I. Introduction

II. Vesiculo-Bullous Diseases
   A. Herpes Simplex Infections
      1. Etiology
         a. herpes simplex virus
      2. Clinical features
         a. vesiculo-ulcerative eruption occurs in the oral and perioral tissues
         b. following resolution, virus migrates to the trigeminal ganglion
         c. prodromal symptoms of tingling, burning or pain occur followed by multiple short-lived vesicles that become ulcerated
         d. lesions heal without scarring in 1-2 weeks
      3. Differential diagnosis - aphthous ulcers, ANUG, erythema multiforme
      4. Treatment and prognosis
         a. limited success with acyclovir, valacyclovir, and other prescription meds may be better; somewhat faster healing with 10% docosanol (Abreva)
         b. fluids, rest, oral lavage and antipyretics
         c. 40% have secondary herpes
         d. asymptomatic shedding of contact virus particles in saliva and other secretions may occur in previously infected individuals; may recur within 2 weeks
   
   B. Herpes Zoster (Shingles)
      1. Etiology
         a. reactivation of the Varicella-Zoster virus
      2. Clinical features
         a. usually in older adult population and immunocompromised individuals
         b. prodromal symptoms of pain or parasthesia appear followed by a well-delineated unilateral maculopapular rash
         c. unilateral distribution with abrupt ending at midline is diagnostic characteristic of herpes zoster
         d. rash becomes vesicular, pustular and ulcerative
      3. Differential diagnosis - herpes simplex, hand, foot and mouth disease, varicella
      4. Treatment and prognosis
         a. remission occurs in several weeks; treatment similar to herpes simplex with use of antiviral meds (valacyclovir appears to show better results than acyclovir), also can use nsaids, narcotic analgesia, wound dressings and bland lotions (ie, Calamine); steroids are used in severe cases
         b. complications include secondary infection of ulcers; post-herpetic neuralgia, and motor paralysis
         c. vaccination recommended for patients age 60 and older

   C. Herpetic Whitlow
      1. Etiology
         a. herpes simplex virus
         b. occurs in dental practitioners who have been in physical contact with infected individuals
2. Clinical features
   a. vesiculo-ulcerative eruptions on the digits along with signs and symptoms of primary systemic disease
   b. raw, redness and swelling are prominent
3. Treatment and prognosis
   a. may last 4-6 weeks and may recur
   b. limited treatment success with 5% acyclovir ointment, 3% ointment of vidarabine or idoxuridine
   c. must use universal precautions

D. Pemphigus Diseases
1. Pemphigus vulgaris – severe progressive autoimmune disease affecting skin and mucous membrane; characterized by acantholysis-intraepithelial blister formation that results from breakdown of cellular adhesion between epithelial cells; has autoantibodies that are reactive against components of epithelial cell attachment mechanism causing blistering; 50% of cases first seen in oral cavity with painful widespread ulceration following rupture of blisters leads to debilitation, fluid loss, electrolyte imbalance; treatment involves high doses of corticosteroids, azathioprine and methotrexate are used in combination with steroids; once life-threatening, mortality rate is 8-10% in 5 years usually related to complications of steroid treatment, SLE, RA and Sjögren’s may occur with Pvm
2. Cicatricial Pemphigoid – also known as mucous membrane pemphigoid – chronic autoimmune disease affecting oral mucosa, conjunctiva, genital mucosa and skin, not as severe as Pvm, may cause scarring, lesions results from cleavage of the epithelium from the underlying connective tissue; most common site is gingiva, formerly called desquamative gingivitis; erythema and ulceration of free and attacked gingiva, +Nikolski’s sign, bullae and erosions may occur persisting for 24-48 hours, ocular lesions may cause blindness; treatment consists of topical corticosteroids in mild cases, systemic steroids in severe cases; disease is difficult to control and slow to respond to tx, episodes of exacerbation and remission
3. Bullous pemphigoid – 80% occur in those older than 60, no sex predilection, circulating autoantibodies are usually detectable and do not correlate with disease activity; oral lesions less common but occur in 1/3 of patients, gingival lesions resemble Cp, other mucosal lesions are large and painful; high doses systemic steroids and nsaids used for tx

