

**Periodontitis: aetiology,
pathogenesis, classifications,
pathomorphology and clinic of
various forms**

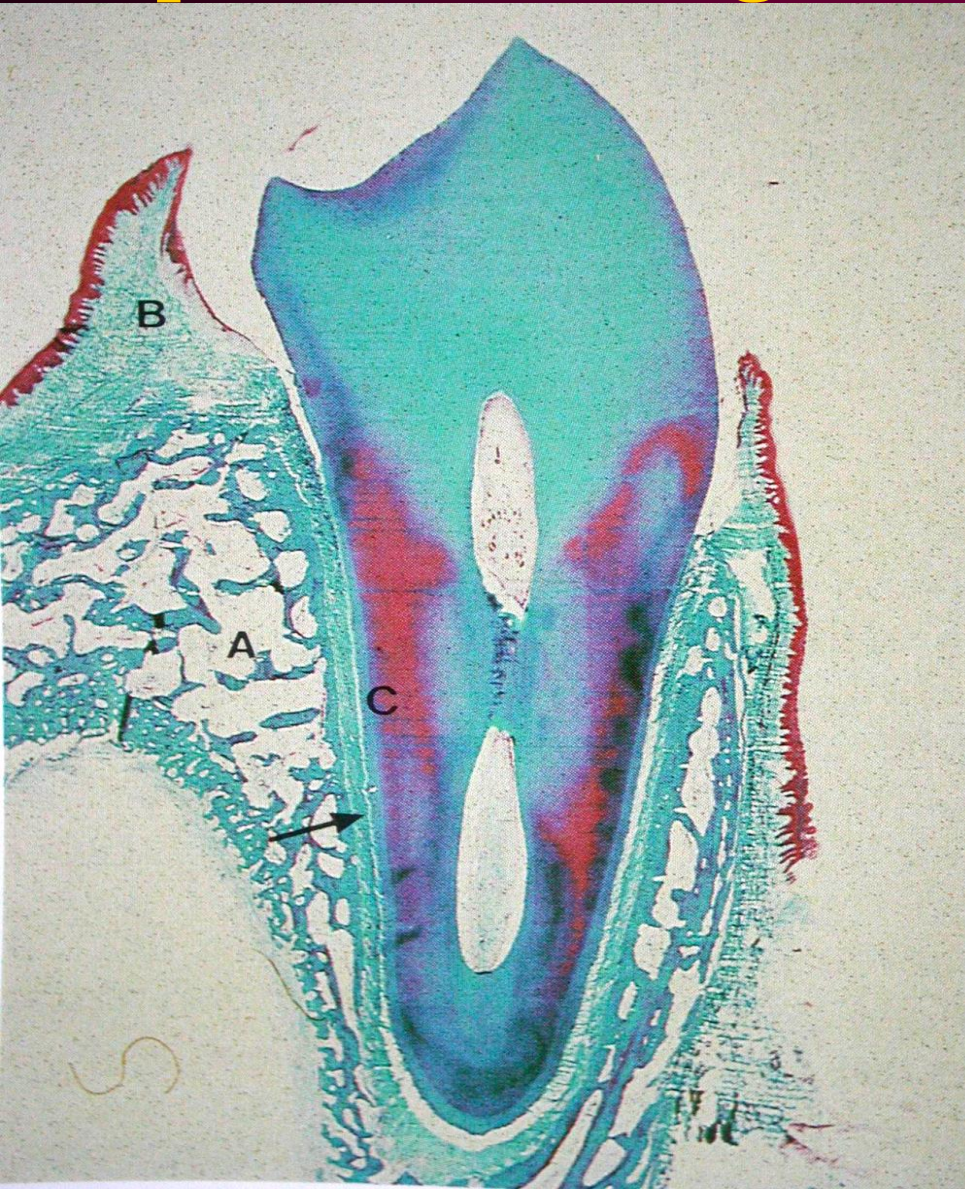
**Lecture for 3-rd year students
of international faculty**

**The lecturer: PhD in Medical Sciences,
Associate Professor
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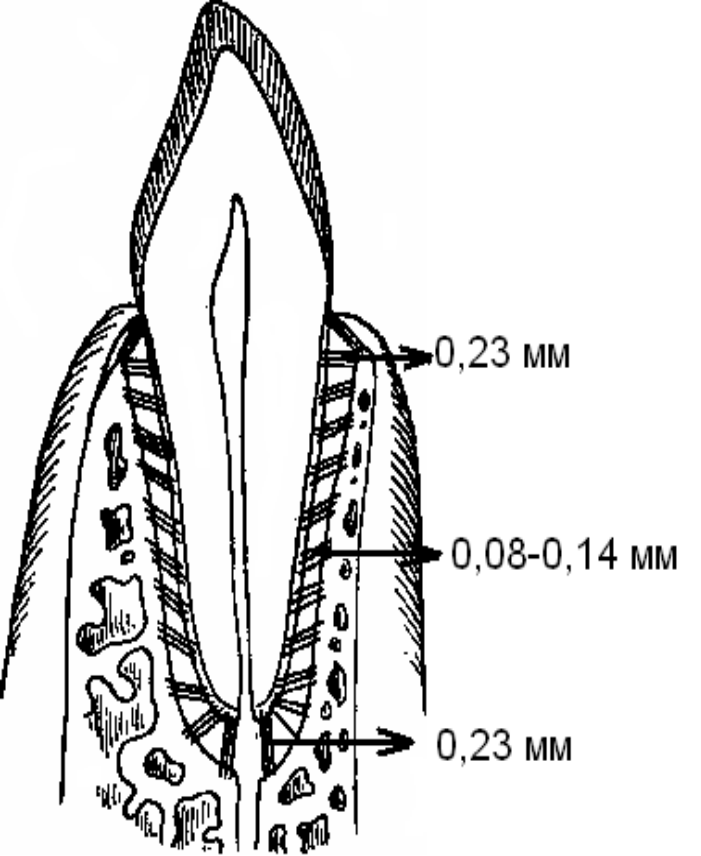
THE PLAN OF LECTURE

1. The structure of periodontium : anatomic, histological, functional features.
2. The aetiology of periodontitis : infectious; traumatic; medicamentous.
3. The pathogenesis of acute and chronic forms of periodontitis.
4. The classification of periodontitis
5. The pathomorphology and clinic of acute forms of periodontitis.
6. The pathomorphology and clinic of chronic forms of periodontitis.
7. The pathomorphology and clinic of chronic periodontitis exacerbation.

Apical periodontitis – inflammation of periodontal ligament around root apex

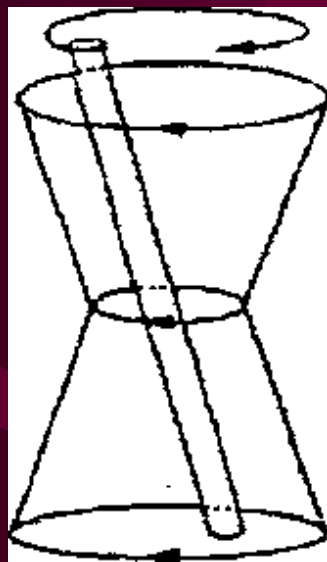


Periodontal ligament (peri – around, dent – a tooth) is the connective tissue which fills periodontal space between tooth root and an alveolus, connecting with root cement on the one side and an internal compact plate of an alveolus on another.



On the average the width of periodontal space on the maxilla is 0.20-0,25 mm, on the mandible is 0,15 - 0,22 mm.

The sizes of periodontal space are not-identical in various sites of an alveolus and make: near the neck and the bottom of an alveolus - 0,23 - 0,27 mm; in a middle third of root - 0,08 - 0,14 mm. Thus periodontal space in an average part of a root has narrowing and under the form reminds a sand-glass.



All this causes feature of micromovements of a tooth in alveolus. The tooth acts as the lever of the first sort with a point of rotation which settles down in an middle part of a root. The greater shoulder is crown and the top half of root, smaller - the lower part of a root. Thus, scope of movement is more in the top part of an alveolus where periodontal space there is wider, than in its lower part.

The width of periodontal space is able to change with the age, tooth development, its function, as a result of pathological process.

After loosening of the antagonist the width decreases to 0,1 – 0,15 mm (because the tooth does not receive functional loading and the periodontium will be atrophy). At the increased loading on a tooth or with age its dilation appears due to the periodontium thickening (hypertrophy). At the hypercementosis contours and size of periodontal space can change (decrease).

