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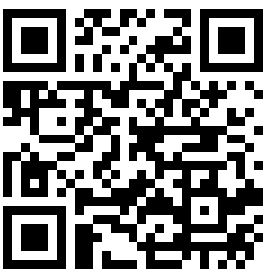

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CHARACTERIZATION AND STAINING OF PORCELAIN FUSED TO METAL RESTORATIONS

by

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Professional paper submitted in partial fulfillment of the requirements of the U.S. Army Dental Residency Program, Walter Reed General Hospital, Walter Reed Army Medical Center, Washington, D.C. 20012

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CHAPTER I

Introduction

When the patient leaves the office, especially those with anterior restorations, they often ask: "How does it look?". For the answer to be: "It matches!" and this response to be consistant, staining procedures and techniques must be developed.

Almost every porcelain fused to metal restoration must be stained and characterized to match the natural tooth. At present the custom is for many Dentists to write a prescription detailing his needs. The staining and characterization is accomplished in the laboratory. When the restoration is returned to the Dentist, he will ultimately have to refine the contour and shade of the restoration.

This paper will outline a practical method of staining and characterizing teeth in the dental office. The goal should be to satisfy the Dentist's need to simulate nature and the patient's need for esthetics.

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CHAPTER II

Historical Review

The art of enameling goes back several centuries before Christ. It was from the early works of these unknown artisans that today's technique of fusing porcelain to metal evolved. Enamels were first used by the Turks in 9000 B.C. and by the Chinese in 1500 B.C.[1] It was the Egyptians, who developed the original form of enameling on metal, although they were limited to two vitreous colors, blue and white. They fused glass around a piece of metal.[2,3] It was not until the early Christian Era that true enameling, or as it was called originally, "electron", was used. The Byzantine period saw the refinement of enameling into two forms, cloisonne and champleve. The first technique was developed by the goldsmiths who soldered gold wire around the object to be enameled. They over-packed the remaining space with powdered enamel or paste, fired the porcelain and polished its surface. The second technique, champleve, was developed by the coppersmiths who completely fabricated the metal object, then carved out the area to be enameled. The packing and treatment of the porcelain was the same as for cloisonne.[2,3]

These refinements allowed enameling to slowly cover Europe, with the inevitable use of many varieties of metals

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and porcelains. It was not until the 18th century when enameling was found to have a protective effect on sheet metal and cast iron did it become an industry.[3] The bath-tub became its greatest example.

The first reference made concerning the fabrication of enameled dental restorations was by C. Mouton in 1740. He applied enameling to his anterior crowns which he called Calottes D'or.[4] The next reference was not until one hundred and thirty-one years later, in 1871, when B. J. Bing developed the porcelain pontic with platinum extensions for anchorage to the adjacent teeth.[5] The first staining of artificial teeth was presented by Dunn.[6] Low fusing porcelain was developed in 1886, and it was not until 1889, that C. H. Land developed the porcelain crown fused to a platinum matrix. In 1898, N. S. Jenkins perfected porcelain that would, when fused, develop stability, durability and color stability. From 1900 until 1951, porcelain fused to metal restorations became obscure in fixed prosthodontics, due to technical difficulties.[5]

Between 1951 and 1964, new alloys and porcelains were developed in conjunction with high speed preparation techniques, and porcelain fused to metal restorations became popular. This popularity of porcelain fused to metal restorations immediately increased the need for the dentists and technicians to understand the colors present in natural

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teeth and the need to develop a technique of staining and characterization of restorations.[5,7]

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CHAPTER III

Color

The development of the metal alloys and stable porcelain adjuvants for the fabrication of porcelain fused to metal restorations has provided the clinician with an acceptable restoration that will harmonize with his remaining natural dentition. The completed restoration still depends on the individual clinicians ability to reproduce the character and color necessary to create this illusion.

This has required the clinician to develop an understanding of the phenomenun of color. In 1933 Clark[8] said:

> "color, like form, has three dimensions, but they are not in general use. Many of us have not been taught their names, nor the scales of their measurement. In other words, we as Dentists are not educationally equipped to approach a color problem".

This is still the problem.[9] Color is an observable phenomena. It is the result of a change in light by a colorant and the pigment used in coloring materials. The color seen by the eye (a physiophysical process), is relayed to the brain for interpretation.[10,11] Since color exists only in the mind of the viewer, it has no value in our interrelationship with man. To find its worth, one must translate color into a physical stimulus.[11,12,13] For this, the production of color requires three things: a source of light, an object which it illuminates, and the eye and brain to per-

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ceive the color (Figure 1).[3,10-15]

Before entering into a discussion about color systems, several factors should be taken into account. First, the relative insensitivity of the eye limits us to the visible spectrum (Figure 2), which is from 3900 to 7700 A.U.[3,11] Sir Isaac Newton (1730) demonstrated that by passing white light through a prism, it was dispursed into its major visible component colors. These were dependent on their wavelengths and reflective indices. When the parts were returned through a similar prism, white light was again obtained (Figure 3). Each color is represented by their dominant hue.[10,16] This means that the character of the transmitted light influences the colors present, since rays present in the original light cannot be reflected or transmitted.[3,11] Thus the source of light in a room, for example the filaments of light bulbs, fluorescent lamps, and the sun can affect our final restoration. As light hits an object it is reflected, refracted and dispursed. It is noted (Figure 4) that when the light beam (incident beam) hits the object and is reflected, the angle of incidence and reflection are the same. At the same time, part of the light will be absorbed into the object. Simultaneously, part of the transmitted light is dispursed through-out the object. In the case of the enameled restorations, all this will depend on the crystalline structure of the porcelain.[3,11,16] This

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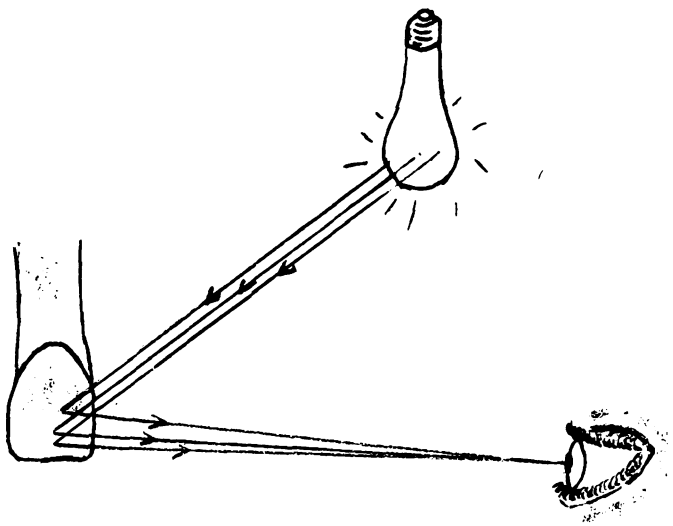

Figure 1: Production of Color

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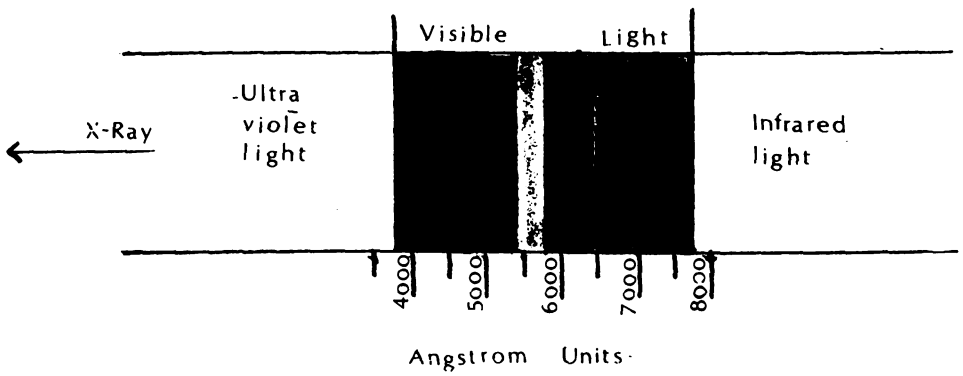

Figure 2: Range of Color Spectrum

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Applications of fluoride to immature or slightly demineralized enamel (initial stage of caries) may provide micro-spaces for fluoride ion uptake, thereby increasing the enamel resistance to subsequent acid attacks. It is also possible to remineralize the subsurface layer of dentin which is demineralized, but not infected.[16,26,37]

The reaction between hydroxyapatite and sodium mono-fluro-phosphate has been shown to be inhibited by salivary o-phosphate and bicarbonate. Therefore, as the flow of saliva is stimulated, the bicarbonate level rises and the inhibition of the reaction becomes more marked because of the increase in pH. Conversely, the decrease in pH will favor the reaction. Current research has shown that a greater amount of fluorapatite (the desired end product) rather than CaF_2 is formed in enamel which is pre-treated with DCPD (dicalcium phosphate dihydrate) and then exposed to a dilute (500ppm) fluoride solution. This minimizes etching of the enamel surface and the deleterious loss of minerals which pre-treating with acid causes. This data also demonstrated that the fluoride penetrated to a deeper layer when DCPD was used as opposed to acid.[38,39] However, decalcification of enamel has been shown to be independent of penetration depth of fluoride.[31] In research using DCPD as a pretreatment solution, the proper amount of

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fluoride must be used in respect to the phosphate ions available in order that the optimum amount of fluorapatite is formed. This proper amount is much less than that found in the concentrated gels discussed earlier. In addition, it was stated that, "It is yet to be shown that significant amounts of fluorapatite are produced in tooth enamel by any of the presently used topical fluoride agents."[38]

Another author suggests that fluoride is not effective in the prevention of post-irradiation dental lesions because the patient's tissues are too sore to use the fluoride carriers.[14] Nevertheless, much clinical evidence has borne out the effectiveness of fluoride gels in the prevention and treatment of post-irradiation dental demineralization. The use of them should be strongly encouraged for irradiated patients on a regular basis. Close Follow-up is mandatory.

D DIET

Another important step in controlling and preventing dental caries in the irradiated patient is to reduce the cariogenic sucrose intake in the diet.[16] Difficulty in eating due to xerostomia makes it necessary for irradiated patients to ingest a soft or liquid diet since food particles cling to the teeth. Soft diets are usually high in carbohydrates and, with the lack of salivary lubrication,

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produce heavy plaques which correlate with an increased cariogenic effect.[11,14,17,18]

Recognized as the most cariogenic food factor, sugar (sucrose) is an absolutely unessential part of our diet.[40,41] The cariogenicity of all food is directly related to its sucrose content. Studies have shown that a 5% solution of sucrose is necessary to decrease the pH of plaque sufficiently to decalcify tooth mineral. Sugar levels higher than 5% further decrease the pH. Most mints, candies, cookies and breakfast cereals contain a percentage of sucrose which far exceeds the 5% level.[41,42] Processing of natural foodstuffs increases the sucrose content and consequently its cariogenicity. Therefore, natural foods such as fruits and nuts should be substituted for "sweets" especially for between meal and bedtime snacks since salivary flow is lowest at these times.

Stimulated saliva, well known for its buffering capacity, decreases the destructive action of mealtime sweets upon enamel in comparison to between meal snacks eaten when salivary flow is greatly diminished. Other factors that influence the seriousness of the caries attack are the consistency of the saliva, the time period of the sugar in the mouth, and the presence of caries inhibiting factors such as fat or phosphate.[13,16] Since the quantity

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and quality of saliva in the irradiated patient is reduced, these controllable factors must be altered favorably.

Streptococcus mutans and other acid producing bacteria utilize any sucrose supplied by the diet for dextran formation which becomes part of the plaque, and forms an ideal hiding place for bacteria. Other sugars are used by the plaque bacteria for acid production which lowers the pH. Demineralization of the enamel commences when the plaque PH drops below 5.6 and continues until the pH rises above this critical point. Therefore, if high sucrose containing plaque is allowed to remain around the necks of teeth, as is often the case in irradiated patients, the pH level never gets a chance to return to the remineralization level and the result is caries.[43]

"To a large extent, good nutrition fosters oral health, whereas poor nutrition contributes to the disease process. No other etiological factors can influence the outcome of a disease such as dental caries as much as the type and quantity of food and the frequency with which it is consumed."[44] Nutritional guidance has been neglected as one of the major factors in the prevention of dental caries in the irradiated patient as well as routine dental patients. The dentist's lack of detailed knowledge and understanding in this area, his misled belief that people's eating

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habits cannot be changed, and his unwillingness to spend time instructing the patient, all contribute to the failure of dentistry in recognizing an important disease control.[43]

The one area of investigation in the prevention of dental caries that has yet to produce any direct results is that of direct bacterial elimination. If we subscribe to the premise that caries is basically a bacterial disease, then the ultimate elimination of this disease in the irradiated patient, as in any afflicted patient, will depend on the eradication of the causative microbial agent. "With the current trend toward development of antibiotics that are targeted for specific types of microorganisms, it is possible to expect with some confidence that an antibiotic will be found that will attack the cariogenic microflora specifically, without upsetting the normal balance of oral microflora."[16, 45]

E RESTORATION

It is much easier to prevent dental lesions associated with irradiation than it is to treat them. Careful examination and evaluation, thorough prophylaxis, education of the patient and careful follow-up are mandatory. These procedures take significantly less time than does dealing with post-irradiation caries and its sequela.[15] The dental demineralization associated with irradiation occurs in all

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the teeth and not just those restricted to the primary beam area. However, the most consistent site for these dental lesions is the mandibular incisor region regardless of the site of the exposure.[11,46]

Frank and associates described the clinical appearance of dental lesions, as did Del Regato, in these different groups. The classic type of decay localizes at the cervical region of the tooth and progresses superficially all around the neck of the tooth often resulting in amputation of the crown. In some patients, the teeth become heavily stained brown-black. Most frequently the teeth are effected by generalized superficial defects that occur on the buccal-lingual surfaces and incisal or occlusal edges. This decay process is extremely similar to that found in other severe xerostomia patients. Histologically, radiation caries is similar to other types of caries.[47]

In a report published three years later by Poyton, he described the dental lesions in irradiated patients as a saucer-shaped loss of tooth substance in the cervical area in which the surface was not decalcified the same as in routine caries. He noted that it was not possible to remove tooth with an excavator but that on examination with an explorer, it was noted that the degree of hardness was less than that of a normal tooth.[21]

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In dealing with the initial stages of caries, whether in the irradiated patient or not, the most conservative treatment is the best. In several studies, it has been shown that the early, mild stages of demineralization can be treated successfully with fluoride gels on a routine basis and by frequent follow-ups.[13,16,28] Enamel impregnated with adhesives has also been shown to afford great resistance to the further progress of incipient caries.[33] As mentioned earlier in the paper, fluoride also has been shown to be effective in preventing recurrent caries around metallic restorations when used in treatment of cavity preparations.[35,36]

In the treatment of teeth in which the decay process has advanced, most practioners feel that amalgam is the material of choice.[7,15,22,23] Brekhus reported that not only was amalgam the most commonly used restorative material, but also it was the most effective. He found only a ten percent failure rate.[48] Since that time, others have reported that 42 percent of all amalgam restorations studied were defective.[49] The main reason for amalgam failure is improper cavity preparation (56%), faulty manipulation of material and/or contamination (40%).[50] The inherent problems associated with amalgam restorations are marginal breakdown, fracture, and residual mercury content.[51] Amalgam

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restorations most often have to be replaced because of new marginal caries (58%).[52]

Composite resin materials have been used in anterior restorations for aesthetic purposes in irradiated patients with some success,[15,24] but silicates are definitely contraindicated. The durability of silicate cement is directly related to the pH of the saliva that washes it. The lower the pH, the higher the failure rate. Silicates do the poorest where debris and food plaques collect on the restoration.[52,53,54]

Irradiated patients who present with highly advanced dental lesions should immediately have the disease brought under control by excavating all carious lesions followed by the placement of zinc oxide and eugenol temporaries. Where indicated, endodontic treatment of the teeth should be performed. Those teeth which have lesions too large to restore with amalgam should be restored with full coverage crowns.[19,22] Well fabricated, full coverage crowns, from a non-economic standpoint, are probably the most ideal restoration for irradiated patients. They are the most durable, cover the greatest amount of tooth structure, and are the most tissue compatible of any restoration agents.[19,24]

Darby, in 1884, speaking of restoring radicular caries similar to that seen in the irradiated patient says:

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> "The decay process is not different from that in other areas of the teeth except that decalcification is much slower in teeth of more dense structure. When caries of this character attacks teeth....it does not often arrest itself. Filling does not always arrest it. Absorption of the gums continues and softening around and below the filling follows, and in short time, a larger filling is demanded. There is no class of operations which the writer is called upon to perform, the result of which is less satisfactory than these. For treatment, make the best operations and direct washes of lime water." [55]

It is no wonder then why many practitioners have treated similar dental lesions in irradiated patients by palliation and ultimate extraction or by allowing the remaining decayed root tip to exfoliate on its own.[2,5,10]

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Chapter III

PATIENT MANAGEMENT AT OUR CLINIC

Due to the significant number of patients being treated at this center for head and neck cancer, the Department of Dentistry, Walter Reed Army Medical Center in late 1969, developed a comprehensive program for the dental management of these patients. The main objective was to improve the quality of life for the patient following his cancer treatment. In order to do this, several other treatment objectives had to be met. These were as follow:

1. Eliminate dental conditions that could increase the morbidity of cancer treatment.
2. Reduce the incidence of post-irradiation caries.
3. Educate the patient in his responsibilities in the control of dental disease.
4. Establish an oral environment easily maintainable by the patient.
5. Increase alternatives for prosthetic management after cancer treatment.
6. Improve patient's speech, deglutition, mastication and esthetic function following cancer treatment.

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7. Provide a follow-up program for reinforcement of home care responsibilities and timely recognition of deteriorating dental conditions requiring early treatment.

The dental management of these patients is broken down into three phases. The first phase is diagnosis and treatment prior to cancer therapy. The second phase is supportive treatment carried out during and immediately following cancer therapy. The third phase consists of all treatment and follow-up examinations following cancer therapy.

During phase I, joint treatment planning conferences are held in which all dental problems are explored in depth. After all alternatives are considered, a treatment plan is developed that combines predictability of results with the lowest possible risk. It is at this time also that the caries control program is initiated. Those teeth which are to be maintained are restored in the best possible manner. A thorough dental prophylaxis is accomplished and patients are instructed in good plaque control with subsequent appointments for reinforcement. All patients are supplied with 0.4% SnF_2 gel and instructed in its daily application. Dietary counseling is carried out where the patients are advised as to the hazards of high carbohydrate diets and

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encouraged to eat well-balanced meals.

All other phases of dentistry such as oral surgery, endodontic treatment, periodontal therapy and removable prosthodontic treatment are carried out with the utmost care and bearing in mind that future dental management will be in an irradiated area.

During phase II, dental treatment consists mainly of supportive and adjunctive procedures carried out during radiation therapy. Plaque control and fluoride application highlight this phase of the program.

Phase III management consists of all subsequent definitive dental procedures and routine follow-up examinations with continued reinforcement of plaque control, dietary counseling and fluoride application.

When dental caries occurs, it is managed either by attempting remineralization with SnF_2 gel or by restoration with a suitable restorative material. More severe caries effecting the pulps of the teeth are usually managed by root canal therapy. Non-restorable post-irradiation caries is delt with by meticulous surgical extractions with particular attention paid to adequate alveolplasty, generous wound irrigation, water-tight soft tissue closure and proper antibiotic coverage. At this time, we have no signigicant complications following surgical removal of teeth in

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irradiated patients.

Since the initiation of this program, dental treatment for patients undergoing head and neck cancer therapy has improved. Fewer patients are rendered edentulous with a subsequent decrease in the incidence of post-irradiation caries from 46% to 12%. Periodic follow-up has resulted in earlier recognition of dental disease thus reducing the number of teeth lost following cancer treatment. Some teeth have been removed post-irradiation but without serious sequelae.[56]

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Chapter IV

DISCUSSION & CONCLUSIONS

The term "radiation caries" has been used throughout the years to describe the demineralization of tooth structure which is typically seen in patients who have had radiation treatment to the head and neck. To put the significance of this problem in its proper perspective, one should be aware of some of the basic facts relating to its incidence. First, cancer of the head and neck accounts for not more than five percent of all cancers, depending upon which study you read.[7,20] Of course, cancer is not the only reason for irradiation of this area, but it is by far the major reason. Out of this small percentage of cancer patients, not all will receive irradiation. The decision for the use of this treatment will be made based on many factors, such as type, stage and location of tumor; the patient's general health, age, and acceptance; and the treatment facility available. Then, out of those patients who receive irradiation to the head and neck, the number of those who are even likely to develop dental lesions associated with this treatment depends upon the amount and

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type of irradiation, the location of radiation ports, the period of time over which the treatment was given, and the patient's own physiological factors and dental status (presence of teeth.) In dealing, then, with this fractional number of patients, the problems which are anticipated and may evolve can be managed in a logical manner as presented here. This is not an insignificant problem even though it affects but a few numbers. To the irradiated patient, caries can be a life-threatening situation.

It must be remembered that post-irradiation caries is still a form of dental caries and, as such, requires three basic factors in order to occur - the host, bacteria and substrate.[1,43,44] To remove any one of these three elements would result in the elimination of the disease. The difference in the irradiated patient is that the tooth may be somewhat unfavorably altered due to the direct effect of the radiation. This is controversial and questioned as to its relationship with caries.[57,58] The bacteria have a greater chance for survival because of alteration in the saliva and oral environment. The substrate is generally more conducive to caries activity due to the increased carbohydrate content, soft adhesive nature and lack of fiber content of the diet. Consequently, to protect the irradiated patient against dental caries, we must employ all

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techniques available to combat the great odds against which we have to work.

To extract the teeth eliminates the host, thereby eliminating the disease. However, this increases the morbidity of the treatment because of altered functional ability on the part of the patient in terms of mastication, speech, and social acceptance. Teeth which are found hopeless in any situation should, of course, be removed and those which are left should be restored as indicated. The decision to extract teeth should be based on a combination of factors: the type of cancer treatment the patient is to receive, the patient's predicted prognosis, the overall evaluation of the patient in respect to his ability, mentally and physically, to maintain the teeth following treatment, and the patient's prior dental history. When it is decided by all concerned that the patient who is to undergo radiation therapy shall maintain his teeth, we, the dental profession, must combine all phases of prevention to help give the patient the best prognosis both dentally and medically.

It is agreed that the most important stage of prevention is plaque control. This, in the irradiated patient, can be a difficult procedure due to the secondary effects of irradiation - mucositis and xerostomia. The patient must

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be made aware of the extreme importance of this phase of prevention and encouraged time and time again. Mouthwashes of an alkaline nature should be supplied and the use of them advocated and demonstrated to the patient by a health professional. Strengthening the tooth substance itself is one proven effective means of prevention. This is done most efficiently with the use of fluoride gels applied to the teeth daily. This, of course, makes the tooth more impervious to demineralization.

The irradiated patient's nutrition is altered due to local factors. Frequently, saliva is drastically diminished (depending upon location of irradiation ports and dose), and, consequently, food particles adhere to the teeth and swallowing is difficult. This condition begins during the course of irradiation and persists until the saliva returns to within normal limits, which may be up to several months or possibly never. This problem, in conjunction with mucositis, forces the patient to consume a soft or liquid diet. A diet of this nature naturally lacks the cleansing ability of fiber-type foods, and is usually high in carbohydrate content. Again, patients must be informed of the dangers of high sucrose diets and directed toward foods which are as palatable, but more nutritious and less cariogenic. It is the dental profession's

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responsibility to have this information available and to relate it to the patient or at least refer the patient to a nutritionist who is familiar with the problems at hand.

When our preventive means fail, we must then be prepared to restore the teeth in the best manner possible. Initial caries can often be halted with frequent applications of fluoride or with adhesives. Advanced caries must be treated by removal and restoration. To date, the best material available for restoration of classical post-irradiation caries is amalgam possibly in conjunction with fluoride treatment of the cavity preparation. Full coverage of the clinical crowns with cast restorations is sometimes necessary if too much tooth structure is lost or if the tooth has been treated endodontically. This type of restoration, done properly, is probably the optimum restoration in all irradiated cases if time and economics were not a concern.

Dental lesions affecting the root surfaces of teeth, similar to those described in irradiated patients, have been reported in the literature as early as 1884.[34,55,59] Although it is not within the scope of this paper to discuss the etiology of caries, it is important to realize that caries of this nature occurs when a combination of factors are present. Consequently, the best treatment relies

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on controlling as many of these factors as possible. In order to control these factors, the patient must be made aware of their importance. He must be educated, encouraged and entrusted to do his part in managing his disease.

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Chapter V

SUMMARY

"Radiation caries" has been described as reported in the literature. Although extraction of the teeth prior to irradiation may be one means of preventing caries, it may not necessarily be the best treatment overall. Each tooth needs to be evaluated on its own merit, and those which would have a poor prognosis should be removed. The patient, in general, should be evaluated also, and on this is based the ultimate decision in retaining teeth.

When teeth are retained in the irradiated patient, the key word is "prevention." Our main tools for prevention are plaque control, fluoride, and diet. It is the dental profession's responsibility to provide the information to the patient for maintaining his teeth and entire oral cavity in the healthiest possible condition. When preventive measures fail, restoration and preservation of that which remains with the best methods and materials at hand is essential. Dental caries in the irradiated patient can mean the difference between life and death.

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BIBLIOGRAPHY

1. Kimeldorf,D.J., Jones, D.C. and Castanera, T.J.: The Radiobiology of Teeth. Rad. Res. 20: 518, 1963.

2. Del Regato, J.A.: Dental lesions observed after roentgen therapy in cancer of the buccal cavity, pharynx and larynx. Am.J. of Roentgenol. 42: 404, 1939.

3. Robinson, J.E.: Dental management of the oral effects of radiotherapy. J. Prosth. Dent. 14:582, 1964.

4. Daly, T.E., Drane, J.B. and MacComb, W.S.: Management of problems of the teeth and jaws in patients undergoing irradiation. Amer. J. of Surgery. 124: 539, 1972.

5. Topanziaro, D.S.: Prevention of osteoradionecrosis of the jaws. O.S., O.M. & O.P. 12: 530, 1959.

6. Marciani,R.D. and Plezia, R.A.: Management of teeth in the irradiated patient. J. Amer. Dent. Assoc. 88: 1021, May 1974.

7. Krajicek, D.D.: Oral radiation in prosthodontics. J. Amer. Dent. Assoc. 78: 320, February 1969.

8. King, E.R., Elizay, R.P. and Dettman, P.M.: Effects of ionizing radiation in human oral cavity and

Digitized by Google

oropharynx. Radiology. 91: 1001, 1968.

9. Elizay, R.P., King, E.R. and Dettman, P.M.: Dental prosthesis and relation to the jaws: A survey of prosthodontists and radiotherapists. J. Amer. Dent. Assoc. 77 (4): 856, 1968.

10. Wildermuth, O and Cantril, S.T.: Radiation necrosis of the mandible. Radiology. 61: 771, 1953.

11. Hayward, J.R. et. al.: The management of teeth related to the treatment of oral cancer. Cancer - A Journal for Clinicians. 19 (2): 98,1969.

12. MacComb, W.S.: Necrosis in treatment of intraoral cancer by radiation therapy. Am. J. Of Roentgenol. 87: 431, 1962.

13. Rahn, A.O. and Drane, J.B.: Dental aspects of the problems, care and treatment of the irradiated oral cancer patient. J. of Amer. Dent. Assoc. 74:957,1967.

14. Coffin, F.: Management of radiation caries. Brit. J. of O.S. 11: 54, 1973.

15. Daly, T.E. and Drane, J.B.: Management of dental problems in irradiated patients. Refresher Course: The Radiological Society of North Am. Nov. 26, 1972.

16. Massler, M.: Changing concepts in prevention and treatment of dental caries. Univ. of Ill. at the Med. Center, 1968.

Digitized by Google

17. La Dow, C.S.: Osteoradionecrosis of the jaw. Oral Surg. 3: 582, 1950.

18. Wescott, W.B., Starcke, E.N. and Shannon, O.L.:Chemical protection against port-irradiation dental caries: case reports. Oral Disease Research Lab,Veterans Admin. Hosp., 1974.

19. Frisch, J. and Sproull, R.C.: Dental treatment after irradiation. J. Of Prosth. Dent. 12: 182, 1962.

20. Bascom, P.: Oral cancer and prosthodontics. J. of Prosth. Dent. 19 (2): 164, 1968.

21. Poyton, H.G.: The effects of radiation on teeth. O.S., O.P. & O.M. 26 (5): 639, 1968.

22. Carl, W., Schaaf, N.G. and Chen, T.Y.: Oral care of patients irradiated for cancer of the head and neck. Cancer. 30: 448, August, 1972.

23. Hinds, E.C.: Dental care and oral hygiene before and after treatment radiation caries. J. Amer. Med. Assoc. 215 (6): 964, 1971.

24. Personal experiences.

25. Ericsson, Y.: The chemistry of the enamel-saliva interface. Pla. J. Med. Sci. 5: 256, 1968.

26. Navia, J.M.: Prevention of dental caries. Int. Dent. J. 22 (4): 427, 1972.

27. Englander, H.R., Keys, P.H., Gestwicki, M. and Sultz,

Digitized by Google

H.A.: Chemical anticaries effect of repeated topical sodium fluoride applications by mouthpieces. J. Amer. Dent. Assoc. 75: 638, September 1967.

28. Cole, W.L. and Stern. M.H.: Control of radiation-induced demineralization of human dentition with topical fluoride gel and enamelite. J. Dent. Res. 52 (Special Issue): 120, February 1973.

29. Keyes, P. and Englander, H.R.: Fluoride therapy in the treatment of dentomicrobial plaque diseases. J. Of Prev. Dent. (JASPD): Jan-Feb., 1975.

30. Cowan, R.D. and Shannon, I.L.: Protective effectiveness of a stannous fluoride gel. Aust. Dent. J. 17: 293, 1972.

31. Fischer, E.E.: Inhibition of enamel demineralization by repeated treatments with sodium and stannous fluoride solutions. J. Dent. Res. 41: 392, 1962.

32. Proc. Finn. Dent. Soc. 69: 202, 1973.

33. Buonocore, M.G.: Adhesives in the protection of caries. J. Amer. Dent. Assoc. 87 (Special Issue): 1000, 1973.

34. Brudevold, F., McCann, H.G., Nilsson, R., Richardson,B. and Coklica, V.: The chemistry of caries inhibition problems and challenges in topical treatments.

Digitized by Google

J. Dent. Res. 46: 37, 1967.

35. Alexander, W.E., McDonald, R.E. and Stookey, G.K.: Effects of Stannous Fluoride on recurrent caries. Results after 24 months. J. Dent. Res. 52 (5): 1147, 1973.

36. Shannon, I.L., Uribe, T.R. and Wightman, J.R.: Topical treatment of prepared cavities. Tex.Dent. J. 91: 6 December 1973.

37. Ingram, G.S.: Some factors affecting the interaction of hydroxyapatite with sodium monofluorophosphate. Caries Res. 7: 315, 1973.

38. Chow, L.C. and Brown, W.E.: Formation of $CaHPO_4 \cdot 2H_2O$ in tooth enamel as an intermediate product in topical fluoride treatments. J.Dent. Res. 54 (1): 65, 1975.

39. Levine, R.S. and Rowles, S.L.: Further studies on the remineralization of human carious dentin in vitro. Archs. Oral Biol. 18: 1351, 1973.

40. McBean, L.D. and Speckmann, E.W.: A review: The importance of nutrition in oral health. J. Amer. Dent. Assoc. 89: 109, 1974.

41. Masters, D.H. and Lewis, H.: The sour side of sugar. JASPD: 23, Jan-Feb. 1975.

42. Shannon, I.L. and Edmonds, E.T.: High sucrose snacks:

Digitized by Google

are they doubly dangerous? Tex. Dent. J. 91: 8, August 1973.

43. Truuvert, M.: Diet Counseling to prevent caries. Cana. Dent. Hygienist 7(2): 27, 1973.

44. Navia, J.M.: Prospects for prevention of dental caries: dietary factors. J. Amer. Dent. Assoc. 87 (Special Issue): 1010, 1973.

45. Fitzgerald, R.J.: The Potential of Antibiotics as Caries-control agents. J. Amer. Dent. Assoc. 87: 1006, 1973 (Special Issue).

46. Cernea, M.M.P. and Batailk: Les alterations dentaires provoquées por la radiothérapie ou la curietherapie de la region cervicale. Rev. Stomalol 48: 509, 1947.

47. Frank, R.M., Herdly, J. and Phillppe, E.: Acquired dental defects and salivary gland lesions after irradiation for carcinoma. J. Amer. Dent. Assoc. 70: 868, 1965.

48. Brekhus, P.J.: Your teeth. Minneapolis, The Univ. of Minn. Press: 36,1941.

49. Moore, D.L., and Stewart, J.L.: Prevalence of defective dental restorations. J. Pros. Dent. 17: 372,1967.

50. Healey, H.J. and Phillips, R.W.: A clinical study of amalgam failures. J. Dent. Res. 28:439, 1949.

51. Nadal, R.: Amalgam restorations: cavity preparation

Digitized by Google

condensing and finishing. J. Amer. Dent. Assoc. 65: 66, 1962.

52. Barnes, G.P., Carter, H.G. and Hall, J.B.: Causative factors in the replacement of dental restorations: a survey of 8891 restorations. Milt. Med. 138: 736, 1973.

53. Wilson, A.D. and Batchelor, R.F.: Dental silicate cements III: environment and durability. J. Dent. Res. 47: 115, 1968.

54. Henschell, C.J.: Observations concerning in vivo disintegration of silicate cement restorations. J. Dent. Res. 28: 528, 1947.

55. Darby, E.T.: The etiology of caries at the gum-margins and the labial and buccal surfaces of the teeth. Dent. Cosmos. 26: 218, 1884.

56. McCasland, J.P.: Dental considerations in head and neck patients. Management of dental and jaw problems. Walter Reed Army Medical Center, Washington, D.C. 20012 (unpublished.)

57. Jerve, P.: X-ray diffraction investigation on the effect of experimental and in-situ radiation on mature human teeth. ACTA Odont. Scand. 28 (5): 623, November 1970.

58. Colby, R.A.: Radiation effects on structures of the oral cavity: a review. J. Amer. Dent. Assoc.

Digitized by Google

29: 1446, 1942.

59. Schamschula, R.G., Keyes, P. H. and Hornabrock, R.W.: Root surface caries in Lufa, New Guinea: clinical observations. J. Amer. Dent. Assoc. 85: 603, 1972.

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AN ANALYSIS OF THE EFFECT OF CHANGING THE VERTICAL DIMENSION OF OCCLUSION ON THE PRESSURES EXERTED BENEATH COMPLETE MAXILLARY DENTURES

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CHAPTER I

REVIEW OF THE LITERATURE

Force application to complete dentures and its results have been widely studied using various approaches. Ledley in a study published in 1968 states that, during function, food exerts an applied force to the teeth of complete dentures; this force causes the denture to be displaced slightly into the "soft" mucosal tissue that supports the denture.[1] Studies by Howell and Brudevold showed that a maximal force of 7.2kg could be exerted against the first molar tooth of a complete denture when chewing raisins.[2] Ohashi, Woelfel and Paffenbarger in a study containing 21 subjects showed that as much as 15.8 kg of pressure could be exerted centrally using a modified Coble balancer plate in the maxillary denture. The bearing area of the force was found to be from 22.8 to 36.6 cm^2 in the maxillary denture.[3] Thomson in a separate study found the area that primarily resists force in a maxillary denture to be a mean of 22.96 cm^2.[4] Yurkartas and Curley found that the maximum force required to masticate food was 12kg, the average being .3 to 1.8kg.[5] However, the relationship of the vertical dimension of occlusion to the force applied to specific sites beneath complete maxillary dentures has not been fully investigated.

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Most studies concern the amount of pressure exerted at the interface between the maxillary denture and the mandibular denture or natural teeth. Tueller showed in an investigation involving 40 subjects that there was only a difference of .5mm when comparing the vertical dimension of occlusion established by maximum mandibular force with the vertical dimension of occlusion established by pre-extraction records, phonetic and anatomic measurements.[6] The effect of increasing or decreasing the vertical dimension of occlusion within limits in order to alter the magnitude of force exerted to the tissues beneath complete dentures is still questionable. It has been suggested by some authors that reducing inter arch distance in complete denture patients reduces biting forces and thereby reduces soreness and trauma.[7] Furthermore, it has been written that if the opposing teeth are the least bit too far apart, muscular contraction causes a destruction of the supporting structures.[8] When evaluating this concept, what is the role of magnitude versus duration of force? Is there a range within which the vertical dimension of occlusion can be changed without changing the pressure applied to the supporting tissues? If so, how much change is possible before the pressure is altered? Is there a direct relationship between the pressure applied to the tissues beneath complete

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dentures and the alteration of the vertical dimension of occlusion?

These questions and others prompted the author to design an experiment to establish whether or not increases or decreases from a selected vertical dimension of occlusion can be related to the amount of pressure applied to the tissues beneath complete maxillary dentures.

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CHAPTER II

METHODOLOGY

The relationship of the pressures exerted beneath complete maxillary dentures to vertical dimension of occlusion was measured using three patients, two males and one female. The age range was from 30 for one of the males, to 67 for the female. The duration of denture wearing was from two months, for one of the males, to 25 years for the female. All three subjects wore both maxillary and mandibular complete dentures with monoplane posterior teeth. The patients were chosen primarily on the basis of health, dependability, and availability with no regard to age, sex, palatal form or any other physiological criteria.

Primary impressions were made of the maxillary arch of each subject using an irreversable hydrocolloid impression material* after edentulous trays were border molded with utility wax. After one layer of baseplate wax was applied

* Jeltrate - Type II Normal Set, L.D. Caulk Co.

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to the primary casts for relief, custom trays were fabricated using autopolymerizing acrylic tray material.* These trays were checked intraorally for adaptation and red modeling plastic** added to the tissue surface of the trays. This was flamed and tempered in a water bath at 132° for 10 seconds and then inserted into the mouth. The subject was instructed to carry out some movements of the muscles to insure a proper extension of the modeling plastic impression. After one minute the impression was removed and checked for accuracy. After the author was satisfied, the borders were relieved 2mm. Green modeling plastic*** was added in small increments to the borders and inserted after flaming and tempering. Physiologic movements of the musculature by the subject were used to accurately mold the borders of the impression. Finally,a thin layer of zinc oxide-eugenol impression paste**** was added as a wash material and the impression reinserted. After the paste had set the impression was removed, checked for accuracy, cleaned of saliva, and poured in improved stone after boxing was accomplished.

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* Fastray, Harry T. Bosworth Co.

** Kerr Mfg. Co.

*** Kerr Mfg. Co.

**** CoeFlo, Coe laboratories

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It was decided that denture bases with interchangeable occlusal tables should be used for each subject to reduce variables. Metal bases of Ticonium 100* were fabricated with a triangular key extending from first molar area to first molar area. After fitting the base to the master cast, auto-polymerizing acrylic** was added to the peripheries and post-dam areas. Wax cones of of impression wax*** were placed in the first molar areas of the maxillary denture base and the closest speaking space recorded by having the subject pronounce a series of sibilant containing words and phrases. The wax cones, which had been previously tempered in a water bath, were flattened at the closest speaking space between the maxillary base and the mandibular denture of the subject. The wax cones were chilled and the maxillary base reinserted. Small scratch marks on the maxillary base and the mandibular denture were made and the closest speaking space measured intraorally using calipers. This procedure was repeated until three measurements fell within $\frac{1}{5}$mm of each other.

* Ticonium Division, CMP Industries, Inc.

** Speed-liner, L. D. Caulk Co.

*** Kerr Mfg. Co.

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The tentative vertical dimension of occlusion was established at a position 1mm closed from the recorded speaking space and checked for esthetics. One criteria for acceptable intraoral records is that they be repeatable and this method, as claimed, seems to be.[9]

Next, a centric jaw relation record was made utilizing green stick modeling plastic.* A facebow transfer was made and the maxillary base on a remount cast was mounted on a semiadjustable articulator.** An impression of the subject's mandibular denture was made using irreverseable hydrocolloid impression material and a mounting cast poured in improved stone.*** The mandibular cast was mounted using the centric jaw relation record.

One millimeter holes were cut through the base over the crest of the ridge in the second bicusped areas. Next, holes were cut one centemeter lingual to the incisive papilla on the anterior slope of the palate and one centemeter anterior to the posterior extent of the base. Both palatal holes were located in the midline of the palate. After all the holes were prepared, four 18 ga needles were contoured to fit the

* Kerr Mfg. Co.

** Model H2, Hanau Co.

*** Silky Rock, Whip-Mix Corp.

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outer palatal slope of the base. Each needle was inserted into its respective hole, the hubs brought together anteriorly and held in place with sticky wax.* Clear autopolymerizing acrylic** was applied the length of the needles and over the holes to seal them. Base plate wax was adapted over the metal key on the base and monoplane teeth set against the cast of the opposing denture at the selected vertical dimension of occlusion. No anterior teeth were set so that ample room would be present for the needles to exit anteriorly. The vertical dimension was opened 5mm and closed 5mm at the pin from the selected vertical dimension of occlusion and posterior teeth set for each of these positions. Screw holes in the metal base lined with autopolymerizing acrylic and self threading screws were used to hold the occlusal tables after they were processed with heat cured acrylic resin.***

After processing, the teeth which were set at the selected vertical dimension of occlusion were screwed to place on the metal base. A new centric jaw relation record was made and the subject's mandibular denture mounted to the maxillary test denture. The occlusion was adjusted for processing error.

* S.S. White Mfg. Co.
** L.D. Caulk Co.
*** Duraflo, Myerson Co.

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Subsequently, each of the other two occlusal tables was screwed to place and adjusted.

A Sanborn 350 series recorder* was utilized to record positive and negative pressures at the four test sites. The recording was done through a #267 Sanborn carrier physiologic pressure transducer as in a previous study in which the author participated.[10] Hoses were attached to each of the needle hubs and attached at the opposite ends to the pressure transducers. After seating the test denture in the subject's mouth, room temperature water was introduced to evacuate all air from the system and form a closed hydraulic system. The subject was instructed to close firmly on cotton rolls placed in the second bicuspid areas while the recorder was calibrated. This procedure required a minimum of 15 minutes in each case.

Peanuts were used as a test food for all subjects and each subject was instructed to chew bilaterally simultaneously to avoid tipping the denture thus creating a leak in the system. Recordings were made at each of the three vertical openings for each subject.

* Sanborn Co.

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Chapter III

RESULTS

Measurements were made of twelve complete chewing cycles for each subject at each of the three vertical positions. These were recorded in millimeters of mercury per centemeter. Next, they were converted to pounds per square inch and will, for the remainder of this paper, be referred to in this manner. Though all measurements of pressure will be referred to in the paper in terms of pounds per square inch, it should be understood that one pound equals 1/2.20 kilograms. This is mentioned for comparison with previous studies.

The phrase, " selected vertical dimension of occlusion", will be abbreviated SVDO and will refer to that vertical dimension of occlusion determined to be suitable for use in constructing the subject's complete dentures. The highest and lowest single values for each test site at each vertical dimension were omitted and the rest used to obtain average values.

Subject One

Subject one was a thirty year old male who had been

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wearing complete dentures for ten years. The highest average positive pressure recorded in the test site over the left ridge was .82. This average value was obtained at a vertical dimension of occlusion increased 5mm from the subject's SVDO. This was an increase of .15 when compared to the average positive pressure recorded at the same site at the subject's SVDO.

The highest average positive pressure recorded at the test site over the right ridge was 1.31. This was also recorded at a vertical dimension of occlusion increased 5mm from the SVDO. This value was .85 greater than the average positive pressure value recorded at the SVDO.

The average value for positive pressure at the test site in the anterior palate was again greatest at an increased vertical dimension of 5mm. This value was .51 greater than that recorded at the subject's SVDO.

Average positive pressure values recorded at the test site in the posterior palate were greater at each vertical dimension tested than those recorded at the other test sites. The highest average positive pressure value in the posterior palate was 3.61 and was recorded at a vertical dimension of occlusion increased 5mm from the SVDO.

The greatest single positive pressures recorded were:

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1.90 for the left ridge, 2.48 for the right ridge, 2.28 for the anterior palate and 5.7 for the posterior palate. All of these were recorded at an opening 5mm from the subject's SVDO. The greatest ranges, the differences between the greatest single positive and negative pressures, were also recorded at the increased vertical dimension of occlusion.

Generally, the average positive pressures at three of the test sites which were recorded at a position 5mm closed from the subject's SVDO varied little from the averages at the same sites at the subject's SVDO. One test site, the left ridge, was not included. No recording could be obtained at the decreased vertical dimension. The test was repeated with the same result. The subject was rescheduled for testing but was lost to the study.

Average negative pressures were calculated in the same manner as average positive pressures. The ranges were computed for each test site at each vertical dimension of occlusion. The highest range value recorded in subject one was in the posterior palate at a vertical dimension of occlusion increased 5mm from the SVDO. This value was 6.46. The highest ranges recorded for each of the other sites were 2.09, 3.05, and 2.47 for the left ridge, right ridge, and anterior palate respectively. These were recorded also at the increased

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vertical dimension of occlusion.

Subject Two

Subject two was a 43 year old male. The highest average positive pressure recorded over the left ridge was 1.30 while the lowest was .48. The higher average was recorded at the subject's SVDO. The difference in the averages was .82.

The highest average positive pressure recorded over the right ridge was 2.61, the lowest .59. The higher value was recorded at the SVDO, the lower 5mm closed from the SVDO. The difference in the two averages was 2.02.

The highest average positive pressure in the anterior palate was .66 and the lowest .09. The highest average was recorded at the SVDO, the lowest at an opening increased 5mm from the SVDO.

The computed average positive pressure of 1.33 was the greatest average recorded in the posterior palate and was recorded at the subject's SVDO. The lowest average positive pressure value recorded in the posterior palate of subject two was .70. This was recorded at a position 5mm closed from the SVDO.

The highest average positive pressure recorded anywhere was 2.61 and was recorded over the right ridge at the subject's

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SVDO. The least positive pressure average recorded anywhere was .09 and was recorded in the anterior palate at a position 5mm greater than the SVDO.

The greatest single positive pressures recorded were: 1.90 for the left ridge, 3.04 for the right ridge, .95 for the anterior palate, and 1.90 for the posterior palate. These were all recorded at the subject's SVDO.

In subject two, all of the high average and single values, both positive and negative, were recorded at the SVDO. In all except the anterior palate all of the low values were recorded at a position closed 5mm from the subject's SVDO.

The largest ranges recorded for subject two were recorded at the SVDO. These were 2.47, 4.94, 1.43, and 3.04 for the left ridge, right ridge, anterior palate, and posterior palate respectively.

Subject three

Subject three was a 67 year old female who had worn complete dentures for 25 years. The highest average positive pressure over the left ridge was only .29 pounds per square inch while the lowest was .11. The higher average positive pressure was recorded at the subject's SVDO, the lower value at both 5mm open from and 5mm closed from the SVDO. The difference in the averages was .18.

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The highest average positive pressure recorded over the right ridge was .57, the lowest .48. The higher average value was recorded at the SVDO, the lower one at both 5mm open from and 5mm closed from the SVDO. The difference between the high and low averages was small, being only .07 pounds per square inch.

The highest positive pressure average value in the anterior palate was only .10 pounds per square inch. The lowest was also .10 and the same average values were recorded at all three vertical openings, indicating little activity at the test site in the subject's anterior palate. The greatest positive pressure average value recorded in the posterior palate was .38 and was recorded at the subject's SVDO. The lowest average positive pressure value recorded in the posterior palate was .25 and was recorded at a position 5mm open from the subject's SVDO.

In testing subject three, the highest average positive pressure recorded anywhere was .57 and was recorded over the right ridge at the subject's SVDO. The least positive pressure average recorded anywhere was .10. This was recorded at all three vertical openings in the anterior palate.

The greatest single positive pressures recorded at the four test sites were: .38 for the left ridge, .86 for the right ridge, .10 for the anterior palate, and .48 for the

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posterior palate. The values for the left and right ridges and the posterior palate were recorded at the subject's SVDO. The values for the anterior palate did not change when the vertical dimension of occlusion was altered.

In subject three, all of the high values, average and single, positive and negative, were recorded at the subject's SVDO with the exception of the negative values in the posterior palate. They were greatest at an opening 5mm greater than the SVDO.

The highest values for the negative pressures were generally recorded at the subject's SVDO. This was true except for the posterior palate where the highest average and single values were recorded at a position increased 5mm from the SVDO. The largest ranges were also recorded at the SVDO. The highest ranges were: .57 for the left ridge, 1.81 for the right ridge, .20 for the anterior palate, and .56 for the posterior palate.

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Chapter IV

DISCUSSION

At the outset of this experiment, the author realized that the correct or optimal vertical dimension of occlusion to be used for a particular complete denture patient is subjective and emperical. There are guides and techniques but in the final analysis, that vertical opening to be used depends on the eyes and experience of the operator.

