RATIONALE OF ENDODONTIC TREATMENT

CONTENTS

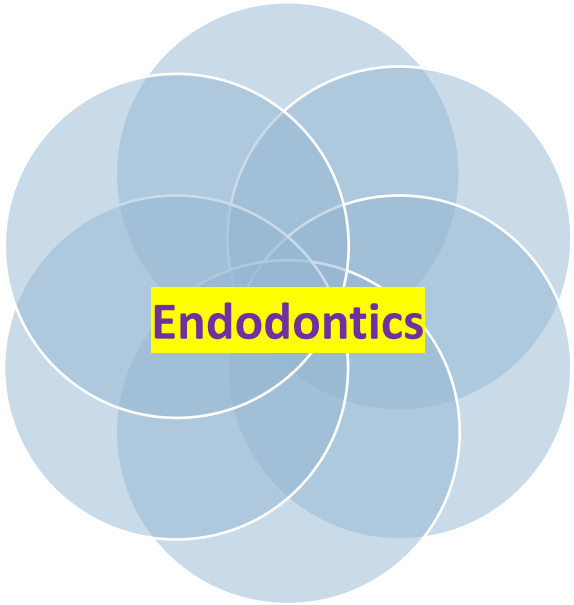
- Tissue Changes
- Endodontic Implications
- - Zones of Infection
- - Kronfeld’s Mountain Pass Theory
- Scope of Endodontics
- Principles of Endodontic Treatment
- Case Selection
- Contraindications of Endodontic Treatment
- Conclusion
- References

INTRODUCTION

- The word **"Rationale"** means fundamental reason or logical basis
- The rationale of endodontic therapy is based on the belief that a natural tooth functions more efficiently and comfortably than a bridge, partial denture, etc.
- **Endodontics** is that branch of dentistry that is concerned with the morphology, physiology and pathology of the human dental pulp and peri-radicular tissues

Basic clinical sciences

Treatment of Pulpal & Peri-radicular diseases and injuries



Biology of the normal pulp

Prevention

Etiology

Diagnosis

- Endodontic treatment is to eradicate the infection, to prevent microorganisms from infecting or re-infecting the root canal and/or peri-radicular tissues
- Endodontic infections

- Constitute almost 40—50% of the overall oral diseases

- Often the result of dental caries

- Most common prelude to endodontic treatment

- Reason of failures in endodontic therapy

Narayanan LL, Vaishnavi C. Endodontic microbiology. J Conserv Dent. 2010;13(4): 233-9.

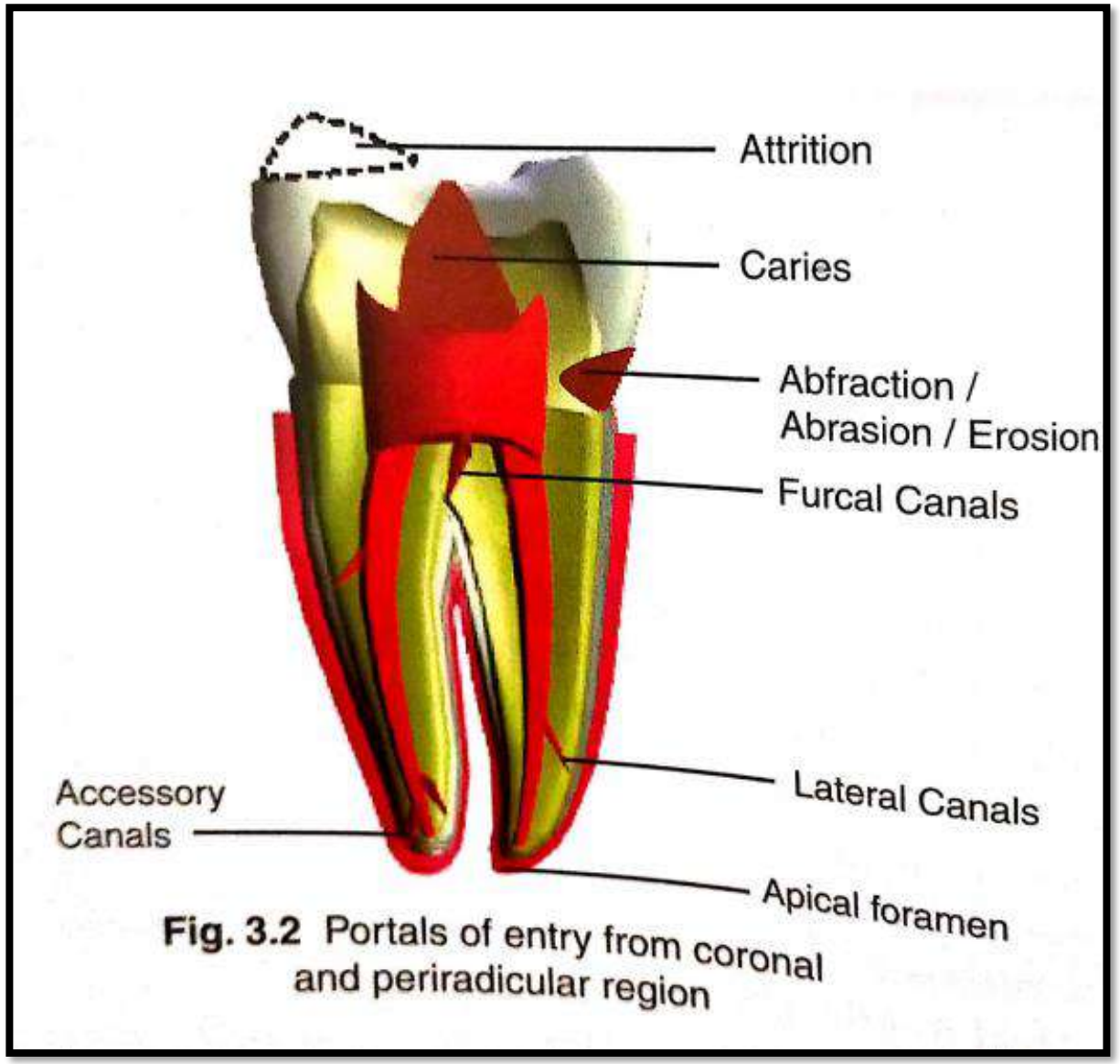
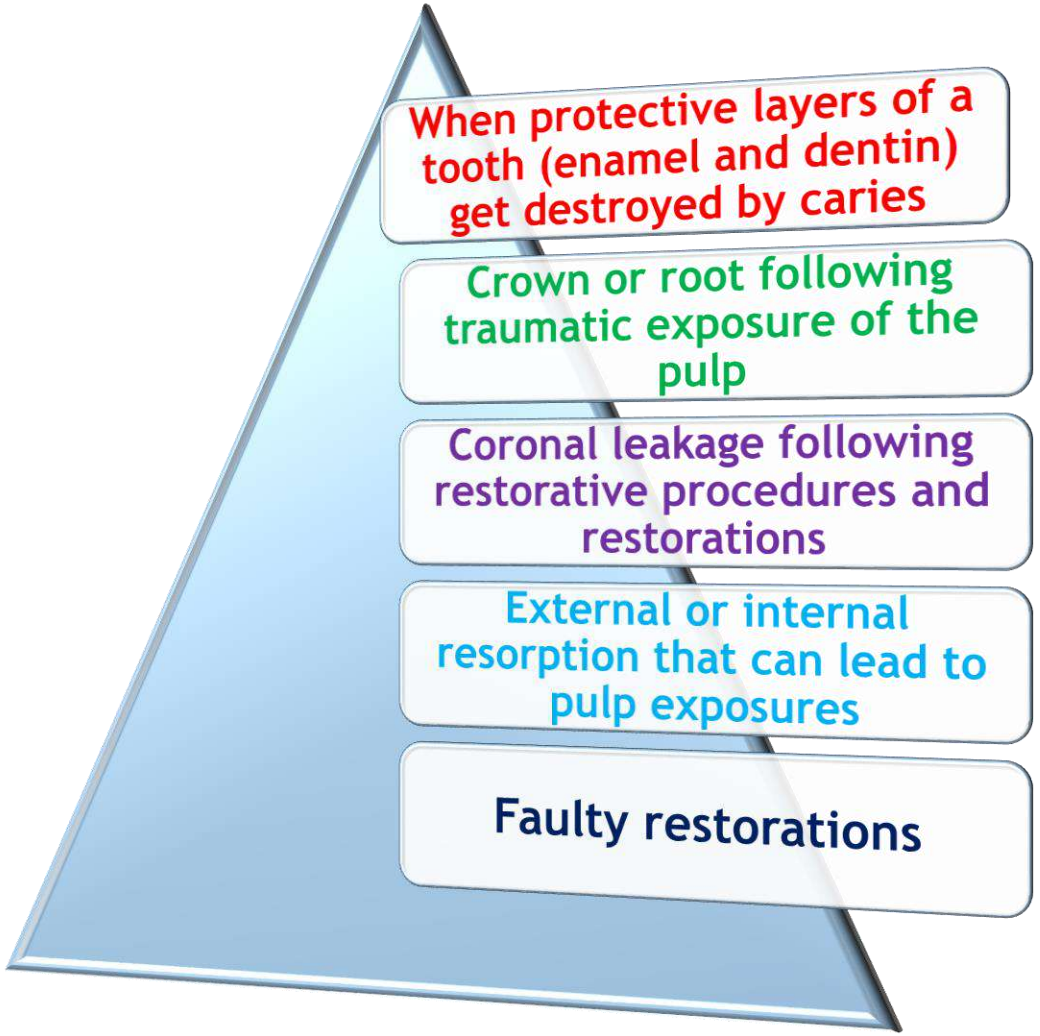


Fig. 3.2 Portals of entry from coronal and periradicular region

- **Entry through Open Cavity**
  - Common way of entry of microorganisms



Cohen's Pathways of the Pulp: 11th Edition

- Gingival Sulcus

Periodontal pathology or treatment

↓

Removal of cementum

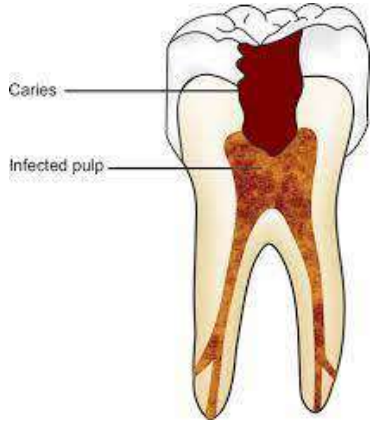
↓

Exposure of dentinal tubules to oral flora

↓

Entry of microorganisms into pulp via accessory and lateral canals

- Lymphatic or hematogenous route



Traumatized or inflamed pulp

↓

Localization of transient bacteria

↓

Anachoresis

↓

Endodontic infections

Inflammation

⇩

Phagocytic cells

⇩

Digest bacteria or cellular debris

⇩

Recognize antibodies

⇩

Attack and destroy foreign matter

⇩

Edema or Dilution of fluid

⇩

Neutralize the irritant

⇩

Limits spread of inflammation

* HOW INFLAMMATION IS DISTINCT FROM INFECTION????

