

POLTAVA STATE MEDICAL UNIVERSITY
THERAPEUTIC STOMATOLOGY PROPAEDEUTICS CHAIR

PULPIT

(First part)

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

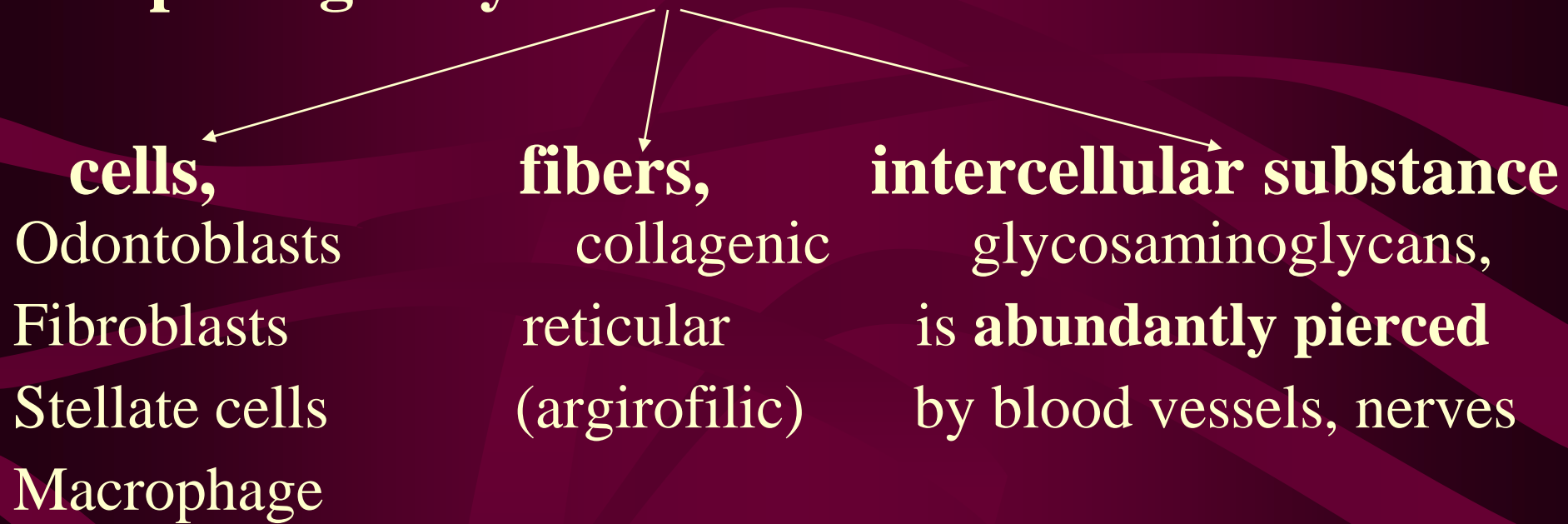
**The lecturer: PhD in Medical Sciences,
Associate Professor
Marchenko Iryna Yaroslavovna**

PLAN OF LECTURE

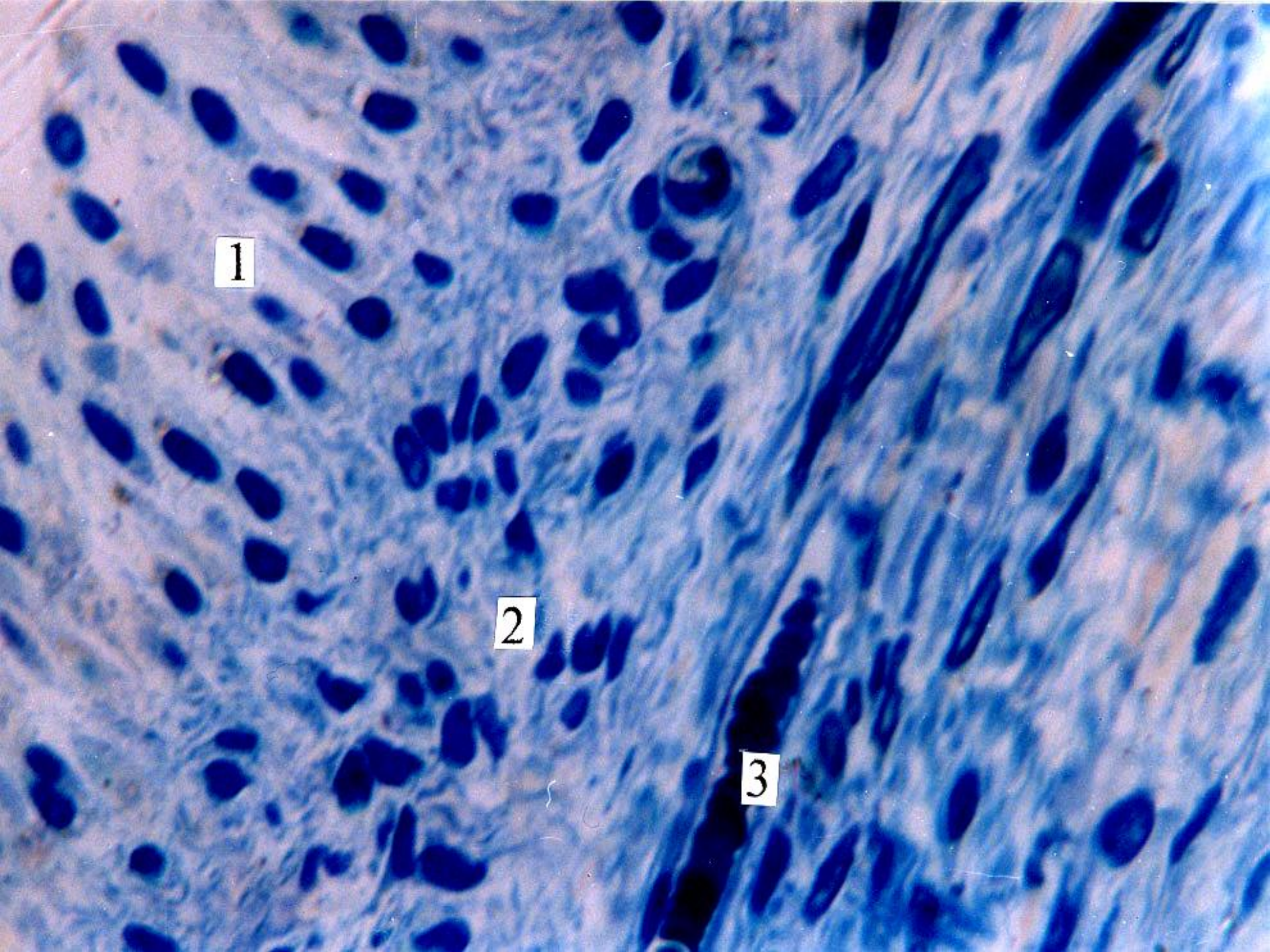
- Definition of concept.
- Structure of pulp: anatomic, hystological, functional features.
- Etiology of pulpitis. Ways of penetration of an infection into a pulp.
- Pathogeny of pulpitis.
- Classification of pulpitis.
- General symptomatology of acute pulpits.
- Clinic and pathomorphology of acute forms of pulpitis.