E. Behcet Syndrome
1. chronic, recurrent autoimmune disease, multisystem – affects GI, cardio, ocular, CNS, pulmonary, dermal and oral; average age of onset is 30; increased prevalence in those from Mediterranean regions and Asia; autoantibodies to human mucosa present and HLA B5 and HLA B51
2. Etiology – immunodysfunction with vasculitis
3. Clinical features
   a. oral ulcers resemble aphthous; may be painful and very large
   b. genital ulcers are small, located on the scrotum, base of penis and labia majora
   c. ocular lesions begin with photophobia and can have conjunctivitis, uveitis, retinitis
   d. skin lesions show papular pattern of pustules commonly on trunk and limbs
   e. diagnosis based on 2 of 3 manifestations (oral, ocular, genital)
   f. a pustular lesion that develops after a needle puncture with saline is highly suggestive of Behcet = pathergy
4. Treatment – with systemic and topical corticosteroids, chlorambucil used for ocular lesions, immunosuppressives used prn
III. Ulcerative Conditions
   A. Aphthous Ulcers (Recurrent Aphthous Stomatitis, RAS)
      1. Etiology
         a. unknown, but may be related to immunologic factors (defect in humoral immune system), microbiologic factors (HSV), nutritional factors (B2, folic acid deficiencies), etc.
      2. Clinical features
         a. three forms: minor, major and herpetiform
         b. painful recurrent ulcers
         c. occasional prodromal symptoms of tingling or burning
      3. Differential diagnosis - secondary oral herpes, trauma, Behcet's Syndrome
      4. Treatment and prognosis
         a. for minor - no treatment needed; can use 5% amlexanox oral paste (Aphthasol) or Rincinol p.r.n. mucosal coating; heals 7-10 days; no scar
         a. for major - corticosteroids, antibiotics (tetracycline oral suspension), combination of tetracycline oral suspension, nystatin oral suspension and hydrochloride elixer (Benadryl) may control disease, but no cure is apparent; sometimes laser surgery is helpful
         b. for herpetiform – tetracycline oral suspension or combination tetracycline and nystatin oral suspensions; heals w/in 7-10 days

   B. Erythema Multiforme
      1. Etiology
         a. unknown, but may be related to a hypersensitivity reaction to infections such as HSV, TB, or histoplasmosis, or to drugs such as barbiturates and sulfonamides
      2. Clinical features
         a. tends to be seasonal
         b. presents with cutaneous lesions; 25-50% have oral lesions
         c. classic lesion is the target or iris lesions consisting of concentric erythematous rings separated by rings of normal color tissue; the skin in the center of the lesions may be erythematous or tan, indicating resolution
         d. oral lesions tend to be multiple, superficial, wide-spread ulcers occurring anywhere in the mouth
         e. a variation of this disease is Stevens-Johnson Syndrome; a triad of ocular, oral and genital lesions
      3. Differential diagnosis – HSV, aphthous ulcers, pemphigus vulgaris, cicatricial pemphigoid. erosive lichen planus
      4. Treatment and prognosis
         a. corticosteroids with anti-fungals helps control disease
         b. supportive measures such as oral irrigation, adequate fluid intake, and use of antipyretics may provide substantial relief
         c. runs a course of 2-6 weeks, recurrences are common

   C. Systemic Lupus Erythematosis (SLE)
      1. Etiology
         a. autoimmune process may be influenced by genetic or viral factors
      2. Clinical features
         a. erythematous rash classically seen over the malar processes and bridge of nose - butterfly pattern; also effects face, trunk, and hands
         b. oral lesions include ulceration, erythema and keratosis usually on vermilion, buccal mucosa, gingiva and palate; healing of lesions may result in fibrosis/scarring;
lesions may have secondary infection of candidiasis

c. systemic expression involves fever, weight loss, malaise and progresses to joints, kidneys, heart, lungs and other vital organs

3. Differential diagnosis - oral lesions resemble erosive lichen planus, speckled erythroplakia, candidiasis

4. Treatment and prognosis
   a. systemic steroids, immunosuppressives, antimalarial and non-steroidal anti-inflammatory drugs  
   b. treat any secondary candida infections with antifungal meds
   c. use salivary substitutes if xerostomia is present
   d. clients taking steroids may require supplemental dosages during dental treatment
   e. complications occur due to vital organ damage and toxic effects of meds; mortality is due typically to renal failure or infection; pregnancy exacerbates condition in those with nephritis or htn

D. Basal Cell Carcinoma (Skin Cancer)

1. Etiology
   a. ultraviolet light

2. Types
   a. nodular – most common; nodular in shape with ulcerated center
   b. morpheaform – indistinct borders; prone to incomplete excision and recurrence
   c. superficial multicentric – eczematous patch that advances peripherally without deep invasion; found usually on the trunk
   d. pigmented
   e. basosquamous

