in their lives and have a good sense of humor and attitude about it.

The effects of the previously discussed factors are different for each amputee. Each individual who undergoes an amputation will deal with it in a unique, personal manner. The amount of actual disability resulting from the loss of a limb depends on how well the injured person handles the loss physically and emotionally. For example, research indicates that there is no direct relationship between the extent of physical loss and the individual's emotional difficulties: the difficulties are more dependent on the personality (including coping skills) of the individual than on the type of amputation. One person with a "limited" physical loss may have greater adjustment problems than someone with a "major" loss. For help in reaching an accommodation with your feelings, you may want to read Kushner's book titled *When Bad Things Happen to Good People*. The philosophy behind this book is highly controversial, but many people feel that it gives you a basis for further thinking about your disability in relation to God and fate in general.

In summary, the way you handle your loss emotionally may have as much or more impact in determining how disabled you are in the future than the physical problem itself.

CHAPTER 6

Physical Rehabilitation

Overview

Rehabilitation after an amputation is a long process. If a leg or foot has been amputated (lower extremity amputee), most of your training in stump care and walking will be done by physiotherapists or physical therapists (called P.T.s). These professionals have four or more years of training in helping people recover from a wide variety of debilitating problems. They are highly skilled in helping you learn to take proper care of your stump and in learning to walk with an artificial leg. You will be taught to take care of and wrap your residual limb (stump). You will be fitted with the artificial limb (prosthesis) most likely to help you develop a good way of walking (gait), carry out your daily activities, return to work, and participate in sports. If an arm or hand has been amputated (upper extremity amputee), you will probably work with an occupational therapist (called an O.T.). These professionals have four or more years of training in helping people overcome handicaps and disabilities. They are experts in such areas as teaching you to carry out all kinds of tasks with one hand or in modifying activities to make the best use of your prosthesis.

You will probably get to go home from the hospital as soon as you can get around by yourself safely, with or without an artificial limb. You will have to plan to return to the hospital or a local rehabilitation center very frequently for continued training and treatment.

One vital part of rehabilitation is extra care for remaining limbs. An intact lower limb tends to take more of your weight at first. A remaining upper limb tends to take over much of the work done by an amputated arm or hand. The extra stress on these limbs may be more than they can easily take, so you have to give them extra care and be sure not to overuse them.

Extent of Recovery

The extent of your recovery depends largely on your physical condition prior to amputation. If you were in good physical shape before the amputation, you will probably recover within a few months and be able to perform most of the activities you did before your amputation. Many amputees who are in good physical condition play basketball, hike, hunt, swim, work, and do most of the other activities their peers do.

Extent and speed of recovery depend mostly on these factors:

1. Your age and the length of the healing process. People heal more slowly as they get older. The more complex the amputation and its wound, the slower healing is likely to be.
2. The extent of other medical problems associated with the amputation such as burns, or diseases causing general debilitation such as diabetes, or not enough blood supply to the limb (vascular insufficiency), all of which tend to lengthen the recovery process.
3. Learning to use your prosthesis is hard work, so your overall physical condition and health will play an important role in how quickly you can progress.
4. How closely you follow the instructions of your physiotherapist. This is especially true of how much you do. It is easier to prevent problems than to cure them, so don't overdo!
5. How much you want to recover and learn to use your prosthesis. There is simply no substitute for determination. If you do not want to work at learning these difficult tasks, you will not get very far.
6. There are many psychological factors that can speed up or slow down your recovery. Support from family and friends, various social and economic factors can play an important role in either speeding up or virtually stopping your recovery.

Postsurgical Dressings

Just after surgery, any or all of the following basic types of dressings for your residual limb may be used.

1. Rigid dressing. Just after surgery, many patients are fitted with a rigid dressing (cast) to assure control of swelling and provide comfort. The end of the cast is made to take a simple training prosthesis usually called a "pylon" so training in standing and walking can be started immediately. A typical pylon is shown in Figure 5. This cast may have to be changed after several days or

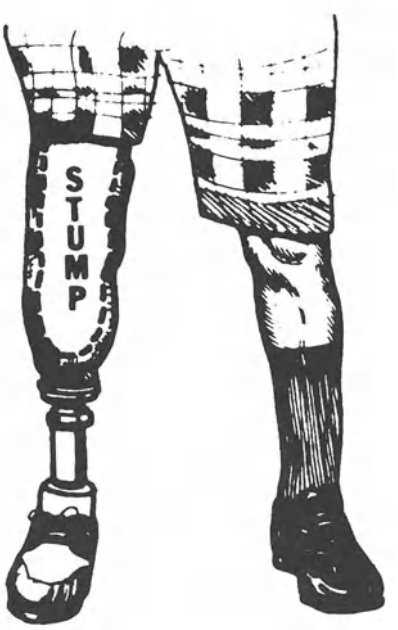

Figure 5. Temporary artificial leg for below-the-knee amputees.

weekly because it becomes loose from the shrinkage of your residual limb. It is held on (suspended) by straps going to a waist or shoulder belt to hold it in place. These casts are normally designed to take only a quarter of the body's weight, or 30 to 35 pounds. Too much weight being put on the cast may result in a delay in healing or even cause the wound to open.

2. Ace wrap. These elastic bandages are used to prevent swelling and encourage shrinkage of the residual limb, as discussed above. Bandaging techniques will be demonstrated and must be followed very carefully. Wrapping is started as soon as possible, even before complete healing.
3. Jobst compression pump. These air-filled sleeves (similar to blood pressure cuffs) place constant, equal pressure on all sides of the residual limb to shrink it rapidly and to shape it appropriately.
4. Stump shrinkers. These are elastic stockings used before your final prosthesis is made and adjusted. When you are not wearing your prosthesis, you will wear a "stump shrinker" or tensor bandage if there is a possibility that your residual limb will swell. They are

frequently worn at night as well as during the day. Initial shrinkage and shaping take about 6 weeks to 3 months, depending on your response and condition.

Residual Limb (Stump) Care

Residual limb care is a vital function that must be done correctly for the rest of your life. Just after amputation, the stump must be wrapped to help it shrink to its optimal size and shape as well as to avoid painful and dangerous swelling. You can not be fitted properly with a prosthesis unless the stump has been correctly taken care of and swelling is under control. The general objective of stump care is to insure that your stump can be easily fitted with a prosthesis and that you will have the physical capacity to operate it.

In order to be fitted for and use a prosthesis successfully, you will have to shape your stump into a cylinder with the help of proper wrapping techniques and keep it free from swelling, infections, sores, wounds, and irritations.

Skin and Joint Problems

Chronic skin disorders and stump contractures (where the muscles and tendons shorten up from disuse so that a joint is bent and cannot be straightened) can be long, painful processes to overcome. So you must do all that you can to keep this from happening. You can cause the muscles and tendons to shrink up and shorten so much that you cannot straighten your limb or use the joint by developing bad habits such as sitting with a BK (below the knee) stump bent. Allowing the stump to hang down causes the same problem. You will be given detailed instructions on how to avoid problems. You will also be given exercises to help avoid problems and to strengthen your muscles. If you follow them carefully, you should avoid most complications. The most important positions to avoid are illustrated in Figure 6.

Residual Limb Hygiene

While you are wearing your prosthesis, fresh air is not able to get to the end of the stump, and moisture cannot evaporate. This is particularly true if the prosthesis is one of the suction-socket types discussed later. The lack of air circulation may promote skin infections and abrasions, which may

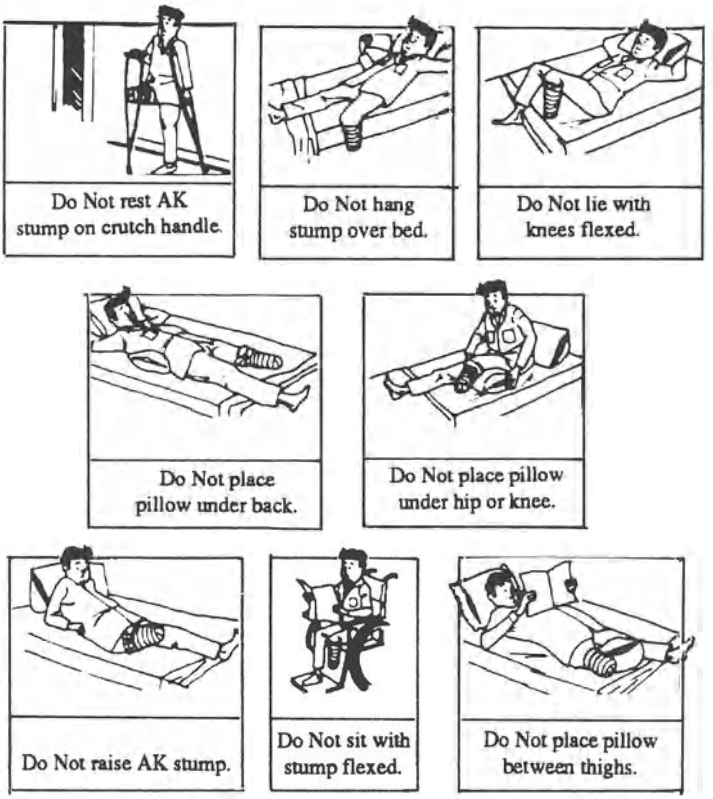

Figure 6. Positions new amputees should avoid. Figure adapted from one appearing in *Limb Prosthetics*, 4th edition, 1979, published by Hanger, Inc. of St. Louis, MO.

keep you from wearing the prosthesis. Daily use of a prosthesis puts your skin under a lot of stress and can cause the skin to break down or become irritated very quickly if you are not careful.

You will be instructed on how to care for the prosthesis, the special stump socks worn with most prostheses, and especially for your skin. Each evening the limb and the prosthesis must be washed with warm water and dried with a soft towel, and powder or corn starch must be applied. Stump shrinkers and socks must be changed and washed daily. These items are delicate and must be hand washed, dried on a flat surface (rather than hung on a clothes line or dried in a dryer). The key is to keep you and your

equipment clean and dry. It takes only a few days to cause skin damage that can take months to heal.

Wrapping and Bandaging

When the residual limb has started to heal, bandaging is important to prevent swelling (edema) and assure that the limb is properly shaped. Only after the limb has become stable in size will you be able to be measured for your final prosthesis. Both you and, if possible, a close family member will be instructed in the proper application and correct pressure of the various bandages. The initial swelling (edema) decrease rapidly, but edema usually lasts from 6 months to a year, so expect lots of changes in your limb and prosthetic socket.

When the residual limb has healed and is relatively stable in size and shape, you will be measured for a final prosthesis. As soon as your stump is healed, you will be instructed in stump wrapping. This is necessary to prevent swelling and properly shape the stump. The stump is shaped by wrapping an elastic bandage around the stump using just the correct amount of tension and pressure. The limb is usually rewrapped every 2 to 3 hours in order to allow the stump to be exposed to fresh air and to adjust the bandage's tension.

Preprosthetic Exercise Program

The object of an exercise program is to ensure that you have the best physical ability you are capable of in order to operate the prosthesis safely and to walk efficiently.

If you do not use a part of any limb for a month or so, it may change so much it may never be useful again. The muscles literally shrink to almost nothing while the tissues that connect the muscles to the bones (ligaments and tendons) will shrink to match the shortest length they can be. Thus, if you keep your knee bent for a month or so, the tissues will shrink up (called a contracture) and may never be able to stretch out again. You will have to learn how to avoid these kinds of problems and to strengthen your residual limb so that it can take on the task of using the prosthesis when you are ready.

Walking with a crutch will probably be your first "functional" activity. You may have to start walking between two parallel bars until your balance has improved enough for you to safely use crutches. Muscle strength and endurance are important. The exercise program will be developed especially for you depending on your current condition and problems as well as

your needs. Resistive exercises, pulley and mat exercises, push-ups, sit-ups, balancing, hopping independently on the unaffected limb, and crutch exercises are important to increase your confidence, strength, endurance, and, especially, safe control of the prosthesis.

Prosthetic Training

The Initial "Training" Artificial Limb

The artificial limb you will use to walk or to assist you to grasp items is called a prosthesis. Soon after amputation most people are fitted with a training or trial prosthesis. This is a very simple device to use so you can walk normally or carry out many of the functions of your hand. A typical pylon (training prosthesis) was shown in Figure 5. As soon as the residual limb is stable (measurements are unchanged for about 2 to 3 weeks), an expert in making and fitting prosthetic limbs (called a "prosthetist") will measure you and build a prosthesis to your exact needs and shape. Modern prostheses may be made from a variety of materials or combinations of materials. The best choice of materials for your prosthesis is determined by many factors. For someone who wants to engage in vigorous sports, material strength is an important consideration. For a frail individual, the weight of the materials might be a greater concern. Your prosthetist can help you decide on the materials most suitable for the construction of your prosthesis. The process begins by creating a plaster mold of your residual limb; then a plastic socket for the limb is created from the mold. This socket is then fastened temporarily to an adjustable temporary leg for beginning of training in walking. Adjustments may have to be made occasionally before the leg is finished.

Please note that you can not bear weight on the end of your residual limb! If you did, the bone at the bottom of the stump would crush the skin flap and destroy it. Your foot is probably eight to ten inches long by three or so wide. The bone at the bottom of the stump is probably less than an inch by an inch. The weight is simply too concentrated for the skin to be able to take it.

Steps and Timing

It take time to learn to use any kind of a prosthesis. You must be patient and learn each step in the process. Skimping on time and rushing now will slow down your eventual recovery and cause you considerably more pain than caused by the training itself. You can expect training to take anywhere

from 2 weeks to 2 months if you are in good physical condition. It will be proportionately longer the further your physical condition is from good.

The objectives of prosthetic training and care are to ensure that you achieve the skills needed for using your prosthesis in daily activities, that you develop an efficient gait (way of walking), and that the prosthesis provides the best possible function, comfort, and appearance. You will have to learn to care for your prosthetic device properly. You need to develop the balance, coordination, and motor skills to perform activities of daily living with your prosthesis. You have to learn how to put on the prosthesis correctly. You will be taught to develop correct habits in putting on and using the prosthesis so that it will always be second nature for you to do it right. You will learn how to put on and use special socks that go on over the end of your residual limb (stump socks). Some people have to add stump socks during the day in order to maintain total contact with the prosthesis. As your residual limb changes over time, stump socks may have to be added to maintain correct contact with the socket. Your body's weight is borne on different parts of the prosthesis depending on the type of amputation. In the below-the-knee prosthesis, the weight is borne on the large tendon below the knee-cap and the flares of the shin bone. In an above-knee prosthesis, weight is distributed evenly over the skin surface area and to a lesser extent on the buttock muscle.

Artificial Legs (Lower Extremity Prostheses)

A wide variety of artificial limbs are available. Which one is best for you depends on many factors including your overall health and physical condition, the condition of your stump, what type of activities you want to perform while using the limb, the type and level of amputation, and, most importantly, which works best for you! For example, an older person would need a safety knee and a much lighter prosthesis than a very active, younger person. Your stump and your prosthetic needs continue to change throughout your life, so you may occasionally have to make corresponding changes in your prosthesis. You may occasionally have to change sockets as your residual limb shrinks or as the socket wears out.

After the optimal set of parts comprising the prosthesis have been selected for your needs at your particular stage in recovery, your prosthesis has to be aligned especially for you. This means that it must be adjusted so it works as much like a real limb as possible. This takes a lot of tinkering and adjustment over a period of weeks or even months. Don't expect it to be perfect at the first attempt. Work with the prosthetist so you get your prosthesis working as well as possible.

Your artificial limb will not move as your leg did prior to amputation. This is especially evident for above-the-knee amputees because the lower limb swings like a pendulum. It develops momentum of its own, which must be controlled. You need to learn to do this. Several newer types of prosthesis have computer-controlled motions. They work by having motion sensors on your leg and back that provide information about your gait to the computer so it can adjust motion in the prosthesis. These work well but are new and expensive.

General Types of Prostheses

1. Endoskeletal with foam cover. The basic weight is through a socket onto a steel or strong plastic rod, which is covered with soft foam for cosmetic purposes.
2. Exoskeletal with hard plastic laminated socket. This looks like a plastic arm or leg. Many are so realistic that even close observation cannot distinguish between artificial and real in either looks or function. Color can be arranged to suit the individual. There are a variety of ways to attach the prosthesis to your residual limb. Which prosthesis is used depends largely on the type and level of amputation, the health of the residual limb, and the use to which you will put the prosthesis. For example, a prosthesis intended for tramping in heavy woods is different from one used around the house or office.