1. A pulpitis is inflammation of tooth pulp.
2. Pulp (pulpa dentis) – connective tissue formation, filling of a tooth cavity (pulp chamber).

Morphologically consists of:



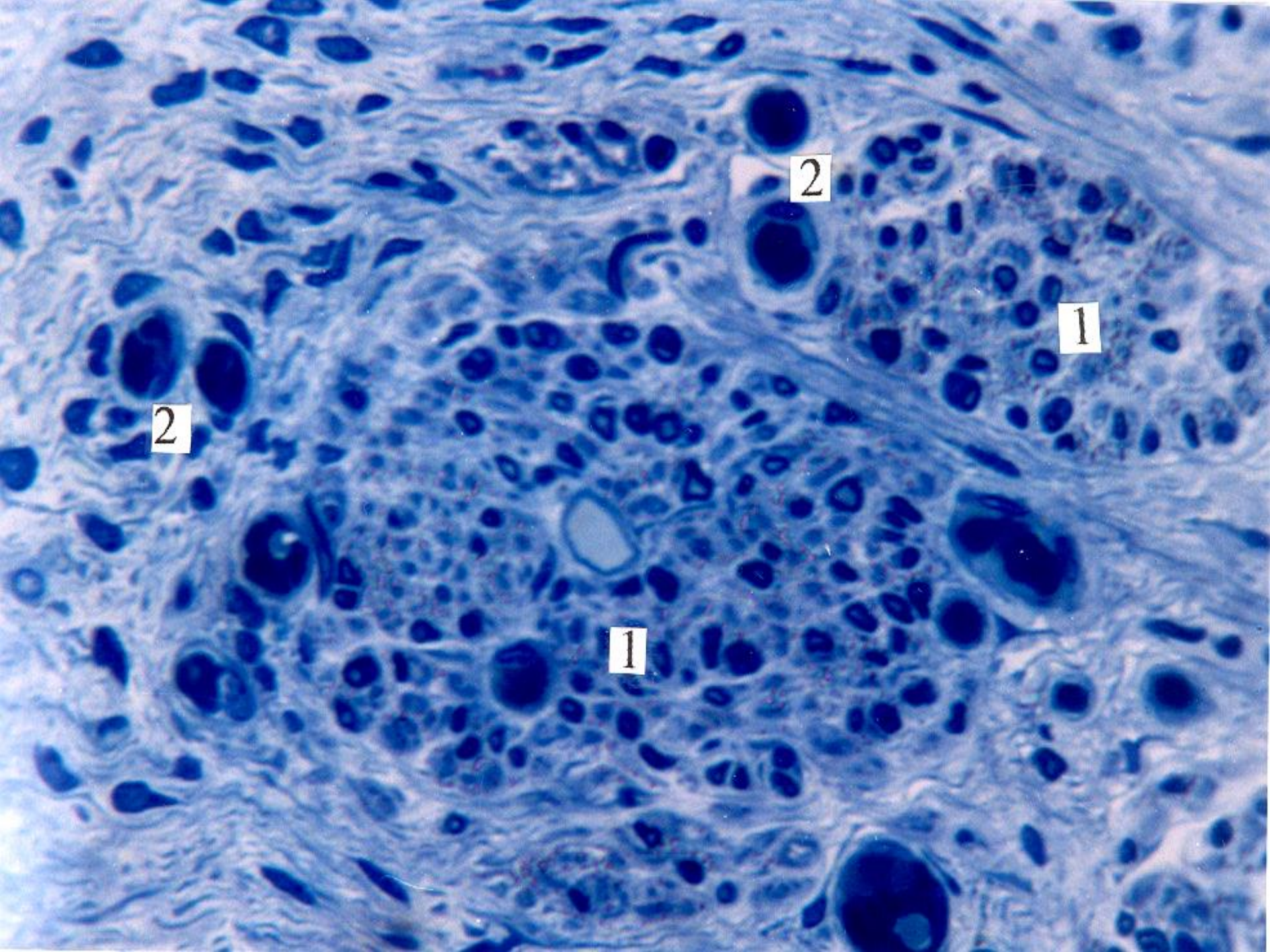
3 layers: 1. Odontoblastic;
2. Subodontoblastic;
3. Central.

