TOP TIPS

Top tips for managing enamel infractions, cracks and fractures – Part 1: Diagnosis

By R. J. R. Smith,[1] E. McColl[2] and G. E. Bryce[3]

Tooth infractions, cracks and fractures (ICF) represent mechanically induced discontinuities within the dental hard tissues. Such findings are commonly observed in clinical practice, and whilst often asymptomatic, may be associated with a range of symptoms and/or aesthetic issues. In part 1 of this two-part series, we offer top tips for assessment and diagnosis of these problems.

1. Terminology

a. Key definitions are shown in Table 1 and Figures 1 and 2. Definitions are adapted from Abbot and Leow.[1]

b. Patients with cracked and fractured teeth may present with a range of symptoms including: no symptoms, pain on biting (or release), temperature sensitivity, or (if pulpitis) spontaneous pain. Such symptoms in cracked teeth are often collectively referred to as 'cracked tooth syndrome'. However, this terminology is erroneous because the word 'syndrome' relates to a 'disease', whereas a crack is a clinical finding not a disease.[1]

Table 1 Definitions of tooth infractions, cracks and fractures

	Definition
Enamel Infraction	Micro-cracks confined to enamel (Fig. 1).
Crack*	Defect where there is a break between two parts of a tooth, without separation of the fragments (Fig. 2) May involve dentine +/- enamel +/- cementum +/- direct communication with pulp.
Fracture*	Defect where there is a break between two parts of a tooth and the fragments have separated (Fig. 2) May involve one or more of the following hard tissues: enamel, dentine, or cementum +/- direct communication with pulp.

*The terms 'crack' and 'fracture', whilst often used interchangeably, represent distinct clinical situations.

2. Aetiology

a. The high inorganic content of enamel makes it hard but inflexible, with a flexural modulus variously reported to be within the range 10–130 GPa.[2] However, the relative flexibility of dentine (flexural modulus within the range 10–20 GPa),[3] periodontal (suspensory) ligament, (and to a much lesser extent) alveolar bone, combined with neuromuscular occlusal force feedback mechanisms, reduces the risk of overloading of teeth. However, where the flexural strength of tooth tissue is exceeded, crack initiation may occur and, if the crack propagates, fracture with separation of the fragments may ultimately occur.

b. ICFs may be caused by acute dental trauma related to events such as accidents, physical assault and sporting mishaps. Such incidents should be screened for within the history taking.

c. The muscles of mastication can generate large occlusal forces. One study reported a patient who could generate a biting force of 575N within the molar/premolar region.[4] Frequent or sustained occlusal forces (associated with parafunctional activity) can be problematic, and a correlation between bruxism and cracks and fractures in teeth has been reported.[5] Parafunction can take several forms, and it is useful to screen for the following habits: clenching/holding teeth together, tapping teeth together, grinding, fingernail chewing, and cheek chewing.

d. Hard diets (eg nuts/bone/ice) may cause ICFs.

e. Unfavourable inter-occlusal relationships (eg non-working side interferences, edge to-edge incisors/canine relationship, and cusps that occlude with the opposing marginal ridges) can

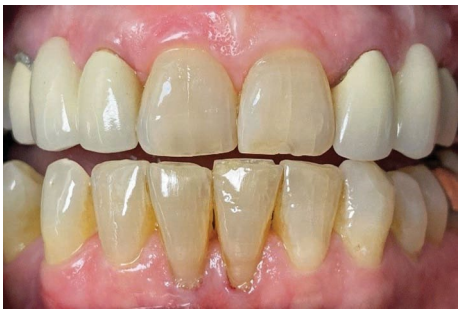


Fig. 1 Enamel infractions in anterior teeth

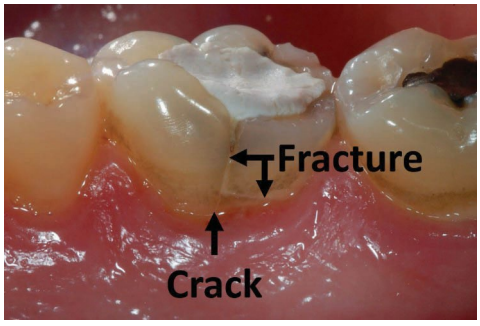


Fig. 2 Lower right 6 with vertical crack-line in mid-lingual aspect, and fracture of the disto-lingual cusp

