PDQ

ORAL DISEASE Diagnosis and Treatment

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پرونده‌ها در قالب نرم‌افزارهای پزشکی (VHS، DVD، VCD، eBook) و ارائه این پرونده‌ها کامی کوچک در راه ارتباط سطح علمی متخصصین کلیه رشته‌های پزشکی کشور به صورت سمعی و بصیری برمی‌داریم. امید است مشوق ما در این راه باشیم.

لذا علاقه‌مندان می‌توانند برای دریافت هر یک از محصولات اراش‌شبد به ازاء هر مبلغ CD ۳۰۰۰ تومان به حساب جاری ۱۳۴۴۶۳۱۲ بانک رفاه کارگران شعبه میدان انقلاب کد شعبه ۱۱۲ به نام مرکز خدمات فرهنگی سالکان و ارز و پس از فاکس فیش فوق به همراه نشان دهنده دقیق نسبت به خرید اقلام و دریافت کالای مورد نظر خود اقدام نمایند. لازم به ذکر است در صورت نیاز به مکرره اطلاعات تکمیلی می‌توانند به نشانه مرکز مراجعه و یا با تلفن زیر تماس حاصل نمایند.

نشان مرکز: تهران، میدان انقلاب – خ. کارگر جنوب – خ. لیافی‌نژاد بین کارگر و جمال‌زاده برای سه‌سیمین ۷۹ کاله ۳۸۹۸۴۶۰۳۰
Notice: The authors and publisher have made every effort to ensure that the patient care recommended herein, including choice of drugs and drug dosages, is in accord with the accepted standard and practice at the time of publication. However, since research and regulation constantly change clinical standards, the reader is urged to check the product information sheet included in the package of each drug, which includes recommended doses, warnings, and contraindications. This is particularly important with new or infrequently used drugs. Any treatment regimen, particularly one involving medication, involves inherent risk that must be weighed on a case-by-case basis against the benefits anticipated. The reader is cautioned that the purpose of this book is to inform and enlighten; the information contained herein is not intended as, and should not be employed as, a substitute for individual diagnosis and treatment.
Preface

This book is designed to serve as a primary source for the identification of diseases and conditions of the mouth and jaws. Dentists and physicians can readily access a wide spectrum of entities with brief, essential text accompanying representative clinical photographs and radiographs. Oral mucosal diseases, salivary gland disorders, and odontogenic diseases are included, as well as general medical conditions reflected or manifested in the mouth. A therapeutics section outlines practical treatment approaches to many oral diseases within the format of a prescribing formula to guide the clinician.

Although the text and illustrations are not fully comprehensive, we have included typical examples of each entity. A text style has been adopted that allows the reader to quickly ascertain the essence of the disease or condition without scanning through sentence prose. We feel the initial encounter of the clinician with the patient is best served by a synoptic clinical approach. With that in mind, we have intentionally not included histopathologic illustrations. It is understood by all clinicians that photomicrographs and detailed histopathology are often the underpinning of fully understanding the disease process, with a more thorough investigation by the reader accomplished by accessing reference texts and original literature sources. A basic list of references is suggested at the conclusion of this guide.

Following the identification of the disease or condition (by way of reviewing the photographs and accompanying text material) and the establishment of a rational and logical differential diagnosis, the clinician can access the therapeutics section. Treatment for selected diseases is streamlined and, in many cases, is presented in the form of the written prescription.

We and the publishers feel that the clinical, presentation-directed chapter design and format will best assist the clinician since the patient's presentation provides the first clue to the diagnostic process. We have endeavored to provide a useful, accurate, and
practical text that will be efficient and time saving for the dermatologist, otolaryngologist, and oral clinician, including the general dentist and dental specialist.

We express our deepest thanks to our families and our office staff for their understanding and help through the planning and execution of this work. To the staff of BC Decker Inc., we offer our collective gratitude for their helpful suggestions, care, and skill in bringing our effort to fruition.

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White Lesions

Actinic (Solar) Cheilitis

Etiology
- Chronic, excessive exposure to solar radiation; ultraviolet spectrum (ranging from 290 to 320 nm) most damaging
- Fair-complexioned people more severely affected than others
- May progress to cutaneous actinic keratosis and/or squamous cell carcinoma

Clinical Presentation
- Vermilion portion of lower lip
- Pale irregularly opaque (keratotic) surface with intervening red (atrophic) zones
- Obfuscated to effaced cutaneous-vermilion border
- More advanced lesions are scaly, crusted and/or indurated.
- Progression to carcinoma often heralded by persistent ulceration or erosion

Microscopic Findings
- Hyperkeratosis
- Epithelial atrophy
- Variable degrees of epithelial dysplasia
- Amphophilic to basophilic change in submucosa (elastosis)
- Telangiectasia

Diagnosis
- Thermal/chemical burn ruled out by history
- Chronic ultraviolet light exposure
- Biopsy findings

Differential Diagnosis
- Exfoliative cheilitis
- Squamous cell carcinoma
**Treatment**

- Prevention of further damage with sunscreens blocking long-wave ultraviolet A (UVA) and short-wave ultraviolet B (UVB) light
- Biopsy of clinically suspicious areas
- CO₂ laser vermilionectomy
- Topical 5-fluorouracil or vermilionectomy for severe disease
- Excision or resection-reconstruction if malignant transformation has occurred

**Prognosis**

- Lifelong follow-up
- Up to 10% develop into squamous cell carcinoma.
- When carcinoma develops, growth tends to be slow and metastasis occurs late; 85 to 90% long-term survival
Candidiasis

**Etiology**
- Infection with a fungal organism of the *Candida* species, usually *Candida albicans*
- Associated with predisposing factors: most commonly, immunosuppression, diabetes mellitus, antibiotic use, or xerostomia (due to lack of protective effects of saliva)

**Clinical Presentation**
- Acute (thrush)
  - Pseudomembranous
  - Painful white plaques representing fungal colonies on inflamed mucosa
  - Erythematous (acute atrophic): painful red patches caused by acute *Candida* overgrowth and subsequent stripping of those colonies from mucosa
- Chronic
  - Atrophic (erythematous): painful red patches; organism difficult to identify by culture, smear, and biopsy
  - “Denture-sore mouth”: a form of atrophic candidiasis associated with poorly fitting dentures; mucosa is red and painful on denture-bearing surface
  - Median rhomboid glossitis: a form of hyperplastic candidiasis seen on midline dorsum of tongue anterior to circumvallate papillae
  - Perlèche: chronic *Candida* infection of labial commissures; often co-infected with *Staphylococcus aureus*
  - Hyperplastic/chronic hyperplastic: a form of hyperkeratosis in which *Candida* has been identified; usually buccal mucosa near commissures; cause and effect not yet proven
  - Syndrome associated: chronic candidiasis may be seen in association with endocrinopathies

**Diagnosis**
- Microscopic evaluation of lesion smears
- Potassium hydroxide preparation to demonstrate hyphae
- Periodic acid–Schiff (PAS) stain
- Culture on proper medium (Sabouraud’s, corn meal, or potato agar)
- Biopsy with PAS, Gomori’s methenamine silver (GMS), or other fungal stain of microscopic sections
**Differential Diagnosis**
- Allergic or irritant contact stomatitis
- Atrophic lichen planus

**Treatment**
- Topical or systemic antifungal agents
  - For immunocompromised patients: routine topical agents after control of infection is achieved, usually with systemic azole agents
  - See “Therapeutics” section
- Correction of predisposing factor, if possible
- Some cases of chronic candidiasis may require prolonged therapy (weeks to months).

**Prognosis**
- Excellent in the immunocompetent host
Exfoliative Cheilitis

Etiology
• Causes may be atopic, contact, factitious, infectious, systemic, or medication induced.

Clinical Presentation
• Usually involves lower lip (in both genders); can involve both lips
• Tender or asymptomatic crusts and impacted scale of vermillion
• Minimal inflammation

Diagnosis
• Clinical appearance
• Nonspecific microscopy results

Differential Diagnosis
• Atopic cheilitis
• Actinic cheilitis
• Contact cheilitis

Treatment
• Determination of cause
• Supportive care
• Topical or intralesional corticosteroids, including lip ointments/pomade (hypoallergenic)
• Topical tacrolimus ointment

Prognosis
• Chronic
• Psychologic support for factitial cheilitis
White Lesions
Fordyce’s Granules

Etiology
• Ectopic sebaceous glands within the oral mucosa and vermilion portion of the lips

Clinical Presentation
• Multiple, scattered, yellowish pink, maculopapular granules
• Buccal mucosa and vermilion of lips predominantly affected
• Asymptomatic
• Increasingly prominent after puberty

Diagnosis
• Bilateral distribution and appearance
• Lack of symptoms
• If biopsy performed, normal sebaceous glands in the absence of hair follicles noted

Differential Diagnosis
• Candidiasis

Treatment
• None
• Reassurance

Prognosis
• Excellent
Geographic Tongue

Etiology
- Unknown; may be familial
- May be related to atopy
- Small percentage associated with cutaneous psoriasis

Clinical Presentation
- May be symptomatic in association with spicy or acidic foods
- Focal red depapillated areas bordered by slightly elevated, yellowish margin
- Dynamic behavior: changes in shape, size, intensity day to day
- Dorsal and lateral tongue surfaces affected predominantly
- Ventral tongue and other areas less often involved
- Often associated with fissured tongue

Diagnosis
- Location and appearance
- Biopsy confirmation usually unnecessary

Differential Diagnosis
- Reiter’s syndrome
- Lichen planus
- Lupus erythematosus
- Candidiasis
- Psoriasis

Treatment
- None, if asymptomatic
- Topical corticosteroids, if symptomatic

Prognosis
- Excellent
- No malignant potential
- May last months to years with periods of remission
White Lesions
Hairy Leukoplakia

Etiology
- Probably due to opportunistic Epstein-Barr virus (EBV) infection of epithelial cells
- Usually in an immunocompromised or immunosuppressed host

Clinical Presentation
- Usually arises on lateral tongue border
- Early lesions are fine, white, vertical streaks with an overall corrugated surface
- Later lesions may be thickened to be plaque-like
- Extensive lesions can involve dorsum of tongue and buccal mucosa
- May serve as a pre-AIDS (acquired immunodeficiency syndrome) sign

Diagnosis
- Incisional biopsy findings show characteristic EBV nuclear inclusions in upper-level keratinocytes

Differential Diagnosis
- Frictional hyperkeratosis
- Lichen planus
- Hyperplastic candidiasis

Treatment
- None necessary; predisposing condition to be investigated
- Can be suppressed with acyclovir for esthetics
- Antiviral acyclovir
- Podophyllin resin topically

Prognosis
- May herald human immunodeficiency virus (HIV) disease in vast majority of cases
- Also may be present after AIDS is established
Hairy Tongue

Etiology
- Generally unknown
- May be related to poor oral hygiene, soft diet, heavy smoking, systemic or topical antibiotic therapy, radiation therapy, xerostomia, or use of oxygenating mouth rinses (H₂O₂, sodium perborate)

Clinical Presentation
- Elongated, hyperkeratotic filiform papillae on tongue dorsum producing a “furred” to “hairy” texture
- Color varies from tan to brownish yellow to black depending upon diet, drugs, chromogenic organisms
- Symptoms usually minimal; may produce gagging or tickling sensation on palate

Diagnosis
- Clinical features
- Culture or cytologic studies not helpful

Treatment
- Physical débridement (brushing with a soft-bristled toothbrush, 5 to 15 strokes, once or twice daily)
- Topical podophyllin (5% in benzoin) followed by débridement
- Elimination of cause, if identified

Prognosis
- Excellent
White Lesions
Leukoedema

Etiology
• Unknown
• Benign; common in general population, with racial clustering in Blacks

Clinical Presentation
• Symmetric, asymptomatic
• Buccal mucosa involved by gray-white, diffuse, milky surface with an opalescent quality
• Wrinkled surface features at rest
• Dissipation of changes with stretching of mucosa

Diagnosis
• Clinical recognition is sufficient.
• Biopsy findings will show marked intracellular edema of spinous layer.
• Individual cells with clear cytoplasm and compact nuclei
• Normal basal cell layer

Differential Diagnosis
• Cheek chewing
• Hereditary benign intraepithelial dyskeratosis
• White sponge nevus
• Lichen planus
• Candidiasis

Treatment
• None necessary; no relation to dysplasia/carcinoma
• Reassurance

Prognosis
• Excellent
Leukoplakia

Etiology
- Essentially unknown, although many cases related to use of tobacco or areca nut in its various formulations
- Other possible factors include nutritional deficiency (iron, vitamin A) and infection (Candida albicans, human papillomavirus).

Clinical Presentation
- An idiopathic white (sometimes white-and-red) patch
- Most common on lip, gingiva, buccal mucosa
- Increased risk of dysplasia or carcinoma when occurring on tongue, floor of mouth, vermilion portion of lip
- Clinical subsets include homogeneous, verrucous, speckled, and proliferative verrucous leukoplakia (proliferative form may be multiple and persistent)
- Cases may advance or regress unpredictably—reflective of a dynamic process
- Most occur in the fifth decade and beyond
- Progress to dysplasia or malignancy may occur with little or no change in clinical appearance.

Diagnosis
- Performance of a biopsy is mandatory after elimination of any suspected causative factors
- Multiple biopsies of large lesions are needed to be performed due to microscopic heterogeneity within a single lesion.

Differential Diagnosis
- Other white lesions
  - Frictional keratosis
  - Hyperplastic candidiasis
- Burn (thermal/chemical)
- Lichen planus
- Genetic alterations (genodermatoses)
  - White sponge nevus
  - Dyskeratosis
- Hereditary benign intra-epithelial dyskeratosis

Treatment
- Excision modalities (surgery, laser ablation, cryosurgery)
- Option to observe lesions diagnosed as benign hyperkeratosis or mild dysplasia
• Possibly photodynamic therapy
• Topical cytotoxic drugs (bleomycin) remain experimental.
• Recurrences common following apparent complete excision

Prognosis
• Guarded
• Observation with repeat biopsies to be performed

Prevention
• Elimination of tobacco use and heavy alcohol consumption
• Recurrences may be reduced by systemic retinoid therapy.
• Possible dietary measures
Lichenoid Drug Eruptions

Etiology
• Hypersensitivity to drugs including sulfasalazine, angiotensin-converting enzyme inhibitors, nonsteroidal anti-inflammatory drugs, β-blockers, gold, antimalarials, sulfonylurea compounds
• Contact hypersensitivity
• Idiopathic reaction to dental restorations including amalgam, composites, gold, other metals

Clinical Presentation
• White striae or papules, as with lichen planus
• Lesions may appear ulcerative with associated tenderness or pain.
• Most often in buccal mucosa and attached gingiva, but any site may be involved

Diagnosis
• Identification and elimination of causative substance
• Biopsy of areas unresponsive to elimination strategy to demonstrate characteristic keratosis and interface inflammation and associated changes
• Patch testing performed to confirm contact allergens

Differential Diagnosis
• Lichen planus
• Leukoplakia
• Dysplasia/carcinoma

Treatment
• Alternative drugs or material to be chosen
• Topical corticosteroid applications
• Topical tacrolimus applications

Prognosis
• Good
• Observation while lesions exist
Lichen Planus

Etiology
- Unknown
- Autoimmune T cell–mediated disease targeting basal keratinocytes (antigen unknown)
- Lichenoid changes associated with galvanism, graft-versus-host disease (GVHD), certain drugs, contact allergens

Clinical Presentation
- Up to 3 to 4% of population have oral lichen planus
- 0.5 to 1% of population have cutaneous lichen planus; 50% also have oral lesions (25% with oral lesions have concomitant skin lesions)
- White females (60%)
- Occurs in fourth to eighth decades
- Variants: reticular (most common oral form); erosive (painful); atrophic, papular, plaque types; bullous (rare)
- Bilateral and often symmetric distribution
- Oral site frequency: buccal mucosa (most frequent), then tongue, then gingiva, then lips (least frequent)
- Skin sites: forearm, shin, scalp, genitalia

Microscopic Findings
- Hyperkeratosis
- Basal keratinocyte necrosis
- Lymphocytes at epithelial-connective tissue interface

Diagnosis
- Examination of oral mucosa, skin, genitalia
- Negative ocular mucosa history; no history of blistering
- Use of drugs, galvanism, GVHD to be ruled out
- Biopsy
- Direct immunofluorescence–fibrinogen and cytoid bodies at interface help confirm

Differential Diagnosis
- Lichenoid drug eruptions
- Lupus erythematosus
- Mucous membrane pemphigoid
- Erythema multiforme
- Contact stomatitis
**Treatment of Oral Lichen Planus**

- Mild to moderate: topical corticosteroids
- Severe: systemic immunosuppression, chiefly with prednisone
- Corticosteroid-sparing drugs with prednisone
- Topical tacrolimus ointment

**Prognosis**

- Control, not cure, can be expected.
- Good prognosis; rare malignant transformation (0.5–3%)
- May be cyclic; may last for years/decades
- Tends to be chronic
Morsicatio Buccarum/Labiorum (Cheek and Lip Chewing)

Etiology
• Chronic, low-grade biting habit

Clinical Presentation
• Shaggy, white, keratotic surface
• Surface often appears granular to macerated
• More uniform keratotic surface may develop over time if habit continues
• Most common sites are lip and buccal mucosa

Microscopic Findings
• Very irregular, fimbriated surface keratin
• Surface bacterial colonization
• No connective tissue changes

Diagnosis
• Presentation
• Biopsy

Differential Diagnosis
• Leukoedema
• Leukoplakia
• Lichen planus
• Lichenoid tissue reactions

Treatment
• Elimination of hyperfunction habit

Prognosis
• Excellent
Proliferative Verrucous Leukoplakia

Etiology
- Some associated with human papillomavirus types 16 and 18
- Role of tobacco and other risk factors
- Represents a clinicopathologic spectrum of disease
- Multiple lesions develop from hyperkeratosis and/or verrucous hyperplasia to verrucous carcinoma or papillary squamous cell carcinoma

Clinical Presentation
- Slowly progressive and persistent
- Initially a flat hyperkeratotic to warty surface
- Surface may be friable
- Typically multiple and recurrent
- Seen in middle-aged to elderly patients

Diagnosis
- Based upon appearance, clinical course, and microscopic diagnosis (ie, clinical-pathologic correlation)
- Microscopic diagnoses include epithelial hyperplasia, hyperkeratosis, verrucous hyperplasia, “atypical papillary-verrucal proliferation,” verrucous or well-differentiated squamous cell carcinoma

Differential Diagnosis
- Idiopathic leukoplakia
- Oral warts/condyloma
- Verrucous/squamous cell carcinoma

Treatment
- Surgical excision
  - Mucosal stripping or excision for benign lesions
  - Wide excision to resection for advanced lesions
  - Laser ablation for benign/atypical lesions
  - Systemic retinoids to control keratosis
Prognosis
- Progression to carcinoma frequently occurs, usually many years after initial lesion(s) develops.
- Fair to good prognosis after malignant transformation
- Frequent follow-up visits recommended and surgical intervention as new/recurrent lesions develop
Smokeless Tobacco Keratosis (Snuff Pouch)

Etiology
- Persistent habit of holding ground tobacco within the mucobuccal vestibule

Clinical Presentation
- Usually in men in Western countries
- Powdered snuff use prevalent in Southeast United States often by women
- Mucosal pouch with soft, white, fissured appearance
- Surface may be pumice-like to verrucous
- Leathery surface due to chronic tobacco use over many years

Microscopic Findings
- Hyperkeratosis with parakeratotic “chevron sign” at surface
- Increased vascularity
- Older lesions with hyalinization in submucosa and minor salivary glands
- Epithelial dysplasia and carcinoma may evolve.

Diagnosis
- Clinical appearance
- Biopsy

Differential Diagnosis
- Leukoplakia (idiopathic)
- Mucosal burn (chemical/thermal)

Treatment
- Discontinuation of habit
- If dysplasia is present, stripping of mucosal site

Prognosis
- Generally good with tobacco cessation
- Malignant transformation to squamous cell carcinoma or verrucous carcinoma occurs but less frequently than does smoking-related carcinoma.
White Lesions
Submucous Fibrosis

Etiology
- Results from direct mucosal contact with a quid containing areca (betel) nut, tobacco, and other ingredients; alkaloids and tannin in the areca nut are liberated by action of slaked lime within the quid, which is wrapped with the betel leaf
- Risk of oral squamous cell carcinoma is increased several-fold

Clinical Presentation
- Early phase: tenderness, vesicles, erythema, burning, melanosis
- Later phase: mucosal rigidity, trismus
- Sites most often affected: buccal mucosa, soft palate
- Leukoplakia of surface with pallor
- Deep scarring, epithelial atrophy in cheeks, soft palate

Microscopic Findings
- Biopsy results show submucosal deposition of dense collagen.
- Epithelial thinning, hyperkeratosis
- Epithelial dysplasia found in up to 15% of cases

Diagnosis
- Appearance
- History

Differential Diagnosis
- Lichen sclerosus

Treatment
- Intralvesional corticosteroid placement
- Surgical release of scar bands in latter stages
- Careful follow-up and vigilance for development of squamous cell carcinoma

Prognosis
- Irreversible
- Fair
White Lesions

Both photographs courtesy of Dr. John S. Greenspan.
White Sponge Nevus

Etiology
- Hereditary (autosomal-dominant) disorder of keratinization affecting nonkeratinizing oral, esophageal, and anogenital mucosal epithelium
- Point mutations in the keratin 4 and/or 13 genes

Clinical Presentation
- Asymptomatic
- Deeply folded, thickened, white mucosa
- Buccal mucosa chiefly affected
- No functional impairment
- Increased prominence during second decade

Microscopic Findings
- Parakeratosis, acanthosis, intracellular edema
- Perinuclear condensation of keratin

Diagnosis
- Clinical appearance
- Family history
- Microscopic findings

Differential Diagnosis
- Idiopathic leukoplakia
- Chemical/thermal burn
- Chronic low-grade trauma (morsicatio)

Treatment
- None required
- No malignant potential

Prognosis
- Excellent
Red/Blue Lesions

Ecchymosis

Etiology
- Soft tissue hemorrhage
- Blood dyscrasia with secondary thrombocytopenia, hemophilia
- Vascular wall defects
- Coagulopathy
- Trauma

Clinical Presentation
- Larger than pinpoint spots (ie, larger than petechiae)
- Nonvesicular, macular surface
- Lesions do not blanch with pressure
- Red to reddish blue to brown color

Diagnosis
- Characteristic size, color
- History
- Blood count, coagulation profile

Differential Diagnosis
- Hemophilia, Kaposi’s sarcoma, hemangioma, thrombocytopenia, von Willebrand’s disease, leukemia, trauma

Treatment
- Identification of etiology, and corresponding treatment

Prognosis
- Excellent
Erythroplakia

Etiology
- Unknown: a red patch that cannot be clinically attributed to another condition
- Contributing factors include tobacco use, alcohol consumption

Clinical Presentation
- Red, often velvety, well-defined patch(es)
- Most common on floor of mouth, retromolar trigone area, lateral tongue
- Usually asymptomatic
- May be smooth to nodular
- Chiefly in males

Diagnosis
- Appearance; history of tobacco/alcohol use
- Biopsy results differentiate from inflammatory and atrophic lesions

Differential Diagnosis
- Erythematous (atrophic) candidiasis
- Kaposi’s sarcoma
- Ecchymosis
- Contact stomatitis
- Vascular malformation
- Squamous cell carcinoma
- Geographic tongue/erythema migrans

Treatment
- Surgical excision if proven dysplastic/malignant

Prognosis
- Fair to good depending upon microscopic diagnosis
- Almost all cases are premalignant to malignant upon initial discovery.
Fissured Tongue

Etiology
• Unknown
• May be hereditary
• Occurs with greater prevalence as population ages

Clinical Presentation
• Multiple crenations or fissures
• May be seen in association with erythema migrans/geographic tongue
• Prominence increases with age
• Usually asymptomatic
• A component of Melkersson-Rosenthal syndrome
• May be a source of halitosis

Diagnosis
• Characteristic appearance
• If symptomatic (pain, burning), may be related to the following:
  • Secondary candidiasis (antifungal prescription)
  • Idiopathic factors

Treatment
• Usually none
• With candidal colonization, topical antifungal preparations are effective.
• Careful débridement with soft-bristled brush, 5 to 15 strokes, once or twice daily

Prognosis
• Excellent
Red/Blue Lesions
Hemangioma

Etiology

- Benign developmental anomalies of blood vessels that may be subclassified as congenital hemangiomas and vascular malformations
- “Congenital hemangioma” usually noted initially in infancy or childhood (hamartomatous proliferation)
- Congenital hemangioma due to proliferation of endothelial cells
- “Vascular malformations” due to abnormal morphogenesis of arterial and venous structures

Clinical Presentation

- Congenital lesions usually arise around time of birth, grow rapidly, and usually involute over several years.
- Malformations generally are persistent, grow with the child, and do not involute.
- Color varies from red to blue depending on depth, degree of congestion, and caliber of vessels
- Range in size from few millimeters to massive with disfigurement
- Most common on lips, tongue, buccal mucosa
- Usually asymptomatic
- Sturge-Weber syndrome (trigeminal encephaloangiomatosis) includes cutaneous vascular malformations (port wine stains) along trigeminal nerve distribution, mental retardation, and seizures.

Diagnosis

- Aspiration
- Blanching under pressure (diascopy)
- Imaging studies

Differential Diagnosis

- Purpura
- Telangiectasia
- Kaposi’s sarcoma
- Other vascular neoplasms
Treatment
• Observation
• Congenital hemangiomas typically involute, whereas vascular malformations persist.
• Surgery (scalpel, cryosurgery, laser [argon, copper])—congenital hemangiomas usually are circumscribed and more easily removed than are vascular malformations, which are poorly defined. (Vascular malformations are associated with excessive bleeding and recurrence.)
• Sclerotherapy
• Microembolization followed by resection for large malformations or if bleeding is problematic

Prognosis
• Guarded
Hereditary Hemorrhagic Telangiectasia (Rendu-Osler-Weber Syndrome)

Etiology
- Not uncommon, familial (autosomal-dominant) mucocutaneous vascular disease
- Some cases may be nonfamilial (spontaneous mutation).
- Arteriovenous (eg, pulmonary) malformations also can occur.

Clinical Presentation
- Multifocal, macular to slightly papular red lesions of skin and mucosa
- Most common on lips, tongue, buccal mucosa, finger tips
- Commonly associated with epistaxis due to involvement of nasal mucosa
- Increase in number and prominence with age
- Blanch under pressure (diascopy positive)
- Lesions can affect gastrointestinal mucosa, which may rupture with associated signs of chronic gastrointestinal blood loss; may produce anemia

Diagnosis
- Family history
- Distribution of lesions

Differential Diagnosis
- CREST syndrome (calcinosis cutis, Raynaud’s phenomenon, esophageal dysfunction, sclerodactyly, telangiectasia)
- Chronic hepatitis
- Radiation-induced vascular alterations

Treatment
- Observation
- Monitoring of pulmonary lesions; embolization if indicated

Prognosis
- Lifelong follow-up/monitoring
- 4 to 10% death rate from complications of the disease
Red/Blue Lesions
Kaposi’s Sarcoma

Etiology

- Several forms
  - Classic idiopathic form affecting extremities
  - Endemic form (African)
  - Immunosuppression-associated form
  - Acquired immunodeficiency syndrome (AIDS)-associated form
- All forms, especially AIDS-associated and immunosuppression-associated forms, may be caused by or closely related to a herpesvirus (human herpesvirus 8 [HHV-8] or Kaposi’s sarcoma–associated herpesvirus [KSHV]).

Clinical Presentation

- Classic form associated with slow but pernicious growth over many years; oral lesions rarely seen
- Endemic form more rapid; oral lesions rarely seen
- AIDS-associated KS most commonly seen on keratinized mucosa/mucoperiosteal tissues; strong predilection for hard palate, followed by gingiva, buccal mucosa, and tongue (prevalence decreasing with treatment for AIDS)
- Evolution from bluish macule to nodule(s)
- Evolution to multiple lesions
- May precede or follow cutaneous lesions
- Usually asymptomatic

Diagnosis

- Location and appearance
- May occur in up to one-third of AIDS patients
- Biopsy showing spindle cell proliferation with vascular slits, extravascular red blood cells

Differential Diagnosis

- Hematoma
- Hemangioma
- Ecchymosis
- Malignant melanoma
- Pyogenic granuloma
- Amalgam tattoo
Treatment of AIDS-Associated Form

- Radiation therapy: single fraction or equivalent fractionated therapy of 800 cGy
- Intralesional therapy: interferon-α, vincristine, vinblastine (2 mg/cc), sclerosing agents (sodium morrhuate)
- Systemic chemotherapy: interferon-α, vincristine, vinblastine, bleomycin, daunorubicin
- Most treatment is palliatively directed.

Prognosis

- Variable, depending upon host’s immune status, but generally poor in AIDS-associated form
Petechiae

Etiology
- Viral infection (Epstein-Barr virus [EBV]-mononucleosis; measles), rickettsial infection
- Thrombocytopenia, leukemia
- Disseminated intravascular coagulation (DIC)
- Trauma: prolonged coughing, frequent vomiting, giving birth, fellatio, violent Valsalva maneuvers

Clinical Presentation
- Pinpoint hemorrhage into mucosa/submucosa
- Asymptomatic
- Usually involves the soft palate
- No blanching on pressure (diascopy)

Diagnosis
- Clinical features
- History, determination of underlying cause

Differential Diagnosis
- See “Etiology”

Treatment
- None; observation only

Prognosis
- Variable, depending upon etiology
Plasma Cell Gingivitis

Etiology
• Usually represents a hypersensitivity phenomenon to an agent such as the following:
  • Cinnamon/cinnamon flavoring
  • Candy flavors
  • Toothpaste/mouthwash
  • Plaque antigens

Clinical Presentation
• Reddened, velvety gingival surface
• Surface epithelium becomes nonkeratinized.
• Limited to attached gingiva

Diagnosis
• Response to elimination of possible etiologic agents
• Biopsy results show plasma cell infiltration within the submucosa and lamina propria beneath an acanthotic epithelium.
• Patch testing

Differential Diagnosis
• Lupus erythematosus
• Wegener’s granulomatosis
• Chronic candidiasis
• Lichen planus
• Mucous membrane pemphigoid

Treatment
• Elimination of causative factor

Prognosis
• Reversal with removal of causative agent
Pyogenic Granuloma

Etiology
• A reactive hyperplasia of capillaries and fibroblasts
• Related to chronic, persistent trauma or irritation (eg, calculus or foreign body)
• Misnomer—neither pyogenic nor granulomatous

Clinical Presentation
• Occurs at any age, but usually in children, young adults, and women
• Red, lobular to smoothly contoured appearance
• When ulcerated, a yellow fibrinous exudate covers the lesion.
• Sessile to pedunculated commonly on gingiva, but also on areas that are traumatized (eg, lower lip, buccal mucosa)
• Bleeds easily but is painless

Microscopic Findings
• Hyperplastic granulation tissue
• Often lobular aggregation of proliferative vascular tissue
• Acute and chronic inflammation may be present, especially if ulcerated

Diagnosis
• History of gradual to rapid onset
• Identification of a stimulus or causative factor (eg, trauma, physical irritant)
• Histologic evaluation

Differential Diagnosis
• Peripheral giant cell granuloma
• Peripheral ossifying fibroma
• Metastatic tumor
• Kaposi’s sarcoma
• Vascular malformation
Red/Blue Lesions

Treatment
• Local excision, scalpel excision
  • Laser ablation
  • Electrosurgery
• If on gingiva, excision should be extended to the periosteum or periodontal ligament

Prognosis
• Excellent
• Recurrence occasional
Varices

Etiology
- An abnormal venous dilatation
- Congenital or from damage to vessel wall (trauma, ultraviolet light)
- Occur with increasing frequency over 40 years of age

Clinical Presentation
- Blue, lobulated surface
- Painless, evolves slowly
- Common on lower lip, sublingual regions
- Blanches with compression (diascopy)
- May become thrombosed

Diagnosis
- Clinical appearance
- Histologic viewing of large-caliber, thin-walled vein

Differential Diagnosis
- Mucocele
- Vascular neoplasm
- Blue rubber bleb nevus syndrome
- Hereditary hemorrhagic telangiectasia (Rendu-Osler-Weber syndrome)

Treatment
- Observation only, if stable
- Elimination by excision, sclerotherapy, or laser ablation

Prognosis
- Excellent
Red/Blue Lesions
Vesiculobullous Diseases

Epidermolysis Bullosa

Etiology

• A diverse group of predominantly cutaneous, but also mucosal, mechanobullous diseases
• Inherited form: autosomal dominant or recessive patterns may occur
• Acquired form (acquisita): autoimmune from autoantibodies (immunoglobulin G [IgG]) to type VII collagen deposited within the basement membrane zone and upper dermis or lamina propria

Clinical Presentation

• Variable, depending upon the specific form of many subtypes recognized
• Mucosal lesions range in severity from mild to debilitating, depending on subtype:
  • Inherited forms have wide range of oral mucosal involvement, with most severe form (autosomal recessive, dermolytic) also demonstrating enamel hypoplasia and caries
  • Acquisita form with mucous membrane pemphigoid variant shows oral and conjunctival erosions/blisters
• Mucosal involvement absent in several variants
• Scarring and stricture formation common in severe recessive forms
• Mucosa is often friable, but it may be severely blistered, eroded, or ulcerated.
• Loss of oral anatomic landmarks may follow severe scarring (eg, tongue mucosa may become smooth and atrophic with episodes of blistering and scarring).
• Obliteration of vestibules, reduction of oral opening, ankyloglossia
• Scarring can be associated with atrophy and leukoplakia, with increased risk for squamous cell carcinoma development.
**Microscopic Findings**
- Bullae vary in location depending upon the form that is present:
  - Intraepithelial in nonscarring forms
  - At epithelial–connective tissue junction in dystrophic forms
  - Subepithelial/intradermal in scarring forms
- Ultrastructural findings are as follows:
  - Intraepithelial forms associated with defective cytokeratin groups
  - Junctional forms associated with defective anchoring filaments at hemidesmosomal sites (epithelial–connective tissue junction)
  - Dermal types demonstrate anchoring fibril or collagen destruction.

**Diagnosis**
- Distribution of lesions
- Family history
- Microscopic evaluation
- Ultrastructural evaluation
- Immunohistochemical evaluation of basement membrane zone using specific labeled antibodies as markers for site of blister formation

**Differential Diagnosis**
- Varies with specific form
- Generally includes the following:
  - Bullous pemphigoid
  - Mucous membrane (cicatricial) pemphigoid
  - Erosive lichen planus
  - Dermatitis herpetiformis
  - Porphyria cutanea tarda
  - Erythema multiforme
  - Bullous impetigo
  - Kindler syndrome
  - Ritter’s disease

(continued)
Treatment

• Acquisita form:
  • Some recent success with colchicine and dapsone
  • Immunosuppressive agents including azathioprine, methotrexate, and cyclosporine may be effective

• Acquisita and inherited forms:
  • Avoidance of trauma
  • Dental prevention strategies including extra-soft brushes, daily topical fluoride applications, dietary counseling

Prognosis

• Widely variable depending on subtype
Erythema Multiforme

Etiology
- Many cases preceded by infection with herpes simplex; less often with *Mycoplasma pneumoniae* or other organisms
- May be related to drug consumption, including sulfonamides, other antibiotics, analgesics, phenolphthalein-containing laxatives, barbiturates
- Another trigger may be radiation therapy.
- Essentially an immunologically mediated reactive process, possibly related to circulating immune complexes

Clinical Presentation
- Acute onset of multiple, painful, shallow ulcers and erosions with irregular margins
- Early mucosal lesions are macular, erythematous, and occasionally bullous.
- May affect oral mucosa and skin synchronously or metachronously
- Lips most commonly affected with eroded, crusted, and hemorrhagic lesions (serosanguinous exudate) known as Stevens-Johnson syndrome when severe
- Predilection for young adults
- As many as one-half of oral cases have associated erythematous to bullous skin lesions.
- Target or iris skin lesions may be noted over extremities.
- Genital and ocular lesions may occur.
- Usually self-limiting; 2- to 4-week course
- Recurrence is common.

Diagnosis
- Appearance
- Rapid onset
- Multiple site involvement in one-half of cases
- Biopsy results often helpful, but not always diagnostic

Differential Diagnosis
- Viral infection, in particular, acute herpetic gingivostomatitis
  (Note: Erythema multiforme rarely affects the gingiva.)
• Pemphigus vulgaris
• Major aphthous ulcers
• Erosive lichen planus
• Mucous membrane (cicatricial) pemphigoid

**Treatment**
• Mild (minor) form: symptomatic/supportive treatment with adequate hydration, liquid diet, analgesics, topical corticosteroid agents
• Severe (major) form: systemic corticosteroids, parenteral fluid replacement, antipyretics
• If evidence of an antecedent viral infection or trigger exists, systemic antiviral drugs during the disease or as a prophylactic measure may help.
• See “Therapeutics” section for details.

**Prognosis**
• Generally excellent
• Recurrences common
Hand-Foot-and-Mouth Disease

Etiology
- A very common enterovirus infection (coxsackievirus A10 or A16), which may occur in mild epidemic proportion, chiefly in children
- Incubation period is short, usually less than 1 week

Clinical Presentation
- Oral mucosal lesions with focal herpes simplex–like appearance, usually involving nonkeratinized tissue (soft palate, floor of mouth, labial-buccal mucosa)
- Accompanying palmar, plantar, and digital lesions are deeply seated, vesicular, and erythematous
- Short course with mild symptoms

Diagnosis
- Concomitant oral and cutaneous lesions
- Skin lesions commonly involve hands and feet.
- Skin lesions may involve buttocks.
- Antibody-titer increase measured between acute and recovery phases

Differential Diagnosis
- Herpangina
- Herpes simplex infection
- Acute lymphonodular pharyngitis

Treatment
- Symptomatic treatment only
- Patient should be cautioned against the use of aspirin to manage fever.

