Chapter 2

Inflammation and Repair
Outline

- Injury
- Natural (Innate) Defenses against Injury
- Inflammation
- Regeneration and Repair
- Injuries to Teeth
- Injuries to Soft Tissues
- Reactive Connective Tissue Hyperplasia
- Inflammatory Periapical Lesions
The Body’s Responses to Injury Are Inflammation, Immunity, and Repair

- Injury: The result of an alteration in the environment that causes tissue damage or necrosis
  - Less severe injury: Hyperplasia, hypertrophy, atrophy

- Inflammation: Allows the body to eliminate injurious agents, contain injuries, and heal defects
Natural (Innate) Defenses against Injury

- Physical barrier: Intact skin or mucosa
- Mechanical defense: Respiratory system’s cilia and mucus
- Antibacterial activity: Enzymes in saliva
- Removal of foreign substances: Flushing action of tears, saliva, urine, and diarrhea
- Inflammation process: White blood cells
Inflammation

- Nonspecific response
- Extent and duration of injury → extent and duration of inflammatory response
- Local or systemic
- Acute, chronic, or combination
Inflammation: Classic or Cardinal
Clinical Signs

- Localized signs:
  - Redness
  - Heat
  - Swelling
  - Pain
  - Loss of normal tissue function
Inflammation: Classic or Cardinal Clinical Signs

- Systemic signs:
  - Fever
  - Leukocytosis
  - Elevated C-reactive protein
  - Lymphadenopathy
Sequence of Microscopic Events

1. Injury to tissue
2. Constriction of microcirculation
3. Dilation of microcirculation → hyperemia → erythema and heat
4. Increase in permeability
5. Exudate leaves microcirculation → transudate
6. Increased blood viscosity
7. Decreased blood flow
8. Margination and pavementing of white blood cells (WBCs) → chemotaxis
9. WBCs enter tissue → emigration → exudate and edema
10. WBCs ingest foreign material → phagocytosis
Inflammation: Microscopic Events and Clinical Signs of Inflammation
Inflammation: Sequence of Microscopic Events

- **Hyperemia**
  - Increased blood flow in capillary beds of injured tissue
    - Will produce erythema (redness) and heat

- **Exudate**
  - Increased blood plasma and proteins in injured tissue
    - Helps dilute injurious agents, but results in excess fluid in tissues: edema
  - Serous (watery) exudate
    - Mainly plasma fluids and proteins, a few WBCs
  - Purulent exudate (suppuration)
    - Contains plasma fluids and proteins, tissue debris, and many WBCs
Inflammation: Sequence of Microscopic Events

- Transudate
- Exudate
- Edema
- Heat
- Abscess
- Fistula
- Pain
Inflammation: Sequence of Microscopic Events

Drain for purulent exudate

Margination and pavementing of neutrophils (N)
Inflammation: Vocabulary Review

- Emigration
  - The process by which WBCs escape from blood vessels through gaps in endothelial cells

- Chemotaxis
  - Directed movement of WBCs toward the site of injury

- Phagocytosis
  - The process by which WBCs ingest and then digest foreign substances
  - May include pathogenic organisms and tissue debris
White Blood Cells in the Inflammatory Response

- White blood cells or leukocytes
  - Neutrophils: Polymorphonuclear leukocytes
  - Monocytes circulating in blood → macrophages in tissue
  - Lymphocytes and plasma cells
    - Seen in chronic inflammation and the immune response
  - Eosinophils and mast cells
    - Seen in both inflammation and the immune response
White Blood Cells in the Inflammatory Response

- Acute
  - Neutrophil
  - Lymphocyte and plasma cell
  - Macrophage

- Chronic

Amount vs. Time

Acute → Chronic
Inflammation → Immune response

Neutrophils: Polymorphonuclear Leukocytes

- Function: Phagocytosis
- Microscopic appearance: Multilobed nucleus and granular cytoplasm that contains lysosomal enzymes
- Constitute 60% to 70% of WBC population
- Derived from stem cells in bone marrow
Neutrophils: Polymorphonuclear Leukocytes
Macrophages

