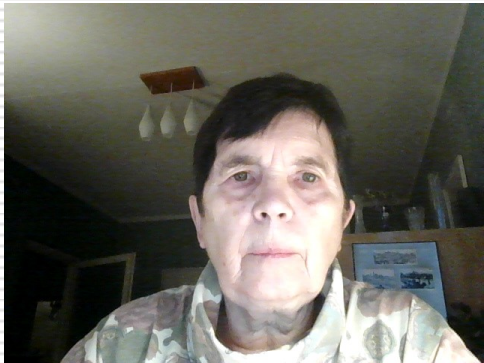
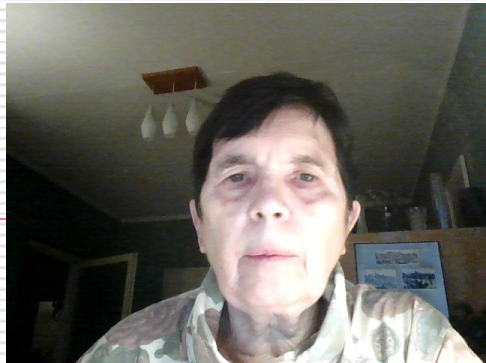
Histology and Physiology of the dental pulp
Inflammation

Prof. MUDr. M. Kukletová

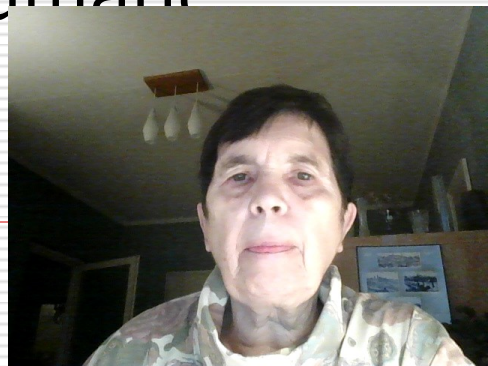


- Nature of inflammatory response
- Inflammation is the reaction of living tissue to any type of injury
- Mobilization and coordination of
- vascular
- neurologic processes
- cellular
- humoral

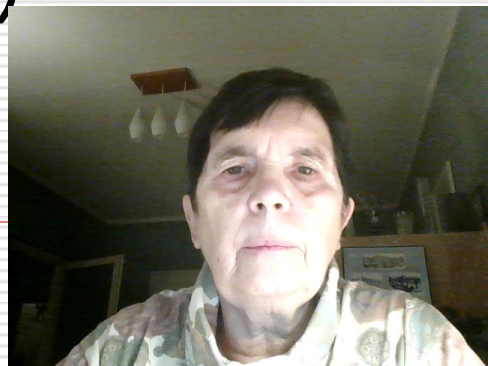


Pathway of entry of microorganisms into the dental pulp

- caries
- trauma
- from surrroundins
- accessory root canals
- retrograde (periodontal pockets)
- anachoresis
- Neurogenic pulpitis -irritation - traumatic occlusion

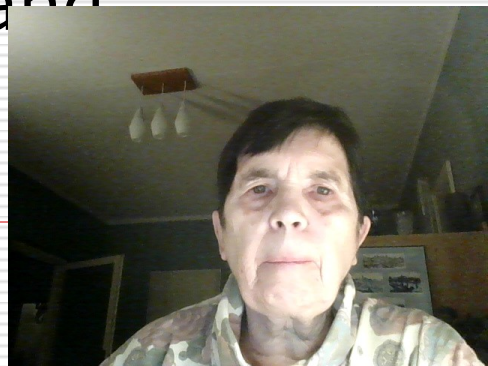


- Objectives - to destroy the irritant
- to neutralize it by dilution
- heat
- redness (rubor)
- swelling (tumor)
- pain (dolor)
- loss of function (functio laesa)
- to set the stage for repair



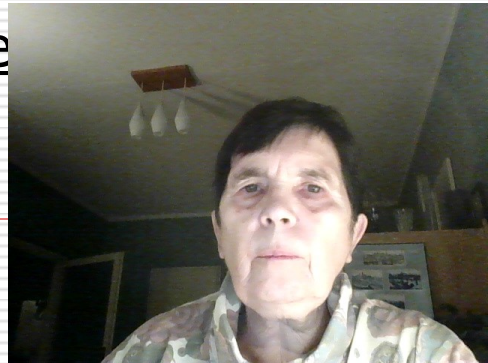
Exudative - acute response

- Initial response of the pulpal tissue to any irritant
- mechanical, chemical, thermal, microbial
- Emergency action - to neutralize the injurious action by:
- an influx of fluid exudate (inflammatory edema) to dilute and detoxify
- white cells (neutrofiles) to ingest and immobilize

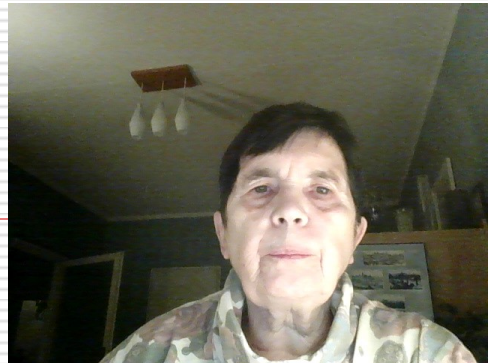


Proliferative (chronic) response

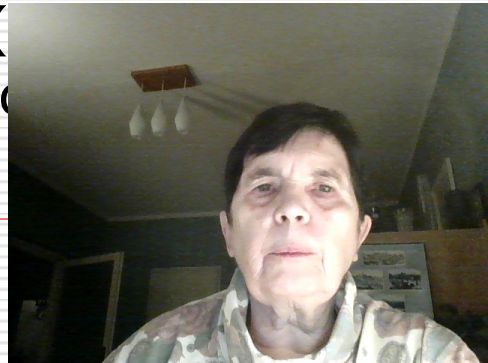
- secondary (delayed) action
- connective tissue forms
- new cells (fibroblasts)
- blood vessels and fibres
- granulation tissue - formed to replace and repair the damaged tissue
- granulomatous tissue = granulation tissue + lymphocytes, plasma cells, macrophages in great quantities
- It is a defense tissue - microorganisms are



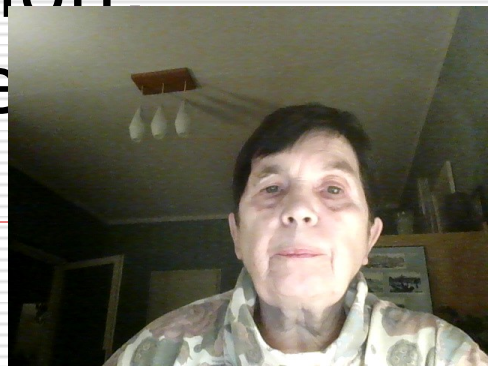
- **Primary factors that initiate the acute inflammation**
- Responsible for inflammation:
- **Neurogenic factor (neural response)**
- **Tissue injury factor (cellular response)**



- **The neurogenic factor** - activation of neural response by environmental irritants (mechanical, chemical, microbial)
- may cause
  - immediate and transient pain perception - result of nerve fibers irritation
  - vasodilatation - persists = increase in capillary permeability, fluid exudation, leucocytes infiltration= beginning of the exudative phase
  - increased intrapulpal pressure = result of the increased blood volume (hyperemia) and tissue exudate
  - secondary (spontaneous) pain response (
- The pain persists - the presence of necroti



- **The tissue injury factor** - is due to release of mediators by the injured odontoblasts
- chemical substances
- the same effect as the nerve factor
- prolonged vasodilatation, fluid exudation, leucocytes infiltration, increase of intrapulpal volume, pressure, pain



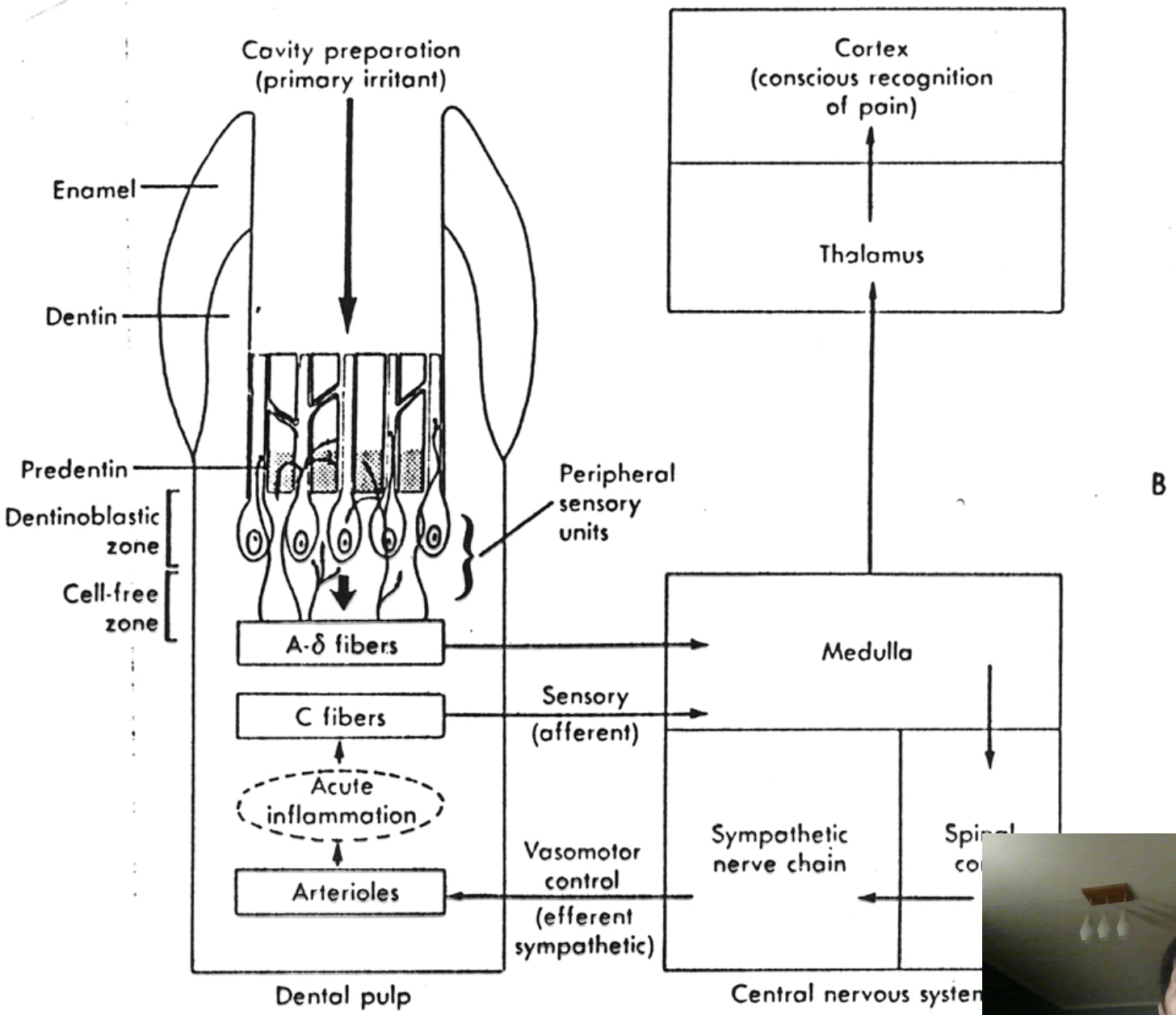


Fig. 3-20, cont'd. **B,** Afferent-efferent pathway for sensory and vasomotor impu
severe dentinal stimulation.

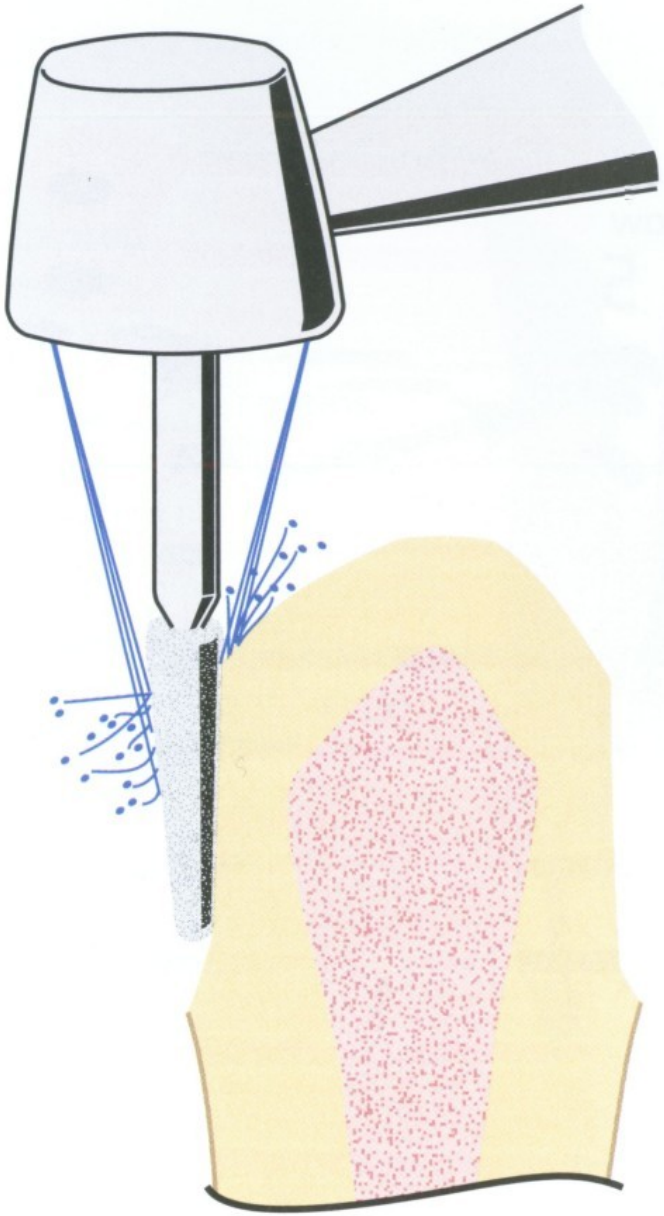
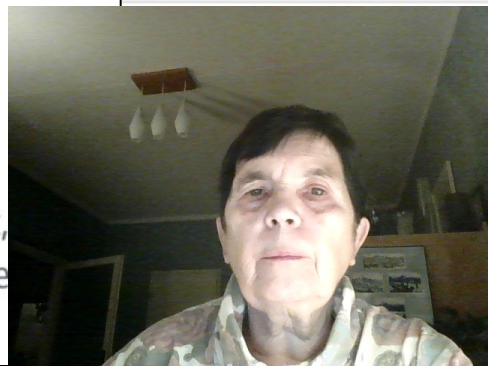


Fig. 3.13 Preparing teeth for restorations generates frictional heat, causes dehydration and tissue damage to the pulp. Such injury is lesse proper water irrigation during the cutting procedure.



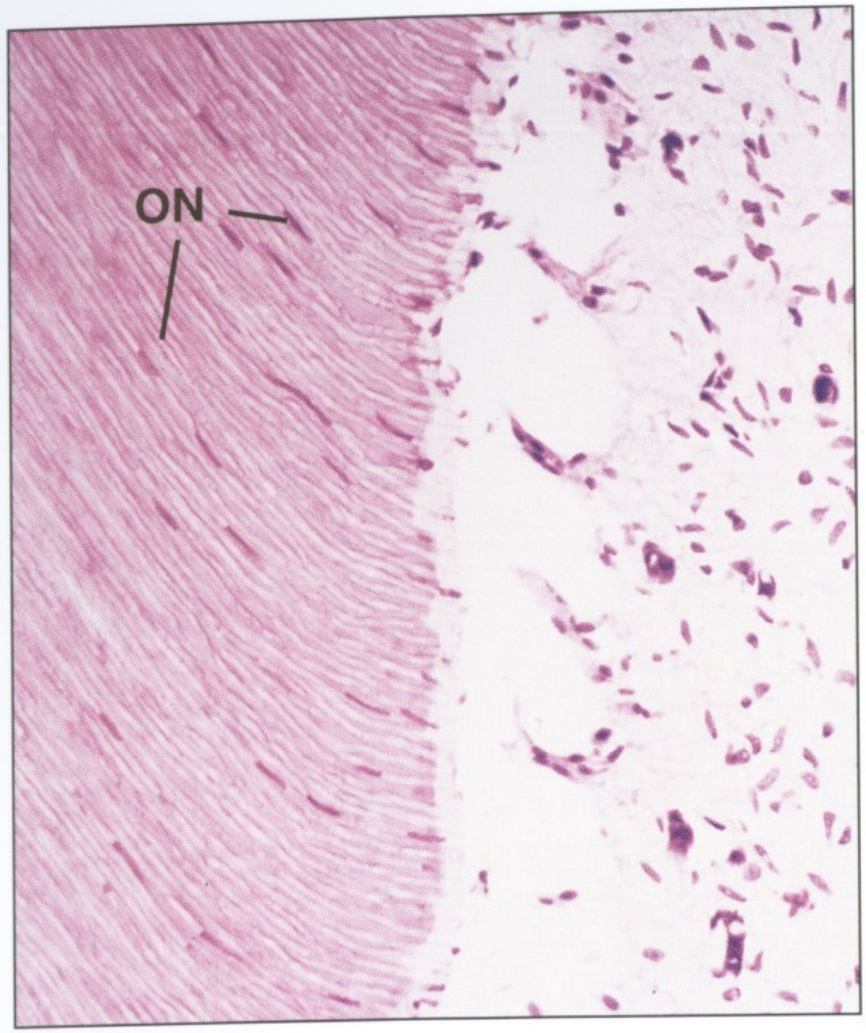
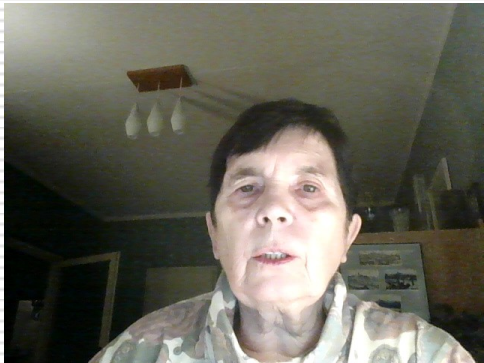
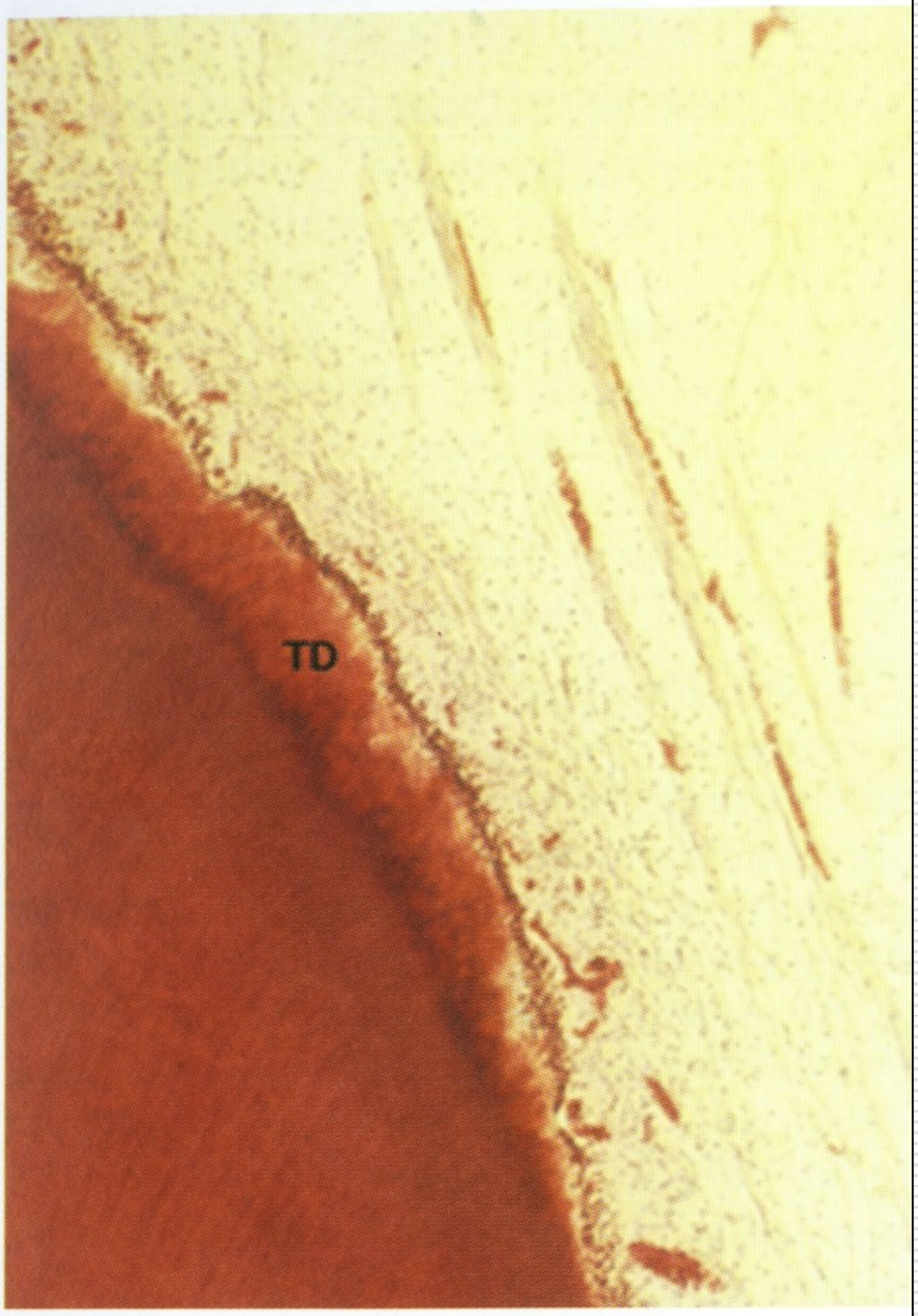
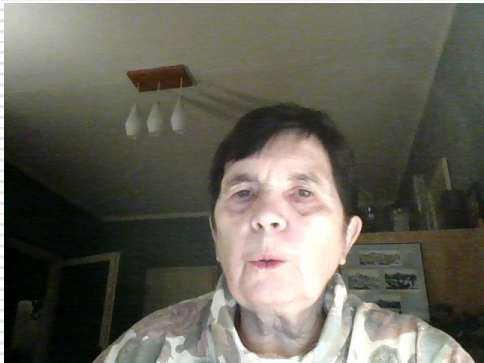


