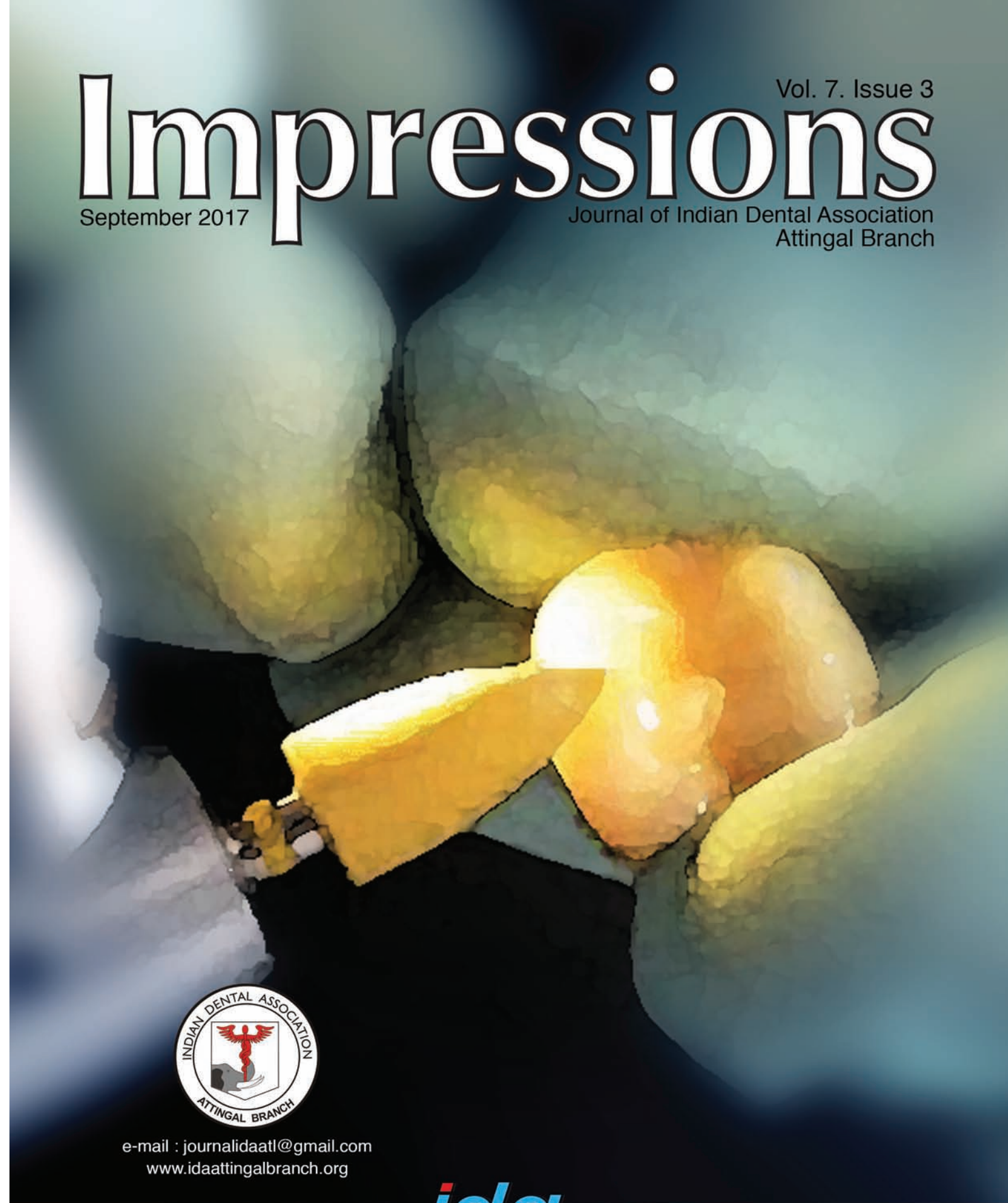
Vol. 7. Issue 3

# Impressions

September 2017

Journal of Indian Dental Association
Attingal Branch



e-mail : journalidaatl@gmail.com
www.idaattingalbranch.org

Best Journal State Award for 2013 and 2014

ida ATTINGAL BRANCH

Best Journal National Award - Runner up 2013 & Winner 2014

# Impressions

September 2017
❖ Vol. 7 ❖ Issue No. 3

Journal of Indian Dental Association Attingal Branch



Indian Dental Association
Attingal Branch

*Office:*

The Editor-In-Chief,
Impressions-Journal of IDA
Attingal Branch,
Prathyusha Dental Care
IIIrd Floor YCDC, Opp
Vydyuthi Bhavanam, Pattom,
Thiruvananthapuram-695004.

Mobile : 9447060374

e-mail: journalidaatl@gmail.com

# President's Message

Dear members,

Welcome to the third issue of journal of IDA Attingal branch. With the excellent support of my executive team and cooperation of our members, we surpassed 3/4th of the branch activities of 2017 in an astonishing manner.

The IMPRESSIONS 3rd issue itself is a very apt example of the efficiency of our executive teams untiring efforts in the form of fetching sponsorships, to collection of articles, to printing, to packing, to posting in time. Hats off to editor journal Dr Pradeep Dathan for his untiring efforts.

All the conveners did a marvellous job this year. The CDE wing deserves a special mention for the outstanding performances of programs conducted. CDH wing was also no exception in their performance like observing No Tobacco Day, Onam Celebration etc were some of the highlighted ones.

I convey my gratitude to each and every member of my Team of Office bearer's and to my beloved member's for your support and cooperation and wish to continue the same.

Thank you

With warm regards,

**Dr Deepak S Das**
President
IDA Attingal Branch.

# Secretary's Message

Dear colleagues,

It gives me great pleasure to forward this message in IMPRESSIONS 3rd issue.

This year the number of excellent papers submitted so far for our journal itself speaks volumes of interest shown by our members. Thanks to all the relentless efforts of all the members of the editorial board especially our editor journal Dr Pradeep C Dathan.

During the last three months, we conducted many CDE program's, CDH activities etc, among them the most appreciated program was the Onam celebration cum Charity program held on 17th sept 2017 at Sneehateeram (a unit of sister's of mercy) Mitirmala, Trivandrum District, where we spend a day with the mentally challenged, homeless women and provided them onam feast, dress, conducted games with orchestra.

Simillarly the last CDE held on 24th sept 2017 at Doubloon, Kallambalam was an outstanding one where we could provide lecture and free handson to all the attended participants in the topic Advanced Endodontics.

Our forthcoming programs for the last quarter of 2017 are 1) State CDE hosted by IDA Attingal and Trivandrum branch combined, 2) FDI CDE by IDA Kerala state at Amrita Dental college Ernakulam, 3) AGM and 4) Installation 2018.

I appeal to all our members to participate in all our future programs and make them a grand success.

Thank you,

With warm regards,

**Dr Anil Kumar**
Honorary Secretary
IDA Attingal Branch.

## ABOUT IDA ATTINGAL

IDA Attingal, symbolizes & represents, updates & educates, promotes & supports the local dental community of erstwhile Attingal, in delivering, quality dental health care to the general public. Maintenance of proper standards & ethical manner in practice, better interpersonal relations, as well as willingness to share knowledge, among members, has provided a high degree of respectability to the organization. Effective follow up of organizational proceedings at the state & national level by the branch executive, ensures that the members are kept abreast of all IDA activities. Regular representation at IDA events & healthy interaction with other branch members, has made IDA Attingal quite popular & a force to reckon. Adding to this would be a plethora of eminent leaders from the branch, who have raised to higher echelons in IDA. Through various Scientific programmes, presentations, journals & newsletters, the branch creates awareness of the latest advancements in dentistry, among members.

Impressions
Journal of Indian Dental Association Attingal Branch
September 2017
❖ Vol. 7 ❖ Issue No. 3

# Contents

# Editorial

## You can do practice and research in the clinic

Research in dentistry has been restricted to academic centres for many years. It is generally felt that this type of research is conducted in an ivory tower environment andhence the transfer ofknowledge from research findings into changes in clinical practice is extremely slow. The time lag between discovery and its generalized adoption by the dental profession is calculated to be 20 years. The academic research takes nearly 10 years to get published through a reputed journal. It may receive the attention of the profession provided it has direct clinical application and revolutionary in nature. Academic based clinical trials and practice based cross sectional studies on the same subject area might give conflicting results and this indicates the limitation of exclusive academic research. The best method to meet the situation is to involve practising clinicians into research.

This journal provides you clinically relevant information which is adapted to our clinical situations. A careful reading of our journal will definitely enrich your experience. At the same time practising clinicians should try to engage in clinical research and publish it through our journal. If you find any interesting situation, do not hesitate to take photographs and document it in your lap top. Your treatment details and the final results should also be recorded. You can send it to this journal and we would gladly publish it. The editorial office can help you in getting a good format for your article.

I request every member to cooperate with the idea of clinical research.

**Dr. Pradeep C. Dathan**
Editor, Impressions

OVERVIEW

# Polishing of newer ceramics

*** K. Chandrasekharan Nair, ** Bheemalingeswara Rao, ***Aswinikumar, ****Pradeep Dathan, *****Murukan P A,**

* Professor Emeritus, ** Professor of Prosthodontics, Vishnu Dental College, Bhimavaram, AP;
*** Reader of Prosthodontics, Amrita School of Dentistry, Kochi, Kerala;
**** Professor of Prosthodontics, Sri Sankara Dental College, Akathumuri, Kerala;
*****Professor of Prosthodontics, Annoor Dental College, Muvattupuzha, Kerala

*Send correspondence:* Dr. K. Chandrsekharan Nair, E-mail: chandrasekharannair.drk@gmail.com

Dental porcelain has optimized the aesthetic requirements, biological safety, strength to withstand masticatory efficiency and cost of the fixed dental prosthesis. Recent emphasis in research has been directed towards the enhancement of its strength, machinability and aesthetic properties. Metal ceramic restorations using conventional laboratory technology, were the most popular till recently but at present those are getting replaced with CAD/CAM restorations. Many of the ceramic materials are adapted to machining and that has forced impressions, casts, wax patterns and casting to be the techniques of the past with historic relevance. Common all ceramic materials currently used in dentistry andtheir clinical recommendations are as follows:

**Popularly used all ceramic materials**

| *Type of ceramic* | *Fabrication process* | *Use* |
|---|---|---|
| Feldspathic ceramic (Vitablocs Mark II,Cerec) | CAD/CAM | Inlays, onlays, veneers, anterior and posterior crowns |
| Leucite reinforced glass ceramic (IPS Empress | Pressable | Inlays, onlays, veneers, anterior crowns |
| Leucite reinforced glass ceramic (IPS Empress CAD/CAM | CAD/CAM | Inlays, onlays, veneers, anterior and posterior crowns |
| Lithium disilicate glass ceramic (IPS E.Max) | Pressable | Inlays, onlays, veneers, anterior and posterior crowns |
| Lithium disilicate glass ceramic (IPS E.MaxCAD/CAM) | CAD/CAM | Inlays, onlays, veneers, anterior and posteriorcrowns, anterior fixed partial dentures |
| Glass infiltrated alumina (In ceram Alumina) | CAD/CAM | Onlays, anterior and posterior crowns,anterior fixed partial dentures |
| Glass infiltrated alumina -ZrO added (In ceram Zirconia) | CAD/CAM | Onlays, posterior crowns and posterior fixed partial dentures |
| Polycrystalline alumina (Procera) | CAD/CAM | Anterior and posterior crowns |
| Polycrystalline zirconia (Y TZP) (Lava Zirconia) | CAD/CAM | Anterior crowns, posterior crowns, anterior andposterior fixed partial dentures |

Occlusal contacts of all ceramic restorations are frequently adjusted at the time of try-in and/or in the post-insertion phase. In minor occlusal adjustments, rubber abrasive polishing wheel is commonly used. A diamond is used when a significant adjustment is indicated. Reduction with diamond is to be followed with polishing to achieve a smooth surface. A polished ceramic surface is less abrasive than the glazed surface. Hence there is no need for reglazing of a ceramic restoration after polishing. Three different tools used in polishing ceramics are 1. diamonds 2. rubber polishers 3.polishing pastes.

Diamond: Fine grit diamond run at a speed of 20000 rpm is generally preferred. Micromotors will give greater control on the speed and cause less heat generation. Light touch will produce smoother surface. (Fig 1-3)

Flexible polishing tips/discs: Micromotors run at 10000/15000 rpm are ideal for flexible polishing tips. Rubber/Poly urethane is used for making these tips. Micromotors have constant torque even at a slow speed, so they are more effective than air driven hand pieces. These are available in coarse, medium and fine grits.. A recent addition is Feather Lite polishing system (Brasseler USA) which consists of diamond-impregnated, polyurethane, multi-use polishers with unique flexible spirals designed to separate and "feather" out. The feathering in the disc helps to maintain surface differentiation when polishing porcelain restorations, allowing the instrument to quickly adapt to any surface including occlusal, interproximal and contact areas. Available in a 26mm extra-oral head size and a 14mm intra-oral head size (Fig. 4-8). Polishing kits are available for both Lithium disilicate and Zirconia restorations.(Fig 9).

Polishing paste: Polishing paste contains diamond particles of 4 micron grit. It is available in syringes of 2g. While polishing, light force, not

Diamond Grit Sizes

| Code | Description | µ |
|---|---|---|
| SF | Super Fine | 30 |
| F | Fine | 50 |
| M | Medium | 107-120 |
| C | Coarse | 150-180 |
| SC | Super Coarse | 180-250 |

*Fig 1.* Grit sizes in diamonds

*Fig 2.* Different shapes of abrasive tips

*Fig 3.* Initial grinding with diamond

*Fig 4.* Medium grit polishing wheel

*Fig 5.* Fine rubber polishing tip

*Fig 6.* Coarse flexible spiral

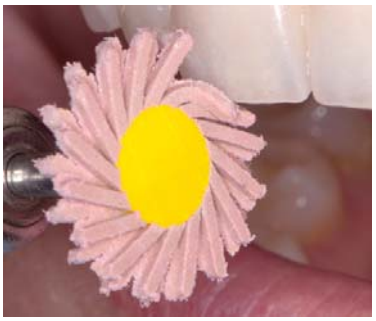


*Fig 7.* Medium flexible spiral

*Fig 8.* Fine flexible spiral


Fig 9. Ceramic polishing kits

*Fig 10.* Polishing paste applied with bristle brush


*Fig 11.* Dialite polishing system

exceeding 0.5 lbs of pressure is applied through rubber cup or bristle brush. Low speed of 6000rpm is used for 15 seconds to obtain high gloss.