This study did not take into account denture experience, subject age, basalar bone relationships, palatal form, or the quality or quantity of the bone making up the ridges of the subjects. The only common denominator was that all subjects wore complete maxillary and mandibular dentures with monoplane teeth set on a flat plane. Subject one had a narrow, tapering arch form with a thin maxillary ridge and a "v" shaped palatal vault. The posterior palatal test site was immediately below the apex of the "V". Subject one also had a class III jaw relationship. Subject two had a much broader, ovoid shaped arch with full, rounded ridges and a class I jaw relationship. Subjects one and two were in the same general age group being 30 and 43 respectively. Subject three had a ovoid shaped arch with moderately well formed

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ridges and a flatter palatal form than the other two. She was 67 years old and had a class II jaw relationship. In retrospect, I believe these factors should be considered and subjects with as many similarities as possible including all of the previously mentioned ones should be selected for study. All of these play a role in the patient's ability to function with his dentures.

One inconsistancy noted in the results obtained from subject one was that he was able to exert more positive pressure in all areas at a vertical dimension of occlusion increased 5mm from the one that had been determined best for him using the technique described in the methodology. It is quite possible that he would have been more efficient and tolerated a vertical dimension of occlusion slightly increased from the one selected for his denture fabrication. He functioned adequately, spoke well, and looked acceptable with the SVDO but may have tolerated an increase. It is interesting to note that the values recorded at the increased vertical dimension of occlusion compared favorably with that of subject two at his SVDO at all test sites.

The subjects did not apply forces to their ridges which were equal even when instructed to chew bilaterally simultaneously. The hardness of the test food, peanuts, may have had something to do with this. However, patients, without

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exception, admit to me that this is unnatural and very difficult for them to do. I believe that we must still have our patients attempt this in order that a situation for equal force distribution will exist to some degree. Subject one applied more average positive pressure to the left ridge than to the right at the SVDO. However, he applied more pressure to the right ridge at an increased vertical dimension of occlusion. Subject two consistantly applied more pressure to the right ridge than to the left at each of the vertical positions used. At his SVDO, he applied more than twice the amount of average positive pressure to the right ridge than to the left. Subject three also applied more pressure to the right ridge at all three of the vertical openings. When questioned as to having a preferred chewing side, subject one declared none while two and three preferred the right side. This seems to be borne out by the data.

The fact that all three subjects recorded generally low positive pressue values in the anterior palate was an interesting finding. While the anterior component of force does exist in complete denture patients, its destructive pressure on the anterior slope of the palate may be less than previously thought.

There was a great deal of activity in the center of the posterior palate of all subjects when compared to the other

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test sites. Activity at the posterior palatal test site seemed to increase from subject to subject with the increasing taper of the palatal vault. This would seem to indicate that as the palatal slope increases, more pressure is applied at the apex of the vault, whereas the pressure is more evenly distributed over a broader, flatter palate.

The data collected seems to indicate that the subjects were able to exert less pressure in all areas tested when the vertical dimension of occlusion was reduced 5mm from the SVDO. If one considers that subject one's SVDO could have been 5mm greater than the one used, all three subjects demonstrated this finding. This is especially significant when we consider that a 5mm reduction at the pin of an articulator translates into about a 2mm reduction in the molar region. It is also interesting to note that two of the three subjects demonstrated a decrease in pressures exerted to the underlying tissues when the vertical dimension of occlusion was increased 5mm. On the basis of the data collected from these three subjects, the SVDO for each subject as determined by the technique described seems to be sensitive to changes as small as 5mm. With 5mm increases or decreases the pressures that the subjects exerted were generally less than those recorded at the SVDO. Reducing the vertical dimension of occlusion would, as stated in the premise, not only reduce the

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duration of pressure by allowing the patient to keep his teeth apart in a rest position, but the magnitude of pressure as well.

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Chapter V

CONCLUSIONS

1. Measurement of pressures at specific sites beneath complete maxillary dentures is possible using the Sanborn 350 recorder.

2. There is often negative pressure which follows the production of positive pressure at the same site beneath a denture.

3. Jaw relationships, denture experience, palatal form, and quality and quantity of the bony ridges are important factors when one considers the pressures that a patient is able to exert at specific sites beneath a denture.

4. Patients do not, even when instructed, apply pressures evenly to their ridges when masticating hard food such as peanuts.

5. The anterior component of force, though present, does not seem to exert great amounts of pressure to the anterior slope of the palate.

6. On the basis of data collected from three subjects, a change of 5mm from the SVDO alters the subject's ability to apply pressures to the underlying tissues.

7. More investigation in this area is needed utilizing greater numbers of subjects with as many simililarities as possible.

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Chapter VI

SUMMARY

An experiment was designed utilizing three subjects to attempt to analyze the effect of changing the vertical dimension of occlusion on pressures exerted at four test sites beneath complete maxillary dentures. On the basis of the data collected from these subjects, it was determined that one could measure pressures beneath maxillary dentures using a Sanborn 350 recorder. It was confirmed, as in previous experiments, that positive and negative pressures are produced at the same sites during mastication. These subjects demonstrated that patients do not exert equal pressures to both ridges when attempting to chew bilaterally simultaneously. They further demonstrated that there are many variables between subjects to be considered when studying the pressures that they are able to produce with their dentures. The anterior component of force, though present, did not seem to exert great pressure on the anterior slope of the palate. Based on this experiment, a change of as little as 5mm can effect the amount of pressure exerted beneath complete dentures. More subjects

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should be included and further investigation accomplished in this area.

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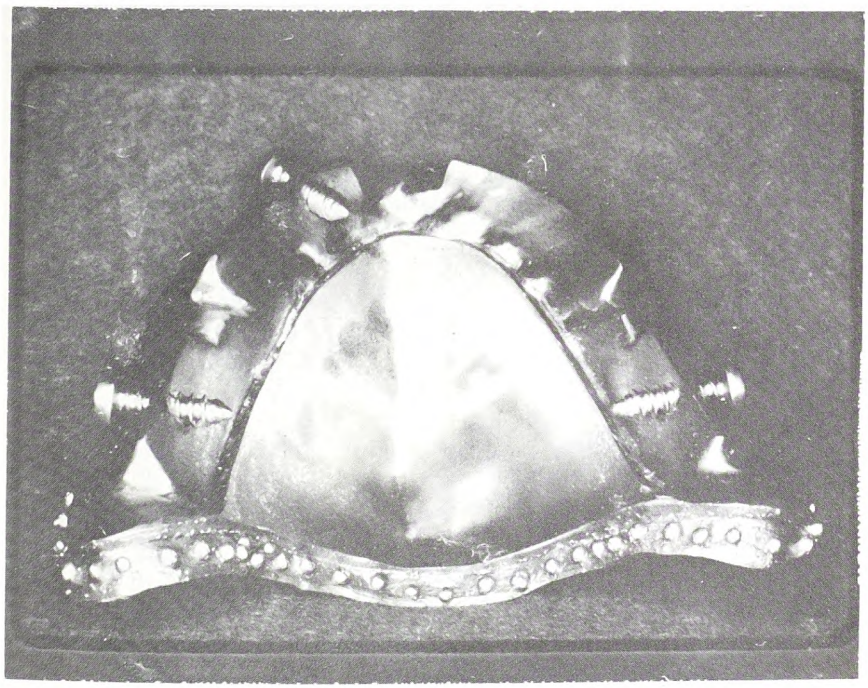

FIGURE 1. Metal base of test denture prior to placing occlusal table.

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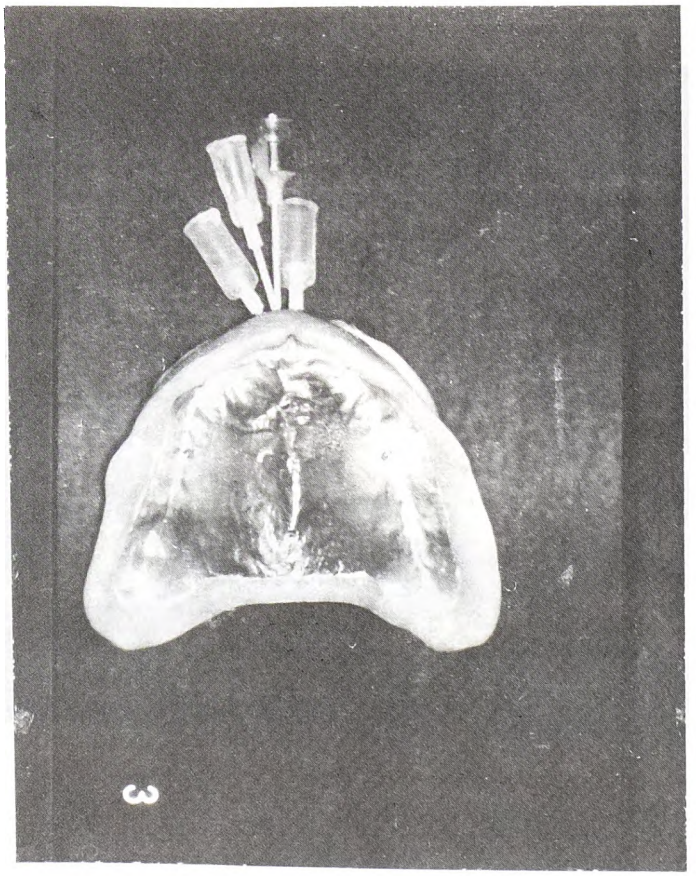

FIGURE 2. Tissue side of test denture showing the four test sites.

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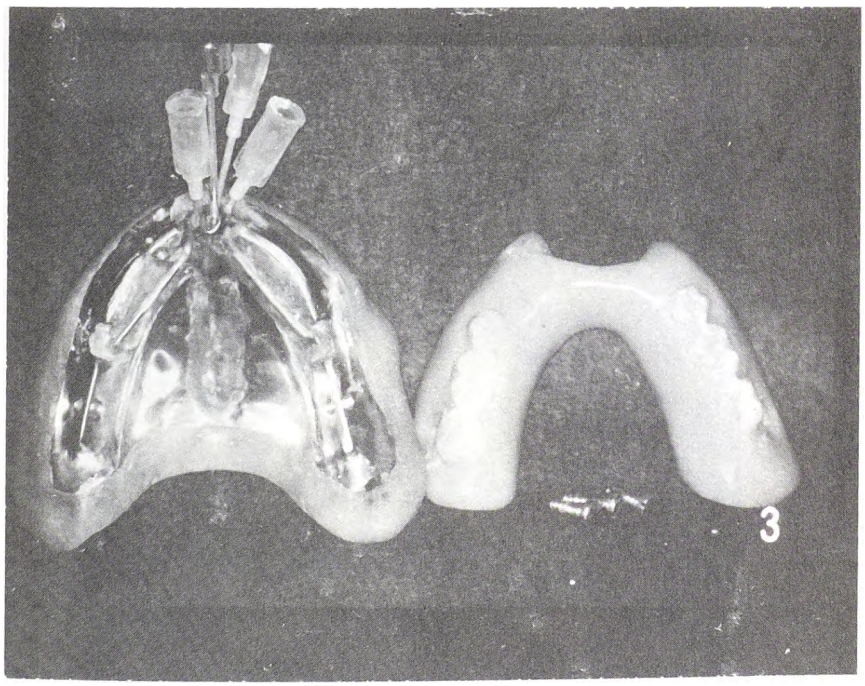

FIGURE 3. Test denture with occlusal table removed.

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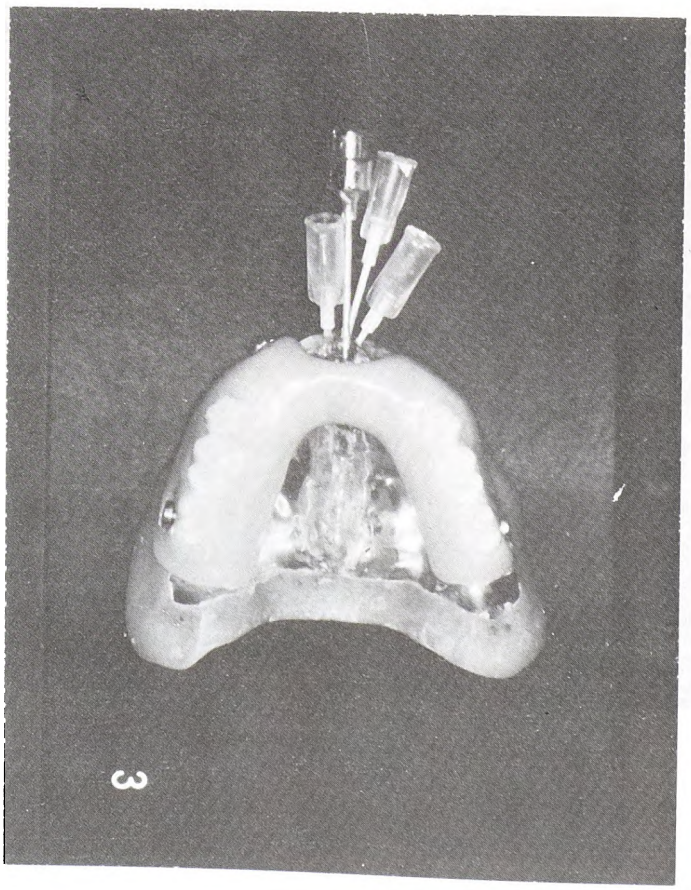

FIGURE 4. Test denture with occlusal table in place.

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TABLE I

Average and Single Values Recorded for Subject One*

Left Ridge	SVDO	+5	-5
Average + pressure	.67	.82	---
High + pressure	.86	1.90	---
High - pressure	.19	.19	---
Average - pressure	.15	.11	---
Range	1.05	2.09	---
Right Ridge			
Average + pressure	.46	1.31	.46
High + pressure	.57	2.48	.57
High - pressure	.19	.57	.57
Average - pressure	.17	.17	.15
Range	.76	3.05	.76
Anterior Palate			
Average + pressure	.61	1.12	.63
High + pressure	.86	2.28	.95
High - pressure	.19	.19	.19
Average - pressure	.11	.15	.13
Range	1.05	2.47	1.14
Posterior Palate			
Average + pressure	1.24	3.61	.89
High + pressure	1.71	5.70	1.33
High - pressure	.38	.76	.38
Average - pressure	.20	.44	.32
Range	2.09	6.46	1.71

* In pounds per square inch

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Table II

Average and Single Values Recorded for Subject Two*

Left Ridge	SVDO	+5	-5
Average + pressure	1.30	.76	.48
High + pressure	1.90	1.80	1.04
High - pressure	.57	.38	.29
Average - pressure	1.34	.24	.17
Range	2.47	2.18	1.33
Right Ridge			
Average + pressure	2.61	1.28	.59
High + pressure	3.04	1.61	.95
High - pressure	1.90	1.12	.57
Average - pressure	1.44	1.04	.44
Range	4.94	2.73	1.52
Anterior Palate			
Average + pressure	.66	.09	.59
High + pressure	.95	.09	.76
High - pressure	.48	.19	.38
Average - pressure	.15	.09	.15
Range	1.43	.28	1.14
Posterior Palate			
Average + pressure	1.33	1.23	.70
High + pressure	1.90	1.80	1.14
High - pressure	1.14	.95	.57
Average - pressure	.93	.55	.36
Range	3.04	2.75	2.71

* In pounds per square inch

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Table III

Average and Single Values Recorded for Subject Three*

Left Ridge	SVDO	+5	-5
Average + pressure	.29	.11	.11
High + pressure	.38	.19	.13
High - pressure	.19	.10	.10
Average - pressure	.11	.10	.10
Range	.57	.29	.23
Right Ridge			
Average + pressure	.57	.48	.48
High - pressure	.86	.57	.57
High - pressure	.95	.29	.29
Average - pressure	.76	.15	.20
Range	1.81	.86	.86
Anterior Palate			
Average + pressure	.10	.10	.10
High + pressure	.10	.10	.10
High - pressure	.10	.10	.10
Average - pressure	.10	.10	.10
Range	.20	.20	.20
Posterior Palate			
Average + pressure	.38	.25	.29
High + pressure	.48	.38	.29
High - pressure	.08	.29	.29
Average - pressure	.10	.19	.01
Range	.56	.67	.58

* In Pounds per square inch

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BIBLIOGRAPHY

1. Ledley, Robert S.: Theoretical Analysis of Displacement and Force Distribution for the Tissue-Bearing Surface of Dentures. J. Dent. Res., 47,318-22, 1968.

2. Howell, A.H. and Brudevold, F.: Vertical Forces Used During Chewing Food. J. Dent. Res., 29,133-36, 1950.

3. Ohashi, M. and Woelfel, J B. and Paffenbarger, G.: Pressures Exerted On Complete Dentures During Swallowing. J. Amer Dent Assoc., 73,625-30, 1966.

4. Thomson, J.C.: The Load Factors In Complete Denture Intolerance. J. Pros Dent., 25,4-11, 1971.

5. Yurkstas, B.S. and Curley, W.A.: Force Analysis Of Prosthetic Appliances. J. Pros. Dent., 22,284-88, 1969.

7. Swenson, Merrill G.: Vertical Relations. Swenson's Complete Dentures., 155, 1964.

8. Sears, Victor H.: Jaw Relation Records and Their Recording. Principles and Technics For Complete Denture Construction., 178, 1949.

9. Cutright, D.E , Brudvik, J.S., Gay, W.D., and Selting, W.J.: Tissue Pressure Ranges Produced Beneath Complete Dentures. Currently unpublished.

10. McCasland, J.P.: Vertical Dimension. Resident and Staff Lectures, Dental Service. Fitzsimmons Gen. Hosp., 1966.

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THE PLAQUE REMOVING ABILITY OF A PROTOTYPE PORTABLE ROTARY PROPHYLAXIS INSTRUMENT AS COMPARED TO MANUAL BRUSHING*

by

MICHAEL D. ALLEN, MAJOR, DENTAL CORPS, USA

Professional paper submitted in partial fulfillment of the requirements of the U.S. Army Dental Residency Program, Walter Reed General Hospital, Walter Reed Army Medical Center, Washington, D.C. 20012

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Chapter I

Review of the Literature

Numerous researchers, utilizing a wide range of subjects and methods have clearly demonstrated a significant relationship between inadequate oral hygiene and the incidence of periodontal disease.[1-6] The principle causative factor is bacterial plaque, and as a result, many instruments and techniques have been devised to facilitate plaque removal by the patient.[7-16] Unfortunately, many of the instruments and techniques designed for this procedure require a relatively high degree of manual dexterity and consequently produce poor results. The tooth surfaces adjacent to the gingival margin and sulcus are the anatomical sites in which inadequate plaque removal is most frequently observed.[1,9,17-20] It is, therefore, essential to provide patients with a simple and effective method of plaque removal in these areas in order to maintain an optimal level of periodontal health.

The dentist and hygienist both utilize the prophylaxis handpiece and rubber cup to obtain a smooth, plaque-free surface during in-office cleaning procedures.

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The use of a portable, hand-held, rotary prophylaxis device has been reported by Levin[8] to provide improved plaque removal when used on a one-time supervised basis, as compared with manual brushing techniques.

The purpose of this study was to test the unsupervised plaque removing efficiency and gingival effects of a prototype, portable, rotary cleaning instrument over a period of four weeks in adult volunteers.

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Chapter II

Materials and Methods

A single blind study was undertaken utilizing eleven adult volunteers, ranging in age from eighteen to forty-three years. The average age was twenty-six years. Prior to the study all potential test subjects were subjected to a complete dental examination. Those individuals found to have clinically detectable, active disease processes were eliminated from the study. This examination also insured that none of the individuals selected had complicating systemic problems. Each of the test subjects had a complete dentition with the exception of the third molars. Those volunteers judged acceptable were then scaled and polished. This was accomplished twice with a period of two weeks intervening. The first scaling and polishing was carried out by a hygienist and the second by a dentist. This insured that, prior to the test period, all of the project volunteers were as free of tooth accumulated debris as possible.

Following the second scaling and polishing, each patient was given instructions in the mechanical operation and application of the rotary instrument. This instrument consists of a prophylaxis contra-angle which

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attaches by means of a machined plastic sleeve-adaptor piece to a motor housing unit as pictured in Figure 1A. The electric motor power is supplied by a rechargeable cadmium battery located in the motor housing unit. The instrument is equipped with an on-off switch located on the base of the motor housing unit. The instrument is stored between uses in a recharging receptacle which draws current for recharging from any 110 volt alternating current wall outlet. The recharging receptacle is shown in Figure 1B. The motor design characteristics provide for rotation stoppage in the event excessive torque is generated during use. This was incorporated as a safety feature to eliminate the possibility of damage to the soft tissue. In addition to this safety feature the possibility of an inordinant amount of tooth surface abrasion was compensated for by the use of an ultra-low abrasiveness dentifrice which was supplied by the manufactuerer for use with the instrument.

Each of the volunteers was instructed to use the instrument only on the experimental quadrants. Each patient was supplied with a soft, polished, nylon bristle toothbrush for use on the control quadrants. The dentifrice used on both the control and experimental sides was the same ultra-low abrasiveness dentifrice which

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was supplied by the manufacturer of the rotary instrument. Utilizing the split-mouth technique, the experimental and control quadrants consisted of either the maxillary and mandibular right or left sides. These sides were assigned to the volunteers by another dentist and were unknown to the examiners.

Prior to plaque accumulation measurements and scoring of the test subjects, a test of inter-examiner reliability was made. Patients presenting for routine examination on two different days with an interval of one week, were randomly selected for this trial examination and scoring. Twenty patients were scored for plaque accumulation during these two periods. Each examiner made independent measurements and scored each of these patients. The results were then compared. In the first examination period inter-examiner agreement was established at eighty-five percent and at eighty-nine percent in the second examination period. This level of agreement was considered to be sufficient to eliminate inter-examiner variation as a significant variable in the experimental design.

Each of the patients were examined and scored at seven day intervals. Erythrosin disclosing tablets*

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*Strong Cobb Arner Inc., Cleveland, Ohio 44120

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were utilized to demarcate clearly the extent of plaque accumulation. Each of the two examiners made independent measurements and scored each of the test subjects. These measurements and scores were obtained utilizing the Navy Plaque Index (modified).[20] In this system the tooth is divided into three major zones, the occlusal, the middle, and the gingival zones. The gingival zone lies apical to an imaginary line connecting the crests of the interdental papillae and roughly parallels the marginal gingiva. This area is subdivided into a mesial, distal, and middle zone, with each having a small area, not exceeding 1.0 mm, adjacent to the gingival tissue. The occlusal zone is coronal to the contact area or height of contour. The middle zone extends between the occlusal and gingival zones and is divided into mesial and distal areas. By assigning all areas a score of one, more emphasis is placed on plaque adjacent the gingival tissues since the surface area is much smaller. This method of scoring is illustrated in Figure 2.

At the end of the fourth week the examiners made a final measurement and scoring of plaque accumulations. Immediately following the final examination, biopsy specimens were obtained from both the control and experimental sides. Utilizing local anesthesia, the entire

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free margin and approximately two millimeters of the attached gingiva from the palatal aspects of teeth numbers four and thirteen were removed. The incision used to free the tissue was similar to that employed in a discontinuous type gingivectomy incision. The biopsy sites were covered utilizing a standard periodontal dressing for a period of one week. Healing in all the test subjects was uneventful and appeared to be complete at two weeks. Normal gingival contour was evident eight to ten weeks following the biopsy. Each of the biopsy specimens was stored in a ten percent formalin solution prior to being submitted for histologic examination. These biopsy specimens were subsequently embedded in paraffin, sectioned at six microns, stained with hemotoxylin and eosin and evaluated to determine the degree of inflammatory infiltrate, degree of surface keratinization, and evidence of trauma or degeneration.

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Chapter III

Results

The experimental side was compared against the control side in each patient for each of the four sessions at which plaque accumulation measurements and scorings were made. A comparison of total score for all controls against total score for all experimentals during each examination period was also made. The data obtained from these measurements were analyzed by a mixed analysis of variance with repeated measurements. The results of these comparisons are shown in Tables 1 and 2. The results of each individual examination period demonstrated a significant difference in the amount of plaque accumulation between the experimental and control sides. The overall comparison between the total scores of the experimental and control sides demonstrates a similar result. The experimental side had a higher plaque score in all but nine of forty-four examinations.

The histologic findings (Figures 3-7) showed that there was no significant difference microscopically between the biopsy specimens obtained from the experimental and control sides. The degree of inflammatory infiltrate and keratinization was similar in all the tissues exa-

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mined with only minor and insignificant differences. All of the specimens demonstrated the presence of a mixed chronic inflammatory infiltrate with a lymphocyte predominance. All of the specimens revealed the presence of keratin formation, either orthokeratin or parakeratin. There was minor rete ridge proliferation and some increased vascularity in the underlying connective tissue. None of the specimens showed any evidence of ulceration, liquifaction degeneration of basal cells, epithelial dysplasia or increased collagenation of the connective tissue.

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CHAPTER IV

Discussion

The results of the study were directly opposite from the expected. Manual brushing with a soft polished nylon bristle brush was found to be significantly more effective than the prototype instrument. Initially it was felt that the instrument would produce superior results since the basic design and application so closely paralleled that of the conventional prophylaxis instruments used routinely in the dental office. Two possible factors may account for these findings. Either the patients employed in the study were unable to utilize the instrument properly or the instrument did not perform as the design characteristics implied that it might. To determine the cause of the poor performance, a survey was made of nine of the eleven project volunteers. Two patients had moved from the area and could not be contacted. Of the nine patients surveyed, seven felt that the instrument did not generate sufficient torque during operation and ceased rotation before enough force could be applied to produce effective plaque removal. Two of the nine patients felt that the instru-

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ment was difficult to manipulate, but that inadequate torque was the major problem.

The histologic evaluation of the biopsy specimens showed only minor and insignificant differences. The degree of inflammatory infiltration was higher than expected when compared to the clinical appearance of the tissues. The absence of traumatic injury or degeneration of the tissues examined indicated that the safety feature provided by limitation of the instrument torque may have precluded damage to the gingival tissues. This limitation of torque also apparently resulted in the relative ineffectiveness of the instrument in plaque removal.

The use of a minimally abrasive dentifrice was intended to eliminate or at least minimize abrasive action on the tooth surface. No clinically detectable abrasion of the teeth in the experimental quadrants was noted by either of the two examiners. The four week duration of the test period was probably an insufficiently long enough time to properly evaluate the potential abrasive action of the instrument. Also, if the amount of torque was apparently inadequate to produce effective plaque removal, there is a high probability that even if the test period had been extended, no significant

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abrasion of the tooth surface would have occurred.

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Chapter V

Summary

The portable rotary prophylaxis instrument investigated in this study was used on a once-a-day, unsupervised schedule by eleven adult volunteers for a period of four weeks. These patients were examined at seven day intervals and were scored using the Navy Plaque Index (modified) for plaque accumulations. Immediately following the examination and scoring at the end of the fourth week, gingival biopsies were obtained from each patient on both the experimental and control sides and subsequently submitted for histological examination and evaluation.

It was found that the rotary instrument was significantly less effective than manual brushing in removing plaque in the patients tested. Histologically there were no significant differences between the biopsies taken from the experimental and control quadrants.

These findings would appear to indicate that the rotary instrument is relatively inefficient as it is presently constructed. The present design characteristics apparently do not permit sufficient torque to be generated and this results in premature stoppage. Improvements in the design of the instrument could make it a very

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home aid for plaque removal. The instrument is obviously unsuitable for removal of plaque accumulations in the interproximal areas.

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BIBLIOGRAPHY

1. Mandel, I.D.: Dental plaque: Nature, formation and Effects. J. Periodontol. 37:357-367, 1966.

2. Suomi, J.D.: Oral hygiene and periodontal disease in an adult population in the United States. J. Periodontol. 43:677-681, 1972.

3. Suomi, J.D., Green, J.C., Vermillion, J.R., Doyle, J., Chang, J.J. and Leatherwood, E.C.: The effect of controlled oral hygiene procedures on the progression of periodontal disease in adults: results after third and final year. J. Periodontol. 42:152-160, 1971.

4. Schwartz, S.R., Massler, M. and Le Beau, L.J.: Gingival reactions to different types of tooth accumulated materials. J. Periodontol. 42:144-151, 1971.

5. Loe, H., Theilade, E. and Jensen, S.B.: Experimental gingivitis in man. J. Periodontol. 36:177-187, 1965.

6. Committee Report. The etiology of periodontal disease. World Workshop in Periodontics. Rumfjord, S. and others (eds.). The University of Michigan Press, pg. 169, 1966.

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7. Ayer, W.A.: Efforts to improve oral hygiene practices. J. Am. Dent. Hyg. Assoc. 46:437-443, 1972.

8. Levin, M.P. and Vandrak, R.F.: A portable rotary dental prophylaxis instrument. J. Acad. Gen. Dent. 21:27-30, 1973.

9. Cohen, M.M.: A pilot study testing the plaque removing ability of a newly invented toothbrush. J. Periodontol. 44:183-187, 1973.

10. Elliot, J.R., Bowers, G.M., Clemmer, B.A. and Rovelstad, G.H.: A comparison of selected oral hygiene devices in dental plaque removal. J. Periodontol. 43:217-220, 1972.

11. Caucro, L.P., Paulovich, D.B., Klein, K. and Picozzi, A.: Effects of a Chlorhexidine Gluconate Mouthrinse on Dental Plaque and Calculus. J. Periodontol. 43:687-691, 1972.

12. Arnim, S.S.: The use of disclosing agents for measuring tooth cleanliness. J. Periodontol. 34:227-245, 1963.

13. Glickman, I., Petralis, R. and Marks, R.M.: The effect of powered toothbrushing and interdental stimulation upon microscopic inflammation and surface keratinization of the interdental gingiva. J. Periodontol. 36:108-111, 1965.

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14. Cantor, M.I. and Stahl, S.S.: The effects of various interdental stimulators upon the keratinization of the interdental col. Periodontics 3:243-247, 1965.

15. Merzel, J., Viegas, A.R. and Munhoz, C.G.: Contribution to the study of Keratinization in human gingiva. J. Periodontol. 34:127-133, 1963.

16. Ash, M.M.: A review of the problems and results of studies on manual and power toothbrushes. J. Periodontol. 35:202-213, 1964.

17. Parfitt, G.J.: Cleansing the subgingival space. J. Periodontol. 34:133-139, 1963.

18. Bahn, A.N.: Microbial Potential in the Etiology of Periodontal Disease. J. Periodontol. 41: 603-610, 1970.

19. Cohen, D.W., Stoller, N.H., Chace, R. and Lester, L: Comparison of bacterial plaque disclosants in periodontal disease. J. Periodontol. 43: 333-338, 1972.

20. Elliot, R.J., Bowers, G.M., Clemmer, B.A. and Rovelstad, G.H.: Evaluation of an oral physiotherapy center in the reduction of bacterial plaque and periodontal disease. J. Periodontol. 43:221-224, 1972.

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TABLE NO. 1

Mean Plaque Scores

TIME PERIOD	ROTARY INSTRUMENT	MANUAL BRUSHING	DIFFERENCE
1st week	26.09	16.36	9.73
2nd week	25.00	18.27	6.73
3rd week	27.09	17.82	9.27
4th week	31.45	21.00	10.45
Totals	109.63	73.45	36.18
Overall Ave.	27.41	18.36	9.05

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TABLE NO. 2

Statistical Analysis

SOURCE	SS	df	ms	F
Between Groups	126.27	1	126.27	0.56
Experimental Error	2029.34	9	225.48	
Subtotal	2155.61	10	F. 95(1.9) =	5.12
Between Visits	324.09	3	108.03	0.72
Groups x Visits	283.89	3	94.63	0.63
Experimental Error	4051.62	27	150.06	
Subtotal	4659.60	33	F. 95(3.27) =	2.96

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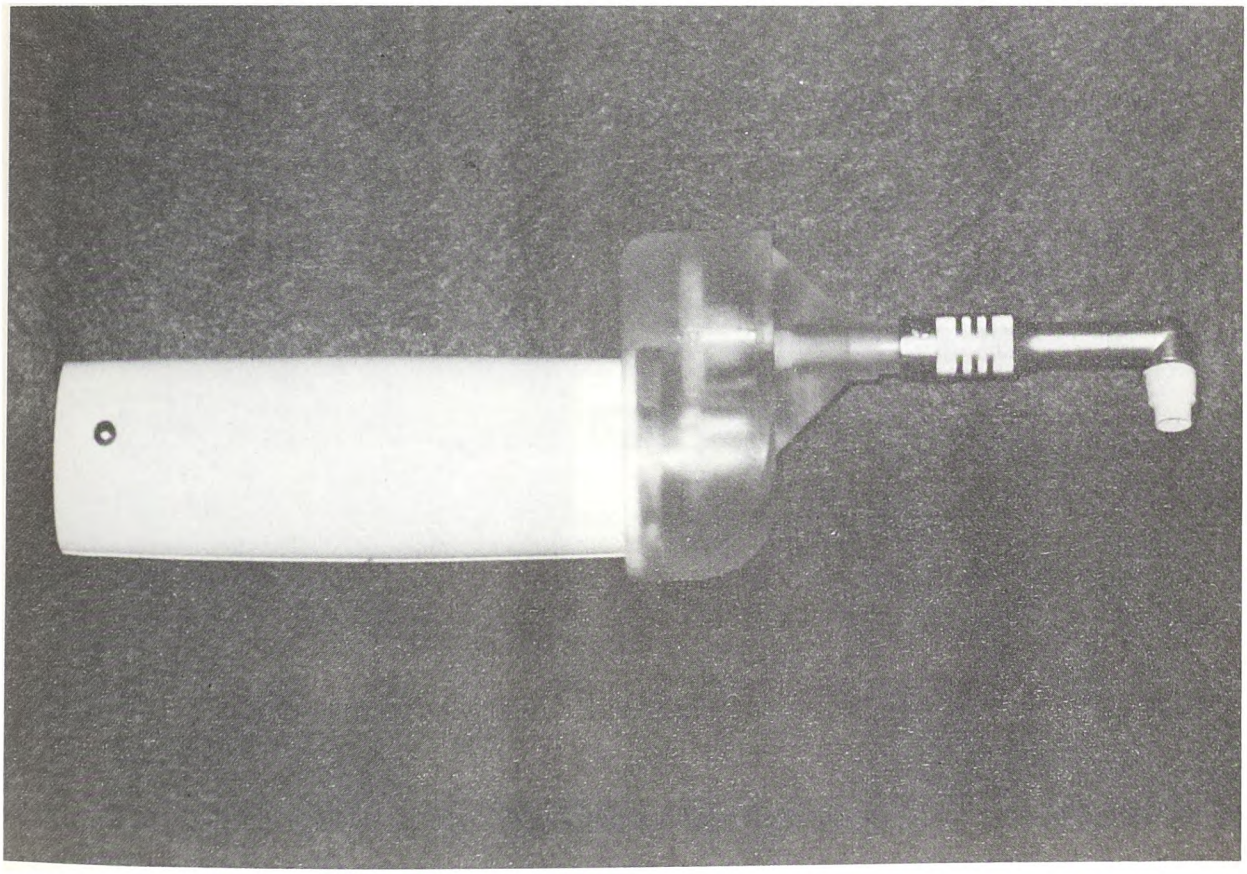

FIGURE 1A. Experimental instrument with prophylaxis angle and rubber prophylaxis cup attached.

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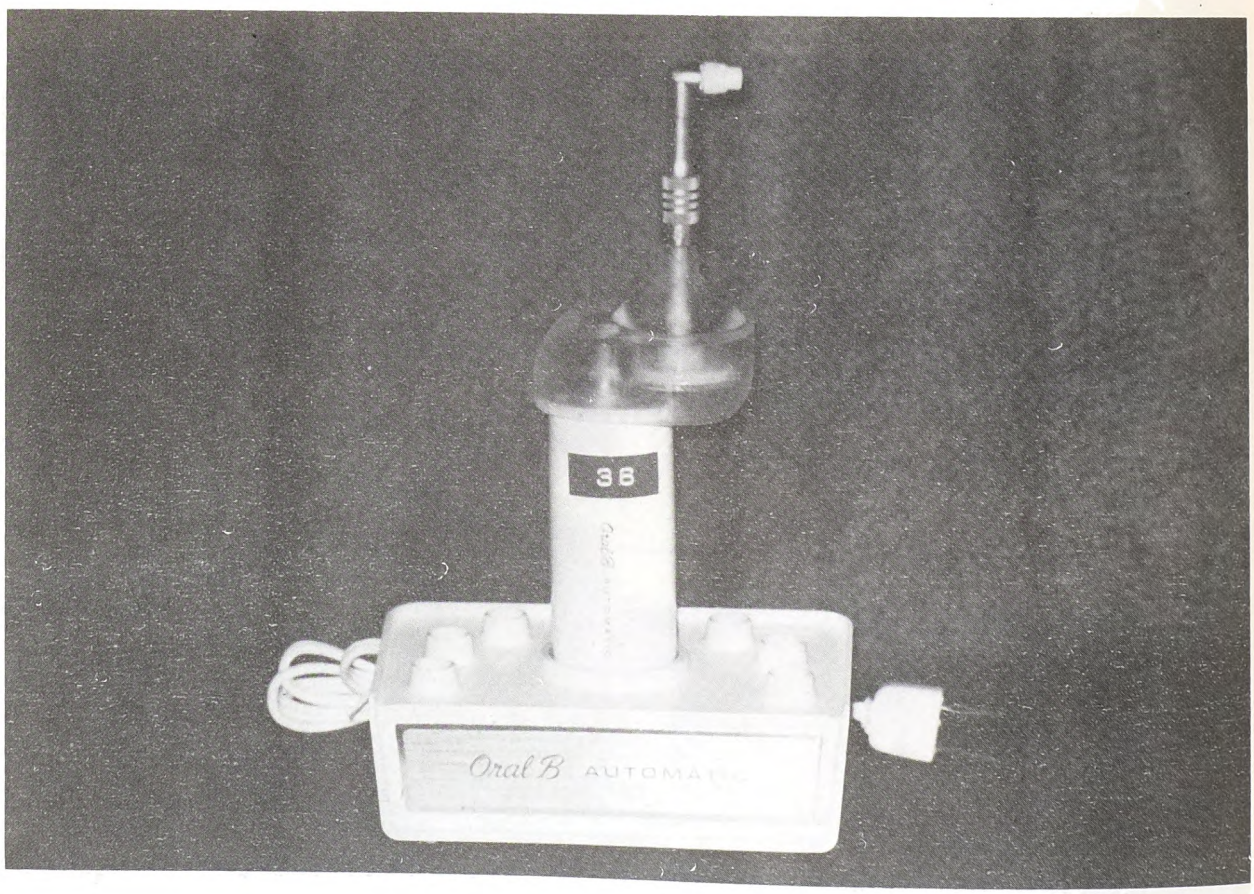

FIGURE 1B. 110 Volt A.C. recharging receptacle with experimental rotary instrument inserted for recharging.

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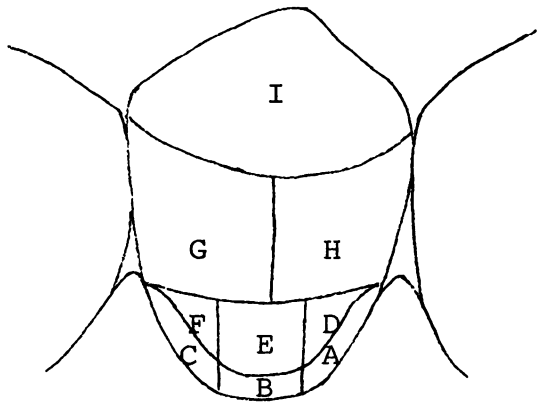

FIGURE 2. Navy Plaque Index (modified) Scoring System:

Area A, B, C SCORE 1. A thin line of stained plaque approximately 1 mm or less adjacent to the gingival tissue, both facial and lingual.

Area D,E,F SCORE 1. The stained plaque extends further into the gingival zone.

Area G & H SCORE 1. The mesial and distal halves of the middle zone area, both facial and lingual.

Area I SCORE 1. The occlusal zone.

Score facial and lingual areas. The total score for each tooth is the sum total of all areas of stained plaque on that tooth.

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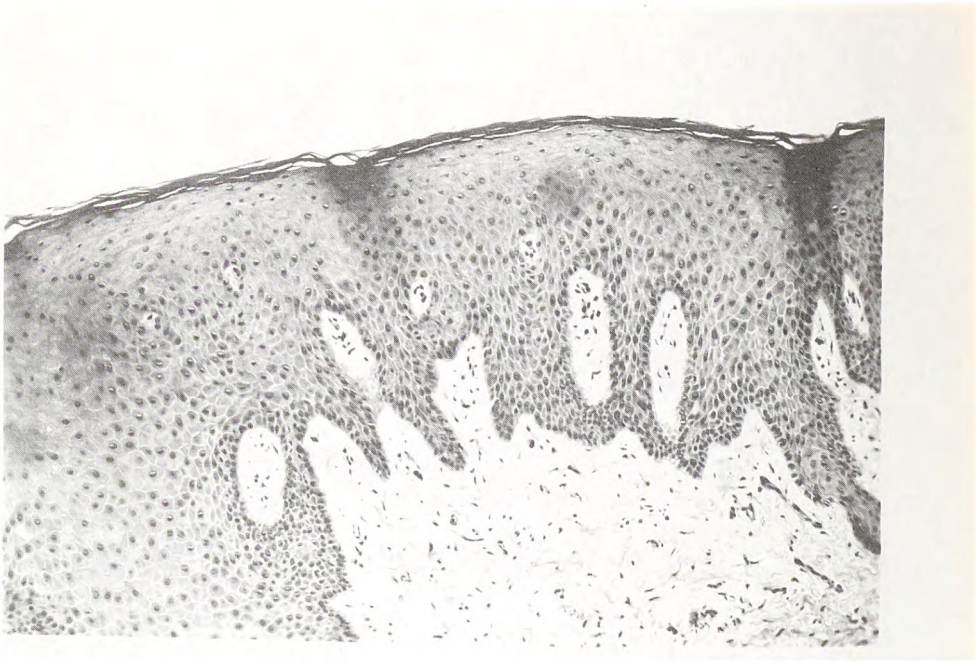

FIGURE 3. Typical biopsy specimen at 100x. Note moderate degree of chronic inflammatory infiltrate and well keratinized epithelial surface.

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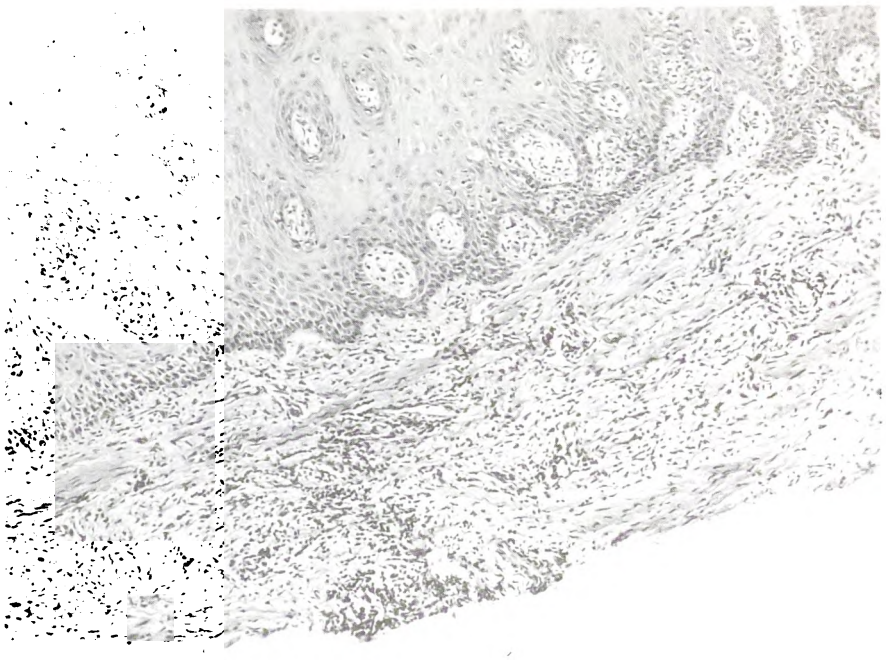

FIGURE 4. Typical biopsy specimen at 100x. Note moderate to heavy degree of chronic inflammatory infiltrate and vascularity of underlying connective tissue.

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FIGURE 5. Typical biopsy specimen at 250x. Note moderate to heavy degree of chronic inflammatory infiltrate.

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FIGURE 6. Biopsy specimen immediately following exicision demonstrating size.

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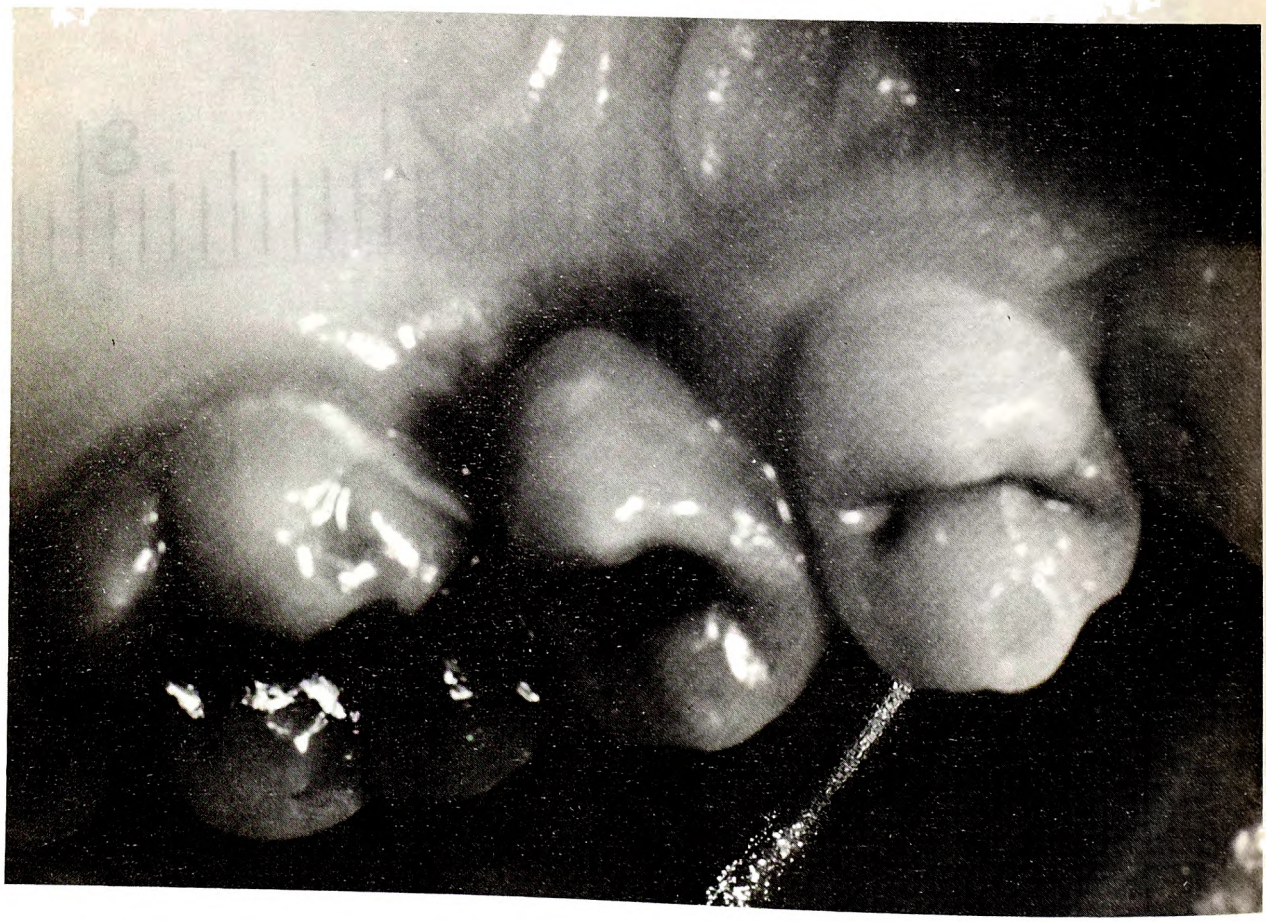

FIGURE 7. Biopsy site one week post-biopsy demonstrating healing.

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PRIOR PLANNING OF FIXED PARTIAL DENTURES

by

WILLIAM F. BOWLES III, LTC, DENTAL CORPS, USA

Professional paper submitted in partial fulfillment of the requirements of the U. S. Army Dental Residency Program, Walter Reed General Hospital, Walter Reed Army Medical Center, Washington, D. C. 20012

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CHAPTER I

INTRODUCTION

The major objective of the dental profession today is the preservation of the structures of the oral cavity.[3,4] This is to include both the dentulous and edentulous structures. The successful accomplishment of this preservation of oral structures for the maximum number of patients is dependent upon the fullest possible utilization of the dentist's time. In Fixed Prosthodontics a carefully prepared treatment plan is essential if our time is to be fully utilized. This treatment plan may significantly reduce patient appointments and the length of appointments if our planning includes a visit-by-visit treatment plan. This type of planning allows each member of the dental team to be prepared for each visit and to plan their time accordingly.