Prognosis
- Excellent
- Lifelong immunity, but it is strain specific
Herpangina

Etiology
- Most often by members of coxsackievirus group A (7, 9, 10, and 16) or group B (1–5)
- Occasionally due to echovirus 9 or 17

Clinical Presentation
- Incubation period of 5 to 9 days
- Acute onset
- Usually endemic in young children; usually occurs in summer
- Often subclinical
- Posterior oral cavity, tonsillar pillars involved
- Macular erythematous areas precede short-lived vesicular eruption, followed by superficial ulceration
- Accompanied by pharyngitis, dysphagia, fever, malaise, headache, lymphadenitis, and vomiting
- Self-limiting course, usually under 2 weeks

Diagnosis
- Other viral illnesses to be ruled out or separated
- Course, time of year, location of lesions, contact with known infected individual

Differential Diagnosis
- Hand-foot-and-mouth disease
- Varicella
- Acute herpetic gingivostomatitis

Treatment
- Soft diet
- Hydration
- Antipyretics
- Chlorhexidine rinses
- Compounded mouth rinses

Prognosis
- Excellent
Herpetic Stomatitis: Primary

Etiology
• Herpes simplex virus (HSV)
• Over 95% of oral primary herpes due to HSV-1
• Physical contact is mode of transmission

Clinical Presentation
• 88% of population experience subclinical infection or mild transient symptoms
• Most cases occur in those between 0.5 and 5 years of age.
• Incubation period of up to 2 weeks
• Abrupt onset in those with low or absent antibody to HSV-1
• Fever, anorexia, lymphadenopathy, headache, in addition to oral ulcers
• Coalescing, grouped, pinhead-sized vesicles that ulcerate
• Ulcers show a yellow, fibrinous base with an erythematous halo
• Both keratinized and nonkeratinized mucosa affected
• Gingival tissue with edema, intense erythema, pain, and tenderness
• Lips, perioral skin may be involved
• 7- to 14-day course

Diagnosis
• Usually by clinical presentation and pattern of involvement
• Cytology preparation to demonstrate multinucleate virus-infected giant epithelial cells
• Biopsy results of intact macular area show intraepithelial vesicles or early virus-induced epithelial (cytopathic) changes
• Viral culture or polymerase chain reaction (PCR) examination of blister fluid or scraping from base of erosion

Differential Diagnosis
• Herpangina
• Hand-foot-and-mouth disease
• Varicella
• Herpes zoster (shingles)
• Erythema multiforme (typically no gingival lesions)
Treatment
- Soft diet and hydration
- Antipyretics (avoid aspirin)
- Chlorhexidine rinses
- Systemic antiviral agents (acyclovir, valacyclovir) if early in course or in immunocompromised patients
- Compounded mouth rinse

Prognosis
- Excellent in immunocompetent host
- Remission/latent phase in nearly all those affected who have adequate antibody titers
Impetigo

Etiology
- Cutaneous bacterial infection: *Streptococcus* and *Staphylococcus* species
- Is spread through direct contact
- Highly contagious

Clinical Presentation
- Honey-colored, perioral crusts preceded by vesicles
- Flaccid bullae less common (bullous impetigo)

Diagnosis
- Clinical features
- Culture of organism (usually group A, β-hemolytic streptococci or group II *Staphylococcus aureus*)

Differential Diagnosis
- Herpes simplex (recurrent)
- Exfoliative cheilitis
- Drug eruptions
- Other vesiculobullous diseases

Treatment
- Topical antibiotics (mupirocin, clindamycin)
- Systemic antibiotics

Prognosis
- Excellent
- Rarely, poststreptococcal glomerulonephritis may develop.
Mucous Membrane Pemphigoid

Etiology
- Autoimmune; trigger unknown
- Autoantibodies directed against basement membrane zone antigens

Clinical Presentation
- Vesicles and bullae (short lived) followed by ulceration
- Multiple intraoral sites (occasionally gingiva only)
- Usually in older adults
- 2:1 female predilection
- Ocular lesions noted in one-third of cases
- Proclivity for scarring in ocular, laryngeal, nasopharyngeal, and oropharyngeal tissues

Microscopic Findings
- Subepithelial cleft formation
- Linear pattern IgG and complement 3 (C3) along basement membrane zone; less commonly IgA
- Direct immunofluorescence examination positive in 80% of cases
- Indirect immunofluorescence examination usually negative
- Immunoreactants deposited in lamina lucida in most patients

Diagnosis
- Biopsy
- Direct immunofluorescent examination

Differential Diagnosis
- Pemphigus vulgaris
- Erythema multiforme
- Erosive lichen planus
- Lupus erythematosus
- Epidermolysis bullosa acquisita

Treatment
- Topical corticosteroids
- Systemic prednisone, azathioprine, or cyclophosphamide
- Tetracycline/niacinamide
- Dapsone
- See “Therapeutics” section for details.
**Prognosis**

- Morbidity related to mucosal scarring (oropharyngeal, nasopharyngeal, laryngeal, ocular, genital)
- Management often difficult due to variable response to corticosteroids
- Management often requires multiple specialists working in concert (dental, dermatology, ophthalmology, otolaryngology)
Paraneoplastic Pemphigus

Etiology
- Autoimmune, triggered by malignant or benign tumors
- Autoantibodies directed against a variety of epidermal antigens including desmogleins 3 and 1, desmoplakins I and II, and other desmosomal antigens, as well as basement membrane zone antigens

Clinical Presentation
- Short-lived vesicles and bullae followed by erosion and ulceration; resembles oral pemphigus
- Multiple oral sites
- Severe hemorrhagic, crusted erosive cheilitis
- Painful lesions
- Cutaneous lesions are polymorphous; may resemble lichen planus, erythema multiforme, or bullous pemphigoid
- Underlying neoplasms such as non-Hodgkin’s lymphoma, leukemia, thymoma, spindle cell neoplasms, Waldenström’s macroglobulinemia, and Castleman’s disease

Microscopic Findings
- Suprabasilar acantholysis, keratinocyte necrosis, and vacuolar interface inflammation
- Direct immunofluorescent testing is positive for epithelial cell surface deposition of IgG and C3 and a lichenoid tissue reaction interface deposition pattern
- Indirect immunofluorescent testing is positive for epithelial cell surface IgG antibodies
- Special testing with mouse and rat bladder, cardiac muscle, and liver may demonstrate paraneoplastic pemphigus antibodies that bind to simple columnar and transitional epithelia

Diagnosis
- Biopsy of skin or mucosa
- Direct immunofluorescent examination of skin or mucosa
- Indirect immunofluorescent examination of sera including special substrates
Differential Diagnosis
- Pemphigus vulgaris
- Erythema multiforme
- Stevens-Johnson syndrome
- Mucous membrane (cicatricial) pemphigoid
- Erosive oral lichen planus

Treatment
- Identification of concurrent malignancy
- Immunosuppressive therapy

Prognosis
- Good with excision of benign neoplasms
- Grave, usually fatal, with malignancies
- Management is very challenging.
Pemphigus Vulgaris

Etiology
- An autoimmune disease where antibodies are directed toward the desmosome-related proteins desmoglein 3 or desmoglein 1
- A drug-induced form exists with less specificity in terms of immunologic features, clinical presentation, and histopathology

Clinical Presentation
- Over 50% of cases develop oral lesions as the initial manifestation
- Oral lesions develop in 70% of cases
- Painful, shallow irregular ulcers with friable adjacent mucosa
- Nonkeratinized sites (buccal, floor, ventral tongue) often are initial sites affected
- Lateral shearing force on uninvolved skin or mucosa can produce a surface slough or induce vesicle formation (Nikolsky sign)

Microscopic Findings
- Separation or clefting of suprabasal from basal layer of epithelium
- Intact basal layer of surface epithelium
- Vesicle forms at site of epithelial split
- Nonadherent spinous cells float in blister fluid (Tzanck cells)
- Direct immunofluorescence examination positive in all cases
- IgG localization to intercellular spaces of epithelium
- C3 localization to intercellular spaces in 80% of cases
- IgA localization to intercellular spaces in 30% of cases
- Indirect immunofluorescence examination positive in 80% of cases
- General correlation with severity of clinical disease

Diagnosis
- Clinical appearance
- Mucosal manifestations
- Direct/indirect immunofluorescent studies
Differential Diagnosis
- Mucous membrane (cicatrical) pemphigoid
- Erythema multiforme
- Erosive lichen planus
- Drug reaction
- Paraneoplastic pemphigus

Treatment
- Systemic immunosuppression
- Prednisone, azathioprine, mycophenolate mofetil, cyclophosphamide
- Plasmapheresis plus immunosuppression
- IVIg for some recalcitrant cases
- See “Therapeutics” section for details.

Prognosis
- Guarded
- Approximately a 5% mortality rate secondary to long-term systemic corticosteroid–related complications
Recurrent Herpetic Stomatitis: Secondary

**Etiology**
- Herpes simplex virus
- Reactivation of latent virus

**Clinical Presentation**
- Prodrome of tingling, burning, or pain at site of recurrence
- Multiple, grouped, fragile vesicles that ulcerate and coalesce
- Most common on vermilion border of lips or adjacent skin
- Intraoral recurrences characteristically on hard palate or attached gingiva (masticatory mucosa)
- In immunocompromised patients, lesions may occur in any oral site and are more severe (herpetic geometric glossitis).

**Diagnosis**
- Characteristic clinical presentation and history
- Viral culture or PCR examination of blister fluid or scraping from base of erosion
- Cytologic smear
- Direct immunofluorescence examination of smear

**Differential Diagnosis**
- Erythema multiforme
- Herpes zoster (shingles)
- Herpangina
- Hand-foot-and-mouth disease

**Treatment**
- Acyclovir or valacyclovir early in prodrome
- Supportive
- Acyclovir may be used for prophylaxis for seropositive transplant patients
- Ganciclovir may be used for human immunodeficiency virus (HIV)-positive patients, especially those co-infected with cytomegalovirus.
- For recurrent herpes labialis, see “Therapeutics” section.
Prognosis

- Excellent
- Healing without scarring within 10 to 14 days
- Protracted healing in HIV-positive patients
Stevens-Johnson Syndrome

Etiology
• A complex mucocutaneous disease affecting two or more mucosal sites simultaneously
• Most common trigger: antecedent recurrent herpes simplex infection
• Infection with *Mycoplasma* also may serve as a trigger.
• Medications may serve as initiators in some cases.
• Sometimes referred to as “erythema multiforme major”

Clinical Presentation
• Labial vermilion and anterior portion of oral cavity usually affected initially
• Early phase is macular followed by erosion, sloughing, and painful ulceration
• Lip ulcers appear crusted and hemorrhagic.
• Pseudomembrane; foul-smelling presentation as bacterial colonization supervenes
• Posterior oral cavity and oropharyngeal involvement leads to odynophagia, sialorrhea, drooling
• Eye (conjunctival) involvement may occur.
• Genital involvement may occur.
• Cutaneous involvement may become bullous.
• Iris or target lesions are characteristic on skin.

Microscopic Findings
• Subepithelial separation with basal cell liquefaction
• Intraepithelial neutrophils
• Epithelial and connective tissue edema
• Perivascular lymphocytic infiltrate

Diagnosis
• Usually made on clinical grounds
• Histopathology is not diagnostic.

Differential Diagnosis
• Pemphigus vulgaris
• Paraneoplastic pemphigus
• Mucous membrane (cicatricial) pemphigoid
• Bullous pemphigoid
• Acute herpetic gingivostomatitis
• Stomatitis medicamentosa

Treatment
• Hydration and local symptomatic measures
• Topical compounded oral rinses
• Systemic corticosteroid use controversial
• Recurrent, virally associated cases may be reduced in frequency with use of daily, low-dose antiviral prophylactic therapy (acyclovir, famciclovir, valacyclovir).
• May require admission to hospital burn unit

Prognosis
• Good; self-limiting usually
• Recurrences not uncommon
Varicella and Herpes Zoster

Etiology
- Primary and recurrent forms due to varicella-zoster virus (VZV)
- Primary VZV (chickenpox): a childhood exanthem
- Secondary (recurrent) VZV (herpes zoster/shingles) infection: most common in elderly or immunocompromised adults

Clinical Presentation
- Varicella (chickenpox)
  - Fever, headache, malaise, and pharyngitis with a 2-week incubation
  - Skin with widespread vesicular eruption
  - Oral mucosa with short-lived vesicles that rupture forming shallow, defined ulcers
- Herpes zoster (shingles)
  - Unilateral, dermatomal, grouped vesicular eruption of skin and/or oral mucosa
  - Vesicles may coalesce prior to ulceration and crusting.
  - Lesions are painful.
  - Prodromal symptoms along affected dermatome may occur.
  - Pain, paresthesia, burning, tingling
  - Postherpetic pain may be severe.

Diagnosis
- Clinical appearance and symptoms
- Cytologic smear with cytopathic effect present (multinucleated giant cells)
- Viral culture or PCR examination of blister fluid or scraping from base of erosion
- Serologic evaluation of VZV antibody
- Biopsy with direct fluorescent examination using fluorescein-labeled VZV antibody

Differential Diagnosis
- Primary herpes simplex/acute herpetic gingivostomatitis
- Recurrent intraoral herpes simplex
- Pemphigus vulgaris
- Mucous membrane (cicatricial) pemphigoid
**Treatment**

- Symptomatic management in primary infection
- Antiviral drugs (especially acyclovir) in immunocompromised patients or patients with extensive disease
- Systemic corticosteroids may be used to help control/prevent postherpetic neuralgia.
- Pain control to prevent “CNS imprinting”

**Prognosis**

- Generally good
- Recurrences more likely in immunosuppressed patients
Ulcerative Conditions

Actinomycosis

Etiology
• An infection caused by one of the Actinomyces group of bacteria, chiefly the israelii species

Clinical Presentation
• Most common site is the mandible, producing cervicofacial disease
• Associated facial pain, paresthesia, low-grade fever, and persistent swelling and discharge
• Bone lesions may be destructive in nature, with accompanying rarefaction and/or sclerosis or periostitis.
• Cervicofacial form is usually insidious in onset, with a long-term, low-grade course.
• Presents typically as a hard, chronic enlargement of the jaw; extraoral abscess formation may be noted with drainage fluid containing yellow sulfur granules (bacterial colonies)

Diagnosis
• Sulfur granules (1–4 mm) in exudate
• Peripheral club-like structures in bacterial colonies microscopically
• Aerobic and anaerobic culture; actinomycyes are anaerobic or microaerophilic

Differential Diagnosis
• Infection: nocardiosis, fungal, staphylococcal, streptococcal
• Neoplasm (malignant)

Treatment
• Surgical débridement followed by prolonged antibiotic course (penicillin is drug of choice)
• Surgical revision of extraoral drainage sites if indicated

Prognosis
• Excellent
Acute Necrotizing Ulcerative Gingivitis (Vincent’s Infection)

Etiology
• *Fusobacterium nucleatum*, *Borrelia vincentii*, and other bacterial species including *Prevotella* and oral treponemes
• Infection requires modification of local or systemic factors including immunosuppression, local hygiene, nutritional deficiencies, intense smoking, and psychological stress.

Clinical Presentation
• Engorged, enlarged, and blunted interdental papillae with crateriform necrosis
• Symptoms include pain, regional lymphadenitis, fetid breath, fever, and malaise.
• Ulcerated areas covered with grayish pseudomembrane
• Often accompanied by dental plaque and calculus
• Bleeding noted spontaneously or with minimal tissue manipulation
• Extension of disease process into adjacent soft tissues noted on occasion

Diagnosis
• Observation of characteristic blunted, necrotizing interdental papillae with “punched-out” appearance
• Lesions on gingiva only

Differential Diagnosis
• Leukemia
• Immunosuppression-related conditions
• Primary herpetic gingivostomatitis
• Acute forms of leukemia
• Vesiculobullous mucosal diseases (mucous membrane [cicatricial] pemphigoid, erosive/bullous lichen planus, pemphigus vulgaris, paraneoplastic pemphigus)

Treatment
• Local débridement, ultrasonic scaling, good oral hygiene, and home care
• Rinses of chlorhexidine, topical povidone-iodine
• Systemic antibiotics (tetracycline, metronidazole) may be beneficial.
• Identification and elimination of predisposing factor(s)
• Underlying immunosuppression should be suspected if no improvement noted

**Prognosis**
• Excellent
Aphthous Stomatitis

Etiology
- Unknown—probably represents a focal immunodysfunction; no viral or other infectious agent identified
- Triggers vary from case to case (eg, increased stress/anxiety, hormonal changes, dietary factors, trauma)
- Alterations in barrier permeability may be a factor, as occur with human immunodeficiency virus/acquired immunodeficiency syndrome (HIV/AIDS), bone marrow suppression, neutropenia, gluten sensitivity, Crohn’s disease, ulcerative colitis, food allergy, Behçet’s disease, and dietary deficiencies (iron, folate, vitamin B₁₂, zinc).
- Although likely immunologic in nature, the specific mechanism is undetermined.
- Human leukocyte antigen (HLA) subtype susceptibility a factor in some cases (-B₁₂, -B₅₁, and others)
- Affects 18 to 27% of the population; prevalence is approximately 20%

Clinical Presentation
- Recurrent, self-limiting, painful ulcers
- Usually restricted to nonkeratinized oral and pharyngeal mucosa (not hard palate or attached gingiva)
- Well-demarcated ulcers with yellow fibrinous base and erythematous halo
- Three clinical forms: minor ulcers, major ulcers, herpetiform lesions
  - Minor variant (most common subtype)
    - Occasional
    - Single but more often multiple
    - Less than 1 cm in diameter
    - Oval to round shape
    - Healing within 7 to 14 days
  - Major variant (Sutton’s ulcers)
    - 1 cm or greater in diameter
    - Single or less commonly several
    - Deep
    - To ragged edges with elevated edematous margins
• May persist for several weeks to months
• Often heal with scarring
• Herpetiform variant (least common variant)
  • Grouped superficial ulcers 1 to 2 mm in diameter; crops of 10 to 100 lesions
  • In nonkeratinized and keratinized tissues
  • Healing within 7 to 14 days
• No etiologic role for herpes simplex virus
• Recurrent aphthous stomatitis occurs as simple (minor) aphthosis (common) and complex (major) aphthosis (uncommon)
  • Complex aphthosis (severe, almost continuous ulcerations; disabling, large, or severe lesions)
  • Simple aphthosis (mild; episodic: 1–4 episodes/yr; few lesions, usually minor or herpetiform)
• In AIDS patients, lesions are typically more severe and may occur on any oral surface.

**Diagnosis**
• Usually has diagnostic clinical appearance of focal, well-defined ulcers involving nonkeratinized mucosa
• History helpful; a recurrent process
• Positive family history

**Differential Diagnosis**
• Traumatic ulcer
• Chancre
• Recurrent intraoral herpes simplex stomatitis
• Cyclic neutropenia

**Treatment**
• Symptomatic therapy may be adequate.
• Systemic causative factors, if present, should be addressed.
• Tetracycline-based oral rinses may be helpful.
• Corticosteroid therapy is the most rational approach and is a consistently effective treatment.
  • Topical corticosteroids as gels, creams, or ointment 4 to 6 times/d to early lesions
  • Intralesional corticosteroid injections
  • Short-duration systemic corticosteroids (low to moderate doses)

(continued)
• Other immunomodulating drugs may be helpful (dapsone, hydroxychloroquine, topical tacrolimus, ameloxanox).
• Colchicine (0.6–1.2 mg/d) is sometimes beneficial.
• Thalidomide treatment has shown efficacy in clinical trials.
• See “Therapeutics” section for details.

Prognosis
• Simple aphthosis
  • Excellent
  • Cannot be cured
  • Good control with corticosteroids is usually possible.
  • Typically, severity decreases as patient ages.
• Complex aphthosis
  • Needs medical evaluation for intercurrent disease
  • Chronic problem
Behçet’s Disease

Etiology
• A multisystem disease secondary to an immunodysfunction associated with certain HLA subtypes
• HLA-Bw51 clusters in those of Middle Eastern and Northern Asian descent
• HLA-B12 noted more in those of European and North American descent, with mucocutaneous presentation

Clinical Presentation
• Classic signs noted in the oral cavity, eye, and genitalia
• Painful, sometimes debilitating, oral and genital aphthous ulcers
• Ocular lesions: painful conjunctivitis, uveitis, iritis, retinitis, and hypopyon
• Cutaneous signs include the following:
  • Erythema nodosum–like lesions
  • Pustular folliculitis
  • Thrombophlebitis
  • Acneiform eruptions
• Positive pathergy sign is characteristic: sterile pustule at site of sterile intradermal saline injection 48 hours prior
• Other systems, usually secondary to vasculitis, may be involved in the following manner:
  • Central nervous system (headache, paralysis, meningoencephalitis)
  • Gastrointestinal problems (diarrhea, inflammatory bowel disease)
  • Vascular thrombosis, hematologic and other organ system manifestations

Diagnosis
• Oral aphthous ulcerations occurring at least three times per year in association with characteristic manifestations within other systems (ocular, genital, cutaneous problems)

Differential Diagnosis
• Erythema multiforme
• Reiter’s syndrome
• Crohn’s disease
• Mucous membrane (cicatricial) pemphigoid
• Vulvovaginal-gingival variant of erosive lichen planus

**Treatment**
- Systemic corticosteroids
- Immunosuppressive drugs (eg, interferon, TNFα inhibitors)
  - Azathioprine, cyclosporine, chlorambucil, methotrexate
  - Thalidomide has been proven helpful.
  - Dapsone and colchicine may be of value in some cases.

**Prognosis**
- Chronic
- Manageable
Blastomycosis

Etiology

• *Blastomyces dermatitidis* produces the North American form of this disease; *Paracoccidioides brasiliensis* causes South American form and some endemic outbreaks in the United States.

• Transmission is usually by spore inhalation; most infections are confined to the lungs. Extrapulmonary spread is hematogenous to skin, mucosa, bone, viscera, meninges, and the genitourinary tract.

Clinical Presentation

• Acute: pneumonitis, fever, weight loss, night sweats, productive cough

• Chronic: granulomatous lesions of oropharyngeal mucosa, skin; pulmonary signs mimicking tuberculosis

• Skin and mucosal lesions are characterized by proliferative verrucous growth, ulceration, and scarring. Mucosal lesions may mimic carcinoma. Mucocutaneous disease indicates disseminated disease.

Diagnosis

• Cytologic or histopathologic examination of tissue with identification of organism

• Culture of sputum or fresh biopsy material

• Potassium hydroxide preparation from lesion scraping

Differential Diagnosis

• Malignant tumor

• Tuberculosis

• Tertiary syphilis

Treatment

• Systemic antifungals: oral itraconazole

Prognosis

• Guarded

• Untreated disease slowly progressive, fatal
Crohn’s Disease

Etiology
• A granulomatous disease of unknown etiology
• Genetic factors coupled with environmental influences appear of greatest importance

Clinical Presentation
• Extraintestinal/oral findings may include the following:
  • Nodular submucosal nodules of lips (granulomas)
  • Polypoid masses with fissures and ulceration along the buccal/labial sulcus
  • Oral ulcers of nonspecific/aphthous type may develop.
  • May present as orofacial granulomatosis such as granulomatous cheilitis
  • Lesions of pyostomatitis vegetans may be associated.

Diagnosis
• Correlation of mucosal lesions with intestinal symptoms of cramping, diarrhea, and associated weight loss
• Oral mucosal biopsy results demonstrate noncaseating, epitheloid granulomas within submucosa

Differential Diagnosis
• Deep fungal diseases including blastomycosis
• Mycobacterial infections
• Tertiary syphilis and other treponemal infections
• Major aphthous ulcers

Treatment
• Management of underlying intestinal symptoms (nonsteroid anti-inflammatory drugs, systemic corticosteroids)
• Local, oral mucosal lesions: monthly intralesional corticosteroid injections in areas of ulceration until improvement is noted; treatment as needed
• Episodic burst of systemic corticosteroids in association with local management of oral lesions; if condition persists, 7 to 10 days of prednisone with rapid taper to zero, with monitoring
• Management of any associated malabsorption may be helpful.
• Metronidazole, 5-aminosalicylic acid, ileal-release budesonide

**Prognosis**
• Related to response of intestinal symptoms to treatment
Histoplasmosis

Etiology
- A fungal infection caused by *Histoplasma capsulatum*
- Fungus endemic to Ohio and Mississippi River valleys
- Transmission: spore inhalation
- Oral lesions usually secondary to pulmonary lesions with hematogenous dissemination

Clinical Presentation
- General symptoms usually mild
- Oral lesions associated with disseminated form/prior pulmonary lesions
- Chronic ulcerations with necrosis, elevated nodular margins
- May resemble squamous cell carcinoma
- On the tongue, a cobblestone ulcer may be noted.
- Oral ulcers persist until treatment of systemic disease.

Diagnosis
- Demonstration of organisms in biopsy specimen
- Culture
- Serologic demonstration of antigen or antibodies

Differential Diagnosis
- Tuberculosis
- Squamous cell carcinoma
- Other deep fungal infections (eg, coccidioidomycosis, cryptococcosis, and blastomycosis)
- Chronic traumatic ulcer
- Tertiary syphilis

Treatment
- Sometimes none required
- Amphotericin B
- Ketoconazole, fluconazole, itraconazole

Prognosis
- May recover spontaneously
- Generally good unless immunosuppression present
Lupus Erythematosus

Etiology
- An autoimmune-/immunologically mediated condition
- Antibodies demonstrable against an array of cytoplasmic and nuclear antigens
- Most often occurs in women

Clinical Presentation
- Three forms are as follows:
  - Chronic cutaneous (CCLE) or discoid (DLE)
  - Subacute cutaneous (SCLE)
  - Systemic (SLE)
- Black females have highest incidence
- Predominates in women over 40 years
- 80% of patients have concurrent cutaneous findings
- 30 to 40% of SLE patients have oral mucosal findings
- Oral mucosal lesions may appear lichenoid, keratotic, and erosive.
  - Labial vermilion with crusted, exfoliative, erythematous, and keratotic appearance
- Oral findings are most common in CCLE or DLE.

Diagnosis
- Direct immunofluorescent examination of mucosal biopsy
- Serologic correlation (antinuclear antibodies: anti–SS-A, –SS-B, and double-stranded deoxyribonucleic acid)

Differential Diagnosis
- Lichen planus
- Candidiasis
- Hypersensitivity/lichenoid eruption
- Leukoplakia

Treatment
- Complex—dependent on LE variant present and level of disease expression
- Systemic corticosteroids and immunosuppressive agents for SLE
- Topical corticosteroid agents for intraoral lesions
- Low-dose hydroxychloroquine
- Intralesional corticosteroid injections
Ulcerative Conditions

**Prognosis**
- Good prognosis with CCLE or DLE form
- Variable prognosis with SLE
- SCLE has an intermediate prognosis between that of SLE and CCLE or DLE forms.
Mucormycosis (Zygomycosis)

Etiology
• Organisms of Zygomycetes class: *Rhizopus, Absidia, Mucor* genera
• Noted chiefly in immunosuppressed individuals and in uncontrolled diabetics

Clinical Presentation
• Large, irregular, necrotizing ulcers
• Most often involves the palate with concomitant paranasal sinus involvement

Radiographic Findings
• Maxillary sinus opacification
• Irregular sinus wall destruction

Microscopic Findings
• Tissue necrosis with fungal invasion into blood vessels
• Nonseptate, branching, broad hyphae

Diagnosis
• Radiographic findings
• Microscopic findings

Differential Diagnosis
• Maxillary sinus neoplasia
• Maxillary sinus aspergillosis
• Soft tissue infarction
• Soft tissue radionecrosis
• Other deep fungal infections

Treatment
• Surgical débridement
• Intravenous antifungal agents (amphotericin B, ketoconazole)
• Control of underlying disease process

Prognosis
• Good, depending upon underlying systemic factors
Photograph courtesy of Dr. John Knapp.
Neutropenic Ulcer

Etiology
- Idiopathic or iatrogenic neutropenia
- Usually noted when neutrophil count falls below 1,500/mm$^3$

Clinical Presentation
- Sharply defined ulcer(s), often with minimal peripheral erythema
- Ulcer base covered by fibrinous exudate
- Wide variation in size of ulcers

Diagnosis
- Clinical appearance correlated with results of peripheral blood count

Differential Diagnosis
- Major aphthous ulcer
- Traumatic ulcer
- Necrotizing sialometaplasia
- Squamous cell carcinoma

Treatment
- Identification and management of underlying neutropenia
- Supportive therapy

Prognosis
- Relative to ability to manage underlying neutropenia
Radiation-Induced Mucositis

Etiology
• Local tumoricidal doses of ionizing radiation
• Destruction of germinative layers of oral mucosal epithelium within radiation portal
• May be enhanced by intraoral gram-negative bacteria

Clinical Presentation
• Mucosal erythema, atrophy, necrosis, ulceration, and pseudomembrane formation
• Generalized pain and dysfunction
• Ultimately, broadly based, contiguous ulcers form.
• Usually begins within 7 to 10 days following the start of treatment
• Exacerbated by radiation-induced xerostomia or chemotherapy/cytoreductive therapy

Diagnosis
• History and appearance

Differential Diagnosis
• Erythema multiforme
• Chemotherapy-induced stomatotoxicity
• Acute erythematous candidiasis
• Neutropenic ulcer

Treatment
• Systemic antiviral, antibacterial therapy
• Topical agents
  • Water-soluble polymer films
  • Antimicrobials
  • Saline rinses
  • Antifungal agents
• Granulocyte-macrophage colony-stimulating factor
• Local and hygiene measures

Prognosis
• Good
• Improves slowly subsequent to treatment
Squamous Cell Carcinoma

Etiology
• Majority (approximately 80%) related to tobacco and alcohol abuse
• Some cases may be virus associated (human papillomavirus types 16 and 18)
• Stepwise progression of genetic alterations now defined from normal to dysplasia to carcinoma

Clinical Presentation
• Early, usually a white or red-white focal surface alteration
• Later stages with ulceration, induration, elevated margins
• Most common sites: lateral tongue, floor of mouth
• Lower lip vermilion surface also a common location
• Advanced-stage disease has associated limitation of movement, trismus, cervical lymph node metastases

Radiographic Findings
• May erode or invade adjacent bone in later stages
• Irregular, destructive, ill-defined margins in later stages

Microscopic Findings
• Usually well differentiated to moderately differentiated
• Invasive islands, cords of epithelial cells
• Individual cells with nuclear pleomorphism, increased nuclear-to-cytoplasmic ratio, dyskeratosis
• Architectural disorganization of proliferating cells

Diagnosis
• Microscopic analysis of tissue specimen (biopsy)

Differential Diagnosis
• Chronic traumatic ulcer
• Primary syphilis
• Deep fungal infection
• Palatal necrotizing sialometaplasia
• Keratoacanthoma (labial)
Ulcerative Conditions

**Treatment**
- Surgical excision is the treatment of choice.
- Combined surgery and radiation therapy for more advanced-stage lesions
- Adjuvant chemotherapy plays a role in advanced disease.

**Prognosis**
- Results are stage related as follows:
  - Stages 1 and 2: generally good prognosis
  - Stages 3 and 4: generally fair to poor prognosis
Syphilis

Etiology
• *Treponema pallidum* spirochete

Clinical Presentation
• Four clinical types (primary, secondary, and tertiary stages; congenital form) plus neonatal form
  
  • Primary stage (oral)
    • Initial sign usually a firm nodule/papule
    • Labial ulceration most common presentation
    • Ulcer firm, indurated, painless
    • Intraoral chancre is an ulcer covered by a pseudomembrane
    • Lesion is highly infectious
    • Regional cervical lymphadenopathy
    • Spontaneous resolution
  
  • Secondary stage
    • Evolves after 6 weeks to 6 months if patient is untreated
    • Reddish brown macular rash periorally; generalized cutaneous rash
    • Oral lesions are split papule at lip commissures, irregular lesions, serpiginous ulcers, or erosions
    • Mucous patches orally (ulcers covered by mucoid exudate)
    • Lymphadenopathy
    • Highly infectious
    • Spontaneous resolution
  
  • Tertiary stage
    • Develops over 3 to 10 years after primary infection if untreated or inadequately treated (more rapidly in immunocompromised patients)
    • Glossitis: atrophic, leukoplakia features
    • Gumma: destructive, painless, solitary granulomatous ulcer; midline of tongue, palate, especially
  
  • Congenital form
    • Hutchinson’s triad including mulberry molars, barrel-shaped incisors

Diagnosis
• Clinical history, appearance
• Direct smear in primary- and secondary-stage lesions (dark field)
• Serologic studies (eg, VDRL [Venereal Disease Research Laboratories] test): positivity noted in last phase of primary stage
• Biopsy

**Differential Diagnosis**
• Deep fungal infection
• Traumatic ulcer
• Squamous cell carcinoma
• Leukoplakia
• Midline granuloma/Wegener’s granulomatosis

**Treatment**
• Antibiotics
  • Parenteral penicillin (penicillin G benzathine) or ceftriaxone
  • Oral tetracycline or doxycycline

**Prognosis**
• Excellent in primary and secondary stages
• Fair in late (tertiary) phase
Traumatic Granuloma
(Traumatic Eosinophilic Ulcer)

Etiology
• A benign, self-limiting, reactive process of oral mucosa of unknown origin
• Some cases with no history of antecedent trauma

Clinical Presentation
• Rapid onset
• Painful, indurated, crateriform ulcer
• Several weeks mean duration
• 60% occur on the tongue (lateral/ventral)
• Average diameter 1 to 2 cm

Microscopic Findings
• Crateriform ulcer with fibrinous surface
• Deeper areas with granulation tissue, endothelial proliferation
• Inflammatory infiltrate with prominent macrophages
• Underlying muscle injury present with eosinophilic infiltrate

Diagnosis
• History and appearance
• Biopsy results/microscopic findings

Differential Diagnosis: Clinical
• Squamous cell carcinoma
• Major aphthous ulcer
• Lymphoma
• Syphilis
• Granulomatous disease
  • Sarcoidosis
  • Wegener’s granulomatosis
  • Tuberculosis

Differential Diagnosis: Microscopic
• Lymphoma
• Angiolymphoid hyperplasia with stromal eosinophilia
Treatment
• Excision
• Observation only
• Topical or intralesional corticosteroids

Prognosis
• Healing usually within 10 days if excised
• Most lesions heal after a few to several weeks without recurrence.
• Rare cases have multiple recurrences.
**Traumatic Ulcer**

**Etiology**
- Accidental/functional or factitious injury

**Clinical Presentation**
- Tender to very painful
- Ulcer with yellow, fibrinous center and well-defined margins
- Inflammatory/erythematous periphery

**Microscopic Findings**
- Fibrinous surface
- Mixed inflammatory infiltrate
- Granulation tissue at base of lesion

**Diagnosis**
- History of trauma
- Evaluation for ill-fitting dental prosthesis or orthodontic appliance
- Nonspecific histologic findings

**Differential Diagnosis**
- Aphthous ulcer
- Neutropenia-related ulcer
- Factitial ulcer
- Squamous cell carcinoma
- Primary syphilis (chancre)

**Treatment**
- None except elimination of cause
- Healing within 10 days

**Prognosis**
- Excellent
Tuberculosis

Etiology
- *Mycobacterium tuberculosis* usually; less commonly *M. avium-intracellulare*
- Oral lesions form in relation to extension of disease beyond the pulmonary focus
- Increased incidence in immunocompromised patients

Clinical Presentation
- Chronic, nonhealing ulcer with induration
- Borders may be raised or rolled.
- Intrabony lesions are lytic and sequestrate with radiographic features of osteomyelitis.

Microscopic Findings
- Centrally necrotic granulomas with peripheral multinucleated Langhans’ giant cells
- Positive Fite or Ziehl-Neelsen tissue staining of microorganisms

Diagnosis
- Clinical appearance and lesion persistence
- Histopathology
- Tuberculin skin test; two-step Mantoux test
- Culture

Differential Diagnosis
- Squamous cell carcinoma
- Syphilis
- Deep mycotic infection
- Traumatic eosinophilic ulcer
- Lymphoma

Treatment
- Systemic chemotherapy: isoniazid, rifampin, streptomycin, and others
- Note: Multidrug-resistant organism may be present.

