FOREWORD TO THE FIFTH EDITION

Since its introduction over fifteen years ago, the Guide has helped thousands of practitioners and students do a more rapid job of diagnosing and treating oral diseases. This edition has been extensively updated to provide you with the most current data available in the critical area of oral pathology diagnosis and treatment. The Guide will help you quickly diagnose and treat the majority of oral diseases likely to be found in practice and will illustrate and discuss normal variations which are commonly mistaken for disease states.

The narrative discusses each disease state in terms of etiology, clinical appearance, and treatment of differential diagnosis. An additional reading list, which will direct you to more detailed discussions, is provided at the back of the Guide.

There are also excellent photographic references of what are considered “classic” examples of various oral diseases.

Complete slide sets of the material contained in this book are available through our offices at the School of Dentistry, University of Missouri-Kansas City. To obtain more information please write:

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**Foliate papillae**

*Fig. 1*

*Fig. 2*

**Description:** Foliate papillae appear as an area of vertical folds and grooves located on the extreme posterior-lateral surface of the tongue. They are occasionally mistaken for tumors or inflammatory disease. The grooves are best seen when air from an air syringe is directed at them. Their long axis is “up and down”, that is, they are at right angles to the long axis of the tongue. Our experience has been that they are bilaterally symmetrical, but one publication states this is not always so. In some people, the papillae are small and inconspicuous whereas in others they are prominent. Those familiar with the basic fold and groove structure are not apt to confuse these normal structures with an abnormality.

**Etiology:** They are normal anatomical structures.

**Treatment:** None required.

**Prognosis:** Good.

**Differential diagnosis:** Lingual tonsils, squamous carcinoma, soft tissue tumors.

**Lymphoid aggregates**

**Description:** Lymphoid aggregates appear as small, slightly elevated nodules that may be normal colored or more red than the surrounding mucosa. Those illustrated here are in the soft palate. They may be found anywhere in the mucosa but are especially common where the mouth meets the throat, including the base of the tongue. This lymphoid rich area has been called Waldeyer’s ring. When they occupy the same area as the foliate papillae, the papillae may take on a more nodular appearance. In the tongue they have been referred to as “lingual tonsils”.

*Fig. 3*

**Etiology:** They represent a variation of normal in which lymphoid tissue is found in ectopic locations.

**Treatment:** None required.

**Prognosis:** Good. They may enlarge or regress in tune with oral or upper respiratory infections.

**Differential diagnosis:** Foliate papillae are often mistakenly identified as lingual tonsils. Although papillae and lymphoid aggregates may occupy the same area, they are different entities.

**Varix (plural: varices)**

*Fig. 4*

**Description:** Varices appear as red, blue, or deep purple broad based elevations in oral mucosa. The size is usually less than 5 mm. The buccal mucosa is a common place to find them, however, they are also
found in lip mucosa and ventral and lateral mucosa of the tongue and floor of the mouth. On ventral tongue they are apt to be multiple and the term “caviar tongue” has been used to describe them. They are seen more commonly in the elderly.

**Etiology:** A varix is a distended vein which has bulged enough to elevate the overlying mucosa. The reason for venous distention is unclear but may be related to weakening of the vessel wall secondary to aging.

**Treatment:** None usually required. They often thrombose but this is of little clinical consequence.

**Prognosis:** Good

**Differential diagnosis:** Mucocele and hemangioma.

**Torus palatinus and torus mandibularis**

**Description:** Bony exostoses in the midline of the hard palate and on the lingual aspect of the mandible are referred to as torus palatinus and torus mandibularis respectively. Some studies suggest they are inherited. Fig. 5 shows a palatal torus and fig. 6 is a mandibular torus. They start in childhood and reach peak incidence in young adults. Once they have reached “programmed size”, their growth stops.

Some are so subtle they hardly constitute an abnormality whereas others are so large they frighten the uninitiated observer.

In the mandible, single masses are commonly seen. However, they may form a row of nodules as illustrated here. In some individuals they occur bilaterally. Those in the palate may become divided by deep grooves to form a cluster of nodules. Exostoses entirely similar to tori occur elsewhere on the alveolar bone but we have no specific name for them. It has been estimated that palatal tori occur in 20% of the population. Mandibular tori are less common, about 10% of the population are affected.

**Etiology:** Tori are developmental overgrowths and as previously stated they may be inherited.

**Treatment:** Tori and other exostoses seldom produce much in the way of patient complaints. Because they extend above the level of surrounding normal mucosa, they invite trauma. Small traumatic ulcers are therefore commonly seen on the mucosa covering tori. Tori may interfere with prosthetic appliances and for that reason may require removal.

**Prognosis:** Good

**Differential diagnosis:** Tori have such a characteristic clinical appearance and history that differential diagnosis is seldom a problem.

**Idiopathic osteosclerosis**

**Description:** Osteosclerosis is an area of dense but normal bone in the jaws. It may occur anywhere in the jaws and in some instances may appear to be attached to a tooth as seen in fig. 7. The shape ranges from round to linear streaks to occasional
angular forms. They are more common in the mandibular molar-premolar area. They are usually discovered on x-ray taken during the course of ordinary dental care.

![Fig. 8](image)

**Etiology:** Osteosclerosis is presumably of developmental origin although a reactive etiology is difficult to rule out in some cases.

**Treatment:** Because osteosclerosis is not a disease, no treatment is required. If there is doubt about the diagnosis, periodic x-rays should be taken. Osteosclerosis does not change with time.

**Prognosis:** Good

**Differential diagnosis:** Condensing osteitis.

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**Osteoporotic bone marrow defect**

![Fig. 9](image)

**Description:** As the name implies this is a localized increase of marrow elements which create a radiolucent radiographic defect. They occur more commonly in women in the midyears and show a predilection for the molar region of the mandible. They are especially common in extraction sites. Radiographically they seldom have as sharp a border as cysts and tumors. Scattered trabeculae may extend short distances into the defect or in some instances through it giving the defect a fairly characteristic appearance. Naturally there are no associated clinical symptoms.

**Etiology:** The exact etiology remains unknown. No connection has been found linking the osteoporotic bone marrow defect with systemic disease or systemic need for increased blood production.

**Treatment:** Once the diagnosis is established no treatment is required.

**Prognosis:** Good

**Differential diagnosis:** This defect may easily be mistaken for cysts or tumors. In those cases where there is doubt about the diagnosis, biopsy should be done.

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**Fordyce granules**

![Fig. 10](image)

**Description:** Fordyce granules appear as flat or elevated yellow plaques or grains just beneath the mucosal surface. The most common site is buccal mucosa although they may be found anywhere in oral mucosa. Development of the oral glands parallels those of the skin, reaching maximum numbers at puberty. Eighty percent of the population are affected. The number of granules is quite variable. Fig. 11 illustrates a large number of granules on buccal mucosa.

**Etiology:** They are ectopic sebaceous
glands, presumed to be a developmental anomaly.

**Treatment:** None required.

**Prognosis:** Good

**Differential diagnosis:** The clinical appearance is so characteristic that differential diagnosis is not a problem.

**Leukoedema**

![Image of Leukoedema](image)

**Description:** Leukoedema appears as a filmy, opaque, white to slate gray discoloration of mucosa, chiefly buccal mucosa. Redundancy of the mucosa may impart a folded or wrinkled appearance to the relaxed mucous membrane. It partially disappears when the mucosa is stretched. It is stated to be seen in 90% of Blacks and 40% of Whites, although in our experience the latter figure is high.

**Etiology:** Leukoedema is a variation of normal which should not be confused with something ominous. Intracellular edema of the superficial epithelial cells coupled with retention of superficial parakeratin is thought to account for the white appearance.