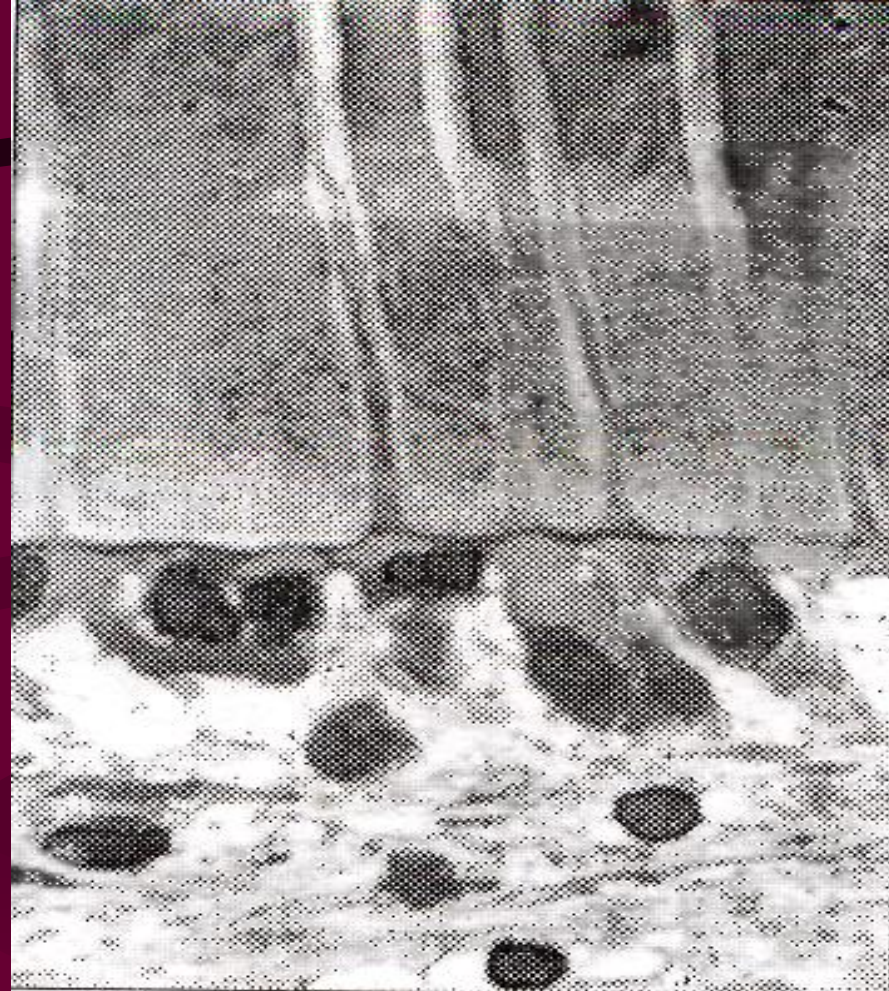
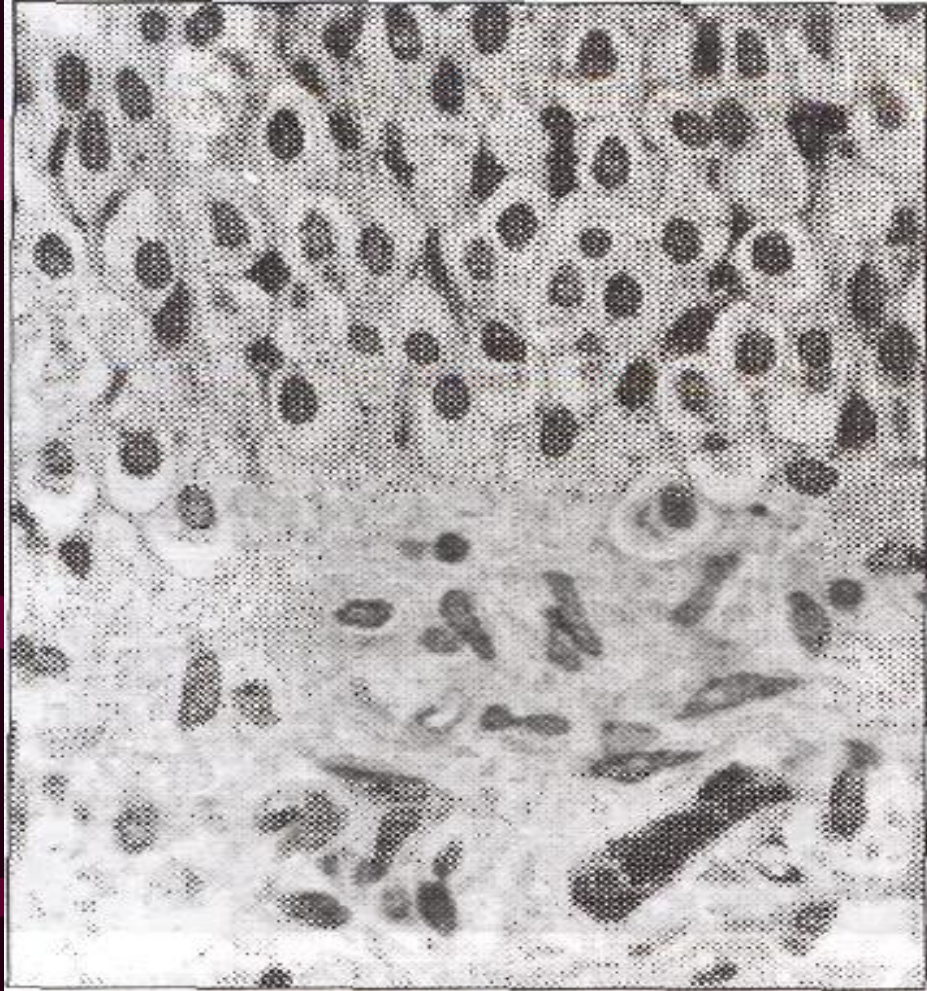