- Function: Phagocytosis and plays a role in immune system
- Microscopic appearance: Single round nucleus and does not have granular cytoplasm
- Constitutes 3% to 8% of WBC population
- Derived from stem cells in bone marrow
Macrophages
Biochemical Mediators of Inflammation

- Cause many of the events in the inflammatory response
  - Basic mediators can recruit other mediators and immune mechanisms
- May be derived from:
  - Blood
  - Endothelial cells
  - White blood cells and platelets
  - Pathogenic organisms as they injure the tissue
Biochemical Mediators of Inflammation

- Three interrelated systems
  - Interaction takes place during activation, among their products, and within their various actions
- Kinin system
- Clotting mechanism
- Complement system
Kinin System

- Active in early phases of inflammation
- Activated by substances in plasma and injured tissue
- Causes increased:
  - Dilation of blood vessels at the site of injury
  - Permeability of local blood vessels
- Induces pain
Clotting Mechanism

- Clots blood and mediates inflammation
  - Some of the clotting mechanism products that are activated during tissue injury cause local vascular dilation and permeability by activating kinin
Complement System

- Involves the production of a sequential cascade of plasma proteins
  - They are present in blood in an inactive form
  - A trigger (usually an antibody-antigen complex) initiates the sequence of steps
  - These plasma proteins function in inflammation and immunity
- Some components cause WBCs known as mast cells to release histamine
  - Histamine causes an increase in vascular permeability and vasodilation
- Other components cause cell death, form chemotactic factors for WBCs, and enhance phagocytosis
Other Biochemical Mediators of Inflammation

- Released by the body:
  - Prostaglandins
    - Cause increased vascular dilation and permeability, tissue pain and redness, and changes in connective tissue
  - Lysosomal enzymes
    - Act as chemotactic factors
    - May cause damage to connective tissues and to the clot
Other Biochemical Mediators of Inflammation

- Released by pathogenic microorganisms:
  - **Endotoxin**
    - Produced by cell walls of gram-negative bacteria
    - Serves as chemotactic factor; can activate complement, function as an antigen, and damage bone and tissue
  - **Lysosomal enzymes**
    - Have a similar chemical composition and action as those released by WBCs
Systemic Manifestations of Inflammation

- Fever
- Leukocytosis
- Elevated C-reactive protein
- Lymphadenopathy
Systemic Manifestations of Inflammation: Fever

- Controlled by the hypothalamus

- Pyrogens
  - Fever-producing substances produced by WBCs and pathogens
  - Pyrogens act on the hypothalamus

- The hypothalamus increases body temperature by way of prostaglandins
Systemic Manifestations of Inflammation: Leukocytosis

- Normal number of WBCs per mm$^3$ of blood: 4000 to 10,000
- In a systemic inflammatory response, the number increases to 10,000 to 30,000/mm$^3$ of blood. This is leukocytosis, or an increase in circulating WBCs
- It is the body’s attempt to provide more cells for phagocytosis
- The type of WBC that is increasing in number can aid in differential diagnosis
  - Viral infection: Increase in lymphocytes
  - Bacterial infection: Increase in neutrophils
  - Allergic reaction: Increase in eosinophils
Systemic Manifestations of Inflammation: Lymphadenopathy

- Enlarged and palpable superficial lymph nodes
- The enlarged nodes occur because of changes in lymphocytes, which are the primary cells of the immune response
  - Hyperplasia: An increase in the number of cells
  - Hypertrophy: Enlargement of individual cells
Lymphadenopathy
Systemic Manifestations of Inflammation: Elevated C-Reactive Protein

- Produced in the liver
- Interacts with the complement system and the clotting mechanism
- Levels can be used to help assess rheumatoid arthritis and systemic lupus erythematosus
- Used to monitor tissue healing
- Used as early infection detection system
- Chronically increased level is associated with an increased risk for cardiovascular disease
- Possible marker for periodontal disease
Systemic Manifestations of Inflammation: Chronic Inflammation

- Caused by persistent injuries
- Repair cannot be completed until source of injury is removed
- Cells involved:
  - Macrophages
  - Lymphocytes
  - Plasma cells
  - Neutrophils
  - Monocytes
  - Fibroblasts
Systemic Manifestations of Inflammation:
Chronic Inflammation:
Granulomatous Inflammation

