

*Medicinska edukacija/  
Medical education*

DIFERENT ASPECTS OF OROFACIAL PAIN  
(PART V)  
DENTAL CAUSE OF FACIAL PAIN

RAZLIČITI ASPEKTI OROFACIJALNOG  
BOLA (PETI DEO)  
ZUBOBOLJA KAO UZROČNIK FACIJALNOG  
BOLA

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*Abstract*

Pain is a reason for seeking dental treatment. Pain is also a reason for avoiding dental treatment. Just as dental treatment can rid a patient of pain, it can also be painful. Occasionally, the treatment may cause postoperative pain. To many people pain is closely connected with dental treatment. Even if some person has not experienced pain himself or herself, they willingly share „their own problems”, or use others’ real or imaginary experiences. Root canal treatment, or the more vulgar version a „root canal”, is currently used in the press as the standard for excruciating pain. A pain is equal to or worse than „a root canal”. You wonder how much damage this journalism expression has caused, how many unnecessary sleepless nights, how much worry, how many appointment cancelations?

*INTRODUCTION*

Dental pain is common. It has been estimated that in the North America 15 million working days are lost yearly due to dental pain <sup>(1)</sup>. Everybody knows what a toothache is. Everybody knows, except the dentist (?!), who is trying to figure origin of pain out whether it is pulpal or periapical pain or pain of another origin. One third of all dental emergencies are of pulpal or periapical origin. When you look only at the emergency cases with pain, approximately 90% of these are of endodontic origin. This does not mean that all these teeth need endodontic treatment. Extraction may be the treatment of choice if a tooth has no value for the whole dentition or it cannot be restored properly. Because endodontic cases dominate among patients in pain, to many clinicians the words endodontics and pain have become more or less synonymous. Even if the endodontic cases are in the majority among patients with acute oral pain, however, pain in the oral cavity does not equal endodontics. Tooth eruption problems, periodontal abscesses, temporomandibular joint disorders, sinusitis, and neurologic disorders are among the many conditions that must be considered.

Endodontics has during the years developed certain techniques and tools for examining patients: palpation and percussion tests, thermal tests, electric pulp tests, and mechanical tests. In most instances one single test does not give the full answer. It is necessary to rely on multiple tests and radiographs to make the correct diagnosis. The most important diagnostic tool is the dentist. The clinician must weigh the results of the tests, compare them to the patient’s history, interpret the radiographs, and then use his or her own knowledge and experience to come to a diagnosis. Often this process is fast. The clinician may reach the diagnosis in seconds. Sometimes, however, especially in cases with chronic pain, this can be a long process that involves many specialists.

Patients are different. It is wise to determine how a patient reacts to testing before the suspected tooth is tested. An easy way to find out the reaction pattern of the patient is to test an innocent tooth first. The palpation and percussion tests determine areas of tenderness usually related to inflammation. The tests to determine pulp vitality, specifically pulp sensitivity, are the thermal, electric and mechanical tests.

### *PALPATION*

An exacerbating periapical inflammation can be confirmed mostly by palpation of the buccal and lingual surfaces that cover the periapical area of the tooth in question. In cases with recessed gingiva it is often difficult to discriminate between apical and marginal tenderness. The use of an instrument smaller than a fingertip, for example, the end of a mirror handle, makes the palpation more precise.

### *PERCUSSION*

Teeth can respond to percussion for many reasons. A response means that somewhere in the periodontal membrane or in the surrounding tissues there is a sensitive area most likely caused by inflammation.

### *THERMAL TESTS*

Cold test is the most reliable clinical, noninvasive vitality test. Cold tests can be carried out with ice, ethyl chloride, or carbon dioxide ice. Heat test is useful only if the patient complains of pain or sensitivity to heat. Most commonly, heated base-plate gutta-percha is used.

### *ELECTRIC PULP TEST*

Just as the thermal tests cannot grade the response, it must be emphasized that electric tests are also strict yes or no tests. Numerical values given by an electric pulp tester do not mean that the instrument is precise beyond the yes or no level. Numerical values can be useful to compare test results after trauma if the tests are performed identically at different time periods. Electric pulp tests should be supplemented with another form of vitality test such as the cold test.

### *MECHANICAL TEST*

A mechanical test is the most reliable vitality test. Probing often reveals a vital response in a deep cavity<sup>(2)</sup>. If there is a carious cavity or an old filling that must be replaced, excavation without anesthesia tells whether the tooth is vital. In a tooth that cannot be tested otherwise, for example, due to a crown, a test cavity is a reliable vitality check. This is an invasive procedure and there must be a valid reason for performing it. It is necessary to explain clearly to the patient that the drilling is started without anesthesia as a test. The patient is told to tell the operator as soon as he or she feels something. It must be emphasized to the patient that it is a matter of feeling, not a matter of feeling pain, and that as soon as a response has been obtained, the tooth is anesthetized<sup>(3)</sup>. After proper explanation patients seldom insist on anesthesia. Anesthesia if given too early makes a correct diagnosis impossible.

