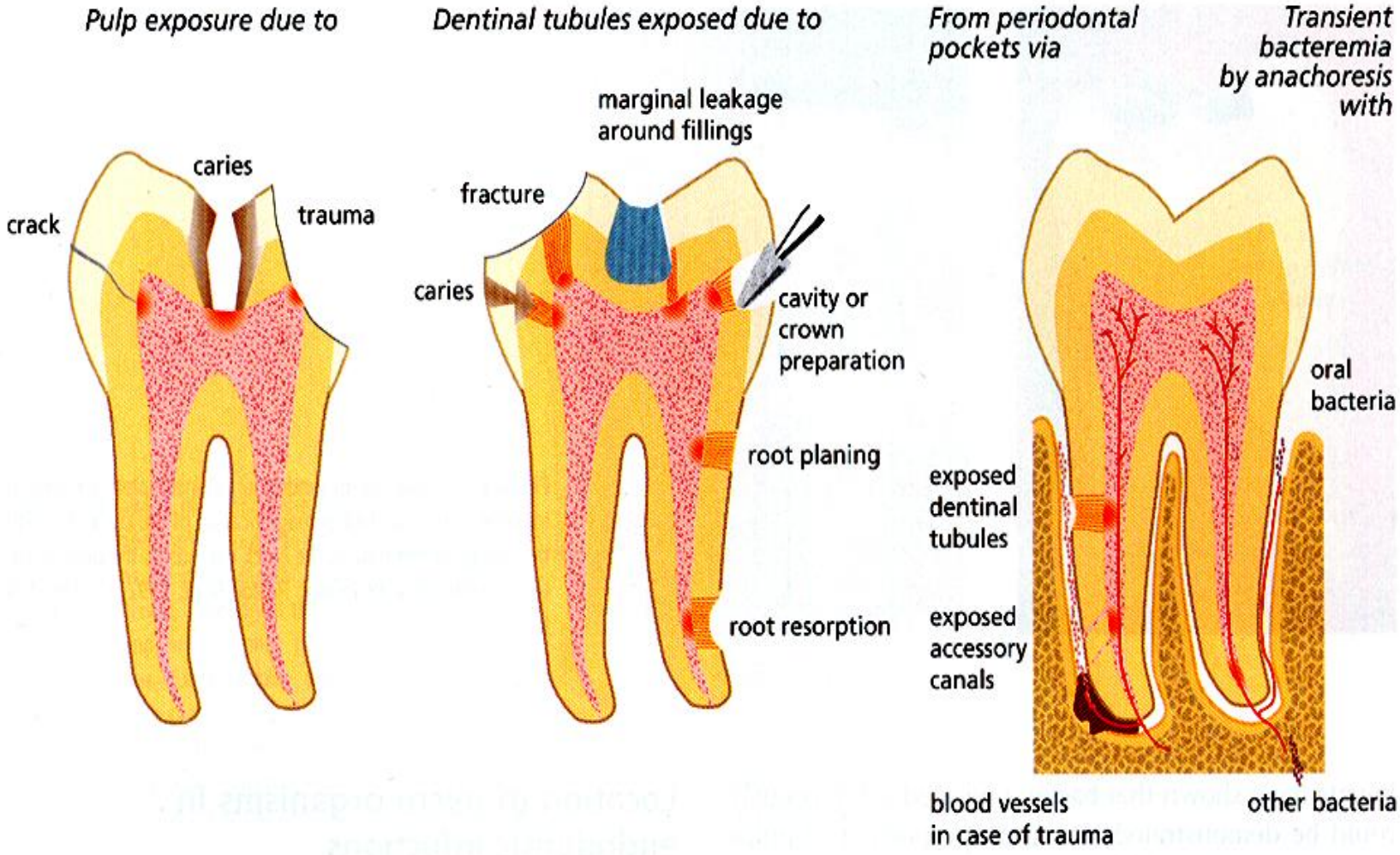
Introduksjon: Pulpitt og apikal periodontitt: etiologi og patogenese; mikrobiologi

Dag Ørstavik

UiO 2012 08 29

Pathogenesis

- Infection
  - Caries
  - Leakage
  - Cracks, fractures
  - Dentin tubule exposure
  - Pulpal tissue
  - Pulp ramifications
  - Dentin tubules
  - Cementum
  - Extraradicular infection
- Tissue response
  - Dentin sclerosis
  - Tubule obliteration
  - Tertiary dentin
  - Pulpitis (productive, degenerating and pyogenic)
  - Pulpoperiodontitis
  - Apical periodontitis
  - Resorptive processes
  - Bone sclerosis



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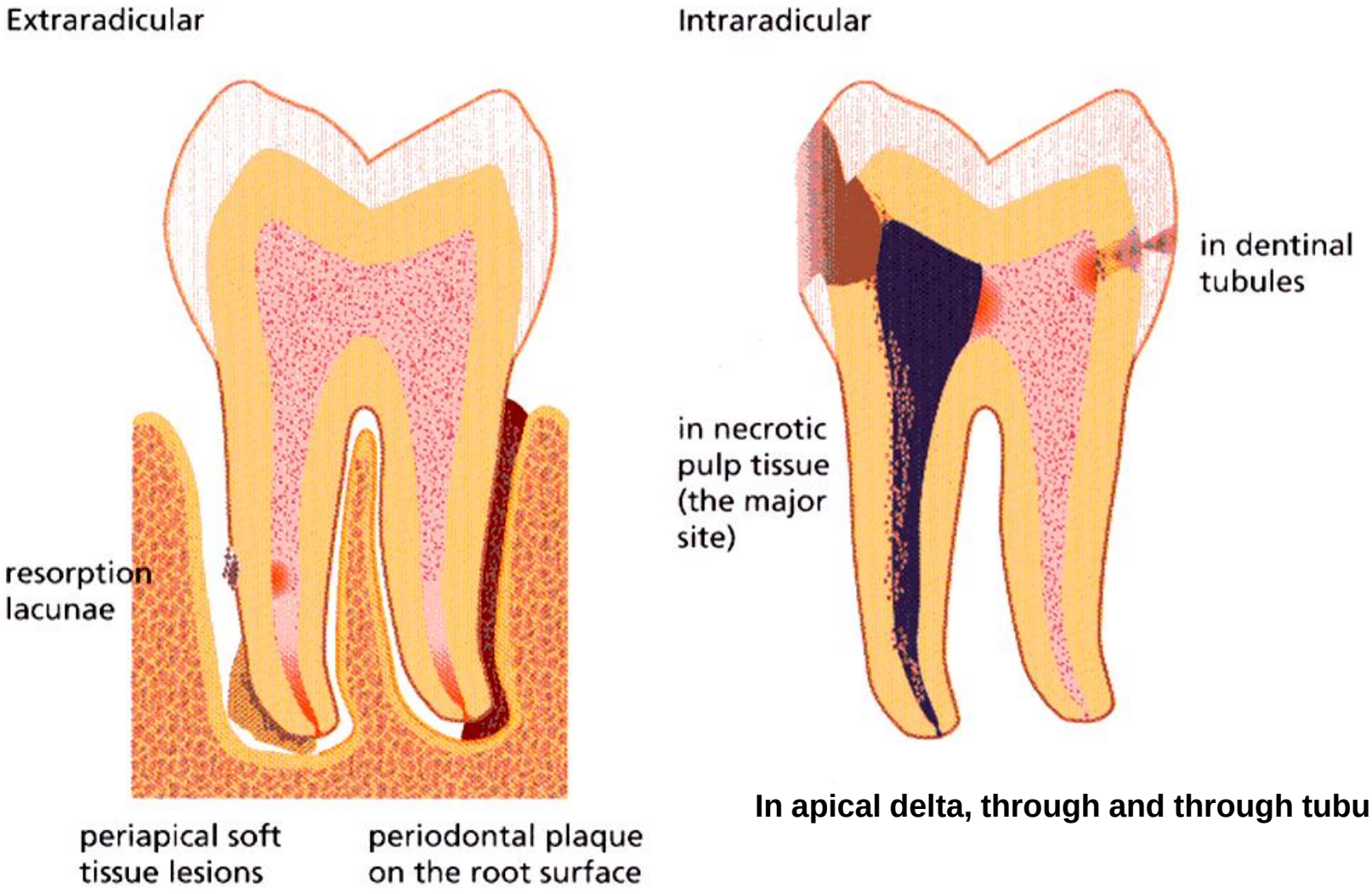
Location of microorganisms in endodontic infections



In apical delta, through and through tubules

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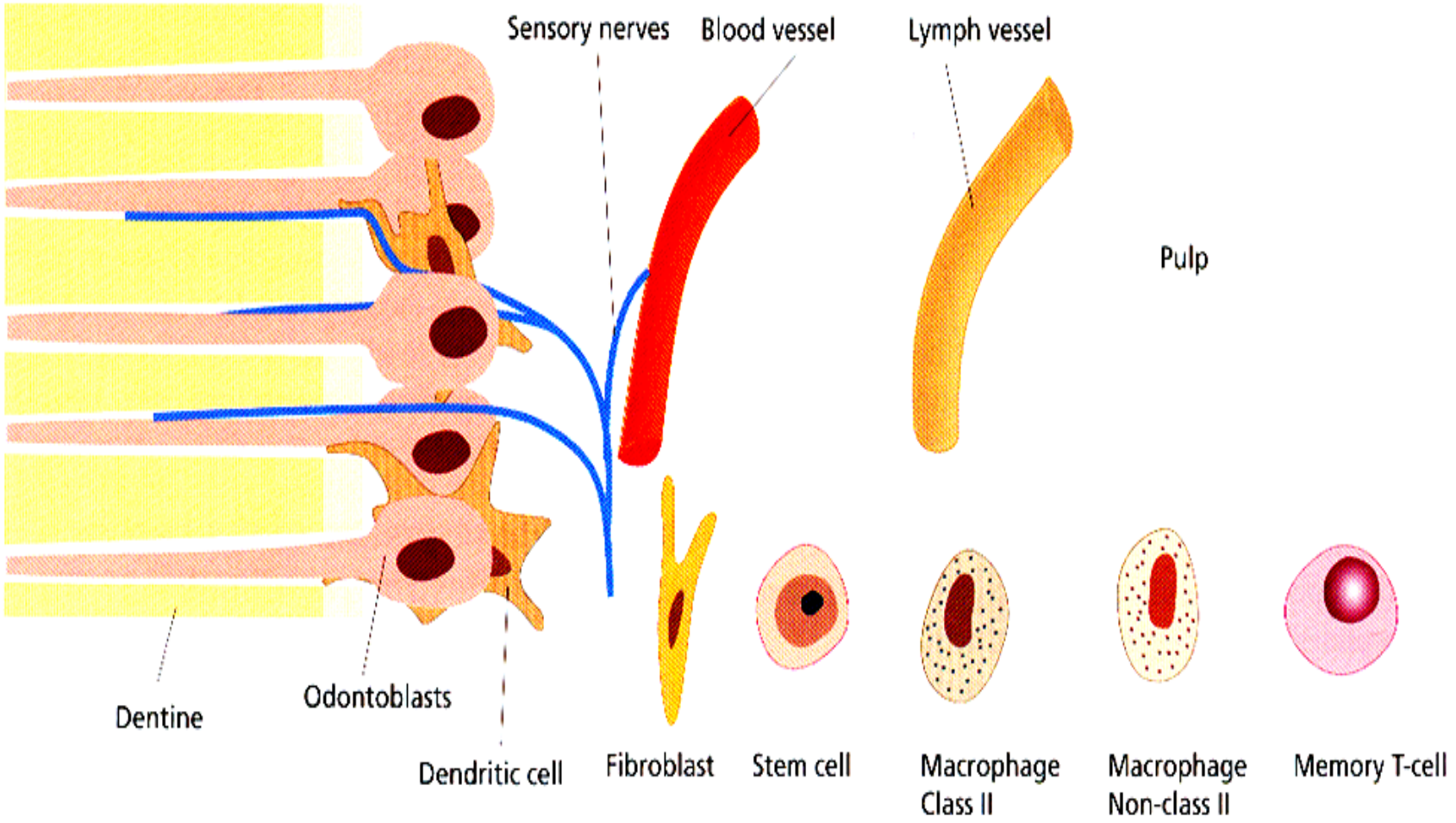

Fig. 3.11 Constituents of primary significance in the defense of the pulp against foreign substances, including bacterial elements, make up the innate 'first line of defense'.

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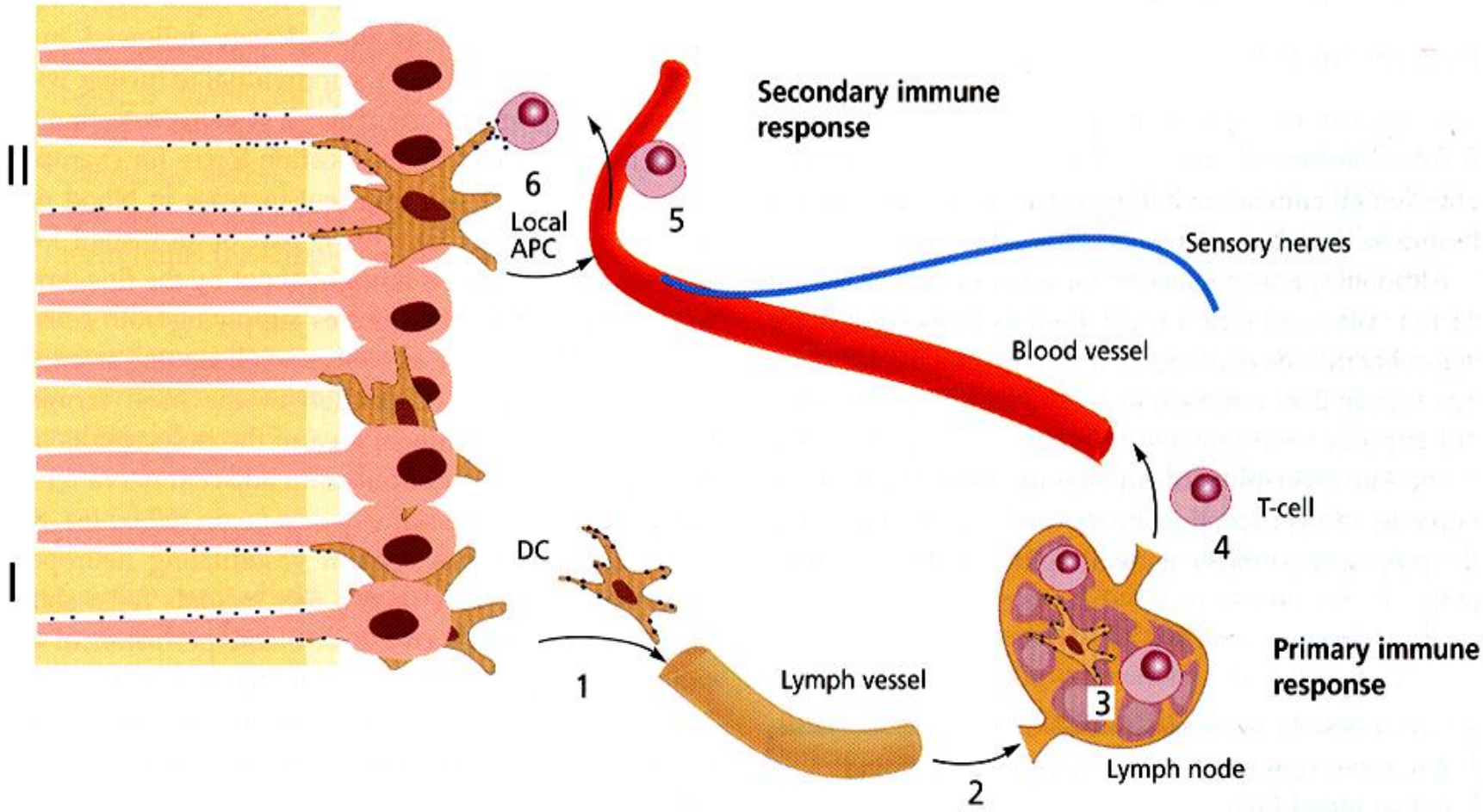

Fig. 3.12 Antigen-specific T-cells are developed in the pulp following primary (I) and secondary (II) antigen exposures along dentinal tubules. Dendritic cells (1 in figure) capture protein antigen for processing to peptide fragments and carry (2) and present peptide fragments in the context of the Class II molecules on their cell surface to naïve T-cells in the regional lymph nodes (3: primary immune response). Following clonal expansion, these cells enter the circulation (4 in figure). Following their patrolling of tissues as memory T-cells, they may participate in secondary immune responses at local sites, e.g. in the pulp (5 in figure), if exposed to the appropriate antigen by local APC (6 in figure). This route constitutes adaptive pathogen-specific immunity.

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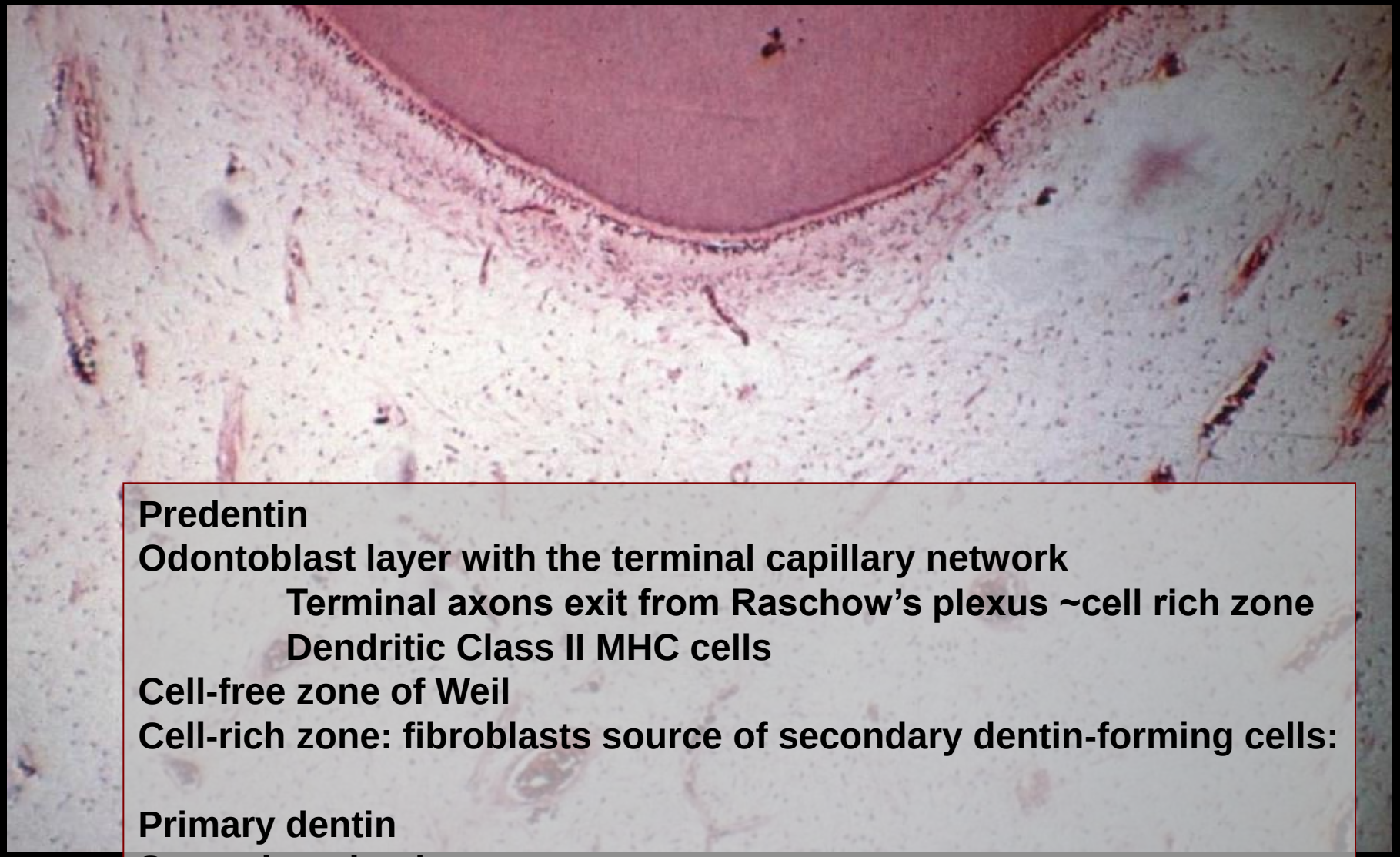

Predentin
Odontoblast layer with the terminal capillary network
Terminal axons exit from Raschow's plexus ~cell rich zone
Dendritic Class II MHC cells
Cell-free zone of Weil
Cell-rich zone: fibroblasts source of secondary dentin-forming cells:

Primary dentin
Secondary dentin
Tertiary dentin: reactionary and reparative

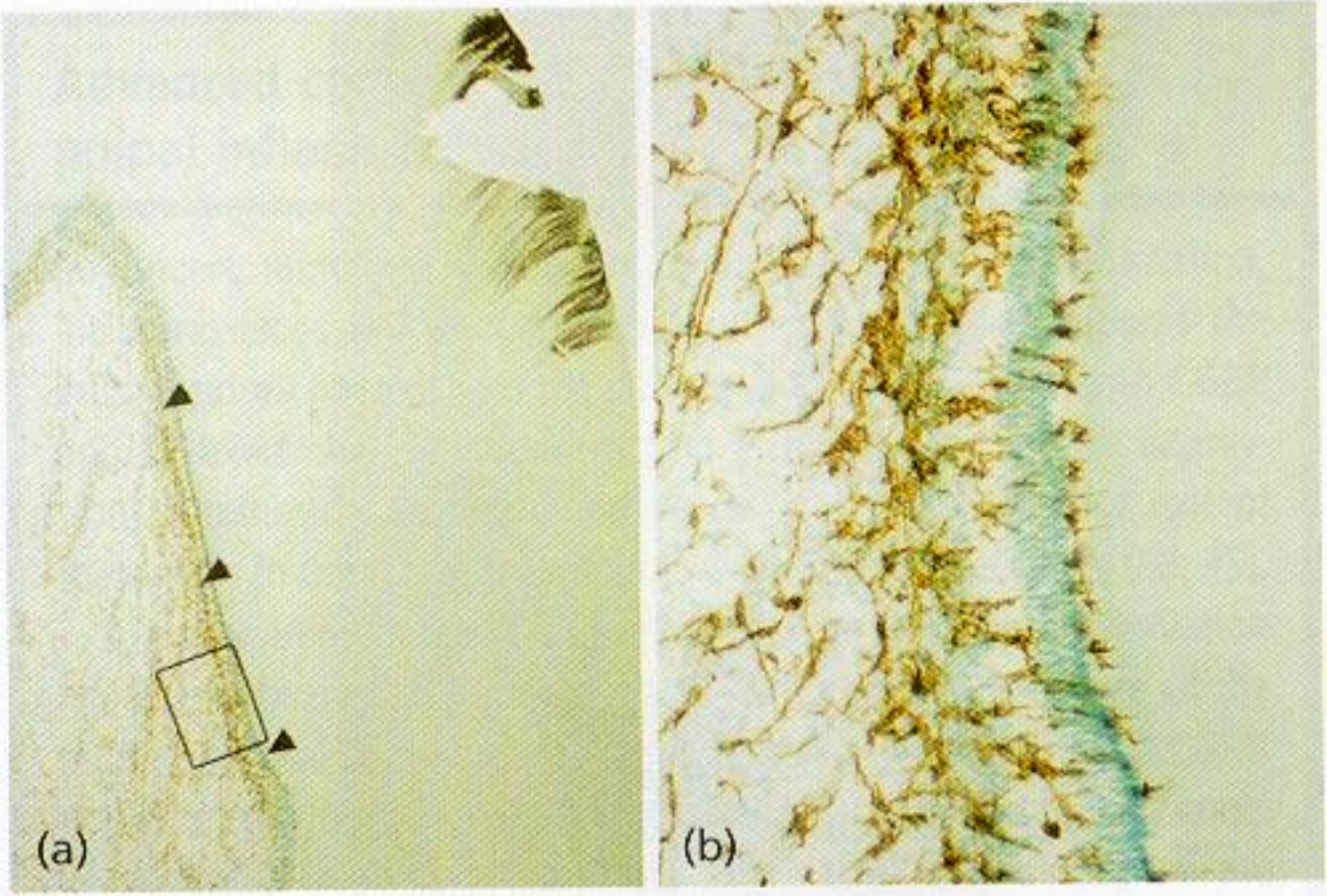

Fig. 3.19 (a) Numerous Class II molecule-expressing cells (stained brown) accumulated underneath a superficial caries lesion, extending into the dentine of a human tooth (dark stain, upper right). (b) Extension of dendrites into the tubules. (Courtesy of Dr T. Okiji.)

