RATIONALE OF ENDODONTICS

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CONTENTS

- INTRODUCTION
- THEORIES OF SPREAD OF INFECTION
- CULPRIT OF ENDODONTIC PATHOLOGY
- PORTALS FOR ENTRY OF MICROORGANISMS
- INFLAMMATION
- TISSUE CHANGES FOLLOWING INFLAMMATION
- INFLAMMATORY CELLS
- INFLAMMATORY RESPONSE TP PERIAPICAL LEASION
- ANTIBODIES
- ROLE OF IMMUNITY IN ENDODONTICS
- ENDODONTIC IMPLICATIONS (PATHOGENESIS OF APICAL PERIODONTITIS AS EXPLAINED BY FISH)
- KRONFELD'S MOUNTAIN PASS THEORY
- RATIONALE OF ENDODONTIC THERAPY
- CONCLUSION

INTRODUCTION

- Endodontic pathology is mainly caused any injury to the tooth which can be physical, chemical or bacterial
- Causes reversible or irreversible changes to pulp and periradicular tissues
- Changes depend on intensity, duration, pathogenicity of stimulus and host defense mechanism
- Rationale of endodontic therapy is complete debridement of root canal followed by three dimensional obturation.

THEORIES OF SPREAD OF INFECTION

- **Focal infection** – it is localized or generalized infection caused by the dissemination of microorganisms or toxic products from a focus of infection.
- **Focus of infection-** this is a circumscribed area of tissue, which is infected with exogenous pathogenic microorganisms and is usually located near a mucous or cutaneous surface.

ORAL FOCI OF INFECTION

- Possible sources of infection in oral cavity which may later set up distant metastases are
  1. infected periapical leasions
     I. Periapical granuloma
     II. Periapical abscess
     III. Periapical cyst
  2. Teeth with infected root canals
  3. Periodontal diseases with specific refeerence to tooth extraction

CULPRIT OF ENDODONTIC PATHOLOGY

- Root canal infections are multibacterial in nature.

PORTALS OF ENTRY OF MICROORGANISMS

- Microorganisms may gain access through several routes.

- Eg; Dental caries, mechanical or traumatic injury, gingival sulcus, periodontium and via blood stream.

- Anachoresis- refers to the attraction of blood borne bacteria in the areas of inflammation.

- It is the process by which microorganisms are transported in the blood to an area of inflammation where they establish an infection

INFLAMMATION

- Inflammation is defined as the local response of living mammalian tissue to injury due to any agent.
- Signs of inflammation
  - Rubor
  - Calor
  - Tumor
  - Dolor
  - Loss of function

