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**PULPITIS: ETIOLOGY,
PATHOGENESIS,
CLASSIFICATION**

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МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ БЕЛАРУСЬ
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1-я КАФЕДРА ТЕРАПЕВТИЧЕСКОЙ СТОМАТОЛОГИИ

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**ПУЛЬПИТ: ЭТИОЛОГИЯ, ПАТОГЕНЕЗ,
КЛАССИФИКАЦИЯ**

**PULPITIS: ETIOLOGY, PATHOGENESIS,
CLASSIFICATION**

Учебно-методическое пособие



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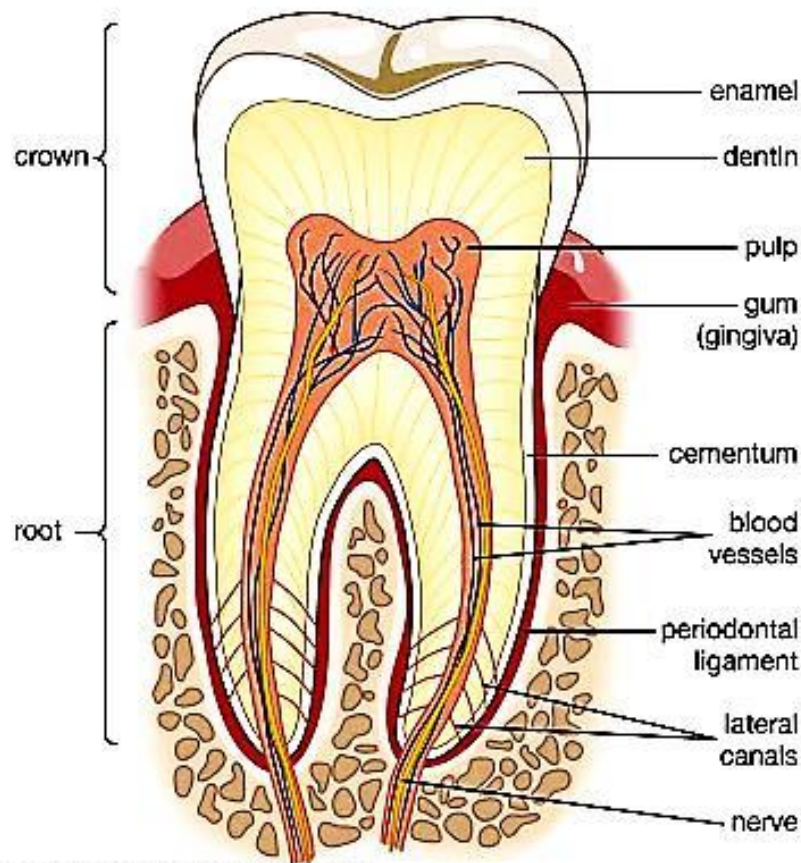
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DEFINITION

The dental pulp is soft tissue of mesenchymal origin located in the center of a tooth (fig. 1). It is the principle source of pain in oral cavity and also a major site of attention in endodontics and restorative procedures. It consists of specialized cells, odontoblasts, arranged peripherally in direct contact with dentin matrix. This close relationship between odontoblasts and dentin is known as «pulp-dentin complex». The pulp is connective tissue system composed of cells, ground substance, fibers, interstitial fluid, odontoblasts, fibroblasts and other cellular components. Pulp is actually a microcirculatory system consisting of arterioles and venules as the largest vascular component. Due to lack of true collateral circulation, pulp is dependent upon few arterioles entering through the foramen. Due to presence of the specialized cells, i. e. odontoblasts as well as other cells which can differentiate into hard tissue secreting cells; the pulp retains its ability to form dentin throughout the life. This enables the vital pulp to partially compensate for loss of enamel or dentin occurring with age. The injury to pulp may cause discomfort and the disease. Consequently, the health of pulp is important for successful completion of the restorative procedures.



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Fig. 1. Relation of pulp with its surrounding structures

Thus the knowledge of pulp is essential not only for providing dental treatment, but also to know the rationale behind the treatment provided.

Composition of the pulp:

1. Cells:

- odontoblasts;
- fibroblasts;
- undifferentiated mesenchymal cells;
- defense cells (macrophages, plasma cells, mast cells).

