THE MUCOSA OF THE HARD PALATE

UNDER THE DENTURE BASE

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A treatise submitted in partial requirement for

the degree of Master of Dental Surgery.

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1978
ACKNOWLEDGEMENTS

I wish to record my sincere thanks to

.....Eric M. Bevan B.D.S. M.D.S. FRACDS. FICD. for his support, guidance
and valuable advice during the preparation of this treatise.

.....Members of the Department of Prosthetic Dentistry, Faculty of
Dentistry for their encouragement and tolerance.

.....The Librarian and staff of the Fairfax Reading Dental Library,
University of Sydney for their friendly and helpful assistance.

.....The National Library of Australia for its assistance in the initial
search for references.

.....Miss Ann Broughton for her immaculate preparation of this treatise

and

The research workers and their assistants for carrying out
investigations in the field of this topic.

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PREFACE

The mucosa of the hard palate is an extremely abused tissue in a normal dentate individual. It has to withstand extremes in temperature and trauma (masticatory function, chemical irritations, etc.). However, when a "foreign material" denture base is placed over this tissue, it is abused even further, by the new environment, in which it finds itself enveloped.

How often does the prosthodontist blame the base for the inflammatory deterioration of the tissue under it, without considering that he may have to accept the major portion of the blame for having placed the base on unhealthy tissue. Many investigations have been made of the tissue after the placement of the base, without examining it prior to insertion.

It is of paramount importance that strict and thorough examination be made of the tissues before they are used as the seat for the denture base.

In the first part of this treatise, a brief summary of the anatomy, microanatomy and function of the mucous membrane of the hard palate is made. Interpretation of form compared to function is emphasised together with an outline of the process of keratinization. The particular problem of aging of the tissue is also discussed with respect to its tolerance to sustain trauma by the denture base.

The effects that may occur and that have been seen to occur with the placement of the denture base on the mucous membranes, are examined in the second section. The controversy of whether the epithelium becomes hyperkeratotic (the surface cells contain keratin with no nuclei
present) or parakeratotic (keratin, present as well as retained pyknotic nuclei in the surface cells) is analysed as well as the possible allergic reactions that some researchers believe, is caused by the chemical components of the denture base material.

The ensuing irritation, that may occur to the tissues of the hard palate, after the placement of the denture base, has been classified by many writers in numerous ways. For the purpose of completeness, it is discussed in this treatise in three separate chapters: denture sore mouth (D.S.M.); inflammatory papillary hyperplasia; and traumatic reactions. Although, there may be a great deal of overlap as far as the aetiology and clinical appearance, it allows the author to summarize and compare all the research that has been carried out in these areas.

As well as the denture base being a major aetiological factor of denture stomatitis, supporting "prosthetic materials and products" such as "liners", "cleansers" and "adhesives" have been found to have both beneficial and deleterious effects on the tissue. Denture retention, taste perception and the effect on the mucosa when the patient suffers from metabolic disorders are included for completeness of this topic.
PART I

THE ANATOMY AND MICROANATOMY OF THE MUCOSA

OF THE HARD PALATE
CHAPTER 1

ANATOMY

The palate, 47, 81, 162 while forming the roof of the mouth, separates the mouth from the nasal part of the pharynx and the nasal cavities. It extends in a posterior direction into the cavity of the pharynx. The palate consists of two parts, the hard and soft palates, which are both covered by a mucous membrane.

The hard palate is the anterior part and is a horizontal shelf of bone separating the oral and nasal cavities. It is formed mainly by the palatal processes of the two maxillae and behind these, by the horizontal plates of the palatine bones which meet in the median plane.

Anteriorly and laterally, the hard palate is continuous with the maxillary alveolar arch. There is a median raphé, 48 extending along the midline of the palate, ending in front in the incisive papillae with transverse ridges fanning out from the anterior part of this raphé. There is a pronounced transverse curvature in the central incisor tooth area; small pits corresponding to the inferior openings of the incisive canals can be observed.

Through these openings pass the naso-palatine vessels and nerves as they leave the nasal cavity and travel along the roof of the mouth, to provide the nerve and blood supply to the anterior two-thirds of the palate. The irregularly shaped ridges of connective tissue, covered with a mucous membrane and found in the anterior third of the hard palate, are referred to as "rugae." 153 Many believe these
rugae are important in speech, while others are of the opinion that they are vestigial.
CHAPTER 2

PRENATAL FORMATION AND DEVELOPMENT

On either side of the developing tongue of the human foetus, there are downward hanging palatal folds.46 With growth in size of the mouth cavity, these folds fuse and elevate. This occurs in the foetus, between the eighth and eleventh weeks. Later the united palatal folds are invaded by bone in front, which forms the hard palate and by muscle (soft palate) behind. The bone extends from the premaxillary, maxillary and palatine centres of ossification to form the hard palate.146

During the first few years after birth, there is a certain amount of growth at the sutures between the maxilla and facial bones including the palatine and lacrimal bones and also between these and the elements of the anterior cranial segment. The maxilla is thrust downward and forward by the growing eyeballs and nasal septum, and this separation of the facial bones permits growth to take place at the sutures. The chief method of facial growth during the second decade is by surface deposition of new bone on the facial surface of the maxilla and their alveolar processes and upon the under surface of the hard palate. This deposition of bone is associated with resorption of the interior of the maxillae and by increasing the sizes of the sinuses of the upper surface of the hard palate, thereby increasing the depth of the nasal cavities. The amount of alveolar growth is related to the downward and forward growth of the mandible relative to the growth of the upper face. The space between the jaws is filled by alveolar bone so that the teeth are maintained in occlusion.
The growth of the hard palate increases by one third after the first year with it becoming twice as large at seven years and by adulthood as much as four times its size at birth. During the first years, growth probably takes place at the palatal sutures, but after, growth is associated with deposition of bone on the undersurface of the palate and the downward and outward growth of the alveolar processes.84
CHAPTER 3

MICROSTRUCTURE OF THE MUCOSA OF THE HARD PALATE

The mucous membrane of the hard palate is firmly fixed to the underlying periosteum and is therefore immobile.\textsuperscript{86} It comprises of a masticatory mucosa, a dense layer of connective tissue, called the lamina propria and a submucosa which differs in various parts of the hard palate.

Van Scotter and Boucher (1965)\textsuperscript{214} stated that the cells comprising the mucosa, by their form and arrangement provide a mechanism of defence. It is by their capacity of reproduction and adaptability that cells maintain their individuality.\textsuperscript{147,152}

3.1 Epithelium
3.1.1. Structure\textsuperscript{12,55,80,150,160,197}

The epithelium is stratified squamous in type. Unlike that of skin, it does not undergo complete cornification. It can be observed as being divided into definite layers.

(i) Basal layer
(ii) Prickle Cell layer
(iii) Granular layer
(iv) Keratinous layer.

The epithelium accounts for only a small fraction of the total mucosa but is an important factor in the protection of it. It forms a homogeneous layer to stop chemical and bacterial agents
entering into the deeper layers of the mucosa.

(i) The basal layer is made up of a single layer of high cuboidal cells that are held to the basement membrane by protoplasmic processes and mediated by hemi-desmosomes. Although these cells are large, the average cell size is small and they have an angular shape.

In electron micrographs of the basal cells, a prominent nucleus with one or more nucleoli, numerous mitochondria, a Golgi complex, lysosomes, a few endoplasmic reticulum and abundant free ribosomes are seen. There are fine protein strands, some 6 mm in diameter, which may appear singly as tonofilaments or be arranged together in bundles to form the tonofilibrils visible. The tonofilaments represent a synthetic product that is retained within the cell as structural protein.

Cohesion is maintained by the presence of cement substance in the intercellular spaces, presumably secreted by the cells themselves. There may be some desmosomes present representing a mechanical link so that the forces applied to an epithelial surface will be transmitted via the cytoplasmic tonofilaments inserted in the attachment plaques. The tight junction is another intercellular attachment recognized morphologically by the close apposition of adjacent plasma membranes, so that apparently no intercellular space remains.

(ii) Superficial to the basal layer is the prickle cell layer made up of layer cells that are irregularly polyhedral. As the cells migrate into the prickle cell layer, there is a concentration of tonofilaments and ribosomes. Between these cells, there are intercellular spaces and in these spaces, there are intercellular bridges. These bridges are, in fact, tonofibrils that pass from adjacent cells and come into contact by means of the specialized structures, desmosomes.
Therefore, there is no continuity between the cells, as the fibrils do not pass through the "bridges" without interruption.

The cells towards the surface of this layer become flattened and widen as they proceed to the other layers. Mitochondria are seen in the cytoplasm near the nucleus, along with numerous bundles of tonofilaments. In the superficial cells of the prickle cell layer, a new organelle, "Odland body" or "membrane coating granule" was identified. As the cells enter the next layer, these granules are arranged along the superficial plasma membrane. These granules are circular or elongated and invariably contain a series of parallel lamellae that give a striated appearance. They are often classified as lysosomes because of their acid phosphatase content.

(iii) The cells move from the prickle cell layer into the granular layer. The cells of this layer contain irregular shaped keratohyalin granules with pyknotic nuclei and closely associated ribosomes. These cells are usually flattened and are arranged in three to five layers. With the gradual increase in their volume and number, the nucleus disintegrates and becomes pale. At the same time, the intercellular spaces become narrow and the processes from the cells become shorter and less distinct. The organelles present in the cells of the lower layers are present in the deeper cells of this layer, with the cytoplasm predominantly occupied by tonofilaments. This layer probably represents the first stage in the formation of the highly resistant membrane enclosing the keratinized squamae.

Dramatic changes take place in the superficial layers of this granular layer; virtually all organelles including the nuclei and keratohyalin granules disappear and the adjacent cells of the keratinized layer are filled entirely with closely packed fibrils.
(iv) The **keratinized layer** consists of flattened cells filled with fibrils showing the typical keratin pattern, with all other cytoplasmic organelles having disappeared. The cell margin forms villous processes which interdigitate with adjacent cells. The plasma membrane, while showing the intracellular thickening laid down in the granular layer, also develops an additional external thickening described by Frithiof (1970). Desmosomes are sometimes visible, but have lost their connection with the tonofilaments. The superficial cells are always shed and are found in the saliva. This layer is one fifth hundredth of the total mucosa.

3.1.2. Biochemical Features of the Epithelium

Anaerobic glycolysis plays an important role in providing energy in the epithelial tissue. Since there are no capillaries in stratified squamous epithelium, only cells at or close to the basement membrane, have a direct access to the blood supply. The more superficially placed cells, therefore have to depend on the raw materials and nutrients that have to pass through or between the deeper cells. This results in a progressive decrease in oxygen tension.

Glycolysis takes place in the supernatant fraction of the cytoplasm and is not associated with an organelle, while aerobic energy conversion takes place in the mitochondria. The mitochondria are concentrated in the basal cells of the epithelium, with their numbers decreasing towards the surface. Presumably glycolysis makes up for the deficit in the energy supply resulting from the decreased concentration of mitochondria.

There is a large amount of structural protein that accumulates in the differentiating cells of the epithelium. These proteins are represented almost entirely by filaments, which are seen in all cell
layers. In keratinizing epithelium, they are most concentrated in the stratum corneum where they may be found embedded in a matrix. The matrix may be related to the keratohyaline granules of the granular layer.

Keratinized tissues are marked by their insolubility in water, buffers, dilute acids and alkalis and organic solvents. Cell membranes of the keratinized layer are resistant to digestion by a variety of enzymes to which tonofilaments, keratohyalin granules and desmosomes are susceptible. This resistance of intracellular structural proteins and cell membranes is important in enabling keratinized epithelia to act as protective surfaces.

3.1.3. Keratinization

Weinmann (1940) stated that the highest degree of keratinization of the oral epithelium studied was seen in smears from the mucous membrane of the hard palate. The epithelium of the hard palate is orthokeratinized. Most theories of keratin formation are based on the sequence of events seen in histological and electron microscope preparations.

The superficial acidophilic layer of keratinized epithelium consists largely of insoluble fibrous protein, with a high proportion of the sulphur containing amino acid, cystine. This protein is basically termed keratin. Electron micrographs show that keratin consists of aggregates of fine fibrils. These filaments are probably bound together by the same attractive forces which operate between all polypeptide chains. This system of filaments within a matrix, is contained within the original cell membrane which has become thickened.

The characteristic intercellular feature of the glandular layer of the epithelium is the presence of the keratohyaline granules,
which show considerable morphological differences between various keratinizing epithelia. In the hard palate, granules of irregular size and shape that show intimate association with tonofilaments, are found. Ribosomes usually surround the tonofilaments. There is no clear evidence as to the origin of the keratohyalin granules. Biochemical analyses suggest that they contain protein and labelled amino acids. It thus seems likely that they are synthesized by ribosomes. All organelles, including the nuclei and keratohyalin granules disappear and the adjacent cells of the keratinized layer are filled entirely with closely packed fibrils. As described previously, the superficial layer of the epithelium consists of flattened cells with fibrils showing typical keratin patterns, with all the other cytoplasmic organelles having disappeared.