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CHAPTER II

DISCUSSION

If the dental profession is to achieve its objective of preserving the patient's remaining dentition over a maximum number of years, certain factors must be considered. In 1968 James Greeley[10] stated that planning and organizing for a fixed prosthesis are extremely important if the patient is to receive the optimum treatment. Sauser[18] stated that technical failures resulting from incorrect design, poor fabrication, or structural weakness of restorations are not the most serious causes of failure in treating partially edentulous patients. Of much greater concern to him are those failures which result from inadequate original diagnosis. Dr. Tylman in his book states,

> "Before any preventive or restorative treatment can be intelligently applied, it must be preceeded by a complete and thorough roentgenographic and clinical examination and evaluation of findings."[21]

Dykema, Cunningham and Johnston state in their book,

> "The majority of partial denture failures can be attributed to erratic treatment instituted without recourse to facts and guidelines readily available from thorough diagnostic procedures."[8]

McCracken[14] feels that errors in diagnosis and treatment occur more often as a result of too limited a consideration

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for the long term needs of the patient than for any other reason. It becomes apparent that a thorough diagnosis and a plan of treatment are essential in the planning of a fixed prosthesis if it is to be successful.

A diagnosis may be defined as the recognition of an abnormality and a thorough investigation of the severity of this condition and the reason why it has developed.[12] Bartels[1] indicates adequate study must be made during this diagnosis to determine the causative factors of destruction in the oral cavity. It may be said in a dental practice, every replacement of tissue by a prosthesis requires careful diagnosis and thorough study preparatory to treatment. Even though diagnosis may become increasingly involved as the prostheses required becomes more extensive, a poorly restored single tooth may lead to far reaching distress in the life of the patient.[5] Dawson[6] states our objective in dentistry is optimum oral health for our patients by the elimination of all factors which cause deterioration.

The first step in a diagnosis is a definite and systematic method of gathering information. Each doctor should develop his own method. However, the more consistant and systematic his approach, the more reliable and valuable his diagnosis.

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EXAMINATION

Tylman states,

> "The ability to make a satisfactory examination is the result of careful study, thoroughness, and experience."[21]

The initial phase of gathering this information for study should be an interview with the patient. Ideally the interview should take place somewhere other than the dental operatory. Initially we should ascertain why the patient is seeking treatment at this time. If a definite reason is present, it is well to listen to his account of the symptoms and his reason for these existing troubles. It may be necessary to treat an emergency condition prior to completeing a thorough examination. If the patient has no specific chief complaint and only desires a general dental check-up, you may proceed. Usually this discussion with the patient should be conversational in nature with as few direct questions as possible.

A pertinent medical history should be elicited with well chosen questions to initiate the patient talking. It should be ascertained if the patient is presently under the care of a physician, if he is taking any medications, what were the results of his last physical examination and how long ago this was. The patient's history as to serious illnesses in the past or present, especially

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those involving the cardiovascular and pulmonary systems must be determined. He also must be questioned about a history of rheumatic fever, congenital heart disease, hypertension, hypotension, diabetes, allergies, and cancer. If a history of cancer is given it must be determined if radiation therapy was part of the treatment. Today with the increase in hepatitis within our South East Asia returnees and the recent increase in venereal diseases in the United States it may be pertinent to include these areas in our medical history. It is always valuable to obtain the name and address of the patient's physician.

Along with the medical history a dental history must be illicited. If the patient has missing teeth, why are they missing? Have there been any complications following dental therapy? Does the patient or his family have any known facial or dental deformities?

Following this interview portion of the examination an extra oral examination is conducted. The oral cavity must not be considered as a part removed from the body. Among factors that may be noted are facial profile, any asymmetry, deviations upon opening or closing the mandible, and variations in color, texture or pigmentation of the skin. Any lesions or eruptions that suggest local or systemic disease should be noted. Palpation of the tempromandibular joint and the neck should be accomplished.

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This also is an excellent time to note the position and contour of the lip line and the amount of tooth showing during speech and at rest.

The intra oral examination follows this extra oral examination. A thorough soft tissue exam precedes any examination of the teeth. If no soft tissue pathology is found and a brief examination of the teeth indicates no emergency conditions exist, this is an excellent time to accomplish a prophylaxis. After this prophylaxis, each individual tooth and tooth surface can be viewed more critically. The teeth are examined to determine the number remaining, their size, shape, clinical length, position in the arch, inclination and vitality. Also to be recorded are caries, new or recurrent; exposed root surfaces, erosion, facets, mobility, open contacts and periodontal pockets. The relationship of one arch to the other and whether centric occlusion coincides with centric relation should be observed at this time.

The next phase of the examination is making a complete set of roentgenograms to include posterior bite wings.

Accurate impressions must be made for study casts. The necessary records to mount these casts in an adjustable articulator must also be made at this time. It is the

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opinion of some authors that these resultant casts are only study casts until they are mounted on an adjustable articulator and then they become diagnostic.[5,12] A duplicate set of these casts should be mounted also so the original set may be preserved as a permanent record of the condition of the patient before treatment.[2,15]

EVALUATION

This data collected during the examination must be subjected to critical evaluation. Much of this can be after the patient is dismissed and at your leisure before the next appointment to conserve time. If the medical history reveals a medical condition, past or present, that may influence the plan of treatment, a clarification by consultation with the patient's physician may be necessary. Diagnosis and treatment planning are many times governed by systemic considerations and subsequent care.[13]

The dental history must also be considered. How has the patient accepted previous dental therapy? If complications have arisen following previous dental therapy this must be evaluated. Any patient who has previously received oral hygiene instructions or has had any form of periodontal therapy can be evaluated as to how he has responded to these. Many times this response will provide a guide as to how well the patient is capable of

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maintaining his oral enviroment. The ratio of successful cases will be much greater in those patients who exhibit good oral hygiene habits.

The roentgenographic survey must next be evaluated. Good quality roentgenograms will reveal osseous pathology, retained roots, impacted teeth and foreign bodies. The crown and root to bone ratio can be determined and the size and form of the roots can be visualized. The size and proximity of the pulp can be determined, the width of the periodontal membrane can be seen and the character of the supporting bone can be observed. This may be especially valuable in areas where stress has been applied such as adjacent to tipped teeth and in areas of traumatic occlusion. This may indicate future expected reactions to additional stresses placed on abutment teeth. The overall quality of previously placed restorations may be judged as well as the location and approximate depth of carious lesions. Another important condition to be noted is the approximation of adjacent teeth considered for fixed restorations. Teeth in close approximation at the expected margin of a restoration may be contraindicated for fixed restorations unless an altered preperation can be planned. Teeth tipped more than 24^{o} from vertical should not be considered as abutments.[21]

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The evaluation continues with a thorough study of the mounted diagnostic casts. As stated before some authors feel casts of the arches cannot be termed diagnostic until they are mounted on an adjustable articulator.[10,12,13] It now becomes simple to disclose the causative factors of destruction to the teeth and their supporting structures.[1] These casts can now be viewed from the lingual as well as the buccal without obstruction. This lingual view is not attainable by any other method. Any discrepancy between centric relation closure and centric occlusion or maximum intercuspation can be visualized and measured. The deflective and interceptive occlusal contacts can now be detected that are easily overlooked in the mouth. Facets become very apparent on the dry models. The plane of occlusion can now be readily seen. The teeth that extend beyond it or those that have not achieved it are readily apparent. The amounts of horizontal and vertical overlap can be seen with clarity. An excessive vertical overlap with the upper and lower teeth locked is more readily analyzed.[1] The clinical crown lengths as well as inclinations and rotations are obvious. The arch form, shape, and contour of the edentulous areas as well as the teeth can be observed. These casts offer a mass of details not available from other methods.[2,4,16,17,18,20]

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TREATMENT PLANNING

The next step is planning of the proposed treatment. The accumulation of all this diagnostic data and its evaluation is pointless unless it is intelligently integrated into a plan of treatment.[9,10] The first and most basic question the prosthodontist must answer is should the patient be treated? By treating the patient, can we improve his oral condition and do anything to help preserve the remaining structures? Dawson[5] states the main consideration in treatment planning should be to do the minimum required to achieve optimum oral health. We must strive to condition the supporting structures to optimum health, then restore the teeth to a stabilized relationship that the patient can maintain. This stable occlusion of the teeth requires simultaneous bilateral reciprocal contact of all teeth at the final point of closure of the jaws.[3,18] Restoratively this stable occlusion necessitates proximal contacts, occluding forces directed along long axes of teeth and forces transmitted from a fixed or removable partial denture to abutment teeth that do not exert undue torque to these teeth.[3] When we decide to accept this patient for prosthodontic care the next obvious question is whether he will best be served by fixed or removable prosthetics.[19]

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McCracken[14] states that the dentist in deciding on a course of treatment must have an open mind regarding the relative merits of fixed and removable restorations. It has been stated that unless a fixed restoration is contra-indicated for some reason, one cannot justify the use of a removable restoration for arbitrary reasons alone.[14,20]

Some general indications for fixed prosthetics are: 1) Single or multiple teeth to be restored individually. 2) Tooth-bounded edentulous regions if the adjacent teeth are capable of supporting the prosthesis. Ante's law may be used as a guide.[12] The length of the span and the periodontal support of the abutments must also be considered. 3) Closing of modification spaces in cases that require a combination of fixed and removable partial dentures. This will reduce the complexity of the removable partial denture. Examples of these are the closing of anterior spaces and to support lone standing abutment teeth. An exception to this may be when the modification space is opposite a unilateral free end area. 4) Splinting of adjacent teeth when indicated.

Some general indications for removable prosthetics are: 1) Edentulous areas with no posterior abutments present. A possible exception is if molars are missing in only one side of the arch and no treatment is indicated

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unless it is needed to maintain a tuberosity in position. 2) Interim replacements after recent extractions. 3) Long spans that require some support from the soft tissues. 4) Cross-arch splinting may be accomplished with a removable partial denture.[14] 5) Esthetics may be better served in the anterior region, especially where there has been excessive bone loss or resorption. 6) Excessive residual ridge loss in any area.

If we arrive at the point now where we determine fixed prosthetics is the answer for this case or part of the answer we may proceed. We must decide if the prosthesis is to be fabricated at the existing occlusal position or if it is necessary or advisable to fabricate the restoration in harmony with centric relation. It is not indicated to attempt harmonizing centric relation and centric occlusion in the healthy mouth as it is not indicated to treat in the absence of pathosis.[16] Another answer to this question may be in the magnitude of restoration needed. One or two single crowns or a single fixed partial denture replacing one or two teeth will usually not indicate the need to harmonize centric relation and centric occlusion.[16] However, more extensive treatment usually requires the harmonization of centric relation and centric occlusion. A duplicate set of

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mounted diagnostic casts can be used to determine the amount and location of tooth structure necessary to be removed to achieve this harmony. These mounted casts have many other very useful purposes. They are useful in evaluating forces that act upon the individual teeth and the proposed restorations. They may be used to select, contour, and position facings. They may be used as a guide in planning and preparing retainers or restorations. The plan of procedure for entire mouth is resolved at this time. Alternatives and contengencies for future treatment are considered and incorporated in the treatment plan if necessary. The proposed plan can be from one tooth restoration at a time to a complete reconstruction of all the teeth at one time. A single tooth replacement for a patient with good quality restorations and whose occlusal relationship is satisfactory can be expected to serve many years. In these cases several single restorations over a prolonged period may achieve satisfactory results. However, if the treatment requires replacement of all restorations, the outcome is doubtful as single restorations are usually placed in opposition to and adjacent to teeth which are not usually restored to proper form and function. Many times quadrant treatment is accomplished if the opposing occlusion is

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satisfactory or can be modified or adjusted so as not to interfere until it can be replaced. The next step to consider is the reconstruction of one side of the mouth at a time. This has the advantage of : 1) Vertical dimension is maintained. 2) The anatomy and function of the reconstructed side can be formed and adjusted to the best possible relationship as the only limitations are the opposite side which has been carefully equilibrated before the case was initiated. 3) The patient maintains one normal side to function on until the other side is completed. Last, the simultaneous reconstruction of all the teeth can be considered. This allows maximum freedom in the development of a new occlusal relationship.

To do any of these types of restorations the proposed restorations can be drawn on the diagnostic casts and then actually prepared on the casts to act as a guide to mouth preparation.[17] The greatest number of failures in fixed prosthodontics are probably attributable to a violation of the basic principles of resistance and retention form.[7] These preparations are especially valuable as a guide to paralleling preparations and establishing a path of insertion. Also the amount of tooth reduction necessary to accomplish the objectives of the restorations can be easily determined. This may

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include reduction to bring a tooth into the plane of occlusion, to correct the cusp to fossa relationship, or selective reduction of tilted or rotated teeth.

After the preparations have been planned these modified casts may now be used to complete a diagnostic wax-up. This will be especially helpful in diagnosing more extensive or difficult problems. The completion of the diagnostic wax-up will aid the dentist in visualizing the finished product and will better acquaint him with the difficult or critical areas in the planning of the case.[1,2] It may even be necessary to further alter the proposed restorations to better achieve the desired results. This self teaching exercise will aid to alert the dentist to difficult areas in planning cases where a diagnostic wax-up is not used. This very seldom used procedure probably offers more information to the fixed prosthodontist than any other procedure in treatment planning.

As the final stages of treatment planning are reached these questions as stated by McCracken must be answered:

"1. By which method of treatment will the patient best be served?
2. Is the prosthetic service recommended the best dentistry has to offer? If compromises are necessary, are the alternative methods

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of treatment acceptable by today's standards?

3. Is the dentist willing to assume the professional responsibility for the accomplishment of the treatment being recommended?"[14]

If the diagnosis and treatment planning have been comprehensive the first two questions should be readily answerable. Each doctor must know his own capabilities to answer the remaining question. There should be no reason to undertake a proposed plan of treatment if the doctor has any doubts of his professional skill to accomplish the final result.

In the presentation of the treatment plan to the patient, the comparison of the original diagnostic casts and the diagnostic wax-up of the proposed treatment will enable the patient to clearly visualize and understand the necessity for change.[10] This will include the types of restorations proposed and the extent of the proposed changes to the total dentition.

These waxed diagnostic casts can be used for other purposes to save valuable time in the office. Custom impression trays for final impressions may be fabricated from these casts. The temporary restorations to be utilized may be fabricated on these casts before the treatment is actually begun. These type of temporaries can be made to reflect the new occlusion to be fabricated in the final restoration.

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CHAPTER III

SUMMARY

This article was not written to offer new knowledge or techniques. Rather it is meant to suggest the maximum use of our knowledge and skills be utilized in the careful diagnosis and planning of treatment for our patients. It has been said a dentist's personal observations, his clinical experience and his knowledge of basic science are his capital tools.[5]

Every replacement of tissues by a prosthesis deserves careful diagnosis and a thorough study before treatment is initiated. Rudd states: "No thorough and precise method of planning should be regarded as superfluous when the health of the patient is safeguarded and when the professional procedures of the dentist are improved and hastened."[17]

Prosthodontic cases that are well thought out and planned in advance proceed more easily and quickly. These cases also will have less tendency for failure and will not require retreatment.

As a profession we will be better able to fulfill our objectives of preservation of the structures of the

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oral cavity if we are able to treat our patients as efficiently and effectively as possible. This allows for the treatment of the maximum number of patients in the least amount of our time. This is best accomplished if each patient has a thorough diagnosis and plan of treatment. The diagnosis and plan of treatment is formulated from a thorough examination and a careful evaluation of the findings from the examination. The prostheses constructed in accordance with a carefully predetermined plan to encompass the qualities of stability, comfort, and maximal efficiency are the result of the concept of preservation of the remaining teeth, their supporting structures and adjacent tissues.[11]

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BIBLIOGRAPHY

1. Bartels, J.C.: Diagnosis and treatment planning, J. Pros. Den. 7:657 (September) 1957.
2. Chestner, Stanley B.: A methodical approach to the analysis of study casts, J. Pros. Den. 4:622 (September) 1954.
3. Cohn, L.A.: Factors of dental occlusion pertinent to the restorative and prosthetic problem, J. Pros. Den. 9:256 (March-April) 1959.
4. Contino, R.M. and Stallard, H.: Instruments essential for obtaining data needed in making a functional diagnosis of the human mouth, J. Pros. Den. 7:66 (January) 1957.
5. Contino, Raymond M.: Diagnosis needed in applying fixed partial denture protheses, Dent. Clin. of N. Amer. 103 (March) 1965.
6. Dawson, Peter E.: Personal Communication, 1973.
7. Douglas, Gordon D.: Principles of preparation design in fixed prosthetics, J.A.G.D. 21:25 (April) 1973.
8. Dykema, R.W., Cunningham, D.M., and Johnston, J.F.: Modern Practice in Removable Partial Prosthodontics, Philadelphia, W.B. Saunders Co., 1969, chap. 2.

Digitized by Google

9. Friedman, S.: Effective use of diagnostic data, J. Pros. Den. 9:729 (September-October) 1959.

10. Greeley, J.H.: Planning for fixed prosthesis, J. Den. 20:412 (November) 1968.

11. Johnston, J.F.: Preparation of mouths for fixed and removable partial dentures, J. Pros. Den. 11:456 (May-June) 1961.

12. Johnston, J.F., Phillips, R.W., and Dykema, R.W.: Modern Practice in Crown and Bridge Prosthodontics, Second Ed., Philadelphia, W.B. Saunders Co., 1965, chap. 2.

13. Klenda, H.M.: Consultation, examination and treatment planning, J.A.D. 58:86 (February) 1959.

14. McCracken, W.L.: Differential diagnosis: fixed or removable partial dentures, J.A.D.A. 63:767 (December) 1961.

15. Myers, George E.: Textbook of Crown and Bridge Prosthodontics, Saint Louis, The C.V. Mosby Co., 1969 chap. 9.

16. Pruden, William H.: Occlusion related to fixed partial denture prosthesis, Dent. Clin. of N. Amer. 121 (March) 1962.

17. Rudd, K.D.: Making diagnostic casts is not a waste of time, J. Pros. Den. 20:98 (August) 1969.

Digitized by Google

18. Sauser, C.W.: Pretreatment evaluation of partially edentulous patients, J. Pros. Den. 11:886 (September-October) 1961.

19. Smith, Gilbert P.: Factors affecting the choice of partial prosthetics---fixed or removable, Dent. Clin. of N. Amer. 3 (March) 1959.

20. Smith, Gilbert P.: Objectives of a fixed partial denture, J. Pros. Den. 11:463 (May-June) 1961.

21. Tylman, S.D.: Theory and Practice of Crown and Fixed Partial Prosthodontics (Bridge), Sixth Ed., Saint Louis, The C.V. Mosby Co., 1970, chap. 2.

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OSTEORADIONECROSIS IN THE HEAD AND NECK CANCER PATIENT: A REVIEW

by

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Professional paper submitted in partial fulfillment of the requirements of the U. S. Army Residency Program, Walter Reed Army Medical Center, Washington, D. C. 20012

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CHAPTER I

INTRODUCTION AND DEFINITIONS

It is the purpose of this paper not only to review the literature on the subject of osteoradionecrosis, but to provide a clear knowledge of the disease, its diagnosis, and various methods of treatment.

Before the task of reviewing this paper is undertaken, it is necessary that some of the terms used are defined, and that the effect of radiation on cells be discussed. This has been lacking in all of the papers reviewed. At the time various methods of treatment are discussed, some of the fundamentals of hyperbaric oxygenation will be covered.

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DEFINITIONS

1. Alpha particle:[4] Helium nuclei in rapid motion.

2. Beta particle:[4] Negatively charged electrons.

3. Diaphysis:[1,3] The portion of a long bone between the ends or extremities, formed from a primary center of ossification.

4. Endarteritis obliterans:[1] Inflammation of the intima layer of an artery followed by collapse and closure of the smaller branches.

5. Epiphysis:[1,3] The end of a long bone, formed from a secondary center of ossification.

6. Involucrum:[1] A covering or sheath, such as that which contains the sequestrum of necrosed bone.

7. Ionization:[1] The dissociation of a substance in solution into ions.

8. Osteitis:[1] Inflammation of a bone, involving the haversian spaces, canals and their branches, and generally the medullary cavity; and marked by enlargement of the bone, tenderness and dull aching pain.

9. Osteoradionecrosis:[1] Necrosis of bone following radiation. That pathological process which sometimes follows heavy irradiation of bone and is characterized by a chronic, painful infection and necrosis; accompanied by late sequestration and sometimes permanent deformity.

10. Periarteritis:[1] Inflammation of the external coats of an artery and of the tissues around the artery.

11. Rad:[1] A unit of measurement of the absorbed dose of ionizing

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radiation; it corresponds to an energy transfer of 100 ergs per gram of any absorbing material (acronym for radiation absorbed dose).

12. Radiation:[1] Electromagnetic waves such as those of light or particulate rays, such as alpha, beta and gamma rays, given off from some source.

13. Radioactivity:[1] The quality of emitting or the emission of corpuscular or electromagnetic radiations consequent to nuclear disintegration. Spontaneous decomposition of an atom accompanied by the emission of alpha, beta and gamma rays.[5]

14. Radionecrosis:[1] Destruction of tissue or ulceration caused by radiant energy.

15. Roentgen:[1] It is the quantity of x or gamma radiation such that the associated corpuscular emission per 0.001293 gram of air produces in air ions carrying one electrostatic unit of electrical charge of either sign.

16. Saucerization:[1] The excavation of tissue to join a shallow shelving depression usually performed to facilitate drainage from infected areas of bone.

17. Sequestrum:[1] A piece of dead bone that becomes separated during the process of necrosis from the sound bone.

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CHAPTER II

RADIATION THEORIES; ALSO, A HYPOTHESIS ON THE MECHANISM OF RADIATION EFFECTS ON CELLS

Radiation[1,4] is an electrostatic wave such as that of light, or a particular ray such as alpha, beta or gamma, given off from some source such as radium, plutonium, estrontium, uranium and X-ray machines. The type of radiation referred to here is ionized radiation, which carries sufficient energy to produce ionization of the material into which it is absorbed. The ionization[4] may be visualized as a momentary loss of an electron from an atom to form an ion pair, followed by prompt reversal of the process and a return to a stable state. The state of ionization is estimated to persist for only 10^{-8} seconds. Ionizing radiation appears to produce its biological effects almost entirely through the mechanism of ionization.

In spite of general agreement that biological effects are casually related to ionization, there is no clear explanation of the precise mechanism of cell injury. Basically, there are two hypotheses[4] or theories of cell injury: 1)There are within cells vital structures of limited volume, and if one or several ion pairs are formed within this sensitive volume, the cells will be severely damaged or killed. This concept is known as the "direct action theory," "quantum hit theory," or "point heat theory." 2) That changes of a more different nature occur within

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cells or in the surrounding tissue fluids, releasing injurious substances usually assumed to be protein breakdown products or peroxides.

C. C. Pollard[5] suggested that radiation damage to the cell involves four processes of major importance. These are: 1) Scission of DNA followed by enzymatic degradation with some DNA being more susceptible than others. 2) Halting of genetic transcription, with the possibility of subsequent recovery. 3) Reduction in the rate of DNA synthesis. 4) Involvement in the repair processes which are effective to different extents at different stages in the cell cycle. It probably works like this: if there is damage to the DNA molecule, there is no transcription of DNA and no messenger, so there are no new enzymes or proteins being produced. When cells are radiated the major target is usually the macromolecules such as nucleic acids, DNA, or proteins. Proteins[5] are a critical target of ionizing radiation. Although these protein molecules may not be unique, their position (chemical) in a cell structure may make them difficult to replace. It may not be possible to repair or replace them quickly enough to save the cell structure.

There is evidence[5] that radiation damage to protein molecules tends to be localized at the sulfhydryl^{S-H} and disulfide^{S-S} groups.

In summary radiation has three effects on cells: 1) It kills cells. 2) It induces mutations including chromosomal arrangements. 3) It delays cell division.

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Cells in general respond in three different ways to the lethal action of radiation: 1) Reproductive death through mitotic inhibition. 2) Genetic death through loss of genes essential to cell survival. 3) Lytic death.

When cells, viruses and other tissues are exposed to high doses of radiation, growth rate decreases, and mortality and chromosomal damage with mutations increase. When mamallian cells,[5] are radiated with 130r, 37% survive and the rest die. When the dose is increased to 200r or more, a break is produced. This is interesting in that this is approximately the same dose as that found for plant cell chromosomes.

Cells[4] are very sensitive to radiation at the time of division, especially in the early prophase or late premitotic phase.

Mature bone is relatively radioresistant, but growing bone is radiosensitive. Injury to bone is most marked at the sites of ossification and remodeling, and consists of necrosis and disorganization of osteoblasts, with the disappearance of osteocytes from the haversian system. Gross structural integrity is characteristically maintained.

Some authors claim that there is an endarteritis[6] or periarteritis of the blood vessels, which will decrease the blood flow to different areas, resulting in the formation of a blood clot and necrosis of tissue. This will leave the bone without any line of defense for either formation of new bone or for fighting an infection. This re-

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sults in osteoradionecrosis of bone.

The amount of radiation required to cause bone injury varies with age and radiation factors such as the type of radiation, amount of radiation, time, and portals. With intense radiation even mature bone may undergo massive necrosis and pathological fracture.

There is no satisfactory clinical method[4] of discovering damage to the bone matrix or even death of bone, unless there is an associated change in the concentration or distribution of calcium salts detectable by radiographs. Necrotic bone is highly vulnerable to infection.

Salivary glands are very radiosensitive. Upon radiation they may suffer temporary or permanent loss of function. Accompanying these changes there is diminution and thickening of saliva. A dose of 3000REM or more causes lasting atrophy, fibrosis, and dysplasia of salivary glands in rats.

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CHAPTER III

FACTORS IN OSTEORADIONECROSIS

According to Kanthack[7] and others,[9,14,15,17,23,24] there are three different factors which seem to be conducive to osteoradionecrosis. These are: 1) Irradiation, 2) Trauma, 3) Infection.

Irradiation[8] of osseous tissue not only tends to destroy the normal resistance of the tissue to infection and its response to healing, but also to alter the entire process of healing.

Shields Warron states, "that the reaction of formed bone to radiation is primarily a lowering of vitality, which in some instances progress to necrosis." This may occur without change in the bone structure or general appearance. The secondary rays[17,23] act mainly on the lamellar substance altering in some way the physiological constitution of this substance without producing gross evidence of change as long as bone remains free from trauma and infection.

An alteration[17] in the ground substance of bone may be responsible for the greater susceptibility to infection and less susceptibility to histolytic solvents.

The function of periosteum[9] is also altered by either primary and secondary radiation. After sizeable therapeutic doses[11] of radiation to bone, gross swelling and thickening of the periosteum

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occurs, which allows it to be stripped away from bone very easily. Does this mean the attachment of periosteum to bone is destroyed? At this time there is no research that can answer this.

Cellular elements within the bone-marrow space are also changed. The blood vessels are occluded by thrombosis or a proliferating fibrosis. There is closure of the canaliculi between osteocytes. The blood vessels tend to become thickened and are finally obliterated. In three cases studied by Niebel,[17] part of the mandible received over 6000r and was subsequently excised. He found that the predominant change was the presence of a diffuse fibrosis. In certain areas there was new vessel formation and telangiectasis consistent with granulation tissue formation. In the cortical bone a few lacunae were empty, which shows death of the osteocytes.

In summary,[8] when bone is compared with soft tissue, bone is known to absorb more radiation energy because it contains more of the heavier elements. This results in death of the osteocytes, thrombosis of blood vessels, fibrosis, and lack of resistance to infection.

TRAUMA

Trauma such as that of a tooth extraction,[9,15,16] acts of mastication, decubitus ulcers,[12] wearing of dentures[13,15] or partial dentures, abrasion, and pressure necrosis are all factors to be concerned with. True trauma in itself,[9] however, is not always an etiological factor. Poor oral hygiene leading to periodontal infection and radiation caries following irradiation therapy of the jaws may be the only predisposing

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factors. Topazian[21] cites other types of traumatic factors such as toothpicks, chewing rough foods, fractured teeth, and hot beverages that can cause or initiate the pathological process of osteoradionecrosis.

INFECTION

The access of organisms[17,23] into the irradiated tissues appear to be the single most consistent factor in the initiation of osteoradionecrosis. Infection[9] changes are observed following the removal of teeth or in periodontal infections in patients that have received radiation to the jaws. The clinical appearance simulates that of chronic osteomyelitis. Loss of vascularity due to vessel ligation such as that performed in radical neck surgery definitely decreases tissue regeneration and resistance to bacterial infection. Niebel[17] claims that he has never seen a purely aseptic case of osteoradionecrosis. The primary avenues of bacterial invasion to the maxillae and mandible are by direct communication of the alveolar bone with the oral flora, and by way of badly broken teeth or due to periodontal infection, or by trauma caused by dentures. If these factors could be controlled, infection in turn could be controlled, and the pathological process of osteoradionecrosis blocked.

According to Rankow and Weissman,[19] there are not only three, but seven, etiologic factors or predisposing factors that may predispose the patient undergoing radiation therapy to osteoradionecrosis, These are: 1) Size of tumor, 2) Irradiation dose, 3) Energy of ra-

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diation, 4) Field size and number of irradiated fields, 5) Oral hygiene, 6) Dental trauma or extractions, 7) Radical neck dissection after radiation.

SIZE OF TUMOR

The size of the tumor is directly related to the incidence of radiation osteitis.

Rankow and Weissman [19] reported that four of the patients, though free of a tumor, had lesions staged as T_{2-3}, these patients developed osteoradionecrosis.

Grant and Fletcher[18] reported a 15% higher incidence of osteoradionecrosis in patients staged as T_{3-4} than as T_{1-2}.

IRRADIATION DOSE

Combined interstitial and external radiation appear to be related to a high incidence of osteoradionecrosis. In Grant and Fletcher's[18] study, 33.3% of their patients received external radiation and interstitial implantation of radioactive sources. These patients developed bone exposure and osteoradionecrosis and had to be treated by a hemimandibulectomy.

In another series reported by MacDougall,[12] in a total of 180 patients treated by medium voltage radiation and radium needle implants, nine patients developed osteoradionecrosis. In another series of six cases with osteoradionecrosis, reported by Niebel,[17] four patients were treated with external radiation and also with radon implants, and all developed osteoradionecrosis.

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In summary, it seems reasonable to assume that when there is external radiation supported by interstitial radiation utilizing radium needles and radon seeds, the incidence of osteoradionecrosis is very high.

ENERGY OF RADIATION

With the use of orthovoltage 250KVP X-rays, the energy absorption of bone is almost double and sometimes two and a half times that of the surrounding soft tissues. When megavoltage is used such as cobalt-60, the energy deposited in bone is equal to that of the surrounding soft tissues. Using megavoltage as the primary mode of treatment for squamous cell carcinoma of the tonsillar area, Grant and Fletcher[18] found a rather high incidence of osteoradionecrosis, 37%. It is interesting to note that in their series, 45.4% of the bony exposures were in patients having a T_{3-4} tumor size. As mentioned previously, the bigger or the higher the tumor size, the higher the possibility of developing osteoradionecrosis. All forty-three of the sixty-nine patients who developed bone exposures or radionecrosis healed with conservative treatment.

In a series of 180 cases reported by MacDougall,[12] in which the radiation treatment was accomplished by either medium voltage or radium implants, only nine cases developed osteoradionecrosis. The figures in his study are somewhat complicated due to the use of radical surgery in the patients treated from 1952-1959. These 180 patients were then treat-

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ed by cobalt-60, some of them with radium implants and moulds and some with radical surgery. Of these 180 patients, nine developed osteoradionecrosis. MacDougall[12] claims that the low incidence of osteoradionecrosis was due to the use of cobalt-60; however, it is very difficult to support that statement due to the different modalities of treatment used in these patients.

Meyer[26] did a study with cobalt-60 and found that its use is less damaging to bone and dental tissues than the use of 200KVP radiation, using the same radiation dose.

Hoffmeister[25] also concurs in the opinion that when you use megavoltage, the post radiation necrosis and osteomyelitis of the mandible is less that when you use orthovoltage. Robinson[22] claims the use of radium needles and randon seeds have produced some of the most severe damage; however, he also states that, "although some authors claim that radiation from cobalt-60 spare covering structures, there is no real difference in bone sparing effects."

FIELD SIZE AND NUMBER OF IRRADIATED FIELDS

Osteoradionecrosis is more frequent and more severe when a single homolateral field is used in comparison to a technique using opposite ports. Grant and Fletcher[18] found that when using homolateral fields in 50 patients, 48% (24 patients) had bone exposure. Of these 24 patients, nine (18%) required a hemimandibulectomy for the treatment of osteoradionecrosis. When the technique used was that of unequally load-

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ed parallel opposing fields in 64 patients, 22 patients (34.4%) developed bone exposure and four patients needed a mandibulectomy for treatment of osteoradionecrosis. When the technique used was equally loaded parallel opposing fields in fourteen patients, only two(13.2%) developed bone exposure, and neither of these two patients needed radical surgery for the treatment of osteoradionecrosis; they healed as a result of conservative treatment.

ORAL HYGIENE

The oral hygiene in a patient who is receiving or who has received radiation treatment in the head and neck area has to be very meticulous, especially after radiation. MacDougall,[12] Scanlo,[13] Francisco,[14] Topazian,[21] Niebel,[17,23] Wildermuth and Cantril,[28] Hinds,[24] Daly[27] and DelRegato[33] all agree that radiation therapy to head and neck cancer decreases the amount of saliva produced as well as changing the acidity (pH), making it a relatively ineffective oral cleansing agent. The decrease in pH is probably due to lessened glandular function.[23] In Daly's[27] work he states,

> "The oral hygiene is an important factor in preventing progression of radiation decay. . . the amount of plaque formation or debris present is directly proportional to the advancement of decay process. . . ."

DENTAL TRAUMA OR DENTAL EXTRACTIONS

Extractions of teeth[30] before radiation treatment has been the causative factor in osteoradionecrosis. Extraction of teeth[13,28,31] after radiation treatment has been the causative factor in osteoradio-

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necrosis. These completely opposite statements are made because in reviewing the literature, it was found that in the twenties and thirties osteoradionecrosis was due to an infection caused by a dental abscess which was brought about by radiation caries.[8,13,14,15,16,22,29] On the other hand, there are many reports[15-19,28] that claim that the extraction of teeth prior to the radiation treatment was the causative factor for osteoradionecrosis to develop.

Rankow and Weissman[19] recommend a full mouth extraction 10-14 days prior to the onset of radiation therapy, and a careful and thorough alveoloplasty with primary mucosal closure without tension. If this treatment is accomplished prior to radiation treatment, the patient will have the best chance of avoiding osteoradionecrosis.

In Chambers' work,[22] he found that mandibular osteomyelitis did not occur in dogs following irradiation when an intact mucosa was present and that the removal of teeth shortly before irradiation of the dogs' mandibles significantly reduced the incidence of osteomyelitis. There are two schools of thought in the problem of teeth and radiation. One advocates the removal of all of the teeth prior to radiation therapy.[8,9,14,16,17,19,29,31,32,36] The other claims that only the teeth that cannot be salvaged should be removed[12,18,21,22,24,25,27,28,34,35,37] and that the other teeth should be preserved.

The second procedure, that of extracting nonsalvageable teeth prior to radiation treatment, is the one being used here at Walter Reed Army Medical Center and at the M. D. Anderson Hospital. This

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method appears to be effective, but more time is needed before a definite conclusion can be reached. Each case should be evaluated individually.

RADICAL NECK DISSECTION AFTER RADIATION

This has not been previously considered as a predisposing factor. The bearing of the inferior border of the mandible[19] during radical neck dissection results in a decreased periosteal arterial blood supply to the mandible, as well as venous congestion. This creates a situation in which the slightest trauma or infection may induce osteoradionecrosis. Three of twelve patients[19] developed osteoradionecrosis after radiation therapy and then radical neck surgery. The treatment of choice was a hemimandibulectomy.

Certainly there are other factors involved in osteoradionecrosis. In a report of Daly's[27,34,35] work at the M. D. Anderson Hospital, it is noted that the patients that ultimately developed osteoradionecrosis of the jaws have many characteristics and predisposing factors in common. The following is a list of such factors: 1) Previous surgery done shortly before start of radiation therapy without obtaining adequate healing. 2) Lesions being irradiated that are in close proximity to bone, such as squamous cell carcinoma of the floor of the mouth, trigone area, etc. 3) The use of high dosage of radiation with or without proper fractionation. 4) The use of external radiation and implants (previously mentioned). 5) Poor oral hygiene with the continued use of irritants. 6) Poor patient

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cooperation in managing the irradiated tissues. 7) Indiscriminated use of prostheses after radiation therapy without resolving all treatment sequelae.

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CHAPTER IV

DIAGNOSIS AND TREATMENT

DIAGNOSIS

The usual and universal complaint is pain along or in the side of the mandible, associated with a small intraoral area of ulceration.[19] The pain can be described[21] as deep and boring, with associated swelling, trismus, and eventually the development of soft tissue ulcers. Teeth in the area are sensitive to hot or cold fluids.[19] Ulceration[21] of the skin or oral mucosa may be present. In severe cases there is sequestrum formation, and this may consist of the entire mandible. Denuded[15] or exposed bone is almost present in the area of osteoradionecrosis. Oroantral and oronasal fistulas can develop later in patients with osteoradionecrosis of the maxillae. Orofacial fistulas can be present in the mandible. Fetor oris is also present due to the infection and to the poor oral hygiene. The most definite[38] radiographic alteration in early disease is that of increased radiodensity, followed by osteolysis in the affected area; however, the patient is generally asymptomatic. In the late disease, X-rays[19] may show multiple punched out irregular areas and tomography may show a pathological fracture. Demineralization[22] with rarefaction is diffuse in the involved bones. There are radiolucent, poorly defined cystic areas intermingled with dense shadows of hypercalcification in a mottled configuration.

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TREATMENT

The best treatment of osteoradionecrosis is prevention.[14,21,24,25,27,32,34,35,38]

There are three schools of thought in the treatment of osteoradionecrosis. 1) Conservative treatment.[17,21,22,23,24,25,27,28,32,34,35] 2) Radical treatment.[12,16,29,32,36,40,42] 3) Combination of the above,[8,9,14,18,29,38] i.e., first conservative treatment; if ineffective, then radical treatment.

The first management of osteoradionecrosis should be prevention. This should start before radiation treatment is begun on the patient. A competent general dentist, oral surgeon, and prosthodontist, as well as a periodontist and endodontist, should be consulted in the treatment planning of patients with cancer of the head and neck area. Once osteoradionecrosis is present, there are three methods of treatment: conservative, radical and combination of both. In the last years there has been some reports on the treatment of osteoradionecrosis with a hyperbaric oxygenation.

The first management of bone necrosis (osteoradionecrosis) should be the conservative treatment.[27,34,35] No surgical intervention should be attempted unless there has been continuous failure using conservative procedures. Surgical intervention may extend the necrotic site to include areas that clinically are not necrotic. Healing of bone exposure and necrosis is promoted if infection is brought under control. Conservative treatment such as zinc perox-

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ide[27,34,35] packs, 1% neomycin solution packs and systemic antibiotics are useful for the treatment of limited areas of necrosis. Other conservative methods are the removal of bony spicules above the gingival crest, good oral hygiene and a soft diet. Grant and Fletcher[18] recommend removal of irritating factors such as tobacco, hot beverages, poorly fitting dentures, and in the final analysis the removal of all irritating substances. If bone is exposed, the primary therapeutic approach is conservative treatment such as the use of salt and soda irrigations. Operative intervention should be resorted to only after conservative treatment has failed.

Hinds[24] recommends the use of baking soda irrigations for cleaning the oral cavity and to promote good oral hygiene. The use of antibiotics has been advocated for the treatment of infection and of osteoradionecrosis. Alexander,[39] in his work using tetracyclines for controlling osteoradionecrosis in rat mandibles, found that rats receiving tetracycline 400mg./kg. of weight/day before, during and after extractions of teeth developed chronic osteoradionecrosis in fewer instances than rats that received no tetracycline therapy. Also, he observed that changes associated with the development of osteoradionecrosis in heavily radiated bone such as necrosis, sequestrum formation, and decreased osteoblastic activity, were observed more frequently in rats not receiving tetracyclines. These observations indicate that tetracycline will reduce the incidence and development of widespread osteo-

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radionecrosis in heavily radiated rat mandibles. This study confirms the premise that antibiotics such as tetracycline and others [38] are very helpful, but they are not a substitute for careful planning before irradiation. Guttenberg [38] found that the use of vibramycin (doxycycline) and cleocin (clindamycin) have been effective in the treatment of mild cases of septic osteoradionecrosis as well as preventing infection when surgery must be performed on the irradiated jaw. If teeth are to be removed from irradiated areas, Niebel[23] recommends the use of elastic bands similar to orthodontic bands to remove teeth. Although this procedure is primarily one of bone resorption, a certain amount of extrusion takes place. Between 3-16 weeks are required to remove a tooth by this method. If extractions are performed prior to radiation, MacDougall[12] recommends that they be done with minimal trauma, and alveoloplasty with the removal of interdental bone to permit a tension free closure of the mucoperiosteum over a smooth bony base is mandatory. Before closure is accomplished all debris must be removed and the edges of the mucoperiosteum freshened to produce a continuous apposition of viable tissue. Adequate pre-post-operative antibiotic coverage should be instituted. The sutures are left in place from five to seven days, and radiation therapy is delayed for fourteen days. Ladow[9] recommends that the treatment of osteoradionecrosis should be essentially symptomatic, that is, the treatment of the symptoms. With the use of analgesics [9,15] and narcotics

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for pain, the oral cavity should be irrigated with hydrogen peroxide 1.5% to help loosen the slough. Surgery may consist of single excision following sequestration or complete removal of the mandible. He also recommends the use of a liquid diet and a daily intake of 1750-2000 calories per day, and stresses that this diet is better tolerated in small and repeated feedings. The reason behind this diet is that patients who have osteoradionecrosis due to the pain and disability do not eat. This diet should be instituted as part of the overall treatment so as to bring the patient back to a healthy state. The use of a high caloric and high protein diet with vitamins is encouraged.

Hoffmeister[25] recommends the removal of the diseased bone only, with no need for a safe margin, in contrast to cancer surgery; where you should have a clear or safe margin free of disease. The surgery for osteoradionecrosis should only carry the involved portion of bone and should stop as soon as bleeding bone is reached. The rongeur forcep[22,25] is the instrument of choice, not the saw. Slow, step by step removal of bone is the technique of choice, not radical resection. Soft tissue coverage cannot be overemphasized. It provides not only protection for exposed bone of a low vitality against organisms indigenous to the oral cavity, but it also provides a source of blood supply. Furthermore, Hoffmeister is against the use of coagulation [15,25] of necrotic bone as recommended by Regaud.[6] The super imposition of thermal trauma

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on bone of already lowered vitality is contrary to the basic principles of tissue preservation, and contributes nothing but disaster. Regaud[6] recommends the use of a cautery of the necrotic bone to hasten extension of the necrosis to the site of reactive bone and true involucrum formation. The alternative is surgical excision[28] of the irradiated portion of the mandible at the onset of osteoradionecrosis. This procedure is the shortest, and according to Wildermuth and Cantril,[28] the treatment of choice.

As was mentioned previously, there are authors who recommend a radical treatment for osteoradionecrosis.[8,12,14,16,18,29,38]

MacDougall[12] recommends a radical treatment, more commonly a hemimandibulectomy, because anything less than the removal of the whole of the irradiated area is certain to lead to failure. The operation of choice should be by way of an intraoral approach for the removal of the jaw, as recommended by Marchetta,[16,40] Rankow and Weissman.[19] Dodson[8,29] recommends that to prevent osteoradionecrosis, the teeth, irrespective of their condition, should be removed before irradiation is started. Francisco[14] also recommends that to prevent osteoradionecrosis, the teeth should be removed, but only the diseased teeth and those in the path of radiation. For the treatment of osteoradionecrosis, Francisco states that, "It should start with local measures, but when there is extensive involvment, surgical intervention should be considered and sometimes radical surgery such as the removal

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of the mandible is the only treatment indicated."

As can be seen, the literature is full of reports as to what should be done to prevent and treat osteoradionecrosis. Some of the preventive methods have been discussed, as well as the treatment for osteoradionecrosis. In summary, there are authors who recommend all the teeth be removed prior to radiation, while others recommend that only the diseased teeth be removed, and still others, who maintain that only those teeth in the path of radiation should be removed, and that a radical alveolectomy should be accomplished at the same time. Once the presence of osteoradionecrosis has been established, there are different methods of treatment: conservative, radical and a combination of the two.

As was suggested earlier, an oral surgeon, general dentist, prosthodontist, endodontist and a periodontist should be part of the team when patients with head and neck cancer are being considered for treatment. All members of this team should review the work done by Daly,[27] at the M. D. Anderson Hospital. In his study he divided the patients into four groups: Group I: Edentulous, Group II: Poor dental status, Group III: Fair, primarily the teeth of these patients are restorable by ordinary dental procedures, Group IV: Good; patient with good oral hygiene, few caries, good periodontal condition.

In 6½ years of followup, Daly found that the incidence and severity of bone necrosis has decreased. The incidence is 22% as compared

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to 37% prior to his work. Those patients who had the benefit of fluoride treatment, good oral hygiene and home care instructions exhibited less post-irradiation decay of the teeth than those who did not have the benefit of prophylaxis, home care instruction, and fluoride application. The first management of osteoradionecrosis should be conservative,[27] as we mentioned earlier.

Radical treatment should be instituted only when the conservative method fails and there is intractable pain, recurrent severe infection, or trismus. The treatment may be a partial or total mandibular resection. When possible this should extend outside of the irradiated portion, since the cut end of the bone may also become necrotic.

The following are the results of treatment for osteoradionecrosis. Of 304 patients studied, only 67 patients developed osteoradionecrosis. The methods of treatment were as follows: 1) Conservative treatment - healed 36%. 2) Conservative treatment-still present, 9.5%. 3) Conservative treatment - patients dead, 16%. 4) Jaw surgery with disease, 9.5%. 5) Jaw surgery without disease, 28%. That 36% of the patients who developed osteoradionecrosis healed with conservative treatment is a strong indication of its effectiveness. Usually the earlier the necrosis is diagnosed and the various phases of conservative treatment started, the greater the chance for rapid healing.

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No surgical intervention [27] should be attempted unless there has been continuous failure using conservative procedures. Surgical intervention in irradiated bone may extend the necrotic site to include areas that clinically are not necrotic; therefore, the decision on how much bone should be removed should be made at the time of surgery after careful deliberation. Healing of bone necrosis is promoted if infection is brought under control. If gross infection is not present and the area of bone necrosis is not too great, the body will treat the bone fragments as foreign bodies and seek to sequestrate them. This process may take months or years. The use of topical solutions, antibiotics, and pain medications, coupled with good oral hygiene, and a well balanced diet, are indicated in cases of limited necrosis. If this fails, radical methods for the treatment of osteoradionecrosis may be employed.

Another method of treatment for osteoradionecrosis is hyperbaric oxygenation. Although a discussion of hyperbaric oxygenation is beyond the scope of this paper, it is in order to explain and discuss some of the basics of how it works.

The primary effect of hyperbaric oxygenation is to increase the partial pressure of oxygen in the plasma.[43,44,45,46,47,48,49] Hyperbaric oxygenation is oxygen therapy applied within a pressure chamber under circumstances which provide an alveolar partial pressure of the gas (oxygen) to exceed those possible from inhalation of pure oxygen

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at atmospheric pressure.

The resultant hyperoxemia is intermediate in the production of elevation in tissue tension of oxygen. The objective is the development of an elevated tension of oxygen, specifically within the mitochondria, the presumed site of its most significant cellular activity.

The oxygen content of air is 20.0 volume%.[43,44,45,46,47] This represents a partial pressure of oxygen of 158mm.Hg. The alveolar partial pressure of oxygen is about 100mm.Hg. The total oxygen content of 100cc. of blood is about 20cc. Most of this oxygen is combined with hemoglobin which is about 97% oxygen saturated. Only 0.3cc. of oxygen are dissolved in the plasma if pure oxygen is breathed at normal room conditions. Its alveolar partial pressure reaches 650mm.Hg.[45] At this level, the change in oxygen of arterial blood is from 20.0 % to 22%. This increased partial pressure of oxygen will not significantly affect the transported oxyhemoglobin level of the blood, which is only increased from 97 to 100% saturated, but it will increase the dissolved oxygen content of plasma to a level of approximately 2ml. per 100cc. of blood or 2 volume %. This change is a 700% increase in the dissolved oxygen and equals 10% of the oxygen carried by the hemoglobin. Further increases in the partial pressure of oxygen serves only to increase the dissolved oxygen in the plasma.