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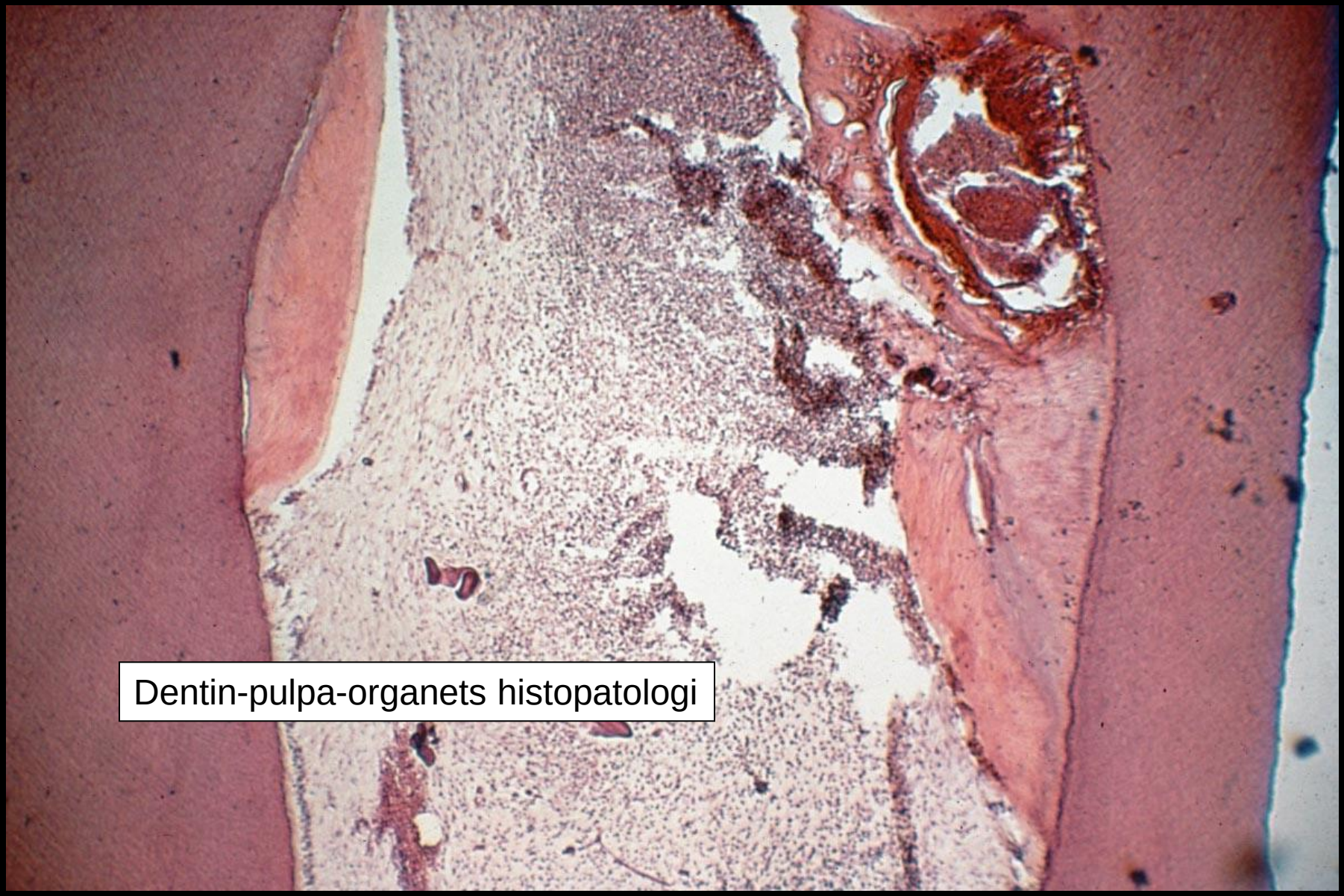
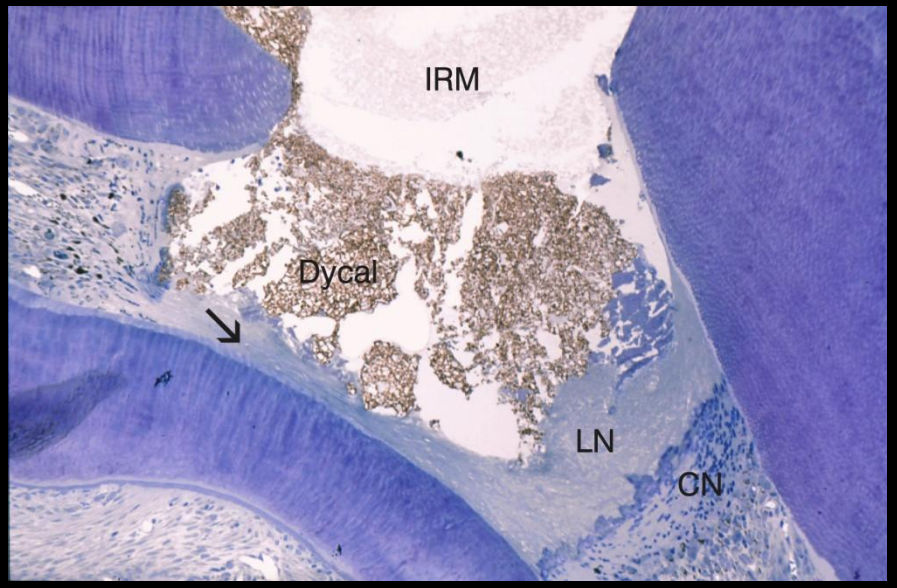
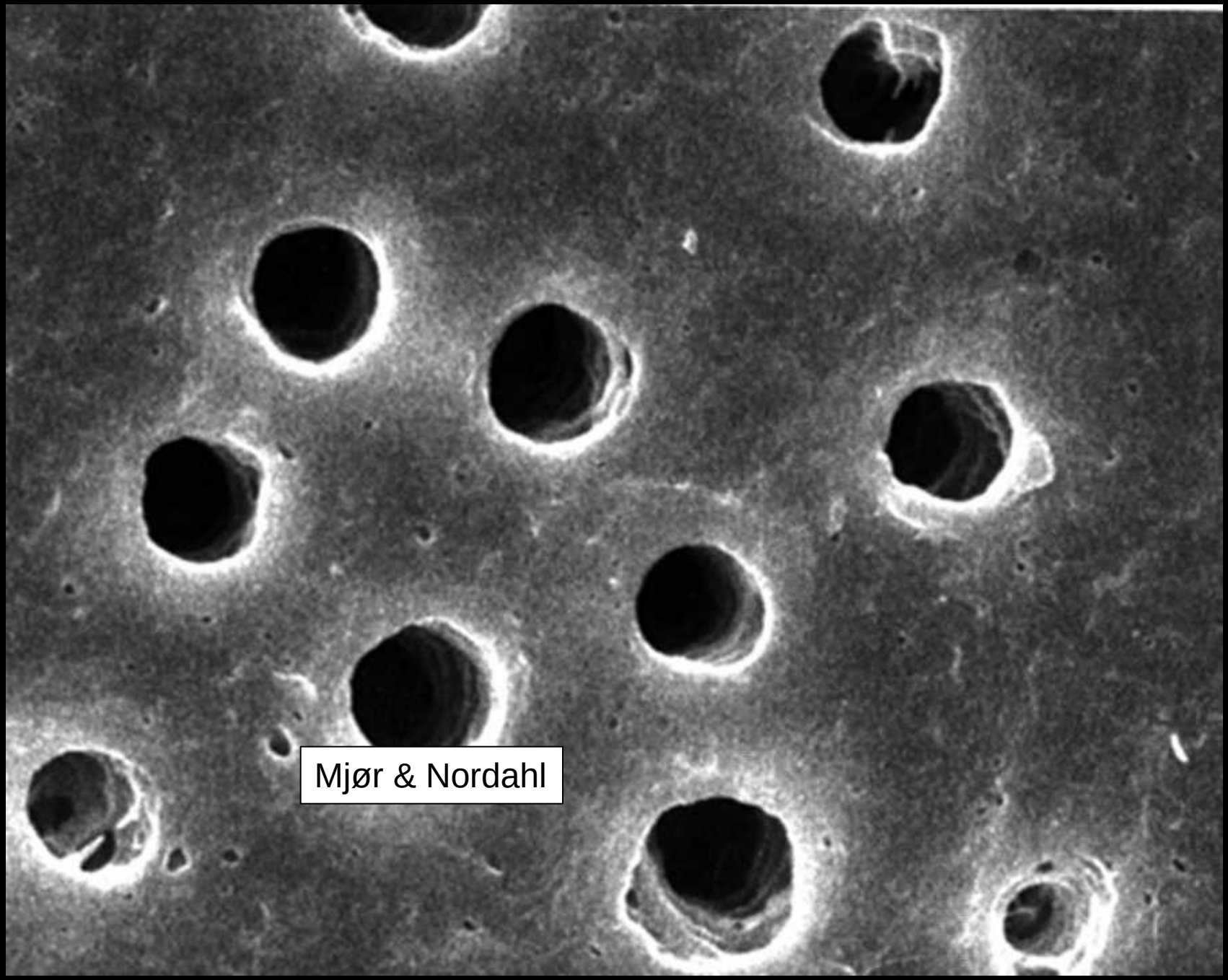
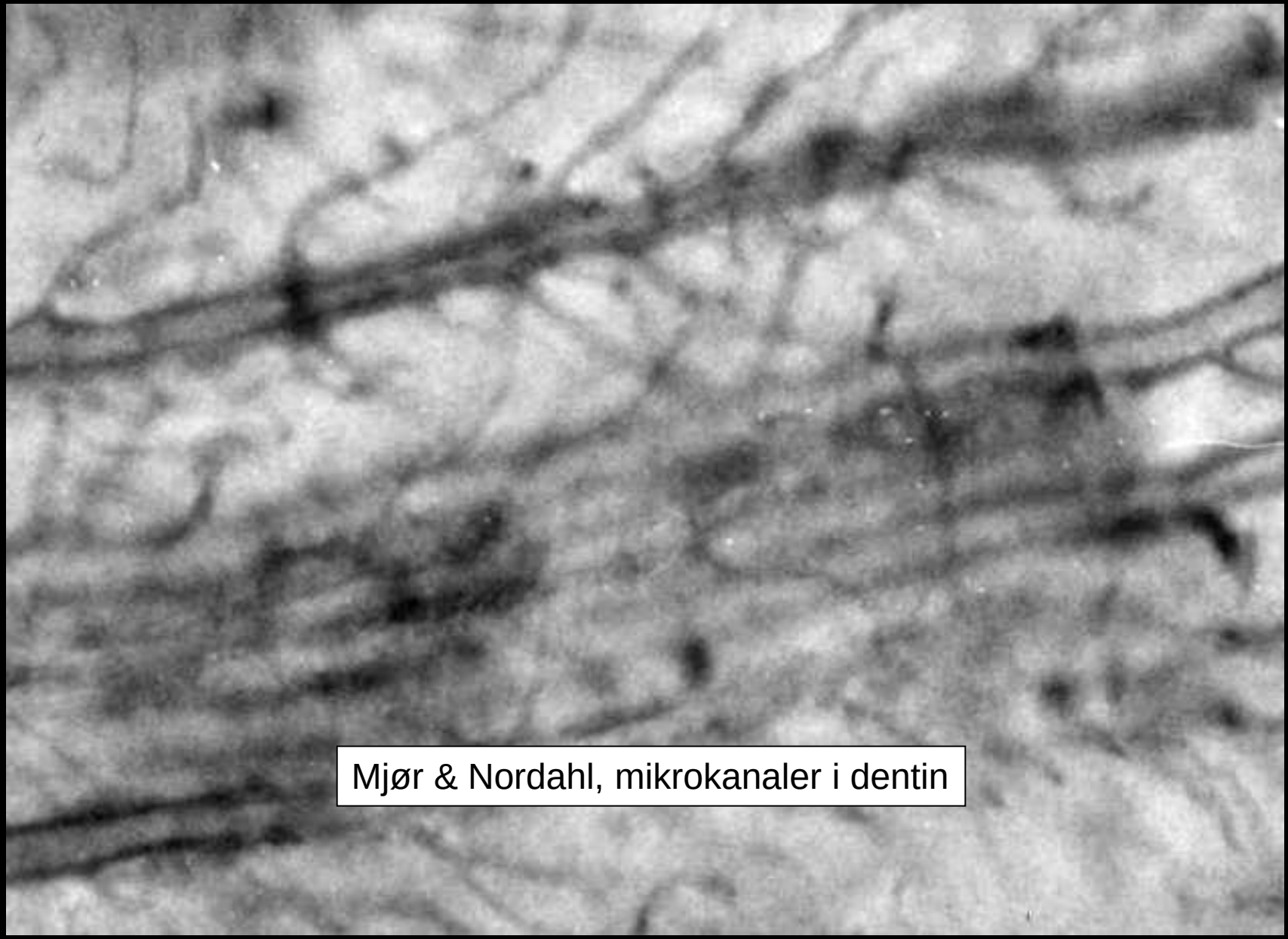

Dentin-pulpa-organets histopatologi

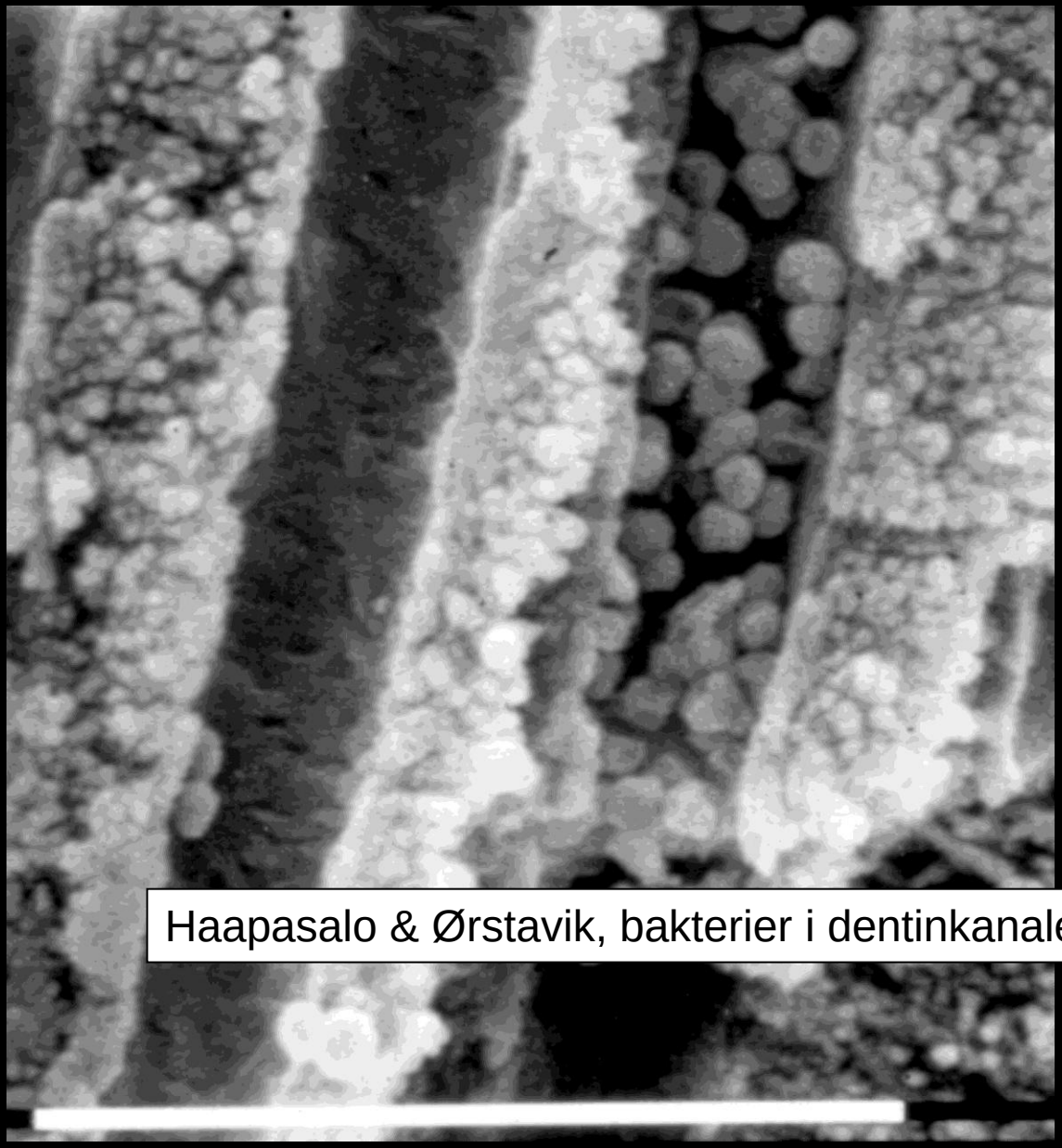


Hørsted-Bindslev & Løvschall 2004)

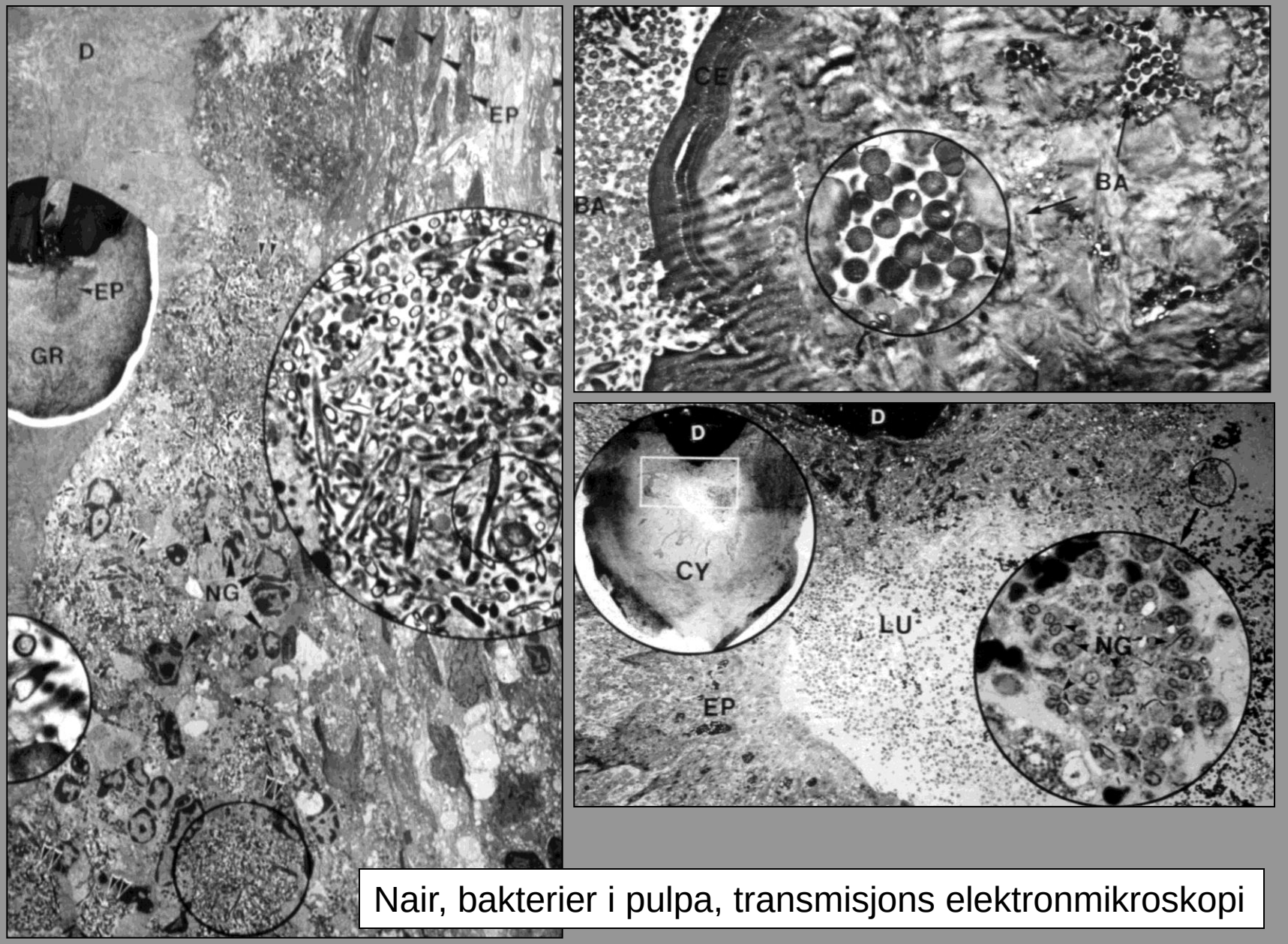
Dentin-pulpa-organets evne til barrieredannelse