Minimising the surface roughness is important in controlling aesthetics, wear, mechanical properties, and plaque accumulation of dental ceramic restorations. For metal ceramic restorations, surface finish can be achieved by either glazing or polishing. For all ceramic restorations, however, polishing is the only viable option since occlusal adjustment is performed after cementation. This limitation has stimulated greater interest among researchers in the field of ceramic polishing.A glazed surface was thought to produce smoother, more cleansable surfaces and stronger mechanical properties. Conventional polishing is not done routinely on all ceramics for fear that it would introduce more surface flaws and weaken the material. With advances in polishing instruments, it became possible to achieve acceptable surface smoothness by rotary equipment. Previous studies have established that in addition to producing smoother surfaces, polishing may also produce surfaces, which are less abrasive than glazed surfaces[1,2]. Grinding, polishing andoverglazing significantly increased the flexural strength of dental ceramics by 15% to 30 %, and re-firing of the ground and polished ceramic decreased the flexural strength significantly from 11% to 18 %.[3]

## Polishing Monolithic IPS e.max® and Zirconia

Hardness is a challenging factor to be recognized with newer monolithic materials. They are much harder than natural tooth structure and which makes them more difficult to re-polish after occlusal adjustments when compared to conventional porcelains. Harder surfaces won't self-adjust. A smooth zirconia surface left in supra-occlusion might not be abrasive but the potential risk of impact fracture cannot be ruled out. Obtaining superior polish prior to delivery of the restoration is important and having an effective system to adjust and re-polish after cementation of the restoration is also essential. Polishing systems used for softer conventional porcelains are not well suited for the harder monolithic materials. Another challenge of effective post-cementation re-polishing is generating sufficient torque from chairside air-driven handpieces. Dialite LD Extra-Oral Polisher System and Dialite ZR Zirconia Intra-Oral Adjustment/Polishing system were tested and found to be extremely effective when used with higher torque electric handpieces. These systems also offer special diamonds and stones (grinders) that are recommended for gross reduction if necessary. They generate less heat when grinding than conventional stones and diamonds, which helps minimize potential for post-processing adjustment. IPS e.max, which has a lower strength and hardness, is also polished reasonably well with air-driven handpieces but set at a higher air pressure and that might not be practical in a private dental office. (Fig 10, 11)[4,5]

The transition from metal ceramic to all ceramic systems is widely appreciated by clinicians mainly because of its aesthetics and strength. While strength increases, scope of adjustments of occlusion and repolishing gets minimized. Hence advanced polishing systems have to be made use of.

## References


1. Jagger DC, Harrison A. An in vitro investigation into the wear effects of unglazed, glazed, and polished porcelain on human enamel. Prosthet Dent 1994; 72: 320-3.
2. Jacobi R, Shillingburg HT, Duncanson MG. A comparison of the abrasiveness of six ceramic surfaces and gold.Prosthet Dent 1991; 66: 303-9.
3. Giordano R, Cima M, Pober R. Effects of surface finish on the flexural strength of feldspathic and aluminous dental ceramics. lnt 1 Prosthodont 1995; 8: 311- 9.
4. McLaren et al. Polishing monolithic IPS e.max and Zirconia restorations.BrasselerInside Dentistry.2015. 11:12-7. (Special Issue 3 insidedentistry.net/go/Clinical Success)
5. http://brasselerusadental.com (28.8.2017)

OVERVIEW

# Redefining complete denture esthetics

***Rubina Mathew Stephen, **Sudeep S., ***Dinesh N.**

*PG student **Prof & HOD ***Prof, Dept. of Prosthodontics,
PMS college of Dental Science and Research, Vattapara, Trivandrum

*Send correspondence:* Dr. Rubina Mathew Stephen, E-mail: rubinastephen@gmail.com

## Abstract

In rehabilitating complete denture patients, esthetics have an exemplified role. Denture aesthetics is the effect produced, which improves beauty and appeal for an individual. Hence, an artistic effort is required in treating edentulous patient. The changing physical personality traits of a patient as well as physiological age changes in the tissues must be considered. Conventional pearl like dentures can be redefined by incorporating personnel characteristic features in teeth and denture bases in order to contribute to a Life-Like appearance. This should begin right from treatment planning onwards. This review paper aims at putting forward the various principles involved in redefinition of a complete denture.

**MATERIALS AND METHODS**

A review of literature was done which engaged most of the articles published in peer-reviewed journals pertaining to the subject published in English and limited to "Characterisation" from 1953 to 2015, in Medline (PubMed) and Google Scholar.

## Introduction

Characterization is a procedure in which the character or collective qualities of a person are introduced in the complete denture, either by modification of teeth or denture bases, to make it appear more natural for that particular person.[1] Complete dentures must be esthetic as well as functional. The two elements that must be considered in denture esthetics are teeth and their supporting denture base.

Complete denture can be characterized by two basic methods.

1. Characterization by selection, arrangement and modification of artificial teeth.
2. Characterization by tinting the denture bases.[1,2]

| Characterization of artificial teeth | Characterization of denture bases |
|---|---|
| Modifications in teeth arrangement | contouring |
| Modifications in shape of tooth | • Festooning |
| | • Stippling |
| | • Alveolar eminance |
| | • Gum fit dentures |
| | tinting |
| | • Custom |
| | • Photocured denture coating |
| | • Light cured gum shading |
| Modifications in specific tooth | |

**Characterization of atificial teeth**

Fisher stated that gender, personality, and age can be used as guidelines for tooth selection, arrangement, and characterization to "enhance the natural appearance of the individual.

**Teeth arrangement**

1. Varying the direction of the long axis of teeth.[1,2,3]

2. Place the teeth so that the tips of the maxillary lateral incisors show when the patient speaks seriously depending on the age and sex, less for old than for young people and more for woman than for men.[1,2,3]

3. Create asymmetry in the divergences of the proximal surfaces of the teeth from the contact points.[1,2,3]

4. Use an eccentric midline.[3,4]

5. Place one maxillary central and lateral incisor parallel to the midline and rotate the other central and lateral incisors slightly in a posterior direction.[2]

6. Place one maxillary central incisor slightly in an anterior direction to the other central incisor.[2]

7. Place the neck of one maxillary central incisor in a posterior direction and the neck of other central incisor in an anterior direction.[2]

8. Create asymmetry for the maxillary right and left cuspids. Rotate one in a posterior direction than the other.[3]

9. Overlapping, tilting, rotation and incisal variations[2]

10. Slight diastema can be created between the lateral incisor and the cuspid on one side. Diastema given should exceed 2-3mm and should be wider at the incisal edge than the base otherwise fibrous food tends to be trapped and can be a source of embarrassment.[4] (Fig 2)

**Shape modification**

1. Teeth abrade with age, so reshaping the incisal edges and mesiodistal diameter give this appearance.[5] (Fig 3)

2. Peg lateral can be given to patients who had them (Fig 4)

**Modifications on specific tooth**

1. A hair line crack can be given in the teeth.
2. Gold or alloy restorations can be placed[6]
3. Silver filling can be given on posterior teeth.
4. Cast crown can be given on posterior teeth.[7]
5. A discolored tooth (as R.C.T treated) can be shown by selecting one or two teeth of darker shade.
6. Dental jewellery can be given on lateral incisors.
7. Gingival tissues recede with age. Selecting a long tooth, contouring the wax to show gingival recession and then staining it a bit, can reproduce this recession.[7]
8. Tobacco staining on tooth

**characterization of denture bases**

Pound in 1951 was the first to suggest a method of tinting acrylic denture bases to simulate the gingival colour. Kemnitzer used a combination of blue and brown stain to reproduce the melanotic pigmentation of the gingiva.

Indication for characterization of denture base[2]

1. Patients with an active upper lip.
2. Patients with a prominent pre-maxillary process.

*Fig 1* Mild diastema

*Fig 2* Overlappig, Tilting, Rotation of incisors

*Fig 3* Shape modification of tooth

*Fig 4* Peg lateral

*Fig 5* Stippling, Festooning, Alveolar prominence

*Fig 6* Flangeless Dentures

*Fig 7* Light cured gum shading

3. Actors, singers and others who may expose gum tissues areas during their performances.

4. The psychological acceptance of the dentures by the patient.

**Contouring of denture bases**

1. Festooning (Fig 5)

Intermittent elongated prominences corresponding to the root contours, light is randomly reflected and dispersed giving natural appearance.[8]

2. Stippling

Orange peel appearance in the attached gingiva. Positive stippling seems to collect less debris and calculus, and is easier to clean than the indentations made by negative stippling techniques. Care must be taken in waxing the denture to the proper thickness and finishing and polishing the denture after processing[8]

Various methods used are

1) toothbrush technique.
2) offset bur technique.
3) blow wax technique.

3. Alveolar eminence

The labial flange of denture should show a series of swellings corresponding to the alveolar eminencies over the roots of the teeth markedly in the anterior region and less markedly in the posterior region.[8]

4. Gum fit dentures

In cases with severe maxillary proclination, avoiding the labial flange, in order not to accentuate the labial fullness further, using careful techniques not to compromise on retention is an accepted modality. Hence, the labial flange of the denture base is trimmed to make it gum fit (Fig 6)

**Tinting of denture bases**

1. Custom tinting

Color characterization may be done by the dentist or technician after the denture has been processed using

Denture tinting chart, Soft tissue shade guide, #6 camel hair brush, Acrylic resin stains or shade modifiers in a variety of colors including red, brown and black, Dappen dishes and Pressure pot or a light curing unit for curing the stains.[9]

2. Photocured denture coating

The clear coating provides a hard, high gloss which makes the polishing of dentures unnecessary. The coating seals the surface which promotes the color stability of the base and the stains. Abrasion resistance of the denture base and custom staining should be greatly improved. It is claimed that the coatings render the denture more wettable and retentive.[9]

3. Light cured gum shading

It consist of micro filled composite resin, can be applied in multilayered technique and can delivers unlimited possibilities for gingival reproduction.[9]

**Ideal requirements of tinting material**

1. It should be readily miscible with methyl methacrylate resin.

2. It should be non-toxic

3. It should not add appreciable bulk to denture bases.

4. It should be stable and non-fading.

5. It should be resistant to loss from abrasion in cleaning and in normal function.

6. It should not alter the properties of the denture base resins.[9]

**Conclusion**

Although this review discusses most of the characterization methods in complete dentures, the real success of a denture is the patient's acceptability. Thus complete denture fabrication not only replaces missing teeth but also restores esthetics, phonetics and function. The natural look can be reproduced so faithfully that dentures are no longer a cosmetic problem. Therefore, every dentist should work towards preserving and enhancing a pleasing smile without impairing function.

**Refrences**


1. Dr. Ravi Kumar C.M., Dr. Kumar D.R.V., Dr. Jain Sorabh R., Dr. Bajaj Krushnan V; Virtualising Natural Effects in Complete Dentures; Sch. J. App. Med. Sci., 2014; 2(1D):364-368.
2. Rajeev Srivastava, Vivek Choukse:Characterization of Complete Denture: INTERNATIONAL JOURNAL OF DENTAL CLINICS 2011:3(1):56-59
3. Martone AL. Effects of complete dentures on facial esthetics. The Journal of Prosthetic Dentistry 1964;14(2):231-55.
4. Lombardi RE. The principles of visual perception and their clinical application to denture esthetics. The Journal of Prosthetic Dentistry 1973; 29 (4):358-82.
5. Esposito SJ. Esthetics for denture patients. The Journal of Prosthetic Dentistry1980;44(6):608-15.
6. Levin EI. Dental esthetics and the golden proportion. The Journal of Prosthetic Dentistry 1978; 40(3):244-52.
7. Ku YC, Shen YF. Simple method for making a metal crown for a complete denture. Journal of Prosthetic Dentistry 2001;86(2):214-5.
8. Tillman EJ. Molding and staining acrylic resin anterior teeth. The Journal of Prosthetic Dentistry 1955; 5(4):497-507.
9. Becker CM, Smith DE, Nicholls JI. The comparison of denture-base processing techniques. Part II. Dimensional changes due to processing. The Journal of Prosthetic Dentistry 1977; 37(4):450-9.

OVERVIEW

# Neutral zone concept in complete denture

*** Himanath K., ** Sudeep S., ***Dinesh N.**

*PG Student, **Professor & HOD, ***Professor, Dept. of Prosthodontics, PMS College of Dental Science and Research,Vattappara, Trivandrum

*Send correspondence:* Dr. Himanath K, E-mail: himanathekm@gmail.com

## Abstract

When all of the remaining natural teeth are removed, there exists within the oral cavity a void that may be called the potential denture space. Within the denture space there is an area that has been termed the neutral zone. The neutral zone is that area in the mouth where the forces of the tongue pressing outward are neutralized by the forces of the cheeks and lips pressing inward.Soft tissues that form the internal and external boundaries of the denture space greatly affect and influence the stability of the dentures and help to determine the peripheral borders, tooth position, and external contours of the dentures.So teeth placement is not over the crest of ridge or buccal or lingual to it but should be dictated by the muscles and vary for different persons.

