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A Comparison of Crevicular Fluid Volume: Pre and Post Periodontal Surgery

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A COMPARISON OF CREVICULAR FLUID VOLUME:
PRE AND POST PERIODONTAL SURGERY

by

David L. Koth, B.S., D.D.S.

A Thesis Submitted to the Faculty of the Graduate School
of Loyola University in Partial Fulfillment of
the Requirements for the Degree of
Master of Science

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VITAE

David L. Koth was born in Kalamazoo, Michigan on September 17, 1934. Elementary and secondary education took place in Kalamazoo where he was graduated from Kalamazoo Central High School in June 1952.

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CHAPTER I

INTRODUCTION AND STATEMENT OF PURPOSE

An optimal time for commencement of restorative procedures after periodontal treatment has been an elusive object. Studies have been done attempting a valid and objective answer (Waerhaug, 1960; Ramfjord and Costich, 1968; Suppipat, 1976). These studies have contributed greatly to our knowledge on the subject. However, they have been lacking two important ingredients: longitudinality and objectivity.

This project was undertaken to quantitatively evaluate the progress of periodontal wound healing.

An attempt was made to find whether periodontal health could be maintained over a period of time by quantitating crevicular fluid volume.

Finally, tissue sections were examined microscopically to determine whether a relationship existed between the morphologic gingival tissue content and the degree, intensity, or stage of inflammation.

Most studies on periodontal wound healing have been qualitative. Numerical values have been assigned to these qualities and in some instances these numbers have been used for quantitative statistical analysis. On closer

examination, however, it can be deduced these were truly dichotomous scales, unsuitable for quantitative analyses and not always reproducible.

The relationship between the presence of gingival crevicular fluid and inflammation is generally accepted (Brill, 1960; Egelberg, 1964; Daneshmand and Wade, 1975; Golub and Kleinberg, 1976). This association is evidenced as a positive correlation between the volume of crevicular fluid and the degree of inflammation. Crevicular fluid measurements have been done in various ways; the correlation between these various methods and the degree of inflammation has been previously established. The recent development of a fluid measuring device, "Periotron"*, has presented an opportunity to quantify crevicular fluid volumes in a relatively simple and reliable manner.

*"Periotron" is the trade name assigned to the crevicular fluid measuring device utilized in this research. The instrument was developed and is distributed by Harco Electronics Ltd., Winnipeg, Canada. Throughout this paper, excepting the literature review, this will be the reference for the noun "Periotron".

CHAPTER II

REVIEW OF THE LITERATURE

INTRODUCTION

The credibility of a direct positive correlation between the volume of crevicular fluid and gingival inflammation is doubted by few. Whether this fluid represents inflammatory exudate or transudate which may be altered in the presence of inflammation, remains a subject of some debate. This dubiety exists in part because of lack of knowledge regarding the contents, origin, and mechanism of movement of crevicular fluid.

The direct association between an increase in inflammation and artificial crowns is well established (Waerhaug, 1960; Larato, 1975; Jameson, 1976). However, to what degree this inflammation is affected by crown contour and/or margin placement remains controvertible (Eissman, 1971; Yuodelis, 1973; Newcomb, 1974). Whether or not there may be an intrinsic factor relating to tissue response (such as a predisposition for periodontal disease) remains an area of scant research.

Histologic studies have been done in numbers and their correlation with clinical parameters of inflammation assessed (Oliver, 1969; Rudin, 1970; Daneshmand and Wade,

1975). In general there is correlation, although the degree of relationship seems to depend on which parameters of each are compared.

CREVICULAR FLUID

Historical Perspective

Presence of fluid in the gingival crevice was described as early as 1817 (Serres). The occurrence of crevicular fluid was postulated to be due to a secretion by "gingival glands". Occurrence of crevicular fluid was substantiated by histological examination later in the nineteenth century by Black (1887). At this time the appearance of fluid was still attributed to glandular secretion; however, it was postulated that origination was from gingival glandular tissue in a plexus clustered among the principal fibers of the periodontal membrane.

During the second decade of the twentieth century the presence of this fluid was widely accepted. The function of the gingival crevicular fluid was postulated to be; facilitation to the formation of subgingival calculus (Black, 1920), a solution which became acidic (therefore tissue destructive) as a result of traumatic occlusion (McCall, 1924), and a serous secretion which aided in cleansing the enamel surface which it circumscribed.

Studies by Bodecker (1931) failed to confirm presence of gingival glands previously described by Black (1899).

Bodecker later (1933) supported Mc Calls theory (1924) and utilized litmus paper in an attempt to correlate erosion and the acidity of crevicular fluid. Later the term crevicular exudate was defined (Miller, 1938). Change in this exudate was presumed to be a clinical sign of incipient periodontal disease. Kronfeld's text (1933) exemplified dentistry's studies of crevicular fluid at that time.

Intensive study of the composition and flow of gingival crevicular fluid, particularly in response to gingival and periodontal disease, originated from Waerhaug's basic studies (Waerhaug and Steen, 1952; Waerhaug, 1955). In these studies he demonstrated gingival fluid originated from serum and was the medium in which subgingival calculus forms. Since then, knowledge has expanded substantially, meriting an extensive review (Cimasoni, 1974) and research efforts which have resulted in the development of diagnostic techniques allowing a more scientific approach to the care of the periodontal structures and the dental patient.

Formation of Gingival Crevicular Fluid and its Relationship to Inflammation

To discover whether gingival crevicular fluid is an inflammatory exudate or a serum transudate which becomes an inflammatory exudate only in the presence of inflammation has been the purpose of many investigations.

In 1952, Waerhaug demonstrated the dynamic nature

of the gingival sulcus by placing India ink in healthy sulci of young dogs. Subsequent examination of the gingival fluid showed emigration of leukocytes through the sulcular epithelium along with an increased transudation of fluid. This led to the conclusion: "In healthy pockets, where a normal epithelial cuff is closely fitted around the tooth to the gingival margin, the secretion is rather minute." The same investigator (Waerhaug and Steen, 1952) introduced pure cultures of pathogenic bacteria into bacteria free gingival crevices of dogs; histologic tissue reaction was noted over a forty-eight hour period. This resulted in the conclusions: Fluid was always present in the gingival crevice; Healthy, calculus free gingival crevices were sterile; and, Bacteria in the crevice caused necrosis of the epithelium and inflammation of the connective tissues which lead to the subsequent exudate formation.

Experiments by Brill (1959 a,b,c) on dogs and humans tended to illustrate continual presence of fluid as a transudate and inflammatory exudate. Injecting dogs with Evans' blue (which binds to plasma albumin and gamma-globulin), fluid flow in the sulcular crevice was assessed as to the effect of histamine, mechanical stimulation, and inflammatory state. The conclusions were: Evans' blue from plasma passed through capillary walls in small amounts and appeared in the gingival crevice as the result of mechanical

stimulation, intravenous injection of histamine, and inflammation. In another experiment (Brill, 1959b), human subjects chewed paraffin. This resulted in increased gingival crevicular fluid flow. It was further noted both stimulated and non-stimulated crevicular fluid contained amino acids. This led to the postulation: gingival stimulation created pressures on the gingival vascular bed which caused escape of plasma. This may have enhanced the antimicrobial substances. Using standardized charcoal particles, Brill (1959c) showed the flow of gingival crevicular fluid was able to remove particulate matter, including bacteria (postulation), from gingival pockets.

The bacteriological contents of gingival crevicular fluid in clinically healthy patients has been studied (Gavin and Collins, 1961a). The results of this study disputed Waerhaug and Steen's theory of a sterile, healthy crevice, by demonstrating microorganisms in a majority of cases. Subsequently the same investigators (Gavin and Collins, 1961a) found fluid from clinically healthy patients to demonstrate no bacteriocidal or bacteriostatic effect on various strains of oral bacteria.

Harvey (1962) placed standardized silver alloy particles in clinically healthy gingival crevices of dogs and humans. The results of his study substantiated the transudation theory: normal gingival crevices maintained

their health by constant flushing with tissue fluid; this fluid flow increased in the presence of inflammation.

Gingival fluid was collected from human subjects via filter paper strips after the subjects were given fluorescein orally (Mann, 1963). This study demonstrated continuous fluid flow. The rate of flow increased as inflammation became more severe.

Other investigations have indicated gingival crevicular fluid was a function of inflammation. As such it was not present in the form of a transudate in healthy gingival crevices. Loe (1961) studied the turnover rate of epithelial cells and the presence of leukocytes in the crevicular fluid of dogs. In this study the gingival crevices were sealed from saliva. He found, by histological examination, support for the theory of constant renewal of the epithelial cuff. He observed neutrophilic leukocytes in the sulcus and reasoned, "they migrate through the epithelial lining under physiologic conditions" and "there is a continuous transudation of tissue fluid into the clinically normal gingival pockets."

Weinstein and others (1967), using chemical, immunochemical, immunological, and electron microscopic techniques demonstrated and characterized the existence of fluid in clinically normal gingival sulci in humans. Their divergence from the findings of Loe, et al. (1965) was ex-

plained as a difference in criteria for "clinically normal" and uncontrolled differences in technique. The authors stated their technique (utilizing fluorescein labeling) was one hundred times more sensitive detecting proteins than the ninhydrin technique employed by Loe.

A study involving microcirculation of healthy periodontium in dogs, injecting one percent of Patent Blue V in one carotid artery and Pelikan carbon black in the other was carried out (Turner, et al., 1969). Patent Blue V passed quickly through the epithelial attachment and the sulcular epithelium. This indicated to these investigators the existence of fluid as a transudate. The Pelikan carbon black (200 to 500 A) did not pass through intact capillary walls. The authors concluded there may have been a "free" flow of fluid through the gingiva and sulcular epithelium. Further study should determine how this "transudate" is transmitted through the complex network and the mediating influence on the process.

Recent studies, employing the use of a quantitating device (Harco), have shown there usually was a varying amount of fluid in clinically normal gingiva in human subjects (Jameson, 1976).

In a later study (Loe, et al., 1965) crevicular fluid from 118 adults was measured by the extra crevicular technique and an intracrevicular technique. Using biomicroscopic techniques they noted capillary compression when

inserting filter paper strips into the crevice (Munktel No. 3 filter paper strip, 1.5mm. wide X at least 10mm. long). They suggested this may have been the reason for finding fluid in healthy gingival crevices. Their conclusions were contrary to earlier reports (Brill, 1959b; Brill and Kraase, 1959; Brill, 1962; Loe, 1962). Crevices of healthy human gingiva did not exhibit fluid flow. Mechanical stimulation of the periodontium did not produce fluid from such crevices. Crevicular fluid flow began prior to appearance of clinical changes in the gingiva and persisted for some time after the clinical changes had disappeared. Gingival fluid was considered an inflammatory exudate and the detection of gingival crevicular fluid represented a definite clinical criterion in a refined distinction between normal and inflamed tissue.

The exudate theory was further supported by Oliver, et al., (1969) in a study involving 53 patients. The authors investigated the relationship between a gingival index (Loe and Silness, 1967), gingival fluid measurements, and histological inflammatory cell density. The results showed strong positive correlation between gingival fluid and the gingival index. They further concluded: in patients with "no clinical evidence of gingival inflammation there is not exudate in the vast majority of crevices."

Flame photometry was used to determine the relative

proportions of sodium, potassium, and calcium (Krasse and Egelberg, 1962). The findings indicated an increase in the proportion of potassium from clinically healthy gingiva. They supposed this to be intra-cellular potassium augmenting the extra-cellular fluid as it passed into the gingival crevice. Their conclusion was "gingival pocket fluid can not be regarded as a simple filtration product, but rather as an inflammatory exudate."

Egelberg (1963a) compared the cellular content of gingival crevicular fluid from healthy gingiva with fluid collected from chronically inflamed gingiva. He observed no difference in cell types (epithelial cells, neutrophilic leukocytes, lymphocytes, and bacteria). There was, however, an increase in the number of inflammatory cells, compared to epithelial cells, in fluid samples from inflamed gingiva. These findings seemed to support his earlier research with Krasse (1962) and supported the theory, "fluid in healthy pockets may be regarded as an inflammatory exudate."

Further investigations by the same author (Egelberg, 1966) led to the conclusion fluid discovered in healthy gingiva was an iatrogenically introduced artifact, resulting from irritation to the epithelium induced by insertion of filter paper strips into the gingival crevice.

In an attempt to explain opposing experimental results, describing gingival crevicular fluid either as a physiological transudate or a pathological inflammatory exudate,

Alfano (1974) discussed a theoretical model of fluid transport. His explanation was the origination of gingival fluid via two distinctly separate mechanisms: the generation of a standing osmotic gradient generated by macromolecular by-products of the bacteria present in subgingival dental plaque; and, initiation of a classical inflammatory response. This led to the conclusion, "at various times or in different areas of the mouth, gingival fluid may progress from an initial osmotically modulated to a secondary inflammatory exudate, with consequent alterations in its composition."

Golub and Kleinberg (1976) reviewed the literature regarding crevicular fluid and explored its implications to clinical periodontal therapy. They basically found support for Alfano's (1974) theory: crevicular fluid was modulated by macromolecules generated by dental plaque by-products. They proposed, however, that instead of creating an osmotic gradient, these macromolecules allowed fluid escape into the crevice by directly altering the crevicular epithelium and connective tissue cells. They suggested further investigation directed toward the role of nonionized ammonia (alkaline pH of crevices favored formation of this compound; Kleinberg and Hall, 1969) and urea in gingivitis (Golub, et al., 1971) gingival crevicular urea could serve as a substrate for ammonia formation. The conclusion was: monitor-

ing gingival crevicular fluid flow could 1) detect subclinical gingival pathology, 2) enable quantification of the severity of inflammation, and 3) monitor the response of the gingiva to periodontal therapy. They further suggested collecting gingival fluid on filter paper strips could lead to development of screening tests for systemic diseases if appropriate biochemical and microbiological analysis could be developed.

Squier (1975) discussed the permeability of oral mucous membranes. He suggested diffusion as the appropriate description of movement through oral epithelium. He also suggested oral epithelium might be described as "leaky". The rate of penetration was directly proportional to the concentration of the penetrant. He stated inflammation was a major cause of increased permeability. He also suggested some factors from plaque may have damaged the epithelium initially, allowing enzymes, toxins and antigens to enter the host tissue which caused an immune inflammatory response.

Gingival Crevicular Fluid and its Relationship to Inflammation

The dental literature contained continuously accumulating evidence which demonstrated a positive correlation between the quantity of crevicular fluid and the severity of gingival inflammation.

Fluorescein was used (orally administered) by Brill and Bjorn (1959) in studies of the permeability of human crevicular epithelium, nasal epithelium, and oral epithelium. Their results showed the epithelial lining of the gingival crevice and nasal mucosa were permeable to fluorescein molecules. They noted a correlation between the degree of inflammation and the quantity of fluorescein recovered on filter strips placed in the gingival crevice. More fluorescein was recovered in patients with extensive restorations than in patients with normal healthy gingiva.

In another study, Brill (1959a), using protein bound Evans blue dye found it (the dye) was recovered in the presence of inflammation, mechanical stimulation and histamine injection.

Harvey (1962) suggested crevices are "flushed" with fluid and the amount of fluid was increased as a result of an acute inflammatory response to inflammation.

Another study using fluorescein was performed (Mann, 1963) which supported Brill's (1959) contention that crevicular fluid flow increased as inflammation of the marginal gingiva increased. This contention was further supported by additional studies (Egelberg, 1964; Loe and Holm-Pederson, 1965).

Oliver, et al. (1969) further supported correlation between gingival fluid flow and inflammation according

to a gingival index system (Loe and Silness, 1963).

In an experiment regarding carbohydrate components of the gingival exudate (Hara and Loe, 1969), the authors corroborated previous studies. They noted gingival exudate increased in volume as the severity of gingival inflammation increased.

Shern, et al. (1974), studying crevicular fluid flow and cytopathology, assumed the accuracy of gingival fluid volume as an indicator of local inflammation.

Daneshmand and Wade (1975), using a ninhydrin technique and the orifice method of collection, found moderate correlation between gingival crevicular fluid volume and a gingival index (Loe and Silness, 1963).

In an experiment to determine the relationship of gingival pocket depth and crevicular fluid flow (Suppipat, 1976), a trend towards positive correlation between fluid volume and the intensity of inflammation was demonstrated.

