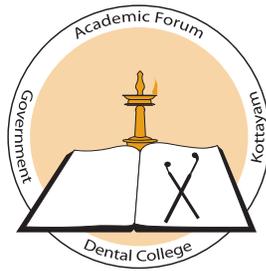


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Govt. Dental College
Kottayam



Journal of Clinical Dentistry

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Contents

Editorial	4
Effect of povidone-iodine mouth rinsing on post-scaling bacteraemia - A microbiological study Amitha Ramesh, Vidya Jayasheela, Biju Thomas, Veena Shetty	5
Rehabilitation of a partially edentulous patient with cleft lip and cleft palate Indu Raj	8
Lasers in endodontics Shiji Dinakaran	10
Chronic desquamative gingivitis - A review Raseena Beevi N.	15
Reverse hybridisation - The brand new bond Shibu Aman	19
Nuclear medicine- through times Asish R., Anita Balan	22
The concept of working width- The forgotten dimension Sheena P., Sam Joseph V G	27
Temporary anchorage devices in orthodontics Elbe Peter, Baiju RM, Joby Peter	31
Multiple Angiomas of oral cavity - A Review and report of a case L. S. Sreela, Sudheesh M, George Varghese	37
Photodynamic therapy (pdf) - A review Sam Joseph V G, Raseena Beevi N., Sheena P	40
Dental Management of a Patient with Cleidocranial Dysplasia- A Case Report. Supriya S., Kavita Rai, Amitha M. Hegde	43
Management of trismus using Trismus Appliance: A Case Report Sandhya Gopalakrishnan	46

The Academic Forum of Govt. Dental College, Kottayam – An Overview

The Dental College Kottayam started functioning from the year 2002. An Academic forum was constituted in the year 2003, with the aim of creating a platform for exchange of knowledge and to impart quality dental education comparable to international standards.

This report from the Chairperson's desk is penned with immense joy, as a long cherished dream is being materialized.

In the year 2003, Dr Baiju R.M and Dr Harikumar K., - two of our young enthusiastic teachers took the responsibility of nurturing the infant 'Academic Forum'. They started with fortnightly meetings, when topics of interdisciplinary interest were presented and discussed. In the year 2005, they succeeded in organizing a grand CDE program 'Gurukulam' which was targeted for the undergraduate students. The veteran gurus like Dr BRR Varma, Dr Varghese Mani, Dr K. Chandrasekharan Nair and Dr K. Nandakumar enlightened their academic grand children.

The academic forum was reconstituted in the following year with Dr L S Sreela as the chairperson and Dr Elbe Peter as the treasurer. Regular monthly meetings were held with the final year BDS students presenting interesting topics from every department, thus encouraging the students to learn how to prepare short presentations and improve their preparation skills.

In the year 2009, a series of CDE programs were held – the first of the series was on "Practice management and medico legal implications in Dentistry" (15 Feb. 2009). The participants were graduates and private practitioners in and around Kottayam. The program was well attended and was a source of inspiration to the organizers.

The next program "Minor Oral Surgical Procedures & surgical removal of impacted teeth" (22 mar 2009), was a venture from the OMFS department. There was such an enormous response that the number of participants for hands on exceeded our expectation.

The third program (29 mar 2009) on "Basic Life Support & Medical Emergencies in the Dental Office" was presented by the renowned team from MIMS, Calicut. The demo video CD was distributed to the participants.

Hands on CDE with surgical demonstration of the various types of Implant Placement for the faculty of GDC, Kottayam was held on 21 Nov 2009.

In March 2010, a program "Stress Management for Professionals" was organized jointly by our academic forum and the department of Psychiatry, MCH, Kottayam.

Here, we proudly present the first issue of our journal, which would not have materialized but for the blessings showered on us by the almighty and the untiring efforts of all our staff members in general and a few in particular whose names need special mention – Dr Baiju R M & Dr Sheena P the chief editor and Asst Editor. We take this opportunity to congratulate all those who have contributed the scientific articles and thank the sponsors for their timely financial help. Last but not the least all good things that happen in our campus are just the fruit of the toil by our mentor and guide Dr George Varghese, who is the soul of this institution.

Dr. Elbe Peter
Treasurer

Dr L.S. Sreela
Chairperson



MESSAGE

I am delighted to learn that under the editorial ship of Dr.R.M.Baiju a new journal ' Journal of Clinical Dentistry' is being published. This attempt will surely bridge the gap between academic interests and clinical relevance. Contribution and active participation from all concerned i.e.students, academicians and practitioners will aid sharing of their knowledge and this in turn decides the ultimate success of the journal. At the same time the editorial board has to painstakingly review and select the articles keeping in mind to guarantee a high standard of the journal so that in due time it will be an indexed journal. The right proportion in the nature of articles in the form of original articles, reviews, seminars and case report has to be maintained in all the forthcoming issues.

I wish the editorial board all success in the new endeavour.

May God bless you.

Dr. K. George Varghese
Principal-in-charge
Govt.Dental College, Kottayam
and
Dean, Faculty of Dentistry
University of Health Sciences Kerala

Principal's Message

Editorial

It is indeed satisfying to bring forth the first issue of Journal of Clinical Dentistry. In the era of evidence based decision making in medicine and dentistry, health related information should reach out to health care professionals across the globe.

Indian Dental Research has struggled due to the lack of publication avenues. But the scenario is changing. Recently a few of our journals have become indexed with Pubmed. There is a rise in the number of new publications offering platform for dental researchers and clinicians. Still we lack proper data of disease statistics. Epidemiological data of dental diseases is the corner stone for fundamental research in clinical dentistry.

The journal of clinical dentistry is a humble beginning towards the goal of igniting interest in young graduates and post graduates to pursue clinical research and scientific writing. I am sure, there will be a lot of shortcomings in the first issue and hope that the readers will bear with us. We also expect our well wishers to help us with constructive criticism.

I wish to acknowledge our principal and all the faculty members of Government Dental College, Kottayam for their advice and suggestions in bringing out this book.

'Dentistry is Interesting'



Dr. Baiju R.M.

Effect of Povidone-Iodine Mouth Rinsing on Post-Scaling Bacteraemia - A Microbiological Study

Abstract

Periodontal diseases are infections of periodontium, caused by bacteria. It is well established that transient bacteremia can result from periodontal treatment such as scaling.

Aims and objectives

- To assess the effect of povidone iodine mouth rinsing on the incidence of post-scaling bacteraemia.
- To investigate the magnitude and microbial profile of bacteremic isolates after scaling.

Sample size:

Total 60 subjects between the age group of 20 – 60 year were randomly selected and divided in to two groups. One with povidone iodine before ultra sonic scaling and other without povidone iodine before ultra sonic scaling; the sample was done. In this study bacterial isolates were recovered from the baseline blood sample of one subject and consisted of staphylococci.

conclusion

In this study we did not find bacteremia after ultrasonic scaling in subjects who did not rinse with povidone iodine prior scaling as well as in subjects who rinsed with povidone iodine before scaling.

Introduction

Periodontal diseases are infections of periodontium, caused by bacteria. It is well established that transient bacteremia can result from periodontal treatment such as scaling. A feature that is unique to the oral bacterial biofilm, particularly the subgingival plaque biofilm is, its close proximity to a highly vascularized milieu. Although innate defense by polymorphonuclear neutrophils is highly developed at the dentogingival junction and backed up by a highly organized lymphatic system, the oral biofilms, if left undisturbed, can establish themselves permanently on nonshedding tooth surfaces subjacent to the dentogingival junction. Under these circumstances, any disruption of the natural integrity between the biofilm and the subgingival epithelium, which is at most about 10 cell layers thick, could lead to a bacteremic state.⁶

While the occurrence of transient bacteraemia after dental procedure does not lead to any complications in healthy individuals, as the bacteria that gain access to the blood stream are generally rapidly removed by the reticulo-endothelial system. However in susceptible patients with acquired or congenital endocardial defects or cardiac prostheses, circulating bacteria may reach the defective endocardium and cause bacterial endocarditis. Although pretreatment antibiotic prophylaxis may decrease the risk of bacterial endocarditis, antibiotics cannot prevent transient bacteremia. Also, bacterial endocarditis can occur after dental treatment in patients who have received antibiotics. The recent guidelines by American Heart Association (AHA) recommend the use of topical antimicrobials such as povidone-iodine as an adjunct to systemic antibiotic cover to enhance the effect of current prophylactic measures. Mouth rinsing is considered preferable to subgingival irrigation as the latter has been shown to induce bacteremia and for this reason is not currently recommended by AHA.¹ Povidone iodine (POV-I) is widely used as topically applied antiseptic. It is an iodophor in which iodine is linked to povidone (polyvinylpyrrolidone). POV-I is microbicidal for gram-positive and gram-negative bacteria, fungi, mycobacteria, viruses and protozoans. This study is designed to assess the effectiveness of pretreatment mouth rinsing with povidone iodine in patients with plaque-induced gingivitis who are undergoing ultrasonic scaling.

Review of literature

1. A single blind parallel study of 2 week duration was conducted on 30 volunteers with untreated periodontal disease. Results showed that incidence of bacteraemia following ultrasonic scaling (13%), periodontal probing (20%) and tooth brushing (3%).³
2. A randomized placebo-controlled trial conducted on 60 patients with

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gingivitis. In which 30 rinsed with 0.9% saline and 30 with 7.5% povidone-iodine for 2min, before ultrasonic scaling of FDI teeth 31-35. Blood samples before and after scaling were cultured by lysocentrifugation. Oral bacteraemia occurred in 33.3% of the saline group and 10% of the povidone-iodine group.¹

3. A study was conducted on 120 patients to determine, whether irrigation of gingival sulcus with one of the two antiseptic solution i.e., povidone iodine and chlorhexidine would affect the incidence and type of bacteraemia after dental treatment. It was found that, bacteraemia in blood culture of 21 control subjects (52.5%), 11 povidone iodine subjects (27.5%) and 18 chlorhexidine subjects (45%). Streptococcus viridians were detected in 15 culture of control group and 4 povidone iodine group and 14 chlorhexidine group.²
4. A study was conducted to determine the ideal time span and concentration of povidone iodine that should be used as pre-procedural rinse. It was reported that the greatest decrease of streptococci was attained when povidone iodine was diluted 1/1, creating a 5% concentration and applied for 30seconds.⁴
5. A study determined the incidence of bacteraemia after single irrigation with 0.12% chlorhexidine gluconate mouth rinse as well as after subsequent scaling and root planing during the same visit. There was no significant difference between the incidence of bacteraemia associated with rinsing with irrigation by chlorhexidine or sterile water. There was also no significant difference in the incidence of bacteraemia after scaling and root planing between the chlorhexidine and sterile water irrigation groups and in patient who did not receive irrigation (control group).⁵

Aims and objectives

- To assess the effect of povidone iodine mouth rinsing on the incidence of post-scaling bacteraemia.
- To investigate the magnitude and microbial profile of bacteremic isolates after scaling.

Materials and methods

Source of data:

Subjects reporting to the Dept of Periodontics of A.B. Shetty Memorial Institute of Dental Sciences, Deralakatte, Mangalore, were selected for the study.

Sample size:

Total 60 subjects between the age group of 20 – 60 year were randomly selected and divided in to two groups. Group A – 30 subjects who would rinse with povidone iodine before ultrasonic scaling. Group B – 30 control subjects without povidone iodine rinse prior to ultrasonic scaling.

Inclusion criteria:

- Subjects with plaque induced gingivitis
- Subjects with good systemic health and not received any periodontal therapy in the past 6months.

Exclusion criteria:

- Subjects with any systemic disorders and allergy to iodine
- Any cardiac defects or other conditions requiring antibiotic cover.
- Pregnant women, lactating women and women in their menstrual phase.
- Subjects currently on antibiotics, steroids.

Clinical examination:

- Medical and Dental history was taken.

- Non-invasive clinical parameters such as gingival inflammation and plaque were recorded before scaling.
- Plaque was assessed on buccal and lingual surfaces of selected teeth using plaque index (PI; Silness & Loe 1964)
- Gingival inflammation was assessed on maxillary and mandibular incisors, canines and premolars using modified papilla, margin, attached gingiva index (mPMAI; Schour & Massler 1947) before ultrasonic scaling.

Study design:

- Informed consent was taken from the subjects before the procedure.
- In the test group 5ml of venous blood sample was taken at the baseline. After which the subjects were asked to rinse the mouth with 10ml of povidone iodine 5% solution for 2 minutes and asked to expectorate. Ultrasonic scaler was used at maximum power and maximum water flow to clean the gingivitis involved teeth supra and subgingivally for a total of two minutes. One more blood sample was taken after the ultrasonic scaling.
- In the control group ultrasonic scaling was done without prior povidone iodine mouth rinsing. The blood samples will be taken at the baseline and after the scaling.

Microbiological analysis:

The bacterial culturing of the blood samples was done in the central research laboratory of A.B. Shetty Memorial Institute of Dental Sciences, Deralakatte, Mangalore.

The blood samples were immediately sent to the laboratory and centrifuged at 4,000rpm for 10min. The supernatant was discarded and the sediment was cultured on blood agar and mutans sanguis agar and incubated both anaerobically as well as aerobically at 37°C for 48 hours to 1week. The cultures were checked daily for bacterial growth.

Results:

Total 60 subjects between the age group of 20 – 60 year were randomly selected and divided in to two groups.

Group A – 30 subjects who would rinse with povidone iodine before ultrasonic scaling.

Group B – 30 control group without povidone iodine rinse prior ultrasonic scaling.

The clinical data from the two groups are represented in the table.

The above table shows the mean scores of plaque index and mPMAI index of the two study groups. Both the groups show fair oral hygiene and moderate gingivitis.

In this study bacterial isolates were recovered from the baseline blood sample of one subject and consisted of staphylococci. However, these microorganisms were considered to be skin contaminants from blood sampling procedure. No microorganisms of oral origin were found in any of the baseline blood samples as well as from the post-scaling blood samples of both the study groups.

Clinical data	Mean Score	
	Group A (n=30)	Group B (n=30)
Plaque index	1.1	1.3
mPMA index	1.2	1.3

Discussion:

Transient bacteremia frequently occur secondary to several periodontal procedures. The purpose of the present study was to investigate the effects of povidone iodine mouth rinsing on the incidence of post-scaling bacteraemia. Generally, inflammatory conditions such as gingivitis and chronic periodontitis, which are precipitated by the buildup of plaque biofilms, the periodontal vasculature proliferates and dilates, providing an even greater surface area that facilitates the entry of microorganisms into the bloodstream. Often, these bacteremias are short-lived and transient, with the highest intensity limited to the first 30 min after a trigger episode. Recently it has been reported that the peak incidence and magnitude of bacteremia occurred 30s after completion of full mouth scaling gingivitis patient but the duration of scaling was not disclosed.⁹

Different forms of aerobic and anaerobic culture methods have been utilized. However, irrespective of the culture method, a larger volume of blood drawn does not necessarily concur with a higher incidence of bacteremia⁹.

The results of studies of postoperative transient bacteremia vary from one study to another, depending on the type of surgical treatment and the method used for isolation of microorganisms from blood. Fastidious microorganisms requiring special conditions or nutrients may not survive or grow in many common blood culture systems. Because of the rapid growth of some microorganisms, they may inhibit or outnumber other organisms. Phagocytic cells and antimicrobial substances in blood may prevent the growth of microorganisms in blood cultures. Antimicrobial agents in blood from patients on antimicrobial treatment may inhibit the growth of susceptible microorganisms.

In the present study blood samples were collected at baseline and 2min after ultrasonic scaling from patients with fair oral hygiene and moderate amount of plaque. All the subjects included in this study had mild to moderate gingivitis. The presence of inflammation in the periodontal site is an important contributory factor to bacterial dissemination. Studies have shown increased incidence of bacteremia in subjects with periodontitis compared to those with gingivitis (where the degree of inflammation is considered less severe) and those with healthy periodontium. For instance, the incidence of bacteremia varies from 5 to 75% for subjects with periodontitis, as opposed to 5 to 20% for subjects with gingivitis.⁷

In the present study blood culturing was done at the baseline and 2min following ultrasonic scaling but no positive cultures were obtained at either instances. However occurrence of bacteremia cannot be ruled out, it is more likely that the magnitude of bacteremia following ultrasonic scaling was very less, that could not be evidenced by the culturing methods. Studies have observed that bacteremia differed considerably in quality and quantity between different treatment groups.⁹ Therefore, more sophisticated and sensitive methods, such as PCR of 16S rRNA genes may need to be employed to identify such small degree of bacteremia. However, this technique was not within the confines of present study. On the contrary, two studies that employed PCR showed lower incidences of bacteremia due to oral manipulations than that reported by culture techniques. Comparison of different experimental

bacteremia studies is difficult because there is no uniform agreement between timing of sampling, volume of blood drawn, and bacteriological identification methods.

Conclusion:

Bacteremia is more likely to develop after certain treatments, specifically invasive procedures. Studies have reported higher incidence of bacteremia after dental extraction, subgingival scaling and intraligamentary injection². In this study we did not find bacteremia after ultrasonic scaling in subjects who did not rinse with povidone iodine prior scaling as well as in subjects who rinsed with povidone iodine before scaling. However risk for bacteremia following ultrasonic scaling cannot be ruled out. The absence of the bacteremia in our study groups could be explained by various factors. Such as,

- All the subjects included in this study had good systemic health. In healthy subjects any transient bacteremia would be quickly cleared by the host immune system.
- The subjects included in the present study had only moderate gingivitis. The degree of inflammation is also known to affect the incidence of bacteremia, as studies have shown less incidence of bacteremia in gingivitis patients compared to periodontitis patients⁷.
- Ultrasonic scaling could be considered less traumatic to the tissue compared to other invasive procedures, thus resulting in very less magnitude of bacteremia that could not be isolated from the blood culture.

Further investigations need to be carried out with increased sample size and may be with more sophisticated bacterial identification methods to demonstrate the transient bacteremia and also to establish the usefulness of mouthrinsing with antimicrobial agents in preventing such bacteremia following invasive dental procedures.

References

1. Cherry M, Daly CG, Mitchell D, Highfield J. Effect of rinsing with povidone-iodine on bacteraemia due to scaling: a randomized-controlled trial. *J Clin Periodontol* 2007; 34: 148-155.
2. Rhan R, Schneider S, Diel O, Schafer V & Shah P. Preventing post-treatment bacteraemia: Comparing topical povidone-iodine and chlorhexidine. *Journal of the American Dental Association* 1995; 126, 1145-1148.
3. Kinane D, Riggio M, Walker K, Mackenzie D & Shearer B. Bacteraemia following periodontal procedures. *Journal of Clinical Periodontology* 2005; 32: 708-713.
4. Rhan R. Review presentation on povidone-iodine antiseptics in the oral cavity. *Postgrad Med J* 1993; 69(Suppl. 3): S4-S9.
5. Lofthus J, Waki M, Jolkovsky D, Otomo Corgel J, Newman M, Fleming T & Nacharni. Bacteraemia following subgingival irrigation and scaling and root planing. *Journal of Periodontology* 1991; 62: 602-607.
6. N. B. Parahitiyawa, L. J. Jin, W. K. Leung, W. C. Yam, and L. P. Samaranyake: Microbiology of Odontogenic Bacteremia: beyond Endocarditis. *Clinical Microbiology Reviews*, January 2009, p. 46-64, Vol. 22, No. 1.
7. Forner, L., T. Larsen, M. Kilian, and P. Holmstrup.. Incidence of bacteremia after chewing, tooth brushing and scaling in individuals with periodontal inflammation. *J. Clin. Periodontol* 2006. 33:401-407.
8. Kinane, D. F., M. P. Riggio, K. F. Walker, D. MacKenzie, and B. Shearer. Bacteraemia following periodontal procedures. *J. Clin. Periodontol* 2005. 32:708-713.
9. Anders Heimdahl, I Gunnar Hall, Maria Hedberg, Hans Sandberg, Per-osten Soder, Kajsa Tunér, and Carl Erik Nord. Detection and Quantitation by Lysis-Filtration of Bacteremia after Different Oral Surgical Procedures. *Journal of clinical microbiology*, Oct. 1990, p. 2205-2209 Vol. 28, No. 10.

Rehabilitation of a partially edentulous patient with cleft lip and cleft palate

One of the most commonly occurring maxillofacial deformity is cleft lip and cleft palate. Incidence of cleft lip ranges between 20% and 30% among all facial clefts. Cleft lip is more common in males and cleft palate is more common in females. Cleft palate incidence ranges between 30-45%. 85% Cleft lip are unilateral. More than two third occur on left side.

Pathogenesis:-

Shafer et al stated that cleft palate appears to represent a disturbance in the normal fusion of the palatal shelves, which is a "failure to unite due to lack of force, interference by the tongue, or a disparity in the size of the parts involved."

Rehabilitation

Historical perspective:-Unfortunate victims of nature were scorned, ridiculed or even ostracized from society. Some were worshipped as deities, whereas others were feared and condemned to death. In 16th century, barber-surgeon Ambroise Pare and his student Franco tried to surgically close "harelips". They outlined the basic treatment principles for cleft patients. The treatment of choice to seal off "fissures of the palate" during this period was the obturator. In 1511, Amatus Lusitonus constructed the first known prosthesis designed to improve the speech of the cleft palate patient. In 1875, Passavant's velum lengthening procedure and Schoenborn's pharyngeal flap improved the tonal quality of the patient's speech. In 1881, Pierre Fauchard advocated nasal and pharyngeal extension to a denture base to aid in speech. In 1929, Fitzgibbons designed a fixed obturator made of gold. Most modern cleft palate prostheses are rigid made of autopolymerising acrylic resin, heat cure acrylic resin or metal.

Advances in the management of patients with cleft lip and cleft palate is very dramatic. The treatment of cleft palate is a combined effort by the Plastic surgeon, Orthodontist, Prosthodontist and Speech Therapist [**Team Concept by Ivy and Cooper**]

Natal and primary dentition period:

[A] Lip surgery (cheiloplasty) "the rule of tens" - 10 wks, 10 lbs, Hb 10 ie, when the baby is at 10th week, attains 10 lb wt and Hb level is 10.

Goal of lip and nose repair: Restoration of lip function & improvement in nasal appearance.

Definitive repair attempted only after dental, orthognathic and maxillary components treated successfully. Primary palatal closure should not be attempted at that time.

[B] Palatal repair

Objectives:-enhance normal speech, provide anatomical palatal closure and minimize maxillary growth inhibition and dentoalveolar deformities. Timing ->from 12 months to 4 years. Most accepted-> Zurich 2-stage palatoplasty.

Primary and mixed dentition:

Orthodontic treatment attempted. Dental irregularities, constricted maxillary arch and lateral shift of the mandible require early correction. Expand the maxilla & correct crossbite. Maxillary expansion appliances. Standard orthodontic treatment is done in the permanent dentition.

[A] Surgical intervention

Velopharyngeal discrepancies corrected by:-Pushback and palatal closure initially(9-12months)&superiorly based pharyngeal flap procedure (3-7years). Interim intervention by Prosthodontist if needed. Speech therapy if needed.

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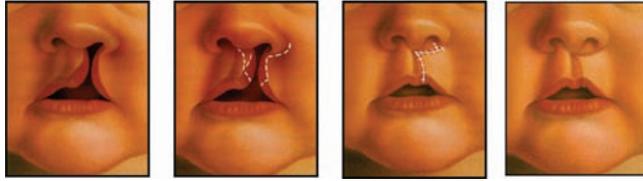


FIG.1



FIG.2



FIG.3



FIG.4



FIG.5



FIG.6



FIG.7

[B] Bone grafting the alveolar cleft- By plastic surgeon

Current trend-close oronasal fistula and bone graft the alveolar cleft with iliac cancellous bone and marrow during the late mixed dentition.

Goals-

- i) separate oral and nasal cavities.
- ii) stabilize maxillary segments
- iii) to provide normal quality of bone in the alveolus for orthodontic movement and support for the teeth.
- iv) to provide adequate 3-dimensional bone volume for placement of implants.

Definitive Prosthodontic management

Ideal time 25 years of age.

Well-fitting interim removable partial denture to replace any missing teeth if a bone graft is scheduled in the future.

1] Removable partial denture residual alveolar cleft after bone grafting.

2] Fixed partial dentures

- Size and colour deficiencies corrected by-Bonded composite restorations or porcelain veneers.
- Maryland bridges
- For conventional FPDs-better to choose more abutments.

3] Osseointegrated implants

Advantages:-

- ◆ Abutment tooth preparation not required.
- ◆ Increased loading of abutment teeth is avoided.
- ◆ Implants in the alveolar clefts may transfer functional forces to the graft which could decrease resorption of the graft.

Case report

A 24 year old female patient referred by an Orthodontist reported to our department for maintaining the space in the maxillary anterior region.

Oral examination revealed a deep cleft palate, underdeveloped maxilla, loss of vestibular depth and missing maxillary anterior and posterior teeth. After

fabricating an obturator with anterior teeth, the patient was referred to department of plastic surgery for expert management of cleft palate. The cleft palate was repaired by alveolar bone grafting with iliac crest graft and the patient was sent for speech therapy. The patient reported back around one year. The interim RPD was acting as a space maintainer and contributing to the aesthetics.

All the missing teeth and spaces created during teeth alignment were closed with fixed partial dentures.

