ULCERATIVE LESIONS
OF THE
ORAL MUCOUS MEMBRANE.

This critical review of the literature is submitted in support of candidature for the degree of Master of Dental Surgery.

N. Quinnan
One of the commonest pathological lesions of the mouth for which patients seek relief is ulceration of the oral mucous membrane. Aetiological factors are many and varied. In some they are obvious and easily remedied; in others they require careful investigation; and some have an unknown aetiology and are resistant to known methods of treatment. For these we should heed the advice of the late Sir James Paget (1870):

"We ought not to set them aside with idle words about 'curiosities and chances'. Not one of them is without a meaning. Not one of them but might be the beginning of excellent knowledge".
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CHAPTER 1.

THE ORAL MUCOUS MEMBRANE.

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INTRODUCTION

A mucous membrane is defined as "the lining of any body cavity opening to the outside" (Fer-Mar, 1951; Silverman, 1961). This lining membrane is thin and vascular, and since it secretes a viscid, protective substance, mucous, it is termed a "mucous membrane". Such a membrane is always composed of epithelium and connective tissue. The outer layer of epithelial cells, highly specialized to protect, absorb and secrete, overlies the connective tissue from which it derives its nourishment. Epithelial cells are cemented closely to one another with a matrix resembling the "ground substance" in which connective tissue cells are loosely arranged (Bloom and Fawcett, 1962). The basic structure of a mucous membrane is modified as its function varies. In all parts of the body during normal function it is subject to "wear and tear", and therefore it must be capable of rapid cell division to renew the tissue.

DEVELOPMENT OF THE ORAL MUCOUS MEMBRANE.

Arey (1960), describes the primary tissue of the digestive system as endoderm. Except for two small parts of ectoderm at the caudal and cephalic ends, endoderm furnishes the epithelial lining of the digestive tract and gives rise to both large and small glands - the liver, pancreas, gastric and intestinal glands.
The accessory coats of the alimentary canal, such as muscle and connective tissue, are derived from an investment of mesenchyme (Cunningham, 1953).

After the loss of the bucco-pharyngeal or oral membrane, in the fourth week of embryonic life it is impossible to determine the exact junction of ectoderm and endoderm in the mouth, (Le Gros Clark, 1958). The portion of the oral cavity derived from the fore-gut is lined with endoderm, and that derived from the cephalic ectodermal portion, or "Stomodeum", is lined with ectoderm.

Hamilton et al., (1959), and Cunningham (1958), consider that the boundary may be represented by an imaginary plane extending from the anterior part of the body of the sphenoid bone to the floor of the mouth, lingual to the mandibular process. Arey (1960) states that the epithelium of the roof and much of the sides of the mouth and lower lip is probably ectodermal in origin.

The endoderm lining the alimentary tract is at first lined by a simple epithelium consisting of a single layer of cuboidal or columnar cells. Where the processes of absorption predominate in the adult, this type of epithelium persists relatively unaltered. In other regions, such as in the mouth, the need for protection against mechanical injury is met by the development of a multi-layered epithelium in which the cells are arranged in "stratified" formation (Le Gros Clark, 1958).
GENERAL STRUCTURE OF THE ORAL MUCOUS MEMBRANE

The oral mucosa is a multi-layered coat in which there is constant growth outward from the deeper layers. Undifferentiated germinal cells are transformed into structureless cells by a series of complicated structural and chemical alterations. All cells except the basal cells are removed from direct contact with the vascular supply and exchange of metabolites is maintained by diffusion across fluid-filled intercellular spaces. Cells are linked by a system of tonofibrils and intercellular bridges which enable the epithelium to combine a high degree of flexibility with considerable mechanical strength. In fact, the oral mucosa is "a complicated architectural system in which the very building blocks are in constant flux" (Rothman, 1954).

The structure of the oral mucosa appears to vary in accordance with the function of specific areas and the biophysical influences relating to them, (Gardner, 1961; Silverman, 1961; Orban, 1962). Around the teeth and on the hard palate the epithelium is toughened or "keratinized", to withstand heavy masticatory forces. On the floor of the mouth, which is largely protected by the tongue, the mucosa does not require re-inforcing. Le Gros Clark (1953), states that any columnar epithelium, if exposed to mechanical stress, may be converted into stratified, keratinised epithelium.

The stratified squamous epithelium lining the oral cavity is separated from the underlying lamina propria and submucosa by a basement membrane, (See Figure 1). This membrane is a condensation of intercellular connective tissue substance, (Bloom
Fig. 167. Diagrammatic drawing of oral mucous membrane (epithelium, lamina propria, and submucosa).

(Orban, 1962)
Fig. 100. Basal Layer of the Epithelium

Fig. 102. Granular and Cornaceous Layers

(Cahn, 1941)
and Fawcett, 1962). Cahn et al (1961), assume that it is permeable to intercellular fluids which nourish the basal cells. The single layer of elongated basal cells is anchored to the basement membrane by short, protoplasmic processes, (Orban, 1962), (See Figure 2). Above it are several layers of larger polyhedral cells, separated from each other by intercellular spaces in which the intercellular bridges appear prominent (See Figure 2). Because of these connecting "spines" this layer is referred to as the "prickle cell" layer, or "stratum spinosum". Nearer the surface the prickle cells flatten and widen considerably and, if a surface keratin layer is present, the cells grade into a "granular" layer or "stratum granulosum", in which the cytoplasm contains numerous basophilic-staining, keratohyalin granules (See Figure 3). The outer layer of keratin, or "stratum corneum", appears structureless since individual cell outlines and cell nuclei have practically disappeared, (Scott and Symons, 1961).

The outer layer of connective tissue comprising the oral mucosa, the lamina propria, is a layer of dense connective tissue of variable thickness giving off papillae to interdigitate with the epithelial "rete pegs" (See Figure 1). The papillae carry a nervous and vascular supply and by forming a continuous network of ridges, facilitate exchange of metabolites between blood vessels and epithelium, (Noyes, 1960; Orban, 1962).

The submucosa attaches the mucosa to underlying structures and carries a vascular and nervous network which extends into the
lamina propria and so supplies the epithelium, (Orban, 1962). It varies from a dense, firm, collagenous tissue to a loose, elastic one, according to the mobility and function of the mucosa.

CLASSIFICATION OF THE ORAL MUCOUS MEMBRANE

Orban and Sicher (1946), in an important contribution to the literature, introduced a detailed classification of the oral mucosa, (See Table 1). Their broad subdivisions of the oral mucosa into; (a) "masticatory", (b) "lining", and, (c) "specialized" types, have been retained to the present day, (Orban and Wentz, 1960; Noyes, 1960; Sicher, 1960; Gardner, 1961; Scott and Symons, 1961; Silverman, 1961; Orban, 1962).

(a) The Masticatory Mucosa includes the gingivae and covering of the hard palate which are subject to strong forces of pressure and friction. Both areas have a keratinized surface layer and a thick and firm lamina propria with firm attachment to the underlying structures. Gardner (1961) compares the keratinized masticatory mucosa with other areas of the body withstanding great mechanical stress, such as the skin of the palms of the hands. However, some earlier authors such as Sognnaes and Albright (1956), reported that keratinization of the oral mucosa is not as complete as that of the skin, although it is more complete than that of other mucous membranes.

(b) The lining mucosa of the oral cavity is a simple,
## CLINICAL CLASSIFICATION

<table>
<thead>
<tr>
<th>CLINICAL CLASSIFICATION</th>
<th>Physiological Characteristics</th>
<th>Histologic Characteristics</th>
<th>Attachment Type</th>
<th>Underlying Structure</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>I. MASTICATORY MUCOSA</strong></td>
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<tr>
<td>A. SIMPLE</td>
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<tr>
<td>1. Marginal gingivae</td>
<td>Immobile</td>
<td>Fused into single layer</td>
<td>Firm</td>
<td>Bone of tooth</td>
</tr>
<tr>
<td>2. Attached gingivae</td>
<td></td>
<td>thick, dense, compact</td>
<td></td>
<td>structure</td>
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<tr>
<td>B. CUSHIONED</td>
<td>Resistant to pressure and stress</td>
<td>Thick, dense non-elastic</td>
<td></td>
<td>Bone</td>
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<tr>
<td>(Hard palate)</td>
<td></td>
<td>Bands of thick collagen, Spaces filled with</td>
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<tr>
<td>1. Anterior fatty zone</td>
<td></td>
<td>- 1. Fat cells</td>
<td></td>
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<tr>
<td>2. Posterior glandular zone</td>
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<td>2. Glands</td>
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<td><strong>II. LINING MUCOSA</strong></td>
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<tr>
<td>A. TIGHTLY ATTACHED</td>
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<tr>
<td>1. Soft palate</td>
<td>Not specialised to resist friction or pressure. Ability to change surface area and volume.</td>
<td>Fused to fascia</td>
<td>Muscle (cheek or soft palate)</td>
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<td>2. Cheeks or lips</td>
<td></td>
<td>Thin, dense elastic</td>
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<td>3. Inferior surface of tongue</td>
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<td>1. Strands of dense connective tissue</td>
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<tr>
<td>B. LOOSELY ATTACHED</td>
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<tr>
<td>1. Vestibular mucosa</td>
<td>Great mobility and ability for rapid absorption</td>
<td>Thin, dense elastic</td>
<td>Loose connective tissue</td>
<td>Loose Bone or muscle</td>
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<tr>
<td>2. Alveolar mucosa</td>
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<tr>
<td>3. Sublingual mucosa</td>
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<tr>
<td><strong>III. SPECIALIZED MUCOSA</strong></td>
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<tr>
<td>Dorsum of tongue</td>
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<td></td>
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<tr>
<td>A. Filiform, fungiform papillae anteriorly</td>
<td></td>
<td>Partially Fused to fascia</td>
<td>Firm Muscle</td>
<td></td>
</tr>
<tr>
<td>B. Vallate papillae</td>
<td></td>
<td>Dense, filiform elastic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C. Lingual follicles</td>
<td></td>
<td>Filiform papillae</td>
<td></td>
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<tr>
<td>posteriorly.</td>
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</table>
protective covering characterized by a thin, non-keratinized epithelium and a thin lamina propria. It is either loosely attached to the underlying tissue to permit mobility or firmly attached to prevent folding and injury (Orban and Wentz, 1960).