Mjør & Nordahl


Mjør & Nordahl, mikrokanaler i dentin



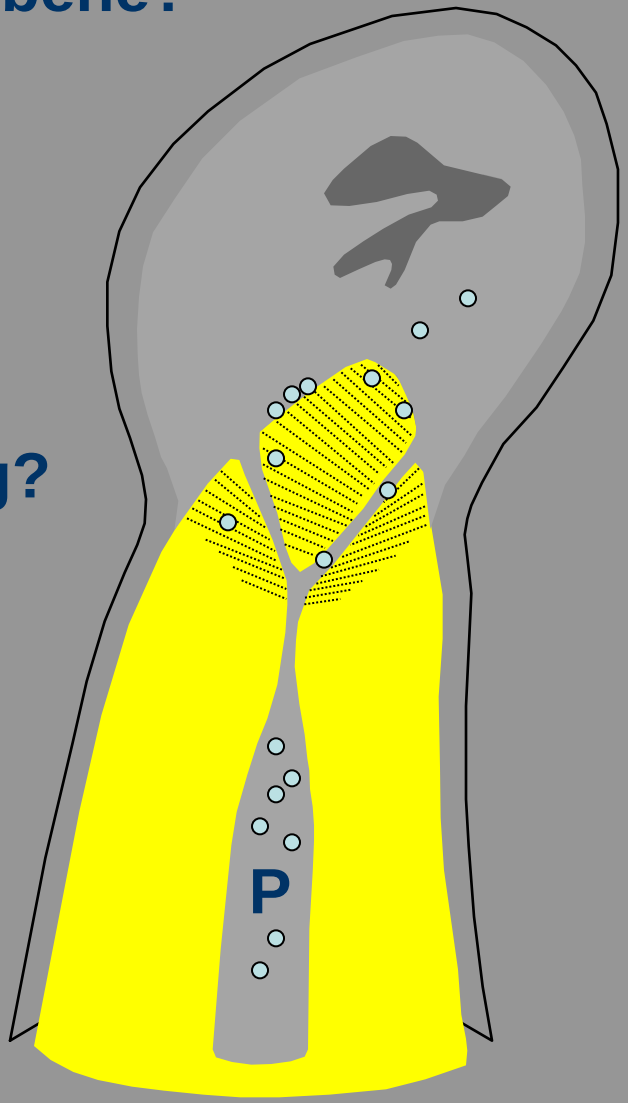
Haapasalo & Ørstavik, bakterier i dentinkanaler

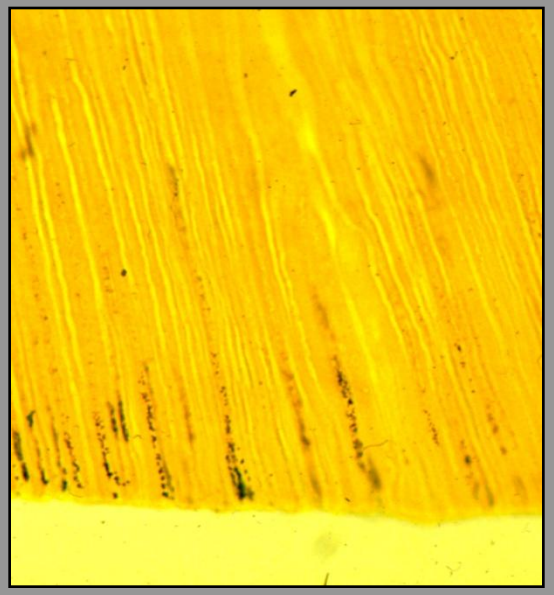


Nair, bakterier i pulpa, transmisjons elektronmikroskopi

Hvor er mikrobene?

Hvordan affiserer de vevene omkring?





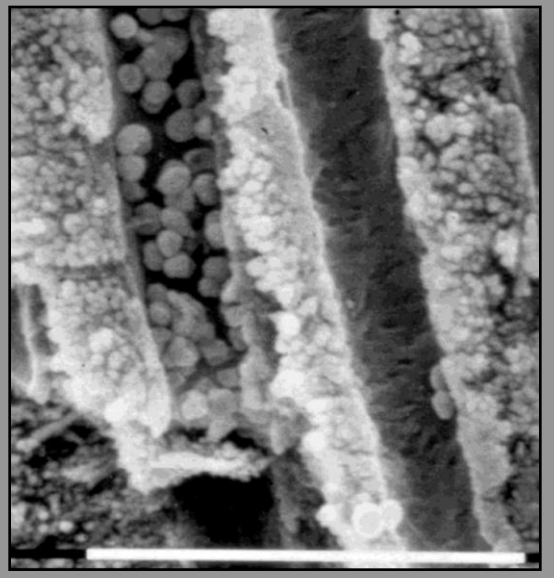
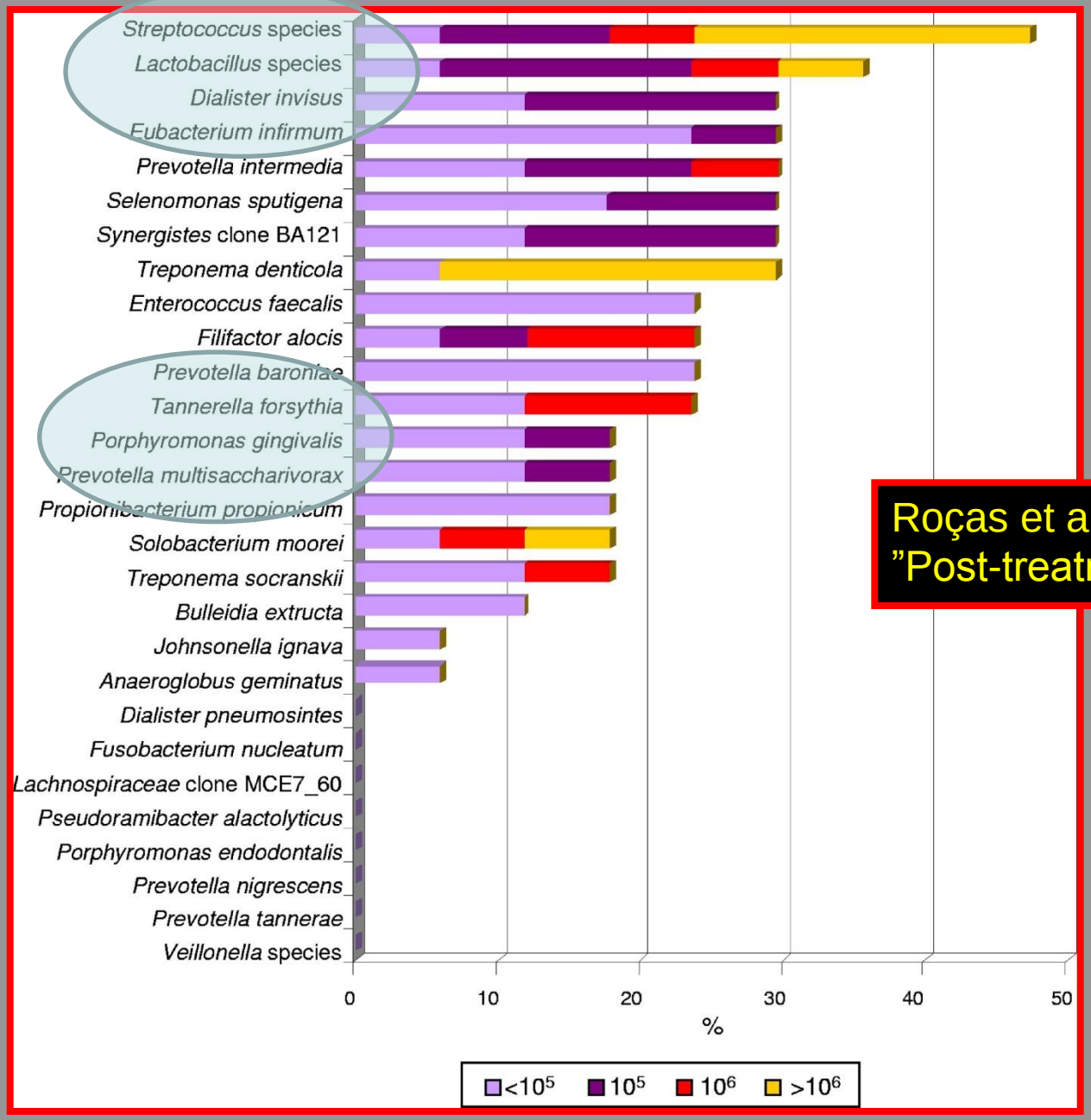


TABLE 2. Classification of bacterial strains isolated from initial, postinstrumentation, and postmedication samples (n = 288)

	IS		PIS		PMS	
	One Visit	Two Visits	One Visit	Two Visits	One Visit	Two Visits
Anaerobs						
Fusiform rods	22	19	7	6	3	2
Prevotella spp.	34	29	8	7	5	1
Porphyromonas spp.	5	3	1		1	
Campylobacter spp.	1	1		1		
Capnocytophaga spp.	2		1			
Actinomyces spp.	1	1		1	1	1
Anaerobic lactobacilli	1		1		1	
Bifidobacterium sp.		1				
Other G+ anaerobic rods	20	20	9	10	4	4
Veillonella spp.		1	1			
Peptostreptococcus spp.	20	15	10	7	3	1
No. of anaerobs	106	90	38	32	18	9
Facultatives						
Coliform rods		3				
Klebsiella spp.	1		1			
Proteus spp.	1		1		1	
Pseudomonas spp.	1		1			
Other G− aerobic rods		1				
Lactobacillus spp.	6	15	2	5		4
Other G+ aerobic rods	1	2	1	2	1	
G+ aerobic spore-forming rods	3		1			1
Streptococcus spp. (NPSP)*	17	13	6	7	5	2
Streptococcus spp. (PSP)†	13	6	5	2		4
Enterococcus spp.	4	4	1	3	1	3
Staphylococcus spp.	5	3	5	5	3	7
Micrococcus spp.	1	1	1			
No. of facultatives	53	48	25	24	11	21
Total no. of strains	159	138	63	56	29	30
Semiquantification						
Very sparse growth	13	12	19	23	11	11
Sparse growth	25	31	28	25	11	6
Moderate growth	73	68	11	4	6	11
Heavy growth	47	26	4	4	0	2
Very heavy growth	1	1	1	0	1	0

* Nonpolysaccaride-producing species.
† Polysaccaride-producing species.
IS = initial sample; PIS = postinstrumentation sample; PMS = postmedication sample.
Results of the semiquantification also are displayed.

Kvist et al 2004



Roças et al., 2008:
”Post-treatment disease”

Core concept 8.3 Ecological determinants of the endodontic microflora

- Adhesion to root canal tissues
- Coaggregation of populations
- Low oxygen concentration
- Reduced redox potential
- Nutrition:
  - —necrotic pulp tissue
  - —tissue fluid and exudate
  - —microbial food chains
- Microbial interactions:
  - —synergistic
  - —antagonistic
- Endodontic treatment:
  - —mechanical debridement
  - —antimicrobial agents

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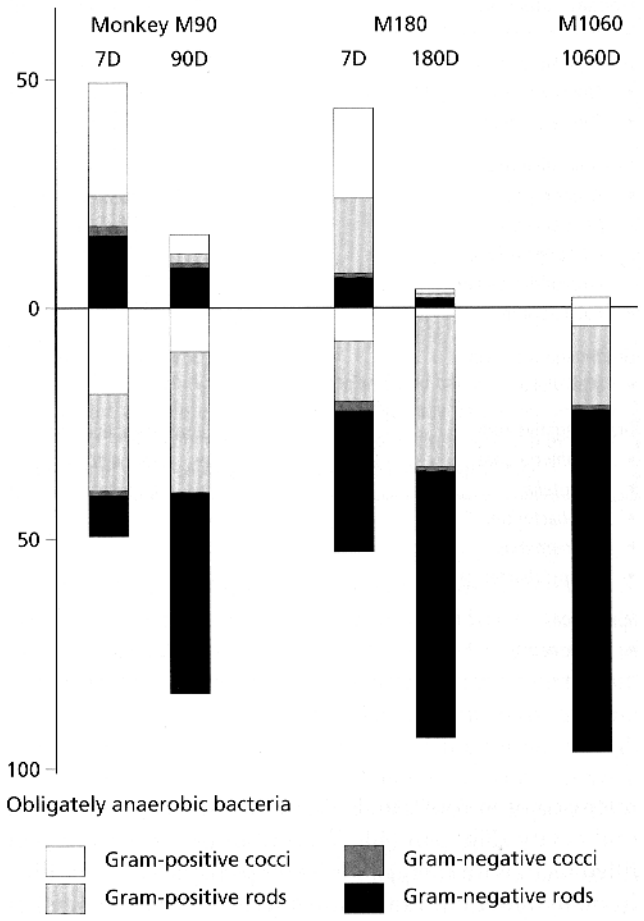


Fig. 8.10 Mean percentage of obligately and facultatively anaerobic bacteria in root canals in three experimental monkeys (M90, M180 and M1060) at different times (7, 90, 180 and 1060 days) after sealing the canals following mechanical removal of the pulp and exposure to the oral microflora for 1 week. From: Fabricius L, *et al. Scand. J. Dent. Res.* 1982; 90: 134–44 (20).

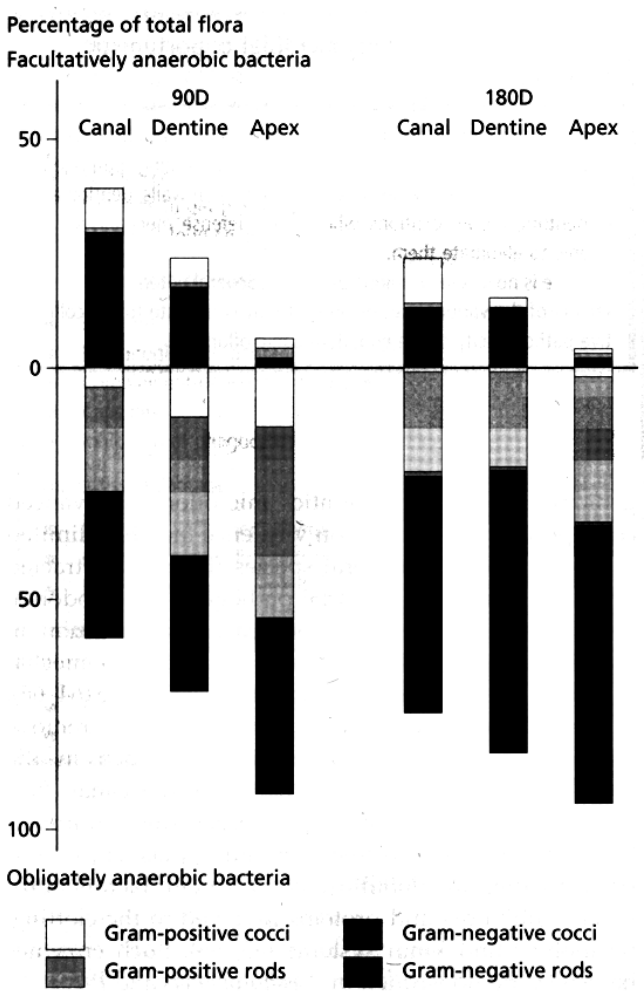
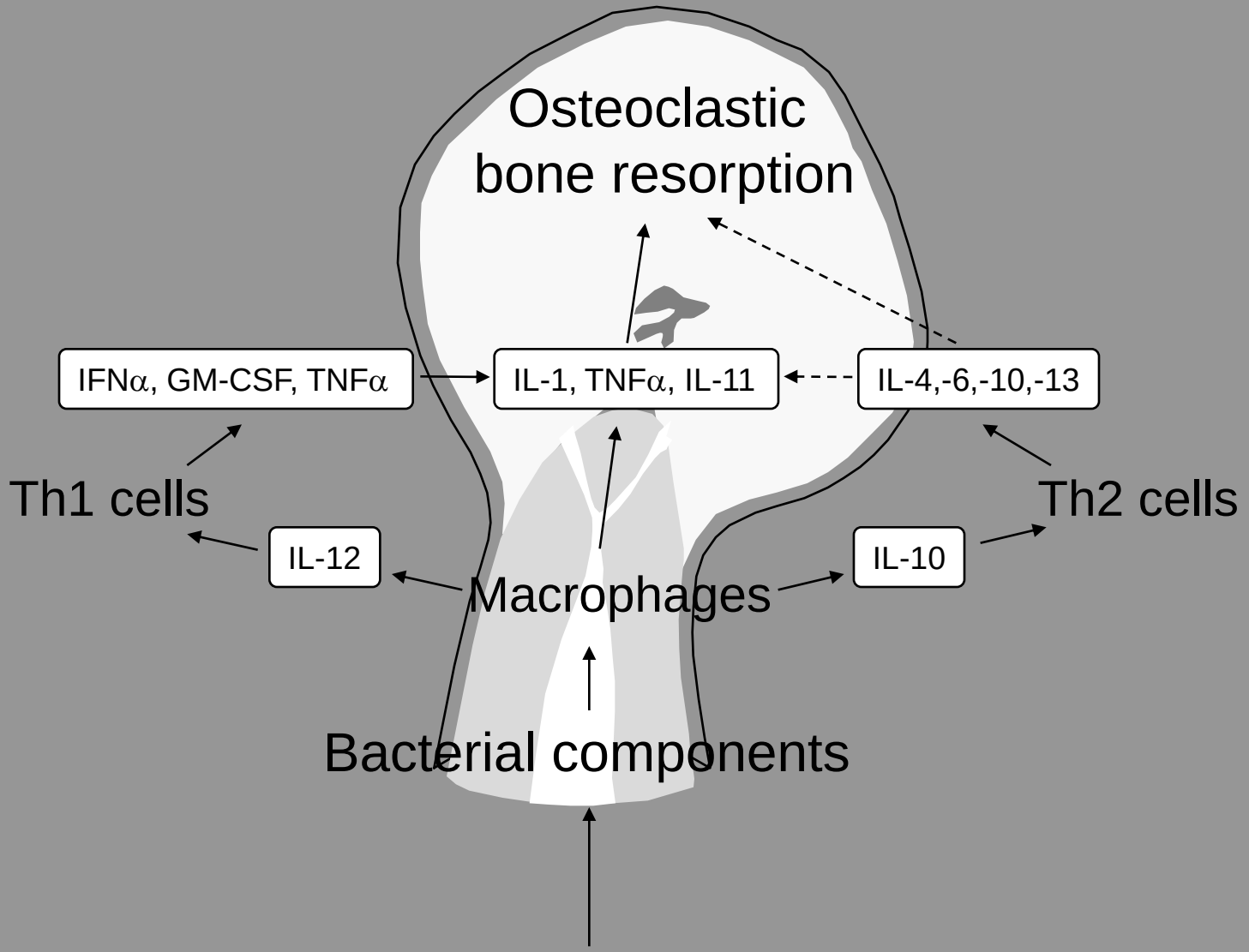
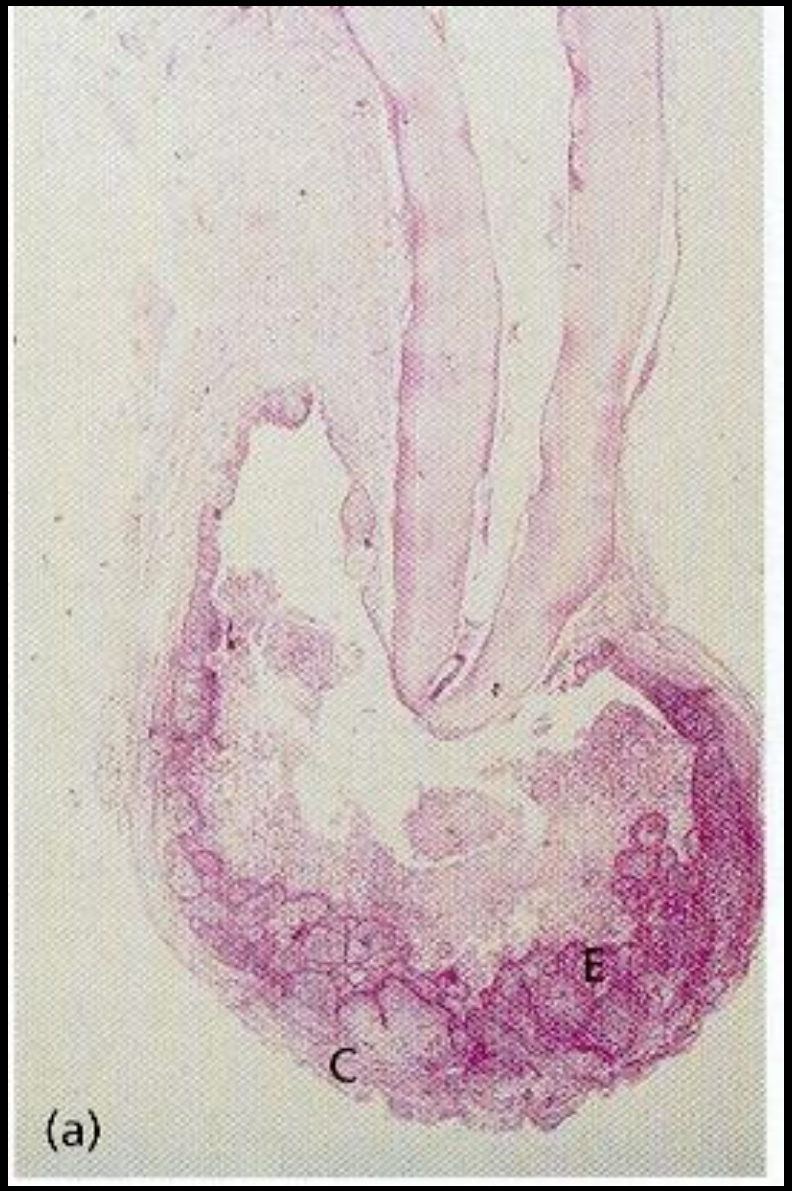


Fig. 8.11 Mean percentage of obligately and facultatively anaerobic bacteria from different parts of the root canal system in final samples in experimental monkeys (M90 and M180) 90 and 180 days after sealing the canals following mechanical removal of the pulp and exposure to the oral microflora for 1 week. From: Fabricius L, *et al. Scand. J. Dent. Res.* 1982; 90: 134–44 (20).