Discussion

Cleft anomaly is not life threatening, but if left untreated or not treated properly, the patient can become physically and psychologically handicapped. Disabilities can range from minor cosmetic discrepancies to a major functional disability combined with cosmetic disfigurement. Rehabilitation is a complex process of restoration of a previous state following a major change. To restore the patient's physical state and quality of life to acceptable levels often requires major efforts, time, expertise and expense.

Within the past 50 years, advancements in technology, genetic and craniofacial research, better diagnostic methods, and the team effort have done much to elevate the standard of care for the cleft palate patient.

Conclusion

The total care rendered to a patient with a cleft anomaly can be successfully concluded only if the involved specialists work as a team and exercise objectivity, commitment and mutual respect.

References

- 1) John Beumer III, D.D.S., M.S., Thomas A. Curtis, D.D.S., Maxillofacial Rehabilitation: Prosthodontic and Surgical Considerations.
- 2) Thomas D. Taylor, DDS, MSD, Clinical Maxillofacial Prosthetics
- 3) Rosenstiel, Land, Fujimoto, Contemporary Fixed Prosthodontics, fourth edition.
- 4) DCNA Maxillofacial Prosthodontics-Vol 34; No.2, April 1990.
- 5) The Journal of Indian Prosthodontic Society, Vol.2, No.1, March, 2002.
- 6) The Journal of Indian Prosthodontic Society, Vol.3, No.1, March, 2003.

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- 1) Dr. K. Harshakumar MDS Professor.
- 2) Dr. T. Sreelal MDS Prof. and HOD, Dept. of Prosthodontics, Govt. Dental College, Thiruvananthapuram

Lasers in endodontics

Abstract

In this fast changing era, not a single aspect of our life is left untouched by lasers. Laser technology enables dentist to perform treatment procedures better than how they have been performed conventionally. It offers incredible precision, less pain and faster healing. Lasers have positively affected every discipline of dentistry and are no longer limited to treating soft tissue conditions. They have become widely accepted even in modern dental care such as cosmetic dentistry. The word laser has a magic hold on society and is perceived to be modern, high-tec and better. This article gives an overview of dental lasers and discusses their clinical applications in endodontics.

Key words

Laser, Endodontic application

Introduction

The concept of laser was first predicted in 1917 by Sir Einstein. Almost immediately after development of ruby laser by Theodore H. Maiman in 1960⁽¹⁾, researchers postulated that it could be applied to a variety of areas including medicine and dentistry. In dentistry lasers find application in diagnosis, caries prevention, endodontics, periodontics, pain control and even cavity preparation. A pulsed Nd: YAG laser was the first to be cleared for dental use by U.S. Food and Drug administration. Subsequently CO₂, Ho: YAG, Argon were cleared. The latest are the Er: YAG lasers for hard tissue removal. The first to use lasers in endodontics were Dr. Weichman and Dr. Johnson (1970) who attempted to seal the apical foramen of freshly extracted teeth using high power (CO₂) laser⁽²⁾. The word LASER is an acronym for Light Amplification by Stimulated Emission of Radiation.

Laser Device

Optical cavity in the centre of the device has a core of chemical elements, molecules or compounds called the active medium. There are two mirrors one at each end of the optical cavity placed parallel to each other. Surrounding this core is an excitation source, either a flash lamp strobe device or an electrical coil which provides the energy into the active medium. Other mechanical components include cooling system, focusing lenses and other controls.

The mirrors at each end of the active medium reflect the photons produced by stimulated emission back and forth to allow further stimulated emission and successive passes through the active medium increase the power of the photon beam. This is the process of amplification. One of the mirrors is slightly transmissive allowing light of sufficient energy to exit the optical cavity.

All available dental laser devices have emission wavelengths of approximately 0.5 μ m (500nm) to 10.6 μ m (10,600nm). They are therefore within the visible or invisible infrared nonionizing portion of the electromagnetic spectrum and emit thermal radiation.

Characteristics of laser beam.

Unlike ordinary light laser light is monochromatic (has one specific colour). In dental applications that colour may be visible or invisible. Laser light possesses three additional characteristics

-Collimation (Light beam emitted from laser device has specific size and shape)

-Coherency (Light waves produced are all in phase with one another and have identical wave shapes)

-Efficiency (thermal energy)

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Classification of lasers

1. Based on application

- Soft tissue lasers Eg : Argon, CO₂, diode
- Hard tissue lasers Eg : Er : YAG
- Resin curing lasers Eg : Argon

2. Based on mode of application

- Used in contact mode Eg : Nd : YAG
- Used in non contact mode Eg : CO₂

3. Based on level of energy emission

- Soft lasers having low level energy Eg : He-Neon, diode
- Hard lasers having high energy level Eg : Er : YAG, Nd : YAG

4. Based on wave length of the beam

- Ultra violet rays – 140 to 400 nm
- Visible light – 400 to 700 nm
- Infrared – 700 to microwave spectrum

5. According to the nature of active medium

- Gas lasers Eg : CO₂ lasers, Argon lasers
- Liquid lasers Eg : Dye lasers
- Solid state lasers Eg : Ruby lasers, Nd : YAG lasers
- Semi conductor lasers Eg : Gallium, Arsenide

6. According to ANSI and OSHA standards lasers are classified as

Class I

Do not pose a health hazard. These devices are totally enclosed and the beam does not exit the housing.

Class II a

Low power output visible lasers that are hazardous when viewed directly for >1000 seconds.

Class II b

Low powered visible lasers that are hazardous when viewed for >0.25 seconds

Class III a

Medium powered lasers that are normally hazardous if viewed for >0.25 seconds without eye protection

Class III b

Medium powered lasers that can be hazardous if viewed directly for any duration

Class IV

High powered lasers (>0.5W) that produce ocular, skin and fire hazards.

Laser delivery systems

There are 2 delivery systems

1. Flexible hollow wave guide or tube that has an interior mirror finish. Laser light is reflected along this tube and exits through a hand piece at the surgical end with the beam striking the tissue in a noncontact fashion. An accessory tip of sapphire or hollow metal can be connected to the end of the wave guide for contact with the surgical site.

2. Glass fiber optic cables with diameter ranging from 200 to 600 μ m. The glass is enclosed in a resilient sheath and cannot be bend into a sharp angle. The fiber fits

snugly into a hand piece with the base end protruding or with attached sapphire / quartz tip (for erbium lasers). This system can be used in both contact and non contact modes.

The laser device can emit the light energy in 2 modalities as a function of time, constant on or pulsed on and off. If the laser is in a pulsed mode the targeted tissue has time to cool before the next pulse of laser energy is emitted. In continuous wave mode the operator must cease the laser emission manually to provide thermal relaxation of the tissue.

Sterilization of laser devices

Small flexible optic fibers, hand pieces or tips must be steam sterilized in separate sterilization pouches after use and kept in these pouches until ready for use. Large diameter erbium fiber optic cables are not designed for steam sterilization and must be disinfected with spray disinfectant wipe⁽³⁾.

Tissue response to laser

Depends on the wave length of the laser used and optical properties of the target tissue. Responses produced can be reflection, absorption, transmission or scattering of the laser energy.

Clinical applications in endodontics

1. Vitality assessment of pulp
2. Alleviating dentinal hypersensitivity
3. Cleaning and Shaping of the root canal system
4. Endodontic surgery
5. Pulp capping and Pulpotomy
6. Tooth fractures
7. Removal of a calcified attached denticle
8. Root canal retreatment
9. Tooth bleaching
10. Diagnosis of residual pulp tissue in canals.
11. Sterilization of instruments
12. Obturation of root canals

1. Vitality assessment of pulp

Laser Doppler flowmetry (LDF) technology was first used to differentiate between vital and nonvital pulps in human in 1986 by Gazelius et al⁽⁴⁾. This technology employs a beam of infrared (780 to 820 nm) or near infrared (632.8 nm) light directed into the tissue by optical fibers. As the light enters tissue it is scattered by stationary tissue cells and moving blood cells. Photons that hit stationary cells are scattered but there will be no frequency shift. Those that hit moving blood cells are scattered and frequency shifted according to the Doppler principle. A small portion of the light containing both Doppler shifted and transmitted light is backscattered to photo detectors built into the probe, from which the laser light is beamed. Signal is then calculated with a preset algorithm in the Laser Doppler flowmetry machine. The outcome signal depends on the number and velocity of the illuminated RBC's termed flux. Flux is the number of moving red cells per second times their mean velocities. Most current laser Doppler instruments gives a read out in perfusion units (PUs).

Advantages

1. Assures objective measurement of pulp vitality

2. Could be used for patients with communication difficulties or for young children whose responses may not be reliable.

3. Non painful technique

4. Can be used in teeth with history of recent trauma or those located in the part of jaw affected following orthognathic surgery.

Disadvantages

1. Medications such as cardiovascular drugs and nicotine may affect pulpal blood flow and thus invalidate the results.

2. Requires custom made stents to ensure accurate and reproducible positioning of the probe at each session.

3. Molars with thicker enamel and dentin and variability in the position of the pulp within the tooth may cause variations in blood flow.

4. Differences in sensor output and inadequate calibration by the manufacturer may dictate the use of multiple probes for accurate assessment.

5. Dark dental dam or aluminium foil should be used to cover the gingival tissues prior to testing to avoid background noise from the gingival tissues.

2. For alleviating dentinal hypersensitivity

Dentinal hypersensitivity is characterized as a short sharp pain from exposed dentin that occurs in response to provoking stimuli such as cold, heat, evaporation, tactility, osmosis or chemicals. According to the hydrodynamic theory dentinal stimulation causes rapid dentinal fluid flow which serves as the final stimulus in activating intradental nociceptors. SEM (Scanning Electron Microscopy) of teeth with dentinal hypersensitivity shows a significantly higher number of patent dentinal tubules per millimeter⁽⁵⁾ and greater mean diameter per tubule than control teeth⁽⁶⁾.

The rationale for laser induced reduction in dentinal hypersensitivity is based on two possible mechanisms that differ greatly from each other. First mechanism implies the direct effect of laser irradiation on the electric activity of nerve fibers within the dental pulp where as the second involves modification of the tubular structure of the dentin by melting and fusing of the hard tissue or smear layer and subsequent sealing of the dentinal tubules. Lasers used for the treatment of dentinal hypersensitivity may be divided into 2 groups – low output power lasers (helium – neon and gallium / aluminium / arsenide [diode]) and middle output power lasers (Nd: YAG and carbon dioxide). Senda et al were the first to apply helium – neon laser in treating dentinal hypersensitivity using an output power of only 6mW⁽⁷⁾. It was claimed that Helium – Neon laser irradiation affects electric activity (action potential) rather than A α or C fiber nociceptors. In 1972 Kantola used CO₂ laser which caused structural change in dentin so that it closely resembled the crystalline structure of normal enamel hydroxyapatite⁽⁸⁾. Nd: YAG laser effect on dentin hypersensitivity is related to the laser induced occlusion or narrowing of the dentinal tubules⁽⁹⁾. Direct nerve analgesia and a suppressive effect achieved by blocking the depolarization of A α and C fibers also were

considered possible mechanisms for the effect of Nd: YAG laser irradiation in reducing dentinal hypersensitivity. Laser usually dramatically reduces sensitivity in one treatment though sometimes a second appointment is required about two weeks after initial lasing. Power levels used are 1/10 or 1/20 of that required for surgical procedures.

3. Cleaning and shaping of the root canal system

Successful endodontic therapy, which mainly depends on the elimination of micro organisms from the root canal system; is accomplished by means of biomechanical instrumentation of the root canal. During instrumentation a smear layer covering the instrumented walls of the root canal is formed. The smear layer consists of a superficial layer on the surface of the root canal wall approximately 1 to 2 μ m thick and a deeper layer packed into the dentinal tubules to a depth of upto 40 μ m. It contains inorganic and organic substances that also include microorganisms and necrotic debris. Smear layer also can protect the bacteria already present in the dentinal tubules by preventing the application of successful intracanal disinfection agents. Studies have clearly demonstrated that more than 35% of the canals' surface area remains unchanged following instrumentation of the root canal using nickel-titanium preparation techniques⁽¹⁰⁾.

In various laser systems used in dentistry, the emitted energy can be delivered into the root canal system by a thin optical fiber (Nd-YAG, erbium, chromium: yttrium-scandium-gallium-garnet[Er, Cr: YSGG], argon and diode) or by a hollow tube (CO₂ and Er:YAG). Laser irradiation emitted from laser systems used in dentistry has the potential to kill microorganisms. In most cases, the effect is directly related to the amount of irradiation and to its energy levels.

Limitations associated with the intracanal use of lasers are that it is almost impossible to obtain uniform coverage of the canal surface using a laser⁽¹¹⁾. Another limitation is the safety of such a procedure because thermal damage to the periapical tissues is possible. Direct emission of laser irradiation from the tip of the optical fibers in the vicinity of the apical foramen of a tooth may result in transmission of the irradiation beyond the foramen. This transmission of irradiation may affect the supporting tissues of the tooth adversely and can be hazardous in teeth with close proximity to the mental foramen or mandibular nerve.

Stabholz and colleagues recently reported the development of a new endodontic tip that can be used with an Er:YAG laser systems⁽¹²⁾. This endodontic tip allows lateral emission of the irradiation (side-firing) rather than the direct emission through a single opening at its far end. The new endodontic side-firing spiral tip (RC Lase; Lumenis; Opus Dent; Israel) was designed to fit the shape and volume of root canal prepared by nickel-titanium rotary instruments. It emits the Er:YAG laser irradiation laterally to the walls of the root canal through a spiral slit located all along the tip. The tip is sealed at its far end, preventing the transmission of irradiation to and through the apical foramen of the tooth. SEM of the lased root canal walls revealed clean surfaces, free of smear layer and debris. Open dentinal tubules were clearly distinguishable.

Erbium family of instruments provides the tissue interactions characteristics to perform effective root canal treatment. Removal of pulp and dentin is easily accomplished. There are challenges such as

(1) To maintain the water spray during the hard tissue ablation so that the temperature of the target tissue and surrounding structures is kept from being elevated.

(2) Design flexible and durable fibers to conduct laser energy

Conventional fiber optic cables (Nd:YAG, Diode) can be placed directly into the root canals. Wave guide and air cooled fiber optic delivery (Er:YAG, Er:YSGG, CO₂) have hand piece attachments that can deliver laser energy into root canals. A variety of endodontic attachments are available for laser handpiece. Studies have shown that Er:YAG laser was more effective than 17% EDTA in removal of smear layer from the root canal walls⁽¹³⁾. Erbium is the only group of wavelengths that can help in instrumentation, removal of smear layer and shaping of the canals.

4. Endodontic surgery

The goal of all endodontic surgery is to eliminate the disease and to prevent it from recurring. Egress of irritants from the root canal system into the periapical tissues is considered the main cause of failure following apicoectomy and retrograde filling⁽¹⁴⁾. Irritants penetrate mainly through the gap between the retrograde filling and the dentin. Second possible pathway of irritants to invade the periapical tissues is through the dentin of the cut root surface after apicoectomy and retrograde filling. It was shown that the dentin of apically resected roots is more permeable to fluids than the dentin of nonresected roots⁽¹⁵⁾.

Weichman and Johnson (1970) attempted to seal the apical foramen of freshly extracted teeth in which the pulp had been removed from the root canal, found that CO₂ laser used caused melting of enamel and dentin with eventual "cap" formation that can be dislodged easily⁽²⁾. Miserendino applied CO₂ laser to the apices of freshly extracted human teeth, found the recrystallized structure was smooth and suitable for retrograde filling⁽¹⁶⁾. Rationale for laser use in endodontic periapical surgery include improved hemostasis and concurrent visualization of the operating field, potential sterilization of the contaminated root apex, potential reduction of the permeability of the root surface dentin, reduction in postoperative pain and reduced risk of surgical site contamination by eliminating the use of aerosol producing air turbine hand pieces for apicoectomy. Although the cutting speed of the laser is slightly slower than the dental handpiece, absence of discomfort and vibration and less chance for contamination of surgical site as well as reduced risk of trauma to adjacent tissues may compensate for the additional time required.

5. Pulp capping and Pulpotomy

Pulp capping is defined by the American Association of endodontists as a procedure in which a dental material is placed over an exposed or nearly exposed pulp to encourage the formation of irritation dentin at the site of injury. Pulpotomy entails surgical removal of a small

portion of vital pulp as a means of preserving the remaining coronal and radicular pulp. Pulp capping is recommended when the exposure is very small (1mm or less) and the patients are young. Pulpotomy is recommended when the young pulp is already exposed to caries and the roots are not yet fully formed (open apices).

Melcer et al showed that CO₂ laser produced new mineralized dentin formation without cellular modification of pulp tissue when tooth cavities were irradiated in beagles and primates⁽¹⁷⁾. Shoji et al applied CO₂ laser energy to the exposed pulp of dogs using a focused and defocused laser mode and a wide range of energy levels (3,10,30 and 60W)⁽¹⁸⁾. Charring, coagulation necrosis and degeneration of the odontoblastic layer occurred, although no damage was detected in the radicular portion of the pulp. In some specimens dentinal bridge was formed.

In case of deep and hypersensitive cavities, indirect pulp capping should be considered. Nd: YAG and 9.6 μ m CO₂ can be used to reduce the permeability of dentin by sealing dentinal tubules. 9.6 μ m CO₂ is well absorbed by the hydroxyapatite of enamel and dentin causing tissue ablation, melting and resolidification. Use of laser for pulpotomy and pulp capping leads to a potentially bloodless field as the laser has the ability to coagulate and seal small blood vessels. Laser tissue interactions make the treated wound surface sterile and improve the prognosis of the treatment.

6. Tooth fractures

Lasers are used in repairing incomplete vertical fractures by causing fusion of the fracture. Studies have shown that CO₂ laser combined with bioactive glass paste is a potential regimen for treatment of vertical root fractures⁽¹⁹⁾.

7. Removal of a calcified attached denticle

A pulsed dye laser emitted at 504nm was used for the removal of attached denticle⁽²⁰⁾.

8. Root canal retreatment

The rationale for using laser irradiation in nonsurgical retreatment may be ascribed to the need to remove foreign material from the root canal system that may be otherwise difficult to remove by conventional methods⁽²¹⁾. The time required for removal of any root canal filling material using laser ablation was significantly shorter than that required using conventional methods. It appears that following laser irradiation some orifices of dentinal tubules were blocked with melted dentin.

9. Laser bleaching

Argon and CO₂ lasers are used. Once laser energy is applied hydrogen peroxide break down to water and free oxygen radical which combines with and thus removes stain molecules. Argon laser has affinity to dark stains and this ensures that yellow brown stains are removed. CO₂ lasers have no color requirement and is unrelated to the colour of the teeth. It penetrates 0.1 mm into hydrogen peroxide and water where it is absorbed and also enhances effect of whitening after initial argon laser process.

After initial prophylaxis, protection and isolation of soft tissues, a focused infrared spot light is used to make tooth surface warmer for better hydrogen peroxide penetration. 50% hydrogen peroxide and patented catalyst painted over tooth; argon laser is activated and used in each tooth for 30 seconds in a sequential manner. Spent mixture is removed by suction and the process repeated several times. Once the tooth whitens up, argon light is replaced by non color specific CO₂ lasers. Activated CO₂ laser hand piece is carefully used over the mixture in progressive continuous circular motion and repeated several times depending on intensity of stain. To finish whitening procedure, fluoride gel is applied over tooth, CO₂ lasers activated. Patient is given maintenance trays and final instructions. Whole process takes about two hours. 980 nm GaAlAs diode had been recently accepted by FDA for tooth whitening in addition to argon and CO₂ lasers.

10. Diagnosis of residual pulp

Application of excimer laser system emitting light at 308 nm is recommended for residual tissue detection within the root canals.

11. Sterilization of instruments

Argon, CO₂, Nd: YAG lasers have been used successfully to sterilize dental instruments⁽³⁾. Laser could be used for sterilizing endodontic files, reamers and sharp surgical instruments without destroying their effectiveness.

12. Obturation of root canals

Rationale in introducing laser technology to assist in obturating the root canal system is based on one or two major assumptions

(1) Ability to use the laser irradiation as a heat source for softening the gutta-percha

(2) For conditioning the dentinal walls before placing the obturating bonding material

The first laser assisted root canal filling procedure involved using the wavelengths of Argon (488nm) laser to polymerize a resin that was placed in the main root canals⁽²²⁾. Er:YAG laser beam (200mJ,4Hz) for 60 seconds enhanced the adhesion of epoxy resin based sealers in comparison with zinc oxide based root canal sealers⁽²³⁾.

Laser safety

Surgical lasers currently used in dentistry fall in class III or class IV groups. Eye protection is a must for dentist, assistant, patient and others. Regardless of the eye protection, a practitioner should never look directly at the laser beam. Wearing the specific protective eyewear for the specific wavelength is necessary. Most surgical lasers used in dentistry are capable of producing smoke, toxic gases and chemicals. Airborne contamination must be controlled by ventilation, using surgical mask and adequate suction.

Conclusion

Dental laser research and application has grown steadily from its modest and unheralded beginning in 1960 to a state of development that pretends dramatic implications for the clinical practice in the current 21st century and

beyond. FDA approved lasers for endodontic purposes are very few at present. So further research in this field is needed and scientific literature followed for new developments. With the development of thinner, more flexible and durable laser fibers, laser applications in endodontics will increase. It should be remembered that lasers are an adjunct to conventional therapeutic devices and approaches and not a panacea. Enthusiasm for their use must be balanced with careful judgment of cost and benefit. Dental laser has emerged to forefront now and presages a substantial contribution to the future of clinical practice of endodontics. Lasers represent a phenomenal change in dentistry and in the future the laser may be just as commonplace as the dental handpiece in the dental office.

References

1. Atkins P W, Physical chemistry 3rd edition New York W H Treeman;1986
2. Weichman J A,Johnson F M,Laser use in endodontics.A preliminary investigation. Oral Surg Oral Med Oral Pathol1971;31:416-20
3. Stabholz A,Moshonov J,Sahar-Helft S,Lasers in endodontics Dent Clin N Am 2004;48:809-32
4. Gazelius B,Olgart L,Edwall B, Edwall L.Non -invasive recording of blood flow in human dental pulp.Endod Dent Traumatol 1986;2:219-21
5. Kimura Y, Wilder-Smith P, Yonaga K, Matsumoto K. Treatment of dentin hypersensitivity by lasers. J Clin Periodontol 2000; 27:715-21
6. Absi EG,Addy M,Adams D. Dentine hypersensitivity.A study of the patency of dentinal tubules in sensitive and non sensitive cervical dentine J Clin Periodontol1987;14:280-4
7. Senda A,Gomi A,Tani T,Yoshino H,Hara G.A clinical study on "Soft Laser 632", a He-Ne low energy medical laser Aichi-Gakuin J dent Sciences 1985;23:773-80
8. Kantola S. Laser induced effects on tooth structure IV. A study of changes in the calcium and phosphorous content in dentin by electron probe microanalysis. Acta Odontol Scand 1972;30:463-74
9. Lan W H,Liu H Treatment of dentin hypersensitivity by Nd:YAG laser J Clin Laser Med Surg,1996;14:89-92
10. Peters O A,Schonenberger K,Laib A. Effects of four Ni-Ti preparation techniques on root canal geometry assessed by micro computer tomography.Int Endod J 2001;34:221-30
11. Stabholz A, Zeltzser R,Sela M, Peretz B, Moshonov J, Ziskind D.The use of lasers in dentistry :principles of operation and clinical applications. Compendium 2003 ;24:811-24
12. Stabholz A.The role of laser technology in modern endodontics.Elsevier Science BV Int Congr Series2003; 1248:21-7
13. Takeda F H,Harashima T,Kimura Yet al Comparative study about the removal of smear layer by three types of lasers. J Clin Laser Med Surg 1998;16:117-22
14. Altonen M,Matila K.Follow up study of apicoectomized molars.Int J Oral Surg 1976;5:33-40
15. Ichesco E, Ellison R,Corcoran J.A spectrophotometric analysis of dentinal leakage in the resected root{abstract}. J Endod 1986;12:129
16. Miserendino LL. The laser apicoectomy: endodontic application of CO₂ laser for periapical surgery. Oral Surg Oral Med Oral Pathol 1988; 66:615-19
17. Melcer J,Chaumate M T,Melcer F, Merard R et al.Preliminary report of the effect of CO₂ laser beam on the dental pulp of the Macaca Mulatta primate and the beagle dog.J Endod 1985;11:1-5
18. Shoji S, Nakamura M,Horiuchi H. Histopathological changes in dental pulps irradiated by CO₂ laser :J endodontics 1985;11:379-84
19. Arakawa.S,Cobb C M,Repley J Wm,Killoy W J,Spencer P.Treatment of root fracture by CO₂ and Nd:YAG :An in vitro study;Journal of Endodontics 1996;22:662-67
20. Rocca J O,Jasmin J R,Duprez J P. Removal of calcified attached denticle with a pulsed dye laser -A case report. Oral Surg Oral Med Oral Pathol 1994; 77:281-284
21. Anjo T,Ebihara A,Takeda A,et al.Removal of two types of root canal filling material using pulsed Nd:YAG laser irradiation;Photo med laser Surg 2004;22:470-6
22. Potts T V,Petrou A.Laser photopolymerization of dental materials with potential endodontic application, J Endodontics1990;16:265-8
23. Sousa -Neto M D,Marchesan M A,Decora J D et al. Effect of Er:YAG laser on adhesion of root canal sealers, J Endodontics1990;26:185-7

Chronic desquamative gingivitis - A review

Abstract

Desquamative gingivitis is an ambiguous term used to describe a range of chronic gingival diseases characterized by intense erythema, desquamation and ulceration of the free and attached gingiva. Use of clinical and laboratory parameters have revealed that vast majority of desquamative gingivitis cases have a dermatologic genesis. Cicatricial pemphigoid and lichen Planus account for over 95% of the dermatologic cases. In this paper the recent concepts of pathogenesis, differential diagnosis and treatment criteria are reviewed.

