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Determination of the Gingival Crevicular Fluid Volumes Associated with Restored (Fixed and Removable Prosthodontics) and Non-Restored Teeth

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DETERMINATION OF THE GINGIVAL CREVICULAR FLUID VOLUMES ASSOCIATED WITH RESTORED (FIXED AND REMOVABLE PROSTHODONTICS) AND NON-RESTORED TEETH

By

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DEDICATION

To Virginia, my Mother, to thank her for the years of unselfish love, patient guidance and gentle encouragement, I dedicate this thesis.

In loving memory of Victoria Bugaj, my Grandmother, for sharing her wisdom and love; and to thank her for the lilacs and other miracles of Nature.

To my dear sister, Roxane, not only for her love and understanding, but most importantly, for her friendship.

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VITA

The author, Pamela Denise Golasz, is the daughter of Daniel and Virginia (Bugaj) Golasz. She was born June 12, 1953 in Chicago, Illinois.

Her elementary education was obtained from the Franz Peter Schubert School in Chicago, Illinois; and her secondary education from the Maine Township High School South of Park Ridge, Illinois where she graduated in 1971.

In May of 1975, she graduated from DePaul University, Chicago, Illinois where she received the degree of Bachelor of Science, majoring in Biology.

In September of 1975, she entered Loyola University School of Dentistry, Maywood, Illinois and in May of 1979, received the degree of Doctor of Dental Surgery.

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CHAPTER I

INTRODUCTION AND STATEMENT OF PURPOSE

The primary objective of restorative dentistry is the replacement of lost teeth or portions of carious and/or fractured teeth. Tissue trauma is an inherent problem (Jameson, 1979) even for the most accomplished and exacting operator. Utilization of the most delicate and innocuous manipulation of the supporting structures can still result in an inflammatory response of soft tissues. One controversial aspect within restorative dentistry is the deleterious effect of applied stress on abutment teeth for both fixed and removable prosthesis. The length of the span of the prosthesis has never been objectively assessed.

Studies (Waerhaug, 1960) have indicated subgingival margin termination and subsequent plaque accumulation were critical considerations in tissue response. The actual contour of the crown proved to be a major determinant in the type of tissue response noted. Youdelis (1973) stated "overcontouring encourages the accumulation of particulate and microbial matter in an area inaccessible for cleaning by the patient". Caruso (1979) determined the morphologic contour of an artificial tooth and/or of a fixed prosthesis should be undercontoured rather than overcontoured. The type of dental materials employed and their relative degree of surface roughness (Volchansky, 1974) were critical to inflammatory response observed in the soft tissue. Jameson (1979) stated margin termination, independent of the dental material employed to construct the crown was

crucial, and felt chronic inflammation was indigenous to subgingival placement of full coverage margins.

Investigators (Tylman, 1978) have eluded to the fact that stresses promoted by "free ended" removable partial dentures are ultimately the cause of loss of abutment. Schuyler, 1941, stated "the semi edentulous mouth presents to the dentist one of his most complex problems. Fixed restorations supplying one, two or a greater number of teeth have served more adequately in maintaining oral function and health than the removable partial appliance as commonly planned and constructed." Cohn (1956) sited "a common existing cause of periodontal disease is the faulty removable partial denture, or fixed partial denture, in which anchorages are improperly selected or incorrectly fabricated. A physiologic result can be obtained from a dental prosthesis only when the strain exerted on abutment teeth is considered in relation to the biologic union of the tooth to the supporting structures, and the mechanical principles involved."

These implications as to the detrimental nature of partial denture prosthesis command additional credence when partial dentures are utilized in patients whose dentitions are handicapped by periodontal disease. Comparisons by restorative dentists have been difficult because removable partial dentures are usually placed in mouths in which restoration via fixed prosthodontics is contraindicated. Despite this fact, some evidence should be demonstrated to validate the concept that removable partial denture abutments respond poorly to the increased occlusal load and torque.

Until recently, no quantitative measurement could be obtained to

assess the degree of response (inflammation) of the soft tissue adjacent to the abutments. Traditionally, the measurement of gingival response to restorative procedures has been evaluated by subjective clinical indices. Loe and Silness (1967) correlated various degrees of observable inflammation by numerical assessment of gingival inflammation on a scale ranging from zero (0-- no observable inflammation) to three (3-- evidence of severe gingival inflammation). Consistency from one researchers observations and recordings to anothers has proven to be a deficient aspect of this clinical procedure. Recently, the introduction of an instrument* that utilizes volumetric quantitation of crevicular fluid as a diagnostic measuring tool of gingival inflammation has lead to a more objective and consistent assessment of soft tissue response to chronic irritation. Crevicular fluid flow increases with an increase in inflammation, and increased crevicular fluid flow occurs prior to clinical manifestations.

Brill (1959) first suggested using crevicular fluid as a measure of gingival inflammation to provide a quantitative means to monitor the physiology of the sulcular region. He noted a greater fluid flow from patients with extensive restorative work compared to clinically healthy, non-restored marginal gingiva. Egelberg (1964) found a correlation between the amount of gingival exudate collected on filter paper strips and the area of inflammatory cell infiltration noted in histological examination. There remains some dispute as to the nature of crevicular fluid, be it exudative or transudative. The conclusion of Bang and Cimasoni (1971) stated "because the gingival fluids have a capacity to

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* Periotron, Harco Electronics Limited, Winnipeg, Canada.

carry high molecular weight compounds such as proteins, this is confirmation that gingival fluid is an inflammatory exudate that passes the crevicular barrier as a result of increased capillary permeability." Cimasoni (1974) qualitatively determined the following components were present in crevicular fluid: Immunoglobulins G, A, and M, Globulins alpha 1 and 2 and beta 1, albumin, fibrinolytic factors, lactic acid, urea, hydroxyproline, endotoxins, bradykinins, and lysosomal enzymes.

The purpose of this investigation is to determine and compare the inflammatory changes in the marginal gingiva of abutment teeth for fixed and removable prostheses.

CHAPTER II

REVIEW OF THE LITERATURE

A. Historical Perspective

In G.V. Black's (1887) histological assessment of the periodontal membrane, he noted the existence of a gingival fluid. "Close clinical examination makes it apparent that there is a slight secretion at this point that is not quite satisfactorily explained even yet by microscopic study of the part." Black noted the existence of a system of cells located about the principle fibers of the periodontal membrane. Referring to these cells as salivary corpuscles, he noted that this tissue was affected first in salivation with certain drugs such as mercury and iodine. Black (1899) described a plexus of loops of glandular tissue associated in a parallel fashion to the root and anastomosing freely. Black (1922) postulated that subgingival calculus formation was augumented by the constant fluid present in the subgingival space.

Stillman and McCall (1927) sited "the cleansing of the enamel surface over which it lies by the serous secretion from its crevicular surface" as a function of the marginal gingiva.

McCall (1924) proposed that teeth in traumatic occlusion resulted in an acidic exudate of the gingival crevice by alteration of the periodontal tissue.

Boedecker (1931) disagreed with Black's (1899) description of a plexus of gingival glands based upon histological findings. Boedecker

did concur with McCall's theory and utilized litmus paper to examine human gingival fluid and to determine "the relation of an acid exudate of the gingival crevice to erosion."

Miller (1938) spoke of "crevicular exudate" as a "discharge from the gingival crevice". He postulated alterations in the crevicular exudate as evidence of subclinical signs of periodontal disease "since microscopic examination of the crevicular fluid reveals the presence of an unusual number of pus cells." Miller noted increased amounts of gingival fluid occurred in crevices adjacent to teeth with erosion.

B. Classical Literature

Waerhaug's (1952) investigations indicated after injection of India ink into the gingival sulcus of young dogs, an increased transudation of fluids and emigration of leukocytes through the sulcular epithlium could be observed within one hour; and the majority of the India ink particles had been removed by this fluid within two hours. Waerhaug postulated saliva could not penetrate below the gingival margin and felt the gingiva could defend itself against injury due to the close approximation of the "epithelial cuff" around the tooth.

Waerhaug and Steen (1952) observed the histologic tissue reaction over a forty-eight hour time period after the deposition of pure cultures of pathogenic bacteria into bacteria free gingival pockets of dogs. Conclusions drawn included: 1. Bacteria within the gingival sulcus result in epithelial necrosis and inflammation of the connective tissue with a subsequent exudate formation. 2. "From all pockets, there is a

constant flow of cellular elements and tissue fluid."

Brill and Bjorn (1959) in attempting to differentiate crevicular epithelium from other oral and nasal epithelium; studied the permeability of human crevicular epithelium to orally administered fluorescein molecules. Both the epithelial lining of the gingival crevice and the nasal mucosa proved to be permeable to fluorescein molecules. A correlation between the amount of inflammation present and the quantity of fluorescein recovered on filter strips placed in the gingival sulcus was observed in that patients with extensive restorations as opposed to non-restored teeth lead to the recovery of a greater amount of fluorescein.

Brill and Krasse (1959) demonstrated an increased flow of tissue fluid from the gingival sulcus that followed short term mechanical stimulation (externally and within the sulcus) of clinically healthy marginal gingiva that returned to the original flow rate within ten minutes in their studies with dogs. Two explanations for the increased fluid flow were postulated: 1. Mechanical stimulation of a vascular bed results in arteriole dilation with subsequent increased pressure and increased permeability allowing the egress of plasma, and thereby increasing the concentration of interstitial fluid. 2. A sol $\longleftrightarrow$ gel mechanism exists for the ground substance of the connective tissue matrix that remains a gel unless mechanical stimulation is applied and the sol state results.

Brill (1959) injected dogs with Evan's Blue (vital dye T-1824 that binds to plasma albumin in low concentration and a globulin in higher concentrations) to evaluate the modification of gingival fluid flow under

the influence of histamine, mechanical stimulation and the inflammatory state. Brill postulated the dye bound to plasma proteins could be employed to evaluate changes in the gingival vessel permeability, and this change could be measured by the quantity of dye that was recovered via filter paper strips placed into the sulcus. Conclusions included protein bound Evan's Blue from plasma can pass through capillary walls in minute concentrations and is observed in the gingival sulcus following mechanical stimulation, IV injection of histamine, and inflammation.

Brill (1959) demonstrated chewing paraffin in human subjects lead to an increase in the gingival tissue fluid flow and would affect the state of the gingival health. "When gingival structures are stimulated by chewing, the antimicrobial effect may be increased, because mechanical stimulation of the gingival vascular bed stimulates escape of fluid from the vessels and plasma contains several antimicrobial substances."

Brill (1959) observed the flushing action of gingival fluid on non vital particles and living bacteria. "The flow of tissue fluid from sub epithelial structures of marginal gingiva is able to remove particulate matter, including bacteria, from gingival pockets." Brill coined the term "pocket" to be that space located between the gingival epithelium and the tooth substance.

Brill and Bronnestan (1960) utilized immunoelectrophoretic analysis of human sulcular fluid to determine the presence of seven different serum protein components including alpha 2 macroglobulin, beta and gamma globulin. They postulated the source of the gingival fluid is tissue

and is formed extracellularly via capillary filtration.