INFLAMMATION	INFECTION
A protective response by the body to variety of etiologic agents (infectious or non-infectious)	Harmful microbial invasion into the body and their resultant ill-effects by toxins
Caused by non-living agents	Caused by invasion of cells by microorganisms
Always present with infection	Not always present with inflammation

Harsh Mohan Test Book Of Pathology 7th Edition

Infective Agents

Immunological agents

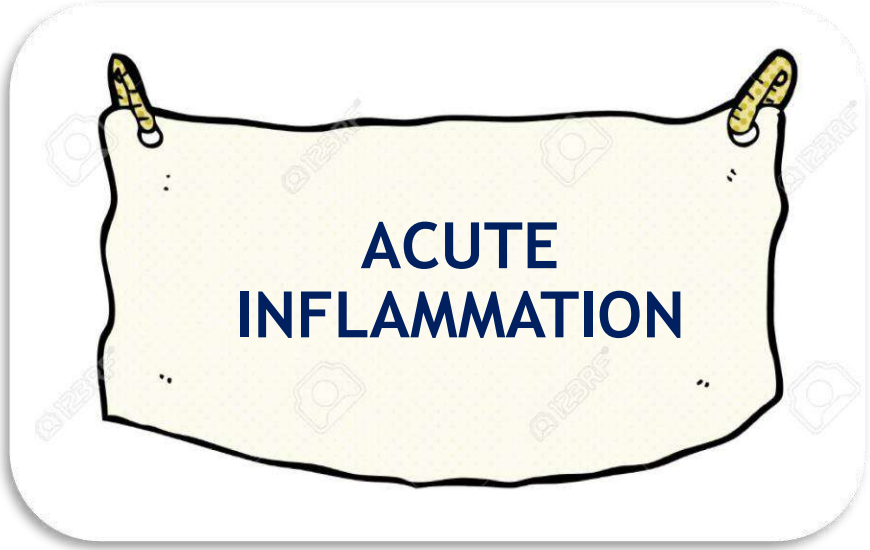
Physical Agents

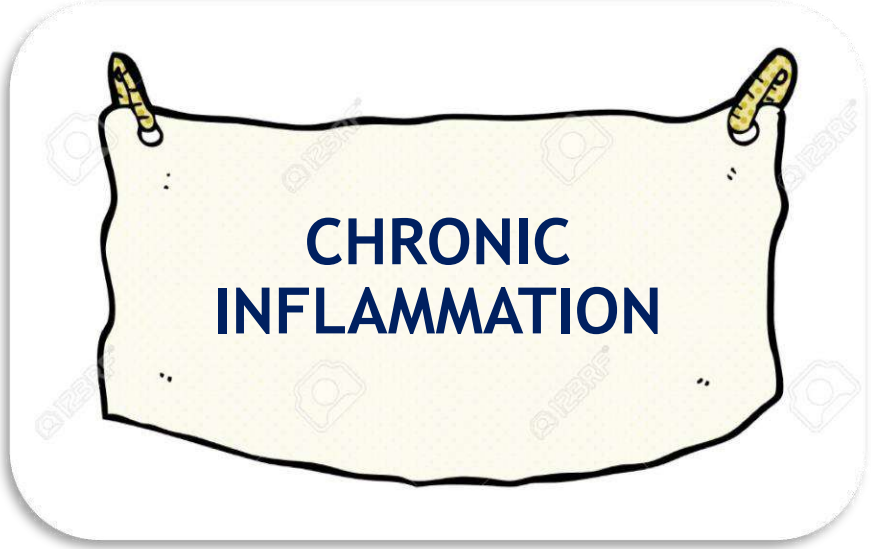
Chemical Agents

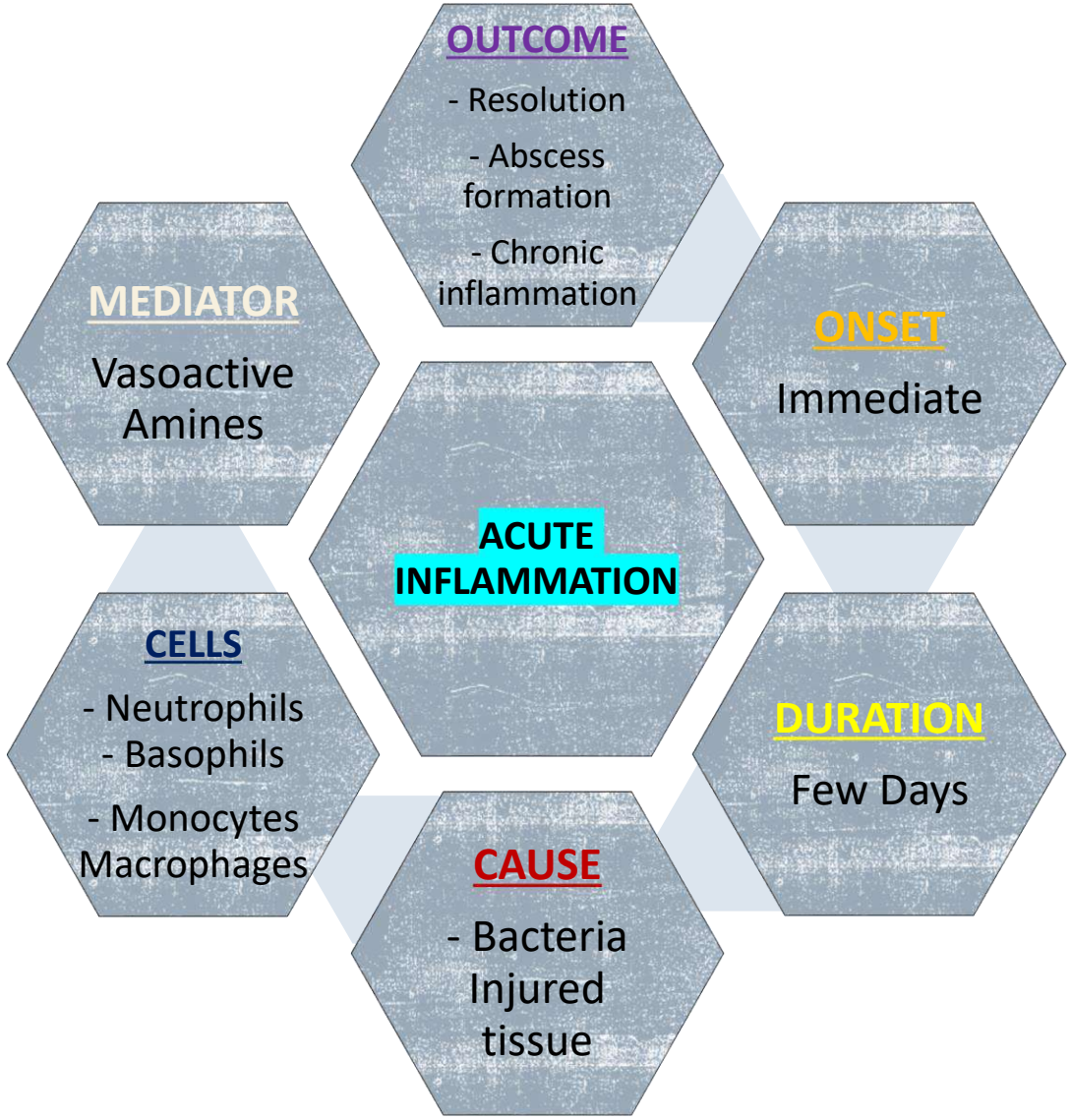
Inert Materials

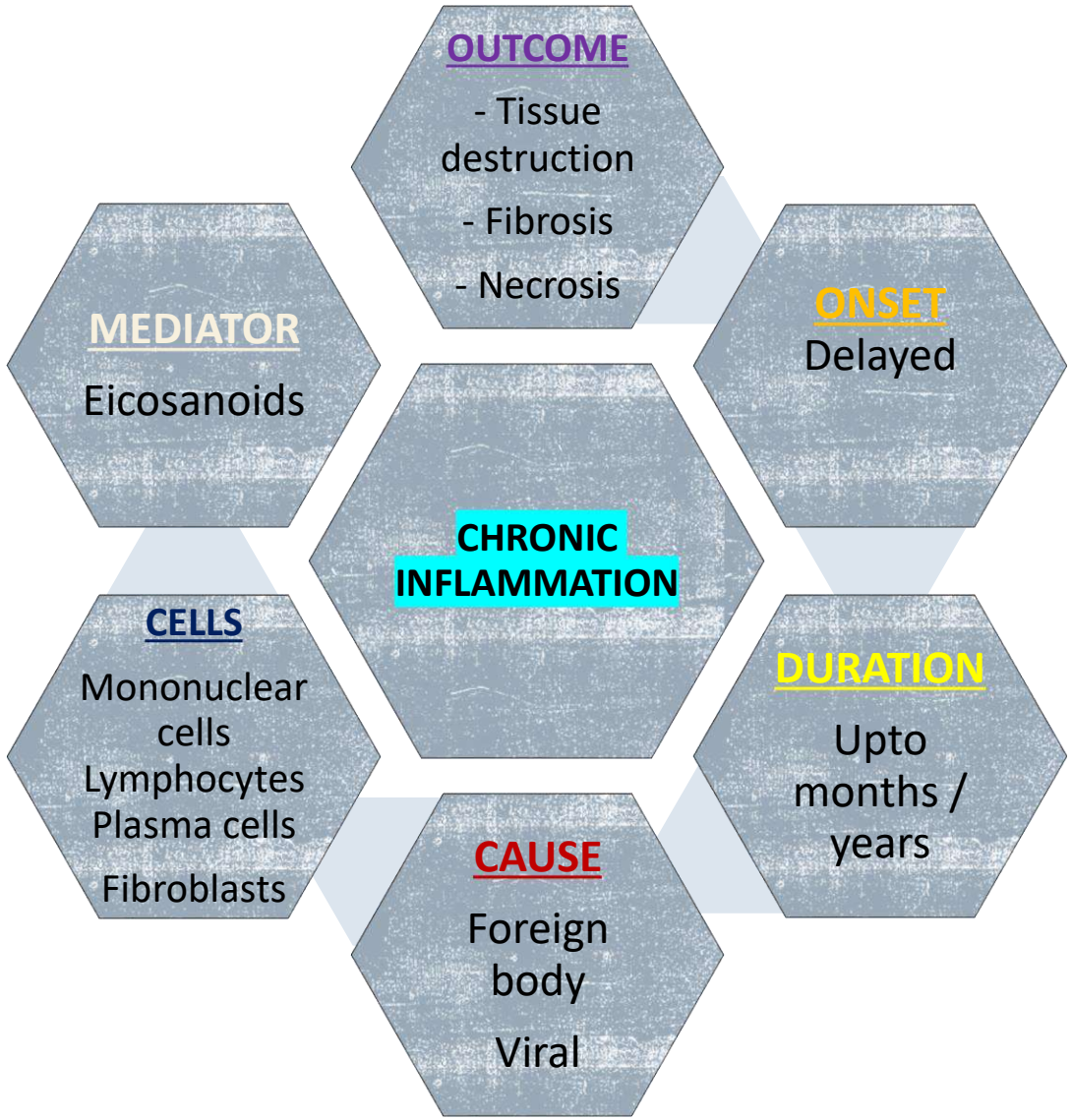
