Restorative Dentistry

Cracked tooth syndrome diagnosis: integrating the old with the new

Oliver Bailey BDS(Hons); MFDS RCSEd; PGCert Implant; FHEA; MFDTEd

Clinical Fellow Newcastle University School of Dental Sciences; GDP North East

John Whitworth BChD(Hons); PhD; FDSRCS; FDSRCS(Rest)

Professor of Endodontology Newcastle University School of Dental Sciences

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Abstract

This article is the first of a two part series on cracked tooth syndrome (CTS). It seeks to aid the clinician in understanding the pathogenesis and clinical features of the condition and review established and new diagnostic tests that will allow greater confidence and predictability in diagnosing teeth with CTS.

Clinical relevance

Gives the clinician greater confidence and predictability in diagnosing teeth with CTS.

Objectives

Explains the pathogenesis and clinical features of CTS and reviews established and new diagnostic tests

Introduction

CTS refers to the signs and symptoms of pain in a posterior tooth with a vital pulp, that is directly attributable to an incomplete fracture involving the dentine, which occasionally extends into the pulp or periodontal ligament.[1] It commonly presents with sharp pain on chewing and thermal sensitivity, and can be difficult to distinguish from other pulpal and periapical conditions[2] (see later).

A crack has been defined by Oxford Dictionaries as, 'a line on the surface of something along which it has split without breaking apart'. At this point it could be described as an incomplete fracture (Figs 1a,b&2), as there is no visible separation of the segments divided by the crack. Cracks can be symptomatic which would support a diagnosis of CTS (Figs 1a&b), or asymptomatic which would not (Fig 2). A complete fracture would demonstrate visible separation and independent movement of one or more segments (Figs 3a-c).

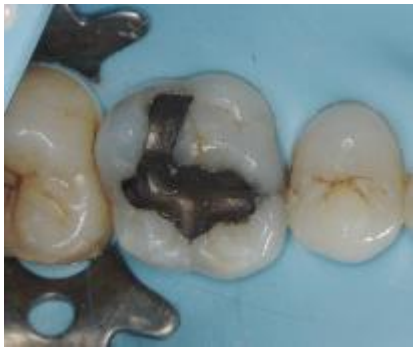

Fig 1a Crack (incomplete fracture) of mesio-palatal cusp UR6. Symptoms included pain on biting. Pain reproduced by biting pressure and release of biting pressure on mesio-palatal cusp. Diagnosis: CTS

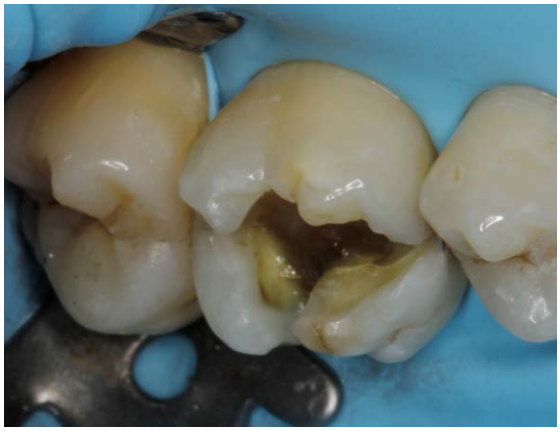

Fig 1b Oblique crack (incomplete fracture) undermining mesio-palatal cusp UR6 evident following restoration removal.

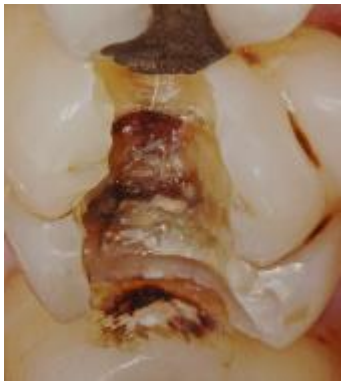

Fig 2 Vertical crack (incomplete fracture) in another UR6 evident running mesio-distally following removal of caries and existing restoration. Tooth asymptomatic, not CTS