Attachment

Artificial limbs have to be attached in some way so they will not fall off or twist while in use and so they will provide enough stability for you to carry out the tasks at hand in safety and comfort. The common types of lower extremity prostheses and methods of attachments are detailed below. Figures 7 and 8 show typical artificial limbs for BK (below-the-knee) amputees. They are held on by various types of straps and wedges. Figure 9 shows a typical leg for a Syme's amputation, which is held in place by closing a window to complete a circle around the residual limb. Figure 10 shows prostheses for above-the-knee (AK) amputees. They are held on by a combination of straps. Figure 11 illustrates a very different method of holding on an artificial leg. This is the "suction" socket, which is held in place by contact between the stump and the prosthesis. This "air-tight" seal holds the limb on the same way a suction cup dart is held to a wall. This

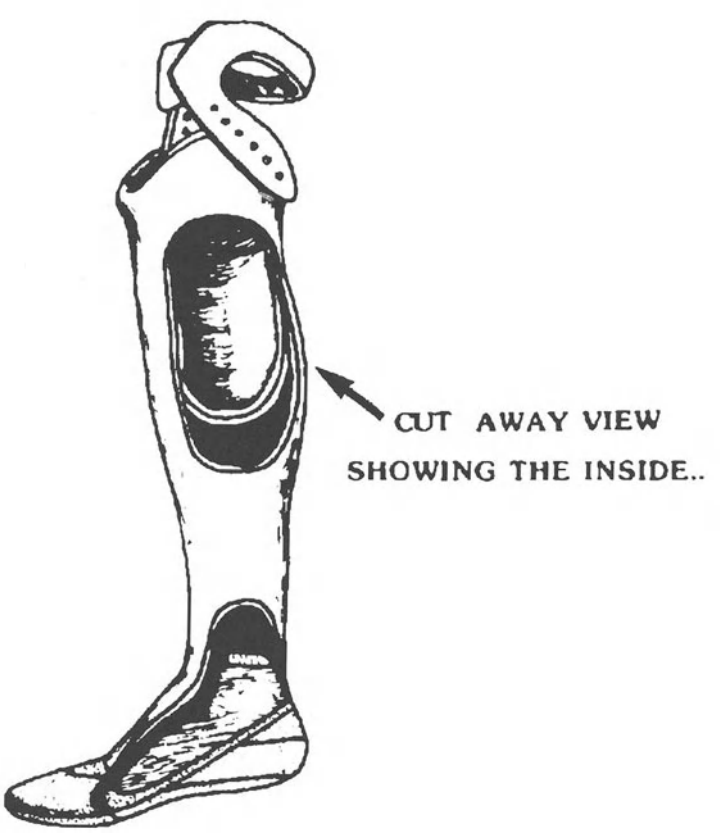

Figure 7. Artificial leg and foot with strap support for below-the-knee amputees. This leg has a strap that holds it just above the knee. The strap provides both stability and strong thigh support.

type of socket can be used only after the residual limb has been stable for a year or so. No socks are worn with this type of limb, so considerable adjustment is required.

As mentioned above, the socket is made to your specifications from a plaster mold. It is modified to avoid excessive contact with your particular tender areas. Computer-aided fitting of sockets is now coming into use. This has the advantages of easier modification to your needs and remembering your exact specifications, so molds of your leg do not have to be kept for manufacture of future sockets.

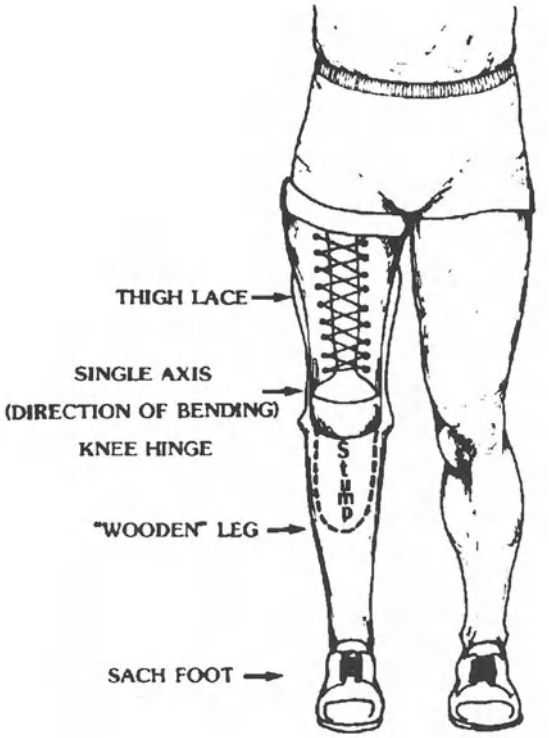

Figure 8. Artificial leg and foot for below-the-knee amputees with a "thigh lacer" for support and stability.

Types of Sockets (Which Your Stump Fits Into)

Sockets for Above-the-Knee Amputees

1. Suction socket: Strapless model discussed earlier that is held on with a vacuum.
2. Semi-suction: Similar to that above, but a little looser.
3. QTB: Weight is borne on the pelvis seat.

Sockets for Below-the-Knee Amputees

1. Traditional total-contact above-the-knee: Prosthesis discussed earlier (either solid or soft versions).
2. PTB: weight borne on the knee cap's tendon.
3. PTS: supracondylar suspension for below-the-knee amputations, has an above-the-knee wedge.

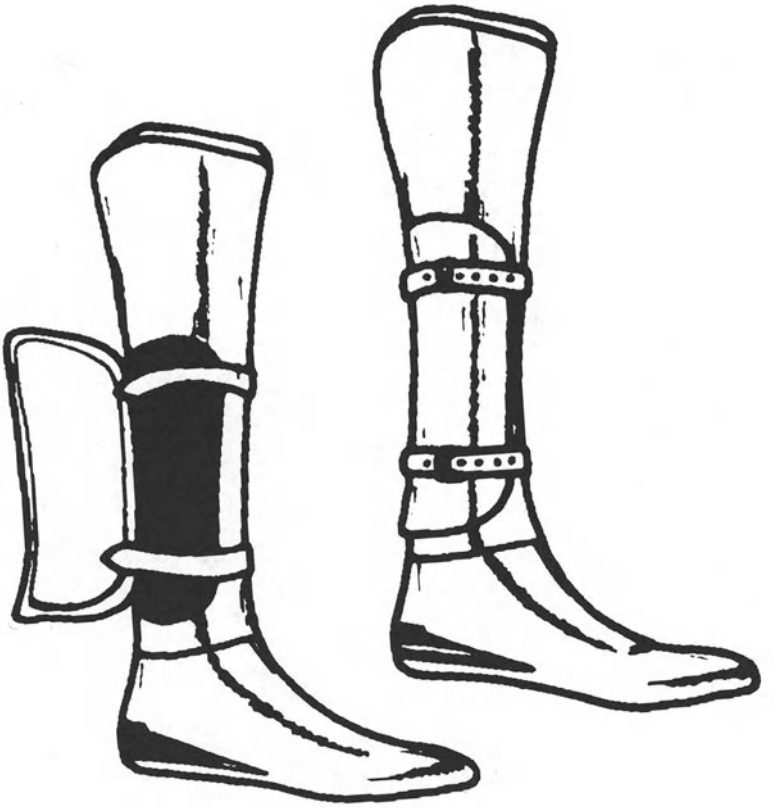

Figure 9. Artificial foot for Syme's "at-the-ankle" amputees. This leg has a hinged "window" that allows room for the ankle and heel to get inside the prosthesis. Once the residual limb (stump) is inside, the window is closed for support and stability during walking.

Sockets for Hip Disarticulation. For those with no actual residual limb because the limb was removed at the hip joint.

Sockets for Hemipelvectomy. When the entire limb and half of the hip bones are removed.

Types of Suspensions (Specific Ways to Attach the Limb to You)

Below-the-Knee

1. PTS supracondylar prosthesis suspended by supracondylar flares.
2. Wedge suspension.

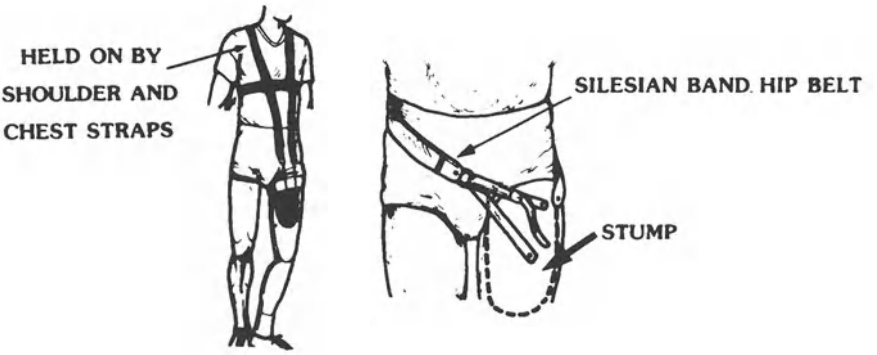

Figure 10. Strap methods for attaching an artificial limb for above-the-knee amputees. For some amputees, a shoulder and chest strap is needed to keep the leg in place. Others do well with just a hip belt.

3. PTB: Traditional plastic laminated hard socket worn with soft petite liner, kick strap, and pelvic band suspension.
4. Auxiliary straps and bands such as thigh lacers. Supracondylar straps
5. Thigh lacers.

Above-the-Knee. Weight is borne on the ischial tuberosity and suspended with silesian bands or pelvic bands with steel hinges.

Percutaneously (through the skin) Implanted Prosthetic Holders

The idea of these devices is that weight was meant to be borne by your bones, so a strong metal rod is implanted into the end of the bone that is at the bottom of your stump and clamped into place. It has several "arms" that go from the end of the rod protruding from the bone out through your skin to a jig that a regular artificial limb can be attached to. The system has the great advantage of avoiding all of the problems with sockets. Although they have been tried off and on since World War II, they are currently only experimental devices because of the high rate of infections getting into the bones along the rod "arms" that go through the skin. These infections are difficult or impossible to stop and can be deadly. Our laboratory and others are working on ways to solve this problem, but the studies have not been completed yet. Thus, it is unlikely that you will be offered such an attachment system at this time.

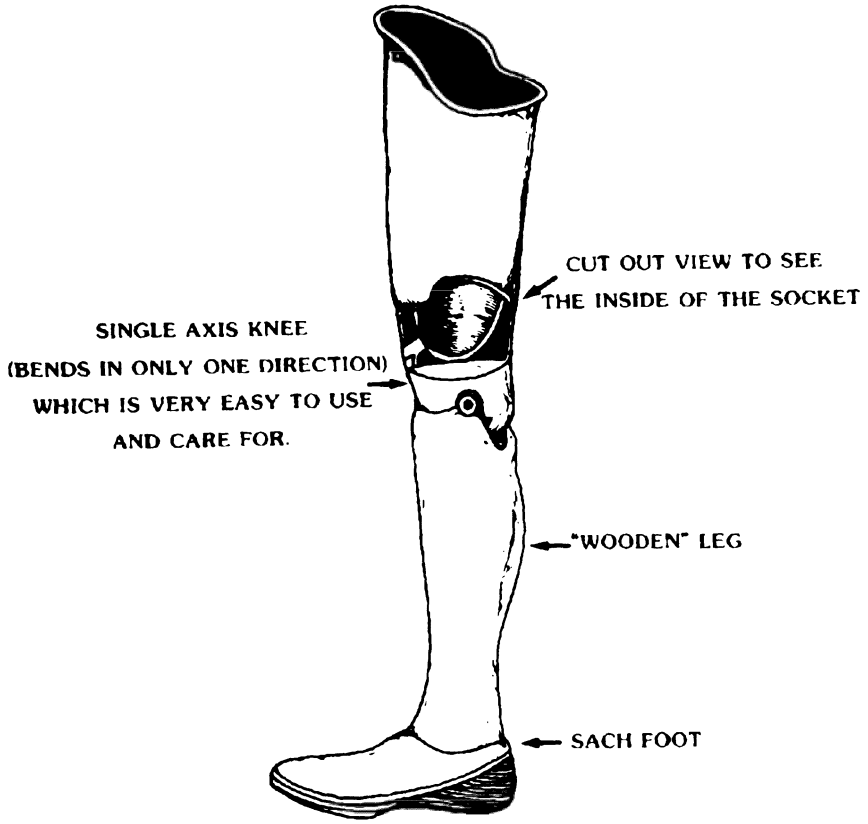

Figure 11. Suction socket method for attaching an artificial limb for above-the-knee amputees. This "suction socket" is held in place by the suction created between the residual limb (stump) and the prosthesis rather than by belts or straps.

Knees

The knees shown in Figure 11 are all "single axis." They can bend only one way—forward and back—rather than rotating in and out the way human knees do. This is a simpler system to learn to use but does make walking on uneven surfaces more difficult. There are many types of artificial knees. They range from those that take a lot of energy to use but give a great deal of stability (such as the hydraulic knee) to those that take little energy to use but are not as stable (such as the single-axis knee). You and your health care team will have to explore the alternatives as your reha-

bilitation progresses. You may go through several types before finding the best one for your own needs. Typical types of knees include:

1. Spring extension-assist: No stance control, lighter weight for older or weak people with little stamina.
2. Hydraulic unit: Swing-phase control to the heel rise. Accommodates differences in high speed to produce a more normal gait.
3. Constant friction: No moving parts and much more control demanded of the patient. Stance-phase control only with constant friction. Stays locked until heels rise. Doesn't buckle. Better for older people.

Feet

If you think about the movements your foot and ankle have to make while you are walking—especially if the surface is sloped or uneven—you will realize that the foot must be flexible to accept the changes in angle of the leg as you move through a step, must be able to adapt to a variety of uneven surfaces, and must provide support for the entire leg. Artificial feet are being improved all of the time. Figures 12 and 13 illustrate the basic principles of these feet. The Veterans Administration (VA) has recently developed the "Seattle" foot, which provides such natural support and movement, it is almost as good as the real one.

Different prosthetic feet are available. Each can take on basic shoe styles. A variety of special shoe styles such as elevated heels up to two and a half inches in height also are available. It is far easier and safer to walk in a relatively flat shoe. Typical types of feet are illustrated in Figures 12 and 13. They include:

1. SACH—solid ankle cushioned heel—an all wood foot shaped like a human foot with a cushioned heel, bumpers, toe-break, and single axis ankle joint. This is a very durable foot.
2. SAFE—stationary ankle flexible endoskeletal—more flexibility for walking on uneven ground. Needs occasional maintenance.
3. Four-way—Inversion/eversion as well as flexion. Moves almost like a human foot. However, the more moving parts, the more upkeep, wear and cost.

Learning to Move Using an Artificial Leg

Learning to walk again is a slow process that requires considerable time and patience. Problems vary with each patient depending on age,

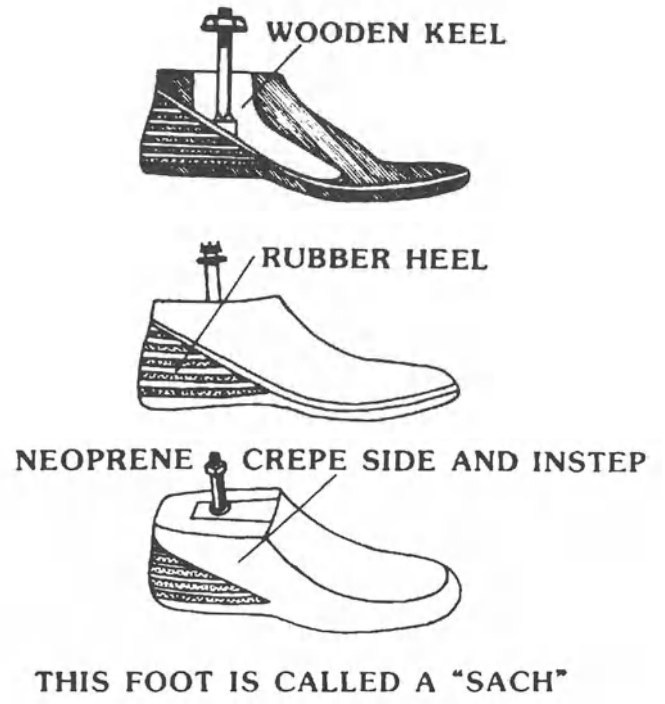

Figure 12. Details of a "SACH" foot. "SACH" is an acronym for solid-ankle/cushioned heel.