Morphologically consists of:

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graph TD; A[Morphologically consists of:] --> B[Cells,]; A --> C[Fibres,]; A --> D[Interceular substance]; B --> B1[Fibroblasts]; B --> B2[Osteoblasts]; B --> B3[Histiocytes]; B --> B4[Cementoblasts]; C --> C1[Collagenic]; C --> C2[Oxytalanic,]; C --> C3[Argirophilic]; D --> D1[Glycosamynoglicans]; D --> D2[abundantly]; D --> D3[penetrated with]; D --> D4[the blood vessels]; D --> D5[and nerves];
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Cells,

Fibroblasts

Osteoblasts

Histiocytes

Cementoblasts

Fibres,

Collagenic

Oxytalanic,

Argirophilic

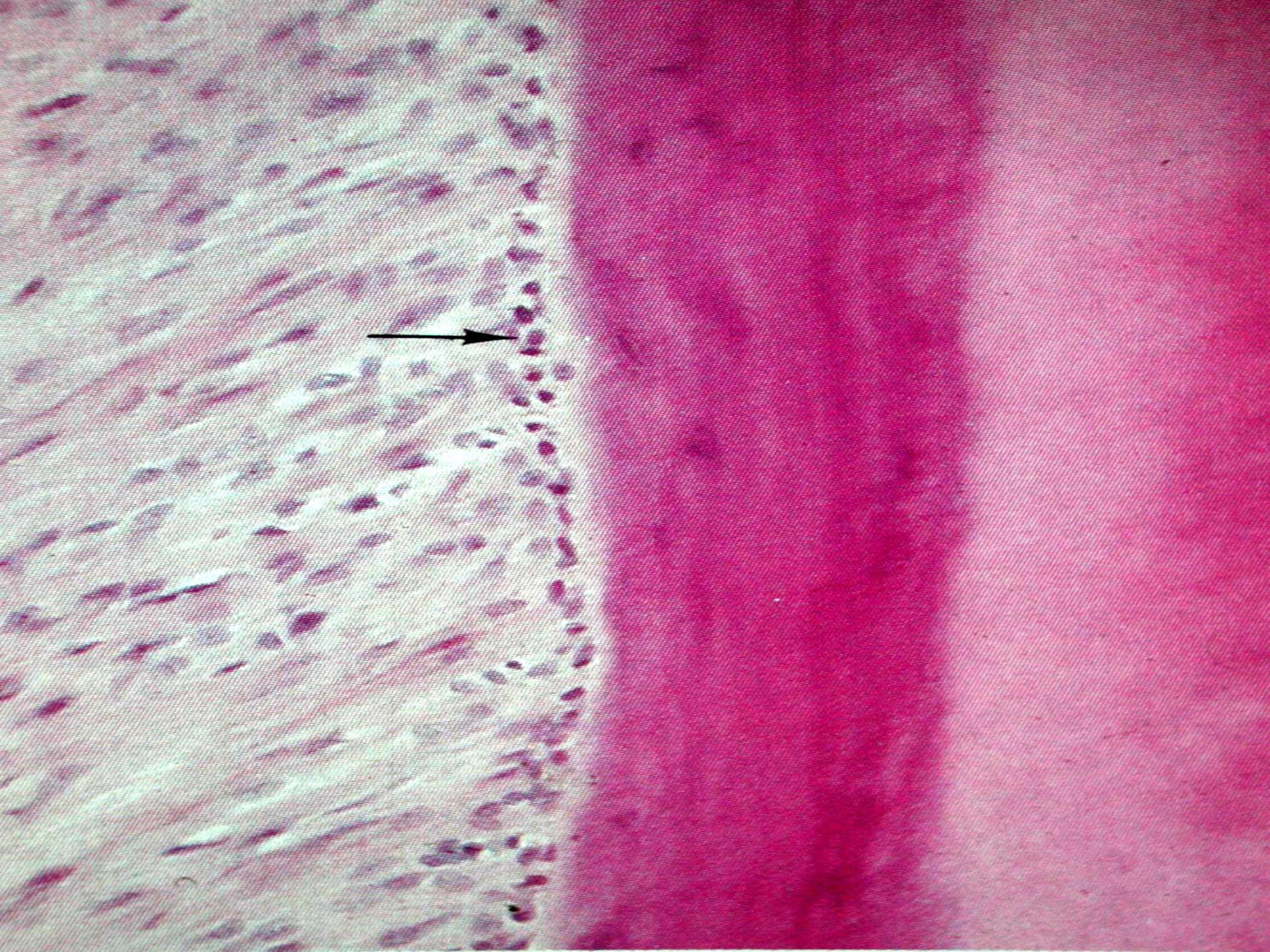
Interceular substance

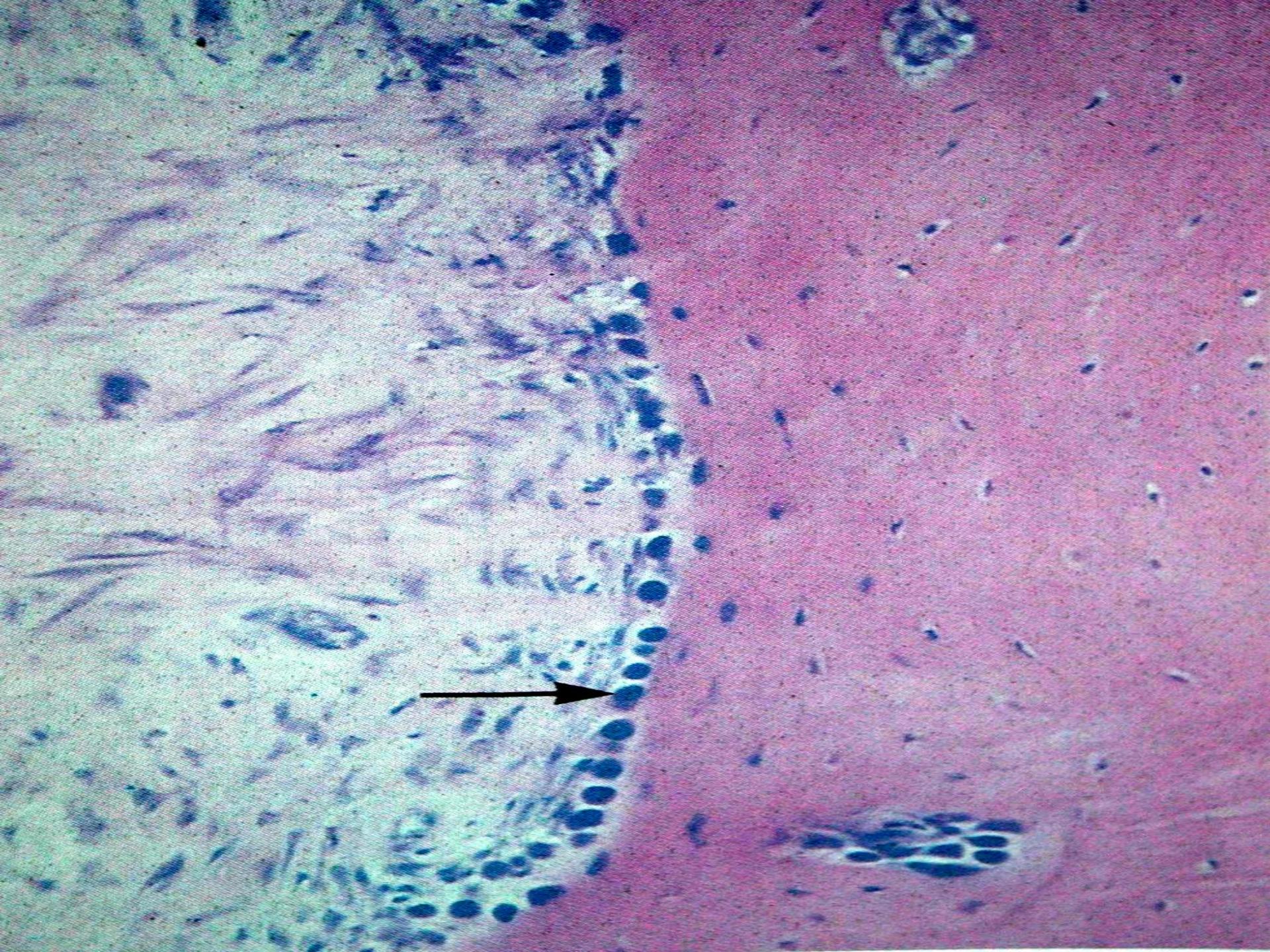
Glycosamynoglicans

abundantly

penetrated with

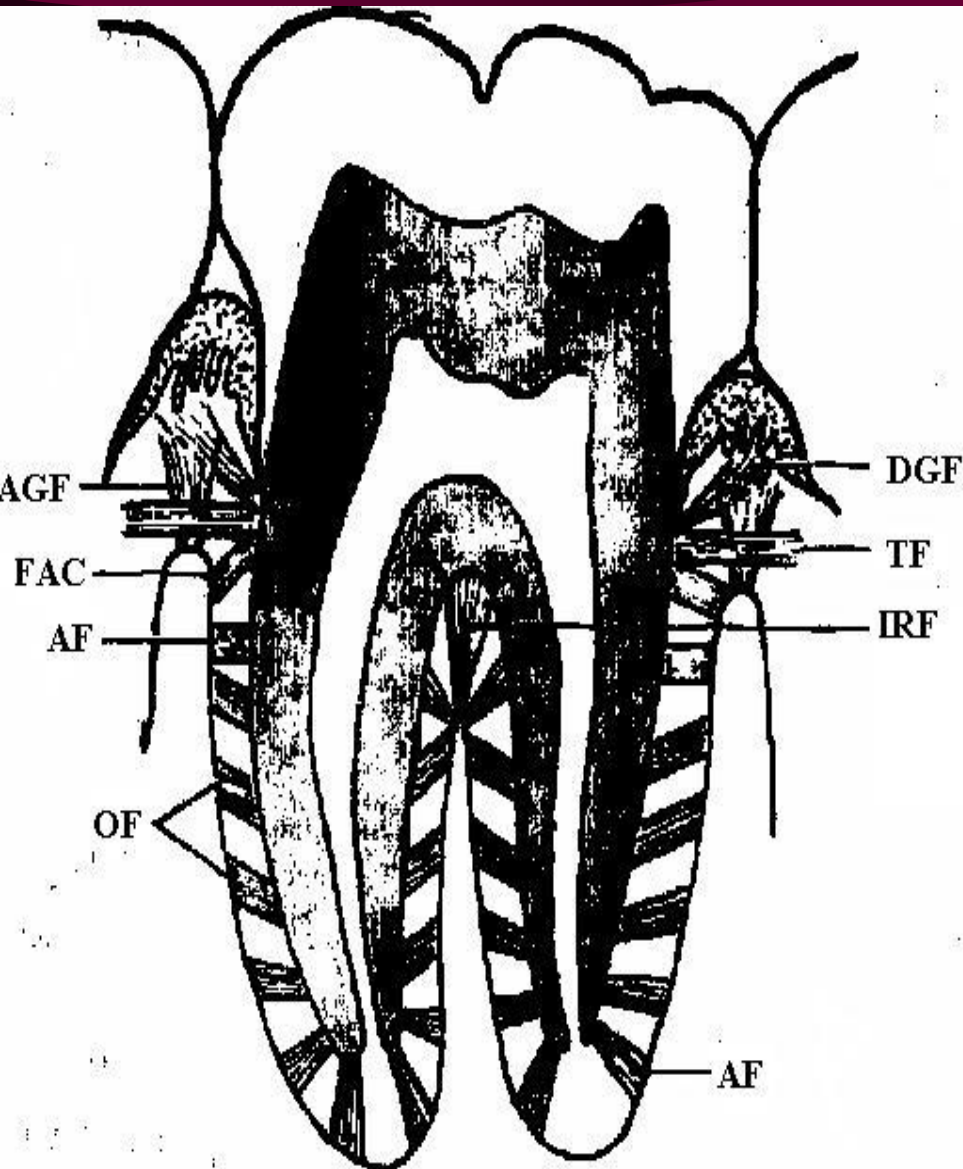
the blood vessels
and nerves







Periodontium fibres



DGF – dentogingival fibres

AGF– alveologingival fibres

FAC – fibers of alveolar crest;

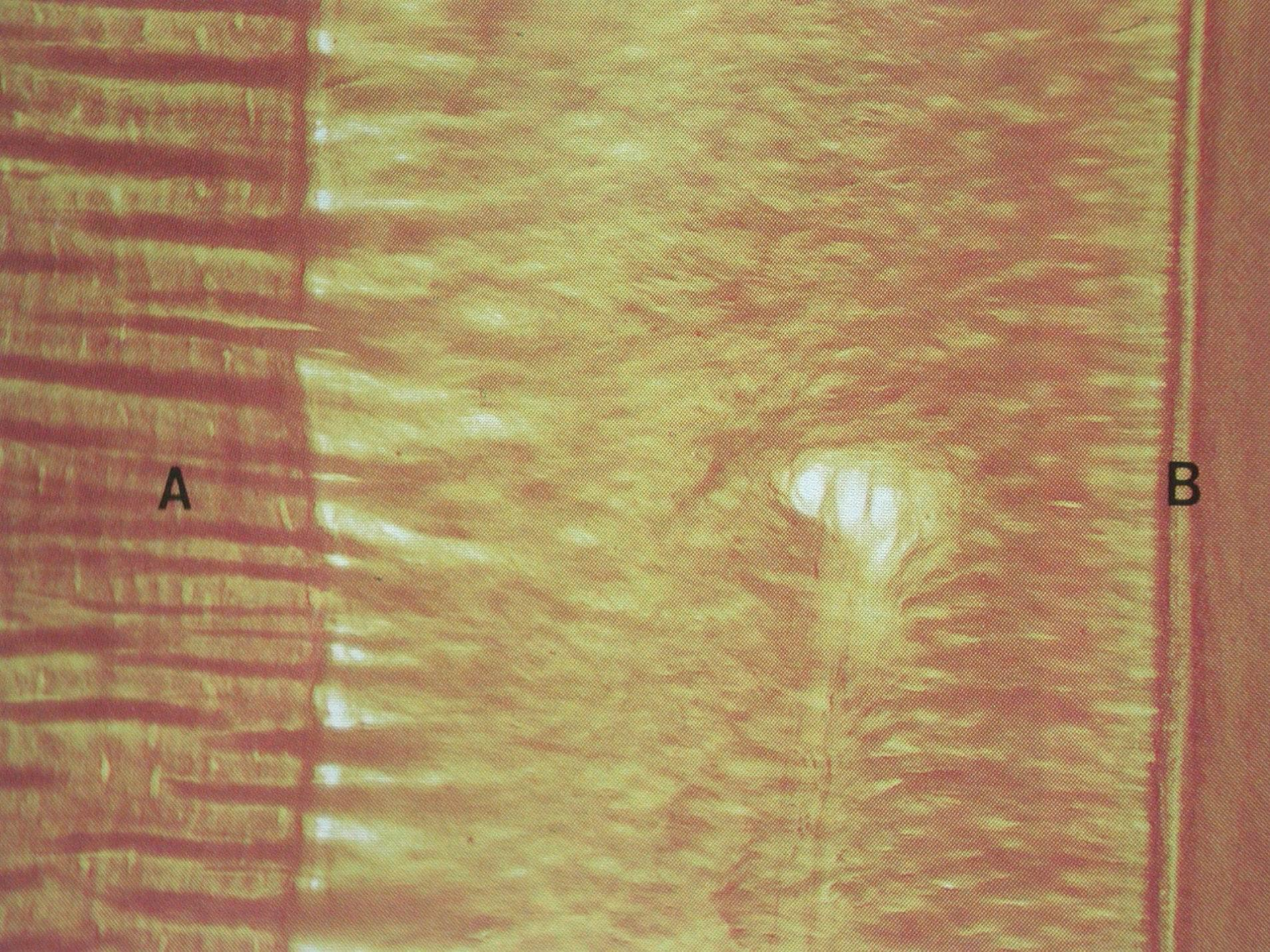
TF – transseptal fibres

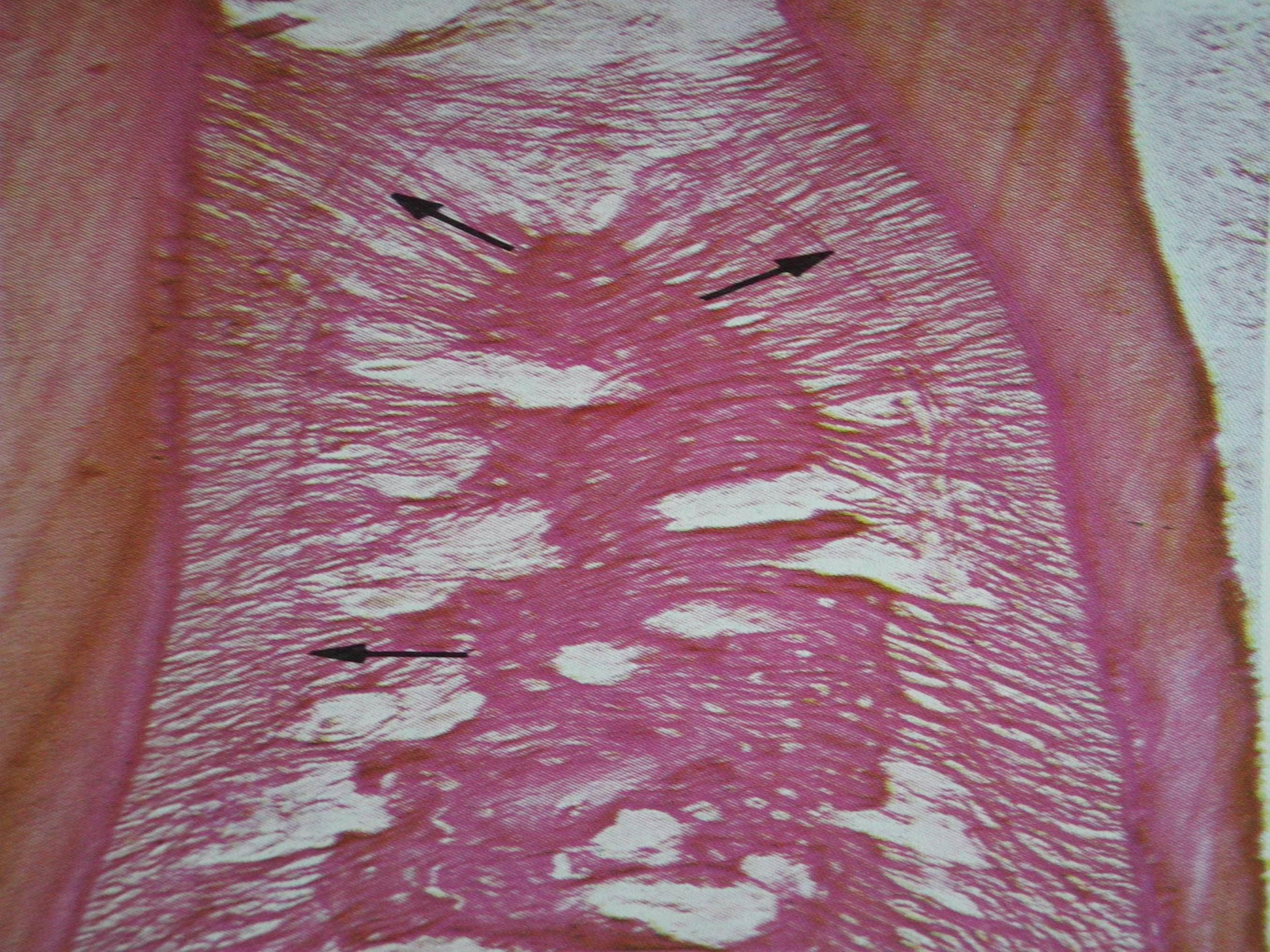
HF – horizontal fibres;