[1]Enhanced Practitioner, Defence Centre for Rehabilitative Dentistry, Evelyn Woods Road, Aldershot, GU11 2LS, UK; [2]Director of Clinical Dentistry, Peninsula Dental School (University of Plymouth), Plymouth, UK; [3]Consultant in Restorative Dentistry, Defence Centre for Rehabilitative Dentistry, Evelyn Woods Road, Aldershot, GU11 2LS, UK.

predispose to ICFs. In addition to clinical examination to identify these features, a history of repeated failure of restoration(s) should signal that unfavourable occlusal relationships/forces may be at play.

f. ICFs may unfortunately also be induced iatrogenically. Awareness of the potential for this to occur, and undertaking mitigating action, should reduce the incidence of this. Areas for consideration include: ultrasonics (use lowest power suitable to the task), rubber dam clamp selection (plastic vs metal, or alternatively use of elastic rubber dam stabilising cord), avoidance of excessive wedging forces during compaction of root canal fillings and amalgam, and avoiding use of dentine pins and threaded posts.

3. Pathophysiology

a. Dentinal pain, characterised by 'sharp'/intense/short-lasting pain upon application of suitable stimulus (eg thermal/sweet/drying/mechanical), may arise in vital teeth with exposed dentine (+/- porosities in enamel) +/- cracks or fractures. The hydrodynamic theory of dentine sensitivity postulates that dentinal pain is related to the induction of transient fluid movement within dentinal tubules (caused by application of a suitable stimulus, which in turn stimulates Aδ nociceptors within the pulp/dentine complex[6]). If upon removal of the stimulus, the symptoms cease immediately, this suggests that at that time the problem is confined to dentine, and that significant pulpal inflammation has not yet occurred.

b. Coronal cracks/fractures may act as a portal for bacteria and their by-products to infiltrate the dentine and pulpal complex, with complications including: tertiary dentine formation/calcific metamorphosis (causing yellow discolouration of the crown) (Figures 3 and 4), and pulpitis (reversible or irreversible) that may ultimately lead to pulpal necrosis and apical disease.

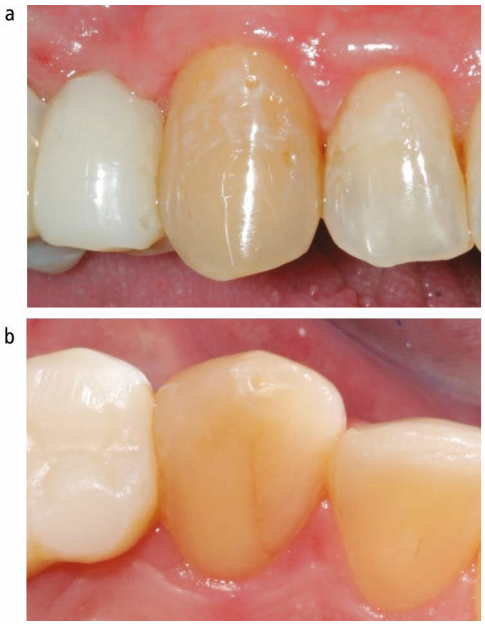


Fig. 3 a, b) Labial and palatal views of an upper right 3 (13) with yellow discolouration. The tooth had been traumatised five years previously. There was a crack-line in the palatal aspect. The tooth was asymptomatic and tested positive with electric pulp test (but negative with cold testing)

c. Cracks extending into the root may initiate additional complications within the periodontal ligament complex. For example, periodontal tissues adjacent to an infected crack or fracture will undergo inflammatory events, which classically result in periodontal attachment loss that vertically aligns with the crack forming a deep narrow periodontal pocket. With further crack propagation to the apex (especially in the case of root fractures), more extensive periodontal destruction may ensue resulting in the radiographic appearance of crater or (classically) 'J-shaped' bone loss. A further (unusual) presentation of note is an apico-coronal root fracture in a tooth testing 'vital', which can present with a peri-apical radiolucency +/- symptoms of apical periodontitis (typically presenting in maxillary second molars with 'spindly' roots).