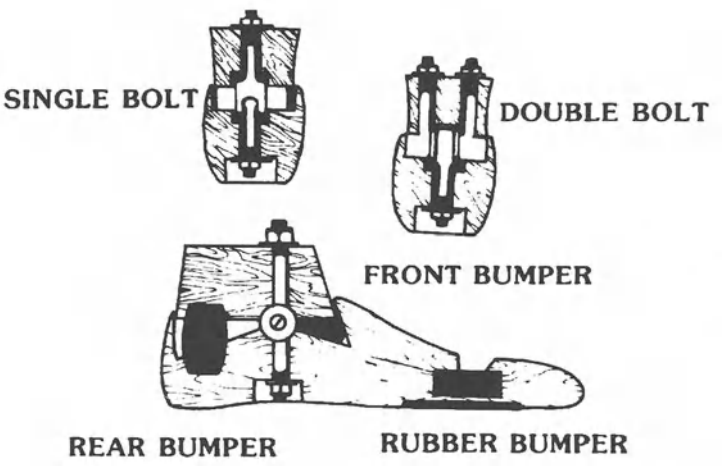

Figure 13. How an artificial foot replicates the motion of a real foot.

general health, and extent of amputation. Some patients can learn to walk with a few lessons, and others take between several weeks and months and continue to improve throughout the first year. You do not have to be hospitalized during this period and can usually be treated as an outpatient.

Below-the-knee (BK) amputees become better ambulators than others because the knee joint has been preserved. It requires approximately three to four times more energy for an above-the-knee amputee to walk than it does for a person with normal legs.

Your training for walking with a prosthesis will probably start on the "parallel bars." This is a pair of adjustable rails set slight more than shoulder width apart at about hip height. Most of your weight is supported by your arms while you learn to control and put weight on your prosthesis. Walking is only one of the movements you must master. In order to be independent, you will have to learn to rise from a chair, to shift your weight and balance, to go up and down stairs, ramps, and hills, and, of vital importance, how to fall safely and then get up again.

Use of Artificial Hands and Arms: Upper Extremity Prosthetic Care and Training

You will probably work with an occupational therapist (OT) to learn about prosthetic hygiene, how to put on and remove your prosthesis, parts of the prosthesis, routine maintenance, and use in activities of daily living including self-care, work, and leisure. You will learn how to clean the prosthesis, and how often to clean your residual limb. You will practice putting on and taking off the prosthesis with and without help (when possible). The type of prosthesis and your abilities will determine whether it is possible to do it alone.

Your ability to adjust to wearing the prosthesis for extended periods of time (called "tolerance") involves your attitude toward the prosthesis as well as your body's ability to bear the strain. You will probably begin by wearing it for half an hour, after which your stump will be inspected for signs of irritation. If none are noted, it will be put on again, and you will wear it for another half-hour. This may be repeated until you can wear it for 2 hours without irritation. If irritation does occur, the prosthesis must not be reapplied until the redness clears up. Do not be surprised if you have several one- or two-day pauses during your training. If you allow an irritation to get bad by ignoring it and keeping a "stiff upper lip," you may cause severe skin and muscle damage that can take weeks to months to heal.

Training beings with learning to control the prosthesis's movements and operations. The basic movements are frequently learned in the first session. As you develop skill in controlling your prosthesis, you become ready to begin learning to use the prosthesis for the activity you want to carry out. For example, to grasp a jar, hook "fingers" must be perpendicular to the table or vertical. Prosthesis awareness and skills are readily learned by using the prosthesis in activities that require two hands such as cutting meat and tying shoes. Necessary acts that have to be carried out frequently for a comfortable, normal life are called "activities of daily living." The particular activities chosen depend on your needs and interests. You will have to learn to recognize and deal with the limitations of your prosthesis. If you do not take the time to learn to work with the device and adapt to its limitations, you may decide not to use one at all, which would limit the range of activities you can carry out. This could unnecessarily make you a cripple. The type of prosthesis chosen and the training you get depend not only on the type of amputation and your physical abilities but also on the types of activities you need to carry out. A pros-

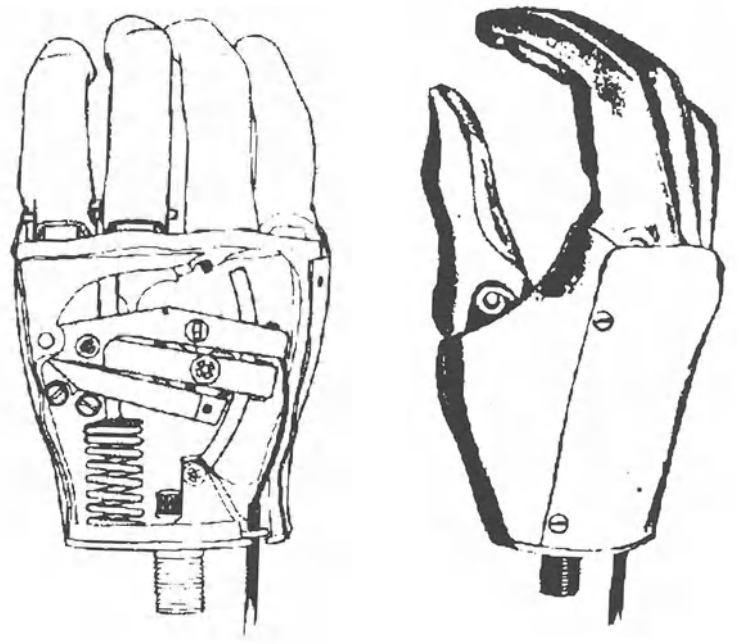

Figure 14. Example of a functional prosthetic hand. The figure is adapted from one appearing in *Limb Prosthetics*, 4th edition, 1979, published by Hanger, Inc. of St. Louis, MO, USA.

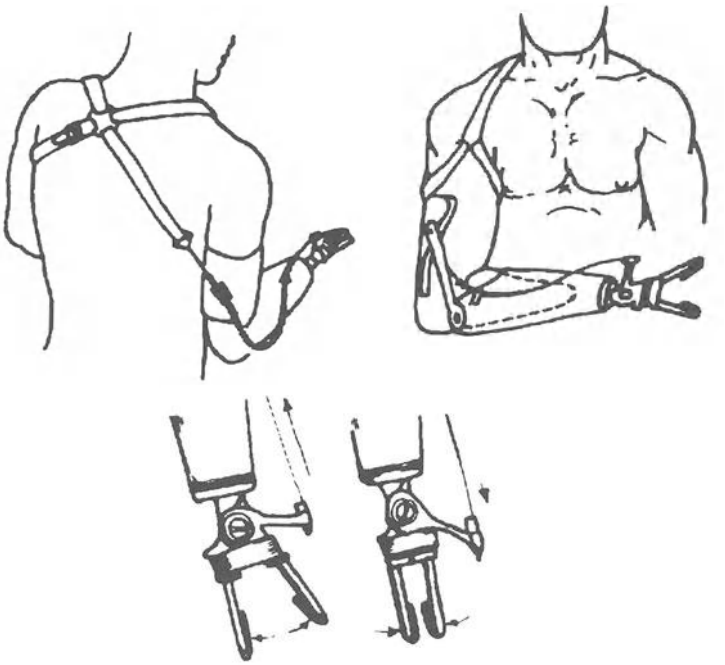

Figure 15. Attachment and use of a typical below-elbow prosthesis.

thesis worn just for looks (cosmetic appearance) is very different from one used to put parts together in a factory. Figure 14 shows a typical prosthetic hand. Figure 15 illustrates how a typical below-elbow (BE) artificial hand and arm is held on and controlled.

Some prostheses are partially powered by batteries. They are controlled by tensing various muscles in your residual limbs and shoulder, so they are called "myoelectric prostheses." They are more complex and expensive than most and have problems with reliability, performance of many tasks, weight, availability of parts and repair. Some of the very new prostheses are computer controlled. The computer helps regulate the amount and type of motion the arm makes so it matches the motion you wish to make better than one with a simple joint can. Some of the prostheses are capable of different grip strengths. They can signal you in various ways to let you know when you are gripping strongly enough to pick up a raw egg without dropping it but not so hard that it is crushed. They are more cosmetic, eliminate the need for harnessing and require less energy and movement to operate. There is still some question as to whether the electronically powered hand is more functional than the muscle-

powered one. Your treatment team will do a careful evaluation of your needs and abilities. Then they will recommend the type of prosthesis most likely to meet your needs. Each amputee has to decide based on individual experience.

Quite frankly, most women do not use functional upper extremity prostheses. They tend to stick with cosmetic ones largely because they don't like the looks of a pair of hooks sticking out the end of their sleeves. This is really unfortunate because the newer types of prostheses can be both very functional and relatively cosmetic. It is true that they don't yet look and act like a living hand, but they are coming closer. Restricting yourself to a cosmetic hand puts severe limits on the types of work and recreational activities you can do.

Pain in the Fully Health Residual Limb

"Stump" pain and phantom pain are very common even after the residual limb has been properly shaped, has healed properly, and has been properly used with a prosthesis for years. Pain in the residual limb is frequently caused by using the prosthesis too much, by skin problems related to the prosthesis, or by changes in the limb that come with age. As discussed earlier, your physician can usually help relieve almost all stump pain. See Chapters 3 and 4 for extensive discussions of mechanisms underlying and treatments for phantom and stump pain.

CHAPTER 7

Living with an Amputation

What Can't You Do?

You can do almost anything you want to regardless of the type of amputation you have if you are in good physical health and condition. Numerous amputees sky dive and do distance swimming, mountain climbing, hiking, fishing, etc. However, you are limited by the differences between a natural limb and prostheses. This is especially true of the amount of activity you can do without causing problems in the residual limb. There is a real limit to how much irritation your skin can take from the pressure, sweat, and twisting of the socket. So, do, but don't overdo. You have to learn your limits.

Planning for Your Return Home after the Amputation

Start planning before you have your amputation! Your ability to move independently may, at least temporarily, be quite different after your amputation than before it. You need to plan how you will get around your home, neighborhood, and work environments. This is probably the first time you and your family have faced this situation. You can get help from many professionals, especially social workers, disablement resettlement officers, and representatives of amputee groups, who know what is likely to happen and can give you guidance about what to expect and where to get help in changing your life style and living environment.

You have to plan for any changes needed in your home, including the doorway, bathroom, bedroom, and kitchen. Apparently minor activities such as getting out of a deep armchair and a bathtub suddenly become events. It is much easier to trip and fall when learning to use a prosthesis, so it is critically important to change or move anything you can trip over or slip on (such as highly waxed floors). Plan as soon as possible for changes in your occupation. If you will not be able to work as a result of the

amputation, get help as early as possible in looking into disability allowances, etc. This is not only to stabilize your economic situation but to help reduce your level of stress. You can't concentrate on rehabilitation if you are worried "sick" about your family's economic survival.

Keeping up Your Health to Keep Your Mobility and Independence

It takes slightly more energy to walk with some prostheses than with normal limbs. Older people tend to notice this more than relatively young folks. This increased energy expenditure, along with pain and irritation, can limit how far you can walk. Double amputees can expect considerable trouble with long hikes. If you find that you are really having problems, you may need a different type of prosthesis or to have your present one adjusted significantly. If you have difficulty getting onto trains and buses, you may be able to get a mobility allowance (frequently available from such sources as the U.S. Veterans Administration and the British government) to help you adapt a vehicle to your needs.

Because it is less convenient and sometimes more painful to move around and be normally active, many amputees tend to adopt a more sedentary life style than is typical of others of similar age. This is potentially dangerous for amputees because decreased activity leads to weakened muscles and decreased vascular flow in the residual limb. If the limb begins to waste away, numerous problems set in quickly. Of special importance is that the skin breaks down more easily, bruises and infections heal more slowly, the bones break more easily, and the prosthesis's fit changes more quickly and radically. The overweight that comes with obesity quickly adds extra strain on the skin–socket interface and changes the fit of the socket. The extra strain on the spine and joints accelerates the development of osteoarthritis and other problems. A regular exercise program is vital to continued good health and ability to use your prosthesis. You and your physiotherapist can work together to design a program that avoids putting extra strain on your residual limb while providing an interesting variety of activities. Promising yourself that you will regularly do a strenuous program of boring, repetitive physical exercise is likely to prove futile before long.

Probably the best way to keep fit is to identify leisure activities that interest you and do a variety of them so you don't get burned out on any one. Many activities do not require extensive use of the lower extremities and can still be done by almost anyone with normal upper extremities. These include archery, swimming, rowing, canoeing, sailing, riding, and

crafts such as woodworking. Almost all amputees can walk for varied distances over a variety of terrains without damaging themselves.

Getting the Help, Training, and Support You Need

The saying "no one is an island" goes for amputees a well as for everyone else. If you are in good health and condition, you need not be more dependent on others than you were before the amputation. If your health and condition have deteriorated because of age, disease, disuse, etc., you will need correspondingly more help.

However, when you first have your amputation, you are not likely to be aware of what techniques exist to make your life easier (such as tying a shoe lace with one hand or adapting an automobile) or what you can do to make your life as effective as possible and reduce your limitations. The next chapter lists the major organizations that can help you help yourself. It also lists some of the written material that can provide information and direction. Too many amputees suffer needlessly because they don't make the effort to find out what techniques and help are available. There is no need to make your life more difficult than it has to be.

Will Your Residual Limb Ever Be "Normal"?

Not entirely. The blood supply to the end of the limb will probably never grow back entirely. Blood carries all of the heat to your limbs. Because less blood is flowing through the end of the limb than is normal, most amputees find that the end of the stump is usually colder than the rest of the limb. When it is very cold, the limb is more easily affected than the rest of the body. Your stump will probably also always be more sensitive to touch, rubbing, etc. than other parts of your body. This means that it could be damaged more easily than you might expect.

Life-Long Changes in You and Your Prosthesis

Your residual limb will continue to change. It swells every day as you use it, and it gradually shrinks with age. It will change in overall configuration as you alter the amount and type of use to which you put your prosthesis. As you age, the way you move, especially your gait, changes naturally. All of these factors affect how well your prosthesis fits. Pros-

theses are machines. Just like any other machine, they get out of whack and break with time and use. They need to be kept up properly and tuned up. The newer devices have computers, muscle tension and motion sensors, computer-controlled joints, tiny motors, etc. You can expect them to give you and your prosthetist more problems and have more "down time" than relatively simple mechanical prostheses.

Between the changes in your body and those in the prosthesis, you can expect to have the prosthesis adjusted and repaired at apparently random intervals. A common problem is that people who have been very comfortable with a socket for years gradually become aware that it no longer fits properly or is becoming irritating and painful. They have the socket replaced with an identical one and are surprised when it doesn't work out. You will have to be remeasured every few years at most and can expect to keep changing various components of the prosthesis as the years pass.

Because your body changes over time, the way your prosthesis fits changes. This can result in changes of several inches in the length your prosthesis has to be so that your legs are the same length. The importance of having a prosthetic limb of the correct length can not be overstated. If you walk with a tilt for several years, you will cause undue strain on your back, pelvis, and your other leg. This can lead to far worse osteoarthritis in your hips and spine than you might otherwise experience. These are major, disabling problems that can be ameliorated but not fixed, so don't let them start.

Critical Final Points

You must take proper care of your residual limb and of your prosthesis! It is a real bother and impingement on your time to keep changing stump socks, cleaning and powering your stump, caring for your socket, etc. when nothing seems to be wrong. Unfortunately, it only takes a few days of slacking off for terrible sores to develop that can take months to heal.

You cannot overuse your prosthesis and get away with it. The price of overuse is crippling pain, sores, and disability that can last for months. You can easily become unable to use your prosthesis for months after a single "binge" of overuse.

You change with time, and things randomly go wrong with you and your prosthesis. This means that you must have an excellent, continuing working relationship with your health care team—especially your physician and prosthetist. You need to be able to tell them when something is

just starting to go wrong—not wait until you become disabled or need surgery to fix what would have been corrected with a simple adjustment.

You are ultimately responsible for how well you live with your amputation. There is simply no way to duck the responsibility. Others, especially your health care team and your family, can give guidance and encouragement, but, in the end, it's all up to you.