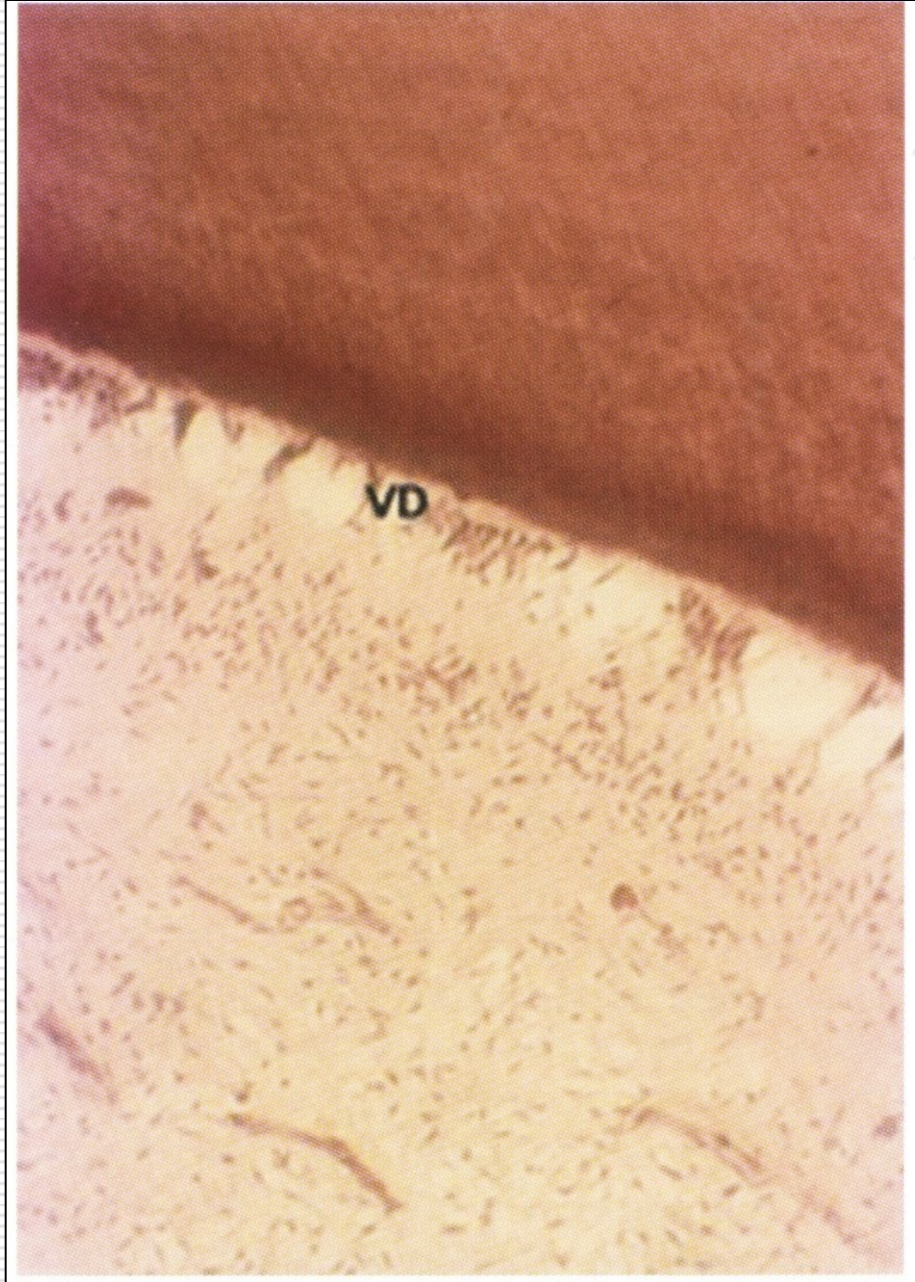
Fig 2-13 Photomicrograph of a demineralized section showing odontoblastic nuclei (ON) displaced into the dentin as a result of cavity preparation with inadequate cooling of the bur. The odontoblastic layer is disrupted and vacuolized. (Hematoxylin-eosin stain; original magnification ×60.)



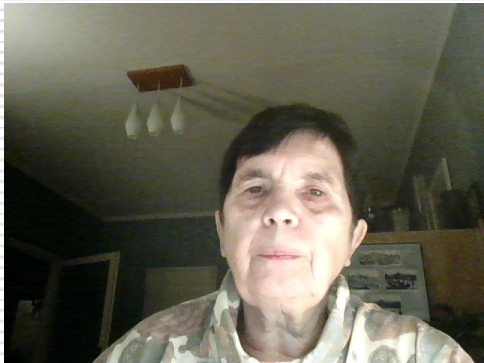


Obr. 2.8 Vrstva terciárního dentinu (TD) vytvořená za tři týdny po preparaci kavity turbinou.





Obr. 2.9 Vakuolární degenerace (VD) odontoblastů nalezená v pulpě za 30 dnů po preparaci kavity turbinou.



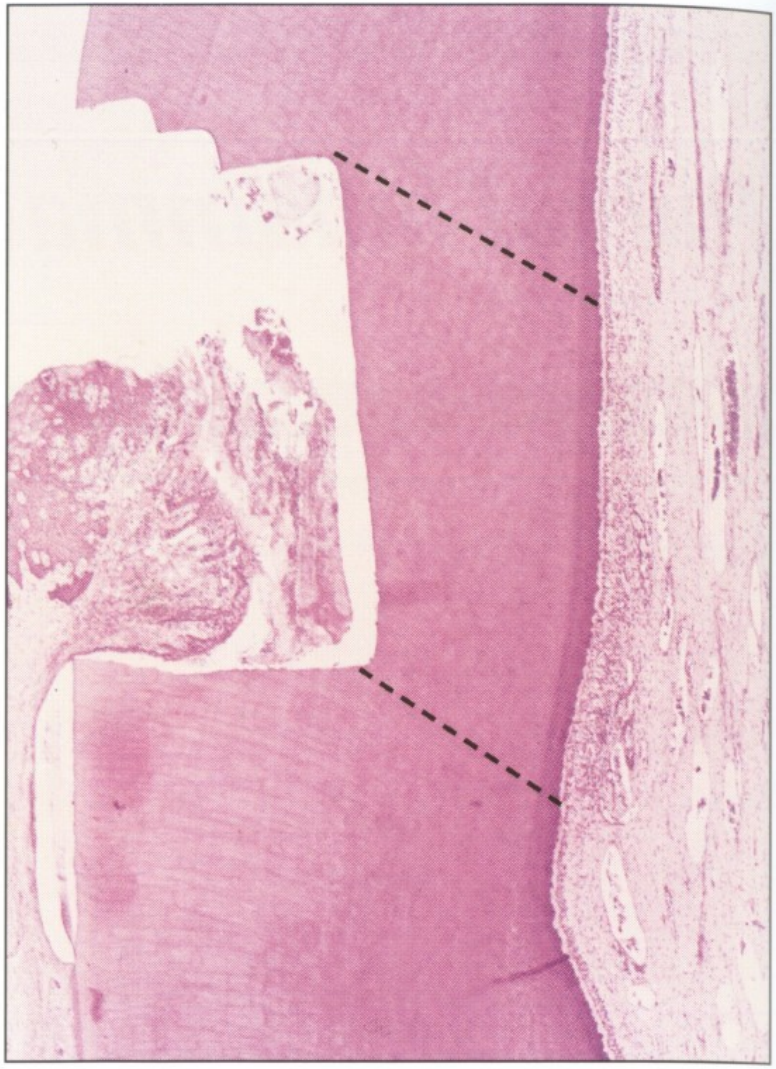
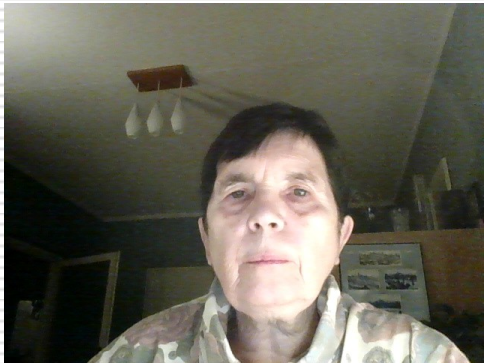


Fig 3-3 Photomicrograph of a moderate pulpal reaction.[6] Many cells have accumulated subjacent to the affected tubules, but they are well delimited to the cavity tubules (between dotted lines). (Hematoxylin-eosin–stained, demineralized section; original magnification ×10.)



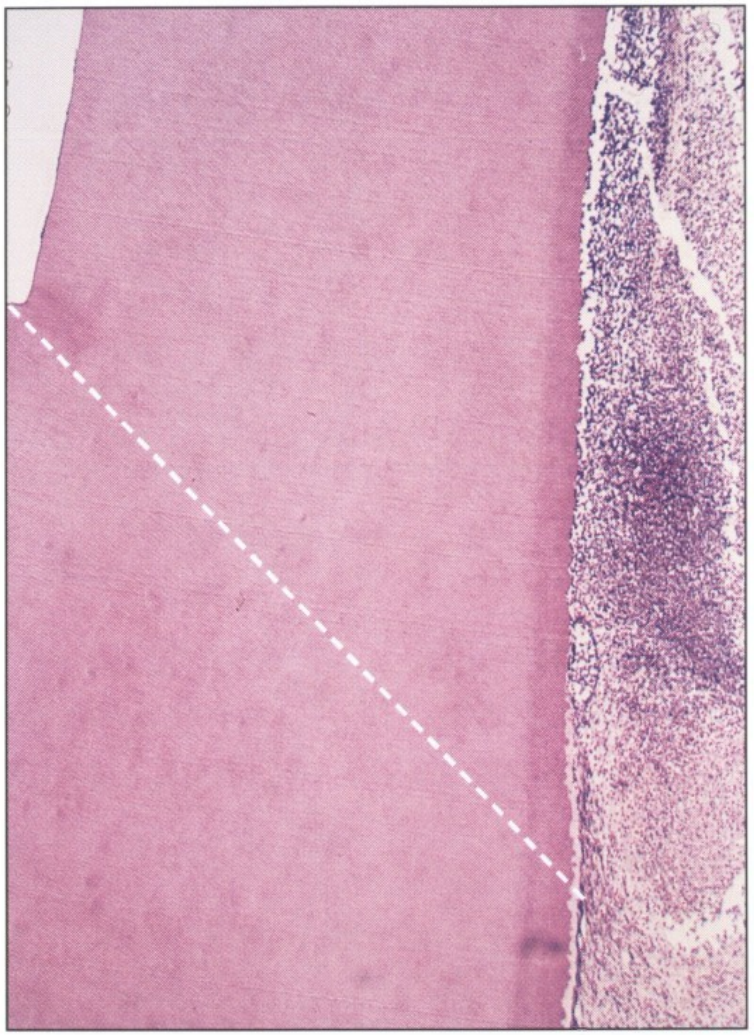
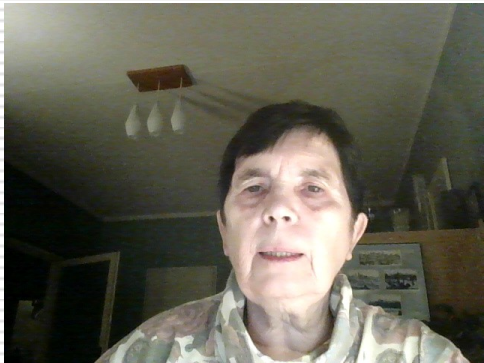


Fig 3-5 Photomicrograph of a severe pulpal reaction.[6] The cellular infiltration is localized to the cavity tubules (above the dotted line), is intense subjacent to the exposed tubules, and corresponds to that of an abscess. (Hematoxylin-eosin–stained, demineralized section; original magnification ×10.)



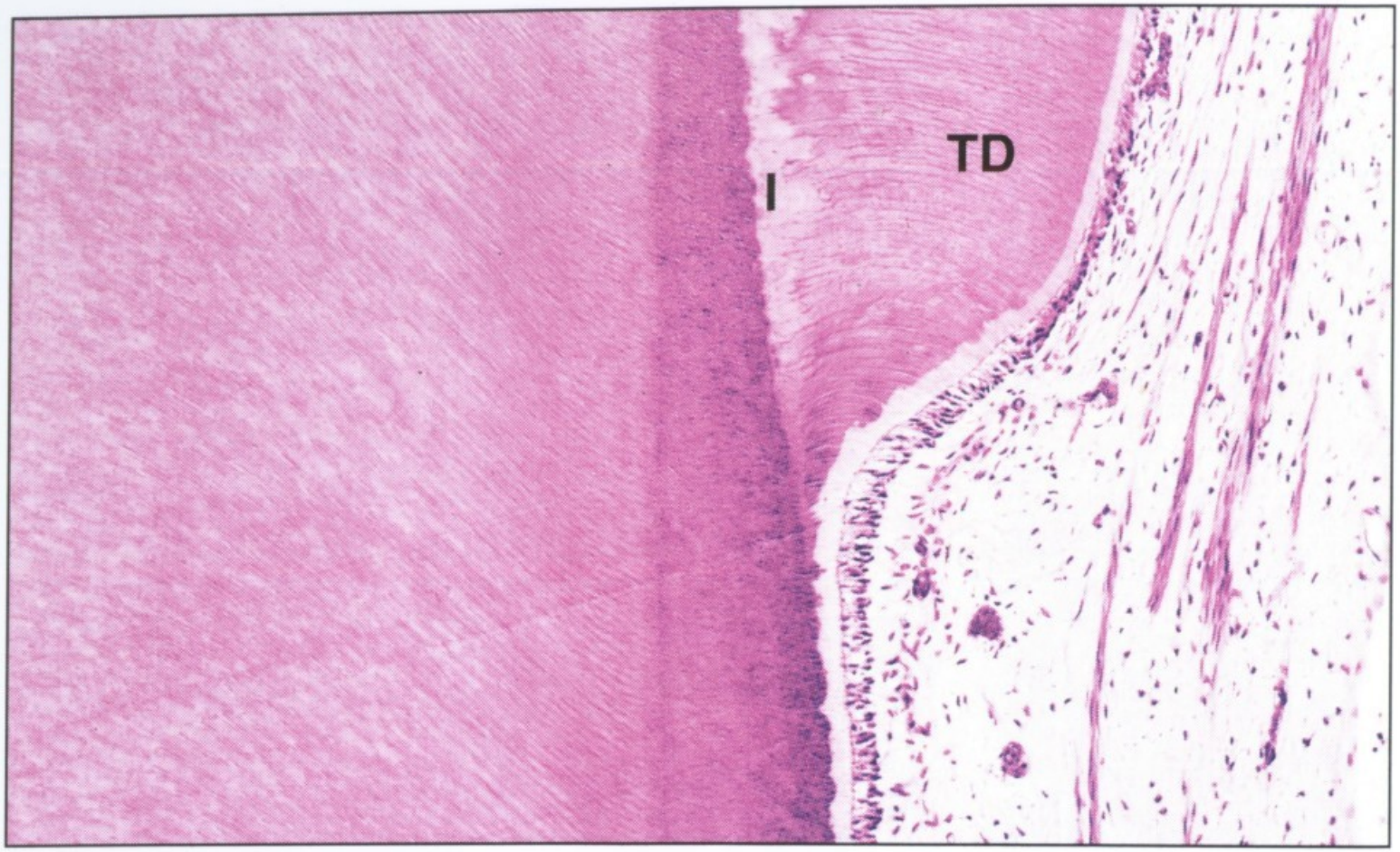
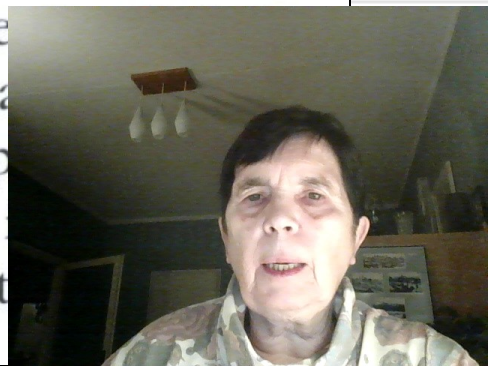
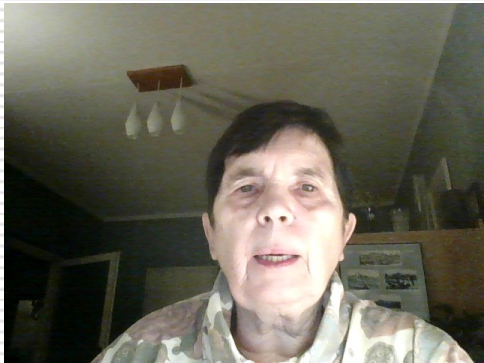
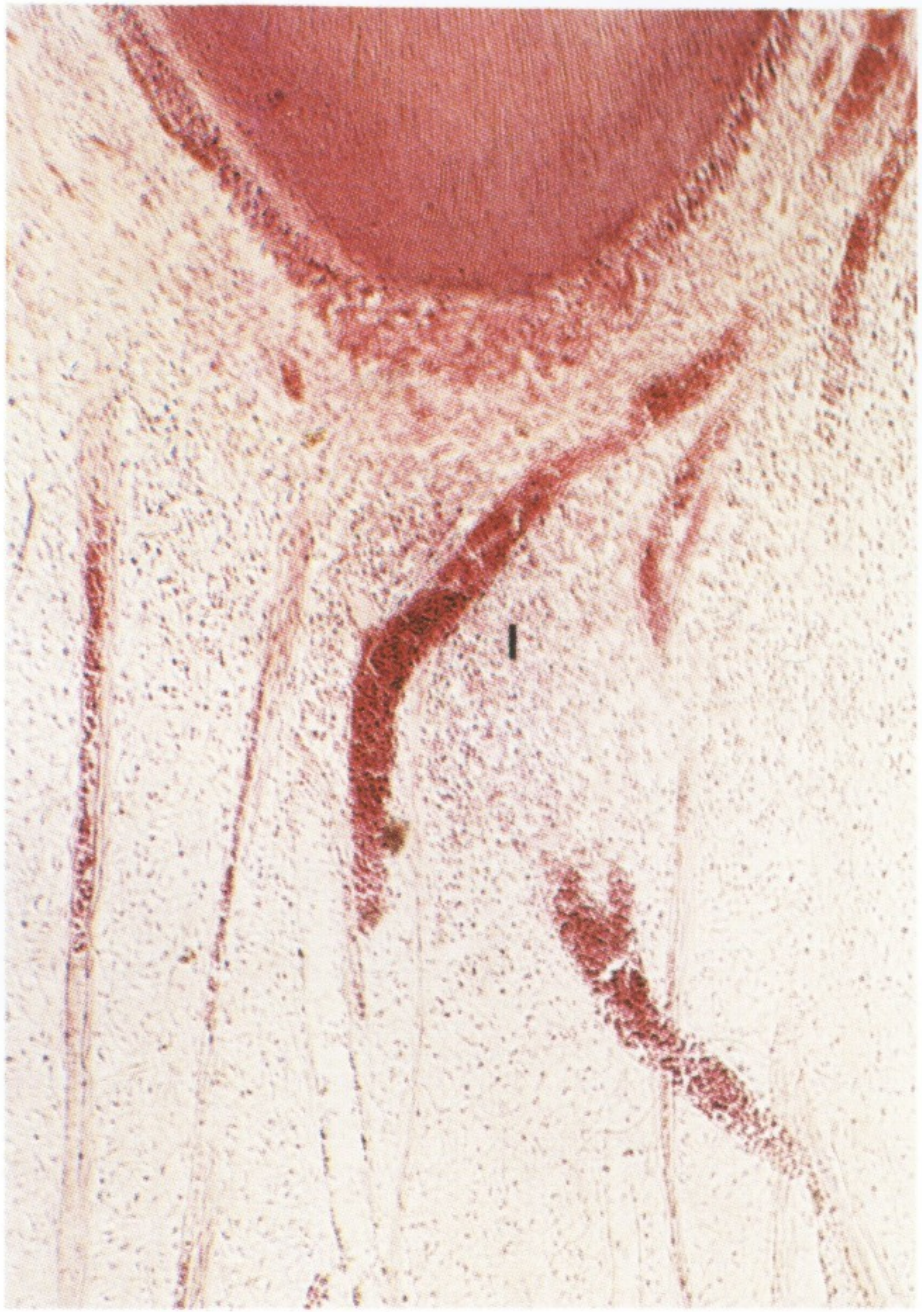


