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Persistent dento-alveolar pain disorder (PDAP): Working towards a better understanding

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KEYWORDS

Non-odontogenic tooth pain, chronic tooth pain, atypical odontalgia, phantom tooth pain, review, diagnosis, epidemiology, mechanisms

SUMMARY POINTS

- New terminology, persistent dento-alveolar pain disorder (PDAP), and diagnostic criteria have been put forward to address the shortcomings of existing nomenclature, which are associated with unclear criteria.
- Arriving at an accurate diagnosis of PDAP is based on excluding other possible aetiologies, and may involve different care providers.
- Synthesis of published data suggests that PDAP has a frequency of occurrence following root canal therapy of around 1.6%.
- The putative risk factors involved in PDAP are largely unknown, but seem to be similar to those being identified with other post-surgical chronic pain disorders.
- The underlying mechanisms involved in the development of and/or perpetuating PDAP are unknown and the approach to treatment remains empiric in nature.

Introduction

Pain in the orofacial region is very common to the human condition, and tooth-related pain is the most prevalent of such pains (1). Most often this pain is a symptom of dental disease and as such is effectively addressed by dental professionals, as reviewed in this journal (2). However, persistent pain perceived in teeth or adjacent dento-alveolar tissues may occur without any readily identifiable local dental aetiology; this pain can be referred to as non-odontogenic 'tooth' pain (3).

Non-odontogenic pain presents a complex problem for care providers for two reasons: Firstly, the challenge to arrive at an accurate diagnosis for the symptom of intraoral pain, and secondly the subsequent ability to provide an effective treatment for such pain – which is highly predicated on the success of the first step. Despite the fact that chronic non-odontogenic pain has been observed and reported in academia for centuries (4), treatment for this disorder continues to be empiric and often involves at some point a deafferenting dental procedure, such as root canal therapy or tooth extraction (5-7).

The purpose of this narrative is to review current literature regarding persistent dento-alveolar pain in order to i) highlight recent developments in the field and ii) identify areas requiring further research, with the ultimate goal of improving patient care. We will point out the challenges in the definition of the disorder and that the research community has now reached an accepted case

definition. Given various previous case definitions, we will describe how frequent the disorder is, present which risk factors have been explored, and describe the mechanisms suggested to be involved. Finally, we present an overview about patient management.