## Introduction

The primary objective of complete denture prosthesis is to construct dentures that will satisfy the three basic requirements of the edentulous patient: maximum comfort, efficiency, and esthetic appearance. This objective can be achieved only if the dentures are both stable and retentive. Complete dentures are primarily mechanical appliances, but since they function in the oral cavity, they must be fashioned so that they are in harmony with normal neuromuscular function. All oral functions -speech, mastication, swallowing, smiling, laughing involve the synergistic actions of the tongue, lips, cheeks, and floor of the mouth, which are very complex and highly individual in themselves. Failure to recognize the cardinal importance of tooth position, flange form, and contour may result many times in dentures that are unstable and unsatisfactory, even though skillfully designed and expertly constructed. The coordination of complete dentures with neuromuscular function is the foundation of successful, stable dentures[1].

### The neutral zone and denture space

When all of the remaining natural teeth are removed, there exists within the oral cavity a void that may be called the potential denture space. Within the denture space there is an area that has been termed the neutral zone. The neutral zone is that area in the mouth where the forces of the tongue pressing outward are neutralized by the forces of the cheeks and lips pressing inward. Since these forces are developed through muscular contraction during the various functions of chewing, speaking, and swallowing, they vary in magnitude and direction in different individuals and in different periods of life. The way these forces are directed against the dentures will either help, to stabilize them or will tend to dislodge them. These soft tissues that form the internal and external boundaries of the denture space greatly affect and influence the stability of the dentures and help to determine the peripheral borders, tooth position, and external contours of the dentures. Therefore, a basic understanding of the anatomy and physiology of the muscles is essential.[1]

Sir Wilfred Fish[21] described a denture as having three surfaces, with each surface playing an important role in the overall fit, stability, and comfort of the denture – the impression surface, occlusal surface and the polished surface. The third surface is mostly denture base material, but it consists of those surfaces of the teeth that are not contacting or articulating surfaces. The buccal and lingual surfaces of the posterior teeth and the labial and lingual surfaces of the lower anterior teeth are not part of the occlusal surface but are part of the polished surface of the denture. The upper anterior teeth actually belong to two surfaces, both the occlusal and the polished surfaces. When the teeth are in contact, the lingual surfaces of the upper anterior teeth are part of the occlusal surface. When the teeth are apart as in speaking or at rest, these surfaces are part of the polished surface. The polished surface is in contact with the cheeks, lips, and tongue. One can visualize that, based on a square unit of area, the polished surface is as large as or larger than the impression and occlusal surfaces combined, depending on the anatomic structure.

### Influence of forces on denture surfaces.

The more the ridge loss, the less the area of the denture base and the less the influence of the impression surface area will have on the stability

and retention of the denture.As the surface area of the impression surface decreases and polished surface area increases, the development and contour of the polished surface becomes more critical. In other words, where more of the ridge has been lost, the more the denture stability and retention is dependent on the polished surface than on the impression surface.

The forces on the polished surface are constantly changing in magnitude and direction during swallowing, speaking, and mastication. It is only when the mouth is completely at rest that the forces are constant. When the occlusal surfaces of the teeth are not in contact, the stability of the denture is determined by the fit of the impression surface and the direction and amount of forces transmitted through the polished surface.

If a person had his teeth in contact all the time, the polished surface would be relatively unimportant in denture stability. Conversely, if a person never brought his teeth into contact, the occlusal surface would be relatively unimportant and the stability would be dependent on the forces on the polished surface as transmitted to the impression surface. In order to construct dentures that function properly not only in chewing but also in speaking and swallowing, we must develop the fit and contour of the polished surface just as accurately and meticulously as the fit and contour of the impression surface and the occlusal surface. It is therefore important to have a knowledge of the anatomic structures and their functions and we must utilize techniques and materials to record the effect of these structures on the polished surface and incorporate it into the denture.

The dental arch is formed by the effect of forces exerted on the teeth by the muscles of the tongue, lips, and cheeks along with certain inherent and local environmental factors. When the teeth are erupting into the mouth during childhood and adolescence the muscular activity and habits that develop will continue through life. Even after the teeth are lost, the forces created by these habits and actions still persist and will have a great influence upon any complete or extensive partial removable prosthesis that is placed into the mouth. It is therefore extremely important that the teeth be placed in that part of the mouth and with an arch form that falls within the area formed by muscular forces.[1]

## Direction of forces

For the muscular forces to be of a stabilizing nature, the dentures must be so constructed that they will receive these forces at the proper angle. Dr.Fish[21] described the cross section of stable dentures in the molar area to be triangular in shape, with the tooth being the apex and the denture periphery the base of a triangle. A force exerted on an inclined plane may be broken down into two components. One component acts in the direction parallel to the inclined plane. The other component, called normal force, acts perpendicularly to the inclined plane. If the inclined planes of the polished surface are properly fashioned and the forces are of equal magnitude, the resultant normal force will be in a seating direction. By the same token, if the dentures are triangular, but not properly located within the neutral zone, the lateral force will be unequal and not provide the equilibrium necessary for a stable denture. This will result either in the dislodgement of the denture or unequal pressure on the ridge.[1]

## Reversed sequence in denture construction

The usual sequence for complete denture is to make primary impressions, make final impressions, and then fabricate stabilized bases. Occlusal rims is used to establish occlusal vertical dimension and centric relation. With the neutral zone approach to complete dentures, the procedure is reversed. Individual trays are constructed first. These trays are adjusted in the mouth for overextensions and checked for stability during opening, swallowing and speaking. Next modelling compound is used to fabricate occlusal rims. These rims, which are molded by muscle function, locate the patients neutral zone. After a tentative vertical dimension and centric relation have been established, the final impressions are made with a closed mouth procedure. Only when final impressions are completed are the occlusal vertical dimension and centric relation finally determined.

The shape of the polished cameo surface will determine whether the muscular forces will stabilize or dislodge the denture. Additionally, this helps patients control their dentures even when the residual ridges have atrophied and the fit is no longer accurate.[2] The proper position of the teeth is not in the center of the ridge, nor labial or buccal to it, but where the cheek pressure and tongue pressure balance each other.

Mahmoud et al[3] found that the residual ridge type (prominent ridges and flat ridges) had no effect on the neutral zone, suggesting that muscular forces rather than the ridge itself were the determining factor. The authors also demonstrated that the width of the neutral zone is smallest at the occlusal plane level and increases as it goes up and down and that as the occlusal vertical dimension increases, the width of the neutral zone also increases and vice versa

The techniques most commonly used for recording the neutral zone were found to be

swallowing[4,5,6] and phonetics[7,8,9]. However, other techniques such as sipping[8,9,10] water, licking[4,8,9] smiling[8,11,12] pursing the lips[9,13,14] sucking[6,15,16] masticating[17] mouth exercises (including tongue movements, blowing, protruding of the tongue, exercise movements of the lips, cheek, and tongue, facial expression, opening and closing)[3,5,6,14,18] and whistling[11,18] have also been reported.

## Materials

Tench et al[1] were the first in this field and have proposed modelling plastic impression compound as the material to be used for recording the neutral zone. Although this advice is widely followed other materials such as tissue conditioner,wax, zinc oxide eugenol impression material,silicone material, chairside relining material, and acrylic resin are also described for this technique[19]. These materials are either used for the initial recording of the neutral zone or at the evaluation appointment. Modelling plastic impression compound, being a thermoplastic material, is easy to manage and has the advantages of low cost and ease of availability, whereas wax is temporarily stable and can be contoured over a period of time by functional movements. A tissue-conditioning material was preferred by many authors because of the ease of mixing, elective initial viscosity, and slow-setting properties that enabled capture of the movable tissue morphology in the functional state. Moreover, this material also allows for an incremental molding procedure, which is important in patients with focal neurological deficits and slow or false reactions to various commands[9,20]. A disadvantage of this material is its relatively high cost. Light-polymerized acrylic resin provides sufficient working time and polishes to a high luster; however, irritation due to the monomer may be a problem[13]. Whichever materials are used for recording the neutral zone, it seems that 2 factors cannot be ignored: the neutral zone should be recorded at an established occlusal vertical dimension, and the material used for recording should be reasonably slow setting so that oral musculature shapes it into proper contour and dimension[17].

## Conclusion

The neutral zone philosophy is based on the concept that for each individual there exists within,the denture space,a specific area where the function of the musculature will not unseat the denture and, at the same time,where the forces generated by the tongue are neutralized by forces generated by the lips and cheeks. So teeth placement is not over the crest of ridge or buccal or lingual to it but should be dictated by the muscles and vary for different persons.

The neutral zone has not been given enough importance,in the literature but as a determinant of occlusion,it cannot be ignored. Complete denture failures are often related to noncompliance with neutral zone factors.

Regardless of the method of treatment, any part of dentition out of harmony with the neutral zone will result in instability,interference with functionor some degree of discomfort to the patient. Thus neutral zone must be considered as an important factor while rehabilitating the edentulous patients. The operator should try to neutralize forces acting on complete dentures, which will make the prosthesis more functionally physiologically and psychologically acceptable to the patient.

## References


1) Victor E Beresin and Frank J Schiesser. The neutral zone in complete dentures; .J Prosthet Dent Feb 2006; vol 95(2) 93-100
2) Schiesser FJ. The neutral zone and polished surfaces in complete dentures. J Prosthet Dent 1964;14:854-65
3) Khamis M, Razek A, Abdalla F. Two-dimensional study of the neutral zone at different occlusal vertical heights. J Prosthet Dent 1981;46:484-9.
4) Miller WP, Monteith B, Heath MR. The effect of variation of the lingual shape of mandibular complete dentures on lingual resistance to lifting forces. Gerodontology 1998;15:113-9
5) McCord JF, Grant AA. Impression making. Br Dent J 2000;188:484-92.
6) Walsh JF, Walsh T. Muscle-formed complete mandibular dentures. J Prosthet Dent 1976;35:254-8
7) Ikebe K, Okuno I, Nokubi T. Effect of adding impression material to mandibular denture space in Piezography. J Oral Rehabil 2006;33:409-15.
8) Gahan MJ, Walmsley AD. The neutral zone impression revisited. Br Dent J 2005;198:269-72
9) Kursoglu P, Ari N, Calikkocaoglu S. Using tissue conditioner material in neutral zone technique. N Y State Dent J 2007;73:40-2
10) Cagna DR, Massad JJ, Schiesser FJ. The neutral zone revisited: from historical concepts to modern application. J Prosthet Dent 2009;101:405-12.
11) Raja HZ, Saleem MN. Neutral zone dentures versus conventional dentures in diverse edentulous periods. Biomedica 2009;25:136-45
12) Sadighpour L, Geramipanah F, Falahi S, Memarian M. Using neutral zone concept in prosthodontic treatment of a patient with brain surgery: a clinical report. J Prosthodont Res 2011;55:117-20
13) Ohkubo C, Hanatani S, Hosoi T, Mizuno Y. Neutral zone approach for denture fabrication for a partial glossectomy patient: a clinical report. J Prosthet Dent 2000;84:390-3.
14) Suzuki Y, Ohkubo C, Hosoi T. Implant placement for mandibular overdentures using the neutral zone concept. Prosthodont Res Pract 2006;5:109-12.
15) Fahmy FM. The position of the neutral zone in relation to the alveolar ridge. J Prosthet Dent 1992;67:805-9
16) Raybin NH. The Polished Surface of Complete Dentures. J Prosthet Dent 1963;13:236-9
17) Lynch CD, Allen PF. Overcoming the unstable mandibular complete denture: the neutral zone impression technique. Dent Update 2006;33:21-2, 24-6
18) Russell AF. The reciprocal lower complete denture. J Prosthet Dent 1959;9:180-90
19) Amit Porwal,Keiichi Sasaki: Current status of the neutral zone: A literature review;, (J Prosthet Dent 2013;109:129-134)
20) Karkazis HC. Prosthodontic management of a patient with neurological disorders after resection of an acoustic neurinoma: a clinical report. J Prosthet Dent 2002;87:419-22
21) Fish E W.Principles of full denture prosthesis 7 th edition. London,Staples press Ltd,1948.

OVERVIEW

# Nutrigenomics—a way to healthy periodontium

*** Arathi S., **Sarath C, ***Seema G**

* Post Graduate Student, **Senior Lecturer, ***Professor, Department of Periodontics,
Sri Sankara Dental College, Akathumuri, Varkala, Trivandrum.

*Send correspondence:* Dr. Arathi S., *E-mail: arathisreedeviapril17@gmail.com*

## Abstract

Nutrition influences growth, development, metabolic activities of periodontium.In nutritionally deprived state, periodontal diseases are evident. The extent and intensity of gingival inflammation is indirectly affected by the innate resistance of periodontal tissues to infection. The differnce in clinical response of a disease in individual is contibuted by their genetic constitution which is unique for every individual.Thats how the concept of nutrigenomics came into existence in the field of treatment.It is the relationship between nurition and genome.It deals with study of interaction between nutrients and dietary bioactive with gene at molecular level.

**Keywords:** Periodontitis,Nutrition, nutrigenomics, vitamins.

## Introduction

Periodontitis is defined as "an inflammatory disease of the supportive tissues of the teeth caused by specific microorganisms or groups of specific microorganisms, resulting in progressive destruction of the periodontal ligament and alveolar bone with increased probing depth formation, recession, or both"[1]

Nutrients have major role in maintaining periodontal health.It may be important in redressing the balance between microbial challenge and the host response because it has been implicated in a number of inflammatory diseases and conditions, including type 2 diabetes, cardiovascular disease, rheumatoid arthritis and inflammatory bowel disease, all of which have also been associated with periodontal disease[2]. Destruction of periodontal tissues is brought about by the production of inflammatory factors released from immune cells. Polymorphonuclear leukocytes are attracted to the site of periodontitis due to the presence of bacteria. Following stimulation by bacterial antigens, leukocytes produce reactiveoxidative species (ROS), enzymes, and defensins that degrade the pathogens during phagocytosis.[3] Limited number of studies do give some indication of a relationship between various nutrients and the periodontal diseases. Results from a prospective, observational study carried out over 14 years revealed that men with a high consumption of wholegrain were 23% less likely to develop periodontitis than were those who had the lowest consumption of wholegrain.[2]

The aim of this article is to reflect the impact of nutrients on periodontium and the significance of nutrigenomics.

