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The Effect of Fixed Prosthodontics on Crevicular Fluid Volume

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THE EFFECT OF FIXED PROSTHODONTICS ON CREVICULAR FLUID VOLUME

by

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VITAE

Clifford A. Zmick was born in Burlington, Wisconsin, on July 24, 1950. Shortly thereafter he became a resident of the City of Chicago where he attended elementary and secondary schools. He was graduated from St. Ignatius College Prep in June, 1968.

After completing a program of preprofessional studies, he received a Bachelor of Science Degree in June, 1972, from the University of Notre Dame, Notre Dame, Indiana.

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In June, 1976, he entered Graduate School at Loyola University to pursue a specialty in Fixed Prosthodontics and a Master of Science Degree in Oral Biology. Currently he is also a clinical instructor at the School of Dentistry.

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CHAPTER I

INTRODUCTION AND STATEMENT OF PURPOSE

The dental profession has known for many years extensive restorative therapy is accompanied by unfavorable supportive tissue responses. This is particularly true when the gingival margins of the restorations are placed below the crest of the gingiva. To what degree these effects are detrimental has been the subject of this thesis.

It is believed the instrumentation during tooth preparation, soft tissue manipulation, and temporization has adversely affected the homeostasis of the marginal gingival tissues. Jameson (1976) stated a certain degree of inflammation is apparently indigenous to restorations with subgingival margins.

It is the purpose of this investigation to determine the inflammatory changes in the marginal gingiva prior to, during, and after the placement of a fixed partial denture. This study will hopefully help to establish a means of objectively evaluating inflammation associated with fixed prosthodontics. Longitudinal studies could also be designed to lend credence to various techniques utilized in fixed prosthodontics.

The quantitative method of crevicular fluid measurement was selected to monitor tissue inflammation. The presence of gingival inflammation has been shown to be accompanied by a corresponding increase in crevicular fluid (Egelberg, 1964; Golub and Kleingerg, 1976). This positive correlation between the volume of crevicular fluid and the degree in inflammation (Valazza, 1972) made this study possible.

The recent development of a crevicular fluid measuring device has made the measurement of the fluid volume more reliable. The Harco Periotron* measures minute fluid volumes by relating electronically a reduction in capacitance. Two

sensors are calibrated with a dry standardized filter paper strip. A fluid containing strip then alters the capacitance between the sensors and the volume of fluid is recorded.

With the use of this refined monitoring device, the crecicular fluid increase as a measure of inflammation can be detected before clincial signs are evident.

In summary, it was the goal of this project to (1) study the alteration of the crevicular fluid of the marginal gingival tissue during the tissue manipulation for restorative techniques; and (2) to monitor the tissue recovery from inflammation and irritation after form and function has been restored.

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*"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.

CHAPTER II

REVIEW OF THE LITERATURE

GINGIVAL CREVICULAR FLUID

A. THE RELATIONSHIP TO INFLAMMATION

The gingival sulcus is the shallow groove between the tooth and the normal gingiva. The bottom of this sulcus is formed by the free surface of the junctional epithelium and the sulcular termination of the oral epithelium (Schroder and Listgarten, 1971). Fluid in this crevice was described as early as 1817 (Serres), and was attributed to glandular secretion by Black (1887). Studies by Boedecker and Cahn (1931) refuted the presence of glands from histologic evidence. Miller (1938) was the first to define the term crevicular "exudate" and believed the exudate to be a clinical sign of incipient periodontal disease. The term "exudate" was used rather loosely in the research of McCall (1924) who attempted to correlate tooth erosion and the acidity of crevicular fluid.

Gingival crevicular fluid was usually considered an inflammatory exudate until the perceptive studies of Waerhaug (1952) demonstrated the dynamic nature of the gingival sulcus. He perfused the healthy sulci of young dogs with India ink. Most of the ink particles were removed after two hours indicating increased transudation of fluid. Leucocyte emigration through the sulcular epithelium was also found giving further support to transudation.

In a further study, pure cultures of pathogenic bacteria were placed in the apparently bacteria-free crevice of young dogs (Waerhaug and Steen, 1952). Histologic tissue reaction was noted over a forty-eight hour period. This resulted in the conclusions: 1) Fluid was always present in the gingival crevice; 2) Healthy, calculus free gingival crevices were sterile, and 3) Bacteria in the crevice caused necrosis of the epithelium and inflammation of the connective

tissue which lead to the subsequent exudate formation.

Gavin and Collins (1961) questioned this sterility. The presence of microorganisms was demonstrated in a majority of cases. They theorized "The entry of organisms into the crevice is favored by the capillary action of the crevice, by the trauma and movement of mastication, and by the constant bathing of the crevice by saliva with its extremely numerous organisms."

Although this discounted Waerhaug's findings of a sterile gingival sulcus, the question as to whether the gingival fluid was an exudate or transudate still had not been resolved. A study by Turner et al. (1969) involved the microcirculation of healthy sulci in young dogs. A 1% solution of Patent Blue V was injected in one carotid artery and Pelikan carbon black in the other. The Patent Blue V passed quickly through the epithelial attachment whereas Pelikan carbon black which measured 200 to 500 Å did not pass through intact capillary walls. Thus the investigators concluded crevicular fluid was a transudate in agreement with Waerhaug, although the precise transmission was not described.

Additional agreement with Waerhaug was found in a study by Löe (1961). He recorded the turnover rate of epithelial cells and the presence of leucocytes in the gingival fluid of dogs. The marginal cuff of 49 areas was sealed with an alcoholic solution of colophony (rosin) and an acrylic crown locked in place with amalgam. He found, by histologic examination at short (1-6h) and long (12-48h) intervals, neutrophilic leucocytes migrated through the epithelial lining under physiologic conditions. "There is a continuous transudation of tissue fluid into the clinically normal gingival pockets."

These are the major works in support of crevicular fluid as a serum transudate. On the other hand, Brill's work (1959) on dogs and humans tended to illustrate continual presence of fluid as a transudate and inflammatory exudate. He injected Evans blue which binds to plasma albumin and gamma-globulin and

assessed crevicular fluid flow under the effects of histamine, mechanical stimulation, and inflammatory state. The results showed the Evans blue was recoverable from the gingival crevice. This demonstrated the role of increased vascular permeability in the production of crevicular fluid. Other experiments were conducted by Brill to lend greater credibility to this role.

In one, human subjects chewed paraffin and demonstrated increased crevicular flow. This was due to mechnical stimulation on the gingival vascular bed which caused escape of plasma.

More data in this direction was provided by Brill and Bjorn (1959) who introduced sodium fluorescein into human subjects by oral administration. Their result demonstrated "the epithelial lining of gingival pockets are generally permeable to the molecules of fluorescein sodium whether clinically healthy or inflamed." More fluorescein was, however, recovered from inflamed gingiva. Herein the question of crevicular fluid being a transudate or an exudate was re-examined: Was the shift of fluid from subepithelial compartments through epithelium physiologic or pathologic?

An answer came from Brill and Krasse (1959) with a study involving dogs injected with sodium fluorescein. Mechanical stimulation in the form of brushing demonstrated increased crevicular fluid flow (Brill 1959). Ten minutes after ceasing stimulation, the flow rate returned to normal. The vascular reaction to stimulation was increased permeability allowing plasma to escape more freely until stimulation ceases and adaptation occurred. This vascular reaction was seen in the early stages of inflammation (Brill 1959).

Another hypothesis was related to the connective tissue. Tissue manipulation may cause the gel-like structure of the connective tissue to become edematous, dissolving the ground substance; thus a sol is created out of the gel substance. Their conclusions were "although reactions in the vessels, the

connective tissue, and the epithelium have been mentioned separately they are not mutually exclusive." No confirmation of the interrelationships have been evidenced in the literature, but the explanation has merit.

As the controversy of whether the crevicular fluid is a transudate or exudate continued, Mann (1963) collected fluid and correlated fluid volume with pocket depth and gingivitis in 27 human subjects. His results indicated a positive correlation between pocket depth, gingivitis, and fluid flow. A conclusion of the study stated inflammation and fluid flow was more interrelated than pocket depth and fluid flow. More comparisons of crevicular fluid and inflammation tended to define this fluid as an exudate.

Conclusive evidence to confirm gingival fluid as an exudate was offered by Löe and Holm-Pederson (1965) who measured 118 adults. Their technique of collection was less traumatic than others. Utilizing biomicroscopic techniques they noted capillary compression when inserting filter paper for fluid collection. They suggested this may have been the reason for finding fluid in healthy gingival crevices. Sulcular epithelium of healthy human gingiva did not exhibit fluid flow, and this was contrary to earlier reports (Brill, 1959; Brill and Krasse, 1959). They also concluded fluid flow is indicative of inflamed tissue and can be ascertained prior to clinical observations.

Similarly, flame photometry was used to determine the relative proportions of sodium,potassium, and calcium found in crevicular fluid (Krasse and Egelberg, 1962). The findings indicated an increase in the proportion of potassium from clinically healthy gingiva. They supposed this to be intracellular potassium augmenting the extracellular fluid as it passed into the gingival crevice. Their conclusion was "gingival pocket fluid cannot be regarded as a simple filtration product, but rather as an inflammatory exudate."

