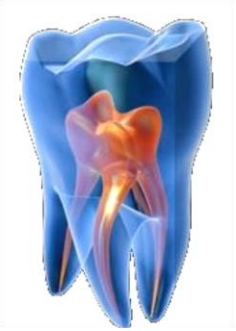
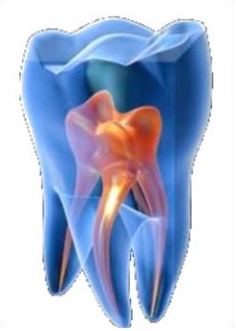
# PULPITIS, ETIOLOGY AND PATHOGENESIS OF PULPITIS, CLASSIFICATIONS, DIAGNOSIS



# Definition

**Pulpitis** - an inflammation of the pulp of a tooth. Pulpitis, in most cases occurs as a complication of tooth decay. Pulp inflammation develops as a result of combined effects of microbial products of their life and decay of organic matter of dentin. Most often in various forms of pulpitis Association found streptococci and lactobacilli bacteria, at least - negative staphylococci.

From the cavity microorganisms penetrate into the pulp to dentinal tubules. This is the most likely route of penetration of microorganisms into the pulp. Less common hematogenous route of penetration (through the apical hole microbes get into the pulp with sinusitis, periodontitis, etc.).



# Etiology

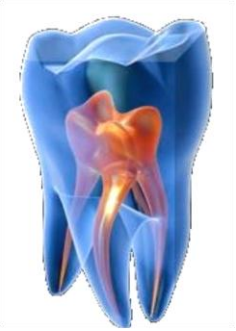
Pulpitis (pulpitis) - inflammation of the dental pulp as a result of exposure to various stimuli.

1. The most common cause of pulpitis are the microorganisms and their metabolic products falling within the pulp chamber of the cavity through the dentinal tubules.

   Most authors consider the main causative agents of inflammation of the pulp coccoid forms of bacteria, including hemolytic and no hemolytic streptococci, diplococci, staphylococci, gram-positive rods, streptobatsilly and lactobacillis.

   Infection of the pulp probably accidental opening the pulp chamber during the treatment of caries, as well as the retrograde route from the deep periodontal pocket, the maxillary sinus when it is inflammation (sinusitis), osteomyelitis, or periodontitis in the adjacent tooth.

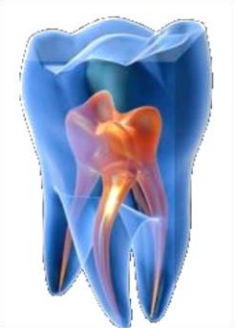
   Perhaps the penetration of infection into the pulp through the blood and the lymphatic system in acute infectious diseases.



2. The second most common etiologic factor for the development of pulpitis may be the acute or **chronic injury**: mechanical, physical and chemical.

**Acute mechanical injury** occurs when you pluck a part of the crown of the tooth exposing the pulp horns, at the turn of the root, with injury of neurovascular bundle in the root apex (eg, stroke),the opening of the pulp horns in the treatment of dental caries, or tooth for a crown dissection.

For chronic mechanical trauma of the pulp are **pathological dental abrasion**, which can lead to exposure of the pulp horns, over-filling the treatment of deep caries, pressure dentiklis (calcification stone).

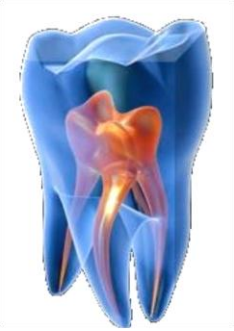


2a. Physical injury can lead to overheating of the pulp during the preparation of cavities or tooth for a crown.

- This can occur when using the blunt burs, or at work on a turbine installation for a long time without water cooling [Ivanov, BC, 1968].
- The imposition of amalgam fillings with isolating thing base sand leads to chronic physical irritation of the pulp, as the metal is a good conductor of thermal stimuli.