Stashenko 1998, prinsipper for apikale immunreaksjoner





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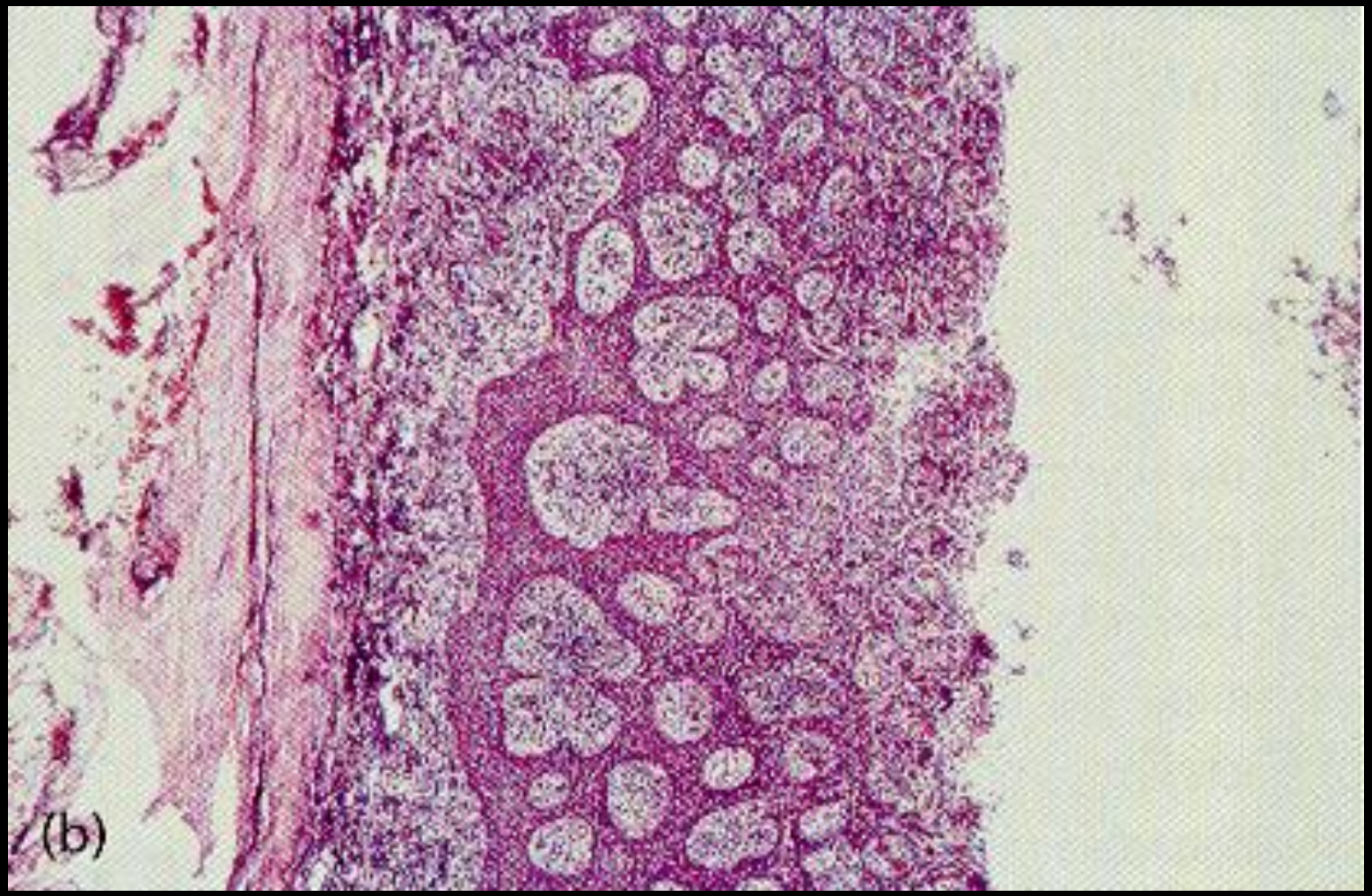

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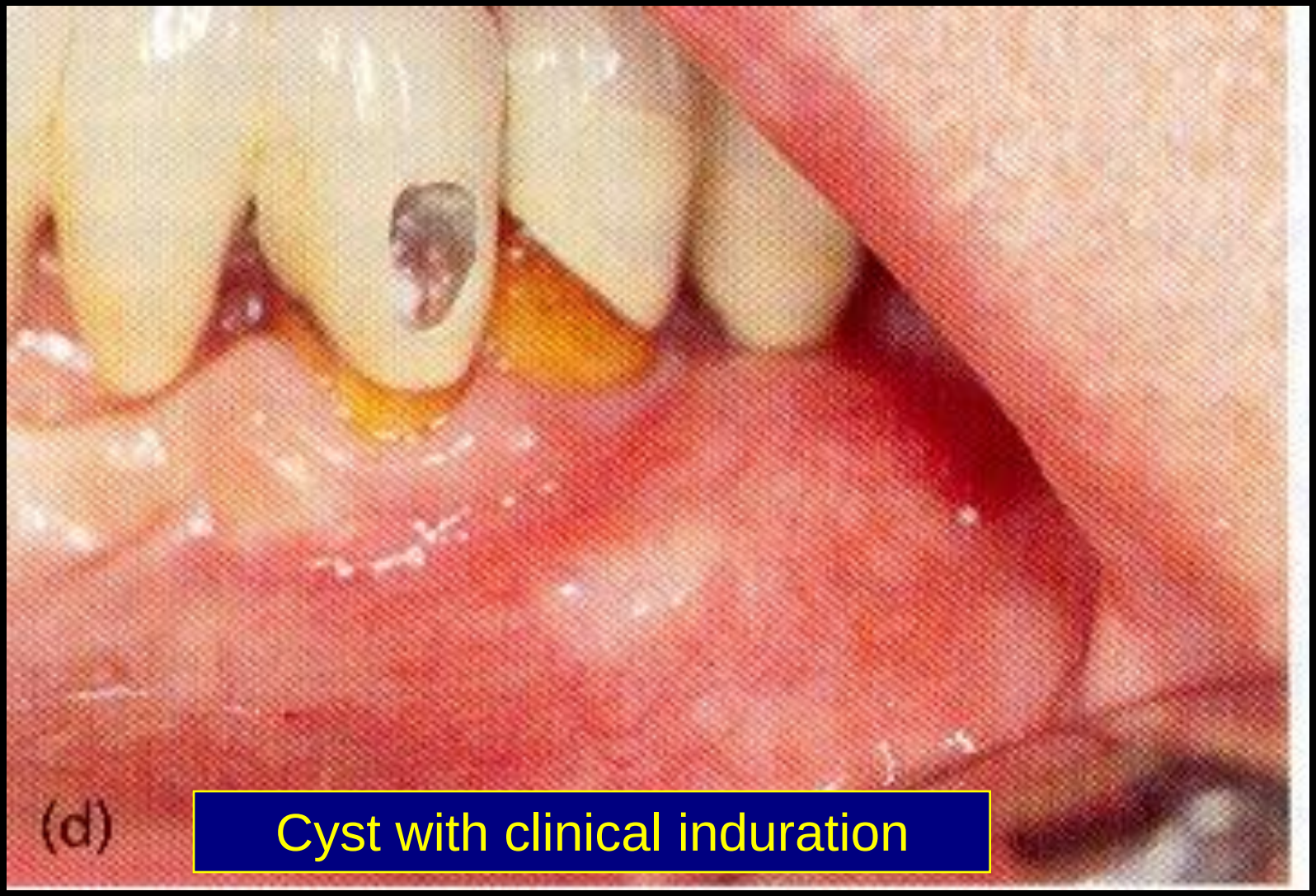
A skull of a woman from a heathen grave at Hólaskógi in Thjórsárdal. It is probable that a dental infection in the upper jaw was the cause of her death.

Photo and text of display at
Iceland's National Museum in Reykjavik



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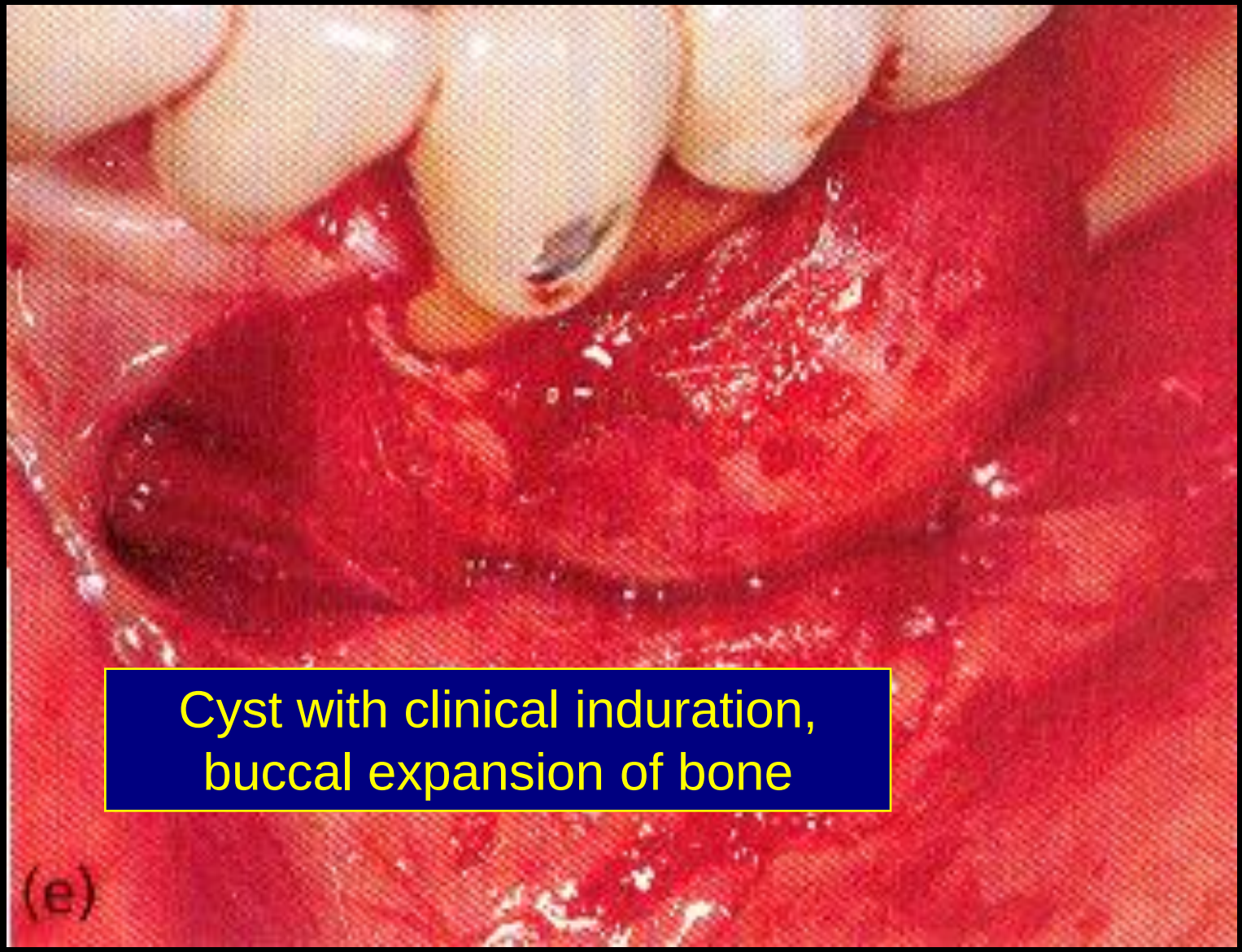

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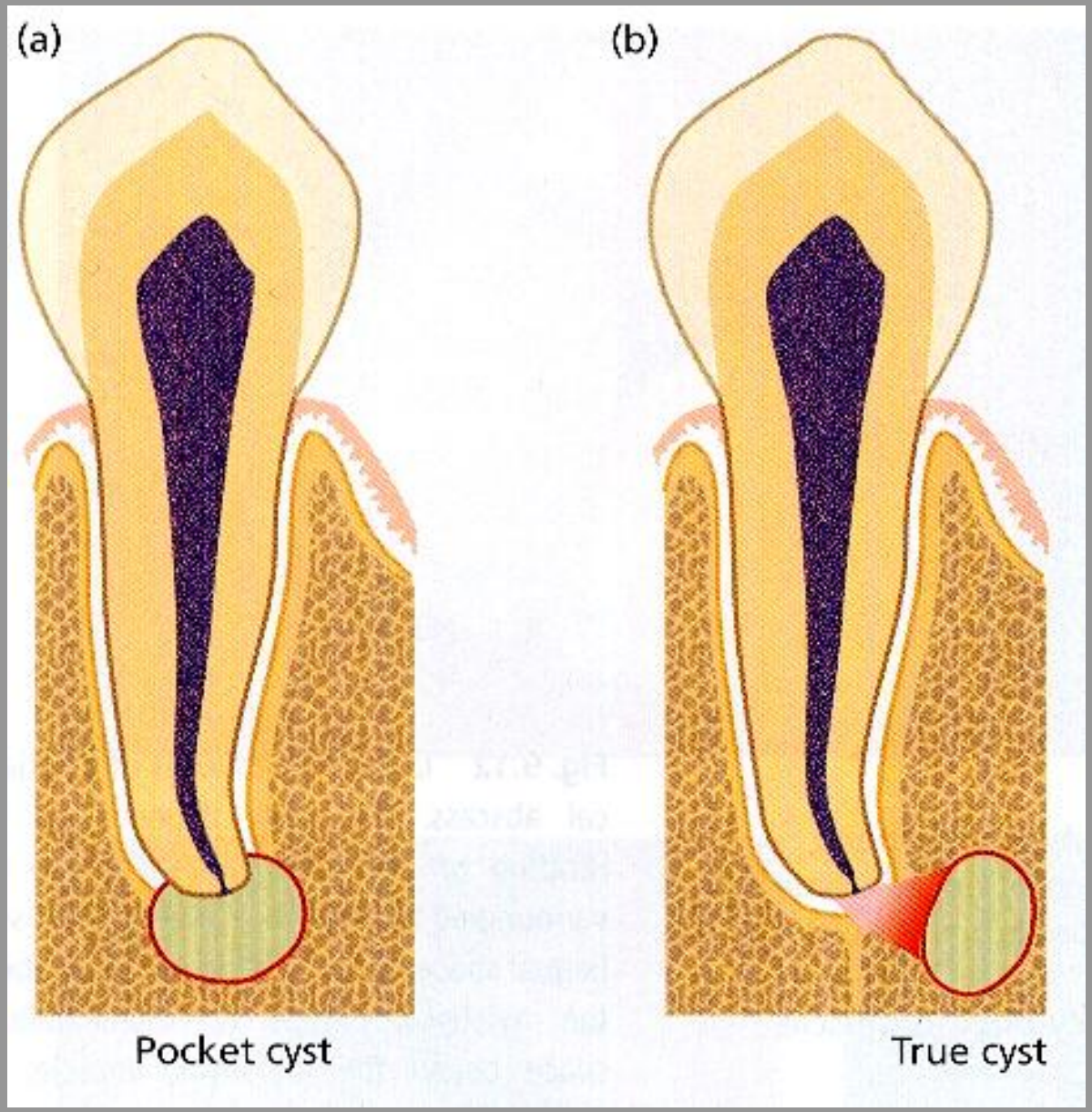

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Key literature 9.2

In a classic experiment in primates, Valderhaug (49) removed the pulp tissue in teeth and left the root canals open to the oral environment for up to 360 days.

Although initially severe inflammatory lesions, including migration of epithelial cells, were seen in the apical area, it took more than 200 days before cyst formation developed. The observation suggests that the inflammatory process in the apical periodontium is capable of inducing proliferation of the epithelial rests of Malassez and that radicular cysts may result if apical periodontitis is left untreated for a long period of time.

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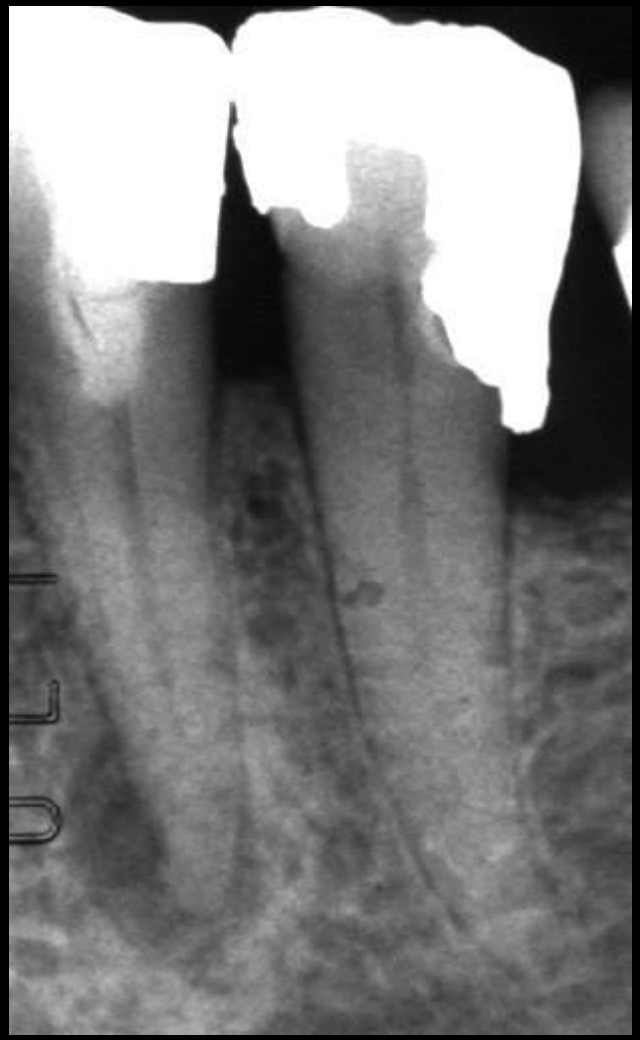

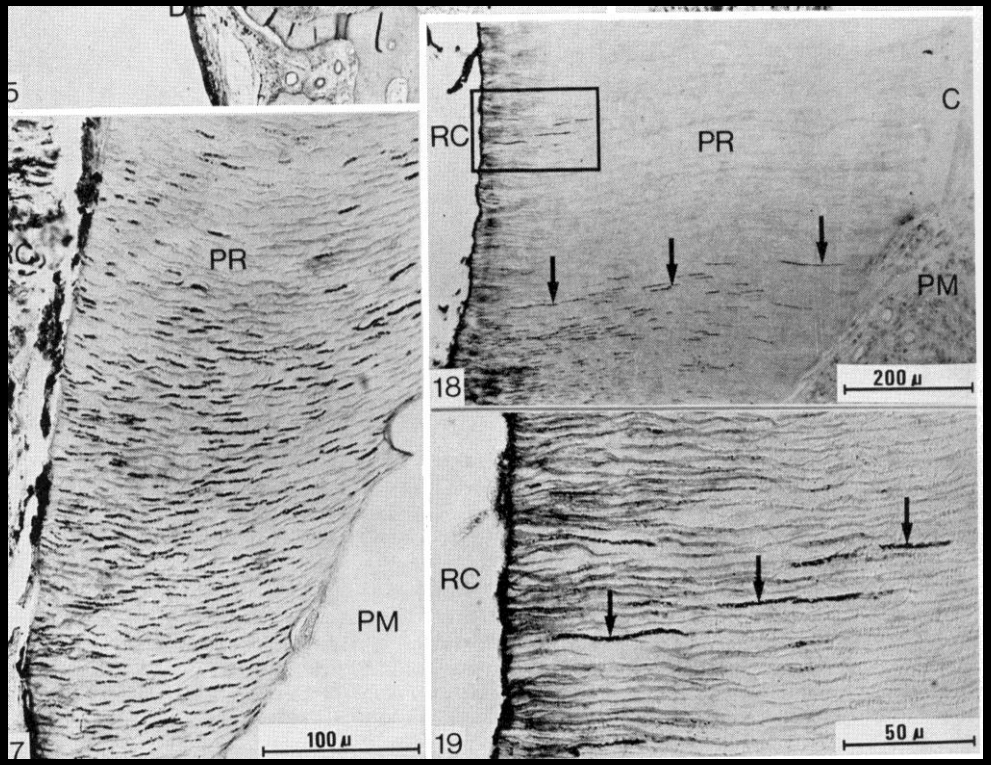
PR
PM
100 µ
RC
PR
C
PM
200 µ
RC
50 µ

Advanced concept 9.2 Pathogenesis and growth of radicular cysts

The factors that initiate proliferation of the epithelial rests of Malassez are not well known. Both bacterial endotoxins as well as cytokines of inflammatory cells have been implicated (26). There is also evidence that epidermal growth factors are involved in this process (22, 23, 47). Once started, epithelial proliferation will continue for as long as stimulating factors are present.

The mechanism behind the development of the cyst cavity has been the focus of much speculation. Two hypotheses still prevail (32). One states that when the epithelial mass increases in size, the central cells will undergo degeneration and necrosis due to lack of nutritional supply. The necrotic material in turn attracts neutrophils, which, together with tissue exudate, result in the formation of microcavities that eventually coalesce to form a radicular cyst. Another theory is built on the assumption that epithelial cells grow to form an epithelial lining on the inner aspect of an abscess cavity.

Also, the exact mechanism for the subsequent slow increase in the size of the radicular cysts has not received its final explanation. Some believe that increased osmotic pressure in the cyst cavity is a key element (38). Increased osmosis leading to the passage of fluid from the surrounding tissue into the cyst lumen is likely to occur due to breakdown of epithelial and inflammatory cells. Furthermore, cyst expansion is related to the release of bone-resorbing factors from mononuclear leukocytes present in the cyst wall, including interleukin, mast cell tryptase and prostaglandins (10, 25, 6, 46).