A good history is the most important step toward a correct diagnosis. The most important thing is to listen to the patient. This cannot be emphasized enough. Almost always the key to the diagnosis can be found in what the patient tells you. It has been stated that if a clinician does not want to take the time to listen to the patient, it is better to refer the patient to a veterinarian. Veterinarians are trained to deal with patients who cannot give a history. Before any kind of treatment can be considered, a diagnosis must be estab-

lished. If the reason for the pain is not known, no treatment should be started. A treatment should never be started without a diagnosis.

Sometimes mistakes are made. For example, a patient presents with symptoms of pain and a pulpectomy treatment is carried out. The patient returns later and the pain symptoms are still the same. Obviously, endodontic treatment was initiated wrongly. On the other hand, the treatment has proved that this tooth is not the source of pain. One should take a step back and think and reexamine the patient. What are the symptoms? What is the patient really telling? Unfortunately, it happens often that the dentist looks at the problem with blinders and can see only teeth. It is easy to fall into the trap and treat the neighboring tooth (and then the next). If the tooth was treated based on the diagnosis made during the previous visit and it is obvious that the treatment did not solve the pain situation, it is possible that the cause of the pain does not involve any tooth. If the symptoms described by the patient do not coincide with the classic symptoms of pulpal and periapical inflammation the conclusion must be that the pain is emanating from something other than a tooth.

Acute pain is often of endodontic origin; chronic pain is mostly not. Longstanding pain seldom is caused by pulpal or periapical inflammation. Frequently a patient points to a root filled tooth when asked to pinpoint the location of chronic pain. It is also common that the patient states that the symptoms began after the root canal treatment. It may take some detective work to figure out whether the pain actually started after endodontic treatment or, which is mostly the case, the pain was present before the treatment. It is often found that the pain was the reason for the treatment even if in hindsight it may appear to have been wrong. As time has passed the patient has forgotten the true order of events and it becomes logical to blame the pain on a procedure. Too many teeth have been unnecessarily root filled by well-meaning clinicians who want to help the patient even if the origin of the pain is unknown. Also, endodontic retreatments are often carried out and the reason given is that the root filling was too short, too long, or had some other technical problem so it must be the reason for the pain.

It cannot be emphasized enough that unless there are obvious endodontic symptoms, there is no reason for a retreatment just because a root filling does not look like a text book illustration. Unfortunately, dentists are expected to help patients by doing something. It appears that if the treatment does not involve drilling, the patient feels that no real treatment has been done. This places a lot of pressure on dentists and in cases of uncertainty it may be easier to give in than to tell the patient that you do not know why the patient is in pain. Also drugs and different forms of treatments have a placebo effect. Chronic pain patients often experience relief for some time after a treatment. Unless the treatment is correct, however, the pain returns. It is essential to listen carefully to the patient and examine him or her carefully, and if it still is not clear why the patient is in pain, no treatment must be rendered. The patient can be given analgesics, a new appointment, or be referred to a specialist. Most importantly, however no treatment should be started.

In most instances when a patient considers an endodontic treatment to be the origin of chronic pain, it turns out after a thorough check of old charts that the pain was present before the endodontic treatment. There are rare instances in which pain develops sometime after pulpectomy and root filling or apical surgery, however. There is a persistent ache; no form of therapy seems to improve the situation. Marbach<sup>(4)</sup> has named this phantom tooth pain. Dental pulp amputation or extirpation may result not only in a lesion at the apex but also could influence the CNS. This pain can last long after the radiographic healing has occurred. It has been suggested that there could be a neuroma formation at the apex of the treated tooth<sup>(5, 6)</sup>.

### VITAL TEETH

Basically, three origins of pain are related to the tooth: pain that emanates from exposed dentin, pulpal inflammation, or periapical inflammation<sup>(7)</sup>. There may be combinations of these three groups. Dentin and pulp are related closely and it is absolutely imperative to look on dentin and pulp as one functioning unit. All procedures performed in dentin are essentially performed in both dentin and pulp.

Dentin is a unique hard tissue<sup>(8)</sup>. It is sensitive to touch and changes in temperature. Because enamel is not sensitive, this sensitivity of dentin has been a mystery (a). Historically three main theories have attempted to explain why dentin is sensitive.

The first theory deals with direct innervation. All other tissues that are sensitive contain nerves so it appears logical that dentin also should contain nerves. Early studies reported the presence of nerves in dentin from the pulp to the dentinoenamel junction<sup>10- 3S</sup>; however, it appears that at least some reports were based on artifacts. Later studies using transmission electron microscopy (TEM)<sup>(9-20)</sup> found unmyelinated nerve fibers close to odontoblastic processes. Byers and Kisch<sup>(15)</sup> and Lilja<sup>(21)</sup> found nerve fibers only in the innermost part of tubules in the circumpulpal dentin. Apparently the hypothesis that nerve terminals are situated at the dentin enamel border must be rejected.

