1984

The Quality of Stainless Steel Crowns Related to Crevicular Fluid Flow

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THE QUALITY OF STAINLESS STEEL CROWNS RELATED TO CREVICULAR FLUID FLOW

by

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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

1984

ACKNOWLEDGMENTS

The author wishes to acknowledge the members of the Advisory Committee: Dr. Patrick Toto, Dr. Sandrick, and Dr. Wayne Milos.

A special recognition to my parents for unlimited encouragement and support through my education.

VITA

The author, Renee Balthazar, is the daughter of Dorothea Shoger Balthazar and Dr. Eugene Regis Balthazar. She attended primary and secondary schools in Aurora, Illinois. In 1971, she graduated from Michigan State University with a Bachelor of Arts degree.

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CHAPTER I

INTRODUCTION AND STATEMENT OF PURPOSE

During the past decade, the use of various preformed stainless steel crowns to restore primary teeth has grown in popularity. Much literature recommends their use. They offer many advantages for conserving deciduous teeth which have extensively involved caries and for restoring them when they exhibit anomalies of enamel and dentin. They sometimes serve as a source of anchorage for space maintainers or offer complete coverage after pulpal therapy. However, errors in the placement of stainless steel crowns are frequently observed and periodontal problems appear to be associated with defective crowns (Goto, 1970; Henderson, 1973; Myers, 1975). Clinical observation suggests that the response of the gingival tissue to a stainless steel crown is influenced by proper contour and adaptation (Goto, 1970; Henderson, 1973; Myers, 1975).

Thus, the detrimental effects of SSC on the periodontium, specifically the marginal gingiva, have been observed clinically. However, no quantitative measurement has been made available to assess the degree of change. A gingival crevicular fluid meter has been developed, the Periotron, which electronically measures minute fluid volume by reduction in capacitance between two sensors when in contact with a standardized filter strip containing fluid.

It is know that crevicular fluid volume increases with an

increase in inflammation (Brill, 1962; Mann, 1963; Egelberg, 1964; Oliver, Holm-Pedersen, and Loe, 1969; Spranger, 1980). Therefore the purpose of this investigation is to determine the crevicular fluid volume utilizing a crevicular fluid meter and to assess changes in the gingiva after insertion of stainless steel crowns. Evaluation will be made to see if a correlation exists between stainless steel crowns, their quality, and the amount of crevicular fluid response. In addition, the frequency in errors in placement of stainless steel crowns and the role each plays in any accompanying gingivitis will be studied.

Some authors suggest that periodontal problems which occur in adults have their origin in the early years of adolescence (Baer, 1974; Ciamsoni, 1974). Thus, the prevention and treatment of periodontal disease in early stages might reduce its occurrence in adulthood. If we can promote good restorative treatment, which is compatible with a healthy gingiva in childhood, further periodontal complications may be prevented in future years.

CHAPTER II

REVIEW OF LITERATURE

The presence of fluid in the gingival crevice has been known for many years (Serres, 1817; Black, 1920). Miller (1938) defined the term crevicular exudate as a discharge from the gingival crevice and listed changes in the exudate as a clinical sign of incipient periodontal disease. He stated frequently that even without clinical signs of periodontal disease microscopic evaluation of crevicular fluid reveals the presence of an unusual number of "pus cells."

However, serious study of crevicular fluid composition and flow, particularly in response to gingival and periodontal disease, begin with the studies of Waerhaug (1952) and Brill (1958).

Waerhaug (1952) placed India ink in gingival sulcus of young dogs and observed, within an hour, an emigration of leukocytes through the sulcular epithelium concomitantly with an increase flow of fluid. Most of the ink was removed by crevicular fluid after two hours. He concluded that saliva could not penetrate below the gingival margin and in healthy pockets, where a normal epithelial attachment is closely adapted around the tooth to the gingival margin, the secretion is rather minute. However, there was a "constant flow of cellular elements and tissue fluid from all pockets."

Brill (1959) studied the flushing action of gingival pocket fluid in dogs by introducing suspensions of charcoal particles and

bacteria into healthy gingival crevices. Six to twenty minutes later both kinds of material could be found on filter strip papers placed intracrevicularly. He concluded that fluid flowing out of gingival pockets can remove particles and bacteria and should be described as a defense mechanism.

Brill and Krause (1960) injected fluorescein intravenously into dogs and humans whose gingivae were clinically healthy. It was recovered thirty seconds later on filter paper strips inserted into the gingival crevice, but not from other intact epithelial surfaces. Thus, it was shown that fluid containing small molecules of fluorescein passes from subepithelial spaces, through the epithelium of clinically healthy or moderately inflamed pockets, and enters the oral cavity by the gingival crevice. "These findings seem to indicate a functional difference between epithelium lining gingival pockets and oral epithelium. The fluid which can be recovered from gingival pockets might be called gingival pocket fluid."

Brill (1959) performed another study to determine the source of gingival fluid. He injected Evans Blue, a dye which binds to plasma protein, into young dogs. Small amounts of blue colored material were recovered via filter strip papers. He injected another dog with Evans Blue and histamine and, after toothbrushing, the gingival samples stained bright blue. Moreover, upon heavy mechanical stimulation of gingival margins, "a heavy outpouring of blue labeled material" into the gingival pockets of dogs resulted. He concluded that the original source of gingival pocket fluid to be the blood stream. When capillary walls were stimulated chemically by histamine and mechanically by

toothbrushing, permeability increased with an outflow of a protein rich fluid. In addition, it showed that epithelium lining gingival pockets, besides being transversed by small molecules like fluorescein, are also transversed by large, complex protein molecules. It was also found that gingival pocket fluid yielded a positive ninhydrin reaction indicating the presence of amino acids.

Brill and Bronnestam (1960) analyzed the crevicular fluids for plasma proteins by immunoelectrophoresis and demonstrated seven different proteins originating from plasma including gamma globulin, transferin and albumin. The author speculated on the importance of gamma globulin as a source of protective antibodies.

Brandtzaig and Mann (1961) studied lysozyme activity of human gingival pocket fluid, serum and saliva. There was no correlation between lysozyme activity of the three fluids. However, there was a high lysozymal activity with increased periodontal inflammation which was not seen in serum and saliva. It was hypothesized that lysozyme within the gingival fluid is from leukocytes. Lysozyme can lyse gram-negative bacteria releasing endotoxin and enzymes, such as hyaluronidase, chonodrotin sulfatase, and collagenase.

Löe (1961) studied the components of gingival fluid by sealing forty-nine clinically healthy gingival pockets in three dogs. He examined them histologically after varying intervals. The pockets contained desquamated epithelial cells, neutrophils and an amorphous substance thought to be remnants of the gingival tissue. The author noted that the presence of neutrophils in the region from the CEJ to the gingival margin tended to show that the passage of fluid occurs

along the entire sulcular epithelium. Also, neutrophils were seen within the crevicular epithelium indicating cells migrate from connective tissue into the sulcus. Gusaffson and Nilsson (1961) studied the fibrinolytic activity of gingival fluid. After collecting pocket fluid from healthy gingiva, they found that it contained plasmin, plasminogen, and fibrinogen. These components are an essential part of inflammatory reactions capable of lysing fibrin.

Krause and Egelberg (1962) studied the relative concentration on Na^{+}, K^{+}, and Ca^{++} in gingival pocket fluid from clincally healthy and inflamed gingiva. The results indicated that the Na/K ratio of the fluid in both cases was lower than that of plasma, showing that intracellular fluid changed as it left the tissue to enter the sulcus. Therefore, it was concluded "that gingival pocket fluid can not be regarded as a simple filtration product, but rather an inflammatory exudate."

