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SALIVARY FLOW AND DENTAL HEALTH

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BDS Bombay

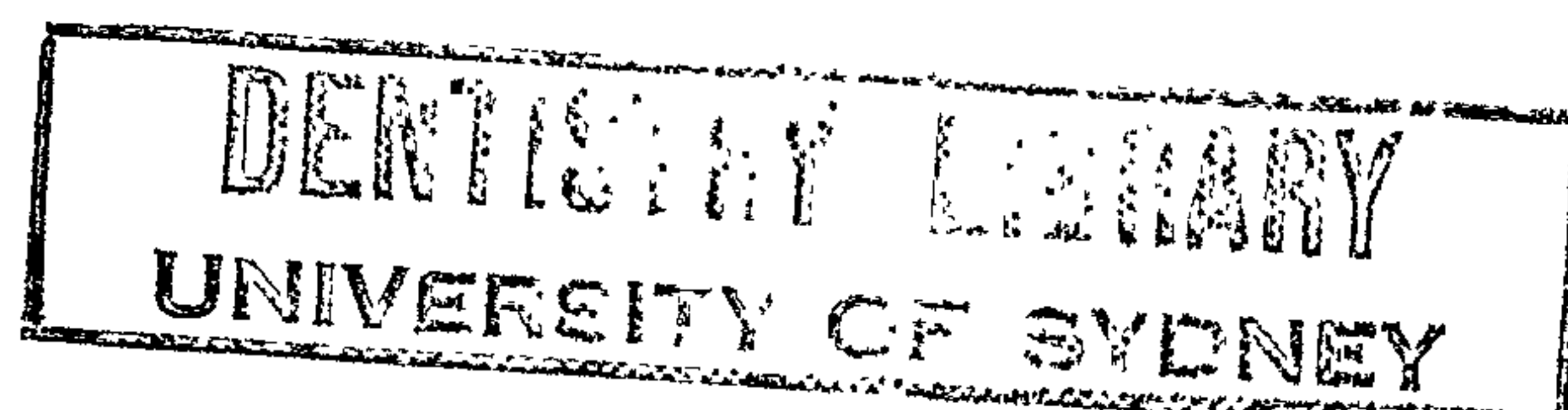
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SUMMARY

This thesis reviews the physiological aspects of salivary glands, dental caries, dental plaque, xerostomia, salivary flow and dental health, salivary clearance and its effect on oral health.

Saliva is produced by three paired exocrine organs the parotid, submandibular and sublingual glands. Together they synthesise and secrete more than 90% of saliva. Seven percent is derived from the minor salivary glands. When present, saliva enables people to enjoy some of life's more serene pleasures. The delicate sensation of taste, the joy of eating and the exquisite sound of the human voice. Life goes on in the absence of saliva but the quality of life diminishes. The importance of saliva composition lies in the ways saliva and its elements supports oral functions. The mouth has developed to facilitate communication and alimentation. These functions may be compromised in the absence of normal salivation. The patients with diminished secretions have difficulty speaking, chewing, forming a food bolus and swallowing. Additionally there is a rapid and substantial increase on caries and mucosal infection. Taste and soft tissue complaints are also more frequent.

Root caries is the oldest recorded form of caries among human populations. But inspite of this it is only in the last fifteen years that interest has been reawakened in this particular manifestation of dental disease. Surveys carried out in a number of industrialised countries have pointed to a substantial decline in edentulousness in adults, an increasing life expectancy and increasing prevalence of root caries. With some justification root caries, can be regarded as a separate disease entity; with respect to its location, its microbiology and the pattern of its occurrence.

Plaque is a normal part of the oral microflora and bacteria maintain a commensal relationship with the host. This relationship makes periodontal disease difficult to control. Normal flora will always re-establish itself. However, several authors have suggested that a

reservoir of calcium phosphate and/or fluoride in dental plaque may be beneficial by maintaining the saturation with respect to enamel mineral, especially when plaque pH decreases.

The problem of Xerostomia is increasing as the proportion of the population of the elderly increases. Xerostomia is not a result of old age itself but is usually iatrogenic and potentially preventable. It is important to find out if the patient is suffering from a deficiency of gland tissue or an under functioning of the salivary glands. If it is the former then saliva substitutes are needed. Gum chewing between meals by students resulted in raising unstimulated saliva flow rates especially among slow secretors, an effect still detectable several weeks after gum chewing had finished, which implies some structural effect on the glands. This suggests that exercising the salivary glands by chewing gum might increase the resulting flow in xerostomic patients. Stimulation of residual gland activity by the use of sugar-free sweets especially chewing gum can have an immediate benefit in stimulating saliva perhaps a longer term benefit in encouraging salivary gland function. Unstimulated flow rates vary widely between subjects and those with low rates do not always have symptoms of dry mouth. The main stimulus to salivation is the sensation of taste (particularly acid). Stimulated saliva is better able because of changes in its composition to prevent demineralisation and to favour remineralisation than is unstimulated saliva. Water is a salivary substitute, but to produce more sophisticated salivary substitutes, the viscoelastic properties of saliva should be incorporated. The use of high molecular weight polyethylene oxide solution could be considered. There are some commercial products containing animal mucin and others using carboxymethylcellulose to make a viscous solution. These substitutes have a role to play in managing dry mouth but their utility is limited.

The clearance pattern of a substance is very important, when trying to estimate the oral bio-availability of a specific substance. There are

many factors involved, and clearance patterns for substances like sucrose, fluoride, chloride and chlorhexidine are quite dissimilar. Individual variations in clearance patterns can be both harmful and beneficial. Mechanisms of salivary clearance of carbohydrates from food, acids from plaque and therapeutic substances (for example fluoride) help to explain differences in oral health between different individuals and between different sites within a single mouth.

Maiwald and Beu (1990) suggest that prevention of oral diseases must focus not only on completely eliminating the factors identified as the cause but rather on reducing them to a level with which our inherent defence mechanisms can cope. It would be beneficial to consider alternatives that increase resistance and defence performance such as the use of the fluorides or boosting the rinsing and buffering action of saliva through additional steps like chewing gum. It appears that chewing gum may have a hydrodynamic pumping effect on plaque which might not be expected with other mechanical stimuli (for example sucking a stone), or with a combined mechanical and chemical stimuli (for example, sucking on a lozénge). Products with hydrodynamic effect might be superior to nonhydrodynamic mechanical and chemical stimuli. Levine R (1989) suggests that the condition with most patients with dry mouth is drug induced. These patients are already taking a large number of drugs, so a nonpharmacological salivary stimulant might be preferred.

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DEDICATION

TO

CARL and OLAV D'SOUZA

WITH LOVE AND AFFECTION

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1. INTRODUCTION

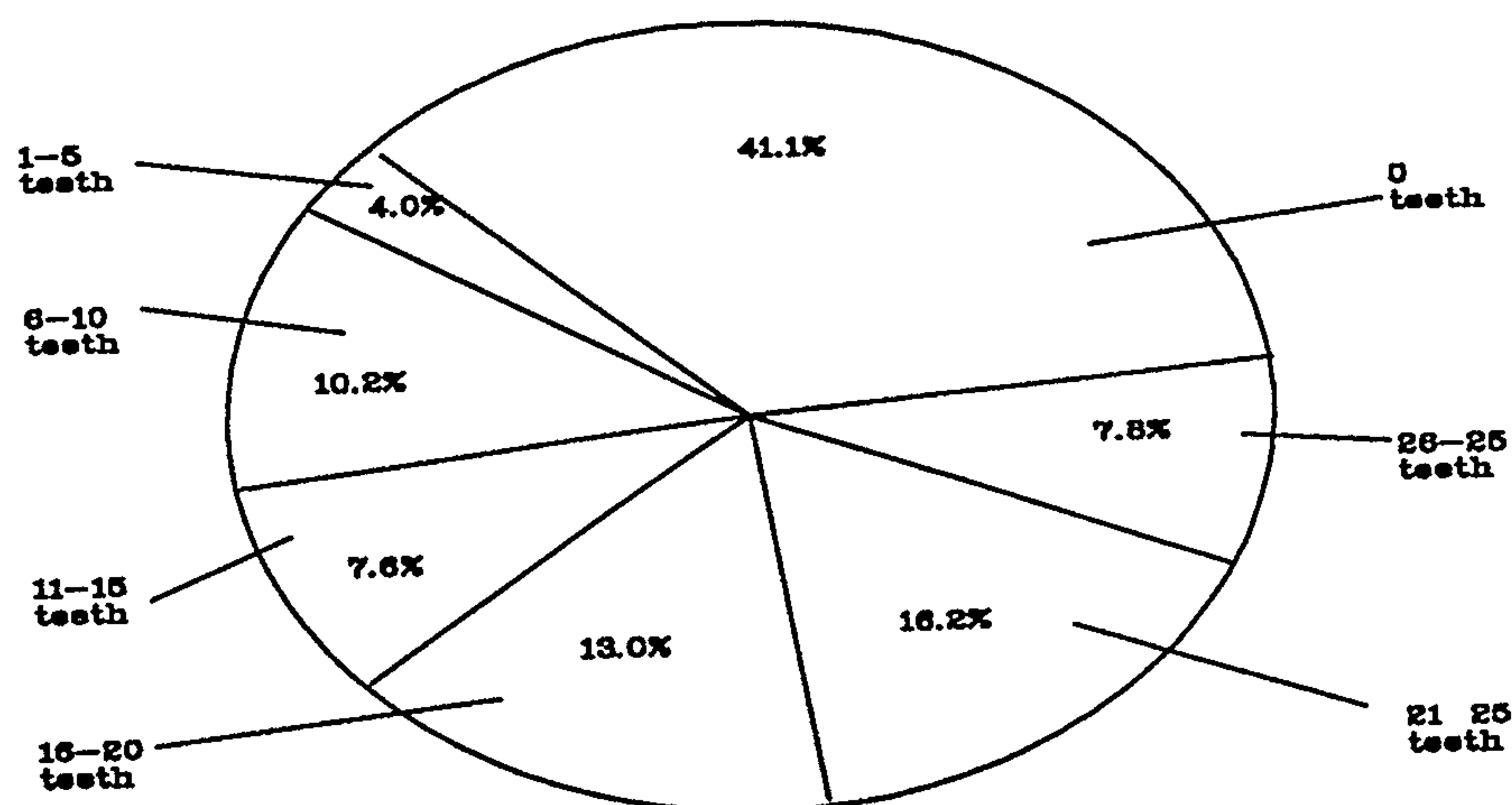
In 1658 Thomas Wharton wrote of the duct which now bears his name. He wrote that a canal opens into the mouth and issues salivary fluid and the concept of salivary secretion was born. It is these secretions that have been referred to as "aqua-vita" (water of life) of the mouth. Saliva is a unique biological fluid of enormous complexity. It plays a principal role in the protection of oral tissues in the alimentary process and in speech. The protective function primarily served by its ubiquitous basal secretions. The food related functions are served by the process of mastication, deglutation, gustation and olfaction.

One hundred years ago W.D. Miller described the basic mechanism of caries etiology, which was the foundation of our understanding today. While Miller's experiments showed saliva is an essential in the pathogenesis of caries, a little more than twenty years later Head recognised the role of saliva in protecting enamel and repairing the effect of the caries attack. This concept was taken further by Bunting & Ricket, who in 1918 described the post eruptive maturation of enamel under the influence of saliva (Levine R 1989). The most important properties of saliva from the caries point of view namely is its buffering power and its saturation with calcium phosphate. These factors increase as the pH are raised and are reduced when the pH falls sufficiently (Jenkins 1990). Saliva is of crucial importance in providing a means of buffering the acids produced in plaque from food debris. In supplying the inorganic ions (calcium phosphate, hydroxyl, fluoride) required to remineralize incipient lesions and in diluting and washing away sugars from around the teeth. Stimulated saliva is more effective than unstimulated saliva. Salivary flow falls during sleep and so intake of food and drinks should be avoided just before retiring. Caries occurs when the chemical conditions at the tooth surface favour demineralisation over remineralisation. That is when the concentration of calcium, phosphate and hydroxyl ions are too low to prevent the apatite-like crystals of the tooth dissolving. The hydrogen ion concentration largely determines the demineralisation forces. While even low level of fluoride ion greatly favour

remineralisation. Doctor John Featherstone of the Eastman Dental Center, Rochester, NY, pointed out "a little bit of demineralisation provided even low levels of fluoride are present may be good for you" by replacing a most soluble with a less soluble enamel crystal. This is believed to be the main way in which fluoride strengthens the teeth. In addition, fluoride reduces demineralisation provided it is present in fluoride ions in solution in the tooth environment (Edgar 1990). Saliva plays a crucial role here by acting as a vehicle for fluoride derived from tooth paste and other sources.

The fall in dental caries in western industrialised countries over the last decade have tended to induce some complacency and convey the impression that the caries problem is solved and that further research on saliva should have a low priority (Jenkins 1990). This attitude overlooks several factors. Firstly, the decline seems now to have stabilised in the western industrialised countries, leaving about half the previous incidents still requiring attention. Secondly, with increasing tooth retention the total number of decayed and filled enamel surfaces is not decreasing as yet in the 35 to 65 year category. Figure 1 shows a pie chart depicting the remaining number of teeth of US older adults.

Figure 1 Remaining teeth of older adults.
Source: Berkey (1988).



In addition, the increased gingival recession that accompanies tooth retention places vulnerable surfaces at risk. In persons who are above fifty five year old, root caries is increasing (Mandel 1989) as shown in Figure 2.

Figure 2 Decayed and filled root surfaces. Oral health of US adults, NIDR Survey, 1985.
Source: Mandel (1989).



Dentists can anticipate greater attention to be given to secondary and recurrent decay and a greater concern for root caries as the population ages. Thirdly, in developing countries with a population shift from rural to urban areas, a westernisation of diet and a greater availability of sugar the caries increase has been rapid and profound. Until public health programs can be instituted and dental care appreciably increased the caries problem in these countries will continue to grow (Mandel 1989).

Xerostomia is a clinical manifestation of salivary glands dysfunction but in itself does not represent a disease entity. It has been recognised for over a hundred years having first being described by Bartley in 1868. The condition presents as a dryness often accompanied by a burning sensation in the oral mucosa. Eating and sometimes speech is difficult and the sensation of taste is reduced. The mucosa is often pale and atrophic with a dry and sticky feel. The tongue may be inflamed and show atrophy of the papilla together with fissuring and cracking. Infection is a direct cause of xerostomia, however, once the condition is established the oral mucosa especially that of the tongue may become prone to infection with the opportunist organisms like candida albicans, introducing an added complication to the management of the disorder (Levine R 1989).

The problem of Xerostomia is increasing as the proportion of the population of the elderly increases. Xerostomia is not a result of old age itself but is usually iatrogenic and potentially preventable. It is important to find out if the patient is suffering from a deficiency of gland tissue or an under functioning of the salivary glands. If it is the former then saliva substitutes are needed. Gum chewing between meals by students resulted in raising unstimulated saliva flow rates especially among slow secretors, an effect still detectable several weeks after gum chewing had finished, which implies some structural effect on the glands (Jenkins, Edgar 1989). This suggests that exercising the salivary glands by chewing gum might increase the resulting flow in xerostomic patients. Stimulation of residual gland activity by the use of sugar-free sweets especially chewing gum can have an immediate benefit in stimulating saliva perhaps a longer term benefit in encouraging salivary gland function (Edgar 1989).

Clinicians should be aware of the nature of saliva, the role that it plays in the mouth and the consequence of any failure in its production. Newbrun referred to a change from an earlier view of

saliva as an obstacle to the placement of restorations ("Saliva be dammed") towards one in which an evaluation of salivary function could become an everyday part of patient evaluation and history taking much as a general medical practitioner would measure blood pressure. By taking the relevant history, general dental practitioners can identify patients who may have reduced salivary flow and recommend procedures to combat their potentially greater risk of caries. People who complain of dry mouth do not necessarily have a very low flow rate and conversely those with a low unstimulated flow do not always complain of dry mouth. It is more significant to know whether or not flow rate has changed adversely in a particular individual (Edgar 1989). It would therefore be advantageous if dentists had a measure of flow rates to diagnose xerostomia accurately.

AIMS

The aims of the thesis are:

To highlight the complexity and unique qualities of saliva and to stress the essential role of saliva in controlling the environment of the teeth and the benefits to be gained by stimulation.

To discuss ways of identifying patients who have reduced salivary flow and the need to undertake measurement of salivary flow rates as a part of routine patient screening.

To discuss salivary stimulants.

2. PHYSIOLOGICAL ASPECTS OF SALIVARY GLANDS

2.1 LOCATION OF SALIVARY GLANDS

Saliva is produced by three paired exocrine organs the parotid, submandibular and sublingual glands. Together they synthesise and secrete more than 90 per cent of saliva. Seven percent is derived from the minor salivary glands. The parotid glands are located under and ventral to the ears and are the glands that become characteristically inflamed in patients with mumps infection. The submandibular glands are located at the midpoint of the mandible. The sublingual glands are located under the tongue in the floor of the mouth. In addition to the major glands numerous minor salivary glands are present through out the mouth. These are on the inner aspects of the lips the soft palate the buccal mucosa and the dorsal surface of the tongue.

2.2 STRUCTURE OF SALIVARY GLANDS

Salivary glands consist of acinar units that produce most of the salivary products and fluid, and the ducts that carry the acinar secretions to the mouth. The acinar cells are pyrimidal in shape and contain large numbers of secretory granules which are located at the apical aspect of the cell. The granules contain the macromolecules that are released into the saliva when they receive the proper cell signal. Simultaneously with the release of the granule contents the acinar cells secrete fluid to aid in carrying the secreted macromolecules into the ducts and into the mouth. The ducts not only carry the acinar secretions to the mouth but also secrete certain salivary macro-molecules and resorb and secrete various electrolytes. However only the acinar units, not the ducts, produce salivary fluid.

2.3 COMPOSITION OF SALIVA

Saliva consists of two major components. The macromolecules and the salivary fluid. In addition to these substances produced by the salivary glands saliva contains serum products and components that are transported into the saliva including drugs and viruses. Whole saliva is the total fluid content of the mouth it is colourless, opalescent, slightly foamy and viscid solution. It contains many non salivary constituents including serum products and blood cells (gingival, crevicular fluid and the mucosa), bacteria and bacterial products, food debris and bronchial secretions. The parotid glands produce more watery proteinaceous secretion compared to the submandibular sublingual saliva which is more viscous with lower protein content. The latter secretions are considered together because they empty onto the mouth floor through a common major excretory duct. The minor gland secretions are extremely mucinous with a high secretory IgA concentration. In an unstimulated state salivary output is low with stimulation such as mastication, output increases as much as ten to twenty times with significant composition alterations. A list of the proteins and non protein constituents of saliva is given in Table 1.

Table 1 Protein and nonprotein constituents of saliva.
Source: Fox PC (1989) .

Albumin	Histatins	Nonspecific buffers
Ammonia	IgA (sIgA)	Phosphatases
Amylase	IgG	Phosphorus
Bicarbonate	IgM	Potassium
β -glucuronidase	Iodine	Proline-rich proteins
Calcium	Kallikrein	Ribonucleases
Chloride	Lactoferrin	Serum proteins(trace)
Cretinine	Lactoperoxidase	Sialic acid
Cystatins	LDH	Sodium
Esterases	Lysozyme	Statherin
Fluoride	Magnesium	Sulfates
Glucose	Mucins	Thiocyanate
Gustin	Nitrogen	Uric acid

Whole saliva is 99 per cent water and includes a mixture of inorganic ions. The major ones being Na^+ , K^+ , Cl^- , HCO_3^- , Ca^{++} , Mg^{++} , HPO^- and minor ones including I^- and SCN^- and F^- . The resting pH varies

between 6.7 and 7.4 for whole saliva, while that of pure parotid secretion which is easiest to obtain varies from 5.2 to 6.8. Saliva also contains a wide array of organic molecules. Some are simple proteins such as enzymes, albumin and free amino acids. But the bulk of the organic component is made up of complex glycoproteins, the mucins. These are important macromolecules which make up the protein backbone of saliva (Levine R 1989).

The presence of excessive amounts of foam in whole saliva and patients complaints that their saliva feels rope-like often indicates disease (Sreebny 1989). The viscosity of whole saliva depends on the ratio of the contribution made to it by the various glands. Schneyer observed that the respective viscosities for stimulated saliva from the parotid, the submandibular and the sublingual were 1.5, 3.4 and 3.4 centipoise (Schneyer 1955). Saliva also demonstrates an ability to be stretched into a thread. This property is referred to as "spinnbarkeit" and is measured in mm. The spinnbarkeit ratios for the parotid, submandibular, sublingual and minor salivary glands are 1:2.4:10:45 (Codipilly et al 1989). This indicates that the minor salivary gland secretions are 45 times as viscid as those of the parotid glands and 4.5 times as viscid as those from the sublingual. The mucous secretion coat and protect the oral mucus membrane.

2.3.1 Factors affecting salivary gland composition

Many factors can affect the composition of saliva. A list of the factors affecting salivary composition is given in Table 2.

Table 2 Factors affecting salivary composition.
Source: Edgar WM, O'Mullane DM (1990).

Species	Hormones
Glandular source	Pregnancy
Flow rate	Genetic polymorphism
Duration of stimulation	Antigenic stimulus.
Previous stimulation	Exercise
Biological rythms	Drugs
Nature of stimulus	Various diseases
Plasma composition (diet)	

The relative contributions from various glands: The parotid gland normally contributes 20 per cent of the total volume of unstimulated saliva secretion, while the submandibular contributes 65 per cent, the sublingual 7 to 8 per cent and the minor mucous glands 7 to 8 per cent. At high flow rates the parotid becomes the dominant gland contributing about 50 per cent to the whole saliva secretion. Since the parotid gland secretes calcium at a lower concentration than the submandibular gland, the calcium content of whole saliva is reduced at high flow rates. All amylase in saliva is produced by the parotid glands. Blood groups are mainly derived from the minor mucous glands. Appreciable differences occur in the proportions of its constituents according to the source of the saliva. For instance parotid saliva is rich in amylase and phosphates and poor in mucous and calcium compared with submandibular and sublingual secretions. The minor mucous glands secrete saliva which contain phosphate and have chloride as their main anions.

The flow rate: The main factor affecting the composition of saliva is its flow rate. It is subject to physiological regulation and has a marked effect on the composition of saliva. The flow is increased not only by a direct stimulation of taste and olfactory receptors but also by the other forms of oral stimulation, such as those experiencing during dental treatment. If the flow rate is increased above the unstimulated rate the sodium, calcium, chloride, bicarbonate, protein concentrations and pH increases whereas phosphate magnesium, urea concentration decreases and potassium shows little change. At very high rates of flow the composition of saliva which is normally hypotonic tends to approach that of plasma (Cole, Eastoe 1977).

The nature of stimulus: This also has an effect on the composition, but mainly because of the effect of different stimuli on the rate of flow. However, using constant flow conditions the effect of the four basic taste stimuli - salt, acid, bitter and sweet- on salivary composition were examined. (Edgar, O'Mullane 1990). It was found that the type of stimulus used had virtually no effect on the electrolyte composition, while the taste of salt stimulated the highest protein content.

The duration of stimulus: Saliva collected at a constant flow rate for two minutes will have a different composition from saliva collected for ten to fifteen minutes. The composition will vary depending on whether the gland has been stimulated within the last hour, the time of day, and so on.

2.4 FUNCTION OF SALIVA

When present, saliva enables people to enjoy some of life's more serene pleasures. The delicate sensation of taste, the joy of eating and the exquisite sound of the human voice. Life goes on in the absence of saliva but the quality of life diminishes.

2.4.1 Oral functions

The importance of saliva composition lies in the ways saliva and its elements supports oral functions. The mouth has developed to facilitate communication and alimentation. These functions may be compromised in the absence of normal salivation (Fox 1985). The patients with diminished secretions have difficulty speaking, chewing, forming a food bolus and swallowing. Additionally there is a rapid and substantial increase on caries and mucosal infection. Taste and soft tissue complaints are also more frequent. A list of major functions of saliva and the components that support them is given in Table 3.

Table 3 **Functions of saliva.**
Source: Fox (1989).

Function	Salivary component involved
Lubrication	Mucins, Proline-rich protein, H ₂ O.
Antimicrobial	Lactoferrin, Lysozyme, Lactoperoxidase, sIgA, Mucins, Histatins, Cystatins, proline-rich proteins.
Remineralization	Ca, P, Pl, Statherin, Anionic proline-rich proteins.
Cleansing	H ₂ O.
Buffering	HCO ₃ , PO ₄ .
Digestive	Amylase, Lipase, Proteases, H ₂ O, Nucleases, Mucins, Gustin.
Mucosal Integrity	H ₂ O, Electrolytes, Mucins.

Saliva is a lubricant acting between soft tissues; between the dentition and soft tissues; and between the soft tissues and food. In addition to water, mucin and proline-rich glyco proteins contribute to the lubrication properties of saliva. There are numerous anti-microbial systems in saliva; all contribute to modulating the oral flora and protecting the oral tissues. The salivary layer on teeth and mucosa may create a surface charge and influence microbial attachment. Mucin and proline-rich polypeptides, as well as electrolytes govern microbial attachments. secretory IgA binds to bacteria and fungi leading to aggregation in the fluid phase. IgA also may lead to compliment-mediated killing. Histidine-rich poly peptides (histatins) are a group of cationic agents that bind to cell walls, disrupting bacteria and fungi. They have been shown to effect candida albicans and may play an important role in preventing oral candidiasis (Fox 1989). Cysteine-rich polypeptides (cystatins) are known to have an antiviral action in herpes viruses. Fox et al (1988) did a study on the inhibition of HIV-1 infectivity by saliva. This study demonstrated the ability of HIV-1 to infect human peripheral blood lymphocytes can be completely inhibited by incubation of virus in unstimulated whole saliva in vitro for one hour at 37 degrees C. In all subjects studied, incubation of virus in submandibular saliva resulted in a decided reduction in infectivity. This finding demonstrated that the inhibitory activity found in whole saliva is secreted by major salivary glands and does not exist in whole saliva simply because of leakage of serum components through oral lesions or through gingival crevice. Results of this study demonstrated that this antiviral activity is likely to have a major role in the oral defence against AIDS.

Lysozyme attacks bacterial cell wall bonds and increases permeability. It also promotes aggregation of Candida albicans. Irons are known to interfere with the action of lysozyme. Enzyme activity is enhanced when lactoferrin chelates iron. Lactoferrin also limits bacterial growth by reducing iron availability. The peroxidase-thiocyanate

-halide system generates active oxidising species upon activation by microbial contact, and exerts an effective killing mechanism.

