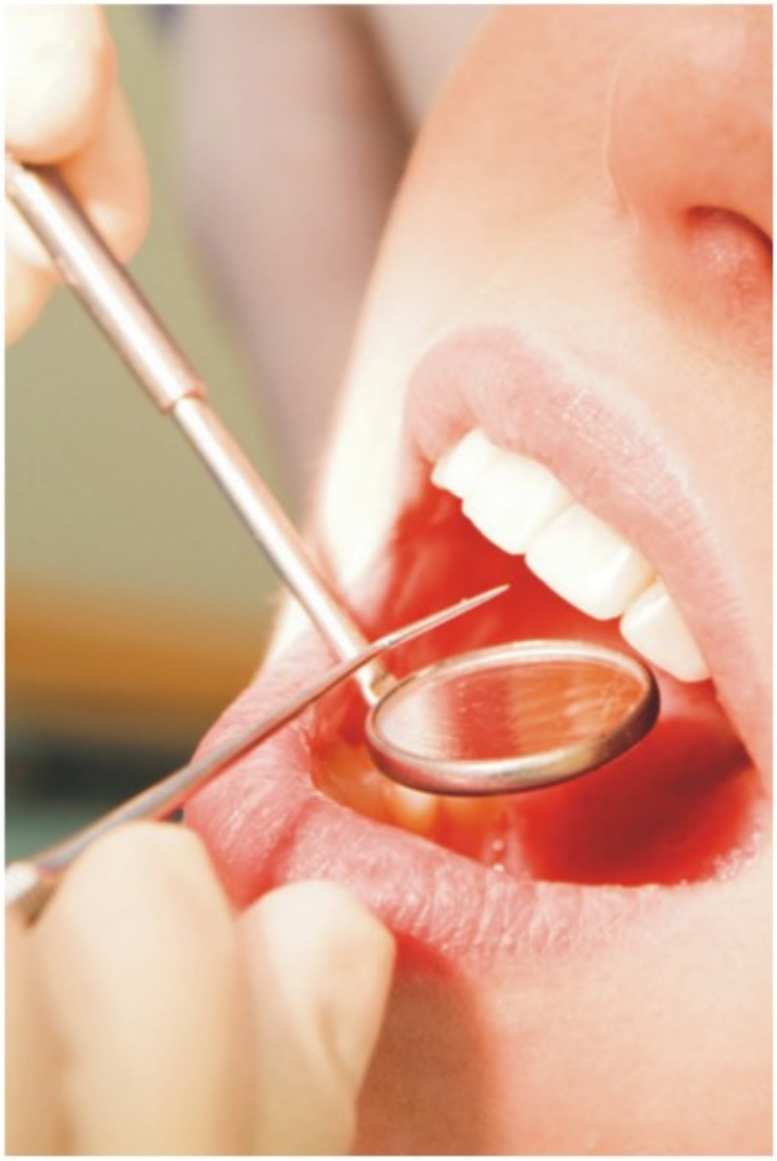

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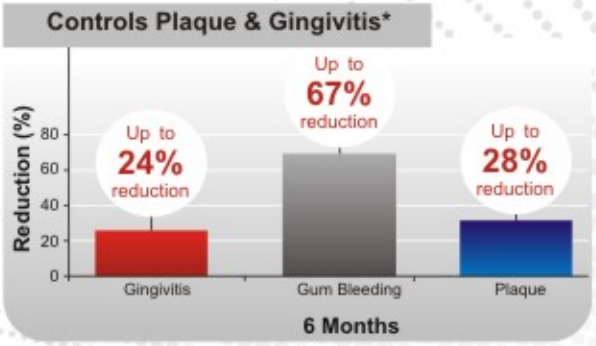

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Original Article

Smoking- A Risk Factor For Periodontal Disease In Adult Males of Mumbai

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Abstract

The present study was undertaken to evaluate smoking as a risk factor for periodontal disease in Mumbai in a large group of patients in Mumbai. A total of 457 male patients between the age group of 20-55 years with the habit of smoking either cigarette or bidi participated in this study. The patients were categorised as: age group(20-25yrs,26-35yrs,36-45yrs &46-55yrs),frequency of smoking(>5units/day,6-10units/day,11-20units/day & ,20units/day).Duration of smoking (>2yrs, 3-5yrs, 6-10yrs & <10yrs).Periodontal status was assessed by community periodontal index. Results showed increase in gingival inflammation, calculus formation among younger age groups (20-35yrs) and moderate pocket formation between 36-55yrs of age. Calculus formation amongst 46-55yrs was 42% where as bleeding in age group of20-35yrs was 31%.The duration of smoking had significant influence on periodontal health since smoking for more than 10yrs being more liable for presence of pockets(almost twice)than smokers for >5yrs.

Key words
Smoking, Risk Factors, Periodontal Diseases

INTRODUCTION

The association between tobacco and its products represents a significant public health problem. Most epidemiological studies indicate that smoking is directly related to incidence & prevalence of a variety of medical problems including pulmonary, cardiovascular, gastrointestinal disease, low birth weight and cancer. Numerous studies have also demonstrated role of tobacco use on the incidence, prevalence, severity of pocket depth, attachment and alveolar bone loss. Smoking plays a significant role in the development of refractory periodontitis. Smokers have also reported to have poorer success rate with scaling and curettage. Smokers have increased levels of salivary antibodies (IgA) and serum IgG antibodies to P intermedia and F Nucleatum. In addition smokers appear to have depressed number of helper lymphocytes which are important components of the immune systems.

In order for the host to efficiently deal with bacterial infection, fully functioned neutrophils are required. Tobacco smoke can have deleterious effects on neutrophil functions for e.g. it can impair chemotaxis and phagocytosis. The nicotine in tobacco smokers is only one of over 2000 potentially toxic substances. Besides this there are many more components in tobacco smoke which could directly damage the normal cells of the periodontium. These deleterious effects can greatly affect the reparative and regenerative potential of the periodontium in tobacco users.

Another aspect of tobacco and its related products relates to their potential abilities to reduce gingival blood flows besides the direct damage to the periodontal tissues.

MATERIALS AND METHOD

20-55 years males from Mumbai city with the habit of cigarette/bidi smoking participated in this study. For assessment of tobacco disease and periodontal disease the sample were categorised into following groups: Age groups: 20-25yrs,26-35yrs,36-45yrs and 46-55yrs Frequency : average<5units/day,6-10units/day,11-20units/day ,>20units/day. Duration of smoking: <2yrs, 3-5yrs, 6-10yrs and >10yrs.

Individuals currently smoking at least 1 cigarette/bidi were considered as smokers. Individuals smoking one cigarette/bidi in 3 days were considered as occasional smokers. Smoking category included use of either cigarette or bidi or both. Both the forms are most popular in Mumbai reason being cost effective and easy availability. To eliminate bias 26 individuals with systemic problems like diabetes mellitus and heart disease were eliminated.47 individuals who were in a habit of both smokeless form of tobacco consumption and smoking were excluded.

Intraoral examination was conducted by a single examiner, using mouth mirror and WHO probe (CPI).The following scores on 10 index teeth recommended by WHO were recorded:

0- Healthy
1- Bleeding
2- Calculus
3- Pocket of 4-5mm
4- Pocket of 6mm

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RESULTS

Cigarette/Bidi smoking had a significant impact on rate of plaque accumulation and hence the severity of periodontal disease. The risk of periodontal disease increases as the duration and frequency of smoking increases.

Table 1-Background and general sample characteristics

		Frequency	Percentage (%)
Age	20-25	154	34.3
	26-35	125	27.3
	36-45	121	26.4
	46-55	57	12.4
	Healthy	12	2.6
	Bleeding on probing	202	44.2
Periodontal	Presence of calculus	165	36.1
Status	Pockets 4-5mm	64	14
	Pockets 6mmor more than 6	14	3.0
	Non smokers	52	11.3
Tobacco Usage	Occasional Smokers	58	12.6
	Regular Smokers	347	75.9
	<2 yrs	72	15.7
Duration of	3-5 yrs	193	35.6
smoking	6-10yrs	153	33.4
	>10yrs	39	8.5
	1-5 units per day	82	17.9
Frequency of	6-10 units per day	129	28.2
smoking	11-20 units per day	143	31.2
	21 or more units per day	103	22.5

Table 2- Sample distribution based on frequency of tobacco consumption in accordance with CPI scores in different age groups

Age	Healthy		Bleeding		Calculus		Pockets 4-5mm		Pocket >5mm		Total	
	Mod	Heavy	Mod	Heavy	Mod	Heavy	Mod	Heavy	Mod	Heavy	Mod	Heavy
20-25	0	20	8	50	2	38	0	18	0	0	10	126
26-35	0	0	13	25	18	34	0	29	0	0	31	88
36-45	0	0	9	26	16	22	2	32	0	0	27	80
46-55	0	0	9	30	5	12	0	35	0	4	14	81
Total	0	20	39	131	41	106	2	114	0	4	82	375

Table 1 illustrates the general characteristics of the sample. The youngest and the oldest age groups constituted for a major and minor proportion of the final sample size with 34.3% and 12.4% respectively. There were few subjects (8.5%) who were smoking since 20 years.