- Formation of granuloma: Microscopic groupings of macrophages surrounded by lymphocytes and plasma cells
  - These macrophages group together to form multinucleated giant cells
  - Associated with foreign body reactions and some infections such as tuberculosis
Systemic Manifestations of Inflammation: Antiinflammatory Drugs

- Nonsteroidal antiinflammatory drugs (NSAIDs)
  - Acetylsalicylic acid (aspirin)
  - Ibuprofen
- Steroidal antiinflammatory drugs
  - Prednisone
- Antihistamines
- Cancer treatment drugs
  - Methotrexate
  - Sulfasalazine
  - Leflunomide
  - Cyclophosphamide
  - Mycophenolate
Reactive Tissue Responses: Hyperplasia, Hypertrophy, and Atrophy

- Hyperplasia
  - An increase in the number of cells, often in response to chronic irritation or abrasion
  - May return to normal if the insult subsides, or may persist after removal of the irritant

- Hypertrophy
  - An increase in the size of cells
  - May be seen in cardiac muscle as a response to hypertension

- Atrophy
  - A decrease in size or function of a cell, tissue, organ, or entire body
Hyperplasia: Increased Epithelial Thickness
Regeneration and Repair

- **Regeneration**: The process by which injured tissue is replaced with tissue identical to that present before the injury.

- **Repair**: The restoration of damaged or diseased tissues.
Microscopic Events That Occur during Repair

Small injury involving epithelium and connective tissue (CT)

Clot forms

Migrating epithelial cells form a new surface layer

Granulation tissue forms

Scar tissue
Microscopic Events: Day of Injury

- Blood flows into injured tissue to produce a clot
  - The clot contains fibrin, clumped red blood cells (RBCs), and platelets
Microscopic Events: One Day after Injury

- Neutrophils migrate from the microcirculation into injured tissue in an acute inflammatory response
- Phagocytosis occurs
Microscopic Events: Two Days after Injury

- Monocytes change to macrophages in tissue
- Macrophages continue phagocytosis and secrete growth factors that stimulate growth of new blood vessels in a process called angiogenesis
- Neutrophils are reduced in number
- Fibroblasts increase in number and produce new collagen fibers in a process called fibroplasia
- Granulation tissue (connective tissue, CT) is formed
- Epithelialization, the process by which new surface tissue is created, occurs
- Blood clot acts as a scaffold for new CT
- Lymphocytes and plasma cells migrate to the area as chronic inflammation and the immune response begin
Microscopic Events: Seven Days after Injury

- Inflammatory and immune responses are completed, *if* the source of injury is removed
- Fibrin is digested by tissue enzymes
  - It sloughs off and the initial repair is complete
- The new tissue is relatively red
  - New epithelium is thin
  - New connective tissue is highly vascularized
- Immature collagen fibers are present and fragile
- Fibroblasts differentiate into myofibroblasts
Microscopic Events: Two Weeks after Injury

- Initial granulation tissue and its fibers have been remodeled
- Matured, fibrous CT is called *scar tissue*
  - It is whiter and paler because of increased collagen and decreased vascularity
Factors Affecting Amount of Scar Tissue

- Heredity
- Strength and flexibility needed in the tissue
- Tissue type
- Type of repair
  - Healing by primary intention
  - Healing by secondary intention
  - Healing by tertiary intention
Types of Repair

- Healing by primary intention
  - Healing of an injury where there is little loss of tissue
  - The margins are close together and very little granulation tissue forms
Types of Repair

- Healing by secondary intention
  - The edges of the injury cannot be joined during healing
  - A large clot forms, resulting in increased granulation tissue
  - May result in excess scar tissue: A keloid
Keloid Formation: Excessive Scar Tissue
Types of Repair

- Healing by tertiary intention
  - Delaying surgical tissue repair until infection is resolved
    - An injured area may become infected, especially with puncture wounds
    - In some situations, an infected injury is left open until infection is controlled
Factors That Impair Healing