Prognosis
- Good
- In the immunosuppressed patient, prognosis is fair.
Wegener’s Granulomatosis

Etiology
• Unknown
• A necrotizing vasculitis with preferential involvement of the respiratory tract early in its course
• Oral involvement unusual and characterized by ulceration and tissue destruction

Clinical Presentation
• Jaw pain, gingival inflammation with petechiae or hyperplasia, palatal ulceration with possible perforation
• Painful salivary gland enlargement may be encountered.
• Classic triad of upper airway, lung, and kidney involvement not required for diagnosis
• May remain localized for prolonged periods prior to multi-organ involvement

Microscopic Findings
• Vasculitis, necrosis, and granulomatous inflammation

Diagnosis
• Oral or upper airway biopsy is helpful in less than one-half of cases.
• Tissue biopsy for microscopic features
• Laboratory studies show the following:
  • Antineutrophil cytoplasmic antibodies (ANCAs)—two staining patterns: cytoplasmic ANCA (high specificity) and perinuclear ANCA
  • Mild, normocytic, normochromic anemia (in 50%)
  • Elevated erythrocyte sedimentation rate

Differential Diagnosis
• Lymphoma, including midline granuloma
• Late-stage syphilis
• Deep fungal infection
• Tuberculosis
• Major aphthous ulcers
Treatment
- Cyclophosphamide/prednisone
- Trimethoprim/sulfamethoxazole (as monotherapy or in combination with immunosuppressive therapy)

Prognosis
- Fair
- Secondary complications related to long-term immunosuppression
Pigmentary Disorders

Addison’s Disease

Etiology
- Adrenal cortical atrophy—85% idiopathic (?autoimmune)
- Oral manifestations due to secondary melanocyte stimulation by increased levels of adrenocorticotropic hormone (ACTH) or β-lipotropin

Clinical Presentation
- Brown macular pigmentation of local or diffuse quality
- Pigmentation usually seen in association with cutaneous bronzing, weakness, weight loss, salt craving, nausea, vomiting, hypotension

Diagnosis
- Confirmation of hypoadrenocorticism by plasma ACTH levels after challenge/stimulation
- Biopsy of mucosa shows melanosis

Differential Diagnosis
- Smoker’s melanosis
- Physiologic/ethnic pigmentation
- Heavy metal deposition/argyrosis
- Medication-related pigmentation
- Peutz-Jeghers syndrome

Treatment
- Management of underlying adrenal insufficiency by corticosteroid replacement therapy

Prognosis
- Good with replacement therapy
Amalgam Tattoo

Etiology
• Implantation or passive/frictional transfer of dental silver amalgam into mucosa

Clinical Presentation
• Gray to black focal macules, usually well defined, but may be diffuse with no associated signs of inflammation
• Typically in attached gingiva, alveolar mucosa, buccal mucosa
• Occasionally may be visible radiographically

Diagnosis
• Radiographs may be useful (intraoral film placement)
• Biopsy may be necessary if clinical diagnosis is in doubt or to rule out lesions of melanocytic origin

Differential Diagnosis
• Vascular malformation
• Mucosal nevus
• Melanoma
• Mucosal melanotic macule
• Melanoacanthoma

Treatment
• Biopsy or observation only

Prognosis
• Little clinical significance if untreated
Melanoacanthoma

Etiology

• A reactive and reversible alteration of oral mucosal melanocytes and keratinocytes
• Usually associated with local trauma

Clinical Presentation

• Unilateral dark plaque; rarely multiple, bilateral
• Most often noted among Blacks and other non-Caucasians
• Occurs more often in women than men by a ratio of 3:1
• History of trauma and local irritation
• Forms rapidly, most often on buccal/labial mucosa
• Asymptomatic melanotic pigmentation

Diagnosis

• Clinical history of rapid onset
• Histologic evaluation
  • Scattered dendritic melanocytes within spongiotic and acanthotic epithelium
  • Increased number of melanocytes along basal layer as single units

Differential Diagnosis

• Melanoma
• Drug-induced pigmentation
• Smoker’s melanosis
• Mucosal melanotic macule
• Mucosal nevus
• Amalgam tattoo

Treatment

• None after establishing the diagnosis
• Often resolves spontaneously

Prognosis

• Excellent
Mucosal Malignant Melanoma

Etiology
- Unknown
- Cutaneous malignant melanoma with relation to sun exposure or familial-dysplastic melanocytic lesions

Clinical Presentation
- Rare in oral cavity (< 1% of all melanomas) and sinonasal tract
- Most cases occur in those older than 30 years of age.
- Usually arises on maxillary gingiva and hard palate
- May exhibit early in situ phase: a macular, pigmented patch with irregular borders
- Progression to deeply pigmented, nodular quality with ulceration
- May arise de novo as a pigmented or amelanotic nodule
- Rarely may be metastatic to the oral cavity as a nodular, usually pigmented mass

Microscopic Findings
- Early stage: atypical melanocytes at epithelial–connective tissue interface, occasionally with intraepithelial spread
- Later infiltration into lamina propria and muscle
- Strict correlation to cutaneous malignant melanoma is not well established, although, as in skin, a similar horizontal or in situ growth phase often precedes the vertical invasive phase.
- Amelanotic forms may require use of immunohistochemical identification: S-100 protein, HMB-45, Melan-A expression

Diagnosis
- Biopsy
- High index of suspicion

Differential Diagnosis
- Mucosal nevus
- Extrinsic pigmentation
- Melanoacanthoma
- Kaposi’s sarcoma
- Vascular malformation
- Amalgam tattoo
- Mucosal melanotic macule
**Treatment**
- Surgical excision
  - Marginal parameters related to depth of invasion and presence of lateral growth
  - Wide surgical margins; resection (including maxillectomy) for large, deeper lesions
- Neck dissection in cases of deep invasion (< 1.25 mm)

**Prognosis**
- Generally poor for most oral malignant melanomas
- Less than 20% survival at 5 years in most studies
Mucosal Melanotic Macule and Ephelides

Etiology
- Most idiopathic, some postinflammatory, some drug-induced
- Multiple lesions suggest syndrome association, as follows:
  - Peutz-Jeghers syndrome
  - Laugier-Hunziker phenomenon
  - Carney’s syndrome
  - LEOPARD syndrome

Clinical Presentation
- Most in adulthood (fourth decade and beyond)
- Most are solitary and well circumscribed
- Lower lip vermilion border most common site, mostly in young women (labial melanotic macule)
- Buccal mucosa, palate, and attached gingiva also involved (mucosal melanotic macule)
- Usually brown, uniformly pigmented, round to ovoid shape with slightly irregular border
- Usually < 5 mm in diameter

Microscopic Findings
- Normal melanocyte density and morphology
- Increased melanin in basal cells and subjacent macrophages (mucosal melanotic macule)
- Increased melanin in basal cells with elongated rete pegs (ephelides)

Diagnosis
- Biopsy

Differential Diagnosis
- Melanoacanthoma
- Mucosal melanotic macule
- Congenital syndromes (Carney’s, Peutz-Jeghers, LEOPARD, Laugier-Hunziker)
Treatment
• Observation
• Biopsy for esthetics
• If increase in size or development of atypical signs occurs, macule should be removed to rule out malignant melanoma, particularly if on palate or alveolar mucosa.

Prognosis
• Excellent
Mucosal Pigmentation: Extrinsic
(Drug or Metal Induced)

Etiology

• Occupational exposure—metals vapors (lead, mercury)
• Therapeutic—metal salt deposits (bismuth, cis-platinum, silver, gold); also nonmetal agents, such as chloroquine, minocycline, zidovudine, chlorpromazine, phenolphthalein, clofazimine, and others

Clinical Presentation

• Focal to diffuse areas of pigmentary change
• If heavy metals are the cause, a typical gray to black color is seen along the gingival margin or areas of inflammation.
• Palatal changes characteristic with antimalarial drugs and minocycline
• Most medications cause color alteration of buccal-labial mucosa and attached gingiva.
• Darkened alveolar bone with minocycline therapy (10% at 1 year, 20% at 4 years of therapy)

Diagnosis

• History of exposure to, or ingestion of, heavy metals or drugs
• Differentiation from melanocyte-related pigmentation by biopsy if necessary

Differential Diagnosis

• When localized: amalgam tattoo, mucosal melanotic macule, melanoacanthoma, mucosal nevus, ephelides, Kaposi’s sarcoma, purpura, malignant melanoma, ecchymosis
• When generalized: ethnic pigmentation, Addison’s disease
• If asymmetric, in situ melanoma must be ruled out by biopsy.

Treatment

• Investigation of cause and elimination if possible

Prognosis

• Excellent
Nevus

Etiology
• Unknown
• Lesion of melanocytic origin within mucosa and skin

Clinical Presentation
• Usually elevated, symmetric papule
• Pigmentation usually uniformly distributed
• Common on skin; unusual intraorally
• Palate and gingiva most often involved

Microscopic Findings
• Most are intramucosal (“dermal”)
• Blue nevi are deeply situated and are composed of spindled nevus cells.
• Other variants are rare; junctional and compound nevi (no dysplastic nevi occur orally)
• Nevus cells are oval/round and are found in unencapsulated nests (theques).
• Melanin production is variable.

Diagnosis
• Clinical features
• Biopsy

Differential Diagnosis
• Melanoma
• Varix
• Amalgam tattoo/foreign body
• Mucosal melanotic macule
• Kaposi’s sarcoma
• Ecchymosis
• Melanoacanthoma

Treatment
• Excision of all pigmented oral lesions to rule out malignant melanoma is advised.
• Malignant transformation of oral nevi probably does not occur.

Prognosis
• Excellent
Nevus of Ota

Etiology
• Idiopathic/congenital
• A proliferation of dermal melanocytes over a specific anatomic distribution

Clinical Presentation
• Macular, grayish blue discoloration of skin and mucosa over the distribution of the ophthalmic and maxillary branches of the trigeminal nerve
• Unilateral distribution
• Sclera on the involved side may be affected.

Microscopic Findings
• Diffuse unencapsulated proliferation of spindle-shaped melanocytes within dermis/submucosa, parallel to surface
• Pigment production may be florid.

Diagnosis
• Clinical presentation

Treatment
• None
• Cosmetic

Prognosis
• Excellent
Pigmentation Disorders: Drug Induced

Etiology
• Therapeutic drug-related tissue pigmentation
• Many drugs may cause change—see listing below

Clinical Presentation
• Macular mucosal discoloration (brown, gray, black)
• Palate and gingiva are most common sites affected
• In addition to mucosal changes, teeth in adults and children may be bluish gray owing to minocycline/tetracycline use (see “Tetracycline Staining” on page 138).

Microscopic Findings
• Most cases are due to increased melanin production. Some are related to the deposition of a drug complex or a metabolized drug.

Diagnosis
• History
• Clinical appearance

Differential Diagnosis
• Physiologic changes
• Smoker’s melanosis
• Mucosal melanotic macule

Treatment
• Drug withdrawal

Prognosis
• Good

Drugs Capable of Producing Tissue Pigmentation
• Antimalarials: chloroquine, mepacrine, quinidine, old-time antimalarials
• Antibiotics: tetracycline group, minocycline
• Antivirals: azidothymidine
• Phenothiazine: chlorpromazine
• Clofazimine
• Heavy metals: gold, mercury salts, silver nitrate, bismuth, lead
• Hormones: ACTH, oral contraceptives
• Cancer/chemotherapy drugs: busulfan, cyclophosphamide, cis-platinum
• Other: methyldopa
Pigmentation Disorders: Physiologic

**Etiology**
- Normal melanocyte activity

**Clinical Presentation**
- Seen in all ages
- Symmetric distribution over many sites, gingiva most commonly
- Surface architecture, texture unchanged

**Diagnosis**
- History
- Distribution

**Differential Diagnosis**
- Mucosal melanotic macule
- Smoking-associated melanosis
- Superficial malignant melanoma

**Treatment**
- None

**Prognosis**
- Excellent
### Pigmentation Disorders: Smoker’s Melanosis

#### Etiology
- Melanin pigmentation of oral mucosa in heavy smokers
- May occur in up to 1 of 5 smokers, especially females taking birth control pills or hormone replacement
- Melanocytes stimulated by a component in tobacco smoke

#### Clinical Presentation
- Brownish discoloration of alveolar and attached labial gingiva, buccal mucosa
- Pigmentation is diffuse and uniformly distributed; symmetric gingival pigmentation occurs most often.
- Degree of pigmentation is positively influenced by female hormones (birth control pills, hormone replacement therapy).

#### Microscopic Findings
- Increased melanin in basal cell layer
- Increased melanin production by normal numbers of melanocytes
- Melanin incontinence

#### Diagnosis
- History of chronic, heavy smoking
- Biopsy
- Clinical appearance

#### Differential Diagnosis
- Physiologic pigmentation
- Addison’s disease
- Medication-related pigmentation (drug-induced pigmentation by chloroquine, clofazimine, mepacrine, chlorpromazine, quinidine, or zidovudine)
- Malignant melanoma

#### Treatment
- None
- Reversible, if smoking is discontinued

#### Prognosis
- Good, with smoking cessation
Tetracycline Staining

Etiology
- Prolonged ingestion of tetracycline or its congeners during tooth development
- Less commonly, tetracycline ingestion causes staining after tooth formation is complete: reparative (secondary) dentin cementum may be stained.

Clinical Presentation
- Yellowish to gray (oxidized tetracycline) color of enamel and dentin
- May be generalized or horizontally banded depending on duration of tetracycline exposure
- Alveolar bone may also be stained bluish red (particularly with minocycline use, 10% after 1 year and 20% after 4 years of therapy).

Diagnosis
- Clinical appearance and history
- Fluorescence of teeth may be noted with ultraviolet illumination.

Differential Diagnosis
- Dentinogenesis imperfecta

Treatment
- Restorative/cosmetic dental techniques

Prognosis
- Good
Verrucal-Papillary Lesions

Condyloma Acuminatum

Etiology
• A sexually transmitted disease
• Associated with human papillomavirus (HPV) types 6, 11, 16, and 18 most often
• Can result in autoinoculation of other sites via trauma
• Lesions located at the site of contact/traumatic event

Clinical Presentation
• Usually on nonkeratinized tissues in immunocompetent patients (soft palate, lingual frenum)
• Pink to whitish pink, exophytic papillary growths with pedunculated outline
• May be solitary or multiple and variably sized, up to 2 to 3 cm
• Can present as papillomatosis of upper respiratory tract

Diagnosis
• Location and appearance
• Demonstration of koilocytotic cellular changes on biopsy
• In situ hybridization or polymerase chain reaction reveals specific HPV subtype
• Electron microscopy demonstrates intranuclear virions

Differential Diagnosis
• Focal epithelial hyperplasia
• Multiple intraoral verruca vulgaris
• Squamous papilloma

Treatment
• Conservative removal
  • Conventional surgery
  • Laser ablation
  • Topical podophyllin
Prognosis

- Recurrences common
- Contagiousness and autoinoculation are considerations.
Focal Epithelial Hyperplasia

Etiology
• A viral infection (HPV 13 or 32), usually found in childhood
• Familial/ethnic clustering often noted, probably secondary through horizontal viral transmission
• Often occurs in native Americans

Clinical Presentation
• Numerous, slightly raised whitish pink asymptomatic papules and irregular plaques that may become confluent
• Size of lesions ranges from a few millimeters to coalescent papules several centimeters in dimension

Microscopic Findings
• Well-defined acanthotic features
• Broadened, anastomosing epithelial ridges with occasional superficial koilocytotic changes

Diagnosis
• Multiple, characteristic lesions
• Biopsy findings
• In situ deoxyribonucleic acid hybridization to demonstrate HPV subtype
• Ultrastructural localization of intranuclear virions

Differential Diagnosis
• Condyloma acuminatum
• Multiple verruca vulgaris

Treatment
• None; lesions usually regress spontaneously
• Excision if esthetic needs demand
• Intralesional interferon therapy

Prognosis
• Excellent
• No reported malignant transformation
Keratoacanthoma

Etiology
• Unknown, may be related to several factors, as follows:
  • Viral—HPV subtypes 11, 13, 24, 33, 57
  • Altered expression of cell cycle proteins including cyclin E, p53, PCNA
  • Keratinocyte dedifferentiation reflected in deficient desmoglein production
  • Immunosuppression
  • Sun damage
• May represent a highly differentiated form of squamous cell carcinoma
• May indicate underlying alimentary neoplasia (Muir-Torre syndrome)

Clinical Presentation
• Usually solitary on sun-exposed areas, including lip
• Initially erythematous papule
• Rapid growth over 4 to 8 weeks
• Nodular, hemispheric, firm nodule
• Central keratin core
• Occasionally regresses spontaneously
• Extremely rare intraorally

Diagnosis
• Clinical evaluation, follow-up
• Histopathology shows keratin plus normal, peripheral epidermis and mature, premature keratinization; no invasion below adnexa; marked pseudoepitheliomatous hyperplasia

Differential Diagnosis
• Squamous cell carcinoma
• Molluscum contagiosum
• Warty dyskeratoma
• Verruca vulgaris
• Pilomatrixoma
• Condyloma acuminatum
• Squamous papilloma
**Treatment**
- Observation and careful follow-up
- Local excision
- Cryotherapy
- Intrallesional chemotherapy (methotrexate, 5-fluorouracil, or bleomycin)

**Prognosis**
- Excellent
Lymphangioma

Etiology
- A benign proliferation of lymphatic vasculature
- Usually congenital in nature

Clinical Presentation
- Superficial or deep in location
- Typically waxes and wanes in size
- Most commonly involves the tongue followed by lips, buccal mucosa, palate
- Facial asymmetry may be a presenting sign.
- Superficial mucosal lymphangiomas resemble caviar or frog’s eggs.
- Deeper lesions present as painless fluctuant masses such as macroglossia.
- Often combined with blood vessels
- Rare variant may occur bilaterally on mandibular alveolar ridge of neonates

Diagnosis
- Biopsy
- Lymphangiography

Differential Diagnosis
- Neurofibroma (deep)
- Hemihypertrophy syndromes

Treatment
- Excision
- If large lesions are stable, observation
- Sclerotherapy

Prognosis
- Variable, depending upon depth and extent of lesion
- Cavernous variant has guarded prognosis
Verrucal-Papillary Lesions
Papillary Hyperplasia (Palatal Papillomatosis)

Etiology
• Generally attributed to ill-fitting maxillary denture
• Often associated with 24 h/d denture wearing
• *Candida albicans* overgrowth common
• May be noted in habitual mouth breathers (nondenture wearers)

Clinical Presentation
• Erythematous palatal vault beneath denture
• Nodular papillary excrescences
• Generally asymptomatic

Diagnosis
• Clinical appearance
• Biopsy results show fibrous and epithelial papillary hyperplasia; may note pseudoepitheliomatous hyperplasia

Differential Diagnosis
• Contact stomatitis
• Chronic candidiasis
• Denture stomatitis

Treatment
• Establishment of good oral hygiene
• Possible antifungal therapy
• Surgical removal of affected mucosa, if excessive tissue hyperplasia
• Relining/remaking of denture

Prognosis
• Excellent
Pyostomatitis Vegetans

Etiology
• A pustular eruption usually associated with inflammatory bowel disease and skin disease
• Liver dysfunction (sclerosing cholangitis) may be associated in some cases.

Clinical Presentation
• Mucosal pustules, erythema, edema
• Erosions and ulcers may form with serpiginous outlines (“snail tracks”).
• Folds of nodular to hyperplastic tissue (“cobblestoning”)

Microscopic Findings
• Neutrophilic and eosinophilic infiltrate into epithelium producing microabscesses
• Infiltration between epithelial clefts
• Epithelial hyperplasia

Diagnosis
• Correlation with underlying gastrointestinal disease, such as the following:
  • Ulcerative colitis
  • Crohn’s disease
  • Sclerosing cholangitis
  • Malabsorption syndrome

Differential Diagnosis
• Oral Crohn’s disease
• Pseudomembranous (acute) candidiasis
• Melkersson-Rosenthal syndrome
• Orofacial granulomatosis
• Acanthosis nigricans

Treatment
• Successful management of underlying gastrointestinal disease
• Local anti-inflammatory agents
• Dapsone or sulfapyridine systemically
Prognosis

- Correlates with that of systemic disease
Squamous Papilloma

Etiology
- A benign epithelial proliferation
- HPV is found in most cases; several subtypes have been identified, especially HPV 6 and 11.

Clinical Presentation
- Exophytic, papillary mass, measuring less than 1 cm
- Usually pedunculated and soft in texture
- White
- Usually solitary; may be multiple
- Favors soft palate; uvula, tongue, gingiva, buccal mucosa may also be involved

Microscopic Findings
- Epithelial hyperplasia with fibrovascular cores
- Papillary projections may be sharp to blunt.
- Epithelium may be dysplastic in some lesions from human immunodeficiency virus–positive patients.

Diagnosis
- Clinical appearance
- Biopsy features

Differential Diagnosis
- Condyloma acuminatum
- Verruca vulgaris
- Focal epithelial hyperplasia
- Verrucous carcinoma

Treatment
- Surgical excision

Prognosis
- Low recurrence rate
Verruca Vulgaris (Oral Warts)

**Etiology**
- Infection of mucosal epithelium by members of the human papillomavirus group—usually HPV 2, 4, 6, or 11

**Clinical Presentation**
- Papular to nodular and exophytic appearance
- Surface texture is cauliflower-like or verruciform in nature
- Perioral skin lesions may be brownish.
- Oral mucosal lesions are usually white to pink.
- May be pedunculated or broad based
- Intraoral sites of predilection include the lips, palate, and attached gingiva.
- Multiple oral lesions may be evident in immunocompromised patients.

**Microscopic Findings**
- Surface hyperkeratosis
- Granulosis
- Koilocytosis
- Acquired immunodeficiency syndrome–associated oral warts may appear dysplastic microscopically.

**Diagnosis**
- Clinical appearance
- Microscopic findings
- Immunohistochemical demonstration of HPV common antigen

**Differential Diagnosis**
- Focal epithelial hyperplasia
- Keratoacanthoma
- Papillary squamous carcinoma
- Squamous papilloma
- Condyloma acuminatum
Verrucal-Papillary Lesions

Treatment
- Excision
  - Laser surgery
  - Cryosurgery
  - Electrosurgery

Prognosis
- Excellent in immunocompetent host
- Recurrence not uncommon
Verrucous Carcinoma

Etiology
• A well-differentiated, exophytic and endophytic squamous cell carcinoma often associated with tobacco use, especially smokeless tobacco
• A primary or ancillary role for HPV is suspected.
• May be preceded by keratotic patch (see “Verrucous Hyperplasia” on page 158)

Clinical Presentation
• One-half of cases involve the buccal mucosa.
• Attached gingiva is involved in one-third of cases.
• Early, superficial lesions often are interpreted as verrucous hyperplasia; lesions become exophytic, irregular, and indurated.
• Advancing lesions push into adjacent tissues.
• Late lesions invade the periosteum and destroy bone.
• Metastases are rare.

Microscopic Findings
• Well-differentiated, blunt masses of epithelium extending into submucosa
• Intense lymphocytic infiltrate adjacent to invasive front

Diagnosis
• Microscopic findings
• Full-thickness specimen is necessary to establish diagnosis

Differential Diagnosis
• Verrucous hyperplasia
• Papillary squamous cell carcinoma
• Proliferative verrucous leukoplakia

Treatment
• Wide excision
• Radiation therapy may be effective.
• Dedifferentiation may occur spontaneously or after radiation therapy.
Prognosis

- Excellent
- Local recurrence is a distinct possibility.
Verrucous Hyperplasia

Etiology
- Unknown; tobacco (smokeless) associated most commonly
- Role of HPV is unclear.
- A possible precursor to verrucous carcinoma

Clinical Presentation
- Exophytic, papillary, keratotic fronds of epithelium
- May be part of the proliferative verrucous leukoplakia spectrum

Microscopic Findings
- Papillary to verruciform surface projections
- Keratin varies in thickness
- Broad, bosselated epithelial ridges
- Well-differentiated cellular features
- Some similarity to early verrucous carcinoma

Diagnosis
- Microscopic features

Differential Diagnosis
- Verrucous carcinoma
- Papillary squamous cell carcinoma
- Proliferative verrucous leukoplakia

Treatment
- Excision or ablation (eg, laser, electrocautery)
- Continued observation

Prognosis
- Good with complete excision
- Recurrence is common.
Verrucal-Papillary Lesions
Connective Tissue Lesions

Cementoblastoma

Etiology
• An uncommon, benign tumor of mesenchymal odontogenic origin
• Unknown stimulus

Clinical Presentation
• Usually affects patients under the age of 30 years
• Mandibular molar-premolar region is most common site
• Jaw expansion, pain, or tenderness
• Radiopacity with peripheral radiolucent halo
• Mass fused to root of affected tooth

Radiographic Findings
• Focal radioactivity
• Obscuration of root apex
• Thin radiolucent rim

Diagnosis
• Radiographic findings
• History of pain/tenderness
• Histologically: abundant number of cementoblasts are associated with an irregular network of hard tissue (cementum)
• Microscopically similar to osteoid osteoma

Differential Diagnosis
• Focal osseous dysplasia
• Ossifying fibroma
• Osteoma

Treatment
• Enucleation (with associated tooth)

Prognosis
• Excellent
Cheilitis Glandularis

Etiology

• Unknown; may be familial in some cases
• Alternative etiologies include infectious, actinic, atopic, and factitious origins

Clinical Presentation

• Usually involves lower lip of adult males (may occasionally involve both lips)
• Tender eversion and enlargement of lip
• Mucoid to purulent secretion at minor salivary gland orifices

Diagnosis

• Microscopy shows nonspecific inflammation.
• Biopsy results may show labial salivary gland hyperplasia and ductal ectasia.
• Sialadenitis

Differential Diagnosis

• Granulomatous cheilitis (orofacial granulomatosis)
• Atopic cheilitis
• Actinic or photosensitivity cheilitis

Treatment

• Surgical excision
• Suppressive therapy with broad-spectrum antibiotics
• Intraliesional corticosteroid injections

Prognosis

• Chronic
• Good
Connective Tissue Lesions
Fibroma: Traumatic

Etiology
- Chronic low-grade trauma/irritation
- A reactive (hyperplastic), rather than neoplastic, process

Clinical Presentation
- Firm
- Same as or more pale than surrounding tissue
- Pedunculated or broadly based (sessile) fibrous mass with a smooth, elevated surface
- Asymptomatic and slow growing
- May be secondarily ulcerated or keratotic
- Typically in regions accessible to trauma: buccal mucosa, lateral tongue margin, lower lip

Diagnosis
- Appearance
- Location

Differential Diagnosis
- Other submucosa/connective soft tissue tumors, as follow:
  - Granular cell tumor
  - Neurofibroma
  - Lipoma
  - Schwannoma
- Salivary tumor
- Metastatic tumor

Treatment
- Conservative local excision

Prognosis
- Excellent
Fibrous Dysplasia

**Etiology**
- Unknown
- A dysplastic process or developmental lesion of bone

**Clinical Presentation**
- An asymptomatic tumor-like mass or deformity of bones
- May be monostotic (80%) or polyostotic (20%)
- Uncommonly, the polyostotic form may occur with endocrine hyperfunction and focal cutaneous pigmentation (McCune-Albright syndrome).
- Slow-growing, painless swelling of affected bone(s)
- Mandibular body most common site
- Facial asymmetry a frequent presenting sign
- Tooth displacement and malocclusion common
- Maxillary jaw lesions may be accompanied by involvement of the zygoma, sphenoid, and, less commonly, the occiput (the craniofacial variant).

**Radiographic Findings**
- Ill-defined, uniformly radiopaque enlargement with “ground-glass” qualities and diffuse, blended margins
- Mandibular lesions may show loculation.
- The craniofacial form may show skull base thickening.
- Intraoral films may show lamina dura obscurity.

**Diagnosis**
- Clinical appearance
- Radiographic qualities
- Biopsy showing typical irregular trabeculae of woven bone (“Chinese characters”) and fibroblastic, vascularized stroma

**Differential Diagnosis**
- Chronic sclerosing osteomyelitis
- Localized Paget’s disease
- Osteosarcoma
- Cemento-osseous dysplasias
Treatment
- Cosmetic recontouring, if necessary
- Observation only, if lesions are minimally developed and stable

Prognosis
- Most stabilize in adult life
- Some relapse noted in up to one-half of surgically recontoured cases
- Malignant transformation rare unless previously irradiated
Fibrous Hyperplasia: Denture-Related (Epulis Fissurata)

Etiology

• Trauma resulting from an ill-fitting denture
• Exuberant fibrous tissue repair secondary to repeated inflammation and trauma

Clinical Presentation

• Found on vestibular mucosa, usually at facial aspect of denture flange
• Rounded folds of broadly based fibrous tissue surrounding the overextended denture flange
• Ulceration often noted at depth of tissue folds
• More common in anterior segment of the jaws
• May occur on hard palate as a polypoid or leaf-like mass

Diagnosis

• Location and presence of a chronically ill-fitting denture

Differential Diagnosis

• Lymphoma
• Soft tissue tumor
• Metastatic tumor

Treatment

• Excision of all tissue
• Relining or reconstruction of new dentures after excision

Prognosis

• No recurrence anticipated with properly fitting denture
Florid Osseous Dysplasia
(Florid Cemento-osseous Dysplasia)

Etiology
- Unknown
- Strong predilection for middle-aged to elderly black females

Clinical Presentation
- Bilateral and symmetric mandible involvement
- Body and posterior segment of mandible chiefly involved
- Usually asymptomatic

Radiographic Findings
- Early stage is mostly a radiolucent process.
- Later stages have multiple, mixed, radiolucent to radiopaque nodularities.
- Purely lucent areas representing simple bone cysts may be seen in conjunction with opacities.

Diagnosis
- Radiographic appearance
- Involved teeth are vital.

Differential Diagnosis
- Chronic, diffusing, sclerosing osteomyelitis
- Fibrous dysplasia
- Osteosarcoma
- Paget’s disease

Treatment
- In uncomplicated, asymptomatic cases, observation only
- If symptomatic, surgical removal of calcified masses with concomitant antibiotic coverage
- Bone saucerization and open packing may hasten progress.

Prognosis
- Good
- When infected, osteomyelitis-related morbidity occurs.
Gardner’s Syndrome

Etiology
• Autosomal-dominant condition
• Association of multiple osteomas, colonic and rectal polyposis, cutaneous and mesenteric fibromas, epidermoid cysts of skin
• Gene is located on chromosome 5q

Clinical Presentation
• Early clinical indicators may be osteomas of jaws and facial bones.
• Colorectal polyps usually develop after osteomas.
• Facial asymmetry
• Multiple impacted teeth and odontomas not uncommon

Radiographic Findings
• Well-defined, sclerotic, opaque masses
• Endosteal or periosteal origin
• Impacted and supplementary teeth are usually noted.
• Multiple odontomas may be noted.

Diagnosis
• Family history
• Multiple jaw bone, facial bone osteomas
• Concomitant polyposis of colon

Differential Diagnosis
• Exostoses of mandible/maxilla

Treatment
• Osteoma treatment is elective/cosmetic.
• Prophylactic colectomy as all patients ultimately develop colon adenocarcinomas
• Genetic counseling

Prognosis
• Relates to colon adenocarcinoma development and behavior
Giant Cell Granuloma

Etiology
- Probably reactive or responsive in nature
- Speculation suggests it may represent a developmental anomaly.

Clinical Presentation
- Bony expansion
- Most cases arise in those less than 30 years of age
- Female predominance
- Near exclusivity in mandible or maxilla; rarely in facial bones
- Occurrence in mandible predominates 3:1 over that in maxilla.
- Usually anterior to molar teeth
- Most cases are nonaggressive, slow growing, and asymptomatic, with no cortical breakthrough or root end resorption.
- Some cases are recurrent and exhibit aggressive behavior with pain, perforation, and rapid enlargement.
- No radiographic or histologic features can be used to separate nonaggressive lesions from aggressive lesions.

Radiographic Findings
- Usually multilocular, occasionally unilocular, radiolucency
- Margins are usually well defined; borders may be scalloped.
- Can displace teeth; less commonly it resorbs tooth roots
- Wide size variation at time of presentation

Diagnosis
- Incisional biopsy
- Primary hyperparathyroidism should be ruled out.

Differential Diagnosis
- Odontogenic lesions
  - Ameloblastoma
  - Odontogenic myxoma
  - Odontogenic keratocyst
- Nonodontogenic lesions
  - Hemangioma
  - Aneurysmal bone cyst
  - Traumatic bone cyst
Connective Tissue Lesions

- Hyperparathyroidism
- Giant cell tumor

**Treatment**
- Thorough curettage
- Marginal resection, if aggressive or recurrent
- Calcitonin may be successful in some cases.
- Intralesional corticosteroid placement in small lesions may be successful.

**Prognosis**
- Aggressive variant has high recurrence rate
- Generally good
Gingival Hyperplasia: Generalized

Etiology
- May be nonspecific and reactive to local factors (plaque, calculus)
- May be related to hormonal changes (pregnancy, puberty)
- May be associated with drug use, including phenytoin, cyclosporine, calcium channel blockers (especially nifedipine)
- A familial form exists
- Some cases may be related to Cowden disease and syndromes such as Zimmerman-Laband, Rutherford’s, Cross, or Murray-Puretic-Drescher.
- May be secondary to leukemic infiltrate

Clinical Presentation
- Bulky enlargement of free and attached gingiva
- Blunted interdental papillae
- Soft and boggy to firm and dense in texture
- Pink to reddish blue

Diagnosis
- Medical history
- Biopsy

Differential Diagnosis
- See “Etiology.”

Treatment
- Identification and elimination of cause, if possible
- Gingivoplasty and improvement of oral hygiene may be indicated in some cases.

Prognosis
- Good
- Often requires repeated excision
Granular Cell Tumor (Granular Cell Myoblastoma)

Etiology
• A benign neoplasm, probably derived from a Schwann cell precursor

Clinical Presentation
• Most common site is the tongue
• Wide age range
• Nontender, asymptomatic submucosal mass, covered with intact epithelium
• Usually dome-shaped, sessile mass; rarely pedunculated

Diagnosis
• Biopsy

Differential Diagnosis
• Traumatic fibroma
• Salivary gland tumor
• Neurofibroma
• Other soft tissue neoplasm (eg, rhabdomyoma, lipoma)

Treatment
• Excision

Prognosis
• Recurrence is rare.
• Rarely multiple tumors
Leukemia

Etiology
- Unknown
- Probably multifactorial: genomic instability including genetic predisposition, syndromic association, prior chemotherapy, environmental factors

Clinical Presentation
- Oral expression most common with myelomonocytic and myelocytic forms, followed by lymphocytic form
- Petechiae, ecchymosis
- Spontaneous gingival hemorrhage
- Mucosal ulceration (neutropenic ulcers)
- Cervical lymphadenopathy
- Loose teeth (due to infiltration of periodontal ligament)

Radiographic Findings
- Osteolytic lesions in up to one-half of childhood cases
- Expanded, coarse marrow spaces and trabeculae
- Alveolar bone destruction
- Loss of lamina dura and border of developmental dental crypts
- Periosteal reaction with “onion skin” effect

Diagnosis
- Peripheral blood analysis
- Bone marrow biopsy analysis

Differential Diagnosis
- Medication-induced gingival hyperplasia
- Idiopathic thrombocytopenia
- Lymphoma

Treatment
- Chemotherapy regimen(s)—these vary with form of disease
- Bone marrow transplantation

Prognosis
- Varies with form of disease and response to treatment
Lingual Bone Defect (Stafne Bone Cyst; Static Bone Cyst)

Etiology
• Developmental depression of the lingual side of the mandible
• The aberrant lobe of the submandibular salivary gland and/or adipose tissue fills the body of mandible defect. The depression created produces characteristic radiographic findings.

Clinical Presentation
• No symptoms
• Discovered incidentally

Radiographic Findings
• Round to ovoid radiolucency below inferior alveolar canal, above inferior border, and below third molar area
• Well defined by a dense hypercorticated margin
• Size range of 1 to 3 cm
• Rarely noted in premolar and canine areas

Diagnosis
• Radiographic appearance

Treatment
• None; recognition only

Prognosis
• Excellent
Connective Tissue Lesions
Lingual Thyroid

Etiology
• Failure of thyroid primordium to descend from foramen cecum into anterior neck

Clinical Presentation
• Midline mass at foramen cecum area
• Dark, well vascularized
• May interfere with swallowing and breathing in infancy
• Bleeding may occur.

Diagnosis
• Clinical appearance, location
• Demonstration of activity with radionuclide scan (technetium 99m)
• Confirmation of presence of cervical thyroid

Differential Diagnosis
• Thyroglossal duct cyst
• Squamous cell carcinoma
• Lymphoma

Treatment
• Removal if functional thyroid is present in usual location
• Move/transplant to alternative site if other thyroid tissue does not exist

Prognosis
• Excellent
Connective Tissue Lesions

[Image of a mouth with visible connective tissue lesions]
Lipoma

Etiology
• A benign neoplasm of adipose cells
• Uncommon in the oral cavity

Clinical Presentation
• Asymptomatic, slow-growing; usually circumscribed, sessile, or pedunculated
• Soft and compressive with doughy consistency
• Most common sites include buccal mucosa, tongue, floor of mouth
• May be deep seated with no color alteration
• Yellowish, lobulated quality when superficially located
• Surface of larger lesions often is covered by telangiectatic vessels.

Diagnosis
• Mature fat cells in lobular pattern
• Usually well circumscribed by thin fibrous capsule
• Several microscopic variations including infiltrating, pleomorphic, angioid, myxoid, and spindle cell types

Differential Diagnosis
• Other soft tissue tumor
• Minor salivary gland neoplasm
• Metastatic disease

Treatment
• Excision

Prognosis
• Recurrence rare with exception of infiltrating and intramuscular types
Macroglossia

Etiology
• Macroglossia is a clinical sign caused by one of the following many conditions:
  • Angioedema/allergic reaction
  • Infection/abscess formation
  • Inflammation related to trauma
  • Granulomatous disease (sarcoidosis, tuberculosis, deep fungal infections)
  • Congenital disease (muscle hypertrophy or lymphangioma)
  • Metabolic alteration (amyloidosis)
  • Endocrine-related condition (acromegaly)
  • Neoplasia/hamartomatous condition (neurofibromatosis)

Clinical Presentation
• Usually diffuse enlargement; infectious forms may be asymmetric and focal
• Transient forms often arise quickly.
• Persistent forms evolve slowly, may cause splaying of teeth and scalloping of tongue borders, and may result in functional problems (speaking, swallowing, deglutition)

Diagnosis
• Biopsy

Differential Diagnosis
• See “Etiology.”

Treatment
• Management is related to etiology and extent of involvement
• Surgical reduction in certain cases

Prognosis
• Related to etiology; from excellent to fair
Masseteric Hypertrophy

Etiology
• Usually secondary to hyperfunction (habitual)
• May be related to dystrophic or metabolic disease of muscle
• May be idiopathic

Clinical Presentation
• Usually bilateral masseter muscle enlargement
• Painless, symmetric, evenly contoured
• Emphasized when muscle is contracted (ie, jaw is clenched)
• Responds to functional or hyperfunctional demands

Diagnosis
• History
• Muscle biopsy if metabolic disease suspected

Differential Diagnosis
• Sialoadenosis
• Parotid gland enlargement
• Bacterial infection
• Viral infection (mumps, ? cytomegalovirus)
• Autoimmune condition (Sjögren’s syndrome)
• Neoplastic infiltration

Treatment
• Dependent upon etiology
• Usually relates to defining cause and includes the following:
  • Observation
  • Management of underlying cause when appropriate

Prognosis
• Excellent
Melkersson-Rosenthal Syndrome

**Etiology**
- A chronic, idiopathic condition
- Part of orofacial granulomatosis spectrum that includes Crohn’s disease, sarcoidosis, cheilitis granulomatosa

**Clinical Presentation**
- Classic triad
  - Chronic intermittent swelling of lip(s) or oral tissues
  - Fissured tongue
  - Facial nerve palsy (20%)
- Occasionally manifests as painless labial swelling (granulomatous cheilitis/Miescher’s granuloma)
- May be seen in association with intestinal Crohn’s disease
- Lobulated, thickened mucosa of cheeks and tongue may be seen.
- Gingival surface granularity may be present uncommonly.
- “Correctable” causes must be excluded before an idiopathic cause is accepted.