Table 2 demonstrates sample distribution based on frequency of smoking in accordance with CPI scores in different age groups

DISCUSSION:

Majority of population studies undertaken in India the sample belongs to low socio economic level. These patients have poor or no access to formal oral health care services. Ironically 78% of the individuals mentioned that they had never been to a dentist. This study also revealed poor oral hygiene habits in the subjects, since most of them restricted to finger with tooth powder and
neem sticks to clean their teeth. The association between the age of the subjects and their periodontal status was evident. Studies have demonstrated a relationship between the prevalence of moderate to severe periodontal disease and the number of cigarettes smoked per day, and the number of years that the patient has smoked. In the present study also long time and heavy smokers were more liable for presenting periodontal pockets.

There is a significant impact of smoking on the severity of periodontal diseases. Also the risk of periodontal pockets increased as the duration and frequency of smoking.

CONCLUSION

The profession of dentistry makes us deal with people addicted to tobacco every day. Different people have different reasons for being enslaved to this habit. Illiterate workers generally are habituated to gutka or paan whereas highly educated corporate workers use smoking as stress buster or fashion symbol. Setting up a clinical practice where some time is allotted for the tobacco deaddiction counselling can not only have a positive impact on society but also aid in maintaining a strong patient dentist relationship.

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Original Article

PRINCIPLES OF MANAGEMENT OF CALCIFIED CANALS

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Abstract

The future of teeth with calcified canals appears much brighter today than ever before due to the advancements in diagnostic aids and instrumentation techniques Use of liquid EDTA may aid in locating the orifice. Always advance instruments slowly in calcified canals When a fine instrument has reached the approximate canal length, do not remove it; rather, obtain a radiograph to ascertain the position of the file. Use ultrasonic instruments in the pulp chamber to loosen debris in the canal orifices. The use of newer nickel titanium rotary orifice – penetrating instruments should be considered.

Key words

Calcifications, EDTA, Sclerosis, Reparative Dentin, Calcificmetamorphosis, Atrophy

INTRODUCTION

Uncontrolled mineralization due to failure of enzyme, pyrophosphatase, reduced capillary permeability and reduced blood supply leads to calcifications.

.Root canal in teeth in which calcific deposits have blocked access to the canal (s), treatment efforts are often thwarted.1 An effort to locate the residual canal may remove large amounts of dentin and there is a risk of perforating or fracturing the root. 1

CALCIFICATION OF THE PULP CAN OCCUR DUE TO

1. Mineralization in response to various irritants
2. Aging.

TUBULAR / DENTINAL SCLEROSIS

Milder or moderately irritating agents produce tubular sclerosis. It is a condition of the primary dentinal tubules where they are ultimately occluded.2 Sclerotic dentin, on the other hand, may be considered a defense mechanism of the pulp dentinal complex because its formation alters the permeability of the tubules, blocking the access of irritants to the pulp.2,3 Certain drugs such as calcium hydroxide and corticosteroids, when placed on the dentin after cavity preparation have also been demonstrated by studies to cause sclerosis. Apparently, some remineralisation also occurs when sedative dressings, such as zinc oxide eugenol, are placed in carious cavities.2

SECONDARY DENTIN

Continuous deposition of the dentin, which tends to reduce the volume of the pulp takes place throughout life. Schour has claimed that 4 microns of secondary dentin is deposited daily. The deposition of dentin is slow and gradual but does increase after the age of 35-40 years. There is no firm evidence that systemic conditions such as arteriosclerosis leads to a higher incidence in pulpal calcifications.4In molars, deposition is seen greatest on the pulpal floor, to a lesser extent on the roof, and least on the side- walls. Therefore, with age, pulp chambers decrease significantly in height i.e. in occlusoradicular direction but not extensively in width i.e. in mesiodistal direction.5,2 As a result, the horns of the pulp in molars are often left behind. They also recede, but not as much as the rest of the pulp tissue.2 In upper anterior teeth, the greatest dentin deposition occurs on the lingual wall of the pulp chambers, as a result of masticating forces with subsequent deposition in the incisal tip and floor of the pulp chamber.

REPARATIVE DENTIN

Localized secondary dentin laid down in areas of focal injury in response to various irritants is more haphazardly organized and is termed as reparative secondary (irregular, irritation or tertiary) dentin.6 In humans, the average rate of reparative dentin formation has been reported to be 2.8 microns for deciduous teeth and 1.5 microns for permanent teeth. Operations on the dentin, with resultant damage to the involved odontoblasts, cause temporary derangement in mineralization shown by the formation of a basophilic line. This has been termed as a calciotraumatic reaction. The presence of abundant amounts of reparative dentin does not appear to be correlated with pulp- test readings.2

RETROGRESSIVE PULP CHANGES

These altered pulp conditions are

1. Atrophy and fibrosis
2. Dystrophic calcification (calcific metamorphosis)6

P.E. Lovdahl and J.L. Gutmann have summarized pulpal responses to irritation as follows7:

1. Rapid death with canal patency.
2. Irritational response with pulpal demise
3. Extensive irritational response and pulp system

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exposure

CALCIFICATIONS OF PULP

. The classification by Kronfield is most commonly used.4
1.Discrete pulp stones (denticles, pulp nodules)
2.Diffuse calcification.
According to Seltzer, denticles may be classified as
a. According to structure – true or false.
The difference between the two is morphologic, not chemical
b. According to size -fine, diffuse mineralizations (also called fibrillar mineralization) and denticles.
c. According to location – embedded or interstitial, adherent and free denticles.

CALCIFIC METAMORPHOSIS

Calcific metamorphosis is defined as a pulpal response to trauma that is characterized by deposition of hard tissue within the root canal space and is commonly found in young adults in the anterior region of the mouth.8

TREATMENT PLANNING

It should be monitored radiographically and treated only if an area of rarefaction or clinical symptoms develop. 9

- Radiographs -Reducing the kVp and increasing the milliamperage accordingly increases the contrast and may make film interpretation easier.
- Multiple preoperative views may help the clinician locate root canals or establish the presence of additional root or canals.
- The use of buccal object rule and radiographic markers such as cotton pellet- stabilized burs/ segments of foil/ gutta percha can help determine the bucco- lingual orientation in posterior teeth. However, to help determine the labio- lingual position during the non- surgical treatment of calcified canals in anterior teeth one is usually forced to rely more on the visual aspect.
- With magnification, a world of remarkable lucid detail within the tooth becomes available, facilitating considerably more accurate,thus more conservative ,penetration through dentin.10

MANAGEMENT

NONSURGICAL MANAGEMENT
ORIFICE RECOGNITION

An important fact to remember is that the canal space in normal root canal anatomy is always in the cross- sectional center of the root. Similarly the pulp chamber is (or was, before calcification) located in the cross sectional center of the crown.11In a tooth with a calcified pulp chamber, the distance from the occlusal surface to the projected pulp chamber is measured from the preoperative periradicular film, or preferably from a bite- wing film, which maximizes accuracy. They applied the buccal object rule for the determination of calcified root canals as follows:

After the initial access opening, the bur is left in place and three radiographs are taken:

- 1.Straight – on to the bucco- lingual dimension to determine the position of the head of the bur in the root canal in the mesio- distal dimensio
2. Radiograph taken with a 20 o horizontal angulation with the cone shifted distally.
3. Radiograph taken with a 20 o horizontal angulation with the cone directed mesially.

The last two radiographs give information regarding the relation of the bur to the canal lumen in the bucco- lingual dimension.12

Krasner and Rankow have given certain laws which are particularly useful in locating calcified canal orifices. These are:

1. Law of symmetry 1: Except for maxillary molars, the orifices of the canal are equidistant from a line drawn in a mesiodistal direction through the pulp- chamber floor.
2. Law of symmetry 2: Except for maxillary molars, the orifices of the canals lie on a line perpendicular to a line drawn in a mesiodistal direction across the center of the floor of the pulp chamber.
3. Law of color change: The color of the pulp chamber is always darker than the walls.
4. Law of orifices location 1: The orifices of the root canals are always located at the junction of the walls and the floor.
5. Law of orifices location 2: The orifices of the root canals are always located at the angles in the floor- wall junction.
6. Law of orifices location 3: The orifices of the root canals are located at the terminus of the root development fusion lines.