The epithelium is composed of a constantly renewing cell population, so that under normal circumstances the number of new cells produced, is just sufficient to match those lost from the surface due to normal wear and tear. There is considerable difference of opinion as to what really constitutes the germinative layers of the epithelium of the hard palate. Recently the problem has been re-examined by Løe, Karring and Hara (1972), using serial section and reconstruction techniques. The observations were that almost all dividing cells do in fact lie on the basement membrane and that all are within three cell layers of it. As the surface cells are lost by desquamation, there is a constant replacement by divisions in the basal layer so that the epithelium maintains a constant thickness.

3.1.4. Permeability of the Epithelium.

There are at least three processes, by which substances have been shown to cross various epithelial membranes. They are:

(i) Carrier-Facilitated Transport
It is believed that the passage of many large, strongly hydrogen-bonded molecules is accomplished by the combination of the molecules with a component of the membrane. After diffusing across the membrane, the combination is dissolved.

(ii) Pinocytosis and Phagocytosis

These processes are not important in transport across the oral mucosa. They are mostly observed in white blood cells.

(iii) Diffusion

This process is achieved by a concentration gradient with molecules moving from the higher to the lower concentration. Most molecules are transferred across the oral mucosa in this manner.

In order to pass across a tissue by simple diffusion, a substance must be sufficiently lipid soluble to dissolve in the cell membranes or be of sufficiently small size to pass through the membrane "pores". The rate of diffusion of weak acids and bases depends upon their degree of dissociation. Factors such as molecular weight, mass-sectional area of the mucosa, the pH of the solution and the equilibrium distribution ratio between the membrane and the aqueous solution are important to the diffusion rate.

3.2 The Basement Membrane

The term, basement membrane, has been used at a histological level, to describe the junction between the epithelium and connective tissue. It appears as a continuous but relatively structureless layer, 1-2 mm thick. There are two discernable layers of the basement membrane caused by differing electron opacities, the light layer, or lamina lucida, and the lower layer, the lamina densa, or dense layer. Associated with the
lamina densa are striated fibrils called anchoring fibrils which form loops through which run the collagen fibrils of the connective tissue. Therefore, it may be said, that this is not a true membrane but a complex of fibrils and matrix.

It is a product of the epithelial cells, although the anchoring fibrils are probably products of the connective tissue. It is composed of protein with small amounts of carbohydrates and glucosamine.

While the majority of metabolites entering or leaving the epithelium must cross the basement membrane, it is evident that at a molecular level, the basal lamina can exert a filtering effect so that only particles of a certain size can pass through. It is also suggested that a similar mechanism operates in the opposite direction. As well, the basement membrane functions for the attachment of the epithelium to the connective tissue. Two factors appear to be largely responsible for this union, one is the adhesion between the epithelial cells and the lamina densa and the other is the attachment of the basal lamina to the adjacent connective tissue. The material constituting the basal lamina has many similarities to that of intercellular cement substances found between the epithelial cells, and so the same sort of adhesion may exist between the basal cells and the basal lamina as between epithelial cells. There may be filaments running between the basal lamina and the hemi-desmosomes providing direct physical attachment.

3.3 Fibrous Connective Tissue

This is represented by:

(i) a lamina propria, and

(ii) a submucosa.
3.3.1. The lamina propria

The lamina propria is a layer of dense connective tissue which is thicker in the anterior than in the posterior part of the palate. The basal cell layer of the epithelium is firmly attached to this by means of the basement membrane. The connective tissue fibres run parallel to the epithelial surface. Its papillae which indent the epithelium, carry blood vessels and nerves. Some nerve fibres may pass into the epithelium. The papillae vary considerably in length and width in different areas. The arrangement of the papillae increases the area of contact between the lamina propria and the epithelium and facilitates the exchange of material between blood vessels and epithelium.

The lamina propria is divided by these papillae into the outer papillary and inner reticular layers. In the lateral areas of the hard palate, the lamina propria is fixed to the periosteum by bands of dense fibrous connective tissue that are at right angles to the surface and divide the submucous layer into irregularly shaped spaces. The distance between the lamina propria and periosteum is smaller in the anterior than in the posterior parts. In the posterior section of the oral cavity, the lamina propria contains many lymphocytes, which are often found migrating into and through the epithelium.

3.3.2. The Submucosa

The submucosa consists of connective tissue of varying thickness and density. It is most dense in the anterior portion as far as the first molar tooth region and then it appears to thin out and become more elastic. It attaches the mucous membrane to the underlying structures. The firmness of this attachment depends upon the character of the submucosa. Glands, blood vessels, nerves and adipose tissue are
present in this layer.

There is no submucosal layer along the midline of the palate. In spite of the presence of a well defined submucosal layer in the wide lateral areas of the hard palate, the mucous membrane is immovably attached to the periosteum of the maxillary and palatine bones. The attachment is accomplished by dense bands and trabeculae of fibrous connective tissue that join the lamina propria of the mucous membrane to the periosteum. The submucous space is subdivided into irregular intercommunicating compartments of various sizes. These are filled with adipose tissue in the anterior part and with mucous glands in the posterior part of the hard palate. The glandular layer of the hard palate continues posteriorly into that of the soft palate.

The adipose tissue found in the hard palate, lies in two symmetrical fields, that are bounded laterally by the residual alveolar ridge and medially by the ridge of the intermaxillary suture and occupies the area extending from the incisive foramen to a frontal plane in the region of the first molar tooth region. It is present in the entire space between the periosteum and the mucosa.

In the groove between the alveolar processes and the hard palate, the anterior palatine vessels and nerves, are surrounded by loose connective tissue. This region, wedge-shaped in cross section, is relatively large in the posterior part of the palate and gradually diminishes in size anteriorly.

The connective tissue components of the mucosa must contain cells responsible for:

1) the synthesis, secretion and maintenance of fibres and ground substances. This is carried out by fibroblasts, which have an
 ultrastructure that is directly related to its level of metabolic activity. These cells contain abundant rough endoplasmic reticulum; well developed Golgi apparatus, moderate amounts of mitochondria and the occasional lysosome. Current opinion is that newly synthesized topocollagen aggregates within the internal of the endoplasmic reticulum, and probably within Golgi vessels, from which it is secreted after fusion with the plasma membrane.

ii) the synthesis and storage of fat. This is carried out by the adipose cells.

iii) the defence of the tissue. The macrophage, the mast cell and other "inflammatory cells" (derived from leucocytes), carry out this task.

The macrophage's function is to ingest and break down microorganisms, foreign material and fragments of damaged tissue following injury. The mast cells are responsible for the synthesis, storage and secretion of pharmacological agents. They play a role in maintaining tissue and vascular homeostasis by the controlled liberation of anticoagulants.

iv) undifferentiated mesenchymal cells, which because of their dormancy, cannot be assigned to one of the above functional groups.

v) the constituent cells of vascular and lymphatic channels.

vi) the constituent cells of the neural element.

vii) the constituent epithelial cells of salivary glands and their ducts.
CHAPTER 4

BLOOD SUPPLY OF THE HARD PALATE

The palate is principally supplied by the right and left greater palatine arteries, which arise from the internal maxillary arteries. It is also supplied by the ascending palatine branch of the facial artery and the palatine branch of the ascending pharyngeal artery.

The basic external pattern is in the form of an arcade system, comprising of arterial anastomoses with the primary arcade consisting of the right and left greater palatine arteries. Secondary arcades are more or less symmetrically arched anastomoses, interconnecting parts of the primary arcade. Tertiary arcades interconnect the secondary arcades. Many arcades also have arteries of small lumen, through which the blood may escape to enter the lumina of nearby or distant capillaries. The nearby capillaries are in the submucosa and include those supplying nervous tissue, fat islands of the connective tissue and the tissues of the principal artery.

Maher and Swindle (1963) investigated and stated that the greater palatine nerve is a primary function of the greater palatine artery. The two structures are enveloped within a common sheath of connective tissue called the epineurium. These workers also observed the palatal venous pattern by injecting ink into the ascending thoracic aorta. After escaping into the capillaries from the arterial arcade, the ink emptied into the small collecting vessels, to reach the vein-to-vein anastomoses of the valved venous arcade system at the submucosal
level. It eventually flowed back to the two main collecting veins that lead to a venous network in the soft palate. As many of the arterial and venous arcades in the hard palate are closely related, the nervous tissue associated with the arteries, sometimes envelope parts of the veins.

The afferent vessels leave the submucosa of the hard palate by entering the reticular and papillary layers of the lamina propria in a straight line. The terminal arteriole will eventually form into the terminal plexus of true capillaries after it gives rise to metarterioles and precapillaries. Both the metarterioles and precapillaries have muscular elements present, whereas the true capillary is without muscular components. The efferent vessels of the papillary plexus are similar to the terminals of the capillaries which they drain.
CHAPTER 5

NERVE AND LYMPH SUPPLY OF THE HARD PALATE

5.1 Nerve Supply

The hard palate is supplied by branches of the sphenopalatine ganglia. One of the branches, the greater palatine nerve, divides in the roof of the mouth into branches which run in grooves in the hard palate, extending forward nearly to the incisor teeth, where they may communicate with branches of the naso-palatine nerve. The lesser palatine nerves emerge from the lesser palatine foramen and are distributed to the hard and soft palate.

The palatine nerves may contain sensory fibres from the facial as well as from the maxillary nerve and are accompanied by secretory and vasomotor fibres.

The free nerve endings in the epithelium of the hard palate commonly arise from the myelinated fibres, which pass through the dermis almost to the epithelium before they divide into two or more non-myelinated lateral branches. The branches may run closely related to the basal edge of the epithelium for some distance and send fine short twigs into the epithelium, which become extremely delicate and ramify freely among the epithelial cells. Sometimes fine branches from non-myelinated fibres, pass into the epithelium to form free endings there.

There are also nerve endings found in the middle of the dermal papillae. In some papillae, there are no organised endings but numbers
of nerve fibres of different calibre, organised in a complex pattern. Some endings in the dermal papillae are characterised by one or several unbranched extensions which pass into the epithelium. These fibrils do not branch and terminate just short of the free surface of the epithelium. Sometimes endings may be found in the dermis deep to the papillae.

Dixon (1962)\textsuperscript{53} found that a greater proportion of intraepithelial fibres appear to be located in the epithelium of the rugae than in the epithelial floor between the adjacent rugae. Their position suggests that the reason for the majority of such fibres being confined to the deeper layers of the epithelium, is to reduce the sensitivity of the mucosa. He also stated that the mode of origin of the nerve endings was important in sensory discrimination of the mucosa.

With age, Taylor et al. (1964)\textsuperscript{206} showed that the number of nerve endings in the rugae region of the hard palate, decreases slightly.

5.2 Lymph Supply

The lymph vessels of the hard palate run posteriorly and medially to join those of the soft palate. They proceed laterally to the tonsil and the palatoglossal arch, to drain into the upper deep cervical lymph nodes.
CHAPTER 6

GLANDS OF THE HARD PALATE

The glands of the hard palate are found in the post-rugal region and lie between the periosteum and mucous membrane, supported by a dense framework of connective tissue. Posteriorly these glands merge with those of the soft palate and form a thick layer between the mucous membrane and the palatal muscles.

The glands are mucous secreting with short intercalated ducts. They undergo mucous transformation and function as part of the mucous terminal portion.

There are about two hundred and fifty independent glandular aggregates in the hard palate. There are only a few glands found near the median raphé. Mostly they are clustered about the junction of the median and middle third of the area between the alveolar ridges and the raphé.
CHAPTER 7

STRUCTURES OF THE HARD PALATE

7.1 Rugae of the Hard Palate

The rugae were first described in 1889, by Dr. Harrison Allen as "a series of ridges running in a transverse direction and usually having a curved outline". They are irregular and often asymmetric in man, and were thought to be ridges of mucous membrane extending laterally from the incisive papilla and the anterior part of the raphe. However Lund (1924) stated that they were not simple elevations of mucous membrane but contain at their base, a connective tissue core called "a rugae nucleus", which consists of a tissue of immature character, rich in cells and interwoven with very delicate connective tissue fibres.

With increase in age, there is a decrease in the amount of fat in the submucosa deep to the rugae, and hence a reduction in the number of them. The rugae occupy usually about half the hard palate and those of the smallest radius of curvature are found anteriorly, with those of increasing radius further posteriorly.