Chapter 8

Further Information

This brief introductory guide cannot supply all the details you will need to be fully informed about amputations and attendant problems. The best source of information about the amputation itself and your immediate rehabilitation is your health care team. After you leave the hospital, information is more difficult to come by. You may also feel alone and isolated in attempting to deal with your problems. Because you are not an expert on amputations, you may not know what is normal or what is important. You may feel hesitant to "bother" your physician very often with what may be minor problems. With rare exceptions, your physician will not be an amputee, and you may not know any other amputees, so you may have difficulty communicating your feelings. This further increases the feelings of isolation. You also may not know where to get information on ways to further you rehabilitation. It really helps if you can contact other amputees or organizations interested in working with amputees. Some literature is available that may be of help in furthering your rehabilitation and in letting you know what kinds of problems to expect. We have included most of the readily available publications that we know of.

Organizations

1. British Limbless Ex-Service Men's Association (BLESMA):
   Frankland Moore House, 185 High Road
   Chadwell Heath, Romford, Essex RM6 6NA, England
   Phone: 081-590 1124
2. National Amputation Foundation (NAF):
   73 Church Street
   Malverne, NY 11565, USA
   Phone: (516) 887 3600

3. American Amputee Foundation (AAF):
   P.O. Box 55218 Hillcrest Station
   Little Rock, AR 72225, USA
   Phone: (501) 666-2523
4. The Amputee Association of Northern Ireland:
   Enterprise House, Balloo Ave.
   Bangor BT19 7QT, Northern Ireland
   Phone: 0247 271525
5. US Veterans Administration
   Central Office
   810 Vermont Ave. N.W.
   Washington, DC 20420, USA
6. Paralyzed Veterans of America
   801 18th St. N.W.
   Washington, DC 20006, USA
   Phone: (202) 872-1300
7. Disabled American Veterans:
   807 Maine Ave. S. W.
   Washington, DC 20024, USA
   Phone: (202) 554-3501
8. National Association of the Physically Handicapped:
   76 Elm St.
   London, OH 43140, USA
   Phone: (614) 852-1664
9. National Information Center for the Handicapped (US):
   1201 16th St. N.W.
   Washington, DC 20036, USA
   Phone: (202) 833-1460
10. National Handicapped Sports and Recreation Association:
    4105 E. Florida Ave.
    Denver, CO 80222, USA
    Phone: (303) 757-3381
11. National Association for the Limbless Disabled:
    134 Martindale Rd.
    Hounslow, Middlesex, TW4 7HQ, England
    Phone: 01-572-5337
12. Disabled Living Foundation
    380 Harrow Road
    London W9 2HV, England
    Phone: 01-289-6111
13. War Amputees of Canada:
    (Helps both children and adults; need not be war related)

2277 Riverside Drive Suite 207
Ottawa, Ontario K1H 7X6, Canada
Phone: (613) 731-3821

14. Amputées de guerre Quebec:
606 Cathcart St., Suite 530
P.O. Box 11027, Station Downtown
Montreal, Quebec H3C 4W6, Canada
Phone: (514) 398-0759

Literature Intended to Help Amputees Help Themselves

Much of this literature is available from amputee organizations. Most large public libraries can borrow books from other libraries at no cost to you.

1. *Survivor*
Consumer Survival Kit, Owings Mills, MD 21117, USA
2. *Single Handed* (a guide for getting along with one hand), edited by B. Garee, 1978
Accent Special Publications, Cheever Publishing, Inc.,
P.O. Box 700, Bloomington, IL 61701, USA
3. *Amputee's Guide: Above-the-Knee* by A. Alexander, 1978
Medic Publishing Co.,
P. O. Box 0; Issawuah, WA 98027, USA
4. *Amputee's Guide: Below-the-Knee* by A. Alexander, 1978
Medic Publishing Co.,
P.O. Box 0; Issawuah, WA 98027, USA
5. *A Manual for Below-Knee Amputees* by A. Muilenburg and A. Wilson
P.O. Box 8313, Houston, TX 77004, USA
6. *A Manual for Above-Knee Amputees* by A. Muilenburg and A. Wilson
P.O. Box 8313, Houston, TX 77004, USA
7. *How to Get Behind the Wheel: Information for Amputees Wishing to Drive a Car*
UK Forum of Driving Assessment Centres
Banstead Mobility Centre, Damson Way, Queen Mary's Ave., Carshalton, Surrey SM5 4NR, England
Phone: 081 770 1151
8. *Guide for the Disabled*
Booklet from the Automobile Association (British) covering ho-

tels, guesthouses, and farm houses catering to the needs of the disabled driver
5 New Coventry St., London, W1V 8HT, England
Phone: 01-930-2462.

9. *Physical Fitness: Sports and Recreation for Those with Lower Limb Amputation*, by Bernice Kegel
Journal of Rehabilitation Research and Development Clinical Supplement 1, 1985
Office of Technology Transfer, Veterans Administration Medical Center, 50 Irving St., N.W., Washington, DC 20422, USA
(Excellent source of information for activities and groups)
10. *Which Benefit* (British)
Booklet from the DHSS available from the Social Work Department covering all current DHSS benefits available
11. *Disability Rights Handbook*
Disability Alliance, 25 Denmark St., London WC2H 8NJ, England
Covers many aspects of disability.
12. Various publications from the US National Information Center (funded by the US Government)
308 Mullen Library
The Catholic University of America
Washington, DC 20064, USA
13. *On the Road to Recovery—General Information for Patients with Lower Limb Amputations*
by J. Dayan and E. Moore, 1981
Burke Rehabilitation Center
Available through the National Amputation Foundation
14. *Strong at the Broken Places* by Max Cleland, 1980
Berkley Books, New York, USA
15. *The One Hander's Book: A Basic Guide to Activities of Daily Living* by Veronica Washam, 1973
John Day Company, New York, USA
16. *Dictionary of Information Resources for the Handicapped* compiled by the staff of Ready Reference Press, 1980
Ready Reference, Santa Monica, CA, USA
17. *Limb Prosthetics*
J. G.Hanger Corp., 1979
Albany, GA 31701, USA
18. *Pocket Guide to Federal Help for the Disabled Person* (American)
Office of Information and Resources for the Handicapped, U.S. Dept. of Health, Education and Welfare, Washington, DC 20201, USA

19. *Care and Use Guide for the Below-Knee Amputee*
American Academy of Orthotists, 717 Pendleton Street, Alexandria, VA 22314, USA
Phone: (703) 836-7118
20. *Hygienic Problems of the Amputee*
American Orthotics and Prosthetics Association, 719 Pendleton Street, Alexandria, VA 22314, USA
21. *Information Booklet for Patients and Relatives Regarding Amputation*
Camberwell Health Authority, Dulwich Hospital, East Dulwich Grove, London, SE22, England

General Literature of Interest

1. *When Bad Things Happen to Good People* by Harold S. Kushner, 1981 Schocken Books, New York, NY, USA

Literature in the Scientific Press Intended Mostly for Scientists and Health Care Providers Rather than Being Directed toward Amputee Patients

Much of this information may be too technical for the average reader to understand completely but may be of interest for finding out about specific areas of interest.

1. *Driving after Amputation: Information for Professionals*; available through the UK Forum of Driving Assessment Centers, Banstead Mobility Centre, Damson Way, Queen Mary's Ave, Carshalton, Surrey SM5 4NR, England;
phone: 081 770 1151.
2. *Amputation Surgery and Lower Limb Prosthetics*, edited by G. Murdoch and R. Donovan, 1988, Blackwell Scientific Publishers, London, England, 1988.
3. Phantom limb and stump pain, by R. Sherman; chapter in R. Portenoy, ed. *Neurologic Clinics of North America* 7(2), 249–264, W. B. Saunders Co., Philadelphia, PA, 1989.
4. Phantom limb pain: Mechanisms, incidence, and treatment, by R. Sherman and J. Arena, *Critical Reviews in Physical and Rehabilitation Medicine* 4, 1–26, 1992.

5. Phantom limb pain: Mechanism based management, by R. Sherman, *Clinics in Podiatric Medicine and Surgery: Pain Management* 11, 85–106, 1994.
6. *The Psychological Rehabilitation of the Amputee*, by L. Friedman, Charles C. Thomas, Springfield, IL, 1978.
7. *Occupational Therapy for Physical Disabilities* by K. Trombley and A. Scott
Williams & Wilkins, Baltimore, MD, year.
8. *Report to the Veterans' Administration Department of Medicine and Surgery on Service-Connected Traumatic Limb Amputations and Subsequent Mortality* by Z. Hrubeck and R. Ryer *Bulletin of Prosthetic Research, 16*, 29–53, 1979.
9. *The Challenge of Pain* by R. Melzack and P. Wall, Basic Books, New York, NY, 1983.
10. Aerobic training exercises for individuals who had amputation of the lower limb, by K. Pitetti, P. Snell, J. Stray-Gundersen & F. Gottschalk, *Journal of Bone and Joint Surgery* 69A, 914–921, 1987.
11. Reactions to loss of limb: Physiological and psychological effects by W. Haber, *Annals of the New York Academy of Science*, 74, 14–24, 1958.
12. Better health for the amputee by H. Pearson, available through BLESMA (address in preceding section).
13. *Lower Extremity Amputation: A Guide to Physical Therapy Management*, by L. Karacoloff, Aspen Systems, Rockville, MD, 1985.

Chapter 9

Definition of Terms

Abrasion: Rubbing off the skin leaving a "raw" area
AE: Above-the-elbow amputation
AK: Above-the-knee amputation
Alignment: Relative position of socket to the heel/foot of the prosthesis
  Dynamic alignment: Position of the socket stump during motion
  Static alignment: Initial position of socket to stump
Aligment apparatus: Adjust the prosthesis so that the gait is as normal as possible
Ambulation: Walking
Amputation: Surgical removal
Anterior: Toward the front
Axilla: The depression in the armpit
BE: Below-the-elbow amputation
BK: Below-the-knee amputation
Cadence: Rhythm of walking
Check socket: A test socket to evaluate the inital fitting of the socket; the material is usually clear to allow visual inspection for problem areas
Chronic pain: Pain that lasts for at least 6 months
Condyle: A rounded bump at the end of a bone
Contracture: Tightening of muscles, tendons, and ligaments around a joint, causing decreased motion
Delayed primary closure (DPC): When the amputation site is closed 3–5 days after surgery
Dependent: Hanging down (not usually used to mean needing others)
Distal: A direction; to the end part of the limb relatively away from the trunk of the body (also see "proximal")
Edema: Swelling (usually of the residual limb)
Elastic wrap: Elasticized bandage used to prevent swelling and encourage shrinkage of the residual limb
Endoskeletal: Soft outer finish with an interior support
Exoskeletal: Hard outer support and finish

Extension: Unbending or stretching
Femur: The thigh bone
Fibula: Smaller of the two bones in the lower leg
Flexion: Bending
Flexion contracture: Inability to extend through the normal range of motion
Gait: Speed, rhythm, and style of walking
Inferior: Below or directed downward
IV: Intravenous; usually refers to a thin plastic line run from a bag into a vein through a needle that is kept in the skin for up to several days at a time; blood can be sampled through this tube and antibiotics, and other medications can be run in through it
Lateral: Away from the midline of the body
Mature: Usually refers to the residual limb that has stabilized in volume and shape (usually within 1 to 2 years after amputation)
Medial: Toward the midline of the body
Modular limb: A type of artificial limb based on a central pillar (pylon) that normally contains at least one joint such as a knee; it is surrounded by plastic foam to imitate the contours of a normal limb
Occupational therapist: An expert in upper extremity rehabilitation.
Oedema: Swelling (of the tissues with fluid) (British spelling; see "edema")
PAM: A very temporary artificial limb used with someone who has just had an amputation so he or she can practice standing and some walking; it is held to your stump by an inflatable sleeve and contains a rod (pylon) that acts as a leg and foot.
Patella: Kneecap
Phantom limb pain: Pain that seems to come from the portion of the limb that was removed; these are painful phantom sensations
Phantom sensations: The normal "ghost" image and feelings that seem to come from the part of the limb which was removed
Physiotherapist: A physical therapist; the therapist who gives you exercise and trains you in how to walk properly
Pistoning: Stump slipping up and down in the prosthesis
Ply: Thickness of stump stocking material
Posterior: Behind or toward the rear
Prosthesis: Artificial limb
Prosthetist: Person who constructs and fits artificial limbs
Proximal: A direction; part of the limb close to the trunk of the body
PTB: Patella tendon bearing—a type of below-knee prosthesis; takes your weight on the relatively insensitive area just below the knee
PTS: Patella tendon supracondylar—a type of below-knee prosthesis suspended by femoral condyles
Pylon: Metal shaft inside the prosthesis; also temporary, simple prosthesis

Residual limb: The part of the limb remaining after the amputation (commonly called the stump)
Revision: To surgically do the stump over again, usually results in a shorter length
SACH foot: Solid-ankle/cushion-heel—type of artificial foot in which a foam heel imitates normal ankle movements
SAFE foot: Solid ankle flexible (endoskeletal)—type of artificial foot
Shrinker: An elasticized prosthetic sock used to prevent swelling and to encourage shrinkage of the residual limb
Shrinking: Usually refers to swelling going down in the residual limb rather than decreased amount of muscle
Skin breakdown: Any bleeding or disruption of the normal skin surface (e.g., blisters and sores)
Slip socket: Protects a tender stump against chafing
Socket: The part of the prosthesis that fits around the residual limb
Soft insert/liner: Cup-shaped form that fits inside the permanent BK prosthesis
Soft socket: Soft lining built into a socket to provide cushioning or permit muscle function
Stump: The residual limb or the end of the residual limb
Suction socket: A way of holding on an artificial limb using air pressure rather than straps
Superior: Above or upwardly directed
Supracondylar: Above the condyles
Suspension: How the artificial limb is held on
Syme's amputation: A type of amputation at the ankle
Tibia: Larger of the two bones in the lower leg
Tubercle: A small protuberance on a bone, usually forming an attachment point of a muscle
Volume changes: Swelling or shrinking of the residual limb
Wedge: Triangular insert that helps to hold some BK prostheses in place
Weight-bearing area: An area of the residual limb able to tolerate pressure and stabilizing forces

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Much of the text of the booklet was originally written by Dr. Sherman for a 1985 booklet entitled *What to Expect When You Lose a Limb: A Guide for Patients Expecting or Having Recently Had an Amputation*. It is conceptually the first edition of the current booklet. It was widely distributed through the U.S. Government's Printing Office and various amputee organizations but was never published. We are very grateful to Jeffrey Ernst, PhD, Betty Dodd, RPT, Saundra Turner, RN, and Roger Brown, OT, for their contributions to the first edition. Many of their ideas are contained in this booklet.

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References

Abt, L.(1954). Psychological adjustment of the amputee. In P. Klopsteg & P. Wilson (Eds.), *Human limbs and their substitutes* (pp. 139–158). New York: McGraw–Hill.

Ackerly, W., Lhamon, W., & Fitts, W. T. (1955). Phantom breast. *Journal of Nervous and Mental Disease, 121,* 177–178.

Adams, R. D., & Victor, M. (Eds.). (1989). *Principles of neurology* (pp. 151 and 155). New York: McGraw–Hill.

Ahmad, S. (1984). Phantom limb pain. *Southern Medical Journal, 77,* 804.

Albe-Fessard, D., & Lombard, M. C. (1983). Use of an animal model to evaluate the origin of and the protection against deafferentation pain. *Advances in Pain Research, 5,* 691–700.

Alexander, F. (1950). *Psychosomatic medicine.* New York: Norton.

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

Amir, R., & Devor, M. (1995) Mutual interaction among sensory cells in dorsal root ganglia. *Encephalography and Clinical Neurophysiology, 95,* 80–81.

Anani, A., & Körner, L. (1979). Discrimination of phantom hand sensations elicited by afferent electrical nerve stimulation in below-elbow amputees. *Medical Progress in Technology, 6,* 131–135.

Appenzeller, O., & Bicknell, J. M. (1969). Effects of nervous system lesions on phantom experience in amputees. *Neurology, 19,* 141–146.

Arena, J., Blanchard, E., & Andrasik. F. (1984). The role of affect in the etiology of chronic headache. *Journal of Psychosomatic Research, 28,* 79–86.

Arena, J., & Sherman, R. (in press). A comparison of the MMPIs of amputees with chronic phantom limb pain and age–sex matched pain free intact controls.

Arena, J., Sherman, R., & Bruno, G. (1989). The relationship between humidity level, temperature, and phantom limb pain: Preliminary analysis. *Biofeedback and Self-Regulation, 14(2),* 128.

Arena, J., Sherman, R., & Bruno, G. (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.