Mann (1963) collected 307 samples of crevicular fluid via filter strip paper from subjects who were given fluorescein sodium by mouth. The condition of the marginal gingiva was assessed and the depth of the pocket was measured. He showed that inflammation has a higher correlation to the amount of fluid which flows into the crevice than does pocket depth. "Inflammation is the main factor contributing to flow. There is a minimal flow in the healthy state and an increased flow in conditions of inflammation and trauma."

Egelberg (1964) studied the amount of exudate that can be obtained from clinically healthy gingiva via filter strip papers stained by ninhydrin. He also tested for variations in fluid flow

from different regions on the tooth. He showed that small amounts of gingival exudate were obtained from healthy pockets and that the larger amount was present in the mesial papilla area as compared to the buccal or palatal marginal gingiva. He further showed a positive correlation between the amount of gingival exudate and clinical gingival inflammation. In addition, he found a positive histologic correlation between the amount of exudate and the degree of inflammatory cell infiltration of the gingiva of dogs. Egelberg stated that there was much evidence to indicate that the gingival exudate as measured with filter paper strip is a good indicator of the degree of inflammation of the gingiva. Such measurements are in fact measures of a fundamental reaction in inflammation--the increase of vascular permeability. Thus, there is a good basis for the introduction of such measurements for determining degrees of inflammation. With this method at hand, an increased possibilities to study the effect of various factors on inflammation of the gingiva have probably been achieved.

Loe and Holm-Pedersen (1965) investigated whether fluid was present in normal, non-inflamed gingiva compared to inflamed gingiva. After clinically assessing via Loe Gingival Index system, the gingiva fluid was collected extracrevicularly (placing filter strip next to the tooth surface, the gingival margin, and the attached gingiva), and intracrevicularly (filter paper strip placed at the entrance of the orifice). By the biomicroscopic technique, they observed that inserting filter paper into sulcus resulted in capillary compression and trauma. They concluded that crevices of healthy gingiva produce no flow even after stimulation. However, inflamed gingiva showed that the

presence of fluid and its amount varied according to its severity. Gingiva that at the start of the experiment did not exhibit flow did so as bacterial activity increased and shortly before clinically observable gingivitis. They suggested that 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.

Hara and Loe (1969), using filter strip papers, tested for the presence and amounts of carbohydrates, free glucose, hexosamine, and glucuronic acid in exudate. Samples were taken from individuals with mild to severe degrees of gingival inflammation. The amount of gingival exudate increased with the severity of inflammation. The study described the presence and quantity of free glucose, glucuronic acid and hexosamine in the gingival exudate. It was theorized that carbohydrates represent breakdown products of the inflamed tissue, but that both their absolute and relative amount may be influenced by the oral microflora.

Orban and Stallard (1969) compared the amount of crevicular fluid flow with clinical scoring techniques, such as Ramfjord's Periodontal Index, Green and Vermillion's oral hygiene index, and biopsy specimens scored on a scale from zero to ten depending on the extent of inflammatory infiltrate. They found that crevicular fluid scores <u>did not</u> correlate with the biopsy score while correlation <u>did</u> exist between plaque scores and biopsy scores. Therefore, they concluded: "a better indication of the inflammatory status of the gingival tissue, as revealed by biopsies, is the evaluation or measurement of dental

plaque."

Oliver, Holm-Pedersen, and Loe (1969) evaluated the relationship between a gingival index (Loe-Silness, 1963), gingival exudate measurements, and histological inflammatory cell density. There was a close relationship between the Gingival Index scores and exudate measurements by ninhydrin staining. When inflammation increases clinically, there was a corresponding increase in the flow of exudate and histological inflammatory infiltrate. When there was no clinical evidence of gingival inflammation, there was no exudate and a minimum of inflammatory cells.

Rudin (1970) measured the sulcular fluid by the ninhydrin staining technique. The severity of marginal inflammation was scored and biopsies of the marginal gingiva taken. Clinical inflammation and sulcus fluid, clinical and histological signs of inflammation, sulcus fluid and round cell infiltration all were positively correlated. The study showed that assessment of sulcular fluid flow rate permits quantitation of the severity of marginal gingivitis.

Wilson and McHugh (1971) study did not agree with the claims of Loe et al. (1965) that there is no gingival exudate, if there is no gingivitis, via the modified-intracrevicular method or orifice method. Of 202 gingival areas which were graded clinically as free from gingivitis, exudate was collected from all but one. The gingival exudate correlated well with Gingival Index (Loe-Silness, 1963) for individual gingival surface areas.

Paunio (1971) determined the presence of hydroxyproline containing components in gingival fluid. The fluid was collected with

filter paper strip from more than 100 individuals and analyzed via gel permeation, chromatography and dialysis. The results supported findings of Hara and Loe (1969) concerning occurrence of hexosamines and uronic acid. However, they feel hydroxyproline in the gingival exudate arose from blood serum leaking into the crevice, from collagen degraded by enzymes originating from the gingival tissue itself, or from plaque itself. This can be considered a sign of collagen metabolism and/or of plaque enzyme action on the connective tissue in the gingiva.

A study done at the University of Minnesota (1972) stated that the composition of gingival fluid varies with the degree of inflammation of the gingival source. The fluid from clinically healthy gingiva can be compared with that from inflamed crevices thusly:

	Healthy Gingiva	Inflamed Crevices
fluid flow rate	low	increased
serum protein	low	higher
[urea]	higher than serum	lower
ph	higher than saliva	lower
# or PMN's	low	high
[electrolytes]	higher than serum	lower

Shillitoe and Lehner (1972) measured the levels of IgG, IgA, IgM, and C^3 in crevicular fluid and serum and IgA in saliva on twenty-five patients with periodontal disease. High concentrations of IgG, IgA, IgM, and C^3 were found in the crevicular fluid although the levels were below those in serum. The serum crevicular fluid ratio

were consistent with the hypothesis, that in the gingival crevice, complement-dependent immune reactions may occur between microbial antigens and antibodies favoring the IgG, so that the ratios for IgG and C^3 are similar.

Alfano (1974) presented a theory concerning the origin of gingival fluid. He thought that it was unclear whether the fluid resulted from physiological or pathological processes. Controversy existed whether the fluid resembles a physiological transudate due to its protein concentrations while to others it appeared as an inflammatory exudate substantiated by its Na/K ratio. He believed that gingival fluid may arise by two distinct mechanisms: (1) a standing osmotic gradient, and (2) initiation of inflammation. The gradient is generated by macromolecular by-products of the bacteria which reside in subgingival plaque. These by-products diffuse through the crevicular epithelium and accumulate in the sulcus. Thus, there is an increase in solute concentration and an osmotic gradient established. These solvent molecules which are drawn across the membrane by this gradient, will raise intercellular hydrostatic pressure and cause the exudation of gingival fluid. This may occur in healthy gingiva. However, if plaque is not removed, macromolecular by-products will eventually penetrate the basement membrane with enzymatic, toxic and antigenic properties initiating an inflammatory exudate. Thus, gingival fluid may progress at different times in various areas of the mouth, from an osmotic exudate to a secondary inflammatory exudate with different composition.

In 1974, Cimasoni presented the following composition of

crevicular fluid in his review of it.

Cellular Elements:

1) Epithelial cells

Leukocytes

Bacteria

2) Electrolytes

Na, K, and other ions Ca^{++}, Cl^{-}, Mg^{++}

3) Organic Compounds

Carbohydrates

Proteins: α, β, a_2, a_1- globulins, albumin

4) Metabolic and Bacterial products

1) Lactic Acid

2) Urea

3) Hydroxyproline

4) Endotoxins

5) Cytolytic factors

6) Antibacterial enzymes

Enzymes

1) acid phosphatase; 2) alkaline phosphatase,

3) B-Glucuronidase; 4) lysozyme; 5) cathepsin D;

6) Proteases; 7) Lactic Dehydrogenase

Egelberg and Attström (1974) compared the orifice method (filter strip paper gently placed in the orifice of the crevices) or the intracrevicular method (inserted as deep as possible in the crevice) for measuring gingival fluid. They measured crevicular fluid from dental students, starting with healthy gingiva and compared the

amount collected from such students who refrained from cleaning their teeth for four days. By each of the methods used, differences in fluid amounts were obtained after one day, indicating gingival fluid measurement as a sensitive technique. "Both techniques seemed to be comparable for evaluation of intra-individual changes of gingival inflammation."

