Oral Pathology

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Chapter 1

Mouth Disease

Mouth disease	
ICD-10	K00.-K14.
ICD-9	520-529
MeSH	D009057

Stomatognathic disease or **mouth disease** refers to the diseases of the mouth ("stoma") and jaw ("gnath"). The etymology is similar to that of the term Gnathostomata. It is the term used by MeSH (along with the synonym **dental diseases**), but other organizations use different terms.

The mouth is an important organ with many different functions. It is also prone to a variety of medical and dental disorders.

The American Dental Association uses the term "oral and maxillofacial pathology", and describes it as "the specialty of dentistry and pathology which deals with the nature, identification, and management of diseases affecting the oral and maxillofacial regions. It is a science that investigates the causes, processes and effects of these diseases."

The World Health Organization uses the term "Diseases of oral cavity, salivary glands and jaws."

Salivary glands

There are both major-major and minor-minor salivary gland in the mouth which secrete saliva and a variety of enzymes to help process foods and make swallowing easy. These salivary glands can get infected or inflamed and can also be very painful; sometimes the salivary glands also develop benign and malignant cancers. However, the most common problem with salivary gland is formation of stones in the small ducts which prevent free

flowing of saliva. The gland swells as they cannot empty and often get infected. While most stones in the duct may resolve, sometimes surgery and antibiotics are required.

Mumps

Mumps of the salivary glands is a viral infection of the parotid glands. This results in painful swelling at the sides of the mouth in both adults and children. The infection is quite contagious. Today mumps is prevented by getting vaccinated in infancy. There is no specific treatment for mumps except for hydration and Tylenol. Sometimes mumps can cause inflammation of the brain, testicular swelling or hearing loss.

Bad breath

Bad breath (halitosis) has many causes including smoking, alcohol, poor care of dentures, gum disease, chronic lung disease, breathing through the mouth, sinusitis, liver disease, diabetes, pregnancy, not brushing or flossing on a regular basis. Medications that cause dryness in the mouth can also cause bad breath. These include antidepressants, anti histamines and antipsychotics. The best way to prevent bad breath is to brush teeth frequently, clean the tongue, keep the nose and sinus clean and drink adequate water.

Canker sores

Canker sores are small ulcers that appear on the inside of the mouth, lips and on tongue. Most small canker sores disappear within 10-14 days. Canker sores are most common in young and middle aged individuals. Sometimes individuals with allergies are more prone to these sores. Besides an awkward sensation, these sores can also cause tingling or a burning sensation. Unlike herpes sores, canker sores are always found inside the mouth and are usually less painful. Good oral hygiene does help but sometime one may have to use a topical corticosteroid.

Fungus infections

Candida is a very common infection of the mouth in immunocompromised individuals. Individuals who have undergone a transplant, HIV, cancer or use corticosteroids commonly develop candida of the mouth and oral cavity. The typical signs are a white patch that may be associated with burning, soreness, irritation or a white cheesy like appearance. Once the diagnosis is made, candida can be treated with a variety of anti fungal drugs.

Herpes

Another very common disorder of the oral cavity is herpes simplex infection (HSV). This virus causes blisters and sores around the mouth and lips. HSV infections are not only annoying but also painful and may keep on recurring. Although many people get infected with the virus, only 10% actually develop the sores. The sores may last anywhere from 3-

10 days and are very infectious. Some people have recurrences either in the same location or at a nearby site. Recurrent infections tend to be mild in nature and may be brought on by stress, sun, menstrual periods, trauma or physical stress.

Burning mouth

Burning mouth syndrome (BMS) is a very painful annoying disorder that causes a sensation of burning on the lips, tongue, mouth and gums. The disorder can affect anyone but tends to occur most often in middle aged women. BMS has been linked to a variety of dental and medical disorders like menopause, dry mouth and allergies. Some individual develop one episode of BMS and others develop recurrent episodes which last months or years. Other features of this distressing disorder include anxiety, depression and social isolation. There is no cure for this disorder and treatment includes use of hydrating agents, pain medications, vitamin supplements or the usage of antidepressants.

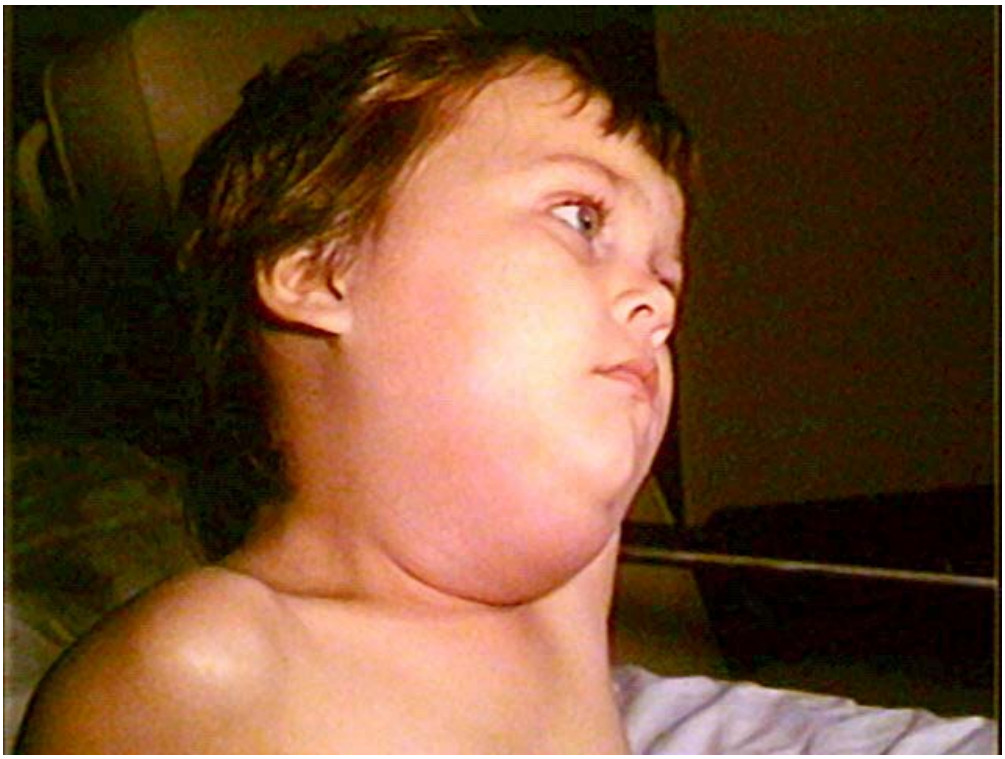
Cancers

Oral cancer may occur on the lips, tongue, gums, floor of the mouth or inside the cheeks. The majority of cancers of the mouth are known as squamous cell cancers. Oral cancers are usually painless in the initial stages or may appear like an ulcer. Causes of oral cancer include smoking, excessive alcohol consumption, exposure to sunlight (lip cancer), chewing tobacco, and infection with the human Human papillomavirus. The earlier the oral cancer is diagnosed, the better the chances for full recovery. If you have a suspicious mass or ulcer on the mouth which has been persistent, then you should always get a dentist to look at it. Diagnosis is usually made with a biopsy and the treatment depends on the exact type of cancer, where it is situated, and extent of spreading.

Chapter 2

Mumps

Mumps



Child with mumps.

ICD-10	B26.
ICD-9	072
DiseasesDB	8449

MedlinePlus	001557
eMedicine	emerg/324 emerg/391 ped/1503
MeSH	D009107

Mumps and **epidemic parotitis** is a viral disease of the human species, caused by the mumps virus. Before the development of vaccination and the introduction of a vaccine, it was a common childhood disease worldwide. It is still a significant threat to health in the third world, and outbreaks still occur sporadically in developed countries.

Painful swelling of the salivary glands (classically the parotid gland) is the most typical presentation. Painful testicular swelling (orchitis) and rash may also occur. The symptoms are generally not severe in children. In teenage males and men, complications such as infertility or subfertility are more common, although still rare in absolute terms. The disease is generally self-limited, running its course before receding, with no specific treatment apart from controlling the symptoms with pain medication.

Signs and symptoms

The more common symptoms of mumps are:

- Parotid inflammation (or parotitis) in 60–70% of infections and 95% of patients with symptoms. Parotitis causes swelling and local pain, particularly when chewing. It can occur on one side (unilateral) but is more common on both sides (bilateral) in about 90% of cases.
- Fever
- Headache
- Orchitis, referring to painful inflammation of the testicle. Males past puberty who develop mumps have a 30 percent risk of orchitis.

Other symptoms of mumps can include dry mouth, sore face and/or ears and occasionally in more serious cases, loss of voice. In addition, up to 20% of persons infected with the mumps virus do not show symptoms, so it is possible to be infected and spread the virus without knowing it.

Prodrome

Fever and headache are prodromal symptoms of mumps, together with malaise and anorexia.

Cause

Mumps is a contagious disease that is spread from person to person through contact with respiratory secretions such as saliva from an infected person. When an infected person coughs or sneezes, the droplets aerosolize and can enter the eyes, nose, or mouth of another person. Mumps can also be spread by sharing food and drinks. The virus can also survive on surfaces and then be spread after contact in a similar manner. A person infected with mumps is contagious from approximately 6 days before the onset of symptoms until about 9 days after symptoms start. The incubation period (time until symptoms begin) can be from 14–25 days but is more typically 16–18 days.

Diagnosis

A physical examination confirms the presence of the swollen glands. Usually the disease is diagnosed on clinical grounds and no confirmatory laboratory testing is needed. If there is uncertainty about the diagnosis, a test of saliva, or blood may be carried out; a newer diagnostic confirmation, using real-time nested polymerase chain reaction (PCR) technology, has also been developed. An estimated 20%-30% of cases are asymptomatic. As with any inflammation of the salivary glands, serum amylase is often elevated.

Prevention

The most common preventative measure against mumps is immunization with a mumps vaccine, invented by Maurice Hilleman at Merck. The vaccine may be given separately or as part of the MMR immunization vaccine which also protects against measles and rubella. In the US, MMR is now being supplanted by MMRV, which adds protection against chickenpox. The WHO (World Health Organization) recommends the use of mumps vaccines in all countries with well-functioning childhood vaccination programmes. In the United Kingdom it is routinely given to children at age 15 months. The American Academy of Pediatrics recommends the routine administration of MMR vaccine at ages 12–15 months and at 4–6 years. In some locations, the vaccine is given again between 4 to 6 years of age, or between 11 and 12 years of age if not previously given. The efficacy of the vaccine depends on the strain of the vaccine, but is usually around 80%.[7] The Jeryl Lynn strain is most commonly used in developed countries but has been shown to have reduced efficacy in epidemic situations. The Leningrad-Zagreb strain commonly used in developing countries appears to have superior efficacy in epidemic situations.

Because of the outbreaks within college and university settings, many governments have established vaccination programs to prevent large-scale outbreaks. In Canada, provincial governments and the Public Health Agency of Canada have all participated in awareness campaigns to encourage students ranging from grade 1 to college and university to get vaccinated.

Some anti-vaccine activists protest against the administration of a vaccine against mumps, claiming that the attenuated vaccine strain is harmful, and/or that the wild

disease is beneficial. There is very little evidence to support the claim that the wild disease is beneficial, or that the MMR vaccine is harmful. Claims have been made that the MMR vaccine is linked to autism and inflammatory bowel disease, including one study by Andrew Wakefield (paper retracted in 2010) that indicated a link between gastrointestinal disease, autism, and the MMR vaccine. However, all further studies since that time have indicated no link between vaccination with the MMR and autism. Since the dangers of the disease are well known, while the dangers of the vaccine are quite minimal, most doctors recommend vaccination.

The WHO, the American Academy of Pediatrics, the Advisory Committee on Immunization Practices of the Centers for Disease Control and Prevention, the American Academy of Family Physicians, the British Medical Association and the Royal Pharmaceutical Society of Great Britain currently recommend routine vaccination of children against mumps. The British Medical Association and Royal Pharmaceutical Society of Great Britain had previously recommended against general mumps vaccination, changing that recommendation in 1987. In 1988 it became United Kingdom government policy to introduce mass child mumps vaccination programmes with the MMR vaccine, and MMR vaccine is now routinely administered in the UK.

Before the introduction of the mumps vaccine, the mumps virus was the leading cause of viral meningoencephalitis in the United States. However, encephalitis occurs rarely (less than 2 per 100,000). In one of the largest studies in the literature, the most common symptoms of mumps meningoencephalitis were found to be fever (97%), vomiting (94%) and headache (88.8%). The mumps vaccine was introduced into the United States in December 1967: since its introduction there has been a steady decrease in the incidence of mumps and mumps virus infection. There were 151,209 cases of mumps reported in 1968. Since 2001, the case average was only 265 per year, excluding an outbreak of >6000 cases in 2006 attributed largely to university contagion in young adults.

Treatment

There is no specific treatment for mumps. Symptoms may be relieved by the application of intermittent ice or heat to the affected neck/testicular area and by acetaminophen/paracetamol (Tylenol) for pain relief. Aspirin is not used due to a hypothetical link with Reye's syndrome. Warm salt water gargles, soft foods, and extra fluids may also help relieve symptoms. According to the Department of Health of Minnesota there is no effective post-exposure recommendation to prevent secondary transmission, as well as the post-exposure use of vaccine or Immune Globulin is not effective.

Patients are advised to avoid fruit juice or any acidic foods, since these stimulate the salivary glands, which can be painful.

Prognosis

Death is very unusual. The disease is self-limiting, and general outcome is good, even if other organs are involved.

Known complications of mumps include:

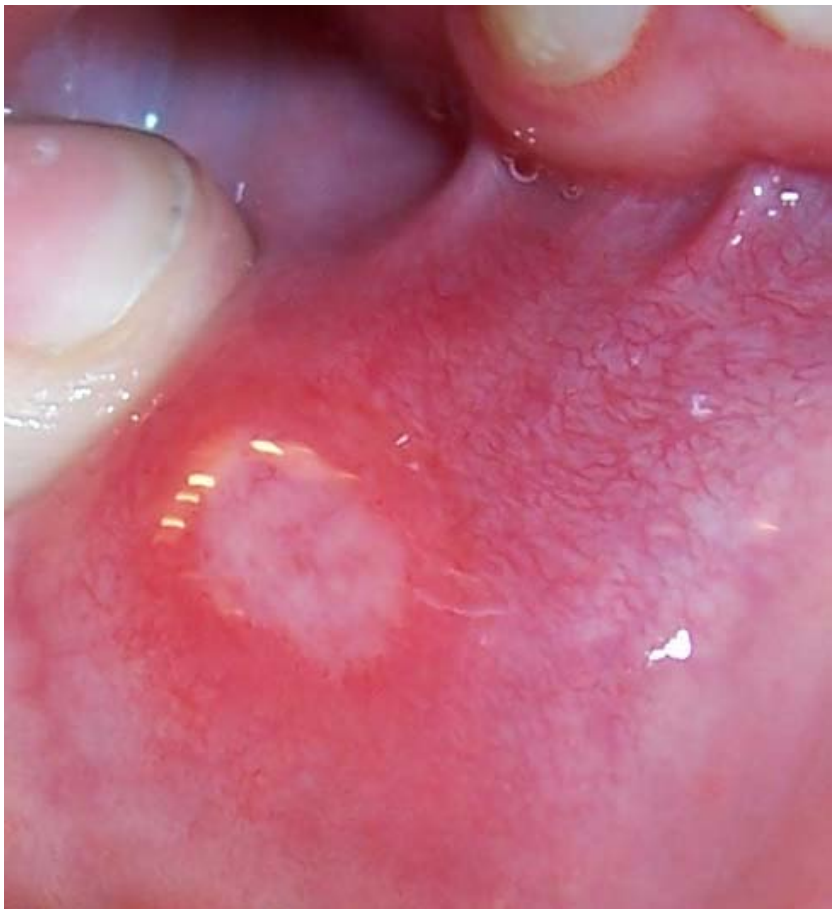
- Infection of other organ systems
- Mumps viral infections in adolescent and adult males carry an up to 30% risk that the testes may become infected (orchitis or epididymitis), which can be quite painful; about half of these infections result in testicular atrophy, and in rare cases sterility can follow.
- Spontaneous abortion in about 27% of cases during the first trimester of pregnancy.
- Mild forms of meningitis in up to 10% of cases (40% of cases occur without parotid swelling)
- Oophoritis (inflammation of ovaries) in about 5% of adolescent and adult females, but fertility is rarely affected.
- Pancreatitis in about 4% of cases, manifesting as abdominal pain and vomiting
- Encephalitis (very rare, and fatal in about 1% of the cases when it occurs)
- Profound (91 dB or more) but rare sensorineural hearing loss, uni- or bilateral. Acute unilateral deafness occurs in about 0.005% of cases.

After the illness, life-long immunity to mumps generally occurs; reinfection is possible but tends to be mild and atypical.

Chapter 3

Aphthous Ulcer

Aphthous ulcer



Mouth ulcer on the lower lip

ICD-10	K12.0
ICD-9	528.2

MedlinePlus	000998
eMedicine	ent/700 derm/486 ped/2672
MeSH	D013281

An **aphthous ulcer** also known as a **canker sore**, is a type of mouth ulcer, appears as a painful open sore inside the mouth or upper throat characterized by a break in the mucous membrane. Its cause is unknown, but they are not contagious. The condition is also known as **aphthous stomatitis,** and alternatively as **Sutton's Disease**, especially in the case of major, multiple, or recurring ulcers.

The term ***aphtha*** means **ulcer;** it has been used for many years to describe areas of ulceration on mucous membranes. Aphthous stomatitis is a condition characterized by recurrent discrete areas of ulceration that are almost always painful. Recurrent aphthous stomatitis (RAS) can be distinguished from other diseases with similar-appearing oral lesions, such as certain oral bacteria or herpes simplex, by their tendency to recur, and their multiplicity and chronicity. Recurrent aphthous stomatitis is one of the most common oral conditions. At least 10% of the population has it, and women are more often affected than men. About 30–40% of patients with recurrent aphthae report a family history.

Classification

Aphthous ulcers are classified according to the diameter of the lesion.

Minor ulceration

"Minor aphthous ulcers" indicate that the lesion size is between 3 mm (0.1 in)-10 mm (0.4 in). The appearance of the lesion is that of an erythematous halo with yellowish or grayish color. Pain that affects quality of life is the obvious characteristic of the lesion. When the ulcer is white or grayish, the ulcer will be extremely painful and the affected lip may swell. They may last about 2 weeks.

Major ulcerations

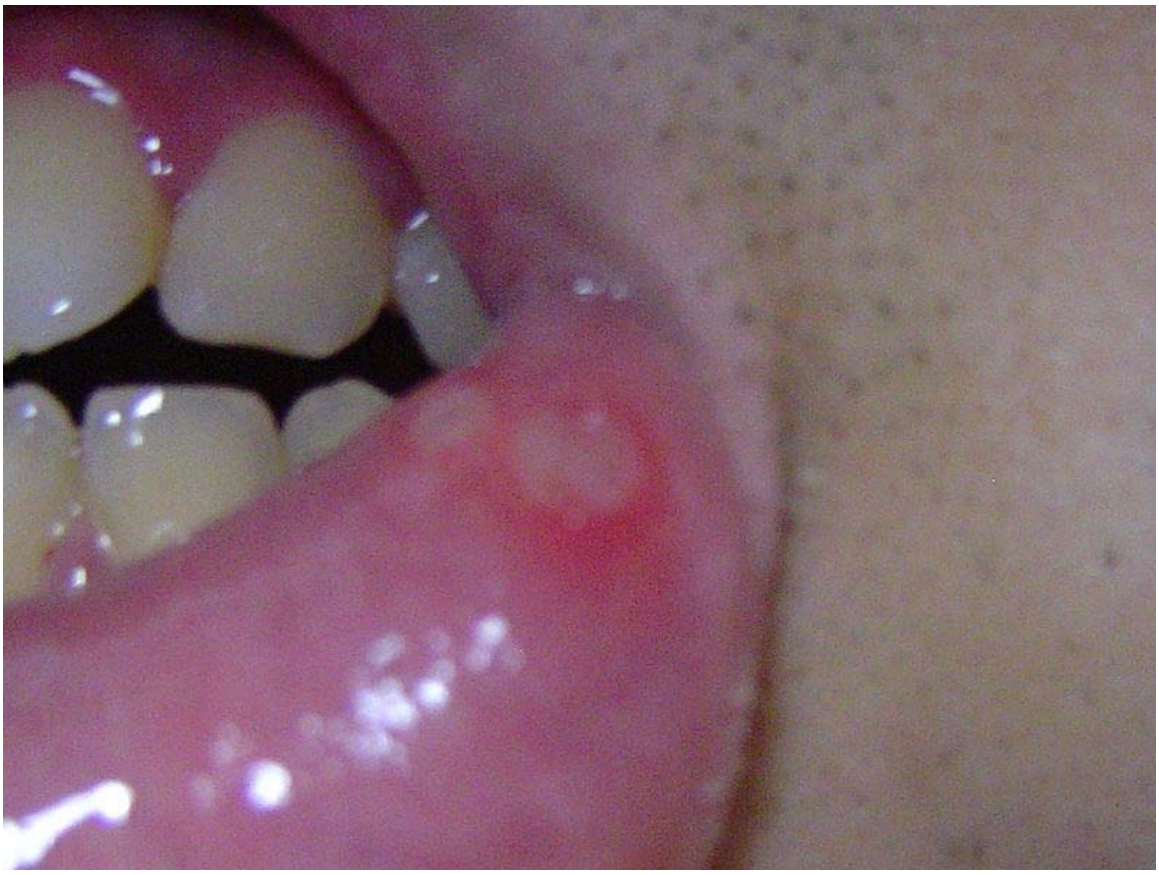
Major aphthous ulcers have the same appearance as minor ulcerations, but are greater than 10 mm in diameter and are extremely painful. They usually take more than a month to heal, and frequently leave a scar. These typically develop after puberty with frequent recurrences. They occur on movable non-keratinizing oral surfaces, but the ulcer borders may extend onto keratinized surfaces.

Herpetiform ulcerations

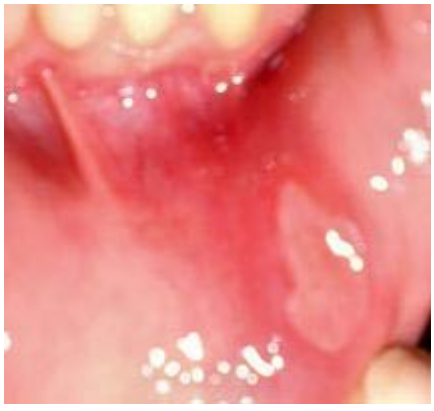
This is the most severe form. It occurs more frequently in females, and onset is often in adulthood. It is characterized by small, numerous, 1–3 mm lesions that form clusters.

They typically heal in less than a month without scarring. Supportive treatment is almost always necessary.

Signs and symptoms



Apthous ulcer



Large aphthous ulcer on the lower lip

Aphthous ulcers usually begin with a tingling or burning sensation at the site of the future aphthous ulcer. In a few days, they often progress to form a red spot or bump, followed by an open ulcer.

The aphthous ulcer appears as a white or yellow oval with an inflamed red border. Sometimes a white circle or halo around the lesion can be observed. The gray-, white-, or yellow-colored area within the red boundary is due to the formation of layers of fibrin, a protein involved in the clotting of blood. The ulcer, which itself is often extremely painful, especially when agitated, may be accompanied by a painful swelling of the lymph nodes below the jaw, which can be mistaken for toothache; another symptom is fever. A sore on the gums may be accompanied by discomfort or pain in the teeth.

Causes

The exact cause of many aphthous ulcers is unknown but citrus fruits (e.g., oranges and lemons), physical trauma, stress, lack of sleep, sudden weight loss, food allergies, immune system reactions, and deficiencies in vitamin B_{12}, iron, and folic acid may contribute to their development. Nicorandil and certain types of chemotherapy are also linked to aphthous ulcers. One recent study showed a strong correlation with allergies to cow's milk. Aphthous ulcers are a major manifestation of Behçet disease, and are also common in people with Crohn's disease.

Trauma to the mouth is the most common trigger. Physical trauma, such as that caused by toothbrush abrasions, laceration with sharp or abrasive foods (such as toast, potato chips or other objects, like toothbrushes), accidental biting (particularly common with sharp canine teeth), after losing teeth, or dental braces can cause aphthous ulcers by breaking the mucous membrane. Other factors, such as chemical irritants or thermal injury, may also lead to the development of ulcers. Using a toothpaste without sodium lauryl sulfate (SLS) may reduce the frequency of aphthous ulcers One smaller study found no connection between SLS in toothpaste and aphthous ulcers. Celiac disease has been suggested as a cause of aphthous ulcers; small studies of patients (33% or 1 out of 3) with Celiac disease did demonstrate a conclusive link between the disease and aphthous ulcers vs control group (23%) but some patients benefited from eliminating gluten from their diets.

There is no indication that aphthous ulcers are related to menstruation, pregnancy, and menopause. Smokers appear to be affected less often.

Prevention

Oral and dental measures

- Regular use of non-alcoholic mouthwash may help prevent or reduce the frequency of sores. In fact, informal studies suggest that mouthwash may help to temporarily relieve pain.

- In some cases, switching toothpastes can prevent aphthous ulcers from occurring, with research looking at the role of sodium dodecyl sulfate (sometimes called sodium lauryl sulfate, or with the acronymes SDS or SLS), a detergent found in most toothpastes. Using toothpaste free of this compound has been found in several studies to help reduce the amount, size, and recurrence of ulcers.
- Dental braces are a common physical trauma that can lead to aphthous ulcers and the dental bracket can be covered with wax to reduce abrasion of the mucosa. Avoidance of other types of physical and chemical trauma will prevent some ulcers, but, since such trauma is usually accidental, this type of prevention is not usually practical.

Nutritional therapy

- Zinc deficiency has been reported in people with recurrent aphthous ulcers. The few small studies looking into the role of zinc supplementation have mostly reported positive results particularly for those people with deficiency, although some research has found no therapeutic effect.

Treatment

A number of different treatments exist for apthous ulcers including: analgesics, anesthetics agents, antiseptics, anti-inflammatory agents, steroids, sucralfate, tetracycline suspension, and silver nitrate.

Amlexanox paste has been found to speed healing and alleviate pain.

Vitamin B_{12} has been found to be effective in treating recurrent aphthous ulcers, regardless of whether there is a vitamin deficiency present.

While dietary supplements of L-lysine can be effective in treating cold sores/herpetic lesions, there is no evidence of an impact on canker sores.

Suggestions to reduce the pain caused by an ulcer include: avoiding spicy food, rinsing with salt water or over-the-counter mouthwashes, proper oral hygiene and non-prescription local anesthetics. Active ingredients in the latter generally include benzocaine, benzydamine or choline salicylate.

Anesthetic mouthwashes containing benzydamine hydrochloride have not been shown to reduce the number of new ulcers or significantly reduce pain, and evidence supporting the use of other topical anesthetics is very limited, though some individuals may find them effective. In general, their role is limited; their duration of effectiveness is, in general, short and does not provide pain control throughout the day. Such medications may also cause complications in children.

Evidence is limited for the use of antimicrobial mouthwashes but suggests that they may reduce the painfulness and duration of ulcers and increase the number of days between ulcerations, without reducing the number of new ulcers.

Milk of magnesia is useful against aphthous ulcers when used topically.

Corticosteroid preparations containing hydrocortisone hemisuccinate or triamcinolone acetonide to control symptoms are effective in treating aphthous ulcers.

The application of silver nitrate will cauterize the sore; a single treatment decreases pain but does not affect healing time though in children it can cause tooth discoloration if the teeth are still developing. The use of tetracycline is controversial, as is treatment with levamisole, colchicine, gamma-globulin, dapsone, estrogen replacement and monoamine oxidase inhibitors.

While commonly used, Magic mouthwash, a combination of a number of ingredients including viscous lidocaine, benzocaine, milk of magnesia, kaolin-pectate, chlorhexidine, or diphenhydramine, has little evidence to support its use in the treatment of aphthous ulcers.

There is the hypothesis that pasteurized goat milk can help with disease symptoms. At the current time, a clinical trial is conducted to check these claims.

Epidemiology

Canker sores are a very common oral lesion. Epidemiological studies show an average prevalence between 15% and 30%. Canker sores tend to afflict women more than men and people less than 45 years old. Canker sores occur most frequently among 16- to 25-year-olds, and they rarely occur in anyone over 55. The frequency of canker sores varies from less than 4 episodes per year (85% of all cases) to more than one episode per month (10% of all cases) including people suffering from continuous RAS.

Chapter 4

Oral Cancer

Oral cancer	
ICD-10	C00.-C08.
ICD-9	140-146
DiseasesDB	9288

Oral cancer is a subtype of head and neck cancer, is any cancerous tissue growth located in the oral cavity. It may arise as a primary lesion originating in any of the oral tissues, by metastasis from a distant site of origin, or by extension from a neighboring anatomic structure, such as the nasal cavity or the maxillary sinus. Oral cancers may originate in any of the tissues of the mouth, and may be of varied histologic types: teratoma, adenocarcinoma derived from a major or minor salivary gland, lymphoma from tonsillar or other lymphoid tissue, or melanoma from the pigment producing cells of the oral mucosa. There are several types of oral cancers, but around 90% are squamous cell carcinomas, originating in the tissues that line the mouth and lips. Oral or mouth cancer most commonly involves the tongue. It may also occur on the floor of the mouth, cheek lining, gingiva (gums), lips, or palate (roof of the mouth). Most oral cancers look very similar under the microscope and are called squamous cell carcinoma. These are malignant and tend to spread rapidly.

Signs and symptoms

Skin lesion, lump, or ulcer:

- On the tongue, lip, or other mouth areas
- Usually small
- Most often pale colored, may be dark or discolored
- Early sign may be a white patch (leukoplakia) or a red patch (erythroplakia) on the soft tissues of the mouth

- Usually painless initially
- May develop a burning sensation or pain when the tumor is advanced

Additional symptoms that may be associated with this disease:

- Tongue problems
- Swallowing difficulty
- Mouth sores that do not resolve in 14 days
- Pain and paraesthesia are late symptoms.

Causes

Oncogenes are activated as a result of mutation of the DNA. The exact cause is often unknown. Regardless of the cause, treatment is the same: surgery, radiation with or without chemotherapy. Risk factors that predispose a person to oral cancer have been identified in epidemiological studies. India being member of International Cancer Genome Consortium is leading efforts to map oral cancer's complete genome.

In many Asian cultures chewing betel, paan and Areca is known to be a strong risk factor for developing oral cancer. In India where such practices are common, oral cancer represents up to 40% of all cancers, compared to just 4% in the UK.