- Local factors that impair healing:
  - Bacterial infection
  - Tissue destruction and necrosis
  - Hematoma
  - Excessive movement of injured tissue
  - Poor blood supply

- Systemic factors:
  - Malnutrition
  - Immunosuppression
  - Genetic connective tissue disorders
  - Metabolic disorders
Bone Tissue Repair

- Osteoblasts create new bone tissue:
  - Factors delaying bone formation:
    - Blood supply at site
    - Growth factors
    - Edema
    - Injury
    - Infection
    - Removal of osteoblast-producing tissues
    - Excessive or inadequate movement of bone tissue

- Factors influencing repair of bone:
  - Nutrition
  - Age
  - Tobacco use
Injuries to Teeth

- Attrition
  - Bruxism
- Abrasion
- Abfraction
- Erosion
- Bulimia
- Methamphetamine abuse
Attrition

- Tooth-to-tooth wear
  - May be observed both in primary and permanent dentition
Bruxism

- Grinding and clenching teeth for nonfunctional purposes, such as
  - Occlusal interferences
  - Stress
  - Tension
  - Seizure disorders
Bruxism

- Signs and symptoms
  - Wear facets
  - Abnormal rate of attrition
  - Hypertrophy of masticatory muscles
  - Increased muscle tone
  - Muscle tenderness
  - Muscle fatigue
  - Cheek biting
  - Pain in the temporomandibular (TM) joint area
  - Tooth mobility
  - Pulpal sensitivity to cold

- Management: Occlusal adjustments to eliminate occlusal interferences and fabrication of an acrylic splint
Abrasions

- Pathologic wearing away of tooth structure that results from a repetitive mechanical habit
- Most frequently seen as a notching on root surfaces with gingival recession
Abfraction

- **Cause:** Microfracture of tooth structure in areas of concentration of stress
  - May be related to fatigue, flexure, fracture, and deformation of tooth structure
  - May occur in combination with abrasion

- **Appearance:** Typically appears as wedge-shaped lesions at the cervical areas of teeth

- **Preventive treatment:** Fabricating an acrylic splint
Erosion

- Loss of tooth structure as a result of chemicals, without bacterial involvement
- Tooth structure may be lost around a restoration, making the restoration stand out, distinguishing it from abrasion or attrition
- Correlate location of erosion and abrasion with patient’s history
Erosion

Potential causes:
- Industrial factors
- Intraorally applied cocaine hydrochloride drug abuse
- Overuse of soft drinks
- Baby bottle caries
- Sucking on lemons
- Chronic vomiting
Bulimia: An eating disorder characterized by food binges followed by self-induced vomiting

- The patient with bulimia maintains a normal body weight but is secretive about eating habits.
  - May see electrolyte imbalance and/or malnutrition
  - Irritation of oral mucosa and lips
  - Traumatic lesions on the back of the fingers
Bulimia

● Management of oral health:
  ➢ Fluoride rinse and toothpaste
  ➢ Rinse with water after purging
  ➢ Avoid brushing immediately after vomiting
  ➢ Use very soft toothbrush
  ➢ May require full-coverage restorative dental treatment
Methamphetamine Abuse: Meth Mouth

- Rapid destruction of teeth as a result of:
  - Methamphetamine acid content
  - Decreased salivary flow
  - Cravings for high-sugar beverages
  - Lack of oral hygiene
Injuries to Oral Soft Tissues

- Aspirin burn
- Phenol burns
- Electric burns
- Other burns
- Lesions associated with cocaine use
- Lesions from self-induced injuries
- Hematoma
- Traumatic ulcer
- Frictional keratosis
- Linea alba
- Nicotinic stomatitis
- Tobacco pouch keratosis
- Traumatic neuroma
- Amalgam tattoo
- Melanosis
- Solar cheilitis
- Mucocele
- Necrotizing sialometaplasia
- Sialolith
- Acute and chronic sialadenitis
Aspirin Burn

- Topical application is a common misuse of this product
  - The tissue becomes necrotic and white
  - The surface may slough off, leaving a painful ulcer
  - The ulcer usually heals in 7 to 21 days
Phenol Burn

