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INVITED REVIEW

The painful tooth: mechanisms, presentation and differential diagnosis of odontogenic pain

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Abstract

Pain arising for the teeth and supporting structures is a very common complaint, affecting around 9% of adults in the UK, and it can sometimes be difficult to determine the exact cause. In this narrative review, we explore the underlying neurophysiology of odontogenic pain and describe the relevance of this to clinical practise. We discuss characteristic features of pain arising from the various dentoalveolar structures and common oral disorders, and consider that non-odontogenic pain may occasionally present as toothache. As well as describing current approaches to reaching a reliable diagnosis, we also discuss some innovative techniques and potential future developments in this area.

Introduction

Toothache is very common, with 9% of adults in the UK reporting pain from their teeth[1], and this has the potential to impact significantly on quality of life. There is also an impact to society as a result of time away from work and education, as well as lost productivity for sufferers[2,3]. In patients experiencing odontogenic pain, sensory neurones of the trigeminal nerve transmit signals from injured or inflamed tissues resulting in the experience of painful symptoms. The tissue most commonly responsible for pain of odontogenic origin is the dentine–pulp complex, often as a result of dental caries, followed next by pain arising from the periodontium. Although diagnosis may often be straightforward, unpicking the cause can sometimes pose a diagnostic conundrum. In this article, we explore the mechanisms of odontogenic pain, discuss typical presentations and propose a rational approach to accurate diagnosis; finally, we discuss some novel and innovative approaches to diagnosis.

Applied neurophysiology

Dental afferent nerve fibres, whose cell bodies reside within the trigeminal ganglion, enter the tooth via the apical foramina. Their signals are carried from the pulp via the mandibular and maxillary divisions of the trigeminal nerve to the brainstem where they synapse with second-order neurones in trigeminal nuclei. The fibres cross the midline and ascend to the thalamus where they synapse with third-order neurones which project to higher brain centres. Signals from the majority of afferent nerve fibres within the pulp are perceived as pain[4,5]. In addition to these afferent fibres, there are also a number of sympathetic efferent fibres which are thought to play a role in hemodynamic control by producing vasoconstriction[6]; parasympathetic fibres are yet to be observed. The periodontium contains numerous mechanoreceptors which contribute to the sensation of pressure and vibration, thus allowing proprioception during mastication[7,8]; as a result, pain arising from the periodontal tissues is fairly well localised. Conversely,

the pulp has a paucity of such receptors and thus pain arising from the pulp is poorly localised.

Pulpal nerve fibres are largely of two types: myelinated, fast-conducting A-fibres which are present in the periphery of the pulp and inner dentine along with the odontoblast processes (Fig. 1); and the smaller diameter, unmyelinated C-fibres which are more numerous in the body of the pulp and have slower conduction velocities[9]. A-fibres primarily respond to stimuli such as heat, cold, desiccation and direct mechanical stimulation of exposed dentine[10], and the signals transmitted by these fibres usually produce pain which is rapid in onset and sharp in character[11]. Although a number of A-fibres terminate in the radicular part of the tooth, many more form a dense plexus below the odontoblast layer (plexus of Raschkow; Fig. 1), and their endings project some distance into the dentine tubules[12]. Movement of fluid within dentine tubules caused by drying, changes in osmotic pressure and temperature change are thought to produce action potentials in A-fibres innervating the innermost part of the dentine through the activation of mechanosensitive ion channels[13]. Another family of ion channels however – thermo-sensitive transient receptor potential channels (thermo-TRPs) – which are present in dental primary afferent fibres and odontoblasts are now known to contribute to the sensation, and transduction into pain, of hot and cold stimuli[14–16]. It has been suggested that the experience of pain via exposed dentine may therefore involve both the transduction of mechanical stimuli and direct activation of thermo-TRPs, perhaps involving an interaction of both afferent fibres and odontoblasts[17]. C-fibres are polymodal and respond to intense heat and cold and the presence of inflammatory mediators such as histamine and bradykinin[18–20]. Stimulation of C-fibres produces a deeper, duller, aching sensation which is poorly localised, and fibres are less responsive to direct mechanical stimulation[19].