The LN bur (Caulk/ Denstply, Tulsa, OK, USA), the Mueller bur (Brasseler, Savannah, GA, USA) and thin ultrasonic tips are especially useful for locating calcified canals. Another important instrument for orifice location is the DG-16 explorer. At this point a fine instrument, usually a No. 8 or No. 10 K –file is placed into the orifice, and an attempt is made to negotiate the canal. An alternative option is to use instruments with reduced flute, such as a Canal Pathfinder (JS Dental, Ridgefield. Conn.) or instruments with greater shaft strength such as the Pathfinder CS (Kerr Manufacturing Co.), which are more likely to penetrate even highly calcified canals.Remove the cervical ledge or bulge. If the orifice still cannot be negotiated with a fine instrument, drill 1-2 mm into the center of the orifice with a No.2 round bur on slow speed and use the explorer to re-establish the canal orifice. When counter- sinking or troughing in an area where an orifice is located, be sure the pulp chamber is dry. The bur rotating at a slow speed will remove whitish chips that then accumulate in the orifice. After a light stream of air is blown into the chamber, these chips appear as white spots on the dark floor of the chamber and serve as markers for exploration or further countersinking. This approach can be used if the fourth canal of the maxillary molar or a separation of the mesio-buccal and mesiolingual canals is anticipated in mandibular second molars.Recently a combination of access refinement ultrasonic tips and magnification has revolutionized the basic concept of access cavity preparation. The uncovering of the floor of the pulp chamber can be accomplished with the help of the CPR 2D or BUC 1 tips. The pulp stones sometimes can be vibrated or teased out by the CPR 2D or BUC 1 tips; at other times, they can be planed with the help of a BUC 2 tip- a process similar to planning the root surface. Grind the floor until the dark- colored dentine becomes visible. is of critical importance because it dictates and guides the extension of access cavity.13 Locating canals and initial penetration under the microscope is also aided by fine instruments like the Micro- Orifice Opener(Denstply Maillefer, Ballaigues, Switzerland.).

BIOMECHANICAL PREPARATION

Coronal flaring in a crown- down fashion is preferred.Incremental instrumentation is achieved by creating new increments between the established widths by cutting off a portion of the file tip, thus making it slightly wider in diameter. For example, if a 1 mm segment is clipped from a size 10 file, the instrument becomes a size 12, by trimming sizes 15, 20 and 25, instruments of sizes 17, 22 and 27 respectively can be created. In extremely sclerotic canals, only 0.5 mm segments are trimmed, increasing the instrument width by 0.01mm and making a size 10 into a size 11, etc. because cutting the shaft imparts a flat tip, a metal nail file is used to smooth the end and reestablish a bevel after the removal of any segment.6

CHELATING AGENTS-IS THERE ANY ROLE

Chelator preparations have been advocated frequently as adjuncts for root canal preparation, especially in narrow and calcified root canals. Apical dentin is more frequently sclerosed, and is more mineralized. The authors recommend liquid EDTA solution be introduced into the

pulp chamber (pipette, cotton pellet) to identify the entrance to calcified canals.14

TIME TO STOP LOOKING FOR CANAL

In the treatment of calcified canals it is common to find a total occlusion of the canal space at any level.11 Smith reviewed the literature concerned with calcific metamorphosis and collected the following data:

- Calcified teeth that were not treated endodontically developed radiographic or clinical symptoms in upto 16% of the cases.
- Studies of the success and failure of conventional endodontic therapy reported failure rates of 10% to 19%.

When these failure rates are compared, it appears that a calcified tooth treated endodontically would have no better chance of success than if it were left alone.8

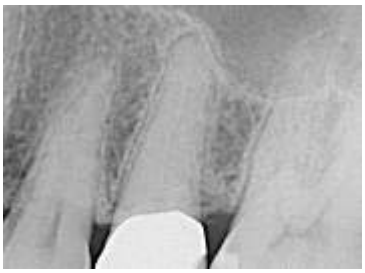

Fig 1 :PRE-OPERATIVE

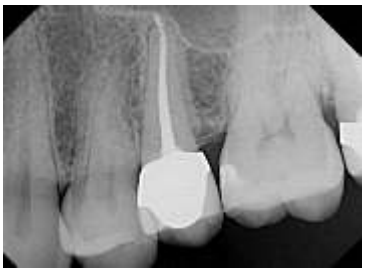

Fig 2: POST-OPERATIVE

The patient reported with pain and swelling in 25
Fig 1 shows a case with calcified canal.
After locating the canal, final obturation was done. (Fig 2)

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Souce of Support: Nill, **Conflict of Interest:** None declared

Case Report

Chaetomium Infection In A HIV Positive Patient

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Abstract

A case of phaeohyphomycosis caused by species of Chaetomium is reported. The patient was HIV positive. Saboraud's Dextrose Agar inoculated with saliva sample as well as scraping from the ulcer showed no growth. However, on 25th day, whitish ruffled growth was seen on the agar with characteristic black areas. These are called perithecia and are characteristic of Chaetomium. The diagnosis was also confirmed by slide culture test & LCB preparation. Infections due to Chaetomium species are rare

Key words
Chaetomium, Fungal Growth, Agar

INTRODUCTION

Chaetomium is a dematiaceous filamentous fungus found in soil, air, and plant debris. As well as being a contaminant, Chaetomium spp. are also encountered as causative agents of infections in humans (1, 2).

CASE REPORT

A 44 years male reported to Dept. of oral medicine and diagnosis of HIDS, Paonta sahib with chief complaint of non healing ulcer for past four months on the tip of the tongue. Careful history revealed that he had chronic productive cough for past two years. Patient was also suffering from chronic diarrohea for past eight months. On oral examination, it was revealed that he had three teeth left in his oral cavity i.e. 12,13,21. Patient was a chronic smoker having around two packets of bidis daily for past thirty years. Cervical lymph nodes were palpable & non tender.
Patient was on medication from a local doctor for past 3-4 months but with no relief from pain.
Following investigations were carried out:
1.Blood samples was obtained and sent to laboratory for testing of HIV antibodies, Hb, TLC, ESR.
2.A saliva sample was obtained by making the patient rinse his mouth with normal saline and expectorate was collected in a sterile container. This was inoculated on Saboraud's Dextrose Agar.
3.A scrapping was obtained from the ulcer and was inoculated on Saboraud's Dextrose Agar.
The patient was recalled after three days to collect his report. The patient was also referred to the Deptt. Of Medicine, Puranchand Hospital for the treatment of diarrohea, cough, etc.
The blood investigations confirmed that the patient was anemic & HIV positive. But the patient never reported back. Saboraud's Dextrose Agar inoculated with saliva sample as well as scraping from the ulcer showed no growth. However, on 25th day, whitish ruffled growth was seen on the agar with characteristic black areas. These are called perithecia and are characteristic of Chaetomium. The diagnosis was also confirmed by slide culture test & LCB preparation.

DISCUSSION

The genus Chaetomium was established by Gustav Kunze (59) who in 1817 published a description of a hitherto unknown genus (3). The genus Chaetomium is a member of subphylum Ascomycotina (family chaetomiaceae) in which the osteolate ascocarps (perithecia) are covered with thick walled pale to dark brown, straight elaborately branched or coiled hairs or septae. Chaetomium are occasionally encountered as contaminant in clinical specimens (4). The genus Chaetomium contains several species. The most common ones are Chaetomium atrobrunneum, Chaetomium funicola, Chaetomium globosum, and Chaetomium strumarium. Chaetomium spp. are among the fungi causing infections wholly referred to as phaeohyphomycosis. Fatal deep mycoses due to Chaetomium atrobrunneum have been reported in an immunocompromised host. Brain abscess, peritonitis, cutaneous lesions, and onychomycosis may also develop due to Chaetomium spp. [4,5,6, 7,8].
Chaetomium colonies are rapidly growing, cottony and white in color initially. Mature colonies become grey to olive in color. From the reverse, the color is tan to red or brown to black [1, 5]. Microscopic Features-Septate hyphae, perithecia, asci and ascospores are visualized. Perithecia are large, dark brown to black in color, fragile, globose to flask shaped and have filamentous, hair-like, brown to

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black appendages (setae) on their surface. Perithecia have ostioles (small rounded openings) and contain asci and ascospores inside. Asci are clavate to cylindrical in shape and rapidly dissolve to release their ascospores (4 to 8 in number). Ascospores are one-celled, olive brown in color, and lemon shaped [1, 5].

Though this fungus has been reported in English literature this is the first case in which it has been isolated from the oral cavity. Not much is known about the oral manifestations of this fungus. If these cases are ignored and the fungus is allowed to spread it will result in increased mortality and morbidity.

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Souce of Support: Nill, **Conflict of Interest:** None declared

Case Report

HEMISECTION AS AN ALTERNATIVE TREATMENT FOR DECAYED MULTIROOTED TOOTH

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Abstract

Hemisection denotes removal or separation of root with its accompanying crown portion of mandibular molars. This procedure represents a form of conservative dentistry, aiming to retain as much of the original tooth structure as possible. The results are predictable and success rates are high. In this paper a case is presented in which hemisection was done because the tooth was grossly carious along with furcation involvement. Mesial half of tooth was extracted and the remaining tooth was restored as premolar which helped to reduce the masticatory load.