TYPES OF INFLAMMATION

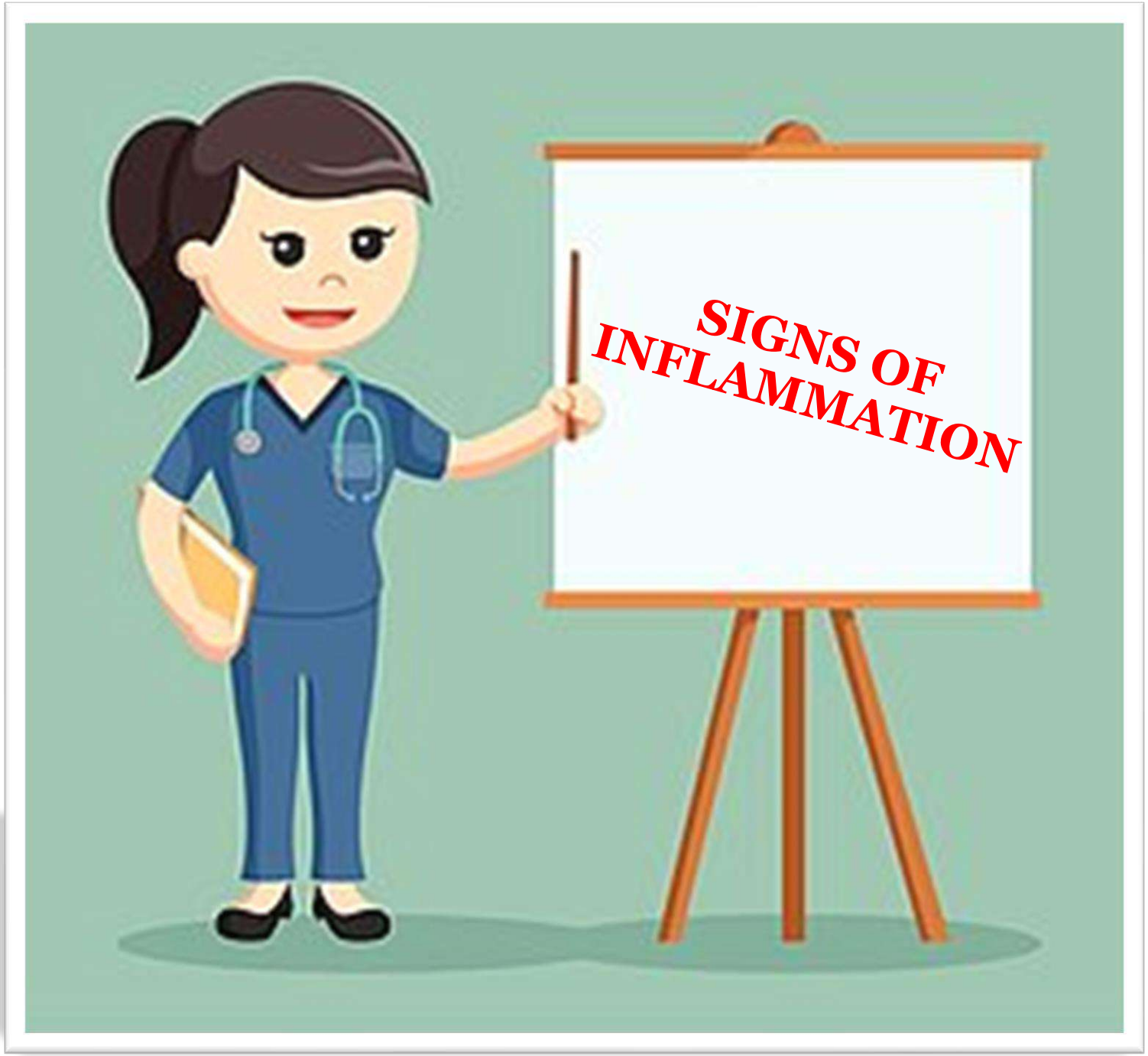
* Depending upon the defense capacity of the host and duration of response



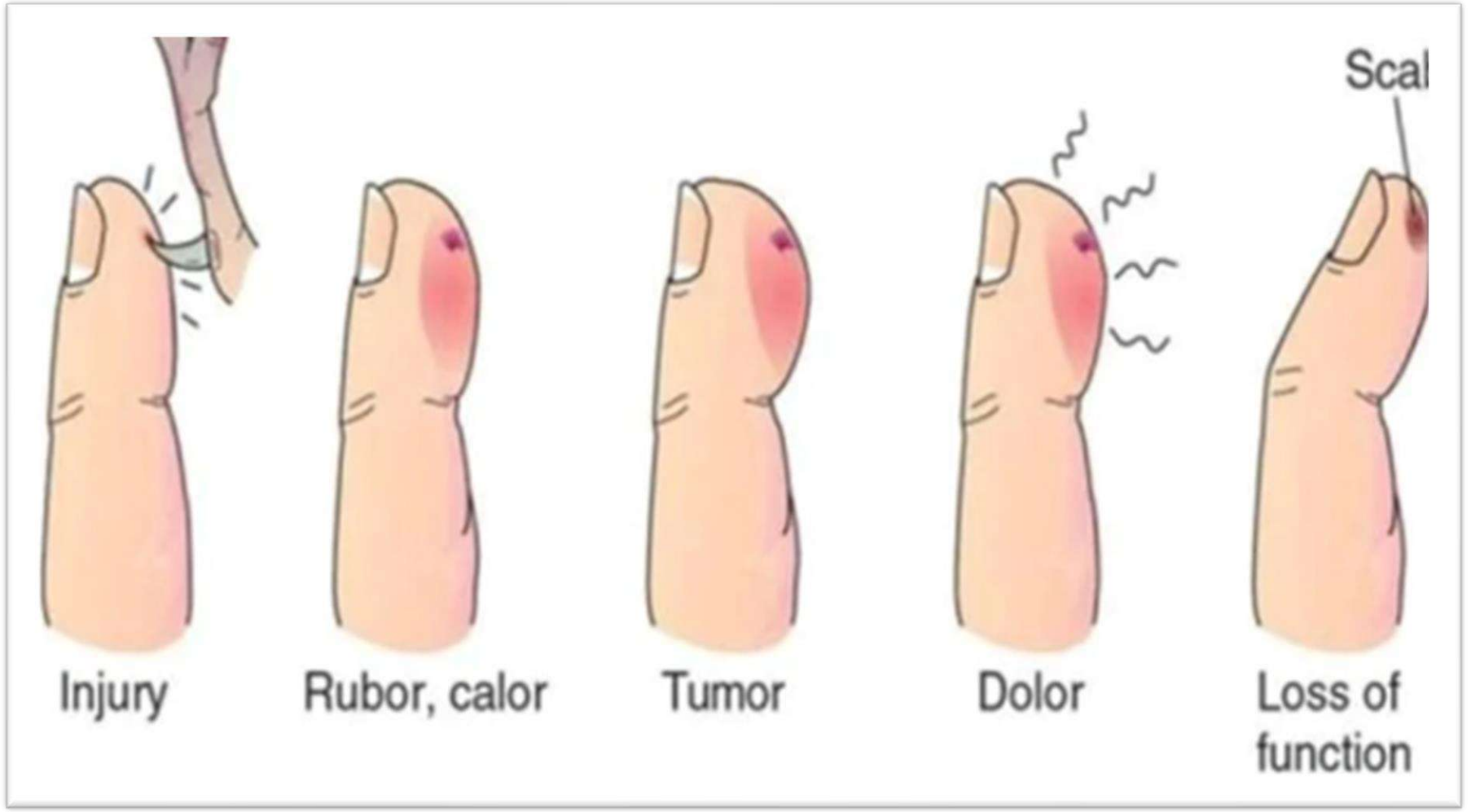



OUTCOME
- Resolution
- Abscess formation
- Chronic inflammation
MEDIATOR
Vasoactive Amines
ONSET
Immediate
ACUTE INFLAMMATION
CELLS
- Neutrophils
- Basophils
- Monocytes Macrophages
DURATION
Few Days
CAUSE
- Bacteria
Injured tissue


OUTCOME
- Tissue destruction
- Fibrosis
- Necrosis
MEDIATOR
Eicosanoids
ONSET
Delayed
CHRONIC INFLAMMATION
CELLS
Mononuclear cells
Lymphocytes
Plasma cells
Fibroblasts
DURATION
Upto months / years
CAUSE
Foreign body
Viral