Meyer and Medak (1962), point out that a difference in the degree of stretch rather than the degree of stress, constitutes the chief difference between the buccal and palatal mucosae. These authors suggest that a completely keratinized surface layer is absent in the buccal tissues because such an inflexible covering would be unsuitable as a lining for muscular tissue which is continually changing its surface area.

(c) The specialized oral mucosa comprises the covering of the dorsal surface of the tongue. In many animals it has a masticatory function. However, in man this function is considered insignificant, (Sicher, 1960). The specialized structures consist of the prominences of the filiform, fungiform and vallate papillae and on the pharyngeal surface of the tongue, the lingual follicles.
Differences between the structure of the skin and that of the oral mucous membrane

"Superficial examination of the skin and the oral mucosa might lead one to believe that the two are identical structures, since both are covered by a layer of stratified squamous epithelium resting on fibrous tissue" (Levy, 1963). However, distinct differences exist between the two tissues. The appendages of the skin include sebaceous glands, hair follicles and sweat glands. The normal oral mucous membrane does not exhibit these structures and has as its appendages mucous glands, salivary glands and teeth. In the maturation process of the epithelial cells of the skin a "clear layer", or "stratum lucidum", is present immediately beneath the stratum corneum. A stratum lucidum does not occur in either the keratinized or non-keratinized oral mucosa (Gardner, 1961). Levy (1963), emphasises that wet and dry tissue surfaces exhibit certain differences. For example, the surface epithelial cells of the skin, which are exposed to air, tend to be dehydrated in comparison with the surface epithelial cells of the oral mucosa, which are normally exposed to a wet environment.

The vermillion border of the lip is the transitory zone between the skin covering the outer surface of the lip and the oral mucosa. The lip epithelium is non-keratinized and contains densely-arranged connective tissue papillae which carry tall
capillary loops close to the surface. Since sebaceous glands are sparse in this region, the epithelium is subject to drying out if not moistened by the tongue (Orban, 1962).

VASCULAR AND NERVIOUS SUPPLY OF THE ORAL MUCOUS MEMBRANE

A rich blood supply is an important factor in promoting rapid healing of the oral mucosa. The small arteries which enter the lamina propria from the submucosa, divide to form a subepithelial network in the papillae. Here the veins which also originate from the capillary network, follow the course of the arteries. In addition, a rich lymphatic network accompanies these vessels, (See Figure 1).

Jenkins (1961), considers the tip of the tongue, the lips, gingival and hard palate as among the most sensitive structures in the body. Dixon (1963), found these areas contain dense nerve plexuses. In fact, in all parts of the oral mucosa nerve endings radiate out into the lamina propria from submucosae networks. All types of nerve terminals exist in the papillae; some of these enter the epithelium where they terminate between the epithelial cells as free nerve endings (Orban, 1962), (See Figure 1).
GLANDS OF THE ORAL MUCOUS MEMBRANE

A. Salivary Glands.

Apart from the outpourings from three pairs of major salivary glands located outside the mucosal wall, the oral mucous membrane is constantly moistened by the secretions from numerous accessory salivary glands. These small glands are situated chiefly in the submucosa and open with numerous, narrow ducts on to the surface of the mucous membrane (Sicher, 1960), (See Figure 1). Lockhart et al (1959), estimate that if the surface epithelium is scraped away from an average-sized soft palate of an adult, approximately 100 duct openings can be seen.

These minor salivary glands can be classified according to their site (See Table 2). The "labial" and "buccal" glands have the free terminals of their secretory ducts directed to the inner surface of the lips and cheeks respectively. The secretions are mucous, except for the serous secretions of the posterior part of the anterior lingual gland and the glands of von Ebner; the vestibular glands have a "mixed" secretion.
TABLE II.

CLASSIFICATION OF MINOR SALIVARY GLANDS
Adapted from Orban (1962), and Sicher (1960).

<table>
<thead>
<tr>
<th>Clinical Classification</th>
<th>Site</th>
<th>Secretion</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Glands of Vestibule</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Labial—superior</td>
<td>Near inner surface of lips — closely packed — variable size</td>
<td>Mixed</td>
</tr>
<tr>
<td>-inferior</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. Buccal</td>
<td>Continuous with labial glands in check — more numerous posteriorly.</td>
<td>Mixed</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Glands of the floor of the mouth</td>
<td>Fused with anterior extension of major sublingual gland or lie on its superior surface</td>
<td>Pure Mucous.</td>
</tr>
<tr>
<td>-minor sublingual</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-glossopalatine</td>
<td>Isthmus region and a continuation posteriorly of lesser sublingual glands — ascend into glossopalatine fold.</td>
<td>Pure Mucous.</td>
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</tr>
<tr>
<td>2. Glands of the tongue</td>
<td>Embedded in musculature of inferior aspect near apex (glands of Blandin-Nuhn)</td>
<td>Ant.—mucous</td>
</tr>
<tr>
<td>-anterior lingual</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-posterior lingual</td>
<td>von Ebner glands empty into trough of vallate papillae</td>
<td>Post.—mucous cells cap demilunes of serous cells Serous</td>
</tr>
<tr>
<td>-glands at the base of the tongue</td>
<td></td>
<td>Pure mucous.</td>
</tr>
<tr>
<td></td>
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<td></td>
</tr>
<tr>
<td>3. Palatine glands</td>
<td>form an almost compact body in the submucosa of the palate — seldom extend anteriorly past a line drawn from first molar to first molar.</td>
<td>Pure mucous.</td>
</tr>
</tbody>
</table>
B. Sebaceous Glands.

Sebaceous glands are not generally considered to be normal constituents of the oral mucosa. However, Thoma and Goldman (1960), estimate that ectopic sebaceous glands, known as Fordyce's Spots, occur in the mouths of about two-thirds of the adult population. They develop as a result of misplaced anlage of the sebaceous glands of the skin (Orban and Wentz, 1960), and usually become more prominent after puberty.

Fordyce's Spots occur singly or in groups as small, yellowish, slightly elevated spots, usually on the inner surfaces of the lips or on the buccal mucosa opposite the occlusal plane. Histological examination shows these sebaceous glands to be in the submucosa and Chambers (1928), showed by means of wax reconstruction models that many of them have ducts opening to the surface. From these openings a greasy substance, sebum, may exude. Miles (1958), reports that these glands have no known function, but that they could contribute significantly to the oral environment.

PIGMENTATION OF THE ORAL MUCOUS MEMBRANE

"Physiologic", or naturally occurring oral pigmentation may occur in individuals of any racial origin, particularly in Negroes, Southern Europeans, Asiatic races and dark-complexioned persons. All investigative work indicates that melanin is the primary oral mucosal pigment, but Bolden (1960), feels that the evidence for
this is sparse and has been derived from isolated observations of only a few regions of the oral cavity.

According to Dummett (1960), when melanin is present it is found in greatest concentration in the gingivae and, in order of decreasing incidence, in the buccal mucosa, hard palate, tongue, soft palate and floor of the mouth.

The melanin granules appear to be formed in the cytoplasm of the melanocytes, which are dendritic cells situated in the basal cell layer of the epithelium (Volker and Kennedy, 1960). Once formed, the melanin granules are transferred to other types of basal cells (Orban and Wentz, 1962). Many of the biochemical factors regulating the complex process of melanin formation are still to be defined. There is general agreement with the theory that the complex enzyme tyrosinase catalyzes the oxidation of tyrosine to 3-4 dihydroxyphenylalanine. This in turn is oxidized to melanin, (Dummett, 1959; Volker and Kennedy, 1960; Montagna, 1962).

**REGENERATION OF THE ORAL MUCOUS MEMBRANE.**

Physiological regeneration of the oral mucous membrane is governed by the rate of its destruction (Gardner, 1961). In the healthy individual a delicate balance is maintained between desquamation and regeneration of the surface cells. A large number of mature cells are lost from the oral mucosa each day. Gardner (1961), estimates that replacement of the oral mucosa
occurs approximately once monthly or 840 times during a life span of 70 years.

Few studies of mitotic activity in the oral mucosa have been reported. Schour et al (1952), using experimental animals, found that 60% of the mitoses took place in the basal cell layer. Trott and Gorenstein (1963), studied rates of mitotic division of cells of the oral epithelium in experimental animals and reported the following daily percentage of cells dividing:

<table>
<thead>
<tr>
<th>Location</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>In the hard palate</td>
<td>6.76%</td>
</tr>
<tr>
<td>In the buccal mucosa</td>
<td>7.25%</td>
</tr>
<tr>
<td>In the crevicular gingiva</td>
<td>6.21%</td>
</tr>
<tr>
<td>In the attached gingiva</td>
<td>2.27%</td>
</tr>
<tr>
<td>In the epithelial attachment</td>
<td>9.5%</td>
</tr>
</tbody>
</table>

However, a different point of view is presented by Meyer and Medak (1962), who estimate that the buccal epithelium is replaced almost three times more rapidly than the epithelium of the hard palate. According to Trott and Gorenstein (1963), the average mitotic rates in all locations were lowest in the evening and reached a peak in the morning.
KERATINIZATION OF THE ORAL MUCOUS MEMBRANE.