2. Matrix:

- collagen fibers (type I, type II);
- ground substance (glycosaminoglycans, glycoproteins, water).

3. Blood vessels (arterioles, venules, capillaries).

4. Lymphatics (draining to submandibular, submental and deep cervical nodes).

5. Nerves:

- subodontoblastic plexus of Raschkow;
- sensory afferent from V nerve and superior cervical ganglion.

When pulp examined histologically, it can be distinguished into four distinct zones from periphery to center of the pulp.

Zones of pulp are:

- Odontoblastic layer at the pulp periphery.
- Cell free zone of Weil.
- Cell rich zone.
- Pulp core.

Odontoblastic layer

Odontoblasts consist of cell bodies and cytoplasmic processes. The odontoblastic cell bodies form the odontoblastic zone whereas the odontoblastic processes are located within predentin matrix. Capillaries, nerve fibers (unmyelinated) and dendritic cells may be found around the odontoblasts in this zone.

Cell free zone of Weil

Central to odontoblasts is subodontoblastic layer, termed cell free zone of Weil. It contains plexuses of capillaries and small nerve fiber ramifications.

Cell rich zone

This zone lies next to subodontoblastic layer. It contains fibroblasts, undifferentiated cells which maintain number of odontoblasts by proliferation and differentiation.

Pulp core

It is circumscribed by cell rich zone. It contains large vessels and nerves from which branches extend to peripheral layers. Principal cells are fibroblasts with collagen as ground substance.

Important features of the pulp:

- Pulp is located deep within the tooth, so defies visualization.
- It gives radiographic appearance as radiolucent line.
- Normal pulp is a coherent soft tissue, dependent on its normal hard dentin shell for protection. Therefore, once exposed, it is extremely sensitive to contact

and temperature but this pain does not last for more than 1–2 seconds after the stimulus is removed.

- Pulp is totally surrounded by dentin which limits the area for expansion and restricts the pulp's ability to tolerate edema.

- Pulp has almost total lack of collateral circulation, which severely limits its ability to cope with bacteria, necrotic tissue and inflammation.

- Pulp consists of unique cells, the odontoblasts, as well as cells that can differentiate into hard-tissue secreting cells. These cells form dentin and/or irritation dentin in an attempt to protect pulp from injury.

- Pulpal responses are unpredictable.

- The innervation of pulp tissue is both simple and complex. Simple in that there are only three nerve endings and consequently the pulp lacks proprioception. Complex because of innervation of the odontoblast processes which produces a high level of sensitivity to thermal and chemical changes.

- Correlation of clinical signs and symptoms with corresponding specific histological picture is often difficult.

Pulpitis is a condition in which the pulp (nerve) of the tooth becomes inflamed, causing pain and pressure in the tooth. There are varying degrees of pulpitis, from mild to severe (S. Watson, 2014).

When the pulp becomes inflamed, pressure in the pulp chamber affects the nerve and connective tissue in the tooth. Extreme cases of pulpitis may result in a phenomenon called referred pain, causing pain from pulpitis to be detected in unrelated areas of the face and mouth, ultimately making it difficult for the patient and the dentist to pinpoint the exact tooth causing the pain.

Pulpitis is inflammation of dental pulp tissue. Pulpitis is mainly caused by bacteria infection which itself is a secondary development of caries (tooth decay). It manifests itself in the form of a toothache (Wikipedia).

ETIOLOGY

The noxious stimuli responsible for pulp inflammation, necrosis, and dystrophy are legion, ranging from bacterial invasion to hereditary dwarfism. Without question, bacterial invasion from a carious lesion is the most frequent initial cause of pulp inflammation. Paradoxically, an alarming amount of pulp involvement is induced by the very dental treatment designed to repair the carious lesion. An increase in automobile and cycle accidents, as well as accidents from body contact sports, has also brought about an increase in pulp death owing to trauma.