Methods of Measurement and Collection of Gingival Crevicular Fluid Flow

Calibrated micro-capillary tubes have been used to collect gingival crevicular fluid (Kaslick, et al., 1970; Krasse and Egelberg, 1962; Shillitoe and Lehner, 1972). This method allowed direct collection; however, most investigators have found it an impractical method because collection times of 10 to 15 minutes were necessary for moderate to severely

inflamed tissues (Golub and Kleinberg, 1976).

Weinstein, et al. (1967) used pre-weighed twisted thread. The thread was inserted into the crevice and the volume of gingival crevicular fluid determined by weighing the sample after removal. This also was found to be unsatisfactory because of the long time required for collection (10 minutes). Evaporation during weighing provided further inaccuracies (Golub and Kleinberg, 1976).

The most widely used method for collection of gingival crevicular fluid was collection onto a filter paper strip placed into or near the orifice.

The sample of fluid has been stained with ninhydrin and measured by gauging optically (Egelberg, 1964; Oliver, et al., 1969). It has also been stained by the administration of fluorescein systemically and quantitated optometrically (Brill, 1959a; Weinstein, et al., 1967).

Others have viewed the wetted strip with no stain under a microscope fitted with a net reticle (Golub, et al., 1971; Egelberg and Attstrom, 1973).

More recently the fluid has been quantitated electronically.

Golub and Kleinberg (1976) described gingival crevicular fluid as a diagnostic aid in managing the periodontal patient. Their review discussed the various aspects of the crevicular fluid. They recommended the use of "a newly

developed gingival crevicular fluid meter (Harco Electronics, Winnipeg, Canada)."

A crevicular flow meter (Harco - HAR.600) was utilized for quantitating gingival fluid flow (Borden, et al., 1974). These investigators collected fluid from 10 subjects and a total of 59 gingival crevices. They utilized the intracrevicular and extracrevicular techniques. The quantities of fluid collected were compared for correlation with the Gingival Index (Loe and Silness, 1963) and gingival pocket depth. The authors further investigated the effect of repeated intracrevicular placement of filter strips (strips were inserted 6 times at 30 second intervals for a period of 3 seconds). The results indicated the 3 second technique intracrevicularly to be more sensitive than the extracrevicular technique. Since repeated intracrevicular measurements did not significantly alter the crevicular fluid flow a technique of measurement was suggested as follows: 1) emptying of the crevicular pool of fluid by insertion of a filter strip for a short time, 2) allowing the pool to fill again for 30 seconds, 3) collecting the quantity of new fluid for 3 seconds and quantitating with the fluid meter. The results of this investigation indicated the extracrevicular technique of collecting gingival fluid was unsatisfactory for slightly inflamed gingiva (G.I. = 1); fluid may have had difficulty escaping well adapted sulcular areas.

Shern, et al. (1974) compared ninhydrin staining with the crevicular flow meter (Harco). The crevicular fluid flow from 60 human male subjects was quantified. They concluded "precision, accuracy and reliability of measuring fluid flow proved greater using a flow meter than using the ninhydrin technique." They found further, using a combination of crevicular fluid flow measurements and cytologic smears "could provide valuable physiologic measurements for clinical trials."

In a thesis study Suppipat (1976) investigated a crevicular flow meter (Harco - HAR 600) for use in clinical research. He found measuring gingival crevicular fluid flow a sensitive and objective method for evaluation of the condition of the marginal gingiva. For his study he utilized the "orifice" method (Loe and Holm-Pederson, 1965).

Different methods of collecting fluid from the crevice have been used. The intracrevicular method (Brill, 1962; Bjorn, et al., 1965; Egelberg, 1964), involved inserting a filter strip into the crevice until resistance was felt. The fluid was then collected for several minutes.

Egelberg (1966) demonstrated even gentle insertion of a filter paper strip in the gingival crevice caused sufficient damage to alter the permeability of the epithelium so the amount of gingival fluid increased.

Loe and Holm-Pederson (1965) illustrated the use of

a less traumatic method in which the strip was inserted just into the orifice of the gingival crevice.

Composition of Crevicular Fluid

Because of the high degree of correlation between the gingival crevicular fluid flow and inflammation found by most investigators, several studies have been done in an effort to determine the composition of this fluid and its origin.

Brill (1959a) found that vigorous chewing of paraffin by human subjects stimulated gingival crevicular fluid flow. On qualitative analysis (utilizing ninhydrin, tri-ketohydrindenehydrate staining technique) he found sulcular fluid from stimulated and non-stimulated sulci contained alpha amino-acid.

In further studies Brill and Bronnestam (1960), by immuno electrophoretic analysis, found seven separate serum protein components. These included α_2 -, beta-, and gamma-globulin. From this information they hypothesized the fluid originated from tissue fluid and was extracellularly formed by filtration through capillaries.

Gustafsson and Nilssen (1961), in an assessment of human gingival crevicular fluid from clinically healthy gingiva, found fibrinolytic factors. They reasoned these fibrinolytic factors could have been significant in counteracting the deposition of fibrin and other proteins at the junction

Between gingival epithelium and tooth.

Krasse and Egelberg (1962) did flame photometry studies on human gingival fluid to determine the relative proportions of sodium, potassium, and calcium. Their results showed a proportional increase in potassium from clinically healthy gingiva. They deduced the cause for this was intracellular potassium added to the extracellular fluid as it left the sulcular epithelium. This led to the conclusion gingival crevicular fluid was not a simple filtration product but a true inflammatory exudate.

A study of the cell content of crevicular fluid (Egelberg, 1963a) showed a relative increase in inflammatory cells (PMNs, lymphocytes) compared to epithelial cells from inflamed gingiva.

Lysozymal activity of human gingival fluid has been examined (Brandtzaeg and Mann, 1964) with serum and saliva (eleven patients: seven with gingivitis, five with periodontitis). Turbimetric spectrocolorimetry techniques were utilized with *Micrococcus lysodeikticus* substrate. They found crevicular lysozymal activity showed a tendency to increase with severity of inflammation while serum and saliva did not. Since there was no observed correlation between the three body fluids they concluded the lysozymal enzyme to be primarily of local origin, possibly derived from leukocytes.

In another study involving gingival fluid, saliva

and serum (Sueda, et al., 1966), gingival fluid was found to contain proteins, lipids, polysaccharides bound to proteins (muco-, glyco-, or lipo-proteins). They noted histochemical reactions were similar in gingival fluid and serum, but weak or absent in saliva. This led them to confirm a previous hypothesis (Cimasoni, 1966): a micropolysaccharide substance, identified earlier (Toto and Sicher, 1964), found between the enamel and crevicular epithelium of humans could have been condensed gingival fluid.

Carbohydrate components have been demonstrated in gingival crevicular fluid (Hara and Loe, 1969). These investigators collected the fluid on filter paper strips. Concurrent with the fluid sampling of the crevice, a blood sample for serum glucose analysis was obtained from the earlobe of each patient. The authors analysed both fluids according to the quantity of glucose, hexosamine and hexuronic acid. The glucose in exudate was 1.13 - 7.09 μ g/mg; in serum the concentration was normal 0.90 - 1.24 μ g/mg. Gingival fluid also showed an increase in hexosamine. They hypothesized: hydrolyzing enzymes released during inflammatory state may have caused separation of the connective tissue intercellular matrix. This would have allowed passage of small molecular substances like hexuronic acid and hexosamine. The latter were present in the tissue because of increased capillary permeability. Similar enzyme activity probably was affect-

ing cell walls of the microorganisms. They concluded it may have been inferred glucose, hexosamine and hexuronic acid represented a breakdown of products of inflamed gingiva. However, both the absolute and relative amounts of these substances may have been influenced by local microflora.

Recent work carried out (Bang, et al., 1970) indicated sodium concentration to be 28% higher than that of serum, while the potassium concentration was nearly double that of serum. This supported previous studies (Krasse and Egelberg, 1962) lending credence to the interpretation a plasma-like fluid became modified as it passed through the sub-crevicular interstitial tissue space where cells were undergoing inflammatory changes. Thus the fluid gained potassium escaping from damaged or dying cells.

Golub, et al. (1971) demonstrated a much higher concentration of urea in gingival crevicular fluid from clinically normal tissue than was present in serum. This could have indicated the water reabsorption (which usually followed the movement of sodium to maintain osmotic balance; Windhager, et al., 1959) resulted in hyperconcentration of urea. This study also demonstrated an increase in the amount of sodium and decreases in the urea concentration in inflamed gingival tissues.

Golbu, et al. (1976) found collagenase activity of the fluid under gingival flaps of partially erupted third

molars varied directly with the collagenase activity of the tissue itself and inversely with its collagen content. Further, they found the breakdown products of collagen produced by this fluid similar to those produced by the gingival crevicular fluid collagenase.

Cimasoni (1974) presented an excellent review of crevicular fluid components. He correctly stated the problems involved in assaying small volumes of fluid and interpolating general statements. This was particularly so with reference to enzyme activity and origin.

Correlation With Other Evaluators of Gingival Disease

Interdental gingival biopsies were examined by Brenier (1950). He determined all gingival tissues, regardless of their clinical state, showed some degree of inflammation. He concluded it would be impractical to establish a clinical index of gingival disease since tissue changes preceded clinically detectable changes.

Brill (1960) studied specimens from dogs with both healthy and inflamed gingiva. His findings showed inflammatory cells sparsely distributed beneath the crevicular epithelium in clinically normal tissues. In clinically inflamed gingival tissues he found a greatly increased number of inflammatory cells. In this study, the author found a relationship to exist between crevicular fluid measurements and the microscopic appearance of the tissue.

In another study (Egelberg, 1964) using planimetric analysis showed significant correlation between the size of areas of inflammation in clinically normal and inflamed gingiva and the amount of gingival exudate in three dogs.

Zachrisson and Schultz-Haudt (1968) utilized the appearance of the pocket epithelium, density, and distribution of inflammatory cell infiltration to evaluate the degree of gingival inflammation. Their results showed a relationship between clinical and histological diagnosis of inflammation in about fifty percent of cases. They also found inflammatory cells in small quantities in all clinically healthy gingiva. These investigators suggested the degree of inflammation be classified histologically.

In a study of crevicular fluid flow, Orban and Stallard (1969) attempted a correlation with Ramfjord's PDI index and Greene and Vermillion's OHI-S. The fluid was collected from six upper anterior teeth (assumption because it was not stated) and stained with .01% ninhydrin. Biopsies were obtained from the same teeth and other areas (not specified). These samples were collected from a "group of dental students". The biopsies were graded on a scale of 1-10 for inflammation (parameters not stated). The investigators found a correlation between their histological index and fluid volume. ($r=+.050$, no P value.) Between the plaque score and their histologic index they found $r = +0.71$, $P <$

0.01. The authors stated the former showed no correlation and the latter was significant. Their conclusion, among others, was consecutive measurements of the gingival fluid were of value in relating the response of gingival tissues to environmental, physiologic, and pathologic conditions and changes.

Oliver, et al. (1969) attempted to find a relationship between the Gingival Index (Loe and Silness, 1963), crevicular exudate flow and histologic inflammatory exudate. Sixty areas in 53 patients were selected and all studies were accomplished in these areas. The histologic study was done by scoring the density of the inflammation: 0 = no inflammatory cells in the connective tissue; 1 = sparse distribution of inflammatory cells in connective tissue; 2 = moderate accumulation of inflammatory cells in isolated areas, sparse elsewhere; 3 = dense infiltration throughout the connective tissue. They found there was no fluid generally when there was no inflammatory exudate. Clinically normal gingiva tended to show an absence of inflammatory cells microscopically. These investigators found a close relationship between the Gingival Index and exudate measurements.

Another study (Rudin, et al., 1970) measured gingival fluid related to 30 maxillary and mandibular teeth. Filter strips with a standardized notch were utilized so only minimal entrance into the crevice was insured. The M

Index was used to assess the degree of clinical inflammation. Biopsies were evaluated morphometrically. The percentage of inflammatory round cells, collagen fibers, connective tissue cells and blood vessels was studied. These investigators found a positive correlation between the M Index and crevicular fluid, as well as between the M Index and the degree of inflammatory cell infiltration. A high positive correlation also existed between the quantity of gingival fluid and round cell infiltration. A negative correlation existed between crevicular fluid volume and the percentage of collagen fibers.

Daneshmand and Wade (1975) utilized a histologic index assessing crevicular epithelium and the amount of round cell infiltration. They graded inflammation at 100 X microscopically according to the following criteria:

0 = No break in continuity of crevicular epithelium; absence of inflammatory cells.

1 = No epithelial breakdown; sparse distribution of inflammatory cells.

2 = Crevicular epithelial disintegration; round cell infiltration not reaching basal layer of the outer gingival epithelium.

3 = Disintegration of crevicular epithelium; round cell infiltration extending to the basal layer of the outer gingival epithelium.

These investigators also employed a morphometric

system they called the inflammatory cell index, in which the inflammatory cells in the corium crossed by the horizontal and vertical lines of the grid were counted. The gingival fluid flow was evaluated by the ninhydrin staining technique, utilizing the orifice method of fluid collection (Loe and Holm-Pederson, 1965). The degree of inflammation clinically was assessed according to the Gingival Index (Loe and Silness, 1963). Low positive correlations of approximately the same magnitude ($r = 0.3$) were found between both histological indices and the Gingival Index and crevicular fluid volumes. A moderate correlation was found between the amount of gingival crevicular fluid and the Gingival Index scores. The strongest relationship they found was between the Histological Index and the Inflammatory Cell Index. They concluded the different indices probably evaluated different aspects of the inflammatory response. The authors' suggestion was "in small samples it may be wise to incorporate parameters which evaluate both macroscopic and microscopic characteristics as well as measurement of gingival fluid whenever practicable."

Mahajan (1976), in a histologic study utilizing a mitotic index, showed the presence of inflammation in gingival crevicular tissues. She illustrated increased mitotic activity, especially in those tissues surrounding full crown restorations.

Gingival Crevicular Fluid Flow as a Monitor of Healing

Arnold, et al. (1966) demonstrated crevicular fluid flow rose to a maximum within one week following gingivectomy. It then decreased slowly for about five weeks, when it reached a level approximately equal to the preoperative readings. This experiment was done on five patients and a total of 85 pockets were evaluated. The initial rapid rise was attributed to removal of the epithelial barrier and marked inflammatory response following surgical trauma. The author did not state whether there was preoperative periodontal preparation. The mean reading (ninhydrin staining) presurgically was 2.7mm., the mean readings at the end of five weeks was 2.4mm.

More favorable results were reported by Sandalli and Wade (1969). In a study involving 12 patients, 19 to 50 years of age, they measured the fluid flow in 120 maxillary and mandibular incisors. The measurements were done immediately prior to scaling, prior to surgery (surgery performed two weeks following scaling and oral hygiene instructions), and at 2, 3 and 4 weeks post surgery. Treatment was accomplished via gingivectomy and flap procedures in a paired sample method. It was found the crevicular fluid flow was reduced significantly following initial preparation. The levels of fluid flow then decreased slowly after surgery and at four weeks had reached levels significantly lower than presurgical levels.

Interproximal maxillary and mandibular anterior teeth in twenty patients between the ages of 18 and 59 were studied by Golub, et al. (1971). The patients did not brush for 12 hours before collecting fluid. Fluid was collected, Gingival Index scored and pocket depth measured. The day following fluid measurements, scaling and root planing were carried out. One week later gingival surgery was performed preceded by gingival fluid volume readings. One month post-surgically, crevicular fluid was measured along with an estimation of the other parameters. The volume of fluid was measured directly. The results indicated scaling had no effect on gingival inflammation or pocket depth, according to the parameters G.I. and fluid flow. This was in contrast to the results of Sandalli and Wade (1969). Surgery had a significant effect on all parameters. The authors speculated further upon the content and effect of urea concentrations in the gingival fluid. Among other things, the authors indicated presurgical scaling may have improved the health of the tissues, which resulted in a better healing rate than shown by Arnold (1966).

RELATIONSHIP OF RESTORATIVE MARGINS AND CONTOUR TO GINGIVAL HEALTH

Margins of restorations were evaluated histologically (Waerhaug, 1960) in relation to the gingiva. This

study inferred gingival inflammation was caused by plaque and not necessarily initiated by the nature of the material nor its surface roughness. It was implied, however, subgingival margins contributed to the etiology of periodontitis by retaining plaque microorganisms in the area of the restoration-tooth margin. He further suggested margin placement must involve consideration of caries rate; predisposition of the host to periodontitis; oral hygiene and aesthetics.

