Phantom Tooth Pain: A New Look at an Old Dilemma

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ABSTRACT

The aim of this paper is to review the current knowledge of phantom tooth pain, a neuropathic facial pain disorder, thought to result from peripheral nerve injury. Phantom tooth pain is a deafferentation pain disorder of persistent toothache in teeth that have been denervated (usually by root canal treatment) or pain in the area formerly occupied by teeth prior to their extraction. The pain usually extends to the facial structures adjacent to tissues that have undergone deafferentation. The clinical characteristics, differential diagnosis, epidemiology, and treatment of phantom tooth pain are reviewed. Suggestions for further research include the need for controlled treatment trials and modification of current criteria.

Conclusions. Phantom tooth pain has much in common with other phantom pain disorders. In the absence of controlled clinical trials specifically directed to phantom tooth pain, treatment should be guided by standards used for other neuropathic pain disorders. Revised diagnostic criteria for phantom tooth pain are proposed.

Introduction

Throughout the past half century, those concerned with the classification of facial pain disorders have struggled with many attempts to bring order to the field. In the absence of a generally accepted classification system for facial pain, numerous labels are employed to describe essentially the same conditions. In addition, ‘wastebasket’ categories such as ‘atypical facial pain’ (AFP) are commonly used. Here heterogeneous facial pain conditions are placed in one category. The common thread among these heterogeneous neuropathic disorders is that they can not be explained by one etiology or anatomic lesion, and that treatment response remains unpredictable.

We present a review of the clinical characteristics for one disorder frequently labeled as AFP, namely phantom tooth pain (PTP), and propose revised diagnostic criteria for PTP. PTP is a syndrome of persistent pain and paraesthesia in the face, teeth, and other oral structures. The onset usually follows nerve injury to the face often accompanying dental or surgical procedures. In the case of tooth extraction, the pain is found in the edentate area. This latter condition is analogous to stump pain following limb amputation [1-3].

Phantom pain is customarily associated with limb amputations [4]. However, phantom pain is not confined to limbs. For example, phantom phenomena are reported in 20-25% of post mastectomy cases, 50% of them painful [5]. Data suggests that an individual surviving the amputation of any anatomic structure can experience phantom pain phenomenon. Yet, despite the fact that teeth are probably the most commonly amputated structures among members of industrialized societies, relatively little attention has been paid to orofacial phantom pain. Moreover, teeth are unique in that the neural structures that serve them can be entirely eliminated routinely without amputating the entire part. One could argue quite legitimately that the term “phantom” should not apply to postendodontic neuropathic pain. Rather than attempting to introduce a unique term to describe this situation, subsuming this clinical state under the rubric of phantom pain appears to be the most parsimonious approach. There is precedent in the literature for other unusual manifestations of “phantom pain” [6-9]. While a similar process may accompany spinal cord injury, for example, with PTP, elimination of neural tissue occurs in the course of root canal therapy. In spinal cord injury, phantom-like pains appear to develop...
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[9] and are readily identifiable as stereotypical phantom pain, given the extreme alteration in function and sensation that accompany such an injury. In the case of PTP, the idea of phantom pain may be much less apparent to the clinician and patient alike.

Although the past decade has witnessed wider recognition of PTP [10], its systematic study is a relatively recent phenomenon. The term PTP was first used in the English-language literature in 1978 [11]. At virtually the same time, the term PTP appeared in French [12] and German publications [13]. In all, Medline lists 23 papers using the term PTP since 1975. In the same period, Medline lists 175 papers under the heading “atypical facial pain,” 70 of which have been published since 1990. Despite the heterogeneity and ambiguity implicit in the term atypical facial pain, it continues to be used. In contrast, we advocate the use of terms that imply greater homogeneity of clinical presentation and etiology, such as PTP. This approach lends itself to the goals of establishing reliable diagnoses, providing more specific treatment, and facilitating research on pathogenesis. Classification systems are important to clinicians. They help them in the selection of treatments, provide a gauge for prognosis, direct the search for risk factors, and alert one to comorbid conditions. To be useful, a taxonomy system must reliable [14]; different examiners applying the diagnostic standard should reach the same conclusions. To be reliable, the system must possess operational criteria and use inclusion and exclusion criteria. It should also be comprehensive, applying to milder, more diagnostically subtle cases as well as the more obvious, severe ones. Nevertheless, the taxonomy of facial pain continues to be problematic for reasons that will become apparent.