Some oral cancers begin as leukoplakia a white patch (lesion), red patches, (erythroplakia) or non healing sores that have existed for more than 14 days. In the US oral cancer accounts for about 8 percent of all malignant growths. Men are affected twice as often as women, particularly men older than 40/60. In Indian subcontinent Oral submucous fibrosis is very common. This condition is characterized by limited opening of mouth and burning sensation on eating of spicy food. This is a progressive lesion in which the opening of the mouth becomes progressively limited, and later on even normal eating becomes difficult. It occurs almost exclusively in India and Indian communities living abroad.

Tobacco

Smoking and other tobacco use are associated with about 75 percent of oral cancer cases, caused by irritation of the mucous membranes of the mouth from smoke and heat of cigarettes, cigars, and pipes. Tobacco contains over 60 known carcinogens, and the combustion of it, and by products from this process, is the primary mode of involvement. Use of chewing tobacco or snuff causes irritation from direct contact with the mucous membranes.

Alcohol

Alcohol use is another high-risk activity associated with oral cancer. There is known to be a very strong synergistic effect on oral cancer risk when a person is both a heavy smoker and drinker. The risk is greatly increased compared to a heavy smoker, or a heavy

drinker alone. Recent studies in Australia, Brazil and Germany point to alcohol-containing mouthwashes as also being etiologic agents in the oral cancer risk family. Constant exposure to these alcohol containing rinses, even in the absence of smoking and drinking, lead to significant increases in the development of oral cancer. A 2008 study suggests that acetaldehyde (a break-down product of alcohol) is implicated in oral cancer.

Human papillomavirus

Infection with human papillomavirus (HPV), particularly type 16 (there are over 120 types), is a known risk factor and independent causative factor for oral cancer. (Gilsion et al. Johns Hopkins) A fast growing segment of those diagnosed does not present with the historic stereotypical demographics. Historically that has been people over 50, blacks over whites 2 to 1, males over females 3 to 1, and 75% of the time people who have used tobacco products or are heavy users of alcohol. This new and rapidly growing sub population between 20 and 50 years old is predominantly non smoking, white, and males slightly outnumber females. Recent research from Johns Hopkins indicates that HPV is the primary risk factor in this new population of oral cancer victims. HPV16 (along with HPV18) is the same virus responsible for the vast majority of all cervical cancers and is the most common sexually transmitted infection in the US. Oral cancer in this group tends to favor the tonsil and tonsillar pillars, base of the tongue, and the oropharnyx. Recent data suggest that individuals that come to the disease from this particular etiology have some slight survival advantage.

Diagnosis

An examination of the mouth by the health care provider or dentist shows a visible and/or palpable (can be felt) lesion of the lip, tongue, or other mouth area. As the tumor enlarges, it may become an ulcer and bleed. Speech/talking difficulties, chewing problems, or swallowing difficulties may develop. A feeding tube is often necessary to maintain adequate nutrition. This can sometimes become permanent as eating difficulties can include the inability to swallow even a sip of water.

There are a variety of screening devices that may assist dentists in detecting oral cancer, including the Velscope, Vizilite Plus and the identafi 3000. While a dentist, physician or other health professional may suspect a particular lesion is malignant, there is no way to tell by looking alone - since benign and malignant lesions may look identical to the eye. A non-invasive brush biopsy (BrushTest) can be performed to rule out the presence of dysplasia (pre-cancer) and cancer on areas of the mouth that exhibit an unexplained color variation or lesion. The only definitive method for determining if cancerous or precancerous cells are present is through biopsy and microscopic evaluation of the cells in the removed sample. A tissue biopsy, whether of the tongue or other oral tissues and microscopic examination of the lesion confirm the diagnosis of oral cancer or precancer.

Management

Surgical excision (removal) of the tumor is usually recommended if the tumor is small enough, and if surgery is likely to result in a functionally satisfactory result. Radiation therapy with or without chemo is often used in conjunction with surgery, or as the definitive radical treatment, especially if the tumour is inoperable. Surgeries for oral cancers include

- Maxillectomy (can be done with or without Orbital exenteration)
- Mandibulectomy (removal of the mandible or lower jaw or part of it)
- Glossectomy (tongue removal, can be total, hemi or partial)
- Radical neck dissection
- Moh's procedure or CCPDMA
- Combinational e.g. glossectomy and laryngectomy done together.
- Feeding tube to sustain nutrition.

Owing to the vital nature of the structures in the head and neck area, surgery for larger cancers is technically demanding. Reconstructive surgery may be required to give an acceptable cosmetic and functional result. Bone grafts and surgical flaps such as the radial forearm flap are used to help rebuild the structures removed during excision of the cancer. An oral prothesis may also be required. Most oral cancer patients depend on a feeding tube for their hydration and nutrition. Some will also get a port for the chemo to be delivered. Many oral cancer patients are disfigured and suffer from many long term after effects. The after effects often include fatigue, speech problems, trouble maintaining weight, thyroid issues, swallowing difficulties, inability to swallow, memory loss, weakness, dizziness, high frequency hearing loss and sinus damage.

Survival rates for oral cancer depend on the precise site, and the stage of the cancer at diagnosis. Overall, survival is around 50% at five years when all stages of initial diagnosis are considered. Survival rates for stage 1 cancers are 90%, hence the emphasis on early detection to increase survival outcome for patients.

Following treatment, rehabilitation may be necessary to improve movement, chewing, swallowing, and speech. speech and language pathologists may be involved at this stage.

Chemotherapy is useful in oral cancers when used in combination with other treatment modalities such as radiation therapy. It is not used alone as a monotherapy. When cure is unlikely it can also be used to extend life and can be considered palliative but not curative care. Biological agents, such as Cetuximab have recently been shown to be effective in the treatment of squamous cell head and neck cancers, and are likely to have an increasing role in the future management of this condition when used in conjunction with other treatments.

Treatment of oral cancer will usually be by a multidisciplinary team, with treatment professionals from the realms of radiation, surgery, chemotherapy, nutrition, dental

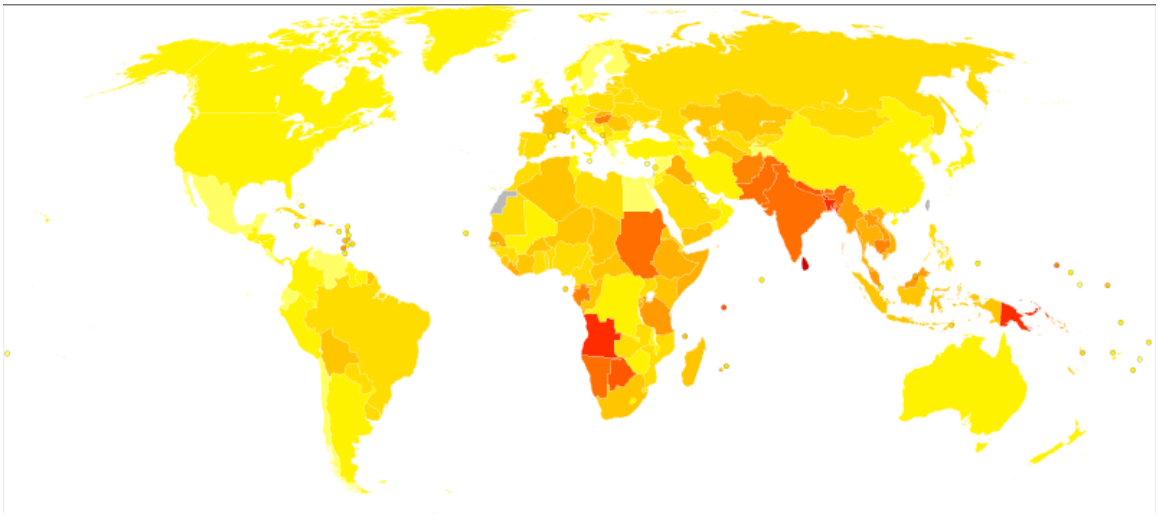
professionals, and even psychology all possibly involved with diagnosis, treatment, rehabilitation, and patient care.

The Oral Cancer Foundation is a website devoted to in depth medical information about all oral cancers including treatment, side effects and even lists of the nation's best cancer treatment centers. The Oral Cancer Foundation has a forum where patients and their caregivers assist each other. It is monitored by the founder and administrators who ensure accurate up to date information is exchanged. This website has the most comprehensive amount of information devoted to oral cancer.

Prognosis

- Postoperative disfigurement of the face, head and neck
- Complications of radiation therapy, including dry mouth and difficulty swallowing
- Other metastasis (spread) of the cancer
- Significant weight loss

Epidemiology



Age-standardized death from oro-pharyngeal per 100,000 inhabitants in 2004.

- no data
- less than 2
- 2-4
- 4-6
- 6-8
- 8-10
- 10-12
- 12-14
- 14-16
- 16-18
- 18-20

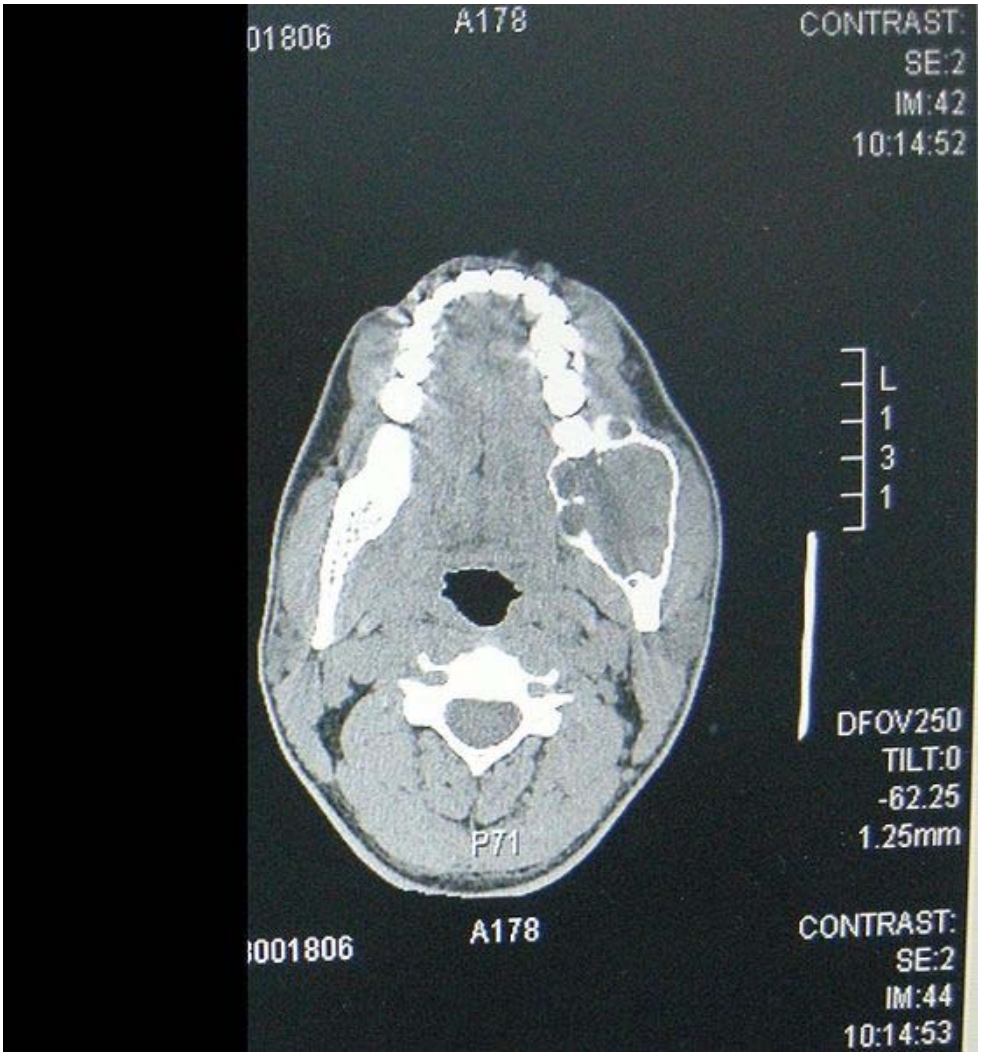

20-25
more than 25

In 2008, in the United States alone, about 34,000 individuals were diagnosed with oral cancer. 66% of the time these will be found as late stage three and four disease. Low public awareness of the disease is a significant factor, but these cancers could be found at early highly survivable stages through a simple, painless, 5 minute examination by a trained medical or dental professional.

Chapter 5

Ameloblastoma

Ameloblastoma



A CT scan of a patient suffering from an ameloblastoma

ICD-10 D16.5

ICD-9	213.1
ICD-O:	9310/0
DiseasesDB	31676
MeSH	D000564

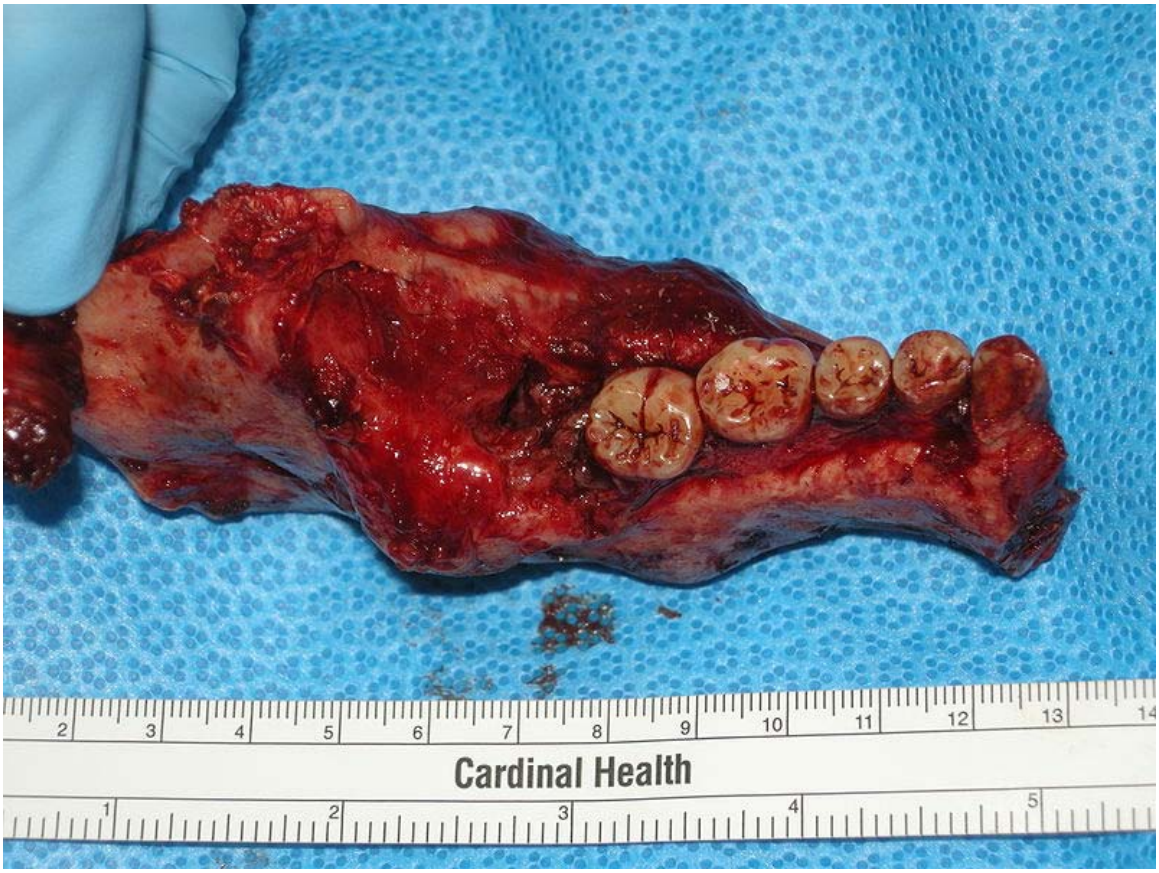
Ameloblastoma (from the early English word *amel*, meaning enamel + the Greek word *blastos*, meaning germ) is a rare, benign tumor of odontogenic epithelium (ameloblasts, or outside portion, of the teeth during development) much more commonly appearing in the mandible than the maxilla. It was recognized in 1827 by Cusack. This type of odontogenic neoplasm was designated as an *adamantinoma* in 1885 by the French physician Louis-Charles Malassez. It was finally renamed to the modern name *ameloblastoma* in 1930 by Ivey and Churchill.

While these tumors are rarely malignant or metastatic (that is, they rarely spread to other parts of the body), and progress slowly, the resulting lesions can cause severe abnormalities of the face and jaw. Additionally, because abnormal cell growth easily infiltrates and destroys surrounding bony tissues, wide surgical excision is required to treat this disorder.

Subtypes

There are three main clinical subtypes of ameloblastoma: unicystic, multicystic, peripheral. The peripheral subtype composes 2% of all ameloblastomas. Of all ameloblastomas in younger patients, unicystic ameloblastomas represent 6% of the cases. A fourth subtype, malignant, has been considered by some oncologic specialists, however, this form of the tumor is rare and may be simply a manifestation of one of the three main subtypes. Ameloblastoma also occurs in long bones, and another variant is Craniopharyngioma (Rathke's pouch tumour, Pituitary Ameloblastoma.)

Clinical features



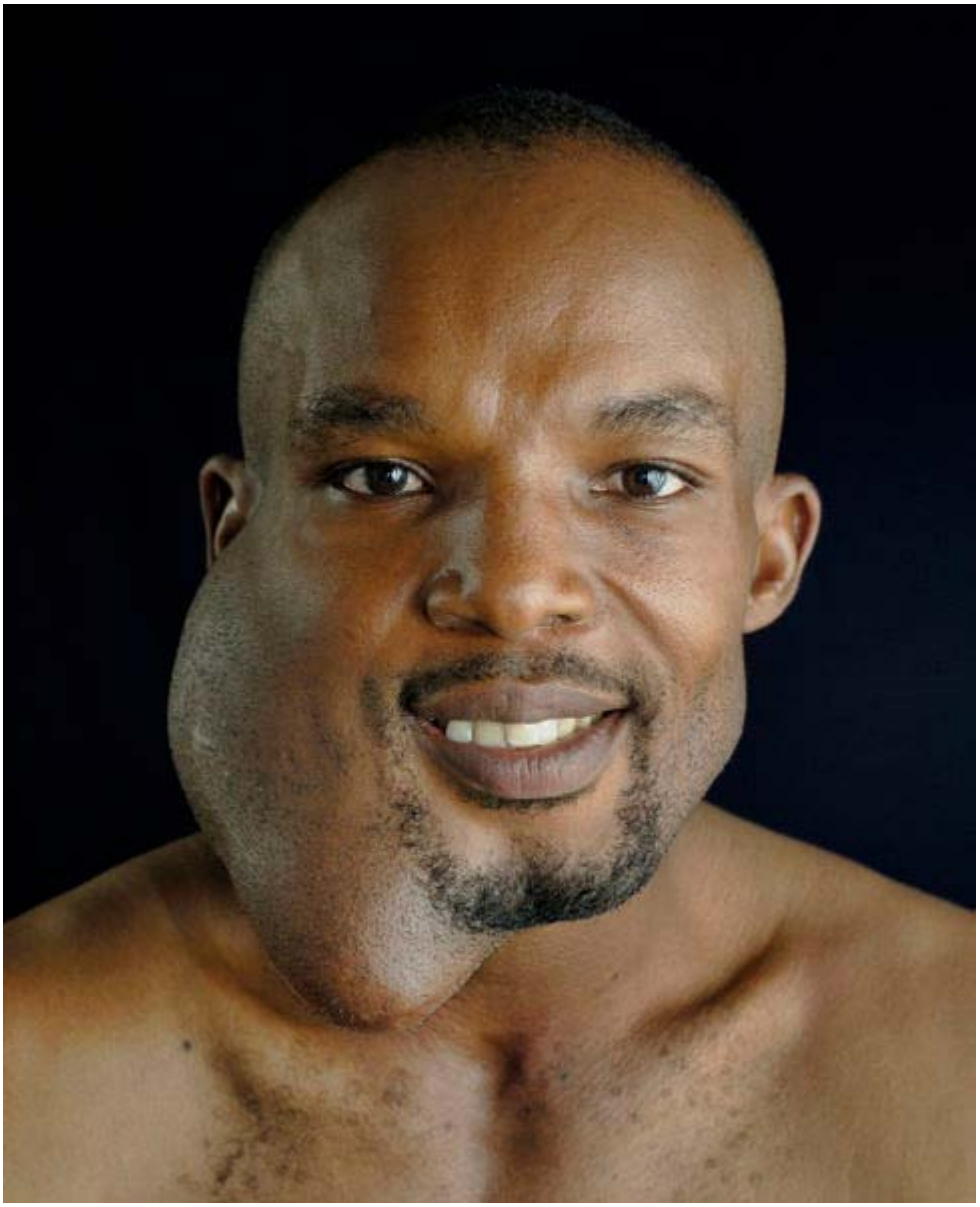
The resected left half of a mandible containing an ameloblastoma, initiated at the third molar

Ameloblastomas are often associated with the presence of unerupted teeth. Symptoms include painless swelling, facial deformity if severe enough, pain if the swelling impinges on other structures, loose teeth, ulcers, and periodontal (gum) disease. Lesions will occur in the mandible and maxilla,although 75% occur in the ascending ramus area and will result in extensive and grotesque deformitites of the mandible and maxilla. In the maxilla it can extend into the maxillary sinus and floor of the nose. The lesion has a tendency to expand the bony cortices because slow growth rate of the lesion allows time for periosteum to develop thin shell of bone ahead of the expanding lesion. This shell of bone cracks when palpated and this phenomenon is referred to as "Egg Shell Cracking" or crepitus, an important diagnostic feature. Ameloblastoma is tentatively diagnosed through radiographic examination and must be confirmed by histological examination (e.g., biopsy). Radiographically, it appears as a lucency in the bone of varying size and features—sometimes it is a single, well-demarcated lesion whereas it often demonstrates as a multiloculated "soap bubble" appearance. Resorption of roots of involved teeth can be seen in some cases, but is not unique to ameloblastoma. The disease is most often found in the posterior body and angle of the mandible, but can occur anywhere in either the maxilla or mandible.

Ameloblastoma is often associated with bony-impacted wisdom teeth—one of the many reasons dentists recommend having them extracted.

Histopathology

Histopathology will show cells that have the tendency to move the nucleus away from the basement membrane. This process is referred to as "Reverse Polarization". The follicular type will have outer arrangement of columnar or palisaded ameloblast like cells and inner zone of triangular shaped cells resembling stellate reticulum in bell stage. The central cells sometimes degenerate to form central microcysts. The plexiform type has epithelium that proliferates in a "Fish Net Pattern". The plexiform ameloblastoma shows epithelium proliferating in a 'cord like fashion', hence the name 'plexiform'. There are layers of cells in between the proliferating epithelium with a well-formed desmosomal junctions, simulating spindle cell layers.



Ameloblastoma

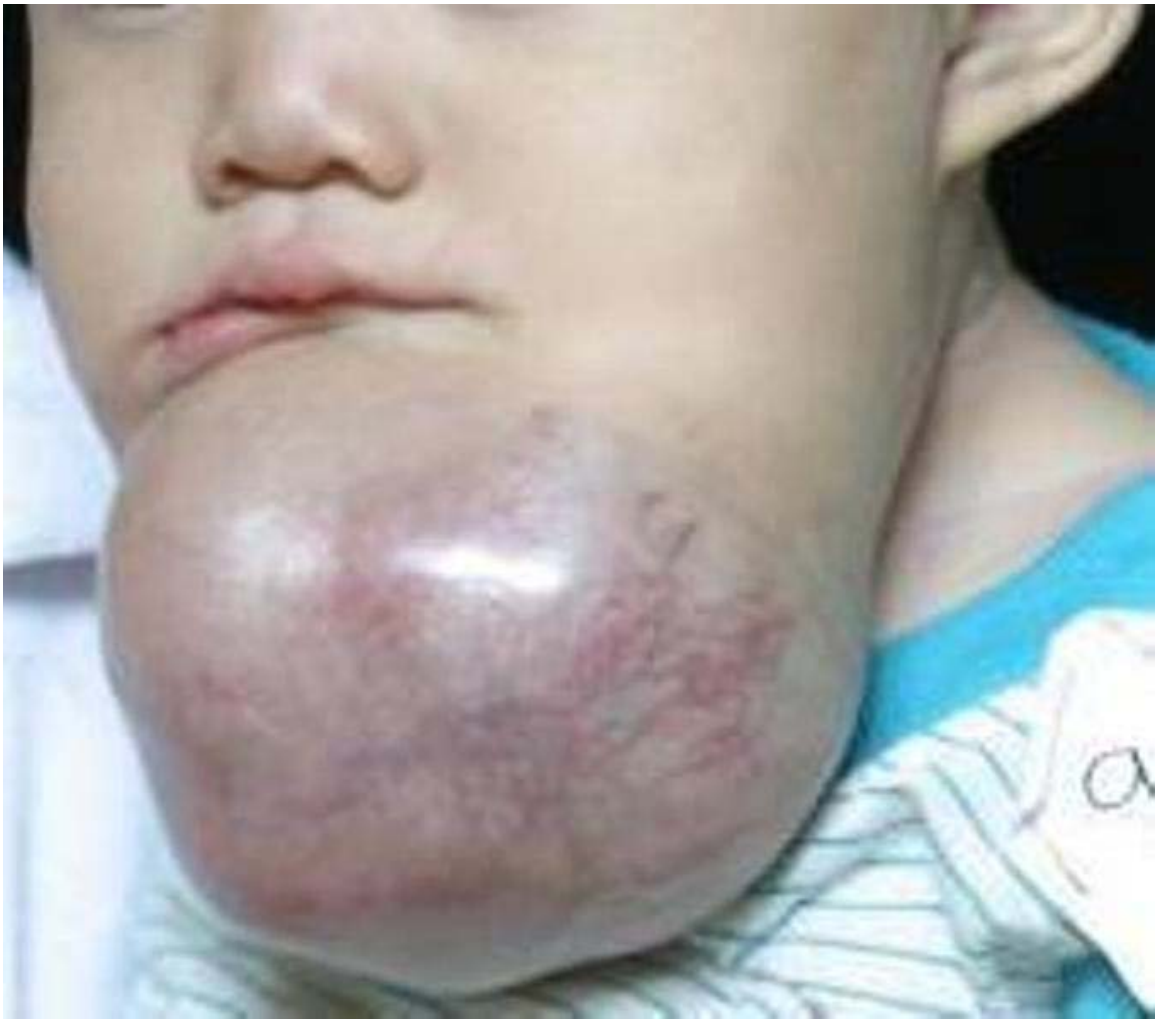
Variants

The six different histopathological variants of ameloblastoma are desmoplastic, granular cell, basal cell, plexiform, follicular, and acanthomatous.

The acanthomatous variant is extremely rare.

One-third of ameloblastomas are plexiform, one-third are follicular. Other variants such as acanthomatous occur in older patients. In one center, desmoplastic ameloblastomas represented about 9% of all ameloblastomas encountered.

Treatment



Tracheal intubation is anticipated to be difficult in this child with a massive ameloblastoma.

While chemotherapy, radiation therapy, curettage and liquid nitrogen have been effective in some cases of ameloblastoma, surgical resection or enucleation remains the most definitive treatment for this condition. In a detailed study of 345 patients, chemotherapy and radiation therapy seemed to be contraindicated for the treatment of ameloblastomas. Thus, surgery is the most common treatment of this tumor. Because of the invasive nature of the growth, excision of normal tissue near the tumor margin is often required. Some have likened the disease to basal cell carcinoma (a skin cancer) in its tendency to spread to adjacent bony and sometimes soft tissues without metastasizing. While not a cancer that actually invades adjacent tissues, ameloblastoma is suspected to spread to adjacent areas of the jaw bone via marrow space. Thus, wide surgical margins that are clear of disease are required for a good prognosis. This is very much like surgical treatment of cancer. Often, treatment requires excision of entire portions of the jaw.

Radiation is ineffective in many cases of ameloblastoma. There have also been reports of sarcoma being induced as the result of using radiation to treat ameloblastoma. Chemotherapy is also often ineffective. However, there is some controversy regarding this and some indication that some ameloblastomas might be more responsive to radiation that previously thought.

While the Mayo Clinic recommends surgery for almost all ameloblastomas, there are situations in which a Mayo Clinic physician might recommend radiation therapy. These include malignancy, inability to completely remove the ameloblastoma, recurrence, unacceptable loss of function, and unacceptable cosmetic damage. In the case of radiotherapy, oncologists at the Mayo Clinic would use intensity-modulated radiotherapy.

Molecular biology

There is evidence that suppression of matrix metalloproteinase-2 may inhibit the local invasiveness of ameloblastoma, however, this was only demonstrated *in vitro*. There is also some research suggesting that $\alpha_5\beta_1$ integrin may participate in the local invasiveness of ameloblastomas.

Recurrence

Recurrence is common, although the recurrence rates for block resection followed by bone graft are lower than those of enucleation and curettage. Follicular variants appear to recur more than plexiform variants. Unicystic tumors recur less frequently than "non-unicystic" tumors. Persistent follow-up examination is essential for managing ameloblastoma. Follow up should occur at regular intervals for at least 10 years. Follow up is important, because 50% of all recurrences occur within 5 years postoperatively. Recurrence within a bone graft (following resection of the original tumor) does occur, but is less common. Seeding to the bone graft is suspected as a cause of recurrence. The recurrences in these cases seem to stem from the soft tissues, especially the adjacent periosteum. Recurrence has been reported to occur as many as 36 years after treatment.

To reduce the likelihood of recurrence within grafted bone, meticulous surgery with attention to the adjacent soft tissues is required.

Epidemiology

The annual incidence rates per million for ameloblastomas are 1.96, 1.20, 0.18 and 0.44 for black males, black females, white males and white females respectively. Ameloblastomas account for about one percent of all oral tumors and about 18% of odontogenic tumors. Men and women tend to be equally affected, although women tend to be 4 years younger than men when tumors first occur and tumors appear to be larger in females.

Chapter 6

Dental Caries

Dental caries

Destruction of a tooth by cervical decay from dental caries.
This type of decay is also known as root decay.