**Treatment:** None required.

**Prognosis:** Good

**Differential diagnosis:** White sponge nevus, hereditary benign intraepithelial dyskeratosis, and dyskeratosis congenita. All are extremely rare.

**Aphthous stomatitis (Canker sore)**

**Description:** This is one of the most common oral diseases. The exact incidence is unknown but estimates range from 20% to 60% of the population. Lesions appear as painful ulcers ranging in size from less than 1 mm to 2 centimeters. They may be single or multiple. Small lesions (less than 0.5 cm) have been referred to as minor aphthae (Fig. 13) and large lesions (more than 0.5 cm) have been called major aphthae (Fig. 14). Major aphthae are also known as Sutton’s disease. An uncommon variety of this disease appears as multiple, pinpoint areas of ulceration that seldom exceed 1 mm (Fig. 15).

![Image of Aphthous Stomatitis](image)

Each lesion begins as a red macule, less often a papule but not as a blister. It soon ulcerates and the ulcer becomes covered by a pyogenic membrane producing the characteristic yellow-white center with surrounding erythematous flare. The shape is usually round to oval but may be elongated in natural folds such as the vestibule.

Aphthous stomatitis occurs on freely movable mucosa which does not overlie bone. The lips, cheeks, vestibule, soft
palate, floor of mouth, ventral and lateral tongue are often involved but gingiva, hard palate and dorsal tongue are seldom affected.

Aphthous lesions affect all age groups from young to old but young adults, especially women, are more affected. Elapsed time between recurrences is extremely variable; some unfortunate patients have almost continuous disease whereas others go from months to years between episodes.

Etiology: The cause is unknown. The concept that canker sores are caused by an L form of Streptococcus sanguis has been superceded by theories revolving around an immunopathogenesis. The deposition of antibodies and complement within epithelium and basement membrane during the early stages of the disease suggests a humoral immune response and the influx of lymphocytes rather than neutrophils in early lesions points to a cellular immune reaction as well. It is yet to be learned if the immune response is directed against self (autoimmunity) or against an extrinsic antigen such as bacteria or viruses. To further cloud the issue, a variety of other factors have been implicated. Withdrawal of certain foods such as cheese, tomato products and gluten has been claimed to help some patients whereas in others, correction of iron, B12 and folate deficiencies have brought about a cure. Improvement of aphthous lesions during the last stages of pregnancy with exacerbation after delivery suggests that gonadal hormones may play a role. The appearance of canker sores which appear during menstruation also suggest a hormonal basis. To add a final element of mystery, aphthous stomatitis has been reported to worsen when cigarette smoking is discontinued.

There are too many theories for them all to be correct. Aphthous lesions, like cancer, may not be a single disease with a single cause but instead, a variety of diseases all manifest by painful mouth sores.

Treatment: Patients with few lesions are treated with topical analgesics or anti-inflammatory agents such as Orabase® with Benzocaine, Orabase® HCA Oral Paste or trimcinolone acetonide dental paste USP 0.1%. For stronger anti-inflammatory action, fluocinonide ointment is recommended. For severe disease, favorable results have been reported using dexamethasone elixir mouthwash combined with azathioprine tablets 50 mg. bid and ibuprofen 600 mg. qid. A word of caution: azathioprine is a powerful immuno-suppressant and should not be prescribed by those not experienced in its use. Prednisone is a safer immune suppressing drug. For moderate to severe disease, we prescribe 20 mg. daily for 7-10 days. Patients should be cautioned about adverse effects of steroid therapy including osteoporosis, aseptic necrosis of the femoral head, cataracts, fluid retention, depression, increased appetite and exacerbation of diabetes. There is little risk of inducing the Cushingoid syndrome in short term therapy.

Prognosis: Cure is seldom achieved but palliation and long term remission may be achieved by above mentioned treatment. Without treatment, healing time varies from 4 days for a small lesion to a month or more for major aphthae. Major aphthae may also cause scarring.

Differential diagnosis: Aphthous stomatitis must be differentiated from herpetic stomatitis, the disease with which it is most often confused. Recurrence is a feature that helps to differentiate aphthous stomatitis from intraoral herpes infections. Intraoral recurrences from herpes virus infections are uncommon.

Lesions indistinguishable from aphthous stomatitis have been reported in Behcet’s syndrome, Reiter’s syndrome, Crohn’s disease and celiac disease.

Herpesvirus infections

Description: Oral infection with herpesvirus occurs in three clinical forms. The most common type consists of recurrent small blisters on the lips commonly referred to as fever blisters or secondary herpes labialis. The second type is a generalized oral infection called primary herpetic stomatitis. The third and least common form of oral herpes infection
consists of small ulcers usually localized on palatal mucosa.

Herpes labialis is illustrated in (Fig. 16 and 17). This lesion is well known and unlikely to be a diagnostic problem. It tends to be a recurrent disease in teenagers and adults. Elapsed time between recurrences varies from person to person. Recurrences are thought to be triggered by exposure to sunlight, febrile diseases, physical and psychogenic trauma, and other irritants.

Generalized involvement of the oral mucous membrane is called primary herpetic stomatitis (Fig. 18 and 19). It is more commonly seen in children, but teenagers and adults are also affected. Patients initially have gingivitis with swollen and red marginal gingiva then small blisters appear throughout the mouth. The blisters break so quickly they are seldom seen by the dentist or physician. After they break, the lesions appear as small ulcers which resemble small aphthous lesions. This generalized infection is accompanied by fever, cervical lymphadenitis, and inability to eat or drink without considerable pain.

Patients who suffer localized intraoral herpes are few in number. For reasons yet to be explained, recurrent intraoral herpes infections tend to occur as small ulcers, mainly on the hard palate mucosa as shown in (Fig. 20).

Etiology: Herpesvirus hominis. Most oral lesions are caused by Type I virus but approximately 10% are thought to be caused by Type II.
Treatment: Acyclovir has shown promising results in the treatment of both first episode and recurrent genital lesions with expectations of the same for oral lesions. Acyclovir ointment (5%) applied 5 times daily to lip lesions shortens healing time by one day, and if applied early, increases the number of abortive lesions. Systemic acyclovir reduces both duration and symptoms of first episode genital lesions and markedly reduced the recurrences. No similar have been reported with oral lesions. (A 0.1% aqueous solution of idoxuridine sprayed into the mouth 3 times each hour has been reported to be effective but needs confirmation in a larger number of patients.)

A number of treatments are of questionable value. These include topical ether, biotinoid-ascorbic acid complex and intramuscular adenosine. Photodynamic inactivation therapy has been abandoned because of possible carcinogenicity. Lysine has been shown to be useless.

Prognosis: The outlook is good but lip lesions commonly reoccur. Once the virus has entered the body, it travels through nerve trunks to the nearest ganglion where it may lie dormant for the remainder of the patient’s life. Future recurrences are thought to be brought about by the “reawakening” of the virus which retraces its steps to cause new lesions in the same general area as the original point of entry. Thus, each recurrence is not a new and different infection from the outside but a recurvulence of the original infection. The ability of the virus to literally hide in deep ganglia makes total eradication almost impossible and will likely frustrate attempts at treatment for the foreseeable future.

Patients with widespread herpetic stomatitis should maintain liquids to prevent dehydration. A broad-spectrum antibiotic is commonly given to control secondary bacterial infection.

Clinicians should be aware that the herpesvirus may cause disseminated infection including encephalitis in which case the prognosis is extremely grave.