In certain areas of the mouth a layer of keratin, because of its unusual insolubility and toughness, protects the underlying mucous membrane. In a study of four areas of this mucous membrane of adult males, aged from 20 - 30, Miller et al (1951), report that the gingivae exhibit the highest degree of keratinization, followed in decreasing order by the dorsum of the tongue, the cheek, and lastly the ventral surface of the tongue. They did not examine the hard palate which Noyes (1959), and Orban (1962), consider to be highly keratinized.

Gardner (1961), notes that at birth there is no keratin in the oral cavity. Its development is generally interpreted as a form of protective adaptation to function. Robinson and Kitchin (1948), found that keratinization increased in response to artificial stimulation of the gingivae with a toothbrush. However, in my opinion, the theory of teleological development of keratin has not yet been positively proven.

Most writers use the terms "keratinization" and "cornification" synonymously. Robinson (1962), emphasises that cornification applies to the histological development of the stratum corneum, and that keratinization applies to the concomitant complex chemical process. This differentiation has not been confirmed by other writers.

Keratins comprise a large group of fibrous proteins. The keratin which is present in the oral cavity belongs to the group of
"soft" keratins (Rothman, 1954). It desquamates easily and when examined with an electron microscope appears to consist of flattened, elongated cells (4-8 in thickness), whose long axes are arranged parallel to the surface (Rhodin and Reith, 1962; Zelickson, 1963).

The process of keratinization has been studied intensely, yet its mechanism has not yet been fully elucidated. Matolsty (1960), Serri and Montagna (1961), Rhodin and Reith (1962), and Zelickson (1963) refer to keratohyalin granules and tonofibrils, two elements present in epithelium which they consider to be associated with the keratinization process. Rhodin and Reith (1962), postulate a theory of keratinization based on electron microscopic observations. Keratohyalin granules and tonofibrils appear in the deeper epithelial layers, mature in successive layers, and finally associate to form a terminal product which stabilizes as keratin.

A relationship between vitamin A and keratinization of epithelial tissues has been recognised for many years, but still remains to be defined (Bern and Lawrence, 1962). Other factors which influence keratinization are; (a) the action of oestrogen and testosterone (Ziskin, 1941; Ziskin et al, 1946; Papic and Glickman, 1950); (b) the glycogen content of the epithelium (Montagna et al, 1951; Rothman, 1954; Turesky et al, 1957; Klingsberg et al, 1961); (c) age of the subject (Papic and Glickman, 1950).
PROTECTION OF THE ORAL MUCOUS MEMBRANE BY SALIVA.

The oral mucous membrane is constantly exposed to mechanical, chemical and thermal irritants and the action of an environment rich in pathogenic micro-organisms. However, clinical observation indicates that infection of the oral mucous membrane is relatively infrequent and seldom of a serious nature (Bartels, 1961). In fact, Stuart et al. (1935), state that "there is in operation in the oral cavity a complex and remarkably efficient self-protective mechanism". The contribution of saliva to this mechanism can be considered under the following headings:

A. MECHANICAL FACTORS.

(I) Flushing Mechanism. Bibby (1949), reports that suppuration rarely occurs in regions of the mouth accessible to a free flow of saliva.

(II) Mastication. Bartels (1961), believes that the movement of the food bolus around the mouth frees bacterial aggregations adhering to the teeth, and aids the desquamation of the oral mucous membrane.

(III) Currents in Saliva. Bloomfield (cited by Payling Wright, 1956), and Stuart (1935), demonstrated that bacteria rapidly pass into the throat and oesophagus under the action of currents of saliva and mucous combined with the movements of the tongue and cheeks. Theus (cited by Bartels, 1961), considers most oral pathogenic micro-organisms are carried to the throat before they can become established in the soft tissues.
(IV) **Crevicular Fluids.** Krasse and Brill (cited by Bartels, 1961), and Schultz-Haudt (1963), describe a tissue fluid present in the gingival sulcus which exerts a mechanical cleansing effect on the gingival tissues. Its origin and composition have not yet been defined.

(V) **Action of Mucins.** Mucins are a secondary product of mucous membranes and comprise a group of different chemical substances whose most striking property is their high viscosity (Florey, 1962b). According to Knox (1951), Pigman and Tsuiki (1959), and Bartels (1961), salivary mucins appear to protect the mucous membrane against chemical and digestive enzymes and to lubricate the passage of the food bolus.

**B. ANTIBACTERIAL FACTORS.**

(I) Lysozyme, a protein enzyme present in saliva, is capable of lysing certain Gram - bacteria, and preparing other microorganisms for digestion by polymorphonuclear leucocytes, (Burnett and Scherp, 1957; Florey, 1962a; Schultz-Haudt, 1963).

(II) Kanter and Appleton (1940), demonstrated that salivary filtrates inhibited the growth of tubercle bacilli.

(III) Antibacterial substances of microbial origin. According to Burnett and Scherp (1957), much of the antibacterial activity of saliva is apparently due to the activity of the indigenous oral flora. Bacterial antagonism has been shown by oral micro-organisms to corynebacterium diphtheriae (Thompson and Shibuya, 1946), clostridium tetani (Bartels and Blechman, 1959),
certain strains of lactobacilli (Zeldow, 1959), and β-haemolytic streptococci (Bartels et al, 1958).

(IV) Antigenic components of saliva. Results of analyses by Leach et al (1963), suggest that there are seven serum proteins present in saliva. Earlier, Bartels (1961), noted that these proteins, one of which is γ-globulin, may explain some of the bactericidal effects produced by saliva.

(V) Leucocytes. Since one of the primary roles of leucocytes is that of phagocytosis, their presence in saliva is probably important. Their origin in saliva has not been explained. As early as 1940 Lundquist announced that, since the epithelium lining the gingival sulcus is rarely intact, it could be the site of transfer of leucocytes from the subepithelial tissues to the saliva. This theory has been supported by the work of Calonius (1958), Sharry and Krasse (1960) and Klein (1962), who showed a positive correlation between the presence of leucocytes and the presence of teeth. Orban (1962), states that lymphocytes at the base of the gingival sulcus have a protective function, but he does not amplify this statement. After observing a large number of leucocytes on the non-keratinized regions of the oral mucous membrane, Weinmann (1940), concluded that leucocytes and keratinization are complementary in protecting the oral mucous membrane. This observation has been confirmed by Ziskin et al (1941), and Montgomery (1951).
BASIC HISTOPATHOLOGICAL CHANGES OF THE ORAL MUCOUS

MEMBRANE.

The basic response of any living cell to injury is strictly limited. Spouge and Diamond (1963), believe that where damage involves principally nuclear function, a mild stimulus may initiate proliferation of the individual cell or tissue; if the damage is more severe or prolonged, it may bring about cell degeneration and death.

Changes in the oral mucous membrane may involve the epithelium, the underlying connective tissue or both structures. Usually changes affecting only one such component are of short duration for soon the contiguous tissue becomes involved, (Cahn, 1941). Because the oral epithelium is separated from the blood supply of the underlying connective tissue its inflammatory response is limited. As a result it is able to contribute significantly to the histological and clinical picture, (Spouge and Diamond, 1963).

Several basic histopathological changes may be associated with the many ulcerative lesions of the oral mucosa. These are described as follows:-

(1) Hyperkeratosis.

Hyperkeratosis is an hypertrophy of the stratum corneum which is common in the oral cavity, particularly in the buccal mucosa and on the tongue. Thoma and Goldman (1960), consider
that the horny cells fail to desquamate, or are produced so rapidly that they form an excessively thick, hornified layer. Hyperkeratotic changes in the epithelium are generally associated with hyperplasia, but Cahn (1936), states that they may accompany epithelial atrophy.

(2) **Parakeratosis.**

In parakeratosis the stratum corneum is enlarged but imperfectly cornified. This reaction results from rapid keratinization, the cells being produced so quickly that the transformation to mature keratin is incomplete, (Bernier, 1959; Thoma and Goldman, 1961). The surface cells retain their spindle-shaped nuclei and spinous processes and adhere closely to one another. This tends to prevent the process of desquamation taking place.

Parakeratosis is commonly associated with intercellular and intracellular oedema of the prickle cell layer, the function of which appears to be impaired. Cahn et al (1962), showed that parakeratosis is frequently accompanied by an increased glycogen content in the cells. They report that in such lesions there is a positive correlation between the absence of glycogen and the presence of a malignant potential.

(3) **Acanthosis.**

Under certain stimuli the cells of the prickle-cell layer may revert to the basal-cell form and, as a result, produce increased numbers of cells, (Bernier, 1959; Thoma and Goldman, 1960).
Fig. 6.—Parakeratosis of oral epithelium. Note nuclei and irregular cells present in cornified layer, A.

(Bernier, 1943)

Fig. 158
Intracellular oedema of the buccal mucosa, with vesicle formation at the bottom of the picture. Haematoxylin and eosin. × 500.

(Rushton and Cooke, 1959)
This causes a thickening of the entire epithelium with broadening and fusing of the rete pegs. Acanthosis may accompany increased keratin formation, as in leukoplakia, or it may occur alone, as in a non-keratinizing papilloma (Hushton and Cooke, 1959).

