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Determination of the Gingival Inflammatory Levels Associated with Abutment Teeth Used for Fixed Prosthodontics

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DETERMINATION OF THE GINGIVAL INFLAMMATORY
LEVELS ASSOCIATED WITH ABUTMENT TEETH
USED FOR FIXED PROSTHODONTICS

by

Thomas E. Yuhas, D.D.S.
Loyola University, 1980

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

June

1980

DEDICATION

To my parents for allowing me to pursue an education
for which they had no opportunity.

To my best friend, Marge.

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A note of gratitude is extended to Harco Electronics for their assistance during the investigation.

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VITA

The author, Thomas E. Yuhas, is the son of Emery and Marjorie Yuhas, Jr. He was born April 25, 1953 in Peoria, Illinois.

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In May, 1980, he received his specialty certificate in Prosthodontics.

TABLE OF CONTENTS

	Page
DEDICATION	ii
ACKNOWLEDGEMENT	iii
VITA	iv
LIST OF TABLES	v
LIST OF ILLUSTRATIONS	vi
CONTENTS OF APPENDIX	vii
INTRODUCTION	1
REVIEW OF THE LITERATURE	4
METHODS AND MATERIALS	18
EXPERIMENTAL RESULTS	27
DISCUSSION	32
SUMMARY AND CONCLUSION	37
BIBLIOGRAPHY	39
APPENDIX	44

LIST OF TABLES

Table	Page
1. Compiled Data	29
2. Analysis of Variance Table	30
3. Unadjusted and Adjusted Deviation Table	31

LIST OF ILLUSTRATIONS

Figure	Page
1. Periotron	21
2. Filter Paper Strips	22
3. Filter Paper Strip	23
4. Filter Paper In Sulcus	24
5. Filter Paper In Sensors Of Periotron	25
6. Periodontal Probe In Gingival Sulcus	26

CONTENTS OF APPENDIX

	Page
PATIENT CONSENT FORM	44
STATISTICAL ANALYSIS	
MESIAL ASPECT	45
DISTAL ASPECT	46
COMPILED RAW DATA	47

CHAPTER I

INTRODUCTION

Inflammation and gingival recession are frequently observed around teeth with artificial crowns. Adverse gingival responses appear to be further modified if the artificial crowns are retainers on abutment teeth for a fixed prosthesis.

The irritation of crown margins in the gingival sulcus is commonly accompanied by bacterial plaque accumulation which is necessary to initiate the inflammation (Waerhaug, 1956). Subgingival margins associated with complete coverage restorations apparently alters the gingival environment and probably contributes to the inflammatory process (Karlson, 1970; Jameson, 1979).

The dentoepithelial junction and gingival sulcus area function, in part, as defense systems. Gingival inflammation also represents a defensive response against micro-organisms and noxious stimuli entering the sulcus. An increase in inflammation is accompanied by a rise in crevicular fluid volume from the gingival sulcus. This rise in crevicular fluid levels is usually sponsored by increased vascular compartments. Hence, crevicular fluid

measurements provide a quantitative means of monitoring the inflammation of the sulcular area.

Fortunately, crevicular fluid enters the gingival sulcus and reflects the degree of sulcular inflammation before clinical signs are evident. In addition, the identification of known lysosomal enzymes (Cimasoni, 1974) in gingival crevicular fluid has suggested a significant role of these enzymes in the pathogenesis of periodontal disease possibly by component reactions of the immune system.

Evaluation of gingival tissue responses to restorative dentistry have previously been reviewed using color, texture and pocket depth of the soft tissue as criteria. Various indices are employed which require the dentist to subjectively assign numerical values to these varying inflammatory stages and pocket depths (Loe, 1967). Monitoring crevicular fluid would provide a more objective, predictive method of measuring gingival responses. Until recently, it was difficult to measure or gauge this gingival crevicular fluid quantitatively. Now, by means of a crevicular fluid monitor developed by Harco Electronics* which electronically measures minute fluid volume, this can be accomplished in a standardized manner.

However, controversy still exists concerning the optimal method of evaluating the condition of abutment teeth and surrounding soft tissue. The effects of increased load and function upon the adjacent gingiva, epithelial attachment and bone are not clear. Evaluation of abutment teeth with crevicular fluid recordings has not been previously performed.

The purpose of this study was to compare the degree of gingival inflammation of teeth with single complete crowns and fixed bridge abutments to similar non-restored teeth within the same oral cavity. Inflammation would be monitored by measuring crevicular fluid volume using the Harco crevicular fluid meter.

CHAPTER II

REVIEW OF THE LITERATURE

Controversy concerning the existence of gingival crevicular fluid as a normal physiologic filtration product or an inflammatory exudate still persists. Nevertheless, there is consensus agreement an inflammatory exudate is present in the unhealthy gingival sulcus. There has been description of cells or groups of cells responsible for a fluid that demonstrated a remarkable healing capacity of the tissue surrounding teeth.

Perhaps a more comprehensive view of this gingival fluid can be forwarded by an historical description of concepts held by former researchers.