3. Clinical Features
   a. most common cancer of the head and neck
   b. occurs in midface region most frequently
   a. presents as indurated, smooth nodules that eventually ulcerate, crust and erode adjacent structures
   b. pattern of appearing to heal and then recur

4. Differential Diagnosis – scrofula, squamous cell carcinoma (lip locations), melanoma, intradermal nevus, seborrheic keratosis

5. Treatment and prognosis
   a. surgery – excision, cryosurgery, electrosurgery, Moh’s chemotherapy
   b. radiation – used for poor surgical candidates, have multiple lesions or tumors in locations for which resection would cause significant morbidity
   c. slow growing and rarely metastasize; excellent prognosis – 90% cure rate

E. Squamous Cell Carcinoma

1. Etiology
   a. tobacco
   b. alcohol consumption
   c. infections (syphilis, HIVD, HPV subtypes 16 and 18, etc.)
   d. mucosal diseases (iron deficiency associated with Plummer-Vinson Syndrome, lichen planus, etc.)
   e. ultraviolet light (lip cancer)
   f. exposure to ionizing radiation, arsenic or industrial chemicals
   g. chronic irritation (poor oral hygiene, poor restorative dentistry)
   h. pre-existing burns and scars
   i. immunosuppression

2. Warning Signs
   a. a lump or thickening in the oral soft tissues
   b. soreness or difficulty in chewing or swallowing
c. ear pain
d. difficulty moving the jaw or tongue
e. hoarseness
f. numbness of the tongue or other areas of the mouth
g. swelling of the jaw that causes dentures to fit poorly or become uncomfortable
h. repeated bleeding from the mouth or throat
i. red, white, or discolored lesions in the mouth or on the lips

3. Clinical features
a. may be white, red, or speckled red and white patches, nodules, or ulcers
b. early tumors may be painless and vary in appearance
c. advanced lesions form indurated fixed ulcers with raised and rolled edges
d. tissue necrosis leads to pain and infection
e. metastases occur to regional lymph nodes and may spread to distant sites such as the lungs, brain and bones

4. Differential diagnosis – tuberculosis, syphilis, chronic trauma

5. Treatment and prognosis
a. surgery, radiation, chemotherapy or a combination depending upon lesion location, histologic type, etc.
b. side effects of treatment may include mucositis, pain, candidiasis, dermatitis, erythema, alopecia, xerostomia, cervical caries, osteonecrosis, etc.
c. prognosis depends on both the histologic subtype (grade) and clinical extent (stage) of the tumor
d. survival rate is 40-50%; approximately 20% (but as high as 40%) of oral cancer patients will develop a second primary lesion usually in the first-second year of follow-up; patients should undergo a thorough exam of the upper digestive tract and airway at regular intervals at the time of diagnosis and after treatment

IV. White Lesions

A. Hairy Leukoplakia

1. Etiology
   a. opportunistic infection related to the presence of the Epstein-Barr virus
   b. associated with subsequent or concomitant development of HIV

2. Clinical features
   a. asymptomatic
   b. variable appearance - irregular surface contour that is folded or corrugated, or smooth and macular
   c. majority are located on lateral borders of the tongue
   d. may have a superinfection of candidiasis
   e. herpetic ulcerations and lymphadenopathy may also be present

3. Differential diagnosis - idiopathic leukoplakia, candidiasis, lichen planus

4. Treatment and prognosis
   a. antiviral and antifungal medications used
   b. limited success with treatment
   c. prognosis influenced by HIV

B. Nicotine Stomatitis

1. Etiology
   a. smoking

2. Clinical features
   a. palatal mucosa is keratinized with red papules representing inflammation of the ductal elements of the underlying minor salivary glands
   b. may have an overall roughened appearance

3. Differential diagnosis - squamous cell carcinoma, idiopathic leukoplakia
4. Treatment and prognosis
   a. elimination of smoking - lesions may resolve
   b. increased risk for the development of malignancies in other regions especially retromolar or facial regions probably a result of the heat of the tobacco smoke

C. Geographic Tongue (Benign Migratory Glossitis, Erythema Migrans)
   1. Etiology
      a. unknown
   2. Clinical features
      a. small, round to irregular areas of dekeratinization and desquamation of filiform papillae
      b. the desquamated areas become red, slightly tender, with elevated margins showing a white or yellow-white border
      c. lesions appear to move across the dorsum of the tongue
      d. positive correlation exists with fissured tongue
   3. Differential diagnosis – candidiasis, leukoplaikia/erythroplakia, lichen planus, lupus erythematosus
   4. Treatment and prognosis
      a. symptomatic treatment with a topical steroid containing an antifungal agent may be indicated
      b. may recur