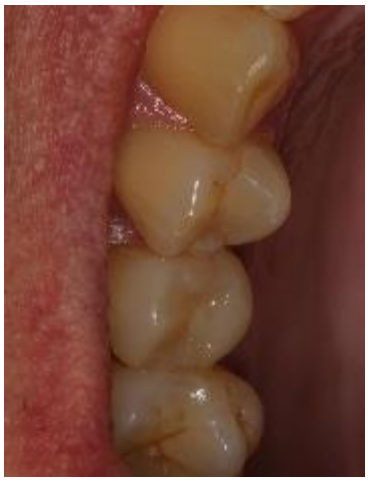

Fig 3a Pain on biting UR4

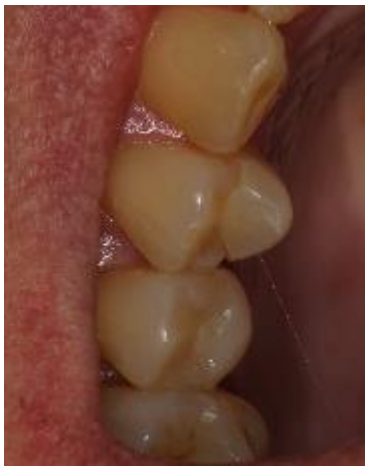

Fig 3b Visual separation with digital pressure. Diagnosis: complete fracture, not CTS.

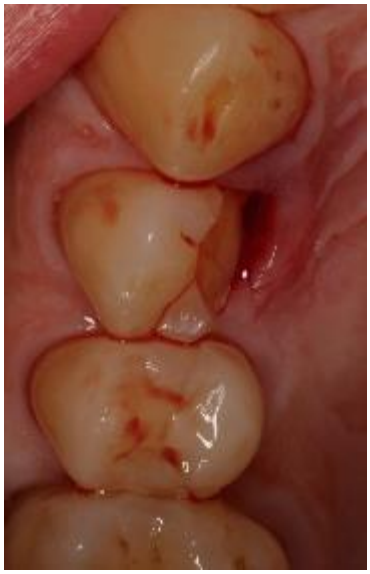

Fig 3c UR4 after removal of mobile portion to assess restorability

Dentine cracks

Internal vs external initiation

A diagnosis of CTS relies on the presence of a painful crack within dentine, not necessarily the overlying enamel, and the presence of an enamel crack does not necessarily indicate that the underlying dentine is cracked[3] (Figs 4a&b). Cracks are mainly initiated and propagated by occlusal loading, with some progressing internally from an initiation point on the external aspect of the tooth, whilst others develop from internal stress concentrators, such as the line angles of cavities and propagate externally (Fig 5). Such cracks are not always associated with visible crack-lines in enamel, which may complicate diagnosis, classification and appropriate clinical management (see later).

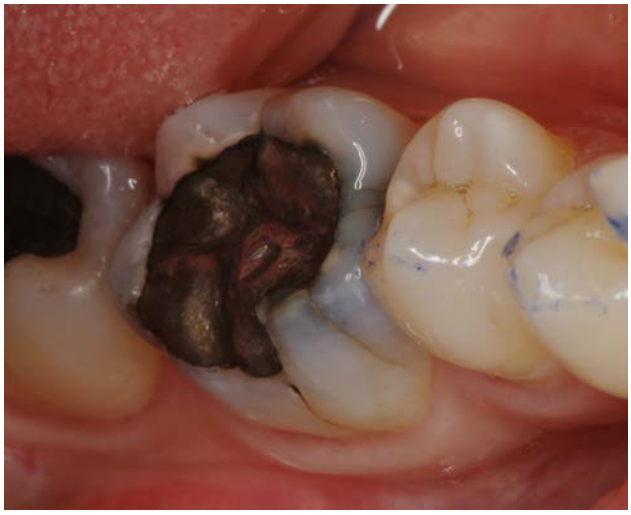

Fig 4a. Reproducible pain on biting pressure MB cusp LR6. Multiple enamel cracks visible pre-operatively.

