


Histology and Physiology of the dental pulp

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- Compositin of the dental pulp

- connectve tissue - loose

collagen fibres

- ground substance

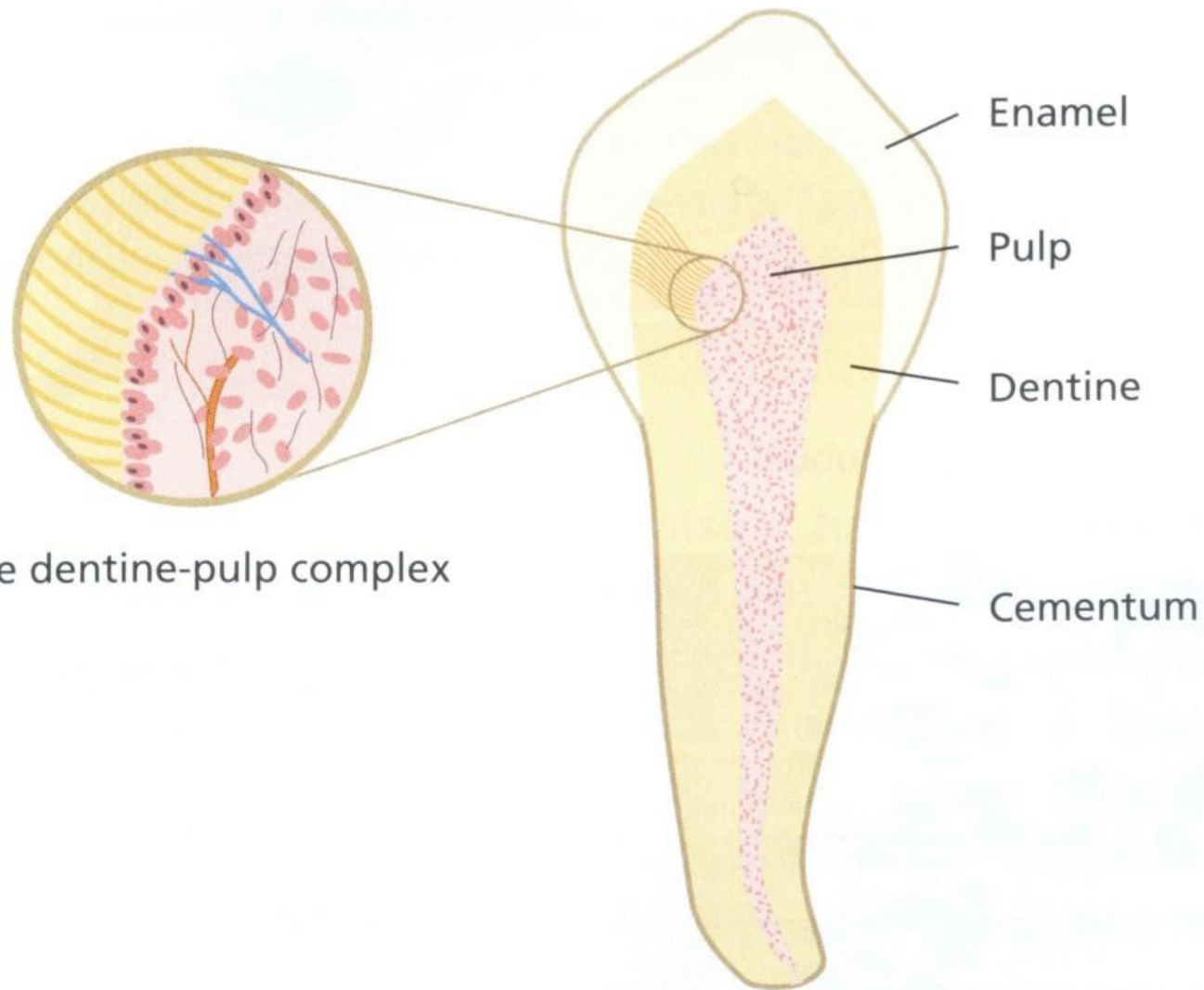
interfibrillar substance

- connective tissue cells

- blood vessels

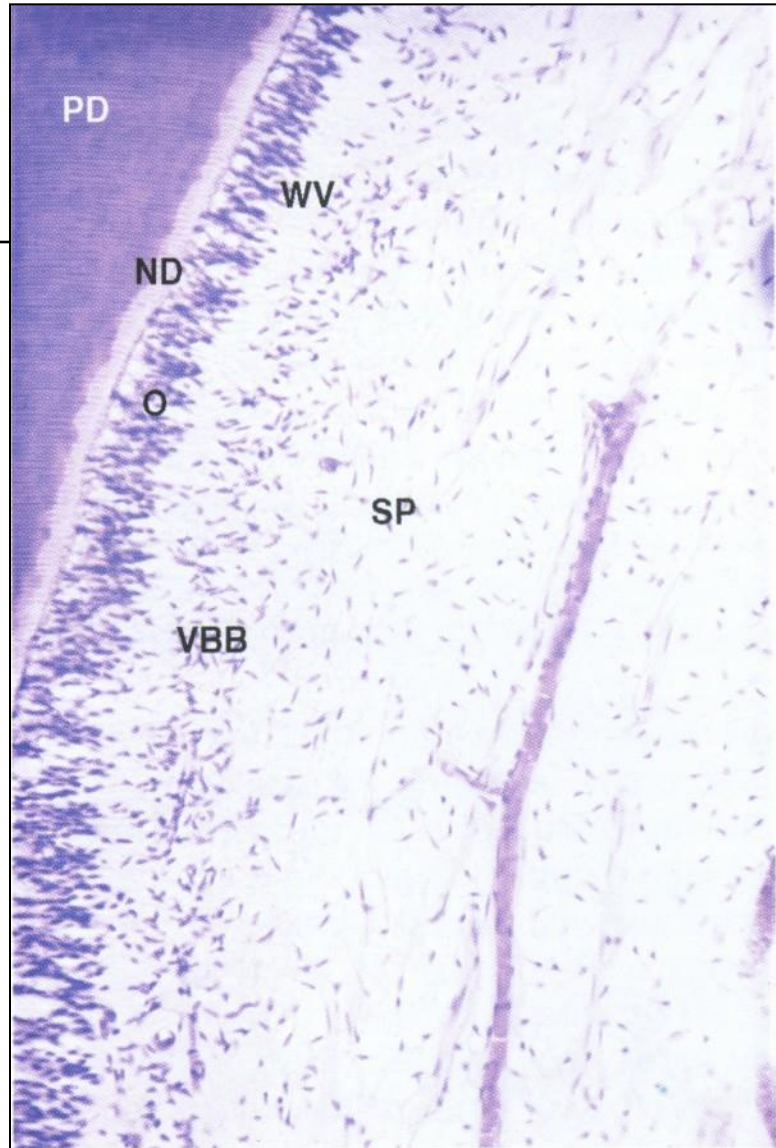
- nerve fibres

- odontoblasts - dentine forming cells



The dentine-pulp complex

Fig. 3.1 Soft tissue of the pulp surrounded by dentine and enamel and cementum. Inset depicts the interface between dentine and pulp.



Obr. 2.1 Mladá lidská zubní dřeň v horním premoláru třináctiletého pacienta (histologický preparát).

4 zones

1. central zone - larger nerves and blood vessels
2. cell rich zone - reserve cells (undifferentiated mesenchymal cells), fibroblasts
3. cell free zone (zone of Weil) - terminals of naked nerve fibres
4. odontoblastic zone

