VIBROTACTILE DEVICES FOR PAINLESS LOCAL ANESTHESIA: A REVIEW

Dr. UMME AZHER

Dr. Umme Azher
Reader, Department of
Pedodontics and Preventive Dentistry,
Sri Rajiv Gandhi College of
Dental Sciences and Hospital
Cholanagar, Hebbal
Bengaluru, Karnataka

ABSTRACT
In pediatric dentistry, the control of dental operative pain requires the routine use of local anesthesia. But the administration of local anesthesia itself produces pain and anxiety that may lead to subsequent unfavorable behavior in children. Some of the newer local anesthetic delivery systems which have been developed with the aim of reducing fear of the needle take advantage of the gate control theory of pain management, which suggests that pain can be reduced by simultaneous activation of nerve fibers through the use of vibration.

KEY WORDS
local anesthesia, vibration, vibrotactile devices.

INTRODUCTION
Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage[1]. Pain management is one of the most important aspects of patient care in pediatric dentistry. But unfortunately the methods used to control dental operative pain may themselves be painful.

Behavioral and physiologic observations suggest that the injection of local anesthetic is among the most feared and anxiety producing procedures during dental intervention[2,3]. Children who perceive significant pain during dental procedures may exhibit greater behavior problems at subsequent visits, resulting in more need for restraint and longer procedures[4]. Of even greater concern, children who experience discomfort may avoid necessary dental care and may be more likely to avoid future care as adults[5].

The pain induced by infiltration of local anesthetic agents can be reduced in a number of complementary methods such as application of topical analgesics[6], distraction technique[7], counter irritation[8,9], varying the rates of infiltration[10], buffering the local anesthesic [11-14], reduced speed of injection[15,16] and use of vibration [17-22].

Vibratory stimulation is a potential method for the treatment of pain. It is one of the several non-pharmacological techniques used to reduce pain[23].

GATE CONTROL THEORY
The vibrotactile devices, which are aimed at easing the fear of the

For correspondence:
Dr. Umme Azher
E-mail: drummeazher@yahoo.com

needle take advantage of the "Gate control theory" of pain management[24], which suggests that pain can be reduced by simultaneous activation of nerve fibers through the use of vibration.

In 1965, Canadian Psychologist Ronald Melzack & British Physiologist Patrick Wall, described the Gate Control theory of nerve conduction. They suggested a gating mechanism within the dorsal horn of the spinal cord that closed in response to normal stimulation of the fast conducting "touch" nerve fibres; but opened when the slow conducting "pain" fibers transmitted a high volume and intensity of sensory signals. They further stated that the gate could be closed again if these signals were countered by renewed stimulation of large fibers. So based on the stimuli this can either permit a pain sensation to travel up the spinothalamic tract to the brain or block those signals from being perceived as pain.

The sensation of pain is conducted slowly along thin, unmyelinated C nerve fibers traveling at about 2 meters per second, whereas an impulse such as vibration is conducted rapidly along thick myelinated A beta fibers at a rate of 75 meters per second. When these two sensations occur at the same time, the sensation of vibration reaches the sensory area of the brain first, causing a release of inhibitory inter-neurons which prevents the activation of projection neurons in the dorsal horn of the spinal cord, resulting in a closure of the gate and the sensation of pain is blocked (Fig 1).

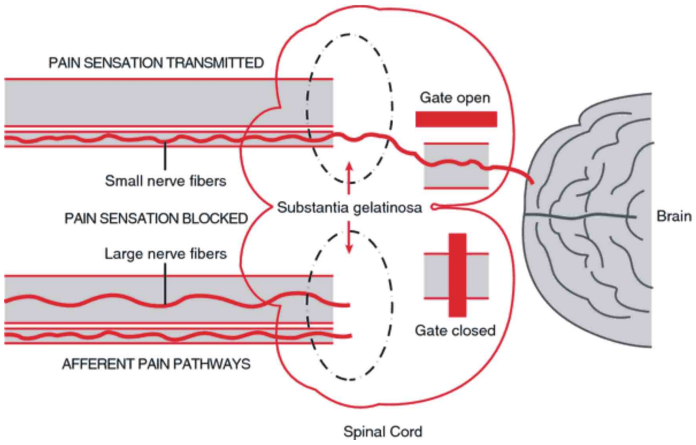


Fig 1: The Gate-Control theory of pain. From Linton et al., 2000.

Vibrotactile Devices

VibraJect

VibraJect (Miltex Inc, York, PA)(Fig 2) is a small device that uses vibrations to block pain sensation during local anesthetic injections. It is a battery operated device which has an attachment that snaps onto the standard dental syringe. It produces vibrations at high frequency onto the needle which inhibits pain sensation at the time of injecting anesthetic. Nanitos and colleagues[19] found this device to be effective in decreasing injection pain and used the gate control theory to explain their findings. Effectiveness of VibraJect, however, has been mixed.

Fig 2: Vibraject

DentalVibe

DentalVibe (BING Innovations LLC, USA) (Fig 3) is a handheld cordless injection system, which consists of a U-shaped vibrating tip attached to a microprocessor-controlled Vibra-Pulse motor. The device delivers a pulsed, percussive vibration with enhanced amplitude, which gently taps the mucosa in a synchronized but changing pattern which keeps the α-β nerve fibers activated thus diverting the pain sensation[25]. The activated U-shaped vibrating tip is first applied to the injection site, and the dental needle may be inserted anywhere in the vibration zone. It also illuminates the injection area and has an attachment to retract the lip or cheek.

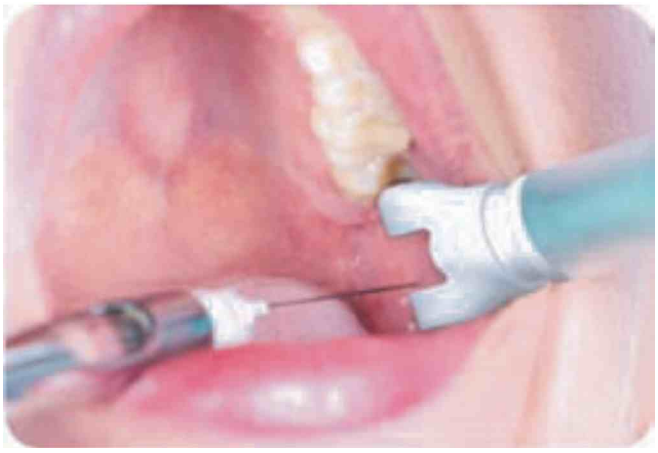


Fig 3: Dentalvibe

Accupal

The Accupal (Hot Springs, AR, USA) (Fig 4) is a cordless battery operated device used for inferior alveolar blocks and palatal injections. It uses vibration as well as pressure to precondition the alveolar or palatal mucosa. Accupal provides pressure and vibrates the injection site, 360° proximal to the needle penetration; this shuts the pain gate mechanism which blocks the pain sensation[26]. The device has a hole headed slot which is attached to motor. The head is placed at the injection site with moderate pressure. The head begins to vibrate & illuminates the area; needle is then placed through a hole in the head to express anesthetic solution without pain to the patient.

Fig 4: Accupal

Syringe Micro Vibrator (SMV)

Syringe microvibrator is a new device introduced to alleviate pain and anxiety of intra oral injections. It has an off-set rotating micro vibration creator with ultra high frequency and ultra low altitude that can be easily placed on any standard dental syringe and some disposable syringes. Upon mounting on a conventional dental anesthesia injection syringe, its motor is switched on and the clinician then uses normal injection technique to administer the anesthetic. Its parallel mounting on the syringe allows clinician to rotate the syringe while in the mouth, if necessary. As it has ultra low vibration altitude, it has no adverse effect on the clinician's dexterity and accuracy during injection and it does not interfere with pin point localization of injection site.

CONCLUSION

Local anesthesia is the corner stone of dental treatment and a great deal of advancement has been made in this field with the aim of allaying the patient's fear of the needle. It is important for the clinician to be familiar with all the available local anesthesia devices and techniques to make treatment less stressful for patient and provide greater comfort in the period following treatment.

REFERENCES

1) IASP Task Force on Taxonomy: Part III: Pain Terms, a Current List with Definitions and Notes on Usage. In Classification of Chronic Pain.. 2 edition. Edited by: Merskey H, Bogduk N. WA, Seattle; IASP Press; 1994:209-214.

2) van Wijk A, Hoogstraten J. Anxiety and pain during dental injections. J Dent 2009;37: 700-4.

3) Vika M, Skaret E, Raadal M, Ost LG, Kvale G. Fear of blood, injury and injections and its relationship to dental anxiety and probability of avoiding dental treatment among 18-year-olds in Norway. Int J Pediatr Dent 2008;18:163-9.

4) Milgrom P, Vignehsa H, Weinstein P. Adolescent dental fear and control: Prevalence and theoretical implications. Behav Res Ther 1992;30:367-73.

5) Berggren U, Meynert G. Dental fear and avoidance: causes, symptoms and consequences. J Am Dent Assoc 1984;109: 247-51.

6) O'Brien L, Taddio A, Lyszkiewicz DA, Koren G: A critical review of the topical local anesthetic amethocaine (Ametop) for pediatric pain. Paediatr Drugs 2005, 7:41-54.

7) Touyz LZ, Lamontagne P, Smith BE: Pain and anxiety reduction using a manual stimulation distraction device when administering local analgesia oro-dental injections: a multi-center clinical investigation. J Clin Dent 2004, 15:88-92.

8) Aminabadi NA, Farahani RM, Balayi Gajan E: The efficacy of distraction and counter stimulation in the reduction of pain reaction in intraoral injection by pediatric patients. The J Contemp Dent Pract 2009; 9:33-40.

9) Ong EL, Lim NL, Koay CK: Towards a pain free venopuncture. Anaesthesia 2000; 55:260-262.

10) Scarfone RJ, Jasani M, Gracely EJ: Pain of local anesthetics: rate of administration and buffering. Ann Emerg Med 1998; 31:36-40.

11) Colaric KB, Overton DT, Moore K: Pain reduction in lidocaine administration through buffering and warming. Am J Emerg Med 1998; 16:353-365.

12) Masters JE: Randomized control trial of pH buffered lignocaine with adrenaline in outpatient operations. Br J Plast Surg 1998; 51:385-387.

13) Orlinsky M, Hudson C, Chan L, Deslauriers R: Pain comparison of unbuffered versus buffered lidocaine in local wound infiltration. J Emerg Med 1992; 10:411-415.

14) Younis I, Bhutiani RP: Taking the 'ouch' out-effect of buffering commertial xylocaine on infiltration and procedure pain - a positive, randomized, doubleblind, controlled trial. Ann R Coll Surg Engl 2004; 86:213-7.

15) Bartfield JM, Crisaffulia KM, Raccio-Robak N, Salluzzo RF: The effects of warming and buffering on pain of infiltration of lidocaine. Acad Emerg Med 1995; 2:254-8.

16) Fitton AR, Ragbir M, Milling MA: The use of pH adjusted lingnocaine in controlling operative pain in the day surgery unit: a positive randomized trial. Br J Plast Surg 1996; 49:404-8.

17) Kakigi R, Shibasaki H: Mechanisms of pain relief by vibration and movement. J Neurol Neurosurg Psychiatry 1992 ; 55:282-286.

18) Lundeberg T, Nordemar R, Ottoson D: Pain alleviation by vibration stimulation. Pain 1984; 20: 25-44.

19) Nanitsos E, Vartuli R, Forte A, Dennison PJ, Peck CC: The effect of vibration on pain during local anaesthesia injections. Aust Dent J 2010; 54:94-100.

20) Roy EA, Hollins M, Maixner W: Reduction of TMD pain by high-frequency vibration: a spatial and temporal analysis. Pain 2003; 101:267-74.

21) Weerakkoby NS, Percival P, Hickey MW, Morgan DL, Gregory JE, Canny BJ, Porske U: Effects of local pressure and vibration on muscle pain from eccentric exercise and hypertonic saline. Pain 2003; 105:425-435.

22) Yarnitsky D, Kunin M, Brik R, Specher E: Vibration reduces thermal pain adjacent dermatomes. Pain 1997; 69:75-7.

23) Dahlin L, Lund I, Lundberg T, Molander C: Vibratory stimulation increases the electro-cutaneous sensory detection and pain thresholds in women but not in men. BMC Complement Altern Med 2006; 6:20.

24) Melzac R, Wall PD. Pain mechanisms: A new theory. Science 1965;150:971 9.

25) Available at: http://www.Dentalvibe.com.

26) Available at: http://www.accupal.com.