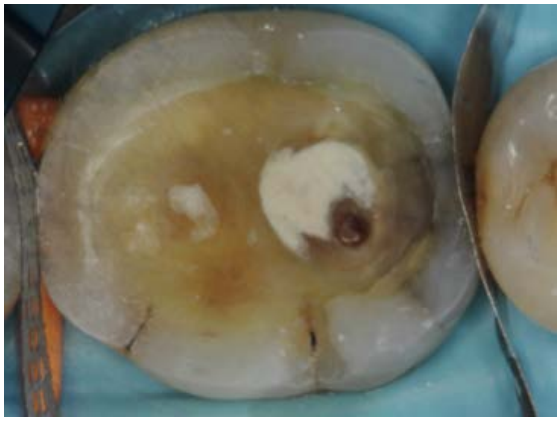

Fig 4b. Following LR6 cavity preparation, multiple stained cracks and fissures confined to enamel evident. Oblique dentine crack MB region most likely responsible for symptoms, but not visible pre-operatively.

Propagation resistance

Dentine is a tough, resilient material, and will resist crack propagation through the formation of micro-cracks ahead of the main crack. These serve to dissipate energy and can lead to 'crack blunting'. Unbroken 'ligaments' of intertubular collagen behind the tip of the crack also serve to resist propagation[4] (Fig 5).

Critically, this suggests that a tooth with a dentine crack is still capable of functioning without fully removing the crack.

Cyclical loading has a greater propensity to propagate cracks than static loading,[5] suggesting bruxists may fare worse than clenchers. Hydration of dentine improves crack blunting,[4] suggesting that root filled teeth and teeth with non-vital pulps may be at greater risk, above and beyond their structural compromise. Aging beyond around 30 years also reduces fracture resistance.[6]

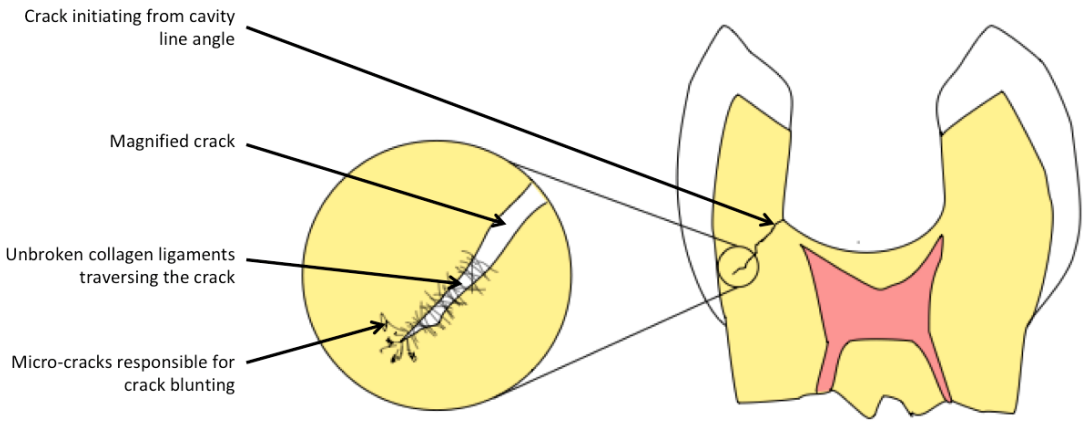

Fig 5 Internally initiated crack demonstrating propagation resistance

Aetiology

Suggested predisposing factors for CTS include previous cavity preparation, restorative material compaction or bonding procedures, tooth morphology, cervical tooth surface loss, function, parafunction and trauma, all of which may lead to crack initiation or propagation.[1, 7]

Diagnosis

Diagnosis can be difficult, with many CTS affected teeth originally misdiagnosed.[2] Commonly there is a history of pain on chewing, and sensitivity that can range from transient to lingering. Sensitivity is often elicited by thermal stimuli (mainly cold) or sweet food and drinks.[8]

A crack can lead to secondary involvement of the pulp or periodontium. The history, clinical examination and any tests should look to assess their potential involvement, whilst considering other aetiologies in the differential diagnosis.

Clinical examination and visual inspection may be enhanced by magnification and transillumination.[7] The presence of an enamel crack is often not diagnostic in the absence of other clinical signs. Visible crack separation, which would give a diagnosis of a complete fracture, can be assessed by attempting to separate cusps manually (Figs 3a-c). A probe catch, or bubbles forming at the gingival margin adjacent to a crack as it opens and closes under digital or biting pressure can be an early sign of a complete fracture. Crack extension to the periodontium may result in localised deep periodontal probing depths.[9]

Tests should look to predictably reproduce the presenting symptoms and localise the source of the pain.