SIGNS OF
INFLAMMATION

GIVEN BY CELSUS 1st century AD & VIRCHOW



Harsh Mohan Test Book Of Pathology 7th Edition

Pain

Action of **cytotoxic agents** from cellular, microbial elements

Swelling

Filtration of macromolecules and fluids into the affected tissues

Heat

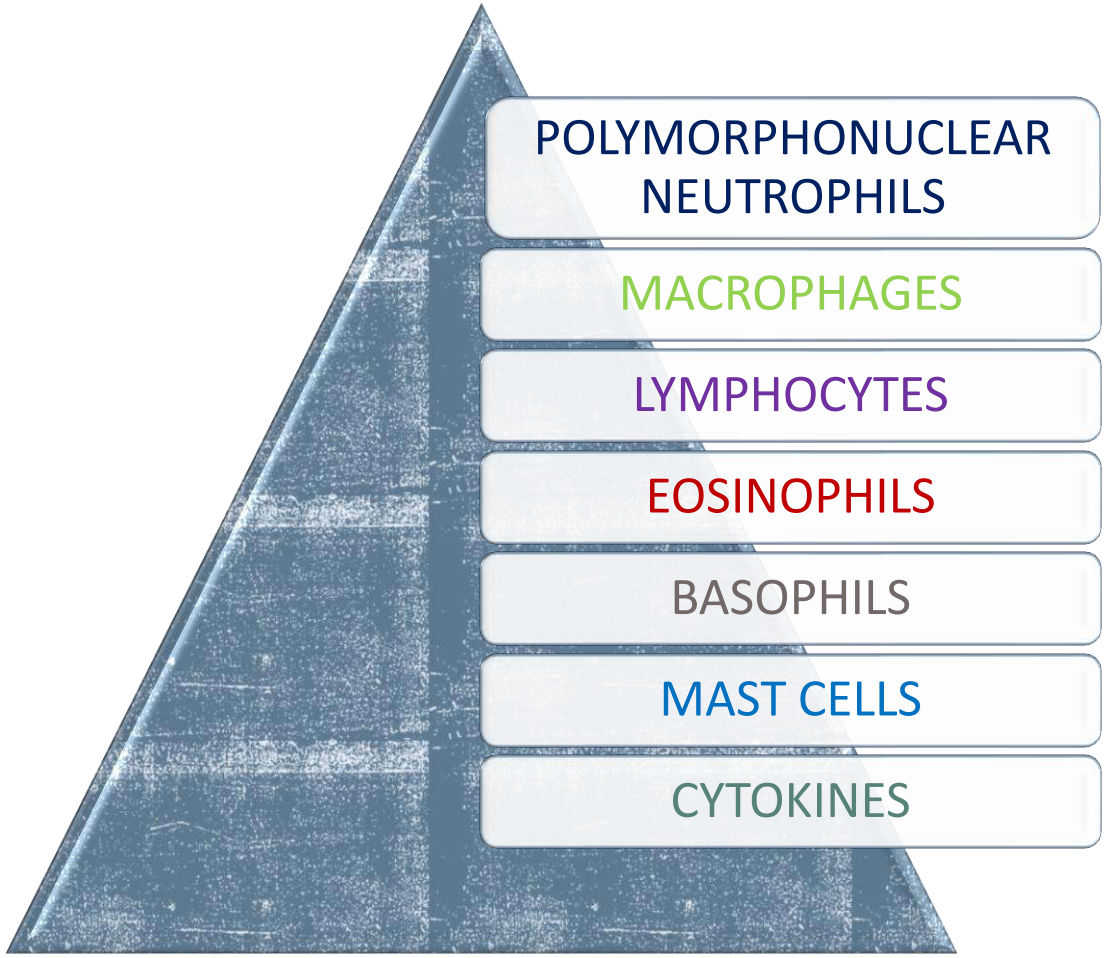
Vasodilatation of the vessels

Rushing of blood to the affected tissues

Function Disturbance

Changes in the affected tissues

CELLULAR MEDIATORS OF INFLAMMATION



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Morphology and Functions of Inflammatory Cells

POLMORPHS

1.Initial phagocytosis of bacteria

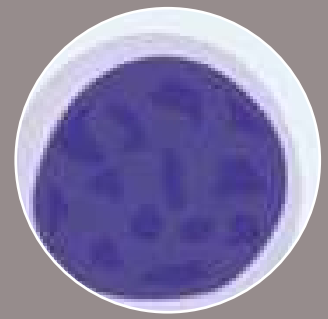
2.Acute inflammatory cell

MONOCYTES/MACROPHAGE

1.Bacterial phagocytosis

2.Chronic inflammatory cell

3.Regulate lymphocytes response

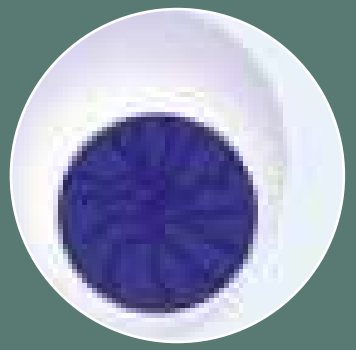


LYMPHOCYTES

1.Humoral and cell mediated response

2.Chronic inflammatory cell

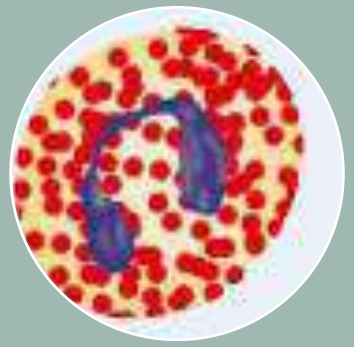
3.Regulate Macrophage response



PLASMA CELLS

1.Derived from B-cell

2.Chronic inflammatory cell

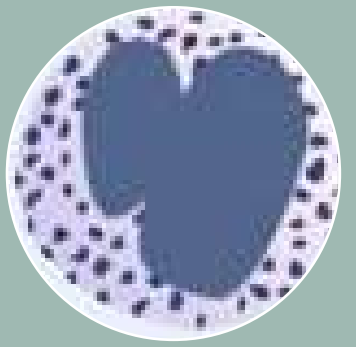


EOSINOPHILS

1.Allergic states

2.Parasites infection

3.Chronic inflammatory cell

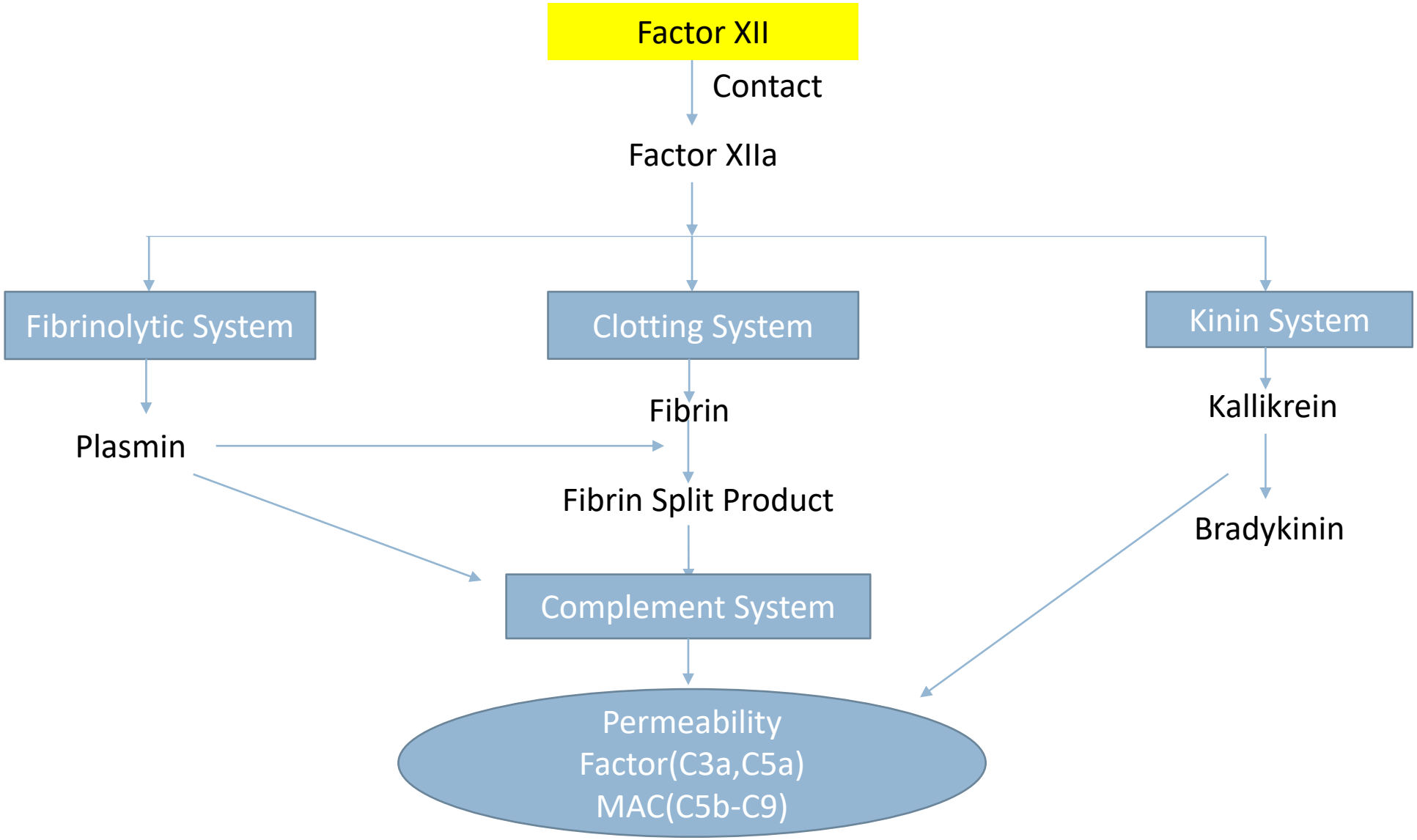


BASOPHILS/MAST CELL

1.Receptor for IgE molecules

2.Dense granules

❑ Plasma Protein-derived Mediators (Plasma Proteases)



Robins And Coltran Pathology Of Disease 8th edition

The action of activated component system in inflammation are as under:

- C3a,C5a,C4a (anaphylatoxins)- Activate mast cells and basophils to release of histamine
- C3b- An Opsonin
- C5a- Chemotactic for leukocytes
- **Membrane attack complex (MAC)**(C5b-C9)- A lipid dissolving agent that causes holes in the phospholipid membrane of cell

Robins And Coltran Pathology Of Disease 8th edition

INFLAMMATION OF DENTAL PULP

- Local physiologic reaction of the body to noxious stimuli or irritants

- Local response of living mammalian tissues to injury from any agents

- A body defense reaction in order to eliminate or limit the spread of injurious agent, followed by removal of necrosed cell and tissue

- John Hunter(1793): Inflammation is not a disease but a non-specific response that has a effect on its host

- Inflammation may be either symptomatic or asymptomatic

- Recognized only at the histologic level

- Depend on type of cells in the lesion

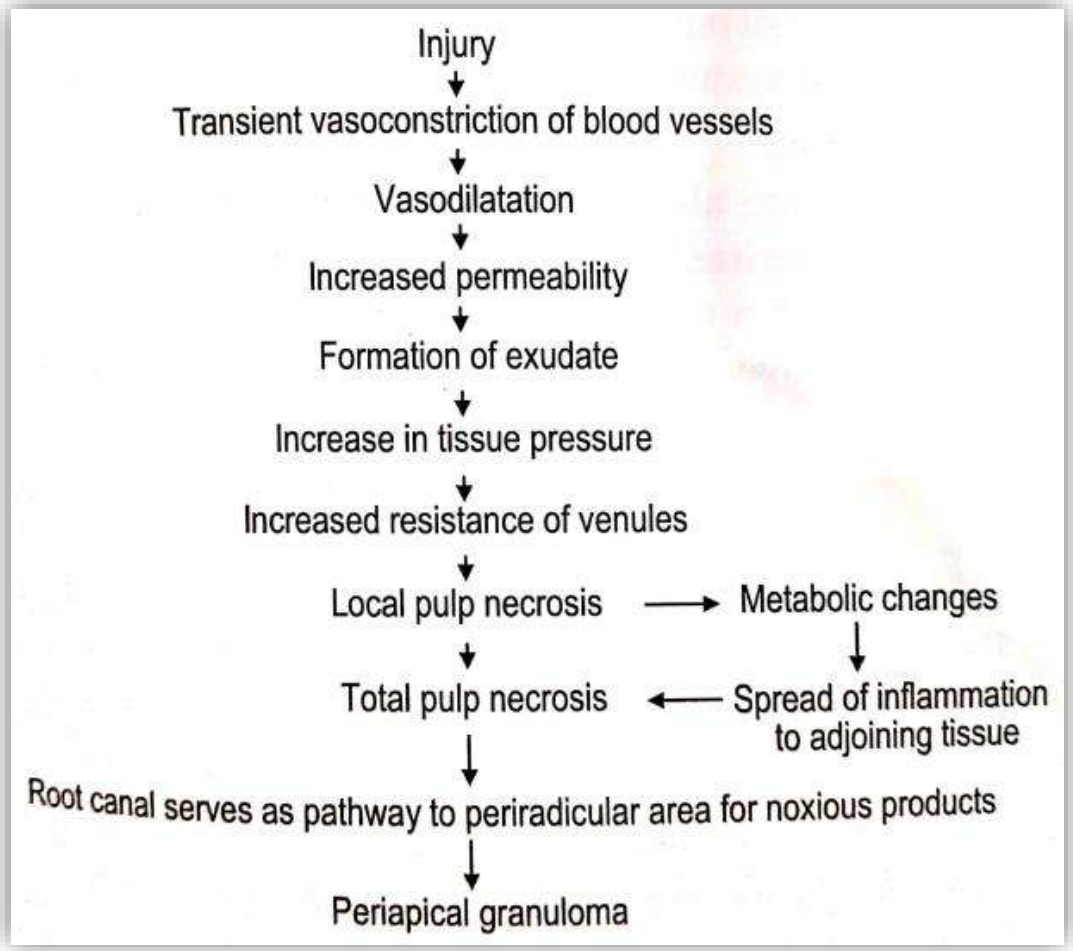
- **Symptomatic acute inflammatory Lesion**