In G.V. Black's (1887) histological description of the periodontal membrane, he referred to a very peculiar system of cells resembling those of the lymphatics which clustered about the principal fibers of the membrane. These cells, which he called salivary corpuscles, were "augmented" with every irritation of the membrane. He noted these cells were first affected in salivation with mercury. Black (1899) described loops of glands running lengthwise to the root and anastomosing freely and ceasing

in a rather thick mass before reaching the gingival border. He concluded from this study these glands were easily disturbed by certain drugs such as mercury and iodine and they were often disturbed by poisonous substances floating in the bloodstream. He felt "phagedenic pericementitis" had its beginning in these glands.

Bodecker (1933), in his investigation of dental erosion and its possible causes, concluded the gingival fluid was acid in dentitions suffering from traumatic occlusion. He found there was more gingival fluid from the crevices around teeth with erosion.

Waerhaug (1953) noted after injecting India ink into gingival pockets, that within one hour transudation of fluid and the emigration of leucocytes took up the grains of ink; and after three days all the ink had disappeared. Waerhaug differed from Gottlieb's theory of the presence of an organic connection between the epithelium and tooth surface. He felt this was an histologic concept and that it did not coincide with the clinical observations. Waerhaug agreed with G.V. Black's concept, the gingiva was closely fitted around the tooth and preferred the term epithelial cuff to epithelial attachment. He felt it was possible for the gingiva to defend itself against injury

such as a temporary ingress of bacteria on a matrix band.

Waerhaug (1952) concluded the flow of gingival fluid in the pocket was in a coronal direction, and the gingival fluid originated from the blood serum. In healthy gingival tissues the flow of gingival fluid was very slight. His investigation showed pockets without calculus or deposits were as a rule, sterile. He also felt it possible to obtain bacteria free pockets around artificial crowns where no bacterial retention possibilities existed.

Hagerman (1955) described the connective tissue fibers of the free gingiva as a "dynamic, living, responsive tissue functioning as a protective cover for the underlying periodontium".

Jensen and Zanders (1958) believed the effects of cementing material on the gingival tissue showed the poor marginal seal of a restoration contributed to gingival irritation. With poor adaptation there developed plaque and bacterial accumulation which caused gingival irritation.

Brill and Bjorn (1959) administered fluorescein orally to humans and by collection on filter paper strips were able to detect it in the gingival sulcular fluid. They found the quantity of fluorescein recovered on the strips increased with an increase in the severity of gingival

inflammation. Also, patients with extensive dental restorations showed more fluorescein in the gingival sulcus than clinically healthy gingiva adjacent to non-restored teeth. Their study showed the fluid in the gingival pocket originated from interstitial fluid and increased with gingival irritation and disease.

Brill (1959) injected dogs with Evan's Blue, a dye that binds to plasma proteins; and concluded plasma proteins passed through capillary walls and entered the gingival sulcus in increased amounts as a result of gingival irritation and inflammation and mechanical stimulation.

In a separate study, Brill and Krasse (1959) again working with dogs, showed brief mechanical stimulation of clinically healthy pockets provoked and increased flow of tissue fluid through pocket epithelium.

Brill (1959) demonstrated chewing paraffin produced an increase in gingival tissue flow.

In a study involving dogs, Brill (1959) concluded gingival tissue fluid washed away particulate matter and bacteria from pockets.

Brill (1960) performing an immuno-electrophoretic study of tissue fluid from gingival pockets found the fluid which

flows through the epithelial cuff into human gingival pockets contains at least seven different proteins corresponding to those of normal human serum. He, therefore, believed the source of this gingival fluid was tissue fluid. Ordinarily the fluid was confined within the body by epithelium and drains away as lymph. This study indicated the gingival fluid drained through the epithelial cuff into the gingival pockets. This observation supported the contention gingival fluid: 1) arose from the bloodstream, 2) passed through connective tissue, and 3) entered the gingival pockets through the epithelium.

Krasse (1960) demonstrated marked flow of gingival fluid from gingival pockets with marginal gingivitis. This investigation was performed on dogs, in which the consistency of the diet was also altered. He noted a harder consistency produced an increase in the gingival flow.

Waerhaug (1960) demonstrated the remarkable healing capacity of the epithelial attachments. He noted there was a certain leakage of fluid constantly going on. He viewed this as a defense mechanism. Again he emphasized the epithelial attachment was actually an epithelial cuff that

envelops the tooth by the circular collagenous fibers and the blood pressure within the capillaries. He stated emphatically "the normal gingival pocket is only a potential pocket". "It opened up when instruments were inserted, but it closed again when they were taken out." In the histologic section the potential pocket was not experienced as an open space.

Orban (1960) refuted Waerhaug's conception of the lack of attachment. He preferred to use the term attached epithelial cuff.

Loe (1961) in describing the physiological aspects of the gingival pocket, believed there was a continuous transudation of tissue fluid into the clinically normal gingival pocket. This was in agreement with the observation made by Brill. Loe also agreed it originated from blood. No glands could be demonstrated in the area so the fluid cannot be a product of secretion. He noted mitotic figures along the entire length of the non-keratinized epithelial lining of the pocket and the desquamation of the surface cells supported the view that the epithelial cuff was constantly renewed.