The identification of those at high risk for PTP needs further exploration. Vulnerability to phantom pain has been demonstrated to be genetic in animals [15-17] and hypothesized to be so in humans [15,18]. Given the universal loss of deciduous teeth, perhaps evidence for the neuromatrix theory can be found in children. Humans appear to experience no lasting sensory sequelae from the loss in contrast to adult dentition.

While acknowledging the frequent use of the term “atypical facial pain” (AFP), Merskey singles out and “deliberately rejected” AFP from the International Association for the Study of Pain taxonomy [19]. He suggests that such patients can better be served by attempting to define them by other, more specific diagnoses, e.g., temporomandibular pain syndrome, atypical odontalgia, etc. Despite his advice, the diagnoses atypical facial pain (AFP) and atypical facial neuralgias persist in the literature and frequently appear on clinical charts. AFP continues to be used as a euphemism for medically unexplained chronic facial pain [20]. However, like other heterogeneous diagnostic labels, such as temporomandibular disorders (TMD) [21], AFP leads to confusion regarding the specific nature of the complaint and often results in inconsistent approaches to treatment. For example, Ford [22] writes that “treatment for patients with atypical face pain is essentially the treatment indicated for depression.” As we will see, Ford’s conclusion was based on a body of literature that predates modern criteria for various facial pain disorders.

Putative Mechanisms for Phantom Pain

Phantom limb phenomenon following amputation is almost universal [1]. Most individuals report the vivid impression that the amputated member is still present and, less often, painful. Mounting evidence from animal and now human studies identify long-term cortical reorganization of the somatopic arrangement can follow alterations of peripheral input.

Neurophysiologic mechanisms of phantom pain

Fields and colleagues [23] describe three different, but nonmutually exclusive, mechanisms to explain neuropathic deafferentation pain. In the first type, they speak of an “irritable nociceptor.” Here an anatomically intact but physiologically abnormal primary afferent nociceptor results in mechanical allodynia due to central sensitization of pain transmission neurons. In other patients, extensive degeneration of C-fibers is present with the allodynia. A third mechanism accounts for both constant pain and sensory loss without allodynia, i.e., anesthesia dolorosa. Here deafferentation may result in changes in the activation state of central nervous system (CNS) pain transmission neurons. Although based largely on animal studies, indirect evidence from humans suggests that more than one of these mechanisms can cooccur in the same individual, and even change in time by means of synaptic reorganization.

Melzack [18,24,25] has posited a provocative hypothesis regarding phantom phenomenon. He suggests that: (1) the bodily sensations that we perceive in our brain are started and maintained, typically, by input derived directly from our bodies. However, because phantom sensations feel so vivid, they too are probably subserved by the same neural processes; (2) All sensations we feel from our bodies, such as pain, can be felt without input from the
body. From this we conclude that the sensory experiences lie innately in the neural “hardware” of the brain because external stimuli may trigger the sensations but do not create them. They are produced by the brain itself. Other examples of physiologic responses that appear to occur without external stimulation include the vivid sexual responses to dreams; (3) The sense of self is generated in the CNS, not from the sensations derived though the peripheral nervous system (PNS). The uniqueness we perceive physically as ourselves (not someone else) takes place in the brain; (4) The CNS processes that regulate the recognition of the body are genetically specified, though probably modified by experience. These four elements comprise Melzack’s theory of the “neuromatrix.”

In summary, the neuromatrix theory of Melzack posits that loss of input to the CNS by deafferentation following injury or amputation produce localized abnormal neural activity. Tissues near the site of injury, visceral sensory nerves, from small afferents in the sympathetic chain, and from higher psychoneural processes trigger prolonged firing, resulting in chronic pain in discrete areas of the denervated body parts and even more remote body sites. The neuromatrix provides an intriguing theoretical framework for understanding one of the most perplexing of pain disorders, phantom pain, and associated phenomena.