- Polymorphonuclear neutrophil

- **Asymptomatic chronic inflammation**

- Lymphocytes, Plasma cells, Monocytes, Macrophages

VASCULAR CHANGES



CELLULAR CHANGES

- **Acute inflammation**

- PMNs (/neutrophils)

- **Chronic inflammation**

- Monocytes - Lymphocytes

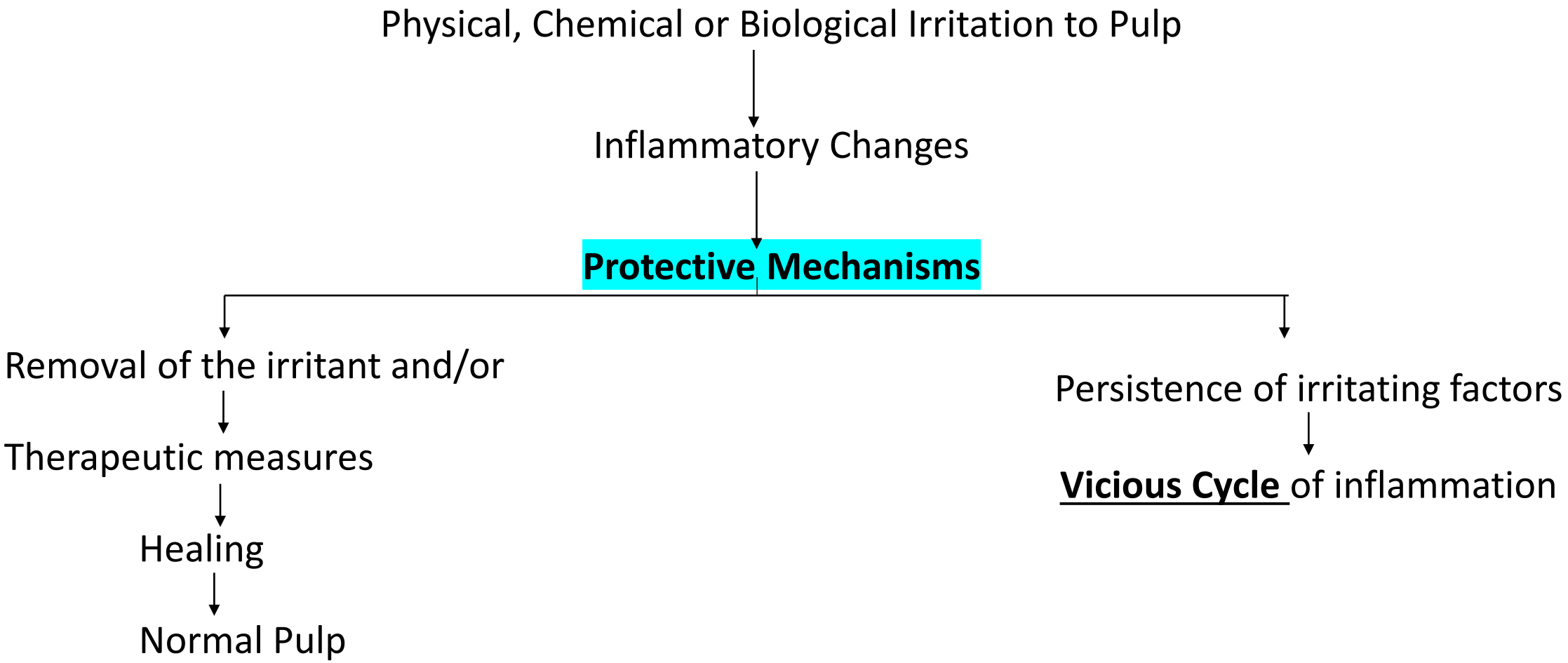
- Macrophages - Plasma cells

- **Other Cells**

- Eosinophils - Basophils

Mithra N. Hegde Textbook of Endodontics:2nd Edition

Sequelae of Pulpal Inflammation



Protective Mechanisms

Limits the Pressure increase within the affected Pulp

↓

Net absorption of interstitial fluid from adjacent capillaries in uninflamed tissues

Increased interstitial fluid pressure

↓

- Increases lymphatic drainage
- Oppose further filtration

Discontinuity in the endothelium and fenestration of pulpal capillaries

↓

Facilitate exchange mechanisms

* Mechanism of Vicious Cycle of Pulpal Inflammation (Kim S. [1985] and Van Hassel H.J. [1971])

Irritation

(Dental caries, trauma, or operative procedures)

⇩

Localized inflammation

Vasodilatation

Increased local tissue pressure

⇩

Localized necrosis

Venous collapse

Reduced blood flow

Waste product accumulation

Progression of inflammation

Wider zone of inflammation

Spread of vascular disturbance

Generalized necrosis

Necrosis of tissue

Release of lysosomal enzymes

Extensive collagen destruction

PERI-RADICULAR MANIFESTATIONS

Overwhelming of **inflammatory response** of pulp

⇩

Partial or total necrosis

⇩

Root canal = Pathway to the peri-radicular area

⇩

Noxious products of tissue necrosis

⇩

Inflammatory and immunologic responses

⇩

On reaching the peri-radicular area

⇩

Release of noxious products

Bone resorption

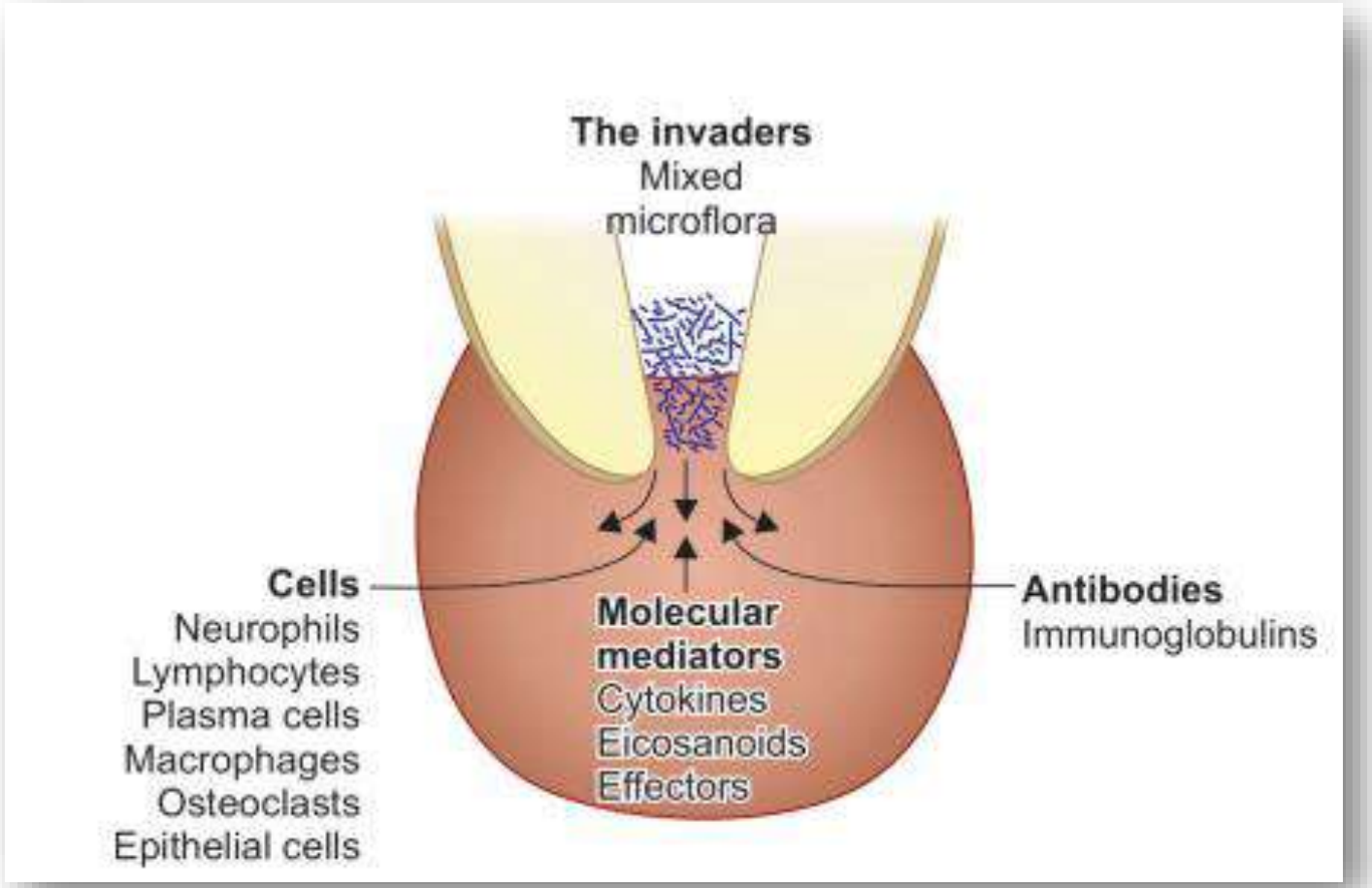
⇩

Formation of granulation tissue

* Immunologic reactions play in the physiology and pathology of the peri- radicular tissues