(4) **Dyskeratosis.**

Dyskeratosis refers to an abnormal maturation of the epidermal cells (Bernier, 1959; Thoma and Goldman, 1960). It is characterized by disorderly or premature keratinization of the basal cells through their successive layers. Bernier (1959), believes that the cell changes in dyskeratosis are multiple and may exist in many combinations which vary in intensity. Some of the more frequently occurring are; (a) alterations in the size and shape of the cells, (b) presence of abnormal mitoses with respect to mode of cell division and frequency of occurrence, (c) alteration in staining capacity of the cell, (d) presence of abnormal nuclear and cytoplasmic inclusions, (e) changes in the rate at which differentiation occurs, (f) loss of, or alteration in, functional activity. Robinson (1962), emphasises that while there may be suggestions of abnormal mitoses and keratinization in the cells of the basal layer, the basement membrane remains intact.

Bernier (1959), feels that "it is reasonable to assume that when dyskeratosis has occurred without actual invasion, in sufficient time the abnormal cells will break their retaining basal layer and extend into adjacent tissue." Spouge and Diamond (1963), warn that
dyskeratosis indicates a severe or ominous degree of disorientation of the epithelial cells.

(5) **Spongiosis.**

This condition represents an increase in the amount of intercellular fluid in the prickle-cell layer. As the cells are forced apart the intercellular bridges become more prominent. Rushton and Cooke (1959), consider that spongiosis often predisposes to parakeratosis and that polymorphonuclear leucocytes or lymphocytes may filter into the intercellular fluid. This excessive collection of fluid may, because of augmented cellular nutrition, result in acanthosis and later rupture of the intercellular bridges to form vesicles, (Bernier, 1959; Thoma and Goldman, 1961). This loss of intercellular bridges is called "acantholysis".

(6) **Vesicles and Bullae.**

A vesicle is a circumscribed, superficial elevation of the skin or mucous membrane caused by the collection of serum, plasma or blood between the epithelial cells, (Bhaskar, 1961). Rushton and Cooke (1959), state that a vesicle arises in two ways:

(a) Intercellular oedema may be such that the intercellular protoplasmic bridges are parted, resulting in acantholysis.

(b) Epithelial cells may themselves degenerate and the empty, swollen cells form an open, meshed reticulum. This type of degeneration, known as "reticular degeneration", is seen in infections with herpes simplex virus.

While histologically vesicles and bullae are similar, the
latter are usually larger than the former. In the oral cavity, because of attendant trauma, vesicles quickly rupture producing shallow, painful ulcers.

(7) **Pseudoepitheliomatous Hyperplasia.**

This condition is characterized by a marked epithelial hyperplasia and is often confused with dyskeratosis and squamous-cell carcinoma. Outstanding microscopic features of this proliferation are: (a) the absence of dyskeratosis, (b) the normal appearance of the epithelial cells (Bhaskar, 1961), (c) maintenance of cell cohesiveness (Bernier, 1959), (d) the presence of inflammatory cells (Thoma and Goldman, 1960). Thoma and Goldman (1960), find that this condition is frequently accompanied by marked acanthosis and extension of the rete pegs. Bhaskar (1961), reports that pseudoepitheliomatous hyperplasia occurs either without any apparent cause, or in association with a variety of conditions such as fungus infection, chronic epithelial ulcers and bony sequestrae.

(8) **Leukoderma.**

Leukoderma is recognised by Sandstead and Lowe (1953), Burkett (1961), Shafer and Waldron (1961), and Shafer et al (1963), as a clinicopathological entity characterised by an increase in epithelial thickness, intracellular oedema of the spinous cells and broadening and elongation of the rete pegs. Shafer et al (1963), note that the amorphous epithelial surface is non-keratinized and that the oedematous cells appear extremely large and pale.
7. Epithelium of oral mucosa showing acanthosis. A. and column, B.

(Bernier, 1943)

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1. Leucoplasia of tongue. The late lesion with an irregularly thickened layer of keratin, and some individual cell dyskeratosis, and irregular downgrowth of the epithelial ridges. Haematoxylin and eosin. × 100.

(Fushton and Cooke, 1959)
Clinically, leukoderma frequently resembles leukoplakia. Shafer et al (1963), state that nearly all patients with leukoplakia exhibit leukoderma in the adjacent mucosa. Shafer and Waldron (1961), report that a similar, if not identical, histological picture is found in an unusual disease known as "white folded gingivo stomatitis", or "oral white sponge nevus".

(9) Leukoplakia.

"Leukoplakia" is a term frequently used to describe a disorder of the mucous membranes characterized by the presence of abnormal white patches or plaques, which do not conform to the picture of identified "white" lesions. In general, authors consider leukoplakia to be a premalignant lesion, although the estimated frequency of malignant transformation varies from 2.5 per cent to practically 100 per cent (Shafer and Waldron, 1961). Smith (1963) believes that "of all the controversial lesions of the oral cavity there is probably none that has evoked so much dissension and confusion regarding definition, cause and ultimate prognosis" as has leukoplakia.

Kollar et al (1954), declare that there is no precise histopathological entity which is accepted by the majority of pathologists as being "leukoplakia". Sections from clinically diagnosed leukoplakias differ greatly. There may be present hyperplasia, hyperkeratosis, acanthosis, dyskeratosis or squamous-cell carcinoma. The basal cell layer is intact and
the underlying connective tissue exhibits varying degrees of chronic inflammatory cell infiltration (Robinson, 1962). Some writers, (Tiecke and Bernier, 1954; Cheraskin and Langley 1956; Bhaskar 1961; Thoma and Goldman, 1961) require the histological presence of dyskeratosis before making a diagnosis of leukoplakia. In the absence of dyskeratosis these authors refer to the disorder as focal keratosis, hyperkeratosis, hyperkeratosis simplex, pachyderma oris or nonspecific keratosis. At the same time, others such as Stones (1954), Rushton and Cooke (1959), Zegarelli et al (1959), do not agree that dyskeratosis is necessary for the histological diagnosis of leukoplakia. Rushton and Cooke (1959) describe an advanced leukoplakia lesion as one which may exhibit (a) marked acanthosis, (b) frequent mitotic figures, (c) many vacuolated prickle cells (d) disruption of the basal cell layer, and, (e) degeneration of elastic fibres in the underlying connective tissue. Hahn et al (1961), conclude that leukoplakia is a dynamic process which begins in the basal layer of the epithelium and involves changes in the mitochondria, the formation of tonofibrils and the keratinization process.

Because of the disparity between clinical and histological findings, it has been suggested by Kollar et al (1954), Russ (1957), Robinson (1962), and Smith (1963), that the term "leukoplakia" be discarded as a diagnostic one. As early as 1954 Kollar et al
presented the following simple classification of maturation disorders of the oral mucous membrane:

1. Hyperplasia.

Hyperkeratosis

( 2. Hyperkeratosis.

simplex.  ( 3. Hyperkeratosis with inflammation.

Hyperkeratosis

( 4. Hyperkeratosis, dyskeratosis and inflammation.

complex  ( 5. Intraepithelial oedema.

6. Carcinoma.

This classification eliminates discussion of the histology of leukoplakia and avoids argument as to whether the leukoplakic lesion is benign, cancerous or precancerous.

It has been observed that leukoplakia is a disease of middle and advanced age and is more common in men than in women. It is now conceded by most investigators that the aetiology is a varied one. The initiation of the condition is believed to depend not only upon extrinsic local factors but also upon intrinsic predisposing factors (Shafer et al., 1963). Extrinsic factors most commonly suggested are tobacco, alcohol and local physical irritants, while the systemic predisposing factors include vitamin deficiency, endocrine dysfunction and a past history of syphilis. Burket (1961), notes that in about half the cases of leukoplakia neither extrinsic nor intrinsic factors can be discovered.
HEALING OF THE ORAL MUCOUS MEMBRANE.

The oral mucosa, like the epidermis of the skin, possesses considerable powers of regeneration (Muir, 1958; Le Gros Clark, 1958). Morris (1949), points out that epithelium carries out its function as a protective layer by quickly covering any wound or break in its continuity.

Wound healing is a composite, biological phenomenon which Arey (1937), divides into three periods or phases. The first phase is a provisional closure of the wound, in which inflammatory defence mechanisms play an important role. The second phase is characterized by death and removal of injured cells and absorption of exudates; the products of cell destruction appear to exert a reparative stimulative effect on the tissues. The final period of constructive repair involves epithelial and connective tissue proliferation. Fayling Wright (1956), considers that the phases of wound healing cannot be clearly demarcated since the cellular reactions exhibited in each merge gradually into one another.

1. PRIMARY REPAIR OF THE ORAL MUCOUS MEMBRANE.

"Primary Repair", or "healing by first intention", usually takes place following surgical incisions in the oral cavity where the clean edges of a wound are held in close approximation by sutures (Muir, 1958; Shafer et al, 1963). Within a few minutes of injury, the wound edges are glued together by a thin layer of coagulum in which connective tissue cells and new capillary buds proliferate. These migrate into the injured tissues while the epithelium proliferates to re-establish an unbroken surface (Muir, 1958; Florey and Jennings, 1962; Shafer et al, 1963).
Essentially the same stages follow vesiculation of the epithelium: the impermeable, keratinized layer is raised by fluid accumulation in the underlying tissues. It acts as a protective cover under which the deeper, more moist layers rapidly proliferate to restore the original stratification (Arey, 1937; Florey and Jennings, 1962).