The causes of pulp inflammation, necrosis, and dystrophy are arranged below in logical sequence, beginning with the most frequent irritant, microorganisms (**John I. Ingle**):

I. Bacterial			
A. Coronal ingress		B. Radicular ingress	
1. Caries		1. Caries	
2. Fracture	a. Complete	2. Retrogenic infection	1. Periodontal pocket
	b. Incomplete (cracks, infraction)		2. Periodontal abscess
3. Nonfracture trauma		3. Hematogenic	
4. Anomalous tract	a. Dens invaginatus (aka dens in dente)		
	b. Dens evaginatus		
	c. Radicular lingual groove (aka palatogingival groove)		
II. Traumatic			
A. Acute	1. Coronal fracture	B. Chronic	1. Adolescent female bruxism
	2. Radicular fracture		2. Traumatism
	3. Vascular stasis		3. Attrition or abrasion
	4. Luxation		4. Erosion
	5. Avulsion		
III. Iatral			
A. Cavity preparation		1. Heat of preparation	
		2. Depth of preparation	
		3. Dehydration	
		4. Pulp horn extensions	
		5. Pulp hemorrhage	
		6. Pulp exposure	
		7. Pin insertion	
		8. Impression taking	
B. Restoration		1. Insertion	
		2. Fracture	
		a. Complete	
		b. Incomplete	
		3. Force of cementing	
		4. Heat of polishing	
C. Intentional extirpation and root canal filling			
D. Orthodontic movement			
E. Periodontal curettage			
F. Electrosurgery			
G. Lase burn			
H. Periradicular curettage			
I. Rhinoplasty			
J. Osteotomy			
K. Intubation for general anesthesia			

IV. Chemical	
A. Restorative materials	1. Cements
	2. Plastics
	3. Etching agents
	4. Cavity liners
	5. Dentin bonding agent
	6. Tubule blockage agents
B. Disinfectants	1. Silver nitrate
	2. Phenol
	3. Sodium fluoride
C. Desiccants	1. Alcohol
	2. Ether
	3. Others
V. Idiopathic	
A. Aging	
B. Internal resorption	
C. External resorption	
D. Hereditary hypophosphatemia	
E. Sickle cell anemia	
F. Herpes zoster infection	
G. Human immunodeficiency virus (HIV) and acquired immune deficiency syndrome (AIDS)	

Coronal caries is by far the most common means of ingress to the dental pulp for infecting bacteria and/or their toxins. Long before the bacteria reach the pulp to actually infect it, the pulp becomes inflamed from irritation by preceding bacterial toxins. *Langeland* observed pulp reactions “with certainty” when superficial enamel fissure caries was found clinically. *Brännström and Lind* observed inflammatory changes in the pulps of 50 of 74 premolars with initial enamel caries on proximal surfaces but with no radiographic evidence of penetration.

Brännström and his associates also demonstrated the alarming rapidity with which bacteria penetrate the enamel. From incipient carious lesions, without cavitation on the enamel surface, microorganisms can reach the dentinoenamel junction. The ensuing gap between the enamel and dentin is completely filled with microorganisms. The infection is then shown to spread not only laterally along the dentinoenamel junction but pulpally as well. It is quite conceivable that a degree of pulp inflammation could develop well before a visual or radiographic break in the enamel becomes apparent. To compound the problem, *Douglass et al.* have claimed that only 60 % of dental caries lesions can be detected by radiographs alone. *Seltzer et al.* have described these pulp changes, from early irritation dentin formation under initial caries, through scattered macrophages and lymphocytes under moderately developed caries, to frank chronic inflammatory exudate under deep carious lesions.

Etiology of pulpal diseases can be broadly classified into three groups:

1. Bacterial:

- caries;
- microleakage around a restoration;
- periodontal pocket and abscess;
- anachoresis.

2. Physical:

a. Mechanical:

- trauma:
 - acute trauma like fracture or avulsion of tooth;
 - iatrogenic dental procedures:
 - pathologic wear like attrition, abrasion, etc.;
 - barodontalgia due to barometric changes.

b. Thermal:

- heat generated by cutting procedures;
- heat from restorative procedures;
- heat generated from electrosurgical procedures;
- frictional heat from polishing restorations.

3. Chemical:

- acids from erosion;
- use of chemicals like monomers, liners, bases, phosphoric acid, or use of cavity desiccants like alcohol.

WEIN classified causes of pulpal inflammation, necrosis or dystrophy in a logical sequence beginning with the most frequent irritant, microorganisms.

1. Bacterial

Most common cause of pulpal injury is bacteria or their products which may enter the pulp through the break in dentin either from:

- caries;
- accidental exposure;
- fracture;
- percolation around a restoration;
- extension of infection from gingival sulcus;
- periodontal pocket and abscess;
- anachoresis (process by which microorganisms get carried by the bloodstream from another source localize on inflamed tissue).