## Definition of nutrient and its classification

Nutrient is defined as a source of nourishment, such as food, that can be metabolized by an organism to give energy and build tissue whereas nutrition is the organic process by which an organism assimilates food and uses it for growth and maintenance. Nutrients can be divided into six major classes, i.e. fats, carbohydrates, proteins, minerals, vitamins, and water; these can be further subdivided into two broad categories, "macronutrients" (fats, carbohydrates, and proteins) which are required in large quantities from the diet and "micronutrients" (minerals, vitamins, trace elements, amino acids, and polyunsaturated fatty acids [PUFA]) which are

only required in small quantities in the diet and which are essential for a range of biological processes important in supporting optimal health. (1) Specific nutrients (antioxidant vitamins A, C, E) and trace element selenium, copper, zinc can modulate the immune and inflammatory responses that maintain epithelial cell integrity and structure. The nutrients get depleted during inflammation with generation of Reactive Oxygen Species (ROS) causing damage to the cellular tissues.[3] Vitamin C acts as a powerful scavenger of free radical. The association between low intake of vitamin C and occurrence of periodontitis has been demonstrated, in a study by Nissada 2010.[2] Vitamin E terminates free radical chain reaction, stabilizes membrane structure. It is shown to have mitigatory effects on inflammation and collagen breakdown. A low level of vitamin E in gingival tissues of periodontitis patients has been reported.[5]

## Nutrigenomics

Nutrigenetics and nutrigenomics are defined as the science of the effects of genetic variation on dietary responses and the role of nutrients and bioactive food compounds in gene expression, respectively[6]. It is important to note that both terms are closely related but not interchangeable. Nutrigenetics research involves genetic inheritance and its variations in the response to nutrients and dietary patterns[7] whereas nutrigenomics investigations focus on dietary effects on genome stability, epigenome alterations, RNA and miRNA alterations, protein expression and metabolite changes.The term genomics describes the process by which all genes present in the genome of a given species can be mapped, sequenced and characterized[8]. Nutrigenomics aims to reveal the relationship between nutrition and the genome and to provide the scientific basis for improved public health through dietary means. Researchers have demonstrated antioxidant depletion in periodontitis locally in the periodontium and within plasma, where investigators established an inverse relationship between reduced concentrations of plasma total antioxidants and vitamin C and increased prevalence of periodontitis.[9]


## History

- 1st April 1869, the first isolation of DNA was made by Friedrich Miescher.
- 25th April 1953, Watson and Crick published "the molecular structure of DNA".
- 1997, the first nutrigenomics company was launched.
- 1999, the name nutritional genomics was changed to genomics by Nancy Fogg-Johnson and Alex Merolli which provides powerful means of discovering hereditary factors in disease[10]

## Practical applications of nutrigenomics[11]

1.Genes and proteins expressed differentially in health and disease that are modifiable by nutrients are identified.

2. Genes, proteins, and metabolites are influenced by specific nutrients that are known to be beneficial or harmful are identified.

3. Genetic variations that alter the nutrient–gene interactions in applications 1 and 2 are identified.

## Possible role for nutrigenomics

Plasma lipid concentrations are major modifiable risk factors for cardiovascular disease. Peroxisome proliferator activated receptor alpha is primarily expressed in tissues that actively oxidize fattyacids, including the liver, muscle; also expressed in vascular endothelium, smooth muscle, and in cells of the immune system. Fibrates, the synthetic ligands for peroxisome proliferator activated receptor alpha. Treatment with fibrates has been shown to lower the amount of plasma triglycerides, raise the amount of high-density-lipoprotein cholesterol and be preventative against cardiovascular disease[12].

## Diabetes and periodontal disease:

The vast majority of cases of DM fall into two broad etiopathogenetic categories: type 1 and type 2 DM (T1DM and T2DM, respectively). T1DM, previously named insulin-dependent diabetes or juvenile-onset diabetes, results from cellular-

mediated autoimmune destruction of pancreatic â cells; therefore, patients are dependent on exogenous insulin. Individuals with T1DM are considered to have a genetic predisposition, although environmental factors, such as dietary components, also contribute to T1DM development.[13] Type 2 diabetes is a complex trait characterized by decreased insulin secretion and decreased insulin action at target tissues. A number of observational studies have reported an increased prevalence of periodontal disease in patients with type 2 diabetes and it is now widely accepted in the periodontal profession that type 2 diabetes is an important risk factor for periodontal disease.[11] Common genetic risk factors may be partly responsible for cross-susceptibility between periodontal disease and type 2 diabetes and that these genetic risk factors may be modulated by diet.A more recent population-based study has confirmed this finding and identified a significant association between the variant genotypes of both interleukin-1A (interleukin-1A-889) and interleukin-1B (interleukin-1B+3954 and interleukin-1B-511) and periodontitis in subjects with type 2 diabetes, which was not seen in nondiabetic controls.[14]

### Interaction of vitamins-gene in DM pathogenesis

Another group of nutrients with anti-diabetic properties are vitamins. For example, it has been demonstrated that vitamin D may improve β cell function by limiting chemokine expression, partially normalizing the expression of major histocompatibility complex (MHC) class I molecules and decreasing the density of MHC class I proteins on β cells.[15] Vitamin D modifies the expression of approximately 250 genes, particularly genes related to functional groups involved in immune responses, chemotaxis, cell death and pancreatic β cell function/phenotype[16].

## Conclusion

Nutrient-deprived people will not heal as fast; gingivitis and periodontal disease will progress faster if patients have poor diets. Diet has a modifying role in the initiation and progression of periodontal disease. Currently the focus is on nutrient- gene interactions, its influence in inflammatory conditions including periodontal diseases. Thus it gives an idea about how to modify the dietary factors so that we can maintain periodontal tissues in healthy state.

## References


1. Newman, G.M.; Takei, H.H.; Klokkevold, R.P.; Carranza, A.F. Carranza's Clinical Periodontology. Classification of Diseases and Conditions Affecting the Periodontium. In Carranza's Clinical Periodontology, 12th ed.; Michael, G.N., Henry, H.T., Perry, R.K., Fermín, A.C., Eds.; Elsevier: Amsterdam, The Netherlands, 2012; pp. 45–67.
2. Van der Velden U, Kuzmanova D, Chapple IL. Micronutritional approaches to periodontal therapy. J Clin Periodontol 2011;38 Suppl 11:142-58
3. Semba RD, Tang AM. Micronutrients and the pathogenesis of human immunodeficiency virus infection. Br J Nutr 1999:81(3):181–189
4. Enwonwu CO, Ritchie CS. Nutrition and inflammatory markers. J Am Dent Assoc 2007: 138: 70–73
5. Slade EW, Bartuska D, Cohen DW, Rose LF. Vitamin E and periodontal disease. J Periodontol 1976;47(6):352-354.
6. Corella, D.; Ordovas, J.M. Nutrigenomics in cardiovascular medicine. Circ. Cardiovasc. Genet. 2009, 2, 637–651.
7. Brennan, R.O. Nutrigenetics: New Concepts for Relieving Hypoglycemia; M. Evans and Company Inc.: New York, NY, USA, 1976.
8. Kramer JA, LeDeaux J, Butteiger D, Young T, Crankshaw C, Harlow H, Kier L, Bhat BG. Transcription profiling in rat liver in response to dietary docosahexaenoic acid implicates stearoyl-coenzyme a desaturase as a nutritional target for lipid lowering. J Nutr 2003: 133: 57–66.
9) Guzman S, Karima M, Wang HY, Van Dyke TE. Association between interleukin-1 genotype and periodontal disease in a diabetic population. J Periodontol 2003: 74: 1183-1190.
10) Schulze M.B., Hu F.B. Primary prevention of diabetes: What can be done and how much can be prevented? Annu. Rev. Public Health. 2005;26:445–467.
11) Merchant AT, Pitiphat W, Franz M, Joshipura KJ. Wholegrain and fiber intakes and periodontitis risk in men. Am J Clin Nutr 2006: 83: 1395–1400
12) Low YL, Tai ES. Understanding diet-gene interactions: lessons from studying nutrigenomics and cardiovascular disease. Mutat Res 2007: 622: 7–13.
13) Dib S.A., Gomes M.B. Etiopathogenesis of type 1 diabetes mellitus: Prognostic factors for the evolution of residual beta cell function. Diabetol. Metab. Syndr. 2009;1:1–8. doi: 10.1186/1758-5996-1-25
14) Struch F, Dau M, Schwahn C, Biffar B, Kocher T, Meisel P. Interleukin-1 gene polymorphism, diabetes, and periodontitis: results from the study of health in Pomerania (SHIP). J Periodontol 2008: 79: 501–507.
15) Gysemans, C.A.; Cardozo, A.K.; Callewaert, H.; Giulietti, A.; Hulshagen, L.; Bouillon, R.; Eizirik, D.L.; Mathieu, C. 1,25-Dihydroxyvitamin D3 modulates expression of chemokines and cytokines in pancreatic islets: Implications for prevention of diabetes in nonobese diabetic mice. Endocrinology 2005, 146, 1956–1964.
16) Wolden-Kirk, H.; Rondas, D.; Bugliani, M.; Korf, H.; van lommel, L.; Brusgaard, K.; Christesen, H.T.; Schuit, F.; Proost, P.; Masini, M.; et al. Discovery of molecular pathways mediating 1,25-dihydroxyvitamin D3 protection against cytokine-induced inflammation and damage of human and male mouse islets of Langerhans. Endocrinology 2014, 155, 736–747.

# Temporomandibular Disorders (Part 2)- Diagnosis diseases

*** T. Mohan Kumar, ** Pradeep C Dathan, ***Smitha Raveendran**

* Prof & Head Dept of Prosthodontics, Mahe Institute of Dental Sciences, Mahe; ** Prof & Head Dept of Prosthodontics, *** Reader Dept of Prosthodontics, Sri Sankara Dental College, Varkala

*Send correspondence:* Dr. T. Mohan Kumar, E-mail: tmkumar59@gmail.com

This is the second part in the series on TMD which will briefly consider the diagnosis of TMDS.

The previous article addressed the general aspects of TMD including definition, classification and nomenclature of this condition. The classification highlighted was the internationally accepted Research Diagnostic Criteria for TMD (RDC/TMD). This classification divides TMD into two Axes- Axis I and Axis II.

This series of articles will as mentioned earlier, focus only on the Axis I disorders. This is because these are the most commonly encountered among the various categories of TMD, by General Dental Practitioners(GDP).

Axis I disorders, as the reader would recall, has 3 subdivisions-namely, Myalgia, Internal Derangements and Arthralgia.

Myalgia includes Myofacial pain and Myofacial pain with limited opening.

Internal Derangements are subdivided into 3. Viz:

(a) opening/closing of the mouth accompanied by clicking,

(b) opening of mouth with clicking with limited opening,

(c) opening <25 mm but without clicking (closed lock).

Arthraligia- is the presence of continuous pain in the joint not relieved by rest.

Thus it is clear that TMD can or have two origins.

1. A muscular origin
2. Intra-articular or joint origin.

But irrespective of the source TMDS have all or some of the following characteristics:-

1. Pain in and around the peri-auricular region including the surrounding musculature.
2. Restriction of mouth opening
3. Sounds from the joint (clicking).

**Diagnosis of TMD**

A diagnosis for TMD is made by utilizing a screening interview[1]. The screening interview is done once dental / medical problems have been ruled out

The screening interview will have the following 5 questions.

(1) Do you have pain when you open your mouth wide, chew -once a week or more.

(2) Pain in temples, face, TMJ or jaws once a week or more

(3) Have you lately registered that the jaw is locked or that you cannot open wide.

(4) Do you have headaches more than once a week.

(5) Do you hear noises from the joint during eating, opening/ closing with or without pain.

If the patient responds positively to all or some of the questions then a diagnosis of TMD can be made.

The next step would be to correctly identify the source (muscular or joint) of the TMD.

This is the key to the management of TMD. If the distinction is not made then frustration and disappointment will follow.

Okeson[1] suggests 7 steps to differentiate between the two.

1. History 2. Restriction of movement. 3. Mandibular interference

4. Joint loading. 5. Functional manipulation 6. Occlusion 7. Diagnostic anaesthetic blockade.

To these may be added Imageology and instrument aided methods. Although it would be difficult to examine each of the seven steps in detail owing to space constraints (Please refer Okeson's classic Text Book on the subject) the last two techniques needs to be examined a little more in detail. This is because there appears to be a some confusion regarding their usefulness in the diagnosis of TMD.

| Features of Intra capsular (Joint) origin of TMD. | Features of Extra Capsular joint (muscle related) origin of TMD |
|---|---|
| Patients usually can remember an incident | No apparent cause mentioned by patient |
| There is deviation on protrusion to affected side | A 'soft' end feel |
| A hard 'end feel'* is felt | No restriction of lateral movements |
| Lateral movements are often restricted to less than 8mm | Deviation but no Deflection<br>No restriction for lateral movements |
| A report of a recent change in bite: heavy contact on posteriors. | No pain on loading |
| Deflection on opening | Functional manipulation# of individual muscles of mastication elicits pain. |
| Clicking Sounds from joint on opening/ closing or chewing with or without pain | Resting of the joint temporarily relieves the pain. E.g no pain immediately on awakening from sleep. |
| Constant pain not relieved by rest (Arthralgia) Anaesthetic blockade of Auriculo-Temporal nerve can rule in/out an intra-articular origin. | |

Table I

Conventional dental or medical imageology for example OPG, lateral Skull views, CBCT etc are not useful as these cannot reveal soft tissue pathoses. Unless gross changes have occurred to the bony structures of the TMJ these tools are unable to give any valid information. However these can be used to rule out bony or dental related pathoses.