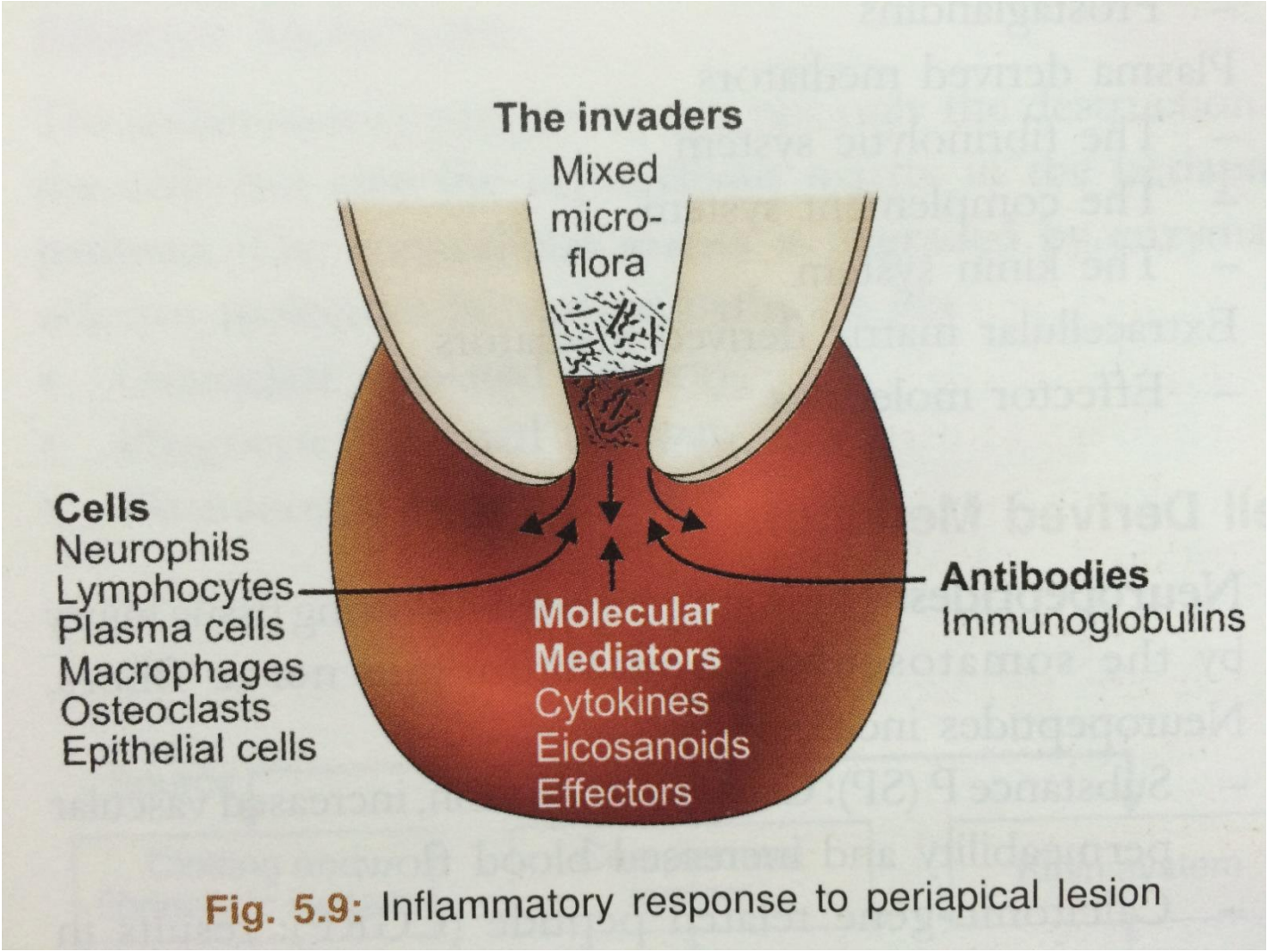


Fig. 5.9: Inflammatory response to periapical lesion

- Inflammation is of 2 types
  - Acute – PMNLs and macrophages
  - Chronic – lymphocytes, plasma cells, macrophages.

- In an infected canal, the microbes usually remain with the canal
- Host defence mechanism cannot completely remove bacteria
- Resulting in, inflammatory response in periapex

TISSUE CHANGES FOLLOWING INFLAMMATION

- 2 TYPES
  - DEGENERATIVE
  - PROLIFERATIVE

a)Degenrative changes in pulp can be

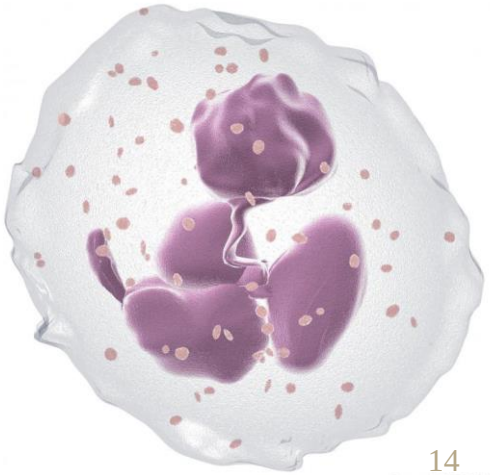
- Fibrous
- Resorptive
- Calcific
- Suppuration

b)Proliferative

- Irritants are mild and act as stimulant
- In the inflame area the irritant may be strong enough to produce degeneration or destruction
- The principal cells of proliferation or repair are the fibroblast

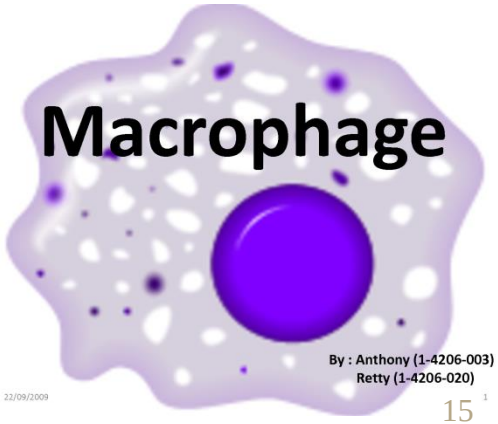
INFLAMMATORY CELLS

- **NEUTROPHILS-** PMNLs, basophils, eosinophils, also called as granulocytes
- Reach site of injury within 24 hrs
- Phagocytose bacteria and cellular debris
- In low Ph, neutrophils die and release proteolytic enzymes, prostaglandins and leukotrienes
- This breaks down tissue leading to abcess formation

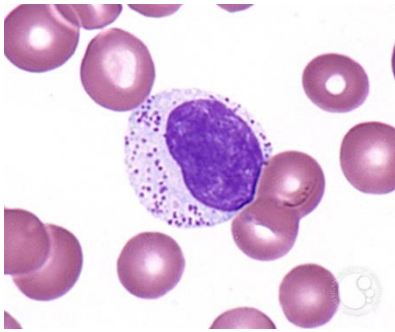


wiseGEEK

- **Macrophages** – circulation monocytes reaches site of inflammation and change to macrophages
- Remain at site for longer time – 2 months
- Function- phagocytosis, pinocytosis, secrete lysosomal enzymes, complement protein and prostaglandin secretion
- Provide antigen to immunocomplement cells
- Scavenger of dead cells
- Produce multinucleated giant cells