ICD-10	K02.
ICD-9	521.0
DiseasesDB	29357
MedlinePlus	001055

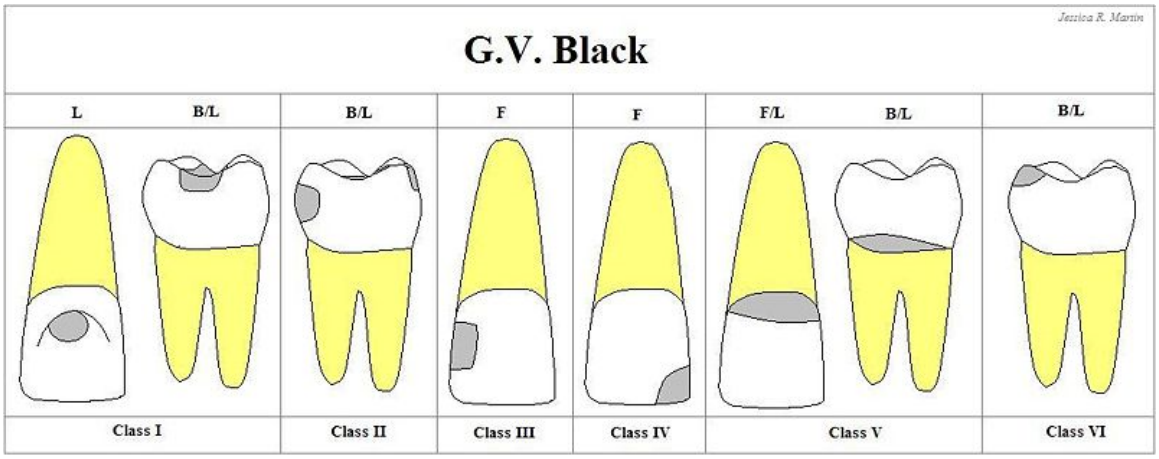
Dental caries, also known as **tooth decay** or a **cavity**, is a disease where bacterial processes damage hard tooth structure (enamel, dentin, and cementum). These tissues progressively break down, producing dental caries (cavities, holes in the teeth). Two groups of bacteria are responsible for initiating caries: *Streptococcus mutans* and *Lactobacillus*. If left untreated, the disease can lead to pain, tooth loss, infection, and, in severe cases, death. Today, caries remains one of the most common diseases throughout the world. Cariology is the study of dental caries.

The presentation of caries is highly variable; however, the risk factors and stages of development are similar. Initially, it may appear as a small chalky area that may eventually develop into a large cavitation. Sometimes caries may be directly visible, however other methods of detection such as radiographs are used for less visible areas of teeth and to judge the extent of destruction.

Tooth decay is caused by specific types of acid-producing bacteria that cause damage in the presence of fermentable carbohydrates such as sucrose, fructose, and glucose. The mineral content of teeth is sensitive to increases in acidity from the production of lactic acid. Specifically, a tooth (which is primarily mineral in content) is in a constant state of back-and-forth demineralization and remineralization between the tooth and surrounding saliva. When the pH at the surface of the tooth drops below 5.5, demineralization proceeds faster than remineralization (meaning that there is a net loss of mineral structure on the tooth's surface). This results in the ensuing decay. Depending on the extent of tooth destruction, various treatments can be used to restore teeth to proper form, function, and aesthetics, but there is no known method to regenerate large amounts of tooth structure, though stem cell related research suggests one possibility. Instead, dental health organizations advocate preventive and prophylactic measures, such as regular oral hygiene and dietary modifications, to avoid dental caries.

Classification

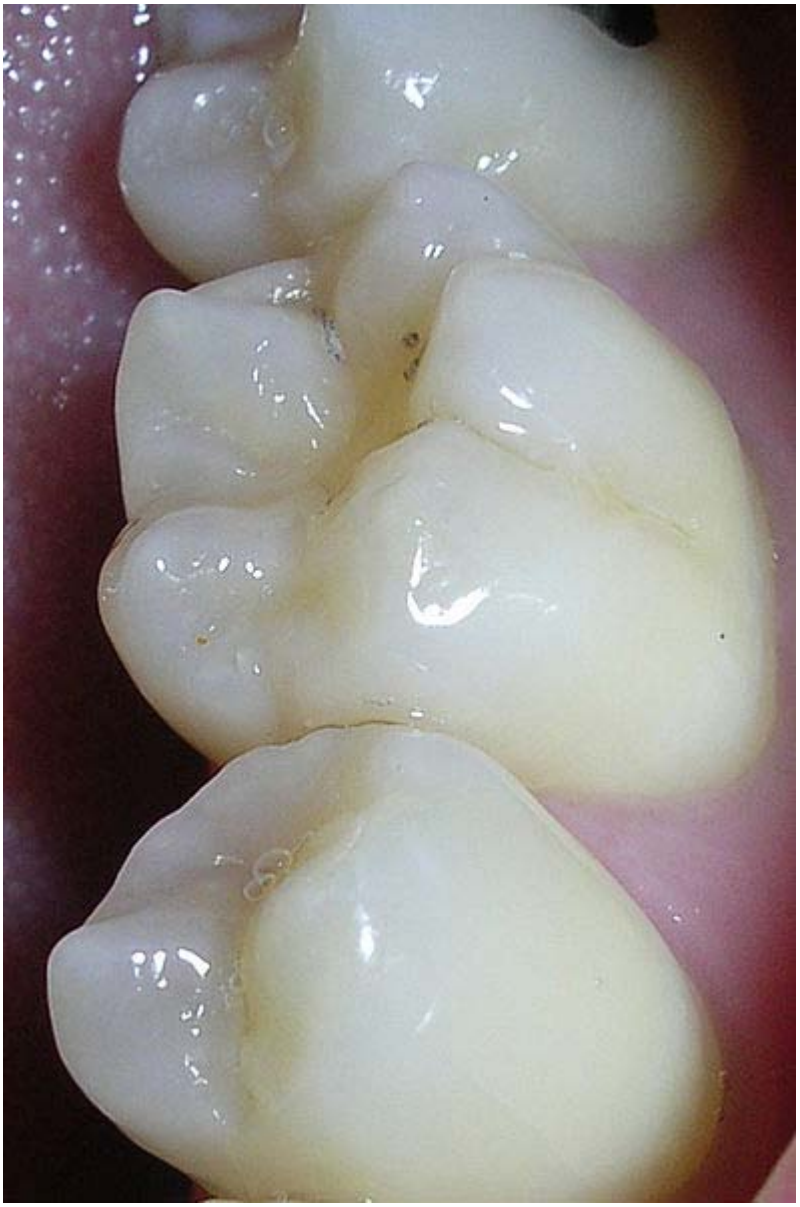
Caries can be classified by location, etiology, rate of progression, and affected hard tissues. These forms of classification can be used to characterize a particular case of tooth decay in order to more accurately represent the condition to others and also indicate the severity of tooth destruction.



G.V. Black Classification of Restorations

Location

Generally, there are two types of caries when separated by location: caries found on smooth surfaces and caries found in pits and fissures. The location, development, and progression of smooth-surface caries differ from those of pit and fissure caries. G.V. Black created a classification system that is widely used and based on the location of the caries on the tooth. The original classification distinguished caries into five groups, indicated by the word "Class", and a Roman numeral. Pit and fissure caries is indicated as Class I; smooth surface caries is further divided into Class II, Class III, Class IV, and Class V. A Class VI was added onto Black's Classification of Caries Lesions and also represents a smooth-surface carious lesion.



The pits and fissures of teeth provide a location for caries formation.

Pit and fissure caries (class I dental caries)

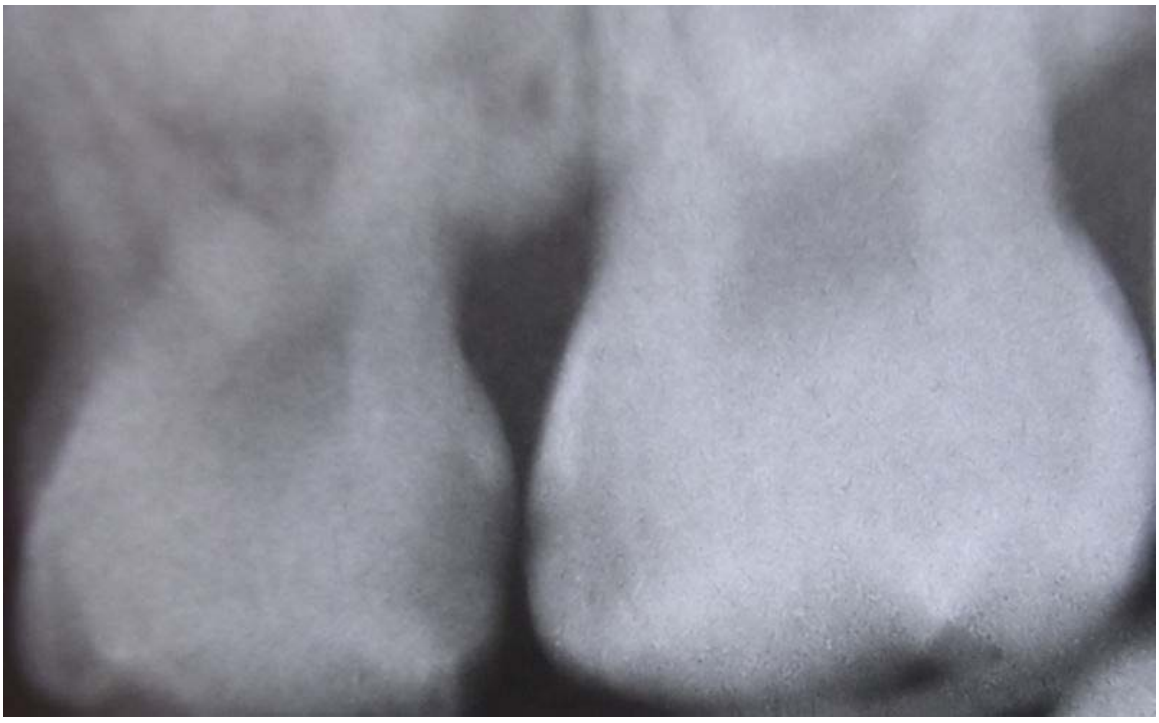
Pits and fissures are anatomic landmarks on a tooth where the enamel folds inward. Fissures are formed during the development of grooves but the enamel in the area is not fully fused. As a result, a deep linear depression forms in the enamel's surface structure, which forms a location for dental caries to develop and flourish. Fissures are mostly located on the occlusal (chewing) surfaces of posterior (rear) teeth and palatal surfaces of maxillary anterior (front) teeth. Pits are small, pinpoint depressions that are most commonly found at the ends or cross-sections of grooves. In particular, buccal pits are found on the facial surfaces of molars. For all types of pits and fissures, the deep

infolding of enamel makes oral hygiene along these surfaces difficult, allowing dental caries to develop more commonly in these areas.

The occlusal surfaces of teeth represent 12.5% of all tooth surfaces but are the location of over 50% of all dental caries. Among children, pit and fissure caries represent 90% of all dental caries. Pit and fissure caries can sometimes be difficult to detect. As the decay progresses, caries in enamel nearest the surface of the tooth spreads gradually deeper. Once the caries reaches the dentin at the dentino-enamel junction (DEJ), the decay quickly spreads laterally. Within the dentin, the decay follows a triangle pattern that points to the tooth's pulp. This pattern of decay is typically described as two triangles (one triangle in enamel, and another in dentin) with their bases conjoined to each other at the DEJ. This base-to-base pattern is typical of pit and fissure caries, unlike smooth-surface caries (where base and apex of the two triangles join).

Smooth-surface caries

There are three types of smooth-surface caries. Proximal caries, also called interproximal caries, form on the smooth surfaces between adjacent teeth. Root caries form on the root surfaces of teeth. The third type of smooth-surface caries occur on any other smooth tooth surface.



In this radiograph, the dark spots in the adjacent teeth show proximal caries.

Proximal caries are the most difficult type to detect. Frequently, this type of caries cannot be detected visually or manually with a dental explorer. Proximal caries form cervically (toward the roots of a tooth) just under the contact between two teeth. As a result,

radiographs are needed for early discovery of proximal caries. Under Black's classification system, proximal caries on posterior teeth (premolars and molars) are designated as Class II caries. Proximal caries on anterior teeth (incisors and canines) are indicated as Class III if the incisal edge (chewing surface) is not included and Class IV if the incisal edge is included.

Root caries, which are sometimes described as a category of smooth-surfaces caries, are the third most common type of caries and usually occur when the root surfaces have been exposed due to gingival recession. When the gingiva is healthy, root caries is unlikely to develop because the root surfaces are not as accessible to bacterial plaque. The root surface is more vulnerable to the demineralization process than enamel because cementum begins to demineralize at 6.7 pH, which is higher than enamel's critical pH. Regardless, it is easier to arrest the progression of root caries than enamel caries because roots have a greater reuptake of fluoride than enamel. Root caries are most likely to be found on facial surfaces, then interproximal surfaces, then lingual surfaces. Mandibular molars are the most common location to find root caries, followed by mandibular premolars, maxillary anteriors, maxillary posteriors, and mandibular anteriors.

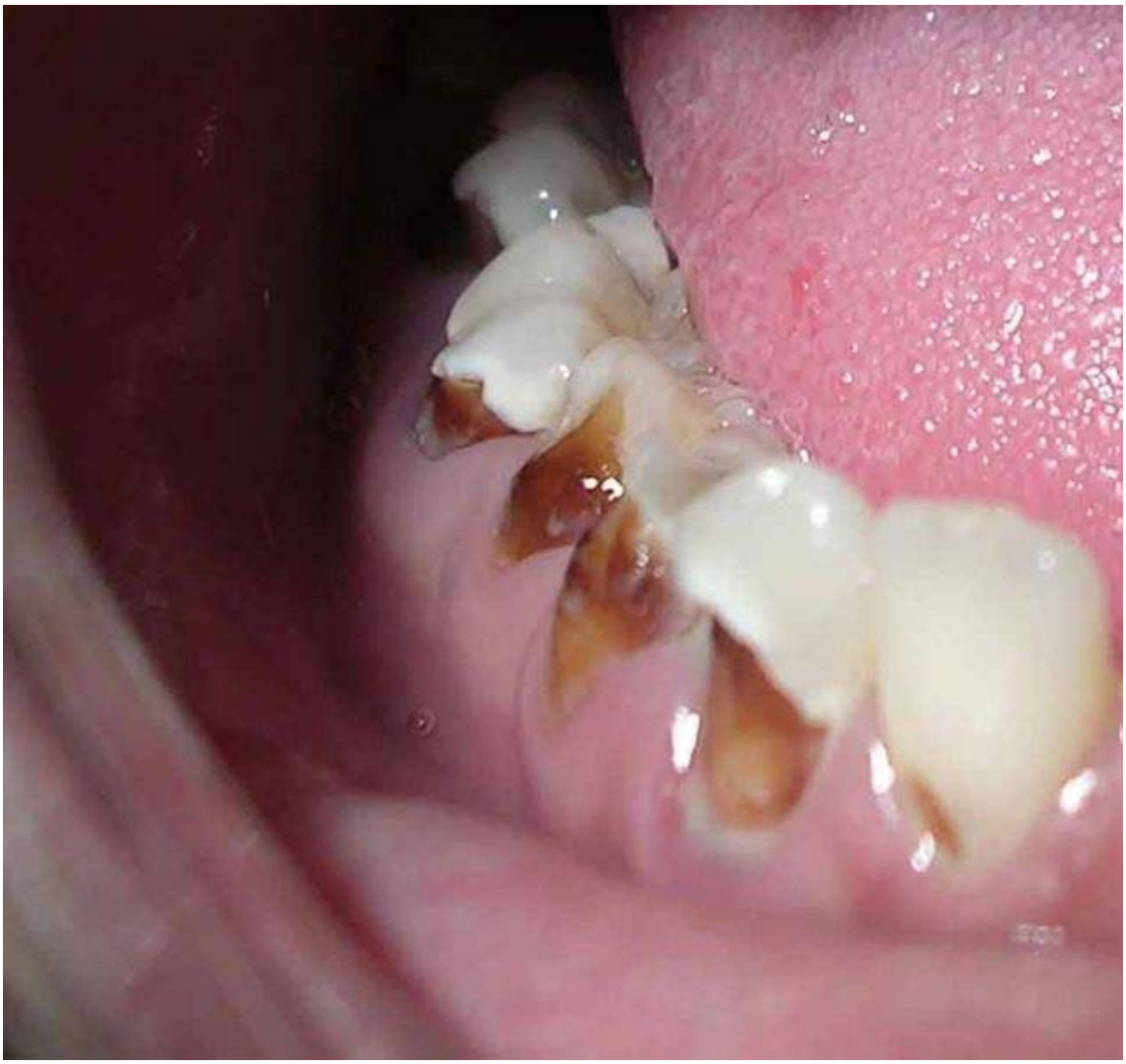
Lesions on other smooth surfaces of teeth are also possible. Since these occur in all smooth surface areas of enamel except for interproximal areas, these types of caries are easily detected and are associated with high levels of plaque and diets promoting caries formation. Under Black's classification system, caries near the gingiva on the facial or lingual surfaces is designated Class V. Class VI is reserved for caries confined to cusp tips on posterior teeth or incisal edges of anterior teeth.

Other general descriptions

Besides the two previously mentioned categories, carious lesions can be described further by their location on a particular surface of a tooth. Caries on a tooth's surface that are nearest the cheeks or lips are called "facial caries", and caries on surfaces facing the tongue are known as "lingual caries". Facial caries can be subdivided into buccal (when found on the surfaces of posterior teeth nearest the cheeks) and labial (when found on the surfaces of anterior teeth nearest the lips). Lingual caries can also be described as palatal when found on the lingual surfaces of maxillary teeth because they are located beside the hard palate.

Caries near a tooth's cervix—the location where the crown of a tooth and its roots meet—are referred to as cervical caries. Occlusal caries are found on the chewing surfaces of posterior teeth. Incisal caries are caries found on the chewing surfaces of anterior teeth. Caries can also be described as "mesial" or "distal." Mesial signifies a location on a tooth closer to the median line of the face, which is located on a vertical axis between the eyes, down the nose, and between the contact of the central incisors. Locations on a tooth further away from the median line are described as distal.

Etiology



Rampant caries.

In some instances, caries are described in other ways that might indicate the cause. "Baby bottle caries", "early childhood caries", or "baby bottle tooth decay" is a pattern of decay found in young children with their deciduous (baby) teeth. The teeth most likely affected are the maxillary anterior teeth, but all teeth can be affected. The name for this type of caries comes from the fact that the decay usually is a result of allowing children to fall asleep with sweetened liquids in their bottles or feeding children sweetened liquids multiple times during the day. Another pattern of decay is "rampant caries", which signifies advanced or severe decay on multiple surfaces of many teeth. Rampant caries may be seen in individuals with xerostomia, poor oral hygiene, stimulant use (due to drug-induced dry mouth), and/or large sugar intake. If rampant caries is a result of previous radiation to the head and neck, it may be described as radiation-induced caries. Problems can also be caused by the self destruction of roots and whole tooth resorption when new teeth erupt or later from unknown causes. Dr. Miller stated in 1887 that

"Dental decay is chemico-parasitic process consisting of two stages, the decalcification of enamel, which results in its total destruction and the decalcification of dentin as a preliminary stage followed by dissolution of the softened residue." In his hypothesis, Dr.Miller assigned essential roles to three factors:

1. Carbohydrate substrate.
2. Acid which caused dissolution of tooth minerals.
3. Oral micro organisms which produce acid and also cause proteolysis.

Rate of progression

Temporal descriptions can be applied to caries to indicate the progression rate and previous history. "Acute" signifies a quickly developing condition, whereas "chronic" describes a condition which has taken an extended time to develop where thousands of meals and snacks, many causing some acid demineralisation that is not remineralized and eventually results in cavities. Fluoride treatment can help recalcification of tooth enamel.

Recurrent caries, also described as secondary, are caries that appears at a location with a previous history of caries. This is frequently found on the margins of fillings and other dental restorations. On the other hand, incipient caries describes decay at a location that has not experienced previous decay. Arrested caries describes a lesion on a tooth which was previously demineralized but was remineralized before causing a cavitation. Using fluoride treatments can help with recalcification.

Affected hard tissue

Depending on which hard tissues are affected, it is possible to describe caries as involving enamel, dentin, or cementum. Early in its development, caries may affect only enamel. Once the extent of decay reaches the deeper layer of dentin, "dentinal caries" is used. Since cementum is the hard tissue that covers the roots of teeth, it is not often affected by decay unless the roots of teeth are exposed to the mouth. Although the term "cementum caries" may be used to describe the decay on roots of teeth, very rarely does caries affect the cementum alone. Roots have a very thin layer of cementum over a large layer of dentin, and thus most caries affecting cementum also affects dentin.

Signs and symptoms

The tip of a dental explorer, which is used for caries diagnosis.

A person experiencing caries may not be aware of the disease. The earliest sign of a new carious lesion is the appearance of a chalky white spot on the surface of the tooth, indicating an area of demineralization of enamel. This is referred to as incipient decay. As the lesion continues to demineralize, it can turn brown but will eventually turn into a cavitation ("cavity"). Before the cavity forms, the process is reversible, but once a cavity forms, the lost tooth structure cannot be regenerated. A lesion which appears brown and shiny suggests dental caries were once present but the demineralization process has stopped, leaving a stain. A brown spot which is dull in appearance is probably a sign of active caries.

As the enamel and dentin are destroyed, the cavity becomes more noticeable. The affected areas of the tooth change color and become soft to the touch. Once the decay passes through enamel, the dentinal tubules, which have passages to the nerve of the tooth, become exposed and causes pain in the tooth. The pain may worsen with exposure to heat, cold, or sweet foods and drinks. Dental caries can also cause bad breath and foul tastes. In highly progressed cases, infection can spread from the tooth to the surrounding soft tissues. Complications such as cavernous sinus thrombosis and Ludwig's angina can be life-threatening.

Causes

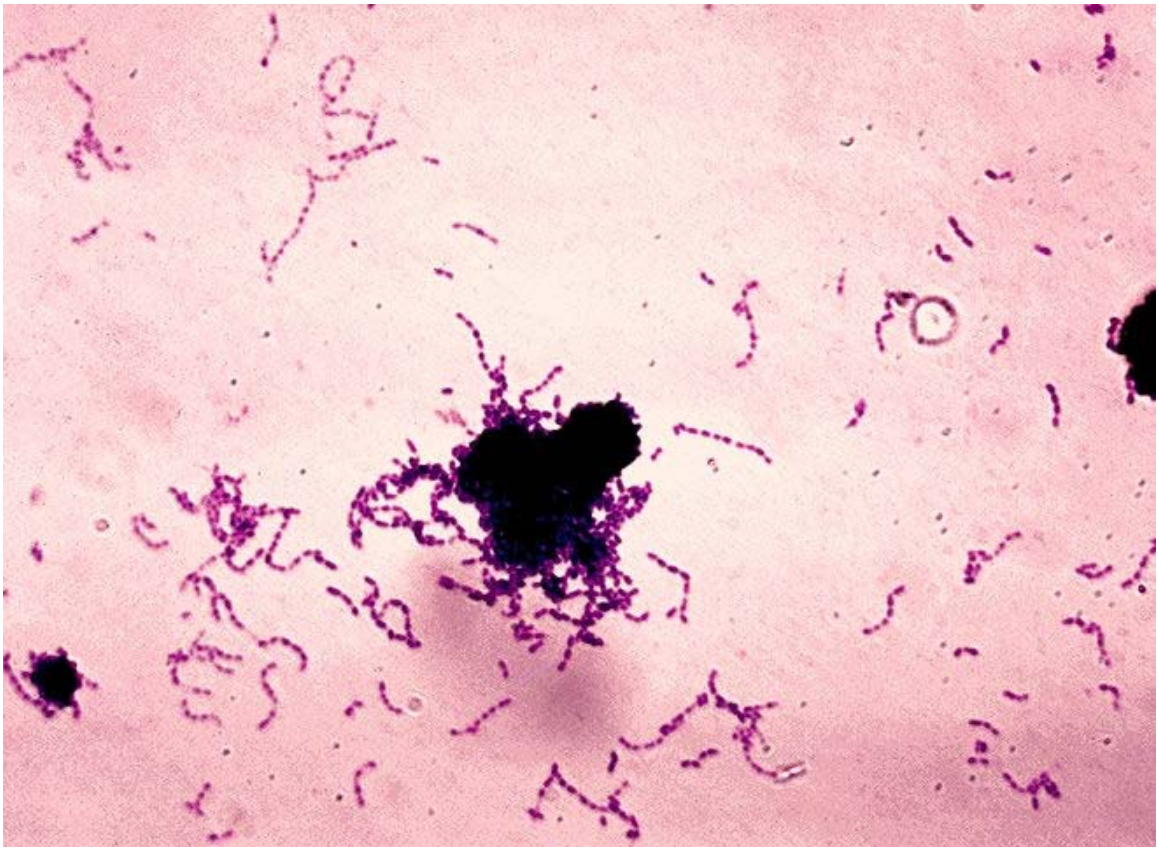
There are four main criteria required for caries formation: a tooth surface (enamel or dentin); caries-causing bacteria; fermentable carbohydrates (such as sucrose); and time. The caries process does not have an inevitable outcome, and different individuals will be susceptible to different degrees depending on the shape of their teeth, oral hygiene habits, and the buffering capacity of their saliva. Dental caries can occur on any surface of a tooth which is exposed to the oral cavity, but not the structures which are retained within the bone.

Teeth

There are certain diseases and disorders affecting teeth which may leave an individual at a greater risk for caries. Amelogenesis imperfecta, which occurs between 1 in 718 and 1 in 14,000 individuals, is a disease in which the enamel does not fully form or forms in insufficient amounts and can fall off a tooth. In both cases, teeth may be left more vulnerable to decay because the enamel is not able to protect the tooth.

In most people, disorders or diseases affecting teeth are not the primary cause of dental caries. Ninety-six percent of tooth enamel is composed of minerals. These minerals, especially hydroxyapatite, will become soluble when exposed to acidic environments. Enamel begins to demineralize at a pH of 5.5. Dentin and cementum are more susceptible to caries than enamel because they have lower mineral content. Thus, when root surfaces of teeth are exposed from gingival recession or periodontal disease, caries can develop more readily. Even in a healthy oral environment, however, the tooth is susceptible to dental caries.

The anatomy of teeth may affect the likelihood of caries formation. Where the deep grooves of teeth are more numerous and exaggerated, pit and fissure caries are more likely to develop. Also, caries are more likely to develop when food is trapped between teeth.



A gram stain image of *Streptococcus mutans*.

Bacteria

The mouth contains a wide variety of oral bacteria, but only a few specific species of bacteria are believed to cause dental caries: *Streptococcus mutans* and *Lactobacilli* among them. *Lactobacillus acidophilus*, *Actinomyces viscosus*, *Nocardia spp.*, and *Streptococcus mutans* are most closely associated with caries, particularly root caries. Bacteria collect around the teeth and gums in a sticky, creamy-coloured mass called plaque, which serves as a biofilm. Some sites collect plaque more commonly than others. The grooves on the biting surfaces of molar and premolar teeth provide microscopic retention, as does the point of contact between teeth. Plaque may also collect along the gingiva.

Fermentable carbohydrates

Bacteria in a person's mouth convert glucose, fructose, and most commonly sucrose (table sugar) into acids such as lactic acid through a glycolytic process called fermentation. If left in contact with the tooth, these acids may cause demineralization, which is the dissolution of its mineral content. The process is dynamic, however, as remineralization can also occur if the acid is neutralized by saliva or mouthwash. Fluoride toothpaste or dental varnish may aid remineralization. If demineralization continues over time, enough mineral content may be lost so that the soft organic material

left behind disintegrates, forming a cavity or hole. The impact such sugars have on the progress of dental caries is called cariogenicity. Sucrose, although a bound glucose and fructose unit, is in fact more cariogenic than a mixture of equal parts of glucose and fructose. This is due to the bacteria utilising the energy in the saccharide bond between the glucose and fructose subunits. *S.mutans* adheres to the biofilm on the tooth by converting sucrose into an extremely adhesive substance called dextran polysaccharide by the enzyme dextransucranase.

Time

The frequency of which teeth are exposed to cariogenic (acidic) environments affects the likelihood of caries development. After meals or snacks, the bacteria in the mouth metabolize sugar, resulting in an acidic by-product which decreases pH. As time progresses, the pH returns to normal due to the buffering capacity of saliva and the dissolved mineral content of tooth surfaces. During every exposure to the acidic environment, portions of the inorganic mineral content at the surface of teeth dissolves and can remain dissolved for two hours. Since teeth are vulnerable during these acidic periods, the development of dental caries relies heavily on the frequency of acid exposure.

The carious process can begin within days of a tooth erupting into the mouth if the diet is sufficiently rich in suitable carbohydrates. Evidence suggests that the introduction of fluoride treatments have slowed the process. Proximal caries take an average of four years to pass through enamel in permanent teeth. Because the cementum enveloping the root surface is not nearly as durable as the enamel encasing the crown, root caries tends to progress much more rapidly than decay on other surfaces. The progression and loss of mineralization on the root surface is 2.5 times faster than caries in enamel. In very severe cases where oral hygiene is very poor and where the diet is very rich in fermentable carbohydrates, caries may cause cavities within months of tooth eruption. This can occur, for example, when children continuously drink sugary drinks from baby bottles.

Other risk factors

Reduced saliva is associated with increased caries since the buffering capability of saliva is not present to counterbalance the acidic environment created by certain foods. As result, medical conditions that reduce the amount of saliva produced by salivary glands, particularly the submandibular gland and parotid gland, are likely to lead to widespread tooth decay. Examples include Sjögren's syndrome, diabetes mellitus, diabetes insipidus, and sarcoidosis. Medications, such as antihistamines and antidepressants, can also impair salivary flow. Stimulants, most notoriously methylamphetamine, also occlude the flow of saliva to an extreme degree. Abusers of stimulants tend to have poor oral hygiene. Tetrahydrocannabinol, the active chemical substance in cannabis, also causes a nearly complete occlusion of salivation, known colloquially as "cotton mouth". Moreover, sixty-three percent of the most commonly prescribed medications in the United States list dry mouth as a known side effect. Radiation therapy of the head and neck may also damage the cells in salivary glands, increasing the likelihood of caries formation.

The use of tobacco may also increase the risk for caries formation. Some brands of smokeless tobacco contain high sugar content, increasing susceptibility to caries. Tobacco use is a significant risk factor for periodontal disease, which can cause the gingiva to recede. As the gingiva loses attachment to the teeth, the root surface becomes more visible in the mouth. If this occurs, root caries is a concern since the cementum covering the roots of teeth is more easily demineralized by acids than enamel. Currently, there is not enough evidence to support a causal relationship between smoking and coronal caries, but evidence does suggest a relationship between smoking and root-surface caries.