A second theory focuses on odontoblasts and their close relationship with nerves. It was hypothesized that the odontoblasts could act as sensory receptors and transmit information to nerve fibers by way of synapses. It has been shown that dentin can be sensitive even if the odontoblasts are destroyed, however.<sup>(11, 12, 21-31)</sup>

A third theory is based on hydrodynamics. Bánóczy J.<sup>(32)</sup> suggested that nerve fibers could be stimulated by pressure from fluid movement in dentin tubules. Later Brannstrom<sup>(11-12)</sup>, Brannstrom and Astrom<sup>(13)</sup> and Ahlquist et al.<sup>(5)</sup> showed that rapid movement of dentin fluid causes pain whether the stimuli are osmotic, chemical, mechanical, or temperature related. Odontoblast processes and nerves are present in the inner third of dentin. External stimuli cause movement of the dentin fluid creating large shear forces in the inner part of the dentin.

The hydrodynamic theory is currently generally accepted. This theory explains how dentin is sensitive. The remaining question is why dentin is sensitive. It is not likely that nature has this elaborate system for dentin sensitivity so that

we shall be able to feel an open class five cavity. It appears that the hydrodynamic system and the intradental nerves are important for examination of what we chew and for control of masticatory forces (c). Aii experimental study demonstrated that a force that slightly deformed a cat tooth triggered a jaw opening reflex. Simultaneously, activity from pulpal nerves was recorded.<sup>(33)</sup> It also has been shown in clinical experiments that patients register loading earlier if a tooth is vital than if it is root filled<sup>(34)</sup>. Earlier it was demonstrated that patients with buccal or lingual cavities experienced pain when the cusp occlusally to the cavity was loaded<sup>(35)</sup>.

Further support for this has been given in studies that demonstrate that teeth can sense the consistency of objects during biting. It appears that the hydrodynamic forces that create pain when dentin is exposed are part of a system that makes it possible for us to feel the consistency of food and to avoid overloading of teeth during mastication. It can be assumed that a slight deformation during loading of a tooth would result in movement of intratubular fluid<sup>(35)</sup>. This movement is registered by intrapulpal nerve endings that send signals to regulate the bite pressure. Three types of sensory nerves are in the pulp. A-delta fibers are small and myelinated. Stimulation of A-delta fibers evokes a sensation of sharp pain. Impulses in these fibers are conducted more slowly than in touch-sensitive nerve fibers in the skin. C-fibers conduct more slowly than A-delta fibers. Activation of C-fibers results in a dull, aching, poorly localized pain. The third type is the A-beta fibers. They respond to the same type of stimuli as A-delta fibers and they appear to be more sensitive to electric stimulation<sup>(27, 36, 37)</sup>. The pulp is well equipped with sensory nerves, which supports the idea that the tooth is a sensory organ that registers load.

### EXPOSED SENSITIVE DENTIN

Exposed dentin is often sensitive, especially to cold and touch. Tooth sensitivity as a result of energetic tooth brushing, scaling, and root planning is a common complaint in clinical practice. Historically, a multitude of treatments, from crushed beetles to fluoride, have been advocated for this pain. The high number of treatment forms shows that no treatment is really successful. Also, given time the pulp produces hard tissue and the symptoms dissipate<sup>(38)</sup>. This in itself makes a high percentage of nonfunctioning methods seem efficient. Since the introduction of the hydrodynamic theory for dentin sensitivity<sup>(13)</sup>, the treatments have been aimed at preventing movement of fluid in the dentin tubules. Restorative materials, including hydrophobic bonding agents, have been used with mixed results. Dentin fluid slowly flows out of the tubules and wets the dentin surface. The introduction of hydrophilic bonding agents has improved the adhesion to the dentin surface<sup>(14)</sup>. When using a composite, or glass ionomer, to treat exposed sensitive dentin, the acid etched dentin becomes sensitive. It is advisable to use local anesthesia during the procedure. Oxalates have been used as a noninvasive way of treating sensitive dentin. Oxalate salts react with calcium ions in the dentin fluid and calcium oxalate crystals precipitate in the tubules<sup>(38)</sup>. This reduces the functional diameter of the tubules and limits the move-

ment of the fluid. The use of potassium oxalate has the additional benefit that potassium ions can reduce nerve activity. This salt has been used in clinical trials and is a reliable treatment form for sensitive, exposed dentin.