- Used in dentistry as a cavity-sterilizing agent and a cauterizing agent
- Will cause whitening and sloughing of the area as a result of tissue destruction
Dental Materials That Can Cause Burns:

- Phenol
- Sodium hypochlorite
- Ferric sulfate
- Formocresol
- Eugenol
Electric Burn

- May be seen in infants or young children who have chewed an electrical cord
- May be quite extensive, damaging oral tissue and even tooth buds
- May cause permanent disfigurement and scarring

Treatment
- Plastic surgery
- Oral surgery
- Orthodontic therapy
Other Burns

- Hot food burns
  - From soup or cheese on pizza
- Products containing hydrogen peroxide or eugenol
Lesions Associated with Cocaine Use

- Lesions located at the midline of the hard palate may vary from ulcers to keratotic lesions to exophytic reactive lesions as a result of smoking crack cocaine.

- Necrotic ulcers of the tongue and epiglottis have been reported as a result of free-basing cocaine.
Lesions from Self-Induced Injuries

- Chronic lip, cheek, or tongue biting
- Trauma to the gingiva from a fingernail
- Lesions may range from ulceration to epithelial hyperplasia and hyperkeratosis
Hematoma

- Accumulation of blood within tissue as a result of trauma
- Appears as a red to purple to bluish-gray mass
- Frequently seen on labial or buccal mucosa
Traumatic Ulcer

- Cheek, lip, or tongue biting
- Denture irritation
- Mucosal injury
- Overzealous brushing

Treatment

- Usually heals within 7 to 14 days unless the trauma persists
- May require a biopsy
Traumatic Granuloma

- The result of persistent trauma
- Appearance: Hard (indurated), raised lesion
- Heals rapidly after biopsy
Frictional Keratosis

- A form of hyperkeratosis
- Cause: Chronic rubbing or friction against an oral mucosal surface; resembles a callus on skin
- Appearance: Opaque white
Frictional Keratosis

- Treatment
  - Identify the traumatic cause of the lesion
  - Eliminate the cause

- Must be differentiated from idiopathic leukoplakia because leukoplakia may be premalignant
Linea Alba

- A white, raised line most commonly on the buccal mucosa at the occlusal plane
  - May be the result of a teeth-clenching habit
  - Sometimes the pattern of the teeth can be seen in the lesion
  - Microscopic appearance: Epithelial hyperplasia and hyperkeratosis
- No treatment necessary
Nicotinic Stomatititis

- A benign lesion typically associated with pipe and/or cigar smoking; may also occur with cigarette smoking
Nicotinic Stomatitis

- Initial appearance: Erythema
- Increased opacity as keratinization occurs
- Raised red areas occur at the openings of ducts of inflamed minor salivary glands
Tobacco Pouch Keratosis: Smokeless Tobacco Keratosis

- A white lesion located where chewing tobacco is placed, most often in the mucobuccal fold
  - Early lesions may have a granular or wrinkled appearance
  - Long-standing lesions may be more opaquely white and have a corrugated surface
Tobacco Pouch Keratosis

- Treatment:
  - Tobacco cessation
  - May require biopsy
- Long-term exposure to chewing tobacco has been associated with increased risk of squamous cell carcinoma
Traumatic Neuroma

- A lesion caused by injury to a peripheral nerve
  - When the nerve sheath of Schwann cells is disrupted, occasionally the proximal end of the damaged nerve proliferates into a mass of nerve and Schwann cells mixed with dense fibrous scar tissue
  - Painful, ranging from pain on palpation to severe, intractable pain

- Diagnosis
  - Biopsy and microscopic examination

- Treatment
  - Surgical excision
Palisaded Encapsulated Neuroma (PEN)

- Benign lesion
- Clinical appearance: Mucosal nodule
- Microscopic appearance: Well-circumscribed lesion that is composed of nerve tissue partially surrounded by fibrous connective tissue
- Considered a reactive, hyperplastic lesion
Amalgam Tattoo

- A flat, bluish-gray lesion of the oral mucosa, caused by the introduction of amalgam into tissue
- May occur during placement or removal of an amalgam restoration or during an extraction
- May be seen in any location in the oral cavity, most commonly on the gingiva or alveolar ridge
Amalgam Tattoo