GLASS IONOMER CEMENT PAST, PRESENT AND FUTURE : A REVIEW

SOWMYA RAO[1], N. SREE NAGA SWETHA[2], SUDHAMANI K.H[3]

1. Mrs. Sowmya Rao
Lecturer
Dept. of Prosthodontics
Sri Rajiv Gandhi College of
Dental Sciences and Hospital
Cholanagar, Hebbal
Bengaluru, Karnataka

2. Dr. N. Sree Naga Swetha
Lecturer
Dept. of Prosthodontics
Sri Rajiv Gandhi College of
Dental Sciences and Hospital
Cholanagar, Hebbal
Bengaluru, Karnataka

3. Dr. Sudhamani K.H
Lecturer
Sri Rajiv Gandhi College of
Dental Sciences and Hospital
Cholanagar, Hebbal
Bengaluru, Karnataka

For correspondence:
Mrs. Sowmya Rao
Email: sowmyarao67@yahoo.com

ABSTRACT
Glass ionomer cements (GICs) are clinically elegant dental materials that have certain exceptional properties that make them useful as restorative and luting materials. This includes adhesion to moist tooth structures and base metals, anticariogenic properties due to release of fluoride, thermal compatibility with tooth enamel, biocompatibility and low toxicity. The use of GIC in a mechanically loaded situation, however, has been hampered by their low poor mechanical properties. Low fracture strength, toughness and wear, limits their extensive use in dentistry as a filling material in stress-bearing applications. In the posterior dental region, GIC is mostly used as a temporary filling material. The requirement to strengthen those cements has lead to an ever increasing research effort into reinforcement or strengthening concepts.

KEYWORDS
glass ionomer, resin modified glass ionomer cement, compomer, giomer

INTRODUCTION
In dentistry, adhesion of restorative materials to tooth substance is an important objective. The glass ionomer cements are one of the products developed in this direction[1].

Glass-ionomer cements (GIC) was were invented in 1969 and reported by Wilson and Kent in the early 1970's[2]. Glass ionomer is a combination of 'Glass' powder and 'ionomer'-ic acid[3].

GIC can be defined as water based material that hardens following an acid-base reaction between the basic fluoro aluminosilicate glass powder and an acidic solution of polyacrylic acid. Significant amounts of fluoride ions are released during this reaction[4].

HISTORICAL DEVELOPMENT
Invention of the glass ionomer cement in 1969 (reported in Wilson and Kent, 1971) resulted directly from basic studies on dental silicate cement (Wilson et al 1972) and studies where the phosphoric acid in silicate cement was replaced by organic chelating acids (Wilson1968). Glass ionomer cement is a hybrid of dental silicate cement and zinc polycarboxylate cement. GICs first clinical use was reported by Dr.Wilson and, McLean in 1975 and

released for use as ASPA (aluminosilicate polyacrylate) cement[5].

EARLY DEVELOPMENT

Scientific development of GIC has been in two steps. First, effort was devoted to improving properties to make it a fully practical material (example - ASPAI, ASPA II, ASPAIII, ASPAIV, ASPAX, ASPA IVa, and ASPAV[5]) for various applications[5].

LATER DEVELOPMENT

Here properties of GIC are modified in order to extend its range of application[5]. This includes:

Reinforced glass-ionomer cement:

Strength of GIC can be improved by modifying the chemical composition of the original glass powder[1].

A. Disperse phase glasses

1980- Wilson et al observed that clear glasses yielded weaker cements than glasses containing droplets of a disperse phase. So, novel glasses were prepared with deliberately large amount dispersed phases of strengthening crystallites in order to improve the strength (Prosser et al 1986). Suitable disperse phases were found to be corundum (Al2O3), rutile (TiO2), baddeleyite (ZrO2) and tielite (Al2TiO5)[1].

B. Fibre reinforced glasses

The incorporation of filler such as alumina fibre, glass fibre, silica fibre, carbon fibre etc to glass powder is mainly to improve the flexural strength of the cement. Unfortunately these composite materials are very difficult to mix and are having low resistance to abrasion due to lack of bonding between fibre and matrix[1].

C. Metal reinforced glass ionomer cement:

Metal-containing glass-ionomer was developed in the 1980s in an attempt to create stronger and more durable cement. One approach was to add silver-tin alloy powder to GIC called Miracle mix (Miracle Mix, GC America), and another was glass-silver metal hybrid material called a cermet (Ketac Silver, 3M ESPE)[6].

i. Miracle mix (silver alloy admix)

Sced and Wilson (1980) and Simmons (1983) introduced this cement. Simmons mixed amalgam alloy powder into the cements and developed this system clinically under the name "Miracle mix". He used this alloy / glass - ionomer mix for core building and for the treatment of mouth with high caries incidence. However, their aesthetics are poor and they do not take burnish .Their resistance to abrasion is also less than that of regular GIC (More et al,1985) and silver tin amalgam alloy particle did not adhere strongly into the cement matrix[1].

ii. Cermet ionomer cement

In 1985, Mclean and Gasser developed this cement by incorporating metal powder to GICs. Ion leachable calcium aluminium fluorosilicate glass powder is mixed with metal powder (gold and silver). This cement is grayish and more radiopaque and achieves a strong bonding of the metal to the glass[1]. Cermet ionomer cement has improved resistance to abrasion and flexural strength. However, their strength is still insufficient to replace amalgam alloys and their use should be confined to low stress bearing cavity preparations. Fluoride release rate decreases over a time because a portion of the glass particle was metal- coated[1].

D. Resin modified glass ionomer cement

In 1992, Resin modified GIC or hybrid ionomer were developed that could be light cured. Addition of the resin not only decreases initial hardening time and handling difficulties, but substantially increases wear resistance and physical strength of the cement. In these materials, the fundamental acid base reaction is supplemented by a second curing process, which is initiated by light or chemical activation. These products could be dual cure or tricure. Dual cure (material sets by acid base and light activated polymerization reaction). Tri cure cement (material sets by acid base + light + chemical activated polymerization) .They consist of the same components as conventional glass ionomer that is basic glass powder, poly acrylic acid with the addition of a small quantity of a resin such as hydroxyethyl methacrylate (HEMA) or Bis GMA in the liquid. They also contain photosensitive

initiator to bring about free radical polymerization. The first such resin modified GICs developed and marketed were lining cements and restorative versions being introduced later[1,7].

Vitrabond (now spelled "Vitrebond"), a resin-modified glass ionomer base/ liner material, was first introduced by 3M Dental Products Division. Vitrebond is supplied in a powder/liquid format and needs to be spatulated by hand. The liquid polyacid component includes a photopolymerizable resin which hardens the material substantially when a visible light beam (40 seconds) is applied .Once the resin component has been cured, the glass ionomer hardening reaction continues[2]. Light-hardened, resin-modified glass ionomer restorative cements were introduced in the early 1990s. Two of these materials were provided in predosed disposable capsules (Photac-Fil, [3M ESPE, and Fuji II LC, GC), and the other was available only in bottles for hand spatulation (Vitremer, 3M). Fuji II LC was also available in a hand-mixed version. Like Vitrebond, the resin-modified glass ionomer restorative cement harden initially by free radical photo polymerization. Forty seconds of visible light beam exposure substantially hardens these cements initially, and a chemical resin polymerization reaction and the glass ionomer setting reaction subsequently progress[2].

When compared to conventional GIC, resin modified glass ionomers show improved properties like fracture toughness, fracture resistance, and resistance to wear, less early contamination by moisture, less microleakage, and improved adhesion to enamel and denti and significant improvement in esthetic properties[2]. However, resin modified glass ionomer cements suffered from certain drawbacks such as setting shrinkage, limited depth of cure especially with more opaque lining cements.[2]

The development of compomer (Polyacid modified composite) has largely forced this group of materials in to the market[1].

E. Polyacid-modified resin composites (Compomers)

'Compomer' possesses a combination of the characteristics of both composites and glass ionomer. 'Compomer' which contains the major ingredient of both composite and glass ionomer cements (polyalkenoate acid and glass filler component) except for water. Compomer sets by dual cure mechanism. The dominant setting reaction is the resinous photo polymerization and no acid-base reaction can occur until later when the material absorbs water[1,6,7].

F. Easily mixable glass ionomer cements:

In conventional glass ionomer cements dispensing and mixing of the powder and liquid are critical and may introduce a considerable variability in the mechanical and physicochemical properties of the set cement. Hence, other modified versions of dispensing system of glass ionomer cements were introduced[1].

i. Capsules

GIC is available in the form of capsule system. These capsules contain pre measured glass ionomer powder and liquid, which ensures correct ratio, consistency of mix and a predictable result. These capsules have angled nozzle that act as a syringe for accurate placement of the material in to a cavity or a crown for cementation[1].

ii. Paste dispensing system

This dispensing system was designed with the objectives of providing optimum ratio, easy mixing, easy placement, total reliability, using a specially designed cartridge and an easy to use material dispenser. In order to provide the material in a paste paste consistency, an ultra-fine glass powder was designed specifically. The low particle size provides the mixed cement with a thixotropic creamy consistency[1].

iii. Modified powder - liquid system

Specialized processing procedure for powder was followed (Specialized granulates). This system has improved wetting of the powder by the liquid rendering the mixing process much easier and faster[1].

RECENT DEVELOPMENT

1. Glass ionomer stabilization and protection (Fuji VII.) material:

There is need for a material to control the active carious lesions where a temporary restoration is required to seal the cavity during the period of disease stabilization and to protect the susceptible tooth surfaces during "at risk" for intermediate/high risk patients. To accomplish these objectives, specially designed glass ionomer were developed. One commercially available product under this category is Fuji VII. It has a pink chroma for easy identification of margins and as a visual reminder of its temporary nature. Fuji VII is designed as a high fluoride release and thus offers greater protection to surrounding tooth surfaces and its free-flowing consistency to ensure effective wetting and intimate adhesion to tooth surfaces. The fine fluoroalumino-silicate glass filler allows a smooth surface finish and the incorporation of strontium in the glass provides radiopacity, enhanced remineralization capabilities and a sharp snap set. Fuji VII sets in around four minutes. The setting reaction of the pink shade of Fuji VII can be accelerated by light curing for 20-40 seconds using a halogen curing light (the pink color absorbs light energy which accelerates the setting reaction)[1].

2. Amino acid modified glass ionomer cements:

One of the factors affecting the strength of glass ionomer is the chemical composition of the polymer matrix. Most of the conventional glass ionomer contain homopolymer or copolymer of unsaturated mono-, di - and tri carboxylic acids. With these formulations, the major problem lies in that the acrylic acid homo or copolymers have COOH groups which are directly attached to the backbone and are closely oriented to each other, resulting in a rigid polymeric structures. It is presumed that strength or fracture resistance of the ionomer material is weakened due to this steric hindrance, which brings about a significantly reduced COO Al+++ interaction in the set cement. So acrylic acid copolymers were modified with N acryloyl- or N-methacryloylamino acids, such as N methacryloyl glutamic acid, providing a possible path to improved conventional glass ionomers. These newly formulated polyacids have flexible side chains tethering the carboxylic acid groups at various distances from the main chain polymer backbone, allowing for more freedom and less steric hindrance when the carboxylic acid groups are undergoing chemical reactions. This type of modification has improved the fracture toughness of the glass ionomer cement. The copolymers with pendant amino acid residues have also been developed for preparing visible light cure formulations. The monomer N Vinylpyrrolidinone (NVP) has been explored for modification of poly (acrylic acid co itaconic acid), providing a path to new polyelectrolytes for formulating glass ionomer. Formulations containing NVP residues have also developed for VLC[1].

3. Giomer

In recent years, a new type of glass filler known as GIOMER has been receiving attention in clinical papers. Giomers are relatively a new type of restorative material. Although widely accepted in Japan for the past 15 years, it has only recently caught on in the United States. The name "giomer" is a hybrid of the word "glass ionomer" and "composite". They have properties of both glass ionomer (fluoride release, fluoride recharge) and resin composites (excellent esthetics, easy polishability and bio-compatibility).

Composition of giomer- Bisphenol A glycidyle di methacrylate, TEGDMA, inorganic glass filler, aluminoxide, silica, pre-reacted glass ionomer filler, DL-camphoroquinone[8].

PRG technology in Giomer- There is a pre-reaction of Fluoroalumino silicate glass fillers with Polyacrylic acid, the reaction produce a glass ionomer which is more stable. This phase is called "WET SILICEOUS HYDROGEL". This material is freeze dried, milled, treated with silane and then ground to produce PRG fillers, then these glass fillers are added to the resin matrix (GIOMER)[8].

PRG Technology is used in production of two types of fillers. They are S-PRG (surface pre- reacted glass ionomer) marketed as BEAUTIFIL (Shofu) and F-PRG (fully pre-reacted glass ionomer)[8].

Giomer are similar to compomer and resin composites, and are light-curable. They need to be bonded using the typical resin bonding systems (FL Bond II), which involve surface treatment and the application of the bonding agents. Shofu has successfully incorporated the material into composite resins such as BEAUTIFIL Flow Plus, BEAUTIFIL II and the bonding agent FL Bond II with great clinical success. Only one giomer is commercially available at this time, Shofu's Beautifil, which uses the S-PRG technology. According to Shofu, Beautifil is indicated for restoring Class I through V lesions as well as for treating cervical erosion lesions and root caries. It is available in 13 shades and is supplied in syringes [6,8,9]. Little published research is available on the properties or performance of giomer. One recently published study compared the fluoride release of a glass ionomer, a resin-modified glass ionomer, a giomer, and a compomer. It found that while the giomer released fluoride, it did not have an initial "burst" type of release like glass ionomers, and its long-term (i.e., 28-day) release was lower than that of the other materials. Another study found that a giomer, after polishing with Sof-Lex disks, had a smoother surface than a glass ionomer, (giomer are easier to polish than glass-ionomers)[9]. A three-year clinical study comparing the performance of a giomer with that of a micro fill resin composite in Class V erosion/abrasion/fraction lesions has also been done. After measuring eight performance characteristics, no significant differences between the two materials were found[9].