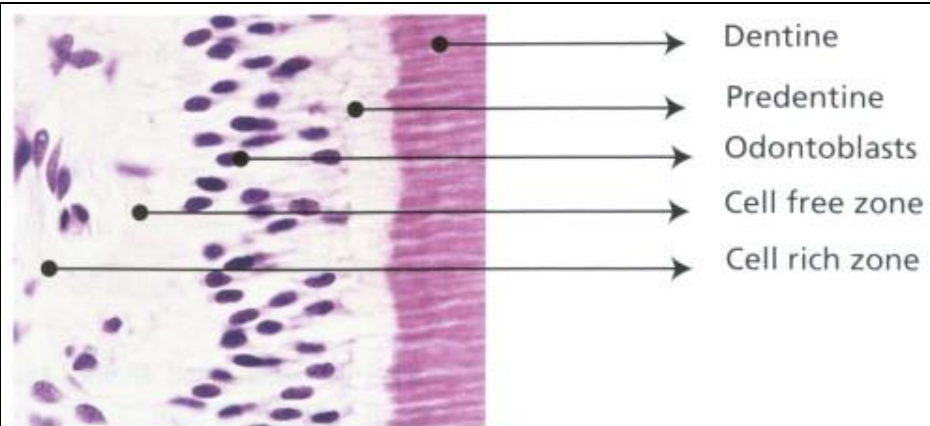



Fig. 3.4 Tissue section stained with hematoxylin and eosin showing dentine, predentine and pulp tissue proper with odontoblasts lining the periphery.

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- Structural elements of the dental pulp cells
 - intercellular substance
 - intercellular fluid

Cells of the dental pulp

- **fibroblasts** - production of intercellular substance, collagen fibres
may change into odontoblasts
age changes - more fibres
- less interfibrillar substance
- **reserve cells** - change into odontoblasts
macrophages
odontoclasts
- **histiocytes** - defense cells - phagocytosis (bacteria, foreign bodies, dead cells)
- **macrophages**, polymorphonuclear leucocytes
- **Odontoblasts** - highly specialized cells - dentin
- dental pulp
- **Dendritic cells** - immunocompetent cells, can induce lymphocyte T-cell proliferation
- **Stem cells**