Key words
Hemisection, Mandibular Molars, Root Resection

INTRODUCTION

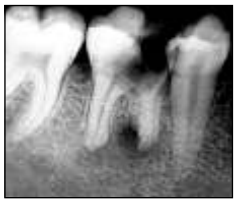
Hemisection (removal of one root) involves removing significantly compromised root structure and the associated coronal structure through deliberate excision.1 Because of two roots present in mandibular molars, one half of the crown and associated root is removed. Thus tooth resection procedures are used to preserve as much tooth structure as possible rather than sacrificing the whole tooth. It differs from bicuspidization, in which a separation is made between the two roots in the furcation area without removal of any root. The separated roots along with its crown part are then restored as premolars2. Selected root removal allows improved access for homecare and plaque control with resultant bone formation and reduced pocket depth.

This procedure is indicated3

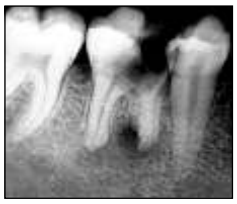
- If there is severe bone loss limited to one root or involvement of a Class III furcation that could produce a stable root after hemisection.
- If the patient is unable to perform appropriate oral hygiene in the area.
- Extensive exposure of the roots because of dehiscence is another indication for excision of one root.
- Indicated for failure of an abutment within a fixed prosthesis, provided a portion of the tooth can be retained to act as the abutment for the prosthesis.
- Untreatable endodontic failure due to perforations and broken instruments.
- Vertical root fracture confined to a single root of a multirooted tooth or any severe destructive process that is confined to a single root, including caries, external root resorption and trauma.

CASE REPORT:

A 30 years old male patient reported to the department with the complaint of pain in left mandibular first molar. On examination, the tooth was tender to percussion and was grossly carious. On probing the area, there was a deep periodontal pocket around the mesial root of the tooth. On radiographic examination, furcation involvement was evident. The bony support of distal root was completely intact (Fig. 1). It was decided that the mesial root should be hemisected after completion of endodontic therapy of the tooth.



Biomechanical preparation was done with in the distal root only (Figure 2).



Hemisection of the mesial root and crown was done with a vertical cut method. After vertical incision and sulcular incision, a mucoperiosteal flap was reflected. The crown was cut with a long shank, tapered fissure carbide bur till the furcation is reached(Figure 3). Once the separation was

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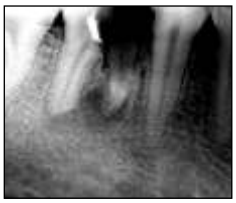

complete, the mesial half was extracted. The empty socket was thoroughly irrigated and the flap was sutured back into its position.
After the complete healing of the extraction socket , the crown of the remaing thooth was restored with FPD on 45,46 so as to distribute the occlusal stresses(Figure 4).

DISCUSSION:

Before selecting a tooth for hemisection, patient's oral hygiene status, caries index and medical status should be considered. Also the accessibility of root furcation for ease of operation as well as good bone support for the remaining roots should be assessed. The furcation region is carefully smoothed, to allow proper cleansing and thus to prevent accumulation of plaque.4 Root fracture is the main cause of failure after hemisection, so occlusal modifications are required to balance the occlusal forces on the remaining root.5
Contraindications include the presence of a strong abutment tooth adjacent to the proposed hemisection, which could act as an abutment to prosthesis. The remaining root may be inoperable for the necessary root canal treatment6. Also, fusion or proximity of the roots may prevent their separation3.
Hemisection may be a suitable alternative to extraction and implant therapy and should be discussed with patients during consideration of treatment options

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Souce of Support: Nill, **Conflict of Interest:** None declared

Case Report

AN UNUSUAL CASE OF TALON CUSP ON MAXILLARY CENTRAL INCISOR

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Abstract

Dens evaginatus is a developmental anomaly characterized by the occurrence of an extra cusp shaped tubercle projecting from the palatal or buccal surfaces (talon cusp). Commonly it occurs in either maxillary or mandibular anterior teeth in both the primary and permanent dentition. This article reports a case of talon cusp and its management.

Key words
Dens Evaginatus, Talon Cusps, Eagle's Talon.

INTRODUCTION

The talon cusp, or dens evaginatus of anterior teeth, is a relatively rare developmental anomaly characterized by the presence of an accessory cusp like structure projecting from the cingulum area or cemento-enamel junction of the maxillary or mandibular anterior teeth in both the primary and permanent dentition. This anomalous structure is composed of normal enamel and dentin and either has varying extensions of pulp tissue into it or is devoid of a pulp horn.1-4 In its typical shape, this anomaly resembles an eagle's talon,2 but it could also present as pyramidal, conical or teat-like.1-5The prevalence of talon cusp varies considerably among populations, ranging from 0.06% to 7.7%.6,7 The permanent dentition is affected more frequently than the primary dentition, and the anomaly is more common in males than in females.1,4,5,8,9 predominantly 65%of the talon cusps occurs in males 10 and prevalence varies considerably among ethnic groups ranging from 0.06% to 7.7% 11. Almost 92% of the affected (taloned) teeth in the permanent dentition have been found in the maxilla, with the lateral incisors being the most frequently involved (55%) followed by the central incisors (36%) and the canines.1,9

The etiology of talon cusp is not well understood, but appears to have both genetic and environmental components.1, 4 Similar to other abnormalities of tooth shape, talon cusp originates during the morpho-differentiation stage of tooth development. It may occur as a result of outward folding of inner enamel epithelial cells and transient focal hyperplasia of the peripheral cells of mesenchymal dental papilla. It can also occur as an isolated finding or in association with other dental anomalies such as peg-shaped lateral incisor, agenesis or impacted canines, mesiodens, complex odontomes, megadont, dens evaginatus of posterior teeth, shovel-shaped incisors, dens invaginatus and exaggerated Carabelli cusp.1-5 The talon cusp has not been reported as an integral part of any specific syndrome, although it appears to be more prevalent in patients with Rubinstein-Taybi syndrome, Mohr syndrome, Sturge-Weber syndrome, incontinentia pigmenti achromians and Ellis-van Creveld syndrome.1,8,12

CASE REPORT

A healthy looking 29 year old male presented to the dental OPD of department of Conservative Dentistry and Endodontics, U.P. Dental College and Research Centre, Lucknow, India for a dental check-up. It was his first visit to the dentist. He did not present any significant medical history. Oral examination showed good oral hygiene, maxillary right central incisor was displaced labially with an accessory cusp on the palatal aspect. There was a negative family history of such dental anomaly from the patient and there was no associated systemic disorder. The cusp projected from the cemento - enamel junction and curved towards the incisal edge of the incisor (Figure 1)

which was around 3mm wide (mesiodistally),4mm (incisocervically) and 3mm thick (labiolingually) extending from cingulum area to the 0.5mm short of incisal edge . The affected tooth was labially placed in the arch with occlusal interferences on talon cusp during occlusion.

A periapical radiograph revealed an inverted V-

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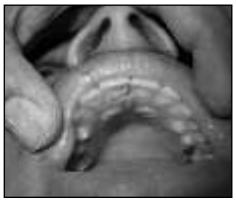
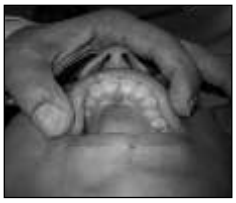
shaped radiopaque structure on the maxillary right central incisor. The extent of pulp tissue into the cusp could not be determined on the radiograph. A diagnosis of type 1 talon cusp was made. The condition and the planned periodic and gradual reduction of the cusp with topical fluoride application and Composite resin facing was explained to the patient. Orthodontic alignment of the displaced central incisor was also planned. With his consent, after oral prophylaxis, a minimal reduction of the talon cusp was carried out using a diamond bur in a high-speed water-cooled handpiece (Figure 2).



Acidulated Phosphate Fluoride (APF) gel was applied to the surface of the reduced cusp (Figure 3)



to avoid any postoperative sensitivity.

DISCUSSION

Talon cusp is an odontogenic anomaly of tooth shape that represents the extreme of continuous variation progressing from an enlarged cingulum (trace talon) through a small accessory cusp (semitalon) to a talon cusp.1 Small talon cusps are usually asymptomatic and need no treatment. Large talon cusps may cause clinical problems including occlusal interference, displacement of the affected tooth, irritation of the tongue during speech and mastication, carious lesion in the developmental grooves that delineate the cusp, pulpal necrosis, periapical pathosis, attrition of the opposing tooth and periodontal problems due to excessive occlusal forces.1-5Talon cusps also present diagnostic and treatment difficulties. On unerupted tooth, the anomalous cusp can radiographically be mistaken for a supernumerary tooth or compound odontomas, leading to unnecessary surgical intervention. This diagnostic problem is especially significant because approximately 90% of all supernumeraries occur in the maxilla and half of these in the incisor region.13

Hattab et al classified this anomaly into 3 types on the basis of the degree of cusp formation and extension. Type I (talon) has an additional cusp that projects from the palatal surface of an anterior tooth and extend at least one half the distance from thecement enamel junction to the incisal edge. Type II (semitalon) has an additional cusp 1 mm or more in length but extending less than one half the distance from the cement enamel junction to the incisal edge. Type III (trace talon) manifest enlarged and prominent cingula and their variation14. The talon cusps described in the current case classified as type1 (talon). Furthermore it is important to remember that talon cusp is occasionally combined with other systemic and dental anomalies15. However, none of these alterations was found in this case.