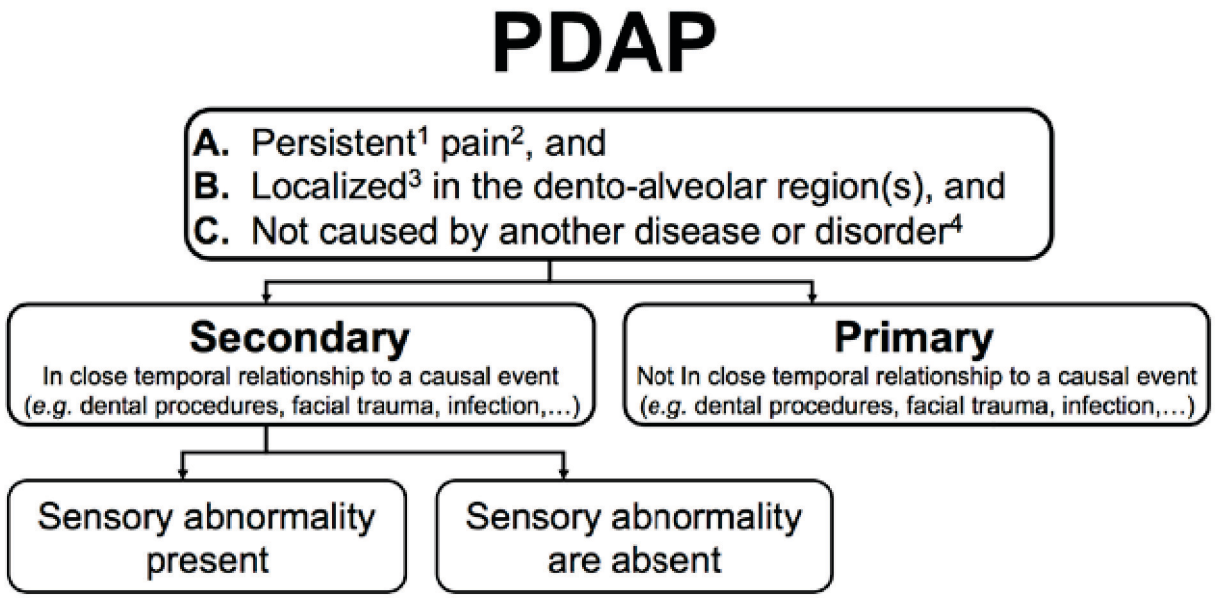
Difficulties with diagnosing PDAP

Problems within pain taxonomy

At present, diagnosing chronic pain disorders is based mainly on clinical signs and symptoms, since the mechanisms underlying the pathophysiological processes are largely unknown. This also applies to conditions that present as chronic pain within the orofacial tissues. Unfortunately, except for temporomandibular disorders (TMD), there is a lack of research assessing the validity of such diagnoses and classification systems. It is therefore unclear how various disorders relate to each other, since there is no over-arching taxonomy, and this is likely to perpetuate discipline-based diagnostic thinking.

The absence of taxonomical data does not however imply an absence of knowledge or theory about taxonomy of orofacial pain conditions. An international collaboration, building from the experience of TMD diagnostic research, has attempted to conceptualise how various orofacial pain disorders may be related and therefore most appropriately defined, with a view to leading eventually to a functional taxonomy (see http://www.rdc-tmdinternational.org/default.aspx).

One group chose to address the topic of non-odontogenic 'tooth' pain as a working example. This group applied ontological principles to the classification of one type of orofacial pain disorder, using the descriptive name persistent dento-alveolar pain (PDAP), and produced initial diagnostic criteria (Figure 1)(8). The key concepts behind this effort were to specify unambiguous terminology without conflations, and to produce diagnostic criteria that could then be tested and refined in the future. The aim was to improve the clinical phenotyping of the disorder so that more extensive epidemiological research and more accurate mechanism-based research could follow. All members of the group readily admitted that the criteria were expert-derived and not evidence-based, and as such were less than ideal. The main reasons for opinion-based criteria included lack of consensus in the literature, and the lack of research data to support previously proposed criteria. Therefore the expert panel's proposition was thought to be the best next step towards garnering discussion on the topic and developing a workable taxonomy.



Criteria
1 Persistent - meaning pain present at least 8 hours/day ≥15 days or more per month for ≥3 months duration
2 Pain - as defined as per IASP criteria (includes dysaesthesia)
3 Localized - meaning the maximum pain defined within an anatomical area
4 Extent of evaluation non-specified (dental, neurological exam +/- imaging, such as intra-oral, CT and/or MRI)

Figure 1 Diagnostic criteria for PDAP

Multiple terms and various diagnostic criteria

Intertwined with the problems involving ontology is the multiplicity of classification systems proposed to define chronic orofacial pain perceived intraorally (9-12). The different terms and varying criteria provide great confusion among clinicians and scientists alike, as well as fodder for disagreement. A previous article in this journal provided the background for all chronic orofacial pain conditions and presented the most widely accepted classification (13). The author presented the groups of orofacial pain conditions and used the term 'persistent idiopathic facial pain (PIFP)' to classify PDAP, which is an apt description that highlights previous thinking. Dental professionals seem to agree that there is a distinct clinical entity that patients seek care about, that has a chronic continuous pain symptom located in the dento-alveolar region that cannot be explained within the context of other diseases or disorders (8). The list of diseases or disorders that need to be ruled out include local or adjacent dental pathosis, referred pain from regional structures (e.g. sinus (14), muscles of mastication (15), heart (16), vascular (17) or brain (18)), or headaches presenting in the orofacial region (CN V2 & V3 distribution (19) as opposed to fronto-orbital V1 or parieto-occipto-cervical (cervical 1-3) distribution).

Looking through the literature one can get a sense that many different terms have been used to describe this clinical scenario, such as: atypical odontalgia, phantom tooth pain, neuropathic tooth pain, and also as a subgroup within persistent idiopathic or atypical facial pain to name a few. Even though it is unlikely that all these terms refer to the same disorder characterised the same way, it seems safe to assume that these conditions share more commonalities than not, hence the move to rename the entity to PDAP (8).