Reproducing thermal pain

Air from a 3-in-1 often elicits symptoms from cracked teeth. Pulp sensibility testing is advised, and an exaggerated response from the affected tooth may aid diagnosis.[10] The pulp may present in variable states. Assessing whether a pulpitis is reversible or irreversible will guide management, however this may be difficult until after the crack is stabilized,[11] and these clinical diagnoses may not accurately represent the histological diagnosis.[12] 98% of teeth presenting with CTS that exhibited pain lingering for up to 45 seconds after ethyl chloride application were successfully managed, resolving pain on biting and maintaining pulp vitality at one year.[13]

Reproducing pain on biting

Percussion in an occluso-apical direction is often painless, whilst lateral percussion can elicit characteristic symptoms. Rebound pain on release of

pressure is classically described as being highly suggestive of a diagnosis of CTS,[14] however data suggests that pain on application of pressure is more common than pain on release, or the presence of both phenomena.[2] Each can cause fluid movement within or out with the tubules and consequent pain.[15] Common tools used to elicit these responses are the Tooth Slooth (Professional Results, Inc, California, USA) (Fig 6), the FracFinder (Denbur, Oak Brook, Illinois), and cotton wool rolls. Biting on cotton wool rolls has limited application because cotton is non-rigid, and rolls are usually too large to be applied in a controlled manner to individual cusps. Each cusp of all teeth in the affected area should be assessed, and painful responses should be checked for reproducibility. It is important to consider opposing teeth, as these are inadvertently loaded during testing.

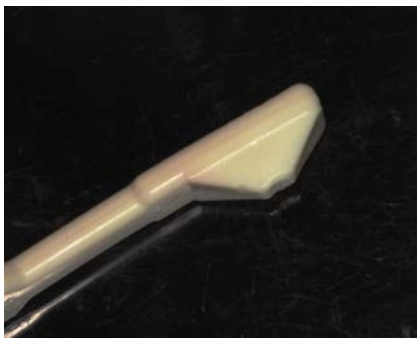

Fig 6 Tooth Slooth- small cupped tip allows stable application to, and testing of, individual cusps

It is always prudent to check both the static and dynamic occlusion and consider occlusal trauma in the differential diagnosis. A study reported non-resolution of symptoms from a tooth initially diagnosed with CTS and managed with an adhesive composite restoration.[16] Subsequent occlusal adjustment resolved the pain.

Where doubt exists over the diagnosis, a trial direct composite splint (DCS) (also called a direct supra-coronal resin onlay restoration or direct coronal onlay splint) can be useful.[17] If the pain resolves after the application of non-bonded composite that wraps over and constrains the cusps (Figs 7a-d), the clinician may be confident of a CTS diagnosis. If the pain on biting does not resolve, it is prudent to reconsider the diagnosis. Differential diagnoses may include apical periodontitis, irreversible pulpitis (uncomplicated by a crack) and occlusal trauma.

Radiographs are useful to identify other pathologies that may be confused with CTS, such as pulpitis associated with caries, or symptomatic apical periodontitis, but are of limited value in diagnosing undisplaced dentine cracks.

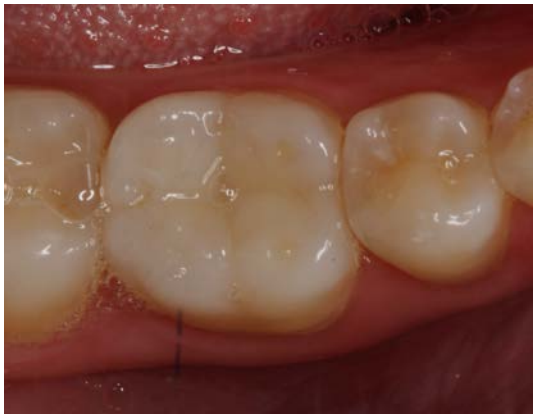

Fig 7a Pain on biting from a minimally restored LR6. Occlusal composite placed 5yrs previously. Pain reproduced on release of biting force on lingual cusps using Tooth Slooth. Pulp responds vital to thermal testing.