Morris (1962) discussed artificial crown contours and their relationship to gingival health. He reappraised earlier views regarding buccal and lingual contours which were supposed to deflect food from entering the crevice. He stated using bulges in crowns to protect the gingival crevice produced crown contours in excess of anything found in nature and caused, rather than prevented, gingival pathology.

Loe (1962) stated investigations have shown "any known type of dental restoration extending into the subgingival area causes damage to the periodontal tissues; either by providing possibilities for bacterial retention and/or by a direct irritational effect of the material per se." He further stated the concept of "extension for prevention" was no longer valid. He called for a "new theoretical basis for a combined prophylactic treatment of caries and periodontal disease."

An interesting histological study was undertaken by Marcum (1967) in which 12 teeth were prepared from crowns in each of 6 dogs. Four finish lines were above the gingival crest, four were even with the crest and four were prepared below the crest. The dogs were subsequently sacrificed and histologic sections prepared. The results showed margins located at, or even with, the gingival crest effected the least inflammatory response, while those margins above and below produced slight to severe inflammatory response. The author inferred: the poor gingival response observed around restorations placed supragingivally may have been due to plaque formation and adherence of food debris; the favorable tissue reaction to adjacent margins may have been due to better marginal adaptation and deflective contour.

The effects of rough surfaces on marginal gingiva have been investigated. In an histologic study (Trevedi and Talim, 1973) there were no clinical changes evident. Histologically there was evidence of inflammation in two-thirds of the patients. The inflammation was maximal adjacent to silicate cements and acrylic restorations. They concluded, "the gingival response appeared to be caused by chemical injury, unpolished restorative materials, poor marginal fit, and inadequate oral hygiene.

Morphologic changes of capillaries in human gingiva adjacent to teeth restored with complete crowns have been

observed utilizing a capillary microscope (Maruyama, et al., 1976). The results of this study showed more than one-fourth of the capillary loops nearest complete crowns exhibited dilatation (diameter greater than 20mm.). This dilatation (in agreement with Stallard, 1967) represented inflammation. Dilatation and complex capillary loops existed in the clinically normal gingiva adjacent to complete crowns. These readings were done in the mid-labial aspect of an upper central incisor.

Newcomb (1974) did an in depth study of anterior veneer crowns. These crowns (34 porcelain jacket crowns and 35 ceramo-metal crowns) were used on 59 patients, the average age of the crowns was 8.23 months. He used the Gingival Index (Loe and Silness, 1963) and the Plaque Index. The extension of the margin into the sulcus was measured by subtracting the distance of the crown margin from the gingival crest from the pocket depth. The margin extensions were divided into four groups according to the distance from the base of the crevice: 0.25 mm., 0.5 mm., 0.75 mm., and 1.00 mm. The results showed: all crowned teeth had a higher Gingival Index score than non-crowned contralateral controls; there was no difference in the Gingival Index between controls; a significant difference between Gingival Indices of crowned teeth with group 1 (0.25 mm.) having the highest score, and group 4 (1.00 mm.) the lowest score. Groups 1

and 2 showed a significant difference in sulcus depth between crowned and control teeth; groups 3 and 4 showed no difference in sulcus depth between crowned and control groups. As the degree of inflammation increased so did the crevice depth and depth of crown margins. The distance between crevice depth and margin depth decreased with increased inflammation. The author concluded: the nearer a subgingival crown margin approached the base of the gingival crevice the more likely there would be severe inflammation; the least inflammation associated with subgingival margins occurred when the subgingival margin was placed at the crest of the gingiva or just below. This apparently substantiated the work of Marcum (1967).

In a literature review done for the American Academy of Restorative Dentistry, Ramfjord, et al. (1974) reviewed a three year study done in Denmark. This work evaluated the gingival status of 334 abutment teeth in 110 patients. The results showed gingivitis to be more severe, with deeper pockets and more loss of attachment around teeth with subgingival margins compared with supragingival margins and nonrestored teeth. Interestingly the study showed 64% of all margins were placed subgingivally initially; at the end of the three year study only 46% of these were still subgingival.

A study was designed to find whether cast crowns

with subgingival margins were more frequently associated with pathologic depths than were unrestored contralateral teeth (Larato, 1975). The influence of frequency of tooth brushing on pocket depth adjacent to teeth was also investigated in this study. One hundred eleven male patients were examined. This study revealed the average pocket depth adjacent to teeth with crowns associated with subgingival margins was 3.4mm. Average pocket depth adjacent to nonrestored teeth was 2.7mm. Fifty-four nonrestored teeth had at least one pocket depth greater than 3 mm., while eighty-four crowned teeth had at least one or more measurement greater than 3 mm. No positive relationship could be found between tooth brushing frequency and the pocket depth adjacent to teeth restored with complete cast crowns. In nonrestored teeth pocket depth increased with reduced frequency of tooth brushing. Pocket depth also increased with increased age of the patient in both restored and nonrestored teeth, and with the cast crown.

In a discussion in *Dental Clinics of North America*, Eissman, et al. (1971), regarding crown contours and margin placement, suggested supragingival margins were preferred. The authors, however, realized this was not always possible clinically. They offered the following guidelines for margin placement: supragingival margin placement on enamel wherever possible; restorations placed at, or beneath, the gingival

crest represent a compromise and should permit a decreasing axial curvature in direct proportion to the occlusal gingival length of the restoration.

In the same article the authors proposed a premise for physiologic contouring. They refuted the in vitro textbook projections of contours and suggested substitution of cognate physiologic concepts. The authors suggested axial contour was related to four factors: 1) clinical crown length; 2) tissue architecture; 3) contour of adjacent teeth; and, 4) character of the opposing occlusion. As the distance from the occlusal table to the free gingival margin increased, the necessity for protective convexities decreased and the accentuation of natural concavities became increasingly important. It has been stated physiologic tooth contouring was directed toward minimizing plaque retention by exposure of the largest possible area of the clinical crown to cleansing actions.

Yuodelis (1973) reviewed the literature on the subject of crown contours. Using their observations in combination with this review they concluded plaque retention was the primary cause of both caries and periodontal disease and plaque retention in turn was aided by overcontours widely accepted as normal. They stated: 1) the greater the degree of facial and lingual bulge, the more plaque was retained in the crevical region; the flatter the contour, the less plaque

retained; 2) if root portions of teeth must be covered by complete artificial crowns subsequent to periodontal therapy, the final restorations should not follow the original anatomic crown and should recreate the original contours of the root position.

CHAPTER III

MATERIALS AND METHODS

Patients for this study were selected randomly from those undergoing periodontal surgery at Loyola University, School of Dentistry. Each patient signed a witnessed consent form (Appendix) describing the procedure. Brief medical and dental histories were obtained. Twenty-seven adult patients between the ages of 25 and 61 were selected with a mean age of 45 (Table 2).

Each patient selected for this study had undergone presurgical preparation which included oral hygiene instruction, diet counseling, scaling and polishing, and where indicated, deep scaling, root planing and curettage (non-surgical). The initial crevicular fluid volume readings were taken immediately prior to surgery before administration of anesthetic or any other drugs.

The areas selected for measurement were the interproximal papillae adjacent to, and including the mesial of, the first molar to the mesial of the contralateral first molar, including both maxillary and mandibular arches. Table 2 shows the frequency distribution for teeth evaluated, according to arch and tooth number. At the time of

the initial measurement of crevicular fluid volume, the marginal gingiva was evaluated in the area of the fluid measurement via the Gingival Index* method. Pocket depths were gauged in these areas after the crevicular fluid was collected.

The biopsy specimen was obtained at this appointment in the following manner: the initial reverse bevel incision was done to the level of the alveolar bone, an intracrevicular incision was accomplished and the remaining adherant tissue removed. From this piece of tissue a papilla was removed and placed immediately into 10% Formalin solution. These were then imbedded in paraffin and sectioned 6-10 microns in thickness. Hematoxylin and eosin staining technique was used.

*Gingival Index according to Loe and Silness,
1967.

- 0 - Absence of Inflammation.
- 1 - Mild Inflammation: Slight change in color and little change in texture.
- 2 - Moderate Inflammation: Moderate glazing, redness, edema and hypertrophy. Bleeding on pressure.
- 3 - Severe Inflammation: Marked redness and hypertrophy, tendency to spontaneous bleeding, ulceration.

The term Gingival Index or G.I. as used in the remainder of this paper has reference to this method of qualitative analysis of the marginal gingiva.

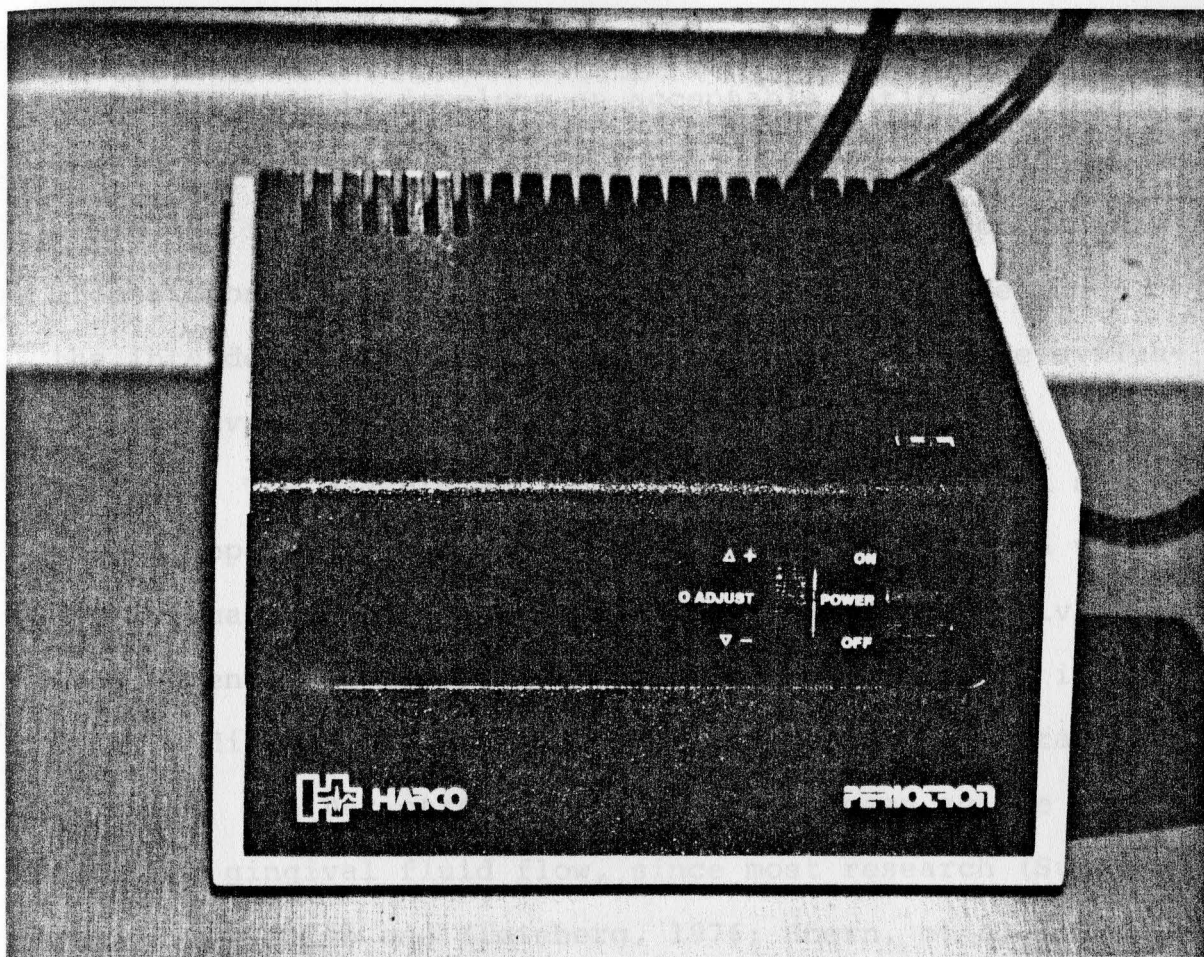
Fluid measurements were obtained initially and again at weeks four, six, eight, and ten following surgery. These data were recorded and subsequently subjected to statistical analysis. Because of the number and spacing of patients it was not possible to evaluate each patient at each time interval. To ensure confidence of the final results, a random sample size of approximately one-third was evaluated at each time interval. The measurements of fluid volume were accomplished by three persons trained in use of the Periotron. In an attempt to diminish possible variance of readings between operators the same person did at least two readings on each patient. As can be seen from the tabulation (Table 4), each patient averaged three readings; therefore, the same operator recorded at least two-thirds of the readings for each patient.

The assessment of pocket depth was made by one operator, while the Gingival Index was rated according to at least two trained observers. This was carried out at the initial reading appointment.

The histologic study was accomplished by one operator (with skillful guidance) in a blind study.

The interproximal area is generally the most seriously affected (Egelberg, 1964); it may be generally stated the tissues surrounding anterior teeth are associated with a less severe inflammation than posterior

structures in the same individual. In order to fairly
 evaluate the entire oral cavity it was decided to assess



horden, et al. 1974) indicates this instrument is more
 sensitive to change; **THE PERIOTRON** than other methods.

Periopaper* supplied by Harco was utilized for
 collecting fluid (Figure 2). The orifice method accord-

*Periopaper - Trade name for filter paper manu-
 factured by Harco Electronics, Limited, Winnipeg, Canada.
 (1.5 mm. X 13 mm.)

FIGURE 3

structures in the same individual. In order to fairly evaluate the entire oral cavity it was decided to assess the periodontal tissue of bicuspid and cuspid. An attempt was made to develop an acceptable ratio between mandibular and maxillary structures. Furthermore, because the interproximal area demonstrates some homogeneity of periodontal structures between teeth, it was decided the interdental papilla of adjacent teeth should be evaluated wherever possible.

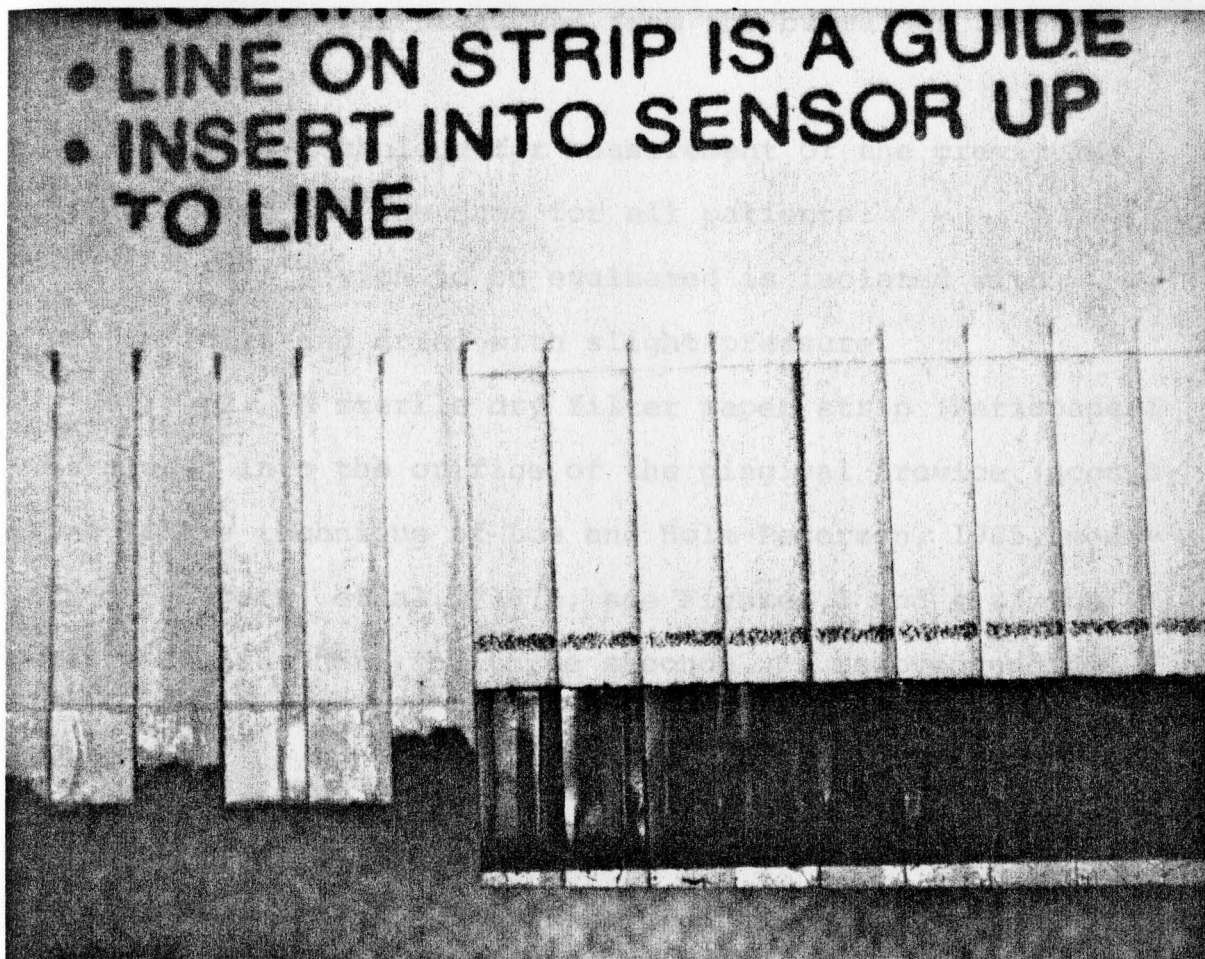
The measurement of fluid volume is subject somewhat to operator variance. For this reason the mean values for evaluated papillae were used instead of the individual measurements, as there is less likelihood of errant individual readings affecting the total aggregation of data.