2b. Chemical pulp injury may occur as a result of the following factors:

- The use of strong antiseptic in high concentration in deep cavities (3% solution of chloramines, 96% solution of alcohol, 3% solution of hydrogen peroxide, ether);
- applied of a permanent obturation without curative line for treatment of deep caries, the use of filling materials that have toxic effects, with isolate base (Acriloksid, Carbodent, Evicrol, etc.);
- the application of therapeutic pads with a strong alkaline medium pH 12-14 (Calmetsin, Caltsin-paste), which can lead to necrosis of the pulp;



- adding a crystal of thymol, iodoform, potassium nitrate in the base;
- use of allergenic filling materials and drugs without adverse allergic status of the patient (acrylic, zinc-eugenol paste sulfanilamidy, antibiotics, etc.), the treatment of deep caries in elderly people of age with a low reactivity of the organism with decompensated form of tooth decay.

Among the etiologic factors include pulpitis denticlis and petrificats (calcification stones) in the cavity of the tooth.

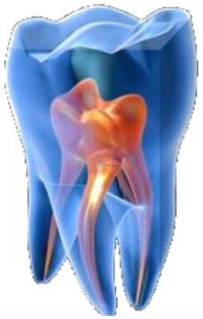
Petrificats (calcification stones) dental pulp can be single or multiple, that is, there is focal or diffuse deposition of mineral salts, which can irritate the nerve endings in the pulp and squeeze the vascular formation, disrupting the microcirculation.

The deposition of calcium salts can be traced mainly along the large vessels and nerves, which, of course, can lead to spontaneous pain in the tooth, especially when changing position of the head.

Dentiklis (Bricked) - is similar to dentin formation of various sizes and shapes that are formed in the crown and the root pulp as a result of chronic mechanical trauma of the tooth or by somatic chronic diseases.

There are:

- free,
- parietal,
- interstitial (bricked) dentiklis.

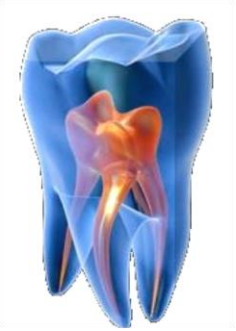


# Pathogenesis

The pulp is a powerful anti infective barrier. With the depletion of protective forces of the pulp and the impact of virulent microorganisms and their toxins develops an inflammatory reaction that may be reversible at some stages.

Nonspecific and specific factors of resistance of the organism involved in the regulation of flow and the elimination of inflammation of the pulp of a tooth. The outcome (stopped) of the inflammation depends on the following factors:

- the virulence of microorganisms and their toxins;
- the duration of the impact of stimuli;
- the resistance of the pulp;
- the general condition of the human body;
- the age of the patient;
- the intensity of dental caries;
- the periodontal status.



Sensitization of the pulp must be considered, which develops well before clinical signs of inflammation as a result of existing caries process.
In the early inflammatory changes in the pulp of developing a functional nature, which are later transformed into the structure.
In response to prolonged exposure to microorganisms and their toxins are cell death and release of large quantities of lysosomalenzymes (proteolytic, glycolytic, lipolytic), which initiates a cascade of reactions.

Time of acute pulpitis does not exceed14 days.

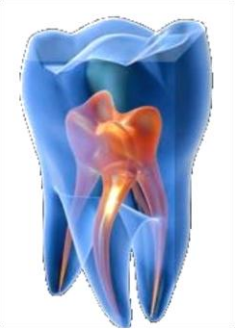
Any inflammatory response consists of three components: the alteration, exudation and proliferation.

In the process of alteration of subcellular structures are damaged, in particular, mitochondrial damage causes a decrease in redox processes [A. Grigorian, 1965].

As a result of damage and the collapse of lysosomes released a large number of hydrolytic enzymes, enzymes of glycolysis, lipolysis, are activated by hydrolysis, released organic acids of the Krebs cycle, lactic acid, amino acids.

This leads to saturation of the pulp by hydrogen ions and an increase in osmotic pressure.

After a brief constriction of arterioles is their expansion, while expanding venules and capillaries.



There is increased blood flow, blood clots, swelling of the vascular wall in an acidic medium, the distance parietal white blood cells (leukocytes), increased blood clotting, thrombosis [Panikarovskiy V.V., 1989].