Similarly, or even more, is the negligible role of certain instrument aided techniques like surface EMG (Electro myograph), JVA (Joint Vibratory Analysis) and Jaw Tracking in the diagnosis of TMD. These instruments do not have the necessary scientific credibility (evidence) to be part of the diasgnostic protocol.

Table I would summarise the seven steps (Okeson). This can help the clinician to differentiate between TMDs of muscular origin from those of Joint origin.

End-feel refers to the 'feel' of jaw opening when sustained force is applied by the clinician. A patient who responds with increased opening following force application is said to demonstrate a 'soft' end-feel. On the contrary if the jaw remains virtually immobile it is said to show a 'hard' end feel. The former is characteristic of a TMD of a muscular origin whereas the latter in a sign of an intra-artticular origin of the TMD

Functional manipulation is a dynamic method of testing muscle involvement. To functionally evaluate a muscle it is made to execute its function and the reaction noted for example, the Masseter / Temporalis are functionally evaluated by requesting the patient to clench while the lower head of the Lateral Pterygoid can be functionally evaluated by requesting the patient to protrude against resistance. A painful/pain free reaction will help the clinician to assess muscular involvement in the TMD.

Deviation- an alteration in opening pathway that returns to normal midline relationship at maximum opening

Deflection- an alteration in opening pathway that does not return to normal midline relationship at maximum opening. The alteration may even increase on maximum opening

By applying these criteria the clinician can arrive at a definite conclusion regarding the origin of the TMD. There upon the clinician can proceed to initiate various management strategies to help resolve the problem. These will be discussed in the third and last part of the series.

## References

1. Recommendations for the GDP- European Academy of CMD- 2007.
2. Management of Temporomandibular Disorders and Occlusion, J.P. Okeson, 6th Ed. MOSBY.

# Recent advances in diagnosis of dental caries

***A. Naveen, *R. Reshma, **R. Rahul, ***MS Deepa**

*CRRI, Azeezia College of Dental Sciences and Research, Meeyannoor, Kollam, Kerala; **Senior Lecturer, *** Prof & HOD, Dept. of Oral Medicine and Radiology, Azeezia College of Dental Sciences and Research, Meeyannoor, Kollam, Kerala.

*Send correspondence:* Dr. Naveen Aravind,, E-mail: naveenaaravind@gmail.com

## Abstract

Dental Caries is a complex, chronic disease and one of the most common illness in dentistry. Caries diagnosis is one of the most basic diagnostic skills that a dentist must learn. The recent advances of early detection of dental caries provide health and economic benefits. This article provides an overview of the state of the art methodologies for the detection and assessment of early carious lesion.

**Keywords:** Dental Caries, Caries detection methods, Fluorescence.

## Introduction

Dental Caries is an irreversible microbial disease of the calcified tissues of the teeth, characterized by demineralisation of the inorganic part and destruction of the organic portion which leads to cavitation (Latin: caries = rot or decay).[1] It is a complex and dynamic process where a multitude of factors initiate and influence the progression of the disease. Although effective methods are known for its prevention and management, it is a major health problem with manifestations persisting throughout life despite treatment.[2]

Caries diagnosis implies deciding whether a lesion is active, progressing rapidly or slowly or whether is already arrested. The proper management of caries in clinical practice requires an accurate clinical diagnosis. Accurate diagnosis can only be achieved by systematic and methodical collection of data. At the clinical dental practice level, caries diagnosis also has a significant impact since it rules treatment decisions. The diagnosis of early caries lesions has been considered as the cornerstone of cost-effective health care delivery and quality of dental care.

Early diagnosis of caries lesion is important because the carious process can be modified by preventive treatment so that the lesion does not progress. If the caries disease can be diagnosed at an initial stage, the balance can be tipped in favor of arrestment of the process by modifying diet, improving plaque control, and appropriate use of fluoride. Using non-invasive quantitative diagnostic methods, it is possible to detect lesions at an initial stage and subsequently monitor lesion changes over time during which preventive measures could be introduced.

## Caries diagnostic methods:

I. Conventional Diagnostic Methods: [3,4,5]
   a. Visual- Tactile method.
   b. Use of Caries Detection Dyes
   c. Use of Dental Floss

II. Novel Diagnostic Methods: [3,4,5,12]
   a. Radiographic Techniques
   - Conventional Radiography—IOPA, Bitewing
   - Digital Radiography
   - Digital Substraction Radiography

   b. Enhanced Visual Techniques
   - Fibre Optic Transillumination (FOTI)
   - Digital Imaging Fibre Optic Transillumination

   c. Fluorescent Techniques
   - Quantitative Light Induced Fluorescence

   d. Laser Induced Fluorescence
   - DIAGNODENT
   - Fluorescence Camera (VISTA PROOF)
   - LED Technology (MID-WEST CARIES 10)

   e. Detection systems based on Electrical Current Measurement
   - VANGUARD electrical Caries Detector
   - Cariesmeter L

   f. Ultrasound Techniques

**Visual-tactile method**

A mouth mirror and a blunt explorer (probe) should be used. The probe must be used very lightly, on the side rather than the point, to test the firmness and texture of the tooth surface.[5]

### Radiographs

Traditional IOPAR, Bitewing radiographs[11] are still mandatory as a diagnostic aid for the caries detection. Newer techniques include Digital Radiography with minimal radiation and Digital Subtraction Radiography[5]; where two radiographs taken at different times with same projection geometry are compared[10].

### Transillumination

Radiation free technique. It's a practical method of imaging teeth in presence of multiple scattering. Transillumination will assist in confirming the presence of a relatively large cavity but should be used in conjunction with radiograph.

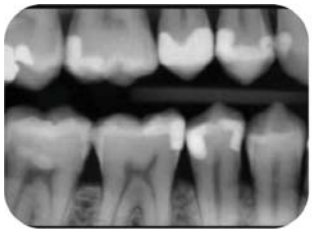
### Ultraviolet illumination

Ultraviolet light has been used to increase the optical contrast between the carious lesion and surrounding sound tissue. The natural fluorescence of tooth enamel, as seen under ultraviolet light illumination is decreased in areas of less mineral content such as in carious lesion, artificial demineralization or developmental defects. The carious lesion appears as a dark spot against a fluorescent background.

### Electronic caries detector

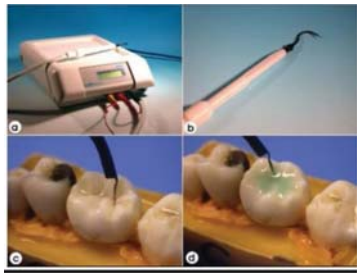
The concept of testing for caries through electrical impedance was first suggested by Pincus in 1951. Subsequently, the device called the Vanguard Caries Detector (Massachusetts manufacturing) was developed. The device consists of a small electrode for the patient to hold and a fine contact point to be placed on the tooth to explore the fissure. Any area of demineralisation will be porous; the porosities filled are with saliva and other electrolytes, so there will be a differential potential between these areas and fully mineralised tooth structure. The recording dial shows number from 0 to 10 and a picture of "FACE" that smiles

Visual Tactile Method



Radiographs

Transillumination

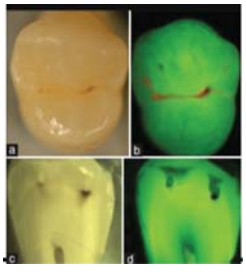


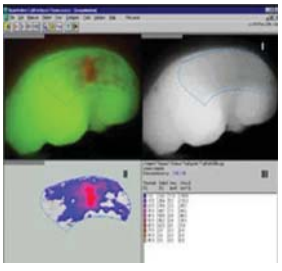
Electronic Caries Detection

← Caries Detection Dyes →

Diagnodent Pen

Diagnodent





Quantitative Light Induced Fluorescence

upto a valve of 5 and frowns when the valve is greater than 5. This level indicates that there is sufficient demineralisation to justify surgical intervention.[4,5]

### Dyes for detection of carious enamel

Various dyes have been tried to detect carious enamel, each having some advantages and disadvantages. They are Procion Dyes, Calcein dye, Brilliant Blue. Dyes have also been used to enhance the diagnostic quality of fiberoptic transillumination.[5]

### Dyes for detection of carious dentin

Dyes have been tried to differentiate between these 2 zones of dentinal caries. 0.5% basic fuchsin in propylene glycol has been proved to be successful for the purpose.

Basic Fuchsin dye was considered to be carcinogenic; therefore it has been replaced by Acid Red and Methylene Blue. Methylene Blue is also slightly toxic so Acid Red is preferred.

### Modified dye penetration method

Bakhos et al. developed an Iodine penetration method for measuring enamel porosity of incipient carious lesions. Potassium iodide is applied for a specific period of time to a well defined area of the enamel and thereafter the excess is removed. The iodine, which remains in the micropores is estimated and indicates the permeability of enamel.

### Diagnodent: (Laser Fluorescence)

The source of light is diode laser, emits laser wave length of 655 nm. It catches decay with more thoroughness, timeliness and accuracy.[4] Most minute problems are detected at an early stage, simple and comfortable to use. Laser diode provide pulsed light of a defined wavelength from the handpiece, which when directed onto the tooth it meets a change in tooth structure (decay) and stimulates fluorescent light of different wave length. This reflected fluorescent light is taken back by the handpiece receptors and later they convert into acoustic signals. Electronic system evaluates to give readings between 1-100.[13]

### Quantitative light induced fluorescence (QLF)

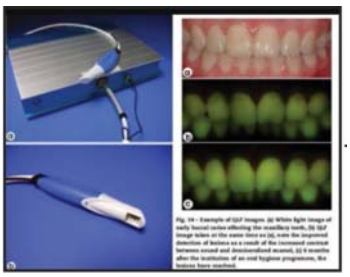
QLF is a dental diagnostic tool for invivo and invitro quantitative assessment of dental caries, plaque, calculus, staining. QLF method is based on autofluorescence of teeth. When teeth are illuminated with high intensity blue light they will start to emit light in the green part of the spectrum. The fluorescence of the dental material has a direct relation with the mineral content of the enamel.[4,14]

### Digital fiberoptic transillumination (DIFOTI)

Digital imaging fiberoptic transillumination (DIFOTI) is a relatively new methodology that was developed in an attempt to reduce the perceived short comings of FOTI by combining FOTI and a digital CCD camera.[5]

Images captured in camera are sent to a computer for analysis using dedicated algorithms. The use of CCD allows instantaneous image to be made and projected, and images taken during different examinations can be compared for clinical changes between several images of same tooth over time. DIFOTI provides clear signatures of different types of frank caries on all types of teeth, and


Digital Fiberoptic Transillumination



Ultrasound Caries Detector

DIFOTI can detect incipient or recurring caries before they are visible on radiographs.[4,9]

### Ultrasound caries detector:

The demineralisation of natural enamel is assessed by ultrasound pulse echo technique. It is observed that there is a definite correlation between the mineral content of the body of lesion and the relative echo amplitude changes. Ultrasonic probe is used which sends longitudinal waves to the surface of the tooth and also serves the function of receiving the waves. Initial white spot lesions which extend only upto enamel produce no or weak surface echoes. The sites with visible cavitation produce echoes with substantially higher amplitude.[5]

### Endoscope / videoscope:

Endoscopic technique is based on observing the fluorescence that occurs when tooth is illuminated with blue light in the wavelength range of 400-500 nm. Difference is seen in fluorescence of sound enamel and carious enamel, when this fluoresced tooth is viewed through a specific broad band gelatine filter, white spot lesions appears darker than enamel. Similarly a white light source can be connected to an endoscope by a fiberoptic cable so that the teeth can be viewed without a filter. This technique is referred to as White Light Endoscopy. Additionally, a camera can be used to store the image. The integration of the camera with the endoscope is called a Videoscope. A miniature color video camera is mounted in a custom made metal mirror holder. This is designed in such a way that image of the surface of enamel can be viewed directly over a television screen.[9]

### Future of caries detection

- Polarization-Sensitive Optical Coherent Tomography (OCT): OCT uses near Infrared light to image teeth with confocal microscopy and low coherence interferometry resulting in very high resolution images at 10-20 microns. The accuracy of OCT is so detailed that early mineral changes in teeth can be detected in vivo after exposure to low pH acidic solutions in as little as 24 hrs by using differences in reflectivity of the near infrared light.[6]
- Frequency-Domain Laser-Induced Infrared Photothermal Radiometry & Modulated Luminescence (PTR/LUM): This technology relies on the absorption of infrared laser light by the tooth with measurement of the subsequent temperature change, which is in the 10C or less range.[6] This optical to thermal energy conversion is able to transmit highly accurate information regarding tissue densities at greater depths than visual only techniques.[7,8]

## Conclusion

It is clear from the above discussion that the differences in caries presentations and behavior in different anatomical sites make it unlikely that any one diagnostic modality will have adequate sensitivity and specificity of detection of carious lesions for all sites; a combination of diagnostic tools will help us diagnose lesions earlier and detect failing restorations sooner, all to avoid more costly, destructive dental procedures and truly take dentistry into the preventive rather than reactive mode.