Independent evaluation of S-PRG materials conducted by the University of Florida and published in JADA, at eight years, none of the restorations failed, no sensitivity was reported, anatomical form was well-maintained and, most notably, no secondary caries were present in any of the patient[9].

iv. Bioactivity of glass ionomers

In recent years, the ability of glass ionomers to release ions apart from fluoride, notably calcium and aluminum, has been studied, and there is evidence to show that they promote remineralization of the tooth. This seems to be related to their ability to buffer lactic acid, an effect that was originally thought to be negative, because of its association with loss of cement by erosion. However, very recently, it has been found that lactic acid at the pH of active caries can be buffered to the pH of arrested caries within less than 30 seconds, and with negligible erosion. This effect is likely to be beneficial, and would inhibit the development of secondary caries around a glass ionomer restoration[2].

CONCLUSION

In recent years there have been considerable changes in the formulations, properties and handling properties of the glass ionomer cements for different clinical applications. It is certain that no material is perfect, but with the current level of intensive research on glass ionomer, the deficiencies that exist seem to be eliminated or at least reduced, resulting in an ever improving range of materials of this type. Almost assuredly, many other GIC products will become available in the future.

REFERENCES

1. Upahya NP and Kishore G. Glass ionomer cement-the different generations. Trends Biomater Artif.Organs, 2005; 18(2):158-65.
2. Croll TP and Nicholson JW, Glass ionomer cements in pediatric dentistry a review of the literature. Pediatric Dent, 2002; 24: 423-5.
3. Mahesh Singh TR, Suresh P, Sandhyarani J.Sravanthi J. Glass ionomer cement in dentistry-a review. IJPAES, 2011; 1: 26-30.
4. Lohbauer U. Dental glass ionomer cement as permanent filling material- properties, limitation and future trends. Materials 2005; 3:76-96.
5. Wilson AD, McLean JW. *Glass Ionomer Cement*, Chicago: Quintessence publishing .Co; 1988:13-20.
6. Nicholson JW, Update on glass ionomer cement. Dental forum 2005; 23:73-9.
7. Hse K.M.Y, Leung S.K and Wei S.H.Y. Resin

ionomer restorative materials for children - a review. Australian Dental Journal 1999;44(1):1-11.

8. GIOMER materials: Essential ingredients for a healthy smile. Dental Tribune Daily U.S. Edition, Feb. 25, 2012:5-11.

9. Composites/Compomers/Giomers [Internet]: *USAF Dental Evaluation and Consultation Service* (cited Jan 2003). Available from: WWW.afms.af.mil/shared/media/document/AFD-130327-261.pdf.

MOLAR INCISOR HYPOMINERALISATION: A REVIEW

SMITHA M [1], SANTHOSH T. PAUL [2]

1. Dr. Smitha M
Post Graduate Student
Dept of Pedodontics
& Preventive Dentistry
Sri Rajiv Gandhi College of
Dental Sciences
Bangalore, Karnataka

2. Dr. Santhosh T. Paul
Professor and HOD,
Dept of Pedodontics
& Preventive Dentistry
Sri Rajiv Gandhi College of
Dental Sciences
Bangalore, Karnataka

For correspondence:
Dr. Smitha M
Email: smithjuno@rediffmail.com

ABSTRACT
Molar Incisor Hypomineralisation (MIH) is a common developmental condition resulting in enamel defects in first permanent molars and permanent incisors. The prevalence of MIH in the different studies ranges from 2.4-40.2%. Several factors are associated with etiology of MIH. Early diagnosis is essential since rapid breakdown of tooth structure may occur, giving rise to acute symptoms and complicated treatment. Management of these teeth should take into consideration their long-term prognosis, as well as management of the presenting features such as pain.

KEY WORDS
dental enamel abnormalities, hypomineralisation, molars, incisors.

INTRODUCTION
During the past few decades, there has been a decline in caries prevalence in all age groups, especially in industrialised countries[1]. Although, occlusal caries still accounts for the majority of caries experience in children, a rapid caries progression in first permanent molars is not common anymore in contemporary populations. However, pediatric dentists are still confronted with large defects in first permanent molars during or soon after eruption, referred to as Molar Incisor Hypomineralisation (MIH)[2]. In the dental literature, a lot of terms have been used to refer to hypomineralized molars including enamel opacities, non fluoride enamel opacities, cheese molars, non fluoride hypomineralisation and idiopathic enamel opacities[3]. Molar Incisor Hypomineralization (MIH) is a qualitative and quantitative defect of the dental enamel. Clinically, MIH molars can lead to various problems for the dentist as well for the affected child. For dentists, the problems are related to rapid development of caries in the erupting first permanent molar, difficulty to anesthetize the MIH molar when treatment is indicated and unpredictable behavior of apparently intact opacities. The child on the other hand, will experience pain and sensitivity even while brushing. General dental practitioners tend to neglect the fact that the affected molars are lost due to the rapid progression of caries and often do not consider interdisciplinary treatment. Early identification and treatment results in successful management of the affected molars and

incisors.

Nomenclature

Weerheijm et al [2] suggested the term Molar Incisor Hypomineralisation and defined it as hypomineralisation of systemic origin of one to four permanent first molars frequently associated with affected incisors. This description was chosen to put emphasis on the fact that molars are always involved in the phenomenon and often there is a combination of molars with demarcated opacities of the incisors.

Prevalence

The epidemiologic data, present in the literature of the last fifteen years, are only limited and turned mostly to studies of prevalence. Many prevalence studies for MIH were carried out in various countries and large variations were found in the prevalence rates. The prevalence of MIH is reported to vary between 2.4 and 40.2% in normal child populations [4]. The large variation could be because of difference in recording methods, indices used and different age or population investigated. Presently there is no conclusive evidence that maxillary molars are more susceptible for MIH than mandibular molars or vice versa[3].

Etiology

The combination of affected molars together with incisors suggests that in case of MIH there is a specific influence on the development of enamel during a limited period of time. Enamel formation is a sensitive process, which can be divided into several stages. A disturbance occurring during the maturation phase will be clinically visible as an enamel opacity, which suggests that in case of MIH the ameloblasts are affected in the (early) maturation stage. In MIH, the lesions in the first permanent molars are often seen together with those in the maxillary and, more rarely, the mandibular incisors. These findings indicate a systemic upset during the first years of a child's life, more precisely during the period in which the crowns of permanent first molars and incisors are mineralised. In general, the defects of the incisors are milder than those of the molars. As masticatory forces on the opacities in incisors are absent, the enamel substance does not disintegrate so easily after eruption. Many studies have analyzed a wide variety of causes for MIH [5,6].

1. **Prenatal period:** The last trimester of pregnancy is a critical period during which the amelogenesis of permanent first molars and incisors teeth starts. Multiple episodes of maternal high fever, viral infections like rubella and chickenpox, prolonged medications during the last month of pregnancy, prolonged vomiting up to last month, urinary infections, maternal hypertension, maternal diabetes, renal deficiency, malnutrition during the last trimester of pregnancy are some of the presumed causative factors listed [6,7,8,9].

2. **Perinatal period:** In the peri-natal period different medical conditions alone or in combination may affect the welfare of a child [5, 9]. In a Greek study, the most common peri-natal problems/conditions associated with MIH were caesarian section, prolonged/complicated delivery, premature birth and twinning. Hypoxia, low birth weight, hemorrhage and detachment during delivery are other peri-natal causes for defective ameloblast function [9].

3. **Post-natal period:** Special attention has been paid to prolonged childhood illnesses, prolonged high fever due to infections, repeated/prolonged medications (antibiotics like amoxicillin)[10,11] and exposure to environmental contaminants such as polychlorinated biphenyls and polychlorinated dibenzop-dioxins / dibenzofurans (dioxins) [12]. Infections such as otitis media, pneumonia, asthma, bronchitis, upper respiratory tract infections, urinary tract infections and exanthamatous diseases like chickenpox, rubella, measles have been positively associated with MIH [8,9,13].

However, various investigators have concluded that MIH is not caused by one factor alone but by many different ones, and that several factors may act together at sensitive stages of amelogenesis, increasing the risk [6,14].

Diagnostic criteria for MIH

For accurate diagnosis of the condition of MIH, a simple, reproducible scoring index is needed.

European Academy of Paediatric Dentistry held a meeting at Athens in 2003, and outlined the judgment criteria for MIH in epidemiological studies [15]. An examination for MIH should be performed on wet teeth after cleaning. Eight years of age was considered as the best time for any examination for the condition [16]. The most appropriate teeth to be examined are the 4 first permanent molars and 8 permanent incisors. For a patient to be diagnosed as suffering from MIH, they should have at least one permanent first molar affected with or without the involvement of incisors. However, if a patient has opacities affecting the incisors only, the condition is not MIH. Judgements related to individual teeth should be recorded for:

- absence or presence of demarcated opacities;
- posteruptive enamel breakdown;
- atypical restorations;
- extraction of molars due to MIH;
- failure of eruption of a molar or an incisor.

Table 1 shows the definitions of the criteria just mentioned above and Fig 1 illustrates the definitions of the criteria given in Table 1.

Demarcated Opacity	A demarcated defect involving an alteration in the translucency of the enamel, variable in degree. The defective enamel is of normal thickness with a smooth surface and can be white, yellow or brown in colour.
Post eruptive enamel breakdown (PEB)	A defect that indicates deficiency of the surface after eruption of the tooth. Loss of initially formed surface enamel after tooth eruption. The loss is often associated with a pre-existing demarcated opacity
Atypical restoration	The size and shape of restorations are not conforming to the temporary caries picture. In most cases in molars there will be restorations extended to the buccal or palatal smooth surface. At the border of the restorations frequently an opacity can be noticed. In incisors a buccal restoration can be noticed not related to trauma.
Extracted molar due to MIH	Absence of a first permanent molar should be related to the other teeth of the dentition. Suspected for extraction due to MIH are: opacities or atypical restorations in the other first permanent molars combined with absence of a first permanent molar. Also the absence of first permanent molars in a sound dentition in combination with demarcated opacities on the incisors is suspected for MIH. It is not likely that incisors will be extracted due to MIH.
Unerupted	The first permanent molar or the incisor to be examined are not yet erupted.
Notes	In cases of a large caries lesion with demarcated opacities at the border of the cavity or on the non caries surfaces, these teeth should be judged as MIH. Other changes in dental enamel such amelogenesis imperfecta, hypoplasia, diffuse opacities, white spot lesions, tetracycline staining, erosion, fluorosis, white cuspal and marginal ridges should be excluded from the types of enamel defects outlined as above.

Table 1. Definitions of the judgement criteria to be used in diagnosing MIH for prevalence studies.

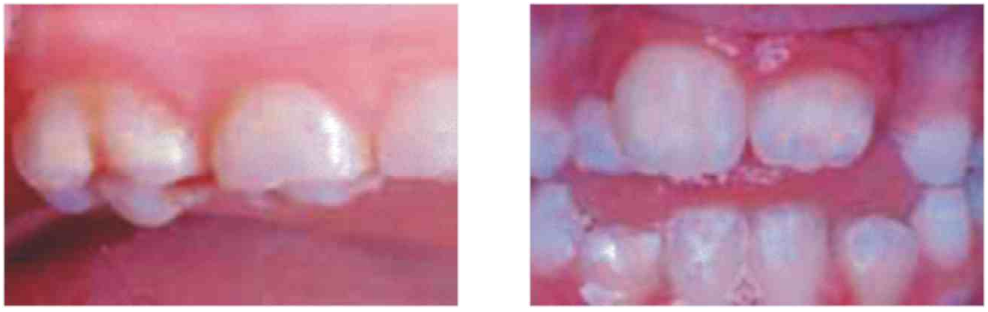


Fig 1 A. Demarcated opacities in enamel of molars and incisors

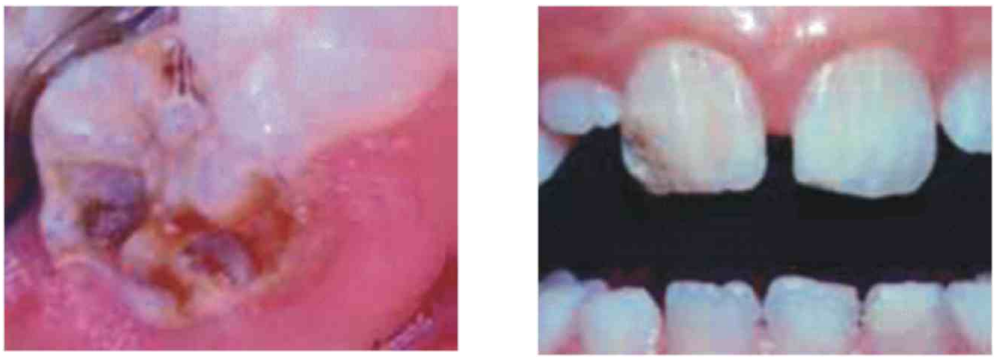


Fig 1 B. Disintegrated enamel of molars and incisors.