Differential diagnosis: Primary herpetic stomatitis may resemble oral lesions of erythema multiforme, but herpes can be diagnosed by exfoliative cytology. A characteristic multinucleated cell appears in the smear of herpes infections. Culture of the virus is possible if a viral laboratory is available. Lesions of herpangina and hand, foot, and mouth disease, both caused by Coxsackievirus, may clinically resemble oral herpes virus infections.

Papillary hyperplasia (PH) and denture sore mouth (DSM)

Description: Long treated as separate entities, there is evidence that PH and DSM may be different expressions of the same disease. Both are related to the wearing of dentures. The mildest form of denture sore mouth appears as small, localized and asymptomatic red spots on the posterior palatal mucosa. As the condition worsens, large confluent areas turn crimson red (Fig. 21). This is the classic form of DSM. In later stages, hyperplasia of palatal mucosa occurs and produces the red, pebbly appearances of papillary hyperplasia (Fig. 22). In some cases of PH, the mucosa has a more mossy than mulberry appearance and the hyperplasia is not apparent until a gentle
blast of air opens the crevices revealing the papillary nature of the lesion. Whether or not DSM always evolves into PH is uncertain.

Etiology: The cause is unknown but there is evidence that Candida albicans is at least contributory. DSM has been called chronic atrophic candidiasis. Organisms are found more often in PH and DSM than in normal controls. Treatment with the antifungal drugs, nystatin and amphotericin B has been reported to bring about remission in most cases, especially in DSM, but recurrences are common when the treatment is discontinued. Since organisms have been shown to colonize the tissue surface of the denture, sterilization of the denture with fungicide is indicated.

Factors other than Candida albicans seem to be involved but it is difficult to assess the role of denture trauma and bacterial pathogens. Because the disease is limited to the area covered by the denture, it is often assumed that the patient is allergic to denture base material. There is little evidence to support this view. Patients with palatal lesions ordinarily do not have lesions under the lower denture contrary to what would be expected if the patient were truly allergic.

Treatment: We know of no effective therapy other than nystatin or clotrimazole in the usual dose for oral candidosis. Good oral and denture hygiene may help. The denture should fit well and not be worn at night. In cases of excessively redundant papillary hyperplasia, surgical excision to the periosteum may provide a better denture base.

Prognosis: Good, the condition is benign. For many years papillary hyperplasia had the undeserved reputation of being premalignant.

Differential Diagnosis: The disease has such a characteristic appearance that diagnosis is seldom a problem.

Epulis fissuratum
(Inflammatory fibrous hyperplasia)

Description: This lesion occurs in those who wear dentures. The lesion consists of two or more folds of soft tissue separated by

Fig. 23

a central groove into which fits the denture border (Fig. 23). It most often is found in the buccal vestibule of the anterior maxilla but any mucosal area touched by a denture border is vulnerable including the lingual aspect of the mandible. In a study of 583 cases, 64% were found in females. Those in the fifth and sixth decade are most often affected. Duration ranged from 1 week to 10 years, 40% of the patients reported a duration of 6 months to 2 years. Symptoms are absent except in ulcerated lesions which may be painful. Histologically the excessive tissue is composed of cellular, inflamed fibrous connective tissue.

Etiology: This is an inflammatory fibrous hyperplasia of oral mucosa caused by an over-extended denture border.

Treatment: Surgical excision of the lesion and reduction of the denture border.

Prognosis: Good

Differential diagnosis: The lesion has such a characteristic clinical appearance that differential diagnosis is not a problem. In questionable cases, biopsy should be done.

Irritation fibroma (traumatic fibroma)

Fig. 24

Description: Traumatic fibroma is a dome-shaped soft tissue mass usually
found on buccal mucosa along the line of occlusion. Less frequently they may be found on lips and tongue. They are among the most common oral soft tissue lesions. The color is usually the same as the surrounding mucosa and the consistency is surprisingly soft. Patients are generally aware of the lesion being present months to years with little change. Histologically, they are fibrous hyperplasia which is collagenous and relatively acellular.

**Etiology:** The presumed etiology is trauma to the affected mucosa. Accidental biting probably accounts for most of these lesions.

**Treatment:** Excision

**Prognosis:** Good

**Differential diagnosis:** Salivary gland neoplasm, varix, and hemangioma.

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### Papilloma

**Description:** Papillomas appear as pedunculated or sessile, white or normal colored cauliflower-like projections that arise from the mucosal surface. In a study of 464 oral papillomas, it was learned that the average size is less than 1.0 cm, only 8% were larger than 2.0 cm. Our experience has been that they are smaller, many are only 3 or 4 millimeters. The same study revealed the mean age of patients with papilloma was 36.4 years with a range from 2 to 91 years. There was no strong sex preference. The most common site was the palate-uvula area followed by tongue and lips. Of all sites, the soft palate was the most common and accounted for 20% of the lesions. The durations ranged from weeks to 10 years but 50% of the papillomas were present between 2 to 11 months.

**Etiology:** The etiology remains unknown. Viral origin has always been suspect but studies are still inconclusive. Immunoperoxidase techniques have identified antigens of the human papilloma virus (HPV) in a small number of cases. The same virus may be found in verruca vulgaris, condyloma acuminatum and focal epithelial hyperplasia all of which may resemble papillomas both clinically and microscopically.

**Treatment:** Conservative surgical excision, recurrence is rare.

**Prognosis:** Good, there is no evidence that papillomas are premalignant.

**Differential diagnosis:** The rare intraoral verruca vulgaris and condyloma acumi-
natum resemble papillomas and microscopic examination may be required to distinguish between them. Large papillomas may resemble early verrucous carcinoma.

**Peripheral fibroma**

**Description:** This lesion appears as a mass of tissue arising from the gingiva adjacent to teeth or between teeth. It favors children and young adults. Those arising between teeth may separate the teeth and produce pressure resorption of the interdental bone. It's not unusual to see a “saddle” lesion straddling the ridge with a labial and lingual lobe. Color is normal or slightly red.

![Fig. 27](image)

Histologically the bulk of this lesion is moderately cellular fibrous connective tissue frequently containing foci of bone, cementum, or dystrophic calcification. When inflammation is present, plasma cells frequently predominate.

**Etiology:** Unknown

**Treatment:** Excision. They may recur. Extraction of the adjacent teeth is seldom necessary.

**Prognosis:** Good

**Differential diagnosis:** Peripheral fibroma bears a great resemblance to pyogenic granuloma and peripheral giant cell granuloma. Histologic examination may be necessary to distinguish between them.

**Pyogenic granuloma**

**Description:** The pyogenic granuloma is a red, nodular overgrowth of granulation tissue that arises from the mucosal or skin surface. Approximately two-thirds of oral lesions are found on the gingiva followed in descending order by the lips, tongue, buccal mucosa, palate, vestibule and edentulous areas. The interdental papilla of the maxillary facial gingiva is the single most common site. A review of more than 800 cases disclosed the mean size to be approximately 1.0 cm. with a range of 3 mm. to 4 cm. Females were more often affected (72%). Duration varied widely with a mean of 5.5 months. Because of the vascular nature of pyogenic granuloma, they bleed easily and some cause mild pain. They commonly develop during pregnancy. The association with pregnancy is so common that the lesion has also been called granuloma gravidarum or pregnancy tumor. Because pus is infrequently found in this lesion, the term pyogenic granuloma is a misnomer but remains the preferred term.

![Fig. 28](image)

**Etiology:** The stimulus that provokes this overgrowth of granulation tissue is unknown although mild trauma and infection are prominently mentioned.