2. SECONDARY REPAIR OF THE ORAL MUCOUS MEMBRANE.

"Secondary Repair", or "healing by second intention", is the principal means by which ulcerations or other wounds of the oral mucous membrane are healed.

Following the initial tissue injury the wound is flooded with blood released from the opened capillaries, and a yellowish serous fluid exudes from the surface. The latter contains a considerable amount of protein which clots on the surface, thereby covering the wound with a transparent, fibrin layer containing leucocytes (Florey, 1962). Toto and Abati (1963), consider that the clot and polymorphonuclear leucocyte infiltration are a temporary defence which permits proliferation of connective tissue cells following injury.

The defect caused by tissue destruction is slowly filled with a mass of newly-formed, organizing tissue which grows upwards from its base (Payling Wright, 1956). Because of its granular appearance this healing tissue is referred to as "granulation tissue".

Cellular proliferation commences around the wound periphery when fibroblasts and endothelial cells grow into the clot along fibrin strands (Shafer et al, 1963). Inflammatory cells, initially
polymorphonuclear leucocytes, and later lymphocytes and monocytes, migrate into the granulation tissue from adjacent vessels to phagocytose tissue debris and micro-organisms.

Within the next few days the surface of the wound assumes a reddish, finely granular appearance. Each of these "granulations" consists of a core of newly-formed blood vessels surrounded by young connective tissue (Arey, 1937). The granulation tissue increases in thickness until the entire tissue defect is filled by new tissue.

During this marked cellular activity in the connective tissue layers, the epithelial cells at the intact edges of the wound proliferate and advance until the surface is whole again. Rothman (1954), believes that this thin sheet of epithelial cells is derived from the lower strata of the prickle-cell layer and that, once the defect is covered, the new epithelium becomes stratified, and the cells in contact with the connective tissue assume the characteristics of basal cells.

As the granulation tissue matures, the collagen bundles condense and the tissue becomes more fibrous. Cellular and vascular contents decrease. Wounds of the oral cavity rarely heal with scarring (Bhaskar, 1961).

3. FACTORS AFFECTING WOUND HEALING.

Many conditions exist which serve to vary the mode of the healing process in the mouth and the time for its completion. They include (a) location of the wound relative to a rich vascular supply; (b) the degree of infection; (c) age of the patient; (d) concentration of vitamin C and protein; (e) action of adrenal cortical steroids.
CHAPTER 2.

THE ULCERATIVE LESION.

Description.

Diagnosis.
THE ULCERATIVE LESION

Definition.

The word "ulcer" is derived from the Latin "ulcus", meaning "a sore". The lesion is defined by Woodruff (1954), as "a breach of continuity of a surface resulting from death of superficial tissue." Fleming (1958), prefers to think of it as "an open sore other than a wound; a loss of substance on a cutaneous or mucous surface, causing gradual disintegration and necrosis of the tissues."

Whatever the accepted definition, an ulcer can be produced by any method of surface destruction. It is necessary to distinguish between ulceration and gangrene, in which a mass of tissue dies "en bloc" (Fish, 1948).

Phases of the Ulcerative Lesion.

Woodruff (1948), and Reade (1961), describe three stages in the course of an ulcer of the oral mucosa:

1. Extension. During extension the floor of the lesion is covered with exudate and sloughing tissue. The base is firm and the edges are well defined. There is a heavy leucocytic infiltration beneath the denuded surface where microorganisms are usually in evidence. At this stage discharge from the surface may be purulent, serous or sanguinous (See Figure 2).

2. In the transitional stage, repair processes have already commenced. The base of the ulcer becomes more loosely attached
to the underlying structures. The sloughing is separated off as small red granulations protrude from the floor of the ulcer and the discharge becomes more serous. Some chronic ulcers may remain in this stage for months or even years.

3. As healing progresses, the granulation tissue growing up from the floor of the lesion reaches the level of the surrounding tissue. Thin epithelial sheets extend out from the intact edges of the ulcer to cover the new connective tissue and the inflammatory reaction of the surrounding tissues subsides. (See Figure 2.)
Diagram of spreading ulcer (above) and healing ulcer (below).

**ULCER**

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**Fig 2.** (Blakiston's dictionary, 1956)

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**a.**

**TUBERCULOUS ULCER**—undermined edges and relatively deep base

**b.**

**TERTIARY SYPHILITIC ULCER**—punched out and deep

**c.**

**EPITHELiomATOUS ULCER**—raised rolled everted edges and a raised base

**d.**

**RODENT ULCER**—raised beaded edge and a raised base

**e.**

**NON SPECIFIC ULCER**—raised edge and a base of variable depth

**f.**

**HEALING ULCER**—shelving edges and shallow base

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**Fig. 1.**—Diagrams of typical cross-sectional shapes of ulcers.

**Fig 3.** (Beade, 1961)
Female, Aged 58 years.

HEALING ULCER OF THE PALATE.

An excision biopsy was performed for a fibroma of the posterior palate and when seen one week later the biopsy wound was healing satisfactorily. The lesion was not painful and examination of the surface showed healthy granulations protruding from the underlying connective tissue. The patient was advised to use frequent warm saline mouth rinses.

Healing took place uneventfully in approximately three weeks.
DIAGNOSIS OF ULCERATIVE LESIONS OF THE ORAL MUCOUS MEMBRANE.

The patient presenting with ulceration of the oral mucosa poses a diagnostic problem which may confuse the most experienced clinician. Textbooks often give precise descriptions of ulcers with the implication that their aetiology may be determined from a visual examination. However, investigators such as Fleming (1958), consider that oral ulcerations assume such variable patterns that too much faith must not be attached to their "naked-eye" appearance in trying to determine their aetiology. A diagnostician should not hesitate to refer a patient for further consultation if he is unable to arrive at a definite diagnosis. Thoma and Robinson (1960), consider that "to refer a case for consultation is often evidence of good judgment for which the referring dentist gains respect."

1. **The History.**

The history of the ulceration must be carefully evaluated for each patient as a requisite to any differential diagnosis. It should include a detailed description of the present illness, relevant medical and dental histories, and dietary and social histories, (Reade, 1961). Fleming (1958), points out that co-operation of the dentist with the patient's physician is essential if the patient is in poor general health. Unrecognised
or neglected systemic conditions will unduly prolong the necessity for local treatment or even invite complete failure of the treatment.

2. **The Clinical Examination.**

Clinicians vary with regard to the detail necessary to identify ulcer types. For example, Woodruff (1954), describes an ulcer with respect to its edge, floor and base. Reade (1961), requiring considerably more detail, refers to its size, shape, site, discharge, foetor oris, associated lesions and lymphadenopathy and whether it occurs singly or in association with other ulcers.

The Clinical Appearance of the Ulcer.

(a) **SITE.** Some oral ulcers have a typical distribution. For instance, those which occur following the breakdown of vesicles of Herpes zoster are situated on the path of a sensory nerve and are therefore frequently unilateral; acute necrotising gingivitis, in the early stages of the condition, is confined exclusively to the interdental papillae; tuberculous ulcers are most commonly seen on the tongue, and recurrent herpetic ulcerations on the lower lip.

(b) **The FLOOR of an ulcer may consist of granulation, fibrous or connective tissue, of muscle or of bone.** According to Fish (1948), the floor of a simple ulcer is approximately level with the surface. In a tuberculous or tertiary syphilitic ulcer the floor is usually depressed below the level of the surrounding tissue. The nature
of the floor of the ulcer may be also significant, (Reade, 1961). In acute necrotizing ulcerative gingivitis and diphtheria it is membranous, in a tuberculous ulcer it consists of exudate-covered granulations, and in a healing ulcer it is of healthy pink granulation tissue, (See Figure 3).

(d) The EDGE of an ulcer may give an indication of the local and general response of the body to the lesion. Oral aphthae have typically irregular, hyperaemic, elevated edges; tuberculous ulcers have irregular, undermined edges; malignant ulcers have hard, everted, elevated edges and rodent ulcers have elevated, beaded edges.

(e) The BASE of an ulcer is the zone of tissue immediately underlying its floor and is examined by palpation. Most ulcers are freely movable over their base. In the case of malignant ulcers the nature of the base has particular significance for it is often indurated and fused with the underlying tissues, (Reade, 1961).

(f) EXUDATE from an oral ulcer is rarely significant because the ulcer is continually bathed in saliva. A simple ulcer may exhibit thick, creamy pus due to staphylococcal infection. Fish (1948), considers that a syphilitic ulcer has a rubbery discharge and a malignant ulcer one that is often foul-smelling.

(g) ASSOCIATED LYMPHADENOPATHY is significant of malignant ulcers.
(h) FOETOR ORIS may be marked in cases of advanced malignancy, acute necrotizing ulcerative gingivitis, advanced pemphigus and Erythema multiforme.

(i) Reade (1961) emphasises that the NUMBER of ulcers present in the mouth is of diagnostic importance. Neoplastic, traumatic and tuberculous ulcers usually occur singly, while primary herpetic ulcers are usually multiple in number.

(j) Other types of lesions are frequently present with oral ulcers. Their absence at the time of clinical examination does not negate the possibility of oral manifestations occurring later, (Reade, 1961).

3. **Clinical Symptoms.**

Clinical symptoms may be systemic and local.