Intrauterine and neonatal lead exposure promote tooth decay. Besides lead, all atoms with electrical charge and ionic radius similar to bivalent calcium, such as cadmium, mimic the calcium ion and therefore exposure may promote tooth decay.

Salivary and dietary iodine seems to play an important role in pathogenesis of dental caries and in salivary glands physiology. Saliva is rich in peroxidase enzymes and has high secretion of iodides. Iodine is able to penetrate directly through intact enamel in dentine, pulp and periodontal tissues and according to some researchers it is able to prevent some dental pathologies directly with antibacterial action, and also indirectly with an antioxidant mechanism.

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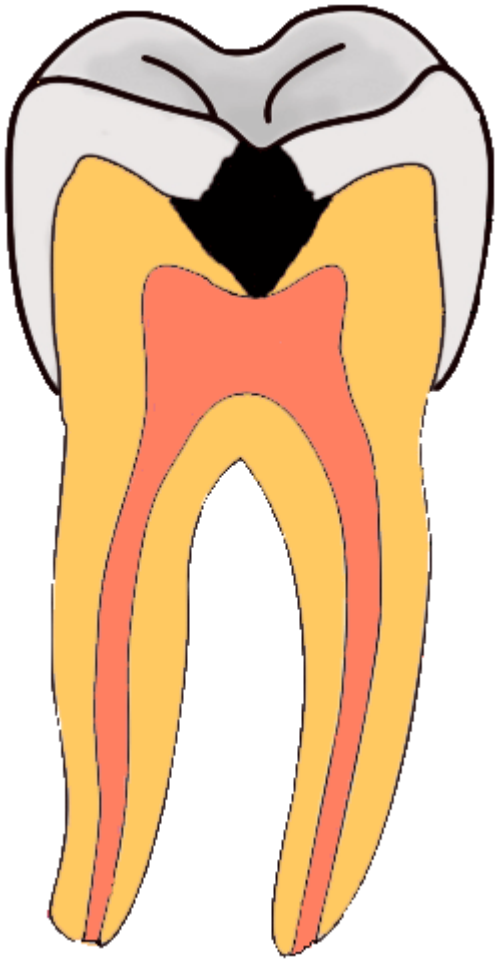
Pathophysiology



The progression of pit and fissure caries resembles two triangles with their bases meeting along the junction of enamel and dentin.

Enamel

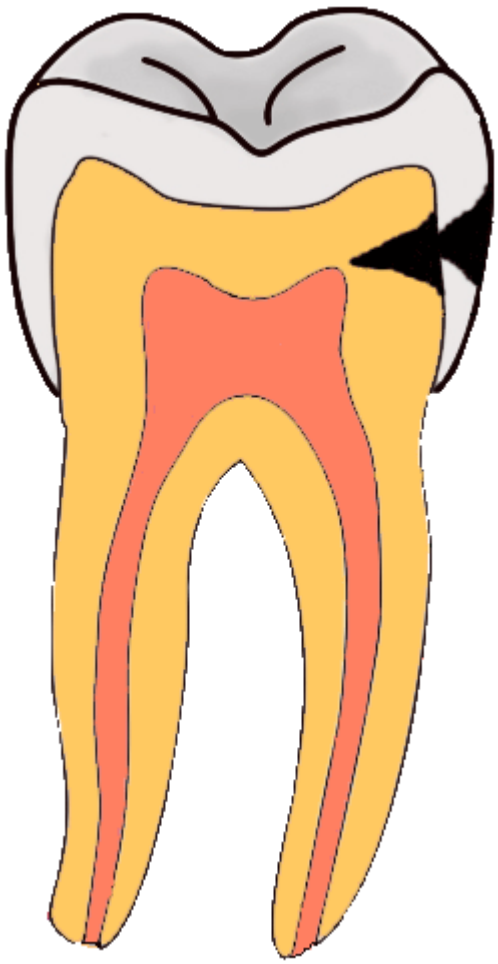
Enamel is a highly mineralized acellular tissue, and caries act upon it through a chemical process brought on by the acidic environment produced by bacteria. As the bacteria consume the sugar and use it for their own energy, they produce lactic acid. The effects of this process include the demineralization of crystals in the enamel, caused by acids, over time until the bacteria physically penetrate the dentin. Enamel rods, which are the basic unit of the enamel structure, run perpendicularly from the surface of the tooth to the dentin. Since demineralization of enamel by caries generally follows the direction of the enamel rods, the different triangular patterns between pit and fissure and smooth-surface caries develop in the enamel because the orientation of enamel rods are different in the two areas of the tooth.

As the enamel loses minerals, and dental caries progresses, the enamel develop several distinct zones, visible under a light microscope. From the deepest layer of the enamel to the enamel surface, the identified areas are the: translucent zone, dark zones, body of the lesion, and surface zone. The translucent zone is the first visible sign of caries and coincides with a one to two percent loss of minerals. A slight remineralization of enamel occurs in the dark zone, which serves as an example of how the development of dental caries is an active process with alternating changes. The area of greatest demineralization and destruction is in the body of the lesion itself. The surface zone remains relatively mineralized and is present until the loss of tooth structure results in a cavitation.

Dentin

Unlike enamel, the dentin reacts to the progression of dental caries. After tooth formation, the ameloblasts, which produce enamel, are destroyed once enamel formation is complete and thus cannot later regenerate enamel after its destruction. On the other hand, dentin is produced continuously throughout life by odontoblasts, which reside at the border between the pulp and dentin. Since odontoblasts are present, a stimulus, such as caries, can trigger a biologic response. These defense mechanisms include the formation of sclerotic and tertiary dentin.

In dentin from the deepest layer to the enamel, the distinct areas affected by caries are the translucent zone, the zone of destruction, and the zone of bacterial penetration. The translucent zone represents the advancing front of the carious process and is where the initial demineralization begins. The zones of bacterial penetration and destruction are the locations of invading bacteria and ultimately the decomposition of dentin.



The faster spread of caries through dentin creates this triangular appearance in smooth surface caries.

Sclerotic dentin

The structure of dentin is an arrangement of microscopic channels, called dentinal tubules, which radiate outward from the pulp chamber to the exterior cementum or enamel border. The diameter of the dentinal tubules is largest near the pulp (about 2.5 μm) and smallest (about 900 nm) at the junction of dentin and enamel. The carious process continues through the dentinal tubules, which are responsible for the triangular patterns resulting from the progression of caries deep into the tooth. The tubules also allow caries to progress faster.

In response, the fluid inside the tubules bring immunoglobulins from the immune system to fight the bacterial infection. At the same time, there is an increase of mineralization of the surrounding tubules. This results in a constriction of the tubules, which is an attempt to slow the bacterial progression. In addition, as the acid from the bacteria demineralizes the hydroxyapatite crystals, calcium and phosphorus are released, allowing for the

precipitation of more crystals which fall deeper into the dentinal tubule. These crystals form a barrier and slow the advancement of caries. After these protective responses, the dentin is considered sclerotic.

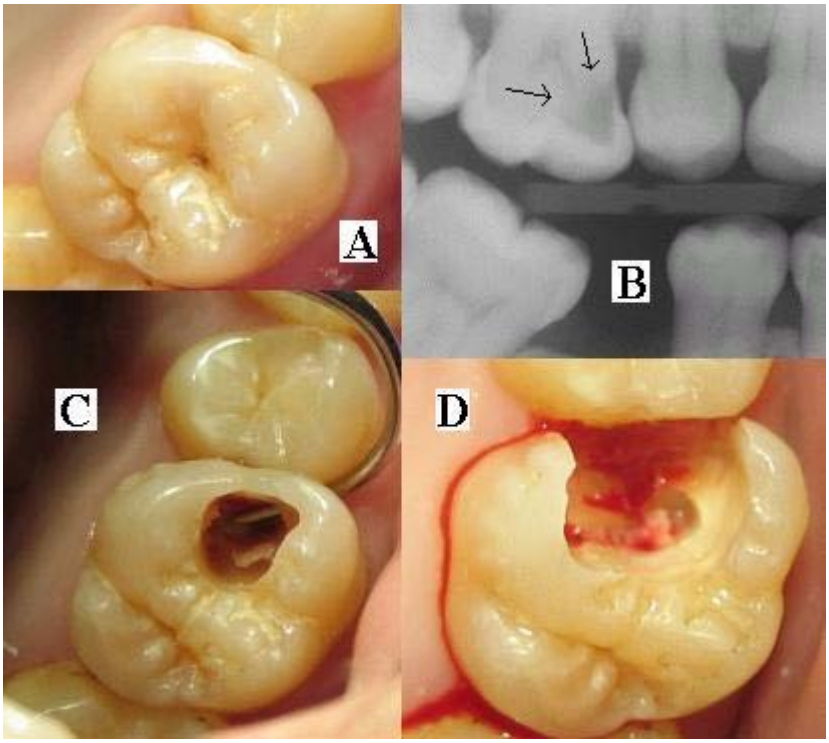
Fluids within dentinal tubules are believed to be the mechanism by which pain receptors are triggered within the pulp of the tooth. Since sclerotic dentin prevents the passage of such fluids, pain that would otherwise serve as a warning of the invading bacteria may not develop at first. Consequently, dental caries may progress for a long period of time without any sensitivity of the tooth, allowing for greater loss of tooth structure.

Tertiary dentin

In response to dental caries, there may be production of more dentin toward the direction of the pulp. This new dentin is referred to as tertiary dentin. Tertiary dentin is produced to protect the pulp for as long as possible from the advancing bacteria. As more tertiary dentin is produced, the size of the pulp decreases. This type of dentin has been subdivided according to the presence or absence of the original odontoblasts. If the odontoblasts survive long enough to react to the dental caries, then the dentin produced is called "reactionary" dentin. If the odontoblasts are killed, the dentin produced is called "reparative" dentin.

In the case of reparative dentin, other cells are needed to assume the role of the destroyed odontoblasts. Growth factors, especially TGF-β, are thought to initiate the production of reparative dentin by fibroblasts and mesenchymal cells of the pulp. Reparative dentin is produced at an average of 1.5 μm/day, but can be increased to 3.5 μm/day. The resulting dentin contains irregularly shaped dentinal tubules which may not line up with existing dentinal tubules. This diminishes the ability for dental caries to progress within the dentinal tubules.

Diagnosis



(A) A small spot of decay visible on the surface of a tooth. **(B)** The radiograph reveals an extensive region of demineralization within the dentin (arrows). **(C)** A hole is discovered on the side of the tooth at the beginning of decay removal. **(D)** All decay removed.

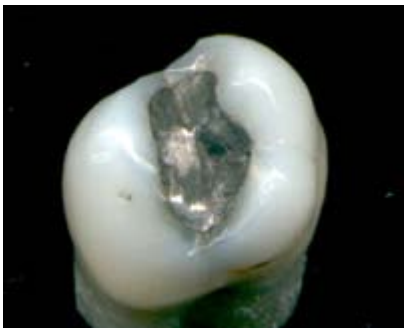
Primary diagnosis involves inspection of all visible tooth surfaces using a good light source, dental mirror and explorer. Dental radiographs (X-rays) may show dental caries before it is otherwise visible, particularly caries between the teeth. Large dental caries are often apparent to the naked eye, but smaller lesions can be difficult to identify. Visual and tactile inspection along with radiographs are employed frequently among dentists, particularly to diagnose pit and fissure caries. Early, uncavitated caries is often diagnosed by blowing air across the suspect surface, which removes moisture and changes the optical properties of the unmineralized enamel.

Some dental researchers have cautioned against the use of dental explorers to find caries. In cases where a small area of tooth has begun demineralizing but has not yet cavitated, the pressure from the dental explorer could cause a cavity. Since the carious process is reversible before a cavity is present, it may be possible to arrest the caries with fluoride and remineralize the tooth surface. When a cavity is present, a restoration will be needed to replace the lost tooth structure.

At times, pit and fissure caries may be difficult to detect. Bacteria can penetrate the enamel to reach dentin, but then the outer surface may remineralize, especially if fluoride is present. These caries, sometimes referred to as "hidden caries", will still be visible on

x-ray radiographs, but visual examination of the tooth would show the enamel intact or minimally perforated.

Treatment

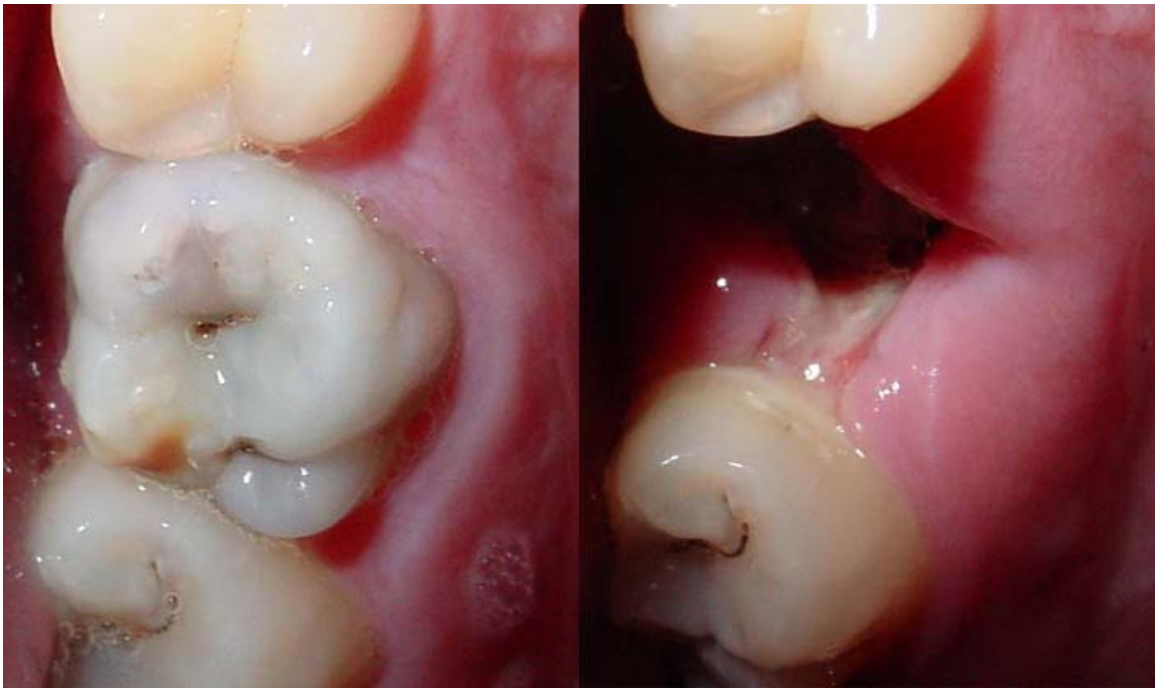


An amalgam used as a restorative material in a tooth.

Destroyed tooth structure does not fully regenerate, although remineralization of very small carious lesions may occur if dental hygiene is kept at optimal level. For the small lesions, topical fluoride is sometimes used to encourage remineralization. For larger lesions, the progression of dental caries can be stopped by treatment. The goal of treatment is to preserve tooth structures and prevent further destruction of the tooth.

Generally, early treatment is less painful and less expensive than treatment of extensive decay. Anesthetics—local, nitrous oxide ("laughing gas"), or other prescription medications—may be required in some cases to relieve pain during or following treatment or to relieve anxiety during treatment. A dental handpiece ("drill") is used to remove large portions of decayed material from a tooth. A spoon, a dental instrument used to remove decay carefully, is sometimes employed when the decay in dentin reaches near the pulp. Once the decay is removed, the missing tooth structure requires a dental restoration of some sort to return the tooth to functionality and aesthetic condition.

Restorative materials include dental amalgam, composite resin, porcelain, and gold. Composite resin and porcelain can be made to match the color of a patient's natural teeth and are thus used more frequently when aesthetics are a concern. Composite restorations are not as strong as dental amalgam and gold; some dentists consider the latter as the only advisable restoration for posterior areas where chewing forces are great. When the decay is too extensive, there may not be enough tooth structure remaining to allow a restorative material to be placed within the tooth. Thus, a crown may be needed. This restoration appears similar to a cap and is fitted over the remainder of the natural crown of the tooth. Crowns are often made of gold, porcelain, or porcelain fused to metal.



A tooth with extensive caries eventually requiring extraction.

In certain cases, endodontic therapy may be necessary for the restoration of a tooth. Endodontic therapy, also known as a "root canal", is recommended if the pulp in a tooth dies from infection by decay-causing bacteria or from trauma. During a root canal, the pulp of the tooth, including the nerve and vascular tissues, is removed along with decayed portions of the tooth. The canals are instrumented with endodontic files to clean and shape them, and they are then usually filled with a rubber-like material called gutta percha. The tooth is filled and a crown can be placed. Upon completion of a root canal, the tooth is now non-vital, as it is devoid of any living tissue.

An extraction can also serve as treatment for dental caries. The removal of the decayed tooth is performed if the tooth is too far destroyed from the decay process to effectively restore the tooth. Extractions are sometimes considered if the tooth lacks an opposing tooth or will probably cause further problems in the future, as may be the case for wisdom teeth. Extractions may also be preferred by patients unable or unwilling to undergo the expense or difficulties in restoring the tooth.

Medicinal plants in the treatment of dental caries

S. No	Botanical Name	Part used	Inhibition Organisms
1.	Acacia leucophloea	Bark	Streptococcus mutans
2.	Albizia lebbeck	Bark	Streptococcus mutans
3.	Abies canadensis	Whole plant	Streptococcus mutans
4.	Aristolochia	Whole plant	Streptococcus mutans

	cymbifera		
5.	Annona senegalensis	Whole plant	Streptococcus mutans
6.	Albizia julibrissin	Whole plant	Streptococcus mutans
7.	Allium sativum	Bulbs	Streptococcus mutans
8.	Anacyclus pyrethrum	Root	Streptococcus mutans
9.	Areca catechu	Nuts	Streptococcus mutans
10.	Breynia nivosus	Whole plant	Streptococcus mutans
14.	Citrus medica	Roots	Streptococcus mutans
15.	Coptidis rhizoma	Whole plant	Streptococcus mutans
16.	Caesalpinia martius	Fruits	Streptococcus mutans, Streptococcus oralis, Lactobacillus casei
17.	Cocos nucifera	Whole plant	Streptococcus mutans
18.	Caesalpinia pyramidalis	Whole plant	Streptococcus mutans
19.	Chelidonium majus	Whole plant	Streptococcus mutans
20.	Drosera peltata	Whole plant	Streptococcus mutans, Streptococcus sobrinus
21.	Embelia ribes	Fruit	Streptococcus mutans
22.	Erythrina variegata	Root	Streptococcus mutans, Streptococcus sanguis
23.	Euclea natalensis	Whole plant	Streptococcus mutans
24.	Fiscus microcarpa	Aerial part	Streptococcus mutans
25.	Gymnema Sylvester	Leaves,Roots	Streptococcus mutans
27.	Glycyrrhiza glabra	Root	Streptococcus mutans
28.	Hamamelis virginiana	Leaves	Preveotella spp., Actinomyces odontolitycus
29.	Harungana madagascariensis	Leaves	Actinomyces, Fusobacterium, Lactobacillus, Prevotella, Propioni bacterium, Streptococcus spp.
30.	Helichrysum italicum	Whole plant	Streptococcus mutans, Streptococcus sanguis, Streptococcus sobrinus
31.	Ginkgo biloba	Whole plant	Streptococcus mutans
32.	Juniperus virginiana	Whole plant	Streptococcus mutans
33.	Kaemperia pandurata	Dried rhizomes, root	Streptococcus mutans
34.	Legenaria sicerania	Leaves	Streptococcus mutans
35.	Mentha arvensis	Leaves	Streptococcus mutans
36.	Mikania lavigata	Aerial parts	Streptococcus mutans, Streptococcus sobrinus
37.	Mikania glomerata	Whole plant	Streptococcus cricetus
38.	Melissa officinalis	Whole plant	Streptococcus mutans, Streptococcus sanguis

39.	Magnolia grandiflora	Whole plant	Streptococcus mutans, Streptococcus sanguis
40.	Melissa officinalis	Whole plant	Streptococcus mutans, Streptococcus sanguis
41.	Magnolia grandiflora	Whole plant	Streptococcus mutans, Streptococcus sanguis
42.	Nicotiana tabacum	leaves	Streptococcus mutans
43.	Physalis angulata	Flower	Streptococcus mutans
44.	Pinus virginiana	Whole plant	Streptococcus mutans
45.	Pistacia lentiscus	mastic gum	Porphyromonas gingivalis
46.	Pistacia vera	Whole plant	oral Streptococci
47.	Piper cubeba	Whole plant	periodontal pathogens
48.	Polygonum cuspidatum	Root	Streptococcus mutans, Streptococcus sobrinus
49.	Rheedia brasiliensis	Fruit	Streptococcus mutans
50.	Rhus corriaria	Whole plant	Streptococcus mutans, Streptococcus sanguis
51.	Rhus corriaria	Whole plant	Streptococcus mutans, Streptococcus sanguis
52.	Rosmarinus officinalis	Whole plant	Streptococcus mutans
53.	Quercus infectoria	Gall	Streptococcus mutans
54.	Rhus corriaria	Whole plant	Streptococcus mutans, Streptococcus sanguis
55.	Syzygium cumini	Bark	Streptococcus mutans
56.	Sassafras albidum	Whole plant	Streptococcus mutans
57.	Solanum xathaocarpum	Whole plant	Streptococcus mutans
58.	Syzygium aromaticum	Dried flower	Staphylococcus aureus
59.	Thymus vulgaris	Whole plant	Streptococcus mutans, Streptococcus sanguis
60.	Tanacetum vulgare	Whole plant	Staphylococcus aureus
61.	Thuja plicata	Whole plant	Staphylococcus aureus
62.	Ziziphus joazeiro	Whole plant	Staphylococcus aureus

Prevention

Toothbrushes are commonly used to clean teeth.

Oral hygiene

Personal hygiene care consists of proper brushing and flossing daily. The purpose of oral hygiene is to minimize any etiologic agents of disease in the mouth. The primary focus of brushing and flossing is to remove and prevent the formation of plaque. Plaque consists mostly of bacteria. As the amount of bacterial plaque increases, the tooth is more vulnerable to dental caries when carbohydrates in the food are left on teeth after every meal or snack. A toothbrush can be used to remove plaque on accessible surfaces, but not between teeth or inside pits and fissures on chewing surfaces. When used correctly, dental floss removes plaque from areas which could otherwise develop proximal caries. Other adjunct hygiene aids include interdental brushes, water picks, and mouthwashes.

However oral hygiene is probably more effective at preventing gum disease than tooth decay. Food is forced inside pits and fissures under chewing pressure, leading to carbohydrate fueled acid demineralisation where the brush, fluoride toothpaste and saliva have no access to remove trapped food, neutralise acid or remineralise demineralised tooth like on other more accessible tooth surfaces food to be trapped. (Occlusal caries accounts for between 80 and 90 percent of caries in children (Weintraub, 2001). Chewing fibre like celery after eating, forces saliva inside trapped food to dilute any carbohydrate

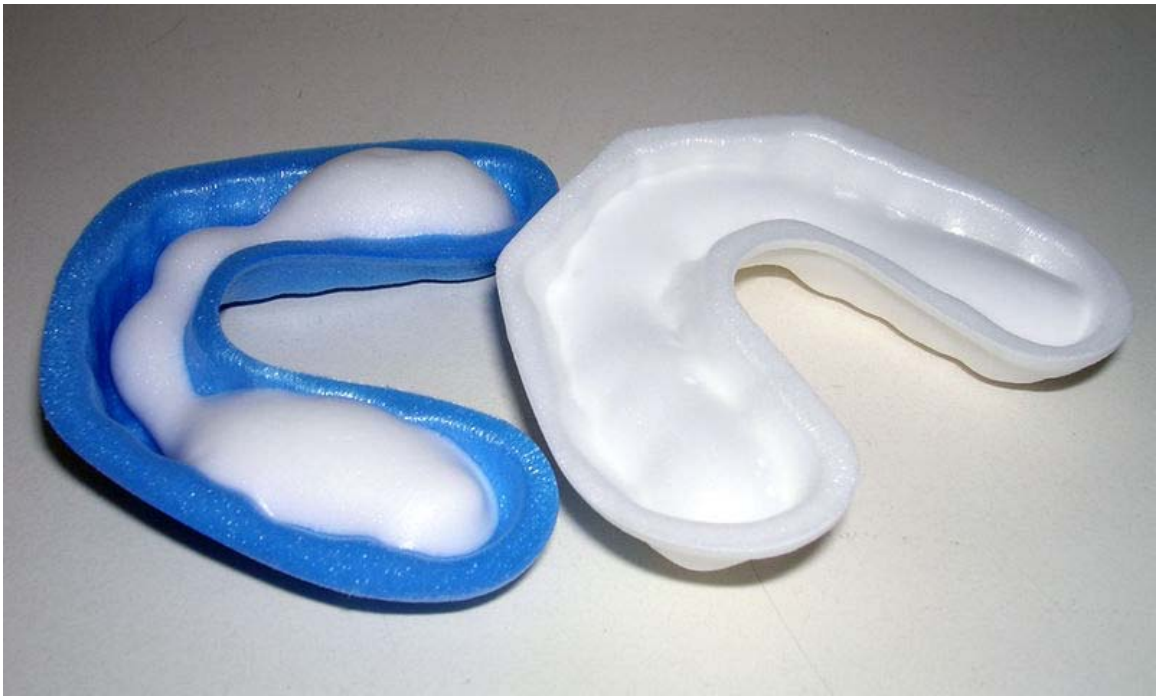
like sugar, neutralise acid and remineralise demineralised tooth. (The teeth at highest risk for carious lesions are the first and second permanent molars.)

Professional hygiene care consists of regular dental examinations and cleanings. Sometimes, complete plaque removal is difficult, and a dentist or dental hygienist may be needed. Along with oral hygiene, radio-graphs may be taken at dental visits to detect possible dental caries development in high risk areas of the mouth.

Dietary modification

For dental health, frequency of sugar intake is more important than the amount of sugar consumed. In the presence of sugar and other carbohydrates, bacteria in the mouth produce acids which can demineralize enamel, dentin, and cementum. The more frequently teeth are exposed to this environment, the more likely dental caries are to occur. Therefore, minimizing snacking is recommended, since snacking creates a continual supply of nutrition for acid-creating bacteria in the mouth. Also, chewy and sticky foods (such as dried fruit or candy) tend to adhere to teeth longer, and consequently are best eaten as part of a meal. Brushing the teeth after meals is recommended. For children, the American Dental Association and the European Academy of Paediatric Dentistry recommend limiting the frequency of consumption of drinks with sugar, and not giving baby bottles to infants during sleep. Mothers are also recommended to avoid sharing utensils and cups with their infants to prevent transferring bacteria from the mother's mouth.

It has been found that milk and certain kinds of cheese like Cheddar can help counter tooth decay if eaten soon after the consumption of foods potentially harmful to teeth. Also, chewing gum containing xylitol (a sugar alcohol) is widely used to protect teeth in some countries, being especially popular in the Finnish candy industry. Xylitol's effect on reducing plaque is probably due to bacteria's inability to utilize it like other sugars. Chewing and stimulation of flavour receptors on the tongue are also known to increase the production and release of saliva, which contains natural buffers to prevent the lowering of pH in the mouth to the point where enamel may become demineralised.



Common dentistry trays used to deliver fluoride.

Other preventive measures

The use of dental sealants is a means of prevention. A sealant is a thin plastic-like coating applied to the chewing surfaces of the molars. This coating prevents food being trapped inside pits and fissures in grooves under chewing pressure so resident plaque bacteria are deprived of carbohydrate that they change to acid demineralisation and thus prevents the formation of pit and fissure caries, the most common form of dental caries. Sealants are usually applied on the teeth of children, shortly after the molars erupt. Older people may also benefit from the use of tooth sealants, but their dental history and likelihood of caries formation are usually taken into consideration.

Calcium, as found in food such as milk and green vegetables, are often recommended to protect against dental caries. It has been demonstrated that calcium and fluoride supplements decrease the incidence of dental caries. Fluoride helps prevent decay of a tooth by binding to the hydroxyapatite crystals in enamel. The incorporated calcium makes enamel more resistant to demineralization and, thus, resistant to decay. Topical fluoride is also recommended to protect the surface of the teeth. This may include a fluoride toothpaste or mouthwash. Many dentists include application of topical fluoride solutions as part of routine visits.

Other products with little or less supportive scientific evidence for effectiveness for the purpose of remineralization include DCPD, ACP, calcium compounds, fluoride, and Enamelon.

Remineralization can also be performed professionally at the dentist.

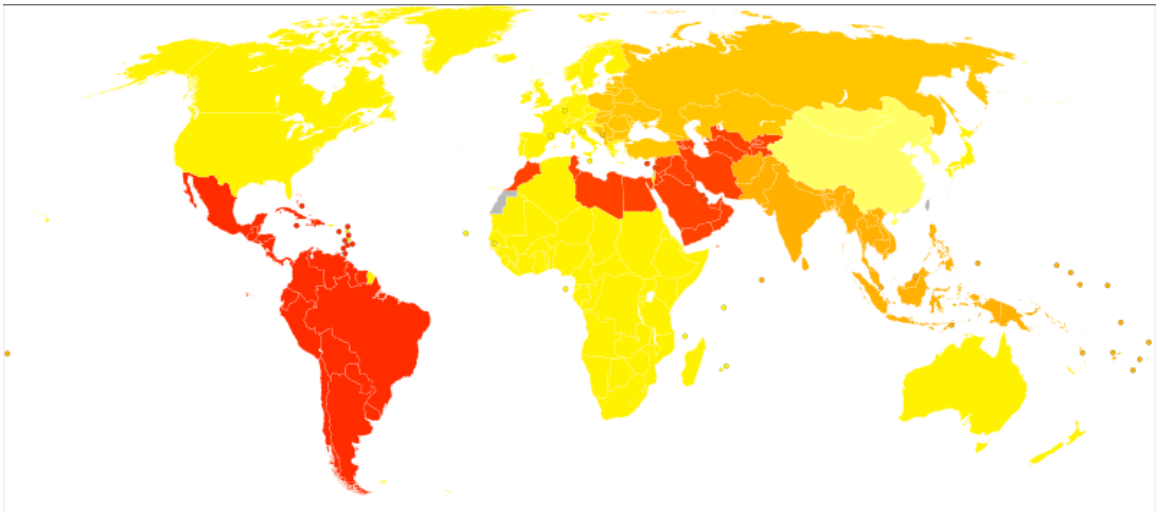
Furthermore, recent research shows that low intensity laser radiation of argon ion lasers may prevent the susceptibility for enamel caries and white spot lesions.

