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"Role of Indices and the Correlation of Clinical and Clinical and Radiological findings in Periodontal Disease".

Thesis submitted as part of the requirements for the Diploma in Public Health Dentistry, University of Sydney.

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I. INTRODUCTION.

II. THE PERIODONTIUM AND PERIODONTAL DISEASE.

(A) Periodontium

(1) Gingiva.
(2) Alveolar bone.
(3) Periodontal Membrane.
(4) Cementum.

(B) General Classification of Periodontal Disease.

(a) Classification based on Pathology.
(b) Classification based on Etiologic Factors.
(c) Classification based on Anatomical Features of Periodontium.

(C) Clinical Pathology and Radiographic Appearance.

(D) Distribution of Periodontal Disease.

2. Prevalence of Periodontal Diseases (including alveolar bone).
3. Epidemiological Characteristics.

III. ROLE OF INDICES IN PERIODONTAL DISEASE.

(1) P.M.A. Index.
(2) P.I. (Russell's Index).
(3) Radiographic Indices of Bone Loss.
(4) Gingival Recession Index.
(5) Ramfjord Index.
(6) Periodontal Disease Rate Index.
(7) Gum-Bone Count Index.
(8) Oral-Hygiene Index.
IV. **CORRELATION OF CLINICAL AND CLINICAL AND RADIOLOGICAL FINDINGS.**

1. Gingivitis and Calculus.
2. Periodontal Disease, Oral Hygiene.
3. Correlation of Pocket Formation, Tooth Mobility and Suppuration with Periodontal Disease.
5. Periodontal Pocket Formation and Gingival Recession.
6. Oral Hygiene and Alveolar bone loss.
7. Clinically Observed Periodontal Disease and Radiographically detected Bone loss.
8. Gingival Recession and degree of Alveolar Resorption.

V. **DISCUSSION.**

VI. **SUMMARY.**

VII. **BIBLIOGRAPHY.**
INTRODUCTION.

It is obvious from the literature on the subject that periodontal disease has existed from the earliest times and historical records show that there was an awareness of this disease and need for its care. It has been described as the commonest of all diseases amongst Egyptians 4,000 years ago. Oral hygiene was practised by Sumerians of 3,000 B.C.; and the elaborately decorated golden toothpicks found in the excavations at Ur in Mesopotamia suggest an interest in cleanliness of the mouth. The Babylonians and Assyrians following the early Sumerian Civilization apparently suffered from periodontal conditions and they had practised the use of some medicinal substances for mouth washes and local applications. In the oldest Chinese Medical work written about 2,500 B.C. there is a description of 'gingival-inflammation', 'periodontal abscess' and 'ulceration'. Amongst the ancient Greeks, Hippocrates of Cos (89) (46)-355 B.C.), the father of modern medicine, had also described the function and eruption of teeth and the causes of periodontal disease and he believed that this disease was due to deposition of pituita or calculus. Amongst the Romans, Aulus Cornelius Celsus (1st Century A.D.) referred to diseases which affected the soft parts of the mouth. He also advised the treatment with chewing of unripe pears and apples and advocated the removal of the stains by rubbing the teeth. Rhazes (850-923), an Arabian also recognised this disease and advocated the use of honey and opium for its
treatment. Albuconis (936-1013) stressed the care of supporting tissues of teeth and had also designed a set of scalers. Later in the fifteenth century, Vaelseus of Montpellier (1382-1417) also emphasized the need of such instruments for the treatment of this disease. Bartholomeus Eustachius published a book in Venice in 1565, in which he referred to the attachment of the root with the bone by means of ligaments, which kept the tooth firm in its place.

With the beginning of the eighteenth century dentistry assumed the early signs of scientific curiosity. Pierre Fauchard (1678-1761), the father of modern dentistry, had written a good deal about periodontal disease. John Hunter, English physiologist and surgeon, also published some books on this subject and had mentioned the disease of the alveolar process which he believed was the site of suppurative-periodontal disease. The nineteenth century brought new names and developments to the periodontal field.

With the turn of the nineteenth century and the passing of the first half of the twentieth century there developed a profile group of clinicians and scientists throughout the world. Dentists and members of the allied medical field have devoted time to furthering knowledge of this disease and at the present time many new techniques have been developed for diagnostic and treatment procedures. In spite of all this, this disease is widely spread throughout the world and it constitutes a major problem in public health. In most countries
it has a high prevalence, affecting approximately half the child population and almost the entire adult population. With the tremendous increase in population and limited availability of dentists, only a small fraction of the periodontal disease problem is being tackled at the present time. There is hardly any attention given towards the preventive and curative care of this wide spread condition, which is one of the important needs of the communities. This may be, firstly, due to inadequate classification (40) of this disease that has existed for many years, secondly, the unreliable epidemiological data due to lack of satisfactory system for examination and accurate diagnosis, and the absence of uniformity in methods employed in the assessment of the disease in its various forms (40). A number of other reasons could be advanced to account for the comparative absence of reliable data on the incidence and prevalence of this disease. Before describing the role of different indices used for measuring its severity and their correlation in the clinical and radiological findings, a brief description of the periodontium, classifications of disease, and its distribution in the world is presented.
II. THE PERIODONTIUM AND PERIODONTAL DISEASE.

A. PERIODONTIUM.

The term 'periodontium' has been defined as the tissues in immediate proximity to, and surrounding, the tooth, including cementum, periodontal membrane, alveolar bone and gingiva. (83). The periodontium is a functional concept and its different tissues do not attain their final form until the tooth has been subjected to the forces of mastication. Each of the components of periodontium are briefly described.

1. Gingiva.

It is a part of the firm oral masticatory mucosa which surrounds the neck of the tooth and covers alveolar processes of jaws, protecting the underlying structures by its firm structure and is well adapted to resist masticatory forces. On clinical and microscopic features it has been divided into three parts. (83, 22).

Marginal Gingiva is a portion which extends along the cervical level of the tooth at the labial and lingual surfaces and tapers to a knife edge-like structure. It is normally separated from the tooth surface by a shallow gingival sulcus, of approximately one m.m. depth. (91).

Attached Gingival. It extends from the free gingival groove to the muco-gingival junction and is immovably anchored to the underlying cementum and alveolar process. Its surface is stippled resembling that of an orange peel.
Papillary Gingiva is a portion of free gingiva which fills the interproximal space between the two adjacent teeth, and is pyramidal in shape, with the base resting over the crest of the interseptal alveolar bone and its apex tapering to the contact points and normally filling the embrasures in between the teeth. If the teeth are over crowded the papillary gingiva is also crowded accordingly and if there is no contact, the papillary gingiva is reduced. (57).

Gingival Sulcus is a trough like depression and is bounded on one side by the dental cuticle and on the other side by the epithelium of the sulcus. The apical end is called the bottom of the gingival sulcus and is limited by the level of the intact epithelial attachment. In ideal conditions the sulcus is absent or very shallow (57) and it has a particular significance because the initial stages of periodontal disease usually commence there.

The weakest point of the periodontium is the epithelial attachment between the tooth and the oral epithelium. The periodontal membrane is firmly attached to the cementum by strong connective tissue fibers, whereas the epithelial cuff is only pressed against the tooth by the surrounding connective tissue. The mechanical support is strongest close to the cemento-enamel junction where the epithelial cells hardly move in relation to the tooth. The strength of the mechanical attachment decreases towards the gingival margin, just beneath it the epithelium will move in relation to the tooth during
mastication, massage and tooth brushing. Foreign bodies may also be pressed between the epithelial cuff and the tooth i.e. into the physiological gingival pocket. However, the cuff is not absolutely tight and a certain seepage of tissue fluid takes place constantly and this, together with leukocytes, seems to be an important part of the defence mechanism against invading micro-organisms. In spite of these dynamic potentialities, the epithelial cuff is apparently the weakest point in the periodontium. Even if non-attached micro-organisms do not stay in the healthy crevice for any length of time, micro-organisms may be retained on the tooth surface in the form of a plaque. The plaque, if not removed daily, will grow between the epithelial cuff and the tooth, and it is apparent that the defence mechanism has not the same chance to attack the micro-organisms retained in a plaque. These anatomical conditions give the pathological processes in the periodontium their typical features. The tooth facilitates retention of plaque, which is responsible for inflammation in the adjacent soft tissues.

**Epithelial attachment.** It normally extends from the bottom of the gingival sulcus towards the cemento-enamel junction and often passes the latter, and is characterized by a uniform width of stratified squamous epithelium. The relationship between the epithelial invagination and the tooth is still controversial. (57, 83). One school of thought maintains that there is a real attachment between the two and
that the bottom of the gingival crevice is located at the coronal limit of the epithelial attachment. Another school of thought maintains that after eruption of the tooth the epithelial cells only adhere to the enamel surface and that the bottom of the gingival crevice is found at the deepest point of the epithelial cuff. (57, 83). According to this concept the entire epithelial invagination is kept in contact with the tooth surface by circular fibers of the gingiva and the blood pressure.

Since there is continuous movement of the clinical crowns occlusally in order to maintain contact with their antagonists as wear takes place, the epithelial attachment also migrates apically over the root surface. Normally, the distance between the apical end of the epithelial attachment and alveolar bone crest vary from 1 to 2 mm. (83).

Clinical appearance of the Gingiva. (83). Marginal gingiva and interdental papilla are smooth, while attached gingiva is stippled. Stippling disappears during pathologic conditions. In the adult dentition the gingiva has a festooned appearance, corresponding with the contours of the roots. Normally the colour of the gingiva is pale pink due to pigmentation, but it may vary in different individuals according to the amount of general pigmentation, and thickness of the epithelial tissue. In a fair complexioned person it is usually lighter pink than a dark or swarthy individual. If the epithelial tissue is thinner then the colour will appear light
red, because of the underlying capillaries. Sometimes, due to
the presence of melanin in melanoblasts of the basal layer
of epithelium, the colour may appear deep purple in healthy
gingiva. At times pigmentation may come from metals, such as
bismuth, arsenic, mercury and lead and give colours of black,
bluish red, deep blue or silver. (83). Sometimes colour may
be due to food that contains colouring agents. But one should
be familiar with the healthy normal gingiva, in spite of
possibility of different colours.

2. Alveolar bone.

It is those portions of maxilla and mandible which
support the teeth by projecting from the jaw bone and form the
socket in which the roots of the teeth are held by their
periodontal membranes. Its structure is moulded in response
to the functional demands of the teeth and disappears when
they are lost. Alveolar process commences approximately 1 m.m.
below the apical portion of the epithelial attachment. (83). It
has been divided into three parts - the alveolar bone proper
(known as lamina-dura), the cortical plate, and the supporting
bone (known as spongiosa). Each is briefly described.