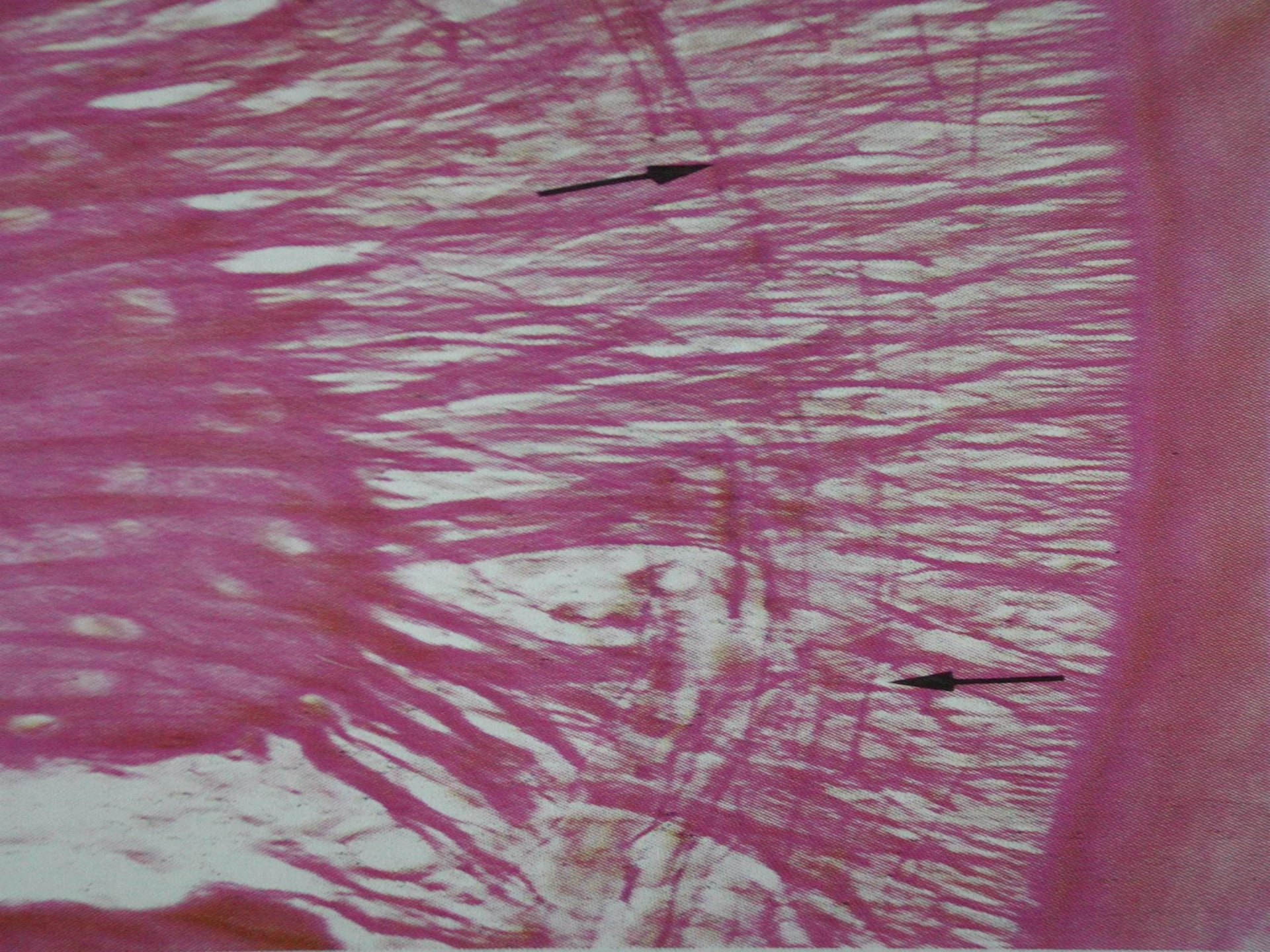
OF – oblique fibres;

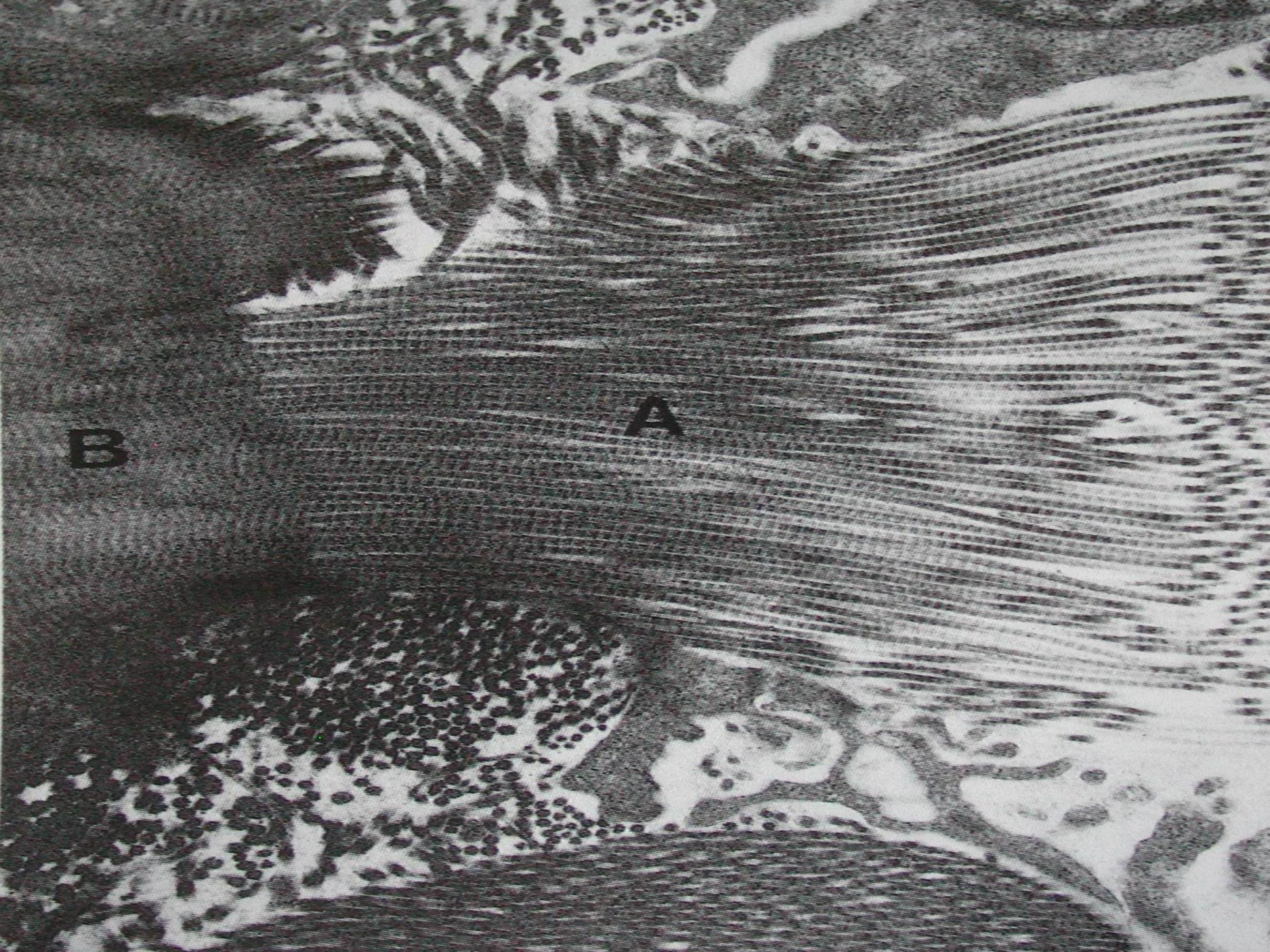
AF – apical fibres;