Egelberg (1963) compared the celluar 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 leucocytes, etc.). There was, however, an increase in the number of inflammatory cells in fluid samples from inflamed gingiva. These findings seemed to support his earlier research with Krasse (1962) that "fluid in healthy pockets may be regarded as an inflammatory exudate."

Further investigations by the same author (Egelberg, 1966) led to the conclusion that 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. This conclusion left little doubt that the production of fluid was intimately related to an abnormal increase in permeability of the vessels of the crevicular plexus.

The state of health or inflammation of the whole marginal region played a major role in the permeability of the junctional and crevicular epithelium. It has been shown by Thilander (1964) the permeability of the skin and mucous membranes is altered by chemical or mechanical stimulation. Furthermore, the pocket epithelium did not constitute the same effective barrier to certain substances, as do other epithelia. It was thin and had no stratum corneum (Thilander, 1964). The marginal region was regularly exposed to mechanical stimuli, especially during mastication. In addition, the constant presence of leucocytes even in an otherwise normal crevicular epithelium was a common finding; these cells were probably attracted to the area by microorganisms or their products (Löe, 1961). All of the above facts related crevicular fluid as an exudate rather than a transudate. The exact mechanism by which substances pass from the subepithelial tissue spaces through the lining epithelium has received

little attention (Cimasoni, 1974).

A study of permeability of the oral mucosa was completed by Squier and Johnson (1975) and these investigators reported oral mucosa was not a highly permeable membrane. Most substances were passed across by simple diffusion in which "the rate of penetration is directly proportional to the concentration of penetrant." The presence of plaque was a cause for an inflammatory response and thus the permeability of the sulcular epithelium was enhanced.

Oliver, et al., (1969) presented further correlations between gingival inflammation, fluid exudate, and gingival index (Löe and Silness, 1967). Results showed a direct proportion between gingival index and crevicular fluid volume. When there was no clinical evidence of inflammation, there was very little or no exudate.

In conclusion, a study done by Alfano (1974) proposed a theory which explains whether gingival fluid was a physiologic transudate or an inflammatory exudate. His explanation was the origination of gingival fluid via two distinctly separate mechanisms: the generation of a standing osmotic gradient generated by macromolecular byproducts 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." Although this treatment has many followers (e.g. Golub and Kleinberg, 1976) there was no convincing data. Currently the crevicular fluid is best referred to as an inflammatory exudate.

B. THE MEASUREMENT AND METHOD OF COLLECTION

Essentially two techniques have been utilized, with minor variations, for the collection of the crevicular fluid: the method of filter paper strips and

that of capillary tubes (Cimasoni, 1974). Most of the time, the collection had been made along the maxillary anterior teeth, where the contamination by saliva was least probable.

In the original work on the dog, Brill and Krasse (1958) utilized filter paper strips. Leirskar (1971) found in a chromatographic study the paper quality was an important factor when measuring crevicular fluid volume. Different paper types should not be compared because gingival fluid exudate exhibits increased mobility of protein solutions with increasing dilution. Löe and Holm-Pederson (1965) proposed to place the extremity of the strip at the entrance to the sulcus. This technique was employed by Egelberg (1966) and was termed the orifice method.

Brill (1962) described his technique of gingival fluid recovery as inserting the filter paper stirp "until minimum resistance was felt." This method was employed by Mann (1963), Sueda et al. (1966), and Egelberg (1964).

The two above techniques were termed the intracrevicular methods and the volume collected has been evaluated four ways.

Staining of the strip with an alcoholic solution of ninhydrin has been employed by Brill (1962), and Orban and Stallard (1969). After staining, most investigators had measured the length of paper stained. This planimetry was also used by Mann (1963).

In those investigations employing sodium fluorescein, fluorescence of the strips was observed under ultraviolet light (Brilla and Bjorn, 1959; Mann, 1963). No quantitative measurements were done. Weinstein et al. (1967) demonstrated fluorescein labelling was 100 times more sensitive to proteins than was ninhydrin staining.

Intracrevicular sampling was also evaluated by weighing before collection in a sealed microcentrifugation tube and by subsequently remeasuring

after collection (Valazza et al., 1972).

Another method recently developed is the harco Periotron which was evaluated by Suppipat (1976) and was found to be a sensitive and objective method for evaluation of the marginal condition. He proposed calibration of the machine with each use by means of a dry sterile filter paper strip. In this study, he employed the orifice method (Löe and Holm-Pederson, 1965) and found: 1) none or very little fluid is found in clinically healthy gingiva, 2) "gingival inflammation has a stronger relationship with gingival fluid flow than has pocket depth" (in agreement with Mann, 1963), and 3) there was a higher success rater after scaling when the pocket depth was 3 mm. or less.

Any fluid collected by this method runs the risk of variation of fluid flow during the collection procedure. Mann (1963) performed a series of 10 collections on 3 patients and observed an increase of fluid flow with time. It was observed the flow remained constant up to 5 and 10 minutes, but increased by about 20 percent in the following 10 minutes. This increase in flow was probably due to the irritation and increase in vascular permeability brought about by the intracrevicular sampling technique (Egelberg, 1966).

Another technique for sampling fluid with filter paper was termed the extracrevicular method. Herein the strip is placed in such a way as to fit closely to the tooth surface, the gingival margin, and the attached gingiva, thereby bridging the entrance of the gingival sulcus (Löe and Holm-Pederson, 1965). This procedure minimized any increase in vascular permeability due to the mechanical irritation of filter paper insertions.

Borden et al. (1974) also utilized the Harco Periotron studied by Suppipat (1976) to collect fluid from 10 subjects and a total of 59 gingival crevices. They employed both the intracrevicular and extracrevicular techniques.

The quantities of fluid collected were compared for correlation with the Gingival Index (Löe and Silness, 1967) and gingival pocket depth. The authors further investigated the effect of repeated intracrevicular placement of filter strips. These 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 indicated the extracrevicular technique of collecting gingival fluid was unsatisfactory for slightly inflamed gingiva; fluid may have difficulty flowing out of such crevices."

Although the data collected by Borden et al. (1974) gave reason for these conclusions, they were intrinsically at odds with Egelberg (1966). He showed the introduction of a filter paper strip in a healthy crevice did indeed represent a mechanical stimulation sufficient to cause an increase in vascular permeability. If so much as a single insertion of filter paper caused such a reaction, then how much more will be caused by six insertions at 30 second intervals?

In argument with this statement, Shern et al. (1974) compared ninhydrin staining with the crevicular flow meter. They concluded "precision, accuracy and reliability of measuring fluid flow proved greater using a flow meter than using the ninhydrin technique."

The greatest hazard in the collection of gingival fluid by any means was the contamination by saliva. Cimasoni (1974) suggested collection be made along the upper anterior teeth where contamination by saliva was least probable. Weinstein et al. (1967) performed immuneoelectrophoresis of samples of gingival

fluid which they had collected in humans by means of filter papers: immunoelectrophoresis of gingival fluid showed precipitation lines when reacted with antihuman sera. This indicated the presence of several serum proteins in gingival fluid. "When gingival fluid was reacted with antiserum to parotid saliva and antiserum to submaxillary saliva no precipitations accrued, thus indicating the absence of salivary proteins in gingival fluid."

Another technique for the collection of crevicular fluid utilized micropipettes. In 1962, Krasse and Egelberg used micropipettes in their study on the sodium and potassium content of the gingival exudate. Mann (1963) and Egelberg (1963) also employed this method.

In a study of the precise evaluation of crevicular fluid, Kaslick et al. (1968) collected fluid by capillarity and then centrifuged the contents for 3 minutes to remove cellular elements and debris. He then determined the fluid volume by a wash technique with a known amount of water.

Sueda et al. (1969) also employed this technique of microcentrifugation. As opposed to the wash technique of Kaslick, the volume of fluid supernate was recovered from the centrifugation tubes by using calibrated aspirating micropipettes.

Still another method collected crevicular fluid by means of gingival washings which completely shielded the gingiva from salivary contamination. Oppenheim (1970) constructed a hard acrylic appliance which covered the entire area of each margin of each tooth. Channels were provided within this appliance for sulcular rinsing with a known quantity of fluid. It was suggested the increased amount of fluid after wash procedures was due to crecivular fluid recovery. There was little possibility of salivary contamination.

Regardless of the method for gingival fluid collection, the flow of crevicular exudate has been shown to follow a circadian pattern (Bissada et al.,

1967; Suppipat, 1976). Considerable variation in the amount of fluid between different individuals, as well as in the same individual at different times of sampling, was demonstrated by Bissada et al. (1967). The results indicate, as a group phenomenon, that fluid flow "was found to increase gradually from 0600 to 2200 h, and to decrease afterwards. At 2200 h the average amount of gingival fluid was about 1.5 times higher than that recorded at 0600 h, the difference being statistically significant." The circadian pattern in the amount of fluid followed the daily body temperature variation. The maximum of crevicular flow followed the evening peak in body temperature by about 4 hours.