The alveolar bone proper. It is the inner wall of the
tooth socket. It is a thin definite wall pierced by small
openings for blood and lymph vessels. It fuses with cortical
plates at the border of the alveolar process on the labial and
lingual sides. The bundles of fibers of the periodontal
membrane are anchored in this alveolar bone proper.
The Cortical plate. The cortical plate of the alveolar process is the outer and inner layers of the maxilla and mandible. It is continuous with the cortical or compact layers of the body of the maxilla and mandible. It is thinner in the maxilla than in the mandible. In the mandible, in the region of the anterior teeth, little or no supporting bone is present and two cortical plates fuse with the alveolar bone proper on the labial and lingual sides respectively.

The supporting bone. It is also known as the spongiosa or cancellous bone and is located between the alveolar bone proper and the cortical plates. It contains marrow spaces and is continuous with the spongiosa of the body of the jaws. It is thicker on the lingual than on the labial or buccal side. In the anterior region it is absent or is very thin, while in posterior teeth it increases in bulk.

Constant changes are going on in the bone. Formation and resorption are going on all the time and normally they are well balanced. In a roentgenogram a normal alveolar process will appear as a composite of many layers of bone — the two cortical layers, the two supporting layers and true alveolar bone or lamina dura of the tooth itself. Normal alveolar bone will appear to have an even density from the crest to the apical region. The meshlike pattern is relatively constant, with slight variations universally found in bone because of anatomic variations. The lamina dura is intact, covering the alveolar crest and continuing around each tooth root to form the border
of the periodontal space. The normal periodontal space, containing the periodontal membranes is so small that frequently it is not visible in the roentgenogram, the tooth blending with the surrounding alveolar process.

3. **The Periodontal Membrane.**

   It is the connective tissue which fills the space between the surface of the root and the bony wall of the alveolus, and continues to surround the root and crown as far as the tissues are attached and also extends into the free gingival margin holding it close to the surface of the tooth and supporting them in the interproximal spaces. This gingival segment of the periodontal structure is of considerable importance at the beginning of periodontitis.

   In the non-functioning teeth the periodontal fibers are normally orientated parallel to the tooth surface and they are not organised to form strong bundles. As function commences, differentiation begins and the fibers become oriented more or less perpendicular to the tooth surface.

   The principal fibers are slightly wavy and arise from the cementum and attach at the termination to the supporting and investing tissues of the tooth – the connective tissue supporting the epithelium, the fibrous matter of gingiva, the cementum of the approximating tooth, the outer layer of the peristium at the border of alveolar process. These fibers bear the stress and needs of various levels of the tooth and have been classified into different groups.
These fibers are attached to the bone in the same way as to the cementum, in either case as Sharpey's fibers. It requires a year of function before the periodontal membrane gains its full strength (57), as experienced clinically by the fact that it is much easier to extract newly erupted teeth than teeth which have been in function for some years.

The function of the periodontal membrane is to transfer the masticatory forces from the teeth to the jaws. Increased function will increase the strength and orientation only to a certain extent. If the forces surpass this limit, damage will ensue, when circulatory disturbances and resorption of the alveolar will will take place.

4. **Cementum**

Is a calcified tissue, covering the anatomical root of the tooth and by this the tooth is attached to the connective tissue fibers of the periodontal membrane. In view of its function it becomes one of the most important of the dental tissues, for no matter how perfect the teeth may be, without firm attachment, they become useless and are soon lost. (57). Under normal conditions the cementum grows continuously by opposition of new layers. It reacts to inflammation and functional stress, either favourably by means of hyperplasia of cellular cementum or unfavourably by means of resorption and loosening of the tooth.
THE PERIODONTIUM

Gingival Sulcus
Gingiva
Epithelial Attachment
Cementum
Periodontal Membrane
Alveolar Bone

DIAGRAM OF THE BONE OF THE MANDIBLE

Alveolar Bone Proper
Cortical Plate
Spongiosa

THE INTERDENTAL PAPILLA
FILLING THE AMBRASURE

Free or Marginal Gingiva
Attached/Alveolar Gingiva
Alveolar Mucosa

SUBLISSIONAL DEMARCATION OF THE ATTACHED AND MARGINAL GINGIVA
B. GENERAL CLASSIFICATION OF PERIODONTAL DISEASE.

In the broadest sense periodontal disease has been defined as being all those pathological processes that involve one or more tissues of the periodontium. There is a curious paradox in present nomenclature and classification of this disease, despite the improvement in terminology, which is becoming more and more precise, there is no generic term for this disease formerly known as "pyorrhea alveolaris" (18). In fact A.R.P.A. (18) uses the term "parodontopathy" (periodontal disease) to designate all periodontal complaints, which covers everything from simple gingivitis, whether of local or systemic origin, up to neoplastic lesions. The American Academy of Periodontology (38) uses the term "periodontal disease" which includes practically all gingival and periodontal pathology. It is also termed "alveolar osteitis", "periodontal osteitis" and "periodontitis proper". (80). All these terms refer to the diseases which affect one or more tissues of the periodontium.

Many classifications of periodontal disease have been developed to provide a useful guide for clinical assessment and management of disease. A review of all existing classifications suggests that the periodontal diseases have been differentiated from each other on the basis of three criteria (22) which are:

(a) nature of pathologic changes.
(b) etiologic factors.
(c) clinical features.

Each will be briefly described and their importance shown
from the point of view of role of indices in measuring or assessing the severity of disease.

(a) **Classification based on Pathology.**

This Classification has been adopted by the "American Academy of Periodontology (38, 24). The disease has been characterized by the different morbid processes which constitute deviation from the normal behaviour of the tissues. It is accepted that three fundamental types of pathologic processes differing distinctly in their characteristics, origin, and course have been observed. This classification has been suggested on the basis of differentiation of these pathological processes, which are:-

(i) **Degenerative.**

(ii) **Neoplastic.**

(iii) **Inflammatory.**

(i) **Degenerative disease of periodontium.**

It applies to regressive changes related to a general or local metabolic deviation,(59, 48,) which are characterized by structural changes resulting in the disappearance from the histological picture of certain elements, or the appearance of substances foreign to the normal composition of the cells or tissues.

In the periodontium, the degenerative process may involve the gingiva, the alveolar bone and the periodontal membrane. The gingival tissues may alone be affected – for which the term *gingivosis* has been used. (80). It is a rare disease and
was first described as occurring among starving children in post war Italy (Schour and Kassler). It is, clinically, similar to desquamative gingivosis, which is occasionally observed in women during menopause. Since underlying tissues of periodontium are closely linked to each other, so degenerative involvement of one of these tissues may lead to changes in the adjoining tissues. For example, from the gingiva it may spread to alveolus and periodontal membrane. When marginal part of alveolar bone is involved the term 'marginal alveolar bone atrophy' has been used, which develops without any inflammatory symptoms or pocket formation. When periodontal membrane is initially affected the term 'periodontosis' (24,38) has been used. It is characterized by the resulting increased mobility and migration of the teeth. There is rapid formation of deep pockets and secondary inflammatory symptoms may appear. X-ray will reveal vertical bone loss and the thickening of periodontal space.

(ii) Neoplastic disease of periodontium (24):

The periodontium may be the site of multiple neoplasms derived from its various constituents e.g. epithelium, connective tissue and vessels. According to the particular case, these neoplastic processes may take the form of diffused or localized hyperplasia (commonly known as epulis) or more rarely, an invading and destructive process. The neoplastic manifestations of the periodontium may be fibroma, angioma, osteoclastoma, periodontal cysts, cementoma.
(iii) Inflammatory disease of periodontium:— (46)

An inflammatory process develops whenever a given factor attacks the integrity of the tissues by physical or chemico-infectious action. It is a type of reaction and repair involving the non differentiated connective tissue and its circulatory elements. The differentiated connective tissue like bone and cementum (61), the parenchymatous tissues and the neighbouring epithelia may also undergo secondary changes.

The irritating agents act on the periodontium either directly on the surface (marginal), or indirectly in the deeper tissues (through the intermediary of the tooth). In the first case, lesions caused by chemicals, bacteria, and most varied mechanical agents are involved. In the second case, mechanical factors like abnormal occlusion act in depth on the bone and periodontal membrane and then inflammatory process leads to destruction of the tissues.

Tissues affected are divided:

(1) **Gingivitis**. (22, 24, 83).

(2) **Periodontitis** (22, 24, 83). Each is briefly mentioned —

(1) **Gingivitis** — It has been differentiated according to the duration of its occurrence:

**Acute** —

**Acute ulcerative** or necrotizing gingivitis — when the marginal part of the gingiva, particularly the papillae, are the seat of necrotic or ulcerative process leading to
destruction. Onset is with sudden pain in the region with general malaise. If neglected, the acute phase may allow the inflammation to extend in depth.

**Acute non specific gingivitis** - It is the result of local trauma, or other diseases such as herpetic stomatitis, streptococcal throat, blood dyscrasias.

**Chronic** -

**Simple gingivitis** - Where papillary, marginal and attached gingival are involved but alveolar bone and periodontal membrane are intact.

**Hyperplastic gingivitis** - Where gingival inflammation of a proliferative nature takes place, with the formation of new collagenous fibers and weak cellular reaction. There are large false pockets.

These two varieties of chronic gingivitis may be modified by systemic disease or conditions such as pregnancy or the use of drugs such as diphenylhydantoin.

(2) **Periodontitis** - It has also been differentiated according to the duration of its occurrence.

**Acute** -

Such as periodontal abscess, or developing from acute ulcerative gingivitis. Mostly it is due to local tissue injury, followed by healing or further destruction.

**Chronic** -

**Periodontitis simplex**: Where marginal horizontal bone resorption has taken place and true pockets have formed which
are usually of regular depth. This may be the result of marginal irritation and possibly aggravated at some stage by occlusal trauma.

**Periodontitis complex:** Where irregular bone resorption has taken place and pockets of varying depth are present. It is often observed in young individuals and is associated with recognizable systemic diseases, but may commence as periodontosis. Sometimes it is considered to be primarily a degenerative lesion with inconspicuous local factors e.g. occlusal trauma.

(b) **Classification based on etiologic factors.**

Ferain A. Garanza (14) has suggested the classification, where terminology of the periodontal disease is based on etiologic factors are:

(i) **Determining factors**

(A) Inflammation.

(B) Trauma.

(C) Combination of Inflammation and Trauma.

(ii) **Predisposing factors.**

Each will be briefly mentioned with reference to the suggested terminology of the disease.

(i) **Determining factors:** These are of local origin e.g. materia elba, calculus, over hanging fillings etc., but their action can be aggravated by certain systemic factors like hormonal, nutritional and gastrointestinal disturbances.