Borden, Golub and Klinberg (1974) compared the two techniques of collecting fluid intracrevicularly and extracrevicularly and their correlation with Löe's gingival index and pocket depth were compared. Gingival crevicular flow was measured by the Harco gingival crevicular flow meter. They concluded that repeated intracrevicular measurements gave similar flow values and that the three second intracrevicular measurement was more sensitive than the extracrevicular method. They recommended, when using the Harco G C F meter initially to empty the crevicular pool with a paper strip and thirty seconds later place a fresh strip intracrevicularly for three seconds. Collecting fluid extracrevicularly was unsatisfactory for slightly inflamed gingiva crevices since the fluid has difficulty flowing out of such crevices.

Shern, Von Mohr, and Joly (1974) compared the ninhydrin staining method and the crevicular fluid flow meter (Harco) for quantifying crevicular fluid flow. They found "precision, accuracy and reliability of measuring crevicular flow greater when using a flow meter than using the ninhydrin dye method." They concluded that using a combination of crevicular fluid flow measurements and cytological smears "could provide valuable physiologic measurements for clinical trials."

Squier and Johnson (1975) discussed the permeability of gingival and sulcular epithelium. Enzymes, toxins and antigens from plaque invade the host tissue and produce inflammation and destruction of tissue supporting the teeth and this reaction is primarily due to an immunological response. Substances pass across skin and oral mucosa by simple diffusion and obey Ficks' Law (the rate is directly proportional to the concentration of the penetrant). They summarized: (1) the external aspect of the gingiva did not demonstrate penetration through the epithelium; (2) the sulcular epithelium was found to be permeable to substances up to a molecular weight of 1.0×10^6; and (3) exogeneous substances enter the junctional epithelium by way of intercellular pathways, since no intercellular barrier such as membrane coating granules exists in the junctional epithelium. They hypothesized that crevicular fluid leaves the tissue at the junction of the oral sulcular epithelium and junctional epithelium. They concluded that "even in the intensely studied gingival area, we have little information concerning the intrinsic permeability of this tissue."

Golub, Slakew, and Singer (1976) removed gingival crevicular fluid from twenty-one patients with and without periodontal disease. The collagenase activity was measured using a radioactive collagen fibril assay. Gingival crevicular fluid collagenase activity, as well as pocket depth and flow amount, were positively correlated to the severity of gingival inflammation. Examination of collagen breakdown products revealed that the fluid collagenase was of tissue origin rather than bacterial. This differs from Smith, Rule, and Rosen (1974) who could detect no activity in gingival crevicular fluid collagenase.

Eisenberg (1977) evaluated lysozyme activity in G C F and its relationship between periodontal health and disease states. Twenty-six subjects were classed according to GI (gingival index) into three groups: normal, gingivitis, and severe. There was no significant difference in lysozyme concentration between non-inflamed gingiva and gingivitis. However, there was higher concentration in patients with periodontitis.

Schenkein and Genco (1977), studying gingival fluid from eighteen subjects with chronically inflamed gingiva, found a marked decrease in C_3 levels in most if not all gingival fluids, and a marked decrease of C_4 levels in some gingival fluids. This suggested that complement might be activated during periodontal inflammation. In a following study, they found C_3 activation products C_3c and C_3d in gingival pocket fluid from patients with severe periodontitis. They stated that the alternate pathway of complement activation occurs in the periodontal pockets, as demonstrated by the conversion of C_3-proactivator (Factor B) to C_3-activator Bb. They also found that C_4 is present in an altered form in some, but not all, gingival fluids.

Golub and Kleinberg (1976) reviewed the literature concerning crevicular fluid and its implications in clinical periodontal therapy. They agreed with Alfano's theory of an osmotic gradient generated by accumulation of macromolecular by-products of bacterial metabolism or degradation. However, those by-products would also increase flow by "directly affecting the crevicular epithelium and the cells of underlying connective tissue rather than simply increasing the osmotic pressure."

They advocated that the dentist should monitor the gingival crevicular flow to assess the severity of gingival disease and the response of tissues to therapy. "One could monitor the response of gingival tissue to various restorative and prosthetic procedures to insure that these procedures do not aggravate the periodontal tissues or induce gingivitis." The chemical or microbial constituents of gingival crevicular fluid could be analyzed in order to evaluate the status of periodontal breakdown.

Suppipat (1976) investigated the clinical usefulness of the HAR-600 Gingival Crevice Fluid Meter. Although readings may vary with different strip location in the machine, room climate, and viscosity of the fluid, he found that measuring gingival fluid is a sensitive and objective method for evaluating the condition of the marginal gingiva. He found that: (1) using the modified intra-crevicular method (Loe and Holm-Pedersen, 1965), little gingival fluid was recovered from clinically healthy gingiva; and (2) gingival fluid was related more to inflammation than to pocket depth.

Smith (1977) proposed that increased awareness of the value of gingival crevicular fluid could result in greater use of it as a diagnostic aid in the clinical situation. It can (1) act as a measurement of severity of gingival inflammation, (2) monitor the effectiveness of oral hygiene, (3) evaluate local periodontal tissue destruction, (4) indicate healing following gingival surgery, (5) measure the effectiveness of periodontal therapy, and (6) demonstrate systemic disease. Dombrowski (1978) uses crevicular fluid as a "rapid chairside test for the severity of periodontal disease."

Spranger (1981) describes the clinical daily usage of measuring crevicular fluid flow by a Periotron. "It is simple, practical, reproducible, objective, and permits rapid quantitative evaluation" of the effectiveness of oral hygiene or periodontal therapy on proper wound healing.

Garnick, Pearson, and Harrell (1979) evaluated the Periotron and found that the Periotron may be useful in assessment of the presence of gingival inflammation. Data from the Periotron showed a high correlation with increased known volumes of fluid and with assessment of gingival inflammation using the Loe's Gingival Index. Successive recordings of the same fluid volume varied from five to eleven percent due to: (1) variations in procedures in obtaining volume especially in the insertion of paper strips between the jaws; (2) fluid evaporation, room temperature, and humidity; and (3) use of the gas chromatographic syringe especially in release of such small quantities of substances.

The literature is replete with studies related to the incidence and prevalence of gingivitis in children.

Wade found that there was a low level of gingivitis at five years (9%), which rose to a peak at eleven to thirteen years (65%). The consequences may vary from mild to severe and if left untreated, may result in degenerative changes (Tsamtsouris and Saadia, 1981). Finn (1973) stressed that periodontal disease is a slow progressive disease which extends over many years and the early stages are common in children.

Stratford (1975) presented a review of literature concerning

gingivitis in children. He concluded that only by early detection of gingivitis can satisfactory preventive measures by instituted at a stage where they are most effective. He promoted the attainment of gingival index which reliably records the initial attack on gingival tissues even though this may not be possible by a clinical observable method. He hoped an index of gingivitis using the gingival exudate will be the answer.

Most investigations of periodontal disease and its relationship to gingival crevicular fluid has concentrated on the adult age group. The following focused on children.

Sandalli and Wade (1971) collected crevicular fluid via filter strip papers from 104 children between three to five years. He concluded that a small measurable fluid flow always exist in children when samples were allowed to be taken. An increase flow occurred when gingivitis is present. A correlation between the amount of gingival flow and crevicular depth is low when gingivae are healthy, but is increased when inflammation is present.