Fig 6-7 Tertiary dentin (TD) formed as a response to the healing of a lesion similar to that shown in Fig 6-5. Note stained, atubular interface dentin (I) and the dentina the tertiary dentin. The odontoblasts lining the pulp the tertiary dentin are short, and the cell-free zone this area. (Hematoxylin-eosin stain; original magnificat

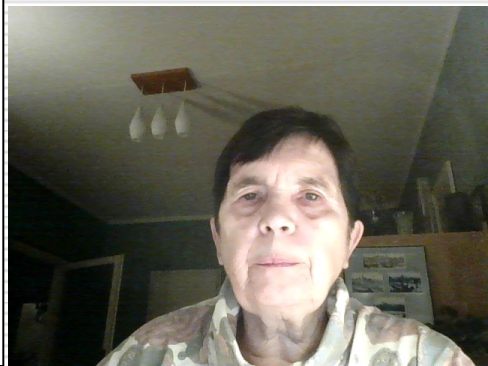


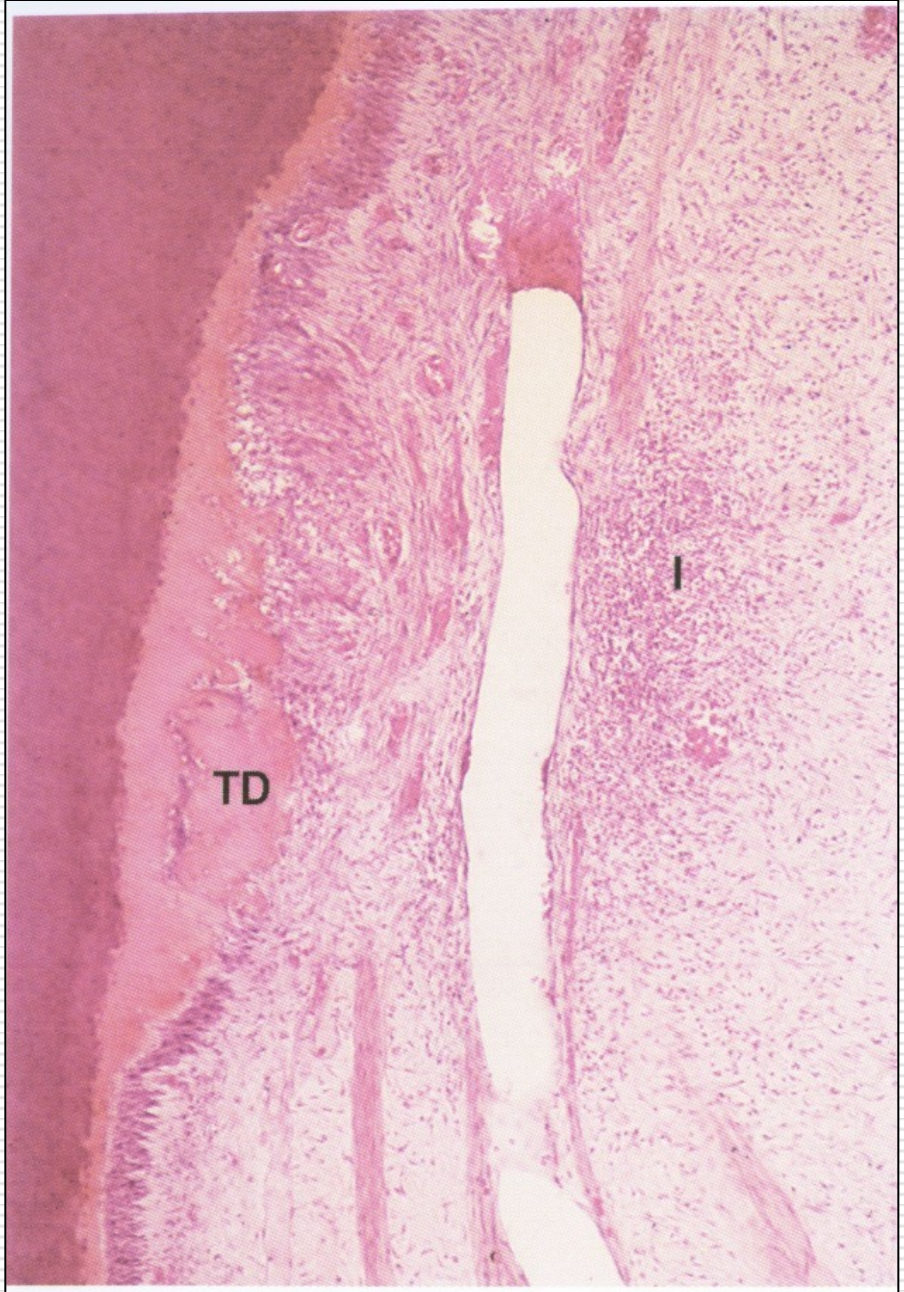
Obr. 2.11 Výrazné poškození pulpy po preparaci turbinou s omezeným chlazením a razantním vysušení kavity alkoholem.



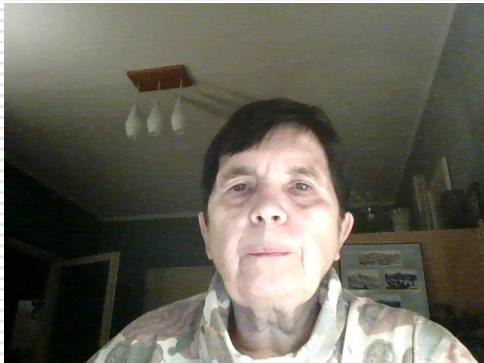


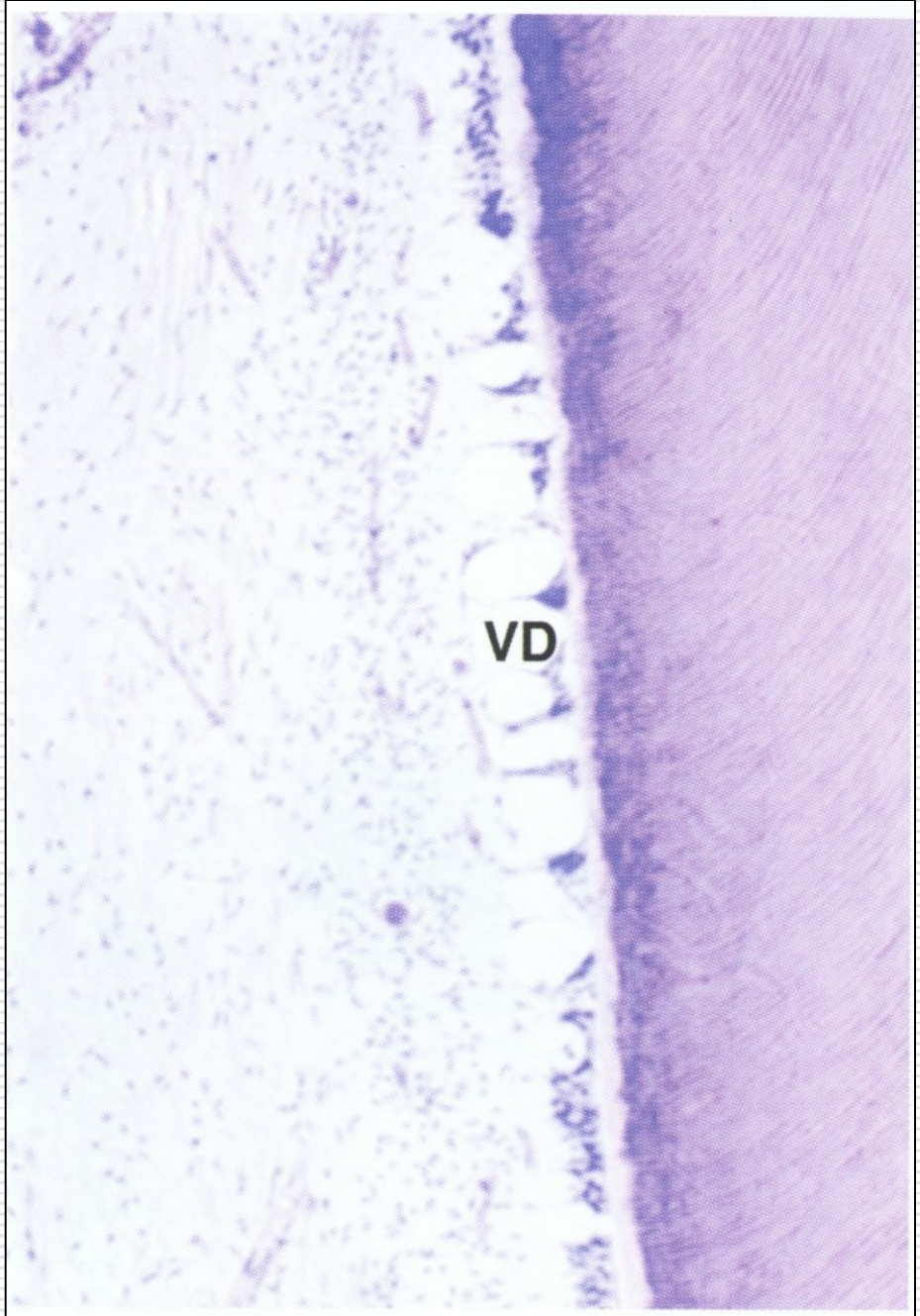
Obr. 2.12 Zánětlivý infiltrát (I) za tři týdny po zaplnění kavity silikátovým cementem.



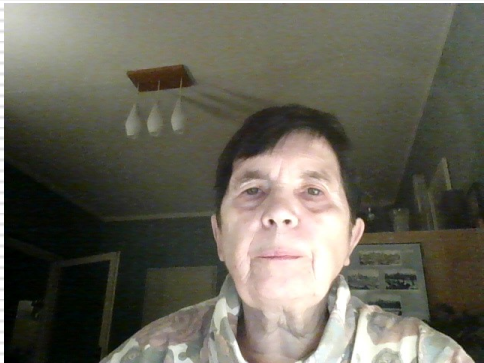


Obr. 2.13 Zánětlivý infiltrát (I) a překotná lokální tvorba terciárního dentinu (TD) v zubní dřeni, kde před třemi týdny byla kavita zaplněna GIC.





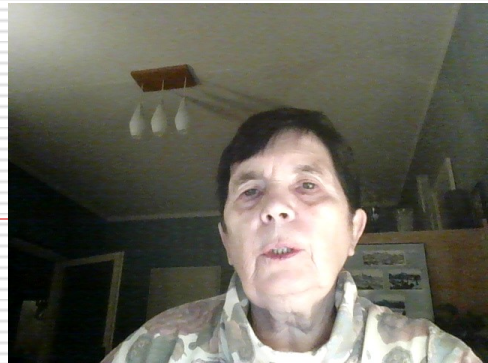
Obr. 2.15 Vakuolární degenerace (VD) odontoblastů.



Inflammation of the dental pulp

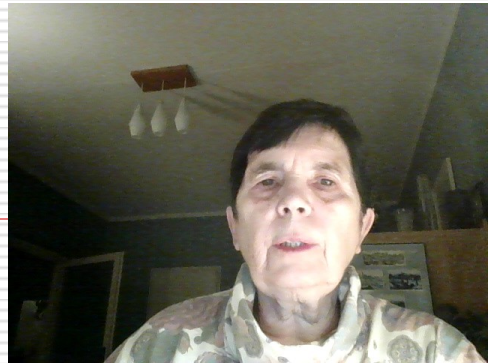
- **According to the nature**

- Reversible
- Irreversible



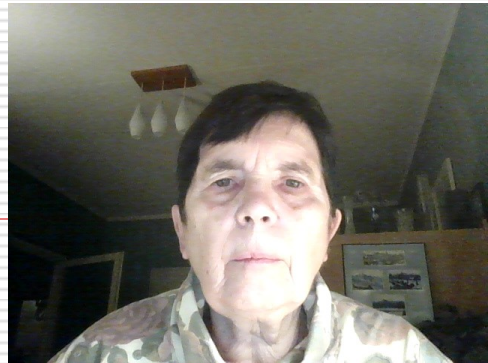
Reversible pulpitis - Hyperemia

- Capillary bed - enlargement, vasodilatation, elevated capillary pressure, increased vascular permeability, potentially reversible response

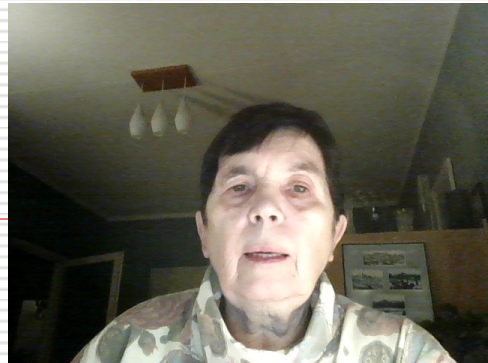


Irreversible pulpitis

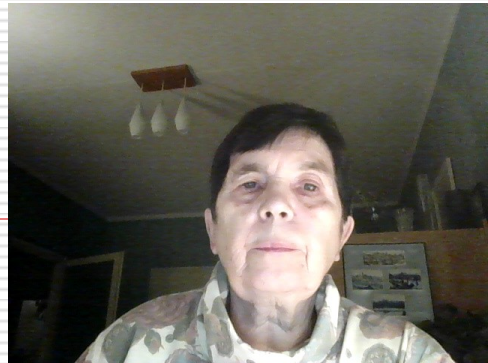
Acute - hyperactivity of exudative forces
polymorphonuclear leucocytes, later macrophage
Chronic - proliferative phase
granulomatous tissue
pain is usually absent
lymphocytes, plasma cells, macrophages
limited number of PMNS



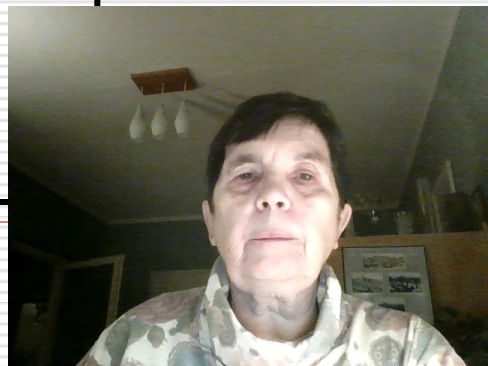
- **Duration and severity**
- acute
- chronic
- subacute



- **Presence/absence of pain**
- painful
- non painful



acute	chronic
partial serous suppurative	open ulcerative polypous
total serous suppurative	closed
retrograde	



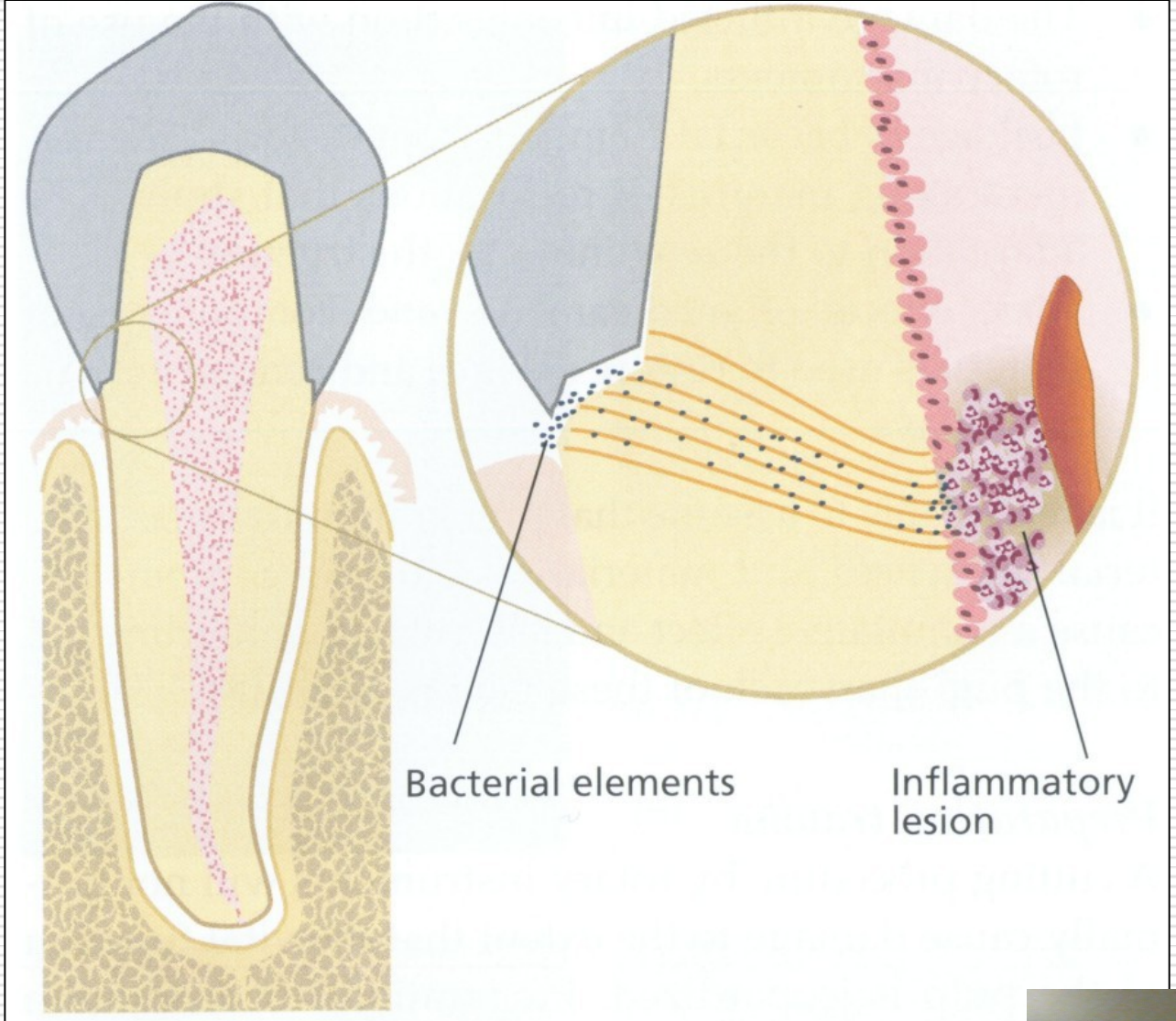
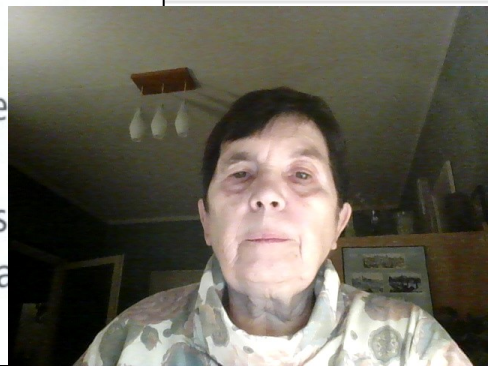
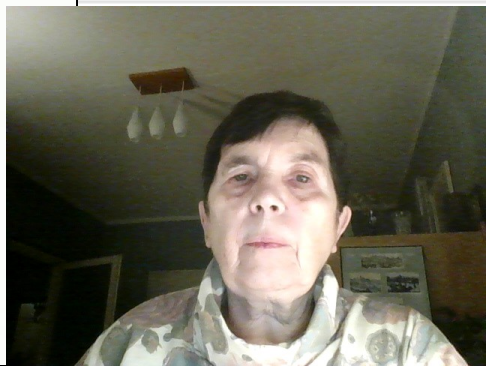


Fig. 3.24 In contraction gaps or after incomplete coverage of de
lowing restorative procedures, bacterial elements in the oral cavity
access to pulp along the exposed dentinal tubules. This is regarded as
threat to the pulp because it may induce painful symptoms and infla
lesions in the pulp.





