Dental Reference Manual

1. Dental caries ................................................................. 2
   a. Pathophysiology .................................................. 4
   b. Classification ....................................................... 7
   c. Rate of Progression ............................................. 9
   d. Signs and Symptoms ............................................. 10
   e. Diagnosis ............................................................ 11
   f. Treatment ............................................................ 12
   g. Prevention ........................................................... 13

2. Gingivitis ................................................................. 14
   a. Causes ................................................................. 15
   b. Symptoms ............................................................ 16
   c. Prevention ........................................................... 17
   d. Diagnosis ............................................................ 17
   e. Treatment ............................................................ 17

3. Oral Ulcer ............................................................... 18
   a. Causes ................................................................. 18
   b. Prevention ........................................................... 20
   c. Treatment ........................................................... 21

4. Oral Cancer ............................................................ 22
Dental Caries

Causes

There are four main criteria required for caries formation: a tooth surface (enamel or dentin); cariogenic (or potentially 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 that is exposed to the oral cavity, but not the structures which are retained within the bone.

 Teeth

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, the tooth is susceptible to dental caries.

The anatomy of teeth may affect the likelihood of caries formation. In cases 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.

Bacteria

The mouth contains a wide variety of bacteria, but only a few specific species of bacteria are believed to cause dental caries: Streptococcus mutans and Lactobacilli among them. Particularly for root caries, the most closely associated
bacteria frequently identified are Lactobacillus acidophilus, Actinomyces viscosus Nocardia spp., and Streptococcus mutans. These 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. In addition, the edges of fillings or crowns can provide protection for bacteria, as can intraoral appliances such as orthodontic braces or removable partial dentures.

Bacteria in a person's mouth convert sugars (glucose and fructose, and most commonly sucrose - or 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; suitable minerals are available in the mouth from saliva and also from preventative aids such as fluoride toothpaste, dental varnish or mouthwash. The advance of caries may be arrested at this stage. If sufficient acid is produced over a period of time to the favor of demineralization, caries will progress and may then result in so much mineral content being lost that the soft organic material left behind would disintegrate, forming a cavity or hole.

**Time**

The frequency of which teeth are exposed to cariogenic (acidic) environments affects the likelihood of caries development. After meals or snacks containing sugars, the bacteria in the mouth metabolize them resulting in acids as by-products which decreases pH. As time progresses, the pH returns to normal due to the buffering capacity of saliva and the dissolved mineral content from 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 2 hours. Since teeth are vulnerable during these periods of acidic environments, the development of dental caries relies greatly on the frequency of these occurrences.

The carious process can begin within days of a tooth erupting into the mouth if the diet is sufficiently rich in suitable carbohydrates, but may begin at any other time thereafter. The speed of the process is dependent on the interplay of the various factors described above but is believed to be slower since the introduction of fluoride. Compared to coronal smooth surface caries, proximal caries progress quicker and take an average of 4 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 cavitation within months of tooth eruption. This can occur, for example, when children continuously drink sugary drinks from baby bottles. On the other hand, it may take years before the process results in a cavity being formed, if at all.

**Other Risk Factors**

In addition to the four main requirements for caries formation, reduced saliva is also associated with increased caries rate since the buffering capability of saliva is not present to counterbalance the acidic environment created by certain foods. As a result, medical conditions that reduce the amount of saliva produced by salivary glands, particularly the parotid gland, are likely to cause widespread tooth decay. Medications, such as antihistamines and antidepressants, can also impair salivary flow.

The use of tobacco may also increase the risk for caries formation. Smokeless tobacco frequently contains high sugar content in some brands, possibly increasing the susceptibility to caries. Tobacco use is a significant risk factor for periodontal disease, which can allow 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 in comparison to enamel.

**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 progress, they 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 1-2% 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, 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 bacterial penetration, and the zone of destruction. 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.
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 µm) 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 hydroxypatite 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 the 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.

**Classification**

Caries can be classified by location, etiology, rate of progression, and affected hard tissues. When used to characterize a particular case of tooth decay, these descriptions more accurately represent the condition to others and may also indicate the severity of tooth destruction.

**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.

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

**Pit and Fissure Caries**

Pits and fissures are anatomic landmarks on a tooth where tooth enamel infolds creating such an appearance. Fissures are formed during the development of grooves, and have not fully fused (unlike grooves), thus possessing a unique linear-like small depression in enamel's surface structure, which would be a great place for dental caries to develop and flourish. Fissures are mostly located on the occlusal (chewing) surfaces of posterior teeth and palatal surfaces of maxillary
anterioir teeth. Pits are small, pinpoint depressions that are found at the ends or cross-sections of grooves. In particular, buccal pits are found on the facial surface 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 be common 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, 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 dentino-enamel junction (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.