- **Lymphocyte-** seen in apical periodontits. Two types



  - T-lymphocytes
  - B- lymphocytes
- **Osteoclast** – in case of apical periodontitis, they proliferate and fuse on stimulation by cytokines and other mediators to form osteoclasts.
- Causes demineralization, dissolution of organic matrix
- Results in bone resorption

- **Epithelial cells-** cytokines and other mediators stimulate the dormant cell rests of malassez.
- Results in inflammatory hyperplasia

INFLAMMATORY RESPONSE TO PERIAPICAL LESION

- Non specific mediators of periradicular lesions. Can be classified as:
  - Cell derived
    - Neuropeptides
    - Arachidonic acid derivatives
    - Cytokines
    - Lysozomal enzymes
    - Platelet activating factor
    - Vasoactive amines
    - Prostaglandins

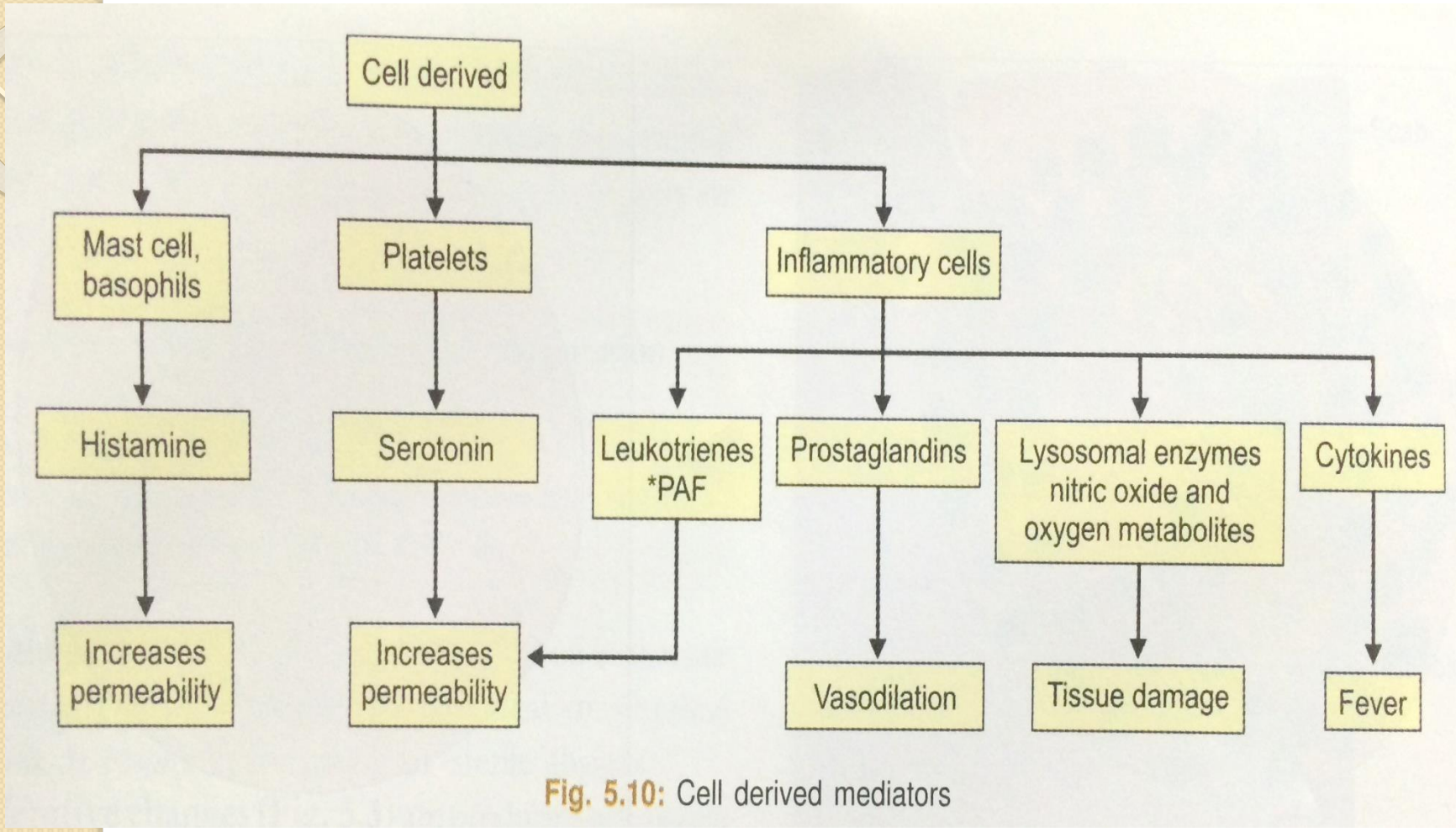


Fig. 5.10: Cell derived mediators

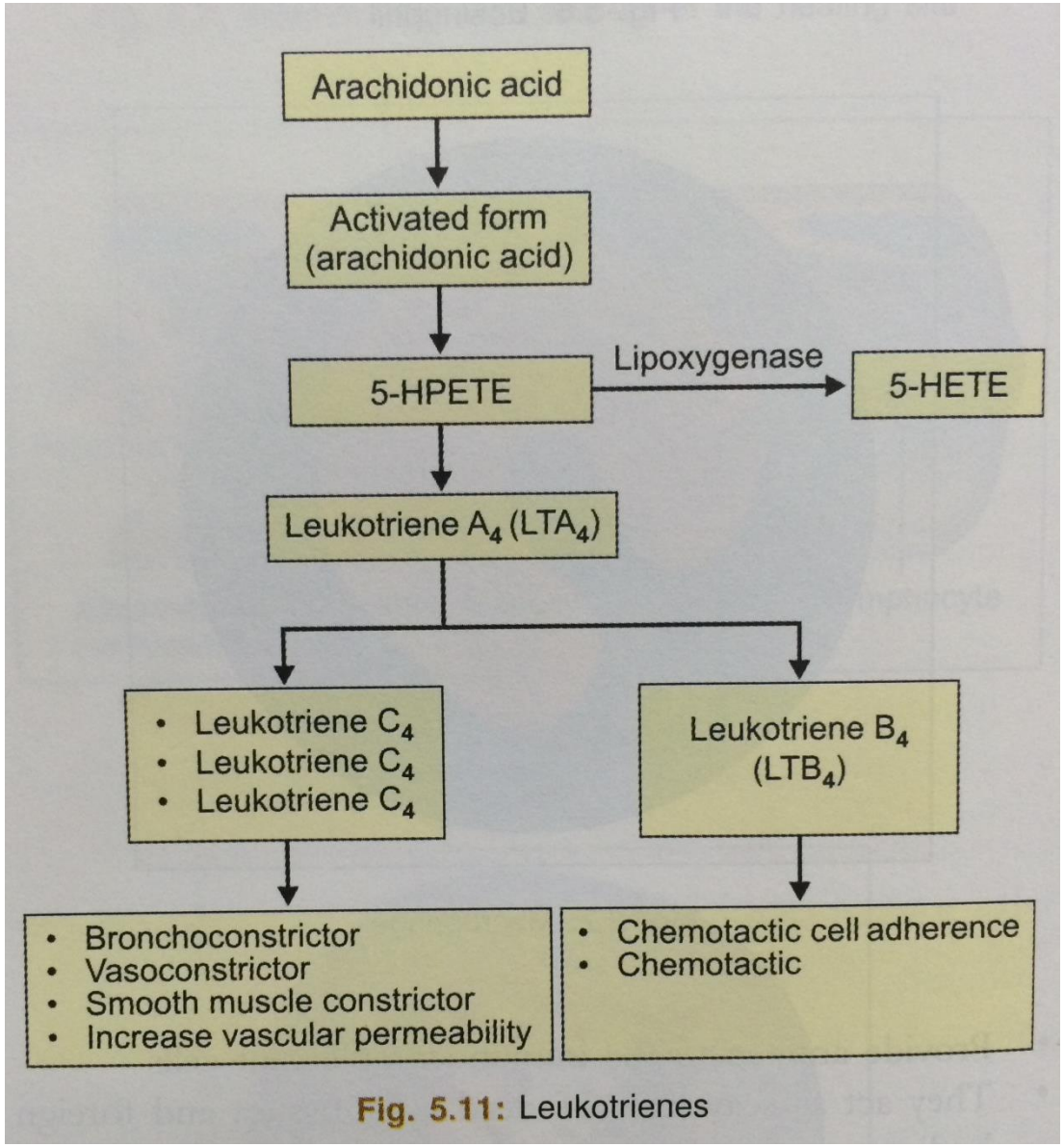


Fig. 5.11: Leukotrienes

- Plasma derived mediators
  - Fibrinolytic system
  - Complement system
  - Kinin system
- Extracellular matrix derived mediators
  - Effector molecules