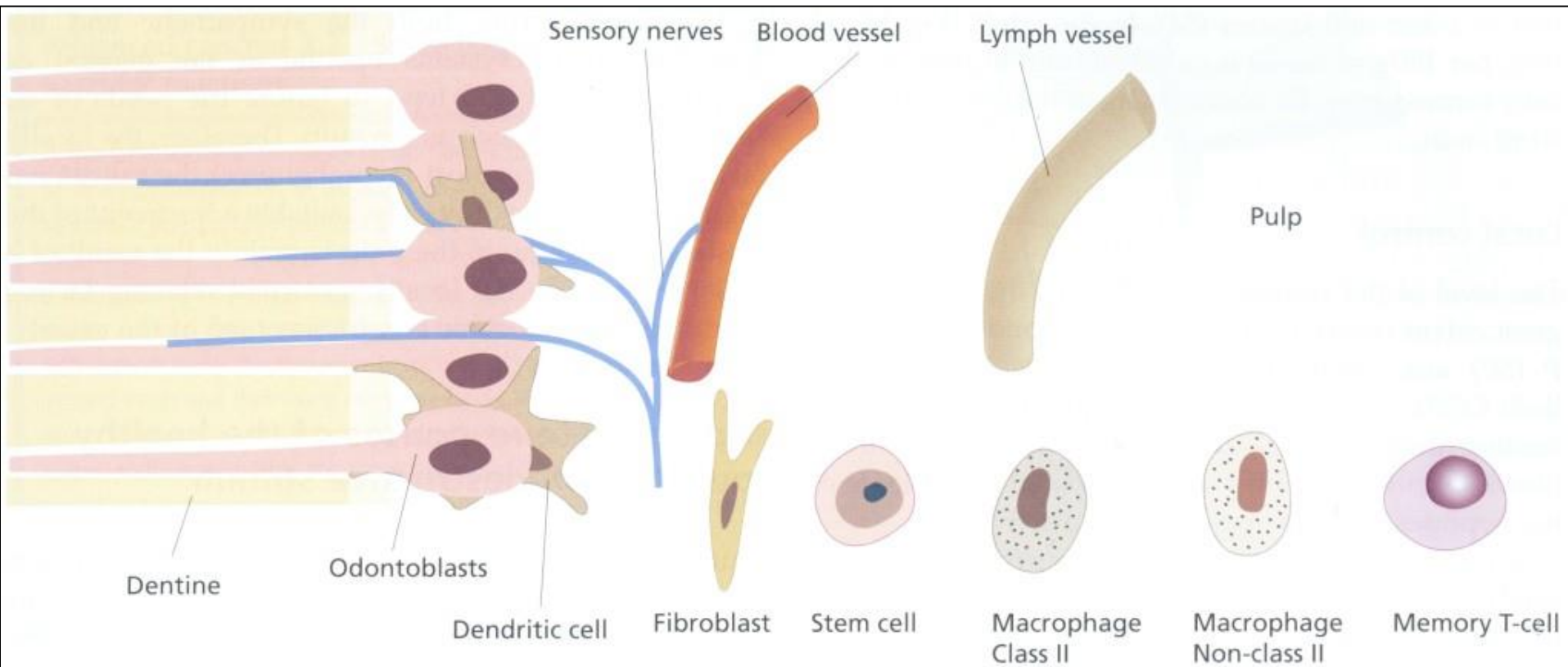


Fig. 3.11 Constituents of primary significance in the defense of the pulp against foreign substances, including bacterial elements, make up the innate 'first line of defense'.

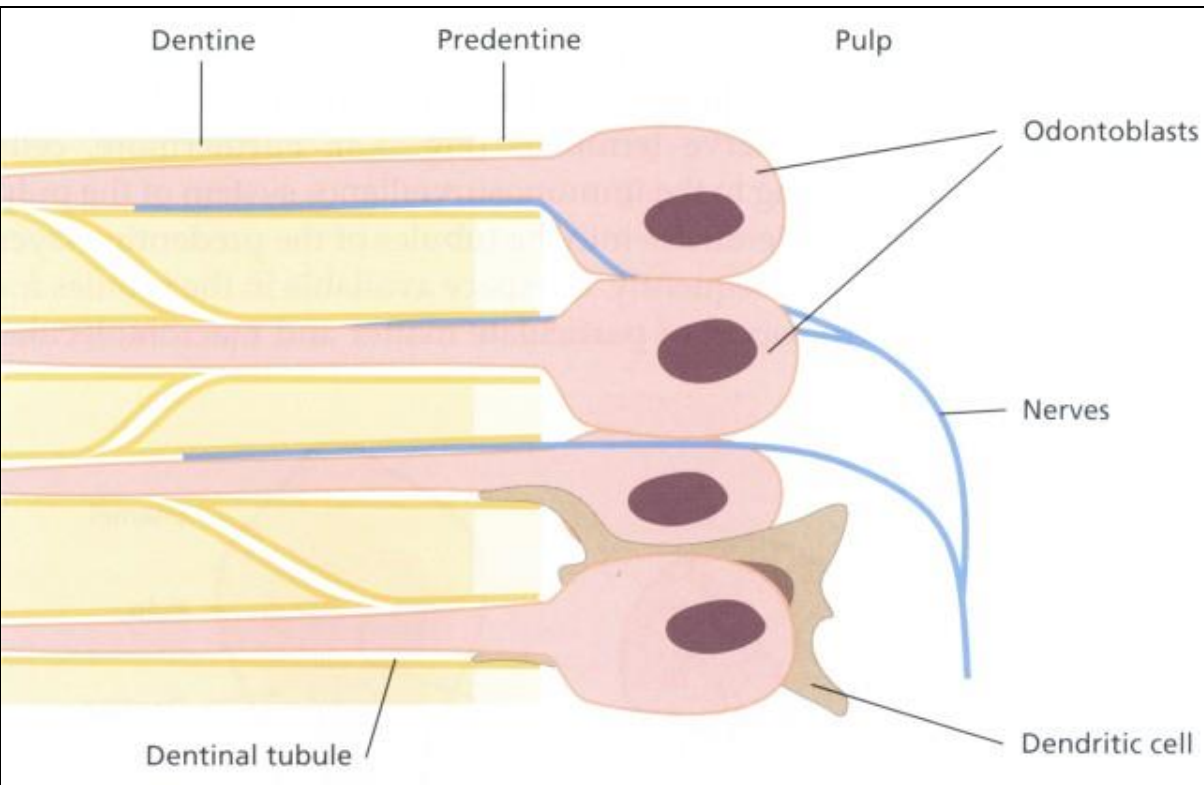


Fig. 3.3 Cellular extensions of odontoblasts, nerves and cells of the immune system (dendritic cells) that occupy the pulpal ends of the dentinal tubules.

Function of the dental pulp

- Formative - dentine formation through the life
- Nutritive - dental pulp maintains the vitality of dentine
- Nervous function
 - afferent
 - efferent
- Defensive function

