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ETIOLOGY, PATHOGENESIS AND TREATMENT OF PULPITIS

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ANATOMICO-PHYSIOLOGICAL CHARACTERISTIC OF THE PULP

The dental pulp, 75% water and 25% organic, is a viscous connective tissue of collagen fibers and ground substance supporting the vital cellular, vascular, and nerve structures of the tooth. It is a unique connective tissue in that its vascularization is essentially channeled through one opening, the apical foramen at the root apex, and it is completely encased within relatively rigid dentinal walls. Therefore, it is without the advantage of an unlimited collateral blood supply or an expansion space for the swelling that accompanies the typical inflammatory response of tissue to injurious conditions. However, the protected and isolated position of the pulp belies the fact that it is a sensitive and resilient tissue with a great potential for healing.

The dental pulp occupies the pulp cavity in the tooth. Anatomically the pulp organ is divided into:

- the *coronal pulp* located in the pulp chamber in the crown portion of the tooth, including the pulp horns that are directed toward the incisal ridges and cusp tips;
- the *radicular pulp* located in the pulp canal(s) in the root portion of the tooth.

The radicular pulp is continuous with the periapical tissues by the connecting through the apical foramen or foramina of the root. Accessory canals may extend from the pulp canal(s) laterally through the root dentin to the periodontal tissues. The shape of each pulp conforms generally to the shape of each of the respective teeth.

The pulp is a unique, specialized organ of the human body serving four functions:

1. Formative or developmental
2. Nutritive

3. Sensory or protective
4. Defensive or reparative.

The *formative* function is the production of primary and secondary dentin by the odontoblasts as well as the protective response of reactionary or reparative dentin.

The *nutritive* function supplies nutriments and moisture to the dentin through the blood vascular supply to the odontoblasts and their process.

The *sensory* function provides sensory nerve fibers within the pulp to mediate the sensation of pain. Dentin receptors are unique because various stimuli elicit only pain as a response. The pulp usually does not differentiate between heat, touch, pressure, or chemicals. Motor fibers initiate reflexes to the muscles of the blood vessel walls for the control of circulation in the pulp.

The *defensive* function of the pulp is related primarily to its response to irritation by mechanical, thermal, chemical, or bacterial stimuli and removing detrimental substances through its blood circulation and lymphatic systems. Such irritants can cause the degeneration and death of the involved odontoblastic processes and corresponding odontoblasts and the formation by the pulp of replacement odontoblasts (from undifferentiated mesenchymal cells) that lay down irregular or reparative dentin. The deposition of reparative dentin by the replacement odontoblasts lining the pulp cavity acts as a protective barrier against caries and various other irritating factors. This is a continuous but relatively slow process; taking 100 days to form a reparative dentin layer 0.12 mm thick. In cases of severe irritation the pulp responds by an inflammatory reaction similar to any other soft tissue injury. However, the inflammation may become irreversible and can result in the death of the pulp because the confined, rigid structure of the dentin limits the inflammatory response and the ability of the pulp to recover.

If, however, the irritant is very mild, such as cutting the odontoblastic process more than 1.5 mm peripheral of the pulp at high speed with air-water coolant during cavity preparation, and although the processes and corresponding odontoblasts then die, no replacement odontoblasts are formed and thus no reparative dentin. Therefore, there is no barrier (except for the smear layer) between the dead tracts remaining and the pulp. This may explain why many teeth have pulpal problems cavity preparation and restoration. Newer dentin bonding agents are promising for sealing the cut dentinal surfaces.

Knowledge of the contour and size of the pulp cavity is essential during cavity preparation. In general, the pulp cavity is a miniature contour of the external surface of the tooth. The size varies among the various teeth in the same mouth and among individuals. With advancing age, the pulp cavity usually decreases in size. Radiographs are an invaluable aid in determining the size of the pulp cavity and an existing pathological condition. Also with advanced age, the pulp generally becomes more fibrous because of episodes of irritation and may contain pulp stones or denticles. The latter are nodular, calcified masses usually appearing in the pulp chamber but also may be in the pulp canal. These may be attached to the pulp cavity wall or free in the mass of pulp tissue. Timely root canal therapy is advised before a stone is formed since it can be a significant problem for the root canal therapist.

Morphology

The pulpal tissue is traditionally described in histologically distinct, concentring zones: the innermost peripheral pulp core, the cell-rich zone, the cell-free zone, and the peripheral odontoblastic layer.

The radicular and coronal pulp core is largely ground substance, an amorphous protein matrix gel surrounding cells,

discrete collagen fibers, and the channels of vascular and sensory supply. The gel serves as a transfer medium, between widely spaced pulp cells and vasculature, for transport of nutrients and by-products. Terminal neural and vascular components, which divide and multiply extensively in the subodontoblastic zones, converge into larger vessels and trunks and together from a main trunk passing through the pulp core to or from the apical foramina. Both matrix and collagen components are formed and maintained by a dispersed network of interconnected fibroblastic cells.

Fibrocytes and undifferentiated mesenchymal cells are particularly concentrated in the outer coronal pulp to form the cell-rich zone subjacent to the peripheral layer of odontoblastic cells. Functioning like troops in reserve, the mesenchymal cells and/or fibrocytes are capable of accelerated mitotic differentiation and collagen matrix production to serve as functional replacements for destroyed odontoblastic cells. They produce reparative dentin when bacteria or their by-products breach the permeable dentinal wall or a pulpal exposure occurs. A dense and extensive capillary bed and nerve plexus from the cell-free zone, infiltrate the cell-rich zone, and separate it from the cellular bodies of the peripheral odontoblastic layer.

Odontoblastic layer

The peripheral cellular layer of the pulp, the odontoblasts, produce primary, secondary, and reactionary dentin. This layer may also regulate or influence tubular mineralization and sclerosis as a defense mechanism. Postmitotic and irreplaceable, the columnar cell bodies line the predentin wall of the pulp chamber in a single layer. From each cell, a single process extends into at least one third of the tubule and adjacent dental substrate that it formed. Each cell has an indefinite life span, but crowding from continued

deposition of secondary dentin constricts the pulpal chamber to reduce the initial number of cells by half. The odontoblastic cells are packed closely together, with both permanent and temporary junctions between the cellular membranes. Just as the peripheral processes of the odontoblasts are physically interconnected, a third type of intercellular interface, a communicating junction, mediates transfer of chemical and electronic signals that permit coordinated response and reaction of the odontoblastic layer. Thus, as an additional protective response, the integrity and spacing of the odontoblastic layer mediates the passage of tissue fluids and molecules between the pulp and the dentin. Routine operative procedures, such as cavity preparation and air-drying of the cut dentinal surface, can temporarily disrupt the odontoblastic layer and may sometimes inflict permanent cellular damage.