Krasse and Egelberg (1962) analyzed the Na/K ratio of human gingival fluid and from their results concluded

the fluid in the gingival crevice was an inflammatory exudate.

Egelberg (1963) in this study applied histamine (an inflammatory mediator) to the gingiva of humans and dogs and found there was an increased permeability and the flow of fluid in the sulcus increased.

Egelberg (1963) concluded fluid from healthy pockets could be considered an inflammatory exudate and not a simple filtration product.

Mann (1963) collected gingival fluid from human subjects on filter paper strips and concluded severity of gingival inflammation was the main factor determining the rate of fluid flow.

Loe and Holm-Pederson (1965) concluded: 1) healthy gingival crevices did not exhibit fluid flow, 2) crevicular fluid began prior to the appearance of any clinical changes, and 3) monitoring fluid flow provided the clinician with a more sensitive technique for detecting early gingivitis than waiting for clinical signs to develop.

Nagao (1967) studied the influence of prosthetic appliances upon the flow of crevicular tissue fluid and concluded the insertion of crowns increased crevicular

fluid flow and poor fitting crowns resulted in more crevicular fluid than crowns that fit well.

Listgarten (1967) demonstrated the oral epithelium had the ability to reconstitute an attachment apparatus after gingival surgery similar to the one in the normal state. There were hemidesmosomes and basement lamina and cuticular structure present in the newly formed epithelial attachment. To achieve this, no reduced enamel epithelium was needed.

Gavin (1968) studying the ultrastructure of the crevicular epithelium of cat gingiva concluded there existed a peculiar permeability of the gingival crevice region of the oral mucosa to tissue fluid and leucocytes. He found the intercellular spaces were wide and extended as a continuous network from the basal lamina to the epithelial surface. One important difference of this epithelium in comparison with other epithelium was the continuity of its intercellular spaces with the oral cavity and the presence within it of large numbers of leucocytes. He theorized the wide intercellular spaces could be a route for the inward passage of materials through the epithelium.

Sandalli and Wade (1969) concluded scaling, polishing and supervised oral hygiene reduced gingival fluid flow.

In addition, they also found there was a considerable reduction in gingival fluid flow four weeks after periodontal surgery, at which time the crevicular epithelium has had a chance to become re-established.

Schwartz (1971) studied gingival reaction to different types of tooth accumulated materials and found the most severe reaction occurred with plaque on calculus, followed by bacterial plaque.

Alfano (1974) performed an interesting and detailed study on the origin of gingival fluid. He contended plaque and bacterial by-products establish an osmotic gradient which initiated gingival exudation. This was a pre-inflammatory exudate which could progress from osmotically-modulated exudate to a secondary inflammatory exudate. A change in composition occurred with this. Some of Alfano's conclusions were (and these were in agreement with other workers), "1) gingival fluid may be recovered from clinically and histologically healthy tissues, 2) the intercellular spaces in the sulcular epithelium dilated during inflammatory exudation, 3) gingival fluid flow increased several days prior to clinical inflammation, 4) the Na/K ratio of the pre-inflammatory gingival fluid was comparable to an inflammatory exudate but the protein composition

of this fluid was lower than an inflammatory exudate, 5) gingival inflammatory exudation was associated with interruption in the basement membrane, 6) the protein/Ca ratio increased in the gingival fluid from increasingly inflamed tissues, and 7) the flow of gingival fluid increased concurrently with the accumulation of dental plaque."

Cimasoni (1974) concluded his monograph with the following statements: "measuring the intensity of gingival fluid flow was probably the most reliable and sensitive procedure for a quantitative evaluation of gingival inflammation". "The measurements should, however, always be made using standardized techniques."

Waerhaug (1978) noted the junctional epithelium becomes readapted to the tooth surface in areas that had been previously covered with subgingival calculus and plaque.

Demetris (1978) found a similar composition between gingival and mixed and parotid salivary IGA content. The presence of immunoglobulins in the crevicular fluid tended to support the contention the fluid played a role in a defense mechanism.

In more clinically related literature, in regard to crown restoration and gingival reactions, we found Wheeler (1931) expressed a strong awareness of the function of

the cervical tissue and the importance of proper management of this area in complete crown restorations. He recognized the cervical margin was the most difficult and perhaps the most important part of crown preparation. He recommended leaving 1mm of enamel above the normal attachment if possible.

Marcum (1967) found the margins of crown restorations that were even with the gingival crest produced the least inflammatory response from the gingival tissues. Margins that were located above and below caused the most inflammation.

Klecinic (1968) stressed the need to eliminate all irritants to the gingiva, to restore tooth form deformities, the proper patient care and hygiene in preparation of restorative dentistry for periodontally comprised dentitions.

Perel (1971) in studying the periodontal considerations of crown contours, concluded defective contours are not desirable. Defective contours resulted in lack of stimulation of the gingiva along with food and plaque accumulation. This resulted in an inflammation of the gingiva.

Skurow (1971) stressed the importance of minimizing the irritation to gingival tissues.