**Treatment:** Conservative excision.

**Prognosis:** Good. Recurrence is infrequently seen.

**Differential diagnosis:** Peripheral giant cell granuloma and peripheral fibroma.
Peripheral giant cell granuloma

Fig. 30

Description: The peripheral giant cell granuloma appears as a nodular red soft tissue mass arising from gingiva or alveolar mucosa. Most are approximately a centimeter in size, although they may be larger. Most patients are under 30; lesions are more common in females. There is almost equal distribution between maxillary and mandibular gingiva. The term "peripheral" is included in the name to separate this lesion from a histologically similar lesion which occurs inside the jaws. Jaw lesions are referred to as the "central" giant cell granuloma. The peripheral granuloma may cause pressure resorption of underlying alveolar bone and less commonly resorption of the adjacent tooth. They are not painful. Histologically this lesion consists of fibroblasts and multinucleated giant cells.

Etiology: Unknown

Treatment: Conservative excision

Prognosis: Good. The lesion should be followed since recurrence is occasionally seen.

Differential diagnosis: Pyogenic granuloma and peripheral fibroma.

Traumatic ulcer

Fig. 31

Description: An ulcer by definition is a localized area on the skin or mucosa in which the surface epithelium has been destroyed. The shape and size of traumatic ulcers are so variable as to defy a simple description. They are usually painful and of short duration.

Etiology: Common causes of traumatic ulcers include: denture irritation, biting injuries, hard foods, chemicals, toothbrush and dry cotton rolls.

Treatment: The treatment is to remove the cause if it is known. Relief of pain can be achieved with topical anesthetics such as Orabase-B® with Benzocaine.

Prognosis: The ulcer should heal if the cause is removed. An ulcer which does not heal within two to three weeks should be biopsied to rule out malignancy.

Differential diagnosis: Traumatic ulcers must be differentiated from squamous carcinoma and ulcerative mucosal diseases such as lichen planus.

Nicotine stomatitis

Fig. 32

Description: The classic form of this disease occurs in the palate of those who smoke pipes. The palatal mucosa is white and criss-crossed by fissures, giving the appearance of a dried creekbed. Small red elevations scattered throughout the lesion are thought to be inflamed orifices of minor salivary gland ducts. It produces no symptoms and may be discovered in a routine oral examination.

Etiology: This lesion is caused by smoking, chiefly pipe smoking. A report of thermally induced "nicotine" stomatitis in a woman who drank scalding hot tea and soup suggests heat rather than tobacco products are responsible for this condition.

Treatment: This disease usually disappears after discontinuance of smoking.
**Differential diagnosis:** The clinical appearance of nicotine stomatitis coupled with a history of pipe smoking is virtually diagnostic. Biopsy is seldom necessary.

**Dilantin gingival hyperplasia**

**Description:** Dilantin has been used for 50 years to control convulsive disorders. Approximately 50% of those who take it develop fibrous overgrowth of the gingiva. Within 2 weeks to 3 months after initiation of therapy, there is enlargement of the interdental papillae and marginal gingiva. This may progress to the point that the teeth are virtually submerged. Uncomplicated hyperplasia produces a firm pink growth; superimposed gingivitis or periodontitis may cause the gingiva to become boggy and red masking the true nature of the lesion. The anterior gingiva is more severely affected than are posterior and lingual areas. Gingival growth is most pronounced in the first year of therapy. Children and adults under 30 are more susceptible to the condition than are those who are past 30 when treatment begins. Most authors agree that unclean teeth may contribute to the enlargement and scrupulous dental hygiene is required to minimize the inflammatory component of the condition. The term Dilantin hyperplasia is an inappropriate term. Dilantin is the Parke-Davis Company’s proprietary name for phenytoin (5,5-diphenylhydantoinate).

**Etiology:** Phenytoin therapy. As stated above, the condition may become aggravated by superimposed gingivitis and periodontitis. There is evidence that phenytoin may impair the secretion of collagenase by gingival fibroblasts permitting the accumulation of excessive gingival collagen.

**Treatment:** The inflammatory component may be reduced by good dental hygiene. The fibrous overgrowth requires surgical removal. Discontinuance of phenytoin therapy may result in gradual regression of the overgrowth within one year.

**Prognosis:** Good

**Differential diagnosis:** Hereditary gingival fibromatosis, hyperplastic gingivitis from dental neglect, leukemic infiltrates of the gingiva, hyperplasia associated with cyclosporin A therapy and calcium channel blockers such as nifedipine, diltiazem and verapamil.

**Geographic tongue (benign migratory glossitis)**

**Description:** The lesions of this disease are so characteristic that recognition is instantaneous. The dorsal tongue displays map-like areas that are smooth and red with a whitish-yellow rim at the perimeter. Atrophy of filiform papillae accounts for the red areas. Parakeratin accumulation is seen in the rim. The disease may involve...
any oral mucosal surface in which case the name “stomatitis areata migrans” is given.

All ages are affected. We have seen it in a child six months old. The number of lesions varies from one to many. Old lesions heal and new ones form, waxing and waning in rhythm with unknown forces. Most patients are without symptoms which is fortunate, since there is no known successful treatment for the disease. Some complain of a mild burning sensation.

Etiology: Unknown
Treatment: None is required.
Prognosis: This is a chronic disease lasting months to years which may show periods of remission and exacerbation.
Differential diagnosis: The lesions are characteristic and differential diagnosis is not a problem.

Lichen planus

Description: Mucosal lesions of this disease appear in three forms: reticular, erosive and plaque. In the reticular type (Fig. 36) a pattern of lacy, white lines is characteristic. In the erosive type, the same reticular pattern is seen but there are areas of ulceration (Fig. 37). This is the most common form of lichen planus. In the plaque type, the lacy pattern is lost and the lesion appears as a solid white lesion. Rarely, vesicles will form. Skin is more frequently affected than is mucosa. Early skin lesions appear as red, maculopapular pruritic areas a few millimeters in diameter. Oral lesions may occur on any surface but the buccal mucosa is the most common site.

Etiology: The cause is unknown but a hypersensitivity reaction is suspected. Recent reports discount the role of stress. The role of mercury is unclear, but there are accounts of lichen planus-like lesions adjacent to amalgam restorations that heal when the fillings are replaced with non-mercury containing restorations. There are also reports of composite filling materials producing a similar effect. Stomatitis caused by drugs may resemble lichen planus; thiazide diuretics are chief offenders.

Treatment: The reticular and plaque types of the disease are ordinarily asymptomatic and require no treatment. In the erosive or ulcerative variety, relief is achieved with topical steroids. If ulceration is too widespread to control with topical treatment, systemic prednisone is indicated. Topical tretinoin 0.1%, a metabolite of vitamin A has been reported to be beneficial as have rinses with cyclosporin A.

Prognosis: The outlook is variable. The disease may last for years, few patients experience spontaneous remission. Topical steroids and vitamin A analogs provide relief but not a cure. Systemic steroids are effective but there are side effects and the disease may recur following discontinuation of therapy. There are reports that lichen planus predisposes the patient to oral cancer. The risk is placed at approximately 1%. The premalignant nature of lichen planus is not universally accepted. Some authors cite evidence that examples of lichen planus turning into cancer were originally dysplastic lesions masquerading as lichen planus. Such lesions have been referred to as lichenoid dysplasia. Until the dispute is settled, it is prudent to advise patients to have regular oral examinations for as long as they have the disease. Some clinicians recommend a biopsy of all patients with lichen planus.
**Differential diagnosis:** Lupus erythematosus, benign mucous membrane pemphigoid, leukoplakia (keratosis), erythroplasia, candidosis and lichenoid drug eruptions.