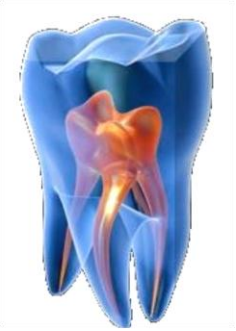
As a result of these processes is hampered by removal of the products of metabolism, increases oxygen starvation.

Depolymerized ground substance, it is alkalization, increases tissue hypoxia. The protective function of the intermediate material is sharply reduced due to changes in its structure (voids interspersed with dense areas).

There are severe disturbances in the microcirculatory system, small hemorrhages. There is a disorganization of odontoblasts, vacuolization of them, cariopicnoz, cariorexis and cariolisis.

With the stage of alteration is developed and is exacerbated by the stage of exudation.

In pulp tissue swelling increases, there is a serous fluid, which in rare cases it may dissolve, that is, inflammation becomes the opposite development, but more often 6-8 hours from the beginning is transformed into a purulent inflammation.



Pulp abscess is formed, around which there is a serous inflammation subsides to the periphery.

In dentinal tubules in a cavity partially out inflammatory transudate than the pressure drop is reached in the pulp cavity of the tooth and the pain subsided for a while.

If the abscess is opened in the cavities, the acute inflammation becomes chronic.

Another outcome of acute diffuse pulpitis may be the death of the pulp as a result of severe hypertension with subsequent irreversible changes.

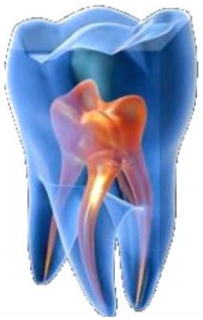
In the transition of acute to chronic fibrous pulpitis on the periphery of the abscess initially formed granulation tissue.

In the future site of the inflamed pulp undergoes fibrous degeneration, which corresponds to the stage of proliferation.

Acute pulpitis may become chronic in the case of gangrenous putrefactive microorganisms entering into the cavity of the tooth through the drain hole in the dentin.

Morphologically, necrosis of the observed surface area, adjacent to the cavity, and the proliferation of granulation tissue in the underlying layer.

The site of necrosis over time extend to the entire coronal part, and then the root pulp, which ultimately leads to the development of periodontitis.



In some cases, acute pulpitis may become a chronic hypertrophic, which is more common in young age.

In this case, the stage of proliferation predominates over the stages of alteration and exudation.

The site of the pulp abscess after opening it in a cavity is replaced by granulation tissue, which is a result of chronic injury (thermal, mechanical, chemical) grows in the direction of the cavity.

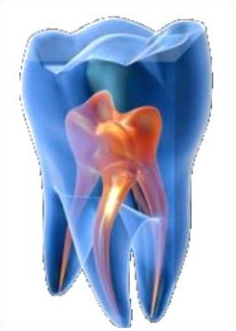
In case of contact with the stratified squamous epithelium of the mucous membranes of the mouth on this fabric is formed of pulp polyp.

Chronic hypertrophic pulpitis may become chronic gangrenous pulpitis and periodontitis.

Chronic forms of pulpitis may from time to time to give an exacerbation, which is observed during the mechanical closing of the drain hole from the cavity of the tooth, reducing the reactivity of the organism as a result of acute and chronic common illnesses, stress.

In the pulp, along with changes characteristic of chronic inflammation (the appearance of foci of sclerosis, fibrosis, areas of salt deposits), there are areas of acute inflammation, microabscesses, edema and exudation phenomena.

Thus, in various forms of pulpitis effects of alteration, exudation and proliferation must exist, but is a predominance of one process over another. For example, in acute forms of pulpitis prevalent phenomenon of exudation in chronic gangrenous - alterations in chronic hypertrophic - proliferation.