C. THE COMPOSITION

Crevicular exudate was found to be a fluid consisting of five ingredients: 1) cellular elements, 2) electrolytes, 3) organic compounds, 4) enzymes, 5) metabolic and bacterial products (Cimasoni, 1974). All of these elements were reported to interreact simultaneously. Great difficulty was experienced when an explanation of a single phenomenon became necessary. Nonetheless, an evaluation of these determinants was attempted.

The epithelium which faced the hard dental structure was dying epithelium. Beagrie (1963) determined mitotic activity in the junctional and sulcular epithelium was at a higher rate than the oral epithelium. This implied the presence of the shed cells in gingival fluid samples.

This fact was confirmed and studied by Lange and Schröder (1971) who utilized smears to examine the specimens with cytochemical techniques. These samples were collected; 1) with the Papanicolau technique, and 2) with fixed, centrifuged suspensions prepared for electron microscopy. The results showed the junctional epithelium cells at the level of the sulcus bottom and coronal to it were characterized by various stages of nuclear karyolysis. Vacuoles

were also shown to originate from former cellular organelles. Junctional epithelial cells undergo a progressive necrosis during their course of exfoliation, from beneath the sulcus bottom into the sulcular area.

Inflammation affected both the rate of renewal of the gingival epithelium and the structural characteristics of the desquamating cells. In man, the study of Marwah et al. (1960) has shown a marked stimulation of cell division in the presence of dense accumulations of inflammatory cells in the connective tissue.

In addition to the epithelial cells, leucocytes have been present in saliva since Colonius (1950) showed a very small leucocyte count in edentulous patients, as compared to persons with teeth. Sharpy and Krasse (1960) found leucocytes constitute an average of 49% of the somatic cells from the gingival sulcus. Also, the number of leucocytes in gingival fluid increased predictably with the intensity of the inflammatory reaction (Egelberg, 1963). Salivary leucocyte level has been positively correlated with the degree of gingivitis by Woolweaver et al. (1972).

A more detailed analysis of the relative proportion of various types of leucocytes collected at the marginal border was published by Attstrom (1970). Sampling of crevicular leucocytes was done by "Styroflex" strips applied to the teeth and later stained. The differential counts showed 95-97% neutrophils, 1-2% lymphocytes, and 2-3% monocytes. The absolute number of cells increased with the intensity of the inflammatory process but the proportions remained the same (Egelberg, 1963).

The presence of bacteria has been known to attract these leucocytes and the investigation of Kaess and Llory (1972) confirmed the fact bacteria were cultured from crevicular exudate (Gavin and Collins, 1961). This study compared cultures obtained from crevicular material with adjacent plaque material. The

number of positive cultures from crevicular fluid was found to be significantly higher. This difference was accounted for by actinomyces, fusobacteria, veillonella, and Bacteroides melaninogenicus.

Electrolytes were also found in the sulcular fluid. There was no concrete consensus available which showed agreement in regards to the measurement and/or concentration. Kaslick and Matsue investigated this entity separately in regards to sodium, potassium, and calcium. Most of the publications agreed the crevicular fluid contained a significantly higher amount of sodium than serum. Also it was stated that sodium concentrations tended to increase in cases of more severe inflammation (Matsue, 1967; Kaslick, 1970). In regards to potassium, Matsue (1967) reported the potassium concentration was much higher in the crevicular fluid than in serum. The potassium content of the crevicular exudate also tended to increase in cases displaying advanced inflammation (Bang et al., 1970). Results obtained by other investigators were inconsistent (Kaslick et al., 1970).

Krasse and Egelberg (1962) compared the sodium: potassium ratio of crevicular fluid to serum. If the fluid passed through intact tissues, it contained the same proportions of sodium and potassium as plasma and extracellular fluid. If the fluid passed through damaged tissues, a decreased sodium: potassium ratio was found because of the accumulation of intracellular potassium from the disrupted cells. The resulting ratio was 3.9 whereas the normal ratio is about 28. From these facts, the authors believed crevicular fluid was an "exudate." A comparison of this ratio to the severity of gingival inflammation rendered inconsistent results (Kaslick et al., 1970; Bang et al., 1970).

The presence of calcium and other ions was studied by Kaslick et al. (1970), with insignificant conclusions. The only action perceived was calcium in the crevicular fluid helped trigger precipitation of mucoprotein along the enamel

surface. The tendency for deposits increased near the sulcular area (Saxton, 1973).

In addition to the presence of cellular elements and electrolytes, crevicular fluid has been found to contain extensive organic components. The greatest concentration of these are carbohydrates and proteins (including enzymes). These were attributed to the host. Metabolic activity under the influence of the local microflora was discussed in a later section.

Hara and Löe (1969) conducted a quantitative carbohydrate investigation on glucose, hexosamine and hexuronic acid in crevicular fluid. The method of collection followed the Brill technique and exudate was measured by a weighing procedure (Valazza, 1972). Exudate glucose content showed values 3-6 times higher than serum from the same patient. It decreased in cases of noninflamed gingiva. Their conclusions contained no further correlations, and the activity of the microflora in the immediate vicinity was not readily controlled.

More data has been available in regards to the presence of proteins in gingival fluid. Bang and Cimasoni (1971) confirmed the inflammatory nature of the crevicular "exudate" due to its capacity to carry high molecular weight compounds, i.e., proteins. Increased capillary permeability was cited as the cause for the presence of gingival exudate (Bang and Cimasoni, 1971; Egelberg, 1966).

Brandtzaeg (1965) and Gustafsson and Nilsson (1961) provided further evidence by reporting the presence of fibrinogen in gingival exudate. This protein was not found in nonvascular fluids unless inflammation was present.

Histochemically, it was determined by Sueda et al. (1966) that crevicular exudate contained proteins similar to those found in serum. These authors utilized filter paper strips soaked with gingival fluid and serum. After staining, nearly identical results were reported.

Quantification of this protein was done by Brill and Brönnestam (1960)

by employing immunoelectrophoresis. It was found the concentration of protein in gingival exudate was as low as 1:10 of that of serum.

In opposition to these results, Mann and Stoffer (1964) utilized paper electrophoresis and obtained densitometric tracings of crevicular fluid collected by the capillary technique. The relative concentration of 5 proteins (gamma, beta, alpha 2 alpha 1, globulin, and albumin) were found to be identical in gingival fluid and serum from the same patient. Holmberg and Killander (1971) supported this evidence by a technique of radial immunodiffusion. They determined "immunoglobulins G, A, and M are present in gingival fluid in concentrations comparable to those of serum."

In further search of proteins in crevicular fluid, there has been extensive investigation of enzymes. Few have been the subject of quantitative research, but no less than 35 were identified in the lysosomal fraction of various cell types. Only six types have been the subject of intensive research and these are the following: 1) acid phosphatase, 2) beta-Glucuronidase, 3) lysozyme, 4) cathepsin E, 5) proteases, and 6) alkaline phosphatase (Cimasoni, 1974). Most of these enzymes in gingival exudate originated from either the various gingival components or from the abundant bacterial flora of the marginal region. The proportion of production by either the host or by the microorganism was not known (Cimasoni, 1974).

Gustafsson and Nilsson (1961) initiated enzyme research in crevicular fluid by demonstrating the presence of a fibrinolytic system. They collected crevicular exudate by means of triangular pieces of filter paper and determined fibrinolytic activity by placing the papers on fibrin plates. Lysis obtained around the sample detected the presence of plasmin (a protease). Crude cultures of bacteria collected along the marginal region did not produce any lysed areas. Therefore, they concluded plasmin was present in crevicular exudate, and was not

present in the bacterial proteases.

In a study to determine if collagenolytic activity was detectable in sulcular fluid, Golub et al. (1974) collected fluid and placed it on a treated collagen gel. He related this data to gingival index and pocket depth. Results exhibited gingival fluid samples which had no visible lysis with a mean crevicular fluid value of 0.16 $\pm$ 0.02 ul. Positive activity was seen with fluid values of 0.26 $\pm$ 0.05 ul. the Gingival Index (Löe and Silness, 1967) and pocket depths correlated positively with these results. Their conclusions were "collagenolytic activity in human gingival fluid tends to be associated with gingiva exhibiting increased inflammation."

The activity of several enzymes were tested by histochemical technique by Sueda et al. (1967). They qualitatively compared acid phosphatase in the crevicular fluid with serum and saliva. Acid phosphatase was usually investigated as lysosomal marker (e.g. collagenase). Gingival fluid consistently contained 10-20 times more acid phosphatase than blood serum.

Crevicular acid phosphatase was also studied by Tynelius-Bratthal, and Attstrom (1972). Dogs with inflamed gingiva were evaluated and then these same dogs were treated by cleaning and polishing until the gingival health was reestablished. The results showed more than twofold reduction in acid phosphatase with a concomitant reduction in crevicular leucocytes and volume of gingival fluid.

Another enzyme, beta-Glucuronidase, has been associated with polymorphonuclear leucocytes and thereby considered a determinant of inflammation. Bang et al. (1970) collected gingival exudate from 23 patients with periodontitis, and in assessing this enzyme, they found beta-Glucuronidase concentration to be 10 times higher than serum. Also, a positive correlation was found between this enzyme concentration and the depths of the periodontal pockets.