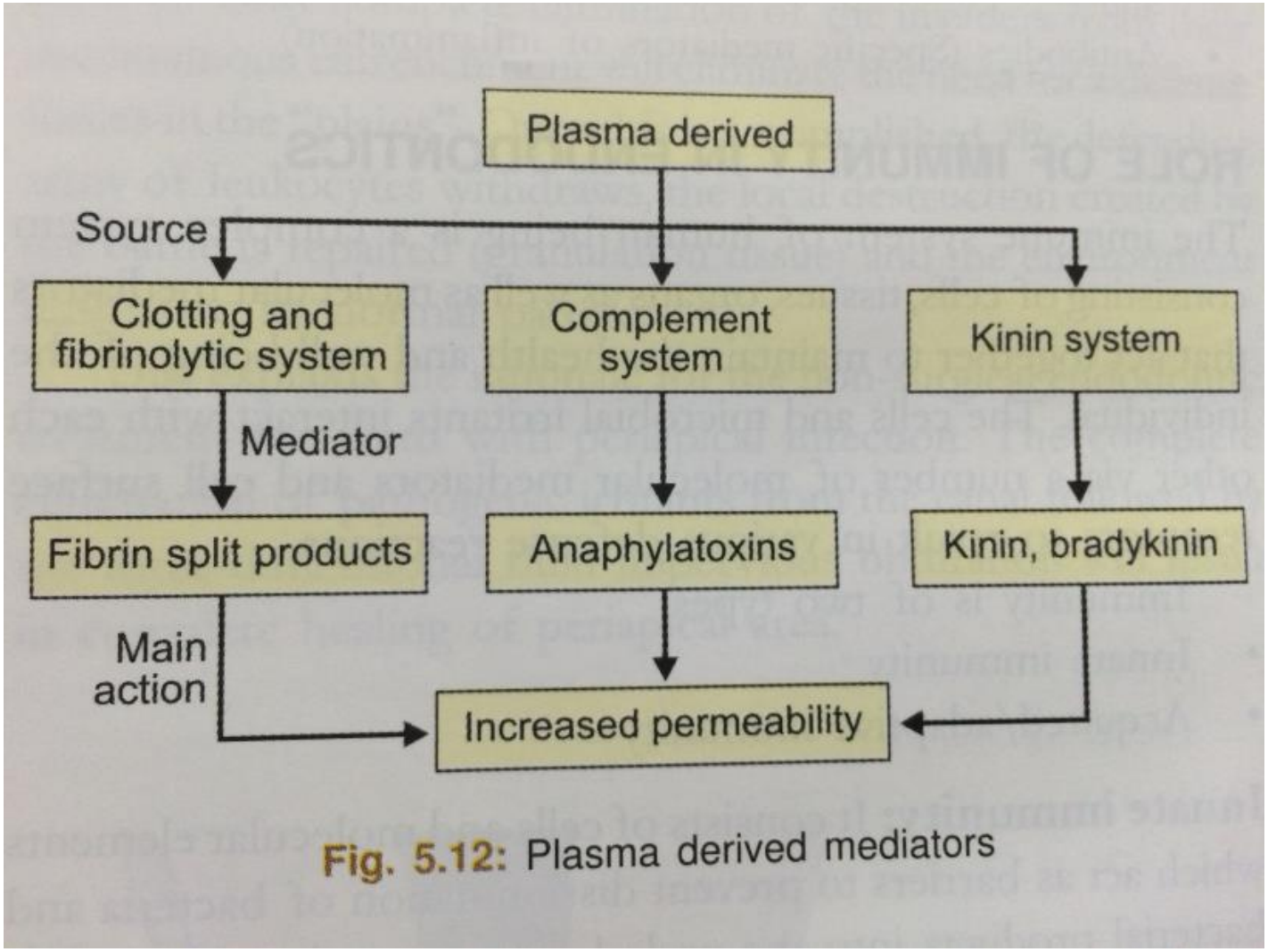


Fig. 5.12: Plasma derived mediators

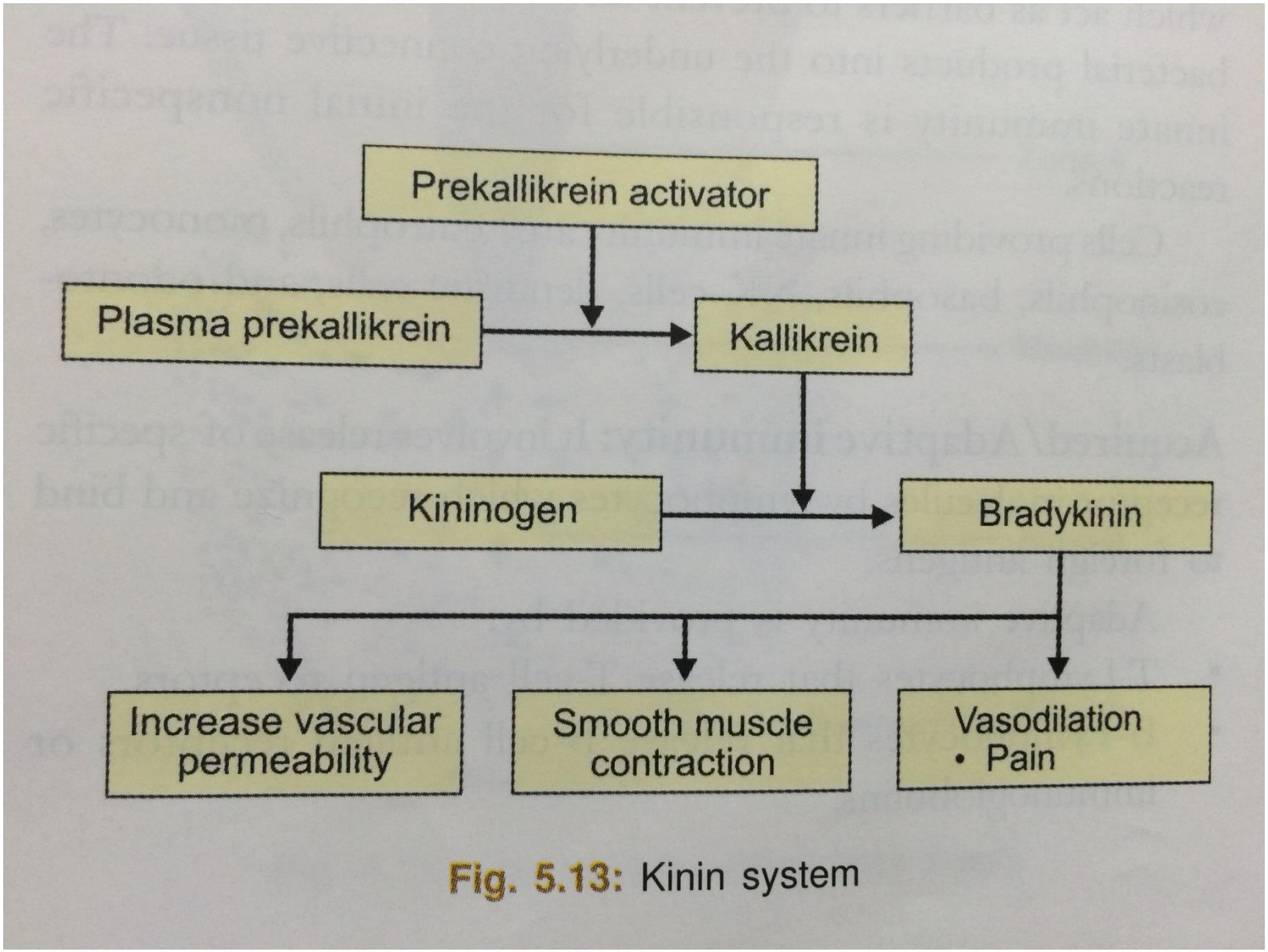


Fig. 5.13: Kinin system

CELL DERIVED MEDIATORS

- **Neuropeptides** - substance P and calcitonin - gene related peptide

- **Eicosanoids** - form prostaglandins and leukotrienes

  - Prostaglandins include
    - PGE_2
    - PGD_2
    - PGF_{2a}
    - PGI_2

  - Leukotrienes produced by the activation of lipoxygenase pathway of arachidonic acid

- **<u>Cytokines</u>**- low molecular weight polypeptides
- They cause development and perpetuation of periradicular lesions
- 2 types
  - Pro inflammatory cytokines – IL1,IL6, IL8
  - Chemotactic agents – TNFα, TNFß, TNFɣ
- IL1ß and TNFα are predominant in periradicular inflammation.
- TNFα is seen in root canal exudates and chronic apical lesions.

- **Lysosomal enzymes-** alkaline phosphatase, peroxidases, collagenases, increase vascular permeability, activation of complement system, bradykinin formation

- **Platelet activating factor-** released from IgE, increases vascular permeability, chemotaxis, adhesion of leukocytes to endothelium

- **Vasoactive amines-** like histamine, seratonin. Increase tissue permeability and vasodilation

PLASMA DERIVED MEDIATORS

- **Fibrinolytic system-** fibrinopeptides and fibrin degradation products, increase vascular permeability and leukocytiv chemotaxis
- **Complement system-** activated by trauma to periapex, causes swelling pain and tissure destruction
- **Kinin system-** causes smooth muscle contraction, vasodilatation, increase in vascular permeability.

ANTIBODIES

- Polyclonal antibodies- IgG, IgM
- Monoclonal antibodies- form antigen antibody complexes and bind to plateletes.
- In acute abcess- complexes enter into systemic circulation
- In chronic lesions- complexes are confined within lesion.

ROLE OF IMMUNITY IN ENDODONTICS

- The immune system of human being is a complex system consisting of cells, tissues, organs as well as mediators
- Immunity is of two types;

  a) innate immunity – consists of cells and molecular elements which acts as barrier to prevent bacteria into the underlying connective tissue

  b) acquired immunity – release of specific receptor molecules by lymphocytes which recognize and bind to foreign antigen

Endodontic Implications (Pathogenesis of Apical Periodontitis as Explained by Fish)

- Fish, in 1939, described the reaction of periradicular tissues to bacterial products, noxious products of tissue necrosis and antigenic agents from root canals.
- Theorized that the zones of infection are not an infection by themselves but the reaction of the body to infection.