Gavin and Collins, (1961) disputed Waerhaug and Steen's 1952 conclusion that the healthy gingiva is sterile through their investigation into the bacteriologic status of the clinically healthy human gingiva. "The clinically healthy gingival crevice appears to contain microorganisms in the majority of cases." A subsequent study (1961b) lead to the conclusion human gingival fluid from clinically healthy gingival crevices exhibits neither bacteriostatic nor bacteriocidal effects on oral bacteria including Neisseria, Diptheroides, Streptococci, and Staphylococci.

Löe (1961) in his studies of the epithelial cell turn over rate made the following conclusions upon histologic examination: 1."Mitotic figures along the entire length of the epithelial lining of the pocket and the desquamation of the surface cells support the view that the epithelial cuff is constantly renewed", 2. The aggregation of neutrophilic leukocytes within the gingival sulcus indicated "they migrate through the epithelial lining under physiological conditions," and 3. "There is a continuous transudation of tissue fluid into the clinically normal gingival pockets."

Gustafsson and Nilssen (1961) noted in their studies of gingival fluid from clinically healthy human gingiva since fibrinolysis could be observed, plasmin and activator must be present in the crevicular fluid. "The fibrinolytic factors in the crevicular fluid might be of significance in counteracting the deposition of fibrin and other proteins at the junction between the gingival epithelium and the tooth."

Harvey (1962) upon observation of the physiologic flow of tissue fluid in flushing extraneous matter from the sulcus of dogs postulated "the normal gingival crevice maintains its hygienic state by constant flushing with tissue fluid which is increased as a result of an acute inflammatory response to irritation."

Krasse and Egelberg (1962) utilized flame photometry to analyze the Na/K ratio of human crevicular fluid and concluded "gingival pocket fluid cannot be regarded as a simple filtration product but rather as an inflammatory exudate."

Egelberg (1963a) in his comparison of cellular content of gingival sulcus fluid from clinically healthy gingiva to fluid from chronically inflamed gingiva observed that although no difference in quality of cells could be detected, an increase in inflammatory cells compared to epithelial cells from the inflamed gingival samples could be observed. Again, his conclusion was "fluid in healthy pockets may be regarded as an inflammatory exudate."

Egelberg (1963b) demonstrated that topical application of histamine solution to marginal and attached gingiva of both human subjects and dogs was able to penetrate the gingival crevicular epithelium but not the attached gingival epithelium. This contradicted Waerhaug (1955) and Brill (1959c) who concluded the gingival fluid was capable of preventing penetration of substances into the crevicular area.

Mann (1963) collected gingival fluid from human subjects on filter paper strips after oral administration of fluorescein sodium (325 mg.).

Results indicated as the severity of the gingival inflammation increased, the crevicular fluid flow increased, and "inflammation, (not pocket depth), was the main factor contributing to the rate of fluid flow."

Egelberg (1964) studied crevicular fluid (human and canine) to determine if a relationship existed between clinical and histologic criteria for gingival inflammation and the quantity of gingival exudate recovered by filter paper strips. Conclusions included: 1. A higher degree of inflammation was observed in the gingival papilla area. 2. A correlation existed between the quantity of gingival exudate recovered on filter paper strips and the area of inflammatory cell infiltrate.

Brandtzaeg and Mann (1964) in their study of the lysozymal activity of human gingival fluid, serum and saliva (from patients with either gingivitis or periodontitis) concluded crevicular lysozymal activity increased with the severity of inflammation; whereas serum and saliva did not. "The enzyme in gingival fluid is assumed to be primarily of local origin, possibly derived from leukocytes."

Weinstein and Mandel (1964) attempted to classify crevicular fluid as specifically altered transudate from serum since: 1. Sulcular cells are constantly sloughed off and the intracellular contents become part of the transudate. 2. An active ionic transport mechanism may be exhibited by the cells. 3. A cytocellular fluid may be contributed by crevicular cells through micropores in the cell walls.

Löe and Holm-Pedersen (1965) employed both an extracrevicular technique and an intracrevicular technique to measure crevicular fluid.

Biomicroscopic techniques lead to the revelation that deep insertion of filter paper strips into the sulcus resulted in capillary compression. Conclusions included the healthy human gingival crevices do not exhibit fluid flow (disputing the conclusion of Brill, 1962) and "mechanical stimulation of the periodontium does not produce fluid from such crevices (disputing the conclusions of Brill, 1959b and Brill and Krasse, 1959)." In addition, crevicular fluid flow commences prior to clinical manifestations and persists for some time after clinical inflammation indices have disappeared. "Gingival fluid is an inflammatory exudate and that the absence or presence of fluid may represent the definite clinical criterion in the refined distinction between normal and inflamed gingiva."

Sueda's, et al., (1966) histochemical study of human gingival fluid, blood serum and saliva lead to confirmation of gingival fluid components; proteins, lipids, and polysaccharides bound to proteins (muco-, lipo-, or glyco-proteins). The results of their study seemed to confirm Cimasoni's (1966) hypothesis that the mucopoly-saccharide substance identified by Toto and Sicher (1964) located between enamel and crevicular epithelium in humans could represent a condensation of gingival fluid.

Stallard (1967) utilized the injection of plastic microspheres into the external carotid artery of squirrel monkeys to study periodontal microcirculation. Microspheres became trapped in areas of chronic inflammation "possibly associated with abnormal coiling of microvessels observed beneath the "col" and sulcular epithelium." Stallard noted the

existence of a correlation between the integrity of microvasculature within the sulcular epithelium and the presence of gingival crevicular fluid. "Initially, the inflammatory reaction, with its characteristic vascular alterations, is a physiologic defense mechanism; however, it appears that in the cases of periodontal disease, the inflammatory response eventually becomes pathologic."

Weinstein, et al., (1967) demonstrated and characterized the presence of gingival crevicular fluid in human gingival sulcus labelled, "clinically normal". The authors cited deviation in criteria for "clinically normal" and uncontrolled differences in technique for their contradiction of the findings of Löe, et al., (1965). The technique utilized by Weinstein (fluorescein labelling) was one hundred times more sensitive in detecting proteins than the technique employed by Löe (ninhydrin staining).

Nagao (1967) studied crevicular fluid by weighing filter paper strips before and after fluid collection and by ninhydrin staining. He concluded that crevicular fluid flow increases following the insertion of crowns and higher crevicular fluid levels were associated with crowns that did not fit well as opposed to a well fitting prosthesis.

Sandalli and Wade (1969) concluded the following from their examination of crevicular fluid volume following periodontal surgery: Gingival fluid flow was reduced following scaling, polishing, and supervised oral hygiene. (Gingival fluid flow decreased four weeks post operatively in concurrence with Löe and Holm-Pedersen's findings, 1965). "There is

a relation between the depth of pocket and the amount of gingival fluid: measuring this amount may be of value in scoring the periodontal status."

Orban and Stallard (1969) from their studies to determine if a correlation between fluid volume and clinical scoring techniques and biopsy specimens existed; postulated alteration of the intercellular cementing substance occurs through numerous factors (hyaluronidase, chewing, brushing, gingival massage, circadian rhythm and hormones) with the overall result being an increase in the permeability of the sulcular epithelium. In addition, crevicular fluid scores did not correlate with biopsy scores based upon inflammatory infiltrate present. "A better indication of inflammatory status of the gingival tissues, as revealed by biopsies, is the evaluation or measurement of dental plaque."

Oliver, et al., (1969) in human studies to determine if a relation between gingival index (Löe and Silness, 1967), gingival fluid volumes, and histologic inflammatory cell densities existed, concluded correlation exists between gingival index system and the volume of gingival exudation (ninhydrin staining technique). In patients with "no clinical evidence of gingival inflammation; there is no exudate in the vast majority of crevices."

C. Recent Literature

Egelberg and Attstrom (1973) in human and canine studies to

evaluate the orifice and intracrevicular methods for measuring gingival fluid concluded both methods were "comparable for evaluation of intra-individual changes of gingival inflammation" and "the orifice method showed statistically significant differences at a few more time point comparisons than the intra crevicular method." The orifice method demonstrated less variation between samples.

Alfano (1974) in an attempt to explain the mechanism and origins of gingival fluid be it a physiologic transudate or a pathologic inflammatory exudate concluded gingival crevicular fluid arises via two pathways: 1. Generation of a standing osmotic gradient by macro-molecular by-products of the bacteria present in the subgingival plaque and 2. The initiation of the classical inflammatory pathway. "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." Borden, et al., (1974) utilized a fluid meter (Harco Electronics LTD., Winnipeg, Canada) to compare extra crevicular and intra crevicular collection techniques and note any correlation between gingival fluid flow and the gingival index (Löe and Silness, 1967). Results obtained indicate repeated intra crevicular measurements yielded similar gingival fluid flow levels but did not significantly stimulate the flow of fluid within the sulcus. The technique recommended for measurement of gingival fluid with the gingival crevicular fluid meter includes depleting the crevicular fluid pool present with a sterile filter paper strip followed by a thirty second

interval and placement of a new sterile filter strip for three seconds to measure the gingival crevicular fluid flow. The three second intracrevicular measurement of gingival crevicular fluid proved to be more sensitive than the extracreviuclar technique.

Shern, et al., (1974) in comparison studies of ninhydrin staining method and the crevicular fluid flow meter (Harco) for quantification of human crevicular fluid flow found "precision, accuracy and reliability of measuring crevicular flow proved greater using a flow meter than using the ninhydrin dye method". A combination of gingival crevicular fluid meter fluid flow measurements and cytological smears was suggested as "valuable physiologic measurements for clinical trials."

Golub, et al., (1974) in studies on collagenolytic activity of human gingival crevicular fluid found collagenolysis in human gingival crevicular fluid was associated with gingiva that exhibited an increase in inflammation.

Lie and Selvig (1975) constructed an experimental dental cuticle on surfaces of human enamel, dentin, cementum, and a control material (epoxy resin with exposed synthetic hydroxyapatite crystals). They postulated the dental cuticle is partly formed by "adsorption of material from serum and tissue fluid which may have seeped through the junctional epithelium as gingival exudate"; and the cuticle is not an anatomical structure but is derived from "the ubiquitous presence of inflammation of the gingiva and exudation through the junctional epithelium."

Holm-Pedersen, et al., (1975) in induced human gingivitis studies determined gingival inflammation was more rapid and aggressive in the elderly and was accompanied by a slightly slower tissue recovery once oral hygiene was reinstated. This age differential was more pronounced with gingival crevicular flow measurements than with the gingival index (Löe and Silness, 1967) and was therefore deemed more sensitive. "Observed differences in development of gingivitis suggest an altered host response to the plaque microorganisms with age".

Squier and Johnson (1975) in discussing the nature of the permeability of the oral mucosa noted most substances utilize diffusion and obeyed Fick's law in which "the rate of penetration is directly proportional to the concentration of the penetrant" in movement across skin and oral mucosa. The skin and oral mucosa were found to differentiate based upon polarity; thereby, only facilitating the diffusion of polar molecules and macromolecules. It was postulated foreign substances utilize intercellular pathways to enter junctional epithelium as no intercellular barrier exists in junctional epithelium as opposed to the membrane coating granules present in oral epithelium that affect permeability.