IRF– interroot fibres





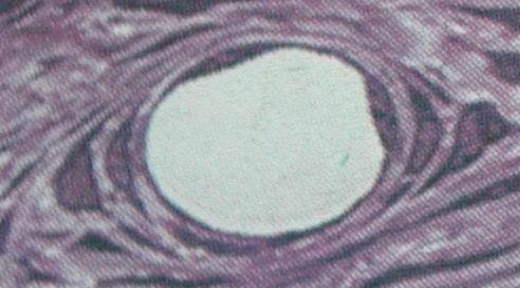




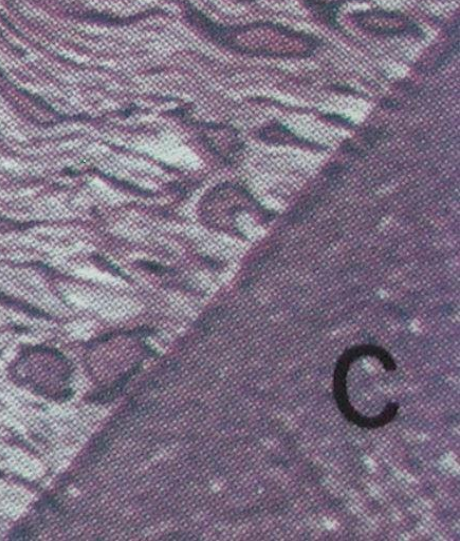
A



B



C



The functions of periodontal ligament

- Hold-supporting;
- Amortized (it is provided with wavy structure of fibers, their direction);
- Sensory (reflexogenic) – perception of chewing loading due to abundance of nervous receptors;
- Barrier (protective) (histiocytes, plasmatic cells);
- Trophic;
- Plastic (reparation) (fibroblasts, osteoblasts, cementoblasts)

3. By aetiology:

Periodontitis

Infectious

Not infectious

Traumatic

Medicamentous

acute
trauma

chronic
trauma

toxic

allergic

Microflora:
association of
Streptococcus,
Lactobacterium
Gram + bacillus
Fusospirochetes
and their toxins

Household,
sport, work
trauma,
accompanied
with injury,
bruise,
dislocation,
fracture

Overconture
filling,
pressure of
mouthpiece,
bad habits:
gnawing of
pen, holding
of needles,
hobnail
between teeth

Arsenium,
Formalinum,
Tricresolfor-
malinum,
Phenolum,
Resorcinum,
AgNO₃,
Eugenol

Antibiotics,
iodine-
containing
agents,
Phenolum,
Eugenol

Ways of entering of infection to the periodontium

1. Through a tooth cavity and root canals;
2. Through odontogingival pocket;
3. By continuation (per contineatatem) - from nearby infection focus (osteomyelitis, osteitis, antritis, purulent periodontitis of the nearby tooth, suppuration of cyst);
4. Hematogenous, lymphogenous.

4 .The pathogenesis – the mechanism of development of inflammation

- 1.Alteration:**
1. Damage of the connective tissue cells (periodontium) with aetiologic factors.
 2. Degranulation of labrocytes, leukocytes, etc.
 3. Release in tissues BAS (serotonin, histamine) mediators of immunoinflammatory reactions.
- 2.Exudation:**
4. Reaction of MCB of pulp with infringement of rheological properties of blood.
 5. Increase of vascular permeability.
 6. Emigration of blood cells, constituent of plasma
 7. Phagocytosis, pinocytosis.
 8. Formation of serous, then purulent exudation, fusion of the periodontium tissues.
- 3.Proliferation:**
9. Reproduction of the connective tissue cells
 10. Differentiation and transformation of cells.

Now it is proved, that the chronic inflammation and the subsequent destruction of a bone tissue around the root apex is not stimulated with bacteria, but with their antigens, including components of a cellular membrane - lipopolysaccharide (endotoxin).

Bacterial toxins directly stimulate production of transcriptional factors which predetermine differentiation of osteoclasts (huge multinuclear cells, which are derivatives of haemopoietic founder cell) capable to resorb bone due to wide arsenal of lysosomal enzymes.

One activated osteoclast is capable to destroy about 200 000 mm³ of bone tissue in a day. Such quantity of bone can be produced with 7-10 generations of osteoblasts which average life lasts about 15-20 days!

The principles of classification

1) by location:

- apex;
- marginal (localized paradontium);
- diffusive.

2) by current (streaming):

- acute;
- chronic;
- exacerbation of chronic.

3) by clinicomorphologic forms;

4) radiological;

5) by aetiology.

Classification of periodontitis by I.G.Lukomsky

1. Acute forms:

- 1) acute serous apex periodontitis;
- 2) acute purulent apex periodontitis;

2. Chronic forms:

- 1) chronic fibrous periodontitis;
- 2) chronic granulating periodontitis;
- 3) chronic granulomatosis periodontitis.

3. Exacerbation of chronic forms of periodontitis.

Classification of periodontitis by M.I.Groshikov

1. Acute periodontitis:

- 1) Phase of periodontium intoxication;
- 2) Phase of expressed exudation process (serous, purulent).

2. Chronic periodontitis:

- 1) Fibrous;
- 2) Granulating;
- 3) Granulomatosis;
- 4) Cyst of root.

3. Exacerbation of chronic forms of periodontitis.

Classification by S.A.Vajndruh

Phases or stages of process	Forms of periodontitis	
	A. Proliferative	B. Purulent
1. Active (progressive) 2.Stabilized (local) 3.Reparative (renewaling)	1A.Granulating periodontitis; 2A. Granuloma 3A. Fibrotisation of process	1B. Abscessing periodontitis 2B. Local purulent 3B. Purulent periodontitis in regenerative phase

Pathomorphology of acute serous Pt

The picture inflammatory hyperemia is observed:

1. Overblooded vessels;
2. Perivascular cellular infiltration with moderate quantity of leukocytes, histiocytes, labrocytes;
3. Hemorrhages in tissue;
4. Tissue edema;
5. Producing of serous exudation.

Changes in bone are not marked usually. If the reasons of acute serous periodontitis are eliminated it calms down, also it is able full restoration of periodontium structure.

Clinic of acute serous periodontitis

This form of inflammation can arise at medicamentous, traumatic and infectious periodontitis. This stage of infectious periodontitis lasts during short-term.

COMPLAINTS: on protracted, constant, not sharp expressed pain (aching pain). It is localized in field of causal tooth, amplifies at biting on a tooth. There is a feeling of "extended tooth". The pain does not irradiate, therefore the patient accurately specifies the causal tooth.

ANAMNESIS: pain disturbs about 1-2 days, the days before tooth was probably treated (arsenic pasta was imposed, root canals were processed or filling up, there was a trauma and other).

OBJECTIVELY: Examination: the causal tooth can be changed in colour, depriving of natural shine with a deep carious cavity or filling. Probing – c/c is connected with pulp chamber, probing is painless. In a pulp cavity disintegration of a pulp or the rests of filling material is observed.

Percussion - painful. The gum in the field of a causal tooth is more often without inflammatory changes or weakly hyperemic, slightly odematic. Palpation is more often painless, less often - weakly painful.

Thermal diagnostic – painless. EOD: more than 100 mkA.

X-ray test does not show any changes in periapecal tissues.

Pathomorphology of acute purulent Pt

It occurs:

1. Increase of tissue infiltration with polymorph nuclear leucocytes;
2. Fusion of tissue and abscess formation.

The nearest sites of a periodontium, jaw bone, gum, periosteum, soft tissues of cheek, regional lymphonodules are in condition of reactive perifocal inflammation. If help will not be rendered to the patient at this stage – to create outflow of exudation – this process complicates with periostitis (if exudation exits under periosteum), osteomyelitis (if exudate distributes on body of jaw with necrotic affection of osteocytes of bone marrow).