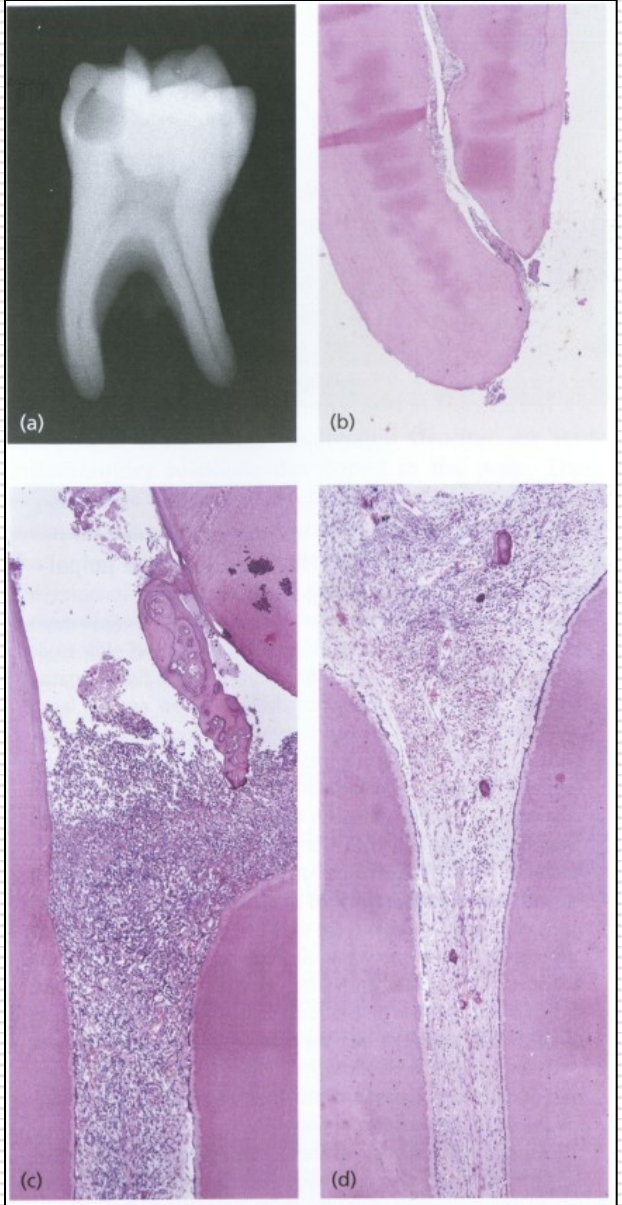
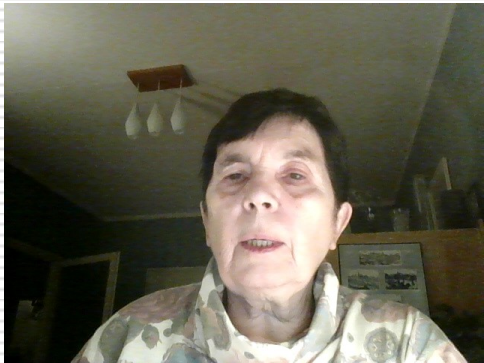
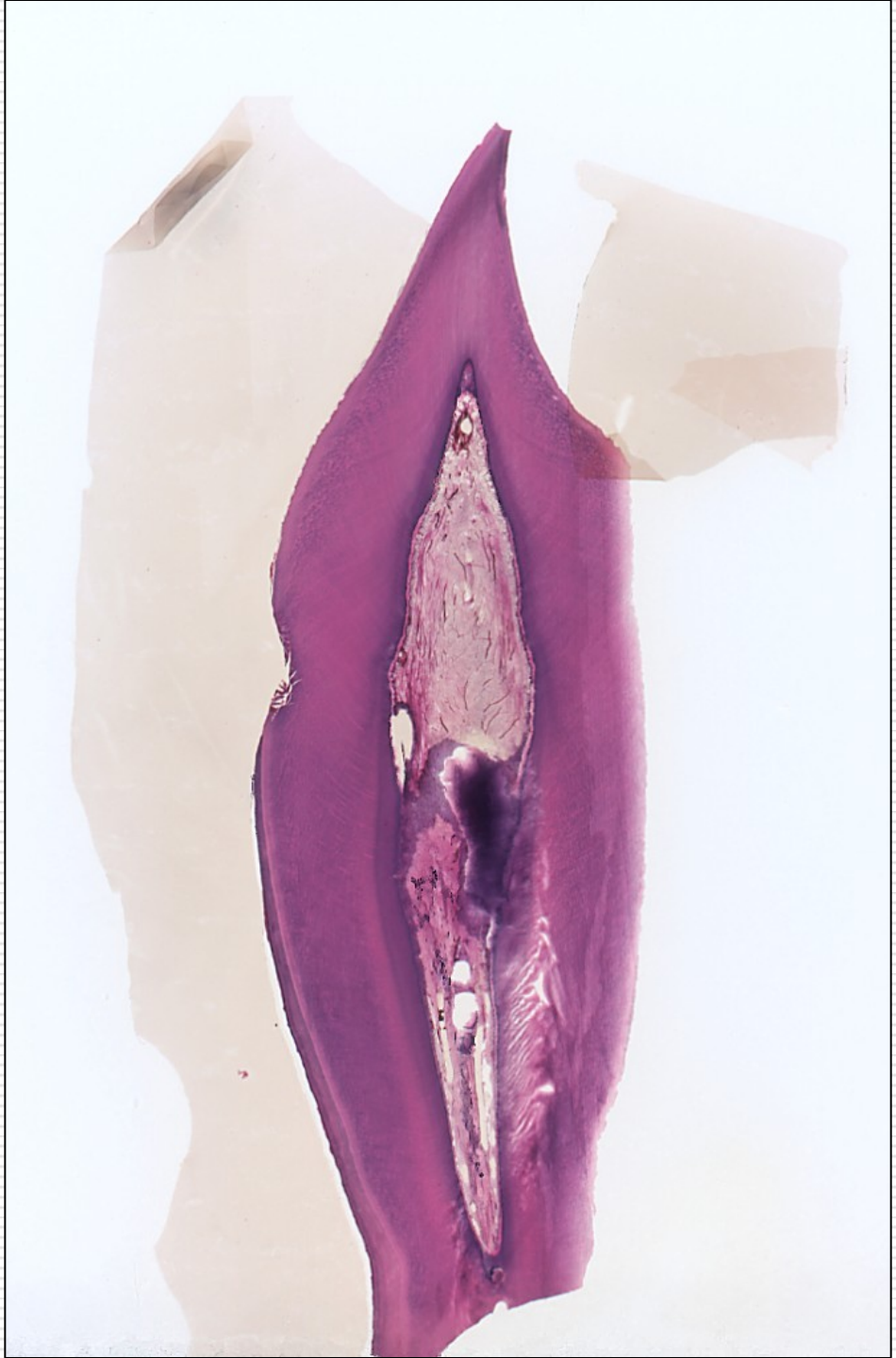
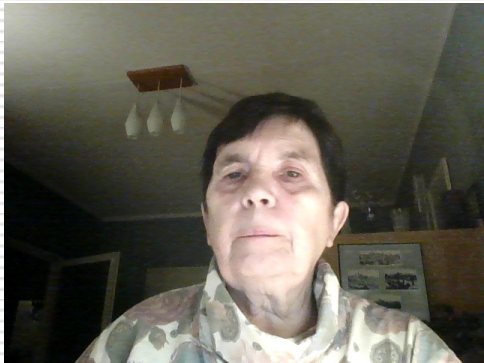
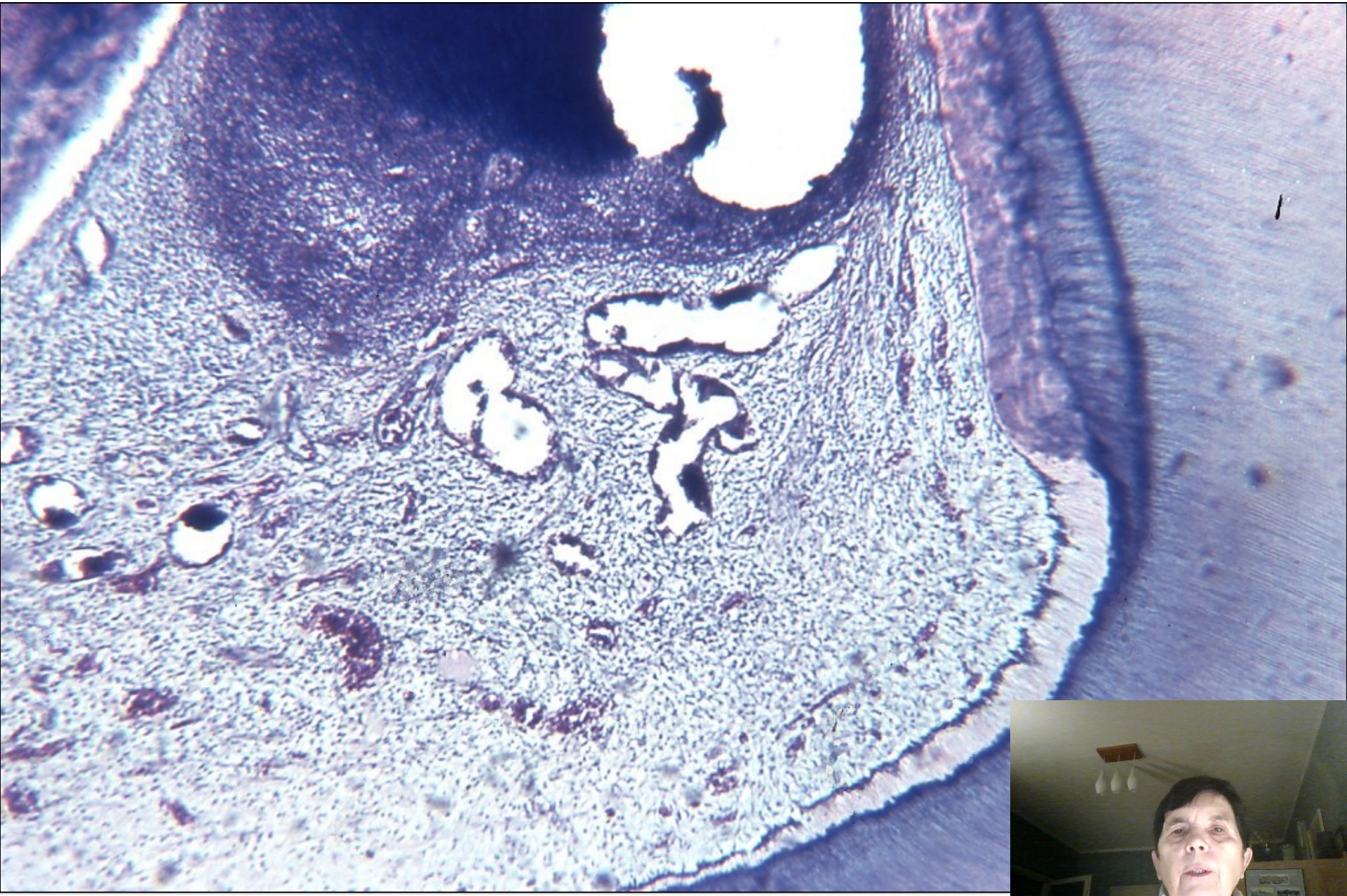


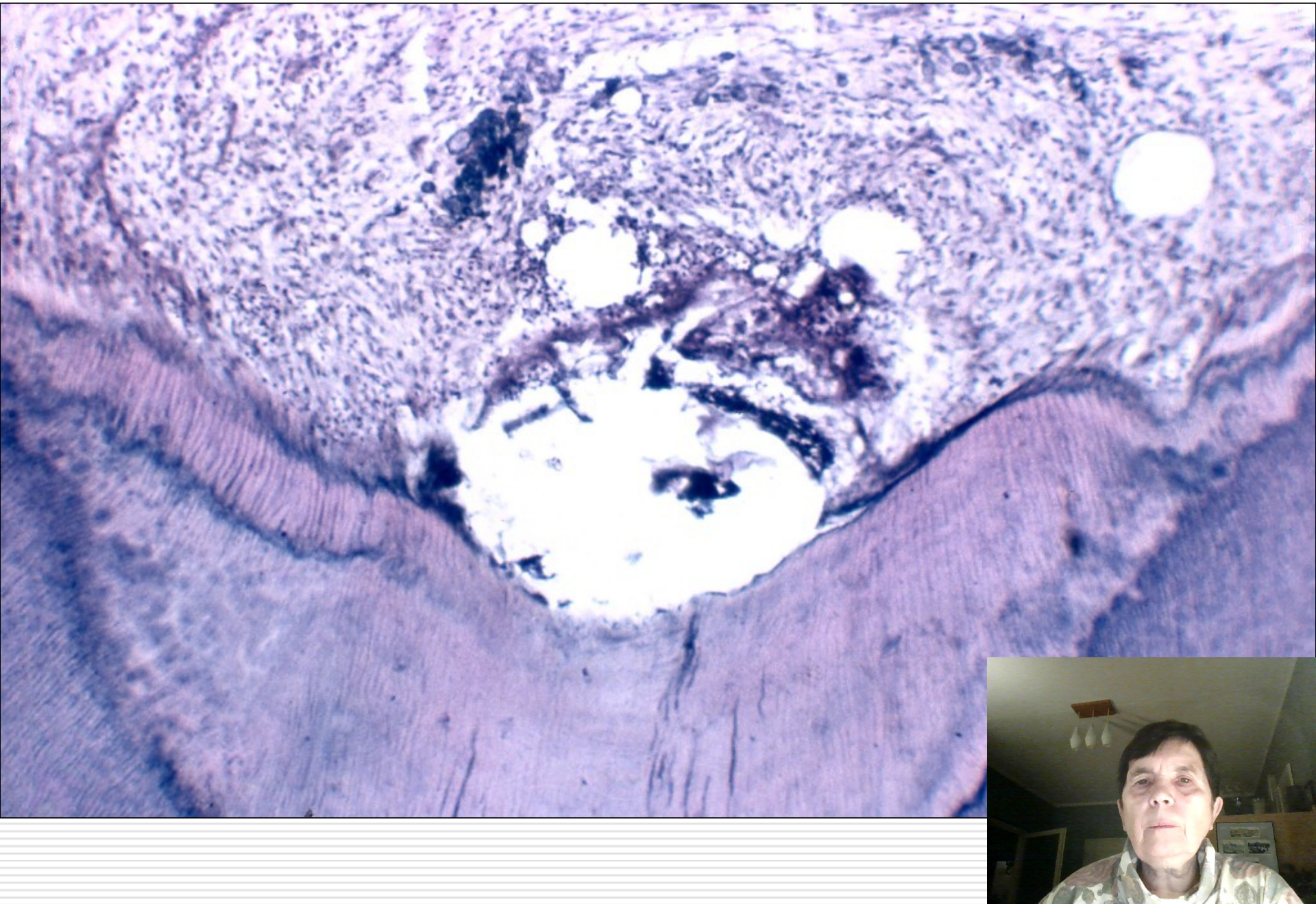
Fig. 3.22 (a) Radiograph showing a deep, mesio-occlusal caries lesion that has advanced to the pulp in a lower molar. Histological examination of the pulp in the extracted tooth reveals partial pulpal breakdown at the break-through of the caries lesion. (b) The apical pulp displays a normal appearance. (c) An intense inflammatory infiltrate extends into the orifice of the mesial root canal. (d) The pulp tissue of the distal root canal shows less leukocyte infiltration with an intact odontoblast cell layer. (Courtesy of Dr Domenico Ricucci.)