When a subject is placed in an environment at two atmospheres of

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pressure, the partial pressure of oxygen will increase from 760mm.Hg. to 1520 mm.Hg. This will increase the dissolved oxygen content of plasma to a level of 4cc. per 100cc. of plasma, with no additional increase in the oxygen carried by hemoglobin. If the pressure is increased to three atmospheres, the partial pressure of oxygen will increase to 2280mm.Hg. (breathing pure oxygen). At this partial pressure, the increase of dissolved oxygen will be from 4cc. to 6cc. per 100cc. of blood. According to Pascale,[45] this dissolved oxygen can provide most, if not all of the oxygen extracted by the brain, the visceral organs, the kidneys, and the inactive muscles. A large portion of the oxygen extracted by the myocardium can be supplied by this unbound (dissolved) oxygen. At these elevated partial pressures of oxygen, the diffusion of oxygen into areas of hypoxia and anoxia is facilitated so that oxygenation in regions of anaerobic phlegmon, anaerobic malignant tissue, or ischemic areas is more readily achieved.

Some of the contraindications to hyperbaric oxygenation therapy are limited ventilation, pulmonary edema, pulmonary fibrosis, and intra or extra pulmonary right to left shunts. The main problem with hyperbaric oxygenation therapy is that of oxygen toxicity, a complex phenomenon which is still an enigma. Some authors, such as Captain George B. Hart,[46] thoracic surgeon, recommend the use of vitamin E or disulfiram (Antabuse) to prevent or diminish the oxygen toxicity. This toxicity[43,44] can be manifested by central nervous system signs, changes in visual

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acuity, rising of diastolic blood pressure, facial pallor, dilatation of the pupils, dizziness, nausea, convulsions, seizures and death. The following are some of the clinical applications of hyperbaric oxygenation:[43,44,45,46,47] 1) Anaerobic infection (gas gangrene). 2) Cardiac surgery. 3) Peripheral arterial insufficiency. 4) Vascular surgery. 5) Burns. 6) Osteomyelitis and osteoradionecrosis. 7) Carbon monoxide poisoning. 8) Cassion disease (compressed air illness).

Some authors[49,50,51] recommend treatment of osteoradionecrosis with hyperbaric oxygenation. Boyne[49] reported treating four cases of osteoradionecrosis with hyperbaric oxygenation. He states that:

> "Vascular fibrous tissue advances from the edges of the living marrow of the viable bone into the intertrabecular spaces of the dead bone, as the vascular fibrous tissue advances rapid resorption of necrotic bone takes place without suppuration and new bone is laid down along the trabeculae of the nonviable bone."

A distinct increase in arterial and venous oxygen pressure can be demonstrated during hyperbaric oxygenation. The direct effect of oxygen on cell mitosis and cell proliferation in areas of tissue repair results in favorable changes in the healing environment. In another report by Boyne, he cited eleven cases of osteoradionecrosis treated with hyperbaric oxygenation. The usual treatment of these cases consisted of the evacuation of pus, debridement, sequestrectomy when indicated, antibiotic therapy, daily irrigation with aminoacridine, and hyperbaric oxygenation. Vitamin E was given daily as part of the treatment to all of

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the patients. The daily dose was 100mg. per day.

The treatment with hyperbaric oxygenation is usually done with 100% pure oxygen and two atmospheres of pressure, one or two hours per day for thirty days. At the present time, this has been changed and the therapy consists of 80-120 hours of treatment; this can be regulated by the resolution of the disease. The use of hyperbaric oxygenation therapy[48,49,50,51] appears to have a definite place in the future management of patients suffering from osteoradionecrosis.

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CHAPTER V

SUMMARY AND CONCLUSIONS

A review of radiation, its theories, and the probable mechanism of radiation effects on cells has been presented. Osteoradionecrosis of the jaws is a complication encountered in patients who received radiation for the treatment of head and neck cancer. Its incidence is directly related to the radiation dose, tumor size, and site(primary), field size and the number of irradiated fields used, oral hygiene, dental condition of the patient, dental trauma, radical neck surgery, and infection. All of these factors were discussed with regard to the relation of each to the incidence of osteoradionecrosis.

The best treatment of osteoradionecrosis is the prevention of it. This could be accomplished with a thorough understanding of the disease. Different means of prevention were discussed, these included the extraction of teeth with radical alveolectomies prior to radiation treatment, restoration of dental health, use of fluorides, intensive oral hygiene as well as close followup, and sound treatment planning.

Treatment of osteoradionecrosis was discussed. The first management of osteoradionecrosis should be conservative. This includes the use of irrigations such as zinc peroxide, hydrogen peroxide, aminoacridine, neomycin solutions, soda irrigations, and systemic antibiotics. If

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conservative treatment fails, radical treatment should be considered. This includes sequestrectomy, saucerization, hemimandibulectomy and total mandibulectomy.

A new method for the treatment of osteoradionecrosis, that of hyperbaric oxygenation, was discussed: its application and mode of action were explained. This should be considered in the treatment of osteoradionecrosis.

In the treatment of head and neck cancer patients, each case must be evaluated and treated individually. No two cases are the same, and should not be treated equally.

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BIBLIOGRAPHY

1. Dorland: Medical Dictionary. W. B. Saunders Co., Vol. 23, 1957.
2. Shafer, Hine, Levy: Oral Pathology. W. B. Saunders Co., June, 1961.
3. Ham, A.: Histology. J. B. Lippincott Co., Seventh Ed., 1974.
4. Anderson, W. A.: Pathology. C. V. Mosby Co., Fourth Ed., 1961.
5. Schwartz, E. E.: The Biological Basis of Radiation Therapy. J. B. Lippincott Co., 1966.
6. Regaud, C., and Ferroux, R.: Protection against roentgen injury. Bull. Acad. de. med. Par. 87:280, March 7, 1922.
7. Kanthak, F. F.: X-ray irradiation and osteonecrosis of the jaws. J. Am. Dent. A. 28: 1925-1929, 1941.
8. Cook, T. J.: Late radiation necrosis of the jaw bones. J. O. S. Vol. 10: 118-137, April, 1952.
9. LaDow, C. S.: Osteoradionecrosis of the jaw. Oral Surg., Oral Med., and Oral Path. 3: 582-590, May, 1950.
10. Watson, W. L. and Scarborough, J. E.: Osteoradionecrosis in intraoral cancer. Am. J. Roentgenol. 40: 524-534,

Digitized by Google

October, 1938.

11. Thoma, K. H.: Oral Surgery. C. V. Mosby Co., Fourth Ed., 713-718.

12. MacDougall, J. A., Evans, A. M., and Lindsay, R. K.: Osteoradionecrosis of the mandible and its treatment. Am. J. Surg. 106: 816-818, 1963.

13. Scanlon, P. W.: Split dose radiotherapy. J. A. M. A. Vol. 220, #3: 400-401, April, 1972.

14. Francisco, J. V.: Osteoradionecrosis of the jaws. J. O. S. Vol. 24: 247-252, May, 1966.

15. Dodson, W. S.: Irradiation osteomyelitis of the jaws. J. Oral Surg., Anesth. and Hosp. D. Serv. Vol. 20: 467-474, November, 1962.

16. Marchetta, F. C., and Solomon, H. A.: Osteoradionecrosis of the mandible. J. O. S. Vol. 16, #1: 69-72, January, 1958.

17. Niebel, H. H. and Neenan, E. W.: Dental aspects of osteoradionecrosis. Oral Surg., Oral Med., and Oral Path. Vol. 10, #10: 1011-1024, October, 1957.

18. Grant, B. P. and Fletcher, G. H.: Analysis of complications following megavoltage therapy for squamous cell carcinoma of the tonsillar area. Am. J. Roentgenol, Radium. Ther. Nucl. Med. 96: 28-36, January, 1966.

19. Rankow, R. M. and Weissman, B.: Osteoradionecrosis of the mandible. Ann. Otol. #80: 603-610, 1971.

Digitized by Google

20. Chambers, F., Ng, E., Ogden, H., Coggs, G., and Crane, J.: Mandibular osteomyelitis in dogs following irradiation. Oral Surg., Oral Med., and Oral Path. Vol. 11, #8: 843-859, August, 1958.

21. Topazian, D. S.: Prevention of osteoradionecrosis of the jaws. Oral Surg., Oral Med., and Oral Path. Vol.12, #5: 530-538, May, 1959.

22. Robinson, D. W., Masters, F. W., and Ketchum, L. D.: Management of osteoradionecrosis. J. Am. A. Vol. 217, #7: 950-951, August, 1971.

23. Niebel, H. H., Neenan, E. W., Walsh, R. P. and Weimer, J. B.: Removal of teeth from irradiated tissue. J. O. S. Vol. 15: 313-319, October, 1957.

24. Hinds, E. C.: Dental care and oral hygiene before and after treatment, radiation caries. J. Am. A. Vol. 215, #6: 964-966, February, 1971.

25. Hoffmeister, F. S., Macomber, W. B., and Wang, M. K. H.: Radiation in dentistry-surgical comments. J. A. D. A. Vol. 78: 512-516, March, 1969.

26. Meyer, I., Shklar, G., and Turner, J.: A comparison of the effects of 200KV. radiation and cobalt-60 radiation on the jaws and dental structure of the white rat. Oral Surg., Oral Med., and Oral Path. Vol. 15, #9: 1098-1108,

Digitized by Google

September, 1962.

27. Daly, T. E.: Management of dental problems in irradiated patients. Refresher course, M. D. Anderson Hospital Tumor Institute at Houston, Texas. 1972.

28. Wildermuth, C. and Cantril, S. T.: Radiation necrosis of the mandible. Radiology. 61: 771-785, November, 1953.

29. Rominger, C. J., Looby, J. and Duncan, J.: Role of alveolectomy in prevention of radionecrosis of the jaws and oral soft tissues. J. Oral Surg., Anesth. and Hosp. D. Serv. Vol. 20: 72-77, January, 1962.

30. Beumer, J., Silverman, S., and Benak, S. B.: Hard and soft tissue necroses following radiation therapy for oral cancer. J. Prosthetic Dentistry. Vol. 27, #6: 640-644, June, 1972.

31. Cook, T. J.: Osteomyelitis and osteoradionecrosis. Oral Surg., Oral Med., and Oral Path. Vol. 16, #3: 257-260, March, 1963.

32. Narang, R. and Wells, H.: The avoidance of osteoradionecrosis of the mandible after extraction of a number of teeth in a patient given radiotherapy for oral carcinoma. J. O. S. Vol. 29, #5: 656-659, May, 1970.

33. DelRegato, J. A.: Dental lesions observed after roentgen therapy in cancer of the buccal cavity, pharynx and larynx. Am. J. Roentgenol Radium Ther. Nucl. Med. 42: 402, 1939.

Digitized by Google

34. Daly, T. E., Drane, J. B., and MacComb, W. S.: Management of problems of the teeth and jaw in patients undergoing irradiation. The Am. J. of Surgery. Vol. 24, October, 1972.

35. Daly, T. E.: Radiation complications in head and neck cancer. Cancer Bulletin, 20: 90-91, 1968.

36. Shearer, H. T.: Effect of cobalt-60 radiation on extraction healing in the mandibles of dogs. J. O. S. Vol. 25: 115-121, March, 1967.

37. Fayos, J. V.: Management of squamous cell carcinoma of the floor of the mouth. The Am. J. of Surgery. Vol. 123: 706-711, June, 1972.

38. Guttenberg, S. A.: Osteoradionecrosis of the jaw. The Am. J. of Surgery. Vol. 127: 326-332, March, 1974.

39. Alexander, R., Elzay, R. P., Dettman, P., and King, R.: Tetracyclines for controlling osteoradionecrosis in rat mandibles. J. O. S. Vol. 25: 503-509, November, 1967.

40. Marchetta,F. C., Sako, K., and Holyoke, E. D.: Treatment of osteoradionecrosis by intraoral excision of the mandible. Surgery Oynecology and Obstetrics. 1003-1008, November, 1967.

41. Marciani, R. D., and Bowden, C. M.: Osteoradionecrosis of the maxillae. J. O. S. Vol. 31: 56-58, January, 1973.

Digitized by Google

42. Freestone, J. T., Look, F., and Caulder, S. L.: Intraoral mandibular resection for osteoradionecrosis. J. O. S. #31: 861-864, November, 1973.

43. Pittinger, C. B.: Hyperbaric Oxygenation. Charles C. Thoma Pubs., 1966.

44. Fundamentals of hyperbaric oxygenation. National Academy of Sciences. Washington, D. C., 1966.

45. Pascale, L. R., and Wallyn, R. J.: Surgical applications of the hyperbaric chamber. Surgical Clinics of North America. Vol. 48, #1: 63-70, February, 1970.

46. Whieldon, D.: So what happened to hyperbaric oxygenation? Hospital Physician. #5: 76-81, 1973.

47. Sippel, H. W. and Nyberg, C. D.: Hyperbaric Oxygenation as an adjunct to the treatment of chronic osteomyelitis of the mandible. J. O. S. 27: 739, 1969.

48. Mainous, E. G., Boyne, P. S., Hart, G. B., and Terry, B. C.: Restoration of resected mandible by grafting with combination of mandible homograft and autogenous iliac marrow, and post-operative treatment with hyperbaric oxygenation. Oral Surg., Oral Med., Oral Path. 13-20, January, 1973.

49. Mainous, E. G., Boyne, P. S., and Hart, G. B.: Elimination of sequestrum and healing of osteoradionecrosis of the mandible after hyperbaric oxygenation therapy. J. O. S. Vol.

Digitized by Google

31, #5: 336-339, May, 1973.

50. Irby, W. B.: Current Advances in Oral Surgery. C. V. Mosby Co., 242-263, 1974.

51. Mainous, E. G., Boyne, P. G., and Hart, G. B.: Hyperbaric oxygen treatment of mandibular osteomyelitis. J. A. D. A. Vol. 87: 1426-1430, December, 1973.

52. Carl, W., Schaaf, N. G., and Sako, K.: Oral Surgery and the patient who has radiation therapy for head and neck cancer. Oral Surg., Oral Med., and Oral Path. Vol. 36, #5: 651-657, November, 1973.

53. Woodward, H. Q., and Coleg, B. I.: The correlation of tissue dose and clinical response in irradiation of bone tumors and of normal tumor. Am. J. Roentgenol Radium Ther. Nucl. Med. 57: 464-471, April, 1947.

54. Ozarda, A. T.: Complication of irradiation for mammary carcinoma. Southern Medical Journal. Vol. 63, #3: 369-370, March, 1972.

55. Kruger, G.: Oral Surgery. 587, C. V. Mosby Co., Fourth Ed.

56. Ercison, B. K.: Osteoradionecrosis of the jaws. Thesis presented to the faculty of the graduate school of the University of Minesota, August, 1965.

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A COMPARISON OF THE GLUCOSE AND TOTAL SOLUBLE PROTEIN OF GINGIVAL FLUID FROM DIABETICS AND NONDIABETICS

by

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Chapter I

Review of the Literature

Since the time Brill first observed the passage of tissue gluid into the gingival pocket,[1-4] gingival fluid has been the object of considerable research. Its basic nature is still under discussion by different investigators;[5-9] while its significance has been investigated in relation to gingival inflammation and periodontal disease,[5-19] serum proteins[20-24] and cellular contents,[25-26] and its relation to pregnancy and the menstrual cycle.[27-29] Since little emphasis has been placed on the relation between gingival fluid and systemic diseases, this study was set up to compare the glucose and total soluble protein content of gingival fluid from diabetic and nondiabetic patients.

Hara and Löe[30] studied the presence of free glucose in gingival fluid, and Bang and Cimasoni[31] determined the total protein content, both studies being performed, however, on normal (nondiabetic) patients. Much work has been done on the nature of diabetic gingiva as compared to nondiabetic with light and electron microscopy revealing marked increase in the thickness of capillary basement membranes (as much as four times) and the thickness of small vessel walls in diabetics with disorientation of cells in the vessel walls.[32-34] Since diabetic gingiva does not utilize oxygen and thus oxidize glucose as readily as nondiabetic gingiva, a fact particularly important to gingival fluid study, Rabinowitz, et. al.[36] pointed out that the gingiva from both diabetics (untreated) and nondiabetics does not possess glycogen as

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part of the polysaccharide component. All these factors, especially the decreased oxygen diffusion and the accumulation of metabolic wastes, have been implicated in the severity of periodontal disease changes in diabetics.[34-35] The number of years a person has been a diabetic is also an important consideration[37-38] due to decreased resistance of the diabetic patient.[39]

The values used to define the diabetic state are agreed upon by most authors, with this study utilizing the two-hour postprandial blood glucose determination, in which any value for circulating glucose of 100 mg. or less per 100 ml. of blood was considered normal or nondiabetic.[40] Since most investigators agree that the upper limit for normal is 120 mg. percent,[38] 100 mg. percent should allow for a reasonable margin of error.

The gingival sites for collection of gingival fluid and the actual technique used was that employed by Kaslick, et. al.[11-13] Maxillary premolars and molars were used, because of ease of isolation of these areas, the faster descending flow rate of gingival fluid[14] from the crevice or "capillary space,"[41] and because the use of capillary tubes[5, 11, 16] for collection made the technique more easily accomplished.

The collection sites were scored according to Löe,[15, 38] each site being given both a gingival and a plaque index. Most writers concur that there is a high correlation between clinical scoring and the amount and rate of fluid flow and the inflammatory state of the tissue, the more inflamed the tissue, the greater the flow.[7, 10, 12, 16, 17, 34, 37] Orban and Stallard,[18] however, feel that the plaque index alone is sufficient and, in their estimate, a more accurate determination of the gingival state.

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Glavind, et. al.[37] observed that the GI and PlI were the same for the diabetic group, while Cohen,[39] in a two-year longitudinal study of diabetes mellitus and periodontal disease, observed higher GI scores and decreased PlI scores (less soft deposits) in the diabetic group. Thus, to eliminate confusion, both indices were used to classify the collection sites.

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Chapter II

Materials and Methods

The study samples consisted of eleven patients in the diabetic and ten patients in the nondiabetic groups for a total of 21 patients. The study samples consisted primarily of male patients with the exception of three female patients in the diabetic group. Since the amount of fluid flow as related to hormonal influences[18] as observed during menstruation,[29] pregnancy, or with women taking oral contraceptives[27] was not a consideration here, nevertheless, it should be noted that none of the female patients were pregnant nor taking oral contraceptives at the time of gingival fluid collection. The ages of the patients ranged from 22 to 65 years with a mean age of 45.

Those patients who presented with excellent medical health and were not taking any drugs of any sort were selected for the nondiabetic group. Additionally and as an added precaution, these patients were instructed not to take any medications during the 24 hour period prior to gingival fluid collection. The diabetic group, on the other hand, consisted of patients presenting with diabetes mellitus, with varied degrees of severity, types of control regimes, and lengths of time as a known diabetic. The control regimes varied from simple diet control to diet control plus insulin (or various combinations). Also, several of the patients utilized were newly diagnosed diabetics--namely, within the two weeks prior to gingival fluid collection. The individual control regimes were not altered in any way

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prior to gingival fluid collection.

All patients underwent a two-hour postprandial blood glucose determination to separate the two groups[34] and to establish a blood glucose at the time of gingival fluid collection. The control group (nondiabetic) values were considered normal if they were 100 mg. percent or less, while the diabetic group values were accepted if they were 120 mg. percent or greater,[40] since they were known diabetics following a specified control regime. The two-hour postprandial was performed in the morning following an overnight fast of twelve hours. The patient presented himself and consumed a breakfast of 100 gm. of carbohydrate and two hours later a single blood sample was drawn to determine the blood glucose. At the same time a separate blood specimen was drawn from which the total soluble protein was determined.

The fluid was then collected immediately after the two-hour postprandial tests were performed in order to maintain the same glucose conditions. Gingival fluid was collected from the interproximal areas of maxillary premolars and molars with the exception of one maxillary lateral incisor (control group patient) using glass capillary tubes as proposed by Kaslick.[11] Before the fluid was collected, however, a clinical evaluation of the degree of gingival inflammation in those areas from which fluid was to be collected was made, using the method patterned after the Gingival and Plaque Indices of Löe.[15] The Gingival Index (GI) system is as follows:

"0 = normal gingiva.
1 = mild inflammation - slight change in color, slight edema. No bleeding on probing.

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2 = moderate inflammation - redness, edema and glazing. Bleeding on probing.
3 = severe inflammation - marked redness and edema. Ulceration. Tendency to spontaneous bleeding."[15]

The Plaque Index (PlI) is as follows:

"0 = no plaque in the gingival area.
1 = a film of plaque adhering to the free gingival margin and adjacent area of the tooth. The plaque may only be recognized by running a probe across the tooth surface.
2 = moderate accumulation of soft deposits within the gingival pocket, on the gingival margin and/or adjacent tooth surface, which can be seen by the naked eye.
3 = abundance of soft matter within the gingival pocket and/or on the gingival margin and adjacent tooth surface."[15]

Each collection site, therefore, was given a GI and PlI value. Only those areas that had a Gingival Index of 1 or 2 were used, with no tissue rating of "nearly normal" (GI = 1) unless the pocket depth was less than 4 mm.[12]

The following technique used for the collection of the fluid was patterned after Kaslick's method.[11] Glass capillary tubes of known internal diameter and length (Unopette System) were placed at the opening of the gingival pocket (Figure 1), (once the area had been isolated, wiped clean of plaque, and the pocket dried with a gentle "blast" of air) care being taken to avoid trauma to the gingiva. Slight finger pressure was applied to the tooth to simulate physiologic function,

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with all tubes tinged with blood being discarded (Figure 2),[11] and additional fluid then being collected using a new tube.

Once the fluid was collected the tube was plugged at one end and centrifuged for five minutes in a micro-hematocrit centrifuge at 12,500 rpm. to remove cellular elements and debris. The tubes were then scored at the junction of the sediment and supernatant fluid, snapped apart, and the portion containing the supernatant fluid was placed into a holder called a flag. The fluid column was then measured (from meniscus to meniscus) with a Boley gauge to the nearest tenth of a millimeter, the volume of the fluid then being calculated (one tube = 10 ul.).[11]

The fluid was then transferred from the tubes to reservoirs containing 1.50 ml. of deionized water. The reservoirs were then stored in a frozen state to avoid breakdown of the glucose by the bacteria present. At a convenient time the fluid was thawed and analyzed for glucose and total soluble protein. The glucose was analyzed by a specific fluorometric direct assay technique,[42] and the protein determined by the Lowry technique (micro) for protein measurement with the folin phenol reagent.[43]

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FIGURE 1. The capillary tube collecting gingival fluid from within the gingival pocket interproximally between the maxillary left first and second premolars.

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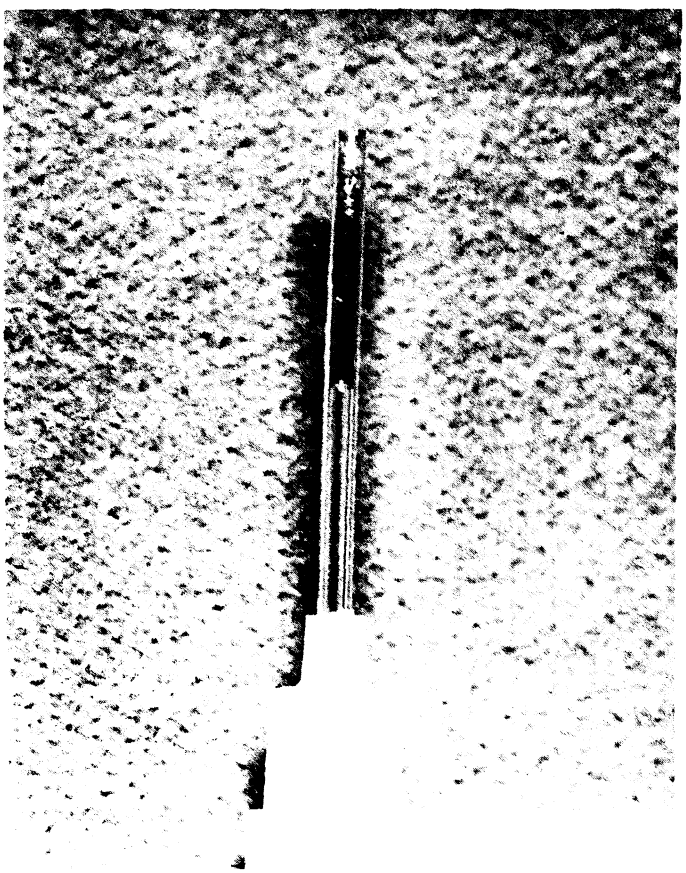

FIGURE 2. Gingival fluid in a glass capillary tube immediately after collection and before plugging and centrifuging. The appearance of the fluid is generally clear or slightly straw colored.

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Chapter III

Results

The two groups were evaluated statistically to determine whether differences existed relative to age, gingival fluid glucose, and gingival fluid total soluble protein. The statistical tests used were the Student's t-test employing a two-tailed determination and Pearson's product moment correlation or r.

The ages of the patients utilized for the study ranged from 22 to 65 years with a mean age of 45 and showed no significant statistical difference with $t = .55$ at $df = 19$. There was also no significant statistical difference relative to gingival fluid protein with $t = .41$ at $df = 19$. The values obtained for gingival fluid glucose, however, did show a significant difference. The level of statistical significance obtained or p value was less than .001 with $t = 5.074$ at $df = 19$. (Table 1)

The Pearson's r values for protein showed very little difference with $r = .28$ for the control group as compared to $r = .26$ for the experimental group, neither being significant. The slope and intercept values also varied very slightly. The glucose values, however, again showed a wide range of difference. The r for the control group was equal to .29 with a .75 for the experimental. While the $r = .29$ was not significant, the $r = .75$ showed a p value less than .01. The corresponding slope and intercept values also varied greatly. These values were utilized to plot respective regression curves for gingival fluid glucose and gingival fluid protein (Figure 3). The

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ordinate or y values were calculated based on the known abscissa or x values. Gingival glucose or protein was plotted on the abscissa with blood glucose or protein on the ordinate.

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	Experimental Group Diabetic							Control Group Nondiabetic						
				Blood		(X) Gingival Fluid		(X) Gingival Fluid		Blood				
Sample No.	GI	PlI	pocket depth mm.	glucose mg%	total soluble protein gm%	glucose mg/ul	total soluble protein ug/ul	total soluble protein ug/ul	glucose mg/ul	total soluble protein gm%	glucose mg%	pocket depth mm.	PlI	GI
1	2	3	4.0	134	6.3	.971	40.41	56.50	.648	7.5	89	5.0	0	2
2	0	1	2.0	236	7.1	2.336	115.69	109.27	1.342	7.2	94	5.0	2	2
3	1	1	2.5	128	6.7	1.560	87.92	106.29	1.117	6.3	96	3.0	1	1
4	1	1	3.0	260	7.5	2.022	59.85	100.86	.932	7.2	90	3.0	2	2
5	2	1	3.0	142	7.3	1.840	106.65	84.19	.793	7.2	90	3.0	1	1
6	2	2	4.0	148	7.3	1.635	90.72	148.56	.777	7.9	99	3.0	1	1
7	1	3	2.5	183	7.5	2.253	94.44	69.42	.633	7.2	88	3.0	1	2
8	2	3	3.0	124	8.6	1.126	88.63	91.95	.851	8.2	99	4.0	0	2
9	1	0	3.0	310	7.2	1.689	179.46	99.82	.826	7.4	90	3.0	2	1
10	2	3	3.0	320	7.8	1.871	83.04	56.25	.814	6.6	98	2.5	2	1
11	2	2	2.0	1020	8.5	3.023	134.21							

TABLE 1. Blood and gingival fluid glucose and total soluble protein findings with corresponding GI, PlI, and pocket depth recordings.

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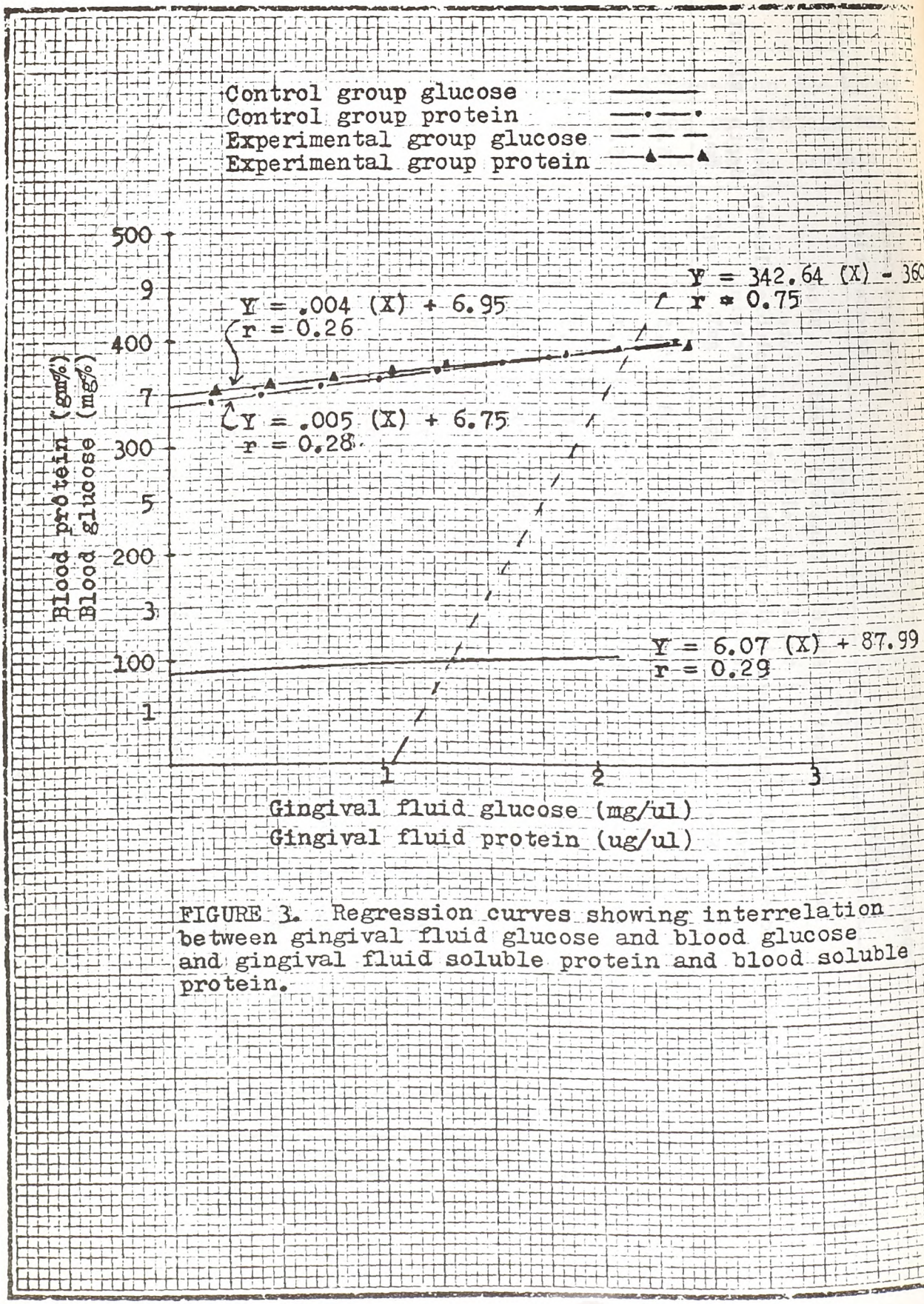

FIGURE 3. Regression curves showing interrelation between gingival fluid glucose and blood glucose and gingival fluid soluble protein and blood soluble protein.

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Chapter IV

Discussion

The purpose of this study as initially described was to compare the glucose and total soluble protein of gingival fluid from diabetics and nondiabetics. Inherent in this purpose also was the intent to determine a possible screening test that could be used to aid in the detection of previously undiagnosed diabetes; and to investigate one aspect as regards the etiology of periodontal disease.

The results described show a strong correlation between blood glucose and gingival fluid glucose with protein showing little difference if any between blood and gingival fluid content. It should be noted further that the techniques used to determine the glucose and protein content were very specific permitting one to determine minute concentrations thus establishing the above noted relationships. Also, sites marked by moderate gingival changes were chosen for fluid collection. Normal clinically healthy gingival tissue could not be used, since it was most difficult to collect fluid with capillary tubes from these sites.

The protein was evaluated as well as the glucose in order to determine whether the vascular changes as noted in patients with diabetes mellitus[32-34] would have any effect on the protein content of gingival fluid. The fact that gingival fluid proteins and serum proteins are similar has been well substantiated in the literature.[20-24] It was, therefore, necessary to determine what fraction of the fluid collected was protein and glucose respectively.

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The relative importance of glucose in gingival fluid is significant not just with regard to developing a possible screening test, but also with regard to the environment of periodontally diseased teeth. Stein and Nebbia[44] performed a screening study using firm cellulose strips impregnated with glucose oxidase and a chromogenic indicator system. One drop of gingival blood was placed on the strip and allowed to "sit" for one minute. The color and intensity was then compared to a color chart. The test can indicate values from 40 to 250 mg. percent glucose, and showed a 2.4 percent detection of incipient disease. Utilizing this concept one might place impregnated stripes into the gingival pocket for a specified time and then check the strip to determine gingival glucose and subsequently, blood glucose.

While this study showed a significant relationship between blood glucose and gingival fluid glucose, Hara and Löe[30] expressed disappointment with their study's results. They attempted to determine free glucose concentrations in gingival fluid and felt that their results were influenced by local environmental factors -- such as the local microflora and the liberation and activation of hydrolyzing enzymes. Gingival glucose concentrations of 3-6 times that of serum were noted with no definite correlation.

Bang and Cimasoni,[31] on the other hand, studying total soluble protein in human gingival fluid yielded results concurring very favorably with this study's findings. Bang and Cimasoni obtained gingival fluid protein values which compared to serum values with no significant difference in concentrations. These findings concerning protein are significant in that the relationships remained unchanged for both normal and diabetic

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patients, irrespective of the latter presenting with obvious vascular changes.

The fact that diabetic gingiva is unable to utilize oxygen and thus oxidize glucose as readily[35] supports this study's findings of high concentrations of glucose in the gingival fluid. These findings together with the cell's impaired ability to completely regulate glucose by insulin, whether endogenous or exogenous, also contributes to the presence of increased glucose in the gingival pocket. The importance of this glucose content relative to the etiology of periodontal disease may rest in several areas. Glucose may contribute as a substrate for the production of certain acids or enzymes by microorganisms, or, it may play a role in plaque metabolism. These factors, then, together with the established fact that a diabetic's resistance to infection is decreased, may play an active role in the development of severe periodontal disease often noted in these patients.

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Chapter V

Summary

Gingival fluid glucose and total soluble protein from diabetics and nondiabetics were compared. The findings showed a high correlation between the gingival fluid glucose and the blood glucose of diabetics with little or no difference between the protein levels. This high correlation for gingival fluid glucose thus suggests a possible screening test for diabetes detection technique utilizing a specially impregnated filter paper strip placed in the gingival pocket for a specified length of time. A positive test would strongly indicate the necessity for more extensive laboratory workup.

The possible role of increased glucose in the gingival pockets of diabetics was also evaluated with regard to the possible etiologic significance of increased severity of periodontal disease as often manifested by these patients.

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BIBLIOGRAPHY

1. Brill, N. and Krasse, B.: The passage of tissue fluid into the clinically healthy gingival pocket. Acta Odont. Scand. 16:223-245, 1958.
2. Brill, N. and Björn, H.: Passage of tissue fluid into human gingival pockets. Acta Odont. Scand. 17:11-21, 1959.
3. Browne, R. M.: A preliminary study of the fluid flow from the gingival sulcus. Proc. Roy. Soc. Med. 55:486-8, 1962.
4. Browne, R. M.: Some observations on the fluid flow from the gingival crevice. Dent. Pract. 14:470, 1964.
5. Krasse, B., and Egelberg, J.: The relative proportions of sodium, potassium, and calcium in gingival pocket fluid. Acta Odont. Scand. 20:143-52, 1962.
6. Löe, H.: Physiology of the gingival pocket. Acad. Rev. 13:6-14, 1965.
7. Löe, H. and Holm-Pedersen, P.: Absence and presence of fluid from normal and inflamed gingivae. Periodontics. 3:171-7, 1965.
8. Sueda, T., Imagawa, Y., and Araya, S.: Tissue fluid flowing into gingival pocket. Bull Tokyo Med. Dent. Univ. 12:159-67, 1965.
9. Weinstein, E., Mandel, I. D., Salkind, A., Oshrain, H. I., and Pappas, G. D.: Studies of gingival fluid. Periodontics. 5:161-6, 1967.
10. Björn, A. L.: Evaluation of gingival fluid measurements. Odont. Rev. (Malmo) 16:300-7, 1965.

Digitized by Google

11. Kaslick, R.S., Chasens, A.I., Weinstein, D., and Waldman, R.: Ultramicromethod for the collection of gingival fluid and quantitative analysis of its sodium content. J. Dent. Res. 47:1192, 1968.

12. Kaslick, R.S., Chasens, A.I., Mandel, I.D., Weinstein, D., Waldman, R., Pluhar, T., and Lazzara, R.: Quantitative analysis of sodium, potassium and calcium in gingival fluid from gingiva in varying degrees of inflammation. J. Perio. 41:93-97, 1970.

13. Kaslick, R.S., Chasens, A.I., Mandel, I.D., Weinstein, D., Waldman, R., Pluhar, T., and Lazzara, R.: Sodium, potassium and calcium in gingival fluid. A study of the relationship of the ions to one another to circadian rhythms, gingival bleeding, purulence, and to conservative periodontal therapy. J. Perio. 41:442-8, 1970.

14. Leirskar, J.: In vitro experiments on gingival exudate measurements. II. The influence of different filter papers and ascending and descending chromatography on mobility. J. Perio. Res. 6:23-27, 1971.

15. Löe, H.: The gingival index, the plaque index and the retention index systems. J. Perio. 38:610-16, 1967.

16. Mann, W.V.: The correlation of gingivitis, pocket depth and exudate from the gingival crevice. J. Perio. 34:379-87, 1963.

17. Oliver, R.C., Holm-Pedersen, P., and Löe, H.: The correlation between clinical scoring, exudate measurements, and microscopic evaluation of inflammation in the gingiva. J. Perio. Res. 4:160, 1969.

Digitized by Google

18. Orban, J. E., and Stallard, R.E.: Gingival crevicular fluid: A reliable predictor of gingival health. J. Perio. 40:231-235, 1969.

19. Weinstein, E., and Mandel, J.D.: The fluid of the gingival sulcus. J.A.S.P. 2:147-53, 1964.

20. Brandtzaeg, P.: Immunochemical comparison of protein in human gingival pocket fluid, serum, and saliva. Arch Oral Biol. 10:795-803, 1965.

21. Brill, N.: Influence of capillary permeability on flow of tissue fluid into gingival pockets. Acta Odont. Scand. 17:23-33, 1959.

22. Brill, N. and Brönnestam, R.: Immuno-electrophoretic study of tissue fluid from gingival pockets. Acta Odont. Scand. 18:95-100, 1960.

23. Mann, W.V., and Stoffer, H.R.: The identification of protein components in fluid from gingival pockets. Periodontics. 2:263, 1964.

24. Sueda, T., Cimasoni, G., and Held.: Histochemical study of human gingival fluid. Parodontologic. 20:141-7, 1966.

25. Egelberg, J.: Cellular elements in gingival pocket fluid. Acta Odont. Scand. 21:283-7, 1963.

26. McMillan, L., Burrell, D.Y., and Fosdick, L.S.: Some observations on the exudate from periodontal pockets. J.A.D.A. 57:484-7, 1958.

27. Lindhe, J., Attström, R., and Björn, A.: The influence of progestogen on gingival exudation during menstrual cycles. A longitudinal study. J. Perio. Res. 4:97-102, 1969.

28. Holm-Pedersen, P., and Löe, H.: Flow of gingival exudate as related to menstruation and pregnancy. J. Perio. Res. 2:13-20, 1967.

Digitized by Google

29. Lindhe, J. and Attström, R.: Gingival exudation during the menstrual cycle. J. Perio. Res. 2:194-198, 1967.

30. Hara, K., and Löe, H.: Carbohydrate components of the gingival exudate. J. Perio. Res. 4:202-7, 1969.

31. Bang, J. S. and Cimasoni, G.: Total protein in human crevicular fluid. J. Dent. Res. 50:683, 1971.

32. Campbell, M. J.: A light and electron microscope study of blood vessels from the gingival tissues of nondiabetic and diabetic patients. Aust. Dent. J. 16:235-9, 1971.

33. Frantzis, T., Reeve, C.M., and Brown, A.L. Jr.: The ultrastructure of capillary basement membranes in the attached gingiva of diabetic and nondiabetic patients with periodontal disease. J. Perio. 42:406-11, 1971.

34. Howe, K., and Stallard, R.: Diabetes and the periodontal patient. J. Perio. 41:713-8, 1970.

35. Campbell, M. J.: The oxygen utilization and glucose oxidation rate of gingival tissue from nondiabetic and diabetic patients. Arch Oral Biol. 15:305-10, 1970.

36. Rabinowitz, J. L., Brayer, L., Bailey, T.A., Shelton, J. W., and Cohen, D.W.: Polysaccharides and the absence of glycogen in the gingiva from normal and diabetic patients. Arch Oral Biol. 15:1307-14, 1970.

37. Glavind, L., Lund, B., and Löe, H.: The relationship between periodontal state and diabetes duration, insulin dosage and retinal changes. J. Perio. Res. 4:164-5, 1969.

Digitized by Google

38. Löe, H.: Endocrinologic influences on periodontal disease, pregnancy, and diabetes mellitus. Ala. J. Med. Sci. 5:336-48, 1968.

39. Cohen, D.W., Friedman, L.A., Shapiro, J., Kyle, G.C., and Franklin, S.: Diabetes mellitus and periodontal disease: Two-year longitudinal observations. Part I. J. Perio. 41:709-12, 1970.

40. Todd-Sanford, Clinical Diagnosis By Laboratory Methods, Edited by Israel Davidsohn and John Bernard Henry. W. B. Saunders Company, Philadelphia, 1969.

41. Gustafsson, G. T. and Nilsson, I. M.: Fibrinolytic activity in fluid from gingival crevice. Proc. Soc. Exp. Biol. Med. 106:277-80, 1962.

42. Lowry, O.H. and Passonneau, J. V., A Flexible System Of Enzymatic Analysis, Academic Press Inc., New York, 1972.

43. Lowry, O. H., Rosebrough, N. J., Farr, A.L., and Randall, R. J.: Protein measurement with the folin phenol reagent. J. Biol. Chem. 193:265-275, 1951.

44. Stein, G.M. and Nebbia, A.A.: A chairside method diabetic screening with gingival blood. Oral Surg. 27:607-12, 1969.

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A HISTOLOGIC STUDY OF THE SUPERIOR LABIAL FRENUM

by

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Professional paper submitted in partial fulfillment of the requirements of the U. S. Army Dental Residency Program, Walter Reed General Hospital, Walter Reed Army Medical Center, Washington, D.C. 20012

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ILLUSTRATIONS

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Chapter I
Introduction

The superior labial frenum, a fold of oral mucosa extending from the maxillary midline area into the vestibule and central upper lip, is a structure significant in several aspects of dental treatment. Historically, the frenum's importance in periodontic, orthodontic, and prosthetic considerations has generated considerable interest concerning its function and repositioning. Surgical treatment for a high frenum attachment which interferes with oral hygiene practices, contributes to persistant diastemata, or complicates design and function of a prosthesis has been described by Goldman, [1] Taylor, [3] Ceremello, [4] Kazanjian, [5] and many others. As recently noted by Ewen, [6] however, a disagreement continues to exist concerning the histologic features of the frenum. Thus it is felt that a histologic study of the frenum and its relation to the mucogingival junction might contribute to a better understanding of its microscopic anatomy and influence on dental deformity.

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Chapter II
Literature Review

Sicher [7] describes the superior labial frenum originating as a post eruptive remnant of the tecto-labial bands, which are embryonic structures which appear about three months in utero and connect the tubercle of the upper lip and the palantine papilla. Growth of the alveolar process normally causes the relocation of the attachment from the papilla area to the anterior aspect of the alveolar process. The timing and degree of this relocation is the basis for some of the pathologic changes attributed to the frenum. Sicher [7] further states that the frenum is normally restricted to the alveolar mucosa and does not enter the gingival area. Hirschfeld [8] correlated attachment at the gingival margin with an inability to perform adequate oral hygiene. Goldman [1] also felt that food impaction was likely when the attachment was at the gingival margin. Gottsegen [2] found inflammation and periodontal destruction occured as a result of high frenum location with accompanying oral hygiene problems.

Failure of the attachment to relocate results in a persistent band of fibrous tissue between the central incisors. This residual band has been implicated by Taylor [3] as a factor in retention of the midline diastema. His study found that 98% of the 6-7 year age group had the midline diastema but only 7% did not close spontaneously by age 12 to 18. The "abnormal" frenum linked to the retained diastema was described by Shirazy [9] as any frenum existing between the central

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incisors in the erupted permanent dentition. Dewell [10] stated that frena which increased in size with age were pathological and Curran [11] wrote that a frenum which caused blanching of the papilla when the lip was lifted should be termed abnormal. Bisnoff [12] and West [13] have reported that these diastemata and associated high frenum attachments may occur on a hereditary basis.

Anderson's Practical Orthodontics [14] noted a lack of agreement among orthodontists concerning surgical correction of the abnormal frenum, but West [13] and Hirschfeld [15] have expressed the current consensus that diastemata presisting after cuspid eruption should be treated by frenectomy in conjunction with orthodontics to approximate the central incisors. West [13] states that frenectomy alone may result in scar formation which would tend to prevent mesial movement of the centrals in the same manner that frenum did originally. Hirshfeld [15] and Corn [16] report that using only orthodontic treatment occasionally results in relapse separation of the teeth after removal of the orthodontic appliances. It would appear that this would also tend to locate the attachment at the gingival margin.

Dewel [10] and Gibbs [17] were less than enthusiastic about frenectomy because they found the intermingling of fibers of the frenum with the transeptal fibers. They believed excision of the frenum would also sever the transeptal fibers and tend to reduce the forces acting to bring the central incisors together. Baum, [18] however, indicated the transeptal fibers did not cross the midline but inserted into the raphe overlying the midline suture of the maxilla.

Similar disagreement exist concerning histologic morphology of the frenum in regard to the presence or absence of muscle.

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Gottsegen [2] and Bisnoff [12] reported that fibers of the orbicularis oris were sometimes found in the maxillary frenum. Archer [19] states that the depressor septi muscle "originates by fusion with the dense connective tissue between the centrals" and that "its fibers fan out widely to interweave with the red zone of the orbicularis oris." Knox and Young [20] have indicated that the frenum originated in the orbicularis oris and stated that their sections contained elastic and collagen fibers, muscle fibers, and fat. Corn [21] concurs that the frenum is completely understood and includes fat, loose connective tissue, and muscle fibers. In contradiction, Taylor, Shirazy, and Dewel's articles, [3,9,10] and current texts such as Orban's Histology [22] and Sicher's Oral Anatomy, [7] all state no muscle is present.

The disagreement in the literature regarding existence of muscle fibers in the frenum was the fundamental justification of this study. The histology of the maxillary labial frenum was investigated with regard to locality and quantity of elastic fibers and the characteristic dense collagenous tissue of the frenum as well, but the previous controversy centered on the muscle fibers.

It was anticipated that the information gleaned from the histological examination of both normal and "abnormal" frena would give a wider spectrum than the use of the only one of these types. Use of both would indicate if the presence of muscle fibers occurred only in the pathological frena, in all frena, or in none.

The differentiation, if found, would dictate dissimilar treatment for various classes of frena.

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The further definition of the histology of the maxillary labial frenum would then both add to the literature on this controversial subject as well as possibly indicate the most appropriate management of this structure.

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Chapter III

Materials and Methods

Eleven fresh biopsy specimens were obtained from orthodontic patients referred for frenectomy. (figures 1a and 1b) The technique employed was excisional and consisted of parallel incisions three to four millimeters apart extending from the palantine papellae to past the mucogingival junction. The incisions were to bone and the specimen thus included approximately a three millimeter width of periosteum. A suture passed through the frenum was utilized to manipulate it during surgery and to transfer it to the 10% buffered formalin in which it was preserved. Sutures were placed and periodontal dressings applied. At one week the sutures were removed, with uneventful healing occuring in all cases.

In addition, three autopsy specimens were obtained from sagitally sectioned human heads. The side containing the frenum was incised in the same manner as the biopsy specimens but, in addition, a portion of the adjacent alveolar bone was resected. These specimens were included to permit a comparison with less prominent frena and to enable a more complete histologic investigation by the inclusion of adjacent bone. The autopsy frena were maintained in 10% buffered formalin and decalcified prior to further preparation. All fourteen frena were divided longitudinally and the parts positioned side by side with the incised surface upward. After paraffin imbedding, sections we cut a eight microns until the blocks were exhausted. Alternate sections were stained with 1) Hematoxylin and Eosin 2) Masson's Trichrome and 3) Weigerts Elastic Stain. They were then examined for

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location and quantity of dense collagenous tissue, muscle fibers, and elastic fibers. To facilitate identification of these elements, control slides of human tissues using the same stains were prepared and used for comparison. A total of over five hundred slides were prepared and examined in this manner.