Introduction

The term chronic desquamative gingivitis was coined in 1932 by Prinz to describe a peculiar condition characterized by intense erythema, desquamation and ulceration of the free and attached gingiva. McCarthy and colleagues in 1960¹ reconsidered the literature on desquamative gingivitis and suggested that desquamative gingivitis was not a specific disease entity but rather, a non specific gingival manifestation of a variety of systemic disturbances

Classification/ Etiology

A provisional classification can now be suggested based on etiologic considerations.

1. Dermatoses
 - a) Lichen Planus
 - b) Mucous membrane pemphigoid
 - c) Bullous pemphigoid
 - d) Pemphigus
 - e) Lupus erythematoses
 - f) Epidermolysis bullosa acquisita
2. Endocrine imbalance
 - a) Estrogen deficiency in females following hysterectomy with oophorectomy or after menopause.
 - b) Testosterone deficiency in males
3. Aging (senile Atrophic gingivitis)
4. Metabolic disturbances
 - a) Nutritional deficiency
5. Abnormal response to local irritants
6. Chronic infections
 - a) Tuberculosis
 - b) Chronic candidiasis
 - c) Histoplasmosis
 - d) HIV infections
7. Drug Reactions
 - a) Toxic – antimetabolites
 - b) Allergic- barbiturates, antibiotics, mouth washes, Chewing gum etc

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Clinical features

Patients may be asymptomatic, however, when symptomatic, their complaints range from a mild burning sensation to an intense pain. Patient cannot tolerate condiments and tooth brushing causes painful denudation of the gingival surface. Approximately 50% of desquamative gingivitis cases are localized to the gingiva, although involvement of the gingiva plus other intraoral and even extraoral sites is not uncommon². Most cases were diagnosed in women in the fourth to fifth decades of life suggesting a hormonal imbalance.³

Use of clinical and laboratory parameters have revealed that approximately 75% of desquamative gingivitis cases have a dermatologic genesis. Cicatricial pemphigoid and lichen Planus account for over 95% of the dermatologic cases.⁴

Diagnosis of desquamative gingivitis

For the establishment of an accurate final diagnosis, the clinical examination has to be coupled with a thorough history and routine histologic and immunofluorescent studies.⁵ It should be mentioned, however, that despite the diagnostic approach, the cause of desquamative gingivitis cannot be elucidated in up to one third of the cases.⁶

Management

Once the diagnosis is established, the dentist has to choose the optimum management for the patient. This is accomplished according to the following three factors.

1. Practitioner's experience 2) Systemic impact of the disease and 3) Systemic complications of the medications

On diagnosis of a lichen Planus lesion, the dental practitioner takes direct and exclusive responsibility for treatment of the patient since erosive lichen Planus is responsive to topical steroids. In case of a cicatricial pemphigoid case where ocular lesions are present referral to the ophthalmologist is essential.

On diagnosis of pemphigus vulgaris the patient is immediately referred to a dermatologist for further evaluation and treatment.

When oral treatment is provided, periodic evaluation is needed to monitor the response of the patient to the selected therapy. Initially, the patient should be evaluated at 2 to 4 weeks after beginning treatment to ensure that the condition is under control. This observation should continue until the patient is free of discomfort. Appointments every 3 to 6 months would then be appropriate. It is clear that dentists play an important role in the diagnosis and management of desquamative gingivitis

Lichen Planus

Lichen Planus is a relatively common, chronic, dermatosis characterized by the presence of cutaneous violaceous papules that may coalesce to form plaques. The current evidence suggests that lichen Planus is an immunologically mediated mucocutaneous disorder where host T Lymphocytes play a central role.⁷ Majority of patients with oral lichen planus are middle aged and older females.

Oral lesions

The most common are the reticular and erosive subtypes. The typical reticular lesions are asymptomatic, bilateral and consist of interlacing white lines on the posterior region of the buccal mucosa. The lateral border and dorsum of the tongue, hard palate, alveolar ridge and gingiva are also affected. The erosive subtype of lichen planus clinically manifests as atrophic and erythematous areas.

Gingival lesions:-

Up to 10% of patients with oral lichen planus have lesions restricted to the gingival tissue, that may occur in one or more forms

1. Keratotic lesions: Raised white lesions may present as groups of individual plaques, linear or reticulate lesions.

2. Erosive lesions: These extensive erythematous areas with a patchy distribution may present as focal or diffuse hemorrhagic areas

3. Vesicular or bullous lesions: These raised fluid filled lesions are short lived on gingiva resulting in an

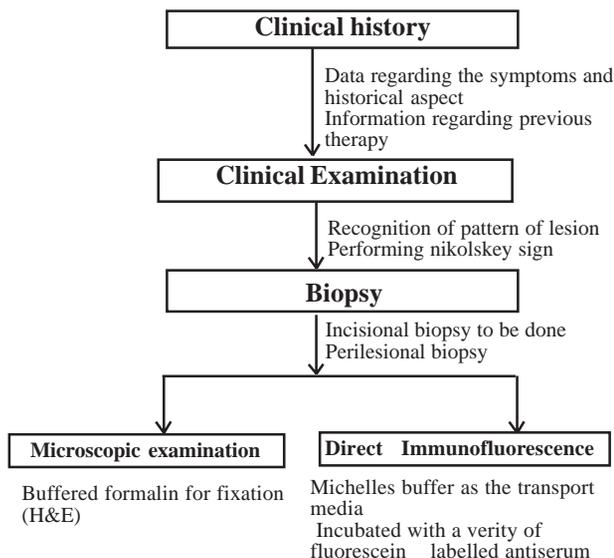


Fig1. An approach to diagnosing desquamative gingivitis

ulceration

4. Atrophic areas:-Atrophy of the gingival tissues results in erythema confined to gingiva.

Histopathology

Microscopically, three main features characterize oral lichen planus

1)Hyperkeratosis or parakeratosis 2) Hydropic degeneration of basal layer and 3) A dense, bandlike infiltrate primarily of T lymphocytes in the lamina propria. Classically the epithelial rete pegs have a "Saw-tooth" appearance. Colloid bodies (Civatte bodies) are often seen at the epithelium connective tissue interface.

Direct immunofluorescence demonstrates the presence of fibrinogen in the basement membrane.

Differential diagnosis.

- Lichenoid drug reaction
- Lupus erythematosus
- Chronic ulcerative stomatitis.
- Cicatricial pemphigoid
- Pemphigus vulgaris

Treatment

Topical application and local injection of steroids have been successful. The erosive, bullous and ulcerative lesions are treated with 0.05% fluocinonide ointment. (Lidex 3 times daily). Intralesional injections of triamcinalone acetonide (10 to 20 mg) has also been used successfully. Other treatment modalities are retinoids, hydroxychloroquine, Cyclosporine and free gingival grafts.

Cicatricial pemphigoid: (Mucous membrane pemphigoid) :

It is a chronic vesiculobullous autoimmune disorder of unknown cause that predominantly affects women in the fifth decade of life.

Clinical features: It is characterized by subepidermal blister formation with subsequent scarring of mucosal surfaces. While the oral and ocular mucosa are most often involved, other mucosal surfaces may also be affected.

Oral lesions: The most characteristic feature of oral involvement is the presence of desquamative gingivitis with typical areas of erythema, desquamation, ulceration and vesiculation of the affected gingiva. Nikolsky's sign

may also be seen.

Histopathology

It is a subepithelial or sub-basal clefting disorder. Lamina propria is infiltrated by lymphocytes.

Differential Diagnosis:

- Bullous pemphigoid
- Bullous lichenplanus
- Dermatitis herpetiformis
- Linear Ig A disease
- Erythema multiforme

Treatment:

Fluocinonide (0.05%) and clobetasole propionate (0.05%) in an adhesive vehicle can be used three times a day for up to 6 months. When lesions do not respond to steroids, systemic Dapsone has proven to be effective. Because of the systemic side effects to this drug including hemolysis, referral to a dermatologist is often indicated.

Bullous Pemphigoid

It is the most common blistering disorder often seen in elderly individuals usually presenting with urticaria-like lesions.

Clinical features:-

Lesions characteristically appear in skin, although concomitant vesiculobullous lesions may occur. Other areas include soft palate, mucosa and floor of the mouth.

Histopathology

Bullae are subepithelial in bullous pemphigoid similar to those in cicatricial pemphigoid. Ultrastructurally the basement membrane is cleaved at the level of lamina lucida

Treatment:

The primary treatment is a moderate dose of systemic prednisone. Steroid sparing strategies (Prednisone plus other immunomodulator drugs) are used when high doses of steroid are needed or steroid alone fails to control the disease.⁸ For localized lesions of bullous pemphigoid, high potency topical steroids or tetracycline with or without nicotinamide can be effective.⁹

Pemphigus vulgaris

Pemphigus vulgaris is the most common of the pemphigus diseases (Pemphigus vulgaris, pemphigus foliaceus, pemphigus vegetans and pemphigus erythematous)¹⁰. Pemphigus vulgaris is a potentially lethal chronic condition (10% mortality rate) with a worldwide incidence of 0.1 to 0.5 cases per year per 100,000 individuals¹¹. In approximately 60% of patients with pemphigus vulgaris, the oral lesions are the first sign of the disease and may herald the dermatologic involvement by a year or more.¹²

Oral lesions:

Oral lesions of pemphigus range from small vesicles to large bullae. When the bullae rupture, they leave extensive areas of ulceration. Virtually any areas of oral cavity can be involved, but multiple lesions often develop at sites of irritation or trauma.

Histopathology:

Lesions of pemphigus demonstrate a characteristic intraepithelial separation, which occurs above the basal layer. Occasionally, the entire superficial layer of epithelium are lost, leaving only the basal cells attached to the underlying lamina propria, conferring a characteristic "tombstone" appearance to the epithelial cells. Acantholysis, a separation of the epithelial cells of the lower stratum spinosum, takes place and is characterized by the presence of round rather than polyhedral epithelial cells. The intercellular bridges are

lost and the nuclei are large and hyperchromatic.¹³

Differential diagnosis

1. Bullous and cicatricial pemphigoid
2. Erythema multiforme
3. Bullous lichenplanus

Therapy

The main therapy for pemphigus vulgaris is systemic corticosteroid therapy with or without the addition of other immunosuppressive agents like azathioprine, methotrexate or cyclophosphamide. Minimization of oral irritation is important in patients with oral pemphigus vulgaris. Optimal oral hygiene is essential, because there is usually widespread involvement of the marginal and attached gingivae in pemphigus vulgaris which can be exacerbated by plaque induced gingivitis and periodontitis. To prevent flare ups, patient in the maintenance phase should receive prednisone before professional oral prophylaxis and periodontal surgery.

Linear 1gA disease (LAD)

It is an uncommon mucocutaneous disorder with predilection in women. Clinically it represents as a pruritic vesiculobullous rash, characteristic plaque or crops with an annular presentation surrounded by a peripheral rim of blisters.

Oral lesions

It consists of vesicles, painful ulcerations or erosions. The hard and soft palate are usually affected. Rarely oral lesions may be the only manifestation before the presentation of cutaneous lesions.

Histopathology

The microscopic features of LAD are similar to those observed in erosive lichen planus.

Immunofluorescence:- Linear deposits of IgA are observed at the epithelial connective tissue interface. They differ from the granular pattern observed in dermatitis herpetiformis.

Differential Diagnosis

1. Erosive lichenplanus
2. Pemphigus vulgaris
3. Bullous Pemphigoid
4. Lupus Erythematosus

Treatment

The primary treatment comprises combination of sulfones and Dapsone. Small amounts of prednisone (10-30mg) can be added if the initial response is inadequate.¹⁴ Alternatively, tetracycline (2g per day) combined with nicotinamide (1.5 g Per day) have shown promising results¹⁵.

Dermatitis Herpetiformis

It is a chronic condition that usually develops in young adults and has slight predilection for males.¹⁶ The cause is unknown but most patients have an associated gluten sensitive enteropathy.

Clinical features:

Periods of exacerbation and remission characterize the disease. Lesions are usually bilateral and symmetric pruritic papules or vesicles seen on the extensor surfaces of the extremities. Oral cavity may be affected.

Histopathology

Collections of neutrophils, eosinophils and fibrin are seen at the papillary tips of dermis. Subsequent exudation at this location contributes to epidermic separation.

Treatment

It is generally treated with dapsone, sulfoxone and sulfapyridine. Because patients often have an associated enteropathy, a gluten-free diet may also be a part of the therapeutic regimen.

Table : Diseases clinically presenting as desquamative gingivitis

	Lichen planus	Cicatricial pemphigoid	Bullous Pemphigoid	Pemphigus vulgaris	Dermatitis herpetiformis	Linear IgA disease
Clinical features	<ul style="list-style-type: none"> • Bilateral white striae • Purple pruritic papule • Seen in middle age • Buccal mucosa commonly affected 	<ul style="list-style-type: none"> • Multiple painful ulcers • Preceded by bullae • Positive Nikolsky's sign • Middle aged or elderly women • May affect mucous membranes of oral cavity, eyes and genitalia 	<ul style="list-style-type: none"> • Skin disease with infrequent oral lesions • Ulcers preceded by bullae • No scarring • Seen in elderly persons 	<ul style="list-style-type: none"> • Multiple painful ulcers preceded by bullae • Middle age • Positive Nikolsky's sign • Progressive disease 	<ul style="list-style-type: none"> • Skin disease with rare oral involvement • Vesicles and pustules • Exacerbation and remission • Young and middle aged 	<ul style="list-style-type: none"> • Vesicles, painful ulcerations • Erosive gingivitis
Histopathologic features	<ul style="list-style-type: none"> • Hyperkeratosis • Hydropic degeneration of the basal layer • Saw toothed rete pegs • Colloid bodies present • Dense infiltration of T lymphocytes 	<ul style="list-style-type: none"> • Subepithelial clefting with epithelial separation from lamina propria leaving an intact basal layer 	<ul style="list-style-type: none"> • Subepithelial clefting with epithelial separation from lamina propria leaving an intact basal layer 	<ul style="list-style-type: none"> • Intraepithelial clefting above the basal cell layer • "Tombstone" appearance of basal cells • Acantholysis present 	<ul style="list-style-type: none"> • Collection of neutrophils, eosinophils and fibrin in connective tissue papillae 	<ul style="list-style-type: none"> • Separation of basement
Diagnosis	<ul style="list-style-type: none"> • By clinical appearance • Fibillar deposits of fibrin at the dermal epithelial junction 	<ul style="list-style-type: none"> • Based on clinical features • Linear deposits of C3 with or without IgG at basement membrane 	<ul style="list-style-type: none"> • Sub-epidermal blister on histologic examination • Linear deposits of C3 with or without IgG at basement membrane 	<ul style="list-style-type: none"> • Based on clinical features • Intercellular deposits in epithelium, IgG in all cases • C3 in most cases 	<ul style="list-style-type: none"> • Characteristic histopathology and demonstration of specific IgA immunofluorescence 	<ul style="list-style-type: none"> • Linear deposits of IgA at basement membrane zone
Treatment	<ul style="list-style-type: none"> • Topical or systemic steroids • Retinoids may be helpful • Follow-up examination necessary 	<ul style="list-style-type: none"> • Topical or systemic steroid 	<ul style="list-style-type: none"> • Systemic steroids/immunosuppressive drugs 	<ul style="list-style-type: none"> • Systemic steroids, occasionally immunosuppressive drugs for their steroid sparing properties 	<ul style="list-style-type: none"> • Dapsone, sulfazone, sulfapyridine 	<ul style="list-style-type: none"> • Sulfonyl or corticosteroids

Drug Eruptions

Drugs can act as an allergen either alone or in combination, sensitising the tissues and resulting in allergic reaction of skin and oral cavity. Stomatitis medicamentosa is characterized by eruptions in the oral cavity resulting from sensitivity to drugs that have been taken by mouth or parenterally. The local use of medicaments in the mouth is referred to as stomatitis venenata or contact stomatitis, examples are aspirin burn or stomatitis due to topical penicillin.

In general, drug eruptions in the oral cavity are multiform. Vesicular and bullous lesions occur most commonly, but pigmented or nonpigmented macular lesions are also found. Erosions, often followed by deep ulceration with purpuric lesions, may also occur. The lesions are seen in different areas of the oral cavity, with the gingiva often affected.¹⁷ Some of the compounds that may cause contact allergy in the gingiva are, mercurial compounds (Amalgam), tartar control tooth pastes (Pyrophosphates) etc. Cinnamon compounds can result in intense erythema of gingival tissue (Plasma cell gingivitis). Elimination of the offending agent usually leads to resolution of the lesion within a week.

Conclusion

Chronic desquamative gingivitis is not a specific disease entity, but a gingival response associated with a variety of conditions. Evidence suggests that about 75% of desquamative gingivitis cases have a dermatologic origin. Females in the fourth to fifth decades of life are mostly affected. Diagnosis of the cases require a thorough clinical history, clinical and histopathological examination. For the management of oral lesions periodic evaluation is needed to monitor the response of the patient to the selected therapy. If the disease have a systemic involvement, an interdisciplinary approach with a dental practitioner,

dermatologist and ophthalmologist are essential for the management of the disease.

References

1. Mc Carthy FP, Mc carthy PL, Shklar G: Chronic desquamative gingivitis; A reconsideration. *Oral surg* 1960; 13:1300.
2. Nisengard R, Levin R; Diagnosis and management of desquamative gingivitis. *Periodontal Insights* 1995;2:4
3. Merritt AH; Chronic desquamative gingivitis. *J Periodontol* 1933; 4:30
4. Nisengard RJ, Neiders M; Desquamative lesions of the gingiva. *J Periodontol* 1981; 52:500.
5. Yih WY, Maier T, Kratochvil FJ, et al: Analysis of desquamative gingivitis using direct immunofluorescence in conjunction with histology. *J Periodontol* 1998; 69:678.
6. Rees T D; Adjunctive therapy. *Proceedings of the world workshop in clinical periodontics, Chicago. The American Academy of periodontology, 1989, X1-X31.*
7. Malmstrom M, Kontinen Y T, Jungell P, et al: Lymphocyte activation in oral lichen planus in situ. *Am J clin Pathol* 1988; 89:329.
8. De Vita S, Neri R, Bombardieri S: Cyclophosphamide pulses in the treatment of rheumatic disease; an update. *Clin Exp. Rheumatol* 1991; 9: 179. 9. Nisengard R; Periodontal implications: Mucocutaneous disorders. *Ann Periodontol* 1996; 1:401.
10. Robinson JC, Lozada-Nur F, Frieden I: oral Pemphigus vulgaris: a review of the literature and a report on the management of 12 cases. *Oral surg Oral med Oral pathol Oral Radiol Endod* 1997; 84: 349.
11. Yih WY, Maier T, Kratochvil FJ, et al: Analysis of desquamative gingivitis using direct immunofluorescence in conjunction with histology. *J Periodontol* 1998; 69:678
12. Siegel MA, Balciunas B A: Oral Presentation and management of vesiculobullous disorders. *Sem Dermatol* 13: 78-86, 1994.
13. Zegarelli DJ, Zegarelli EV: Intraoral pemphigus Vulgaris. *Oral Surg* 1977; 44: 384.
14. Chorzeliski TP, Jablonska S, Maciejowska E: Linear IgA bullous dermatosis of adults. *Clin Dermatol* 1992; 9:383.
15. Peoples D, Fivenson DP.; Linear IgA Bullous Dermatitis: Successful treatment with tetracycline and nicotinamide. *J Am Acad Dermatol* 1992; 26:498.
16. Economopoulou P, Laskaris G: Dermatitis herpetiformis: Oral lesions as an early manifestation. *Oral Surg Oral Med Oral Pathol* 1986; 62:77.
17. Gallagher G T: Oral mucous membrane reaction to drug and chemicals *Curr Opin Dent* 1991;7:77.
18. Newman, Fermin Carranza. *Clinical Periodontology* Ninth edition, WB Saunders.
19. Shantiriyaa Reddy, *Essentials of clinical periodontology and periodontics*. Second edition. Jayee.
20. Newman, Fermin Carranza. *Clinical Periodontology* Seventh edition, WB Saunders

Reverse hybridisation - The brand new bond

Abstract

Composite Resin restorations are slowly but surely replacing amalgam as a restorative material. Years of research have eliminated many short comings of composite resins. But micro leakage leading to discolouration and secondary caries remain to be unsolved mysteries till date. The cause attributed is due to incomplete penetration of binding resins of the collages fibrils exposed after acid conditioning. Research studies have shown that complete resin penetration of collagen fibrils does not take place.

Research Studies directed at removal of the exposed collagen fibrils completely have yielded better bond strength following application of bonding resin. This novel approach of deproteinisation of collagen fibrils exposed after acid conditioning & subsequent bonding to achieve stronger & predictable bond between resin & dentine. Thus bonding on to a new type of dentine substrate which is physically different from conventional dentine surface formed after acid conditioning alone seems to be an effective solution to micro leakage associated problems of composite resin restorations.

This article deals with the nature of a new type of dentine substrate and new type of hybrid layer formed – Reverse Hybrid Layer. An insight into Reverse Hybridization- a new paradigm.

Key words: hybrid layer, reverse hybridisation, smear layer, resin tag, de proteinisation

Introduction

The possibility of bonding restorative materials to dentin and enamel has intrigued the dental profession for many years. Strong adhesion between the two would eliminate the need for retentive undercuts and prevent formation of marginal gaps and thus the penetration of bacteria and coloring matter. Adhesion of restorative material and enamel has become routine and reliable aspect of modern adhesive restorations. But dentinal adhesion proves to be more difficult and less predictable. Much of the difficulty in bonding to dentine is the result of the complex histological structure and variable composition of dentine itself.

Modern day dentine bonding is the result of two fundamental processes-

1. Mineral phase of dentine removed without damaging the collagen matrix with the help of acid conditioning¹.
2. Microscopic spaces created by removing the inorganic minerals must be filled with an adhesive resin that penetrates the exposed collagen fibril network extending into the partially demineralised dentine which is later polymerised hard¹.

This resin infiltrated dentine layer is called hybrid layer and the mechanism is called hybridisation- first described by Nakabayashy et al in 1982².

Optimal bond strength is derived from complete resin penetration of exposed collagen fibril network. It has been suggested that dentine bonding agents do not fully diffuse through the collagen network that remain after acid conditioning. This results in the formation of a weak porous layer of collagen protected neither by hydroxyapatite nor encapsulated by the resin. Subsequent hydrolysis of exposed collagen fibrils would lead to degradation of the dentine- resin bond resulting in decreased bond strength and micro leakage.

Nakabayashy and others³ have suggested that the demineralized zone should be kept to a minimum to avoid long term bond degradation caused by incomplete penetration of resin through collagen network.

Reverse Hybridization - Future of dentine bonding?

Recent studies have concentrated on removing this collagen network completely preventing the formation of unsupported collagen fibrils. Removal of organic collagen following acid conditioning and subsequent bonding onto partially demineralized dentine layer may produce more durable adhesion to the hydroxy apatite component of the dentine substrate. Removal of collagen fibril network can be accomplished by washing with 5% NaOCl for 2 minute. This is followed by rinsing and drying to remove excess water. Later the bonding agent is applied and polymerized insitu.

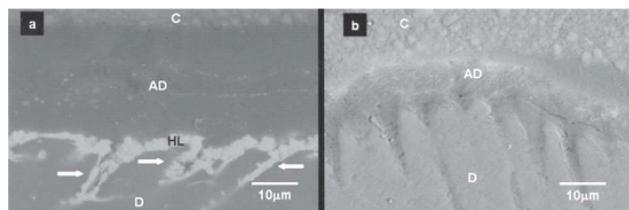
This deproteinisation of dentinal surface done after acid conditioning has found to be a better substrate for bonding when used along with 4th and 5th generation bonding agents.

Studies have been carried out with different concentration of NaOcl - 1.5% for 2

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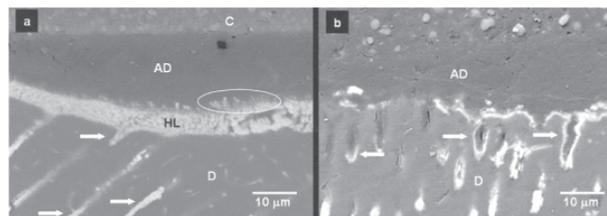


a: Intense accumulation of silver grains within hybrid layer & along dentinal tubules

D= dentin, C=composite resin, HL=hybrid layer, AD= adhesive resin.¹¹

Fig. 1

b: With NaOCl. No silver deposition could be observed along the interface.



a: acid etched specimen showing silver deposits within the hybrid layer.

b: acid etched & NaOCl treated specimen-hybrid layer not apparent.