Practical considerations regarding the diagnostic process

Diagnosis of PDAP is dependent of the ruling out of all other potential sources for the symptom of pain (i.e. referred pain from another site) and other pain disorders with such an anatomic presentation (i.e. TMD, headache disorders). Therefore the process to arrive at a diagnosis of PDAP includes more than a traditional dental evaluation and intraoral imaging. Given this, and the low sensitivity of dental radiographs, some researchers have assessed the diagnostic yield when three-dimensional radiography is used to image the dento-alveolar region involved in PDAP pain. The authors reported that the addition of advanced imaging improves the ability to ascertain the absence of local bone destruction (20), something that would exclude the presence of local disease and suggest that dental interventions are not indicated.

Besides local disease interacting with the primary afferent neurone resulting in pain, chronic orofacial pain mimicking PDAP has been reported to be referred from intracranial structures (18). A case-series of brain MRIs taken of a mixed group of chronic facial pain revealed that 7 of the 38 patients imaged (18%) had structural lesions impinging on the 5th cranial nerve (21). This is not a trivial proportion, and has prompted the authors of this article to routinely obtain brain MRIs on their patients, resulting in the anecdotal experience that between 5 and 10% of PDAP patients have similar structure lesions.

How big is the problem, and who is at risk of getting it?

In general, epidemiological evidence for PDAP is largely unknown. Given that local dental disease, sensory perception of pain, and presence of irreversible treatments are inexplicitly related, recent research on this topic has focused on the combined presentation of these three factors within the provision of root canal therapy. This seems like a reasonable initial approach to exploring PDAP since root canal therapy is a common procedure with over 20 million performed every year in the United States (22). Furthermore, root canal therapy is provided for patients with diverse characteristics (i.e. age, gender, socioeconomic status, health status) and thus allows for the exploration of a number of risk factors in a broad patient population.

Research on the prevalence of PDAP

Evidence of the true prevalence of PDAP is still lacking since all known studies have used convenience sampling from clinical populations, which is not representative of the population at large. One could argue that this is the appropriate strategy for such research given that: a) PDAP is at present intertwined with the pre-existence of local disease, the symptom of pain, and the provision of deafferenting procedures; and b) such a sampling strategy recruits a high percentage of those seeking care. Initial reports, their limitations notwithstanding, suggested that PDAP is not an uncommon outcome following root canal therapy with historic estimates ranging from 2.5 to 3.1% (23,24).

Table 1 Prevalence data abstracted from available articles that clinically confirmed diagnosis of PDAP

Author	Year published	Total sample enrolled	Follow up rate (%)	Clinical cases of PDAP	Frequency of PDAP (%)
Marbach et al (24)	1982	732	70	8	1.1
Campbell et al (23)	1990	118	100	6	5.1
Pollmann (26)	1993	2,620	100	25	1.0
Jacobs et al (28)	2002	500	35	8	1.6
Polycarpou et al (29)	2005	400	44	21	5.3
	Totals	**4,370**	**82**	**68**	**1.6**

A recent systematic review assessing the presence of non-odontogenic pain six months or longer following root canal therapy, to give an upper limit estimate for PDAP, found that 3.4% of patients reported pain that was unexplained by local disease (25). Given that most studies were not designed specifically to capture PDAP and other non-odontogenic pain, such as referred muscle pain and headache disorders presenting in the dento-alveolar region, this is likely to be a high estimate and can be considered an upper limit.

Since these early articles by Marbach et al (24) and Campbell et al (23), there have been three articles investigating the frequency of PDAP (see Table 1). Pöllmann, in 1993, published an article in which he evaluated people prior to employment (26). Consistent with the idea of sensory disturbances following deafferentation as a consequence of limb amputation (27), he identified 44 people with such alteration in feeling, of whom 25 had a painful component and 19 reported no pain. Since 2,620 people of the entire sample (N=3,126) had a missing tooth, they were considered the 'at risk' group since the others had not undergone a deafferenting dental procedure and therefore presumably did not have the combined presentation of dental disease and interventional treatment.

Jacobs et al in 2002 reported on 500 dental patients treated with either root canal therapy or tooth extraction (28). Of the 176 questionnaires returned they identified 10 with altered perception in the treated dento-alveolar area that was clinically evaluated; 8 were painful and 2 were painless.

Polycarpou et al in 2005 reported on 400 dental patients receiving tertiary endodontic care (29). This prospective study followed up 175 patients, and identified 12 as having PDAP.

