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REVIEW

Persistent Cold-Sensitivity in Root Canal Treated Teeth: Mechanisms, Cracked Roots, and Clinical Implications

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Abstract: Root canal treatment (RCT) removes necrotic and inflamed pulp tissue with the objective of eliminating pain and sensitivity. Nonetheless, some treated teeth may have persistent sensitivity to cold. This symptom is counterintuitive and poses a diagnostic challenge. This article reviews current knowledge base on how cold sensitivity can persist in RCT teeth. Cold sensitivity may indicate residual neural tissue, periodontal/periapical innervation, altered sensations, cracked roots, and restoration marginal leakage. Cracked roots may indeed induce "cold-only" sensitivity. A narrative review, diagnostic and clinical management considerations are presented as well.

Keywords: endodontic, proprioception, sensitivity, post operative pain, dental pulp

Introduction

Root canal therapy (RCT) is a basic treatment in dentistry. It entails the removal of infected or inflamed pulp tissue, disinfection of the canal space, and filling of the canal space. A subsequent restoration may then be performed. An unexpected outcome is persistent sensitivity to cold in the treated tooth. Patients sometimes do, but rarely, report that a previously root-canaled tooth reacts to cold stimuli.[1–4]

What Follows is a Narrative Review

Odontoblasts line the pulp chamber and have processes that extend into dentin. The hydrodynamic theory states that microscopic dentinal tubules are filled with fluid and when a cold stimulus is applied to the external tooth surface, the temperature change causes fluid movement in the tubules, that stimulates mechanosensitive A-delta nerve fibers at the pulp-dentin interface.[5,6]

Understanding how cold sensitivity can persist or reappear, even after RCT is important for an appropriate diagnosis to avoid unnecessary retreatments.[7,8] This endeavor reports articles from literature searches in clinical post treatment endodontics with respect to mechanisms of cold sensitivity and the important role of cracked roots. It is important for the clinician to be aware of this phenomenon to prevent misdiagnoses or unneeded treatment.

A basic literature was done in PubMed using the keywords: cold sensitivity, post root canal treatment, cracked tooth roots, fractured tooth roots, cold symptoms in teeth. After exclusions for relativity only a few articles were found and are discussed and included in the references.

Case Report

A 35-year-old female presented with the chief complaint of "cold sensitivity" in her mandibular left molar. She was told by her previous dentist that her symptoms were coming from tooth #18 since #19 had been treated with root canal therapy and crown. (Figure 1) Nonetheless, she was referred to the endodontist who treated #19 who dismissed her symptoms. She then

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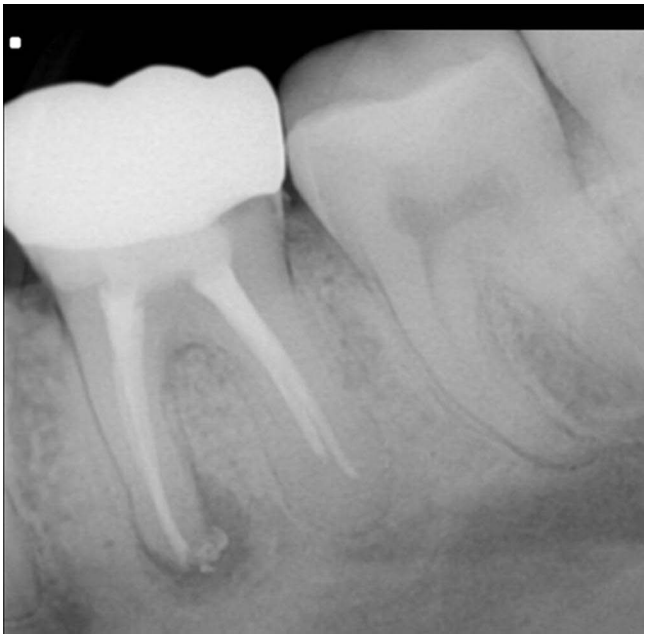

Figure 1 Periapical radiograph of the mandibular left first molar four years after endodontic treatment with a symptom of cold sensitivity.