Berman (1973) concluded the success or failure of

complete crown restorations was determined by how well a clinician handled the soft tissues. He noted two basic requirements for successful tissue retraction. One, the tissue should be normal, and two, it should be capable of repair. He described a beveled shoulder preparation technique which provided the proper space for a gold collar marginal termination without infringing on the soft tissues. He concluded generally normal gingiva creeps over and covers the thin walled gold collars of complete crowns. Whereas, abnormal tissue fails to respond to preparation, retraction and impression making. When the abnormal tissue heals, recession and exposure of terminal margins of the preparation results.

Trivedi (1973) concluded most gingival inflammation adjacent to restorative materials was possibly due to imperceptible roughness, debris, defective margins or chemical injury.

Richter (1973) concluded from his study involving margin placement the fit and finish of full crown restorations was more important than whether the margin was supra or sub-gingival.

Nemetz (1974) emphasized the need to condition the

soft tissues prior to final preparations. He stressed the importance of proper instrumentation and proper tissue retraction.

Borden (1974) measured human crevicular fluid flow using the Harco fluid meter to compare extracrevicular and intracrevicular collection techniques. Their results showed: 1) repeated intracrevicular measurements gave similar gingival fluid flow rates, 2) the three seconds intracrevicular measurement of crevicular fluid flow was more sensitive than the extracrevicular technique, 3) repeated intracrevicular measurements did not significantly stimulate fluid flow, 4) the recommended technique for measuring crevicular fluid with GCF meter involves initially emptying the crevicular pool with a paper strip followed by a thirty seconds interval and three seconds placement of a fresh filter paper strip for measurement, and 5) measuring gingival crevicular fluid flow was an objective method for monitoring gingival inflammation. They stated the intracrevicular measurement was a more satisfactory technique.

Volchansky (1974) using a scanning electron microscope studied surface roughness of enamel, cementum, amalgam, gold inlay, porcelain fused to gold and calculus. The conclusion was, "enamel is probably the smoothest and most

acceptable surface in the mouth, and all natural and restored surfaces should be compared to it".

Jameson (1976) in comparing the crevicular fluid volumes of restored and non-restored teeth, found a statistically significant difference between them. He concluded the placement of subgingival margins resulted in a predictable degree of inflammation. This degree of inflammation was considered indigenous to complete crown restorations placed subgingivally.

Sweetnam (1977) reported no significant difference between semi-precious metal and Type III gold in complete gold crowns on posterior teeth. All teeth crowned with subgingival margins regardless of metal type showed a pronounced increase in crevicular fluid volume when compared to the control teeth.

Garvin (1979) studied the effect of self-curing acrylic treatment restorations (temporization) on the gingival sulcus area after complete crown preparation by measuring the gingival crevicular fluid volume.

CHAPTER III

METHODS AND MATERIALS

Thirty patients were randomly selected who met the following criteria:

- 1) a tooth which was a full cast retainer for a bridge abutment,
- 2) a tooth which was restored with a full cast crown, and
- 3) a non-restored tooth.

The patients ranged from 28 to 65 years in age with a mean age of 46.4. Twenty-one of the thirty patients were female.

The involved teeth were isolated with cotton rolls and gently dried with a warm air syringe. A crevicular fluid reading was taken and recorded from the mesial-buccal and distal-buccal of the observed teeth.

The gingival sulcus was emptied first by using a sterile filter paper strip (Periopaper - Harco) and inserted into the gingival crevice with a cotton forceps. This strip was left in for a period of three seconds to empty the crevicular fluid pool. A second filter strip was inserted into the same area and allowed to remain for a period of twenty-seven seconds. This second filter paper strip was then

placed between the sensors of the Periotron instrument. The filter paper was inserted a uniform distance into the sensors by inserting it to the line on each filter strip. A digital readout on the face of the instrument would indicate a quantitative measurement of the fluid collected. This number, when divided by 200, would show the amount of fluid collected in micro-liters. After obtaining both mesial and distal readings on each tooth, the gingival pocket depth was measured and recorded. Lastly, a subjective gingival index determination was made using the Loe and Silness gingival index.

Loe and Silness
Criteria for the Gingival Index System

- 0 = Absence of inflammation
- 1 = Mild inflammation - slight color change, little texture change
- 2 = Moderate inflammation - moderate redness, glazing, edema, bleeding upon probing
- 3 = Severe inflammation - marked redness and hypertrophy, ulceration, spontaneous bleeding

All the readings were performed by the same investigator. The order of sequence of procedures was followed throughout the investigation.

The recorded data was compiled into a single table

and mean values for all measurements were computed. The gingival crevicular fluid volumes were subjected to analysis of variance to determine if a statistically significant difference existed between the non-restored, restored and abutment teeth.