## Reference


1. B Sivapathasundharam and A R Raghu(2012) Dental Caries; Shafer's Textbook of Oral Pathology, 7th Edition, pg 419-420.
2. Anil Govindrao Ghom(2011);Dental Caries; Textbook of Oral Medicine, 2nd edition, pg 516-532.
3. Prachi Mital, Neha Mehta, Aditya Saini, Deepak Raisingani, Medhavi Sharma;(2014) Recent Advances In Detection and Diagnosis of Dental Caries; Journal of Evolution of Medical and Dental Sciences, vol 3 issue 1, pg 177-191.
4. Zangooei booshehry, M Fasihinia, H Khalesi, M Gholami(2010); Dental Caries Diagnostic Methods; DJH, Vol 2, no. 1, pg 1-12.
5. Nikhil Marwah, Manju Gopakumar(2014) Diagnostic aids in Dental Caries; Textbook of Pediatric Dentistry, 3rd Edition, pg 506-515.
6. Amaechi BT. Emerging technologies for diagnosis of dental caries: The road so far. Journal of Applied Physics 2009;105(10):102047-9.
7. Jeon RJ, Matvienko A, Mandelis A, Abrams SH, Amaechi BT, Kulkarni G. Detection of interproximal demineralised lesions on human teeth in vitro using frequency-domain infrared photothermal radiometry and modulated luminescence. Journal of Biomedical Optics(2007);12(3):034028—13.
8. Jeon R, Sivagurunathan K, Garcia J, Matvienko A, Mandelis A, Abrams S,editors. Dental Diagnostic Clinical Instrument. Journal of Physics: Conference Series:2010: IOP Publishing.
9. Haak R, Wichet Mj, et al.; The validity of proximal caries detection using magnifying visual aids. Caries Research 2002;36:249-55.
10. Wenzel A. Digital radiography and caries diagnosis. Dentomaxillofac Radiol 1998;27:3-11.
11. King NM, Shaw L. Value of bitewing radiographs in detection of occlusal caries. Community Dental Oral Epidemiol 1979;7:218-21.
12. Iain A Pretty. Caries detection and diagnosis: novel technologies. Journal of dentistry.2006;34:727-39.
13. Lussi A, Imwinkelried S, Pitts N, Longbotton C, Reich E. Performance and reproducibility of a laser fluorescence system for detection of occlusal caries in vitro. Caries Research. 1999;33(4):261-66.
14. Thoms M. Detection of intraoral lesions using a fluorescence camera. Proceedings of SPIE Lasers in Dentistry XII.2006;6137(5): 1-7.

# NSAID induced liver injury - Do we care enough?

***Rammohan R.S., **Radhika S Chandran**

* Reader, Department of Oral and Maxillofacial Surgery, Sri Sankara Dental College, Varkala - 695 318
* Professor, Department of Oral and Maxillofacial Surgery, Noorul Islam College of Dental Sciences, NIMS Medicity, Aralumoodu, Trivandrum - 695 123

*Send Correspondence:* Dr. Rammohan R.S.; email:

## Abstract

Liver is the principal site of drug metabolism. So any drug prescribed has its implications in liver. It is a well known fact that liver disease alters drug metabolism producing drug toxicity. Reciprocally, drugs can cause hepatotoxicity in an otherwise healthy liver. This phenomenon termed as Drug Induced Liver Injury, DILI is seen with NSAIDs also. The various NSAIDs producing DILI are discussed in detail, providing insight for timely recognition and management to the dental practitioner.

The liver is basically a protective organ. It prevents accumulation of substances in the body by converting them from lipid-soluble to water-soluble metabolites, so that it can be easily excreted by the kidneys. Its unique blood supply is tailor-made for its metabolic role. Apart from the normal hepatic artery and vein doing its respective role as in any other organ, liver has an additional portal system of veins designed to port substances to be metabolised from GIT.

Liver disease affects drug metabolism by impairing its clearance, biotransformation, and pharmacokinetics. Paradoxically a drug itself may induce liver disease, the phenomenon being termed as Drug induced liver injury, DILI. Paracetamol, the drug popularly believed to be the most innocuous is also malicious to the liver cells. The conventional practice of minimising the dosage or duration of the drug will not deter a potent hepatotoxin its manifestations. This article aims to discuss NSAID related DILI and their implications in dental practice.

DILI is a broad term applied to any injury to the liver by a drug manifesting as a spectrum from asymptomatic liver test elevations to Acute liver failure.Risk factors for drug-induced liver injury (DILI) include age ≥ 18 yr, obesity, pregnancy, concomitant alcohol consumption, and certain genetic polymorphisms.[1]

DILI can be predictable and dose-related or unpredictable and unrelated to dose.The most common example of a drug causing predictable DILI is acetaminophen. This type of drug injury has a short latency period and is the most common form of DILI observed. On the contrary, idiosyncratic DILI is unpredictable, has longer/variable latency, and is less common. Examples of idiosyncratic DILI include those related to amoxicillin/clavulanate, nonsteroidal anti-inflammatory drugs, and isoniazid. Biochemically, DILI can be hepatitic (hepatocellular injury), cholestatic, or mixed. DILI can also be categorized as immune (hypersensitivity reaction) or nonimmune.[2]

Because there is no confirmatory diagnostic test, other causes of liver disease, especially viral, biliary, alcoholic, autoimmune, and metabolic causes, need to be excluded. Drug rechallenge, although it can strengthen evidence for the diagnosis, should be avoided. Management emphasizes drug withdrawal, which, if done early, usually results in recovery. Antidotes for DILI are available for only a few hepatotoxins; such antidotes include N-acetylcysteine for acetaminophen.

### NSAIDs and hepatotoxicity

Clinically apparent liver injury from NSAIDs is rare (~1-10 cases per 100,000 prescriptions) and typically presents as acute hepatitis within 1 to 3 months of starting the medication[3]. Cases of fatal hepatitis tend to present much later – after 12 to 15 months. asymptomatic elevations in serum

aminotransferase levels occur in up to 18% of patients taking NSAIDs over a prolonged period. The rate of such aminotransferase abnormalities varies by the different NSAIDs with the lowest rate for ibuprofen (0.4 %) and Ketoralac 1% and mefenamic acid (5%). These drugs are considered liver safe with very rare instances of hepatitis reported. Sulindac, nimesulide and diclofenac are comparatively hepatotoxic with reports of symptomatic hepatitis to acute liver failure. drugs, closely related to sulphonamide like nimesulide, sulindac exhibit cross hepatic sensitivity among themselves and to sulpha drugs[4]. COX2 inhibitors and piroxicam can also cause clinically apparent liver disease. The pattern of injury is mainly hepatocellular, although cases of cholestatic (sulindac), and mixed (naproxen) injury have been reported. Women and the elderly, as well as patients with chronic hepatitis C may be more susceptible.[5]

Hepatotoxicity of aspirin and acetaminophen is due to intrinsic toxicity and usually associated with use of high doses, while for other NSAIDs it is, most likely idiosyncratic.

**Acetaminophen/Paracetamol**

Acetaminophen overdose is one of the established causes of fulminant hepatic failure. But when acetaminophen dosing is limited to less than 4 g/d, it is exceedingly rare[6]. Acute overdose (1-time ingestion of >12 g in an adult or 250 mg/kg in a child)[7] or long-term ingestion of supratherapeutic doses (>4 g/d) has resulted in acute liver failure.

The US Food and Drug Administration advises clinicians to limit combination analgesics to 325 mg of acetaminophen per unit. The action typically targets opioid combinations, since dose escalation is seen once patients develop tolerance to opiods. "There are no available data to show that taking more than 325 mg of acetaminophen per dosage unit provides additional benefit that outweighs the added risks for liver injury.[8]

The prevailing mechanism of acetaminophen-induced hepatotoxicity is due to accumulation of a hepatotoxic intermediate, N-acetyl-p-benzoquinone imine (NAPQI) more than the reserves of glutathione which normally conjugates NAPQI. In contrast, Glutathione is predictably depleted in the setting of long-term alcohol consumption or malnutrition. rendering alcoholic patients more susceptible to drug-induced liver injury[9]. less than 4 g/d of acetaminophen appears safe with mild to moderate alcohol intake, but most hepatologists advocate for lower dosing at 2 g or less per day[9].

**Aspirin**

Aspirin-related hepatotoxicity has been reported, but this is a dose-related phenomenon related to intrinsic salicylate hepatotoxicity, and generally only occurs when aspirin is used in higher doses[10]. Patients on long term, moderate to high dose aspirin therapy frequently have elevations in serum ALT levels. With high doses, ALT elevations are common and can be marked and associated with mild increases in alkaline phosphatase and bilirubin. The more dramatic examples of aspirin hepatotoxicity usually occur with doses of 1,800 to 3,200 mg daily (>100 mg/kg) and with salicylate levels of greater than 25 mg/dL[11]

**Reye Syndrome**

A special form of aspirin hepatotoxicity is Reye Syndrome, the development of lactic acidosis, microvesicular fat and hepatic dysfunction with encephalopathy and coma. Reye syndrome usually occurs in children or young adults developing a few days to a week after a prodromal febrile illness, typically influenza B or varicella. It is often rapidly fatal, but in milder cases recovery is rapid. Subsequently, case reports followed by careful epidemiological surveys linked the occurrence.[12]

Patients with severe hepatic reactions to aspirin can safely take acetaminophen or other NSAIDs, and usually tolerate lower doses of aspirin without problems. In the case of Reye syndrome, aspirin has been shown to inhibit mitochondrial function and the combination of a systemic viral illness with drug induced mitochondrial dysfunction is thought to underlie the pathogenesis of Reye syndrome[12].

**Other NSAIDs**

Nearly all of the NSAIDs have been implicated in causing liver injury. It is hepatocellular in nature[13]. Diclofenac, and particularly sulindac, are reported to be more commonly associated with hepatotoxicity[14]. In one study, sulindac use was associated with a 5–10 fold higher incidence of hepatic injury than other NSAIDS[15]. Although ibuprofen is considered to be liver-safe, sporadic cases of subacute hepatic failure have been reported. Riley[16] reported a case series of three hepatitis C patients who developed a greater than five-fold increase in liver transaminases following ingestion of therapeutic doses of ibuprofen. NSAIDs like nimesulide have been withdrawn from clinical use because of associated hepatotoxicity. The new more selective COX-2 inhibitors (e.g. celecoxib, rofecoxib, ) are also associated with hepatotoxicity, although celecoxib is said to have less potential

for hepatotoxicity.[17]

NSAID induced injury is hepatocellular, the mechanisms being impairment of ATP synthesis by mitochondria, or production of active metabolites with direct cytotoxicity. It most commonly occurs within 6–12 weeks of initiation of therapy. and the clinical pattern can be acute hepatitis with jaundice, fever or chronic active hepatitis[18] (serological ANF-positive).

Female patients aged >50 years, with autoimmune disease, and those on other potentially hepatotoxic drugs, appear to be particularly susceptible[19]. Liver function test abnormalities generally settle within 4–6 weeks of stopping the causative drug. However, some patients may develop acute liver failure. hepatotoxicity to a NSAID, often produce the same reaction if the drug is restarted or a structurally similar drug is given, e.g. sulpha drugs.[20]

## Conclusion

NSAID-induced hepatotoxicity must be considered in the differential diagnosis of all patients presenting with a spectrum of disease, ranging from isolated deranged liver function tests, to fulminant hepatic failure. Because of the availability of these drugs over the counter, many patients will not disclose their use of these agents to their doctor as they do not perceive them to be 'prescribed' medication.

Patients who develop NSAID-induced hepatotoxicity must be advised to stop taking NSAIDs. Paracetamol remains the analgesic drug of choice for these patients, even if they are jaundiced. They may also safely use aspirin in the future. This is because the toxicity of NSAIDs relates to their their diphenylamine ring molecular structure, which aspirin does not have. even though hepatotoxicity is listed as a class warning for NSAIDs, diclofenac and sulindac seem most commonly associated with the problem.

## References


1. Manns MP, Czaja AJ, Gorham JD, et al. Practice guidelines: diagnosis and management of autoimmune liver disease. Hepatology 2010; 51: 1–29. [Ref list]
2. Leise MD1, Poterucha JJ2,Talwalkar JA. Drug induced liver injury. Mayo Clin Proc. 2014 Jan;89(1):95-106.
3. National library of medicine. www.livertox.nih.gov
4. Tarantino G, Di Minno MND, Capone D. Drug-induced liver injury: Is it somehow foreseeable? World Journal of Gastroenterology/: WJG. 2009;15(23):2817-2833.
5. Riley TR 3rd1, Smith JP. Ibuprofen-induced hepatotoxicity in patients with chronic hepatitis C: a case series.Am J Gastroenterol. 1998 Sep;93(9):1563-5
6. Temple AR, Lynch JM, Vena J, Auiler JF, Gelotte CK. Aminotransferase activities in healthy subjects receiving three-day dosing of 4, 6, or 8 grams per day of acetaminophen. Clin Toxicol (Phila)2007;45(1):36-4
7. Makin AJ, Wendon J, Williams R. A 7-year experience of severe acetaminophen-induced hepatotoxicity (1987-1993). Gastroenterology 1995;109(6):1907-1916
8. Acetaminophen Overdose and Liver Injury — Background and Options for Reducing Injury. https://www.fda.gov/downloads/AdvisoryCommittees/.../UCM164897.pdf
9. Benson GD, Koff RS, Tolman KG. The therapeutic use of acetaminophen in patients with liver disease. Am J Ther.2005;12(2):133-141
10. Fry SW, Seeff LB. Hepatotoxicity of analgesics and anti-inflammatory agents. Gastroenterol Clin North Am. 1995;24:875–905.
11. Wolfe JD, Metzger AL, Goldstein RC.Acute enzyme elevations due to high dose aspirin therapy.Aspirin hepatitis. Ann Intern Med 1974; 80: 74-6.
12. Norman MG, Lowden JA, Hill DE, Bannayne RM. Reye syndrome in a child with juvenile rheumatoid arthritis. Encephalopathy and fatty degeneration of the viscera in childhood: II. Report of a case with isolation of influenza B virus. Canad Med Assoc J 1968; 99: 522-6.
13. Aithal GP, Day CP. Nonsteroidal anti-inflammatory drug-induced hepatotoxicity. Clin Liver Dis. 2007;11:563–575, vi-vii
14. Hussaini SH, Farrington EA. Idiosyncratic drug-induced liver injury: an overview. Expert Opin Drug Saf. 2007;6:673–684
15. Guldenschuh I, Hurlimann R, Muller A, Ammann R, Mullhaupt B, Dobbie Z, Zala GF, Flury R, Seelentag W, Roth J, et al. Relationship between APC genotype, polyp distribution, and oral sulindac treatment in the colon and rectum of patients with familial adenomatous polyposis. Dis Colon Rectum. 2001;44:1090–1097
16. Riley TR 3rd, Smith JP. Ibuprofen-induced hepatotoxicity in patients with chronic hepatitis C: a case series. Am J Gastroenterol 1998; 93: 1563-5.
17. Benichou C. Criteria of drug-induced liver disorders. Report of an International Consensus Meeting. J Hepatol. 1990; 11: 272-276.
18. Masubuchi Y, Yamada S, Horie T. Possible mechanisms of hepatocyte injury induced by diphenylamine and its structurally related NSAIDS. J Pharmacol Exp Ther 2000; 292: 982–7.
19. O'Connor N1, Dargan PI, Jones AL. Hepatocellular damage from non-steroidal anti-inflammatory drugs. QJM 2003 Nov;96(11):787-91.
20. Carrillo-Jimenez R, Nurnberger M. Celecoxib-induced acute pancreatitis and hepatitis: a case report. Arch Intern Med 2000; 160: 553-4.