Golub and Kleinberg (1976) in their review of the literature pertinent to crevicular fluid and clinical applications in periodontal therapy agreed with Alfano (1974) in concluding by-products of plaque modulate crevicular fluid; but the mechanism postulated was plaque macro molecule by-products increase crevicular fluid flow directly by

alteration of the crevicular epithelium and connective tissue cells. Based upon the fact that gingival crevicular fluid flow increases prior to clinical observation of gingival pathology, they suggested collection of gingival fluid on filter paper strips as an aid to screening individuals for systemic diseases following the development of biochemical and microbiological analyses of components of gingival crevicular fluid.

Suppipat (1976) utilized the HAR-600 Gingival Crevice Fluid Meter (Harco Electronics LTD., Winnipeg, Canada) in clinical research and determined if the orifice collection method is employed (Löe and Holm-Pedersen, 1965), clinically healthy gingiva yielded at best very slight amounts of gingival fluid and a greater correlation exists between gingival inflammation and fluid flow than with gingival inflammation and pocket depth (Mann, 1963).

D. Clinical Literature

Waerhaug (1960) in histological evaluations of crown margins in relation to gingival inflammation determined plaque to be the etiology of gingival inflammation irregardless of the material employed in the construction of the restoration or its degree of surface roughness. While subgingival margin determination was found to be contributory, the cause of the inflammatory response was plaque and by-products of component microorganisms. Decisions regarding termination of a restoration margin must include an accurate assessment of incidence of caries, oral hygiene, esthetics and potential for periodontal disease.

Morris (1962) discussed crown contour and its influence on gingival

health and concluded (contrary to Amsterdam, et al., 1959) artificial buccal and lingual crown contours contribute to gingival pathology rather than prevent it. In conclusion, he re-emphasized the fact crown contour should contribute to proper muscle action and gingival health.

Loe (1962) discussed the concept of extension for prevention in relation to crown restorations and concluded "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."

Marcum (1967) concluded from histological margin termination studies on dogs crown margins terminated at the gingival crest resulted in a minimal inflammatory response; however, a slight to severe inflammatory response was associated with crown margins terminated either above or below the gingival crest. Citing "plaque formation and adherence of food debris" as an explanation for inflammation associated with margins above the crest of the marginal gingiva, he also concluded "better marginal finish and a better crown contour that deflects food away from the gingival crevice" contributed to the minimal inflammatory response associated with margin termination at the crest of the marginal gingiva.

Waerhaug (1968) discussed the fact margin termination of crowns should be supragingival to minimize the contributory effect of plaque retention on crown margins to periodontal disease. "Many patients would

be better off if bridges or partial dentures were not constructed."

Mount (1970) in an analysis of literature pertinent to crown gingival margin termination concluded adequate preparation is the most critical component when the gingival crevice is involved.

Berman (1973) stated "the anatomy and physiology of the sulcus, coupled with refractory vision, present an insurmountable task in preparation." Clinicians do not possess an innocuous technique of tooth preparation that is non injurious to the delicate gingival soft tissues.

Trivedi and Talim (1973) performed clinical and histologic examinations on fifty-four human teeth in which Class V restorations of polished amalgam, unpolished amalgam, silicate cement, acrylic resin or gold alloy were placed. Clinically, no changes in the gingiva were noted adjacent to the restorations; however, histologic examination revealed the greatest degree of inflammation in tissue adjacent to those teeth restored with silicate cements and acrylic resin, and no inflammation was associated with the gold alloy restoration. One third of all histological samples did not reveal any incidence of pathology irregardless of the material employed in the restoration. "Gingival response appeared to be caused by chemical injury, unpolished restorative materials, poor margin adaptation and inadequate oral hygiene."

Volchansky, et al., (1974) performed scanning electron microscopic studies to evaluate the surface topography of enamel, calculus, cementum, gold inlay, porcelain fused to gold, and amalgam. In addition, a Taylor-Hobson Talysurf model 3 surface roughness testing machine was employed

to determine five distinct levels of surface roughness and concluded "enamel is probably the smoothest and most acceptable surface in the mouth, and all natural and restored surfaces should be compared to it."

Mormann (1974) observed the effect either polished or roughened inlays had upon the crevicular fluid rate (utilizing ninhydrin staining technique) and plaque formation (utilizing the Löe-Silness scoring technique, 1967). The margins were well adapted and terminated at least one millimeter into the sulcus. Mean gingival fluid flow rates for non-restored teeth were significantly lower than fluid flow rates from the restored teeth. In addition, the rough surface served as a nidus for plaque formation to a greater extent than did smooth polished surfaces. In conclusion, "even perfectly adapted and well polished proximal gold inlays cause gingival inflammation."

Ranfjord, et al., (1974) in his review of a longitudinal study from the University of Oslo that pertained to margin termination of bridge abutments emphasized the following: 1. Subgingival margin termination was directly correlated to periodontal pathology and did not yield the anticipated protection against recurrent caries., 2. Gingival recession will occur subsequent to long term provisional utilization,, and 3. If the sulcular epithelium is invaded or removed, the potential for increased sulcular epithelial permeability is increased.

Newcomb (1974) employed clinical studies to assess the degree of gingival inflammation in relation to subgingival margin termination utilizing Gingival Index, Löe Silness Gingival Plaque Indes, 1967, and pocket

depth determination. Conclusions obtained were; 1. "The nearer a subgingival crown margin approaches the base of the gingival crevice, the more likely it is that severe gingival inflammation will occur," and 2. "Least amount of inflammation is observed when subgingival crown margins are placed at the gingival crest or just into the gingival crevice."

Strauss, et al., (1975) employed the Gingival Crevice Meter (Harco) to examine subclinical inflammatory changes under major connectors of mandibular partial dentures and determined changes in crevicular fluid flow can be measured with no changes in pocket depth or in Löe-Silness Gingival Index, 1967. "Removable partial denture components can modify the crevicular flow of adjacent gingival crevices."

Burch (1975) emphasized the necessity for supragingival margin termination wherever possible citing buccal, lingual, proximal and transitional line contours in restorations as mandatory aspects in the maintenance of gingival health.

Mahajan (1976) performed histological evaluations of inter dental papillae adjacent to full coverage restorations and non restored teeth. Conclusions included: 1. Clinically normal tissue adjacent to non restored teeth exhibited few inflammatory cells compared to normal tissue adjacent to restored teeth which showed inflammatory cell infiltrate and dilated blood vessels., 2. Inflamed gingiva next to restored teeth demonstrated heavy inflammatory cell density and dilated blood vessels., 3. A loss of collagen fibers around blood vessels was observed in inflamed areas., and 4. The squamous epithelium mitotic index increased with full coverage restorations.

Caruso (1979) in histological evaluation of crown contour of cast full coverage restorations in Rhesus monkey studies utilizing parameters of gingival index, plaque index, crevicular fluid volume and inflammatory cell density concluded both overcontoured and undercontoured cast crowns exhibited adverse gingival tissue response. "Undercontoured cast crowns with margins terminating at or slightly above the gingival crest did not effect the gingival tissue as much as overcontoured crowns."

CHAPTER III

MATERIALS AND METHOD

The purpose of this investigation is to determine and compare the inflammatory changes in the marginal gingiva of abutment teeth for fixed and removable prostheses.

Data was obtained from those patients seeking treatment at the Loyola Dental School clinic. Thirty patients that had an existing partial denture with at least one restored abutment (full coverage) and a non restored tooth in the same dental arch were utilized as candidates for the thesis. Parameters of Gingival Crevicular Fluid volume, Löe-Silness Gingival Index (1967), and determination of the depth of the gingival sulcus were recorded for the restored abutment under the existing partial denture, the non restored tooth (control), as well as for a second abutment of the partial denture. In addition, the type of material utilized to fabricate the full coverage restoration, the margin termination of the restoration, the total sum of the edentulous span, the type of partial denture and type of opposing dentition were recorded for each candidate.

Data was also obtained from thirty patients that had fixed prosthodontic restorations including a terminal abutment for a fixed prosthesis, a single full coverage restoration and a non restored tooth (control) in the same dental arch. The parameters recorded included the Gingival Crevicular Fluid volume, Löe-Silness Gingival Index (1967),

and determination of the depth of the gingival sulcus.

The method of Gingival Crevicular Fluid collection employed involved initially isolating the area to be measured with a sterile cotton roll and gently drying the marginal gingiva with a warm air syringe.

The existing pool of crevicular fluid present in the gingival sulcus was emptied by gently inserting a sterile filter paper strip (see Figure 4) into the orifice of the gingival sulcus and allowing it to remain in place for an elapsed time of three seconds before it was removed and disgarded. A second sterile filter paper strip was then inserted into the orifice of the gingival sulcus (see Figure 5), allowed to remain in place for twenty-seven seconds, removed and immediately inserted between the recording sensors (see Figure 6) of the Gingival Crevicular Fluid Meter so that the entire moistened area of the filter paper was in contact with the sensors (the filter paper strip is inserted to the line indicated on each strip as indicated in Figure 4).

The digital read out value of the Gingival Crevicular Fluid Meter (Periotron-Harco Electronics LTD., Winnipeg, Canada) rises to a maximum level and then decreases. The value recorded was the highest numerical reading obtained. Digital numerical values are converted to fluid volume (microliters) by dividing the readings by a conversion factor of 200 (eg. a digital read out of 10 = 0.05 ul)

Following each measurement, the sensors were dried with a sterile cotton roll.

In the aforementioned manner, data was obtained from the orifice of the gingival sulcus adjacent to the mesiobuccal and distobuccal aspects of each tooth measured.

In addition to the parameter of the Gingival Crevicular Fluid volume, a periodontal probe was utilized to determine the mesial and distal depth of the gingival sulcus (see Figure 7) and a Gingival Index determination was obtained utilizing the Löe-Silness Gingival Index as outlined below.

Gingival Index - Löe and Silness

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.

Information from each data sheet (see Appendix) was compiled into single tables (see Tables 1 through 5) and the mean values for all measured parameters computed.

The data was subjected to a t-test analysis to determine if a statistically significant difference existed in the Gingival Crevicular Fluid volumes between the restored single unit full coverage restoration and the restored single unit full coverage restoration (abutment) under an existing partial denture; as well as to determine if a statistically

significant difference existed in the Gingival Crevicular Fluid volumes between the restored abutment for fixed prosthesis and the restored abutment under an existing removable prosthesis (partial denture).

Regression analysis was also employed to determine which variables of an abutment for a removable prosthesis contributed significantly to the Gingival Crevicular Fluid volumes obtained.