Nano leakage pattern show thin discontinuous silver depositions along resin-dentine interphase & between resin tags & tubule walls.¹¹

Fig. 2

minutes and 5% NaOCl for 2 minutes. Both have shown to eliminate the collagen layer left back after acid conditioning^{4,5}. SEM evaluation conducted in regard to deprotonization (collagen removal) by NaOCl shows morphology of acid etched and deprotonized dentine was completely different from dentine etched alone. Inaba et al demonstrated that diameter of open dentinal tubules increased from 1.8 to 4 μm after 10% NaOCl wash. It was found out that diameter of tubule orifice increased after NaOCl treatment of acid demineralized dentine due to loss of peritubular dentine. Diameter of the lateral branch of tubule also increased and became more numerous when compared with a conventional etching procedure⁶.

The phosphoric acid/NaOCl treatment produced a 'new dentine substrate. As suggested by Inaba et al it had porous but mineralized surface. This substrate is more similar to enamel than dentine in nature. Predictable long term bonds may be possible onto this mineralized substrate. Prati C⁶ et al studied the effect of various dentine adhesives on the acid etched /NaOCl treated dentine substrate. He showed that adhesive system like Optibond Solo (Kerr manufacturing company) are more acidic in nature and they were able to re-etch the mineral phase of the collagen free dentine surface to a depth of 0.3 – 0.5 μm which is too shallow to be detected by SEM. This would then create a "nano hybrid layer or nano RIDL" (resin infiltrated dentine layer)⁷.

More over molecular size of this bonding system is much smaller which accounts for its increased permeability through collagen free mineralized surface⁶.

Larger resin tags formed after acid/NaOCl treatment contributes to increased total bond strength.

Moreover these days, filled adhesive systems are available. This results in resin tag formation with filler particles inside the tags (demonstrated by Perdiago)⁶. These filled resin tags again add to the total shear bond strength.

This new dentine substrate which is acid/NaOCl treated when impregnated with a bonding resin results in the formation of a new type of hybrid layer. Instead of conventional collagen- resin hybrid layer formed, here a hydroxyl apatite – resin hybrid layer is formed. This new hybrid layer is termed reverse hybrid layer.

Let us observe what happens during reverse hybrid layer formation. Acid conditioning removes smear layer and exposes the collagen fibrils of dentine matrix. This is followed by NaOCl wash for 2 min. which not only removes the exposed collagen fibrils but also solubilizes the fibrils down into the underlying partially mineralized dentine to

create submicron porosities within the mineral phase. Cylindrical channels (0.1 μm in diameter) previously occupied by collagen fibers are now available for resin infiltration within the mineralized matrix⁶. These spaces are coated with bonding resin and polymerized hard.

Acid etching of mineralized dentine decreases its modulus of elasticity from a relatively stiff 17GPa to a very low 5Mpa due to removal of apatite crystals⁶. Infiltration of resin into this demineralized dentine partially restores the stiffness of dentine matrix to a about 2-6 GPA⁶. The modulus of elasticity of the acid/NaOCl treated dentine may be better owing to improved mechanical properties of the mineralized substrate as well as increased diameter of resin tags in dentinal tubules.

Research studies are underway regarding this new concept of bonding. Studies conducted by C. Prati, S. Chersoni and D. H. Pashley on the effect of removal of surface collagen fibrils on resin – dentine bonding showed that high bond strength can be obtained by "reverse hybrid layer" phenomenon when 5th generation bonding agents were used⁶.

Similar studies were carried out by M.A. Vargas, D.S Cobb and S. R. Armstrong. They compared shear bond strength of 4th generation bonding agents with and without hybrid layer and found that ALL BOND 2 adhesive give better bond strength in the absence of collagen matrix which was removed prior to priming and bonding³.

Paulette Spencer and James R Swafford examined the nature of unprotected protein (collagen) left back after acid conditioning at the adhesive interface. They found that this unprotected collagen left back after acid conditioning served as the major inadequacy in time line bond stability⁴.

Studies conducted by Inai, Kanemura and Tagani. H⁸ showed that bonding agent based on acetone system yielded better bond strength after removal of collagen matrix with help of NaOCl.

Contradicting studies have also been reported. Franken Berger and N. Kramer compared IV & V generation bonding agents with & without hybrid layer. He reported shear bond strength were considerably low without hybrid layer.

Gwinnet⁹ concluded that the collagen layer does not significantly contribute to the interfacial strength of resin to dentine. The study also throws light on the significance of removing the exposed collagen matrix completely after acid conditioning which may otherwise under go hydrolysis due to incomplete penetration of the resin, ultimately leading to resin – dentine bond failure.

This relatively new idea of removing collagen matrix after acid conditioning and subsequent bonding to partially demineralized dentine could eliminate or substantially reduce the microleakage associated with polymerization shrinkage of composite resin. As the dentine substrate after acid etching/NaOCl treatment is more of a mineralized one it can resist the polymerisation shrinkage stress more effectively. This could eliminate the two potential disadvantages commonly associated with composite resin namely secondary caries associated with microleakage and discoloration.

Fig 1 & 2 shows back scattered SEM images of resin-dentine interface with and without hybrid layer

Conclusion

Modern day adhesive system have succeeded in achieving optimal initial bond strength. But the major short coming is the longevity of the bond. Long term bond degradation leading to microleakage and discoloration still remain as the Achilles heel for composite restorations. Reverse hybridizations seems to be novel solution in preventing this long term bond degradation. Extensive clinical studies need to be done to authenticate this procedure on a regular basis. Nevertheless reverse hybridization seems to offer light at the end of the tunnel for sure.

References

1. Nakabayashi, 1998, "Hybridization of Dental Tissue"
2. Edward. J. Swift, J. Perdiago, 1995, "A brief history and state of the art in enamel and dentine bonding". Quintessence International; Volume, 26 –No:2.
3. Vargas. M. A, D. S. Cobb, S. B Armstrong, 1997, "Resin dentine Shear bond strength and interfacial ultra structure with & without hybrid layer. Operative Dentistry: July/August, Vol 22, No:4, Page 159.
4. Paulette Spencer, James R Swaford 1999, " Nature of unprotected protein at dentine –adhesive interface".
5. Richard. S Schwartz, " Fundamentals of operative dentistry"
6. Prati. C, S. Chersoni, D.H Pshley, 1998 " Effect of removal of surface collagen fibrils on resin dentine bonding" Dental Materials: 15.323-331.
7. V de Pa Saboia, A. L Rodriguez, 2000. "Effect of collagen removal on Shear Bond Strength of two single bottle adhesive systems." Operative dentistry; 25:395-400.
8. Inai. K. Kanemura & Tagani. J, 1998 "Adhesion between collagen depleted dentine and adhesive". American Journal of Dentistry; June; 11(3) 123-71.
9. Gwinnet AJ, Tay FR & Wei SH, 1996 "Quantitative contribution of collagen network in dentine hybridization". American Journal of Dentistry: August ;9 (4) 140-4.
10. Chan D. C. N, J. W Rainhardt and D.B Boyeer, 1985" Composite resin compatibility and bond longevity of a dentine bonding agent – Invitro study." Journal of Dental Research 64 (12): December; 1402-1404.
11. Patricia de Britto, Eduardo Dasilva, Journal of applied oral science (july/aug 2009)

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Nuclear medicine - through times

Introduction

Nuclear medicine is a branch of medicine that uses radiation to provide information about the functioning of a person's specific organs or to treat disease. It comprises of diagnostic examinations that result in images of body anatomy and function. The images are developed based on the detection of energy emitted from a radioactive substance given to the patient, either intravenously or by mouth. Generally, radiation to the patient is similar to that resulting from standard x-ray examinations.

Nuclear medicine was developed in the 1950s by physicians with an endocrine emphasis, initially using iodine-131 to diagnose and then to treat thyroid disease. Nuclear medicine images can assist the physicians in diagnosing tumors, infection and other disorders; can be detected by evaluating organ function.

Specifically, nuclear medicine can be used to evaluate the fracture of bones, infections, arthritis or tumors, determine the presence or spread of cancer, analyze kidney function, to locate tumors in ovary, breast, prostate and colorectum, blood flow and function of the heart, for respiratory problems, identify blockage of the gallbladder, identify bleeding into the bowel, locate the presence of infection and assess thyroid and salivary gland functions.

The practice of Nuclear Medicine includes both diagnostic and therapeutic techniques. Most of the procedures are related to organ imaging using internally distributed radioactive material. There are also diagnostic techniques which quantitatively measure physiologic function (such as thyroid radio uptake). Several therapeutic procedures are done using radioactive materials.

Diagnostic techniques in nuclear medicine use radioactive tracers which emit gamma rays from within the body. These tracers are generally short-lived isotopes linked to chemical compounds which permit specific physiological processes to be scrutinized. They can be given by injection, inhalation or orally. The single photons are detected by a gamma camera which can view organs from many different angles. The camera builds up an image from the points from which radiation is emitted; this image is enhanced by a computer and viewed by a physician on a monitor.

The standard detector system for Nuclear Medicine is a scintillation camera. The scintillation camera uses a sodium iodine crystal to convert the energy of the gamma photon into a flash of light. Then it is detected by a photo-multiplier tube array which views the side of the scintillating crystal away from the patient. There is a collimator between the patient and the scintillating crystal so that the image will record only primary photons moving directly from the organ to the crystal. The collimator is analogous to the grid used for conventional radiography.

The photo-multiplier tubes produce an electrical pulse when they are exposed to light from the scintillating crystal. The output from all of the photo-multiplier tubes can be summed to determine the total energy of the gamma photon. This allows the spectrometer to limit the recorded photons to only those coming from the radiopharmaceutical administered for the current test. The output from each photo-multiplier tube can be evaluated in relation to the other tubes in the array to determine the exact site where the gamma photon interacted with the scintillating crystal. Once the position (on an x-y axis) of the scintillation has been determined, a point of light is placed in a corresponding position on a video display.

Discussion

Ernest Lawrence, inventor of the cyclotron, recognized the possibilities for radio medicine. Treating a patient with leukemia, John Lawrence had administered a radioactive isotope of phosphate. It was the first time that a radioactive isotope had been used in the treatment of a human, and he became known as the father of nuclear medicine.²⁴

In 1937, Joseph Hamilton was the first to use radioactive tracers to study circulatory physiology. Using radioactive sodium, he realized that these radioisotopes with a short half-life, has a property which allows them to be use without medical side effects. In 1938, technetium-99m which remains the most commonly used isotope in medicine, was discovered by Emilio Segre.²⁴

In the 1950s, Hal Anger conducted studies on medical imaging. From 1952 to 1958, he gradually developed the scintillation camera, also known as the Anger Camera, which enables physicians to detect tumors by imaging gamma rays emitted by radioactive isotopes. Developed forty years ago by Anger, these techniques remain

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the most commonly used tools in nuclear medicine.

Anger's scintillation camera evolved into modern imaging systems such as PET (positron emission tomography) and SPECT (single photon-emission computed tomography).⁵ Anger and his colleagues contributions include the multi-crystal whole body scanner (1970), gated single gamma tomography (1974), dynamic, gated PET (1978). Anger Positron Cameras were used to diagnose bone tumors in 1960's. In 1972, Yukio Yano devised a technetium-99m/phosphate system for bone scanning. In 1979, rubidium-82 was used for dynamic PET diagnosis of heart disease.

The first skeletal images were made using 85 Strontium in 1970s and these images were satisfactory for demonstrating metastases.²⁴ Using 18-fluorine, imaging technique limited because of the relatively short physical half-life of the isotope. A major improvement in skeletal imaging occurred when technetium labeled compounds were developed. Originally, the radiopharmaceutical was based on a polyphosphate complex. Since the polyphosphate chain length was unpredictable, other phosphate compounds were developed and diphosphonates labeled with 99m technetium are used.

A gallium scan uses a special camera to take pictures of specific tissues in the body after a radioactive tracer (radionuclide or radioisotope) administration and makes them visible. Each type of tissue that may be scanned (including bones, organs, glands, and blood vessels) uses a different radioactive compound as a tracer.^{27,28} The radioactivity of the tracer decreases over a period of weeks. It remains in the body temporarily before it is eliminated in the urine or feces.

During a gallium scan, radioactive gallium citrate as tracer is injected into a vein in the arm. It travels through the bloodstream and into the body's tissues, primarily the bones, liver, intestine, and areas of tissue where inflammation or a buildup of white blood cells is present. It usually takes the tracer a few days to accumulate in these areas. Most of the cases scan is done at 2 days and repeated at 3 days after the tracer is injected. Areas where the tracer accumulates in higher-than-normal amounts show up as bright or "hot" spots in the pictures. These areas may be caused by infection, certain inflammatory diseases, or a tumor.

The radiopharmaceutical has two components. There is a radioactive label which allows external detection. The important part of the radiopharmaceutical is the chemical moiety which determines exactly where and how the radiopharmaceutical will be localized. It is absolutely necessary to have a radiopharmaceutical which precisely localizes the radioactivity to the organ and the physiologic function. The technical characteristics of the radiopharmaceutical are extremely important. The gamma photon emission from the radioactive isotope must in the range of 80-200 KeV. It is desirable to have a radioactive label which only produces gamma rays. Beta particles are not energetic enough to reach the detector.^{22,43}

The most commonly used liquid radionuclides

- Ø Technetium – 99m
- Ø Iodine – 123 & 131
- Ø Thallium – 201
- Ø Gallium – 67
- Ø Fluorine – 18
- Ø Indium – 111

The most commonly used gaseous/aerosol radionuclides

- Ø Xenon – 133
- Ø Krypton – 83

The most important half-life in determining radiation exposure to the patient is the effective half-life. The effective half-life is calculated using the physical and biologic half-lives of the radioactive material. Generally radioisotopes are made in nuclear reactors and some in cyclotrons. Neutron-rich made in reactors; neutron-depleted ones are made in cyclotrons.

Reactor Radioisotopes: Half life and uses

Ø Molybdenum-99 (66 hours): Used as the 'parent' in a generator to produce technetium-99m.

Ø Technetium-99m (6 hours): To image the skeleton, heart muscles, brain, thyroid, lungs, liver, spleen, kidney, gall bladder, bone marrow, salivary and lacrimal glands.

Ø Bismuth-213 (46 minutes): Targeted alpha therapy (TAT), especially cancers.

Ø Chromium-51 (28 days): Label red blood cells and quantify gastro-intestinal protein loss.

Ø Cobalt-60 (10.5 minutes): Formerly used for external beam radiotherapy.

Ø Copper-64 (13 hours): Study genetic diseases affecting copper metabolism.

Ø Erbium-169 (9.4 days): For relieving arthritic pain in synovial joints.

Ø Holmium-166 (26 hours): Diagnosis and treatment of liver tumors.

Ø Iodine-125 (60 days): Brachytherapy (prostate and brain), evaluate the filtration rate of kidneys and to diagnose deep vein thrombosis in the leg. Used in radioimmunoassays for detecting show the presence of hormones.

Ø Iodine-131 (8 days): Used in treating thyroid cancer, imaging the thyroid, diagnosis of abnormal liver function, renal blood flow and urinary tract obstruction.

Ø Iridium-192 (74 days): used as internal radiotherapy source for cancer treatment.

Ø Iron-59 (46 days): Used in studies of iron metabolism in the spleen.

Ø Palladium-103 (17 days): To make brachytherapy permanent implant seeds for early stage prostate cancer.

Ø Phosphorus-32 (14 days): Used in the treatment of Polycythemia vera.

Ø Potassium-42 (12 hours): Determination of exchangeable potassium in coronary blood flow.

Ø Rhenium-186 (3.8 days): Pain relief in bone cancer.

Ø Rhenium-188 (17 hours): Irradiate coronary arteries from an angioplasty balloon.

Ø Samarium-153 (47 hours): Relieving the pain of secondary cancers lodged in the bone, prostate and breast cancer.

Ø Selenium-75 (120 days): Study the production of digestive enzymes.

Ø Sodium-24 (15 hours): Studies of electrolytes within the body.

Ø Strontium-89 (50 days): Effective in reducing the pain of prostate and bone cancer.

Ø Xenon-133 (5 days): Pulmonary ventilation studies.

Ø Ytterbium-169 (32 days): Cerebrospinal fluid studies.

Ø Yttrium-90 (64 hours): Cancer brachytherapy, relieving the pain of arthritis in larger synovial joints.

Ø Radioisotopes of cesium, gold and ruthenium are also used in brachytherapy.

Cyclotron Radioisotopes: Half life and uses

Ø Carbon-11, Nitrogen-13, Oxygen-15, Fluorine-18: Positron emitters used for studying brain physiology and pathology, for localizing epileptic focus, for psychiatric, neuropharmacologic, cardiologic and oncologic studies.

Ø Cobalt-57 (272 days): Marker to estimate organ size.

Ø Gallium-67 (78 hours): Tumor imaging and localization



Fig-1 Ernest lawrence

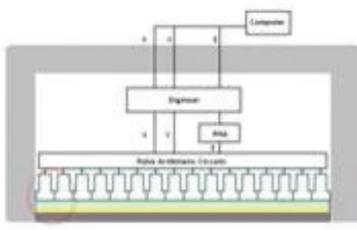


Fig-2 Pet diagram



Fig-3 John lawrence



Fig-4 Hal Anger

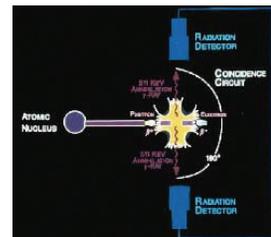


Fig-5 Gamma camera detector

of inflammatory lesions.

Ø Indium-111 (2.8 days): Brain studies, infection and colon transit studies.

Ø Iodine-123 (13 hours): Diagnosis of thyroid function.

Ø Krypton-81m (13 seconds): Functional images of pulmonary ventilation, and for the early diagnosis of lung diseases.

Ø Rubidium-82 (65 hours): PET agent in myocardial perfusion imaging.

Ø Strontium-92 (25 days): Used as the 'parent' in a generator to produce Rb-82.

Ø Thallium-201 (73 hours): Diagnosis of coronary artery disease and location of low-grade lymphomas

A bone scan means that it uses tiny amounts of radioactive materials called tracers (radionuclide) which accumulates in certain organs and tissues.^{11,23} Once introduced into the body, tracers emit gamma waves of radiation, which are detected by a special camera. This camera produces images that are interpreted by radiologists or nuclear medicine specialists. It is a diagnostic procedure used to evaluate abnormalities involving bones and joints. A radioactive substance is injected intravenously, and the image of its distribution in the skeletal system is analyzed to detect certain diseases or conditions.

Methylene-diphosphonate (MDP) can be preferentially taken up by bone. By chemically attaching technetium-99m to MDP, radioactivity can be transported. For imaging it is attached to bone via the hydroxyapatite.^{7,12} Any increased physiological function, such as due to a fracture in the bone, will usually mean increased concentration of the tracer. This often results in the appearance of a 'hot-spot' which is a focal increase in radio-accumulation, or a general increase in radio-accumulation throughout the physiological system.

Inflammatory conditions of the TMJ demonstrate increased uptake as seen in condylar hyperplasia.²² The clinician must carefully assess the history, clinical examination, laboratory data, and imaging data to arrive at the proper diagnosis. Certain conditions and situations can confound the results of the bone scan like active periodontal disease can result in an increased uptake of the radiopharmaceutical in the alveolar processes of the mandible and maxilla.

Thyroid imaging was the historical origin for Nuclear Medicine imaging. Radioactive iodine (¹³¹I) used to evaluate thyroid function in earlier days. The beta particle produces too much radiation exposure and no longer used for thyroid scanning. Now the most commonly used radiopharmaceutical is the technetium pertechnetate (TcO₄). The technetium is chemically very similar to iodine and it is actively localized in the thyroid gland.²⁴ In the human thyroid, no significant organification of technetium occurs. It does not persist in the gland in the same way that of iodine. It is useful for thyroid imaging because it has a favorable gamma emission and relatively short physical half-life results in a low level of radiation exposure. Thyroid imaging is commonly done with several radioiodine isotopes like,

¹²³I and ¹²⁵I.

Liver scan used for the purpose of finding space occupying lesions within the liver. The best way to anatomically examine the liver using nuclear imaging is to localize the radioactivity using the reticuloendothelial system phagocytes. The most frequently used radiopharmaceutical is ^{99m}Tc sulfur colloid (technetium heptasulfide). Hepatocyte function can also be assessed in the scan. More recently, iminodiacetic acid compounds have been developed. These are labeled with ^{99m}Tc and produce much better images of the liver and biliary tract.¹⁴ These radiopharmaceuticals have gone through several evolutions beginning with iminodiacetic acid or IDA, then HIDA, followed by PIPIDA and finally DECIDA.

There are different types of brain scans have been done over the history of nuclear imaging.⁹ Currently used brain scan is a study based on the distribution of cerebral perfusion. These images are usually displayed in parasagittal and trans-axial tomographic sections (SPECT).^{15,38} Brain SPECT tomoscintigraphy is used in the diagnosis of brain ischemic attack, localisation of epileptogenic focus, brain tumors, AIDS-related diseases, functional vascular residual capacity test.^{6,9,16,20,44}

Ventilation and perfusion imaging of the lungs most commonly been used to detect the presence of pulmonary embolization. It also used to give a quantitative indication of regional lung function. This kind of quantitative information is used to decide the extent of the surgical resection. In perfusion imaging localization of the radioactivity is done by using the pulmonary capillary bed as a filter to trap radioactive particles, ^{99m}Tc macro-aggregated albumen which are placed into the venous circulation.^{29,30} The albumen is heat aggregated so that 90% of the particles are between 10-90 microns in maximum dimension. After the radiopharmaceutical is localized in the lungs, images the anterior, posterior, both laterals and both posterior oblique views are taken. The radiopharmaceutical has a persistence half time (biologic half time) in the lungs of about 3.5 hours. Ventilation imaging of the lungs is generally used to show the distribution of inspired air and is performed by inhale a radioactive gas, ¹³³Xe.

The frequently used cardiac imaging procedures in nuclear medicine are infarct imaging, myocardial function study and myocardial perfusion study. Infarct imaging method uses a radioactive phosphate compound which localized in areas of necrotic tissue. This localization is not dependent on any organ function and is of the chemical changes that occur died tissues. Myocardial perfusion study method uses ²⁰¹Tl. Currently, other chemicals are used and are labeled with ^{99m}Tc. These chemicals are localized in the myocardium proportional to blood flow.²⁴ The myocardial perfusion study is usually done using a tomographic technique. Myocardial function studies measures the ability of the left ventricle to pump blood and

is done by making the blood pool radioactive.

A salivary gland scan is a nuclear medicine test that examines the uptake and secretion in the salivary glands of a radioactively labeled marker substance. Salivary gland dysfunction is encountered in various pathologic processes commonly presents as a dry mouth. The diagnosis of salivary gland disease relies mainly on the clinical presentation. Salivary gland scan is useful in detecting cause of swelling in the major salivary glands, differentiate benign and malignant lesions, and to find the cause of dry mouth. Quantitative analysis of ^{99m}Tc -pertechnetate salivary gland scintigraphy has been used in the evaluation of salivary gland function and is a noninvasive technique.^{18,36}

The patient is positioned under a gamma scintillation camera that detects radiation. Immediately after the injection, imaging begins. For accurate results, the patient must stay still during imaging. After several images, the patient is given lemon drop candies to suck on, which stimulate the salivary glands. Another set of images is made for comparison purposes. The entire process takes about ten minutes for the injection and 30-45 minutes for the scan.^{4,34}

Salivary scintigraphy has been useful for investigation of various diseases affecting the salivary glands.²¹ In Sjögren's syndrome, pertechnetate imaging can demonstrate the severity of salivary gland involvement.^{1,2,3,8} In clinical situations, like iatrogenic irradiation of the salivary glands for therapy of head and neck tumors or radioiodine treatment of thyroid cancer, salivary scintigraphy helps to assess functional damage.^{2,32,39,42} Also helpful in monitor recovery in these patients. Salivary scintigraphy has been refined toward providing quantitative information on changes in gland function after parenchymal insult, due to inflammation or radiation. Salivary scintigraphy would provide an objective means of diagnosis. It a reproducible tool for follow-up of salivary function impairment.^{13,17,18,32}

The TMJ is a synovial joint, diseases and disorders that affect other parts of the musculoskeletal system can also affect the TMJ. Internal derangement, degenerative joint disease (arthrosis), inflammatory arthritis and infection all are detected in TMJ scintigraphy.² The initial examination used to image TMJ symptoms is usually plain radiographs/conventional tomography. The arthritic changes and congenital bone abnormality are visualized fairly well on plain films. The radionuclide imaging using technetium Tc 99m methylene diphosphonate/hydroxymethylene diphosphonate (^{99m}Tc MDP/HMDP) and single-photon emission computed tomography (SPECT) using ^{99m}Tc MDP/HMDP have been found to be sensitive techniques in the diagnosis of TMJ disorders.^{2,10,26}

Imaging allows accurate staging of internal derangement for operative planning. TMJ arthrography implies radiographic imaging after introduction of radiographic contrast into the upper or lower or both joint spaces of the TMJ and is indicated for evaluating soft tissue components, especially disk position, morphology and to evaluate disk function during opening and closing movements. If the disk is displaced and reduces on jaw opening, the dynamic events are depicted more clearly with fluoroscopic analysis.^{43,45} The smooth to-and-fro flow of contrast material from the anterior recess in the closed-jaw position to the posterior recess in the open-jaw position indicates normal function. A continual collection of contrast material in the anterior recess of the lower joint compartment and progressive deformity with jaw opening help to confirm the diagnosis of disk displacement. Perforations are detected easily during initial filling of the joint by observing contrast material flow from the inferior compartment to the superior

compartment.