2. Traumatic

- acute trauma like fracture, luxation or avulsion of tooth;
- chronic trauma including parafunctional habits like bruxism.

3. Iatrogenic

Pulp inflammation for which the dentist's own procedures are responsible are designated as dentistogenic pulpitis. Various iatrogenic causes of pulpal damage can be:

- thermal changes generated by cutting procedures, during restorative procedures, bleaching of enamel, electrosurgical procedures, laser beam, etc. can cause severe damage to the pulp if not controlled;

- orthodontic movement;
- periodontal curettage;
- periapical curettage;
- use of chemicals like temporary and permanent fillings, liners, bases and use of cavity desiccants such as alcohol.

4. Idiopathic:

- aging;
- resorption: internal or external.

Radiation injury to pulp

Pulp cells exposed to ionizing radiation may become necrotic, show vascular damage and the interference in mitosis of cells. Irradiation also affects salivary glands causing decreased salivary flow, thereby increasing predisposition to dental caries and pulpal involvement. Radiation damage to teeth depends on dose, source, type of radiation, exposure factor and stage of tooth development at the time of irradiation.

PATHOGENESIS

IMMUNE RESPONSE

Pulpitis is characterized as the immune and neurological responses that are mainly triggered by the invasion of caries-related microorganisms into dentinal tubules and pulp (fig. 2). Pulp reacts to various irritants as do other connective tissues. Degree of inflammation is proportional to intensity and severity of tissue damage. For example, slight irritation like incipient caries or shallow tooth preparation causes little or no pulpal inflammation, whereas extensive operative procedures may lead to severe pulpal inflammation.

In the pulp, just as in other areas of the body, inflammation can be present. Inflammation of the pulp does not take place only when the bacteria in the decay have reached the pulp. Bacterial products may reach the pulp much earlier and begin the inflammatory response. The inflammation may be acute or chronic because just like other tissues in the body, the pulp will react to irritants with innate and/or adaptive immune responses.

Innate immunity in the pulp is not specific but uses receptors to recognize molecular patterns common to microbes to initiate bacterial killing (phagocytosis). The components of the innate response of the dentin/pulp complex to caries include at least the following six: 1) outward flow of dentinal fluid; 2) odontoblasts; 3) neuropeptides and neurogenic inflammation; 4) innate immune cells, including immature dendritic cells (DCs), natural killer (NK) cells, and T cells, as well as 5) their cytokines and 6) chemokines. Although the first two items are not classic components of innate immunity, they are uniquely involved in the initial inflammatory response to caries.

Odontoblasts, (the cells that form dentin) have cellular processes that extend into dentinal tubules and are the first to encounter the caries bacterial antigens. They express low levels of interleukin 8 (IL-8) and genes related to chemokines

and chemokine receptors. The odontoblasts have been shown to attract immature dendritic cells.