1

2

3





Features of pulp inflammation: it takes place in closed (secluded) box, which is formed by intractable dentine, therefore inflammation proceeds quickly, accompanied the rapid squeezing of pulp, leads to disturbance of pulp trophicity and its death

3. By etiology

pulpitis

distinguish :

infectional

non-infectional

traumatic

chemical

thermic

Microflora:
associations
streptococci,
lactobacterias,
Gram+ bacilli,
fusospirochetes
and their toxins

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

Perforation
and trauma
of pulp
during
preparation

Toxic
action of
alcohol,
ether,
eugenol,
orthophos-
phoric acids,
monomers
from filling
materials

Overheating
of pulp
during
preparation
of carious
cavities
or under
artificial
crown

Ways of penetration of infection into a pulp

- Through carious cavity;
- Retrograde:
 - a) through gingival pocket;
 - b) from near-by infectious center;
 - c) hematogenic;
 - d) lymphogenic.

4. Pathogeny – a mechanism of inflammation development

- 1. Alteration :**
1. Damage of connecting tissue cells (pulp) by etiologic factors.
 2. Degranulation of leucocytes, labrocytes;
 3. Elimination of BAS in tissues – mediators of inflammation - serotonin, histamine.
- 2. Exsudation:**
4. Reaction of pulp MCB with disturbance of rheologic properties of blood.
 5. Increase of vascular permeability.
 6. Emigration of blood cells, components of plasma
 7. Phagocytosis, pinocytosis .
 8. Formation of serous, then purulent exudates infiltration
- 3. Proliferation:**
9. Reproduction of connecting tissue cells.
 10. Differentiation and transformation of cells.

5. Classification of pulpitis by E.M. Gofung

1. Acute pulpitis (*acuta*)

- a) partial (*partialis*);
- b) general (*diffusa communis totalis*);
- c) purulent (*purulenta*).

2. Chronic pulpitis (*chronica*)

- a) fibrous (*simplex*);
- b) hypertrophic (*hypertrophica*);
- c) gangrenous (*gangraenosa*).

Classification of pulpitis

by **E.S.Yavorskaya, L.I.Urbanovich (Kiev, 1964)**

I. Acute pulpitis (p. acuta).

1. Hyperemia of pulp (hyperaemia).
2. Traumatic (traumatica):
 - a) by chance bare pulp;
 - b) at the break of crown or root.
3. Partial (partialis).
4. Diffuse (totalis, communis).
5. Purulent (purulenta).

II. Chronic pulpitis (p. chronica).

1. Fibrous (fibrosa).
2. Hypertrophic (hypertrophica).
3. Gangrenous (gangraenosa).
4. Concrementous (concrementosa).

III. Exacerbation of chronic pulpitis (chronica exacerbatio).

IV. Pulpitis, complicated with periodontitis.

Works classification of pulpitis

I. Acute pulpitis (p. acuta).

1. Traumatic (traumatica):
 - a) at preparing (without or with denudation of pulp);
 - b) at the break of crown or root.
2. Hyperemia of pulp (hyperaemia).
3. Partial (partialis).
4. General (communis).
5. Purulent (purulenta).

II. Chronic pulpitis (p. chronica).

1. Fibrous (simplex).
2. Hypertrophic (hypertrophica).
3. Gangrenous (gangraenosa).
4. Concrementous (concrementosa).
5. Root (radicis dentis).