Arena, J., Sherman, R., Bruno, G., & Smith, J. (1988). The relationship between situational stress and phantom limb pain: Preliminary analysis. *Biofeedback and Self-Regulation, 13(1),* 55.

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

Arner, S., & Meyerson, B. A. (1988). Lack of analgesic effect of opioids on neuropathic and idiopathic forms of pain. *Pain, 33,* 11–23.

Ax, A. F. (1983). The physiological differentiation between fear and anger in humans. *Psychosomatic Medicine, 15,* 433–442.

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.

Baik-Han, E. J., Kim, K. J., & Chung, J. M. (1990). Prolonged ongoing discharges of sensory nerves as recorded in isolated nerves in the rat. *Jounal of Neuroscience Research, 27,* 219–227.

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

Baron, R., & Maier, C. (1995). Phantom limb pain: Are cutaneous nociceptors and spinothalamic neurons involved in the signaling and maintenance of spontaneous and touch-evoked pain? A case report. *Pain, 60,* 223–228.

Bartusch, S. L., Sanders, B. J., D'Alessio, J. G., & Jernigan, J. R. (in press). Clonazepam for the treatment of phantom limb pain. *Clinical Journal of Pain.*

Basbaum, A. I., Gautron, M., Jazat, F., Mayes, M., & Guilbaud, G. (1991). The spectrum of fiber loss in a model of neuropathic pain in the rat: an electron microscopic study. *Pain, 47,* 359–367.

Bates, R. E., & Stewart, C. M. (1991). Atypical odontalgia: Phantom tooth pain. *Oral Surgery, Oral Medicine, Oral Pathology, 72,* 479–483.

Bennett, G. J., Kajander, K. C., Sahara, Y., Iadorala, M. J., & Sugimoto, T. (1989). Neurochemical and anatomical changes in the dorsal horn of rats with an experimental painful peripheral neuropathy. In F. Cervero, G. J. Bennett, & S. M. Headley (Eds.), *Processing of sensory information in the superficial dorsal horn of the spinal cord* (pp. 463–472). New York: Plenum Press.

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.

Bini, G., Hagbarth, K. E., Hynninen, P., & Wallin, G. (1980). Thermoregulatory and rhythm-generating mechanisms governing the sudomotor and vasoconstrictor outflow in human cutaneous nerves. *Journal of Physiology (London), 306,* 537–552.

Blanchard, E. B., Andrasik, F., & Arena, J. (1984). Personality and chronic headache. In B. Maher & W. Maher (Eds.), *Progress in experimental personality research* (vol. 13, pp. 303–364). New York: Academic Press.

Blood, A. M. (1956). Psychotherapy of phantom limb pain in two patients. *Psychiatry Quarterly, 30,* 114–122.

Blumberg, H., & Jänig, W. (1981). Neurophysiological analysis of efferent sympathetic and afferent fibers in skin nerves with experimentally produced neuromata. In J. Siegfried and M. Zimmermann (Eds.), *Phantom and stump pain* (pp. 15–31). New York: Springer-Verlag.

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

Bornstein, B. (1949). Sur le phénomène du membre fantôme. *Encéphale (Paris), 38,* 32–46.

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

Botterell, E., Callaghan, J., & Jousse, A. (1954). Pain in paraplegia. *Proceedings of the Royal Society of Medicine, 47,* 280–288.

Boyle, M., Tebbe, C. K., Mindell, E. R., & Mettlin, C. J. (1982). Adolescent adjustment to amputation. *Medical and Pediatric Oncology, 10,* 301–312.

Bradley, K. C. (1955). *The sequelae of amputation.* Canberra: Trustees of the Services Canteens Trust Fund.

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

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

Bressler, B., Cohen, S. I., & Magnussen, S. (1955b). The problem of phantom breast and phantom pain. *Journal of Nervous and Mental Disease, 123*, 181–187.

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

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

Brooke, R. I. (1980). Atypical odontalgia. *Oral Surgery, Oral Medicine, Oral Pathology, 49*, 196–199.

Browder, J. G., & Gallagher, J. P. (1948). Dorsal cordotomy for painful phantom limbs. *Annals of Surgery, 128*, 128.

Buijk, C. (1988). Use and usefulness of low limb prostheses. *International Journal of Rehabilitation Research, 11*, 361–367.

Bullitt, E. (1991). Abnormal anatomy of deafferentation: Regeneration and sprouting within the central nervous system. In B. S. Nashold, Jr. & J. Ovelman-Levitt (Eds.), *Advances in pain research and therapy* (pp. 71–80). New York: Raven Press.

Burchiel, K. J. (1984). Spontaneous impulse generation in normal and denervated dorsal root ganglia: Sensitivity to alpha-adrenergic stimulation and hypoxia. *Experimental Neurology, 85*, 257.

Burke, D. & Woodward, J. (1976). Pain and phantom sensation in spinal paralysis. *Handbook of Clinical Neurology, 26*, 489–499.

Campbell, A. W. (1905). *Histological studies on the localization of cerebral function*. (p. 7). Cambridge: Cambridge University Press.

Campbell, J. (1987). Painful sequelae of nerve injury. *Pain, 34*, 334.

Campbell, J. N., Meyer, R. A., Davis, K. D., & Raja, S. N. (1992). Sympathetically maintained pain: A unifying hypothesis. In W. D. Willis (Ed.), *Hyperalgesia and allodynia*. New York: Raven Press, 141–149.

Campbell, J. N., Meyer, R. A., & Raja, S. N. (1992). Is nociceptor activation by alpha-1 adrenergic receptors the culprit in sympathetically maintained pain (SMP)? *American Pain Society Journal, 1*, 3–11.

Campbell, J. N., Raja, S. N., & Meyer, R. A. (1993). Pain and the sympathetic nervous system: Connecting the loop. In L. Vecchiet, D. Albe-Fessard, U. Lindblom, & M. A. Giamberadino (Eds.), *New trends in referred pain and hyperalgesia* (pp. 99–107). Amsterdam: Elsevier.

Campbell, J. N., Raja, S. N., Meyer, R. A., & MacKinnon, S. E. (1988). Myelinated afferents signal the hyperalgesia associated with nerve injury. *Pain, 32*, 89–94.

Campbell, J. N., Raja, S. N., Selig, D. K., Belzberg, A. J., & Meyer, R. A. (1994). Diagnosis and management of sympathetically maintained pain. In H. L. Fields & J. C. Liebeskind (Eds.), *Pharmacological approaches to the treatment of chronic pain: New concepts and critical issues* (pp. 85–97). Seattle: IASP Press.

Cannon, W. B. (1920). *Bodily changes in pain, hunger, fear and rage* (2nd ed.). New York: Appleton.

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

Chabal, C., Jacobson, L., Russell, L. C., & Burchiel, K. J. (1992). Pain responses to perineuromal injection of normal saline, epinephrine, and lidocaine in humans. *Pain, 49*, 9–12.

Charcot, J. M. (1892). *Leçons du mardi à la Salpêtière*, 2nd edition. (Vol. 1, pp. 344–355). Paris: Publications du Progrès Médicale.

Christensen, K., Blichert-Toft, M., Giersing, U., Richardt, C., & Beckmann, J. (1982). Phantom breast syndrome in young women after mastectomy for breast cancer. *Acta Chirurgica Scientifica, 148,* 351–354.

Christopher, R. P., & Koepke, G. H. (1963). Peripheral nerve entrapment as a cause of phantom sensation and stump pain in lower extremity amputees. *Archives of Physical Medicine and Rehabilitation, 44,* 631–634.

Clark, S. A., Allard, T., Jenkins, W. M., & Merzenich, M. M. (1988). Syndactyly results in the emergence of double digit receptive fields in somatosensory cortex in adult owl monkeys. *Nature, 332,* 444–445.

Coderre, T. J., Abbott, F. V., & Melzack, R. (1984). Effects of peripheral antisympathetic treatments in the tail-flick, formalin and autotomy tests. *Pain, 18,* 13–23.

Coderre, T. J., Grimes, R. W., & Melzack, R. (1986a). Autotomy after nerve sections in the rat is influenced by tonic descending inhibition from locus coeruleus. *Neuroscience Letters, 67,* 82–86.

Coderre, T. J., Grimes, R. W., & Melzack, R. (1986b). Deafferentation and chronic pain in animals: An evaluation of evidence suggesting autotomy is related to pain. *Pain, 26,* 61–84.

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.

Coderre, T. J., & Melzack, R. (1986). Procedures which increase acute pain sensitivity also increase autotomy. *Experimental Neurology, 92,* 713–722.

Coderre, T. J., & Melzack, R. (1987). Cutaneous hyperalgesia: Contributions of the peripheral and central nervous systems to the increase in pain sensitivity after injury. *Brain Research, 404,* 95–106.

Colado, M. I., Del Rio, J., & Peralta, E. (1994). Neonatal guanethidine sympathectomy suppresses autotomy and prevents changes in spinal and supra spinal monoamine levels induced by peripheral deafferentation in rats. *Pain, 56,* 3–8.

Conine, T., Hershler, C., Alexander, S., & Crisp, R. (1993). The efficacy of farabloc in the treatment of phantom limb pain. *Canadian Journal of Rehabilitation, 6,* 151–161.

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

Critchley, M. (1955). Quelques observations relatives a la notion de la conscience du moi corporel. *Encephale, 44,* 501–535.

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

Crone-Munzebrock, A. (1950). Phantomgefuhl und phantomschmerz nach mammaamputation. *Langenbecks Archiv für Klin Chir 266,* 569–575.

Cronholm, B. (1951). Phantom limbs in amputees. *Acta Psychiatrica et Neurologica Scandinavica, 72,* 1–310.

Dahl, J. B. (1994). Neuronal plasticity and pre-emptive analgesia: Implications for the management of postoperative pain. *Danish Medical Bulletin, 41,* 434–442.

Daniel, W. (1983). *Biostatistics: A foundation for analysis in the health sciences* (3rd edition). New York: John Wiley & Sons.

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.

Davar, G., & Maciewicz, R. J. (1989). Deafferentation pain syndromes. *Neurologic Clinics, 7,* 289–304.

Davis, K. D., Tasker, R. R., Kiss, Z. H. T., Hutchinson, W. D., & Dostrovsky, J. O. (1995). Visceral pain evoked by thalamic microstimulation in humans. *NeuroReport, 6,* 369–374.

Davis, L., & Martin, J. (1947). Studies upon spinal cord injuries. *Journal of Neurosurgery, 4,* 483–491.

Davis, R. (1993a). Successful treatment for phantom pain. *Orthopedics, 16*, 691–695.

Davis, R. (1993b). Phantom sensation, phantom pain, and stump pain. *Archives of Physical Medicine and Rehabilitation, 74*, 79–91.

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.

DeMarinis, M., Fraioli, B., Esposito, V., Gagliardi, F. M., & Agnoli, A. (1992). Changes in vasomotion and nociception in trigeminal deafferented humans. In F. Sicuteri (Ed.), *Advances in pain research and therapy* (Vol. 20, pp. 197–201). New York: Raven Press.

Dennis, S. G., & Melzack, R. (1979). Self-mutilation after dorsal rhizotomy in rats: Effects of prior pain and pattern of root lesions. *Experimental Neurology, 65*, 412–421.

Devor, M. (1983). Nerve pathophysiology and mechanisms of pain in causalgia. *Journal of the Autonomic Nervous System, 7*, 371–384.

Devor, M. (1988). Central changes mediating neuropathic pain. In R. Dubner, G. F. Gebhart, & M. R. Bond (Eds.), *Pain research and clinical management* (Vol. 3, pp. 114–128). Amsterdam: Elsevier.

Devor, M. (1991). Sensory basis of autotomy in rats. *Pain, 45*, 109–110.

Devor, M. (1994). The pathophysiology of damaged peripheral nerves. In P. D. Wall & R. Melzack (Eds.), *Textbook of pain* (3rd ed., pp. 79–100). Edinburgh: Churchill Livingstone.

Devor, M. (1996). Pain mechanisms. *The Neuroscientist* (in press).

Devor, M., Basbaum, A. I., Bennett, G. J., Blumberg, H., Campbell, J. N., Dembowsky, K. P., Guilbaud, G., Janig, W., Koltzenburg, M., Levine, J. D., Otten, U. H., & Portenoy R. K. (1991). Group report: Mechanisms of neuropathic pain following peripheral injury. In A. I. Basbaum & J. M. Besson (Eds.), *Towards a new pharmacotherapy of pain* (pp. 417–440). New York: John Wiley & Sons.

Devor, M., & Bernstein, J. J. (1982). Abnormal impulse generation in neuromas: Electrophysiology and ultrastructure. In J. Ochoa & W. Culp (Eds.), *Abnormal nerves and muscles and impulse generators* (pp. 363–380). Oxford: Oxford University Press.

Devor, M., Govrin-Lippmann, R., & Raber, P. (1985). Corticosteroids suppress ectopic neural discharge originating in experimental neuromas. *Pain, 22*, 127–137.

Devor, M., & Jänig, W. (1981). Activation of myelinated afferents ending in a neuroma by stimulation of the sympathetic supply in the rat. *Neuroscience Letters, 24*, 43–47.

Devor, M., Jänig, W., & Michaelis, M. (1994). Modulation of activity in dorsal root ganglion neurons by sympathetic activation in nerve-injured rats. *Journal of Neurophysiology, 71*, 38–47.

Devor, M., Lomazov, P., & Matzner, O. (1994). Na^+ channel accumulation in injured axons as a substrate for neuropathic pain. In J. Boivie, P. Hansson, & U. Lindblom. (Eds.), *Touch, temperature and pain in health and disease: Wenner-Gren Center Foundation symposia* (pp. 207–223). Seattle: IASP Press.

Devor, M., & Raber, P. (1990). Heritability of symptoms in an experimental model of neuropathic pain. *Pain, 42*, 51–67.

Devor, M., & Wall, P. D. (1981). Plasticity in the spinal cord sensory map following peripheral nerve injury in rats. *Journal of Neuroscience, 1*, 679–684.

Devor, M., & Wall, P. D. (1990). Cross excitation among dorsal root ganglion neurons in nerve injured and intact rats. *Journal of Neurophysiology, 64*, 1733–1746.

Devor, M., White, D. M., Goetzl, E. J., & Levine, J. D. (1992). Eicosanoids, but not tachykinins, excite C-fiber endings in rat sciatic nerve-end neuromas. *NeuroReport, 3*, 21–24.

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.

Dougherty, P. M., & Lenz, F. A. (1994). Plasticity of the somatosensory system following neural injury. In J. Boivie, P. Hansson, & U. Lindblom. (Eds.), *Touch, temperature and pain in health and disease, Wenner-Gren Center Foundation symposia* (pp. 439–460). Seattle: IASP Press.

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

Dubner, R. (1991). Neuronal plasticity and pain following peripheral tissue inflammation or nerve injury. In M. R. Bond, J. E. Charlton, & C. J. Woolf (Eds.), *Proceedings of the Vita World Congress on Pain* (pp. 263–276). Amsterdam: Elsevier.

Dubner, R., & Ruda M. (1992). Activity-dependent neuronal plasticity following tissue injury and inflammation. *Trends in Neuroscience, 15,* 96–103.

Durance, J., & O'Shea, P. (1988). Upper limb amputees: A clinic profile. *International Disability Studies, 10,* 68–72.

Easson, W. M. (1961). Body image and self-image in children. *Archives of General Psychiatry, 4,* 619–621.

Echlin, F. (1949). Pain responses on stimulation of the lumbar sympathetic chain under local anesthesia. *Journal of Neurosurgery, 6,* 530–533.

Editorial [unsigned]. (30 June, 1979). Phantom breast. *Lancet, 1(2),* 1386.

Elizaga, A., Smith, D., Sharar, S., Edwards, T., & Hanson, S. (1994). Continuous regional analgesia by intraneural block: Effect on postoperative opioid requirements and phantom limb pain following amputation. *Journal of Rehabilitation Research and Development, 31,* 179–187.

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

Engkvist, O., Wahren, L. K., Wallin, E., Torebjork, E., & Nyström, B. (1985). Effects of regional intravenous guanethidine block in posttraumatic cold intolerance in hand amputees. *Journal of Hand Surgery, 10,* 145–150.

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

Farley, D., & Smith, I. (1968). Phantom rectum after complete rectal excision. *British Journal of Surgery, 55,* 40.