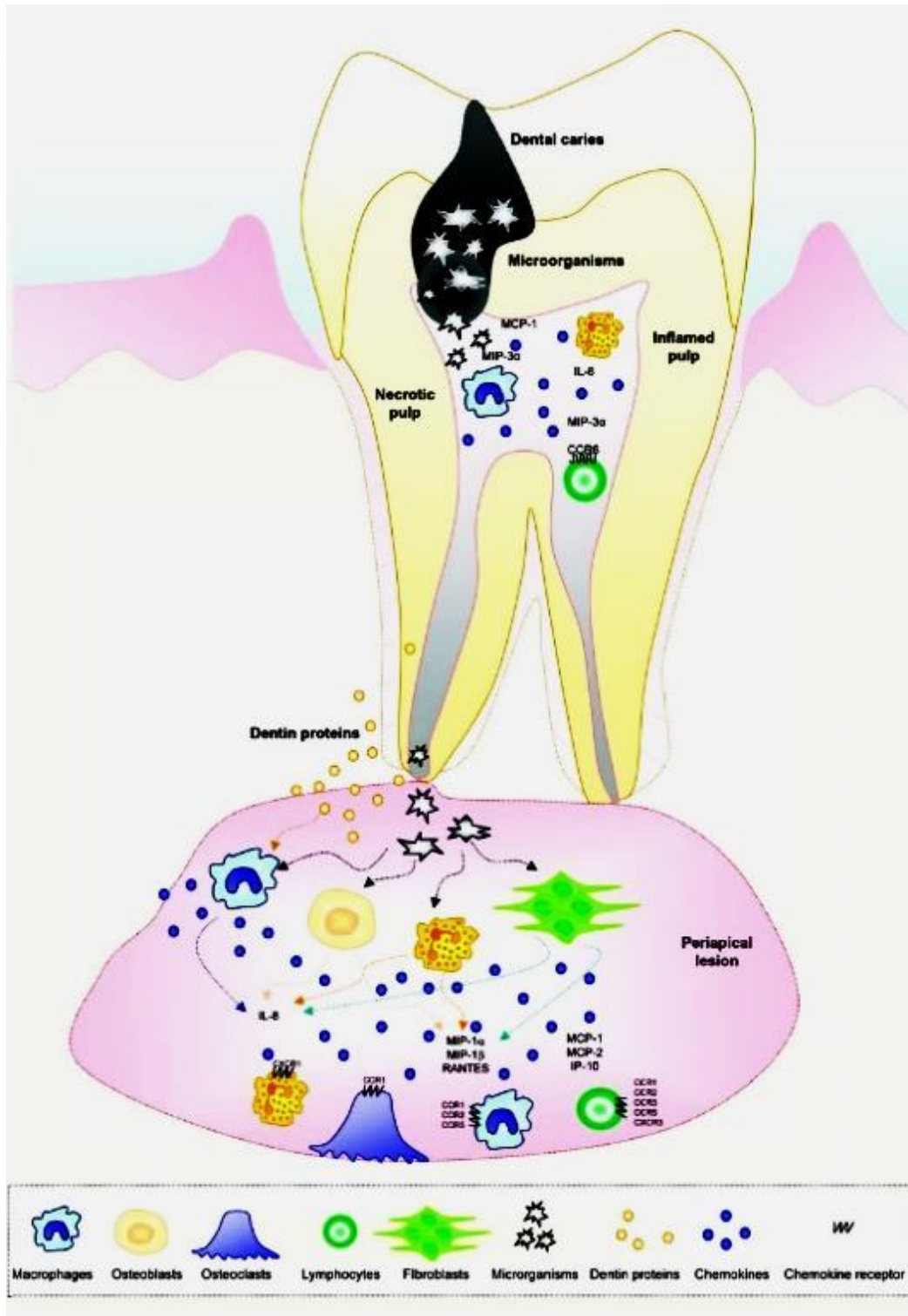


Fig. 2. Immune and neurological responses of the pulp

Dendritic cells (DCs) are a heterogeneous leukocyte (white blood cell) population. DCs in healthy peripheral tissues (steady state) are in an immature state. The cells are capable of sensing microbes as well as antigen capture and processing capabilities. A rapid accumulation of pulpal DCs has been observed beneath cavity preparations, and an increased number of DCs accumulated under

caries. Immature DCs are therefore considered to be part of the innate phase of pulpal immune response.

Persistent infection leads to the activation of adaptive immunity. A transition to an adaptive immune response will take place in the dental pulp as caries and bacteria approach the pulp. Antigens are recognized individually and lines of lymphocytes are developed to produce specific antibodies which attach to the recognized cells and initiate their destruction. Phagocytes remove the remains. B cells and T cells are the major lymphocytes involved.

A variety of cytokines have been observed in the pulp. Patients with symptomatic and asymptomatic irreversible pulpitis have been shown to have an almost 23-fold increase in the cytokine IL-8 in the pulp. Cytokines in the pulp interact with each other. The ultimate effect on pulpal inflammation and healing is dependent upon the integrated actions of these inflammatory mediators.

In addition to the lymphocytes, macrophages also provide defense against certain intracellular pathogens. Activated macrophages can function as class II antigen-presenting cells, similar to pulpal dendritic and B cells. In addition, activated macrophages secrete many inflammatory mediators.

Macrophages in the pulp become activated after receiving two signals. The first is a priming stimulus and the second is an activating signal. The priming stimulus is secreted by activated T-helper cells. The activating stimulus may include bacterial lipopolysaccharides, muramyl dipeptide, and other chemical mediators.

