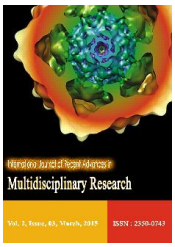

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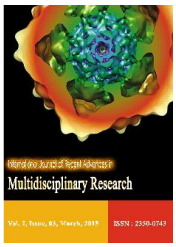

Research Article

CRACKED TOOTH SYNDROME- A REVIEW

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ABSTRACT

Cracked Tooth Syndrome is a common condition. In a major percent of cases etiology is primarily the after effect of a dental procedure .The patient is often unable to identify the offending tooth or quadrant involved, and may report a history of a dental procedure with unsatisfactory results. Clinicians need to have a thorough understanding regarding this concept to mitigate the commencement of crack propagation in tooth after a restorative procedure. This article is a comprehensive literature review about the incidence, prevalence, etiology and pathogenesis of CTS from contemporarily available data.

INTRODUCTION

Cracked Tooth Syndrome (CTS) is a group of symptoms characterized by incomplete fracture (infraction) of a vital tooth that involves the both enamel and dentin and occasionally extends into the pulp (Cameron, 1964). It is also known as green stick fracture (Sutton, 1962). They mostly occur in posterior teeth, especially in mandibular second and first molars. Sharp pain with no evident signs of any problem with the teeth is the hallmark of CTS. The Patients would often present a protracted history of pain of varying intensity; the origin of which may be difficult to locate. While intermittent pain on biting is the most consistent complaint associated with these teeth, cracks in teeth may present with a wide array of symptoms ranging from occasional discomfort to severe and prolonged pain. Symptoms are often dependent on the depth and direction of the crack and the tissue involved (Ehrmann and Tyas, 1990). Cracks in teeth may occur in both horizontal and vertical directions involving the crown and/or root. The etiology is generally excessive occlusal force, developmental defects or restorative procedures. These are usually incomplete fractures found in the crown of posterior teeth extending from an internal line angle at the floor of a restoration and often involving a marginal ridge with the fracture extending in a mesiodistal direction (Goel *et al*., 1992). The fracture commences in the crown and may terminate near cemento-enamel junction or extend apically into the root. The forces placed on the dentition during normal masticatory functions are small when compared to the maximum biting force. Anderson measured occlusal loads on mandibular molars using strain gauge on chewing meat, biscuit or carrots and it reached up to 14.9kg (Anderson, 1956), while De Boever reported a maximum force of 7.9kg with molars on occlusal loading (De Boever *et al*., 1978). This wide variation in readings suggests that functional chewing forces are variable from session to session and it changes with the consistency and viscosity of the food (Table 1).

The functional chewing force is very minimal when compared with the forces the stomatognathic system can exert. Arnold stated that the ratio of force on molars, premolars and incisors is 4:2:1, with far heavier forces on the most posterior teeth close to muscles of mastication (Attanasio, 1991). Dental tissues respond biomechanically to stresses and strains occurring during mastication. Hood *et al*, in his studies on deflection of premolar cups on occlusal loading found that deflection is as minimum as 11µm in unrestored tooth and progresses as the size or restoration increase from pit and fissure cavities preparation to class II preparation and complex restorations. Deflection is maximum in root canal treated tooth and it goes up to 27.5µm (Hood, 1991) (Table 2).

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Table 1. Load on mandibular molars while chewing

Author	Force
Anderson	Ranges from7.2kg to 14.9 kg (70.6 and 146 N)
De Boever	Forces between 2.4 and 7.2 kg (23.5 and 70.6 N)
Howell and Manly	90 kg (880 N),
Helkimo	10-73 kg (98-715 N) with an average of 45.7 kg (448 N) for males and 36.4 kg (357 N) for females

Table 2. Deflection of cusps in premolar teeth in various clinical situations (Hood)