Smith, Golub, and Duperon (1974) evaluated the crevicular fluid of sixty-four children divided into various developmental stages of the dentition: primary, mixed and permanent. They concluded that tooth type had no effect on fluid volume, crevice depth or crevicular pH in children of the same age. While young children showed less crevicular fluid volume, shallower crevices, and lower pH than did adolescents in the healthy non-inflamed group. Increased inflammation caused changes in children similar to adults with exception of crevicular pH which remained unchanged.

Hakim (1977) studied sixty children between the ages of seven and thirteen using filter strip papers and periotron. The results showed a difference of the mean value of crevicular fluid flow of 6.5 from non-inflamed gingiva as opposed to 16.8 for inflamed. Thus, it is generally agreed that crevicular fluid increases its flow with increased inflammation.

Since 1940, the stainless steel crown has found wide acceptance among dental practitioners as a valuable technique within the deciduous dentition for teeth requiring complete coverage. The literature contains many articles concerning proper placement (Myers, Full, Humphrey, and Moore, 1975). However, errors in the placement of stainless steel crowns are frequently observed and gingivitis appears to be associated with defective crowns (Goto, Myers, Henderson, and Wilson, 1970). However, little evidence of a specific correlation between preformed stainless steel crowns and gingival health has been presented.

Goto et al. (1970) adapted stainless steel crowns on 250 primary teeth of sixty-four children ages two to nine. The crowns were observed clinically for thirty to 1,637 days and x-rays taken. An evaluation of the fit of the crown revealed the following results: good, 54 percent; fairly good, 34 percent; failure, 12 percent. They noted a higher percent of gingivitis and loosening of the crown occurred in cases that had been classified as failures. However, gingivitis was noted in 13 percent of the cases that had been classified as good.

Henderson (1973) evaluated 13 percent primary teeth with stainless steel crown from children ranging from four to thirteen years.

He reported a significantly higher marginal gingivitis score on the facial margin as compared to the lingual. Evaluation of the Marginal Gingivitis Index of the various categories of crowns (good, fair, poor) revealed no significant difference in the gingivitis index of the crowns that were evaluated as good or fair. However, there was a higher gingival index for the crowns that were evaluated as poor. The presence of gingivitis was determined clinically by the Ramfjord Index. No matter how accurately the preformed stainless steel crown was trimmed, adapted, and polished in the present study, some inflammation was always observed due to differences in form and contour between crown and tooth.

Myers (1975) evaluated the association between defective stainless steel crowns and a clinical sign of gingivitis. He used Gingival Index of Loe and Silness for gingival evaluation. Seventy-one of the 110 stainless steel crowns had a defect. Improper crown length was the most common error followed by crimp and contour. Several crowns were identified as having more than one error. There was a significant association between crown defects and clinical evidence of gingivitis.

The purpose of this study is to determine the response of gingival tissue to stainless steel crowns by measuring the crevicular fluid content using a periotron. This will be done by quantifying objectively the effect of stainless steel crown on the gingiva in contrast to previously done subjective evaluations. Secondly, the purpose is to determine the correlation between the quality of the stainless steel crown and amount of crevicular fluid flow. Thirdly, the type of error detected in placed stainless steel crowns, its

frequency, and its location will be evaluated.

CHAPTER III

MATERIALS AND METHODS

Thirty children, ranging in age four to ten, participated in this study. None of the children participating had history of systemic disease or were presently taking medication. A consent form was signed by patient's parent (appendix A) after describing the study.

Teeth selected for measuring the crevicular fluid were primary molars, one with a stainless steel crown, and a control tooth with no stainless steel crown located in the same arch. The marginal gingiva in the area of fluid collection of the control tooth was assessed and only that which showed a Gingival Index was required for the patient to participate in this study. All patients with generalized inflammation were eliminated.

Six measurements, mesio-buccal, middle, disto-buccal, mesio-lingual, middle lingual, and disto-lingual, were taken. Therefore, a total of 360 fluid volume measurements were taken. All measurements were taken by the same investigator.

The method for collection used in this research was the intracrevicular method. Fluid is collected from the intracrevicular sulcus employing homogenous sterile papers 1.5 mm. x 13 mm. The collection technique is as follows: (1) the region examined was isolated with cotton balls and dried with air; (2) a filter paper strip is placed into the gingival sulcus orifice for three seconds to empty the

crevicular pool and is discarded; and (3) twenty-seven seconds later, a fresh filter strip was placed in the sulcus for three seconds and the strip placed in the Periotron. The highest number was recorded.

The Periotron is a gingival crevicular fluid flow meter. The meter contains two jaws that function like the plates of an electrical condenser. When a drip strip is inserted, the capacitance is as its maximum, but with the appropriate electronic circuits registering zero on the viewer. Insert of a wet strip provokes a reduction in the capacitance and results in a rise of the reading. This rise is directly proportional to the wet area, specifically to the volume of fluid collected. The standardized strips were marked by a line of insertion between the jaws of the Periotron in order to standardize the sampling. Because of the electronic device a three second insertion would give an indication of the fluid flow with very little tissue irritation.

After the periotron readings were completed, a second examiner evaluated the marginal gingiva of the SSC according to Gingival Index established by Loe and Silness (1963). Also the crown itself was evaluated for errors as listed on the data sheet (appendix B). Using a mirror and explorer, the disto-buccal, buccal, mesio-buccal, disto-lingual, lingual and mesio-lingual areas of the stainless steel crown were evaluated. The interproximal areas, the mesial and disto, were examined by radiography. The crowns were evaluated for errors in length, crimp, contour, polish, position, cement removal, and contact. If no error existed, the designated square on the data sheet was left blank. If an error was found, a positive sign (+) was written.

Gingival Index -- Loe and Silness (1963)

Score

0 Absence of inflammation.

1 Mild inflammation. Slight change in color and little change in texture.

2 Moderate inflammation. Moderate glazing, redness, edema, and hypertrophy. Bleeding on pressure.

3 Severe inflammation. Marked redness and hypertrophy. Tendency to spontaneous bleeding, ulceration.

The length of the crown was defective if the margin was above the crest of the gingiva tissue or below the cemento-enamel junction. Crimping was correct if no space existed between the margin of the crown and the tooth, into which an explorer tip could fit. The position of the crown was unsatisfactory if the crown showed marked rotation and incorrect axial inclination.

The crown contour had to approximate normal tooth contour and not appear flat. The polish was considered smooth if no plier marks or scratches were present, clincially. Cement removal was evaluated radiographically. This is the same criteria as presented by Myers (1975).

CHAPTER IV

EXPERIMENTAL RESULTS

Table 1 is the compiled bibliographical information from the individual data sheets (appendix B). The patient's number, name, sex, primary molar teeth involved, experimental and control, and the date the stainless steel crown was cemented, tested, and their difference in months were recorded. They ranged in age four to eleven with the mean age seven years ten months old. Eighteen of the thirty children were females. The stainless steel crowns had been cemented in from two to sixty-one months, with a mean of nineteen months. Sixteen of the primary molar teeth tested were from the mandible.

The mean Periotron readings for the experiment and control teeth, at the six sites and for each patient are shown on Table 2. The difference between experimental and control mean values are shown. Experimental mean periotron readings at the six sites ranged from 6.93 (buccal midpoint) to 12.30 (disto-buccal papillae). Control values ranged from 2.63 (buccal midpoint) to 4.30 (disto-buccal papillae). Experimental values were significantly different (p 0.05) from control values at all six sites.