CASE REPORT

# Comparitive evaluation of gingival healing following gingevectomy split mouth technique - using laser and scalpel

*** Tintu Madona Joy, **Mintu M Kumar, **Seema G.**

* Post Graduate Student, **Senior Lecturer, Department of Periodontics, Sri Sankara Dental College, Akathumuri, Varkala, Trivandrum.

*Send correspondence:* Dr. Tintu Madona Joy, E-mail: tintujoy1983@gmail.com

## Abstract

Objective-This case report investigates gingival healing after gingevectomy and adjunctive use of laser therapy in an inflammatory gingival enlarment during fixed orthodontic therapy.

The wound healing takes place by secondary intention and is associated with significant patient morbidity during the healing period. Newer techniques like the use of laser can be employed to enhance patient comfort during procedure and better healing of the gingivectomy sites. Laser use is beneficial as it requires minimal anaesthesia, lesser surgical time,less post operative pain and good post-operative healing.

**Key words:** Gingivectomy, laser, wound healing

## Introduction

A perfect smile can be crafted only on a healthy gingiva. The goal of periodontal therapy is to reestablish anatomical and physiologic conditions conducive to long term health and function of periodontium. Esthetic improvement is one of the most common reasons a patient wishes to undergo orthodontic treatment.

Gingival position and shape plays a vital role in esthetics of an orthodontic case. Unfortunately, after orthodontic treatment the gingival position and shape commonly possesses non-ideal properties, negatively impacting the end result. For these patients, a soft tissue gingivectomy procedure is needed to position the gingival unit in the correct position.

Traditionally, gingivectomies were performed using a scalpel with satisfactory results however, with the advent of laser technology, diode lasers have been increasingly utilized for intraoral soft tissue applications such as gingivectomies. The purpose of the present study was to compare gingivectomy performed with a scalpel and those performed with an 980 nanometer diode laser in regards to postoperative pain, gingival healing, gingival relapse, and operative time.

Gingivectomy is a procedure employed to eliminate the pseudopockets seen in the enlarged gingiva. The procedure is employed to remove the diseased tissue, to improve the esthetics, for prosthetic and orthodontic purposes and to reduce the probing pocket depth of periodontal pockets.

The wound healing is a slow process following gingivectomy and takes place by secondary intention; it takes a few weeks to establish the normal contour of the gingiva. Several methods such as topical application of medicaments, antibiotics, or amino acids have been tried to improve the healing by secondary intention. Furthermore, techniques that cause lesser tissue damage would allow the wound heal quickly and uneventfully.[1]

Laser therapy is a fast growing, simple and atraumatic technique, which has been used in dentistry since the 1990s. Improved infection control, reduced postoperative pain and sensitivity, reduced patient anxiety and minimizing the need for anesthesia are the other advantages of laser.

Gingivectomy using lasers may prove to be beneficial to the patient as decreases patient morbidity and results in faster healing as compared to the conventional technique. With this background in view, the following study was conducted to evaluate the post gingivectomy healing in scalpel and laser treated surgical wounds.[3]

A study by Konikoff et al found that greater than 60% of patients finishing orthodontic

treatment had some type of gingival asymmetry. This gingival encroachment of the clinical crowns was most often due to either inflammatory changes of the soft tissue or an altered or delayed passive eruption of the teeth. Thus, Konikoff et al. concluded that in order to achieve an ideal smile in a majority of orthodontic patients, gingevectomy should be considered to any asymmetries.[3]

### Disadvantages of Laser Therapy[3]

1. The mild postoperative discomfort usually delayed up to one or two weeks.
2. Epithelial regeneration is delayed, and the wounds take a longer time to reepithelialize than following conventional surgery, is thought to be the result of the lack of wound contraction and thus failure of the area to reduce in size.
3. High cost of equipment.
4. The procedure usually will take longer time especially, where abundant of tissue removal is required.
5. Is the possible retinal injury to the eyes of the patient or practitioner with use of the diode laser.
6. Some clinicians have reported less tactile sense, slower cutting, poorer wound healing, and greater tissue dessication when using the diode laser as compared with use of a scalpel.
7. The plume that is created when removing the soft tissue with a diode laser can contain certain pathogens and have a burning flesh odor and therefore, a high-filtration face mask is recommended

### Case report

A 14-year-old male patient, undergoing fixed orthodontic treatment reported to the Department of Periodontics, Sri Sankara Dental College, Akathumuri, Varkala, Trivandrum with localized gingival enlargement in the right and left upper and lower back region. Gingivectomy had been planned for the patient In order to compare the healing following treatment, it was decided to treat tooth no 13,14,15,43,44,45 (Fig 1) using a diode laser and tooth numbers 23,24,25,33,34,35 (Fig 2) using the conventional scalpel technique and teeth

Presurgical preparation consisted of scaling, oral hygiene instructions, intra oral periapical radiographs and blood investigations. A written informed consent was obtained from the patient. Following local infiltration of the tissue a conventional gingivectomy procedure was performed on teeth 23,24,25,33,34,35 using a scalpel excising the soft tissue wall to the base of the pocket (Fig 3). Teeth 13,14,15,43,44,45 were lased with a diode laser 980 nm (ZOLAR) excision mode 3 W, pulsed contact mode, pulse interval and length 0.1 ms, scanning an area of about 1 cm2 while holding the delivery tip perpendicular to the tissue surface (Fig 4).

## SURGICAL TECHNIQUE

*Fig 1* Preoperative view

*Fig 2* Preoperative view

*Fig 3* Scalpel gingevectomy

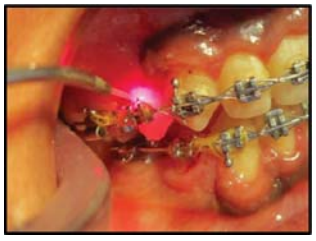


*Fig 4* Laser gingevectomy

*Fig 5* Post operative view laser

*Fig 6* Post operative view Scalpel

After the procedure, all surgical sites were covered with a periodontal dressing (Coe-Pak). The periodontal dressings were removed after seven days. Postoperative instructions were given and patient was asked to monitor the pain response. Photographs were taken of the treatment sites at pre-surgery, immediately post-surgery The patient was asked to assess the overall pain experience and on either of the operated sites based on the McGills pain questionnaire reported more discomfort on the scalpel treated site as compared to the lased site.

**Clinical evaluation**

The surgical sites were evaluated for the following:

- ✧ Tissue color (red, pink or bluish);
- ✧ Tissue color (red, pink or bluish);
- ✧ Tissue contour (normal, hyperplastic, or atrophic)
- ✧ Clinical status of the healing wound for the specific time interval (normal, better than normal, worse than normal).
- ✧ For the purpose of analysis, a three point scale was used to score healing of the surgical wound

Score +1: Indicating superior healing of laser-treated wounds compared to control sites.

Score 0: Indicating that laser-treated test sites and control sites exhibited the same degree of healing response.

Score –1: Indicating that control exhibited superior healing when compared to lasertreated test sites.

The test site were reevaluated after 7 days, 2 weeeks and after one month respectively concluding that the laser applied sites healed better than the scalpel treated sites. Laser showed better results after healing completed and was a relatively painless and easy procedure as assessed and confirmed by McGill's pain questionnaire.

**Results**

Healing was uneventful in both techniques, gingivectomies performed with the laser had less postoperative inflammation, lesser pain and better healing earlier, compared to scalpel technique

**Discussion**

Healing of periodontal tissue after surgical treatment has long been a subject of study. In this case report post-gingivectomy wounds were assessed over a number of days to clarify whether laser treatment could or could not improve the healing process and post-surgical patient comfort

Data suggest that the laser-treated site had a faster recovery in these periods, with a reduction of pocket depth compared to scalpel technique.This finding indicates that, in this period, the irradiated wounds underwent a better healing process than the wounds from the control group, probably because of higher collagen production leading to a better remodeling of the connective tissue and a reduction of the probing depth.[1]

The reduction of the probing depth in the early stages of healing is a very positive finding, because it makes it easier for the patient to keep the area clean, allowing better oral hygiene.[2]

The advantage of using the scalpel technique over other modalities, such as soft tissue lasers or electrosurgery, is the lack of tissue damage beyond the edge of the incision. These other modalities generate heat during use and can cause collateral tissue damage adjacent to the incision.The disadvantages of using the scalpel include the lack of hemorrhage control and resultant difficulty in visualizing the surgical field as well as difficulty in thinning and recontouring gingival tissues.[2]

Laser treatment is a unique noninvasive approach that offers infection control, precision surgical technique and encouraging postoperative benefits.Treatment using soft tissue lasers has dramatically improved surgical technique and wound healingas has been demonstrated in several studies.

Therefore, in this case postgingivectomy wounds were assessed over a number of days to clarify whether laser treatment could improve the healing process and postsurgical results,during the surgical procedure profuse bleeding was observed in the scalpel treated site, whereas the laser site was relatively blood less lack of swelling, pain or scar tissue formation and eventually good wound healing.

Laser treatment causes destruction of epithelial and stromal cells but leaves much of the connective tissue matrix intact and the basement membrane resists laser irradiation. The laser induced wounds heal through reparative synthesis of matrix proteins.[10] The relative resistance of matrix proteins against laser irradiation and the slow removal and replacement of the residual matrix accounts for the lack of scarring and contraction observed in laser-treated areas.

Laser-treated sites also have improved adenosine triphosphate synthesis, fibroblast proliferation; collagen synthesis, phagocytosis of macrophages, and acceleration of the inflammatory phase of wound healing. All these mechanisms can result in cellular proliferation and acceleration of the wound healing process.[11]

There are certain limitations in the use of lasers for gingivectomy like excessive heating of the tissue due to charring, cemental burns result when the laser tip comes in contact with the tooth; also laser

procurement is costlier when compared to other modalities.

## Conclusion

The diode laser provides an adequate alternative to the traditional scalpel for soft tissue gingivectomy procedures. The diode laser performs the procedure more efficiently with greater hemostasis than does the scalpel. Also, gingival healing and inflammation,are significantly improved post surgically with the diode laser as compared to the scalpel, although relapse of the gingival tissue in a coronal direction is greater with the laser than with the scalpel. Although the diode laser does provide some added benefit over the scalpel technique, there are some drawbacks, so the clinician must decide if the added expense of the laser unit is worth the benefit that is achieved.

## Reference


1. Clinical Study of the Gingiva Healing after Gingivectomy and Low-Level Laser Therapy, Photomedicine and Laser Surgery, Amorim et al,Volume 24, Number 5, 2006.
2. Histological and clinical evaluation of gingival healing following gingivectomy using different treatment modalities,Meenawat, et al,JJournal of the International Clinical Dental Research Organization, January-December 2013 Vol 5 Issue 1.
3. A Comparison of two soft tissue gingevectomy techniques by James R Durham, University of Alabama at Birmingham,Alabama 2009
4. Van der Geld, P., Oosterveld, P., Van Heck, G., & Kuijpers-Jagtman, A. M. (2007). Smile Attractiveness. Angle Orthodontist, 77 (5), 759-765.
5. Waldrop, T. C. (2008). Gummy Smiles: The Challenge of Gingival Excess: Prevalence and Guidelines for Clinical Management. Seminars in Orthodontics, 260-271.
6. Garber, D. A., & Salama, M. A. (1996). The aesthetic smile: diagnosis and treatment. Periodontology 2000, 18-28.
7. Sarver, D. M. (2005). Soft-Tissue-Based Diagnosis Treatment Planning. Clinical Impressions, 21-26.
8. Konikoff, B. M., Johnson, D. C., Schenkein, H. A., Kwatra, N., & Waldrop, T. C. (2007).
9. Clinical Crown Length of the Maxillary Anterior Teeth Preorthodontics and Postorthodontics. Journal of Periodontology, 645-653.
10. Ma riotti A. Dental plaque-induced gingival diseases. AnnPeriodontol 1999;4:7-19.
11. Buchelt M, Kutschera HP, Katt erschafk a T, Kiss H, Lang S, Beer R,et al. Erb:YAG and Hol:YAG laser osteotomy: The eff ect of laser ablation on bone healing. Lasers Surg Med 1994;15:373-81.
12. Pourzarandian A, Watanabe H, Ruwanpura SM, Aoki A, Ishikawa I. Eff ect of low-level Er: YAG laser irradiation on cultured human gingival fi broblasts. J Periodontol 2005;76:187-93.


