The Cracked Tooth Syndrome

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THERE IS a well-known clinician and lecturer who is frequently introduced to dental societies as the man who has written more, published more, lectured more, and just plain talked more on the subject of toothache than any other man, living or dead. He stresses that of all the causes of toothache, pulpitis and acute periapical abscesses account for 87% of the incidence, and 90% of these are due to neglected caries which has been allowed to involve the pulp. "All that is needed to prevent these toothaches," he stated, "is to simply eliminate the caries".[1]

Recently, he was rather embarrassed to admit to have lost two upper molars due neither to neglected caries, periodontal disease, nor any other discernible local cause. These serviceable teeth supported inlays with sufficient protective bases for a quarter of a century. There was just no apparent reason for their pulps to die.

The first hint of trouble was that occasionally, as he was eating and happened to bite on a hard piece of food, there was a sharp, very disconcerting pain for just a moment. As time passed, these episodes occurred more frequently. Numerous examinations were made, including x-rays and pulp vitality tests without disclosing the reason for the difficulty. As the years passed, the symptoms became more pronounced and the transitory pain gave way to dull persistent ache lasting for an hour or longer. The electric pulp test was no longer positive, and there

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was slight radiographic evidence of periapical tissue change.[2] Such a case history exemplifies the "cracked tooth syndrome."

Ritchey, Mendenhall, and Orban,[3] Sutton,[4] and Cameron[5] repeatedly emphasized to the dental profession that the phenomenon of an *incomplete tooth fracture* does occur, and that it can create a very difficult and frustrating diagnostic problem. Whenever the dentist is confronted with otherwise unexplainable discomfort of this nature, the possibility of an incomplete tooth fracture must be considered.

CLINICAL CHARACTERISTICS

Experienced clinicians state that these patients present some of the most bizarre symptoms encountered in clinical practice, ranging from an unexplained, vague discomfort, or a mild pain occurring during mastication over a period of years, to a severe pain which may be relatively constant, or even a quick unbearable stab lasting for only a few seconds.[3,5,6,7,8]

The predominent symptom and most frequent complaint is discomfort or pain elicited at the initiation or release of chewing pressure. Patients may complain of sensitivity to thermal changes or chemical changes while chewing sweet or acidic food. Frequently, patients express no association of sensitivity with any environmental stimuli.

However, the symptoms gradually intensify with time, becoming progressively annoying with increasing frequency and duration, and eventually manifest the excruciating symptoms of a severe toothache.

Incomplete fractures of the posterior teeth rarely occur in persons under 40 and many involve non-carious, unrestored teeth, making it difficult to believe that a problem exists. Several of the teeth in an affected quadrant may respond abnormally to diagnostic tests, but in the majority of cases none of the teeth respond abnormally to the diagnostic tests whether they be thermal, percussive, radiographic, or vitality, thus adding to the difficulty in diagnosis.

Percussion alone, surprisingly enough, is usually not helpful, yet biting on a foreign object such as an applicator stick, cotton roll, or a small rubber wheel, may give the spreading or torquing action needed to elicit pain. One can be reasonably certain that whenever a sharp pain is elicited upon the release of biting pressure, the portion of the tooth under the object is partially separated from the rest of the tooth by an incomplete fracture. The pulp tester customarily gives a normal reading unless the pulp is directly involved. Thermal tests may be

helpful only if a hot or cold stream is played directly upon the tooth, but hot gutta percha or ice are usually valueless. [3,5,6,7,8]

If the fracture does not involve the pulp initially, the symptoms continue as the crack gradually deepens. Dramatic relief may be obtained either by the completion of the fracture or by inclusion of the fracture in the preparation of the tooth during a restorative procedure. If the deepening fracture in the dentin eventually spreads and permits a separation of a cusp, it may cause a sudden lancinating pain. After a cusp has broken away, even though sensitive dentin is exposed, the symptoms by comparison may be temporarily relieved. The symptoms can be intensified, however, if the pulp of the tooth is further aggravated by additional cutting procedures, which may at the same time progressively weaken the tooth.

If the pulp is eventually involved in the fracture, the symptoms usually become intensified to an excruciating pulpitis as the pulp becomes infected and abscessed.[3,5,6,8]

Massler[9] has pointed out that pain is a very poor indicator of pulpal status in terms of the degree or type of pulpal inflamation and repair potential. Numerous histologic studies have shown no correlation between clinical appearance of the deep dentinal lesion, symptoms of pain, and pulpal pathosis. Massler feels that part of the misconception of clinicians who relate pain to pulpal status is their failure to differentiate between the sharp lancinating character of dentinal pain from the more dull throbbing quality of pulpal pain. He further establishes the fact that pure dentinal pain may occur even under a very superficial lesion at the dento-enamel junction in which there is no corresponding evidence of pulpal inflamation.