III. Exacerbation of chronic pulpitis (chronica exacerbatio).

IV. Necrosis and gangrene of pulp (gangraena et necrosis pulpa).

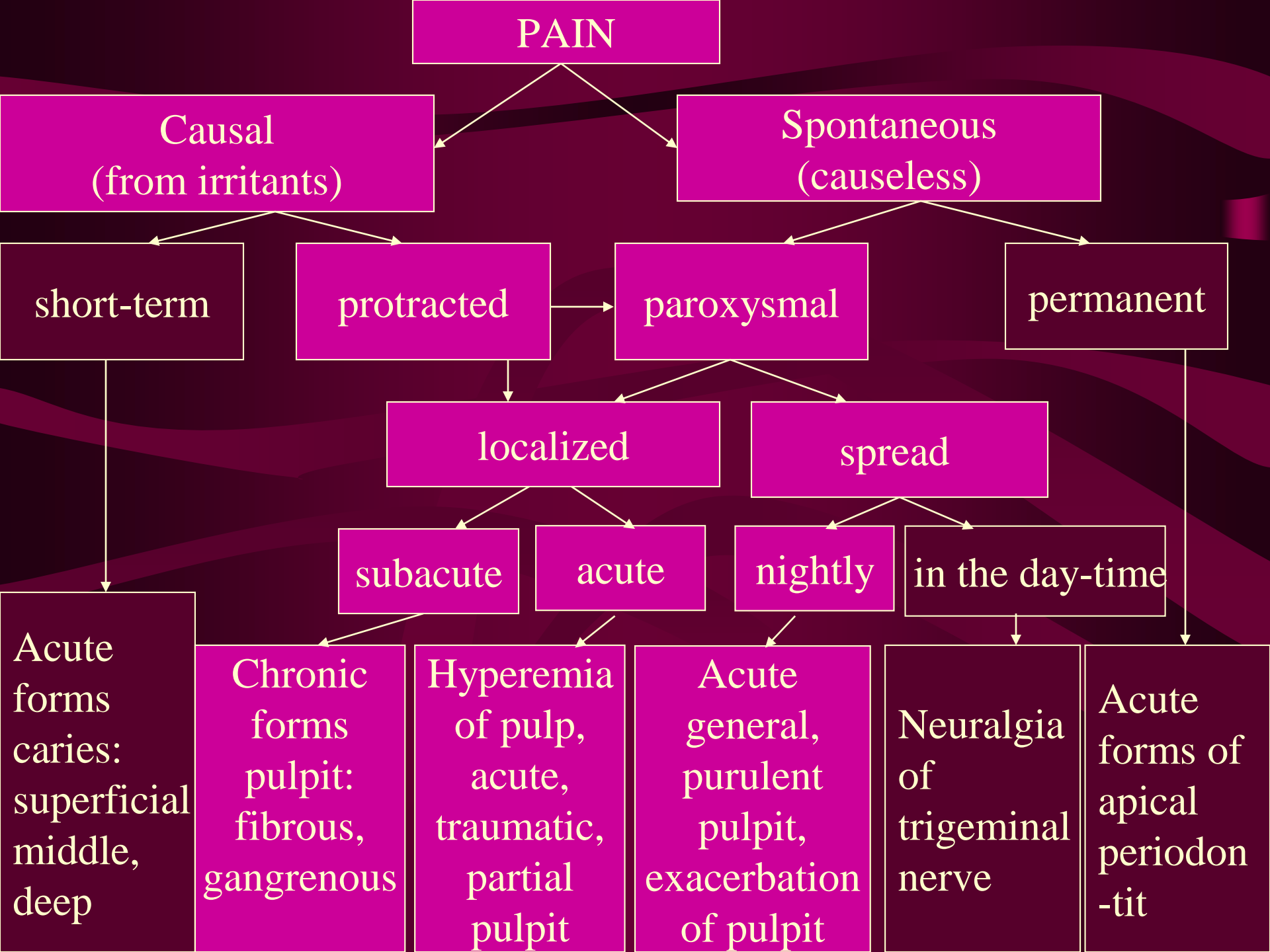
V. Atrophy of pulp (atrophia pulpa).

ICD-10 classification

- K04 Diseases of the pulp and periapical tissues
- K04.0 Pulpitis
- K04.00 Initial (hyperemia)
- K04.01 Acute
- K04.02 Purulent (pulpal abscess)
- K04.03 Chronic
- K04.04 Chronic ulcerative
- K04.05 Chronic hyperplastic (pulp polyp)
- K04.08 Other specified pulpitis
- K04.09 Pulpitis, unspecified
- K04.1 Pulp necrosis Pulp gangrene
- K04.2 Denticle pulp degeneration, pulp petrification
- K04.3 Incorrect formation of hard tissues in the pulp. Secondary or irregular dentin
- K04.9 Other and unspecified diseases of the pulp and periapical tissues

American Association of Endodontist Terminology for Pulpitis Diagnosis

- Healthy pulp
- Reversible pulpitis
- Symptomatic irreversible pulpitis
- Asymptomatic irreversible pulpitis
- Pulp necrosis
- Previously treated
- Previously initiated (started) treatment
(amputation, incomplete extirpation)



ACUTE TRAUMATIC PULPIT

COMPLAINTS: acute pain, arising up during preparation of carious cavity. In the case of trauma of tooth - spontaneous pain and pain from all irritants, bleeding.

ANAMNESIS: household, sporting, works trauma

OBJECTIVELY EXAMINATION: at survey - on the bottom of caries cavity there is connection with pulp chamber, drop of blood; or break of part (or all) crown, denudation or transmission of red pulp.

Probing - acutely painful.

Percussion - painless. In the case of trauma and injury or dislocation of tooth - painful, sensitive.