# Classifications of Pulpitis

**WHO classification (World Health Organization) – Russian Version:**

К.04. Diseases of the pulp:

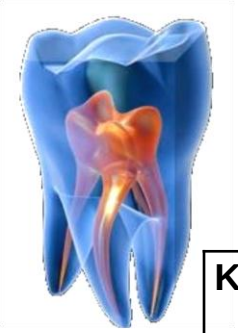
К.04.0. Pulpitis

- К.04.00. hyperemia of the pulp;
- К.04.01. acute
- К.04.02. Pus (abscess of the pulp)
- К.04.03. chronic
- К.04.04. chronic ulcerative
- К.04.05. chronic giperplazic pulpitis (pulp polyp)
- К.04.08. Other specified pulpitis
- К.04.09. unidentified pulpitis

К.04.1. Necrosis of the pulp. Gangrene of the pulp

К.04.2. The stones of the pulp. Dentriklis.

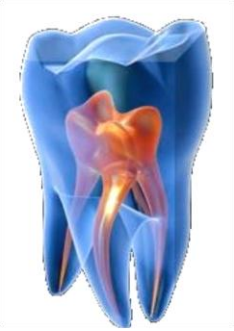
К.04.3. Abnormal formation of dental hard tissues in the pulp. Irregular secondary dentine.



## WHO classification (World Health Organization) – Original Version:

Diseases of pulp and periapical tissues

| | |
|---|---|
| **K04.0 Pulpitis:** | • NOS (National Occupational Standards) acute<br>• chronic (hyperplastic)(ulcerative)<br>• irreversible<br>• reversible |
| **K04.1 Necrosis of pulp** | Pulpal gangrene |
| **K04.2 Pulp degeneration** | Denticles<br>Pulpal:<br>• calcifications<br>• stones |
| **K04.3 Abnormal hard tissue formation in pulp** | Secondary or irregular dentine |
| **K04.4 Acute apical periodontitis of pulpal origin** | Acute apical periodontitis NOS |
| **K04.5 Chronic apical periodontitis** | Apical or periapical granuloma<br>Apical periodontitis NOS |
| **K04.6 Periapical abscess with sinus** | • Dental abscess with sinus<br>Dento-alveolar abscess with sinus |
| **K04.7 Periapical abscess without sinus** | • Dental abscess NOS<br>• Dento-alveolar abscess NOS<br>Periapical abscess NOS |
| **K04.8 Radicular cyst** | Cyst:<br>• apical (periodontal)<br>• periapical<br>• residual radicular<br>***Excl.:*** lateral periodontal cyst (K09.0) |
| **K04.9 Other and unspecified diseases of pulp and periapical tissues** | |



## Classification of E.M. Gofung:

I. Acute pulpitis:
- a partial,
- a general,
- purulent.

II. chronic:
- Simple,
- hypertrophic,
- gangrenous.

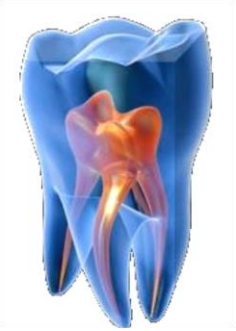
## Classification of the Moscow Medical Stomatological Institute:

Acute pulpitis:
- focal,
- diffuse.

Chronic pulpitis:
- fibrotic,
- gangrenous,
- hypertrophic.

Exacerbation of chronic pulpitis.

State after a partial or complete removal of the pulp.



## Classification of lesions of the pulp of M. Ghafar and C. Andreescu (Bucharest 1990):

I. Acute pulpitis:
Serous;
- ❑with limited morphologic lesions (acute coronary partial or serous pulpitis);
- ❑with the morphological lesions of the pulp all over the (full or acute coronary root serous pulpitis);

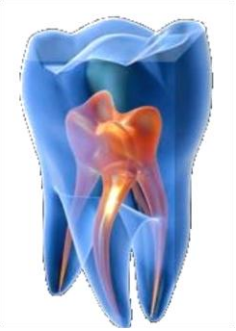
Purulent :
- ❑With limited lesions (acute coronary serous or purulent partialpulpitis);
- ❑covering the entire pulp (full or acute coronary root purulent pulpitis);

2. Chronic pulpitis:
Closed (not communicating with the external environment);
- ❑chronic pulpitis proper closed;
- ❑chronic granulomatous pulpitis internal (internal granuloma Palace).