Many attempts have been made to relate symptoms from assumed inflamed pulps to the histologic image of the pulp (17). It was found that there was little correlation between clinical symptoms and histologic findings in the pulp (39). Since then numerous studies have confirmed Greth's findings. In other words, it is not possible to make a correct histopathology diagnosis based on clinical symptoms (40). The causes for pulpal pain are not fully understood. Even if we know that the pulp is inflamed, it is often free from symptoms. Also, two pulps with similar histopathology features may show different clinical symptoms. It is conceivable that an increased osmotic pressure caused by accumulated bacterial products causes an outward flow of dentin fluid, thus eliciting firing of pulpal nerve endings (41). It is not possible, however, to demonstrate this by means of histologic techniques. This may explain to some extent discrepancies between clinical and histopathology findings. It is also conceivable that differences in the micro flora of different carious lesions may affect nerve endings differently. This leaves us in a gray area when it comes to understanding the true state of a pulp. A common way of discriminating between pain to cold that emanates from exposed dentin or from pulpal inflammation is to determine the duration of the pain. Pain that lingers for a few seconds is considered as being a sign of pulpal inflammation. This is a clinical rule of thumb that probably leads us astray occasionally. Currently, however, there is no better yardstick.

A true clinical diagnosis is based on objective symptoms and determines the nature and location of a disease. A treatment decision is then made based on the diagnosis, available procedures, and experience of the clinician. In the uncertain world of pulpal diagnostics the terms reversible and irreversible pulpitis are often used instead of true diagnoses. These terms do not describe the nature of the disease. By using these terms the clinician has made a decision whether he or she believes that the pulpal inflammatory process will reverse after excavation of caries and placement of a filling. The World Health Organization advocates pulp diagnoses based on clinical findings and our knowledge of pulpal reactions. Caries is an infectious disease (25). The bacterial by-products from the carious lesion pass through the dentinal tubules and cause pulpal inflammation (35, 42, 43). So, from studies we know that a tooth with a carious lesion has an inflamed pulp. If this tooth has symptoms the diagnosis is symptomatic pulpitis. If there are no symptoms, the diagnosis is asymptomatic pulpitis. The treatment is then based on the diagnosis but also clinical factors such as depth of cavity after excavation, exposure or no exposure of pulp, and stage of tooth development.

#### *PULPAL PAIN/HYPERSENSITIVE TEETH*

Often these teeth have been called hyperemia. Because we do not know much about the actual blood flow in these teeth, however, this term is a misnomer. These teeth are sensitive to changes in temperature, especially cold, and the duration of the pain is usually a few seconds. There may be several reasons for this type of increased sensitivity such as

exposed dentin, leakage under a restoration, or an iatrogenic reason such as preparation without sufficient water coolant. Sometimes it may be difficult to find a reason. In most instances endodontic treatment is not indicated. Replacement of fillings or treatment of exposed dentin with potassium oxalate usually takes care of the problem.

#### *PULPAL PAIN/PULPAL INFLAMMATION*

As mentioned earlier this is a gray area unless the pulp is exposed. Clinical symptoms and findings, including radiographs and the clinician's knowledge, lead to a treatment decision. As a rule of thumb the duration of the pain is used as a guide for whether endodontic treatment should be initiated. When questioning a patient regarding pain duration it is not productive to use subjective expressions such as long or short. It is better to give the patient examples: „When the pain starts, does it last for seconds, minutes, hours?“ Usually it is an indication for endodontic treatment if the pain lasts beyond seconds and lingers when the stimulus is removed. The stimulus can be hot, cold, or sweet. Sensitivity to temperature changes is a sign of a vital pulp. Pain that lingers after temperature changes is a sign of a troubled pulp. Endodontic treatment should be initiated. In severe cases, a patient has a constant, intense pain. Occasionally, pain relief can be gained by cooling the tooth with ice water. It has happened more than once that a patient who occasionally takes a „swig“ from a bottle with ice water to get pain relief has been accused of drunken behavior.

Caries and old fillings are removed in a tooth with pulpal pain. When there is no pulp exposure after excavation, the clinician must make the decision whether to access the tooth. In most instances the symptoms sub-side when a temporary filling (e.g., zinc oxide-eugenol based cement) is placed. Such a tooth must be tested later for vitality before the final restoration is placed.

When excavation has exposed the pulp, a pulpectomy, including cleaning of the canal, is the optimal treatment. Time is at a premium when dealing with emergency patients who are often added to the daily schedule. If there is a time constraint the second best treatment is a pulpotomy. If there is not sufficient time to carry out a careful pulpectomy treatment, it is better not to enter the root canals during this visit. Sources of infection such as caries and leaky fillings should be removed together with the most inflamed part of the pulp. Sometimes it is tempting to perform a quick pulpectomy but it has been found that both the proper pulpectomy and pulpotomy have better chances of rendering a symptom-free tooth than the quick pulpectomy (4). After an emergency pulpotomy, many drugs such as eugenol, cresatin, and camphorated phenol have been advocated as intervisit medications (36). It has been shown, however, that a sterile cotton pellet is as effective as any medicament and the use of various drugs does not influence the pain relief. The important part of the treatment is the removal of the infected material (26). If it is difficult to obtain good retention, the temporary filling material (e.g., zinc-oxide eugenol cement) can be allowed to fill the whole access cavity without any cotton pellet.