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Chapter IV

Findings

All specimens, both biopsy and autopsy, were found to contain loose connective tissue, densely packed collagen, and quantities of elastic fibers.

The only muscle fibers seen were in the autopsy specimens and were located in the vestibular portion of these more generously-excised specimens, and were not felt to constitute a part of the frenum proper. There was no evidence of muscle fibers coursing within the frenum or inserting at the alveolar bone.

The elastic fibers were seen in moderate quantities (fig. 2a and 2b) in both the normal and abnormal frena. The pattern of elastic fibers ended abruptly at levels varying with individual specimens and this was assumed to indicate locations of the coronally positioned mucogingival junction within the frenum. In most slides, the mucogingival groove could not be seen co-located with this transition from elastic to non-elastic containing tissues. It was also noted that the elastic fibers did not extend as far as the alveolar crest in even the most clinically formidable of the "abnormal" frena.

Thus, the elastic did not extend between the central incisors. In the autopsy specimens, only a few elastic fibers reached the alveolar periosteum, with most being seen at some distance from the osseous surface.

The predominent feature of all slides, of course, was the dense collagenous tissue (fig. 3). It formed a continuous, homogenous mass extending to the papilla area at the lingual termination of the specimens.

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Chapter V

Summary

In order to help clarify a disputed anatomical feature, the superior labial frenum was examined histologically in this study of normal and abnormal frena. The results indicated that no muscle was present and that the destructive capacity of the displaced frenum, therefore, could not be due to direct muscle tension.

All specimens, both biopsy and autopsy, were found to contain loose connective tissue, densely-packed collagen, and elastic fibers. The elastic fibers were seen in moderate quantities (fig 2a and 2b) in both the "normal and "abnormal" frena. This pattern of elastic fibers ended abruptly at a level varying with separate specimens. The level of elastic fibers may be related to the coronal position of the mucogingival junction in the frenum area. However, it was seen that the elastic fibers never extended all the way between the centrals even in the case of the heaviest fibrous band between the centrals.

Muscle fibers were absent in all areas of all sections except in the autopsy specimens. In these, a few muscle fibers were found in the vestibular portion, but not in the frenum proper. There was no evidence of muscle fibers traversing the frenum with insertion on the alveolar bone.

The lack of muscle fibers negates the concept of "muscle pull" exerted by the maxillary frenum. It would seem that the elastic nature of the frenum would be amenable to simple incisional treatment rather than the broad dissection excisional procedure used by many clinicians. Also, it is to be noted that separation of the frenum does not involve the musculature of the lip and should in no way decrease the function of that structure.

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Figure 1a. Frenum of a dense fibrous nature separating the maxillary centrals

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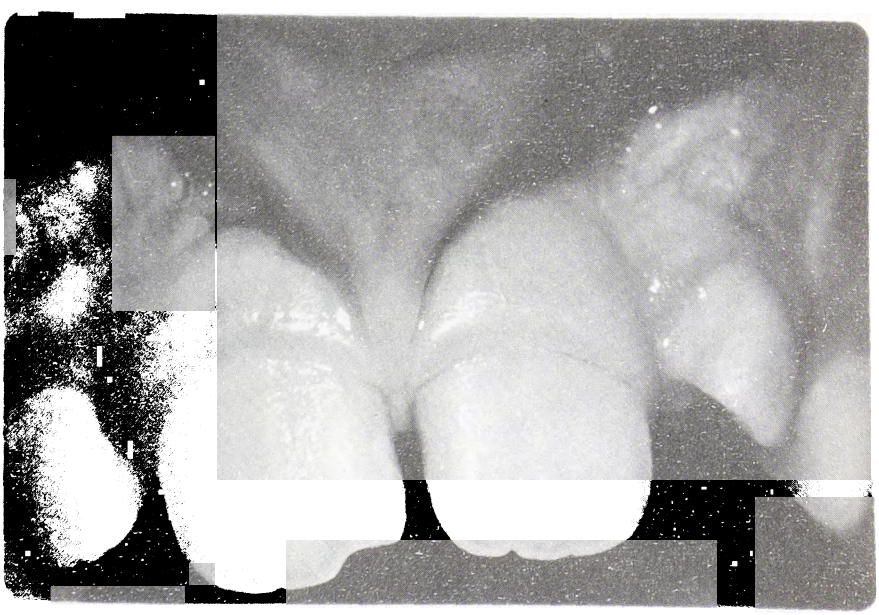

Figure 1b. Insertion of a frenum at the level of the gingival margin

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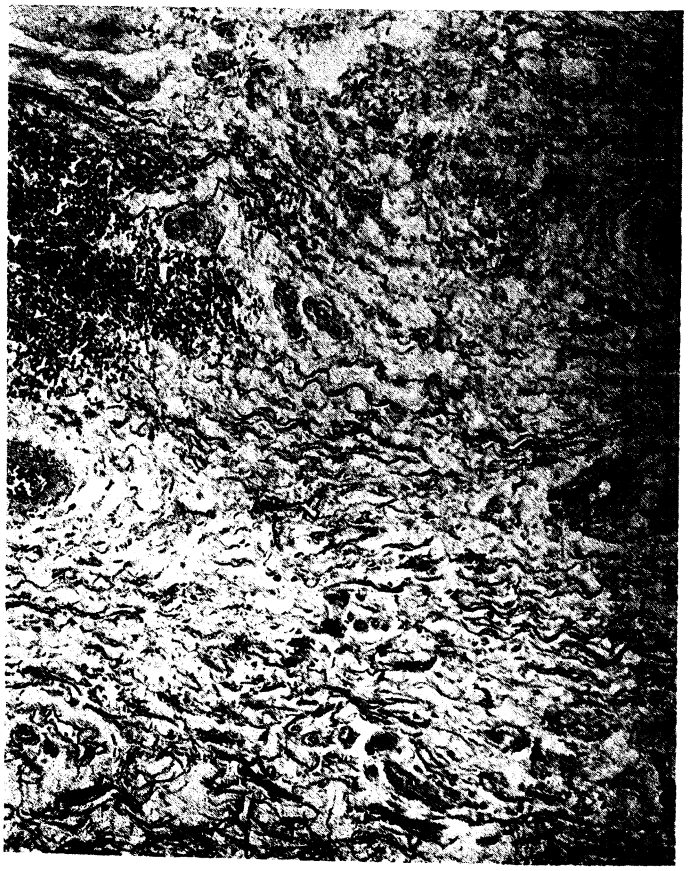

Figure 2a. Loose connective tissue portion of frenum exhibiting preponderance of elastic fibers (Weigerts Elastic Stain)

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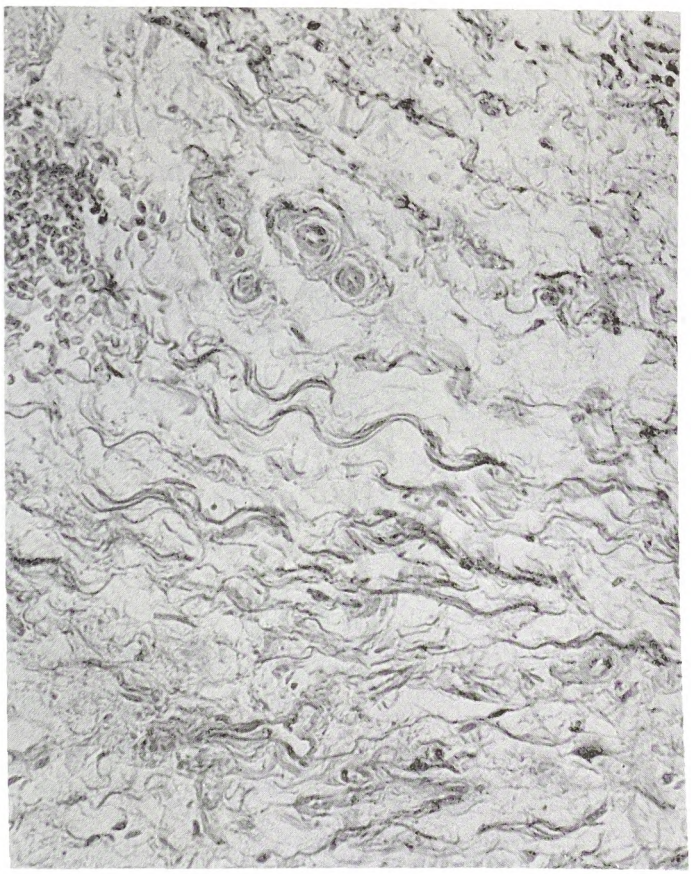

Figure 2b. Higher magnification of the thin, wavy elastic fibers (Weigert's Elastic Stain)

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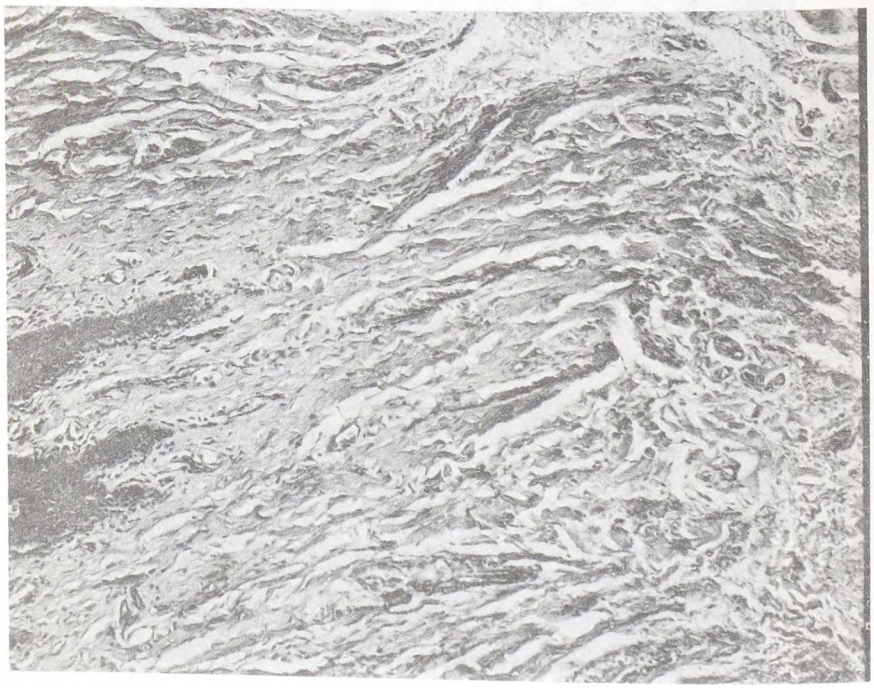

Figure 2c. Dense collagenous tissue seen throughout all specimens (Masson's Trichrome Stain)

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BIBLIGRAPHY

1. Goldman, H. M.: Periodontia, 3rd Ed., C.V. Mosby Co. St. Louis, 1953.
2. Gottsengen, R.: Frenum position and vestibular depth in relation to gingival health. Oral Surg., 7:1069,1954.
3. Taylor, J. E.: Clinical Observations Relating to the Normal and Abnormal Frenum Labii Superioris. Am J. Ortho and Oral Surg., 25:646,1939.
4. Ceremello, P. J.: The Superior Labial Frenum and the Midline Diastema and Their Relations to Growth and Development of the Oral Structures. Am. J. Orth., 39:120,1953.
5. Kazanjian, V. H.: Surgery as an Aid to More Efficient Service with Prosthetic Dentures, J.A.D.A. 22:566,1935.
6. Ewen, S. J.: Frena - Their Roles, Especially in Periodontics, N.Y. State Dental J. 34:626,1968.
7. Sicher, H.: Oral Anatomy, 5th Ed., C. V. Mosby Co., St. Louis,1970.
8. Hirschfeld, I.: The Toothbrush, Its use and Abuse. J.A.D.A., 26:1237,1937.
9. Shirazy, E.: Frenum Labii Superioris. J.A.D.A. and Dental Cosmos, 25:761,1938.
10. Dewel, B. F.: Normal and Abnormal Labial Frenum Differentiation. J.A.D.A., 33:318,1964.
11. Curran, M.: Superior Labial Frenotomy. J.A.D.A., 41:419,1950.

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12. Bisnoff, H. L.: The Labial Frenum. The Dental Outlook, 31:146,1944.

13. West, E. E.: Diastema, A Cause For Concern. D.C.N.A., July 1968.

Anderson, G. M.: Practical Orthodontics: 8 th Ed. C. V. Mosby Co., St. Louis, 1955.

15. Hirshfeld, L. and Geiger, A.: Minor Movement in General Practice, 2nd Ed., C. V. Mosby Co., St. Louis, 1966.

16. Goldman, H. M. and Cohen, D. W.: Periodontal Therapy. Mucogingival Surgery - H. Corn 4th Ed., C. V. Mosby Co.,1968.

17. Gibbs, S. L.: The Superior Labial Frenum and Its Orthodontic Considerations. N. Y. State Dental J., 34:550,1968.

18. Baum, A. T.: The Midline Diastema. J. Oral Med. 21:30,1966.

19. Archer, W. H.: Oral Surgery, 4th Ed., C. V. Mosby Co. St. Louis,1966.

20. Knox, L. R. and Young, H. C.: Histological Studies of the Labial Frenum. I.A.D.R. Abstracts, 1962.

21. Corn, H.: Technique for Repositioning the Frenum in Periodontal Problems: D.C.N.A. Mar, 1964 p79.

22. Orban, B.: Orban's Oral Histology and Embryology. 7th Ed., C. V. Mosby Co., St. Louis, 1972.

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TEMPOROMANDIBULAR JOINT PAIN-DYSFUNCTION SYNDROME

by

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Professional paper submitted in partial fullfillment of the requirements of the U.S. Army Oral Surgery Residency Program, Walter Reed General Hospital, Walter Reed Army Medical Center, Washington, D. C. 20012

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CHAPTER I

INTRODUCTION

The temporomandibular joint pain-dysfunction syndrome is a clinical enity which is often misunderstood, misdiagnosed and subsequently mistreated. It is a common complaint the dentist encounters almost daily. It is often the initial agent which places the patient at the disposal of the dentist. The evaluation of the complex nature of pain is the first step in arriving at an accurate diagnosis of cause. An understanding of the basic mechanisms, anatomy and physiology of this complex syndrome is reflected in the ease or difficulty of treating the patient. The dentist must become involved in challenging and difficult psychosomatic problems. Physiologic and psychologic makeup, individual constitution, life situations and experiences serve as a background for this syndrome. Effective management of mandibular dysfunction must be directed toward the whole patient and all his emotional and physical problems. To do less is to invite recurrence.

It is the purpose of this paper to present a limited review of the literature on temporomandibular joint pain-dysfunction syndrome, and to discuss the etiology, diagnosis and treatment.

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CHAPTER I

INTRODUCTION

The temporomandibular joint pain-dysfunction syndrome is a clinical enity which is often misunderstood, misdiagnosed and subsequently mistreated. It is a common complaint the dentist encounters almost daily. It is often the initial agent which places the patient at the disposal of the dentist. The evaluation of the complex nature of pain is the first step in arriving at an accurate diagnosis of cause. An understanding of the basic mechanisms, anatomy and physiology of this complex syndrome is reflected in the ease or difficulty of treating the patient. The dentist must become involved in challenging and difficult psychosomatic problems. Physiologic and psychologic makeup, individual constitution, life situations and experiences serve as a background for this syndrome. Effective management of mandibular dysfunction must be directed toward the whole patient and all his emotional and physical problems. To do less is to invite recurrence.

It is the purpose of this paper to present a limited review of the literature on temporomandibular joint pain-dysfunction syndrome, and to discuss the etiology, diagnosis and treatment.

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CHAPTER II

REVIEW OF THE LITERATURE

Review of the literature of temporomandibular joint pain-dysfunction, indicates three basic etiologic concepts: (1) the muscular concept,[1] (2) the occlusal concept,[2] and (3) the psychic concept.[3] The term temporomandibular joint pain-dysfunction syndrome was initially introduced by Dr. Lazslo Schwartz.[4] This term is the most descriptive of the condition since it includes occlusal disharmony, psychic tension and muscle hypertonicity as part of the syndrome. Some of the earliest work done in linking occlusal discrepancies with masticatory muscle incoordination was done by Moyers.[5] He used electromyography and reported that class II malocclusions had masticatory muscle changes not seen in class I cases. This work was amplified by the studies of Perry and Harris in 1954.[6] In 1956, Jarabac[7] showed greater and more irregular masticatory muscle contractions in patients who had occlusal interferences.

Based on these earlier studies, Ramfjord[8], in 1961 presented some classic findings which have become, in part, the basis of the occlusal-psychic-muscular concept of understanding the temporomandibular joint pain-dysfunction

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syndrome. Ramfjord demonstrated, using electromyography, that occlusal prematurities between centric relation (the most retruded mandibular position) and centric occlusion (maximum intercuspation) caused disharmony in masticatory muscle contractions. He further found that occlusal adjustment eliminated these prematurities and allowed the muscle activity to return to a normal range. A number of recent review articles give excellent coverage of the literature and the reader is directed to the reviews of Bell[9] and Stone[10] for a more comprehensive treatment of the literature.

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CHAPTER III

DISCUSSION

There are two aspects to the successful management of any disease process: one is the establishment of an accurate diagnosis; the other is an understanding of its etiology so that a rational treatment plan can be formulated. Lack of diagnostic discrimination can account for many of the failures in the management of patients with TMJ disorders.

The pain-dysfunction syndrome is different from either rheumatoid or degenerative joint disease in that the disorder is initially a problem of the masticatory muscles rather than an arthropathy.[11] The movements of the TMJ depend upon coordinated muscle action. When the teeth are brought into functional contact, they must work in precise harmony with the muscles. Thus, the muscles and the dentition become integral parts of joint function. Consequently, disruption of normal muscle action is expected to have immediate influence on masticatory function. When this occurs, the initiating symptoms are masticatory muscle pain (myalgia) and masticatory dysfunction due to spasm in the masticatory muscles. Precise symptoms depend upon which muscles are involved and to

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what degree. The discomfort is associated with the stretching and contracting of these painful muscles. The dysfunction is associated with restriction of movement due to shortening of the muscles, acute malocclusion due to displacement of the mandible by muscle spasms, and interference with movement due to the increased inter-articular pressure from spasm of the elevator muscles.

Because of a finding of some variation of agreement as to what constitutes the TMJ pain-dysfunction syndrome, a brief consideration of the essential signs and symptoms that places a patient in this category is both helpful and necessary to make an accurate diagnosis. The most common finding is pain of unilateral origin.[12] The pain usually is described by the patient as a deep dull ache felt in the preauricular area just anterior to the tragus of the ear. The discomfort may radiate from this region either superiorly into the temporal fossa, inferiorly down the ramus of the mandible, posterior inferiorly along the trapezius muscle.

Occasionally, the patient experiencing this ache will note a transient, unstimulated, sharp pain which will travel along the pathway of the auditory canal. The pain can be constant, but can be worse on arising, or mild in the morning and gradually worsening as the day progresses. It is frequently exacerbated at mealtime.

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The next most common finding is muscle tenderness.[13] The muscle most often involved in spasm is the external pterygoid muscle. By placing the index finger in the buccal space (the area just posterior to the molar bone) it is possible to palpate anatomic parts which can give important clues in diagnosis. A normal buccal space is free from pain on slight finger compression. If the muscles are in spasm, this area will be extremely painful on the affected side. By pressing the index finger upward and laterally against the coronoid process, a determination whether or not the insertion of the temporalis muscle is involved. A myositis of the insertion of one temporal muscle can cause a complete trismus; a slight myositis can affect mandibular function. By pressing the index finger upward and medially, it is possible to obtain information as to the condition of the lateral pterygoid muscle. If it is in spasm, there will be pain upon compression.

The third most common symptom is the clicking or popping noise in the TMJ. This symptom must be accompanied by pain or tenderness or both. A study by Stone and Dunn[10] demonstrated that 38% of the patients diagnosed as having temporomandibular joint pain-dysfunction syndrome have joint sounds. This may indicate that chronic joint sound, if it is not caused by the head of

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the condyle passing over the articular eminence, is a precursor to a more acute problem.

The fourth cardinal symptom is limitation of jaw function. Trismus or limitation of mandibular motion has been referred to by several authors[14,15] as being one of the most common clinical findings in this syndrome. Stone[10] found in his patients that the most significant and common clinical sign is deviation of the mandible laterally, during opening. Eighty per cent of the patients demonstrated such a midline shift and more than four-fifths of these patients exhibited a marked mandibular shift toward the involved or painful side. Besides having one or more of the four cardinal symptoms of pain, tenderness, clicking and limitation, the patient considered to have TMJ pain-dysfunction syndrome must also have these negative characteristics; absence of clinical, radiographic, or biochemical evidence of organic changes in the TMJ ; lack of tenderness in the TMJ when this area is palpated via the external auditory meatus.[1] The significance of these negative characteristics in establishing the final diagnosis lies in their indication that the primary site of the problem is in the masticatory musculature rather than in the structures of the joint. This distinction forms the essential basis for understanding the etiology of the pain-dysfunction

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syndrome.

The role of occlusion and occlusal interferences in the cause of functional TMJ pain-dysfunction syndrome is constantly debated. According to recent investigations, patients with this problem do not have any more occlusal interferences than individuals without the disturbances.[16] On the other hand, it has been shown that such disturbances can unquestionably be eliminated in the overwhelming majority of cases by removal of occlusal interferences. The disturbances can very easily be precipitated again by placement of a single occlusal interference in the same patient. A number of patients can also relate their symptoms to insertion of dental restorations or appliances, and muscle pain has been experimentally produced in denture patients by changes in occlusion.[17]

It is evident that one of the most important factors in the development of these disturbances is the individual patient's lack of adaptation to a less than ideal occlusion.[7] This adaptive capacity is very closely related to a patient's psychic status of stress, and emotional tension or tranquility and emotional stability. Onset, remissions and exacerbations of temporomandibular joint pain commonly follow or coincide with episodes of nervous tension (emotional conflicts, college exams, etc.).

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This threshold of psychic irritability, as it relates to occlusal interferences that trigger abnormal action of the jaw muscles, varies from individual to individual and from time to time in the same individual. Lupton[18] has shown that about seventy five per cent of an unselected sample of women patients from the Temporomandibular Joint Research center whom he subjected to psychological testing, fell within a dominant personality category. They were characterized best as "hypernormal" individuals. He has suggested that the attempt of these patients to maintain the facade of hypernormality, leads to generalized somatic tension. This tension accounts for their frequent addiction to tension-relieving oral habits.

There can be little doubt that a definite relationship exists between occlusal disharmony, psychic tension and TMJ pain-dysfunction syndrome. However, occlusal disharmony alone will not lead to pain in these structures unless the important factor of psychic tension also is present. In some extreme instances, psychic tension may be so severe that very little occlusal interference is necessary to start muscle spasms; or occlusal interferences may be so severe that it takes very little psychic tension to initiate excessive force and produce spasm. The majority of cases are found between these two extremes.[19]

Many modes of treatment have been used in correction of the temporomandibular joint pain-dysfunction syndrome.

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Bell[20] feels that occlusal disengagement is the treatment of choice. In the presence of myalgia or myospasm, the teeth should be disoccluded. Properly motivated, informed, cooperative patients can frequently accomplish occlusal disengagement by voluntarily avoiding clenching the teeth. When voluntary methods are ineffective, disengagement is accomplished with bite plates or occlusal splints. Muscle tonicity and pain will usually decrease within a few weeks after the use of these appliances. Bite plates or occlusal splints must be properly designed and adjusted. Improper appliances may worsen or perpetuate mandibular dysfunction. A modified Hawley bite plate appliance is simple to fabricate. A flat plateau of acrylic resin, built up behind the maxillary anterior teeth, serves as a stop for the lower incisor teeth. The occlusion is raised just enough to prevent contact of the posterior teeth and permit all jaw movements without tooth contact. Because it allows extrusion of posterior teeth, it should not be used for longer than three to six weeks.

After elimination of the pain, the patient is instructed to wear the bite plate only during those intervals when he is most likely to grind or clench his teeth. Consideration is also given at this time to indicated occlusal adjustment.

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Physiotherapy plays an important role in the treatment of myalgia and muscle spasm. Heat and cold are important adjuncts in therapy. Heat is probably the most practical modality for home and office use. Heat produces sedation, analgesia and relief of muscle tension. Effective use of superficial heat requires 30 minutes of exposure. This should be hot moist heat. Superficial heating will not, however, penetrate to a spastic medial pterygoid muscle. Treatment of these muscles requires the use of ultrasound diathermy. Ultrasound, the most effective method of deep heating available at present, heats to a depth of 4 to 5 centimeters in contrast to the 1 to 2 centimeter penetration of superficial heat. It can pinpoint a temperature rise in the deep cervical and pterygoid muscles and TMJ space to therapeutic levels within three to ten minutes.[21] It is contraindicated, however, in the orbital region and in the condylar area during the growth period. Ultra sound should be discontinued if the desired therapeutic effect has not been attained after ten days.

One of the cardinal principles of physiotherapy is voluntary restriction of mandibular movement. This is accomplished to stop the pain-cycling effect of continuous muscle use. The patient is instructed to limit the amount of mandibular opening. He is placed on a soft diet for a period of two weeks. If the patient

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cannot voluntarily restrict excessive mandibular movements, immobilization of the mandible is sometimes indicated. This can be accomplished with Ivy loops or arch bars, and intermaxillary wires or elastics. The immobilization period usually lasts no longer than one week. Most often, this is adequate time to break the myospasms. Attention can then be directed at occlusal disharmonies.

Drug therapy plays an important role in the treatment of TMJ Pain-dysfunction Syndrome. Psychic tension and anxiety are a dominant factor in the pathogenesis and perpetuation of pain-dysfunction problems.[22] Since 1954, when Thorazine was introduced as an effective antidepressant for psychotic patients, numerous drugs have been synthesized for treating emotional disorders and relaxing skeletal muscles. Many of these drugs are effective in treating symptoms of neurotic tension and anxiety. Valium is a safe drug for treating situationally related anxiety and tension of relatively short duration. I have found that 5 mg taken three times a day gives excellent results. An oral analgesic such as aspirin can be prescribed in combination with the diazepam.

Controlled pharmacologic and clinical studies of these drugs have failed to demonstrate that any one of them directly relieves peripheral myospasm when given orally.[23] Their effects on muscle tone or spasm are

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probably due to a nonspecific depressant action on the central nervous system. Diazepam may be clinically effective as a centrally acting muscle relaxant in conditions where spasticity interferes with performance.[24] Drug dependency is a potential hazard with any agent, so they should not be prescribed for long periods of time. Because drowsiness and incoordination may occur, patients should be warned against these possibilities.

Corticosteroid drugs are occasionally used for palliative treatment of the arthralgia associated with mandibular dysfunction. Their application is predicated on the presence of intra- or periarticular inflammation. When severe inflammation is present, the judicious short term use of an agent like prednisolone may be advisable in the smallest possible dose that will provide symptomatic relief of pain. For maximum effect, it should be given in combination with an oral analgesic such as aspirin.

Intra- and periarticular injections of a corticosterioid can be given a limited clinical trial in cases where systemic therapy is ineffective, where the affected joint is severely painful and inflamed, and where limited jaw-opening prevents occlusal disengagements. They must be used with caution since repeated injections may induce

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degenerative joint chances. When symptomatic relief is attained, it usually occurs within 48 hours and lasts from a few days to a month.

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CHAPTER IV

SUMMARY

It has been the purpose of this paper to discuss the temporomandibular joint pain-dysfunction syndrome. It has been described as a myofascial pain-dysfunction syndrome, which is characterized by deep, dull, localized pain in the pre-auricular area, the side of the face, the mandible and occasionally involving the neck region. The pain is usually unilateral and results in a limited motion of the mandible. The diagnosis is made on history of onset, symptoms, and a positive area of tenderness produced by palpation of the muscles of mastication. It is felt that the major single cause of pain is a sustained contraction or spasm of the muscles of mastication. Painful muscular spasm can result from such seemingly insignificant causes as minor malocclusion, a chill or exposure to cold, and emotional stress. A pain-spasm pain cycle is produced. The muscles contract, resulting in pain, which in turn produces an increase in contractions and resulting increase in pain.

The therapy employed for temporomandibular joint pain-dysfunction syndrome varies with the dentist's background and education. Every field of dentistry has become involved. The prosthodontist has relied heavily on bite-opening appliances, and full mouth reconstruction.

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The periodontist has endeavored to correct premature occlusal interferences, or negate the occlusion with bite planes. The oral surgeon has utilized roentgenographic interpretation, drug therapy, immobilization and injection of steroids. As I stated at the beginning of this paper, the effective management of mandibular dysfunction must be directed toward the whole patient and all of his emotional and physical problems. To do less is to invite recurrence.

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BIBLIOGRAPHY

1. Laskin, D. M.: Etiology of Pain-Dysfunction Syndrome, J.A.D.A., 79:147, 1969.

2. Ramfjord, S. P.: Dysfunctional Temporomandibular Joint and Muscle Pain, Journal of Prosthetic Dentistry, 11:353, 1961.

3. Moulton, R. E.: Emotional Factors in Non-organic Temporomandibular Joint Pain, Dental Clinics of North America, Nov., 1966.

4. Schwartz, L. L.: Disorders of the Temporomandibular Joint, Philadelphia, W.B. Saunders Co., 1959.

5. Moyers, R. E.: Temporomandibular Muscle Contraction Patterns in Angle Class II Malocclusions, American Journal of Orthodontics, 35:837, 1949.

6. Perry, H. T. and Harris, S. C.: The Role of the Neuromuscular Systemic Functional Activity of the Mandible, Journal of the American Dental Association, 48:665, 1954.

7. Jarabok, J. R.: An Electromyographic Analysis of Muscular and Temporomandibular Joint Disturbances Due to Imbalances in Occlusion, Angle Orthodontics, 26:170, 1956.

Digitized by Google

8. Ramfjord, S. R.: Bruxism, a Clinical and Electromyographic Study, Journal of the American Dental Association, 62:21, 1961.

9. Bell, W. H., and Ware, W. H.: Management of Temporomandibular Joint Pain-Dysfunction Syndrome, Dental Clinics of North America, April, 1971.

10. Stone, S. and Dunn, M.: The General Practitioner and the Temporomandibular Joint Pain-Dysfunction Syndrome, Journal of the Massachusetts Dental Society, Fall, 1971.

11. Bell, W. E.: Clinical Diagnosis of the Pain-Dysfunction Syndrome, Journal of the American Dental Association, 79:154-160, 1969.

12. Greene, C. S. and others: The TMJ Pain-Dysfunction Syndrome: Heterogeneity of the Patient Population, Journal of the American Dental Association, 79:1168-1172, 1969.

13. Ibid.

14. Dachi, S. F.: Diagnosis and Treatment of Temporomandibular Joint Syndrome, Journal of Prosthetic Dentistry, 20:53, 1968.

15. Zarb, G. A.: Assessment of Treatment of Patients with Temporomandibular Joint Dysfunction, Journal of Prosthetic Dentistry, 25:432, 1971.

Digitized by Google

16. Dixon, A. D.: Structure and Functional Significance of the Intraarticular Disc of the Human Temporomandibular Joint, Journal of Oral Surgery, 15:48, 1962.

17. Black, L. S.: Tensions and Intermaxillary Relations, Journal of Prosthetic Dentistry, 4:204, 1954.

18. Lupton, D. E.: A Preliminary Investigation of the Personality of Temporomandibular Joint Pain-Dysfunction Patients, Psychotherapy, Psychosomatic Medicine, 14:199, 1966.

19. Ramfjord, R., and Ash, M.: Occlusion, Philadelphia, W.B. Saunders, 1971.

20. Bell, William H.: Nonsurgical Management of the Pain-Dysfunction Syndrome, J.A.D.A., 79:161, 1969.

21. Krusen, F. H.: Handbook of Physical Medicine, Philadelphia, W.B. Saunders Co., p.279, 1965.

22. Laskin, D. M.: Etiology of Pain-Dysfunction Syndrome, J.A.D.A., 79:147, 1969.

23. Choice of Drugs for Emotional Disorders, Medical Letter, 6:45, June 5, 1964.

24. Farrell, D. F. and Hofman, W. W.: A Quantitative Evaluation of the Effect of Diazepam in Huntington's Chorea, Archives of Physical Medicine, 49:586, Oct, 1968.

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CURRENT CONCEPTS IN PREVENTION AND TREATMENT OF POST-IRRADIATION CARIES

by

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Professional paper submitted in partial fulfillment of the requirements of the U.S. Army Dental Residency Program, Walter Reed General Hospital, Walter Reed Army Medical Center, Washington, D. C. 20012

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Chapter I

INTRODUCTION

"Radiation caries" is a broad term used to describe the rapidly destructive carious process involving the teeth of patients who have had radiation treatment to the head and neck. A more accurate term for this disease may be post-irradiation caries. Typically, the demineralizing process attacks the radicular portion of the tooth and other areas where the protective enamel layer has worn thin or is non-existent. Radiographically, the lesions typically appear as punched-out areas, usually below the cemento-enamel junction.

The true cause of this rapidly progressive process is still an area of controversy among those who see and treat these patients. It is speculated that it is due either to the direct effect of the radiation on the tooth itself or to an indirect effect of the radiation which causes secondary changes in the oral cavity thereby affecting the teeth, or a combination of both causes.

It has been difficult to evaluate and summarize the effects of radiation on the teeth of man reported in the clinical literature. Most of the information has been obtained primarily from case histories of patients treated by

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irradiation for various types of tumors. The multiplicity of exposure techniques and therapeutics employed, the sporadic nature of dental observations, and the paucity of information regarding the general oral condition prior to irradiation limit the value of most case reports.[1]

It is a fact that, in the presence of oral cancer, teeth have a special significance regarding morbidity and rehabilitation following therapy. As the cure rate for oral cancer improves, our attention turns from the immediate concern for irradiation of the disease to some considerations of the long term problems of dental and oral health in these patients.

The object of this paper will be to describe current methods advocated in the prevention of post-irradiation caries and the materials and methods used in treating such lesions that do occur.

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Chapter II

REVIEW OF THE LITERATURE

Del Regato[2] was among the first to describe the rapidly progressive dental lesions associated with the irradiated patient. He noted that these lesions were most frequently seen as a decay of the neck of the tooth which extended around the tooth and finished by complete amputation of the crown. He was also the first to report that "these lesions seemed to be produced by an indirect mechanism because very often they are observed in patients whose teeth or maxillae have not been irradiated." It was suggested that irradiation of the salivary glands and modifications in the secretion of saliva resulting therefrom, either qualitative or quantitative, should be considered as one of the factors in the production of dental lesions. He therefore suggested that a pre-irradiation extraction of greater numbers of teeth would be a prudent procedure.

A EXTRACTIONS

In order to be certain of preventing the dreaded dental defects seen in so many head and neck irradiated patients, which may ultimately lead to osteoradionecrosis

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of the jaws, many dental practitioners and radiation therapists have chosen pre-irradiation extraction as their first line of defense.

Opinions on which teeth to extract prior to irradiation range from the extraction of all teeth[3-9] to the extraction of none.[10] Many believe that only those teeth in the direct line of irradiation need to be extracted[3,4,6] along with other teeth that are badly cariously or periodontally involved.[5,7,8,9] Others recommend that only those teeth which are extensively involved cariously or periodontally, those which are partially errupted or impacted, and those which are not necessary for reconstruction and are not self-cleansing should be extracted.[11] Some suggest extracting all teeth which might need extraction within one year of irradiation[12] or all teeth which might be deeply cariously involved and have a possibility of exposure.[13] Many treatment centers prefer to classify patients according to their presenting dental condition at the time of initial examination and then extract teeth accordingly.[4,6] Still others would recommend removing all teeth with crowns on them "because crowns prevent clinical or radiographic visualization of caries."[7] There are reports in the literature of high incidences of osteoradionecrosis following irradiation occurring in areas of pre-irradiation

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extraction.[4] This is probably due to the permanent delay in remodeling of the mandibular alveolar ridge and continuous threat to the integrity of the overlying mucosa.[10] Because of this, some treatment centers have recommended a more conservative approach to extractions immediately prior to irradiation.[4] Nevertheless, in certain circumstances, such as when the salivary glands are to be irradiated, or when the patient's ability to cooperate is poor, or when pre and post-irradiation dental treatment may not be adequately available, it is suggested by some that all the teeth should be extracted pre-irradiation with sufficient time allowed for healing.[5-9] It has been said that "based on past experience, it may be radical to keep the teeth and conservative to extract them."[7] In a survey in which 127 radiotherapists and 93 prosthodontists responded, 25 percent of both groups believed that all teeth should be removed prior to head and neck irradiation. Forty-six percent of the prosthodontists believe that only the teeth in the direct field of radiation should be removed, whereas 65 percent of the radiotherapists indicated that all teeth with decay or associated inflammation in the radiation field should be extracted. Twenty-four percent of the prosthodontists and 20 percent of the radiotherapists believed that if salivary glands are irradiated, any teeth

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which might be effected by xerostomia should be extracted. Greater than 80 percent of both groups agreed that a decrease in saliva production due to irradiation of salivary glands is instrumental in the production of post-irradiation problems of teeth.[8,9]

B PLAQUE CONTROL

When it is elected that teeth shall be preserved in irradiated patients, those who are responsible for the treatment of these patients seek the best methods available for preserving the dental organs in the face of an altered hostile environment.

Of course, all teeth which are to be kept are restored to as near optimum condition as possible, preferably prior to irradiation, by using meticulous care and the best available materials and methods.

It has been suggested by some that full coverage acrylic splints should be made and fitted in order to insulate the natural teeth from the unnatural, dry environment of the irradiated oral cavity. These splints are left in place, covering all remaining teeth, until the salivary flow has returned to near normal. This may be a total of two years or more.[14]

Most all agree that at the top of the list in prevention should be good home care with emphasis on

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plaque control. The statement which in general reflects the concensus of opinion is, "the amount of plaque formation or debris present is directly proportional to the advancement of the decay process."[15] Therefore, it is most important that the patient is made aware of the problem of caries and is assisted in maintaining his oral environment in as optimal condition as possible.[3,16] Patients who have previously demonstrated poor plaque control must be taught proper techniqes and placed on an intensified program of plaque control. They must be thoroughly dentally educated and impressed continuously with the importance of diligent home care.[3,6] A patient's original susceptibility to caries and a patient's status of oral hygiene are two important factors which dictate the severity, type and extent of caries in the irradiated patient.[11] It has been shown, without a doubt, that the increase of microorganisms that accompanies poor oral hygiene is a definite contributing factor to dental decay.[7,14] Neglect of the oral tissue and teeth following irradiation may lead to infection possibly resulting in osteoradionecrosis of the jaws. This may ultimately lead to the death of the patient even though the cancer has been eliminated. Therefore, it is suggested by some that, if not convinced that the patient will maintain satisfactory plaque control

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and oral care for the rest of his life, all teeth should be extracted prophylacticly.[5,7,18,19,20] One report stated that it was possible that overvigorous toothbrushing in an attempt to remove the material accumulated around the necks of teeth caused the cervical erosion typically seen in irradiated patients.[21] However, most therapists who treat these patients agree that meticulous plaque control at home is our greatest ally in prevention of these dental lesions.[3,6,11,15,18,22,23]

It is a recognized fact that one of the greatest deterrents to the patient in maintaining satisfactory plaque control is the presence of locally painful tissues resulting from mucositis and xerostomia. These secondary sequella of irradiation often prevent the vigorous cleansing regime that such conditions warrant. Many recommend the use of an oral lavage containing an alkaline solution in warm water for relief of mucositis and xerostomia.[3,11,22] This may be a useful adjunct as well as glycerin rinses and troches.[3] One report in the literature indicates that mouthwashes are sometimes the most that can be tolerated. In such cases, a dilute solution of chlorhexidine is recommended, "which if used regularly (b.i.d.), maintains cleanliness far better than anything else (other mouthwashes) and appears to inhibit calculus deposition."[14]

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Life threatening medical complications coupled with the side effects of irradiation: dryness, loss of taste, hypersensitivity of intact teeth and the onset of dental decay worry both the patient and practitioner. These problems along with an ingrained fear of dental procedures may oftentimes understandably cause a patient to forgo dental care at a time when it is needed most.[14,24]

C FLUORIDE

Enamel is composed of calcium phosphate crystals embedded and enveloped by a membranous-like matrix. In the normal unaltered oral environment, these crystals are in physio-chemical equilibration with the fluids that bathe them, so a tidal wave is constantly taking place, whereby the enamel crystalites are dissolved and then recrystallized. However, in the irradiated patient, the saliva is frequently altered in quantity, quality, and pH.[2,22] Saliva has been found to exert a major influence in protecting the tooth structure.[25] For instance, when fluoride is present in saliva or in plaque on the enamel surface, the remineralization process is enhanced by the deposition of calcium phosphate salts which are apparently apatitic. Remineralization is largely responsible for the maintenance of enamel. When enamel is demineralized through bacterial action, it will normally remineralize

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again if the bacterial attack on the enamel surface is not constant and overwhelming, thereby resulting in decay. The quality and ability to resist dissolution of the remineralized enamel will depend upon the availability of trace elements such as fluoride at the time in order to influence the acquisition of stable apatite crystals. "The most acceptable explanation of fluoride efficacy is that it reacts through the dissolution of the calcium phosphate crystal and precipitates with the fluoride as a fluorapatite."[26]

The rationale behind the use of fluoride then is to make tooth surfaces less susceptable to demineralization thereby aiding in the prevention of the decay process. However, if the decay process is advanced, fluoride will only slightly halt the progression.[14] It has been shown, as in plaque control, that with an increased cooperation in the use of fluoride, a lower incidence of decay occurs in the irradiated patient.[15,23]

Several studies indicate that the application of 1% sodium fluoride for five minutes a day via a fluoride carrier covering all the teeth will significantly reduce the amount of cariously effected teeth in irradiated patients.[11,13,15,22,27,29] The teeth should be thoroughly cleaned before application in order to obtain maximum benefit. It is advocated that the fluoride

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applications should be started before or at least by the beginning of irradiation.[11,13,15] Some suggest that, during radiation therapy, a trained therapist should apply topical fluoride daily to the patient's teeth.[6] The daily use of the 1% sodium fluoride has been known to be effective in control of rampant caries reducing the hypersensitivity of root surfaces and in control of radicular caries, all of which are common occurrences in irradiated patients. The use of the fluoride carriers has many advantages. They maintain a high concentration of fluoride in contact with the teeth for a specific period of time. Saliva will not dilute the fluoride solution. Biting pressure on the trays forces the preparation into inaccessible areas around the teeth. The soft tray material is comfortable and largely restricts the material from the throat area.[5]

It has been suggested that the sodium fluoride gel could be thickened and used for a toothpaste with hydrogen peroxide on the brush.[29] Other types of fluoride preparations in a variety of forms have been investigated as to their usefulness in the reduction of acid solubility of tooth structure. The development of post-irradiation dental caries was effectively prevented in a study using 0.4% SnF_2 gel daily in conjunction with an oral hygiene program.[18,30] In another study, SnF_2 was shown to be

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superior to sodium fluoride in reducing acid solubility of intact enamel when applied twice daily.[31] Acidulated phosphate fluoride gel demonstrated no superiority in anticarious effect when compared with sodium fluoride on a similar basis.[27] Studies with amine fluoride have shown that the optimal length of time of application is ten minutes. Increasing the length of time that the fluoride is in contact with the tooth beyond this time limit does not increase the fluoride uptake. In this same study, retention of fluoride was not improved by a surface coating of the teeth or by treatment with a fluoride-varnish preparation.[32] However, because it is well known that a portion of the fluoride acquired during topical application leaches away during the first few hours (especially if the penetration rate is rapid), methods have been studied to seal the fluoride next to the teeth in order to obtain greater benefits from the fluoride ions. A similar procedure may be feasible in sealing cavity margins to prevent recurrent caries in amalgam restorations in which the cavity preparation walls were treated with fluoride solutions for fifteen to thirty seconds prior to restoration.[16,35,36] This does not endanger the pulp. The actual condition of the tooth surface can have a bearing on the degree of benefit imparted to it from the fluoride.

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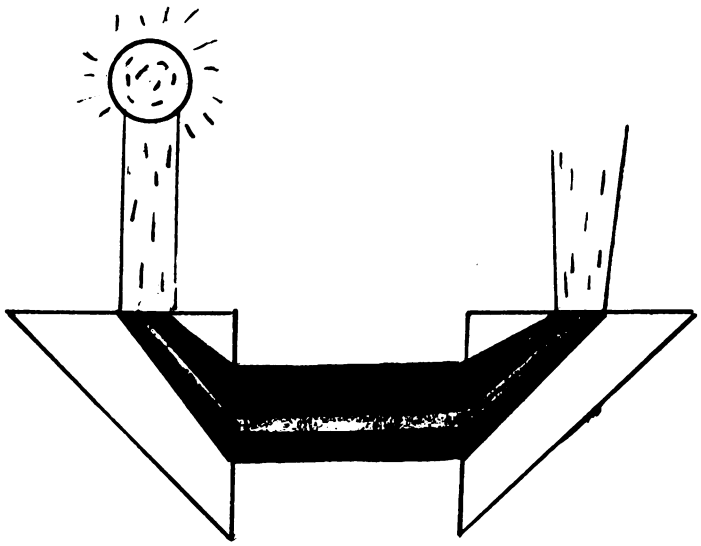

Figure 3: Light Dispersion in a Prism

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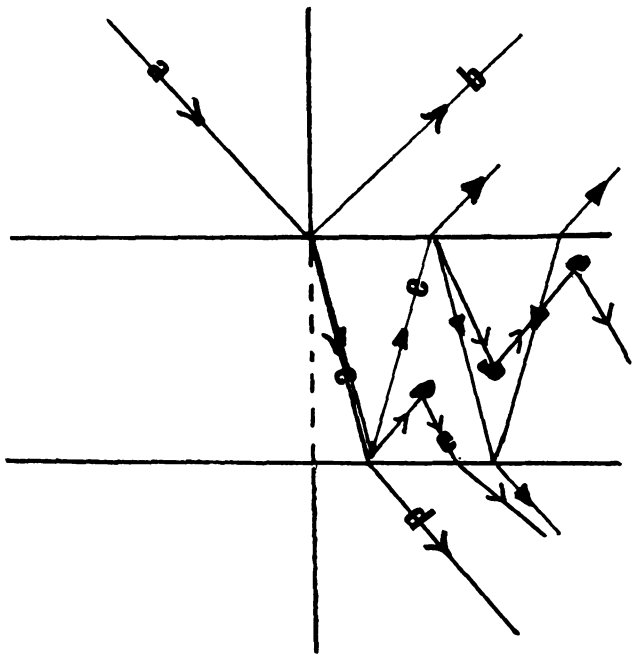

Figure 4: Light Reflectance

a. Incident Beam

b. Reflected Beam

c. Refracted Beam

d. Transmitted Beam

e. Dispersed Light

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becomes important to the clinician when trying to interpret the form of the tooth, A major portion of its final appearance depends on the size, shape, surface texture and particle content of the restorations.[5,12,17]

A method must be provided which will allow the clinician to duplicate the color he selects, since the previously mentioned effects influence both the color and character of the restoration. By the use of the spectrophotometer and colorometers industry has come up with a number of color systems, which provide predictable duplication of colors. The more commonly described systems are those of Ridway, Ostwald, and Munsell.[3,10,11] The Munsell system, which is the best known, is based:

> "on the guiding principle of equal visual perception of small color differences, the Munsell system is both a collection of samples painted to represent equal intervals of visual perception and a system for describing all possible colors in terms of three coordinates".[11]

The Munsell color coordinates are hue, value and chroma.[3,10-12] The literature has described these parameters by other names, depending on who is describing color. The physicist call them dominant wavelengths, reflectance, and purity, while the psychologist and some dental texts name them hue, brightness, and saturation.[12,18,19] The coordinates or parameters can be defined as follows:[3,5,10-12]

Hue: The quality of color which allows us to distinguish one color from another. Red from green, yellow from violet.

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Value: The quality which distinguished a light color from a dark color, and is related to the achromatic (colorless) polar axis in the Munsell system. This is represented on a black and white scale with gray as an intermediate. The black has a low value (zero) and the white has a high value (ten).

Chroma: This is the property which distinguishes the difference between a strong color and weak color. It deals with the intensity of the hue in a color.