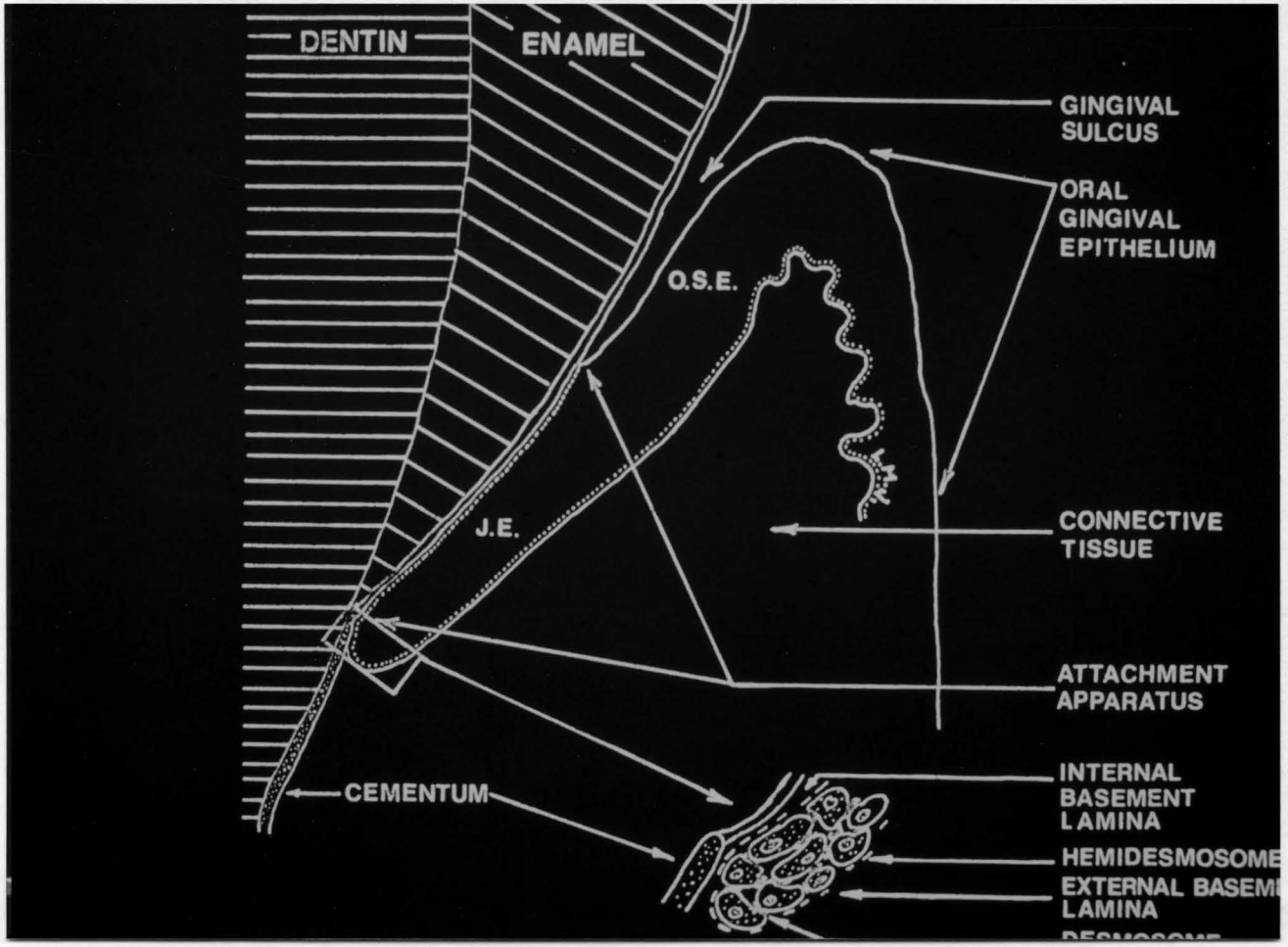

Figure 1: Diagram of the dentinoepithelial junction. (After L.M. Jameson)

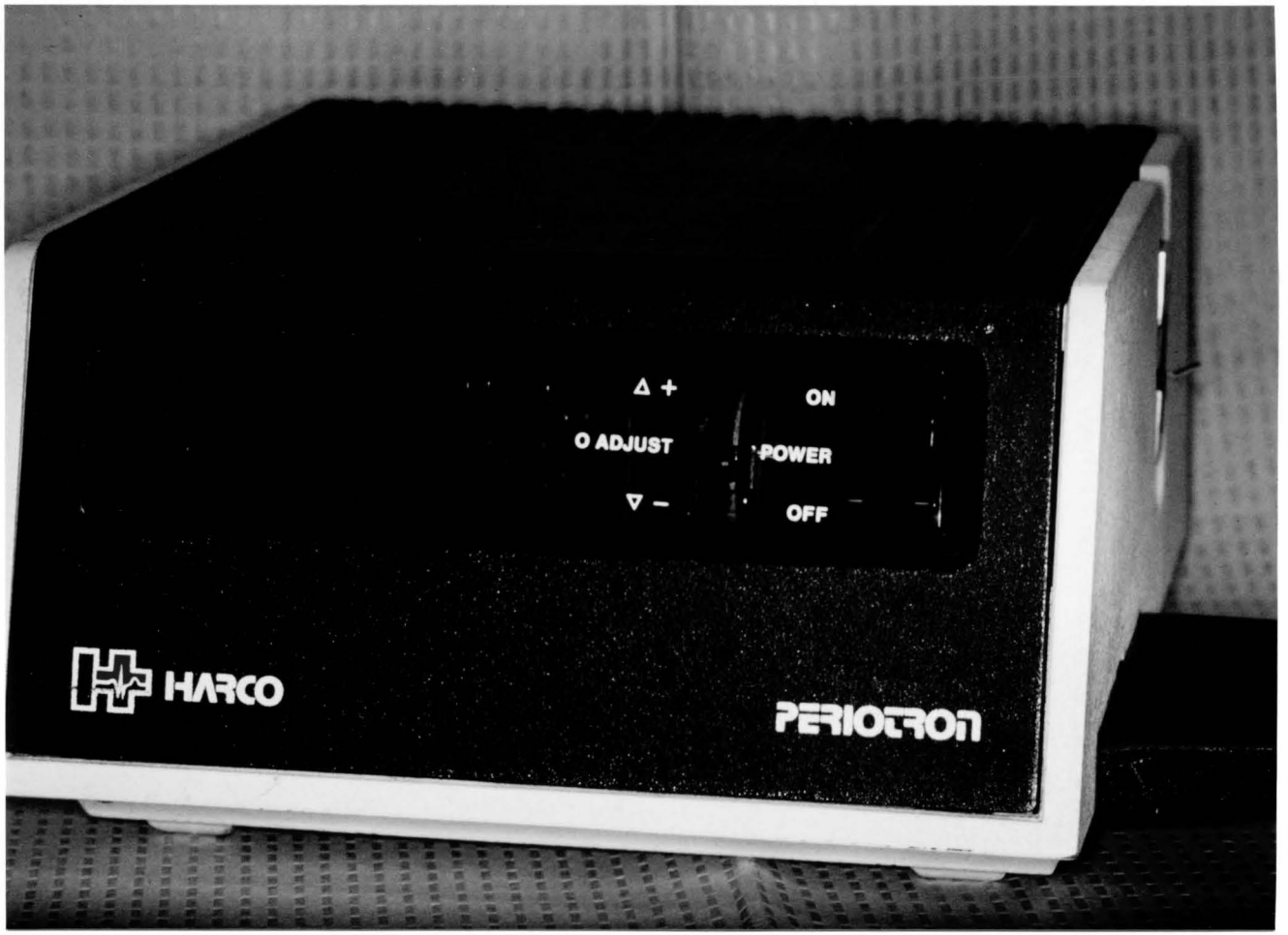

Figure 2: Periotron (Gingival Crevicular Fluid Meter - Harco Electronics LTD., Winnipeg, Canada).

Figure 3: Sterile Filter Paper Strips (Periopaper-Harco Electronics LTD., Winnipeg, Canada).

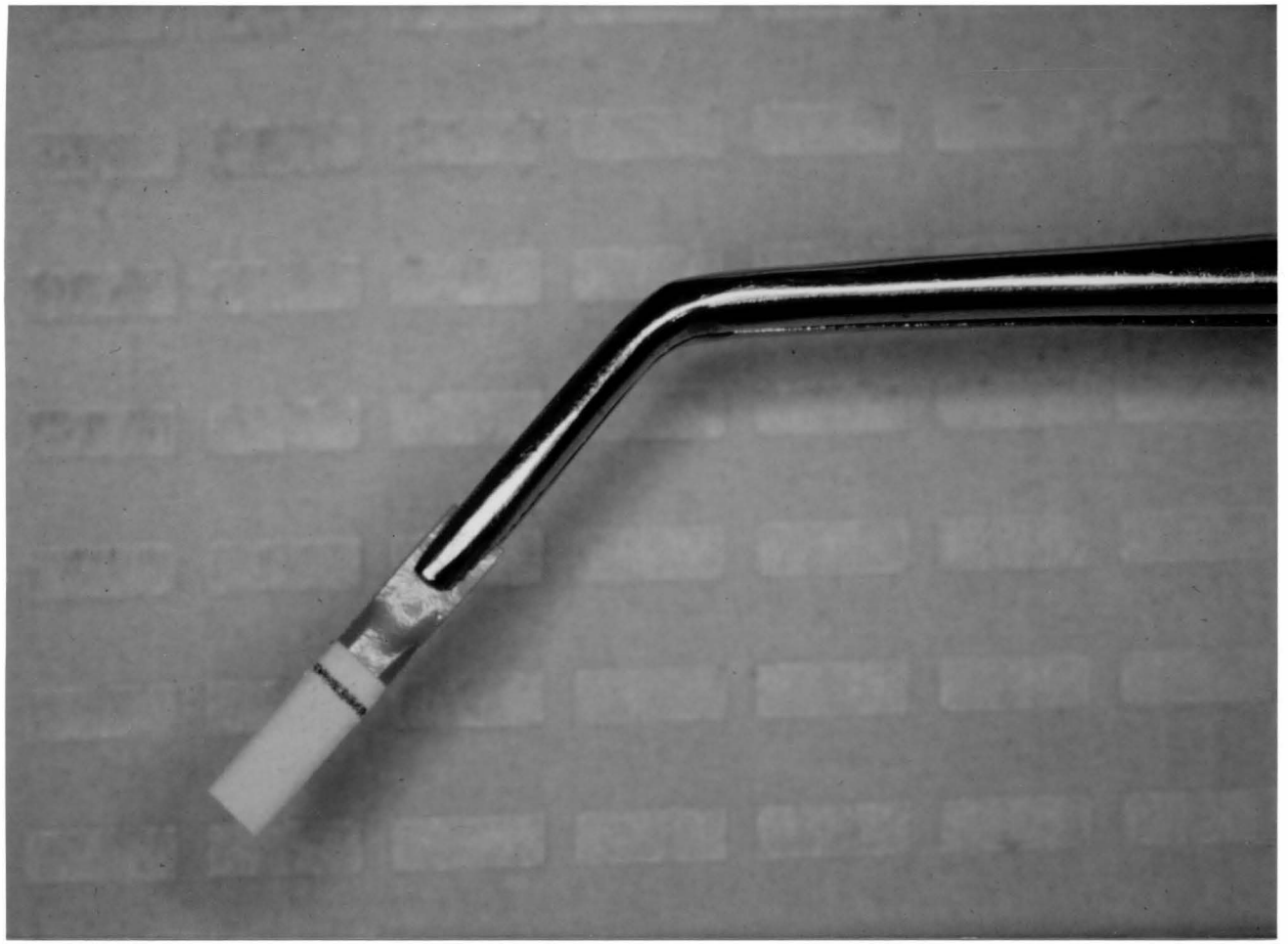

Figure 4: Filter Paper Strip Prior to Collection of Crevicular Fluid.