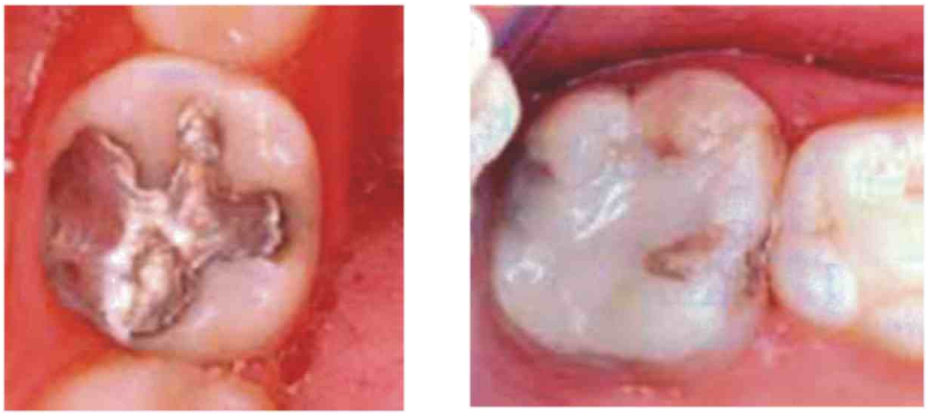


Fig 1 C. Atypical restorations

Initially hypomineralized areas were categorized into three grades of severity: severe (loss of enamel in association with affected dentin), moderate (loss of enamel only), and mild (colour change: white, yellow or brown) [17]. After encountering certain difficulties in differentiating between moderate and severe cases, Lygidakis et al[16] proposed a classification with mild and moderate-severe categories. The mild category includes intact enamel surfaces with occasional sensitivity and mild aesthetic concerns. Post-eruptive breakdown, persistent or spontaneous hypersensitivity and strong concerns regarding aesthetics form the moderate-severe category. Severity can vary between individuals and can also vary within the mouth of a single individual. Fig 2 and 3 illustrates the difference in severity within the mouth of a single individual.

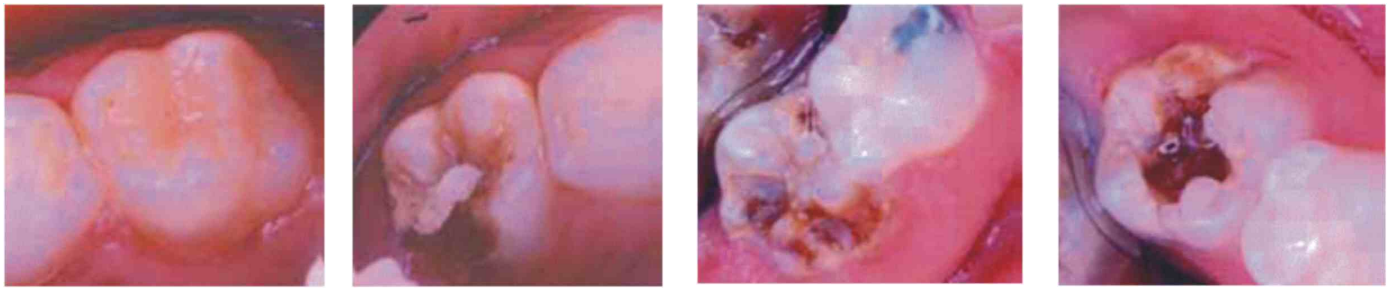


Fig 2. Asymmetric appearance in molars. The four molars are from the same child.

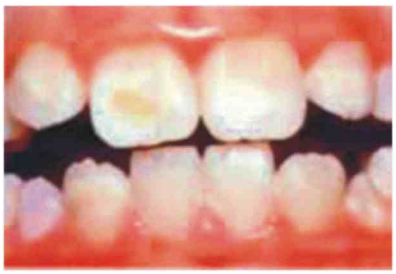


Fig 3. Asymmetric appearance in incisors

Problems specific to MIH in a young child

MIH presents with the eruption of the first permanent molars and permanent incisors. Therefore, at age six to eight years significant dental treatment may be required, which can prove a challenge in such a young age group. Hypersensitivity is a common complication of MIH, making oral hygiene and eating difficult, while further compromising the defective teeth. Hypersensitivity may also complicate the clinical management of MIH. When present, profound analgesia will be essential for all procedures. Therefore, even non-invasive preventive clinical procedures such as fissure sealants may pose significant discomfort for these young children, increasing their anxiety and causing behaviour management problems.

The rapid post-eruptive enamel breakdown that may arise in MIH poses another clinical problem for this group of children. By the time the affected molar is fully erupted, preventive measures may no longer suffice and a more extensive restoration is needed. Should the degree of severity warrant a more radical solution to care, an endodontic or orthodontic opinion should be sought. A combined team approach to treatment planning will maximise options and ensure the best treatment outcome. Incisor involvement may give rise to aesthetic problems, which again require early intervention to deal with unsightly defects or opacities.

Optimal treatment should be established on a case-dependent basis. The child's compliance, severity of hypomineralization, occlusion, extent of treatment required, financial cost, investment of time, and the long-term prognosis of the teeth are just some of the many factors that may determine the appropriate treatment option [18].

Management

MIH may lead to extensive treatment need. If an erupting first permanent molar shows signs of opacities and/or post-eruptive breakdown, the child should be monitored frequently until the moment that all four molars have completely erupted. In order to minimize the loss of enamel and any damage due to caries, both preventive and interceptive treatment is required. The management of MIH depends largely on the severity of the enamel defect. Determining the severity of MIH will provide the basis for selecting an appropriate treatment. The severity will be largely determined by the size of the lesion and the degree or extent of hypomineralization [16]. The larger degree of enamel loss with compromised masticatory function is followed by rapid breakdown of the tooth. This leads to a more extensive and potentially difficult treatment. A very useful 6-step management approach for MIH has been proposed by William et al [5]. It includes risk identification, early diagnosis,

remineralisation and desensitization, prevention of dental caries and post eruptive enamel breakdown, restorations or extractions and maintenance. Treatment protocol based on severity of MIH is given in Table 2.

Tooth	Mild MIH	Moderate MIH	Severe MIH
Incisor	Remineralization solution, Tooth mousse, Fluoride Mouthrinses.	May require micro abrasion, bleaching followed by Composite resin veneers	May require micro abrasion, bleaching followed by composite resin veneers and later porcelain veneer.
Molars	Dental sealants, Fluoride varnish	Preventive resin restoration	Restorable -Stainless steel crown Non restorable - Extraction followed by orthodontic treatment
Recall	Biannually	Thrice annually	Biannually

Table 2. Treatment protocol based on severity of Hypomineralization.

CONCLUSION

The first permanent molar has a significant role in development of occlusion and its early loss can have a considerable effect on dental health of the child in future. It seems advisable to monitor children with a poor general health more frequently in the first four years after birth for MIH [13]. It is important that MIH is diagnosed early. This ensures that appropriate treatment can be provided in an optimum timeframe. It also ensures that the risk and complications of post-eruptive enamel breakdown are minimized. In all cases of MIH, it is essential that the young child be reviewed on a regular basis in order to assure their long-term dental health.

REFERENCES

1. Petersen PE . The World Oral Health Report 2003: Continuous improvement of oral health in the 21st century the approach of the WHO Global Oral Health Programme. Community Dentistry and Oral Epidemiology (2004) 31 (Suppl 1): 3-24.
2. Weerheijm KL, Jälevik B, Alaluusua S. Molar-Incisor Hypomineralisation. *Caries Res* 2001; **35**: 390-91.
3. Weerheijm KL. Molar incisor hypomineralization (MIH). Eur J Paediatr Dent. 2003; 4(3):115-20.
4. Jalevik B. Prevalence and diagnosis of Molar-Incisor- Hypomineralisation (MIH): a systematic review. Eur Arch Paediatr Dent 2010; 11: 59-64.
5. William V, Messer LB, Burrow MF. Molar incisor hypomineralization: review and recommendations for clinical management. Pediatr Dent. 2006; 28(3):224-32.
6. Crombie F, Manton D, Kilpatrick N. Aetiology of molar-incisor hypomineralization: a critical review. Int J Paediatr Dent. 2009; 19 (2):73-83.
7. Mahoney EK, Morrison DG. The prevalence of

Molar-Incisor Hypomineralization (MIH) in Wainuiomata children. N Z Dent J. 2009; 105(4):121-7.

8. Whatling R, Fearne JM. Molar incisor hypomineralization: a study of aetiological factors in a group of UK children. Int J Paediatr Dent. 2008;18(3):155-62.

9. Lygidakis NA, Dimou G, Marinou D. Molar-incisor-hypomineralization (MIH). A retrospective clinical study in Greek children. II. Possible medical aetiological factors. Eur Arch Paediatr Dent. 2008; 9(4):207-17.

10. Hong L, Levy SM, Warren JJ, Dawson DV, Bergus GR, Wefel JS. Association of amoxicillin use during early childhood with developmental defects. Arch Pedatr Adolesc Med 2005; 159:943 -948.

11. Laisi S, Ess A, Sahlberg C, Arvio P, Lukinmaa PL, Alaluusua S. Amoxicillin may cause Molar Incisor Hypomineralization. J Dent Res 2009; 88 (2):132 -136.

12. Laisi S, Kiviranta H, Lukinmaa PL, Vartiainen T, Alaluusua S. Molar Incisor Hypomineralisation and Dioxins: New findings. Eur Arch Paediatr Dent. 2008; 9 (4):224- 227.

13. Beentjes VE, Weerheijm KL, Groen HJ. Factors involved in the aetiology of molar-incisor hypomineralization (MIH). Eur J Paediatr Dent. 2002; 3(1):9-13.

14. Alaluusua S. Aetiology of molar-incisor hypomineralization: a systematic review. Eur Arch Paediatr Dent 2010; 11(2):538.

15. Weerheijm KL, Duggal M, Mejàre I, Papagiannoulis L, Koch G, Martens LC, Hallonsten AL. Judgement criteria for molar incisor hypomineralization (MIH) in epidemiologic studies: a summary of the European meeting on MIH held in Athens, 2003. Eur J Paediatr Dent. 2003; 4(3):110-3.

16. Lygidakis NA, Wong F, Jälevik B, Vierrou AM, Alaluusua S, Espelid I. Best Clinical Practice Guidance for clinicians dealing with children presenting with Molar-Incisor-Hypomineralization (MIH): An EAPD Policy Document. Eur Arch Paediatr Dent. 2010; 11 (2):75-81.

17. Alaluusua S, Lukinmaa PL, Vartiainen T, Partanen M, Torppa J, Tuomisto J. Polychlorinated dibenzo-pdioxins and dibenzofurans via mother's milk may cause developmental defects in the child's teeth. Environ Toxicol Pharmacol 1996; 1:193-7.

18. Daly D, Waldron JM. Molar Incisor Hypomineralisation: clinical management of the young patient. Journal of the Irish Dental Association 2009; 55 (2); 83- 86.

OZONE THERAPY : A REVIEW

JOGULINE VINITA[1], VIJAY K RAGHAVA[2], UMESH YADALAM[3]

1. Dr.Joguline Vinita
Post graduate student,
Department of Periodontics,
Sri Rajiv Gandhi College of
Dental Sciences and Hospital
Cholanagar, Hebbal
Bengaluru, Karnataka

2. Dr.Vijay K Raghava
Reader,
Department of Periodontics,
Sri Rajiv Gandhi College of
Dental Sciences and Hospital
Cholanagar, Hebbal
Bengaluru, Karnataka

3. Dr.Umesh Yadalam
Reader,
Department of Periodontics,
Sri Rajiv Gandhi College of
Dental Sciences and Hospital
Cholanagar, Hebbal
Bengaluru, Karnataka

For correspondence:
Dr. Joguline Vinita
Email: joguline@yahoo.co.in

ABSTRACT
This review of literature is an attempt to summarize different modalities of ozone application in dentistry. Ozone gas has a high oxidation potential and is effective against bacteria, viruses, fungi, and protozoa. It also has the capacity to stimulate blood circulation, platelets, and immune response. Gingivitis and periodontitis are most common inflammatory diseases of supporting tissues of teeth. Role of microbial etiology and host response in progression of gingival and periodontal diseases has been well established. Because of the beneficial biological effects of ozone, due to its antimicrobial and immunostimulating effect, it is well indicated in the treatment of gingival and periodontal diseases. Owing to the anti inflammatory, immunomodulating effects of ozone and its positive influence on the microcirculation, the ozone therapy is now widely used in dental field

KEYWORDS
antimicrobial, immunostimulating, antihypoxic, biosynthetic

INTRODUCTION
Ozone is a colorless gas form of oxygen and is present in atmosphere. It is one of the most important gases in the stratosphere due to its ability to filter ultraviolet rays, which is critical for the maintenance of biological balance in the biosphere. It has been used to purify water throughout the world for many years as it is highly effective in killing bacteria present in different forms. It effectively kills bacteria, fungi, viruses and parasites at a lower concentration[1].

Ozone is a chemical compound consisting of three oxygen atoms (O3triatomic oxygen), a higher energetic form than normal atmospheric oxygen (O^2). Molecular weight of ozone is 41.98 g/mol. It is a powerful oxidizer[2]. It protects living organisms by surrounding the earth at altitudes of 50,000 100,000 feet. One molecule of ozone is equal to 3000 - 10,000 molecules of chlorine and kills pathogenic organisms 3500 times faster[3].