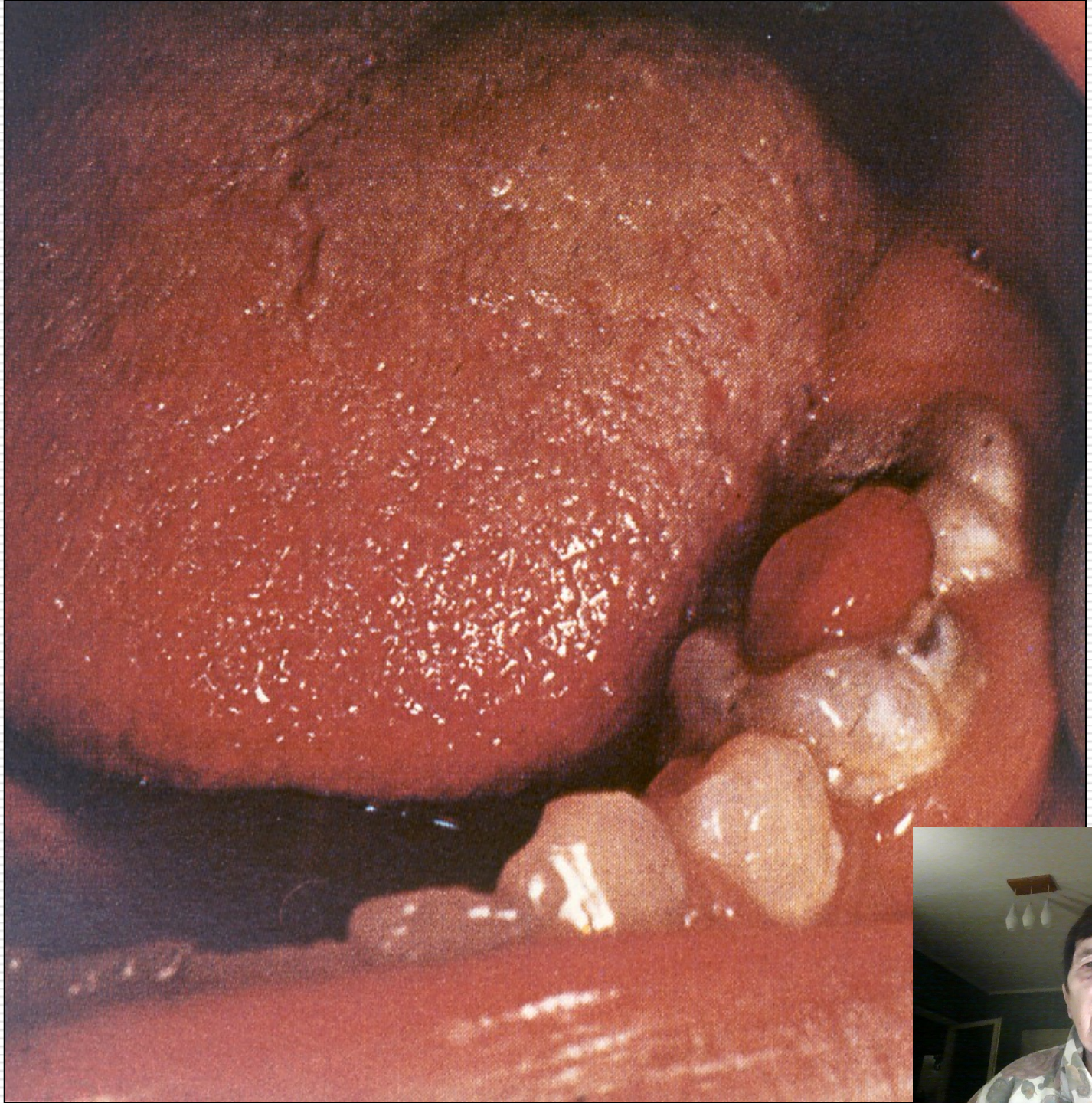


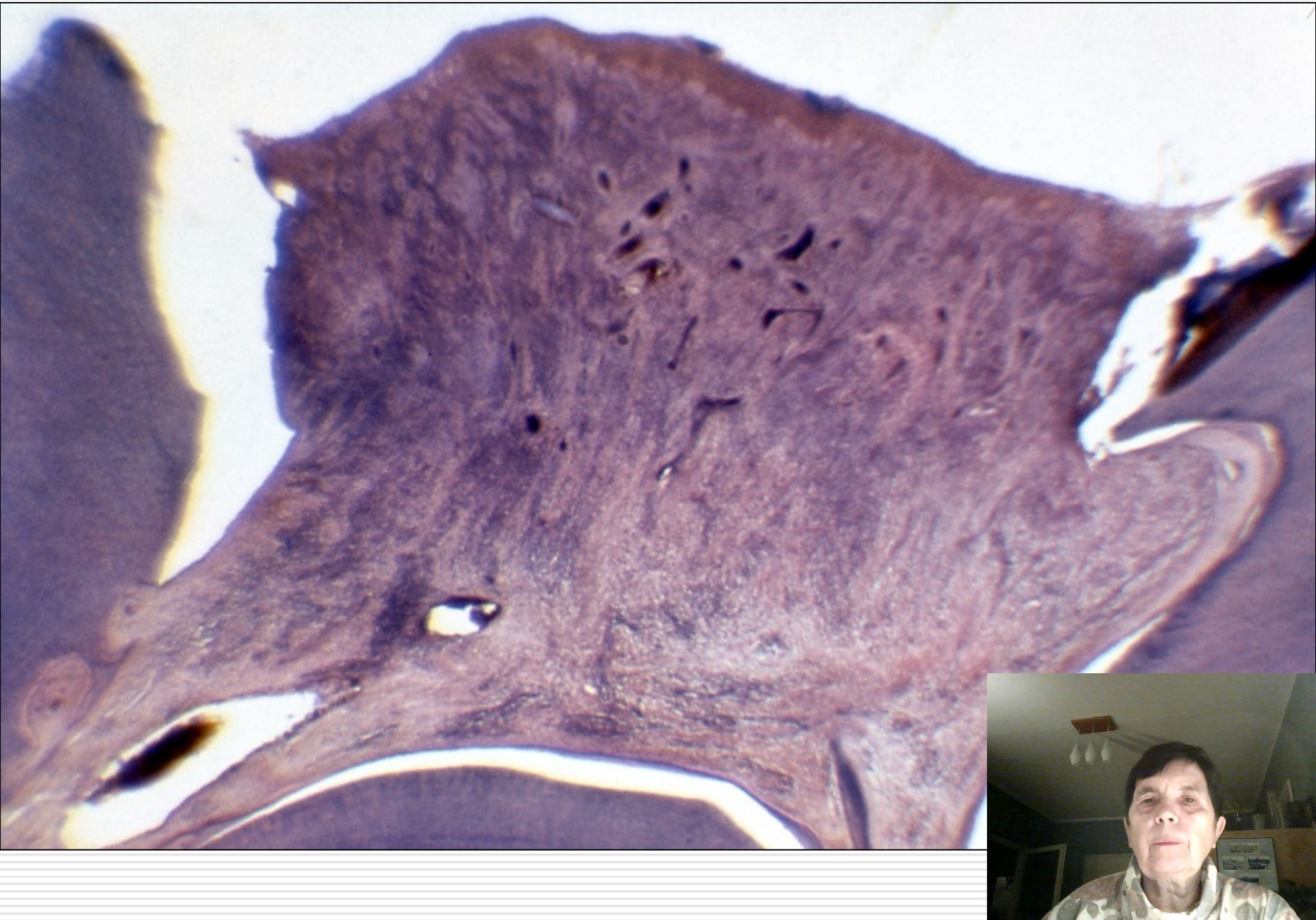












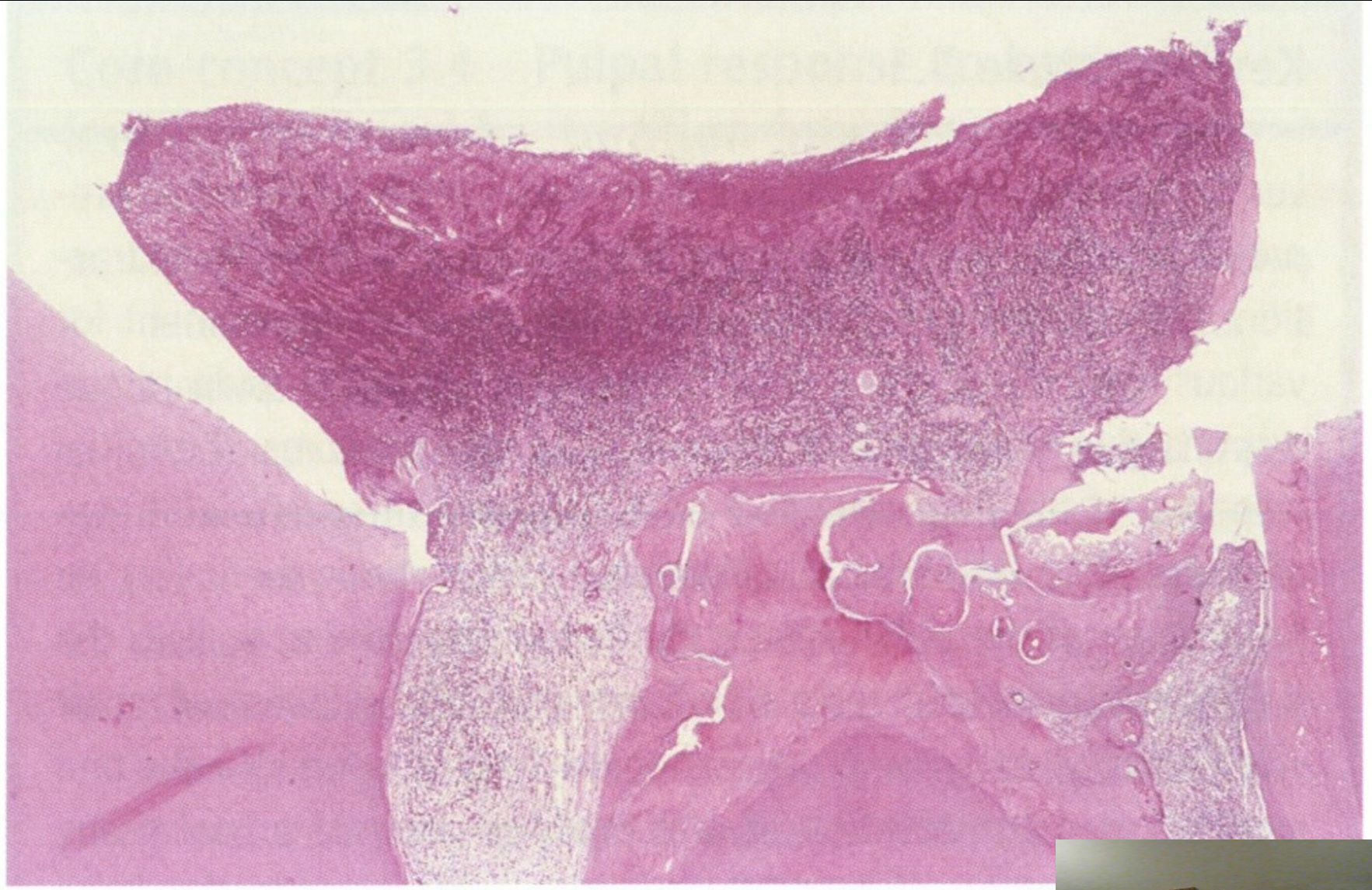
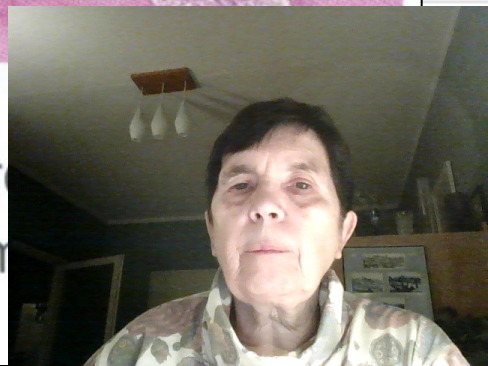
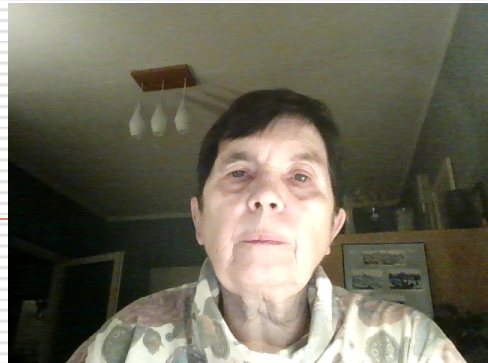


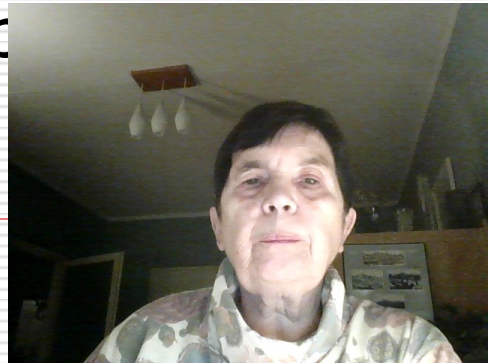
Fig. 3.23 Microphotograph of a pulp polyp extending fr
young tooth broken down by caries. Note the dense inflamm
the proliferating tissue. (Courtesy of Dr Domenico Ricucci.)



Acute - hyperactivity of exudative forces
polymorphonuclear leucocytes, later macrophage
Chronic - proliferative phase
granulomatous tissue
pain is usually absent
lymphocytes, plasma cells, macrophages
limited number of PMNS

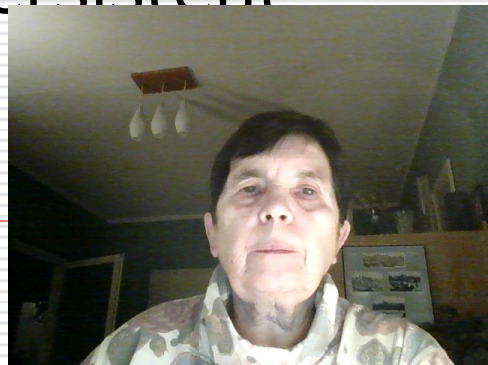


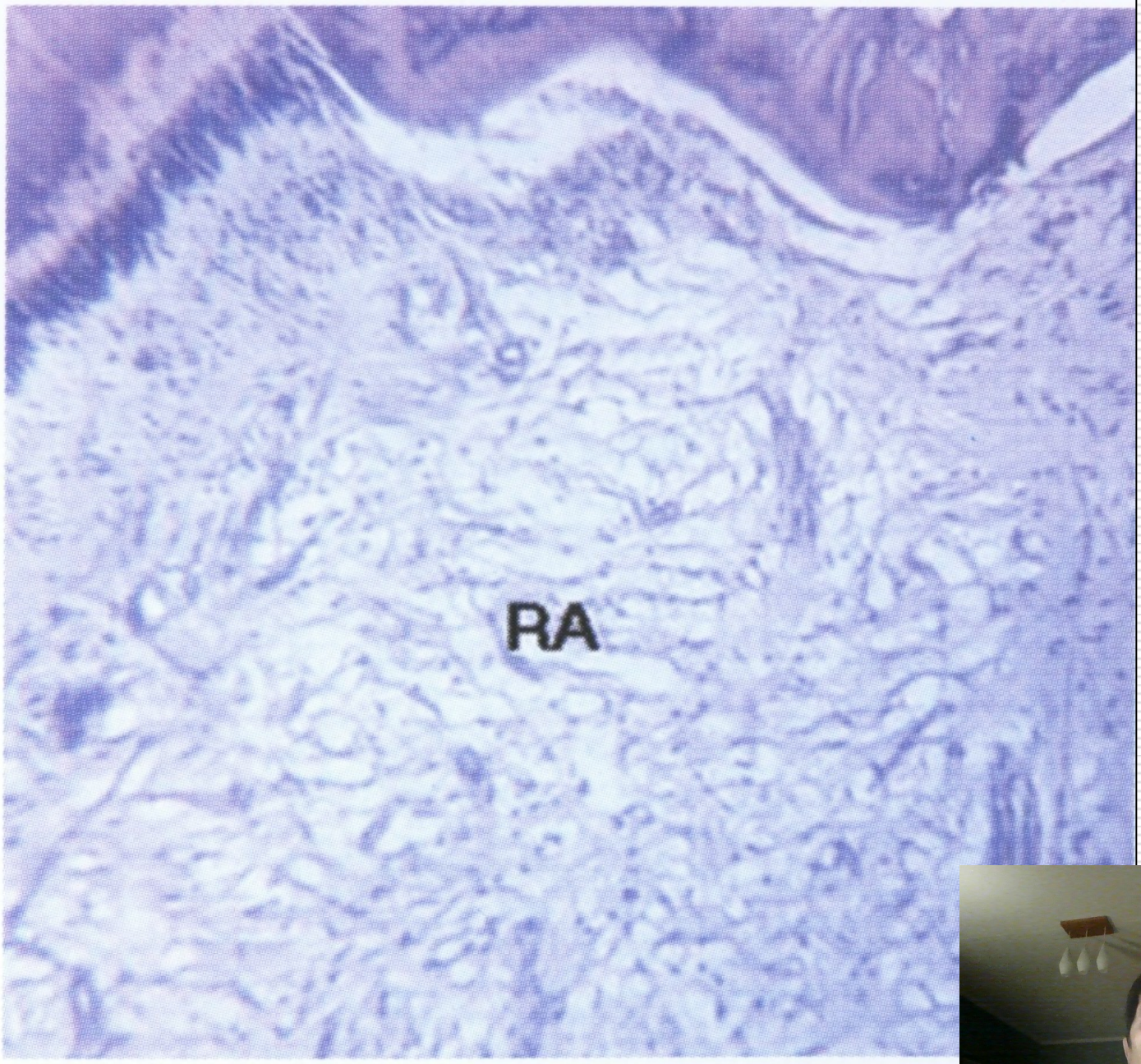
- Irreversible
  - painful pulpitis
- acute pulpitis
- subacute pulpitis
  - nonpainful pulpitis
- chronic ulcerative pulpitis (due to caries)
- chronic pulpitis (no caries)
- chronic hyperplastic pulpitis (pulp p



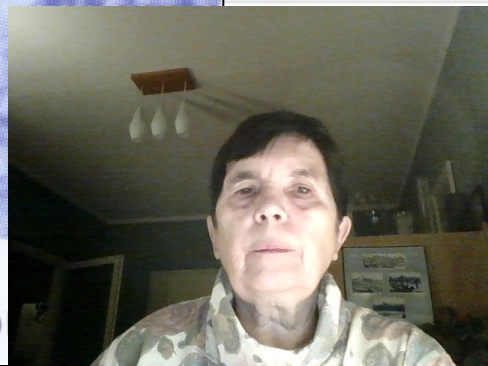
Additional pulp changes

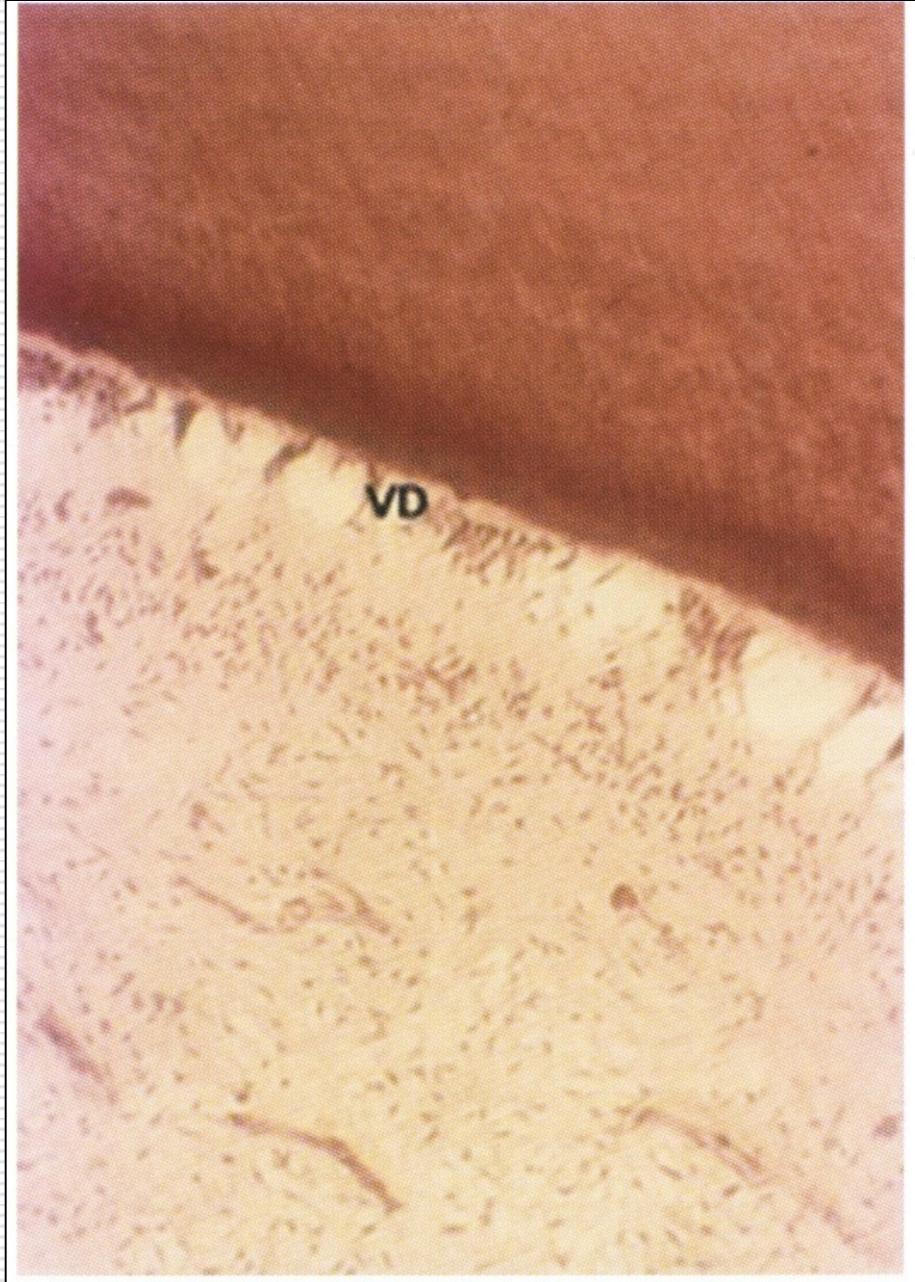
- retrogressive changes
  - atrophy, fibrosis, vacuolization of odontoblasts, hyalinization
- dystrophic calcification
- pulp stones
  - internal resorption (sequel to persistent chronic inflammation)