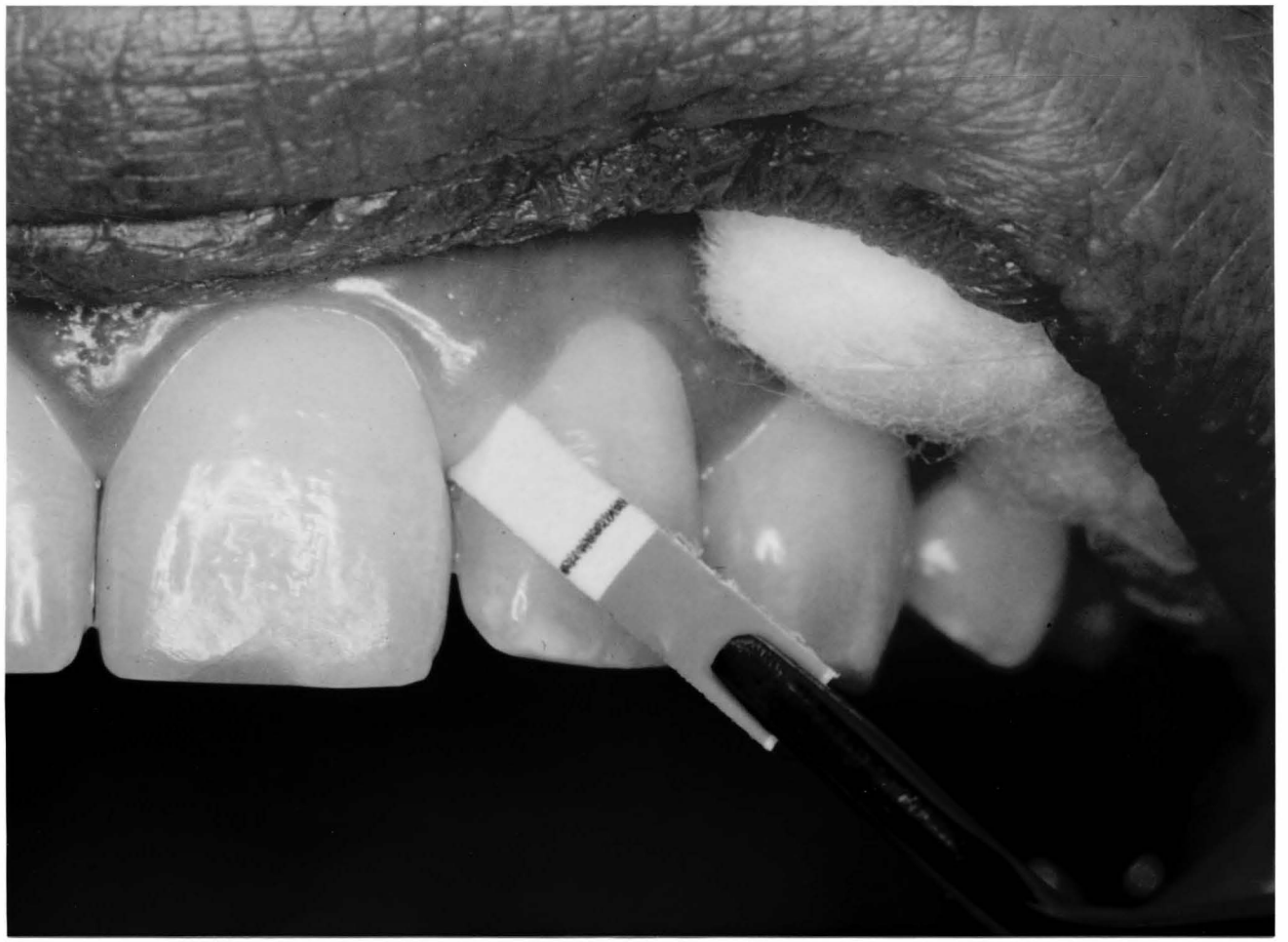

Figure 5: Gingival Crevicular Fluid Collection Technique.

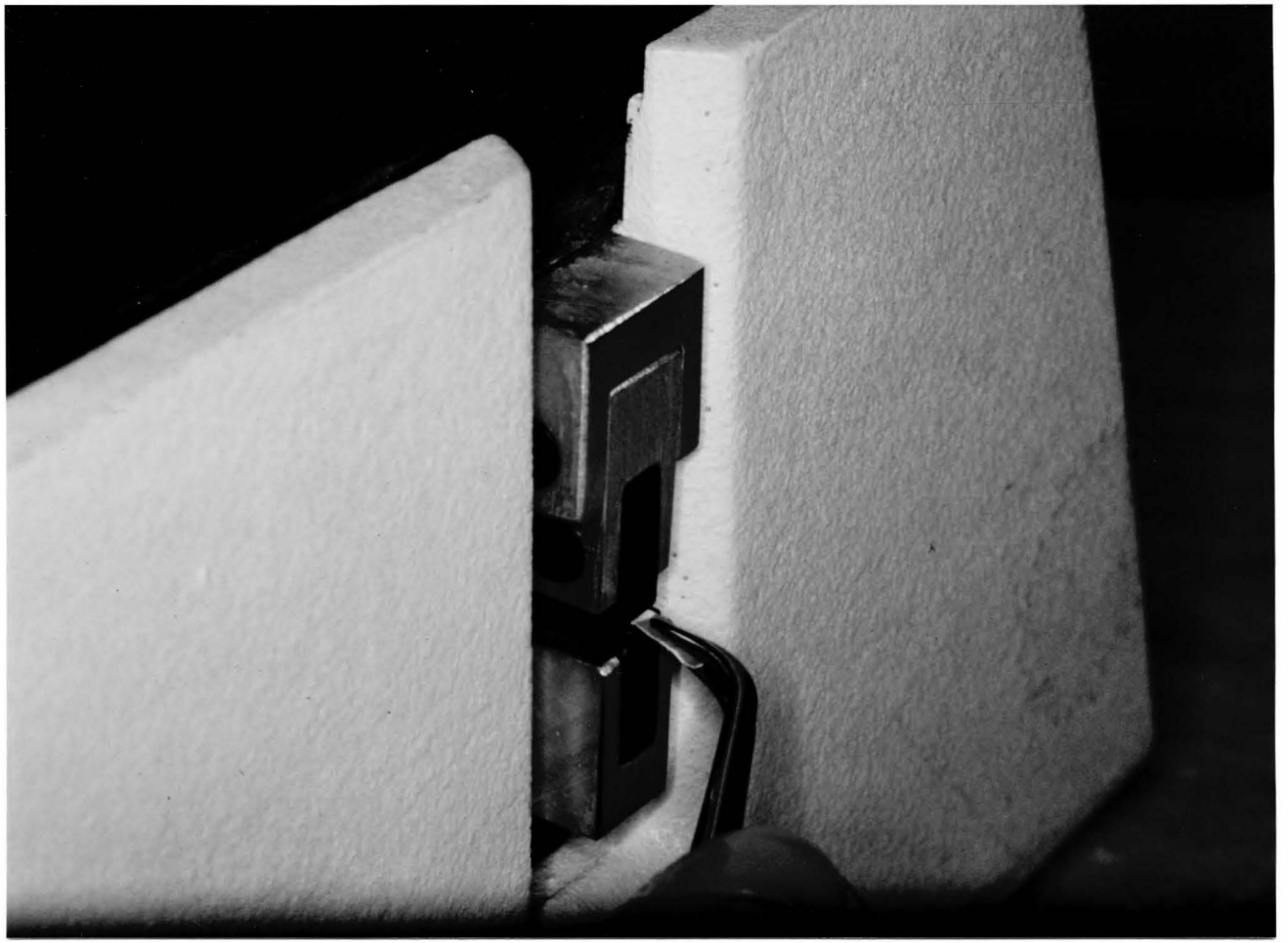

Figure 6: Placing Filter Paper Strip Between Recording Sensors of the Gingival Crevicular Fluid Meter (Periotron).

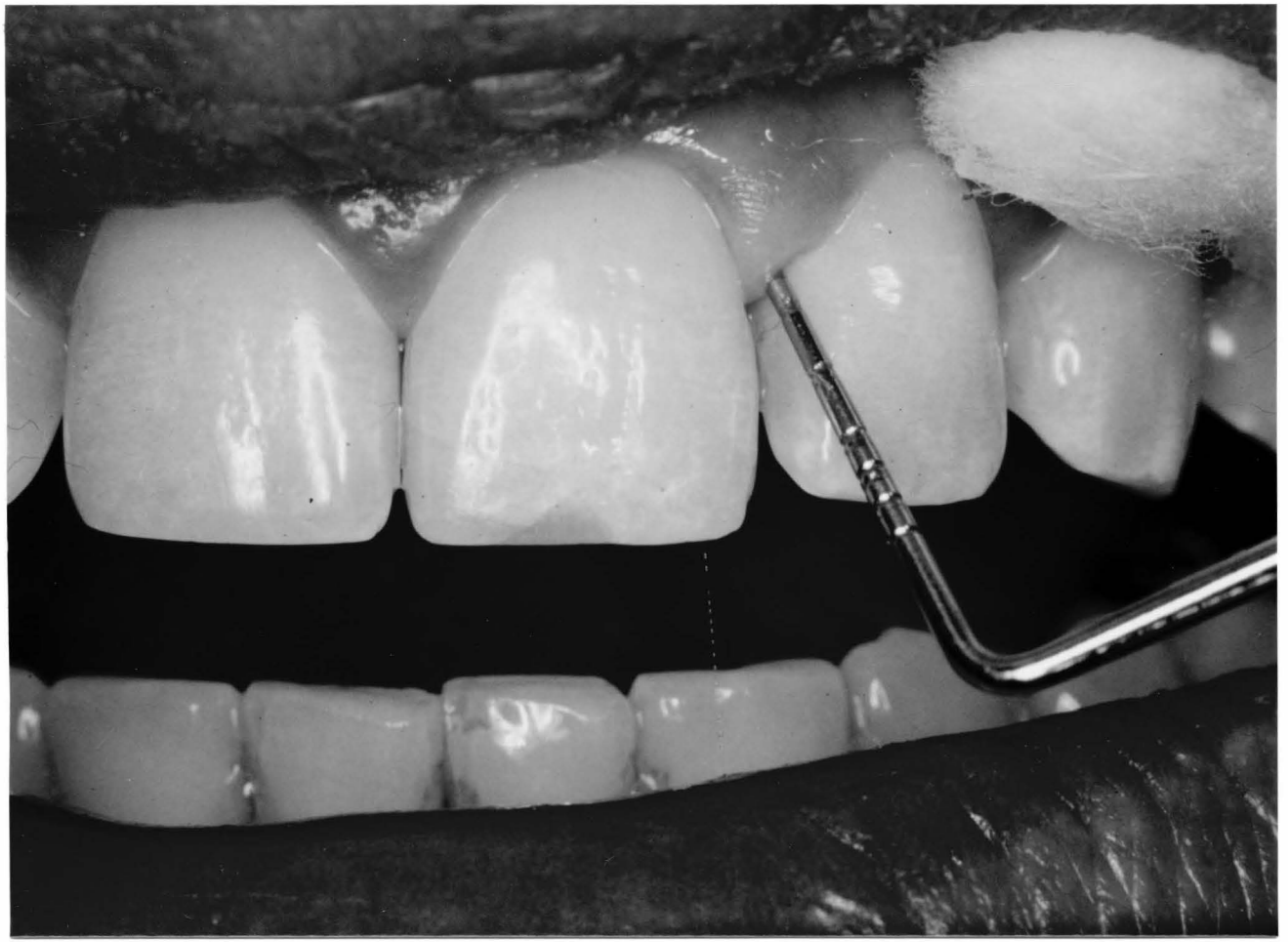

Figure 7: Measuring the Depth of the Gingival Sulcus.