FIGURE 1

Periotron

(Clinical GCF Meter - Harco Electronics LTD, Winnipeg, Canada)

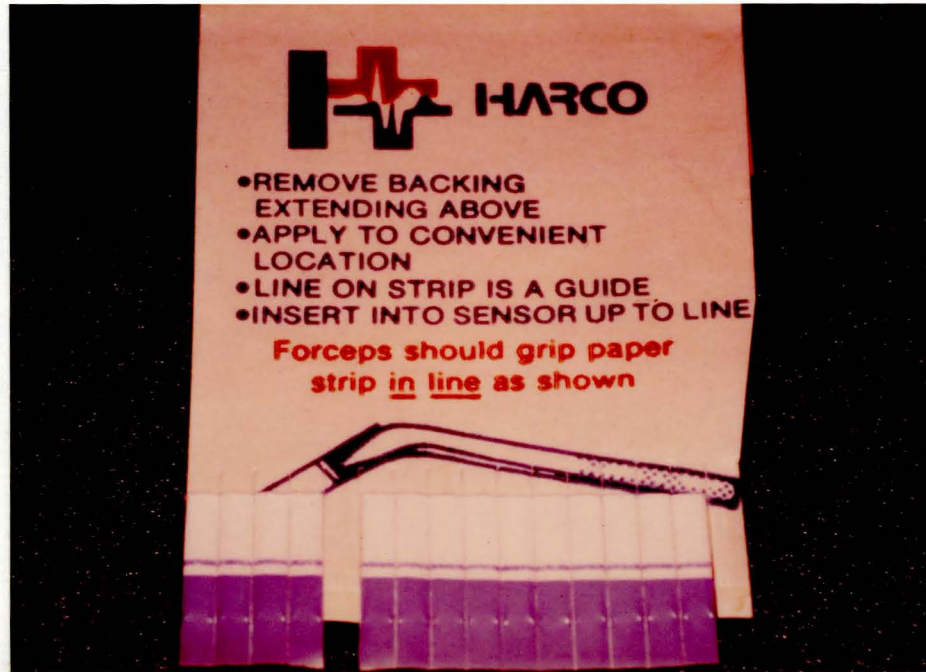


FIGURE 2

Sterile Filter Paper Strips
(Periopaper - Harco Electronics LTD, Winnipeg, Canada)