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.

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".

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.

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. Recurrent caries, also described as secondary, is 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.
Signs and Symptoms

Dental explorer used for caries diagnosis.

Until caries progress, a person may not be aware of it. The earliest sign of a new carious lesion, referred as incipient decay, is the appearance of a chalky white spot on the surface of the tooth, indicating an area of demineralization of enamel. As the lesion continues to demineralize, it can turn brown but will eventually turn into a cavitation, a "cavity". The process before this point is reversible, but once a cavitation forms, the lost tooth structure cannot be regenerated. A lesion which appears brown and shiny suggests dental caries was 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 further, the cavitation 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 cause the tooth to hurt. The pain can be worsened by 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 which may become life-threatening, as in the case with Ludwig's angina.
Diagnosis

This preoperative photo of tooth #3, (A), reveals no clinically apparent decay other than a small spot within the central fossa. In fact, decay could not be detected with an explorer. Radiographic evaluation, (B), however, reveals an extensive region of demineralization within the dentin (arrows) of the mesial half of the tooth. When a burr was used to remove the occlusal enamel overlying the decay, (C), a large hollow was found within the crown and it was discovered that a hole in the side of the tooth large enough to allow the tip of the explorer to pass was contiguous with this hollow. After all of the decay had been removed, (D), the pulp chamber exposed and most of the mesial half of the crown was either missing or poorly supported.

Primary diagnosis involves inspection of all visible tooth surfaces using a good light source, dental mirror and explorer. Dental radiographs produced when X-rays are passed through the jaw and picked up on film or digital sensor, may show dental caries before it is otherwise visible, particularly in the case of caries on interproximal (between the teeth) surfaces. Large dental caries are often apparent to the naked eye, but smaller lesions can be difficult to identify. Unextensive dental caries were formerly found by searching for soft areas of tooth structure with a dental explorer. Visual and tactile inspection along with radiographs are still employed frequently among dentists, particularly for pit and fissure caries.
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 is a dental instrument used to remove decay carefully and 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.

In certain cases, root canal therapy may be necessary for the restoration of a
tooth. Root canal therapy, also called "endodontic therapy", 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.

**Prevention**

**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. A toothbrush can be used to remove plaque on most surfaces of the teeth except for areas between teeth. 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. 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.

**Dietary Modification**

For dental health, the 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. 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. 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.

**Other Preventive Measures**

The use of dental sealants is a means of prevention. Sealants are thin plastic-like coating applied to the chewing surfaces of the molars. This coating prevents the accumulation of plaque in the deep grooves and thus prevents the formation of pit and fissure caries, the most common form of dental caries.

Fluoride therapy is often recommended to protect against dental caries. It has been demonstrated that water fluoridation 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 fluoride 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.

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

**Gingivitis**

Gingivitis ("inflammation of the gums") (gingiva) around the teeth is a general term for gingival diseases affecting the gingiva (gums). As generally used, the term gingivitis refers to gingival inflammation induced by bacterial biofilms (also called
plaque) adherent to tooth surfaces.

**Causes**

Gingivitis is usually caused by bacterial plaque that accumulates in the small gaps between the gums and the teeth and in calculus (tartar) that forms on the teeth. These accumulations may be tiny, even microscopic, but the bacteria in them produce foreign chemicals and toxins that cause inflammation of the gums around the teeth. This inflammation can, over the years, cause deep pockets between the teeth and gums and loss of bone around teeth - an effect otherwise known as periodontitis.

Since the bone in the jaws holds the teeth into the jaws, the loss of bone can cause teeth over the years to become loose and eventually to fall out or need to be extracted because of acute infection. Regular cleanings (correctly termed periodontal debridement, scaling or root planing) below the gum line, best accomplished professionally by a dental hygienist or dentist, disrupt this plaque biofilm and remove plaque retentive calculus (tartar) to help prevent inflammation. Once cleaned, plaque will begin to grow on the teeth within hours. However, it takes approximately 3 months for the pathogenic type of bacteria (typically gram negative anaerobes and spirochetes) to grow back into the deep pockets and restart the inflammatory process. Calculus (tartar) may start to reform within 24 hours. Ideally, scientific studies show that all people with deep periodontal pockets (greater than 5mm) should have the pockets between their teeth and gums cleaned by a dental hygienist or dentist every 3-4 months.