Vascular system

The circulatory system supplies the oxygen and nutrients that dissolve in and diffuse through the viscous ground substance to reach the cells. In turn, the circulation removes waste products, such as carbon dioxide, by-products of inflammation, or diffusion products that may have permeated through the dentin before they accumulate to toxic levels. The equilibrium between diffusion and clearance may be threatened by use of long-acting anesthetics that contain vasoconstrictors such as epinephrine. An intraligamental injection of a canine tooth with 2% lidocaine with 1:100.000 epinephrine will cause pulpal blood flow to cease for 20 minutes or more. Fortunately, the respiratory requirements of mature pulp cells are low so that no permanent cellular damage ensues.

Inflammation, the normal tissue response to injury and the first stage of repair, is somewhat modified by the pulp chamber. A stimulus producing cellular damage initiates

neural and chemical reactions that increase capillary permeability so that proteins, plasma fluids, and leukocytes spill into the confined extracellular space, producing elevated tissue interstitial fluid pressure. Theoretically, elevated extravascular tissue pressure could collapse the thin venule walls and start a destructive cycle of restricted circulation and expanding ischemia. However, the pulpal circulation is unique because it contains numerous arteriole "U-turns", or reverse flow loops, and arteriole-venule anastomoses, or shunts, to bypass the affected capillary bed. Also, at the periphery of the affected area, where high tissue pressure is attenuated, capillary recapture and lymphatic adsorption of edematous fluids are expedited. These processes confine the area of edema and elevated tissue pressure to the immediate inflamed area. Although tissue pressure at an area of pulpal inflammation is two to three times higher than normal, it quickly falls to nearly normal levels approximately 1.0 mm from the affected area.

Another protective effect of evaluated but localized pulpal tissue pressure is a vigorous outward flow of tubular fluid to counteract the pulpal diffusion of noxious solutes through permeable dentin. However, an inflammatory condition and higher tissue pressure may also induce hyperalgesia, a lowered threshold of sensitivity of pulpal nerves. Thus, an afflicted tooth exposed to the added stress of cavity preparation and restoration may become symptomatic or hypersensitive to cold or other stimuli.

Innervation

Dental nerves are either efferent autonomic C fibers to regulate blood flow or afferent sensory nerves derived from the second and third divisions of the fifth intracranial (trigeminal) nerve. Nerves are classified according to purpose, myelin sheathing, diameter, and conduction velocity. Although

a few large and very high-conduction velocity A-β(beta) nerves with a proprioceptive function have been identified, most sensory interdental nerves are either myelinated A-δ(delta) nerves or smaller, unmyelinated C fibers. The innervation of the premolar, for example, consists of about 500 individual A-δ nerves that gradually lose their myelin coating and Shwann cell sheathing as they branch and form a sensory plexus of free nerve endings around and below the odontoblastic layer. The A-δ nerves have conduction velocities of 13.0 m/s and low sensitization thresholds to react to hydrodynamic pressure phenomena. Activation of the A-δ system results in a sharp, intense "jolt".

There three to four times more of the smaller, unmyelinated C fibers, which are more uniformly distributed through the pulp. The conduction velocities of C fibers are slower, 0.5 to 1.0 m/s, and C fibers are only activated by a level of stimuli capable of creating tissue destruction, such as prolonged high temperatures or pulpitis. The C fibers are also resistant to tissue hypoxia and are not affected by reduction of blood flow or high tissue pressure. Therefore, pain may persist in anesthetized, infected, or even nonvital teeth. The sensation resulting from activation of the C fibers is a diffuse burning or throbbing pain, and the patient may have difficulty locating the affected tooth.

The afferent transmission of painful sensations, commonly experienced although unreliable as a warning signal, may not be the primary protective function of pulpodentin innervation. Experimentally denervated teeth exposed to trauma suffer greater pulpal damage than innervated controls. The initiation and coordination of the inflammatory cascade; the vascular, tissue, and tubular fluid dynamics; and the immunocompetent response are important protective functions of the neural components.

PECULIARITIES OF PATIENT ASSESSMENT WITH PULP PATHOLOGY. PULP INFLAMMATION. ETIOLOGY AND PATHOGENESIS OF PULPITIS. PATHOMORPHOLOGY. CLASSIFICATION

Peculiarities of patient assessment with pulp pathology

The patient's complaint should be recorded in his or her own words. It is wise to listen carefully to the patient's description of his problem as it often gives the operator most of the information he needs to make a diagnosis.

Valuable information may be acquired by asking the patient specific questions about the symptoms:

- How long have you had the pain?
- When did you first notice the pain or discomfort?
- Can you point to the tooth or area that bothers you?
- Does it hurt to bite on the tooth or to the touch it?
- Describe the pain: sharp or dull, throbbing, mild or severe, localized or radiating, pulsating, nagging, sudden, off and on, constant, getting better or worsening.
- Does the tooth start hurting by itself or on its own?
- Does it hurt most during the day or at night, and how long does it last?
- What makes it hurt: hot, cold, sweets, chewing/biting, air, other?
- Does the pain linger?
- Have you taking anything to relieve the pain? If so, does it relieve the pain? For how long?

An indication of pulp vitality can be obtained through the results of thermal tests, an electric pulp test, and a test cavity. In addition, before any tooth is restored with a casting, pulpal evaluation should be performed. To conduct a *thermal test*, a cotton applicator tip sprayed with a freezing agent or hot

gutta-percha is applied directly to the tooth. Hot and cold testing should elicit from the healthy pulp a response that will subside within a few seconds following removal of the stimulus. Pain lasting 10 to 15 seconds or less after stimulation by heat or cold suggests a ***hyperemia,*** an inflammation that may be reversed by timely removal of the irritant(s). Intense pain of longer duration from hot or cold usually suggests ***irreversible pulpitis,*** which can only be treated by root canal therapy or extraction. Pain that results from heat but is quickly relieved by cold also suggests irreversible pulpitis. Lack of response to thermal tests may indicate that the pulp is necrotic. Adjacent and/or contralateral unaffected teeth should be tested for baseline comparisons as the duration of pain may differ among individuals.