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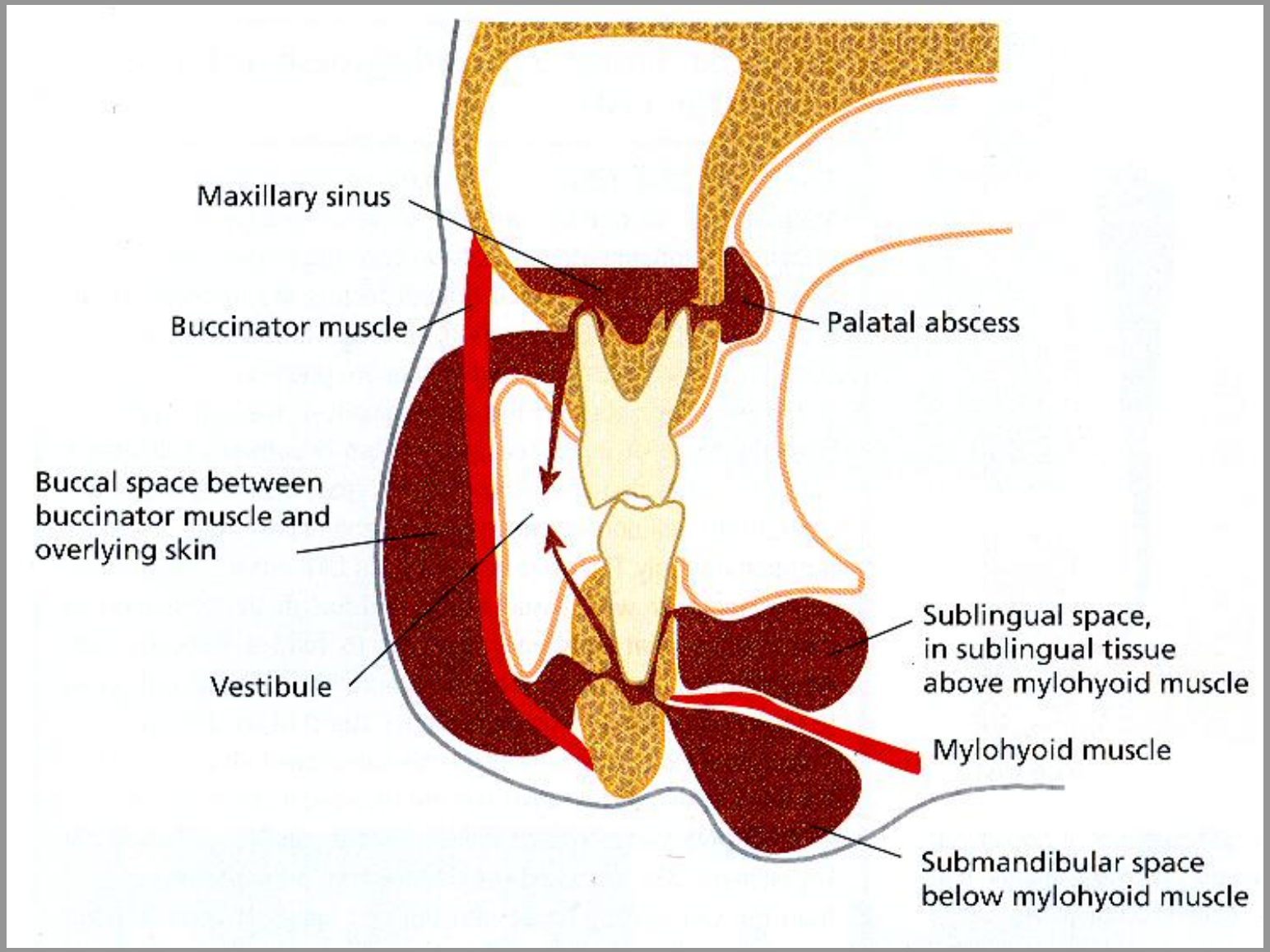

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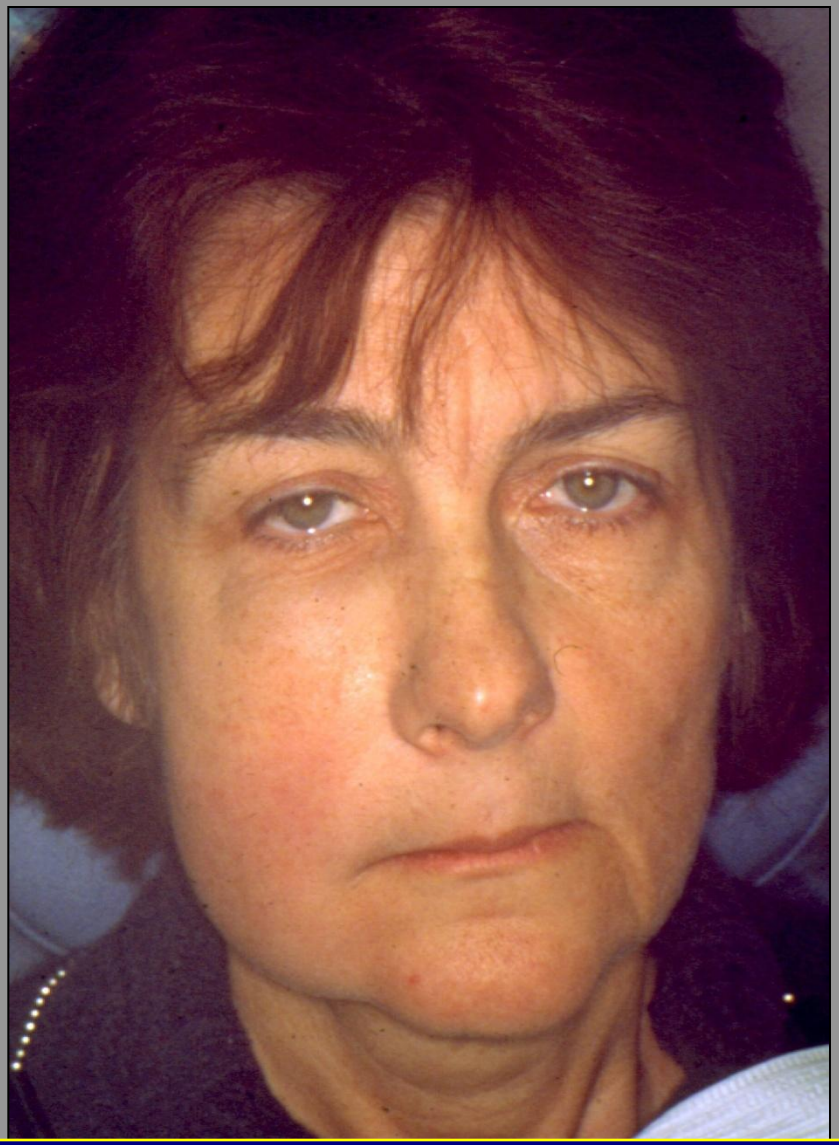
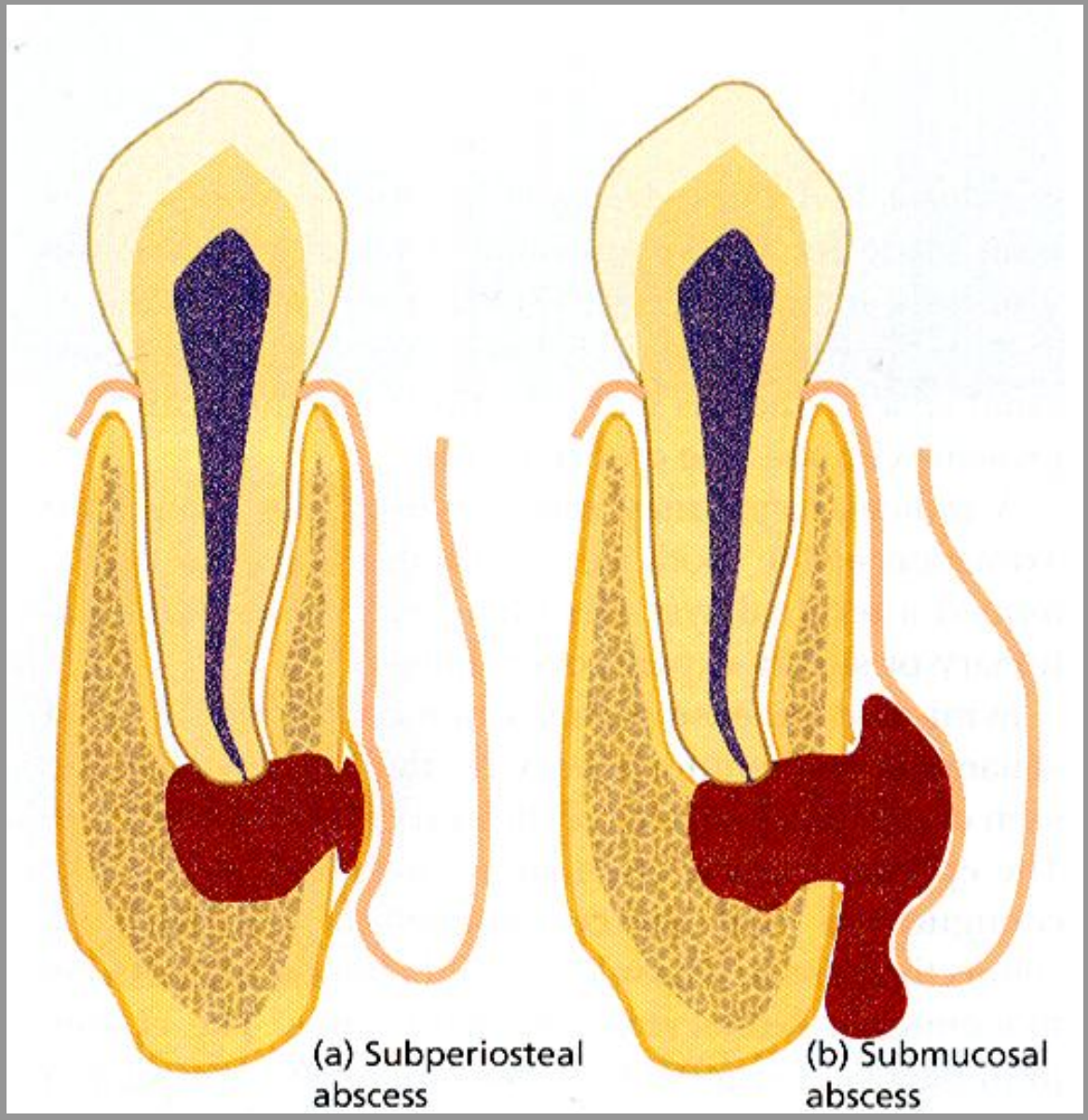


Infection from 46 with abscess in buccal space



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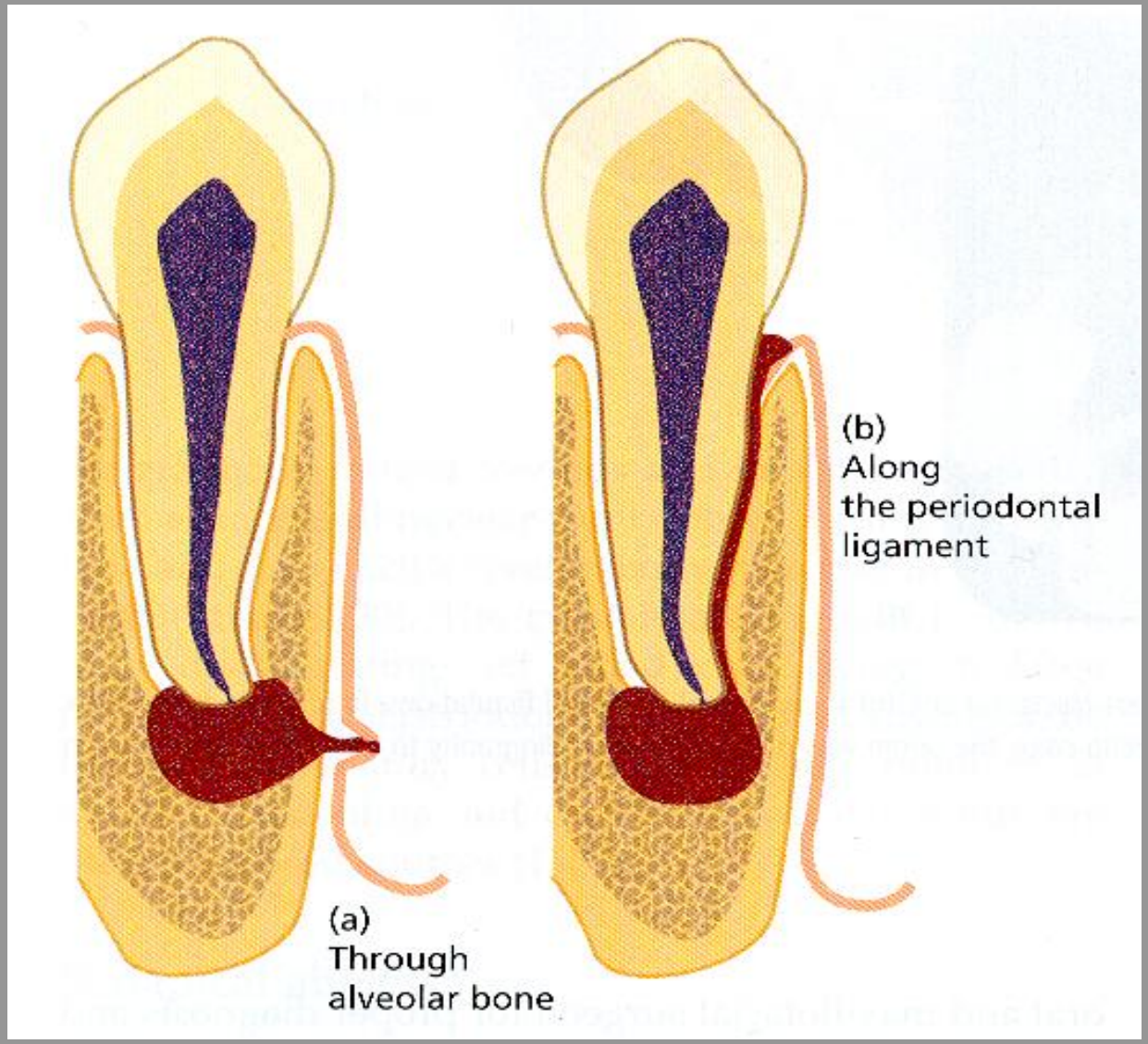

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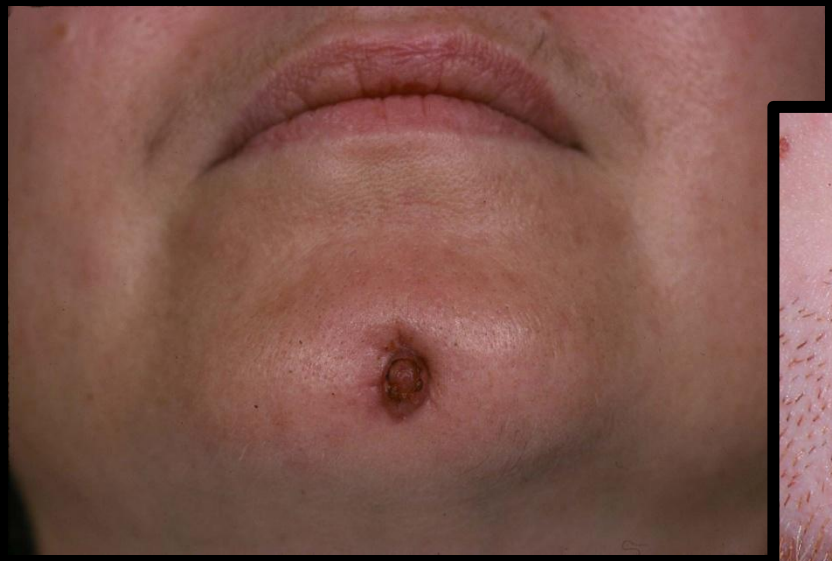
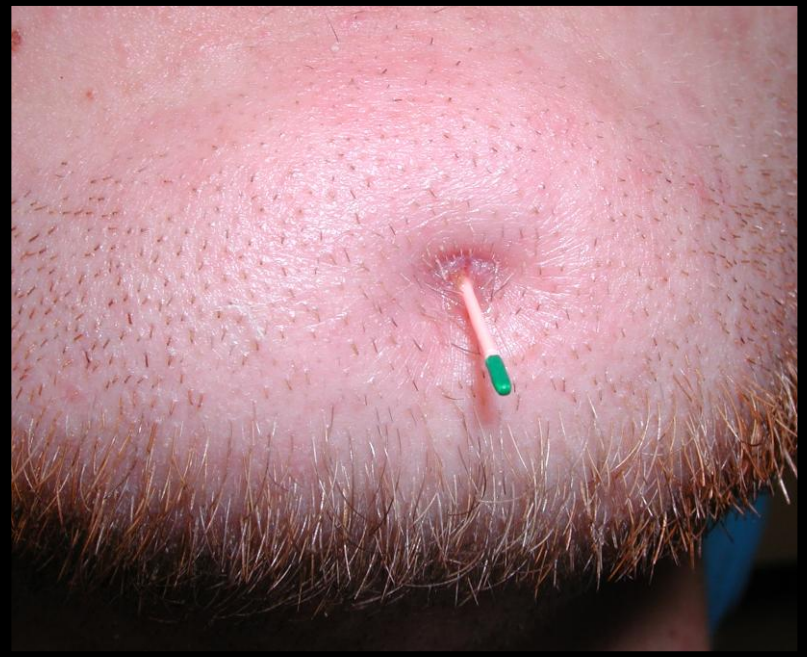
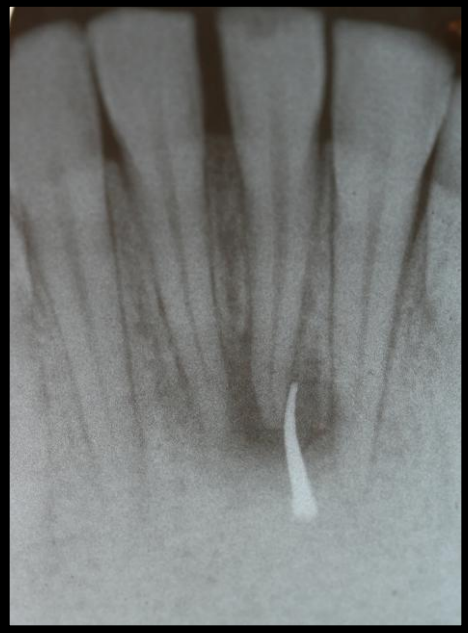
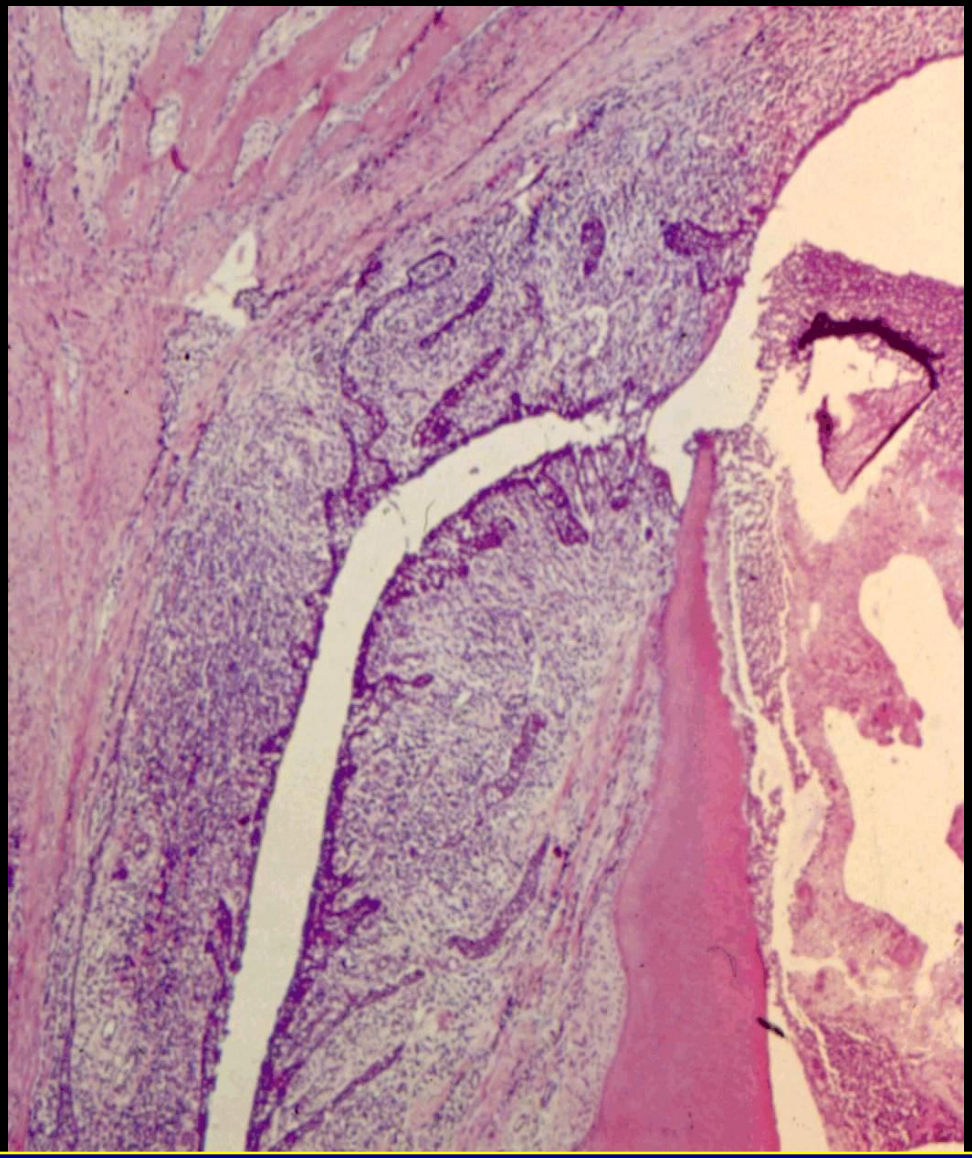