The opinion is divided as to the real physiology of the palatal rugae, but some suggestions put forward, include the provision of an area for efficient mastication and the facilitating of the orientation of the tongue in the oral cavity and hence aiding in enunciation.
7.2 The Incisive Papilla

The pear shaped, or oral papilla is formed of dense connective tissue. It contains the oral parts of the vestigial nasopalatine ducts. They are blind epithelial ducts lined by simple or pseudo-stratified columnar epithelium. Small mucous glands open into the lumen of the ducts. Irregular islands of hyaline cartilage often surround the ducts.
CHAPTER 8

THE AGING OF THE MUCOSA OF THE HARD PALATE

Age changes in both the mucosa and submucosa can be observed with the occurrence of a loss of moisture and elasticity.

There is a loss of surface area and thinning of the epithelium. Acanthosis of the prickle cell layer with a thinning of the granular layer, also occurs. There is a decrease in cell division of the basal layer as well. There are contradictory studies on the keratinous layer of the hard palate in the older patient. Clinical observation and histology suggest increased keratinization with age but the reverse situation is observed with exfoliative cytological studies.

Richman and Abarbanel, in 1943, reported an increased keratinization, sometimes resulting in the formation of white plaques. Shklar in 1966 found a tendency for increased keratinization after studying the oral mucosa of one hundred patients over the age of sixty. Somewhat different results emerged from the studies, employing exfoliative cytology, from Zimmermann and Zimmermann in 1965. They stated that there was a statistically significant reduction in the proportion of keratinized squamae from the hard palate.

In the submucosa, there is also a decrease in thickness and hence resiliency, due to loss of adipose tissue and glandular tissue. The collagen content of the connective tissue is replaced by a flabbier tissue and hence the firm surface and connection to the periosteum
slowly vanishes. There is degeneration of the elastic fibres and replacement of the muscle fibres by connective tissue. The vessels and nerves because of this process are vulnerable to pressure, especially in the area of the greater palatine and incisive foramina. With the loss of glandular tissue, there is a gradual disappearance of palatal glands from the anterior of the palate, causing a reduction in salivary glands, which may lead to xerostomia.

Pickett, Appleby and Osborne (1972) looking at the gross changes that occur in the aging process found "fatty degeneration of the salivary glands results in a reduction of the amount of excretion. The mucin content increases, and the saliva becomes more viscous. The reduced occlusal vertical dimension often seen in elderly patients hinders the flow of saliva". They went on to say that "the diminished salivary production often causes stickiness in the oral cavity" and the lack of cleansing action of the saliva "renders the tissues more susceptible to infection," with painful bleeding, cracks and fissures often resulting. These workers also found an excessive growth of Candida albicans in the aged.

Burzynski (1965) examined the chemical changes in the palate of guinea pigs in relation to the aging process. He found that there was a decrease in the carbohydrate content of the non-collagenous fraction of the palate tissues with aging and an increase in the carbohydrate content of the collagenous fraction.
PART II

THE DENTURE BASE AND THE MUCOSA OF THE HARD PALATE
CHAPTER 9

INTRODUCTION

"One of the reasons that a professional dental education is essential for the man who assumes responsibility for the denture patient, is the possibility of involvement of the associated tissues in disease processes." 173

The dentist has the responsibility of diagnosis, prevention and treatment of lesions in the denture patient. Thorough visual examination including the taking of radiographs, is the first requisite in assessing the patient. Past medical histories often give clues to aid in diagnosis. Palpation of all masses should be carried out including the nodes of the neck.

Biopsy is often essential to complete the diagnosis and should be carried out on any lesion which cannot be definitely and finally identified by other routine clinical methods.

Lesions of the palate may not be related directly or indirectly to the denture but may be exacerbated by its placement on an already diseased tissue. On the other hand, some lesions may be attributable to the denture through factors such as faculty construction, age, patient habits and poor clinical observations and histories.

This section will attempt to discuss the changes and lesions that occur to the mucous membrane of the hard palate when covered by a denture base.
CHAPTER 10

THE MUCOSAL REACTION TO THE PLACEMENT OF THE DENTURE BASE

The response of the oral epithelium to the placement of a denture base, is a contentious issue. Research has been carried out, with opinion divided as to whether the keratinized layer of the surface becomes parakeratotic or hyperkeratotic. It must be assumed that better keratinization represents a better functional adaption of the mouth to an artificial appliance.

Perhaps the main question should be as Kapur and Shklar put in 1963, whether the mucosa of the palate was parakeratotic before the insertion of dentures. They carried out a biopsy study and the observations were classified into clinical and microscopic findings. The results clinically were that there was no difference with regard to texture or colour of the mucosa before and after insertion. Microscopically they tabulated their results in two categories.

1. Mucosa prior to denture insertion.

There was a distinct layer of keratin - often of parakeratotic variety with cell nuclei visible within the stratum corneum. The connective tissue was often infiltrated by varying numbers of lymphocytes, plasma cells and histiocytes. When stimulated with a toothbrush for periods of four weeks, there was revealed a generalised increase in the width of the stratum corneum compared with that of the unstimulated. The zone of keratin was increased in width, but in many places, it appeared to be irregular and discontinuous. The stimulated mucosa
presented greater downward extensions of the rete pegs in some instances.

2. Mucosa subsequent to denture insertion.

(There is a period after the insertion of a denture where the tissues of the palate, in contact with the denture base, adjust to the new environment. Inflammation, minor ulceration and patient discomfort often occur initially, but can be resolved with time and slight adjustment.)

Histologically, there was a distinct increase in the width of the stratum corneum. The keratin layer was generally of the hyperkeratotic variety, but there were zones of parakeratosis. Chronic inflammation was minimal and there was no evidence of ulceration or severe inflammation. The connective tissue collagen appeared dense and well formed.

Kapur and Shklar came to the conclusion that after wearing dentures for a given time, there resulted an excellent tissue response, coupled with the development of an obvious and well formed stratum corneum. This zone appeared to increase in width, compared with biopsy specimens that were taken prior to the insertion of dentures. It appeared that stimulation from a well adapted denture was greater than that of toothbrushing.

They commented: "If keratinization is a mechanism whereby the tissues gain a greater degree of protection against local irritation and trauma, then it appears that a well adapted denture stimulates the underlying mucosa to produce keratin. The lack of inflammation in a subjacent connective tissue indicates that the denture is not an irritant".

Their studies suggest that well adapted dentures stimulate rather than irritate the underlying mucosa, resulting in healthy mucosa with slight hyperkeratinization, rather than atrophic changes easily
susceptible to local trauma.

Markov (1969)\textsuperscript{128} agreed and stated that keratinization increased when dentures were made with respect to sound principles. The purpose of his study was to try to determine whether the amount of keratinization under dentures is related to the principles of denture construction such as occlusion, denture stability, retention and vertical dimension. It was found that basic biomechanical principles of good denture construction were conducive to better keratinization of soft tissue under dentures.

The method Markov used, was to take scrapings from various areas of the maxilla, including the palate. He found that keratinization was increased with i) satisfactory occlusion

ii) stability

iii) correct vertical dimension

iv) palatal relief present.

Keratinization was increased with lack of retention. However, this was deemed to be insignificant. With an increase in wear facets on the tooth, there was a decrease in keratinization.

However the same writer, one year earlier in 1968,\textsuperscript{127} stated that keratinization under the denture base was related to age and inversely proportional to the period of time the denture was worn. The amount of keratinization was determined by comparing "the proportion of enucleated squamae to the total cell population found in smears from scrapings of the mucosa under dentures".

His method was to divide his 105 subjects, who had experience or no experience in denture wearing into three groups. They were defined as i) edentulous patients who had never worn dentures
ii) patients who had worn complete dentures for up to four years

iii) patients who had worn complete dentures for six years or more.

Each patient's particulars, including age, smoking habits and the number of years of denture experience were noted. Again he took tissue scrapings from areas of the maxillae. The material was stained and slides prepared. 200 to 300 cells were counted in each of several fields to determine the number of completely keratinized cells in relation to all other cells.

It appeared from his results, the covering of the mucosa of an edentulous mouth with dentures, eliminates "stimulation of keratinization by food and tongue". Some low values for keratinization, in this study, were probably due to pathosis of the oral mucosa, as substantiated by clinical appearance. His investigation showed high values of keratinization for the edentulous palate when no dentures were worn and a rapid decline once dentures were placed. The high values were apparently due to food and tongue stimulation of the palatal mucosa. However once the denture was placed, this area "becomes that of the greatest clinical pathosis" because of the palate of the denture is deformed upward during the processing and finishing procedures. This region was also most susceptible to trauma during lateral and upward anterior movement of the maxillary denture.

With respect to age, as the patient grows older, the thickness of the epithelium decreases as does the stratum corneum. Thus trauma may be more painful and clinically significant in older than in younger patients even though the keratinization values, obtained by means of cytologic smears, were the same.
Men tended to show more keratinization as did patients who smoked. If the dentures were regularly removed from the mouth at night, keratinization was increased.

McMillan (1972)\textsuperscript{123} however, disagreed saying the reactions, observed by Markov were related neither to age nor to the length of time for which dentures had been worn. He investigated, cytologically, the degree of keratinization in both non-denture and denture wearing subjects. The conclusions he made were:

i) the tissue response to denture wearing is individual and is not related strictly to clinical appearance.

ii) under dentures, the mucosal surface may be as well keratinized as when no dentures are worn.

iii) when the palatal mucosa appears normal clinically, it may be poorly keratinized in a minority of subjects. The probability is that they will be females.

iv) where the palatal mucosa is erythematous, the potential for keratinization is reduced in males but extinguished in females.

v) a reduction in palatal keratinization under dentures is related neither to the age of the patient nor to the length of time for which the dentures have been worn.

Elfenbaum (1965)\textsuperscript{67} asserted that if a denture base fitted well and the palate was properly cushioned by an adequate mucosa and submucosa, and if the patient gave the denture proper care, the tissues were stimulated by mastication, phonation and expression. He backed up his statements by showing that well fitting dentures, if studied by slow motion radiography, would move constantly under masticatory forces, thus stimulating the soft tissues and increasing the keratinization of the mucosa.
Calonius in 1961, 32 went to tremendous trouble to show that
denture wearing did not cause any observable changes in the mucosa of
the palate and that fully keratinized cells accounted for about 90-95%
of the smear population of both denture and non-denture wearing patients.
Perhaps, that this may have been due to his index that labelled
"keratinized" cells as nucleated as well as anucleated - thus preventing
identification of parakeratosis.

But there exists another school of thought. Pendleton, using
biopsy material in 1951, 163 found that the horny layer was typical where
no dentures were worn, whilst parakeratosis was usual under dentures.
His view was supported by Hedégard 87 in his studies a decade later.

However, where Pendleton and Hedégard used histologic
procedures, Al-Ani, Shklar and Yurkstas in 1966, 1 used cytologic
observations of smears obtained from the "entire area of contact with
the denture". Their findings coincided with the observations that
denture wearing results in a decrease in keratinization. They stated
that the palatal mucosa, with its boney support, was more commonly
subjected to trauma, but with the coverage of it, by the denture base,
it is protected and hence did not have to be fully keratinized.
Sillevis Smith (1973) 191 agreed, with studies involving the insertion
of denture bases in patients which covered half the palate - leaving
the other half in a normal environment. This arrangement allowed for the
mechanical stimuli, provided by the tongue and food bolus during chewing,
speaking and swallowing. He found that within two weeks, the covering
of the mucosa with a plate had caused cytological changes in the
epithelium of the palate, indicating the keratinization had begun to
differ from that of the uncovered half. Again he explained, that this
was because of the reduction of the mechanical stimulation to which the
mucosa was subjected during speaking and chewing. Östlund (1958)\textsuperscript{157} observed, with the use of histological material, that the palatal mucosa could appear clinically "normal", while its epithelial surface was parakeratotic.

He showed that with denture wearing, the glandular tissue was decreased and was replaced by mainly connective tissue. The areas of glandular tissue were diminished by about 50%. However, Butcher and Mitchell (1968)\textsuperscript{27} stated that no measurements from individuals were observed in this study, over a period of time. They believed that the degenerative changes seen by Östlund, could be partly due to the ageing process.

Butcher and Mitchell carried out studies on the effect of cold cured acrylic resin dentures on the palate of monkeys under general anaesthesia. The results seen in the hard palate, were as follows.

\textbf{After the first week} - the glands of the hard palate were greatly degenerated.

\textbf{After the second week} - there was a dilation of the ducts and a reduction in the height and size of the cells of the alveoli.

\textbf{After three weeks} - vacuolation and dissolution of the cells continued to occur and most of the acini had completely disappeared. The glands were mainly replaced by dense connective tissue.

Their conclusions were that the glands of the palate, had undergone regression, resulting from the occlusion of the duct orifices or pressure on the glands.