Relevant to this discussion was research by Schultz-Haudt and Sherp (1955) in which beta-Glucuronidase was identified from cultures of streptococci isolated from human gingival crevices. The origin of this enzyme remained singularly elusive. Most of the other enzymes found in gingival fluid could be produced both by bacteria and by cells of the host. Persistently, then, investigators were confronted with the difficult task of making a distinction between the two possible sources. This controversial issue led to interesting investigations regarding the metabolic and bacterial products found in crevicular fluid.

Urea was demonstrated in gingival fluid by Golub et al. (1971). Its concentration in the fluid was shown to be several times higher than serum and saliva. The presence of urea in crevicular fluid could be responsible for the elevated pH in the gingival crevice, through the production of ammonia by the crevicular microorganisms.

The presence of bacterial endotoxins in gingival exudate has been examined by Simon et al. (1970). They believed the severity of gingival inflammation was positively correlated with the concentration of endotoxin. This approach was confirmed by a histologic study in 1971.

Green and Kass (1970) demonstrated gingival exudate to possess antibacterial factors. Waerhaug and Steen (1952) had suggested this phenomenon earlier. Utilizing rabbits and radiolabelled samples of Sarcinia marcescens, Green and Kass showed complete bacterial elimination after 2 hours.

In contrast to these findings, Collins and Gavin (1961) failed to show any bacteriocidal activity in crevicular fluid. These authors introduced gingival exudate to previously inoculated blood agar plates and observed no reaction. Some question was therefore present as to the existence of a mechanism in situ.

RELATIONSHIP OF RESTORATIVE MARGINS AND CONTOUR TO GINGIVAL HEALTH

It has been established in the preceding section of the review of literature the volume of crevicular fluid was determined by the state of inflammation of the periodontium. Among the multitude of contributing factors, the morphological contours of restorations had been one of the primary parameters of responsibility for the dentist. Variations of tooth contour and margin placement were considered in order to protect the free gingival margin from the traumatic effects of mastication. Lack of attention to these areas caused increased vascular permeability due to mechanical (or chemical) stimulation and increased gingival fluid flow which preceded periodontal breakdown (Mann, 1963).

Recreation of the original tooth anatomy had previously included facial and lingual "deflective" bulges. Their purpose, as stated by Wheeler (1961), was to protect soft tissues by the deflective bulge resisting food contact with the marginal tissue during mastication.

This theory had not been formally challenged until the classic work of Yuodelis et al. (1973). These authors combined observations with current literature and concluded plaque retention was the primary cause of both caries and periodontal disease. The retention of plaque 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 cervical region; the flatter the contour, the less plaque retained; 2) if root portions of teeth must be covered by complete artifical crowns subsequent to periodontal therapy, the final restorations should not follow the original anatomic crown and should recreate the original contours of root position (fluting)." All attention was directed at minimizing the areas for plaque accumulation.

Experimental data utilized for Yuodelis's research was taken from investigations of Perel (1971) on young dogs. In Perel's studies, the deflective contours were removed and after nine weeks of study, no significant clinical change was noted. Overcontouring was also examined by overbuilding the buccal and lingual surfaces with a filled acrylic resin. These restorations were placed 0.5 mm. above the gingiva and at no time was the soft tissue disturbed. After only two weeks, redness of the marginal gingiva was seen; and after four weeks, occlusal tissue proliferation was measured at 0.5 to 1 mm. Bleeding was easily elicited upon probing.

Perel found increased leucocytes (of all types) after two weeks. Blood vessel engorgement was detectable at the marginal tips. Increased crevicular fluid volume was recorded. Examination at four weeks showed microscopic destruction of the epithelial parakeratotic layer near the sulcular entrance. Perel concluded "overcontouring was extended as an awning over the marginal gingiva resulting in: 1) accumulation of debris, 2) prevention of gingival margin massage during mastication, and 3) prevention of access with the tongue for bolus movement."

Similar opinions were expressed by Morris (1962) when he reappraised earlier views on buccal and lingual contours. 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." With the resultant tissue profiles, increased crevicular fluid volume had been established (Mann, 1963).

The importance of natural tooth contour without deflective bulges was reaffirmed by Burch (1971). He advocated such surfaces increased the maintainability of oral hygiene procedures, and "lended to an individual and collective self protection of the masticatory apparatus."

It had been established overcontoured restorations had been associated with the incipience of inflammatory reactions. Similarly, the subgingival margins of restorations had been observed to elicit marginal tissue inflammation (Jameson, 1976). The morphologic changes of capillaries in human gingiva adjacent to teeth restored with complete crowns was studied by Maruyana et al. (1976). This study utilized a capillary microscope and concluded inflammation was present in clinically normal tissues surrounding complete crown restorations. Histologic evidence primarily included dilitation and complex capillary loop formation.

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

Additional histologic evaluation of gingival margins was researched by Waerhaug (1960). In this classic article, subgingival margins were shown to contribute to the etiology of periodontitis. The inflammation found in the marginal area was concluded to be due to the retention of plaque microorganisms in the area of the gingival margin of restorations. Gingival inflammation was hypothesized to be caused by plaque, and not necessarily the nature of the restorative material nor its surface roughness. This was an important conclusion of the study.

Confirmation of Waerhaug's hypothesis was undertaken by Trevedi and Talim (1973). These authors studied the effects of rough surfaces on marginal gingiva histologically. Of the sample used, no clinical change was evident. However, histologically, two-thirds of those subjects displayed inflammation. The extent or degree of rough surface did not cause any greater degree of inflammation than the smoother surface with the same amount of plaque accumulation.

Löe (1962) disagreed in part with the previous studies. He suggested the "direct irritational effect of the material per se" could be a primary factor in marginal tissue breakdown. Nonetheless, he concluded as did Waerhaug, dental restorations extending subgingivally caused inflammation due to bacterial retention.

It was established that gingival margins caused inflammation, Marcum (1967) examined the sulcus to determine the effect of various levels of marginal finish lines. Three different finish lines were evaluated histologically in dogs: above the tissue, even with the sulcus crest, and subgingival. 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 responses.

These conclusions were substantiated by the work of Newcomb (1974) who examined 59 patients with anterior crowns. He utilized the Gingival Index (Löe and Silness, (1963) and the Plaque index as well as the extension of the margin into the sulcus. Examination of margin placement necessitated measurement of sulcus or pocket depth. The results showed all restored teeth had a higher Gingival Index score than non-crowned contralateral controls. As the pocket depth and margin depth increased, the degree of inflammation proportionately increased. 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 margin was placed at the crest of the gingiva.

Larato (1975) continued study on subgingival margins and sulcular degeneration due to restorative efforts. He investigated pocket depth adjacent to teeth with subgingival crowns on one hundred eleven male patients. This study revealed the average pocket depth surrounding teeth with subgingival crowns was

3.4 mm. Average pocket depth adjacent to nonrestored teeth was 2.7 mm. Fifty-four nonrestored teeth had at least one pocket depth greater than 3 mm., while 84 crowned teeth had at least one or more measurements greater than 3 mm. A positive relationship was not determined between toothbrushing frequency and the pocket depth adjacent to teeth restored with subgingival crowns.

The recurring theme throughout this research review had been the presence of inflammation which was indigenous to artifical crowns, especially those placed subgingival. Ramfjord et al. (1974) seems to have confirmed this belief by reviewing longitudinal research performed at the University of Oslo. This work evaluated the gingival status of 334 abutment teeth in 110 patients for three years. The results showed gingivitis to be more severe, with deeper pockets and more loss of attachment around teeth with crowns which had 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.

The sulcular area had been shown to be very labile and artificial crowns with subgingival margins irritated the crevicular epithelium and caused inflammation (Maruyama, 1976). The presence of plaque also caused inflammation which was compounded by improper contour (bulges) of crown restorations (Yuodelis, 1973). Together these factors affected the marginal permeability (by inflammation) and resulted in increased crevicular fluid flow (Egelberg, 1966). Jameson (1976) measured 57 teeth with artifical crowns and compared them to the unrestored teeth on the opposite side of the arch. His results confirmed the fact a certain degree of inflammation is indigenous to subgingivally placed margins on artificial crowns.

CHAPTER III

MATERIALS AND METHODS

This clinical study involved a total of thirteen (13) patients from Loyola University School of Dentistry and from private practice. There were seven (7) females and six (6) males between the ages of 20 and 60, with the mean age of 36.6. Each patient was required to sign a witnessed consent form which described the intended procedures.

Thirty (30) teeth from the thirteen (13) patients were selected for evaluation. Each patient qualified for the study by needing a posterior fixed bridge which involved at least one pontic and two abutment teeth unilaterally. These teeth had no previous restorations involving the sulcular area. Each patient selected possessed unrestored teeth on the opposite side of the dental arch. The unrestored side was used as a control. Photographic records were made for each patient to provide records prior to the restoration of one side of the arch. It also helped to provide clinical records for the gingival tissue index evaluation.

All prostheses, except one, were prepared by the investigator; and all patients received porcelain fused to gold with porcelain occlusal surfaces. Recognized licensed dental laboratories were employed to complete the prostheses.