A tooth with a manifest, painful pulpal inflammation is sometimes difficult to anesthetize. For example, when a mandibular block has been given and all signs indicate that

the anesthesia is working, the tooth in question still may be sensitive. Additional anesthesia in the form of intraligamental or intraosseous injections is helpful. If it is possible to reach the pulp during excavation, supplemental intrapulpal anesthesia can be given. Birchfield and Rosenberg (7), have demonstrated that it is a prerequisite for anesthesia to obtain back pressure during intrapulpal injection. If it is difficult to obtain back pressure directly, a temporary filling material can be used to seal the area around the injection needle.

### NONVITAL TEETH

The nonvital tooth with a necrotic pulp is free from symptoms. Bacteria easily can enter the necrotic pulp by way of caries, however. Also, exposed dentinal tubules without the protection given by a vital pulp act as path-ways for bacteria (19). The periapical inflammation is caused by infection in the root canal. Bacterial breakdown products such as endotoxins seep out through the apical foramen and create a local immune response to the infection in the root canal. The body needs space to line up protective layers of inflammatory cells, and inflammatory host factors trigger bone resorption to create this space. The more bacterial toxins, the more inflammatory cells are necessary for the defense. In other words the radiograph can be used as a guide to estimate the amount of bacteria. The more bacteria, the larger is the radiolucency (26). During most of its existence the periapical inflammation is free from symptoms. There appears to be a balance between the infection and the defending forces. It is important to keep this in mind when examining a patient in pain. A radiolucent area per se is not an origin of pain. The patient's history and clinical examination findings, including radiographs, determine the need for therapy.

An exacerbating periapical inflammation shows all the classic symptoms of inflammation: redness, pain, swelling, and lack of function. The tooth is tender to percussion and periapical palpation. It is a time-consuming procedure to treat this condition, but there is no way around it. It is necessary to remove the infection to make the tooth free from symptoms. This means cleaning the root canals. It is essential to use an antibacterial irrigation solution (e.g., sodium hypochlorite). Occasionally, there is an early flare-up of a periapical inflammation before radiographic signs are visible; however, it is usually not any problem to identify these teeth because they are painful and tender to percussion.

When a flare-up has reached a later stage of development there is an abscess formation. A mature abscess should be drained either through the tooth or through an incision. Sometimes it may be difficult to obtain complete drainage by way of the root canal. In these instances an incision helps. It is mostly possible to dry and close a canal after instrumentation and irrigation. The irrigation syringe can be used to extract exudate from the canal to aid the drying. The tooth may be left open overnight if it is not possible to obtain a dry canal. It is essential to bring the patient back the next day to clean the canal further and close it with an antibacterial dressing in place. To leave a tooth open for longer time periods makes it possible for new bacteria to establish themselves in the root canal system. The cleaning of the root canal is the most important part of the antibacterial treat-

ment. An intracanal medicament is also essential. It helps kill microorganisms that remain after canal instrumentation (16). Liquid medicaments seep out through the apical foramen into the body. They also react with periapical tissues. This leaves the canal without any antibacterial protection after some time. Calcium hydroxide mixed with a sterile solution stays in the canal and provides a long-term antibacterial effect. It is essential that the calcium hydroxide be mixed with water to have it ionized. Slurries based on oils and calcium hydroxide are not ionized and do not have any antibacterial effect. Because the use of calcium hydroxide gives a long-term antibacterial effect, it gives freedom when it comes to scheduling appointments.

Usually a periapical flare-up does not require the use of antibiotics. The cleaning of the root canal and, when appropriate, the draining of an abscess takes care of the infection. This rids the patient of the symptoms of pain. If the patient has fever or malaise or if an abscess is situated in the submandibular area, however, the use of antibiotics is required. Studies on the bacterial flora of infected root canals have given us the opportunity to choose antibiotics suited for different instances. The necrotic, infected root canal to a great extent has gram-negative anaerobic bacteria, which makes metronidazole the primary choice. Failing endodontic treatment with infection in previously instrumented and root filled canals provides an environment in which food is sparse for the bacteria. The predominant microorganisms that can survive in these canals are intestinal bacteria such as *Escherichia coli* and *S. faecalis* (44). This makes penicillin the drug of choice.

The endodontic treatment itself also can cause postoperative pain. If a patient shows signs of symptomatic apical periodontitis after root filling, time can be used as a guide for finding the cause. When the pain symptoms come soon or immediately after treatment, it is most likely a mechanical reason for the pain. Over-instrumentation, over-filling, or a reaction to the filling material is a likely cause. The patient should be given an explanation for the symptoms and analgesics. When there is a delay in the onset of postoperative symptoms the cause is most likely infection. Removal of the root filling material and renewed antibacterial treatment takes care of the problem.