**Angular cheilosis**

**Description:** This lesion appears as fissuring and maceration at the commissures of the lips. The term cheilitis and cheilosis have both been used to describe the same disease.

![Fig. 38]

**Etiology:** Experimental subjects on a riboflavin deficient diet develop angular cheilosis. It is doubtful that vitamin deficiency contributes to the disease in the United States. Two factors may work in concert to produce angular cheilosis. Overclosure from the loss of vertical interdental dimension creates a moist skin fold at the commissures. This area is susceptible to infection with oral and skin bacteria and fungi. *Candida* is thought to be one of the major pathogens.

**Treatment:** In those patients who have obvious overclosure, restoration of vertical dimension is of benefit. Application of nystatin ointment to eliminate *Candida* organisms is indicated.

**Prognosis:** Good

**Differential diagnosis:** The disease is so characteristic that it cannot be confused with any other lesion.

**Candidosis (Candidiasis, Moniliasis, Thrush)**

**Description:** Infection with *Candida albicans* is known as candidiasis or candidosis.

There is virtually no organ or tissue immune to this fungus but skin, mouth, and genital lesions are most common. Severity of infection varies from small localized areas to generalized stomatitis. Involved mucous membrane develops a white slough consisting of necrotic mucosa and organisms. Because of uneven distribution of lesions, a speckled white on red appearance is common. In contrast to most other white lesions, the white can be wiped off leaving a denuded red surface that usually bleeds.

![Fig. 39]

**Etiology:** As previously stated, this disease is caused by an infection with *Candida albicans*. It is frequently stated that this disease occurs in four groups: (1) the very young; (2) the very old; (3) those with reduced resistance and (4) those on long term antibiotic therapy or immunosuppression.

**Treatment:** There are several topical and systemic drugs available. One of the more common treatments consists of a mouthwash of nystatin oral suspension 400,000 to 600,000 units four times daily for at least one week. This is the adult dose. The drug works by direct contact and thorough swishing in the mouth for 3-5 minutes before swallowing is mandatory. Recently a clotrimazole troche has become available. Each troche is 10 mg, adult dosage is 1 troche dissolved in the mouth 5 times each day for 14 days. Ketoconazole (Nizoral), a systemic antifungal drug is available as a 200 mg tablet, adult dosage is one each day. Ketoconazole has been shown to be toxic to the liver. Long term use requires monitoring of liver function.
should be screened for diabetes or other immunosuppressive diseases such as leukemia.

**Differential diagnosis:** Oral infection with Geotrichum is said to be identical to candidosis. Culture is necessary to distinguish between them.

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**Pulpitis**

**Description:** Inflammation of the pulp is one of the most common lesions seen in dental practice. No illustration is shown because pulpitis is not amenable to clinical photography. Pulpitis usually causes a toothache (pulpalgia) ranging from mild to excruciating although, in some cases, there are no symptoms.

**Etiology:** Pulpitis is usually brought about by pulp infection occurring as a consequence of caries, but may also be caused by physical trauma, dental instrumentation, and irritating restorative materials used by dentists.

**Treatment:** Treatment consists of elimination of the responsible agent. Irreversible cases are treated by endodontic procedures or extraction.

**Prognosis:** The prognosis depends on the extent and severity of the pulp involvement.

**Differential diagnosis:** It is frequently stated that periodontal infections, sinusitis, and referred pain may masquerade as pulpitis.

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**Dental caries**

**Description:** Caries ranks alongside gingivitis and periodontitis as one of the most common oral diseases. Treatment of these diseases and their sequelae constitutes the bulk of the practice of dentistry. Caries is the only disease that attacks that portion of the tooth exposed to the oral environment. For those who read this and may not be familiar with caries a photograph is shown in (Fig. 40). Typical caries are most commonly located in the occlusal pits and fissures of molars and premolars, as well as beneath the contact points on the interproximal surfaces. Special consideration should be given to two types of caries namely radiation caries and nursing bottle caries. Radiation type caries characteristically occur as multiple lesions in the cervical region of the teeth immediately adjacent to the gingiva. These may completely encircle the tooth causing amputation at the gingiva (Fig. 41). Nursing bottle caries are multiple rampant lesions that occur in deciduous teeth of nursing infants and small children (Fig. 42).

**Etiology:** Caries is caused by the decalcification of the tooth enamel and destruction of the protein matrix. Acid produced by bacteria, mainly Streptococcus mutans, in dental plaque is the precipitating factor. After the enamel is destroyed bacteria enter the dentin and may extend to the pulp of the tooth. Radiation or cervical caries are usually related to xerostomia and/or chem-
ical changes in saliva. Radiation therapy to
the head and neck region usually exceeding
4000 rads, is the most common cause.
However, many common medications
and systemic chemotherapy may also cause
dry mouth and radiation type caries. Nurs-
ing bottle caries are due to frequent nursing
with solutions containing high concentra-
tions of sugar such as milk, soft drinks,
and juices. These solutions allow bacteria
to break down the sugars to produce exces-
sive acid thus lowering the pH of both
plaque and saliva.
Treatment: Prevention is the best treat-
ment. Most caries can be prevented by
daily brushing and flossing to remove
plaque. A well-balanced diet without exces-
sive sugars is also beneficial. Topical
and systemic fluoride are highly effective
in reducing caries, especially if given
during the formative years of the teeth.
Fluorides have significantly reduced the
incidence of caries in the United States in
the last several decades. Radiation type
caries can also be prevented by a daily
regime of topical fluoride. Fluoride treat-
ments either brushed on or applied with a
custom mouth guard should begin as soon
as radiation or chemotherapy is started or if
xerostomia is discovered. Patients who
have received head and neck radiation
should continue daily treatments for life to
prevent possible osteoradionecrosis.

Once caries have developed, dental restorative procedures are the only treat-
ment, although there is now evidence that
very early lesions, under intact surface
enamel (white spots), may be remineral-
ized. Topical fluoride enhances this pro-
cess.

Prognosis: Prognosis is good if the disease
is treated early. If ignored caries is a major
cause of tooth loss, and a potential source
of both local and systemic infections.

Differential diagnosis: None

Marginal gingivitis

Description: Inflammation of the marginal
gingiva is among the mildest but most
common human ailments. The free margi-
nal gingiva is slightly swollen, red and
bleeds easily. It is not painful. It may show
patchy involvement with skip areas or it
may involve virtually the entire marginal
gingiva. If untreated, some patients show
progression to bulky enlargement of the
gingiva called hyperplastic gingivitis (fig.
44). Gingivitis is stated to be enhanced by
pregnancy and puberty.

Etiology: The gingival inflammation is
caused by the irritating effects of bacterial
plaque located on the adjacent tooth
surface.

Treatment: Treatment consists of cleaning
the teeth and the practice of good oral
hygiene.

Prognosis: The disease is easily treated and
the prognosis is good. If ignored, the in-
flammation may spread to deeper peri-
dontal tissues in which case the patient
is said to have periodontitis.

Differential diagnosis: Desquamative gingi-
vitis, dilantin hyperplasia, and leukemic
infiltrate.

Necrotizing ulcerative gingivitis
(Vincent's infection, trench mouth)

Description: This is a specific type of infec-
tion of oral mucosa whose lesions are
found chiefly on marginal gingiva. Ne-
crosis of the interdental papillae which spreads to involve adjacent facial and lingual marginal gingiva is virtually diagnostic. The ulcerated gingiva is covered by creamy exudate. Patients have pain and halitosis and in severe cases, fever and cervical lymphadenitis. Spread of lesions into the throat has been referred to as Vincent's angina. The disease occurs in all age groups but is uncommon in children.