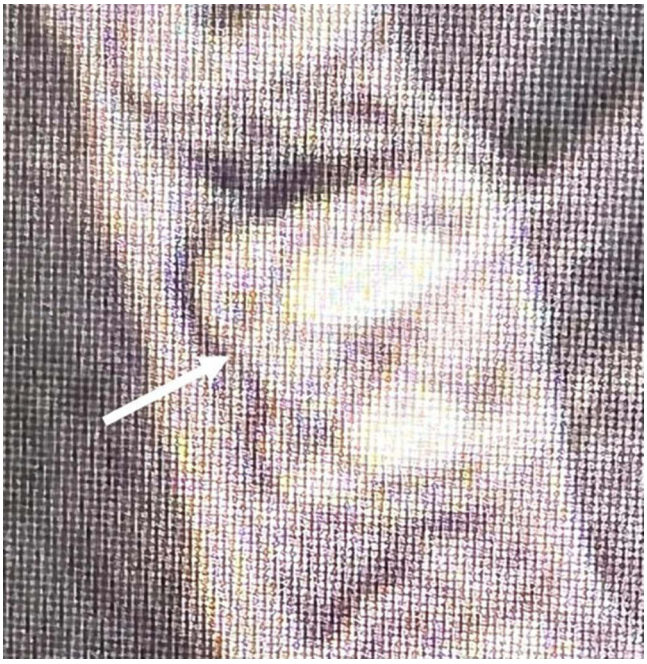

Figure 2 Cone beam computerized tomogram axial view of the molar showing a lingual root fracture (arrow).

sought a third opinion. The periapical radiograph showed an apical radiolucency at the M root apex. (Figure 1) A CBCT was taken that showed a "J" shaped lesion around the M root and a lingual root fracture (Figures 2 and 3). The tooth was deemed unrestorable and extraction was recommended. Figure 4 shows the immediate post root canal therapy (Figure 4).

Cold Sensation in Vital Teeth

Nociception is the physiologic process of primary afferent nerve fibers of the somatosensory system that recognizes noxious stimuli. Indeed, nociceptors are a specialized group of somatosensory neurons primary to the detection of

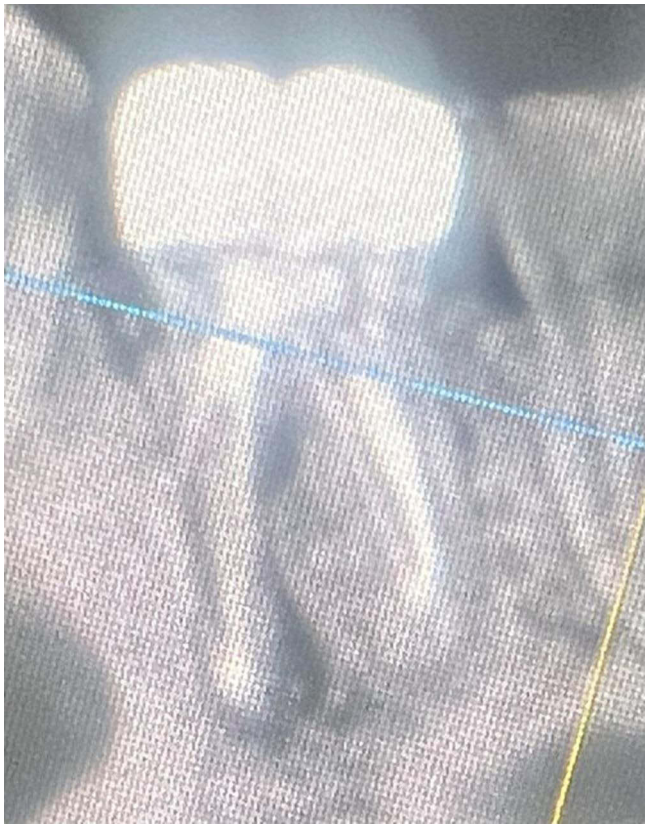

Figure 3 Cone beam computerized tomogram sagittal view showing a "J" shaped root lesion.