Macrophages are professional phagocytes in innate immune responses. Activated macrophages are effective killers that eliminate pathogens in both innate and adaptive immune responses, and are also important in tissue homeostasis, through the clearance of senescent cells, and in remodeling and repair of tissue after inflammation. The number of macrophages increases with the progression of caries and is always higher than that of DCs at all stages of the caries invasion.

NEUROLOGICAL RESPONSE

According to the Brännström's hydrodynamic theory, activated nociceptors from fluid movement and other irritants through the patent dentine tubules result in pulp pain. Unmyelinated, slow conducting C-fibers aid in feeling a slowset, burning pain. According to neuronal studies, 70–80 % of pulpal axons are unmyelinated. Highly myelinated A δ -fibers, which allow for fast conduction, are responsible for the sharp, shooting pain.

Thus, the stimulus intensities are based on various fibers. Fast-conducting A β and A δ -fibers provide the lowest stimulus intensities (typically referred to as prepain sensations), and those sensations eventually receive higher stimulation levels. The dull aches are associated with C-fibers and slow A δ -fibers. As inflammation intensifies, the A-fibers are increasingly activated. C-fiber innervation and A δ -fibers are polymodal receptors that are sensitive to capsaicin and inflammatory mediators.

The pain mechanisms associated with pulpitis are similar to those of the rest of the body (i. e. receptors, intracellular signaling, transmitters, etc.). The inflammatory mediators act on specific receptors relating to nociceptive neurons, leading to the production of second messengers and activation of phospholipases and protein kinases. The second messengers regulate receptors ion channels that deal with sensitization. The ion channels open based on pain stimuli propagating action potentials in sensory neurons.

In addition, dental caries is more likely to develop pulpitis due to less time for the dental pulp to react and protect itself by occluding the dentinal tubules. Based on the tooth injury, sensory nerve fibers react to pulpitis by growing terminal branches into the adjacent surviving pulp, which also changes the cytochemical phenotype. This neural growth typically lasts a few days and function and form is retained. Thus, pain is poorly localized, and the level of pain stemming from pulpitis varies based on severity, quality, duration, onset, trigger.

Thus, depending on condition of pulp, severity and duration of irritant, host response, pulp may respond from mild inflammation to pulp necrosis. These changes may not be accompanied by pain and thus may proceed unnoticed.

Pulpal reaction to microbial irritation:

- Carious enamel and dentin contain numerous bacteria.
- Bacteria penetrate in deeper layers of carious dentin.
- Pulp is affected before actual invasion of bacteria via their toxic byproducts.
- Byproducts cause local chronic cell infiltration.
- When actual pulp exposure occurs, pulp tissue gets locally infiltrated by PMNs to form an area of liquefaction necrosis at the site of exposure.
- Eventually necrosis spreads all across the pulp and periapical tissue resulting in severe inflammatory lesion.

Degree and nature of inflammatory response caused by microbial irritants depends upon:

- Host resistance.
- Virulence of microorganism.
- Duration of the agent.
- Lymph drainage.
- Amount of circulation in the affected area.
- Opportunity of release of inflammatory fluids.

THEORIES OF PULPITIS

The traditional theory which explained the pulpal inflammation was referred to as strangulation theory.

Strangulation theory

On irritation, there is local inflammation in pulp, which results in vasodilatation, increased capillary pressure and permeability. These result in increased filtration from capillaries into tissues, thus increased tissue pressure.

By this, thin vessel walls get compressed resulting in decreased blood flow and increased venous pressure. This results in vicious cycle, because increase in venous pressure further increases capillary pressure. Consequently, choking/strangulation of pulpal blood vessels occurs because of increased tissue pressure. This results in ischemia and further necrosis.

Current theory

Many studies have shown that increase of pressure in one area does not affect the other areas of pulp. Therefore local inflammation in pulp results in increased tissue pressure in inflamed area and not the entire pulp cavity.

It is seen that injury to coronal pulp results in local disturbance, but if injury is severe, it results in complete stasis of blood vessels in and near injured area. Net absorption of fluid into capillaries in adjacent uninflamed area results in increased lymphatic drainage thus keeping the pulpal volume almost constant.