Remineralization of the tooth is enhanced through supersaturation in saliva with respect to enamel, minerals, calcium and phosphate, is maintained because of the presence of specific protein. Proline-rich salivary proteins and tyrosine-rich protein called statherin which are thought to inhibit spontaneous calcium and phosphate from the saliva (Peretz 1990). Saliva provides a constant supply of ions to the enamel surface. It also contains buffers which resist pH changes. Saliva dissolves and disperses food particles and serves as a clearing agent for soluble food stuffs. Saliva can be looked upon as "the blood-stream of the teeth" (Peretz 1990).

2.4.2 Cleansing and digesting

Saliva by virtue of its water content, acts as a mechanical cleansing agent. It also possesses buffering properties because of its bicarbonate content. However, both phosphate and macromolecules can contribute to buffering to a lesser extent.

A range of digestive enzymes are present in saliva including amylase, proteases, lipase, and nucleases. Mucins are important for the formation of the food bolus. For a substance to be tasted, it must be in an aqueous solution. Water of saliva functions as a solvent. There is some preliminary work to suggest that other salivary factors may be involved in taste bud integrity but this remains to be confirmed (Fox 1989). It has been shown that the normal gustatory functions can be preserved in the absence in salivary output (Fox 1989).

Saliva probably contributes to the mucosal integrity by virtue of the hydrated surface film. Little is known about the factors responsible but mucin and water are assumed relevant. In addition, the electrolytic function of saliva can influence the charge of these

protein molecules. Saliva can also contain intrinsic mucosal protective factors yet to be identified.

When saliva function is impaired by disease or medications, it can have profound effect on oral health and functions. It has been demonstrated that complaints of oral dryness, the most common symptom of salivary dysfunction, do not reliably reflect actual gland performance (Fox 1990). Some individuals may have significant decline in salivary output and offer no subjective complaints, while others have no measurable hypofunction, yet are markedly symptomatic. This has led to confusion when studies utilize only subjective criteria for evaluation of salivary capabilities. It is essential that salivary gland function be assessed in a careful, standardised, and objective manner. Such methods of evaluation are available and should be used to classify salivary functional status.

3. CARIES

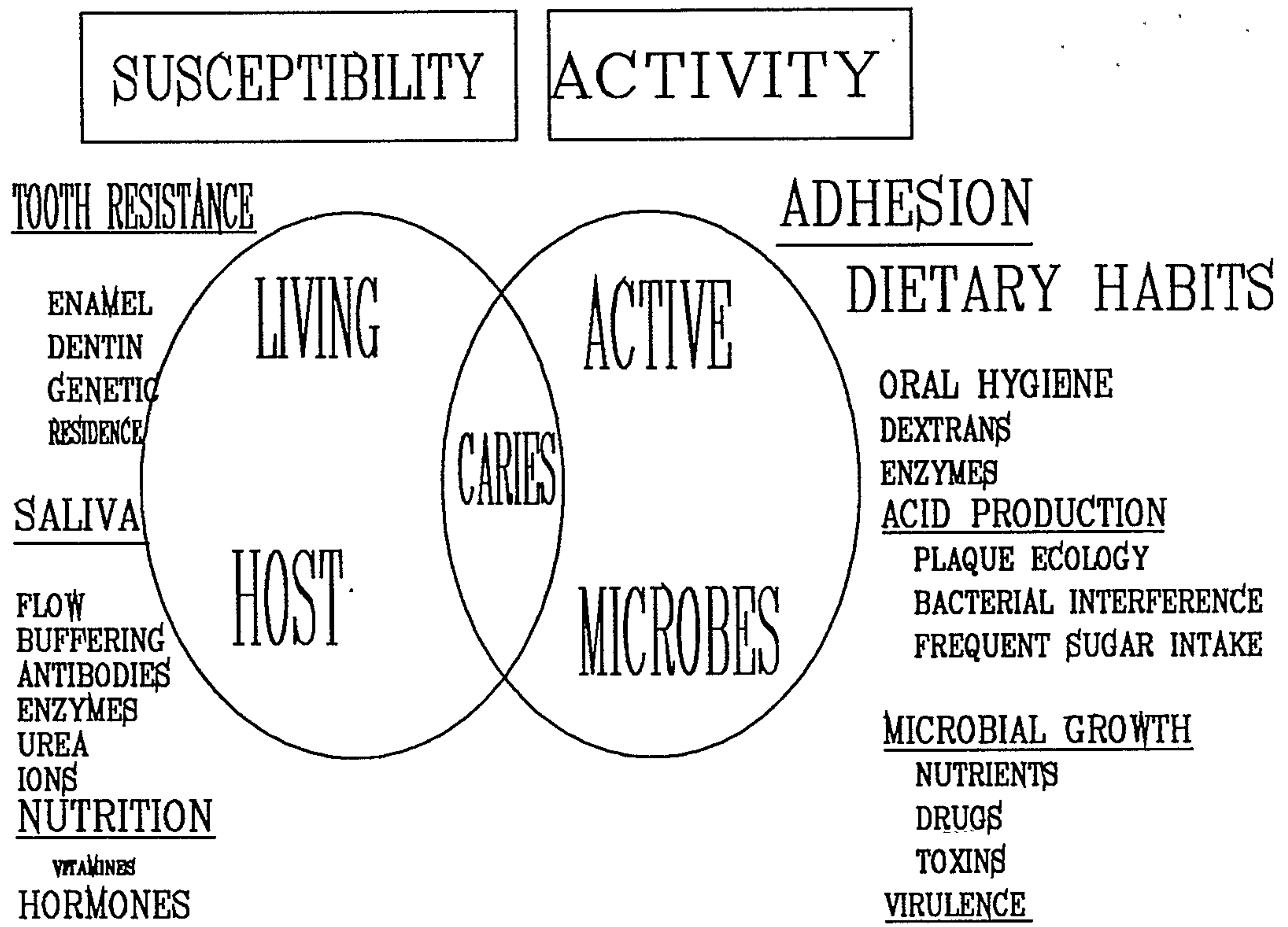
Dental caries has been described as a socio-medical problem because the disease attacks almost a hundred percent of population. The pattern of caries has undergone changes with time. In industrialised countries prevalence of caries in children and adolescence is showing a definite downward trend. However, because of increased access to dental care more teeth are retained in the older age group and in these more cases of root caries are being recorded. The caries picture in developing countries is being reported by epidemiologist to be the opposite with a dramatic increase in adolescents which is mainly attributed to exposure to a western type diet (Krasse 1985). A western type diet includes fast food snacks and carbonated drinks which encourage frequent snacking. It is a diet which is high in fat, protein and refined sugar. Opinions differ as to the causes of caries reduction in the industrialised countries. Some attribute it to the increased use of fluoride, while others to the changes in dietary habits (Newbrun 1989). It also seems possible that a wide spread use of fluoride toothpaste may have changed the microflora in such a way that the cariogenic microorganisms of low virulence increased to the detriment of highly virulent strains and lessened the caries risk as a result (Krasse 1985).

3.1 ETIOLOGY

For caries to develop the three main factors of microflora diet and host have to be present and interacting. The importance in observing the relationship between these factors for the diagnosis treatment and prevention of caries should be emphasized continuously. Newbrun (1989) states that a fourth factor, the time dimension must be considered in the etiology of caries. The clinical development of caries takes more than one year (Newbrun 1989). From the factors mentioned the living host susceptibility and the microflora activity are the two primary elements. (Larmas 1985).

Figure 3 shows that for caries to occur numerous factors must be acting concurrently via either the living host (susceptibility) or the microflora (activity).

Figure 3 Susceptibility and activity.
Source: Larmas (1985).

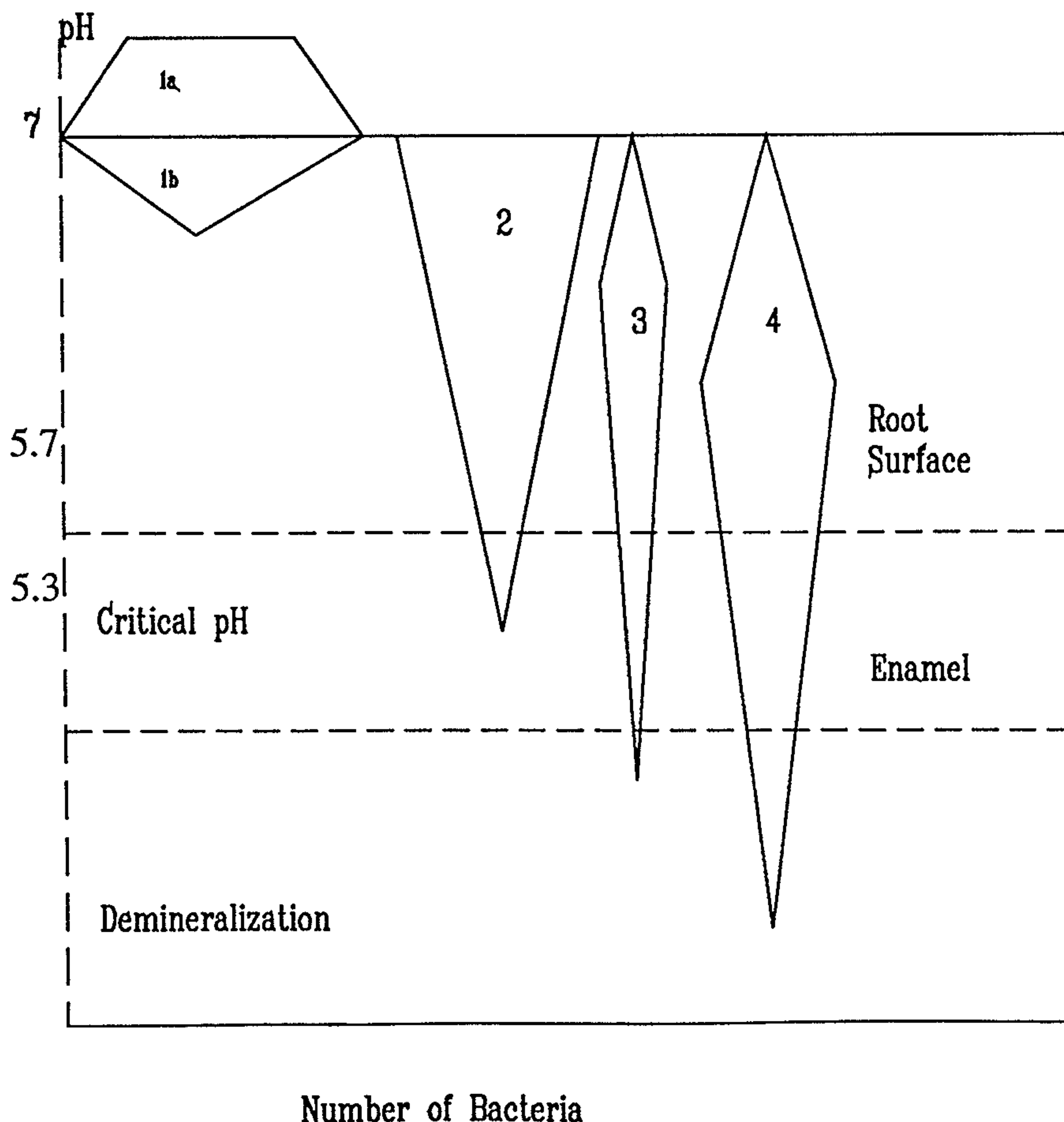


3.1.1 Microflora.

Some microorganisms are more important than others in the pathogenesis of dental caries, namely, streptococcus mutans, lactobacilli and some actinomyces species. Streptococcus mutans is generally associated with the initial development of caries and lactobacilli with the further development of the lesions and actinomyces with the root surface caries (Krasse 1985). Both lactobacilli and streptococcus mutans have the capacity to grow and produce substantial amounts of acids at a low pH of around 5.0. Low pH values favour the establishments of streptococcus mutans on the tooth surface. The ability to grow and produce acid at a low pH is of ultimate importance in the role of the microorganism for the development of caries.

Figure 4 shows that it is the function of the microorganisms and not the total number of plaque bacteria that determines whether or not demineralisation will occur. Figure 4 also shows acid production of different microorganisms in plaque. Some bacteria might produce alkali (1a) others only slight amount of acid (1b). Other micro-organisms (2) compromise a large proportion of the total number of the plaque bacteria which although they produce large amounts of acid never cause the pH to reach the so called critical level. The critical pH is the level at which the tooth substance is dissolved to a considerable degree. This level is believed to be between pH 5.3 and 5.7. some microorganisms like streptococcus mutans and lactobacilli bacilli (3) and (4) show optimal growth at a lower pH than other plaque bacteria and reach a final pH below the critical level. It is evident from Figure 4, that it is not the number of microorganisms but the function of certain bacteria that determine whether demineralisation will occur or not. Thus in order to evaluate the caries risk it is important to know the extent to which the person is carrying caries inducing organisms like streptococcus mutans and lactobacilli (Krasse 1985).

Figure 4 Microorganisms, bacteria and demineralisation.
Source: Krasse (1985)



Streptococcus mutans: Studies clearly show a strong correlation exists between streptococcus mutans and dental caries (Krasse 1985). The reason why this microorganism is cariogenic is not fully known but is most probably due to its unique combination of properties. It colonises the teeth., It produces a large amount of extracellular polysaccharides that enables voluminous plaque formation. It produces large amounts of acid even at low pH values. It breaks down some salivary glycoproteins which might be of great importance for the initial development of a carious lesion. Hunter et al (1988) suggests that streptococcus mutans have the biochemical properties to be a caries promoting plaque and there is a close correlation between streptococcus mutans and dental caries.

Lactobacilli: These microorganisms are also associated with caries at least under special circumstances. The conditions favouring these organisms is one where a mouth is subjected to high and repeated intake of sugar between meals and related well to the carbohydrate intake (Newbrun 1989). Thus the lactobacilli count can be used for an evaluation of the caries risk and assessment of the dietary changes (Harris, Park 1987). It is possible to easily cultivate and identify streptococcus mutans and lactobacilli and apply this information in the diagnosis treatment and prevention of dental caries (Klock 1984). Where there is a high prevalence of these microorganisms in the plaque there is generally a high number of these microorganisms in the saliva and therefore saliva can be a useful vehicle in routine examination to evaluate caries risk (Krasse 1985).

Actinomyces: Actinomyces strains especially , especially actinomyces viscosus, and actinomyces naelundii and actinomyces odontolyticus have been associated with the development of root surface caries (Narracott 1988). These microorganisms are poor acid producers and the development of root surface lesions is comparatively slow when these microorganisms are the causative factor. Selective media for cultivating actinomyces in plaque and saliva have been developed.

3.1.2 Diet

The importance of various dietary factors in the etiology of caries should be understood. Sugar appears to be the most important dietary item in caries etiology (Rugg-Gunn 1989). Hunter et al (1988) suggests that streptococcus mutans are sucrose dependant. Sucrose is the only sugar from which glucan can be formed. There is a close correlation between streptococcus mutans and dental caries.

Sucrose: The central role of sucrose in the development of dental caries is documented by a series of accurate observations. (Krasse 1985). These include:

1. Studies of the history and the geographical variation in the prevalence in dental caries.
2. Observation of isolated populations for which the environmental conditions have been changed.
3. Clinical studies and clinical experiments.
4. Observations of persons with hereditary fructose intolerance, who cannot eat sucrose and who remain almost caries free on a western type diet.
5. Experimental studies of animals.
6. Studies of relationship between streptococcus mutans and caries.

The Vipeholm study (Gustaffson et al 1954) demonstrated conclusively that the frequency of sugar intake rather than the total sugar intake was of decisive importance in the development of caries. The concentration and stickiness of the sugar influences its clearance. Starch is less caries accelerating than monosaccharides. Sucrose is five times more cariogenic than starch. Sucrose is a small unchanged molecule that easily diffuses into the dental plaque. It is highly soluble and acts as a substrate for both production of extracellular polysaccharides and for acid production (Newbrun 1989). Plaque microorganisms produce acids at the same rate of speed as from disaccharide sucrose as from glucose and fructose. Sucrose favours the establishment of streptococcus mutans on teeth (Harris, Christen 1987). A high sucrose intake gives rise to voluminous plaque formation. Sucrose does not contain any inhibitory action on plaque microorganisms. Sucrose plays a key role in the intercellular adherence of streptococcus mutans to form colonies (Krasse 1985).

3.1.3 Host

It was once considered the resistance of enamel to dissolution was of vital importance to the progress of dental caries. However, opinion has altered. The resistance of the tooth to caries is now thought to be partly dependent on whether it becomes colonised by cariogenic microorganisms (Newbrun 1989). The composition of saliva and the nature of the film to glycoproteins deposited on the tooth surface, the pellicle governs the establishment of the microflora on the tooth surface. The secretion rate and buffering capacity of saliva can be used to assess the caries risk (Krasse 1985). The salivary factors are affected by a number of physiological and pathological conditions (Krasse 1985).

3.2 CARIES PROCESS

The initial caries lesion develops when accumulations of organic acids interfere with the dynamic equilibrium of ion exchange causing more minerals to leave the tooth and form a demineralised subsurface lesion. This process is first recognised by the clinician as a "white spot lesion". In its early form it is reversible. (Peretz et al 1990).

3.3 HISTOLOGY OF CARIOUS LESION

Histologically a carious lesion on the smooth surface of enamel is conical in shape with its apex towards the dentine. Light microscopy studies of initial enamel lesions without surface cavitation have revealed four distinct zones representing varying degrees of hard tissue formation. (Peretz et al 1990). Starting from the surface of the lesion these zones are :

1. A relatively intact surface zone with 1 percent to 5 percent pore volume. (Normal enamel has 0.1 percent pore volume.)
2. The body of the lesion is markedly radiolucent. Its crystal size is 10-30 nm compared to 35-40 nm in sound enamel and its pore volume is 5-25 percent. The most common crystal damage occurs in the centre (core defect) due to the fact that the carbonate which is mostly located in the centre of the crystal is preferentially dissolved in acid.
3. A dark zone with crystal diameter of 80-100nm and 2-4 percent pore volume.
4. The last zone is an intermediary zone called the translucent zone, with minimal loss of mineral 1 percent pore volume and crystal size of 25-30 nm (Peretz et al 1990).

3.4 CRIES RISK

Ten to twenty per cent of the western population are of high caries risk. (Krasse 1985). Actual caries risk describes to what extent the person at a particular time runs the risk of developing a carious lesion. In most circumstances the actual risk is generally considered high or low.

3.4.1 Importance of caries risk assessment

An assessment of caries risk influences the planning of therapy, selection of restorative material, recall examination and forms the basis of various decisions in the daily work of the dental practitioner. The assessment is based on clinical examination and case history. If there is uncertainty towards treatment and prevention of disease these clinical examinations should be supplemented by dietary history, salivary and bacteriological samples (Krasse 1985).

3.5 ROOT CRIES

Root caries is the oldest recorded form of caries among human population. But in spite of this it is only in the last fifteen years that interest has been reawakened in this particular manifestation of dental disease (Comment 1989). Surveys carried out in a number of industrialised countries have pointed to a substantial decline in edentulousness in adults, an increasing life expectancy and increasing prevalence of root caries. With some justification root caries, can be regarded as a separate disease entity; with respect to its location, its microbiology and the pattern of its occurrence. (Comment 1989).

3.5.1 Definition

Root surface caries is defined as a soft progressive lesion on the root surface and is known by a variety of terms including cemental caries and senile caries. (Mount 1986).

3.5.2 Histopathology of root caries

A number of investigators (Phankosol et al 1985) have described the microscopic appearance of root caries. In Gram stained sections a dense mat of bacterial plaque extends across the lesion and adjacent exposed cemental surfaces. The plaque mass appears as a filamentous pallisade. The initial change appears to be an irregular pattern of surface erosion of cementum which precedes bacterial invasion of dentine. In more advanced lesions filamentous forms of coccoid cells actively invade the dentinal tubules with a secondary spread between tubules. Ultra structural studies (Narracott 1988) showed the distinctive pattern of dentinal invasion and seemed to demonstrate the unique importance of Actinomyces forms in the invasive process. At the advancing front of the lesion, tubules are closely packed with organisms and appear enlarged so that the peritubular dentine is reduced in thickness. Some of the enlarged tubules coalesce to form bacteria packed cavities representing a liquefaction focus.

3.5.3 Epidemiology of root caries

Epidemiological studies of root caries have been reviewed by Banting (1986) who has stressed the lack of consistency used in the wide range of population groups investigated. It does appear, however, that in healthy ambulant urban adults the prevalence of root caries ranges from 20-40 per cent. Prevalence increases with age and with special groups such as chronically ill institutionalised people. Banting (1986) has discussed the importance of relating observed root caries to the proportion of susceptible surfaces displayed. When such an approach is used prevalence rates, while only slightly higher, display a very much more linear trend with advancing age. A similar view that the unit of measurement in root caries prevalence studies should be surfaces exhibiting gingival recession and therefore "at risk" to lesion development has led to the development of a Root Caries Index (RCI) which is a measure of true attack rate. (Katz 1982). Table 4 shows the RCI formula.

Table 4 **Root caries index.**
Source: Katz (1982) .

$$\frac{(R-D) + (R-F)}{(R-D) + (R-F) + (R-N)} \times 100$$

(R-D): recession with decay root surface

(R-F): recession with filled root surface

(R-N): recession with sound root surface

Assessment of root surfaces most at risk to root caries is difficult to document since in older susceptible individuals tooth loss may already be considerable and indeed due to root caries itself rather than periodontal disease which has traditionally been accepted as a major cause of tooth loss in subjects (Narracott 1988). Katz et al (1982) have reported that the most commonly affected teeth, in rank order, mandibular molars, mandibular premolars and maxillary canines. The least affected were the mandibular incisors. In terms of surfaces affected, the RCI rate for proximal surfaces for maxillary molars was four times that for buccal surfaces. Interestingly, mandibular premolars exhibited a three fold increase in root caries compared with maxillary premolars inspite of the fact that recession occurred at approximately the same rate for each tooth type.

Leske and Ripa (1989) did a study on 796 adult patients (mean age 39.9 yrs.) who resided in fluoride deficient communities on Long Island, N.Y.. Incremental caries data were analysed to provide descriptive information about the susceptibility of individual teeth and surfaces to root caries. Molars were most prone to root caries/fillings, followed in decreasing order by premolars, canines and incisors. While canines and incisors had nearly identical increment, since there are half as many canines as incisors in the mouth, canines are actually twice as susceptible to root caries/fillings. Facial surfaces comprised 53 per cent of the increment followed by distal, lingual and mesial surfaces. Approximately 70 per cent of the DFS for facial and lingual surfaces were fillings, compared to approximately

50 per cent for mesial and distal surfaces suggesting that part of the increment for facial and lingual; surfaces may be treated as abrasion areas rather than caries. When comparing their study to Katz et al (1982), Leske, Ripa (1989) found their incidence data showed the same relationship for the incisors. On the other hand maxillary and mandibular molars exhibited a similar incidence. Table 5 shows a the number of root surface lesions and fillings during the three year period study according to arch and individual tooth type.

Table 5 **Number of root surface lesions and fillings during three-year study period according to arch and individual tooth type.**
Source: Leske, Ripa (1989).

Tooth Type and Arch											
		Incisors		Canines		Premolars		Molars		All Teeth	
Age Range	N	Max	Mand	Max	Mand	Max	Mand	Max	Mand	Max	Mand
20-24	49	0	0	0	0	0	0	0	0	0	0
25-34	217	0	0	1	0	0	0	0	4	1	4
35-44	224	11	1	6	2	14	9	13	18	44	30
45-54	200	7	4	7	3	14	24	35	31	63	62
55-65	106	20	3	24	5	14	39	30	27	88	74
Total											
Number	796	38	8	38	10	42	72	78	80	196	170
Percentage(*)		82.6	17.4	79.2	20.8	36.8	63.2	49.4	50.6	53.6	46.4

(*) Percentage represents the distribution between the maxillary and mandibular arches for each individual tooth type and for all teeth(last column).

3.5.4 Etiology of root caries.

The development of root caries surface takes place following the exposure of the root surface due to gingival recession. On this exposed root surface colonisation and invasion of bacteria occurs. Studies with experimental animals have suggested that several types of oral bacteria may result in root caries (Narracott 1988). As early as 1964 the association between *Actinomyces* species and root surface caries had been demonstrated in human dentition leading to a search for similar organisms in human root surface lesions (Jordan, Hammond 1972). When samples from deeper areas of root caries were cultured a number of *Actinomyces* were recovered particularly *A. viscosus*, *A. naeslundii* and *A. odontolyticus*. Other investigators have also isolated *Actinomyces* species from sound colonised surfaces in almost equal numbers. Hence it may be suggested since such organisms do not require dietary sugars but can utilise them when present they become invasive when suitable forms and concentrations of *actinomyces* species available (Jordan 1986).

The role of *streptococcus mutans* is unclear but the number of *streptococcus mutans* is higher in a root caries surface than on sound surface, while *lactobacilli* are found only occasionally in such lesions. (Keltjens et al 1987).

Dietary factors: The demineralisation of cementum and dentine follows the production of acids from dietary carbohydrates by plaque bacteria. In older people the cariogenicity of food stuffs may also be increased by the diminution of salivary flow. The side effects of a range of drugs include dry mouth "which contributes considerably to the problem".

Tavares et al (1991) did a study to assess the level of root caries in a population of diabetic adults. The purpose of the study was to explore the prevalence of root caries in an adult population with restricted ingestion of refined carbohydrates. Table 6 shows the sugar and starch restrictions reported by diabetic and non diabetic subjects.

Table 6 **Sugar, starch and diabetes.**
Source: Tavares et al (1991).

	Diabetics	Non-diabetics
"Slightly" or "not at all"	2	29
"Moderately", "nearly completely" or "completely"	28	1

Chi-square = 51.25; $p < 0.001$.

The results shown in **Table 7** show a highly statistically significant difference in the root surface filled in the diabetics and non-diabetics. The conclusion to this study was the difference in filled root surfaces can be viewed as a difference in caries experience. The reduced root caries rate in diabetics is very likely the result of a diet that significantly restricts the ingestion of refined carbohydrates. (Tavares et al 1991).

Table 7 **Means and standard deviations of root surface variables in the diabetic and non-diabetic subjects.**
Source: Tavares et al (1991).