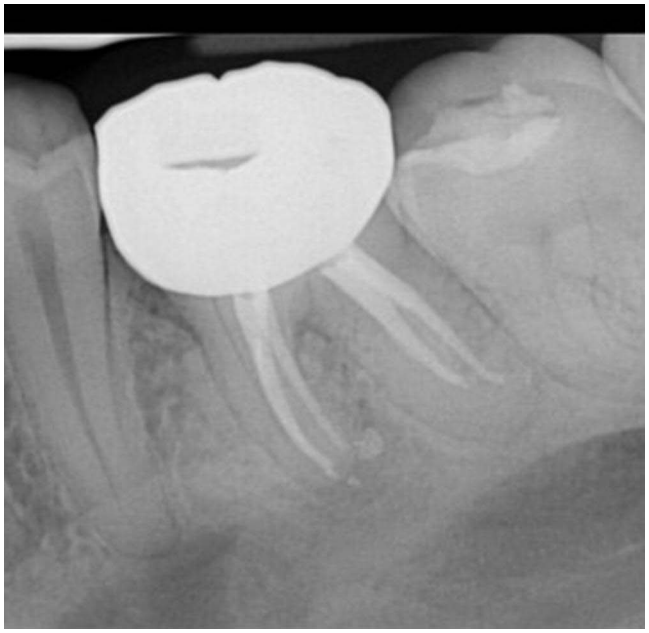

Figure 4 Periapical radiograph of the molar just after initial root canal therapy four years prior.

noxious stimuli. Transient receptor potential (TRP) channels are found in the membranes of some of these cells that are activated by a variety of stimuli. There are three members of the transient receptor potential (TRP) ion channel family, TRPV1, TRPM8, and TRPA1. These are molecular detectors of thermal and chemical stimuli that activate sensory neurons to produce acute or persistent pain.[9–15]

TRPs are found in a large family of cells with non-selective permeable ion channels that perceive environmental stimuli.[9–15] TRPs have a regulatory role in pulp physiology, oral mucosa sensation, dental pain nociception and salivary gland secretion. TRPs mediate signal perception and transduction of mechanical, thermal, and osmotic stimuli in odontoblasts, periodontal ligament, oral epithelial, salivary gland cells, and chondrocytes of temporomandibular joints. TRPs are also involved in dental pain nociception.[9–15] The various TRP channels play an important role in the transduction of external stimuli to intracellular signals.[14–16]

There are six members of the TRP channel, TRPV1, TRPV2, TRPV3, TRPV4, TRPM8, and TRPA1 and are the primary afferent nociceptors. They act as transducers for mechanical, chemical, and thermal stimuli as a first step for provoking pain. TRP ion channels activated by temperature, thermo TRPs, are important molecular actors in acute, inflammatory, and chronic pain. Heat activates four TRP channels (TRPV1–4), while cold activates two thermo TRP channels TRPM8 and TRPA1. TRPV4 perceives tensile forces, thermal and chemical stimuli.[14–16]

Many TRP channels have temperature-dependent gating properties. Thermo TRPs are thermo-sensors in the somatosensory system. These cause steep changes in depolarizing currents as a response to cooling or heating.[14–16]

Research on odontoblast TRPC5 cold transduction shows that cold sensitivity is mediated via these channels in healthy teeth. If similar pathways or residual structures remain after root canal cleaning and shaping, this may be due to cracks, via TRPC5 and TRPA1 channels.[14–16]

A-delta fibers are activated by heat and/or cold, play a role in periodontal nociception and non-odontoblastic neural cells that signal from periodontal ligament and bone.[6] Additionally, A-delta fibers will respond to cold stimuli applied to alveolar bone overlying the periodontal ligament.[6]

After RCT, in rare cases, cold sensitivity persists, this means that some other sensibility route or a residual sensory cell must be involved.

After Root Canal Treatment There Are Histological and Neural Changes

1. There is a loss of pulpal neural fibers but may be a persistence or remnant neural elements. When pulpectomy or complete RCT is performed, the main pulpal neural trunks are removed.[17] Nonetheless, some residual neural elements may persist in accessory or lateral canals. Additionally, sometimes regeneration or rearrangement of neural fibers near the apical or periapical region may occur.[17,18] Periapical neural changes occur after a pulpectomy and there is a derangement of the periodontal plexus around the apical third of the root. Neural branching, sprouting, or axonal growth may persist for a year.[17,18]
2. Inflammation and periapical healing.