The treatment of talon cusp involves careful clinical judgment and review of whether the cusp contains or is devoid of a pulp horn. Earlier reports, based on radio graphic examination, stated that removal of the cusp would inevitably lead to pulp exposure that would require endodontic treatment.2 However radiographic tracing of the pulpal configuration inside the talon cusp has inherent difficulties because the cusp is superimposed over the affected tooth crown. Similarly, histological examination of extracted talon teeth failed to show the presence of a pulp horn in the talon cusp.15-18 Pitts and Hall removed 3 mm of the anomalous cusp in one visit, without pulp exposure.19Several times, we have reduced 1.0 mm to 1.5 mm of talon cusp in one appointment without exposing the pulp.1,5 However, this does not imply that all talon cusps are devoid of pulp horn.

CONCLUSION

Talon cusp is a not an harmless defect, as it may provide a challenge duringdiagnosis and treatment planning to clinician. Early diagnosis may minimize certain problems such as caries, periodontal disease and malocclusion.

The management and treatment outcome of talon cusp depends on the size, presenting complications and patient cooperation.

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Case Report

"Buccal Connector Immediate Partial Denture"

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Abstract

A 37-year old male patient presented with a complaint of long- standing dissatisfaction with the esthetics of his anterior teeth. He was insistent that he would not consider retaining his unsightly anterior teeth. His smile and self-confidence were affected by his dissatisfaction with his dental esthetics. The unsightly teeth had to be removed and an immediate prosthesis provided. The treatment was complicated by the deep bite which did not permit conventional partial denture service. The treatment carried out utilised a modification of Removable Partial Denture involving Buccal connectors. This modification succeeded in overcoming the limitations quite satisfactorily.

Key words
Buccal Connector, Immediate Denture, Modification, Removable Partial Denture

INTRODUCTION

Immediate denture is defined as "any removable dental prosthesis fabricated for placement immediately following the removal of a natural tooth/teeth"1

The concept of Immediate dentures is not new. As early as 1860, Richardson2 described the use of immediate dentures. Various workers have published about the use of complete dentures immediately following dental extractions.3,4,5 In the early years, this treatment modality was considered a luxury and offered only to few patients. According to Seals et al, 6 other than few exceptions, maxillary anterior teeth should always be replaced with immediate dentures.

Advantages of Immediate Dentures 6

Many advantages of Immediate Dentures have been given-

- Prevent patient embarrassment- at no time is the patient without teeth
- Promote dental health- with the option of immediate dentures, patients are less Reluctant to get diseased anterior extracted.
- Easier to achieve natural esthetics- as there is an opportunity to replicate the natural Dentition closely
- The natural teeth provide a guide for Vertical Dimension. Also there are less changes in the facial musculature.
- Unfavourable speech and chewing habits are less likely to develop.
- Promote better healing- as the immediate dentures act as a stent and protect the Wound. This prevents dislodgement of blood clot, and also reduces bleeding and postextraction pain.
- May decrease the Residual Ridge Resorption, and also promotes better ridge form.
- Prevent collapse of facial musculature
- Patients adapt to the dentures more easily and faster

Disadvantages of Immediate Dentures6

- Increased treatment complexity
- Try- in is not possible
- Increased treatment cost- as it is suggested to re-line or re-make the dentures within few months.
- Increased patient visits- due to the follow- up treatments and care required.

Contra-indications of Immediate Dentures6

- Patients who have undergone head- and- neck radiotherapy
- Patients with bleeding disorders or impaired wound healing.
- Aged or medically compromised patients.
- Patients with cysts, abscesses or those requiring extensive surgical removal of bone
- Patients with psychological disorders or diminished mental capacities.

Connector design

Most partial dentures are constructed using either palatal or lingual connectors. In rare cases, in some mandibular dentures, labial bars have been used as the major connector of choice. This is a variation of conventional Removable Partial Denture design in which the parts of a prosthesis on one side of the arch are connected with those on the opposite side by a component which passes labial to the remaining natural teeth.

The most common indication for labial bar is the presence of lingually tipped anterior teeth.

Other indications include severe crowding of anterior teeth, large mandibular tori and severe soft tissue undercuts on the lingual side of the anterior teeth.7

Facial connectors may be in the form of either Labial or Buccal Connectors in the form of either bars or

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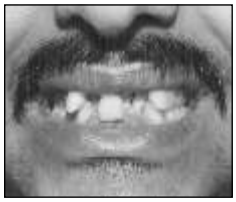
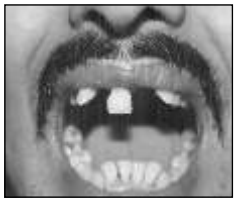
plates. Labial bar has been used by some workers in maxillofacial prostheses8,9,10. It has been also suggested as an alternative to more conventional designs in cast partial dentures.11,12,13,14 A further modification of Labial- bar is the Swing-Lock design.7 However, the Buccal- bar or Buccal- plate has rarely, if ever, been used in Removable Partial Dentures.

Usually, these connectors are cast in metal as part of the denture framework. Also, since the prosthesis is not intended for long term use, it was considered prudent to keep the treatment simple and cost-effective by utilising an all- acrylic denture design.

Case Report

The patient, a 37 year old male presented to the Out- Patient Department, with a complaint of dis-satisfaction with the esthetics of his anterior teeth and smile. On examination, it was observed that his maxillary left central incisor was missing. Also, both the first and second molars on both left and right sides of maxilla were missing. There was compromised periodontal health with almost all teeth exhibiting Grade I mobility. The maxillary right central incisor and both the maxillary lateral incisors were seen to have Grade III mobility.The treatment plan was complicated by the presence of 100% deep bite, with the edges of the lower incisors touching the palate in the rugae area. [figs. 1, 2]





The patient had earlier got a removable denture fabricated for the missing central incisor from some other dentist. He reported he could never use this denture as it interfered with his occlusion.

The patient was suggested orthodontic treatment, which he declined. He was insistent upon removal of the mobile and unsightly teeth. He would not accept any treatment plan which would preserve the mobile teeth (viz. Elective pulpectomies and post- and- core to correct the alignment). The patient was open to any form of replacement of the extracted teeth- he was willing to consider removable or fixed replacements. The patient was healthy and there was no significant medical history.

Keeping in mind the patients requirements, it was decided to extract the mal-aligned teeth and provide an Immediate Removable replacement.

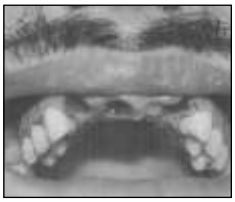
Technique

An innovative design was considered because a conventional design was not possible as the lower incisor teeth would strike the denture base of a conventional prosthesis.

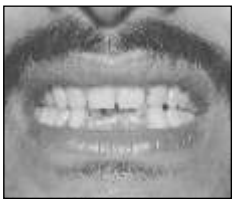
Procedure

1. Basic oral prophylaxis was carried out.
2. Impressions were made of the maxillary and mandibular arches using Irreversible hydrocolloid (Zelgan, Dentsply, India)
3. A jaw- relation record was made in Maximum- Intercuspation position using modelling wax (Y-Dents, MDM Corporation, Delhi, India). Double- thickness of wax was used in horse- shoe shape and softened by immersion in warm water. The patient was asked to bite into the wax till complete closure.
4. The impressions were poured in Type III Dental Stone (Kalstone, Kalabhai, Mumbai, India)
5. The gross undercuts were blocked- out using Type II Dental Plaster. (Neelkanth,)
6. The dental casts were mounted on a non- adjustable articulator. (Jabbar)
7. The maxillary cast was modified with removal of the teeth to be extracted. The removal of stone teeth was done one- at- a- time, and artificial teeth replaced accordingly. This technique is useful in achieving more natural esthetics. The teeth were placed in a more superior plane than that existed naturally.
8. After teeth arrangement, wax- up was completed. Palatally, the wax- up was kept open in the rugae area. The buccal connectors were created in the wax- up.
9. The waxed- up denture was de-mounted, flasked, and processed in heat cure denture base resin (Trevalon, Dentsply, India) in the usual manner.
10. The denture was retrieved, finished and polished and ready for insertion [figs. 3,4]

11. The patient was recalled, and administered Local Anesthesia- using naso-palatine nerve block. This was supplemented with labial infiltrations.
12. The maxillary right central incisor and both the lateral incisors were extracted. [fig. 5] The extractions were done atraumatically and minor labial plate alveoloplasty was done.