When trying to understand the Munsell color system, it is best to apply it to only one color. First, turn to Figure 5 and study those boxes assigned letters. It will be seen that the color blue, A, is the original hue at the fourth value level (column 1). Any color to the left of the original hue has, by the addition of various quantities of gray of the fourth value, reduced the chroma, B, although the hue has not been affected. If a gray of higher value was added to the original hue the insuing color would have a lesser chroma, higher value, but the same hue, C. If a lower value (darker) gray is added, the chroma would be lessened, D, the value would be decreased, although the hue would remain the same.[10,14,15]

What must be emphasized, is that by adding more and more gray of the same value, to any color, only reduces the chroma of the hue. It is the value of the gray added which will give a lower or higher value to the hue.[11]

One can apply the knowledge of the Munsell system and that advocated by Ceramco[18] to porcelain restorations. The

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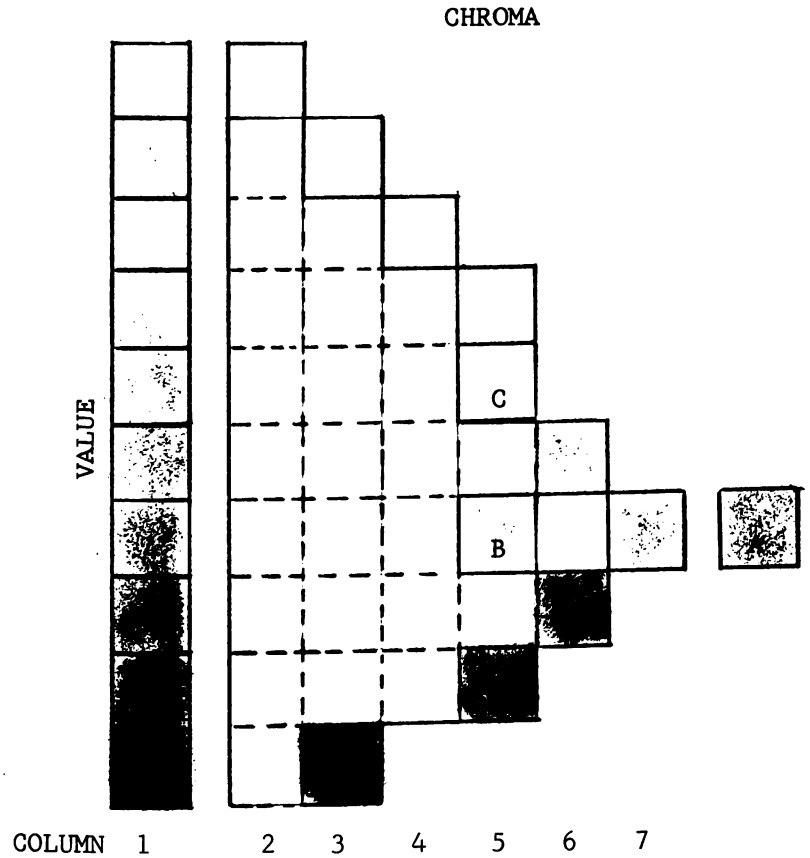

Figure 5: Munsell Color System

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Ceramco system is an adaption of the Munsell system, although only six colors are used. It is based on the primary colors: red, yellow, and blue and their complimentary colors: orange, green, and violet (Figure 6). This system arranges these colors in a circle, starting at the top with red, followed by orange, yellow, green, blue, and violet. The center of which is gray. This system provides a method, by which, we are able to obtain the proper shade to communicate with our colleagues in regard to the hue, chroma, and value of our restorations. If a restoration has too much orange present, it can be corrected by the proper use of the Ceramco color system.[18] The color chart tells us that orange sits between red and yellow and contains a mixture of each. By the addition of red's complimentary color green, the two colors cancel each other out to form gray. The yellow then becomes the dominant color present. If the chroma of the yellow is found to be too intense, it can be reduced by the addition of a gray with a similar value. By the addition of various amounts of gray of different values. The value of the hue can be changed.[10-12]

Prior to using any color system for the staining of porcelain restorations, one must have a knowledge of both dental and oral anatomy. As light enters the mouth it is lost in the darkness of the oral cavity. The translucent incisal edges may take on a grayish appearance due to the

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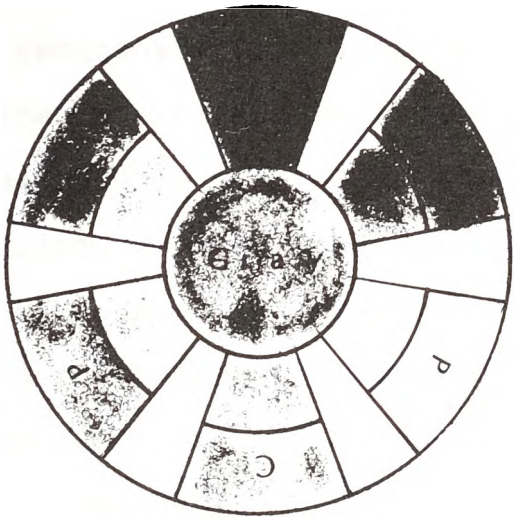

Figure 6: Ceramco Color Guide

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black background. The gingival one third of the teeth may have a reddish tint, due to the light reflection from the gingivae. The anatomy of the teeth will help determine their shade, since a tooth with thick enamel will reflect less of the underlying yellow dentin than a thin enameled tooth. In the end, one must realize that dentin, enamel and fused porcelain all have different optical properities. We are, therefore, attempting to blend our artifical prosthesis into an area surrounded by natural teeth.[12]

Man has not discovered how the cone pigments of the eye function and how color is transmitted to the brain. It is known that the human eye sees color as variable combinations of the three so-called primary colors. What dentists have failed to realize is that the discription and interpretation of color is based on the Munsell system, which will allow the clinician to communicate ideas on color to each other. The concept that man can choose and combine colors in two fundamentally different ways has been lost in the rhetoric. The methods are by addition or by subtraction of colors.[13,16] The former can be demonstrated by projecting the three primary light colors, red, green, and blue-violet on a white background. The combination of red and green being yellow, while all three will give us white. In the subtraction method, the primary colors are red, yellow and blue, thus blue and yellow gives green.

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This process is brought about by the taking of colors away from white light and permitting the remainder to come through.[16]

It should be noted that in each of the methods, the primary colors of one are the complimentary colors of the other and vice versa. In the additive method we start with three light (colored) sources on a white background, while in the subtraction method we start with a white light source and by the use of primary colors we mix colors to produce color. In all dental offices a combination of both methods are occuring at the same time. The appearance of the natural teeth may vary according to the light in which they are observed. It is impossible to imitate all the optical systems with artificial restorations. The dentist can approach the characteristics of natural teeth sufficiently, so that the difference is only noticable to the trained eye.[20]

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CHAPTER IV

Dental Porcelains

Dental porcelains come in three types: one used in artificial teeth, one employed in jacket crown fabrication and one used in the fabrication of the porcelain fused to metal restorations. The fabrication of the dental porcelain is the same in all three cases. It is the contents of each group of porcelains which give them their inherent properties. The medium and low temperature maturing porcelains are those porcelains used in jacket crowns and porcelain fused to metal restorations. Their basic properties of temperature maturing and esthetic characteristics are of interest to us. Dental porcelains are made by firing the raw ingredients until they are fused and then they are quenched. The quenched dental porcelains are then ground into a fine powder, the frit.[3,20]

This is the foundation from which all porcelains are made. At this point several compounds are added or omitted to produce specific types of porcelains. All dental porcelains are translucent, except opaque which has had Zurconium added.[20]

Dental porcelain restorations are made by firing the frit into a cohesive mass. Small increments of porcelain can be added to the fused material, until the desired shape

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is obtained. The porcelain is still porous at this time and dull in appearance. By glazing the surface of the restoration the glossy appearance of the natural dentition can be achieved. The surface of the dental porcelain frit is completely melted together to form a single phase glass. It can also be achieved by the addition of a glazing powder whose maturing temperature is lower than the body porcelains.[3,12,20]

The final procedure in the fabrication of porcelain fused to metal restorations is the staining of the outer surface. The stains are obtained by the adding of pigmented oxides to the frit. The blending of colors in enamels is difficult, because in addition to the color interaction, chemical reactions between the oxides can occur.[3]

The frit used in stains must be milled to a much greater fineness than that of body porcelains. This is necessary in order to have an even distribution of frit and pigmented oxides in the stains.[3,12,20]

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CHAPTER V

A Technique of Staining Enameled Restorations

Materials: The materials required to stain porcelain fused to metal restorations are minimal. The three main items necessary are the staining kit, furnace, and good light. The kit should contain the following items:

Porcelain Stains: It is very important that the stains be compatible with the body porcelain.[18] At present, the Ceramco stain kit is the most popular, because of its great variety of tints. The Wilkinson ceramic glazing kit is an excellent kit, especially for gingival characterization.[18,20]

Liquid Media : This can be distilled water, glycerine, or any media present in the kits.

Staining Slabs : The stains can be retained on the artist's palate from one staining to another. A useful addition to ones armamentarium is an artist's palate with wells. It will help prevent the spreading of the stains into each other.

Spatula : Metal spatula #324.

Paint Brushes : Several sable or camel hair brushes are nice. They should be thin and pointed. It is very difficult to sharpen any brush, therefore, pointed brushes are important. Although one of the brushes should be striped of almost all bristles. The one or two bristles left are useful for very fine staining, for example craze lines.

Furance : It should be a compact furnace which will fit easily into the operatory. It should have the capability to rise slowly or rapidly to its maximum temperature, 2000°F, in 10-20 minutes, and hold at that temperature for heat soaking.[12,20]

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Sagger Pegs and Tray: The tray should have fixed and movable pegs for versatility.

Light Source : If at all possible several sources of light should be available in a staining procedure.[22] It is recommended that a full specturm type bulb be utilized in dental operatories and laboratories. The Verd-A-Ray Criticolor and G. E. Chroma 75 lights appear to meet most of the criteria needed.[21,22]

Staining Technique

Step 1: The shade of the tooth is selected prior to any tooth preparation. This will be the base line from which future staining procedures are predicated. It is important, at this time, to select a shade which is similar to the original tooth or slightly lighter, because dark colors are more difficult to lighten later. Stains have a tendency to slightly decrease the color value of the restoration.[17,20] The Dentsply International Trubyte Bioform Shade Guide is an excellent guide from which to obtain the basic shade.

Step 2: The enameled restoration should be returned from the laboratory before final glazing. This allows the restoration to be tried in the mouth and recontoured. The margins and occlusion are checked and adjusted, as required. The restoration is then moistened with the patient's saliva or water.

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This allows the clinician to evaluate the value, chroma and hue of the enameled restoration. When the shade is going to be evaluated the clinician should first look at an area with a blue background.[22] Because when one fixates on a color intently for 30 seconds to two minutes and then looks away, the after-image remaining in the mind is that of the complimentary color.[25] The complimentary color of blue is orange, which is in the shade range of most teeth. Whether or not the restoration is to be stained it is now cleaned in the ultrasonic cleaner in distilled water. If no staining is necessary it goes on the glazing phase.

Step 3: The cleaned restoration is dried and placed loosely into the mouth. The area is isolated with cotton rolls or by 2x2 gauze. It is important that the cleaned porcelain surface is not touched. Sodium chloride on the hand may react with some of the pigmented oxides to cause a chemical reaction.

Step 4: The staining technique itself is not difficult and can be accomplished quickly if a few rules are followed. It is best to first cover the surface of the restoration with a thin layer of the liquid media. This gives the restoration the illusion

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of being glazed, and the clinician can better evaluate the amount of staining required.

A tooth which appears ovoid can be tapered by the addition of a little blue stain along the proximal line angles. A tapered tooth can be made to look ovoid by the addition of brown along its proximal line angles.[19]

Yellow, brown or orange-brown can be added along the gingival one third to match dentin showing through thin enamel. The incisal edge of the mandibular teeth can be grooved, a brown stain can be placed in the groove surrounded by a yellow, orange-brown mixture. This helps match worn incisal edges, whose dentin has been stained by natural causes or discolored from the use of tobacco or coffee. A light brown can be placed in occlusal anatomical grooves when attempting to simulate the staining of older teeth.[12,17-19]

Brown can also be used to show stained check lines or cracks. If the crack appears deep, the restoration should be etched with an inverted cone bur or razor blade and the area stained.[12,17]

An unstained craze line can also be shown by spreading a light blue stain in one direction. This should end abruptly in a straight line. The

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border is then accentuated by placing a little white along its length.

Blue or Blue-Gray is used to increase the translucency along the incisal edges. This can be applied either buccally, lingually or both. If the incisal edge appears too blue a little complimentary color, orange, can be added. The translucency can be accentuated by bordering the blue with a little orange. If the restored tooth is next to a tooth with a large M.O.D. amalgam, its enamel may have a bluish-gray appearance. This can be simulated by staining the buccal surface with blue-gray stains with white flecks. This will give the restoration the illusion of having an alloy and will give the dental arch harmony.[18]

Hypoplastic enamel can be shown by the addition of white with dashes of gray or blue. Mottling can be shown as brown areas circumscribed by a rim of light orange or white.

Bridges can be stained interproximally by the addition of blue, violet, brown or gray stains. In older patients brown stain helps, where as in the younger patient blue-gray may look the best. In women the use of violet or light gray is appropriate.[18]

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All stains added after the biscuit bake can be removed by wiping them away prior to glazing or ground off after glazing. The inherent shades of the restoration require shade modifications. This is accomplished by either adding stains to decrease its value or to increase its value, the latter is the most difficult of the two. Sometimes this can be done by the use of the tint's complimentary color.[15,18]

Step 5: Once the staining has been completed, the restoration is carefully placed on a sagger peg and tray. It is best to remove the restoration from the tooth by placing a finger on the lingual surface of the restoration and slowly slipping it off. The sagger tray and restoration is placed in front of the furnace muffle and the stains are allowed to dry. Once the stains are set, the restoration is fired.

The dental porcelain can be glazed in two ways. The simplest and by far the hardest glaze obtained is by self glazing. The dental porcelain is heated rapidly (10-15 minutes) to its fusion temperature. The glazing temperature of dental porcelain is 1800^{o}F and it is held there for 5 minutes. The porcelain becomes pyroplastic and forms a vitreous layer which is a single phase glass. If a glazing powder

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is used, the restoration is taken up to 1100-1200°F and held at this temperature for several minutes for the removal of impurities. The temperature is then raised to its glazing temperature around 1640°F, at the rate of 100°F per minute.[12,20]

The glazed and stained restoration is removed from the oven and cooled. When the restoration is cool, the gold is polished and the restoration is ready for cementation. If at any time further changes are desired, the restoration can be restained and refired.

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CHAPTER VI

Summary

This paper has briefly explained some of the history of porcelains, color and its systems, porcelain manufacture, and the materials needed for staining. A practical technique has been described to help the clinician satisfy the patient's need for esthetics. The clinicians ability to duplicate nature has yet to be achieved. This will not occur until we have a greater knowledge of light sources, available stains and better restorative materials. We must continue our search for materials which are biologically acceptable, optically similar, and technically workable if we are finally going to achieve our ultimate goal of duplicating nature.

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BIBLIOGRAPHY

1. Savage, G.: Pottery and Porcelain. Encyclopedia Britanica, 1966, Vol. 18, pg.342.

2. The Editors: Enamelwork. Encyclopeida Britanica, Macropedia, 1974, Vol. 6, pg.778-779.

3. Andrews, A. I.: Porcelain Enamels, Ed., The Gerrard Press Publishers, Champaign, Illinois, pg.1-5; 78-83; 97-106, 365-369.

4. Brown, L. P.: The Antiquities of Dental Prosthesis. Dental Cosmos, 1934, Vol. 76, pg.830.

5. Tylman, D.: Theory and Practice of Crown and Fixed Partial Prosthodontics (Bridge), 6th Ed., The C. V. Mosby Co., St. Louis, Illinois, 1970, Chapter 1 and 25.

6. Morison, K. N. and Warmick, M.: Staining of Porcelain Bonded to Metal Restorations. Journal of Prosthodontic Dentistry, Vol. 15, July and Aug. 1965, pg. 713-716.

7. Silver, M., Howard, M. C., and Klein, G.: Porcelain Bonded to Cast Metal Understructures. Journal of Prosthodontic Dentistry, Vol. 11, 1961, pg.132.

8. Clark, E. B.: Seventy-fourth Annual Session of the American Dental Association, Buffalo, N. Y., Sept. 15, 1932.

Digitized by Google

9. Sproul, R. C.: "Color: Frustrations or Pleasure". Paper presented before the 108th Annual Session of the American Dental Association, Washington, D.C., 1967.

10. Sproul, R. C.: Color Matching in Dentistry, Part I, The Three-Dimensional Nature of Color. Journal of Prosthodontic Dentistry, Vol. 29, No. 5, May 1973, pg. 416-424.

11. Billmeyer, I. W., and Saltzman, M.: Principles of Color Technology, New York, J. Wiley and Sons, Inc., pg. 1-89.

12. Johnston, J. F., Phillips, R. W., Dykema, W. R.: Modern Practice in Crown and Bridge Prosthodontics, 3rd Ed., W. B. Saunders, Co., 1971, pg. 348-356, 374, 381-388.

13. The Editors: Color. Encyclopeida Britanica, Macropedia, 1974, Vol. 3, pg. 22-23.

14. Sproul, R. C.: Color Matching in Dentistry, Part II, The Three-Dimensional Nature of Color. Journal of Prosthodontic Dentistry, Vol. 29, No.5, May 1973, pg. 556-566.

15. Sproul, R. C.: Color Matching in Dentistry, Part III, Color Control. Journal of Prosthodontic Dentistry, Vol. 31, No. 2, Feb. 1974, pg. 146-154.

Digitized by Google

16. Chaitin, P., Maxwell, J.: Color, Life Library of Photography, Time-Life Books, New York, 1970.

17. Culpepper, W. D., Mitchell, P. S., Blass, M. S.: Esthetic Factors in Anterior Tooth Restorations. Journal of Prosthodontic Dentistry, Vol. 30, No. 4, Part 2, Oct. 1973, pg. 576-582.

18. Ceramco Color System. Ceramco Equipment Corp., Revised Ed., 1968.

19. Wilkinson, A. H. B.: The Science of Color in Dentistry. National Association of Certified Dental Laboratories Journal, Vol. 27, No. 11, Nov. 1970, pg. 6-10.

20. Ralph, W. Phillips: Shinner's Science of Dental Materials .W. B. Saunders Co., Philadelphia, Pa., 7th Ed., 1973, Chapter 31.

21. Bergen, S. F., McCasland, J. P.: Dental Operatory Lighting (Color) Study. Unpublished, 1974.

22. Bergen, Stephen F.: Veterans Administration Hospital, New York City, New York, Dental Service, Personnel Communication.

23. Sturdivant, C. M.: The Art and Science of Operative Dentistry, The Blakiston Division, McGraw-Hill Book Co., 1968, pg. 479-480.

24. Baum, L.: Advanced Restorative Dentistry, Modern Materials and Techniques, W. B. Saunders Co., Philadelphia, Pa., 1973, pg. 334-335.

Digitized by Google

25. Owen, Peter A.: Art of Painting. Encyclopedia Britanica, Macropedia, Vol. 3, 1974, pg. 22-23.

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AN EVALUATION OF SKIN GRAFTS IN MANDIBULAR VESTIBULAR EXTENSIONS

by

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Professional paper submitted in partial fulfillment of the requirements of the U.S. Army Dental Residency Program, Walter Reed General Hospital, Walter Reed Army Medical Center, Washington, D.C. 20012.

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CHAPTER I

INTRODUCTION

As life expectancy increases, more individuals have edentulous jaws that have become unfavorable for the support of complete dentures. The desirability of surgical intervention to aid the prosthodontist in cases of shallow vestibular sulci or where advanced atrophy and loss of nomobile covering mucosa had occurred has been well established.[1]

It is the purpose of this paper to assess the relative merits of a skin grafting procedure by measuring the area of denture bearing tissues before and after the surgery.

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CHAPTER II

REVIEW OF THE LITERATURE

Various surgical techniques for extending the residual atrophic edentulous ridge to provide a more satisfactory denture foundation using grafted skin have been reported since 1916.

Moskowicz[2] and Esser[3] first described a skin grafting procedure using an extraoral approach from an incision in the submental region.

Weiser[4] and Pickerill[5] improved the technique by using an intraoral approach. The graft-covered stent was inserted through the mouth and mucous membrane was closed over it.

The buccal inlay technique was first described by Gillies[6]. He used this technique in the maxilla in which the labial sulcus was insufficient.

Pichler[7] was the first to apply skin grafts to raw surfaces of the postmaxillectomy cavities to prevent shrinkage of the soft tissue and to hasten healing.

Schuchhardt[8] suggested that the periosteal surface,

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not the soft tissue side, be covered with skin. He sutured the free mucosal edge in the depth of the newly created vestibular sulcus to the periosteum. The shrinkage of the labial tissues, which is a problem in the buccal inlay technique, could be lessened.

The combination of vestibuloplasty with skin grafting and simultaneous lowering of the floor of the mouth, as described by Obwegeser[9], has been another improvement to provide a better foundation for a prosthesis in the mandible. This technique is the one used by the Oral Surgery Service at Walter Reed Army Medical Center.

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CHAPTER III

DISCUSSION

The split thickness skin graft to the mandibular ridge with vestibuloplasty and lowering of the floor of the mouth is a combination prosthetic, surgical management of the grossly resorbed mandibular ridge.

This procedure is not recommended for every patient that presents with extensive loss of residual mandibular ridge. The prosthodontist and oral surgeon should hold a joint evaluation prior to any decision to perform surgery and plan prosthodontic treatment.[10]

A complete dental history is necessary. From this may be determined some idea for the etiology of the loss of the residual ridge. All of the patients basically exhibit the same symptoms. They state that the lower denture "flops" around with no stability. Another complaint is soreness on the mandibular ridge associated with chewing. Adjusting for a sore spot will give them temporary relief. They will return in a few days with a similar complaint of a sore spot in another area. Patients usually give a history of wearing their denture twenty four hours a day with no concept of the proper way

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to function with their dentures or how to maintain their oral tissues. An oral and roentgenographic exam will reveal severe atrophy of alveolar bone with little or no attached mucosa. Their existing dentures will be underextended, unstable and have an unsatisfactory occlusion. There is excessive wear on the occlusal surfaces of the posterior teeth with a resultant loss in vertical dimension of occlusion. This brings the anterior teeth into contact which has set up unfavorable stresses on the anterior part of the ridge.

The first phase in the treatment is to make a diagnostic set of dentures using accepted prosthetic techniques with adequate extension, establishing a more favorable occlusion. Proper instructions to the patient are necessary to make sure he understands the correct way to function with these dentures. I am referring the necessity of him chewing bilaterally simultaneously in the posterior area with no anterior function. If the patient can manage with the new dentures all well and good, but if all this fails, and the patient still has problems, the split thickness skin graft procedure will be recommended with the ultimate goal of providing a more resistant tissue for the denture bearing area which will hopefully

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improve stability and retention of the lower denture.

While we are in this diagnostic phase of treatment, it is important to evaluate the mental attitude of the patient to determine what degree of acceptance, responsibility and cooperation can be expected. The patient must understand the extensiveness of the procedure, particularly the necessity for post operative prosthodontic observations and adjustments. His attitude must be such that he not only desires the treatment, but is willing to understand and accept the limitations of denture performance prior to treatment.

The patient's physical status and medical history should be carefully reviewed to reduce any surgical or post operative complications. He should fully understand the proposed surgical procedures, the expected tissue responses, the post operative difficulties and anticipated prognosis. A good working rapport, which allows the patient to be an active participant, is essential.

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CHAPTER IV

MATERIALS AND METHODS

For the purpose of this study, it was decided to use a 28 gauge relief metal, (Ash Mfg. Co.), in conjunction with a weight-volume formula, to obtain our measurements of the surface area of the denture bearing tissues. A total of three cases will be discussed. The measurements were made on stone casts poured from final impressions using accepted prosthetic techniques both pre and post operatively. The post operative impression was made approximately four months after surgery.

Using a Torsion Balancer, a square centimeter section of relief metal was found to weigh .469 grams. Using a micrometer the height or thickness of the metal was determined to be .04293 centimeters. The density of the metal was then calculated by using the following formula:

$$\text{Density} = \frac{\text{Weight}}{\text{Volume}} = \frac{.469 \text{ grams}}{1 \text{ cm}^2 \times .04293 \text{ cm}} = 10.925 \text{ gm/cc}$$

The density (10.925 gm/cc) and height (.04293 cm) are standard figures which will be used throughout our formulas to determine the measurements of the surface area.

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An outline of the total denture bearing area was drawn on each cast. A section of relief metal was cut and adapted to this outline and then weighed. A second outline of just the crest of the ridge area was then drawn on each cast. The previously mentioned section of relief metal was then cut back and adapted to this area and then weighed.

Using the following formulas, the volume and area of each section of metal could then be determined.

$$\text{Volume} = \frac{\text{Weight}}{\text{Density}} \quad (10.925 \text{ gm/cc})$$

$$\text{Area} = \frac{\text{Volume}}{\text{Height}} \quad (.04293 \text{ cm})$$

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CHAPTER V

RESULTS

		Pre. Op.	Post Op.	
Total Denture Bearing Area	Case # 1.	19.239cm^2	17.528cm^2	— 8.9%
	2.	26.755cm^2	24.106cm^2	— 10%
	3.	22.525cm^2	20.643cm^2	— 9.1%
Crest of Ridge Area	Case # 1.	8.896cm^2	12.243cm^2	+ 27.4%
	2.	12.772cm^2	14.187cm^2	+ 10%
	3.	11.250cm^2	15.468cm^2	+ 27.3%

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CHAPTER VI

CONCLUSIONS

The measurements show that the total denture bearing area decreased. The area over the crest of the ridge increased. This results from the surgical procedure itself. Most of the buccal pouch is obliterated. A certain amount of vestibular height is lost because of scarring at the skin-mucosa junction after final healing. The highly displaceable soft tissue over the ridge is replaced with a better denture bearing surface of grafted skin.

All of the patients who have undergone this graft procedure have responded well. They claim they have better stability and retention than they had before their ridges resorbed with their original dentures. Prior to the surgery, the thin, movable, elastic tissue with many sensory nerve endings could offer no resistance to the occlusal forces disseminated under the denture. The substitution of a keratinizing, non-elastic tissue with less sensory nerve endings has significantly reduced the complaint of soreness on the mandibular ridge associated with chewing.

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CHAPTER VII

SUMMARY

1. The total available denture bearing area has decreased.

2. The denture bearing area over the crest of the ridge has increased.

3. Non-elastic, keratinizing tissue with less sensory nerve endings has been substituted for highly thin, elastic, non-keratinizing tissue with many sensory nerve endings.

4. The floor of the mouth has become non-displacing and is effectively separated from the buccal mucosa.

5. Subjective approval of the procedure has been unanimous.

It is very gratifying to see the response of these patients after the graft procedure. With careful case selection, some of these hopeless situations can be treated with this combined prosthetic-surgical technique to provide a more favorable tissue for the mandibular denture bearing region.

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BIBLIOGRAPHY

1. Guernsey, L. H. : Preprosthetic Surgery, Dental Clinics of N. Am., p. 447, April 1971.
2. Moskowicz, L. : Ueber die Verpflanzung Thierscher Epidermislaeppchen in die Mundhoehle. Arch Klin Chir 108:216, 1916.
3. Esser, J. F. : Studies in Plastic Surgery of the Face, Ann. Surg., 65:297, March 1917.
4. Weiser, R. : Ein Fall von ankylose, Verlust des Alveolarfortsatzes und der Vestibulum oris im Bereich fast des ganzen Unterkiefers. Oest Ungar Vierteljahrsch Zahnheilk 34:147, 1918.
5. Pickerill, H. P. : Intra-oral Skin-Grafting.: The Establishment of the Buccal Sulcus, Brit. J. D. Sci. 32:135-141, 1919.
6. Gillies, H. D. : Plastic Surgery of the Face: Based on Selected Cases of War Injuries of the Face Including Burns, London, 1920, Henry Frowde and Hodder and Stoughton, pp. 8-12.
7. Pichler, H. : Zur Behandlung bosartiger Oberkiefer-geschwulste, Arch. Klin. Chir. 167: 769-775, 1931.

Digitized by Google

8. Schuchhardt, K. : Die Epidermistransplantation bei der Mundvorhofplastik. Deutsch Zahnaerztl Z 7: 364, April 1952.

9. Obwegeşer, H. : Surgical Preparation of the Maxilla for Prosthesis. J. Oral Surg., 22:127, March 1964.

10. Heartwell, C. M. and Peters, P. B. : Surgical and Prosthodontic Management of Atrophied Edentulous Jaws, J. Pros. Den., 16:614, 1966.

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A CLINICAL AND HISTOLOGIC EVALUATION OF THE EFFECTS OF SELECTED DENTIFRICES ON HUMAN PALATAL MUCOSA

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INTRODUCTION

As early as 1930, the American Dental Association recognized the need for controlling the ingredients of dentifrices with regard to both safety and effectiveness. Yet in 1947, the Council on Dental Therapeutics decided to "...discontinue the consideration of cleansing dentifrices with a view toward their acceptance,...and give particular attention to dentifrices for which therapeutic claims were made."[1]

The American Public spent $386,000,270 on dentifrices in 1971.[2] This is more than double the amount spent in 1953,[3] and may be the reason that dentifrice advertising is so conspicuous and competitive in nature. Recently, much emphasis has been placed on the cosmetic qualities such as "whiter teeth" and "fresher breath". Most dentifrice products consist of from six to ten components (Table #1).

Dentifrices which have only cleansing, refreshing, and/or deodorizing action are defined as cosmetics, and are not required to prove both safety and effectiveness as required by the 1962 Kefauver-Harris Amendment to the Federal Food, Drug, and Cosmetic Act.[4]

Recent clinical and experimental evidence suggests that some dentifrices have adverse effects on oral tissues. If research continues to support these findings, perhaps dentifrices should be considered medicinal agents and regulated and evaluated as such.

REVIEW OF THE LITERATURE

Much attention has been directed to the effects of dentifrice abrasives on the hard dental structures,[5,6] but very little information exists concerning the effects of dentifrice components on the soft tissues of the oral

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cavity. For the purpose of review, the adverse effects on the soft tissues may be classified as true hypersensitivity reactions to one or more components, or chemical irritation with resultant tissue response.

HYPERSENSITIVITY: Allergic reaction to essential oil flavoring agents is not uncommon, although not widespread. These oils are used in combination and are present in 0.5 to 2.0 per cent concentration in dentifrices.[7] In addition to the main flavors of spearmint, peppermint, wintergreen, and cinnamon mint, many other oils are blended with the main agent to lend distinctive flavor to the dentifrice.

In 1948, Winter[8] reported an allergy to Orris Root used in tooth paste. Oral examination of the patient revealed a bright red appearance of the gingival tissues, enlargement of the interdental papillae, and loss of stippling. A thorough history implicated the patient's dentifrice as the etiologic agent. When a similar dentifrice was compounded without Orris Root, the tissues improved markedly within ten days, and returned to normal within a month.

Cheilitis was reported as a manifestation of allergy to oil of cinnamon.[9] Angular lesions of two weeks duration healed in five days following disuse of the dentifrice. Subsequent testing of the individual components confirmed the allergic nature of the disorder.

Smith[10] reported an acute allergic reaction following the use of tooth paste. The patient displayed massive edema of the mandibular tissues, floor of the mouth, neck and pharynx. Following initial treatment, patch tests were positive for the essential oil, Oleum Menthae Piperitae.

Hjorth and Jervoe,[11] as quoted by Guarnieri,[12] reported five cases of allergy to essential oils. One of the patients could tolerate a brand of dentifrice not containing fluoride but reacted to the same brand with fluoride. Further analysis, however, revealed that the essential oil com-

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position was different for the two dentifrices and that an essential oil and not the fluoride was to blame. If careful analyses were not done in this case, fluoride might have been mistaken for the offensive agent. For this reason, care must be exercised when interpreting some of the literature implicating fluoride as an allergen.

Douglas[13] reported one hundred and thirty-three cases of allergy to fluoride tooth paste. He reported that oral ulcerations were refractory to antibiotic therapy but cleared when a substitute non-fluoride tooth paste was used. No attempt was made to test individual components for allergic response, and therefore the conclusions drawn from his observations are subject to question.

Antiseptic agents such as Diclorophene, once used in an ammoniated dentifrice, have been found to be allergenic.[14] Nine cases were observed over a six month period which displayed clinical findings such as glossitis, stomatitis, and circumoral dermatitis. These were confirmed with patch testing procedures.

Hjorth and Jervoe[11] tested an essential oil combination which had a proven allergenic potential on two hundred and six consecutive patients in their practice and six demonstrated hypersensitivity. The incidence of true allergy to the components of a dentifrice, therefore, is probably quite small. There is increasing evidence, however, that there are far more subtle yet widespread effects of dentifrice on the oral tissues which affect a much higher percentage of the population than might be expected.

CHEMICAL IRRITATION: The components most often implicated in chemical irritation are the surfactants, the essential oils and additive agents.

(1) Surfactants or detergent wetting agents: The following references[15, 16, 17] are quoted by Guarnieri[12] from the foreign dental literature.

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Mourgues[15] feels that wetting agents can penetrate the tissues causing cellular edema and inflammation. When compared to a hypertonic saline control solution, wetting agents were shown to produce cellular edema. However, no specified number of patients was stated, the agent was not identified, and the agent was not used in combination with other agents found in dentifrices.

Bossard[16] feels that wetting agents attract water and result in hyperhydration of the tissues. He states that Haim found morphologic changes in the tissues and a change in the distribution of the mucopolysaccharide component of connective tissue.

Riethe[17] studied the effects of commercial wetting agents on the oral tissues of ninety patients. He found that the adverse effects of these agents were dependent on their concentration.

Kitchen and Graham[18] reported that one of the commonly used surfactants (sodium alkyl sulphate) had no deleterious affects on oral mucous membranes of four dogs which were studied histologically, and thirty-nine dental students on which clinical evaluation was done. However, this is only one of several similar agents in common use.

The efficacy of these agents in the oral cavity has not been proven. Accepted Dental Therapeutics[19] reports that since these agents lower surface tension they are able to wet surfaces which are normally water repellent. Perhaps their ability to disperse the lipid fraction of tooth accumulated materials is helpful in allowing agents such as fluoride to gain access to enamel, but this has yet to be proven. Probably the most important reason for their use is that the public apparently prefers a foaming dentifrice.[7]

(2) The Essential Oils and Additive Agents: The volatile oils are capable of causing chemical irritation as well as hypersensitivity even

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though used in relatively small concentrations.[7]

Product surveys have shown that taste is the most important single factor governing the selection of a family dentifrice.[3] The search for pleasing taste sensations has led to the incorporation of higher percentages of essential oils. In some instances these are used in combination with chloroform. Gershon and Pader[7] point out that it is extremely difficult to accurately determine the level of essential oils in a given dentifrice with current analytical techniques. Since these are volatile oils, a fresh tooth paste may be more pungent than an aged one of the same type. They advise caution in the use of chloroform in a dentifrice because it can decompose to hydrogen chloride and attack certain metals used in fabricating the containers. A recent edition of Accepted Dental Therapeutics refers to the possible effects of chloroform on the tissues in these terms:

> "Chloroform acts locally as a penetrating agent and fairly powerful irritant which may even result in blisters."[19]

Although the concentration of this agent is only three to four per cent in some dentifrices, the potential for irritation certainly exists.

(3) General Reaction to Tooth Pastes: Wannenmacher,[20] as quoted in the World Workshop in Periodontics 1966,[21] used exfoliative cytology to study the effects of dentifrice on the soft tissues. He demonstrated that all tooth pastes tested tended to increase the number of folded cells and pyknotic nuclei when compared to the oral tissues of non-dentifrice users. The Workshop concluded:

> "Efforts might be directed to the development of dentifrice materials less harsh to the oral tissues."

In 1969, Surface[22] noted that many of his patients began to complain of thermal sensitivity and gingival irritation which he traced to the newly

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marketed, highly advertized "get'em white" tooth pastes. He found that symptoms subsided in most cases ten days after discontinuing the use of the dentifrice. Cervical sensitivity at times persisted. He concluded that the "super white" dentifrices are detrimental to oral health causing both marginal gingivitis and cervical sensitivity in many people. Whether or not this firm a relationship can be established based on the evidence the author presented is questionable. Perhaps irritation of the tissues by the tooth pastes led to inadequate oral hygiene procedures which then led to the clinical observations. In any case, his report may have direct clinical significance.

The most comprehensive study on the effects of dentifrice on oral tissues can be found in ~~an unpublished~~ a report by Guarnieri.[12] His work can be divided into preliminary animal studies and follow-up human studies.

In the animal experiments, chloroform, the essential oil concentration, and detergent concentration were evaluated as variables. Different combinations and concentrations of these variables were used in a common dentifrice base. Test compounds were applied to the lower labial gingiva of adult guinea pigs with a cotton swab for one minute, three times a day for five days. In the control group, water was applied in the same manner. Distilled water was used to remove the test agent and the area was dried with absorbent cotton. Visual examinations were made each day and prior to sacrifice. Erythema, denudation and ulceration were selected to serve as the criteria for a clinical grading system from zero (same as baseline) to four. Histologic sections were graded on the basis of cellular infiltration, ulceration, and thickness of the keratin layer. Animals were sacrificed on the sixth day, the tissues were processed and stained with Hematoxylin

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and Eosin and Periodic Acid Schiff. Significant findings in the animal studies are summarized below:

(1) A dentifrice containing chloroform (3 to 4%), essential oils (1.3%), and sodium N-lauroyl sarcosinate (1.1%),* produced ulceration of the guinea pig tissues under the experimental conditions.

(2) Sodium N-lauroyl sarcosinate (1.1%)+ was the only one of these three variables to show tissue reaction when used alone in a common dentifrice base.

(3) When chloroform and the essential oils were used in combination, there appeared to be an additive irritating effect.

The latter finding points to the necessity for testing the ingredients used in dentifrices not only separately but in the combinations and concentrations used in the commercial preparation.

Guarnieri followed this work with two human clinical studies. In the first study, forty-four dental students were used in a supervised brushing experiment. The purpose was to compare the effects of Ultra Brite which had produced ulceration in the guinea pigs with one which had been commercially available for a longer period of time and had gained wide acceptance by the American Public.#

Each subject was given a soft, multitufted, nylon brush. The first week, the students brushed two times a day for one minute. Week two, they brushed three times a day for one minute. The third week, the students brushed three times a day for two minutes with an extra ribbon of dentifrice added at the end of the first minute. The subjects were examined initially

*Ultra Brite, Colgate-Palmolive Co., New York, N. Y., 10022
+Gardol, Patent by Colgate-Palmolive Co.
#Colgate with MFP, Colgate-Palmolive Co.

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and at the end of each week for oral changes. Also, if a subject had an oral complaint an examination was given at that time. Differences in response were statistically significant only at the end of the third week. At this time, 66.7 per cent of those using Colgate MFP showed clinical inflammatory changes, compared to 88.9 per cent in the Ultra Brite group. Moreover, at the end of the third week, the Colgate group had voiced 110 complaints while the Ultra Brite group had 240 complaints. This finding also proved statistically significant. The most frequent complaints in order of decreasing frequency were:

"burning"

"tingling sensation"

"soreness"

A second study was designed to determine which ingredients were most irritating to the oral tissues. The subjects were prisoners at a local correctional institution. Four groups of seventy-five subjects were utilized. They were given the following test dentifrices:

(1) Ultra Brite

(2) Ultra Brite without chloroform.

(3) Ultra Brite with reduced essential oil concentration (REOC).

(4) Ultra Brite with REOC and without chloroform.

The subjects were supervised daily for three brushings of one minute duration for four weeks. The oral tissues were examined weekly and a cytologic smear was taken from the lower anterior vestibule and evaluated with Papanicolaou stain. Results of the clinical portion at the end of the four weeks did not show any statistically significant differences among the four groups. When the baseline clinical evaluation charts were compared with the clinical findings at the end of four weeks, it was found that there was

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a highly significant difference with regard to oral irritation present. Oral irritation was defined as color changes (mild, moderate, severe erythema), denudation, or ulceration as compared to adjacent tissues. The differences in the cytology were found to be significant when comparing the chloroform to the non-chloroform containing dentifrices. The blue to yellow cell ratios were altered. The yellow cell counts were highest for the chloroform groups and lowest for the groups not using the chloroform containing dentifrice. Just the opposite was true for the blue cell counts indicating a more acute inflammatory reaction with the chloroform containing tooth pastes. These changes were seen throughout the population tested suggesting that they were not of an allergic nature.

In summary, the results of these studies implicated three components of the test dentifrices as possible etiologic agents in oral inflammatory changes. These three components were:

(1) Sodium N-lauroyl Sarcosinate: a detergent wetting agent, the only component found to be capable of producing lesions in guinea pigs when used alone.

(2) Chloroform: an agent used to enhance flavor, associated with significant cytologic changes in humans.

(3) Essential Oils: flavoring agents whose concentration appeared to be directly related to inflammatory changes especially when used in combination with chloroform.

Several clinical studies have supported Guarnieri's basic finding of irritation with the use of certain dentifrices. The United States Army Institute of Dental Research conducted several pertinent studies. Nine hundred and twenty-nine patients were asked by a receptionist at an Army treatment center, in the absence of the examining dentist, which dentifrice

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they used. These patients were then given an oral examination in which an "extreme effort was made to rule out any possible local or systemic etiological factors in any case where symptoms of gingivitis or stomatitis were present. When no other etiological factors could be found, and the patient reported recent continuous use of one dentifrice, these cases were diagnosed as dentifrice stomatitis."[23] McCleans, Ultra Brite, and Colgate(MFP) were found to be associated with the highest incidence of "stomatitis." While this study can be critisized for the lack of controls, uncertainty of the cause-effect relationship, and vagueness of the term "stomatitis", it probably has clinical significance. This study was repeated by different investigators at a different location and the results paralleled those of the initial study.

More recently, Stec[24] has reported a possible relationship between desquamation and the use of dentifrice. In a clinical evaluation of desquamation as indicated by the use of disclosing agents, she reported that 109 of 213 patients evaluated had some degree of desquamation. Five patients who did not use dentifrice did not show the characteristic epithelial slough. Subjective complaints such as "burning" or "hot" feeling were found with some patients. Some dentifrices particularly the "whitener" brands, showed a very high incidence of desquamation. Since some tissue changes were seen with all dentifrices, she concluded:

"We no longer recommend dentifrices to our patients."

While this action may be too drastic, these and other findings point to the necessity for careful regulation of tooth paste composition. With patient complaints such as "burning" and "soreness", one might expect detrimental effects on the performance of oral hygiene. A sore mouth certainly does not encourage thorough disease control practices by the patient.

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PURPOSE

None of the above mentioned studies or surveys have demonstrated adequate controls in humans. No information is available as to the histological effects of dentifrice on human tissues. This study was designed to evaluate the clinical and histological effects of selected dentifrices on human masticatory mucosa.

MATERIALS AND METHODS

24 volunteer subjects were selected for the clinical evaluation according to the following criteria:

(1) All patients denied a history of allergy to dentifrice.

(2) All patients presented with palatal mucosa which appeared clinically normal.

(3) All patients retained enough maxillary teeth to hold an acrylic stent firmly in position against the palatal mucosa.

(4) No patients wore maxillary removable prostheses.

In addition, the first ten subjects underwent biopsy for histologic examination of the palatal mucosa.

An alginate maxillary impression was secured for each patient and a palatal stent constructed with six receiving depots (figure 1A). All depots were beaded at the periphery (figure 1B) to help localize the test dentifrices and to demarcate both test and control sites on the palatal mucosa (figure 1C). Control sites were located between the two test depots bi-laterally to minimize the potential interaction of the test dentifrices. Wire clasps were used to retain the stent in close apposition with the palatal tissues and to facilitate removal of the appliance (figure 1A).

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On the basis of the literature review, four commerically available dentifrices* were selected for study. The dentifrice positions in the test depots were rotated (figure 2) so that each test dentifrice was located an equal number of times in the anterior and posterior hard palate. This was done to minimize examiner set and reduce the possible effect of antero-posterior anatomical differences in the hard palate.

Ten days prior to the experiment, each patient discontinued the use of dentifrice and used a diluted standard mouth wash+ with his regular oral hygiene routine. On the day of the test, dentifrices were placed in the appropriate receiving depots (figure 1B) by a dental assistant who took care to remove all excess dentifrice from beyond the beaded areas of the stent. Each subject wore the stent for one hour at which time it was removed, thoroughly cleansed of dentifrice and replaced. The appliance was worn for the next seven and one half hours to protect the test sites from interference by the tongue and to allow the beading to delineate the periphery of the depots on the palate (figure 1C). The stent was then removed and the six sites evaluated by a single examiner (ALA) who was not aware of the dentifrice placement schedule. The following clinical grading system was used:

0 - no observable color change

1 - slight erythema present

2 - moderate to severe erythema apparent

Kodachrome II photographs were taken of the palatal tissues.

*Colgate (MFP), Colgate-Palmolive Co., N. Y., N. Y. 10022
Crest, Proctor and Gamble, Cincinnati, Ohio 45202
McCleans, Beecham Inc., Clifton, N. J. 07012
Ultra Brite, Colgate-Palmolive Co., N. Y., N. Y. 10022

+Mann's Concentrate, Mann Company Inc., Louisville, Kentucky

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Following clinical and photographic recordings, approximately 8 hours following exposure to the test dentifrices, biopsies were taken with the Paquette instrument* (figure 3) in a manner similar to that used when securing donor tissue for gingival grafting.[25] An attempt was made to remove a full thickness strip of tissue[26] which encompassed the test and control sites. The tissue was then gross sectioned using the indentations formed by the beading as guide lines. The six segments were placed in separate labelled specimen bottles containing 10% formalin for fixation and storage until the completion of all biopsies. All specimens were then embedded in paraffin, sectioned at 5 micra, and stained with H & E and Mallory's connective tissue stain.

Sections were then examined microscopically for epithelial and connective tissue characteristics. Epithelium was evaluated for the type and degree of keratinization, the arrangement of cells and cell layers, presence or absence of necrosis, and intra-epithelial inflammatory infiltrate. The connective tissue was evaluated for fiber integrity and orientation, hyperemia and grossly assessed for inflammatory infiltration.

RESULTS

Clinical Findings: Palatal erythema was frequently noted within the peripheries of the test sites but was not observed within the controls. The individual scoring and the Chi Square analysis is depicted in Table 2. Statistical differences among the scores for the individual dentifrices were shown to be significant at .001 level. Individual dentifrices were

*Surgical knife designed to hold a portion of an ultra-sharp, steel razor blade, courtesy Dr. Omar Paquette. Hu-Friedy Mfg. Co., Inc.

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then compared to control sites with regard to erythema. The tissue response to Crest did not differ significantly from the control, while the differences between the other three dentifrices and the control were significant at .001 level. When individual dentifrices were compared with each other, Crest produced responses which differed significantly from Colgate (MFP) (< .01), McCleans (< .001) and Ultra Brite (< .001). Ultra Brite differed from Colgate (MFP) (< .05) but not from McCleans. These differences are depicted graphically in Figure 5. Subjects frequently noted "tingling", "numbness" and "burning" sensations associated with the test sites. These were subjective findings which were sometimes difficult to localize and could not consistently be attributed to any individual dentifrice.

Histological Findings: All sections were intermediate or full thickness specimens of masticatory mucosa. Occasionally, in the posterior specimens, the transition from masticatory to posterior lining mucosa could be seen. Mucous glands were sometimes observed in the thicker specimens.

The lamina propria in both control and experimental areas consisted of dense, functionally arranged collagen bundles which were roughly parallel to the surface in the deeper layers and perpendicular within the connective tissue papillae. Blood vessels were apparent in all layers but were especially prominent within the papillary projections. Generally, the connective tissues in both the experimental and control sections appeared normal, although generalized hyperemia and acute inflammation was associated with the most severe reactions in the experimental sections. Occasionally, focal areas of chronic inflammatory cells were seen immediately subjacent to the epithelium and were associated with a loss of

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continuity of the basal layer of epithelium. These were noted in all sections and therefore were felt to be a result of previous masticatory trauma not associated with the present experiment.

The epithelium, in contrast to the connective tissues, showed marked differences between many of the control and the experimental sections. All sections within the controls were predominantly orthokeratinized. Infrequent islands of parakeratin were noted as described previously[27] as a feature of normal palatal mucosa. Varying thicknesses of orthokeratin were associated with a distinct granular layer which appeared more prominent with thicker layers of keratin (figure 7). Occasionally, areas of hydropic degeneration were noted within the spinous cell layer, but the predominate finding within all controls was one in which the spinous and basal cell layers were regularly arranged and consistent with a mature fully keratinized oral epithelium (figures 6,7).

Many experimental sites, however, showed marked deviation from the normal palatal epithelium seen in the adjacent control areas. Out of the 40 experimental sites, 5 areas in 4 patients contained sections in which generalized necrosis and intra-epithelial abscess formation occurred (figure 11). 9 sites in 6 patients contained epithelium which showed marked widening of the intercellular spaces, isolated areas of intra-epithelial necrosis and a thickened parakeratin-like surface layer (figure 10). 14 sites in 10 patients exhibited a slightly thickened parakeratin-like surface layer with normal to slightly widened intercellular spaces (figure 9). This surface layer was frequently associated with superficial sloughing of orthokeratin and often contained remnants of keratohyalin granules (figure 15). Several authors[27-29] have noted the nearly complete absence of keratohyalin granules in the presence of

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normal parakeratin. The finding of the keratohyalin granules, the sloughing orthokeratin and the loss of cellular integrity at the interface of the surface and spinous layers in many experimental sections strongly suggested a causal relationship between the parakeratin-like surface layer and the test dentifrices.