   RCT aims to resolve infection and inflammation in periapical tissues. With an appropriate obturation and coronal seal, peri-radicular tissues generally heal, with a decrease in inflammatory cell infiltrate and apical bone regeneration. Nonetheless, when there is leakage of the fill coronally or apically, fill material overfill, or residual bacteria, then inflammation may persist, potentially stimulating local neural tissue.[19] If there is a coronal restoration with microbial leakage and fluid ingress that reaches root dentin, this can stimulate residual dentin tubules or peri-radicular tissues.[10,19]

1. Periodontal ligament (PDL) and peri-radicular innervation

   After the pulp is removed, the PDL remains innervated. The PDL contains mechanoreceptors, nociceptors, sympathetic fibers, and other sensory fibers. In the case of inflammation, infection, or mechanical forces, these fibers can be sensitized or undergo changes in connectivity or nerve sprouting in the PDL that respond to cold.[18]

1. Neural plasticity and injury response

   Nerves endings can respond to injury by sprouting, forming neuroma-like structures, or sensitize in response to inflammatory mediators. The neural response may change the peri-radicular plexus and activate with cold.[7,18,20]

1. Histological studies: Although direct histology of cracked roots in RCT teeth with cold-only sensitivity is sparse, studies of periapical and PDL tissues show increased innervation and inflammation in response to residual infection.[21,22]

Mechanisms for Cold-Only Sensitivity in RCT Teeth

1. Residual or accessory pulpal tissue or nerves
   a. There may be pulp remnants, sensory afferents responsive to cold, in lateral or accessory canals. These may not be well perfused by vasculature, but may respond to cold stimuli if a direct thermal stimulation via a crack or restoration leakage.[18,19]
   b. In appropriately done RCT, all pulpal tissue is removed, but sometimes the entirety of the pulp is not fully extirpated, especially when there are complex pulpal anatomies.[17]
2. Extension of stimuli to PDL and peri-radicular tissues
   a. Cold applied to a crown or a cracked root might conduct the thermal stimulus through dentin to the PDL or peri-radicular tissue. The sensory fibers in the PDL or adjacent apical bone can respond. Because these neural fibers have different conduction thresholds, the response may be to preferentially cold only.[23]
   b. Periapical nerve sprouting in response to residual inflammation may produce hypersensitive fibers.[24]

1. Fluid conduction via cracks or micro-fissures
   a. A hallmark of cold sensitivity in vital teeth is hydrodynamic stimulation of dentin tubules. In an RCT tooth, dentin tubules may be exposed at root surfaces as with gingival recession or along a root crack. A cold stimulus may cause inward or outward fluid shifts in tubules, generating a mechanosensitive signal in the PDL or any residual pulp.[25]
   b. Cracks may act like conduits, allowing the cold stimulus to reach deeper tissues, more readily than heat. This depends on thermal conductivity of the dentin and the fluid movement response.[21,22,25] Microcracks may enhance sensitivities.[21,22]
4. Altered thermal conduction and insulation
   a. Restorative materials, post and core build-ups, crowns, posts, cracks, and dehiscent root surfaces can change the thermal conductivity pathways. A crack may reduce dentin insulation properties and allow cold to reach tissues quicker. Heat tends be dissipated. Thus, cold may produce sharper, more localized stimuli.[10]
5. Inflammatory sensitization can lead to lower sensory thresholds
   a. When periapical or periodontal inflammation is present, inflammatory mediators such as prostaglandins and other cytokines can reduce the threshold of sensory fibers. Cold, which ordinarily might not cause a neural threshold to be reached, may do so in the presence of inflammation.[7,26]
   b. Neural plasticity as with sprouting, can change ion channel action. This may increase the responsiveness of cold-sensitive.[7]
6. Selective nerve injury or loss of neural fibers
   a. Damage or loss of specific types of nerve fibers may lead to the persistence of only certain modalities. If fibers mediating heat or pressure are completely damaged and cold-sensitive fibers survive then their actions may be exaggerated.[27]
7. Overfilled materials or irritants
   a. Overfilling beyond the tooth apex of materials that induce chemical irritation can provoke neural reactions in the PDL. This may sensitize periapical nociceptors, potentially making them respond to cold. Though such reactions often also produce spontaneous or heat-related pain, in some cases cold stimuli might be the only provoking trigger.[28]

Types of root cracks and fractures[23,29,30]

- Vertical root fractures: cracks that extend longitudinally from the crown/root toward or through the root may penetrate PDL.
- Lateral or oblique cracks: possible under occlusal loads.
- Microcracks and craze lines: very small cracks are usually not visible on radiographs and may be present in root surfaces.

Cracks may expose internal dentin and tubules that may form pathways for fluids or bacteria and thus allow thermal penetration.[2,23]

1. Localization via PDL

   Cracks that extend to the PDL allow thermal, mechanical, and fluid stimuli to reach PDL cells. The PDL contains mechanoreceptors and nociceptors which can respond to cold. PDL fibers are more responsive to movement or pressure, but in a cracked root, thermal conduction may generate fluid shifts that these fibers detect. A cold substance may trigger pain.