The pericoronal tissue of these thirty (30) prepared teeth and thirty (30) control teeth were evaluated in three distinct manners:

1) The depth of the gingival sulcus was measured and recorded at five (5) time intervals.
2) The marginal tissue was assessed in terms of a gingival index after Löe and Silness (1967).
3) The crevicular fluid was collected in two (2) areas of each

abutment tooth with the Harco Gingival Crevicular Fluid Meter at five (5) time intervals.

Measurements of the Gingival Index refers to the clincial appearance of the marginal gingiva. A Löe and Silness (1967) criterion consists of:

Score	Clinical Findings
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 areas selected for measurement were the mesiobuccal and distobuccal line angles because of easy access.

The periotron (figure 1) was chosen to moderate gingival fluid flow, since most research (Suppipart, 1976; Golub and Kleinberg, 1976; Shern, et al., 1974; Borden, et al., 1974) indicated this instrument was more sensitive to change in fluid volume than other methods.

Periopaper (figure 2) was utilized for collecting fluid. The orifice method according to Löe and Holm-Pederson (1965) as modified by Borden, et al., (1974) was the technique employed. All measurements were made by the investigator in the following manner:

1. Region to be evaluated was isolated with cotton rolls and dried with slight pressure.

2. A sterile dry filter paper strip (Periopaper) was placed into the orifice of the gingival crevice (according to the technique of Löe and Holm-Pederson, 1965, modified by Shern, et al., 1974). The initial strip remained here

for three seconds, was removed, and was discarded.

3. The emptied fluid pool was allowed to fill for twenty-seven (27) seconds, and another sterile strip was placed in the same location. This strip was allowed to collect fluid for three seconds. The total elapsed time was thirty (30) seconds (figure 3).

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

5. The Periotron presented a digital readout, which automatically is held after 19-20 seconds. This reading was entered on the data sheet. This numerical reading may be converted to volume in microliters by dividing by 200 (e.g., a digital reading of 10 = 0.05 ul).

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

Each tooth was evaluated three (3) times with five (5) minute intervals between collections. Also, the time of day was kept consistent for each determination due to the possible interference of circadian periodicity (Bissada, et al., 1967).

The control of this data was the assessment of the teeth on the opposite side of the same arch for the same patient. These control teeth were without restorations. They were evaluated in the same manner as the prepared teeth.

Measurements of the pericoronal tissue of 60 teeth included 120 surfaces and each were assessed in three (3) trials. Five (5) determinations were collected for each patient at the following intervals: 1) before treatment, 2) at time of temporary removal for cementation of prosthesis, 3) one week after