Stimulation of pulp afferent fibres causes the release of several peptides from the neurone such as calcitonin-gene-related peptide (CGRP), substance P and neurokinin A[21,22]. These substances cause vasodilation within the pulp and the release of inflammatory mediators from pulp fibroblasts, attracting immune cells in a process called neurogenic inflammation[23,24]. As inflammation proceeds, inflammatory mediators such as histamine, bradykinin, prostaglandins and leukotrienes are released which reduce the thresholds required for nociceptors to generate action potentials; as a result, there is an exaggerated and prolonged response to painful stimuli known as hyperalgesia, and stimuli which would not usually be painful in the healthy tooth produce intense pain – a phenomenon termed allodynia. One mechanism by which this occurs is the sensitisation of thermo-TRPs by various inflammatory mediators, producing thermal hyperalgesia[25]. The voltage-gated sodium channels (NaCh) responsible for initiation and propagation of neuronal action potentials also change during pulpal inflammation; for example,

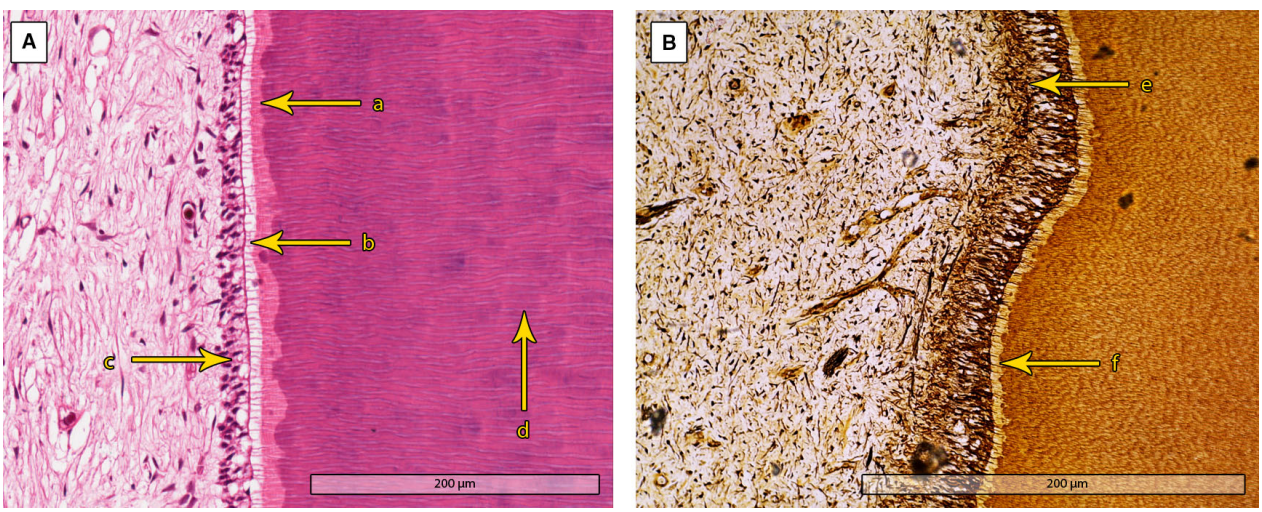

Figure 1 Demineralised histologic sections of human tooth stained with haematoxylin and eosin (A) and silver stained (B, neural tissue stained dark brown) showing the dentine–pulp interface. (a) Odontoblast processes projecting into predentine layer; (b) Odontoblast processes visualised by separation of odontoblast layer from dentine–pulp interface during processing; (c) Odontoblast layer; (d) Dentine, showing dentinal tubules; (e) nerve plexus of Raschkow in the periphery of the pulp, closely related to odontoblast layer; (f) nerve fibres extending into predentine layer.

one type of NaCh involved in pain sensation, $Na_v1.7$, becomes more numerous in the painful pulp, potentially leading to greater sensation of pain[26]. Additionally, nerve fibres sprout new branches, increasing the area from which they receive sensation[27], and fibres which are usually dormant in the healthy pulp begin transmitting signals. The result of these processes is that nociceptors become more sensitive, and the experience of pain becomes more intense and prolonged – this is termed peripheral sensitisation. In addition, central sensitisation of second- and third-order neurones occurs in the brain and brainstem[28], causing an increased perception of pain and pain spreading over a larger area over time[29].

Although the A-fibres responsible for pain transmission are classically assumed to be Aδ-fibre nociceptors, in 2011 Fried, Sessle and Devor proposed the concept of the low-threshold algoneuron[30]. Their reasoning was that light mechanical stimulation (which produces pain in exposed dentine) does not usually result in pain that in other areas of the body, as this does not stimulate the high-threshold Aδ and C-fibre nociceptors found in these tissues. To explain this apparent paradox, they proposed the presence of low-threshold mechanoreceptors (LTM) in the pulp, with conduction velocities in the Aβ range. These fibres normally encode the sensation of touch elsewhere, and may contribute to the perception of pain in the pulp; this concept appears to be consistent with the evidence, as many pulpal afferent neurones have histological appearances typical of LTMs and express nociceptive neurotransmitters at the level of the trigeminal ganglion[30].