Based upon the above findings, a grading system was developed in an attempt to access the severity of the epithelial reaction.

0 - Epithelium is predominantly orthokeratinized and contains a distinct granular cell layer, regular intercellular spaces and an orderly arrangement of all 4 cell layers.

1 - Epithelium exhibits a predominant parakeratin-like surface, slight widening of intercellular spaces and loss of distinct granular layer. Spinous and basal cell layers exhibit no areas of necrosis and no intra-epithelial inflammatory infiltrate.

2 - Epithelium possesses thickened "parakeratin-like" surface, no granular layer, local or generalized areas of necrosis. Intra-epithelial inflammatory infiltrate may be present.

Individual histologic results and Chi Square analysis are depicted in Table 3, Chi Square analysis revealed differences among the experimental sites which were significant at $< .01$. When sites exposed to Crest were compared to control areas no statistical difference was seen, while the controls differed significantly from Colgate (MFP) ($< .001$), McCleans ($< .01$) and Ultra Brite ($< .01$). Likewise, histological grading of Crest differed from Colgate (MFP) ($< .05$), McCleans ($< .05$), and Ultra Brite ($< .01$). Colgate (MFP) differed from Ultra Brite ($< .05$) but not from McCleans, while the latter did not differ statistically from Ultra Brite.

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Finally, a comparison was made between the histological and clinical findings in the first 10 subjects. When individual sites were compared with regard to clinical and histological grade, a 75 per cent agreement was noted (Table 4). The scores are depicted graphically in Figure 12.

DISCUSSION: The results support the contention that some commercially available dentifrices have the potential to produce pathologic changes in normal masticatory mucosa. Under the described conditions, there appeared to be a graded response among the dentifrices tested which in some cases was statistically significant. Crest appeared to produce a very mild reaction which did not differ significantly from the control areas clinically or histologically, while Colgate (MFP), McCleans and Ultra Brite produced pathologic changes which were readily apparent within the epithelium of the test areas. Colgate (MFP) was most often associated with mild changes, and McCleans and Ultra Brite with the more severe responses. The clinical observations roughly paralleled the histological findings.

The clinical grading system was probably less reliable than the histological system. The reliability of the clinical examiner could not be accurately accessed from this experiment. However, when the clinical examiner graded Kodachrome slides of the palatal tissues on two separate occasions an 84 per cent agreement was found. This finding may have bearing on the examiner's clinical reliability. Massler[30] feels the color photograph provides an accurate means for recording color changes in the gingiva in epidemiological studies. The decision was made to use the Kodachrome only as a check on reliability of the clinical examiner because of the small number of subjects involved and the possibility of masking subtle changes in erythema with the minute reflections and shadows

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found on Kodachrome slides. It is possible that memory played a role in the per cent agreement found. This factor was minimized by photographing at approximately 1:1 magnification, including only three sites on each slide and providing minimal memory cues.

Grading of the histologic material was done independently by two examiners. There was a 92 per cent agreement on the scoring of individual sites. The histologic grading system developed was therefore felt to be a very reliable indicator of the pathologic changes observed.

There appeared to be very little interaction among the contents of the depots. Pathologic changes were rarely found in the centrally located control depots, and then only in isolated areas which appeared to represent "breaks" in the orthokeratin, (figure 13). Occasionally, an abrupt transition was noted between the "parakeratin-like" surface layer and adjacent normal heratin. A slight depression in the surface epithelium at this interface was suggestive of the influence of the peripheral beading of the depot (figure 14). Therefore, an accurately adapted stent of this design will probably confine two adjacent dentifrices.

Clinically, it was more difficult to evalute erythema in the anterior sites when prominent rugae were present. It would have been preferable to use only relatively smooth masticatory mucosa for the clinical evaluation. Since the dentifrices were rotated (figure 2) this finding probably had little effect upon the results.

It is difficult to access the clinical significance of these findings. The potential for adverse tissue changes with certain dentifrices has been demonstrated. The orthokeratinized palatal mucosa would seem to be one of the most resistant areas in the oral cavity to external irritants if mature keratin has a protective function as believed. It is not

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known, however, how long dentifrice remains in contact with the tissues in actual use. Possibly with gingival contours altered by periodontal disease and with current crevicular brushing methods, the period of contact extends beyond that required for oral hygiene measures. If so, certain dentifrices could initiate or contribute to an existing inflammatory condition. One of the dentifrices tested produced relatively little change in the palatal tissues even with this extended contact. Care should be exercised in the formulation and testing of all dentifrices to insure distribution of a product which is compatible with the oral tissues.

SUMMARY: A study was undertaken in 24 human volunteer subjects to determine the possible effect of selected commercially available dentifrices on clinically normal masticatory mucosa. An acrylic stent was fabricated which was designed to hold four test dentifrices in close apposition to the palatal tissues for one hour. Clinical and histological evaluation approximately eight hours later revealed a response which was evident clinically and histologically. Clinically, erythema was associated with many of the experimental sites but not with the control areas. Histologically, changes were observed mainly in the epithelium. Intra-epithelial abscess formation and areas of necrosis were observed in the most severe reactions. A para-keratin like surface layer with desquamation of the orthokeratin was noted in the more mild reactions. The control areas were all predominantly orthokeratinized and normal appearing masticatory mucosa. Crest was associated with the milder reactions and did not differ from the controls statistically. Colgate (MFP) reactions differed from control but were frequently mild to moderate while McCleans and Ultra Brite were often associated with the more severe reactions noted histologically.

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Although the clinical significance of these findings could not be readily assessed, the potential for irritation of human palatal mucosa has been demonstrated. These findings suggest that care should be exercised in the formulat-on and testing of all dentifrices to insure distribution of a product which is compatible with the oral tissues.

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TABLE #1 - Tooth Paste Composition

	%	Compounds	Function
1. Abrasives	40-47	Calcium Carbonate	Cleaning and polishing Agents
		Dibasic Calcium Phosphate Dihydrate	
		Calcium Pyrophosphate	
2. Surfactants	1-3	Sodium Lauryl Sulphate	Synthetic detergents which emulsify, lower surface tension. Value not established in oral cavity.
		Sodium N-lauroyl Sarcosinate	
3. Humectants	20-25	Glycerol	Moisture retainers, non-toxic.
		Sorbitol	
		Propylene Glycol	
4. Binders	1-2	Gum Traganth	Lend viscosity to the mass and prevent colloidal separation
		Sodium Alginate	
		Irish Moss Extr.	
5. Flavoring Agents	1-2.5	Essential Oil Mixt.	Single most important factor in public acceptance.
		Saccharine	
		Cyclamates	
6. Miscellaneous	.4-1.0	Formaldehyde	Preservation
		Chloroform (3-4%)	Flavor enhancer, organic solvent
		Flouride	Therapeutic agent.
7. Water	25		
	100 total		

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CLINICAL RESULTS - 24 SUBJECTS

	0	1	2
COLGATE (COL)	8	14	2
CREST (CR)	20	4	0
McCLEANS (MC)	7	10	7
ULTRA BRITE (UB)	3	11	10

CHI SQUARE ANALYSIS

	df	X^2	P
ALL DENTIFRICES	6	35.564	.001
CONTROL - CR	2	4.364	N.S.
CR - COL	2	12.70	.01
CR - MC	2	15.96	.001
CR - UB	2		.001
COL - MC	2	3.50	N.S.
COL - UB	2	7.98	.05
MC - UB	2	2.16	N.S.

TABLE 2

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HISTOLOGICAL RESULTS - 10 SUBJECTS

	0	1	2
COL	1	7	2
CR	7	3	0
MC	2	3	5
UB	2	1	7

CHI SQUARE ANALYSIS

	df	X^2	P
ALL DENTIFRICES	6	21.88	.01
CONTROL - CR	2	3.86	N.S.
CR - COL	2	8.10	.05
CR - MC	2	7.76	.05
CR - UB	2	12.28	.01
COL - MC	2	3.20	N.S.
COL - UB	2	7.76	.05
MC - UB	2		N.S.

TABLE 3

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CLINICAL AND HISTOLOGICAL: 10 PATIENTS

A. CLINICAL	0	1	2
COL	3	6	1
CR	9	1	0
MC	3	3	4
UB	0	5	5

B. HISTOLOGICAL	0	1	2
COL	1	7	2
CR	7	3	0
MC	2	3	5
UB	2	1	7

C. # OF EXPERIMENTAL

SITES	AGREE	DISAGREE	% AGREEMENT
40	30	10	75

TABLE 4

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REFERENCES

1. Council on Dental Therapeutics of the American Dental Association: New Policy on Dentifrices Announced by the Council. J. Am. Dent. Ass., 35:522, 1947.
2. Office of Business Economics: Personal Communication. 29 March 1973.
3. Bureau of Economic Research and Statistics: Family Dental Survey. J. Am. Dent. Ass., 47:573, 1953.
4. Rosenthal, M. W.: Mouthwashes. "Cosmetics Science and Technology." Eds Balsam, M. S., and Sagarin, E., Wiley Interscience, 533-563, 1972.
5. Miller, W. D.: Experiments and Observations on the Wasting of Tooth Tissue Variously Designated as Erosion, Abrasion, Chemical Abrasion, Denudation, etc. Dental Cosmos, 49:1, 109, 225, 1907.
6. Stookey, G. K. and Muhler, J. C.: Laboratory Studies Concerning the Enamel and Dentin Abrasion Properties of Common Dentifrice Polishing Agents. J. Dental Res., 47:524, 1968.
7. Gershon, S. D. and Pader, M., Dentifrices. "Cosmetics Science and Technology." ibid, 423-531, 1972.
8. Winter, G. R.: Case Report: Allergic Manifestations Caused by the Use of a Dentifrice Containing Orris Root Powder. J. Periodont., 19:108, 1948.
9. Laubach, J. L., Malkinson, F. D., and Ringrose, E. J.: Chelitis Casued by Cinnamon (Cassia) Oil in Toothpaste. J. Am. Med. Ass., 152:404, 1953.
10. Smith, I. L. F.: Acute Allergic Reaction Following the Use of Tooth-Paste. Brit. Dent. J., 125:304, 1968.

Digitized by Google

11. Hjorth, N. and Jervoe, P.: Allergish Kontaktstomatitis Ogkontakdermatis Fremkaldt of Smagsstoffer I Tandpasta. Tandlaegebladet, 71:937, 1967. (Danish)

12. Guarnieri, L. J.: The Effect of Dentifrice Components on the Oral Tissues of Humans and Guinea Pigs. Dissertation for the Degree of Doctor of Philosophy in Dental Science, Submitted to the Faculty of the Graduate School of Indiana University, 1970.

13. Douglas, T. E.: Fluoride Dentifrices and Stomatitis. Northwest Med., 56:1037, 1957.

14. Fisher, A. A. and Tobin, L.: Sensitivity to Compound G-4 (Dichlorophene) in Dentifrices. J. Am. Med. Ass., 151:998, 1953.

15. Mourgues, F.: Certaines Fausses Conceptions de Hygiene Buccodentaire, Causes Possibles des Insucces dans le Traitment des Parodontopathies, Paradontopathies (Geneve), 18:171, 1966. (Fr)

16. Bossard, F. J.: Quelle est L'hygiene buccale Preconisce Comme Prophylaxie et therapeutique Paradontale? Paradontopathies (Geneve), 18:341, 1966. (Fr)

17. Reithe, P.: Beitrag zur Wirking Grenzflachenaktiver Stoffe auf die Gingiva. Paradontopathies (Geneve), 18:347, 1966. (Ger)

18. Kitchen, P. L. and Graham, W. C.: Sodium Alkyl Sulphate as a Detergent in Toothpaste. J. Am. Dent. Ass. and Dent. Cos., 24:736, 1937.

19. Council on Dental Therapeutics of the American Dental Association: "Accepted Dental Therapeutics", 1973-1974. 35th Ed., Chicago, Am. Dent. Ass., 1972, p. 239.

20. Wannenmacher, M.: Studien Uber die Einwirkungen Von Zahpflegmitteln

Digitized by Google

auf die Gingiva and die Mundschleimhaut nach Cytologischen Untersuchen. (Effects of Dentifrice on the Oral Mucosa). Deut. Zahnarztebl. 18:413, 1964. (Ger)

21. "World Workshop in Periodontics": The American Academy of Periodontology and The University of Michigan. Ed. Ramfjord, S. M., Kerr, D. A., and Ash, M. M., Ann Arbor, Mich., 1966.

22. Surface, J. S.: New Dentifrices: A Report. J. Acad. Gen. Dent., 17:56, 1969.

23. United States Army Institute of Dental Research; Reprint #12, 1 September 1971.

24. Stec, I. P.: A Possible Relationship Between Desquamation and Dentifrice. J. Am. Dent. Hyg. Ass., 46:42, 1972.

25. Soehren, S. E., Allen, A. L., Cutright, D. E., and Seibert, J. S.: Clinical and Histologic Studies of Donor Tissues for Free Grafts of Masticatory Mucosa. J. Periodont., 44:727, 1973.

26. Sullivan, H. C. and Atkins, J. H.: Free Autogenous Gingival Grafts. I. Principles of Successful Grafting. Periodontics, 6:121, 1968.

27. Meyer, J. and Gerson, S. J.: A Comparison of Human Palatal and Buccal Mucosa. Periodontics, 2:284, 1964.

28. Wentz, F., Maier, A., and Orban, B.: Age Changes and Sex Differences in the Clinically "Normal" Gingiva. J. Periodont. 23:13-24, 1952.

29. Trott, J.: An Histologic Investigation into the Keratinization found in Human Gingiva. Brit. Dent. J., 103:421, 1957.

30. Massler, M. A.: The P-MA Index for the Assessment of Gingivitis. J. Periodont. 38:592, 1967.

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FIGURE 1A: The acrylic palatal stent in position on stone model.

FIGURE 1B: The tissue surface of the acrylic stent with test dentifrices in position. Note peripheral beading of control depot. Excess was removed (arrow).

FIGURE 1C: At the time of evaluation note the demarcation of depots on the palatal mucosa.

FIGURE 2: Technique for rotation of test dentifrices. Depots numbered 2, 5 are controls, while 1, 3, 4, 6 are test depots.

FIGURE 3: Removal of tissue with the Paquette instrument. Note the presence of depression caused by beading posteriorly (arrow).

FIGURE 4: Gross sectioning of the tissue into separate biopsy bottles using peripheral indentations as guide lines.

FIGURE 5: Graph of clinical results based upon degrees of erythema observed.

FIGURE 6: Control area showing normal but thin orthokeratinized palatal mucosa.

FIGURE 7: Control area showing normal but thick orthokeratinized palatal mucosa. Note prominent granular layer.

FIGURE 8: Experimental area given histologic grade zero.

FIGURE 9: Experimental area given grade one. Note superficial sloughing of orthokeratin.

FIGURE 10: Experimental area given grade two. Note areas of necrosis subjacent to thickened parakeratin-like surface layer of epithelium.

FIGURE 11: Experimental area given grade two severe. Note PMN infiltration of epithelium.

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FIGURE 12: Graphic representation of the clinical and histologic scores for the first 10 subjects.

FIGURE 13: Control area. Note the break in the orthokeratin with underlaying "parakeratin" (arrow).

FIGURE 14: Experimental area. Note the abrupt transition between thickened parakeratin and the adjacent keratin. Depression is suggestive of influence of peripheral beading on stent (arrow).

FIGURE 15: Experimental area. Note keratohyalin granules within parakeratin-like surface layer of epithelium (arrow).

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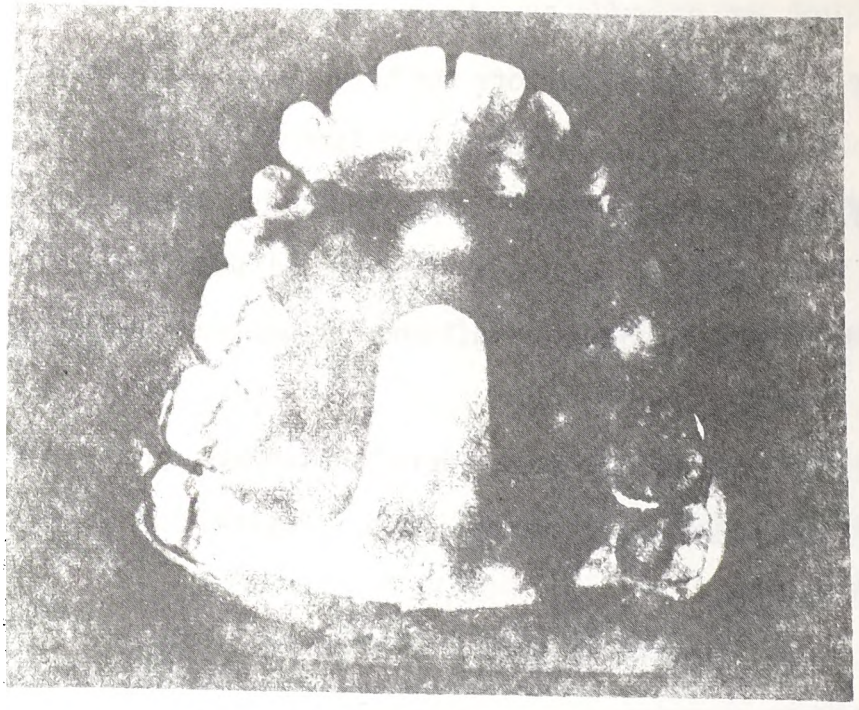

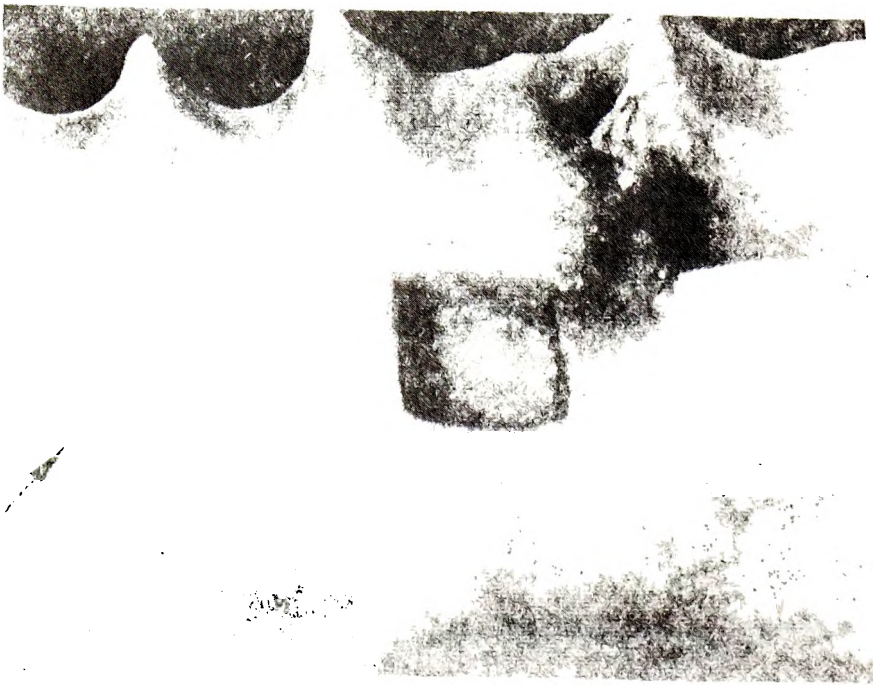

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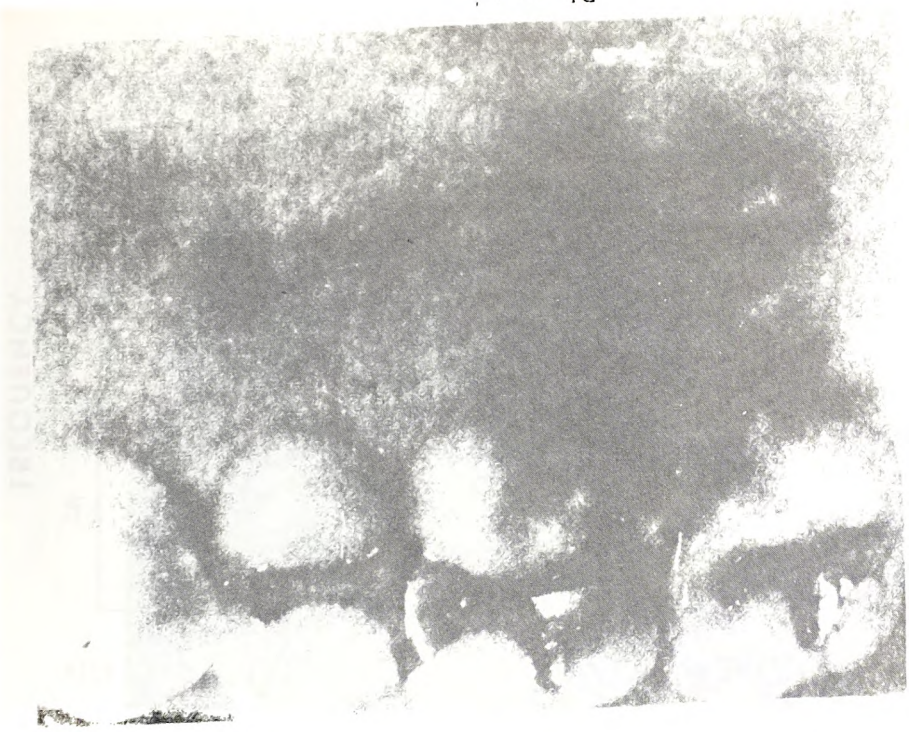

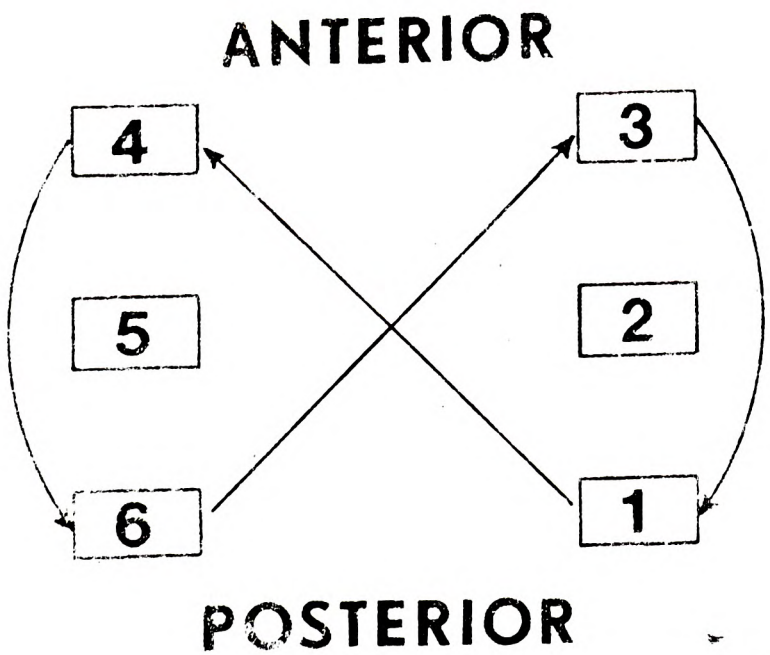

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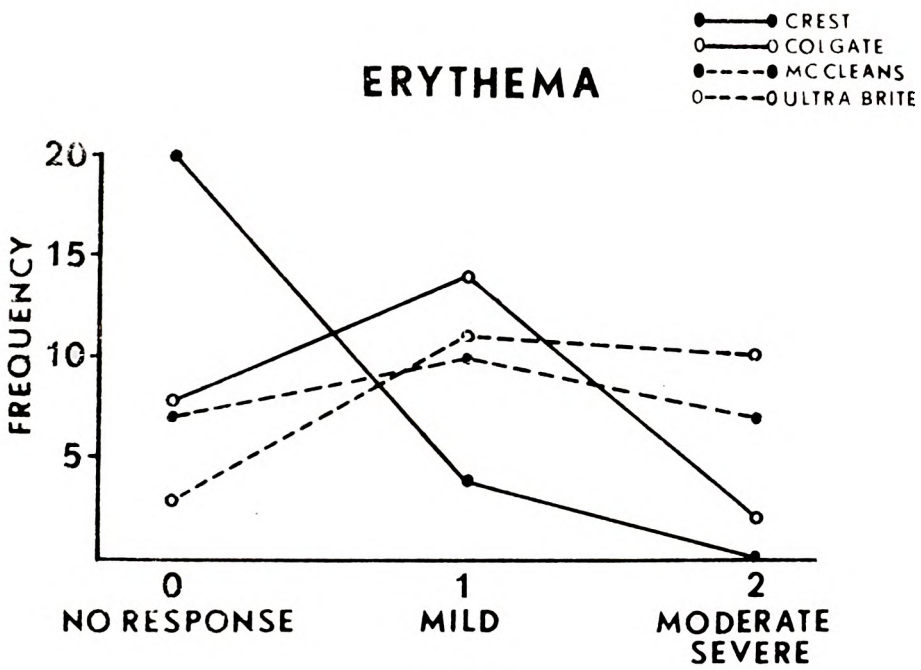

FIGURE* 5

FIGURE 6

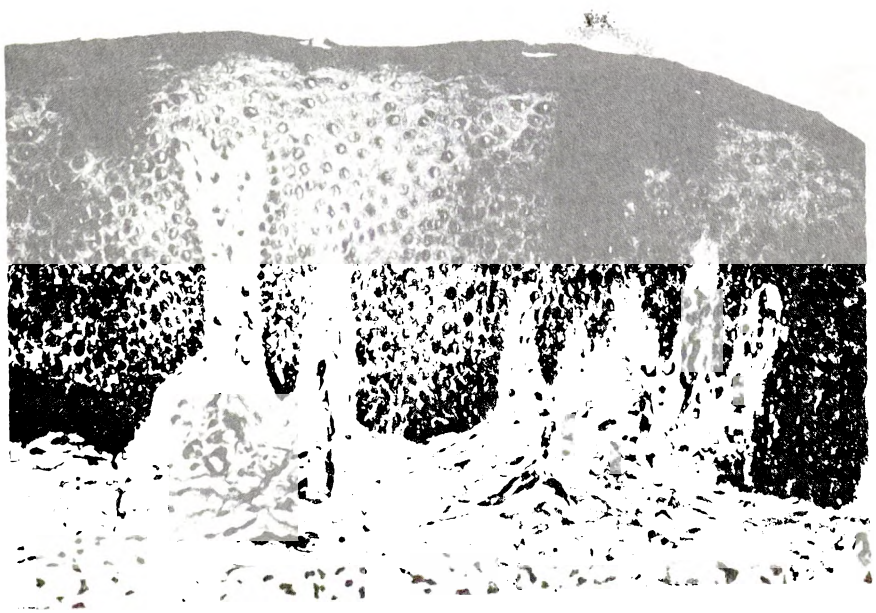

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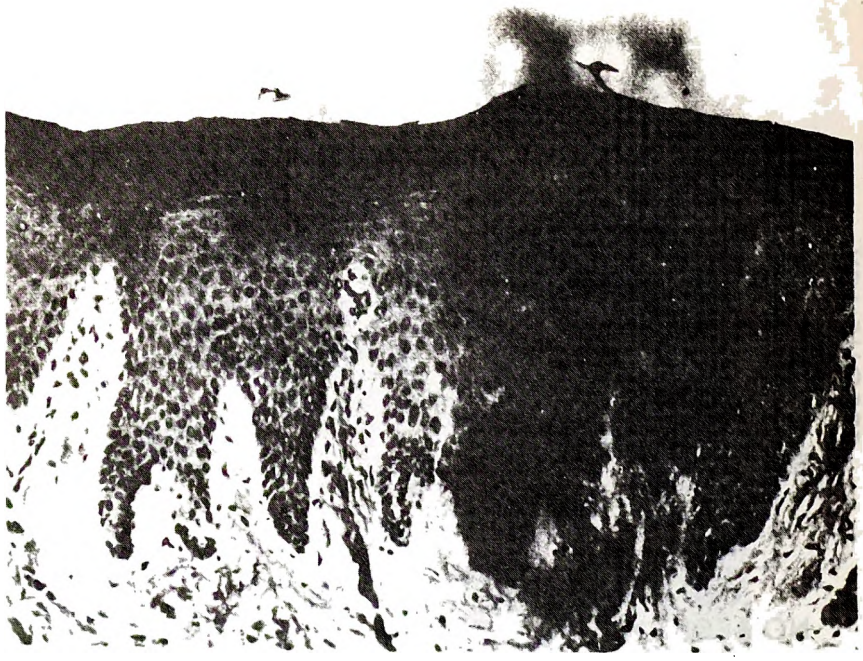
FIGURE 7



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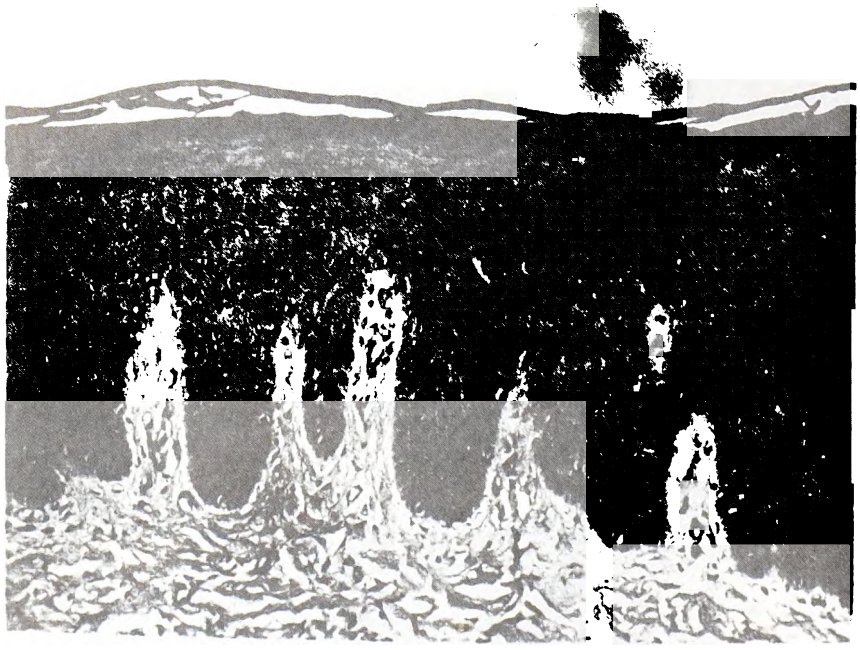

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FIGURE 9

FIGURE 10

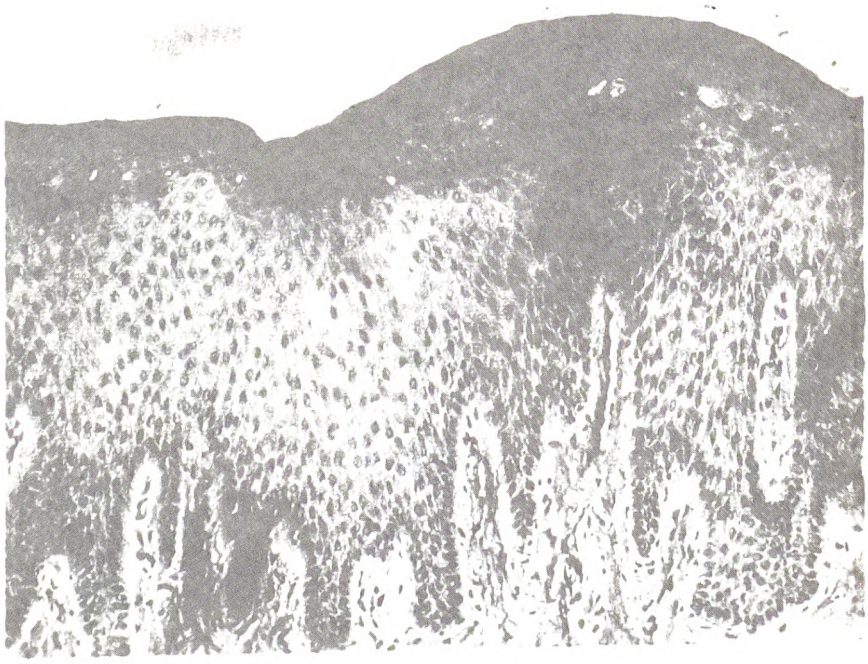

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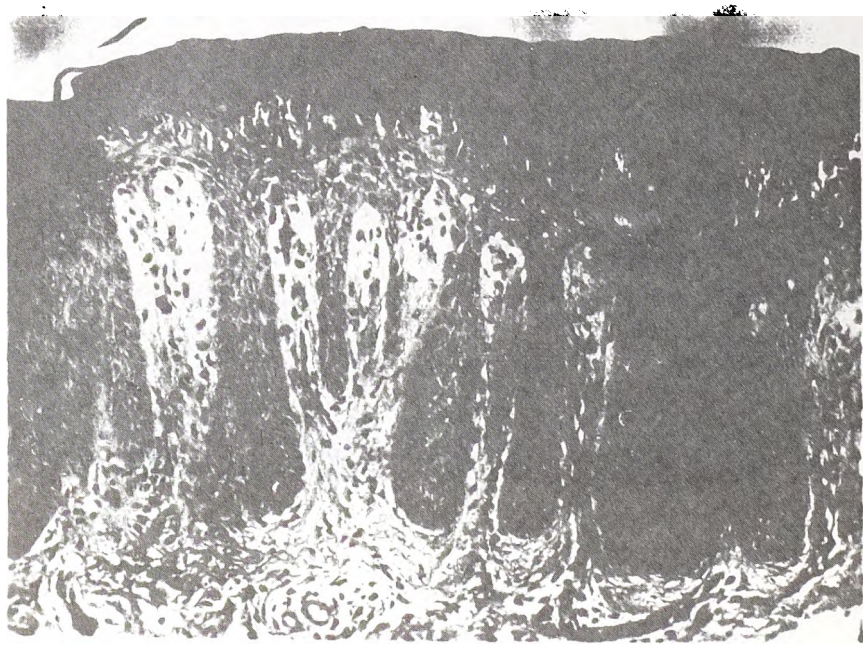
FIGURE 11



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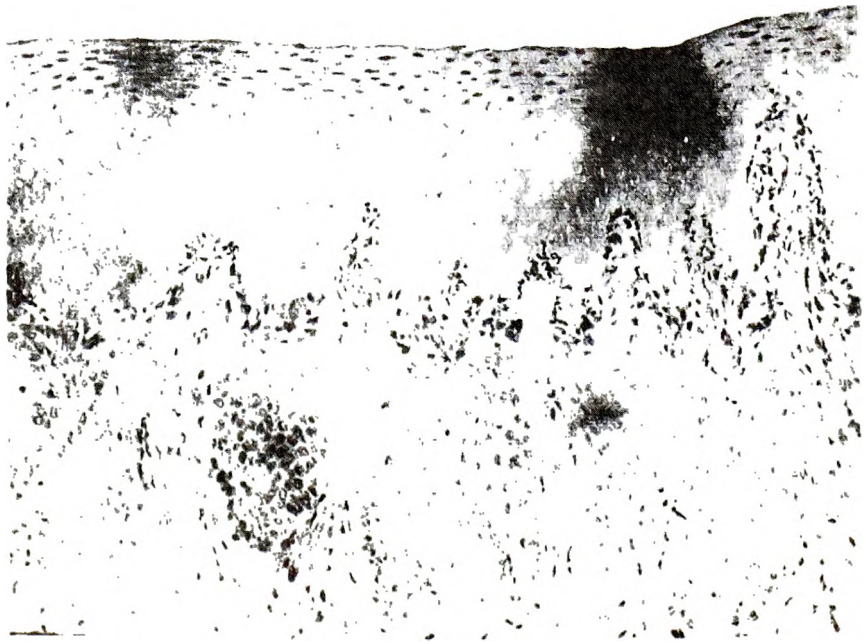

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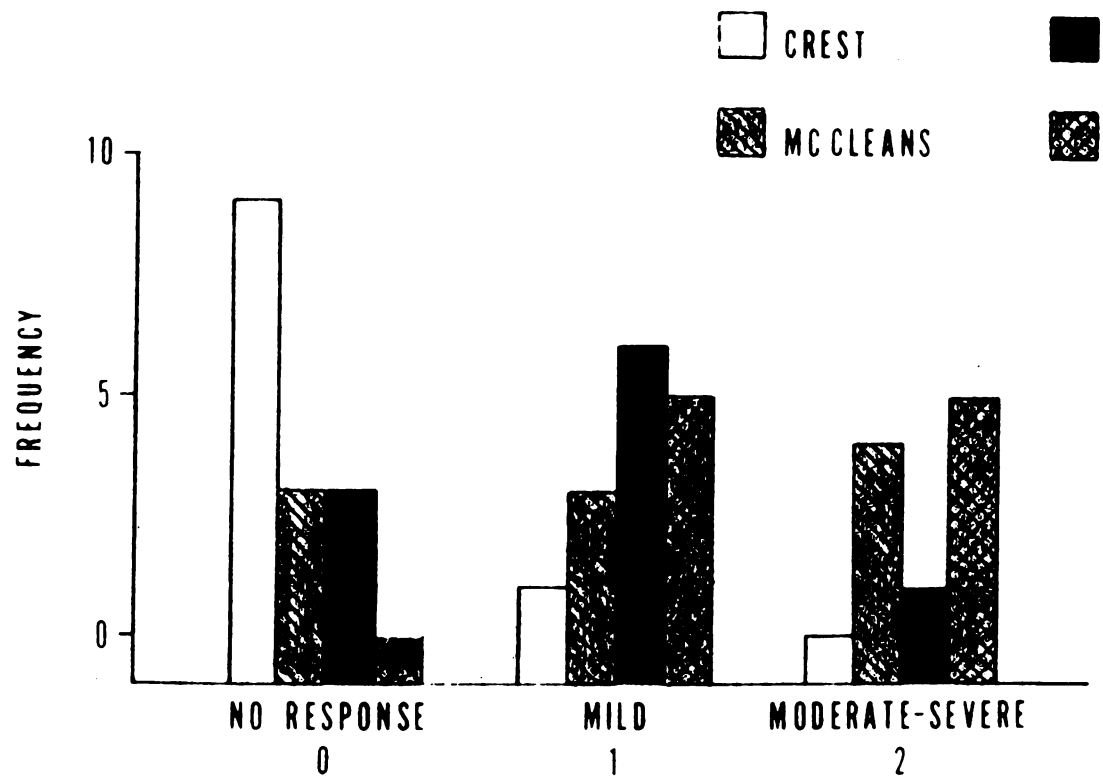
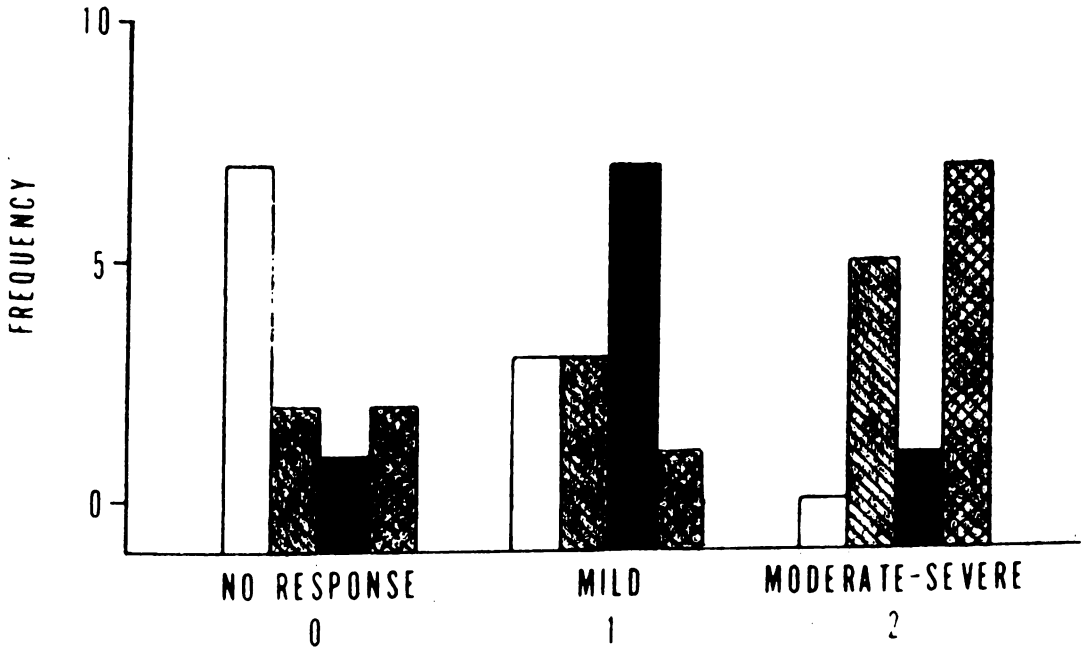


A. CLINICAL ERYTHEMA - 10 PATIENTS



B. HISTOLOGICAL 10 PATIENTS

FIGURE 12

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FIGURE 13

FIGURE 14

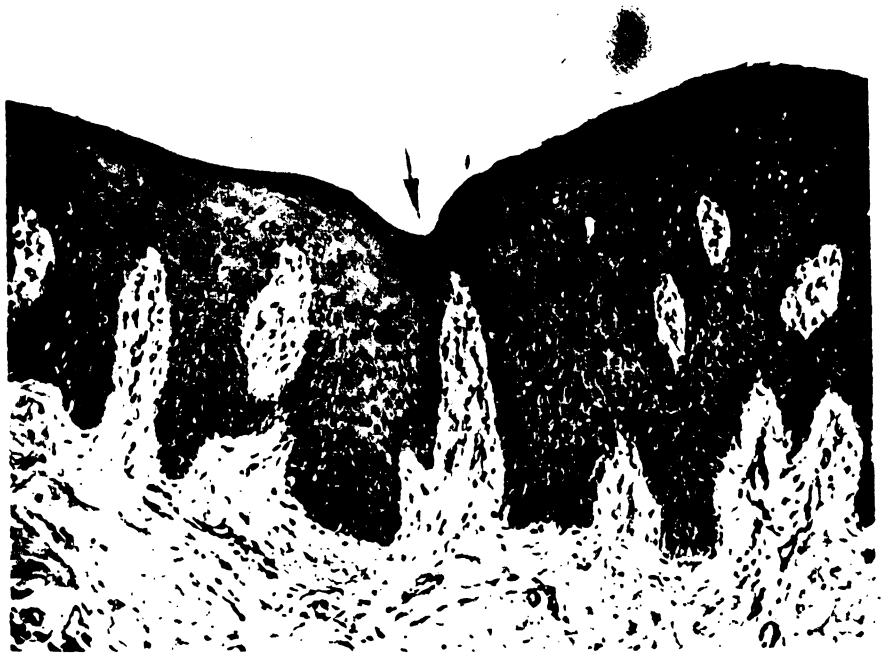

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FIGURE 15

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SCANNING ELECTRON MICROSCOPE STUDY OF RESHARPENING PERIODONTAL SCALERS

by

Charles J. Antonini
MAJ. Dental Corps, USA

Professional paper submitted in partial fulfillment of the requirements of the U.S. Army Dental Residency Program, Walter Reed Army Medical Center Washington, D.C. 20012

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CHAPTER I

INTRODUCTION

The periodontal scaler's working edge is designed to remove tooth accretions. A working edge is formed by grinding the scaler's facial and lateral surface, with natural and artificial stones commonly being used to form this working edge. Grinding techniques are accomplished by either grinding first on the facial surface and last on the lateral surface or vice versa. Evaluation of grinding stones and techniques on the working edge has ranged from clinical testing to dissecting microscopic observations, while scientific investigation of the working edge can be accomplished by a profilometer or a scanning electron microscope.

> The nature of surfaces prepared as a result of machining is characterized at present by the profilometer, which gives an indirect plot of surface roughness without giving a direct photograph of the area. In the SEM the nature of the surface is directly examined. Not only should this be of value in establishing meaningful machine standards, but it should be useful also in understanding the phenomena involved in machining metals.[1]

The scanning electron microscope is the instrument of choice when metallic surface characteristics are investigated.

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CHAPTER II

REVIEW OF THE LITERATURE

G. V. Black has stated that nothing in a dental technical procedure is more important than the care of cutting edges.[2] A periodontal scaler's cutting edge depends upon the angle formed by the facial and lateral sides. When there is an exact meeting of these sides of the instrument, an effective working edge is produced. Dull instruments are resharpened using natural or synthetic stones which act as abrasives. Because of its fine abrasiveness or grit, natural stones like Arkansas are usually recommended. It is claimed that Arkansas stones re-establish a better working edge by producing a smoother surface and a finer cutting edge. Synthetic stones (aluminum oxide, diamond, silicon carbide, and coarse) are said to produce unnecessary metal removal, rough surfaces, and wire edges.[3,4,5] Restoration of an effective cutting edge can be accomplished by grinding either the lateral or facial surface.[4,6] (See figure 1A.)

The purpose of this experiment was to compare the effects of three grinding stones and two grinding techniques on the cutting edge of a #3 Morse sickle scaler.

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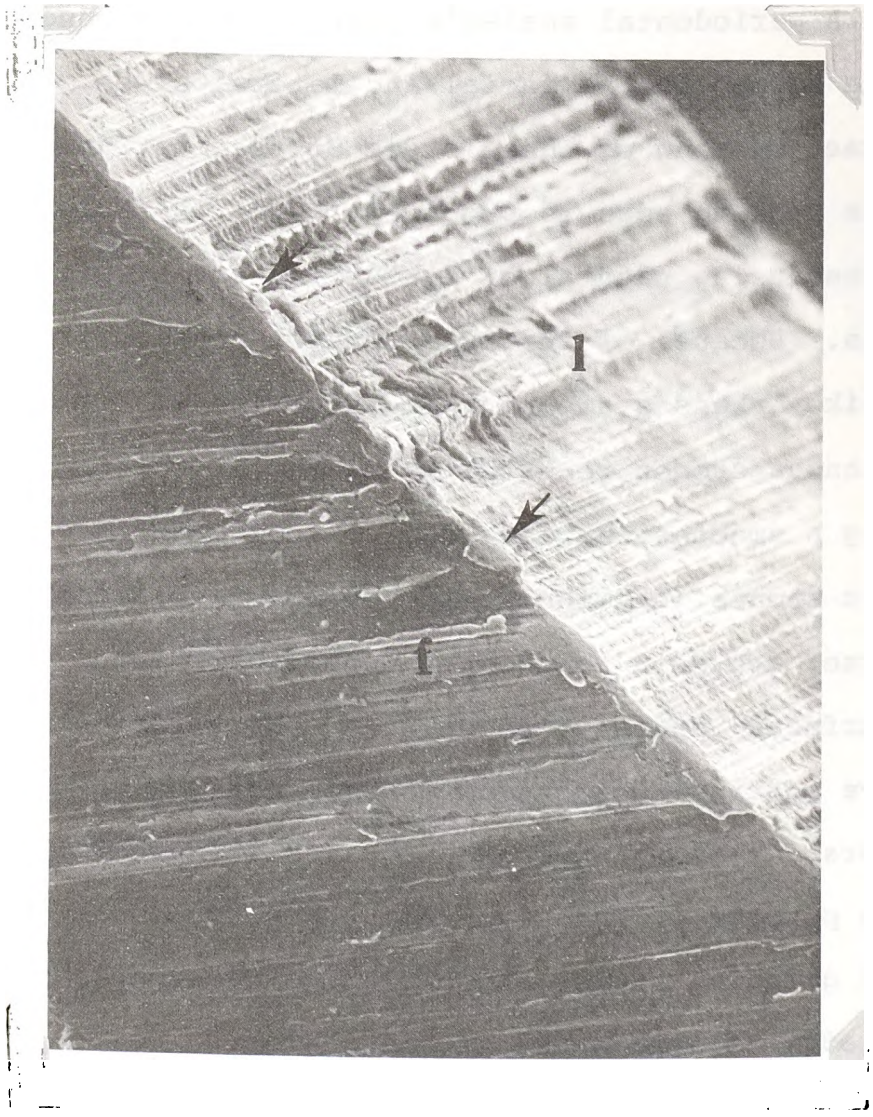

Figure 1A. Factory sharpened scaler. "f" is the facial surface and "l" marks the lateral surface. Arrows indicate the wire edges extending from the lateral surface. 500x.

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CHAPTER III

MATERIALS AND METHODS

Twenty randomly selected #3 Morse scalers were investigated at a point 1-2 mm. from the tip utilizing an American Metal Research Scanning Electron Microscope at KV 20 with a magnification multiplier of 1.00x. Each scaler was photographed at four different stages (factory edges, dulled edges, resharpened, and definned edges). Each scaler was cleaned with acetone before being placed in the SEM.

The manufacturer's sharpening technique was as follows:

> The #3 Morse scaler point is ground first on the inside (facial surface - auth.) with a 220 grit aluminum oxide wheel. The sides (lateral surface - auth.) are then ground on a 150 grit silicon carbide belt. All grinding operations are rotary.[6]

Dullness, as determined by the fingernail test,* was obtained by scaling on extracted teeth above the clinical epithelial attachment.

Fifteen scalers were ground on the facial surface by a R_x Reciprocating Honing Machine. The machine grinding schedule was five scalers by Arkansas stone (grit 500), five by diamond stone (grit 320), and five by coarse stone (grit 320).