$M + AgAb \rightarrow MAgAb$

$MAgAb + Ca^{++} \rightarrow MAgAb\ C\ \overline{1}$, activated C 1

$MAgAb + C\ 4 \rightarrow MAgAb\ C\ \overline{1\ 4}$

C 1 esterase cleaves

C 4 and C 2 attached to membrane M

Mg^{++}

$MAgAb\ C\ \overline{1\ 4} + C\ 2 \rightarrow MAgAb\ C\ \overline{1\ 4\ 2}$

C 4 2 an enzyme activated next C 3 esterase

$MAgAb\ C\ \overline{1\ 4\ 2} + C\ 3 \rightarrow MAgAb\ C\ \overline{1\ 4\ 2\ 3} +$

C 3 fragment, C 3a, which is chemotactic for leukocytes and is vasoactive.

C 3 cleavage also occurs by C 3 activator in the "alternative" pathway.

$MAgAb\ C\ \overline{1\ 4\ 2\ 3} + C\ 5, C\ 6, C\ 7 \rightarrow MAgAb\ C\ \overline{1\ 4\ 2\ 3\ 5\ 6\ 7} +$

C 5 fragment, C 5a, which is chemotactic for leukocytes and is vasoactive.

Figure 8: Pathway of Complement Activation in Gingival Tissue (M). (Suzuki, J., Gargiulo, A., and Toto, P.)

CHAPTER IV

EXPERIMENTAL RESULTS

Table one is the compiled data from individual data sheets (see Appendix) listing the sex, age, type of existing partial denture, teeth involved in the collection of data, designation of type of abutment or control, type of restorative material, margin termination, gingival crevicular fluid volumes, Löe-Silness index, pocket depth, opposing dentition and length of edentulous span.

Table two lists the frequency distribution for sex, material utilized for crown restoration; and mean values for age, gingival crevicular fluid volume, Löe-Silness index and pocket depth for the fixed prosthodontic non restored tooth (control), restored bridge abutment, and single restored tooth.

Table three lists the frequency distribution for sex, type of partial denture, type of opposing dentition; and mean values for age, gingival crevicular fluid volume, Löe-Silness index, pocket depth, and total length of edentulous span for the removable prosthodontic non restored tooth (control), restored abutment under a partial denture, and non restored abutment under a partial denture.

Table four summarizes the mean values for the various categories under the fixed prosthodontic groups. The lowest mean gingival crevicular fluid volume values, Löe-Silness index, and pocket depth determinations were associated with the non restored (control) group. These

values increased for the restored single crown; however, the highest mean values were associated with the restored bridge abutment.

Table five summarizes the mean values for the various categories under the removable prosthodontic groups. The lowest mean gingival crevicular fluid volume values, Löe-Silness index, and pocket depth determinations were associated with the non restored (control) group. These values increased for the non restored partial denture abutment group; however, the highest mean values were associated with the restored partial denture abutments.

Table six summarizes mean values of gingival crevicular fluid volume, Löe-Silness index and pocket depth determination for all comparison groups.

Table seven summarizes the t-test analysis comparing the gingival crevicular fluid volume, Löe-Silness index and pocket depth determination between the single unit restored tooth (Fixed Prosthodontics) and the restored abutment under an existing partial denture. The gingival crevicular fluid volume is statistically higher ($p < .001$) for the restored abutment under a partial denture as compared to a single restored tooth.

Table eight summarizes the t-test analysis comparing the gingival crevicular fluid volume, Löe-Silness index and pocket depth determination between the restored bridge abutment (Fixed Prosthodontics) and the restored abutment under an existing partial denture. No statistically significant difference ($p < .001$) occurs between the two groups

for either total fluid volume, Löe-Silness index or pocket depth determination.

The data from the Removable Prosthodontic categories was subjected to a multiple regression analysis using Mesial and Distal gingival crevicular fluid volumes, and Löe-Silness index as dependent variables and employing the type of opposing dentition, material for the crown, margin termination, type of partial denture and total length of edentulous span as independent variables. Table nine highlights the statistically significant ($p < .001$) independent variables of type of material used in construction of the crown and margin termination that had the greatest influence upon the increased gingival crevicular fluid volumes and Löe-Silness index.

NO.	SEX:	AGE:	TYPE OF PARTIAL:	TOOTH #:	ABUTMENT OR CONTROL:	CROWN:	MARGIN PLACEMENT:	GCF LEVEL: vol.µl	LÖE-SILNESS INDEX:
	1=Fe 2=M		A=Max. B=Mand. 1=Bilateral distal ext. 2=Unilateral distal ext. Right 3=Unilateral distal ext. Left 4=Tooth supported.		A=Non restored abutment. B=Control C=Restored abutment	1=PFM 2=CVGC 3=Acrylic to gold 4=PJC 5=Enamel	1=Sub. 2=At Gingival crest. 3=Supra. 4=Natural tooth.	M. D.	M. D. 0=Absence of inflammation 1=Mild inflam. 2=Moderate inflammation 3=Severe inflammation

POCKET DEPTH: mm.		OPPOSING DENTITION:	LENGTH OF SPAN:
M.	D.	1=Partial denture 2=Denture 3=Natural dentition 4=Natural and fixed	R.L. ANT.

DETERMINATION OF THE GINGIVAL CREVICULAR FLUID VOLUMES ASSOCIATED WITH RESTORED (FIXED AND REMOVABLE PROSTHODONTICS) AND NON-RESTORED ABUTMENTS.

Pamela Denise Golasz, D.D.S.
Loyola University School of Dentistry
June, 1980

Table 1: Pages 40 through 53

NO.	SEX:	AGE:	TYPE OF PARTIAL:	TOOTH #:	ABUTMENT OR CONTROL:	CROWN:	MARGIN PLACEMENT:
1	2	58	A-1	5	C	1	1
				8	B	5	4
				14	C	1	1
2	2	62	B-1	21	C	2	3
				24	B	5	4
				29	C	2	3
3	1	67	B-1	21	C	3	2
				24	B	5	4
				28	C	3	2
4	1	49	B-1	22	C	1	1
				24	B	5	4
				27	C	1	1
5	1	49	A-1	5	C	1	1
				6	B	5	4
				11	C	1	3
6	1	66	B-1	21	C	3	1
				23	B	5	4
				27	C	3	1
7	1	66	A-1	6	C	1	1
				8	B	5	4
				11	C	1	1
8	2	63	B-1	22	C	2	3
				24	B	5	4
				27	C	1	1
9	1	68	A-2	8	B	5	4
				10	C	1	1
				14	C	2	3
10	1	62	B-2	20	C	1	3
				23	B	5	4
				28	C	1	1

NO.	GCF LEVEL: vol.µl M.	D.	LÖE-SILNESS INDEX: M.	D.	POCKET DEPTH: M.	D.	OPPOSING DENTITION:	LENGTH OF SPAN: R.	L.	ANT.
1	0.13	0.115	2	2	2	2	1	3	1	0
	0.05	0.035	1	1	3	2				
	0.08	0.135	3	3	2	7				
2	0.03	0.07	1	2	1	2	2	3	4	0
	0.03	0.05	1	1	1	1				
	0.035	0.09	1	2	1	2				
3	0.14	0.15	2	3	3	3	4	3	3	0
	0.07	0.08	1	1	2	2				
	0.1	0.085	2	3	3	4				
4	0.2	0.14	3	2	6	3	1	3	3	0
	0.075	0.07	2	2	2	3				
	0.085	0.075	2	2	3	2				
5	0.05	0.13	1	1	2	3	1	2	2	2
	0.04	0.06	1	1	1	1				
	0.075	0.045	1	1	3	2				
6	0.07	0.105	1	1	2	3	2	4	3	0
	0.03	0.055	1	1	3	2				
	0.11	0.075	2	1	4	2				
7	0.04	0.015	0	0	1	2	3	3	4	2
	0.02	0.015	0	0	1	2				
	0.04	0.02	0	1	1	2				
8	0.04	0.055	1	1	1	2	1	4	4	0
	0.015	0.02	0	0	1	1				
	0.02	0.04	0	1	1	2				
9	0.025	0.02	1	2	3	3	4	4	0	3
	0.1	0.045	2	1	2	3				
	0.065	0.14	2	1	2	3				
10	0.03	0.035	1	2	3	2	4	3	1	0
	0.035	0.03	1	1	2	2				
	0.1	0.1	2	1	3	2				

NO.	SEX:	AGE:	TYPE OF PARTIAL	TOOTH #:	ABUTMENT OR CONTROL:	CROWN:	MARGIN PLACEMENT:
11	1	65	A-2	6	C	1	2
				9	B	5	4
				11	C	1	3
12	1	75	A-2	6	C	2	1
				9	B	5	4
				15	C	2	1
13	2	68	B-1	21	A	5	4
				22	B	5	4
				29	C	1	3
14	1	65	B-1	21	C	2	3
				24	B	5	4
				28	A	5	4
15	2	66	B-1	21	A	5	4
				24	B	5	4
				28	C	3	3
16	1	75	B-1	20	A	5	4
				23	B	5	4
				29	C	2	3
17	2	48	A-2	8	A	5	4
				9	B	5	4
				11	C	3	3
18	1	57	B-2	22	C	1	1
				24	B	5	4
				28	A	5	4
19	2	66	A-2	6	C	3	1
				11	B	5	4
				12	A	5	4
20	2	57	A-3	5	A	5	4
				9	B	5	4
				11	C	1	1