- Amalgam particles may be seen on radiograph, aiding in diagnosis
- Patient history may help
- Must be differentiated from malignant melanoma
- Treatment
  - None, providing melanoma has been ruled out
Melanosis

- Normal physiologic pigmentation of oral mucosa
  - May be genetic
  - May occur as a result of inflammation: Postinflammatory melanosis
  - If presenting as a macule, a biopsy may be warranted
  - Labial melanotic macule on vermilion of lips
  - Smoker’s melanosis
Solar Cheilitis (Actinic Cheilitis)

- A degeneration of the tissue of the lips, caused by exposure to the sun

- Appearance:
  - Lips appear dry and cracked
  - The vermilion appears pale pink and mottled
  - The interface between lips and skin is indistinct
  - Microscopically: Epithelium is thinner than normal; degenerative CT changes
Solar Cheilitis (Actinic Cheilitis)

- Smoking and alcohol use increase risk of squamous cell carcinoma
- Biopsy may be indicated for persistent scaling or ulceration
- Prevention
  - Avoid sun exposure
  - Use sun-block agents
Mucous Retention Lesion: Mucocele

- A lesion formed when a salivary gland duct is severed and the mucous salivary gland secretion spills into the adjacent CT

- Not a true cyst because it is not lined with epithelium
Mucous Retention Lesions: Mucocele, Mucous Cyst, or Mucous Retention Cyst

- Dilated salivary gland ducts that developed as a result of duct obstruction

- Treatment: Removal of affected minor salivary gland
Mucous Retention Lesion: Ranula

- A unilateral mucocele-like lesion that forms on the floor of the mouth
  - Associated with the ducts of submandibular and sublingual glands
Sialolith

- A salivary gland stone
  - May be found in both minor and major salivary glands
  - Formed by precipitation of calcium salts around a central core
  - May often be seen on radiograph
Sialolith

- **Treatment**
  - Sometimes the calcification can be “milked” from the duct
  - It may require surgical removal; this may damage the duct
Necrotizing Sialometaplasia

- A benign condition of salivary glands
  - Moderately painful swelling and ulceration
  - Thought to result from blockage of the blood supply to the affected area, resulting in necrosis of the salivary gland
Necrotizing Sialometaplasia

- Salivary gland epithelium is replaced by squamous epithelium
- The ulcer usually heals by secondary intention
- Biopsy is needed to establish diagnosis
Acute and Chronic Sialadenitis

- Painful swelling of the involved salivary gland caused by obstruction of the salivary gland duct

- Diagnosis
  - May involve injection of a radiopaque dye into the gland, followed by a radiograph (sialogram)

- Treatment
  - May require antibiotics
Reactive Connective Tissue Hyperplasia

- Pyogenic granuloma
- Giant cell granuloma
- Irritation fibroma
- Denture-induced fibrous hyperplasia
- Papillary hyperplasia of the palate
- Gingival enlargement
- Chronic hyperplastic pulpitis
Reactive Connective Tissue Hyperplasia

- Proliferating, exuberant granulation tissue and dense fibrous connective tissue resulting from overzealous repair
  - May be a response to a single event or chronic low-grade injury
Pyogenic Granuloma

- A proliferation of connective tissue containing numerous blood vessels and inflammatory cells occurring as a response to injury
  - The name is a misnomer; the lesion is neither pyogenic (pus forming) nor a true granuloma
Pyogenic Granuloma

- **Appearance:**
  - Ulcerated
  - Soft to palpation
  - Bleeds easily
  - Deep red to purple
  - Generally elevated, may be sessile or pedunculated
  - Most commonly observed on the gingiva, it may be seen on other intraoral areas
  - May vary in size from a few millimeters to several centimeters
  - Usually develops rapidly and then remains static
  - Most common in teenagers and young adults, but may occur at any age
  - If seen in a pregnant female, it is called a *pregnancy tumor*

- **Treatment**
  - Surgically excised if it does not regress spontaneously
Pregnancy Tumor

- A pyogenic granuloma seen in a pregnant woman
  - The lesions are identical to those seen in men and nonpregnant women
  - May be caused by hormonal changes and increased response to plaque
  - They often regress after delivery
Peripheral Giant Cell Granuloma