13. The finished and polished prosthesis was tried and upon minor adjustment, was suitable for insertion. The prosthesis was checked for high points and the occlusion adjusted accordingly. The patient was satisfied with the esthetics of the prosthesis. [fig. 6]



14. The patient was advised post- extraction precautions and discharged. He was to report after 24 hours and not remove the prosthesis in the meantime.
15. Upon 24- hour recall, the prosthesis was removed, checked and cleaned. The sockets seemed to be healing well and the patient did not have any pain. The patient was further advised about the use of the prosthesis and asked to report after 1 week to plan

the definitive treatment.

Discussion

Immediate removable dentures are a very satisfying treatment modality for both patients and dentists. The patients get the benefit of improved confidence, continued dental esthetics and improved comfort. The dentist finds satisfaction in providing a very acceptable treatment to the patient. The usual design of immediate dentures was not suitable for this patient. The patient presented with dissatisfaction with dental esthetics and consequently, his smile. His dental situation was further complicated by the presence of 100% deep- bite which was traumatising the palate in the rugae region. A unique design of Immediate Partial Denture has planned for this patient. This design of Denture was successful in overcoming the problems of deep bite and lack of space needed for a conventional Partial Denture. The initial retention and stability was good. The patient was able to maintain satisfactory oral hygiene and the patient had no complaints- either esthetic or functional. This design utilised buccal connectors of denture base acrylic. The strength of acrylic is not adequate for making either labial or buccal connectors intended for long- term use. However, this prosthesis was intended for use as an Immediate, Interim Denture. Therefore, it was concluded that this design can be used as a suitable alternative to conventional removable partial denture design in some rare cases. This design is advocated for interim usage only. Long- term use, as in definitive prostheses, would require cast frameworks.

CONCLUSION

Clinical practice in dentistry entails all kinds of services including restorative procedures.Most prosthesis designs are fairly straightforward and require simple application of established procedures and concepts. However, some patients with unique complications and requirements force us to think “out-of-the-box”. If we use sound scientific principles and customise them with common sense, we may be able to provide simple solutions to such patients. The case presented here was satisfactorily dealt with by using a minor modification of a tried and tested concept. It can be safely concluded that the design utilised may be used for similar cases.

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Case Report

Functional And Esthetic Rehabilitation Of Severely Worn Anterior Dentition Caused By Anterior Interferences

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Abstract

Anterior interferences seen commonly with retroclined maxillary incisors may lead to severe attrition of the anterior teeth, anterior spacing, gingival recession or intra capsular derangement of the TMJ. These attritioned incisors are usually treated with bite opening followed by veneers and full coverage crowns. However this approach does not pay attention to correcting the inter incisal angle and hence establishing correct incisal guidance which leads to failure of the restoration. This case report documents the orthodontic and restorative treatment of destructive occlusal wear in the anterior region.

Key words
Anterior Interference, Attritioned Teeth, Inter Incisal Angle

INTRODUCTION

The term occlusal interference is generally understood to denote an undesirable contact between some aspect of the maxillary and mandibular dentitions, with a consequent disruption of fluid mandibular closure into maximum intercuspation, or unhindered movement of the mandible in protrusive or lateral excursive movements. Anterior interferences have been defind as : An occlusal interference on the anterior teeth, identified during unforced closure of the mandible, sometimes associated with a distalizing effect on condylar position. They are seen more commonly in cases with retroclined maxillary incisors and under torque upper central incisors. These interferences may lead to severe attrition of the anterior teeth, anterior spacing, gingival recession or intra capsular derangement of the TMJ. This case report documents the orthodontic and restorative treatment of destructive occlusal wear in the anterior region.

A 34 year old female presented with presented with a single, irregular, reddish, pedunculated 2x2 cms gingival swelling on lingual side w.r.t 47,48 (Fig.1).

CASE REPORT:

The patient, a 46 year old male reported with a chief complaint of hypersensitivity and attrition of lower front teeth. Excessive incisal wear was noted on the mandibular central incisors.(fig 1) However, there was virtually no discernible wear on the lateral incisors, and it is quite unlikely that the effects of bruxism would be this localized without some other factor being involved. The type of excessive incisal wear noted in this example is an obvious deleterious effect of an anterior interference. The lower left lateral incisor was non vital. Molar relation was class I, over jet was 1mm and overbite was 80%. The oral hygiene was acceptable, the probing depths were normal. Cephalometric examination revealed retroclined maxillary incisors and correctly inclined mandibular incisors. Radiological examination presented no other significant finding.

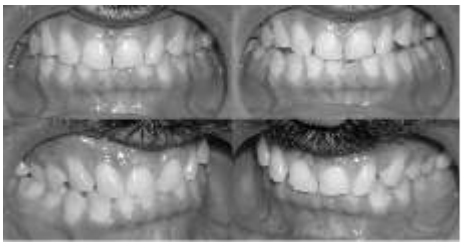

Figure 1: Pre-Treatment photographs of the patient showing retroclined incisors and severe attrition of the mandibular incisors.

TREATMENT

Treatment plan was presented to the patient. The patient was informed regarding orthodontic bite opening procedure, correcting the axial inclination of teeth and establishing a correct interincisal angle and hence the incisal guidance. The patient understood the plan and gave his consent for the orthodontic treatment and restorative procedures.

Endodontic treatment of 32 was performed. A deprogramming splint was fabricated and adjusted for the patient. Patient was comfortable with it in 1 week. He was recalled every two weeks for 2 months. Slight bite opening was observed. Orthodontic treatment was instituted using 018 slot Roth prescription only in the upper dentition. No orthodontic intervention was done in the lower jaw. Alignment and leveling was done with round and

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later with rectangular 16 x 22 NiTi wires. This resulted in correction of inclination of upper incisors and bite opening. After bite opening with intrusion of maxillary incisors adequate overjet and over bite was achieved with consolidation of the spaces that appeared between the anterior teeth. (Fig 2).

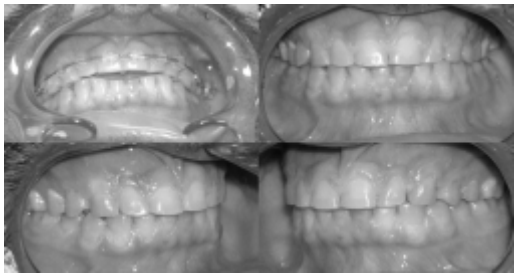

Figure 2. Photograph showing adequate overjet and over bite achieved to allow restoration of the attrided lower incisors.

Filtek Z 350 (3m Unitek) was used to restore the lower anteriors.(Fig 2). A Hawley's retainer with anterior bite plane was fabricated and delivered to the patient.

CONCLUSION

The purpose of this article was to introduce the concept of correction anterior interferences before restoring the worn off dentitions and not just restoring the teeth after raising the bite. It is of paramount importance to understand that correct axial inclination of maxillary and mandibular anteriors and not just bite opening will ensure the longevity of the restorations.

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Review Article

LINGUAL ORTHODONTICS – AN OVERVIEW

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Abstract

Man has been striving for generations to improve his outlook, his appearance- what he calls the aesthetic value. The face [in general] and the teeth have also come to play a part in his presentation to the outside world. To enhance this desire, attention has been given to correct malformations of teeth. In earlier times this was done by crude methods. And then evolved the concept of "braces"; fixed on the labial surfaces. Orthodontists have given a new dimension to their arena of operations, shifting from the labial to the lingual so as to give rise to the concept of "invisible braces' or Lingual Orthodontics

Key words
Invisible Braces, Bracket Design, TARG

LINGUAL ORTHODONTICS – AN OVERVIEW

Man has been striving over generations to improve his outlook, his appearance – what he calls the esthetic value. The face [in general] and teeth have also come to play a part in his presentation to the world. To enhance this desire, attention has been given to correct the malformations of teeth. Earlier it was done by crude methods. But with advancements in science, newer methods and materials have evolved. Orthodontists are also striving towards this goal and have produced the best of results by giving a new dimension to their arena of operations, changing from the labial to the lingual so as to give rise to a new concept " the Invisible Braces" concept or " Lingual Orthodontics". The value of invisible braces is not in the hardware, but perhaps best expressed in the word "invisible".

PATIENT SELECTION

Lingual therapy is demanding on both the patient and the dentist. So the patient must be made aware that it will require greater effort and chair time. The most important factors in selecting patients for lingual treatment seem to be their personalities and reasons for seeking treatment. The patient should be informed of the rationale and the effects of lingual appliance, speech, soreness, bite opening] and told that their attitude should be one of understanding and a desire to do whatever is necessary to accomplish the optimum results.

BRACKET DESIGN

To compensate for the tooth form and shape seen on the lingual, some considerations in bracket design need to be followed:

1 Since inter bracket distance is reduced on the lingual, the bracket must be designed to be as narrow as possible.

2 Because of decreased bracket width, mesio-distal root control becomes difficult which can be taken care of using vertical slots for auxiliaries.