(a) **Systemic Symptoms.**

If present, systemic symptoms vary in presentation and in degree of severity. They include headache, nausea, pyrexia, tachycardia, anorexia and tachypnoea. Reade (1961), lists the following:

i. Systemic symptoms rarely occur in cases of oral aphthae, minor traumatic ulcers, early neoplastic and recurrent herpetic ulcers.

ii. Mild systemic symptoms are associated with acute necrotizing ulcerative gingivitis, Erythema multiforme, Varicella, Herpes zoster and ulcerative lichen planus.
iii. Severe systemic symptoms are characteristic of primary herpetic stomatitis, advanced malignancies, blood dyscrasias, pemphigus, Stevens-Johnson syndrome, Variola.

(b) Local Symptoms, may vary widely:

I. Initiation:

Reade (1961), describes the commencement of oral ulcers according to:

i. Method of onset

- sudden (acute necrotizing ulcerative gingivitis, primary herpetic stomatitis)
- insidious (neoplastic, syphilitic and tuberculous ulcers)

ii. Initial appearance:

- ulcer (acute necrotizing ulcerative gingivitis and others)
- vesicle (Herpes zoster, herpetic stomatitis, pemphigus and others)
- nodule (some oral neoplasms, oral aphthae and tuberculous ulcers).

II. Causes of Initiation:

These may be traumatic, (decubital ulcer or thermal burn), infective, (Herpes zoster), psychosomatic (ulcerative lichen planus), or develop because of lowered tissue resistance
(acute necrotizing ulcerative gingivitis). In some instances of oral ulceration the initiating factor is unknown (lichen planus, pemphigus, oral aphthae and neoplasms).

III. Duration of the ulcer may have diagnostic significance. "Oral ulcers persisting for more than three weeks should be strongly suspect and if healing is not evident a biopsy should be performed", (Reade, 1961). Herpetic ulcers, aphthous ulcers and similar lesions are of short duration, while neoplastic, tuberculous and tertiary syphilitic ulcers are of long duration. The period between episodes is usually greater in recurrent herpetic ulcerations than in recurrent aphthous ulcerations. Chronological association with other factors may be important. For example, seasonal changes may be associated with the appearance of acute necrotizing ulcerative gingivitis; menstrual periods are sometimes connected with recurrent aphthous ulceration.

IV. Pain from oral ulceration can be acute (oral aphthae), moderate (Erythema multiforme), or absent (primary syphilis).

V. Relief of pain is usually achieved with drugs administered after a diagnosis has been made. In cases of traumatic ulceration removal of irritants may produce rapid healing; this may be important in distinguishing between neoplastic and traumatic ulceration.
4. **Special Laboratory Investigations.**

There are several laboratory procedures which may be used to establish or confirm diagnosis of oral ulceration. Bernier (1959), points out that indication for their use depends on interpretations gained from many factors associated with the patient's condition:—

(a) Haematological examinations are of value in determining the systemic response of the patient to oral infection and in eliminating any blood disorder as the cause of the oral ulceration.

i. **Full Blood Count.** Oral ulceration may be associated with deviations from the normal red and white blood cell counts (Leukaemias, anaemias, leukopenia, pancytopenia).

ii. **Differential Blood Count.** Numerical variation in the leucocyte count may be of significance in diagnosing oral ulceration due to agranulocytosis, neutropenia and leukaemia.

iii. **The Colour Index** is a measure of the amount of haemoglobin carried by the individual erythrocytes and is a valuable aid in classification and diagnosis of the various anaemias.

Other routine haematological estimations such as platelet count, prothrombin time, capillary resistance, bleeding and coagulation times are usually not required for the diagnosis of oral ulceration.
(b) Urine Analysis. A routine urine analysis includes the macroscopic appearance of the specimen, the specific gravity, the microscopic examination of sediment for casts, cells or organisms, and a qualitative test for sugar and albumin. Such an analysis may be important when oral ulceration is associated with diabetes mellitus.

(c) Bacteriological Examination. Diagnosis of oral ulceration in syphilis, tuberculosis, diphtheria, histoplasmosis, blastomycosis and other infections depends on the microbiological identification of the specific organism. The sensitivity of the organism to chemotherapeutic agents can readily be ascertained by culture methods. Fleming (1958), stresses that bacteriological investigation should be made before any treatment has commenced.

(d) Serological Tests. The Kahn Flocculation and Wassermann Reaction tests are important for the diagnosis of tuberculosis and syphilis respectively.

(e) A Dietary Analysis frequently reveals that a patient with oral ulceration has a poor diet. Correction of a vitamin or protein deficiency aids the effective treatment of many oral ulcerations, particularly recurrent aphthous ulcers and those due to the herpes simplex virus.

(f) Allergy Tests. If allergy is suspected as the cause of oral ulceration, enquiry may lead to the identification of a particular
allergen; in cases where the allergen is not apparent skin tests should be done by an allergist.

(g) Biopsy and pathological examination of any non-healing oral ulcer should be done immediately. It is impossible to separate squamous-cell carcinoma, basal-cell carcinoma, and some chronic inflammatory conditions without a biopsy. Identification of various types of granulomatous lesions, such as those in tuberculosis and certain fungus infections is impossible without the aid of a histological examination of tissue.
CHAPTER 3.

MECHANICAL, PHYSICAL AND CHEMICAL INJURIES

A. Mechanical.

B. Burns
1. Thermal
2. Chemical
3. Electrical
4. Radiation.
A. TYPES OF TRAUMATIC ULCERS.

1. THE SIMPLE TRAUMATIC ULCER.

The commonest ulcerative lesion of the oral mucous membrane is the simple traumatic ulcer, which is usually due to some form of mild irritation. Simple injuries which may cause oral ulceration include accidental biting or chewing of the lip, tongue or cheeks during mastication; abrasions from rough foods such as toast, fruit stones, fishbones and bone chips; injudicious use of the toothbrush, toothpick or dental floss; roughened surfaces of teeth or margins of restorations; ill-fitting crowns, bridges, orthodontic and prosthetic appliances and injuries from dental instruments during routine dental procedures (Hirschfeld, 1933; Bernier, 1959; Kerr and Ash, 1960; Ship, 1963). Oral ulceration in children frequently follows trauma from foreign bodies placed in the mouth for investigation (Jacobs, 1956).

There is no uniformity of the type of lesion produced by these various agents and for a correct diagnosis to be made, careful observation and a complete history are sometimes necessary. The simple ulcer is usually localized and irregular in outline; the colour of the adjacent tissue is normal and the peripheral zone of the ulcer is rarely undermined. The base is usually reddish or reddish-yellow in colour and it is often covered by a moist fibrinous exudate. Beneath this covering are dilated blood vessels, usually seen in association with a lymphocytic and plasma cell infiltration (Bernier, 1959; Ship, 1963). Fibroblastic activity is sometimes prominent and macrophages may be present in moderate numbers.
Once the cause of irritation has been removed these simple ulcers usually heal rapidly without scar formation. Healing of the oral mucosa is primarily established by formation of granulation tissue. However, delayed healing of simple traumatic episodes in the mouth may be the initial manifestation of some systemic disease and should be investigated. For example, the patient with uncontrolled diabetes mellitus, a blood dyscrasia, or a malignancy will frequently exhibit a disturbed healing process. Sometimes a patient may not be aware of a lesion in his mouth, such as a slowly-growing tumour, until painful surface ulceration requires him to seek dental or medical advice.

2. **The Decubital Ulcer**
   (Acute Denture Sore, Pressure Ulcer)

The word "Decubitus" is described by Prinz and Greenbaum (1933), as originally signifying "a bedsore". This meaning has been extended to describe certain types of ulceration of the oral mucosa. Bernier (1949), refers to a decubital ulcer as "a contact manifestation resulting from pressure exerted in a confined area over a long period of time." These ulcers are produced by acute or chronic irritation, usually from ill-fitting dentures, sharp teeth or certain habits such as biting the lips and cheeks and sucking loose areas of tissue into spaces between the teeth.

The decubital ulcer has a well-defined border and corresponds to the area over which pressure has been applied. When such an ulcer is associated with over-extended denture flanges it is frequently accompanied by fissuring in the area of contact (Orban and Wentz, 1960).
Female, Aged 40 years.

**TRAUMATIC ULCER FROM MECHANICAL INJURY.**

The patient complained of an extremely painful ulcer on the upper anterior alveolar ridge which had been present for six days. It was aggravated when the upper vulcanite denture was inserted.

The patient was advised not to wear the denture for one week and to apply tannic acid and glycerine to the lesion. At the end of this time the ulcer had completely healed.

The denture was found to be most unsatisfactory and the patient was strongly advised to have a new acrylic denture constructed.
Cassie (1957), considers that decubital ulcers most frequently occur on the lateral border of the tongue. They are usually frank, painful ulcerations of various sizes and characterized by irregular erythematous margins and oedema. Severe decubital ulcers may be associated with regional lymphadenopathy and usually require several weeks for complete healing. Goadby (1931), Shira (1957), Bernier (1959), Colby et al (1961), describe chronic decubital ulcers which so resemble malignant lesions that histological examination is essential to differentiate between them.

Histological examination of a decubital ulcer shows an abrupt termination of the normal epithelium at the edge of the ulcer and a diffuse infiltration of the submucosa and underlying muscular tissue with chronic inflammatory cells. Bernier (1959), notes that dyskeratosis may be present. Goadby (1931), and Woodruff (1954), emphasise that this type of chronic ulcer may degenerate into a malignant form.