**Diagnosis**
- Biopsy
- Key feature is noncaseating epithelioid granulomas with multinucleated giant cells
- Patch testing for contact allergy

**Differential Diagnosis**
- Angioedema
- Sarcoidosis
- Cellulitis/erysipelas of lip

**Treatment**
- If necessary, intralesional corticosteroid injections
- Tapering dose of systemic corticosteroids
- Clofazimine in slowly tapering dosage
- Dapsone and other nonsteroidal anti-inflammatory drugs

**Prognosis**
- Generally good
- May be persistent
Mucosal Neuroma

Etiology
- A component of the multiple endocrine neoplasia syndrome type III (MEN III), (also called type 2b)
- Syndrome is related to proliferation of neural crest derivatives, as follows:
  - Medullary carcinoma of thyroid
  - Pheochromocytoma
  - Mucosal neuromas
  - Ganglioneuromatosis of the bowel
- Autosomal-dominant transmission
- Gene is located on chromosome 10

Clinical Presentation of MEN III
- Thickened, prominent lips and frenula
- Conjunctival neuromas, corneal nerves
- Oral mucosal neuromas: tongue, lips, cheeks, commissures
- Marfanoid facies and habitus
- Medullary thyroid carcinoma
- Oral mucosal neuromas may be initial sign of syndrome

Laboratory Findings of MEN III
- Increased serum calcitonin levels
- Increased urinary vanillylmandelic acid

Microscopic Findings of Mucosal Neuroma
- Plexiform bundles of neural tissue
- Axons within bundles

Diagnosis
- Confirmation of neuroma presence
- Demonstration of increased serum calcitonin

Differential Diagnosis
- Neurofibromatosis

Treatment
- Thyroidectomy
- Follow-up for pheochromocytoma development
- Genetic counseling
Prognosis

- Guarded, with 100% risk of medullary carcinoma
- 50% risk of pheochromocytoma
Neurofibroma

Etiology
- A benign neoplasm of peripheral nerve
- Concomitant proliferation of perineural fibroblasts and Schwann cells
- Multiple lesions suggest neurofibromatosis syndrome.
- Syndromic form associated with autosomal-dominant inheritance pattern due to mutation of \( NF1 \) or \( NF2 \) genes

Clinical Presentation
- Tongue, buccal mucosa, mucobuccal fold most common sites
- Soft tissue findings: discrete nodules or diffuse lobular lesions
- Skin lesions with syndromic forms: café-au-lait macules, (characteristically six or more); uniform pigmentation with smoothly contoured borders

Radiographic Findings (When Intrabony Lesions Are Present)
- “Blunderbuss” expansion of inferior alveolar foramen
- Uniformly expanded alveolar canal in body of mandible

Microscopic Findings
- Usually unencapsulated mass of spindle cells with gently wavy to twisted nuclei
- Stromal background is delicately fibrillar
- Scattered mast cells in lesions

Diagnosis
- Microscopic findings

Differential Diagnosis
- Localized neurofibroma
  - Neuroma
  - Fibroma
- Widespread neurofibroma
  - MEN 2b/III
  - Proteus syndrome
**Treatment**
- When solitary, excision
- When multiple, verification of the diagnosis, and treatment directed toward function and/or esthetics

**Prognosis**
- When isolated, excellent
- When syndrome related, up to 15% risk of malignant transformation to neurogenic sarcoma
Paget’s Disease

Etiology
- Unknown, although several theories exist including the following:
  - Inborn error of connective tissue metabolism
  - Paramyxovirus or slow virus infection
  - Autoimmune-mediated vascular disorder
  - Possible association with alterations involving chromosomes 6 and 18
  - Familial form exists

Clinical Presentation
- Nearly one-fifth of cases involve the mandible and maxilla.
- Maxillary and mandibular involvement is usually bilateral and symmetric.
- Maxilla predominates over mandible by 2:1
- Jaw and skull enlargement common
- Often deep aching pain in affected bone(s)
- Neurologic complications, as follows, in later phases of uncontrolled or advanced cases:
  - Vertigo, headache
  - Auditory/visual disturbances
  - Facial paresis
- Monostotic involvement occurs but rarely
- Dental patients often complain of ill-fitting prostheses or slow separation of teeth.

Radiographic Findings
- Initially ill-defined, often multiple lytic lesions noted
- Later stage shows patchy radiopaque pattern (like cotton wool)
- Hypercementosis recognizable by “drumstick” appearance of root outline
- Lamina dura–periodontal membrane space may become obliterated.

Laboratory Findings
- Increased serum alkaline phosphatase
- Serum calcium and phosphate normal
- Elevated urinary calcium and hydroxyproline levels
Microscopic Findings
• Early phase predominantly has osteoclastic resorption, fibrous tissue replacement of bone, and prominent blood vessels
• Late phase (sclerotic) has predominantly osteoblastic function, which results in dense bone with numerous reversal lines

Diagnosis
• Radionuclide imaging to determine extent and distribution of lesions

Differential Diagnosis
• Osteosarcoma
• Fibrous dysplasia
• Acromegaly

Treatment
• Bisphosphonate therapy
• Calcitonin
• Pain control

Prognosis and Complications
• Slowly progressive
• Deformities and neurologic complications in late phases
• Malignant transformation may occur (osteosarcoma) in 1% of cases, secondary to loss of heterozygosity in chromosome 18q.
Periapical Cemento-osseous Dysplasia

Etiology
• A dysplastic lesion of periodontal membrane origin with no known cause

Clinical Presentation
• An asymptomatic focus of periapical alteration in the mandibular region; usually anterior
• May involve apices of one or more teeth
• Noted usually in fourth and fifth decades
• Associated teeth are always vital.

Radiographic Findings
• Initially, findings of a periapical radiolucency include the following:
  • Well-defined, noncorticated border
  • Less than 1 cm in diameter
  • Lamina dura usually lost
  • With aging, radiodensity increases

Diagnosis
• Radiographic features
• Associated tooth (teeth) is vital

Differential Diagnosis
• Multiple periapical abcesses
• Florid osseous dysplasia

Treatment
• None; observation only

Prognosis
• Excellent
Connective Tissue Lesions
Scleroderma

Etiology
• Autoimmune, with local and systemic or multiorgan effects secondary to excessive collagen deposition
• Localized sclerosis (morphea) is a distinct disorder sharing only histologic features with systemic sclerosis.

Clinical Presentation
• Oral findings in progressive systemic sclerosis are as follows:
  • Limited oral opening (microstomia)
  • Narrowing of lips (“purse string” sign)
  • Raynaud’s phenomenon (an early finding)
  • Facial skin becomes taut and mask-like.
  • Tongue becomes “bound down” and hypomobile.
  • Telangiectases over facial skin, lips, tongue

Radiographic Findings
• Prominent antegonial notch on panoramic radiograph
• Variable resorption of condyles and coronoid processes
• Uniform widening of periodontal membrane space
• Root resorption

Diagnosis
• Clinical features
• Microscopic features of skin or mucosal biopsy
• Serology: demonstration of anticentromere antibodies or antitopoiso merase I (anti-Scl 70)

Differential Diagnosis
• Lichen sclerosus
• Submucous fibrosis
• Postradiation scarring

Treatment
• Systemic corticosteroids
• Immunosuppressive agents
• Vasodilators
- Systemic d-penicillamine
- Control of local effects of disease

**Prognosis**
- Dismal for systemic form
Torus: Palatal and Mandibular

Etiology
• Focal cortical bone overgrowth of unknown cause

Clinical Presentation
• Palatal: slow-growing, asymptomatic, paramedian, nodular bony mass
• Mandibular: bilateral, smooth and lobular bony masses along lingual surface in cuspid-premolar area
• Larger lesions may interfere with function or dental prosthesis.
• Larger lesions may show surface ulceration and may lead to focal osteomyelitis.

Microscopic Findings
• Dense hyperplastic cortical bone with few marrow spaces

Diagnosis
• Clinical presentation is usually diagnostic.
• Microscopic findings

Treatment
• Observation
• Surgical removal under the following conditions:
  • If there is interference with seating of prosthesis
  • If condition is excessive or symptomatic

Prognosis
• Excellent
Salivary Gland Diseases

Mucocele

Etiology
• Extravasation type
  • Physical-traumatic injury to minor gland excretory duct
  • Mucus extravasation into periductal soft tissue produces a local inflammatory response and granulation tissue “encapsulation.”
• Variant
  • Superficial mucocele
  • Mucus pool at epithelial–connective tissue junction
  • Possibly trauma or systemic (hormonal) etiology

Clinical Presentation
• Lower lip most common site; also buccal mucosa, anterior ventral tongue
• Painless bluish hue when mucin is near surface
• Often waxes and wanes in size

Microscopic Findings
• Mucus pool surrounded by granulation tissue
• Macrophage and neutrophil response to free mucin
• Focal chronic sialadenitis

Diagnosis
• Presentation
• Microscopic findings

Differential Diagnosis
• Hemangioma/varix
• Pyogenic granuloma
• Salivary neoplasm
• Connective tissue neoplasm
Treatment
• Excision with associated local minor salivary glands

Prognosis
• Occasional recurrence
**Mucus Retention Cyst**

**Etiology**
- Represents dilatation of salivary excretory duct due to obstruction
- Duct obstruction may be due to a mucous plug or sialolith formation

**Clinical Presentation**
- Major or minor salivary glands affected in adulthood
- Asymptomatic, soft mucosal swelling
- Can occur at any intraoral minor salivary gland site, especially upper lip

**Microscopic Findings**
- Thin, dilated, epithelial-lined salivary excretory duct
- Lining is cuboidal to columnar with occasional mucus-producing cells present
- Adjacent salivary gland lobules minimally altered but may show obstructive inflammatory changes

**Diagnosis**
- Microscopic findings

**Differential Diagnosis**
- Extravasational mucocele
- Salivary gland neoplasm, especially mucoepidermoid carcinoma

**Treatment**
- Excision of cyst with adjacent gland(s)

**Prognosis**
- Recurrence is rare.
Necrotizing Sialometaplasia

Etiology
- Local ischemic injury of salivary gland lobules
- May be preceded by trauma or local anesthetic injury, or it may appear spontaneously

Clinical Presentation
- Both major and minor salivary glands can be affected.
- Hard palate most common site, usually unilateral
- Initially a painful to dysesthetic submucosal swelling
- Ultimately, a central necrotic crater develops.
- May extend to and involve deep soft tissue and palatal bone

Microscopic Findings
- Salivary gland inflammation and lobular necrosis (necrosis is not always demonstrable on biopsy)
- Ductal squamous metaplasia (bland cytology)
- Lobular architecture of salivary glands persists

Diagnosis
- Microscopic findings

Differential Diagnosis
- Salivary gland neoplasm
- Squamous cell carcinoma
- Granulomatous disease

Treatment
- Follow-up only

Prognosis
- Excellent
Ranula

Etiology
• Obstruction of the sublingual (usually) or submandibular salivary gland by a sialolith or by trauma
• Secondary to obstruction, extravasation of saliva into the soft tissue of the floor of the mouth

Clinical Presentation
• Unilateral, fluctuant, soft tissue mass on the floor of the mouth
• Usually has a bluish, slightly translucent quality
• When above the mylohyoid muscle, presentation is intraoral.
• If extravasation extends below the mylohyoid muscle, a plunging ranula forms.
• Occlusal radiographs may demonstrate a suspected sialolith.

Diagnosis
• Demonstration of sialolith
• Soft tissue imaging (T2-weighted magnetic resonance image)
• Aspiration of mucinous salivary fluid
• Excised tissue with granulation tissue lining around mucin pool

Differential Diagnosis
• Dermoid cyst
• Salivary gland tumor
• Soft tissue tumor
• Cystic hygroma
• Thymic cyst

Treatment
• Marsupialization as an initial procedure
• Excision of the involved gland (extravasation type)
• Sialolithectomy (in obstructive type)

Prognosis
• No recurrence with sialadenectomy
• Recurrence risk with sialolithectomy secondary to duct scarring or reformation of stone
Sialorrhea (Sialosis)

Etiology
• Varied; may include idiopathic paroxysmal sialorrhea, parkinsonism, stomatitis (acute), newly inserted oral appliances, expectorants, neostigmine, and others

Clinical Presentation
• Excess saliva resulting in drooling
• Angular cheilosis
• Diffuse parotid/submandibular salivary gland enlargement

Diagnosis
• Direct observation and analysis of history
• Flow-rate measurement

Differential Diagnosis
• See “Etiology.”

Treatment
• Scopolamine
• If related to medication use, an alternate medication should be chosen, if possible.

Prognosis
• Guarded/indeterminate
Sjögren’s Syndrome

Etiology
• An autoimmune disease resulting in exocrine gland dysfunction secondary to mononuclear cell infiltration
• Increased prevalence of human leukocyte antigen DR/DQ alleles
• Autoantibody production against nuclear antigens SS-A and SS-B
• No specific agent identified; postulations include the following:
  • Potential role for viruses/retroviruses as cofactors
  • Possible role of cytokine and hormonal influence on signal transduction and secretion

Clinical Presentation
• Decrease in exocrine gland function
• Xerostomia
• Xerophthalmia/keratoconjunctivitis sicca
• Salivary and lacrimal gland enlargement (one-third of cases)
• Secondary effects of exocrine dysfunction are as follows:
  • Dental caries
  • Oral candidiasis
  • Ocular/corneal discomfort
• Primary form: exocrine dysfunction dominates
• Secondary form: exocrine dysfunction; other associated autoimmune conditions—usually rheumatoid arthritis, less often lupus erythematosus

Diagnosis
• Demonstration of objective xerostomia and xerophthalmia
• Serologic demonstration of associated SS-A or SS-B antibodies
• Correlation of clinical and serologic findings with labial salivary gland biopsy; demonstration of presence of periductal lymphocytic sialadenitis

Differential Diagnosis (Xerostomia/Parotid Gland Swelling)
• Sarcoidosis
• Human immunodeficiency virus–associated exocrinopathy
• Drug side effects
• Lymphoma
• Depression
• Autonomic neuropathy
• Graft-versus-host disease
• Bulimia
• Alcoholism
• Diabetes mellitus
**Treatment**

- Directed at associated connective tissue or autoimmune disease
- Systemic corticosteroids if acute symptoms arise
- Usually symptomatic and preventative therapies are used, including the following:
  - Reduction of oral dryness
  - Pilocarpine
  - Cemiveline
  - Oral moisturizing agents (saliva substitutes)
  - Gustatory stimulation
  - Ocular moisture replacement
  - Saline
  - Synthetic glycoprotein solutions
  - Carboxymethylcellulose sodium
  - Ocular punctual occlusion
  - Frequent dental/ophthalmic examinations

**Prognosis**

- Guarded
- High risk of lymphoma compared with risk in those without autoimmune disease
Lymphoid Lesions

Burkitt’s Lymphoma

Etiology
- B lymphocyte malignancy associated with genetic mutations: C-MYC, P53, and others
- Causative association with Epstein-Barr (EB) virus, and malaria cofactor believed to increase the risk for gene-translocation accidents
- African form closely associated with EB infection; North American form less strongly associated

Clinical Presentation
- Rapidly progressive facial asymmetry, chiefly of the mandible
- Proptosis in children may occur in association with maxillary lesions.
- Pain and paresthesia associated with jaw lesions
- Children predominately affected
- Facial presentation noted in 25% of North American (nonendemic) cases; nearly 100% in African children (endemic cases)
- Abdominal (retroperitoneal) presentation usually noted initially in nonendemic form and in a wider age range than in endemic form

Radiographic Findings
- Ill-defined radiolucency
- Loss of lamina dura and developmental crypt(s) around unerupted teeth
- Uniform widening of periodontal membrane space
- Teeth may be displaced, causing malocclusion and/or exfoliation.

Diagnosis
- A diffuse proliferation of small noncleaved lymphoid cells (B lymphocyte–derived cells)
- Tumor cells have round nuclei and prominent nucleoli.
- Scattered macrophages with abundant pale cytoplasm contain-
ing pyknotic cellular debris represent the “stars” in the so-called starry sky appearance.

**Differential Diagnosis: Clinical**
- Other jaw malignancies of childhood (Ewing’s sarcoma, osteosarcoma)
- Acute infection

**Differential Diagnosis: Microscopic**
- Other round cell malignancies of childhood (neuroblastoma, leukemia, embryonal rhabdomyosarcoma)

**Treatment**
- Multiagent chemotherapy
- Overall cure rate in children is now up to 90% with intensive, high-dose fractionated therapy

**Prognosis**
- Fair
Lymphoepithelial Cyst

Etiology
• Entrapment of oral mucosal epithelium within lymphoid tissue in foliate papillae, floor of the mouth, ventral tongue, soft palate

Clinical Presentation
• Yellow to white nodule
• Asymptomatic, slow growing
• May drain or decompress spontaneously
• Most common in second then fourth decades
• Overlying mucosa intact, smooth

Diagnosis
• Histologic demonstration of lymphoid tissue with germinal centers surrounding true cyst lumen filled with epithelial debris

Differential Diagnosis
• Lipoma
• Minor salivary gland neoplasm or sialolith
• Mucocele

Treatment
• Excision

Prognosis
• Excellent
Lymphoma

Etiology
- Idiopathic
- Long-term immunosuppression
- ?Post radiation

Clinical Presentation
- Relatively common in head and neck region
- Most common in middle-aged and older individuals
- Mass of reddish blue tissue with ulceration, pain
- Paresthesia of lip when occurring in mandible
- Initial presentation in oral cavity is uncommon (2%)
- Predominant oral sites: palate, gingiva, buccal mucosa, mandible
- May arise within lymph nodes or extranodally in soft tissue
- Ill-defined radiolucency in bone
- Hodgkin’s lymphoma rare in oral cavity
- Burkitt’s lymphoma arises in children.

Microscopic Findings
- Non-Hodgkin’s lymphoma predominates; almost always B cell type
- Varied, depending upon type/subtype: cell size, pattern, maturation level
- Classification schemes dependent upon microscopic features
- Human immunodeficiency virus–associated lymphomas are typically diffuse, large cell, high-grade lymphomas.
  - EB virus is often evident in tumor cells.
- Revised European American Lymphoma Classification and Working Formulation are current microscopic classifications

Diagnosis
- Biopsy, immunohistochemical studies
- Flow cytometry
- Staging work-up involves the following:
  - Bone marrow biopsy
  - Whole body computed tomography scan

Differential Diagnosis
- Salivary neoplasm
- Metastatic tumor
• Soft tissue tumor (primary)
• Leukemia

**Treatment**
• Dependent upon extent/clinical stage and microscopic features
• For localized (stage I) disease: radiation therapy
• Chemotherapy or combined chemotherapy-radiation therapy for more widespread disease
• Very-low-grade lesions may be observed only since treatment typically has no effect on outcome.

**Prognosis**
• Dependent upon clinical stage and histologic subtype; acquired immunodeficiency syndrome–associated lymphoma has a poor outcome
• Very-low-grade lesions have excellent prognosis
Myeloma

Etiology
• Neoplastic proliferation of malignant plasma cells
• Monoclonal immunoglobulin (κ or λ light chain) production

Clinical Presentation
• Occurs exclusively in adulthood (males 2:1 over females)
• Bone pain and paresthesia
• Mucosal involvement may occur as polypoid to lobular masses.
• Purpura and woody induration of the tongue (macroglossia) or gingiva may be the initial manifestation.
• Solitary presentation invariably becomes multiple myeloma.
• The extramedullary form occasionally becomes multiple myeloma.

Radiographic Findings
• Sharply defined radiolucencies, usually of many bones
• Absence of marginal hyperostosis or opaque lining

Laboratory Findings
• Monoclonal gammopathy by serum electrophoresis
• Bence Jones proteinuria
• Plasma cells in bone marrow aspirate

Microscopic Findings
• Monotonous, diffuse plasma cell proliferation
• Variable levels of differentiation and mitotic activity
• Immunohistochemical demonstration of monoclonality (κ or λ light chains)

Diagnosis
• Biopsy
• Immunohistochemical evaluation

Differential Diagnosis
• Lymphoma
• Primary osseous tumor
• Metastatic tumor
• Traumatic bone cyst
Lymphoid Lesions

**Treatment**
- Chemotherapy
- Local radiation therapy

**Prognosis**
- Poor
Cysts

Aneurysmal Bone Cyst

Etiology
- Unknown
- Possibly represents a vascular response/repair to jaw injury (an arteriovenous malformation)
- Three phases: incipient, destructive, stabilization

Clinical Presentation
- Bony expansion and occasionally mild pain
- Chiefly occurs in mandible
- Female predilection

Radiographic Findings
- Expansile, multiloculated, destructive bony lesion
- Surrounding bone may be sclerotic
- Angiogram demonstrates intense vascularity

Diagnosis
- Radiographic lytic lesion
- Honeycombed quality of large vascular sinusoidal spaces and bony septa
- May be confused microscopically with central giant cell granuloma

Differential Diagnosis
- Ameloblastoma
- Odontogenic keratocyst
- Odontogenic myxoma
- Hemangioma
- Giant cell granuloma

Treatment
- Excision to en bloc resection

Prognosis
- Good to excellent
Calcifying Odontogenic Cyst

Etiology
- An odontogenic cyst with characteristic microscopic pattern
- May be noted in association with other odontogenic tumors
- Origin is residual odontogenic epithelium in the jaws; stimulus unknown

Clinical Presentation
- Usually a unilocular, well-defined radiolucency, chiefly of maxilla
- Scattered opacities seen in up to 50% of cases
- May be associated with the crown of an unerupted tooth
- An extraosseous form may occur (usually anterior to first molar)
- May be more solid than cystic (odontogenic ghost cell tumor)

Radiographic Findings
- Well-defined radiolucency or lucency with opaque foci (dystrophic calcification of keratin produced by lining epithelium)
- Tooth displacement or root resorption may be seen.

Diagnosis
- Stratified squamous lining with prominent basal layer
- Budding or extension of epithelium into the cyst wall may be noted.
- Characteristic ghost cell keratinization required for diagnosis
- Ghost cells may undergo dystrophic calcification.
- Foreign body reaction may occur when ghost cells come in contact with connective tissue.
- The solid or tumorous form shares microscopic features with ameloblastoma.

Differential Diagnosis: Radiographic
- Calcifying epithelial odontogenic tumor
- Ossifying fibroma
- Ameloblastic fibro-odontoma
Treatment
- Enucleation/excision
- If noted in association with another odontogenic tumor, consideration must be given to the behavior of the accompanying lesion.

Prognosis
- Some recurrence potential, especially in association with solid lesions
- Overall prognosis is very good
- Excellent prognosis for peripheral (gingival) lesions
Dental Lamina Cyst (Bohn’s Nodules; Gingival Cyst of Newborn)

Etiology
• Cystic degeneration of residual dental lamina/odontogenic epithelium
• Found in over 80% of newborns

Clinical Presentation
• Small (1–2 mm), usually multiple, yellow-white nodules over the alveolar crest in neonates
• Usually involute following spontaneous cyst rupture

Diagnosis
• Appearance and location
• Histologically, parakeratinized epithelial lining with keratin-filled cyst cavity is noted.

Differential Diagnosis
• Eruption cyst

Treatment
• None; observation only

Prognosis
• Excellent
Cysts
Dentigerous Cyst

Etiology
- A developmental odontogenic cyst arising subsequent to separation between dental follicle and the crown of an associated unerupted tooth
- Proliferation of reduced enamel epithelium lining the follicle, with fluid accumulation between epithelium and impacted tooth crown
- Alternatively, degeneration of the stellate reticulum component of the enamel organ occurs during odontogenesis.

Clinical Presentation
- Most commonly involves frequently impacted teeth: mandibular third molars, followed by maxillary canines
- Usually noted during second and third decades
- Asymptomatic; discovered on routine radiographic examination
- Painless jaw/alveolar expansion may occur; cortex is thinned and rarely perforated

Radiographic Findings
- Well-defined radiolucency enclosing crown of unerupted tooth
- Corticated/opaque margins unless infected
- May produce root resorption of adjacent erupted teeth
- Usually unilocular; less commonly multilocular

Diagnosis: Microscopic
- Cysts without secondary inflammation
  - Thin, cuboidal, nonkeratinized epithelial lining two cell layers thick with flat epithelial–connective tissue interface
  - Loosely arranged collagen bundles, occasionally containing scattered odontogenic epithelial rests
- Cysts with secondary inflammation
  - Hyperplastic, nonkeratinized squamous epithelial lining with epithelial ridge development
  - Variable chronic inflammatory cell infiltrate within condensed collagen stroma
**Differential Diagnosis: Radiographic**
- Odontogenic keratocyst
- Ameloblastoma

**Treatment**
- Cyst enucleation and extraction of associated tooth
- Marsupialization prior to excision may be considered if the cyst is very large.

**Prognosis**
- Excellent
- Possible complications
  - Pathologic fracture with large lesions
  - Neoplastic transformation of epithelial lining (ameloblastoma and, rarely, squamous cell carcinoma)
Dermoid Cyst

Etiology
• Cystic degeneration of entrapped epithelium within the midline fusion zone between the first and second branchial arches
• Alternative etiology relates to in utero traumatic epithelial implantation into floor of mouth area

Clinical Presentation
• Slowly enlarging, usually asymptomatic, sublingual or floor-of-mouth mass
• May present as a soft and compressible paramedian swelling or deformity
• Overlying mucosa/skin is thinned, but is otherwise unremarkable
• May have doughy consistency because of sebum and/or keratin in cystic cavity

Microscopic Findings
• Epithelial lining (stratified squamous)
• Cyst contents may include keratin debris, hair follicles/hair, and sebaceous and sweat glands.
• Rarely find gastric mucosal characteristics present in cyst lining

Diagnosis
• Aspiration may yield cellular debris, sebum, keratin, mucus
• Histologic demonstration of hair follicles, sebaceous glands, keratinizing cystic lining

Differential Diagnosis
• Cellulitis of odontogenic origin
• Sublingual sialadenitis
• Ranula (superficial or deep/plunging)

Treatment
• Intraoral surgical excision

Prognosis
• Excellent
Cysts
Eruption Cyst

Etiology
• Soft tissue cyst of attached gingiva secondary to fluid accumulation within the follicular space of an unerupted tooth

Clinical Presentation
• Gingival swelling on the alveolar crest
• Usually soft, translucent to bluish (“eruption hematoma”)

Diagnosis
• Location
• Radiographic demonstration of erupting tooth

Differential Diagnosis
• Gingival cyst

Treatment
• Usually none is necessary as tooth typically erupts through lesion
• Possibly unroof cyst to facilitate eruption

Prognosis
• Excellent
Glandular Odontogenic Cyst

Etiology
• A developmental odontogenic cyst
• A unique jaw cyst with microscopic evidence of glandular differentiation

Clinical Presentation
• Slow growing; may be expansile
• Located chiefly in the anterior mandible
• May present a lateral periodontal relationship

Radiographic Findings
• Usually a multilocular cystic radiolucency
• Sharply defined with hyperostotic margins
• May be extensive, locally invasive; may perforate cortical bone

Diagnosis
• Radiographic qualities
• Incisional biopsy results show cystic epithelium with mucous cells and pseudoduct formation

Differential Diagnosis
• Giant cell lesion
• Ameloblastoma
• Odontogenic keratocyst
• Lateral periodontal cyst

Treatment
• Excision, peripheral ostectomy
• En bloc excision
• Primary reconstruction

Prognosis
• Recurrence may be associated with conservative management.
Lateral Periodontal Cyst

Etiology
- Stimulus unknown
- Dental lamina remnant proliferation within the alveolar segment of the jaw, separate from the periodontal ligament

Clinical Presentation
- Asymptomatic
- Usually occurs in fourth decade and beyond
- Usually in mandibular canine/premolar region (65%)
- In the maxilla, the lateral incisor area predominates.

Radiographic Findings
- Well delineated, round to ovoid lucency with thin, opaque (corticated) margin
- Located lateral to vital tooth roots
- Usually unilocular; may be multilocular (botryoid odontogenic cyst)

Diagnosis
- Thin, nonkeratinized epithelial lining
- Nodular epithelial thickening along cyst lining
- Lining cells are cuboidal with interspersed clear glycogen-filled cells.

Differential Diagnosis
- Inflammatory, lateral radicular cyst
- Primordial cyst/odontogenic keratocyst
- Odontogenic tumor
- Glandular odontogenic cyst

Treatment
- Conservative enucleation
- The botryoid variant requires more aggressive curettage.

Prognosis
- Recurrence uncommon
- Increased risk of recurrence with botryoid variant; longer-term follow-up necessary
Nasopalatine Duct Cyst

Etiology
• Developmental, nonodontogenic cyst
• Cystic degeneration of epithelial remnants of the vestigial nasopalatine duct

Clinical Presentation
• Usually develops in adulthood
• May be incidental on routine dental radiographs
• Palatal mass with tenderness and drainage
• Adjacent teeth are vital.

Radiographic Findings
• Well-defined, median-paramedian radiolucency in anterior maxillary midline, greater than 5 to 6 mm in diameter
• Border usually sclerotic
• Round, ovoid, or heart shaped

Diagnosis
• Radiographic features
• Biopsy confirmation

Differential Diagnosis
• Apical/radicular cyst
• Other odontogenic cysts
• Odontogenic tumor

Treatment
• Enucleation

Prognosis
• Rarely recurs
Nevoid Basal Cell Carcinoma Syndrome

Etiology
• Autosomal-dominant condition
• Loss of heterozygosity at chromosome 9q22.3
• Mutation of PTCH tumor suppressor gene

Clinical Presentation
• Multiple jaw cysts (odontogenic keratocysts)
• Numerous cutaneous basal cell carcinomas, which arise early in life and are independent of sun exposure
• Bifid ribs
• Calcification of falx cerebri
• Ocular hypertelorism
• Mandibular prognathism
• Broad nasal bridge
• Medulloblastoma
• Palmar and plantar pits

Radiographic Findings
• Multiple jaw radiolucencies
• Lamellar calcification of falx cerebri
• Bifid rib on abdominal radiograph

Diagnosis
• Radiographic and clinical findings

Differential Diagnosis
• Other syndromes, such as the following:
  • Charcot-Marie syndrome
  • Waardenburg’s syndrome

Treatment
• Excision of basal cell carcinomas and odontogenic keratocysts
• Excision of other related aggressive tumors at other sites
• Genetic counseling

Prognosis
• Guarded
Odontogenic Keratocyst

Etiology
• A benign, aggressive developmental odontogenic cyst; may be associated with mutation of PTCH tumor suppressor gene

Clinical Presentation
• 5 to 15% of odontogenic cysts
• Usually occurs sporadically as an isolated finding
• Approximately 5% are associated with nevoid basal cell carcinoma.
• 5% of patients have multiple odontogenic keratocysts (OKCs) and no syndrome

Radiographic Findings
• Can occur in any area of maxilla or mandible
• Rarely may arise in gingival soft tissue only (peripheral)
• Mandible is preferred site in 65 to 78% of cases
• Often (40%) seen in a dentigerous relationship
• Discrete radiolucency, usually in relation to teeth (apical, lateral radicular, pericoronal to impacted tooth)
• May be unilocular to multilocular

Microscopic Findings
• Thin, parakeratinized epithelial lining (6–10 cells thick)
• Wavy, corrugated surface configuration
• Prominent, palisaded, cuboidal to low-columnar basal cell layer
• Basal layer “budding” into fibrous stroma is seen occasionally
• Satellite or daughter cyst formation noted frequently

Diagnosis
• Radiographic features
• Microscopic findings

Differential Diagnosis
• Odontogenic cysts: dentigerous, radicular, lateral periodontal, or glandular odontogenic
• Nonodontogenic cyst: nasopalatine duct
• Odontogenic tumors: ameloblastoma, myxoma
• Giant cell granuloma
• Central mucoepidermoid carcinoma

**Treatment**
• Excision with curettage of bony confines

**Prognosis**
• The recurrence rate varies from 10 to 30% (solitary OKCs).
• Recurrence rates are greatest in patients with a syndrome.
Primordial Cyst

Etiology
• A developmental odontogenic cyst arising from cystic degeneration of the enamel organ prior to formation of hard tissue

Clinical Presentation
• Invariably has the microscopic appearance of odontogenic keratocyst
• Rare
• Radiolucent lesion of jaw
• Occurs in place of a tooth, usually a third molar

Radiographic Findings
• A well-defined radiolucency
• Most commonly in the posterior mandibular quadrants

Diagnosis
• Radiograph shows a cyst instead of a tooth
• Histologically an odontogenic keratocyst

Differential Diagnosis
• Odontogenic tumor
• Other odontogenic cyst
• Central giant cell granuloma

Treatment
• Enucleation with bone curettage

Prognosis
• Significant recurrence rate
• Long-term follow-up mandated
Radicular Cyst

Etiology
• Preceded by periapical granuloma; arises as follows:
  • Secondary to necrosis of dental pulpal tissue
  • Stimulation of epithelial network (Malassez’s rest) at tooth root apex results in cystification
• Cyst growth continues secondary to effects of osmotic gradient across epithelial lining layers, mediators of inflammation, and epithelial proliferation

Clinical Presentation
• Asymptomatic unless there is an acute exacerbation
• Usually a limited process at root apex or lateral to root surface
• Radiograph shows a round and well-defined lucency, usually with a sclerotic margin.
• Generally 1 cm or less across, but can be significant in size
• Root resorption uncommon

Microscopic Findings
• Stratified squamous epithelial lining
• Lumen filled with cell debris, fluid, cholesterol
• Connective tissue wall with mixed inflammatory infiltrate

Diagnosis
• Documentation of nonvital tooth
• Radiograph shows alteration of apical bone

Differential Diagnosis
• Periapical granuloma
• Central giant cell granuloma
• Odontogenic and nonodontogenic tumors
• Metastatic tumor

Treatment
• Endodontic therapy or
• Periapical surgery and biopsy or
• Tooth extraction and biopsy
Prognosis
• Excellent
• Occasional recurrences
Thyroglossal Duct Cyst

Etiology
• Cystic change associated with thyroglossal duct remnants that failed to involute (tenth week of development)
• Rarely may be hereditary in origin (autosomal dominant or recessive)

Clinical Presentation
• Soft, painless, and slowly enlarging mass in anterior midline of the neck of children and young adults
• Usually unilocular as seen by ultrasound examination
• Mass is usually mobile
• Most occur above the hyoid bone.
• May involve the tongue

Microscopic Findings
• Cyst lining of squamous, transitional, ciliated columnar epithelium composite
• The cyst wall may contain residual thyroid tissue.

Diagnosis
• Ultrasonography
• Clinical presentation of midline neck mass
• Histopathology

Differential Diagnosis
• Base-of-tongue carcinoma
• Base-of-tongue salivary tumor
• Thyroid carcinoma arising within cyst

Treatment
• Surgical excision

Prognosis
• Excellent
• Recurrence owing to tortuous morphology
• Rarely, carcinomatous transformation of duct lining or remnants of thyroid parenchyma are noted.
Traumatic Bone Cyst

Etiology
• Unknown in most cases
• May be due to traumatic injury producing intramedullary hemorrhage and subsequent clot resorption
• Alternative theory suggests degeneration of primary intrabony pathology

Clinical Presentation
• Peaks in second decade
• Usually in body of mandible
• Painless in most cases
• Swelling noted in one-fourth of cases

Radiographic Findings
• Clearly defined radiolucency
• Margins may be uneven but clear.
• May extend between tooth roots creating a scalloped pattern

Diagnosis
• Radiographic appearance
• Clinical finding of an empty bony space (pseudocyst)
• Collagen and fibrin line the dead space (no epithelium).
• Lamellar bone may be noted along the bony margin.

Differential Diagnosis
• Central giant cell granuloma
• Fibro-osseous lesion (early)
• Hemangioma

Treatment
• Surgical exploration
• Observation for resolution

Prognosis
• Excellent
• Small risk of recurrence
Odontogenic Tumors

Adenomatoid Odontogenic Tumor

Etiology
• Derivation from epithelial component of the enamel organ
• Represents less than 10% of odontogenic tumors
• Biologic behavior allows for distinction from ameloblastoma

Clinical Presentation
• Narrow age range, 5 to 30 years, with most cases noted during second decade
• Female predilection
• Anterior jaw location common
• Association with unerupted tooth
• Asymptomatic; occasionally produces expansion of alveolar bone
• Rarely occurs in gingival soft tissue (peripheral)
• May produce root divergence of adjacent teeth

Radiographic Findings
• Well defined, unilocular, often adjacent to crown of unerupted tooth
• Opaque foci may be scattered within the lucency in a “snowflake” or “salt and pepper” pattern.

Microscopic Findings
• Characteristic intraluminal/intracystic growth with well-defined capsule
• Dual cell population: spindle cells and cuboidal to columnar cells forming tubules or pseudoducts
• Foci of dystrophic calcification or eosinophilic droplets may be noted.

Diagnosis
• Radiographic features
• Microscopic findings
Differential Diagnosis
• Dentigerous cyst
• Odontogenic keratocyst
• Calcifying odontogenic cyst
• Lateral root cyst
• Calcifying epithelial odontogenic tumor

Treatment
• Enucleation

Prognosis
• No recurrence
Ameloblastic Fibroma and Ameloblastic Fibro-odontoma

Etiology
- Ameloblastic fibroma: a benign mixed odontogenic tumor with concomitant epithelial and mesenchymal neoplastic proliferation
- Ameloblastic fibro-odontoma: as for ameloblastic fibroma with the addition of an odontoma
- Spontaneous; no known cause for either

Clinical Presentation
- Noted mostly in first and second decades
- Approximately 70% in mandible, usually posterior region
- No gender predilection
- May cause jaw expansion
- Asymptomatic

Radiographic Findings
- Well defined with hyperostotic margin
- Unilocular to multilocular
- Often associated with an unerupted tooth
- Ameloblastic fibro-odontoma has opaque component(s) related to enamel and dentin in the odontoma component

Diagnosis
- Lobulated, cellular mesenchymal component with proliferating odontogenic epithelium in cords and islands
- Enamel matrix, dentin formation associated with odontoma (when present)

Differential Diagnosis
- Ameloblastoma
- Dentigerous cyst
- Odontogenic keratocyst
- Odontogenic myxoma
- Central giant cell granuloma
Treatment
• Conservative surgical excision/curettage

Prognosis
• Excellent
Ameloblastoma

Etiology
- A benign, aggressive jaw tumor of odontogenic epithelial (ectodermal) origin; the most common odontogenic tumor after the odontoma
- Incidence of 0.3 cases per million people

Clinical Presentation
- Peak incidence during third to fifth decades
- 80% occur in the mandible, chiefly in molar and ramus region
- Often presents in association with unerupted third molar teeth
- May produce marked deformity, facial asymmetry
- Extraosseous or peripheral variant arises in gingival tissues of older adults (fifth to seventh decades)
- Typically slow growing, but persistent

Radiographic Findings
- Osteolytic or radiolucent with sclerotic, smooth, even borders
- May be unilocular to multilocular
- Root resorption or tooth displacement may be seen.
- Can expand affected jaw in any plane
- Cortical perforation may occur.