Psychiatric mechanisms

An extensive literature exists regarding the controversy as to whether phantom pain has a psychological or physical basis [26]. Many attribute the etiology of phantom pain and of AFP to psychological factors [27–29]. A standard neurology textbook associates AFP with women, depression, anxiety, and hysteria [30]. Indeed, as with many other phantom pain disorders, most PTP patients could meet DSM-IV criteria for somatoform pain disorder [31]. A psychological attribution to the diagnosis is not surprising when the patient’s symptoms appear to fit no known physical disorder, the symptoms are notoriously recalcitrant to traditional dental and surgical treatment, and examination discloses high rates of psychiatric symptomatology [27]. Nevertheless, it remains plausible that psychological abnormalities observed in phantom patients are a consequence of the stress associated with the pain, as this explanation has recently been confirmed for other orofacial pain conditions [32]. There is no evidence currently that PTP is characterized by a pre-morbid personality [27]. Few pain clinicians now raise the specter of a psychological basis for PTP.

Definitions and Subgroups

Phantom Tooth Pain

The most common form of orofacial phantom pain is PTP. There are reports of other facial phantom pains involving eyes, noses, ears, and tongues. Like phantom pain due to spinal cord injury, teeth can be denervated yet still be attached to the individual. What is obviously ‘missing’ in the case of many other phantom pains, such as limb amputation or mastectomy, may be much less obvious with PTP.

PTP is a deafferentation syndrome of persistent toothache in teeth that have been denervated (root canal) or in the area formerly occupied by teeth before their extraction. The pain often extends to the facial structures adjacent to the tissues that had undergone deafferentation. Table 1 presents a revision of the 1992 criteria for PTP. These revised criteria are distinguished from the earlier published version [33] in that they attempt to aid differential diagnosis rather than simply provide a comprehensive description of the syndrome.

Usually PTP follows dental or surgical procedures such as root canal therapy, apicoectomy, or tooth extraction. Other facial traumas and surgical procedures may precede the onset of PTP. PTP is characterized primarily by persistent pain. Neither repeated endodontic treatment, apicoectomy, nor

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<th>Table 1</th>
<th>Diagnostic Criteria for PTP</th>
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<td>1. Pain is located in the face or described as a toothache.</td>
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<td>2. The pain is described as a constant dull, deep ache. (Less than 10% of sufferers report occasional spontaneous sharp pains that overlap the ache. Sharp pain is not essential to meet criteria).</td>
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<td>3. A brief (seconds to minutes) pain-free period is reported upon awakening from sleep. There are no other refractory periods.</td>
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<td>4. Pain develops (or continues) within one month following endodontic treatment or tooth extraction, or other trauma or medical procedure related to the face.</td>
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<td>5. Overlying the area of dental (or other) treatment (usually on the surface of the face but occasionally intraorally) is a location with a much lowered pain threshold (hyperalgesia), often surrounded by a larger area with less severe hyperalgesia.</td>
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<td>6. Sleep is undisturbed by pain or other phantom sensations.</td>
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<td>7. No radiographic or laboratory tests suggest other sources of pain.</td>
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Note: Prevalence does not differ by sex. The loss of deciduous teeth does not result in phantom tooth pain.
more tooth extractions render the affected area free of pain. On the contrary, procedures and other surgical interventions, such as trigeminal rhizotomy and microvascular decompression, frequently exacerbate pain severity and, in addition, may increase the distribution of pain in the trigeminal nerve [17]. Animal data suggest that dental pulp amputation not only result in a lesion at the tooth apex, but consistent with other nerve injuries, also alters the CNS to generate ongoing pain [34]. This central “generator” could account for the lack of response to analgesic measures taken at the PNS. Some suggest that those neuropathic pains that have a predominantly central “generator” comprise the so-called deafferentation pain syndromes [35]. This theory does not exclude the possibility that a peripheral lesion is required to sustain the pain resulting from the central generator [35,36].

Because PTP is often incessant, incapacitating, and inscrutable, the patients present a vexing problem for clinicians. Complicating matters for the clinician are difficulties regarding differential diagnoses. PTP is mistaken frequently for temporomandibular joint disorder (TMJ) or trigeminal neuralgia, sinusitis, or even ill-fitting dentures (see section on differential diagnosis, below). All too often, chronic facial pain disorders appear superficially similar one to another. Moreover, the clinician cannot turn to a ‘gold standard.’ Confirmatory diagnostic laboratory and radiographic tests are unavailable. While a physician with a specialty pain practice is sensitive to this, many dentists are used to relying on ‘objective’ signs to make a diagnosis. Dentists are trained to depend strongly on radiographs. Like all radiographs, dental radiographs are open to interpretation and can lead to equivocal treatment interventions. This frequently compounds frustrating, expensive, and lengthy delays in arriving at the correct diagnosis while diagnostic tests and treatments are pursued and repeated.