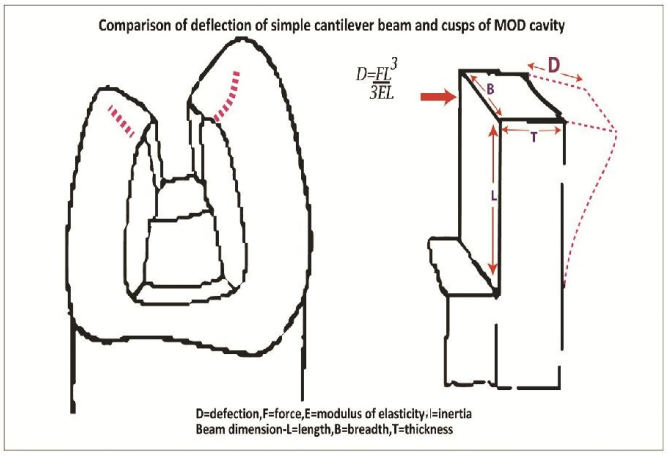
For the intact tooth	11 µm
For minimal occlusal class I cavities	16 µm
Minimal width MO cavities	20 µm
Minimal width MOD cavities	24 µm
extended MOD cavities	32.5 µm
After pulpotomy 10 kg (98 N) load	a) 27.5 µm deformation b) palatal cusp fracture after 18 µm deformation

Endodontically treated teeth become progressively weaker and are prone to further damage on masticatory function. Preparation of teeth by removal of caries has also been shown to significantly reduce tooth rigidity (Table 3).

Table 3. Cuspal movement on loading mandibular molar teeth at a rate of 20 N/s for 5 sec, generating a maximum load of 100 N (Pantivisai and Messer)

Intact teeth.	Low cuspal movement of up to 1 µm
MO	<2 µm
MOD	3-5 µm
Endodontic access	2-3 fold increase in cuspal movements, which were greatest in the MOD group.
On single cusp loading.	25-50 µm

In a class II cavity preparation, the cusps may be considered as cantilever beams. If the cusp height is double that of cavity floor, the deflection will increase by 8 times (because of the L cubed factor) (Figure 1). Similarly if cusp width is reduced by half, deflection will again increase eight times (because of the t cubed factor) (Homewood, 1998).



Change the 'L' in denominator of the formula to 'I'

Figure 1. (from HOOD[8])

There are two classic patterns of crack formation. The first one occurs when the crack is centrally located, and follows the dentinal tubules and may extend to the pulp (Figure 2); the second is where the crack is more peripherally directed and may result in cuspal fracture (Rosen, 1982) (Figure 3). Most fractures tend to occur in a direction near parallel to the forces on the cuspal incline.

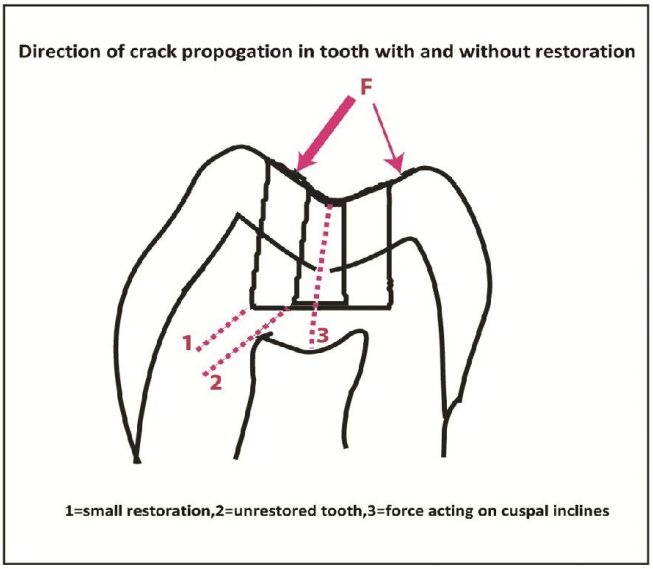


Figure 2. (from HOMEWOOD[9])