The experimental values for the distal papilla periotron readings (11.75) were higher than mesial papilla values (8.82) which in turn were higher than midpoint values (7.85). Lingual periotron values (9.46) were higher than buccal values (9.15). The control

TABLE 1

BIBLIOGRAPHICAL DATA

#	Last Name	First Name	Age	Sex	Teeth		Date (tested)	Date (cmnt)	Months
					Exp	Cnt			
01	Ascensio	Kimberly	10	F	L	S	03/23/81	08/29/77	43
02	Zellergus	Charles	7	M	A	I	03/18/81	01/22/81	2
03	Holk	Tanya	5	F	K	L	03/13/81	11/06/80	4
04	Scipta	Ellen	9	F	J	A	03/21/81	02/20/79	25
05	Malden	Kyle	7	M	A	I	03/13/81	01/11/80	14
06	Zahn	Richard	6	M	S	L	03/18/81	01/16/81	2
07	Minnich	Ralph	6	M	K	T	03/12/81	11/10/80	4
08	Gibson	Heather	10	F	I	B	03/12/81	01/04/80	14
09	Kippa	Joyce	6	F	T	K	03/13/81	04/10/80	11
10	Munich	Paul	4	M	K	L	03/20/81	01/19/81	2
11	Parrish	Virginia	9	F	K	T	02/25/81	02/25/77	48
12	Dancler	Frank	10	M	K	T	02/11/81	01/20/78	37
13	Benko	Allison	10	F	J	A	02/25/81	12/03/76	51
14	Jackson	Figgiero	7	M	B	I	02/25/81	08/22/80	6
15	Vargas	Pauline	7	F	B	I	03/03/81	12/28/80	3
16	Ko	Laura	11	F	J	A	02/05/81	08/18/77	42

TABLE 1 (continued)

#	Last Name	First Name	Age	Sex	Teeth		Date (tested)	Date (cmnt)	Months
					Exp	Cnt			
17	Blake	Karrie	7	F	I	J	03/12/81	11/10/78	28
18	Daczman	Natalie	11	F	T	K	03/21/81	01/15/76	62
19	Broderich	Rachael	9	F	S	L	02/09/81	11/12/80	3
20	Richardson	Amy	8	F	J	B	03/12/81	03/28/78	35
21	Gilfarb	Elaine	6	F	K	L	02/20/81	10/03/80	5
22	Saura	Gregory	7	M	B	I	04/11/81	04/11/78	36
23	Garcia	Jamy	7	M	K	T	04/09/81	11/12/80	6
24	Sanchez	Adam	5	M	L	S	03/11/81	09/26/80	6
25	Zeman	Valerie	11	F	A	J	02/12/81	04/17/79	22
26	Gorecki	Susan	11	F	A	J	02/22/81	02/26/79	24
27	Winter	John	8	M	K	T	03/17/81	01/04/81	2
28	Jurkowski	Ryan	9	F	S	T	01/15/81	02/21/78	35
29	Vickroy	John	5	F	T	K	01/20/81	10/15/80	3
30	Facio	Tracy	7	F	B	O	02/12/81	12/09/80	2

TABLE 2

PERIOTRON READINGS: BY SITE, BY TOOTH,
EXPERIMENTAL AND CONTROL

PT#	B:MP	B:DP	B:MID	L:MP	L:DP	L:MID	$\overline{X}$	Difference Expy-Con
01E	04	20	16	05	13	12	11.67	
01C	01	01	04	01	05	02	2.33	9.33
02E	02	08	02	03	09	02	4.33	
02C	03	04	01	00	05	00	2.17	2.17
03E	02	08	05	05	03	06	4.83	
03C	02	03	02	02	02	01	1.83	3.00
04E	18	20	22	19	15	14	18.00	
04C	04	12	02	03	11	07	6.30	11.5
05E	07	14	04	15	16	08	10.67	
05C	07	02	01	01	02	01	2.33	8.33
06E	08	06	04	08	05	05	6.00	
06C	02	01	01	01	01	06	2.00	4.00
07E	05	07	02	02	03	02	3.50	
07C	02	05	03	02	03	01	2.67	.83
08E	07	06	01	03	10	12	6.50	
08C	05	06	02	04	06	05	4.67	1.83

E = experimental
C = control
B = buccal
L = lingual
MP = mesial papilla
DP = distal papilla
MID = middle
$\overline{X}$ = average of periotron readings for tooth

TABLE 2 (continued)

PT#	B:MP	B:DP	B:MID	L:MP	L:DP	L:MID	$\overline{X}$	Difference Expy-Con
09E	02	01	01	04	09	04	3.50	
09C	01	01	01	04	04	08	3.17	0.33
10E	07	04	12	14	08	05	8.33	
10C	04	07	08	04	05	03	5.17	3.17
11E	24	27	07	13	12	14	16.17	
11C	04	03	02	04	03	02	3.00	13.17
12E	01	08	09	14	08	03	7.17	
12C	02	01	03	10	07	06	4.83	2.33
13E	12	23	09	22	25	20	18.50	
13C	04	06	03	06	03	05	4.50	14.00
14E	08	18	09	09	22	09	12.50	
14C	06	08	02	09	02	03	5.00	7.5
15E	11	15	07	07	13	09	10.33	
15C	01	14	07	01	08	03	5.67	4.67
16E	06	08	06	08	02	06	6.00	
16C	00	02	00	02	02	00	1.00	5.00
17E	01	09	01	01	02	01	2.50	
17C	04	02	01	01	01	01	1.67	0.83

E = experimental
C = control
B = buccal
L = lingual
MP = mesial papilla
DP = distal papilla
MID = middle
$\overline{X}$ = average of periotron readings for tooth

TABLE 2 (continued)

PT#	B:MP	B:DP	B:MID	L:MP	L:DP	L:MID	$\bar{X}$	Difference Expy-Con
18E	12	14	09	09	13	06	10.50	
18C	03	05	00	03	08	06	4.17	6.33
19E	11	09	03	23	13	13	12.00	
19C	08	05	01	09	13	07	7.17	4.83
20E	12	05	06	06	13	07	8.17	
20C	04	04	03	06	03	02	3.67	4.50
21E	05	35	10	07	27	20	17.33	
21C	00	05	00	02	01	00	1.33	16.00
22E	09	09	05	07	05	02	6.17	
22C	05	09	02	02	08	04	5.00	1.17
23E	11	05	06	06	06	02	6.00	
23C	08	05	04	02	03	02	4.00	2.00
24E	10	20	09	19	18	30	17.67	
24C	05	01	01	04	03	01	2.50	15.17
25E	10	24	03	12	07	04	10.00	
25C	01	05	02	11	05	09	5.50	4.50
26E	14	21	12	11	11	10	13.17	
26C	12	03	13	09	03	02	7.00	6.17

E = experimental
C = control
B = buccal
L = lingual
MP = mesial papilla
DP = distal papilla
MID = middle
$\bar{X}$ = average of periotron readings for tooth

TABLE 2 (continued)

PT#	B:MP	B:DP	B:MID	L:MP	L:DP	L:MID	$\overline{X}$	Difference Expy-Con
27E	07	10	12	12	10	01	8.67	
27C	05	03	03	02	03	05	3.50	5.17
28E	01	01	01	01	12	02	3.00	
28C	01	01	00	00	00	00	.33	2.67
29E	10	02	06	13	09	30	11.67	
29C	01	04	03	01	05	01	2.50	9.17
30E	10	12	07	10	17	04	10.33	
30C	08	01	05	01	01	01	2.83	7.50
E	8.23	12.30	6.93	9.60	11.20	8.77		
C	3.77	4.30	2.63	3.57	4.20	3.13		
	4.46	8.0	4.3	6.03	7.0	5.64	Overall	5.94

E = experimental
C = control
B = buccal
L = lingual
MP = mesial papilla
DP = distal papilla
MID = middle
$\overline{X}$ = average of periotron readings for tooth

values for the distal papilla, mesial papilla, and midpoint showed the same order of rank: 4.25, 3.67, 2.88, respectively. The lingual mean control values were 3.64, while the buccal 3.57.