Limited increase in pressure within affected pulpal area is explained by the following mechanism:

- Increased pressure in inflamed area favors net absorption of interstitial fluids from adjacent capillaries in uninflamed tissues.
- Increased interstitial tissue pressure lowers the transcapillary hydrostatic tissue pressure difference, thus opposes further filtration.
- Increased interstitial fluid pressure increases lymphatic drainage.
- Break in endothelium of pulpal capillaries facilitates exchange mechanism.

Infectious sequelae of pulpitis include apical periodontitis, periapical abscess and osteomyelitis of the jaw. Spread from maxillary teeth may cause purulent sinusitis, meningitis, brain abscess, orbital cellulitis, and cavernous sinus thrombosis. Spread from mandibular teeth may cause angina, parapharyngeal abscess, mediastinitis, pericarditis and empyema.

CLASSIFICATION

International classification ICD-10

K 04 Diseases of pulp and periapical tissues *

K04.0 Pulpitis

- K04.00 Initial (hyperaemia)
- K04.01 Acute
- K04.02 Suppurative [pulpal abscess]
- K04.03 Chronic
- K04.04 Chronic, ulcerative
- K04.05 Chronic, hyperplastic [pulpal polyp]
- K04.08 Other specified pulpitis
- K04.09 Pulpitis, unspecified

K04.1 Necrosis of pulp

- Pulpal gangrene

* The original version of the International Classification ICD (ICD-DA, WHO Geneva, 1995).

K04.2 Pulp degeneration

- Denticles
- Pulpal calcification
- Pulpal stones

K04.3 Abnormal hard tissue formation of pulp

K04.3X Secondary or irregular dentine

- Excludes:** pulpal calcifications (K04.2)
pulpal stones (K04.2)

Pulpitis classification (E. M. Gofung, 1928)

1. Acute pulpitis:
 - a) Partial.
 - b) General.
 - c) Purulent.
2. Chronic pulpitis:
 - a) Fibrous (simple).
 - b) Hypertrophic.
 - c) Gangrenous.

American Association of Endodontists Classification*

- Normal pulp
- Reversible pulpitis
- Symptomatic irreversible pulpitis
- Asymptomatic irreversible pulpitis
- Pulp necrosis
- Previously treated
- Previously initiated therapy

Grossman's Clinical Classification

1. Pulpitis: inflammatory disease of dental pulp.
 - a. Reversible pulpitis:
 - Symptomatic (Acute).
 - Asymptomatic (Chronic).
 - b. Irreversible pulpitis:
 - Acute
 - Abnormally responsive to cold.
 - Abnormally responsive to heat.

* Endodontics: Colleagues for Excellence. Published for the Dental Professional Community by the American Association of Endodontists.

- Chronic
 - Asymptomatic with pulp exposure.
 - Hyperplastic pulpitis.
 - Internal resorption.
- 2. Pulp degeneration:
 - a. Calcific (Radiographic diagnosis).
 - b. Other (Histopathological diagnosis).
- 3. Pulp necrosis:
 - a. Coagulation necrosis.
 - b. Lique faction necrosis.

Ingle's Classification

1. Inflammatory changes:
 - a. Hyperreactive pulpalgia.
 - Hypersensitivity.
 - Hyperemia.
 - b. Acute pulpalgia.
 - Incipient.
 - Moderate.
 - Advanced.
 - c. Chronic pulpalgia.
 - d. Hyperplastic pulpitis.
 - e. Pulp necrosis.
2. Retrogressive changes:
 - a. Atrophic papulosis.
 - b. Calcific papulosis.

Baume's Classification

- Based on clinical symptoms:
- Asymptomatic, vital pulp which has been injured or involved by deep caries for which pulp capping may be done.
 - Pulp with history of pain which is amenable to pharmacotherapy.
 - Pulp indicated for extirpation and immediate root filling.
 - Necrosed pulp involving infection of radicular dentin accessible to antiseptic root canal therapy.

Seltzer and Bender's Classification

- Based on clinical tests and histological diagnosis:
1. Treatable without pulp extirpation and endodontic treatment:
 - Intact uninflamed pulp.
 - Transition stage.

- Atrophic pulp.
 - Acute pulpitis.
 - Chronic partial pulpitis without necrosis.
2. Untreatable without pulp extirpation and endodontic treatment:
- Chronic partial pulpitis without necrosis.
 - Chronic total pulpitis.
 - Total pulp necrosis.

Репозиторий БГМУ

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Репозиторий БГМУ

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