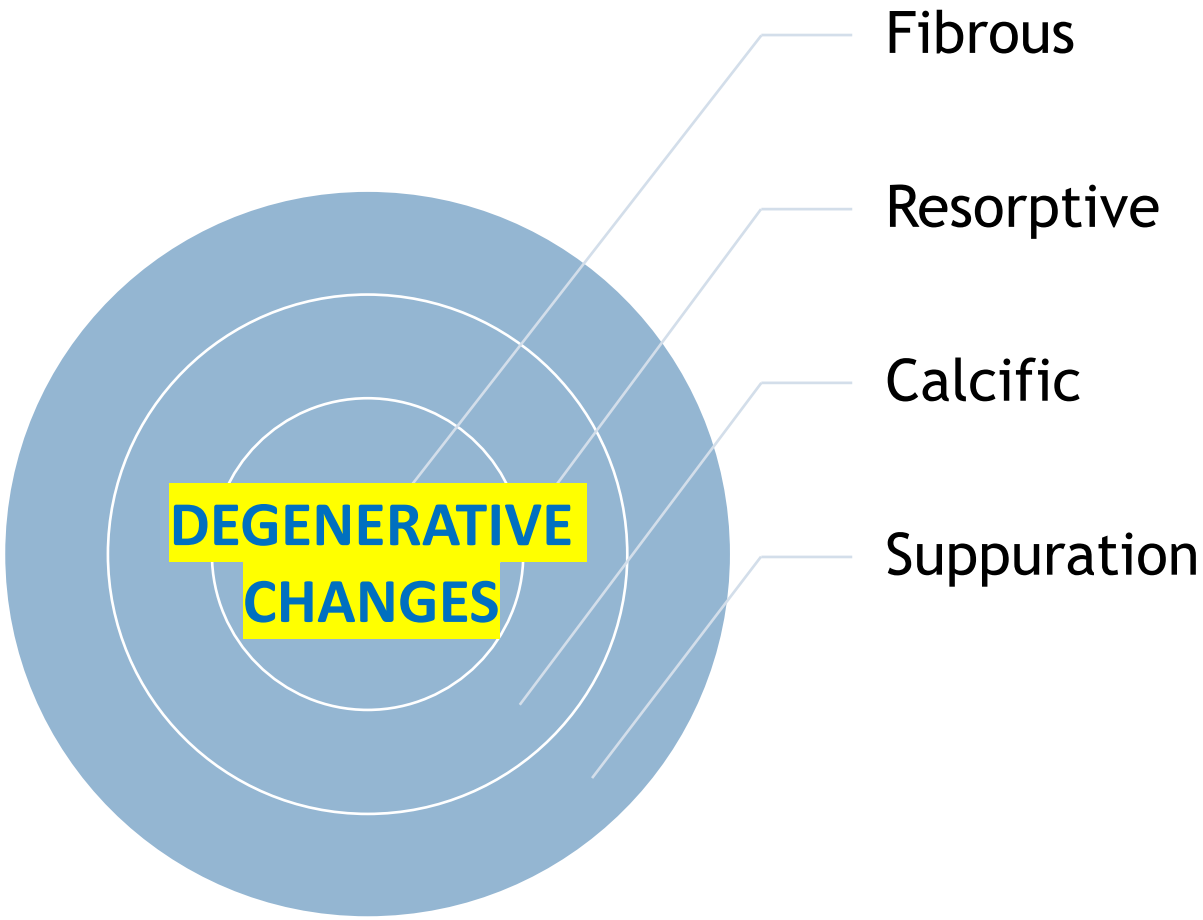
- Endodontic flare-ups are mediated by IgE reactions

- Bone resorption is mediated by a lymphokine called **osteoclast-activating factor**



Schematic representation of inflammatory response of peri-radicular area to root canal irritants

TISSUE CHANGES FOLLOWING INFLAMMATION



Requisites for Suppuration

- Necrosis of tissue cells
- Sufficient number of polymorphonuclear leukocytes
- Digestion of the dead material by proteolytic enzymes

Degeneration

⇩

Thrombosis of the blood vessels

⇩

Damage to the tissue cells

⇩

Release of leukotoxin

⇩

Injury to Polymorphonuclear cells

⇩

Release proteolytic enzymes

⇩

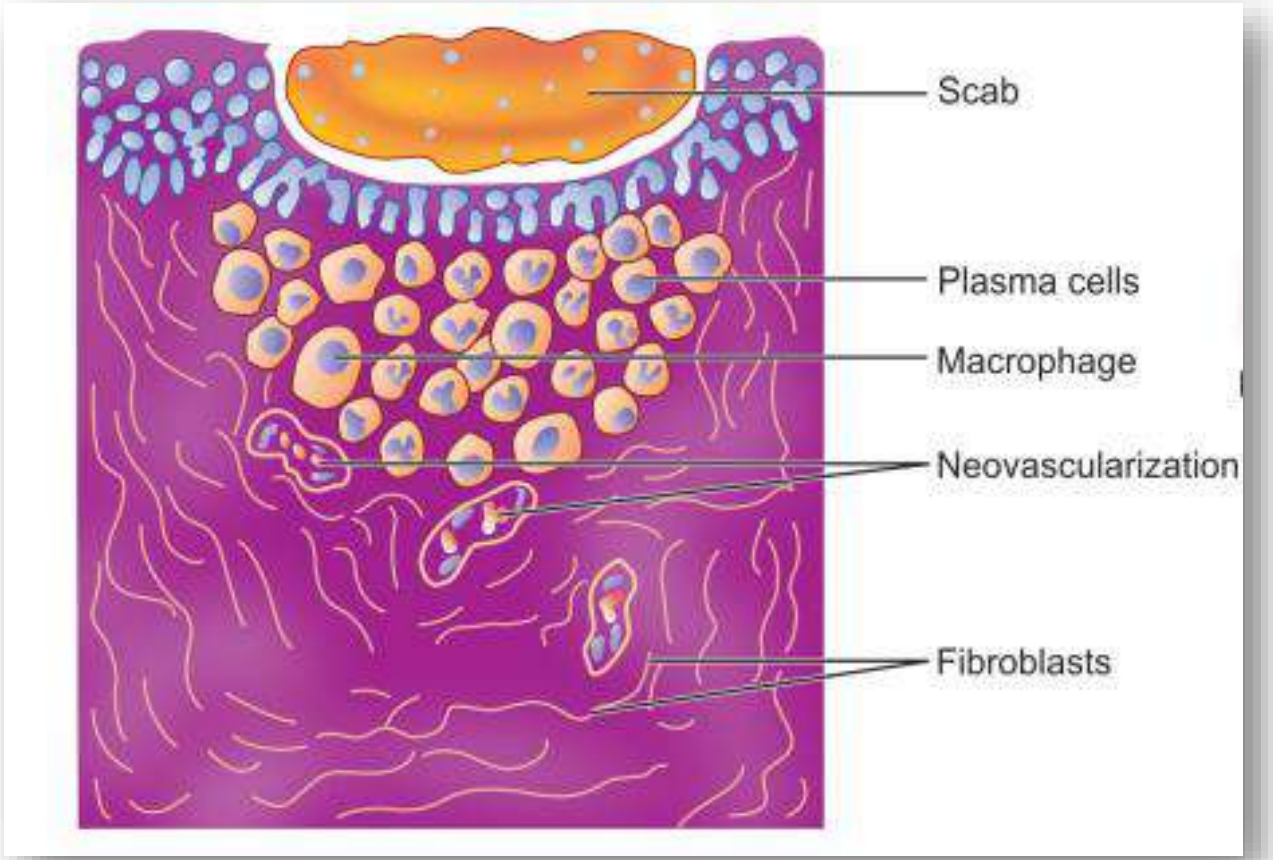
Necrosis

⇩

Liquefaction of the dead tissue

⇩

Suppuration or formation of pus



Inflammatory cells present at the healing site

* Center of the inflamed area (Plasma cells and macrophages)

* Periphery (Proliferation or repair cells, i.e. Fibroblasts)

PROLIFERATIVE CHANGES

- Produced by irritants mild enough to act as stimulants

* **Center** of the inflammatory area

Irritant (Strong)

Degeneration or destruction

⇩

* **Periphery** of the inflammatory area

Irritant (Mild)

Proliferation

* If the tissue is in apposition

(An incision for root resection)

Fibroblastic repair will take place

⇩

* When a gap is present between the tissue parts

Repair with granulation tissue (Resistant to infection)

ENDODONTIC IMPLICATIONS

Root canal

⇩

Site of infection

⇩

Microorganisms do not move from the root canal to the peri-radicular tissues

⇩

Multiply sufficiently to grow out of the root canal

⇩

Metabolic or toxic products of tissue necrosis

⇩

Diffuse to the peri-radicular tissues

Theories of Spread of Infection

Oral Sepsis Theory
(W. D. Miller, 1888)

- Human mouth as focus of infection
- Necrotic pulp could act as a centre of infection resulting in alveolar abscess

Theory of Elective Localization
(E. C. Rosenow,1909)

- Bacteria located within a certain focus of infection could cause systemic infection by localizing within a specific target organ or tissue

Focus of infection
(Frank Billing, 1904)

- Circumscribed area of tissue, which is infected with exogenous pathogenic microorganisms
- Usually located near a mucous or cutaneous surface

Focal infection
(1950)

- Localized or general infection caused by the dissemination of microorganisms or toxic products from a focus of infection

Mechanism of Focal Infection

- Metastasis of micro-organisms from infected focus
- **Hematogenous** or **lymphogenous** spread