Diagnosis
- Sheets, strands, islands of odontogenic epithelium
- Peripheral layer of cuboidal to columnar ameloblast-like cells enclosing a cell population analogous to stellate reticulum of the enamel organ
- Cystic degeneration common within stellate reticulum component
- Several histologic patterns described have no clinical relevance.
- A biologic variant, cystic (unicystic) ameloblastoma, occurs in younger patients; has a less aggressive clinical course and is managed more conservatively
- Malignant variants rarely seen

Differential Diagnosis
- Dentigerous cyst
- Odontogenic keratocyst
• Odontogenic myxoma
• Central giant cell granuloma

**Treatment**
• Varies with subtype, size, location
• Solid/multicystic lesions generally require local excision or resection.
• The cystic variant requires local excision, as recurrences may follow curettage only

**Prognosis**
• Generally good; recurrence rates higher with conservative treatment
• Recurrence rates of up to 15% following marginal resection
• Very good prognosis for cystic ameloblastoma
• Long-term follow-up necessary
Calcifying Epithelial Odontogenic Tumor

Etiology
- A benign odontogenic tumor of uncertain histogenesis
- Stratum intermedium component of enamel organ is favored cell of origin

Clinical Presentation
- Chiefly in posterior mandible
- Painless, slow growing
- Mean age of occurrence is approximately 40 years
- Occasional soft tissue origin (peripheral) noted as a sessile gingival mass
- Jaw expansion a common clinical presentation

Radiographic Findings
- Usually noted in association with an impacted tooth
- Multilocular; most often with mixed radiolucent and radiopaque features
- Impacted tooth often obscured by tumor-associated calcification
- Margins may be well defined or sclerotic and vague.

Diagnosis
- Radiographic features
- Biopsy findings of polyhedral epithelial cells, nuclear pleomorphism, amyloid material, and concentric calcifications with epithelial islands

Differential Diagnosis
- When radiolucency predominates: dentigerous cyst, odontogenic keratocyst, ameloblastoma, odontogenic myxoma
- With mixed radiolucent and radiopaque features: calcifying odontogenic cyst, adenomatoid odontogenic tumor, ameloblastic fibro-odontoma, fibro-osseous lesion, osteoblastoma

Treatment
- Local, conservative excision including a thin rim of normal bone (so-called ostectomy) versus conservative en bloc removal
- Peripheral lesions with a narrow periphery of normal-appearing mucosa
Prognosis

• Very good
• Recurrence rate is low, from 10 to 15%
• Long-term follow-up recommended
Odontogenic Myxoma

Etiology
• A benign odontogenic tumor
• Unknown origin

Clinical Presentation
• A lesion of adulthood (average occurrence at 30 years)
• Equal male:female and mandible:maxilla occurrences
• Wide age range: second through sixth decades
• Usually asymptomatic
• May produce jaw expansion

Radiographic Findings
• Well-defined, unilocular to multilocular radiolucency
• Loculi range from small “honeycomb” to large “soap bubble” shapes
• Cortical thinning may be present with larger lesions.
• Perforation of the cortex is uncommon.

Microscopic Findings
• Minimal cellularity, myxoid background
• Variable amounts of collagen
• Scattered residual bony trabeculae
• Odontogenic epithelial rests are rarely noted.

Diagnosis
• Radiographic features
• Microscopic findings

Differential Diagnosis
• Other odontogenic tumor: ameloblastoma
• Odontogenic cysts: odontogenic keratocyst, dentigerous cyst, glandular odontogenic cyst
• Central giant cell granuloma

Treatment
• Excision with bony curettage
• Large lesions may require en bloc resection.
Prognosis

- Good
- Can be aggressive rarely
- Recurrences not uncommon, secondary to gelatinous quality and lack of capsule
Odontoma

Etiology
• A hamartomatous or benign mixed odontogenic tumor of the jaw
• Composed of enamel, dentin, cementum, and pulp tissue

Clinical Presentation
• Two forms, as follows:
  • Complex: a randomly arrayed mixture of dental tissues with no gross resemblance to a tooth
  • Compound: multiple, tooth-like structures
• Mean age of occurrence, 12 to 16 years
• Asymptomatic, usually small and discovered incidentally
• Jaw expansion may be present with large lesions.
• Presence may be heralded by an over-retained primary tooth or by alveolar swelling.

Radiographic Findings
• Well-localized, mixed radiolucent and radiopaque lesion
• Within alveolar segment of jaws
• Complex form most commonly noted in mandibular molar area
• Compound form favors anterior jaw region, usually the maxilla; may contain a few small teeth or large numbers of tiny tooth-like structures

Diagnosis
• Radiographic presentation
• Histologic demonstration of dental hard tissues

Differential Diagnosis
• Ameloblastic fibro-odontoma
• Adenomatoid odontogenic tumor
• Calcifying odontogenic cyst
• Focal sclerosing osteitis, osteoma

Treatment
• Conservative excision/curettage

Prognosis
• Excellent
Peripheral Odontogenic Fibroma

Etiology
- A benign proliferation neoplasm of fibroblastic and odontogenic epithelial origin

Clinical Presentation
- Asymptomatic, firm, slow-growing mass of the attached gingiva
- Overlying mucosa unremarkable and intact
- Sessile growth pattern
- Usually along facial or buccal aspect of gingiva
- Calcifications may be present radiographically.
- Underlying alveolar bone is spared.
- Uncommon to rare
- Also seen centrally (within bone)

Diagnosis
- Fibrous to myxoid stromal tissue
- Scattered islands and strands of odontogenic epithelium
- Some cells may be vacuolated.
- The degree of epithelial proliferation may vary from minimal to prominent.

Differential Diagnosis
- Peripheral giant cell granuloma
- Pyogenic granuloma
- Peripheral fibroma
- Peripheral ameloblastoma

Treatment
- Excision: local and conservative

Prognosis
- Excellent
Benign Nonodontogenic Tumors

Carotid Body Tumor

Etiology

• Rare neoplasm arising from nonchromaffin paraganglia in carotid artery bifurcation
• Heredofamilial (autosomal-dominant) form can occur (in less than 10%)
• Can be multiple, bilateral, or multicentric

Clinical Presentation

• Typically presents as a mass in the lateral neck
• May be associated with bruit, hoarseness, dysphagia

Diagnosis

• Ultrasonography as a screening measure
• Angiography of both carotid systems

Differential Diagnosis

• Metastatic tumor
• Vagal nerve sheath tumor

Treatment

• Surgical removal
• Radiation therapy
• Combined surgical and radiotherapy

Prognosis

• Generally good
• Can be locally invasive
• May metastasize in 5 to 25% of cases
Exostosis

Etiology
• Unknown
• Probable reactive phenomenon (stimulus undetermined)

Clinical Presentation
• Asymptomatic, bony, nodular masses
• Cortical bone enlargement of the jaws; usually bilateral and symmetric
• Usually multiple; slow growing
• Most commonly along buccal/facial aspects of the maxillary and mandibular alveolar ridge
• Overlying mucosa intact, unremarkable
• Usually develops in adults

Diagnosis
• May appear radiographically as homogeneous opacities

Differential Diagnosis
• Peripheral fibroma
• Periostitis
• Periosteal/parosteal osteosarcoma

Treatment
• None required
• May need to be removed for prosthesis (denture) construction

Prognosis
• Excellent
Juvenile Ossifying Fibroma

Etiology
- A rapidly evolving variant of ossifying fibroma of the young
- Cause unknown

Clinical Presentation
- Onset between 5 and 15 years of age
- Rapid growth over several weeks
- Maxilla and paranasal areas predominate
- Tooth displacement common

Radiographic Findings
- Well-defined radiolucency
- Focal mineralization may be noted.
- Adjacent bone may be eroded or destroyed.

Microscopic Findings
- Prominent stromal cellularity
- Woven bone and/or psammomatous calcifications
- Plump osteoblast rimming

Diagnosis
- Correlation of histologic and radiographic findings

Differential Diagnosis
- Osteosarcoma
- Central giant cell granuloma
- Odontogenic tumor

Treatment
- Wide local excision or resection
- Reconstruction

Prognosis
- Recurrence rate of 30 to 50%
Benign Nonodontogenic Tumors
Langerhans Cell Disease
(“Histiocytosis X,” Idiopathic Histiocytosis)

Etiology
• Unknown
• Proliferation of Langerhans’ cells (immune surveillance cells) normally found in skin, mucosa, bone marrow, and lymph nodes

Clinical Presentation
• A broad spectrum, typically divided into three subsets, as follows:
  • Unifocal or multifocal chronic disease of bone (eosinophilic granuloma)
  • Widely disseminated chronic disease of bone and soft tissue (Hand-Schüller-Christian disease)
  • Acute, disseminated disease with bone marrow involvement (Letterer-Siwe disease)
• Most arise in childhood; eosinophilic granuloma often arises in adolescents and adults.
• Jaw lesions noted in up to 20% of cases with tenderness, loose teeth (focal to segmental), gingival inflammation, and friability

Radiographic Findings
• Bone lesions often punched out, sharply circumscribed
• “Floating teeth” appearance with alveolar bone involvement
• Skeletal survey should be performed to rule out multiple bone involvement

Diagnosis
• Radiographic demonstration of lytic bony lesions
• Infiltrate of mononuclear cells, often with clefted nuclei
• Often accompanied by a variety of other cell types, including eosinophils, lymphocytes, giant cells, plasma cells
• Immunohistochemical demonstration of CD1a staining
• Langerhans’ cells also stain for S-100 protein, although the antibody is less specific.
• Ultrastructural demonstration of cytoplasmic racquet-shaped Birbeck granules

Differential Diagnosis: Clinical
• Cat-scratch disease
• Juvenile xanthogranuloma
Benign Nonodontogenic Tumors

Differential Diagnosis: Radiologic
• Juvenile periodontitis, endocrinopathies, hypophosphatasia, leukemia, bony malignancy (primary/metastatic)
• In adults: myeloma

Treatment
• Localized variant
  • Surgical curettage of bony lesions
  • Low-dose radiation therapy of inaccessible lesions
• Widespread variants
  • Chemotherapy including methotrexate, vincristine, cyclophosphamide
  • Bone marrow transplantation for resistant/recurrent disease

Prognosis
• Varies with form of disease, as follows:
  • Localized variant: very good
  • Disseminated variant: fair to poor
Ossifying Fibroma

Etiology
• A benign fibro-osseous lesion of bone
• Cause unknown

Clinical Presentation
• Expansile lesion of bone
• Cortices intact
• May produce deformity, malocclusion, dysfunction
• Mandibular lesions are more common than are maxillary.

Radiographic Findings
• Well-delineated, smooth contours
• Quality varies from lucent to opaque
• Margins may be sclerotic.
• Can resorb roots and displace teeth
• May displace mandibular canal

Microscopic Findings
• Fibrovascular stroma
• Islands/trabeculae of osteoid, woven bone
• Cementum droplets may be present.

Diagnosis
• Correlation of histologic and radiographic findings

Differential Diagnosis: Radiographic
• Odontogenic cyst
• Giant cell lesion
• Odontogenic tumor

Differential Diagnosis: Histologic
• Fibrous dysplasia (must have clinical-pathologic correlation)

Treatment
• Conservative excision
• Enucleation with peripheral bony curettage
Prognosis

- Excellent
Osteoma

Etiology
• Sporadic form is idiopathic
• May be a component of Gardner’s syndrome
• Excludes maxillary and mandibular tori

Clinical Presentation
• Sporadic form with frontal and sphenoid sites predisposed
• May be multiple
• Solitary lesions rare in jaws

Radiographic Findings
• Well circumscribed, dense, sclerotic
• May be subperiosteal or medullary

Diagnosis
• Radiographic features
• Microscopic features: normal cortical and trabecular bone

Differential Diagnosis
• Tori, exostoses
• Ossifying fibroma
• Osteoblastoma
• Focal sclerosing osteitis

Treatment
• Usually none
• Local resection, if compromising

Prognosis
• Excellent
• Little recurrence potential
• When associated with Gardner’s syndrome, malignant conversion of intestinal polyps is assured.
Peripheral Ossifying Fibroma

Etiology
- A reactive hyperplasia of the gingiva; may be related to chronic irritation
- Periodontal ligament/membrane origin postulated

Clinical Presentation
- Exclusive gingival location; commonly interdental
- Nodular, sessile to pedunculated, usually ulcerated mass
- Slow growing; may rarely displace teeth
- Usually in young adults and adolescents
- Early lesions may bleed easily.
- Anterior maxillary arch is favored site

Diagnosis
- Central islands or trabeculae of bone/cementum
- Fibroblastic proliferation in a sheet-like configuration
- Usually ulcerated with granulation tissue base

Differential Diagnosis
- Pyogenic granuloma
- Peripheral giant cell granuloma
- Peripheral fibroma
- Peripheral odontogenic tumor
- Osteosarcoma/chondrosarcoma
- Metastatic neoplasm

Treatment
- Excision including underlying periosteum or associated periodontal ligament

Prognosis
- Recurrence occasionally seen; believed to be related to incomplete excision
Benign Nonodontogenic Tumors
Angioedema

Etiology
- Usually triggered by ingested antigens (eg, shellfish, nuts, fruits, medications)
- Mechanism associated with immunoglobulin E (IgE)-mediated mast cell degranulation with subsequent histamine release
- Drug reactions resulting in release of inflammatory mediators (bradykinin)
- Some cases have a genetic basis: C1 esterase inhibitor deficiency or inhibitor dysfunction (autosomal recessive)
- May be correlated with disease states characterized by the presence of circulating immune complexes

Clinical Presentation
- Soft, diffuse, painless swelling of face, lips, and neck
- Overlying skin and oral mucosa appear noninflamed
- Mucosa may become secondarily erythematous, ulcerative, or, rarely, vesicular
- Usually short-lived (24–48 hours)

Diagnosis
- Nonspecific histology
- Correlation of history and clinical findings

Differential Diagnosis
- Trauma (physical, cold)
- Cellulitis
- Vascular malformation
- Acute contact stomatitis
- Melkersson-Rosenthal syndrome (early stages)
- Orofacial granulomatosis (early stages)

Treatment
- Elimination of possible etiologic/precipitating factor(s)
- Antihistamines, corticosteroids, adrenaline
Prognosis

- Good to excellent
Cheilitis Granulomatosa

Etiology
- Isolated, idiopathic, and chronic lip enlargement
- May be an incompletely expressed or oligosymptomatic form of Melkersson-Rosenthal syndrome

Clinical Presentation
- One or both lips may be diffusely enlarged and nontender.
- Episodic swelling initially, with progression to a persistent enlargement
- Less often, superficial labial exfoliation or surface weeping/crusting may be noted.
- Lip swelling may herald similar changes of the gingiva, buccal mucosa, or palate.
- May be associated with Crohn’s disease, sarcoidosis, contact sensitivity, dental abscesses

Microscopic Findings
- Demonstrates noncaseating epithelioid granulomas
- Absence of organisms

Diagnosis
- History of intermittent to persistent asymptomatic lip swelling
- Characteristic appearance
- Lip or soft tissue biopsy (involved gingiva)
- Rule out sarcoidosis (chest radiograph, serum angiotensin-converting enzyme levels)
- Patch testing for contact allergens
- Dental radiographs to rule out asymptomatic periapical pathology

Differential Diagnosis
- Angioedema
- Cellulitis/erysipeloid reaction
- Sarcoidosis
- Crohn’s disease
- Melkersson-Rosenthal syndrome
- Cheilitis glandularis
- Contact stomatitis
Treatment
- Local intralesional triamcinolone injections under local anesthesia
- 5 to 10 mg total dose in depot fashion every 3 to 4 weeks to achieve response
- Local treatment may be coupled with an initial systemic course of glucocorticoids.
- Clofazimine 100 mg daily for 60 days with reduction to a maintenance dose of 30 mg on alternate days
- Metronidazole may also be effective at 250 mg three times daily for 1 month. This may be coupled with intralesional corticosteroid placement.
- Dapsone may be effective (as per dermatitis herpetiformis dosing)
- Surgical reduction (cheiloplasty) may be necessary.

Prognosis
- Guarded
- Must remain aware of possible neurologic manifestations, ophthalmologic involvement, psychological effects
Drug-Induced Stomatitis (Stomatitis Medicamentosa)

Etiology
- Oral changes found in approximately 5% of those with cutaneous reaction to drugs
- Mucosal alterations may result from the following:
  - Myelosuppression
  - Direct cytotoxic or cytostatic effect(s) on dividing epithelial cells
  - Xerostomic effects
  - Alterations of oral microbial flora

Clinical Presentation
- Painful, erythematous, erosive, or ulcerative lesions
- Nonkeratinized locations often affected initially
- Fixed form of drug-associated eruptions relatively uncommon intraorally
- Pseudomembranous necrotic surface may be noted

Diagnosis
- History
- Clinical appearance

Differential Diagnosis
- Chemical or thermal burn
- Erosive lichen planus
- Pemphigus vulgaris
- Mucous membrane (cicatricial) pemphigoid
- Erythema multiforme
- Acute herpetic gingivostomatitis
- Candidiasis

Treatment
- Identification and withdrawal of offending drug
- Symptomatic management including topical preparations (see “Therapeutics” section)
• Systemic corticosteroids if mucosal reaction is not related to antineoplastic treatment

**Prognosis**
• Generally excellent
Garré’s Osteomyelitis

Etiology
• Chronic, low-grade, dentoalveolar infection
• Resultant bony inflammation extends to the periosteum, producing a reduplication of the cortex (“onion skin” effect).

Clinical Presentation
• Usually an asymptomatic, unilateral, mandibular, bony hard asymmetry
• Limited to children and young adults

Radiographic Findings
• Medullary mottling with (lucent and opaque) ill-defined margins
• Periosteal-cortical expansion
• Occlusal radiograph shows concentric or parallel layering of cortex

Diagnosis
• Carious mandibular tooth, usually first permanent molar
• Radiographic features
• Biopsy results showing periosteal osteoblastic reaction, minimally inflamed fibrous marrow

Differential Diagnosis
• Ewing’s sarcoma
• Langerhans cell disease (histiocytosis X)
• Osteosarcoma
• Fibro-osseous lesion
• Metastatic disease

Treatment
• Elimination of the infected focus (carious tooth to be extracted or filled)
• Antibiotic administration early in treatment phase

Prognosis
• Good
Gingivitis

Etiology
• Variable
• Most are microbiologic or plaque associated (simple marginal gingivitis).
• Some are modified by hormonal changes, such as those in pregnancy (pregnancy gingivitis).
• Fusospirochetal gingivitis plus poor oral hygiene and poor nutrition are associated with acute necrotizing ulcerative gingivitis.
• Rarely, some forms are associated with contact allergy (“plasma cell gingivitis”).

Clinical Presentation
• Dependent on etiology, as follows:
  • Plaque associated: marginal inflammation to more generalized erythema and blunting of interdental papillae with rolled margins
  • Hormonally related: diffuse erythema and hyperplasia
  • Fusospirochetal: necrotic, blunted, ulcerated interdental papillae with spontaneous bleeding; foul odor
  • Allergy based: hyperplastic and bright red, granular to velvety surface alteration

Diagnosis
• Identification of cause
• Patch testing for contact allergens

Differential Diagnosis
• Acquired immunodeficiency syndrome–associated periodontal disease
• Oral lichen planus
• Mucous membrane (cicatricial) pemphigoid
• Acute herpetic gingivostomatitis
• Pemphigus vulgaris
Inflammatory Diseases

**Treatment**
- Local débridement and chlorhexidine rinses in cases of bacterial origin
- Reduction of hormonal dosage
- Elimination of allergen

**Prognosis**
- Excellent
Median Rhomboid Glossitis

Etiology
- A benign, inflammatory condition
- Often related to yeast colonization (erythematous candidiasis)
- Inflammatory process noted in response to overlying Candida population
- Exact mechanism is unclear

Clinical Presentation
- Well-defined, asymptomatic erythematous patch on dorsum of tongue
- Paramedian erythema, usually with focal atrophy of filiform papillae
- Chronic forms may become multinodular.
- Rarely may be hyperkeratotic
- May be mistaken for a benign or malignant tumor

Microscopic Findings
- Papillary, atrophic or hyperplastic epithelium
- Candidal colonization of surface
- Heavy, chronic inflammatory infiltrate

Diagnosis
- Clinical appearance, location

Treatment
- Topical and/or brief course of systemic antifungal therapy (optional)
- Observation

Prognosis
- Excellent
Inflammatory Diseases

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Osteomyelitis

Etiology
- An acute or chronic inflammatory process within the medullary space or along the cortical surface of bone
- Usually due to extension of a periapical abscess
- Other common causes include physical trauma (fracture) or bacteremia.
- Most common organisms include staphylococci and streptococci

Clinical Presentation
- Pain, swelling, fever, lymphadenitis
- Sequestrum formation
- Lower lip paresthesia, occasionally with acute disease in mandible
- Associated soft tissue swelling

Radiographic Findings
- Acute phase may be unremarkable
- Ill-defined, patchy radiolucency (“moth eaten”)

Diagnosis
- Presentation and radiographic findings
- Microscopic evidence of intrabony inflammation, marrow fibrosis, osteoclastic resorption, reduced osteoblastic activity, nonviable bone

Differential Diagnosis
- Osteosarcoma
- Local extension of malignant tumor
- Metastatic tumor
- Osteoradionecrosis

Treatment
- Drainage and antibiotics for acute disease
- Débridement, sequestrectomy, antibiotics for chronic disease
- Reconstruction if necessary after disease is resolved

Prognosis
- Good
Osteoradionecrosis

Etiology
- A serious complication of tumoricidal doses of radiation to the head and neck, usually > 60 Gy (6,000 rads)
- Radiation produces damage to the microvasculature, permitting a hypoxic state, which, in turn, leads to a hypocellular bony environment.
- Minor damage to the irradiated bone produces a nonhealing wound, forming dead bone—necrosis.

Clinical Presentation
- Usually affects the mandible
- Bone pain
- Exposed necrotic bone within radiation portal
- External fistula formation
- Pathologic fracture

Radiographic Findings
- Irregular zones of mixed radiopacity and radiolucency
- Separation of nonvital bone (sequestrum) from remaining viable bone

Diagnosis
- Radiographic and clinical features
- Biopsy results show nonvital bone.

Differential Diagnosis
- Metastatic tumor
- Locally recurrent tumor
- Osteomyelitis
- Osteosarcoma
- Radiation-induced sarcoma

Treatment
- After biopsy, débridement of bone preceded and followed by hyperbaric oxygen therapy
- If necessary, resection and reconstruction
• Necessary tooth extraction and elimination of focal infection within radiation portal 21 days prior to treatment
• Excellent preventive dental care

**Prognosis**
• Guarded
Periapical Granuloma

Etiology
• A mass of inflamed granulation tissue
• Forms secondary to pulp necrosis of the associated tooth
• May develop following periapical abscess formation or may form as pulpal death eventuates without abscess precursor

Clinical Presentation
• Usually asymptomatic
• With acute exacerbation, pain and sensitivity develop.
• Tenderness at root apex on palpation
• Pain on biting or percussion of tooth

Radiographic Findings
• Radiolucency at apex of tooth
• Size ranges up to 1 to 2 cm in diameter
• Root resorption not uncommon

Diagnosis
• Radiographic features
• Demonstration of nonvital pulpal component

Differential Diagnosis
• Periapical cemental dysplasia
• Periapical cyst

Treatment
• Conventional endodontic therapy
• Apical curettage/root end amputation if above measures fail
• Extraction of involved tooth

Prognosis
• Excellent
Sarcoidosis

Etiology
• Unknown; granulomatous disease process
• May represent a systemic response to a single provoking agent; mycobacteria has been suggested but not proven
• Possible role of genetic factors coupled with disordered reactions to foreign antigens

Clinical Presentation
• Mucocutaneous
  • Red to brown nodules/plaques with erythema nodosum features
  • Minor salivary glands of the lips and palate may be involved.
  • Erythematous, hyperplastic gingiva
• Salivary/lacrimal
  • Parotid, submandibular, and lacrimal glands may be enlarged.
• Multiple organ systems, such as the following, may be involved:
  • Particularly the lung, but also liver, endocrine glands, the heart, and the reticuloendothelial and musculoskeletal systems
  • Heerfordt’s syndrome may be related to sarcoidosis (uveitis, parotid gland enlargement, fever, cranial nerve palsies).

Diagnosis
• Demonstration of sarcoidal (noncaseating epitheloid) granulomas in at least two organ systems
• Elevated serum angiotensin-converting enzyme levels are usually present.
• Over 90% of cases have abnormal chest radiograph.
• Other causes of granulomatous inflammation must be ruled out.

Differential Diagnosis
• Tuberculosis
• Lymphoma (non-Hodgkin’s, Hodgkin’s)
• Deep fungal infection
• Crohn’s disease
Treatment
- Corticosteroids, if symptoms demand
- Severe or unresponsive cases: methotrexate
- Cutaneous lesions only: hydroxychloroquine
- Intralesional corticosteroids

Prognosis
- Generally good
Tooth Abnormalities

Abrasion

Etiology
- Excessive or abnormal wearing of teeth
- Commonly associated with use of smokeless tobacco, abrasive dentifrices, cigars, and pipes, habitual grinding, improper tooth-brushing techniques
- Pathologic wear of teeth associated with abnormal habits or function or consumption of abrasive to coarse diets
- The so-called toothbrush abrasion at the gingival margin may be related to abnormal incisal or occlusal forces producing abfraction injury to enamel at the cementoenamel junction

Clinical Presentation
- Causally related appearance includes the following:
  - Incisal/occlusal wear related to habit or abrasive diet or substance
  - Cervical wear of posterior teeth with chronic, low-grade toothbrush injury
- At occlusal and incisal surfaces, a generalized loss of crown height
- At the cervical margin (cementoenamel junction), horizontal V-shaped to saucerized notches (abfraction injury)
- With exposure of significant root surface, abrasion-related injury, diffuse loss of cementum and dentin
- Pulp canals, containing tertiary dentin, are visible in advanced cases.

Diagnosis
- Clinical appearance

Differential Diagnosis
- Amelogenesis imperfecta
- Dentinogenesis imperfecta
Tooth Abnormalities

Treatment
• Restorative dental techniques
• Correction of habits, occlusal force discrepancies

Prognosis
• Good
Amelogenesis Imperfecta

Etiology
- Intrinsic enamel defect that affects all teeth of both dentitions
- Results from defective amelogenin genes on X and Y chromosomes and also chromosome 4 (tuftelin gene)
- At least 16 variants noted based upon inheritance pattern, enamel qualities, and radiographic features
- Frequency of 1:14,000 to 1:16,000 of population

Clinical Presentation
- One of three basic alterations of enamel may be seen: hypoplasia, hypomaturation, or hypocalcification
- Enamel hardness varies depending upon type of defect: normal hardness in hypoplastic form but deficient amounts of enamel; soft enamel in the hypocalcified variant but normal amounts of enamel
- Color ranges from normal (hypoplastic) to dark yellow-brown (hypocalcified)
- Radiographic changes range from normal density (hypoplastic) to less dense (hypocalcified)
- May be noted in association with taurodontism (coronally enlarged dental pulps)
- X-linked form demonstrates random vertical bands of normal and hypoplastic enamel

Diagnosis
- Clinical and radiographic features
- Family history (autosomal, X-linked forms)

Treatment
- Full-crown restorations for esthetics
- Genetic counseling
Tooth Abnormalities
Attrition

Etiology
- Defined as a physiologic wearing of teeth secondary to normal function/mastication
- Can involve all surfaces of teeth including interproximal, incisal-occlusal, buccal, and lingual
- Abnormal occlusal-incisal relationship can predispose to accelerated rates of attrition

Clinical Presentation
- Primary and permanent dentitions can be affected.
- Flattened occlusal surfaces and reduction of incisal height
- The loss of interproximal tooth surface (usually enamel only) leads to gradual dental arch shortening.
- Asymptomatic

Diagnosis
- Characteristic appearance
- Generally proportional to age

Treatment
- Usually does not require specific management
- Elective restoration of occlusal/incisal surfaces to prevent over-closure of jaws in function

Prognosis
- Excellent
Tooth Abnormalities
Bulimia

Etiology

• A compulsive-eating disorder characterized by repeated episodes of binge eating followed by vomiting or another form of purging, including laxative abuse
• Cause may be biologic (neurometabolic disturbance), psychological (societal pressure for extreme thinness), or combined (biopsychosocial)
• Nearly 1.2 million adolescent and young adult females are affected in the United States; males are considerably less affected. Up to 20% of college-age women are affected.

Clinical Presentation

• Oral signs include erosion of teeth, gingivitis, xerostomia, painless parotid gland enlargement, increased caries rate, thermal hypersensitivity of teeth
• Specific patterns of enamel destruction (perimolysis) are noted along the palatal and occlusal aspects of maxillary teeth, sparing the buccal and labial surfaces.
• Mandibular teeth usually are affected less severely.

Diagnosis

• Recognition of oral signs
• Coordination of oral signs with other findings including dermatologic signs: lanugo-like body hair, brittle hair and nails, asteatotic skin, hand or finger calluses (related to self-induced vomiting)

Differential Diagnosis

• Diet-induced enamel loss
• Chronic gastric reflux disease

Treatment

• Combined aggressive medical management, psychotherapy, behavioral management, food intake management, and nutritional counseling
Prognosis

- Fair to good
- Mortality (estimates range from 1–15%) is divided equally between medical complications (electrolyte disturbance, acute renal failure, cardiac complications) and suicide.
Dentinal Dysplasia

Etiology
- An inherited disorder (autosomal dominant) of circumpulpal dentin with associated alterations of root morphology; no other organs affected

Clinical Presentation
- Premature tooth loss
- All teeth affected
- Two forms, as follows:
  - More severe form (type I) characterized by “rootless teeth,” with normal-colored crowns, obliterated pulp chambers, multiple periapical radiolucencies (periapical granulomas/cysts)
  - Less severe form (type II) characterized by amber-colored primary teeth with susceptibility to wear; permanent teeth of normal color with thistle-shaped pulp chambers; frequent pulp stones noted

Diagnosis
- Combined clinical and radiographic features
- Normal clinical color of permanent teeth and periapical lesions help to distinguish from dentinogenesis imperfecta
- Clinical crowns of primary teeth are amber and opalescent.
- Absent root formation (type I)
- Thistle-shaped pulp chambers and pulp stones (type II)

Differential Diagnosis
- Chemotherapy-/radiation therapy–induced root development alteration
- Pulpal dysplasia

Treatment
- Teeth usually unsalvageable (type I)
- Observation
- Genetic counseling

Prognosis
- Guarded
Dentinogenesis Imperfecta

Etiology
- Hereditary disorder (autosomal dominant) of dentin (1:8,000 frequency in population)
- May be seen in association with osteogenesis imperfecta
- Altered dentin matrix is related to the defective degradation of dentin phosphoprotein during dentinogenesis.

Clinical Presentation
- Primary and permanent dentition exhibit gray to brownish opalescence
- Normal enamel fractures easily from defective underlying dentin
- Severe tooth abrasion related to exposed dentin following enamel loss
- Radiographically, roots are slender to spiked with pronounced cervical constriction and obliterative pulpal calcification
- Constricted tooth cervix gives molar crowns a “tulip” profile

Diagnosis
- Clinical and radiographic appearance
- Family history

Differential Diagnosis
- Osteogenesis imperfecta

Treatment
- Functional and esthetic restorations (full crowns)
- Genetic counseling
Erosion

Etiology
• Acid dissolution of enamel and dentin
• Loss of enamel and, less commonly, dentin secondary to chemical (usually acids) action/demineralization
• Intrinsic sources relate to stomach acid presence within the oral cavity.
• May be due to the following:
  • Occupational exposure to acids
  • Diet with acid exposure (phosphoric acid–containing beverages; sucking on lemons)
  • Chronic regurgitation/gastroesophageal reflux
  • Bulimia-related vomiting

Clinical Presentation
• Loss of enamel initially along lingual surfaces of anterior teeth (bulimia, reflux)
• Labial enamel loss (beverage related, occupation related)
• Cupped dentin noted to occur more rapidly than adjacent enamel loss on occlusal surfaces
• Existing metallic restorations (inlays, amalgam fillings) and any protected enamel may be above the surrounding dentin, creating a “ledge” effect.
• Smooth to polished appearance of maxillary incisors in chronic, high-volume consumers of beverages (phosphoric or citric acid–containing)

Diagnosis
• Correlation of appearance with diet, habits, environmental exposure, underlying eating disorder, or chronic acidic reflux

Differential Diagnosis
• Amelogenesis imperfecta
• Factitial injury

Treatment
• Identification and elimination of cause
• Treatment of underlying etiology
• Dental restorative treatment subsequent to complete functional evaluation, vertical dimension, and esthetics

**Prognosis**

• Excellent
Fluorosis: Chronic Endemic

Etiology
• Excessive dietary levels of fluoride: greater than one part per million in drinking water (can lead to fluorosis in a dose-dependent relationship)
  • Childhood ingestion of fluoride dentifrice on a chronic basis
  • Tooth enamel hypomaturation resulting from prolonged ingestion of abnormally high levels of fluoride during tooth development, usually between 2 and 3 years of age

Clinical Presentation
• Enamel alterations ranging from local pitting to white opacity or deeper brown mottling
• Distribution is symmetric and bilateral and occurs in all quadrants of the jaws.

Diagnosis
• Characteristic appearance and distribution
• Data concerning fluoride concentration in drinking water should be obtained.

Differential Diagnosis
• Amelogenesis imperfecta
• Tetracycline-associated staining

Treatment
• Restorative dental treatment
• Cosmetic bleaching

Prognosis
• Excellent
Tooth Abnormalities
Fusion

Etiology
• Merging of two tooth germs to create a single tooth

Clinical Presentation
• A single large tooth (macrodont) is noted.
• One less tooth will be present in the dental arch.

Radiographic Findings
• Common or separate pulp canals and roots

Diagnosis
• Radiographic evaluation
• One less tooth present in dental arch

Treatment
• If esthetics demand, removal and replacement

Prognosis
• Not applicable
Tooth Abnormalities
Natal Teeth

Etiology
• Usually indicates prematurely erupted deciduous teeth

Clinical Presentation
• Erupted teeth at birth
• Almost always are incisors
• 85% appear in mandible

Treatment
• If mobile, extraction
• Possible retention for functional, esthetic reasons
Malignant Nonodontogenic Tumors

Ewing’s Sarcoma

Etiology
- Unknown
- Chromosomal translocations t(11;22), t(7;22), t(7;21) noted
- Gene rearrangement often noted, that is, (22;q12) and expression of the MIC2 gene
- Genetically related to primitive peripheral neuroectodermal tumor via translocations t(11;22), (q24;q12)

Clinical Presentation
- 60% in males; over 95% in those under 20 years of age
- Chiefly in bone and soft tissues
- Highly malignant
- Pain, numbness, and swelling often early complaints
- Diffuse, irregular, lytic bone lesion
- Cortical expansion variable
- Second most common bone tumor of children/adolescents
- Soft tissues of head and neck account for 11% of extraskeletal sites

Diagnosis
- Radiographs often show “moth-eaten” appearance and laminar periosteal bone reaction
- Cortex may be eroded or expanded

Differential Diagnosis
- Osteosarcoma
- Lymphoma
- Peripheral neuroectodermal tumor of bone
- Primitive rhabdomyosarcoma
- Neuroectodermal tumor of infancy
Treatment
• Radiation and multiagent chemotherapy

Prognosis
• 54 to 74% 5-year survival rate in localized osseous form
• Late relapse not uncommon
Metastatic Cancer

Etiology
• Spread of a primary malignancy to the oral cavity structures or jaws (usually from lung, breast, prostate, colon, kidney)
• Accounts for < 1% of oral malignancies

Clinical Presentation
• Usually manifests in the jaws with pain and swelling
• Not uncommon is loosening of teeth or pathologic jaw fracture
• Soft tissue location is rare.
• Most frequent sites of primary neoplasms are kidney, lung, breast, colon, prostate, stomach
• Intraosseous lesions with lytic, ill-defined radiolucentes

Microscopic Findings
• As with the primary tumor
• Tumor marker studies (immunohistochemical) may be necessary to define the site of origin.

Diagnosis
• Radiographic findings
• Biopsy

Differential Diagnosis
• Primary soft tissue tumor
• Primary osseous tumor
• Periodontitis (localized)
• Osteoradionecrosis

Treatment
• Local radiation
• Combination chemoradiotherapy

Prognosis
• Poor
Osteosarcoma

**Etiology**
- May be associated with pre-existing bone disease such as the following:
  - Paget’s disease (10 to 15%)
  - Fibrous dysplasia (0.5%)
  - Mutation/amplification of p53, c-myc, c-JUN, c-fos, MOM2, CDK4, SAS

**Clinical Presentation**
- May present with pain paresthesia, trismus, nasal or paranasal sinus obstruction
- May masquerade as an odontogenic infection
- Intraoral signs are as follows:
  - Tooth mobility (vertical)
  - Periapical radiolucency (teeth vital)
  - Distal displacement of terminal molar
  - Jaw mass may be ulcerated.

**Radiographic Findings**
- Early intraoral findings
  - Displacement of teeth
  - Root resorption
  - Absent or attenuated lamina dura
  - Uniformly widened periodontal membrane space
- Later jaw bone findings
  - Lytic, “moth-eaten” destruction
  - Cortical destruction
  - Soft tissue extension
  - Erosion of mandibular canal
  - 25% of cases have “sunburst effect” (radiating radiopaque spicules)

**Microscopic Findings**
- Sarcomatous stroma
- Osteoid production by neoplastic cells
- Four basic patterns (no prognostic significance) are as follows:
  - Osteoblastic
  - Chondroblastic
  - Fibroblastic
  - Telangiectatic
Diagnosis
• Correlation of clinical, radiographic, pathologic findings

Differential Diagnosis
• Fibro-osseous lesion
• Osteomyelitis
• Osteoradionecrosis
• Metastatic tumor
• Other form of sarcoma

Treatment
• Radical ablative surgery
  • Hemimandibulectomy
  • Partial maxillectomy ± orbital exenteration
• Adjuvant chemotherapy/radiotherapy

Prognosis
• Survival ranges from 12 to 58% at 5 years
• Mandibular lesions are associated with a greater survival rate than are maxillary lesions.
Metabolic and Genetic Disorders

Amyloidosis

Etiology
- May be primary (idiopathic), secondary to systemic disease, or familial
- Formation of a fibrillar protein deposited in soft tissues and visceral organs with associated levels of dysfunction

Clinical Presentation
- The primary form may produce obvious tongue enlargement (macroglossia) and associated purpura, or nodular submucosal alterations.
- The secondary form may be subtle; gingival tissues may contain deposits of amyloid.