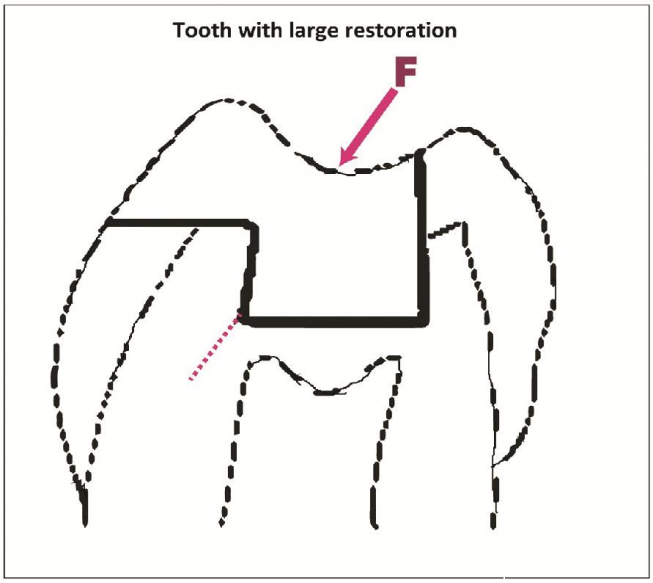


Figure 3. (from HOMEWOOD[9])

Hence, with larger restorations, the crack tends to be more superficial and may produce less severe symptoms, or rather no symptoms. With smaller restorations the cracks can be much deeper and closer to the pulp and may produce more severe symptoms. If wedging forces are placed on both buccal and lingual cuspal inclines, the resultant crack may occur in the midline of the tooth and propagate towards the pulp, especially in unrestored teeth (Homewood, 1998).

Terminology and definition

The term Cracked tooth Syndrome was first coined by Cameron in 1964 (Cameron, 1964 and Cameron, 1976), who noted a correlation between restoration size and its occurrence.

Table 4. The etiology of cracked tooth syndrome

Classification	Factors	Clinical Situations
Developmental	Hypocalcification	Occurrence in unrestored teeth
Restorative procedures	Inadequate design features	Over-preparation of cavities
		Insufficient cuspal protection
		Deep cusp–fossa relationship
	Stress concentration	Pin placement
		Hydraulic pressure during seating of tightly fitting cast restorations
		Physical forces during placement of restoration
		Non-incremental placement of composite restorations
		Torque on abutments of long-span bridges
Occlusal	Functional forces	Large untreated carious lesions
		Cyclic forces
	Parafunction	Bruxism
	Masticatory accident	Sudden and excessive biting force
	Damaging horizontal forces	Eccentric contacts
Miscellaneous	Thermal cycling	Enamel cracks
	Foreign body	Lingual barbells
	Dental instruments	High speed rotary instruments

Mention is made in the earlier literature of pulpal pain resulting from incomplete tooth fractures, and also of "greenstick fractures" of the crown (Ritchey *et al*., 1957). Ellis defined it as "a fracture plane of unknown depth and direction passing through tooth structure that, if not already involving, may progress to communicate with the pulp and/or periodontal ligament" (Ellis, 2001). Cameron reported that only 75% of teeth with "cracked tooth syndrome" will have vital pulps. Many practitioners confuse the terminology by illustrating teeth with "cracked tooth syndrome" which are in fact teeth with vertical root fractures (Geurtsen *et al*., 2003).

Etiology of CTS

Tooth with developmental defects tends to develop cracks on normal occlusal loading or on a slight increase in biting pressure (Christopher *et al*., 2002 and Hiatt, 1973). Restorative procedures will structurally compromise the tooth structure. Normal occlusal contact occurring on extensive occlusal or proximo-occlusal intracoronal restorations may subject the remaining weakened tooth structure to crack (Bales, 1975). Deep cusp–fossa relationships due to over-carving of restorations, or cast restorations placed without proper consideration for cuspal protection, also render the tooth vulnerable. Excessive condensation pressures, hygroscopic expansion of amalgam, placement of retentive pins and extensive composite restorations placed without due care for incremental technique (resulting in tensile forces in the tooth structure due to polymerization contraction) predispose the tooth for fracture formation (Trushkowsky, 1991).