Occasionally, a special form of postoperative symptom can be found in teeth with apices located close to the bone surface. The classic situation is as follows: a tooth without periapical radiolucency has been treated. After a few months the patient returns complaining about sensitivity to tapping of the buccal crown surface. Clinical examination reveals that the tooth is tender when percussed on the buccal surface. Usually it is less tender when percussed on the occlusal surface and not tender when the lingual surface is percussed. (Use a contraangle amalgam carrier, plastic instrument, or some other instrument that can reach.) A radiograph shows an intact periapical contour. There are no signs of inflammation caused by infection. The reason for this pain to tapping is that the apex is located closely to the bone surface. Endodontic treatment causes a slight periapical inflammation that often results in a minor bone resorption. This is a transient and symptom-free condition. When the apex is close to the bone surface, however, this resorption results in

thin apical bone or the periosteum placed directly on the apex. When the patient taps the buccal crown surface the root apex is moved buccally toward the periosteum, which is sensitive. The treatment of this condition is to explain the situation to the patient (a drawing tells more than a thousand words) and tell the patient to stop tapping. A few patients become „addicted tappers” and their continued tapping keeps the tooth tender. Apical surgery to shave the apex to create space between the bone surface and the root cures the condition.

### *REFERRED PAIN*

A case in which the origin of pain is in one location and the patient experiences pain in another is rather common. One tooth may be described as painful by the patient. If local provocation and testing do not trigger or increase the pain, referred pain should be suspected<sup>(45)</sup>. It can require a great deal of work to find the origin of the pain. Selective local anesthesia is many times useful. Teeth on the same side of the midline are tested. Referred pain may not always originate from another tooth, however. Certain forms of heart problems, such as angina pectoris, sometimes give pain symptoms in the mandibular molar areas. Most commonly the left side shows these symptoms, but in some persons the right side instead is involved.

### *CRACKED TEETH*

Cracked teeth often give atypical symptoms. These teeth can be difficult to locate and diagnose. All the problems and challenges surrounding these teeth have suggested the term cracked tooth syndrome. In the beginning cracks are small and hard to detect. As time goes by the crack increases, becomes stained, and is easier to detect. Dye solutions, fiber optic lights, and removal of restorative materials for inspection can help during the search for a suspected crack. It may take a long time before a crack develops into a detectable stage. In the meantime the patient often goes from dentist to dentist in search of a solution. Vital teeth with cracks often exhibit sudden sharp pain attacks. Movement in the fracture line causes movement of fluid in dentin tubules, which triggers pain. Sharp, intense pain, especially during chewing, should make us suspect a crack. Teeth with cracks that do not reach the pulp show this symptom until the crack becomes a fracture and the undermined portion becomes loose. When a crack goes into the pulp, the crack becomes an avenue for infection. In addition to the mentioned symptoms, sensitivity to changes in temperature also develops as the pulpal inflammation becomes advanced.

Cracks in root-filled teeth are often caused by intradental wedges, also known as posts. In addition to this it appears that patients place more load on root filled teeth than on vital teeth, thereby increasing the risk for cracks”<sup>(7)</sup>. Symptoms from cracked root-filled teeth are often vague or difficult to localize. During late stages, when the crack or fracture has reached the root surface, the advancing bacteria cause a periodontal inflammation. When this inflammation is established it is often possible to probe from a pocket to the apical region.

### *PHARMACOLOGIC MANAGEMENT OF PULPAL AND PERIAPICAL PAIN*

There are basically two situations that require drugs to subdue pain of endodontic origin. The first is when the patient calls and is in need of medication before the treatment can start. The second is when postoperative pain is predicted. With the exception of pain from exposed dentin, pain of dental origins is caused by inflammation. Therefore, no steroidal anti-inflammatory drugs (NSAIDs) are the first choice (?). When postoperative pain is expected, the use of long-lasting local anesthesia should be considered. This often can take care of the first postoperative period. For a detailed description of the use of pharmaceutical agents in the treatment of pain, please see the article by Dionne elsewhere in this issue.

### *AVOIDING OR REDUCING POSTOPERATIVE PAIN*

One measure to reduce postoperative pain after endodontic instrumentation is to reduce the occlusion. Recently, Rosenberg et al<sup>(46)</sup> found that occlusal reduction should not be prescribed generally but used during certain circumstances. It aids in the reduction of post-instrumentation pain when the patient has preoperative pain, vital pulp, percussion sensitivity, or the absence of periapical radiolucency. Patients who exhibit all these conditions benefit most from occlusal reduction; however, one (or more) of these conditions is enough to demonstrate a need for occlusal reduction.