TYPICAL CASE HISTORIES

CASE 1. A 62-year-old white female patient experienced an atypical facial neuralgia for several weeks. On examination, the crown of a mandibular right second molar contained an occlusal gold restoration and a fracture in the enamel extending through the mesial marginal ridge and progessing apically down the mesial surface of the crown. There was no radiographic evidence of pathology, but the crack was such that there was no consideration given to salvage. The tooth was extracted.

The pulp was described as chronically inflamed with foci of necrosis, massive pulp stones, and an atrophic, degenerative odontoblastic layer. Colonies of microorganisms were present. A separation

within the dentin, possibly representing a portion of the clinically apparent fracture, was detectable on the microscopic section.

CASE 2. A 60-year-old white male patient had endured a long history of pain in the area of the mandibular left second molar which exhibited considerable occlusal attrition but no evidence of caries or restorations. Eventually, the patient presented with a draining buccal fistula, radiographic evidence of bone rarefaction, and a mesiodistal split of the crown of the tooth, which was also extracted.

The pathologist described a non-vital pulp chamber filled with debris, a thick layer of irregular dentin covering the roof of the pulp chamber, and numerous colonies of microorganisms.

DISCUSSION

It is impossible to set up a clinical research project to further study the "cracked tooth syndrome" in which incompletely fractured and painful teeth are left *in situ*. What information then is presently available which will permit us to extrapolate from conditions simulating the "cracked tooth syndrome" and provide us with some understanding of the pathology of the latter?

When an incomplete fracture occurs, the time interval is considerably shortened as compared to advancing caries in which the usual environmental irritants, such as saliva, bacteria, and chemicals, manage to reach the pulp. The closer the fracture line approaches the pulp, as it extends into dentin, and through the dentin to the pulp, the more critical the situation and the poorer the prognosis. The degree of fracture is not critical but only whether or not the pulp has been involved and made accessible to the oral fluids and microbiota. The irritants have immediate access to the pulp by entering a fracture, and the pulp does not have the usual time to protect itself with the formation of reparative dentin.[10]

The affects of saliva on exposed dentin and its relation to the "cracked tooth syndrome"

Fish,[11] Van Huysen and Gurley,[12] and Brännström[13] have all shown how irritating saliva and its constituents are to the pulp. Fish described, in dog and monkey teeth, the occurrence of intrapulpal foci of chronic granulation and abscess formation beneath cavities left open to saliva for 8 to 12 weeks. Van Huysen and Gurley demonstrated, in young dog teeth, marked leukocytic infiltration of the pulp as early as four days. Brännström, by grinding off the buccal cusp of premolars to obtain a large plane surface of transected dentinal tubules,

found that the dentin surfaces became more sensitive to touch after exposure to saliva for one week, and that the subjacent pulp contained many leukocytes.

Lundy and Stanley[14] found in 28 human teeth that had received Class V cavity preparations, cut at 250,000 rpm (Borden high speed handpiece) with an air-water spray and exposed to saliva for 1 to 11 days, that the amount of pulpal pathology was quite severe initially, and intrapulpal blister and abscess formation occurred in more than one third of the specimens (Figs. 1 and 2).

It was also obvious that 1.5 mm or more of remaining dentin between the floor of the cavity and the pulp considerably reduced the intensity of the response, apparently due to salivary exposure, and that preoperatively formed irregular dentin usually prevented a re-

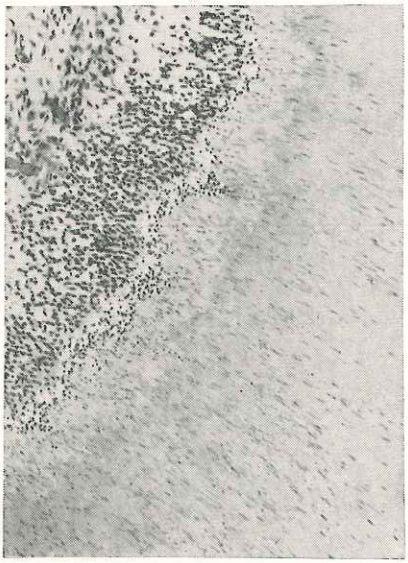

Figure 1 — 661064-10-3. One day postoperative. R.D. 1.28 mm. Specimen prepared at approximately 200,000 rpm with an air-water spray, a No. 2 round bur, and exposed to saliva for one day. Note the number of cells displaced into the dentinal tubules and the accumulation of leukocytes between the predentin and odontoblastic layers. Mag., x100.