**Etiology:** Isolation of *Borrelia Vincentii* and *Fusobacterium fusiformis* from the lesion is reported. They are suspected of being the chief etiologic agents although reinoculation of these organisms into tissues of volunteers has not reproduced the disease. Reduction of patient resistance is thought to play an etiologic role.

**Treatment:** Treatment consists of debridement and cleaning the teeth plus a broad spectrum antibiotic in those with fever and cervical lymphadenitis. Topical anesthetics may provide palliation.

**Prognosis:** Good

**Differential diagnosis:** Agranulocytosis, cyclic neutropenia, and acatalasia.

**Periodontitis**

**Description:** Extension of gingival inflammation into the underlying bone and periodontal ligament is referred to as periodontitis. It is best seen on radiographs since bone resorption is the outstanding feature. Periodontitis is a silent disease with an occasional acute exacerbation in the form of local, painful abscesses. The gingiva is usually inflamed. The chief indicators of this disease are increase in gingival sulcus depth as determined by gingival probing, and loss of alveolar bone as seen on radiographs. The conventional form of this disease starts in the teens or early adult years and without treatment shows gradual progression throughout life. It is the major cause of tooth loss in adults.

Other than common, conventional periodontitis, three subtypes have been identified: (1) rapidly progressive periodontitis (RPP) affecting young adults; it resembles the conventional disease except that bone destruction is accelerated and occurs over a period of weeks or months rather than years; (2) juvenile periodontitis (JP) formerly known as periodontosis and typically affecting teenagers and characterized by destruction of alveolar bone around only first permanent molar teeth and incisor teeth; and, (3) prepubertal periodontitis (PP) affecting the deciduous teeth in children around age 4.

**Etiology:** Bacterial plaque is thought to be responsible for periodontitis. In the rapidly progressive type, there is evidence for increased activity of *Bacteroides* and *Actinobacillus, Eikenella,* and *Capnocytophaga* organisms coupled with defects of leukocyte chemotaxis. In the juvenile (periodontosis) type, research has implicated a similar array of gram negative anaerobic rods as pathogens but there is no plausible explanation for the selective in-
volvement of first molar and incisor teeth other than the “first erupted-first involved” theory. In the prepubertal form of periodontitis, an abnormality of the immune system in the form of decreased chemotaxis of monocytes and neutrophils has been identified. From the above, it is obvious that alterations in plaque flora and reduced immunity are encountered in the subtypes of periodontitis but there is uncertainty about a cause and effect relationship.

**Treatment:** In conventional periodontitis, prevention is achieved through good dental hygiene. Unless treated, continued loss of alveolar bone eventually necessitates extraction of teeth. In those patients who still have adequate bone support, periodontal surgery to reduce the sulcus depth may be of benefit. Consult the reference list in the back of this book for information regarding treatment of the subtypes of periodontitis.

**Prognosis:** The prognosis depends on the stage of the disease at the time treatment is instituted. A thorough discussion is beyond the scope of this handbook.

**Differential diagnosis:** Most forms of periodontitis are easily recognized with periodontal probing and dental radiographs. Histiocytosis X and Burkitt’s lymphoma should be ruled out in prepubertal periodontitis. Children with prepubertal periodontitis combined with hyperkeratosis of the palms and soles are said to have the Papillon-Lefèvre syndrome. Prepubertal periodontitis has also been described in children with Ehlers-Danlos syndrome.

**Periapical cyst (radicular cyst)**

**Description:** This is a cyst at the apex of a non-vital tooth and is a sequela of pulpitis. Periapical cysts bear a great resemblance to dental granulomas. About the only substantive difference is the presence of an epithelium lined central cavity in the cyst. The associated tooth is usually asymptomatic. Acute infectious episodes may cause pain. The lesion appears as a sharply circumscribed radiolucent lesion around the apex of the associated tooth. It is often stated to have a thin sclerotic rim at the border but this feature is absent as often as present.

**Etiology:** This cyst is a direct sequela of inflammation of the pulp which has extended into the adjacent periapical tissues.

**Treatment:** Treatment consists of endodontic therapy or extraction of the associated tooth with curettage of the cyst.

**Prognosis:** Good

**Differential diagnosis:** Dental granuloma.

**Comment:** The periapical abscess has not appeared as a special entity in this handbook. A periapical infection with suppuration is correctly called a periapical abscess. An abscess may arise de novo or in a preexisting granuloma or cyst. Drainage of pus provides considerable relief of pain and hastens healing.

**Periapical dental granuloma**
Description: The dental granuloma is a focus of chronic inflammation around the apex of a tooth and is a sequela of pulpitis. Radiographically it appears as a periapical radiolucency. The border is usually distinct but this cannot be relied upon. The overlying tooth is ordinarily insensitive to electric pulp testing. While sensitivity to percussion may be present, many patients are totally asymptomatic. An acute infectious episode will result in pain, and often results in a formation of a draining sinus tract and/or parulis formation. Osteomyelitis and cellulitis are an ever present danger.

Etiology: Like the periapical cyst, the dental granuloma is a direct sequela of inflammation of a pulp which has extended into the surrounding periapical tissue. The pulpitis is in itself usually caused by infection secondary to caries but may be caused by trauma.

Treatment: Treatment consists of endodontic therapy or extraction.

Prognosis: Good

Differential diagnosis: Differential diagnosis includes radicular cyst and periapical abscess.

Condensing osteitis

Description: Condensing osteitis is a reaction to infection. It differs from other periapical inflammatory diseases in that there is a bone production rather than bone destruction. The result is a radiopaque lesion. This sclerotic reaction is apparently brought about by good patient resistance coupled with a low degree of virulence of the offending bacteria. It is more commonly seen in the young and seems to show special predilection for the periapical region of lower molars. The associated tooth is carious or contains a large restoration. We are reluctant to state the reaction of the tooth to electric pulp testing because of lack of sufficient personal experience and paucity of published information. Theoretically, the results should be abnormal. Whether or not the pulp is irreversibly diseased is not known. Current level of knowledge suggests that the pulp is irreversibly inflamed. Uncommonly, condensing osteitis occurs as a reaction to periodontal infection rather than dental infection.

Etiology: Infection of periapical tissues by organisms of low virulence.

Treatment: It has been our policy to treat only those cases which are symptomatic. This is done by endodontic therapy or extraction. In those cases which are asymptomatic in which there is no obvious caries in the associated tooth, we follow them with periodic x-ray examination.

Prognosis: In those cases in which the offending tooth is extracted, the area of condensing osteitis may remain in the jaws indefinitely.

Differential diagnosis: Idiopathic osteosclerosis and cementoblastoma. An abnormal result with electric pulp testing strongly suggests condensing osteitis and tends to rule out osteosclerosis and cementoblastoma.

Pericoronitis

Fig. 51

Description: The term pericoronitis refers to inflammation in the gingival tissue around the crown of a partially erupted tooth. The lower molars are the usual site. Gingiva around the crown is red, swollen, and painful. It is seen almost exclusively in those teeth which are partially erupted.

Etiology: Pericoronitis is caused by bacte-
rials infection of the gingiva. The anatomical circumstances that sets the stage for this disease develops when the crown of an erupting tooth has partially erupted through gingiva. The pericoronal dental follicle becomes a cul-de-sac extension of the mouth. This is an excellent place for bacterial growth, nurtured by stagnant fluid movement and accumulation of debris.

**Treatment:** Treatment consists of flushing out the space with sterile water or saline. Systemic antibiotics are indicated in severe infections. If the associated tooth will not erupt completely, it should be extracted.