Effect on bacteria, virus, fungus, protozoa
Bacteria
Ozone acts on bacterial cell membranes, by oxidation of their lipid and lipoprotein components. There is evidence for interaction with proteins as well. Ozone seems to render the spores defective

in germination, perhaps because of damage to the spore's inner membrane[4].

Virus

All viruses are susceptible to ozone; yet differ widely in their susceptibility.Lipid-enveloped viruses are especially sensitive to ozone. Analysis of viral components showed damage to polypeptide chains and envelope proteins impairing viral attachment capability, and breakage of viral RNA[5,6,7].

Fungal and protozoa

Ozone inhibits cell growth at certain stages[7].

Effect on blood cells

Ozone reduces or eliminates clumping of red blood cells and its flexibility is restored, along with oxygen carrying ability. There is a stimulation of the production of glutathione peroxidase, catalase, and superoxide dismutase which act as free radical scavengers[8,9].

Effect on leukocytes

Ozone behaves as a weak cytokine such as tumor necrosis factor-α (TNF-α), interleukin-2, interleukin-6, interleukin-8, transforming growth factor-β [TGF-β]) inducer[10-13]. Ozone reacts with the unsaturated fatty acids of the lipid layer in cellular membranes, forming hydrogen peroxides (H_2O_2), one of the most significant cytokine inducers[14].

Platelets

H_2O_2 generated by blood ozonation activate phospholipase C, phospholipase A2, cyclo-oxygenases and lipooxygenases, and thromboxane synthetase, allowing a step increase of intracellular calcium, release of prostaglandin E2, prostaglandin F2a, and thromboxane A2 with irreversible platelet aggregation[15-17].

Ozone generators

There are three different systems for generating ozone gas: [18]

Ultraviolet System: produces low concentrations of ozone, used in esthetics, saunas, and for air purification.

Cold Plasma System: used in air and water purification

Corona Discharge System: produces high concentrations of ozone. It is the most common system used in the medical/ dental field. It is easy to handle and it has a controlled ozone production rate.

Medical grade ozone is a mixture of pure oxygen and pure ozone in the ratio of 0.05% to 5% of O3 and 95% to 99.95% of O2. Due to the instability of the O3 molecule, medical grade ozone must be prepared immediately before use.

ROUTES OF ADMINISTRATION

Gaseous ozone Ozone can be used in gaseous form via an open system or via a sealing suction system to avoid inhalation and its adverse effects.

Ozonated water Ozonated water has been shown to be very effective against bacteria, fungi and viruses.

Ozonized oil In addition to gaseous and aqueous form, oils that are ozonized also seems extremely convenient.

Though gaseous ozone was shown to have more effective microbicidal properties than aqueous form, due to its toxic effects if inhaled, ozonated water is the most preferred form for use in dentistry. Therefore a safe system for applying gaseous ozone

into the periodontal pocket that avoids inhalation still needs to be developed[19].

APPLIANCES PRODUCING OZONE FOR DENTAL USE

1. HealOzone by KaVo is air-based and the application of the gas takes place in a closed circuit.

2.OzonyTron by MYMED Gmb H. - Oxygen activation generator (OzonytronXBiozonix, München, Germany) uses the power of high frequency and voltage. Activated oxygen (ozone) concentration can be adjusted in 5 levels via current strength. There is no closed circuit here, therefore, ozone can be applied to the places that are difficult to reach, e.g. gingival pockets or root canals.

3. Product photo (Prozone) by W&H - It is characterized by its ease of use and safety of application (present tissue compatible dosages in the indication areas of periodontitis and endodontitis). Prozone ensures a hygienic procedure during the gassing of the pockets due to its exchangeable plastic attachments (Perio tips or Endo tips).

BIOCOMPATIBILITY OF OZONE

A study investigated cytotoxic effects of gaseous ozone and aqueous ozone on human oral epithelial (BHY) cells and gingival fibroblast (HGF-1) cells compared with established antiseptics chlorhexidine digluconate (CHX) 0.2%; sodium hypochlorite (NaOCl) 5.25%, 2.25%; hydrogen peroxide H2O2 3%. Aqueous ozone revealed the highest level of biocompatibility of the tested antiseptics[20].

Advantages

- Disinfectant
- Anti - inflammatory
- Activation of intracellular metabolism of oral mucosa and dental wounds
- Improvement of regional circulation
- Stimulation of regenerative processes
- Hemostasis in capillary bleedings
- Painless procedures

Disadvantages

- Ozone toxicity if the level increases at 0.0007% per application
- Instability
- Not readily available

Indications

- Chronic or recurrent infections in the oral cavity[21]
- Prophylaxis and prevention of dental caries
- Remineralization of pit and fissure caries, root and smooth surface caries
- Bleaching of discolored root canal treated teeth.
- Sterilization of cavities, root canals, periodontal pockets, herpetic lesions
- Desensitization of extremely sensitive tooth necks
- Pre washing of surgical sites
- Plaque control
- Contamination control

- **Contraindications**

The following are contraindications for use of ozone therapy:[22]

- Pregnancy
- Glucose-6-phosphate-dehydrogenase deficiency (favism)
- Hyperthyroidism
- Severe anaemia
- Severe myasthenia
- Active hemorrhage
- Acute alcohol intoxication

- Recent Myocardial infarction

BIOLOGICAL ACTIONS

Antimicrobial effect

Ozone works destructively against bacteria, fungi and viruses. The antimicrobial effect of ozone is a result of its action on cells by damaging its cytoplasmic membrane due to ozonolysis of dual bonds and also ozone induced modification of intracellular contents because of secondary oxidant effects. This action is selective to microbial cells but does not damage human body cells because of their major antioxidative ability[23].

Immunostimulating Effect

Ozone influences cellular and humoral immune system. It stimulates proliferation of immunocompetent cells and synthesis of immunoglobulins. It also activates function of macrophages and increases sensitivity of microorganisms to phagocytosis. Ozone causes the synthesis of biologically active substances such as interleukins, leukotrienes, and prostaglandins which is beneficial in reducing inflammation and wound healing. Ozone in high concentrations causes immunodepressive effect whereas in its low concentration immunostimulating effect[24].

Antihypoxic Effect

Ozone improves the transportation of oxygen in blood, which results in change of cellular metabolism activation of aerobic processes (glycolysis, Krebs cycle, β oxidation of fatty acids) and use of energetic resources. Ozone improves the metabolism of inflamed tissues by increasing their oxygenation and reducing total inflammatory processes[25].

EFFECT OF OZONE ON BACTERIA

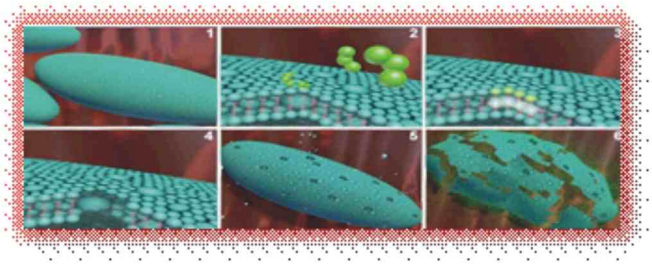


Fig-1 Ozone molecule coming into contact with bacterial cell wall - penetrating & creating pores in bacterial cell wall resulting in cell lysis

Biosynthetic effect

It activates mechanisms of protein synthesis and increases amount of ribosomes and mitochondria in the cells. These changes on the cellular level explain elevation of functional activity and regeneration potential of tissues and organs.

GOALS OF OZONE THERAPY

Setting the standard-of-care and therapeutic goals are based on sound evidence-based science is critical. Therapeutic goals are inclusive and not exclusive of standard of care.

The goals of oxygen/ozone therapy are: [26]

1. Elimination of pathogens.
2. Restoration of proper oxygen metabolism.
3. Induction of a friendly ecologic environment.
4. Increased circulation.
5. Immune activation.
6. Simulation of the humoral anti-oxidant system.

USES IN DENTISTRY

- **Periodontology-** Gingivitis, Periodontits, Periimplantitis, Surgical cuts, Prophylaxis
- **Dental and oral pathology-** Caries, Enamel cracks, Root canal treatment, Tooth whitening, Dentinal hypersensitivity, Abscess, Granuloma, Fistulae, Apthae, Herpes infection, Stomatitis Candidiasis

- **Surgery-** Implantation, Re-plantation, Extraction, Wound Healing, Coaguloapathy - prolonged bleeding
- **Prosthodontics and Restorative dentistry-** Stumps and crown disinfection, Cavity disinfection
- **Orthodontics and Orthopedics-** TMJ dysfunctions, Trismus, Relaxation, Myoarthtopathy
- **Diagnostics -** Vitality test
- **Decontamination of dental unit water line-** Water becomes stagnant when the units are not in use. Detachment of microorganisms, splatter, and aerosols from dental procedures may possibly infect health care personnel. Ozone has been used for purification of water due to its efficiency and lack of side effects. Kohno et al published their results that indicated acidic electrolyzed water could be applied as an appropriate measure against bacterial contamination of the DUWL[27,28,29].

In model dental unit water lines, ozone achieved a 57% reduction in biofilm and a 65% reduction in viable bacteria in spite of being used in a very low dose and with a short time of application[30].

Uses in periodontology

Ozonated water inhibited the accumulation of experimental dental plaque in vitro. Ozonated water had strong bactericidal activity against bacteria in plaque biofilm. It was found that ozonated water (0.54 mg/L) was highly effective in killing of both gram-positive and gram-negative microorganisms. Gram-negative bacteria, such as Porphyromonas gingivalis, Porphyromonas endodontalis were more sensitive to ozonated water than gram-positive oral streptococci and Candida albicans in pure culture[31].

One study concluded that the addition of ozone to a ultrasonic cleaning system containing different experimental solutions resulted in antibacterial activity against Staphylococcus aureus. Ozonated water has an excellent anti- inflammatory capacity. Researchers choose the NF-kappaB system, a paradigm for inflammation-associated signaling/transcription. Their results showed that that NF - kappaB activity in oral cells in periodontal ligament tissue from root surfaces of periodontally damaged teeth was inhibited following incubation with ozonized medium[32,33,34].

The use of ozone around implants is supported by published research showing that ozone not only effectively sterilizes the surfaces of both the implant and bone, but also initiates the reparative mechanisms allowing tissue regeneration around implant surface[35,36].

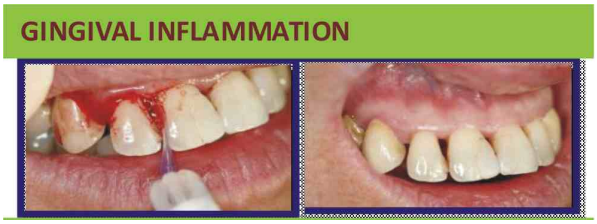


Fig.2

IRRIGATION WITH OZONATED WATER

Application modalities in Periodontology

According to the clinical case, different applications modalities are available using ozone gas, irrigation with Ozonated water and in-office use of ozonized oil as well as home use[37].

I. NON SURGICAL PERIODONTAL THERAPY

1) **Gas application via a customized thermoformed dental appliance**
2) **Irrigation with Ozonated Water -** Ozonated

water can be used to irrigate the affected area during and after scaling, root surface planning, and non-surgical pocket curettage.

3) **In-office and Home Use of Ozonized Olive Oil -** After in-office treatment with ozone gas or Ozonated water, pockets can be filled with ozonized olive oil using a blunt 25G needle or any other appropriate tip. Patient can be given some of the oils for home use. In-office ozonized oil application can be repeated once a week.

II. Surgical therapy

Surgical Procedures- Ozonated water can be used as an irrigant during the surgical procedure and/or as a final surgical site lavage. The sutures can be covered with a thin layer of ozonized oil and the patient can be instructed to apply the oil 3-4 times a day.

III. TREATMENT OF PERI IMPLANTITIS

Peri-Implantitis- Peri-implantitis is very bothering to both the dentist and the patient.Ozone gas infiltrations can also be used in this situation. Ozonated water can be used as an irrigant during debridement and curettage. Patient can be advised to apply ozonized oil on the treated area 3-4 times/day.

IV. TREATMENT OF SENSITIVE ROOTS

Desensitization of sensitive root necks - Quick and prompt relief from root sensitivity has been documented after ozone spray for 60 seconds followed by mineral wash onto the exposed dentine in a repetitive manner. This desensitization of dentine lasts for longer period of time.

OZONE TOXICITY AND IT'S MANAGEMENT

Ozone toxicity

Ozone inhalation can be toxic to the pulmonary system and other organs. Complications caused by ozone therapy are infrequent at 0.0007 per application. Known side-effects are epiphora, upper respiratory irritation, rhinitis, cough, headache, occasional nausea, vomiting, shortness of breath, blood vessel swelling, poor circulation, heart problems and at times stroke[38].