The *electric pulp tester* also has value in determining the vitality of the dental pulp. The electric pulp tester is placed on the tooth and not on a restoration. A small electric current delivered to the tooth causes a tingling sensation when the pulp is vital and no response when the pulp is nonvital. It is important to obtain readings on adjacent and contralateral teeth so the tooth in question can be evaluated relative to the responses of the other teeth. Results of an electric pulp test should not be the sole basis for a pulpal diagnosis since false positives/negatives can occur. Instead, electric pulp test results provide additional information that, when combined with other findings, may lead to a diagnosis. Electric pulp testing is sometimes not possible in teeth with large or full-coverage restorations.

A *test **cavity*** can be performed to help in the evaluation of pulpal vitality when a large restoration in the tooth may be resulting in false negative responses with other evaluation methods. This test particularly is an option for diagnosing questionable pulpal vitality of a tooth contemplated for a replacement casting restoration. By using a round bur and no

anesthetic, a test cavity is made through the existing restoration into the dentin. Lack of sensitivity (response) when the dentin is cut may indicate a nonvital pulp. However, sclerosed dentin can result in a false negative. Moreover, on a multiple-rooted tooth, one region of the dentin may respond, whereas there may be no response at another site, possibly indicating a degeneration of a portion of the pulp. Furthermore, heat generated by the bur might cause a response, but the pulp may not be healthy. Though there are indications for the test cavity, its use and diagnostic information attained are limited.

Pulpal abnormalities such as *pulp stones* and *internal resorption* may be identified in the anterior *periapical radiographs.*

Causes of pulpal inflammation

Like others soft tissues, the pulp reacts to an irritant with in inflammatory response. It was previously believed that pulpal inflammation was the result of toxic effects of dental materials. More recent evidence, however, demonstrates that pulpal inflammatory reactions to dental materials are mild and transitory; significant adverse pulpal responses occur more as the result of pulpal invasion by bacteria or their toxins. Even early enamel caries lesions that extend less than one fourth of the way to the dentinoenamel junction have been shown to induce a pulpal reaction, particularly when the caries lesion has advanced rapidly. This is probably due to increase in the permeability of enamel, allowing the transmission of stimuli along enamel rods.

As a lesion progresses deeper into the tooth, pulpal reaction increases. When actual pulpal encroachment by bacteria and/or their toxins occurs, severe inflammation or pulpal necrosis frequently occurs. The outward flow of fluid through dentinal tubules does not prevent bacteria or their

toxins from reaching the pulp and initiating pulpal inflammation. The caries process also induces the formation of reparative dentin and reactive dentin sclerosis, which increases the protective effects of the remaining dentin.

When bacterial contamination is prevented, favorable responses in pulpal tissue adjacent to many restorative materials have been found. Those materials include amalgam, light-activated resin composite, autocured resin composite, zinc phosphate cement, silicate cement, glass-ionomer cement, and acrylic resin. Acid etching of dentin has long been considered detrimental to the pulp, but the pulp can readily tolerate the effects of low pH if bacterial invasion is prevented.

A number of instrumentation techniques elicit pulpal responses as well. The most common are rotary instruments used in high- and low-speed handpieces for tooth preparation. Tooth preparation can be traumatic to the pulp, and a number of factors affect pulpal reaction. The degree of pulpal reaction is depended on the amount of friction and desiccation. The key to controlling both is water spray at the site of contact between the bur and tooth structure. This is more important than the amount of water that is used on a rotating bur. Frictional heat generated by tooth preparation can result in bur lesions in the pulp and abscess formation. While it is often advantageous to refine aspects of a cavity preparation without water spray to aid visibility, this must be done conservatively. The pulp can tolerate dry preparation in a limited area, but the severity of the pulpal reactions increases as the area of dentin subjected to preparation without water spray increases. Another adverse consequence of desiccating dentin in a preparation is that the dentinal fluid is lost from the tubules. The lost fluid may be replaced with chemicals that can elicit a harmful pulpal reaction.

The temperature rise is considerably greater when enamel or a combination of enamel and dentin is prepared

versus preparation of dentin alone. Additionally, research has shown that pressure applied during rotary instrumentation has a greater effect on temperature rise than does rotational speed, which is probably why preparation using low-speed rotary instrumentation has been shown to be more traumatic to the pulp than high-speed preparation. Diamonds tend to produce more temperature increase than do carbide burs, and the reaction of the pulp tends to increase as the depth of the cavity preparation increases. Considering these latter two findings, it should not be surprising that an occasional consequence of full-coverage restorations in pulpal necrosis. Key to controlling temperature rise and minimizing adverse pulpal reaction from rotary instrumentation are adequate air/water spray coolant and light pressure during preparation. Two new methods for tooth preparation are available – lasers and kinetic cavity preparation, also known as air abrasion. Animal studies have shown that air-abrasion cavity preparation is no more traumatic to the pulp than rotary instrumentation. Likewise, the use of a variety of lasers, including CO_2, Er: YAG, and free electron lasers (FEL) on tooth structure has demonstrated minimal pulpal response, comparable to that of high-speed rotary instrumentation.

Etiological factors in pulpitis origin.

1. Infection.
2. Thermal irritants.
3. Mechanical trauma.
4. Chemical factors.
5. Concrements.

Causes of pulpal pain

The causes of pulpal pain and sensitivity, while not fully explained, are becoming better understood. Increased intrapulpal pressure on nerve endings, secondary to an

inflammatory response, is one mechanism that may explain pain as a result of bacterial invasion. However, this interpretation fails to explain sensitivity that occurs in the absence of inflammation. The explanation for pulpal pain in the absence of inflammation that is most accepted is the hydrodynamic theory. In a vital tooth with exposed dentin, there is a constant slow movement of fluid outward through the dentinal tubules. The hydrodynamic theory proposes that when a stimulus causes the slow fluid movement to become more rapid, nerve endings in the pulp are deformed, a response that is interpreted as pain. Stimuli such as tooth preparation, air drying, and application of cold have been suggested as causes of this sudden, rapid movement of fluid.

Classification of pulp diseases (K 04)

K 04.0 - Pulpitis: acute, chronic (hyperplastic, ulcerous, and purulent).

Pulpal abscess.

Pulpal polyp.

K 04.1 - Necrosis of pulp: pulpal gangrene.

K 04.2 – Regeneration/degeneratioin of the pulp: pulp stones (denticles), pulp calcification.

K 04.3 – Abnormal creation of hard tissues in the pulp.

Classification of pulpitis (K 04.0)

According to E. Platonov:

Acute: local, diffuse (generalized).

Chronic: fibrous, gangrenous (necrotic), hypertrophic (proliferative).