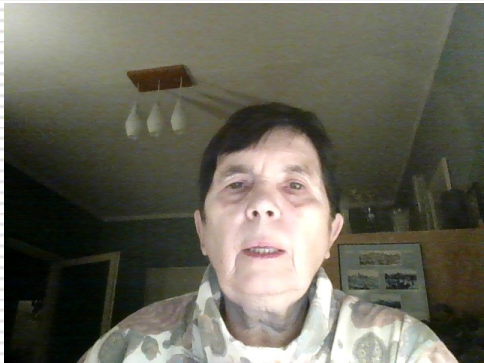


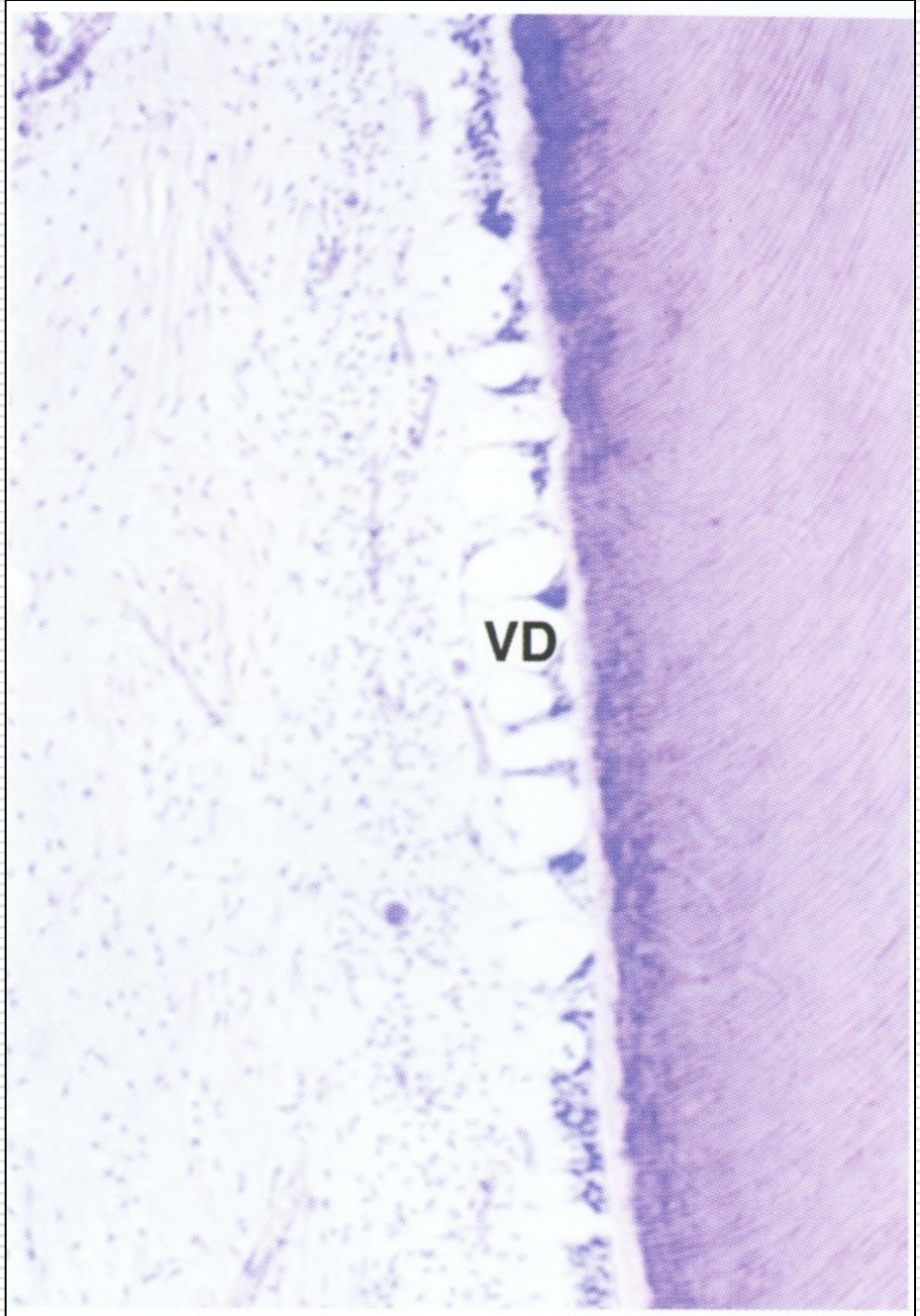
Obr. 2.17 Síťovitá, retikulární atrofie (RA) p



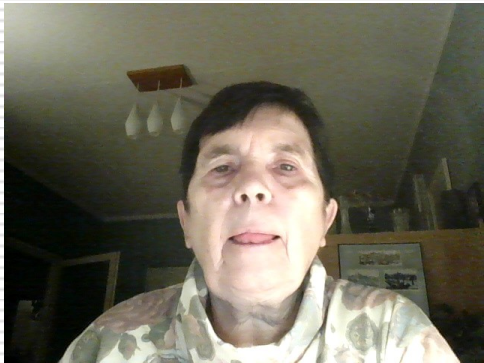


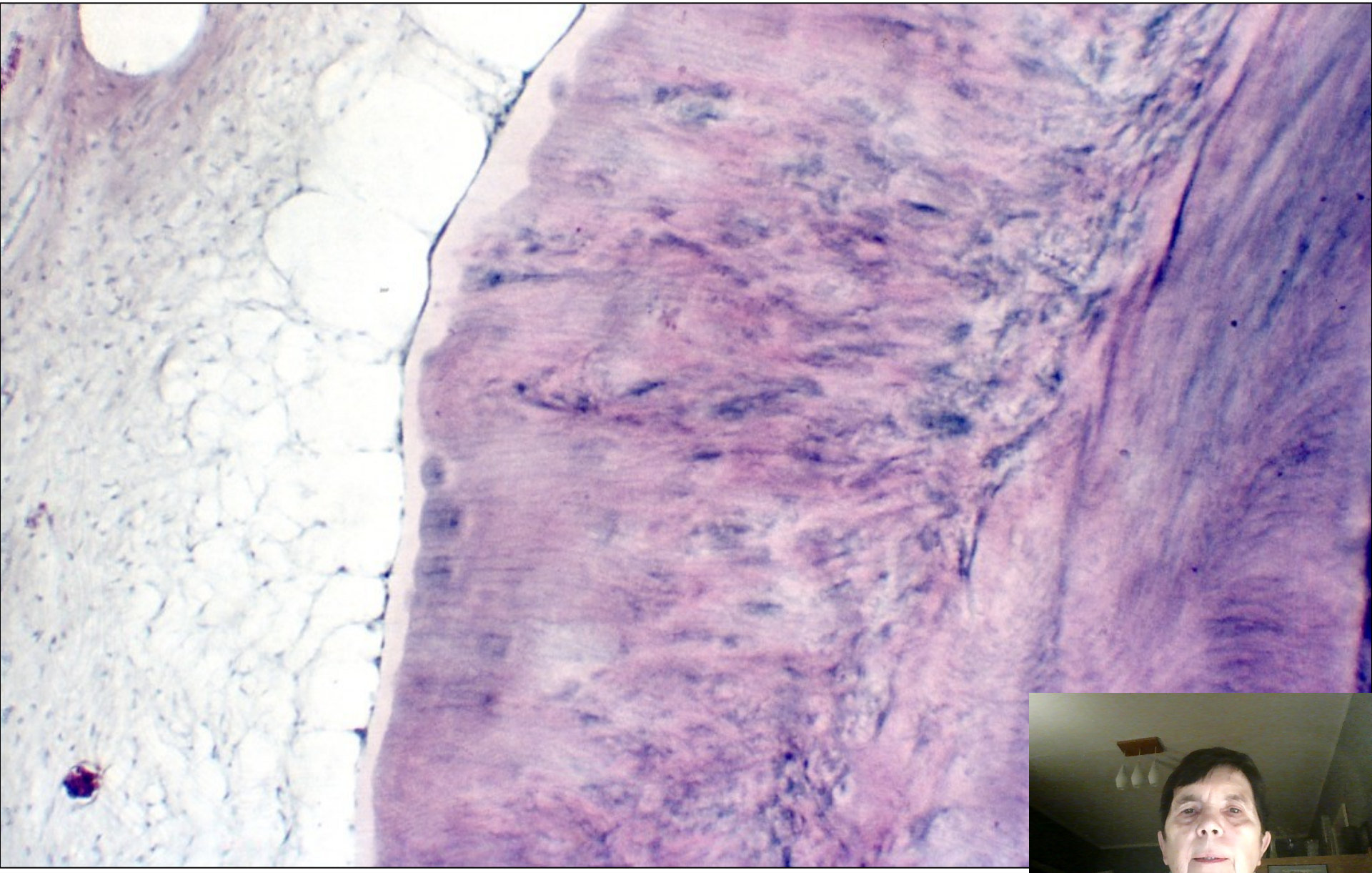
Obr. 2.9 Vakuolární degenerace (VD) odontoblastů nalezená v pulpě za 30 dnů po preparaci kavity turbinou.

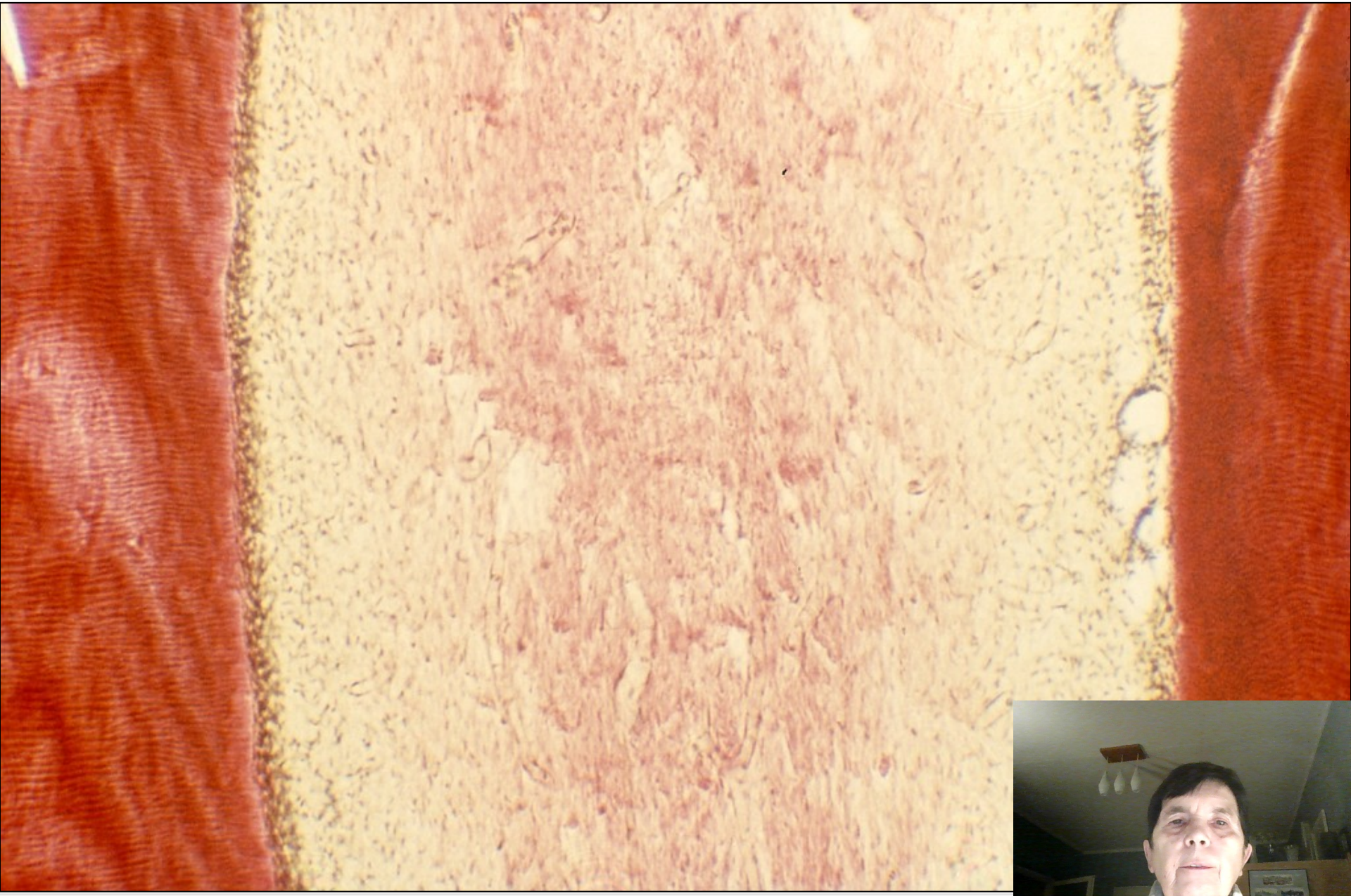


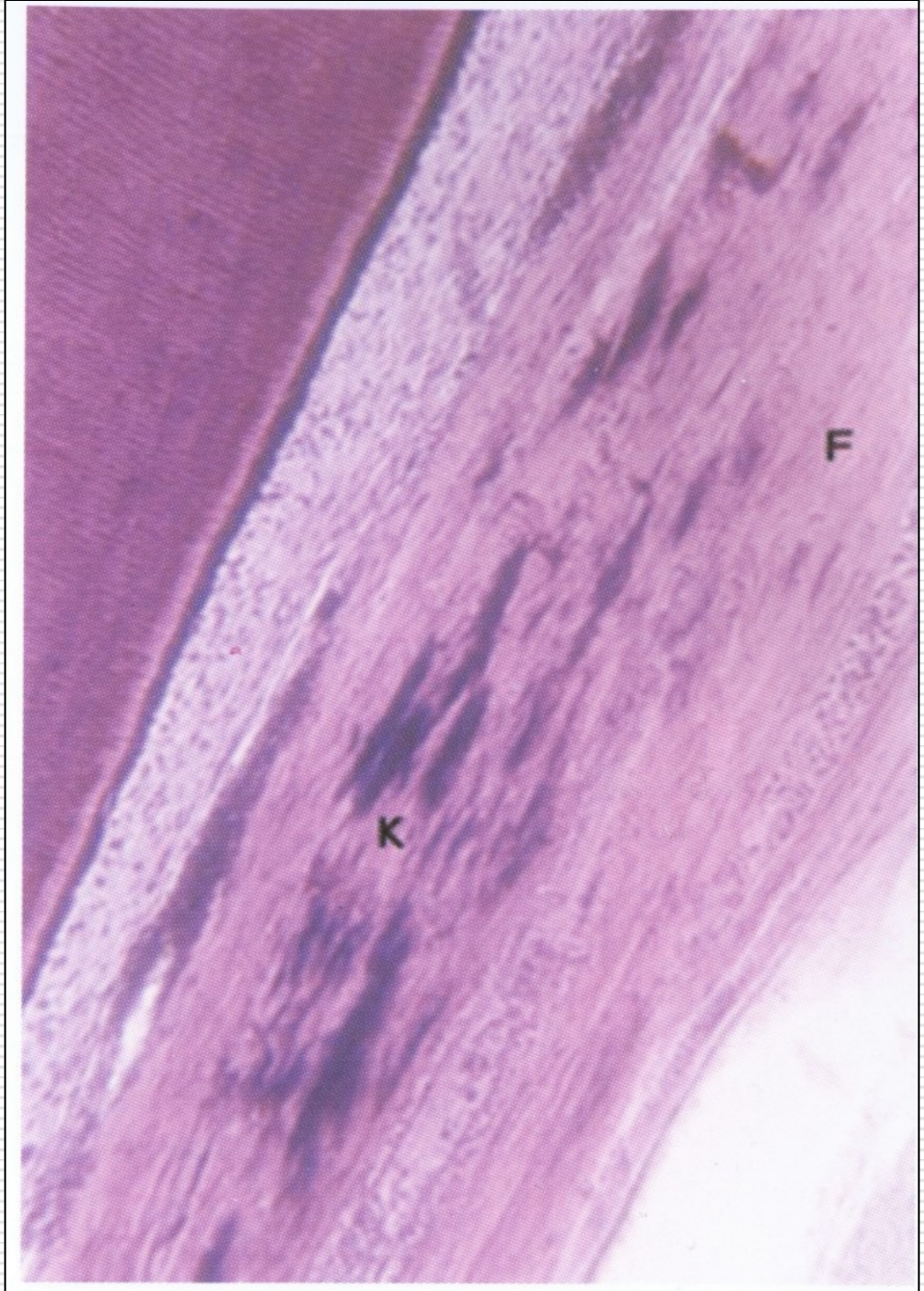


Obr. 2.15 Vakuolární degenerace (VD) odontoblastů.