# Guidelines to the Authors

- Article will be evaluated for publication on the understanding that the work submitted has not been published elsewhere, that it has not and will not be submitted to another journal until the editor has made a decision on its acceptability for this journal, and that, if accepted, its contents will not be published elsewhere without the editor's permission. Accepted papers become the copyright of the Impressions-Journal of IDA Attingal branch and permission must be sought from the publishers before they can be reprinted elsewhere.
- Each page must be numbered in consecutive order from the title page, through the abstract, text, references.
- Manuscript must contain a structured abstract of a maximum of 200 words, with the headings, purpose of the study, materials and methods, results, and conclusions.
- Use standard headings: introduction, materials and methods, results, discussion, conclusions.
- References shall be given according to the Vancouver style: the author's surname and initials (if there are more than five authors, additional authors are designated as et al.); full title; journal title (in standard abbreviated form); year; volume number; and page numbers from start to finish.
- A version of the manuscript must be submitted on CD as well.
- Correspondence may be sent to the following address:

The Editor-In-Chief, Impressions-Journal of IDA Attingal Branch,
Prathyusha Dental Care IIIrd Floor YCDC, Opp Vydyuthi Bhavanam, Pattom,
Thiruvananthapuram - 695004
Mobile: 9447060374, e-mail: journalidaatl@gmail.com

CASE REPORT

# Comparative Results of Frenectomy by Conventional technique and Laterally displaced flap

*** Navya L V., **Sarath C., ***Seema G**

* Post Graduate Student, **Senior Lecturer, ***Professor, Department of Periodontics, Sri Sankara Dental College, Akathumuri, Varkala, Trivandrum.

*Send correspondence:* Dr. Navya L V., E-mail: navyalv123@gmail.com

## Abstract

An aberrant frenum gives rise to aesthetic and functional problems and needs to be eliminated. Various procedures were introduced to eliminate the aberrant frenum which led to delayed healing, loss of interdental papilla and unesthetic scar. This led to more conservative approaches with their technical and esthetic limitations. An alternative approach for primary closure in midline and to avoid unesthetic scar by creating a zone of attached gingiva is frenectomy with laterally displaced flap. The interdental papilla is left surgically undisturbed and healing takes place by primary intention. A case report of frenectomy with laterally displaced flap is presented.

**Key words:** aberrant frenum, frenectomy, laterally displaced flap

## Introduction

A frenum is an anatomic structure formed by a membranous fold of mucous membrane and connective tissue, sometimes muscle fibres. The superior labial frenum is triangular in shape and attaches the lip to the alveolar mucosa and/or gingiva. It extends over the alveolar process in infants and forms a raphe that reaches the palatal papilla. This attachment generally changes as the alveolar process grows to assume the adult configuration.[1] Taylor has observed that a midline diastema is normal in about 98% children between 6 and 7 years of age but the incidence decreases to only 7 % in persons 12-18 yrs old.[2] But in some instances the infantile arrangement is retained. Depending upon the extension of attachment of fibres, frena have been classified as [3]

1. Mucosal – fibres attached up to mucogingival junction.
2. Gingival – fibres inserted within attached gingiva.
3. Papillary – fibres extending into interdental papilla
4. Papilla penetrating – the frenal fibres cross the alveolar process and extend up to palatine papilla.

Clinically, papillary and papilla penetrating frena are considered as pathological and have been found to be associated with loss of papilla, recession, diastema, difficulty in brushing, alignment of teeth.[4,5] Miller has recommended that the frenum should be characterised as pathogenic when it is unusually wide or there is no apparent zone of attached gingiva along the midline or the interdental papilla shifts when the frenum is extended.[6] In such cases, it is necessary to perform a frenectomy for esthetic, psychological and functional reasons. Aberrant frena are detected visually, by applying tension to see the movement of papillary tip or blanch produced due to ischemia of the region.[7] There are numerous surgical techniques for the removal of labial frenum. In the "classical frenectomy" by Archer[8] and Kruger[9] the frenum, interdental tissue and palatine papilla are completely excised leading to exposure of underlying alveolar bone and thus leading to scarring. This technique resulted into an unesthetic scar, but this approach was advocated to assure removal of muscle fibres, supposedly connecting the orbicularis oris with the palatine papilla. It was thought that if this was not done, the diastema would reopen.

Henry et al. in his study of histological constituents of frenum found dense collagenous tissue, loose connective tissue and elastic fibres but no muscle fibres.[1] So Edward[10], evaluating 308 patients who demonstrated either a diastema or an abnormal frenum or a combination of both, advocated a "conservative surgical procedure". His method consisted of three procedures:

1. Apically repositioning of the frenum with denudation of alveolar bone.

2. Destruction of the trans-septal fibres between the approximating central incisors.

3. Gingivoplasty of any excess labial and/or palatal tissue in the interdental area.

One of the characteristics of Edward's technique was the esthetic maintenance of the interdental papilla. But the healed scar in the midline appeared unesthetic to the subjects. Coleton[11] and Lawrence[12] have used free gingival graft combined with frenectomy. This procedure avoids the scar and increases the attached gingival in midline, but a mismatched gingival colour in midline and need of a second surgical site to achieve donor tissue complicated the technique. Laser has been used by various clinicians which has its relative advantages and disadvantages.[13,14] Miller in 1985 presented a surgical technique combining the frenectomy with a laterally positioned pedicle graft.[7] Esthetically acceptable attached gingiva across the midline was attained by laterally positioned gingiva and healing by primary intention. No attempt was made to dissect the transseptal fibres and hence, interdental papilla remained undisturbed. Esthetically and functionally better results were obtained.[7] This article is a comparison of clinical cases of an aberrant frenum which were approached by techniques like conventional (classical) technique and Miller's technique using unilaterally displaced pedicle flap.

## Surgical technique

### Conventional (classical) technique

A 20 year old female patient was reffered from department of orthodontics with high frenal attachment. On clinical examination it was found that patient had a type III frenal attachment. The situation was explained to the patient and was advised to undergo a frenectomy procedure by conventional technique. The area was anaesthetized, using 2% lignocaine with 1:80000 adrenalines. The frenum was engaged with a haemostat which was inserted into the depth of the vestibule and incisions were placed on the

*Fig 1* Preoperative View

*Fig 2* Frenum was engaged with hemostat

*Fig 3* Frenum Excised

*Fig 4* Suturing done

*Fig 5* Coe Pack given

*Fig 6* 2 weeks postoperative view

*Fig 7* 6 months postoperative view

upper and the under surface of the haemostat until the haemostat was free. The triangular resected portion of the frenum with the haemostat was removed. A blunt dissection was done to relieve the fibrous attachment. The edges of the diamond shaped wound were sutured using 4-0 black silk with interrupted sutures. The area was covered with a periodontal pack. The pack and the sutures were removed 1 week post-operatively.

### Frenectomy using unilateral single pedicle flap (miller's technique)

A 43 year old male came to the department of periodontology with chief complaint of spacing between the front teeth. On clinical examination it was found that patient had a type III frenal attachment. The situation was explained to the patient and was advised to undergo a frenectomy procedure. After local anaesthesia, incision was taken to separate the frenum from the base of interdental papilla. This incision was extended apically up to the vestibular depth to completely separate the frenum from alveolar mucosa. Any remnant of frenal tissue in the mid line and on the under surface of lip was excised. A vertical parallel incision was taken on the mesial side of lateral incisor, 2-3mm apical to marginal gingiva, up to vestibular depth.

The gingiva and alveolar mucosa in between these two incisions were undermined by partial dissection to raise the flap. A horizontal incision was then given 1-2 mm apical to gingival sulcus in the attached gingiva, connecting the coronal ends of the two vertical incisions. Flap was raised, mobilised mesially and sutured to obtain primary closure across the midline. The surgical area was dressed with COE PAK (GC America Inc., USA). Dressing and the sutures were removed 1 week later. A healing zone of attached gingiva was clearly visible with no loss of interdental papilla.

### Results

The healing of the surgical procedure was uneventful for both techniques. Conventional frenectomy technique leads to the scaring in the midline. The unilateral pedicle flap shows complete healing with zone of attached gingiva, no scar and colour of gingival tissue was comparable to the adjacent. Healing was obtained by primary intention. No loss of interdental papilla was observed. No complication was noted during healing period.

### Discussion

In the era of periodontal plastic surgery, more conservative and precise techniques are being adopted to create more functional and esthetic results. The technique for management of aberrant frenum has undergone changes from Archer's8 and Kruger's[9] "classical techniques" of total frenectomy to Edward's[10] more conservative approach. Recent techniques added frenal relocation by Z-plasty[15], frenectomy with soft-tissue graft[11,12] and Laser[13,14]

Preoperative Views (Fig 8,9)

Frenum Excised (Fig 10)

Incision for lateral flap (fig 11)

Flap displaced and sutured (Fig 12)

Coe Pack given (Fig 13)

2 weeks postoperative view (Fig 14)

6 Months postoperative view (Fig 15)

applications to avoid typical diamond shaped scar and facilitate healing. A frenum is evaluated in relation to vestibular depth, zone of attached gingiva, interdental papilla and diastema. A zone of attached gingiva is considered to prevent recession and it also gives an aesthetically pleasant appearance. Miller's technique combined with a laterally positioned pedicle graft[7] was attempted in this case. This technique offers two distinct advantages. First, on healing there is a continuous band of gingiva across the midline rather than unesthetic scar. The second advantage is that transseptal fibres are not disrupted surgically, to avoid any trauma to interdental papilla. This prevents loss of interdental papilla. Miller, in his study observed that interdental papillae were maintained in all 27 cases surgically treated by this method. But the classical technique leaves a longitudinal surgical incision and scarring, which may lead to periodontal problems and an anaesthetic appearance, thereby necessitating other modifications.

## Conclusions

In conclusion, the conventional (classical) technique fails to provide satisfactory aesthetic results in the case of a broad, thick hypertrophied frenum. This may be due to an inability to achieve primary closure at the centre, consequently leading to secondary intention healing at the wide exposed wound. It may become a matter of concern in the case of a high smile line exposing anterior gingiva. The unilateral pedicle flap technique shows complete healing with zone of attached gingiva, no scar formation and colour of gingival tissue was comparable to the adjacent tissue. This technique may be suitable in situations where anterior aesthetics is of primary importance. The technique is reliable and easy to perform and provides excellent aesthetic results.

## Acknowledgement

I am grateful to Professor and HOD Dr. Seema G in guiding and discussing the case. I am also grateful to Dr. Sarath C (Senior Lecturer, Department of Periodontics) for his support during the clinical procedure. Also thankful to the patient and his family members for the utmost cooperation.

## References

1. Henry SW, Levin MP, Tsaknis PJ. Histological features of superior labial frenum. J Periodontol 1976;47:25-28.
2. Taylor JE. Clinical observation relating to the normal and abnormal frenum labii superioris. Am J Orthod Oral Surg 1939;25:646.
3. Placek M, Miroslavs, Mrklas L. Significance of the labial frenal attachment in periodontal disease in man. Part 1; Classification and epidemiology of the labial frenum attachment. J Periodontol 1974;45:891-94.
4. Dewel B F,The labial frenum, midline diastema and palatine papilla: a clinical analysis. Dent Clin North Am 1966;175-84.
5. Maria E Diaz-Piaz, Manual O Lagravere, Rita Villera. Midline diastema and frenum morphology in the primary dentition. J Dent 2006;26:11-14.
6. Miller P D Jr. The frenectomy combined with a laterally positioned pedicle graft; functional and aesthetic consideration. J Periodontol 1985;56:102-106.
7. Gottsegen R. Frenum position and vestibule depth in relation to gingival health. Oral Surg 1954;7:1069-72.
8. Archer W H (ed). Oral surgery- a step by step atlas of operative techniques, 3rd edition. Philedelphia, W B Saunders Co.,1961;192.
9. Kruger GO (ed). Oral surgery, 2nd edition. St. Louis, The C.V. Mosby Co.,1964;146.
10. Edwards J G. The diastema, the frenum, the frenectomy: a clinical study. Am J Ortho1977;71:489.
11. Colten S M. Mucogingival surgical procedures employed in re-establishing the integrity of the gingival unit(III):The frenectomy and the free mucosal graft. Quintessence Int 1977;8:53-61.
12. Lawrence G B, Fowler E B, Moore E A, Murray D J. The free gingival graft combined with the frenectomy; A Clinical review. Gen Dent 1999;47: 514-18.
13. Coluzzi D J. Fundamentals of dental laser, science and instruments. Dent Clin North Am 2004;48:751- 70.
14. Gontizo F, Navarro R S, Haypek P, Ciamponi A L, Haddad A S. The application of diode and Er;YAG lasers in labial frenectomy in infant patients. J Dent Child 2005;72: 10-15.
15. Tait CH. Median frenum of upper lip and its influence on spacing of upper central incisor teeth. N Z Dent J 1929;25: 116.

# IDA Attingal Branch

## Reports & Activities

**JULY**

CDE program "LAYERS OF BEAUTIFICATION" by Dr Ratan Salecha was conducted on 9th July 2017 at Travancore hall, Technopark. It was a well attended program by our members

The brach collected rupees 2,00,000 from its members who willingly contributed and handed over to Dr Gijo Eldred's family as an aid towards the treatment expenses of Dr Gijo Eldred

**AUGUST**

First General Body meeting was conducted on August 6th at Lions Club Hall. 39 members attended. On the same day, after the GB a CDE programme was conducted. Topic "IDEAL RADIOGRAPHS In Dental Office". The faculty for the programme was our branch member Dr Rahul R.

Womens wing of IDA Attingal branch conducted a free dental camp for cancer patients in connection with Oral Hygiene Day at Palode. Cancer awareness class was conducted and the ill effects of tobacco usage was described to them. oral hygiene instructions were given, Basic dental treatment was provided medicines were distributed free of cost.

**SEPTEMBER**

Onam celebration of our branch was arranged at "Snehatheeram", Mitirmala near Karette on 17th September. Snehatheeram is an orphanage for mentally challenged women. Cultural events were conducted in which Inmates of "Snehatheeram" actively participated along with our members. "Ona sadhya. "the branch also provided dresses, groceries, books and eatables to them.

On 24th September another CDE programme along with handson was conducted. Topic "Workshop On Advanced Endodontics"

by Dr Digesh Burfiwala at Hotel Dobloon, Kallambalam. No additional charge was collected from participients for handson.

One executive committee meeting was conducted in August.

*Distribution of Onam gift to the inmates of Snehatheeram by WDC Chair Person Dr Deepa. S*


*CDE IDEAL RADIOGRAPHS In Dental Office". "by Dr Rahul R.Dhoble*

*Inauguration of Onam celebration at Snehatheeram by the state vice president Dr Arun Roy*