All these studies had limitations, including omissions of reported data (i.e. baseline pain status, duration of symptoms, dental diagnoses) and/or significant loss in follow up. Nonetheless, pooling the data across those studies allowed us to estimate the frequency of occurrence of PDAP. Using a conservative calculation, which is the total of clinically determined cases of PDAP (68) divided by the total number of patients enrolled and receiving care (4,370), an estimate for occurrence of PDAP in these five studies was calculated to be 1.6% (Table 1). Given the frequency of root canal therapy being performed, this is not an insignificant number of patients experiencing this painful outcome.

Risk factors for the development of PDAP

As expected, given the few epidemiological studies reporting on PDAP, the understanding of risk factors involved in the development of this chronic pain state are lacking. Only one study calculated odds ratios and it found that extended duration of pre-operative pain, presence of other chronic pain problems, female gender, and a history of painful treatment in the orofacial region are statistically significant risk factors for PDAP following root canal therapy (29). These are interesting findings, especially in the face of a selected population sample with more than 50% loss to follow-up, because they are consistent with risk factors observed in other surgical procedures that assessed for the outcome of persistent pain (30-33). Exploring the long-term outcome of patients with PDAP, one study followed a cohort over a 9 to 19 year period and reported that 10 of the 45 (22%) subjects followed did not report orofacial pain (34). The authors further reported that patients continuing to obtain dental interventions faired worse than those who did not, but potential diagnostic misclassification in this study could hinder the certainty of such an inference. Clearly, more epidemiological research is needed to understand basic questions on putative risk factors for PDAP.

Proposed mechanisms underlying PDAP

As a consequence of the lack of adequate epidemiological data that identify the causal pathway(s) involved in PDAP, existing research into the systems underlying the disorder has been based on clinical observations. Studies have therefore focused on the phenomenology of PDAP, exploring the presumed mechanisms that largely investigate either a psychological aetiology (35) or neuropathic aetiology (36). Besides the long-term goal of elucidating mechanisms, which this approach cannot fully realise until employed prospectively, this research approach has value in improving the characterisation of the disorder such as identifying intermediate or endophenotypes.

Psychosocial factors

In the last decade there have been three articles published that assessed psychosocial factors in patients with PDAP, all employing a case-control study design (28,37,38). The first study used the SCL-90 questionnaire to assess this domain for ten cases plus ten matched controls. They found no differences with individual scales between those with pain and their pain-free controls, but when combined as an assessment of overall psychological distress slightly higher values were observed in the group with PDAP (28).

The second study used the SCL-90 and the SF-36 questionnaires to assess 46 cases plus 35 matched controls (37). With the SCL-90 they observed a significant difference in both somatisation and depression domains, and with the SF-36, four of the eight domains were found to be significantly different from pain-free controls (bodily pain, role-physical, social functioning, and vitality).

The third study was restricted to patients being referred from psychiatric facilities and compared patients with PDAP to those with burning mouth syndrome (BMS), but with no pain-free controls (38). Of the 36 patients with PDAP and pre-existing records, the referring clinic had given the following diagnoses: 19 (53%) had a somatoform disorder, 8 (22%) a mood disorder, and 14 (39%) had no psychiatric diagnosis. Given that all three studies assessed psychosocial variables in patients with PDAP that had been experiencing intra-oral pain for some extended period of time, it is impossible to draw a conclusion regarding causation. Furthermore, over one third of PDAP patients not receiving a psychiatric diagnosis suggest that their symptom of pain may result in increased rating on these non-specific questionnaire-based instruments. This notion is partly supported by a follow-up study that compared the same 46 PDAP patients with a cohort of patients with TMD pain and

found no difference in the psychosocial domains between these two groups of patient with orofacial pain disorders (39). The inconclusive data assessing psychosocial factors in relationship to PDAP prevents any conclusions regarding their role in the development and/or maintenance of this chronic pain disorder.

Neuropathic factors

The category of potential neuropathic factors is vast and includes a wide range of possible peripheral and central mechanistic changes that have been hypothesised to occur with peripheral nerve injury (40). To date the various articles investigating the potential differences in the somatosensory function between cases of PDAP and pain-free controls can be grouped in studies assessing: i) the psychophysical response to a variety of presented stimuli, and ii) the pain response to a pharmacological challenge. Six articles have explored the psychophysical to some degree, ranging from the application of a single provoking stimulus (41,42,43) to the systematic application of a battery of stimuli (28,37,44) applied in an attempt to characterize those with PDAP (45). Baad-Hansen and colleagues observed no differences in blink reflex, a measure of trigeminofacial brainstem function, between the affected and pain-free sides; it was not altered by the application of capsaicin (41,42) but was delayed when compared to matched pain-free controls (42). Moana-Filho and colleagues observed that a dynamic pressure pain stimulus over dento-alveolar tissues evoked greater pain in cases of PDAP compared to matched pain-free controls and had significant discriminative ability in separating cases from controls (area under a ROC curve of 0.99) (43).