Pain associated with pulpitis

The term 'pulpitis' describes inflammation of the pulp, and although inflammation may be present without pain[31], pulpitis is usually painful. The classical distinction of pulpitis as 'reversible' where symptoms are transient and do not linger, or 'irreversible' where they are persistent or spontaneous[32] is a useful heuristic which is widely used clinically; this clinical distinction does not always match the histological status of the pulp however, or its ability to maintain vitality after treatment[31,33]. A number of authors have suggested that a proportion of teeth with 'irreversible pulpitis' may have the potential to regenerate, and have suggested the need to reconsider how pulpitis is classified clinically[34–36]. One proposed classification system[36] favours 'mild pulpitis', where there is a heightened and lengthened response to cold testing but no spontaneous symptoms; 'moderate pulpitis' where symptoms are prolonged and occasionally spontaneous; and 'severe pulpitis' where there is clear spontaneous pain and prolonged pain to warm and cold, with possible pain to percussion and lying down; this system is, however, yet to be clinically validated and widely adopted. Despite the usual assumption that teeth with 'irreversible pulpitis' require conventional endodontic treatment or extraction, consensus is shifting in favour of minimally invasive endodontics, and vital pulp therapies such as pulpotomy for teeth with symptoms suggestive of inflammation of the coronal pulp, or with deep caries, with the aim of preserving pulpal integrity[34,35,37–39]. Despite this more conservative treatment, some teeth may undergo 'irreversible' degenerative changes requiring conventional endodontic treatment or extraction, and in some cases this may occur without the patient experiencing painful symptoms[39].

The cause of pulpitis in most cases is bacterial invasion arising as a result of caries; however, bacterial ingress may occur during placement of deep restorations (Fig. 2), dentoalveolar trauma or secondary to extensive attachment loss in periodontitis, where extra-radicular biofilm may reach the apical area allowing bacteria to enter the pulp via the apical foramina. As caries progresses through the enamel and then into dentine, bacterial byproducts diffuse towards the pulp through dentinal tubules resulting in inflammation of the coronal pulp[40]. There is subsequent increased vascularity, and the activation and sensitisation of A-fibre nociceptors, causing sharp pain on stimulation. Changes in NaCh expression and sensitisation of thermo-TRPs as discussed may contribute to thermal hyperalgesia at this stage. As caries progresses to include the full width of dentine, bacteria invade the pulp and severe inflammation ensues with areas of necrosis[41,42]. As inflammation reaches the centre of the pulp, more C-fibres are affected and the spontaneous, intense, lingering pain typical of severe pulpitis develops. Severe pulpitis is more likely to produce C-fibre-mediated pain because these fibres are more tolerant of the tissue hypoxia found in the severely inflamed pulp than A-fibres[19,20]; C-fibres therefore remain active longer in the degrading tissue. Because of the limited proprioceptive capacity of the pulp and the fact that single afferent nerve fibres may branch to serve multiple teeth, pain associated with pulpitis is typically poorly localised[43].

Clinical diagnosis clearly cannot be based upon histological examination of the pulp, as in most cases