Clinic of acute purulent periodontitis

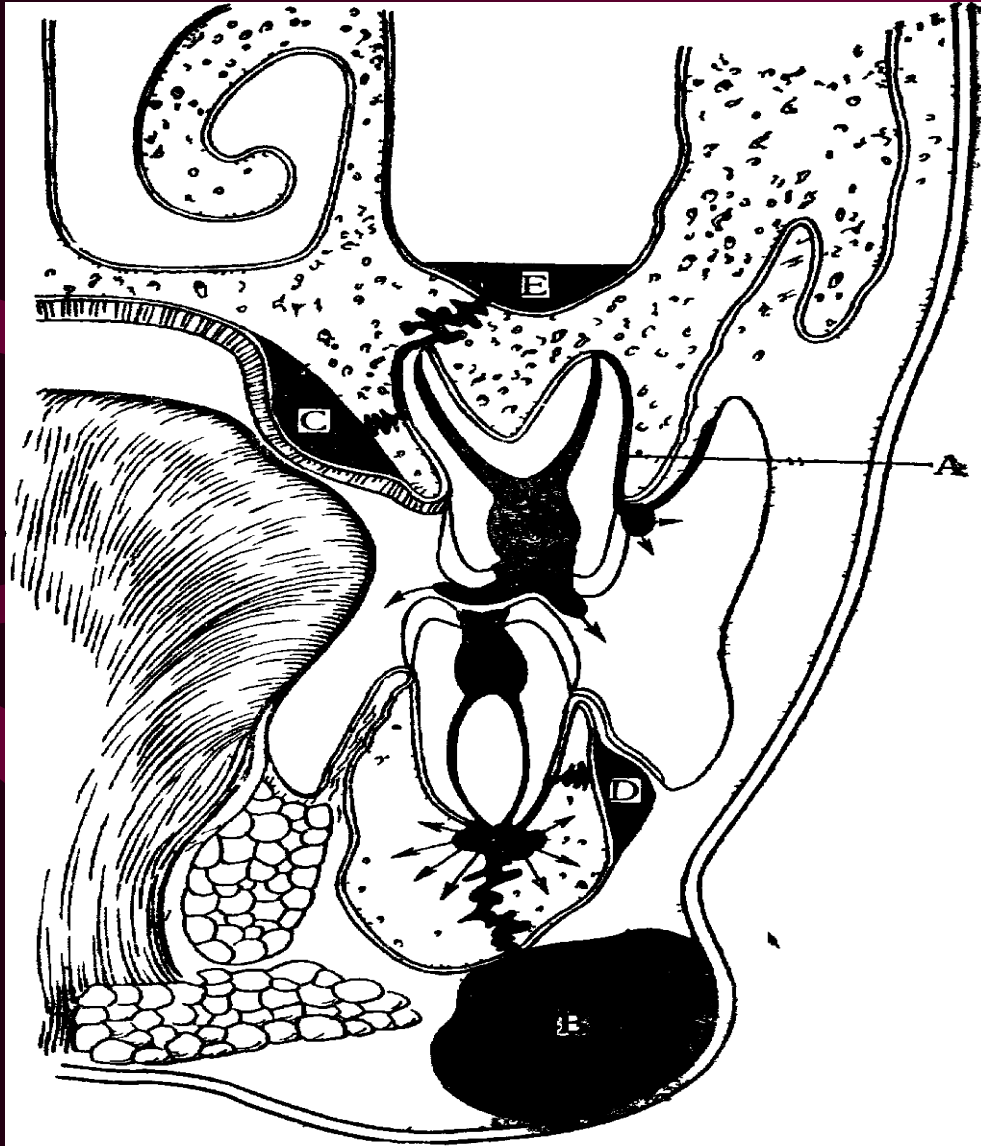
This form of an inflammation can develop as continuation of acute purulent pulpitis without intermediate stage of serous periodontitis.

COMPLAINTS: on a spontaneous sharp constant, intensive, pulsing pain, with irradiation on corresponding branches of a trigeminal nerve. But the patient always defines sick tooth as there is feeling of "an extended" tooth. The least touching to a tooth, even with tongue, strengthens a pain, therefore the patient holds a mouth the half-opened. The general condition of patient is suffered: head ache, sleep disturbance, difficulty or impossibility of eating. **ANAMNESIS:** pain disturbs about 3-5 days.

OBJECTIVELY: Examination: the causal tooth can be changed in colour, depriving of natural shine with a deep carious cavity or filling. Mobility of causal tooth is marked. Probing – c/c is connected with pulp chamber, probing is painless. In a pulp cavity disintegration of a pulp or the rests of filling material is observed. Vertical and horizontal percussion – sharply painful. The gum in the field of projection of root apex is hyperemic, edematic. Palpation of a mucosa in the field of apex projection of tooth root is painful. Regional lymph nodes are increased, painful. Thermal diagnostic – painless. EOD: more then 100 mkA.

X-ray test does not show any changes in periapical tissues or insignificant widening of periodontal space in area of root apex.

The scheme of distribution of exudation from periodontium of maxilla and mandible molars (by Kutteer).



A – marginal way (through odontogingival pocket);

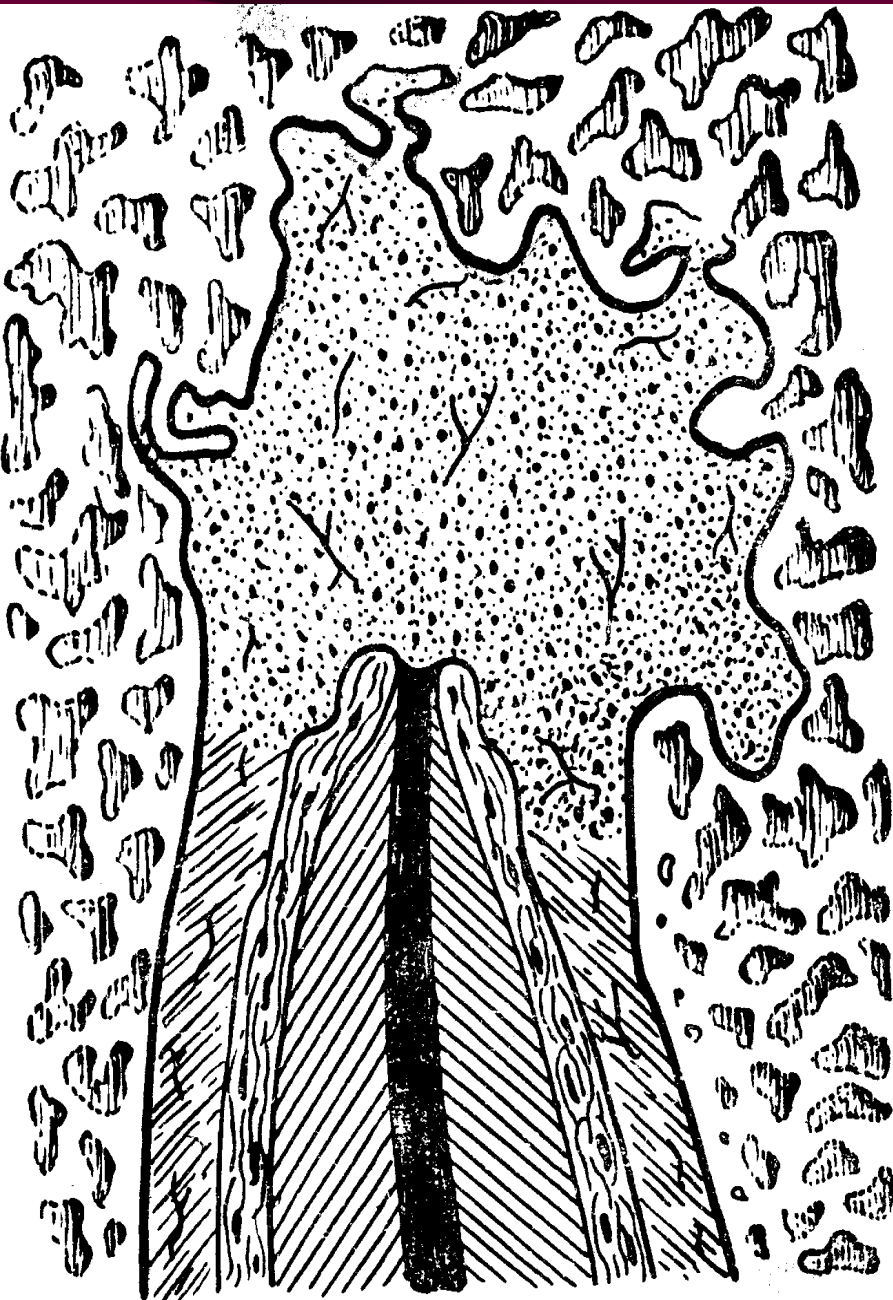
B – in soft tissues of jaw-facial area;

C, D – under periosteum;

E – in additional sinus (antritis);

The most favorable way—through the root canal and pulp chamber

Chronic granulating Pt



1. Intensive osteoclastic resorption of bone tissue, which is not separated from healthy bone;
2. Resorption of cement, and sometimes of root dentin;
3. Formation of granulation tissue – young connective tissue which contains a considerable quantity of capillaries and cellular elements, small number of nerves;
4. Formation of fistulas due to germination of granulations in marrowy spaces of a jaw and perforation cortical layer of bone, periost, and can go out on mucosa membrane of vestibule of oral cavity. At exacerbation of this process serous and purulent exudation can be discharged through it.