Zagury and colleagues observed that the after-stimulus sensation of pain following application of cold to the alveolar mucosa was significantly longer for cases of PDAP than controls and that the test was able to differentiate these groups (44). The electrical pain threshold was elevated for both sides in cases of PDAP, while electrical detection, warm detection and heat pain detection thresholds of the face were similar (44). Other researchers observed increased sensitivity to light-touch in PDAP cases, but not to two-point discrimination or thermal discrimination intra-orally, suggesting only A-beta fibre hyper-function at the site of pain (28).

List and colleagues observed a range of hypersensitivity, hyposensitivity, and normal sensitivity in cases of PDAP compared to matched controls with multiple stimuli and found minimal group-wise differences (37). When comparing the absolute difference between sides, these researchers observed greater variability in PDAP cases for light-touch threshold, pin-prick pain threshold, pressure pain threshold, wind-up ratio, dynamic mechanical allodynia, and vibration detection threshold but not warm detection, heat pain detection or cold detection thresholds (37). Despite the modest number of studies involving psychophysical stimuli and the variety of stimuli applied, their results suggest that PDAP cases present greater amounts of intra-group variability and increased pain evoked by supra-threshold stimuli. Assessments of different challenges to pharmacological agents revealed a modest increased pain response to capsaicin (42), lack of pain reduction to fentanyl or ketamine (46), and mixed pain reduction to injected local anaesthetic agents (44) – even when administered in a double-blind randomised fashion (47). Given the variation in these study findings, it seems that PDAP may very well involve several different psychophysical characterisations, similar to that being observed in other chronic pain conditions, and as such warrants a profiling approach to its characterisation (48).

Patient management and treatment

Diagnosis of PDAP is not straightforward, given the possibility of multiple different tissues being involved and the interaction with psychosocial variables. Expert opinion suggests an inter-disciplinary approach to the management of patients with PDAP similar to other chronic pain conditions, which includes diagnostic work-up and treatment implementation (49-53). Furthermore, experts agree that earlier recognition and initiation of therapeutic modalities is more beneficial than delayed diagnosis and treatment (54,55). Given the uncertainty of the mechanisms, diagnosis, and treatment of PDAP, these ideas seem reasonable but remain to be substantiated with evidence.

A recent review of treatments for neuropathic orofacial pain conditions did not find any randomised controlled trials for conditions consistent with PDAP and ultimately concluded: 'Based on available evidence from other neuropathic pain conditions, TCAs or gabapentin would be the first drugs indicated' (56). Case-series data suggest that some patients may respond to peripherally applied medications (57-59) and several sets of observational data recommend against interventional procedures, such as root canal therapy or extraction (34,60,61). Together, it seems that the best course of action is to follow the NICE guidelines for the treatment of neuropathic pain with oral medications (62), try topical medications when practical, and avoid irreversible treatments that involve local tissue injury. From a clinician's perspective, as well as someone suffering from PDAP, this scant data regarding treatment is unacceptable, difficult to admit, and yet not surprising.

Conclusions

Confusion in diagnostic criteria and terminology prompted the recent development of consensus-driven diagnostic criteria for persistent-dento-alveolar pain disorder (PDAP). The diagnostic process may involve multiple care providers to rule out various conditions that may refer the symptom of pain to the dento-alveolar region; the diagnosis of PDAP is in essence one of exclusion. Due to the often presentation of pre-existing dental disease, frequent report of pre-operative pain sensation, and surgical deafferentation as treatment to address both, the outcome of PDAP following root canal therapy needs to be cautiously interpreted. The frequency of prevalence for PDAP associated with root canal therapy is estimated to be around 1.6%, but methodological issues limit the validity of these results. Initial epidemiological research suggests that similar risk factors are involved with the development of PDAP as with other post-surgical chronic pain disorders. The mechanisms underlying the disorder are largely unknown, with psychosocial and neuropathic

factors being the most commonly explored, whilst treatment at present time remains to be based on empirical evidence.