(A) **Inflammation:** When 'Inflammation' is the
causative factor, and disease is localised only to the gingiva, producing the clinical picture of chronic marginal gingivitis, without true pocket formation the term inflammatory gingival syndrome has been suggested.

Further, due to the severity of inflammation the epithelial attachment has proliferated apically with a true pocket formation and involvement of the alveolar bone and periodontal membrane, the term inflammatory periodontal syndrome has been used for this condition. When reparative capacity of bone is normal and only superficial bone loss has taken place, the condition has been termed - superficial inflammatory syndrome. But where reparative capacity of bone is poor and severe bone loss has resulted, the term deep inflammatory syndrome has been used.

Clinically inflammatory periodontal syndrome has been characterized by supra-alveolar, supra-boney pocket formation, gingival bleeding, suppuration, subgingival calculus, and horizontal bone loss. Tooth mobility has shown to be a late sign. Clinically, superficial inflammatory syndrome has shallower pockets and a slower progress than deep inflammatory syndrome.

(B) Trauma: Traumatizing factors produce certain lesions, where disorganization (53, 3, 62, 69, 85) of periodontal membrane takes place and bone loss is favoured and this condition has been termed traumatic periodontal syndrome.

When traumatizing influence is not severe and lesions
produced are repaired and no clinical manifestations, except a slightly increased tooth mobility at bite pressure is observed—this condition has been described as compensated traumatic lesions. Further when traumatic forces exceed the reparative potentialities of the tissues, causing degenerative changes in the periodontal membrane and giving rise to tooth mobility, migration of teeth and formation of diastema, without pocket formation and presenting widening of periodontal space and fuzziness or absence of lamina dura and reduction of alveolar crest in radiographs. This condition has been termed uncompensated traumatic lesions. The cause of traumatic forces may be occlusal disharmony, bruxism and lip biting.

(c) Combined lesions: These are of both inflammatory and traumatic origin and in combination share their characteristics, and have been termed combined periodontal syndrome.

When the deleterious action of these combined factors remains below the threshold of resistance of the tissues, the clinical picture of chronic marginal gingivitis, with a slightly increased tooth mobility is clinically observed and this condition has been termed compensated combined periodontal syndrome. Further, where trauma and inflammation act upon weakened periodontal structures, going beyond the threshold of resistance, producing tooth mobility, deep pocket formation, calculus, suppuration and migration and this condition has been called uncompensated combined periodontal syndrome. X-ray will reveal widening of periodontal space localized or generalized loss of lamina dura and vertical bone loss.
(ii) Predisposing factors:-(75) These are of systemic origin and have been put under two groups. Each is briefly mentioned.

(A) A deficiency in the mechanism of bone formation which may be altered due to lack of thyroid hormones, lack of androgens, lack of somatotrophin, and lack of function. Bone matrix may not be normal due to lack of proteins, Vit. C, deficiency, which may be primary or secondary. Mineralization and enzymatic factor may be defective due to primary calcium deficiency, adequate Ca/P ratio, lack of magnesium or vitamins, increased calcium or Vit. D, excretion.

(B) An exaggeration in the mechanism of bone resorption— as a result of hyperparathyroidism, acidosis, renal failure, or osteolytic neoplasm.

These systemic factors constitute the real internal predisposing factors for periodontal disease by altering the normal metabolic interchanges of bone and periodontal membrane and reparative capacity of tissues. (19,20,25,84,37). They are seldom diagnosed but favour the destruction produced by local factors.

(c) Classification based on anatomical features of the periodontium. (80).

The terminology of the disease is based on the particular structure of the periodontium involved in it. It has been put into three groups:-
(i) Gingival diseases.

(ii) Diseases of alveolar bone.

(iii) Disease of bone and gingiva.

Each is briefly described.

(i) **Gingival diseases:** Those affecting the gingival tissue only and not involving the bone and the periodontal membrane. It has been further subdivided, according to the anatomical classification of gingiva in terms of its three component portions - papillary, marginal and attached gingiva. (79,83). This gives rise to the more precise designation of papillary gingivitis, marginal gingivitis and attached gingivitis, depending which region is predominantly involved in a given case. Where the inflammation of the marginal gingiva is the predominating characteristic, marginal gingivitis is the term used, which is more accurate than just gingivitis.

(ii) **Diseases of alveolar bone:** Those affecting the alveolar bone or cementum and periodontal membrane, without involving the gingival tissues. This stage normally follows when gingivitis advances and follows the course of the blood vessels and spreads into the bone marrow and its periosteal surface and has spread into the periodontal membrane. But at times the disease may have started in the periodontal membrane and then proceeded to the bone, as in periodontosis (80) and finally into the gingival tissues. In such cases clinical diagnosis becomes difficult and X-ray is essential. When
alveolar bone is involved the term Alveolar osteitis has been used and when periodontal membrane is involved the term periodontitis proper has been used.

(iii) Disease of bone and gingiva:— Those affecting both the gingival and bony tissue along with the periodontal fibers. This has been termed the true periodontal disease. Initially gingiva is involved and then spreads into the alveolar bone involving periodontal fibers.

The general classification, as briefly described above, shows how the different terminology has been used for different characteristics of the periodontal disease. It is accepted that most valid basis for classification of periodontal disease is the one based on general pathology. But for the purpose of epidemiologic surveys of this disease, this classification is not suitable, as it needs histopathological examination, which is impossible. Consideration of etiology, although important, can play only a secondary and accessory part in a classification by contributing certain details for minor sub-divisions. Study of causes is also an unsuitable basis for classification in the epidemiological studies, as many etiological factors, some possibly unknown, may be involved in any particular case.

The classification based on anatomical features of the periodontium appears to be quite suitable for the purpose of epidemiologic surveys. Specially because etiology of periodontal disease is very complex and its frequency is very high throughout the communities and the only suitable way of
assessing is by adopting clinical examination. The subdivision of gingivitis, on the basis of anatomical regions is relatively simple, since they are clinically visible. The disease of alveolar bone and periodontal membrane is also easy to diagnose clinically in its late stage—which will be shown by the pocket formation, suppuration, mobility of tooth. But in the early stage of its onset it is difficult to diagnose clinically. This can be recognised only by roentgenographic examination. The writer feels that classification based on anatomical features is quite adequate for the epidemiological investigation of periodontal disease.

C. CLINICAL PATHOLOGY AND RADIOGRAPHIC APPEARANCE.

Clinically it is not possible to detect the histopathological changes which take place in the tissues of the periodontium, and it is also not possible to examine the status of disease by histopathological investigations in field studies. Therefore in assessing the severity of periodontal disease it is essential to correlate the clinical and radiological findings with the histopathological state, which is possible to a certain extent, and acts as guide in assessing its severity.

(1) Main features of clinical pathology of the periodontium (22, 83) are as follows:—

(1) Colour: If the gingiva is red or magenta, this is a sign of inflammation. The colour also depends on the degree of keratinization and thickness of epithelium, the amount of oedema, and the presence or absence of pigments. In some cases
keratinization may be absent; if so, the capillaries will shine through even in the absence of inflammation. In other cases pigmentation may camouflage an underlying inflammation. A severe inflammation in the part of the gingiva bordering the crevice or pocket is often not evident from the oral surface. Therefore while examining the colour of the gingiva one has to be careful, because sometimes it may be misleading.

(2) **Contour:** Periodontal disease, mostly gives rise to the thickening of the gingival margins and swelling of papillary region. But sometimes these signs may be the result of vigorous brushing of the teeth or may merely represent anatomical variations. Therefore, much attention should be paid to reach the correct diagnosis.

(3) **Bleeding:** In the presence of inflammation there is either spontaneous bleeding or bleeding may take place on digital pressure. Bleeding may also occur on probing and instrumentation, which is due to cervicular ulceration and increased vascularity of the inflamed connective tissue of gingiva.

(4) **Pus secretion:** When the pus beads are observed from the pocket, specially on digital pressure, it is a definite sign of severe inflammation.

(5) **Pocket deepening:** In the late stages of inflammation, the pockets become more marked and the depth becomes more than 3 m.m. It is a confirmed sign of pathological lesion. But there may be exceptions such as pockets around erupting teeth and pockets where successful treatment has been
carried out, which are non pathological.

(6) **Increased tooth mobility**: It is in most cases a sign of pathology, where deep seated structures are involved. But in the absence of pathology, it is sometimes observed in teeth that are subjected to heavy function. Therefore other signs of disease must be confirmed before increased mobility can be taken as a sign of periodontitis.

(7) **Tooth Migration**: It is caused by the pressure from the granulation tissue within the walls of the pockets. If a pocket is of irregular depth, this pressure, although slight, will be uneven around the tooth and may cause it to migrate with the development of a diastema.

(ii) **Main features of the radiological appearance of periodontal disease** (22, 83).

It is essential to differentiate between the earliest clinical signs and the earliest radiographic signs of the periodontal disease. A considerable degree of alteration must occur in the periodontal tissues before X-ray shows any sign of changes. When the radiographic changes appear the disease process has progressed to a stage sufficient to alter the structures to a radiographically detectable degree. A sequence of changes observed radiographically in destructive periodontal disease associated with the extension of inflammation, from the gingiva into the underlying structure is briefly presented.

(1) The inflammation extends from the connective tissue
of the gingiva to the underlying bone, through blood and lymph vessels. This happens usually when early stages of gingivitis are not treated in time. This stage of clinical pathology can be detected radiographically, which will produce a radiographic fuzziness, instead of the normal clearcut linear outline, either on the mesial or distal aspect of the septal crest.

(2) When inflammation has progressed further, giving rise to widening of vessel channels, and pocket formation, this stage can be detected in roentgenogram with a break in the continuity of the lamina dura in the area of fuzziness.

(3) When pocket has formed and suppuration has set in, it shows that inflammation has progressed further. This stage can be detected radiographically, and will present a wedge shaped radiolucent area on the mesial or distal margin of the crest of septal bone with an associated widening of periodontal space.

(4) If still deeper extension of inflammation has taken place into the alveolar bone and a severe pocket has formed, X-ray will present clear demarcated finger like radiolucent projections extending from the marginal area into the interdental septum.

(5) With the further extension of inflammation, X-ray will present a progressive bone resorption, where the height of the alveolar bone is reduced. This stage may present certain amount of tooth mobility, or may not, as periodontal space is responsible for its mobility to a certain extent.

In certain conditions like periodontosis, occlusal trauma,
and certain systemic disturbances, these above mentioned radiographic changes may have different picture and may not present any correlation with the clinical pathology.

D. DISTRIBUTION OF PERIODONTAL DISEASE.

From the available data it is apparent that periodontal disease, in some degree in one form or the other, affects large population groups, at all ages and frequently remains undiagnosed until the disease is well advanced. It has become a major public health responsibility, not only an important cause of tooth loss and consequent reduction in masticatory efficiency but also because of its probable influence upon general health through focal infection. (38). A brief description of the prevalence of periodontal diseases is presented.