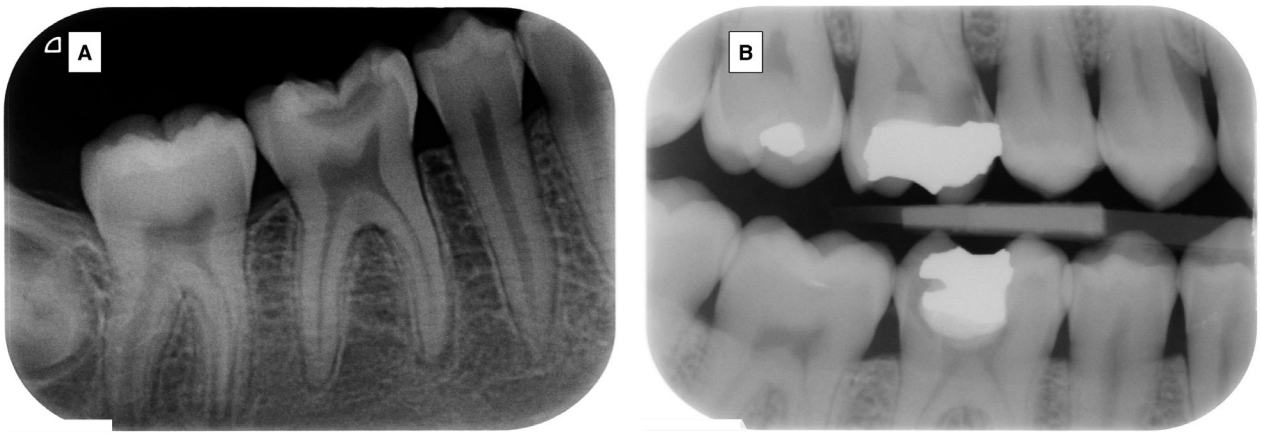

Figure 2 (A) Periapical radiograph showing deep occlusal caries in the lower right first molar; pulpal pain as a result of pulpitis may be expected in this tooth. This may be mild, and resolve following management of caries, or severe and require conservative or conventional endodontic therapy. (B) Bitewing radiograph showing extremely deep secondary caries beneath the mesial surface of a restoration in the upper right first molar, and a deep restoration in the lower right first molar; pulpal pain as a result of pulpitis is likely, and conservative or conventional endodontic treatment may be required.

the aim of treatment is to preserve the tooth wherever possible; therefore, proxy measures are used to arrive at the most likely pulpal status (Fig. 3). These tests are far from infallible[33] and the reliability of sensibility testing may be variable, particularly in heavily restored teeth or those with calcified pulp chambers[44]. Although the degree of pulp inflammation does not reliably correlate with the severity of painful symptoms, application of a causative stimulus (e.g. hot/ cold) to a specific tooth is more likely to result in transient symptoms where the pulp is mildly inflamed, and spontaneous or lingering symptoms are less likely. In severe pulpitis, there is more likely to be pain at rest, and hyperalgesia and allodynia may occur as a result of initially peripheral, and then later central, sensitisation – producing a prolonged and more pronounced painful response to application of provoking stimuli during sensibility testing (cold, heat, electrical, biting), and pain to stimuli which would normally be innocuous.

Pulpal pain attributed to hypersensitivity

Pulpal pain attributed to hypersensitivity can be defined as pain occurring in association with a clinically normal pulp, and is a diagnosis included in the recently published International Classification of Orofacial Pain (ICOP)[45]. Hypersensitivity occurs due to stimulation of pulpal nociceptors as a result of exposed dentine, for example due to gingival recession, tooth wear (Fig. 4) or fractures of teeth.

The prevalence of hypersensitivity on the basis of clinical examination ranges between 1 and 42% depending on the population studied[46–51], and is strongly associated with the presence of erosive tooth wear, clinical attachment loss, acid reflux and frequent vomiting[48]. The most commonly accepted mechanism of hypersensitivity is the hydrodynamic theory whereby desiccation of the dentine surface, warm, cold, sweet and mechanical stimuli cause an increased flow of fluid through open dentinal tubules in exposed dentine[52–54]. This fluid flow generates action potentials in A-fibres within the peripheral pulp and inner dentine by opening mechanosensitive ion channels in these neurones[55,56], resulting in sharp, transient pain. Recent evidence has identified the expression of mechanosensitive ion channels and receptors by odontoblasts, which along with evidence supporting signalling between odontoblasts and pulpal neurones suggests that odontoblasts may play a role in pain transduction from dentine stimulation[12,57].

The pain of hypersensitivity may be poorly localised due to the lack of pulpal proprioception and the fact that multiple teeth in the same area may be affected. It can be hard to differentiate pulpal pain caused by hypersensitivity to that of pulpitis, however in the former there are rarely symptoms consistent with C-fibre activity such as prolonged, aching pain, and other than exposed dentine there may be no other pathology which would be expected to cause pain. Clinical experience however would tell that some patients with hypersensitivity describe lingering pain, and painful symptoms may persist despite occlusion of exposed dentine – this may be due to peripheral and central sensitisation. A