1. Crack as a thermal conduit

   Cold applied outside the tooth can travel more easily along a crack in the form of a fluid or air with direct exposure of dentin. Cracks allow faster transmission of cold to deeper parts of the tooth and the PDL and root apex.[16,23,29]

1. Exposure of dentin and tubules

   Cracked root surfaces may expose superficial and deep dentin tubules. A cold substance can induce fluid movement in exposed tubules that may stimulate any remaining nerve fibers either in residual pulp tissues or in the PDL to stimulate nociceptors.[31]

1. Path for bacterial ingress and inflammation

   Cracks can allow bacteria to penetrate, resulting in localized inflammation near the crack margins, PDL, or periapical region. Local inflammatory mediators can sensitize nerve fibers. These fibers may be more responsive to cold.[6,16,26]

1. Selective activation

   Cold may produce sharp, rapid fluid contraction, generating fluid movement in dentinal tubules which activates A-delta fibers. Heat may expand the tubule and dissipate. Heat may require a sustained thermal exposure to cause a neural activation. Sudden cold stimulus might be sufficient for a nerve activation in a cracked root so a cold stimulus may dominate.[6,16,26]

1. Dynamic effects

   In cracked roots, when the patient breathes cold air, drinks cold liquids, or has external cold contact, the temperature change may be more abrupt at a crack causing sudden contraction of dentinal fluid that reaches nerve fibers or remnants thereof.[15]

1. Transillumination may be useful in identifying a crack.[29,30]

Clinical diagnostic steps.

1. Confirm the source
   a. Isolation and cold testing of the RCT tooth separated from adjacent teeth with cotton rolls or rubber dam, to rule out referred sensitivity. Sometimes patients feel cold on one tooth but the actual responsive tooth is an adjacent vital tooth.[4,31,32]
   b. Use thermal stimuli such as ice or a proprietary product with careful isolation and comparative testing.[4,31–33]
   c. Electric pulp test may not elicit a positive response.[3,4,33]

1. Evaluate restorations and coronal seals
   a. Check margins of crown, fillings, temporary restorations. Poor coronal seal can allow cold exposure into dentin or cracks. Evaluate crown height, fit, and presence of leakage.[10]
3. Assess structural integrity of the root.[29]
   a. Plane film radiographs and CBCT may reveal fractures, cracks, vertical root fracture, or microfractures. Sometimes cracks are only visible under magnification.[34]
   b. Clinical signs: pain on biting, pain on release of the bite, periodontal "J" shaped pocketing adjacent to suspected crack and localized swelling. The "J" shaped periodontal lesion may be solitary.[35]
4. Check for residual infection and periapical pathology[26]
   a. Radiographic evidence of periapical radiolucency, widening of PDL, and bone loss.
   b. Symptoms: swelling, discomfort, and sinus tracts.
5. Look for possible overfilled materials or irritants
   a. Use radiographs to see if the canal sealing material is extruded. Chemical irritation could affect adjacent neural tissue.
6. Time course of symptoms
   a. Question the patient as to when the cold sensitivity started: immediately post-RCT, after placing the crown, or later. Ask if the sensitivity is worsening, improving, or static.

Differential diagnoses.[23]

- Referred pain from adjacent teeth
- Gingival recession exposing root surfaces of adjacent or same tooth
- Decay under crown/restoration
- Leakage of restoration
- Sinus involvement (especially upper posterior teeth)
- Neuropathic pain

Treatment considerations.[23,36,37]

- Monitor for improvement: in early postoperative period, mild sensitivity may resolve as inflammation subsides.
- Improve coronal restoration quality: if a restorative leakage is found it may need replacement or repair.
- Adjust, repair or replace a restoration if there are open margins.
- Address cracks: depending on severity, cracked root may require root amputation, bonded repair, or extraction.
- Medications and desensitizing agents: topical dentin desensitizers, anti-inflammatory therapy (NSAIDs), may be appropriate.
- Retreatment: residual infection or a missed accessory canal may need revision treatment.