4. Top tips for assessment

a. The aspiration is to either directly visualise the ICF, or in symptomatic cases, achieve ICF localisation by precise replication of the patient-reported symptoms.

b. Teeth should be dried (with a 3:1 syringe) and examined under good lighting conditions with magnification (loupes or microscope).

c. Pulp testing with both cold (eg Endo-Frost, Coltène/Whaledent, Langenau, Germany) and Electric Pulp Test (EPT) provides a surrogate measure for pulp vitality.

d. Rubber dam held in place with fingers (or rubber dam clamp) can be readily used to individually isolate suspect and 'control' teeth, prior to application of stimuli relevant to the patient's pain history. For example:
   i. Cold or warm water can be syringed into the dam to form a pool around the tooth and left for several seconds before being aspirated out
   ii. Air may be blown over the tooth using a 3:1 syringe.

e. The character of pain (eg sharp/aching/throbbing etc), intensity of pain (using a visual analogue scale 1–10), and duration (fleeting or lingering) should be determined. Taken together, these findings can help localise the source of pain, and the structure(s) involved (eg specific tooth/cusp, and whether dentine/pulp +/- peri-apical tissues). It should be noted that pulpless teeth with vertical root fractures may sometimes give a false positive test with a hot/

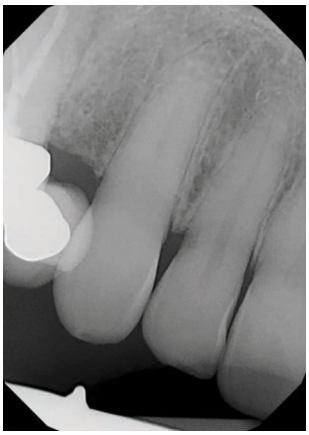


Fig. 4 A long cone peri-apical radiograph demonstrated calcific metamorphosis and an intact periodontal ligament space. No treatment was deemed necessary and the tooth placed under long-term clinical and radiographic review

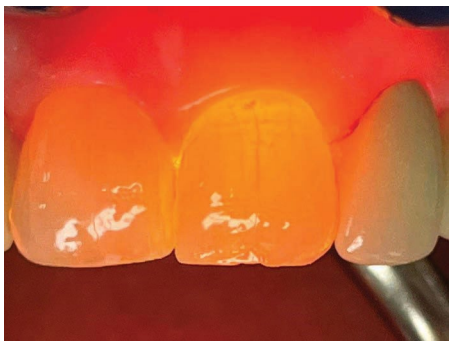


Fig. 5 Same case as shown in Figure 1, with transillumination of the upper left central incisor tooth highlighting enamel infractions

cold stimulus due to fluid percolation through the fracture line to the periodontal ligament.

f. Transillumination of teeth may demonstrate cracks/fractures by virtue of refraction phenomenon at the crack/fracture boundary (Fig. 5). However, the effectiveness of transillumination is reduced in teeth with restorations, and not possible in teeth with full-coverage restorations. Where (direct/indirect) restorations are removed as part of a restorability assessment, it is useful to carry out transillumination (again) once the tooth structure is revealed.

g. A Tooth Slooth (Professional Results, Laguna Niguel, USA) is a plastic device for identifying teeth/specific cusps eliciting pain on biting (or, following biting, upon release). The device should be used methodically, placing it on one specific cusp at a time, and asking the patient to bite onto it. The patient's reported pain experience (if any) should be recorded for each tooth/ cusp of interest.