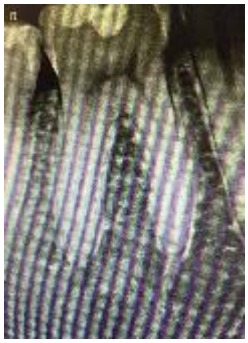

Fig 7b Peri-apical radiograph LR6 shows no obvious apical pathology, a distal radiolucency apparently confined to enamel and a fairly shallow occlusal restoration

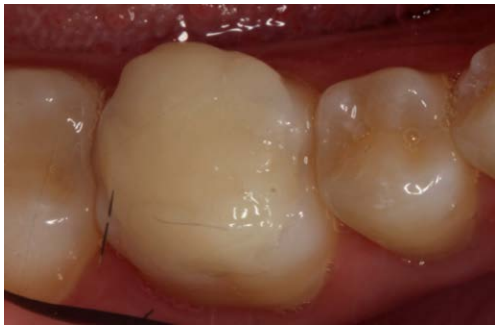

Fig 7c Diagnostic DCS provided by direct application of non-bonded composite resin, 1.5mm thick on the occlusal, with extension over buccal and lingual cusps. Patient asked to close their teeth together, explaining that the bite will feel high. Complete resolution of painful biting symptoms confirms the diagnosis of CTS.

Fig 7d Diagnostic DCS removed simply. The second article in this series will describe ongoing management with a definitive DCS.

Crack classification

Many attempts have been made to classify cracks.[9, 18] An ideal system would allow prevalence data to be recorded in defined populations and guide clinical decision-making for individual patients. One recent system of crack classification[9] stated that the 'location and extent of the crack determine the treatment plan'. However in CTS it is often impossible to know the location and extent of the crack at presentation. Diagnostic testing often gives no indication of the location or extension of the crack(s) (Fig 8). Even when the tooth is operatively explored, by removing existing restorations (Figs 1&8), the true extension is often unclear. A classification system should therefore not over reach by including clinically unknowable variables.

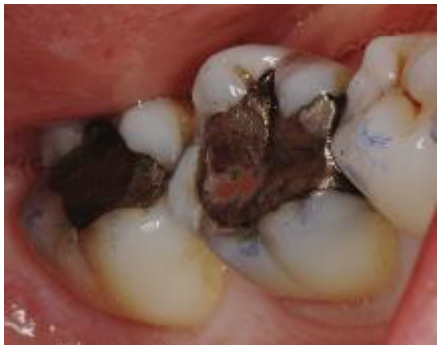

Fig 8a Pain on biting, LR6. Symptoms reproduced by biting pressure applied via Tooth Slooth on distobuccal cusp.

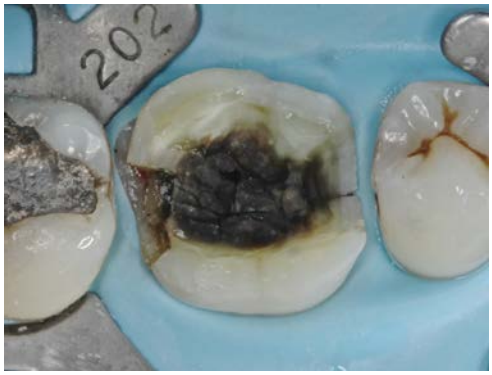

Fig 8b Same tooth as Fig 8a. Disto-buccal portion fell away on removal of restoration, but multiple cracks noted with central vertical crack. Diagnostic testing gave no indication of the location or extension of the cracks.

All that can really be ascertained (and again this may only be possible following operative exploration), is if cracks run obliquely (Figs 1b & 9) or vertically (Figs 2 & 8b). An oblique crack that can be seen both internally in dentine and externally in the overlying enamel (Fig 9) may have clinical relevance and is therefore prudent to include in a crack classification. Unrestored teeth with a suspected crack should ideally not be opened for investigation but managed by non-destructive means if possible (see follow-up paper).