Arthrography can be performed as a single-contrast examination in which iodinated contrast is injected into one or both of the TMJ spaces, or as double-contrast arthrography in which the injection of iodinated contrast is combined with a gas contrast medium. Single-contrast lower-compartment arthrography is better for demonstrating joint dynamics. Double-contrast dual-space arthrography better demonstrates anatomic features of the joint, such as shape of the joint spaces and configuration of the disk in its different mediolateral sections.

A more recent development is Positron Emission Tomography (PET), using isotopes produced in a cyclotron. A positron-emitting radionuclide is introduced, usually by injection, and accumulates in the target tissue. As it decays it emits a positron, which promptly combines with a nearby electron resulting in the simultaneous emission of two identifiable gamma rays in opposite directions. These are detected by a PET camera.³⁵

PET's, with radiopharmaceutical fluorine-18-2-fluoro-2-deoxyglucose (FDG), as the tracer, most accurate non-invasive method of detecting and evaluating most cancers and also used in cardiac and brain imaging. The development of radiopharmaceuticals like FDG made it easier to study living beings, and to diagnose and evaluate the effect of treatment on human disease. During the 1980s the technology that underlies PET advanced greatly. Commercial PET scanners were developed with more precise resolution and images. Over the last several years, the major advance in this technology has been the combining of a CT scanner and a PET scanner in one device. The modern PET/CT scanner allows a study to be done in a shorter amount of time but still provides more diagnostic information.^{10,31,33}

It is often performed studies for brain imaging by single photon emission tomography (SPECT) using thallium, or sestamibi, tetrofosmin, and positron emission tomography using flurodeoxyglucose as a radiotracer. Tumoral lesions and normal brain tissue have different uptake properties for these tracers. ^{99m}Tc glucoheptonate (^{99m}Tc -GHA) used as an early brain SPECT tracer.^{37,40} Its accumulation in the tumors could be attributed to disruption of the blood-brain barrier caused by the tumor, rather than active extraction of the tracer in relation to tumor metabolism.⁴¹ Newly introduced tracers, such as 201Thallium and technetium-based thallium analogs were shown to accumulate in viable myocardium and became used for brain tumor imaging. Their uptake was independent of blood-brain barrier disruption. However, it has been suggested that disruption of the blood-brain barrier is a necessary condition for the uptake of any tumor seeking agent. Plain pertechnetate, whose uptake depends on disruption of the blood-brain barrier by the tumor. ^{99m}Tc -GHA is more economic than tetrofosmin, sestamibi or thallium, and is easily radiolabeled with technetium (^{99m}Tc) in a standard nuclear medicine pharmacy.

PET is considered particularly effective in identifying the presence of cancer, its spread and response to treatment. PET is used for detecting cancers of lung, head and neck, colorectal, esophageal, lymphoma, melanoma, breast, thyroid, cervical, pancreatic, and brain.^{7,25} In early detection PET images can accurately characterize a tumor as benign or malignant. This help in avoiding surgical biopsy, confirmation of distant metastasis that can alter treatment plans. It determines the full extent of disease, especially in lymphoma, malignant melanoma, breast, lung, colon and

cervical cancers. To check and staging and recurrences of cancer it is considered being the most accurate diagnostic procedure. It also differentiates tumor recurrences from radiation necrosis or post-surgical changes. Such an approach allows for the development of a more rational treatment plan for the patient. Assessing the effectiveness of chemotherapy, the level of tumor metabolism is compared on PET scans taken before and after a chemotherapy cycle.

Conclusion

Nuclear medicine is safe because the radioactive tracers, or radiopharmaceuticals, used are quickly eliminated from the body. And these tracers rapidly lose their radioactivity. In most cases, the dose of radiation necessary for a scan is very small. The development of specific tumor seeking radiopharmaceuticals is one of the most promising areas of research in nuclear medicine. The potential for detecting small foci of tumor and perhaps indicating the malignancy to therapy is the goal of many current investigations.

Bibliography

- Aung W, Murata Y, Ishida R, Takahashi Y, Okada N, Shibuya H. Study of quantitative oral radioactivity in salivary gland scintigraphy and determination of the clinical stage of Sjögren's syndrome. *J Nucl Med.* 2001;42:38-43.
- Baur, D. A., T. F. Heston, and J. I. Helman. "Nuclear Medicine in Oral and Maxillofacial Diagnosis: A Review for the Practicing Dental Professional." *Journal of Contemporary Dental Practice* 5 (February 15, 2004): 94-104.
- Bohuslavizki KH, Brenner W, Tinnemeyer S, et al. Quantitative salivary gland scintigraphy derived from 166 normals. *Radiol Oncol.* 1995;29:297-305.
- Chae, S. W., J. H. Sohn, H. S. Shin, et al. "Unilateral, Multicentric Warthin's Tumor Mimicking a Tumor Metastatic to a Lymph Node. A Case Report." *Acta Cytologica* 48 (March-April 2004): 229-233.
- Chang LT. A method for attenuation correction in radionuclide computed tomography. *IEEE Trans Nucl Sci NS* 25: 638-643.
- Choi JY, Kim SE, Shin HJ, Kim BT, Kim JH. Brain tumor imaging with ^{99m}Tc-tetrofosmin: comparison with ²⁰¹Tl, ^{99m}Tc-MIBI, and ¹⁸F-fluorodeoxyglucose. *J Neurooncol*; 46: 63-70; 2000.
- Citrin DL, Tormey DC, Carbone PP. Implications of the ^{99m}Tc diphosphonate bone scan on treatment of primary breast cancer. *Cancer Treat Rep* 1977; 61:1249-1252.
- Demangeat R, Didon-Poncelet A, Cherfan J, Demangeat JL. Stimulated salivary pertechnetate clearance revisited: correlation with dynamic scintigraphic indices in Sicca syndrome. *Clin Nucl Med.* 2000;25:888-894.
- Dooms GC, Hecht S, Brant-Zawadzki M, et al. Brain radiation lesion: MR imaging. *Radiology* 158: 149-155; 1986.
- Even-Sapir E, Martin RH, Barnes DC, Pringle CR, Iles SE, Mitchell MJ. Role of SPECT in differentiating malignant from benign lesions in the lower thoracic and lumbar vertebrae. *Radiology* 1993; 187:193-198.
- Galasko CSB. The pathological basis for skeletal scintigraphy. *J Bone Joint Surg Br* 1975; 57:353-359.
- Genant HK, Bautovich GJ, Singh M, Lathrop KA, Harper PV. Bone-seeking radionuclides: an in vivo study of factors affecting skeletal uptake. *Radiology* 1974; 113:373-382.
- Hermann GA, Vivino FB, Shnier D, Krumm RP, Mayrin V. Diagnostic accuracy of salivary scintigraphic indices in xerostomic populations. *Clin Nucl Med.* 1999;24:167-172.
- Hoffman, D. C.; Ghiorso, A.; and Seaborg, Glenn T., eds. (2000). *The Transuranium People: An Intimate Glimpse*. London: Imperial College Press
- Kaplan WD, Takvorian T, Morris JH, Rumbaugh CL, Conndly BT, Atkins HL. Thallium-201 brain tumor imaging. A comparative study with pathological correlation. *Journal of Nuclear Medicine* 28: 47 - 52; 1987
- Kim KT, Black KL, Marciano D, Mazziotta JC, Guze BH, Grafton S, Hawkins RA, Becker DP. Thallium-201 SPECT imaging of brain tumors: methods and results. *J Nucl Med.* 1990 Jun; 31(6):965-969.
- Klutmann S, Bohuslavizki KH, Kroger S, et al. Quantitative salivary gland scintigraphy. *J Nucl Med Technol.* 1999;27:20-26.
- Kohn WG, Ship JA, Atkinson JC, Patton LL, Fox PC. Salivary gland ^{99m}Tc-scintigraphy: a grading scale and correlation with major salivary gland flow rates. *J Oral Pathol Med.* 1992;21:70-74.
- Lee HB, Blafox MD. Mechanism of renal concentration of technetium-^{99m}-glucoheptonate. *Journal of Nuclear Medicine* 26: 1308-1313; 1985.
- Levielle J, Pision C, Karakand Y et al. Technetium-^{99m} glucoheptonate in brain tumor detection: an important advance in radiotracer technique. *Journal of Nuclear Medicine* 18: 957-961;1977
- Liem IH, Olmos RA, Balm AJ, et al. Evidence for early and persistent impairment of salivary gland excretion after irradiation of head and neck tumours. *Eur J Nucl Med.* 1996;23:1485-1490.
- Mari C, Catafau A, Carrio I. Bone scintigraphy and metabolic disorders. *Q J Nucl Med* 1999; 43:259-267.
- McAfee JG, Reba RC, Majd M. The musculoskeletal system. In: Wagner HN, Jr, Szabo Z, Buchanan JW, eds. *Principles of nuclear medicine*. 2nd ed. Philadelphia, Pa: Saunders, 1995; 986-1012.
- Morrissey, D.; Loveland, W. T.; and Seaborg, Glenn T. (2001). *Introductory Nuclear Chemistry*. New York: John Wiley & Sons.
- Nishiyama Y, Yamamoto Y, Fukunaga K, Satoh K, Kunishio K, Ohkawa M. Comparison of ⁹⁹Tcm-MIBI with ²⁰¹Tl chloride SPET in patients with malignant brain tumours. *Nucl Med Commun* 22: 631-9; 2001.
- Pauwels EKJ, Feitsma RJ. Radiochemical quality control of ^{99m}Tc-labeled radiopharmaceuticals. *Eur J Nucl Med* 2: 97; 1977.
- Ramanna L, Waxman A, Binney G, Waxman S, Mirra J, Rosen G. Thallium-201 scintigraphy in bone sarcoma: comparison with gallium-67 and technetium-MDP in the evaluation of chemotherapeutic response. *J Nucl Med.* 1990 May; 31(5):567-572.
- Rausch JM, Resnick D, Goergen TG, Taylor A. Bone scanning in osteolytic Paget's disease: case report. *J Nucl Med* 1977; 18:699-701.
- Roland Muller-surr. Radiopharmaceuticals: their intrarenal handling and localization. In: *Nuclear medicine in clinical diagnosis and treatment* Vol. 1. New York, US Churchill Livingstone, 1994.
- Rydberg, J.; Liljenzin, J.-O.; and Choppin, Gregory R. (2001). *Radiochemistry and Nuclear Chemistry*, 3rd edition. Woburn, MA: Butterworth-Heinemann.
- Savelli G, Maffioli L, Maccauro M, De Deckere E, Bombardieri E. Bone scintigraphy and the added value of SPECT (single photon emission tomography) in detecting skeletal lesions. *Q J Nucl Med* 2001; 45:27-37.
- Shannon IL, Suddick RP, Dowd FJ Jr. Saliva: composition and secretion. *Monogr Oral Sci.* 1974;2:1-103.
- Shaw E, Arusell R, Scheithauer B, et al. Prospective randomized trial of low-versus high-dose radiation therapy in adults with supratentorial low-grade glioma: initial report of a North Central Cancer Treatment Group/Radiation Therapy Oncology Group/Eastern Cooperative Oncology Group study. *J Clin Oncol* 20: 2267-76; 2002.
- Silvers AR, Som PM. Salivary glands. *Radiol Clin North Am.* 1998;36:941-966.
- Sklar CA. Childhood brain tumors. *J Pediatr Endocrinol Metab* 15 Suppl 2:669-73; 2002
- Solans R, Bosch JA, Galofre P, et al. Salivary and lacrimal gland dysfunction (sicca syndrome) after radioiodine therapy. *J Nucl Med.* 2001;42:738-743.
- Soricelli A, Cuocolo A, Varrone A, et al. Technetium-^{99m}-Tetrofosmin uptake in brain tumors by SPECT: Comparison with Thallium-201 imaging. *Journal of Nuclear Medicine* 39: 802-806; 1998.
- Staudenherz A, Fazeny B, Marosi C, et al. Does (^{99m}) Tcsestamibi in high-grade malignant brain tumors reflect blood-brain barrier damage only? *Neuroimage* 12: 109-11; 2000.
- Stuchell RN, Mandel ID. Salivary gland dysfunction and swallowing disorders. *Otolaryngol Clin North Am.* 1988;21:649-661.
- Tanasescu D, Wolfstein R, Waxman AD. Technetium-^{99m}-glucoheptonate as a brain scanning agent. *Editorial Journal of Nuclear Medicine*18: 1037-1038; 1977.
- Utriainen M, Metsahonkala L, Salmi TT, et al. Metabolic characterization of childhood brain tumors: comparison of ¹⁸Ffluorodeoxyglucose and ¹¹C-methionine positron emission tomography. *Cancer*.95:1376-86; 2002.
- Vigh L, Carlsen O, Hartling OJ. Uptake index and stimulated salivary gland response in ⁹⁹Tcm-pertechnetate salivary gland scintigraphy in normal subjects. *Nucl Med Commun.* 1997;18:363-366.
- Wagner, HN. *Nuclear Medicine: The Road to Smart Medicine and Surgery*. JNM August 1999; 39; 8:13N-34N.
- Waxman AD, Tanasescu D, Siemsen JK et al. Technetium-^{99m}- glucoheptonate as a brain screening agent. *Journal of Nuclear Medicine* 17; 345-348;1977.
- Wellman HN, Schauwecker D, Robb JA, Khairi MR, Johnston CC. Skeletal scintimaging and radiography in the diagnosis and management of Paget's disease. *Clin Orthop* 1977; 127:55-62.

The concept of working width - A forgotten dimension

Abstract

The causes of endodontic failure are multifaceted and endure due to an abundance of misinformation, misconceptions, and perpetuated endodontic myths. Spectacular change is occurring in clinical endodontics and is driven by an explosion of new technologies, instruments, materials, and the emergence of new techniques. Mismanagement of the apical one-third during canal preparation which compromises three-dimensional obturation is one of the most frequent aspects that contribute to the failure. Understanding the current concepts and techniques of working width can minimize and reduce many of the irresolute failures of root canal treatment.

Key Words:

Working width, endodontic therapy, apical constriction, working length

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Introduction

Cleaning & shaping of the root canal comprises the most important phase of endodontic treatment. Clinicians primary concern is to thoroughly cleanse the root canal system mechanically & chemically removing microorganisms and their substances. Therefore the first step for successful treatment is an exact diagnosis of the root canal system and recognition of its variations. Many textbooks and much literature focus on canal instrumentation in terms of filing reaming and other instrument motion and always stress the importance of canal size. Without solid evidence, however it is still not clear how large is large enough. On the basis of micro-computed tomography and other morphometric studies it has been shown that the horizontal dimension of the root canal system is not only more complicated than the vertical dimension (working length) but also more difficult to investigate because the horizontal dimension varies greatly at each vertical level of the canal. Thorough instrumentation of the apical region has long been considered to be an essential component in the cleaning and shaping process. It was discussed as a critical step as early as 1931 by Groove.¹ Simon² later recognized the apical area as the critical zone for instrumentation. Other authors^{3, 4} concluded that the last few millimeters that approach the apical foramen are critical in the instrumentation process. Incidentally, this area of the canal (coronal to the apical constriction) was called, and with good reason, the "Forgotten Dimension"⁵ by Carl Hawrish. This article provides perspectives on the current concepts and techniques to handle WW (Working Width), the horizontal dimension of the root canal system) and its clinical implications.

Canal anatomy

Computer tomography has made visualizing canal systems much simpler task. We've learned that nearly every canal is curved. What may appear as a straight canal in a two-dimensional X-ray almost always has some degree of curvature in an unseen plane. The apical constriction has been carefully examined by a number of authors. Both Kuttler⁶ and Mizutani et al.⁷ showed irregularities in the shape of the cementodentinal junction. These shapes have been described as oval, long oval, ribbon shaped, or round Drummer et al.⁸ has shown the apical constriction to be irregular in a longitudinal direction as well. Twenty-five percent of the apical constrictions in teeth evaluated by Wu⁽⁹⁾ had long oval shapes. In fact, the data demonstrated that the CEJ of

most teeth were never completely round, but tended to be oval. Furthermore, the cross-sectional shape of most canals is not round but oval (mimicking the oval shape of most roots). Lastly, few canals have a constant taper; instead, they exhibit nearly parallel walls in multiple segments throughout the length of the canal. So, a root canal with a graceful, tapering and a single apical foramen ending at the apical foramen is the exception rather than the rule.

Diameter of the Apical Constriction

The apical minor diameter, or minor constriction, is best visualized by studying cross-sections of apical canals as described by Wu et al.⁽⁹⁾ In his classic study he demonstrated the average initial narrow diameter at the apical constriction ranges from 0.3 to 0.4 mm. However just coronal to the apical constriction canal diameters increase significantly, ranging from 0.35 to 1.00 mm and higher.⁽⁵⁾ The clinician should consider introducing a non tapered instrument to working length after coronal flaring because determination of the initial narrow apical canal diameter plays a major factor in identifying the extent of final apical shaping. Because the first non-tapered instrument that binds the apical constriction is larger than the corresponding tapered instrument, it better reflects the actual narrow apical diameter of the canal.

Working width

Working Width relates to canal diameter coronal to the apical constriction. It was first used by Dr. Jou from the University of Pennsylvania. Working Width (WW) is best understood by studying cross-sections of apical canals.¹⁰ Oval canals have two diameters, a minor (smaller) and a major (larger) diameter. The quality of cleaning is dependant on instrumenting to the larger diameter; it's Working Width (*Fig. 1*) If the greater diameter of the original canal is measured, the correct WW is an instrument size slightly larger than that dimension.¹¹

Determination of initial working width at working length

One common method of deciding on the size of apical preparation is to first determine the pre-operative canal diameter by passing consecutively larger instruments to the working length until one binds. This initial apical file estimation is referred to as the determination of MinI WW0 (minimal initial horizontal dimension at working length)¹⁰. The master apical file size Max FWWO (maximal final horizontal dimension at working length) is then to be decided.¹⁰ In the past two guidelines were considered sufficient for instrumentation.

- 1) Enlarge root canal at least three ISO file sizes larger than the initial binding file.
- 2) Enlarge the canal until clean, white dentinal shavings appear in the flutes of the instrument blade.¹²

Both these guidelines cannot be recommended in all cases. The color of dentinal shavings is no indication of the presence of infected dentin or organic debris. Root canals should be enlarged regardless of initial width, to remove irregularities of dentin and to make walls of the

canal smooth and tapered. However, there is no evidence that the instrument which binds first actually reflects the diameter of the canal in the apical region. Thus, the concept of widening the apical canal to three sizes larger than the first file to bind is not based on evidence. Gutierrez and Garcia¹³ showed canals are often improperly cleaned. They attributed inadequate instrumentation to the root canal diameter being larger than the instrument size used for calibration of the initial canal size in each individual case. Ideally, the minimum size to which root canal should be enlarged cannot be standardized and varies from case to case. Recent studies¹⁴ suggest that the first K file and the first light speed instrument that bound at the working length did not actually reflect the diameter of the apical canal. The inaccuracy and discrepancy can come from various morphologic and procedural factors such as canal shape, canal length, coronal interference and the instruments used.

Canal shape

Cross sections at various revealed that root canal anatomy varied from round to oval or triangular. The round canal can be measured more easily because the minimal initial working width and maximal initial working width are the same, but this is not the case with oval root canal. The proper instrument and tactile sensation may determine the Min IWW (minimal initial working width) of the oval, long-oval and the flat canals.

Canal length

When using an instrument to gauge working length, the longer the canal, the greater the frictional resistance. In a very long canal, the frictional resistance may increase to affect the clinician's tactile sense for determining the initial working width correctly. In addition, if the coronal flare is too limited to the coronal third of the canal, then shaft of the instrument may engage the canal wall and cause false/premature conclusion as to the WW.

Canal taper

Most canals do not have a constant taper. In many cases canal walls are parallel in some segments of the canal. This shape does not always lend itself to being instrumented to a tapered form. So it can be inferred that canal instrumentation should closely mimic the original anatomy from orifice to working length. Any tapering discrepancy between the gauging instrument and canal may lead to an early instrument engagement of the canal wall, causing a false sensation of the apical binding. Early coronal flare can increase the taper of the canal and reduce the tapering discrepancy between the gauging instrument and canal wall.

Canal curvature

Most canals are curved in one or more directions. The more severe a curve, the more difficult the treatment. Curved canals can cause deflection of the gauging instrument and increase the frictional resistance. The curvature of the root canal can be categorized into two-dimensional, three dimensional, small radius, large radius, and double curvature (S-shaped, bayonet

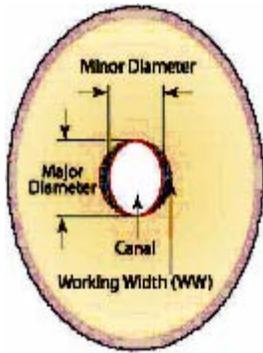


Fig. 1a: In a pure oval canal there are two dimensions, major diameter and minor diameter

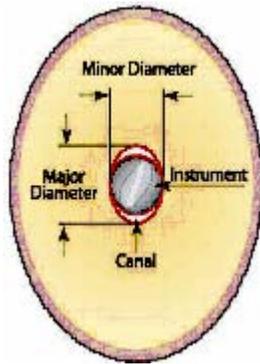


Fig. 1b: An instrument that cuts just at the minor diameter leaves a lot of wall untouched.

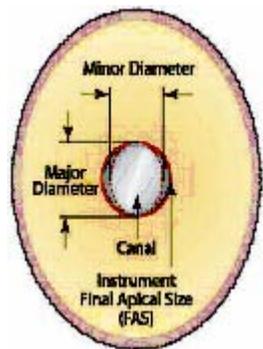
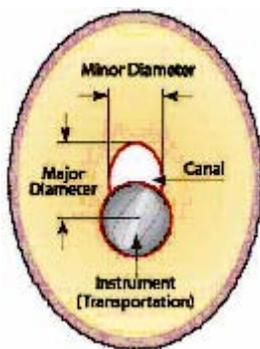


Fig. 1c&d: Instruments must be the right size and be flexible enough to stay centered in the canal. A correct instrument size can do more than good if it isn't flexible and canal transportation occurs.



shaped) and with different degrees of severity. Each of these curvatures has a different effect on a clinician's tactile sense. The combinations of these curvatures make correct determination of the IWW (initial working width) extremely difficult, if not impossible.

Canal content

The content of the root canal may be fibrous in nature. Calcified material may also be part of the canal content. During determination of the working width, the mixed canal contents can create different degrees of frictional resistance against the gauging instrument. It can eventually affect the clinician's tactile sense.

Canal wall irregularities

It has been advocated that continuous & progressive dentin formation on pulp chamber floor creates dentin projections that narrow the canal diameter, especially at the cervical third. Attached pulp stones, denticles and reparative dentin can create convexities on the canal wall surface. Resorption can produce concavities on the canal wall surface. These phenomena can serve as an impacting factor that induces a false estimation of the true canal dimension at working length and other levels.

Minimizing the influence of affecting factors

Before the initial working width determination, it is suggested to widen the orifices to do early coronal flaring and additional canal flaring to ensure effective irrigation and to minimize any interferences with tactile sensation. Carefully selecting the adequate instrument of maximal flexibility and minimal taper such as light speed may avoid interferences and help to achieve better results. Ideally, root canal preparation should follow the exact horizontal dimension of the root canal at every level of the canal. In this ideal condition, especially for long-oval and flattened root canals, they can be cleaned and shaped properly with minimal mishaps of weakening, stripping or perforating the canal. Recent studies^(15,16,17) have indicated that no current instrument technique was able to completely clean dentin walls of the oval, long oval, and flattened root canals.

Instrument for determining the initial working width

The rigidity, flexibility and tapering of the instrument used for determining initial working width can affect accuracy. The selection of the first instrument to fit the apical constriction is achieved by tactile sensation, which is possible only after coronal flaring. Any tapering discrepancy between the gauging instrument and canal may lead to an early instrument engagement of the canal wall altering the tactile sensation. Instruments with a large taper might give a resistance sensation if it comes in contact with the canal walls in the coronal portion of the canal without any contact between the instrument and the canal walls in the more critical apical portion of the canal. In addition, the rigid instrument in a curved canal also lead to a false tactility. If the greater diameter of the original canal is measured, the correct WW is an instrument size slightly larger than that dimension.

Determination of minimal and maximal final working width at working length

The literature shown that root canal systems need to be enlarged sufficiently to remove debris and to allow proper irrigation to the apical third of the canal. Research has shown that canals need to be enlarged to at least #35 file for adequate irrigation to reach the apical third⁽¹⁸⁾. Ram⁽¹⁹⁾ concluded that canals need to be enlarged to a #40 file size so that maximum irrigation is in contact with the apical debris. When smaller files were used, debris was not flushed out by irrigation. Chow⁽²⁰⁾ demonstrated that the canal system had to be instrumented to at least #40 file for proper irrigation. Shuping et al. and Siqueira et al.^(21,22) later confirmed the findings that larger file sizes are needed to allow the irrigating solution to reach the apex. Larger instrumentation sizes not only allow proper irrigation but also significantly decrease remaining bacteria in the canal system

Any investigation of the effectiveness of cleaning the root canal system without carefully estimating the minimal and maximal initial working width in the oval, long oval, and flattened root canals may result in misleading data,

especially if the horizontal canal morphology was not carefully assessed. In an oval, long oval, or flat canal, circumferential instrumentation seems to be the only reasonable way to properly clean and shape the canal. Ideally, during root canal preparation, the instruments and techniques used should always conform to and retain the original shape of the canal to maximize the cleaning effectiveness and minimize unnecessary weakening of the tooth structure to achieve the optimal result. Clinically the heavily infected cervical part of the canal has often been enlarged with Gates-Glidden burs or canal wideners to a round shape instead of following the original oval, long-oval or flat shape.