Feinmann, C., Harris, M., & Crawley, R. (1984). Psychogenic facial pain: Presentation and treatment. *British Medical Journal, 288,* 436–438.

Feinstein, B., Luce, J., & Langton, J. (1954). The influence of phantom limbs. In P. Klopsteg & P. Wilson (Eds.), *Human limbs and their substitutes.* New York: McGraw-Hill.

Fields, H. L. (1987). *Pain.* New York: McGraw–Hill.

Finsen, V., Persen, L., Lovlien, M., Veslegaard, E., Simensen, M., Gasvann A., & Benum, P. (1988). Transcutaneous electrical nerve stimulation after major amputation. *British Journal of Bone and Joint Surgery, 70(B),* 109–112.

Flor, H., Elbert, T., Knecht, S., Wienbruch, C., Pantev, C., Birbaumer, N., Larbig, W., & Taub, E. (1995). Phantom-limb pain as a perceptual correlate of cortical reorganization following arm amputation. *Nature, 375,* 482–484.

Foley, K. M. (1987). Pain syndromes in patients with cancer. *Medical Clinics of North America, 71,* 169–184.

Ford, C. (1983). Chronic pain syndrome. In C. Ford (Ed.), *The somatizing disorders: Illness as a way of life* (p. 121). New York: Elsevier.

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

Frederiks, J. (1963). Occurrence of phantom limb phenomena following amputation of body parts and following lesions of the central and peripheral nervous system. *Psychiatrica, Neurologica, Neurochirurgia, 66,* 73–97.

Fried, K., Govrin-Lippmann, R., Rosenthal, F., Ellisman, M. H., & Devor, M. (1991). Ultrastructure of afferent axon endings in a neuroma. *Journal of Neurocytology, 20,* 682–701.

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

Freeman, W., & Heimburger, R. (1947). Surgical relief of pain in paraplegic patients. *Archives of Surgery, 55,* 433–440.

Fromm, G. H. (1991). Pathophysiology of trigeminal neuralgia. In G. H. Fromm & B. J. Sessle (Eds.), *Trigeminal neuralgia: current concepts regarding pathogenesis and treatment.* (pp. 105–130). Boston: Butterworth-Heinemann.

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

Gerhards, F., Florin, I., & Knapp, T. (1984). The impact of medical, reeducational, and psychological variables on rehabilitation outcomes in amputees. *International Journal of Rehabilitation Research, 7,* 379–388.

Gessler, M. (1984). Relief of phantom pain by stimulation of the nerve supplying the corresponding extensor-muscles. In *Proceedings of the International Society for the Study of Pain,* Hamburg, Germany.

Glickman, L. (1980). *Psychiatric consultation in the general hospital.* New York: Marcel Dekker.

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

Glynn, C. J., Stannard, C., Collins, P. A., & Casale, R. (1993). The role of peripheral sudomotor blockade in the treatment of patients with sympathetically-maintained pain. *Pain, 53,* 39–42.

Gonzales-Darder, J. M., Barbera, J., & Abellan, M. J. (1986). Effect of prior anaesthesia on autotomy following sciatic transection in rats. *Pain, 24,* 87–91.

Gracely, R. H., Lynch, S. A., & Bennett, G. J. (1992). Painful neuropathy: Altered central processing maintained dynamically by peripheral input. *Pain, 51,* 175–194.

Graves, E. J. (1994). Detailed diagnoses and procedures. National Hospital Discharge Survey, 1992. National Center for Health Statistics. *Vital Health Statistics, 13*(119), 1–63.

Greenfield, N. W., & Sternbach, R. A. (1972). *Handbook of psychophysiology.* New York: Holt, Rinehart & Winston.

Gross, D. (1982). Contralateral local anesthesia in the treatment of phantom limb and stump pain. *Pain, 13,* 313–320.

Guilbaud, G. (1991). Neuronal responsivity at supra-spinal levels (ventrobasal thalamus complex and SM1 cortex) in a rat model of mononeuropathy. In J. M. Besson & G. Guilbaud (Eds.), *Lesions of primary afferent fibers as a tool for the study of clinical pain* (pp. 219–232). Amsterdam: Excerpta Medica.

Haber, W. B. (1955). Effects of loss of limb on sensory function. *Journal of Psychology, 40,* 115–123.

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

Haber, W. B. (1958). Reactions to loss of limb: Physiological and psychological aspects. *Annals of the New York Academy of Science, 74,* 624–636.

Hackett, T. (1978). The pain patient: Evaluation and treatment. In T. Hackett & N. Cassem (Eds.), *Handbook of general hospital psychiatry.* St. Louis: C. V. Mosby, 41–63.

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

Hallin, R. G., & Wiesenfeld-Hallin, Z. (1983). Does sympathetic activity modify afferent inflow at the receptor level in man? *Journal of the Autonomic Nervous System, 7,* 391–397.

Hardy, J. D., Wolf, H. G., & Goodell, H. (1952). *Pain sensations and reactions.* New York: William & Wilkins.

Harris, M. (1974). Psychogenic aspects of facial pain. *British Dental Journal, 136,* 199–202.

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

Hendler, N. (1982). The anatomy and psychopharmacology of chronic pain. *Journal of Clinical Psychiatry, 43,* 15–21.

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

Heusner, A. P. (1950). Phantom genitalia. *Transactions of the American Neurological Association, 75,* 128–131.

Hill, A. (1993). The use of pain coping strategies by patients with phantom limb pain. *Pain, 55,* 347–353.

Hockaday, J., & Whitty, C. (1967). Patterns of referred pain in the normal subject. *Brain, 90,* 481–496.

Hokfelt, T., Zhang, X., & Wiesenfeld-Hallin, Z. (1994). Messenger plasticity in primary sensory neurons following axotomy and its functional implications. *Trends in the Neurosciences, 17,* 22–30.

Hono, C. Z., Chena, B. B., Liu, A. Y., & Yu, J. (1990). Local steroid injection: Its effect on the recovery of nerve conduction in experimental neuropathy. *Archives of Physical Medicine and Rehabilitation, 71,* 42–45.

Howe, J. F. (1983). Phantom limb pain—a re-afferentation syndrome. *Pain, 15,* 101–107.

Hrbek, V. (1976). New pathophysiological interpretation of the so-called phantom limb and phantom pain syndromes. *Acta Universitatis Palackinane Olomucenis, 80,* 79–90.

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

International Association for the Study of Pain (IASP). Subcommitee on Taxonomy, Classification of Chronic Pain. (1994). *Pain, 2nd ed.* (p. 70). Seattle: IASP Press.

Jacobson, L., & Chabal, C. (1989). Prolonged relief of acute postamputation phantom limb pain with intrathecal fentanyl and epidural morphine. *Anesthesiology, 71,* 984–985.

Jacobson, L., Chabal, C., & Brody, M. C. (1989). Relief of persistent postamputation stump and phantom limb pain with intrathecal fentanyl. *Pain, 37,* 317–322.

Jacobson, L., Chabal, C., Brody, M. C., Mariano, A. J., & Chaney, E. F. (1990). A comparison of the effects of intrathecal fentanyl and lidocaine on established postamputation stump pain. *Pain, 40,* 137–141

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

Jaeger, H., & Maiger, C. (1992). Calcitonin in phantom limb pain: A double-blind study. *Pain, 48,* 21–27.

Jahangiri, M., Bradley, J. W. P., Jayatunga, A. P., & Dark, C. H. (1994). Prevention of phantom pain after major lower limb amputation by epidural infusion of diamorphine, clonidine and bupivacaine. *Annals of the Royal College of Surgeons of England, 76,* 324–326.

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

Jamison, K., Wellisch, D. K., Katz, R. L., & Pasnau, R. O. (1979). Phantom breast syndrome. *Archives of Surgery, 114,* 93–95.

Jänig, W. (1985). Causalgia and reflex sympathetic dystrophy: In which way is the sympathetic nervous system involved? *Trends in Neuroscience, 8,* 471–477.

Jänig, W. (1987). Pathophysiology of nerve following mechanical and ischemic injury. *Pain, S4,* 335.

Jänig, W. (1990a). Activation of afferent fibers in an old neuroma by sympathetic stimulation in the rat. *Neuroscience Letters, 111,* 309–314.

Jänig, W. (1990b). The sympathetic nervous system in pain: Physiology and pathophysiology. In M. Stanton-Hicks (Ed.), *Pain and the sympathetic nervous system* (pp. 17–89). Boston: Kluwer Academic Publishers.

Jänig, W. (1993). Pain and the sympathetic nervous system: Pathophysiological mechanisms. In R. Bannister & C. J. Mathias (Eds.), *Autonomic failure. A textbook of clinical disorders of the autonomic nervous system* (pp. 231–251). Oxford: Oxford University Press.

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

Jarvis, J. H. (1967). Post-mastectomy breast phantoms. *Journal of Nervous and Mental Disease, 144,* 266–272.

Jensen, T. S., Krebs, B., Nielsen, J., & Rasmussen, P. (1983). Phantom limb, phantom pain and stump pain in amputees during the first 6 months following limb amputation. *Pain, 17,* 243–256.

Jensen, T. S., Krebs, T. S., Nielson, J., & Rasmussen, P. (1984). Non-painful phantom limb phenomena in amputees: Incidence, clinical characteristics and temporal course. *Acta Neurologica Scandinavica, 70,* 407–414.

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. (1986). Phantom limb and related phenomena in patients with amputated extremities. *Ugeskrift for Laeger, 148(a),* 506–509.

Jensen, T. S., & Rasmussen, P. (1994). Phantom pain and other phenomena after amputation. In P. D. Wall & R. Melzack (Eds.), *Textbook of pain* (3rd ed., pp. 651–665). Edinburgh: Livingstone Churchill.

Kaas, J. H., Merzenich, M. M., & Killackey, H. P. (1982). The reorganization of somatosensory cortex following peripheral nerve damage in adult and developing mammals. *Annual Reviews of Neuroscience, 6,* 325–356.

Kajander, K. C., Wakisaka, S., & Bennett, G. J. (1992). Spontaneous discharge originates in the dorsal root ganglion at the onset of a painful peripheral neruopathy in the rat. *Neuroscience Letters, 138,* 225–228.

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

Karstetter, K., & Sherman, R. (1991). Use of thermography for initial detection of early reflex sympathetic dystrophy. *Journal of the American Podiatric Medical Association, 81,* 198.

Katz, J. (1992a). Psychophysiological contributions to phantom limbs. *Canadian Journal of Psychiatry, 37,* 282–298.

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

Katz, J., Clairoux, M., Kavanagh, B. P., Roger, S., Nierenberg, H., Redahan, C., & Sandler, A. N. (1994). Pre-emptive lumbar epidural anaesthesia reduces postoperative pain and patient-controlled morphine consumption after lower abdominal surgery. *Pain, 59,* 395–403.

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 post-operative pain. *Anesthesiology, 77,* 439–446.

Katz, J., & Melzack, R. (1987). Referred sensations in chronic pain patients. *Pain, 28,* 51–59.

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.

Kegel, B., Carpenter, M., & Burgess E. (1978). Functional capabilities of lower extremity amputees. *Archives of Physical Medicine and Rehabilitation, 59,* 109–120.

Kirk, E. J. (1974). Impulses in dorsal spinal nerve rootlets in cats and rabbits arising from dorsal root ganglia isolated from the periphery. *Journal of Comparative Neurology, 2,* 165–176.

Kogerer, H. (1930). On the psychology of the phantom limb. *Zeitschrift für die Gesamte Neurologie und Psychiatrie, 126,* 381–383.

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

Korenman, E. M. D., & Devor, M. (1981). Ectopic adrenergic sensitivity in damaged peripheral nerve axons in the rat. *Experimental Neurology, 72,* 63–81.

Koschorke, G., Meyer, R., & Campbell, J. (1987). Myelinated afferents that innervate neuromas display marked sensitivity to stimuli. *Pain, S4,* 281.

Koschorke, G. M., Meyer, R. A., Tillman, D. B., & Campbell, J. N. (1991). Ectopic excitability of injured nerves in monkey: Entrained responses to vibratory stimuli. *Journal of Neurophysiology, 65,* 693–701.

Krane, E. J., & Heller, L. B. (1995). The prevalence of phantom sensation and pain in pediatric amputees. *Journal of Pain and Symptom Management, 10,* 21–29.

Krebs, D., & Fishman, S. (1984). Characteristics of the child amputee population. *Journal of Pediatric Orthopedics, 4,* 89–95.

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* (Vol. 9, 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., Knudsen, V., Lundby, L., & Hvid, H. (1992). Long term phantom breast syndrome after mastectomy. *The Clinical Journal of Pain, 8,* 346–350.

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.

Kruger, L. (1992). The non-sensory basis of autotomy in rats: A reply to the editorial by Devor and the article by Blumenkopf and Lipman. *Pain, 49,* 153–155.

Kugelberg, E. (1946). "Injury activity" and "trigger zones" in human nerves. *Brain, 69,* 310–324.

Kushner, H. S. (1981). *When bad things happen to good people.* New York: Schocken Books of New York.

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* (Vol. 2, pp. 499–524). Madison, CT: International Universities Press.

LaMotte, R. H., Shain, D., Simone, D. A., & Tsai, E. F. (1991). Neurogenic hyperalgesia: Psychophysical studies of underlying mechanisms. *Journal of Neurophysiology, 66,* 190–211.

Lasagna, L., Mosteller, F., Von Fiesinger, J., & Beecher, H. (1954). A study of the placebo response. *American Journal of Medicine, 16,* 770–779.

Lascelles, R. G. (1966). Atypical facial pain and depression. *British Journal of Psychiatry, 112,* 651–659.

Lasoff, E. M. (1985). When a teenager faces amputation. *RN, 48,* 44–45.

Laughlin, M., & Armstrong, R. (1985). Muscle blood flow during locomotory exercise. *Exercise and Sport Sciences Reviews, 13,* 95.

Lennon, M. C., Link, B. G., Marbach, J. J., & Dohrenwend, B. P. (1989). The stigma of chronic facial pain and its impact on social relationships. *Social Problems, 36,* 117–133.

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.

Lenz, F. A., Tasker, R. R., Dostrovsky, J. O., Kwan, H. C., Gorecki, J., Hirayama, T., & Murphy, J. T. (1987). Abnormal single-unit activity recorded in the somatosensory thalamus of a quadriplegic patient with central pain. *Pain, 3,* 225–236.

Leriche, R. (1939). *The surgery of pain.* (Vol. 5). Baltimore: Williams and Wilkins.

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

Leriche, R. (1947b). A propos des algies des amputés. *Mémoires de l'Academie Chirurgique, 73,* 280–284.

Lesse, S. (1987). Atypical facial pain syndrome of psychogenic facial pain: A masked depression syndrome. In S. Lesse (Ed.), *Masked depression* (pp. 302–317). New York: Jason Aronson.

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* (pp. 121–156). Hillsdale, NJ: Lawrence Erlbaum Associates.

Levine, J. D., Taiwo, Y. O., Collins, S. D., & Tam, J. K. (1986). Noradrenergic hyperalgesia is mediated through interaction with sympathetic postganglionic neurone terminals rather than activation of primary afferent nociceptors. *Nature, 323,* 158–169.

Lhermitte, J., & Susic, Z. (1938). Pathologie de l'image de soi—les hallucinations des amputés: étude clinique et pathologique. *Presse Médicale, 46,* 627–631.

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

Livingston, W. K. (1938). Phantom limb pain. A report of ten cases in which it was treated by injections of procaine hydrochloride near the thoracic sympathetic ganglions. *Archives of Surgery, 37,* 353–370.

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

Loeser, J. D., & Ward, A. A. (1967). Some effects of deafferentation on neurons of the cat spinal cord. *Archives of Neurology, 17,* 629–636.

Loeser, J. D., Ward, A. A., & White, L. E. (1968). Chronic deafferentation of human spinal cord neurons. *Journal of Neurosurgery, 29,* 48–50.

Lombard, M. C., Besse, D., & Besson, J. M. (1992). Deafferentation pain and the opioids in the dorsal horn of the rat spinal cord. In F. Sicuteri (Ed.), *Advances in pain research and therapy* (pp. 29–44). New York: Raven Press.

Lombard, M. C., Nashold, B. S., & Pelissier, T. (1983). Thalamic recordings in rats with hyperalgesia. *Advances in Pain Research, 5,* 767–772.

Maciewicz, R., Bouckom, A., & Martin, J. B. (1985). Drug therapy of neuropathic pain. *Clinical Journal of Pain, 1,* 39–49.