Schilli (1967)\textsuperscript{180} stated that the early reaction of the oral mucosa to a prosthetic device manifested itself by an increase of the
mitotic rate. He experimented by fixing acrylic bite blocks to the palatal mucosa behind the maxillary incisors of adult rats. The rat could bite on the block before reaching occlusion. By means of autoradiography, it was found that all mitoses were found in the basal layer of the epithelium - compared to (say) the case of carcinoma, where mitoses are found in all layers. He concluded that there existed an inhibiting factor that prevented oral mucosa from degenerative changes.
CHAPTER 11

DENTURE SORE MOUTH

(DENTURE STOMATITIS)

(The abbreviation D.S.M. will be used for the term "denture sore mouth").

Miller (1977)\textsuperscript{135} described D.S.M. as "an inflammatory reaction of supporting mucosa that is in direct contact with a denture base. The irritation may be manifested by a beefy redness; in some instances the surface epithelium assumes a dull red, satiny appearance that is usually accompanied by some oedema".

Zegarelli et al. (1961)\textsuperscript{225} further stated that "the inflamed lesion often exhibits sharp, well demarcated borders which are limited by the denture itself". They believed that the lesions might be asymptomatic and frequently the patient was totally unaware of the existence of denture soreness, more in spite of the likelihood that it had been present for several years.

Sutherland (1968)\textsuperscript{203} called D.S.M. a "chronic inflammatory condition of the denture bearing areas of the oral mucosa, commonly the palate". The tissues became inflamed and he believed that a soreness or "burning sensations" accompanied the condition. Ritchie (1969)\textsuperscript{172} disagreed and stated that "the patient is rarely aware of the condition and seldom complains of soreness".

Newton described D.S.M. in 1962,\textsuperscript{142} with Kaaber and Bertram (1971)\textsuperscript{92} agreeing, that it may be categorized into two clinically justifiable types and several transitional forms. Newton believed that
papillary hyperplasia was one form, the other being a simple inflammation. These two workers investigated the cytologic variations and phagocytic activity in relation to the variations in the clinical inflammation pattern. The local palatal mucosal was after reaction recorded in accordance with the following criteria:

i) inflammation without ulceration, slight to moderate erythema without visible rupture of the mucosal surface, and

ii) inflammation with ulceration, moderate to strong erythema with visible rupture of the mucosa, with or without conspicuous exudation.

The collection of cytologic material in these investigations differed from earlier clinical studies, whereby the thick layer of mucin which contained large amounts of desquamated epithelium, was cleansed from the mucous membrane with a water spray. The earlier workers examined the untouched mucous membrane.

The cytologic composition of the investigated material was dominated by neutrophilic granulocytes with a regular occurrence of both macrophages and eosinophilic granulocytes. This finding agreed with the work carried out by Ritchie et al. (1969) who found that "the uncovered normal palatal mucosa exhibited the usual predominantly anucleate pattern as compared to the nucleate pattern from the adjacent affected areas". They further found a "tadpole-shaped intermediate cell" which was normally associated with malignancy, however as they were associated with a benign inflammatory condition, they believed, suggested that they arose in neoplastic ulcers as a result of secondary infection.

However Kaaber and Bertram's methods could differentiate between simple inflammation and its strong migratory activity, and inflammatory
papillary hyperplasia. They found that the strong migratory activity of simple inflammation, reflected an acute inflammation reaction in the tissue with high vascular permeability. Low vascular permeability was reflected in the decreased migratory activity of the hyperplasia.

They concluded, that the general clinical picture suggested that hypersensitive reactions were unusual in D.S.M., although the condition provided numerous opportunities for the sensitization of the mucosa both to its microflora and to the components of the denture materials. This was in direct contradiction to the findings of Budtz-Jørgensen and Bertram in 1970, who believed that poor denture hygiene and the presence of Candida albicans, were the potential factors in the development of this disease.

The conclusions that Ritchie et al. reached from their survey, were that the most common aetiologic factor was denture trauma (93%) and the presence of Candida albicans. They also believed that allergy appeared to be an unlikely cause, however hypersensitivity reactions might occur to "free monomer, toxic breakdown products of foodstuffs, or denture cleansers absorbed into the superficial substance of the denture base". Their survey showed that 74% of the patients examined with D.S.M., wore their dentures at night and 41% also had angular cheilitis associated with the condition.

Rattner in 1954, first described angular cheilitis as "a development of a scaly, eroded, fissured or macerated lesion on a red inflammatory base initiated at the angles of the mouth". In the same year, Liddelow stated that "the elderly frequently exhibited inflammation and fissuring at the corners of the mouth."
Lyon and Chick (1957),

Shuttleworth and Gibbs (1960),

Lehner (1965) and Turrell (1966) all showed there was a relationship between angular cheilitis and D.S.M. and that the infection was caused by the Candida albicans organism.

Lyon and Chick eliminated D.S.M. by the use of antifungal treatment without modification of the denture base. Shuttleworth and Gibbs isolated Candida albicans from angular cheilitis lesions. Cawson in 1963, after examining 29 patients with D.S.M., found 15 of them had angular cheilitis. Turrell in 1966 stated that trauma from unstable dentures was the major cause of the inflamed tissue because this trauma reduced the tissues' resistance to infection, especially to that of Candida albicans, which was a normal inhabitant of the flora of the mouth.

Van Reenen (1973) undertook an extensive microbiological investigation into D.S.M. and observed the following results.

i) Most surface palatal epithelial cells were infected with gram positive cocci.

ii) The numbers of bacteria tended to increase with years of denture wearing and in patients with denture stomatitis. Nucleated epithelial cells were more frequently observed in patients with D.S.M.

iii) The bacteria most frequently found from palates of patients with D.S.M. included streptococci, pneumococci and staphylococci.

iv) Under the scanning electron microscope, normal palatal epithelium had an irregular pitted surface but few bacteria seemed to be attached to the surface.

v) High antibody titres were obtained to streptococci, D. pneumomae, and staphylococci in patients with D.S.M.

vi) Nystatin and penicillin inhibited the growth of a number of the micro organisms isolated from D.S.M.
vii) Removal of dentures cured the condition.

viii) Under experimental conditions, Candida albicans penetrated an acrylic resin used routinely for making dentures. Candida albicans can be grown under reduced pressure, hence stimulating growth. Therefore, retention by maxillary dentures will provide an ideal medium for growth.

He concluded that "these studies suggest, D.S.M. was another example of an endogenous infective disease: a community of microorganisms normally resident on the denture bearing epithelium invades the tissues and causes the lesions. Trauma, the fit of the dentures, and number of other factors could be responsible for initiating this disease".

Sutherland (1968)\textsuperscript{203} agreed that the aetiology of D.S.M. was uncertain but listed the following factors that might be involved singularly or together.

i) Denture traumas, caused by occlusal imbalance, poor adaption, rough surfaces.

ii) Poor denture hygiene and food debris (seeds etc.).

iii) Chemical irritation.

iv) Allergic reactions (doubtful).

v) Sweat Gland Syndrome - Newton (1962)\textsuperscript{142} believed the ducts of the salivary glands were blocked by plugs of keratin, thus forcing the saliva into adjacent tissues, thereby producing inflammation.

Collett (1958)\textsuperscript{41} stated that one of the irritative causes of the wearing of dentures, especially shown on the palate, was that caused by malocclusion. If the centric was not correct then the dentures would tend to rotate. This would occur at least every time the patient swallowed and caused an inflammation reaction which differed in intensity with the amount of denture movement and tissue resistance.
Nyquist (1952)\textsuperscript{144} believed that denture trauma was the dominant factor in D.S.M. with the severity of the trauma not appearing to be related to the area involved. His observations also showed that

i) D.S.M. was more prevalent in women.

ii) Flabby ridges caused instability of the denture base thus initiating the denture stomatitis.

iii) There was no proof that the age of the denture was an important factor.

iv) Day and night wearers of dentures were at a higher risk.

In 1961, Mathews et al.\textsuperscript{132} stated "that the fitting surface of the denture may reproduce the finest detail of the denture bearing area and these fine irregularities may irritate the mucosal surface on slight movements of the denture in normal use. Further aggravation occurs if the dentures are worn at night or when there is any failure in prosthetic technique".

As early as 1928, Stansbery\textsuperscript{199} noted four important facts that should be remembered when placing a denture base on healthy soft tissue.

i) No undue pressure or compression should be put on the tissues when taking impressions for the denture base, as it may impair the blood supply and hence deteriorate the health of the tissues.

ii) There should be proper processing and finishing of the denture base prior to the placement in the patient's mouth.

iii) There should be no "structural defects" within the denture construction such as lack of vertical dimension and incorrect M.M.R.

iv) Since the denture base is made out of non-conducting dental material, there will be a lack of temperature change of the tissues and hence a lack of stimulation of the tissues under the base, so it is important to have periods where the denture base is absent from
the tissues.

Nearly four decades later, Elfenbaum in 1965,\(^{67}\) agreed with Stansbery stating that if the patient was careless, rarely removing the denture and did not clean the denture adequately, the tissues became affected by a lack of ventilation and there was a presence of stale stagnant saliva between the palate and the denture base. The small mucous glands in the palate discharged a thick ropy saliva which was not able to mix with the thinner serous saliva and interfered with denture retention. Circulation of blood and lymph was retarded by constant pressure.

Sharp and Fister (1966)\(^{184}\) stated that a patient with a sore mouth, that is not due to apparent infection or trauma, was one who most likely had an oral soft tissue structure and tone similar to those of low tolerance denture patients. This abnormality was characterized by atrophy and degeneration of both the mucosa and the submucosa. They believed that the changes represented the end result of an underlying abnormal metabolic function and/or a nutritional deficiency. Earlier Nyquist wrote that the sick patient was far more prone to D.S.M. than that of the healthy. Any systemic disease that lowered the resistance of the oral tissues to the oral environmental factors, might be primarily or secondarily responsible for the development of D.S.M. These could include Vitamin B complex or C deficiencies, pernicious or hypochromic microcytic anaemia, diabetes mellitus and iron deficiency.

Other factors might be endocrine abnormalities, especially in post-menopausal women and drug administration. Sutherland found that D.S.M. occurred more often in old women where the oestrogen levels were reduced and where the incidence of moniliasis was high.
McKendrick (1968) concluded that there was no evidence of tetracyclines affecting the incidence of D.S.M. However tetracyclines might enable an established condition to persist. Therefore the action of any antibiotic on the oral flora seemed to depend on the amount of that antibiotic that existed in the saliva and its permeability through the epithelium.
CHAPTER 12

INFLAMMATORY PAPILLARY HYPERPLASIA

Inflammatory papillary hyperplasia of the palatal mucosa is a specific pathologic entity consisting of an inflamed, polypoid hyperplasia of palatal mucosa. Sutherland (1968)\textsuperscript{203} reported that it only occurred usually in the vault of the palate, but in severe cases, the entire palate might become involved. Miller (1977)\textsuperscript{135} stated that it was frequently found in the anterior two thirds of the palate under an ill-fitting maxillary denture. The disease is progressive, painless and increases in size with the age of the lesion. Some researchers believe it to be premalignant.\textsuperscript{78,174}

Papillary hyperplasia is also referred to as pseudo-epitheliomatous hyperplasia and papillomatosis and is reported to occur in 2% to 11% of all denture wearers. It is mostly found on the hard palate in maxillary denture wearers, although some lesions have been reported in non-denture wearers. Fisher and Rashid (1952)\textsuperscript{71} were the first to describe the clinical and histological features of the disease.

In the same year, Thoma,\textsuperscript{208} described a lesion as a "leukoplakia with a papillomatous reaction". Although this lesion appeared malignant, it was thought to be precancerous or a carcinoma in situ. Shaffer et al. (1963),\textsuperscript{183} Thoma and Robinson (1960)\textsuperscript{209} and Waite (1961)\textsuperscript{217} believed that the lesion could transform to a malignant state if not checked. Sutherland, however believed "there is very little to suggest that there are any malignant potentialities".

Since there is only slight tenderness, the affected patient is seldom aware of the lesion.
12.1 Clinical Appearance

Fisher and Rashid described papillary hyperplasia as a diffuse papillary hyperplasia of the palatal mucosa associated with ill-fitting dentures and/or unhygienic dentures.

The mucosa of all lesions exhibited surface projections. Lambson (1966)\textsuperscript{103} put forward a classification based on the anatomy of the projections. He stated that "some areas are distinctly nodular and present large flattened elevations. Other areas are papillary, presenting like a ripple surface resembling a raspberry. They were shorter and narrower than the nodular type. Both the nodular and papillary type are separated by deep fissures". He further reported a "mossy" type which was best seen with a strong jet of air and bright illumination.

Most of the lesions contained more than one type of surface morphology, however they could be classified by their predominant feature. Lambson found that the papillary and/or nodular areas were located in the centre of the lesion; the border areas were generally mossy in type. Fissures separated all these areas. With the wearing of a denture, the nodules and papillae became flattened and edematous with the classical signs of inflammation i.e. redness and swelling. There was no ulceration present.