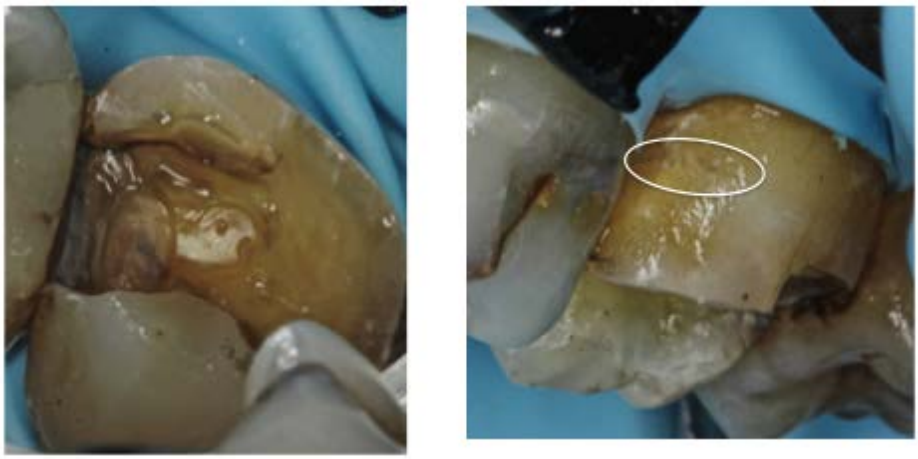

Fig 9 Oblique crack undermining mesio-buccal cusp UL7 in dentine visible in overlying enamel

The extension of a crack, in the absence of frank manifestations of pulpal or periodontal pathology, or an observable exit point, is always unknown. Any attempts to quantify the extension are therefore unhelpful in formulating a treatment plan. This is most often the situation faced when a diagnosis of CTS is made. Cracks commonly harbor biofilm,[19] and may extend to the pulp or the periodontium, but might not necessarily manifest pulpal or periodontal disease. This is reliant on the presence and nature of the biofilm, and the host response to it, which may often be in equilibrium. Subtle shifts in quantity or quality of the biofilm, or in the host response can easily change this balance, favouring either health or disease. The complex dynamics seen in the shift from biofilm influenced health to disease are not fully known.[20]

Crack epidemiology

CTS is most commonly seen in mandibular molars, followed by maxillary molars and then maxillary premolars, with non-functional cusps more commonly affected than functional cusps.[8] Finite element analysis has helped to explain this observation by showing that non-functional cusps generally sustain more damaging tensile stresses, whilst functional cusps generally sustain more favourable compressive stresses.[21] The restorative status of affected teeth varies considerably between studies, with the proportion of unrestored teeth ranging from 5-60%.[22, 23]

There are few good data on the incidence or prevalence of CTS in defined populations. Hilton et al.(2007) reported a 'very high' prevalence of cracked teeth in an American population,[24] though this is likely to have included cracks confined to enamel or 'craze lines', and asymptomatic cracks which are therefore not teeth with CTS. Cracks in dentine are also often asymptomatic[25] (Fig 1c). One study in an American population of patients with observable cracks suggested that the greatest chance of a tooth being symptomatic (CTS) was seen in patients who had the combination of a molar tooth with an observable distal crack that blocked transilluminated light, though the increase in likelihood was modest at

just over 20%. Stained cracks were less likely to be symptomatic.[7] This data does highlight the problem of visually differentiating crack lines which are confined to enamel from those which extend into dentine, and ascribing causation to a visible crack in a painful tooth (Fig 4).

Conclusion

Patients with CTS may present with a confusing collection of symptoms.

Successful clinical management of cracked teeth does not always require the removal of the crack or a segment of tooth tissue.

Current classification systems are not always helpful in guiding clinical management. Classification of cracks in CTS should be limited to known parameters.

Current diagnostic methods may be inconclusive, but when supported by the provision of a diagnostic DCS that resolves the patient's symptoms, may reassure both the patient and practitioner of the diagnosis.

The second article in this series looks at the effective clinical management of teeth with a confirmed diagnosis of CTS, including a discussion of when to bond the diagnostic DCS in supra-occlusion, when it may not be appropriate to do so, and what to do if it is not. The development of a decision tree seeks to clarify the decision making process.

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