Figure 1: The Periotron

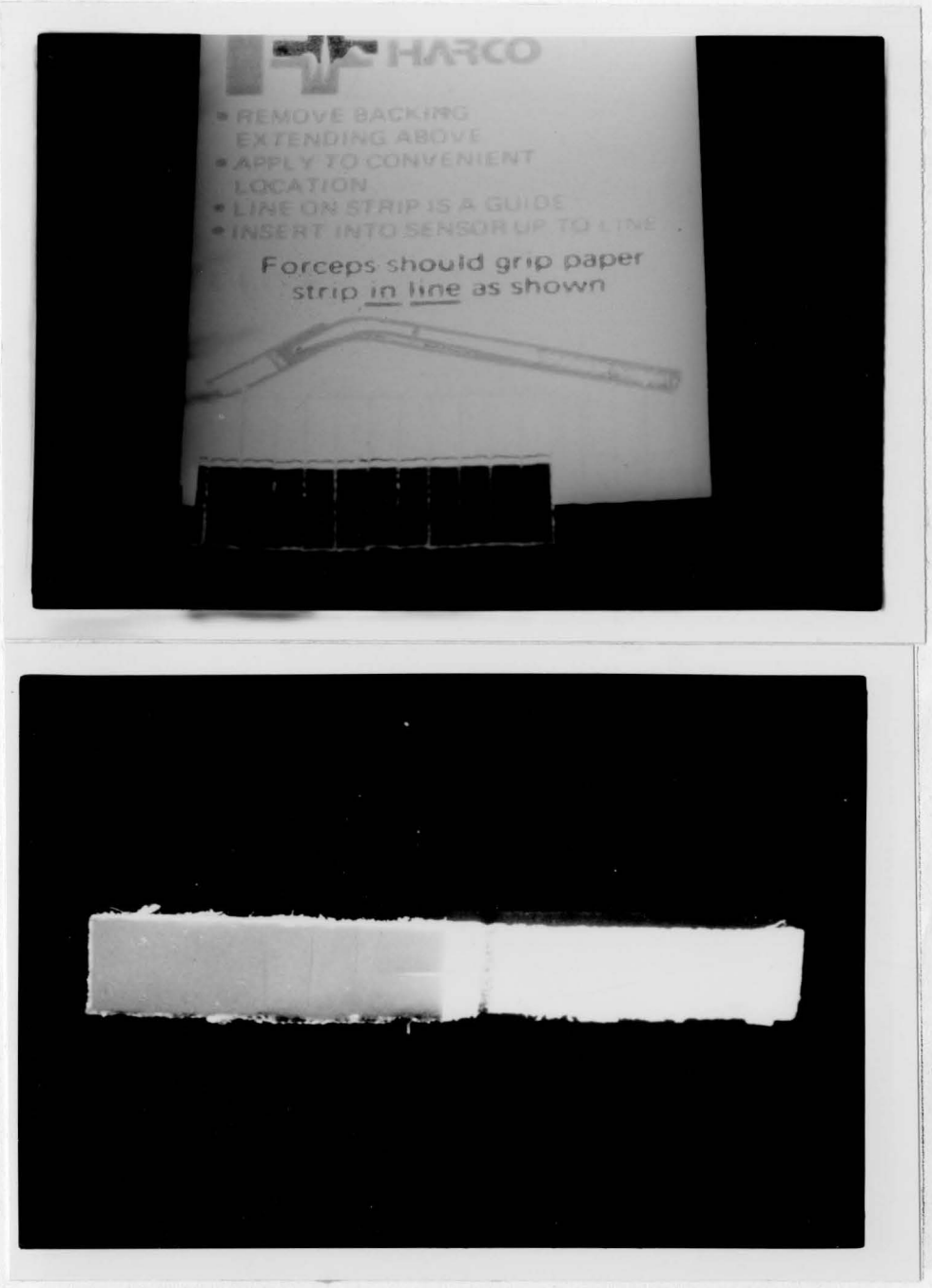

Figure 2: Periopaper, as supplied

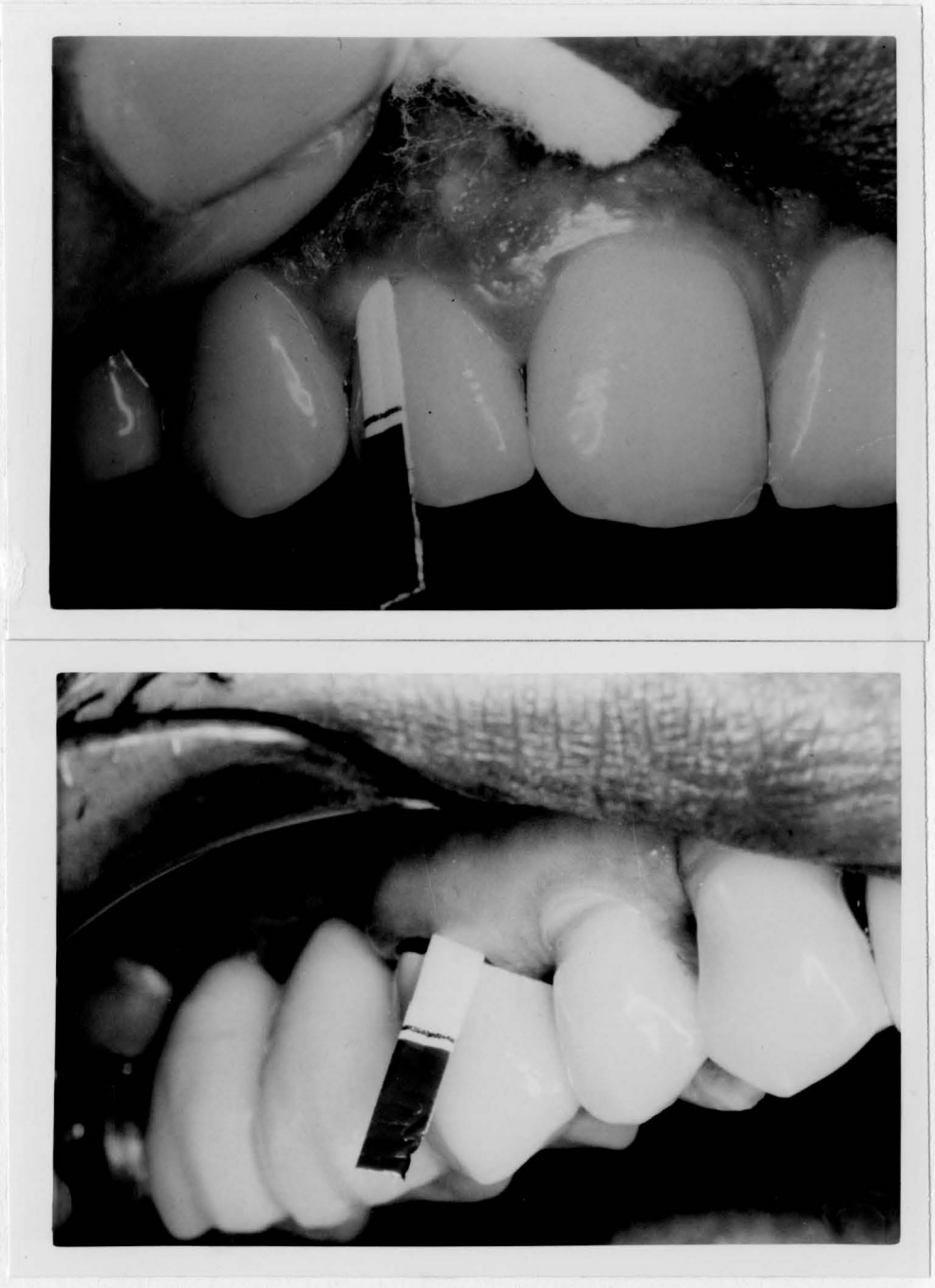

Figure 3: Periopaper in the
Gingival Sulcus

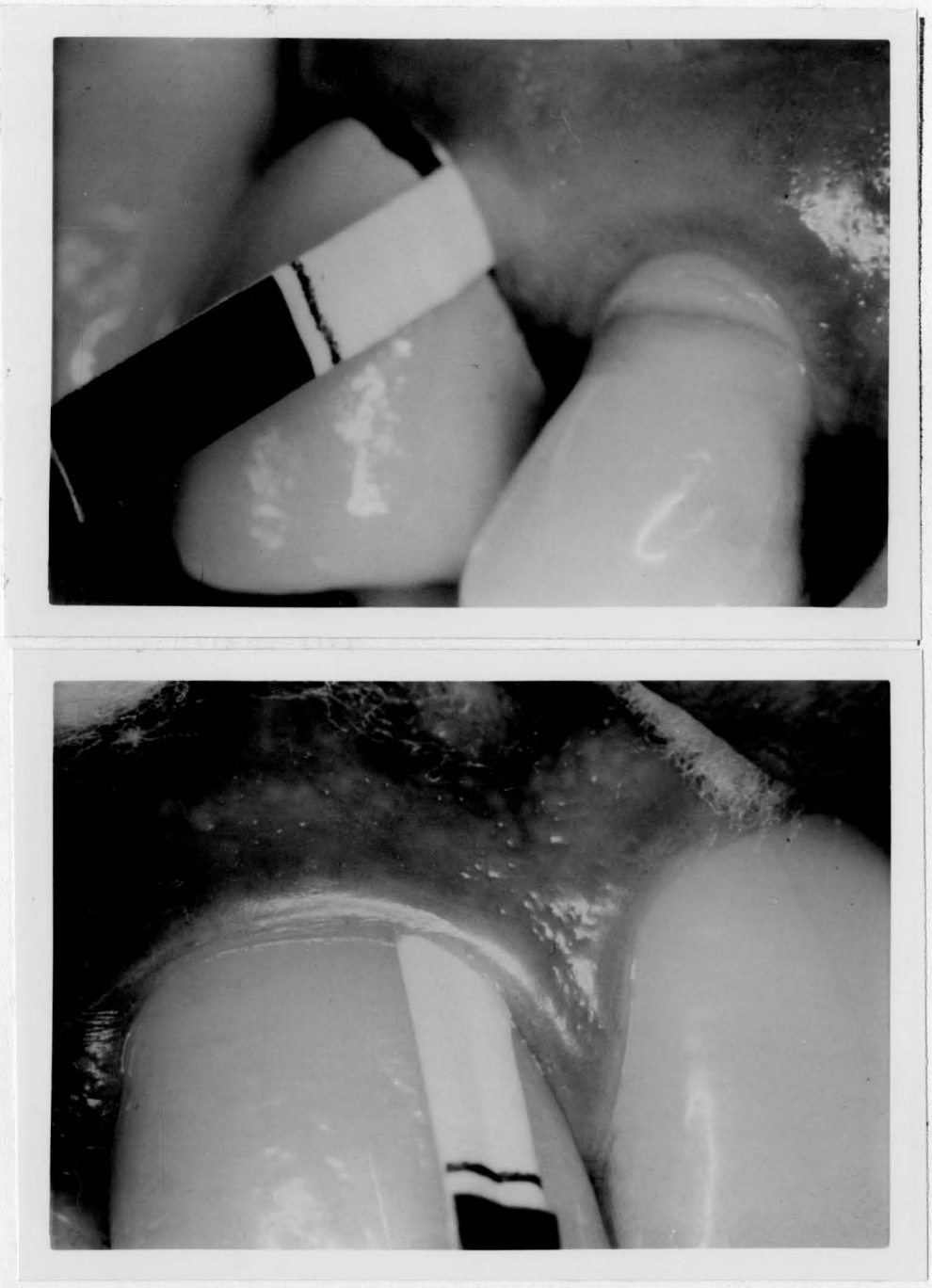

Figure 3 (continued): Periopaper in

the Gingival Sulcus

Figure 4: Periopaper in place between sensors which are separated

cementation, 4) thirty (30) days after cementation, and 5) sixty (60) days after cementation.

The data was treated statistically by paired "T" tests which permitted examination of the interaction between intervals. Significance was assigned to probability less than the 0.05 level.

CHAPTER IV

EXPERIMENTAL RESULTS

Each tooth and its contralateral control was evaluated by the Periotron in three trials at each interval. The means of the raw data are compiled and listed in Table IX for the prepared teeth, and Table X for the control teeth. There is also a record of each patient, their age, and the variation of pocket depth and Gingival Index Score (after Löe and Silness, 1967). This is contained in Table XI. No significant difference was found in pocket depth variation nor Gingival Index Score.

Crevicular Fluid Variation was examined by means of multiple paired "T" tests and thereby the interaction could be readily determined between the intervals of measurement. Analysis of the data begins with Table I and Table II which summarize the means and standard deviation of each interval. The deviation tends to decrease at the later stages of fluid recording.

The intervals first compared are those between prepared and control teeth (Tables III and IV). The only variation, besides which could occur by chance, is the tissue manipulation of tooth preparation. Such was shown to be significant.

Prepared teeth were examined at different time intervals (Table V and VI). A statistically significant change of fluid volume was noted before treatment and sixty days after treatment on the mesial aspect of the restored teeth. An inconsistent value was recorded at these intervals on the distal aspect of the abutment teeth.

Change was shown to be significantly present on mesial and distal aspects when the temporary was removed and the final prosthesis was seated.

To be certain that this variation was initiated during the course of treatment, identical intervals were tested between the control teeth. Some

significance was detected for the mesial aspect of the control teeth.

TABLE I

SUMMARY OF COMPILED DATA: PREPARED TEETH

EXPRESSION OF VARIATION IN PERIOTRON UNITS

TIME OF FLUID MEASUREMENT	MEAN	STANDARD DEVIATION
Mesial before treatment	12.79	13.33
Distal before treatment	13.05	16.58
Mesial temporary removal	21.30	13.67
Distal temporary removal	22.45	15.25
Mesial at one week	11.11	4.91
Distal at one week	14.11	12.41
Mesial at thirty days	8.71	4.53
Distal at thirty days	9.87	6.12
Mesial at sixty days	7.93	4.92
Distal at sixty days	8.15	4.96

TABLE II

SUMMARY OF COMPILED DATA: CONTROL TEETH

EXPRESSION OF VARIATION IN PERIOTRON UNITS

TIME OF FLUID MEASUREMENT	MEAN	STANDARD DEVIATION
Mesial before treatment	10.51	10.20
Distal before treatment	12.99	14.85
Mesial temporary removal	14.23	10.15
Distal temporary removal	11.85	8.07
Mesial at one week	10.10	6.25
Distal at one week	12.05	10.84
Mesial at thirty days	8.57	6.72
Distal at thirty days	9.81	6.09
Mesial at sixty days	8.83	7.34
Distal at sixty days	9.94	9.07

TABLE III

PAIRED "T" TESTS

BETWEEN CONTROLS AND PREPARED TEETH: MESIAL

INTERVAL	"T" VALUE	PROBABILITY
Before treatment and		
Control before treatment	0.66	0.513
Temporary removal and		
Control temporary removal	2.20	0.036*
One week and		
Control one week	0.72	0.477
Thirty days and		
Control thirty days	0.10	0.921
Sixty days and		
Control sixty days	-0.64	0.528

*indicates significant value at $P < .05$

TABLE IV

PAIRED "T" TESTS

BETWEEN CONTROLS AND PREPARED TEETH: DISTAL

INTERVAL	"T" VALUE	PROBABILITY
Before treatment and Control before treatment	0.01	0.989
Temporary removal and Control temporary removal	3.71	0.001*
One Week and Control one week	0.62	0.539
Thirty days and Control thirty days	0.04	0.970
Sixty days and Control sixty days	-1.14	0.265