Mailis, A., & Wade, J. (1994). Profile of caucasian females with possible genetic predisposition to reflex sympathetic dystrophy: A pilot study. *Clinical Journal of Pain, 10,* 210–217.

Marbach, J. J. (1976). Phantom bite. *American Journal of Orthodontics, 70,* 190–199.

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

Marbach, J. J. (1978b). Phantom bite syndrome. *American Journal of Psychiatry, 135,* 476–479.

Marbach, J. J. (1993). Is phantom tooth pain a deafferentation (neuropathic) syndrome? Part I. Review of diagnosis, pathophysiology, treatment. *Oral Surgery, Oral Medicine, Oral Pathology, 75,* 95–105.

Marbach, J. J., Hulbrook, J., Hohn, C., & Segal, A. G. (1982). Incidence of phantom tooth pain: An atypical facial neuralgia. *Oral Surgery, Oral Medicine, Oral Pathology, 53,* 190–193.

Marbach, J. J., Lennon, M. C., Link, B. G., & Dohrenwend, B. P. (1990). Losing face: Sources of stigma as perceived by chronic facial pain patient. *Journal of Behavior in Medicine, 13,* 583–604.

Marbach, J. J., & Lipton, J. A. (1978). Aspects of illness behavior in facial pain patients. *Journal of the American Dental Association, 96,* 630–638.

Marbach, J. J., Varoscak, J. R., & Cloidt, J. C. (1986). The diagnosis and treatment of three chronic facial pain disorders: Deafferentation neuralgia, degenerative joint disease of the temporomandibular joint, and myofascial syndrome. *Journal of the Massachusetts Dental Society, 35,* 169–172,200–204.

Marbach, J. J., & Wallenstein, S. L. (1988). Analgesic, mood and hemodynamic effects of intranasal cocaine and lidocaine in chronic facial pain of deafferentation and myofascial origin. *Journal of Pain and Symptom Management, 3,* 73–79.

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

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.

Matzner, O., & Devor, M. (1987). Contrasting thermal sensitivity of spontaneously active A- and C-fibers in experimental nerve-end neuromas. *Pain, 30,* 373–384.

McGrath, P. A. (1990). *Pain in children: Nature, assessment and treatment.* New York: Guilford Publications.

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.

McKechnie, R. (1975). Relief from phantom limb pain by relaxation exercises. *Journal of Behavior Therapy and Experimental Psychiatry, 6,* 262–263.

McLachlan, E. M., Jänig, W., Devor, M., & Michaelis, M. (1993). Peripheral nerve injury triggers noradrenergic sprouting within dorsal root ganglia. *Nature, 363,* 543–546.

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. (1993). Pain: past, present and future. *Canadian Journal of Experimental Psychology, 47,* 615–629.

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, IL: Charles C. Thomas.

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

Melzack, R., & Wall, P. (1982). *The challenge of pain.* New York: Basic Books.

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

Merskey, H. (1985). Psychological normality and abnormality in persistent headache patients. *Pain, 23,* 35–47.

Merskey, H. (1989). Psychiatry and chronic pain. *Canadian Journal of Psychiatry, 34,* 329–336.

Millstein, S., Bain, D., & Hunter, G. (1985). A review of employment patterns of industrial amputees—factors influencing rehabilitation. *Prosthetics and Orthotics International, 9,* 69–78.

Millstein, S., Heger, H., & Hunter, G. (1986) Prosthetic use in adult upper limb amputees. *Prosthetics and Orthotics International, 10,* 27–34.

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.

Miser, A. W., & Miser, J. S. (1989). The treatment of cancer pain in children. *Pediatric Clinics of North America, 36,* 979–999.

Mitchell, S. W. (1872). *Injuries of nerves and their consequences.* Philadelphia: J. B. Lippincott.

Monga, T., & Jaksic, T. (1981). Acupuncture in phantom limb pain. *Archives of Physical Medicine and Rehabilitation, 62,* 229–231.

Morgenstern, F. S. (1964). The effects of sensory input and concentration on post-amputation 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 *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.

Naliboff, B., Cohen, M., & Yellin, A. (1982). Does the MMPI differentiate chronic illness from chronic pain? *Pain, 13,* 333–341.

Nashold, B. S., & Ostdahl, R. H. (1979). Dorsal root entry zone lesions for pain relief. *Journal of Neurosurgery, 51,* 59–69.

Nashold, B., Ostdahl, R., Bullitt, E., Friedman, A., & Brophy, B. (1983). Dorsal root entry zone lesions: A new neurosurgical therapy for deafferentation pain. In J. Bonica (Ed.), *Advances in pain research and therapy* (pp. 739–750). New York: Raven Press.

Nathan, P. W. (1956). Reference of sensation at the spinal level. *Journal of Neurology, Neurosurgery and Psychiatry, 19,* 88–100.

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.

Nathan, P. W. (1989). The sympathetic nervous system and pain. *Functional Neurology, 4,* 11–15.

Nepomuceno, C., Fine, P., Richards, J. S., Gowens, H., Stover, S., Rantanuabol, U., & Houston, R. (1979). Pain in patients with spinal cord injury. *Archives of Physical Medicine and Rehabilitation, 60,* 605–608.

New York Times. Phantom exercises for phantom pain. January 22, 1995, A4.

Nielson, K., Adams, J., & Hosobuchi, Y. (1975). Phantom limb pain: Treatment with dorsal column stimulation. *Journal of Neurosurgery, 42,* 301–307.

Niv, D., & Devor, M. (1993). Does the blockade of surgical pain preempt postoperative pain, and prevent its transition to chronicity? *IASP Newsletter, Nov/Dec,* 2–7.

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.

Nordin, M., Nyström, B., Wallin, U., & Hagbarth, K. E. (1984). Ectopic sensory discharges and paresthesiae in patients with disorders of peripheral nerves, dorsal roots and dorsal columns. *Pain, 20,* 231–245.

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.

Ochoa, J. L., Torebjörk, E., Marchettini, P., & Sivak, M. (1985). Mechanisms of neuropathic pain: Cumulative observations, new experiments, and further speculation. In H. L. Fields, R. Dubner, & F. Cervero (Eds.), *Advances in pain research and therapy* (Vol. 9, pp. 431–450). New York: Raven Press.

Ochoa, J. L., & Torebjörk, H. E. (1980). Paresthesia from ectopic impulse generation in human sensory nerves. *Brain, 103,* 835–853.

Oille, W. A. (1970). Beta adrenergic blockade and the phantom limb. *Annals of Internal Medicine, 73,* 1044–1045.

Oveson, P., Krøner, K., Ørnsholt, J., & Bach, K. (1991). Phantom-related phenomena after rectal amputation: Prevalence and clinical characteristics. *Pain, 44,* 289–291.

Paredes, J. P., Puente, J. L., & Potel, J. (1990). Variations in sensitivity after sectioning the intercostobrachial nerve. *American Journal of Surgery, 160,* 525–528.

Parkes, C. M. (1973). Factors determining the persistence of phantom pain in the amputee. *Journal of Psychosomatic Research, 17,* 97–108.

Parkes, C. M. (1976). The psychological reaction to loss of a limb: The first year after amputation. In J. G. Howells (Ed.), *Modern perspectives in the psychiatric aspects of surgery* (pp. 515–532). New York: Brunner/Mazel.

Parks, C. (1975). Psycho-social transitions: Comparison between reactions to loss of a limb and loss of a spouse. *British Journal of Psychiatry, 12,* 204–210.

Penfield, W., & Rasmussen, T. (1955). *The cerebral cortex of man.* New York: Macmillan.

Pilowsky, I., & Kaufman, A. (1965). An experimental study of atypical phantom pain. *British Journal of Psychiatry, 111,* 1185–1187.

Pitres, A. (1897). Étude sur les sensations illusoires des amputés. *Annales Médico Psychologiques (Paris), 55,* 5–19,177–192.

Poeck, K. (1964). Phantoms following amputation in early childhood and in congenital absence of limbs. *Cortex, 1,* 269–275.

Pollock, L., Brown, M., Boshes, B., Finkelman, I., Chor, H., Arieff, A., & Finkle, J. (1951). Pain below the level of injury of the spinal cord. *Archives of Neurology and Psychiatry, 65,* 319–322.

Pollmann, L. (1990). Phantomerscheinungen auch nach zahnenfernungen? *Fortschrift der Medizin, 108,* 59–61.

Pollmann, L. (1992). Studies on phantom toothache. In F. Sicuteri (Ed.), *Advances in pain research and therapy* (pp. 281–283). New York: Raven Press.

Portenoy, R. K. (1991). Issues in the management of neuropathic pain. In A. I. Basbaum, J. M. Besson (Eds.), *Towards a new pharmacotherapy of pain* (pp. 393–414). New York: John Wiley & Sons.

Portenoy, R. K., & Foley, K .M. (1986). Chronic use of opioid analgesics in non-malignant pain: report of 38 cases. *Pain, 25,* 171–186.

Purry, N., & Hannon, M., (1989). How successful is below-knee amputation for injury? *British Journal of Accident Surgery, 20,* 32–36.

Raja, S. N. (1993). Diagnosis of sympathetically maintained pain: The past, present, and future. *European Journal of Pain, 14,* 45–48.

Raja, S. N., Treede, R. D., Davis, K. D., & Campbell, J. N. (1991). Systemic alpha-adrenergic blockade with phentolamine: A diagnostic test for sympathetically maintained pain. *Anesthesiology, 74,* 691–698.

Ramachandran, V. S., Stewart, M., & Rogers-Ramachandran, D. C. (1992). Perceptual correlates of massive cortical reorganization. *NeuroReport, 3,* 583–586.

Randall, G. C., Ewalt, J. R., & Blair, H. (1945). Psychiatric reaction to amputation. *JAMA, 128,* 645–652.

Rawlings, C. E., & Wilkins, R. H. (1991). Treatment of the deafferentation pain syndromes of the trigeminal system. In B. S. Nashold, Jr., & J. Ovelmen-Levitt (Eds.), *Deafferentation pain syndromes: pathophysiology and treatment* (pp. 291–300). New York: Raven Press.

Rees, R. T., & Harris, M. (1978). Atypical odontalgia. *British Journal of Oral Surgery, 16,* 212–218.

Reik, L. (1984). Atypical odontalgia: A localized form of atypical facial pain. *Headache, 24,* 222–224.

Reisner, H. (1981a). Phantom sensations (phantom arm) in plexus paralysis. In J. Siegfried & M. Zimmermann (Eds.), *Phantom and stump pain* (pp. 62–65). New York: Springer-Verlag.

Reisner, H. (1981b). Phantom tooth. In J. Siegfried & M. Zimmermann (Eds.), *Phantom and stump pain* (pp. 81–83). New York: Springer-Verlag.

Reith, M. E. A., Sershen, H., & Lajtha, A. (1985). Binding sites for tritiated cocaine in mouse striatum and cerebral cortex had different dissociation kinetics. *Journal of Neurochemistry 46,* 309–312.

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.

Richardson, D., Schmitz, M., & Borchers, N. (1986). Relative effects of static muscle contraction on digital artery capillary blood flow velocities. *Microvascular Research, 31,* 157.

Riddoch, G. (1941). Phantom limbs and body shape. *Brain, 64,* 197–222.

Riese, W., & Bruck, G. (1950). Le membre fantôme chez l'enfant. *Revue Neurologique, 83,* 221–222.

Roberts, W. J. (1986). A hypothesis on the physiological basis for causalgia and related pains. *Pain, 24,* 297–311.

Rodin, B. E., & Kruger, L. (1984). Deafferentation in animals as a model for the study of pain: An alternative hypothesis. *Brain Research Reviews, 7,* 213–228.

Roger, A. G. (1989). Use of amitriptyline (elavil) for phantom limb pain in younger children. *Journal of Pain and Symptom Management, 4,* 96.

Rotter, J. (1966). Generalized expectancies for internal versus external control of reinforcement. *Psychological Monographs, 80.*

Santini, M. (1976). Towards a theory of sympathetic-sensory coupling: The primary sensory neuron as a feedback target of the sympathetic terminal. In Y. Zotterman (Ed.), *Sensory function of the skin in primates with special reference to man* (pp. 15–35). Oxford: Pergamon.

Saris, S. C., Iacono, R. P., & Nashold, B. S., Jr. (1988). Successful treatment of phantom pain with dorsal root entry zone coagulation. *Applied Neurophysiology, 51,* 188–197.

Scadding, J. W. (1981). Development of ongoing activity, mechanosensitivity, and adrenalin sensitivity in severed peripheral nerve axons. *Experimental Neurology, 43,* 345–364.

Scadding, J. W., Wall, P. D., Wynn Parry, C. B., & Brooks, D. M. (1982). Clinical trial of propranolol in post-traumatic neuralgia. *Pain, 14,* 283–292.

Schnurr, R. F., & Brooke, R. I. (1992). Atypical odontalgia: Update and comment on long-term follow-up. *Oral Surgery, Oral Medicine, Oral Pathology, 73,* 445–448.

Schott, G. D. (1993). Pain and the sympathetic nervous system. In R. Bannister & C. J. Mathias (Eds.), *Autonomic failure. A textbook of clinical disorders of the autonomic nervous system* (pp. 904–917). Oxford: Oxford University Press.

Seltzer, Z., Beilin, B. Z., Ginzburg, R., Paran, Y., & Shimko, T. (1991). The role of injury discharge in the induction of neuropatic pain behavior in rats. *Pain, 46,* 327–336.

Sessle, B. J. (1987). The neurobiology of facial and dental pain: Present knowledge, future directions. *Journal of Dental Research, 66,* 962–981.

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. (1980). Special review: Published treatments of phantom limb pain. *American Journal of Physical Medicine, 59,* 232–244.

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. (1985a). Relationships between jaw pain and jaw muscle contraction level: Underlying factors and treatment effectiveness. *J. Prosthetic Dent, 54,* 114–118.

Sherman, R. (1985b). Relationships between strength of low back muscle contraction and intensity of chronic low back pain. *Am. J. Phys. Med., 64,* 190–200.

Sherman, R. A. (1989a). Stump and phantom limb pain. *Neurological Clinics, 7,* 249–264.

Sherman, R. (1989b). Phantom limb and stump pain. In R. Portenoy (Ed.), *Neurologic clinics of North America* (pp. 249–264). Philadelphia: W.B. Saunders Co.

Sherman, R. A., & Arena, J. G. (1992). Phantom limb pain: Mechanisms, incidence, and treatment. *Critical Reviews in Physical and Rehabilitation Medicine, 4,* 1–26.

Sherman, R., Arena, J. G., Bruno, G., & Smith, J. (1988). Precursor relationships between stress, physical activity, meteorological factors, and phantom limb pain. In *Proceedings of the annual meeting of the American Pain Society,* Toronto: American Pain Society.

Sherman, R., Arena, J., & Ernst, J. (1990). The mystery of phantom pain: Growing evidence for physiological mechanisms. *Biofeedback and Self-Regulation, 14(4),* 267–280.

Sherman, R., Arena, J., Searle J., & Ginther, J. (1991). Development of an ambulatory recorder for evaluation of muscle tension and movement in soldiers' normal environments. *Military Medicine, 156,* 245–248.

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., Camfield, M., & Arena J. (1995). The effect of presence or absence of pain on low back pain patients' answers to questions on the MMPI's HY, Hs, and D scales. *Journal of Military Psychology, 7,* 28–38.

Sherman, R., Ernst, J., Barja, R., & Bruno, G. (1986). Application of recent discoveries of physiological bases for phantom limb and phantom body pain to chronic pain mechanisms and treatments. *Medical Bulletin of Europe, 4,* 14–19.

Sherman, R., Ernst, J., Barja, R., & Bruno, G. (1988). Phantom pain: A lesson in the necessity for carrying out careful clinical research in chronic pain problems. *Rehabilitation Research and Development, 25(2),* vii–x.

Sherman, R., Ernst, J., & Markowski, J. (1985). Phantom body pain and near surface blood flow among spinal cord injured veterans. In *Proceedings of the American Paraplegia Society annual meeting.* Las Vegas: American Paraplegia Society.

Sherman, R., Ernst, J., & Markowski, J. (1986). Relationships between near surface blood flow and altered sensations among spinal cord injured veterans. *American Journal of Physical Medicine, 65,* 281–297.