Formative function

Formation of dentine through the life

- primary dentine
- secondary dentine
- tertiary dentine

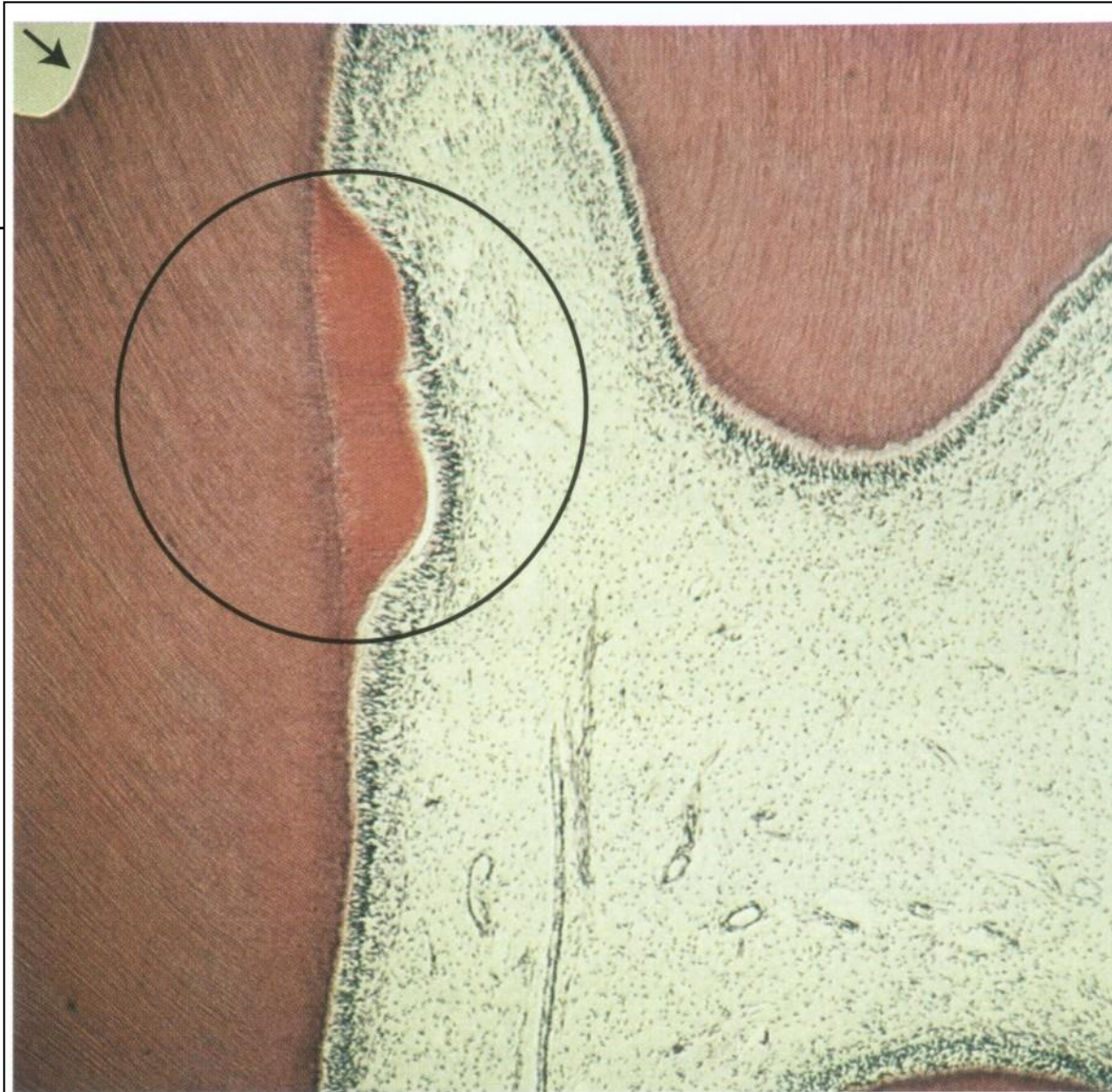


Fig. 3.5 Microphotograph shows hard tissue repair following a cavity preparation (arrow). The circle indicates the bulk of new dentine being formed.

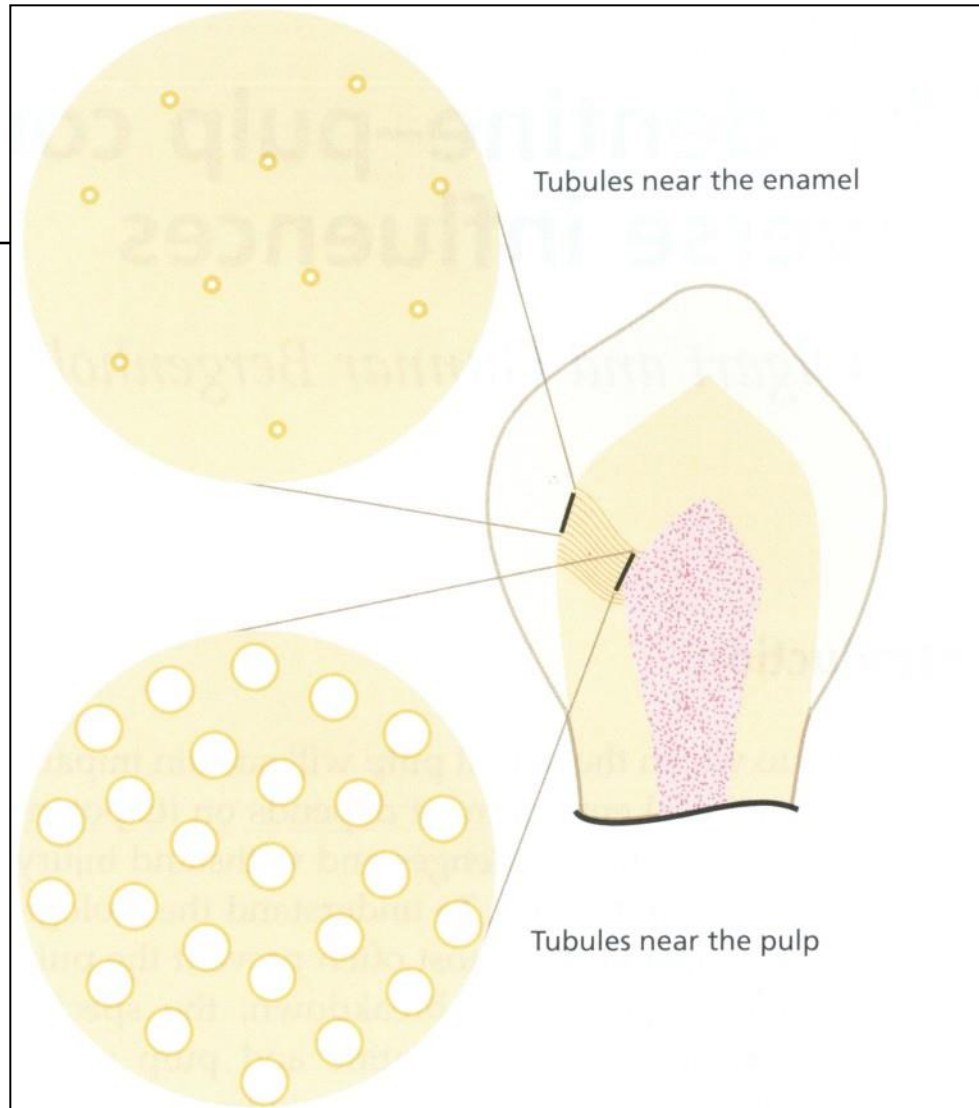


Fig. 3.2 Density of dentinal tubules in various portions of the crown region in teeth. It has been estimated that the surface area taken by cross-cut tubules is ca. 2–3% in the periphery but near the pulp the dentinal tubules assume ca. 25% of the surface area (67).