12.2 Histological Appearance

Waite (1961)\textsuperscript{217} stated that the tissue of a lesion was poorly keratinized with elongated branched rete pegs accompanied by a heavy infiltration of lymphocytes and plasma cells. The lamina propria contained dense collagen fibres and there was a discontinuity of the basement membrane.
Bolender et al. (1965) later described the lesion in detail and stated the degree of hyperplasia could be determined by the extent of the cellular alterations. They found keratin was not prominent, but many crevices were filled with parakeratin. Further the basal cells were well organised and sharply demarcated from the underlying lamina propria. The lamina propria consisted of a loose fibrous connective tissue stroma with a variable amount of oedema. It was heavily infiltrated with plasma cells and lymphocytes.

12.3 **Microbiological Aspects**

It has been shown that the total aerobic and anaerobic microbiological counts of tissues from papillary hyperplasia lesions were significantly higher than comparable counts from non-pathologic palatal tissues from denture wearing patients.

Because of the structure of the hyperplasia, there was a greater surface area for the collection of microorganisms and debris.

There have been conflicting reports on the association of Candida albicans with papillary hyperplasia. Cain (1936) and Bartels (1937) claimed that these organisms were usually responsible for this lesion however Nyquist disagreed, although the moist warm environment between the papillae is an excellent medium for the growth of Candida albicans.

12.4 **Aetiology**

Although there has been no conclusive evidence as to the cause of papillary hyperplasia, there have been many ideas put forward. Waite believed it was due to an "overaccentuation of the relief area" of a maxillary denture, accompanied by irritations from
denture movement and poor oral hygiene.

However, Yrastorza in 1963, with studies of over five thousand patients and sixty-four cases of papillary hyperplasia, found that the relief area was not always associated with the lesion, but poor hygiene and ill-fitting dentures were certainly contributable.

Campbell (1961), after carrying out a survey on the use of relief chambers, stated that the hyperplastic tissue often filled the entire space provided by the relief. He thought the negative pressure stimulated the proliferation of the tissue.

A study was carried out by Schmitz in 1964 to investigate the causative factors of papillary hyperplasia. He found that frictional irritation was the primary cause of the lesion formation caused by "the skidding, plunging motion of ill-fitting dentures". Malocclusion, inadequate direct and indirect retainers or "by the patient that is addicted to the habit of continually seating and unseating his dentures by his facial musculature" may also be possible causes. The lesion occurred whether a relief chamber was present or not and Schmitz showed that the lesion was not limited to the area covered by the chamber. However he stated, that although it was not a "concomitant fact", it must be considered a contributory factor in its formation.

Lambson agreed that relief chambers initiated the lesion and put forward a further argument that as the resorption of bone need not be uniform, any denture base area which did not contact the soft tissue, produced the equivalent of a relief area and hence could be a causative factor. He believed that one outstanding finding was "that the disease developed in patients who wore a maxillary denture too long" and suggested that a denture should serve for only 5 to 6 years.
In 1967, Fairchild continued his investigations along the lines of Lambson, reaching the conclusion that there was always a relief or void in a denture, and that it was due to the movement of ill-fitting dentures.

Peyton and Antony (1963) and Giglio, Lace and Arden (1952) showed that there was a dimensional change in the palatal part of the heat-cured acrylic resin complete denture which occurred during processing, resulting in significant discrepancies between the base material and the palatal part of the processing cast. Shrinkage was also exhibited by casting metal which passed from the molten to the solid state.

Therefore both the acrylic and metal bases created their own relief and Fairchild concluded that the prolonged presence of this void promoted the development of the lesion in the same way as a relief chamber. Also he noted that there was a tendency of hydrocolloids and reversible hydrocolloids to "slump" when impressions of the palatal tissue were taken, resulting in an additional discrepancy of palatal contact.

"The fit of the denture had more influence on the condition of the supporting mucosa than any other single factor..." This was what Lambson and Anderson concluded from their studies in 1967 and therefore suggested that the denture be constantly checked professionally every six months for stability and fit. They also believed that the dentures should be removed at night.

Love, Goska and Mixson (1967) stated that "the fit of the denture had more influence on the condition of the supporting mucosa than any other single factor". Further they stated that "although
all age groups should be urged to good mouth care and denture habits, special efforts must be made with the younger age groups because of their greater responsive potential to stimuli; and thus to inflammation "in adverse circumstances".

Nordenram and Landt (1969) found:

i) the highest incidence of papillary hyperplasia was in the age group between 50 and 60 years.

ii) the incidence in women was greater than men.

iii) in about half of the cases, the patient had worn the same dentures for more than fifteen years.
CHAPTER 13

TRAUMATIC REACTIONS

Dentures may introduce unfavourable mechanical factors that deform the underlying soft tissues and destroy the supporting bone by creating excessive pressures on certain areas of the denture foundation. Often the first sign of the destruction of the residual ridges under ill-fitting dentures, is the deformed and traumatized condition of the soft tissues where there had been excessive pressure.

Van Thiel (1960) stated that the irritation due to a foreign body as caused by dentures, always elicited certain reactions of the tissues with epithelial tissue changes occurring. The connective tissues displayed inflammatory, oedematous and sclerotic disturbances with the oral glands often congested and atrophied.

13.1 Chronic Inflammatory Fibrous Hyperplasia

This is also described as epulis fissuratum, prosthetic ulcers and redundant tissue. This condition typically occurs around the borders or flanges of the denture base and is only rarely seen in the hard palate. It is described as being a raised sessile or pedunculated lesion, composed of dense fibrous connective tissue, resulting from the constant trauma and inflammation by the pressure of the denture.

Lantzman (1968) and Shaffér et al. (1963) both agreed that the mouth of the elderly person was frequently the site of these chronic traumatic lesions of the soft tissue. Nordenram and Landt (1969)
reported that most cases occurred in persons between 50 and 60 years of age (37.7%), with a high incidence in women (81.2%).

Cutright (1974) found only eight of these lesions after studying 583 cases and found that it may occur in any age group. He found no correlation in his study, between this lesion and other pathologic conditions. Acanthosis and hyperkeratosis were present in nearly every case, as these were the first histologic manifestations of the body to trauma and irritation and represented the initial defensive reaction of the epithelium. There was no evidence of any malignant change.

Cutright believed that there was an unusually large number of separate pathologic entities found associated with this fibrous hyperplasia, most of them being associated with denture trauma and inflammation. His other findings included:

i) it was most often asymptomatic

ii) it might be present for weeks or years

iii) papillary hyperplasia could occur in association with fibrous hyperplasia.

13.2 Decubital Ulcer

Although this term, describes a "bed-sore", its use has been broadened to describe traumatic ulcers of the oral mucosa. It is also referred to as an acute denture or pressure sore.

It is rather uncommon to find ulceration in the denture bearing areas probably because the movements of the dentures at the peripheries is greater than that at the primary bearing areas. Rodegerdts (1964) believed that the palate only accounted for about 25% of the pressure spots. This might be because most of the time, the
denture was not in function and consequently there would not be a great deal of pressure. He believed the most prevalent area on the palate is in the region of the rugae.

Sheppard (1970)\(^{187}\) carried out a study for the purpose of determining whether pressure on the palate was also a factor in the aetiology of palatal lesions. He found that this was rare, except in the rugae area, which might contribute to lesions and resorption of the anterior ridge.

The decubital ulcer was usually associated with the insertion of a new denture or an immediate denture and might be found in the folds of inflammatory hyperplastic tissue. It had a well defined border and corresponded to the area where pressure was applied.\(^{43,124,138}\)

Elfenbaum (1969)\(^{68}\) stated that "if a palatal lesion is really a denture sore, it will be red because of the denudation of the superficial epithelial layer. The lesion will have a macerated appearance and its borders will be ragged". He also said that a foreign object, such as a particle of hard food e.g. a seed, trapped under the denture base or a small spicule of acrylic left after processing might also cause this lesion.

A decubital ulcer should not be confused with aphthous ulcers which are whitish lesions under the denture base with discrete borders and red rings around them.\(^{68}\)

13.3 Neoplasms

The basic signs of oral carcinoma\(^{91}\) are

i) ulceration - loss of continuity of the mucosal surface

ii) induration - hardness of the lesion on palpation
iii) fungation – verrucous, cauliflower-like surface
iv) elevation – elevation of part of the lesion above the normal level of the surrounding mucosa
v) fixation – attachment of the lesion to the deeper structures with loss of mobility.

McKenzie (1970)\textsuperscript{122} remarked that oral malignancy could seldom be directly attributable to denture irritation as a single cause of the disease.

The lesion is usually of the epidermoid squamous cell type, although it can be glandular. It might appear as a shallow punched out ulcer with rolled indurated margins, or it might be a papillary growth with a wide base. The adenocarcinomatous lesion is a nodular tumour which might eventually ulcerate. The early detection of palatal cancer is particularly urgent because advanced growth may cause permanent dysfunction by perforating the palate and placing the oral and nasal cavities in communication.

As the squamous cell carcinoma grows, it begins to invade in depth, but the greater initial spread proceeds horizontally across the surface. After invasive growth into the submucosa is established, lymphatic permeation and metastases of cancer cells as emboli can occur at any time.

In the early stages, these carcinomas are almost never painful or even tender and this lack of pain might persist even into relatively advanced stages. This lack of pain in the early stages is a very important point in the differential diagnosis between cancer and inflammatory lesions of the mouth.
A squamous cell carcinoma may present in the basal layers of leukoplakic epithelium. Any area of cracking, fissuring, ulceration or bleeding from leukoplakia must be adequately studied by biopsy immediately, for these changes usually indicate malignant transformation.

Cahn and Slaughter (1962)\textsuperscript{30} believed that ill-fitting dentures might have a definite influence in the initiation of cancer of the mucous membranes. Denture irritation was one of the complaints of patients observed by Sharp and Hazlet (1960)\textsuperscript{185} with oral carcinoma. They further related that atrophy and leukoplakia might be considered to be precancerous, but found together, they were more significant than either one alone.

Castigliano (1965)\textsuperscript{36} agreed that chronic irritation induced by ill-fitting dentures and malocclusion had been shown to be significant in cancer susceptible individuals.

Curran and Whittaker (1973)\textsuperscript{49} described the case of a malignant melanoma of the oral cavity, that presented with a large pigmented area covering the middle of the hard palate. The patient was an elderly woman, who had noticed some discomfort caused by her dentures. It has been stressed by many authors, the importance of pigmented lesions appearing in the oral mucosa.

13.4 Leukoplakia

Leukoplakia is any white patch or plaque of the oral mucous membrane which cannot be scraped off, reversed by removing the irritants or identified clinically as any other specific entity.

Waldron (1965)\textsuperscript{218} saw leukoplakia in more men than women in middle or later life. Both local and systemic factors probably played a role in the development of the lesion. In many patients, it appeared
to be basically a response of the mucosa to local irritation such as a denture base. Nutritional and hormonal factors might also be important. Burket (1965)\textsuperscript{26} agreed with Waldron, summarizing the predisposing factors as systemic or local chronic irritations.

Shaffer et al. (1963)\textsuperscript{183} described the various types of histopathological changes that might be associated with leukoplakia. They included hyperkeratosis, parakeratosis, acanthosis, spongiosis and dyskeratosis.

13.5 Xerostomia

Xerostomia or dryness of the mouth is a clinical manifestation of the salivary gland dysfunction but in itself does not represent a disease entity.

The clinical features of the condition is a decrease in or a complete lack of saliva, with severe alterations occurring in the mucous membrane. The mucosa appears dry and atrophic, rough and fissured, with an unnatural gloss and it loses its elasticity. The mucosa frequently bleeds and becomes secondarily infected. It seems that the vascular circulation is impaired with oxygen being conveyed via the red blood cells to the tissues. The normal balance of the oral bacterial flora is upset, conditioning the debilitated tissues for the invasion of pathogenic organisms.

Saliva is most important to the denture wearing patient for the retention of the appliance and for the lubrication and protection of the mucosa beneath the dentures. The aged edentulous patient is very susceptible to xerostomia because of the destruction of some of the salivary glands and replacement by fibrous and fatty tissue and by the pressure of the denture itself, causing atrophy of the salivary gland tissue.
Wade and Beeley\textsuperscript{216} list many systemically administered drugs that may cause xerostomia. They include

i) Antispasmodics e.g. Atropine, Dicyclonine

ii) Antihistaumases e.g. Cyclizine, Promethazine

iii) Antihypertensives e.g. Reserpine

iv) Antidepressants e.g. Nortmptyline.