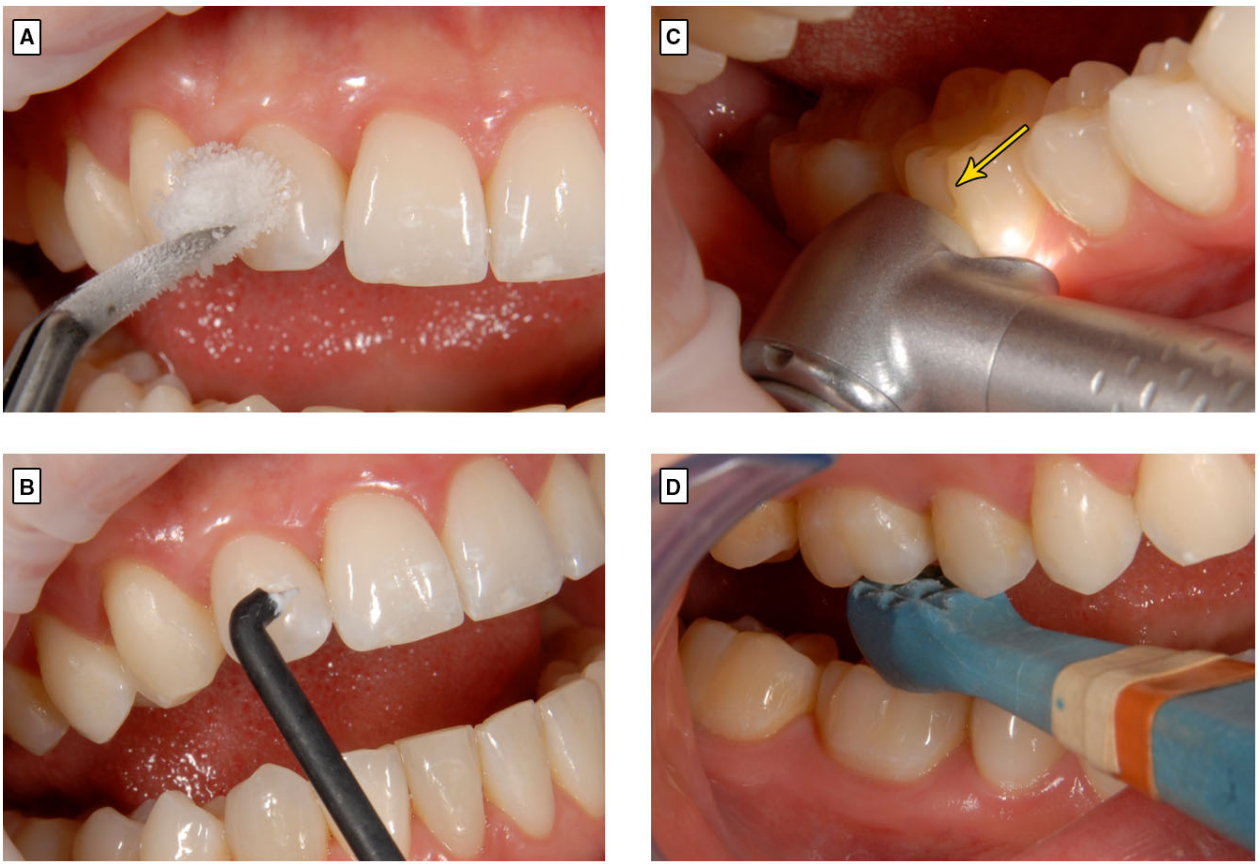

Figure 3 Commonly used methods for evaluating pulpal status (A, Thermal testing using propane/butane spray; B, Electric pulp testing tip application with increasing current), and detection of cracks (C, Transillumination of the lower first molar using the fibre optic light of a dental handpiece – note illumination of a mid-buccal fracture line [yellow arrow]; D, Use of a fracture finding device on each cusp to detect cusp fracture).

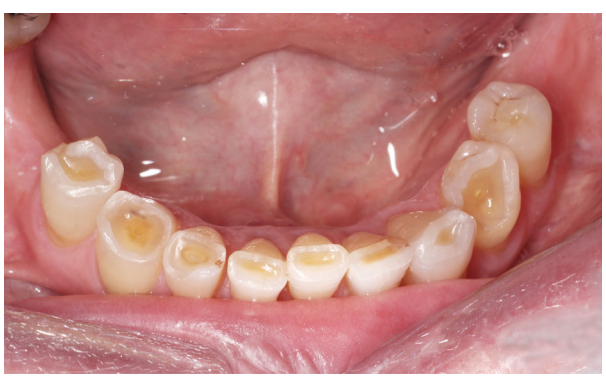

Figure 4 Exposed dentine caused by erosive toothwear. This patient reported a long history of gastro-oesophageal reflux disorder which had only recently been treated and the main dental complaint was poorly localised sharp pain on eating. Cold testing of the majority of the anterior teeth produced familiar, sharp pain and a pain diagnosis of dentine hypersensitivity as a result of erosive toothwear was made.

diagnosis of hypersensitivity should thus be based on the presence of sharp pain evoked by hot, cold and/or sweet stimuli, which lasts for no more than a few seconds, is poorly localised, and the absence of other pathologies would explain the pain (other than exposed dentine).