Over-carving of a restoration, with loss of appropriate occlusal contact can result in the extrusion of a tooth, altering the cusp-fossa relationship and resulting in fracture of the nonfunctional cusp (Christopher *et al*., 2002 and Bales, 1975). However the fracture of cusps may be, whether functional or non-functional, primarily associated with large intra-coronal restorations and carious lesions. Other iatrogenic causes of CTS include excessive hydraulic pressure induced on luting of crown or bridge. Long-span bridges exert excessive torque on the abutment teeth, leading to crack generation. Other common causes include masticatory accidents, such as biting on a hard, rigid object with unusually high force or excessive removal of tooth structure during tooth preparation. Para functional habits such as bruxism are also associated with the development of this condition. Thermal cycling and damaging horizontal forces or parafunctional habits have also been implicated in the development of enamel crack in such unrestored teeth (Christopher *et al*., 2002). The higher incidence of CTS in mandibular second molars may be associated with their proximity to the temporo-mandibular joint, based on the principle of the "lever" effect — the mechanical force on an object is increased at closer distances to the fulcrum [16] (nut cracker effect[1]). Eccentric contacts can also induce crack formation in tooth. These studies are in contrasts with the findings of Cameron, who claimed that the teeth involved were usually quite heavily restored (Cameron, 1964).

Incidence

Cracked tooth occurs primarily in adulthood. Cameron reported that 80% of 102 cracked teeth occurred with patients over 40 years of age (Cameron, 1964). Other reports suggested that teeth with large intracoronal restorations and mandibular molars were most commonly affected (Eakle, 1986). The wedging effect of the prominent mesio-palatal cusp of the maxillary first molar may also be taken into account as a predisposing factor (Geurtsen, 1992). The transverse ridge of the maxillary molars may provide structural reinforcement and accounts for the lower incidence of fracture in these teeth. The maxillary molars and premolars have a similar incidence of fracture, with the mandibular premolars being the least susceptible. The disto-lingual cusp of mandibular molars is the most susceptible cusp for fracture (Khera *et al*., 1990). The findings for the prevalence of cusp fracture in other teeth were not consistent. Nonfunctional cusps may be more susceptible to fracture than functional cusps. This observation is based on the cusp's dimension- functional cusps are significantly larger in a bucco-lingual dimension and are covered with a thicker layer of enamel. The functional cusps are supported on the inner and outer inclines by the opposing tooth on the other hand non functional cusps may be more susceptible to fracture from lateral excursive occlusal forces due to the lack of support from the outer incline. In molar, non functional cusps were found to have a steeper cuspal incline. As the cuspal inclines are the guiding planes for lateral excursive movements for group functional occlusal relationships these cusps may be subjected to greater occlusal forces. If other teeth in the arch have been restored with flatter cuspal inclines then the steeper cusps are further exposed to crack formation.

Pathophysiology

These symptoms can be explained by the hydrodynamic theory of pain (Brännström, 1986).

The movement of dentinal fluid stimulates mechano-receptors that are in close proximity to the odontoblast cell body, which then activate A-delta nerve fibers (faster myelinated fibers), resulting in a short sharp pain. The pain is produced upon movement of dentinal fluid when the crack is opened by pressure on the cusp, and it subsides as the fluid moves back on releasing the pressure Seventy to eighty per cent of nerve fibers entering the pulp are non-myelinated C fibers (Trowbridge, 1986). These fibers are slow-conducting and produce a dull, poorly localized sensation and are activated by inflammation, heat and mechanical deformation. These C-fibers are usually activated by stimuli which cause actual pulpal damage. A tooth with a painful pulpitis can present with a severe, sharp pain, indicative of A-delta fiber activation followed by a prolonged, dull ache that radiates throughout the jaw, suggestive of of C-fiber activation. Classically the symptoms related to these teeth are pain on biting and sensitivity to thermal changes; particularly pain associated with the release of pressure, 'rebound pain' is also a consistent finding. Piezo electric current generation due to friction between the apatite crystals during movement could be another cause of pain.

Management

Treatment of CTS will depend on the position and extent of the crack. Management options vary according to clinical need, from replacement of the fractured cusp with a simple restoration to placement of an extracoronal restoration with adequate cuspal protection or root canal treatment.

Conclusion

Every practitioner should be aware of the nuances of CTS, and the condition must always be considered when a patient complains of pain or discomfort on chewing or biting with no apparently visible defects. A thorough case history will provide crucial guidance for a diagnosis. Careful clinical examination and inspection, supplemented by specialized tests, will be conclusive.

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