D. Lichen Planus
   1. Etiology
      a. epithelial basal cell damage related to a cell-mediated immune process involving Langerhans cells, T-lymphocytes and macrophages; autoimmune response
      b. possibly stress related
   2. Clinical features
      a. chronic inflammatory mucocutaneous disease that presents in a variety of forms – plaque, atrophic, erosive, bullous, reticular
      b. most common type, reticular form, is characterized by the presence of Wickham’s striae that produce a keratotic lacy pattern
      c. the erosive form is characterized by granular and bright erythematous surfaces that may bleed upon slight manipulation
      d. cutaneous lesions are found in 20-60% of patients presenting with oral lesions
   3. Differential diagnosis – candidiasis, leukoplaikia, squamous cell carcinoma, hairy leukoplaikia
   4. Treatment and prognosis
      a. no specific treatment is uniformly successful
      b. corticosteroids (such as tacrolimus and clobetasol) help control the disease but does not cure it
      c. periodic re-evaluation is necessary; 1% of cases may undergo malignant transformation

E. Candidiasis (Moniliasis, Thrush)
   1. Etiology
      a. Candida albicans
      b. opportunistic infection secondary to the short-term use of systemic antibiotic therapy for minor bacterial infections, and to severely debilitated and immunocompromised patients
   2. Clinical Features
      a. variable and numerous forms noted
         1. pseudomembranous (removable white plaques)
2. chronic hyperplastic (leukoplakialike plaques do not rub off)
3. chronic erythematous (patchy or diffuse mucosal erythema)
4. acute atrophic (numerous erosions and intense inflammation)
5. angular cheilitis

b. wiping away plaques will leave an erythematous, eroded or ulcerated surface that is tender

3. Differential diagnosis - chemical burns, traumatic ulcerations, mucous patches of syphilis, erosive lichen planus (red lesions)

4. Treatment and prognosis
   a. topical application of nystatin suspension or cream, or clotrimazol
   b. withdraw antibiotic therapy, if possible
   c. may need to use systemic administration of meds for immunosuppressed patients with associated candidiasis
   d. excellent prognosis, but may recur

V. Red-Blue Lesions
   A. Median Rhomboid Glossitis (Chronic hypertrophic/hyperplastic candidiasis)
      1. Etiology
         a. chronic infection by C. albicans
      2. Clinical Features
         a. red elevated rhomboid or oval lesion in the dorsal midline of the tongue anterior to the circumvallate papillae
         b. usually asymptomatic
      3. Treatment and prognosis
         a. symptomatic treatment with antifungal medications may be necessary

   B. Pyogenic Granuloma
      1. Etiology
         a. overreaction of connective tissue to a known stimulus or injury
      2. Clinical features
         a. red hyperplastic granulation tissue that may become ulcerated due to secondary trauma
      3. Differential diagnosis - peripheral giant cell granuloma, hemangioma
      4. Treatment and prognosis
         a. surgical excision
         b. occasionally recurs due to incomplete excision, failure to remove etiologic agent or reinjury to the area

   C. Epulis Fissuratum (Irritation hyperplasia/Inflammatory fibrous hyperplasia)
      1. Etiology
         a. chronic irritation, poorly fitting denture
      2. Clinical features
         a. initially produces an ulcer that heals incompletely due to repeated trauma
         b. hyperplastic healing results
         c. pink-red, fleshy exuberance of mature granulation tissue is produced
         d. located where denture flange rests
         e. slow growing lesion
      3. Treatment and prognosis
         a. surgical excision and denture adjustment
         b. prognosis good if both treatments applied, otherwise tendency to recur
D. Hemangioma
1. Etiology
   a. vascular neoplasms
2. Clinical features
   a. various types: vascular lesions and congenital lesions (capillary and cavernous)
   b. color ranges from red to blue
   c. when compressed, blanching occurs
   d. may be flat, nodular or bosselated
3. Treatment and prognosis
   a. careful surgical approach with selective arteriole embolization or sclerosant therapy
   b. good prognosis