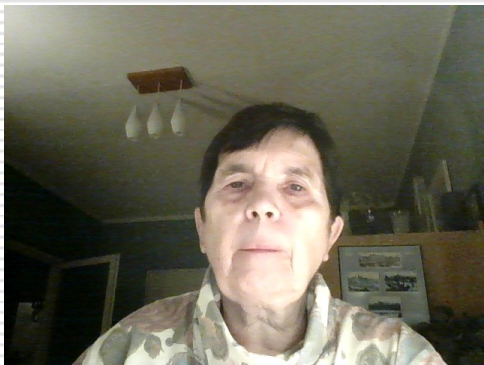


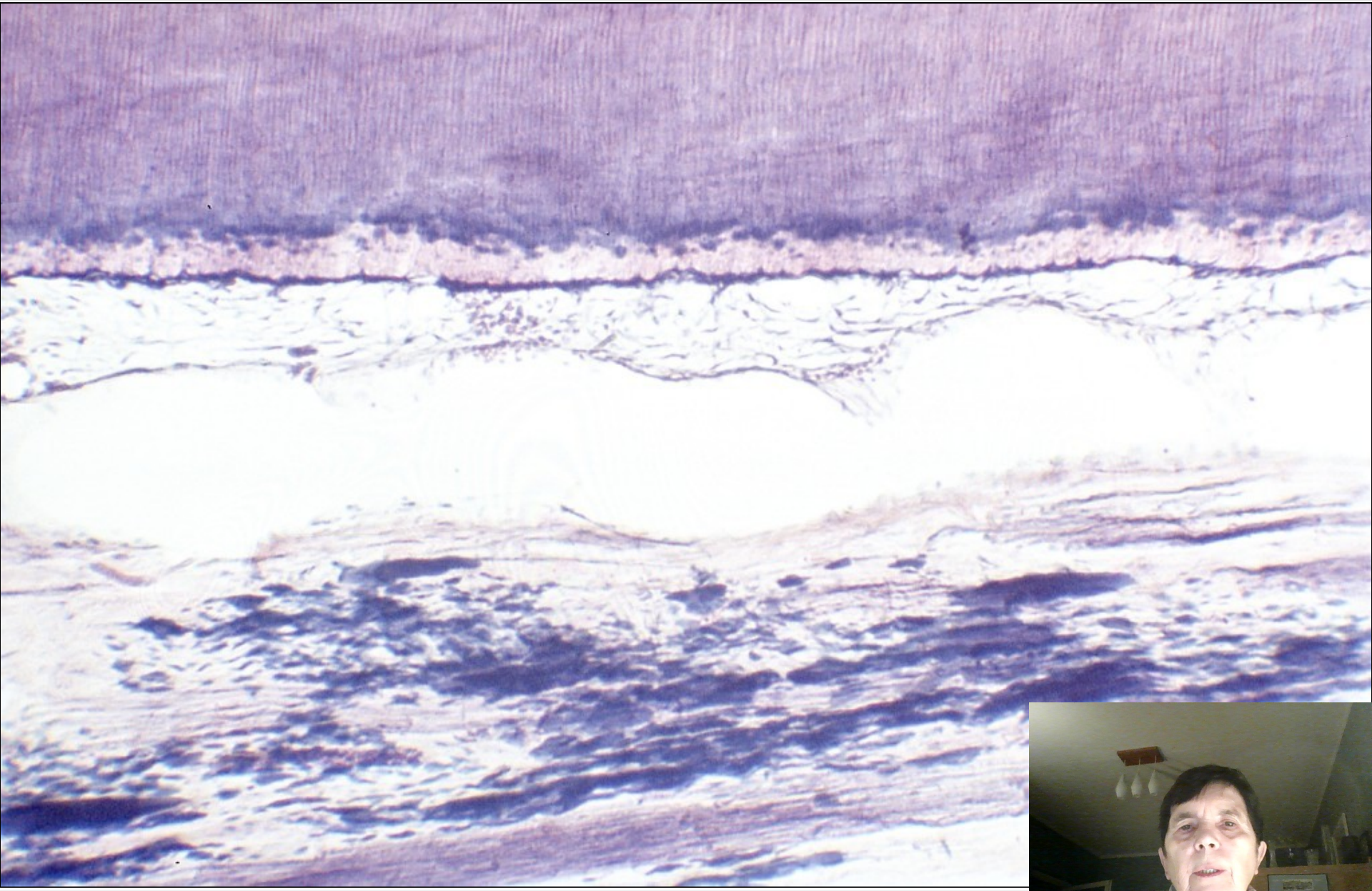


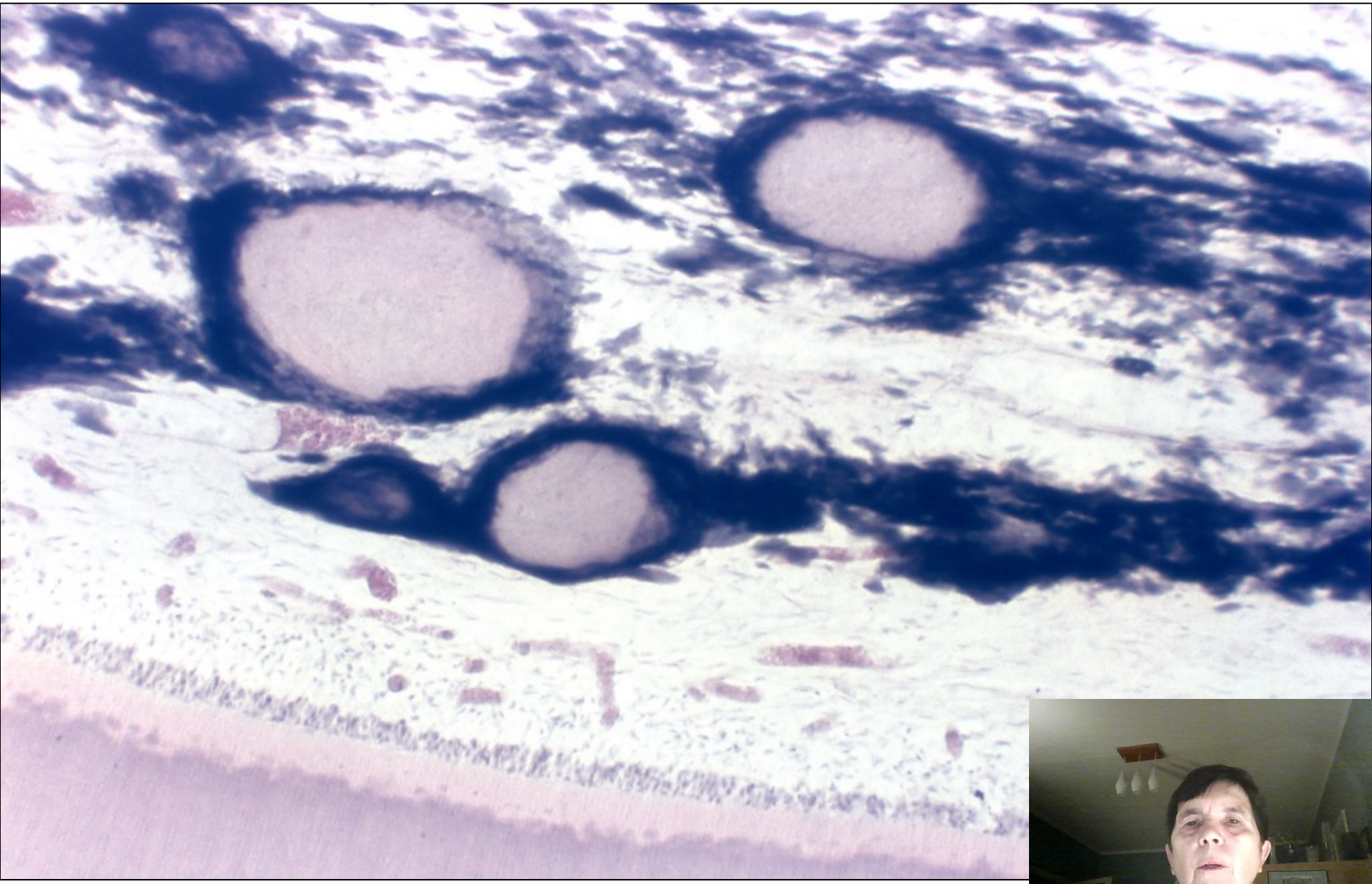


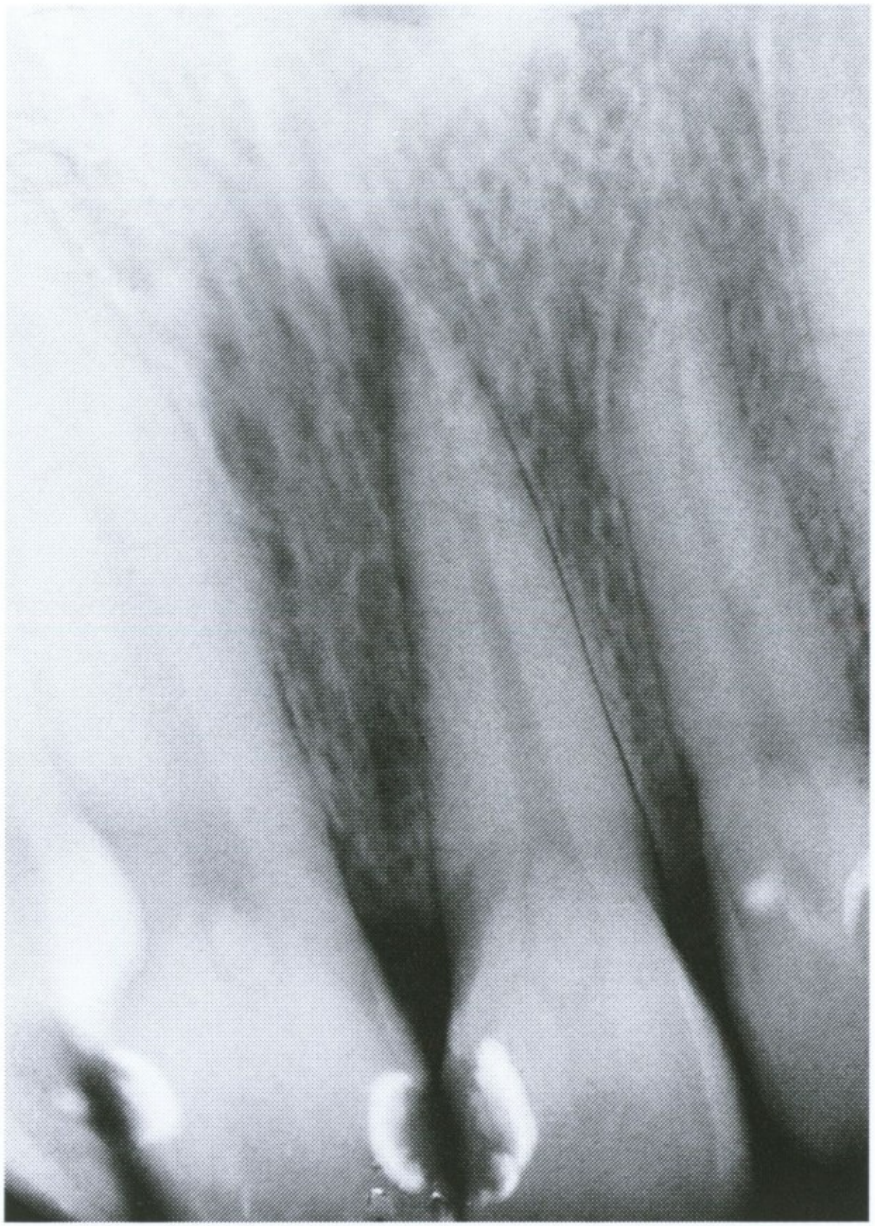


Obr. 2.16 Fibróza (F) pulpy s četnými kalcifikacemi (K) v zubu staršího pacienta (60 let).

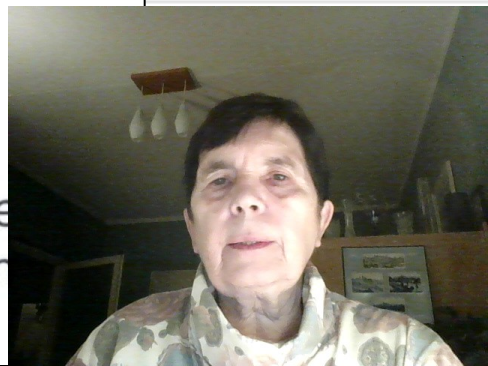


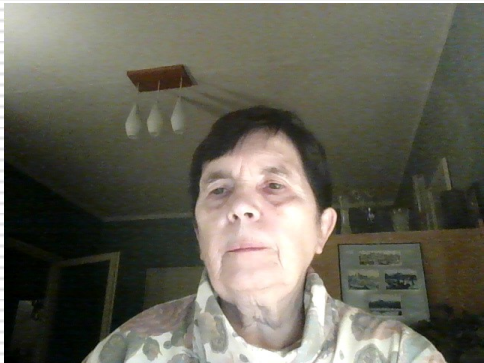


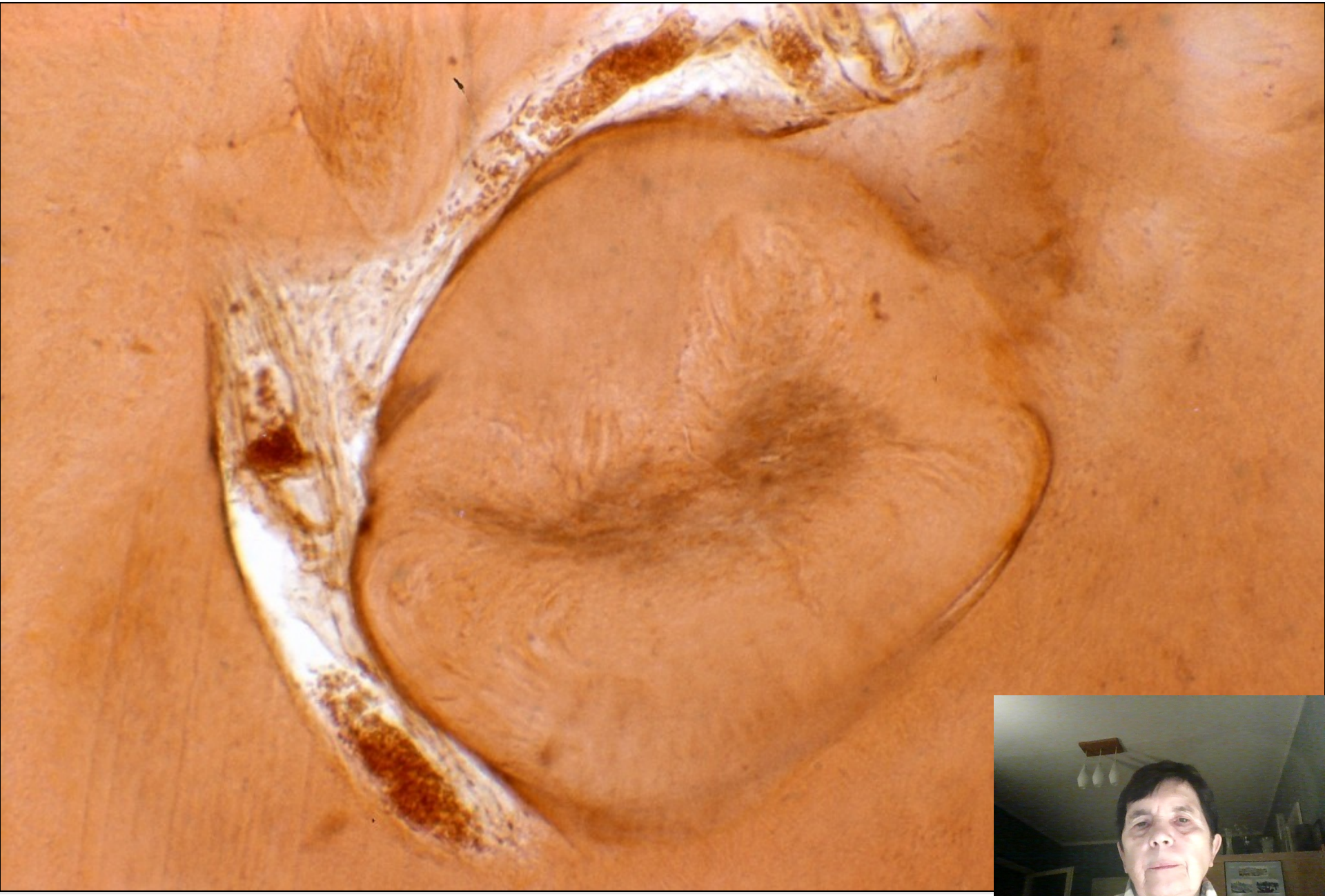


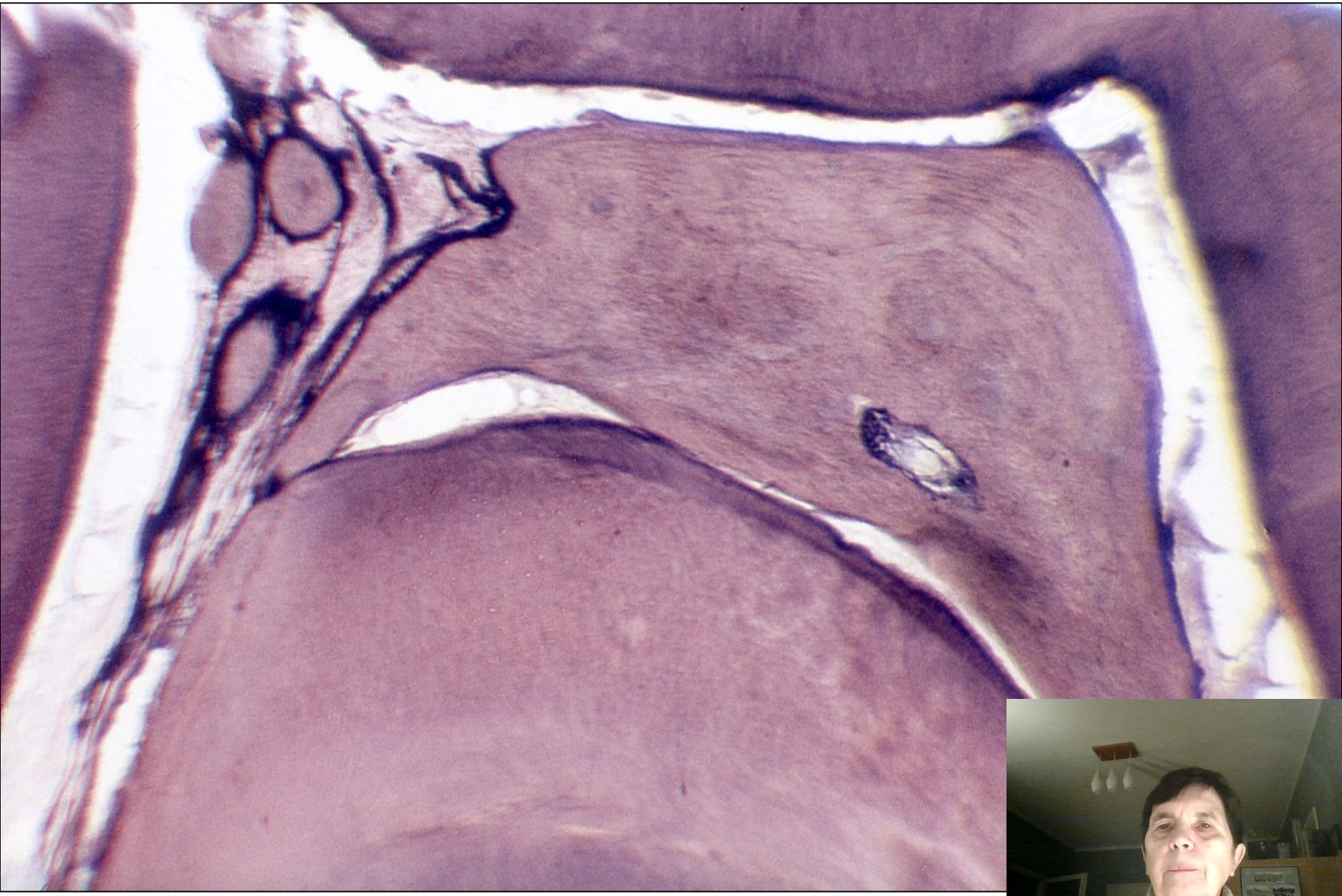


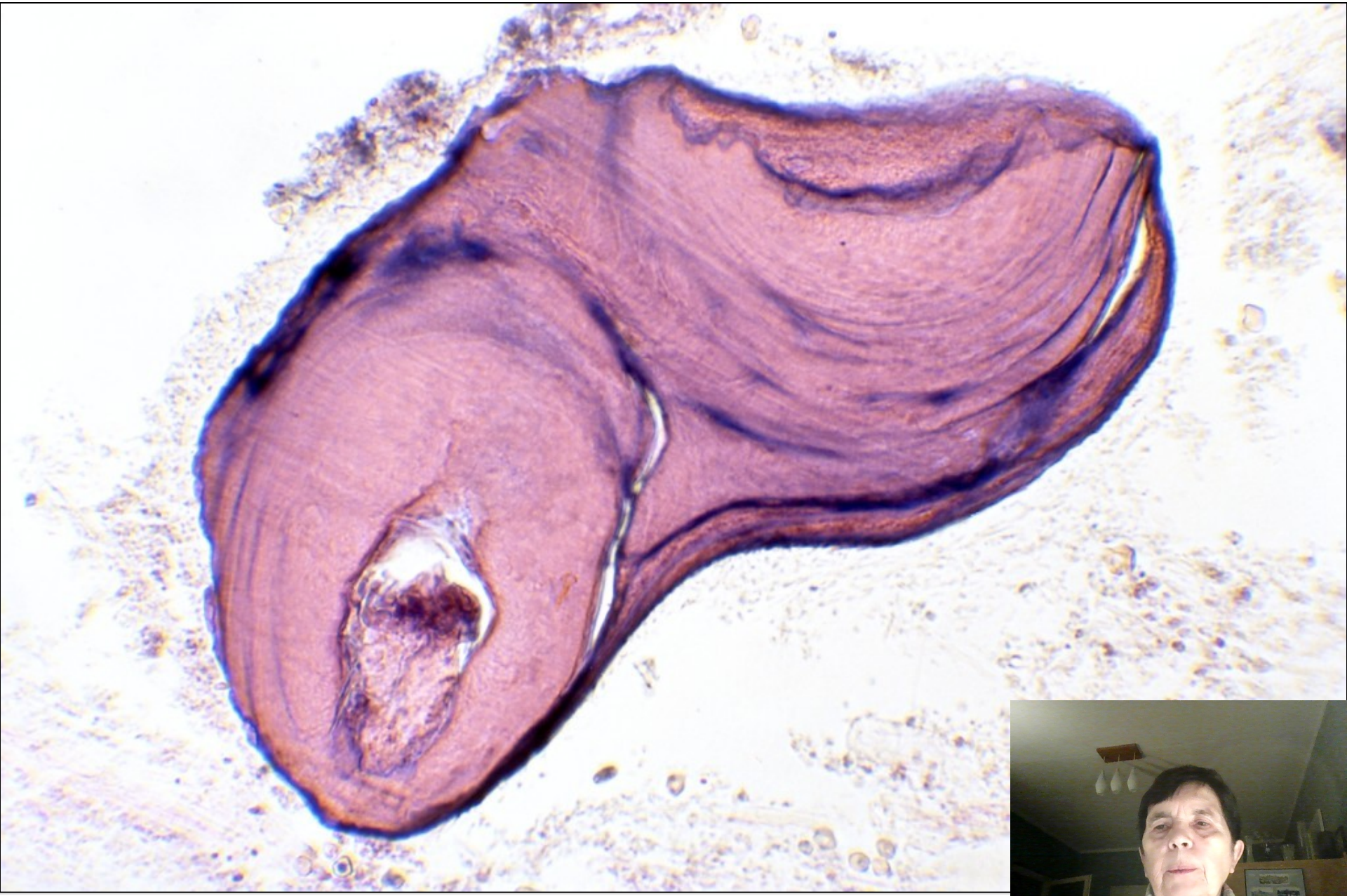
Obr. 2.19 Rentgenogram horních řezáků s ve
vejčitými dentikly ve dřeňové dutině a chron
apikální periodontitidou v zubu 21.