1. Prevalence of gingival disease:

A brief review of the wide range prevalence investigated by different investigators is described. In the United States Massler (45) and others examined more than 1,000,000 white boys and girls aged 6-14 years; and observed gingivitis prevalence of only 3.5 - 8.6 per cent. Brucker (10) also observed a similar low prevalence, 8.7 per cent, in a group of children from 4 to 16 years of age. Marshall Day observed in a small group of 234 children aged 6-18 years, in Rochester, 73 per cent prevalence (38), and McCull showed a prevalence of 98 per cent in his findings (50). Massler (46) et al examined 804 children from 5 to 14 years of age in Chicago and observed 35.7
percent were free from gingival disease. King (32) in a
survey, carried out in England, observed 2.5 to 43.6 percent
freedom in varying groups, at 11 to 14 years of age. Campbell
and Cook (22) also observed gingivitis prevalence, in 2924
cases examined, of only 2.2 percent. Schour and Massler (79)
investigated in Italy and pointed out that 44.7 to 50.7 percent
of examined children, from 6-20 years of age were free from
gingival disease. Marshall Day et al (39) observed 21 to 40
percent freedom from gingival disease in a survey carried out
in Puerto Rico, of children from 6-18 years of age. He also
observed a 43 percent freedom at the age of 6 to 18 years in
the Virgin Islands (40). Lodavala and Harris (33) in Chiang
Mai Provence in Thailand, observed 47 percent of 313 children
examined had some degree of gingivitis. Davis (14) surveyed a
Polynesian group on Pukapuka atol and observed 47 percent out
of 497 persons had some sign of gingival and periodontal
disease. He also concluded that the disease begins early in
life and increases in severity with age. Vivone in Argentina (88)
observed ulcerated gingiva in 36 percent of male and 28 percent
of female school children averaging about 16 and 15 years of
age, and in from 20 to 32 percent of adults.

Marshall Day et al (41), in a study carried out in India,
observed that gingival disease prevalence and severity is
related to socioeconomic status. He observed that children
of high socioeconomic status showed 73.7 percent of gingival
disease prevalence, and in the same age group (7 to 17 years of
age) in a low to middle class group showed 99.4 percent of prevalence, with 73.6 percent in the severe category. Factors inherent in the socioeconomic status and probably contributing to the difference in gingival disease prevalence in India, have been shown as the result of lack of oral hygiene and malnutrition. In the United States, using precisely the same diagnostic criteria, a percentage prevalence of 73.0 was observed at 6 to 18 years of age in Rochester, New York, (38) and 77.0 at 13 to 18 years in Boston (43). Other studies summarized by Cunningham (13) in his review, and those cited suggest that variations in prevalence and severity of disease exist, but it has wide prevalence throughout the world. Variations observed may be due to geographic or racial background as observed by the comparative immunity of Virgin Island Negros and the severity of the gingival changes in children of low economic status in India. But many of the differences observed may be related to the lack of a standardized system of examination and recording and differences in interpretation.

Further, it has been concluded that the prevalence and severity of gingival disease increases with age. In the child, sharp rise in prevalence from 6 to 8 years of age has been related to the eruption of permanent teeth. Marshall Day (38) compared four distinct racial groups and observed that average incidence rises from 6 years of age, with some irregularity to reach a peak at about the age of puberty or
earlier. In United States the peak is reached at age 10 to 12 years (38,43,46). In Puerto-Rico at age 12 years and in the Virgin Islands (Negro) and in India at 14 years. It has been further shown that in these four racial groups the incidence declines quite sharply to age 18 years or beyond, with a subsequent increase again in the early twenties. Then the incidence follows a gradual though steady upward trend, until a prevalence of almost 100 percent is reached between 50 to 60 years of age, as observed by Marshall-Day et al (43). The sharp downward trend during adolescence has been variously related to the end of the puberty and the tooth eruption period, and/or social factors, associated with greater emphasis on oral hygiene and awareness of aesthetic values at this age.

2. Prevalence of periodontal diseases (Including alveolar bone). Studies on the prevalence of chronic destructive periodontal disease, where alveolar bone is involved, have been undertaken less frequently than for the gingival disturbances. Its prevalence, as investigated by different investigators, is briefly presented.

Black in Chicago (8) area observed a prevalence of periodontal infection from 13 percent at 20-24 years of age, increasing to 88 percent at 50 years and over. Miller and Seidler (56) reported the need for periodontal treatment in 62 percent of the males and 56 percent of the females in 1003 hospital cases after roentgenographic analysis. They
They observed actual bone destruction increasing from 9 percent in the 11-15 years age group to 97 percent at age 41-45 years. From a study of 5,014 men in Veterans Administration Dental Clinic in Chicago, Belting, Massler and Schour (20) found that the incidence of combined alveolar bone diseases reached a peak at 45-49 years of age, when 42 percent of the male population was affected. A thorough study carried out in Boston on 1300 persons, by Marshall-Day, Stýphens and Quigly (43) shows that about 80 percent of children showed some evidence of gingival disease. While this prevalence dropped with increasing age, there was compensatory increase in the proportion of persons showing evidence of alveolar bone loss in the roentgenograms. Bone loss was observed in virtually all persons aged 35 years or older. Bossert and Marks (9) found in a study of 12,800 employees of an insurance company in New York, that the active disease was present in only about 30 percent, aged 35-39 years, with prevalence rising with age to about 60 percent in persons 60 years of age or older. Mehta, Grainger and Williams (54), made a series of pocket measurements in 670 Canadian civil servants and observed gingival crevices deeper than 3 m.m. in 85 percent of individuals, aged 15-19 years, and in about 24 out of 25 persons at all older ages. Russell (68) examined 1676 citizens of Baltimore, which included males, females and about one quarter of Negro participants. He disclosed 4 percent of persons with one or more overt periodontal pockets at the
age of 15-19 years, and 56 percent at the age of 60 years and over. He also concluded that as the age advances, the periodontal disease becomes more prevalent. A survey carried out by the American Dental Association in May 1952, which included 3900 persons, showed that periodontal treatment was needed by 9.5 percent of males and 9.7 percent of females aged 15 years and over, for an average of 13.8 and 13.6 teeth per case. It further showed that extractions for periodontal disease were needed by 11.2 percent of males in the same age group, for an average of 8.2 teeth per case, and by 8.3 percent of females, for an average of 7.1 teeth per case. For patients aged 35 years and over in males, and 40 years and older in females, periodontal disease was the reason for between two and three times as many extractions as dental decay. Thus the Association concluded that some degree of periodontal deterioration was present in virtually all adults, and that this deterioration proceeds to gross destruction of tissues in about half of all persons aged 50 years and older and that in middle aged later life, periodontal disease is by far the most important cause of tooth loss.

In other countries studies were carried out on population groups of different ethnic origins and nutritional states. In India, Marshall-Day and Shaurie (44), carried out a roentgenographic survey of bone loss in males, mostly police constables, and observed a figure of 0.19 for average resorption at the age 15-18 years, and 3.12 at the age 50-60
years. They also concluded that chronic destructive periodontal disease is incipient at an early age and progresses more rapidly in older age groups. Greene (30), who determined P.I. scores (which varied from .70 to .8) for boys aged 11, 13, 15 and 17 years in public schools of Bombay (India) and rural Basen; observed the same relative differences, as recorded by Marshall Day (43) et al in their study at Boston. Mehta, Sanjana, Schroff and Doctor (50), in a survey for the extraction of 18,030 teeth in 4278 consecutive registrants at the Noir Dental Clinics in Bombay, found that periodontal disease was the cause of loss of 11,960 teeth (63.3 percent of the total) for patients of all ages, and was responsible for the loss of 79.2 percent of teeth at all ages after 30 years. McCombie and Chu (51) examined national service men in Singapore with an average age about 21 years, and concluded that P.I. scores for 196 Chinese, 130 Malaysians and 73 Indians and Pakistanis were 0.91, 0.70 and 0.83 respectively, about the same or slightly higher than those observed by Greene in Bombay (30). Sinclair, Cameron and Goldsworthy (82) carried out an X-ray examination of native adults in New Guinea, and observed moderate to extensive alveolar bone loss in 53, 69, and 76 percent of the persons in three different groups. Dodds (16) in a summary of the literature, concludes that "periodontal disease" is prevalent among the inhabitants of Africa, but among East-African natives it is less prevalent than among other races. The
disease attacks without regard for race or colour, but its incidence increases with age and is negligible in children. (16) The reports he cites give prevalence figures rather lower than those for other areas; from 30 to 50 percent for Europeans, and as low as six percent in some primitive Bantu Workers or as three percent of severe disease in Masai. Loss (34) noted that reported percentage of prevalence range from nine of 50 percent in Europe. From his general statement it is clear that this disease presents no formidable problem throughout the whole world.

3. Epidemiological Characteristics.

Two population patterns in periodontal diseases have been clearly observed according to the findings of the investigators. Firstly the numbers of people with gross and destructive disease increases as the population ages, and secondly with this increase in numbers there is an increase in the average severity of disease as well. In the susceptible individuals the age of onset may be as early as puberty as shown by Marshall Day et al, (43, 44), and Mehta et al (54). Many investigators have shown that high levels of periodontal disease have been observed in populations suffering from one or another dietary deficiency (19, 85, 15), but this needs further investigation as no consistent relationship has been discovered by Russell and co-workers (69).

Some independent groups have investigated within group differences which seem to be associated in some way with the
status of the individuals in the sub groups. Lovdal and Arno and Waerhaug (35) observed in their findings, carried out for 1202 men in industrial concern, that official personnel engineers, who were university graduates, technicians and foremen, were 'conspicuously better' than ordinary workmen. Mehta, Sanjana, Schroff (53) and Doctor examined 1640 boys, 11-16 years of age dividing them into high, middle and low groups on the basis of family income. They observed some degree of gingivitis in nearly all the boys, severe disease characterized by excessive swelling, extremely deep pockets on buccal and lingual aspects, spongy tissue, profuse bleeding was present, with rare exceptions, only in boys of the low income groups. Russel (70), observed in his findings in the United States that differences between rural and urban children disappeared when comparison was limited to groups of children whose parents had about the same degree of education, and that educational attainment was directly related to their own periodontal condition. Arno (2) and co-workers in their study of an Oslo industrial group, concluded that higher incidence of gingivitis among the workers may be attributed to the poor oral hygiene, rather than environmental factor. They also observed that there was no significant difference in the gingivitis percentage between males and females who belonged to the same occupation, hygiene, tobacco consumption and age group.