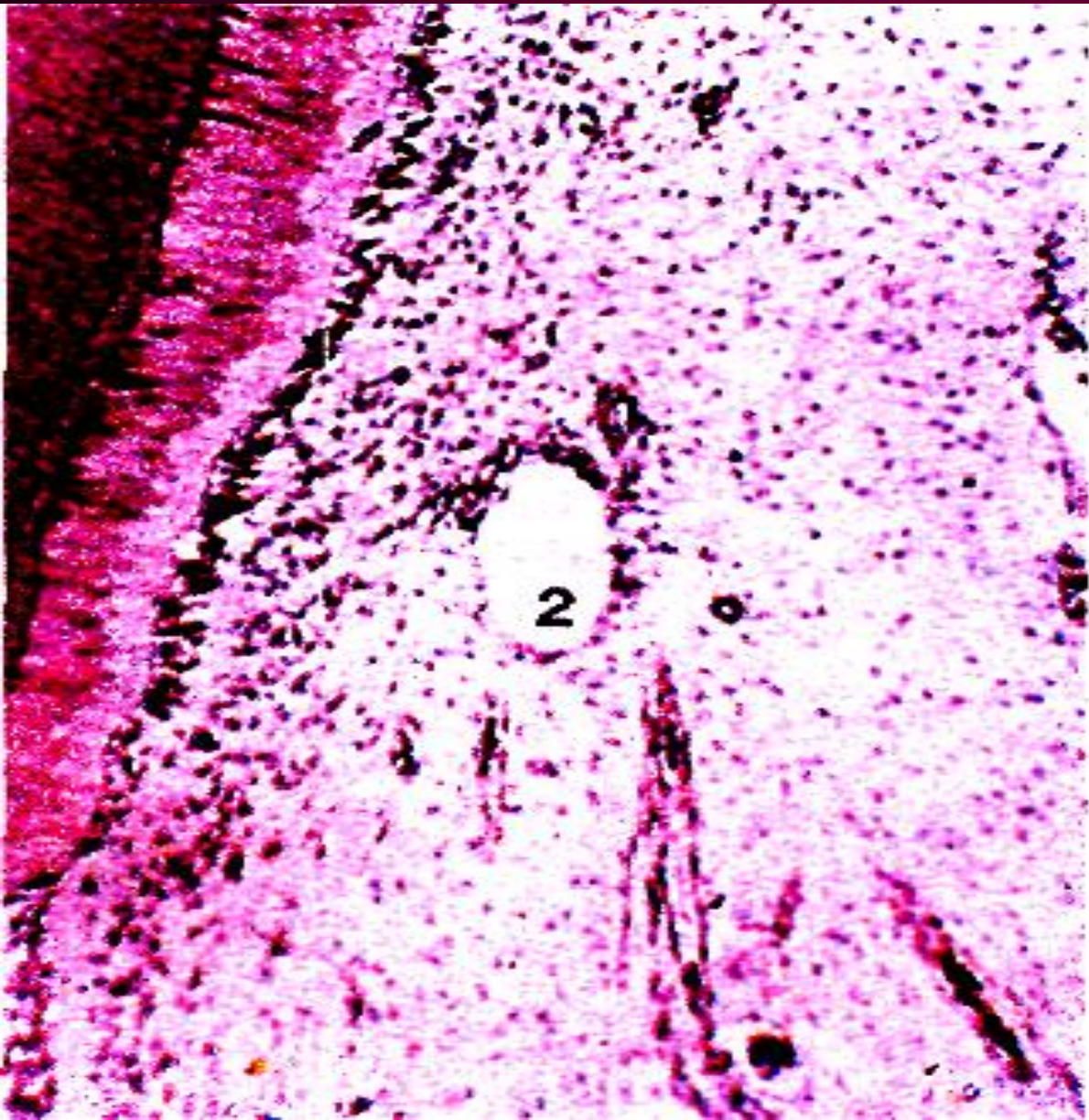
Palpation - painless.

Thermal diagnostic - painful, pain continues after

Acute traumatic pulpitis



PATHOMORPHOLOGY OF HYPEREMIA OF PULP



- Plethora of pulp blood vessels (full-blooded), hyperemia of pulp tissue;
- Stretched vessel with diapedesis of blood corpuscle (single leucocytes)
- Diminishing of number of odontoblast layers, their dystrophy.