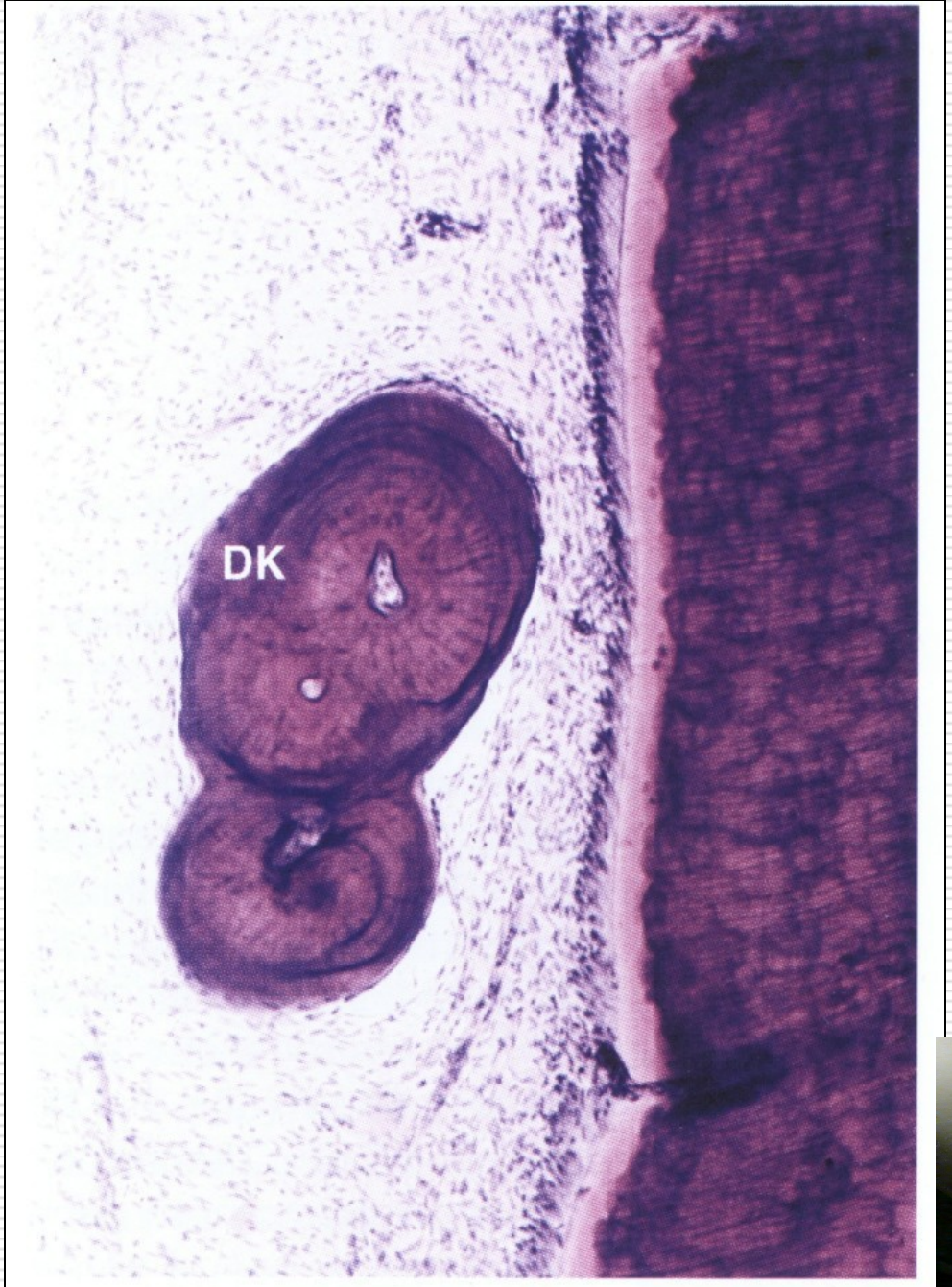




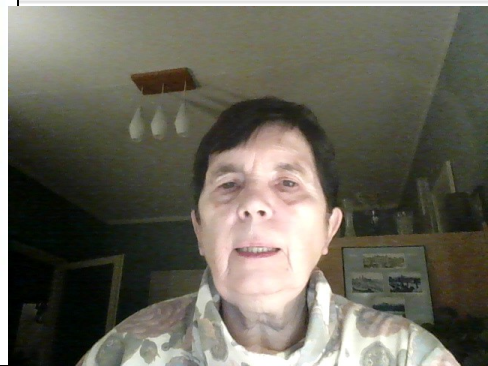






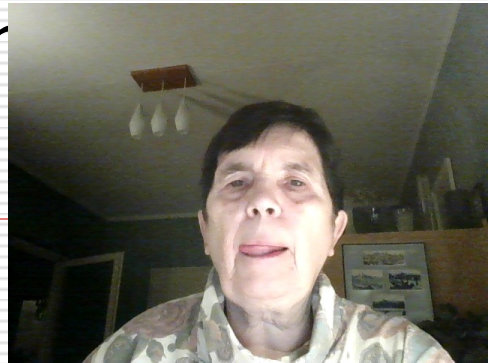


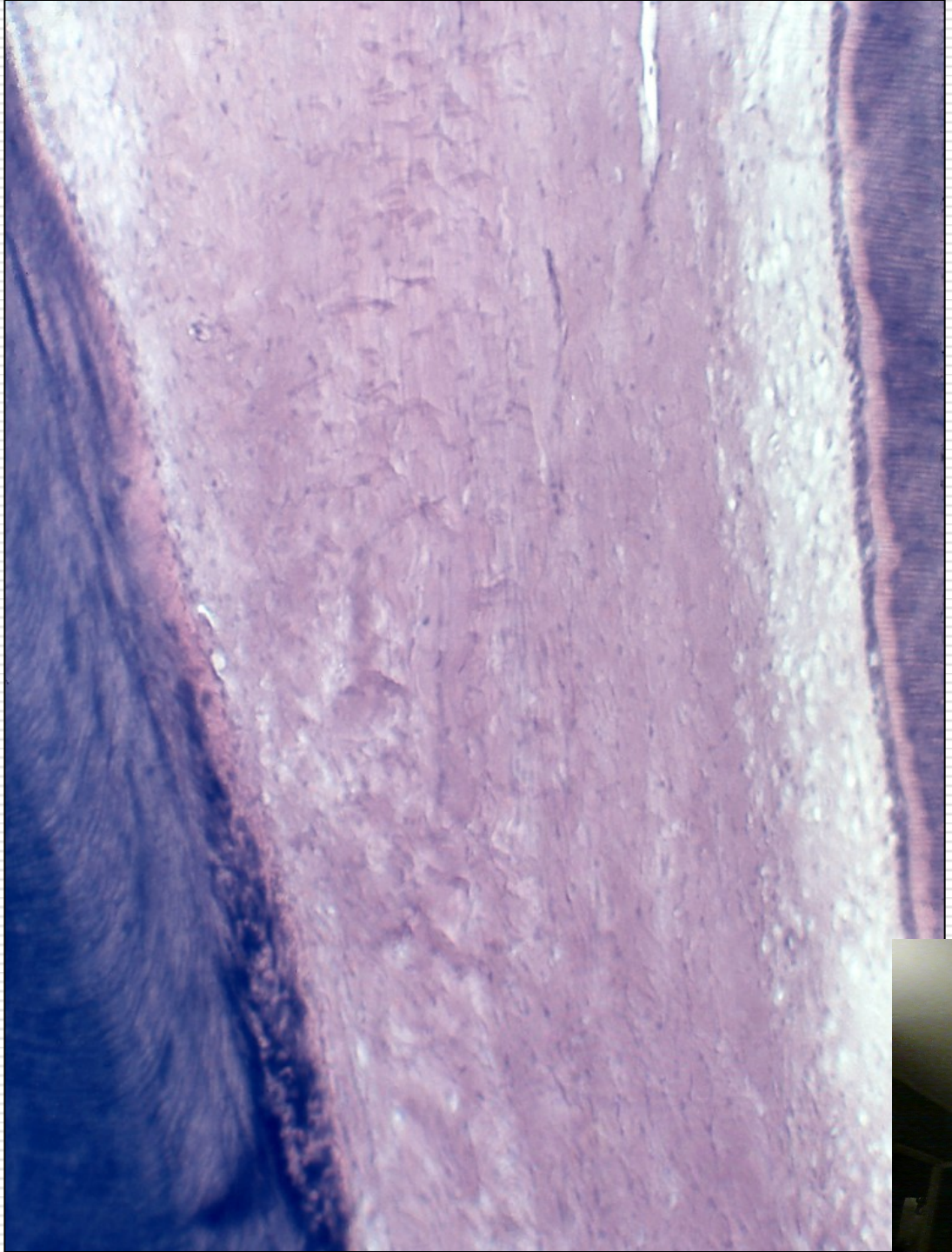
Obr. 2.20 Volný pravý dentikl (DK) v mladé a zdravé pulpě.

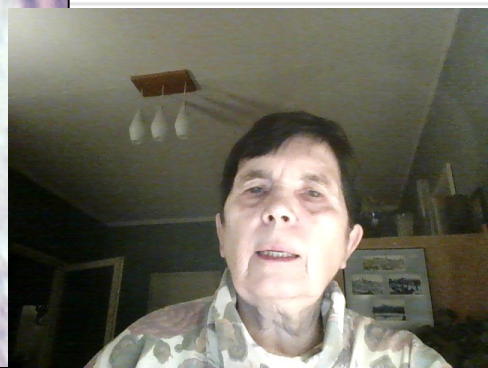


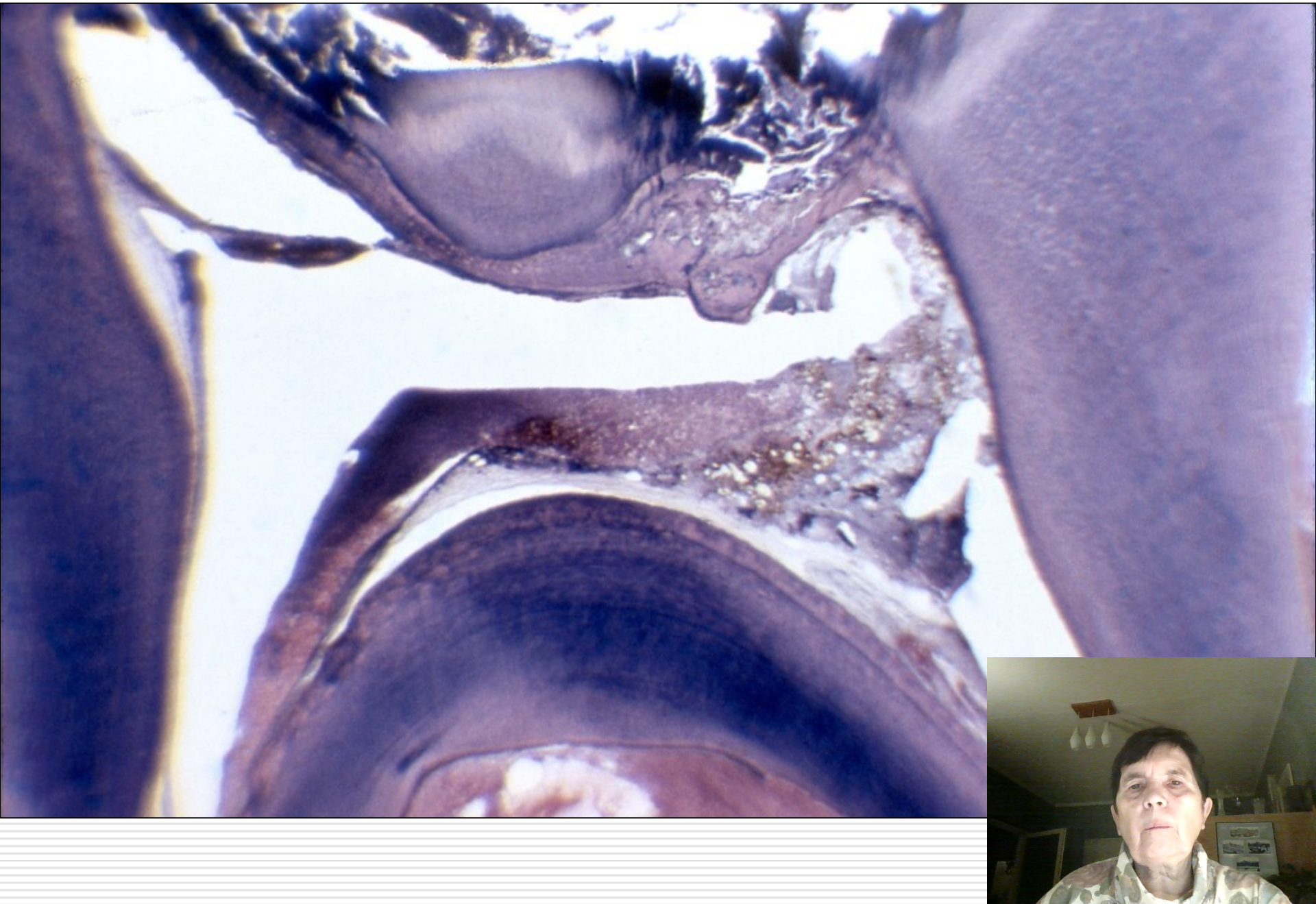
Necrosis

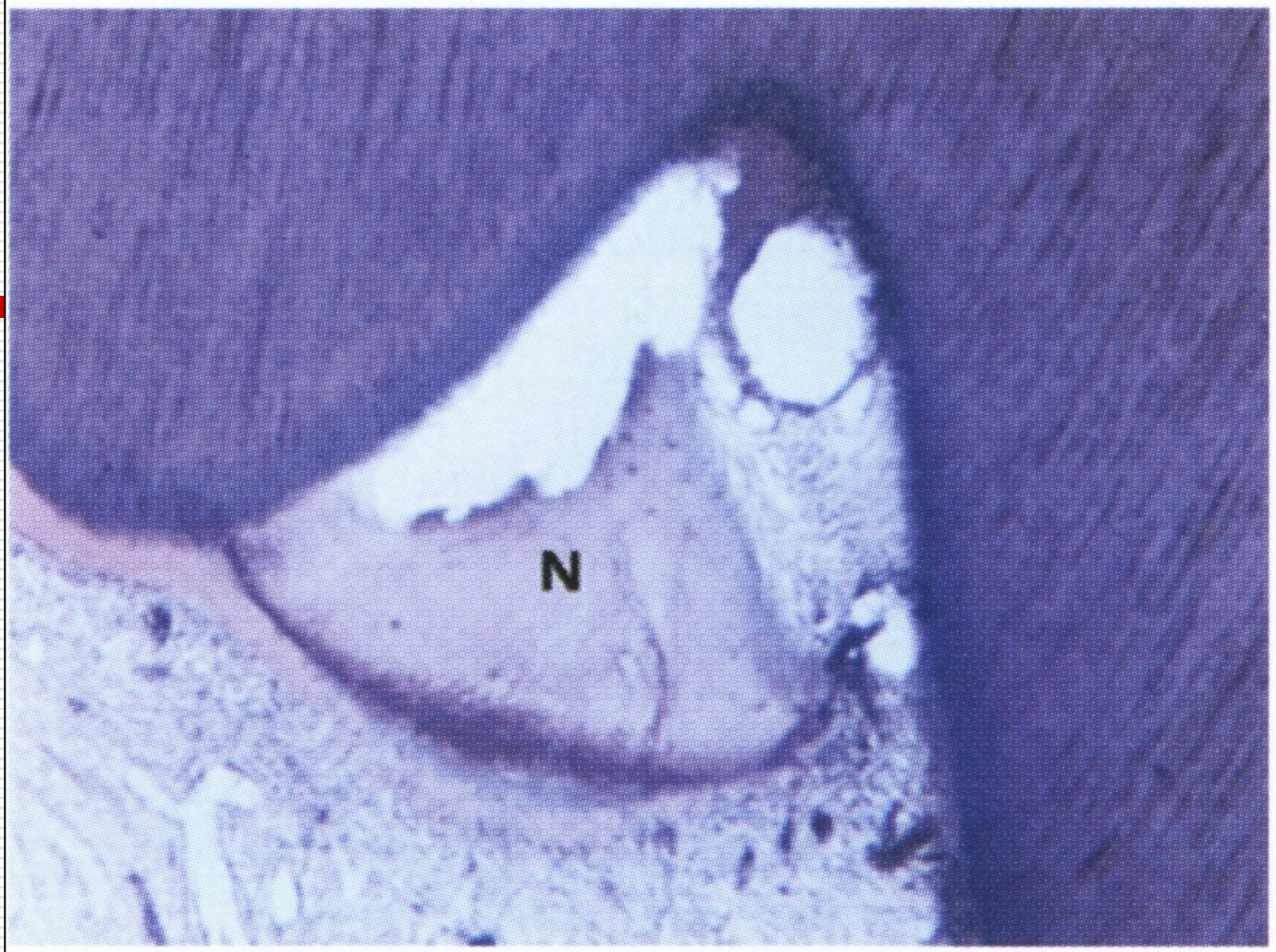
- Is a sequel of acute and chronic inflammation
- liquefaction necrosis
- coagulation necrosis
- products of necrosis - toxic to the periapical tissues =
- inflammatory response, abscess formation, without the presence of microbes
- anaerobic bacteria - decomposition proteins



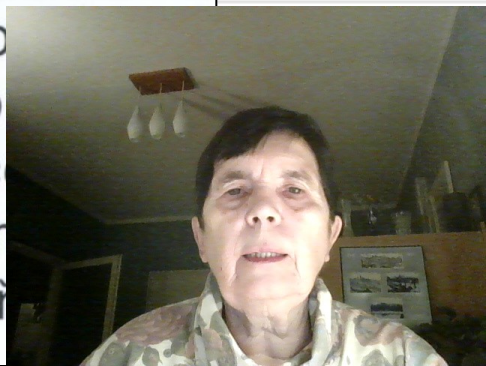








Obr. 2.10 Demarkující se nekróza (N) rohu p
čtyři týdny po preparaci mladého zubu turb
značně omezeným chlazením. Demarkac
vytvořeným málo mineralizovaným dentiner
čí o dobré obranyschopnosti mladé zubní dř



Gangrene

- Intermediate proteolytic products = Foul odour
  - indol, scatol
  - putrescin, cadaverin
  - indican
- End products
  - hydrogen sulphide
  - ammonia
- Endotoxins from bacteria
- Foreign bacterial proteins