The mean periotron reading from all six sites for each control tooth ranged from .33 to 7.17. The mean periotron reading from an experimental tooth ranged from 2.5 to 18.0. The differences between the means of each experimental and control teeth ranged from 0.33 to 16.00. The mean experimental value for all thirty experimental teeth was 9.51, while the mean control average was 3.57. This was significantly different at the 0.05 level (table 4). The overall mean difference between experimental and control was 5.94.

Statistical compilation of data with Loe's clinical readings, for the six sites, for the thirty experimental teeth and for the overall averages is presented in Table 3. On the control teeth, all sites evaluated, showed no clinical sign of inflammation, thus receiving a Loe's reading of 0 (see appendix B).

The range of Loe's values at the six experimental sites ranged from 0.97 to 1.20. Like the periotron readings, the distal papilla showed the largest value (1.15), followed by the mesial papillae (1.10), and midpoints (1.06). In contrast to the periotron readings, the buccal mean (1.14) value is slightly larger than the lingual (1.03).

The range of values from the thirty experimental teeth range from 0 to 2.17. The difference between experimental and control teeth ranged the same. The average mean value for all thirty experimental teeth was 1.09, while the mean control average was 0. This was

TABLE 4

TESTS OF SIGNIFICANCE

Peritron Values
Exp. versus Control

df	T tabulated	T calculated	Significance
29	2.05	7.28	$p<0.05$

Loe's Values
Exp. versus Control

df	T tabulated	T calculated	Significance
29	2.05	9.12	$p<0.05$

TABLE 3

LOE-SILNESS CLINICAL EVALUATION OF
EXPERIMENTAL TEETH BY SITE AND TOOTH

PT#	MB	BU	DB	DL	LI	ML	E	
01	2	2	2	1	1	1	1.50	1.50
02	0	0	0	0	0	0	0.00	0.00
03	1	1	2	1	1	1	1.17	1.17
04	2	2	2	2	2	2	2.00	2.00
05	1	1	1	1	1	1	1.00	1.00
06	1	2	2	1	1	1	1.33	1.33
07	1	1	1	0	0	0	0.50	0.50
08	0	0	1	1	1	1	0.67	0.67
09	0	0	0	1	0	0	0.17	0.17
10	0	0	0	0	0	0	0.00	0.00
11	2	3	3	2	2	2	2.33	2.33
12	1	1	1	1	1	1	1.00	1.00
13	2	2	2	2	2	2	2.00	2.00
14	1	1	2	2	1	1	1.33	1.33
15	2	2	2	1	1	1	1.50	1.50
16	2	0	0	2	0	1	0.83	0.83
17	0	0	0	0	0	0	0.00	0.00
18	1	1	1	1	1	1	1.00	1.00
19	2	1	2	2	3	3	2.17	2.17
20	1	1	1	2	1	1	1.17	1.17
21	1	2	2	2	2	1	1.67	1.67
22	0	0	0	0	1	0	0.17	0.17
23	2	1	1	1	1	1	1.17	1.17
24	2	1	1	1	1	1	1.17	1.17
25	1	2	1	1	1	1	1.17	1.17
26	2	2	2	1	1	1	1.50	1.50
27	1	1	1	1	1	1	1.00	1.00
28	0	0	0	0	1	0	0.17	0.17
29	1	1	1	2	2	2	1.50	1.50
30	2	2	2	1	1	1	1.50	1.50
	1.13	1.10	1.20	1.10	1.03	0.97		
	3.43 = 1.14			3.1 = 1.03			1.09	

Footnote: On the control teeth, all sites showed no clinical signs of inflammation, having a Loe's reading of 0. Thus C is also 0.

PT# = patient number
MB = mesio-buccal
BU = middle of buccal
DB = disto-buccal
DL = disto-lingual
LI = middle of lingual
ML = mesio-lingual

significantly different at the 0.05 level (table 4).

The test of significance for the effect of stainless steel crowns on the gingiva is measured (Table 4) by the difference in mean periotron readings of the thirty experimental teeth and thirty control. A second test of significance for the effects of stainless steel crowns on the gingiva was performed by using Loe's system of evaluation for clinical inflammation. Both methods showed a significant difference between mean experimental and mean control values at the 0.05 level. This can be attributed to the presence of the stainless steel crown with various numbers of errors in them.

Table 5 summarized the crown evaluations tabulated from individual data sheets. The range of errors per tooth is shown and ranged from 0 to 20 with the mean number of errors per tooth 9.7.

The frequency of errors according to sites on the stainless steel crown is summarized on Table 6. The most frequent site of error on a stainless steel crown was the disto-lingual. The most frequent error found was crimp, followed by length and contour. Infrequently found errors included polish, position, cement, and contact. On the average, there was at least one error per site, per tooth.

The number of teeth affected by a particular error at one site is shown on Table 7. Eighty-three percent of the teeth had an error in crimp while 80 percent had an error in length of the stainless steel crowns. Less frequent errors were found in contour (57%) and polish (27%). Infrequently found, were errors in contact, position and cement.

When errors according to location of the crown on tooth is

TABLE 5

STAINLESS STEEL CROWN EVALUATIONS FOR
ERRORS BY TOOTH AND SITE

PT#	LENGTH	CRIMP	CONTOUR	CONTACT	POS'N	POLISH	CEMENT	TOTAL # OF ERRORS ON TOOTH
01		BCH	ABC					6
02	CE	DH	DH			B		7
03	BCEFGH	DE						8
04	ABCDGH	CDEGH	ABCEFG					17
05		BDEFG	BDEFGH					11
06	G	ABCFH	ABCH				E	11
07			ABDEFGH					7
08	EFG							3
09	AE	DH				E		5
10	EFG		B					4
11	ABCEFG	ABCDH	DH		ABH			16
12	E	BCEFGH	ABCDEFGH					15

Key:
A = mesio-buccal
B = buccal
C = disto-buccal
D = disto-interproximal
E = disto-lingual
F = lingual
G = mesio-lingual
H = mesio-interproximal

TABLE 5 (continued)

PT#	LENGTH	CRIMP	CONTOUR	CONTACT	POS'N	POLISH	CEMENT	TOTAL # OF ERRORS ON TOOTH
13	CEFG	ADEFGH	DH			ABC		15
14	CE	DH		D		ABC	D	9
15	DEFGH	ADH	DH					10
16	ADEFGH	ADH						9
17								0
18		CEFH						4
19	ACEFG	CH						7
20	AEH	FH						5
21	ACDEG	EFGH	EFG			EFG		15
22	ABCG							4
23	ACEFG	BDE	ABC			ABCEG		16
24		CEFGH	ABCEFG					11
25	D	GH	ABCG					7

Key: A = mesio-buccal
B = buccal
C = disto-buccal
D = disto-interproximal
E = disto-lingual
F = lingual
G = mesio-lingual
H = mesio-interproximal

TABLE 5 (continued)

PT#	LENGTH	CRIMP	CONTOUR	CONTACT	POS'N	POLISH	CEMENT	TOTAL # OF ERRORS ON TOOTH
26	EFGH	H	ABCEFGH				E	13
27	C	ABCDEFGH*	ABCEFG	H				16
28	B	BDE						4
29	BEFG	ABDEFGH			EFG	ABCEFG		20
30	CDEFGH	ABCDEFGH				A		15

Key: A = mesio-buccal
B = buccal
C = disto-buccal
D = disto-interproximal
E = disto-lingual
F = lingual
G = mesio-lingual
H = mesio-interproximal

TABLE 6

NUMBER OF ERRORS ACCORDING TO LOCATION ON TOOTH

	B: mesial	B: middle	B: distal	D	Distal-Lingual	L: mid	Lingual	M	
Length	9	6	12	6	18	12	16	7 =	86
Crimp	8	10	10	15	13	11	10	21 =	98
Contour	10	11	10	6	8	8	9	10 =	72
Contact				1				1 =	2
Position	1	1			1	1	1	1 =	6
Polish	5	5	4		4	2	3	=	23
Cement				1	2			=	3
	33	33	36	29	46	34	39	40	
									290

TABLE 7

PERCENTAGE OF TEETH AFFECTED
BY A SPECIFIC ERROR

	Length	Crimp	Contour	Contact	Position	Polish	Cement
Number of Teeth Affected	24	25	26	2	2	8	3
Percentage	80	83	57	7	7	27	10
Total Errors	86	98	72	2	6	23	3

contrasted to number of different type of errors, it can be concluded that errors in length, crimp, contour and polish tend to occur more than once on a particular tooth.