According to O. Javors'ka:

Acute pulp inflammation:

- Hyperemia of the pulp

- Acute local pulpitis
- Acute diffuse pulpitis
- Acute purulent pulpitis
- Acute traumatic pulpitis: accidentally naked pulp, accidentally wounded pulp, and naked pulp in cause of fracture of tooth crown.

Chronic pulp inflammation:

- Simple chronic pulpitis
- Chronic hypertrophic pulpitis
- Chronic gangrenous pulpitis
- Concremental pulpitis.

Pulpitis, complicated with focalize pericementitis.

ACUTE PULPITIS. CLINIC, DIAGNOSIS, DIFFERENTIAL DIAGNOSIS

Acute local pulpitis - (pulpitis acuta focalis)

Complaints: attacks of acute, spontaneous colicky pain, which intensificated after all types of irritants and in the nighttime. Pain lasts for 10-30 min. with long light periods (without pain). Patient usually can localize painful tooth.

Objective changes: in the tooth – deep carious cavity filled with soft, pigmented necrotic dentin and food debris. Pulp chamber is closed.

Exploration of the bottom of the cavity is painful in one or two points (near the pulp horns).

Percussion is not painful.

Thermal test is positive.

EPT – 20-25 mcA (indicates to the decreased pulp reactivity).

Rtg – no changes.

Pathological anatomy: inflammatory edema (tumor) and hyperemia of the pulp tissue.

Differential diagnosis.

- *Deep caries* – short-lived pain after irritants which passing after their removing. There is no acute, spontaneous, colicky pain, nighttime pain.
- *Acute generalized pulpitis* – attacks of colicky or continuous throbbing pain with irradiation, which last 1-3 hours; light periods are very short or absent.
- *Chronic fibrous pulpitis* - there is no spontaneous and nighttime pain. In anamnesis – acute pain in the past. Pulp chamber is perforated, exploration of this place caused acute pain and bleeding of the pulp.
- *Papillitis* – inflammation of gingival (interdental) papilla. There is inflammatory blush (hyperemia) and edema (tumor) of interdental papilla, painful and bleed to touch. Tooth is intact or with carious lesion, but there is no inflammation in pulp.

After 1-3 days duration, local pulpitis is transferred into the generalized pulpitis.

Acute generalized (diffuse) pulpitis - (pulpitis acuta diffusa)

Complaints: acute attacks of spontaneous colicky or throbbing pain, often more intensive in the nighttime, with irradiation, which last 1-3 hours without light periods. Patient usually cannot localize the painful tooth.

After 24 hours duration, serous exudate of the inflammatory pulp is transformed into the purulent exudate. In stage of purulent exudation, when the abscess is formed in the pulp tissue, reaction to the thermal irritants is changed. In stage of serous exudation could irritants be caused by the pain, in stage of purulent exudation could irritants be provoked by the pain and hot irritants provoked by the pain.

Objective changes: deep carious cavity filled with soft, pigmented necrotic dentin and food debris. Pulp chamber is closed.

Exploration of the all bottom of the cavity is very painful.

Percussion is sometimes a little painful.

Thermal test is positive (provoked very long attack of pain).

EPT – 40-50 mcA.

Rtg – no changes.

Pathological anatomy: inflammatory edema and hyperemia of the pulp tissue; enlargement of blood vessels, emigration of leucocytes, small hemorrhages, diapedesis of erythrocytes and small abscesses.

Differential diagnosis.

- *Acute local pulpitis* - pain last for 10-30 min. with long light periods (without pain), without irradiation. Patient usually can localize painful tooth. Duration of inflammatory process is 1-2 days.
- *Acute pericementitis* - localized gnawing dull pain, violent pain during and after biting; percussion is very painful; thermal test is negative; EPT>100 mcA; palpation of gingiva in proection of tooth root apex is painful.
- *Neuralgia of trifacial (trigeminal) nerve* – violent colicky pain during conversation, meal or after touching the skin of the face. There are no night attacks of pain.
- *Maxillary sinusitis* – there are several common manifestations: headache, rise of temperature, weakness. Difficult (rough) breathing through the nose, rhinorrhea.
- *Alveolitis* – inflammation of alveole after tooth extraction when blood clot was decomposed or not created at all. Objective changes: alveole is empty,

there are many gray coating on alveole walls, and palpation of gums in this region is very painful.

CHRONIC PULPITIS, EXACERBATION OF CHRONIC PULPITIS. CLINIC, DIAGNOSIS, DIFFERENTIAL DIAGNOSIS

Transformation of acute inflammation into the chronic inflammation takes place after perforation of pulp chamber is arising.

Chronic fibrous pulpitis – (pulpitis chronica fibrosa)

Complaints: often is asymptomatic; sometimes – throbbing pain and discomfort in the tooth, pain after very strong mechanical and thermal irritants. Some times pain in the tooth occurs after sharp changes of temperature (from cold to hot temperature), and after suck out from the tooth.

Objective changes: deep carious cavity with soft and dark-brown pigmented walls and bottom. Pulp chamber is perforated, exploration in this place caused acute pain and bleeding of the pulp. Some times, when the pulp chamber is closed, exploration of the bottom of the cavity is only sensitive.

Percussion is not painful.

Thermal test is positive.

EPT – 25-50 mcA

Rtg – no changes.

Pathological anatomy: vascular and exudative reactions are not expressive. Productive processes are dominated. Transformation of pulp tissue into the fibrous connective tissue, gialinosis and sclerosis of pulp, decrease of pulp cells takes place. Blood vessels are widening.

Differential diagnosis.

- *Profound caries* – short-lived pain after all types of irritants which passing after their removing, there is no acute pain in anamnesis.
- *Acute generalized pulpitis* - attacks of acute, spontaneous colicky pain, which intensificated after all types of irritants and in the nighttime. Pain lasts for 10-30 min. with long light periods, without irradiation. Duration of this type of pulpitis is 2-3 days.
- *Chronic necrotic pulpitis* – attacks of pain after hot irritant, pulp chamber is open, exploration of the pulp is painless (deep exploration of the pulp in the root canals is painful).

Chronic proliferative(hypertrophic) pulpitis – (pulpitis chronica hypertrofica)

This type of pulpitis is developed after chronic fibrous pulpitis, when the perforation of pulp chamber is very big and mechanical irritation of pulp taken place. It causes proliferation of pulp tissue.

Complaints: pain and bleeding occur during mastication and after mechanical irritants.

Objective changes: big carious cavity, pulp chamber is open, and hypertrophic pulp tissue is coming out from the carious cavity.