The Periotron (Figure 3) was chosen as the evaluator of gingival fluid flow, since most research (Suppapat, 1976; Golub and Kleinberg, 1976; Shern, et al. 1974; Borden, et al. 1974) indicates this instrument is more sensitive to change in fluid volume than other methods.

Periopaper* supplied by Harco was utilized for collecting fluid (Figure 2). The orifice method accord-

*Periopaper - Trade name for filter paper manufactured by Harco Electronics, Limited, Winnipeg, Canada. (1.5 mm. X 13 mm.)

ing to Lee and Holm-Pederson (1965) as modified by Larson, et al. (1974) was the technique utilized (Figure 1).



collect fluid for three seconds. The total elapsed time is thirty seconds. PERIOPAPER, AS SUPPLIED

4. The second filter strip is immediately placed between the recording sensors to the depth of the premarked line (Figure 5). The Periotron previously had been switched to the on position and calibrated to zero with a dry, sterile Periopaper.

5. The Periotron gives a digital readout, which automatically is held after 3-29 seconds. This

FIGURE 2

ing to Loe and Holm-Pederson (1965) as modified by Borden, et al. (1974) was the technique utilized (Figure 1). Access to crevicular areas was from the buccal or labial aspect.

The technique for measurement of the crevicular fluid volume was the same for all patients:

1. Region to be evaluated is isolated with cotton rolls and dried with slight pressure.
2. A sterile dry filter paper strip (Periopaper) is placed into the orifice of the gingival crevice (according to the technique of Loe and Holm-Pederson, 1965, modified by Shern, et al., 1974, see Figures 1 and 4). The strip remains here for three seconds, is removed and discarded.
3. The emptied fluid pool is allowed to fill for twenty-seven seconds and another sterile strip is placed in the same location. This strip is allowed to collect fluid for three seconds. The total elapsed time is thirty seconds.
4. The second filter strip is immediately placed between the recording sensors to the depth of the premarked line (Figure 5). The Periotron previously had been switched to the on position and calibrated to zero with a dry, sterile Periopaper.
5. The Periotron presents a digital readout, which automatically is held after 19-20 seconds. This

DIAGRAMATIC REPRESENTATION
ORIFICE METHOD OF FLUID COLLECTION
(Loe and Holm-Pederson, 1965)

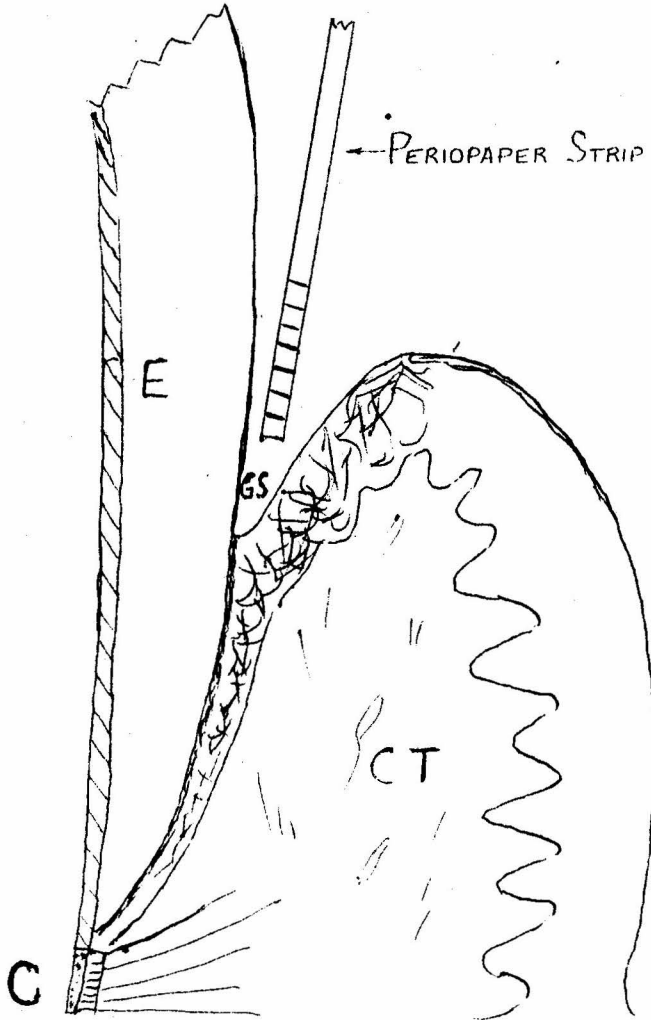
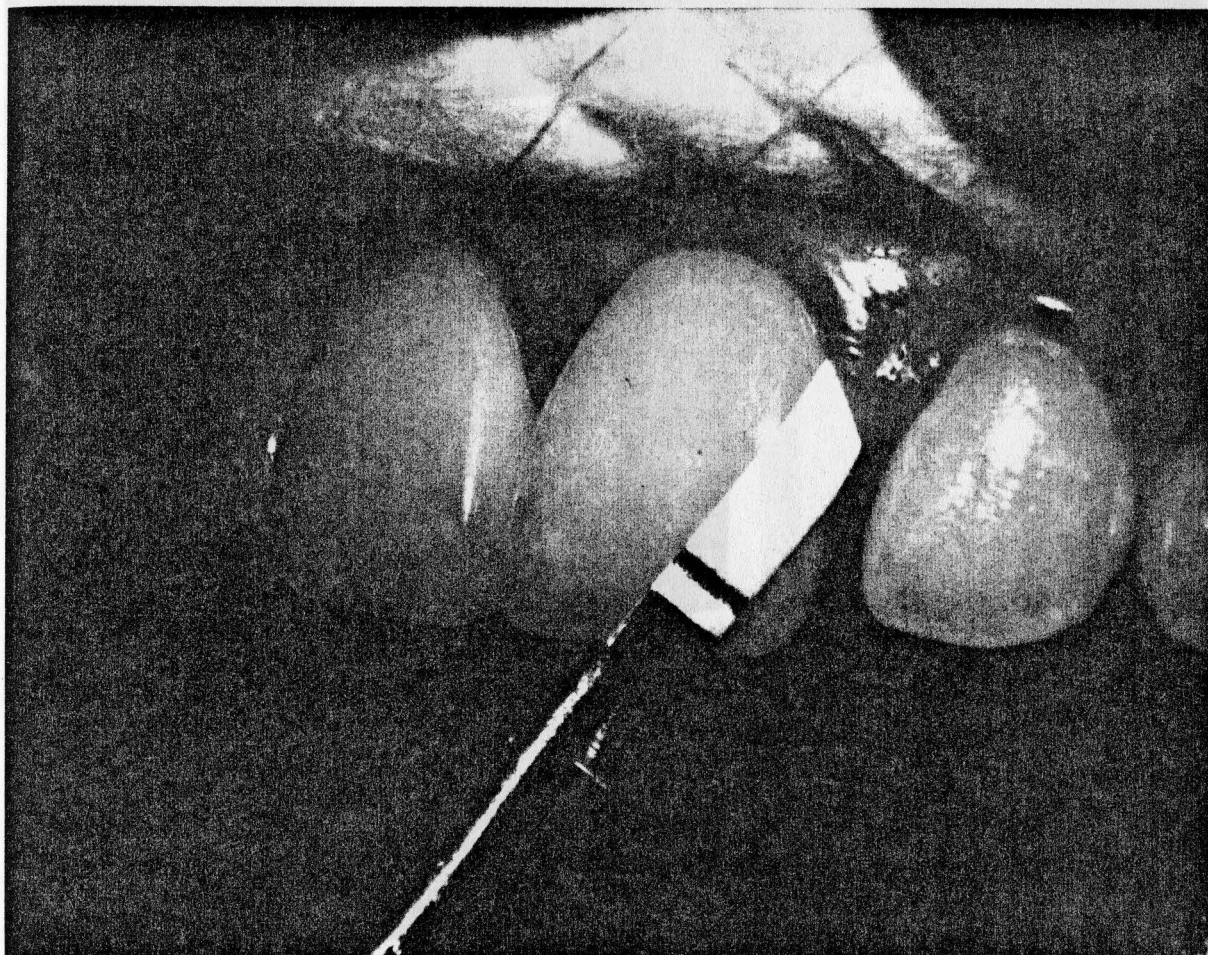


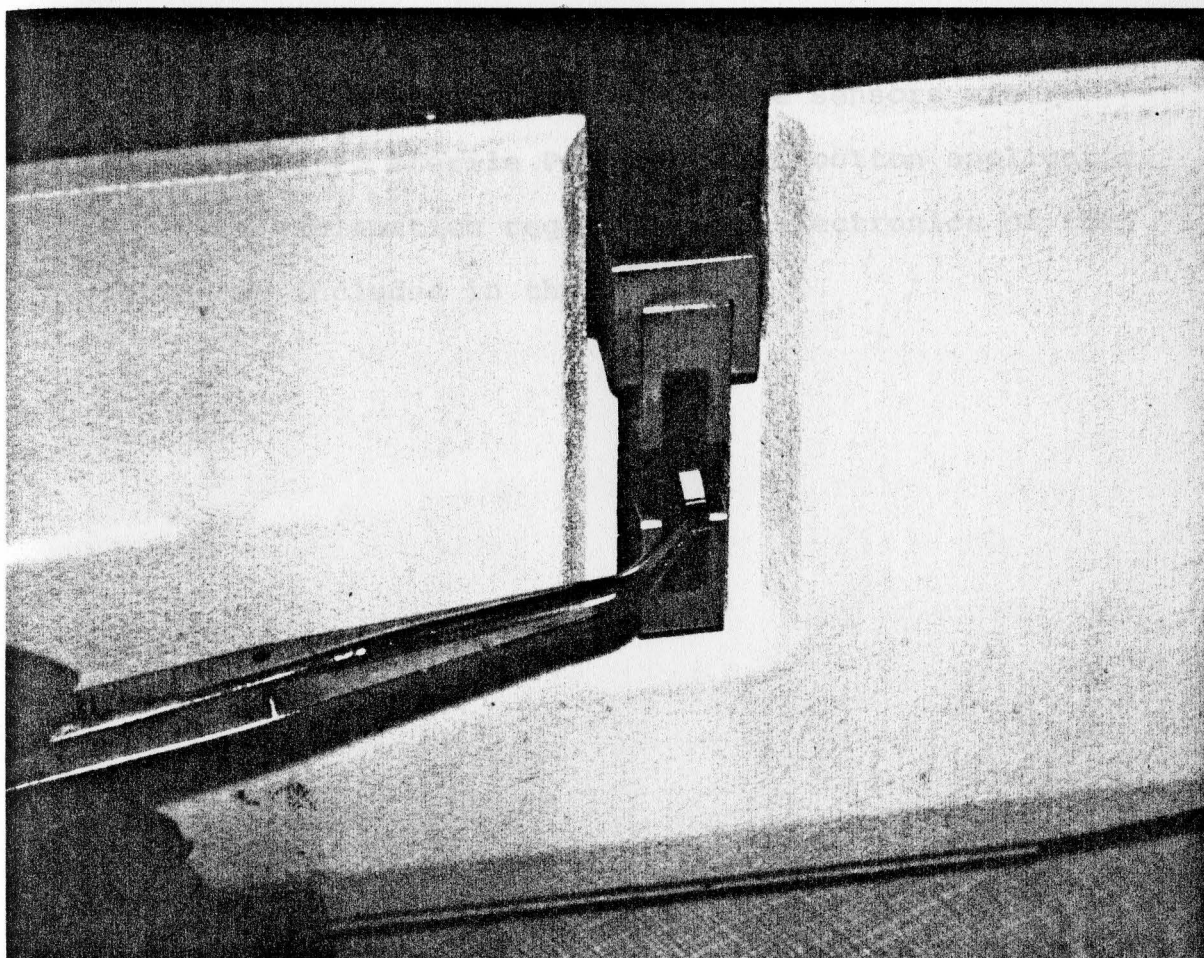
FIGURE 1



PERIOPAPER IN GINGIVAL CREVICE

FIGURE 4

reading is entered on the data sheet. This numerical reading may be converted to volume in microliters by di-



SENSORS, PERIOTRON

FIGURE 5

reading is entered on the data sheet. This numerical reading may be converted to volume in microliters by dividing by 200 (eg. a digital reading of 10=0.05 μ l).

6. After each measurement the sensors are dried with a dry, sterile Periopaper or cotton applicator.

An explanation regarding the electronics of the Periotron is included in the Appendix.

CHAPTER IV

EXPERIMENTAL RESULTS

Table 2 illustrates the frequency distribution and tooth numbers used in the portion of the thesis dealing with monitoring periodontal healing.

Table 3 is a compilation of the individual data sheets (see Appendix).

Table 4 is data compiled from Table 3. Weekly means are shown for weeks 0, 4, 6, 8, and 10. The results of a two tailed paired sample t test are also shown, with appropriate P values. From these data a significant difference can be demonstrated between weeks 0-8 ($P < 0.05$) and weeks 0-10 ($P < 0.05$).

Table 5 is a frequency table compiled from Table 4. It illustrates the patients who started with a reading of 0-10 and those who started with initial readings above 10. These are expressed as percentages and the table shows patients beginning with readings of 0-10 demonstrate about a 50% better prognosis.

Figures 8 through 12 are representative sections of types of inflammation found in this study.