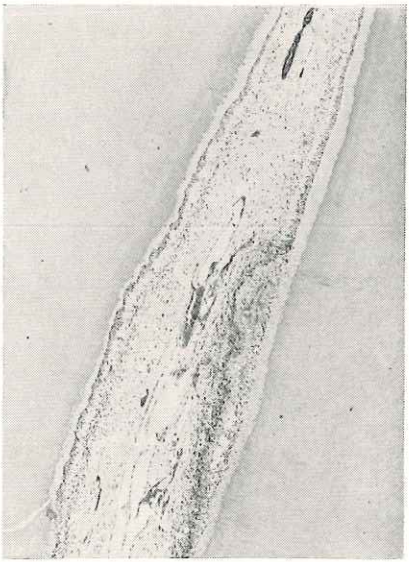

Figure 2 — 661070-10-4. Eight days postoperative. R.D. 0.89 mm. Specimen prepared at approximately 200,000 rpm with an air-water spray, a No. 2 round bur, and exposed to saliva for eight days. A purulent exudate has lifted off the odontoblastic layer and created an intrapulpal blister. Mag., x40.

sponse. If irregular dentin was found under only part of the cavity preparation, the response would be characteristically reduced or absent beneath only such irregular dentin.

In those specimens exhibiting the most severe histopathologic changes, the teeth had responded more quickly to cold stimuli.

The effects of fluid movement within exposed dentinal tubules and its relation to the "cracked tooth syndrome."

The movement of tubular fluids by chemicals, a jet of dry air, dry heat, absorbent paper, and reduced pressure cause tooth pain.

When Anderson and his associates found that a concentrated sugar syrup evoked pain in dentin, it seemed to them that the osmotic characteristics of solutions might have some bearing on their effectiveness to produce dentinal pain.[15] Anderson and Ronning[16] therefore applied the following solutions to human dentin with osmotic pressures ranging between 25 and 2760 atmospheres: ethanol, urea, glycerol, dextrose, NH_4Cl, NaCl, $CaCl_2$, and syrup. The solutions with high osmotic pressures like sugar, syrup, and saturated $CaCl_2$ solutions probably caused pain because they were capable of causing gross osmotic pressure disturbances within the dentinal tubules.

Anderson, Matthews, and Gorretta[17] demonstrated *in vitro* that saturated $CaCl_2$ caused fluid to move through the dentinal tubules towards the cavity preparation at a maximum flow rate of 4.8 nanoliters/second. However, after repeated applications of this solution a decline in flow occurred, coinciding with the earlier findings by Anderson and Ronning[16] that a reduction in pain-producing effects occurred with repeated applications of the test solutions *in vivo*.

Anderson and his associates concluded that pain production and fluid movement through dentinal tubules were related phenomena,[17] and that there are apparently no nervous elements within the dentin. When pain is evoked it is probably due to stimulation of receptor mechanisms in the pulp by a disturbance transmitted through the tubules by non-nervous elements.[18,19]

Ronning[19] observed that tubules, opened by fracturing the dentin, readily allowed dye diffusion both outward and inward but that tubules opening on to a cut surface did not. This inability of the cut surface to transmit dye is probably due to a narrow superficial barrier of precipitate induced by alterations of the protein content of the dentinal tubules. A similar phenomenon can be produced in dentinal tubules opened by fracturing if they are subjected to protein precipitants or heat. Evidently this narrow superficial barrier is of a

temporary nature; otherwise, the severe pulpal lesions due to saliva in the unrestored teeth of the study by Lundy and Stanley[14] would not occur.

Brännström and Astrom,[20] like Ronning,[19] showed that patients presenting dentinal surfaces prepared by drilling responded poorly or negatively to the stimulus of absorbent filter paper. Dentinal surfaces exposed by fracturing, however, being free from debris occluding the dentinal apertures, evoked severe pain.