3) Since the lingual contours of teeth vary a lot, the amount of torque supplied by the bracket will be very sensitive to its occluso-gingival placement. This can be solved by indirect bonding procedures with Pre-angulated pre-torqued brackets.

4) Consideration should be given to the ease of insertion, ligation and removal of the arch wires.

Eventually the method selected for the determination of lingual bracket torques and thickness was to relate the lingual determinants to labial tooth anatomy. Tracings of the labial and lingual profiles were made and a line was drawn through the LA point (now FA point), representing the plane to the arch wire, to define labial torque. Lingual equivalent torque values were then calculated studied statistically and reduced to set of average lingual torque values. Similar studies were conducted to define lingual pad profile and contours, lingual molar bracket torques, rotations, base curvatures and in-out relationships.

BRACKET PLACEMENT

The many variations in tooth size, lingual contour, cingulum and marginal ridge anatomy, inconsistencies in tooth form, shape and inclination of the lingual surface make the use of predetermined bracket placement of no much utility. Smith et al suggested the use of indirect bonding by TARG (torque and angulation reference guide). The TARG instrumentation is designed to transfer bracket prescriptions from the more reliable labial surfaces of each tooth to the lingual at a given bracket height.

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This allows to set customized torque and angulation for each tooth.

ARCH WIRES

There is a dramatic difference in the arch form with lingual treatment. Fujita described the "Mushroom Arch" as necessary in lingual treatment because of the difference in facial – lingual thickness of anterior and posterior teeth; also there is a large constriction in arch width as one proceeds distally form the lingual surface of canine to the bicuspid. Since the brackets are designed to minimize bracket profiles, it is necessary to place compensating 1st order bends interproximally at cuspid-bicuspid and bicuspid-molar regions.

BONDING

For bonding of lingual brackets, the preferred mode of placement is indirect because:-

1. The variation in lingual tooth morphology creates the need for custom measurement for selection of appropriate bracket base thickness and torque.
2. The clinician's lack of familiarity with lingual tooth morphology makes it difficult to visualize angulations and bracket heights.
3. It is difficult to obtain a direct line of sight for bonding.
4. Increased accuracy in bracket placement is required because compensating lingual arch wire bends are more difficult and time consuming to form.

A modified dental surveyor and TARG (torque and angulation reference guide) are used to align the lingual surfaces relative to the labial crown inclinations. Once the bracket slot height and angulation are marked, indirect bonding of the brackets on the lingual is done.

TREATMENT SEQUENCE

Four phases are normally seen:

1. Leveling, aligning, rotational control and bite opening.
2. Torque control.
3. Consolidation and retraction.
4. Detailing and finishing.

CONCLUSION

Many patients would like to have the benefits offered by high quality orthodotontic treatment but do not want to have braces that are visible to their friends and colleagues, a situation considered to be a social stigma by some. Lingual orthodontics is a way out for such patients. The lingual appliance is no panacea, but if patients are carefully selected, lingual braces can be a valuable addition to the orthodontist's armamentarium.

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Review Article

SYSTEMIC DISEASES ASSOCIATED WITH ORAL INFECTION- A REVIEW

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Abstract

It has been recognized that oral infection, especially periodontitis, may affect the course and pathogenesis of a number of systemic diseases, such as cardiovascular disease, bacterial pneumonia, diabetes mellitus, and low birth weight. The purpose of this review is to evaluate the current status of oral infections, especially periodontitis, as a causal factor for systemic diseases.

Key words

Periodontitis, Infection, Systemic Diseases.

INTRODUCTION

Introduction

The theory of focal infection, which was promulgated during the 19th and early 20th centuries, stated that "foci" of sepsis were responsible for the initiation and progression of a variety of inflammatory diseases such as arthritis, peptic ulcers, and appendicitis [1]. In the oral cavity, therapeutic edentulation was common as a result of the popularity of the focal infection theory. Since many teeth were extracted without evidence of infection, thereby providing no relief of symptoms, the theory was discredited and largely ignored for many years. Recent progress in classification and identification of oral microorganisms and the realization that certain microorganisms are normally found only in the oral cavity have opened the way for a more realistic assessment of the importance of oral focal infection. It has become increasingly clear that the oral cavity can act as the site of origin for dissemination of pathogenic organisms to distant body sites, especially in immunocompromised hosts such as patients suffering from malignancies, diabetes, or rheumatoid arthritis or having corticosteroid or other immunosuppressive treatment. A number of epidemiological studies have suggested that oral infection, especially marginal and apical periodontitis, may be a risk factor for systemic diseases.

Human endodontal and periodontal infections are associated with complex microfloras in which approximately 200 species (in apical periodontitis) [2] and more than 500 species (in marginal periodontitis) [3] have been encountered. These infections are predominantly anaerobic, with gram-negative rods being the most common isolates. The anatomic closeness of these microfloras to the bloodstream can facilitate bacteremia and systemic spread of bacterial products, components, and immunocomplexes.

BACTEREMIA

The incidence of bacteremia following dental procedures such as tooth extraction, endodontic treatment, periodontal surgery, and root scaling has been well documented [4]. Bacteremia after dental extraction, third-molar surgery, dental scaling, endodontic treatment, and bilateral tonsillectomy has been studied by means of lysis-filtration of blood samples with subsequent aerobic and anaerobic incubation [5]. Bacteremia was observed in 100% of the patients after dental extraction, in 70% after dental scaling, in 55% after third-molar surgery, in 20% after endodontic treatment, and in 55% after bilateral tonsillectomy. Anaerobes were isolated more frequently than facultative anaerobic bacteria. Another study [6] involving 735 children undergoing treatment for extensive dental decay found that 9% of the children had detectable bacteremias before the start of dental treatment. In addition, a variety of hygiene and conservative procedures, including brushing of the teeth, increased the prevalence of bacteremias from 17 to 40%. Anesthetic and surgical procedures increased the occurrence of bacteremias from 15 to 97%. One recent study by Debelian et al. [7] used phenotypic and genetic methods to trace microorganisms released into the bloodstream during and after endodontic treatment back to their presumed source, the root canal. Microbiological samples were taken from the root canals of 26 patients with asymptomatic apical periodontitis of single-rooted teeth. Blood was drawn from the patients during and 10 min after endodontic therapy. All root canals contained anaerobic bacteria. In group I, where the first three root canal reamers were used to a level 2 mm beyond the apical foramen of the tooth,

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Propionibacterium acnes, Peptostreptococcus prevotii, Fusobacterium nucleatum, Prevotella intermedia, and Saccharomyces cerevisiae were recovered from the blood. In group 2, where instrumentation ended inside the root canal, P. intermedia, Actinomyces israelii, Streptococcus intermedius, and Streptococcus sanguis were isolated from the blood.

As stated above, dissemination of oral microorganisms into the bloodstream is common, and less than 1 min after an oral procedure, organisms from the infected site may have reached the heart, lungs, and peripheral blood capillary system [8].

SYSTEMIC DISEASES ASSOCIATED WITH ORAL INFECTION:

1) Cardiovascular Disease:

Cardiovascular diseases such as atherosclerosis and myocardial infarction occur as a result of a complex set of genetic and environmental factors [9]. The genetic factors include age, lipid metabolism, obesity, hypertension, diabetes, increased fibrinogen levels, and platelet-specific antigen Zwb (P1A2) polymorphism. Environmental risk factors include socioeconomic status, exercise stress, diet, nonsteroidal anti-inflammatory drugs, smoking, and chronic infection. The classical risk factors of cardiovascular disease such as hypertension, hypercholesterolemia, and cigarette smoking can only account for one-half to two-thirds of the variation in the incidence of cardiovascular disease [1].

Among other possible risk factors, evidence linking chronic infection and inflammation to cardiovascular disease has been accumulating [10]. It is clear that periodontal disease is capable of predisposing individuals to cardiovascular disease, given the abundance of gram-negative species involved, the readily detectable levels of proinflammatory cytokines, the heavy immune and inflammatory infiltrates involved, the association of high peripheral fibrinogen, and the white blood cell (WBC) counts [11].

There are several proposed mechanisms by which periodontal disease may trigger pathways leading to cardiovascular disease through direct and indirect effects of oral bacteria. Evidence indicates that oral bacteria such as Streptococcus sanguis and Porphyromonas gingivalis induce platelet aggregation, which leads to thrombus formation [12]. When S. sanguis is injected intravenously into rabbits, a heart attack-like series of events occur. Possibly, antibodies reactive to periodontal organisms localize in the heart and trigger complement activation, a series of events leading to sensitized T cells and heart disease [12]. In one recent study, Deshpande et al. [13] showed that P. gingivalis can actively adhere to and invade fetal bovine heart endothelial cells, bovine aortic endothelial cells, and human umbilical vein endothelial cells. Invasion efficiencies of 0.1, 0.2, and 0.3% were obtained with bovine aortic endothelial cells, human umbilical vein endothelial cells, and fetal bovine heart endothelial cells.