Exploration is sensitive on the surface of the pulp, and painful on the deeper layers. Hypertrophic pulp bleeds during probing.

Percussion is not painful.

Thermal test is negative.

EPT – 40-60 mcA.

Rtg – no changes.

Pathological anatomy **–** hyperplastic processes and considerable growing of connective tissue are dominated.

There are many young granulation tissue in the pulp. Odontoblasts are only in the root part of pulp.

Differential diagnosis.

- *Hypertrophic papilitis* – proliferation of inflamed interdental papilla.
- *Granulation tissue,* coming from the perforation opening in bi- or trifurcation region (X-ray with diagnostic needle is recommended).

Chronic necrotic pulpitis – (pulpitis chronica gangrenosa)

Complaints: throbbing pain after hot irritants, evil smelling, and change of tooth color. There are no spontaneous attacks of pain.

Objective changes: deep carious cavity with thin walls, pulp chamber is open. Necrotic pulp is dark colored. Tooth is changed in color (gray colored).

Exploration of the coronal pulp is unpainful, deep exploration is painful.

Percussion is not painful.

Thermal test is positive (hot water).

EPT – 60-80 mcA.

Rtg – some times show deformation and widening of periodontal slit.

Pathological anatomy: structure of coronal pulp is lost. There are no cells and fibers. There are many colonies of microorganisms, crystals of fat acids, bloody pigments in necrotic pulp tissue.

Differential diagnosis.

- *Chronic fibrous pulpitis* - often is asymptomatic, some times, pain occurs after very strong mechanical or thermal (as a usual, cold) irritants; there is no pain after

hot irritants. Pulp chamber is perforated, exploration in this place caused acute pain and bleeding of the pulp.

- *Chronic apical pericementitis* – acute pericementitis in anamnesis. Superficial and deep exploration is not painful; thermal test is negative; EPT>100 mcA; percussion is sensitive or painful. Some times, on the alveolar gums in projection of root apex can be gingival fistula.

Exacerbation of chronic pulpitis (pulpitis chronica exacerbata)

As a usual chronic fibrous pulpitis is exacerbated. Causes of exacerbation: common diseases, trauma of pulp through the carious cavity, mistakes during previous tooth filling.

Subjective changes: all signs of acute pulpitis, with anamnesis of previous attacks of acute pain with/without irradiation. Some times pain is dull, gnawing and constant (continuous).

Objective changes: deep carious cavity, pulp chamber is open; pulp during exploration is painful; thermal test is positive. Some times there is no pain after irritants.

Differential diagnosis.

- *Acute pulpitis*
- *Acute apical pericementitis*
- *Exacerbation of chronic apical pericementitis*

ANOTHER TYPES OF PULPITIS

Traumatic pulpitis:

- Accidentally naked pulp
- Accidentally wounded pulp
- Naked pulp in cause of fracture of tooth crown

Accidentally naked pulp – occur during cavity preparation, as result of mistakes in operative technique when carious lesion is very deep. As a usual, patient doesn't feel the pain. There are no pathomorphological changes in pulp tissue. On this situation operator must continue his work in sterile conditions. Indirect pulp capping is recommended.

Accidentally wounded pulp – trauma of the pulp by the explorer, excavator or drill during preparation of carious cavity. On this situation pulp always is infected by necrotic dentin. The first sign of pulp wound is a very acute short-lived pain. On the bottom of the cavity – perforation into the pulp chamber and drop of blood. On this situation operator can use a direct pulp capping. Direct pulp capping should be attempted only a small mechanical exposure of an otherwise healthy pulp occurs. The tooth must be isolated with a rubber dam, and adequate hemostasis must be achieved. The exposure should be covered with calcium hydroxide because of its documented ability to provide the highest percentage of success. It must be possible to restore the tooth with a well-sealed restoration that will prevent subsequent bacterial contamination.

Naked pulp in cause of fracture of tooth crown.

Naked pulp tissue during short period of time turns to be infectious. Patient feels the pain after all irritants. Pathologico-anatomical changes are analogous to acute inflammation of pulp.

Restpulpitis –inflammation of root canal pulp tissue in causes of mistakes in endodontic treatment of pulpitis. Clinical changes are like chronic pulpitis.

Concremental pulpitis.

This type of pulpitis is origin as result of reparative dentin formation in the pulp tissue (denticles (pulp stones), petrification of the pulp). More often occurs in patients over 30 years old (with advanced age, the pulp generally becomes more fibrous because of episodes of irritation).

Subjective changes: unbearable attacks of pain with irradiation; occurs without any visual irritants or, some times, after rapid had movements.

Objective changes: as a usually tooth is intact with effaced occlusal surface, or, sometimes, after caries treatment.

Rtg: there are one or many denticles in tooth cavity or root canals. The latter are nodular, calcified masses usually appearing in the pulp chamber but also may be in the pulp canal. These may be attached to the pulp cavity wall or free in the mass of pulp tissue.

Differential diagnosis is made between concremental pulpitis and acute diffuse pulpitis, and neuralgia of trigeminal nerve (X-ray diagnosis is necessary).

Timely root canal therapy is advised before a stone is formed since it can be a significant problem for the root canal therapist.

Pulpitis, complicated with localize pericementitis (pulpitis, periodontiti focali complicata)

Complaints: spontaneous throbbing pain with irradiation, pain increased after all types of irritants, during mastication and at the nighttime. Light periods is very short or absent at all.

Objective changes: deep carious cavity, pulp chamber is close. After perforation of pulp cavity, pulp is painful and bleeds during probing. Alveolar gums under the causative tooth are tumorous, inflammatory blushed, and painful to touch.

Percussion is very painful.

Thermal test is positive.

EPT – >100 mcA

Rtg - no changes in periapical tissues (when pericementitis is chronic - deformation and widening of periodontal slit, resorption or destruction of alveolar bone tissue).