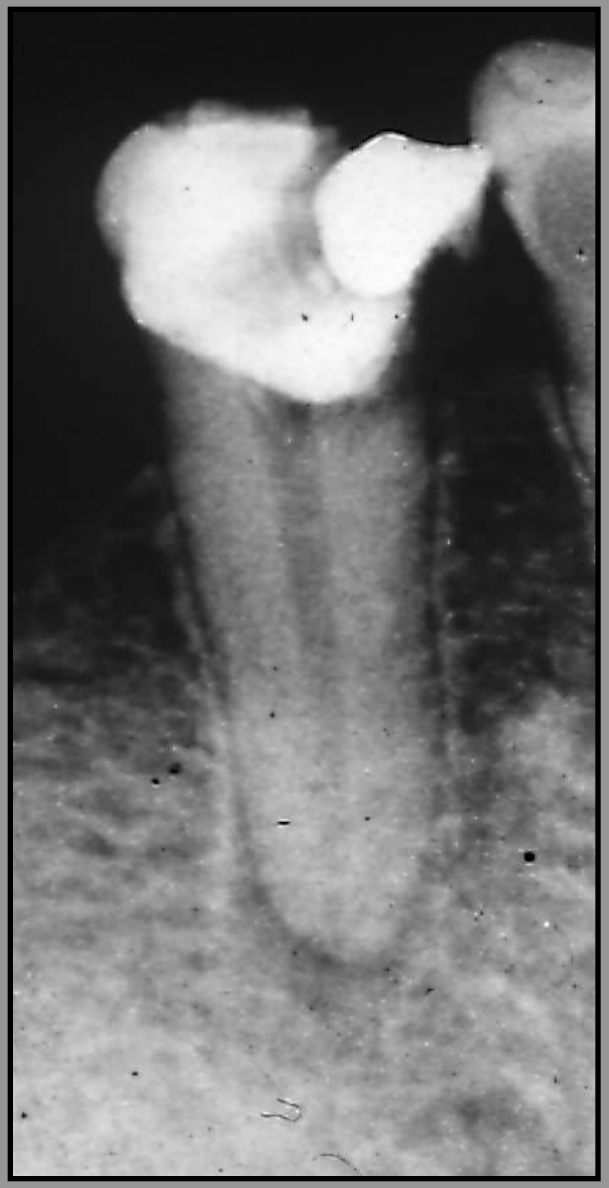


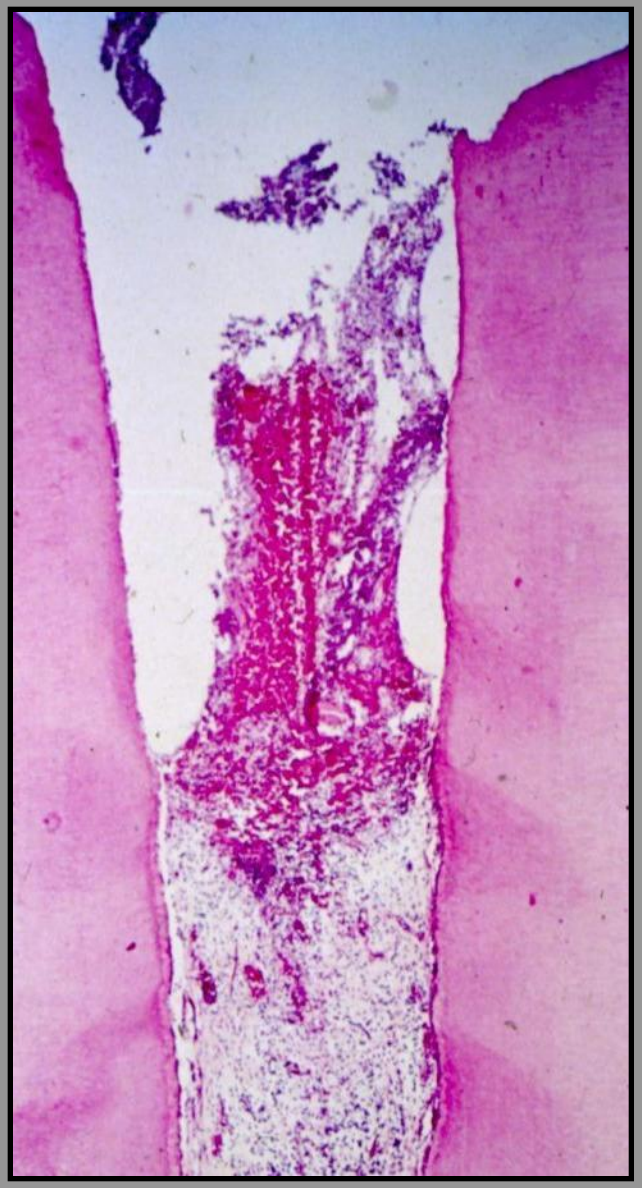


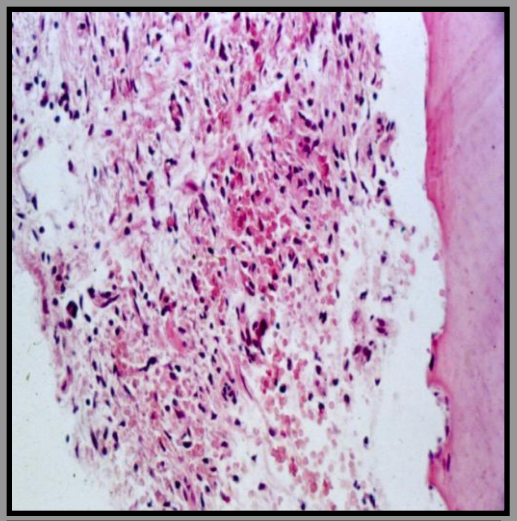
Dra...ng sinus tract from lower central incisor

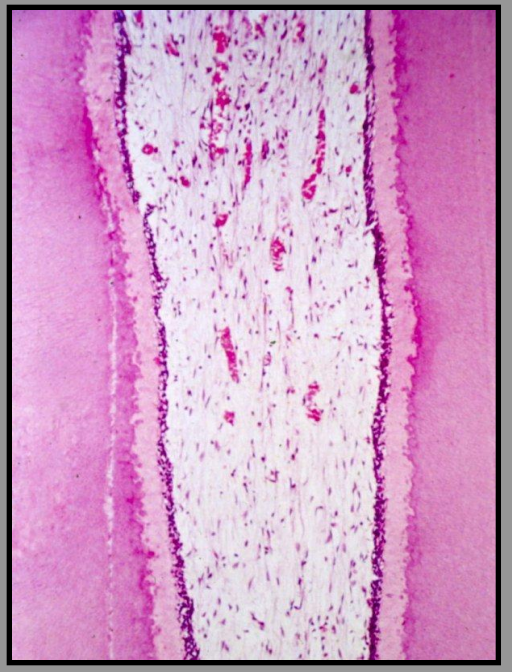


Draining sinus tract with epithelial coverage (Valderhaug)

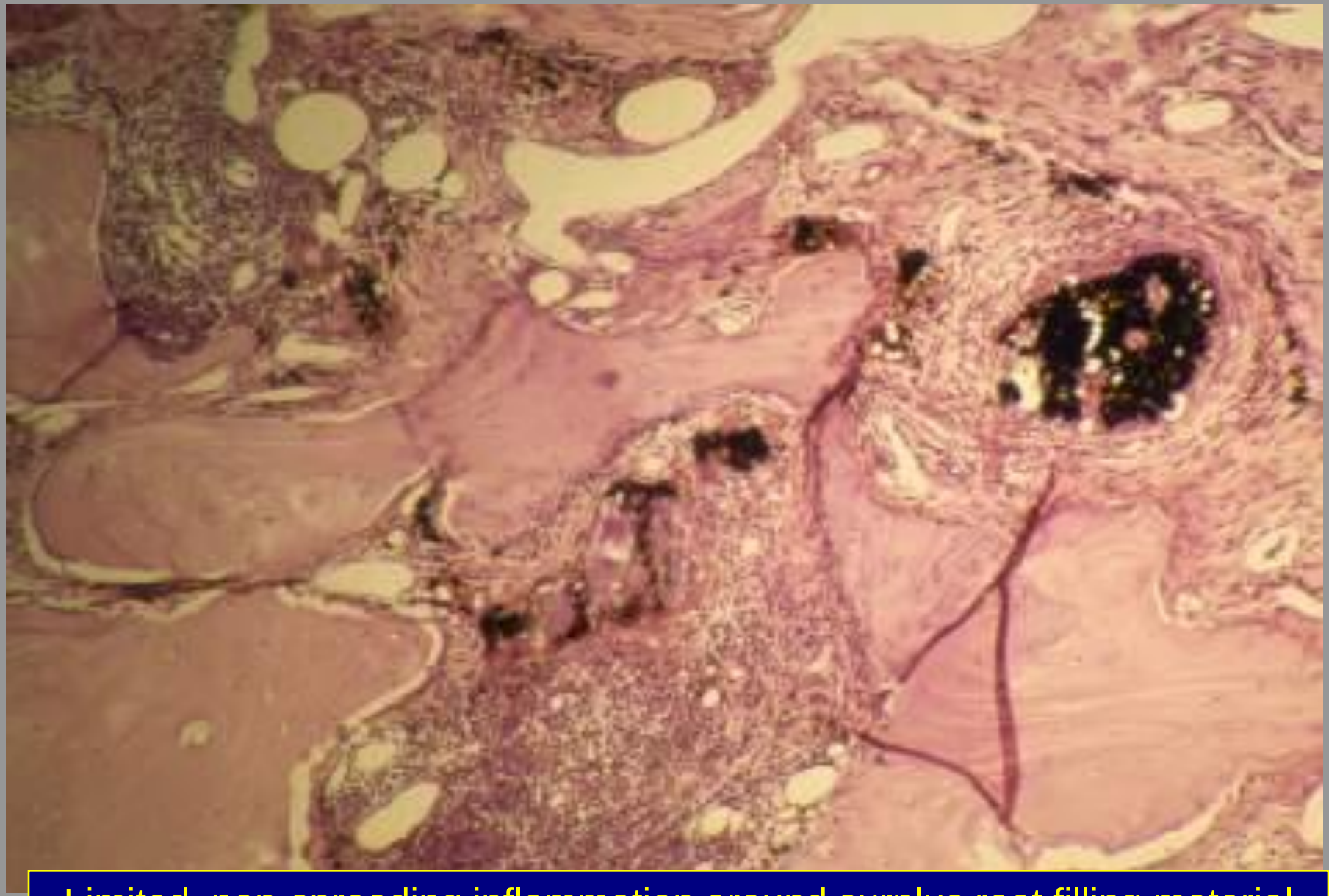






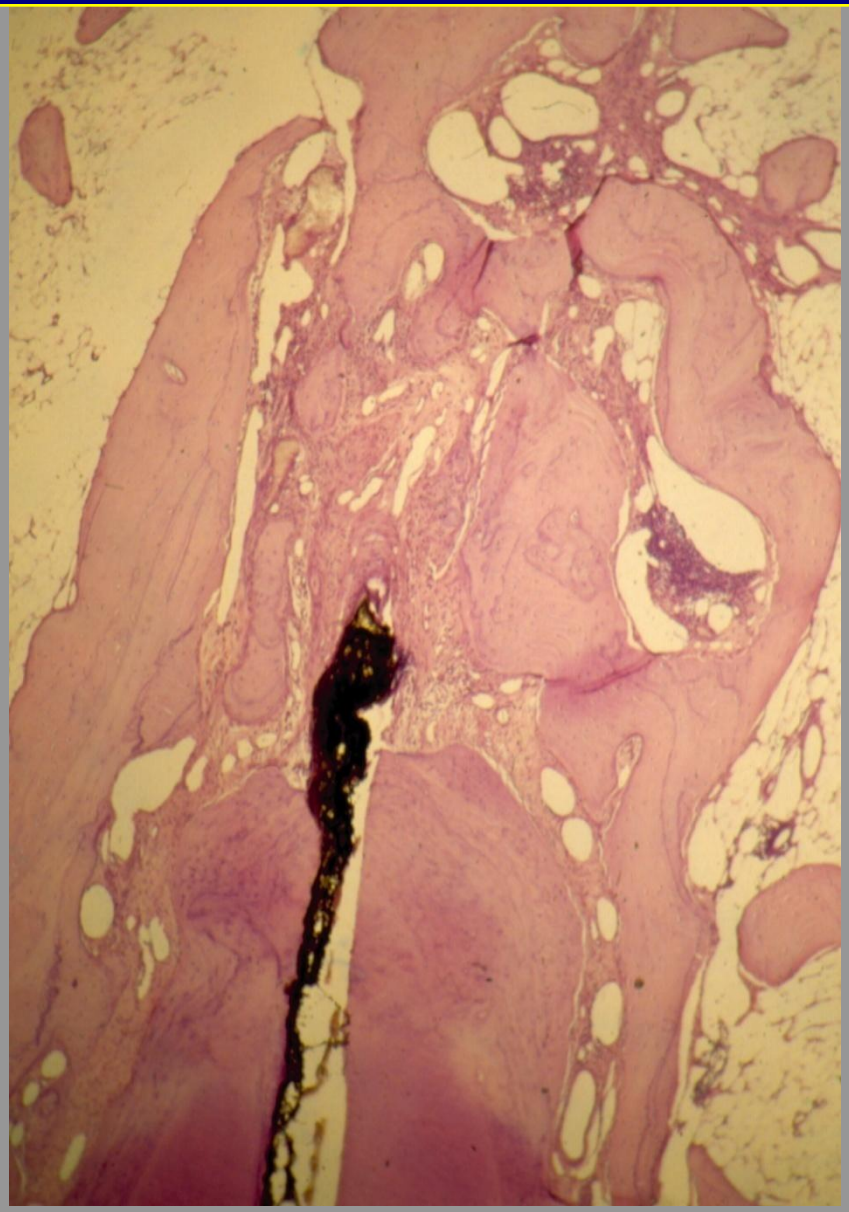


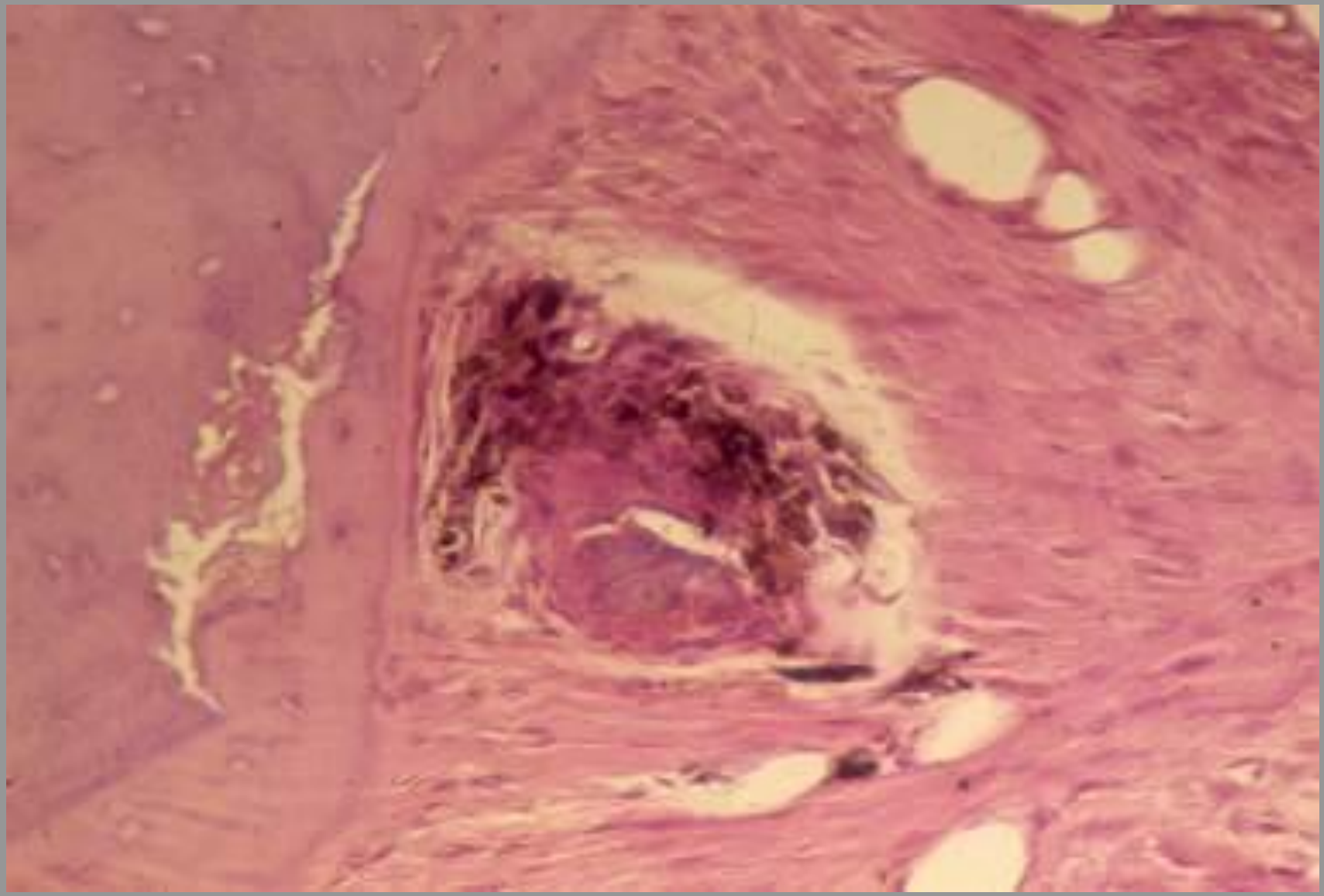
Only partially necrotic/infected pulp, but still apical periodontitis



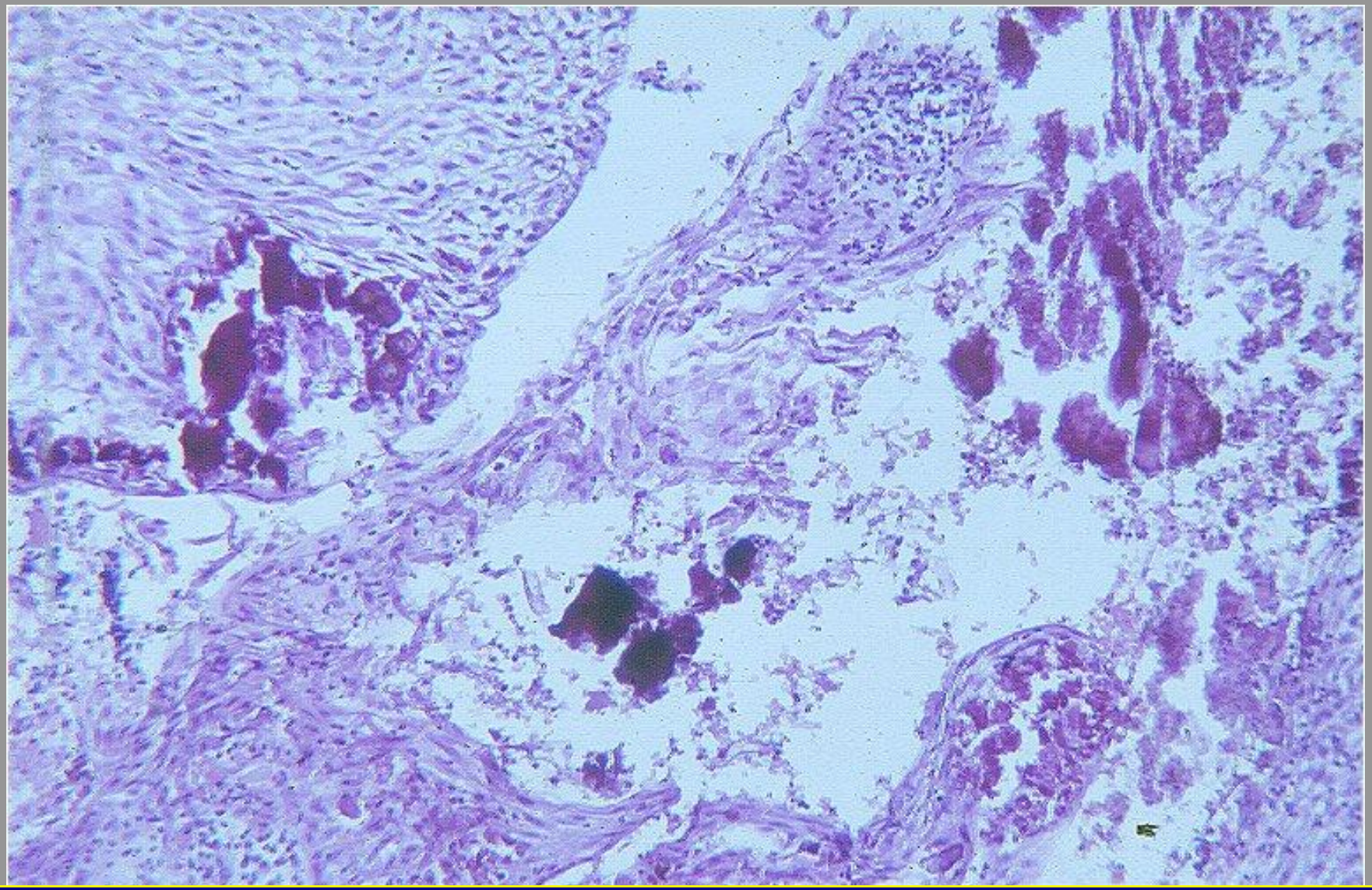
Limited, non-spreading inflammation around surplus root filling material