Clinic of chronic granulating Pt

It is an actively progressing process in a periodontium and surrounding bone.

COMPLAINTS: Unpleasant pain (aching pain) in tooth, sense of gravity, awkwardness, especially at biting on tooth, presence of fistula on a gum, destroyed tooth, change of tooth colour.

ANAMNESIS: tooth has a long history of disease: earlier different kinds of pain could appear (spontaneous, nightly, paroxysmal). Tooth could be treated in case of caries or its complications. Pain has changeable character, is accompanied with periodic exacerbation of process and appearance of a fistula.

OBJECTIVELY: Examination: the causal tooth is changed in colour, depriving of natural shine, deep carious cavity or filling is observed. Often this tooth is not used by the patient during chewing, so it was coated with dental deposit.

Probing – c/c is widely connected with pulp chamber, probing is painless because of full disintegration of pulp. In a pulp cavity there are disintegration of a pulp or the rests of filling material. The gum in the field of causal tooth with signs of a chronic inflammation: obviously weakly edema and hyperemia or hidden symptoms become perceptible:

- ✓ **Symptom by A. Marmasse** (hidden hyperemia): with index finger rubbing 5-10 times on vestibule surface of alveolar process in field of causal and 2 neighboring teeth is performed.

In a result near damaged tooth arises expressed stable hyperemia.

- ✓ **Symptom by Crane** (hidden edema): after pressure of ball-shaped instrument in area of apex root projection of damaged tooth appears depression (recess), which lasts for a long time.
- ✓ **Symptom by I.G.Lukomsky** (vessels paresis): it is the same as symptom Crane, but we estimate colour and expression of depression. At first depression has pale colour, then changes on cyanotic and remains for a long time.

Clinic of chronic granulating Pt

In the field of projection of root apex the fistulous course with the protruded (expanded) granulations is often observed. It is also possible to see cicatrices after fistulas.

Vertical percussion – weakly morbid or painless.

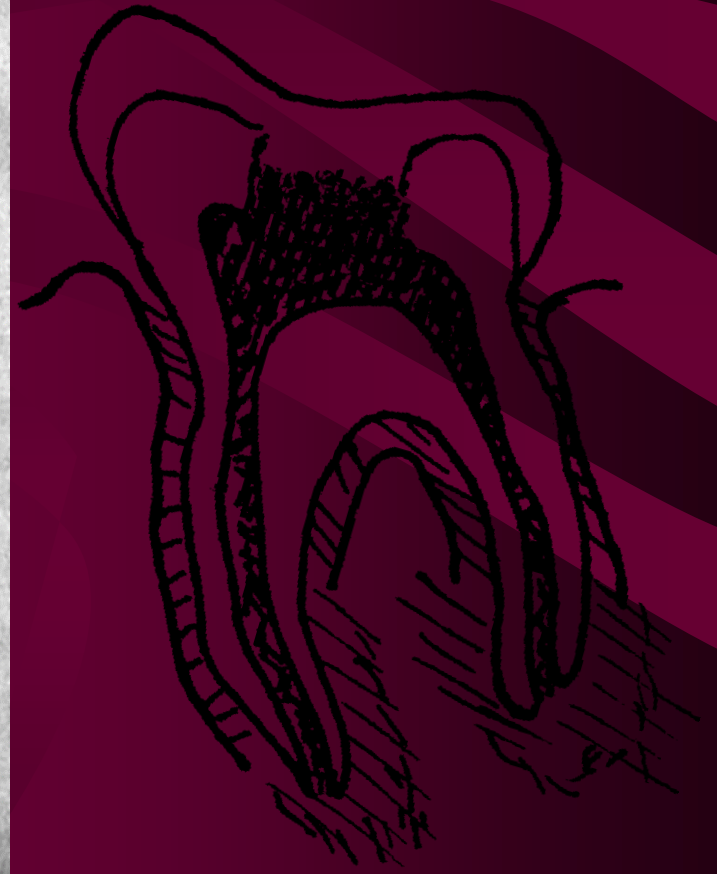
Palpation of a mucosa in the field of apex projection of tooth root - weakly painful. Regional lymph nodes are increased, painful.

Thermal diagnostic – painless.

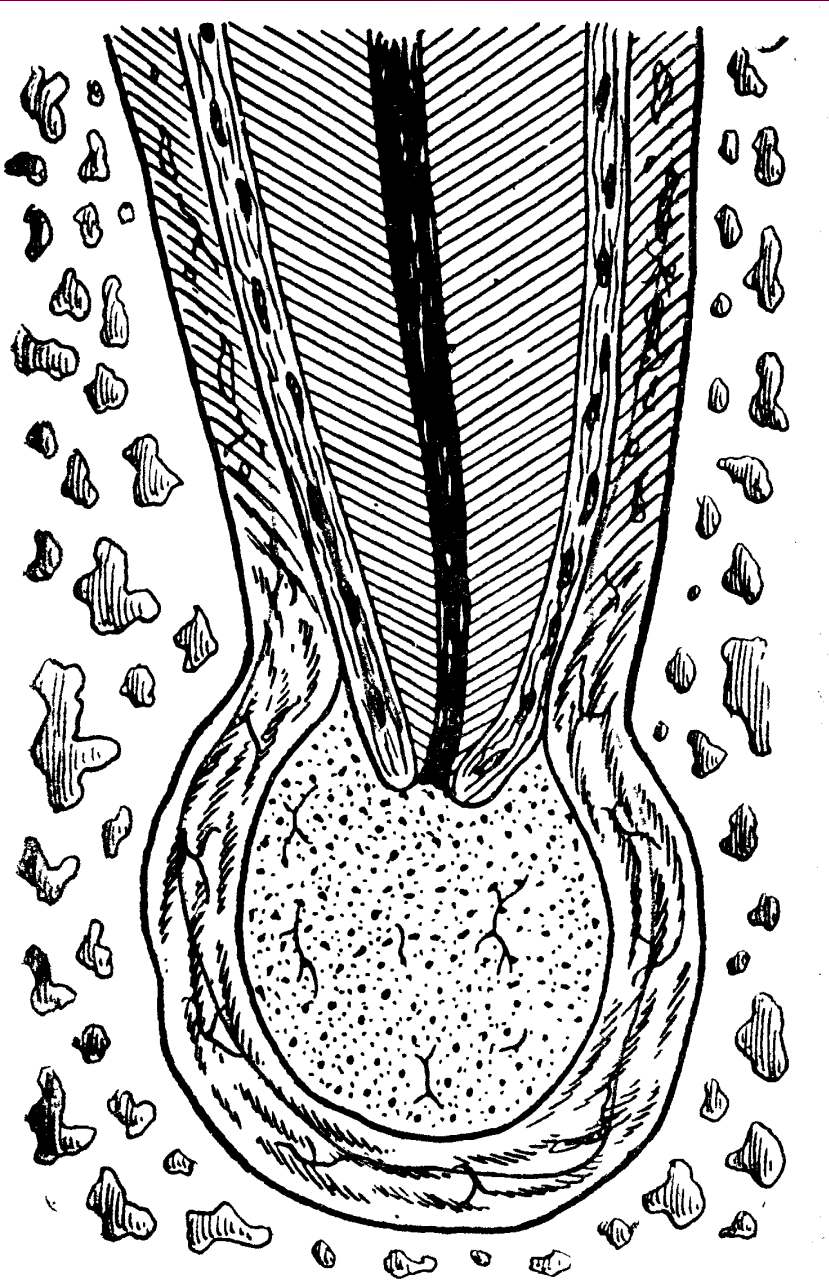
EOD: more than 100 mkA.

X-ray test:

destruction of a periodontal space, resorption or destruction of bone without accurate borders in the form of "tongues of flame". Sometimes resorption of cement and dentine of tooth root apices.



Chronic granulomatosis Pt



Destruction of periodontium and bone tissue which are substituted with granulation tissue, however it is limited from surrounding bone by peripheral fibrous capsule.

By Marmasse:

- Infection zone;
- Destruction zone;
- Inflammation zone;
- Stimulation zone.

Kinds of granulomas

Abricosov distinguishes following kinds of granulomas:

1. Simple;
2. Complex (epithelial);
3. Cystiform (cystogranuloma, root cyst).

Macroscopically simple granuloma is a small knot in size from pea to cherry (to 0,5 cm), surrounded with the connecting capsule and its fibres are intertwined in periodontium. Therefore during of tooth extraction granuloma is taken out together with root.

Microscopically simple granuloma consists of usual granulating tissue limited on periphery with fibrous capsule.

Epithelial granuloma consists of the same cells, but it is penetrated with strands of multilayered flat epithelium.