Diagnosis
- Appearance of tongue
- Systemic complaints
- Biopsy results: demonstration of amyloid deposits in tissues (tongue, gingiva)

Differential Diagnosis
- Hyalinosis cutis et mucosae (lipoid proteinosis)
- Leukemic infiltrate
- Lymphangioma
- Neurofibromatosis
- Hemodialysis-related disorder

Treatment
- Directed to underlying cause (secondary)
- Localized amyloid tumors may be excised.
- Generally symptom related (dialysis, digitalis, depending upon organ involvement)

Prognosis
- When renal impairment exists, transplantation may be necessary.
Cherubism

Etiology
• Autosomal-dominant, fibroblast/giant cell–containing condition
• May be secondary to somatic mutation, mapping to chromosome 4p16.3
• No associated metabolic or biochemical alterations noted
• Possible linkage/association with Noonan’s syndrome

Clinical Presentation
• Early signs in childhood
• Bilateral, symmetric enlargement of mandible
• Maxillary involvement less common and less prominent
• Dental arch/occlusal discrepancies may be noted.
• Unerupted teeth often noted
• Facial features include lower-third fullness and scleral exposure at a forward resting gaze.

Radiographic Findings
• Symmetric, multiloculated, expansile radiolucencies of mandibular body and ramus
• Impacted/displaced teeth common
• Thinned cortices with scalloped medullary margins
• Older patients may exhibit maturation with bone fill in some areas but with preservation of expanded bony profile.

Diagnosis
• Clinical appearance
• Radiographic findings

Differential Diagnosis
• Central giant cell granuloma (multiple)
• Fibrous dysplasia
• Langerhans cell disease (histiocytosis X)
• Hyperparathyroidism
• Multiple odontogenic keratocysts
**Treatment**

- Variable, ranging from cosmetic recontouring to local curettage early in lesion development
- Active surgical intervention should be deferred until after the pubertal growth spurt, if possible.

**Prognosis**

- Stability usually noted by end of skeletal growth
- Often regresses into adulthood, but variably so
Cleidocranial Dysplasia

Etiology
- Autosomal-dominant trait with high penetrance and variable expressivity
- Mutations in SH3-binding protein on chromosome 4p16.3
- Widespread membranous and endochondral defects in craniofacial complex

Clinical Presentation
- Chief head and neck manifestations include the following:
  - Defective ossification
  - Wormian bones with calvarial defects
  - Delayed fontanelle and suture closure
  - Variably developed clavicles often a prominent skeletal finding
  - Long, narrow neck with variably drooped shoulders
  - Midface deficiency secondary to hypoplasia of facial bones and paranasal sinuses
  - Ocular hypertelorism
  - Palate with narrow, high-arched quality
  - Delayed closure of mandibular symphysis
  - Multiple unerupted and malpositioned teeth with lack of cellular cementum
  - Multiple supernumerary teeth

Diagnosis
- Clinical features
- Radiographic findings (skull, jaw, chest)

Differential Diagnosis
- Achondroplasia
- Pyknodysostosis
- Hydrocephalus

Treatment
- Genetic counseling
- For dental abnormalities, treatment options are as follows:
  - Early orthodontic intervention
  - Surgical exposure of unerupted teeth
  - Extraction of supernumerary teeth
• Surgical correction of jaw deformities
• Dental reconstruction

**Prognosis**
• Stability with growth cessation
• Dental and oral rehabilitation can proceed as per usual after surgery (see above) is completed.
Hyperparathyroidism

Etiology
- Primary form usually due to parathyroid adenoma
- Secondary form related to altered renal vitamin D metabolism with secondary hypocalcemia
- Excessive parathormone secretion common to all forms

Clinical Presentation
- Classic triad in patients over 60 years includes the following:
  - Renal calculi/nephrolithiasis
  - Subperiosteal resorption of phalanges
  - Lethargy, psychotic-like state
- Fibrous/lytic bone lesions; chief oral finding is well-defined, cyst-like radiolucencies of jaw(s)
- Osteoporotic bony changes
- Loss of lamina dura
- Duodenal ulcer formation

Diagnosis
- Increased serum calcium levels (primary)
- Increased urinary hydroxyproline levels
- Elevated serum parathormone
- Radiographic changes (phalanges, jaws)
- Histologic findings: identical to central giant cell granuloma of jaws

Differential Diagnosis
- Cherubism
- Renal disease (osteodystrophy)
- Paget’s disease of bone (skull)—early stages
- Multiple odontogenic keratocysts (nevoid basal cell carcinoma syndrome)

Treatment
- Primary hyperparathyroidism: removal of abnormal gland(s)
- Secondary hyperparathyroidism: management of renal disease

Prognosis
- Good
Therapeutics

Actinomycosis
• Systemic therapy: penicillin or tetracycline in large doses for 3–6 mo
• Wide excision of infected tissue

Acute Herpetic Gingivostomatitis
• Systemic therapy
  • Valacyclovir 500 mg #20; 1 tablet twice daily × 10 d
  • Acyclovir 400 mg #50; 1 tablet 5 times daily × 10 d
• Fluids
• Analgesia

Acute Necrotizing Ulcerative Gingivitis
• Débridement of necrotic tissue
• Aggressive oral hygiene and plaque control
• Metronidazole 250 mg #40; 1 4 times daily × 10 d

Angioedema
• Systemic therapy
  • Antihistamine: diphenhydramine 50 mg capsules #12; 1 every 6 h × 2–3 d
  • Doxepin 25 mg tablets #12; 1 every 6 h × 2–3 d
  • Prednisone 10 mg tablets #12; 4 tablets daily × 3 d

Aphthous Stomatitis
• See “Recurrent Aphthous Stomatitis.”

Behçet’s Disease
• Treat as for aphthosis (see “Recurrent Aphthous Stomatitis”).
• Refer to a dermatologist, a rheumatologist, or an ophthalmologist, depending on organ involvement, for ongoing care, which may include systemic immunosuppressive and/or anti-inflammatory drugs.

Candidiasis
• Identify and correct provocative factors.
• Topical therapy
  • Nystatin oral suspension (100,000 units/mL); rinse 5 mL and swallow 4 times/d
  • Clotrimazole (Lotrimin) solution 1%; rinse 5 mL and swallow 4 times/d
• Clotrimazole troches (Myclex) 10 mg; dissolve 1 troche in mouth 5 times/d
• Clotrimazole vaginal tablets 1/2 of 500 mg tablet dissolved in mouth bid
• Systemic therapy
  • Fluconazole (Diflucan) 100 mg #15; 2 tablets on the first day, 1 tablet days 2–7, 1 tablet every other day for days 8–21
  • Ketoconazole (Nizoral) 200 mg #21; 1 tablet every day with breakfast × 21 d
  • Itraconazole (Sporanox) 200 mg #21; 1 tablet every day with breakfast × 21 d
• May use shorter duration for less severe infections

Cheilitis Glandularis
• Challenging to treat
• Trials of therapy
  • Intralesional corticosteroids as triamcinolone acetonide 5–10 mg/mL; inject 1–3 mL per session with sessions at 3–4 wk intervals
  • Systemic antibiotic: tetracycline 500 mg tid
  • Systemic corticosteroid: prednisone 5 mg tablets #40
    – Take each morning for 8 d with breakfast, 8-8-6-6-4-4-2-2 mg, stop
    – Will shorten the course of an individual episode but not change the natural history of the disease

Cheilitis Granulomatosa
• Challenging to treat
• Trials of therapy
  • Intralesional corticosteroids such as triamcinolone acetonide 5–10 mg/mL; inject 1–3 mL per session with sessions at 3–4 wk intervals
  • Systemic antibiotic: tetracycline 500 mg tid
  • Systemic corticosteroids: prednisone 5 mg tablets #40
    – Take each morning for 8 d with breakfast, 8-8-6-6-4-4-2-2 mg-stop
    – Will shorten the course of an individual episode but not change the natural history of the disease
  • Dapsone 25 mg tablets
    – Check baseline complete blood count (CBC), liver function tests, urinalysis, and glucose-6-phosphate red blood cell enzyme level before treatment.
– Take each morning with breakfast, 1 × 3 d, 2 × 3 d, 3 × 3 d, 4 × 7 d, and 5 daily thereafter.
– Check CBC and liver function every month for 3 mo, then every 3 mo thereafter.
– Use for long-term control of disease

**Crohn's Disease**

- Challenging to treat
- Trials of therapy
  - Intralesional corticosteroids such as triamcinolone acetonide 5–10 mg/mL; inject 1–3 mL per session with sessions at 3–4 wk intervals
  - Systemic antibiotic: tetracycline 500 mg tid
  - Systemic corticosteroid: prednisone 5 mg tablets #80
    - Take each morning with breakfast for 16 d as 8/d × 4 d, 6/d × 4 d, 4/d × 4 d, 2/d × 4 d, stop
    - Will reduce disease activity as topical corticosteroids or systemic nonsteroidal anti-inflammatory drugs (NSAIDs) are started
  - Dapsone 25 mg tablets
    - Check baseline CBC, liver function tests, urinalysis, and glucose-6-phosphate red blood cell enzyme level before treatment.
    - Take each morning with breakfast, 1 × 3 d, 2 × 3 d, 3 × 3 d, 4 × 7 d, and 5 daily thereafter.
    - Check CBC and liver function every month for 3 mo, then every 3 mo thereafter.
    - Use for long-term control of disease

**Drug-Induced Stomatitis (Stomatitis Medicamentosa)**

- Topical therapy (compounded rinses)
  - Option 1
    - Diphenhydramine 200 mg, viscous lidocaine 90 mL, Maalox suspension 90 mL, distilled water 180 mL
    - Swish 5 mL for 2 min and expectorate 3–4 times/d.
  - Option 2
    - Dexamethasone 100 mg, viscous lidocaine 60 mL, diphenhydramine 200 mg, sorbitol 15 mL, Maalox suspension to 275 mL
    - Swish 5 mL for 2 min and expectorate 3–4 times/d.
- Systemic therapy: prednisone 5 mg tablets #80
  - Take each morning with breakfast for 16 d as 8/d × 4 d, 6/d × 4 d, 4/d × 4 d, 2/d × 4 d, stop
  - Will reduce disease activity as topical corticosteroids or systemic NSAIDs are started

**Erythema Multiforme**

- Topical therapy (compounded rinses)
  - Option 1
    - Diphenhydramine 200 mg, viscous lidocaine 90 mL, Maalox suspension 90 mL, distilled water 180 mL
    - Swish 5 mL for 2 min and expectorate 3–4 times/d.
  - Option 2
    - Dexamethasone 100 mg, viscous lidocaine 60 mL, diphenhydramine 200 mg, sorbitol 15 mL, Maalox suspension to 275 mL
    - Swish 5 mL for 2 min and expectorate 3–4 times/d.

- Systemic therapy
  - Prednisone 5 mg tablets #80
    - Take each morning with breakfast for 16 d as 8/d × 4 d, 6/d × 4 d, 4/d × 4 d, 2/d × 4 d, stop
    - Will reduce disease activity as topical corticosteroids or systemic NSAIDs are started
  - Acyclovir 200 mg tablets #42 (if triggered by herpes simplex virus infection); 1 tablet every 4 h for 7 d or 1 tablet bid-tid as prophylaxis

**Exfoliative Cheilitis**

- Identify possible topical or drug-related causative agents (eg, gold, toothpaste, mouthwash, lipstick).
- Determine if factitial cause(s) is present.
- Topical therapy: see “Candidiasis”
- Systemic therapy: see “Candidiasis”

**Fissured Tongue**

- Brush tongue surface 10–15 times with dentifrice after meals and at bedtime to remove debris that causes halitosis.

**Geographic Tongue**

- Brush tongue surface 10–15 times with dentifrice after meals and at bedtime to remove debris that causes halitosis.
• Topical therapy
  • Fluocinonide gel/cream 0.05% 60 g; apply after meals and at bedtime
  • Clotrimazole troches (Mycelex) 10 mg; dissolve 1 troche in mouth 5 times/d
  • Clotrimazole vaginal tablets 1/2 of 500 mg tablet dissolved in mouth bid
  • Tacrolimus (Protopic) ointment 0.1% 60 g; apply after meals and at bedtime

**Hairy Tongue**
• Brush tongue surface 10–15 times with dentifrice after meals and at bedtime to remove debris that causes halitosis.
• Topical therapy: dilute H₂O₂ (1 part 3% H₂O₂:1 part H₂O); brush tongue after meals and at bedtime for black hairy tongue

**Hand-Foot-and-Mouth Disease**
• Fluids
• Analgesia
• Recovery expected quickly

**Herpangina**
• Fluids
• Analgesia
• Recovery expected quickly

**Herpes Zoster**
• Topical therapy
  • Calamine lotion for wet, oozing cutaneous lesions
  • Doxepin (Zonalon) cream for pain relief of acute lesions
• Systemic therapy
  • Acyclovir 400 mg tablets #100; 2 tablets 5 times daily × 7–10 d
  • Famciclovir 500 mg tablets #21; 1 tablet 3 times daily × 7 d
  • Valacyclovir 500 mg tablets #42; 2 tablets 3 times daily × 7 d

**Impetigo**
• Topical therapy: mupirocin ointment applied twice daily
• Systemic therapy
  • Penicillin V potassium 250 mg tablets #40; 1 tablet 4 times daily × 10 d
  • Erythromycin base 250 mg tablets #40; 1 tablet 4 times daily × 10 d
  • Dicloxacillin 250 mg tablets #40; 1 tablet 4 times daily × 10 d
Lichen Planus

- **Topical therapy**
  - Betamethasone cream (0.1%) 60 g; apply after meals and at bedtime
  - Fluocinonide gel/cream 0.05% 60 g; apply after meals and at bedtime
  - Tacrolimus (Protopic) ointment 0.1% 30 g; apply after meals 3 times daily and at bedtime, do not eat or drink for 30 min; taper frequency depending on response
- **Intralesional therapy**: triamcinolone acetonide 5–10 mg/mL; inject 1–3 mL per session with sessions at 3–4 wk intervals
- **Systemic therapy**
  - Prednisone 5 mg tablets #80
    - Take each morning with breakfast for 16 d as 8/d × 4 d, 6/d × 4 d, 4/d × 4 d, 2/d × 4 d, stop
    - Will reduce disease activity as topical corticosteroids or systemic NSAIDs are started
  - Dapsone 25 mg tablets
    - Check baseline CBC, liver function tests, urinalysis, and glucose-6-phosphate dehydrogenase enzyme level before treatment.
    - Take each morning with breakfast, 1 × 3 d, 2 × 3 d, 3 × 3 d, 4 × 7 d, and 5 × daily thereafter
    - Check CBC and liver function every month for 3 mos, then every 3 mo thereafter.
    - Use for long-term control of disease.
  - Hydroxychloroquine (Plaquenil) 250 mg #100; 2 tablets with breakfast for 4 wk, then 1 tablet daily for maintenance
    - Baseline ophthalmology consultation; repeat every 6 mo to monitor for retinal toxicity

Lupus Erythematosus

- **Topical therapy**
  - Fluocinonide gel/cream 0.05% 60 g; apply after meals and at bedtime
  - Tacrolimus (Protopic) ointment 0.1% 30 g; apply after meals 3 times daily, do not eat or drink for 30 min
- **Intralesional therapy**: triamcinolone acetonide 5–10 mg/mL; inject 1–3 mL per session with sessions at 3–4 wk intervals
Melkersson-Rosenthal Syndrome
• See “Fissured Tongue.”
• Orofacial granulomatosis—see “Cheilitis Granulomatosa”

Nevus
• All pigmented nevi should be excised, if reasonable from a surgical point of view.

Pemphigoid
• Refer to a dermatologist or an ophthalmologist, depending on organ involvement, for ongoing care, which may include systemic immunosuppressive and/or anti-inflammatory drugs.
• For localized oral pemphigoid/gingival pemphigoid, apply topical therapy: fluocinonide 0.05% gel/cream 60 g
  • Apply to early lesions after meals and at bedtime.
  • Do not apply to ulcers.
  • May be used for 1–2 h with mouthguard for occlusive therapy
• Systemic therapy for severe, chronic disease
  • Prednisone 5 mg tablets #80
    – Take each morning with breakfast for 16 d as 8/d × 4 d, 6/d × 4 d, 4/d × 4 d, 2/d × 4 d, stop
    – Will reduce disease activity as topical corticosteroids or systemic NSAIDs are started
  • Dapsone 25 mg tablets
    – Check baseline CBC, liver function tests, urinalysis and glucose-6-phosphate dehydrogenase enzyme level before treatment.
    – Take each morning with breakfast, 1 × 3 d, 2 × 3 d, 3 × 3 d, 4 × 7 d, and 5 × daily thereafter
    – Check CBC and liver function every month for 3 mo, then every 3 mo thereafter.
    – Use for long-term control of disease
  • Tetracycline and niacinamide
    – 500 mg of each administered tid
    – Use for long-term control of disease

Pemphigus Vulgaris
• Coordinate overall management with patient’s internist/primary care physician since treatment of this disease requires systemic immunosuppression and/or use of anti-inflammatory drugs.
• Management of oral lesions will consist of systemic immunosuppressive agents.
• Local/intralesional therapy may be a useful adjunct following an initial good measurable response to systemic glucocorticosteroid dosing.

• Systemic therapy: prednisone 10 mg tablets #150
  • Take each morning with breakfast at a total daily dose of 1 mg/kg of body weight.
  • Taper slowly over several months as clinical response permits to maintenance dosing.
  • Management of prednisone side effects is important.

• Corticosteroid-sparing systemic therapy
  • Azathioprine 1–3 mg/kg; dosing spaced morning and evening
  • Mycophenolate mofetil 500 mg tablets; 1.5 g bid

• Severe or unresponsive disease
  • Plasmapheresis
  • Pulse cyclophosphamide (Cytoxan) IV for 3 wk
    – Monitor response.
    – Continue on orally administered immunosuppressants.
  • IVIg therapy
  • Local therapy for focal residual lesions: intralesional triamcinolone suspension 10 mg/mL

**Plasma Cell Gingivitis**
• Identify contact allergen(s) and avoid exposure.
• Topical therapy: fluocinonide gel/cream 0.05% 60 g; apply after meals and at bedtime
• Systemic therapy: griseofulvin 250 mg tablets #150; take 1 with each meal for 7 wk

**Pyostomatitis Vegetans**
• Seek the underlying inflammatory bowel disease.
• See “Crohn’s Disease.”

**Radiation-Induced Mucositis**
• Topical therapy
  • Benzydamine rinses
  • Saline/bicarbonate rinses 2.5 mL each in 125 mL water; 5 mL rinsed bid
  • Chlorhexidine 0.12% compounded as alcohol-free formula
    – Store in a light-protective container.
    – 15–30 mL rinsed bid
• See “Drug-Induced Stomatitis.”
• Systemic therapy: analgesics prn
Recurrent Aphthous Stomatitis (Aphthosis)

- Classify disease into simple versus complex

- Simple aphthosis
  - Amlexanox paste 5 g (Aphthasol); apply to ulcers after meals and at bedtime
  - Fluocinonide 0.05% gel/cream 60 g
    - Apply to early lesions after meals and at bedtime.
    - Do not apply to ulcers.
  - Compounded rinse option 1
    - Diphenhydramine parenteral (or 12.5 mg/5 mL non-alcoholic elixer) 200 mg, viscous lidocaine 90 mL, Maalox suspension 90 mL, distilled water 180 mL
    - Rinse 5 mL—expectorate 4–6 times daily.
  - Compounded rinse option 2
    - Dexamethasone (10 mg/mL) 10 mL, diphenhydramine 200 mg, viscous lidocaine 60 mL, Maalox suspension 85 to 275 mL
    - Rinse 5 mL—expectorate 3–5 times daily.

- Complex aphthosis
  - Laboratory evaluation for “correctable causes”: CBC, red blood cell folate, serum ferritin, serum vitamin B₁₂, serum iron studies, serum zinc
  - Topical therapy as for simple aphthosis
  - Systemic therapy for severe, painful, chronic complex aphthosis
    - Prednisone 5 mg tablets #40
      - Take each morning with breakfast for 8 d 8-8-6-6-4-4-2-2 mg, stop
      - Will shorten the course of an individual episode but not change the natural history of the disease
    - Colchicine 0.5 mg tablets
      - Take 1 each morning with breakfast for 1 wk; if tolerated, increase to 2 tablets each morning
      - May suppress disease activity
    - Pentoxifylline (Trental) 400 mg tablets; 1 tablet 3 times/d with meals
    - Dapsone 25 mg tablets
      - Check baseline CBC, liver function tests, urinalysis and glucose-6-phosphate dehydrogenase enzyme level before treatment.
– Take each morning with breakfast, 1 × 3 d, 2 × 3 d, 3 × 3 d, 4 × 7 d, and 5 × daily thereafter
– Check CBC and liver function every month for 3 mo, then every 3 mo thereafter.
– Use for long-term control of disease.

**Recurrent Herpes Simplex Labialis or Stomatitis**

- **Topical therapy**
  - Penciclovir cream (Denavir) 1% 1.5 g tube; apply at the onset of symptoms every 2 h × 4 d
  - Docosanol cream (Abreva) 10%; apply topically at the onset of symptoms q2–3h 5 times daily
  - Acyclovir ointment 5% 3 g tube; apply at the onset of symptoms 6 times daily × 7 d

- **Systemic therapy**
  - Acyclovir 200 mg tablets #35
    - 1 tablet 5 times daily × 7 d
    - Start medication with premonitory symptoms to shorten the course of the episode.
  - Acyclovir 200 mg tablets
    - 3 tablets daily to prevent reactivation in bone marrow transplant recipients

**Sjögren’s Syndrome**

- **Topical therapy**
  - Moisten mouth with cool water or ice chips.
  - Avoid alcohol-containing mouth rinses.
  - Avoid drugs that produce xerostomia.
  - Limit caffeine intake.
  - Use Vaseline on lips at night (a thin coating).
  - Drink milk with meals

- **Saliva substitutes**
  - Liquid, tablet, or gel forms
  - Available over the counter

- **Systemic therapy**
  - Pilocarpine (Salagen) 5 mg tablets #100; take 1 tablet 3 times daily
  - Cevimeline capsules (Evoxac) 30 mg capsules #100; take 1 capsule 3 times daily
Stevens-Johnson Syndrome
• Topical therapy (compounded rinses)
  • Option 1
    – Diphenhydramine 200 mg, viscous lidocaine 90 mL, Maalox suspension 90 mL, distilled water 180 mL
    – Swish 5 mL for 2 min and expectorate 3–4 times/d
  • Option 2
    – Dexamethasone 100 mg, viscous lidocaine 60 mL, diphenhydramine 200 mg, sorbitol 15 mL, Maalox suspension to 275 mL
    – Swish 5 mL for 2 min and expectorate 3–4 times/d
• Systemic therapy
  • Prednisone 5 mg tablets #80
    – Take each morning with breakfast for 16 d as 8/d × 4 d, 6/d × 4 d, 4/d × 4 d, 2/d × 2 d, stop
    – Will reduce disease activity as topical corticosteroids or systemic NSAIDs are started
  • Acyclovir 200 mg tablets #42 (if triggered by herpes simplex virus infection); 1 tablet every 4 h for 7 d or 1 tablet bid-tid as prophylaxis

Tuberculosis
• Systemic therapy (prolonged treatment with at least 2 drugs)
  • Isoniazid 300 mg daily × 6 mo
  • Rifampin 450–600 mg daily × 6 mo
  • Ethambutol 15 mg/kg daily for first 2 mo
  • Pyrazinamide 1.5–2.5 mg/kg for first 2 mo

Wegener's Granulomatosis
• Systemic therapy
  • Sulfamethoxazole/trimethoprim (Bactrim DS) Septra DS 1 twice daily
  • Prednisone 1 mg/kg daily
  • Cyclophosphamide

Zoster
• See “Herpes Zoster.”
White Lesions


مرکز خدمات فرهنگی سالگان
ارائه کننده کتاب و گزینه‌های تخصصی پزشکان

همگام با توسعه علمی و فرهنگی جهان محترم و استفاده روزافزون کامپیوتر در بین جامعات بشری، خصوصاً رشته‌های مختلف علم و استفاده به‌پهنه‌ای از آنها یافته‌ها یکی از آنها این بافتها در قالب نسخه‌های پزشکی تهیه شده‌اند که و VHS، DVD، VCD، eBook، و ... دائماً بر اساس کتاب یا کتاب‌هایی می‌باشد که گزینه‌هایی کامپیوتر علوم سختی و نرم‌سختی بجای وارد نموده و این استرشاد ما در این راه باشد.

لذا علاقهمندان می‌توانند برای دریافت هر یک از محصولات ارتقاء‌های یا از مرکز خدمات فرهنگی سالگان واریز و پس‌باز از فاکس فیش‌های ویا تقسیم‌بندی نسبت به الجزایر، مراکز و پزشکان کالایی مورد نظر خود اقدام نمایند. لازم به ذکر است فقط به سفارشاتی که جهت فرد سفارش به خدمات فوق ذکر واریز شده ترکیب آنها در داده‌ها تولید خواهد شد. لذا خواهشمند است از واریز و به هر گونه حساب دیگری اکیدا خودداری فرمایید.

بازهم در این مرکز کتاب‌های وابسته به نشان نصب مراحم بین واگذاری و ۱۹۹۷-۲۰۰۱ تکمیل و حاصل تمایل.

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۱- رادیولوژی
### 21.1 Computed Body Tomography with MRI Correlation

*Joseph K. T. Lee, Stuart S. Sagel, Robert J. Stanley, Jay P. Heiken* (3rd Edition) *(Lippincott Williams & Wilkins)*

### 21.2 CT Teaching Manual

*Matthias Hofer* (Thieme) *(Saleken E-Book)*

### 23.1 Diagnostic Imaging Expert (A CD-ROM Reference & Review)

*Ralph Weissleder, Jack Witterberg, Mark J. Rieumont, Genevieve Bennett*

2000

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CD Imaging .......................... 21.1

*Computed Body Tomography with MRI Correlation*

Diagnosis Imaging (A CD-ROM Reference & Review) *(Ralph Weissleder, Jack Witterberg, Mark J. Rieumont, Genevieve Bennett)*


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### 24.1 Diagnostic Ultrasound: A Logical Approach

*(John P. McGahan, Barry B. Goldberg)*

#### 25.1 Diagnostic Ultrasound of Fetal Anomalies: Principles and Techniques (CD I,II)

*Principles and Techniques*

#### 26.1 Digital Human Anatomy and Endoscopic Ultrasonography

*(Manoop S. Bhutani, Md, John C. Deutschn, Md)* *(Saleken E-Book)*

#### 27.1 EBUS (Endobronchial Ultrasound)

#### 28.1 Endoscopy and Gastrointestinal Radiology

*(Gregory G. Ginsberg, Michael L. Kochman)*

#### 28.2 Endoscopic Retrograde Cholangiopancreatoigraphy

*(Gregory G. Ginsberg, Michael L. Kochman)*
29.1 Essentials of Radiology

Case 1 CD مطرح‌شده در این بخش موضوع فارغ‌نمایی تا مطابق گرفتن در این بر حسب موضوع فارغ‌نمایی تا مطابق گرفتن در این

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Magnetic Resonance Imaging in Orthopedics and Sport Medicine (David W. Stoller)

In the first edition of this book, MRI was a new and exciting technology that had just begun to revolutionize the way we approach musculoskeletal imaging. Since then, MRI has become an indispensable tool in the evaluation of patients with musculoskeletal disorders. This second edition of the book reflects the rapid growth and development of MRI technology over the past decade. It includes new chapters on MRI of the spine, knee, ankle, shoulder, elbow, and hand, as well as updated and expanded sections on MRI of the hip, foot, and other joints. The book also covers new imaging techniques such as functional MRI and diffusion-weighted imaging, which are now routinely used in clinical practice.

Mammography Diagnosis and Intervention (Ralph S. Smathers, M.D.)

This book provides a comprehensive overview of mammography, from basic principles to advanced techniques. It covers the technology, physics, and clinical applications of mammography, as well as the interpretation of mammograms. The book also includes chapters on mammography in special populations, such as younger women and women with dense breast tissue. The book is designed to be a practical guide for radiologists and other healthcare professionals who perform mammograms.

MR Angiography Thoracic Vessels (O. Ratib & D. Didier)

This book is a comprehensive guide to MR angiography of the thoracic vessels. It covers the basics of MR angiography, including the physics and pulse sequences, as well as advanced topics such as flow-sensitive techniques and contrast-enhanced imaging. The book also includes detailed case studies to illustrate the clinical applications of MR angiography.

MR Imagin Expert (Geir Torhim, Peter A. Rinck) 4th Edition

This version is a special adaptation for "Magnetic Resonance in Medicine: The Basic Textbook of the European Magnetic Resonance Forum" and includes new chapters on the use of MRI in clinical practice. The book covers the basics of MRI technology and physics, as well as advanced topics such as functional MRI, diffusion-weighted imaging, and MR spectroscopy. It also includes case studies to illustrate the clinical applications of MRI.

MRI of the Brain & Spine (Scott W. Atlas) (Lippincott-Roven)

This book is a comprehensive guide to MRI of the brain and spine. It covers the basics of MRI technology and physics, as well as advanced topics such as functional MRI, diffusion-weighted imaging, and MR spectroscopy. The book also includes detailed case studies to illustrate the clinical applications of MRI.

Normal Findings in CT and MRI (Torsten B. Moeller, Emil Reif) (Thieme)

This book provides a comprehensive overview of normal findings in CT and MRI. It covers the basics of CT and MRI technology and physics, as well as advanced topics such as functional MRI, diffusion-weighted imaging, and MR spectroscopy. The book also includes detailed case studies to illustrate the clinical applications of CT and MRI.
The basics of Performing and Interpreting Ultrasound Scans (Matthias Hofer) (With the collaboration of Tatjana Reihs) (Thieme)

VOXEL-MAN 3D-Navigator Brain and Skull (Regional, Functional, and Radiological Anatomy) (IMDM university Hospital Eppendorf, Hamburg) (Springer)

VOXEL-MAN 3D-Navigator Inner Organs (Regional, Systemic and Radiological Anatomy) (IMDM university Hospital Eppendorf, Hamburg)

Whole Body Computed Tomography (Second Edition) (Otto H. Wegener) (Blackwell Science)

Advanced Rhinoplasty Techniques Cosmetic Rhinoplasty (Rollin K. Daniel, M.D.)

Atlas D'ORL Realise avec la collaboration des (Dr Michel Boucherat, Dr Jean-Robert Blondeau)
5.2 Atlas of Head & Neck Surgery Otolaryngology (TEXTBOOK) (Byron J. Bailey, Karen H. Calhoun, Amy R. Coffey, J. Gail Neely)

1- Atlas:

- Head & Neck Surgery:
  - Salivary Gland
  - Nose & maxilla
  - Oral Clarity
  - Ear
  - Neck & Larynx
  - Thyroid & Parathyroid

- Otolologic procedures:
  - Middle Ear and Oscicular Chain
  - Tran temporal Skull Base
  - Congenital Aural Base

- Plastic & Reconstructive Surgery:
  - Laryngoplasty, Rhytidectomy, Rhinoplasty
  - Mandibular Surgery, Local & Regional Flaps

- Pediatric and General Otolaryngology:
  - Frontal Sinus
  - Nasal Polyectomy
  - Ton Sillctomy

2- Bilbo Med Medline:

3- Head & Neck Surgery:
- Textbook
- Drug Reference

4- Facial Plastic Reconstructive Surgery

5.3 Causes of FAILURE in STAPES SURGERY (VCD I) (Howard P. House, TED N. Steffen)

5.4 STAPEDECTOMY (Prefabricated Wire-Loop and Gelfoam Technique) (VCD II)

5.5 Chirurgia Endoscopica Dei Seni Paranasali (A Cura di E. Pasquini G. Farneti)

5.6 Color Atlas of Diagnostic Endoscopy in Otorhinolaryngology (EIIY YANAGISAWA, MD)

5.7 Cobblation Assisted Tonsillectomy (CAT) — Cobblation Assisted Procedures (VCD) (CD I , II)

6.2 Atlas of Rhinoplasty Open and Endonasal Approaches (Gilbert Aiach, M.D.)

1- Atlas:

- Head & Neck Surgery:
  - Salivary Gland
  - Nose & maxilla
  - Oral Clarity
  - Eye
  - Neck & Larynx
  - Thyroid & Parathyroid

- Otolologic procedures:
  - Middle Ear and Oscicular Chain
  - Tran temporal Skull Base
  - Congenital Aural Base

- Plastic & Reconstructive Surgery:
  - Laryngoplasty, Rhytidectomy, Rhinoplasty
  - Mandibular Surgery, Local & Regional Flaps

- Pediatric and General Otolaryngology:
  - Frontal Sinus
  - Nasal Polyectomy
  - Ton Sillctomy

2- Bilbo Med Medline:

3- Head & Neck Surgery:
- Textbook
- Drug Reference

4- Facial Plastic Reconstructive Surgery

1- Basic Science / General Medicine

2- Otolaryngology

3- Otology

4- Facial Plastic Reconstructive Surgery

10. Cobblation Assisted Tonsillectomy (CAT — Cobblation Assisted Procedures (VCD) (CD I , II)

1- Subtotal Cobblation Assisted tonsillectomy

2- Lop — off “CAT” technique

3- Cobblation Assisted tonsilectomy

4- Cobblation channeling of the inferior turbinate

5- Cobblation channeling of the Soft palate

6- Cobblation channeling of the tonsil
Coblation Assisted Tonsillectomy (CAT)

DALLAS RHINOPLASTY  
Nasal Surgery by the Masters (Reducing Tip Projection and Nostril Show Via the Open Approach) (CD I, II)

11.2 VCD: 1

1) Cadaveric Rhinoplasty Dissection Technique
A. Closed endonasal approach
- Intracartilaginous (IC) incision
B. Cartilage delivery technique
- Intracartilaginous incision
C. Open Rhinoplasty approach
- Transcolumnellar incision

2) Tip Alteration
A. Columellar Stat placement
B. Controlling dome angulation and tip defining points
- Intermodal sutures
- Transdomal Sutures
C. Correction ofalar pinching/notching
- Lateral crural strut grafts
- Alar contour grafts
D. Tip grafts
- Infratip graft
- Onlay tip graft

3) Spatial reconstruction
A. Septal reconstruction
B. Inferior turbinate resection (Submucosal)
- Septal resection
C. Modification of the dorsum
- Component dorsum reduction
- Spreader graft placement

4) Osteotomies
A. Medial Osteotomy
B. Lateral Osteotomy
C. External Osteotomy

5) Adjunctive techniques/Closure
A. Alare base resection
- Correction ofalar flaring
B. Closare
C. Spilts

Endoscopic Sinus Surgery (SALEKAN-eBook)

14.2 Diseases of the Sinuses Diagnosis and Management (Darid W. Kennedy, MD, FRCSI, William E. Bolger, MD, FACS, S. James Zinreich, MD)

15.2 Endoscopic Sinus Surgery (SALEKAN-eBook)
16.2 **ENDONASAL SINUSECTOMY WITH CORRECTION OF THE NASAL CAVITY** *(Rikio Ashikawe, Takashi Ohmae, Toshio Ohnisshi, Yutaka Uchida)*

The Endonasal sinusectomy with correction of the nasal cavity *(Takahash's method)* is carried out in seven steps.

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17.2 **Endoscopic Sinus Surgery** NEW HORIZONS *(Nikhil J. Bhatt, M.D.)*

18.2 **EVIDENCE-BASED OTITIS MEDIA** *(Richard M. Rosenfeld, MD, MPH, Charles D. Bluestone, MD)*

...
29.2 کتاب الکترونیکی مشتمل بر 42 صفحه، کتاب کامل و کاربردی در تام مباحث جراحی پلاستیکی می‌باشد. این کتاب به مثابح فلسطینی به جراحی پلاستیکی در تمام سطوح آورش و درمان پزشکی می‌پردازد و بسیاری از استفاده‌های متخصص را در جراحی پلاستیکی در این کتاب نشان می‌دهد.


30.2 رینوپلاستی یک روش کلیه در پایان کاری که محاسباتی شامل جراحی قاب‌زی و انجام تجزیه‌کاری‌های متنوعی می‌باشد. این کتاب در این مباحث واقعیت‌ها و اینکه در این کتاب به گونه‌هایی در حال تغییر است که در این کتاب نشان داده شده است.


31.2 رینوپلاستی یک روش کلیه در پایان کاری که محاسباتی شامل جراحی قاب‌زی و انجام تجزیه‌کاری‌های متنوعی می‌باشد. این کتاب در این مباحث واقعیت‌ها و اینکه در این کتاب نشان داده شده است.


32.2 رینوپلاستی یک روش کلیه در پایان کاری که محاسباتی شامل جراحی قاب‌زی و انجام تجزیه‌کاری‌های متنوعی می‌باشد. این کتاب در این مباحث واقعیت‌ها و اینکه در این کتاب نشان داده شده است.


33.2 رینوپلاستی یک روش کلیه در پایان کاری که محاسباتی شامل جراحی قاب‌زی و انجام تجزیه‌کاری‌های متنوعی می‌باشد. این کتاب در این مباحث واقعیت‌ها و اینکه در این کتاب نشان داده شده است.