<u>47 LOWER TEETH</u>			<u>47 UPPER TEETH</u>		
<u>TOOTH</u>	<u>FREQUENCY</u>	<u>%</u>	<u>TOOTH</u>	<u>FREQUENCY</u>	<u>%</u>
# 3	21	44.68	# 3	18	38.30
# 4	21	44.68	# 4	16	34.04
# 5	2	04.26	# 5	5	10.64
# 2	2	04.26	# 2	3	06.38
# 1	1	02.13	# 1	3	06.38
			# 6	2	04.26
Totals	47	100.00%	Totals	47	100.00%

Number of Patients	Male	Female
27	12	15

Number of Patients	Between Ages	Mean Age
27	25 and 61	45

TABLE 2

PAT- IENT #	AGE	GI	PKT. DPT.	TOOTH #	WEEK 0		WEEK 4		WEEK 6		WEEK 8		WEEK 10	
					GCF	G \bar{C} F	GCF	G \bar{C} F	GCF	G \bar{C} F	GCF	G \bar{C} F	GCF	G \bar{C} F
1	53	2	3	<u>4,3</u>	02.00		01.00		06.50		03.00		02.00	
		2	3	<u>3,4</u>	05.00	03.50	01.00	01.00	05.50	06.00	02.50	02.75	02.00	02.00
2	55	1	3	<u>3</u>	21.00		22.50							
		1	3	<u>3</u>	13.00	16.83	21.50	22.00						
		1	3	<u>4</u>	16.50		22.00							
3	47	2	3	<u>4,3</u>	22.00		12.00		15.00				04.00	
		2		<u>3,4</u>	21.50	21.75	20.50	16.25	33.00	24.00			03.00	03.50
4	30	1	3	<u>4,3</u>	06.50	06.50			05.50	05.50	02.00	02.00		
5	54	2	3	<u>3,4</u>	20.50				15.00		06.00		02.50	
		2	3	<u>2,3</u>	22.50	21.50			22.50	18.75	08.00	07.00	09.50	06.00
6	51	2	4	<u>3</u>	22.00		31.50						18.00	
		3	3	<u>1</u>	20.50		25.00						21.00	
		2	4	<u>3</u>	11.50		31.50						29.50	
		2	4	<u>1</u>	20.50	18.63	28.00	29.00					27.00	23.88
7	25	2		<u>4,3</u>	37.00				21.00		08.50		31.00	
		2		<u>3,4</u>	12.50	24.75				21.00	17.00	12.75	04.50	17.75
8	55	2	4	<u>4,3</u>	21.00		21.00				18.50		19.00	
		2	4	<u>2,3</u>	20.50	20.75	11.50	16.25			16.00	17.25	22.50	21.75
9	36	3	3	<u>3,4</u>	07.00								10.00	
		2	2	<u>1,2</u>	06.00	06.50							01.50	05.75

TABLE 3

PAT- IENT #	AGE	GI	PKT. DPT.	TOOTH #	WEEK 0		WEEK 4		WEEK 6		WEEK 8		WEEK 10	
					GCF	ḠCF	GCF	ḠCF	GCF	ḠCF	GCF	ḠCF	GCF	ḠCF
10	57	1 1	1 1	<u>4,3</u> <u>3,4</u>	09.50 14.50		22.50 14.50		12.50 15.00		11.50 05.00		08.50	
11	25	2 1	3 2	<u>3,4</u> <u>3,4</u>	25.50 07.00		13.50 01.00		08.00 05.00		05.00 04.00		04.50	05.00 02.50 03.75
12	50			<u>4,3</u> <u>3,4</u>	02.00 09.50		02.20 10.50						10.50 16.00	13.25
13	31	2 2	2 2	<u>4,3</u> <u>3,4</u>	13.50 26.00		12.00 12.00						12.50 14.50	13.50
14	45	0 1	1 1	<u>4,3</u> <u>3,4</u>	03.50 09.00		00.00 00.00						02.50 03.50	03.00
15	45	1	3	<u>4,3</u>	10.50	10.50			06.00	06.00				
16	39	2 2	5 6	<u>3,4</u> <u>4,5</u>	20.50 20.00		20.00 17.50						10.50 07.00	08.75
17	34	2		<u>2,1</u> <u>5</u>	12.00 24.00	18.00								
18	40			<u>4,3</u> <u>3,4</u>	08.00 13.50				19.50 02.00		18.00 04.00		11.00	18.75 03.50 11.13
19	52	1 1		<u>4,5</u> <u>3,4</u>	16.50 18.50									
20	50			<u>4,3</u> <u>3,4</u>	04.00 05.50									

TABLE 3

PAT- IENT #	AGE	GI	PKT. DPT.	TOOTH #	WEEK 0		WEEK 4		WEEK 6		WEEK 8		WEEK 10	
					GCF	ḠCF	GCF	ḠCF	GCF	ḠCF	GCF	ḠCF	GCF	ḠCF
21	49	1	3	4,3	02.00	02.00			04.00	04.00				
22	36	2	5	2,3	29.50	29.50					17.50	17.50	15.00	15.00
23	55	1	2	4,5	02.00				04.00				03.00	
		1	2	5,4	08.00	05.00			08.50	06.25			03.50	03.25
24	37	2	4	6,5	22.00				14.00		07.00		04.00	
		2	5	5,6	44.00	33.00			16.00	15.00	06.00	06.50	03.00	03.50
25	61	1		4,3	02.00	02.00	08.00	08.00	09.50	09.50	08.50	08.50		
26	57	1	2	3,4	07.00	07.00					01.00	01.00	00.50	00.50
27	58	1	2	4,3	03.00				00.50					
		2	4	3,4	09.00	06.00			07.00	03.75				

TABLE 3

PATIENT #	WEEK 0 GCF	WEEK 4 GCF	WEEK 6 GCF	WEEK 8 GCF	WEEK 10 GCF
1	03.50	00.10	00.60	02.75	00.20
2	16.83	22.00			
3	21.75	16.25	24.00		03.50
4	06.50		05.50	02.00	
5	21.50		18.75	07.00	06.00
6	18.63	00.29			23.88
7	24.75		21.00	12.75	17.75
8	20.75	16.25		17.25	20.75
9	06.50				05.75
10	12.00	18.50	13.75	08.50	
11	16.25	07.50	06.50	04.50	03.75
12	05.75	06.30			13.25
13	19.75	12.00			13.50
14	06.25	00.00			03.00
15	10.50		06.00		
16	20.25	18.75			08.75
17	18.00				
18	10.75		10.75	11.00	11.30
19	17.50				
20	04.75				
21	02.00		04.00		
22	29.50			17.50	15.00
23	05.00		06.25		03.25
24	33.00		15.00	06.50	03.50
25	02.00	08.00	09.50	08.50	
26	07.00			01.00	00.50
27	00.60		03.75		
MEANS	13.59	12.94	10.77	08.27	09.14
# Pat.	27	12	14	12	17

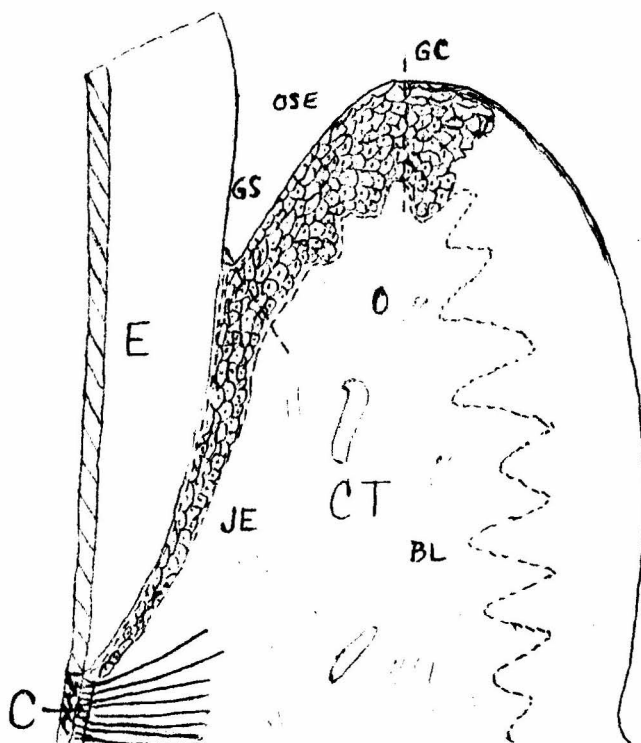
RESULTS OF 2 TAILED PAIRED SAMPLE T TEST

BETWEEN WEEKS 0-4	BETWEEN WEEKS 0-6	BETWEEN WEEKS 0-8	BETWEEN WEEKS 0-10
P=0.156	P=0.309	P=0.013	P=0.007

TABLE 4

	INITIAL READING	
	Group 1 GCF 10	Group 2 GCF 10
# of patients	11	16
# of patients completing study	10	14
	FINAL READING	
	Group 1 GCF 10	Group 2 GCF 10
# of patients	9	7
	90%	50%

TABLE 5



E-enamel; C-cementum; CT-connective tissue; BL-basal lamina; JE-junctional epithelium; OC-oral epithelium; GS-gingival sulcus; GC-gingival crest; OSE-oral sulcular epithelium; the dotted lines indicate the approximate boundaries between junctional, oral sulcular and oral epithelium.

DIAGRAMATIC REPRESENTATION
of the
GINGIVAL CREVICULAR AREA

FIGURE 6

CHAPTER V

DISCUSSION

The so-called gingival margin region includes those portions of the periodontium allowing attachment of the gingiva to the tooth. The gingival sulcus is the shallow groove between the tooth and normal gingiva. The depth of this sulcus may vary in "normal" health but generally is 0.5 mm. or less (Schroder and Listgarten, 1971).

The gingival sulcus seems to function as an initial defense mechanism; as such it shows unique features. The bottom of the sulcus is formed by the free surface of the junctional epithelium. The coronal portion is lined by an epithelium which could be described as the sulcular termination of the oral epithelium. This is depicted diagrammatically in Figure 1. In normal, ideal conditions a few leukocytes can be seen in the intercellular spaces of the junctional epithelium. When inflammation exists, the number of white cells increases in the junctional epithelium, and intercellular spaces increase in size allowing further ingress of inflammatory producing substances. The initial reaction is due to irritation caused by mechanical factors or, most often, irritation induced by plaque microorganisms.

The crevicular area is characterized by a par-

ticular fragility; the epithelium is permeable to various substances, especially in the presence of inflammation. Inflammation in the gingival tissues produces exudation which presents itself clinically as fluid in the crevicular sulcus. Measuring the intensity of this fluid flow is a reliable and accurate quantitative indicator of gingival inflammation.

The process of inflammation is complex. In the presence of microorganisms it becomes increasingly difficult to describe by standard parameters. This is particularly true when one attempts analysis of the minute amount of fluid produced during gingival inflammation (Cimasoni, 1974).

Gingival fluid flow is subject to many variables, both between patients and in the same patient. Examples of this variability are circadian periodicity (Bissada, et al. 1967), pregnancy (Loe, 1965), menstruation (Muhlemann, 1948) and, puberty (Sutcliffe, 1972). Age has been shown to have an influence on the fluid volume (Brankin, 1977). It is probable systemic medication and disease states (clinical and sub-clinical) will affect this also.

With the above observations in mind, it seems reasonable to establish a range of normal crevicular fluid volume. This has been accomplished (Periotron manual) and a range of Periotron readings established as follows: 0-10, healthy; 11-20, mild inflammation; 21-40, moderate

inflammation; over 40, severe inflammation.

Using the above established range, several observations can be made concerning the experimental results.

There was an overall improvement of approximately 71% (Table 4). This general overall improvement can be attributed principally to periodontal surgical procedures, initial oral hygiene instruction and frequent recall following surgery. The recall appointments, generally for the first four to six weeks, included polishing of tooth surfaces and reinforcement of oral hygiene procedures.

Previous studies have shown general improvement postsurgically for about five weeks (Arnold, 1966; Sandalli and Wade, 1969; Golub, et al., 1971). This was attributed to the reestablishment of an epithelial barrier. This assumption seems reasonable. The continued decrease in fluid volume in subsequent weeks (Table 4) can be explained as continuing regeneration involving the connective tissue portions of the gingival tissues. Previously, it has been suggested collagen formation is complete about 72 days after surgery involving flap procedures (Ramfjord and Costich, 1968). In healthy gingivae there should be a regeneration of the periodontal ligament fibers by this time, creating a more closely adapted gingival collar.

It is interesting to note if gingival crevicular fluid is considered to be an inflammatory exudate, the healing process just described takes place in the presence of an inflammatory state.

In the group of patients with initial fluid readings of 0 to 10, 90% remained below 10 (9 out of 10); 100% (10 out of 10) reached a reading of 10 or below sometime during healing. In the group of patients with initial fluid readings greater than 10 there is a reduction in this figure to only 50% (7 out of 14) with 57% ever showing a reading of 10 or less (Table 5). This indicates the degree of inflammation present prior to periodontal surgical procedures is inversely proportional to periodontal health at the end of this study. The above results raise this question: If presurgical readings of the quantity of crevicular fluid indicate a "normal" range, why proceed with surgery? Until the relationship between gingival crevicular fluid and inflammation is better understood we should strive to attain a state of gingival health which yields 0 fluid flow. For the present it seems fair to state: We can definitely improve gingival health via periodontal surgical procedures when the tissues are in a presurgical state of health. Our results, otherwise, are unpredictable. This indicates many other aspects must be considered in a wholistic patient appraisal,

before the decision is made to proceed with periodontal surgery.

For example, it has been shown scaling and oral hygiene instruction produce significant reduction in the inflammatory state (Sandalli and Wade, 1969). If this pre-surgical regimen should fail to produce these results, reappraisal is in order before proceeding with surgical treatment, since improvement is associated with the degree of inflammation present at surgery.

There is a slight increase in mean readings during week 10 (Table 4) which may have been contributed to by several factors. At the time these readings were taken, many of the patients were undergoing some phase of restorative dentistry and a few had received interim partial dentures. A positive relationship between restorations and inflammation has been demonstrated (Waerhaug and Zander, 1957; Brill and Bjorn, 1959). Although there was no plaque index recorded, it was noted after initial postsurgical polishing had been abandoned, there was a tendency for plaque accumulation in some patients. This could be due to decreased intensity of oral hygiene motivation or sensitivity due to exposed root surfaces. Plaque accumulation was noted also by Arnold (1966).

It can be seen from Table 4, the patients who showed increased readings after initial improvement were,

with one exception, patients with initially high readings. This strongly indicates a host/response relationship. This relationship may be with the irritant or microorganism, or an indication of abnormal inflammatory response.

The foregoing discussion illustrates the difficulty associated with obtaining a prognosis for periodontal surgery. Apropos to restorative intervention following periodontal surgery the following remarks apply. The initial (4-5 week) decrease in fluid volume readings indicate reestablishment of an epithelial barrier. The continued healing which takes place through week ten indicates reestablishment of the connective tissue elements. It therefore seems reasonable to state restorations whose preparation will not involve trauma to the connective tissue may be carried out four to five weeks postsurgically. Those restorations whose preparation will involve manipulation of connective tissue (probably the greater number) should not be carried out until at least ten weeks postsurgically. This is true for two reasons: Epithelium covering an immature connective tissue base is less resistant to trauma (Ramfjord and Costich, 1968); The quantity of crevicular fluid demonstrates the presence of inflammation, indicating the epithelium is more permeable at the four-five week stage. Manipulation at this point would allow the ingress of toxic products to increase,

causing a delay in healing (Brill, 1959b; Squier, 1975).

This study indicates a vital ingredient of successful periodontal therapy is regular, periodic debridement of the tissues involved with the crevicular area and oral hygiene reinforcement.

The author feels monitoring gingival healing by quantitating the volume of the gingival crevicular fluid flow is accurate and useful. However, greater knowledge and understanding of the composition of gingival crevicular fluid and its relationship to inflammation are necessary for quantitative monitoring of crevicular fluid to be valued as the sole parameter in assessing gingival health.

There were insufficient representative samples to attempt any type of histologic correlation. This was due to poor biopsy technique. Very few areas of junctional, crevicular and oral epithelium were available in proper relationships.

However, there were specimens with epithelium, either oral or sulcular, intact with their respective connective tissue bases for general examination. Sixteen slides were reviewed for general characteristics. All showed at least a sparse infiltration of inflammatory cells. Approximately 10 showed a more or less dense inflammatory exudate. In one slide nearly all normal collagen was replaced by a

very dense infiltrate, associated with ulceration of the epithelium.

In general these histologic samples indicated a chronic inflammatory state, or a transitional stage between acute and chronic. Cell types were neutrophilic polymorphonuclear leukocytes, plasma cells, macrophage, isolated instances of eosinophils and the ubiquitous lymphocytes. An attempt was made to illustrate acute, chronic and normal sections. These are shown in Figures 8 through 12.

The prevalence of chronic or subacute sections is an expected observation since these patients had undergone presurgical periodontal treatment for a period of time varying from 1 to 3 weeks prior to surgery. The removal of plaque, and in some cases granulation tissue, should allow healing. This is especially true in shallow crevices (3 mm. or less) where the patient has better access for plaque disruption.

The illustrations shown in Figures 8 through 12 are examples of tissue sections obtained from patients in the study immediately prior to surgery. The author has attempted to show sections which would illustrate various stages of inflammation according to cell types and other morphologic considerations. These sections are illustrations of inflammation and do not imply correlations with readings of the Periotron.

Gingival inflammation and periodontal disease are not static events. Therefore, more than one stage of inflammation is probably present at the same time. Morphogenic sections such as those illustrated can show only one portion of the entire inflammatory process present within patients.