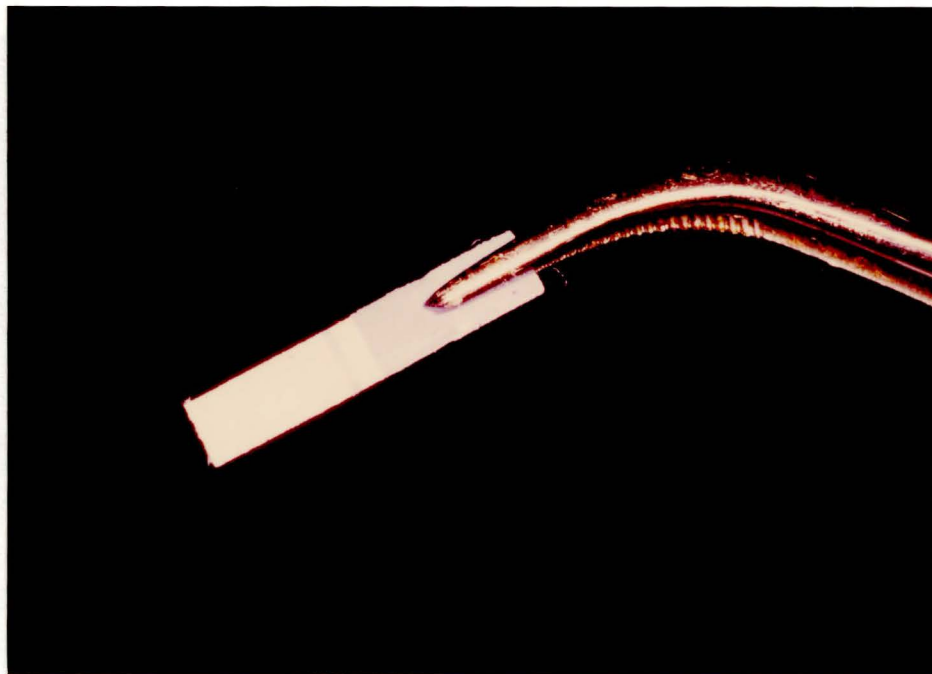


FIGURE 3

Filter Paper Strip Prior to Collection of Crevicular Fluid



FIGURE 4
Gingival Fluid Collection Technique From a Patient Before Preparation

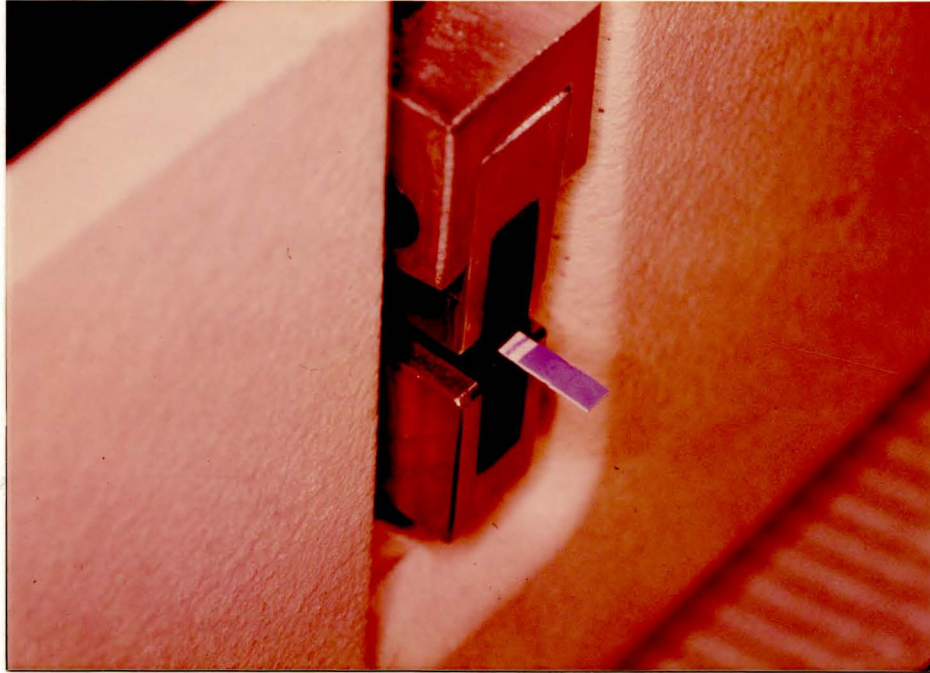


FIGURE 5
Placing Filter Paper Between Recording Sensors



FIGURE 6
Measuring Sulcus Depth
(After Collecting GCF)

CHAPTER IV

EXPERIMENTAL RESULTS

Table I contains the compiled data from a section of the total raw data collected. Listed are the patient number, sex, age, teeth involved in the collection, restorative material and recorded values for crevicular fluid volume, pocket depth (mm) and gingival index. The amount of microliters of fluid collected is determined by dividing the instrument reading by 200. This was incorporated into the computer program.

Table II is the analysis of variance table for the crevicular fluid volumes of the mesial amount, distal amount, and the averaged fluid volume collected per tooth.

In all three analysis it has been shown that there is a significant difference between the three types of teeth observed as to the amount of crevicular fluid present. ($.05 > P > .01$) This statistical method does not show if there is a significant difference between the restored and abutment tooth.

Table III shows the unadjusted and adjusted deviation from the grand mean for mesial volume, distal volume and averaged volume for all three types of teeth observed.

This analysis shows us that there is a significant difference between the non-restored and the other two types observed. It also shows that the abutment tooth had the most variability when compared to the other types.

TABLE I

Patient No.	Patient:	Sex	DOB	Age
	Address:			
	Phone:			

Tooth#	NR	Mat'l.	M-Vol	D-Vol	M-Depth	D-Depth	Index
	R						
	AB						

1)
2)
3)

Gingival Index Code:

- 0) Normal
- 1) Mild Inflammation - no bleeding with probe
- 2) Moderate Inflammation - bleeding with probe
- 3) Severe Inflammation - spontaneous bleeding

NR = Non-Restored R = Restored AB = Abutment

Material: Porcelain to Metal (P)
Acrylic to Gold (A)
Enamel (E)
Gold (G)

M-Vol & D-Vol = Mesial and Distal Volumes as read out on Periotron.

M-Depth & D-Depth = Mesial and Distal Pocket Depth in millimeters.

Gingival Index = Gingival Index Code Number.

μ = Microliters of fluid collected - average volume of mesial plus distal.

TABLE II

ANALYSIS OF VARIANCE
TYPE WITH VOLUME (AVG)

Source of Variation	SS	DF	MS	F	P
Volume	12.520	1	12.520		
Type	3.567	2	1.783	5.234	$P < .01$
Residual	29.302	86	0.341		

UNADJUSTED AND ADJUSTED DEVIATION
FOR TYPE WITH VOLUME

Grand Mean = 1.39

Type	Unadjusted Deviation	Adjusted for Independents and Covariates Deviation
1) Non-Restored	-0.46	-0.29
2) Restored	0.21	0.21
3) Abutment	0.24	0.08

CHAPTER V

DISCUSSION

PART I

The crevicular fluid of the gingival sulcus reflects the host's response against microorganisms and noxious stimuli. The fluid is reportedly (Rudin et al 1970) increased proportionately to the intensity of the inflammatory process present. The general purpose of this investigation was to compare the inflammation of the soft tissue surrounding teeth restored with single, complete crowns and abutments for fixed prosthodontics. This was accomplished by measuring the crevicular fluid of the gingival sulcus using a periotron by "Harco". A more specific intent was to determine if the increased load to abutment teeth substantially altered the crevicular fluid flow. Thirty patients who had a fixed prosthesis, complete crown and a similar unrestored tooth were subjected to crevicular fluid determinations, Loe-Silness index assessment and periodontal pocket depth recording. The data were reviewed statistically using an analysis of variance or independent chi square.

PART II

This study supported the study of Jameson (1977) and

Garvin (1979) that demonstrated the majority of teeth recorded consistently higher crevicular fluid levels. This evidence of increased inflammation is present in all teeth supporting complete coverage restorations regardless of the type of restorative material used.

Some investigators contend there is no Harco Electronics, Winnipeg, Canada.

Crevicular fluid flows in clinically healthy marginal gingiva, but in this study there was always a measureable quantity of fluid in the randomized sample of patients selected. The advantage of monitoring the degree of inflammation is avoiding further tissue deterioration and developing into a degenerative chronic stage. A quantitative increase of fluid seen in the tissue prior to noticeable visible increase in inflammation as demonstrated by Loe and Holm-Pedersen (1965) was seemingly reaffirmed.