It has been shown by Greene (30) that general stage of
hygiene in the mouth, presence or absence, or extent of calculus and debris presented invariably a relationship to the condition of periodontal tissues, when within group comparisons are made. Schei, Lovdal and Arno (76), compared bone resorption measurements for men with poor, with fairly good and with good efficiency in tooth brushing (as indicated by the presence or absence of debris in all or any parts of mouth), and they observed that men with good efficiency had by far the least degree of bone resorption; that men with fairly good efficiency had lost an intermediate degree of bone; and that men whose brushing efficiency was poor had lost by far the greatest amount of bone.
III. ROLE OF INDICES IN PERIODONTAL DISEASE.

Principals and methods of epidemiological concept are being applied to communicable and other diseases, including periodontal disease, throughout the world, which relate to the initiation and progression of the disease in groups of communities, in order to bring about its effective prevention and control. With the development of research in recent years, two approaches to epidemiology have been followed, more or less separately. One group of investigators has been working from the clinical and experimental side, while another group has been studying the actual distribution of disease in a population, in relation to various factors and conditions which affect their distribution. The field studies of periodontal disease have been carried out by using certain methods and techniques for recording its occurrence and classifying its degree of severity. (28,58).

These methods of scoring the disease have been termed periodontal indices, which express the severity and occurrence in population groups, or in the individual person. The objectives (65) for scoring system are as follows:

(1) To map out the distribution of the disease (prevalence)
    (a) In population groups.
    (b) Within each dentition.
    (c) Around each individual tooth (mesial, distal, buccal, and lingual).

(2) To record the progress and behaviour of the disease either by longitudinal studies of the same group or by comparing
prevalence studies of various age groups within the same population (incidence).

(3) To serve as a basis for evaluation of the role of various etiologic factors in the pathogenesis of the periodontal disease.

(4) To allow an estimate of total need for periodontal therapy in population groups.

(5) To achieve a basis for education of the dental profession, the public, and the governmental authorities regarding the need for attention and treatment of periodontal disease; to estimate further needs for dentists and auxiliary personnel.

(6) To observe as a basis for testing and evaluation of various procedures for periodontal treatment.

(7) To serve as a basis for evaluation of the effectiveness of various measures in preventing or delaying the loss of teeth from periodontal disease.

(8) To serve as a basis for evaluation of measures to prevent periodontal disease.

Ramfjord (63), emphasized the importance of these objectives and suggested that the literature on epidemiology of periodontal disease should be evaluated on the basis of whether or not these objectives have been met adequately.

Greene and other investigators have suggested that the appropriate method is dependent upon the project objectives, and the choice of an index in any specific situation must be based upon the nature of information required, the numbers of persons
to be considered, and the type and amount of equipment that the circumstances and the location of the study will permit. He also described certain characteristics for such methods of classifying degrees of severity of periodontal disease for epidemiological studies. These are as follows:—

(1) The method of choice must have definite clear cut criteria which reduce examiners decision to a minimum.

(2) The method should be sufficiently quantitative to provide a measure of the degree of severity of the disease in population groups.

(3) The method should be practicable for use in epidemiological studies of large populations, and should have speed and simplicity of application rather than complexity even when the speed and simplicity require sacrificing some details.

(4) The technique to be employed must be within the abilities of those employing them, and even general practitioners should be able to use it.

(5) The recording should be suitable for reduction to a final score or index which may be subjected to statistical analysis.

It is observed from the various investigations carried out that two measuring devices have been mostly used in estimating the prevalence of periodontal disease, and since these techniques are based on clinical and radiological methods, it is not surprising that the estimates vary.

A single clinical index for this disease has not been
possible to develop so far, because of the multiplicity of
changes in tissues which are utilized to describe each disease.
Mostly two areas are involved —
(a) the gingiva, where gingivitis is usually a reversible
process, being active, indicates response to treatment and can
easily be detected clinically.
(b) the loss of alveolar bone, or true periodontal disease,
where bone loss is chronic, cumulative and irreparable — is an
irreversible process, and is hard to observe clinically.
The role of different commonly used indices is described.

(1) **THE P.M.A. INDEX OF GINGIVITIS.**

In the course of a nutrition survey carried out in 1945 in
Italy, Schour and Massler (79), classified patients on a scale
for gingivitis running from zero through four, based on
examination of the labial gingivae of the lower anterior region.
With this experience as a basis, they devised the P.M.A. index
of gingivitis. Brief descriptions of this scoring system were
published by Massler in 1948 (77) and 1949 (47).

Basic to the method is the concept that extent of
gingivitis indicates the severity of the affection. Schour
Massler and Chopera agreed with King that inflammatory lesions
tend, for the most part, to begin in the papillary gingiva and
to spread from that area into the marginal gingiva. In severe
gingivitis the inflammation then spreads to the attached gingiva
(periodontitis). The degree of severity of the inflammation is
usually indicated also by its location, mild gingivitis being confined to the papillae, moderate gingivitis involving the marginal gingiva and severe gingivitis affecting the attached gingiva — as differentiated by King (32). He recorded three degrees of severity for each gingival "unit" i.e. each area of attached gingiva, marginal gingiva and papillary gingiva. In this form then P.M.A. was not yet one index, but three. Later on Massler and Savara (48), modified this method of analysis to one, in which scores for P.M.A. were added to give one total, which was taken as representing the status of the individual. This was a true weighed scale, where the gingival area mesial to each tooth could be scored from zero through three, depending upon the extent of the inflammation.

The criteria directed that a gingival unit be scored as positive if it departed in any way from normal. Normal was specified as a gingiva of a pale, pink-colour, of firm texture without bleeding on firm digital pressure, of normal contour, and of close adherence of the free gingiva to the teeth just coronal to the cementoenamel junction, and of the attached gingiva to the alveolar bone (46). It is not possible to describe the same things to all men. The phrase "pale, pink-colour and contour" probably convey as many minute differences as there are dental examiners. Further, the skill and judgement of an examiner are put to the greatest strain when he is asked to recognize and report the smaller deviations from any normal. Agreement between P.M.A. examiners may be difficult to attain.
This was shown by Sandler (74) for eight dentists who took part in P.M.A. examination of 10 adults and showed a considerable divergence in their estimates for each of the patients. But it is felt by the author that the P.M.A. index has been designed for a population and not as a diagnostic aid and that disagreement may be essentially random between dentists as shown by Sandler, and this has hardly any importance where a very small sample of ten people is selected.

In the P.M.A. index, no mandatory examination procedures have been prescribed. Examination may include the entire mouth or merely an anterior segment. It may be visual or be supplemented by colour photographs (49). Some investigators have included only the numbers of inflamed gingival papillae (36). Stahl and Morris (86) counted the numbers of inflamed papillae, the numbers of inflamed marginal areas and the numbers of teeth involving gingival recession, and reported these items separately as the mean percent involved per man. In a study limited to maxillary anterior areas, Parfit (61), restored the severity degress originally used by Massler, Schour and Chapra (46), using the severity score for the most highly inflamed area as the score for individual. (61).

P.M.A. index is very sensitive to small changes, when disease is mild and is useful in clinical trials of the effectiveness of agents like mouth washes or tooth brushing on gingivitis. It has been developed solely for assessing the gingivitis and cannot be applied to the disease affecting the deeper periodontal
tissues. It seems to be quite adequate for young children, where all the indices are measures of simple gingivitis. This simplest one to apply under primitive conditions, as the examination can be carried out by visual methods, using only the tongue blade.

(2) PERIODONTAL INDEX.

This index was developed for the assessment of chronic destructive periodontal disease by Russell (68), but there is provision for recording gingivitis also. It is applied by a swift appraisal of the supportive tissues of the individual teeth, and records progressive stages of inflammation of the gingiva, pocket formation with consequent loss of alveolar bone, loss of function of the tooth and eventually loss of the tooth itself. Each tooth is scored according to the clinical conditions of supportive tissues. In the absence of overt inflammation in the investing tissues or absence of loss of function due to destruction of investing tissues - a tooth is assigned a score of zero. The scale has two stages of simple gingivitis, differing only in the extent of inflammation. If there is an overt area of inflammation in the free gingiva, and does not circumscribe the tooth - the score of one is assigned and is described as a mild gingivitis. If the inflammation completely circumscribes the tooth, but there is no apparent break in the epithelial attachment - the score of two is assigned, and this clinical condition is described as gingivitis. Further, when the epithelial attachment has been broken and there is a pocket
(not merely a deepened gingival crevice due to swelling in the free gingiva), but there is no interference with normal masticatory function and the tooth is firm in its socket and has not drifted - a score of six is assigned. This clinical condition is termed gingivitis with pocket formation. Lastly when tissue destruction has advanced so far that the tooth is loose and may have drifted and may sound dull on percussion with a metallic instrument, It may also be depressible in its socket. It is assigned a score of eight and this clinical condition is termed advanced destruction with loss of masticatory function. A definite rule for further guidance is also given, that whenever any examiner is in doubt, a lower score is always assigned. Thus the average score for the teeth in the mouth of an individual is taken as the periodontal score for that person and population scores can be determined. Most persons with a clinical diagnosis of gingivitis score from 0.1 to 1.0, those with frankly established destructive disease from 1.5 to 5.0 and those with disease in terminal stages from about 4.0 to 8.0 - the maximum score. (68).

This index has been developed after a considerable period of trial and error by Russell and his co-workers. Previously it had involved long and tedious examination procedures where a probe was used for measuring the depth of pockets and a history of missing teeth was also recorded in the patient's score. Radiographs were also eliminated because their use raised the scores and was not possible in most of the field studies where
electricity and transport were not available.

According to Russell's findings this index is quite good for comparing population groups. He found that comparability of population findings between trained examiners is as good with this index as on counts of D.M.F. teeth (10). He further concluded that this degree of comparability depends upon mandatory procedures, and a series of criteria designed to place the minimum strain on examiners' judgement. In this index reporting is limited to advanced and quite obvious lesions - those clearly evident at first glance in good light (68). Whenever an examiner is in doubt, he has to score a lesser score.

The writer feels that this degree of comparability has probably been achieved at the cost of an underestimation of the total number of lesions, and further when the object is comparison between two populations the effect of a constant underestimation is constant in each group and is of minor importance.

This index can be applied very swiftly, and a minimum of equipment is required. Comparability seems to be relatively good between and within examiners (68). It is usually sensitive to differences between moderate and advanced stages of disease. It has also been used with success to make comparisons between two populations, within segments of a population and to estimate changes in status of a population over short and long spans of time (54, 68).

Findings may be reported in several ways, each amenable to
statistical analysis. The group scores present both prevalence and relative severity of disease. Prevalence can be shown on several levels, as the proportion of persons with any sign of disease, or with one or more periodontal pockets, or with one or more teeth beyond function. This index can also be used to contrast proportions of persons free of disease in two populations, and then proceed to consider the relative severity of disease in persons in whom disease was present.