3. THE TRAUMATIC ULCER ASSOCIATED WITH THE USE OF LOCAL ANAESTHESIA.

Some patients may inadvertently bite, or by other means injure, anaesthetized soft tissues of the mouth producing localised or extensive ulcers (Shira, 1957). Such effects are frequently seen in young children who may severely traumatise the lower lip following an inferior dental nerve block injection. Marked oedema results and numerous small ulcers may coalesce to form a single large ulcer.
Kerr and Ash (1960), note that, when this type of ulcer forms on the vermillion border of the lip, tissue fluid exuding from the area produces a surface crust. Such an ulcer may form rapidly and alarm the patient and the parent. To avoid this discomfort the patient should be warned to exercise care until the effects of the anaesthetic have subsided.

If a local anaesthetic solution is injected into dense connective tissue under high pressure, tissue necrosis and ulceration may occur (Orban and Wentz, 1960). The adrenalin present in the solution, by producing protracted local ischaemia, further stimulates ulcer formation. These ulcers at the site of injection are localized, shallow and irregular in shape.

4. **BEDNAR'S APHTHAE.**
(Pterygoid ulcers, Pterygoid erosions)

The term "Bednar's aphthae" refers to an unusual clinical condition seen in young infants in which two symmetrical ulcers develop on either side of the median raphe on the posterior third of the hard palate. Such ulcers appear as small, round, sharply contoured yellow or grey lesions. Jacobs (1956), notes that within two to three days they may involve the midline of the palate and Miller (1957), states that occasionally they extend to the pharynx and larynx and are accompanied by general sepsis.

Prinz and Greenbaum (1939), consider that the term "aphthae" is a misnomer since the ulcers are neither histologically nor
clinically related to true aphthae. However, the early appearance of the lesion of both Bednar's aphthae and of true aphthous ulcers is such that it seems possible that they may sometimes be confused.

5. **REGA-FEDÉ'S DISEASE.**

Rega-Fedé's syndrome is a rare condition in young children in which lingual and sublingual traumatic ulcers develop in association with the erupting lower anterior deciduous teeth.

Jacobs (1956), describes its occurrence in a week-old infant born with erupted lower central incisor teeth. The instinctive sucking process produced an ulcer of the lingual frenum and tip of the tongue. Swallow (1963), reports a persistent lingual ulcer in a two-month old infant whose lower right central incisor erupted a few days after birth.

Extraction of the offending tooth or teeth is usually followed by rapid healing. However, Jacobs (1956), points out that destruction of the tip of the tongue may be permanent.

In a five-year old athetotic girl examined by Swallow (1963), anterior tongue thrusting produced a penetrating ulcer of the ventral surface of the tongue. The upper and lower central incisors had been shed and the ulcer coincided with the mesial incisal angle of a deciduous lateral incisor. The ulcer healed following the insertion of a cast silver splint which covered all the lower teeth and included a smooth connecting bar in the place of the shed lower anterior teeth.
6. ULcers ASSOciATED WITh TOOTHBRUSH INJURIES.

Traumatic ulcers caused by toothbrush injuries are common, especially when a hard type of brush is used incorrectly. Erosions and ulcerations may develop on the gingival and alveolar mucosa (Orban and Wentz, 1960). Kerr and Ash (1960), consider that the most severe injuries occur at the crest of the gingival contour over the roots of the teeth. Hirschfeld (1933), reports five cases of toothbrush-inflicted wounds which subsequently became secondarily infected and ulcerated. These wounds include gingival punctures, gingival lacerations, abrasions and blistering. If the trauma is not repeated this type of ulcer should disappear within a week.

7. THE "COTTONWOOL ROLL" ULCER.

These ulcers are sometimes observed after prolonged contact of the oral mucosa by cottonwool rolls during routine dental procedures. They usually occur at the junction of the attached and alveolar gingivae. Abrupt removal of the cottonwool roll may tear or bruise the surface epithelium producing a denuded area which may rapidly become secondarily infected (Cheraskin and Langley, 1958). Scher (1963), reports four cases of cottonwool roll ulcers in young women. In each case vesiculation at the site of contact of the roll with the epithelium occurred the following day. Desquamation, inflammation and ulceration followed. Scher (1963), considers that the starch content of the surface of a cottonwool roll forms approximately 0.7 - 0.8% of the total weight. This starch takes up
sufficient moisture to become adhesive to the epithelium (Prinz and Greenbaum, 1939; Scher, 1963).

TREATMENT OF TRAUMATIC ULCERS OF THE MOUTH.

Following removal of the trauma, these ulcers heal spontaneously within a few days. Orban and Wentz (1960), note that single lesions do not enlarge and heal without scar formation. If the ulcer is painful, the patient should be advised to avoid hot, spicy foods; to use a mild alkaline mouthwash, such as sodium bicarbonate; to apply antiseptic agents to avoid secondary infection and, if necessary, to suck anaesthetic lozenges.

Investigators agree that, if a traumatic ulcer does not show evidence of healing at the end of a week, a biopsy should be made to eliminate the possibility of malignancy.
B. TYPES OF BURNS OF THE ORAL MUCOSA

Since the lesion produced by a burn invariably involves surface tissue destruction, it satisfies the definition of an ulcer. Schultz and Vazirani (1961), point out that burns of the oral cavity are identical to burns elsewhere in the body, with the exception of sunburn. Because the mouth is relatively inaccessible, it rarely sustains severe or extensive burns.

The clinical course of a burn anywhere in the body is influenced by the type of thermal agent involved, the depth of the burn and the age and physical state of the patient (Clarkson, 1963).

The classification of burns as of first, second or third degree applies in the mouth as for the rest of the body: A first degree burn exhibits erythema and oedema and may not produce surface tissue destruction; a second degree burn produces vesiculation and development of bullae, both of which usually degenerate into ulcers; third degree burns vary in colour from grey to black depending on the amount of ischaemia present, extravasation of blood and carbonization of the tissues (Lever, 1953).

1. ULCERATION OF THE ORAL MUCOUS MEMBRANE ASSOCIATED WITH THERMAL INJURY.

The delicate oral mucous membrane is probably subject to greater variations in temperature than any other tissue in the body with the exception of the skin. Schultz and Vazirani (1961), divide thermal burns of the mouth into those caused by extremes of heat and cold on
either side of the body temperature.

(a) **High Temperature Burns.**

Mild burns of the oral cavity from the accidental ingestion of hot foods or beverages are common. Kerr and Ash (1960), point out that, if the hot food is fluid, the burn usually occurs on the anterior third of the tongue and palate. If the food is semi-solid and adheres to the mucosa, the burned area is more localized and the burn more intense. Hot thermal burns may be accidentally caused in smoking or in the dental surgery by hot instruments, wax or impression material. Colby et al. (1961), report a severe burn on the labial mucosa from overheated hydrocolloid impression material. Within 24 hours the coagulated mucous membrane had separated off, producing a large ulcer with a floor of exposed bone. Shira et al. (1959), describe a prolonged case of multiple ulceration of the tongue, deliberately produced by the patient with the burning end of cigarettes. Burket (1960), refers to a symmetrically shaped burn of the hard palate associated with eating "pizza" pies, the hot cheese adhering to the roof of the mouth.

Superficial thermal burns of the oral mucosa produce a vesicle which ruptures to form a raw painful ulcer. There is usually marked inflammation in the surrounding tissues. An ulcer following severe tissue destruction, such as in the careless use of electrocautery, forms scar tissue requiring several weeks to heal. Serious burns, particularly those which involve the skin of the face, may produce deformity and impair jaw movement (Thoma and Goldman, 1961).
Male, Aged 27 years.

TRAUMATICO ULCER FROM PHYSICAL INJURY

(THERMAL BURN)

Following removal of a retained root fragment from the upper left premolar region the patient was advised not to insert his denture.

When seen two days later for the removal of sutures two painful, symmetrical ulcers were seen on the hard palate. They were serpiginous in outline and covered by a yellowish-white necrotic membrane. The patient remembered burning his mouth with hot soup the day following surgery. An aphthous-like ulcer was also present in the upper left buccal sulcus adjacent to the surgery site.

Tannic acid and glycerine was applied to the lesions which completely healed in one week.
(b) **Low Temperature Burns.**

Extremely low temperatures may burn the oral mucosa producing ulceration. This may be caused by the use of refrigerant anaesthesia, such as ethyl chloride, by "dry ice", or by contact with a cold metallic instrument (Bernier, 1959; Schultz and Vazirani, 1960; Burket, 1961).

The lesion produced by excessive cold commences as an irregular white plaque, the raised surface of which is soon lost to reveal a red ulcerated base. According to Bernier (1959), the lesion is usually accompanied by extreme pain and necrosis of tissue.

2. **Ulceration of the Oral Mucous Membrane Associated with Chemical Burns.**

The accidental ingestion or entry into the mouth of strong acids and alkalis produces typical chemical burn. These are frequently associated with ulceration of the pharynx and stomach. Bernier (1959), criticises the tendency of many dentists and physicians to indiscriminately use medicaments for the treatment of minor soft tissue abnormalities of the oral cavity. Thoma and Goldman (1960), state that although saliva will dilute the irritating agent, it also may spread it to all regions of the mouth.

Contact of the injurious agent with the oral mucosa initiates an inflammatory reaction. The mucosa becomes swollen and painful and is characterized by epithelial destruction. A large number of white blood cells collects below the burned surface and separates the
necrotic tissue from the deeper viable tissues (Orban and Wentz, 1961). Sloughing of the necrotic tissue is followed by the formation of an ulcer which is usually covered by a fibrinous exudate.

(a) Aspirin Burn.

The placing of aspirin on the oral mucosa to relieve toothache is a common and unwarranted practice. The corrosive action of the free acetylsalicylic acid may produce a coagulation necrosis with separation and sloughing of the epithelium (Cheraskin and Langley, 1956; Orban and Wentz, 1961; Shafer et al, 1963).