34.2 رینوپلاستی یک روش کلیه در پایان کاری که محاسباتی شامل جراحی قاب‌زی و انجام تجزیه‌کاری‌های متنوعی می‌باشد. این کتاب در این مباحث واقعیت‌ها و اینکه در این کتاب نشان داده شده است.
### 4.3 Advanced Therapy of BRST DISEASE (S. Eva Singletry, MD, Geoffrey L. Robb, MD)

2000

### 5.3 Active Management of Labour (Kieran O'Driscoll, Declan Meagher) (SALEKAN E-BOOK)

2004

### 6.3 American Cancer Society Atlas of Clinical Oncology (Cancer of the Female Lowe Genital Tract) (Patricia J. Eifel, M.D. Charles Levenback, M.D.) (SALEKAN E-BOOK)

2001

- Cervix
- Chemotherapy in Curative Management
- Surgery for Vulvar Cancer
- Surgical Treatment of Invasive Cervical Cancer
- Diagnostic Imaging
- Epidemiology
- Post-treatment Surveillance
- Radiation Therapy for Vulvar Cancer
- Radiation Therapy for Invasive Cervical Cancer
- Screening for Neoplasms
- Pathology
- Palliative Care
- Acute Effects of Radiation Therapy
- Radical Management of Recurrent Cervical Cancer
- Treatment of Squamous Intraepithelial Lesions
- Molecular Biology
- Late Complications of Pelvic Radiation Therapy
- Management of Vaginal Cancer
- Invasive Carcinoma of the Cervix
- Anatomy and Natural History

### 7.3 Atlas of Clinical oncology Breast Cancer (American Cancer Society) (David J Winchester, MD, David P Winchester, MD)

2000

- Genetics, Natural History, and DNA-Based Genetic Counseling in Hereditary Breast Cancer
- Breast Cancer Risk and Management: Chemoprevention, Surgery, and Surveillance
- Screening and Diagnostic Imaging
- Imaging-Directed
- Breast Biopsy
- Histopathology of Malignant Breast Disease
- Unusual Breast Pathology
- Prognostic and Predictive Markers in Breast Cancer
- Surgical Management of Ductal Carcinoma In Situ
- Evaluation and Surgical Management of Stage I and II Breast Cancer
- Locally Advanced Breast Cancer
- Breast Reconstruction

### 8.3 ATLAS OF ENDOSCOPIC TECHNIQUES IN GYNECOLOGY (First Edition) (Jeffrey M. Goldberg, MD, Tommaso Falcone, MD) (©W.B. Saunders, Philadelphia)

2001

1. Instrumentation and Pelvic Anatomy
2. Surgery for Pelvic Support
3. Ovarian Surgery
4. Hysteroscopic Surgery
5. Patient Preparation
6. Surgery for Endometriosis and Pelvic Pain
7. Complications
8. Tubal Surgery
9. New Procedures
10. Uterine Surgery


2001

- Pelvic sling in the treatment of stress incontinence
- Fibro-fatty labial flap (Martius Flat) for vaginal reconstruction
- Transvaginal hysterectomy for severe prolapse
- Transvaginal repair of grade IV cystocele

### 10.3 Atlas of Transvaginal Surgery (Second Edition) (©W.B. Saunders, Philadelphia) (VCD)

2001

- Transvaginal repair of enterocoele and vault prolapse
- Transvaginal repair of vesico-vaginal fistula using a peritoneal flap
- Transvaginal repair of posterior vaginal wall prolapse

### 11.3 COLPOSCOPY an Interactive CD-ROM (Thomas V. Sedlacek, MD, Charles J. Dunton, MD)

2003

- Male impotence
- Female impotence
- Male impotence
- Female impotence

### 12.3 Core Curriculum in Primary Care Patient Evaluation for Non-Cardiac Surgery and Gynecology and Urology (Michael K. Rees, MD, MPH)

2001

- Anatomical classification of the urinary bladder
- Urethral sphincter function
- Female impotence
- Male impotence

### 13.3 Core Curriculum in Primary Care Gynecology (Michael, Isaac Schiff, Keith, Thomas, Annekathryn)

2003

- Anatomical classification of the urinary bladder
- Urethral sphincter function
- Female impotence
- Male impotence

### 14.3 Danforth's Obstetrics and Gynecology (James R. Scott) (9 Edition) (SALEKAN E-BOOK)

2003

- Anatomical classification of the urinary bladder
- Urethral sphincter function
- Female impotence
- Male impotence
Left Salpingectomy (Film: LMP)

Limiting Physician Exposure to Hepatitis B and HIV: Ob / Gyns (R. Viscarello, MD)

Laparoscopic Retropubic Colposuspension For Stress urinary incontinence (Gordon, D. Davis, MD, & R.W. Lobel, MD)

Bi-polar Desication of Vascular Tissue: Laparoscopic Hysterectomy (Paul, D. Indman, MD)

TEXT AND ATLAS OF Female in Fertility Surgery (Robert B. Hunt) (Third Edition) (Mosby)

Triplet Pregnancies and their Consequences (Louis G. Keith, MD, Isaac Blickstein, MD) (SALEKAN E-BOOK)

Urogynecology: Evaluation and Treatment of Urinary Incontinence (Bruce Rosenzweig, MD, Jeffrey S. Levy, MD, Donald R. Ostergard, MD)

TVT Tension-free Vaginal – Tape


27.3 Triplet Pregnancies and their Consequences (Louis G. Keith, MD, Isaac Blickstein, MD) (SALEKAN E-BOOK)

28.3 TVT Tension-free Vaginal – Tape

29.3 Urogynecology: Evaluation and Treatment of Urinary Incontinence (Bruce Rosenzweig, MD, Jeffrey S. Levy, MD, Donald R. Ostergard, MD)
روش‌های جراحی: ابتدا در مورد روش‌های انجام جراحی بحث شده و سپس Procedure اعمال جراحی شرح داده شده است. در قسمت‌های بعدی مقایسه مجدد موقعیت روش‌ها ذکر شده و در آخر Complication این روش‌ها توضیح داده شده است.

incontinence management to private patients • Non surgical therapy • urogynecology as a subspecialty • in mobilisation of private patients • professional considerations • sexuality

30.3 Video Journal of Gynecology (Vaginal Hysterectomy) Wedge morcellisation Technique for the Large Uterus) (The Infertile Couple) (David Olive, MD, George W. Morley MD)

31.3 WOMEN'S HEALTH (MOSBY'S PRIMARY CARE)

32.3 UTEROSALPINGOGRAPHY IN GYNECOLOGY (Hysterosalpingography) It's Application in Physiological And Pathological Conditions (SALEKAN E-BOOK)

33.3 Your Pregnancy, Your Newborn The Complete Guide for Expectant and New Mothers (SALEKAN E-BOOK)
Diagnostic Testing | Blood Studies | Urine Studies | Stool Studies
---|---|---|---
Chemistry Studies | Microbiologic Studies | Immunodiagnostic Studies | Nuclear Medicine Studies
Cytology, Histology, and Genetic Studies | Endoscopic Studies | Ultrasound Studies | Pulmonary Function and Blood Gas Studies
Prenatal Diagnosis and Tests of Fetal Well-Being | Cerebrospinal Fluid Studies | X-ray Studies | Special Systems, Organ Functions, and Post Mortem Studies

2.4 A Slide Atlas of Atherosclerosis (Progression and Regression) (Herbert C. Stary)

2002

3.4 American Society of Hematology (CD 1-5) (44th Annual Meeting)

CD-1: ALL - AML - ASH/ASCO Joint Symposium - Atypical Cellular Disorders
CD-2: CLL - CML - CNS Lymphoma - Cutaneous Lymphoma - E. Donnall Thomas Lecture
CD-3: Enhancing Physician/Patient Communication Regarding Hematologic Disorders - Ham-Wasserman Lecture - Hematology Grants Workshop
CD-4: Myeloma - Myelodysplastic Syndromes Non-Myeloablative Transplantation - Platelets: Thrombotic Thrombocytopenic - Purpura: Plenary Policy Forum
CD-5: Presidential Symposium - Red Cell Antigens as Functional Molecules and Obstacles to Transfusion - Sickle Cell Disease - Stem Cell Transplantation: Supportive Care and Long-Term Complications - Stem Cells: Hype and Reality Update on Epidemiology and Therapeutics for Non-Hodgkin’s Lymphoma

4.4 An Electronic Companion to Microbiology for Majors™ (Mark L. Wheelis) Review, Test Yourself

What Are Microorganisms? | Methods of Microbiology | Eukaryotic Cell Structure | Metabolism & Energy | Gene Regulation | Microbial Ecology | Disease
---|---|---|---|---|---|---
Classification | Prokaryotic Cell Structure | Growth & Reproduction | Microbial Genetics | Viruses | Defenses Against Infection

5.4 Atlas of Hematology


6.4 Atlas of Surgical Pathology (Johns Hopkins) (Jonathan I. Epstein, Neera P. Agarwal-Antal, David B. Danner, Kim M. Ruska)

2003

7.4 Atlas of Medical Parasitology (Dr. K. Ghazvini)

---|---|---|---|---|---|---

8.4 Basic histology: TEXT & ATLAS IMAGE LIBRARY (Tenth Edition) (Luiz Carlos, Juqueirita, Jose CARNEIRO) (A Division of The McGraw-Hill Companies) (Version 1.02)

2000

9.4 Biochemical Interactions An electronic companion to: FUNDAMENTALS OF BIOCHEMISTRY (Donald voet, Judith G. voet, charlotte W. Pratt) (Version 1.02)

1999
<table>
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<tr>
<th>Title</th>
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<th>Edition</th>
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<tr>
<td>BRS Cell Biology</td>
<td>Leslie P. Gartner, James L. Hiatt, Judy M. Strum</td>
<td>Fourth Edition</td>
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<tr>
<td>Clinical Hematology</td>
<td>Victor Hoffbrand, John E Pettit</td>
<td>Second Edition</td>
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<td>Common Problems in Clinical Laboratory Management</td>
<td>Judith A. O'Brien, M.S. CLS(C)</td>
<td>Saleken E-Book</td>
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### Biology Concepts & Connections (Second Edition)

- Introduction: The Scientific Study of Life
- The Life of the Cell
- Cellular Reproduction & Genetics
- Concepts of Evolution
- The Evolution of Biological Diversity
- Animals: Form & Function
- Plants: Form & Function
- Ecology

### Principles and Practice of Hematology (Second Edition)

- Part I: Fundamentals of Hematology: Tools of the trade
- Part II: The Hematopoietic System
- Part III: Stem Cell Disorders
- Part IV: White Blood Cells
- Part V: Hemostasis
- Part VI: Red Blood Cells
- Part VII: Systemic Disease
- Part VIII: Hematologic Therapies
- Part VIII: Appendices

### BRS Cell Biology

- Plasma Membrane
- Nucleus
- Cytoplasm
- Extracellular Matrix
- Connective Tissue
- Cartilage and Bone
- Muscle
- Nervous Tissue
- Circulatory System
- Lymphoid Tissue
- Endocrine System
- Skin
- The Urinary System
- Female Reproductive System
- Digestive System: Oral Cavity and Alimentary Tract
- Special Senses
- Epithelia and Glands
- Blood and Hemopoiesis
- Digestive System: Glands
- Comprehensive Exam

### Clinical Hematology

- Normal Hemopoiesis and Blood Cells
- Leucocyte Abnormalities
- Hemostasis and Bleeding Disorders
- Bone Marrow Transplantation
- Parasitic Infections Diagnosed in Blood
- Anaemias
- Hematological Malignancies
- Coagulation Disorders
- Bone Marrow in
- Blood Transfusion
- Further Reading
- Acknowledgements
- Non-hemopoietic Disease

### Clinical Immunology

- Encouraging Education
- The Acquisition and Maintenance of Laboratory Instrumentation
- Mastering Finances: Billing and Coding
- TAMING TECHNOLOGY: POINT OF CARE TESTING (POCT)

---

لطفاً، تلاش کنید کتاب و سیدی‌های تخصصی پزشکی‌ها را با کتابخانه مالکه سالگان ملاقات کنید.
21.4 Concise Histology (A data of multiple choice question in microscopic) (Bloom & Fawcett's) (Second Edition) 17.4

18.4 Dianostic Hematology
This textbook, Diagnostic Hematology: A pattern approach, is accompanied by a CD-ROM with three knowledge-based systems applied to 237 case studies. The 3 knowledge-based systems are:
1. Professor Petrushka for peripheral blood analysis
2. Professor Fidelio for flow cytometry immunophenotyping
3. Professor Belmonte for bone marrow interpretation

19.4 Discover Biology


21.4 EMBRYO (CD Color Atlas for Developmental Biology) (Gary C. Schoenwolf)
Chapter 1: Frog Embryos
Chapter 2: Chick Embryos
Chapter 3: Pig Embryos
Chapter 4: Gametogenesis

22.4 Essential Cell Biology (with the voice of Julie Theriot designed and programmed by Christopher Thorpe)

23.4 Fields Virology (Forth Edition) (Volume 1) (Lippincott Williams & Wilkins)
Section One: General Virology
Chapter 1-22
Specific Virus Families
Chapter 23-90

24.4 Genetics From Genes to Genomes (Ann Reynolds, Ph.D.)
University of Washington

25.4 Histology EXPLORER

26.4 Gram Stain TUTOR (ANINTERACTIVE TUTORIAL THAT TEACHES THE MICROSCOPIC EXAMINATION OF URINARY SEDIMENT)

1. Introduction
2. Morphology
3. Specimen Sites
4. Case Studies
5. Exam
6. Image Atlas

27.4 HISTOLOGY EXPLORER

28.4 HUMAN HISTOLOGY CD-ROM (Alan Stevens. James Lowe)

29.4 Images of Disease An image database for the teaching of Pathology (Nick Hawkins, Mark Dziegielewski)

30.4 Immunology (Blackwell Science)


32.4 Interactive Embryryo Program The Human Embryo Program (Jay Lash Ph.D.)

33.4 Laboratory Medicine: URINALYSIS (Chemical and microscopic examination of urine Atlas of Microscopic Analysis Procedures for Urinalysis) (Pesce Kaplan Publishers Inc.)
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<td>34.4</td>
<td>Media Supplement for Biochemistry (FOURTH EDITION)</td>
<td>2000</td>
<td>Mosby</td>
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<td>35.4</td>
<td>Microbes in Motion III (Dr. Gloria Delisle and Dr. Lewis Tomalty Queen's University)</td>
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<td>36.4</td>
<td>MICROBIOLOGY AND IMMUNOLOGY (KEN S. ROSENTHAL)</td>
<td>2002</td>
<td>Mosby</td>
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<td>37.4</td>
<td>MICROBIOLOGY AND MIcrobial Infections (Topley &amp; Wilson's) (Albert Balous, Max sussman)</td>
<td>NINTH EDITION</td>
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<td>38.4</td>
<td>MODERN GENETIC ANALYSIS</td>
<td>1999</td>
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<td>39.4</td>
<td>MOLECULAR CELL BIOLOGY 4.0</td>
<td>2000</td>
<td>Mosby</td>
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<td>40.4</td>
<td>NCCL INFOBASE</td>
<td>2002</td>
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<td>41.4</td>
<td>PATHOLOGIC BASIS OF DISEASE Interactive Case Study Companion to ROBBIMS (W. B. Saunders Company)</td>
<td>Sixth Edition</td>
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<td>PATHOLOGY (Alan Stevens. James Lowe)</td>
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<td>Peripheral Blood TUTOR (ANINTERACTIVE TUTORIAL THAT TEACHES THE MICROSCOPIC EXAMINATION OF URINARY SEDIMENT)</td>
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<td>44.4</td>
<td>PRINCIPLES OF Molecular Virology (THIRD EDITION)</td>
<td>2000</td>
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<td>45.4</td>
<td>RAPID REVIEW HISTOLOGY AND CELL BIOLOGY (E. ROBERT BURNS, M. DONALD CAVE) (MOSBY)</td>
<td>SIXTH EDITION</td>
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<td>46.4</td>
<td>Samter's Immunologic Diseases (K. Frank Austen, M.D, Michael F. Frank, M.D., John P. Atkinson, M.D., Harvey Cantor, M.D.)</td>
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</table>
The Cell 1.0: A Molecular Approach (Many Animations, Movies, Photos, and drawn images) (Geoffrey M. Cooper) 1999

UNDERSTAND! Biochemistry (3/e Version) (Lehninger Principles of Biochemistry) 2000

UNDERSTAND! Biochemistry (VERSION 1.0) 1999

Urinalysis TUTOR (AN INTERACTIVE TUTORIAL THAT TEACHES THE MICROSCOPIC EXAMINATION OF URINARY SEDIMENT) (Carla M. Phillips, MLM, MT(ASCP), Paul J. Henderson, MS, MT(ASCP), Claudia Bein, BS, MT(ASCP))

A Slide Atlas of ATHEROSCLEROSIS Progression and Regression (Herbert C. Stary, MD) 2002

A visible improvement in angina treatment (VCD)
2.5 ACCSAP (Adult Clinical Cardiology Self-Assessment Program) (C. Richard Doni, MD, Richard P. Lewis, MD) (AMERICAN COLLEGE of CARDIOLOGY) 2000
3.5 Acute Heart Failure (THE CLEVELAND CLINIC FOUNDATION) (W. Frank Peacock, MD) (The Emergency Department and the Economics of Care) 2004
4.5 American Heart Associations fighting Heart Disease and Stroke Abstracts from Scientific Sessions (Augustus O. Grant, Raymond J. Gibbons) 2002

5.5 Atlas of Transesophageal Echocardiography (Navin C. Nanda, MD, Michael J. Domanski) (Williams & Wilkins)
1. Normal Anatomy
2. Prosthetic Valves and Rings
3. Mitral Valve
4. Ischemic Heart Disease
5. Aortic Valve and Aorta
6. Cardiomyopathy
7. Tricuspid and Pulmonary Valves
8. Congenital Heart Disease

6.5 BEYOND HEART SOUNDS The Interactive Cardiac Exam (John Michael Criley, MD) (VOL 1)

7.5 Cardiac Catheterization, Angiography, and Intervention (SIXTH EDITION) (LIPPINCOTT WILLIAMS & WILKINS) 2000

8.5 Cardiovascular Surgery (VCD) (CD I, II, III) Excerpted from "Medical & Surgical Controversies in CV disease: The Aorta and Peripheral Vessels" Course Directors: Thoralf M. Sundt III, MD and Peter C. Spittell, MD 2004

9.5 Carotid Artery Stenting (Current Practice and Techniques) (Nadim Al-Mubarak, Gary S. Roubin, Siriram S. Laver, Jiri J. Vitek) 2004

10.5 CathSAP Cardiac Catheterization and Interventional Cardiology Self-Assessment Program (Carl J. Pepine, MD, Steven E. Nissen, MD) 2003

11.5 Challenging established treatment patterns in chronic heart failure A Satellite Symposium held during the ESC Heart Failure meeting 2003

12.5 Clinical TRANSESOPHAGEAL ECHOCARDIOGRAPHY (A PROBLEM- ORIENTED APPROACH) (Second Edition) (Steven N. Konstadt) 2003

13.5 Clinical Utility of Contrast Echocardiography Sonovue: An ideal contrast agent for Low MI myocardial Perfusion (Dr. Daniela Bokor, Bracco sa, Milano)

What's new in cardiac echography (Dr. Luciano Agati, University "La Sapienza Roma"

Ischemic coronary artery disease (Dr. Harald Becker, John Radcliffe Hospital, Oxford) 2001
Congestive Heart Failure (NOVARTIS) (CD I , II)

Case report: a Case report and multiple choice test

CHF management

Coronary Heart Disease (J. Hurley Myers, Ph.D., Frank H. Netter, M.D.)

Case report

Drugs for the Heart (Sixth Edition) (Salekan E-Book) (Lionel H. Opie, Bernard J. Gersh)

ECG (Jay W. Mason, MD)

ECG DIAGNOSIS MADE EASY ROMEO VEGHT

ECG-SAP III (Jay W. Mason, MD, FACC)


Echo Lecture (VIDEO SERIES) (7CD) (Mayo)

TEE in the Operating Room (Bijoy K. Khandheria, MD)

Intraoperative echocardiography has become an essential component to the surgical approach to valvular disease. Dr. Bijoy Khandheria discusses the utility of intraoperative echocardiography and its impact on the surgical management of cardiovascular disease.

TEE in Adult Congenital Heart Disease (James B. Seward, M.D.)

Dr. James Seward Presents Adult Congenital Heart Disease. A generation of Have Grown into adulthood and Present with postoperative congenital heart disease. Transesophageal echocardiography is extremely helpful but may not always be necessary in the assessment of adult congenital heart disease. Learn from the expert regarding appropriate use of transesophageal echocardiography and assessment of residua and sequela of adult congenital heart disease.

Understanding Operative Procedures for Patients with Univentricular Heart from Palliation to Fontan (James B. Seward, M.D.)

Dr. Seward gives a detailed overview of complex anomalies and their applicable corrections. Topics included are Blalock, Mustard, Glenn and Fontan corrections. Graphic depictions of each corrective procedure, possible complications and echocardiographic example are included.

Mitril Valve Reguritation: Essential Measurements. Pitfalls and Limitations. (Fletcher A. Miller, Jr, MD)

Dr. Fletcher Miller discusses and presents the current approach to the quantitative evaluation of mitral valve regurgitation. This is an excellent review of current quantitative assessment of mitral valve regurgitation including pitfalls and limitations.

Mitril Valve Reguritation: Evidence-Based Practice (A. Jamil Tajik, MD)

A Classic presentation by Dr. A. Jamil Tajik on a change in clinical practice with regard to the quantitation of regurgitation and then a change in medical management with early surgery and repair of the mitral valve.
6. Evaluating the Patient with Prosthetic Valve (Fletcher A. Miller, Jr., MD)
Dr. Fletcher Miller, an expert on the echocardiographic assessment of prosthetic valves, presents a detailed in-depth review of the quantitative echo Doppler approach to the prosthetic valve. It is important to understand the hemodynamic pitfalls and limitations of the echocardiographic assessment of cardiac prosthetic valves.

7. Stress Echocardiography and Contrast (Patricia A. Pellikka, M.D.)
Stress Echocardiography and Contrast Using illustrative cases, Dr. Pellikka gives an expert presentation and discussion on the role of contrast in stress echocardiography. Pitfalls and limitations of contrast stress echocardiography are also discussed. New Horizons in Stress Echocardiography Dr. Pellikka, an expert in Stress echocardiography, discusses Dobutamine stress echocardiography and its role in preoperative risk stratification. Also discussed are new advances in stress echocardiography such as color kinesis and acoustic quantification, color Doppler imaging, and strain and strain rate imaging.
### 42.5 HEART SOUNDS

**Basic Cardiac Auscultation** Version 3.0 (Leonard Werner, M.D., Brian Pitts, David Gilsdorf)

2003

### 43.5 HEART SOUNDS

**Basic Cardiac Auscultation CD-ROM to Accompany** (M.D., F.A.C.P., Brian Pitts, M.D., David Gilsdorf) (Lippincott Williams & Wilkins)

2003

### 45.5 Highlights ESC Congress

**HURST'S THE HEART** (R. Wayne Alexander, Robert S. Schlant, Valentin Fuster)

2004

### 46.5 Interactive Atlas of Transesophageal Color Doppler Echocardiography

(Raffaele De Simone)

2003

### 47.5 Manual of Cardiovascular Medicine (Second Edition)

(Brian P. Griffin, Eric J. Topol)

2004

### 49.5 Interventional Cardiology Clinical Resource (Disc 1 & 2)

(Evidence . Analysis . Recommendations . Consensus Reports)

University of Vienna, Austria

2003

### 50.5 Highlight

**Intra-Aortic Balloon Catheter Insertion and Removal Technique** (ARROW)

1. INTRODUCTION  2. LAB SELECTION  3. LAB PREPARATION  4. LAB INSERTION  5. LAB CATHETER PREPARATION  6. LAB CATHETER INSERTION  7. LAB REMOVAL

2002

### 51.5 Highlights

**MVP Video Journal of Cardiology** (Maria-Teresa Olivari, M.D., Antonio M. Gotto, M.D., D. Phill.)

2002

### 52.5 Highlights

**MVP Video Journal of Cardiology** (Anthony C. Pearson, M.D., Charles B. Higgins, M.D., William W. O'Neil, M.D.) (VCD)

2002

### 54.5 Movie

**MVP Video Journal of Cardiology** (Maria-Teresa Olivari, M.D., Antonio M. Gotto, M.D., D. Phill.)

2002

### 55.5 Movie

**MVP Video Journal of Cardiology** (Anthony C. Pearson, M.D., Charles B. Higgins, M.D., William W. O'Neil, M.D.) (VCD)

2002
3- Improved understanding of cardioembolic Stroke provided by Transesophageal Echocardiography

Anthony C. Pearson

Majlesi Shodeh: دکتر

تاريخ ترجمه مقالات: این مقاله ترجمه نگرفته است

56.5 MVP VIDEO JOURNAL OF CARDITHORACIC SURGERY

(Patricia M. Applegate, Richard L. Applegate, I)


2003

57.5 Perioperative Transesophageal Echocardiography

2003

59.5 PLUMER'S PRINCIPLES & PRACTICE OF INTERVENTIONAL THERAPY

(SEVEN EDITION) (Sharon M. Weinstein)

2003

60.5 Practical Perioperative Transesophageal Echocardiography

Introduction, instructions and acknowledgements (David Sidebotham, John Faris, Alan Merry, Andrew Kerr)

2002

Textbook of Cardiovascular Medicine

62.5 TEXTBOOK OF CARDIOVASCULAR MEDICINE

(2nd EDITION) (ERIC J. TOPOL)

2003
The Physiological Orgins of HEART SOUNDS and MURMUS

The Physiological Orgins of HEART SOUNDS and MURMUS (John Michael Criley, M.D., Conrad Zalace, David Creley)

Catalog of Lesions

- Normal
- Valvar Lesions
- Pericardial Disease
- Congenital Heart Disease
- Cardiomyopathies
- Myoxma

Vascular Vision (A Liberating Approach to Vascular health Expert Opinions in Dyslipidaemia) (Professor Philip Barter, Dr. John Kastelein, etc.)

Vascular Vision (A Liberating Approach to Vascular health Expert Opinions in Dyslipidaemia) (Professor Philip Barter, Dr. John Kastelein, etc.)

Vascular Vision (A Liberating Approach to Vascular health Expert Opinions in Dyslipidaemia) (Professor Philip Barter, Dr. John Kastelein, etc.)

1. From a new perspective: mitral valve prolapse aortic dissections and aneurysms

2. Surgical and medical management of ascending and descending aortic dissections liporoten (A): a cardiovascular risk factor

3. Laser Angioplasty for coronary Atherosclerotic Disease

Herbert Geschwind
کتب: 
شمار اندیشمندان، نزدیک کسانی‌های بیشماری و طبیعی از کتاب‌های شده است. این کتاب به ۴ قسمت تقسیم شده است:

بخش ۱: Basic Concept
شامل اندیشمندان، نزدیک کسانی‌های بیشماری و طبیعی از کتاب‌های شده است.

بخش ۲: درمان‌های محلی: در این بخش، دستگاه‌های مبلمان (فصل ۳)، درمان‌های ویرانک‌های بیشماری و طبیعی از کتاب‌های شده است.

بخش ۳: Management
شامل تحلیل کتاب‌های ویرانک‌های بیشماری و طبیعی از کتاب‌های شده است.

بخش ۴: در مورد پیشگیری از کسانی‌های بیشماری و طبیعی از کتاب‌های شده است.

۲. ۶ AQUAMIDE: Poly Acryl Amide Ged (ان جی نام علمی: Polysciences) 
توییباثی جاده شده است. در این بخش، از تیم‌های زیادی در این AQUAMIDE می‌باشد. 
در این بخش یکی از این CD به کار رفته اند

۳. ۶ ATLAS OF COSMETIC SURGERY (Michael S. Kaminer, MD, Jeffrey S. Dover, MD, FRCP, Kenneth A. Arndt, MD) (W.B. Saunders Company) 

2002 

PART I 
EVALUATION OF THE COSMETIC SURGERY PATIENT
1. The History of Cosmetic Surgery
2. The History of Cosmetic Dermatologic Surgery
3. Evaluation of the Aging Face,
4. Photoaging: Mechanisms, Consequences, and Prevention
5. Beauty and Society
6. Psychosocial Issues and Their Relevance to the Cosmetic Surgery Patient

PART II 
ANESTHESIA
7. Regional Anesthesia for Aesthetic Surgery
8. Office-Based Sedation and Monitoring
9. Postoperative Pain and Nausea Management

PART III 
COSMETIC SURGERY PROCEDURES AND TECHNIQUES
10. Topical Skin Care
11. Lasers in the Treatment of Vascular Lesions
12. Lasers in the Treatment of Pigmented Lesions
13. Laser Hair Removal
14. Liposuction
15. Hair Transplantation
16. Soft Tissue Augmentation
17. Botulinum A Exotoxin Injections for Photoaging and Hyperhidrosis,
18. Chemical Peels
19. Lasers in Skin Resurfacing
20. Blepharoplasty
21. Surgical Rhinotomomy: Rare and the Endoscopic Forehead Lift
22. Leg Vein Management: Sclerotherapy, Ambulatory Phlebectomy, and Laser Surgery
23. Scar Management: Keloid, Hypertrophic, Atrophic, and Acne Scars

۴. ۶ Atlas of Dermatology (Jhon’s Hopkins) 


۱۹۹۹ 

تاریخچه: تاریخچه درمان‌های بیشماری (uw.edu) 

DOI: Dermatology online Atlas 

نتایج سه شده، در این CD، یک شکل‌گیری در این انتخاب‌های بیشماری و طبیعی از کتاب‌های شده است. از این نمایندگی انتخابی از ۶۰۰ DPO شکل‌گیری در این CD انتخاب در چهار مورد تشخیص‌های بیشماری و طبیعی از کتاب‌های شده است.
7.6 Botulinum Toxin Aesthetic Indications (Mauricio de Maio, Segio Talarico, Benjamin Ascher, Nam Ho Kim South) 2003

8.6 Color Atlas and Synopsis of Clinical Dermatology Common and Serious Diseases Thomas B. (Fitzpatrick, M.D. Richard Allen Johnson, M.D. Dick Suurmond, M.D) —

9.6 COLOR ATLAS OF CLINICAL DERMATOLOGY COMMON AND SERIOUS DISEASES (Salekan E-Book) —


11.6 COSMETIC LASER SURGERY PERFECT THE TECHNIQUES, REDUCE THE RISKS, AND ENJOY THE RESULTS WHEN PERFORMING COSMETIC LASER SURGERY (Richard E. Fitzpatrick Michal P. Goldman) 2000

14.6 Cosmetic Surgery An Interdisciplinary Approach BASIC AND CLINICAL DERMATOLOGY (ALAN R. SHALITA, M.D., DAVID A. NORRIS, M.D) 2001

15.6 COSMETIC SURGERY FOR FACE AND BODY

16.6 Cutaneous Laser Surgery (Second edition) The Art and Science of Selective Photothermolysis (Goldman, Fitzpatrick)
رتبه‌ی هوشمندی از آن به دلیل انتخاب کتاب‌هایی که در وضعیت‌های مصرف مواد مخدر و جراحات کیفیت بالا قرار دارند.

تشخیص و درمان ترکیب‌های بی‌خودی

17.6 Cutaneous Medicine Cutaneous Manifestations of Systemic Diseases (THOMAS T. PROVOST, MD, JOHN A.FLYNN, MD) (Johns Hopkins Medical Institutions Baltimore, Maryland) 2001

18.6 Dermatology: A Multi-Media Teaching File (Disc 1,2) (Gross & Microscopic Symposium) (Mosby) —

19.6 EVIDENCE-BASED DERMATOLOGY (Howard I. Maibach, MD, Sagib J. Bashir, BSc (Hons), MB, CHB, Ann McKibbon, BSc, MLS) 2002

21.6 Hair Removal with Intense Pulsed Light (IPL) (نظریه استفاده از زیرلیزر- زیرلیز، آنتی‌بیوتیکس) —

22.6 HAIR TRANSPLANTATION (The Art of Micrografting and Minigrafting) (Salekan E-Book) 2002

23.6 HANDBOOK OF ORAL DISEASE DIAGNOSIS AND MANAGEMENT (Crips Heal D Hand) (MARTIN DUNITZ) 1999

24.6 Laser Hair Removal (David J. Goldman) (Martin Dunitz) 2000

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hair removal

Management of Facial Lines and Wrinkles (Second Edition) (Salekan E-Book)


Physical Signs in Dermatology (Second Edition) (Clifford M Lawrence, Neil H Cox) (Salekan E-Book)


Radiosurgical Treatment of Superficial Skin Lesions (S. Randolph Waldman, M.D.)

Radiosurgical Vaporization of Dermatologic Lesions (Dr. Stephen Chi Rello)

Reconstructive Facial Plastic Surgery (Salekan E-Book)
REFINEMENT IN HAIR TRANSPLANTATION: Micro and minigraft Megasession

33.6

Refinements in Hair Transplantation: Micro and Minigraft Megasession

2002

Skin Rejuvenation with skin filler (E.A.A. Derm)

34.6

Skin Rejuvenation with skin filler

2002

Textbook of Dermatology (Sixth Editions) (R.H. CHAMPION, J.L. BURTON, D.A. BURNS, S.M. BREATHNACH) (ROOK) (Softcover c Gertion I.T. Consultants Ltd. Version 1.2.0)

36.5

Textbook of Dermatology

1998

The Aging Face A Systematic Approach (Calvin M. Johnson, Jr., Ramsey Alasrawi)

38.6

The Aging Face

2002

Treatment of Skin Disease Comprehensive therapeutic Strategies (Mark G Lewinbuiw R Warren J Heymann, John Beth-Jones, Ian Oranje) (SALEKAN E-BOOK)

39.6

Treatment of Skin Disease

2002
درمان (یا از نظریه، خود خود درمان) یا درمانی که باید بر اساس نوع مطالعات انجام شده در evidence-Based (specific investigations) management strategy (در بالین و مالیه و شرح حال باید به نکات جستجو و د- جدول برای یکی پزشک آزمایش یا پزشکی را درخواست کند)

4- درمان (یا از نظریه، خود خود درمان) یا درمانی که باید بر اساس نوع مطالعات انجام شده در Clinical trial (B) (double blind study) (A) مشخصه (A) - E و Nامگذاری شده است. به عنوان مثال در درمان از درمان که از نظریه، خود خود درمان A و Nامگذاری شده (B) Nامگذاری شده که (A) مشخصه (B) و (A) مشخصه (B) مشخصه (A)

پزشک‌هایی که به پزشکی کمک می‌کنند

- بیماری را برای یکی ۱۲ فرمولیت نو- باعث خاصیت و سایر نو- باعث خاصیت و سایر Nامگذاری شده است. این کتاب شامل ۶۳ بیماری هر هر باعث خاصیت و سایر Nامگذاری شده است. این کتاب شامل ۶۳ بیماری هر هر

40.6 USING BOTULINUM TOXINS COSMETICALLY (Jean Carruthers, Alastair Carruthers)

CD


Segment I: Core Decompression

Segment II: Trauma Case Studies: Retrograde Femoral Nailing

2.7 AO Image Collection AO Principles of fracture Management (T.P. Ruedi, W.M. Murphy)

3.7 AO International AO Teaching Series-LCP (Thomas P. Ruedi, Prof. Michael Wagner)

Foreword-Basics LCP System LCP Cases Literature and studies

Methods of osteosynthesis AO Principles Biomechanical Principles Surgical techniques

Description Implants and instruments Application Indications Operating techniques

Humerus Femur Tibia Periosteogenic

Related Literature Study results

4.7 AO Principles of Fracture Management (Thomas P. Ruedi, William M. Murphy) (CD I, II)

1- AO philosophy and Its basis 2- Decision making and planning 3- Reduction and fixation techniques 4- Specific fractures 5- General topics 6- Complications

5.7 Atlas of Orthopaedics Surgery (Disk 1-6)

Disk 1: Condylar Plate Fixation in the Distal Femur, Malleolar Fracture Fixation, Malleolar Fracture Type B, Malleolar Fracture Type C, Tension Band Wiring on the Elbow Femoral Neck Fracture Large Cannulated System, Fracture of the Radius Shaft 3.5 LC-DCP, Screw Fixation and Plating

Disk 2: Techniques of Absolute Stability, Proximal Humerus Fracture, Reduction with Clamps, Posterior Wall Fracture, Posterior + Transverse Wall Fracture, Undeamed Tibial Nails (UTN), Intraarticular Fracture of the Distal Humerus

Disk 3: Fracture of the Tibial Plateau, Tibia Fracture in Foam LH, Reduction Techniq, The Undeamed Femoral Nail System, Dynamic Condylar Screw (DCS), Dynamic Hip Screw (DHS), Pin Tibial Fractures (Footed Foot)

Disk 4: Application of Large Distractor, AO Asif External Fixator, PC-FIX Point Contact Fixator an Internal Biological, The Proximal Femoral Nail (PFN), Bicondylar Fracture of Tibial Plateau, Minimal Invasive Plating of the Tibia

Disk 5: Direct and Indirect Reduction Techniques, Short Oblique Radius Fracture, Small External Fixator, Intraarticular Fracture Distal Radius, Distal Radius, Open Reduction & Fractures of the Calcaneus, Postoperative Treatment, Internal Fixation of a Humeral Shaft Fracture

Disk 6: High Cinematography of a Butterfly Fracture, Posterior, Pelvic Fixations Symphysis Pubis & Pubic Rami, Pelvic Fixations, Anterior Plate Fixation 53028, The Pelvic C-Clam, Liss Less Invasive Stabilization System, LCP Locking Compression Plate

6.7 Body in Motion (Susan K. Hillman)

- Anatomy - Content - Everything - Anatomy Text - Surface Anatomy Videos - Muscle Action Videos

2003

7.7 CCC (Core Curriculum in Primary Care) Orthopedics/Sport Medicine Section

1- Introduction 2- Orthopedic Procedures: A Rheumatology's Perspective 3- Exercise and Aging A Prescription for life 4- Foot and Ankle Problems Part Two
8.7 Click'X VenttoFix SynCage (J. Webb, O. Schwarzenbach J. Thalgott) (VCD) (AO ASIF OFFICIAL TAPE)  

9.7 FRACTURES IN ADULTS (ROCKWOOD AND GREENS)  
1- General Principles 2- Upper Extremity 3- Spine 4- Lower Extremity  

10.7 FRACTURES IN CHILDREN General Principle Upper Extremity Spine Lower Extremity (ROCKWOOD AND WILKINS) (James H. Beaty, James R. Kasser)  

11.7 FRACTURES OF THE PELVIS AND ACETABULUM (G.F. Zinghi, A. Briccoli, P.Bungaro) (Salekan E-Book)  

12.7 Gait Analysis an introduction (Third Edition) An interactive multi-media presentation produced using polygon software (Micheal W. Whittle)  

33.1 Imaging of Spinal Trauma in Children (Lawrence R. Kuhns, M.D.) (University of Michigan Medical Center)  

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3- Interactive hand therapy  
4- Interactive Hip  
5- Interactive Shoulder  
6- Interactive Knee  
7- Sports Injuries The Knee  
8- Interactive Food and Ankle  
9- Interactive Skeleton  

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14.7 Internal Fixation of a Humeral Shaft Fracture with the UHN (P.M.Rommens, J. Blum)  

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15.7 MASTER TECHNIQUES IN ORTHOPAEDIC SURGERY RECONSTRUCTIVE KNEE SURGERY Southern California Center for Sports Medicine Long Beach, California (DOUGLAS W. JACKSON, M.D.)  