Several guidelines were developed to determine the minimum final working width at the working length. The maximal discrepancy between the maximum final working width and minimum final working width can be six to eight ISO sizes. Complicated by the canal curvature, the instrument used and the techniques implemented, the concepts for determining the final working width seems to be unclear and chaotic. Between the cervical and apical areas, the clinician has the absolute freedom to determine the minimum final working width and the maximum final working width at N mm from working length because the scientific information and evidence are not available yet.

Conclusion

There has been minimal development of concepts, techniques, and technology to measure initial WW and to determine final WW accurately or properly. Understanding the current concepts and techniques of WW can reduce the underestimation of the minimal initial WW and subsequent incomplete cleaning of the root canal system. The detailed information regarding horizontal morphology of the root canal system can help to solidify concepts and improve techniques of cleaning and shaping the root canal system. Carefully maintaining the aseptic chain, using adequate irrigating solutions to enhance efficacy, and cautiously applying current concepts and techniques of WW may provide a better quality of endodontic therapy for the patient.

References

- Grove CJ. The value of the dentinocemental junction in pulp canal surgery. *J Den Res* 1931;11:466-8.
- Simon J. The apex: how critical is it? *Gen Dent* 1994;42:330-4.
- Kasmer Kerekes and Leif Tronstad. Morphometric observations on root canals of human premolars. *J Endodon* 1977; 3: 74-9.
- Kasmer Kerekes and Leif Tronstad. Morphometric observations on the root canals of human molars. *J Endodon* 1977; 3: 114-8.)
- E. Steve Senia. Canal Diameter: The Forgotten Dimension. *Dentistry Today* 2001 (May); 20: 58-62.
- Kuttler Y. Microscopic investigation of root apices. *J Am Den Assoc* 1955;50:544
- Mizutani T, Ohno N, Nakamura H. Anatomical study of the root apex in the maxillary anterior teeth. *J Endod* 1992;18:344-7.
- Dummer PMH, McGinn JH, Rees DG. The position and topography of the apical canal constriction and apical foramen. *Int Endod J* 1984;17:192-8.
- Wu MK, R'oris A, Barkis D, Wesselink PR. Prevalence and extent of long oval canals in the apical third. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2000; 89:739-43.
- Jou YT, Karabucak B, Levin J et al. Endodontic working width: current concept and techniques. *Dent Clin North Am* 2004; 48: 323-5.
- E. Steve Senia, Endodontic success: It's all about the apical third *Endo tribune* 1 march 2008
- Grossman L. *Endodontic Practice*. 10th edition. Philadelphia: Lea & Febiger; 1986
- Gutierrez JH, Garcia J. Microscopic and macroscopic investigation on results of mechanical preparation of root canals. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1968; 25:108-16.
- Wu MK, Barkis D, Roris A, Wesselink PR. Does the first file to bind correspond to the diameter of the canal in the apical region? *Int Endodon J* 2002;35(3):264-6
- Liu DT, Jou YT. A Technique estimating apical constricture with K files and NT Light speed rotary instruments. *J Endodon* 1999;25(4):306
- Weiger R, Lost C. Efficiency of hand and rotary instruments in shaping oval root canals. *J Endodon* 2002;28(8):580-3
- Barbizam JVB. Effectiveness of manual and rotary instrumentation techniques for cleaning flattened root canals. *J Endodon* 2002;28(5):365-6
- Salzgeber RM, Brilliant JD. An in vivo evaluation of the penetration of an irrigating solution in root canals. *J Endod* 1977;3:394-8.
- Ram Z. Effectiveness of root canal irrigation. *Oral Surg* 1977;44:306-12.
- Chow T. Mechanical effectiveness of root canal irrigation. *J Endod* 1983;9:475-9.
- Shuping G, Orstavik D, Sigurdsson A, Trope M. Reduction of intracanal bacteria using nickel-titanium rotary instrumentation and various medications. *J Endod* 2000;26:751-5.
- Siqueira J, Lima K, Magalhaes F, Lopes H, de Uzeda M. Mechanical reduction of the bacterial population in the root canal by three instrumentation techniques. *J Endo* 1999; 25:332-5.

Temporary anchorage devices in orthodontics

Abstract

Temporary skeletal anchorage is a promising new field in orthodontics and already a wide variety of skeletal anchorage devices are available commercially. This review aims to assist clinicians by outlining the principles of bone anchorage and the salient features of the available systems, especially those that may influence the choice of a specific Temporary Anchorage Device (TAD) for anchorage reinforcement.

Key words: Orthodontic anchorage, orthodontic implants, mini-implants, mini-screws, mini-plates, temporary anchorage devices.

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Introduction

Orthodontic anchorage control is a very important part of orthodontic treatment planning and subsequent treatment delivery. Research has focussed on the efficient movement of teeth to minimize anchorage loss by improvements in orthodontic materials, bracket designs (e.g. self-ligating brackets or Tip-Edge) and friction-less treatment protocols (e.g. segmented arch technique, loop mechanics). The methods used to reinforce orthodontic anchorage traditionally involve the use of extra-oral (headgear) and intra-oral (transpalatal arch, Nance palatal button etc.) appliances. However, it is recognized that these conventional anchorage systems are limited by multiple factors such as patient compliance, the relative number of dental anchorage units, periodontal support, iatrogenic injuries and unfavourable reactionary tooth movements.

In recent years, numerous publications have introduced novel ways of reinforcing anchorage using a variety of devices temporarily anchored in bone. Orthodontic bone anchorage is indicated when a large amount of tooth movement (e.g. labial segment retraction or mesial/distal movement of multiple posterior teeth) is required or dental anchorage is insufficient because of absent teeth or periodontal loss. Such devices may also be useful in asymmetric tooth movements, intrusive mechanics, intermaxillary fixation/traction and orthopaedic traction and appear to be rapidly gaining acceptance in routine orthodontic practice.

There is no clear consensus regarding the nomenclature of these devices and are interchangeably used by various authors. The confusing array of names include mini-implants,¹ micro-implants,² microscrew implants,³ miniscrews⁴ temporary anchorage devices (TADs). and Bone anchorage devices (BADs)

In view of the rapidly evolving and complex nature of this topic, this paper aims to assist the orthodontist by reviewing the various design features of currently available temporary anchorage devices and outlining principles of skeletal anchorage and the clinically relevant factors that influence the choice of a specific TAD.

Types of Skeletal anchorage

There are three distinctly different approaches to bone anchorage in terms of the devices' origin and characteristics (Figure 1). Broadly speaking, TADs can either be osseointegrated or mechanically retentive depending on their bone-endosseous surface interface and design features. The latter group can be subdivided according to whether the screw (mini-implant) or plate (mini-plate) components are the principal design elements.

A) Orthodontic implants

The first widely available means of bone anchorage evolved from Branemark's⁵ work on the concept of osseointegration and use of titanium implants to replace missing teeth. These endosseous implants have features to promote both functional and structural integration (osseointegration) at the implant-bone interface, and require an unloaded latency period of up to 6 months.⁵ In 1984, Roberts *et al.*⁶ investigated the tissue response to orthodontic forces applied to restorative implants and concluded that continuously loaded implants remained stable with 100 g force after a 6-week healing period. In a follow-up study on dog mandibles, osseointegration was found in 94% of the implants and it was concluded that less than 10% of endosseous surface area contact with bone was needed to resist forces of up to 300 g for 13 weeks. Subsequently, several manufacturers modified restorative implant designs to produce customized orthodontic fixtures. Clinical studies on the use of osseointegrated implants for orthodontic anchorage have reported a success rate of 86–100%.⁷ The retromolar implants,⁸ Onplant, Straumann Orthosystem and Mid-plant systemTM are examples of osseointegrated BADs.

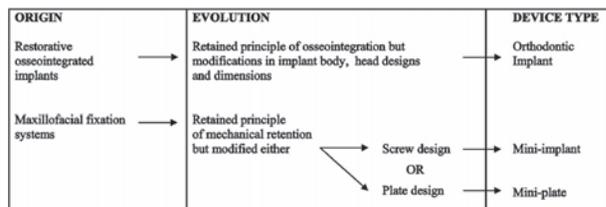


Fig 1 – types of TADs

B) Mini-implants and mini-plate systems

Orthodontic mini-implant and mini-plate systems are derived from maxillofacial fixation techniques and rely on mechanical retention for anchorage (Figure 1). Since these devices use osseous physical engagement for stability, they are less technique sensitive than osseointegrated implants, amenable to immediate orthodontic loading and are easily removed. Osseointegration is neither expected nor desired (in terms of screw removal), although animal studies have demonstrated that a limited and variable level (10–58%) of osseointegration can occur. In 1983, Creekmore and Eklund⁹ reported the use of a vitallium screw, resembling a bone-plating screw, placed in the anterior nasal spine region. This was loaded after 10 days for successful intrusion of the adjacent upper incisors. Subsequent modifications to the design of fixation screws have made them more suitable for use in orthodontics and led to the introduction of customized **mini-implant** kits. In the late 1990s, both Kanomi *et al.*¹ and Costa *et al.*¹⁰ described mini-implants specifically designed for orthodontic use. The Aarhus, Spider screw, Dual Top, Absoanchor and IMTEC are current examples of mini-implant type temporary anchorage devices. (fig 2)

C) Mini Plate

Over the same period, alterations to the design of maxillofacial fixation plates have led to the introduction of **mini-plate systems**. In 1985, Jenner *et al.*¹¹ reported a clinical case where maxillofacial bone plates were used for orthodontic anchorage. In 1998, Umemori *et al.*¹² used L-shaped Leibinger mini-plates in the mandible to intrude molars for anterior open bite correction. They termed this approach the 'The Skeletal Anchorage System' (SAS) and suggested that, when compared with osseointegrated implants, these mini-plates provide stable anchorage with immediate loading. Since then other mini-plate design variations have been introduced, e.g. Bollard Mini Plate implant and C-tube implant (Figure 3). Clinical studies on these non-integrating devices have reported success rates of 86–93% for mini-implants and 93% for mini-plates.

Design Features of Temporary Anchorage Devices

There are several features common to all osseointegrated implants and mini-implants (Figure 4) and therefore these are described together. Mini-plate design features however will be described separately.

Material specifications

Although manufacturers do not give detailed material specifications, most TADs are made of pure titanium or titanium alloy. Titanium has proven properties of biocompatibility, is light weight, has excellent resistance to stress, fracture and corrosion, and it is generally considered to be the material of choice. Surgical grade stainless steel has also been used for Leone mini-implants and SK mini implants. During their manufacture

orthodontic implants undergo a variety of surface alterations to promote osseointegration, e.g. the sand-blasted and acid-etched (SLA) endosseous surface of the Orthosystem. Mini-implants on the other hand are manufactured with a smooth endosseous surface or additional surface treatments (e.g. TOMAS system) to actively discourage osseointegration and therefore simplify their removal.

Dimensions

Orthodontic implants and mini-implants are available in a range of body lengths and diameters. For **orthodontic implants** both physical stability and osseointegration depend on adequate bone-fixture surface contact, which in turn is a balance between the fixture's diameter and length. If the length is small the diameter must be large and vice versa. In practice, an implant's primary stability is related to its intra-osseous length, whilst the threads help to dissipate stress within the trabecular bone. Subsequently, the implant's shape and surface characteristics are important influences on osseointegration, as the load tolerance is proportional to the available osseointegrated surface area. Such orthodontic implants are usually cylindrical in shape (Figure 4) with a relatively short body length (4–7 mm) and large diameter (3–5 mm) as compared with mini-implants. These dimensions provide a large surface area in a limited depth of bone, making them suitable for mid-palatal, retro-molar and edentulous sites.

Conversely, **mini-implants** have long, narrow conical shapes (Figure 4) and are available in 6–15 mm intra-osseous lengths and in 1.2–2.3 mm diameters. An *in vitro* laboratory study has compared the mechanical properties of three types of mini-implants (Leone, M.A.S. and Dentos) on a non-biological bone substitute, and the authors concluded that mini-implants should be at least 1.5 mm in diameter in order to resist fracture.¹³ In terms of a mini-implant's primary stability, the diameter is more important than body length for mechanical interlocking in bone. If excess resistance is encountered during the placement of a mini-implant, it is preferable to first create a pilot hole using a drill whose diameter is less than the fixture body. For example, insertion of a 1.5 mm diameter mini-implant may warrant the use of a 1.1 mm diameter drill in the maxilla and 1.3 mm in the mandible, due to the differential bone density.

Body designs

Orthodontic implants and most mini-implants are commonly described as being self-tapping. Self-tapping body designs often have a special groove in their tip, which cuts or taps the bone during insertion. This feature usually requires a pilot hole to be drilled first and the groove at the tip then creates the thread pattern in bone as the fixture is inserted. Orthodontic implants have broadly similar self-tapping designs to improve the transfer of compressive forces to the adjacent bone, minimize micro-motion and increase the bone-implant surface area. For example, the Straumann Orthosystem relies on the physical shape of its threads to provide primary stability from the time of insertion until osseointegration subsequently occurs. Conversely, mini-



Fig 2 – mini implant screw



Fig 3- mini plate system

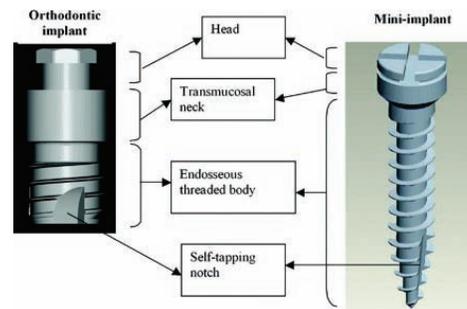


Fig 4- parts of a typical orthodontic and mini implant

implants have been manufactured with a wide variety of thread designs and body shapes. As with maxillo-facial fixation screws, the first mini-implants were tapped into pre-drilled holes. More recently, we have seen the release of self-drilling mini-implants, which can be screwed directly into bone using a driver at an appropriate torque level. This simplifies the insertion stage by avoidance of pre-drilling although some manufacturers indicate that their mini-implants behave in a self-drilling fashion in the maxilla, but may require pre-drilling in the mandible (e.g. IMTEC, Orlus).

Implant Head design

Orthodontic implants usually feature two-piece designs with specific healing abutments and intra-oral attachments. A healing cap or cover screw is usually placed during the latency phase and then replaced by specialized fixtures, which enable connection of orthodontic auxiliaries such as a transpalatal arch (TPA) for indirect anchorage. The majority of available mini-implants feature various one-piece designs. The C-Orthodontic system has a two-piece design, where the head is screwed on to the endosseous base either at insertion or after an apparent osseointegration period of 6–8 weeks.¹⁴ The IMTEC mini-implant system also has a detachable head abutment.

Mini-implant head designs may have hooks, ball ends or grooves to connect orthodontic traction auxiliaries or rectangular/round slots (bracket head designs). These slots have broadly similar dimensions to an orthodontic bracket and can be used to directly engage arch wires. In Bracket head type, two kinds of screws are available (in certain types) depending on the driving directions. Left Handed Screw should turn counter clockwise direction during driving. Depending on the direction of moment, we can choose Right or Left handed screws. The transmucosal neck is that part of the implant or mini-implant, which emerges through the soft tissue superficial to the cortical plate. A smooth polished transmucosal neck of appropriate height is essential to prevent plaque accumulation and harbouring of micro-organisms, and also provide sufficient clearance for the fixture head.

Mini-plate systems

Orthodontic mini-plate systems are broadly similar to maxillofacial plating systems (Figure 3) in terms of their holed baseplates and fixation screws, but have specifically modified ends to engage orthodontic auxiliaries. They are manufactured from titanium and are supplied in kits containing both mini-plates and fixation

screws. The designs may vary in shape and size, but are usually available as two- to five-holed mini-plates with transmucosal neck extensions. These plates are about 1.5 mm in thickness and can be bent or trimmed to adapt them to the cortical plate contour at the insertion site. They are secured with mono-cortical fixation screws of 5–7 mm lengths and 1.2–2.3 mm diameters. The intra-oral end is usually a cylindrical tube with holes through which orthodontic wires may be passed. A locking mechanism is integrated into the cylindrical tube, such that it can be tightened to stabilize the orthodontic wire or auxiliary.

Clinical aspects that influence the choice of a TAD

Thorough treatment planning is essential for the successful use of TADs to ensure a predictable outcome. The factors to be considered are;

- anchorage requirements of the case
- age of the patient
- potential insertion site morphology
- available bone (quantity and quality)

Anchorage specific steps include informed consent, selection of a suitable TAD, planning for accurate positioning, the surgical insertion procedure and biomechanical principles of force application. In addition to study models, a working model assists the orthodontist to plan treatment, identify insertion areas and prescribe a surgical stent. A panoramic radiograph, peri-apical radiographs, and a lateral cephalograph assist in the evaluation of available bone depth and the proximity of adjacent anatomical structures, and to confirm the positional details post-operatively. Some authors have suggested the use of CT scans to assess the bone morphology at potential sites for both orthodontic implants and mini-implants, but this is difficult to justify in routine clinical practice.

Anatomical placement site considerations

The most common sites for **orthodontic implants** are the mid-palatal region, para-median area of palate, and retromolar edentulous areas.⁸ For the anterior palate, bone depth can be assessed on a lateral cephalograph such that the antero-posterior location and inclination of the implant are planned to optimize the available bone depth. This allows for implants of up to 6 mm lengths to be placed in this region (Figure 5). Implants can also be inserted in para-median positions, i.e. 6–9 mm posterior to the incisive foramen and 3–6 mm laterally. This may be a valid option in young patients with a patent mid-palatal suture, although appropriate surgical and

radiological planning is essential. If there are any doubts over the degree of obliteration of the mid-palatal suture the implant should be placed just posterior to the first premolars where ossification is usually more complete.¹⁵

Mini-implants are much more versatile in terms of their potential anatomical sites because of their small diameters. Typical insertion sites are maxillary and mandibular buccal interproximal areas (Figure 6), the maxillary sub-nasal spine region, mandibular symphysis, para-median and mid-palate, retro-molar, infra-zygomatic and maxillary tuberosity areas. A volumetric CT study of 20 patients to assess the hard and soft tissue depths required for mini-implant insertion, indicated that 10 mm length screws could be placed in the symphysis and retro-molar regions and 4 mm lengths were preferable in the mid-palate area, incisive and canine fossae. In another study Poggio *et al.*¹⁶ assessed the interproximal alveolar sites in terms of the vertical insertion levels for mini-implants using 25 volumetric tomographic images of the maxilla and mandible.¹⁶ Mesio-distal and buccolingual distances were evaluated 2, 5, 8 and 11 mm from the alveolar crest. The results suggested that in both the maxilla and mandible, insertion in the buccal inter-premolar areas 5–11 mm from the alveolar crest would avoid damage to roots. The mean mesio-distal width of interproximal bone available was 3.5 mm in maxilla and 4.9 mm in mandible in this vertical range. In the maxilla maximum bone width was available on the palatal aspect of the alveolus; however, in the molar region insertion more than 8 mm from the alveolar crest should be avoided because of proximity to the maxillary sinus. In the interproximal sites, mini-implants should be angled at 30–40° to the vertical axis of teeth to enable insertion of longer ones in the available three-dimensional (3D) bone trough. Although not always necessary, if initial alignment is completed first then there may be more sites available for mini-implant placement through intentional separation of the adjacent roots during this treatment phase.¹⁷

Even when correctly inserted, it is important to be aware that mini-implants do not remain absolutely stationary, as was demonstrated in a clinical study of 16 patients with mini-implants inserted in the zygomatic buttress. When loaded over a period, these fixtures were displaced by 1 to 1.5 mm in the direction of the applied force. Interestingly, a histological animal study has assessed root repair after injury from mini-implant insertion and found that complete root repair occurred within 12 weeks of fixture removal.¹⁸ Finally, in long-term edentulous areas, implant and mini-implant placement should be carefully planned due to likely alveolar resorption and lowering of the maxillary sinus floor.

Recommended sites for the placement of **mini-plates** are the zygomatic process of the maxilla, mandibular body distal to the first molars¹² and the maxillary buccal plate above the premolar/molar roots. Whilst mini-plates may be placed in bony areas remote from the dental roots and important anatomical structures, their disadvantages include the large scale subperiosteal flap surgery necessary to access these remote sites and the associated patient morbidity. Their transmucosal part is adapted such that it emerges through the soft tissue at

an appropriate position and level for orthodontic auxiliaries to be attached.

Surgical stents and guides

The insertion techniques for all TADs should attempt to maximize the available bone volume, whilst avoiding adjacent anatomical structures such as dental roots, naso-maxillary cavities and neurovascular tissues. Clinical experience with palatal implants has shown that accurate 3D positioning is a critical factor in this respect.¹⁹ Several authors have recommended the use of removable stents for orthodontic implants to transfer the pre-surgical prescription to the surgical stage,²⁰

Some authors and manufacturers currently recommend an indirect planning technique for mini-implants, where a brass separating wire or a custom-made wire guide is placed between adjacent teeth and over the insertion site, or added to an adjacent fixed appliance bracket. These markers are then radiographed *in situ* in order to relate them to the planned insertion site and adjacent dental roots.²¹ Arguably, such wire markers only provide limited and indirect topographical and angulation information, but no inclination guidance for mini-implant insertion. To overcome this problem, 3D removable stents have been described for mini-implants.²²

Implantation/explantation

Several studies on endosseous implants have demonstrated that pre-operative prophylactic antibacterial measures reduce post-operative infection and hence early failure rates. A single dose of pre-operative antibiotics is generally recommended before placement of orthodontic implants, but the consensus is that this is not required for mini-implants other than for general medical reasons.²³ Instead, a chlorhexidine mouthwash or swab may be used immediately pre-operatively to reduce the bacterial load.²⁴

Most TADs can be inserted as a chairside procedure under local anaesthesia. A generous surgical access flap is clearly required for mini-plate systems and a localized subperiosteal flap is recommended by some mini-implant manufacturers. Conversely, some mini-implants may be screwed directly through the attached mucosa, or a soft tissue punch may be used to prevent mucosal tearing and provide a clean-cut tissue margin around the transmucosal neck. The soft tissue thickness at the insertion site influences the choice of fixture, such that a longer transmucosal neck should be used in areas with thick soft tissues. The pilot hole (if required) should be drilled as per the manufacturer's recommendations, at a slow speed with adequate cooling using saline irrigation to minimize heat generation (below 47°C) and associated bone necrosis. The fixture may be seated either with digital pressure using a screwdriver (with or without a torque wrench), or a slow speed handpiece depending on operator choice.

The implant placement torque (IPT) is a measure of resistance to fixture insertion and its relationship to mini-implant success rates was studied in 41 patients (124 mini-implants).²⁵ The results showed that the IPT was higher in the mandible than the maxilla, and that the failure rate in the mandible increased when high torque

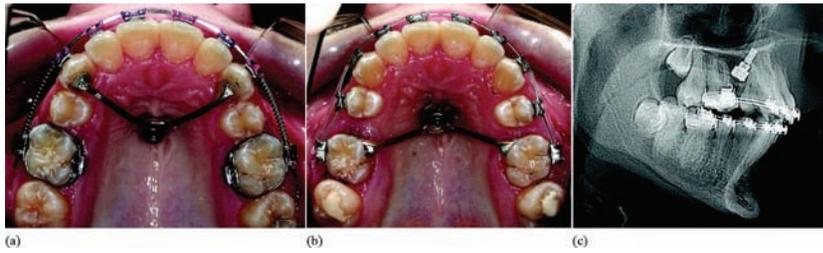


Fig 5 – orthodontic implants in the palatal aspect



Fig 6 – mini implant placed in the buccal aspect

values were encountered during insertion. The authors attributed such failures to excessive stress created in the dense peri-implant bone as indicated by the high IPT values resulting in local ischaemia and bone necrosis. Therefore, it appears that a low IPT may indicate bone deficiency and poor initial stability, whilst a very high torque may be associated with bone degeneration. The authors recommended IPT values within the range of 5–10 Ncm when 1.6 mm diameter mini-implants are used and suggested the use of a relatively larger pilot drill for the mandible than the maxilla.

Good oral hygiene practice and regular chlorhexidine mouthwashes for 1–2 weeks are typically recommended. Clinical studies have shown that inflammation of peri-implant tissue is a contributory risk factor for early failure in both orthodontic implants²⁶ and mini-implants.²⁷ Post-operatively, there should be no signs of pain (including tooth sensitivity), peri-implant inflammation or implant mobility.

Removal of **orthodontic implants** can be done under local anaesthesia using the manufacturer's specific explanation tools. The implant bed is left to granulate and good mucosal coverage occurs within a week. **Mini-plates** require a second episode with full surgical flap access for their removal. Conversely, **mini-implants** are easily removed by unscrewing them using their screwdriver or handpiece adapter and the consensus is that 90% of such episodes do not even require local anaesthesia.²³

Force application on TADs

Straumann recommend that Orthosystem implants are kept unloaded during the initial 12 weeks healing (latency) phase, although there are reports in the literature of this ranging from 2 to 16 weeks.⁷ In a histomorphometric animal study, osseointegrated implants were subjected to continuous forces of 100–300 g.²⁸ This appeared to favourably influence the turnover and density of peri-implant bone, whilst the degree of osseointegration was independent of the amount of loading within this range. A similar experimental study showed that when a continuous uniform force or a static load (e.g. an orthodontic force) is applied, the marginal peri-implant bone is denser than that around implants loaded with a fluctuating (e.g. masticatory) force. Several clinical studies have shown that loaded osseointegrated implants are stable over force levels in the range of 80–600 g.