Variable	Non-diabetics		Diabetics		Adjusted Mean Diff	RankTest p-value
	Mean	S.D.	Mean	S.D.		
Recessed buccal surfaces	17.32	10.41	18.22	13.54	0.30	0.58
C1 surfaces	0.25	0.58	0.31	0.86	0.05	0.65
C2 surfaces	0.58	1.28	0.56	1.05	-0.08	0.90
Filled surfaces	1.76	2.78	0.49	1.01	-1.24	<0.001
Katz Root Caries Index	15.24	17.57	7.06	13.09	-8.03	<0.001
Abraded buccal surfaces	2.44	3.28	2.10	3.41	-0.46	0.02

3.5.5 Clinical features

Unlike coronal lesions which commences in enamel, root caries extends as a shallow lesion under cementum and does not make a wedge shaped penetration into dentine as does coronal caries. Root caries is mainly evident from the mid thirties onwards although no age group is completely free from this condition (Comment 1989). It appears in its early stages as one or more well defined discoloured areas predominantly located along the cemento-enamel junction. The active lesions are softened without obvious cavitation and appear yellowish or light brown in colour. The passive or arrested lesions appear more darkly stained, often almost black, their consistency seems more leathery than active lesions. Frequently, however, the dark lesions with cavitation may be as hard if not harder than the non diseased root surface. (Nyvad, Fejerskov 1982). The lesions spread laterally and coalesce with minor neighbouring lesions and thus eventually encircle the tooth. Commonly, the lesions extend only 0.5-1 mm in depth. Another striking feature of the lesion is that it seldom spreads apically as the gingival margin recedes with continued periodontal breakdown but new lesions may develop later at the level of the new gingival margin (Nyvad, Fejerskov 1982). Table 8 shows the clinical definitions used for the assessment of root caries.

Table 8 **Clinical definitions used for assesment of root caries.**
Source: Tavares et al (1991).

C1 Incipient lesion: A well defined, softened area, yellowish or light brown, but without cavitation upon initial inspection; that is, the morphological integrity of the surface is undisturbed before the surface is probed.

C2 Frank cavitation: A softened area, yellowish or light brown, with a disruption of the normal surface contour; that is, there is a discontinuity or break in the surface, even prior to its being probed.

CF Secondary Caries: Caries at the margin of a restoration as evidenced by a yellowish brown softening at the interface of the restoration and the root surface.

F Root restoration: A restorative material that has been inserted on the root surface only.

R1 or R2 Overlapping lesion or restoration: A lesion or restoration that is primarily on the enamel but extends onto the root and is designated as R1. A lesion or restoration that is primarily on the root but extends on to the enamel is designated as R2.

A Root abrasion: A wedge shaped defect, softly angled in the early stage, sharply angled in later stages, with highly polished exposed dentine.

3.5.6 Treatment of root caries

Root surface lesions particularly those on the proximal surfaces are extremely difficult to restore. Early intervention is most desirable before cavitation has occurred and this approach rests on a sound knowledge on a clinical appearance of the developing root surface lesion. **Table 8** shows a root caries severity index and treatment recommendations. Billings (1986) demonstrated that shallow root lesions could be eradicated prophylactically and root surface recontoured and smoothed using conventional finishing and polishing material. This process may cause sensitivity to some patients and can be treated by daily use of self applied topical fluoride application. Lesions approaching 1mm or more generally require the placement of a restoration. Glass ionomer cement or glass cermets are the material of choice (Billings 1986, Mount 1986, Bryant 1991), while this material remains somewhat technique sensitive its cariostatic properties, ease of manipulation and predictable results make it the material of choice. In case esthetics is a priority the sandwich technique with glass ionomer cement and composite resin is recommended. The proposed treatment of pulpal tissue compromised by root carious exposure differs little from that expected for coronal carious exposure. Root canal therapy is indicated and should be performed immediately due to the possibility of canal calcification. In the case of root caries with deep periodontal pockets the best approach is to apically reposition the flap making the lesion supragingival and potentially restorable (Narracott 1988). A summary of root caries severity and treatment recommendations prepared by Ettinger (1991) is shown in **Table 9**.

Table 9 **Root caries and treatment recommendations.**
Source: Ettinger (1991).

Root caries severity	Treatment recommendations
Grade I - Incipient	Grade I -
a. Surface texture soft irregular, can be penetrated with dental explorer.	Daily topical fluoride treatment (1% sodium fluoride used in trays for 5 min. each day.)
b. No surface defect.	
c. Pigmentation-variable, light tan to brown.	
Grade II - Shallow	Grade II -
a. Surface texture soft, irregular rough, can be penetrated with dental explorer.	Excavate with hand or rotatory instrument to recontour root surface; Smooth and polish root surfaces with a conventional restorative finishing and polishing materials. Daily topical fluoride treatment.
b. Surface defect - less than 0.50mm in depth.	
c. Pigmentation - variable, tan to dark brown.	
Grade III - Cavitation	Grade III -
a. Surface texture - soft, can be penetrated with dental explorer.	Restore with conventional restorative materials. Daily topical fluoride treatment.
b. Penetrating lesion - cavitation present, greater than 0.50mm in depth, no pulpal involvement.	
c. Pigmentation - variable, light brown to dark brown.	
Grade IV - Pulpal	Grade IV -
a. Deeply penetrating lesion with pulpal involvement.	Endodontic therapy and restoration.
b. Pigmentation - variable - brown to dark brown.	

3.6 THE ROLE OF SALIVA IN DEMINERALISATION AND REMINERALISATION OF TEETH

Demineralisation of enamel occurs when under the influence of H^+ ions, the enamel crystals under the plaque dissolve and minerals from the dissolved crystals, particularly calcium and phosphate in their ionic form leave the enamel and enter the saliva and plaque fluid. The process may eventually lead to cavitation. The presence of the fluoride ion inhibits this process.

Remineralisation is the precipitation of calcium and phosphate ions as apatite like crystals in a previously demineralised area. This process occurs during enamel lesion formation and if conditions are favourable, arrest and even reverses the caries process. Presence of fluoride ion enhances this process. (Peretz et al 1990).

Saliva prevents the demineralisation of enamel by calcium, phosphate, fluoride content, its buffering agents mainly bicarbonates and phosphates. Fluoride even at low concentrations, reduces the rate of demineralisation-low levels of fluoride may be provided in saliva from fluoride toothpaste, gels, varnishes and so on. Fluoride also favours remineralisation of enamel lesions, and the mineral which is deposited on the enamel crystals forms a fluoride rich, fluoroapatite-like coating (Edgar, O'Mullane 1990). Table 10 shows the range of calcium, phosphate and fluoride concentrations found in the stimulated whole saliva of several hundred people.

Table 10 Calcium, phosphate and fluoride levels in human stimulated whole saliva.
Source: Edgar, O'Mullane (1990).

	Approximate concentration ranges	
	mmol/L	ppm
Calcium	0.75 - 1.75	30 - 70
Phosphate	2.0 - 5.0	60 - 155
Fluoride	0.0005-0.005	0.01-0.10

Very little of the total fluoride found in the mouth is secreted by the salivary glands about 0.01ppm. The higher levels of fluoride come from extraneous sources such as fluoridated water and toothpaste. Perhaps more important in relation to caries in the composition of plaque fluid. Plaque fluid is the liquid phase of plaque which is in contact with the tooth. It can be regarded as an intermediate reservoir between saliva and the aqueous environment in the diffusion channels between enamel crystals. Calcium and fluoride concentrations in plaque fluid are similar to those found in whole saliva, while phosphate is approximately double. **Table 11** shows calcium, phosphate and fluoride levels in plaque fluid.

Table 11 Calcium, phosphate and fluoride levels in plaque fluid.
Source: Edgar, O'Mullane (1990).

	Mean (SD) values	
	mmol/L	ppm
Calcium ion	0.85 (0.52)	34 (21)
Phosphate	11.5 (3.3)	356 (102)
Fluoride	0.0049 (0.0027)	0.09 (0.05)

This means that plaque fluid is highly supersaturated with calcium and phosphate ions, with respect to tooth mineral, and that free fluoride ions are also present at about 0.1ppm. The caries inhibitory constituents in saliva are listed in **Table 12**.

Table 12 Caries inhibitory constituents in saliva.
Source: Daly (1990).

Calcium, phosphate and Other trace elements (for remineralisation)	Bicarbonate buffer system (helps neutralise acids in plaque)
Antibacterial factors (Lysozyme, Peroxidase System, Immunoglobulins)	

4. DENTAL PLAQUE

Dental plaque has been recognised for over a century, but evidence of its key role in the development of both dental caries, gingival and periodontal disease has been forthcoming only in recent years (McHugh 1986). The importance of bacteria in dental plaque has only been fully appreciated since 1963 when Socransky and his colleagues demonstrated that human plaque contains 7.1×10^{11} organisms per gram wet weight. This is only slightly less than the 2.3×10^{11} organisms found in pure cultures of streptococci and shows that a plaque consists predominantly of bacteria rather than food remnants as had been thought. (McHugh 1986).

The significance of dental deposits for the development of periodontal disease lacked a scientific basis until the middle of the 20th century, when well designed epidemiological studies were performed (Lindhe 1989). It has now been established that removal of plaque is the method of preventing periodontal disease. Final evidence of the statement was presented by the abolition of oral hygiene procedures. (Loe et al 1965, Theilade et al 1966). Plaque is a normal part of the oral microflora and bacteria maintain a commensal relationship with the host. This relationship makes periodontal disease difficult to control. Normal flora will always re-establish itself. However, several authors have suggested that a reservoir of calcium phosphate and/or fluoride in dental plaque may be beneficial by maintaining the saturation with respect to enamel mineral, especially when plaque pH decreases (Pearce et al 1991). Pearce et al (1991) studied the effect of natural plaque in two subjects who withheld oral hygiene for 4 days and mouth rinsed in a mineral-enriching solution for two minutes four times per day during the last two days. The objective of the study was to examine dental plaque by transmission electron microscopy after treatment with calcium phosphate fluoride urea mouth rinse solution to ascertain the distribution, location and form of the mineral deposit. Chemical analysis of the smooth surface natural plaque exposed to the mineral enriched solution showed considerably elevated concentration of calcium phosphate and fluoride in treated subjects compared with

untreated control subjects. Results are shown in Table 13.

Table 13 **Composition of plaque samples.**
Source: Pearce et al (1991).

Treatment	Subject	Calcium mmol/mg	Phosphate mmol/mg	Fluoride mmol/mg
Mouthrinse	A	2.19	1.34	69.3
	B	2.10	0.92	20.5
No Treatment	A	0.27	0.18	1.2
	B	0.97	0.37	2.5

All values based on the dry weight of plaque.

The rationale of the plaque mineral-ion-reservoir concept of caries prevention is that fluorhydroxyapatite crystals, deposited in plaque will dissolve to a small extent when the pH decreases following sugar exposure, thus maintaining saturation of plaque fluid with respect to enamel mineral. This study was restricted to a very small number of subjects, and the results require confirmation by a larger study. However, it does suggest that the urea based mouth rinse solution is an effective means of producing a plaque mineral ion reservoir.

4.1 DEFINITION

Dental plaque is defined as a bacterial aggregation on the teeth or other solid oral structure (Lindhe 1989).

4.2 ECOLOGY OF THE ORAL CAVITY

Ecology of the oral cavity is an open growth system. That is, nutrients and bacteria are repeatedly introduced and removed from the system. Organisms that successfully colonise the mouth are those that adhere because of the different oral surfaces. Different organisms adhere to different surfaces (McHugh 1986). If they cannot adhere they will still colonise by getting stuck in some other way by being retained in pits and fissures. Once adhered or retained they must be available to successfully utilise the nutrients available (Lindhe 1989).

4.2.1 Factors acting against adherence

1. Salivary flow.
2. Flow of gingival crevicular fluid.
3. Chewing.
4. Oral hygiene procedures.
5. Desquamation of epithelial cells.
6. Clearance rate.
7. Buffering capacity of saliva.
8. Oxygen tension (varies and supports different species).

If bacteria can overcome the above factors they are in an ideal environment with high humidity and ideal temperature.

4.3 MICROBIOLOGY OF PLAQUE

In healthy gingiva it was found that gram +ve cocci and rods and a few gram negative cocci and rods were evident. As gingivitis became evident with the build-up of plaque, spirochetes and filamentous organisms began to take over in dominance (Lindhe 1989). This is shown in Figures 5 and 6.

Figure 5 Percentage distribution of organisms in phases of plaque development.
Source: Lindhe (1989).

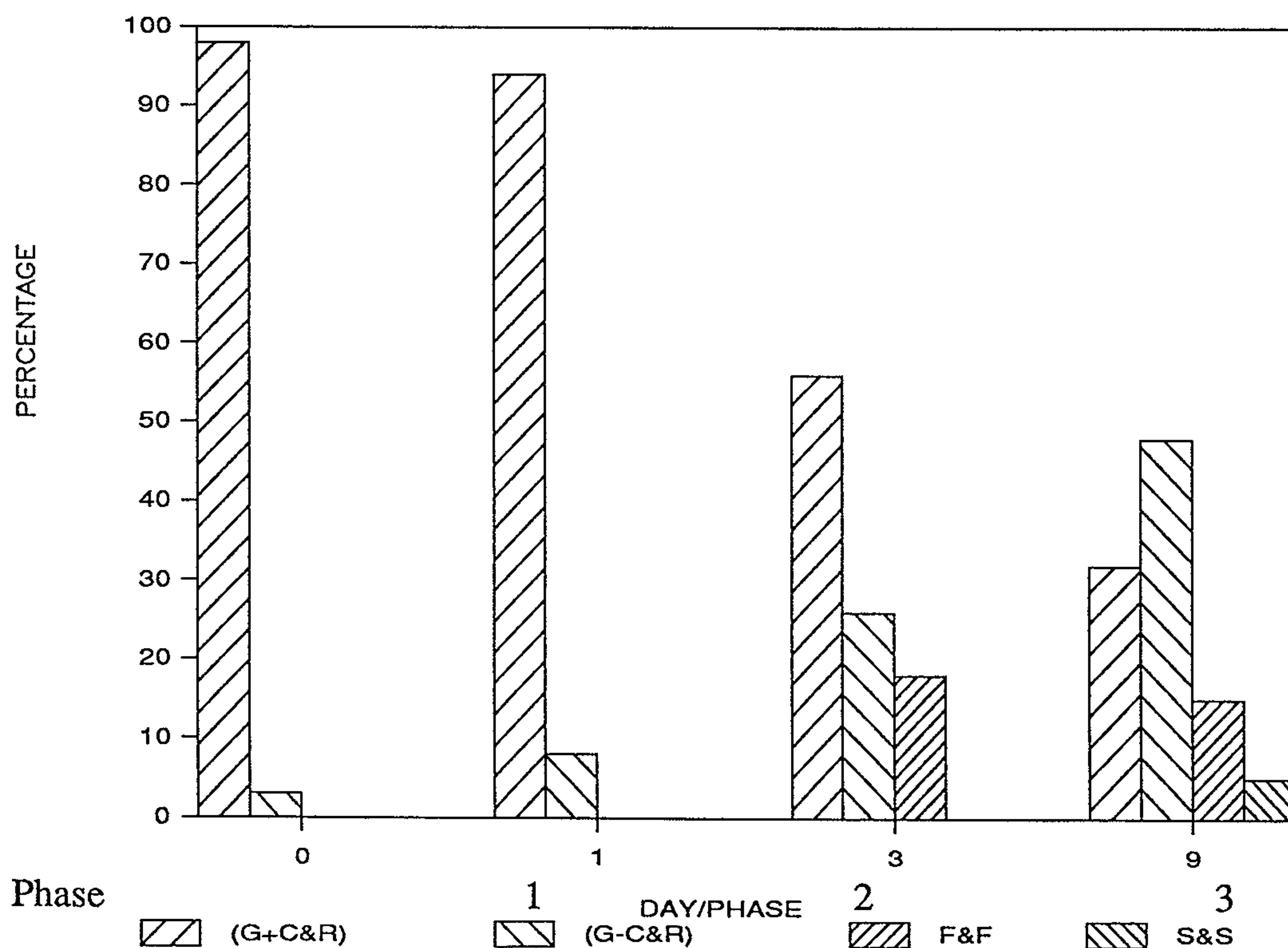
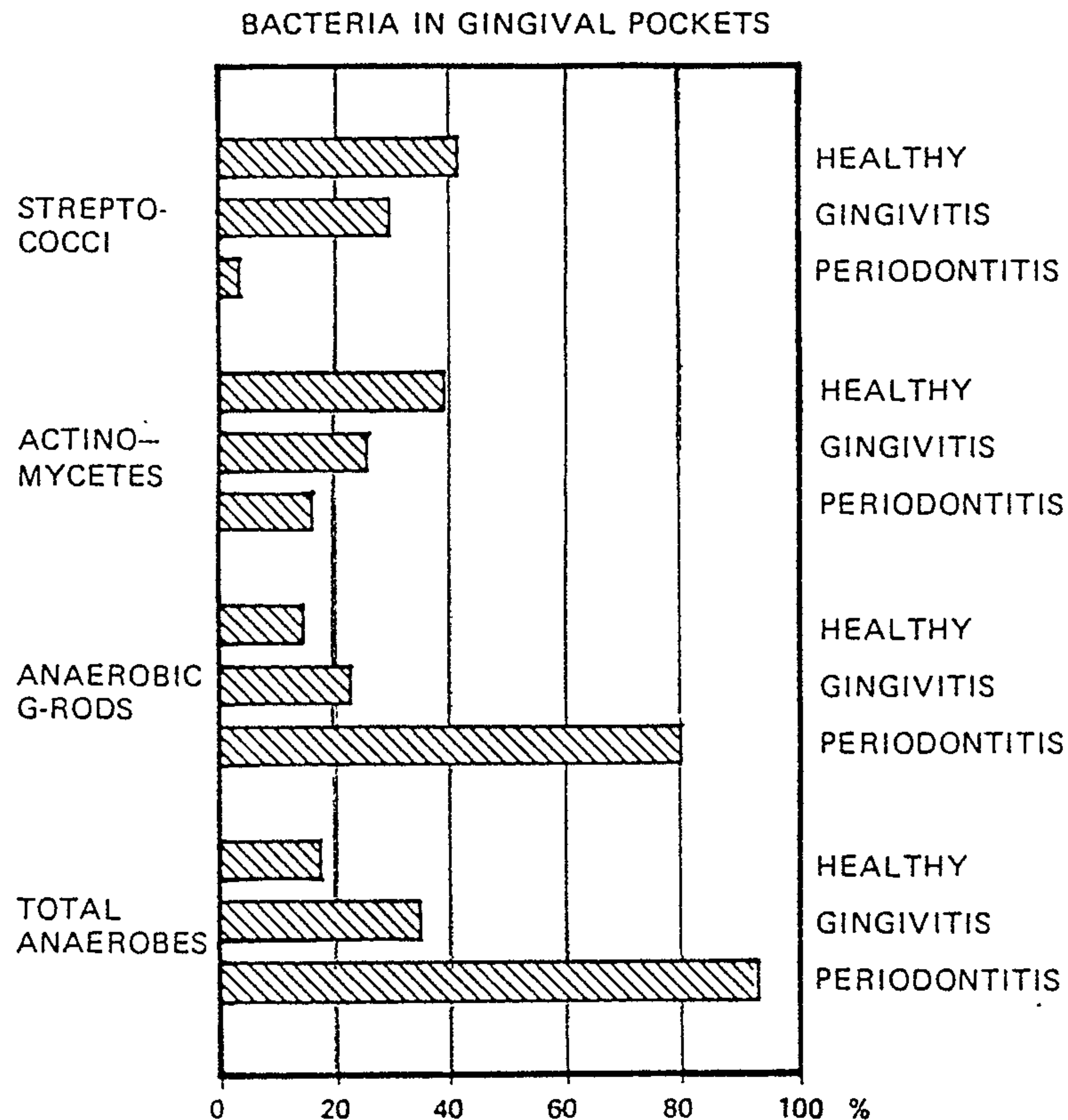


Figure 6 Bacteria in gingival pockets.
Source: Lindhe (1989).



4.4 PLAQUE FORMATION

There are three major phases in plaque formation (McHugh 1986):

1. Pellicle formation.
2. Initial colonisation.
3. Development of complex flora.

4.4.1 Pellicle formation

Pellicle or acquired pellicle is formed rapidly on tooth surfaces by selective absorption of protein containing material originating mostly from saliva, but probably also from soluble products from oral bacteria and saliva. It is initially 0.1 to 0.2 microns in thickness but increases in time upto 15 microns.

Acquired pellicle is a non bacterial plaque matrix. The pellicle has a variable structure appearance and develops between fifteen minutes and two hours. This amorphous proteinaceous membrane which contains

several salivary proteins adsorb into and coats the surface of the enamel. A major part of the acquired pellicle are proline rich proteins which show a selective absorption onto apatitic surfaces (Peretz et al 1990). Acquired pellicle thickness varies from 100nm after two hours to about 400 nm after 24-48 hours (Peretz et al 1990). Small dendritic projections off the pellicle penetrate a small distance into the pores and demineralised spaces of enamel. Initially, the salivary pellicle is predominantly bacteria free (Peretz et al 1990). Due to denaturation of proteins, this absorbed salivary coating becomes highly insoluble with time. The pellicles protective role is manifested by its ability to act as a "permselective membrane", a membrane which influences the mobility of calcium and phosphate ions from the enamel surface to the saliva and the penetration of H^+ ions from the saliva into the enamel. (Peretz et al 1990). When present, the pellicle reduces the rate of demineralisation of tooth surface caused by acidic conditions which arise as products of bacterial metabolism of carbohydrates. The pellicle is the initial foothold of saliva on the enamel surface on which bacterial colonisation first occurs.

4.4.2 Initial colonization

Oral bacteria vary markedly in their ability to colonise surfaces and evidence indicates that this is due more to differences in bacterial attachment than to differential growth rates (Gibbons, Van Houte 1973).

Adherence: Once pellicle has formed a number of factors determine whether or not bacteria will adhere to it and form plaque. The following factors should be considered.

1. Innate capacity to adhere or attach.
2. Capability to adhere to each other.
3. Number of bacteria present.
4. Different species have different rates of growth. This is very dependent upon the nutrient factors present and the quantity of them available from the host ie. whether or not essential substrates are available from the nutrients.

Ultra structural studies of the initial colonisation of cleaned teeth indicate that bacteria adhere additionally as well separated cells or small cell aggregates (McHugh 1986). Plaque seems to develop as a series of microenvironments. Only a small proportion of bacteria in the mouth become attached and most bacteria introduced into the mouth are cleared rapidly by the action of the lips, tongue, and the flow of saliva (McHugh 1986). If the removal forces such as the flow of saliva, the movement of the tongue and the lips, or the mastication of food are below critical limits, the layer of initially adherent micro-organisms will increase in thickness. This increase is mainly dependent on the growth rate of the organisms, although additional organisms often of different types are added to the surface (McHugh 1986). Some types of bacteria such as *B. Melaninogenicus* and other gram negative rods are dependent on prior colonisation by gram negative species (McHugh 1986).

4.4.3 Development of complex plaque flora

A complex flora develops within a few days of plaque formation starting on a clean tooth surface and reaches stability after seven to fourteen days. The flora is characterised by the presence of filamentous organisms and spirochetes (Lindhe 1989).

4.5 STRUCTURE AND COMPOSITION OF PLAQUE

The study of the structure and the composition of plaque is very difficult. Many bacteria have fastidious requirements and are anaerobes. Many attempts have been made to study the structure and composition of plaque. Listgarten (1975) did a study on six volunteers all requiring several full crowns. He made temporary crowns of epoxy resin for the volunteers. These were left in place and were removed in different stages of plaque formation. Plaque was allowed to accumulate so when the temporary crowns were taken out the plaque was still adherent to it. He then embedded this crown and its associated plaque in a block of the same epoxy resin and sectioned it. From this and other studies the structure and formation of plaque has been determined (Schroeder, Baumbauer 1966, Sonju, Rolla 1973).

4.5.1 Supragingival plaque

It consists of the pellicle then a layer of cocci, polymorphs and desquamated epithelial cells. This is covered by a layer of filamentous forms one week after oral hygiene measures are withdrawn. These grow into the coccal mat eventually replacing most of the cocci. Supragingival plaque associated with gingivitis is thicker than plaque associated with healthy tissues, it is predominantly gram positive and contains mostly actinomyces. Gingivitis develops consistently after a complex flora becomes established in supragingival plaque. It plays a primary role in the development of gingivitis periodontitis, enamel caries and cemental caries. Its elimination is greatly to be desired (McHugh 1986).

4.5.2 Subgingival plaque

The subgingival plaque starts its development from the supragingival plaque and is influenced by the subgingival environment. The subgingival area is very stagnant and adhesion is not as critical as for supragingival plaque. Motile bacteria like spirochetes can exist. The plaque adheres to dentine and cementum but not to enamel therefore histologically it has a different adhering surface. As the subgingival areas are not exposed to saliva, there is no pellicle instead there is a cuticle (which is the remains of the epithelial attachment), gingival fluid proteins and secretory products of epithelial cells. Early subgingival plaque is composed of a very dense layer of microorganisms next to the cuticle. These are gram negative and gram positive, cocci, rods and filamentous organisms. Outside this there are bacteria with flagelli and spirochetes. If plaque is present for long enough a pocket may develop. If this happens the microflora becomes a lot more variable and contains many spirochetes and motile organisms.

4.6 THE EFFECTS OF SALIVA ON PLAQUE MICROBIOLOGY

In addition to containing antibacterial activities, organic components in saliva (particularly mucous glycoproteins) support the growth of many oral bacteria; variations in their relative abilities to survive on salivary floral factors can explain some of the variations in plaque microbial composition.

4.6.1 Antimicrobial factors

The antimicrobial factors are listed below in Table 14. These factors are a part of the mouth's defence mechanism against invasion by pathogenic bacteria, while permitting tolerable levels of commensal organisms which are not normally pathogenic.

Table 14 Non-immunoglobulin antimicrobial factors found in saliva.
Source: Edgar, O'Mullane (1990).

	Salivary glands	Salivary exudate
Salivary peroxidase	+	-
Myeloperoxidase	-	+
Lysozyme	+	-
Lactoferrin	+	+
Aggregating factors	+	-
Histidine-rich proteins	+	-
Amylase	+	-
Anionic proteins	?	?

4.6.2 Salivary factors which aggregate oral bacteria and support bacterial growth

Saliva aggregating factors are believed to be important and have been well characterised. They act by clumping bacteria together and facilitating their adherence to surfaces. Saliva acts as a SUBSTRATE to bacterial growth. Invitro studies of sterilised saliva inoculated plaque show carbohydrates are metabolised first then proteins (Edgar, O'Mullane 1990). Saliva acts as a selective medium for oral organisms. Experiments done inoculating plaque material in various liquids show that in saliva a typical microflora develops (Edgar, O'Mullane 1990).