Limited, non-spreading inflammation around surplus root filling material

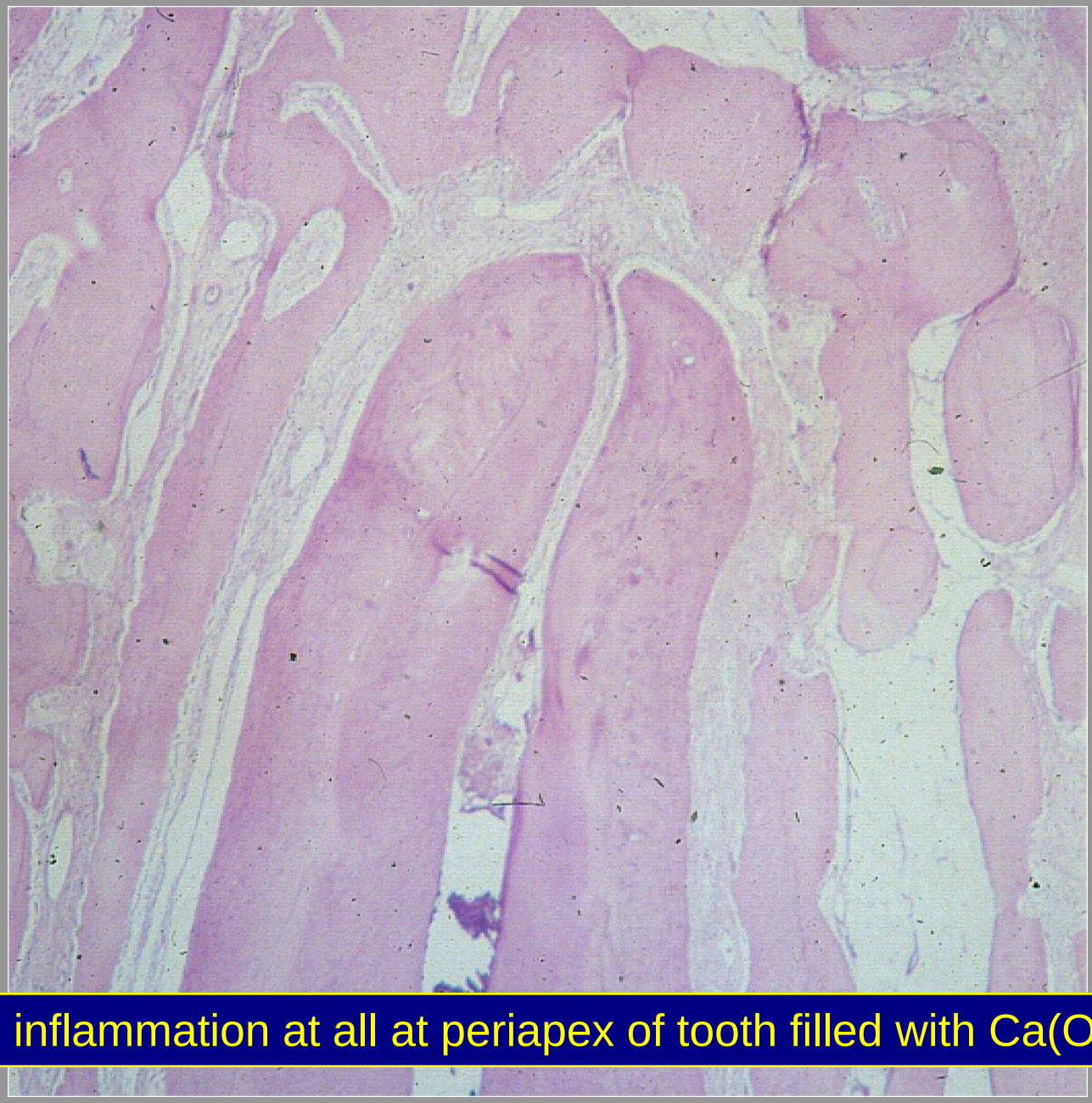




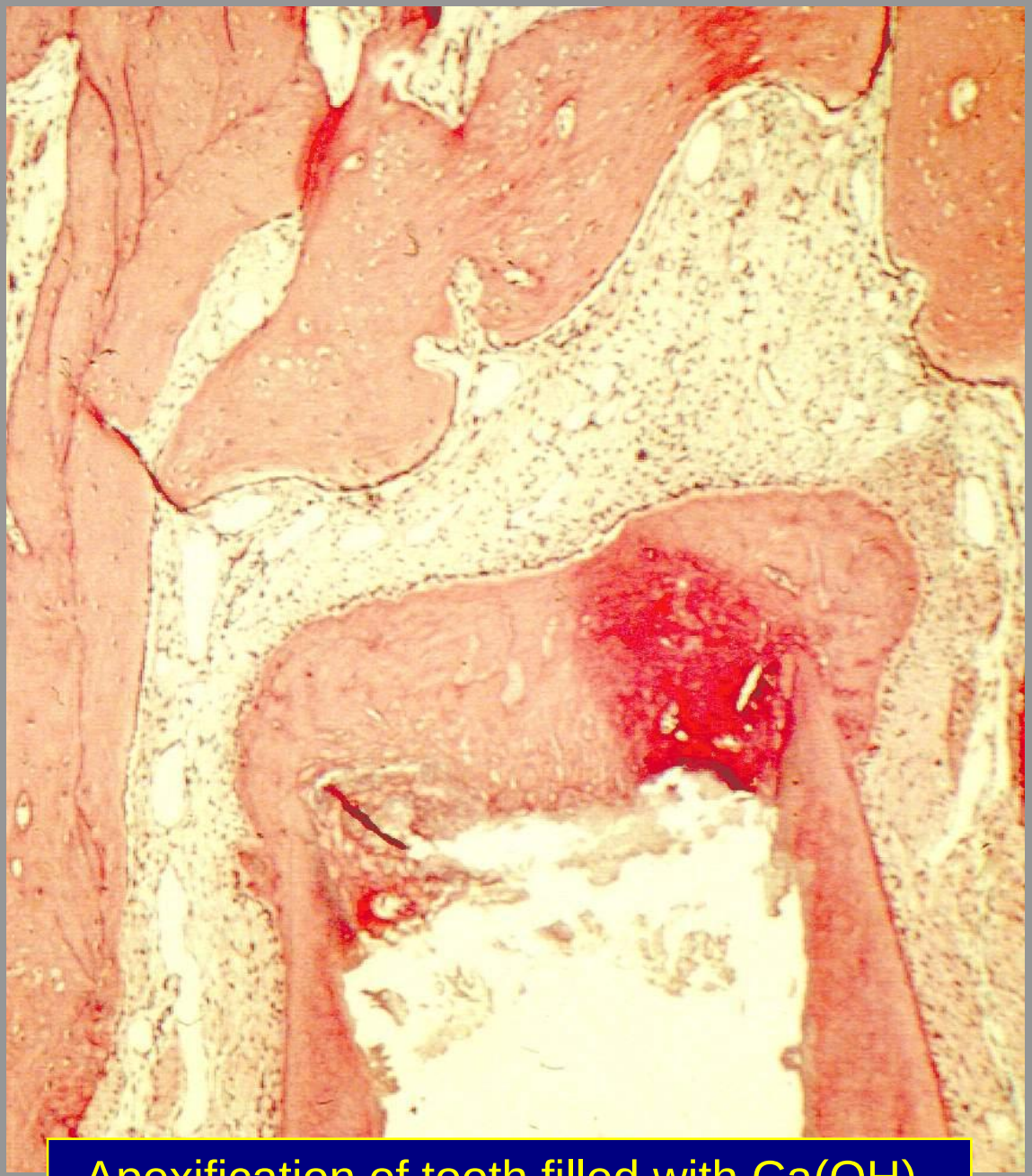
Almost no inflammation around surplus root filling material



Surplus root filling material in biopsy of apical periodontitis: possible etiologic role?



No inflammation at all at periapex of tooth filled with $Ca(OH)_2$



Apexification of tooth filled with $Ca(OH)_2$

Oral Microbiol Immunol. 2005 Oct;20(5):**Detection of bacteria in endodontic samples by polymerase chain reaction assays and association with defined clinical signs in Italian patients.** Foschi F, Cavrini F, Montebugnoli L, Stashenko P, Sambri V, Prati C.

BACKGROUND/AIMS: The presence of selected bacteria (Enterococcus faecalis, Porphyromonas gingivalis, Prevotella intermedia, Tannerella forsythensis, Treponema denticola) in infected root canals was studied using polymerase chain reaction (PCR) assays, and the association of bacteria with clinical signs of endodontic disease was assessed. The null hypothesis, that no difference could be observed between clinical signs of apical periodontitis and a specific bacterial strain, was tested. METHODS: Microbial samples were obtained from 62 teeth in 54 patients with endodontic disease. For each tooth, clinical data including patient symptoms were collected. Teeth were categorized by diagnosis as having acute apical periodontitis (AAP, teeth with clinical symptoms but no periapical radiolucency, n=22), chronic apical periodontitis (CAP, teeth with radiolucency but no clinical symptoms, n=15) or exacerbated apical periodontitis (EAP, teeth with symptoms and radiolucency, n=25). Seventy-one percent of cases were primary endodontic infections, and 29% were recurrent ('secondary') endodontic infections (failing cases). PCR assays were used to detect the presence of the selected bacteria. RESULTS: T. denticola and E. faecalis were each detected in 15 of 62 samples (24%), P. gingivalis in 8 samples (13%), P. intermedia in 5 samples (8%), and T. forsythensis in 4 samples (7%). T. denticola was detected in 56% of teeth with EAP. E. faecalis was found in 60% of teeth with CAP and in 72% of teeth with secondary infection. Statistical analysis demonstrated an association of CAP and secondary endodontic infection with the presence of E. faecalis. ($P<0.01$). EAP was associated with the presence of T. denticola ($P<0.01$). CONCLUSION: T. denticola was associated with symptomatic endodontic disease in the presence of apical bone resorption. E. faecalis was associated with treatment failures. We suggest that these species may play critical roles in endodontic pathology.

J Med Microbiol. 2005 Aug;54(Pt 8):777-83. **Quantification of endotoxins in necrotic root canals from symptomatic and asymptomatic teeth.**

Jacinto RC, Gomes BP, Shah HN, Ferraz CC, Zaia AA, Souza-Filho FJ.

The purpose of this investigation was to quantify the concentration of endotoxin in necrotic root canals and investigate the possible relationship between the concentration of endotoxin and endodontic signs and symptoms. Samples were collected from root canals of 50 patients requiring endodontic treatment due to necrosis of the pulpal tissue. Anaerobic techniques were used to determine the number of c.f.u. in each sample. A quantitative chromogenic Limulus amoebocyte lysate assay was used to measure the concentration of endotoxin in each sample. The presence of c.f.u. was detected by culture in all samples (range 10(2)-5x10(6)). In samples from cases of patients with spontaneous pain, the mean c.f.u. was 1.43x10(6) while in asymptomatic cases it was 9.1x10(4). Endotoxin was present in all the samples studied [range 2390.0-22100.0 endotoxin units (EU) ml-1]. The mean concentration of endotoxin in samples from patients with spontaneous pain was 18540.0 EU ml-1 while in asymptomatic cases it was 12030.0 EU ml-1. Asymptomatic cases generally had lower levels of endotoxin (i.e. a negative association). A positive association was found between endotoxin and symptomatic cases (e.g. spontaneous pain, tenderness to percussion, pain on palpation, swelling and purulent exudates). This study showed that endotoxin is present in high concentrations in root canals of symptomatic teeth. There was a positive correlation between the concentration of endotoxin in the root canal and the presence of endodontic signs and symptoms.

Oral Microbiol Immunol. 2005 Aug;20(4):211-5. **Porphyromonas gingivalis, Porphyromonas endodontalis, Prevotella intermedia and Prevotella nigrescens in endodontic lesions detected by culture and by PCR.**

Gomes BP, Jacinto RC, Pinheiro ET, Sousa EL, Zaia AA, Ferraz CC, Souza-Filho FJ.

The aim of this study was to investigate the presence of four black-pigmented bacteria, Porphyromonas gingivalis, Porphyromonas endodontalis, Prevotella intermedia and Prevotella nigrescens, in endodontic infections by culture and polymerase chain reaction (PCR) analyses. Microbial samples were obtained from 50 teeth with untreated necrotic pulps (primary infection) and from 50 teeth with failing endodontic treatment (secondary infection). Microbiological strict anaerobic techniques were used for serial dilution, plating, incubation, and identification. For PCR detection, the samples were analyzed using species-specific primers of 16S rDNA and the downstream intergenic spacer region. Culture and PCR detected the test species in 13/100 and 50/100 of the study teeth, respectively. The organisms were cultured from 11/50 (22%) of primarily infected root canal samples and from 2/50 (4%) of secondary root canal samples. PCR detection identified the target species in 32/50 (64%) and 18/50 (36%) of primary and secondary infections, respectively. P. gingivalis was rarely isolated by culture methods (1%), but was the most frequently identified test species by PCR (38%). Similarly, P. endodontalis was not recovered by culture from any tooth studied, but was detected by PCR in 25% of the sampled teeth. PCR-based identification also showed higher detection rates of P. intermedia (33%) and P. nigrescens (22%) than culture (13%). In conclusion, P. gingivalis, P. endodontalis, P. intermedia, and P. nigrescens were identified more frequently in teeth with necrotic pulp than in teeth with failing endodontic treatment. Also, a higher frequency of black-pigmented species was detected by PCR than by culture.

J Endod. 2005 Jun;31(6):424-9. **Identification of cultivable microorganisms from primary endodontic infections with exposed and unexposed pulp space.**

Chu FC, Tsang CS, Chow TW, Samaranayake LP.

This study was aimed at comparing the cultivable microorganisms in canals with periapical radiolucencies with exposed and unexposed pulp space. Microbiological samples were taken and analyzed from 45 canals with exposed pulp space, and 43 canals with unexposed pulp space. The canal contents were analyzed by aerobic/anaerobic culture, and conventional identification techniques. There were 211 isolates of bacteria belonging to 28 genera and 55 species recovered from exposed canals. In the unexposed group, 185 isolates of bacteria were recovered, of which 54 species of 28 genera were identified. Among the four most common genera, Prevotella was significantly more common in the exposed group (51/211 in the exposed group versus 30/185 in the unexposed group) ($p = 0.049$), while there were no differences in prevalence of Actinomyces, Peptostreptococcus, and Campylobacter between two groups of canals. In addition, Fusobacterium nucleatum and Propionibacterium acne were significantly more common in the unexposed canals ($p = 0.047$ and $p = 0.0051$, respectively). Similarity in bacterial species in these two groups suggests that pulp space exposure may not be a significant factor in determining the type of bacteria present in infected canals.

Int Endod J. 2005 Jun;38(6):372-80. **Effect of endodontic procedures on enterococci, enteric bacteria and yeasts in primary endodontic infections.**

Ferrari PH, **Cai S**, **Bombana AC**.

AIM: To detect enterococci, enteric bacteria and yeast species from the canals of teeth with primary endodontic infections before and after canal preparation and to test the antibiotic susceptibility of enterococcal strains isolated from infected root canals. METHODOLOGY: Twenty-five single-rooted teeth with pulp necrosis, intact pulp chambers and periradicular lesions were selected for study. Samples were collected from canals before and after instrumentation. Amongst isolated microorganisms from infected root canals only enterococci, enteric bacteria and yeasts were identified by biochemical tests. The in vitro antimicrobial susceptibility of isolated enterococci strains was evaluated by the Etest system. RESULTS: Microorganisms were isolated from 92% of the samples following intracoronal access, 22% were enterococci, enteric bacteria or yeast species. After biomechanical preparation, these species were no longer detected. After 7 days without intracanal dressing, 100% of the canals contained microorganisms, 52% of which were target species. However, after using paramonochlorophenol [PRP (2.0 g), Rinosoro and polyethylene glycol (400 equal parts up to 100 mL)] as an intracanal dressing for 7 days, enteric bacteria and yeasts were not detected; only enterococci were still present. All strains of enterococci were susceptible to ampicillin, but exhibited variable susceptibility to rifampin and ciprofloxacin. CONCLUSIONS: Enterococci, enteric bacteria and yeasts were present in primary endodontic infections. Enterococci, particularly Enterococcus faecalis and E. faecium were resistant to removal by root canal preparation followed by intracanal dressing.

Pesqui Odontol Bras. 2004 Jul-Sep;18(3):242-6. **Susceptibility of some oral microorganisms to chlorhexidine and paramonochlorophenol.**

do Amorim CV, **Aun CE**, **Mayer MP**.

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Since the use of antimicrobial agents is required in endodontic therapies, this study aimed at determining the minimum inhibitory concentrations (MICs) of chlorhexidine digluconate and paramonochlorophenol (PMC) against microorganisms commonly found in endodontic infections. Both agents were tested by agar dilution tests against Pseudomonas aeruginosa, Staphylococcus aureus, Enterococcus faecalis, Escherichia coli, Candida albicans, Prevotella intermedia, Porphyromonas gingivalis, Porphyromonas endodontalis, Prevotella denticola and Prevotella melaninogenica. The MIC of chlorhexidine ranged from 2.67 to 80.00 microg/ml, and the MIC of PMC from 46.67 to 213.33 microg/ml. The highest MIC value of PMC was detected for E. faecalis whereas E. coli was the most susceptible microorganism to this agent. The highest MIC values of chlorhexidine were observed for *P. aeruginosa* whereas *E. coli* and *P. denticola* were the most susceptible microorganisms to this agent. Since the MIC values observed are much lower than the concentrations currently used in the endodontic therapy, it is suggested that both agents are effective in reducing the microbiota in the root canal.

Oral Dis. 2004 Nov;10(6):389-97. **Molecular evaluation of residual endodontic microorganisms after instrumentation, irrigation and medication with either calcium hydroxide or Septomixine.**
Tang G, Samaranayake LP, Yip HK.
BACKGROUND AND OBJECTIVE: The correct choice of antimicrobial agents as inter-appointment medicaments is as important as the instrumentation and irrigation to remove pathogens from infected root canals. Calcium hydroxide [Ca(OH)2] and framycetin sulfate (Septomixine) are common endodontic medicaments. Therefore, we evaluated the efficacy of either calcium hydroxide or Septomixine in eliminating residual intra-canal bacteria, particularly Actinomyces spp., during inter-appointment interval in endodontic therapy using molecular methods. METHODS: A total of 31 single-rooted teeth with primary root canal infections were studied immediately after opening the canals and subsequently after instrumentation, irrigation with sterile saline and 1-week medication with either Ca(OH)2 (n = 25) or Septomixine (n = 6). Whole bacterial genomic DNA was isolated directly from samples and PCR with universal primers performed to detect total intra-canal bacteria. The variable regions of 16S rDNA of bacteria were amplified and labeled with digoxigenin for further hybridization to detect Actinomyces spp. A total of seven oligonucleotide probes specific for A. bovis, A. gerencseriae, A. israelii, A. meyeri, catalase-negative A. naeslundii (genospecies 1 and 2), catalase-positive A. naeslundii genospecies 2 and A. odontolyticus were used to detect Actinomyces spp. in 22 of 31 medicated root canals [Ca(OH)2: n = 17; Septomixine: n = 5]. RESULTS: The PCR results showed that 25 of 31 examined canals were positively detected with residual microorganisms after instrumentation, irrigation with sterile saline and 1-week medication with either Ca(OH)2 (n = 20) or Septomixine (n = 5). Thus, only six canals [Ca(OH)2: n = 5, Septomixine: n = 1] were aseptic after treatment. Hybridization results showed higher detection frequency of both A. odontolyticus and A. gerencseriae after treatment. Significant correlation was found between exposed pulp before treatment and positive detection of Actinomyces spp., particularly A. odontolyticus on the second visit ($P < 0.05$). CONCLUSION: The conventional, 1-week medication of either Ca(OH)2 or Septomixine in endodontic therapy may not effectively inhibit residual bacterial growth in all root canals during inter-appointment intervals. Further investigations using, for instance quantitative real-time PCR analyses, are required to substantiate the present findings.

Crit Rev Oral Biol Med. 2004 Sep 1;15(5):308-20. **Virulence factors of Enterococcus faecalis: relationship to endodontic disease.**

Kayaoglu G, Orstavik D.
Gazi University, Faculty of Dentistry, Department of Endodontics and Conservative Treatment, 82. Sokak 06510 Emek, Ankara, Turkey. guvenk@gazi.edu.tr <guvenk@gazi.edu.tr>

Enterococcus faecalis is a micro-organism that can survive extreme challenges. Its pathogenicity ranges from life-threatening diseases in compromised individuals to less severe conditions, such as infection of obturated root canals with chronic apical periodontitis. In the latter situation, the infecting organisms are partly shielded from the defense mechanisms of the body. In this article, we review the virulence factors of E. faecalis that may be related to endodontic infection and the periradicular inflammatory response. The most-cited virulence factors are aggregation substance, surface adhesins, sex pheromones, lipoteichoic acid, extracellular superoxide production, the lytic enzymes gelatinase and hyaluronidase, and the toxin cytolysin. Each of them may be associated with various stages of an endodontic infection as well as with periapical inflammation. While some products of the bacterium may be directly linked to damage of the periradicular tissues, a large part of the tissue damage is probably mediated by the host response to the bacterium and its products.

J Endod. 2004 Oct;30(10):689-94. **An evidence-based analysis of the antibacterial effectiveness of intracanal medicaments.**

Law A, Messer H.
Postgraduate Endodontics, School of Dental Science, University of Melbourne, Melbourne, Australia. amanda.law@bigpond.com

The authors reviewed the literature evaluating the antibacterial effectiveness of intracanal medicaments used in the management of apical periodontitis. A PICO (problem, intervention, comparison, outcome) strategy was developed to identify studies dealing with calcium hydroxide, phenolic derivatives, iodine-potassium iodide, chlorhexidine, and formocresol. The final inclusion/exclusion criteria eliminated all papers except five that evaluated calcium hydroxide. The total sample size in the included studies was 164 teeth. Microbiologic sampling was performed before endodontic treatment (S1), after instrumentation and irrigation (S2), and after intracanal medication (S3). At S2, 62% of canals were positive. After medication, 27% still showed detectable growth. Of cultures that were positive at S2, 45% were still positive at S3. Most studies did not address issues of culture reversals or false positive and false negative cultures. The main component of antibacterial action appears to be associated with instrumentation and irrigation, although canals cannot be reliably rendered bacteria free. Calcium hydroxide remains the best medicament available to reduce residual microbial flora further.

Oral Microbiol Immunol. 2004 Oct;19(5):327-30.Related Articles, Links

In vitro antimicrobial effect of bacteriophages on human dentin infected with Enterococcus faecalis ATCC 29212.

Paisano AF, Spira B, Cai S, Bombana AC.

Department of Endodontics/University of Sao Paulo, Sao Paulo, Brazil. silvanac@icb.usp.br

This study assessed the effect of bacteriophages on the viability of Enterococcus faecalis. Human dental roots were inoculated with a suspension of E. faecalis at three different multiplicities of infection - 0.1, 1.0 and 10.0. The phage lysate was able to significantly inhibit bacteria growth when incubated at the multiplicities of infection of 1.0, 10.0 and 0.1. The dental roots were also inoculated with bacteria for 6 days to allow bacterial penetration into the teeth tubules. Addition of the phage lysate to the roots following the 6-day incubation period led to a substantial reduction in bacteria viability. Phage therapy may be an important alternative for the treatment of root canal infections refractory to conventional endodontic therapy.