METHODS OF TREATMENT OF PULPITIS. ANESTHESIA IN PULPITIS TREATMENT. INDICATIONS FOR USE AND CHOOSING OF TREATMENT METHOD. CONSERVATIVE TREATMENT OF PULPITIS

Methods of pulpitis treatment

A. Methods of pulp preservation (biological)
   1. Method of full pulp preservation (conservative)
   2. Method of partial pulp preservation (vital pulp amputation, pulpotomy)

B. Methods of pulp removal
   1. Vital extirpation (with anesthesia)
   2. Non vital extirpation (with devitalizing agents)
   3. Combinative method

Choosing of treatment method

Before choosing of treatment method we must take into consideration the type of pulpitis and some conditions, like a:

- Patient age and common condition
- Prolongation of disease
- Ways of infection entrance into the pulp
- Localization of the carious cavity
- Condition of circumpulpal dentin
- Color, consistence and sensitivity of pulp
- Index of EPT
- Condition of periapical tissues
- Rtg information

Anesthesia of inflammatory pulp

1. Extrapulpal methods:
   - General anesthesia (narcosis, analgesia)

- Local anesthesia (injectional: infiltration (subperiosteal, periapical, intraligamentous, intrapapillar, intraosteal), conduction)

2. Intrapulpal methods:
- Application
- Droock-anesthesia (anesthesia under the pressure)
- Intrapulpal injectional anesthesia

Method of full pulp preservation (conservative)

Indications:
- Reversible pulpitis (hyperemia of the pulp)
- Traumatic pulpitis (accidentally naked pulp)

Conditions, when we can use this method:
- Young age (under 30 years old)
- Prolongation of disease is no longer than 2 days
- Entrance of the infection was through the carious cavity
- Carious cavity is localized in limits of anatomic tooth crown
- Presence of connected with pulp dentin on the bottom of the cavity
- No changes in periapical tissues

Reversible pulpitis ⇒***Indirect pulp capping.***

This procedure involves the removal of infected dentin except for the deepest, last small amount, which if removed might expose the pulp. Subsequent placement of restorative materials must adequately seal the cavity and provide thermal, mechanical, and chemical protection. If the pulp is healthy, secondary odontoblasts will differentiate and form a layer of

reparative dentin for further protection. The decision on whether to re-enter the cavity at a later time (at least 6 months) is based on how much infected dentin was left behind during the indirect pulp capping procedure. This decision must consider the possibility for further injury to the pulp from additional operative procedures.

The goals of this procedure are to prevent pulp exposure and aid pulpal recovery by medication. The portion of the remaining softened dentin is covered with calcium hydroxide liner/base and the excavated area is restored with a temporary material. Calcium hydroxide promotes reparative dentin bridges over any area of frank pulpal exposure. Such repair usually occurs in 6 to 8 weeks and may be evident radiographically in 10 to 12 weeks.

Traumatic pulpitis⇒***Direct pulp capping***.

A direct pulp cap is a technique for treating a pulp exposure with calcium hydroxide to stimulate dentin bridge (reparative dentin) formation. If the exposure site is the consequence of infected dentin extending into the pulp, termed a carious pulpal exposure, it is likely that infection of the pulp has already occurred and removal of the tooth pulp is indicated. If, however, the pulp exposure occurs in area of normal dentin (usually as a result of operator error or misjudgment), termed a mechanical pulpal exposure, and bacterial contamination from salivary exposure does not occur, the potential success of the direct pulp cap procedure is enhanced. With either type of exposure, a more favorable prognosis for the pulp following direct pulp capping may be expected if:

- The tooth has been asymptomatic (no spontaneous pain, normal response to thermal testing, and is vital) prior to the operative procedure.
- The exposure is small, less than 0.5 mm in diameter.

- The hemorrhage from the exposure site is easily controlled.
- The exposure occurred in clean, uncontaminated field (such as provided by rubber dam isolation).
- The exposure was relatively atraumatic and little desiccation of the tooth occurred, with no evidence of aspiration of blood into the dentin (dentin blushing).

Animal studies have demonstrated that direct pulpal exposures can heal normally, but a bacteria-free environment is required. The adverse consequences of bacterial contamination of the pulp have been well documented. Therefore, the only reasonable chance that a direct pulp cap has to permit formation of a dentin bridge and to maintain pulp vitality is under the most ideal conditions. If a large number of bacteria from a caries lesion or exposure to the oral flora have contaminated the pulp, the likelihood of regaining or maintaining a healthy pulp is slight. In addition, aged pulps have increased fibrosis and a decreased blood supply, and thus a decreased ability to mount an effective response to invading microorganisms.

In one clinical study of direct pulp capping of 38 patients over 3 years, no relationship between success and factors such as patient age, tooth type, or size of exposure was found. However, in a larger study of both direct and indirect pulp capping involving 592 patients over a 24-year period, age, tooth type, and extent of exposure did have a bearing on success.

The degree of bleeding affects the success of direct pulp capping; increased bleeding is associated with increased likelihood of failure.

Direct pulp capping should be attempted only when a small mechanical exposure of an otherwise healthy pulp occurs. The tooth must be isolated with a rubber dam, and

adequate hemostasis must be achieved. The exposure should be covered with calcium hydroxide because of its documented ability to provide the highest percentage of success. It must be possible to restore the tooth with a well-sealed restoration that will prevent subsequent bacterial contamination.

METHOD OF PARTIAL PULP PRESERVATION (VITAL PULP AMPUTATION) IN PULPITIS TREATMENT. INDICATIONS. TECNIQUE STAGES, MEDICATIONS

Method of partial pulp preservation (vital pulp amputation)

The main point of the method – operative removing of part of inflammation (coronal pulp) and medicamental treatment of root pulp.

Indications:

- Traumatic pulpitis (accidentally wounded pulp)
- Acute local pulpitis
- When direct pulp capping method was not effective

Conditions:

- Young age
- Carious cavity is localized on tooth crown
- There is no changes in periapical tissues

Technique stages:

1. Operative field isolation
2. Anesthesia
3. Carious cavity preparation
4. Opening of pulp chamber
5. Pulp amputation
6. Widening of root canals mouth

7. Hemostasis (diatermocoagulation, haemostatics: sol. of adrenalinum, hemophobinum, absorbable gelatin sponge, hemocollagene, hemofibrine, alstase…)
8. Root pulp capping (calcium hydroxide liners: calcipulpe, pulpomixine)
9. Temporary filling
10. Permanent filling (after 3-4 weeks)

EXTIRPATION METHODS IN PULPITIS TREATMENT. INDICATIONS. TECNIQUE STAGES, MEDICATIONS

Method of vital pulp extirpation

Indications: traumatic pulpitis (fracture of tooth crown), acute generalized pulpitis, chronic fibrous, prolipherative, necrotic pulpitis, concremental pulpitis, exacerbation of chronic pulpitis, pulpitis complicated with apical pericementitis.