**Prognosis:** Good

**Differential diagnosis:** The disease is so characteristic that differential diagnosis is no problem.

**Amalgam tattoo**

![Image of amalgam tattoo](image1)

**Description:** An amalgam tattoo is a localized area of blue-gray pigmentation. The amalgam is relatively inert and usually causes no tissue damage. The discoloration is permanent.

**Etiology:** The accidental and usually unavoidable implantation of dental amalgam in oral soft tissues produces this lesion.

**Treatment:** None required.

**Prognosis:** Good

**Differential diagnosis:** None

**Nasopalatine duct cyst**

![Image of nasopalatine duct cyst](image2)

**Description:** This is an ignoble name for a lesion. The dorsal surface of the tongue appears hairy and is discolored. The hairy texture is imparted by excessive keratinization of the filiform papillae. The keratin may take on the color of extrinsic stains and display a variety of colors. To the artistic minded, the more glorious cases rival the rainbow.

**Etiology:** The exact etiology is unknown, however, it appears to occur more frequently among heavy smokers. Another suggested cause is fungal overgrowth precipitated by antibiotics. In some patients, such as the case illustrated, there is no apparent cause.

**Treatment:** Treatment consists of brushing the tongue with a soft bristle toothbrush. If there is an obvious cause, naturally, it should be eliminated.

**Prognosis:** Good

**Differential diagnosis:** None

**Hairy tongue**

**Description:** This is an ignoble name for a lesion. The dorsal surface of the tongue appears hairy and is discolored. The hairy...
Description: This developmental cyst forms in the nasopalatine duct, and appears on x-ray above the apices of the maxillary incisors. The cyst may overlap the roots of the teeth. It is usually asymptomatic and discovered on routine dental films where it appears as an oval or heart-shaped radiolucent lesion. Rarely this cyst will expand overlying mucosa. It does not interfere with tooth vitality and seldom causes root resorption. It is frequently found in edentulous patients.

Etiology: This is a developmental cyst presumably arising from epithelial remnants in the nasopalatine duct.

Treatment: Surgical enucleation.

Prognosis: Good

Differential diagnosis: Radicular cyst, residual cyst, keratocyst, and central bone tumors.

Dentigerous cyst

![Image](image.png)

Description: This is a cyst that forms around the crown of an unerupted tooth and therefore, appears on x-ray as a pericoronal radiolucency. The size is extremely variable. Small ones are only slightly greater in size than a normal follicle whereas large ones are of sufficient size to virtually hollow out the jaw.

Most patients with this cyst are young or middle age adults. The teeth most commonly affected are third molars and maxillary cusps. No tooth is immune, but deciduous teeth are scarcely ever affected.

Small cysts are without symptoms but large ones expand the affected jaw and may cause pain and less often paresthesia.

Etiology: The accumulation of fluid between the unerupted tooth and the surrounding dental follicle is the accepted etiology. What precipitates this fluid accumulation is unknown.

Treatment: Surgical enucleation. All dentigerous cysts should be submitted for histopathologic examination.

Prognosis: Good

Differential diagnosis: Cystic ameloblastoma, odontogenic keratocyst, odontogenic adenomatoid tumor and calcifying epithelial odontogenic tumor.

Leukoplakia

Description: We use the term leukoplakia to mean a white lesion of mucous membrane. It carries no histologic connotation. Using this definition, biopsy is required for accurate diagnosis.

Biopsy of most oral leukoplakia will show hyperkeratosis, a purely reactive and harmless lesion. Some, however, will show dysplasia, a premalignant lesion. A small number will show carcinoma in-situ or invasive carcinoma.

![Image](image.png)

The leukoplakia shown in figure 56 was dysplastic (premalignant).

Leukoplakia is more common in males and favors older age groups. It produces no symptoms.

Etiology: The exact etiology is unknown although physical trauma, smoking, excessive alcohol intake and vitamin A deficiency are suspected.

Treatment: The treatment depends on the histologic findings and the extent of the lesion. For simple hyperkeratosis, removal of any apparent cause is indicated. Those showing dysplasia, or carcinoma, should be treated with the usual methods used in treatment of cancers.
Prognosis: As stated above, the prognosis depends to a great extent on the precise histologic findings. One study indicates approximately 20% of oral leukoplakia lesions will be dysplastic or malignant on the day of biopsy.

Differential diagnosis: Hyperkeratosis, dysplasia, squamous carcinoma, lichen planus, candidosis, and lupus erythematosus.

Erythroplasia (erythroplakia)

Description: A red but not ulcerated area on mucous membrane is called erythroplasia. The texture may be normal or roughened. Size is variable, some being so small as to virtually escape detection whereas large areas are conspicuous to casual inspection. There are usually no symptoms. Being neither elevated nor depressed, they present as quiet, unpretentious lesions. The border may be sharp or blend imperceptibly into surrounding normal mucosa. It must constantly be kept in mind that early carcinoma frequently appears as an area of erythroplasia.

Fig. 57

Etiology: Because there are several different diseases which appear as erythroplasia, there naturally are multiple etiologic agents. Local irritants and infection are responsible for some lesions. For those which prove to be dysplastic or neoplastic, the etiology is unknown. Tobacco and alcohol are chief suspects.

Treatment: Treatment depends on the histologic findings. In those with an obvious cause, the cause should be eliminated. If the biopsy shows dysplasia, total excision is indicated. Those that show in-situ carcinoma or infiltrating carcinoma are treated appropriately.

Prognosis: This depends on the histologic diagnosis and extent of the lesion. In one study, more than 90% of oral erythroplakias were dysplastic (premalignant) or malignant on the day of the biopsy.

Differential diagnosis: Lesions of physical trauma, chemical burns, infections and non-infectious mucositis, dysplasia, in-situ carcinoma and squamous cell carcinoma.

Comment: As mentioned in the section on carcinoma, there are certain areas of the oral mucosa which seem more prone to develop malignancy. Additionally, oral cancer is more often seen in those over age 40. Because of this, an area of erythroplasia in a cancer prone area in a patient past 40 is highly suspicious for malignancy and should be biopsied on the day it is seen. This is especially true for those lesions whose duration exceeds 2 weeks.

Squamous cell carcinoma (epidermoid carcinoma)

Fig. 58

Fig. 59

Description: In excess of 90% of all oral cancers are of the squamous cell type. As stated in other sections in this monograph, early carcinoma may clinically appear as leukoplakia or erythroplasia. It may also appear as a mixture of erythroplasia and leukoplakia as is illustrated in fig. 58.
Another common clinical appearance is an area of chronic ulceration as shown in fig. 59.

Squamous carcinoma is about four times as common in men as in women. Risk of acquiring the disease increases with each passing decade but is seldom seen in those under forty. According to the American Cancer Society there are about 17,000 new cases in the United States each year. Although no area of oral mucosa is immune, certain areas are more vulnerable. Soft palate, lateral and ventral tongue mucosa, and floor of the mouth are especially prone to develop squamous carcinoma.

**Etiology:** The etiology is unknown. To restate here the numerous suspected etiologic agents would serve no useful purpose. Naturally, suspicion has fallen on smoking and alcohol.

**Treatment:** Usual treatment consists of surgical excision, irradiation, and chemotherapy or combinations of these.

**Prognosis:** The five year survival rate is about 50%. Early diagnosis increases the chance of survival.

**Differential Diagnosis:** Hyperkeratosis, erythroplasia, lichen planus, and traumatic ulceration. A definitive diagnosis can be established only by biopsy.

**Snuff lesion**
*(smokeless tobacco lesion)*

![Fig. 60](image)

**Description:** The lesion develops on the mucosa adjacent to where smokeless tobacco is held. The usual appearance is a white, wrinkled or corrugated thickened mucosa with intervening areas of erythema. The clinical picture may change and is related to the duration of exposure.