Selective sensitivity to cold but not heat or pressure symptoms. The threshold and stimuli dynamics.

a. Cold stimuli can produce rapid inward and outward movement of tubule fluid but such fluid shifts are more abrupt with cooling and this causes contraction of fluid as opposed to heating which causes expansion and diffusion of the fluid. Cold is a more powerful sensitivity trigger for cracks in exposed dentin.[13]

1. Different conduction pathways
   a. Cold may conduct easily along cracks, whereas heat may dissipate slowly or be buffered by surrounding tissues or restoration materials.[13]
2. Neural sensitization to cold
   a. Cold-sensitive ion channels TRPC5 and TRPA1 may be overexpressed or have a lower activation threshold in the presence of inflammation or neural plasticity. Thus, cold stimuli that would ordinarily be subthreshold cause a reaction. Nonetheless, heat or pressure may require a higher magnitude to activate the damaged or residual pathways.[13]
3. Limited modality survival
   a. After RCT or injury, some nerve fiber types may be destroyed or lost preferentially. C fibers mediating heat pain may be more susceptible to injury, while fibers or structures capable of detecting cold remain or regenerate. Thus, only cold sensitivity remains.[13]
4. Stimulus localization
   a. Cold stimuli are often more focal such as with a cold drink and pressure or heat may be more diffuse. A crack may act as a focal conduit for cold, making the stimulus more intense locally. Heat and pressure spread and may not reach the neural threshold.[13]
5. Temporal aspects
   a. Cold stimuli often elicit immediate sharp responses, whereas heat may require prolonged exposure. If exposure is brief, cold may trigger a response while heat may not.[13]

Evidence: Experimental and Clinical Studies.

- Odontoblast TRPC5 channels: One study demonstrated that TRPC5 is necessary for cold transduction in healthy teeth, odontoblasts act as the direct site for TRPC5-mediated cold sensing.[13] This suggests that in vital teeth cold sensitivity depends upon odontoblast functionality. When the pulp is removed, odontoblasts lining the pulpal walls are also removed. Nonetheless, any residual odontoblasts such as in lateral or accessory canals, or periradicular tissue changes may indirectly recruit similar mechanisms.[13]
- Periapical neural changes after pulpectomy: One study observed that after pulpal necrosis and RCT, the neurons exhibit increased axonal branching and sprouting in the apical PDL zone, even when obturation is done.[18] Such changes could underlie enhanced sensitivity to cold stimuli via PDL innervation.
- Histological evaluation of teeth with and without coronal seal: One study looked at roots with or without coronal seal over 2–6 months after RCT. Those with coronal leakage showed significantly more inflammation in periradicular tissues.[38] Inflammation can modulate neural sensitivity.[38]

Apical Limit of Instrumentation & Obturation: One histological study showed that when root canal instrumentation and obturation remain at or short of the apical constriction, periapical tissues show more favorable healing. Extrusion of instruments, sealer or gutta-percha causes local inflammation and foreign body reactions. This kind of irritation could lead to neural sensitization of periapical nociceptors that respond to cold.[19]

Clinical Management (Table 1)

1. Clinical history and symptom characterization

Table 1 Cracked Root Case Scenarios

Scenario	Crack Type Location	Pathway for Cold Stimulus	Neural Substrate Residual Structures	Clinical Presentation
Vertical root fracture extending to PDL	Crack extends from root surface into PDL (longitudinal), perhaps near root apex or middle third	Cold liquid or air enters the crack; cold transmitted directly to dentin and PDL or apex	PDL nerve fibers or periapical nerve sproutings, possibly residual pulp nerve in lateral canal or accessory canal near crack	Sharp, momentary pain on cold (liquid or air), no response to heat, no pressure sensitivity; possible pocket or isolated bone loss on one side.
Microcrack in root surface, uncovered by gingival recession	Crown margin is sealed, but root surface crack near cervical root exposed due to gingival recession	Exposed crack allows cold air or water to affect dentin tubules adjacent to crack; fluid shifts	Tubules + adjacent PDL fibers; no pulp inside, but periradicular nerves and PDL sense mechanical/thermal shifts	Cold sensitivity when breathing or eating cold liquids; may not feel heat; symptoms worsen with cold exposure; no swelling or spontaneous pain.
Crack under a coronal restoration, not visible radiographically	Crack runs coronally under crown margin, partially into root dentin	Cold applied to crown (via restoration or margin) transmits via crack; restoration materials may conduct cold if thin or flawed	Accessory nerves or PDL near crack; possibly residual pulp tissue if canal system not fully sealed; also possible hypersensitive periradicular nerves from residual inflammation	Cold sensitivity localized to that tooth; discomfort when cold drinks; perhaps recurring inflammatory sign if bacteria enter; occasionally some tenderness but not obvious heat sensitivity.