4.6.3 Role of salivary mucins

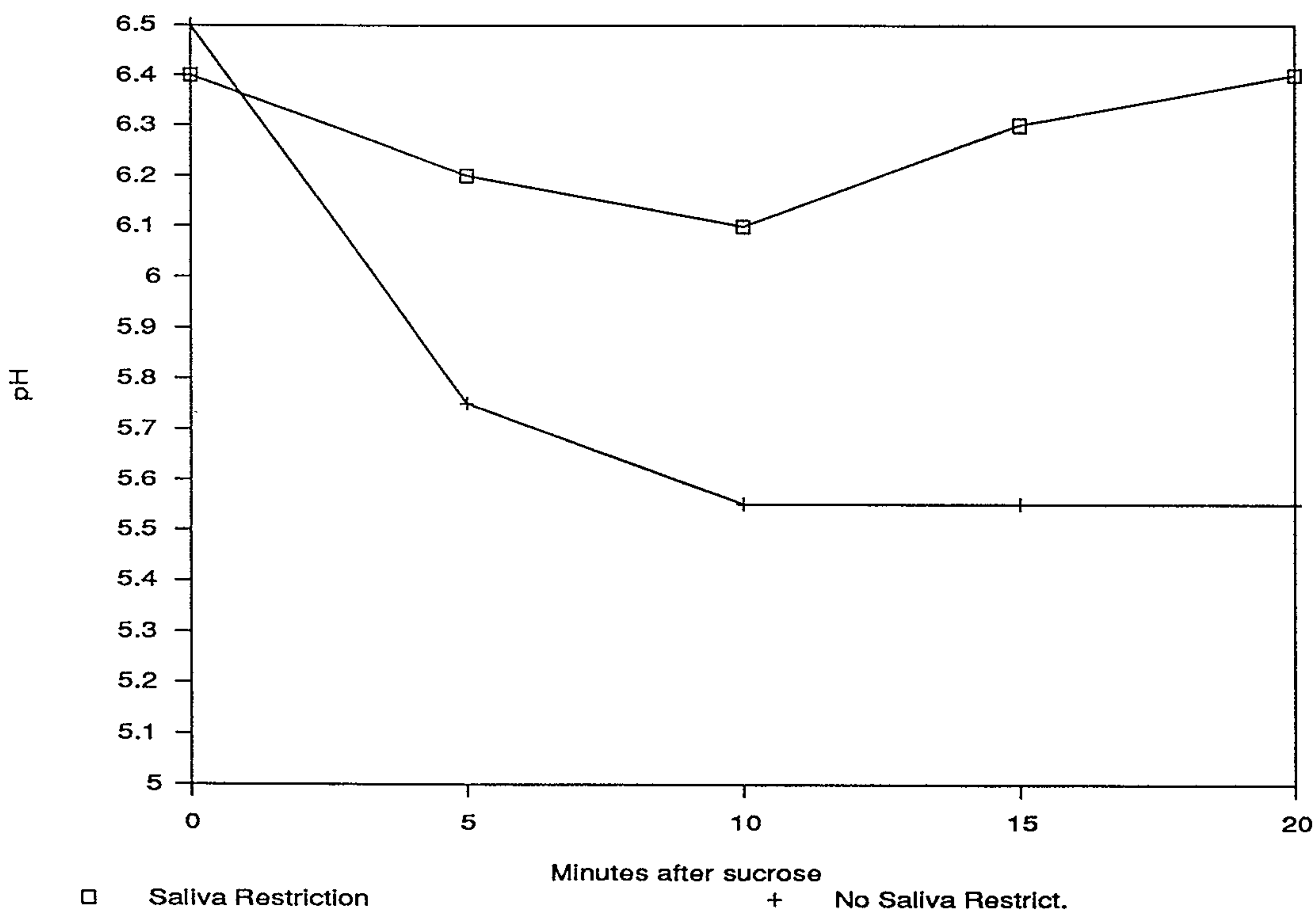
The types of energy source available for growth of microorganisms are very important in determining the microbial composition of plaque. Microorganisms can be grouped according to their preferred substrate. Studies done invitro, and in vivo on monkeys show that streptococcus species grow on mucin but that streptococcus mitior grows better than streptococcus mutans when mucin is the only source of nutrients. (Edgar, O'Mullane 1990). Thus, saliva alone selects for a non cariogenic microflora with low levels of streptococcus mutans. It is important when developing salivary substitutes that substrates are not added which would stimulate the numbers of streptococcus mutans (Edgar, O'Mullane 1990). In the mouth the salivary glycoproteins are also found either as a mucosal film or on the teeth as an acquired pellicle. The pellicle will differ in composition from ductal saliva as only certain components of saliva actually bind to the mucosa or tooth surface. Binding of bacteria to the acquired pellicle must also be taken into account in the selection of the flora.

4.7 EFFECTS OF SALIVA ON PLAQUE pH

Jenkins (1978) compared the Stephan curves produced following a sucrose rinse with or without salivary restriction. The results show that excluding saliva by cannulating the ducts of the major glands and diverting the saliva outside the mouth, lowered the minimum pH and slowed recovery to the base line pH (Jenkins 1978) as in Figure 7.

Figure 7 pH changes in plaque after a sucrose rinse with and without a restriction of saliva flow.

Source: Jenkins (1978).



4.7.1 Buffering systems

A large range of plaque pH values seem to be compatible with oral health. Healthy for one individual and maybe unhealthy for another. This is due to the multifactorial nature of dental caries.

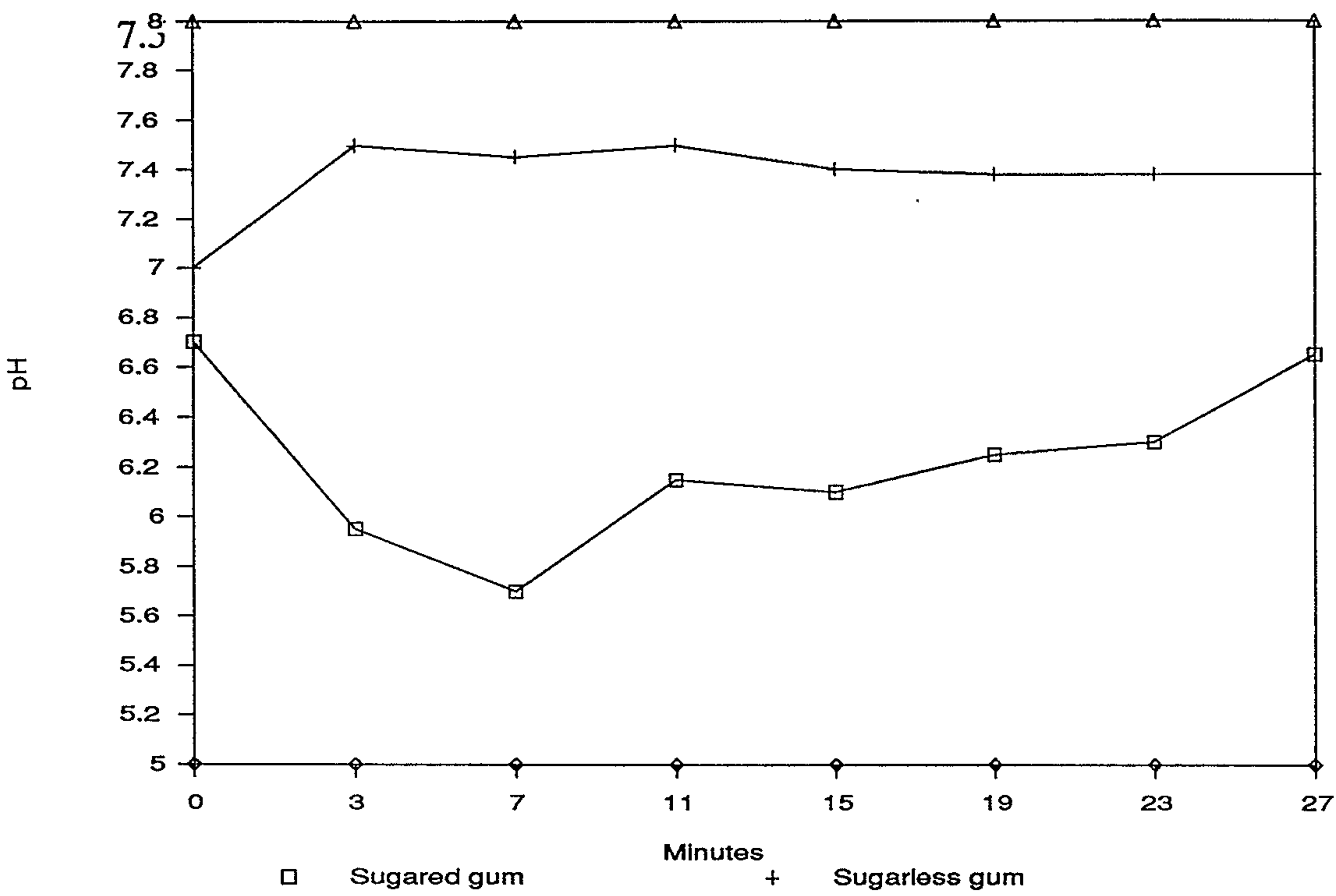
Plaque has an intrinsic buffering capacity due to the presence of phosphates, bicarbonates, proteins, and other macromolecules in plaque (Edgar, O'Mullane 1990). Saliva also contributes to several buffering systems including bicarbonate, phosphates, and proteins. Calcium phosphate crystals are thought to be present even in young plaque and can dissolve under acid conditions to increase greatly the buffering capacity. This can also raise the concentration of calcium and phosphate ions and thus help to oppose the demineralisation of the tooth (Edgar, O'Mullane 1990). A negative correlation exists between calcium phosphate in plaque and caries activity. Metabolically derived bicarbonates increases with salivary gland activity and provides an increasingly effective buffering capacity against acid especially at high flow rates (Edgar, O'Mullane). Salivary pH also rises with the increased flow rate, so in addition to its buffering effect, stimulated saliva neutralises plaque activity.

Saliva has detectable levels of urea and ammonia. Ammonia neutralises acid, urea can be converted by some oral microflora into ammonia. In addition some bacteria can decarboxylate the amino acids from salivary peptides to amines - these are alkaline and also remove hydrogen ions from the system (Edgar, O'Mullane 1990).

4.7.2 Enhanced salivary stimulation and its pH raising effects

The effect of chewing a sugarless gum and a sugar containing gum have been compared. Chewing a sugarless gum produced a rise in plaque pH, reflecting the raised pH of stimulated saliva. But with a sugar containing gum, despite a stimulated flow, there was a decrease in pH which lasted for twenty minutes (Rugg-Gunn 1978). Figure 8 shows the Stephan curves produced by sugared and sugarless chewing gum.

Figure 8 Stephan curves produced by sugared and sugarless chewing gum.
Source : Rugg-Gunn (1978) .



Chewing may therefore seem to have a beneficial on plaque pH by promoting salivary flow, but this effect may be reduced by the presence of fermentable carbohydrate. However, Jensen, Wefel (1989) suggest that sugarless or sugar containing gum is chewed after plaque is acidified by eating fermentable carbohydrate, both have a beneficial effect on neutralising plaque pH, the duration and time are critical. This suggests that additional substrate for acid production provided by the sugared gum is less of a factor. The level of bicarbonate in saliva in response to chewing gum can reach to 12-13 mmol per litre (Edgar, O'Mullane (1990)) and it is remarkable how quickly this can elevate pH.

4.8 FLUORIDE LEVELS AND PLAQUE pH

Salivary fluoride levels even in fluoridated areas and after using fluoride tooth paste are quite low, about 1 mmol/L (Edgar, O'Mullane 1990). It has been shown that an increased systemic intake of fluoride will lead to an elevated level of plasma fluoride and subsequently raised salivary levels. This can lead to increase in plaque fluoride level. It appears that a high level of fluoride is retained in plaque for upto 8 hours after a fluoride rinse (Edgar, O'Mullane 1990). The extent to which systemic fluoride administration can affect bacterial activity in the plaque is not known. But plaque fluoride levels are usually 50-100 times higher than that in whole saliva. Topically administered fluoride have antibacterial actions but is a direct effect and not mediated by saliva. However, fluoride from dentifrices, gels, and other vehicles may precipitate on the tooth surface as calcium fluoride, which then slowly dissolves into the saliva and elevates the salivary fluoride concentration slightly (Stookey 1987). Systemic fluorides have only a small effect on plaque acid production but their effect may be great enough to tip the scales between demineralisation and remineralisation of tooth enamel (Edgar, O'Mullane 1990), because part of the fluoride in plaque is present in a bound form but part is released into solution when the pH falls. This can also be potentially beneficial in favouring remineralisation and modifying subsequent bacterial metabolism.

5. XEROSTOMIA

Xerostomia or dryness of the mouth is a fairly frequent complaint of older patients. Xerostomia is not classified as a specific disease entity but is a symptom associated with a decreased lack of salivary secretion (Sreebny 1989). The clinical management of both , the general discomfort and any associated disease process such as dental caries, periodontal disease and candidiasis often found in patients suffering form xerostomia is difficult and frustrating (Ettinger 1981).

5.1 DEFINITION

Xerostomia is defined as the subjective feeling of oral dryness. This is frequently but not always associated with salivary gland hypofunction (Sreebny 1989).

5.2 EPIDEMIOLOGY

There are very few epidemiological data on xerostomia. A study of a general adult population in Rochester, New York, found a low prevalence of about 2 per cent in normal healthy individuals. This figure increased to 20 per cent, however, in individuals on medication (Edgar, O'Mullane 1990). Xerostomia is likely to become even more common as the population In the developed world shifts towards older people. The elderly more frequently experience the iatrogenic and systemic causes associated with xerostomia (Levine 1989)

5.3 ETIOLOGY

It is convenient to divide the many causes of xerostomia into those which are temporary or transient in nature or those which are permanent. Select diseases/conditions associated with salivary gland hypofunction and xerostomia are shown in **Table 15**.

Table 15 **Select diseases/conditions associated with salivary gland hypofunction and xerostomia.**

Source: Sreebny (1989).

Disease/condition	Examples
Drugs/medication	Anorectics, anticholinergics, antidepressants, antipsychotics, sedatives and hypnotics, antihistamines, antiparkinsonian drugs, antihypertensive agents, and diuretics.
Irradiation	Therapeutic radiation to the head and neck, for example, for cancer.
Organic Diseases	1. Rheumatoid (connective tissue, collagen, autoimmune) conditions, for example, Sjogren's syndrome (primary/secondary), rheumatoid arthritis, systemic lupus erythematosus, progressive systemic sclerosis (scleroderma). 2. Hyposecretory conditions: primary biliary cirrhosis, atrophic gastritis, graft v host disease, pancreatic insufficiency, type V hyperlipoproteinemia. 3. Immunodeficiency disease, example, AIDS. 4. Diabetes mellitus. 5. Hypertension. 6. Cystic fibrosis: Decreased flow in mucous-secreting minor salivary glands. 7. Neurologic disease, for example, Bell's palsy, trauma. 8. Dehydration: Impaired water intake; loss of water through the skin (fever, burns, excessive sweating); blood loss; emesis; diarrhea; renal water loss; polyuria; osmotic diuresis.
Psychogenic Disease	Depression
Decreased Mastication	Animal and limited human data indicate that intake limited to liquid or soft foods leads to a decrease in flow of saliva. Contribution to the feeling of oral dryness is not known.

5.3.1 Temporary causes of xerostomia

Emotional state: Anxiety and depression are well recognised as causes of reduced basal flow (Levine R 1989). They illustrate the powerful influence of the higher cortical centres on saliva secretion. While these conditions may ebb and flow they are often treated by drugs which in themselves are salivary inhibitors.

Duct calculi: Blockage of the duct of a major salivary gland can produce dryness on the effected side, together with pain and swelling in the gland, especially on stimulation. The condition is most common in middle age, with the submandibular gland most likely to be affected. Calcification may occur anywhere along the duct and even in the body of the gland, producing a more diffuse swelling or sometimes cellulitis. The calculi may range from 1mm to more than 10mm in length.

Sialoadenitis: There are many causes of inflammation of the salivary gland which can result in impaired function. Acute infections include mumps and post operative parotitis, while chronic conditions include swellings related to nutritional deficiency and hypersensitivity to iodine. However, many cases of intermittent swelling of the salivary glands are idiopathic and can be grouped under the heading "chronic nonspecific sialoadenitis". This condition is often associated with duct calculi and if untreated may lead to progressive fibrosis of the gland and the development of permanent xerostomia (Levine R 1989)

Drug therapy: One of the most common causes of temporary xerostomia is the use of drugs. These may act on higher cortical centres, but more commonly act on the mechanism of neuro-transmission. A wide variety of drugs may have this effect. They include many antihistamines and antidepressants. Some of the most commonly used drugs are presented in Table 16.

Table 16 **Drugs associated with xerostomia.**
Source: Levine R (1989) .

Drug	Use
Acetazolamide	Diuretic
Amitriptyline	Antidepressant
Amphetamine	CNS stimulant
Chlorpromazine	Antipsychotic
Clonidine	Antihypertensive
Guanethidine	Antihypertensive
Imipramine	Antidepressant
Methadone	Analgesic
Methyldopa	Antihypertensive
Nortryptaline	Antidepressant
Promazine	Antipsychotic
Tetracycline	Antibiotic

The excessive use of diuretics can result in dehydration and xerostomia. In most cases function recovers after the drug is withdrawn. However, as many such drugs are prescribed for chronic recurrent conditions, their use frequently becomes the basis for long term treatment.

Drug related changes vary in intensity from person to person. In dentate persons, the caries risk, especially root caries will depend on: the duration of administration of the drug (Ettinger 1981). The person's susceptibility to caries will also depend on the degree of dietary alteration related to a variety of factors; and the severity of xerostomia the drugs produce; as well as on the effectiveness of the person's oral hygiene regimen. For the elderly, neuromuscular coordination becomes a major factor in the ability to maintain an adequate level of oral hygiene (Ferguson M 1989). A dentist should be aware of the oral side effects of the drugs prescribed for his patients. If side effects are noticed, it may be possible to ask the patients physician either to adjust the drug dosages, to change drugs, or to jointly treat the induced xerostomia.

Dehydration: Any condition which creates a loss of fluid such as vomiting, diarrhoea, sweating or haemorrhage will cause a xerostomia.

5.3.2 Permanent causes of xerostomia

Salivary gland aplasia: Congenital absence of one or more major salivary glands is a rare but recognised condition of unknown etiology without an obvious hereditary basis and does not appear to be associated with other ectodermal dysplasias.

Sjogrens syndrome: This autoimmune disease is usually found in older women and classically consists of the triad: xerostomia, keratoconjunctivitis, sicca, and rheumatoid arthritis. Salivary gland enlargement can occur unilaterally but is usually bilateral and the glands most often involved are the parotids. It has been shown in a number of studies (Ettinger 1981) that the salivary flow rate is reduced and that this is related to the lymphocytic infiltration and replacement of the functional parenchyma by these inflammatory cells. The decreased salivation causes difficulty in swallowing and mastication, abnormalities in taste sensation, oral mucosal soreness and ulceration. The oral mucosa often appears dry, smooth, and glazed with tongue fissuring. The common complications associated with the disease are xerostomia, candidiasis, angular cheilitis and root caries (if teeth are present).

Other systemic disorders: Xerostomia is associated with diabetes mellitus, probably as a consequence of polyurea. The prevalence of xerostomia in diabetic patients is presented in Table 17.

Table 17 **Prevalence of xerostomia in diabetic patients.**
Source: Tavares et al (1991).

<u>Drug Intake, Type</u>	<u>Presence of Oral Dryness</u>		
	Yes	No	Per cent
All types	25	31	45
Xerogenic drugs	9	8	53
None or nonxerogenic	15	24	38

Parkinson's disease, cystic fibrosis, sarcoidosis, cardiac failure, uremia, hypertension, thyroid disease and folic acid deficiency. There are other various disease states or deficiencies which create alterations in salivary composition and may reflect biochemical changes and electrolyte balance in the persons blood, plasma or serum levels. Therefore, a xerostomia of known definable cause may be a symptom of an undiagnosed systemic disease (Ettinger 1981) such a patient should be referred to a physician for medical evaluation.

Xerostomia may also be due to disorders affecting the autonomic outflow pathway such as encephalitis, brain tumours or accidents.

Radiotherapy: A well recognised side effect of radiotherapy to the head and neck for malignancy is the loss of salivary gland function (Daly 1990). Post-radiation atrophy is partly due to reduction in the vascularity of the gland and partly to the direct effect of the x-rays on the specialised epithelial cells in the secretory acini which are highly sensitive to radiation. In some cases there is recovery of function after several months, but in others permanent xerostomia develops. It should be added that there is no evidence that radiation damages the teeth or periodontal tissues directly, the effect on the dentition resulting solely from the reduction in salivary flow (Levine R 1989).

Surgical desalivation: This may occur as a result of surgical intervention for tumours of the salivary glands or related structures or as a result of trauma involving the removal of part or all of a major gland, or damage to its duct or nerve supply.

5.3.3 Physiological Xerostomia

In addition to the pathological and iatrogenic causes of xerostomia, it should be recognised that the sensation of dry mouth can arise as a result of psychological changes. The sensation of dry mouth may occur because of an imbalance between the salivary flow entering the

mouth on one hand, and the loss of water from the mouth as a result of evaporation into exhaled air and absorption of water by the oral mucosa on the other hand. Ten to fifteen per cent of people breathe through the mouth at rest (Levine R 1989). This factor may be an important cause of complaints of dryness.

5.3.4 Aging

It is not entirely clear how the aging process influences the rate of salivary flow. Some investigators have shown that in healthy unmedicated persons, the flow of whole or parotid saliva does not change with age (Parvinen, Larmas 1981). The exception to this may be post menopausal women who demonstrate a slightly reduced rate of secretion (Heft, Baum 1984). Submandibular and minor salivary flow on the other hand may decrease with age (Pedersen et al 1985). Other investigators maintain that the flow of whole saliva declines with age (Navazesh, Milligan 1989). Basic etiologies of salivary gland dysfunction are presented in Table 18.

Table 18 Basic etiologies of salivary gland dysfunction.
Source: Wright (1987).

Systemic auto-immune diseases.

Radiation therapy to fields that expose the salivary glands.

Medications

Infections (which include those reported in homosexuals/AIDS patients)

Idiopathic conditions.

Trauma & surgical excision

5.4 CLINICAL MANIFESTATION

The patient with compromised salivary gland function may present a number of problems (Ben-Aryeh et al 1985, Fox et al 1987):

1. Dry or burning sensation.
2. Difficulty with speech eating and taste.
3. Mucosal infection.
4. Difficulty in coping with dentures.
5. Bacterial sialadenitis.
6. Periodontic disease.
7. Dental caries.

The dry and burning mouth is often the major presenting symptom (Ferguson M 1989). The patient may present deep fissuring of the upper and lower lip, angular cheilitis, a cracked tongue, a dry lobulated or fissured tongue and complain of bad breath. A red glazed lingual mucosa is common after having a dry mouth for a lengthy period of time. Patients present with the entire spectrum of dry mouth symptoms, from mouths that appear completely normal to extremely dry mouths.

It is important for clinicians to accept that underlying salivary disease should not be ruled out, just because the mouth looks reasonable. In addition to salivary factors a clinician must consider other mucosal and psychological problems. If a patient presents with what appears to be a moist mucosa without complaining of a dry mouth, salivary gland disease should not necessarily be discounted.

Patients with xerostomia avoid a variety of foods because of the difficulty of chewing and swallowing. Food debris persists in these individuals longer than in the person with adequate salivary flow and brings further annoyance. There is also a diminution in taste. These patients tolerate dentures poorly, and denture stomatitis or chronic atrophic candidiasis is more common in these individuals (Ettinger 1990). A whitish membrane may also be seen on the posterior part of the tongue or palate.

Enlargement of the salivary gland usually bilateral, painless, usually in the parotid may be due to radiation or disease (Ferguson M 1989). Bacterial sialadenitis is also a potential problem with reduced flow. A whole range of different microorganisms and sometimes mixed organisms may ascend the duct. The patient, with pre-existing inflammatory exocrinopathy, may eventually produces a fistula over the parotid with pus tracking down the side of the face.

The film of dried or thick saliva on the teeth is characteristic in xerostomia. Periodontal disease is a problem in xerostomic individuals. Plaque accumulation, debris in the mouth, and gingivitis appear in patients especially those who have tricyclic antidepressants for a number of months (Ferguson M 1989). In patients who receive radiotherapy, radiation mucositis is also a problem.

The patient who has dry mouth uses a variety of liquids for self treatment like carbonated drinks or suck on candies, acid drops or mints; all obviously predisposing to caries. The patient who has systemic lupus erythematosus and inflammatory exocrinopathy can develop severe problems with widespread periodontal disease and rampant caries (Ferguson M 1989). A frequent complaint voiced by the patient is "my fillings are falling out and my teeth are crumbling away" (Mandel 1989). Clinically the caries susceptibility manifests first on exposed cementum as root caries on both buccal and lingual surfaces, or at the edge of fillings or recently replaced fillings. In time, enamel lesions increase markedly and involve usually decay-resistant surfaces, such as the approximal surfaces of lower anterior teeth and incisal surfaces. With longstanding untreated xerostomia, the caries become circumferential with minimal specific site localisation and an almost total involvement of the crowns and roots. Associated oral and non-oral symptoms presented in Table 19. The deleterious oral sequelae of salivary gland dysfunction is presented in Table 20.

Table 19 Relationship of xerostomia to select oral and nonoral symptoms.

Source: Sreebny (1989).

Symptom	Dry mouth Group, % (N=131)	Wet mouth Group, % (N=378)
Xerostomia	100	100

Oral Symptoms :

Try to keep mouth moist	79	18
Get out of bed to drink	48	16
Difficulty with speech	48	6
Difficulty with swallowing	29	5
Fissures, sores at corners of lips	28	8
Keep fluids at bedside	27	11
Difficulty chewing dry food	25	8
Tongue: burning, tingling feelings	19	3
Problems with taste	13	5

Nonoral Symptoms :

Dry throat	76	33
Blurred vision	61	29
Dry eyes	39	20
Burning sensation, eyes	37	20
Sandy, gritty feeling, eyes	32	16
Use eye drops	9	4
Dry skin	52	32
Breathe through mouth	37	29
Dry nose	34	18
Change in sense of smell	12	9

Females only, N=96 :

Vaginal itching	42	19
History of vaginal fungal infections	38	8
Vaginal burning	28	9
Vaginal dryness	27	17

Table 20 The deleterious sequelae of salivary gland dysfunction.
Source: Wright (1987).

Functional alterations, inconveniences, and discomforts

- difficulty in speaking
- difficulty in swallowing
- difficulty in chewing
- taste aberration
- breath malodor

Soft-tissue alteration and deterioration related to decreases quantity and quantity of saliva

- alteration in oral microflora
- reduced saliva-buffering capacity
- gingivitis
- mucositis
- ulceration
- increased incidence of trauma from teeth, foods, prostheses
- increased incidence of secondary infection (bacterial, fungal, viral)
- swollen, tender salivary glands and inflamed duct orifices
- dry, cracked lips and corners of mouth

Tooth structure deterioration

- decalcification
- dental caries
- incisal margin-chipping
- crown fractures

5.5 INVESTIGATIONS

In the investigations of the patient who presents with a dry mouth, history and examination are of crucial importance. The concepts of flow rate, sialochemistry, gland biopsy for morphological changes, sialography and scintiscanning are all relevant. Methods of evaluation of salivary gland function are shown in Table 21.