FISH ZONES OF REACTION

- 4 well defined zones
  a. Zone of **Infection** or **Necrosis**
  b. Zone of **Contamination**
  c. Zone of **Irritation**
  d. Zone of **Stimulation**

A)ZONE OF INFECTION

- Infection is confined to the center of the lesion.
- Characterized by **polymorphonuclear leukocytes(PMNL)** and **microorganisms**
- Necrotic cells and destruction components from phagocytes.

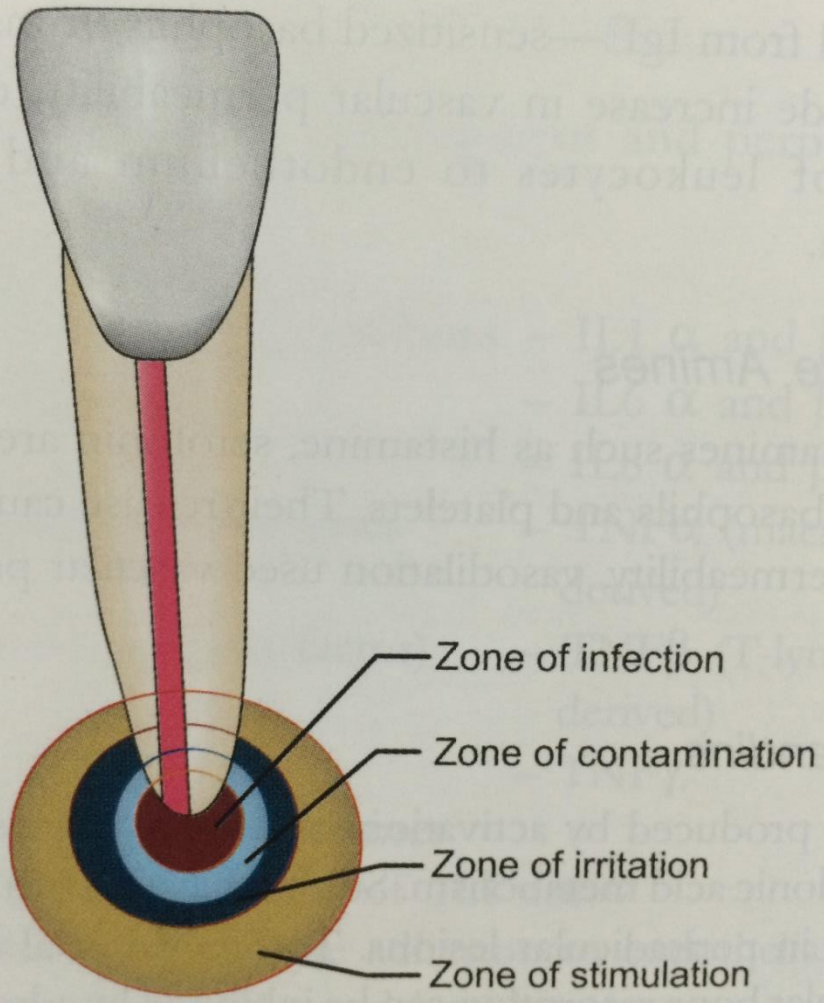


Fig. 5.14: FISH zones

B)ZONE OF CONTAMINATION

- Located around the center
- Area of cellular destruction
- **No bacteria**
- Destruction due to toxins released from central zone.
- Characterized by **round cells**, osteocyte necrosis and empty lacunae
- **Lymphocytes** prevalent

C)ZONE OF IRRITATION

- Zone away from central lesion
- Characterized by **macrophages, histiocytes and osteoclasts.**
- Histologically, looks like **preparation to repair**

D)ZONE OF STIMULATION

- Peripheral zone, toxins act as stimulant
- Characterized by **fibroblasts and osteoblasts**
- Secretion of collagen by fibroblasts and new bone formation by osteoblasts

- Microorganisms from root canal proliferateto grow out of root canal
- Metabolic byproducts or toxic products may get diffused into periradicular tissue.
- If microorganisms are highly virulent- can develop into periradicular lesion, formation of pus and finally in the formation of a chronic abscess.