Pain from the periapical and periodontal tissues

Periodontal pain is defined in ICOP as 'pain caused by a lesion or disorder involving the periodontium: the periodontal ligament and/or the adjacent alveolar (peri-radicular) bone tissue'[45], and therefore may be caused by a number of conditions. As pulp inflammation progresses in the presence of apically advancing bacterial colonisation, the inflammatory process finds its way into the periapical tissues through apical and lateral foramina[41,58]. In many cases, apical inflammation may be observed during pulpitis and before necrosis of the pulp. Local inflammation causes tissue destruction and pain, which is well localised due to the numerous mechanoreceptors found in the periodontal tissues[7,8]. Periapical nociceptors become

sensitised by many of the same mechanisms as in pulpitis[59,60], and this process may produce pain which is constant and aching in character, with exacerbation of pain on biting due to compression of the periapical tissues. The history may reveal preceding pulpitis-pain-like symptoms followed by a relatively painless period due to cessation of nociceptive signals from the pulp where necrosis occurs.

Clinical examination may reveal a mobile tooth due to periodontal destruction, and the tooth will most likely be tender to percussion due to stimulation of periapical nociceptors. Sensibility testing may either elicit no response in the case of pulp necrosis, or produce pain if sensate, inflamed pulp tissue remains[61]. Depending on the degree of bony destruction, and the speed and duration of the inflammatory process, radiographic signs may range from a normal periapical appearance, widening of the periodontal membrane space, loss of the lamina dura of the alveolus or the appearance of frank periapical radiolucency[62] (Fig. 5). Long-standing, chronic lesions are more likely to show radiographic changes compared to more rapidly progressing inflammation, which may show little change in the early stages. Diagnosis is supported by the presence of a well-localised, painful tooth with tenderness to percussion, increased mobility, radiographic changes and a painful or absent response to sensibility testing.

While the inflammation associated with gingivitis may cause pain and discomfort, periodontitis is usually a painless disease[63,64]. Acute periodontal conditions such as periodontal abscess may present with well-localised pain due to the abundance of mechanoreceptors in the periodontal tissues, as well as suppuration, bleeding, swelling and tenderness. There may be tenderness to percussion of the tooth and since the pulp is usually unaffected there may be a normal response to sensibility testing (a short-lasting, slightly painful sensation to thermal testing but absence of lingering pain, and appreciable sensation of the same nature on electrical testing). Periodontal abscesses most often occur in patients with pre-existing periodontitis as an exacerbation of their condition[65]. Necrotising periodontal diseases such as necrotising gingivitis and periodontitis present with severe pain, malaise and halitosis, and are characterised by necrosis and ulceration of the periodontal tissues[66,67].

Pericoronitis is a common cause of pain, particularly associated with erupting third molar teeth. Diagnosis is usually unchallenging due to the presence of a partially erupted or impacted tooth with traumatised, tender and inflamed mucosa surrounding or overlying it. There may also be trismus and suppuration.

Occlusal trauma may be another cause of pain from the periodontal tissues, and presents as pain on biting, potentially in association with increased mobility, tooth migration and widening of the periodontal membrane space on radiographic examination[68,69]. Occlusal trauma results from excessive occlusal forces (e.g. parafunctional habits or occlusal discrepancies) in a normal periodontium, or normal/excessive occlusal forces where there is existing attachment loss. A common example of this is where a newly placed restoration has not been properly contoured, and is 'high' in the occlusion, causing excessive occlusal force and resulting pain. Gingival and periodontal inflammation caused by food impaction beneath defective approximal contacts, fractured teeth or tooth tissue loss due to caries are further sources of pain from the periodontal tissues.