MANAGEMENT OF TOXICITY

- Patient must be placed in supine position
- Inhale humid oxygen
- Ascorbic acid
- Vitamin E
- Acetylcystein

CONCLUSION

Dentistry is changing as we are now using modern science to practice dentistry. In comparison with classical medicine modalities such as antibiotics and disinfectants, ozone therapy is quite inexpensive, predictable and conservative. There is always a chance of development of resistance against antibiotic. Pathogens on the other hand, cannot overcome oxidative challenges of ozone. In addition, there is evidence that ozone directly inactivates bacterial toxins, while antibiotics do not. Indeed, toxins are major contributors to bacterial tissue destruction[39].This state of the art technology allows us to take a minimally invasive and conservative approach to dental treatment. Treating patients with ozone therapy reduces the treatment time with a great deal of difference and it eliminates the bacterial count more precisely. The treatment is completely painless and increases the patients' acceptability and compliance with minimal adverse effects.

REFERENCES

1) Grootveld M, Baysan A, Siddiqui N, Sim J, Silwood C, Lynch E. History of clinical publications of ozone. In Lynch E, editor. Ozone: the revolution in dentistry. London Quintessence Publishing Co 2004;p. 23-30.

2) Stopka P. Ozon. Progresdent 2003;6:8-11.

3) Filippia A. The effect of ozonated water on epithelial wound healing. Douch Dent J 2001;143:96-101

4) Young SB, Setlow P. Mechanisms of killing of Bacillus subtilis spores by Decon and Oxone, two general decontaminants for biological agents. J Appl Microbiol 2004;96:289 -301.

5) Roy D, Wong PK, Engelbrecht RS, Chian ES. Mechanism of enteroviral inactivation by ozone. Appl Environ Microbiol 1981;41:718-23.

6) Burleson GR, Murray TM, Pollard M. Inactivation of viruses and bacteria by ozone, with and without sonication. Appl Microbiol 1975;29:340-4.

7) Bocci VA. Scientific and medical aspects of ozone therapy. State of the art. Arch Med Res 2006;37:425-35.

8) Mudd JB, Dawson PJ, Santrock J. Ozone does not react with human erythrocyte membrane lipids. Arch Biochem Biophys 1997;341:251-8.

9) Bocci V. The case for oxygen-ozone therapy. Br J Biomed Sci 2007;64:44-9.

10) Arsalane K, Gosset P, Vanhee D, Voisin C, Hamid Q, Tonnel AB, et al. Ozone stimulates synthesis of inflammatory cytokines by alveolar macrophages in vitro. Am J Respir Cell Mol Biol 1995;13:60-8.

11) Van Hoof HJ, Zijlstra FJ, Voss HP, Garrelds IM, Dormans JA, Van Bree L, et al. The effect of ozone exposure on the release of eicosanoids in guinea-pig BAL fluid in relation to cellular damage and inflammation. Mediators Inflamm 1997;6:355-61.

12) Bayram H, Sapsford RJ, Abdelaziz MM, Khair OA. Effect of ozone and nitrogen dioxide on the release of proinflammatory mediators from bronchial epithelial cells of nonatopic nonasthmatic subjects and atopic asthmatic patients in vitro. J Allergy Clin Immunol 2001;107:287-94.

13) Cho HY, Zhang LY, Kleeberger SR. Ozone-induced lung inflammation and hyperreactivity are mediated via tumor necrosis factor-alpha receptors. Am J Physiol Lung Cell Mol Physiol 2001;280:1537-46.

14) Reth M. Hydrogen peroxide as second messenger in lymphocyte activation. Nat Immunol 2002;3:1129-34.

15) Maresca M, Colao C, Leoncini G. Generation of hydrogen peroxide in resting and activated platelets. Cell Biochem Funct 1992;10:79-85.

16) Iuliano L, Colavita AR, Leo R, Praticò D, Violi F. Oxygen free radicals and platelet activation. Free Radic Biol Med 1997;22:999-1006.

17) Valacchi G, Bocci V. Studies on the biological effects of ozone: Release of factors from ozonated human platelets. Mediators Inflamm 1999;8:205-9.

18) Nogales CG, Ferrari PA, Kantorovich EO, Lage-Marques JL. Ozone Therapy in Medicine and Dentistry. J Contemp Dent Pract. 2008 May; (9)4:075-084

19) Priyamak AA. Ozone: The Revolution in Dentistry. Copenhagen: Quintessence Publishing; 2004. p. 155-64

20) Huth KC, Jakob FM, Saugel B, Cappello C, Paschos E, Hollweck R, et al. Effect of ozone on oral cells compared with established antimicrobials. Eur J Oral Sci 2006; 114:435-40.

21) Baysan A, Lynch E. The use of ozone in dentistry and medicine. Prim Dent Care 2005;12:47-52.

22) Nogales CG, Ferrari PA, Kantorovich EO, Lage-Marques JL. Ozone Therapy in Medicine

and Dentistry. J Contemp Dent Pract. 2008 May; (9)4:075-084.

23) Seidler V, Linetskiy I, Hubálková H, Stanková H, Smucler R, Mazánek J. Ozone and its usage in general medicine and dentistry. A review article. Prague Med Rep 2008;109:5-13.

24) Teresa B, Wolanska E, Cieszko-Buk M, Orlowski M, Chalas R. Practical use of ozone in dentistry-comments. Ann Universitalis Maria Curie Sklodowska Lubin-Polonia 2008;LXIII:28.

25) Seaverson K, Tschetter D, Kaur T. Patient guide to oxygen/ozone therapy. Health centered cosmetic dentistry. [Online]. [Last cited on 2010 January 13]. Available from: URL:http://www.toothbythelake.net/ozone_therapy.html.

26) Mollica P, Harris R. Integrating oxygen/ ozone therapy into your practice. [Online]. [Cited 2010 January 13];[4 screens]. Available from: URL:http://www. Toxin free smile. Dom/ images/ozone integrating % 20 oxygen ozone 20% therapy your practice.

27) Putnins EE, Di Giovanni D, Bhullar AS. Dental unit waterline contamination and its possible implications during periodontal surgery. J Periodontol 2001;72:393-400.

28) Wirthlin MR, Marshall GW Jr, Rowland RW. Formation and decontamination of biofilms in dental unit waterlines. J Periodontol 2003;74:1595-609.

29) Kohno S, Kawata T, Kaku M, Fuita T, Tsutsui K, Ohtani J, et al. Bactericidal effects of acidic electrolyzed water on the dental unit waterline. Jpn J Infect Dis 2004;57:52-4.

30) Walker JT, Bradshaw DJ, Fulford MR, Marsh PD. Microbiological evaluation of a range of disinfectant products to control mixedspecies biofilm contamination in a laboratory model of a dental unit water system. Appl Environ Microbiol 2003;69:3327-32.

31) Nagayoshi M, Fukuizumi T, Kitamura C, Yano J, Terashita M, Nishihara T. Efficacy of ozone on survival and permeability of oral microorganisms. Oral Microbiol Immunol 2004;19:240-6.

32) Estrela C, Estrela CR, Decurcio Dde A, Silva JA, Bammann LL. Antimicrobial potential of ozone in an ultrasonic cleaning system against Staphylococcus aureus. Braz Dent J 2006;17:134-8.

33) Sechi LA, Lezcano I, Nunez N, Espim M, Duprè I, Pinna A, et al. Antibacterial activity of ozonized sunflower oil (Oleozon). J Appl Microbiol 2001;90:279-84.

34) Huth KC, Saugel B, Jakob FM, Cappello C, Quirling M, Paschos E, et al. Effect of aqueous ozone on the NF-kappaB system. J Dent Res 2007;86:451-6.

35) . Low SP, Williams KA, Canham LT, Voelcker NH. Generation of reactive oxygen species from porous silicon microparticles in cell culture medium. J Biomed Mater Res A 2010;93:1124-31.

36) Low SP, Williams KA, Canham LT, Voelcker NH. Evaluation of mammalian cell adhesion on surface-modified porous silicon. Biomaterials 2006;27:4538-46.

37) Gupta G, Mansi B. Ozone therapy in periodontics. J Med Life 2012;5:59-67.

38) Matsumura K, Hyon SH, Nakajima N, Iwata H, Watazu A, Tsutsumi S. Surface modification of polyethylene-co-vinyl alcohol hydroxyapatite immobilization and control of periodontal ligament cells differentiation. Biomaterials. 2004;25:4817 - 4824.

39) Bocci V. Biological and clinical effects of ozone. Has ozone therapy a future in medicine? Br J Biomed Sci 1999;56:270-9.

RADIOTHERAPY AND CHEMOTHERAPY INDUCED ORAL MUCOSITIS

MARIN ABRAHAM[1], TEJAVATHI NAGARAJ[2], LEENA JAMES[3], SUCHETHA.D.N.[4]

1. Dr. Marin Abraham
Lecturer
Department of Oral
Medicine and Radiology
Sri Rajiv Gandhi College of
Dental Sciences and Hospital
Cholanagar, Hebbal
Bengaluru, Karnataka

2. Dr. Tejavathi Nagaraj
Professor and HOD,
Department of Oral
Medicine and Radiology
Sri Rajiv Gandhi College of
Dental Sciences and Hospital
Cholanagar, Hebbal
Bengaluru, Karnataka

3. Dr. Leena James
Reader
Department of Oral
Medicine and Radiology
Sri Rajiv Gandhi College of
Dental Sciences and Hospital
Cholanagar, Hebbal
Bengaluru, Karnataka

4. Dr. Suchetha.D.N
Senior Lecturer
Department of Oral
Medicine and Radiology
Sri Rajiv Gandhi College of
Dental Sciences and Hospital
Cholanagar, Hebbal
Bengaluru, Karnataka

For correspondence:
Dr. Marin Abraham
Email: marinabraham09@gmail.com

ABSTRACT
Oral mucositis or stomatitis is a spectral and an expensive adverse effect induced by chemotherapy and radiotherapy prevalent in about 40% of victims who fall prey to malignant neoplastic disease. Nevertheless, the high risk group of population to be affected with oral mucositis comprises patients affected with the head and neck cancer. With the onset of oral mucositis, the patient undergoes severe pain and distress that resists the tolerance to chemotherapy and radiotherapy, thereby progressing to a transient withdrawal of the treatment.

KEYWORDS
mucositis, head and neck cancer, radiation, chemotherapy, somatization, cell viablity

INTRODUCTION
The impact of oral mucositis is influenced by the span taken to diagnose the pathology encountered by the patient, his/her age, a good oral hygiene regime, type, dose, and frequency of drug administered to the patient. The incidence of oral mucositis is consistently higher among patients subjected to conditioning therapy for bone marrow/peripheral blood progenitor cell transplantation, continuous infusion therapy for breast and colon cancer, and therapy for tumors of the head and neck associating concomitant chemotherapy and radiotherapy.[1] Oral ulcers could be a preliminary indication of drug toxicity, that could seldom warrant reduction of drug dosage or complete cessation of drug therapy.[2,3] Patients themselves manipulate their diet by decreasing intake of food and sometimes fluids as well.

EPIDEMIOLOGY
The incidence and severity of oral mucositis do not occur with a confined specificity, but can be minimized if the subjects keep up an admirable oral health, those who abstain from smoking, and regulate their blood sugar levels.[4,5] The risk of developing mucosal injury increases with the number of chemotherapy cycles and previous episodes of chemotherapy induced oral mucositis. Damage to salivary glands reduces the flow and composition of saliva and its pH, with variation in the oral microflora, thereby increasing the risk of infection. Patients undergoing radiotherapy are at a greater jeopardy with oral mucositis by an estimate of 80% than their chemotherapy treated counterparts with a lesser incidence of 22%.[6]

PATHOPHYSIOLOGY

The oral mucositis progression is characterized by five phases; initiation that causes DNA damage in basal epithelial cells generating reactive oxygen species (ROS), which induces apoptosis and regulates inflammatory cytokines in cells called signaling. Inflammatory cytokines increases tissue damage known as amplification where the integrity of mucosa is lost, leading to portals of entry for bacteria, virus and fungi causing ulceration and finally healing by proliferation, differentiation and migration of epithelial cells.[7,8]

CLINICAL FEATURES

Mucositis is a form of mucosal barrier injury characterized by oral erythema, ulceration, and pain. Ulcers are typically covered by exudates composed of cells, serum and debris. This stage is the ulcerative/ fibrinous/ pseudomembranous mucositis **(Fig.1)**. Pseudomembrane appears white or opaque when hydrated by saliva and may appear yellowish or greenish due to superficial infection when associated with deep ulcers. A definite observation is mandatory, to secern from candidiasis or viral infection.[8,9]