Mini-implants are usually described as being loaded immediately or after a healing period of 2 weeks. They apparently withstand forces ranging between 50–250 g

and are stable when horizontal or vertical forces are applied provided that these forces cause minimal rotational moments.¹⁰ A study of factors associated with the stability of mini-implants, concluded that the main risk factors for premature loosening were a small diameter, peri-implant inflammation and patients with high mandibular plane angles (who appeared to have thinner buccal cortical bone), but not force levels.

In terms of orthodontic mechanics, either direct or indirect traction may be applied to TADs. For instance, palatal implants usually provide indirect anchorage via a TPA connected to anchor teeth. The TPA can be either soldered to the implant cap or secured with a clamping cap or resin bonding

Conversely, mini-implants usually provide direct anchorage whereby traction is applied to the fixture's head. Occasionally, a mini-implant can be reinforced by combining it with an abutment via a rigid rectangular wire, e.g. to a bracket on the tooth that forms the anchorage unit.

Conclusions

TADs have evolved as viable alternatives to traditional anchorage methods and offer significant advantages in terms of low compliance, efficient, multi-purpose and reliable anchorage. Comparison of the three groups of TADs indicates that once integrated, orthodontic implants provide a reliable method for 'absolute anchorage' and most studies have shown high success rates.⁷ However, they have disadvantages of relatively high costs, invasive placement and removal, elaborate planning and laboratory support, a limited range of anatomical sites for insertion and the requirement for a latency period before clinical loading.

Although, mini-plates can be placed in remote sites independent of the alveolar ridge, this means that surgical access can prove difficult. This is their main disadvantage along with the associated increase in patient morbidity, the degree of invasiveness and relatively high costs. However, they do have advantages of being amenable to immediate loading and versatility in terms of the application of forces in different vectors.

Arguably, mini-implants will be more widely used than the other two TAD groups because of their ease of insertion and removal, wide range of insertion sites, low cost, lower patient morbidity and discomfort, and early/immediate loading. They are also considered more clinician-friendly, since orthodontists can easily insert them as a routine procedure. Although, mini-implants have been shown to displace under loading, they can be safely placed in most inter proximal areas. Their main limitations are dependence on adequate bone quality/

depth for stability, adjacent soft tissue inflammation and a small risk of fracture during insertion or removal. On balance, it appears that as techniques evolve further, mini-implants may be the TAD of choice in most clinical scenarios requiring maximum anchorage reinforcement, whereas implants and mini-plates may be reserved for those cases requiring the use of remote anchorage sites due to over-riding anatomical considerations.

References

- 1 Kanomi R. Mini-implant for orthodontic anchorage. *J Clin Orthod* 1997; **31**: 763–67.
- 2 Kyung HM, Park HS, Bae SM, Sung JH, Kim IB. Development of orthodontic micro-implants for intraoral anchorage. *J Clin Orthod* 2003; **37**: 321–28.
- 3 Park HS, Kwon OW, Sung JH. Micro-implant anchorage for forced eruption of impacted canines. *J Clin Orthod* 2004; **38**: 297–302.
- 4 Dalstra M, Cattaneo PM, Melsen B. Load transfer of miniscrews for orthodontic anchorage. *Orthodontics* 2004; **1**: 53–62.
- 5 Branemark PI, Adell R, Briene U, Hansson BO, Lindstrom J, Ohlsson A. Intra-osseous anchorage of dental prostheses. I. Experimental studies. *Scand J Plast Reconstr Surg* 1969; **3**: 81–100.
- 6 Roberts WE, Smith RK, Zilberman Y, Mozsary PG, Smith RS. Osseous adaptation to continuous loading of rigid endosseous implants. *Am J Orthod* 1984; **86**: 95–111.
- 7 Higuchi KW, Slack JM. The use of titanium fixtures for intraoral anchorage to facilitate orthodontic tooth movement. *Int J Oral Maxillofac Implants* 1991; **6**: 338–344.
- 8 Roberts WE, Marshall KJ, Mozsary PG. Rigid endosseous implant utilized as anchorage to protract molars and close an atrophic extraction site. *Angle Orthod* 1990; **60**: 135–52.
- 9 Creekmore TD, Eklund MK. The possibility of skeletal anchorage. *J Clin Orthod* 1983; **17**: 266–69.
- 10 Costa A, Raffaini M, Melsen B. Miniscrews as orthodontic anchorage: a preliminary report. *Int J Adult Orthod Orthognath Surg* 1998; **13**: 201–09.
- 11 Jenner JD, Fitzpatrick BN. Skeletal anchorage utilizing bone plates. *Aust Orthod J* 1985; **9**: 231–33.
- 12 Umemori M, Suguwara J, Mitani H, Nagasaka H, Kawamura H. Skeletal anchorage system for open-bite correction. *Am J Orthod Dentofacial Orthop* 1999; **115**: 166–
- 13 Carano A, Lonardo P, Velo S, Incorvati C. Mechanical properties of three different commercially available miniscrews for skeletal anchorage. *Prog Orthod* 2005; **6**: 82–97.
- 14 Chung K, Kim SH, Kook Y. The C- Orthodontic microimplant. *J Clin Orthod* 2004; **38**: 478–486.
- 15 Schlegel KA, Kinner F, Schlegel KD. The anatomic basis for palatal implants in orthodontics. *Int J Adult Orthodon Orthognath Surg* 2002; **17**: 133–39.
- 16 Poggio PM, Incorvati C, Velo S, Carano A. 'Safe zones': a guide for microscrew positioning in the maxillary and mandibular arch. *Angle Orthod* 2006; **76**: 191–97.
- 17 Schnelle MA, Beck FM, Jaynes RM, Huja SS. A radiographic evaluation of the availability of bone for placement of miniscrews. *Angle Orthod* 2004; **74**: 832–37.
- 18 Asscherickx K, Vannet BV, Wehrbein H, Sabzevar MM. Root repair after injury from mini-screw. *Clin Oral Implants Res* 2005; **16**: 575–78.
- 19 Wehrbein H, Merz BR, Diedrich P. Palatal bone support for orthodontic implant anchorage—a clinical and radiological study. *Eur J Orthod* 1999; **21**: 65–70.
- 20 Tosun T, Keles A, Erverdi N. Method for the placement of palatal implants. *Int J Oral Maxillofac Implants* 2002; **17**: 95–100.
- 21 Maino BG, Bednar J, Pagin P, Mura P. The Spider Screw for skeletal anchorage. *J Clin Orthod* 2003; **37**: 90–97.
- 22 Kitai N, Yasuda Y, Takada K. A stent fabricated on a selectively colored stereolithographic model for placement of orthodontic mini-implants. *Int J Adult Orthodon Orthognath Surg* 2002; **17**: 264–66.
- 23 Mah J, Bergstrand F. Temporary anchorage devices: a status report. *J Clin Orthod* 2005; **39**: 132–36.
- 24 Lambert PM, Morris HF, Ochi S. The influence of 0.12% chlorhexidine digluconate rinses on the incidence of infectious complications and implant success. *J Oral Maxillofac Surg* 1997; **55**: 25–30.
- 25 Motoyoshi M, Hirabayashi M, Uemura M, Shimizu N. Recommended placement torque when tightening an orthodontic mini-implant. *Clin Oral Implants Res* 2006; **17**: 109–14.
- 26 Forna N, Burlui V, Luca IC, Indrei A. Peri-implantitis. *Rev Med Chir Soc Med Nat Iasi* 1998; **102**: 74–79.
- 27 Cheng SJ, Tseng IY, Lee JJ, Kok SH. A prospective study of the risk factors associated with failure of mini-implants used for orthodontic anchorage. *Int J Oral Maxillofac Implants* 2004; **19**: 100–06.
- 28 Melsen B, Lang NP. Biological reactions to orthodontic loading of oral implants. *Clin Oral Implants Res* 2001; **12**: 144–52.

Multiple Angiomas of oral cavity - A review and report of a case

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Vascular anomalies have been and remain poorly understood. This is in part the result of a lack of a uniformly accepted classification and unclear understanding of the natural history of these lesions. In addition, clinicians from many specialties are involved in the management of these patients and have looked for solutions within their specialty and applied them to all lesions. It is therefore not surprising that the lack of consensus on the terminology of vascular anomalies has resulted in a wealth of publications that are often imprecise and commonly perpetuate misconceptions and inaccuracies about the diagnosis and management of these lesions.

Before the 1980s, the terminology that was used to describe vascular anomalies was confusing and ambiguous. The descriptive terminology used in the past (port wine stain, strawberry hemangioma, salmon patch) conjure up visual approximation to the lesions but have no correlation with the biological behaviour or natural history of these lesions^{1,2}. Mulliken and Glowacki introduced a simple classification in 1982 that was based on the clinical, histochemical, and cellular criteria to distinguish between the various vascular anomalies. They described two distinct entities—hemangiomas and vascular malformations³.

Hemangiomas are tumor like malformations composed of seemingly disorganized masses of endothelium-lined vessels that are filled with blood and connected to main blood vascular system¹. They represent the most common benign soft tissue tumor of childhood, occurring in 4% to 10% of children⁶. They have been described in almost all locations of the body. *Watson and McCarthy* in 1843 studied 1308 lesions and reported that 56% of the lesions occurred in the head and neck region and the remaining part of the total body accounted for only 44%⁴. They may occur as isolated lesions in the oral cavity or as multiple lesions or in association with other developmental anomalies in various angiomatous syndromes⁵.

We report a case of multiple angiomas of oral cavity with multiple phleboliths in a 52 year old lady.

Case Report

A 52 year old lady presented with a swelling over the lower lip and tongue of 2 years duration. The patient had noticed a small diffuse swelling with bluish discoloration over the lower lip two years back that gradually enlarged to the present size, without any associated symptoms. This was followed by similar swellings over the tongue. There was no other relevant medical or family history. On examination, multiple soft, diffuse, non tender swellings over the lower lip, dorsum of tongue, floor of mouth and right buccal mucosa were evident clinically. The swelling over the lower lip was 1 x 1 cm in size, that over the right buccal mucosa was 2 x 2 cm in size and floor of the mouth 3 x 2 cm in size. The body of the tongue showed three nodules of 1 x 1 cm in size. All the lesions had a bluish hue. They were sessile with smooth surface, no ulceration or bleeding. The panoramic view of mandible showed multiple areas of radio-opacity around the mandible which were diagnosed to be phleboliths. A CT Angiogram brain including neck was done. Multiple hypodense lesions with coarse, round calcifications were noted in the following regions:

- Lower pole of right parotid gland - 2.5 x 2 cm
- Right parapharyngeal space - 1.5 x 1 cm
- Right posterior ethmoidal air cells - 2 x 1.5 cm
- Left submandibular region - 3 x 2 cm
- Right buccal space - 2 x 1.4 cm
- Left anterolateral aspect of tongue
- Right masticator space between temporalis and lateral pterygoid



Fig A. Lower lip



Fig B. Floor of the mouth and buccal mucosa



Fig C. Tongue

The lesions appeared to be drained by tributaries of external jugular vein. The external jugular veins on either side also appeared prominent. But no arterial feeders were noted. All the above features were suggestive of **multiple venous vascular malformations**.

Discussion

Hemangiomas are benign, vascular lesions, generally congenital in origin, developing from abnormally differentiated blood vessels. Most lesions are solitary (80%) and girls are more affected than boys (3:1)^{1,3,4}. Facial hemangiomas have a predilection for segmental distribution and for regions of embryological fusion. The most common sign is of a slow-growing palpable mass that can fluctuate⁷. The skin overlying the hemangioma will often show increased vascularity, giving the hemangioma a bluish tint. Pulsations, bruits, or thrills are rarely detectable in hemangioma⁸. It is usually detected by the second or third decade of life. Hemangioma affects as many as 12% of white, but it rarely occurs in darker colored individuals.^{3,4} The exact cause of hemangioma is unknown; however, either trauma or hormonal changes have been postulated as being involved.

The terms capillary and cavernous hemangioma are out of date and the lesions are more appropriately described according to the depth of the lesion as superficial, deep, and compound hemangioma⁴. Superficial hemangiomas originate from the papillary dermis and present as bright red, macular or papular masses (previously called capillary or strawberry hemangioma). Deep hemangiomas originate from the reticular dermis or subcutaneous tissues and appear as bluish or relatively colorless masses (previously called cavernous hemangioma). Compound hemangiomas have superficial and deep components and were previously called capillary cavernous hemangiomas.

Most oral hemangiomas are located on the tongue, where they are multinodular and bluish red^{12,13,14}. The multinodularity is racemose and diffuse. The lip mucosa is another common site of involvement in children. Hemangiomas range from simple red patches (port wine stain) which do not raise the mucosal surface, to large fungating masses which bury teeth and cause serious disfigurement. Lesions close to the surface appear

reddish blue or if a little deeper, a deep blue. Angiomatous lesions occurring within the muscle (intramuscular hemangioma) may fail to show any surface discoloration. *Liston*, in 1843, recorded the first case of intramuscular hemangioma involving the semi membranous muscle in the popliteal space².

Hemodynamics in angiomas is perturbed, and stasis with thrombosis is commonly encountered. Most patent vascular lesions will blanch under pressure; indeed, placing a microscope glass slide over the pigmented area and adding pressure will often demonstrate this feature dramatically. Conversely, when intraluminal clots form, they become palpable and the lesion will usually not blanch. Thrombi in angiomas may eventually calcify, and such lesions will feel hard on palpation. The calcified nodules, or *phleboliths*, may be radiographically evident^{9,10}. Computed tomography, Doppler and conventional ultrasonography, radionuclide-labeled red cell scintigraphic scanning, MRI and superselective microangiography are used to diagnose hemangioma¹¹.

Since many hemangiomas spontaneously involute during teenage years, treatment may be withheld in children. Patients who require treatment can undergo conventional surgery, laser surgery, or cryosurgery. Larger lesions that extend into muscles are more difficult to eradicate surgically, and sclerosing agents such as 1% sodium tetradecyl sulfate may be administered by intralesional injection. These agents result in postoperative pain, and the patient must be managed with a moderate-level analgesic such as oxycodone or aspirin with codeine. Cutaneous port-wine stains can be treated by subcutaneous tattooing or by argon laser. Potassium titanyl phosphate laser may be used in the management of subglottic hamangioma.¹²

Hemangiomas of the skin and oral mucous membrane often coexist with similar lesions of the central nervous system and the meninges. A variety of such angiomatous syndromes have been described.

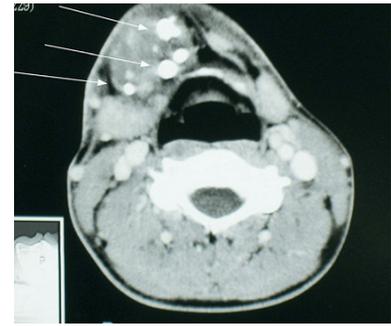
Sturge-Weber syndrome is characterized by unilateral "port wine" telangiectasis in the face (nevus flammeus), with a variable distribution sometimes matching the dermatomes of one or more trigeminal nerve divisions. The intraoral lesions in this syndrome classically occur on the same side of the body as other angiomas in the



Fig. D. Tongue



Panoramic view showing phleboliths (arrow mark)



patient, but the classic pattern is not always found in either the distribution or the expression of the various components of the syndrome. Involvement of the ipsilateral meninges with associated atrophy of the cerebral cortex can lead to epilepsy and intellectual impairment.

Maffucci syndrome or Enchondroma with multiple angiomas was first reported by Maffucci in 1881. It is a rare genetic disorder that affects both males and females. It is characterized by benign enlargements of cartilage (enchondromas); bone deformities; and dark, irregularly shaped hemangiomas. No racial or sexual predilection is apparent. No familial pattern of inheritance has been shown, but the disease manifests early in life, usually around the age of 4 or 5 years, with 25% of cases being congenital.

In 1940, Kasabach and Merritt described a male infant with a discolored, indurated lesion on his left thigh that rapidly grew and affected the entire left leg, scrotum, abdomen, and thorax. In addition, the infant also had consumptive thrombocytopenia. This association has become known as *Kasabach-Merritt syndrome*⁵. The original case is now known to have been associated with kaposiform hemangioendothelioma and thrombocytopenia. These lesions can occur anywhere on the skin which is the organ most commonly affected. Lesions of the internal organs often cause bruising on the skin. Lesions are usually painful and tender. Aggressive infiltration with ulceration and infection is rare but can occur. Bleeding from thrombocytopenia and coagulopathy is frequently observed.

Conclusion

An awareness of the clinical appearance of angiomas is very important. These lesions when present in the teeth bearing areas may lead to fatal post extraction bleed. Multiple angiomas are rare and when present the possibility of associated syndromes must be ruled out.

References

1. Waner M, Suen JY. Hemangiomas and vascular malformations of the head and neck. New York: Wiley-Liss; 1999
2. Mulliken JB, Young AE. Vascular birthmarks. Hemangiomas and malformations. Philadelphia: Saunders; 1988
3. Mulliken JB, Glowacki J. Hemangiomas and vascular malformations in infants and children: a classification based on endothelial characteristics. *Plast Reconstr Surg* 1982; 69:412-22
4. M. Ethunandan, Timothy K. Mellor; Haemangiomas and vascular malformations of the maxillofacial region—A review; *British Journal of Oral and Maxillofacial Surgery* 44 (2006) 263-272
5. Neville BW, Damm DD, Allen CM, Bouquot JE. Oral & maxillofacial pathology. 2nd ed. Philadelphia: Saunders; 2002. p. 337-69.
6. Paolo Scolozzi, Francois Laurent, Tommaso Lombardi, Michel Richter; Intraoral venous malformation presenting with multiple phleboliths; *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2003;96:197-200
7. Hasan Ayberk Altug, Vural Büyüksoy, Kemal Murat Okçu, DDS, Necdet Dogan, Diyarbakır and Ankara; Hemangiomas of the head and neck with phleboliths: Clinical features, diagnostic imaging, and treatment of 3 cases; *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2007;103:e60-e64
8. Addante RR, Donovan MG. Right facial mass. *J Oral Maxillofac Surg* 1994;52:1061-5.
9. Allen PW, Enzinger FM. Hemangioma of the skeletal muscle: an analysis of 89 cases. *Cancer* 1972;29: 8-22
10. Terezhalmay GT, Riley CK, Moore NS. Intramuscular hemangiomas. *Quintessence Int* 2000;31: 142-3
11. Koichi Yonetsu, Eiji Nakayama, Toshiyuki Kawazu, Shigenobu Kanda, Satoru Ozeki, Masanori Shinohara; Value of contrast-enhanced magnetic resonance imaging in differentiation of hemangiomas from lymphangiomas in the oral and maxillofacial region; *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1999;88:496-500
12. Craig L. Hatch; Pigmented lesions of the oral cavity; *Dent Clin N Am* 49 (2005) 185-201
13. Carapeto FJ, Garcia-Perez A, Winkelmann RK. Acral arteriovenous tumor. *Acta Dermatovener (Stockh)* 1977;57:155-8.
14. Connelly MG, Winkelmann RK. Acral arteriovenous tumor. A clinicopathological review. *Am J Surg Pathol* 1985;9:15-21

Photodynamic therapy (PDT) - A review

Abstract

Photodynamic therapy (PDT) is a relatively new treatment modality. It involves the administration of a photosensitizer followed by local illumination with visible light of specific wavelength. In the presence of oxygen, the light illumination of photosensitizer can lead to a series of photochemical reactions and consequently the generation of cytotoxic species. Applications of Photodynamic therapy in dentistry may include treatment of premalignant and malignant oral lesions, and photodynamic antimicrobial chemotherapy (PACT) of bacterial and fungal infections. This review discusses the new developments in PDT basic sciences, clinical applications, and the usefulness of various forms of PDT techniques.

Key words: photodynamic therapy, photosensitiser, light delivery systems.

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Introduction

Photodynamic therapy involves the use of a photoactive dye (photosensitizer) that is activated by exposure to light of a specific wavelength in the presence of oxygen. The exposure of the photosensitizer to light results in the formation of oxygen species, such as singlet oxygen and free radicals, causing localized photodamage and cell death. The advantage of this new approach includes rapid bacterial elimination, minimal chance of resistance development and safety of adjacent host tissue and normal microflora. This review elucidates the evolution and use of photodynamic therapy

Mechanism of action

PDT involves three components: light, a photosensitizer, and oxygen. A photosensitizer or its metabolic precursor is administered to the patient. Upon irradiation with light of a specific wavelength, the photosensitizer undergoes a transition from a low-energy ground state to an excited singlet state. Subsequently, the photosensitizer may decay back to its ground state, with emission of fluorescence, or may undergo a transition to a higher-energy triplet state. The triplet state can react with endogenous oxygen to produce singlet oxygen and other radical species, causing a rapid and selective destruction of the target tissue (Fig. 1).

There are two mechanisms by which the triplet-state photosensitizer can react with biomolecules.

Type I involves electron/hydrogen transfer directly from the photosensitizer, producing ions, or electron/hydrogen removal from a substrate molecule to form free radicals. These radicals react rapidly with oxygen, resulting in the production of highly reactive oxygen species (superoxide, hydroxyl radicals, hydrogen peroxide).

Type II reactions produce the electronically excited and highly reactive state of oxygen known as singlet oxygen. In PDT, it is difficult to distinguish between the two reaction mechanisms. A contribution from both Type I and II processes indicates that the mechanism of damage is dependent on both oxygen tension and photosensitizer concentration

Lastly Type III reaction is a unique PS reaction because it is oxygen independent. These reactions require either high concentration of the PS or a deaerated system, in order to bypass the reaction with oxygen. Under anaerobic system, radicals are generated and these can subsequently react.

Figure1 shows Schematic representation of photodynamic reaction and photodynamic therapy. Light (photon) of an appropriate energy is absorbed by a photosensitizer, which undergoes a transition from a low-energy ground state to the excited-singlet state. The activated photosensitizer interacts with oxygen to produce singlet oxygen and other radical species that cause a toxic effect in tumor cells or microorganisms.

Photosensitizers

A photosensitive molecule is one which on activation by radiation or light causes another molecular component to react (McCaughan 1999, Meisel and Kocher 2005). In general there are three basic groups of PS, Tricyclic dyes, Tetrapyrroles and Furocoumarins (Meisel and Kocher 2005). More than 400 compounds are known with photosensitizing properties including dyes, drugs, cosmetics chemicals, and many natural substances. Most of the sensitizers used for medical purposes belong to the following basic structures

Tricyclic dyes with different meso atoms - .Acridine orange, proflavin, riboflavin, methylene blue, fluorescein, eosin, erythrosine rose Bengal and Toluidine Blue O (TBO; tolonium chloride))

Tetrapyrroles - Porphyrins and derivatives, chlorophyll, phyloerythrin, phtalocyanines

Furocoumarines. Psoralen and its methoxy-derivatives xanthotoxin, bergaptene

Photosensitizer in Periodontal Therapy should be non-toxic & activated upon illumination. It Should bind with bacteria & plaque without causing any cosmetic issues, such as unwanted staining of gingiva, other soft tissues and easily access pathogens present in deeper periodontal pockets

Light Sources

PDT requires a source of light that activates the photosensitizer by exposure to low-power visible light at a specific wavelength. Human tissue transmits red light efficiently, and the longer activation wavelength of the photosensitizer results in deeper light penetration. Consequently, most photosensitizers are activated by red light between 630 and 700 nm, corresponding to a light penetration depth from 0.5 cm (at 630 nm) to 1.5 cm (at 700 nm). In the past, photosensitizer activation was achieved via a variety of light sources, such as argon-pumped dye lasers, potassium titanyl phosphate (KTP)- or neodymium:yttrium aluminum garnet (Nd/YAG)-pumped dye lasers, and gold vapor- or copper vapor-pumped dye lasers. All these laser systems are complex and expensive. At present, diode laser systems that are easy to handle, portable, and cost-effective are used predominantly. For treatment of larger areas, non-coherent light sources, such as tungsten filament, quartz halogen, xenon arc, metal halide, and phosphor-coated sodium lamps, are in use. Recently, non-laser light sources, such as light-emitting diodes (LEDs), are making an impact on PDT. The LED devices are small, lightweight, and highly flexible. In addition, LEDs are highly efficient for second-generation photosensitizers, with absorption wavelengths closer to the LED peak emission.

Applications of PDT in Dentistry

PHOTODYNAMIC ANTIMICROBIAL CHEMOTHERAPY OF DENTAL AND MUCOSAL INFECTIONS (PACT)

In recent years, the emergence of antibiotic resistant strains, such as methicillin resistant *Staphylococcus aureus* and vancomycin-resistant *Enterococcus faecalis*,

stimulated a search for alternative treatments. PACT has the potential to be an alternative treatment modality, replacing antibiotics especially for the treatment of localized infections of the skin and the oral cavity. Micro-organisms that are killed by PACT include bacteria, fungi, viruses, and protozoa. PACT is equally effective against antibiotic-resistant and antibiotic-susceptible bacteria, and repeated photosensitization has not induced the selection of resistant strains.