The writer feels that this index is the index of choice in most of countries where other indices cannot be applied. It is easy for examiners to learn and is quite simple and does not consume much time during field investigations.

(3) **RADIOGRAPHIC INDICES OF BONE LOSS.**

When chronic destructive periodontal disease is present, the loss of alveolar bone which results is an irreversible change. Most of the population indices of alveolar bone loss are based on radiographic examination.

Marshall Day and associates (43, 44) carried out investigations by using this method. They scored resorption of alveolar bone, on a scale from zero through ten, with a grade of ten indicating complete loss of alveolar bone. A grade of five was scored when half of the alveolar support was lost, and a grade of one was scored when there was just enough bone loss to be visible in the film. Thus the average score for the areas read in the mouth was taken as the resorption figure for that person (43, 44).
Schell, Waerhaug, Lovdal and Arno (76) designed a scale where scoring of alveolar bone resorption rated as many as ten divisions. Resorption level in this situation was observed to be closer to the true level.

Miller and Seidler (56) used an index in which each quadrant in the mouth was assigned a value from zero to five, depending upon the amount of bone lost. Zero was scored when no dissolution of bone was seen; three was scored when half the bone in the quadrant had disappeared and five was scored when bone was destroyed to the apices of the roots. The index for the patient was the sum of the four quadrant scores (56). They used the sum of the scores, rather than a mean value and observed that it was necessary to limit their study to persons with 16 or more teeth in the mouth.

Sandler and Stahl (72) used a scale on which a score of zero was assigned when there was no obvious resorption of alveolar bone, a score of one was assigned for alveolar resorption not exceeding one third of the root length, a score of two for resorption greater than one third but less than one half of the root, and a score of three for resorption greater than one half the root length. The mean score for all the teeth present in the mouth was taken as the score for individual (72).

Schell, Waerhaug, Lovdal and Arno (76) observed that overlapping of images on the film and difficulties in locating the alveolar crest or cementoenamel junction, prevented measurements in about 25 percent of the bone septa, in a study of 737 adult
males in Oslo. Schei and co-workers further concluded that a difference of 10 degree in angulation of the X-ray tube, made little difference in estimation of anterior septa, but changed estimates by as much as 20 percent in the molar region. (76). They pointed out that these differences, in equipment, exposure, film angulation, film processing, might lead to differences between one study group and another, which may be misleading in the assessment of alveolar bone resorption.

Schei and his co-workers (76) in their findings also observed, that the alveolar bone resorption due to physiologic aging with advancing age may be another misleading factor. They further emphasized the need of relative measurements for persons of equivalent age for the purpose of comparisons between two populations, as the error would modify the findings with equal force.

Marshall Day and Shear (44), pointed out the limitations of radiographic indices, particularly from the prognostic viewpoint. Roentgenographic differences in alveolar bone type and texture often are of more significance than the amount of bone remaining, and the presence or absence of the normal cortical layer, frequently is more important than the relative degree of resorption of alveolar crest. (44). Frequently it is impossible to obtain a satisfactory view of the labial, buccal and lingual bone loss in roentgenograms, unless an advanced and more complicated modified technique as described by Salkind (71), is used, which is not practicable for such studies. Marshall Day,
further concluded that underestimation of bone resorption on relative measurements of interdental bone are slight and the reading error can be kept random, only so far as it increases sample (44) size, to which the writer also agrees.

Radiographic indices are permanent records and films taken years apart in time can be compared side by side and assessed by identical criteria. This avoids learning effect, where examination procedures and criteria become more skilled with accumulation of experience. X-ray films can be read in the absence of patients who may sway the examiners judgement. It is the best means of detecting periodontitis in incipient stage. When an irreversible population index is desired, the X-ray examination is the method of choice.

The main drawbacks are greater time and expense required for the assessment of each individual. It is not possible to use in field studies, in countries where electricity and transport is not in general use.

(4) **GINGIVAL RECESSION INDEX.**

This is a second irreversible lifetime measure of periodontal destruction. It was used by Stahl and Morris (86) in a study carried out for the American Army. They counted the number of teeth in which gingival recession had exposed the cemento-enamel junction, and the total teeth present in each mouth. Then they expressed these values as a percentage score of recession for an individual. The whole group score was computed as the mean percent per man.
Sandler and Stahl (72) in their study observed that this score was misleading. They examined 1299 males for bone resorption by radiographs and also for gingival recession and indicated that a considerable number of individuals showed high degree of bone resorption with low percentages of gingival recession. They did agree that these two symptoms have some relationship, but do not occur together with sufficient consistency to warrant the reliable prediction of one symptom from the other. On the other hand Russell and co-workers experience was that group gingival recession scores are closely correlated with group bone resorption scores as determined by radiographic findings. They used this index of cumulative periodontal destruction with satisfactory results. (69).

This index seems to be very simple as simple counts of the teeth with exposed cementum can readily be learnt by any examiner and examiner variations can be reduced almost to zero. It will hardly furnish any information in young populations. This index would be difficult to apply and to make counts in mouths with heavy supra gingival calculus or in betel chewers would be a problem.

(5) Ramfjord Index.

This index is based upon reversible and irreversible signs of disease, which can be recorded by its application. It has been recently proposed by Ramfjord (63) and as far as the author's knowledge goes, it has only been given trial by Ramfjord (64), who applied it in his study of periodontal status in India.
This method has added some new principles to compensate for various inadequacies of the previously mentioned systems. Procedures and criteria for this index are mandatory. Six selected teeth are examined in order to save time and effort, and to indicate the periodontal condition of the whole dentition. These are maxillary right first molar, maxillary left central incisor, maxillary left first bicuspid, mandibular left first molar, mandibular right central incisor and mandibular right first bicuspid. The area around each of these teeth is dried with cotton wool and is observed and tested for deviation from health and colour, form, density and bleeding tendency. The inflammation is scored in three degrees.

(i) a score of one for mild to moderate inflammatory gingival changes not exceeding all around the tooth.

(ii) a score to two for mild to moderately severe gingivitis extending all around the tooth, and

(iii) a score of three for severe gingivitis, characterized by marked redness, tendency to bleed and ulcerate.

This gingivitis score is the periodontal score for the teeth, if on probing the epithelial attachment is found to be on the enamel.

Then a pocket probe (number zero) is used for measuring the depth of the periodontal pockets - on mesial, buccal, distal and lingual aspects of each of these six teeth examined. When the epithelial attachment is on enamel, the depth of the crevice is noted but not used in the tooth score. If the gingival crevice
in any of the four measured areas extends apically from the cementoenamel junction, but not more than 3 m.m. (including 3 m.m. measurement) in any area then gingival score is ignored and the tooth is assigned a periodontal disease score of four. If any crevice extends to 6 m.m. (including 6 m.m.) below the cementoenamel junction, the tooth score is five, and if any extends more than 6 m.m. below the cementoenamel junction, the tooth score is six, which is the maximum on the scale. Thus the average tooth score is ten score for that person. When one or more of the specified teeth are absent then the score is based on the remainder of the teeth. The distance from the cementoenamel junction to the depth of the crevice is not measured directly. Independent measurements are made for the distance from the cementoenamel junction to the gingival margin, and for the distance from the gingival margin to the bottom of the crevice. These are then added or subtracted to arrive at the tooth score. Thus the first three grades on the Ramfjord scoring scale refer to gingivitis which is a reversible sign. The last three grades refer to the depth of the gingival crevice or of periodontal pocket, below the cementoenamel junction. This is an irreversible sign of disease.

Theoretically this index appears to be quite adequate. The procedures of examination for recording gingivitis are more detailed and it is impossible to overlook early gingivitis. Further, recording of the depth of the pocket in relation to cementoenamel junction on all the four surfaces, has been given
great consideration. It appears to be quite sensitive for recording the superficial as well as deeper conditions of the periodontium. One can measure the height of the adjacent alveolar bone according to the technique described by Ramfjord and measurements will be as informative as radiographs taken to assess the amount of bone loss and will also give a better description of its architecture. But all this will need a considerable skill on the part of the examiner. If proved successful, it will be quite useful for field studies.

One drawback appears to be the sample of the teeth selected, whether the six teeth chosen are fairly representative of the whole mouth, particularly in adults with extensive tooth loss, still remains undetermined. Further it is a very complicated index and has not been given trial by any independent investigator.

(6) **PERIODONTAL DISEASE RATE INDEX.**

This index has been suggested by Sandler and Stahl (73) and it has been called the "periodontal disease rate". It includes both reversible and irreversible signs and requires the use of both pocket probe and X-ray equipment. All the teeth and surrounding tissues are examined in a given mouth. A tooth is scored as positive if there is (a) gingival necrosis, or hypertrophy or inflammation encircling the tooth, or a purulent discharge from the gingival crevice, or (b) a gingival crevice having a depth of 3 m.m. or more or (c) tooth mobility greater than one m.m. in any direction or (d) radiographic evidence
of resorption of alveolar bone extending more than 3 m.m.
apically from the cementoenamel junction. Thus number of teeth
affected with periodontal disease is determined and computed
as the percentage of all teeth present in the mouth. This is
the score for individual and group score is the mean of the
scores for individuals comprising the group.

It is a simple method in summation, and prevalence of
disease can be expressed in easy terms.

Radiographic equipment is a handicap for field studies and
is not possible in most situations. There is also no
differentiation of the character of the lesion, its chronicity
and the degree of bone loss. Moreover gingival and bone
conditions are combined into a single figure, in which the
relationship between the two tissues is lost from sight.

(7) **GINGIVAL BONE COUNT INDEX.**

This index has been developed recently by James et al (31),
and permits differential recording of gingival and bone
conditions. An explorer is used in exacting specific pockets and
a bitewing X-ray procedure is applied for loss of bone.
Subjective measurements are made on a zero to three scale,
for gingivitis of each tooth and proportional measurements of bone
loss is made on a zero to five scale.

**Gingival Score:** If there is no deviation from normal
gingiva then a tooth is scored zero or negative. For mild
gingivitis involving free gingiva (margin and papilla) it is
scored one, for moderate gingivitis involving both free and
attached gingiva it is scored two, and for severe gingivitis with hypertrophy and easy bleeding it is scored three.

**Bone Score:** One score is assigned for each tooth studied. For no bone loss the tooth is assigned a score zero, for incipient bone loss or notching of alveolar crest it is scored one, for bone loss approximating one fourth of root length or pocket formation — one side not over one half root length, it is scored two, for bone loss approximating one half of the root length or pocket formation — one side not over three fourths root length with slight mobility it is scored three, for bone loss approximating three fourths root length or pocket formation — one side to apex with moderate mobility it is scored four, and for complete bone loss with marked mobility it is scored five. Thus maximum possible gum-bone count per person is shown by score eight, and for obtaining G.B. count in the individual the whole mouth mean scores are added.