h. Deep narrow periodontal defects associated with cracks or fractures may be subtle and can easily be missed. A UNC15 probe is well suited for periodontal assessment and should be carefully 'walked around' the tooth/teeth of interest – noting the pocketing profile. Where inflammation is present, the patient may signal particular discomfort on periodontal probing, and an under-estimation of pocket depth may be made. In such cases, we would advise the administration of infiltrations of local anaesthesia prior to re-probing. It should also be noted that whereas the buccal and palatal/lingual aspects are usually readily accessible for periodontal probing, where a proximal tooth contact exists, access to probing in that area will be compromised. In such cases, where a Class II restoration is present which requires removal, the gingival sulcus at the proximal surface(s) should be re-probed after removal of the restoration.

i. Dyes, such as methylene blue, may also be used in the detection of cracks in teeth.

j. Two-dimensional radiographs will not directly demonstrate fractures unless the x-ray beam is within four degrees of the plane of fracture.[7] However, appropriate intra-oral radiographs may be indicated if there is no previous or recent imaging available in order to screen for caries/defective restorative margins/crestal bone levels/peri-radicular pathology.

k. Three-dimensional radiographic imaging (eg cone beam computerised tomography [CBCT]) has been shown to be able to predictably detect root fractures in the lab-based setting.[8] However, in the clinical situation, the reliability of CBCT for detection of root fractures is limited by voxel size (linked to the need to keep radiation dose as low as reasonably practicable), and degree of separation of the fractured portions potentially being below the limit of resolution. Additionally, where the tooth has been previously root-filled, imaging artefacts caused by gutta percha can frustrate attempts to screen for root fractures.[9] However, as previously mentioned, the finding of deep localised or 'J-shaped' radiolucency (on either 2D or 3D radiographs) is characteristic of the pattern of bone loss associated with cracked or fractured roots (Figures 6 and 7).

5. Top tips for diagnosis

a. Following assessment, relevant provisional or (ideally) definitive diagnoses are made based on triangulation of findings.

b. Causes of signs and symptoms other than cracks/fractures must be identified (and managed) including: dentine sensitivity secondary to gingival recession, caries, non-carious tooth surface loss, or defective restorations.

c. The following diagnoses may be appropriate within the context of cracked or fractured vital teeth:
   i. Enamel infraction
   ii. Enamel or enamel-dentine fracture +/- pulp exposure (eg complicated or uncomplicated)
   iii. (Apico-coronal) root fracture +/- (clinical symptoms of) pulpitis
   iv. Dentine sensitivity secondary to crack/fracture
   v. Reversible pulpitis secondary to crack/fracture
   vi. Irreversible pulpitis secondary to crack/fracture.

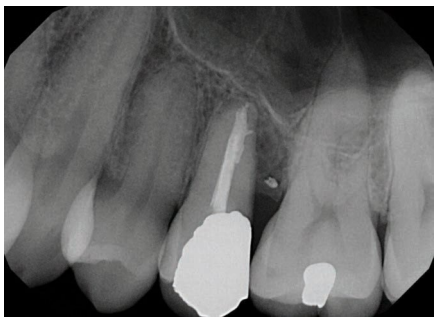


Fig. 6 Localised infrabony defect distal to a root-filled upper left 5 tooth

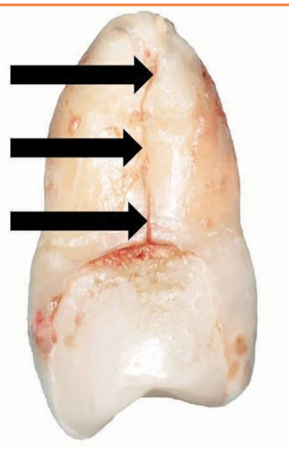


Fig. 7 A restorability assessment of the upper left 5 tooth (shown in Fig. 6) revealed that the root was cracked, deemed not predictably restorable, and to be extracted. Crack-line is arrowed

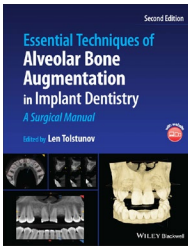
d. The following diagnoses may be appropriate within the context of cracked or fractured non-vital teeth:
   i. Apical periodontitis (acute or chronic) secondary to crack/fracture
   ii. Perio-endo lesion secondary to crack/fracture of root.