As bacteria are a major factor contributing to poor oral health, there is currently research to find a vaccine for dental caries. As of 2004, such a vaccine has been successfully tested on animals, and is in clinical trials for humans as of May 2006.

Chewing gum after eating promotes the flow of saliva which naturally reduces the acidic pH environment and promotes remineralization.

Xylitol lollies and gum also inhibit the growth of Streptococcus mutans.

Epidemiology



Disability-adjusted life year for dental caries per 100,000 inhabitants in 2004.
- no data
- less than 50
- 50-60
- 60-70
- 70-80
- 80-90
- 90-100
- 100-115
- 115-130
- 130-138
- 138-140
- 140-142
- more than 142

Worldwide, most children and an estimated ninety percent of adults have experienced caries, with the disease most prevalent in Asian and Latin American countries and least prevalent in African countries. In the United States, dental caries is the most common

chronic childhood disease, being at least five times more common than asthma. It is the primary pathological cause of tooth loss in children. Between twenty-nine and fifty-nine percent of adults over the age of fifty experience caries.

The number of cases has decreased in some developed countries, and this decline is usually attributed to increasingly better oral hygiene practices and preventive measures such as fluoride treatment. Nonetheless, countries that have experienced an overall decrease in cases of tooth decay continue to have a disparity in the distribution of the disease. Among children in the United States and Europe, twenty percent of the population endures sixty to eighty percent of cases of dental caries. A similarly skewed distribution of the disease is found throughout the world with some children having none or very few caries and others having a high number. Australia, Nepal, and Sweden have a low incidence of cases of dental caries among children, whereas cases are more numerous in Costa Rica and Slovakia.

The classic "DMF" (decay/missing/filled) index is one of the most common methods for assessing caries prevalence as well as dental treatment needs among populations. This index is based on in-field clinical examination of individuals by using a probe, mirror and cotton rolls. Because the DMF index is done without X-ray imaging, it underestimates real caries prevalence and treatment needs.

History

An image from 1300s (A.D.) England depicting a dentist extracting a tooth with forceps.

There is a long history of dental caries. Over a million years ago, hominids such as Australopithecus suffered from cavities. The largest increases in the prevalence of caries have been associated with dietary changes. Archaeological evidence shows that tooth decay is an ancient disease dating far into prehistory. Skulls dating from a million years ago through the neolithic period show signs of caries, excepting those from the Paleolithic and Mesolithic ages. The increase of caries during the neolithic period may be attributed to the increased consumption of plant foods containing carbohydrates. The beginning of rice cultivation in South Asia is also believed to have caused an increase in caries.

A Sumerian text from 5000 BC describes a "tooth worm" as the cause of caries. Evidence of this belief has also been found in India, Egypt, Japan, and China. Unearthed ancient skulls show evidence of primitive dental work. In Pakistan, teeth dating from around 5500 BC to 7000 BC show nearly perfect holes from primitive dental drills. The Ebers Papyrus, an Egyptian text from 1550 BC, mentions diseases of teeth. During the Sargonid dynasty of Assyria during 668 to 626 BC, writings from the king's physician specify the need to extract a tooth due to spreading inflammation. In the Roman Empire, wider consumption of cooked foods led to a small increase in caries prevalence. The Greco-Roman civilization, in addition to the Egyptian, had treatments for pain resulting from caries.

The rate of caries remained low through the Bronze Age and Iron Age, but sharply increased during the Middle Ages. Periodic increases in caries prevalence had been small in comparison to the 1000 AD increase, when sugar cane became more accessible to the Western world. Treatment consisted mainly of herbal remedies and charms, but sometimes also included bloodletting. The barber surgeons of the time provided services that included tooth extractions. Learning their training from apprenticeships, these health providers were quite successful in ending tooth pain and likely prevented systemic spread of infections in many cases. Among Roman Catholics, prayers to Saint Apollonia, the patroness of dentistry, were meant to heal pain derived from tooth infection.

There is also evidence of caries increase in North American Indians after contact with colonizing Europeans. Before colonization, North American Indians subsisted on hunter-gatherer diets, but afterwards there was a greater reliance on maize agriculture, which made these groups more susceptible to caries.

In the medieval Islamic world, Muslim physicians such as al-Gazzar and Avicenna (in *The Canon of Medicine*) provided the earliest known treatments for caries, though they also believed that it was caused by tooth worms as the ancients had. This was eventually proven false in 1200 by another Muslim dentist named Gaubari, who in his *Book of the Elite concerning the unmasking of mysteries and tearing of veils*, was the first to reject the idea of caries being caused by tooth worms, and he stated that tooth worms in fact do not even exist. The theory of the tooth worm was thus no longer accepted in the Islamic medical community from the 13th century onwards.

During the European Age of Enlightenment, the belief that a "tooth worm" caused caries was also no longer accepted in the European medical community. Pierre Fauchard, known as the father of modern dentistry, was one of the first to reject the idea that worms caused tooth decay and noted that sugar was detrimental to the teeth and gingiva. In 1850, another sharp increase in the prevalence of caries occurred and is believed to be a result of widespread diet changes. Prior to this time, cervical caries was the most frequent type of caries, but increased availability of sugar cane, refined flour, bread, and sweetened tea corresponded with a greater number of pit and fissure caries.

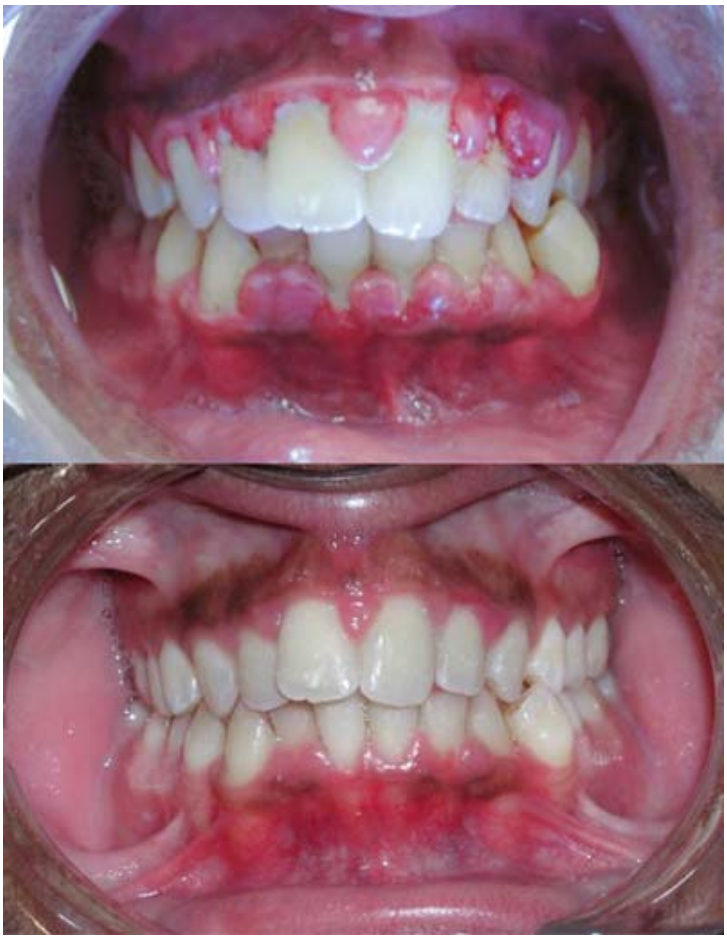
In the 1890s, W.D. Miller conducted a series of studies that led him to propose an explanation for dental caries that was influential for current theories. He found that

bacteria inhabited the mouth and that they produced acids which dissolved tooth structures when in the presence of fermentable carbohydrates. This explanation is known as the chemoparasitic caries theory. Miller's contribution, along with the research on plaque by G.V. Black and J.L. Williams, served as the foundation for the current explanation of the etiology of caries. Several of the specific strains of bacteria were identified in 1921 by Fernando E. Rodriguez Vargas.

Chapter 7

Gingivitis

Gingivitis



Severe gingivitis before (top) and after (bottom) a thorough mechanical debridement of the teeth and adjacent gum tissues.

ICD-10	K05.0-K05.1
ICD-9	523.0-523.1
DiseasesDB	34517
MedlinePlus	001056
MeSH	D005891

Gingivitis ("inflammation of the gum tissue") is a term used to describe non-destructive periodontal disease. The most common form of gingivitis is in response to bacterial biofilms (also called plaque) adherent to tooth surfaces, termed *plaque-induced gingivitis*, and is the most common form of periodontal disease. In the absence of treatment, gingivitis may progress to periodontitis, which is a destructive form of periodontal disease.

While in some sites or individuals, gingivitis never progresses to periodontitis, data indicates that periodontitis is always preceded by gingivitis.

Classification

As defined by the 1999 World Workshop in Clinical Periodontics, there are two primary categories of gingival diseases, each with numerous subgroups:

1. Dental plaque-induced gingival diseases
    1. Gingivitis associated with plaque only
    2. Gingival diseases modified by systemic factors
    3. Gingival diseases modified by medications
    4. Gingival diseases modified by malnutrition
2. Non-plaque-induced gingival lesions
    1. Gingival diseases of specific bacterial origin
    2. Gingival diseases of viral origin
    3. Gingival diseases of fungal origin
    4. Gingival diseases of genetic origin
    5. Gingival manifestations of systemic conditions
    6. Traumatic lesions
    7. Foreign body reactions
    8. Not otherwise specified

Signs and symptoms

The symptoms of gingivitis are somewhat non-specific and manifest in the gum tissue as the classic signs of inflammation:

- Swollen gums

- Bright red or purple gums
- Gums that are tender or painful to the touch
- Bleeding gums or bleeding after brushing

Additionally, the stippling that normally exists on the gum tissue of some individuals will often disappear and the gums may appear shiny when the gum tissue becomes swollen and stretched over the inflamed underlying connective tissue. The accumulation may also emit an unpleasant odor. When the gingiva are swollen, the epithelial lining of the gingival crevice becomes ulcerated and the gums will bleed more easily with even gentle brushing, and especially when flossing.

Cause

Because plaque-induced gingivitis is by far the most common form of gingival diseases, the following sections will deal primarily with this condition.

The *etiology*, or cause, of plaque-induced gingivitis is bacterial plaque, which acts to initiate the body's host response. This, in turn, can lead to destruction of the gingival tissues, which may progress to destruction of the periodontal attachment apparatus. The plaque accumulates in the small gaps between teeth, in the gingival grooves and in areas known as *plaque traps*: locations that serve to accumulate and maintain plaque. Examples of plaque traps include bulky and overhanging restorative margins, claps of removable partial dentures and calculus (tartar) that forms on teeth. Although these accumulations may be tiny, the bacteria in them produce chemicals, such as degrative enzymes, and toxins, such as lipopolysaccharide (LPS, otherwise known as endotoxin) or lipoteichoic acid (LTA), that promote an inflammatory response in the gum tissue. This inflammation can cause an enlargement of the gingiva and subsequent *pseudopocket* formation.

Diagnosis

It is recommended that a dental hygienist or dentist be seen after the signs of gingivitis appear. A dental hygienist or dentist will check for the symptoms of gingivitis, and may also examine the amount of plaque in the oral cavity. A dental hygienist or dentist will also look for signs of periodontitis using X-rays or periodontal probing as well as other methods.

If gingivitis is not responsive to treatment, referral to a periodontist (a specialist in diseases of the gingiva and bone around teeth and dental implants) for further treatment may be necessary.

Prevention

OTC anti-gingivitis mouthwash containing chlorhexidine from Mexico.

Gingivitis can be prevented through regular oral hygiene that includes daily brushing and flossing. Interdental brushes are also useful in cleaning the teeth from plaque. Hydrogen peroxide, saline, alcohol or chlorhexidine mouth washes may also be employed. In a recent clinical study, the beneficial effect of hydrogen peroxide on gingivitis has been highlighted. Rigorous plaque control programs along with periodontal scaling and curettage also have proved to be helpful, although according to the American Dental Association, periodontal scaling and root planing are considered as a treatment to periodontal disease, not as a preventive treatment for periodontal disease. In a 1997 review of effectiveness data the U.S. Food and Drug Administration (FDA) found clear evidence which showed that toothpaste containing triclosan was effective in preventing gingivitis.

In many countries, such as the United States, mouthwashes containing chlorhexidine are available only by prescription.

Researchers analyzed government data on calcium consumption and periodontal disease indicators in nearly 13,000 U.S. adults. They found that men and women who had calcium intakes of fewer than 500 milligrams, or about half the recommended dietary allowance, were almost twice as likely to have gum disease, as measured by the loss of attachment of the gums from the teeth. The association was particularly evident for people in their 20s and 30s.

Preventing gum disease may also benefit a healthy heart. According to physicians with The Institute for Good Medicine at the Pennsylvania Medical Society, good oral health can reduce risk of cardiac events. Poor oral health can lead to infections that can travel within the bloodstream.

Treatment

The focus of treatment for gingivitis is removal of the etiologic (causative) agent, plaque. Therapy is aimed at the reduction of oral bacteria, and may take the form of regular periodic visits to a dental professional together with adequate oral hygiene home care. Thus, several of the methods used in the prevention of gingivitis can also be used for the treatment of manifest gingivitis, such as scaling, root planing, curettage, mouth washes containing chlorhexidine or hydrogen peroxide, and flossing. Interdental brushes also help remove any causative agents.

Recent scientific studies have also shown the beneficial effects of mouthwashes with essential oils.

Complications

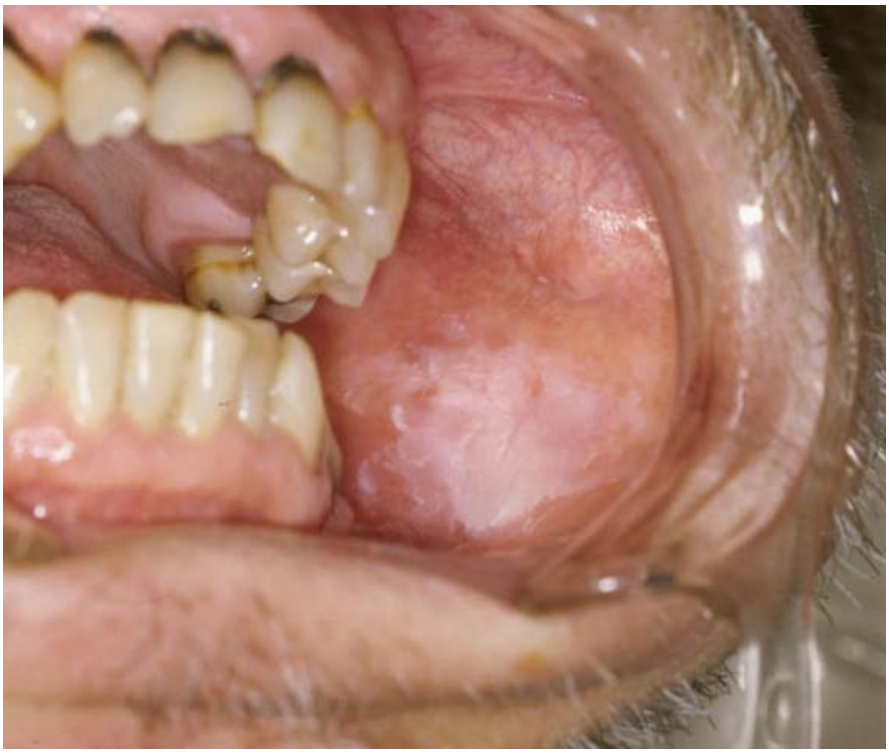
- Tooth loss
- Recurrence of gingivitis
- Periodontitis
- Infection or abscess of the gingiva or the jaw bones
- Trench mouth (bacterial infection and ulceration of the gums)

Chapter 8

Leukoplakia and Mucous Cyst of the Oral Mucosa

Leukoplakia

Leukoplakia



The white lesion is an example of leukoplakia.

ICD-10	K13.2, N48.0, N88.0, N89.4, N90.4
ICD-9	528.6, 530.83, 607.0, 622.2, 623.1, 624.0
DiseasesDB	7438
MedlinePlus	001046
MeSH	D007971

Leukoplakia is a clinical term used to describe patches of keratosis . It is visible as adherent white patches on the mucous membranes of the oral cavity, including the tongue, but also other areas of the gastro-intestinal tract, urinary tract and the genitals. The clinical appearance is highly variable. Leukoplakia is not a specific disease entity, but is diagnosis of exclusion. It must be distinguished from diseases that may cause similar white lesions, such as candidiasis or lichen planus.

It is sometimes described as precancerous.. It is also associated with smoking.

Tobacco, either smoked or chewed, is considered to be the main culprit in its development. (1998-2010 Mayo Foundation for Medical Education and Research (MFMER).

The term "candidal leukoplakia" is sometimes used to describe certain types of oral candidiasis.

Although the term "leukoplakia" often applies to conditions of the mouth, it can also be used to describe conditions of the genitals and urinary tract.

Incidence and prevalence

Leukoplakic lesions are found in approximately 3% of the world's population. Like erythroplakia, leukoplakia is usually found in adults between 40 and 70 years of age, with a 2:1 male predominance.

Causes

Leukoplakia is primarily caused by the use of tobacco. Other possible etiological agents implicated are HPV's, Candida albicans and possibly alcohol. Simultaneously serum levels of patients with leukoplakia were found to be low in Vit A,B-12,C & folic acid,in a study conducted in India. Most result from chronic irritation of mucous membranes by carcinogens. Bloodroot, otherwise known as sanguinaria, is also believed to be associated with leukoplakia.

5% to 25% of leukoplakias are premalignant lesions; therefore, all leukoplakias should be treated as premalignant lesions by dentists and physicians - they require histologic

evaluation or biopsy. Hairy leukoplakia, which is associated with HIV infection and other diseases of severe immune deficiency can go on to develop lymphoma when associated with HIV.

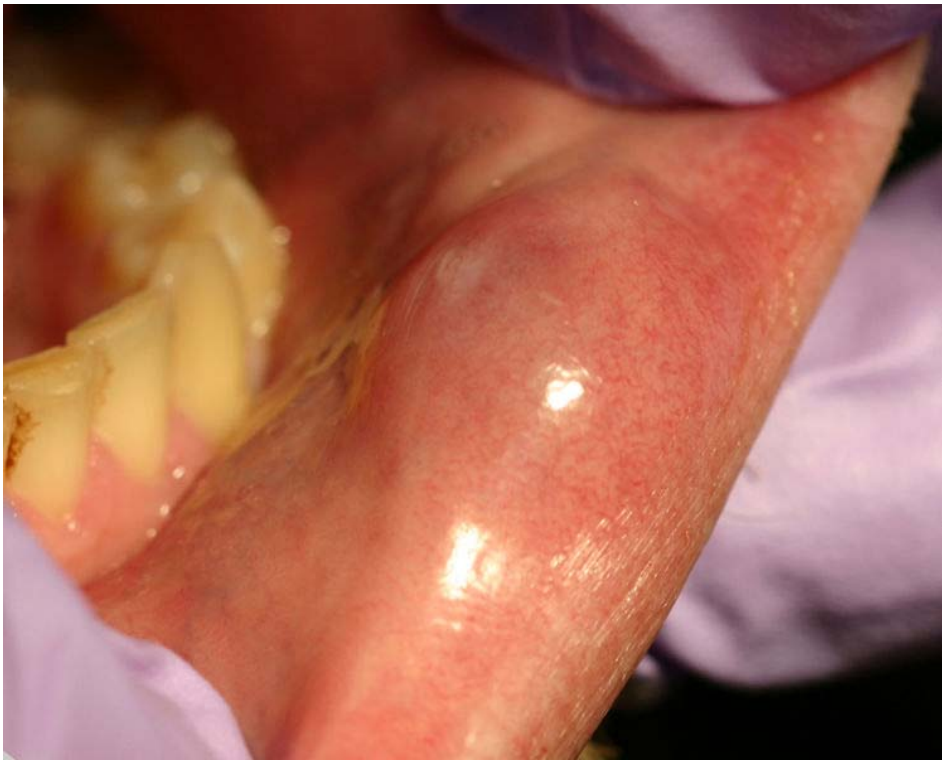
Treatment

The treatment of leukoplakia mainly involves avoidance of predisposing factors — tobacco cessation, smoking, quitting betel chewing, abstinence from alcohol — and avoidance of chronic irritants, e.g., the sharp edges of teeth. A biopsy should be done, and the lesion surgically excised if pre-cancerous changes or cancer is detected.

Taking beta-carotene orally seems to induce remission in patients with oral leukoplakia. Further research is needed to confirm these results.

Mucous cyst of the oral mucosa

Mucous cyst of the oral mucosa



A mucocele on the lower lip.

ICD-10	K11.6
ICD-9	527.6
DiseasesDB	30713
eMedicine	derm/274
MeSH	D009078

A "mucous cyst of the oral mucosa" (also known as a "mucocele") is a clinical term that refers to two related phenomena: **mucus extravasation phenomenon**, and **mucus retention cyst**. The former is a swelling of connective tissue consisting of collected mucin due to a ruptured salivary gland duct usually caused by local trauma, in the case of mucus extravasation phenomenon, and an obstructed salivary duct in the case of a mucus retention cyst. The mucocele has a bluish translucent color, and is more commonly found in children and young adults.

It can be considered a polyp or a cyst.

Locations

The most common location to find a mucocele is the surface of the lower lip. It can also be found on the inner side of the cheek (known as the buccal mucosa), on the anterior ventral tongue, and the floor of the mouth. When found on the floor of the mouth, the mucocele is referred to as a ranula. They are rarely found on the upper lip. As their name suggests they are basically mucus lined cysts and they can also occur in the Paranasal sinuses most commonly the frontal sinuses, the frontoethomidal region and also in the maxillary sinus. Sphenoid sinus involvement is extremely rare. When the lumen of the vermiform appendix gets blocked due to any factor, again a mucocele can form.

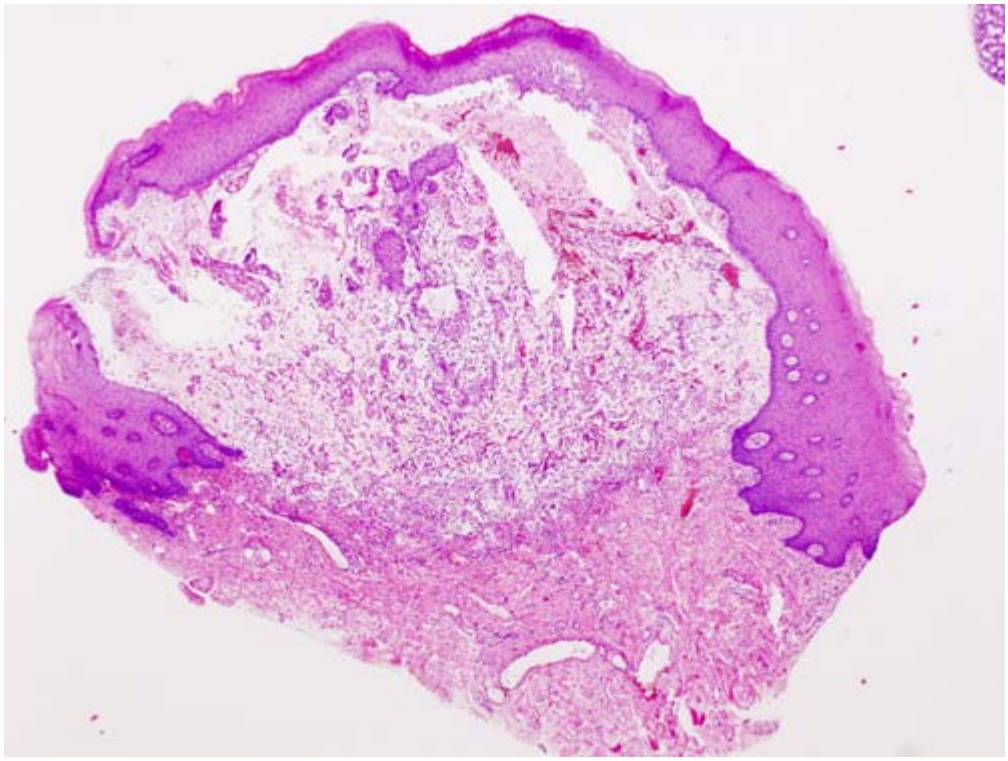
Characteristics

The size of oral mucoceles vary from 1 mm to several centimeters and they usually are slightly transparent with a blue tinge. On palpation, mucoceles may appear fluctuant but can also be firm. Their duration lasts from days to years, and may have recurrent swelling with occasional rupturing of its contents.

Variations

A variant of a mucocele is found on the palate, retromolar pad, and posterior buccal mucosa. Known as a "superficial mucocele", this type presents as single or multiple vesicles and bursts into an ulcer. Despite healing after a few days, superficial mucoceles recur often in the same location.

Histology



Histopathologic image of extravasation type mucocele of the lower lip. H & E stain.

Microscopically, mucoceles appears as granulation tissue surrounding mucin. Since inflammation occurs concurrently, neutrophils and foamy histiocytes usually are present.

Treatment

Some mucoceles spontaneously resolve on their own after a short time. Others are chronic and require surgical removal. Recurrence may occur, and thus the adjacent salivary gland is excised as a preventive measure.

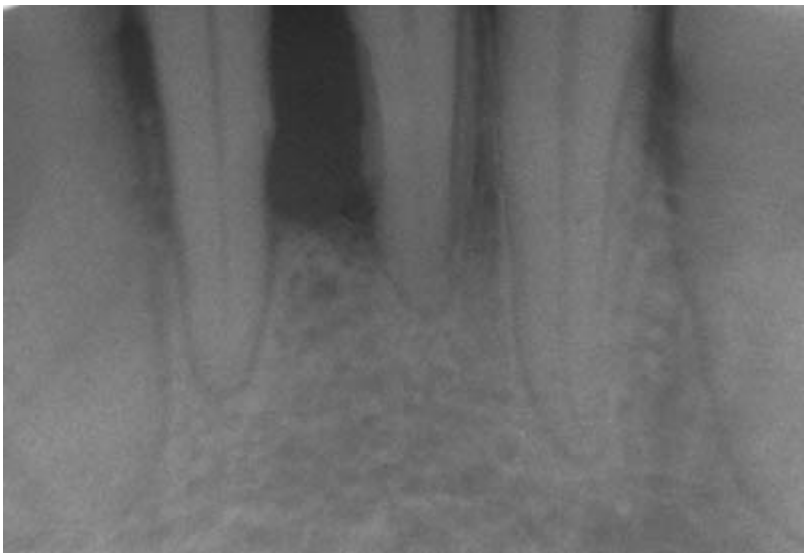
Several types of procedures are available for the surgical removal of mucoceles. These include laser and minimally-invasive techniques which means recovery times are reduced drastically.

A non-surgical option that may be effective for a small or newly identified mucocle is to rinse the mouth thoroughly with salt water (one tablespoon of salt per cup) four to six times a day for a few days. This may draw out the fluid trapped underneath the skin without further damaging the surrounding tissue. If the mucocele persists, individuals should see a doctor to discuss further treatment.

Chapter 9

Periodontitis

Periodontal disease



This radiograph shows significant bone loss between the two roots of a tooth (black region). The spongy bone has receded due to infection under tooth, reducing the bony support for the tooth.

ICD-10	K05.4
DiseasesDB	29362
MedlinePlus	001059
MeSH	D010518

Periodontitis is a set of inflammatory diseases affecting the periodontium, i.e., the tissues that surround and support the teeth. Periodontitis involves progressive loss of the alveolar bone around the teeth, and if left untreated, can lead to the loosening and

subsequent loss of teeth. Periodontitis is caused by microorganisms that adhere to and grow on the tooth's surfaces, along with an overly aggressive immune response against these microorganisms. A diagnosis of periodontitis is established by inspecting the soft gum tissues around the teeth with a probe (i.e. a *clinical exam*) and by evaluating the patient's x-ray films (i.e. a *radiographic exam*), to determine the amount of bone loss around the teeth. Specialists in the treatment of periodontitis are periodontists; their field is known as "periodontology" or "periodontics".

The word "periodontitis" comes from *peri* ("around"), *odont* ("tooth") and -*itis* ("inflammation").

Classification

The 1999 classification system for periodontal diseases and conditions listed seven major categories of periodontal diseases, of which the last six are termed *destructive* periodontal disease because they are essentially irreversible. The seven categories are as follows:

1. Gingivitis
2. Chronic periodontitis
3. Aggressive periodontitis
4. Periodontitis as a manifestation of systemic disease
5. Necrotizing ulcerative gingivitis/periodontitis
6. Abscesses of the periodontium
7. Combined periodontic-endodontic lesions

Moreover, terminology expressing both the extent and severity of periodontal diseases are appended to the terms above to denote the specific diagnosis of a particular patient or group of patients.

Extent

The *extent* of disease refers to the proportion of the dentition affected by the disease in terms of percentage of sites. Sites are defined as the positions at which probing measurements are taken around each tooth and, generally, six probing sites around each tooth are recorded, as follows:

1. mesiobuccal
2. mid-buccal
3. distobuccal
4. mesiolingual
5. mid-lingual
6. distolingual

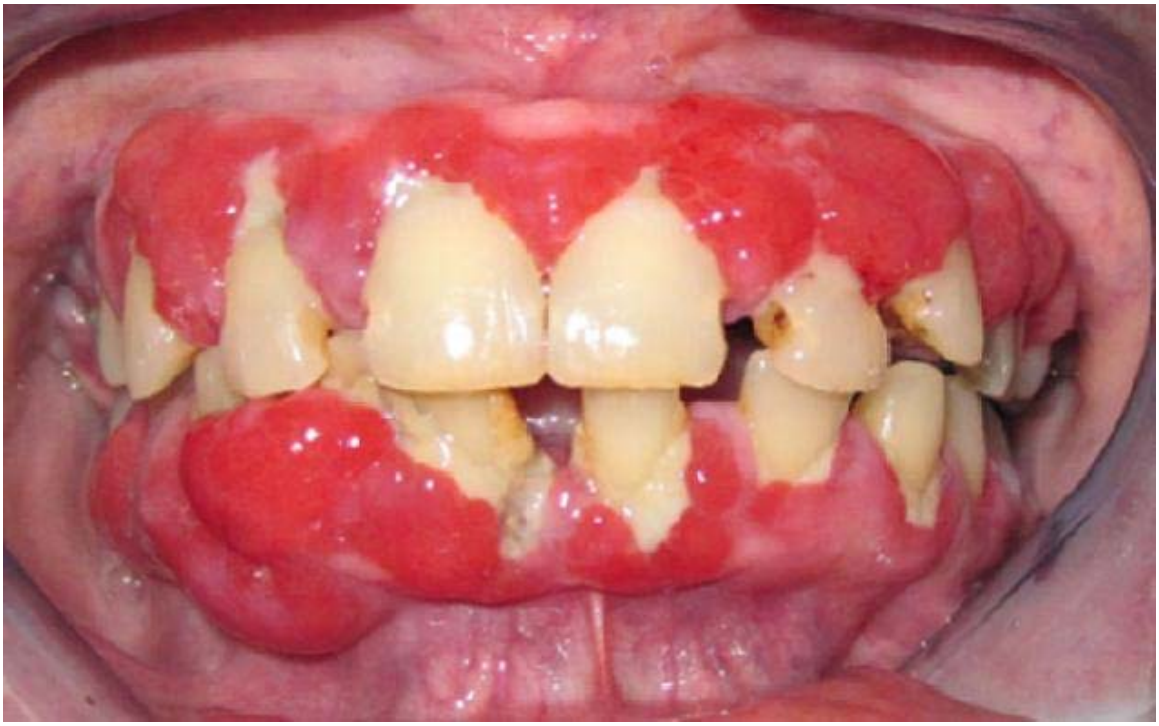
If up to 30% of sites in the mouth are affected, the manifestation is classification as *localized*; for more than 30%, the term *generalized* is used.