The first rule for avoiding postoperative pain during endodontic therapy is to stay inside the canal. Instrumentation beyond the apical foramen can in itself cause pain because of the mechanical trauma. Also, it markedly increases the risk for forcing infected material from the canal into the periapical tissues and causing an exacerbation. The apical foramen is mostly not situated at the anatomic apex but 1 mm to 2 mm, and sometimes more, away from the tip of the root<sup>(47,48)</sup>. Therefore, instrumentation to the radiographic apex, something that previously has been recommended, must be avoided. Instrumentation to the radiographic apex means that the instrument in most instances is outside the root. This not only increases the risk for postoperative pain but it also increase

*Sažetak:*

Bol može biti razlog da potražimo pomoć stomatologa. Takođe može biti i razlog da se izbegava lečenje. Kao što lečenje zuba može da pacijenta oslobodi bola, može biti bolno samo po sebi. Ponekad lečenje zuba može da uzrokuje postoperativni bol. Mnogi ljudi povezuju bol sa lečenjem zuba. Čak i oni koji nisu lično iskusili bol, radije će se opredeliti da sami „reše svoj problem” ili da koriste nečija stvarna ili imaginarna rešenja. Lečenje živca u zubnom kanalu, ili narodski „lečenje korena” se koristi u medijima kao standard za mučni bol. Bilo koji bol je jednak ili još gori od „lečenja korena”. Začuđujuće je koliko štete može prouzrokovati jedan novinarski izraz, koliko bespotrebno besanih noći, brige i otkazanih sastanaka.

**REFERENCES**

- Napeñas JJ. Intraoral pain disorders. *Dent Clin North Am.* 2013 ; 57(3):429-47.
- Paphangkorakit J, Osborn JW. The effect of pressure on a maximum incisal bite force in man. *Arch Oral Biol* 1997; 42: 11-7.
- Sjogren U, Hagglund B, Sundqvist G, Wing K. Factors affecting the long-term results of endodontic treatment. *J Endod* 1990; 16(10): 498-504.
- Marhach JJ. Orofacial phantom pain: Theory and phenomenology. *J Am Dent Assoc* 1996; 127: 221-9.
- Ahlquist M, Franzen O, Coffey J, Pashley D. Dental pain evoked by hydrostatic pressures applied to exposed dentin in man: a test of the hydrodynamic theory of dentin sensitivity. *J Endod* 1994; 29(3): 130-4.
- Arvilt T: Studies on the infrastructure of dental tissues. II: The predentin-pulp border zone. *Odontologisk Revy* 1967; 189(2): 191-208.
- Ali R, O'Sullivan DJ, Gray GB, Vowles RW, Hooper SM. Teaching dental pain with and without underlying oral physiology: learning implications. *J Dent Educ.* 2009 Sep;73(9):1090-4.
- Pashley DH. Clinical considerations of dentin structure and function. *J Prosthet Dent* 1991; 66: 777-81.
- Birchfield J, Rosenberg PA. Role of the anesthetic solution in intrapulpal anesthesia. *J Endod* 1975; 1: 26-7.
- Bjerken E, Wennberg A, Tronstad L. *Endodontisk akutbehandling.* Tandlakartidningen 1980; 72: 314-9.
- Brannstrom M. Reducing the risk of sensitivity and pulp complications after the placement of crowns and fixed partial dentures. *Quintessence Int.* 1996; 27(10):673-8.
- Brannstrom M. The hydrodynamic theory of dental pain: Sensation in preparations, caries and the dental crack syndrome. *J Endod* 1986; 12: 453-7.
- Brannstrom M, Astrom A. The hydrodynamics of the dentin: Its possible relationship to dental pain. *Int Dent J* 1972; 22: 219-27.
- Brännström M, Johnson G, Nordenvall KJ. Transmission and control of dentinal pain: resin impregnation for the desensitization of dentin. *J Am Dent Assoc.* 1979; 99(4): 612-8.
- Byers MR, Kiseh SJ: Definition of somatic nerve ending in rat teeth by radiography of axon transported protein. *J Dent Res* 1976; 55(3): 419-25.
- Bystrom A, Claesson R, Sundqvist G. The antibacterial effect of camphorated para-nitrochlorophenol, camphorated phenol and calcium hydroxide in the treatment of infected root canals. *Endodontics and Dental Traumatology* 1985; 1: 170-5.
- Chase SW. The nerve fiber and Von Korff's fiber in the dental tubules of mammalian teeth. *J Dent Res* 1929; 9: 281-8.
- Chirnside IM. Bacterial invasion of non-vital dentin. *J Dent Res* 1961; 40:134-40.
- Edwardsson S. Bacteriological studies on deep areas of carious dentine. *Odontologisk Revy* 1974; 25(suppl 32): 1.
- Frank RM. Etude au microscope électronique de l'odontoblast et du canalicule dentinaire humain. *Arch Oral Biol* 1966; 11:179-99.
- Lilja J. Innervation of different parts of the predentin and dentin in young human premolars. *Acta Odontol Scand* 1979; 37:337-46.
- Villa-Chávez CE, Patiño-Marín N, Loyola-Rodríguez JP, Zavala-Alonso NV, Martínez-Castañón GA, Medina-Solis CE. Predictive values of thermal and electrical dental pulp tests: a clinical study. *J Endod.* 2013; 39(8):965-9.
- Kakar A, Kakar K. Measurement of dentin hypersensitivity with the Jay Sensitivity Sensor Probe and the Yeaple probe to compare relief from dentin hypersensitivity by dentifrices. *Am J Dent.* 2013; 26 Spec No B:21B-28B.
- Hargreaves K, Troullos ES, Dionne RA. Pharmacological rationale for the treatment of acute pain. *Dent Clin North Am* 1987; 31:675-94.
- Hasselgren G, Reit C. Emergency pulpotomy: Pain relieving effect with and without the use of sedative dressings. *Journal of Endodontics* 1989; 15: 254-6.
- Hildebrand C, Fried K, Tuisku F, et al: Teeth and tooth nerves. *Prog Neurobiol* 45:165-222, 1995
- Kim TS, Caruso JM, Christensen H, Torabinejad M. A comparison of cone-beam computed tomography and direct measurement in the examination of the mandibular canal and adjacent structures. *J Endod.* 2010; 36(7):1191-4.
- Langeland K. Histologic evaluation of pulp reactions to operative procedures. *Oral Surg Oral Med Oral Pathol* 1959; 12:1357-69.
- Langeland K: Management of the inflamed pulp associated with deep carious lesions. *Journal of Endodontics* 1981; 7:169-75.
- Langeland K: Prevention of pulp damage. *Dent Clin North Am* 1972; 16: 709-32.
- Langeland K. Tissue response to dental caries. *Endodontics and Dental Traumatology* 1987; 3: 249-71.
- Bánóczy J. ŠDentin hypersensitivity and its significance in dental practice. *Č. Fogorv Sz.* 2002 Dec;95(6):223-8.
- Olgart L, Gazelius B, Sundstrom F. Intradental nerve activity and jaw-opening reflex in response to mechanical deformation of cat teeth. *Acta Physiol Scand* 1988; 133: 399-406.
- Randow K, Glantz PO. On cantilever loading of vital and non-vital teeth: An experimental clinical study. *Acta Odontol Scand* 1986; 44: 271-7.
- Robinson AD. A preliminary investigation of the pain response to mechanical deformation of the teeth. *Arch Oral Biol* 1964; 281-6.
- Narhi M. The characteristics of intradental sensory units and their responses to stimulation. *J Dent Res* 1985; 64: 654-71.
- Narhi M, Jyvasjarvi E, Virtanen A: Role of intradental A- and C-type nerve fibers in dental pain mechanisms. *Proceedings of the Finnish Dental Society* 1992; 88 (suppl 1) :507-16.
- Pashley DH. Dentin permeability, dentin sensitivity and treatment through tubule occlusion. *J Endodontics* 1986; 12: 465-74.