Nutritive function

- ❑ arterioles branching into terminal arterioles
- ❑ terminal capillary network - peripherally
- ❑ post capillary venules
- ❑ collective venules
- ❑ main venules
- ❑ anastomoses
- ❑ lymphatic vessels

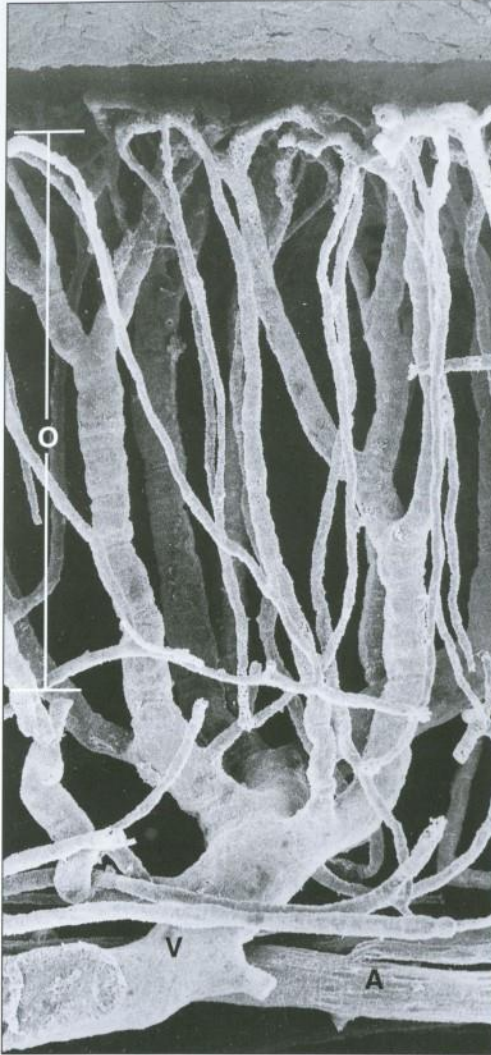


Fig 1-24 Vascularity of the pulp. A monomer is injected into the apical blood vessels and polymerized. The tooth is then demineralized, and the organic components are digested away, allowing examination of the "vascular tree." (O) Odontoblastic region; (V) venule; (A) arteriole. (Original magnification $\times 900$. Courtesy of Dr K. Takahashi.)

Nervous function

- Dental pulp - both vasomotor and sensory nerves (vasomotoric and defense functions)
- **vasomotor nerves** - sympathetic division of the autonomic system (postganglion)
- accompany arterioles

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- **efferent system** - from the central nervous system to the dental pulp multipolar neurons
 - cell bodies - lateral horn of grey matter of the upper thoracic levels of the spinal cord (preganglionic)
and
superior cervical ganglion (postganglionic)
 - ending - in the muscle cells of the vessels
 - function - vasodilatation x vasoconstriction
 - regulation of blood volume
blood flow
 - control of fluid interchange between tissues and capillaries
 - Conscious recognition of irritant - possibility to correct the problem before irreversible effects occur

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- **Afferent neuron** - dendrite - in the dental pulp
cell body - in the semilunar ganglion of the 5th cranial nerve
(trigeminus)
 - **sensory nerves** - branches of the maxillary and mandibular division of the 5th cranial nerve
- trigeminus
 - entrance - foramen apicale
central part of the pulp
branching - plexus of Raschkow
A δ nerves (myelinated, 2-5 μ m)
C, minute non-myelinated fibres (0,3-1,2 μ m)

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- A δ nerves - carry pain sensation (nociceptive)
conduction velocity - 12-30m/sec
 - C-fibres - carry pain sensation at a slower speed
0,5-1m/sec (10% C fibres - sympathetic)
 - A δ nerves - initial momentary sharp pain
response to external stimuli
stimulation without tissue injury
 - C-fibres - continuous, constant throbbing pain
higher threshold of excitation
stimulation is associated with tissue damage
(inflammatory processes)



Theories of pain transmission

- dentin innervation
- hydrodynamic mechanism
- odontoblastic deformation

Dentin innervation

- nerve fibers in the dentinal tubules - 100 - 200 μm
not proved (no synapsis between nerve fibres and odontoblasts)
- Pain producing substances (KCl, histamine, acetylcholine) do not elicit pain when applied to the peripheral dentine.
- Application of anesthetics on the peripheral dentine -
no anaesthesia of the dental pulp

Hydrodynamic mechanism

- The naked nerve endings - sensitive to sudden pressure changes, fluid movements - mechanical deformation
 - from the pulp
- source of movement
 - from the tubules
- dentine tubules contain fluid - capillary tubes