Table 21 Methods for evaluation of salivary gland function.
Source: Fox (1989).

Sialometry
 Sialochemistry
 Salivary Scintigraphy (99m Tc)
 Sialography
 Ultrasound, MRI, CT

5.5.1 History and Examination

Points to establish are; whether the dryness is continual or intermittent; whether it is accompanied by pain or swelling; if unilateral or bilateral; and whether there is any relevant history of anxiety, stress or depression, a systemic disorder, irradiation, trauma, surgery or medication. The patient's occupation and domestic situation are often relevant.

A duct calculus will usually present in a patient as a unilateral dryness with pain or discomfort and swelling in the affective gland on stimulation. In Sjogrens syndrome any swelling is often constant and is accompanied by other symptoms of the syndrome and in many cases lymph node enlargement. As well as looking for evidence of enlargement of salivary glands and lymph nodes and unilateral dryness, the examination should include palpation of the floor of the mouth for evidence of submandibular duct calculi and examination of the major openings, as inflammation or swelling of the orifice may indicate the presence of a distally placed calculus. These are often easily revealed by simple intraoral radiography (Fox 1989).

A question that often arises concerns patients requiring salivary evaluation. Subjective complaints are not reliable indicators of gland dysfunction. In selecting patients who have measurable gland hypofunction from a larger group who complained of dry mouth, Fox PC et al (1987) examined responses to a standardised questionnaire from 100 individuals with the chief complaint of oral dryness (xerostomia). They identified several useful questions for distinguishing individuals with salivary dysfunctions. Questions used to determine individuals with salivary gland hypofunction are presented in Table 22.

Table 22 Questions to determine individuals with salivary gland hypofunction.

Source: Fox et al (1987).

- A. Positive Responses Are Suggestive of Salivary Hypofunction
 - Does your mouth feel dry when eating a meal?
 - Do you have difficulty swallowing foods?
 - Do you sip liquids to aid in swallowing dry foods?
 - Is the amount of saliva in your mouth most of the time too little or don't you notice it?

- B. Positive Responses Did Not Predict Salivary Hypofunction
 - Does your mouth feel dry at night or on awakening?
 - Does your mouth feel dry at other times of the day?
 - Do you chew gum or use hard candies or mints daily to relieve oral dryness?

Positive responses are not a guarantee that gland hypofunction can be found but are suggestive of salivary dysfunction and identify individuals who should be investigated further. Early identification of the patient with salivary gland dysfunction allows for intervention and the opportunity to prevent serious consequences.

5.5.2 Sialometry

Flow rates can be assessed using a range of techniques; different dentists tend to favour the method with which they are most familiar, although a uniform approach would be desirable. Regardless of the technique being used clinicians must use a set value, above which is normal and below which is a clear indicator of disease (Ferguson M 1989). **Table 23** shows the values of daily production of whole saliva. Resting flow rates and gland activity are presented in **Table 24**. The salivary flow rates for parotid, submandibular and sublingual glands are in **Table 25**.

Table 23 The daily production of whole saliva.
Source: Sreebny (1989).

Physiological State	Time hr.	Flow rate		Volume (mL)
		min	(mL/min)	
Sleep	8	480	0	0
Waking State				
Stimulated (Eating)	2	120	2.0	240
Resting	14	840	0.4	336
Total =				576

If stimulated (eating) = 3 hrs, total daily flow = 696 mL.

Table 24 Resting flow rates and gland activity.
Source: Sreebny (1989).

Gland Activity	Flow Rate (mL/min)
All salivary glands functional	0.40
35% loss of activity	0.30
50% loss of activity	0.20
75% loss of activity	0.10

Xerostomia generally associated with flow rates ≤ 0.2 mL/min.

Table 25 Salivary flow rates from parotid and sm/sl* glands.
Source: Sreebny (1989).

Gland	Flow rate (ml/min/gland)	
	Resting	Stimulated**
Parotid gland	0.04	0.5-0.8
Sm/sl gland	0.12	0.5-0.8

* Sm/sl = submandibular and sublingual glands

**stimulated with citric acid

assessing salivary flow both in its basal state and on stimulation is an important diagnostic test. While collection of saliva from individual major glands is a specialised and difficult procedure, an indication of total flow can easily be obtained. Levine R (1989) has suggested a method where a patient is asked to expectorate in to a 25mL glass measuring cylinder for three minutes, first without stimulation and then for 3 minutes in all chewing paraffin wax or sorbitol chewing gum. Flow rates for healthy adults of less than 0.2mL per min at rest and less than 0.5mL per min when stimulated should be regarded as low. The measurement of residual saliva volume has always been difficult however, Levine R (1989) has suggested a rapid and accurate method which involves introducing a small volume of tasteless fluoride solution into the mouth. After allowing a few minutes for dilution into the pool of saliva, a saliva sample is taken and fluoride concentration estimated using a specific ion electrode. A simple calculation of the dilution gives the volume of saliva in the mouth.

Sialometry is the quantitation of saliva output, and can be performed on individual major gland secretion or whole saliva. Parotid saliva is usually obtained using a modified carlson-crittenden collector (Jenkins 1978). This device covers the orifice of stensen's duct and holds to the mucosa with gentle suction. It allows collection of pure gland saliva in a non invasive, nonpainful manner. Collection of submandibular/sublingual saliva presents greater difficulties. Fox (1989) has reported a method that allows for simple sampling with disposable equipment that can be used regardless of the anatomy of the whartons duct area. In this method a micropipette collects the secretions using a continuous gentle suction after the region is isolated with gauze and the parotid orifices are covered. Saliva is drawn into the collecting tube. For this technique, collection is carefully timed so that a flow rate, expressed as mL/min per gland can be derived. If compositional analysis are to be done, saliva should be collected into chilled tubes, kept on ice, and frozen until

analysis. An important consideration, is whether the secretion is unstimulated or stimulated. For the former, subjects should have nothing in the mouth for a minimum of 90 minutes before collection. During this time, they must refrain from any oral activity such as smoking or tooth brushing. Stimulated samples must utilise a standard reproducible stimulus. A gustatory stimulus, such as 2 per cent citric acid solution is useful and easily prepared. It should be applied at regular intervals to a specific area, most commonly the lateral dorsal tongue surface. The most important considerations when sialometry is done, are to control and specify the collection technique. Whatever technique is used, meaningful results will be obtained only if great care is given so that the collection method are well-described and reproducible (Fox 1989).

5.5.3 Sialochemistry

Sialochemistry is valuable in delineating the elements of the salivary secretions and their concentrations. Patterns of salivary concentrations have been described for a number of diseases (Fox 1989). Salivary composition is reflective of gland function and disease but not of a specific diagnosis. Sialochemistry gives information about the extent and location of gland pathology. Salivary chemistries can be considered supportive of diagnosis but not diagnostic.

5.5.4 Salivary scintigraphy

Salivary scintigraphy is an autoradiograph procedure which has varied with time but may be useful when quantitated during concentration and emptying of the salivary gland. It is performed utilising technetium pertechnetate (^{99m}Tc). A gamma-emitting radionuclide with affinity for salivary, gastric and thyroid glands. An intravenous bolus of technetium is injected and the uptake and the secretion of the tracer by the major salivary glands is followed (Fox 1989). Tracer movement can be scanned in arresting and stimulated state. Technetium pertechnetate has been shown to be transported by salivary acinar

cells and serves as a useful indicator of functional tissues (Fox 1989). Pertechnetate passes through the acini and is handled similarly to iodide by the salivary glands (Ferguson M 1989). Scintigraphy provides a means of identifying individuals with gland dysfunction who retain portions of functional parenchyma and may be amenable to treatment to increase salivary output.

5.5.5 Sialography

Sialography has been used in investigating sialiectasis and ductal changes (Ferguson M 1989). Sialography utilises a radio-opaque material injected in a retrograde fashion into the salivary duct system to define gland anatomy (Fox 1989). Another development in sialography is the technique of the continuous infusion of the pressure monitored sialography using aqueous media (Ferguson M 1989). Certain patterns of sialograms are suggestive of specific disorders. Sialography gives little information as to the degree of gland function but is invaluable in demonstrating gland masses or sialolitis (Fox 1989). Disadvantages include technical difficulties, as duct cannulation is required, invasiveness, and the possibility of both acute and chronic reaction to the contrast material.

5.5.6 Ultrasound, magnetic resonance imaging and computerised tomography

Ultrasound, computerised tomography and magnetic resonance imaging have been used to study salivary gland pathology. While there have been favourable reports for specific situations, these methods require further study before their utility or superiority is proven.

5.5.7 Gland biopsy

Gland biopsy is a useful technique. Gland biopsy can be done for minor and major salivary glands.

The labial minor gland biopsy is best established using the vertical or preferably horizontal incisions. In this technique (Ferguson M 1989), 4 to 6 lobes of the minor gland must be taken for inflammatory

exocrinopathy, because there is considerable variation from one lobe to the other. The changes sought are inflammatory infiltrates, acinar destruction, large dilated ducts containing hardened mucoid material and sometimes fibrosis. Ferguson M (1989) has also discussed parotid biopsy which is indicated for patients with parotid swelling where a lymphoma is suspected or in a patient with a cardiac history who would be vulnerable to infectious endocarditis if a labial biopsy was done because a wound on the labial mucosa may not always heal well and repeated episodes of bacteremia may occur. This procedure is less uncomfortable than minor gland biopsy.

5.5.8 Haematology and serology tests

These tests are useful as screening tests in connective tissue disorders and systemic disorders like diabetes mellitus. Ferguson M (1989) has suggested for connective tissue disorders the broad screen of the erythrocyte sedimentation rate and the blood film. The rheumatoid factor, the antinuclear factor, and the dna antibodies should be considered to look for connective tissue disease. Ferguson M (1989) has also suggested that diabetes mellitus could be screened by looking at a random plasma glucose, a fasting plasma glucose, and/or a glucose tolerance curve, and a urine analysis for glucose.

5.6 Management

By the time many patients seek help for xerostomia their problem may become well established and be the cause of considerable distress. While the relief given to many sufferers is limited, patience and consideration, especially towards the elderly can be as important as any positive steps that may be taken. Of equal importance is the need for regular review, which as well as enabling the progress of the condition to be monitored, provides the continuity of care and support which is a great help to many patients (Levine R 1989).

Ferguson M (1989) suggested that in the management of patients who suffer from chronic xerostomia, it is first necessary, to objectively

test whether the patients subjective feeling of dryness is supported by a reduced salivary flow rate. It is important to determine the etiology of the salivary gland hypofunction. According to Ferguson M (1989) the dentist should treat the patient with an established diagnosis of dry mouth by adopting an aggressive active program of management, considering routine oral hygiene measures, diet, salivary substitutes, sialagogues, dental and psychological treatment.

5.7 Therapy

The basic principles of therapy are the elimination or correction of accessible etiological factors, the stimulation of natural salivary flow or its replacement with an artificial saliva, and adjunct therapy for xerostomia must be included. Treatment of xerostomia is difficult and frustrating and a multifactorial approach may be successful (Ettinger 1981). Major components of a successful protocol for xerostomic patients is given in Table 26. The sequence for xerostomia protocol is presented in Table 27.

Table 26 Major components of successful protocol for xerostomia patients.

Source: Wright (1987).

Early diagnosis of salivary gland dysfunction problems.

Program to inform the individual about their present oral status and the potential oral and systemic consequences of xerostomia.

Referral system to trained professionals for special oral/systemic evaluations and utilisation of protocols to improve salivary gland function.

Effective preventive and palliative regimes for patient relief and oral tissue protection.

Maintenance recall schedule to assess compliance and to alter treatment regimes as needed.

The correction of accessible etiological factors and adjunct therapy will be discussed below. The stimulation of salivary flow will be discussed in Chapter 6.

Table 27 Sequence for xerostomia protocol.
Source: Wright (1987) .

Initial oral examination and treatment planning :

- Radiographic survey
- Soft and hard tissue assessment
- Clinical evaluation of present or potential salivary gland dysfunction. .
- Oral prosthesis evaluation.
- Treatment consultation with patient and family.
- Request special salivary and systemic test when appropriate.
- Prescribe or dispense palliative agents to relieve dry mouth (artificial saliva, mild baking soda solution mouthrinse, pocket size water dispenser, lip moisturizer).
- Prescribe topical anaesthetic, special antimicrobial agents as diagnosis indicates.

Initial orientation and treatment appointment by dental hygienist.

- Present slide program to inform patient about deleterious sequelae of salivary dysfunction.
- Calculus removal, root planing, tooth polishing.
- Take impressions to construct fluoride carriers (all patients receiving radiation treatments involving salivary gland areas, Sjogren's syndrome, and other selective individuals with salivary dysfunction).
- Distribute information pamphlets to oncology patients.

Follow-up dental hygiene appointment:

- Personal oral hygiene instruction (brush, floss, use of baking soda paste).
- Dispense and demonstrate use of fluoride gel (carrier or brush on techniques).

Dental treatment appointment:

- Provide a dental therapies to eliminate/control oral infections and other conditions that may lead to episodic problems (extractions, endodontic therapy, restorations, prostheses adjustments, smooth sharp tooth cusps, wax-coat or remove sharp orthodontic fixtures).

Maintenance evaluations and supportive prevention reinforcement:

- Re-evaluation and modulation of management agents/regimes are done as required.
- Conservative dental treatments are performed as oral and medical conditions permit.
- Emphasis is placed on life-long management concepts of the salivary gland dysfunction problems.

5.7.1 Elimination or correction of accessible etiological factors

This depends upon the cause of xerostomia but much can often be achieved, especially where the cause is multifactorial. Cases complicated by iron deficiency and hormonal changes have responded well to replacement therapy, while drug induced xerostomia may rapidly improve when the medication has changed (Levine R 1989). The treatment of Sjogren syndrome is based on the use of systemic steroids and good results are often obtained (Ettinger 1981).

5.7.2 Adjunct therapy for xerostomia

The treatment of xerostomia involves clinical management of both the general discomfort and any associated disease process such as dental caries, periodontal disease and candidiasis often found in patient suffering from xerostomia (Ettinger 1981). Strict attention should be given to involving the patient in a preventive dental program that includes brushing and flossing instruction, fluoridation of teeth, the use of remineralising solutions, dietary advice, monitoring and control (Ferguson M 1989). While there are common principles to the management of all cases of xerostomia each case calls for careful assessment and an individual treatment plan embracing all aspects of whole mouth care. In all cases regular review and encouragement is essential to the patients wellbeing. As the maintenance of good plaque control is often a problem, the assistance of a dental hygienist is of great value. Cervical and root caries must be arrested by rigorous attention to diet, plaque control and use of locally applied fluoride solutions or gels together with a fluoride mouth rinse (Levine R 1989). When restoration of such lesions is required, glass ionomer is the material of choice because of its fluoride content. Once a satisfactory regime has been established; and the symptoms controlled, the review period can be lengthened, however, regular and careful examination must be maintained to guard against a relapse.

Prevention: Before starting to look for the cure, the clinician must consider prevention. There has been some work in radiotherapy in modifying beam geometry. To spare one parotid without compromising treatment of the neoplasm. Thus, reasonable function in at least one parotid could be maintained. This technique has afforded considerable symptomatic relief to patients (Ferguson M 1989). Ferguson M (1989) compared the data in his studies between spared and irradiated sites, using maximal parotid flow rates. He found a mean value of 1.11mL/min compared with 0.28mL/min which confirms that the parotid has been protected.

Due to the many microorganisms found in the mouth, appropriate dental care can reduce the potential for infection during periods of chemotherapy-induced bone marrow suppression (DePaola et al 1986). Programs for oral hygiene should be discussed with these patients and reinforced at regular intervals. A dry mouth is made more bearable by good oral hygiene, which should be encouraged by careful instruction and then carefully be assessed and the instruction reinforced as and when necessary (Levine R 1989).

Such patients often have a number of other systemic problems and require considerable support and guidance with oral hygiene measures. The dentist must consider the difficulties in each individual case. For disabled cases with crippling rheumatoid arthritis, toothbrush handles can be modified and irrigators are helpful. There seems little point in giving severely arthritic patients detailed instructions on flossing when they cannot get the floss near the teeth (Ferguson M 1989).

As xerostomia can predispose patients to rapidly destructive caries and periodontal disease, intensive preventive measures must be taken for dentate patients as well as dental health education aimed at reducing the cariogenicity of the diet and improving the plaque removal. The patient should be given a clear and simple account of

the effect of xerostomia on the teeth and gingival tissues, so that the need for good oral hygiene is appreciated. This should be followed by dietary advice aimed at reducing the intake frequency of sugars including glucose and fructose (natural fruit sugar) which is often used as a sucrose substitutes in foods and drinks (Levine R 1989). Full denture wearers should be instructed to keep the dentures and especially the fitting surfaces clean and not to wear them at night (Ettinger 1981).

The other measures to be considered are use of a chlorhexidine mouthwash as a means of chemical plaque control. While it has the disadvantage of staining enamel and composite restorations, it can be most useful short-term means of reducing plaque formation, especially for patients with sore mouths immediately following radiotherapy. However, it may be unacceptable for long term use (Levine R 1989).

Topical fluoride: The role of topical fluoride treatments in the prevention of dental caries can be successful for patients experiencing dry mouths (Daly 1990). Levine R (1989) suggested the use of topical fluoride in addition to the use of a fluoride toothpaste. This is most conveniently provided by a daily rinse with 0.05 per cent sodium fluoride mouth rinse which is accepted as an effective method of caries control. Meyerowitz et al (1991) did a study to evaluate the effect of twice daily application of 0.05 per cent sodium fluoride mouth rinse on demineralisation and remineralisation in the oral cavities of subjects suffering from radiation-induced hyposalivation. The results of this study suggest that a twice daily mouth rinse with 0.05 per cent sodium fluoride can prevent demineralisation and enhanced remineralisation in subjects with radiation-induced hyposalivation.

While many different fluoride preparations are available for surgery use, fluoride application by patients at home may involve the use of fluoride toothpaste, mouthwashes or gels. The fabrication of a custom

fitted mouth guard provides one effective method for the home application of fluoride gel. The mouth guard should be designed in such a way as to ensure the efficient application of fluoride gel to the cervical and interproximal areas of the teeth as well as to the gingival margins. This aids in the prevention of caries and gingivitis (Daly 1990). The following topical fluoride treatment has been quoted by Daly (1990).

Preliminary treatment:

The objective is to achieve the highest possible level of oral health. The basic regime for dry mouth patients is:

1. Carry out necessary restorative and periodontal treatment.
2. Give patient detailed oral hygiene and dietary instructions.
3. Take impressions for mouthguards.

Home care:

1. Thorough plaque removal daily and use of fluoride toothpaste.
2. Use fluoride gel in mouthguards (5 min each arch) every 1 to 2 days.
3. Rinse with 0.2% chlorhexidine mouth wash once daily.

Follow-up surgery care:

1. Review patient at least every two months.
2. Scale and polish teeth.
3. Monitor oral hygiene and oral diet.
4. Carry out restorative repair as necessary.

Topical fluorides and general prescribing guidelines for fluorides in the USA have been quoted from Ettinger 1991 as follows;

Topical fluorides:

- a. Sodium Fluoride (0.05% NaF) low potency rinse - over the counter (OTC) rinse with 10 mls daily after cleaning teeth nothing post operatively (NPO) 1/2 hour.
- b. Sodium Fluoride (0.2% NaF) high potency rinse - Rx rinse with 10 mls daily after cleaning NPO 1/2 hour.
- c. Fluoride Home-Use Gels - once or twice daily - Rx
 - i. stannous fluoride 0.4% gel (Gel-Kamp)

- ii. acidulated phosphate fluoride 0.5% gel (Karigel)
- iii. neutral sodium fluoride 0.5% gel

GENERAL PRESCRIBING GUIDELINES FOR FLUORIDES

- A. Stannous fluoride products have the same fluoride ion content as regular fluoride toothpaste (1,000 ppm). However, the tin ion in stannous fluoride binds to plaque allowing the fluoride/tin compound to exert anti-streptococcus mutans activity for 8 hours. Therefore, the primary usefulness for stannous fluoride is in periodontally involved area with major plaque removal and bacteria problems.

eg.: For anti-bacterial/anti-plaque therapy.

RX : Omnigel 0.4% (or any stannous fluoride gel)

DSP: 7oz.

SIG: Brush with 1/2 inch strip after cleaning teeth twice daily.
Spit out excess. NPO 1/2 hour.

- B. Acidulated phosphate fluoride products have five times the fluoride ion concentration of regular fluoride toothpaste (1000 ppm). The active fluoride compound in acidulated phosphate fluoride is sodium fluoride. The addition of the phosphoric acid promotes fluoride uptake by reaching the pH threshold for demineralisation. Long term fluoride retention in enamel is probably best with acidulated phosphate fluoride. Initial uptake is no better for acidulated phosphate fluoride than for neutral sodium fluoride. Because the pH of home-use acidulated phosphate fluoride gels is 5.6, they should not be repeatedly applied to porcelain or glass ionomer surfaces as the restorative material may acid etch and become rough nor should they be used with patients with mucositis or xerostomia.

Example: Overdenture Abutment Teeth.

RX : Karigel 0.5% (any acidulated phosphate fluoride gel listed above).

DISP: 30mL

SIG: Place one drop in each depression after cleaning teeth daily or every other day, NPO 1/2 hour.

RX: Omnigel 0.4% (or any stannous fluoride gel listed above)

DISP: 7oz

SIG: Place one drop in each depression after cleaning teeth daily or every other day. NPO 1/2 hour.

- C. Sodium fluoride products have five times the fluoride ion concentration of regular fluoride toothpaste (1000 ppm). Uptake into demineralised concentrations is very rapid. However, the prolonged fluoride content of the lesion may be lower than those treated with acidulated phosphate fluoride (APF). Neutral sodium fluoride products are useful for patients who need remineralisation but cannot tolerate the

acidic pH of APF due to sensitive oral tissue lesions, or the presence of porcelain or glass ionomer restorative materials. In the severely xerostomic patient neutral sodium fluoride gel may be the product of choice because these patients tend to stain with stannous fluoride and may be too irritated with acidulated phosphate fluoride.

Begin with b.i.d. use. After two to three months, monitor on once daily application.

NOTE: APF SHOULD NOT BE USED ON GLASS IONOMER OR PORCELAIN MATERIAL.

Diet: Attention must also be given to the diet which should be palatable, edible and nutritious. These patients often prefer moist and particularly greasy foods. It is important to have a dietitian council these patients, looking at individual preferences and what the patient is eating and then try to establish a diet adequate for the individual. Some of these patients may not be particularly well and the diet must be practical and within their ability to prepare (Ferguson M 1989). Ettinger (1981) has suggested that patients should be advised to avoid;

- a. Dry and bulky foods.
- b. Spicy and acidic foods
- c. Alcoholic beverages.
- d. Carbonated beverages.
- e. Tobacco.

A high fluid intake should be encouraged unless it is medically contraindicated such as for patients being treated with diuretics.

Environmental factors: Maintenance of optimal air humidification in the home is useful especially during sleep. Vaseline can be used to protect the lips (Wright 1987).

Dental: Dentures should be checked and adjusted for any irritations they may be causing. The patient must visit the dentist to make sure that all sharp cusps of teeth and irregular fillings are smoothed (Wright 1987). Periodic oral prophylaxis, fluoride therapy and oral hygiene instructions should be given to the patient (Daly 1990).

Temporary palliation: Mouth washes can be useful to alleviate oral discomfort. Those containing glycerine and methyl cellulose are easy to make up. A suitable mouth wash consists of methyl cellulose 4 per cent, 20 cubic centimeters. Glycerine 10 cubic centimeters, and distilled water with one drop of lemon oil 60 cubic centimeters. The methylcellulose powder dissolves directly in the glycerine (Ettinger 1981). For patients who have had therapeutic radiation or are receiving chemotherapy and are edentulous, mouth rinses with diluted milk of magnesia or sodium bicarbonate are helpful in the debridement of epithelium and the relief of pain due to denuded mucosa during the stage of mucositis (Ettinger 1981). For relief of pain lidocaine 2-5 per cent or diclonine 0.5-1 per cent may be used (Ferguson M 1989). Christensen (1991) has discussed special oral care for seriously ill and unconscious patients. This special oral care is aimed to keep the mouth clean because it is known that these patients, owing to their poor general conditions, are prone to mouth and throat infections. Secondly it is desirable to prevent drying up of the oral mucosa. Fever, breathing through the mouth and certain drugs can cause dryness in the mouth of such patients. This is extremely uncomfortable for the patient and at the same time it reduces the natural resistance of the mucosa to infections. The treatment consists of 0.1 per cent chlorhexidine solution to be rubbed all over the oral mucosa and teeth. One hour after the chlorhexidine is applied a liquid lubricant consisting of glycerol water and lemon juice is rubbed all over the oral mucosa about once every hour to protect the mucous membrane from drying out. However since the liquid lubricant contains lemon juice this treatment should not be used continuously for many months on patients with their own teeth as acid solutions can damage natural teeth.

Treatment of candidiasis: Oral candidiasis is a common finding in patients suffering from xerostomia. Table 28 shows the various forms of oral candidiasis.

Table 28 **Various forms of oral candidiasis.**
Source: Reichl (1990).

Common forms:

Acute pseudomembranous candidiasis (thrush).
 Acute atrophic candidiasis (antibiotic candidiasis).
 Chronic atrophic candidiasis (denture sore mouth).
 Angular cheilitis (perleche).

Rare forms:

Chronic mucocutaneous candidiasis.
 Candida leukoplakia (precancerous).
 Median rhomboid glossitis.
 Profound immunodeficiency mucocutaneous candidiasis (AIDS).
 Juvenile juxtavermillion candidiasis.
 Dentinal candidiasis.

The dental management of the different categories of oral candidiasis patients is presented in **Table 29**.

Table 29 **Dental management of different categories of OC patients.**
Source: Reichl (1990).

1. When obvious predisposing conditions are present, appropriate treatment can be provided solely by the dentist.
 Examples are patients with the following histories :
 - Prior radiation therapy
 - Extended antibiotic usage
 - Steroid treatment
 - Immature immune systems (infants)
 - Diabetes
 - Poor dentures
 - Drug-induced xerostomia
2. When no obvious predisposing condition exists, referral for a medical evaluation should be initiated. Examples include :
 - Suspected HIV positive patients.
 - Potentially undiagnosed diabetes mellitus
 - Xerostomia of unknown origin.
3. Any patient from category 1 that is refractory to conventional treatment. In such cases, a medical evaluation is recommended.