In the absence of a diagnostic gold standard, accurate differential diagnosis of PTP is dependent upon history, physical examination, and such often overlooked tools as epidemiology [37,38]. While only limited information about the epidemiology of PTP is currently available [see below], the epidemiology of other orofacial pain conditions, e.g., TMJ [39], TN [40] is better known. The demographic distribution of these other orofacial pain conditions provides a source of clues for other possible diagnoses. For example, the underlying condition for recent onset facial pain in a 60-year-old male is unlikely to be a TMJ because TMJ is much more likely to be found in younger women [41]. Trigeminal neuralgia, temporal arteritis, or PTP remain possible explanations, with the latter especially likely when a symptom onset was shortly after root canal therapy.

Epidemiology of PTP

Epidemiologic data concerning chronic pain disorders such as PTP are difficult to obtain. Crombie and Davies [42] note that exploring the epidemiology of a disease requires a formal definition of a diagnostic group. PTP is still undergoing revision of its criteria (Table 1) to establish a relatively homogeneous group with diagnosis that is likely to be replicated by different clinical research teams. For example, the International Association for the Study of Pain [19] could identify only PTP because intraoral stump pain syndrome was only conceptualized in 1996 [1,3]. Because no available data on the prevalence of intraoral stump pain exists, the current discussion focuses on PTP, drawing in part from what is known about other types of phantom pain.

As reviewed by Kalauokalani and Loeser [43], the prevalence of nonorofacial phantom pain following amputation is reported to range between 53% and 72%. These rates remain fairly constant for the five years following amputation. Jensen [44] reported a rate of 59% at two years post amputation. Sherman [45] reported a rate of 78% in a large (n = 2694) survey that varied widely in time since amputation. Several investigators report a gradual decline in the prevalence of postamputation phantom pain with the passage of time [43,45]. Preamputation pain is agreed to be the major risk factor predicting postamputation phantom pain [44]. As most root canal procedures are preceded by preamputation pain, it would appear that the risk of post-root-canal pain should be relatively high. Although healthy teeth provide relatively little sensory feedback, toothache can occupy a great deal of one’s attention.

There are two common dental procedures that appear to increase the risk of PTP, tooth extraction and endodontic therapy. Thus, it is surprising that PTP is not more frequently reported in the literature. Several explanations are possible. Underreporting due to lack of recognition is a likely explanation. Alternatively, the quantity of neural tissue associated with a tooth compared with a limb may explain the difference in rates. Melzack’s neuromatrix theory predicts that the general lack of awareness of teeth, unless diseased, would also result in relatively low rates of phantom tooth pain.

Reports of PTP following tooth extraction are unavailable. In a single study [46] of 436 individuals who underwent root canal therapy, 7% reported continued pain more than one month after treatment. Eight of the 11 subjects who were directly
examined meet criteria for PTP. Extrapolating from the subsample on whom follow up physical examination was possible, the postendodontic therapy rate of PTP was 3 to 6%, with no significant differences in rates for men versus women. All those with verified PTP following endodontic treatment reported tooth pain before endodontic therapy, vs. 75% of the entire sample undergoing endodontic therapy. This is consistent with other studies showing that pain before amputation is a risk factor for nonorofacial phantom pain.

If one generalizes to the U.S. population of 260 million people and assumes conservatively that 5%, or 13 million members of the U.S. population, have undergone endodontic treatment and 5% of this group result in PTP, then 650,000 cases of PTP secondary to endodontic therapy would potentially exist. Rates are potentially even higher when accounting for other potential risk factors such as tooth extraction.

The risk of more severe and widespread PTP increases for the individual undergoing multiple endodontic treatments, particularly when the additional therapies are misdirected attempts to treat the unrecognized PTP. Per capita endodontic treatment in the U.S. will likely increase with the aging of the population. With more elders eager and able to afford the services, the rates of PTP are bound to rise.