Collett (1958)\textsuperscript{41} summed up the prosthetic implications of xerostomia as local trauma being caused to the tissues under the denture base because the patients can't form boli of food, since their dentures move in function without salivary lubrication.
CHAPTER 14

ALLERGIC REACTIONS

Allergic contact reactions are a delayed type of hypersensitivity and have the following criteria:171

i) the patient has had previous exposures to the allergen

ii) the reaction conforms to a known allergic pattern, such as redness; necrosis or ulceration

iii) the reaction resolves when the allergen is removed

iv) the reaction recurs when the tissues are re-exposed to the allergens at the same site

v) a "patch test" is positive.

However, Quinta and Zablotsky (1976)77 believed that it was more important in distinguishing an allergic reaction as compared to an irritational reaction. In their experience, the "irrational reaction occurs within a few to several hours after the mucosa is exposed to the resin material, however when the reaction develops after 24 hours, an allergy should be considered".

Kotilainen (1972)98 believed that the constituents of resin were capable of producing positive reactions in skin tests and on oral mucosa but that opinions vary as to whether a true allergy could be demonstrated, especially as regards to polymerized resins.

In 1952, Nyquist144 believed that the components of acrylic resin could be a possible allergen, although it was rare. In 1968, Whitfield221 and Kronman carried out studies using self and heat curing resins. After histological examinations of biopsies obtained of tissues
under denture bases after a period of thirty or more days, it was shown that there was some parakeratosis with a slight increase in inflammatory cells of the cold cured acrylic group.

In their studies, they premised their observations, by noting that metabolic alterations preceded structural changes and the utilization of histochemical procedures would "provide a more sensitive tool for the study than would be provided by solely histomorphologic techniques". They found that there was also parakeratosis under the control specimen, that is, the tissue under a chrome-cobalt alloy base. They noted that changes beneath either acrylic or metal bases were negligible, with the heat and cold cured resin bases eliciting the same response. They showed that the group wearing cold cured acrylic bases exhibited an increase of inflammatory cells in the connective tissue of several of the patients when compared with their controls. In the heat cured acrylic group, there was a marked decrease in inflammatory cells, which they said might indicate that this material was less of an irritant than cold cured acrylic. Evaluation of the histochemical findings indicated little, if any, metabolic alteration detectable between tissues subjected to heat or cold cured acrylic.

Rattner (1936)\(^{169}\) was the first to describe an occurrence of a contact allergy of the oral mucosa being associated with the wearing of a "plastic denture". In 1948, Bradford\(^{19}\) reported on a reaction to the plasticizer in the resin. Reactions to pure methyl methacrylic monomer were produced by Fisher in 1956,\(^{70}\) concluding that the liquid monomer was a potent sensitizer. Nyquist was able to sensitize most of his test group with monomer but not with polymerized resin.

Kreshover in 1958,\(^ {100}\) made the statement that acrylic resin dentures could be "the exciting agent in the development of allergic
manifestations".

Many workers have offered opinions as to what small amounts of the components of the resins were left after polymerization. In 1956, Axelsson, found that the amount of residual monomer of self-cured acrylic was 3 to 6% compared with 0.2% in a properly cured heat-cured resin. He further found in 1958 that this 0.2% of monomer could not be extracted, whereas additional amounts present in an undercured material, could be rapidly leached out by water.

Smith and Baines (1956) found that minute quantities of monomer were left but disappeared in a very short time in laboratory and mouth conditions. Smith (1959) stated that the more benzoyl-peroxide, which was used as an initiator in the polymerization process, that is left after the process, the more residual monomer was found.

Guinta and Zablotsky (1976) and Nealy and del Rio (1969) both studied virtually the identical cases, with their respective patients having a past history of multiple allergies and previous sensitizations to monomer liquid of "nail-extender kits". Guinta and Zablotsky believed that this corroborated the observation that allergic contact stomatitis was usually associated with allergic hypersensitivity of the skin.

Although self curing acrylic resin had been recognised as a tissue irritant, McCabe and Barker (1976) believed that heat cured material, as a possible cause, should not be discounted. Their findings showed that a patient could be sensitive to a denture base which although heat cured, had been incorrectly processed.

Van Scotter and Boucher (1965) asked the question: "Are tissue changes caused by the denture or the individual tissues or both?"
As part of their study, consideration was given to the histologic differences that occurred between the acrylic resin base and the vulcanite base. Their findings showed that the acrylic resin denture base, while providing some protection from foods and liquids to the underlying tissues, was a stimulating factor in itself.

However, the studies that have been discussed, certainly do not prove conclusively that an allergic reaction of the mucous membrane could be initiated by an acrylic resin denture base. It might occur on rare occasions but it could be more relevant to discuss the possibility of bacterial allergic reactions. Richie et al. (1969)\textsuperscript{172} and Salo and Hirvonen (1969)\textsuperscript{179} both observed that the mucosal reaction was a primary irritant reaction "caused by yeasts occluded on the mucosa by the denture".

Kotilainen\textsuperscript{98} reported his observations made on patients using Patch tests with denture materials. He found only one patient out of 43 was allergic to acrylic resin. The positive reactions were shown to be caused by yeasts present in the saliva. Candida albicans organisms were found in most reactions.

Danilewicz-Stysiak (1971)\textsuperscript{51} carried out a study of 40 patients with only four having a positive allergic response. Her conclusions were, that although an allergic reaction to acrylic resin components was rare, it might be allergenic in nature. A response might be elicited in combination with oral flora, or "as a result of their forming undetermined compounds during polymerization".

Stansbery, as early as 1928,\textsuperscript{199} stated that lack of temperature change of the tissues, covered by non-conducting denture material, could lead to lack of stimulation of the tissues and hence cause an
abnormal reaction. This reaction perhaps may be misconstrued as an "allergic" reaction.

Chromium-cobalt alloy denture bases were used as controls by many of the investigations, that have been discussed. The reason for its use, was put forward by Tillotson (1959): 210

"A metal base, being non-porous, can be kept cleaner and it is a conductor, allowing heat to escape from the tissues as it normally should. The tissues take more kindly to metal bases and do not undergo changes that usually follow the use of acrylic base materials."

This statement is not based on histological studies of the oral mucosa.

Choudhary and Boucher in 1965, 40 studied the histologic response of the oral mucosa of cats to metal bases - both dental casting gold and dental chromium-cobalt casting alloy - as compared to acrylic resin bases. The study was limited to changes only in the stratum corneum with special interest shown of the influence of thermal conductivity of the metal on the stratum corneum.

They found that there was no significant difference in the thickness of the stratum corneum between metal or acrylic. That is to say, that the thermal conductivity of the metals, did not affect the thickness. Their findings also showed that keratosis was more predominant than parakeratosis under both bases.

Other workers 101,166 have praised the metal base for various reasons, such as inhibiting bacterial growth, better adaption as compared to the acrylic base and no untoward tissue reaction. There have been unconfirmed reports that the nickel content in the chromium-cobalt denture base may elicit an allergic reaction.
CHAPTER 15

THE EFFECT ON THE MUCOSA OF:

15.1 DENTURE LINERS
   a) SOFT (RESILIENT) LINERS
   b) "HOME" LINERS

15.2 DENTURE CLEANSERS

15.3 CHLORHEXIDINE WASHES

15.4 DENTURE ADHESIVES

15.5 DENTURE CONDITIONERS
15.1 DENTURE LINERS

15.1.1. Soft (Resilient) Liners

"The impression surface of a denture base that is lined with a resilient material, therefore should partially absorb and provide for an advantageous distribution of imposed stresses to its basal seat." This assumption implies that an ideal material will instantaneously recover its original form after an external stress is removed and retain its resilience for an indefinite period of service. Such qualities of resilience are two of the requisite properties for a satisfactory denture liner.\(^{106}\)

Knoblauch and Reynick (1973)\(^{97}\) believed the ideal liner material was one which "functions as an interface to considerably reduce the incidence of trauma and both soft and hard tissue diseases, during the normal masticatory cycles. The trauma, as well as the disease, may be caused by localized excessive occlusal forces transmitted through the denture and liner. Chemical alteration of the salivary interface between the tissue and liner as well as the deterioration of the liner material, can be caused by both of the above as well as by resident bacteria, ultraviolet light and absorption of essential oils".

Storer (1962)\(^{201}\) and Bates and Smith (1965)\(^{9}\) agreed that a material suitable for a resilient lining in a denture should:

i) be dimensionally stable in processing and in service

ii) have a permanence of resilience

iii) minimally absorb fluids in the oral environment

iv) demonstrate a high resistance to abrasion

v) not contribute to the deterioration, weakness or distortion of the parent hard base
vi) have adequate bond strength to the rigid base
vii) be hygienic, colour stable, odourless and tasteless
viii) be non-irritating and non-toxic to the supporting oral mucosa.

Many types of material have been tried, such as natural rubber, polyvinyl chloride, polyvinyl acetate and silicone products, but all were not highly successful clinically. Some didn't achieve permanent resiliency, due to the leaching out of the plasticizer, while others were hard to manipulate due to the critical gelation temperatures, poor bond strength and high water sorption. They act to equalize stress on the mucosa for a short period of time and are sufficient to condition the tissues to loading.

The silicones seem to be the most satisfactory with their pioneer studies being carried out by Barnhart in 1960\(^7\) and Robinson in 1963.\(^{175}\) "Molloplast K-G", "Silastic 390", "Silastic 616" and "Super-Soft" are commercial names of the main "resilient" liners available at present.

Laney (1970)\(^{106}\) after carrying out clinical studies on "Molloplast K-G" and "Silastic 390", believed their use did not in itself increase the retention and stability of the denture, however it might provide mechanical retention or stability when used to engage undercut areas otherwise unavailable to the hard denture base. Its use appeared to be of significant benefit when used where there is a hard median palatal raphé. Since the processed silicone material does not contain plasticizers or organic fillers, it will not harden in service.

Clinical observations of patients with dentures lined with
"Super Soft" or "Silastic 616", over 21 months, were carried out by Means, Rupp and Poffenberger in 1971. They found that both were safe to use against oral tissues; will deteriorate and lose resilience, reducing their effectiveness in a short time; and should be considered temporary. A case report by Davies in 1970 indicated that "Silastic 390" relieved pain considerably by its use.

One of the problems associated with "Silastic 390" is the growth of Candida albicans on and within the material when in use in some patients. These fungal growths destroy the surface quality of the material and may cause irritation of the oral tissues because of a combination of surface roughness and the concentration of exotoxins and metabolic products of fungal colonies. Williamson (1968) found that "some liners were inert, some inhibitory to Candida albicans and some excitatory".

Masella, Dolan and Laney (1975), from their experiments, believed that "Silastic 390" did not supply the nutrients required for the growth of Candida albicans. They stated that daily immersion of the denture lined with "Silastic 390" in a denture cleaner or in water at 60°C, after first cleaning the denture mechanically by brushing, was an effective measure in preventing the growth of Candida albicans on "Silastic 390". Laney, again, summed up the use of soft liners extremely well, by saying:

"The dentist should not recommend use of the processed resilient liner to patients as a permanent solution to their problem unless he is prepared to observe, service and replace the liner at regular intervals... These individuals, particularly, must be thoroughly cautioned as to the limitations and added inconveniences involved in using the resilient materials as other than temporary measures."
15.1.2 "Home" Liners

These home reliners must be considered to be dangerous to the oral health of the patient who uses them. In fact Tautin (1971) believed that all levels of dentistry, from the practitioner in his office to the people on the national level, must eliminate these potentially destructive preparations from the market, and must make the public aware of the danger in using these products.

Terry, Lutes and Ellinger (1967) stated that these materials did not adapt to the basal seat under the denture but created contours that might cause severe and irreparable tissue damage. Means (1964) described the use of these reliners as a "vicious circle, with the patient relining his own dentures that have become loose. After sometime, the patient would add more material, to 'tighten' the dentures once more. Finally through discomfort, the patient will seek professional help".

15.2 DENTURE CLEANSERS

The study of denture cleansers has been undertaken by many researchers.

Langwell (1955) stated that there was a difficulty in devising a means for dealing with the removal of deposits on dentures because of the great variability in the rate of deposition and in the composition of the deposits between individuals, even in different parts of the same individual's mouth. The saliva may also vary according to the state of health or position in the mouth.

Neill (1968) considered three separate phases of "soiling of dentures".
i) Accumulation of mucin and food debris

ii) The "mucilagenous surface contaminants" appeared to gain attachment to the denture base to form a plaque. This plaque acted as a matrix for the deposition of stain derived from the breakdown of food substances. Calcium salts released from the saliva might also invade the plaque.

iii) The "tartar formation stage", where the organic matrix had become completely petrified. This only occurred on those surfaces of the dentures adjacent to the openings of the salivary ducts.