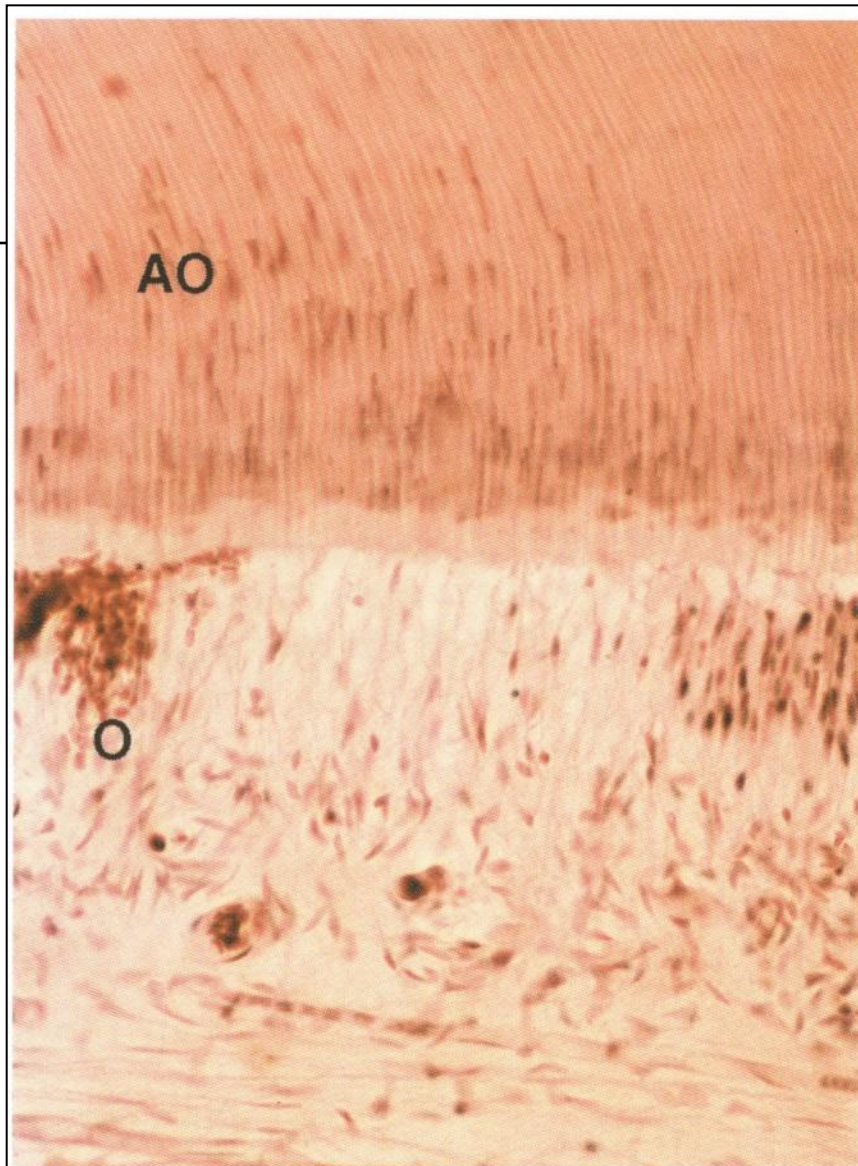
movements of the fluid in tubules:

- ❑ direct mechanical deformation on the low threshold $A\delta$ free nerve endings
- ❑ also movement of odontoblasts
- ❑ these movements cause deformation of nerve membranes
- ❑ increase of Na^+ ions permeability
- ❑ depolarization of $A\delta$ fiber membrane
- ❑ action potential is initiated



Odontoblasts deformation

- ❑ injury - any stimulation (thermal, mechanical, chemical, osmotic)
- ❑ odontoblasts - when deformed or injured - produce stimuli received by free nerve endings
- ❑ release of chemical substances
- ❑ electro-activation - change in the electric surface charges
- ❑ mechano-activation - movements of odontoblasts





Obr. 2.7 Aspirace jader odontoblastů (AO) do dentinových tubulů ihned po preparaci turbinou. V místě aspirace vrstva odontoblastů zcela chybí.




Defense function

- ❑ dentinal pain
- ❑ smear layer
- ❑ tubular sclerosis
- ❑ irritation (tertiary) dentine formation
- ❑ inflammation of the connective tissue

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- ❑ Smear layer - scaling, abrasion, attrition, caries, cavity preparation
 - ❑ microcrystalline debris (smear layer)
 - ❑ extends into the dentinal tubules - covers the dentinal surface (several μm thick)
 - ❑ reduction of dentine sensitivity and permeability (plugging of the tubules).

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- ❑ Tubular sclerosis - by milder or moderately irritating agents (slowly progressing caries, cavity preparation, abrasion, attrition, age changes)
 - ❑ peritubular dentine formation
 - and
 - ❑ intratubular calcification
 - ❑ the tubules become narrower and are closed

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- Tertiary (irritative, irregular) dentine formation
 - defensive barrier against caries progression