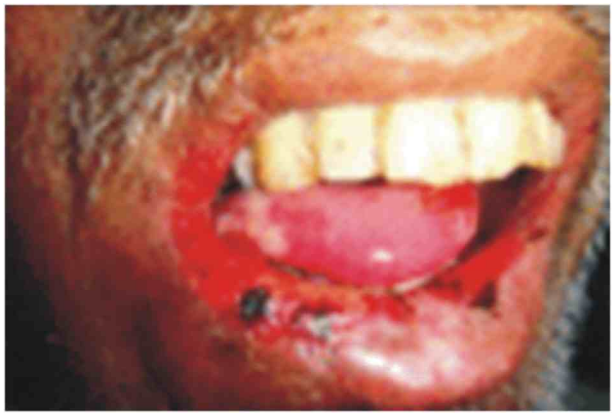


Fig.1: Oral mucositis in a patient who has undergone radiotherapy

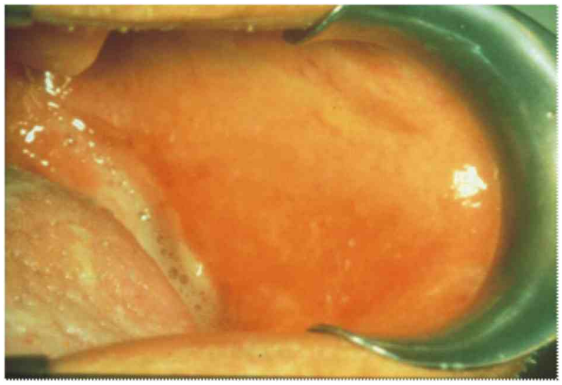


Fig.2: Grade I oral mucositis

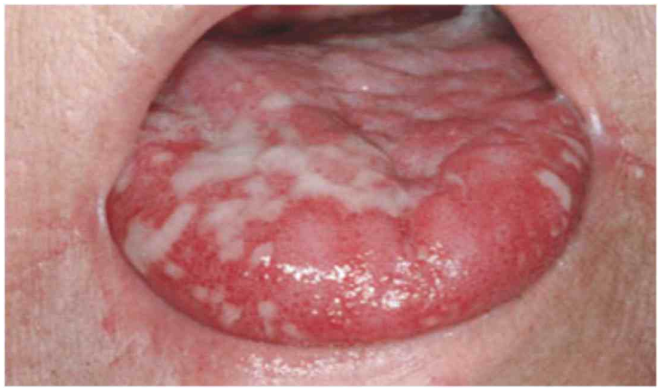


Fig.3: Grade II oral mucositis

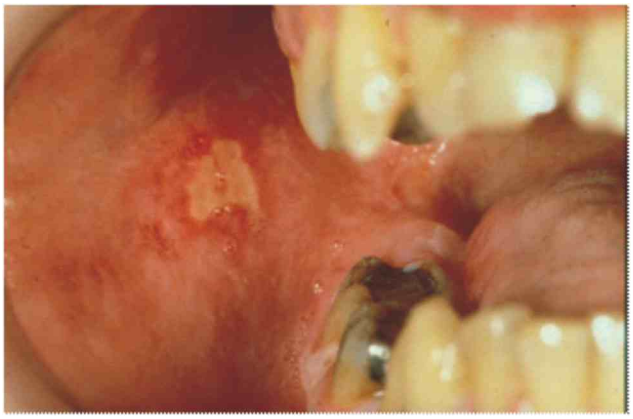


Fig.4: Grade III oral mucositis

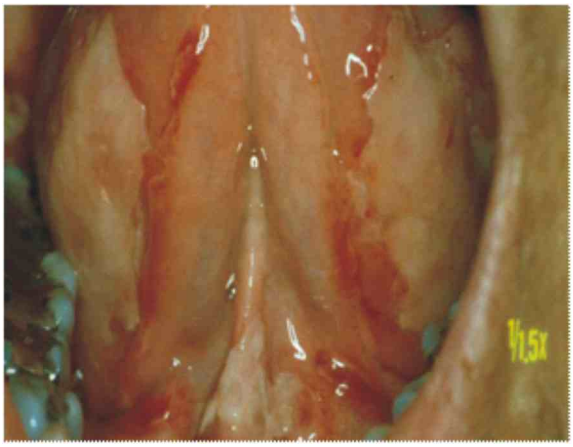
Radiation induced clinical features of oral mucositis:

The clinical signs of grade-1 radiation-induced mucositis commences at the end of the first week of a conventional seven-week radiation protocol (daily dose of 1.8 to 2.0 Gy, five times a week) **(Fig.2).** A white discoloration of the oral mucosa that characterized by hyperkeratinisation of the epithelium, followed by erythema, is observed or viceversae and are mostly asymptomatic. By the end of the second or around the third week of treatment, small foci of ulceration can be appreciated, corresponding to the grade II mucositis **(Fig.3)**. Patient complains of mild pain and can take soft diet. Severe, grade III mucositis presents as ulcers covered by pseudomembranes, extending to larger areas of the oral mucosa, that are extremely painful to rub off, and dysphagia **(Fig.4)**. In Grade IV mucositis, the patient encounters even more severe ulceration, covering almost all mucosal surfaces which mark the severity of the condition. The severely affected regions are the soft palate, the

mucosa of the hypopharynx, floor of the mouth, cheek, base of the tongue, lips, and dorsum of the tongue that persists throughout radiotherapy, and develops at its maximum grade at the cessation of radiotherapy that regresses by the third week following the procedure. The ulcerations can get aggravated with secondary infections like pseudomemberanous candidiasis and herpes virus-1 (HSV-1) reactivation.[9]

Clinical features of chemotherapy induced oral mucositis

Chemotherapy-induced oral mucositis commences with diffuse leukoedema. Oral mucositis begins 5-10 days following the initiation of chemotherapy and lasts 7-14 days; the entire sequalae lasts 2-3 weeks. Oral mucositis marks its onset with areas of erythema and atrophy of the mucosa that then breaks down to form ulcers that are covered by a yellowish-white fibrin clot (pseudomembrane) with an existing peripheral erythema. Ulcers may range from 0.5 cm to greater than 4 cm in maximum dimension. At the peak of its intensity, the patient experiences extreme pain and difficulty to open the mouth. With the administration of chemotherapy, the oral mucosa becomes atrophic, with an altered rejuvenation, local trauma leads to ulceration; the nonkeratinized sites become the most vulnerable. Therefore, lesions occur bilaterally, mainly the buccal mucosa, the ventral and lateral parts of the tongue, the labial mucosa, the floor of the mouth, the soft palate and oro pharyngeal fauces **(Fig.5)**.[10]



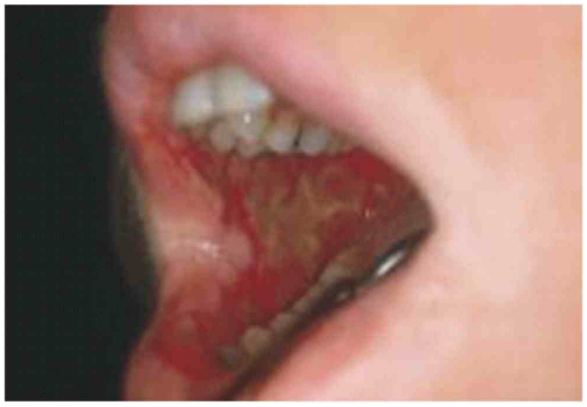


Fig.5: Bilateral ulcerations of the oral mucosa induced by chemotherapy.

TREATMENT MODALITIES

An intensive oral hygiene protocol, treatment of carious teeth and maintaining proper oral hygiene reduces the virulence of oral microflora. Pain is the most unpleasant situation encountered in oral mucositis that affects the nutritional intake, mouth care and quality of life.[11] Non opioid strategy: It comprises the use of variety of adjuvant treatment that can be used simply or combined with opioids to improve the efficacy of pain management. The agents considered are cyclo-oxygenase-2 inhibitor, nonsteroidal anti-inflammatory drugs, gabapentin, cannabinoids, alpha-2 adrenergic receptor agonists consisting of clonidine, nicotine, lidocaine and ketamine.[12-18]

i) Cryotherapy: The patients rub gently ice chips in the mouth for about thirty minutes that commences five minutes prior to each dose of drug; primarily recommended for patients undergoing bolus fluorouracil administration.

ii) Lasers: low level laser therapy reduces levels of reactive oxygen and pro inflammatory cytokines, that contributes to the pathogenesis of mucositis. Laser treatment is performed one day before chemotherapy and once weekly, till the resolution of mucositis.

iii) L-glutamine: an aminoacid required for cell respiration in rapidly dividing cells; Saforis being its proprietary suspension. Its mode of action is by reducing the production of cytokines and related apoptosis and promotes

healing by increasing fibroblast and collagen synthesis.

iv) Gelclair: It is a concentrated gel and Zilactin is a protective film that forms a protective barrier when applied to the traumatized oral mucosa. It is economically favourable and simple for patients to prepare comprising a teaspoon of salt and a teaspoon of baking soda in quarter litre of water.

v) Sucralfate: a basic aluminium salt of sucrose sulfate that forms a paste like protective coat on the ulcerated mucosa by forming iconic bonds to proteins and promotes local production of prostaglandins, a cycloprotectant that stimulates epithelial proliferation, migration blood flow and mucous protection.[13]

vi) Capsacin candies have been encouraged as an alternative analgesic for chemotherapy induced mucositis.

vii) Silver nitrate administration a few days prior to radiotherapy reduces the severity of mucositis by stimulating the un-irradiated mucosa, making it more proliferative.

Mouth washes:

The use of saline mouth wash, topical mouth rinse with anesthetic-2% viscous lidocaine with diphenhydramine provides a short term relief; namely "Magic mouth wash" that comprises of lidocaine, diphenhydramine, magnesium aluminium hydroxide, and morphine mouth wash that contributes to the reduction of the adverse effects, Leucovorin (5-formyltetrahydrofolate) to protect the normal tissues from the toxic effect of high dose methothextrate, a folic acid antagonist.[14]

Anti inflammatory agents:

Benzydamine hydrochloride is a drug with anti-inflammatory, anesthetic, analgesic, antipyretic antimicrobial activities, has been used as a mouthwash to prevent and treat oral mucositis. It has proved to be effective in patients with radiation induced oral mucositis.

RKO_2O_2: is an antioxidant, N-acetyl cystiene in proprietary matrix, a topical applicant reduces the severity of oral mucositis to doses of 50gy radiation therapy.

Mesalazine: (5-aminosalicylic acid) is an anti-inflammatory agent which inhibits activation of one transcription factor involved in transcription of genes that shows some effects when treated for radiation and chemotherapy induced oral mucositis.

EN_{3247}: (triclosan0.1) has broad spectrum antimicrobial activity; at bacteriostatic concentrations inhibits uptake of essential aminoacids and causes disorganization of cytoplasmic membrane. Patients acquiring treatment for oral mucositis experiences a lesser severity of radio and chemotherapy induced oral mucositis.

Chamomile has anti-inflammatory, spasmolytic effects, promotes granulation and mucosal healing. The prophylactic mouthwashes delayed the onset and reduced the severity of radiation mucositis.[15]

Glucocorticoids are anti-inflammatory drugs, the use of high dose of methasone mouthwash before and during radiotherapy showed progressive whitening as radiation progressed, in contrast to erythema and maintained the mucosa virtually ulcer free .

Prostalandin E2, a derivative of misoprostol has anti inflammatory properties that produced favourable results in cases of radiation induced oral mucositis.

Nutrition supplement: Consumption of soft diet and liquid diet, gastrostomy is beneficial in case of severe mucositis.

PTA lozenges,pastillies and povidone iodine: selective decontamination by using lozenges containing polymixin E (P), tobramycin (T) and amphotericin B (A), together provide broad spectrum antibacterial and antifungal cover. These are commonly known as PTA lozenges or PTA pastilles. Povidone iodine has a wide antiseptic effect including antiviral, antibacterial and antifungal efficacy and good tolerability in

radiotherapy and chemotherapy induced mucositis.
Palliation of dry mouth: Can be done by sipping water regularly, to reduce drying of mouth and use of artificial saliva, chewing sugarless gum, use of cholinergic agents.

Oral hygiene maintenence: Oral mucositis can be reduced by proper oral hygiene methods; it reduces the oral toxicity induced by radiotherapy.
Oral debridement: with mucolytic agents such as alkalol to dislodge dry secretions.

Retinoids and beta carotenes: retinoids are known to stimulate wound healing and to reduce oral complications by inhibiting inflammation. It induces epithelial growth and enhances mucosal resistance to cycle-specific toxicities. Supplemental dietary beta-carotene has shown to decrease the severity of chemotherapy and radiotherapy induced mucositis.

Immunoglobulin IgG: Administration of intramuscularly reduces the severity and duration of radiotherapy.
Radiation Shields: fabricated to protect the oral tissues during radiation. By introducing protective stents and sparing blocks, irradiation of uninvolved mucosa can be prevented. Use of CT based target delineation, intensity modulated radiation therapy, custom made intra oral devices, electron beam 3D conformal multibeam, wedged pair can minimize the radiation dose to healthy mucosa.

CONCLUSION

Oral mucositis is a frequent encounter in patients undergoing chemotherapy and radiotherapy for the treatment of various forms of cancer. It is essential to regularly assess any pathology within the oral cavity to minimize acute and chronic oral and systemic sequelae of anti-neoplastic and radiation therapy. Antidepressants would aide to overcome depression and reducing pain somatization in psychologically affected patients. The role played by oral mucositis induced secondary to chemotherapy or radiotherapy though not life-threatening; interferes considerably with the outcome of cancer treatment.