E. Kaposi’s Sarcoma
1. Etiology
   a. genetic predisposition
   b. infection (viral)
   c. immunodisregulation (reduced immunosurveillance)
2. Types
   a. classic – seen in those of Mediterranean descent; 50-70 years of age; usually confined to lower extremities; oral involvement is rare
   b. african – can be similar to classic type or uniquely aggressive form that typically affects children with cutaneous and mucosal lesions as well as visceral organ and lymph node involvement; oral lesions are rare; death occurs from GI hemorrhage
   c. renal transplant-associated KS – oral cavity involvement occurs and appears associated with the degree of immunosuppression
   d. epidemic – seen in patients with AIDS; extremely virulent and deadly
3. Clinical features
   a. malignant neoplasm of endothelial origin
   b. single or multifocal reddish-brown or blue macules or nodules often associated with HIVD and other immunodeficiencies
   c. most common site is the palate (73%) followed by the attached gingiva (36%) and dorsal tongue (15%)
   d. clinical course is rapid and aggressive
   e. occurs in 20% of HIVD patients with half developing oral lesions; initially lesions are asymptomatic but become painful and bleed; careful examination of cutaneous surfaces is recommended
4. Differential diagnosis – hemangioma, melanoma, pyogenic granuloma
5. Treatment and prognosis
   a. surgery, low-dose radiation and chemotherapy, but none are uniformly successful
   b. modest success with intralesional injections of vinblastine and other compounds
   c. poor prognosis – average survival is 21 months; most death is due to opportunistic infections rather than a consequence of KS
VI. Tables of Medications

### Table I: Antiviral Medications

<table>
<thead>
<tr>
<th>Name of Medication</th>
<th>Use</th>
<th>Administration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Topical acyclovir (Zovirax®)</td>
<td>Recurrent herpes labialis</td>
<td>Cream formulation most efficacious ACV 5%; ointment generally not efficacious</td>
</tr>
<tr>
<td>Systemic (oral) acyclovir (Zovirax®)</td>
<td>Recurrent herpes labialis and in prophylaxis of lesions</td>
<td>ACV at 200-400 mg taken 5x/day for 5 days</td>
</tr>
<tr>
<td>Systemic (oral) valacyclovir (Valtrex®)</td>
<td>Recurrent herpes labialis and in prophylaxis of lesions</td>
<td>1000 mg bid may abort lesions</td>
</tr>
<tr>
<td>Penciclovir (Denavir®)</td>
<td>Recurrent herpes labialis</td>
<td>Topical 1% cream</td>
</tr>
<tr>
<td>Famciclovir (Famvir®)</td>
<td>Recurrent herpes labialis</td>
<td>125-500mg – does not suppress RHL, but lesions are smaller and last a shorter period of time</td>
</tr>
<tr>
<td>Topical docosanol (Abreva)</td>
<td>Recurrent herpes labialis</td>
<td>10% docosanol reduces healing time and pain; only OTC drug approved by the FDA</td>
</tr>
<tr>
<td>Topical foscarnet (Foscavi®)</td>
<td>ACV-resistant HSV infection</td>
<td>3% foscarnet cream</td>
</tr>
</tbody>
</table>

### Table II: Topical Prescription and OTC medications for Recurrent Aphthous Lesions

<table>
<thead>
<tr>
<th>Name of Medication</th>
<th>Administration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amlexanox oral paste (Apthasol)</td>
<td>5% paste, apply qid after meals and at bedtime</td>
</tr>
<tr>
<td>Triamcinolone Acetodnide dental paste (Kenalog in Orabase 0.1%)</td>
<td>Apply after each meal and at bedtime</td>
</tr>
<tr>
<td>Chlorhexidine oral rinse (Peridex,PerioGard)</td>
<td>Rinse with 20 ml for 30 seconds tid</td>
</tr>
<tr>
<td>Fluocinonide 0.05% (Lidex ointment mixed 50/50 with Orabase (30 grams total)</td>
<td>Apply when oral inflammatory lesions do not respond to Kenalog in Orabase; use thin layer 4-6x/day</td>
</tr>
<tr>
<td>Benzyl alcohol (Zilactin gel)</td>
<td>Apply q 3-4 hours</td>
</tr>
<tr>
<td>Benzocaine 10% (Zilactin B)</td>
<td>Apply q 3-4 hours</td>
</tr>
<tr>
<td>Lidocaine 2.5% (Zilactin L)</td>
<td>Apply q 3-4 hours</td>
</tr>
<tr>
<td>Diphenhydramine (Benadryl® Elixir)</td>
<td>Swish with 1 tsp for 2 min before each meal (can be swished and swallowed)</td>
</tr>
<tr>
<td>Benzocaine, gelatin, pectin and sodium carboxymethylcellulose (Orabase® with Benzocaine)</td>
<td>Apply 3-4 times/day</td>
</tr>
<tr>
<td>Benzocaine and 2-octyl cyanoacrylate (Colgate Sooth-N-Seal)</td>
<td>Dry lesion and apply q 6 hours</td>
</tr>
<tr>
<td>Tetracaine Hydrochloride 1% (Viractin)</td>
<td>Apply 3-4 times/day up to 7 days</td>
</tr>
</tbody>
</table>
Table III: Antifungal Medications