The function of inflammation is to mobilize the body's defenses, bring them to the injured site and attempt to overwhelm the source of injury. The source of injury may be mechanical, chemical or bacterial. Regardless of the source of injury the tissue response is essentially the same and the purpose is: to bring to the area certain phagocytic cells (neutrophilic polymorphonuclear leukocytes, macrophage, etc.) which dispose of bacteria, dead cells, or other debris; bring antibodies to the site (humoral antibodies are serum proteins and escape from the altered endothelium); neutralize and dilute the irritant (edema); limit the spread of inflammation; and, to initiate repair.

Inflammatory responses may be subdivided into four major types (Bhaskar, 1969): Acute, subacute, chronic and chronic granulomatous. These four types are not distinct entities and transitions from one to the other occur constantly. These types or stages of inflammation are responses to tissue injury and the appearance of each depends on the type and intensity of the irritant and the response

of the host.

Acute inflammation occurs immediately following injury. Initial vasoconstriction is followed by vasodilatation. The cellular components of the blood stream move peripherally and adhere to the vessel wall. The vessels become dilated and white blood cells, particularly polymorphonuclear leukocytes, migrate into the tissue. These cells are attracted to the site of injury by chemotaxis. As inflammation becomes more severe the endothelial spaces widen, allowing the further migration of larger molecular weight substances. These larger weight substances are proteins (albumin, globulins and fibrinogen). Water also leaves the blood stream in large amounts causing edema. The crevicular epithelium evidences changes during this time, initial widening of the interepithelial spaces increases, allowing further ingress of inflammatory products. In an apparent attempt to prevent this the epithelium initially becomes thickened; this is demonstrated histologically and by the increase in the mitotic index (Mahajan, 1976). At the same time the epithelium is undergoing this change, the connective tissue fibers are undergoing degeneration. The ingress of inflammatory cells causes increase in size of the subepithelial layer. This in turn applies pressure to the epithelium which becomes further distended. Unless the inflammatory cause is neutralized or removed

there is eventual ulceration of the epithelium.

Acute inflammation is characterized histologically then by edema, an infiltrate of leukocytes (predominantly polymorphonuclea leukocytes), disintegration and loss of collagen from connective tissue fibers and epithelial changes.

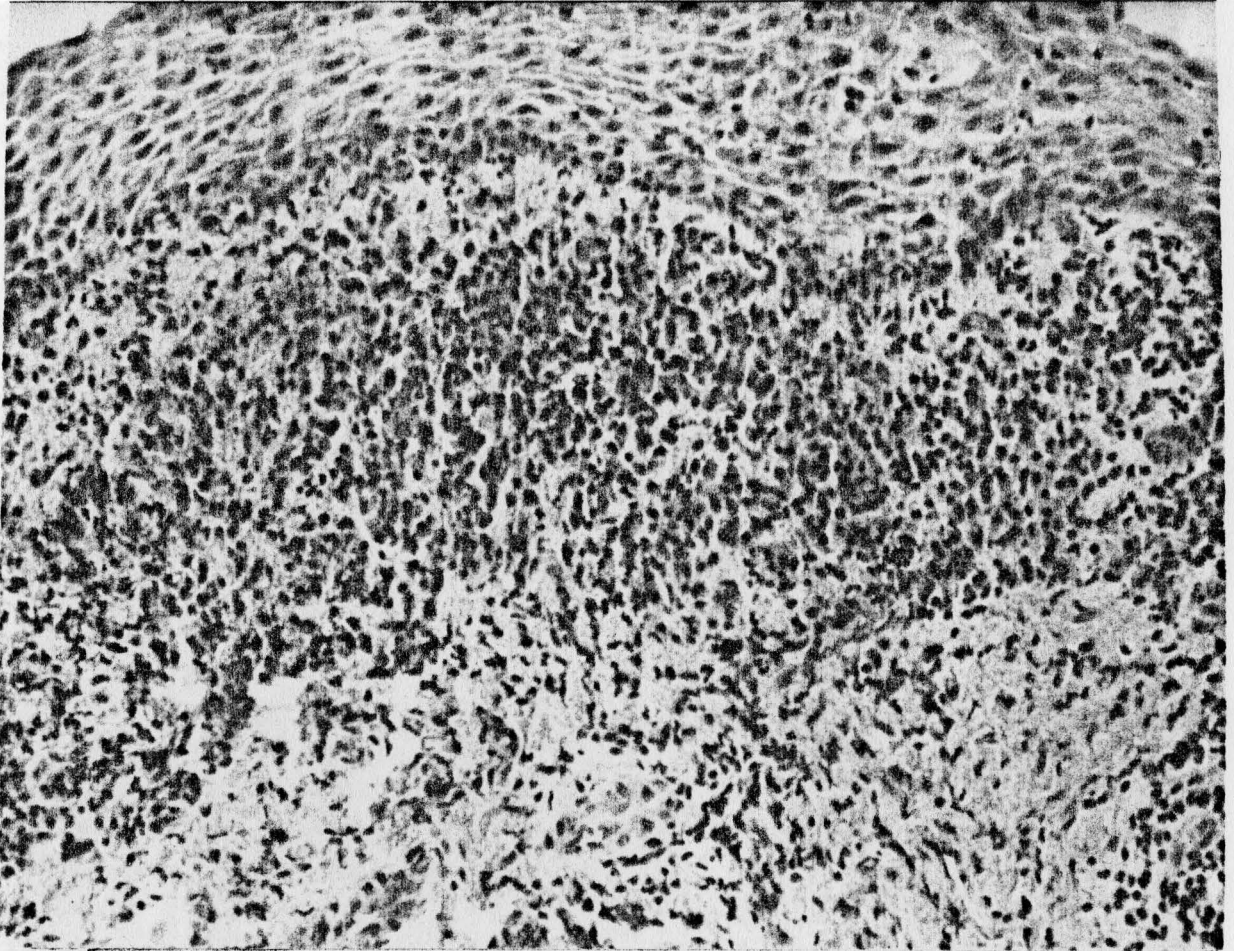
Chronic inflammation is a low grade, prolonged and proliferative type of inflammation. This occurs when the irritation is of low virulence, when host resistance is adequate or as a sequellae to acute inflammation. Chronic inflammation may exist as a continuous reaction of acute inflammation. It may be indistinguishable from the acute stage. The predominant white blood cell is the lymphocyte and in proliferative or healing stages plasma cells and lymphocytes exist together with plasma cells predominant. Microscopically the white cell infiltrate is less dense, lymphocytes and plasma cells predominate. The connective tissue damage may be less severe. Generally collagen loss from connective tissue fibers is not severe enough to allow rapid diffusion of the inflammatory infiltrate. The epithelial changes are less pronounced. Chronic inflammation persists for months to years, whereas acute inflammation lasts from days to two or three weeks. When an area of inflammation demonstrates characteristics of acute and chronic elements it is described as sub-acute.

Occasionally chronic inflammation may be characterized by the presence of large numbers of macrophage. These cells form diffuse, circumscribed masses. This response is called chronic granulomatous inflammation specific for certain types of causitive agents such as foreign body reaction and fungus diseases.

Normal sulcular tissue is demonstrated in Figures 10 and 11. Though this represents the most normal section in this study, it should be noted there are white cells present in the connective tissue. This may be characteristic of normal crevicular tissue (Brenier, 1950).

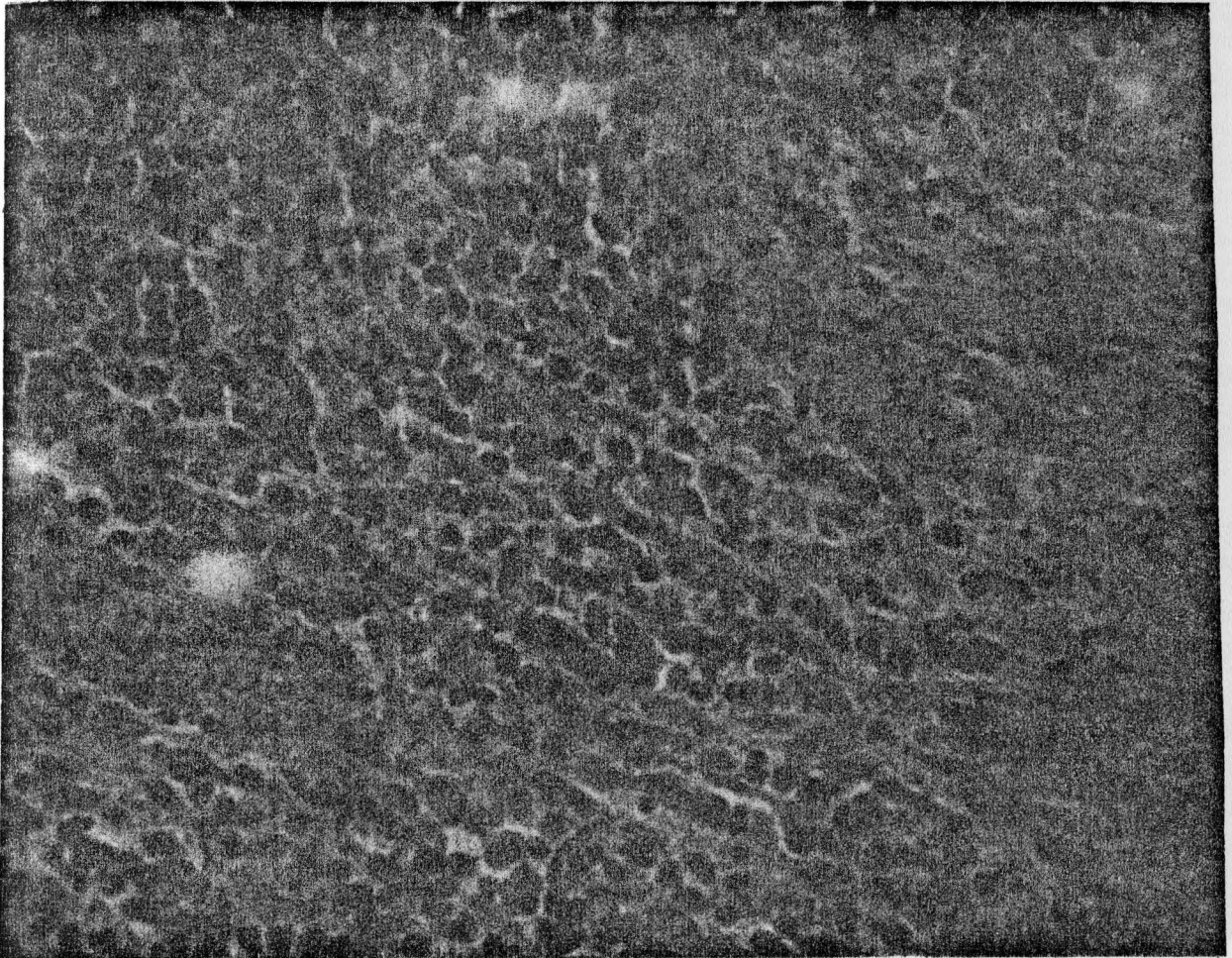
The author would like to emphasize the above descriptions are very general and it is not always possible to show discrete stages. The chemical processes occuring during inflammation have not been considered; these contribute in all phases of inflammation. The immune response of the host may also play an important role by contributing to cell damage, especially when the antigen antibody complex activates compliment.

To summarize, inflammation varies in stages from acute to chronic. Any microscopic specimens then may show any degree of this expansion and the severity may vary from extreme to mild. Judgement of the effects and stage of inflammation are made by: the appearance of the epithelium; density of the infiltrate and cell type; amount of collagen



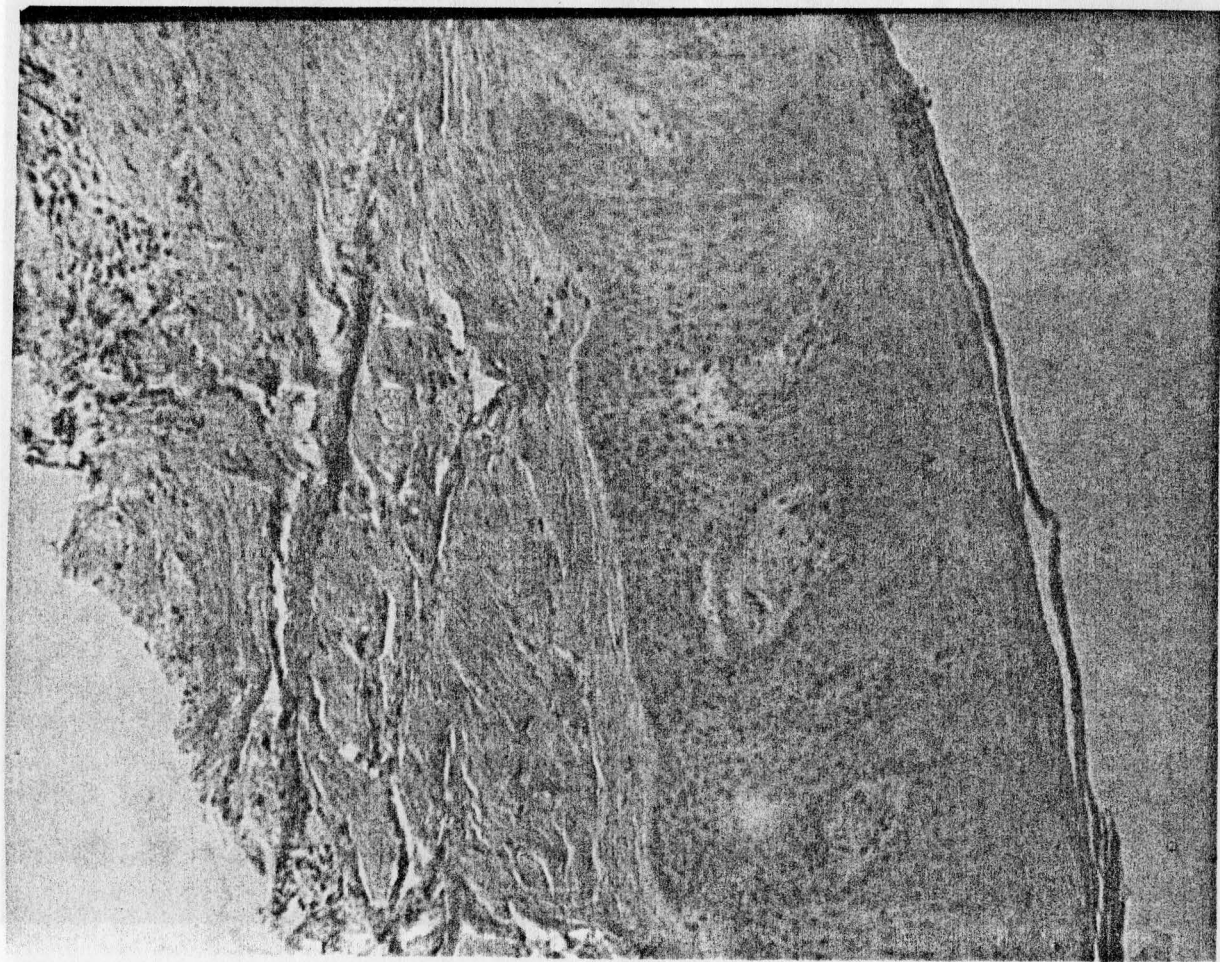
Sulcular epithelium. Sub-acute stage of inflammation. The inflammatory infiltrate shows cell types from chronic and acute stages. It appears PMN and lymphocytes are about equally divided. Breaks are evident in the basal cell layer of the epithelium and leukocytes can be seen in the widened intercellular spaces. The connective tissue central to the infiltrate is unorganized and undergoing dissolution.

FIGURE 8



Example of cell types in inflammation. Monocytes are present with lymphocytes. Polymorphs can be seen migrating through the widened intercellular spaces.