Effects of pact (photodynamic antimicrobial chemotherapy) on oral biofilms

The antimicrobial activity of photosensitizers is mediated by singlet oxygen, which, because of its high chemical reactivity, has a direct effect on extracellular molecules. Thus, the polysaccharides present in extracellular matrix of polymers (EMP) of a bacterial biofilm are also susceptible to photodamage. Such dual activity, not exhibited by antibiotics, represents a significant advantage of PACT. Breaking down biofilms may inhibit plasmid exchange involved in the transfer of antibiotic resistance, and disrupt colonization. The activity of PACT against homogenous and mixed Gram-positive/Gram-negative oral biofilms has been reported for a range of photosensitizers

PACT for Peri-Implantitis and Endodontic Treatment

Biofilms of *Streptococcus intermedius* prepared in artificial root canals and extracted human teeth were subjected to PACT with TBO and a laser diode device (633 nm) equipped with an endotip that allowed light to be transmitted down to the apex of the tooth. *S. intermedius* was present in numbers similar to those found in heavily infected root canals. Photoactivated disinfection significantly reduced the number of bacteria in both types of root canals (Williams et al., 2006). Seal et al. (2002) reported partial inactivation of *S. intermedius* biofilms in root canals of extracted teeth, using TBO and a helium/neon laser (633 nm). In vitro and in vivo studies using PDT have shown that this approach has the potential to maximize root canal disinfection. However, while disclosing and confirming the excellent antibacterial potential of PDT, none of these studies have consistently examined the effectiveness of this procedure in supplementing bacterial elimination after chemomechanical procedures, which is the greatest potential use of this technology with regard to root canal disinfection. PDT application has an adjunctive benefit besides mechanical treatment at sites with difficult access (furcations, deep invaginations, concavities) Necessity for flap operations may be reduced, patient comfort may increase and treatment time decrease. PDT removes the biofilm in residual deep pockets during maintenance phase without the need for a mechanical intervention. PDT may decrease the risk of bacteremia, which routinely occurs after periodontal treatment procedure.

Perspectives and future directions

PACT appears to be most efficient for treatment of localized and superficial infections. Thus, infections in



Figure 1

Application of Photosensitizer

Laser exposing (1 min per tooth)

the oral cavity—such as mucosal and endodontic infections, periodontal diseases, caries, and peri-implantitis—are potential targets. PACT will not replace antimicrobial chemotherapy, but the photodynamic approach may improve the treatment of oral infections, accelerating and lowering the cost of the treatment. Development of new photosensitizers, more efficient light delivery systems, and further animal studies are required to establish the optimum treatment parameters before investigators can proceed to clinical trials and eventual clinical use. The future of PDT will depend on the interactions between clinical applications and technological innovations. Allison et al. (2006) have described PDT as the therapy that “is truly the marriage of a drug and a light”, and, as a result, only interdisciplinary research approaches can overcome all the difficulties and challenges of PDT.

Conclusion

PACT appears most efficient for localized and superficial infections. Thus, infections in the oral cavity — such as mucosal and endodontic infections, periodontal diseases, caries and peri-implantitis — are potential targets. PACT will not replace antimicrobial chemotherapy, but the photodynamic approach may improve treatment of oral infections, speeding up treatment and lowering its cost. In November 17, 2005 the first regulatory approval for photodynamic disinfection therapy from Health Canada was announced. Also it received company-wide ISO-13485 certification of quality management standard for medical device companies. Based on the evidence to date, treatment of periodontal diseases using PDT of any type is still considered experimental. Based on these articles one must conclude that there is a great need to develop an evidence-based approach to the use of lasers for the treatment of periodontitis. It would be prudent to say that there is insufficient evidence to suggest that PDT is superior to the traditional modalities of periodontal therapy. Everything new is not always better so an astute clinician should critically appraise all the literature before making a dramatic change from the current standard of treatment.

References

1. (Williams JA, Pearson GJ, Colles MJ (2006). Antibacterial action of photoactivated disinfection {PAD} used on endodontic bacteria in planktonic suspension and in artificial and human root canals. *J Dent* 34:363-371.)
2. (Seal GJ, Ng YL, Spratt D, Bhatti M, Gulabivala K (2002). An in vitro comparison of the bactericidal efficacy of lethal photosensitization or sodium hypochlorite irrigation on *Streptococcus intermedius* biofilms in root canals. *Int Endod J* 35:268-274.)
3. Fonseca MB, Junior PO, Pallota RC, et al. Photodynamic therapy for root canals infected with *Enterococcus faecalis*. *Photomed Laser Surg* 2008;26:209
4. Garcez AS, Nunez SC, Lage-Marques JL, et al. Efficiency of NaOCl and laser-assisted photosensitization on the reduction of *Enterococcus faecalis* in vitro. *Oral Surg, Oral Med Oral Pathol Oral Radiol Endod* 2006;102:e93-8.
5. Garcez AS, Ribeiro MS, Tegos GP, et al. Antimicrobial photodynamic therapy combined with conventional endodontic treatment to eliminate root canal biofilm infection. *Lasers Surg Med* 2007;39:59-66.
6. Soukos NS, Chen PS, Morris JT, et al. Photodynamic therapy for endodontic disinfection. *J Endod* 2006;32:979-84. Williams JA, Pearson GJ, Colles MJ. Antibacterial action of photoactivated disinfection (PAD) used on endodontic bacteria in planktonic suspension and in artificial and human root canals. *J Dent* 2006;34:363-71.
7. Bergmans L, Moisiadis P, Huybrechts B, et al. Effect of photo-activated disinfection on endodontic pathogens ex vivo. *Int Endod J* 2008;41:227-39.
8. Fimple JL, Fontana CR, Foschi F, et al. Photodynamic treatment of endodontic polymicrobial infection in vitro. *J Endod* 2008;34:728-34.
9. Meire MA, De Prijck K, Coenye T, et al. Effectiveness of different laser systems to kill *Enterococcus faecalis* in aqueous suspension and in an infected tooth model. *Int Endod J* 2009;42:351-9.
10. Lim Z, Cheng JL, Lim TW, et al. Light activated disinfection: an alternative endodontic disinfection strategy. *Aust Dent J* 2009;54:108-14.
11. Garcez AS, Nunez SC, Hamblin MR, et al. Antimicrobial effects of photodynamic therapy on patients with necrotic pulps and periapical lesion. *J Endod* 2008;34: 138-42.
12. Bonsor SJ, Nichol R, Reid TM, et al. Microbiological evaluation of photo-activated disinfection in endodontics (an in vivo study). *Br Dent J* 2006;200:337-41. discussion

Dental Management of a Patient with Cleidocranial Dysplasia- A Case Report.

Abstract

Cleidocranial dysplasia is a generalized skeletal dysplasia leading to multiple abnormalities⁽¹⁻²⁾. The major concern of the patient is most often the oral/dental disorders.⁽³⁾ Cleidocranial dysplasia (CCD) is a rare congenital disorder, (incidence 1:1000 000)⁽⁴⁾ primarily affecting bones that undergo intramembraneous ossification, i.e., generally the calvarian but also clavicular bones. CCD is also known as Marie"Sainton disease, mutational dysostosis, and cleidocranial dysostosis. The dental abnormalities associated with it present a remarkable challenge in orthodontic treatment planning. Early diagnosis is extremely important to give the patient the best treatment options. Patients with cleidocranial dysostosis require a team approach with good communication and cooperation from the patient. Timing of the intervention is critical, and many surgeries might be required.

Here, we report a case of CCD in a 12 "year" old boy, who had come to us with a chief complaint of missing front teeth.

Key Words: Cleidocranial Dysplasia, Syndrome, Cleidocranial Dysostosis, Marie"Sainton Disease, Mutational Dysostosis, and Cleidocranial Dysostosis

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Cleidocranial dysplasia is a generalized skeletal dysplasia leading to multiple abnormalities¹⁻² the major concern of the patient is most often the oral/dental disorders.³ Cleidocranial dysplasia (CCD) is a rare congenital disorder, (incidence 1:1000 000)⁴ primarily affecting bones that undergo intramembraneous ossification, i.e., generally the calvarian but also clavicular bones. Since its first description in 1898 by Pierre Marie and Paul Sainton,⁵ over 1000 cases have been published in the medical literature.

The skull base is dysplastic and reduced in growth. Radiographs of the newborn demonstrate poor or absent ossification of the parietal bones. Owing to reduced growth of bones developing from the chondrocranium, increased skull width and resulting hypertelorism usually appear with associated biparietal and frontal bone bossing. Together with underdevelopment of the facial bones, which often results in a mid face deficiency and narrow paranasal sinuses, the dysostotic growth attributes to many patients an almost familiar resemblance. Chief intraoral expressions include retained deciduous dentition, delayed eruption or retention of the permanent dentition, multiple supernumerary teeth, actual cleft palate, hypoplastic maxilla, enlarged mandibles, a high narrow arched palate and absence or paucity of cellular cementum on the roots.⁶

The thoracic cage is small and bell shaped with short ribs. The pelvis shows a delayed closure of wide symphysis pubis. The dysplastic pelvis often necessitates caesarean section of pregnant female. Hands and feet have pseudoepiphyses at the base of the metacarpal bones and abnormal phalangeal tufts, and often cone-shaped epiphyses of the distal phalanges.

Signs and symptoms were divided into the categories like supernumerary teeth, failure of tooth eruption, hypoplastic maxilla, "clavicular sign" and other skeletal disorders. The clavicular sign was defined by the patient's ability to oppose the shoulders in front as a result of hypoplastic or aplastic clavicles. This criterion helps in easy diagnosis and the reliability of the symptom even if not expressed in all patients.⁷ The wormian bones, small epactal bones in the lambdoid suture, is also recurrently expressed in patients with cleidocranial dysplasia.⁸

In addition, the family history did not reveal any significant findings.

Diagnosis

In 1997, the aetiological factor of cleidocranial dysplasia, RUNX2 gene, was mapped on the short arm of chromosome⁹ by Mundlos et al.⁹ RUNX2 is considered a master gene in the formation of bone and dental tissue. R.Rajendran and B.Sivapathasundharam suggest several chromosome

abnormalities with this syndrome including rearrangement of long arm of chromosome⁸ (8q22) and the long arm of chromosome.⁶ 10 Mutations in the core-binding factor alpha-1 (CBFA1) gene, located on chromosome 6p21, have been shown to be the cause of cleidocranial dysplasia.

A strong correlation between body height and supernumerary teeth as a function of the involvement of the "runt homology domain", a structural feature of the RUNX2 protein involved in the DNA binding site recognition was reported by Yoshida et al.¹¹

Case report

A male patient aged 12 years with short stature reported to our department with a chief complaint of missing permanent teeth. A detailed case history, was taken and upper and lower impressions for study cast, diagnostic radiographs etc. were taken.

Extra oral Findings:

Frontal bossing, hypertelorism, midface deficiency and clavicular sign were present.

Intra oral Findings:

High arched palate, retained primary teeth, maxillary hypoplasia, delayed eruption of the permanent teeth except for the first permanent molars, mobile lower primary incisors etc.

Dentition

In upper, primary two incisors [61 and 62], canines, first primary molar [64], second primary molars [55 and 65] and in the lower arch all the primary teeth were present. All the permanent first molars had erupted.

Radiographic examination of the chest x-ray revealed absence of clavicles and PNS view showed underdeveloped paranasal sinus. An orthopantomograph was taken to evaluate the status of missing permanent teeth and was found that there were multiple unerupted permanent teeth and no supernumerary teeth were present.

Treatment

In the dental management of CCD patients today, total extraction of all permanent teeth followed by full dentures has been completely abandoned. The tendency of maxillary underdevelopment can be neutralised by preservation of the permanent teeth through the natural dento-alveolar compensatory mechanism.

The first molars seem to erupt spontaneously in nearly all patients, presumably because they only have a very thin layer of bone to pass through. Orthodontic treatment seems to have been necessary in nearly all CCD cases described in the literature¹² but it is generally agreed that removal of primary teeth will improve the possibility of spontaneous eruption. Also, bone overlying the normal, permanent teeth should be removed, as it has been shown histologically,¹³ that the alveolar bone in CCD has abnormal dense trabeculation with multiple

reversal lines, indicating incomplete resorption.

Patients with no or only a few supernumerary teeth had a much higher frequency of spontaneous eruption.¹⁴ Hence we also expected an acceptable good result and planned for the extraction of retained primary teeth.

Management of structural deformity can be corrected by orthognathic surgery /distraction osteogenesis.

Treatment Done

Extraction of 71,72,81 and 82 with the surgical exposure of 31,32,41 and 42 was done under local anaesthesia. Patient was kept under observation for the eruption of the permanent teeth for few months and if required can be intervened by fixed orthodontics later.

A slow maxillary expansion screw with reverse pull head gear was given as an attempt to correct the midface hypoplasia till the growth is complete and later to go in for further surgical correction.

Other treatment option which shall be considered is extraction of retained primary teeth and removal of the overlying bone over the unerupted permanent teeth, extrusion of the teeth followed by fixed orthodontic therapy.

Surgical options being distraction osteogenesis, for the correction of midface deficiency as quoted by Seiji et al.¹⁵ There is no doubt that the distraction osteogenesis has become one of the important treatments for facial skeletal deformity, and recent numerous reports showed well the benefit of this treatment.¹⁶

Discussion

Although there are many case reports available in the literature, scientific interdisciplinary studies are rare.

The bone defects in patients with cleidocranial dysplasia chiefly involve the clavicles and skull, although a wide variety of anomalies may be found in other bones. The clavicles are absent, either unilaterally or bilaterally, in about 10% of all cases. More commonly, the clavicles show varying degrees of hypoplasia and malformation. In this case there was absence of the clavicles.

Ishii et al¹⁷ assessed craniofacial morphology in young and adult individuals with cleidocranial dysplasia obtained from lateral head films. They demonstrated that young subjects showed relatively normal jaw proportions and morphology of the mandible, while older individuals tended to express the typical signs of cleidocranial dysplasia. There was a marked discrepancy in the size of maxilla and mandible in this child. These differences can be attributed to pronounced horizontal mandibular growth resulting from lack of vertical maxillary growth as well as impaired eruption of permanent teeth.

The gnathic and dental manifestations are distinctive and may lead to the initial diagnosis. As Neville et al¹⁸ stated the patients often have a narrow, high arched palate.

Prolonged retention of deciduous teeth and delay or complete failure of eruption of permanent teeth are characteristic features. In the current case report we have recorded the presence of all the permanent first molars.

Conclusion

Timing of intervention seems to be of the utmost importance for a successful result. As patients do not experience any functional or psychosocial problems until the age of 9-10 years, referral for treatment will often be too late to reach the 'correct-for-eruption' developmental stage of the incisor. Another reason for late referral might also be the short stature of these children who, in many cases, look very young for their age.

The dentist should take radiographs at least once a year from the age of 5 years and ensure timely intervention.¹⁴ Patients should also be observed for development of distal molars, cysts, etc until late adolescence.¹⁴

It is essential that the pediatric dentist understands the development of dentition, generalized skeletal dysplasia and other disorders associated with cleidocranial dysplasia.

References:

- Gorlin RJ, Cohen M M Jr, Levin L S. Cleidocranial dysplasia. In : Gorlin R J, Cohen M M Jr, Levin LS(eds). *Syndromes of the head and neck*. P249-252. Oxford: Oxford University Press, 1990.
- Jensen B L. Somatic development in cleidocranial dysplasia. *Am J Med Genet* 1990;35:69-74
- Jensen B L, Kreiborg S. Development of the dentition in cleidocranial dysplasia. *J Oral Pathol Med* 1990;19:89-93
- Golan I, Baumert U, Held P, Feuerbach S, Mussig D. Radiological findings and molecular genetic confirmation of cleidocranial dysplasia. *Clin Radiol* 2002; 57:525-529.
- Marie P, Sainton P. Sur la dysostose cleido-cranienne hereditaire. *Rev Neurol* 1898;6:835-838
- Tyndall DA. Cleidocranial dysostosis: a nearly unrecognized case. *Gen Dent* 1983; 31: 390-393.
- Mundlos S, Otto F, Mundlos C, Mulliken JB, Aylsworth AS, Albright S, et al., Mutations involving the transcription factor CBFA 1 cause cleidocranial dysplasia. *Cell* 1997;89:773-779.
- I Golan, U Baumert BP Hrala and D MuBig. Dentomaxillofacial variability of cleidocranial dysplasia: clinicoradiological presentation and systematic review. *Dentomaxillofacial Radiology* 2003;32:347-354.
- Mundlos S, Otto F, Mundolos C, Mulliken JB, Aylsworth AS, Albright S et al. Mutations involving the transcription factor CBFA1 cause cleidocranial dysplasia. *Cell* 1997; 89:773-779.
- Shafer's textbook of oral pathology V Edition page No.994-997.
- Yoshida T, Kanegane H, Osato M, Yanagida M, Miyawaki T, Ito Y, et al., Functional analysis of RUNX2 mutations in Japanese patients with cleidocranial dysplasia demonstrates novel genotype- phenotype correlations. *Am J Hum Genet* 2002, 71:724-738.
- Elomaa E, Elomaa M. Orthodontic treatment of a case of cleidocranial dysostosis *Suom Hammaslaak Toim* 1967; 63:142-151.
- Bishop RG. Dental management if cleidocranial dysostosis. Case report. *Aust Dent J* 1984;29:1-4
- B.L. Jensen and S.Kreiborg. Dental treatment strategies in cleidocranial dysplasia. *Br Dent J* 1992;172:243.
- Seiji Iida, Tomonao Aikawa, Natsuko Yoshimura. Maxillary distraction osteogenesis using the intraoral distractors and the full- covered tooth-supported maxillary splint. *Oral Maxillofac Surg* 2007;65;813-817
- Swennen G, Schliephake H, Dempf R, et al: Craniofacial distraction osteogenesis: A review of the literature: Part 1: Clinical studies. *Int J Oral Maxillofac Surg* 2001;30:89
- Ishii K, Nielsen IL, Vargervik k. Characteristics of jaw growth in cleidocranial dysplasia. *Cleft Palate Craniofac J* 1998; 35: 161-166.
- Neville, Damm, Allen, Bouquot-II Edition. *Oral & Maxillofacial Pathology*-537-539

Management of trismus using Trismus Appliance: A Case Report

Abstract

Abstract: Trismus is a problem commonly encountered by the dental practitioner. It has a number of potential causes, and its treatment will depend on the cause. This article reports management of trismus using treaded tapered screw.

Key words: Trismus, treaded tapered screw, trismus appliances

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Introduction

The term trismus denotes a motor disturbance of the trigeminal nerve, especially spasm of the masticatory muscles, with difficulty in opening the mouth.¹

Causes of trismus

Several conditions may cause or predispose an individual to develop trismus. The aetiology of trismus may be broadly classified as follows.²

1. Pathosis in TMJ like ankylosis and derangement of TMJ
2. Oodontogenic or non odontogenic infections
3. Neoplasm
4. Elongation of coronoid or styloid process
5. Presence of foreign body
6. Disturbance in trigeminal nerve
7. Pathosis in muscles of mastication like trauma, prolonged or extreme stretching
8. Systemic conditions like Tetanus, Scleroderma, Rheumatoid arthritis
9. Scar tissues/adhesions in region of maxilla and mandible caused due to trauma, burns etc
10. Subsequent to radiation therapy

Management of trismus

Treatment for trismus should be directed at eliminating its cause. Diagnostic assessment should be made before any type of therapy is applied. Treatment modalities include.^{2,3,4}

1. Heat treatment and electrotherapy
2. Analgesics and muscle relaxants
3. Surgical intervention
4. Physiotherapy and Soft Diet
5. Trismus appliances

Indications for trismus appliances.⁵

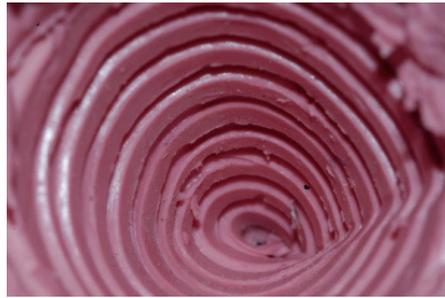
1. Reparative healing before formation of dense scar
2. Trismus resulting from 6-8 weeks of maxillo-mandibular fixation
3. Muscle atrophy, painful motions, weakness
4. Radiation therapy

Actions.⁵

1. Soften and stretch fibrous tissue
2. Increase the range of joint motion



Step 1



Step 2



Step 3

3. Restore circulatory efficiency
4. Increase muscular strength
5. Retains muscular dexterity

Classification of trismus appliances: ⁵

It can be externally activated or internally activated

Externally activated

It employs forcible stretching of elevator muscles by depressing the mandible. Force can be continuous or intermittent; light or heavy; elastic or inelastic.

Internally activated

It uses depressor muscles to open mouth. Action completely depends on patient motivation and cooperation

Types of trismus appliances⁵

1. Dynamic Bite opener introduced by Drane in 1967^{6,7}
2. Threaded tapered screw ⁸
3. Screw type mouth gag by Nakajimo et al
4. Tongue blades
5. Finger pressure advocated by Rouse⁹
6. Continuous dynamic jaw extension apparatus

Case report

55 year old female patient reported to the Department of Prosthodontics with reduced mouth opening following oncosurgical procedures on the left alveolar ridge.

Patient had difficulty to incise and masticate food along with speech problems.

On examination patient had only 14 mm of mouth opening. The normal range of mouth opening varies from patient to patient, within a range of 40–60 mm. The width of the index finger at the nail bed is between 17 and 19 mm. Thus, two fingers' breadth (40 mm) up to three fingers' breadth (54–57 mm) is the usual width of opening. ¹⁰

It was decided to fabricate tapered threaded screw. The reason for this selection is based on ease of fabrication in the dental clinic as a chair side procedure. It is an internally activated device which provides continuous or intermittent force which can be controlled by the patient.

Procedure for fabrication of tapered screw⁸

Step 1

Cone model was prepared in Type II Gypsum product. Modelling wax was cut into 2mm width and adapted on to the cone model at 5mm interval simulating the treads of screw

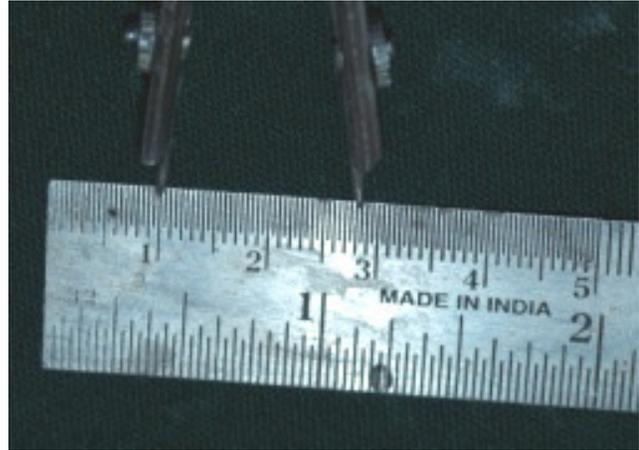
Step 2

Impression was made using irreversible hydrocolloid material.

Step 3

Self cured acrylic resin was mixed and packed into the impression and allowed to cure. The appliance thus obtained was finished and polished.

The patient was advised to place the screw between the posterior teeth and gradually turn it to wedge the



teeth apart. The treads guided the teeth along the increasing taper while the patient could control the timing and degree of pressure required to gradually increase the jaw separation. Inter incisal distance was found to be 18 mm

Application Schedule:

1. Stretch to the point of discomfort, not pain
2. Place appliance between teeth till snug
3. Measure opening by counting rings
4. Hold for 20 seconds
5. Withdraw and rest for 10 seconds
6. Insert to same degree of opening for 20 seconds
7. Repeat for 10-15 minutes, daily 4 times.

Conclusion

Trismus is a condition that impairs eating, interferes with oral hygiene, restricts access for dental procedures, and may adversely affect speech and facial appearance. The success of treatment depends on recognition of the cause and initiation of appropriate management. Trismus appliances used in conjunction with physical therapy is found to be very effective in the treatment of restricted mouth opening.

References:

1. Taylor EJ, ed. *Dorland's Illustrated Medical Dictionary*, 27th ed. Philadelphia: W.B. Saunders, 1998; p.1759.
2. Marien M Jr. Trismus: causes, differential diagnosis, and treatment. *Gen Dent*. 1997 Jul-Aug; 45(4):350-5.
3. Tveterås K, Kristensen S. The aetiology and pathogenesis of trismus. *Clin Otolaryngol Allied Sci*. 1986 Oct; 11(5):383-7.
4. P.J. Dhanrajani, O Jonaidel; Trismus: Aetiology, differential diagnosis and treatment. *Dent Update* 2002; 29: 88-94
5. Lund TW, Cohen JI. Trismus appliances and indications for their use. *Quint Int* 1993; 24: 275-279.
6. Brown KE. Dynamic opening device for mandibular trismus. *J Prostht Dent* 1968; 20: 438
7. Brunello DL, Mandikos MN. The use of a dynamic opening device in the treatment of radiation induced trismus. *Aust Prosthodont J*. 1995;9: 45-8.
8. John Beumer III, *Maxillofacial Rehabilitation Prosthodontic and Surgical Considerations*, Ishiyaku Euro-America, Inc, 1996; p. 525-527
9. Rouse PB. The role of physical therapist in support of maxillofacial patients. *J Prostht Dent* 1970; 24:193
10. Nelson SJ, Nowlin TP, Boeselt BJ. Consideration of linear and angular values of maximum mandibular opening. *Compend Contin Educ Dent* 1992; 13: 362-363