Patients suffering from xerostomia, who wear dentures, often suffer from denture sore mouth (chronic atrophic candidiasis). Ettinger (1990) suggested a ten day course of topical polyene antibiotics such as nystatin or Amphotericin B which are the drugs of choice. Denture associated stomatitis, is believed to be caused by a hypersensitivity reaction of the tissues to the toxins released by *Candida albicans* situated on or in the denture, thus the dentures must also be treated. Denture disinfection with an antifungal solution is best carried out by soaking the dentures for half an hour in either 2 per cent sodium hypochlorite, 1:750 dilution of benzalkonium chloride or 0.2 per cent to 2 per cent chlohexidine gluconate. Benzalkonium chloride is a disinfectant of choice (Ettinger 1989). It is essential that a fresh solution be used for soaking the denture each day as gram negative rods can proliferate in this disinfectant within 24 hours. The patient may also suffer other forms of candidiasis like angular cheilitis and thrush. The medications for treatment of oral candidiasis are presented in Table 30.

Table 30 **Medications for treating oral candidiasis.**
Source: Reichl (1990).

1. Topical antifungal drugs:

Nystatin (Mycostatin) oral suspension 100,000 U/mL.

Disp: 60mL

Sig: Take 2-5 mL four times daily; hold in mouth 2 minutes and swallow, and/or soak prostheses overnight.

Nystatin ointment 100,000 U/gm

Disp: 30 gm.

Sig: Apply liberally to effected areas 4 times daily.

Nystatin pastilles (Mycostatin Pastilles) 200,000 U/troche.

Disp: 60

Sig: Dissolve one in mouth four times daily.

Clotrimazole (Mycelex) troches 10 mg

Disp: 70

Sig: Dissolve 1 in mouth five times daily.

2. Systemic antifungal drug:

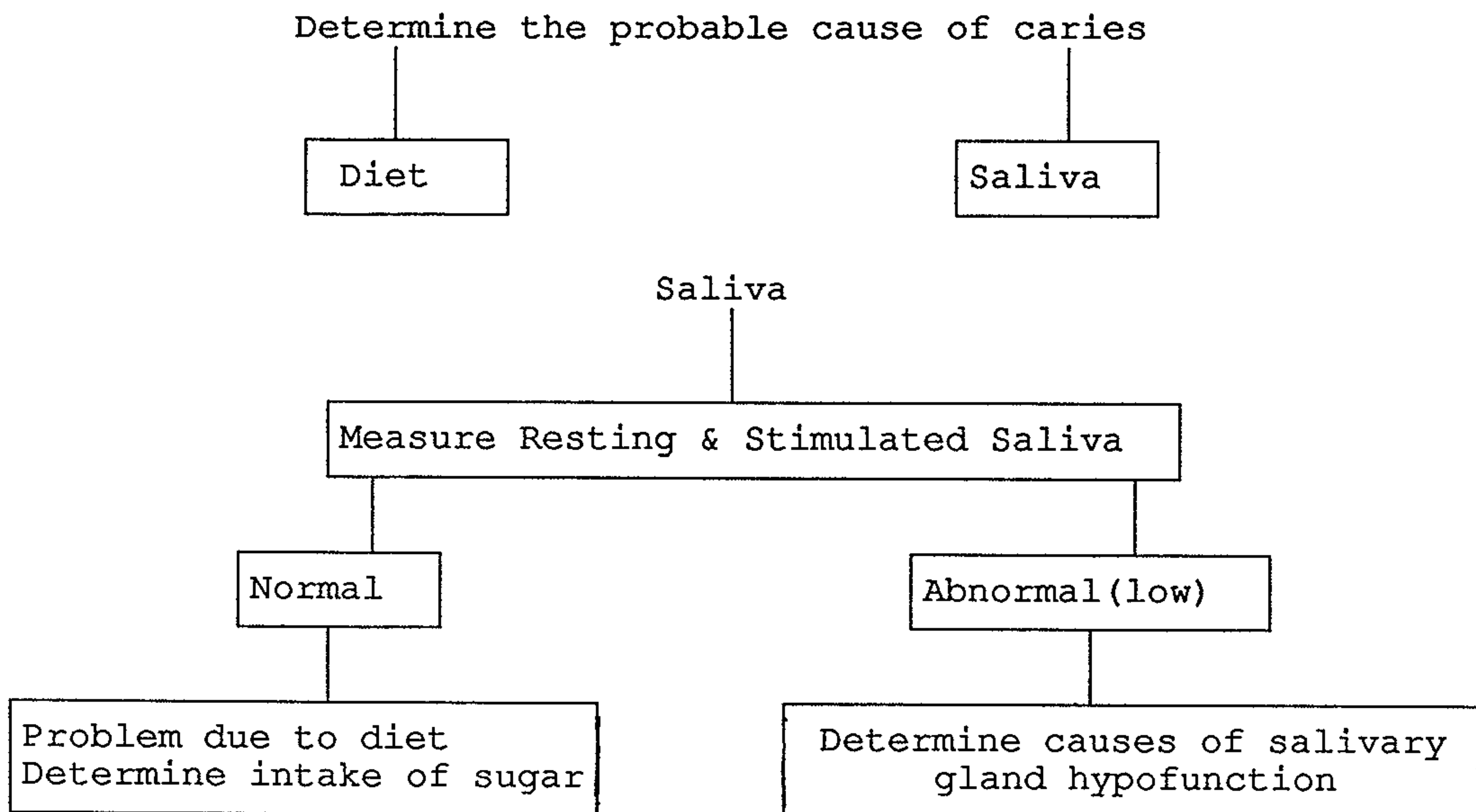
Ketoconazole (Nizoral) tablets 200mg.

Disp: 20.

Sig: Take one daily.

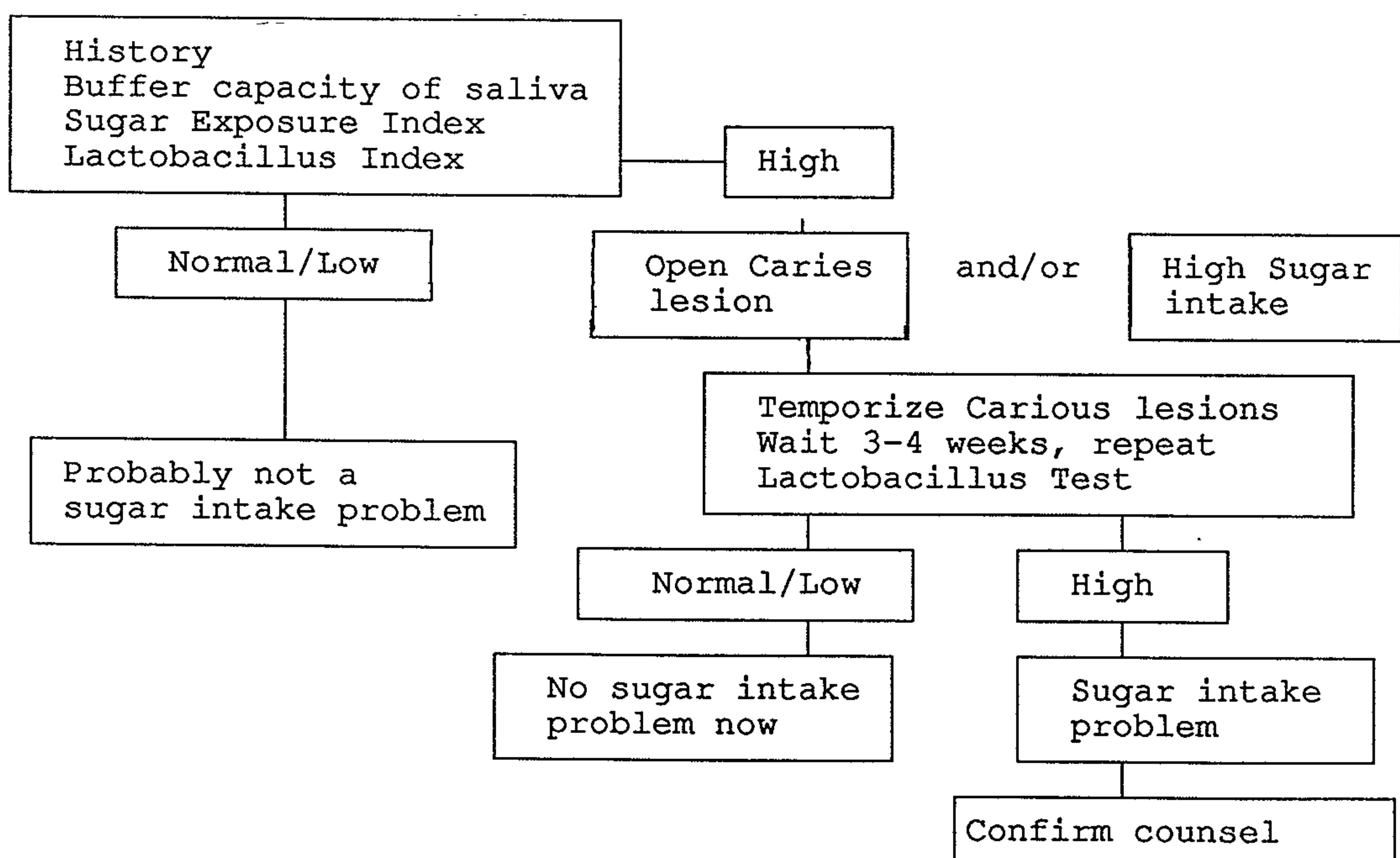
Management of the caries active patient: Xerostomia is related to a high incidence of dental caries (Kidd 1987). This is seen most dramatically in patients who have had radiotherapy in the region of the salivary glands for malignancy. If strenuous attempts are not made to prevent disease, rapid carious destruction of the dentition can occur. A low salivary secretion rate leads to reduced elimination of micro-organisms food remnants and to a reduced tendency to remineralisation or early enamel lesions. In addition there is an increased number of streptococcus mutans and lactobacilli (Kidd 1987). The increased caries activity seen in persons with reduced salivary flow is hardly surprising in view of the dietary changes, the reduced host resistance, and the overwhelming microbial challenge. A low salivary buffer capacity has been linked with high caries activity (Krasse 1985). Since caries is a disease with several causes the approach to its management will have several facets (Newbrun 1989). It will include dietary control, the judicious use of fluorides to slow down lesion progression, The reduction of dental plaque through mechanical removable and chemical prevention of its formation. It is a good practice to stabilise large lesions with temporary restorations but definite restorative dentistry should be deferred until the cause of the disease has been treated (Kidd 1987). It is necessary to be aware that fillings do not prevent further disease, new lesions may form adjacent to the restorations or the fillings may leak allowing caries to progress beneath them. Any restoration in a mouth where caries is not controlled is a "pathway to the pulp" (Kidd 1987). The management of a caries active patient is presented in **Figure 9**. The determination of sugar intake is presented in **Figure 10**.

Figure 9 Management of the caries-active patient.
Source: Sreebny (1990).



(See Figure 10)

Figure 10 Determine sugar intake.
Source: Sreebny (1990).



Psychological treatment: People with persistent dry mouth can be compared to people with chronic pain and should be treated accordingly. Ferguson M (1989) has suggested that the patient should be referred to a psychiatrist and the symptoms that are depressing them should be discussed. The psychiatrist should try and find out if there are any psychological events which would aggravate this factor. This is a beneficial and supportive environment for people with chronic discomfort.

In summary an approach to managing the patient with persistent dry mouth begins with considering the etiology, followed through by investigative steps and is culminated in the development of the treatment plan, utilising currently available therapies.

6. SALIVARY FLOW AND DENTAL HEALTH

Saliva is categorised as resting (unstimulated basal) or stimulated. Basal saliva refers to the saliva present in waking subjects when they are at rest and the glands are under minimal stimulation. Stimulated saliva refers to secretions induced by external stimuli mainly mastication (Sreebny 1989).

Unstimulated flow rates vary widely between subjects and those with low rates do not always have symptoms of dry mouth (Ferguson M 1989). The main stimulus to salivation is the sensation of taste (particularly acid). Stimulated saliva is better able because of changes in its composition to prevent demineralisation and to favour remineralisation than is unstimulated saliva (Jenkins 1990).

6.1 UNSTIMULATED SALIVA

Unstimulated saliva is usually collected with the patient sitting quietly, with the mouth open to allow the saliva to collect into a beaker or similar receptacle over a given time (Sreebny 1989). Alternatively, the patient can spit out the saliva at regular intervals, while swallowing is inhibited. Dawes (1987) has done studies on unstimulated salivary flow rates on healthy individuals. Table 31 shows results of these studies of the flow rates of unstimulated saliva in man.

Table 31 Results from several studies of the flow rate of unstimulated saliva in man (mL.min).
Source: Dawes (1987).

Authors	Type of Saliva	n	Gender	Age	Mean	SD	Range	Percentile	
								10%	90%
Anderson et al (1974)	Whole	100	M & F	10	0.39	0.21	0.10-1.24	0.17	0.67
		96	M & F	13	0.39	0.22	0.05-0.90	0.15	0.70
Becks & Wainwright (1943)	Whole	661	M & F	5-95	0.32	0.23	0.01-1.85		
Heintz et al (1983)	Whole	286	M	15-74	0.36		0.05-2.75	0.15	0.73
		343	F	15-74	0.26		0.03-1.15	0.09	0.60
Shanon (1967)	Paro-tid	4589	M	17-22	0.040	0.031			
Enfors (1962)	Subma ndibu lar.	54	M & F	20-59	0.10		0-0.39		

The average unstimulated flow rate is 0.4mL/min (Sreebny 1989), but the normal range is very large and includes individuals with very low flow rates who do not complain of dry mouth (Edgar, O'Mullane 1990). Such a broad normal range makes it difficult to identify an individual who has abnormally low flow rates. Unless saliva is completely absent one can identify a patient to have a dry mouth only on subjective symptoms. A patient can complain of dry mouth even when salivary flow is easily measured and this complaint may be due to localised regions of dry mucosa which are most likely to occur in the anterior palate region where no major or minor salivary gland secretions enter the oral cavity. Edgar and O'Mullane (1990) report a study done on dental students on salivary flow. The students were asked to note when the onset of dry mouth symptoms began. On average, the symptoms started when salivary flow had fallen by 40-50% of the normal value-despite wide variation in flow rates. Thus it can be suggested that it is the change in the amount of saliva rather than the absolute amount which is important.

In true xerostomia, it is impossible to collect any saliva by conventional means. For such patients salivary function can be monitored by using paper strips, which are applied to the minor mucous glands. The amount of saliva can be estimated using a periotron, an instrument which measures the conductivity of the paper strip (proportional to the amount of fluid), whose main use is in monitoring gingival crevice fluid (Sreebny 1989).

Whether a particular flow rate is high or low is much less important than whether it has changed adversely in a particular individual (Edgar, O'Mullane 1990). Unfortunately dentists do not routinely measure salivary flow rates and when a patient complains of having low salivary flow, it is impossible to judge whether there is a genuine reduction in the salivary flow rate. It would, therefore, be advantageous if dentists included measurement of salivary flow as a part of their regular examination.

6.2 FACTORS AFFECTING UNSTIMULATED SALIVARY FLOW RATE

Dawes (1987) has reported many factors that influence the unstimulated salivary flow rate. These are presented in **Table 32**.

6.2.1 Hydration

This is potentially the most important factor. When body water content is reduced by 8 per cent, the salivary flow rate decreases to virtually zero (Dawes 1987). For a man of about 70Kg, comprising about 50Kg of water, 8 per cent dehydration means a loss of 4 litres. In contrast, hyperhydration will increase the salivary flow rate (Edgar, O'Mullane 1990).

6.2.2 Body posture

Flow rate also varies with position (Sreebny 1989). A person when standing or lying will have a higher or lower flow rate respectively than when seated (Dawes 1987). Flow rate also greatly decreases in the dark, but decreases with cigarette smoking and increases with olfactory stimulation (Sreebny 1989).

Table 32 Factors influencing the unstimulated salivary flow rate in healthy subjects.
Source: Dawes (1987).

Important factors	Unimportant factors
1. Degree of hydration	1. Gender ?
2. Body position	2. Age (above 15)
3. Exposure to light	3. Body weight
4. Olfaction	4. Gland size
5. Smoking	5. Psychic effects
6. Previous stimulation	a) Thought of food
7. Circadian rhythms	b) Sight of food
8. Circannual rhythms	c) Appetite
9. Drugs	d) Mental stress.
	6. Degree of functional stimulation.

6.2.3 Biological rhythm

The flow rate of saliva peaks (acrophase) during the afternoon and drops to almost zero during sleep (Schneyer et al 1956). It is therefore important to standardise the time of day at which saliva is collected (Dawes 1987).

A study carried out by Shannon (1966) has shown a circannual rhythm in the flow rate of parotid saliva with a peak value in winter and lower flow rates in summer.

6.2.4 Drugs

A frequent association of dry mouth and drug utilisation results from iatrogenic interference with salivary control mechanisms normally mediated by both parasympathetic and sympathetic innervation (Widdop 1989). Any agent capable of interfering with the actions of acetylcholine on muscarinic-cholinergic type receptors or nor-epinephrine on alpha-or beta-adrenergic receptors, disrupting the autonomic pathways, will have potential to disturb the production of saliva as a secondary potential to disturb the production of saliva as a secondary effect (Widdop 1989). Many drugs have the capacity to

block neurotransmitter action to depress activity in the brain's salivary centre or both. There are over 430 standard drugs with xerostomic potential as well as nicotine and alcohol (Bahn 1972, Sreebny, Schwartz 1986).

6.3 STIMULATED SALIVA

Stimulated salivary flow in healthy individuals show a wide variation (Dawes 1987). Several studies have been done on stimulated salivary flow rates the results of which are shown in **Table 33**. Unfortunately these studies use various stimuli, with an international agreement on a particular stimulus a valid comparison of the different studies could have been made.

Table 33 Factors influencing salivary flow rate and composition.
Source: Edgar, O'Mullane (1990).

Authors	Type of saliva	Stimulus	Sample number	Mean (mL/min)	SD
Heintze et al. (1983)	Whole	Paraffin wax	629	1.6	(2.1)
Mason et al (1975)	Parotid	Lemon Juice	169	1.5	(0.8)
Shannon et al (1974)	Parotid	Grape candy	368	1.01	(0.46)
Ericson et al (1972)	Submandibular	1% citric acid	28	0.79	(0.38)
Shannon and Frome (1973)	Whole	Chewing gum	200	1.69	(0.57)

6.4 FACTORS INFLUENCING THE STIMULATED FLOW RATE

The maximum flow rate of stimulated whole saliva is 7mL/min. Many factors influence the stimulated flow rate in man and these are listed in **Table 34**.

Table 34 **Factors affecting the flow of stimulated saliva.**
Source: Edgar, O'Mullane (1990).

Nature of stimulus
Unilateral stimulation
Gland size
Gag reflex

6.4.1 Mechanical stimulus

The action of chewing, in the absence of any taste can stimulate salivation, although the flow rate will be low relative to maximum stimulation with citric acid (Edgar, O'Mullane 1990). During mastication the food in the mouth is mixed, thus increasing the distribution of saliva.

6.4.2 Gustatory stimulus

Acid is the most potent of the four basic taste stimuli, the others are salt, bitter and sweet (Dawes 1987). Edgar, O'Mullane 1990 reported a study done on various concentrations of citric acid which found that 5 per cent citric acid would stimulate maximum salivary flow rate. Sreebny (1989) has suggested the 2 per cent solution of citric acid be used for clinical evaluation of a xerostomic patient.

6.4.3 Unilateral stimulus

If a person habitually chews on one side most of the saliva will be produced in the gland on one side unless gustatory stimulation is also present (Edgar, O'Mullane 1990).

6.4.4 Gland size

Stimulated salivary flow rate is directly related to gland size. However, unstimulated flow rate is not related to gland size (Sreebny 1989).

6.4.5 Aging

It is not clear how the aging process influences the salivary flow rate. Some investigators have shown that in healthy unmedicated

persons the flow of whole saliva (Parvinen, Larmas 1981) and parotid saliva (Heft, Baum 1984) does not change with age. Heft and Baum (1984) did studies on unstimulated and stimulated parotid salivary flow rates in individuals of different ages. Both unstimulated and stimulated parotid saliva samples were collected from healthy, unmedicated individuals between the ages of 23 and 81 years. There were no significant difference in flow rate related to age with either unstimulated or stimulated secretion. The exception to this may be post menopausal women, who demonstrate a slightly reduced rate of secretion (Sreebny 1989).

Submandibular and minor salivary gland flow on the other hand may decrease with age (Gandara et al 1985). Pedersen et al (1985) studied age dependent decreases in human submandibular flow rates as measured under resting and post stimulation conditions. Submandibular saliva samples were obtained with a collection device, under resting and post stimulation conditions, for 28 healthy individuals between 70 and 91 years of age and from 30 healthy individuals between 18 and 39 years of age. The salivary flow rates were significantly decreased in the age group compared with the controlled group. The mean resting and post stimulation flow rates for the aged group averaged 22 per cent and 39 per cent respectively of control values.

Other investigators maintain that the flow of whole saliva declines with age (Navazesh, Milligan 1989). Sreebny (1989) did studies on the relationship of the subjective feeling of oral dryness to age. Data from this study suggests that one out of every seven adult patients may suffer from some degree of salivary hypofunction. It is more frequently observed in older subjects (40% of those aged over 65 years). However it is present in adults of all ages.

The reasons why so many people do not complain of xerostomia are complex. Some believe that this is a normal feeling, some are bothered about so many sytemic complaints that, by comparison, the

mouth seems unimportant and some feel that it is an inevitable consequence of the aging process. Studies have shown that with the possible exception of post menopausal women there is no loss in salivary function with aging (Sreebny 1989, Edgar, O'Mullane 1990).

6.4.6 Food

Edgar, O'Mullane (1990) have reported a study that tested the effects of seven foods. Results from this study showed that even from the most bland food (boiled rice) elicited 43 per cent of the maximum flow rate produced by 5 per cent citric acid. Rhubarb pie, which is both acidic and sweet elicited 70 per cent maximum flow rate. The study showed that it was the gustatory stimulus provided by the food rather than the mechanical stimulus of chewing, which was responsible for this relatively high flow rates. In comparison with other foods, chewing gum elicits a low flow rate because most chewing gums provide a sweet stimulus, which is generally the least effective of the taste stimuli. Initially the flow rate is raised but as the flavour and sweetness leaches out, only the mechanical stimulus remains and the flow rate falls.

However studies done by Jensen, Wefel (1989) show that if chewing gum is chewed for a long time, usually 20-30 minutes, it can be beneficial in buffering and clearance of carbohydrates.

6.5 UNSTIMULATED FLOW RATE AND ORAL HEALTH

Unstimulated flow rate is more important than stimulated flow rate for general oral health (Dawes 1983). A study done by Jenkins, Edgar (1989) has shown that sugar-free chewing gum given to students over a long period of time produced a small rise in the unstimulated but not stimulated flow rate. The study suggested that if salivary glands were stimulated even in xerostomia, the activity of the salivary gland may be increased.

6.6 TOTAL DAILY SALIVARY FLOW

The average unstimulated flow rate over a waking period of 16 hours is about 0.3-0.4mL/min or a total of about 300mL of saliva (Edgar, O'Mullane 1990). For paraffin stimulated whole saliva it is approximately 2mL/min (Sreebny 1989). Approximately 5 per cent of the population demonstrates stimulated flow rates less than 0.7mL/min (Parvinen, Larmas 1981). During sleep the maximum flow will fall to 0.1mL/min producing about 40mL of saliva (Sreebny 1989). Assuming an average of 8 hours of sleep per day and approximately two hours per day spent eating, resting saliva would be produced over a period of 14 hours. The data from studies done by Sreebny (1989) indicate that the calculated total daily flow of whole saliva is approximately 500 to 700mL/24 hours. This amount is considerably less than 1 to 1.5 litres per day cited in many physiology texts (Sreebny 1989, Edgar, O'Mullane 1990). The data from the 1989 Sreebny study also suggests that approximately 50 - 60 per cent of the daily output is derived from basal saliva. The profile of daily secretion of saliva is presented in Table 35.

Table 35 Profile of daily secretion of saliva.
Source: Sreebny (1989).

State	Hours/min	Secretion rate	Total (mL/day)
Sleep	8/240	0 mL/min	0
Unstimulated	14/840	0.4 mL/min	336
Stimulated	2/120	2 mL/min	240
Total			576
If stimulated period = 3 hours; total = 696 mL/day.			

6.7 SALIVARY FLOW AND TASTE

By the time the salivary secretion reaches the opening of the glands into the mouth it is very hypotonic compared with plasma (Edgar, O'Mullane 1990). The reason for this is probably to facilitate taste. Taste buds rapidly adjust to the taste of any solution in the mouth including saliva.

Unstimulated saliva is particularly well adapted to facilitate the condition of taste. Besides being low in sodium chloride it is also low in glucose, buffering capacity and urea (Edgar, O'Mullane 1990).

6.8 THE BUFFERING ABILITY OF SALIVA

Proteins: Saliva has a low level of proteins compared with plasma, therefore there are too few amino acids to have a buffering effect at the usual pH of the oral cavity (Edgar, O'Mullane 1990).

Phosphate ions: Saliva contains inorganic phosphates which contribute to its buffering capacity (Cole, Eastoe 1977).

Bicarbonates: Bicarbonates vary from less than 1 mmol/L in unstimulated saliva to almost 60 mmol/L at high flow rates of saliva (Edgar, O'Mullane 1990). Bicarbonates are the most important buffering system but only at high flow rates (Cole, Eastoe 1977). Therefore it is an important buffer against acid produced by dental plaque. However, in unstimulated saliva, the level of bicarbonate ions is too low to be effective (Edgar, O'Mullane 1990).

Salivary pH is dependent on bicarbonate concentration and an increase in bicarbonate concentration results in an increase in pH. At very low flow rates the pH can be as low as 5.3 rising to 7.8 at high flow rates (Edgar, O'Mullane 1990).

6.9 CALCIUM AND PHOSPHATE CONCENTRATION

Calcium and phosphate concentration maintain the saturation of saliva with respect to tooth mineral and are therefore important in calculus formation and caries development (Jenkins 1978). Saliva contains less calcium but more phosphate than does plasma. Different salivary glands have different concentrations of calcium and phosphate. Parotid saliva has less calcium but more phosphate ions than does submandibular saliva while the minor mucous secretions are very low in phosphate ions (Edgar, O'Mullane 1990). Calcium and phosphate concentrations greatly depend on the salivary flow rates. The higher

the flow rate the higher the calcium content in both the parotid and submandibular saliva. The calcium level in the submandibular saliva is higher at all flow rates than in parotid saliva. If the components of the ion products determining the solubility of tooth mineral are considered, all three components (that is calcium ion, phosphate ion and hydroxyl ion) increase with salivary flow. The higher the flow rate the more effective the saliva in reducing demineralisation and promoting remineralisation of teeth (Edgar, O'Mullane 1990).