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35.1 Magnetic Resonance Imaging in Orthopedics and Sport Medicine (David W. Stoller)  

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<td>Intraocular Inflammation and Uveitis (Section 9) (SALEKAN E-BOOK)</td>
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<td>LEO Clinical Update Course on Retina (H. Michael Lambert, Charles. Arr, J. Paul Diechert, Mark W. Johnson, James S. Tiedeman)</td>
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<td>LEO Clinical Update Course on Cataract (Stephen S. Lane, MD, Alan S. Candall, MD, Douglas D. Koch, MD, Roger F. Steinert, MD)</td>
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<td>Loeil Prental Endoscopie du Vitre Phaco Chop (VIDEO Media) (Helen K. WU, MD, Roger F. Steinert, MD, Michael B. Raizman, MD)</td>
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<td>47.8</td>
<td>MOVIMENTQ NATURAL PARA EL OJO ARTIFICIAL (VCD), (AJL OPHTHALMIC, S.A.)</td>
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<td>MVP VIDEO JOURNAL OF OPHTHALMOLOGY</td>
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<td>New England Eye Center Imaging in Glaucoma</td>
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<td>New England Eye Center Photorefractive Keratometry (PRK) Course (Helen K. Wu, MD, Roger F. Steinert, MD, Michael B. Raizman, MD)</td>
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<td>OCULAR PATHOLOGY (FIFTH EDITION) (MYRON YANOFF, MD AND BEN S. FINE, MD) (Mosby) (SALEKAN E-BOOK)</td>
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### Notes
- OCT: Optical Coherence Tomography
- SLO: Scanning Laser Ophthalmoscope
1.9 5 Minute Neurology Consult (SALEKAN E-BOOK) (D. Joanne Lynn)
- Neurologic Symptoms and Signs
- Neurologic Diagnostic Tests
- Neurologic Diseases and Disorders
- Short Topics

2.9 55th Annual Meeting March 29-April 5, American Academy of Neurology (HAWAII)

3.9 Abnormal Psychology LIVE and interactive tutorial (Barlow/Durand's, Durand/Barlow's, Trull/Pharc's)

4.9 American Academy of Neurology 2004 Syllabi

5.9 Advanced Therapy of HEADACHE CONQUERING HEADACHE (SECOND REVISED EDITION) An Illustrated Guide to Understanding The Treatment and Control of Headache (Alan M. Rapoport, Fred D. Sheftell)

6.9 Atlas of Functional Neuroanatomy (Dr. Walter J. Hendelman)

7.9 Boehringer Ingelheim Satellite Symposium International Stroke Conference (Phoenix, Arizona)

8.9 Brainiac!™ Medical Multimedia Systems Presents (Version 1.52) (An interactive digital atlas designed to assist in learning human neuroanatomy)

9.9 Clinical Neurology (G David Perkin Fred H Hochberg Douglas C Miller)

10.9 Comprehensive Textbook of PSYCHIATRY (Seventh Edition CD-ROM) (Benjamin J. Sadock, MD – Virginia A. Sadock, MD) (LIPPINCOTT WILLIAMS & WILKINS)

Unên

- Neurology Symptoms and Signs
- Neurologic Diagnostic Tests
- Neurologic Diseases and Disorders
- Short Topics

Presentation

CD

5 Minute Neurology Consult

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- Neurologic Diagnostic Tests
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Comprehensive Textbook of PSYCHIATRY (Seventh Edition CD-ROM) (Benjamin J. Sadock, MD – Virginia A. Sadock, MD) (LIPPINCOTT WILLIAMS & WILKINS)
Severe Amnesic Syndrome: Anterograde and Retrograde Amnesia Perseverative Verbal Behavior in Amnesia Semantic Memory Loss Fluctuating Sensory In Dementia With

Left Spatial Neglect Eye Movements in Severe Left Spatial Neglect Anosognosia for Hemiparesis Paraphasias

Broca's Aphasia Lewy Bodies Impaired Verbatim Repetition

21.9 DISORDERS OF COGNITIVE FUNCTION (VCD-II) (AMERICAN ACADEMY OF NEUROLOGY) (CONTINUUM) 2002

Wernicke's Aphasia Dysexecutive Syndrome Disinhibited Behavior Grasp Response and Imitation Behavior Positive Signs of Executive Dysfunction Progressive Apraxia

Negative Signs of Executive Dysfunction Prospagnosia and Visual Agnosia Simultanagnosia Optic Ataxia

22.9 DISORDERS OF COGNITIVE FUNCTION (VCD-III) (AMERICAN ACADEMY OF NEUROLOGY) (CONTINUUM) 2002

Basic Mental Status Examination Token Test for Auditory Comprehension Confrontation Naming Finger Constructions Luria 3-Step Test Line Cancellation Gestural Praxis

23.9 EMG Training (Kenneth Ricker, M.D.)

EMG as a Tool for Evaluation and Diagnosis of Neurological and Neuromuscular Diseases

24.9 ENS Teaching Course

Dizziness and vertigo Clinical Neurophysiology Clinical Neuropathology Sleep Disorder Stroke

Neuropathology for Clinicians Neurosurgery for Neurologist Epilepsy Multiple Sclerosis Muscle disorders

Neuroimaging Neurology of Systemic disease Parkinson's disease Ultrasound in Neurology Dementia

TICU in Neurology Movement disorders Neuropathies Current Treatments Neurology

25.9 EPILEPSY The Comprehensive CD-ROM (Jerome Engel, Jr., M.D., Ph.D., Timothy A. Pedley, M.D.) Lippincott Williams & Wilkins 1999

Epilepsy: A comprehensive textbook and reference

26.9 Essentials of Clinical Neurophysiology (Karl E. Misulis MD. Ph.D., Thomas C. Pedley MD.) Lippincott Williams & Wilkins 2002

27.9 Foundations of NEUROBIOLOGY

28.9 Foundations of Behavioural Neuroscience

29.9 FUNDAMENTALS OF HUMAN NEURAL STRUCTURE (S. Mark Williams) (Sylvius™ 2.0)

30.9 General depression and its pharmacological treatment (Professor Brain Leonard) (VCD)

31.9 Guidelines (American Academy of Neurology) (SALEKAN E-BOOK) 2004

Brain Injury & Brain Death - Child Neurology - Dementia - Epilepsy - Headache - Movement Disorders - Multiple Sclerosis - Neuroimaging - Neuromuscular - Stroke and Vascular Neurology - Technology Assessment
بیمارستان RiverView Alzheimer disease group
کانال تیپ کرده دری نست. چندین فلخه فیلم اموزش راجع به نحوه مصاحبه با بیماران میلادی به آزمایش و شرح حال

58.9 THE HUMAN BRAIN (Marion Hall David Robinson)

59.9 THE HUMAN NERVOUS SYSTEM (Springer)

60.9 The Massachusetts General Hospital Handbook of Pain Management (Second Edition) (Jane Ballantyne, Scott M. Fishman, Salahadin Abdil) (SALEKAN-E-book)

61.9 The Movement Disorder Society's Guide to Botulinum Toxin Injections

62.9 Thinking a head (Critical question in ms therapy)

63.9 Understanding and Diagnosing Restless Legs Syndrome

CD علائم

1.10 (AGA Postgraduate Course) A Day and Night in the Life of a Gastroenterologist

2.10 3DClinic (Version 1.0) Seeing is Understanding

3.10 Adult Airway Management Principles & Techniques American Association (afael A. Ortega, M.D., Harold Arkoff, M.D.)

4.10 Advanced Therapy of INFILAMMATORY BOWEL DISEASE (Theodore M. Bayless, MD, Stephen B. Hanauer, MD)

5.10 AGA Postgraduate Course CONTROVERSIES And CLINICAL CHALLENGES (An Intensive Two-Day Course Covering A Diversity of Topics Related to the Pancreas)

6.10 Part 1: Physiological Basis of Gastrointestinal Motility Part 2: Motility Test for the Gastrointestinal Tract

7.10 Atlas of GASTROINTESTINAL MOTILITY in Health and Disease (Second Edition)

2001

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2003

2001
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<td>Marvin M. Schuster, MD, FACP, FAPA, FACG, Michael D. Crowell, PhD, FACG, Kenneth L. Koch, MD</td>
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<td>Atlas of Clinical Oncology Cancer of the Lower Gastrointestinal Tract</td>
<td>Christopher G. Willett, MD</td>
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<td>Atlas of Clinical Rheumatology (2nd Edition)</td>
<td>David J. Nashel, Chief, Rheumatology Section Va Medical Center, Washington, Professor of Medicine Georgetown University</td>
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<td>1. Clinical Atlas of Rheumatic Diseases</td>
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<td>Raymond E. Lenhard, MD, Robert T. Osteen, MD, Ted Gansler, MD</td>
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<td>Ingram Roberts, MD</td>
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<td>G. Michael Besser MD, DSc, FRCP, Michael O. Thorner MB BS, DSc, FRCP</td>
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<td>(Robert R Rich, Thomas A Fleisher, William T Shearer, Brain L Kotzin, Harry W Schroeder)</td>
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<td>Raymond E. Lenhard, J. MD, Robert T. Osteen, MD, Ted Gansler, MD</td>
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<td>Colonoscopy</td>
<td>CB Williams, JD Waye, Y Sakai</td>
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<td>G. Michael Besser MD, DSc, FRCP, Michael O. Thorner</td>
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<td>Jean Bourbeau, MD, MSc, FRCP, Diane Nault, RN, MSc, Elizabet Borycki</td>
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<td>Differential Diagnosis</td>
<td>LC Gupta Abhital Gna Abhiseh Gupta (Salekan E-Book)</td>
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<td>Diseases of the Liver</td>
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<td>General Considerations</td>
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34.10 Linear ECHO ENDOSCOPY  Tome I anatomy  (Dr. Marc Giovannini)
- Equipment  - Environment  - Echo-anatomy

35.10 Menopausal Osteoporosis (Neill Musselwhite, M.D., Herman Rose, M.D.)

36.10 MKSAP® 12  (American College of Physician-Physician-American Society Internal Medicine)
- Gastroenterology and Hepatology  - Endocrinology and Metabolism  - Infectious Disease Medicine  - Rheumatology  - Oncology  - Hematology  - Cardiovascular Medicine  - Pulmonary Medicine

37.10 Oxford Textbook of Medicine (OTM)  (Weatherall, Ledingham, Weatherall)

38.10 Parenting Guide

39.10 Pre-Colonoscopy Education Program  (Dr. Michael Shaw, Dr. Oliver cass Dr. James Reynolds Patricia Tomshine, Rn)
- Reason for Colonoscopy  - The Colon and The Colonoscope  - Preparations  - Day of the Procedure  - About the Procedure  - After the Procedure  - Minor Complications  - Major Complications

40.10 Principles & Practice of Infectious Diseases  A Harcourt Health Sciences Company

41.0 Rheumatology  (John H. Klippel, Paul A Dieppe)
- Rheumatic Diseases  - Signs and Symptoms  - Rheumatoid Arthritis and Spondyloarthropathy  - Infection and Arthritis
- Regional Pain Problems  - Connective Tissue Disorders  - Disorders of Bone, Cartilage  - Management of Rheumatic Disease

42.10 TEXTBOOK OF GASTROENTEROLOGY  (Third Edition)  ATLAS OF GASTROENTEROLOGY  (Second Edition)  (David H. Alpers, MD, Loren Laine, MD)

43.10 Textbook of Rheumatology  (Kelley's)  (W.B. Saunders Company)
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**Textbook of TRAVEL MEDICINE and HEALTH** (Herbert L. Dupont, M.D., Robert Steffen, M.D.) (B.C. DECKER INC)

- Section XI: VASCULITIC SYNDROMES
- Section XII: SCLERODERMA AND MIXED CONNECTIVE TISSUE DISEASES
- Section XIII: STRUCTURE, FUNCTION, AND DISEASE OF MUSCLE
- Section XIV: RHEUMATOID DISEASES OF CHILDHOOD
- Section XV: CRISTAL-ASSOCIATED SYNOVITIS
- Section XVI: OSTEOARTHRITIS, POLYCHONDHRITIS, AND HERITABLE DISORDERS
- Section XVII: ARTHRITIS RELATED TO INFECTION
- Section XVIII: ARTHRITIS ACCOMPANYING SYSTEMIC DISORDERS
- Section XIX: DISORDERS OF BONE AND STRUCTURAL PROTEIN
- Section XX: TUMORS INVOLVING JOINTS
- Section XXI: RECONSTRUCTIVE SURGERY FOR RHEUMATIC DISEASE


- I. General Considerations
- II. Diagnosis of Pain
- III. Therapeutic Options: Pharmacologic Approaches
- IV. Therapeutic Options: Nonpharmacologic Approaches
- V. Acute Pain
- VI. Chronic Pain
- VII. Pain Due to Cancer
- VIII. Special Situations
- Appendices
- Subject Index

**UEGW Gastroenterology** Week 10th United European (Geneva, Switzerland)

**UpToDate** CLINICAL REFERENCE LIBRARY 13.1 (CD I, II) (Burton D. Rose, MD, Joseph M. Rush, MD)

- Adult Primary Care
- Allergy and Immunology
- Cardiology
- Critical Care
- Drug Information
- Endocrinology
- Family Medicine
- Gastroenterology
- Gynecology
- Hematology
- Infections Disease
- Nephrology
- Oncology
- Pediatrics
- Rheumatology
- Pulmonology
- Women's Health

**YEAR BOOK of RHEUMATOLOGY, ARTHRITI, AND MUSCULOSKELETAL DISEASE** (Richrd S. Panush, MD) (SALEKAN E-BOOK)

- Health Sciences, Epidemiology, Economics, & Arthritis Care
- Systemic Lupus Erythematosus and Related Disorders
- Rheumatoid Arthritis
- Vasculitis and Systemic Rheumatic Diseases and Other Related Disorders
- Systemic Sclerosis and Related Disorders
- Osteoarthritis, Crystal-Related Arthropathies, Osteoporosis, Infectious Arthritis, and Spondyloarthropathies
- Regional Pain Syndromes, Non-Articular Musculoskeletal Disorders, and Fibromyalgia
- Miscellaneous Topics

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**11- اطفال**

**1.11** A Major Contributor to Neonatal Infant Morbidity and Mortality (SURVANTA) (Part I, II) (Alan J. Gold, MD, J. Harry Gunkel, Arvin M. Overbach)

**2.11** Atlas of Pediatric Gastrointestinal Disease

**3.11** Basic Mechanisms of Pediatric Respiratory Disease (Second Edition) (Gabriel G. Haddad, MD, Steven H. Abman, MD)

**4.11** Child Development, 9/e (John W. Santrock)
بیست و چهارمین مجله بهداشت، به‌عنوان یکی از مهم‌ترین و معروف‌ترین مطبوعات در زمینه بهداشت و پزشکی در ایران، به فنون و تکنیک‌های جدید و بهبودی رسانی منابع در زمینه درمان و درمان که به وسیله گروه‌های تخصصی و متخصص در زمینه بهداشت و پزشکی ارائه می‌شود.

نتیجه‌گیری

این مطالعه نشان می‌دهد که گروه‌های تخصصی و متخصص در زمینه بهداشت و پزشکی در ایران، به فنون و تکنیک‌های جدید و بهبودی رسانی منابع در زمینه درمان و توانایی بهبودی رسانی منابع در زمینه درمان که به وسیله گروه‌های تخصصی و متخصص در زمینه بهداشت و پزشکی ارائه می‌شود.

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نتیجه‌گیری

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10.12 HEALTH ASSESSMENT (Gaylene Bouska Altman, RN, Ph.D., Karrin Johnson, RN, Robert W. Wallach, MD) 2002

11.12 MCCQE Review Notes and Lecture Series (Marcus Law & Brain Rotenberg) 2000

12.12 Medical Dictionary (Dorland's) (by W. B. Saunders) — 2000

13.12 MEDICAL Encyclopedia For Health Consumers (With Atlas) —

14.12 MedStudy™ (The Best Internal Medicine Board Review) 2000

15.12 Natural Medicine Instructions for Patients (Lara U. Pizzorno, Joseph E. Pizzorno, Jr, Michael T. Murray) 2002

16.12 Patient Teaching Aids 2002

17.12 Practical General Practice (Guidelines for effective clinical management) (Alex Khot, Andrew Polmear) (Third Edition) —

18.12 RAPID REVIEW FOR USMLE STEP 1 (Mosby) 2002

19.12 SPSS 12.0 for Windows 2003


بنچ 1: 150 موردی از انواعی و پزشکی: شامل 150 سهمه سالنی با تفسیر طراحی شده از دستگاه و اندام‌های بدن به همراه اطلاعات مربوط به آن در این بنچ تهیه مطالب انتخابی و فیزیولوژی مورد بررسی است.

بنچ 2: مواردی که در این بنچ در این راهکار قابل قبول و راهبردی در هنگام مسابقه می‌باشد. همچنین مواردی که ساختار انتخابی لازم نیست بررسی شود.

بنچ 3: مواردی که باید در این بنچ بیان شود. در این بنچ کریکت انتخابی می‌باشد و در آن مراحل و علل آن در حالت شایع و نادر باید بررسی شود.

بنچ 4: مواردی که باید در این بنچ بیان شود. در این بنچ کریکت انتخابی می‌باشد و در آن مراحل و علل آن در حالت شایع و نادر باید بررسی شود.

بنچ 5: آنالیز بانک می‌باشد. در این بنچ پیشنهادات مصرف هر ماده به اثر تکیه و استحکام در مورد به معیارهای و انتخابات مشابه بر مبنای معیارهای انتخابی است.
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**Common Chemotherapy Regimens in Clinical Practice**  | **Antimetic Agents for the Treatment of Chemotherapy-Induced Nausea and Vomiting**

2004

22.13 The Herbalist (David L. Hoffman)  
- Basic Principles  
- Human Systems  
- Actions  
- Herbal Materia Medica

23.13 THE MERCK INDEX on CD-ROM (Version 12:3)  
2000

24.13 USP 27-NF 22 Through Supplement Two (U.S. PHARMACOPEIA) (The standard of Quality) (The United States Phamocopeial Convention, Inc)  
2004
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<td>Surgery of the Liver &amp; Biliary Tract 3e: Selected Operative Procedures (L.H. BLUMGART, Y. FONG) (W.B. Saunders)</td>
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The IMZ Implant System (VCD) (Dr. Karl-Ludwing Ackermann, Dr. Axel Kirsch) (CD I, II)

Toothcolored Restoratives

TOOTH-COLORED RESTORATIVES Ninth Edition (Principles and Techniques) (Harry F. Albers, DDS)

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Treatment Planning in Dentistry

UCD Implant

ANATOMY & PHYSIOLOGY (5th Edition) (Gary A. Thibodeau, Kevin T. Patton)

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Interactive Physilogy: RESPIRATORY SYSTEM

Interactive Physilogy: Cardiovascular System

Interactive Physilogy: Urinary System

InterActive PHYSiOLOGY

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| 10.17 | The Interactive Skeleton Tutorial | (Dr. Peter Abrahams of Cambridge University, UK.) | — |
| 11.17 | World of SPORT examined | — |
| 13.17 | Sobotta (Atlas of Human Anatomy) | (Urban & Schwarzenberg) | — |
| 15.17 | Therapeutic Exercise for Lumbopelvic Stabilization | A Motor Control Approach for the Treatment and Prevention of Low Back Pain | (Second Edition) | (Carolyn Richardson, Paul W. Hodges, Julie Hides) | (Salekan E-Book) | 2004 |
| 16.17 | Gray's Anatomy | The Anatomical Basis of Clinical Practice | (Thirty-Ninth Edition) | (Susan Standring) | (CD I, II) | (Salekan E-Book) | 2005 |
| 1.18 | The Oncology Nursing Society presents | THE ADVANCED PRACTICE ONCOLOGY NURSING REVIEW | — |
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| 3.18 | Focus on Nursing Pharmacology | (Lippincott Williams & Wilkins) | 2000 |
| 4.18 | Wongs ESSENTIALS OF Pediatric Nursing | (Mosby) | A Harcoun Health Sciences Company | 2001 |
| 5.18 | Maternal, Neonatal and Women's Health Nursing | By Delmar, a division of Thomson Learning | 2002 |
| 6.18 | Nursing Care of Infants and Children | (Seven Edition) | 2003 |
| 7.18 | McMinn's Interactive Clinical Anatomy | — |
| 8.18 | INERACTIVE ATLAS OF CLINICAL ANATOMY | (Illustrations by Frank H. Netter, M.D.) | — |
| 1.19 | BACK STABILITY | Christopher M. Norris, MSc, MCSP, Director, Norris Associates, Manchester, UK | (Salekan E-Book) | — |
4.19 DIGITAL SHIATSU

- خود ماساژ درمانی (total body)
- مدار کاربردی ماساژ درمانی (self-shiatsu)
- جستجو - اساس و مبانی ماساژ درمانی - راهنما

در این قسمت روش ماساژ صحیح و عمل مامی بدین همانا به ماساژ فیلم و توضیحات کودنده و مبن بایانه می‌شود. در تصاویر طراحی علائم حساسی که در ماساژ درمانی مورد توجه قرار می‌گیرند نمایش داده است.

- همه‌یا به نمایش فیلم و توضیحات کودنده در دو قسمت روش ماساژ درمانی ارائه شده است.
- مدار کاربردی ماساژ درمانی در 32 نمونه درمانی است. (نیا: از آریوپلاستیک، در یک شرکت می‌باشد، قطعات، چنین دارای فیلم و توضیحات خوشنویسی نمایش داده شده است نامیکوشي)
- اصول ماساژ درمانی و روش‌های کلاسیک که نیز تاریخچه متعددی دارد.
- بر اساس نحوه فلسفه می‌توان از این روش‌ها تجزیه و تحلیل نمود و با کمک نمونه به روشی که آن مبنا و محتوای مختلفه.

این برنامه به صورت autorun.exe نصب می‌شود.

5.19 EXERCISE THERAPY PREVENTION AND TREATMENT OF DISEASE

6.19 Fibromyalgia Syndrome Bodywork Management Strategies

Assessment Methods

- Manual Thermal Diagnosis
- Skin on Fascia Adherence
- Hypersensitive Skin Zones reduced Skin elasticity
- Drag palpation for increased hydrosis
- Neuro muscular Technique Evaluation (NMT)

7.19 Fundamentale of Sensation ad Perception (3rd Edition) (M.W. Levine)

8.19 Health & Fitness (DataSel Software, Inc)

9.19 Interactive Atlas of Human Anatomy

10.19 Introduction to Massage Therapy (Mary Beth Braum, Stephanie Simonsoon) (Salekan E-Book)

11.19 MANIPULATION OF THE SPINE, THORAX AND PELVIS.

MANIPULATION OF THE SPINE, THORAX AND PELVIS. An Osteopathic Perspective (Peter Gibbons, Philip Tahan)

این قسمت به صورت نمایش مدار کاربردی فیلم و توضیحات کودنده در دو قسمت روش ماساژ درمانی ارائه شده است.

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HVLA thrust techniques-spine and thorax - Cervical and cervicothoracic spine - Thoracic spine and rib cage - Lumbar and thora Columbar spine

12.19 Massage Therapy Review (interactive Edition) (Mosby) —

13.19 Men's Health GET RID OF THAT GUT STAGE 1: BEGINNERS LEVEL STAGE 2: INTERMEDIATE LEVEL STAGE 3: ADVANCED LEVEL

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22.19 Power Touch —


24.19 Surface and Living Anatomy (Gordon Joslin SotI) 2002

25.19 The Complete Acupuncture —

26.19 The Principles of Harmonic Techniques (Eyal Lederman) (VCD) —
AUA Vide Digest The American Urological association (AUA) Impotence and Infertility

3.21

CD Impotence (Diagnosis & treatment option)

Penile Venous Ligation

Infertility

Rectal Probe Electroejaculation

CD Infertility

Normal Bladder Anatomy and Variants of Normal histology

Flat Urothelial Lesions

Invasive Urothelial Carcinoma

Conventional Morphologic, Prognostic, and Predictive Factors and Reporting of Bladder Cancer

Squamous Lesions

Cystitis

Miscellaneous Nontumors and Tumors

Second any Tumors of the Bladder

Bristol Urological Institute (Computer Aided Learning Program)

Campbell’s Urology

Core Curriculum in Primary Care Patient Evaluation for Non-Cardiac Surgery and Gynecology and Urology

Core Curriculum in Primary Care Gynecology

Nephrology

Core Curriculum in Primary Care Nephrology

CD, Core Curriculum.
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Harvard CCC

Majmooa-ei tohlezooyi be surat-ei, irodalami, hizmat-ei ke in-khojas-ei zaroto.  CD

Harvard CCC

CD I: Clinical Urology - Pediatric Urology - Investigative Urology - Urological Survey

CD: Cystectomy and Construction an Ileocecal Neobladder for Urethral Voiding (John A. Libertino MD, FACS)

Erectile Dysfunction Current Investigation and Management (Ian Eardley, Drishna Sethia)

Hot Topics in UROLOGY (Roger S Kirby, Michael P O'Leary) (SALEKAN E-BOOK)

1- How to erahcate Renal mass/Tumor  2- Drugs vs Diet in Modifying Renal failure  3- Treatment of Mypertension-Special Case  4-Clinical Application of Renal Physiology

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CD: Male and Female Sexual Dysfunction (Allen D. Seffel) (Salkan E-Book)

Pelvic Floor Exercises for Erectile Dysfunction (Grace Dorey phD MSCP)

PRIMER ON KIDNEY DISEASES (Second Edition) (NATIONAL KINDNEY FOUNDATION SCIENTIFIC ADVISORY BOARD)

The Journal of UROLOGY (Spring & Summer) (CD I, II) (Official Journal of the American Urological Association)
16.21 Urogynechology: Evaluation and Treatment of Urinary Incontinence (Bruce Rosenzweig, MD, Jeffrey S. Levy, MD, Donald R. Ostergard, MD)

Types of incontinence • incontinence awareness • incontinence management to private patients • Non surgical therapy • urogynechology as a subdiscipline • professional consideration

Cystoscopy • uroflowmetry • Postvoid residual • Cystometrogram • Pad test • Pessary test • Multi-Channel urodynamics

Stress urinary incontinence

Consideration for the OB/Gyn Generalist

Complication • Procedure • Y • Types of incontinence • incontinence awareness

Consideration for the OB/GYN Generalist

Smith's General Urology (Sixteenth edition) (Emil A. Tanagho, Jack W. Meanin) (Salekan E-Book)

Glenn's Urologic Surgery (Sixth Edition) (Sam D. Graham, James F. Glenn) (Salekan E-Book)

The Kidney (Volume 1-2) Seven Edition (Barry M. Brenner) (E-Book)
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| 12. Diagostic Neuroradiology | Anne G. Osborn | (Mosby) | تک جلدی | 500,000
| 13. Bone and Joint Disorders | Conventional Radiologic Differential Diagnosis | Francis A. Burgener Marti Kormano | (Mosby) | تک جلدی | 400,000
| 15. Radiobiology for the Radiologist | (Fifth Edition) | | تک جلدی | 400,000
| 16. Anatomy Positioning & Procedures Workbook | Steven G. Hayes | | تک جلدی | 470,000
| 17. Atlas of Normal Roentgen Variants That May Simulate disease | (Seven Edition) | (Theodore E. Keats & Mark W. Anderson) | (Mosby) | تک جلدی | 700,000
| 18. مبانی اساسی در سونوگرافی دایر و تجهیزات آن (ترجمه و گردآوری: دکتر پروین علی‌پور) | | | تک جلدی | 50,000
| 19. اصول تشخیصی و درمانی بیماری‌های بیماری (دکتر مصطفی کیانی، دکتر الهام حجی‌محمدیان) | | | تک جلدی | 180,000
| 20. شایع‌ترین‌ها، نادرترین‌ها، تشخیص‌های افتراضی، بین‌سراب‌تشخیص بیماری‌های (تالیف: دکتر احمد علیزاده) | | | تک جلدی | 50,000
| 26. Practical Guide to Abdominal & Pelvic MRI | (John R. Leyendercker, Jeffrey J. Brown) | | | | | تک جلدی |

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| 27. Ultrasonography in Urology | A Practical Approach to Clinical Problems | (Edward I. Bluth-Peter H.) | | | | تک جلدی | 350,000
| 28. Seminars in Ultrasound CT and MR | | | | | | تک جلدی | 70,000
| 29. Diagnostic Ultrasound | (Rumack, Wilson, Charboneau) | (2005) | | | | تک جلدی | 1,400,000

چاب اول این کتاب که در سال 1991 به پایان رسید و به عنوان را یکی از مرجع سونوگرافی در جهان می‌باشد. از آن‌جا که دانش سونوگرافی در طول 8 سال گذشته پیشرفت‌های بسیاری داشته است که به‌بازگشت در این کتاب احساس نمی‌شود.
در این کتاب بیش از یکصد نویسنده متخصص در سونوگرافی ناشی کرده‌اند از خصوصیات دانش سونوگرافی در زمینه تصوربرداری، تشخیص و کاربرد آنها را به روش تجزیه‌درآورده‌اند. فصول کتاب شامل سونوگرافی ناب‌پوش‌سازی و ثبت‌هایی تحت هدایت سونوگرافی تا گزارش کلی کتاب را در کلیه عضوه‌افزای حجم مربوط به سونوگرافی متغیران و راک‌پاژین می‌پوشاند.

تعداد زیادی از تصاویر گزارش‌هایی در رابطه با سونوگرافی زنان و پزشکان و دست‌نوازه‌های متعدد، در تفسیر و درک طبقات در ساختمان انجام شده است. گزارش‌های راهنما و highlight جداول به‌طور کلی در شکل تصویری نشان می‌دهند. مطالب مهم برای تهیه تصویرکشی نشان می‌دهند. و مراحل استفاده به صورت دقیق می‌تواند. نیاز داشته باشند. این کتاب در دو جلد نوشته شده است. جلد اول شامل محتوای اینچرایکال چون مقایسه‌ای از دو جلد کتاب اغلب مطرح و دسترسی به میزان و ذهنی‌گرایان پژوهشی و تحقیقاتی مورد است. جلد دوم کتاب شامل فصل نجم که بحث کامل سونوگرافی زنان و مردان است و نهایتاً فصل ششم سونوگرافی اطلاعات این جلد جدید در مورد سونوگرافی داخلی اطلاعات در اطلاعات به ایله فصل افراد شده است. خوانندان این کتاب متخصصین و دستاران رادیولوژی دانشجویان بررسی و مورد است. گردید.

- زیر جلد
- 500,000

### 31. **Ultrasound A Practical Approach to Clinical Problems** (Edward Bluth, Peter H. Arger Carol B. Benson, Philip W. Rails, Marilyan) (Thieme)
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- 800,000

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**Doppler**

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<td>Vascular diagnosis with Ultrasound Clinical References With Case Studies</td>
<td>Hennerici, Neuerburg-Heusler(Thieme)</td>
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**Notes:**
- TCD = Transcranial Doppler
- AVF = Arteriovenous Fistulae
PART I (Pathology-based diagnoses): Congenital malformations-Trauma Subarachnoid hemorrhage and Aneurisms-Stroke-Vascular Malformations Neoplasm's and Tumor lesions-Primary Non-neoplastic cysts-Infection and Demyelinating Disease-Metabolic/Degenerative Disorders, Inherited-Toxic/Metabolische/Degenerative Disorders, Acquired
PART II (Anatomy-based Diagnoses): Ventricles and Cysterns-Sella and Pituitary-CPA-IAC-Skull, Scalp and Meninges

89. Diagnostic Imaging Orthopaedics  (Stoller.Tirman Bredella) (2004)  تک جلدی 900,000
90. Diagnostic Imaging Head and Neck (Harnsberger) (2004)  تک جلدی 1,000,000
92. DIAGNOSTIC MUSCULOSKELETAL IMAGING (THEODORE T. MILLER, MARK E. SCHWEITZER) (2005)  تک جلدی 450,000
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Imaging of the newborn, infant, and young child  

Borderlands of Normal and Early Pathological Finding in Skeletal Radiography  
(Fifth revised edition)  
(Juergen Freyschmidt, Joachim Broßmann, Juergen Wiens, Andreas Sternberg)  
(Thieme)

Clinical Imaging  
(Ronald L. Eisenberg, Amelia County  
(an atlas of differential diagnosis)  
(Lippincott Williams & Wilkins)  

Atlas Of Normal Roentgen Variants that may Simulate Disease  
(Mosby Inc.)  (2001)  (Seventh Edition)

 otras recomendaciones:

- El papel de la radiología intervencionista en el manejo del dolor.
- Nuevos avances en la arthrografía.
- El uso de las técnicas de CT y MRI en el diagnóstico de lesiones musculoesqueléticas.
- La importancia de la formación en la radiología intervencionista para los residentes.
- La necesidad de una formación continua en la radiología intervencionista.
- El papel de la radiología intervencionista en el manejo del dolor.
- La importancia de la formación en la radiología intervencionista para los residentes.


Robert C. Gilson, M.D., Ph.D., Cleveland Clinic Foundation, Cleveland, Ohio
Charles F. Luminet, M.D., Ph.D., Hospital of the University of Pennsylvania, Philadelphia
Guy Marshall, Ph.D., M.D., Case Western Reserve University, Cleveland, Ohio

MRA (Magnetic Resonance Angiography)
Looking for the number key to the diagrams? Just fold out this page...

A didactically brilliant and unprecedented approach to understanding CT imaging

(Matthias Hofer, MD)  Institute for Diagnostic Radiology, MNR Clinic, Duesseldorf, Germany

Ideal for radiology residents, students and technicians, this concise manual is the perfect introduction to the practice and interpretation of computed tomography.

Designed as a systematic learning tool, it introduces the use of CT scanners for all organs. Finally, self-assessment quizzes – including answers – at the end of each chapter help the reader monitor progress and evaluate knowledge gained.

Special Feature

Includes detachable, pocket-sized cards containing checklists and tables of normal measurements – perfect for study or quick reference when on rounds.

کتاب فوق ذکر در مورد MRI و CT Scan در زمینه نوروزادی‌یولوژی به بحث و بررسی می‌پردازد و شامل 4 بخش اصلی است:

**بخش اول**: ملاحظات تکنیکی باید
- قسم 1 - اصول فیزیکی مرتبط به CT Scan مورد استفاده بالینی
- MRI و CT Scan مورد استفاده بالینی

**بخش دوم**: مغز
- قسم 2 - اناتومی زمردی مغز در MRI و CT Scan
- عفونت‌ها و بیماری‌های اندازه‌گیری

**بخش سوم**: چهارم: ستون فقرات
- قسم 3 - ستون فقرات مغز، مغزگیری و مغز‌یابی
- ستون فقرات و اندازه‌گیری

**بخش چهارم**: ستون فقرات
- قسم 4 - اناتومی زمردی مغز در MRI و CT Scan
- عفونت‌ها و بیماری‌های اندازه‌گیری

لازم به ذکر است که در کتاب فوق، برای بهبود مطالعه از توصیف‌گونه‌های مربوط به استفاده گردیده و برای طبقه‌بندی تکات اساسی از جداول متعدد بهره‌گیری شده است.

## HIGHLIGHTS OF OPHTHALMOLOGY INTERNATIONAL

### WAVEFRONT ANALYSIS, ABERROMETERS and CORNEAL TOPOGRAPHY

**B. BYOD, A. AGARWAL** (2003) 1100,000R

گرچه هنوز هم در بسیاری از نقاط کشورهای امکان عمل جراحی کاتاراكت رسانی به روش‌های نسبتاً قدیمی نیز وجود داشته، عدسی‌های الکترونی به پاس خدمات دانش‌شرکت‌های نوروزادی در بزرگ‌شهر مورگانی، نام می‌گیرند. یکی از اولین خدمات در این زمینه، به نام "Super Vision"، افق‌پردازی به نام Customized LASIK، مورد اکتشاف و ذکر در کتابرسی در کشور از آن جا بعنوان "ویژگی پیشرفته‌ای" شناخته می‌شود. در جهت فروش‌شدن علیه م موجود در این زمینه، این کتاب با عنوان WAVEFRONT ANALYSIS, ABERROMETERS and CORNEAL TOPOGRAPHY پرداخته است.
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<td><strong>OPHTHALMOLOGY MONOGRAPHS Cataract Surgery and Intraocular Lenses</strong></td>
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مکان: جنوب، کارگر نیک، نیاک، خیابان، بین کارگر و ملمارد، نیایشگاه سایت، پلاک ۹۸۷۶۸
تلفن: ۶۹۶۶۸۹۹-۱۶۳۲۷۳۲۱۹۰

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**Red/Blue Lesions**


### Vesiculobullous Diseases


**Ulcerative Conditions**

Lo Muzio L, Mignogna MD, Favia G, et al. The possible association between oral lichen planus and oral squamous cell carcinoma: a clini-


Pigmentary Disorders


**Verrucal-Papillary Lesions**


**Connective Tissue Lesions**


Salivary Gland Diseases

**Lymphoid Lesions**


**Cysts**


**Odontogenic Tumors**


Philipsen HP, Reichart PA, Takata T. Desmoplastic ameloblastoma (including “hybrid” lesion of ameloblastoma). Biological profile based on 100 cases from the literature and own files. Oral Oncol 2001;37:455–60.


Benign Nonodontogenic Tumors


**Inflammatory Diseases**


Tooth Abnormalities


**Malignant Nonodontogenic Tumors**


**Metabolic and Genetic Disorders**