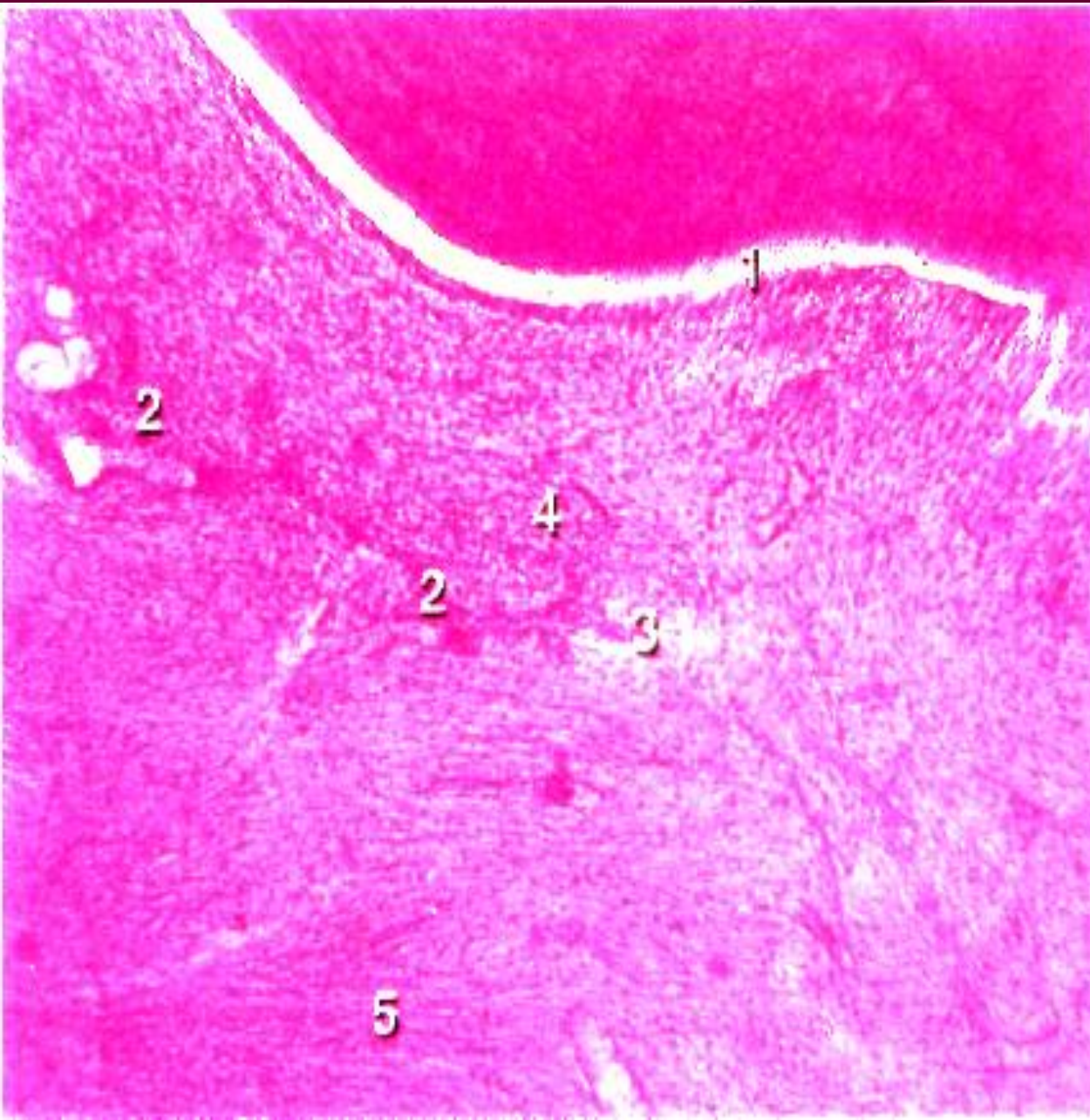
CLINIC OF PULP HYPEREMIA

COMPLAINTS: acute, spontaneous pain during 1-2 min. (lightning, fulminant), intermissions 6-12 hours, pain from all types of irritants, proceeds 1-2 min. after their removal.

ANAMNESIS: episodes of pain are disturbed about 1 day until this pain arose up only from irritants, was short-time.

OBJECTIVELY EXAMINATION: at survey- a deep carious cavity or filling. Probing - the dentine of bottom and walls is softened, caries cavities is not connected with pulp chamber, on a bottom is painful. Percussion (vertical and horiz.) - painless. Palpation - painless. Thermal diagnostic - painful, pain continues 1-2 min.

ACUTE PARTIAL PULPIT



- Dystrophy of same odontoblasts;
- Pulp hyperemia and plethora;
- Swollen of a part of pulp;
- Limited leucocytes infiltration.