People with a healthy periodontium (gums, bone and ligament) or people with gingivitis only require periodontal debridement every 6 months. However, many dental professionals only recommend periodontal debridement (cleanings) every 6 months, because this has been the standard advice for decades, and because the benefits of regular periodontal debridement (cleanings) are too subtle for many patients to notice without regular education from the dental hygienist or dentist. If the inflammation in the gums becomes especially well-developed, it can invade the gums and allow tiny amounts of bacteria and bacterial toxins to enter the bloodstream. The patient may not be able to notice this, but studies suggest this can result in a generalized increase in inflammation in the body and/or cause possible long term heart problems. Periodontitis has also been linked to diabetes, arteriosclerosis, osteoporosis, pancreatic cancer and pre-term low birth weight babies.

Sometimes, the inflammation of the gingiva can suddenly amplify, such as to cause a disease called Acute Necrotizing Ulcerative Gingivitis (ANUG), otherwise
known as "trench mouth." The aetiology of ANUG is the overgrowth of a particular type of pathogenic bacteria (fusiform-spirochete variety) but risk factors such as stress, poor nutrition and a compromised immune system can exacerbate the infection. This results in the breath being extremely bad-smelling, and the gums feeling considerable pain and degeneration of the periodontium rapidly occurs. This can be successfully treated with a 1-week course of Metronidazole antibiotic, followed by a deep cleaning of the gums by a dental hygienist or dentist and reduction of risk factors such as stress.

When the teeth are not cleaned properly by regular brushing and flossing, bacterial plaque accumulates, and becomes mineralized by calcium and other minerals in the saliva transforming it into a hard material called calculus (tartar) which harbors bacteria and irritates the gingiva (gums). Also, as the bacterial plaque biofilm becomes thicker this creates an anoxic environment which allows more pathogenic bacteria to flourish and release toxins and cause gingival inflammation. Alternatively, excessive injury to the gums caused by very vigorous brushing may lead to recession, inflammation and infection. Pregnancy, uncontrolled diabetes mellitus and the onset of puberty increase the risk of gingivitis, due to hormonal changes that may increase the susceptibility of the gums or alter the composition of the dentogingival microflora. The risk of gingivitis is increased by misaligned teeth, the rough edges of fillings, and ill fitting or unclean dentures, bridges, and crowns. This is due to their plaque retentive properties. The drug phenytoin, birth control pills, and ingestion of heavy metals such as lead and bismuth may also cause gingivitis.

The sudden onset of gingivitis in a normal, healthy person should be considered an alert to the possibility of an underlying viral aetiology, although most systemically healthy individuals have gingivitis in some area of their mouth, usually due to inadequate brushing and flossing.

**Symptoms**

The symptoms of gingivitis are as follows:

- Swollen gums
- Mount sores
- Bright-red, or purple gums
- Shiny gums
- Gums that are painless, except when pressure is applied
- Gums that bleed easily, even with gentle brushing, and especially when flossing.
- Gums that itch with varying degrees of severity dark red gums that are
usually pink and bleeding
- Faint blue tearing from eyes

Prevention

Gingivitis can be prevented through regular oral hygiene that includes daily brushing and flossing (mouth wash optional). Rigorous plaque control programs along with periodontal scaling and curettage also have proved to be helpful.

Researchers analyzed government data on calcium consumption and peridontal disease indicators in nearly 13,000 people representing 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.

Research says the relationship between calcium and gum disease is likely due to calcium's role in building density in the alveolar bone that supports the teeth.

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.

Hypervitaminosis A, otherwise known as excess Vitamin A in the diet has also been linked to gingivitis in cats and dogs. Whether this is applicable to humans though, remains unclear.

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.

Treatment

A dentist or dental hygienist will perform a thorough cleaning of the teeth and gums; following this, persistent oral hygiene is necessary. The removal of plaque is usually not painful, and the inflammation of the gums should be gone between one
and two weeks. A gargling of brine water also helps. Oral hygiene including proper brushing and flossing is required to prevent the recurrence of gingivitis. Anti-bacterial rinses or mouthwash, in particular chlorhexidine digluconate 0.2% solution, may reduce the swelling and local mouth gels which are usually antiseptic and anaesthetic can also help.