**Differential diagnosis**

PTP is often confused with the typical neuralgias and myofascial pain. The most common of the typical neuralgias is trigeminal neuralgia (TN) [38,40]. Both TN and PTP patients complain of pain in the face, often specifically in teeth. Here the resemblance ends. The paroxysmal, sharp, sudden, electrical-like stabbing recurrent pain is unlike the dull uninterrupted pain of PTP. The age of onset of TN is usually after the fourth decade with a peak onset in the fifth and sixth decade. Other typical neuralgias are associated with acute herpes zoster, post herpetic neuralgia, and geniculate neuralgia.

In addition, myofascial face pain is often confused with PTP. Myofascial face pain, also known as ‘TMJ’ or myofascial TMD [39], like PTP, but unlike TN, presents as a constant, dull aching face pain. Although myofascial face pain can be comorbid with, and may be secondary to PTP, when the dull aching pain is restricted to the facial muscles, a diagnosis of PTP is excluded.

While not technically a matter of confusion, as stated earlier, PTP is frequently labeled AFP. AFP is a diagnosis made, traditionally, after excluding other possibilities for which the clinician has a physical explanation. In the absence of specific criteria and considering the recent developments in pain disorder taxonomy, the term AFP might best be relegated to one of historic interest.

**Treatment**

The need for more homogeneous groups of neuropathic pain subjects is a recurrent theme among those attempting to interpret the treatment literature. By focusing attention on PTP, it is hoped more researchers will conduct controlled trials. Meanwhile, to date there are few controlled clinical trials for treatment of PTP to act as a guide. Thus, although the following section on treatment focuses on the PTP, we are required to make inferences from controlled studies of more extensively studied neuropathic pain disorders such as trigeminal and post herpetic neuralgia.

The past decade has witnessed the availability of many new adjuvant analgesic drugs and formulations for the treatment of neuropathic pain. Nevertheless, progress in the treatment of PTP has been mixed because of the long delay in diagnosis. Earlier detection of more patients would certainly result in avoiding the more aggressive and egregious errors of the past, namely unnecessary root canal treatments, tooth extractions, and neurosurgical procedures.

With the exception of trigeminal neuralgia, physicians have traditionally relegated the treatment of most chronic facial pain disorders to dental and oral surgeons. Even in the case of trigeminal neuralgia, many patients undergo unnecessary dental procedures before obtaining proper diagnosis and treatment [47]. Ostensibly, the decision to refer patients for dental care is based on the notion that these disorders require special dental expertise outside the scope of conventional medical training. There is little evidence to support this viewpoint. Moreover, physicians assume treatments used by many dentists for other facial pain problems, i.e., bite plates or bite adjustment, are reasonably effective. There is no literature, either anecdotal report or controlled clinical trial, to suggest their efficacy for PTP. Once the correct diagnosis and implicit knowledge of pathophysiology underlying PTP is made, it becomes clear that there is no logic for traditional dental treatment approaches.

Centrally acting drugs are meant to influence different impulses that culminate in central synaptic excitability. This includes the administration of both oral, transdermal, and certain topical drugs. A
second approach is directed to the changes in the chemistry of transported substances in the PNS by means of nerve block injection.

Recently, Virani and colleagues [48] reviewed drug interactions in neuropathic pain. They observed in clinical practice neuropathic pain patients are often treated with multiple drug “cocktails.” This leads to ambiguity about the effect of any given drug and the potential for side effects, e.g., fatigue, depression, or insomnia.

**Centrally acting agents**

Uncontrolled studies make up the majority of the available data on treatment of phantom pain in general and PTP specifically. Nonetheless, such studies often provide clues to etiology and differential diagnosis. Meanwhile, we will refer to non-PTP phantom pain studies if they may be relevant. Specific references below to treatment for PTP come from clinical experience and do not have the authority derived from controlled trials. Unfortunately, this is the only information available currently.

**Anticonvulsants (ion channel blockers)**

Carbamazipine, gabapentin, phenytoin, clonazepam, and lamotrigine have all been used and studied in the treatment of many neuropathic pains (for reviews see 49,50). Their mechanism of action is not well understood. In an adequate dose, carbamazipine is reported to be effective for more than 70% of trigeminal neuralgia patients [51-53]. If carbamazipine is effective in a patient with suspected PTP, one should consider amending the diagnosis to trigeminal neuralgia. For reasons that are still unclear, carbamazipine does not produce analgesia for PTP. Phenytoin is only rarely effective.