Severity

The *severity* of disease refers to the amount of periodontal ligament fibers that have been lost, termed *clinical attachment loss*. According to the American Academy of Periodontology, the classification of severity is as follows:

- *Mild*: 1–2 mm of attachment loss
- *Moderate*: 3–4 mm of attachment loss
- *Severe*: ≥ 5 mm of attachment loss

Signs and symptoms



Periodontitis manifesting as painful, red, swollen gums, with abundant plaque.

In the early stages, periodontitis has very few symptoms and in many individuals the disease has progressed significantly before they seek treatment. Symptoms may include the following:

- Redness or bleeding of gums while brushing teeth, using dental floss or biting into hard food (e.g. apples) (though this may occur even in gingivitis, where there is no attachment loss)
- Gum swelling that recurs
- Halitosis, or bad breath, and a persistent metallic taste in the mouth
- Gingival recession, resulting in apparent lengthening of teeth. (This may also be caused by heavy handed brushing or with a stiff tooth brush.)

- Deep pockets between the teeth and the gums (pockets are sites where the attachment has been gradually destroyed by collagen-destroying enzymes, known as *collagenases*)
- Loose teeth, in the later stages (though this may occur for other reasons as well)

Patients should realize that the gingival inflammation and bone destruction are largely painless. Hence, people may wrongly assume that painless bleeding after teeth cleaning is insignificant, although this may be a symptom of progressing periodontitis in that patient.

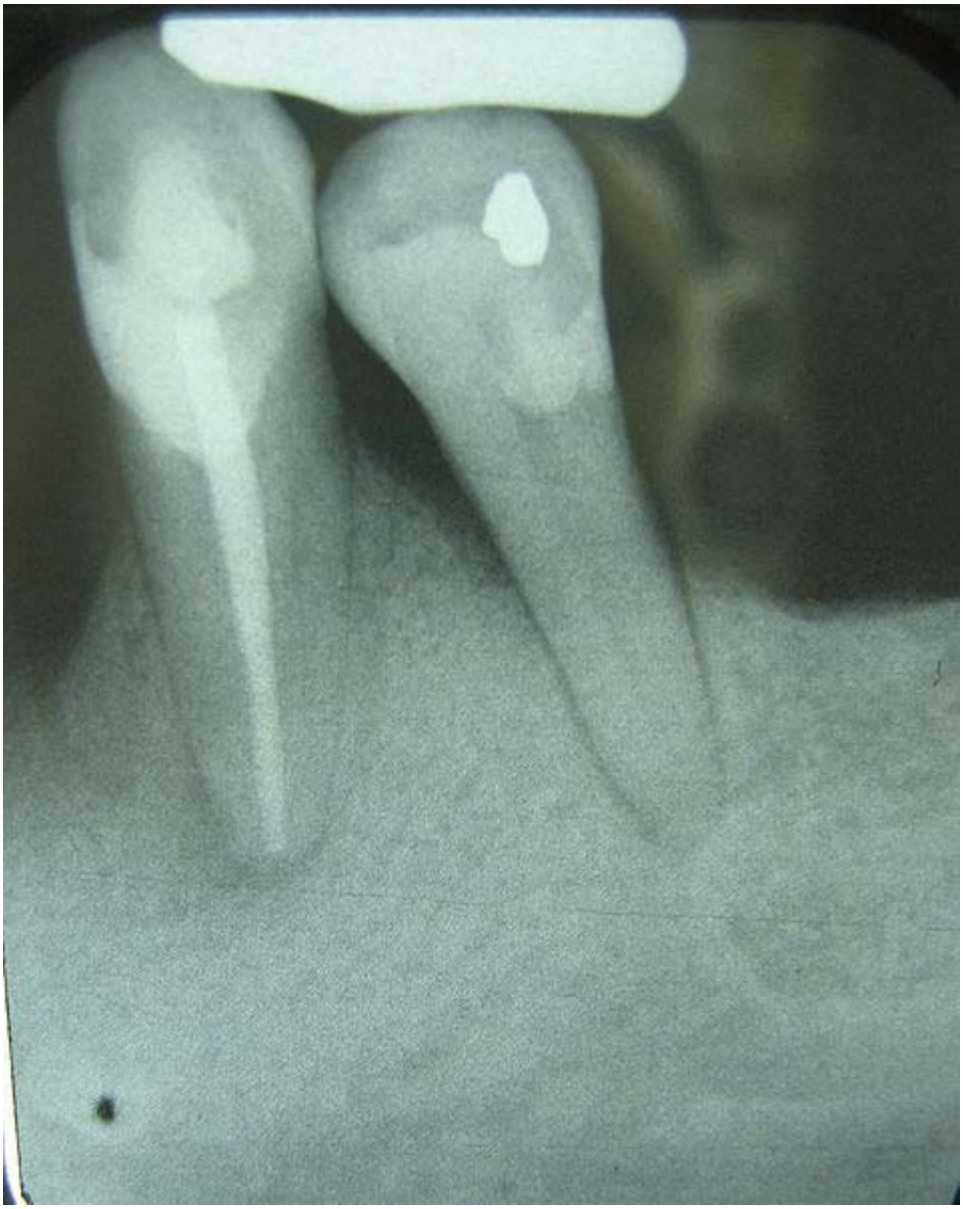
Effects outside the mouth

Periodontitis has been linked to increased inflammation in the body such as indicated by raised levels of C-reactive protein and Interleukin-6. It is through this linked to increased risk of stroke, myocardial infarction, and atherosclerosis. It also linked in those over 60 years of age to impairments in delayed memory and calculation abilities.

Causes

Periodontitis is an inflammation of the periodontium, i.e., the tissues that support the teeth. The periodontium consists of four tissues:

- gingiva, or gum tissue;
- cementum, or outer layer of the roots of teeth;
- alveolar bone, or the bony sockets into which the teeth are anchored;
- periodontal ligaments (PDLs), which are the connective tissue fibers that run between the cementum and the alveolar bone.

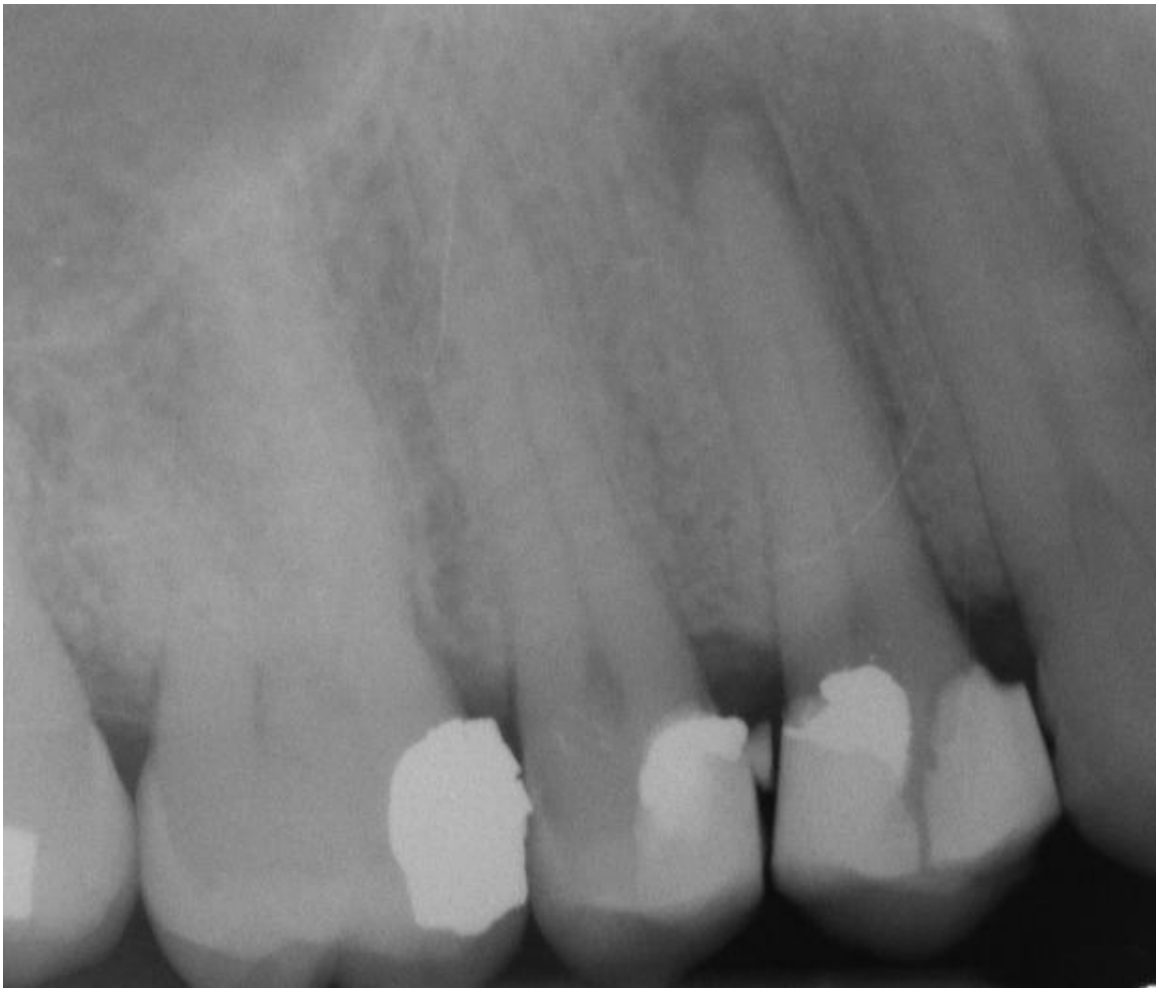


This X-ray film displays two lone-standing mandibular teeth, the lower left first premolar and canine, exhibiting severe bone loss of 30–50%. Widening of the periodontal ligament surrounding the premolar is due to secondary occlusal trauma.

The primary etiology (cause) of gingivitis is poor oral hygiene which leads to the accumulation of a mycotic and bacterial matrix at the gum line, called dental plaque. Other contributors are poor nutrition and underlying medical issues such as diabetes. New finger nick tests have been approved by the Food and Drug Administration in the US, and are being used in dental offices to identify and screen patients for possible contributory causes of gum disease such as diabetes.

In some people, gingivitis progresses to periodontitis - with the destruction of the gingival fibers, the gum tissues separate from the tooth and deepened sulcus, called a

periodontal pocket. Subgingival microorganism (those that exist under the gum line) colonize the periodontal pockets and cause further inflammation in the gum tissues and progressive bone loss. Examples of secondary etiology are those things that, by definition, cause microbic plaque accumulation, such as restoration overhangs and root proximity.



The excess restorative material that exceeds the natural contours of restored teeth, such as these, are termed "overhangs", and serve to trap microbic plaque, potentially leading to localized periodontitis.

Smoking is another factor that increases the occurrence of periodontitis, directly or indirectly, and may interfere with or adversely affect its treatment.

If left undisturbed, microbic plaque calcifies to form calculus, which is commonly called tartar. Calculus above and below the gum line must be removed completely by the dental hygienist or dentist to treat gingivitis and periodontitis. Although the primary cause of both gingivitis and periodontitis is the microbic plaque that adheres to the tooth surface, there are many other modifying factors. A very strong risk factor is one's genetic susceptibility. Several conditions and diseases, including Down syndrome, diabetes, and

other diseases that affect one's resistance to infection also increase susceptibility to periodontitis.

Another factor that makes periodontitis a difficult disease to study is that human host response can also affect the alveolar bone resorption. Host response to the bacterial-mycotic insult is mainly determined by genetics; however, immune development may play some role in susceptibility.

According to some researches periodontitis may be associated with higher stress.

Prevention

Daily oral hygiene measures to prevent periodontal disease include:

- Brushing properly on a regular basis (at least twice daily), with the patient attempting to direct the toothbrush bristles underneath the gum-line, to help disrupt the bacterial-mycotic growth and formation of subgingival plaque.
- Flossing daily and using interdental brushes (if there is a sufficiently large space between teeth), as well as cleaning behind the last tooth, the third molar, in each quarter.
- Using an antiseptic mouthwash. Chlorhexidine gluconate based mouthwash in combination with careful oral hygiene may cure gingivitis, although they cannot reverse any attachment loss due to periodontitis.
- Using a 'soft' tooth brush to prevent damage to tooth enamel and sensitive gums.
- Using periodontal trays to maintain dentist-prescribed medications at the source of the disease. The use of trays allows the medication to stay in place long enough to penetrate the biofilms where the microorganism are found.
- Regular dental check-ups and professional teeth cleaning as required. Dental check-ups serve to monitor the person's oral hygiene methods and levels of attachment around teeth, identify any early signs of periodontitis, and monitor response to treatment.

Typically dental hygienists (or dentists) use special instruments to clean (debride) teeth below the gumline and disrupt any plaque growing below the gumline. This is a standard treatment to prevent any further progress of established periodontitis. Studies show that after such a professional cleaning (periodontal debridement), microbic plaque tend to grow back to pre-cleaning levels after about 3–4 months. Hence, in theory, cleanings every 3–4 months might be expected to also prevent the initial onset of periodontitis. However, analysis of published research has reported little evidence either to support this or the intervals at which this should occur. Instead, it is advocated that the interval between dental check-ups should be determined specifically for each patient between every 3 to 24 months.

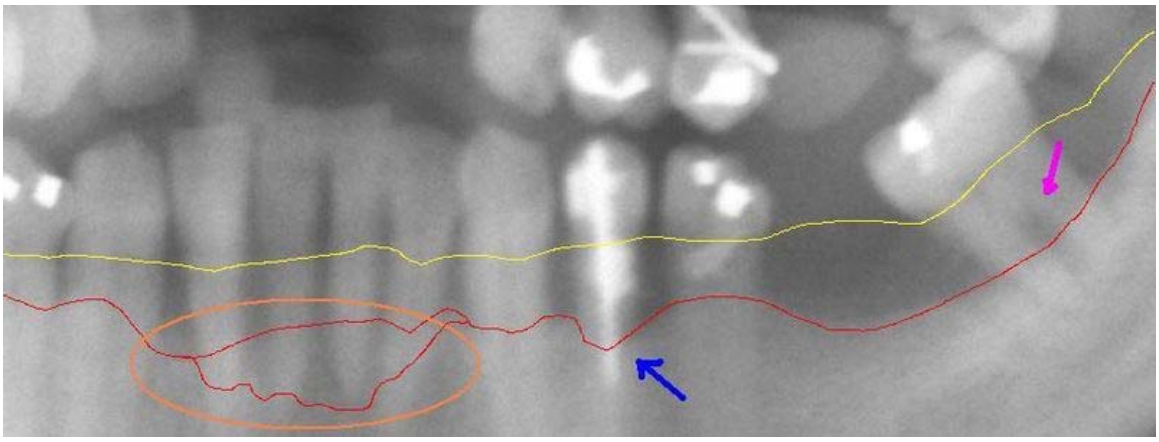
Nonetheless, the continued stabilization of a patient's periodontal state depends largely, if not primarily, on the patient's oral hygiene at home as well as on the go. Without daily

oral hygiene, periodontal disease will not be overcome, especially if the patient has a history of extensive periodontal disease.

Periodontal disease and tooth loss are associated with an increased risk of cancer.

A contributing cause may be low selenium in the diet: "Results showed that selenium has the strongest association with gum disease, with low levels increasing the risk by 13 fold."

Management



This section from a panoramic X-ray film depicts the teeth of the lower left quadrant, exhibiting generalized severe bone loss of 30–80%. The **red line** depicts the existing bone level, whereas the **yellow line** depicts where the gingiva was originally (1–2 mm above the bone), prior to the patient developing periodontal disease. The **pink arrow**, on the right, points to a *furcation involvement*, or the loss of enough bone to reveal the location at which the individual roots of a molar begin to branch from the single root trunk; this is a sign of advanced periodontal disease. The **blue arrow**, in the middle, shows up to 80% bone loss on tooth #21, and clinically, this tooth exhibited gross mobility. Finally, the **peach oval**, to the left, highlights the aggressive nature with which periodontal disease generally affects mandibular incisors. Because their roots are generally situated very close to each other, with minimal interproximal bone, and because of their location in the mouth, where plaque and calculus accumulation is greatest because of the pooling of saliva, mandibular anteriors suffer excessively. The **split in the red line** depicts varying densities of bone that contribute to a vague region of definitive bone height.

The cornerstone of successful periodontal treatment starts with establishing excellent oral hygiene. This includes twice daily brushing with daily flossing. Also the use of an interdental brush (called a Proxi-brush) is helpful if space between the teeth allows. For smaller spaces a product called "Soft Picks" are an excellent manual cleaning device. Persons with dexterity problems such as arthritis may find oral hygiene to be difficult and may require more frequent professional care and/or the use of a powered tooth brush. Persons with periodontitis must realize that it is a chronic inflammatory disease and a

lifelong regimen of excellent hygiene and professional maintenance care with a dentist/hygienist or periodontist is required to maintain affected teeth.

Initial therapy

Removal of microbic plaque and calculus is necessary to establish periodontal health. The first step in the treatment of periodontitis involves non-surgical cleaning below the gumline with a procedure called scaling and debridement. In the past, Root Planing was used (removal of cemental layer as well as calculus). This procedure involves use of specialized curettes to mechanically remove plaque and calculus from below the gumline, and may require multiple visits and local anesthesia to adequately complete. In addition to initial scaling and root planing, it may also be necessary to adjust the occlusion (bite) to prevent excessive force on teeth that have reduced bone support. Also it may be necessary to complete any other dental needs such as replacement of rough, plaque retentive restorations, closure of open contacts between teeth, and any other requirements diagnosed at the initial evaluation.

Reevaluation

Multiple clinical studies have shown that non-surgical scaling and root planing is usually successful if the periodontal pockets are shallower than 4–5 mm. It is necessary for the dentist or hygienist to perform a reevaluation 4–6 weeks after the initial scaling and root planing, to determine if the treatment was successful in reducing pocket depths and eliminating inflammation. Pocket depths which remain after initial therapy of greater than 5-6mm with bleeding upon probing are indicate continued active disease and will very likely show further bone loss over time. This is especially true in molar tooth sites where furcations (areas between the roots) have been exposed.

Surgery

If non-surgical therapy is found to have been unsuccessful in managing signs of disease activity, periodontal surgery may be needed to stop progressive bone loss and regenerate lost bone where possible. There are many surgical approaches used in treatment of advanced periodontitis, including open flap debridement, osseous surgery, as well as guided tissue regeneration and bone grafting. The goal of periodontal surgery is access for definitive calculus removal and surgical management of bony irregularities which have resulted from the disease process to reduce pockets as much as possible. Long-term studies have shown that in moderate to advanced periodontitis, surgically treated cases often have less further breakdown over time and when coupled with a regular post-treatment maintenance regimen are successful in nearly halting tooth loss in nearly 85% of patients.

Maintenance

Once successful periodontal treatment has been completed, with or without surgery, an ongoing regimen of "periodontal maintenance" is required. This involves regular

checkups and detailed cleanings every three months to prevent re-population of periodontitis-causing microorganism, and to closely monitor affected teeth so that early treatment can be rendered if disease recurs. Usually periodontal disease exist due to poor plaque control, therefore if the brushing techniques are not modified, a periodontal recurrence is probable.

Alternative treatments

Periodontitis has an inescapable relationship with subgingival calculus (tartar). The first step in any procedure is to eliminate calculus under the gum line, as it houses destructive anaerobic microorganisms that consume bone, gum and cementum (connective tissue) for food.

Most alternative “at-home” gum disease treatments involve injecting anti-microbial solutions, such as hydrogen peroxide, into periodontal pockets via slender applicators or oral irrigators. This process disrupts anaerobic microorganism colonies and is effective at reducing infections and inflammation when used daily. A number of potions and elixirs that are functionally equivalent to hydrogen peroxide are commercially available but at substantially higher cost. However, such treatments do not address calculus formations, and so are short-lived, as anaerobic microorganism colonies quickly regenerate in and around calculus.

In a new field of study, calculus formations are addressed on a more fundamental level. At the heart of the formation of subgingival calculus, growing plaque formations starve out the lowest members of the community, which calcify into calcium phosphate salts of the same shape and size of the original, organic bacilli. Calcium phosphate salts (unlike calcium phosphate; the primary component in teeth) are ionic and adhere to tooth surfaces via electrostatic attraction. Smaller, free-floating calcium phosphate salt particles are equally attracted to the same areas, as are additional calcified microorganism, growing calculus formations as unorganized, yet strong, “brick and mortar” matrices. The microscopic voids in calculus formations house new anaerobic microorganism, as does the top “diseased layer”.

Because the root cause of subgingival calculus development is ionic attraction, it was hypothesized that the introduction of oppositely charged particles around the formations may chelate calcium phosphate salt components away from the matrix, thus reducing the size of subgingival calculus formations. To accomplish this, a sequestering agent solution consisting partly of sodium tripolyphosphate (STPP) and sodium fluoride (charge -1) was tested on a patient with burnished and new subgingival calculus at a depth of 6 mm. The patient delivered the solution using an oral irrigator, once a day, for 60 days. The results were the successful elimination of all calculus formations studied. This test was conducted using a subgingival endoscopic camera (perioscope) by an independent periodontist.

The promise of this new, alternative treatment is to keep subgingival calculus at bay, in concert with traditional periodontal treatments. In this way, periodontitis may be

controlled by the patient, and complete restoration of dental health can be a collaborative effort between the patient and the dental professional.

Additionally, Periodontitis can be treated in a noninvasive manner by means of Periostat (subantimicrobial dose of doxycycline), an FDA-approved, orally-administered drug that has been shown to reduce bone loss. Its mechanism of action in part involves inhibition of Matrix metalloproteinases (such as collagenase), which degrade the extracellular matrix under inflammatory conditions. This ultimately can lead to reduction of aveolar bone-loss in patients with periodontal disease (as well as patients without periodontitis).

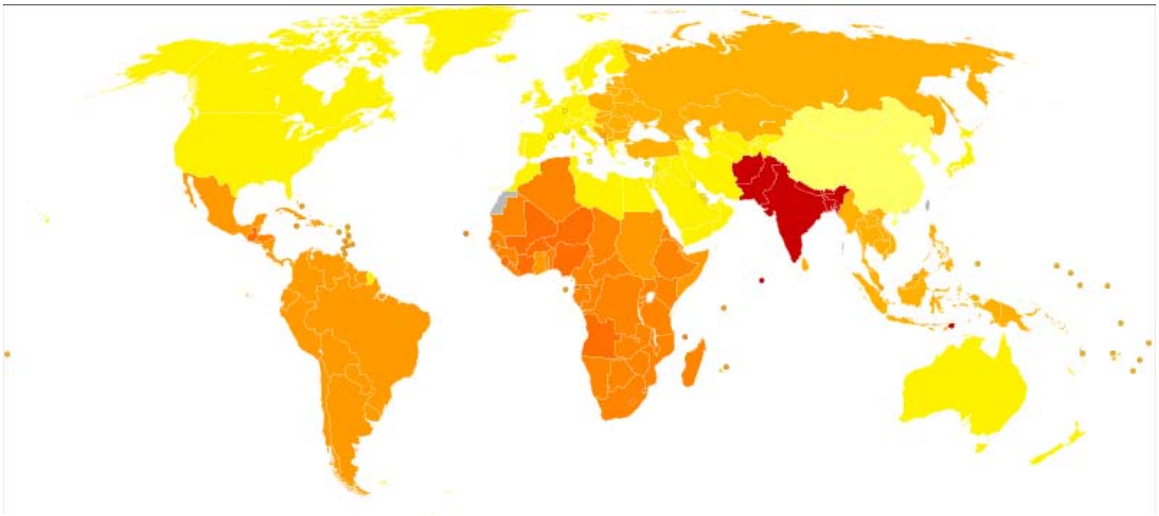
Prognosis

Dentists and dental hygienists measure periodontal disease using a device called a periodontal probe. This is a thin "measuring stick" that is gently placed into the space between the gums and the teeth, and slipped below the gum-line. If the probe can slip more than 3 millimeters below the gum-line, the patient is said to have a gingival pocket if no migration of the epithelial attachment has occurred or a periodontal pocket if apical migration has occurred. This is somewhat of a misnomer, as any depth is in essence a pocket, which in turn is defined by its depth, i.e., a 2 mm pocket or a 6 mm pocket. However, it is generally accepted that pockets are self-cleansable (at home, by the patient, with a toothbrush) if they are 3 mm or less in depth. This is important because if there is a pocket which is deeper than 3 mm around the tooth, at-home care will not be sufficient to cleanse the pocket, and professional care should be sought. When the pocket depths reach 6 and 7 mm in depth, the hand instruments and cavitrons used by the dental professionals may not reach deeply enough into the pocket to clean out the microbic plaque that cause gingival inflammation. In such a situation the bone or the gums around that tooth should be surgically altered or it will always have inflammation which will likely result in more bone loss around that tooth. An additional way to stop the inflammation would be for the patient to receive subgingival antibiotics (such as minocycline) or undergo some form of gingival surgery to access the depths of the pockets and perhaps even change the pocket depths so that they become 3 mm or less in depth and can once again be properly cleaned by the patient at home with his or her toothbrush.

If a patient has 7 mm or deeper pockets around their teeth, then they would likely risk eventual tooth loss over the years. If this periodontal condition is not identified and the patient remains unaware of the progressive nature of the disease then, years later, they may be surprised that some teeth will gradually become loose and may need to be extracted, sometimes due to a severe infection or even pain.

According to the Sri Lankan tea labourer study, in the absence of any oral hygiene activity, approximately 10% will suffer from severe periodontal disease with rapid loss of attachment (>2 mm/year). 80% will suffer from moderate loss (1–2 mm/year) and the remaining 10% will not suffer any loss.

Epidemiology



Disability-adjusted life year for peridontal disease per 100,000 inhabitants in 2004.

- no data
- less than 3.5
- 3.5-4
- 4-4.5
- 4.5-5
- 5-5.5
- 5.5-6
- 6-6.5
- 6.5-7
- 7-7.5
- 7.5-8
- 8-8.5
- more than 8.5

Periodontitis is very common, and is widely regarded as the second most common disease worldwide, after dental decay, and in the United States has a prevalence of 30–50% of the population, but only about 10% have severe forms.

Like other conditions that are intimately related to access to hygiene and basic medical monitoring and care, periodontitis tends to be more common in economically disadvantaged populations or regions. Its occurrence decreases with higher standard of living. In Israeli population, individuals of Yemenite, North-African, South Asian, or Mediterranean origin have higher prevalence of periodontal disease than individuals from European descent.

Presumably, individuals living in East Asia (e.g. Japan, South Korea and Taiwan) have the lowest incident of periodontal disease in the world.

In other animals

Periodontal disease is the most common disease found in dogs and affects more than 80% of dogs aged three years or older. The prevalence of periodontal disease in dogs increases with age but decreases with increasing body weight; i.e., toy and miniature breeds are more severely affected. Systemic disease may develop because the gums are very vascular (have a good blood supply). The blood stream carries these anaerobic microorganisms, and they are filtered out by the kidneys and liver, where they may colonize and create microabscesses. The microorganisms traveling through the blood may also attach to the heart valves, causing vegetative endocarditis (infected heart valves). Additional diseases that may result from periodontitis includes chronic bronchitis and pulmonary fibrosis.

Chapter 10

Warthin's Tumor

Warthin's tumor

This Warthin's tumor presented as a parotid mass in a middle-aged male, who underwent superficial parotidectomy. The tumor, at the right of the image, is well-demarcated from the adjacent parotid tissue and tends to shell out from it.

ICD-10	D11.
ICD-9	210.2
ICD-O:	8561/0
DiseasesDB	31941
eMedicine	plastic/371
MeSH	D000235

Warthin's tumor or **Warthin tumour**, also known as **papillary cystadenoma lymphomatosum** or adenolymphoma, is a type of benign tumor of the salivary glands.