**Oral Ulcer**

An oral ulcer (from Latin ulcus) is the name for the appearance of an open sore inside the mouth caused by a break in the mucous membrane or the epithelium on the lips or surrounding the mouth. The types of oral ulcers are diverse, with a multitude of associated causes including: physical or chemical trauma, infection from microorganisms or viruses, medical conditions or medications, cancerous and sometimes nonspecific processes. Once formed, the ulcer may be maintained by inflammation and/or secondary infection. Two common oral ulcer types are aphthous ulcers (canker sores) and cold sores. Cold sores are caused by the herpes simplex virus.

**Causes**

There are many processes which can lead to ulceration of the oral tissues. In some cases they are caused by an overreaction by the body's own immune system. Factors that appear to provoke them include stress, fatigue, illness, injury from accidental biting, hormonal changes, menstruation, sudden weight loss, food allergies and deficiencies in vitamin Bl2, iron and folic acid. Oral ulcers are also a common result of ceased cigarette smoking, affecting about two out of five quitters. Some drugs, such as nicorandil, have also been linked with oral ulcers. Some recreational drugs cause mouth ulcers.
**Trauma - Minor Physical Injuries** - Trauma to the mouth is a common cause of oral ulcers. A sharp edge of a tooth, accidental biting (this can be particularly common with sharp canine teeth), sharp or abrasive food (particularly if left overnight), poorly fitting dentures, dental braces or trauma from a tooth brush may injure the mucosal lining of the mouth resulting in an ulcer. These ulcers usually heal at a moderate speed if the source of the injury is removed (for example, if poorly fitting dentures are removed or replaced).

**Sugar Injuries** - Eating large amounts of sugar can also lead to oral ulcers. These are not a general worry because these subside within a day or two unless large volumes of sugar continue to be present in a person's diet.

**Chemical Injuries** - Chemicals such as aspirin or alcohol that are held or that come in contact with the oral mucosa may cause tissues to become necrotic and slough off creating an ulcerated surface. Sodium lauryl sulfate (SLS), one of the main ingredients in most toothpastes, has been implicated in increased incidence of oral ulcers.

**Infection** - Viral, fungal and bacterial processes can lead to oral ulceration. One way to cause oral ulceration by this is to touch your chapped lips without washing your hands first.

**Viral** - The most common is Herpes simplex virus which causes recurrent herpetiform ulcerations preceded by usually painful multiple vesicles which burst. Herpes Zoster (shingles), Varicella Zoster (chicken pox), Coxsackie A virus and its associated subtype presentations, are some of the other viral processes that can lead to oral ulceration. HIV creates immunodeficiencies which allow opportunistic infections or neoplasms to proliferate.

**Bacterial** - Bacterial processes leading to ulceration can be caused by Mycobacterium tuberculosis (tuberculosis) and Treponema pallidum (syphilis).

Opportunistic activity by combinations of otherwise normal bacterial fauna, such as aerobic streptococci, Neisseria, Actinomyces, spirochetes, and Bacteroides species can prolong the ulcerative process.

**Fungal** - Coccidioides immitis (valley fever), Cryptococcus neoformans (cryptococcosis), Blastomyces dermatitidis ("North American Blastomycosis") are some of the fungal processes causing oral ulceration.

**Protozoans** - Entamoeba histolytica, a parasitic protozoan is sometimes known to cause mouth ulcers through formation of cysts.
**Immune system** - Many researchers view the causes of aphthous ulcers as a common end product of many different disease processes, each of which is mediated by the immune system.

Aphthous ulcers are thought to form when the body becomes aware of and attacks chemicals which it does not recognize. The presence of the unrecognized molecules garners a reaction by the lymphocytes, which trigger a reaction that causes the damage of an oral ulcer.

**Immunodeficiency** - Repeat episodes of mouth ulcers can be indicative of an immunodeficiency, signaling low levels of immunoglobulin in the oral mucous membranes. Chemotherapy, HIV, and mononucleosis are all causes of immunodeficiency with which oral ulcers become a common manifestation.

**Autoimmunity** - Autoimmunity is also a cause of oral ulceration. Mucous membrane pemphigoid, an autoimmune reaction to the epithelial basement membrane, causes desquamation/ulceration of the oral mucosa.

**Allergy** - Contact with allergens can lead to ulcerations of the mucosa.

**Dietary** - Vitamin C deficiencies may lead to scurvy which impairs wound healing, which can contribute to ulcer formation. Similarly deficiencies in vitamin Bl2, iron, zinc and folic acid have been linked to oral ulceration.