*indicates significant value at $P < .05$

TABLE V

PAIRED "T" TESTS BETWEEN INTERVALS

FOR PREPARED TEETH: MESIAL VALUES

EXPRESSION OF VARIATION AT DIFFERENT TIMES OF MEASUREMENT

INTERVAL	"T" VALUE	PROBABILITY
Before treatment and After temporary removal	-2.43	0.021*
Before treatment and After one week	0.70	0.491
Before treatment and After thirty days	1.64	0.112
Before treatment and After sixty days	2.04	0.050*
Temporary removal and After one week	4.42	< 0.001*
Temporary removal and After thirty days	4.89	< 0.001*
Temporary removal and After sixty days	4.76	< 0.001*
One week and After thirty days	2.15	0.040*
One week and After sixty days	2.74	0.010*
Thirty days and After sixty days	0.97	0.340

*indicates significant value at $P < .05$

TABLE VI

PAIRED "T" TESTS BETWEEN INTERVALS

FOR PREPARED TEETH: DISTAL VALUES

EXPRESSION OF VARIATION AT DIFFERENT TIMES OF MEASUREMENT

INTERVAL	"T" VALUE	PROBABILITY
Before treatment and After temporary removal	-2.47	0.020*
Before treatment and After one week	-0.40	0.691
Before treatment and After thirty days	1.25	0.222
Before treatment and After sixty days	1.53	0.137
Temporary removal and After one week	3.30	0.003*
Temporary removal and After thirty days	4.53	0.000*
Temporary removal and After sixty days	5.21	0.000*
One week and After thirty days	2.56	0.016*
One week and After sixty days	2.69	0.012*
Thirty days and After sixty days	1.68	0.103

*indicates significant value at $P < .05$

TABLE VII

PAIRED "T" TESTS BETWEEN INTERVALS

FOR CONTROL TEETH: MESIAL VALUES

EXPRESSION OF VARIATION AT DIFFERENT TIMES OF MEASUREMENT

INTERVAL	"T" VALUE	PROBABILITY
Before treatment and After temporary removal	-2.41	0.023*
Before treatment and After one week	0.28	0.784
Before treatment and After thirty days	1.26	0.218
Before treatment and After sixty days	0.85	0.404
Temporary removal and After one week	2.94	0.006*
Temporary removal and After thirty days	3.27	0.003*
Temporary removal and After sixty days	2.66	0.013*
One week and After thirty days	1.75	0.091
One week and After sixty days	1.17	0.252
Thirty days and After sixty days	-0.29	0.774

* indicates significant value at ($P < .05$)

TABLE VIII

PAIRED "T" TESTS BETWEEN INTERVALS

FOR CONTROL TEETH: DISTAL VALUES

EXPRESSION OF VARIATION AT DIFFERENT TIMES OF MEASUREMENT

INTERVAL	"T" VALUE	PROBABILITY
Before treatment and After temporary removal	0.48	0.637
Before treatment and After one week	0.30	0.769
Before treatment and After thirty days	1.19	0.245
Before treatment and After sixty days	0.98	0.334
Temporary removal and After one week	-0.10	0.920
Temporary removal and After thirty days	1.76	0.089
Temporary removal and After sixty days	0.96	0.346
One week and After thirty days	1.34	0.189
One week and After sixty days	0.98	0.338
Thirty days and After sixty days	-0.10	0.920

*indicates significant value at $P < .05$

CHAPTER V

DISCUSSION

In this investigation, thirty teeth of thirteen patients were restored. Each patient demonstrated prior tooth loss on their dental history. Fixed partial dentures were to replace the missing teeth. The contralateral side of the restored arch contained the same teeth without restorations.

This provided an experimental control. The preparation, impression with cord retraction and temporization were the only dental procedures performed on the subjects during the period of crevicular fluid measurement. On five occasions, each tooth was evaluated from before treatment to sixty days after the prosthesis was inserted. The subjects in the sample were selected to provide a control using the contralateral teeth. They were also chosen because fixed partial dentures permitted the recording of multiple abutments with one impression and temporization procedure.

Results shown by Jameson (1976) noted that inflammation was indigenous to artificial crown restorations. Of the teeth examined in his study, all the restorations had been present for at least six months. The data was well controlled and a conclusive comment on inflammation was reflected with an increase of crevicular fluid volume.

Some question existed as to whether this inflammation was a result of incomplete tissue healing after the insult of tooth preparation. Does the gingival tissue return to its previous state after such restorative procedures? Data compiled by this investigation indicated the sulcular epithelium can return to its state prior to restorative procedures. Inflammation recorded by increases in gingival fluid was likely due to the presence of the final restoration, not solely by the restorative procedure.

The collection of this data reflected of variability commonly seen in clinical research. This is evidenced by the range of standard deviations found in Tables I and II. An increase of sample size would alleviate this problem. The measurements of crevicular fluid volume, exhibited greater accuracy as experience in recording crevicular fluid increased. This can be noted by the gradual decrease in standard deviations from the one week records to sixty day records in Tables I and II.

In regards to instrumentation variation, one cannot ignore the degrees of irritation due to drying of the gingiva, and the insertion of filter paper strips (Egelberg, 1966). Leirskar (1971) mentions also the variation in the viscosity of gingival fluid that influences the absorability of each filter paper strip.

Consideration must be given to the instrument as well as the instrumentation. Suppipat's (1976) studies on the Periotron illustrated a standard deviation of $\pm$ 15% in day to day readings of a known volume of serum. Similarly, the Bissada et al. (1967) investigation in circadian periodicity and individual variability, displayed no consistency. However, other investigators have data within much closer limits than this study.

Despite variability, the data reflected a clinical evaluation more critical than that of the Löe and Silness Gingival Index. Throughout the restorative procedures, a Löe and Silness score of 2 was uncommon. This indicated the presence of only mild inflammation even during the temporization procedures, which displayed levels of moderate to severe inflammation when measured by the Periotron (Table IX).

The Periotron is capable of monitoring levels of inflammation that may not yet be clinically apparent. Its range of readings are the following: 0-10, healthy; 11-20, mild inflammation; 21-40, moderate inflammation; and greater than 40 units indicates severe inflammation.

Patient variability was also a factor in these results. All the subjects responded differently to the temporization procedures. This varied proportionately with that area of gingiva impinged upon by the temporary, and the individual patient's capacity for tissue repair. Some restorations demanded subgingival margin placement, and more inflammation was noted. Areas kept supragingival responded with the least inflammation (Yuodelis, 1973). One subject (teeth #9, 10) lost the temporary restoration by breakage and, surprisingly, the tissue fluid values were the lowest this patient experienced throughout the study.

Despite the variability, some trends were established. Tissue response to the insult of tooth preparation was readily apparent. After only one week, the prepared teeth had crevicular fluid volumes that were indistinct from their controls (Tables III and IV). Variation at this time could have been due to chance alone and not to any treatment rendered. In examining the time intervals, a significant difference existed in comparison between the before treatment level of gingival fluid and that after sixty days on the mesial side. Such was not the case on the distal side, yet the data reflected a tendency in that direction (Tables V and VI). The sulcular epithelium, it seems, if judged to be at only a slight level of inflammation, is damaged by preparation (Tables III, IV, V, VI). One week after cementation, this epithelium has repaired sufficiently to record the volume of crevicular fluid similar to the volume before preparation. From this level, there appeared to be an increase due to the tissue response to the foreign restoration. If tested at the six month interval, one would expect confirmation of the inflammatory trend found here, and the results noted by Jameson (1976). Inflammation is indigenous to artificial crowns.

Future research will be necessary to confirm these results. A larger sample size with an even more accurate method of collection would make the records more accurate by reducing the variability.

CHAPTER VI

SUMMARY AND CONCLUSIONS

The extent of crevicular fluid volume has been found to be a reliable indicator of the severity of inflammation of the marginal gingival tissue. Ideally, the sulcular epithelium should provide a barrier to fluid flow in either direction. The absence of fluid indicates the absence of inflammation. Yet, it is a clinical reality that crevicular fluid is commonly present. Each tooth is apparently surrounded by some degree of inflammation.

Whether this fluid is an exudate or a transudate of serum is a subject still under investigation. At present, the majority of research dictates this fluid to be an exudate. A volume increase or decrease can be monitored to evaluate the degree of inflammation.

It was the goal of this project to record crevicular fluid alteration during the tissue manipulation of restorative techniques. The following conclusions were reached:

1) Restorative procedures caused an increase in inflammation to the marginal gingival tissue.
2) Temporization after preparation elicited the greatest degree of inflammation. Resolution occurred quickly. The shortest possible time of treatment restoration (temporization) was deemed desirable.
3) Statistical trends in this investigation indicated that the marginal tissue and sulcular epithelium are repaired after restorative procedures; and inflammation increases were due to the presence of the restoration.