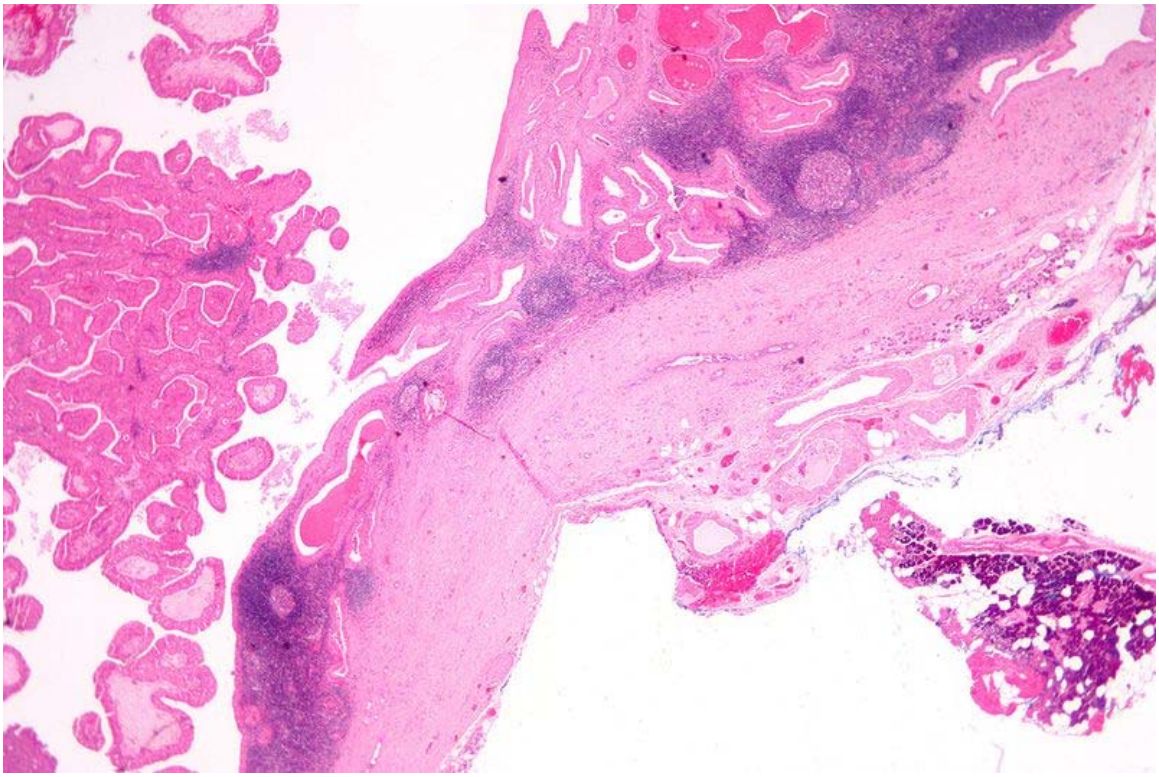
Etiology

Its etiology is unknown, but there is a strong association with cigarette smoking. Smokers are at 8 times greater risk of developing Warthin's tumor than the general population.

Locations

The gland most likely affected is the parotid gland. Though much less likely to occur than pleomorphic adenoma, Warthin's tumor is the second most common benign parotid tumor.

Characteristic



Low magnification micrograph of a **Warthin tumor** arising from the parotid gland.

Warthin's tumor primarily affects older individuals (age 60–70 years). There is a slight female predilection according to recent studies, but historically it has been associated with a strong male predilection. This change is possibly due to the tumor's association with cigarette smoking and the growing use of cigarettes by women. The tumor is slow growing, painless, and usually appears in the tail of the parotid gland near the angle of the mandible. In 5–14% of cases, Warthin's tumor is bilateral, but the two masses usually are at different times. Warthin's tumor is highly unlikely to become malignant.

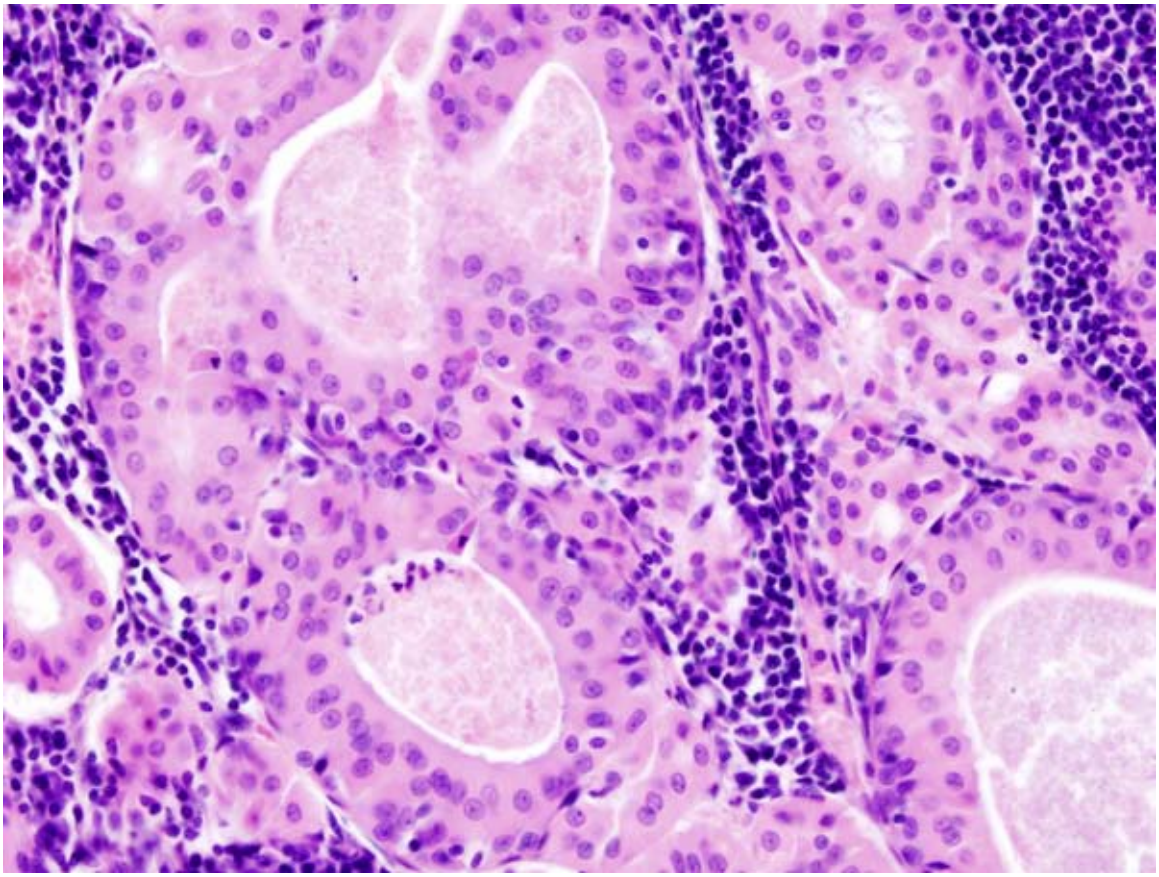
Histology

The appearance of this tumor under the microscope is unique. There are cystic spaces surrounded by two uniform rows of cells with centrally placed pyknotic nuclei. The cystic spaces have epithelium referred to as papillary infoldings that protrude into them. Additionally, the epithelium has lymphoid stroma with germinal center formation.

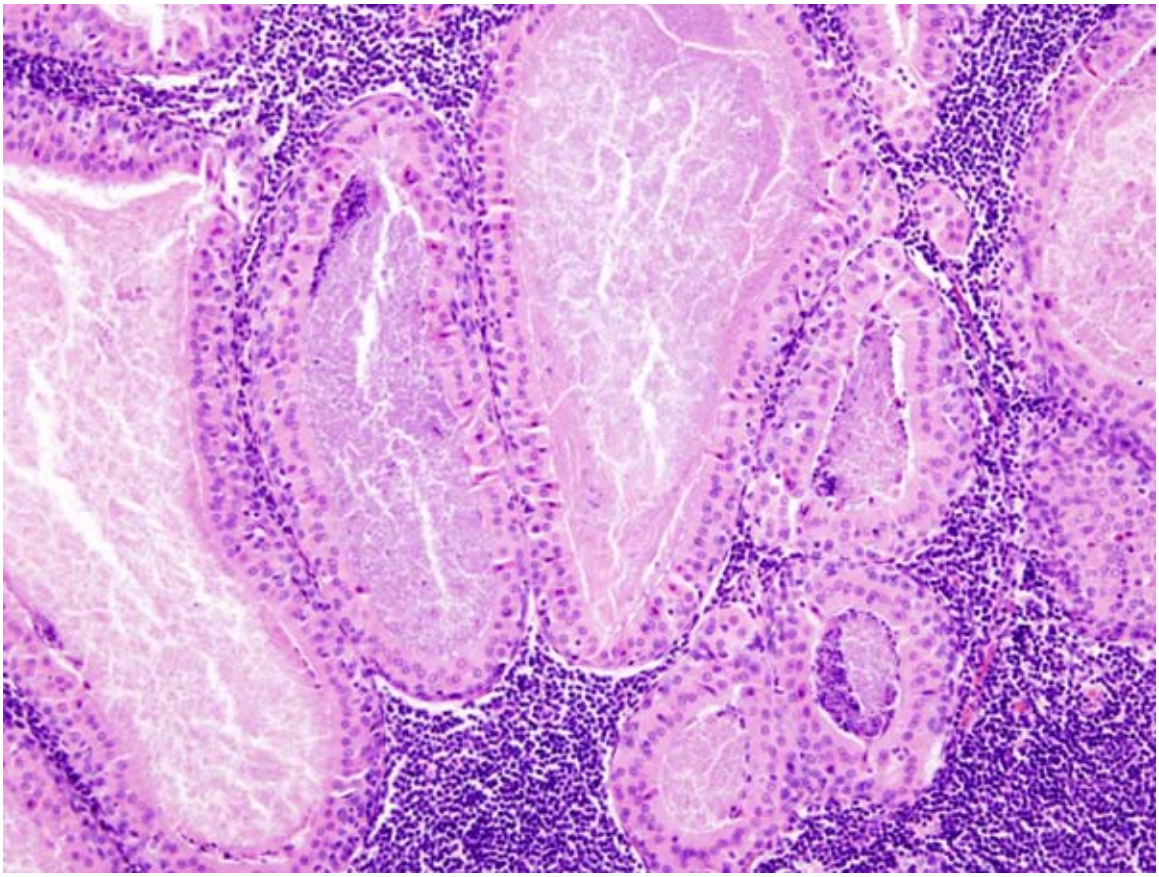
Treatment

Most of these tumors are treated with surgical removal. Recurrence is rare, occurring in 6–12% of cases.

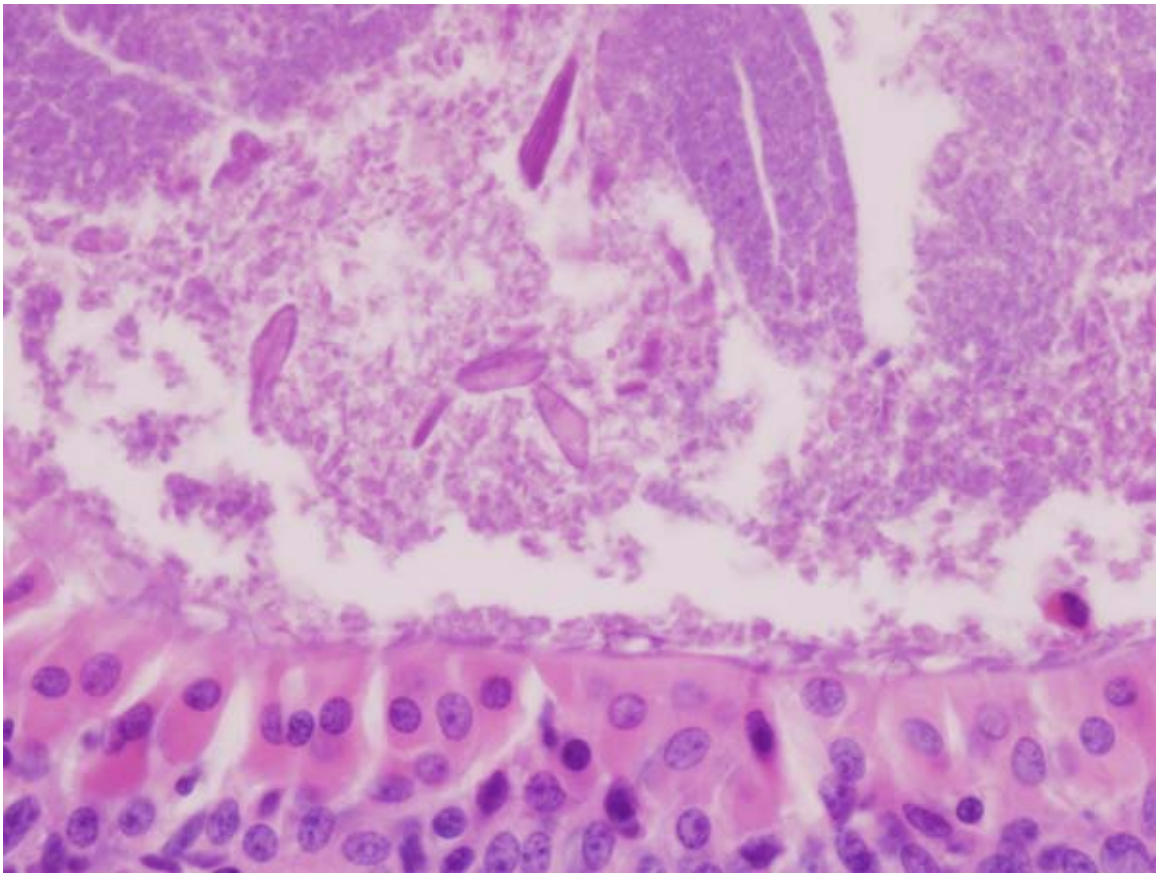
Additional images



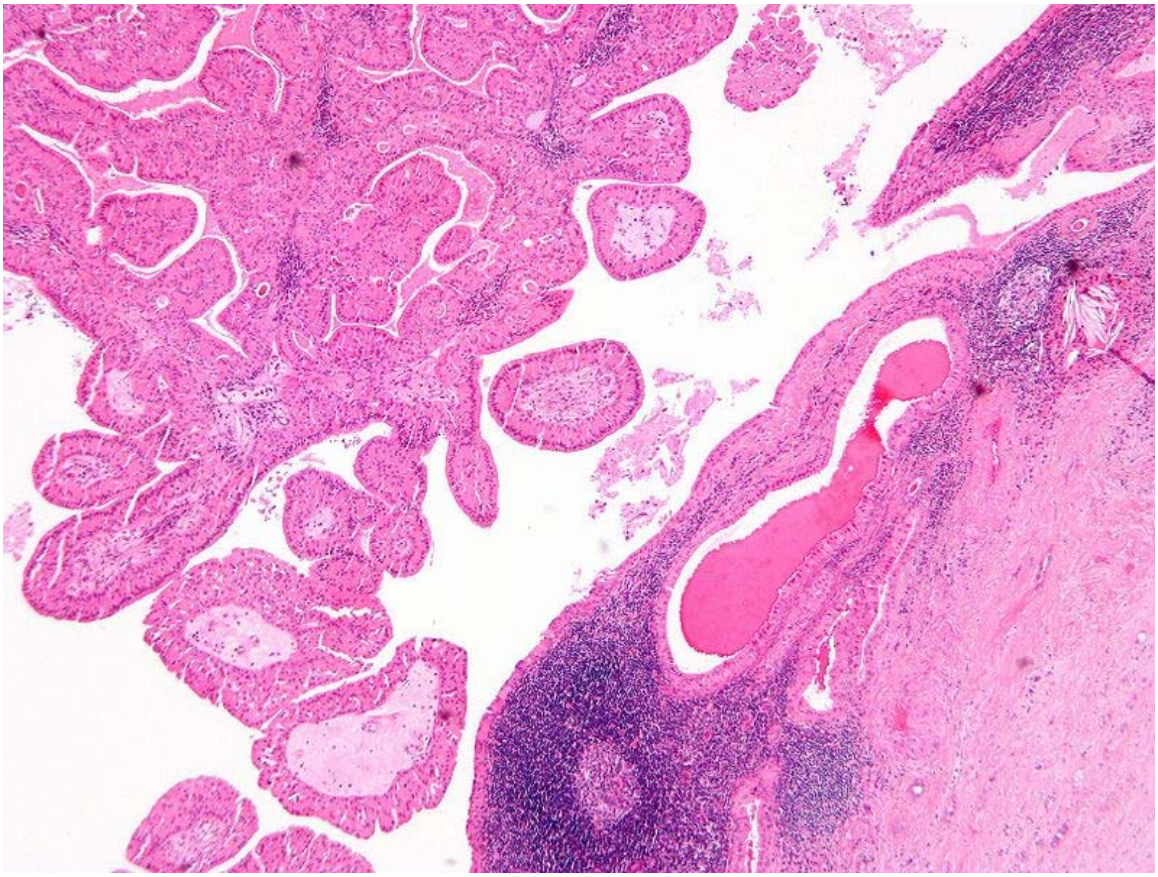
Histopathology of Warthin tumor in the parotid gland. H&E stain.



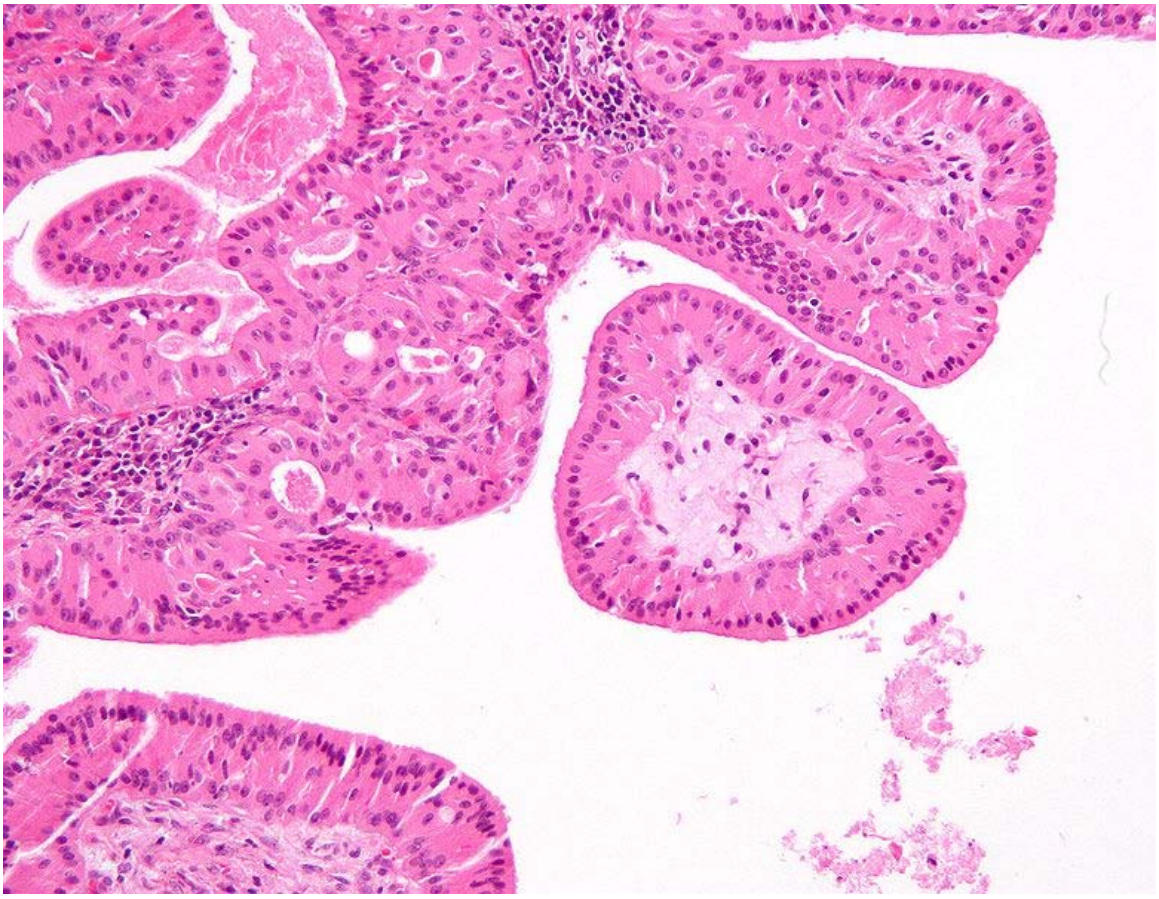
Histopathology of Warthin tumor in the parotid gland. Another view of a file H&E stain.



Histopathology of Warthin tumor in the parotid gland. Higher magnification of a file. H&E stain.



Intermediate magnification micrograph of a **Warthin tumor**.

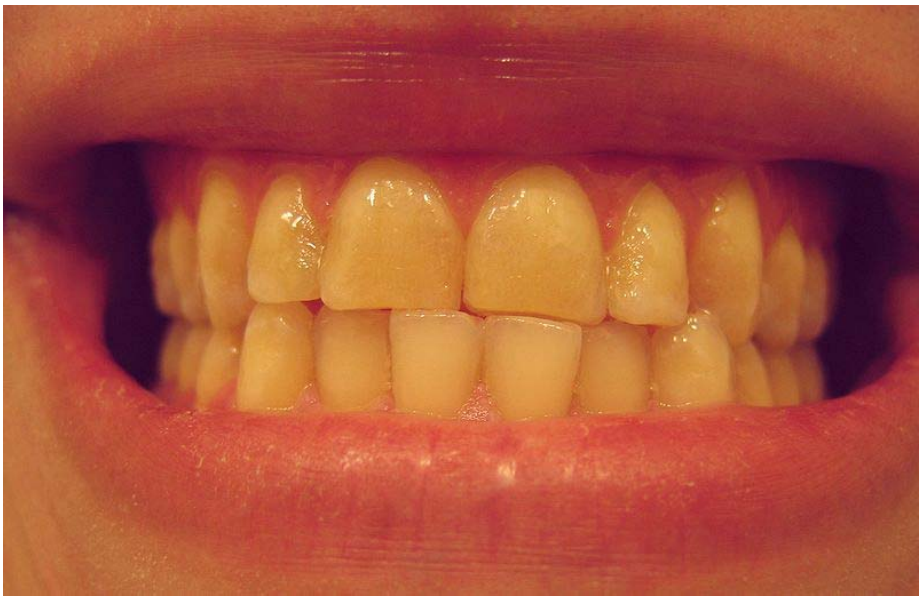


High magnification micrograph of a **Warthin tumor** showing the characteristic bilayered epithelium.

Chapter 11

Bruxism

Bruxism



A profile of a smile, exhibiting significant wear, especially on the maxillary incisors. Even though the teeth are in an edge-to-edge position, the teeth are in maximum intercuspation; this patient has a Class III malocclusion.

ICD-10	F45.8
ICD-9	306.8
DiseasesDB	29661
MedlinePlus	001413
MeSH	D002012

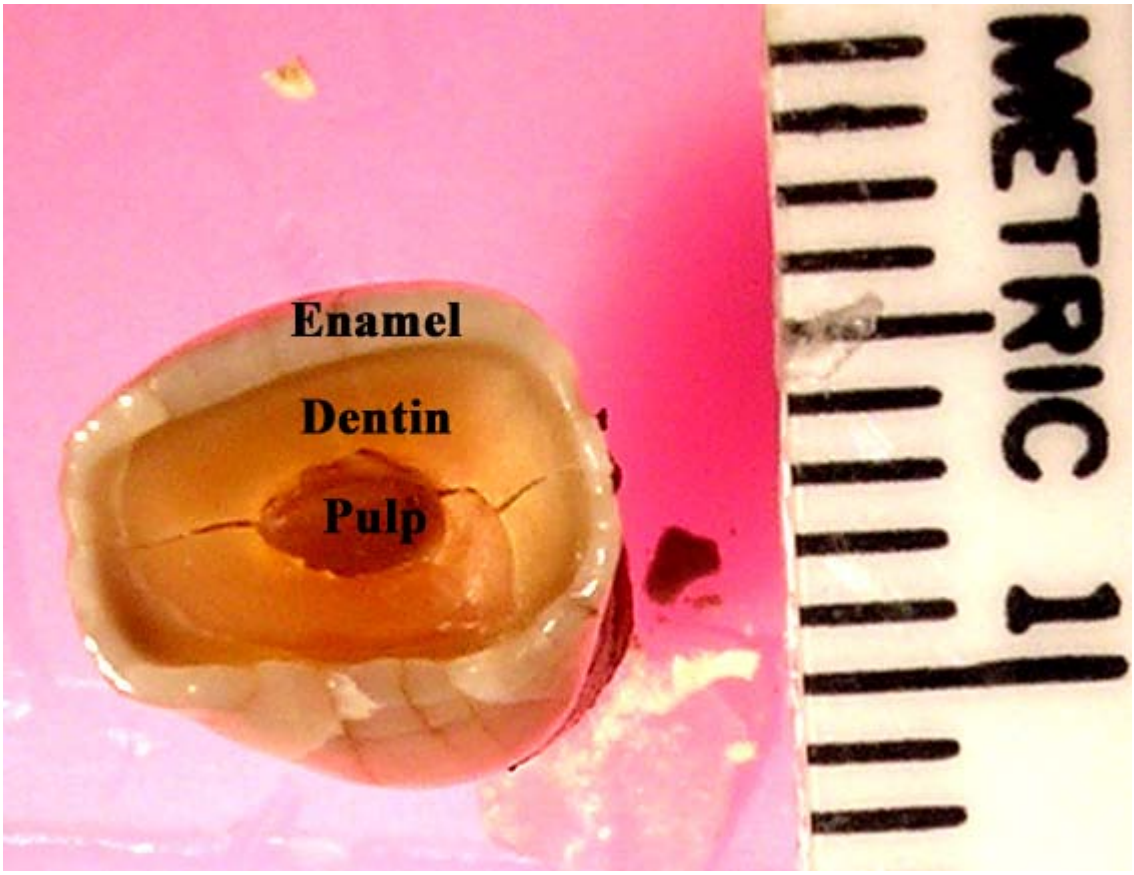
Bruxism (from the Greek βρυγμός (*brygmós*), "gnashing of teeth") is characterized by the grinding of the teeth and is typically includes the clenching of the jaw. It is an oral parafunctional activity that occurs in most humans at some time in their lives. In most people, bruxism is mild enough not to be a health problem. While bruxism may be a diurnal or nocturnal activity, it is bruxism during sleep that causes the majority of health issues and can even occur during short naps. Bruxism is one of the most common sleep disorders.

Causes

Multiple articles have incorrectly cited bruxism as a reflex chewing activity; bruxism is more accurately classified as a habit. Reflex activities happen reliably in response to a stimulus, without involvement of subconscious brain activity, and bruxism does not. All habitual activities are triggered by one kind of stimulus or another, and that does not make the habit a reflex. Chewing is a complex neuromuscular activity that is controlled by subconscious processes, with higher control by conscious processes within the brain. During sleep, (and for some during waking hours while conscious attention is on something else) the subconscious processes can run unchecked while the higher control is inactive, resulting in bruxism. Some bruxism activity is rhythmic with bite force pulses of tenths of a second (like chewing), and some has a longer bite force pulses of 1 to 30 seconds (clenching). Researchers classify bruxism as "a habitual behavior, and a sleep disorder."

The etiology of problematic bruxism can be quite varied, from allergic reactions or medical ailments, to trauma (such as an auto accident) to a period of unusual stress, but once bruxism becomes neurologically established as a habit, the original cause can be removed and the habit remains. It is theorized that certain medical conditions can trigger bruxism, including digestive ailments and anxiety.

Signs



The effects of bruxism on an anterior tooth, revealing the dentin and pulp which are normally hidden by enamel

Most bruxers are not aware of their bruxism, and some dentists estimate that only 5% go on to develop symptoms, such as jaw pain and headaches, which will require treatment. In many cases, a sleeping partner or parent will notice the bruxism before the person experiencing the problem becomes aware of it, although bruxers may be aware of secondary pain symptoms and not realize that those symptoms stem from bruxism.

Bruxism can result in abnormal wear patterns of the occlusal surface, abfractions and fractures in the teeth. This type of damage is categorised as a sign of occlusal trauma.

Over time, dental damage will usually occur. Bruxism is the leading cause of occlusal trauma and a significant cause of tooth loss and gum recession.

In a typical case involving lateral motion, the canines and incisors of the opposing arches are moved against each other laterally, i.e., with a side-to-side action, by the medial pterygoid muscles that lie medial to the temporomandibular joints bilaterally. This movement abrades tooth structure and can lead to the wearing down of the incisal edges of the teeth. People with bruxism may also grind their posterior teeth, which will wear

down the cusps of the occlusal surface. Most (but not all) bruxism includes clenching force provided by masseter and temporalis muscle groups, but some bruxers clench and grind front teeth only, and this type of clenching involves neither masseter nor temporalis muscle groups. Bruxism can be loud enough to wake a sleeping partner. Some individuals will clench the jaw without significant lateral movements. Teeth hollowed by previous decay (caries), or dental drilling, may collapse, as the cyclic pressure exerted by bruxism is extremely taxing on the tooth structure.

Symptoms

Patients may present with a variety of symptoms, including:

- Anxiety, stress, and tension
- Depression
- Earache
- Eating disorders
- Headaches
- Migraines
- Loose teeth
- Tinnitus
- Gum recession
- Neck pain
- Insomnia
- Sore or painful jaw

Sequelae

Eventually, bruxism with lateral movements shortens and blunts the teeth being ground and may lead to myofascial muscle pain, temporomandibular joint dysfunction and headaches. In severe, chronic cases, it can lead to arthritis of the temporomandibular joints. The jaw clenching that is often part of bruxism can be an unconscious neuromuscular daytime activity, which should be treated as well, usually through physical therapy (recognition and stress response reduction).

Diagnosis

Bruxism can sometimes be difficult to diagnose by visual evidence alone, as it is not the only cause of tooth wear. Over-vigorous brushing, abrasives in toothpaste, acidic soft drinks and abrasive foods can also be contributing factors, although each causes characteristic wear patterns that a trained professional can identify. Additionally, the presenting symptoms may be difficult for a physician to attribute to bruxism.

The effects of bruxism may be quite advanced before sufferers are aware they brux. Abraded teeth are usually brought to the patient's attention during a routine dental examination. If enough enamel has been abraded, the softer dentin will be exposed, and

abrasion will accelerate. This opens the possibility of dental decay and tooth fracture, and in some people, gum recession. Early intervention by a dentist is advisable.

The most reliable way to diagnose bruxism is through EMG (electromyographic) measurements. These measurements pick up electrical signals from the chewing muscles (masseter and temporalis). This is the method used in sleep labs. There are three forms of EMG measurement available to consumers for use outside sleep labs. The first is bedside EMG units similar to those used by sleep labs. These units can be purchased for about $2000 and pick up their signals from facial muscles through wires connecting the bedside unit to electrodes that are adhesively attached to the user's face. TENS electrodes or ECG electrodes may be used.

The second type of EMG measurement available to consumers is a self-contained $400 EMG measurement headband sold under the trade name *SleepGuard*, available on loan from some dentists or at a rental rate of $50 per month from the manufacturer. The EMG measurement headband does not require adhesive electrodes or wires attached to the face. While it does not record the exact time, duration, and strength of each clenching incident as the most expensive bedside EMG monitors do, it does record the total number of clenching incidents and the total clenching time each night. These two numbers easily distinguish clenching from rhythmic grinding and allow dentists to quantify severity levels accurately. These self-contained units can also function as a Biofeedback Headband, to help people train themselves out of their bruxism habit.

Bedside EMG units and the self-contained EMG measurement headband can both be used either in silent mode as a diagnosis measurement or in biofeedback mode as a treatment.