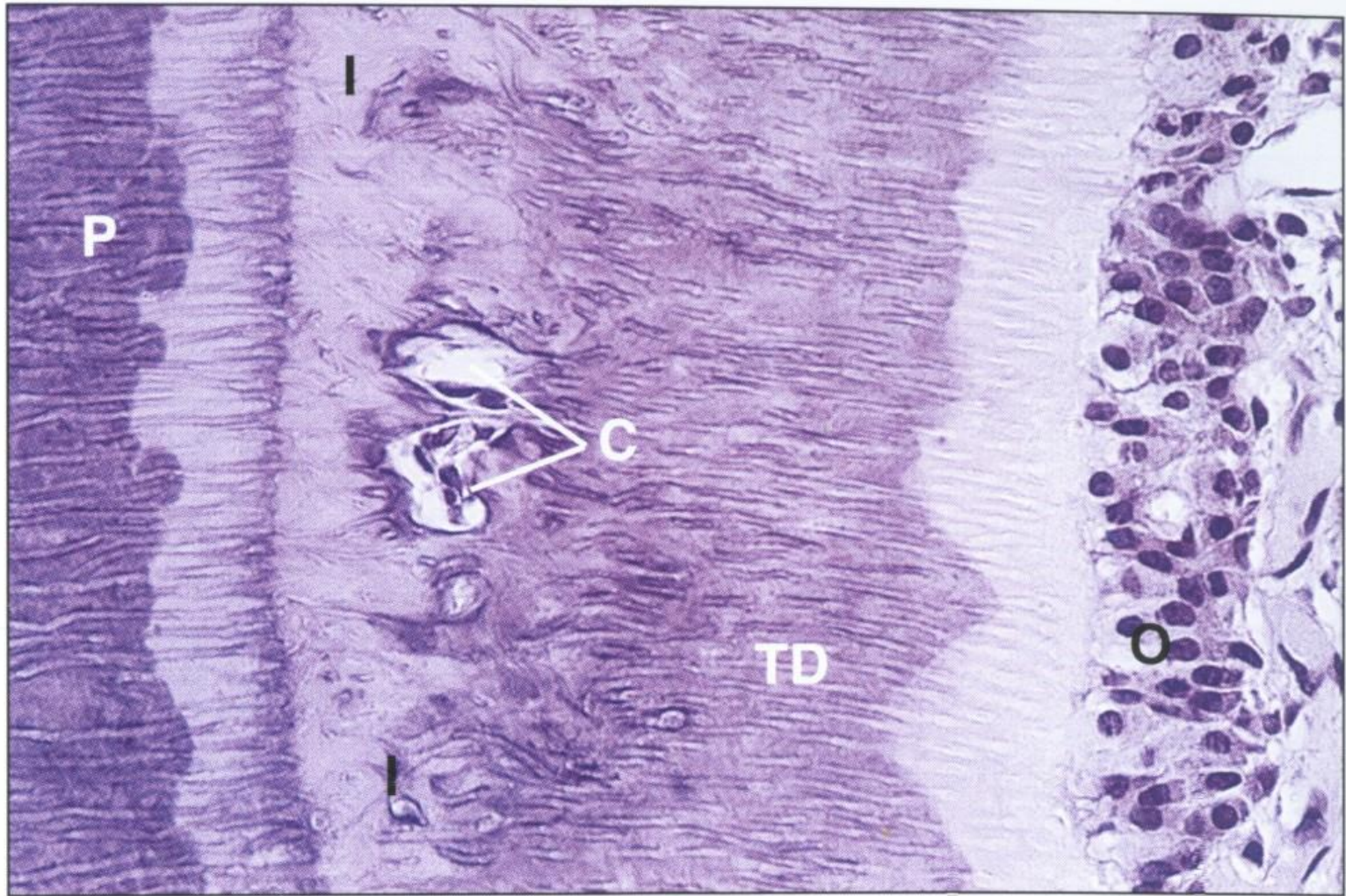


Fig 1-35 Interface dentin (I) between primary (P) and reparative tertiary dentin (TD). Note the irregular structure, including cellular inclusions (C), of the interface dentin. (O) Odontoblasts. (Hematoxylin-eosin stain; original magnification $\times 220$.)



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 the lightly stained, atubular interface dentin (I) and the dentinal tubules in the tertiary dentin. The odontoblasts lining the pulpal aspect of the tertiary dentin are short, and the cell-free zone is lacking in this area. (Hematoxylin-eosin stain; original magnification $\times 65$.)