<table>
<thead>
<tr>
<th>Name of Medication</th>
<th>Administration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mycostatin® pastilles (nystatin)</td>
<td>4-5 troches/day for 14 days</td>
</tr>
<tr>
<td>Mycostatin® oral suspension (nystatin)</td>
<td>1 tsp 4-5 times/day; rinse and hold in mouth for 2 minutes before spit/swallow; do not eat/drink for 30 minutes</td>
</tr>
<tr>
<td>Mycostatin® ointment/cream or powder (nystatin)</td>
<td>Apply 4-5 times/day; apply to dentures prior to each insertion; do not eat/drink for 30 minutes</td>
</tr>
<tr>
<td>Mycelex® troche ( clotrimazole); contains sucrose: use caution in those with diabetes mellitus</td>
<td>Dissolve 1 troche in mouth 5 times/day for 14 days</td>
</tr>
<tr>
<td>Fungizone® oral suspension (amphotericin B); manufacturer is discontinuing</td>
<td>1 ml swish and swallow for 4 times/day for 14 days</td>
</tr>
<tr>
<td>Mycolog® II (nystatin and triamcinolone)</td>
<td>Apply at bedtime and after each meal</td>
</tr>
<tr>
<td>Diflucan® 100 mg (fluconazole) – systemic</td>
<td>200 mg immediately, then 100 mg daily for 10-14 days</td>
</tr>
<tr>
<td>Nizoral® 200 mg (ketoconazole) – systemic</td>
<td>200 mg/day for 10-14 days</td>
</tr>
</tbody>
</table>

Table IV: Examples of Salivary Replacement Therapy

<table>
<thead>
<tr>
<th>Products</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biotene® (Laclede Pharmaceuticals)</td>
<td>Toothpaste, oral gel, mouthrinse, chewing gum; contains 3 key salivary enzymes found in natural saliva – lactoferrin, lysozyme, lactoperoxidase; replicates natural salivary enzyme activity, is water based, xylitol can cause diarrhea</td>
</tr>
<tr>
<td>Orajel (Del Pharmaceuticals, Inc)</td>
<td>Moisturizing gel and spray, toothpaste; gel and spray contain 18% glycerin, sorbitol in the gel, xylitol in the spray, lasts up to 2 hrs; limited fermentation of sorbitol in toothpaste may increase caries risk</td>
</tr>
<tr>
<td>Oasis (GlaxoSmithKline)</td>
<td>Mouthwash or spray; TriHydra technology with hydrophilic polymers, xanthum gum, glycerine and carboxymethylcellulose; gives same feel as saliva, lasts up to 2 hrs, mint flavored</td>
</tr>
<tr>
<td>Nuvora</td>
<td>Saleve oral health lozenge, delivers essential oils and xylitol over several hours; sugar and alcohol free</td>
</tr>
<tr>
<td>Salagen (5 mg pilocarpine hydrochloride)</td>
<td>Cholinergic agonist, need to take drug for min 90 days to see optimum effects, multiple drug interactions</td>
</tr>
<tr>
<td>Evoxac (cevimeline)</td>
<td>Cholinergic agonist, use with caution in pts with cvd, asthma, copd, decreased visual acuity, elderly, those with kidney problems</td>
</tr>
<tr>
<td>Salivart®</td>
<td>Synthetic saliva in aerosol spray</td>
</tr>
<tr>
<td>Omnii products with xylitol</td>
<td>TheraSpray, Theramints, TheraGum</td>
</tr>
<tr>
<td>Mouthkote®</td>
<td>Contains mucopolysaccharide from Yerba Santa plant, closely mimics glycoproteins in saliva, lemon-lime taste</td>
</tr>
<tr>
<td>Tom’s of Maine Natural Anti-cavity Fluoride Mouthwash</td>
<td>Alcohol free fluoride mouthwash with xylitol, antioxidant green tea, aloe vera leaf juice, and chamomile</td>
</tr>
</tbody>
</table>
Selected References


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