Cracked teeth

Teeth with significant fractures which separate the tooth into independently mobile parts are usually easy to diagnose; however, incomplete fractures (cracks) often precede this, particularly in mandibular molar teeth with extensive restorations (although

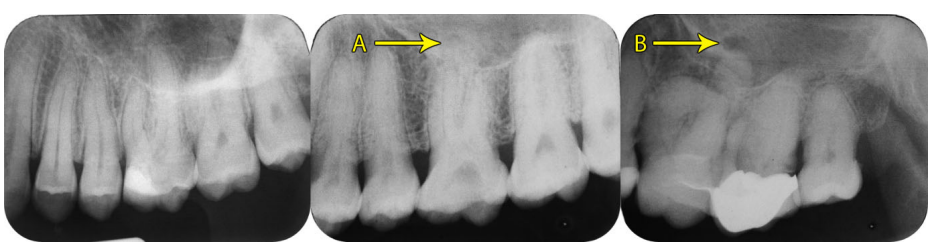

Figure 5 Periapical radiographs from different patients showing molar teeth in the upper left quadrant with varying radiographic signs of periapical disease. In all such cases, pain arising from the periapical tissues may be present. From left to right showing normal apical tissues; very early widening of the periodontal membrane space (A) and established apical radiolucency (B).

the situation also often occurs in unrestored teeth)[70–72]. Cracked teeth have long caused much difficulty in diagnosis and management[73], and patients with such cracks often present with confusing symptoms and findings on examination. This condition can be very difficult to diagnose because cracks may not be visible to the naked eye or may lie beneath otherwise seemingly sound restorations. For example, patients may complain of pain on biting, but the tooth is not tender when percussed with a mirror handle. This is because the periodontal tissues – which are tested by percussion – are not the source of the pain, and as a result the pain is not well localised. Occlusal forces wedge the crack open, and on release of the pressure, rapid movement of fluid in the dentinal tubules which have been exposed by the crack causes sharp pain[74]. Provocation of pain by asking the patient to bite on each cusp in turn using an instrument such as a FracFinder™ (Directa; Upplands Väsby, Sweden) or Tooth Slooth® (Professional Results Inc.; CA, USA) may elicit familiar symptoms (Fig. 3D). The tooth may give an entirely normal response to sensibility testing because if the crack does not extend into the pulp, the pulp may not be inflamed. Some patients however report symptoms of pulpal pain which is exacerbated by hot and cold, or spontaneous, lingering pain. In this situation, either bacterial products have diffused into dentinal tubules towards the pulp as a result of bacterial colonisation within the crack or there may be direct exposure of the pulp by the crack; in either case, the pulp becomes inflamed[75].

It is important to identify these cracks and take steps to prevent their propagation, to prevent progression to more severe pulpitis or terminal fracture of the tooth. Unfortunately, radiographic examination is usually unhelpful in the diagnosis of cracked teeth as the cracks are not well visualised[71] unless they serendipitously run in the direction of the X-ray beam. One clinical feature that may be present if the crack involves the periodontal ligament is a localised area of increased periodontal probing depth due to attachment loss at the site of the crack; although not always present, this feature represents poorer prognosis for the tooth[72] and indicates microbial presence in the crack. Transillumination may be useful in visualising a crack, as light transmitted through the tooth is blocked by discontinuity of the enamel and dentine[71] and a clear boundary will appear (Fig. 3C). Dedicated fibre optic light sources exist for this purpose (among others), although light from a dental handpiece or composite curing light (taking care to protect the operator and assistant's eyes) is often effective. Other techniques such as the use of commercially available dyes usually used for caries detection, and magnification using loupes or an operating microscope also aid detection.

Diagnosis

The key to managing pain is an accurate diagnosis. This cannot be reached unless a comprehensive history, careful examination and appropriate special tests and investigations are performed. The history itself is often the key to the diagnosis and the nature of the pain, its timing, duration, location and precipitating factors guide the clinician to the cause. For example, pain in response to change in temperature rather than pressure may indicate a pulpal cause in favour of a periodontal one, and pain on biting might make pain of periodontal origin or a cracked tooth more likely. Similarly, short-lived pain in response to cold drinks may make pulpal pain as a result of mild pulpitis or hypersensitivity more likely than if the pain was spontaneous and lingering, as might be seen as a result of severe pulpitis.

A careful and thorough examination should identify any pathology affecting the teeth which may indicate a source of pain, and efforts should be made to reproduce the patient's pain by palpation, percussion, selective loading of teeth and response to thermal stimuli. Similarly, suspicious teeth should be tested for sensibility using appropriate methods[44]. A provisional diagnosis is often formed following the history and examination, and radiographic tests serve to confirm or refute this. For example, secondary caries around a deep restoration found on a bitewing radiograph may support a provisional diagnosis of pulpal pain caused by severe pulpitis, and radiolucency around the apex of a tooth which is tender to percussion on examination may confirm a diagnosis of apical periodontitis as the cause of periapical pain.