Technique stages:

I visitation:
1. Operative field isolation (rubber dam)
2. Anesthesia
3. Carious cavity preparation
4. Opening of pulp chamber
5. Pulp amputation (excavator, round carbide drill)
6. Widening of root canals orifice (gates-glidden drill, peeso reamer, orifice opener (widener))
7. Pulp extirpation (barbed (nerve) broash)
8. Hemostasis (diatermocoagulation, hemostatics: sol. of adrenalinum, H_2O_2, hemophobinum, Alustin…)

9. Widening of root canals (step-back, crown-down techniques; chemical widening: Canal+, Largal Ultra, Parcan)
10. Cleaning of the root canals with antiseptics (3% sol. of H_2O_2, sol.NaOCl, 0.2% sol. of Chlorhexidini bigluconatis, 0.5% sol. of Furacilini, Antiformin, Natrium chloratum…)
11. Hermetization of root canals with cotton with antiseptic (Dicamphen, Cresophene, Camphocresol, Camphenol, Falicid, Endotine, Grinazole…)
12. Temporary filling

II visitation (after 1-2 days):

1. Removing of the temporary filling
2. Cleaning of root canals with antiseptics
3. Drying of root canals (septopoints(paper), Sikko Tim, Hydrol)
4. Filling of root canals (*Sealers*: Endomethasone, Endobtur, TubliSeal, Wach's Cement; SealApex, Apexit, CRCS (Calcibiotic Root Canal Sealer); Diaket, ThermaSeal, TopSeal, Lee Endo-Fill; Ketac-Endo. *Fillers*: Gutta-percha points, silver points)
5. Permanent filling of cavity

One-visit method in treatment of pulpitis

Indications: chronic fibrous pulpitis, chronic proliferative pulpitis, traumatic pulpitis, acute local pulpitis; orthodontic and prosthetic indications.

Conditions: one-root tooth, no periapical inflammation.

Technique stages:

1. Operative field isolation
2. Anesthesia
3. Carious cavity preparation
4. Opening of pulp chamber

5. Pulp amputation
6. Widening of root canals orifice
7. Pulp extirpation
8. Hemostasis
9. Widening of root canals
10. Cleaning of the root canals with antiseptics
11. Drying of root canals
12. Filling of root canals
13. Permanent filling of cavity.

Method of non-vital pulp extirpation

Technique stages:

I visitation

1. Operative field isolation
2. Partial preparation of carious cavity
3. Anesthesia (intrapulpal methods (Anesthopulpe, Pulperyl, Pulpomixine…))
4. Opening of pulp chamber
5. Application of devitalizing paste (Caustinerf arsenical – 7days, Caustinerf rapid – 3days, Caustinerf Fort sans arsenic – 7-10days, Periodontique sans arsenic – 7days, Paraformaldegid – 7-10days; Depulpin…)
6. Temporary filling

II visitation

1. Removing of temporary filling
2. Radical preparation of carious cavity
3. Opening of pulp chamber
4. Pulp amputation
5. Widening of root canals orifice
6. Pulp extirpation

7. Widening of root canals
8. Cleaning of the root canals with antiseptics
9. Drying of root canals
10. Filling of root canals
11. Permanent filling of cavity.

MISTAKES AND COMPLICATIONS IN PULPITIS DIAGNOSIS AND TREATMENT; THEIR CORRECTION

1. Common: during anaesthesia, inspiration of endodontic instrument.
2. Local: in diagnosis, during treatment, after treatment.

Fractured instruments.

One of the most time-consuming and frustrating side effects of endodontic treatment is the fracture of an endodontic instrument within a root canal. Prevention of such an accident is preferable to the effort, frequently abortive, expended in the retrieval of fractured portions.

Causes of fracture:

1. Over-use of the instrument.
2. Sterilization at temperature above 170°C (excepting the use of a glass bead sterilizer, in which immersion of the instrument is solely for a period of 10-15 seconds).
3. Mishandling the instrument in the canal.
4. Faulty access cavity.
5. Faults in manufacture.

1. Over-use. The small diameter instruments are most at risk and should be used once or twice only and then discarded. As a rough guide, sizes 8-25 should be used once or twice, sizes 30-50 may be used twice and the larger sizes should be

inspected after use for signs of bluntness or distortion, and rejected if either factor is apparent. Flaming of endodontic instruments is to be condemned out of hand, because it leaves them brittle and apt to fracture.

2. Sterilisation. When instruments are sterilized by dry heat, a temperature of 160˚C for one hour is acceptable from a metallurgical viewpoint. After numerous repeated sterilizations, there is a tendency for the metal of some makes the instrument to become slightly brown in colour. This does not materially affect the properties of the large sizes, but an increased brittleness in the fine diameter reamers and files negates their further usefulness. Ideally, instruments should be stored sterile in glass phials, so that those not used will not suffer repeated thermal cycling.

3. Mishandling. This constitutes the major cause of fracture. No endodontic instrument should be forced into a narrow canal. Barbed broaches and spiral fillers are especially prone to fracture and engine driven reamers invariably fracture, if they jam in a fine canal. No barbed broach or spiral fillers should be used unless it can be inserted freely to the end of the canal.

Reamers and files, especially the finest sizes, should never be rotated more than one quarter turn in the first application, before being withdrawn, cleaned and reinserted. When the apical constriction has been reached, and it becomes possible to rotate the instrument without binding, the canal is irrigated and the next size of instrument is inserted. Filing is continued using a pulling action, whereas reamers operate only when rotated in a clockwise direction.

N.B! Never misses out the next larger size. Any attempt to rotate a reamer in a curved canal can lead to fracture of the reamer or perforation of the root.

If an instrument is jammed, great care must be taken to avoid reverse rotation because the distortion of the flutes leads

quickly to fracture. The canal should be irrigated and then, using a pulling action coupled with a gentle, quarter turn, reciprocatory movement, disengagement can usually be achieved. Clockwise rotation of a jammed reamer or K type file tends to unwind the flutes, resulting ultimately in fracture. Such stresses are set up also in Hedstroem and similar files.

After each application of an endodontic instrument, it is mandatory that the flutes or blades of the instrument be checked for signs of damage. If any damage is found, immediate rejection is necessary.

Exerting excessive force when using fine instruments may lead to bending or kinking. Unless this is minimal, they should not be straightened, but replaced. Blunt endodontic instruments are useless. Their continued use is false economy and the tendency to make them work by the application of greater force may lead to fracture.

For endodontic preparation, a reciprocating handpiece has become increasingly popular. However, it should be used at speeds in the region of 1-2.000 rpm and with minimum force, otherwise fracture of instruments can occur. Peeso reamers and Gates Glidden drills similar require a gentle touch. Their use for the total preparation of canals is to be condemned. They must fit the canal loosely before the engine is operated and be used for flaring the canals with a gentle circumferential pulling movement. They can also be used for removing gutta-percha root fillings.