REFERENCES

1. http://emedicine.medscape.com/chemotherapy induced oral mucositis treatment and management/1079570-overview.
2. Varisto LE, Volpato R, Silva TC, Oliveira TM, Sakai VM, Aparecida M et al. Radiation therapy and chemotherapy induced oral mucositis. Rev Bras De Otorinolaring 2007;73:562-68.
3. Lynch MA, Bright VJ, Greenberg MS. Burkit's oral medicine diagnosis and treatment. 9th ed. Philadelphia (PA): Lippincott raven publishers; 1998.
4. Rao MRU, Naidu MU, Ramana GV, Rani PU, Mohan IK, Suman A et al. Chemotherapy induced/radiation induced oral mucositis-complicating treatment of cancer. Neopl 2004;10:10.
5. Sadashivan R. Chemotherapy induced oral mucositis. US Onc Rev 2010;6;13-16.
6. Oral mucositis.om/920.
7. Roopashri G, Jayanthi K. Radiotherapy and chemotherapy induced oral mucositis prevention and current therapeutic modalities. IJDA 2010;2:12.
8. Kumar PSS, Balan A, Shankar A. Radiation induced oral mucositis. Ind J of Palliat Care PMCID2902123 2009;15:95-102.
9. Pico JL, Avila A, Gravita, Nacchache P. Oral mucositis occurance, consequence and treatment. The Onclgst 1998;3:446-51.
10. Harris DJ. Cancer treatment induced Mucositis Pain : Strategies for Assessment and Management. Ther Clin Risk Manag. 2006 September; 2(3): 251–258.
11. Krishnatry R, Nachankar AA, Gupta T, Agarwal JP. Oral mucositis short review. IJHNS 2011;2:37-43.
12. Turhal NS, Erdal S, Karacay S. Efficacy of treatment to relieve mucositis induced discomfort. Supp Care Canc 2000;8:55-58.

13. Szabo S, Hollander, Scarbrough E, Folkman J. Role of vascular factors in mode of action of sucralfate. AJM 1991;91:158-60.

14. Polting CM, Uitterhoeve R, Reimer WS, Achtenberg T. The effectiveness of commonly used mouthwashes for prevention of chemotherapy induced oral mucositis. Eur J Cancr Care 2006;15:431-39.

15. Filder P, Loprinizi CL. Prospective evaluation of chamomile for prevention of 5FU induced mucositis. Canc1996;77:52-55.

16. Hoffman HT . Oral mucositis, a challenging complication of radio-chemotherapy. Wiley Periodic Inc 2004; 26: 77-84.

17. Biron P, Sebban C, Gourmet R, Chvetz G, Philip I, Blay JY et al. Research controversies in management of oral mucositis. Supp Care Canc 2000;8:68-71.

18. Epstein JB, Schubert MM. Managing pain in mucositis. Sem in Oncol Nurs 2004;20:30-37

SHORTENED DENTAL ARCH: REVISITED

GOUTHAM G.B[1], KARTHIK K[2], SANAM BASHEER[3],

1. Dr. Goutham G.B
Postgraduate student
Dept. of Prosthodontics
Sri Rajiv Gandhi College of
Dental Sciences and Hospital
Cholanagar, Hebbal
Bengaluru, Karnataka

2. Dr. Karthik K
Postgraduate student
Dept. of Prosthodontics
Sri Rajiv Gandhi College of
Dental Sciences and Hospital
Cholanagar, Hebbal
Bengaluru, Karnataka

3. Dr. Sanam Basheer
Lecturer
Dept. of Prosthodontics
Sri Rajiv Gandhi College of
Dental Sciences and Hospital
Cholanagar, Hebbal
Bengaluru, Karnataka

For correspondence:
Dr. Goutham G.B
Email: gouthamgb1@gmail.com

ABSTRACT
With advent of new ideas and care towards oral hygiene, it seems that a new concept like shortened dental arch (SDA) is one of the best treatment modalities wherein treatment of lost dentition is carried out minimally so that the oral function is neither compromised nor the craniomandibular discomfort is experienced by patients. The SDA concept approach appears to fit well with problem solving approach in modern dentistry. This article reviews advantages, disadvantages and views of the clinicians on shortened dental arch.

KEYWORDS
partial denture, shortened dental arch, treatment option

INTRODUCTION
To provide care for the partially dentate or edentulous patient, the dental surgeon has to consider number of factors such as oral functionality, vertical dimension, occlusion, maintenance of hard tissue, temporomandibularjoint (TMJ) health as well as patient comfort. Dentists replace severely decayed, missing and damaged teeth by prosthesis which is fixed or removable to restore or improve masticatory function[1,2]. SDA can be defined as "a specific type of dentition with intact anterior region and a reduction in the occluding pairs of posterior teeth, starting posteriorly"[3].This was first described by Dutch Prosthodontist Arnd Kayser and co-workers at the dental school of university of Nijmegen, Netherlands, in 1981[4]. According to SDA concept, all treatment efforts concentrated on preserving sound anterior and premolar teeth and avoiding extensive restorative treatment in the molar region[5].

BASIS OF SDA CONCEPT
Prosthodontists often approach the treatment of the SDA as a reconstructive or rehabilitative problem. These terms are introduced to differentiate between rehabilitation procedure aimed at restoring dental and temporomandibular joint function and reconstruction that implies more anatomically oriented restoration of tooth surfaces. The decision to restore all the teeth in the mouth should be made only after all intended advantages of such treatment are carefully considered and are found to out weight the benefits of a more limited yet therapeutically sound alternative. The decision based on the aim to create and maintain a healthy occlusion as described by Ash and Ramfjord, implies an

absence of pathology not only on the dentition but also periodontal osseous and muscular components of the oral system. An orthopedically stable centric position, axial loading of dentition and guidance from centric to eccentric positions are essential. The last requirement is adaptability of the system to changes in other components in the system. The shortened dental arch includes thorough examination of the dentition within the functional needs of the stomatognathic systems and the requirements of the dentition to maintain a pathology free system[6].

The World Health Organization (WHO) indicates that a functional, esthetic, natural dentition has at least 20 teeth, while the literature indicates that dental arches comprising the anterior and premolar regions meet the requirements of a functional dentition[1].

WHO goal: 1992

"Oral health by retention of healthy, natural functioning dentition comprising not less than 20 teeth and not acquiring prosthesis"[7].

Classification of SDA

Kayser suggested the following classification for SDA according to the number of teeth remaining in the arch and symmetry of shortening is as follows[4].

Symmetrically shortened dental arch

Extremely shortened dental arch with asymmetry

A system which considers occlusal units as premolar equivalents was also developed in which a molar is equivalent to two premolar units and a premolar equivalent to a single occlusal unit.

Rationale and considerations of SDA:

Following are the rationale and consideration of SDA[3]

Anterior and premolar regions are functionally and esthetically strategic parts of dentition, and are considered a priority in rehabilitation.

It is based on circumstantial evidence.

It does not contradict current theories of occlusion.

It fits well with a problem solving approach.

It meets the requirement of oral function.

Molars are high risk teeth for caries and periodontal diseases.

It reduces the need for complex restorative treatment in the posterior region.

Advantages of SDA concept:

Following are the advantages of SDA[4]

1. Restoration of a SDA meets the characteristics of current theories of an acceptable occlusion by terminating occlusal platform at the second premolar region.
2. It also provides a high standard of care and minimal cost by avoiding restorative treatment for the posterior regions of mouth.
3. Financially, the SDA concept is acceptable to the patient as molars are more prone to be lost by both caries and periodontal diseases and considered most costly teeth to preserve.
4. The oral comfort was compared with patients for SDA, distal extension RPD and subjects with complete dental arches. No significant finding between three groups with respect to pain or distress.
5. According to Armellini and Vonfraunhofer SDA may be beneficial for immunosuppressed patients and those undergoing radiotherapy or chemotherapy.

Disadvantages of shortened dental arches

Following are the disadvantages of SDA[4]

1. Many people with SDA found that their chewing ability hindered or they had to change food preparation practices.
2. Some patients with SDA reported the prevalence of temporomandibular joint problems.
3. A study performed by Witter et al. showed that there is greater prevalence of joint sounds with subjects having only unilateral posterior support and those with no posterior support.
4. SDA may be associated with greater tooth migration and interdental spacing, although migration was deemed small and clinically insignificant.
5. An SDA may also be associated with greater

over eruption of teeth.

6. People with SDA have been found to have more mobile teeth and lower alveolar bone levels.

Following are views of various authors on SDA Concept[7]:

Kreulen and Witter et al. concluded instability was not seen in SDA as long as three to four occluding units existed, but the signs worsened "Extremely shortened dental arches" and age was associated with changes in occlusal integrity. Witter et al. found that interdental spacings occurred following extractions leading to SDA due to reduction of anterior component of occlusal force because of the absence of molar teeth and not due to overload in anterior region. Watanabe et al. reported that during maximum voluntary clenching, TMJ loads were less in SDA compared to complete dentitions because of decreased muscular force in SDA.

According to Kreulen et al. absence of complete posterior occlusal support either unilaterally or bilaterally may aggravate signs of temporomandibular disorders. Witter et al. found that periodontal tissues show favourable response to SDA except in cases of uncontrolled periodontal diseases. According to the study by Hittori et al. neuromuscular regulatory systems control maximum clenching strength under various occlusal conditions and no evidence was seen that SDA caused overloading of teeth and joints.

Prosthodontic Considerations of SDA:

Prosthodontic considerations in treatment of patients include occlusal stability, establishing correct vertical dimension, preserving the health of soft tissue, hard tissue and TMJ. Occlusal stability can be defined as the absence of the tendency for teeth to migrate other than the normal physiologic compensatory movements occurring over time[8]. Occlusal stability is determined by a number of factors, including periodontal support, the number of teeth in the dental arches, interdental spacing, occlusal contacts and tooth wear. Distal tooth migration occurs in shortened dental arches and result in an anterior load which, in turn increases the number and intensity of anterior occlusal contacts as well as interdental spacing. Such effects may be aggravated when unopposed teeth and lone standing teeth have inadequate periodontal support. Likewise tooth migration can cause changes in the vertical and horizontal overlap, occlusal wear, and loss of posterior support, among other effects. SDA comprising anterior and premolar teeth satisfy oral functional demands and show similar vertical overlap and occlusal tooth wear patterns to those found with complete dental arches.

Few studies are reported on the prevalence of TMJ problems in adults with SDA. A study by Sarita PT compared SDA group comprising of 725 subjects with an intact anterior region and 0-8 posterior occluding pairs of teeth with a control group of 125 subjects having complete dental arches. Study reported a greater prevalence of joint sounds with subjects having unilateral posterior support. However there was no difference in pain, mandibular mobility, maximum mouth opening, or clicking of the joints for the SDA and control groups. It was noted, however the tooth wear increased significantly with decreased posterior support while there was no evidence that SDA provokes TMJ problems. It was noted that the risk of pain and joint sounds increased when unilateral or bilateral posterior support was missing[9].

Another study addressed question of whether SDA could cause functional over loading of the teeth and TMJ effects possibly leading to periodontal disease and temporomandibular disorder.

Electromyographic masticatory muscle studies were used to calculate occlusal forces and joint loads using a finite element jaw model and compared these values with actual measured occlusal forces. While the occlusal force on each tooth increased with missing molar occlusion, there appeared to be an overall decrease in joint loads, although occlusal force per root surface area was always greatest on the most posterior tooth. There were no indications that a SDA can cause overloading of the TMJ or teeth, suggesting that neuromuscular regulatory system are efficient in controlling the maximum clenching force under various occlusal conditions[1].

Attitudes of dental professionals towards the SDA concept:

According to Kanno et al. in surveys conducted in Tanzania, Netherlands and European countries, SDA had a role in contemporary clinical practice where elderly and economically poor patients were involved and also in cases where possibilities of restorative care were complicated[10].

CONCLUSION

The dental arches consisting of anterior and premolar regions meet the demands of functional dentition. However, functional demands and number of teeth to satisfy that demand vary with individual to individual and consequently dental treatment must be planned to each individual needs and adaptive ability. The SDA concept does not contradict current occlusal theories and appear to fit well with the problem solving approach. By offering partially dentate patient a treatment option that ensure improved oral hygiene, oral functionality, comfort and possibly reduced cost.

REFERENCES

1. Armellini D. The Shortened dental arch: A Review of the literature. J Prosthet Dent 2004; 92:531-4.
2. Rodriguez AM, Aquilino SA, Lund PS. Cantilever and implant biomechanics: a review of the literature, part 2. J Prosthodont 1994; 3: 114-8.
3. Pradeep KC, George S. An assessment of prosthodontists attitude to the shortened dental arch concept. J Interdiscip Dentistry 2012; 2:104-7.
4. Bansal M, Singh S. Concept of shortened dental arch an overview. Indian journal of multidisciplinary dentistry 2012; 2:560-2.
5. Nassani MK, Tarakji B. Reappraisal of the removable partial denture as a treatment option for the shortened dental arch. Eur J Dent 2013; 7:251-6.
6. Frias V, Toothaker R, Wright R Shortened Dental Arch: A Review of current treatment concepts. J Prosthodont 2004:104-9.
7. Chetan Hegde. Dental arch A new weapon in the arsenal of Prosthodontic rehabilitation. Nitte University, Deralakatte: Mangalore. www.guident.net/prosthodontics/shortened-dental arch a new weapon in the arsenal of prosthodontic rehabilitation.html.
8. Sarita PT. A study on occlusal stability in shortened dental arches. Int J Prosthodont 2003; 16:375-80.
9. Sarita PT. Signs and symptoms associated with TMD in adults with shortened dental arches. Int J Prosthodont 2003; 16:265-70.
10. Kanno T. A review of the shortened dental arch concept focusing on the work by the Kaiser/Nijmen group. J Oral Rehabil 2006; 33:850-62.