6.10 MINOR MUCOUS GLAND SECRETIONS

These differ in two ways from the major gland secretions. They are very low in phosphate and they contain virtually no bicarbonate so they are very poorly buffered (Jenkins 1978). Although their influence on saliva as a whole may be small these glands may have a considerable effect on the environment of teeth and oral mucosa as their secretions are in intimate contact with them for long periods, undiluted by saliva from major glands when these are unstimulated (Jenkins 1978).

6.11 OLFACTORY AND PSYCHIC STIMULI

Generally olfactory stimuli are very poor in increasing salivary secretion relative to gustatory or mastication stimulation. Similarly thinking of food or seeing food are also poor stimuli (Dawes 1987). It may appear that one salivates at the thought of food. But it more likely that one merely becomes aware of the pool of saliva always present in the mouth. Unstimulated salivary flow is unaffected by visual stimuli (Jenkins, Dawes 1966). In general therefore the psychic effect of thinking about or seeing food has little effect on stimulating salivary flow (Dawes 1987).

6.12 SALIVA COLLECTION TECHNIQUE

Collection of whole saliva may be performed by a variety of techniques: drooling, spitting, suction and swab. All four methods provide similar information (Navaseth, Christensen 1982). The spitting and drooling techniques are simple and reliable. In the collection of saliva the most commonly used stimulants are paraffin

wax and citric acid. Other stimuli such as salt and rubber bands have also been used. The flow of saliva varies greatly among subjects and even in the same individual under different circumstances. It is important that the method of saliva collection is standardised. When observed over long periods of time the flow rate of one person tends to be fairly constant (Sreebny 1989).

Not all cases of salivary gland hypofunction are accompanied by oral dryness. There seems to be a cutoff point below which dry mouth is almost always present and above which it may or may not be present. This cut off point is about half the persons resting flow rate (Dawes 1987). People begin to complain about oral dryness when the resting flow rate is below 0.2mL/min (Dawes 1987). Resting flow rate below 0.1mL/min is considered abnormal. For stimulated saliva, the cutoff point is approximately 0.5mL/min. In order to achieve a whole saliva resting flow rate of 0.2mL/min there would have to be a reduction of at least 50 per cent in the overall salivary flow. Resting flow rates and a gland activity are presented in Table 36.

Table 36 **Resting flow rates and gland activity.**
Source: Sreebny (1989).

Gland activity	Flow rate (mL/min)
All glands functional	0.4
25% loss of activity	0.3
50% loss of activity	0.2
75% loss of activity	0.1
1 non functional parotid gland	0.36
2 non functional parotid glands	0.32
1 non functional SM/SL gland	0.25
2 non functional SM/SL glands	0.10
1 non functional parotid + SM/SL gland	0.21
2 non functional parotid + 1SM/SL gland	0.17

Sreebny suggests that a resting flow rate of 0.2mL/min of whole saliva could not be caused by local conditions (for example: sialoliths or

neoplasms). It is probably caused by systemic disorders that cause extensive damage to the glands or interfere with their blood supply or neural control.

Sreebny (1989) suggests the flow rate of saliva may readily be measured in the dental office. All that is needed is a quiet area, paraffin wax, a stop watch and a device to collect saliva and measure the amount secreted. Of these the most difficult is the quiet area. Since the flow of saliva is affected by a hormonally regulated circadian rhythm, the time of collection should be standardised for each patient. The values given for normal and abnormal salivary flow rates should represent a convenient index. They must be adapted and interpreted for each patient.

6.12.1 Collection of resting whole saliva

Sreebny (1989), has discussed the following technique for the collection of whole saliva. Patients are instructed not to swallow any of the secretions during the spitting technique collection procedure. For resting saliva, the patient is requested to sit quietly, tilt the head slightly down, keep eyes open and not move lips, tongue, cheeks or any other body part. Immediately before the test the patient is told to swallow any saliva which may be present and then allow the unstimulated saliva to gradually seep into the mouth. After two minutes, this is expectorated into a graduated collection vessel called sialometer. Two more two minute samples are collected, for a six minute total. The physical characteristic and the volume of saliva are recorded. Flow rate is expressed as mL/min.

6.12.2 Collection of stimulated whole saliva

Sreebny (1989), has discussed the following method for the collection of stimulated whole saliva. A standard piece of paraffin wax (1.5gms, mp 42 degrees C) is used. The wax is placed in the patients mouth and softened. The patient is asked to swallow any accumulated saliva, and is then instructed to continually chew the wax at a normal rate.

Three two minute samples are obtained. Flow rate and physical appearance are recorded. Where paraffin wax cannot be employed for example in edentulous patients, 2 per cent citric acid may be used as a stimulant. The citric acid solution is swabbed on the dorsum of the tongue every 15secs. The clinician should be aware that citric acid may interfere with chemical and microbiological tests on whole saliva.

6.12.3 The pros and cons of whole saliva

Whole saliva consists of secretions of the major and minor salivary glands mixed with food debris, microorganisms, sloughed cells, leukocytes and gingival fluid. It is readily obtained and is an excellent indicator of whole mouth dryness and the diseases of conditions associated with it (Sreebny 1989). Xerostomia is a subjective, not an objective, indicator of salivary gland hypofunction. Flow rates should be determined in all cases where salivary gland hypofunction is suspected. Normal baseline values should be obtained for all patients. These form the standard against which subsequent findings are judged (Edgar, O'Mullane 1990).

6.12.4 Collection of individual gland saliva

Special techniques exist for collection of saliva from major and minor salivary glands. Among these are 1) the "Lashley Cup", to collect secretions from the parotid gland; 2) the "Segregator" to collect secretions from the submandibular or sublingual glands; and 3) cannulation.

A simple method exists to collect the combined secretions from submandibular and sublingual glands (Fox 1985). Gauze may be placed over the openings of "Stensen's" ducts to block or absorb the secretions from the parotid glands. The submandibular/sublingual saliva is aspirated from the floor of the mouth with a small plastic aspirating device and transferred to a graduated cylinder. The collection period is approximately 5-10 minutes. The volume of fluid is measured and the flow rate is calculated.

The minor salivary glands are situated on the submucosa of the lips, the cheeks, the hard and soft palate and the ventral and dorsal surface of the tongue. They are predominantly mucous. It has been estimated that the minor glands contribute less than 10 per cent of the total volume of saliva, but account for about 70 per cent of the mucous secreted (Andros et al 1982, Shern et al 1989). Andros et al (1982), has discussed a method for the collection of minor gland secretions. Secretions from the minor salivary glands, may be readily collected on small absorbent paper strips and assayed with the Periotron GCF Meter, a device which measures small volumes of fluids. Mucosa is dried after two minute samples of saliva are obtained by touching the developing salivary droplet with the paper and reading the volume on the meter. The technique may be used to measure the rate of flow of resting or citric acid stimulated minor salivary gland saliva. It is a simple direct and rapid method.

6.13 CURRENT DIAGNOSTIC USES OF SALIVA

Diagnostic use of saliva has become very extensive particularly in relation to estimation of systemic levels of liquid soluble drugs and hormones. Thiocyanate levels have been used to validate self reported frequency of tobacco smoking and nitrate levels have been assayed to estimate dietary nitrate intakes (Ferguson D 1987). The estimation of steroid hormone concentrations in saliva is now generally recognised as a means of determining systemic steroid levels which offers many advantages over estimation in serum or urine samples. Immunoassay methods now permit measurements of very small concentrations of biologically active substances in saliva (Ferguson D 1987). It is, however, likely that unstimulated whole saliva may be the clinicians preferred sample. This may be less susceptible to variations (Ferguson D 1987).

6.13.1 Salivary flow rates: A diagnostic aid

Salivary flow rate can be a highly significant diagnostic tool in the dental treatment planning of high risk groups (Strahl et al 1990).

Those patients at risk for xerostomia should be tested with the Lashley cup to determine if acceptable flow rates are being maintained or compromised. Strahl et al (1990) conducted the study using 2 randomly selected homogenous groups of black geriatric patients. Group A consisted of 10 patients with dentition and group B consisted of 10 patients that were edentulous. Neither group were taking prescribed medication. Results of the study are shown in Table 37.

Table 37 Salivary flow rates among dentulous and edentulous elderly black patients in Baltimore.
Source: Strahl et al (1990).

Group A	Group B	t-test
Dentulous	Edentulous	
0.70 mL/min	0.45 mL/min	p<0.05

Results show that adequate flow rates for dentulous and edentulous are significantly different. Using an independent t-test there was a significant difference at the p<0.05 level. These are results of a small sample but appear to be worthy of diagnostic consideration and further study.

Both hard and soft tissue are ravished by xerostomia. In edentulous patients, caries rates soar and make the prognosis for fixed
prothetics poor. In edentulous patients there is an ineffective film thickness resulting in high abrasion (sore spots) and decreases in the retention of removable prothesis. Masticatory and gustatory stimulants may help alleviate these symptoms. However, the measurement of salivary flow is an invaluable diagnostic tool in determining the prognosis of alternative treatment plans. Because the elderly are more likely to be taking medication which result in xerostomia this diagnostic tool should be used on a routine basis which geriatric patients (Strahl et al 1990). Just as dentist use the periodontal probe to diagnose periodontal disease and the sphygmomanometer to diagnose high blood pressure, the Lashley cup can

easily be used to diagnose xerostomia. Armed with this knowledge the proper selection of treatment plans can be chosen (Strahl et al 1990).

6.14 STIMULATION OF NATURAL SALIVARY FLOW

Modern technology has allowed us to understand better the functions of saliva and now provides a rationale for developing: (1) diagnostic reagents for monitoring oral and systemic health status, (2) replacement therapies for individuals with salivary dysfunctions. Several areas of dental research are directed at augmenting or enhancing both the quality and quantity of saliva for individuals with dry mouth. An intrinsic approach has been explored utilising medication such as pilocarpine and bromhexine to stimulate the salivary glands to produce more saliva. An extrinsic approach proposes the use topically applied artificial saliva (Levine M et al 1987). Sreebny (1989) has suggested the stimulation of natural salivary flow may be enhanced by removing the cause of the salivary gland hypofunction. It may also be stimulated by mechanical, chemical, electronic, and pharmacological means.

6.14.1 Mechanical stimulation

Mechanical stimuli include the consumption of foods that require vigorous mastication like carrots or the use of materials that require chewing like sugarless gum or the holding of objects in the mouth like cherry or olive pits. For denture wearers where caries considerations can be ignored, the sucking of citric acid flavoured boiled sugar sweets such as acid drops may provide a pleasant and effective form of relief. A more practical approach to the stimulation is to advise chewing of a flavoured non cariogenic agent such as sorbitol chewing gum. Where xerostomia related caries is a problem the use of sorbitol chewing gum immediately after a meal or snack may prove beneficial as its use has been shown to rapidly restore plaque pH following sugar intake (Jensen 1986). While chewing has the advantage of helping to force the saliva into plaque stagnation sites around teeth as well as being a proven salivary stimulus, taste is potentially a greater stimulus and citric acid has been found to be the most effective agent (Watanube, Dawes 1988).

Low sticking gum is useful for patients with dry mouth who wear full dentures. Patients with dentures are less liable to use chewing gum but it is worth recommending. The action of chewing and the flavour of chewing gum promotes salivary flow and it appears that in dentate individuals, a secondary action of the chewing pushes the saliva through the interproximal areas and raises plaque pH (Ferguson M 1989).

6.14.2 Chemical stimulation

Citric acid substances are often used to chemically stimulate the salivary flow. Products containing lemon may be prescribed by the dentist. However, caution should be exercised since low pH products may demineralise the teeth. Sreebny (1989) has discussed the use of Pro-flow a substance which contains 3.5 per cent citric acid and is saturated with dicalcium phosphate which is now being tested. Pro-flow is supposed to stimulate flow but does not decalcify enamel.

Ettinger (1991), has quoted the following dosage of citric acid;

Rx : 5% citric acid in glycerine

Disp: 30mLs

Sig : 5-6 drops under the tongue 4 times daily.

6.14.3 Pharmacological agents

A number of drugs including pilocarpine bromhexine and anethole-trithione have been assessed as salivary stimulants. However none can be endorsed for routine long term therapy and their use at present is within the province of the specialist (Fox 1987). Proposed treatments to stimulate salivary gland function are listed in Table 38.

Table 38 **Proposed treatment to stimulate salivary gland function.**
Source: Fox (1987).

Bromhexine
 Trithio-paramethoxyphenylpropene/Anethole-trithione
 (Sulfarlem S25/Sialor)
 Pilocarpine Hydrochloride
 Betanecol Chloride
 Guaifenesin
 Neostigmine
 Potassium Iodide
 Nicotinic Acid
 Malic Acid
 Vitamin A

Bromhexine: It is a mucolytic agent indicated for use in chronic bronchitis, an obstructive pulmonary disease. It acts to increase the quantity of secretions while decreasing their viscosity. Fox (1987) has indicated that a number of clinical trials have examined the effects of sytemic administration of bromhexine on salivary and lacrimal function of patients with Sjogren's syndrome have failed to demonstrate an increase in saliva output.

Pilocarpine: It can be used as a pharmacological sialagogues. Pilocarpine 5mg t.i.d. has been tested by Sreebny 1989 and shown to be helpful in certain difficult cases. After a period of six months no changes were observed in patient heart rate, blood pressure or E.C.G. (Sreebny 1989).

Ettinger (1989) has quoted the following dosage for pilocarpine;

Rx : Ophthalmic pilocarpine 4%
 Disp: 40 mls
 Sig : 2 drops on the tongue and swallow 4 times daily

Maximum dose : 3 drops 4 times daily

Contraindication: cardiac arrhythmia, bronchial asthma

Some patients are sensitive to pilocarpine to doses as low as 1-2 mgs. Even such small doses can induce excessive perspiration nausea and loose bowels (Ferguson M 1989). Possible alternatives to pilocarpine are listed in **Table 39**.

Table 39 Parasympathomimetic agents.
Source: Ferguson M (1989).

PARASYMPATHOMIMETIC DRUGS

PILOCARPINE

BETHANELOL

CARBACHOL

DISTIGMINE

NEOSTIGMINE

PYRIDOSTIGMINE

6.14.4 Electronic stimulation

An electronic stimulator (Biosonics "SAL" Salivator system can be employed to stimulate the flow of saliva. The device electrically stimulates the oral and pharyngeal afferent nervous system, inducing and increase in salivation (Sreebny 1989). Steller et al (1988) have suggested that preliminary studies of the Biosonics "SAL" salivator system may be a useful adjunct to the techniques dentists use to treat dry mouth.

6.15 SALIVARY SUBSTITUTES OR ARTIFICIAL SALIVA

Water is a salivary substitute, but to produce more sophisticated salivary substitutes, the viscoelastic properties of saliva should be incorporated. The use of high molecular weight polyethylene oxide solution could be considered. There are some commercial products containing animal mucin and others using carboxymethylcellulose to make a viscous solution. These substitutes have a role to play in managing dry mouth but their utility is limited (Ferguson M 1989).

The significant difficulty is delivery. Aerosol containers are expensive. An alternative is carrying around spray bottles to be filled up regularly, but these can become contaminated with bacteria if not kept immaculately clean.

An ideal artificial saliva should be long lasting, provide lubrication, inhibit colonisation of microflora responsible for dental caries and gingivitis, and coat the oral soft tissues for protection against environmental insult and desiccation (Levine M et al 1987).

6.15.1 Composition and properties

Traditionally two approaches have been utilised for the treatment of dry mouth: "intrinsic" and "extrinsic". For example intrinsic agents have been used to maintain or augment hypofunctional glands and can include pilocarpine (Fox et al 1986). An obvious disadvantage of this approach is the potential side effects and the actual formulation and regimen of these compounds are still under experimental study (Fox 1985). Contents of extrinsic saliva substitutes is shown in Table 40.

Table 40 Contents of extrinsic saliva substitutes.
Source: Levine M et al (1987).

1. Carboxymethylcellulose.
2. Animal mucins.
3. Xylitol or Sorbitol.
4. Mineral salts.
5. Fluorides.
6. Preservatives.
 - (a) methyl p-hydroxybenzoate.
 - (b) KSCN + H₂O₂ → hypothiocyanate.

Carboxymethylcellulose is a common ingredient used to impart lubrication and viscosity. Sorbitol or xylitol are added to provide surface activity and act as a sweetner. However, the combination of carboxymethylcellulose and sorbitol results in a highly viscous mixture having a surface tension significantly higher than natural saliva. Animal mucins, usually derived from porcine gastric tissues and bovine submaxillary glands have been added with a concomitant decrease in carboxymethylcellulose content to reduce the viscosity and surface tension of artificial saliva. Levine M et al (1987), have suggested that the more physiological mucin based saliva substitutes facilitate the emulsion of food, aid in swallowing and allow the artificial saliva to distribute more evenly over the oral mucous

membranes. Salts have been added to artificial saliva to mimic the electrolyte content of natural saliva, while calcium phosphate and fluoride ions are included to provide remineralisation potential (Levine M et al 1987). The rehardening properties of artificial saliva appear to be dependent on an appropriate concentration of carboxymethylcellulose, mucin, and sorbitol (Vissink et al 1985). However, this compound has an unpleasant taste and may irritate oral mucous membranes when its concentration increases due to evaporation of water from the artificial saliva.

Levine M et al (1987) have reported the use of a modified lubometer to measure the lubricating properties of a salivary proline rich glycoprotein at enamel glass interface. They have reported preliminary data utilising this modified lubometer to compare the lubrication and viscosity of four commercially available artificial salivas with comparable properties of human submandibular/sublingual and parotid salivas. Three brands of commercially available carboxymethylcellulose based artificial salivas were tested: Moi-stir, Salivart and Xero-lube. All these substitutes contained at least 1 per cent carboxymethylcellulose w/v in an aqueous ionic solution meant to stimulate natural saliva. Orathana was used as a mucin based saliva substitute in these tests. Xero-lube displayed the highest viscosity followed by Moi-stir and Orathana. With the exception of Salivart all artificial salivas had viscosities greater than that of parotid or submandibular/sublingual saliva. The mucin based substitute, Orathana, exhibited relative lubricating values comparable with those of human saliva, while the saliva substitutes not containing mucin displayed very little function.

Vissink et al (1987), studied the efficacy of mucin-containing artificial saliva in eliminating symptoms of xerostomia. The efficacy of a mucin containing saliva substitute was assessed in terms of changes in oral functioning, the psychic and social implications of these changes. In this study 39 patients with xerostomia filled out

a questionnaire before application of the artificial saliva and after six weeks of usage. From the study it was concluded that the application of mucin containing saliva substitute produced the sensation of a dry mouth and improved oral functions such as chewing, swallowing, and speech. There was also less oral discomfort at night. The patients using mucin containing saliva felt less restricted in their social activity, especially in communication.

6.15.2 Rheology of saliva

Rheology is the science associated with the deformation of materials subjected to stresses or forces (Schwarz 1987). A fluid can be defined as a material that will flow indefinitely upon application of a stress of any magnitude. The qualification on the degree of the applied stress is to distinguish fluids from solids and materials with internal structure. Rheology of saliva affects the coating and lubrication of oral surfaces and the consistency of ingested foods (Schwarz 1987).

Saliva is a complex system of mucins, electrolytes, proteins, sugars, bacteria and enzymes (Shellis 1978). Schwarz (1987) did studies on the biochemistry of saliva. He identified components and determined their relative concentrations present in saliva. Constituents such as proteins and polysaccharides in aqueous media form viscoelastic fluids, perhaps having internal structure. Natural saliva is labile and homogenous. It is possible to have simultaneously, a liquid phase, a gaseous phase, a gel phase. Therefore, the rheological properties of whole saliva is highly variable. Schwarz (1987) in his study made rheological measurements on two synthetic saliva samples : Salivart and Xero-Lube. He concluded that a minimum volume of resting saliva must be maintained in the mouth to prevent tissue damage from abrasion and dehydration. The net amount of fluid depends on the difference between the influx and the efflux of saliva. Oral dryness means a reduced influx of fluid and the residual volume in the mouth reduces with time.

Schwarz (1987) defined V_c as the critical volume needed to coat surfaces with a film that is sufficiently protective. It is not practical to administer synthetic saliva at the normal rate of salivary secretion. The design of a competent synthetic saliva must consider periodic dosages, which puts additional constraints on the properties of artificial saliva. The dose cycle for a xerostomic patient receiving artificial saliva starts with delivery of the saliva. Under normal conditions the amount added to existing material in the mouth should not exceed 1.2 mL. If the volume does exceed that amount a swallow is initiated, leaving about 0.9mL for a normal male patient (Lagerlof, Dawes 1984). This remaining fluid (V) is further depleted by subsequent swallows and evaporations. When V is less than V_c , then another dose is applied which completes the cycle.

To reduce the frequency of application of synthetic saliva, it is necessary that one decrease the frequency of swallows, decrease leakage and evaporation and make it difficult to form a bolus. All these can be done by formulating a synthetic saliva to have high viscosity to be a weak gel. It is plausible that the purpose of the mucins in saliva is to form a weak gel or a visco elastic fluid in order to provide proper rheological, water-binding, and surface coating properties (Schwarz 1987).

Schwarz (1987) also suggests the use of weak gels. The proper choice of excess salt and concentration of polysaccharide results in a weak gel with a melting point slightly above body temperature (37 degrees centigrade). The weak gel belongs to a class of materials with internal structure. At rest, the material is viscoelastic, solid and adheres to surfaces. Mild shear disrupts the structure and the material flows like a fluid having a low viscosity.

6.15.3 Current status of artificial salivas

Clinically, artificial salivas have served as a replacement modality for individuals exhibiting hyposalivation. For sale as an "over the counter" item artificial saliva have traditionally been function oriented or formulated to replenish particular functions of saliva such as lubrication, viscosity, tissue hydration, surface tension and/or antimicrobial properties. Currently available products appear to be less than ideal. Since their effects are of limited duration and they may either have an unpleasant taste or irritate the mucosa (Levine M et al 1987).

Levine R (1989) reported formulations based on carboxymethylcellulose or animal mucin with a known cariogenic flavouring agent such as sorbitol, xylitol, saccharin or aspartame and a preservative. Fluoride, calcium and phosphate ions are desirable additions to this basic formula in cases of xerostomia related caries. In the United Kingdom artificial saliva may be provided on NHS prescription or a carboxymethylcellulose based formula given in the dental practitioners formulary. The composition of artificial salivas reproduced from the Dental Practitioners Formulary is presented in Table 41.

**Table 41 Artificial saliva, DPF. (Reproduced from the Dental Practitioners' Formulary, 1988-90).
Source: Levine R (1989).**

Sodium Chloride	100 mg
Hypromellose '4500'	1.3 g
Benzalkonium chloride solution	0.02 mL
Saccharin sodium	10 mg
Thymol	10 mg
Peppermint oil	0.02 mL
Spearmint oil	0.03 mL
Amaranth solution	0.1 mL
Water for preparations	to 100 mL

Saliva Orathana is based on pig mucin and may be unacceptable to some patients for religious reasons. It is provided in a spray bottle which is convenient to use and is now available in a fluoride form. It was proved to be a more effective form of relief than water

(Duxbury et al 1989). Glandosan is a carboxymethyl based artificial saliva and is also available in a spray. However, it has a low pH and does not contain fluoride.

In the USA, there are numerous artificial saliva products currently marketed for non-prescription use by patients. All products were developed to mimic chemical and physical characteristics of natural saliva. Carboxymethylcellulose is used to give artificial saliva a viscosity similar to natural saliva. All products contain calcium and phosphate ions but only three contain 2ppm fluoride (Xero-Lube, Orex, and Saliv-aid). There is no clinical evidence to indicate that these minerals are effective in the remineralisation of tooth surfaces (Ettinger 1991). The Council of Dental Therapeutics does not recognise remineralisation or reduced caries rate claims for these products. Most artificial salivas are sweetened with sorbitol. However, for patients who constantly use these products, in the absence of normal salivary flow, sorbitol can cause gastro-intestinal discomfort as it has a tendency to absorb water (Ettinger 1991). Commercially available common saliva substitutes are presented in Table 42.

Shannon and co-workers, 1977, have produced an artificial saliva known as Salube for use by patients after radiation to the head and neck. Salube contains fluoride and a number of other ionic components. This oral rinse or remineralisation media has been shown to be effective in maintaining the natural dentition after radiation, but is contraindicated in patients on a salt restricted diet because it contains a high concentration of sodium.

Table 42 **Treatment of dry mouth.**
Source: Ettinger (1991).

Common OTC Saliva Substitutes									
Product	Dosage Form	CMC	SOR	FL	KCL	CaCl ₂	NaCl	MgCl ₂	PO ₄
Moi-Stir (Kingswood lab) 336 Heather Dr. P.O.Box 744, Carmel, IN 46032.	Pump Spray 4 oz.	X	X		X	X	X	X	X
Orex (King's Speciality Co.) 305 W. Brackenridge P.O.Box 207 Ft. Wayne, IN 46801	Pump Spray 6 oz.	X	X	X		X		X	X
Salivart (Wesport Pharmaceuti- cals) P.O.Box 816 Westport, CT 06881.	Aerosol 50 mL	X	X		X	X	X	X	X
Xero-lube (Scherer Labs Inc.) 14335 Gillis Road, P.O. Drawer 400009 Dallas, TX 75240	Pump Spray 6 oz.	X	X	X	X	X	X	X	X
Saliv-Aid (Copley Pharmaceu- ticals) 398 West Second St. Boston, MA 02127.	Plastic 2 oz or 6 oz	X	X	X	X	X	X	X	X

6.15.4 Future directions for artificial saliva

The current knowledge of complex structural and functional characteristics of salivary secretion argues for new approaches in the development of artificial salivas (Levine M et al 1987). Consideration should be given to the design and formulation of saliva substitutes that are disease oriented as well as function oriented. Several applications for disease oriented saliva substitutes come to mind. Should artificial saliva be used selectively to augment the protective functions of saliva in individuals with normal salivation? For example should artificial salivas be designed to prevent or regulate plaque mediated diseases? In this instance formulation may be very different for an artificial saliva made to combat the deleterious effects of xerostomia. An artificial saliva may be designed to prevent or treat denture stomatitis. An artificial saliva may be designed for individuals with acquired immunodeficiencies who require artificial saliva tailored to their specific needs. In other words, should the artificial saliva have antiviral properties and also be targeted to soft tissues (Levine M et al 1987).

The design of saliva substitutes for disease requires a knowledge of the structure function relationships of individual salivary molecules. In this regard it is relevant to review briefly information on the composition of human saliva and emphasise area of future research. Levine M et al (1987) suggests that majority salivary molecules are acinar cell products which can be grouped into several families, these are listed in Table 43.

Table 43 Serous and mucous acinar cell products.
Source: Levine M et al (1987).