4. *Faulty access cavity.* It is a sine qua non that the access cavity must be prepared in a direct line with the root canal. In a multi-rooted tooth, extension of the cavity for each canal alignment is mandatory. In lower incisors, this will often result in involvement of part of the incisal margin lingually or even labially. Failure to observe this rule may lead to bending of the instrument with consequent risk of perforation of the

root or fracture of an instrument, the flutes of which have undergone deformation due to jamming.

5. *Faults in manufacture.* Although rare, it is possible to have a flaw in a file or reamer, which can lead to its fracture. It is difficult to assess whether such is the case when an instrument fractures in use. However, misuse is more likely to stress the defect, resulting in fracture.

It is salutary to note that the more experienced the operator, the lower the incidence of fractured files and reamers. Some endodontic instruments may vary in length by ±1.0mm: this should be checked before root canal stops are adjusted.

Retrieval of fractured instruments. When an instrument has been fractured near to the apex of the root, the ability to remove it is related to how closely it fits the canal and how tightly it has been forced against the walls, especially when the canal is curved.

When the fractured part extends coronally into a flared portion of the canal, its removal is made easier. If it reaches the pulp chamber it may be possible to grasp it with Steiglitz forceps or tease it out with a fine sharp excavator or by wedging a Hedstreom file against it and pulling. If this is ineffective, a sharp tap on the beaks of pliers, grasping the handle of the file, may dislodge the fragment.

Frequently it is possible to insinuate a fine file past the fragment and reach the apical constriction. Then, by enlarging the spase with ascending sizes of file, the canal may be prepared and filled, incorporating the fragment into the root filling.

The fracture of barbed broaches. The barbed broach was devised for extirpation of a vital pulp and its sole alternative function is the removal of entrapped fragments of cotton wool or paper points from a root canal. The most likely reason for fracture of a barbed broach is attempting to remove

it from the canal when it has become lodged, and this occurs because it has been used in a canal which is the same size or smaller than the broach.

As a precautionary measure, barbed broaches should be used only in canals in which they do not bind against the walls. Otherwise, it is safer to eliminate pulpal tissue with files.

Removal of a fragment of fractured barbed broach may be extremely difficult but it is best accomplished by attempting to bypass the fragment with fine files.

Alternatively, by widening the canal as far as the obstruction, so that a trephine can be inserted, it may be possible to cut away the dentine surrounding the fragment and thus release it.

The Masseran technique. The mechanical removal of a fragment can be accomplished with the aid of a Masseran kit. A channel is cut around the fragment, extending approximately half its lenghth, so that it may be grasped and removed. A fair amount of dentine is lost in the process and the instruments must be confined to the straight part of the canal. The Masseran kit is especially useful for the removal of fractured posts, instruments and metal points which are lodged within the coronal part of the canal.

There are 14 different diameters of trepan, varying from 1.1mm to 2.4mm, color-coded for easy identification. There are two trepans of each diameter, one short and one long. The wall of the trepan is less than0.25mm thick. Either the trepans may be screwed into the milled handle or mounted in a speed-reducting handpiece. The author recommends the use of the handle because greater control can be exercised than with a handpiece.

The smallest trepan that will fit around the fragment is selected by using one of the gauges provided. The trepan is pressed over the end of the fragment and turned in an anti-

clockwise direction. Irrigation fluid or some other lubricant will facilitate this procedure. The trepan should be withdrawn from the canal after a few turns and the accumulated debris removed. Trepan burs are thin and easily damaged. However, they can be sharpened by the operator, following the instruction provided.

Removal of the fragment. The method of removal depends upon the site of the fragment. In the case of the larger instruments, points and posts, a trepan one size smaller than that used to cut the channel is selected. The end of the trepan has to grip on to the metal until it is sufficiently secured to allow its removal by means of a twisting action combined with apically directed pressure.

If the fragment is sufficiently small an extractor may be used. This consists of a rod, which is screwed into a tube. Close to the end of the tube, internally, is a ridge against which the rod engages. The extractor is pushed over the partially freed fragment and the rod is screwed home, thus gripping the end of the fragment against the ridge and permitting its removal.

Failure to remove or bypass the fragment, coupled with subsequent evidence of periapical pathosis will indicate the adoption of a surgical approach. Either extraction of the tooth, apicectomy or root resection (amputation in a multi-rooted tooth) is indicated.

Perforations.

There are three types of perforations according to their position in the tooth.

Lateral wall of root. The use of engine-operated rotating instruments such as burs or reamers makes perforation of the wall of the root likely. When a lateral perforation is suspected, the site must be established; if it is on the buccal

wall surgical correction should be simple, if it is on the palatal it will be extremely difficult.

Apex. Over-zealous instrumentation of a canal may result in perforation through the apical foramen. Calcium hydroxide may be used provide an apical barrier.

Floor of the pulp chamber. Perforations through the floor of the pulp chamber quickly become periodontal problems with furcal bone loss and pocketing, unless they are treated immediately. The perforation may be repaired with a material with a material, which will provide a good seal. Amalgam, glass-ionic cement, or gutta-percha are commonly used. The larger the perforation and the longer it is left the poorer the prognosis. In certain cases an alternative treatment is the removal of the roots. These teeth are difficult to restore and it is also difficult for the patient to maintain a good periodontal condition. In this case the patient was advised to have the tooth extracted and a bridge placed.

Arsenical intoxication.

It is well known, that Arsenical acid is a prothoplazmatic poison.

Toxic single dose – 0.01gr; lethal dose – 0.08-0.1gr.

Dose, applicated on an open pulp horn in pulpitis treatment – 0.0008gr.

In local application Arsenic acid caused tissue necrosis. Pathomorphological changes: coagulation of proteins, necrosis, widening and laceration of blood vessels, and hematoma into the pulp tissue.

- Arsenical paste tends to cause pain of varying intensity during 1-2 hours, when paste was applicated on close pulp chamber or under great pressure.
- Should the coronal seal be disturbed between visits, the mixture, which usually contains arsenic or formaldehyde, could be enter the oral cavity, producting severe toxic

ulceration (necrosis of intradental papilla, gingival mucous, cheek, and some times alveolar bone ⇒arsenical osteomielitis).

- Application to mach devitalizing paste or to long period between visits caused a toxic arsenical pericementitis. In this case cleaning canal with arsenical antidotes is necessary (2% tincture of J_2 or Unitiol).

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