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Orofacial Pain (Part two) Multiple Choice Questions

Burning mouth syndrome (BMS)

More than one answer may be correct. Select all that apply

1 How long must symptoms be present to allow diagnosis of BMS?

a) 3 weeks
b) 3 months
c) 6 weeks
d) 6 months
e) 12 months.

2 Which of the following is the most likely pain characteristic of BMS?

a) Elicited pain with spicy foods
b) Intermittent neuralgia
c) Constant dull ache
d) Awakening at night
e) Cold allodynia.

3 Which of the following is the most commonly used management of BMS in the UK?

a) Amitriptyline
b) Pregabalin
c) Nortriptyline
d) Baclofen
e) Citalopram.

4 What medication regime is recommended?

a) Dose at 10mg nocte for 3 months
b) Step up weekly (nocte) 10mg, 20mg, 30mg, 40mg maintenance at highest possible dose for 12 weeks
c) Dose at 20mg nocte for 3 months
d) Step up weekly (nocte) 20mg, 40mg, 60mg maintenance at highest possible dose for 12 weeks
e) Dose at 30mg nocte for 2 months.

Primary headache disorders: Focus on migraine

More than one answer may be correct. Select all that apply

1 Which of the following are considered appropriate acute treatment choices in the management of migraine?

a) High dose aspirin
b) Rizatriptan
c) Tramadol
d) Low dose aspirin
e) Co-codamol.

2 Which of the following are first line preventative treatments in the management of migraine?

a) Amitriptyline
b) Topiramate
c) Gabapentin
d) Sodium valproate
e) Coenzyme Q10.

3 Which acute treatment regimens are unlikely to cause medication-overuse headache?

a) Ibuprofen 200mg od daily
b) Ibuprofen 800mg bd twice a week
c) Ibuprofen 400mg od four times a week
d) Ibuprofen 600mg od once a week
e) Ibuprofen 800mg tds twice a week.

4 With which of the following can aura be experienced?

a) Tension-type headache
b) Cluster headache
c) Migraine
d) No headache
e) Hemicrania continua.

5 Which symptoms can typically be experienced in association with a migraine attack?

a) Neck pain
b) A need to remain active during the attack
c) Hunger
d) Sensitivity to chocolate
e) Yawning.

Persistent dento-alveolar pain disorder (PDAP): Working towards a better understanding

Select the most correct response

1 To render a diagnosis of a non-odontogenic chronic pain disorder presenting intraorally, the clinician(s) needs to rule out:

a) pulpitis and apical periodontitis associated with teeth in the region
b) headache disorders with the symptom of pain perceived peri-orally
c) intra- and extracranial lesions that may impinge upon or alter trigeminal somatosensory system
d) trigeminal neuralgia
e) all of the above.

2 The aetiology of PDAP is not known, but the clinical condition observed is often a product of:

a) pre-existing dental disease
b) post-procedural chronic pain
c) idiopathic chronic pain
d) all of the above
e) none of the above.

3 The current estimates for the frequency of non-odontogenic 'tooth' pain and PDAP associated with root canal therapy are:

a) 10% and 2%
b) 15% and 5%
c) 5% and 2%
d) 3% and 1.5%
e) 5% and 1.5%.

4 The putative risk factors involved in PDAP are largely unknown. Limited exploration has been published, involving which system-based mechanisms?

a) Psychological mechanisms
b) Neuropathic mechanisms
c) Inflammatory mechanisms
d) Two of the above
e) None of the above.

5 The underlying mechanisms involved in the development of and/or perpetuating PDAP are unknown, therefore once a diagnosis is established the best course of treatment is:

a) prescribe non-steroidal anti-inflammatory and antibiotic medications
b) extract the tooth closest to the painful area
c) surgical exploration of the involved area
d) prescribe the newest anti-epileptic drug available that the patient has not heard of
e) follow the most recent guidelines for idiopathic chronic pain management.

MCQ answers
Burning mouth syndrome
1 d
2 c
3 c
4 b

Primary headache disorders: Focus on migraine
1 a and b
2 b and d
3 b, d and e
4 a, b, c, d and e
5 a, c and e

Persistent dento-alveolar pain disorder (PDAP): Working towards a better understanding
1 e
2 d
3 d
4 d
5 e