KRONFELD'S MOUNTAIN PASS THEORY

- Kronfeld expalined that granulomas provide and unfavorable environment for bacterial survival.
- He employed FISH concept to explain tissue reaction

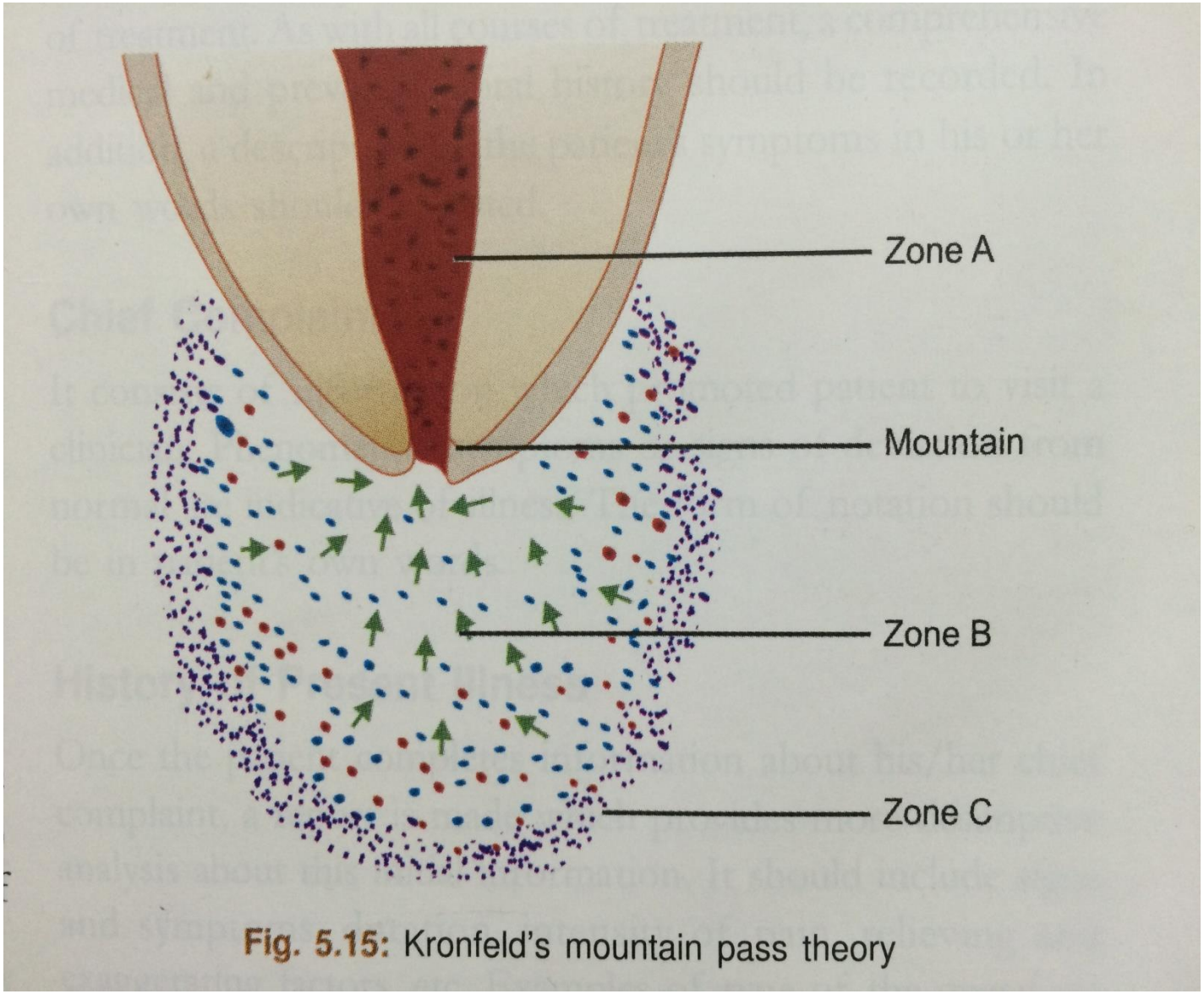


Fig. 5.15: Kronfeld's mountain pass theory

1) ZONE A- *bacteria* in infected root canal as **invaders** behind high, inaccessable "mountains" and *foramina* as **mountain pass**
2) ZONE B- *Proliferative tissue* of granuloma represents an **Army** defending the periapex from the bacteria.
    i. When few bacteria enter, it is destroyed by leukocytes . This results in major battle, analogous to acute inflammation

3) ZONE C- complete elimination of bacteria from the mountainous entrenchment (root canal) will result in elimination of defence forces in plains

RATIONALE OF ENDODONTIC THERAPY

- Rationale of endodontic therapy relies on the fact that non-vital pulp being avascular has no defence mechanisms.
- Breakdown products from damaged tissue diffuses into surrounding tissue leading to periapical irritation.
- Endodontic therapy seals the root canal system 3-dimensionally and prevents percolation of toxic byproducts into periapex.

- Endodontic therapy includes

  a) Non surgical endodontic treatment – includes three phases,

  1) access preparation

  2) shaping and cleaning

  3) obturation

  b) Surgical endodontic treatment – to remove the diseased tissue present in the canal and around the apex , and retrophil the root canal space with biologically inert material


Thank You