PART III

The limitations of this study was the actual collection of data. Consistency in the execution of recording the data is a mandate. Although the instrument is believed to be an accurate, reliable recording of ul of fluid, the response of various patients to the inflammation indigenous to com-

plete, subgingival crowns is variable and somewhat unpredictable.

A redeeming feature of this study is the comparison of bridges, crowns and a control within the same oral cavity. This experimental design should lend credence to the results obtained.

PART IV

The clinical significance of notable increase in restored teeth compared to the controls as the placement of subgingival, complete crowns results in a certain predictable degree of inflammation. This is regardless of the ability of the operator and the diligence of laboratory implementation. There is a comparative reduced level of crevicular fluid where margins are polished and smooth and the general profile of the supporting tissue is healthy prior to tooth preparation. Caution by the dentist during preparation, tissue retraction and treatment restoration placement is necessary. Lack of attention to detail can precipitate an adverse tissue response. Patients who demonstrated the presence of a higher level of crevicular fluid in the sulcus of the control tooth, consistently recorded an increase in crevicular fluid around the complete crown and fixed bridge

abutments. This is compatible with Brill-Bjorn (1959) results.

This inflammation could be attributed to a lesser disciplined oral hygiene program and/or a predetermined innate level of tissue reaction to given patient reaction. Recommendations to reduce this level of response would include:

- 1) detailed comprehensive diagnosis,
- 2) satisfactory tooth preparation which will allow tissue compatible tooth contours in the final restoration,
- 3) fewer complete, subgingival crowns whenever possible,
- 4) supra-gingival termination of crown for patients with periodontally compromised dentitions ... pain permitting,
- 5) increased frequency of recall appraisals by the dentist to review the restorative procedures.

PART V

Future research could explore the value of comparing the qualitative to quantitative measurements of the crevicular fluid. Auto immune responses are verified by the presence of certain immunoglobulins, i.e., IGg, ICM and/or C₃ etc. The presence or absence of these immunoglobulins during the five phases of the auto immune compliment response

might possibly elucidate the role of restorative procedures which commonly follow extensive periodontal surgery. Future investigation should include studies as to the relation between the span of the bridge, the age of the prosthesis, the restorative material, and the medical liability of the patient.

CHAPTER VI

SUMMARY AND CONCLUSIONS

The crevicular fluid volume between nonrestored, single unit and abutment teeth was compared on 30 patients between the ages of 28 to 65. There was a significant difference ($P < .05$) in crevicular fluid volume between the three types of teeth observed. The abutment teeth showed the most variability as to fluid volume. The Periotron^R was used to quantitatively measure the amount of crevicular fluid within the gingival sulcus.

The dynamic nature and delicate balance of the dento-gingival junction and sulcus areas was discussed in relation to gingival inflammation, full coverage restorative procedures, and presence of gingival crevicular fluid. The chemical constituents of gingival crevicular fluid and their possible implications as factors in periodontal pathology were presented. The necessity for further studies associated with gingival crevicular fluid were mentioned.

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

- 1) Subclinical inflammatory changes associated with full coverage restorations can be objectively and quantitatively measured using standardized methods and a gingival crevicular fluid meter.

2) Teeth being used as abutments for a fixed prosthesis show a greater inclination towards subclinical inflammatory change than a nonrestored or a single unit crown. This fact may be attributed to the increased load placed on the abutment teeth and/or the closed contact areas which make plaque more difficult to be removed by the patient.

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APPENDIX

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 periodontal treatment.

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

You are free at any time to ask questions about this project and the associated procedures. If during the procedure you wish to withdraw your participation in this study, you may do so without prejudice.

I HAVE READ THE ABOVE INFORMATION AND WILL PARTICIPATE IN THIS STUDY.

PARTICIPANT'S SIGNATURE _____

DATE _____

WITNESS SIGNATURE _____

DATE _____

ANALYSIS OF VARIANCE
TYPE WITH MESIAL VOLUME

Source of Variation	SS	DF	MS	F	P
Volume	11.731	1	11.731		
Type	3.128	2	1.564	4.405	P < .05
Residual	30.529	86	0.355		

Unadjusted and Adjusted Deviation for Type with Mesial Volume

Grand Mean = 1.39

Type	Unadjusted Deviation	Adjusted for Independents and Covariates Deviation
1) Non-Restored	-0.46	-0.29
2) Restored	0.21	0.17
3) Abutment	0.24	0.11

ANALYSIS OF VARIANCE
TYPE WITH DISTAL VOLUME

Source of Variation	SS	DF	MS	F	P
Volume	9.823	1	9.823		
Type	4.964	2	2.482	6.976	$P < .01$
Residual	30.601	86	0.356		

Unadjusted and Adjusted Deviation for Type with Distal Volume

Grand Mean = 1.39

Type	Unadjusted Deviation	Adjusted for Independents and Covariates Deviation
1) Non-Restored	-0.46	-0.34
2) Restored	0.21	0.23
3) Abutment	0.24	0.11