- A lesion that contains many multinucleated giant cells, well-vascularized connective tissue, RBCs, and chronic inflammatory cells
- Reactive lesion
- Clinical appearance resembles that of pyogenic granuloma
- Treatment: Surgical excision
● Peripheral giant cell granuloma
  ➢ Lesions occurring *outside* of bone

● Central giant cell granuloma
  ➢ Lesions *within* bone of the mandible or maxilla
Irritation Fibroma (Focal Fibrous Hyperplasia, Fibroma, Traumatic Fibroma)

- The most common mass on the gingiva
- Caused by trauma
Irritation Fibroma (Focal Fibrous Hyperplasia, Fibroma, Traumatic Fibroma)

- **Appearance:** A broad-based, persistent exophytic lesion composed of dense, scarlike connective tissue with few blood vessels. Usually a small lesion, less than 1 cm in diameter.
- Irritation Fibroma

- Peripheral Ossifying Fibroma
Denture-Induced Fibrous Hyperplasia (Epulis Fissuratum, Inflammatory Hyperplasia)

- **Cause:** Ill-fitting denture
- **Location:** In elongated folds of tissue adjacent to denture flange
- **Composed of:** Dense, fibrous CT surfaced with stratified squamous epithelium
- **Treatment:**
  - Surgical removal
  - Relining of prosthesis
  - New denture
Papillary Hyperplasia of the Palate (Palatal Papillomatosis)

- Denture-induced hyperplasia
- Appearance: Palatal mucosa covered by multiple erythematous papillary projections; “cobblestone” appearance
Papillary Hyperplasia of the Palate (Palatal Papillomatosis)

- Treatment: Surgical removal of hyperplastic papillary tissue before new denture construction
Gingival Enlargement

- An increase in the bulk of free and attached gingiva, especially the interdental papillae
- Gingival margins are rounded
- Color may vary from normal pink to pale or erythematous depending on the degree of inflammation and vascularity
- May be generalized or localized
Gingival Enlargement

- Reactive response to:
  - Local irritants
  - Hormonal changes
  - Drug induced
  - Hereditary
  - Idiopathic
  - Leukemia

- Treatment
  - Gingivoplasty
  - Gingivectomy
  - Meticulous oral hygiene
Chronic Hyperplastic Pulpitis
(Pulp Polyp)

- An excessive proliferation of chronically inflamed dental pulp tissue
  - Occurs in teeth with large, open carious lesions often in primary and permanent molars
  - Usually asymptomatic
Chronic Hyperplastic Pulpitis
(Pulp Polyp)

- Granulation tissue with inflammatory cells, primarily lymphocytes and plasma cells
- Neutrophils may be present
- Generally surfaced by stratified squamous epithelium

Treatment
  - Endodontic therapy
  - Extraction
Inflammatory Periapical Lesions

- Periapical abscess
- Dental or periapical granuloma
- Radicular cyst (periapical cyst)
- Resorption of teeth
- Focal sclerosing osteomyelitis
- Alveolar osteitis ("dry socket")
Inflammatory Periapical Lesions

- Caries or trauma may result in:
  - Inflammation
  - Infection
  - Chronic hyperplastic pulpitis
  - Necrosis of the pulp

- The inflammatory process begins in pulp and then extends to the periapical area
  - Accessory canals may lead to areas of inflammation on the lateral portion of the root
Acute Periapical Abscess

- Purulent exudate surrounded by connective tissue containing neutrophils and lymphocytes
  - Inflammation produces severe pain
  - Tooth may slightly extrude from tooth socket
  - May or may not test positive with electric pulp testing
Periapical Abscess

- Fistula
- Fistulous tract
- Channel of least resistance
- Presence of fistula warrants a radiographic evaluation
Periapical Abscess

- May develop directly from inflammation in the pulp
- More commonly develops in an area of previously existing chronic inflammation
- Treatment:
  - Drainage and endodontic therapy
  - Extraction
Dental Granuloma (Periapical Granuloma, Chronic Apical Periodontitis)