Sherman, R., Evans, C., & Arena, J. (1993a). Environmental–temporal relationships between pain and muscle tension: Ramifications for the future of biofeedback treatment. In M. Shtark & T. Sokhadze (Eds.), *Biofeedback: Theory and practice.* Novosibirsk: Nauka Publishers.

Sherman, R., Evans, C., Caminer, S., Sherman, S., & Wolf, S. (1994). Shocking phantom limb pain: First evidence of relationships with environmental temperature and successful self-regulation [abstract]. In *Revision to Biofeedback and Self-Regulation* (pp. 284–285). New York: Plenum Press.

Sherman, R., 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., Goeken, A. (in press). Biofeedback for pain: A multipractitioner study [abstract]. *Biofeedback and Self Regulation, 20,* 314–315.

Sherman, R., Griffin, R., Evans, C., & Grana, A. (1992a). Temporal relationships between changes in phantom limb pain intensity and changes in surface electromyogram of the residual limb. *International Journal of Psychophysiology, 13,* 71–77.

Sherman, R., Griffin, V., Evans, C., & Grana, A. (1992b). Temporal relationships between changes in phantom limb pain and in surface EMG. *Biofeedback and Self-Regulation, 17,* 320.

Sherman, R., & Jones, C. (1996). *The Amputee's Guide.* Invited by the British Limbless Ex-serviceperson's Association. Essex, England: M & B (Felstead) Ltd.

Sherman, R., & Sherman, C. (1983). Prevalence and characteristics of chronic phantom limb pain among American veterans: Results of a trial survey. *American Journal of Physical Medicine, 62,* 227–238.

Sherman, R., & Sherman, C. (1985). A comparison of phantom sensations among amputees whose amputations were of civilian and military origins. *Pain, 2,* 91–97.

Sherman, R., & Sherman, C. (1991). Physiological parameters that change when pain changes: Approaches to unraveling the "cause-or-reaction" quandary. *Bulletin of the American Pain Society, 1(4),* 11–15.

Sherman, R., Sherman, C., & Bruno, G. (1987). Psychological factors influencing chronic phantom limb pain: An analysis of the literature. *Pain, 28,* 285–295.

Sherman, R., Sherman, C., & Gall, N. (1980). Survey of current treatment of phantom limb pain in the United States. *Pain, 8,* 85–99.

Sherman, R., Sherman, C., & Grana, A. (1989). Occurrence of acute muscle contractions in the residual limbs of amputees preceding acute episodes of phantom limb pain. *Biofeedback and Self-Regulation, 14(2),* 169.

Sherman, R., Sherman, C., & Parker, L. (1984). Chronic phantom and stump pain among American veterans: Results of a survey. *Pain, 18,* 83–95.

Sherman, R., & Tippens, J. (1982). Suggested guidelines for treatment of phantom limb pain. *Orthopedics, 5,* 1595–1600.

Shukla, G., Sahu, S., Tripathi, R., & Gupta, D. (1982a). A psychiatric study of amputees. *British Journal of Psychiatry, 141,* 50–53.

Shukla, G., Sahu, S., Tripathi, R., & Gupta, D. (1982b). Phantom limb: A phenomenological study. *British Journal of Psychiatry, 141,* 54–58.

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.

Siegel, E. F. (1979). Control of phantom limb pain by hypnosis. *American Journal of Clinical Hypnosis, 21,* 285–286.

Siegfried, J., & Cetinalp, E. (1981). Neurosurgical treatment of phantom limb pain: A survey of methods. In J. Siegfried & M. Zimmerman, (Eds.), *Phantom and stump pain* (pp. 148–155). Berlin: Springer Verlag.

Simmel, M. L. (1956). On phantom limbs. *Archives of Neurology and Psychiatry, 75,* 637–647.

Simmel, M. L. (1962a). Phantom experiences following amputation in childhood. *Journal of Neurology, Neurosurgery and Psychiatry, 25,* 69–78.

Simmel, M. L. (1962b). The reality of phantom sensations. *Social Research, 29,* 337–356.

Simmel, M. L. (1966). A study of phantoms after amputation of the breast. *Nuropsykologica, 4,* 331–350.

Singer, J. A., & Salovey, P. (1988). Mood and memory: Evaluating the network theory of affect. *Clinical Psychology Review, 8,* 211–251.

Skoyles, J. R. (1990). Is there a genetic component to body schema? *Trends in Neuroscience, 13,* 409.

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.

Smythe, H. (1984). Problems with the MMPI. *Journal of Rheumatology, 11,* 417–418.

Sohn, D. L. (1914). The psychic complex in congenital deformity. *New York Medical Journal, 100,* 959–961.

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.

Sotgiu, M. L., Biella, G., & Riva, L. (1994). A study of early ongoing activity in dorsal horn units following sciatic nerve constriction. *NeuroReport, 5,* 2609–2612.

Souques-Poisot, A. (1905). Origine peripherique des hallucinations des membres amputes. *Revue Neurologique (Paris), 13,* 1112–1116.

Staps, T., Hoogenhout, J., & Wobbes T. (1985). Phantom breast sensations following mastectomy. *Cancer, 56,* 2898–2901.

Stein, J., & Warfield, C. (1982). Phantom limb pain. *Hospital Practice, 17,* 166–167.

Stengel, E. (1965). Pain and the psychiatrist. *British Journal of Psychiatry, 111,* 795–802.

Sternbach, R. (1968). *Pain: A psychophysiological analysis* (pp. 117–176). New York: Academic Press.

Sternbach, T., Nadvorna, H., & Arazi, D. (1982). A five year follow-up study of phantom limb pain in post traumatic amputees. *Scandinavian Journal of Rehabilitation Medicine, 14,* 203–207.

Stevens, P. E., Dibble, S. L., & Miaskowski, C. (1995). Prevalence, characteristics, and impact of postmastectomy pain syndrome: An investigation of women's experiences. *Pain, 61,* 61–68.

Stevenson, G. H. (1950). Amputations with special reference to phantom limb sensations. *Edinburgh Medical Journal, 57,* 44–56.

Studies relating to pain in the amputee. (1952, June). *A progress report from the prosthetic devices research project at the Institute of Engineering Research,* Department of Engineering, University of California Berkeley Medical School, Series II, issue 23.

Sugarbaker, P., Weiss, C., Davidson, D., & Roth, Y. (1984). Increasing phantom limb pain as a symptom of cancer reccurrence. *Cancer, 54,* 373–375.

Sunderland, S. (1968). *Nerve and nerve injuries.* Edinburgh: Livingstone.

Sunderland, S. (1978). *Nerves and nerve injuries* (2nd edition). Baltimore: Williams and Wilkins.

Sweet, W. (1975). Phantom sensations following intraspinal injury. *Neurochirurgia (Stuttgart), 18,* 139–154.

Szasz, T. S. (1949). Psychiatric aspects of vagotomy: IV. Phantom ulcer pain. *Archives of Neurology and Psychiatry, 62,* 728–733.

Tasker, R. R. (1984). Deafferentation. In P. D. Wall & R. Melzack (Eds.), *Textbook of pain* (pp. 119–132). Edinburgh: Churchill Livingstone.

Tasker, R. R. (1989). Management of nociceptive, deafferentation and central pain by surgical intervention. In J. L. Fields (Ed.), *Pain syndromes in neurology* (pp. 119–132). Guildford/Surrey: Butterworths.

Taub, E. (in press).

Tebbi, C. K., & Mallon, J. C. (1987). Long-term psychosocial outcome among cancer amputees in adolescence and early adulthood. *Journal of Psychosocial Oncology, 5,* 69–82.

Tebbi, C. K., Petrilli, A. S., & Richards, M. E. (1989). Adjustment to amputation among adolescent oncology patients. *The American Journal of Pediatric Hematology/Oncology, 11,* 276–280.

Teuber, H. J., Kreiger, H. P., & Bender, M. B. (1949). Reorganization of sensory function in amputation stumps; two-point discrimination. *Federation Proceedings, 8,* 156.

Thyregod, H., Jenson, J., Retpen, J., Boberg, G., Ramussen, E., & Jensen, S. (1988). Traumatic amputation of the upper limb: The use of body-powered prostheses and employment consequences. *Prosthetics and Orthotics International, 12,* 50–52.

Torebjork, H. E., Lundberg L. E., & LaMotte, R. H. (1992). Central changes in processing of

mechanoreceptive input in capsaicin-induced secondary hyperalgesia in humans. *Journal of Physiology, 448,* 765–780.

Treede, R. D., Davis, K. D., Campbell, J. N., & Raja, S. N. (1992). The plasticity of cutaneous hyperalgesia during sympathetic ganglion blockade in patients with neuropathic pain. *Brain, 115,* 607–621.

Tsushima, W. (1982). Treatment of phantom limb pain with EMG and temperature biofeedback: A case study. *American Journal of Clinical Biofeedback, 5,* 150–153.

Turk, D. C., & Salovey, P. (1984). Chronic pain as a variant of depressive disease. *Journal of Nervous and Mental Disease, 172,* 398–404.

Usdin, G., & Lewis, J. (1979). *Psychiatry in general medical practice.* New York, McGraw–Hill.

Van Bogaert, L. (1934). Sur la pathologie de l'image de soi (Études anatomo-cliniques). *Annales Médico Psychologiques (Paris), 14,* 519–555,714–759.

Varma, S. K., Lal, S. K., & Mukherjee, A. (1972). A study of phantom experience in amputees. *Indian Journal of Medical Sciences, 26,* 185–188.

Varni, J. W., Rubenfeld, L. A., Talbot, D., & Setoguchi, Y. (1989a). Determinants of self-esteem in children with congenital/acquired limb deficiencies. *Developmental and Behavioral Pediatrics, 10,* 13–16.

Varni, J. W., Rubenfeld, L. A., Talbot, D., & Setoguchi, Y. (1989b). Family functioning, temperament, and psychologic adaptation in children with congenital or acquired limb deficiencies. *Pediatrics, 84,* 323–330.

Varni, J. W., Rubenfeld, L. A., Talbot, D., & Setoguchi, Y. (1989c). Stress, social support, and depressive symptomatology in children with congenital/acquired limb deficiencies. *Journal of Pediatric Psychology, 14,* 515–530.

Varni, J. W., & Setoguchi, Y. (1991). Psychosocial factors in the management of children with limb deficiencies. *Physical Medicine and Rehabilitation Clinics of North America, 2,* 395–404.

Varni, J. W., Setoguchi, Y., Talbot, D., & Rubenfeld Rappaport, L. (1991). Effects of stress, social support, and self-esteem on depression in children with limb deficiencies. *Archive of Physical Medicine and Rehabilitation, 72,*1053–1058.

Vetter, R. J., & Weinstein, S. (1967). The history of the phantom in congenitally absent limbs. *Neuropsychologia, 5,* 335–338.

Wahren, L. (1990) Changes in thermal and mechanical pain thresholds in hand amputees. A clinical and physiological long-term follow-up. *Pain, 42,* 269–277.

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. (1992). The biological function and dysfunction of different pain mechanisms. In F. Sicuteri (Ed.), *Advances in pain research and therapy* (pp. 19–28). New York: Raven Press.

Wall, P. D., & Devor, M. (1981). The effect of peripheral nerve injury on dorsal root potentials and on the transmission of afferent signals into the spinal cord. *Brain Research, 209,* 95–111.

Wall, P. D., & Devor, M. (1983). Sensory afferent impulses originate from dorsal root ganglia as well as from the periphery in normal and nerve-injured rats. *Pain, 17,* 321–339.

Wall, P. D., Devor, M., Inbal, R., Scadding, J. W., Schonfeld, D., Seltzer, Z., & Tomkiewicz, M. M. (1979). Autotomy following peripheral nerve lesions: Experimental anaesthesia dolorosa. *Pain, 7,* 103–113.

Wall, P., & Gutnick, M. (1974a). Properties of afferent nerve impulses originating from a neuroma. *Nature, 248,* 740–743.

Wall, P. D., & Gutnick, M. (1974b). Ongoing activity in peripheral nerves: The physiology and pharmacology of impulses originating from a neuroma. *Experimental Neurology, 43,* 580–593.

Wall, R., Novotny-Joseph, P., & MacNamara, T. E. (1985). Does preamputation pain influence phantom limb pain in cancer patients? *Southern Medical Journal, 78,* 34–36.

Wall, P. D., Scadding, J. W., & Tomkiewicz, M. M. (1979). The production and prevention of experimental anesthesia dolorosa. *Pain, 6,* 175–182.

Wallgren, G. R. (1954). Phantom experience at spinal anaesthesia. *Annales Chirurgicae et Gynaecologicae Fenniae, 43 (Supplementum),* 486–500.

Wallin, G., Torebjörk, E., & Hallin, R. (1976). Preliminary observations on the pathophysiology of hyperalgesia in the causalgic pain syndrome. In Y. Zotterman (Ed.), *Functions of the skin in primates* (pp. 489–499). New York: Pergamon Press.

Wangenstein, O. H., & Carlson, A. (1931). Hunger sensations in a patient after total gastrectomy. *Proceedings of the Society for Experimental Biology and Medicine, 28,* 545–547.

Weinstein, S. (1969). Neuropsychology of the phantom. In A. L. Benton (Ed.), *Contributions to clinical neuropsychology* (p. 73). Chicago: Aldine.

Weinstein, S., & Sersen, E. A. (1961). Phantoms in cases of congenital absence of limbs. *Neurology, 11,* 906–911.

Weinstein, S., Sersen, E. A., & Vetter, R. J. (1964). Phantoms and somatic sensation in cases of congenital aplasia. *Cortex, 1,* 276–290.

Weinstein, S., Vetter, R. J., & Sersen, E. A. (1970). Phantoms following breast amputation. *Neuropsykologica, 8,* 185–197.

Weisenberg, M. (1975). *Pain: Clinical and experimental perspectives.* St Louis: C. V. Mosby.

Weiss, S., & Fishman, S. (1963). Extended and telescoped phantom limbs in unilateral amputees. *Journal of Abnormal and Social Psychology, 66,* 489–497.

Weiss, S., Fishman, S., & Krause, F. (1971). Severity of disability as related to personality and prosthetic adjustment of amputees. *Psychological Aspects of Disability, 18,* 67–75.

White, J. C., & Sweet, W. H. (1969). *Pain and the neurosurgeon.* Springfield, IL: Charles C. Thomas.

Wiesenfeld-Hallin, Z., & Hallin, R. G. (1983). Possible role of sympathetic activity in abnormal behavior of rats induced by lesion of the sciatic nerve. *Journal of the Autonomic Nervous System, 7,* 385–390.

Wilcox, G. L. (1991). Excitatory neurotransmitters and pain. In M. R. Bond, R. Gebhart, & R. Dubnet (Eds.), *Proceedings of the 5th World Congress on Pain* (pp. 263–276). Elsevier: Amsterdam.

Wilentz, J. (1991). Female phantom pain. *Bulletin of the American Pain Society, 1,* 14–15.

Woolf, C. J. (1992). Excitability changes in central neurons following peripheral damage. In W. D. Willis, Jr. (Ed.), *Hyperalgesia and allodynia* (pp. 221–243). New York: Raven Press.

Woolsey, C. N., Theodore, M. D., Erickson, C., & Gilson, W. E. (1979). Localization in somatic sensory and motor areas of human cerebral cortex as determined by direct recording of evoked potentials and electrical stimulation. *Journal of Neurosurgery, 51,* 476–506.

Wynn-Parry, C. B. (1980). Pain in avulsion lesions of the brachial plexus. *Pain, 9,* 41–53.

Yang, T. T., Gallen, C. C., Ramachandran, V. S., Cobb, S., Schwartz, B. J., & Bloom, F. E. (1994). Noninvasive detection of cerebral plasticity in adult human somatosensory cortex. *NeuroReport, 5,* 701–704.

Yarnitzky, D., Barron, S. A., & Bental, E. (1988). Disappearance of phantom pain after focal brain infarction. *Pain, 32,* 285–287.

Yasumo, W., Nishigori, A., & Yamaguchi, Y. (1984). Facilitatory effects of monoamine on autotomy of rats induced by peripheral nerve section. *Pain Supplement, 2,* S446.

Zimmerman, M. (1991). Central nervous mechanisms modulating pain-related information: Interaction with peripheral input. In K. L. Casey (Ed.), *Pain and central nervous system disease: The central pain syndromes* (pp. 183–199). New York: Raven Press.

Zubek, J. P., Flye, J., & Aftanas, M. (1964). Cutaneous sensitivity after prolonged visual deprivation. *Science, 144,* 1591–1593.

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