This index seems to be useful because subjective measurements of gingival condition is combined with simple proportional measurements of bone loss in order to produce a composite score. The original components are well identified and it is more descriptive than the previously mentioned (composite indexes) and gingival and bone conditions are both combined in one.

It is recently developed index and nobody has given it adequate trials. Moreover major drawback is involvement of X-ray equipment, which is not possible in most situations.
(8) **ORAL HYGIENE INDEX.**

It is necessary to describe briefly the role of the oral-hygiene index, because the relation between periodontal disease and calculus or oral debris is so marked and uniform that no consideration of other factors possibly associated can be undertaken unless the effect of these conditions can be held constant in some manner.

The oral hygiene index was developed by Green et al (29) and covers all the teeth fully erupted in the given mouth, and is based on simple clearly defined criteria which reduce examiner variations to a minimum.

This index is composed of the combined 'Debris Index' and 'Calculus Index', and each in turn is based on 12 numerical determinations, representing the amounts of debris or calculus present on the buccal and lingual surfaces of each of three segments of each dental arch, (1) the segment distal to the right cuspid, (2) the segment distal to the left cuspid and (3) the segment mesial to the right and left first bicuspid.

Separate recordings are made for the buccal and lingual surfaces to differentiate oral hygiene status between these surfaces.

Examination is carried out in the prescribed fashion - by using an explorer. Firstly the buccal and the lingual surfaces in the upper right posterior segment, secondly in the upper anterior segment, and thirdly in the upper left posterior segment, and then the lower arch inspection proceeds in the same manner, but from left to right. If there is no presence of
debris stain, a score of zero is assigned, if soft debris covers not more than one third of the tooth surface or the presence of extrinsic stains without other debris regardless of surface area covered, it is assigned a score of one. If soft debris covers more than one third, but not more than two thirds of the exposed tooth surface a score of two is assigned and finally when soft debris covers more than two thirds of exposed tooth a score of three is assigned.

Calculus is scored on the same scale. Zero is scored if no calculus is present. A score of one is assigned for supra gingival calculus covering not more than one third of the tooth surface; a score of two for supra gingival calculus covering between one third and two thirds of the surface, or for the presence of individual flecks of subgingival calculus, and a score of three for supra gingival calculus covering more than two thirds of the tooth surface or for a continuous heavy band of subgingival calculus around the cervical portion of the tooth.

Score for the individual is the average score for the segments examined. It can be calculated separately for debris or for calculus, or as the composite of the two.

The routine procedures used by Ramfjord (63) includes a series of indices which are supplementary to his periodontal disease score. Occlusal and incisial attrition, tooth mobility, lack of contact, plaques and calculus are scored on specified scales running from zero through three, corresponding in general to the clinical description of none, mild, moderate
and severe.

The oral hygiene index suggested by Greene et al (29) is a useful tool when assessing tooth brushing efficiency, when evaluating the dental health practices of a community and the immediate as well as long term effects of dental health education procedures.
IV. CORRELATION OF CLINICAL AND CLINICAL AND RADIOLOGICAL
FINDINGS.

Periodontal disease includes a multiplicity of diseases affecting different tissues of the periodontium. Many indices have been devised to assess its severity in populations and some popular ones have been described by the writer in this paper. Some of these are based on clinical signs of disease and have been applied for soft tissue involvement, clinically visible. Others are based on total tissue damage or bone loss, and have required the use of radiographic examination. But a few are based on clinical as well as radiographic examination and present the status of the soft as well as hard tissue involvement. Any of these indices will provide useful information when applied in appropriate situations. So far no one has shown the correlation of these indices in periodontal disease by applying all of them in one particular situation. This needs further investigation to assess the superiority for one, out of all the indices so it may be applied in field studies where radiographic examination is not possible.

The criteria of different indices described in this paper, are being presented by the writer in a tabular form to show their theoretical correlation in Table I.
<table>
<thead>
<tr>
<th>Dental Caries</th>
<th>Damage to Periapical Tissues</th>
<th>Periodontal Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulpal Exposure</td>
<td>Permanent Root Formation</td>
<td>gingival recession</td>
</tr>
<tr>
<td>Loss of Attachment</td>
<td>Root Resorption</td>
<td>periodontal abscess</td>
</tr>
<tr>
<td>Erosion of Enamel</td>
<td>Alveolar Bone Loss</td>
<td>periodontal pocket</td>
</tr>
<tr>
<td>Caries Progression</td>
<td>Alveolar Bone Loss</td>
<td>periodontal defect</td>
</tr>
</tbody>
</table>

**Caries Severity**
- (a) Dental Caries involving only the tooth
- (b) Caries involving the tooth and surrounding gingival tissue
- (c) Caries involving the tooth, surrounding gingival tissue, and bone

**Damage to Periapical Tissues**
- (a) Periapical Tissue Damage
- (b) Periapical Tissue Pathology
- (c) Periapical Tissue Infection

**Periodontal Disease**
- (a) Gingival Recession
- (b) Periodontal Abscess
- (c) Periodontal Pocket

**Key Concepts**
- Caries progression affects tooth structure.
- Periapical tissue damage indicates infection.
- Periodontal disease impacts bone and soft tissue.
The close relationship of various findings by different investigators using different methods showing some correlation with each other will also be presented.

(1) **Gingivitis and Calculus: (Correlation)**

Ramfjord (64) in a study carried out in India (11 to 17 years of age) using his own Index, observed that there is a gradual increase in the amount of calculus with increasing age. He compared the calculus scores with gingivitis scores and concluded that an obvious correlation existed between the two conditions; that with the increase of calculus score, gingivitis score also increased as shown in table 2.

**TABLE 2.**

**Correlation Between Calculus and Gingivitis**

<table>
<thead>
<tr>
<th>Age</th>
<th>Urban and Rural (India)</th>
<th>Mean Calculus Score</th>
<th>Mean Gingivitis Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>11 yrs.</td>
<td></td>
<td>.99</td>
<td>1.40</td>
</tr>
<tr>
<td>13 yrs.</td>
<td></td>
<td>1.25</td>
<td>1.49</td>
</tr>
<tr>
<td>15 yrs.</td>
<td></td>
<td>1.48</td>
<td>1.53</td>
</tr>
<tr>
<td>17 yrs.</td>
<td></td>
<td>1.53</td>
<td>1.50</td>
</tr>
</tbody>
</table>

(2) **Periodontal Disease and Oral Hygiene:**

In a study carried out by Greene (30) in India, the conditions of periodontal structure was recorded using Russell's method and oral hygiene status using Greene Vermillion (29) method. He observed that periodontal disease score increases with each increase in the calculus and debris score.
and oral hygiene index. Calculus score increases at a more rapid rate than the debris score, and there is a sharp rise in the oral hygiene index with each increase in periodontal score. This presents a close correlation between periodontal index score and oral hygiene index score as shown in fig. 1.

Greene used the same indices for recording periodontal score and oral hygiene score, in a study carried out in Atlanta. He compared it with the India study and he observed similar correlation between the scores and this is shown in fig. 2. He further noted that periodontal disease scores were significantly higher in India than Atlanta, when oral hygiene index was between 2.1 and 5.0.

Other groups were too small for valid comparisons. This difference may be due to either the inflammatory response to debris and calculus in Indian people being greater than in Atlanta, or that there are other important irritants or altering factors active in India which are not active in the Atlanta study group.

Marshall Day et al. (43) also observed the same striking relationship between the oral hygiene score and periodontal score. They also pointed out that clinically demonstrable calculus was markedly increased at 31 years of age, reaching a peak of 91 percent at 56–59 years. They also noted that after 50 years of age a greater proportion of subjects exhibited both supra gingival and subgingival calculus. They also observed a gradual increasing trend of periodontal disease
among the same subjects, 24 percent at the age of 19 years, 90 percent at the age of 31 years, and 100 percent at the age of 56 years and over. They concluded that as the oral hygiene score rises, there is a rise in periodontal disease score.

Lovdahl and co-workers (55) had similar findings, while carrying out a study of employees in an industrial concern in Oslo. They also noticed a very close correlation between oral hygiene and periodontal disease (pocket formation and mobility).

All these findings (also figures 1 and 2) show that the scores of debris and periodontal disease, calculus and periodontal disease, and oral hygiene and periodontal disease are significantly correlated.
FIGURE 1

Mean Oral Hygiene Scores Compared with Periodontal Scores. (11 to 17 Years of Age, Urban-Rural India)\textsuperscript{30}

FIGURE 2

Oral Hygiene Index Compared with Periodontal Score (11 to 17 Years of Age, India-Atlanta)\textsuperscript{30}
(3) Correlation of pocket formation, tooth mobility and suppuration with periodontal diseases.

Marshall Day and his co-workers (43) examined for pocket formation, mobility and suppuration in recording periodontal disease incidence. They observed constantly increasing incidence of pocket formation in all age groups, highest being recorded in the age group 52-55 years. Tooth mobility incidence increased sharply from 25 percent at 35-39 years to 49 percent at age 40-48 years, with a steady increase to 75 percent at age 60 years. Further, almost 40 percent of all subjects showed suppuration at age 40 years as did approximately 50 percent in the older age group. This is shown in fig. 5, and it is clear that there is a relationship existing between pocket formation, mobility of teeth and suppuration. If fig. 5 is compared with fig. 3, it will present certain correlation in between periodontal disease, pocket formation, mobility and suppuration.
PREVALENCE OF PERIODONTAL DISEASE
(13 to 60 years of age, U.S.A.)

POCKET FORMATION, MOBILITY AND SUPPURATION
(PERCENT PERSONS AFFECTED BY AGE)
Gingivitis and Gingival Recession.

Sandler et al (72) in their observations noted that gingival inflammation and gingival recession are directly related as shown in fig. 8. He also pointed out that it was uncommon to find persons with few inflamed gingiva and many receded gingiva and vice versa. They also observed a statistical relationship between the occurrence of inflammation of the interdental papillae and inflammation of the marginal gingivae, which was found to be very close as shown in fig. 9. This shows that when a large proportion of gingival papillae are inflamed then a large proportion of gingival margins are likely to be inflamed and when few of either are inflamed, then few of the other will be inflamed.
FIGURE 8

% of Teeth Affected by Inflammation of Gingival Papilla

% of teeth affected by gingival recession

RELATIONSHIP BETWEEN INFLAMED GINGIVAL PAPILLAE AND GINGIVAL RECESSSION

FIGURE 9

% Teeth Affected Inflammation Gingival Papillae

% of Teeth Affected by Inflammation of Marginal Gingiva

RELATIONSHIP INFLAMED PAPILLA AND MARGINAL GINGIVA
(5) Periodontal pocket formation and gingival recession.