A third method of diagnosis using EMG is available in disposable form under the trade name BiteStrip. The BiteStrip is a self-contained EMG module that adhesively mounts to the side of the face over the masseter muscle. The BiteStrip can only do one night of measurement and does not display the clench count or total clenching time, but rather provides a single-digit display related to bruxism severity. The BiteStrip provides significantly less information than an EMG bedside unit or EMG headband and costs about $60 per day to use.

Associated factors

The following factors are associated with bruxism:

- Disturbed sleep patterns and other sleep disorders (obstructive sleep apnea, hypopnea, snoring, moderate daytime sleepiness)
- Malocclusion, in which the upper and lower teeth occlude in a disharmonic way, e.g., through premature contact of back teeth
- Relatively high levels of consumption of caffeinated drinks and foods, such as coffee, colas, and chocolate
- High levels of blood alcohol

- Smoking
- High levels of anxiety, stress, work-related stress, irregular work shifts, stressful profession and ineffective coping strategies
- Drug use, such as SSRIs and stimulants, including methylenedioxymethamphetamine (ecstasy), methylenedioxyamphetamine (MDA), methylphenidate and other amphetamines, including those taken for medical reasons
- Hypersensitivity of the dopamine receptors in the brain
- GHB and similar GABA-inducing analogues such as Phenibut, when taken with high frequency
- Disorders such as Huntington's and Parkinson's diseases
- Obsessive Compulsive Disorder

Treatment

In cases where the initial cause of bruxism is still present, bruxism may be treated by finding and eliminating such cause. In cases where bruxism has become a habit and the original cause is no longer present, various habit-modification approaches can be taken to alter the pattern of bruxism that has been subconsciously learned. In some cases bruxism may be reduced or even eliminated when the associated factors, e.g., sleep disorders, are treated successfully.

Mouth guards and splints

Ongoing management of bruxism can be based on minimizing the abrasion of tooth surfaces by the wearing of a dental guard, or splint. Such a splints range in price from over $1000 for some dentist-made splints, to $20 for consumer-moldable or consumer-adjustable splints available at pharmacies. Splints obtained at dental offices are usually made from a molds taken from an individual's upper and/or lower teeth, and the fitting process often involves several trips to the dentist for measuring, fitting, and ongoing supervision. There are four possible goals of this treatment: constraint of the bruxing pattern such that serious damage to the temporomandibular joints is prevented, stabilization of the occlusion by minimizing the gradual changes to the positions of the teeth that typically occur with bruxism, prevention of tooth damage, and the enabling of a bruxism practitioner to judge—in broad terms—the extent and patterns of bruxism through examination of the physical indentations on the surface of the splint. A dental guard is typically worn on a long-term basis during every night's sleep. Although mouth guards are a first response to bruxism, they do not help cure it. Dentist-made mouthguards can cost anywhere from $200 to over $1000. Professional treatment may be medically recommended to ensure proper fit and make ongoing adjustments as needed. The consumer should beware that, at dental shows, manufacturers of materials and equipment used in making mouth guards boast the making of mouth guards as "the second most profitable service in dentistry". Some dentists simply recommend their patients buy an inexpensive customizable mouth guard from a pharmacy.

Another device sometimes given to a bruxer is a repositioning splint. A repositioning splint may look similar to a traditional night guard but is designed to change the occlusion, or bite, of the patient. Randomly controlled trials with these type devices generally show no benefit over more conservative therapies.

An NTI-tss (nociceptive trigeminal inhibitor) mouth guard is another option that can be considered. Nociceptors are nerves that sense and respond to pressure. The trigeminal nerve supplies the face and mouth. The NTI appliance is made to snap onto the front teeth. Normally when the mouth is closed, the upper and lower front teeth overlap: The NTI prevents this overlap and, if one attempts to close the jaw normally while wearing an NTI device, the NTI translates the bite force into a forward twisting of the lower front teeth. The intent is that the nerve sensations from this will be interpreted by the brain as undesirable, and the clenching force will automatically subconsciously be reduced. Thus when bruxism starts in the night the pressure which is applied to the two front teeth can, it is claimed, send quite a strong alarm signal to the brain. The NTI device must be fitted by a dentist.

The efficacy of such devices is debated. Some writers propose that irreversible complications can result from the long-term use of mouthguards and repositioning splints. The danger of the NTI device is that if one does clench hard on it, worse damage may result, because the presence of the NTI changes the forces on the teeth and the tempormandibular joint, so patients using an NTI device should have ongoing monitoring by a dentist.

Biofeedback

Various biofeedback devices are currently available, and effectiveness varies significantly depending on whether the biofeedback is used only during waking hours, or during sleep as well. Many authorities remain unconvinced of the efficacy of daytime-only biofeedback. The efficacy of biofeedback delivered during sleep can depend strongly on daytime training, which is used to establish a Pavlovian response to the biofeedback signal that persists during sleep.

The first wearable nighttime bruxism biofeedback device (introduced in 2001), was originally sold under the trademark GrindAlert by BruxCare, and is now sold under the trademark *SleepGuard* by Holistic Technologies, which owns the patents on the technology. The SleepGuard biofeedback headband is a battery-powered device that sounds a tone against the forehead when it senses EMG (electromyographic) muscle activity in the temporalis muscles. The tone starts out very quiet and then gets louder, allowing people to stop clenching without waking up. This standard model records and displays nightly data on the number of bruxism events that last for at least two seconds and the total accumulated duration of those events. A fast-response model is also available, which records and displays nightly data on the number of bruxism events that last for at least 0.2 seconds. The volume of the alarm and the bite force required to trigger the device are adjustable. After proper Pavlovian training during waking hours, more than at least a 60% reduction in bruxism within a few days, and more than an 80% reduction

in bruxism within one month. The biofeedback sound on the headband is designed to come on slowly, allowing users to subconsciously respond in their sleep without waking up. The manufacturer offers a free three-week trial so that only people who find the device works well for them have to pay for it and claims that less than 15% of trial units are returned.

A mild electric shock bio-feedback device for treating Bruxism, GrindCare, has been approved by the European regulatory authorities and was introduced to the market in 2Q2008 - and was approved by FDA Authorities in the US in early 2010. The device works by using simple electrodes adhesively attached to the skin close to the cheek bones prior to sleeping; it detects the initial muscular contractions and immediately provides mild electrical shock pulses to the facial muscles. The electric shocks serve to interrupt bruxism activity. The device is worn on the head and reportedly reduces grinding, usually without interfering with the sleep of the patient as described by Jadidi, Castrillon & Svensson. Thereby facial tension, joint defects and teeth disruption are reportedly reduced.

A taste-based biofeedback method was developed by Moti Nissani, Ph.D. and is called "The Taste-Based Approach to the Prevention of Teeth Clenching and Grinding". The therapy involves suspending sealed packets containing a bad-tasting substance (e.g. hot sauce, vinegar, denatonium benzoate, etc.) between the rear molars using an orthodontic-style appliance. Any attempt to bring the teeth together will rupture the packets and alert the user to the habit. This approach finds favor with some people who prefer to relate to biofeedback as "aversive therapy". The Taste-Based Approach claims to suffer less from desensitization over time than sound-based biofeedback approaches may have, but may interrupt sleep more. (There is effectively no limit to the aversive taste of certain substances.)

One bruxism biofeedback device which was briefly on the market but is no longer available was sold under the trademark Oralsensor. This device consisted of a pneumatic pouch embedded in a soft polymer plate that fits over upper or lower teeth. When the teeth came together with a force that exceeded a set threshold, an alarm is sounded in an earpiece worn by the user; the device is no longer sold.

In 2005, a new type of occlusive device was patented that produces a movement incompatible with teeth clenching. When nighttime bruxism occurs, people breathe through the nose. The device forces people to breathe through the mouth; by forcing the opening of the mouth, the device is claimed to stop clenching. The occlusive device has an electromyogram system that monitors the electric activity of the jaw muscle via wireless electrodes. These electrodes transfer jaw-muscle activity by radio frequency to an external monitoring system. Once the signal has been interpreted by the monitoring system, if a person clenches, the monitoring unit sends a radio frequency signal to a transceiver integrated in a mechanical actuator. The mechanical actuator has two occlusive flaps that block the nostrils, forcing breathing to occur through the mouth. Once the patient stops clenching, the flaps open, allowing breathing through the nose

again. The occlusive device does not wake up people since it blocks nostrils slowly, and it never closes them completely to avoid sleep disruption.

Botox

Botulinum toxin (Botox) can be successful in lessening effects of bruxism, though serious side-effects (including death) are possible. In extremely dilute form (Botox), this toxin is used as an injectable local paralysis agent that weakens (partially paralyzes) muscles and has been used extensively in cosmetic procedures to 'relax' the muscles of the face and decrease the appearance of wrinkles. In April, 2008, a study was published in the *Journal of Neuroscience* that showed that facially injected Botox can and does propagate into the brains of some test animals, and the U.S. Food and Drug Administration (FDA) announced that it was beginning a safety review of Botox and other similar drugs. Less than one microgram of botulinum toxin ingested or inhaled is sufficient to kill an adult human, and although the intent of the injections is that the toxin should stay localized in the muscle, some of it does migrate (as it would if injested or inhaled).

Botox was not originally developed for cosmetic use. It was, and continues to be, used to treat diseases of muscle spasticity such as blepharospasm (eyelid spasm), strabismus (crossed eyes) and torticollis (wry neck). Bruxism can also be regarded as a disorder of repetitive, unconscious contraction of the masseter muscle (the large muscle that moves the jaw). In the treatment of bruxism, Botox works to weaken the muscle enough to reduce the effects of grinding and clenching, but not so much as to prevent proper use of the muscle. The strength of Botox is that the medication goes into the muscle and is not supposed to get absorbed into the body (though the new research shows it does). The procedure involves about five or six simple, relatively painless injections into the masseter muscle. It takes a few minutes per side, and the patient starts feeling the effects the next day. Occasionally, some bruising can occur, but this is quite rare. Injections must be repeated more than once per year, and the risk factor of spread of the botulinum toxin is compounded by this repetition.

The symptoms that can be helped by this procedure include:

- Grinding and clenching
- Morning jaw soreness
- TMJ pain
- Muscle tension throughout the day
- Migraines triggered by clenching
- Neck pain and stiffness triggered by clenching

The optimal dose of Botox must be determined for each person as some people have stronger muscles that need more Botox. This is done over a few touch-up visits with the physician injector. This treatment is expensive, but sometimes Botox treatment of bruxism can be billed to medical insurance. The effects last for about three months. The

muscles do atrophy, however, so after a few rounds of treatment, it is usually possible either to decrease the dose or increase the interval between treatments.

Other authorities caution that Botox should only be used for temporary relief for severe cases and should be followed by diagnosis and treatment to prevent future bruxism or jaw clenching, suggesting that prolonged use of Botox can lead to permanent damage to the jaw muscle.

Dietary supplements

There is anecdotal evidence that suggests taking certain combinations of dietary supplements may alleviate bruxism; pantothenic acid, magnesium, and calcium are mentioned on dietary supplement websites. Calcium is known to be a treatment for gastric problems, and gastric problems such as acid reflux are known to increase bruxism.

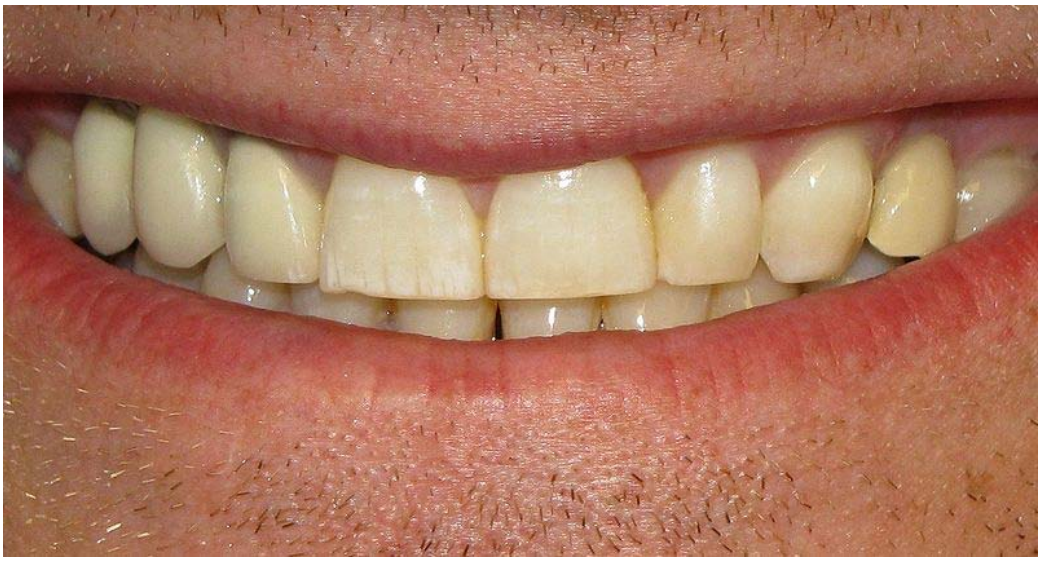
Repairing damage

Damaged teeth can be repaired by replacing the worn natural crown of the tooth with prosthetic crowns. Materials used to make crowns vary; some are less prone to breaking than others and can last longer. Porcelain fused to metal crowns may be used in the anterior (front) of the mouth; in the posterior, full gold crowns are preferred. All-porcelain crowns are now becoming more and more common and work well for both anterior and posterior restorations. To protect the new crowns and dental implants, an occlusal guard should be fabricated to wear during sleep.

Chapter 12

Dental Fluorosis

Dental fluorosis



A mild case of dental fluorosis (the white streaks on the subject's upper right central incisor) observed in dental practice

ICD-10	K00.3
ICD-9	520.3

Dental fluorosis is a health condition caused by a child receiving too much fluoride during tooth development. The critical period of exposure is between 1 and 4 years old; children over age 8 are not at risk. In its mild form, which is the most common, fluorosis appears as tiny white streaks or specks that are often unnoticeable. In its severest form, which is also called mottling of dental enamel, it is characterized by black and brown stains, as well as cracking and pitting of the teeth.

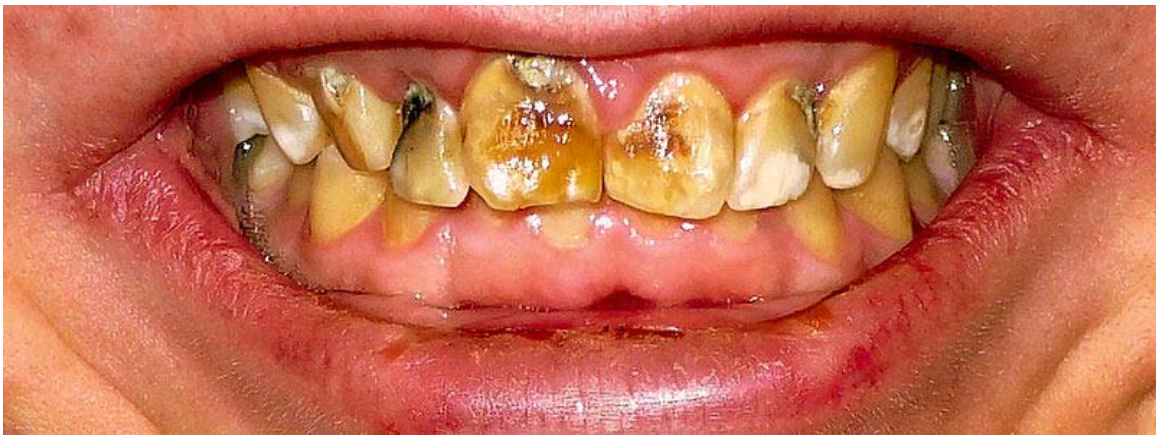
The severity of dental fluorosis depends on the amount of fluoride exposure, the age of the child, individual response, as well as other factors including nutrition. Although water fluoridation can cause fluorosis, most of this is mild and not usually of aesthetic concern. Severe cases can be caused by exposure to water that is naturally fluoridated to levels well above the recommended levels, or by exposure to other fluoride sources such as brick tea or pollution from high fluoride coal.

Physiology

Dental fluorosis occurs because of the excessive intake of fluoride, either through fluoride in the water supply, naturally occurring or added to it; or through other sources. The damage in tooth development occurs between the ages of 3 months to 8 years, from the overexposure to fluoride. Teeth are generally composed of hydroxyapatite and carbonated hydroxyapatite; when fluoride is present, some fluorapatite is generated. Excessive fluoride can cause white spots, and in severe cases, brown stains or pitting or mottling of enamel. Fluorosis cannot occur once the tooth has erupted into the oral cavity. At this point, fluorapatite is beneficial because it is more resistant to dissolution by acids (demineralization). Although it is usually the permanent teeth which are affected, occasionally the primary teeth may be involved.

The differential diagnosis for this condition may include Turner's hypoplasia (although this is usually more localized), some mild forms of amelogenesis imperfecta, and other environmental enamel defects of diffuse and demarcated opacities.

Dean's Index



A severe case of dental fluorosis, or **"mottled dental enamel."**

H.T. Dean's fluorosis index was developed in 1942 and is currently the most universally accepted classification system. An individual's fluorosis score is based on the most severe form of fluorosis found on two or more teeth.

Dean's Index

Classification	Criteria – description of enamel
Normal	Smooth, glossy, pale creamy-white translucent surface
Questionable	A few white flecks or white spots
Very Mild	Small opaque, paper white areas covering less than 25% of the tooth surface
Mild	Opaque white areas covering less than 50% of the tooth surface
Moderate	All tooth surfaces affected; marked wear on biting surfaces; brown stain may be present
Severe	All tooth surfaces affected; discrete or confluent pitting; brown stain present

Prevalence

As of 2005 surveys conducted by the National Institute of Dental Research in the USA between 1986 and 1987 and by the Center of Disease Control between 1999 and 2002 are the only national sources of data concerning the prevalence of dental fluorosis.

NIDR and CDC findings		
Deans Index	**1987**	**2002**
Questionable fluorosis	17%	11.8%
Very mild fluorosis		19%
Mild fluorosis	4%	5.83%
Moderate fluorosis	1%	0.59%
Severe fluorosis	0.3%	
Total	22.3%	37.2%

The U.S. Center of Disease Control found a 9% higher prevalence of dental fluorosis in American children than was found in a similar survey 20 years ago. In addition, the survey provides further evidence that African Americans suffer from higher rates of fluorosis than Caucasian Americans.

The condition is more prevalent in rural areas where drinking water is derived from shallow wells or hand pumps. It is also more likely to occur in areas where the drinking water has a fluoride content greater than 1 ppm (part per million), and in children who have a poor intake of calcium.

Dietary reference intakes for fluoride

Age group	Reference weight kg (lb)	Adequate intake (mg/day)	Tolerable upper intake (mg/day)
Infants 0-6 months	7 (16)	0.01	0.7
Infants 7-12 months	9 (20)	0.5	0.9
Children 1-3 years	13 (29)	0.7	1.3
Children 4-8 years	22 (48)	1.0	2.2
Children 9-13 years	40 (88)	2.0	10
Boys 14-18 years	64 (142)	3.0	10
Girls 14-18 years	57 (125)	3.0	10
Males 19 years and over	76 (166)	4.0	10
Females 19 years and over	61 (133)	3.0	10

If the water supply is fluoridated at the level of 1 ppm, one must consume one litre of water in order to take in 1 mg of fluoride. It is thus improbable a person will receive more than the tolerable upper limit from consuming optimally fluoridated water alone.

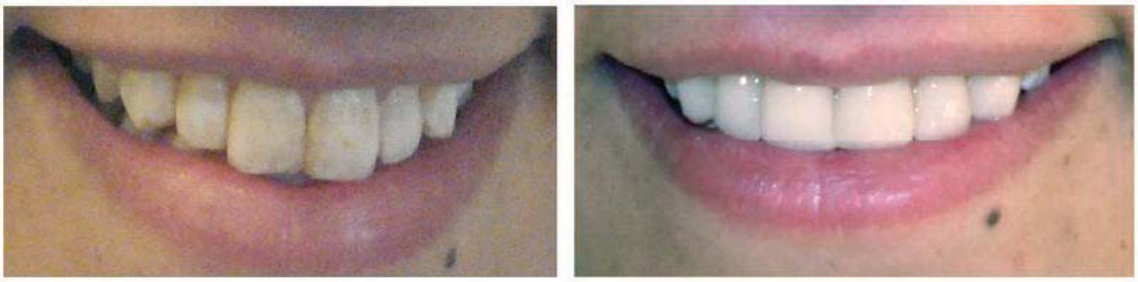
Fluoride consumption can exceed the tolerable upper limit when someone drinks a lot of fluoride containing water in combination with other fluoride sources, such as swallowing fluoridated toothpaste, consuming food with a high fluoride content, or consuming fluoride supplements. The use of fluoride supplements as a prevention for tooth decay is rare in areas with water fluoridation, but was recommended by many dentists in the UK until the early 1990s. Coal burning can pollute air with fluoride: indoor air with approximately 60 μg F/m³ and drinking water with 3.6 mg F/L are similarly toxic to developing permanent teeth.

Dental fluorosis can be prevented by lowering the amount of fluoride intake to below the tolerable upper limit.

American Dental Association advisory

In November 2006, the American Dental Association began recommending to parents that infants from 0 through 12 months of age should have their formula prepared with water that is fluoride-free, or contains low levels of fluoride to reduce the risk of fluorosis.

Treatment



Fluorosis before Treatment

After Treatment(Porcelain Laminate Veneers)

Before and after porcelain laminate veneers

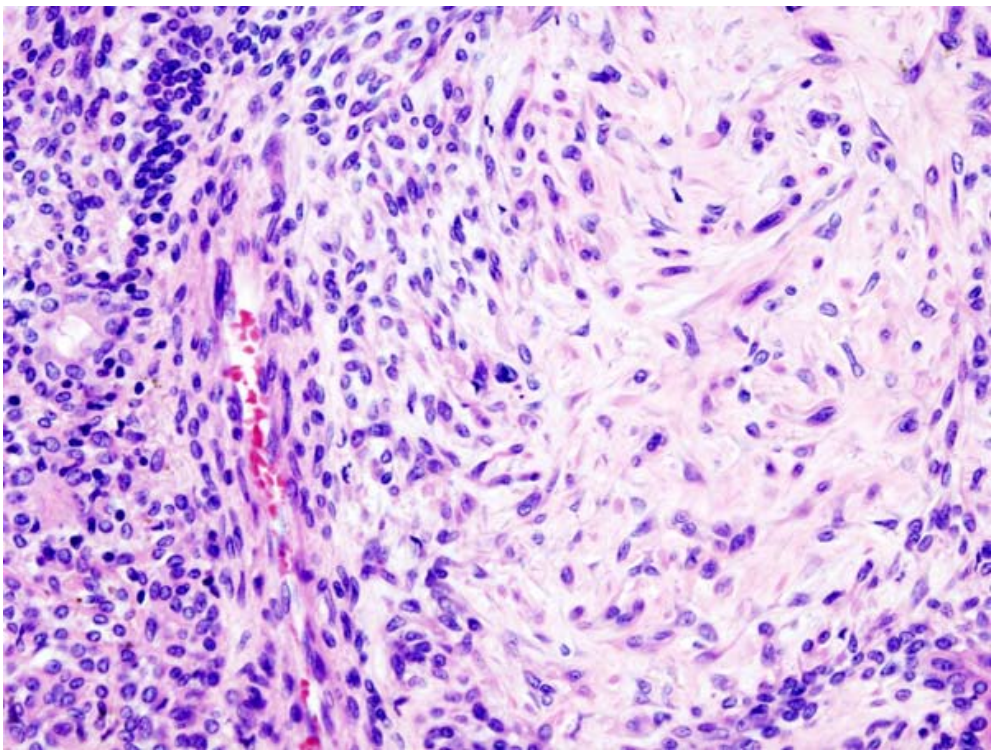
Dental fluorosis can be cosmetically treated by a dentist. The cost and success can vary significantly depending on the treatment. Tooth bleaching, microabrasion, and conservative composite restorations or porcelain veneers are commonly used treatments. Generally speaking, bleaching and microabrasion are used for superficial staining, whereas the conservative restorations are used for more unaesthetic situations.

Chapter 13

Pleomorphic Adenoma and Ossifying Fibroma

Pleomorphic adenoma

Pleomorphic adenoma



Pleomorphic adenoma consists of mixed epithelial (left) and mesenchymal cell components (right). The latter often exhibits myxofibrous appearance and in some

instances shows chondromatous differentiation.

ICD-10	D11.
ICD-9	210.2
ICD-O:	8940/0
OMIM	181030
eMedicine	radio/531
MeSH	D008949

Pleomorphic adenoma is a benign neoplastic tumor of the salivary glands. It is the most common type of salivary gland tumor and the most common tumor of the parotid gland. It derives its name from the architectural pleomorphism (variable appearance) seen by light microscopy. It is also known as "Mixed tumor, salivary gland type", which describes its pleomorphic appearance as opposed to its dual origin from epithelial and myoepithelial elements.

Clinical Presentation

The tumor is usually solitary and presents as a slow growing, painless, firm single nodular mass. Isolated nodules are generally outgrowths of the main nodule rather than a multinodular presentation. It is usually mobile unless found in the palate and can cause atrophy of the mandibular ramus when located in the parotid gland. When found in the parotid tail, it may present as an eversion of the ear lobe. Though it is classified as a benign tumor, pleomorphic adenomas have the capacity to grow to large proportions and may undergo malignant transformation, to form carcinoma ex-pleomorphic adenoma, a risk that increases with time. Although it is "benign" the tumor is aneuploid, it can recur after resection, it invades normal adjacent tissue and distant metastases have been reported after long (+10 years) time intervals.

Histology

Histologically, it is highly variable in appearance, even within individual tumors. Classically it is biphasic and is characterized by an admixture of polygonal epithelial and spindle-shaped myoepithelial elements in a variable background stroma that may be mucoid, myxoid, cartilaginous or hyaline. Epithelial elements may be arranged in duct-like structures, sheets, clumps and/or interlacing strands and consist of polygonal, spindle or stellate-shaped cells (hence pleiomorphism). Areas of squamous metaplasia and epithelial pearls may be present. The tumor is not enveloped, but it is surrounded by a fibrous pseudocapsule of varying thickness. The tumor extends through normal glandular parenchyma in the form of finger-like pseudopodia, but this is not a sign of malignant transformation.

The tumor often displays characteristic chromosomal translocations between chromosomes #3 and #8. This causes the PLAG gene to be juxtaposed to the gene for beta catenin. This activates the catenin pathway and leads to inappropriate cell division.

Diagnosis

The diagnosis of salivary gland tumors utilize both tissue sampling and radiographic studies. Tissue sampling procedures include fine needle aspiration (FNA) and core needle biopsy (bigger needle comparing to FNA). Both of these procedures can be done in an outpatient setting. Diagnostic imaging techniques for salivary gland tumors include ultrasound, computer tomography (CT) and magnetic resonance imaging (MRI).

Fine needle aspiration biopsy (FNA), operated in experienced hands, can determine whether the tumor is malignant in nature with sensitivity around 90%. FNA can also distinguish primary salivary tumor from metastatic disease.

Core needle biopsy can also be done in outpatient setting. It is more invasive but is more accurate compared to FNA with diagnostic accuracy greater than 97%. Furthermore, core needle biopsy allows more accurate histological typing of the tumor.

In terms of imaging studies, ultrasound can determine and characterize superficial parotid tumors. Certain types of salivary gland tumors have certain sonographic characteristics on ultrasound. Ultrasound is also frequently used to guide FNA or core needle biopsy.

CT allows direct, bilateral visualization of the salivary gland tumor and provides information about overall dimension and tissue invasion. CT is excellent for demonstrating bony invasion. MRI provides superior soft tissue delineation such as perineural invasion when compared to CT only.

Treatment

Overall, the mainstay of the treatment for salivary gland tumor is surgical resection. Needle biopsy is highly recommended prior to surgery to confirm the diagnosis. More detailed surgical technique and the support for additional adjuvant radiotherapy depends on whether the tumor is malignant or benign.

Generally, benign tumors of the parotid gland are treated with superficial or total parotidectomy with the latter being the more commonly practiced due to high incidence of recurrence. The facial nerve should be preserved whenever possible. The benign tumors of the submandibular gland is treated by simple excision with preservation of mandibular branch of the trigeminal nerve, the hypoglossal nerve, and the lingual nerve. Similarly, other benign tumors of minor salivary glands are treated similarly.

Malignant salivary tumors usually require wide local resection of the primary tumor. However, if complete resection cannot be achieved, adjuvant radiotherapy should be

added to improve local control. This surgical treatment has many sequellae such as cranial nerve damage, Frey's syndrome , cosmetic problems, etc.

Usually about 44% of the patients have a complete histologic removal of the tumor and this refers to the most significant survival rate.

But the locoregional control rate for the carcinoma pleomorphic adenoma is only 66% in the 5-year duration of the recovery time making the recurrence of the disease invariably fatal, and even the 44% of the disease-related specific cases report to high rate of mortality accounting to 87%.

Ossifying fibroma

Ossifying fibroma

ICD-O:	9262/0
MeSH	D018214

Ossifying fibroma or *peripheral ossifying fibroma* is an oral pathologic condition that appears in the mouth as an overgrowth of gingival tissue due to irritation or trauma. Because of its overwhelming incidence on the gingiva, the condition is associated with two other diseases, though not because they occur together. Instead, the three are associated with each other because they appear frequently on gingiva and they also begin with the letter "p": pyogenic granuloma and peripheral giant cell granuloma. Some researchers believe peripheral ossifying fibromas to be related to pyogenic fibromas and, in some instances, are the result of a pyogenic granuloma which has undergone fibrosis and calcification.

Presentation

The color of peripheral ossifying fibromas ranges from red to pink, and is frequently ulcerated. It can be sessile or pedunculated with the size usually being less than 2 cm. Weeks or months may pass by before it is seen and diagnosed.

There is a gender difference with 66% of the disease occurring in females. The prevalence of peripheral ossifying fibromas is highest around 10 – 19 years of age. It appears only on the gingiva, more often on the maxilla rather than the mandible, and is frequently found in the area around incisors and canines. The adjacent teeth are usually not affected.

Peripheral ossifying fibromas appear microscopically as a combination of a mineralized product and fibrous proliferation. The mineralized portion may be bone, cementum-like, or dystrophic calcifications. Additionally, highly developed bone or cementum is more likely to be present when the peripheral ossifying fibroma has existed for a longer period of time.

Treatment

Treatment usually involves surgical removal of the lesion down to the bone. If there are any adjacent teeth, they are cleaned thoroughly to remove any possible source of irritation. Recurrence is around 16%.