NO.	GCF LEVEL: vol.µl		LÖE-SILNESS INDEX:		POCKET DEPTH:		OPPOSING DENTITION:	LENGTH OF SPAN:		
	M.	D.	M.	D.	M.	D.		R.	L.	ANT.
11	0.05	0.07	0	1	2	2	1	4	1	0
	0.035	0.04	0	0	1	1				
	0.035	0.035	0	1	1	2				
12	0.105	0.095	2	3	3	2	1	4	0	0
	0.055	0.055	1	2	1	2				
	0.09	0.05	3	2	3	2				
13	0.15	0.115	1	1	2	1	1	2	3	0
	0.06	0.025	0	0	2	1				
	0.04	0.065	0	1	2	3				
14	0.105	0.055	0	0	1	1	1	2	2	0
	0.05	0.045	0	0	1	1				
	0.03	0.02	1	1	2	2				
15	0.05	0.105	1	1	2	2	1	3	3	0
	0.06	0.07	1	1	2	1				
	0.09	0.13	2	2	2	3				
16	0.025	0.03	0	1	2	1	1	2	2	0
	0.035	0.04	0	0	1	1				
	0.075	0.05	1	2	2	2				
17	0.035	0.055	0	0	1	2	1	5	1	1
	0.02	0.05	0	1	2	1				
	0.05	0.09	1	1	3	3				
18	0.125	0.16	2	2	1	2	2	2	2	0
	0.08	0.07	0	0	1	1				
	0.175	0.145	1	2	2	2				
19	0.08	0.06	0	1	1	2	1	4	2	2
	0.05	0.03	0	0	1	1				
	0.03	0.06	1	1	2	2				
20	0.045	0.05	1	1	3	2	3	0	3	3
	0.035	0.04	1	1	2	2				
	0.09	0.08	2	2	3	2				

NO.	SEX:	AGE:	TYPE OF PARTIAL:	TOOTH #:	ABUTMENT OR CONTROL:	CROWN:	MARGIN PLACEMENT:
21	1	43	A-3	6	A	5	4
				8	B	5	4
				12	C	1	3
22	2	71	A-3	4	C	2	3
				6	B	5	4
				11	A	5	4
23	1	59	A-3	3	C	3	1
				9	B	5	4
				11	A	5	4
24	1	42	B-3	20	C	2	1
				23	B	5	4
				28	A	5	4
25	1	62	B-4	21	A	5	4
				23	B	5	4
				29	C	2	1
26	1	62	A-4	4	C	2	3
				6	B	5	4
				13	A	5	4
27	1	47	A-4	4	B	5	4
				6	C	2	1
				11	A	5	4
28	2	55	A-4	2	C	2	3
				5	B	5	4
				12	A	5	4
29	1	55	B-4	24	B	5	4
				27	A	5	4
				30	C	2	1
30	2	71	B-4	20	A	5	4
				26	B	5	4
				27	C	3	3

NO.	GCF LEVEL: vol.µl M.	D.	LÖE-SILNESS INDEX: M.	D.	POCKET DEPTH M.	D.	OPPOSING DENTITION:	LENGTH OF SPAN: R.	L.	ANT.
21	0.08	0.085	2	1	3	2	1	2	1	0
	0.045	0.11	1	1	2	2				
	0.1	0.085	2	2	4	3				
22	0.095	0.14	1	2	2	3	1	1	4	1
	0.05	0.055	0	1	1	2				
	0.045	0.015	1	1	2	1				
23	0.09	0.095	1	1	4	3	4	0	3	0
	0.01	0.005	0	0	1	1				
	0.06	0.04	1	0	2	2				
24	0.095	0.055	1	1	2	2	2	1	2	0
	0.015	0.03	0	1	2	2				
	0.015	0.01	0	0	1	1				
25	0.075	0.07	2	1	3	1	4	1	2	0
	0.105	0.035	1	0	1	2				
	0.135	0.16	1	1	1	2				
26	0.065	0.215	0	2	2	3	1	1	1	5
	0.065	0.065	2	2	2	2				
	0.09	0.15	2	2	1	2				
27	0.055	0.02	1	1	2	2	3	0	0	4
	0.115	0.05	1	1	2	3				
	0.04	0.045	1	1	2	1				
28	0.095	0.09	2	3	2	2	4	1	0	6
	0.065	0.07	1	1	3	2				
	0.05	0.085	1	2	1	3				
29	0.02	0.025	0	0	1	1	4	2	3	0
	0.035	0.08	1	1	1	2				
	0.07	0.06	1	1	2	2				
30	0.05	0.075	1	2	2	2	2	3	1	5
	0.03	0.075	1	1	1	1				
	0.105	0.14	2	3	3	5				

Table 2: Fixed Prosthodontic Categories

Category	Sex	Age ($\bar{x}$)	Material Used for Crown	GCF Vol.(µl) M. ($\bar{x}$)	GCF Vol.(µl) D. ($\bar{x}$)	Löe-Sillness Index ($\bar{x}$)
B = Non restored (Control)	Fe = 70% M = 30%	46.4	Enamel = 33% PFM = 54.4% CVGC = 6.7% Acrylic to metal= 5.6%	0.048	0.058	1.389
C = Restored bridge abudment						
D = Restored single tooth						

Pocket Depth mm. M. ($\bar{x}$)	Pocket Depth mm. D. ($\bar{x}$)
2.656	2.689

Table 3: Removable Prosthodontic Categories

Category	Sex Fe = 63.3% M = 36.7%	Age ($\bar{x}$)	Type of Partial Denture	Opposing Dentition
A = Non restored abutment		60.63	Bilat. distal ext.= 38.9% Unilat ext. Right = 24.4% Unilat ext. Left = 16.7% Tooth supported = 20.0%	Partial Denture = 50.0% Denture = 16.7% Natural dentition = 10.0% Nat. dent. plus fixed rest. = 23.3%
B = Control				
C = Restored abutment				

Total Length of Edentulous Span ($\bar{x}$)	Arch	GCF Vol. (µl.) Mesial ($\bar{x}$)	Distal ($\bar{x}$)
5.5 teeth	Max. = 50% Mand.= 50%	0.065	0.070

Löe-Silness Index Mesial ($\bar{x}$)	Distal ($\bar{x}$)	Combined ($\bar{x}$)	Pocket Depth -- mm. Mesial ($\bar{x}$)	Distal ($\bar{x}$)
1.011	1.200	1.105	1.967	2.067

Table 4: Fixed Prosthodontic Categories

Category	Material used for Crown	GCF Vol. (µl). Mesial	Distal	Löe-Silness Index	Pocket Depth mm. Mesial	Distal
B Non restored (Control)	(Enamel = 100%)	0.030	0.036	0.933	2.400	2.433
C Restored bridge abutment	PFM = 76.7% CVGC = 6.7% Acrylic to metal = 16.7%	0.061	0.085	1.633	2.733	2.733
D Restored single tooth	PFM = 86.7% CVGC = 13.3%	0.052	0.055	1.600	2.833	2.900

Table 5: Removable Prosthodontic Categories

Category	Material used for Crown	GCF Vol, (µl). Mesial	Distal	Löe-Silness Index Mesial	Distal	Combined	Pocket Depth mm. Mesial	Distal
A Non restored	(Enamel = 100%)	0.064	0.071	1.200	1.267	1.500	2.033	2.100
B Non restored single tooth (control)	(Enamel = 100%)	0.044	0.046	0.633	0.767	0.800	1.633	1.600
C Restored partial denture abutment	PFM = 36.7% CVGC = 40.0% Acrylic to metal = 23.3% Margin Placement Sub Ging. = 50.0% At crest = 6.7% Supra Ging = 43.3%	0.087	0.093	1.200	1.567	1.600	2.233	2.500

Table 6: Summary of Mean Values for all Comparison Groups

COMPARISON GROUPS	GINGIVAL CREVICULAR FLUID VOL.(µl.) (Mesial)	(Distal)	LÖE-SILNESS INDEX	POCKET DEPTH (mm.) (Mesial)	(Distal)
FIXED PROSTHODONTICS:					
Non Restored Tooth (Control)	0.030	0.036	0.933	2.400	2.433
vs.					
Restored Single Tooth	0.052	0.055	1.600	2.833	2.900
vs.					
Restored Bridge Abutment	0.061	0.085	1.633	2.733	2.733
REMOVABLE PROSTHODONTICS:					
Non Restored Tooth (Control)	0.044	0.046	0.800	1.633	1.600
vs.					
Non Restored Abutment	0.064	0.071	1.500	2.033	2.100
vs.					
Restored Partial Denture Abutment	0.087	0.093	1.600	2.233	2.500
FIXED PROSTHODONTICS:					
Restored Single Tooth	0.052	0.055	1.600	2.833	2.900
vs.					
REMOVABLE PROSTHODONTICS:					
Restored Partial Denture Abutment	0.087	0.093	1.600	2.233	2.500
FIXED PROSTHODONTICS:					
Restored Bridge Abutment	0.061	0.085	1.633	2.733	2.733
vs.					
REMOVABLE PROSTHODONTICS:					
Restored Partial Denture Abutment	0.087	0.093	1.600	2.233	2.500

Table 7: t - test Analysis

Group 1: single restored unit
Group 2: restored abutment under existing partial denture

Variable	Number of Cases	Mean	S. D.	T value	Degrees of Freedom	
M#VOL MESIAL FLUID VOLUME						
Group 1	30	0.0518	0.032	3.85	58	Statistically significant at p<.001
Group 2	30	0.0868	0.038			
D#VOL DISTAL FLUID VOLUME						
Group 1	30	0.0548	0.033	3.74	58	Statistically significant at p<.001
Group 2	30	0.0935	0.046			
VOLUME TOTAL FLUID VOLUME IN MICROLITERS						
Group 1	30	0.0533	0.030	4.29	58	Statistically significant at p<.001
Group 2	30	0.0902	0.036			
LOE#INDEX LOE-SILNESS INDEX LEVEL-COMBINED						
Group 1	30	1.6000	0.498	0.0	58	
Group 2	30	1.6000	0.855			
M#DEPTH MESIAL - POCKET DEPTH						
Group 1	30	2.8333	0.986	2.22	58	
Group 2	30	2.2333	1.104			
D#DEPTH DISTAL - POCKET DEPTH						
Group 1	30	2.9000	1.269	1.50	58	
Group 2	30	2.5000	0.731			

Table 8: t - test Analysis

Group 1: restored bridge abutment (Fixed Prosthodontics)
Group 2: restored abutment under existing partial denture

Variable	Number of Cases	Mean	S. D.	T value	Degrees of Freedom
M#VOL MESIAL FLUID VOLUME					
Group 1	30	0.0613	0.026	3.03	58
Group 2	30	0.0868	0.038		
D#VOL DISTAL FLUID VOLUME					
Group 1	30	0.0850	0.072	0.54	58
Group 2	30	0.0936	0.046		
VOLUME TOTAL FLUID VOLUME IN MICROLITERS					
Group 1	30	0.0732	0.045	1.61	58
Group 2	30	0.0902	0.036		
LOE#INDEX LÖE-SILNESS INDEX LEVEL - COMBINED					
Group 1	30	1.6333	0.669	0.17	58
Group 2	30	1.6000	0.855		
M#DEPTH MESIAL - POCKET DEPTH					
Group 1	30	2.7333	1.048	1.80	58
Group 2	30	2.2333	1.104		
D#DEPTH DISTAL - POCKET DEPTH					
Group 1	30	2.7333	1.143	0.94	58
Group 2	30	2.5000	0.731		