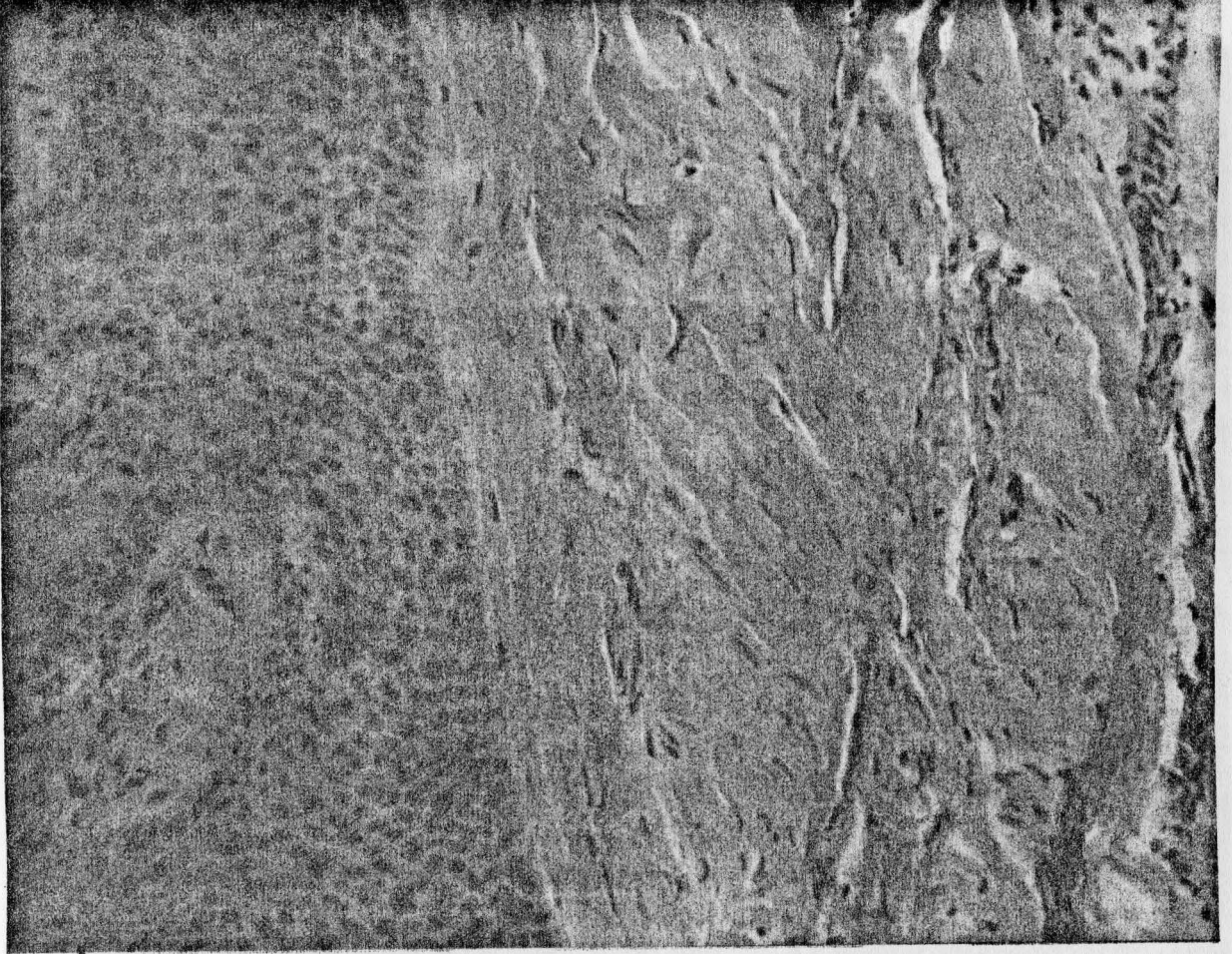
FIGURE 9



Oral epithelium representing normal crevicular tissue. It should be noted there are a few white cells present in the central area of the connective tissue.

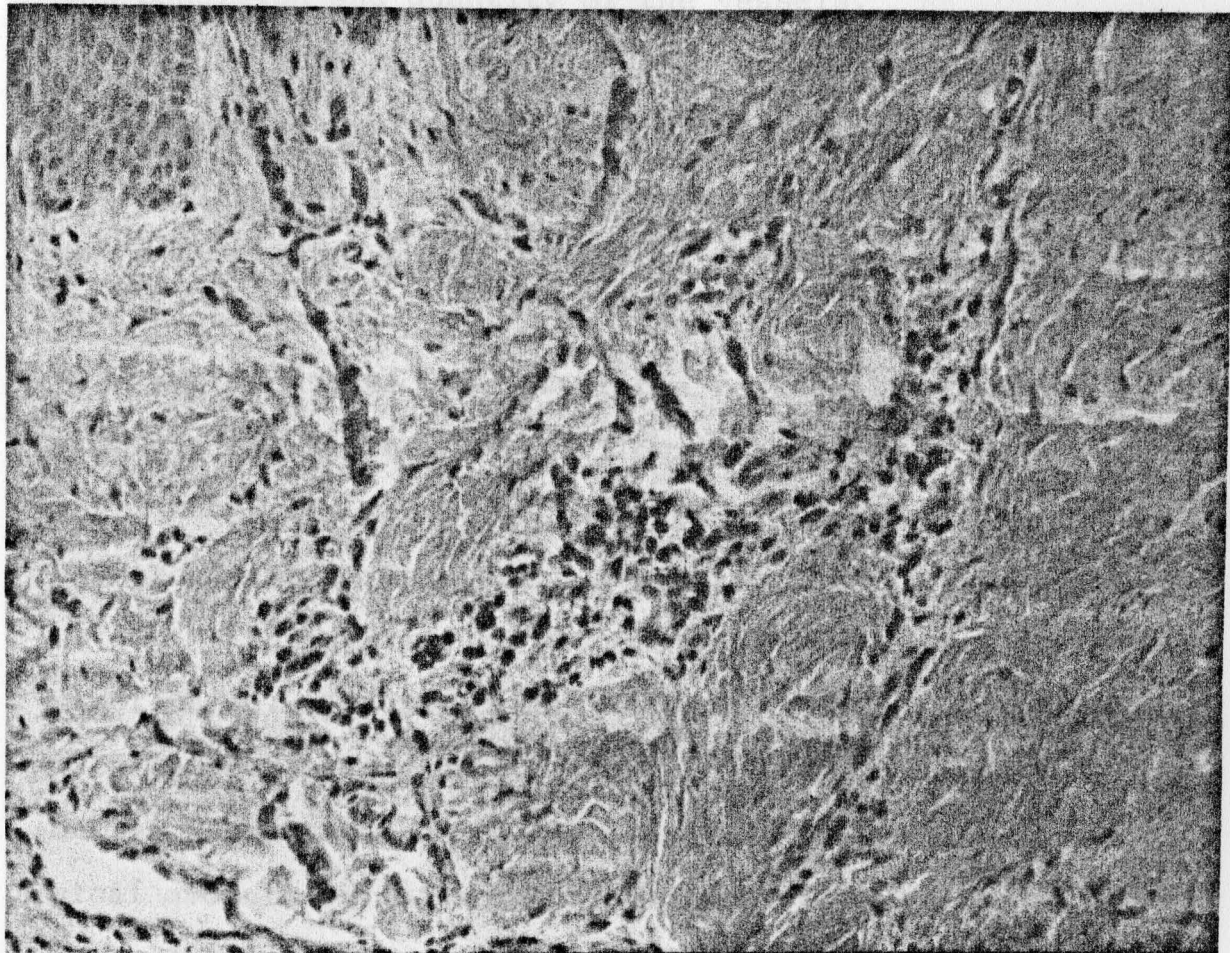
FIGURE 11

FIGURE 10



Enlarged view of oral epithelium representing normal crevicular tissue.

FIGURE 11



An example of a chronic infiltrate. The connective tissue fibers immediately around the infiltrate show a loss of collagen. Peripheral to the infiltrate the fibers appear normal. Plasma cells predominate.

FIGURE 12

dissolution and fiber arrangement of the connective tissue; the condition and quantity of the vessels.

A long term study is desirable involving patients who will need periodontal surgical treatment and extensive restorative dentistry. Fluid readings should be obtained prior to presurgical preparation; after presurgical preparation, before surgery; and, 4 and 10 weeks after surgery. Readings should be done after the restorative phase is complete and periodically following this for at least one year. Controls should be patients who receive surgery and no, or minimal, restorative dentistry.

Confirmation of Newcomb's (1974) research could be of value. The Periotron could be used in conjunction with another parameter to evaluate the severity of inflammation. Because the Periotron is able to detect sub-clinical amounts of inflammation it would be possible to temporarily place extensive restorations. The quantity of fluid could be measured over a period of time to discover whether these restorations are creating an unacceptable amount of inflammation. If the inflammation is unacceptable, it could be possible to correct the restorations before permanent cementation.

Periotron readings should be correlated with other indicies in a long term healing study. This could establish Periotron readings in relation to one or two other well es-

established indicies. The Periotron method is easily accomplished. However, in the author's opinion, it should be used in conjunction with other parameters of gingival health. A strong correlation with another clinical index (eg., G.I.) would provide a reliable objective and qualitative analysis which could be readily accomplished.

CHAPTER VI

SUMMARY AND CONCLUSIONS

There is ample evidence healing occurs during the first four to five weeks following periodontal surgery. The present experiment was designed to monitor healing for ten weeks postoperatively, beginning with postsurgical week four.

Because crevicular fluid has been shown to be a reliable indicator of the severity of inflammation, it was decided this would be the indicator of healing. The Periotron was chosen as the evaluator of fluid volume because of its reported accuracy.

Patients selected for this study presented with periodontal disease which was to be treated surgically. The results of the investigation have revealed:

- 1) The initial healing process (4-5 weeks) is probably the result of regaining the epithelial covering.

- 2) Healing continues at least through week 10 postsurgically. This probably represents the maturity of connective tissue components, with a resultant improvement in tissue tone.

- 3) There is a reduction in the quality of postsurgical healing when patients are treated who show presur-

gical readings above 10 on the Periotron. This reading roughly indicates the high end of a healthy reading. It was shown there is approximately a 50% less chance of attaining "healthy" readings in 10 weeks or less.

4) Presurgical preparation is very important. Ideally it should place the tissue in relative health (reading less than 10) before surgery is performed.

5) The necessity of good presurgical preparation and postsurgical recall and maintenance becomes obvious. This is probably the single most important observation displayed.

6) Crevicular fluid is a reliable estimator of gingival health; however, it should be used in conjunction with other parameters of gingival health.

BIBLIOGRAPHY

- Alfano, Michael C. "The Origin of Gingival Fluid." J. Theor. Biol., Vol. 47, (1974), 127-136.
- Arnold, R., et al. "Alterations in Crevicular Fluid Flow During Healing Following Gingival Surgery." J. Periodont. Res., Vol. 1, (1966), 303-308.
- Bang, J., et al. "Potassium, Sodium, Calcium and Proteins in Human Gingival Fluid." Int. Ass. Dental Research. 49 G.M. Abstr. No. 481, (1970), New York.
- Bhaskar, S.N. Synopsis of Oral Pathology. 2nd ed. Saint Louis: The C.V. Mosby Co., 1965.
- Bissada, N.F., et al. "Circadian Periodicity of Human Crevicular Fluid Flow." J. Periodont., Vol. 38, (1967), 36-40.
- Bjorn, A.L., et al. "Evaluation of Gingival Fluid Measurements." Odont. Rev., Vol. 16, (1965), 300-307.
- Black, G.V. "The Periosteum and Peridental Membrane-Interfibrous Elements of the Peridental Membrane." The Dent. Review, Vol. I, No. 6, (April 15, 1887), 289-302.
- Black, C.V. "The Fibers and Glands of the Peridental Membrane." The Dent. Cosmos, Vol. XLI, No. 2, (February, 1899), 101-122.
- Black, C.V. Special Dental Pathology. 2nd. ed. Chicago: Medico-Dental Publishing Co., 1920.
- Bodecker, Charles F., and Cahn, Lester R. "The Histology and Function of the Gingiva." Dent. Items of Interest, Vol. 53, (February, 1931), 94.
- Bodecker, Charles F. "Dental Erosion: Its Possible Causes and Treatment." The Dent. Cosmos, Vol. IXXV, (Nov., 1933), 1056-1062.

- Borden, S.M., et al. "An Intracrevicular Technique for Monitoring Gingival Crevicular Fluid (GCF) Flow." J. Dent. Res., Vol. 53, Special Issue, Abstract # 482, (Feb., 1974), 175.
- Brandtzaeg, P. and Mann, W.V. "A Comparative Study of the Lysozyme Activity of Human Gingival Pocket Fluid, Serum and Saliva." Acta Odont. Scand., Vol. 22 (1964), 441-445.
- Brankin, G.W. "Determination of Mean Crevicular Fluid Volumes of Males and Females of Different Ages." Unpublished M.S. Thesis, Loyola University, 1977.
- Brenier, J. "The Histological Changes of the Gingival Tissues in Health and Periodontal Disease." Oral Surg. Vol. 3, (1950), 1194-1199.
- Brill, N. "Influence of Capillary Permeability of Flow of Tissue Fluid into Gingival Pockets." Acta Odont. Scand., Vol. 17, (1959a), 23-33.
- Brill, N. "Effect of Chewing on Flow of Tissue Fluid Into Human Gingival Pockets." Acta Odont. Scand., Vol. 17, (1959b), 277-284.
- Brill, N. "Removal of Particles and Bacteria From Gingival Pockets by Tissue Fluid." Acta Odont. Scand., Vol. 17, (1959c), 431-440.
- Brill, N. "Gingival Conditions Related to Flow of Tissue Fluid Into Gingival Pockets." Acta Odont. Scand., Vol. 18 (1960), 421-446.
- Brill, N. "The Gingival Pocket Fluid. Studies of its Occurrence, Composition and Effect." Acta Odont. Scand., Vol. 20, Supple. 32, (1962), 1-115.
- Brill, N. and Bjorn, H. "Passage of Tissue Fluid Into Human Gingival Pockets." Acta Odont. Scand., Vol. 17, (1959), 11-21.
- Brill, N. and Bronnestam, R. "Immuno-electrophoretic Study of Tissue Fluid From Gingival Pockets." Acta Odont. Scand., Vol. 18, (1960), 95-100.

- Brill, N. and Krasse, B. "Effect of Mechanical Stimulation on Flow of Tissue Fluid Through Gingival Pocket Epithelium." Acta Odont. Scand., Vol. 17, (1959), 115-130.
- Cimasoni, G. "The Crevicular Fluid." Monographs in Oral Science, Vol. 3, ed. H.M. Myers, (1974), S. Karger, Basel.
- Daneshmand, H. and Wade, A. "Correlation Between Gingival Fluid Measurements and Macroscopic and Microscopic Characteristics of Gingival Tissue." J. Clin. Periodontal, Vol. 2 (4), (1975), 190-197.
- Egelberg, J. "Cellular Elements in Gingival Pocket Fluid." Acta Odont. Scand., Vol. 21, (1963), 280-287.
- Egelberg, J. "Gingival Exudate Measurements for Evaluation of Inflammatory Changes of the Gingivae." Odont. Rev., Vol. 15, (1964), 381-398.
- Egelberg, J. "Permeability of the Dentogingival Blood Vessels; I, Application of the Vascular Labelling Method and Gingival Fluid Measurements." J. Periodont. Res., (1966), 180-191.
- Egelberg, J. and Attstrom, R. "Comparison Between Orifice and Intracrevicular Methods of Sampling Gingival Fluid." J. Periodont. Res., Vol. 8, (1973), 384-388.
- Eissman, H., et al. "Physiologic Design Criteria for Fixed Dental Restorations." Dent. Clin. N. America, Vol. 15, No. 3, (July, 1971), 543-568.
- Gavin, J.B. and Collins, A.A. "The Occurrence of Bacteria Within the Clinically Healthy Gingival Crevice." J. Periodont., Vol. 32, (1961), 198-201.
- Golub, L.M., et al. "Urea Content of Gingival Crevicular Fluid and Its Relation to Periodontal Disease in Humans." J. Periodont. Res., Vol. 6, (1971) 243-251.
- Golub, L.M., et al. "Collagenolytic Activity of Crevicular Fluid from Periocoronar Gingival Flaps." J. Dent. Res., Vol. 55 (2), (1976), 177-181.