A common cause of ulcers is Celiac disease, in which case consumption of wheat, rye, or barley can result in chronic oral ulcers. If gluten sensitivity is the cause, prevention means following a gluten-free diet by avoiding most breads, pastas, cakes, pies, cookies, scones, biscuits, beers, etc. and substituting gluten-free varieties where available. Artificial sugars, such as those found in diet cola and sugarless chewing gum, have been reported as causes of oral ulcers as well.

**Cancer** - Oral cancers can lead to ulceration as the center of the lesion loses blood supply and necroses.

**Medical Conditions** - The following medical conditions are associated with mouth ulcers: Behcet's disease, Bullous pemphigoid, Celiac disease, Crohn's disease, Gingivo stomatitis, Leukoplacia, oral lichen planus, Lupus erythematosus, Neutropenia, Oral thrush, and Ulcerative colitis.

**Prevention**

The majority of the types of ulceration require treatment of the underlying cause of the oral ulceration for successful prevention; controlling imbalances in vitamins and minerals related to ulceration, managing or restricting the disease processes has
shown to reduce the ulcerative process. For trauma related cases, avoiding the offending source will prevent ulceration, but since such trauma is usually accidental, this type of prevention is not usually practical.

Individuals who have a high incidence of opportunistic bacterial infections subsequent to an accidental oral injury (biting etc.) can prevent the injury from becoming infected by directly bathing the wound with an anti-bacterial mouthwash for one minute every 12 hours for 2 days; it is important to use a small vessel to contain the solution as most antibacterial mouth washes that remain in the mouth for a full minute will have detrimental effects such as a prolonged impairment to the sense of taste and the potential loss of otherwise desirable flora. Quantities around 1ml are more than sufficient. Ideally, the first treatment should occur within 3 hours.

**Treatment**

Treatments based on antibiotics and steroids are reserved for severe cases, and should be used only under medical supervision. Some doctors may also prescribe a local anaesthetic, such as lidocaine or benzocaine, for cases of multiple or severe oral ulcers.

Some people benefit from using an over-the-counter topical gel, like Bonjela, which may contain substances such as choline salicylate to help reduce the pain and inflammation associated with oral ulcers.

Also, putting baking soda directly on the sore and taking L-Lysine has been shown to help with healing and pain. Holding milk in the mouth over the sore can have a temporary soothing effect upon the sore.

Brushing the teeth and rinsing the mouth with Euthymol toothpaste provides relief for a period of a few hours.

Hydrogen peroxide applied locally reduces infection and often improves healing (recommended by Mayo Clinic for cancer patients who’s treatment increase the occurrence of cold sores).

Licorice extract has been used and is now commercially available as "CankerMelts".
Oral Cancer

Oral cancer is any cancerous tissue growth located in the mouth. 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 histologi 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. Far and away the most common oral cancer is squamous cell carcinoma, originating in the tissues that line the mouth and lips. Oral or mouth cancer most commonly involves the tissue of the lips or the tongue. It may also occur on the floor of the mouth, cheek lining, gingiva (gums), 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.

Known Risk Factors

In 2008, in the US alone, about 34,000 individuals will be 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.

All cancers are diseases of the DNA in the cancer cells. Oncogenes are activated as a result of mutation of the DNA. The exact cause is often unknown. Risk factors that predispose a person to oral cancer have been identified in epidemiological studies.

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 19 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.

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

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.

**Human Papilloma Virus**, (HPV) particularly versions 16 and 18 (there are over 100 varieties) is a known risk factor and independent causative factor for oral cancer. 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 females slightly outnumber males. Recent research from Johns Hopkins indicates that HPV is the primary risk factor in this new population of oral cancer victims. HPV16/18 is the same virus responsible for the vast majority of all cervical cancers and is the most common sexually transmitted infection in the US.

**Symptoms**

- Mouth sore that does not heal
- Mouth sore that bleeds spontaneously
- Velvety white, red or speckled (red and white) patch in the mouth that is persistent
- Hard, raised lesion (lump), crusts, eroded areas on the lips, gums, or other areas inside the mouth
- Persistent pain in the mouth
- Difficulty chewing, swallowing, speaking, or moving the tongue
Signs and Tests

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, particularly if the cancer is on the tongue.

While a dentist, physician or other medical professional may suspect a particular lesion is malignant, the only definitive method for determining this 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.

Treatment

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 is often used in conjunction with surgery, or as the definitive radical treatment, especially if the tumour is inoperable. 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.

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 I 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 therapists 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 seldom 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.

Complications

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