Ramfjord (64) in his findings amongst Indian subjects noted that there was a close correlation between periodontal disease and gingival recession as shown in table 3. He concluded that with increase of periodontal disease an increase of gingival recession takes place.

<table>
<thead>
<tr>
<th>Age</th>
<th>Number Examined</th>
<th>Persons with Periodontal Pockets</th>
<th>Persons with Recession</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Number</td>
<td>Percent</td>
</tr>
<tr>
<td>11</td>
<td>542</td>
<td>4</td>
<td>0.7</td>
</tr>
<tr>
<td>13</td>
<td>464</td>
<td>3</td>
<td>0.6</td>
</tr>
<tr>
<td>15</td>
<td>392</td>
<td>8</td>
<td>2.0</td>
</tr>
<tr>
<td>17</td>
<td>217</td>
<td>20</td>
<td>9.2</td>
</tr>
<tr>
<td>19–22 (mean 20.6)–19</td>
<td>6</td>
<td>31.6</td>
<td>3</td>
</tr>
<tr>
<td>23–26 (mean 24)–27</td>
<td>13</td>
<td>48.0</td>
<td>7</td>
</tr>
<tr>
<td>27–30 (mean 28.3)–13</td>
<td>6</td>
<td>46.2</td>
<td>6</td>
</tr>
</tbody>
</table>

(6) Correlation between Oral Hygiene and Alveolar Bone Loss.

Schei (76) and his co-workers in a systemic investigation observed a relationship between the efficiency of oral hygiene and loss of alveolar bone, by using X-ray films for detecting
bone loss, and clinical observation for oral hygiene status; in a group of industrial workers and staff in Oslo. They compared bone resorption measurements for men with good, with fairly good and with poor efficiency in tooth brushing (as indicated by presence or absence of debris in all or any parts of the mouth) and observed that men with good efficiency had by far the least degree of bone resorption, that men with fairly good efficiency had lost an intermediate degree of bone, and that men whose brushing efficiency was not good had lost by far the greatest amount of bone, with differences between the groups progressively more marked as age groups were considered. This finding shows that there is some correlation between oral hygiene status and bone resorption also.

(7) Clinically Observed Periodontal disease and Radiographically detected bone loss.

In a study carried out by Marshall Day et al (43) the clinical method used showed that periodontal disease was rarely found between 13 and 18 years of age, but the incidence increased rapidly from 24 to 69 percent between ages of 19 and 26 years. Thereafter the incidence steadily increased until 45 years of age when almost all the subjects showed some evidence of periodontal disease as shown in fig. 3. Then the same subjects were examined radiographically. They observed that the highest resorption of figure of X-ray index score in age group 13-15 years was 2. Further, the highest resorption figure met between the age of 16 and 22 was 3. Then there was
a sharp rise to 6 in the 23 - 26 years of age group, and increased gradually until age 35-39 years as shown in fig. 4. In the same figure comparisons are also made with North Indian subjects, who were examined by Marshall Day, using same technique.

From results in figures 3 and 4 it is obvious that periodontal disease has its incipiency at an early age and then progresses more rapidly with a greater degree of bone loss in all age groups. There is a reasonable correlation existing between periodontal disease observed clinically and alveolar bone resorption detected radiographically as shown by comparing results in fig. 3 and 4.
FIGURE 4

BONE RESORPTION
(13 to 60 YEARS OF AGE, U.S.A. AND INDIA)
(8) **Gingival Recession and degree of alveolar resorption.**

Recession of gingiva and resorption of alveolar bone are closely associated as shown by Sandler and co-workers (72). They also pointed out that these findings do not occur together with sufficient consistency to warrant the reliable prediction of one symptom from the other as shown in fig. 6.

Marshall Day (43) and co-workers study of total recession of gums without pocket formation as shown in fig. 7, when compared with bone resorption figures of the same subjects (fig. 4), also shows a fairly good correlation though there is no consistent pattern.
FIGURE 6

RELATION BETWEEN MEAN SCORES FOR BONE LOSS AND MEAN SCORES FOR GINGIVAL RECESSION

Degree of Alveolar Bone Resorption

Percentage of Teeth Affected by Gingival Recession
TOTAL RECESSION COMPARED WITH
BONE RESORPTION (U.S.A.)

TOTAL RECESSION COMPARED WITH
BONE RESORPTION (U.S.A.)

TOTAL RECESSION COMPARED WITH
BONE RESORPTION (U.S.A.)
(9) Correlation between Clinical and Clinical plus Radiological findings by Russell.

This study supports the potential value of this index and was carried out by Russell (68), who tested his method of scoring for underestimation by a clinical study as well as by clinical plus radiographic method. He examined 200 subjects by using his own method, which has been previously described, and recorded periodontal scores.

Then radiographic films were secured for each patient. Final diagnosis was made by him after study of the patient's clinical records and the dried mounted X-ray films, with the aid of the notes made at the time of examination which described the colour, texture, tone and general status of the gingival tissues.

The effect of this procedure disclosed that there was a general upward revision at all levels of disease and this had also raised the mean scores for the group as a whole from 1.20 to 1.45 as shown in details in table 4. These two methods used, showed a high degree of correlation between the scores, and the relationship was approximately a straight line as shown in fig. 10.
CORRELATION OF VISUAL AND VISUAL PLUS X-RAY PERIODONTAL SCORES
It is obvious from these given facts that similar findings will be returned whether radiographs are used or whether the estimate is based upon the field examination alone.

TABLE 4.

(The Relationship between periodontal scores assessed by visual examination alone, and by visual examination supplemented by X-ray in 200 patients seen in the Dental Clinic of the Clinical Centre at the National Institute of Health, Bethesda, Maryland).

<table>
<thead>
<tr>
<th>Range of Periodontal Scores Visual alone (clinical)</th>
<th>Number of persons Examined</th>
<th>Mean Scores</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Visual alone (clinical)</td>
</tr>
<tr>
<td>Zero</td>
<td>77</td>
<td>Zero</td>
</tr>
<tr>
<td>.1 - .3</td>
<td>34</td>
<td>.20</td>
</tr>
<tr>
<td>.4 - .9</td>
<td>26</td>
<td>.63</td>
</tr>
<tr>
<td>1.0 - 1.9</td>
<td>23</td>
<td>1.33</td>
</tr>
<tr>
<td>2.0 - 4.9</td>
<td>24</td>
<td>3.41</td>
</tr>
<tr>
<td>5.0 - 8.0</td>
<td>16</td>
<td>6.54</td>
</tr>
<tr>
<td>All Persons Examined</td>
<td>200</td>
<td>1.20</td>
</tr>
</tbody>
</table>

A study was carried out by Zimmerman (92) et al, where 110 long term residents of Bartlett and Cameron, Texas, were examined clinically (Russell's method) as well as radiographically
Mean periodontal scores for persons with no bone resorption and with mild, moderate or severe bone resorption recorded are shown in table 5. He showed that the relationship was quite striking as the periodontal score increased with the increase of bone resorption.

On the basis of these two cited studies it is concluded that the field method of scoring suggested by Russell is quite adequate to determine the relative periodontal status of two groups or populations.

**TABLE 5.**

Relationship between periodontal scores and Bone loss.

<table>
<thead>
<tr>
<th>Bone resorption and X-ray</th>
<th>Number of persons</th>
<th>Mean periodontal scores by field method</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>11</td>
<td>.89</td>
</tr>
<tr>
<td>Mild</td>
<td>51</td>
<td>1.14</td>
</tr>
<tr>
<td>Moderate</td>
<td>34</td>
<td>2.13</td>
</tr>
<tr>
<td>Severe</td>
<td>14</td>
<td>4.81</td>
</tr>
</tbody>
</table>
V. **Discussion.**

The main objective of a periodontal index is to enable comparison between population groups or sub-groups for the purpose of determining extent and severity of periodontal disease or as a basis for development and expansion of dental education, public health, and dental practice. It gives an idea as to the number of trained personnel required, the cost of services involved, and also is useful in stimulating and maintaining public awareness regarding their dental health. It is a very useful tool in keeping the Government informed concerning the need and progress of dental health programmes. It is also very useful aid in finding out the relationship between the periodontal disease and the systemic diseases and in their preventive and control measures.

Any of the described methods appear to be quite useful in providing information, if applied in appropriate situations by skilled workers. The choice of an index depends upon the nature of information required, the number of persons to be surveyed, the amount of time at hand and the type and amount of equipment available in that situation.

P.M.A. index is more useful in young population, where all the indices are measures of simple gingivitis, and it is very simple to apply in primitive conditions. When separation of individuals with complete freedom from disease is desired, P.M.A. index is the best of the methods described. When hand instruments like probe mouth mirror and chip blower are
available with minimal provision for sterilization—P.I. (Periodontal Index), Ramfjord Index, or Gingival Recession Index can be applied. When a real distinction between moderate and severe stage of disease is required P.I. (Periodontal Index) will prove to be more appropriate.

Radiographic Indices are the best means of detecting periodontitis in incipient stages, but gingival condition cannot be recorded. The count of teeth involved in gingival recession is least sensitive to incipient disease. The count of overt pockets in P.I. is less sensitive than radiographic examination for the detection of incipient stage and the sensitivity of incipient disease by Ramfjord Index probably cannot be determined.

The author feels that Periodontal Index (Russell's) is very simple in its application and presents good comparability of the findings for determination of relative group prevalence and is quite adequate for use in field studies with minimum problems.

The criteria of different indices as compared in table 1 appear to be very similar to each other, even though some describe the clinical condition of the soft tissue while others state the condition of the hard tissue visually or radiographically detected. Each of these indices should have a good correlation to the criteria of periodontal conditions chosen although all have not received independent trials. There is good correlation between criteria described by clinical and
radiographic methods and this has been shown by Russell as previously discussed. Further there is definite correlation shown between different indices by the direct correlation that they show to age and bone loss, oral hygiene and periodontal disease. However, the direct correlation of individual indices, one to another, has not been tested by clinical trials and a clinical trial of all indices on the same group would be needed to show superiority of an index in its correlation to periodontal disease. Further investigation is needed in this field.
VI. SUMMARY

Normal state of the periodontium and general classification of its disease, with clinical pathology and radiographic appearance have been described. Distribution of periodontal disease in the group population has been presented, and its epidemiological characteristics shown. Indices for scoring its severity have been described and evaluated. Theoretical correlation of the criteria of these indices has been shown and the close relationship of clinical and clinical plus radiographical findings has been presented. The further need of investigation in this field to assess the superiority of one index over the other has been emphasized.
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