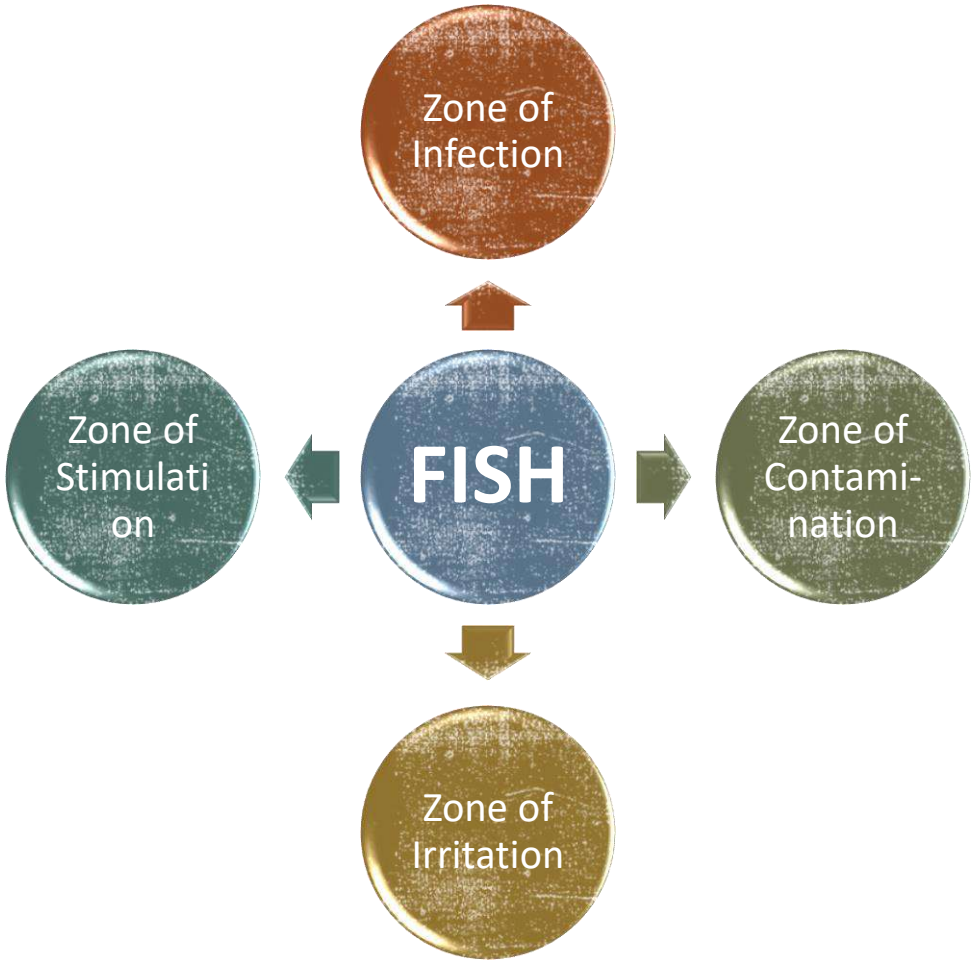
- Carrying of toxins or toxic by-products
- Through **blood stream** and **lymphatic channel** to site where they may initiate a hypersensitive reaction in tissues

Oral Foci of Infection

* Possible sources of infection in oral cavity which may later on set up distant metastasis

- Infected periapical lesions
- Periapical granuloma
- Periapical abscess
- Periapical Cyst
- Teeth with infected root canals
- Periodontal diseases with special reference to tooth extraction

* Described by Fish (1939)



Jaws of guinea pigs

⇩

Drilled openings in the bone

⇩

Packed wool fibers saturated with a broth culture

⇩

of microorganisms

⇩

Established experimental **foci of infection**

ZONE OF INFECTION

- Center of the lesion
- Contains PMNs

ZONE OF CONTAMINATION

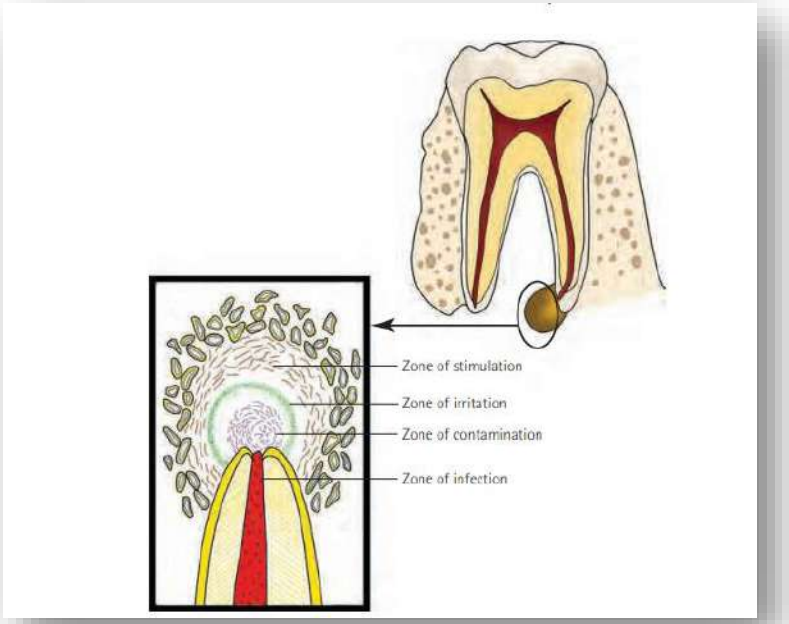
- Round cell infiltration
- Discharged toxins from the central zone

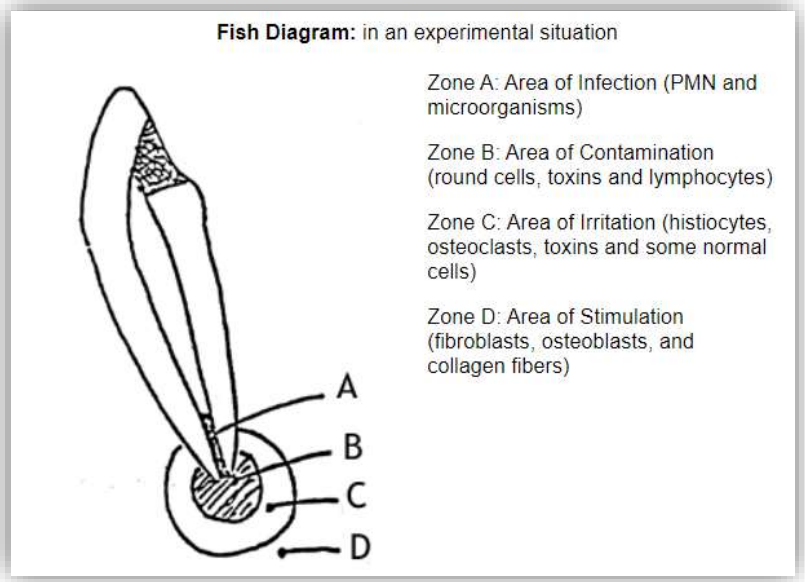
ZONE OF IRRITATION

- Macrophages and osteoclasts
- Diluted toxins
- Histologic picture that signifies a body's attempt to initiate repair

ZONE OF STIMULATION

- Fibroblasts and osteoblasts
- Mild toxin to be a stimulant





Toxic products released by the microorganisms (From A) are destructive to the periapical tissue

Periapical area (B) is surrounded by (C) and (D)

Delicate balance favoring Zones (A) and (B

Continuous existence of periapical pathosis

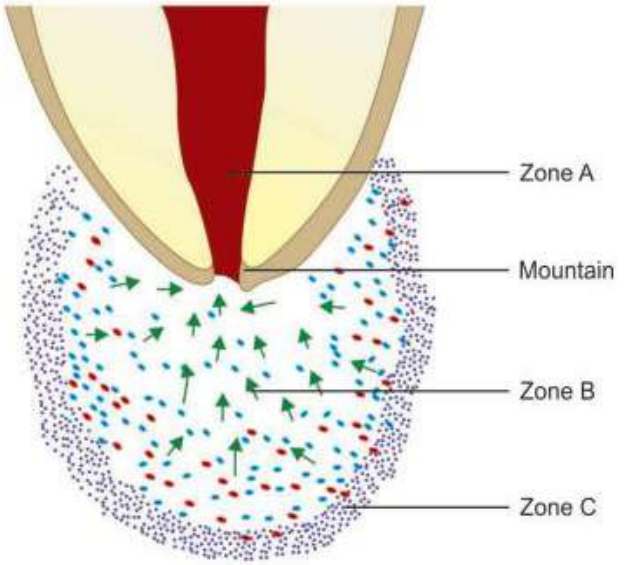
Zone (D) is capable of repairing the area

Elimination of Zone A (the source of the infection) by endodontic therapy will tip the balance to favoring the activity of Zone D

Repair of the periapical tissue and retention of the tooth

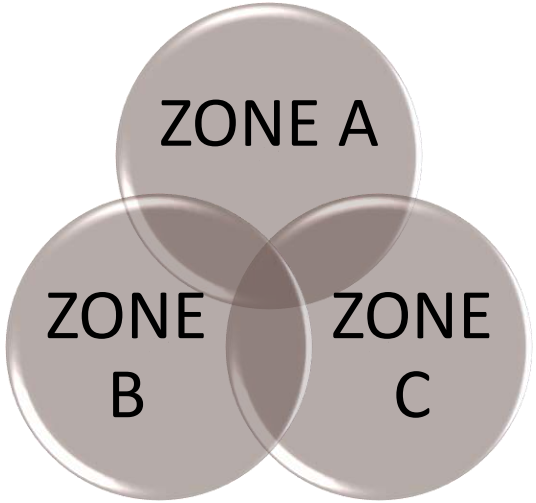
Columbia private Ivy League research university in New York City, Wikipedia

Kronfeld's Mountain Pass theory (1939)

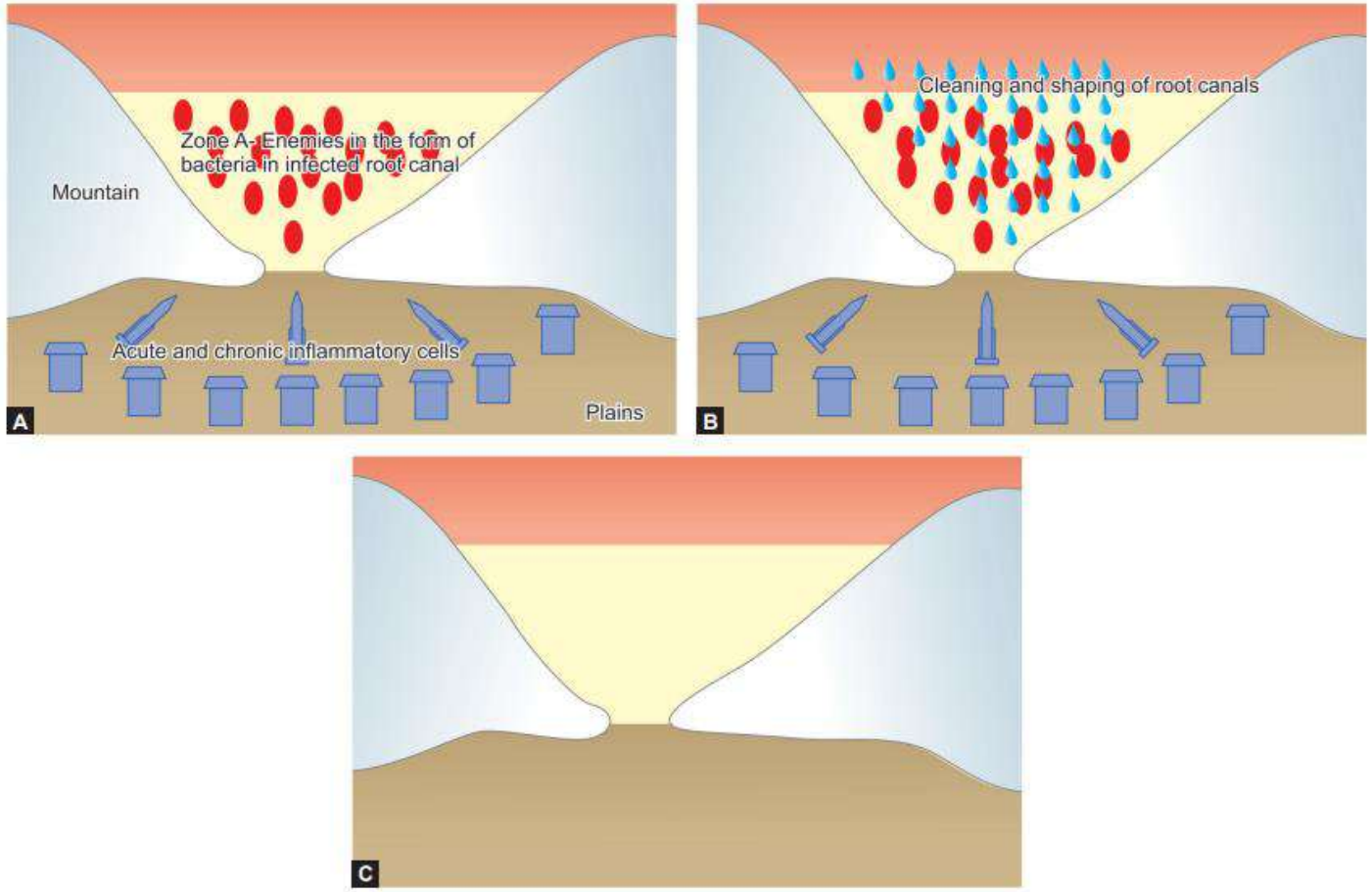


- Helps to understand the close relationship between **pulp and periodontium**

- Employed Fish concept to explain the tissue reaction in and around the granulomatous area