- A localized mass of chronically inflamed granulation tissue that forms at the opening of the pulp canal, generally at the apex of a nonvital tooth root.
Dental Granuloma (Periapical Granuloma, Chronic Apical Periodontitis)

- **Characteristics:**
  - Chronic process
  - Most cases are asymptomatic
  - Tooth may be sensitive to pressure and percussion
  - Tooth may be slightly extruded from the socket

- **Treatment:**
  - Endodontic therapy
  - Extraction
Dental Granuloma (Periapical Granuloma, Chronic Apical Periodontitis)

- Radiographic appearance:
  - May vary from slight thickening of the periodontal ligament space to a diffuse radiolucency, to a distinct, well-circumscribed radiolucency surrounding the root apex
Dental Granuloma (Periapical Granuloma, Chronic Apical Periodontitis)

- Composed of granulation tissue containing lymphocytes, plasma cells, and macrophages
- May also contain neutrophils, areas of dense fibrous connective tissue, or epithelial rests of Malassez

Microscopic appearance showing inflammatory cells at the apex of a tooth
Radicular Cyst (Periapical Cyst)

- A true epithelium-lined cyst
  - Associated with the root of a nonvital tooth
  - The most commonly occurring cyst in the oral region
  - A result of proliferation of the rests of Malassez
  - Usually asymptomatic and discovered on radiograph
Radicular Cyst (Periapical Cyst)

- Radiographic appearance:
  - Radiolucent
  - Well circumscribed
  - Same as periapical granuloma

- Treatment:
  - Endodontic therapy
  - Apicoectomy
  - Extraction and curettage of periapical tissue
Radicular Cyst (Periapical Cyst)

Periapical Cyst → Residual Cyst
Residual Cyst

- Forms after tooth extraction and all or part of radicular cyst is left behind

- Treatment:
  - Surgical removal
**External Resorption**

- Nonreversible resorption of the tooth structure, beginning at the outside of the tooth

- Causes:
  - Inflammation
  - Pressure
  - Reimplantation
  - Idiopathic
Internal Tooth or Root Resorption

- Resorption often associated with an inflammatory response in the pulp or for an idiopathic reason

- Appearance:
  - Clinically: A pinkish area in the crown resulting from the vascular, inflamed connective tissue
  - Radiographically: Radiolucent
Internal Tooth or Root Resorption

- **Treatment:**
  - If the root is not perforated, calcium hydroxide is placed and endodontic treatment is performed in an attempt to save the tooth.
  - If the tooth is perforated, it must be removed.
Focal Sclerosing Osteomyelitis
(Condensing Osteitis)

- A change in the bone near the apices of teeth
  - Thought to be a reaction to low-grade infection
- Generally asymptomatic
- If painful, may be associated with pulpal inflammatory disease
Focal Sclerosing Osteomyelitis (Condensing Osteitis)

- Radiopaque
  - Borders may be diffuse or well defined
- Commonly associated with the mandibular first molar
- No treatment usually necessary
- Biopsy to rule out other radiopaque lesions such as osteoma, complex odontoma, or ossifying fibroma
Alveolar Osteitis ("Dry Socket")

- A postoperative complication following tooth removal in which the blood clot is lost before healing can take place, leaving raw, exposed nerve endings
  - Most often occurring in the mandibular third molar areas
  - The patient may complain of pain, bad odor, and bad taste

- Risk factors:
  - Dissolution of the clot at the surgical site
  - Traumatic extraction
  - Presence of infection before extraction
  - Tobacco smoking after extraction
Alveolar Osteitis ("Dry Socket")

- Treatment:
  - Gentle irrigation
  - Daily application of Dry Socket Paste containing eucalyptol until symptoms are relieved
Discussion Questions

- What cells appear first during an inflammatory response?
- What is the difference between healing by primary, secondary, and tertiary intention?
- What is the difference between regeneration and repair?
- What is the difference between attrition, abrasion, and erosion?
- What injuries to oral soft tissues may be observed within the oral cavity?
- What is the definition of reactive tissue hyperplasia and what forms may be observed within the oral cavity?
- What inflammatory periapical lesions may be observed within the oral cavity?