The typical appearance of the lesion is that of an irregular white patch which cannot be wiped off. If traumatised, this lesion may become secondarily infected and result in ulceration. Healing usually takes place within about seven days. If the chemical irritation is repeated, healing is delayed and when it finally does occur, it is with scar formation.

(b) Sodium Perborate.

This chemical has been widely used as a mouthwash, particularly in the treatment of acute necrotizing gingivitis. Schroff (1938), Glickman and Bibby (1944), Bernier (1959), and Thoma and Goldman (1960) advocate care in its use since repeated applications produce various degrees of inflammation, oedema and ulceration. Ulceration of the mucosa at the site of contact may be deep, painful and accompanied by severe sloughing. The work of Glickman and Bibby (1944), confirmed previous observations that sodium perborate has no therapeutic value in the mouth.
Male, Aged 38 years.

**TRAUMATIC ULCER FROM CHEMICAL INJURY**

*(ASPIRIN BURN)*

Acetylsalicylic acid exerts a coagulative action when it contacts the oral mucous membrane.

The patient had experienced toothache from a carious lower left second molar and in an attempt to alleviate the pain had placed an "aspirin" tablet in the lower left buccal sulcus.

When seen approximately 18 hours later a painful, desquamating lesion was present. The necrotic coagulated epithelium subsequently sloughed and rapid healing took place.
(c) Phenol. (Carbolic Acid).

 Phenol is a strong protoplasmic poison used in dentistry as a cavity sterilizer and cauterising agent. Since the introduction of newer and safer preparations its use is becoming obsolete.

 If Phenol accidentally contacts the soft tissues of the mouth, severe tissue destruction may occur. Burket (1961), states that pain is not severe until sloughing of the necrosed surface tissue takes place.

 Goulding (1960), points out that systemic absorption of phenol may occur by way of the oral mucosa, and repeated application may result in a general toxaemia.

 (d) Chromic Acid.

 Chromic acid is a powerful oxidising agent used in dentistry chiefly for the treatment of inflammatory gingival conditions. Sugarman (1952), reports an instance of prolonged use of chromic acid for the treatment of acute necrotizing gingivitis producing acute ulceration in the buccal and lingual folds. Accepted Dental Remedies (1963), contraindicates the use of chromic acid because of the availability of more satisfactory drugs.

 (e) Other Chemicals.

 Irritation from chemicals such as trichloracetic acid, lye and volatile oils, such as oil of cloves, may produce ulceration of the oral mucous membrane. Colby et al (1961), has observed gingival tissue coagulation caused by a leak of pyrazone from beneath the rubber dam during a bleaching procedure.
3. **ULCERATION OF THE ORAL MUCOUS MEMBRANE ASSOCIATED WITH ELECTRICAL INJURY.**

(a) *Galvanism.*

Galvanism is a condition in which oral lesions are produced by a flow of electric current in the mouth. The presence of dissimilar metals establishes a galvanic cell with saliva or bone fluid acting as the electrolyte. The two metals may be quite separate, or in different parts of a heterogeneous single restoration, such as an amalgam filling or improperly heat-treated gold bridge (Schriefer and Diamond, 1952; Farrell, 1958; Inovay and Banoczy, 1961). The size of the difference in electrical potential created by the metals depends on the nature of the electrolyte, the position of the metal in the electromotive force series, and whether or not the metals are in contact. Farrell (1958), points out that gold or amalgam in adjacent restorations produce the greatest flow of current.

Oral lesions caused by electrogalvanic currents have been described by McDonald (1934), Lain and Caughron (1936), Roome and Dahlberg (1936), Lain et al (1940), Colby et al (1961), and others. Lain and Caughron (1936), examined 1,000 adults with two or more dissimilar metals present in the mouth and found ulceration in approximately 28% of them. The lesions are most frequently found near the offending metals. Ulceration appears to occur only in severe cases of galvanism and the lesions vary in size from a small gingival ulcer to an extensive erosion of the buccal mucosa or tongue. The lesions may not appear for months, or even years, after an electric
cell has been established in the mouth. Other oral manifestations of galvanism include erythema, vesiculation, a dry burning sensation and a metallic taste. All investigators agree that the oral lesions heal rapidly when the injurious restorations are removed.

Burket (1961), considers that galvanic lesions of the mouth are of little importance because of their rare occurrence. However, Lain and Caughron (1936), estimated that evidence of galvanism is present in approximately 60% of the patients whose mouths contain two or more dissimilar metals. Since the use of dissimilar metals in the mouth is a common practice in modern dentistry, the assumption by Inovay and Banoczy (1961), that they are important in the aetiology of oral ulcerations and other lesions, seems reasonable.

(b) Other Electrical Burns of the Mouth.

Electrical burns of the mouth occur chiefly in young children who have been playing with electrical cords. If the insulation is accidentally broken and the bare wire exposed, a surge of current passes through the child's soft tissues (Schultz and Vazirani, 1961; Shafer et al, 1963). The consequent changes range from a mild hyperaemia to severe ulceration and necrosis. When the area of ulceration is extensive it heals slowly and plastic repair of the lip may be necessary. Most patients who suffer electrical burns of the mouth are thrown into a state of shock and require immediate medical attention and hospitalization. Schultz and Vazirani (1961), strongly recommend a more thorough education of the general public to prevent this type of burn.
4. **ULCERATION OF THE ORAL MUCOUS MEMBRANE ASSOCIATED WITH RADIATION BURNS.**

Ionising radiation in the form of radioactive elements is now widely used for the treatment of neoplasms. Following irradiation of the cervicofacial area, changes frequently occur in the soft and hard oral tissues. Meyer et al (1963), state that these changes may be so serious that adequate therapeutic doses of irradiation are contraindicated. The difference between a "tumour-lethal" dose of radiation and that which will produce damage to normal tissue adjacent to the neoplasm, is very small. English and Tullis (1951), and Payling Wright (1956), consider that the epithelial cells of the skin and alimentary tract are extremely sensitive to injury by penetrating radiation.

The changes, or "radioepithelitis", caused in the oral mucous membrane by exposure to radiation are essentially the same as the "radiodermatitis" occurring in the skin (Low-Beer, 1951; Shafer et al, 1963). These changes are directly proportional to the amount of radiation absorbed by the tissues. Mucous membrane injury may vary from a mild localised erythema to an extensive necrosis and destruction of the tissue. Cheraskin and Langley (1956), state that ulceration can occur anywhere in the mouth, but particularly on the gingivae and hard palate. It is frequently associated with osteoradionecrosis and is usually accompanied by severe pain (Bernier, 1959; Burket, 1961). Ulceration is aggravated by a decreased flow of saliva. Silcox (1958), notes that even a liquid diet may be difficult for the patient to ingest. Meyer (1958), reports a case of
mucous membrane ulceration following the administration of 6,100 r to a squamous-cell carcinoma of the left mandibular ridge. An ulcer developed at the site of the original lesion and had a base of exposed bone.

The ulcer caused by irradiation exhibits a necrosis of the surface epithelium which may extend into the submucosa. Thickening of the intimal layer of the vessel walls produces occlusion of the lumen and inflammatory reactions in the connective tissues. Homogenization of collagen and mucoid degeneration may also take place (Stones, 1957; Colby et al, 1960; Thoma and Goldman, 1961).

Bernier (1959), states that the gross tissue ulceration and necrosis which accompany total body irradiation are due to the complete diminution of peripheral leukocytes and the entrance of micro-organisms into the tissues.

Ulcerative lesions produced by radiation burns usually follow the course of a chronic lesion. When healing has taken place the mucosa is usually pale in colour and atrophy of the epithelium and fibrosis of the underlying submucosa occur. The filiform and fungiform papillae of the tongue disappear and there is diminution of sensation and temporary loss of taste (Bertram, 1951).
TREATMENT OF BURNS OF THE ORAL MUCOUS MEMBRANE

Schultz and Vazirani (1961), consider that the following general treatment applies to burns of the mouth:

(a) Control of Pain. All burns are accompanied by some degree of pain and its intensity decides the drug to be used.

(b) Prevention of Shock. Burns of the mouth are rarely accompanied by shock. However, when indicated, emergency treatment should include restoration of body fluids, control of haemorrhage, maintenance of body heat and elevation of the feet.

(c) Prevention of Infection - by using local application of antibactericidal medicaments and antibiotics.

(d) Promotion of Granulation tissue by complete débridement of severely burned areas.

(e) Plastic repair of skin, muscle and mucous membrane at the appropriate time.

1. Thermal Burns.

Treatment consists mainly in relief of pain using anodynes or anaesthetic lozenges. Débridement of necrotic tissue and prevention of secondary infection with the use of antibiotics may be necessary. Thoma and Goldman (1960), recommend painting the wound daily with gentian violet. When severe scar contraction has occurred plastic surgical repair may be necessary.

2. Chemical Burns.

Treatment for chemical burns of the mouth is based on the removal and neutralization of the irritant as rapidly as possible.
Tiecke et al (1959), consider large quantities of water to be the most effective neutralizing agent. Most authors agree that the use of a bland mouthwash is indicated and that for severe ulceration anodynes and the topical use of an anaesthetic should be used. The mouth should be kept clean and free of debris to avoid secondary infection.

For chromic acid burns Sugarman (1952), advises the daily topical application of a 2% aqueous solution of methylene blue. Phenol burns should be immediately washed with 70% alcohol (Burket, 1961). Accepted Dental Remedies (1963), recommends the application of glycerine ether or oil.