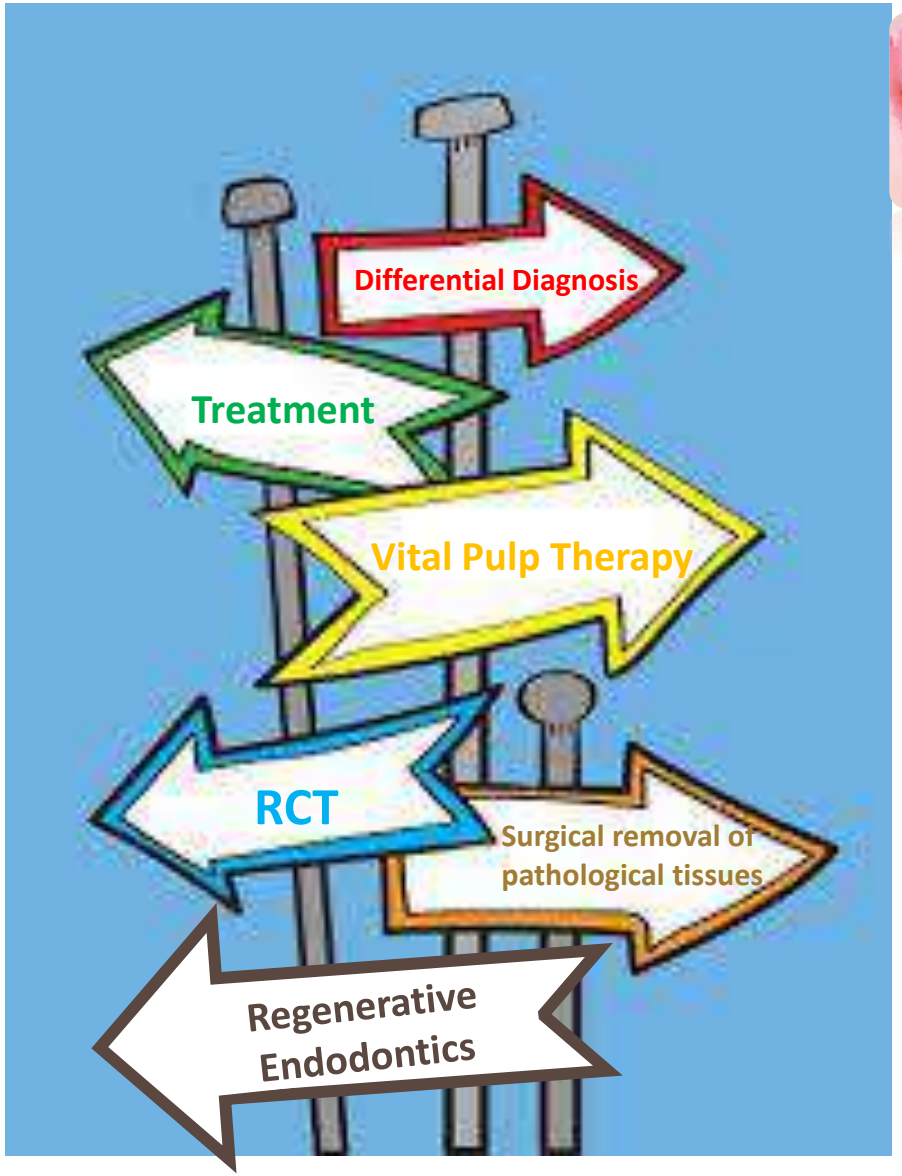


NISHA GARG: TEXTBOOK OF ENDODONTICS; 4TH EDITION



(A) Enemies in form of microorganisms of infected root canals need pass to enter the plains which is guarded by soldiers in form of acute and chronic inflammatory cells
(B) Cleaning and shaping of root canals neutralize the enemies by attacking from behind
(C) If enemies are eliminated by the attack, soldiers are not required. But if mass attack of invaders occur, battle between invaders and soldiers result in acute inflammation.

SCOPE OF ENDODONTICS



Endodontic Implants

- Introduced back in **1960**

- Artificial metallic extension

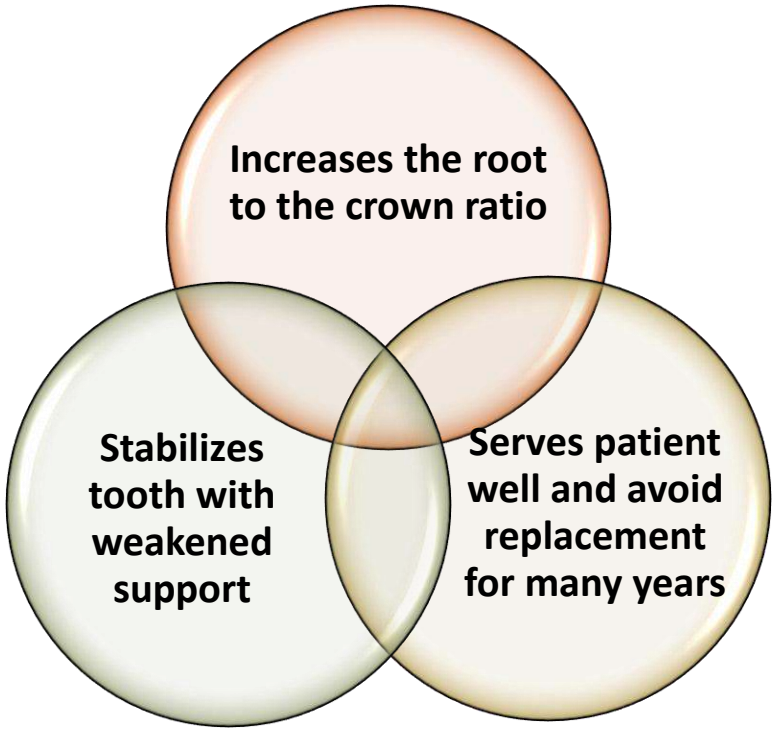
- Safely extend out through the apex of the tooth into sound bone

Orlay	First to use and advocated endodontic implants
Frank	- Standardized the technique - Developed proper instruments and matching implant
Frank & Abram	Showed properly placed endodontic implant was accepted by the apical tissues

Yadav RK, Tikku AP, Chandra A, Wadhwani KK, A, Singh M. Endodontic implants. Natl J Maxillofac Surg 2014;5:70-3

INDICATIONS

- Periodontal bone loss
- Involvement of a single tooth
- Difficult in extraction and replacement
- Horizontal fracture of a tooth
- Too weak remaining coronal portion
- Unfavorable crown-root ratio
- Pathological resorption of the root apex due to chronic abscess
- Pulpless tooth with unusually short root



Yadav RK, Tikku AP, Chandra A, Wadhwani KK, A, Singh M. Endodontic implants. Natl J Maxillofac Surg 2014;5:70-3

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JOURNAL OF ENDODONTICS

Printed in U.S.A
VOL. 15, NO. 10, OCTOBER 1989

Endodontic Endosseous Implants: Case Reports and Update of Materials

Russell M. Larsen, DDS, Jimmy Ray Patten, DDS, PhD, and Blake E. Wayman, DDS, MS

Two cases are presented **with 5-yr follow-ups** in which Vitallium endodontic implants were used successfully to improve the crown-root ratio of central incisors compromised by trauma

* A **15-yr-old** male who had been hit in the mouth with a baseball 4 days earlier presented for treatment

Horizontal mid- root fracture of 11
Intruded apical segment

3 weeks later
Apical segment was removed
Placement of endodontic implant

1-yr postoperative radiograph shows good osseous healing

2-yr post-treatment radiograph shows that the Vitallium implant has been well accepted

5-yr postoperative radiograph shows excellent osseous regeneration and healing

* A 53-yr-old female fallen 6 days earlier and intruded the maxillary central incisor

Apical radiolucency with 11

Following 2 week of stabilization the canal was filled with calcium hydroxide

Due to severe mobility, the Vitallium implant was placed 3 months after the accident

15-month recall radiograph shows a slight thickening of the periodontal ligament and that the Vitallium implant has been well tolerated

5 year recall shows some external resorption, but the tooth is stable and asymptomatic


PRINCIPLES

Factors Determining Case Selection

Competent

Excellent endodontic therapy

Motivation

Financial ability

Age

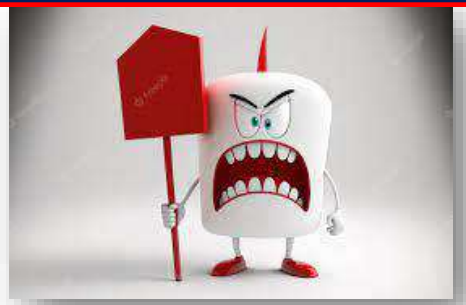
Physical Disability

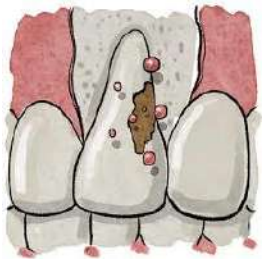
Occupation

Physical Disorders

Columbia private Ivy League research university in New York City, Wikipedia

CONTRAINDICATIONS!!!!





CONCLUSION

- The rationale for endodontic treatment is to eradicate the infection, to prevent microorganisms from infecting or re-infecting the root canal and/or peri-radicular tissues
- Must seal the root canal system three-dimensionally
- Prevent tissue fluids from percolating in the root canal and toxic by-products from both necrotic tissue and microorganisms regressing into the peri-radicular tissues

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THANK YOU!!!!!