Brännström[21] demonstrated that pain could also be evoked by subjecting exposed dentin to a jet of dry air, dry heat, or reduced pressure. He conjectured that these technics caused pain by disturbing, moving, or deforming the tubular contents through evaporation or changes in pressure. *In vitro*, he[13] demonstrated that the outward flow of fluid from the apertures of exposed dentinal tubules was considerable if the dentin surface was continually subjected to air drying, heat, or negative pressure. But with *in vivo* studies, the pain often disappeared with prolonged exposure to an air jet, and the dentin became subsequently insensitive to scraping with a probe.

For several years,[21,22,23] Brännström believed that the cessation of pain was related to the displacement into the dentinal tubules of the odontoblasts subjacent to the cut tubules, thus impairing some receptor mechanism. He also felt that the flow of the tubular contents away from the pulp might be the reason for the odontoblastic displacement. Later, however, he found that the dentinal surface became even more sensitive to touch after it had been exposed to saliva for one week in spite of earlier destruction of large parts of the subjacent odontoblastic layer.[13]

The effects of caries and bacteria on exposed dentin and pulp tissue and its significance to the "cracked tooth syndrome"

Pulp tissue mechanically exposed to saliva and left untreated in germ-free rats will routinely bridge the exposure with new dentin within three weeks. Only in the total absence of bacteria does this repair phenomenon occur. But pulp tissue similarly exposed and left untreated in conventional laboratory rats possessing the usual oral bacterial flora will consistently become non-vital due to bacterial infaction.[24] A question that must be asked and remains unanswered at this point is why human saliva exerts an initially severe pulpal response that saliva in germ-free rats does not.

Similarly, in man, if a fracture involves a pulp horn, bacteria readily gains access to the pulp. Even if the fracture line merely

comes close to the pulp or pulp horn but is separated by a thin layer of dentin, bacteria may enter via patent dentinal tubules.[6,10] However, long before the bacteria, *per se*, reach the pulp their enzymes, toxins, and more particularly their organic acids can span the remaining tubular distance and cause pulpitis. As caries advances through mineralized dentin the acids produced by the cariogenic organisms can be rendered non-irritating by the inorganic compounds of dentin.[25] But in the terminal stages of the carious process, where there is only decalcified dentin between the acidogenic bacteria and the pulp, the acids need only to diffuse through a small amount of this decalcified tissue in order to irritate the pulp.

Reeves and Stanley[26] in 1966 correlated the degree of dentinal carious involvement in terms of depth of bacterial penetration in dentinal tubules and degree of pulpal pathology associated with the carious lesion. They measured the distance between the point of deepest penetration by the bacteria and the pulpal tissue. As long as the bacteria remained more than one millimeter from the pulp, the disturbance to the pulp tissues was insignificant. Once the carious microorganisms approached within 0.5 mm of the pulp, the severity of the pathology became significant, and when the irregular dentin itself was invaded by organisms, the pulp became generally diseased with abscess formation and chronic granulation tissue.

Lundy and Stanley,[14] in a study to determine how quickly oral organisms penetrate patent human dentinal tubules, found that a plaque of microorganisms formed on the floor of unrestored cavity preparations within 48 hours, but that no organisms actually penetrated the opened dentinal tubules until the sixth day (Fig. 3). After 210 days the deepest penetration of the organisms was 3.00 mm., but the average penetration was only 0.52 mm. after an average of 84.2 postoperative days. Apparently, bacteria move through patent dentinal tubules at no great speed. Organisms were found in the pulp tissue only when an actual exposure was present. From this information, one can readily understand the critical nature of the situation when a fracture extends close to the pulp, more so than the presentation of exposed dentinal tubules.

RECOMMENDATIONS

In order to facilitate the diagnosis of a "cracked tooth syndrome," the dye application technic of Viener[7] is valuable. After removing the restoration, a dye such as methylene blue is applied to the tooth, and previously undisclosed cracks may be discovered because they stain darker. If the dye test is at first negative, one can

apply a generous amount of methylene blue and seal the cavity with ZnOE to which additional dye has been incorporated. When the temporary restoration is removed several days later, a careful examination may then reveal a previously unstained crack.

Also some use might be made of the test solutions of Anderson and Naylor[18] to examine teeth suspected of being fractured. Such agents as acetyl choline, KCI (potassium chloride), histamine, and 5-hydroxytryptamine creatinine sulfate do not evoke a response when confined to dentin but readily do so when contacting the pulp tissue.