Family	Functional
1. Mucins	Heterotype complexing Microbial clearance/adherence Tissue coating
2. Proline rich proteins and glycoproteins	Microbial clearance/adherence Modulate calcium-phosphate equilibria Tissue coating
3. Histidine-rich peptides	Modulate calcium-phosphate equilibria Modulate growth of oral flora Salivary buffering
4. Cysteine-containing phosphoproteins	Heterotypic complexing Modulate calcium-phosphate equilibria Modulate growth of oral flora Tissue coating
5. Amylases	Digestion of complex carbohydrates Heterotypic complexing Tissue coating Modulate growth of oral flora

7. SALIVARY CLEARANCE AND ITS EFFECT ON ORAL HEALTH

The clearance pattern of a substance is very important, when trying to estimate the oral bio-availability of a specific substance. There are many factors involved, and clearance patterns for substances like sucrose, fluoride, chloride and chlorhexidine are quite dissimilar. Individual variations in clearance patterns can be both harmful and beneficial. Mechanisms of salivary clearance of carbohydrates from food, acids from plaque and therapeutic substances (for example fluoride) help to explain differences in oral health between different individuals and between different sites within a single mouth (Edgar, O'Mullane 1990).

A large number of substances of different chemical composition pass through the oral cavity every day. Some of these substances, such as sucrose or acids, are a threat to the health of the oral cavity with its unique and vulnerable tissues. Other substances, like fluoride and chlorhexidine, act as a defence, promoting oral health. Many substances will dissolve into saliva, from which they will then diffuse out into the oral tissues. The effect of incoming freshly secreted saliva, however, is to dilute the concentration of dissolved substances, a process that is described as salivary clearance (Edgar, O'Mullane 1990).

7.1 FACTORS THAT INFLUENCE FOOD CLEARANCE FROM THE MOUTH

A number of factors influence the rate at which foods are removed from the mouth by swallowing. These are physical properties such as adhesiveness, the resting salivary flow rate and the degree of salivary flow induced by flow in the mouth. Bibby et al (1986) in their study found that starchy foods remain in the mouth longer than sugary foods. A salivary clearance model by Dawes (1983), describes the oral cavity as an incomplete siphon. According to this model, after swallowing there is a minimum level of saliva remaining in the mouth, called residual volume of saliva. Saliva then flows into the mouth at the rate dependent on the stimulating effect of an ingested substance. The volume of saliva in the mouth increases until a

maximum volume is reached. This stimulates the subject to swallow, clearing some of the substances to be eliminated from the oral cavity. The remainder (dissolved in the residual volume of saliva) is then progressively diluted by the saliva again entering the mouth until the maximum volume is reached, and another swallow occurs. The Dawes model has been used to describe with considerable accuracy the clearance of substances such as sucrose. Bristse and Lagerlof (1987) have shown that intra-oral sugar clearance is quickest in the lower incisor region and slowest in the upper incisor region, with little difference in the clearance rates in the upper and lower molar regions. Lindfors and Lagerlof (1988) found that the greater the salivary concentration of sugars the greater the fall in the plaque pH, and that rapid clearance of cariogenic food was important in caries prevention.

7.2 FLUORIDE CLEARANCE

After a topical fluoride treatment the primary chemical reaction product is the formation of calcium fluoride on the enamel surface. Stookey (1987) has suggested that calcium fluoride formed on the enamel surface following topical fluoride application has two possible fates, a portion of the initial reaction products undergoes further reaction resulting in the formation of fluorohydroxy apatite, while the remainder is lost from the enamel surface to the dental plaque and saliva. Plaque fluoride levels can be elevated for six to eight hours following a rinse and can constitute a "reservoir" of fluoride (Edgar, O'Mullane 1990). Edgar and O'Mullane (1990) suggest that during the early phase of clearance, some of the fluoride will diffuse into the plaque from which it is later redistributed into the bulk saliva component. This will delay clearance of fluoride as will the formation of calcium fluoride deposits on the teeth which can occur at higher fluoride concentrations and alter the clearance pattern. Some fluoride may be swallowed, absorbed into the blood and partly recycled via the salivary glands.

7.3 CLEARANCE OF SUBSTANCES FROM LOCAL SITES

Saliva covers the oral surfaces in a thin film approximately 0.1mm thick. The rate of flow in different areas affects the salivary clearance from different regions in the mouth. Britse and Lagerlof (1987) compared sucrose clearance after a sucrose mouth rinse at four sites with that of the bulk saliva. A much slower clearance was found from between the upper centrals compared to between the lower centrals. When the clearance of sugar from the lower incisor site was measured in different individuals against the measured salivary flow rate, a correlation was seen between the salivary flow rate and individual clearance patterns.

7.4 CLEARANCE OF CARBOHYDRATES

Dental caries is caused by the demineralising effects of organic acids produced in the plaque by micro-organisms that ferment carbohydrates, most notably sucrose (Edgar, O'Mullane 1990). Clearance of carbohydrates from the mouth appears to be one of the most important functions of saliva with respect to prevention of dental caries. Dawes (1983) did a theoretical study of salivary sugar clearance, he identified the most important factors as being the unstimulated salivary flow rate and the volumes of saliva in the mouth before and after swallowing. The lower the values of the latter two parameters the faster is sugar cleared from the mouth.

The reduced salivary flow rate in most patients with dry mouth will reduce the rate of oral sugar clearance and presumably this contributes to the increased caries incidence in such patients (Dreizen et al 1977). Edgar, O'Mullane (1990) found at a slower clearance rate pH was depressed to a greater degree than at a faster rate. Sucrose concentration in the early phase of clearance was also significantly correlated to pH in the latter phase of the Stephan curve. Thus a slower clearance rate not only depresses pH but also retards its recovery to the resting value. The more rapid the flow the faster the carbohydrate is cleared. This is true whether the saliva is unstimulated or stimulated, for example by chewing gum. Clearance will be less enhanced if the gum contains sucrose and other

fermentable carbohydrates, but if it contains sugar alcohols such as xylitol or sorbitol, which are minimally metabolised by plaque bacteria, then the increased salivary flow will be very effective in carbohydrate clearance. Jensen and Wefel (1989) suggest that even sucrose sweetened gum if it is chewed until the sucrose leaches out can enhance clearance.

7.5 CLEARANCE OF CHLORHEXIDINE

Chlorhexidine is used in dentistry to control infection, especially in the form of rinse solutions or gels for plaque control, to prevent both dental caries and periodontitis (Edgar, O'Mullane 1990). A feature of chlorhexidine is its adherence to the oral surfaces. Approximately 30 per cent of the antiseptic is retained in the mouth following a one minute rinse of 0.2 per cent solution (Bonevoll 1974). The adherence to these oral surfaces gives a special clearance pattern and it can remain elevated for many hours.

7.6 THE EFFECT OF A WATER RINSE ON PLAQUE pH

Mouth rinsing after a sugar challenge does not markedly affect the sugar content of organic acids and of amino acids in plaque, whereas chewing paraffin wax has a dramatic effect. This may reflect not only the neutralisation and buffering by bicarbonate, but also the supply of nitrogenous compounds for base production (Edgar, O'Mullane 1990). The lack of effect of mouthrinsing may be because it is done too late. Two minutes after a sucrose challenge, the sucrose concentration in saliva is lower than that in plaque, so rinsing would not be expected to accelerate diffusion markedly, unless the sugar clearance is excessively slow as in the case of xerostomic patients. As far as the removal of acid is concerned it appears outward diffusion may not adequately explain plaque neutralisation as protons (H^+ ions) responsible for the low pH are fixed to bacterial proteins and other fixed buffers in plaque. This is why mobile salivary buffers like bicarbonate are so important, they are able to diffuse in, capture the protons from the fixed buffers and remove them (Edgar, O'Mullane 1990).

7.7 CHEWING GUM

Maiwald and Beu (1990) suggest that prevention of oral diseases must focus not only on completely eliminating the factors identified as the cause but rather on reducing them to a level with which our inherent defence mechanisms can cope. It would be beneficial to consider alternatives that increase resistance and defence performance such as the use of the fluorides or boosting the rinsing and buffering action of saliva through additional steps like chewing gum.

This section contains an array of studies concerning the action of chewing gum. Chewing gum as a means to stimulate salivary flow has been a topic of interest in many recent journal articles and seminars. It has evoked much interest. It appears that chewing gum may have a hydrodynamic pumping effect on plaque which might not be expected with other mechanical stimuli (for example sucking a stone), or with a combined mechanical and chemical stimuli (for example, sucking on a lozenge). Products with hydrodynamic effect might be superior to nonhydrodynamic mechanical and chemical stimuli. Levine R (1989) suggests that the condition with most patients with dry mouth is drug induced. These patients are already taking a large number of drugs, so a nonpharmacological salivary stimulant might be preferred.

Edgar (1990) suggested that stimulation of residual gland activity by the use of sugar-free sweets, especially chewing gum could have an immediate benefit in stimulating saliva and perhaps a longer term benefit in encouraging salivary gland function. Jenkins and Edgar (1989) did a study on gum chewing between meals by students. The study showed that gum chewing between meals resulted in raising unstimulated saliva flow rates especially among slow secretors (an effect still detectable several weeks after gum chewing had finished), which implies some structural effect on the glands.

Basically chewing gum can be divided into sugar-free and sugared gum. Some of the studies done on sugar-free and sugared gum are discussed in the next sections.

7.7.1 Xylitol chewing gum

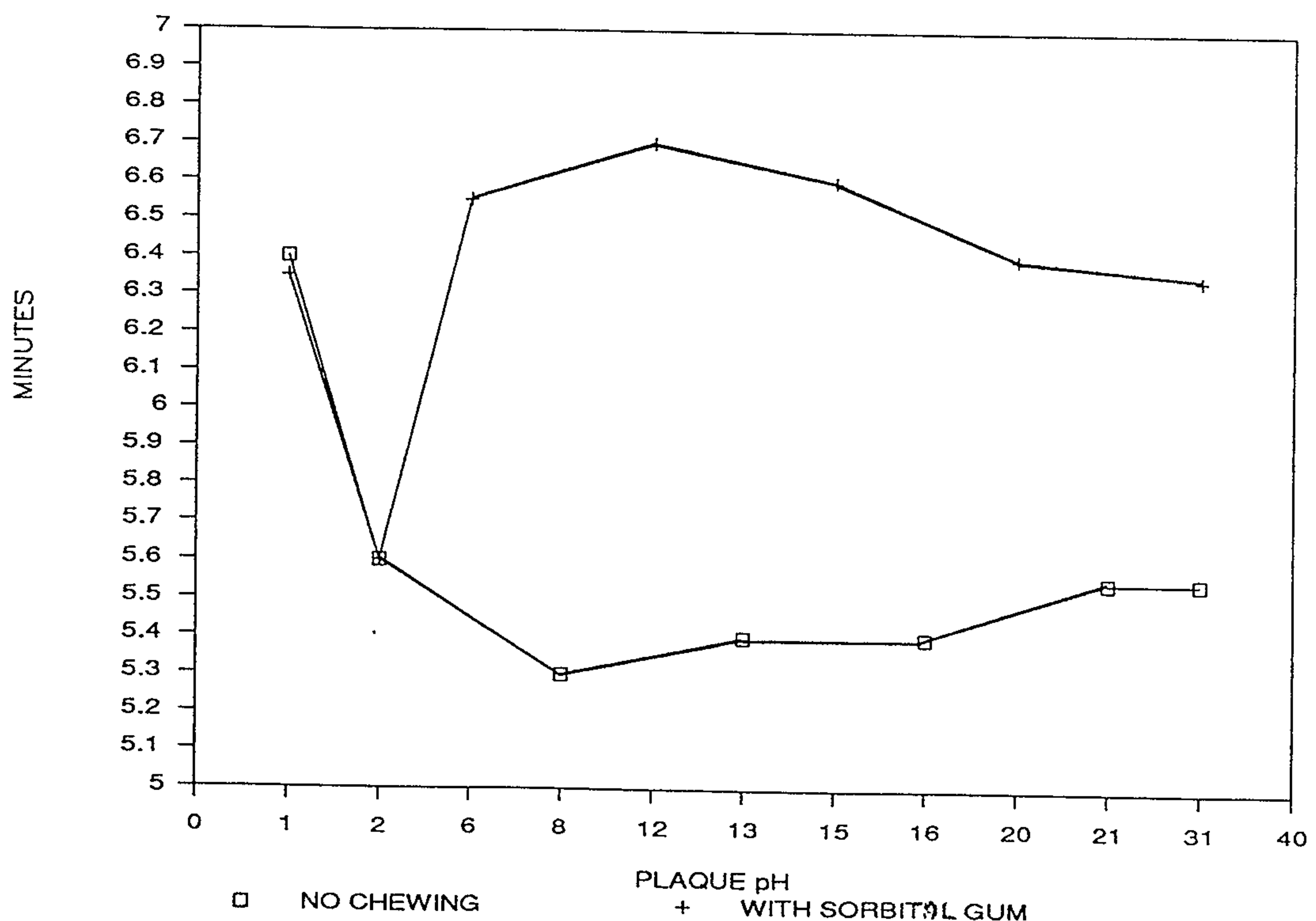
Xylitol is a naturally occurring sugar alcohol of the pentitol-type that is found in small amounts in most fruits and vegetables. Xylitol is the only sucrose substitute that rivals sucrose in degree of sweetness and cannot serve as a substrate for *Streptococcus mutans* and *Lactobacillus casei* (Loesche 1985).

Loesche (1985) commented on the Turku Xylitol Chewing Gum Study carried out by Scheinin et al (1975). The most important aspect of this research was the demonstration in clinical trials that xylitol was non-cariogenic when substituted for sucrose either in foods or in chewing gum. With respect to the effect of chewing gum to remineralisation, the results found xylitol chewing gum had an adequate effect on the remineralisation process but sucrose chewing gum was inadequate in this respect. This is because the xylitol and sucrose chewing gum between meals would effect the composition of the plaque flora in different ways, particularly in regard to the size of the niche occupied by *Streptococcus mutans*. This in turn would either amplify the cariogenic potential of plaque flora in the case of the sucrose chewers or mute the cariogenic potential in the case of the xylitol chewers. Thus the brief exposures of these sweeteners caused the plaque to diverge into two distinct microbial communities in regard to caries potential. The plaque from the sucrose subjects tended to have more *Streptococcus mutans* and *Lactobacilli* and exhibited high levels of extra and intracellular carbohydrates and lactic acid. The plaque from the xylitol subjects exhibited higher levels of enzymes involved in the removal of sugar residues from glycoproteins. This type of plaque metabolic activity would not lower the pH and the mineralising potential of saliva which operates at neutral pH, could come to dominate to the extent that remineralisation of incipient lesions could occur (Loesche 1985).

7.7.2 Sorbitol chewing gum

Markovic et al (1988) did a study on "Sorbitol Gum in Xerostomics". The purpose of this work was to study the effect of chewing sorbitol sweetened gum on salivary flow and plaque pH on xerostomic patients. Salivary flow was studied in 19 dry mouth subjects, while plaque pH response to a fermentable carbohydrate solution was measured in a subset of ten subjects. It was found that the sorbitol sweetened chewing gum significantly increased salivary flow rates. Largely because of this increased salivary flow, the degree and duration of tooth exposure to plaque acids (as measured by changes in plaque pH) was significantly reduced. The findings of this study suggested that chewing a sorbitol sweetened gum may be beneficial for individuals suffering from xerostomia, both from the point of view of increasing comfort and reducing their risk of dental caries. **Figure 11** presents the plaque pH response to a 10 per cent sucrose rinse in 10 subjects with xerostomia.

Figure 11 Plaque pH response.
Source: Markovic et al (1988).



Plaque pH response to a 10 per cent sucrose rinse in 10 subjects with xerostomia. Sucrose solution rinsed with from time 0 to 1 minute for both determinations. Gum chewed from time 2 to 12 minutes during gum chewing.

Leach et al 1989 did a study on the "Remineralisation of Artificial Caries-like Lesions in Human Enamel In Situ by Chewing Sorbitol Gum". This study showed that chewing sugar-free gum for twenty minutes, five times a day after meals and snacks, increased the remineralisation potential of the mouth. The results also suggested that chewing sugar-free gum is not only non cariogenic but may actually be therapeutic in repairing the early stages of carious attack in subjects using a fluoride toothpaste.

Park et al (1989) did a study on the "Impact of Chewing Gum on the Acidogenicity of Meals". The study evaluated the effect of chewing sorbitol gum on plaque pH following a meal. The results indicated that chewing sugar-free gum after a meal significantly raised the lowest pH. It also significantly reduced pH falling below 5.5 and resulted in a reduction in acidogenicity.

7.7.3 Urea chewing gum

Bjornstrom et al (1984) did studies on the effects of sugar-free chewing gum. They did two salivary secretion studies in volunteers who chewed gum in a formulation similar to urea chewing gum (referred to as V6). These studies clearly showed that saliva secretion was increased by chewing this type of gum. By following the recommended chewing time of ten minutes saliva secretion was increased approximately four fold. Results from their studies showed that the demineralisation period is reduced by the chewing of sugar-free chewing gum, and is significantly reduced even further by chewing of sugar-free chewing gum containing 20mg urea.

Bjornstrom et al (1984) did a study to evaluate five saliva stimulants and three saliva substitutes in patients suffering from xerostomia. They were listed as:

Saliva Stimulants:

- 1.Mucidan
- 2.Nicotinamide
- 3.V6
- 4.Salivin
- 5.Ascoxal-T

Saliva Substitutes:

- 6.Saliment
- 7.Salisynt
- 8.Mouth Rinse Solution

Each patient was expected to use the eight different preparations for 14 days in a randomised order. After the 14 day period, the patients were interviewed by dentists using a standard questionnaire. All the preparations tested were considered to relieve the symptoms of dry mouth. However, V6 chewing gum and Salivin were ranked as the best products by the patients.

7.7.4 Cinnamon flavoured chewing gum

Allen and Blozis (1988) did a study on "Oral Mucosa Reactions to Cinnamon Flavoured Chewing Gum". In this study a group of patients had oral mucosal lesions as a consequence of using cinnamon flavoured products primarily chewing gum. The lesions appeared as erythematous areas that had an associated keratosis or ulceration. They usually became apparent as a burning sensation. The severity was variable ranging from a focal "fixed drug" like reaction to a relatively diffuse mucositis. Symptoms and signs usually resolved in a week when patients stopped using the cinnamon flavoured gum. The clinical features of the problem when corroborated with a history provide significant information to make a diagnosis. The histomorphological changes were not diagnostic of themselves but were sufficiently characteristic to alert the pathologist to this possibility.

7.7.5 Sugared chewing gum

Glass (1981) did a study on the effect on dental caries incidence after frequent ingestion of small amounts of sugar and stannous EDTA in chewing gum. The results of this study showed that sucrose containing gum increased caries incidence. Similar conclusions were shown in a study done by Finn and Jamison (1967) on the effect of a dicalcium phosphate chewing gum on caries incidence in children. Unfortunately both studies were conducted without control of the time at which the gum was chewed and the period of chewing. Perhaps this may explain the increased incidence in caries.

Results from the study done by Thompson et al (1986) on the interproximal plaque pH responses to aspartame gum and gelatin dessert showed that when sucrose containing gum was chewed for one minute and then removed the acid challenge is equivalent to a 60 second rinse with 10 per cent sucrose solution.

Edgar and Bateman (1990) did a study on "Remineralisation of Artificial Human Enamel Lesions by Chewing Sucrose Gum". Results showed that when sucrose gum is chewed for twenty minutes after meals and snacks, sucrose gum promotes remineralisation of artificial lesions in situ.

7.7.6 Comparison of sugared and sugar-free chewing gum

Shannon and Frome (1973) did a study on "Enhancement of Salivary Flow and Buffering Capacity". They studied whole saliva flow rate, pH and buffer capacity, under conditions of no exogenous stimulation, in subjects chewing either sugar or sugarless chewing gum. They found chewing either type of gum brought about highly significant increases in rate of flow, in pH and in buffer capacity values. These changes suggested highly beneficial effects in preventing demineralisation of the teeth by acids. While comparing sugared and sugarless gums they found that non fermentable carbohydrates such as sorbitol and mannitol could be substituted for sucrose in a gum formulation without loss of the gums ability to elicit a copious flow of saliva. Flow rate induced by the sugarless formulation was significantly higher than that elicited by the same size bolus of sugared gum. They suggested it was possible to consider the potentialities of sugarless gum in preventive dentistry programs both as an inducer of salivary flow and as a vehicle for topical application and systemic administration of therapeutic agents.

Jensen and Wefel (1989) did a study on "Human Plaque pH Responses to Meals and the Effects of Chewing Gum". This study illustrated that chewing a piece of gum (both sugar-free and sugar containing gum) for twenty minutes following the consumption of meals restores plaque pH to resting levels that are considered "safe for teeth". The study indicated that the meals can be very acidogenic and in addition to normal dental procedures, chewing for twenty minutes after a meal should be considered as a strategy to reduce the cariogenic challenge to teeth.

Maiwald and Beu (1990) did a study on "The Caries Preventative Action

of Sugar Containing and Sugarfree Chewing Gum". They found there was an insignificant difference between sugar and sugar-free gum when considering the benefits of stimulated saliva and rapid rise in pH.

7.7.7 Discussion

A number of studies concerning the action of chewing gum have been discussed. Studies on sugar-free gums show results that sorbitol chewing gum significantly increases salivary flow (Markovic et al 1988), also causes a remineralisation of human enamel lesions (Leach et al 1989) and significantly raises lowered pH when chewed after meals (Park et al 1989).

Studies done on the urea base for chewing gum by Bjornstrom et al (1984) show that the demineralisation period is reduced by chewing sugar-free chewing gum and is significantly reduced even further by chewing sugar-free gum containing 20mg urea.

Clinical field trials with sucrose containing gum have pointed to increased caries incidence levels with this type of chewing gum (Finn, Jamison 1967, Glass 1981). These clinical studies were conducted without control of the time at which the gum was chewed and the period of chewing. This may be the explanation for the increased caries incidence reported in clinical trials with sucrose containing gum. Perhaps the results may have differed if the sucrose containing gum had been provided after meals or fermentable carbohydrates containing snacks and was required to have chew times of twenty minutes or more (Edgar, Bateman 1990).

Shorter chewing times may not be as effective at returning the plaque pH to safe levels. It is also quite probable that sucrose containing gum chewed between meals for short times may contribute substantially to the overall cariogenic challenge. When sucrose containing gum is chewed for one minute and then removed the acid challenge is equivalent to a sixty second rinse with 10 per cent sucrose solution (Thompson et al 1986).

Jensen and Wefel (1989) in their study illustrated that prolonged interproximal plaque pH drops occur following the consumption of a variety of normal meals which contain fermentable carbohydrates. The plaque acid attack following meal consumption can be rapidly reversed during a twenty minute gum chewing period following consumption of the meal.

In conclusion the interproximal plaque pH neutralisation can be observed regardless of whether sugar or sugar-free gum is chewed for twenty minutes after meals (Jensen, Wefel 1989). Bjorstrom et al (1984) in their study on sugar-free gum concluded that sugar-free chewing gum did relieve the symptoms of dry mouth. Stimulation of residual gland activity by the use of sugar-free chewing gum could have an immediate benefit in stimulating saliva and perhaps a longer term benefit in encouraging gland function (Edgar 1990).

Although the tooth cleaning action of chewing gum is no substitute for mechanical plaque removal (Maiwald, Beu 1990) any procedure that reduces the acidic challenge to the dentition is beneficial in a preventive program. Therefore in addition to the optimal use of fluorides, meticulous oral hygiene and the reduction of between meals snacks, the addition of chewing gum for twenty minutes following the consumption of meals should be considered for individuals at risk for dental caries. This simple process is easy for most individuals to comply with and takes advantage of normal host protective mechanisms to reduce the acidogenic challenge at stagnation areas such as interproximal sites. Chewing gum for twenty minutes after meals is one additional step that could be taken to help balance the caries equilibrium process in the direction of less demineralisation and more remineralisation in order to help reduce the risk of caries formation or progression.

8. CONCLUSIONS

Saliva enables people to enjoy some of life's more serene pleasures, the delicate sensation of taste, the joy of eating and the exquisite sound of the human voice. Life goes on in the absence of saliva but the quality of life diminishes. The importance of saliva composition lies in the ways saliva and its elements support oral functions. The mouth has developed to facilitate communication and alimentation. These functions may be compromised in the absence of normal salivation.

Patients with diminished secretions have difficulty speaking, chewing, forming a food bolus and swallowing. Additionally there is a rapid and substantial increase in caries and mucosal infection. Taste and soft tissue complaints are also more frequent. Saliva provides a constant supply of ions to the enamel surface. It also contains buffers which resist pH changes. Saliva dissolves and disperses food particles and serves as a clearing agent for soluble food stuffs. Saliva can be looked upon as "the blood-stream of the teeth"

Dental caries has been described as a socio-medical problem because the disease attacks almost a hundred per cent of population. The pattern of caries has undergone changes with time. In industrialised countries prevalence of caries in children and adolescences is showing a definite downward trend. However, because of increased access to dental care more teeth are retained in the older age group and in these more cases of root caries are being recorded.

Plaque is a normal part of the oral microflora and bacteria maintain a commensal relationship with the host. This relationship makes periodontal disease difficult to control. Normal flora will always re-establish itself. However, several authors have suggested that a reservoir of calcium phosphate and/or fluoride in dental plaque may be beneficial by maintaining the saturation with respect to enamel mineral, especially when plaque pH decreases.

The problem of Xerostomia is increasing as the proportion of the population of the elderly increases. Xerostomia is not a result of old age itself but is usually iatrogenic and potentially preventable.

It is important to find out if the patient is suffering from a deficiency of gland tissue or an under functioning of the salivary glands. If it is the former, then saliva substitutes are needed.

Stimulation of residual gland activity by the use of sugar-free sweets, especially chewing gum, can have an immediate benefit in stimulating saliva perhaps a longer term benefit in encouraging salivary gland function. Unstimulated flow rates vary widely between subjects and those with low rates do not always have symptoms of dry mouth. The main stimulus to salivation is the sensation of taste (particularly acid). Stimulated saliva is better able, because of changes in its composition, to prevent demineralisation and to favour remineralisation than is unstimulated saliva. Water is a salivary substitute, but to produce more sophisticated salivary substitutes, the viscoelastic properties of saliva should be incorporated. The use of high molecular weight polyethylene oxide solution could be considered. There are some commercial products containing animal mucin and others using carboxymethylcellulose to make a viscous solution. These substitutes have a role to play in managing dry mouth but their usefulness is limited.

The clearance pattern of a substance is very important when trying to estimate the oral bio-availability of a specific substance. There are many factors involved, and clearance patterns for substances like sucrose, fluoride, chloride and chlorhexidine are quite dissimilar. Variation in clearance patterns can be either harmful or beneficial.

Studies concerning the action of chewing gum have been discussed. Studies on sugar-free gums show results that sorbitol chewing gum significantly increases salivary flow (Markovic et al 1988), also causes a remineralisation of human enamel lesions (Leach et al 1989) and significantly raises lowered pH when chewed after meals (Park et al 1989).

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