---

*The instrument is placed at the angle used, and small strokes are made against the fingernail. When dull, it glides over the nail without cutting it.

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The five remaining scalers were ground by hand on an Arkansas stone (grit 500). All hand grinding was against the scalers' lateral surfaces and in a downward direction away from the cutting edge. The fingernail test was again used to determine the re-establishment of the cutting edge. Each re-established cutting edge was compared in a double blind study to the factory cutting edge to determine the efficacy of both the grinding stones and the resharpening techniques. The data were analyzed by Chi-square with a Yates' correction and by Fisher's Exact Test.

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CHAPTER IV

RESULTS

Both the factory and machine resharpened scalers exhibited wire edges or fins (see Figures 1A, 1B, 2, 3). However, the wire edges originated from different surfaces with Examiners J.B. and M.L. being asked to designate the surface from which the wire edge originated. The majority of the factory wire edges were found to be extensions of the lateral surfaces: Examiner J.B. - 20 out of 20 and Examiner M.L. - 19 out of 20 (see Figures 1A and 1B and Table 1). Of the fifteen machine ground scalers, Examiner J.B. noted 14 wire edges as extensions of the facial surface and Examiner M.L. notes 13 wire edges as extensions of the facial surface (see Figures 2, 3, and Table 1). Chi-square with a Yates' correction (df = 1) was used to compare data on factory and machine wire edge origins, revealing a statistically significant difference at $p < .001$ between the surface origin of the factory and machine fins. Therefore, the factory sharpening technique produced a wire edge extension of the lateral surface, while the machine grinding technique produced a wire edge extending from the facial surface.

The five hand ground scalers (see Figure 5) exhibited a high frequency of exact meeting between the lateral and facial surfaces: 4 out of 5 scalers (Examiner J.B.) and

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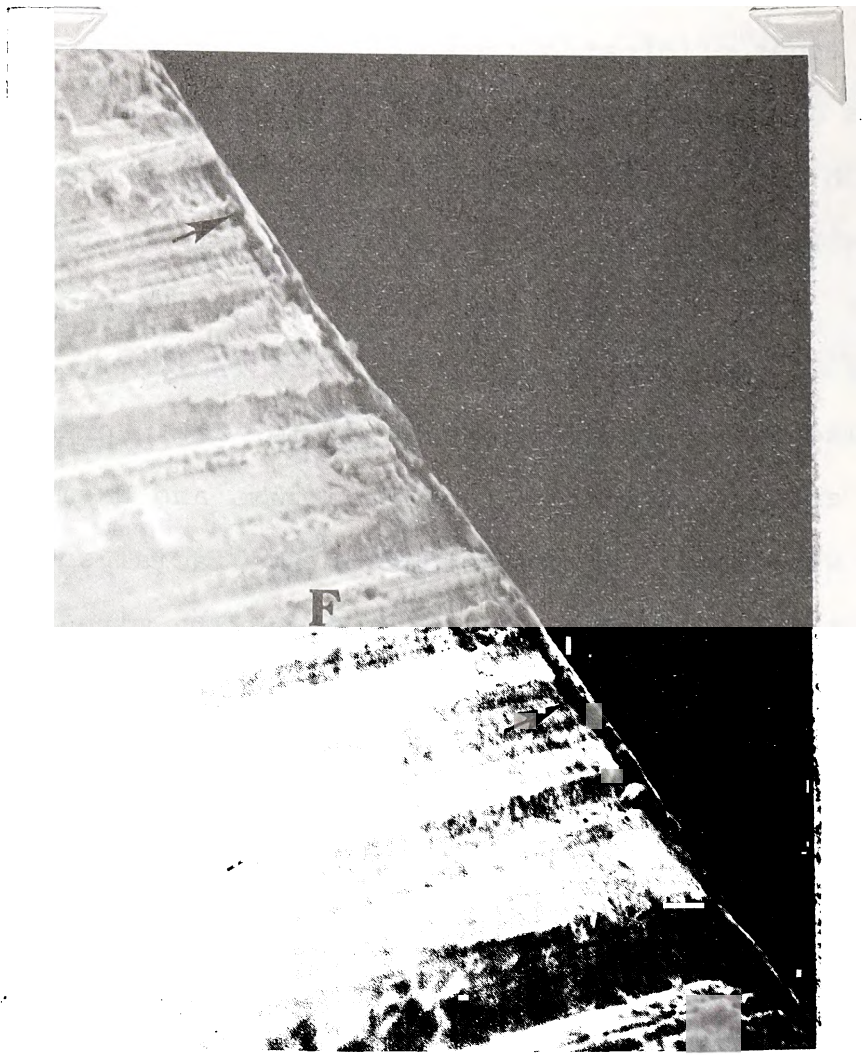

Figure 1B. Factory sharpened scaler. View from the facial surface (F). Arrows indicate wire edge extending from the facial surface. 500x.

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Table 1

Cutting Edge Observations

Grinding Technique	No. of Scalers Observed		Presence of Wire Edges		Origin of Wire Edge				Exact Meeting	
					Facial		Lateral			
	JB	ML	JB	ML	JB	ML	JB	ML	JB	ML
Factory	20	20	20	19	20	19	0	0	0	0
Machine	15	15	14	13	0	0	14	13	1	2
Hand	5	5	1	0	0	0	1	0	4	5
Machine Definned	14	13	0	0	0	0	0	0	14	13

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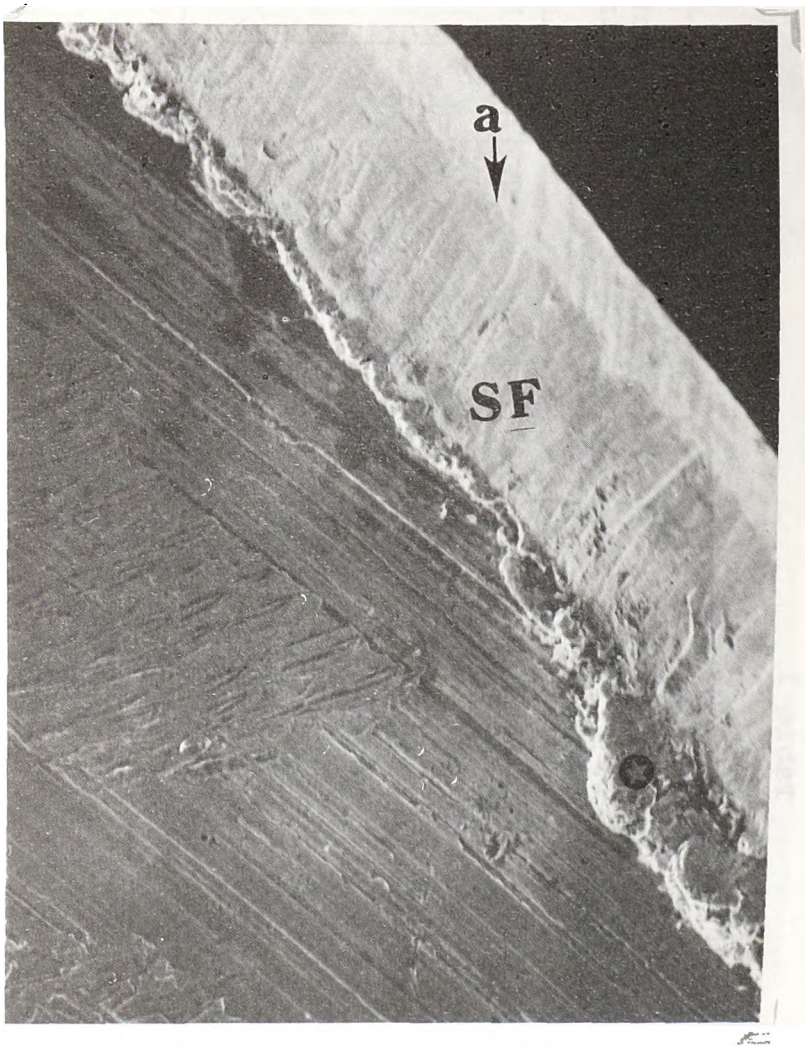

Figure 2. Scaler ground by an Arkansas stone. Star marks non-functional wire edge extending from the facial surface. "a" is the facial metallic deformity. Surface finish (SF) is smoother than the factory surface. Arrow indicates cutting stroke. 500x.

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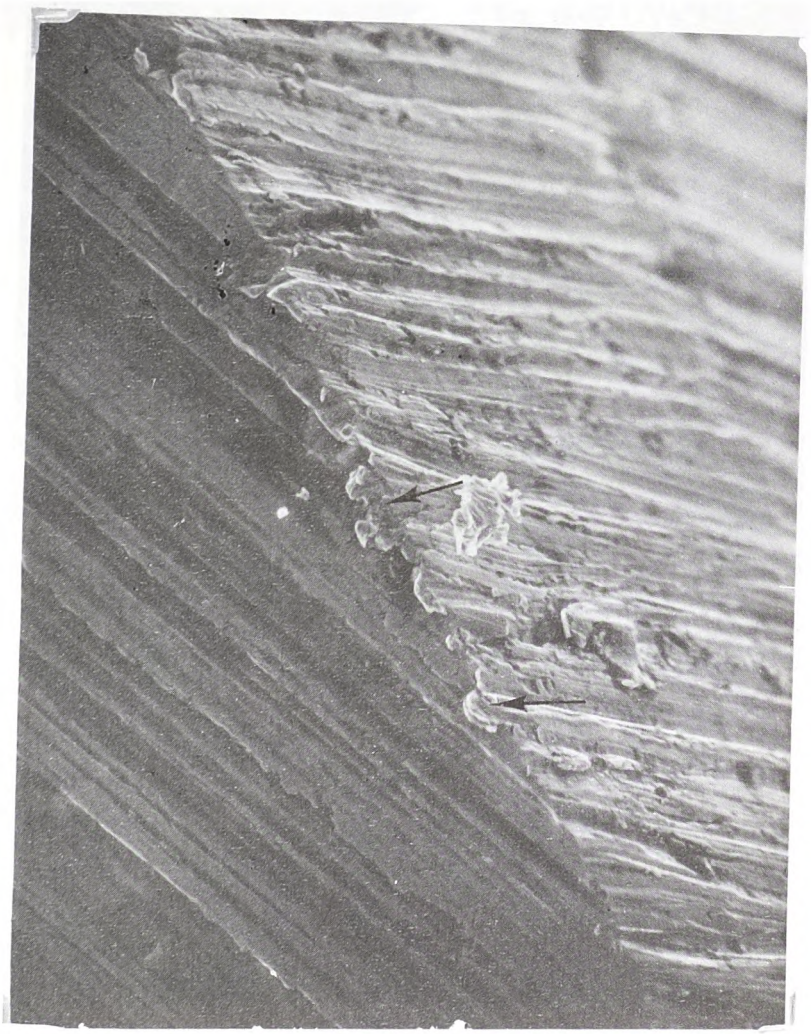

Figure 3. Scaler ground on the facial surface by a diamond stone. The wire edge formed is minimal. 500x.

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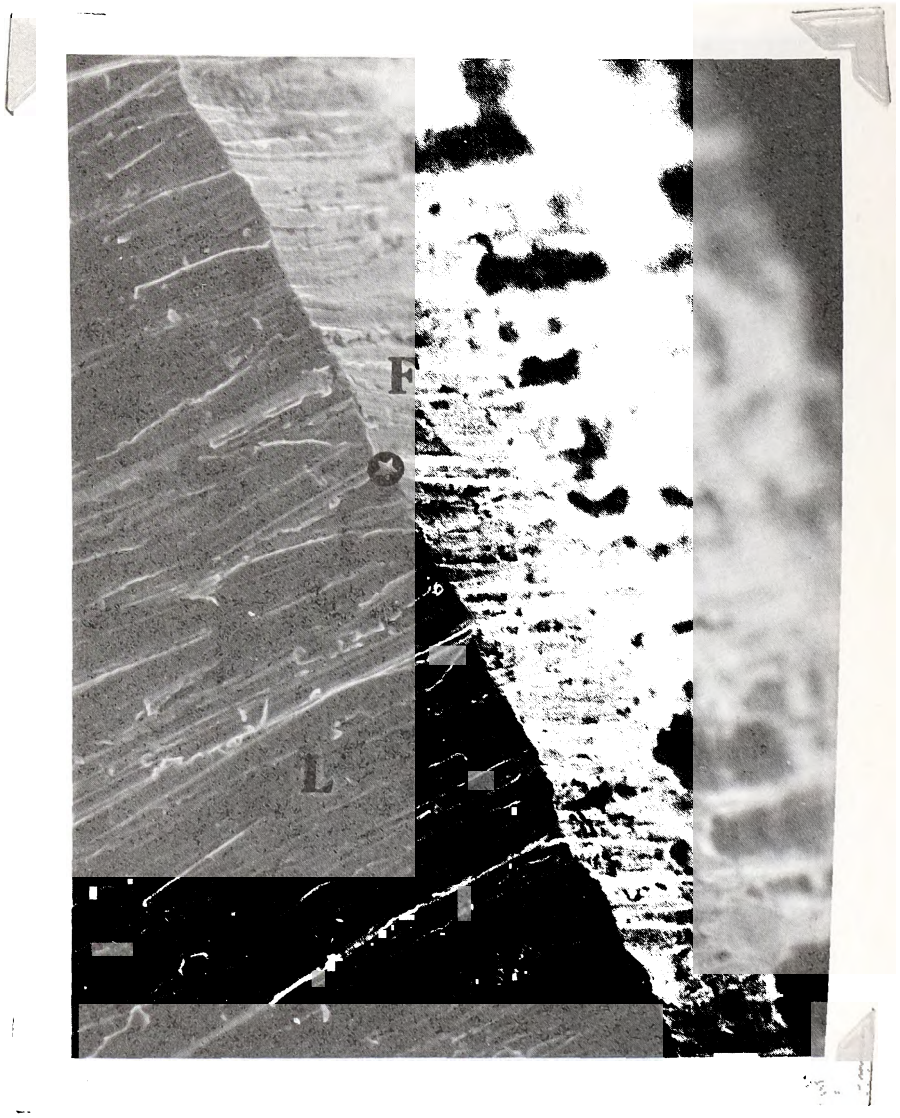

Figure 4. Definned (honed) scaler. The wire edge was removed. The result is an exact meeting of the facial (F) and lateral (L) surfaces. The star illustrates the definned cutting edge. 500x.

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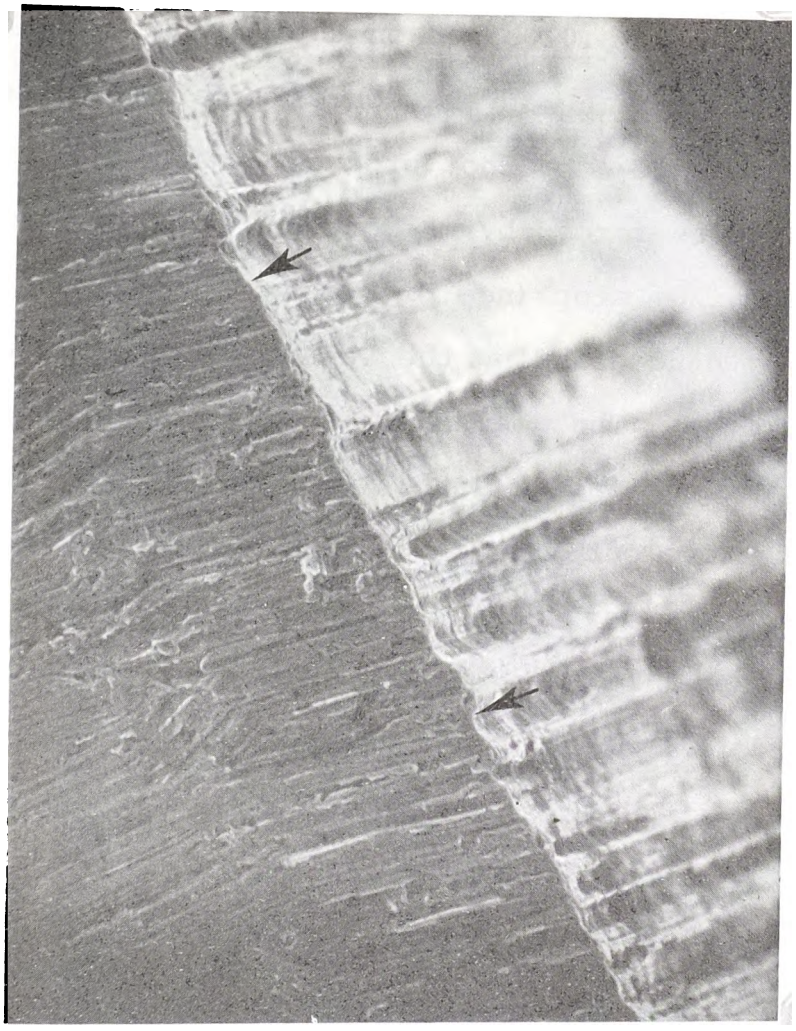

Figure 5. Arrows illustrate the exact meeting of the facial and lateral surfaces. The scaler was ground by hand on the lateral surface. The grinding was away from the cutting edge. 500x.

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5 out of 5 scalers (Examiner M.L). When the hand ground and machine ground cutting edges were analyzed in reference to wire edge formation, there was a statistically significant difference at $p < .01$ based on the Fischer's Exact Test ($df = 1$). Thus, grinding on the scaler's lateral side away from the cutting edges does not produce a wire edge.

The wire edges formed by machine grinding necessitated a definning (honing) step (see Figure 5). Definning was accomplished by hand grinding the lateral surface away from the facial surface. The goal of this step was to produce an exact meeting of the facial and lateral surfaces, thus producing an effective cutting edge. Both examiners were asked to characterize the definned scalers in reference to the presence of absence of wire edges. When data comparing undefinned and definned machine sharpened scalers was analyzed by chi-square with a Yates' correction ($df = 1$), there was a statistically significant difference at $p < .001$. Therefore, definning was an essential step in order to remove the wire edges and produce an exact meeting of the facial and lateral surfaces. The result is the re-establishment of an effective cutting edge.

In observing dullness, both examiners noted significant metallic alterations of the former cutting edge. Apparently, there was metallic loss of the factory wire edge and deformation of the underlying metal (see Figure 6). The wear can be classified as edge deformation.[8] Electron-probing of the dull

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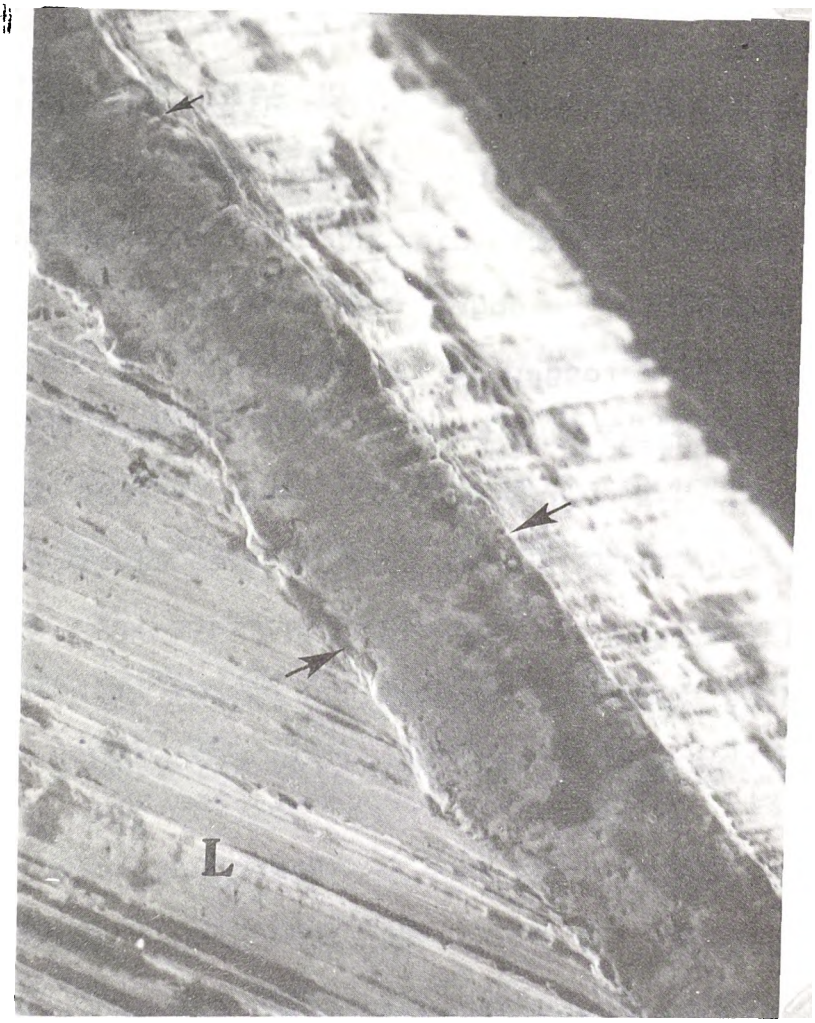

Figure 6. Dull instrument illustrating the edge deformation. "L" is the scaler's lateral surface. Area between arrows shows the deformation of the cutting edge onto the lateral surface. 500x.

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area indicates a metallic composition similar to the scaler. Calcium counts of the dull area showed an insignificant amount of biological material, indicating that the metallically deformed area was indeed metallic and not an accumulation of tooth structure.

Machine grinding stones produced different degrees of finning. Arkansas stones produced moderate to severe wire edges (see Figure 2), while diamond and coarse stones formed minimal to moderate wire edges (see Figure 3).

Facial surface roughness was dependent on the stone's grit.[10] The Arkansas stone (grit 500) produced the smoothest resharpened surface (see Figure 2). The factory ground scalers exhibited a corrugated facial surface (see Figure 1). Arkansas grinding transformed the corrugated surface into a smooth, non-corrugated surface. Diamond and coarse grinding stones removed the corrugation but left irregular gouges (see Figure 3).

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CHAPTER V

DISCUSSION

The factory sharpened scaler exhibited a wire edge which both investigators noted to be a metallic extension of the lateral surface. This type of wire edge was due to the factory's sharpening technique. The lateral surface was ground last and the direction of grinding was toward the cutting edge (see Figures 1A and 1B). This wire edge can be prevented by grinding away (downward) from the cutting edge, or by honing, i.e., lightly grinding the facial surface.[4] This would result in an exact meeting of the facial and lateral surfaces.

> Grain size has a considerable effect on surface roughness. The ratio of the average surface roughness values was approximately equal to the inverse of the mesh number of the abrasive grains.[9]

The factory scalers appeared to have two distinct types of surface roughness. Examiner J.B. noted all lateral surfaces were smoother than the facial surfaces. The factory states:

> The #3 Morse scaler point is ground first on the inside with a 220 grit aluminum oxide wheel. The sides are then ground on a 150 grit silicone carbide belt. All grinding operations are rotary.[10]

One would expect the facial surface to be smoother than the lateral surfaces. However, the examiners noted the opposite.

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The probable reason is that the facial surface was ground by a wheel and the lateral by a belt. The wheel did not give; therefore, more pressure could be placed on the facial surface. The belt was pliable and a fine grit belt would produce a smoother surface.[10]

During the dulling procedure two factors were not considered. One is a rank angle which is clinically not constant. No attempt was made to standardize the rank angle.[11] Second, a fretting process occurs when two components are in contact in a material system. In scaling, the two components are tooth debris and intact cementum. It is not known to what degree fretting affects the cutting edge.[12] The factory cutting edge was a wire edge. Dullness was due to deformation of this wire edge which was physically bent over, with the underlying metallic structure deformed as dullness increased (see Figure 6).

Machine grinding was accomplished in a reciprocal motion on the scaler's facial surface, resulting in a wire edge extending from the facial surface. This type of wire edge was statistically significant at $p < .001$. A facial metallic deformity was noted in four scalers (see Figure 2). The deformity was probably due to two factors: 1) that the scaler was not stabilized against the grinding stone which would result in an uneven grinding; 2) the stone itself was not stabilized at its base, in which case the stone would wobble and cause the facial metal defect.

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Fifteen scalers were ground by three different stone grits. The Arkansas stone with a 500 grit produced the smoothest facial surface (see Figure 2). This would agree with Zohdi that the grain size has considerable effect on the surface roughness.[9] The three stones produced different degrees of wire edges. Arkansas stones produced moderate to excessive fins (wire edges). Coarse stones produced minimal to moderate fins and diamond stones produced minimal fins.

Five scalers were ground on the lateral surface away from the cutting edge. The average number of strokes was 17.2, which is probably the maximum average. A smaller number of strokes might have produced the same cutting edge. Both examiners observed an exact meeting of the lateral and facial surface. Downward grinding by hand on the scalers' lateral surface produced an effective cutting edge (see Figure 5).

From the SEM prints, a scaler wire edge can be defined as an unsupported metallic extension from one of the surfaces forming the cutting edge (see Figures 1A and 1B). Additionally, a wire edge can be classified as functional or non-functional. The factory cutting edge is a functional wire edge, i.e., a metallic projection that is structurally and directionally capable of removing tooth accretion. The functional wire edge must extend in the same direction as the cutting stroke (see Figures 1A and 1B). The machine ground scaler produced

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a non-functional cutting edge. This is a metallic projection(s) that probably cannot remove tooth accretions due to the direction of the extension of the wire edge. The wire edge is not in alignment with the cutting stroke (see Figures 2 and 3).

This experiment re-emphasized the necessity of honing, which should be the last step in the scaler sharpening procedure. The goal of the honing step is to remove wire edges, resulting in a scaler cutting edge which is formed by an exact meeting of the facial and lateral surfaces (see Figure 4). Honing away from the cutting edge produced an effective cutting edge. The number of honing strokes varies. In this experiment, the average number of strokes was 9.2, although fewer honing strokes might have produced the same result.

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CHAPTER VI

CONCLUSION

The factory sharpened scalers exhibited fins which were metallic extensions from the lateral surface. These extensions (fins) were due to two factors. One, the lateral surface was the last surface to be ground and the grinding direction was toward the cutting edge. Two, honing was not employed in order to remove the fins. The result is a continuous fin (wire edge) extending from the lateral surface. These can be considered functional wire edges. The hand grinding technique was against the scaler's lateral surface. The grinding direction was away from the cutting edge. The result was an exact meeting of the scaler's facial and lateral surface. This produced no evidence of fins. The machine grinding technique was a reciprocal motion against the scaler's facial surface. The result was a metallic extension (fin) from the facial surface. A honing step was performed on the scaler's lateral surface away from the cutting edge. The final outcome was an exact meeting of the scaler's facial and lateral surface. Thus, honing is a necessary step in the sharpening technique.

From scanning electron microscope photographs, scaler wire edges can be classified as functional and non-functional. A functional wire edge is a continuous metallic projection

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in the same direction of the cutting stroke. A functional wire edge can remove tooth accretions. A non-functional scaler wire edge is a metallic projection not in the direction of the cutting stroke. This wire edge cannot remove tooth accretions. Finally, an examination of scanning electron microscope prints permits the definition of a cutting edge as an exact meeting of the facial and lateral surfaces.

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CHAPTER VII

SUMMARY

SEM photographs were taken of 20 scalers in factory sharpened state, dulled, and re-sharpened state. Fifteen scalers were re-sharpened by R_x Honing Machine, 5 each on Arkansas, diamond, and coarse stones. Five were hand sharpened on an Arkansas stone. A double blind study of the SEM prints revealed that factory edges are functional wire edges; machine sharpening produces non-functional wire edges; hand sharpening produces an exact meeting of lateral and facial surfaces. A honing step performed on machine resharpened edges removed the wire edges and produced an effective cutting edge.

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BIBLIOGRAPHY

1. Johari, Om. Scanning Electron Microscopy/1968 Proceedings of the Symposium--The Scanning Electron Microscope: The Instrument and its Application, p. 86. April 30-May 1, 1968. ITT Research Institute, Chicago, Ill.

2. Foss, C. L., and Orban, T. R. Sharpening Periodontal Instruments, Journal of Periodontology, 27:135, 1956.

3. Wilkins, E. M. Clinical Practice of the Dental Hygienist. 3rd ed. Lea & Febiger, Philadelphia, p. 186, 1971.

4. Steele, P. F. Dimensions of Dental Hygiene. Lea & Febiger, Philadelphia, p. 211., 1966.

5. Glickman, I. Clinical Periodontology. 2nd ed. W. B. Saunders, Philadelphia, p. 612, 1972.

6. Swenson, H. M. Sharpening of prophylactic instruments. Journal of Am. Dental Hygienists Assoc. 31:6-10, 1968.

7. Ransom and Randolph Co., P.O. Box 905, Toledo, Ohio 43691. Letter to author, December 20, 1974.

8. Lindhe, J., and Jacobson, L. Evaluation of Periodontal Scalers I: Wear following clinical use. Odontologisk Revy. 17:1-8, 1966.

9. Zohdi, M. E. Statistical Analysis, Estimation and Optimization of Surface Finish in the Grinding Process. Journal of Engineering for Industry, 96:119-122, February 1974.

10. Ransom and Randolph Co., P. O. Box 905, Toledo, Ohio 43691. Personal communication, February 18, 1974.

11. Lindhe, J. Evaluating periodontal scalers, II: Wear following standardized orthogonal cutting. Odontologisk Revy., 17:1-8, 1966.

12. Gross, G. L., and Howppner, D. W. Characterization of Fretting Fatigue Damage by SEM Analysis. Wear, 24:77-95, 1973.

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SJOGREN'S SYNDROME:

A REVIEW

by

THOMAS D. TAYLOR, CAPTAIN, DENTAL CORPS, USA

Professional paper submitted in partial fulfillment of the requirements of the U.S. Army Dental Residency Program, Walter Reed General Hospital, Walter Reed Army Medical Center, Washington D.C. 20012

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Chapter I
History

The generalized symptom complex known as Sjogren's syndrome has grown out of clinical research by many individuals. The entity as we know it today, including the primary symptoms of xerostomia, xerophthalmia and enlargement of salivary and lacrimal glands, was not considered as one disease entity until as late as 1953.

In 1882 Leber described an ocular disorder which he described as filamentary keratitis. In 1888 Mikulicz reported a patient whose chief complaint was chronic lacrimal gland enlargement followed by submandibular and parotid gland enlargement. Surgical removal of the lacrimal and submandibular glands followed by microscopic examination revealed massive replacement of acinar parenchyma by lymphocytes with fibrotic degeneration of the glandular tissue.

In 1889, Fischer mentioned the association of arthritis with filamentary keratitis for the first time. Houwer's examination of ten patients with filamentary keratitis in 1927 revealed arthritis in six.

In 1919 Fuchs associated deficient tear secretion with filamentary keratitis 16 years after Schirmer described the physiology of tear flow. In 1927 Schaffer and Jacobsen classified patients with parotid gland enlargement into two groups.

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The first was called "Mikulicz' disease" and was considered an entity itself characterized by lacrimal and salivary gland enlargement. "Mikulicz' syndrome" was reserved for those patients in whom clinical symptoms of the disease were secondary to a variety of other diseases such as tuberculosis and sarcoidosis.

In 1933 Henrik Sjogren, a Swedish ophthalmologist, described thirteen women with filamentary keratitis which he called "Keratoconjunctivitis sicca" associated with arthritis. The frequency with which these disease processes occurred together convinced Sjogren that this combination of symptoms constituted a specific disease entity. Sjogren's syndrome was defined as the occurrance of keratoconjunctivitis sicca, xerostomia, and arthritis together.

Morgan and Castleman were the first to recognize the relationship between Sjogren's syndrome and Mikulicz's disease. Both glandular dysfunction and enlargement showed the same histological picture and were merely different manifestations of the same disease process. The use of the term "Mikulicz' disease" has since been replaced by the term "Sjogren's syndrome" to describe the disease complex.

Since 1933, mainly by clinical observation, Sjogren's syndrome has been associated often with other disease processes mainly of connective tissue systems. In some patients Sjogren's syndrome is the primary disease process while in others it is merely a complication of a more severe systemic disease.

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It seems appropriate at this time to define certain terms which are used frequently in the liturature.

Mikulicz' disease: An entity of unknown etiology characterized by salivary and lacrimal gland enlargement and mononuclear cell infiltration.

Mikulicz' syndrome: Patients with clinical manifestations of Mikulicz' disease secondary to a variety of diseases such as tuberculosis and sarcoidosis. Neither this term or Mikulicz' disease is used with much frequency today. They have been replaced by "Sjogren's syndrome" or the name of the specific process under question.

Filamentary Keratitis: Keratitis of the cornea characterized by twisted filaments of mucoid material on the surface of the cornea associated with and as a result of decreased lacrimal flow, and at times corneal ulceration.

Keratoconjunctivitis sicca: Filamentary keratitis as renamed by Sjogren in 1933.

Sjogren's syndrome: "A chronic systemic disorder manifested by dryness of the eyes (xerophthalmia), nose, mouth (xerostomia), pharynx and vagina often in association with a rheumatic disease."[1]

The major features that permit diagnosis of Sjogren's syndrome may be considered under categories of definite and probable.

Definite: The presence of either (1) objective evidence of keratoconjunctivitis sicca, or (2) characterized histological features in lacrimal or salivary gland tissue.

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Probable: The presence of two out of the following three features. (1) recurrent or chronic "idiopathic" salivary or lacrimal gland swelling, (2) unexplained xerostomia, or (3) connective tissue disease.[2]

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Chapter II
Epidemiology

Many workers have described Sjogren's syndrome as an uncommon disease. However, the frequency with which Sjogren's syndrome is diagnosed depends in large measure on the awareness of the practitioner initially examining patients. It is quite possible that many patients with diagnostic signs of Sjogren's syndrome are never recognized because no treatment is sought or an incorrect diagnosis is made.

It is difficult to establish the age of onset of a chronic disease process in which symptoms are frequently very mild. The highest incidence of Sjogren's syndrome is most frequently described as between 40 and 60 years of age.

The occurance of Sjogren's syndrome in the general population has been calculated by many workers using varied criteria and methods of diagnosis and classification. Approximately 3% of individuals in the general population have diagnosed rheumatoid arthritis. Assuming from other studies that 14.2% of rheumatics exhibit symptoms of Sjogren's, we could expect to see Sjogren's syndrome manifestations in one out of every 525 people. If we also recognize that only 30% of patients with Sjogren's syndrome also have rheumatoid arthritis, the incidence of Sjogren's syndrome in the general population could be even higher. Seifert and Gecter studied the parotid glands of 900 unselected sequential autopsies and found four with classical histological findings of Sjogren's syndrome. This gives a frequency of 0.44% occurance

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in the general population.

The ratio of women over men with Sjogren's syndrome has been reported frequently by many researchers as approximately nine to one. Although the age of onset in women corresponds with the general age of onset of menopause, there is no evidence of a relationship between the two.

There is no documented familial occurrance of Sjogren's syndrome but some have reported a possible familial predisposition to the developement of immunological disorders, one of which is probably Sjogren's syndrome.

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Chapter III

Etiology

Mikulicz and others felt that the enlargement of lacrimal and salivary glands, and the histological picture of round cell infiltration, represented a chronic infectious process involved in Mikulicz'disease and Sjogren's syndrome.

In the 1960's, Bumin, Anderson, and others reported elevated serum IgG, IgM, and IgA levels in Sjogren's syndrome patients consistent with findings seen in patients with nonrelated or nonspecific auto-immune disease processes. Increased frequency of organ specific and system specific antibodies such as those found in renal tubular dysfunction, Hashimoto's thyroiditis, and systemic lupus erythematosis have also been reported in Sjogren's syndrome. Although a common occurrance in Sjogren's patients, specific autoimmune antibodies or consistent levels of serum globulins have not yet been recognized as diagnostic or even typical of Sjogren's syndrome.

In 1970 Shearn, Tu, Stephens, and Lee reported virus like structures in patients with Sjogren's syndrome.[12] These structures were found in renal biopsy specimens of four patients. Similar particles have been recognized frequently in patients with a variety of autoimmune diseases such as systemic lupus erythematosis. In fact, renal biopsy is used routinely as a diagnostic aid in lupus patients. This is consistent with the occurrance of

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Sjogren's syndrome and systemic lupus erythematosis simultaneously in many patients, however the four patients biopsied in Shearn's study did not exhibit signs or symptoms of systemic lupus erythematosis.. Although this is weak evidence for a viral etiology of Sjogren's syndrome, it is intriguing to speculate on the subject of possible viral etiology or involvement in Sjogren's syndrome and many other autoimmune diseases.

Since the disease was first described, Sjogren's syndrome has been considered an infectious disease, an autoimmune disease, and now again, an infectious or at least a microbial disease.

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Chapter IV

Diagnosis

The diagnosis of Sjogren's syndrome has been defined by Shearn as falling into two categories, definite and probable. (see definitions pp.3+4).

Diagnosis of keratoconjunctivitis sicca can be made by recording and analyzing patient symptoms, the Schirmer strip test, and by rose bengal staining.

Ocular symptoms include foriegn body sensation, burning, excess secretion, inability to tear, redness, photosensitivity, "film", itching and difficulty in lid opening. The Schirmer test involves the use of a strip of Whatman No. 41 filter paper 5x35mm which is folded at a right angle 5mm from one end and inserted into the conjuctival sac at the outer third of the lower lid. After five minutes the strips are removed and the length of moistened paper is measured. Less than 15mm of wetting is considered abnormal.

The rose bengal staining test involves the placing of one drop of 1% rose-bengal dye in the conjuctival sac and immediately rinsing with saline. Gross examination of the staining of the conjuctiva and/or cornea is considered abnormal and signifies irritated or necrotic tissue present on the eye surface.

The presence of symptoms and/or positive test results as

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described above are considered diagnostic for keratoconjunctivitis sicca.

Diagnosis of xerostomia or oral and salivary gland manifestations can be made by determining a history of xerostomia or parotid or submandibular gland enlargement, examination of the oral cavity and palpation of the salivary glands, quantitative estimation of parotid and total salivary flow, sialography, sequential salivary scintigraphy, and biopsy of major and minor salivary glands.

Complaints of dryness of the mouth and lips or an awareness of insufficient salivary flow is by definition xerostomia. However, many researchers have pointed out that many patients do not complain about dry mouth and throat even when their salivary flow appears greatly diminished. The presence of fissuring or ulceration of the lips and mouth and ulceration of labial commissures is at times indicative but by no means diagnostic of decreased salivary flow. Clinical enlargement of the major salivary glands is a typical but not a frequent finding in Sjogren's syndrome. The fact that salivary gland enlargement is not a frequent finding in Sjogren's syndrome has contributed to the continued usage of the terms Mikulicz' disease and Mikulicz' syndrome.

Salivary flow rate determinations are useful but inconsistent methods of determining normal or abnormal salivary flow. The

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measuring of parotid flow by means of Lashley cups placed over the orifices of the duct followed by lemon juice stimulation, and having the patient chew paraffin for three minutes while collecting all saliva produced, are two methods commonly used for determination of salivary flow rates.

Sialography is useful in determining the size, presence of pathology, and extent of destruction of major salivary glands. Common findings in patientswith Sjogren's syndrome are punctate sialectasis, globular sialectasis, and large areas of cavitary destructive sialectasis.

Salivary gland immunofluoresence and sequential salivary scintigraphy are relatively recent diagnostic aids being employed on patients suspected of having Sjogren's syndrome. Sequential salivary scintigraphy is a method used for observing the relative rates of uptake, accumulation, and discharge of ^{99m}Tc pertechnatate by major salivary glands. The procedure involves placing the patient in the supine position with his/her head immobilized on the crystal of a gamma scintillation camera and administering an I.V. dose of 10 millicuries of the isotope. Sequential photos are made at varying intervals for a period of an hour and reveal the relative concentration of the isotope in the glands during this period.

Biopsy of major salivary glands for histological examination

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is an involved and potentially hazardous procedure and should be reserved for those instances in which a definite diagnosis cannot be made by minor salivary gland biopsy or there is some question as to the form of lymphoproliferation present.

Minor salivary gland biopsy is a quick, simple, and relatively painless procedure which yields excellent material for examination. The procedure involves making a shallow 1cm incision in the labial mucosa of the lower lip usually one or two centimeters inside the lip. This is followed by blunt dissection of the margins which releases the glands toward the exterior. These may then be snipped off and the incision closed with 4-0 silk sutures. The yield is usually between 3-6 glands which is more than adequate for histological examination.

The histology of Sjogren's syndrome in the minor salivary glands can be described as ranging from mild perivascular and periductular plasma cell and lymphocyte infiltration to massive lymphocytic infiltration and replacement of acinar tissue by fibrous tissue. As the severity of the disease process increases there seems to be a relative decrease in the number of plasma cells present and a marked increase in the number of lymphocytes associated with destruction of glandular (acinar) tissue and apparent sparing of ductular epithelium.

Patients with Sjogren's syndrome also exhibit inconsistently

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high levels of serum globulins, anti-rheumatoid antibodies, anti-nuclear antibodies, anti-salivary duct antibodies and many other factors found in patients with autoimmune type disorders.

Sjogren's patients also exhibit a high frequency of drug sensitivity reactions to various medications, the most common being penicillin.

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Chapter V

Treatment

For many years the treatment for the various manifestations of Sjogren's syndrome has been mainly of a symptomatic nature. Recently, however, Sjogren's therapy has expanded to include more agressive modes of treatment, such as corticosteroids, immunosuppressants, and even cytotoxic agents. The reason for this change in treatment has been the gradual realization that Sjogren's syndrome is not the benign, mild disease as was previously thought. Better case documentation and follow-up has illustrated that Sjogren's patients exhibit a high frequency of coexisting connective tissue disorders and frequent malignant degeneration. Palliative treatment of symptoms is still an important aspect of total therapy, however, long term treatment with associated alleviation of symptoms has been realized with the use of the previously mentioned systemic drugs.

The ocular symptoms of Sjogren's syndrome can be treated by the use of false tears, protective glasses, and cautery of the nasolacrimal duct. The discomfort associated with rheumatoid arthritis is best treated with salicylates. Oral symptoms and resultant complications of Sjogren's syndrome may be treated by methyl cellulose rinses, salivary stimulants, fluoride trays, and close dental supervision to intercept and treat recurrent and root surface caries frequently seen in patients with decreased salivary flow.

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Chapter VI

Summary

In summary, Sjogren's syndrome is a complex of signs and symptoms which in itself is a relatively minor disease entity. When viewed from a systemic basis Sjogren's syndrome is merely one of many diseases which are interrelated and may be life threatening. (Fig. 1)

Simultaneous occurrance of rheumatoid arthritis, systemic lupus erythematosis, polymyositis, and many other connective tissue disorders with Sjogren's syndrome can only serve to illustrate the necessity of complete examination and workup by the practitioner of any patient with symptoms of Sjogren's.

The striking feature of Sjogren's syndrome is its tendency for destruction and/or degeneration of organs of ectodermal origin(salivary, lacrimal, stomach, and sweat glands). Other autoimmune diseases associated with Sjogren's syndrome seem to attack connective tissue(mesoderm) almost exclusively.

The most significant aspect of Sjogren's syndrome for the dentist must be the realization that any patient exhibiting the symptoms of xerostomia, xerophthalmia, or salivary gland enlargement must be screened for the possibility of a coexisting disease process of a much more serious nature.

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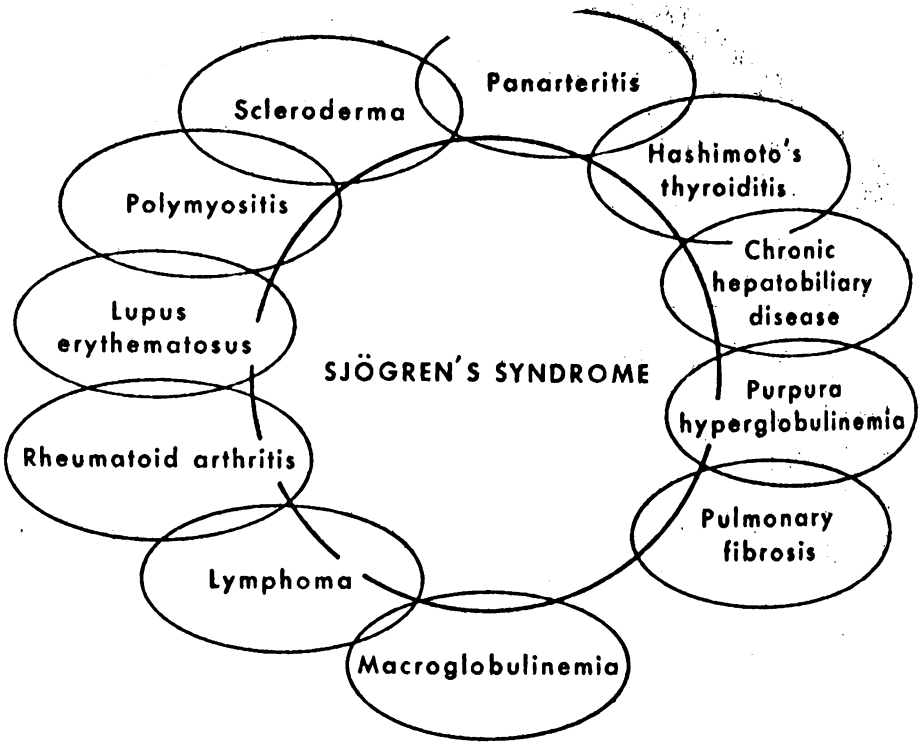

FIGURE 1. The spectrum of autoimmune diseases (by no means complete) associated with Sjogren's syndrome.

Taken from Shearn, Martin.: Sjogren's syndrome. Seminars in Arthritis and Rheumatism 2:165-190 1972.

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BIBLIOGRAPHY

1. Shearn, M.: Sjogren's syndrome. Seminars in Arthritis and Rheumatism 2: 165-190 1972.
2. Mason, A.: Sjogren's syndrome-A clinical review. Seminars in Arthritis and Rheumatism 2: 301-331 1973.
3. Fogel, D.: Sjogren's syndrome with benign purpura hyperglobulinemia. New England Journal of Medicine 261:81-83 1959.
4. Bloch, K.: Buchanan, W., Wohl, M., and Bunim, J.: Sjogren's syndrome: A clinical, pathological, and serological study of sixty two cases. Medicine 44: 187-231 1965.
5. Schall, G., Et. al: Xerostomia in Sjogren's syndrome. JAMA 216: 2109-2116 1971.
6. Kaltreider, H., and Talal, N.: The neuropathy of Sjogren's syndrome: Trigeminal nerve involvement. Annals of Internal Medicine 70: 751-762 1969.
7. Anderson, L. and Talal, N.: The spectrum of benign to malignamt lymphoproliferation in Sjogren's syndrome. Clinical and Experimental Immunology 9: 199-221 1971.
8. Talal, N., Sokoloff, L., and Barth, W.: Extra salivary lymphoid abnormalities in Sjogren's syndrome, reticulum cell sarcoma, "pseudolymphoma," macroglobulinemia. American Journal of Medicine 43: 50-65 1967.
9. Tu, W., Et. al: Interstitial nephritis in Sjogren's syndrome. Annals of Internal Medicine 69: 1163-1170 1968.
10. Whaley, K., Et. al: Sjogren's syndrome I; sicca components. Quarterly Journal of Medicine 42: 279-304 1973.
11. Whaley, K., Et. al: Sjogren's syndrome II; Clinical associations and immunological phenomena. Quarterly Journal of Medicine 42: 513-548 1973.
12. Shearn, M., Et. al: Virus-like structures in Sjogren's syndrome. Cl Lancet: 568-569 1970.
13. Akin, R., Et. al: Sjogren's syndrome. Journal of Oral Surgery 33: 27-33 1975.
14. Anderson, L., Et. al: Cellular versus humoral autoimmune responses to salivary gland in Sjogren's syndrome. Clinical and Experimental Immunology 13: 335-342 1973.
15. Cummings, N., Et. al: Sjogren's syndrome; newer aspects of research, diagnosis, and therapy. Annals of Internal Medicine 75: 937-950 1971.
16. Talal, N., Et. al: Tand B lymphocytes in peripheral blood and tissue lesions in Sjogren's syndrome. Journal of Clinical Investigation 53: 180-189 1974.

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17. Steinberg, A., Green, W., and Talal, N.: Thrombotic thrombocytopenia purpura complicating Sjogren's syndrome. JAMA 215: 757-761 1971.
18. Greenspan, J., Et. al: The histopathology of Sjogren's syndrome in labial salivary gland biopsy. Oral Surg. Oral Med. Oral Path. 37: 217-229 1974.
19. Ericson, S.; The prevalence of hyposalivation in rheumatoid arthritis and its relation to sialographic appearance of the parotid glands. Oral Surg. Oral Med. Oral Path. 38: 315-331 1974.
2o. Leban, S., and Stratigos, G.: Benign lymphoepithelial sialadenopathies, The Mikulicz/Sjogren controversy. Oral Surg. Oral Med. Oral Path. 38: 735-748 1974.
21. Chisolm, D. and Mason, D.: Labial Salivary Gland Biopsy in Sjogren's syndrome. Journal of Clinical Pathology 21: 656-660 1968.

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