Table 9: Regression Analysis

MESIAL GCF VOLUME						
Material used for crown						
R Square 0.1647	Analysis of Variance	DF	Sum of Squares	Mean Square	F	
	Regression	2	0.02049	0.01024	8.576	Statistically significant at p<.001
	Residual	87	0.10391	0.00119		
Margin Placement						
R Square 0.21271	Analysis of Variance	DF	Sum of Squares	Mean Square	F	
	Regression	3	0.02646	0.00882	7.745	Statistically significant at p<.001
	Residual	86	0.09794	0.00114		
DISTAL GCF VOLUME						
Material used for crown						
R Square 0.18398	Analysis of Variance	DF	Sum of Squares	Mean Square	F	
	Regression	2	0.02863	0.01431	9.807	Statistically Significant at p<.001
	Residual	87	0.12697	0.00146		
Margin Placement						
R Square 0.18398	Analysis of Variance	DF	Sum of Squares	Mean Square	F	
	Regression	3	0.02863	0.00954	6.463	Statistically significant at p<.001
	Residual	86	0.12697	0.00148		
LÖE-SILNESS INDEX						
Material used for crown						
R Square 0.16654	Analysis of Variance	DF	Sum of Squares	Mean Square	F	
	Regression	2	10.47536	5.23768	8.692	Statistically significant at p<.001
	Residual	87	52.42464	0.60258		
Margin Placement						
R Square 0.17065	Analysis of Variance	DF	Sum of Squares	Mean Square	F	
	Regression	3	10.73417	3.57806	5.898	Statistically significant at p<.001
	Residual	86	52.16583	0.60658		

CHAPTER V

DISCUSSION

The fragile, cellular microcosm of the dentoepithelium and the gingival sulcus function to maintain periodontal integrity. With the advent of high speed instrumentation, even the most highly skilled operator employing innocuous and delicate techniques of soft tissue manipulation encounters the inherent problem of tissue trauma. An inflammatory response of the marginal gingiva may occur subsequent to the intrinsic irritation provided by full coverage restorations and the various levels of gingival termination. Another detrimental aspect of restorative procedures is the adaptation of the full coverage restoration to the preparation. The cement interface between termination of the restorative material and natural tooth structure provides a nidus for plaque accumulation.

The response to inherent tissue trauma and bacterial by products has been inflammation of the soft tissues. Gingival inflammation is the manifestation of an attempt on the part of the host to dilute, isolate and remove, and render benign substances deemed foreign. They may be of microbial origin or due to some noxious stimuli. A correlation between an increase in gingival crevicular fluid flow and an increase in inflammation has been observed; and crevicular fluid as postulated by Brill, 1960, can be quantitatively measured and thereby serve as an indicator of the level of inflammation present that is far more consistent

than subjective clinical indices that have been utilized historically.

Inflammatory changes have been observed by investigators on a histological basis. Alterred microcirculation and increased inflammatory cell density will ultimately lead to increased vascular permeability that permits an egress of fluid from the vascular compartment (Grant, Stein, and Everett, 1972) into the gingival sulcus. Presence of increased gingival crevicular fluid flow in the sulcus will reflect the level of periodontal inflammation prior to clinical manifestation (Loe and Holm-Pedersen, 1965) of pathology. Subclinical inflammation can thus be monitored and the inflammatory process interrupted to limit the amount of irreversible tissue damage.

The controversy over whether gingival crevicular fluid flow can be demonstrated in clinically healthy gingiva has lead to the theory that crevicular fluid is a reflection of the host's response to noxious or exogenous stimuli and his capacity to release mediators of inflammation. The host's response to foreign invasion will vary with the individual and may be affected by age, genetic capacity for response and predisposition, and the general health of the individual.

Investigators have discussed the relative value of abutments for both fixed and removable prosthodontics. Cohn (1956) stated "a common cause of periodontal disease is the faulty removable partial denture, or fixed partial denture, in which anchorages are improperly selected or incorrectly fabricated." Waerhaug (1968) in emphasizing that margin termination should be supragingival to minimize the contributory effect

of plaque retention on crown margins toward periodontal disease stated "many patients would be better off if bridges or partial dentures were not constructed."

This investigation was performed in an attempt to lend statistical credence to the inflammation levels of restored abutment teeth for both fixed and removable prosthesis. A determination and comparison of the inflammatory changes in the marginal gingiva of abutment teeth for fixed and removable prosthesis was performed. Data was collected from thirty individuals (mean age = 46.4) that exhibited a restored bridge abutment, a restored single unit crown and at least one non restored tooth (control). Thirty additional individuals (mean age = 60.63) served as candidates for the collection of data from at least one restored abutment under an existing partial denture and a non restored tooth (control).

The gingival crevicular fluid volume mean, Löe-Silness Index, and pocket depth mean were determined and the lowest values were correlated with the fixed prosthodontic non restored tooth (control) category. These values tended to increase with the single restored tooth category and were the highest with the restored abutment for a bridge category.

Further increases in the aforementioned parameters occurred in the control group for the removable prosthesis group, increased with the non restored abutment, and were the highest for the restored abutment under an existing partial denture.

A statistically significant increase is observed in gingival

crevicular fluid volume associated with a restored abutment under a removable prosthesis when compared with a single restored crown. In part, this can be attributed to the additional plaque accumulation and associated bacterial by products related to the components and materials used in the fabrication of the partial denture. This increase may also reflect the compromised dentitions (periodontally involved) that necessitate partial denture prostheses. In addition, the statistics obtained may actually be a reflection of the alterred host response to a foreign material, in this case, a removable prosthesis. It may well be that an alterred host response in the form of an autoimmune response that is the body's response to a prosthesis deemed "foreign" or not recognized as "self" is reflected in the data. Further studies to varify the presence of C 3 and IgG in human gingival crevicular fluid would be indicated. (See Figure 8: Pathway of Complement activation, page 35).

The fact that no statistically significant increase in gingival crevicular fluid volumes could be measured between a restored bridge abutment and a restored partial denture abutment appears to be explained by the fact that the restored bridge abutment gingival crevicular fluid values increased to meet the increased gingival crevicular fluid values of the restored partial denture abutment. This increased gingival crevicular fluid volume associated with a restored bridge abutment may well reflect the margin termination, material utilized to construct the prosthesis, the contour of the bridge abutment, and the existence of inadequate embrasure that would have facilitated the maintenance of the prosthesis by the patient.

From the regression analysis of the variables of partial denture prosthesis, the factors that contributed the most to the increased level of gingival crevicular fluid volume were margin placement and type of restorative material employed.

The findings in this study correlate with those of Brill and Björn, (1959) who observed increased gingival crevicular fluid volume with full coverage restorations as compared to non restored teeth: Maruyama and associates, (1976) who observed dilation of capillary loops related to inflammation in gingiva classified as clinically normal; and Mahajan, (1976) whose histologic observation of the interdental papilla adjacent to full coverage restorations indicated that both normal and inflamed gingiva adjacent to restored teeth exhibited an inflammatory cell infiltrate and dilated blood vessels.

CHAPTER VI

SUMMARY AND CONCLUSIONS

The dynamic aspects of the dento-gingival junction and sulcus were presented, and inherent problems in restorative dentistry that contribute to soft tissue inflammation were discussed. Components of gingival crevicular fluid and their possible implications in relation to periodontal pathology have been correlated with a response on the part of the host to exogenous or noxious stimuli.

A determination and comparison of the inflammatory changes in the marginal gingiva of abutment teeth for fixed and removable prostheses as well as for non restored teeth was performed. Data was collected from thirty individuals (mean age = 46.4) that exhibited a restored bridge abutment, a restored single unit crown, and at least one non restored tooth (control). Thirty additional individuals (mean age = 60.63) served as candidates for the collection of data from at least one restored abutment under an existing partial denture and a non restored tooth (control). The gingival crevicular fluid volume, Löe-Silness index, and pocket depth were recorded; data was subjected to a t-test analysis between the two populations, and a regression analysis was performed on the variables recorded for the partial denture prostheses.

The following conclusions can be made in view of the results obtained during this study:

1. In a comparison of the marginal gingiva inflammatory changes associated with restored teeth versus the non restored teeth; for both the Fixed Prosthodontic categories and the Removable Prosthodontic categories, the least amount of inflammation as measured by the parameters of gingival crevicular fluid volume, Löe-Silness index and pocket depth in all cases, were associated with the non restored groups.
2. In a comparison of the marginal gingiva inflammatory changes of the single restored tooth from the Fixed Prosthodontic population versus the restored tooth under an existing partial denture from the Removable Prosthodontic population; the least amount of inflammation as measured by the gingival crevicular fluid volume was associated with the single restored tooth from the Fixed Prosthodontic population.
3. In a comparison of the marginal gingiva inflammatory changes of the restored bridge abutments (Fixed Prosthodontics) to the restored abutments under a partial denture (Removable Prosthodontics); the least amount of inflammation as measured by the gingival crevicular fluid volume was associated with the restored bridge abutment.
4. Löe-Silness index and pocket depth determination may not be as valid a determinant of inflammatory levels of soft tissue as is determination of the gingival crevicular fluid volume.
5. Teeth with subgingival margin placement from both populations were accompanied by predictable inflammatory levels.

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APPENDIX

Patient Data Sheet (Fixed Prosthodontics)

DATE:

PATIENT NAME:	AGE:	SEX:	TOOTH #:	BRIDGE ABMT: SINGLE RESTORED T; CONTROL:	REST. MATERIAL:	GCF VOL: M. D.	LOE SILNESS INDEX:	POCKET DEPTH: M. D.

Patient Data Sheet (Removable Prosthodontics)

DATE:

PATIENT CHART NUMBER: PHONE: AGE:

PARTIAL: Maxillary TYPE: Tooth supported--Bilat. Distal Ext.--Unilateral Dist. Ext. R
Unilateral Dist. Ext. L
Mandibular

TOOTH #	RESTORATION TYPE OR CONTROL TOOTH	MARGIN PLACEMENT	GCF READING (M) (D)	LOE SILNESS INDEX (M) (D)	POCKET DEPTH (M) (D)

DENTITION OF OPPOSING ARCH: Natural Dentition -- Denture -- Partial Denture--
Natural Dentition plus Fixed Prosthesis

LENGTH OF EDENTULOUS SPAN:

SCHEMATIC DIAGRAM OF PROSTHESIS:

APPROVAL SHEET

The thesis submitted by Pamela Denise Golasz, D.D.S., has been read and approved by the following committee:

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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 by the Committee with reference to content and form.

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

6/25/80
Date

William F. Malone
Director's Signature