- Golub, L.M. and Kleinberg, I. "Gingival Crevicular Fluid: A New Diagnostic Aid in Managing the Periodontal Patient." Oral Sci. Rev., Vol. 8, (1976), 49-61.
- Gustafsson, G. and Nilsson, I. "Fibrinolytic Activity in Fluid from Gingival Crevice." Soc. Exper. Biol. and Med. Proc., Vol. 106, (1961), 277-280.
- Hara, K. and Loe, H. "Carbohydrate Components of the Gingival Exudate." J. Periodont. Res., Vol. 4, (1969), 202-207.
- Harvey, P. "Elimination of Extraneous Material from the Gingival Crevice." J. Periodont., Vol. 33, (1962), 231-237.
- Jameson, L. "Comparison of the Volume of Crevicular Fluid from Restored and Nonrestored Teeth." Unpublished M.S. Thesis, Loyola University, 1976.
- Kaslick, R.S., et al. "Quantitative Analysis of Sodium, Potassium, and Calcium in Gingival Fluid from Gingiva with Varying Degrees of Inflammation." J. Periodont., Vol. 41, (1970), 93-97.
- Kleinberg, I. and Hall, G. "pH and Depth of Gingival Crevices in Different Areas of the Mouths of Fasting Humans." J. Periodont. Res., Vol. 4, (1969), 109-117.
- Krasse, B. and Egelberg, J. "The Relative Proportions of Sodium, Potassium and Calcium in Gingival Pocket Fluid." Acta Odont. Scand., Vol. 20, (1962), 143-152.
- Kronfeld, Rudolf. Histopathology of the Teeth and Their Surrounding Structures. Philadelphia: Lea and Febiger, 1933.
- Larato, D. "Effects of Artificial Crown Margin Extension and Tooth Brushing Frequency on Gingival Pocket Depth." J. Prosth. Dent., Vol. 34 (6), (Dec., 1975), 640-643.
- Loe, H. "Physiological Aspects of the Gingival Pocket." Acta Odont. Scand., Vol. 19, (1961), 387-395.

- Loe, H. "Reactions of Marginal Periodontal Tissues to Restorative Procedures." Internat. Dent. J., Vol. 18 (41), (Sept., 1962), 759-778.
- Loe, H. "Periodontal Changes in Pregnancy." J. Periodont., Vol. 35, (1965), 209-217.
- Loe, H. "The Gingival Index, The Plaque Index, and The Retention Index Systems." J. Periodont., Vol. 38, (1967), 61.
- Loe, H., et al., "Experimental Gingivitis in Man." J. Periodont., Vol. 36, (1965), 177-187.
- Loe, H. and Holm-Pedersen, P. "Absence and Presence of Fluid from Normal and Inflamed Gingivae." Periodontics, Vol. 3, (1965), 171-177.
- Loe, H. and Silness, J. "Periodontal Disease in Pregnancy. I. Prevalence and Severity." Acta Odont. Scand., Vol. 21, (1963), 532-551.
- Mahajan, Meera. "Histological Evaluation of Gingiva in Complete Crown Restorations." Unpublished M.S. Thesis, Loyola University, Chicago, 1976.
- Mann, W.V., Jr. "The Correlation of Gingivitis Pocket Depth and Exudate from the Gingival Crevice." J. Periodont., Vol. 34, (1963), 379-387.
- Marcum, James S. "The Effect of Crown Marginal Depth Upon Gingival Tissue." J. Prosth. Dent., Vol. 17 (5), (May, 1967), 479-487.
- Maruyama, T., et al. "Morphology of Gingival Capillaries Adjacent to Complete Crowns." J. Prosth. Dent., Vol. 35 (2), (Feb., 1976), 179-184.
- Mc Call, J. "Studies in the Etiology of Approximal and Gingival Caries." J. Dent. Res., Vol. 6, (Dec., 1924), 461.
- Miller, S.C. Textbook of Periodontia (Oral Medicine). York, Pa: The Maple Press Co., 1938.
- Morris, Melvin L. "Artificial Crown Contours and Gingival Health." J. Prosth. Dent., Vol. 12 (6), (1962), 1146-1156.

- Muhlemann, H.R. "Eine Gingivitis Intermenstrualis." Schweiz. Mschr. Zahnheilk., Vol. 58, (1948), 865-885.
- Newcomb, G.M. "The Relationship Between the Location of Subgingival Crown Margins and Gingival Inflammation." J. Periodont., Vol. 45, (1974), 151-154.
- Oliver, R.C., et al. "The Correlation Between Clinical Scoring, Exudate Measurements and Microscopic Evaluation of Inflammation in the Gingiva." J. Periodont., Vol. 40, (1969), 201-209.
- Orban, J.E. and Stallard, R.E. "Gingival Crevicular Fluid: A Reliable Predictor of Gingival Health?" J. Periodont., Vol. 40, (1969), 231-235.
- Periotron Manual. Harco Electronics Ltd., Winnipeg, Canada.
- Ramfjord, S. and Costich, E. "Healing After Exposure of Periostium on the Alveolar Process." J. Periodont., Vol. 39, (1968), 199.
- Ramfjord, S., et al. "Report of the Committee on Scientific Investigation of the American Academy of Restorative Dentistry." J. Prosth. Dent., Vol. 32, No. 2, (August, 1974), 198-221.
- Rudin, H.J., et al. "Correlation Between Sulcus Fluid Rate and Clinical and Histological Inflammation of the Marginal Gingiva." Helv. Odont. Acta, Vol. 14, (1970), 21-26
- Sandalli, P. and Wade, A.B. "Alterations in Crevicular Fluid Flow During Healing Following Gingivectomy and Flap Procedures." J. Periodont. Res., Vol. 4, (1969), 314-318.
- Schroder, H.E. and Listgarten, M. Fine Structure of the Developing Epithelial Attachment of Human Teeth. Karger, Basel, 1971.
- Serres, A. "Essai sur l Anatomie et la Physiologie des Dents, ou Nouvelle Theorie de la Dentition." Paris: Mequignon, Marvis, 1817.
- Shern, R.J., et al. "Crevicular Fluid Flow and Cytopathology as Objective Clinical Measurements." J. Dent. Res., Vol. 53, (Feb., 1974), 175.

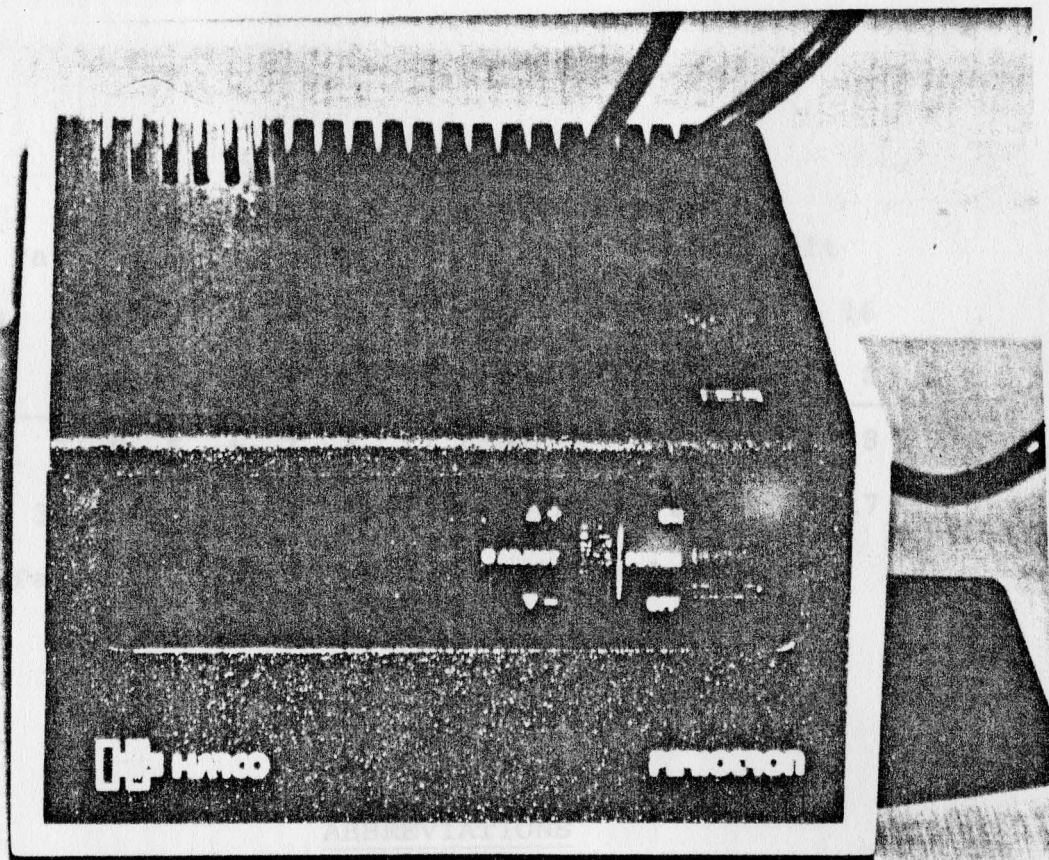
- Shillitoe, E.J. and Lehner, T. "Immunoglobulins and Complement in Crevicular Fluid, Serum and Saliva in Man." Arch. Oral Biol., Vol. 17, (1972), 241-247.
- Squier, C.A. "Permeability of Oral Mucosa." Brit. Med. Bull., Vol. 31 (2), (1975), 169-175.
- Stallard, R.E. "Periodontal Microcirculation and the Gingival Crevicular Fluid." Parodontologie and Academy Rev., Vol. 1 (1), (March, 1967), 34-45.
- Sueda, T., et al. "Histochemical Study of Human Gingival Fluid." Parodontologie, Vol. 20 (4), (Dec. 1966), 141-146.
- Suppipat, N. "The Use of the HAR-600 Gingival Crevice Fluid Meter in Clinical Research." Unpublished M.S. Thesis, University of Oslo, 1976.
- Sutcliffe, P. "A Longitudinal Study of Gingivitis and Puberty." J. Periodont. Res., Vol. 7, (1972), 52-58.
- Toto, P.D. and Sicher, H. "The Epithelial Attachment." Periodontics, Vol. 2 (4), (July/August, 1964). 154-156.
- Trivedi, S.C. and Talim, S.T. "The Response of Human Gingiva to Restorative Materials." J. Prosth. Dent., Vol. 29 (1), (Jan., 1973), 73-79.
- Turner, H., et al. "Visualization of the Microcirculation of the Periodontium." J. Periodont., Vol. 40, (1969), 222-230.
- Waerhaug, J. "The Gingival Pocket: Anatomy, Pathology, Deepening and Elimination." Odont. Tskr., Vol. 60, Suppl. 1, (1952).
- Waerhaug, J. "The Source of Mineral Salts in Subgingival Calculus." J. Dent. Res., Vol. 34, (1955), 563-568.
- Waerhaug, J. "Histologic Considerations Which Govern Where the Margins of Restorations Should Be Located in Relation to the Gingiva." Dent. Clin. No. Am., Symposium on Practical Periodontal Therapy, (March, 1960), 161-176.

- Waerhaug, J. and Steen, E. "The Presence or Absence of Bacteria in Gingival Pockets." Odont. Tskr., Vol. 60, (1952), 1-24.
- Waerhaug, J. and Zander, H. "Reaction of Gingival Tissue to Selfcuring Acrylic Restorations." J. Amer. Dent. Ass., Vol. 54, (1957), 760-768.
- Weinstein, E., et al. "Studies of Gingival Fluid." Periodontics, Vol. 5 (4), (July/August, 1967), 161-166.
- Windhager, E.E., et al. "Single Proximal Tubules of the Necturus Kidney, III. Dependence of H₂O Movement on NaCl Concentration." Amer. J. Physiol., Vol. 197, (1959), 313-318.
- Yuodelis, R., et al. "Facial and Lingual Contours of Artificial Complete Crown Restorations and Their Effects on the Periodontium." J. Prosthet. Dent., Vol. 21 (9), (Jan., 1973), 61-66.
- Zachrisson, J. and Schultz-Hautdt, P. "A Comparative Histological Study of Clinically Abnormal and Clinically Inflamed Gingiva from the Same Individual." Odont. Tdskr., Vol. 76, (1968), 179-197.

APPENDIX

DESCRIPTION OF THE PERIOTRON

The Periotron contains two jaws that function like the plates of an electrical condenser . When a dry strip is inserted, the capacitance is at its maximum but, with the appropriate electronic circuits, registers zero on the instrument. Insertion of a wetted strip reduces the capacitance and results in a rise in the reading. The rise is directly proportional to the area wetted, i.e., to the volume of fluid collected on the paper. The jaws have an additional role of considerably reducing sample evaporation, which is extremely important with minute samples. The Periotron is capable of accurately measuring the fluid on the paper strip whether an extracrevicular or an intracrevicular technique is used. Because of the ease of measurement, the time of insertion in the intracrevicular technique can be reduced to as little as two or three seconds, which should reduce the irritation considerably.



GCF - Gingival Crevicular Fluid
 GCF - Mean Gingival Crevicular
 Fluid
 N - Number in sample
 t - Result of t test
 S.D. - Standard Deviation
 P - Probability

THE PERIOTRON

FIGURE 7

TOOTH NUMBERING SYSTEM

Patients Upper Right								Patients Upper Left							
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
32	31	30	29	28	27	26	25	24	23	22	21	20	19	18	17
Patients Lower Right								Patients Lower Left							

ABBREVIATIONS

- GCF - Gingival Crevicular Fluid
 GCF - Mean Gingival Crevicular Fluid
 N - Number in sample
 t - Result of t test
 S.D.- Standard Deviation
 P - Probability

TABLE 1

Address 68 City _____ State _____ Zip _____ Date _____ Time _____

Home Phone _____ Office Phone _____ Birthdate _____ Age _____

TOOTH #	MOBILITY	POCKET DEPTH mm.		GINGIVAL INDEX	CREVICULAR FLUID VOL.	INTERPROXIMAL		MEAN C.F.V.	WEEK OF STUDY						TISSUE -GRAI	
						DISTAL	MESIAL		0	4	6	8	10	12		
					→											
					→											
					→											
					→											
MEANS																

1. OPERATOR SHOULD BE SAME FOR C.F.V. MEASUREMENT. 2. MORE THAN ONE OBSERVER SHOULD GRADE G.I. AND MOBILITY.

MOBILITY SCALE

- 0 - Absence of Mobility
- 1 - Slight Mobility
- 2 - Moderate Mobility
- 3 - Pronounced Mobility
- 4 - Severe Mobility with Depressibility

GINGIVAL INDEX

- 0 - Normal
- 1 - Mild Inflammation, Slight Redness, Slight Edema, No Bleeding on Probing
- 2 - Moderate Inflammation, Redness, Edema and Glazing, Bleeding on Probing.
- 3 - Severe Inflammation, Redness and Edema, Ulceration, Tendency for Spontaneous Bleeding

MICROSCOPIC GRADING OF INFLAMMATION OF TISSUE SAMPLE - H and E SECTIONS

- Zero (0) - Absence of Inflammatory Cells
- + (1+) - Slight Inflammatory Reaction
- ++ (2+) - Moderate Inflammatory Reaction
- +++ (3+) - Pronounced Inflammatory Reaction
- ++++ (4+) - Severe Inflammatory Reaction

TOOTH NUMBERING SYSTEM

Patient's UR	Patient's UL
5 4 3 2 1	1 2 3 4 5
5 4 3 2 1	1 2 3 4 5
Patient's LR	Patient's LL

Fixed Prosthodontics Department
Crevicular Fluid Volume Measurements

PATIENT CONSENT FORM

The purpose of this study is to determine if there is an increase in the amount of fluid flowing from the space between the teeth and gums after placing a crown (cap).

This preliminary study will utilize an instrument that measures very small amounts of fluid and possibly can be used to detect gum disease before it has progressed to an advanced stage.

The procedure will involve isolating the teeth with sterile cotton rolls, then placing a small sterile piece of filter paper next to the teeth near the gums for about three (3) seconds. Several teeth will be measured in this manner. This will not produce any discomfort or have any ill effect whatsoever on the gums or the teeth. The entire procedure, including filling out the questionnaire, should take approximately twenty (20) minutes.

If at any time during the procedure you want to withdraw your participation in this study, you are free to do so.

I HAVE READ THE ABOVE INFORMATION AND WILL PARTICIPATE IN THIS
STUDY. PARTICIPANT'S SIGNATURE _____

DATE _____

APPROVAL SHEET

The thesis submitted by David L. Koth, B.S., D.D.S. has been read and approved by members of the Department of Oral Biology.

The final copies have been examined by the director of the thesis and the signature which appears below verifies the fact that any necessary changes have been incorporated, and that the thesis is now given final approval with reference to content, form, and mechanical accuracy.

The thesis is therefore accepted in partial fulfillment of the requirements for the Degree of Master of Science.

5/5 /77
Date

William Palmer
Signature of Advisor