Although there are no randomized controlled clinical trials for PTP, gabapentin has been used successfully to treat phantom limb pain in controlled trials [54]. It is considered the drug of choice for PTP. Gabapentin’s presumed action is that of a membrane stabilizer. Gabapentin influences both axonal conduction and synaptic transmission. Its site of action may be at the abnormal peripheral nerve membrane stabilizer. Gabapentin influences both axonal conduction and synaptic transmission. Its site of action may be at the abnormal peripheral nerve or within the central nervous system.

**GABA-B receptor agonist**

Baclofen has been used in a variety of neuropathic disorders. It is helpful in PTP as an adjunct to other medications.

**Sedatives and tranquilizers**

Generally, barbiturates, nonbarbiturate hypnotics and minor tranquilizers have little analgesic activity in PTP, with one exception. Clonazepam, a benzodiazepine derivative, in doses of 1-3 mg/daily, often reduces PTP. A variety of mechanisms that include increased brain serotonin and enhanced GABA binding has been investigated to explain these analgesic effects in a variety of neuropathic pains.

**Antidepressants**

Tricyclic antidepressants (TCA) have been used for many years in the treatment of various deafferentation pain syndromes. McQuay and colleagues [49] conclude from their meta-analysis that, among patients with various forms of neuropathic pain, 30% achieved at least a 50% reduction in pain. Their efficacy is well established but their site of action is unknown [55]. Questions have been raised as to whether TCAs act as analgesics or work by altering mood. Recent evidence supports an analgesic effect [56]. Phenothiazines potentiate the analgesic effects of TCAs in PTP. In severe cases, the author of this article (JJM) has prescribed a combination of TCA and phenothiazines (i.e., perphenazine and amitriptyline, Trival) with excellent results. The clinician should monitor the patient for signs of tardive dyskinesia.

**Narcotic analgesics**

A good deal of controversy focuses on narcotic drug therapy for neuropathic pain. In his review of opioids and neuropathic pain, Dellemijim emphasizes that some inconsistencies in reported response can be attributed to a lack of agreement regarding nomenclature [57]. For example, studies including patients with AFP may include subjects with somatic pain disorders, which confounds results. At present, there are insufficient controlled clinical trials to provide a definitive opinion regarding opioid efficacy for PTP. However, because opioids are frequently prescribed as the only means of pain control, several concepts are clinically useful. Effective treatment and efficacy are not the same. ‘Efficacy’ refers to the activation of the receptor by the opioid. The balance between pain relief and side effects is referred to as ‘effective treatment.’ This implies the need for individual dose titration that targets interindividual variability [35]. Until recently, narcotics were deemed relatively ineffective in the treatment of PTP [33]. There is, however, animal experimental [58] and clinical [35,58] evidence suggesting the contrary. The authors now believe that the use of narcotics has a role in chronic PTP. Subsets of patients suffering from chronic benign pain find relief with a fixed daily dose of oral narcotic analgesic. The author (JJM) prescribes controlled-
release oxycodone, controlled-release morphine, fentanyl [59], ketamine [60], and methadone in selected cases. Patients selected for this therapy recognize that these drugs rarely result in addiction in chronic pain patients, but may result in chemical dependency. Nevertheless, patients are screened for addiction-proneness and medical suitability.

In addition, with one exception, intranasal application of drugs is just beginning to be recognized as useful. The intranasal application of cocaine has been used for analgesic purposes for more than a hundred years [61]. In a controlled double blind study, cocaine has been shown to abolish PTP temporarily. Cocaine, unlike opioids, apparently does not show affinity for specific receptor sites [62], although the mechanisms of these two properties are not necessarily related. The receptor site for cocaine’s action is unclear. In addition to its long recognized local anesthetic effects, cocaine has been reported to show central analgesic effects [63]. Moreover, a large loss of opioid binding sites has been shown to occur after deafferentation [58]. This may explain why cocaine is more effective than opioids in the treatment of PTP. The difference in analgesic effects in of the two types of drugs suggests research approaches to the etiology of PTP. Like the chronic use of opioids, cocaine possesses complex management issues that society has yet to address adequately.

Peripherally applied agents

Nerve Blocks. The author employs local anesthetic injections routinely for the relief of PTP. They are also effective when combined with steroids. Local injection of dexamethasone with various combinations and strengths of local anesthetics is clinically effective. In rats, local steroid injection to the site of nerve compression facilitated recovery of nerve conduction blockade when compared to saline injected and noninjected control groups [64].