Int Endod J. 2004 Aug;37(8):519-24. Erratum in:
Int Endod J. 2005 Nov;38(11):854. (Relates to description of primers.)

Cytomegalovirus infection in symptomatic periapical pathosis.

Slots J, Nowzari H, Sabeti M.

AIM: To compare the presence of human cytomegalovirus (HCMV) and Epstein-Barr virus (EBV) infections in samples from 25 symptomatic and 19 asymptomatic periapical lesions. METHODOLOGY: Periapical samples were collected by sterile curettes in conjunction with apicectomy. cDNA-based HCMV and EBV identification was performed on total mRNAs extracted from peripapical tissues, using primers for genes transcribed during the productive phase of the herpesvirus infection. Statistical analysis was performed using chi-squared test. RESULTS: HCMV was detected in 100% of the symptomatic and in 37% of the asymptomatic study lesions. EBV was identified only in HCMV-infected periapical lesions. The difference in occurrence of HCMV and EBV between symptomatic and asymptomatic periapical lesions was statistically significant ($P < 0.0001$). CONCLUSIONS: The noteworthy finding of this study was the ubiquitous occurrence of HCMV active infection in symptomatic periapical pathosis. EBV may contribute to periapical pathogenesis in a subset of symptomatic lesions. HCMV and EBV infections may cause periapical pathosis by inducing cytokine and chemokine release from inflammatory or connective tissue cells, or by impairing local host defences resulting in heightened virulence of resident bacterial pathogens. Knowledge about the role of herpesviruses in periapical pathosis seems important to fully delineate the pathogenesis of endodontic infectious diseases. HCMV and probably EBV should be added to the list of putative pathogenic agents in symptomatic periapical disease.

Eksamensspørsmål

- Beskriv pulpas respons på antigener som er kommet inn i overfladisk dentin
- Beskriv pulpa-dentin-organets cellulære og nervøse komponenter som aktiveres ved traume eller karies i dentin
- Hvilke bakterier dominerer i en primærinfeksjon av pulpasystemet?

Eksamensspørsmål

- Skisser dentinstrukturen med tanke på spredning av infeksiøst og antigent materiale. Hvilke forskjeller er det mellom koronalt og radikulært dentin, og hvilke aldersforandringer kan forekomme som kan tenkes å ha betydning for rotkanalsinfeksjoner?

Eksamensspørsmål

- Skisser hvordan en primær immunrespons skjer i pulpa mot antigener i dentinkanalene
- Beskriv og forklar primærdentin, sekundærdentin og reaksjonært dentin

Eksamensspørsmål

- Hva og hvor er dendritiske celler i pulpa? Hvilken rolle spiller de i pulparesponser?
- Hvordan kan hårdvevsbarrièrer induseres under perforasjoner? Hvilke celler er involvert, hvor effektiv er prosessen? Hva motvirker slik hårdvevsdannelse?

Eksamensspørsmål

- Hvilke er de fem morfologiske områder mikrobene (kan) finnes ved etablerte pulpainfeksjoner?
- Beskriv hovedtypene av bakterier ved etablerte, primære rotkanalsinfeksjoner
- Beskriv hvordan rotkanals-bakteriefloraens sammensetning endres over tid fra initial infeksjon og over lokalisasjon fra det koronale mot det apikale

Eksamensspørsmål

- Beskriv viktige økologiske determinanter for oppvekst og overlevelse av bakterier i rotkanalsystemet
- Hvilke bakterier har særlig evne til å overleve utenfor tannens overflater?
- Hva er en lommecyste? Og i motsetning til hva?

Eksamensspørsmål

- Hvor kan pussdannende infeksjoner fra pulpa gå?
- Beskriv de kliniske tegnene ved akutt apikal periodontitt fra de første symptomer til en etablert fistel. Legg vekt på ulike tann- og spredningslokalisasjoner.
- Hva definerer en radikulær cyste?
- Beskriv cellulære og morfologiske trekk ved radikulære cyster

Fig. 1: Schematic drawing showing the course of the sympathetic nerves. Postganglionic sympathetic nerves (green) from the SCG travel with the internal carotid nerve, reach the trigeminal ganglion, and supply teeth and supporting structures via the maxillary and inferior alveolar nerve (yellow). Modified from Matthews and Robinson (1980).

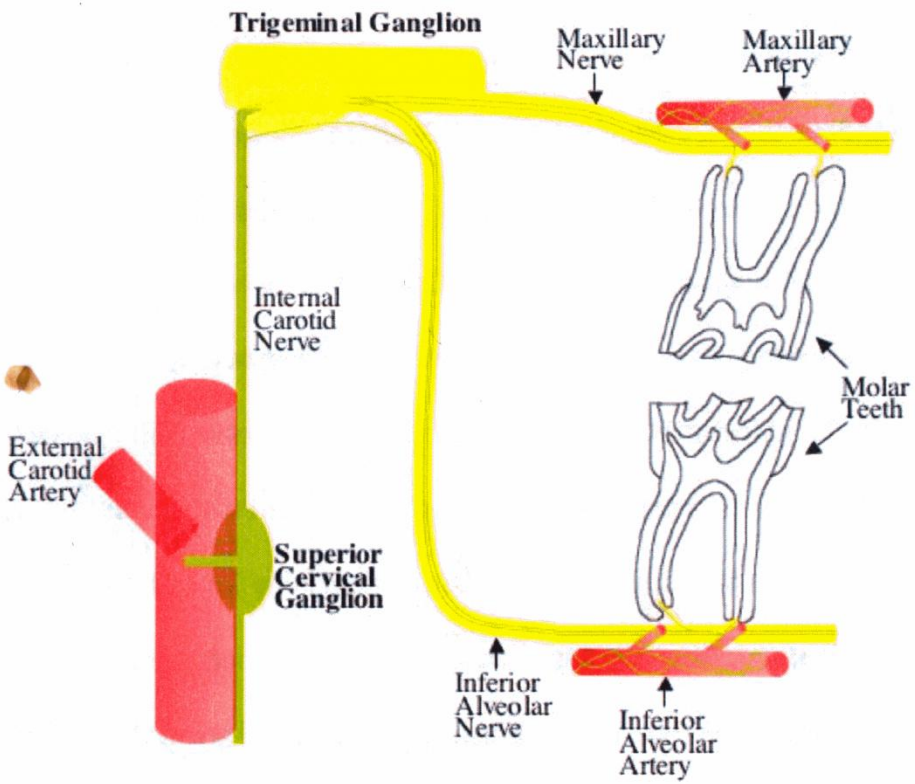
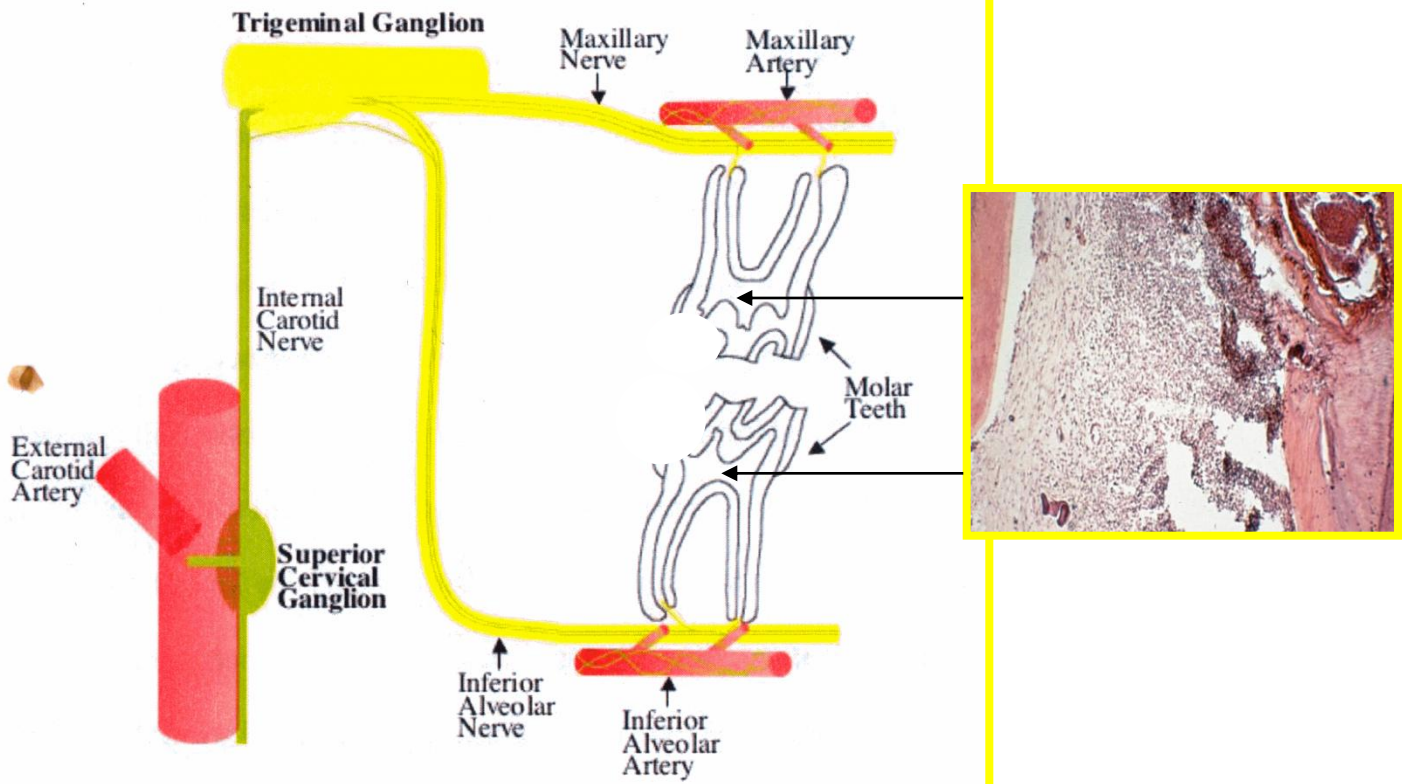
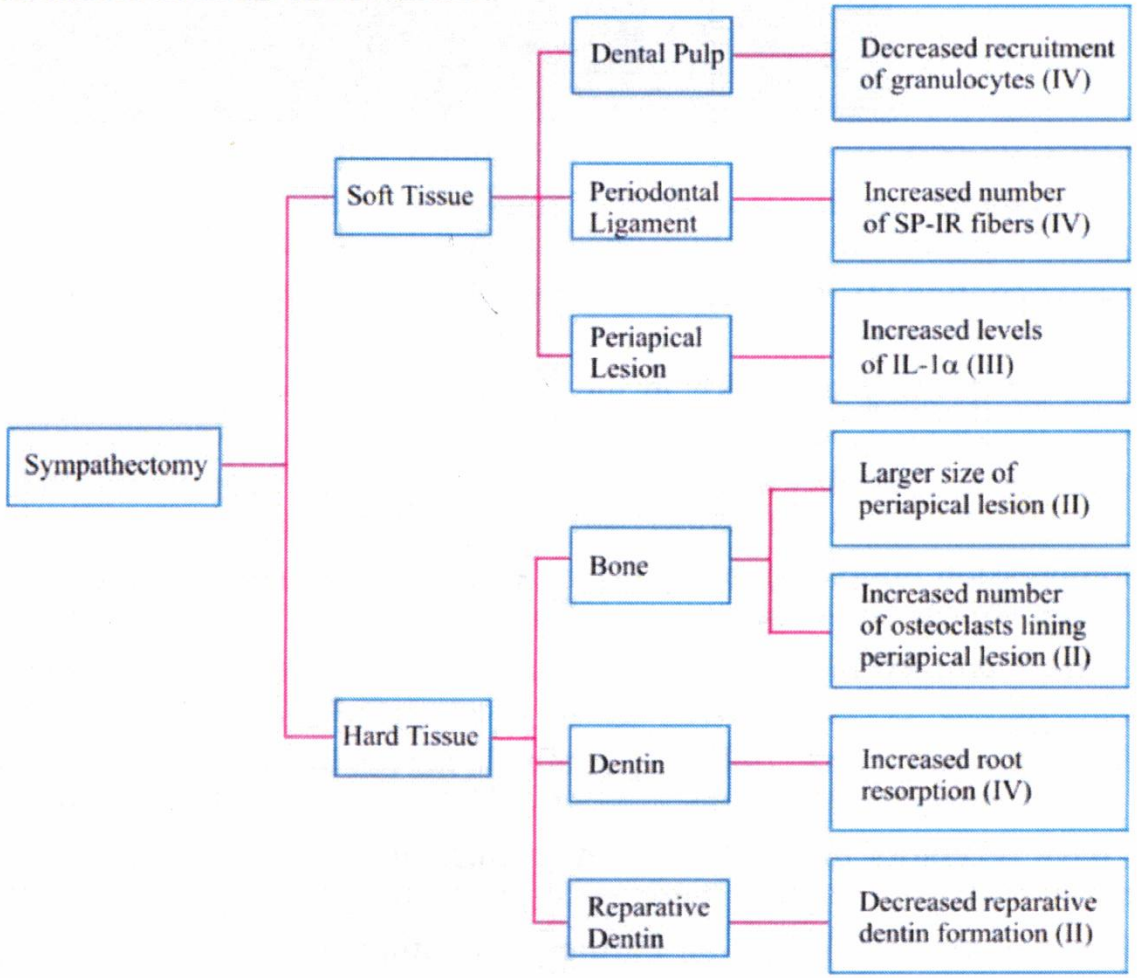


Fig. 1: Schematic drawing showing the course of the sympathetic nerves. Postganglionic sympathetic nerves (green) from the SCG travel with the internal carotid nerve, reach the trigeminal ganglion, and supply teeth and supporting structures via the maxillary and inferior alveolar nerve (yellow). Modified from Matthews and Robinson (1980).



Haug SR: Sympathetic Innervation and Dental Inflammation. Bergen 2003

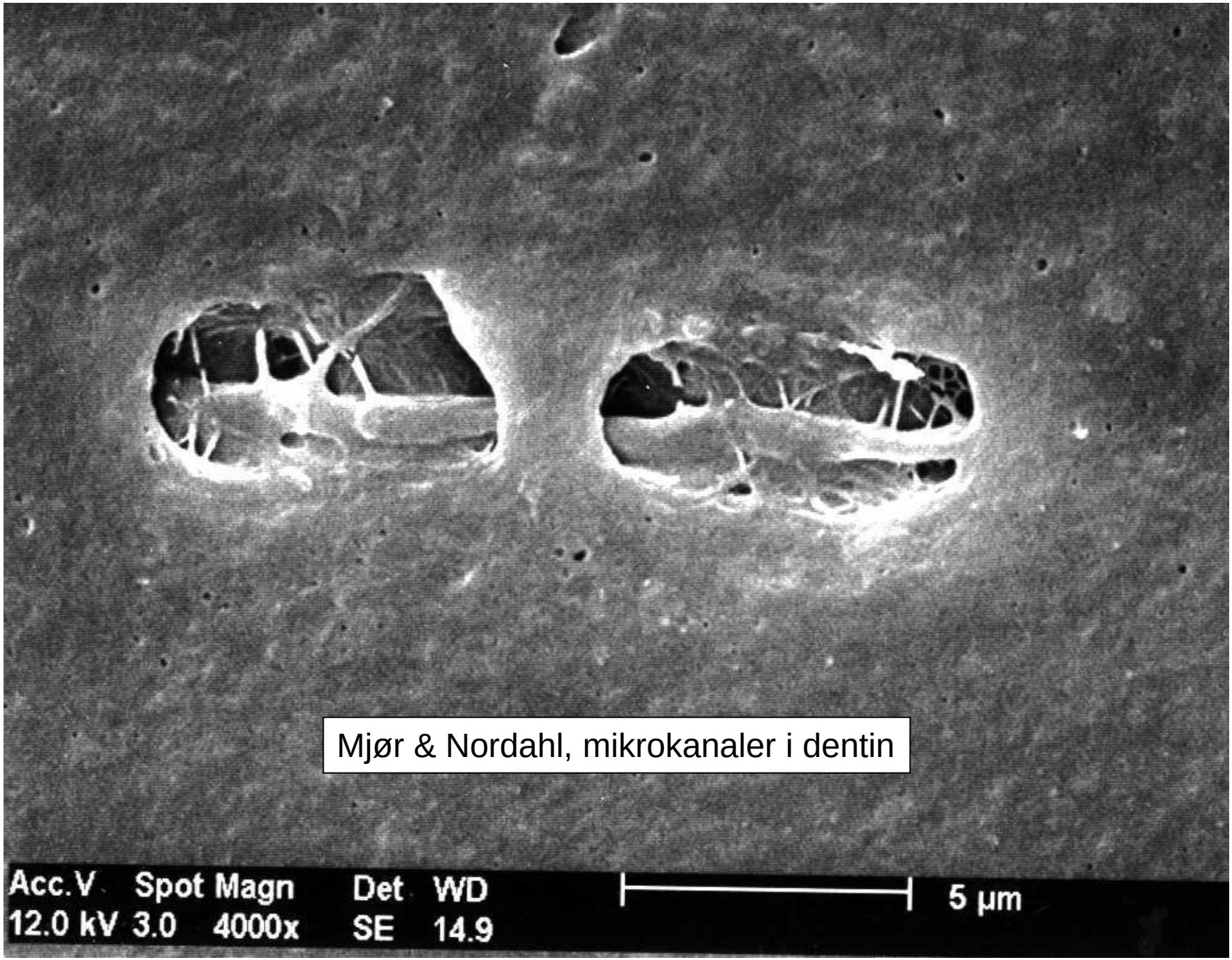
Fig. 5: A flow chart summarizing major findings on the responses of sympathectomy in dental tissue during inflammation. Roman numerals refer to article number in the thesis.



Haug SR: Sympathetic Innervation and Dental Inflammation. Bergen 2003

Guttorm Toverud

- Toverud publiserte mer enn 120 artikler, både vitenskapelige og populærvitenskapelige. Hans viktigste bidrag er The Norwegian Dental Study med data fra nasjonale, årlige undersøkelser av 5000–7000 skolebarn fra 1940–49, og med oppfølging 1952–53. Tannhelsen blant barna ble markant bedre i krigsårene, og Toveruds konklusjon var at reduksjonen i sukkerinntaket var den viktigste årsaken til denne helsegevinsten.


Mjør & Nordahl, mikrokanaler i dentin
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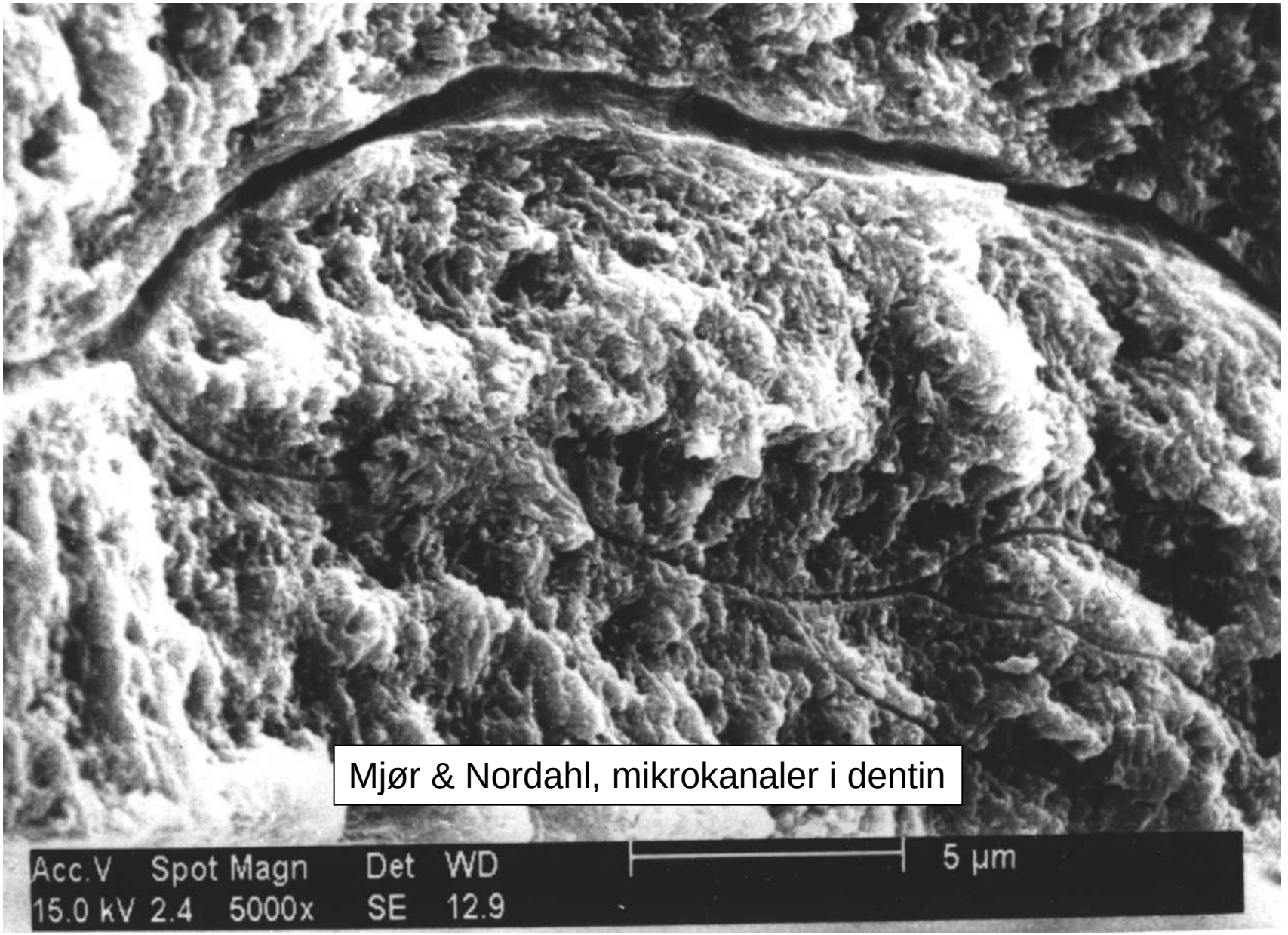
Mjør & Nordahl, mikrokanaler i dentin
Acc.V Spot Magn Det WD
15.0 kV 2.4 5000x SE 12.9
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