MacCallum et al. (1968)\textsuperscript{120} tabulated the properties of denture cleansers, listing their main active constituent and their disadvantages.* They also listed "requirements for an ideal cleanser".

i) Ability to render the organic portion of the deposit soluble as well as the ability to dissolve the inorganic material.

ii) Non-toxic, easy to remove, leaving no traces of irritant or antigenic material.

iii) Harmless to all denture materials.

iv) Not harmful to clothing.

v) Stability during storage.

vi) Preferably bactericidal and fungicidal.

Smith (1966)\textsuperscript{194} distinguished between the loosely adherent food debris and saliva (which is easily rinsed or wiped off) and the more tenacious deposit which required mechanical and/or chemical action to remove it. The composition of the "hard deposits" varied but it was essentially consisting of

1. an inorganic portion of calcium phosphate, calcium carbonate and smaller amounts of other phosphates

and

2. an organic portion, of protein which bands the deposits to the denture base. This was about 15 to 30% of the total deposit.

* See page 67.
**PROPERTIES OF DENTURE CLEANSERS**

<table>
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<tr>
<th>TYPE OF MATERIAL</th>
<th>MAIN ACTIVE CONSTITUENT</th>
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<tr>
<td>a) HYPOCHLORITE SOLUTION</td>
<td>DILUTE SODIUM HYPOCHLORITE</td>
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<td>2. CORROSION OF COBALT–CHROMIUM</td>
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<td>3. ODOUR.</td>
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<td>b) ALKALINE PEROXIDE CLEANSERS</td>
<td>ALKALINE PEROXIDE WITH OXYGEN – LIBERATING AGENT</td>
<td>DO NOT READILY REMOVE HEAVY STAINING.</td>
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<tr>
<td>c) DILUTE MINERAL ACIDS</td>
<td>DILUTE HYDROCHLORIC ACID</td>
<td>CORROSION OF COBALT–CHROMIUM ALLOYS</td>
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<td><strong>ABRASIVE CLEANSERS</strong></td>
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<td>a) DENTURE POWDER</td>
<td>ABRASIVE AGENT</td>
<td>ABRASION OF ACRYLIC DENTURE BASE AND TEETH</td>
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<td>b) PASTES</td>
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<td>2. DIFFICULT TO ENSURE COMPLETE REMOVAL OF CLEANSER</td>
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In brushing tests carried out by Anthony and Gibbons (1958) on heavy staining, the commercial cleansers proved to be quite effective, with stains being removed after a relatively brief brushing period. However this advantage was likely to be outweighed by the disadvantages of loss of polish, the inability to reach remote areas of the denture and the increased possibility of dropping or damaging the denture while it was being scrubbed.

Morden et al. (1956) stated that the use of hypochlorite immersion cleansers would cause corrosive action on metal bases, exhibiting significant weight loss and microscopic evidence of attack.

15.3 CHLORHEXIDINE WASHES

"Continued covering of the surface of the palate results in a progressive regression of the palatal glands. Astringents initiate activity of the glands. Frequent removal of any restoration (denture) and rinsing will keep the mucous membrane in a better physiologic state."

"Rinsing the mouth with an astringent reflexly stimulates palatal glandular activity. This activity and dilation of the ducts cause turgidity within the adjacent tissues." 27

Although chlorhexidine mouthwashes (chlorhexidine gluconate) are found to be of great benefit with peridontal problems, the use of chlorhexidine denture washes have been shown to be advantageous in also maintaining the health of the oral tissues that may be under an appliance.

One of the aetiologic factors of denture sore mouth is poor denture cleanliness, however in clinical, histological and
histochemical tests, the beneficial effects on the oral mucosa were evaluated when employing the antibacterial chlorhexidine in daily denture washing. In this same study, there was light microscopical evidence of the recovering epithelium, comprising an enhanced staining for micropolysaccarides, being possibly involved in the keratinization process and a simultaneous decrease in the glycogen content. The connective tissue exhibited a decrease in round cell infiltration and an increase in visible fibrous components – indicating a diminished inflammatory reaction.

It must be remembered that a suppression of the bacterial factor alone might not entail a complete recovery from the disorders observed in the oral mucosa. Heyden et al. (1971) believed that the possible binding of chlorhexidine to accumulations of organic materials and the acrylic base, might involve a local suppression of the number of bacteria.

Fredén et al. (1972) agreed that a binding might occur between the organic material on the denture surfaces and chlorhexidine with an affinity of the agent to the base material occurring. They further stated that "indirectly chlorhexidine may influence the metabolism and healing of the tissue by means of its antibacterial capacity, sterilizing the acrylic denture base material".

Budtz-Jørgensen & Løe's (1972) studies confirmed the above point of view and, moreover stated that the effect of chlorhexidine was comparable to that of specific antymycotic drugs such as Nystatin. However, they found that the incidence of recurrence was higher with chlorhexidine after treatment, than with Nystatin. The cause of this, might be that disinfection of the maxillary dentures did not completely suppress Candida albicans growth, neither in the palate nor in other sites
of the oral cavity. They suggested that a 0.2% solution, used as a denture disinfectant, would have sufficient strength if mouthrinsing with chlorhexidine was performed simultaneously.

15.4 DENTURE ADHESIVES

Kapur (1967) investigated denture adhesives and concluded that "although denture wearers may say that a denture adhesive helps them chew better, (these) results show that their masticatory performance is not improved". It is thought that the patient might be given a sense of false security and although, the elderly patient might not like to renew his dentures, he should be informed that his dentures, despite the adhesive, will become increasingly ill-fitting and hence cause problems that are associated with this situation.

Adhesives are available either in a gel or powder form and if either type is used, it is important that after daily wear, the remains of the product be removed so that a build-up of the material does not occur, thus preventing a nidus of infection to occur.

15.5 DENTURE CONDITIONERS

In 1961, Chase coined the phrase "dynamic adaptic stress" when referring to a tissue conditioner that is "non-setting, flows and yet is resistant". This material had two uses, one as a conditioning material, the other as an impression material. It is placed in the denture so that it acts as a cushion between it and the inflamed tissues and is worn for several days and nights. The pressures on the denture base, created by oral activity, are the forces utilized to massage the tissues to better form and health, and at the same time condition them to the thrust pattern of the dentures. After a sufficient period of
time, tissues that have been beaten down will recover their form and hyperplastic tissues will become hardened and the inflammation will be reduced.

Many authors have reported studies concerning the chemical compositions of these products.\textsuperscript{20,21,11,223} They are generally manufactured as a powder and liquid. The powder is mainly acrylic resin and is mixed with a liquid plasticizer, generally an alcoholic solvent. The result is the formation of a soft and elastic gel with a high percentage of flow under compression. These conditioners maintain their optimal characteristics from three to fourteen days. It appears that once this material is placed in the denture base, the patient’s saliva, diet and oral hygiene dictate the length of time, it can be left in the denture. "Viscogel" and "Hydrocast" are two commercial brands available at present.

These materials act as soft cushions that allow the deformed mucosa to push against them and gradually recover their normal form. Boos in 1959\textsuperscript{18} stated that even while a denture might be lined with a tissue conditioner, it should be removed by the wearer as often as possible. Krammer (1971)\textsuperscript{99} believed the patient should keep his dentures out of his mouth for prolonged periods, to rest the deformed tissues. This obviously was unrealistic, thus making the tissue conditioning materials an acceptable alternative solution.

Bláhová and Neumann (1971)\textsuperscript{11} found that the quality of retention of dentures, lined with a tissue conditioner, was not improved by their presence. Lower values for retention of dentures, lined with soft-curing resin are caused by the fact that the lining contained a certain quantity of saliva which is easily released. Battersby, Gehl and O’Brien
(1968) found that the retention of dentures was reduced by increasing the thickness of the resin material. This can be explained by the fact that a greater quantity of soft-curing resin contains and also releases a greater quantity of liquid into the space under the denture. Therefore, dentures lined with a thick layer of the resin, would have decreased retention.
CHAPTER 16

RETENTION OF THE DENTURE TO THE PALATE

Inadequate retention of the denture base may cause damage to the underlying mucosa of the hard palate in two ways: by the constant movement of the denture with injury to various sections of the adjacent and supporting tissues occurring; or by the patient acquiring a habit of reseating the denture with the fingers or tongue. In either instance, the physiologic endurance of the mucosa is overtaxed and pathologic problems result. 105

In addition, loose dentures may have a disturbing effect upon the patient's phyle which leads to the formation of "tenacious and stubborn habits".

Lawson (1972) 110 listed three requirements for retention.

i) The denture must cover the greatest possible surface area of the supporting tissue.

ii) The fitting surface of the denture must be in close contact with the supporting epithelium.

iii) There must be a peripheral seal around the entire border of the denture. This implies that the border tissues must be capable of providing a seal, and for this purpose, they must have adequate elasticity.

Hardy and Kapur (1958) 85 arbitrarily classified the variables that might influence the retention of the denture base as physical and physiologic, mechanical, psychologic and surgical. The physical,
physiologic and mechanical variables will be discussed in detail. The psychologic (such as intelligence, expectation, apprehension and gagging of the patient) and the surgical (ridge extension and implants) factors are beyond the bounds of this discussion.

16.1 Physical and Physiologic Factors

The physical classification involves such forces as adhesion, cohesion, capillarity and/or atmospheric pressure, whereas the nature of the supporting tissues, the patient's neuromuscular control, ridge characteristics and related considerations may be included in the physiologic category.

Campbell (1954)\textsuperscript{33} after examining the literature relevant to this topic, revealed "that there is no unanimity concerning the actual physical forces responsible for the retention in situ of the complete maxillary denture. Adhesion and cohesion of molecules, capillary attraction, the "suction" provided by atmospheric pressure, "interfacial surface tension", the pressure of the atmosphere relative to that of the fluid film are mentioned as contributors in whole or in part to the picture".

Retention, other than that due to the muscular or occlusal forces was described by Roydhouse (1960),\textsuperscript{178} as a "phenomenon associated with flow". As the denture moved or tended to move away from the mucosa, the increasing volume beneath, was filled by the flow of air or saliva from the periphery. All features that impeded flow increased retention and vice-versa.

Staintz (1948)\textsuperscript{200} after analysing the part played by the fluid film in denture retention, indicated that it results from the differences
in pressure between the atmosphere and the fluid film. He stated that although the adhesive and surface tension forces were not large, they did not in themselves exert an appreciable retentive force on the denture. They did, however, sustain the pressure difference across the meniscus of the fluid film and this difference, when multiplied by the projected denture area, equalised the force exerted on the denture.

In 1945, Snyder et al., stated that under reduced atmospheric pressure, certain bodily physiologic changes occurred such as a decrease in salivation and a decrease in muscular control. Thus they thought that, although it was a negligible factor, since there was less saliva between the tissues, there would be less adhesion. However, from their experiments, they reported that a 70% decrease in pressure caused a 50% decrease in the retention of dentures. Therefore adhesion was thought to play a significant part, because the retention was not directly proportional to the decrease in pressure.

Campbell (1954) observed that a decrease in the fluid film thickness caused an increase in the retention of a denture base, concluding that in every case where a denture could be dislodged in a particular area, the amount of the force necessary to dislodge it, was actually increased by the apparent lack of the fluid film. However, Östlund (1960) disagreed, believing that the retentive forces decreased in proportion as the surface tension of the "intermediate film" was lowered.

Also in 1960, Craig et al. carried out experiments with results suggesting that "capillary forces are the principal physical forces involved in denture retention". The capillary force was influenced by the continuity of the saliva film and any discontinuity would reduce
the retentive force. Reduction of the outside pressure would contribute to film discontinuity.

Avant (1973)\(^4\) supported the conclusion that both atmospheric pressure and the intimate relationship of the denture base to the underlying tissue, contributed to denture retention. The effect of water sorption of the denture base did not significantly alter the retention of the base. This was in contradiction to Campbell's findings in 1956, that increased water imbibition of the acrylic resin was reflected in increased retention.

16.2 Mechanical Factors

"The functional success or failure of denture construction depends upon the health of the soft tissues which support and retain the denture bases. Denture base support is not a physiologic function of the oral mucosa, but it is an activity imposed upon the mucosa by the dentist with the patient's consent. Dentures constructed in conformity with the biologic needs of the oral mucosa have been found to fulfil all reasonable demands for stability and retention without risk of pathologic changes."\(^{177}\)

The mechanical features contributing to the retention of the maxillary denture base to the hard palate can be divided into four sections:

i) Pressure Areas of the Palate
   a) Palatal Relief
   b) Post Dam Area

ii) Occlusion

iii) Design

iv) Materials.
16.2.1. Pressure Areas of the Palate

a) Palatal Relief.

The torus palatinus (the bony outgrowth in the centre of the hard palate) contributes to the instability of the denture by acting as a fulcrum for undesirable leverage and hence should be relieved. Many researchers believe the centre of the hard palate should always be relieved, however, Raybin (1949)\textsuperscript{169} noted that if the mouth was of uniform texture throughout, the vault would require no central palatal relief. Laney and Gonzalez (1967)\textsuperscript{107} agreed, stating that "the ultimate goal is to distribute the masticatory stresses adequately over as wide an area as feasible, to provide a more lasting retention of the denture and to give the patient maximum comfort".