Success rates of steroid injection appear to be dependent on two factors. First, to avoid side effects, the proper site of injection must be determined as low doses of steroid are necessary when repeated injections are contemplated. Some are at the site of the teeth intraorally and others are on the face at the terminal points of the divisions of the trigeminal nerve. Still others are at sites associated with other neuralgias, e.g., trigeminal and occipital. Repeated clinical trials help to establish the correct injection sites for each patient.

The second factor in success with steroids is early treatment, when possible. Steroids apparently best facilitate peripheral nerve recovery when the injury is fresh [64]. Recall the sequence of physiologic responses detected following injury of a peripheral nerve. After the initial shock and a brief shutdown of neural activity, the injured axon puts forth sprouts. These new sprouts differ from the parent nerve in an essential way. They readily generate action potentials either spontaneously or following mechanical, chemical, thermal or metabolic, (i.e., ischemic) stimulation. If these sprouts connect with the appropriate receptor, more stable electrical characteristics are likely to be established and the hyperexcitability state recedes [65]. Of course in the case of endodontically treated teeth, contact with the appropriate receptors is forever prevented. Deafferentation is the permanent state. Fortunately, as Wall noted, all neuropathies do not always result in pain [65].

Topical and transdermal drugs. Topical drugs are usually formulated as a liquid, cream, or gel. They are applied directly to the skin. They act by penetrating the skin and influencing peripheral tissues, including nerves, directly underlying the site of application.

Transdermal drugs are also applied to the skin, but may act at distant sites, by way of the bloodstream. Transdermal delivery systems are usually patches containing a pooled reservoir. Other drugs are directly applied to skin but act as transdermal vehicles.

Patients are often reluctant to use topical drugs and certainly transdermal patches on the face. Topical capsaicin [66,67] and clonidine applied with a transdermal patch [68] in selected neuropathic pain disorders have been tested. In clinical practice, few PTP patients have continued to use a capsaicin. Several find topical ketamine analgesic. We are presently testing drugs in a topical intraoral delivery system that permits the vehicle to stay attached to the oral mucosa, gradually releasing the drug. At present there is a lack of consistent results from many studies of topically applied drugs [66]. Nevertheless, topical and transdermal therapy has intrinsic advantages and will no doubt continue to undergo investigation.

Surgery. There are occasional indications for surgical treatments for PTP. A thorough appraisal of surgical treatments is not a chief priority of this article.

It follows logically that if local anesthetic blockade of a nerve relieves pain temporarily, surgical interruption at the same site, or central to it, would yield lasting relief. Clinical experience has amply demonstrated, quite to the contrary, that the re-
verse is more often than not the case [69]. The literature on this subject is too vast to review here; however, a brief summary will be attempted.

Many neurosurgical and dental approaches have been tried on PTP, mostly with extremely poor results. Rawlings and Wilkins [17] reviewed the neurosurgical treatment of pain syndromes of the trigeminal system. Their findings were not encouraging. Clinical experience suggests that postneurosurgical patients make up the most recalcitrant group of facial pain patients.

The most common treatment for PTP is further endodontic therapy followed by apicoectomy and tooth extraction (Fig. 1). The logic is apparent to any clinician familiar with endodontics. There are two patterns to the clinical histories of PTP cases. In the first case, a dentist or dental specialist examines 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 mistaken belief that the pain is of dental origin. In the second scenario, routine endodontic treatment is performed expertly but pain persists or is worse 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 sequella of this approach to pain management. Clinicians who consider PTP in the differential diagnosis of unusual dental pain complaints will participate in fewer of these two scenarios.

**Conclusion and Future Research**

The specific purpose of this article is to review current knowledge on PTP based on clinical observation. Beside the obvious need for controlled clinical trials, one other major information gap is apparent. There is a need to refine further the diagnostic criteria for PTP. As Merskey [17,19] emphasizes, the evolution of a taxonomic system is a work in progress. However, before progress can be made in refining criteria for a specific disorder, a critical mass of clinicians and researchers must bring their attention to the problem. The first step is recognition that the disorder exists, is a distinct one, and can be identified reliably. This is why we have presented newly revised criteria for PTP. Our hope is that others will investigate and improve both the taxonomy and treatment of PTP. As these criteria are applied in practice, further revisions and specifications will likely follow.

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