There are 2 points of view about genesis of epithelium in complex granulomas:

1. In periodontium there are small groups of epithelial cells which remained from enamel body and situated like islands – Malasse's islands (in 1885 he has described them at first). In the time of pathological process epithelial cells expand, participating in formation of complex granulomas.
2. Epithelium grows into the tissue of granuloma from mucosa through odontogingival pocket or fistulas. (Lukomsky).

Cystogranuloma develops from complex granuloma, by way of formation of cavity with epithelial covering.

Its sizes are about 0,5 – 0,8 cm

It occurs:

- 1. Vacuolar fatty dystrophy of the central areas of epithelial strands;**
- 2. Decay of epithelial cells;**
- 3. Formation of small cleft-formed cavities which are merging and forming big cavity. The cavity contains proteinous exsudate + cholesterol crystals (because of xanthomous cells decay, which give opalescent shade). This fact is used for cyst diagnostics.**

Radicular cyst – similar formation with size about 1 cm and more.

Clinic of chronic granulomatosis Pt

COMPLAINTS: Very often proceeds asymptotically.

Sometimes there are complaints to unpleasant pain (aching) in tooth, especially at biting on tooth.

ANAMNESIS: tooth has a long history of disease: earlier different kinds of pain could appear (spontaneous, night, paroxysmal). Tooth could be treated in case of caries or its complications. Sometimes at catarrhal diseases or at active chewing arise aching pain, sense of a strain, gravity.

OBJECTIVELY: Examination: the causal tooth is changed in colour, depriving of natural shine, deep carious cavity or filling is observed.

Probing – c/c is widely connected with pulp chamber. In a pulp cavity - disintegration of a pulp or the rests of filling material. Probing is painless. The gum in the field of a causal tooth is without changes or with the phenomena of a stagnant hyperemia.

Vertical percussion – painless or weakly morbid. At horizontal percussion – positive symptom by Shmreker (apex tremors or symptom of the reflected impact) is observed.

Palpation of mucosa in the field of apex projection of tooth root – painless or weakly painful. It is possible to feel a protrusion of an external side of bone because of a reactive thickening of periost.

Regional lymph nodes are increased, painful.

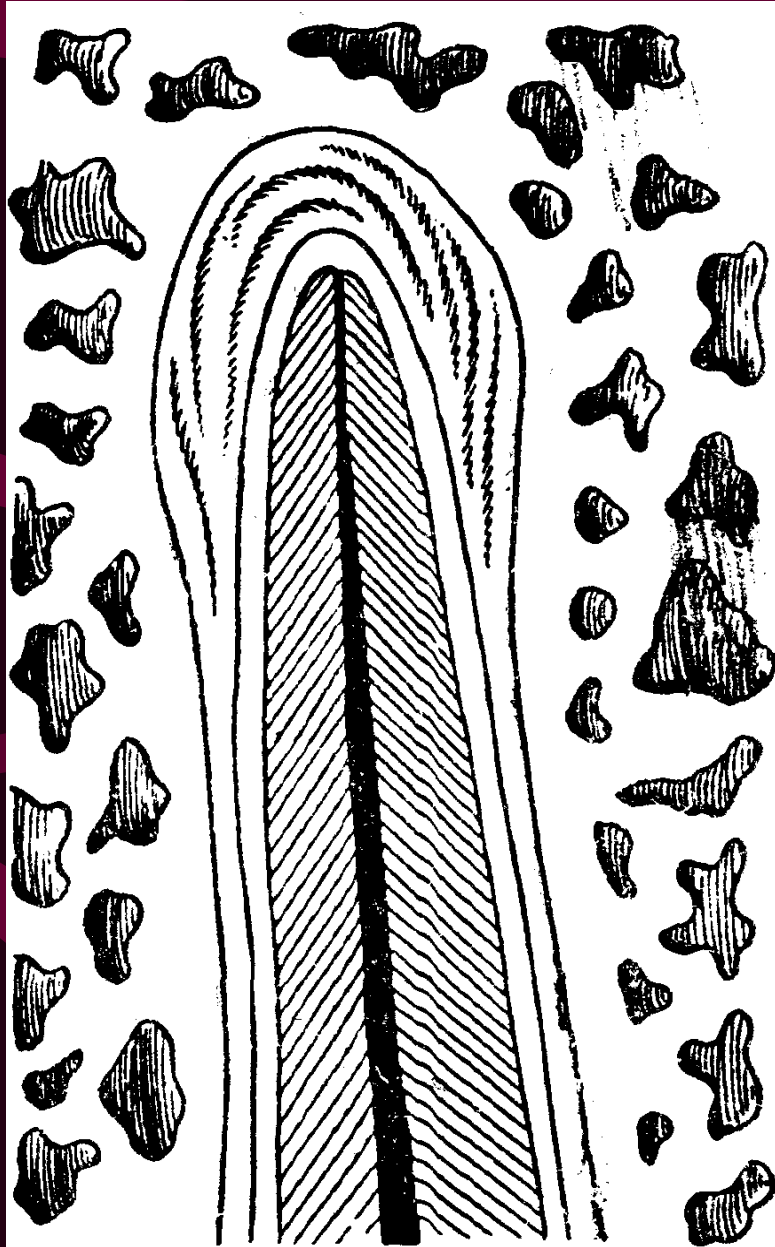
Thermal diagnostic – painless. EOD: more than 100 mkA.

X-ray test:

destruction of a periodontal space, osteoporosis or destruction of bone with accurate contours, oval or spherical shape with dimension about 0,5 sm.



Chronic fibrous Pt



Periodont transforms into coarse-fibrous connective tissue like to cicatrices. There is decrease of cell elements, inordinate growth of fibrous strands. Between its fascicles are located a small focuses of infiltrations. After treating of granuloma and granulating periodontitis regeneration of the bone tissue in alveolar process of jaw with construction of bone beams towards a periodontium is marked. It is occurred new growth of root cement due to activity of cementocytes.

Clinic of chronic fibrous Pt

It is the optimum form of chronic inflammation by current.

COMPLAINTS: it proceeds asymptotically. The patient has complaints on damage of a filling, fracture of a crown or a discoloration of tooth.

ANAMNESIS: tooth has a long history of disease: earlier different kinds of pain could appear (spontaneous, night, paroxysmal). Tooth could be treated in case of caries or its complications.

OBJECTIVELY EXAMINATION:

Survey – the causal tooth is changed in colour, depriving of natural shine, deep carious cavity or filling is observed.

Probing – c/c is widely connected with pulp chamber. In a pulp cavity - disintegration of a pulp or the rests of filling material. Probing is painless. The gum in the field of a causal tooth is without changes.

Vertical and horizontal percussion – painless.

Palpation of a mucosa in the field of apex projection of tooth root – painless.

Regional lymph nodes are not increased, painless.

Thermal diagnostic – painless. EOD: more then 100 mkA.

X-ray test:

deformation of a periodontal space (dilating or waist), is possible a thickening of apical part of root in cause of hypercementosis (like drumstick).



Exacerbation of chronic forms of apical periodontitis

More often chronic granulomatosis and granulating Pt can exacerbate. Reasons of an exacerbation could be general and local.

Local factors: additional infection of the root canal (in the case of the open tooth cavity), the closing of the perforation and root canal with hard food (broken ratio of anaerobic and aerobic microorganisms), trauma, tooth overloading.

General factors: reduction of immunological reactivity of organism (after hypothermia, previous catarrhal diseases).

Clinic of exacerbation of Pt chronic forms

COMPLAINTS: sharp spontaneous constant intensive pulsing pain, which is irradiated on corresponding branch of a trigeminal nerve, feeling of "an extended" tooth, presence the fistula on a gum, destroyed tooth, change of tooth color, facial asymmetry due to collateral edema. The least touching to the tooth, even with tongue, strengthens a pain, therefore the patient holds a mouth the half-opened. The general condition of patient is suffered: head ache, sleep disturbance, difficulty or impossibility of eating.

ANAMNESIS: acute pain disturbs about 3-5 days. Presence of changeable pain, is accompanied with periodic exacerbation of process and appearance of a fistula.

OBJECTIVELY: Examination: the face is asymmetrical due to collateral edema (without hyperemia, painfull palpation, temperature increasing, infiltration). Regional lymph nodes are increased, painful. Causal tooth can be changed in colour, depriving of natural shine with a deep carious cavity or filling. Mobility of causal and neighboring teeth is marked. Probing – c/c is connected with pulp chamber, probing is painless. In a pulp cavity disintegration of a pulp or the rests of filling material is observed. Vertical and horizontal percussion – sharply painful. The gum in the field of a causal tooth has inflammatory changes: hyperemic, edematic, can be observed fistula or cicatrices. It is possible to feel compaction of mucosa membrane of transitional fold or a protrusion of an external side of bone because of a reactive thickening of periost, sometimes the defect bone wall. Palpation of a mucosa in the field of apex projection of tooth root is painful.

Thermal diagnostic – painless. EOD: more then 100 mkA. X-ray test: there is changes in periapical tissues correspond to chronic form of inflammation.



THANK'S FOR ATTENTION!