If relief is required, it is usually near the centre of the stress bearing area of the maxillary denture, thus creating a problem of sacrificing the stress bearing area and retentive seal surface for palatal relief and retention.

b) Post Dam Seal

An exaggerated post-dam in the posterior margin of a denture base, can be a direct cause of instability. Raybin stated that if the post dam was too wide, its presence on the soft tissues of the palate might cause a reverse pressure, thus loosening the denture. If it was too deep, bruising, atrophy and inadequate functioning of the post-dam would follow.

The posterior palatal seal compensated for dimensional changes incurred in processing acrylic dentures. Avant (1973)\textsuperscript{5} carried out studies to determine if a posterior palatal seal was necessary for complete denture retention and if altering the type and location of that seal,
affected retention. His results showed that a posterior palatal seal was necessary for optimum retention of maxillary dentures. This seal may be achieved by impression techniques that place pressure on the selected areas across the palate border of the impression\(^{139}\) or by carving the cast on which the denture is fabricated.\(^{85}\) The former method appears to be more desirable.

16.2.2. Occlusion

Since the soft tissue of the hard palate is exceptionally sensitive to traumatic insults, it is important that the occlusion of the prostheses be developed to function efficiently and with the least amount of trauma to the supporting tissue. The occlusion should be refined in centric relation as well as the range of functional and non-functional movements of the mandible.\(^{156}\)

16.2.3. Design

The retention of acrylic resin denture bases of three different designs was measured clinically under various conditions of contact by Skinner et al. in 1953.\(^{192}\) The results obtained confirmed that the retention of the base was measured by the post-dam and the peripheral seal. They also found that the "roofless" base exhibited the least retention.

They also put forward the idea, that retention was greater in the anterior portion of the denture. "Suction" or "vacuum" chambers appeared to have no influence on retention.

16.2.4. Materials

Swartz in 1966\(^{204}\) tested heat-cured acrylic resin, cold cured resin, chrome-cobalt, aluminous and porcellain materials to assess which
material would exhibit the best retentive properties.

The cast aluminium alloy bases were the most resistant to vertical dislodgement forces in all tests, while the heat-cured acrylic resin bases were consistently dislodged with a small amount of force. The alumina alloy was 99.99% pure in aluminium and magnesium components.
CHAPTER 17

THE EFFECT OF DENTURES ON TASTE PERCEPTION

Widely divergent views exist concerning the role of the palate in gustatory function and hence, whether there is a loss of taste sensation associated with palatal coverage, is a subject of great controversy.

Henkin, Graziadei and Bradley (1969) stated that taste in man had traditionally been considered a function of the tongue. On the tongue, taste had been commonly associated with the function of various structures called taste buds and their associated neural innervation which reside in larger lingual structures (papillae). They continued by saying:

"Both the sour and bitter tastes could be appreciated on the tongue, but quantitatively these qualities are appreciated at higher concentrations on the palate. The palate is the most sensitive anatomical area for the tastes of sour and bitter. Salt and sweet can also be appreciated on the palate but not at concentrations as low as those appreciated on the tongue."

These workers carried out studies of taste thresholds with and without maxillary dentures in place and found that the thresholds for salt and sweet were normal but thresholds for sour and bitter were markedly elevated. However, they concluded by stating:

"...the wearing of a maxillary denture for many years could be a source of frequent mechanical irritation to the structures in the palatal epithelium, especially as the appliance loosens and irritates over time."
In this manner, the palatal papillae and taste buds might be damaged in some as yet unspecified manner."

Mones (1950), Chambers (1937), Giddon et al. (1954) and Martone and Edwards (1962) have indicated a loss of taste sensation due to factors such as inability to press food against the hard palate and the inability to confine the food against the smooth polished surface of the dentures by the tongue.

Laird (1939) and Cohen & Gitman (1959) agreed that there was no correlation between the wearing of dentures and the lack of taste sensation. However Kapur, Collister and Fischer (1967) believed that there was an increased response of the parotid gland when dentures were worn, due, they suspected probably to the confinement of food by the dentures in the areas of taste reception.

Strain (1952) was not convinced that there were taste buds present in the mucosa of the hard palate, thus causing Shannon, Terry and Nakanoto (1970) to discount the role of gustation in the alteration of the rate of flow of the parotid gland and to assert that the decrease rate of flow associated with the tissue coverage, was likely to be related to tactile deprivation.
CHAPTER 18

METABOLIC DISORDERS AFFECTING THE MUCOSA

It has been suggested that the prosthodontist's problem was not really that of unsatisfactory dentures. Margolese (1970) stated that "when it is possible to exclude the faulty denture, local conditions and nutritional deficiencies, the fault must lie in the tissues, the bone and the covering mucosa, since these constitute the primary stress-bearing areas of the ridge and the palate".

He continued:
"A healthy basal seat could probably withstand the stress of something less than a perfect denture. Conversely, a perfect denture would probably injure an unhealthy tissue."

Margolese attempted to simplify the problem of the question: "Why does the tissue fail to fulfil its function?" by investigating the problem from the standpoints of those hormones responsible for cell formation and maintenance (anabolic) and those which cause cellular destruction (catabolic).

He instanced the processes involved in the formation of the mucous membrane.

"Prior to puberty, the mucosa is a thin layer made up of almost entirely of small round cells with large nuclei. Under the influence of oestrogen and progesterone, the cells gradually increase in size with a reduction in size of the nuclei. Thus, the middle of the tissue is made up of intermediate cells. As the process continues, the cells become somewhat less round and have smaller nuclei and they form the outer part of the mucous membrane. These are mature squamous
cells, usually termed cornified."

"Again any imbalance between anabolic and catabolic hormones will cause reversion of the mucous membranes to its prepuberal status or to an intermediate stage depending upon the degree of imbalance."

10.1 Diabetes Mellitus

Diabetes Mellitus is a disorder of carbohydrate metabolism and is characterized particularly by an increase in blood sugar (over 180 mg per cent) and usually glycosuria. The chief hazards are ketosis and vascular changes.

Ulceration may be a fairly common complication, particularly in the lower extremities in the disease. Although, as O'Driscoll (1966) has referred, there are no oral lesions specific to the disease, lesions can occur in the mouth.

The classical and subjective and objective symptoms that may indicate the presence of diabetes is a dry or burning mouth. In an uncontrolled diabetic, there may be in addition generalized edematous mucous membranes.

However, as early as 1942, Sheppard stated:

"The general mouth symptoms of stomatitis, burning, ulceration, dryness and an acetone odour, together with many tongue symptoms, appear to be relics of the preinsulin era. Nearly all cases of diabetes mellitus are treated and controlled and the mouth and tongue differ little from those of the non-diabetic; nor is the reaction to dentures different."

In 1968, Wesson reported a case of mature onset diabetes, that was diagnosed from a palatal ulceration. However, after investigation and treatment, the conclusion was made that trauma from an ill fitting
maxillary denture was the precipitating cause. He believed that diabetes seemed to interfere with the healing of the lesion.

18.2 Anaemia and Vitamin Deficiencies

Changes in the oral cavity associated with various types of anaemias have been recognised for many years. Iron deficiency anaemia and pernicious anaemia all have an effect on the oral mucosal tissue, however very rarely is there seen any changes in the palatal epithelium.

Drinnan (1970)\textsuperscript{66}, writing about iron deficiency anaemia, stated that the occurrence of oral mucosal changes in certain patients with iron deficiency with or without anaemia, was probably the result of many secondary factors such as alterations in the metabolism of pyridoxine, folic acid and iron containing enzymes.

Pernicious anaemia (a macrocytic disease caused by a lack of Vitamin B12 in the bone marrow) related to stomatitis, cannot be characterized by a specific description. But Little (1975)\textsuperscript{113} believed that "nevertheless persistent or recurring non-specific stomatitis of unexplained local origin may be an early clinical manifestation of pernicious anaemia. Long term follow-up of patients with persistent or recurring stomatitis is essential to rule out pernicious anaemia or to intercept and treat the disease when the blood picture becomes diagnostic".

A dry mouth usually accompanies pernicious anaemia, together with a disturbance in taste. However those symptoms may also occur in iron deficiency anaemia as well as other specific and non-specific diseases. Hence the oral symptoms and signs as Hjørting-Hansen & Bertram (1968)\textsuperscript{90} said, are only suggestive. The final diagnosis could
only be established on the basis of laboratory tests.

Kimball (1954)\textsuperscript{96} listed "vitamin antagonists" such as nicotine, alcohol, aspirin and barbituates, as a cause of tissue soreness under the denture. These antagonists would destroy or substitute themselves for vitamins in their essential enzymes systems. He stated that they mainly act as Vitamin C and B complex and could cause a deficiency, thus eliciting a tissue soreness.

He also believed that a deficiency in vitamin B complex and vitamin C could cause chronic soreness beneath dentures with the vitamin C deficiency exhibiting a type of scurvy in the mouth with extreme hyperaemia. A vitamin A deficiency could cause widespread injury through the imbalanced production of keratin and the disturbed sequence in the forming, maturing and shedding of epithelium.
**SUMMARY**

When the tissue of the hard palate is abused by ill-fitting denture bases or the new environment that it is placed, it is important that it be identified, treated and thenceforth prevented. Even the best intended denture base has the inherent potential of causing trauma to the basal seat tissue.

Whether the epithelium of the tissue becomes hyperkeratotic or parakeratotic is difficult to ascertain and perhaps the tissue should be examined prior to the insertion of the base, especially with patients with long standing denture experience, to monitor its degree of keratinization. Also, the area of the palate from where the studied specimen has been taken, may be of great significance. However these observations really are of no real consequence, for it is more important to note the pathological factors that may occur under the base.

It is essential that a clean denture base be placed on an adequately ventilated tissue seat. That is to say, that is important that the denture be removed at regular intervals to maintain a reasonably normal environment for the usually covered tissue. With constant coverage, tissue will gradually become necrotic, due to lack of stimulation and problems such as denture stomatitis will ensue.

It appears, that whether this stomatitis is represented by entities such as denture sore mouth, ulcers or papillary hyperplasia, it is mainly caused by ill-fitting dentures. Therefore the prosthodontist may be largely responsible for causing this diseased state. Great attention should be paid to tissue examination, relief areas, extensions
of the base and design as well as accurate impressions and correct
centric and working movements, to ensure that the retention and
stability of the denture is optimal. This is especially important
in the case of immediate dentures, that have to be checked regularly
in their initial stages. Traumatic ulcers, if left, can lead to
precancerous problems, however the incidence of neoplasms in the palate
is surprisingly low.

Although there is nothing better than the normal environment
of the mouth to maintain the health of the tissue, the best possible
environment that can be achieved in the circumstances, should be maintained
between the tissue and denture base. Clean dentures, regular mouthwashes,
frequent periods without wearing of the denture and perhaps stimulation
will help to maintain the health of the tissue. The patient's
constitution and health may be indirectly important to this environment.
Xerostomia will cause an unsuitable environment between the tissue and
base and inflammation will quickly develop.

Chromium cobalt and gold cast denture bases (especially
partials) enhance the environment of the tissue in as much as it is
not as necessary to cover it to such a degree as is necessary with
acrylic resin bases. The problem of allergic reactions occurring
with the placement of acrylic bases on the tissue of the hard palate
is probably a minor one. It is extremely rare to see a true allergic
reaction on the palate and perhaps those workers that have reported
allergic reactions, might be recognising denture stomatitis caused by
other factors. It is possible that some components of the methylnetha-
crylate base may leach out, thus causing a deterioration in the
environment between the tissue and base. It could be thought that if
the denture base elicited an allergic response to the epithelial
tissue, a reaction might also be expected to be seen on the tongue
or buccal mucosa. Incomplete rinsing of "denture cleansers" from the
base could also be a considered aetiologic factor.

"Denture cleansers" are not really important in the maintenance of denture hygiene. Warm water and soap used with a firm brush will prove to be just as effective. A lingering pleasant taste might be one of the advantages of these "cleansers". The less said about "home reliners" and "denture adhesives" the better. They appear to cause more problems as well as giving the patient a false sense of well being as far as fit and function are concerned.

The mucosa of the hard palate under the denture base is an extremely traumatized tissue. It has to withstand abnormal pressures, nidi of infection and constant abuse. It has to survive in an unnatural environment.

All possible precautions should be utilized by the prosthodontist and patient alike, to care and maintain the tissue in a "near to normal" healthy condition. It is certainly a difficult responsibility, but one that has to be met, especially by denture wearing patients, as they have no alternative to this problem.
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