39. Pashley DH. Dentin permeability and its role in the pathobiology of dentine sensitivity. *Arch Oral Biol* 1994; 39(suppl): 73S-80S.
40. Han G, Hu M, Zhang Y, Jiang H. Pulp vitality and histologic changes in human dental pulp after the application of moderate and severe intrusive orthodontic forces. *Am J Orthod Dentofacial Orthop*. 2013; 144(4):518-22.
41. Pawar R, Alqaied A, Safavi K, Boyko J, Kaufman B. Influence of an apical negative pressure irrigation system on bacterial elimination during endodontic therapy: a prospective randomized clinical study. *J Endod*. 2012; 38(9):1177-81.
42. Nakabayashi N. The hybrid layer: A resin-dentin composite. *Proceedings of the Finnish Dental Society* 1992; 88 (suppl 1): 321-9.
43. Reeves R, Stanley HR. The relationship of bacterial penetration and pulpal pathosis in carious teeth. *Oral Surg Oral Med Oral Pathol* 1966; 22 (1): 59-65.
44. Molander A, Reit C, Dahlen G, Kvist T. Microbiological status of root filled teeth with apical periodontitis. *International Endodontic Journal* 1998; 31:1-7.
45. Okeson JB: History and examination for temporomandibular disorders. *Mandibular Disorders and Occlusion*, ed 2. St Louis, CV Mosby, 1989, pp 256-259.
46. Rosenberg PA, Babick P, Schertzer L, Leung A. The effect of occlusal reduction of pain after endodontic instrumentation. *J Endod* 1998; 24 (7): 492-6.
47. Matthews B. Effects of enamel and dentine thickness on laser doppler blood-flow signals recorded from the underlying pulp cavity in human teeth in vitro. *Arch Oral Biol*. 2013; 58(11):1692-5.
48. Tronstad L, Bjerken E, Borglin E, Hasselgren G, Petersson K, Segerstein M, Wennberg A. Behandling av exacerberande apikal periodontit: Jamforelse mellan in Jaggmedikament. *Tandlakartidningen* 1980; 72(5):234-5.