The correlation coefficients between a number of variables are summarized in Table 8. There was found a highly significant correlation ($p<0.05$) between the total errors found on a tooth and the difference between experimental and control values for both periotron readings and Loe's clinical evaluations. A significant correlation ($p<0.05$) was found between periotron readings and Loe's clinical evaluation (difference of experimental minus control) for each tooth. No relationship was found between the months that a stainless steel crown had been in the mouth and the effect it had on the gingiva as measured by the periotron and Loe's index.

Table 9 summarizes the correlation between the number of errors at a site with the difference in periotron readings at that site. This was an attempt to break down the significant correlation found between total errors on a tooth and the respective reading in Table 8. Even though the highest correlation was found between the periotron and number of errors at a site, only three of the six possible sites showed a significant correlation between Loe's index and the number of errors.

TABLE 8

TABLE OF CORRELATIONS

Independent Variable	Dependent Variable	Correlation Coefficient	Significance
Months SSC in mouth	Periotron reading	0.16	NS
Months SSC in mouth	Loe's clinical evaluation	0.10	NS
Total errors on SSC	Periotron reading	0.57	$p<0.05$
Total errors on SSC	Loe's clinical evaluation	0.66	$p<0.05$
Periotron readings	Loe's clinical evaluation	0.70	$p<0.05$

TABLE 9

CORRELATION BETWEEN NUMBER OF ERRORS AT A SITE AND PERIOTRON AND GINGIVAL INDEX EVALUATION DIFFERENCE (EXPERIMENTAL-CONTROL)

	Evaluation Sites					
	BMP	BMID	BDP	LMP	LMID	LDP
Periotron	0.50*	0.24	0.25	0.58*	0.65*	0.33
Loe's	0.63*	0.32	0.53*	0.44*	0.44*	0.38*

* = significant at $p<0.05$

CHAPTER V

DISCUSSION

In this study, the periotron was used to quantitate the presence and severity of gingivitis. Its validity has been shown many times previously (Brill, 1962; Mann, 1963; Egelberg, 1964; Oliver st al, 1969; Spranger, 1981). Spranger (1981) evaluated the periotron to measure crevicular fluid flow and stated its superiority to other techniques for measuring crevicular fluid flow. The clinician merely collects a sample of exudate on filter paper strips which are scored blindly by the periotron, allowing no bias. The numerical value is directly proportional to the area involved and volume of fluid collected. The periotron is simple, practical, and reproducible and permits rapid quantitative evaluation. A high correlation was found ($p<0.05$) between the Loe-Silness Gingival Index and the crevicular fluid flow readings (table 8). Similar findings between an index based on clinical observation and examination and crevicular fluid flow was reported previously by Oliver et al. (1969), Wilson (1971), and Spranger (1981).

The purpose of the investigation was to try to relate the quality of stainless steel crowns to crevicular fluid flow. Actually, this objective purpose encompasses two questions: (1) the effect of a perfectly made stainless steel crown on the gingiva, and (2) the effects of different errors at specific sites on the stainless steel

crown on the gingiva. However, to find a stainless steel crown with no errors is exceedingly difficult. Indeed, our survey found only one stainless steel crown with no errors. The difference in crevicular fluid flow between experimental and control on this tooth was 0.83, which suggests that a well done stainless steel crown has a minimal, if any, effect on the gingiva as assessed by the periotron.

The Loe-Silness clinical evaluation, although not as precise as the periotron, showed no difference between the effect of the stainless steel crown on the gingiva.

Another way to assess the effect of stainless steel crowns on the gingiva is to analyze those teeth with the fewest errors and extrapolate towards teeth with no errors. If we look at the stainless steel crowns with less than six total errors (an average of less than one error per site), we have a sample of eight patients--#8, 9, 10, 17, 18, 20, 22, 28. The difference between experimental and control on these eight teeth is 2.6, certainly less than the overall average of 5.94 for the thirty patients.

The regression equations for errors versus difference in periotron for these eight patients shows a y- intercept of 0.7. In other words, if these patients are representative of higher quality stainless steel crowns and if this relationship is linear, then we would expect that a stainless steel crown, with no errors, would have a minimal effect on the gingiva as assessed by the periotron. Indeed, the regression equation for errors versus difference in periotron readings for all thirty patients shows a y- intercept of 1.1. Judging from the one perfect stainless steel crown and from the regression line

extrapolations from the eight patients and thirty patient group, the evidence is highly suggestive that the stainless steel crown has a minimal impact on the gingiva as measured by the periotron (see following page).

The literature concerning the effect of stainless steel crowns on the gingiva is not unanimous, largely because different criteria to evaluate gingival health. Weber (1974) found that no gingival changes resulted after placement of stainless steel crowns. He also reported that the length of time did not have a deleterious effect on the gingiva. His evaluation did not encompass subgingival plaque deposits. Myers (1975) examined 110 stainless steel crowns and found 39 without errors. Seventy-five percent of these were assessed to be free of gingivitis.

Henderson (1974), however, found that no matter how well done a stainless steel crown may be, there is always some gingivitis present. Goto et al. evaluated the effect of stainless steel crowns per se and reported a high incidence of gingivitis around well done crowns. However, he did not elaborate on the gingival condition before placement of the crown.

However, one study is more concerned with the second question; does the clinical stainless steel crown with its frequent errors have an effect on the gingiva as measured by the periotron. The tests of significance rejects the null hypothesis that there is no effect (table 4).

This concurs with Goto (1970) who found the highest incidence of periodontal complications resulted from crowns classified with the