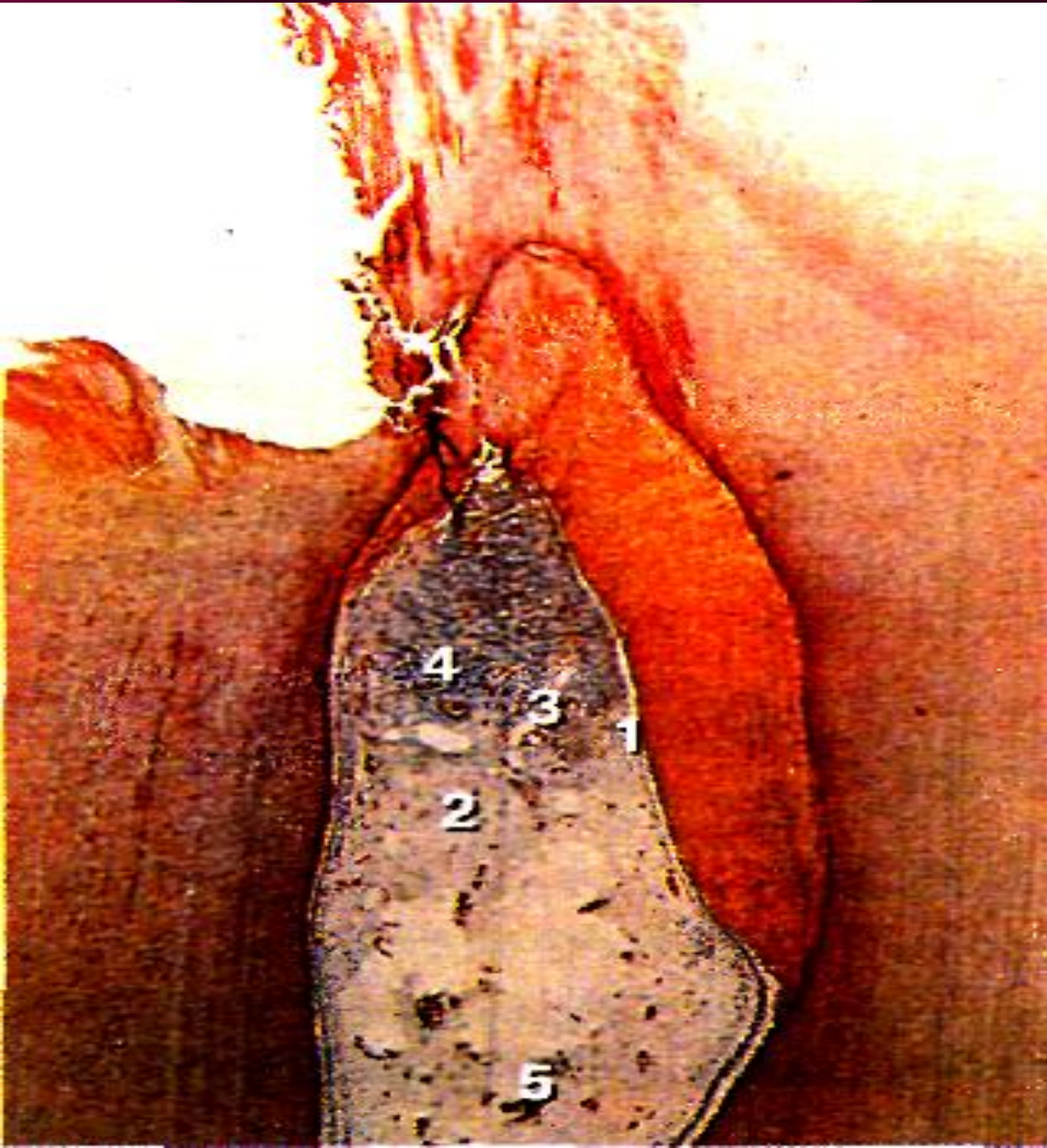
CLINIC OF ACUTE PARTIAL PULPIT

COMPLAINTS: acute, spontaneous, paroxysmal pain during 15-30 min, intermissions about 3-4 hours. Pain is localized. Attacks can arise up from all types of irritants.

ANAMNESIS: episodes of pain are disturbed about 2 days, until this pain arose up only from irritants, was short-time.

OBJECTIVELY EXAMINATION: at survey - a deep carious cavity or filling. Probing - on the bottom of carious cavity acute pain on the area of one horn of pulp. Percussion and palpation - painless. Thermal diagnostic - provokes an episode of pain, protractedly not passing. EOD: 10-15 mkA.

ACUTE GENERAL PULPIT



- Reticular dystrophy of odontoblasts layer adjacent to the replaceable dentine;
- Expansion of vessels;
- Slowed-down of blood stream (sluggish circulation);
- Diapedesis of red corpuscles and formation of small hemorrhages (extravasations)
- Diffuse leucocytes infiltration;
- Edema of tissue.

CLINIC OF ACUTE GENERAL PULPIT

COMPLAINTS: pain acute, spontaneous, irradiating on appropriate branches of trigeminal nerve. Attacks last about 1-2 hours, intermissions about 1 hour. Pain increases at night. Any irritants can provoke pain.

ANAMNESIS: episodes of pain are disturbed about 2-3 days, duration of them grew gradually, and «light intervals» became shorter.

OBJECTIVELY EXAMINATION: at survey - a deep carious cavity or filling. Probing - caries cavities is not connected with pulp chamber, on all bottom is acute pain. Percussion vertical-sensible, horizontal – painless. Palpation - painless. Thermal diagnostic – hardly painful. EOD: 20 - 40 mkA.

ACUTE PURULENT PULPIT



- Hardly extended of pulp vessels;
- Edema of tissue;
- Diffuse leucocytes infiltration;
- Centers of the purulent melting of pulp tissue.

ОДА К ЗУБНОЙ БОЛИ

Роберт Бер

Ты, завладев моей скулой,
Пронзаешь десна мне иглой,
Сверлишь сверлом, пилишь пилой,
Без остановки.
Мечусь истерзанный и злой,
Как в мышеловке.

Так много видим мы забот,
Когда нас лихорадка бьет,
Когда подагра нас грызет
Иль боль в желудке.
И эта боль — предмет острот
И праздной шулки.

Бешшусь я, исходя слюной,
Ломаю стулья как шальной,
когда соседи надо мной
В углу хохочут.
Пускай их бесы бороной
В аду щекочут.

Всегда жила со мной беда —
Неурожай, недуг, нужда,
Позор неправого суда,
Долги, убытки...
Но не терпел я никогда
Подобной пытки!

И я уверен, что в аду,
Куда по высшему суду
Я непременно попаду
(В том нет сомнений!)
Ты будешь первою в ряду
Моих мучений.

О, дух раздора и войны,
Что носит имя сатаны
И был низвергнут с вышины
За своеволие,
Казни врагов моей страны
Зубною болью!

CLINIC OF ACUTE PURULENT PULPIT

COMPLAINTS: acute, spontaneous, specially in a night-time, pulsing, irradiating, tearing, unbearable pain. Pain is almost permanent, a few diminishes from cold.

ANAMNESIS: episodes of pain disturb more than 3 days, became unbearable in the last night.

OBJECTIVELY EXAMINATION: at survey - a deep carious cavity or filling. The tooth can lose natural brilliance.

Probing of the bottom of car/cav can be painless (if a crown pulp is transformed in pus), the perforation of tooth cavity can happen during probing, a drop of pus can appear.

Percussion – sensitive or weakly painful.

Palpation - painless. Thermal diagnostic - pain increases from hot, diminishes from cold. EOD - 50-80 мкА

Result of acute traumatic pulpitis treatment



**Result of
treatment**

**Acute traumatic
pulpitis**





THANK'S FOR ATTENTION!