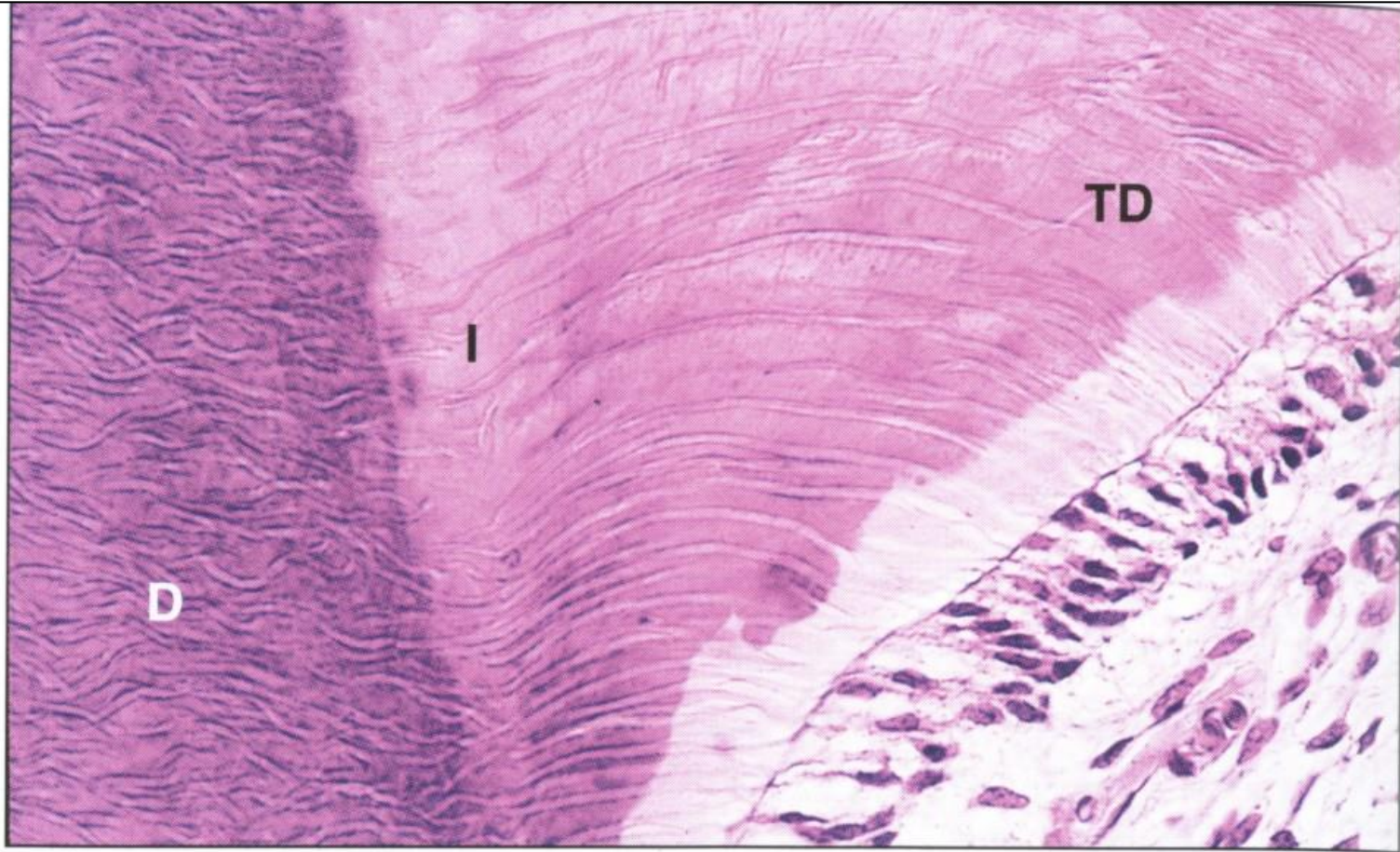


Fig 6-8 Higher magnification of the most peripheral part of the tertiary dentin (TD) shown in Fig 6-7. No or little communication is present between dentinal tubules in the dentin (D) and the tertiary dentin, except in the lower periphery of the illustration. (I) Interface dentin. (Hematoxylin-eosin stain; original magnification $\times 100$.)

□ **prolonged irritation**

- affects the plasma membrane and nucleus = first step towards inflammatory response

□ **severe irritation**

- deep cavity (less than 2mm from the pulp)
- intensified by:
 - inadequate water coolants
 - acid cements
 - inadequate insulation of metalfillings
 - microleakage of restorations

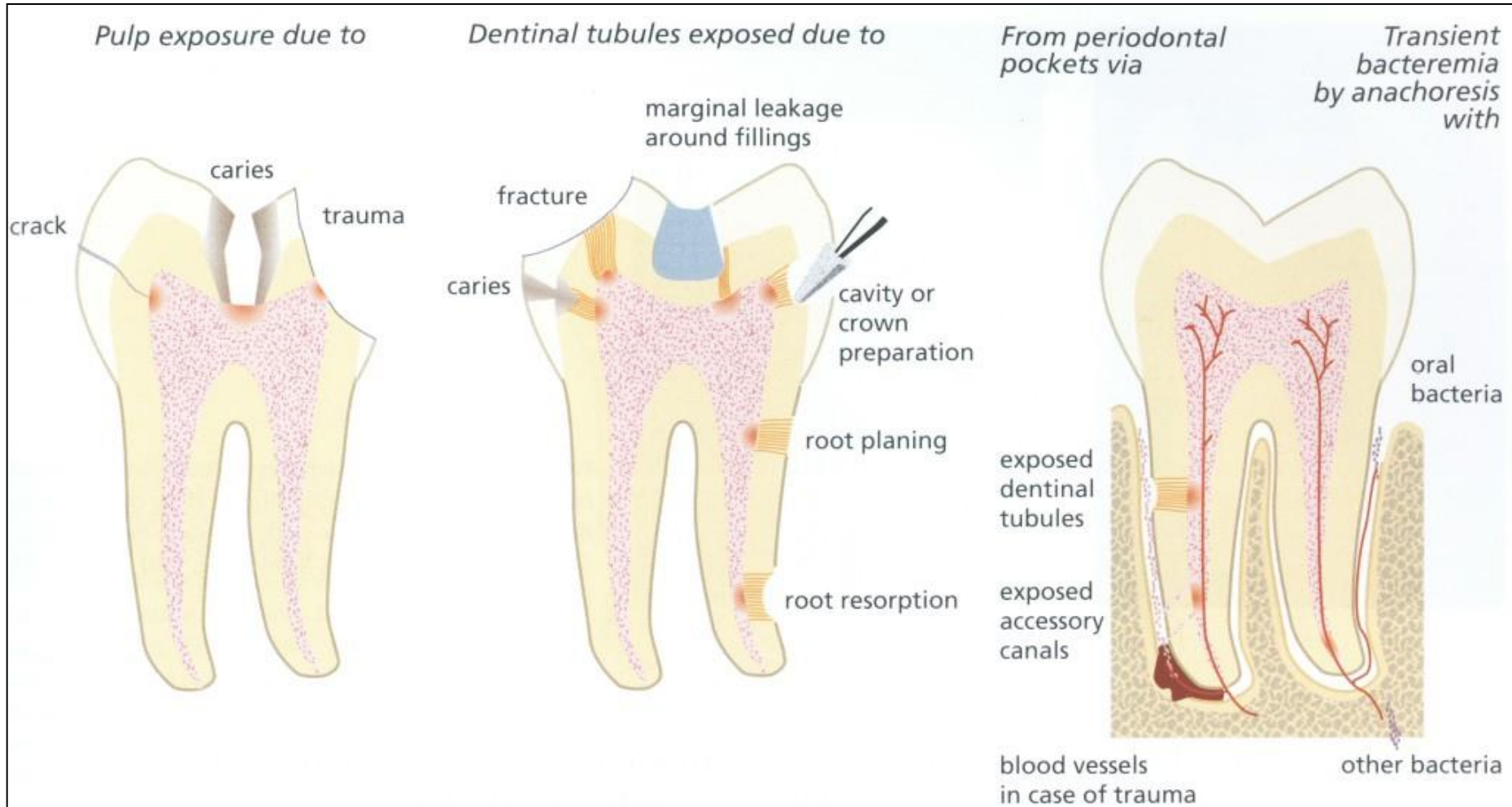


Fig. 8.1 Drawing illustrating the pathways of entry for micro-organisms into the root canal. Obvious ways of entry are pulp exposures due to caries or trauma. Potential pathways are cracks in enamel and dentine due to trauma, and dentinal tubules exposed by caries, fracture, cavity or crown preparation, marginal leakage around fillings, root resorption or root planing. From periodontal pockets, potential pathways are via exposed accessory canals, via exposed dentinal tubules or via blood vessels in the case of trauma. During bacteremia, blood-borne bacteria may colonize an inflamed or necrotic pulp (anachoresis). (See text for details.)