The results of the history, examination and investigations should never be relied upon in isolation – not infrequently the results of each of these can be at odds with each other, and the picture may not be clear. All of the information obtained should be interpreted in combination to reach an accurate diagnosis. Where the diagnosis is not certain, on occasion it may be appropriate to delay treatment until the picture is clearer, in preference to performing an irreversible procedure. Occasionally, the clinical findings do not tally with the patient's symptoms, and although it may be tempting to try to do something to a tooth which seems otherwise

sound when the patient insists it is the cause of their pain, it should always be considered that the cause of the patient's pain may not be odontogenic, and a definite diagnosis should always precede any invasive intervention. It is not unheard of for patients with non-odontogenic pain to have had multiple teeth restored, endodontically treated and extracted by well-meaning dentists with no improvement in their symptoms. Pain arising from a non-odontogenic cause such as temporomandibular disorders, trigeminal neuralgia, post-traumatic trigeminal neuropathic pain and headache disorders including migraine may present similarly to odontogenic pain; this is discussed in depth elsewhere in this issue.

Future directions – how might diagnostics be improved?

Although the approaches we have mentioned form the mainstay of the diagnosis of odontogenic pain, innovative techniques have been reported, although they are not yet widely used in clinical practice. The variable reliability of thermal and electronic pulp testing may be overcome by measuring the level of blood oxygenation in the pulp as a marker of pulp perfusion using pulse oximetry[76,77]. This technique measures the absorption of light of a specific wavelength transmitted through the tooth by placing a probe onto it. Readings are reproducibly and progressively lower in healthy pulp, varying degrees of pulpitis, and pulp necrosis, respectively; this method may provide a non-invasive method of differentiating between these pulp states; however, it requires specialist equipment which may limit its use.

Several inflammatory markers such as tumour necrosis factor-alpha, prostaglandin-E2, interleukin-2 and matrix metalloproteinase-9 have also been reported to be reproducibly greater in severe pulpitis than mild pulpitis; this could prove clinically useful if levels of these markers can be measured *in vivo* to determine the need or otherwise for endodontic therapy in pulpal pain resulting from pulpitis[78,79]. A number of authors have described techniques for measuring these pulpal biomarkers, including sampling of dentinal fluid during cavity preparation[80], or pulpal blood following pulp exposure or during pulpotomy[81]. It remains to be seen however whether these techniques could be simplified sufficiently for routine clinical use in the future.

As discussed, the diagnosis and localisation of cracks in teeth can be challenging; recently, a number of imaging techniques have shown some promise in visualising these. Firstly, imaging of teeth *ex vivo* with magnetic resonance imaging (MRI) using a protocol called sweep imaging with Fourier transformation (SWIFT) and a custom intra-oral MRI coil demonstrates cracks as small as 20 μm, and images are not hampered by streak artefact from metallic restorations as seen when using cone beam computed tomography (CBCT)[82,83]; additionally, MRI avoids using ionising radiation and the problems that may come with this. This is important because MRI using conventional clinical protocols is typically unable to obtain useful signal from dental hard tissues, although currently the SWIFT protocol is not available on most MRI systems. Interestingly, this technique has been used to visualise artificial fluid flow inside the pulp chambers of teeth *in vitro*, which if possible clinically could provide a non-invasive method of quantifying pulpal blood flow[84]. Secondly, although CBCT typically has low accuracy for crack detection, one group have demonstrated the potential utility of a computational technique called isotropic steerable wavelets in their preliminary work[85,86]. This technique allows the automatic analysis of high-resolution CBCT data sets to detect cracks and may help to improve the accuracy of this now widely used modality for crack detection. This work is however very much preliminary.

Conclusions

Pain from the teeth is common, and although the origin is usually easily identified following a thorough assessment, occasionally the clinical presentation may be challenging. We would emphasise the importance of a clear, structured and comprehensive approach to history taking, examination and investigation, and stress that the diagnosis should be reached only after interpreting all the findings in light of the patient's complaint. Once a diagnosis has been reached, pain is usually effectively treated by managing the cause – be that by restoration of teeth, conventional or conservative endodontic treatment, tooth extraction or periodontal therapy. Clinicians should be wary of the uncommon, but real possibility of non-odontogenic disorders presenting as toothache and be confident in recommending further investigation over irreversible intervention where the history does not align with the clinical findings.

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