Open:
- ❑Ulcerative;
- ❑Granulomatous (polypous).

## Classification of International Diagnosis Code (IDC):

ICD-9-CM 522.0 - is a billable medical code that can be used to specify a diagnosis on a reimbursement claim: **2012 ICD-9-CM Diagnosis Code 522.0 - Pulpitis**

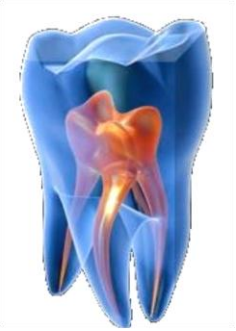


# Diagnostics

**Clinical manifestations of pulpitis are very diverse. Patients complain of severe pain, localized in a particular tooth (acute focal pulpitis). Pain provoked by thermal irritants, in the beginning of the process of pain accur from the cold stimulus, and at later stages -from the hot irritants.**

**The pains continued for 15-20 minutes (acute focal pulpitis).Episodes of pain at first - intermittent , over time - are becoming more frequent. Pain may occur spontaneously, especially in the evening or at night. Attacks of pain may pass spontaneously. But often for pain relief is necessary to use analgesics.**

**Pain attacks in the beginning of the disease short-time (15-20 minutes) with a long "light periods" (painless) - intervals (2-4 hours). With the progression of the disease - increased pain during the 4 hours to 2, and the "light periods" (painless) is reduced to 10-20 minutes.**



Night pain prevails over the day, as night dominated by the parasympathetic nervous system activity of the sympathetic. At night, the heart rate slows, and as a consequence – slows circulation, which leads to accumulation of toxic products in the pulp.

This produces irritation of nerve receptors - there is pain.

Pain can be unbearable, progressive, sharp. In acute diffuse pulpitis French author describes the pain as "crazy tooth".

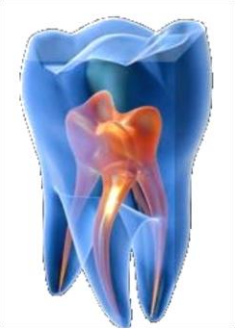
The pain may irradiate from diseased tooth to adjacent teeth and the region.

The pain irradiate along the branches of the trigeminal nerve.

Irradiation zone:

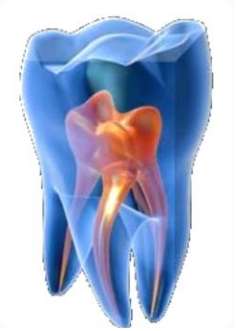
- The temporal region;
- Orbital (eye) area;
- Under the mandible (lower jaw);
- In the occipital region;
- The muscle Sterno-Cleino-Mastoideus.

It is important that the irradiation does not pass the medial (middle) line of the face.



# Clinical examination

- The tooth is a normal color, with a deep cavity with the crushed dentin;
- After removing food debris and pulverized dentine can be seen a thin layer of pigmented dentin;
- Probing causes intense pain reaction (acute focal pulpitis - at one point and diffuse - around the bottom of the cavity).
- Percussion of the tooth (vertical) - painless (acute focal pulpitis) and slightly painful (acute diffuse pulpitis);
- The temperature probe - is an intensive long-time pain;
- EDI is an additional method of investigation. Healthy dental pulp responds to a current of 4–6 uA, with deep caries up to 12-18 uA, with acute focal pulpitis to 20 uA, with acute pulpitis generally up to 30-45 uA, chronic fibrous pulpitis to 35 uA. (1mkA = 1μA = 1uA=0,001mA=0,000001A).



- **Indicators of the current 60 uA talk about the death of the coronal pulp, and more than 100 uA - about death and root pulp.**
- **X-ray diagnosis is used as an additional method to detect hidden cavities, dentiklis (calcification stones) detection and identification of changes in the periodontium.**
- **Changes in the periodontium on radiographs in chronic pulpitis detected in 28% of cases (extensions periodontal ligament, resorption of the root apex).**

# The END