APPENDIX

TABLE IX

COMPILED RAW DATA - PREPARED TEETH

AVERAGES OF THE THREE TRIALS, EXPRESSED IN PERIOTRON UNITS

	BEFORE TREAT		TEMP REMOVAL		ONE WEEK		THIRTY DAYS		SIXTY DAYS	
Subject	M	D	M	D	M	D	M	D	M	D
1	9.6	28.7	25.7	9	5.3	7	8.3	14	3.7	5
2	2.7	1.3	17.3	18.3	5.7	7.3	6.3	4.7	2	1.7
3	18.7	7.3	22	23.7	12.7	25	15	15.7	11	10.7
4	4.3	5.3	16.3	16.7	13.7	22	7.3	13	3	8.3
5	15	35.8	15.7	19	10	7.7	19	16.3	11.3	6
6	8	11.3	44.3	49.7	11.3	9.7	3.7	5.3	3.6	8.7
7	6.7	1	8	7.3	2.3	3	2	2.7	2	1.7
8	7.7	2.3	17.7	18	15.3	7.7	4.3	3.3	1	1
9	23	22.7	2.7	6.7	6	13	10.3	18	20.7	17
10	1.3	10.7	1.3	2	7.3	15.3	11	14	10.7	16
11	51.3	14.7	5	6	17.7	7.3	9	7.3	12	4.7
12	9.7	11.7	11.7	7.3	4	12	9	6.7	3.3	3
13	2.3	1.7	29	43.3	13.3	21.3	10	12.7	15.7	15.7
14	6.7	4.3	20	16.7	16	10.6	10.3	11	11	12.7
15	49.3	6.7	12.7	60	12	11	7	3	10	6
16	18	4.3	53.7	23.7	12.3	20.7	5.7	14	6.7	8.7
17	38.7	35	41.7	23.7	15.7	6.3	13.7	9	9.3	14
18	5.5	1.2	23	23	6.3	9.3	3	6	3	6.6
19	15.3	79.3	26	55.3	17.3	69	5.7	27.3	2.7	5.3
20	6	11.3	10.7	20.3	4	5	4.3	6	2.7	4.7
21	2.3	2.7	14.3	18.7	15.7	6	6	2	6.7	2
22	11.3	2	48	46	17.7	21	13.3	19.3	4	15.7
23	3.3	1.6	17.5	39.3	11.3	28	21.3	8.3	13.7	17.3
24	7.7	29	30.3	11	17.7	18	7.7	13.7	7.7	11
25	7	1.7	4.7	8	6	9	7	1.3	8	2.3
26	2	19.7	12	15.3	17.7	6.5	9.3	9	13.3	5.7
27	2.7	2.3	15	11.3	9	3.3	4	4	9.7	7.3
28	2	1	42.7	24	13.3	6.3	11	4	8.3	6.7
29	2.7	2	22	26	11	19.7	5	9.7	6	7.7
30	18.7	9.7	28	23.3	14	15.3	11.7	14.7	15	11.3

TABLE X

COMPILED RAW DATA - CONTROL TEETH

AVERAGES OF THE THREE TRIALS, EXPRESSED IN PERIOTRON UNITS

	BEFORE TREAT		TEMP REMOVAL		ONE WEEK		THIRTY DAYS		SIXTY DAYS	
Subject	M	D	M	D	M	D	M	D	M	D
1	12	6.3	27	8.3	8.7	2.7	8.3	6	2	1.7
2	35	34.3	28.7	25	14.7	14.7	20.7	18.7	7.4	19.3
3	8.7	5	9.7	7.7	12	6.3	8.3	12.3	9	12.3
4	12.7	24.7	9	11.7	9	9.7	7.3	7.7	10.7	7.7
5	1.7	1.7	4	4.3	4.7	6	12.7	3.3	8.3	9
6	7.7	15.7	11	21.7	9.3	15.7	11.7	20.7	22	18.7
7	11.3	35	6.7	2.3	9.3	4.3	4.7	3	1.7	1.3
8	27.7	17.7	16.7	13.7	7	8	3	3	1.3	2
9	3.7	5.3	2.7	4.7	5	6	8	10.7	6.7	10
10	19	20.3	19.7	15.7	18.3	17.7	14	21.3	15	24.7
11	8.3	5	11.7	5.3	11.3	8.7	6.3	3.3	8.7	5.3
12	31.7	19	20.3	20.7	19.7	16.7	13	12.3	12	18.3
13	34.7	12.7	37.3	25.3	31	24.3	33.7	22	29	21.7
14	9	14.7	10	15.3	10.3	13	14.7	15	15.3	16.7
15	6.3	5	11.7	9.3	6.3	3.3	3.3	5.3	3.3	5.3
16	2.3	1.7	12.3	8	4.3	18	5.7	13	5.3	6.3
17	8.7	8.7	3	18	5.7	22.3	3.7	15.7	4.7	7.7
18	21	26.3	30.3	29.3	2.3	24.3	1	17	1	1.3
19	2.6	1	16.3	8	8	2.7	1.6	1.3	1	2
20	2	4	18.7	9.7	9	9.3	5	7.6	6.7	2.7
21	17.3	15.3	33	51	18.3	56.3	13	12.7	18.7	15.3
22	1.6	7.3	6.3	23.3	6	8.3	3.7	8.7	4	6.7
23	2.7	14.2	2.7	4	1.3	2.3	9.3	6.3	7	8.3
24	3.7	4.3	4.7	4.3	11.7	9.3	12.7	10	27.4	41.7
25	1.3	2	1	1.3	2.3	1.3	1	1	5	1
26	5	2.3	29.7	24	18	22.3	3	4.7	7.3	6.3
27	8.7	3	15.3	10.3	9.7	4.7	8	7.7	10.3	4
28	1	1.3	14.7	5	9.7	6.7	9.3	8.3	7	2.7
29	2.7	3	10.7	3.7	13	5.7	7.7	4.3	3.3	4.3
30	5.2	7.7	2	10.7	7	10	2.7	11.3	4	14

TABLE XI

SUMMARY OF PATIENTS, POCKET DEPTHS, AND GINGIVAL INDEX SCORES

PATIENT	AGE	SEX	TOOTH #	BEFORE TREATMENT M Pocket	BEFORE TREATMENT D D	AFTER TREATMENT M Pocket	AFTER TREATMENT D D	GI_1	GI_2	GI_3	GI_4	GI_5
			18	1	1	2	2					
1	35	M	20	1	1	1	1	0	0	0	0	0
			18	3	2	3	3					
2	25	F	20	2	2	$2\frac{1}{2}$	2	0	1	0	0	0
			2	1	2	$1\frac{1}{2}$	2					
3	43	M	4	1	1	1	1	0	0	0	0	0
			19	2	1	$1\frac{1}{2}$	$1\frac{1}{2}$					
4	20	F	21	$2\frac{1}{2}$	1	$1\frac{1}{2}$	$1\frac{1}{2}$	0	0	0	0	0
			2	$2\frac{1}{2}$	$2\frac{1}{2}$	2	3					
5	36	M	5	2	2	2	2	0	0	0	0	0

TABLE XI - continued

SUMMARY OF PATIENTS, POCKET DEPTHS, AND GINGIVAL INDEX SCORES

PATIENT	AGE	SEX	TOOTH #	BEFORE TREATMENT M Pocket	BEFORE TREATMENT D D	AFTER TREATMENT M Pocket	AFTER TREATMENT D D	GI_1	GI_2	GI_3	GI_4	GI_5
6	31	M	17	3	2	3	2	0	1	0	1	0
			20	2	2	2	1					
			13	3	3	2	$2\frac{1}{2}$	0	0	0	0	0
			15	2	2	2	2					
7	60	M	28	2	3	2	1	0	0	0	0	0
			29	3	$2\frac{1}{2}$	2	3					
			32	3	3	2	3					
8	28	F	28	1	1	1	2	0	1	0	0	0
			30	3	$3\frac{1}{2}$	$1\frac{1}{2}$	1					
9	37	F	11	$2\frac{1}{2}$	$2\frac{1}{2}$	$1\frac{1}{2}$	2	0	1	0	0	0
			13	3	2	2	2					
			15	3	$2\frac{1}{2}$	3	2					
10	26	F	11	2	2	$2\frac{1}{2}$	$2\frac{1}{2}$	0	1	0	1	0
			14	3	3	2	2					

TABLE XI - continued

SUMMARY OF PATIENTS, POCKET DEPTHS, AND GINGIVAL INDEX SCORES

PATIENT	AGE	SEX	TOOTH #	BEFORE TREATMENT M Pocket	BEFORE TREATMENT D D	AFTER TREATMENT M Pocket	AFTER TREATMENT D D	GI_1	GI_2	GI_3	GI_4	GI_5
			4	1	2	1	$1\frac{1}{2}$					
11	48	M	1	3	2	2	2	0	0	0	0	0
			18	4	2	3	3					
12	43	F	21	2	2	2	2	0	1	0	0	0
			12	2	2	2	2					
13	45	F	15	2	1	2	$2\frac{1}{2}$	0	0	0	0	0

LOYOLA DENTAL SCHOOL

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 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 anytime 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:

_____ _____

CLINICAL DATA EXAMINATION FORM

Date: _____

1. Patient's Name: _____
2. Age: _____ Sex: _____
3. Occlusal Relationship: Canine _____ Molar _____
4. Body Type: Endo Meso Ecto
5. Identification of Abutments (#'s)
6. Number of Pontics _____

Abutments		M D	M D	M D	M D
Trial	1				
	2				
	3				
Gingival Index					
Pocket Depth					

Before Treatment	Temporary Removal	One Week Post-Op	Thirty Days Post-Op	Sixty Days Post-Op

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The thesis is, therefore, accepted in partial fulfillment of the requirements for the degree of Master of Science in Oral Biology.

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