PATIENT # & DESCRIPTION	TOOTH#	MAT'L.	VOLUME		DEPTH		INDEX
			M	D	M	D	
#1 Male 52	12 NR	E	10	12	3	3	1
	13 R	P	15	9	3	4	1
	27 AB	P	13	11	3	3	2
#2 Female 56	11 NR	E	11	10	3	4	2
	12 R	P	8	5	4	3	2
	10 AB	P	13	8	3	3	2
#3 Female 37	5 NR	E	6	3	3	2	1
	10 R	P	10	14	2	3	2
	11 AB	P	9	11	3	3	2
#4 Female 34	10 NR	E	4	2	2	2	0
	9 R	P	8	6	2	2	1
	12 AB	A	12	6	3	2	1
#5 Female 37	6 NR	E	5	2	2	2	0
	7 R	P	9	7	3	2	2
	9 AB	P	4	11	2	2	1
#6 Male 49	27 NR	E	5	5	1	2	1
	28 R	P	5	15	2	1	1
	9 AB	P	8	5	2	2	1
#7 Female 61	7 NR	E	3	2	2	2	0
	13 R	P	14	9	3	3	2
	4 AB	A	13	16	3	6	2
#8 Male 59	5 NR	E	3	8	3	4	1
	8 R	P	4	2	1	1	1
	10 AB	P	8	11	3	2	2
#9 Male 48	10 NR	E	6	7	3	3	2
	19 R	G	13	10	6	6	2
	13 AB	A					

PATIENT # & DESCRIPTION	TOOTH#	MAT'L.	VOLUME		DEPTH		INDEX
			M	D	M	D	
#10 Female 48	8 NR	E	9	4	3	2	1
	6 R	P	9	12	3	3	2
	10 AB	P	12	10	2	2	1
#11 Female 38	6 NR	E	2	6	3	2	2
	10 R	P	8	2	3	3	2
	11 AB	P	5	5	1	1	2
#12 Female 56	9 NR	E	5	4	1	2	0
	10 R	P	6	11	2	2	2
	12 AB	P	8	8	2	2	1
#13 Female 44	8 NR	E	3	3	2	2	0
	2 R	G	4	4	3	3	2
	20 AB	G	24	55	4	2	3
#14 Female 45	3 NR	E	9	15	3	3	2
	11 R	P	11	16	3	3	1
	6 AB	P	18	68	3	3	2
#15 Male 55	11 NR	E	5	5	2	1	1
	10 R	P	7	15	3	1	1
	6 AB	P	14	16	2	4	1
#16 Female 28	6 NR	E	6	13	4	4	1
	8 R	P	17	18	3	3	2
	29 AB	P	23	19	3	2	2
#17 Female 34	4 NR	E	8	16	3	3	1
	9 R	P	7	7	4	5	2
	6 AB	P	13	12	3	5	3
#18 Female 34	14 NR	E	6	4	2	1	1
	3 R	P	8	5	4	2	1
	11 AB	P	8	11	1	2	1

PATIENT # & DESCRIPTION	TOOTH#	MAT'L.	VOLUME		DEPTH		INDEX
			M	D	M	D	
#19 Female 52	7 NR	E	1	6	2	2	1
	30 R	G	34	30	3	5	2
	4 AB	A	14	46	5	3	2
#20 Female 45	11 NR	E	20	17	2	3	2
	9 R	P	22	23	2	2	2
	8 AB	P	22	24	2	2	2
#21 Female 41	10 NR	E	3	5	2	3	1
	19 R	P	7	3	3	2	1
	12 AB	P	10	8	2	2	0
#22 Female 37	9 NR	E	4	7	2	1	0
	10 R	P	8	9	2	3	1
	11 AB	P	9	12	2	2	1
#23 Male 54	6 NR	E	6	5	1	3	0
	7 R	P	7	7	2	2	1
	3 AB	P	6	9	2	2	1
#24 Female 34	8 NR	E	4	5	2	2	0
	3 R	P	6	8	3	5	2
	29 AB	P	9	13	3	3	1
#25 Female 51	7 NR	E	5	14	3	3	2
	10 R	P	21	16	4	3	2
	4 AB	P	12	16	3	2	1
#26 Female 58	11 NR	E	5	3	2	1	1
	9 R	P	5	4	1	2	1
	28 AB	G	13	10	2	2	2
#27 Male 61	27 NR	E	10	11	5	4	2
	23 R	P	14	16	2	3	1
	29 AB	A	13	18	4	4	2

PATIENT # & DESCRIPTION	TOOTH#	MAT'L.	VOLUME		DEPTH		INDEX
			M	D	M	D	
#28 Female 47	27 NR	E	11	7	2	3	1
	6 R	P	7	11	3	5	2
	9 AB	P	14	22	3	3	2
#29 Male 32	10 NR	E	2	2	2	2	0
	19 R	G	6	14	3	3	2
	8 AB	P	11	19	2	2	2
#30 Male 65	8 NR	E	5	11	2	2	1
	10 R	P	11	21	3	2	2
	3 AB	P	22	20	6	4	2

APPROVAL SHEET

The thesis submitted by Thomas E. Yuhas has been read and approved by:

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

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

6/25/80

Date

William F. Malone

Signature of Advisor