We hope you have found these hints and tips helpful. Top tips for managing enamel infractions, cracks and fractures will be discussed in part 2 of this series. ■

References

1. Abbott P, Leo W. Predictable management of cracked teeth with reversible pulpitis. *Aust Dent J* 2009; **54:** 306–315.
2. Lu C, Nakamua T, Korach C. Effective property of tooth enamel: monoclinic behaviour. *J Biomech* 2012; **45:** 1437–1443.
3. Kinney J, Balooch, M, Marshall, G, Marshall S. A micromechanics model of the elastic properties of human dentine. *Arch Oral Biol* 1999**; 44:** 813–822.
4. Edmonds H, Glowacka H. The ontogeny of maximum bite force in humans. *J Anat* 2020; **237:** 529–542.
5. Lobbezoo F, Manfredini D, Bracci A. Bruxism. *In BMJ Best Practice.* 2022. Available at: https://bestpractice.bmj.com/topics/en-gb/708 (accessed May 2023).
6. Brännström M. A hydrodynamic mechanism in the transmission of pain-producing stimuli through the dentine. *In* Anderson D (ed) *Sensory mechanisms in dentine.* pp 73–79. Oxford: Pergamon Press, 1963.
7. Rud J, Omnell K. Root fractures due to corrosion. Diagnostic aspects. *Scand J Dent Res* 1970; **78:** 397–403.
8. Guo X L, Li G, Zheng J Q *et al.* Accuracy of detecting vertical root fractures in non-root filled teeth using cone beam computed tomography: effect of voxel size and fracture width. *Int Endod J* 2019; **52:** 887–898.
9. Patel S, Brady E, Wilson R, Brown J, Mannocci F. The detection of vertical root fractures in root filled teeth with periapical radiographs and CBCT scans. *Int Endod J* 2013; **46:** 1140–1152.

BOOK REVIEW



ESSENTIAL TECHNIQUES OF ALVEOLAR BONE AUGMENTATION IN IMPLANT DENTISTRY: A SURGICAL MANUAL (2ND ED)

Editor: Len Tolstunov;
2022; Wiley-Blackwell; £170.99 (eBook); pp. 608;
ISBN: 978-1-119-82734-4

This second edition of *Essential techniques of alveolar bone augmentation in implant dentistry* is a surgical guide that aims to provide clinicians with necessary surgical augmentation techniques to allow for adequate bone width and height before placing a dental implant. The previous edition was in two volumes; however, this is a single volume combining the best material from the previous edition along with new material and techniques. This edition is aimed at young practitioners at the start of their implant training with a step-by-step teaching approach. The book is well laid out with photographs, histological slides and tables complementing the text, which engages the reader on the topic.

The book is made up of seven sections. The first section which is an introduction to essential clinical knowledge gives a background on bone biology and wound healing as well as revisiting basic surgical anatomy of the jaws. There is also a segment that discusses radiographic evaluation of the ridge including the use of CBCT. This section ends by discussing different incision designs and soft tissue flaps as well as considering the biological rationale of surgical bone augmentation procedures.

Section two focuses exclusively on ridge preservation and discusses different types of bone graft materials and surgical extraction techniques to minimise bone loss. Sections three and four explore ridge augmentation in the horizontal and vertical dimensions and discuss the use of membranes, block grafting, ridge split expansion procedures, sinus lifts, use of titanium meshes in the augmentation process and how orthodontic extrusion may help vertical ridge enhancement.

Section five discusses various techniques used for soft tissue grafting and how to create ideal soft tissue contour and thickness, as well as ways to manage the papilla. Section six focuses on alternative techniques for bone augmentations, including short implants, zygomatic and pterygoid implants. Finally, section seven is a brief overview of tissue bioengineering, discussing important topics such as guided tissue regeneration and briefly the use of stem cells.

Overall, this edition provides a comprehensive and detailed summary of the fundamental aspects of bone augmentation in implant dentistry. The book is well laid out in defined sections making it easier for the reader to drop in and out of sections as and when they need to refer to them. There are clinical cases throughout the book that demonstrate the techniques described for readers to learn from, making the book more clinically relevant and applicable.

Ammar Ahmed Zaki