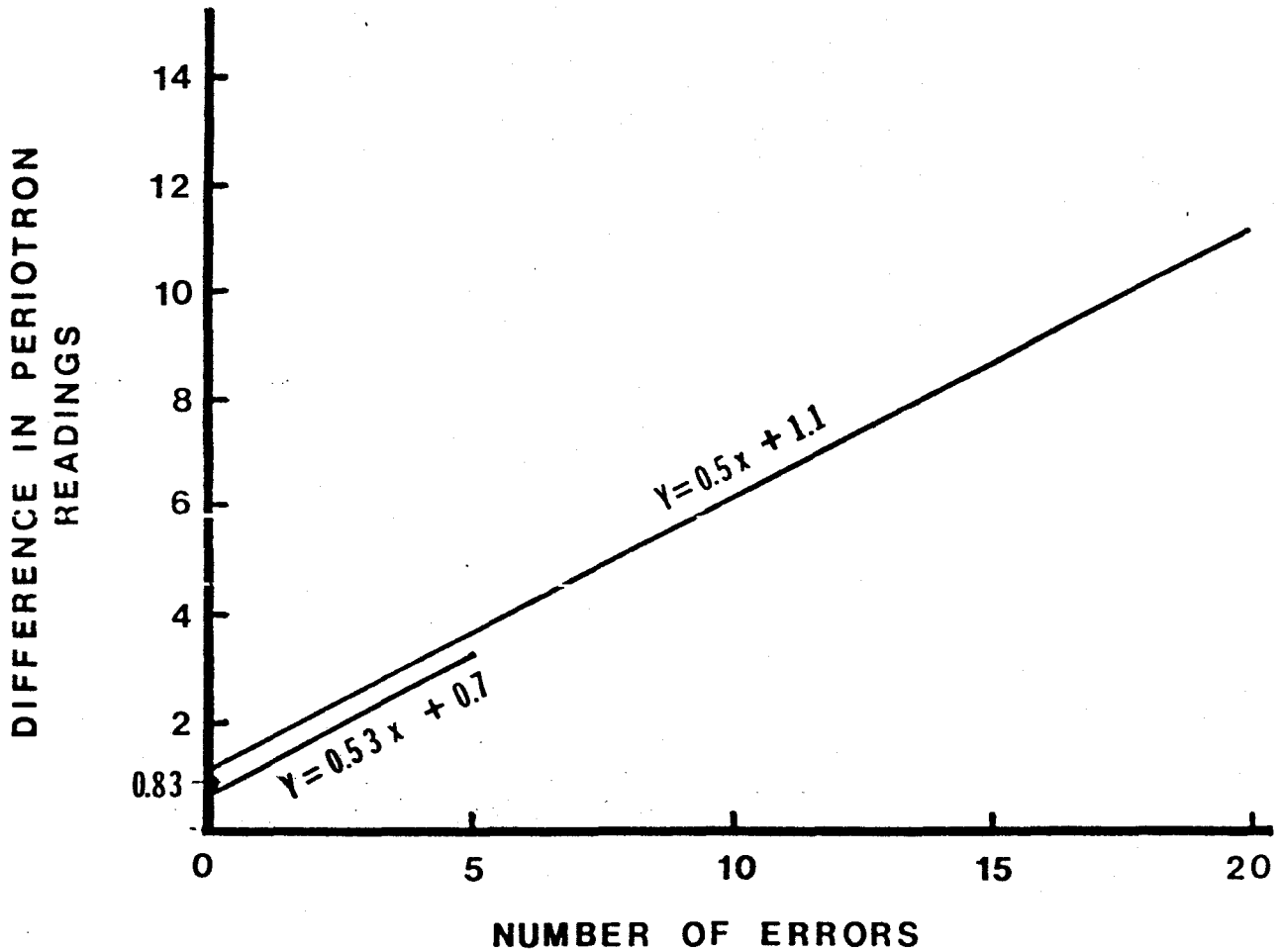
DIFFERENCE IN PERIOTRON READINGS
14
12
10
8
6
4
2
0.83
0
5
10
15
20
NUMBER OF ERRORS
Y=0.5x + 1.1
Y=0.53x + 0.7

FIGURE 1

poorest marginal fit. Henderson (1974) found that the poorer the crown fit, the greater plaque deposits and poor fitting crowns resulted in a higher incidence of gingivitis. Myers (1975) also found that gingivitis was associated with poor crown adaptation. Defects in the case may enhance plaque accumulation, thus accounting for the association between gingivitis and defective stainless steel crown.

This is in agreement with other studies relating errors and gingivitis (Jameson, 1979; Goto, 1970; Henderson, 1974; Myers, 1975).

But it is the correlation coefficient analysis (table 8) which will assess quality. If the number of errors causes an increase in crevicular fluid flow, then we will see a positive and significant correlation coefficient. Table 7 shows this to be the case, that with a greater number of errors on a stainless steel crown you will find an increased crevicular fluid flow. Table 7 also shows that with a greater number of errors on a stainless steel crown, there will be a deleterious change in the gingiva as evaluated by Loe's criteria.

That, however, was just the total number of errors on a stainless steel crown causing a gingivitis. The clinical import of this is that the poorer a stainless steel crown is, the more severe will be the ensuing gingivitis.

This study, however, examined the types of errors and the sites at which they occurred in an attempt to see if the teaching of stainless steel crown preparation could be improved to avoid these pitfalls.

By far, the most frequent errors were in crimp, length, and contour. Since the first two are seen on two-thirds of all crowns examined and contour on one-half, it would seem that more time should

probably be invested in teaching these details. Comparing the total errors with the percentage of teeth affected, it is obvious that these errors, when they occur, tend to occur in multiples.

Ashrafi (1981) reported errors in crown crimp 66 percent of the time. Myers (1975) reported errors in length, crimp and contour; the most common with frequently more than one type of error occurring on a tooth.

The sites that errors occurred followed a pattern. The most frequent site for an error distinctly tended to be on the distal, especially the disto-lingual. The most frequent error at these sites was the length. The most frequent interproximal error tended to be in the crimp, causing an impingement of the crown on the gingiva.

The difference between experimental and control periotron readings at the six sites tended to follow a similar pattern. The highest differences were found at distal sites and on the lingual, which, of course, are the most difficult to see. This agreed with the order of frequency of errors at a site. The middle of either the buccal or lingual, certainly more accessible to vision than either papilla, showed the lowest difference in periotron scores. The buccal was less than lingual. This suggests how good stainless steel crowns can be if the operator is patient and diligent.

Table 9 summarizes an attempt to correlate the number of errors at a site with the difference in periotron readings at that site. This was done to see if an error at one site can cause changes at an adjacent site or not. Since only three of six sites showed a significant correlation ($p<0.05$) interpretation is somewhat chancy. This tends to

show that the errors on a crown have a cumulative effect on the gingiva.

Interestingly enough, Loe's clinical evaluation showed a higher correlation for the whole tooth and a significant correlation in five of six sites. This is important because one would think that since it only has four gradation, instead of the virtual endless possibilities for the periotron, that it would not be as precise. It is also very good that the two evaluations correlate significantly with each other ($p<0.05$).

One last possibility was investigated. It could be that a stainless steel crown could be cemented in, and with time, cause a continuous degenerative effect on the gingiva. The correlation tests showed no significant relationship existed. This means that it is the quality and not the time it is in place that determines the state of gingival health. Marcum (1967) demonstrated histologically, on cast gold crowns placed in dogs, that length of time a restoration was in place had little, if any, effect on severity or degree of inflammation.

CHAPTER VI

SUMMARY AND CONCLUSIONS

Our study found that almost every stainless steel crown had errors and that these errors were largely responsible for the gingivitis as assessed by crevicular fluid flow readings and Loe's clinical evaluation. The most frequent errors were in crimp, contour, and length. These errors were more frequent on the lingual and tended to occur multiply.

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APPENDIX A

Consent Form

The purpose of this study is to determine if there is an increase in the amount of fluid flowing from the space between the teeth and gums after placing a stainless steel crown. The procedure will involve drying each tooth with cotton rolls, then placing a small sterile piece of filter paper next to the tooth near the gums for about three seconds. This will not produce any discomfort whatsoever or have any effect on the teeth or gums.

My child may participate in this study.

Signature _____

Date _____

APPENDIX B

Date

Patient

Operator

Tooth

Date cemented

Crown Evaluation

Periotron Score

	MB	BU	DB	D	DL	LI	ML	M	X	Buccal		
Length										MP		
Crimp										DP		
Contour										M		
Contact										Lingual		
Position										MP		
Polish										DP		
Cement										M		

Clinical Evaluation

Control									
SSC									

APPROVAL SHEET

The thesis submitted by Dr. Renee Balthazar has been read and approved by the following committee:

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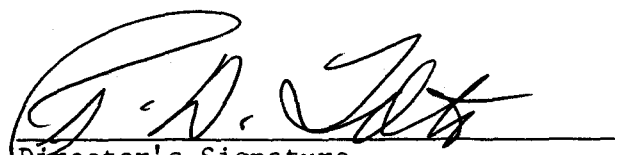
The final copies have been examined by the director of the thesis and the signature which appears below verifies the fact that any necessary changes have been incorporated and that the thesis is now given final approval by the Committee with reference to content and form.

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

3-27-84
Date


Director's Signature