To hopefully reduce the incidence of the "cracked tooth syndrome," one might consider some of the dynamics of cavity and crown preparation, especially at the bur (or diamond stone) — tooth interface as described by Nelson.[27] As the edge of the blade enters the enamel and dentin, ideally only the energy required to shear apart the molecules of the tooth should be applied to the tooth. However, energy is usually applied in excess, resulting in considerable frictional heat causing plastic and elastic deformation of the tooth surface. Because enamel is so brittle and hard (about as hard as bottle glass) its elastic and plastic deformation during cutting is small. However, the dentin surface is changed considerably by the excess energy not involved in the actual cutting. The heat generated may be low in terms of total calories but focally high enough to change the character

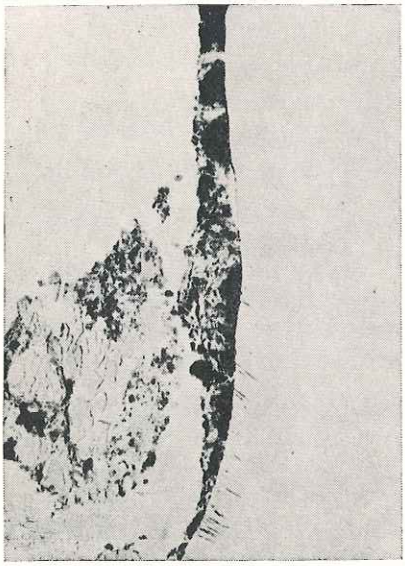

Figure 3 — 661065-5-2. Brown and Brenn Stain. Six days postoperative. R.D. 0.98 mm. The bacterial plaque (black) has accumulated on the floor of the cavity preparation. The dentinal tubules containing penetrating organisms (black) line up in picket-fence fashion. Mag., x125.

of the surface chemically and physically. All of these conditions can be needlessly aggravated by operating without an air-water spray.

Some modification of the technics of investigators like Mahler[28] and Craig and associates[29,30] might well be focused on the problem of the "cracked tooth syndrome." Rather than emphasizing how stress concentration causes failure of restorations, similar studies might enhance our knowledge of tooth fracture itself. Mahler points out that one should recognize that both the restoration and remaining tooth structure must resist structural failure if the restorative attempt is to be successful. The design of the preparation limits the structural effectiveness of both the restoration and the remaining tooth structure. Providing bulk for the restoration can only be done at the expense of tooth structure, and, conversely, conservation of tooth structure can be accomplished only at the expense of the restoration.[28]

Photoelastic stress analysis studies of amalgam restorations, inlays, and crowns have been most revealing.[29,30] Craig and his associates have demonstrated how damaging stress concentration can be and recommend its reduction by: 1) rounding axiopulpal line angles; 2) increasing the bulk of the restoration in the region of tensile stress; 3) having multiple point contact when an antagonistic tooth occludes with the abutment crown; 4) rounding cusps involved in a preparation; 5) avoiding deep groove carving near the center of the tooth; 6) making contact areas as broad as possible on tilted teeth; and 7) making margins of crowns full to increase the bulk of the restoration.

SUMMARY AND CONCLUSIONS

1. Clinicians should be aware that the phenomenon of incomplete tooth fracture is an increasingly more common occurrence.

2. The predominant symptom and most frequently associated complaint is discomfort or pain on chewing pressure.

3. A crack or fracture line shortens considerably the time interval necessary for the usual irritants, such as saliva, bacteria, and chemical substances to reach and affect the pulp.

4. The extensive nature of the fracture is not nearly as important as whether or not the pulp is accessible to the oral fluids and microbioti.

5. Saliva is very irritating to pulp tissue and precipitates a very intense initial response.

6. Certain chemical solutions when applied to exposed dentin can help to distinguish between dentin involvement and pulp involvement.

7. Solutions with high osmotic pressures, capable of causing gross osmotic disturbances in patent dentinal tubules, can cause pain when applied to exposed dentin.

8. Pain can also be evoked by subjecting exposed dentin to a jet of dry air, dry heat, reduced pressure, and absorbent paper.

9. Bacteria do not penetrate patent dentinal tubules, *per se*, very rapidly, the average penetration into the dentinal tubules being only 0.52 mm. after 84.2 days. Bacteria exert little influence on pulp tissues until they approach within 0.5 mm. of the pulp. When irregular dentin itself is involved by organisms, the pulpal pathology then becomes severe. Unless the fracture line comes very close to the pulp, the effects of bacteria will be delayed.

10. Symptomatology the first few months is no doubt due to the exposed dentinal tubules being subjected to physical and chemical irritants rather than to bacterial involvement *per se*.

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