Periodontal degeneration or gingival recession is also a common manifestation with cervical erosion of teeth a less frequent finding. Symptoms are uncommon.

**Etiology:** Prolonged use of smokeless tobacco products such as chewing tobacco or snuff.

**Treatment:** Biopsy should be done to rule out dysplasia, otherwise no treatment is necessary.

**Prognosis:** Verrucous and squamous carcinomas arise in smokeless tobacco lesions more than chance alone can explain. Both the irritation as well as organic carcinogens in the tobacco have been implicated. One article noted almost a 50-fold increased risk of cancers of the gingiva and buccal mucosa in females who were chronic users. The duration necessary to induce dysplastic or malignant change is unknown but appears to be at least 20 years. This potential malignant change is of great concern with the marked increased use by our teenage population. Persistent lesions should be biopsied to rule out dysplastic or carcinomatous change.

**Differential Diagnosis:** The clinical appearance of the lesion plus a history of using smokeless tobacco establishes the diagnosis.

**Cementoma**

**Description:** Cementoma occurs as a self-limiting lesion around the apices of vital teeth, chiefly in women in the middle years. The condition is more common in Blacks, and lower anterior teeth are the principal site. The lesion may be solitary or occur in multiples. The initial lesion is a periapical proliferation of benign fibrous connective tissue in the periodontal ligament. There are no symptoms and x-ray shows a periapical radiolucency ordinarily not exceeding a centimeter. Cementum is slowly formed in the central area and the entire lesion becomes converted to a mineralized mass which appears radiopaque on x-ray. Often a thin radiolucent halo persists around the circumference of the opaque lesion.

**Etiology:** Unknown
Treatment: There is no treatment required. 
Prognosis: Good, the lesion is self-limiting. 
Differential diagnosis: Differential diagnosis during the lytic stage includes periapical cysts, abscesses, and granulomas. The opaque stage must be distinguished from condensing osteitis and osteosclerosis. Pulp vitality tests of associated teeth are an absolute necessity to arrive at accurate diagnosis.

Acquired immune deficiency syndrome (AIDS) 
Description: Acquired immune deficiency syndrome is characterized by relentless destruction of key cells of the immune system. The eventual collapse of both the cellular and humoral arms of immunity leaves the host vulnerable to a wide variety of pathogenic organisms including bacteria, viruses, fungi and protozoa.

It is important for health care workers to recognize that it is difficult to transmit the AIDS virus in the health care setting, from patient to worker or the reverse. However, the opportunistic infectious diseases AIDS patients are apt to have including tuberculosis, herpesvirus infections and hepatitis B are readily transmissible.

Etiology: The causes of AIDS is an RNA retrovirus of the lentivirus group. It is designated the human immunodeficiency virus (HIV) and there are two variants: HIV-1, the cause of most cases of AIDS, and HIV-2. The virus attaches to the surface of cells that bear the CD4 receptor including helper T lymphocytes, B lymphocytes and macrophages. Although they lack a CD4 receptor, astrocytes, skin fibroblasts, and bowel epithelium become infected. The virus destroys the infected cells. With gradual depletion of the key cells of immunity, especially T-helper lymphocytes and macrophages, the host becomes increasingly vulnerable to pathogenic organisms.

Oral manifestations: Candidiasis—Colonization and infection of the oral mucosa by Candida albicans is among the earliest and most common findings in HIV-infected patients. In one study, 88% had oral candidiasis. Lesions range from white to red or red/white combinations. Figure 63 illustrates the typical appearance of candidiasis on the lateral tongue; note the resemblance to hairy leukoplakia. The lesions may be asymptomatic or there may be mild discomfort. The treatment of candidiasis is discussed on page 17 of this text. For stubborn infection in AIDS patients, fluconazole is recommended.

Kaposi's sarcoma—AIDS patients are vulnerable to a variety of oral malignancies including Kaposi's sarcoma, malignant lymphoma and squamous carcinoma. Kaposi's sarcoma is the most common. In one study, 20% of AIDS patients had Kaposi's sarcoma and of these, the tumor was in the oral cavity in 1 of every 5 patients; the palate is the most common site. In the early stage, the tumor appears as a red to purple bruise (fig. 64). The tumor grows and eventually appears as a hemorrhagic mass (fig. 65). The cell of origin is endothelium; thus Kaposi's sarcoma is a variety of angiosarcoma. They are locally invasive, cause pain and bleeding and interfere with normal
function. Radiation is the preferred treatment but laser resection and intralesional vinblastine provide palliation.

Hairy leukoplakia—This variety of leukoplakia was first recognized in HIV-infected patients but has been encountered in other immune deficiency states such as organ transplant patients who are intentionally immune suppressed. The lateral tongue is the most common location (fig. 66). Lesions are of rough texture, adherent and asymptomatic. The diagnosis of hairy leukoplakia can be suspected on routine biopsy specimens, but confirmation requires demonstration of the presence of the causative virus, the Epstein-Barr herpesvirus. This is ordinarily achieved by DNA in situ hybridization. A word of caution: hairy leukoplakia may be confused with candidiasis. In one study, 52% of cases clinically diagnosed as hairy leukoplakia proved to be candidiasis. A patient who presents with a white lesion should be treated with antifungal therapy first. If it fails to heal, it most likely is hairy leukoplakia.

Gingival and periodontal lesions—HIV-infected patients are vulnerable to gingivitis, periodontitis, (fig. 67) and necrotizing ulcerative gingivitis (ANUG-like). The organisms recovered from these lesions are the same as those in non-HIV-positive patients, but they are present in greater numbers. Lesions are treated by dental prophylaxis, debridement, and metronidazole. Good oral hygiene and daily rinses with chlorhexidine are beneficial.

Others—HIV patients also develop major aphthous-like lesions that respond to topical tetracycline and topical steroid therapy. Other patients have painful palatal and gingival ulcers that have been found to harbor cytomegalovirus. The human papillomavirus has also been found in mucosal papules. Herpesvirus may cause painful and protracted oral ulcers that are responsive to treatment with acyclovir. Lastly, xerostomia secondary to a Sjögren's syndrome-like illness has been reported.
Additional references

Foliate papillae

Varix

Tori

Osteosclerosis

Bone marrow defect

Aphthous stomatitis


Herpesvirus infection


Papillary hyperplasia (PH)


Epulis fissuratum
Irritation fibroma

Mucocoele

Papilloma


Peripheral fibroma

Pyogenic granuloma

Peripheral giant cell granuloma

Traumatic ulcer

Stomatitis nicotina


Dilantin hyperplasia


Geographic tongue

Lichen planus


Candidosis
Pulpitis

Dental caries

Marginal gingivitis

Vincent's infection

Periodontitis

Periapical cyst

Dental granuloma

Condensing osteitis

Pericoronitis

Amalgam tattoo

Black hairy tongue

Nasopalatine duct cyst

Dentigerous cyst

Leukoplaikia

Erythroplasia

Squamous cell carcinoma

Snuff lesion

Cementoma
Acquired immune deficiency syndrome

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Dr. Benjamin Warner: fig. 47
Dr. Richard Pascoe: fig. 45
Dr. Glenn Hemberger: fig. 26
C. V. Mosby Co.: fig. 9
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- Gum irritations
- Denture and Mouth Sores
- Orthodontic irritations
- Mouth Burns and Cheek Bites

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- Maximum Strength Pain Relief
- Relieves Pain of Cold Sores, Fever Blisters and Canker Sores
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