Note: These scenarios illustrate how cracks create structural vulnerabilities, enabling cold stimuli to reach neural tissues that normally would be isolated.

   a. Time of onset: shortly after RCT or restoration delivery.
   b. Nature of cold sensitivity: sharp/momentary vs lingering after removal of cold stimulus.
   c. Sensitivity to heat or percussion or compression.
   d. Spontaneous pain, swelling, sinus tract.
2. Diagnostic testing[3]
   a. Thermal tests, cold and heat, of the tooth in question and adjacent teeth.[3,4,33]
   b. Percussion and palpation.
   c. Bite tests such as biting on a wooden stick or "Tooth Slooth" to detect cracks.
   d. Radiographs especially a CBCT to look for fractures, periapical lesions, overfilled material.
3. Evaluate restoration and coronal seal
   a. Evaluate restoration margins for recurrent decay and leakage and occlusion.
4. Assess for cracks
   a. Clinical signs: There may be pain upon release of biting, a localized "J" shaped periodontal pocket adjacent to a suspected crack, presence of sinus tract.
   b. Visual examination with magnification.
   c. Use dye, transillumination, CBCT.[29,30]
5. Treatment options
   a. Mild symptoms may resolve, nonetheless, conservative measures and oral hygiene may suffice. Informed consent is important.
   b. Coronal leakage may be repaired or require restoration replacement.
   c. Occlusion adjustment may be in order.
   d. A crack may be repaired.
   e. A significant crack, as with a vertical fracture that compromises the PDL and is irreparable, then extraction may be best.
   f. A delayed extraction of a cracked or fractured tooth may allow bacteria to vegetate in medullary bone and later colonize an implant placed onto that site causing a failure to osseointegrate.[39]
6. Patient counselling
   a. Explain that cold sensitivity in an RCT tooth is unusual, but not impossible.
   b. Discuss risks and benefits of possible retreatment vs extraction vs waiting. Waiting may allow medullary bacterial vegetation that may colonize a future implant placement.
   c. Monitor symptoms: worsening, new symptoms (heat, spontaneous pain), swelling should prompt a re-assessment.

Issues for Further Research

- Direct histological evidence of cold-only sensitivity in RCT teeth with cracked roots is lacking. Most studies are animal studies, or relate to vital teeth or periapical neural changes, but not exactly to this clinical phenomenon.
- The exact ion channel expression or molecular changes in periradicular or PDL nerves post-RCT especially in cracked roots are not well characterized. PDL fibers express TRPC5 after RCT/inflammation and cold sensitivity in those fibers may operate via different molecular mechanisms.
- The response of residual pulp tissue in accessory or lateral canals in individual patients is difficult to measure clinically.
- Diagnostic imaging for cracks is imperfect and many cracks go undetected until symptoms appear.
- There is little prospective data on outcomes when "cold-only sensitivity" after RCT is observed and many of these symptoms resolve spontaneously while others progress to complete root fracture.

Conclusion

Cold sensitivity in a root canal treated tooth is possible through several converging pathways. Residual neural or odontoblastic elements in may remain in accessory or lateral canals in the tooth root. Periapical or PDL innervation and inflammation may induce sensitized sensory neurons to produce a temperature sensitivity. There may be structural defects such as cracks, exposed dentin, or poor coronal margins may allow cold to reach deeper structures. Differential stimulus dynamics occur where cold has faster thermal conduction or fluid shifts that trigger a response faster than heat. Cracked roots play a central role. Cracks undermine structural integrity and expose openings for cold to reach responsive neurons.

Persistent cold sensitivity after root canal treatment is an uncommon but clinically significant phenomenon. Although pulp removal should eliminate thermal sensitivity that is mediated through the pulp, residual neural tissue in lateral or accessory canals or in periapical tissues, PDL innervation, structural defects such as cracked roots, and altered sensitivity thresholds from inflammation or neural plasticity may all contribute.

Clinicians faced with this symptom should methodically evaluate for cracks, leakage, residual infection, and restoration integrity. Management should be tailored to the situation. A conservative restoration or, nonetheless, an extraction may be indicated.

A long-standing root crack may allow deep penetration of bacteria into medullary bone that may vegetate and subsequently colonize a dental implant causing an early or late failure. Thus, a cracked tooth may be best extracted as soon as practicable.

Disclosure

The author reports no conflicts of interest in this work.

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