Ebersole et al. [14] found that patients with adult periodontitis have higher levels of C-reactive protein (CRP) and haptoglobin than subjects without periodontitis. Both CRP and haptoglobin levels decline significantly after periodontal therapy. Another recent study [15] evaluated the relationship of cardiovascular disease and CRP. Groups of adults who had neither periodontal nor cardiovascular disease, one of these diseases, or both of them were assembled. In those with both heart disease and periodontal disease, the mean level of CRP (8.7 g/ml) was significantly different from that (1.14 g/ml) in controls with neither disease. The authors also showed that treatment of the periodontal disease caused a 65% reduction in the level of CRP at 3 months. The level remained reduced for 6 months.

Recently, a specific heat shock protein, Hsp65, has been reported to link cardiovascular risks and host responses [16,17,18]. Heat shock proteins are important for the maintenance of normal cellular function and may have additional roles as virulence factors for many bacterial species [19]. In animal studies, Xu et al. [18] demonstrated that immunization of rabbits with bacterial Hsp65 induces atherosclerotic lesions. A subsequent large-scale clinical study found a significant association between serum antibody levels to Hsp65 and the presence of cardiovascular disease [20]. Their theory, consistent with their clinical findings, is that bacterial infection stimulates the host response to Hsp65, which is a major immunodominant antigen of many bacterial species. The interaction between expressed Hsp65 and the immune response induced by bacterial infection is hypothesized to be responsible for the initiation of the early atherosclerotic lesion [18]. It has been suggested that chronic oral infection stimulates high levels of Hsp65 in subjects with high cardiovascular risk [21]. Thus, if antibodies directed towards bacterial heat shock proteins cross-react with heat shock proteins expressed in the host tissue, especially if they are found in the lining of blood vessels, then some oral species might well be the link between oral infection and cardiovascular disease [21].

Finally, oral infection can also cause tooth loss. Evidence has shown that edentulous persons with and without dentures and dentate individuals with missing teeth change their eating habits[22] . They may thereby avoid certain nutritious foods because of difficulty in chewing and select high-calorie, high-fat food. When the foods cannot be well pulverized, this has an adverse effect on the internal absorption of nutrients. Such dietary preferences would predispose such individuals to the type of high-fat foods that are recognized as risk factors for cardiovascular disease [23]. In dentate individuals with many missing teeth, the diet-induced elevation of serum low-density lipoprotein has been shown to upregulate monocytic responses to LPS [24]. In these subjects, one would have both the diet-induced sensitization of monocytes and the plaque-laden teeth that could provide the LPS challenge to these cells. Instead of having hyperresponsive monocytes reacting to any LPS introduced from the plaque, there would be elevated secretion of inflammatory cytokines by monocytes stimulated by elevated low-density lipoprotein levels. This interaction between LPS and monocytes may explain the severity of gram-negative infections in certain diabetic patients [24], but it could also be operating in individuals who change to a high-fat diet because of missing teeth. Thus, all the mechanisms by which poor oral hygiene and periodontal disease may contribute to cardiovascular disease described above could also come into play as a result of certain dietary changes secondary to missing teeth [21].

2) Coronary heart disease:

Atherosclerosis and Myocardial infarction:

Atherosclerosis has been defined as a progressive disease process that involves the large- to medium-sized muscular and large elastic arteries. The advanced lesion is the atheroma, which consists of elevated focal intimal plaques with a necrotic central core containing lysed cells, cholesterol ester crystals, lipid-laden foam cells, and surface plasma proteins, including fibrin and fibrinogen [25]. The presence of atheroma tends to make the patient thrombosis prone because the associated surface enhances platelet aggregation and thrombus formation that can occlude the artery or be released to cause thrombosis, coronary heart disease, and stroke. Overall, about 50% of deaths in the United States are attributed to the complications of atherosclerosis and resulting cardiovascular diseases [26]. A myocardial infarction is the damaging or death of an area of the heart muscle resulting from a reduced blood supply to that area. Myocardial infarction is almost always due to the formation of an occlusive thrombus at the site of rupture of an atheromatous plaque in a coronary artery [27].

3) Stroke:

Stroke is a cerebrovascular disease that affects blood vessels supplying blood to the brain. It occurs when a blood vessel bringing oxygen and nutrients to the brain bursts or is clogged by local thrombus formation or by aggregates of bacteria and fibrin from other sources such as the heart. In an average population, the annual incidence of new strokes is 2 per 1,000 [27]. Studies on the pathology of stroke indicate that 80 to 85% of these lesions are due to cerebral infarction; 15 to 20% are caused by hemorrhage [27].

The inflamed periodontium releases inflammatory cytokines, LPS, and bacteria into the systemic circulation, and they may promote atherosclerosis and affect blood coagulation, the function of platelets, and PG synthesis, thereby contributing to the onset of stroke. In a case-control study [28], 40 patients under the age of 50 with cerebral infarction and 40 randomly selected community controls matched for sex and age were compared for dental status. Poor oral health, as assessed by total dental index and orthopantomography, was more common in the patients with cerebral infarction than in individuals of the control group.

Another cross-sectional study of 401 veterans showed that several dental and oral conditions were significantly associated with the diagnosis of a cerebral vascular accident when included in a multivariate logistic regression model with and without many of the known risk factors for cerebral vascular accident [21].

4) Infective Endocarditis:

Infective endocarditis is a bacterial infection of the heart valves or the endothelium of the heart. It occurs when bacteria in the bloodstream lodge on abnormal heart valves or damaged heart tissue. Endocarditis occurs rarely in people with normal hearts. However, people who have certain preexisting heart defects are at risk for developing endocarditis when a bacteremia occurs [25].

Infective endocarditis is a serious and often fatal systemic disease that has been associated with dental diseases and treatment. There are over 1,000 case reports associating dental procedures or disease with the onset of endocarditis [29]. Three controlled studies have recently been conducted, all showing an association of dental procedures with bacterial endocarditis [30]. In addition, multiple animal models (rats, rabbits, and pigs) have shown that oral bacteria and even dental extraction can create histologic evidence of endocarditis under experimental conditions [31]. It appears that dental procedures, especially extractions and possibly scaling, meet currently accepted epidemiological criteria for causation of endocarditis [32]. No other systemic diseases or conditions have been studied so extensively, although several other disorders may be linked to dental diseases.

Drangsholt [29], searched the world biomedical literature from 1930 to 1996 and concluded that the incidence of infective endocarditis varies between 0.70 and 6.8 per 100,000 person-years; over 50% of all infective endocarditis cases are not associated with either an obvious procedural or infectious event 3 months prior to developing symptoms; 8% of all infective endocarditis cases are associated with periodontal or dental disease without a dental procedure; the risk of infective endocarditis after a dental procedure is probably in the range of 1 per 3,000 to 5,000 procedures; and over 80% of all infective endocarditis cases are acquired in the community, and the associated bacteria are part of the host's endogenous flora. A new causal model of dental disease- and procedure-associated endocarditis has been proposed [29] that involves early and late bacteremia. The early bacteremia may "prime" the endothelial surface of the heart valves over many years and promote early valve thickening. This renders the valves susceptible to late adherence and colonization with bacteria. The late bacteremia may work over days to weeks and allows bacterial adherence and colonization of the valve, resulting in the characteristic fulminant infection.

5) Bacterial Pneumonia:

Pneumonia is an infection of pulmonary parenchyma caused by a wide variety of infectious agents, including bacteria, fungi, parasites, and viruses. Pneumonia can be a life-threatening infection, especially in the old and immunocompromised patient, and is a significant cause of morbidity and mortality in patients of all ages. Total pneumonia mortality in low-risk individuals over 65 years of age is 9 per 100,000 (0.009%), whereas in high-risk individuals who are likely to aspirate, the mortality can be almost 1,000 per 100,000 (1%) or higher [33]. Pneumonias can be broadly divided into two types, community acquired and hospital acquired (nosocomial). These types of pneumonia differ in their causative agents.

The lung is composed of numerous units formed by the progressive branching of the airways. The lower respiratory tracts are normally sterile, despite the fact that secretions from upper respiratory tracts are heavily contaminated with microorganisms from the oral and nasal surfaces. Sterility in the lower respiratory tract is maintained by intact cough reflexes, by the action of tracheobronchial secretions, by mucociliary transport of inhaled microorganisms and particulate material from the lower respiratory tract to the oropharynx, and by immune and nonimmune defense factors [34,35,36]. The defense factors are present in a secretion which also contains surfactant and other proteins such as fibronectin, complement, and immunoglobulins, which coat the pulmonary epithelium. The lung also contains a rich system of resident phagocytic cells which remove microorganisms and particulate debris [36].

Microorganisms can infect the lower respiratory tracts by four possible routes: aspiration of oropharyngeal content [37], inhalation of infectious aerosols [38], spread of infection from contiguous sites [35], and hematogenous spread from extrapulmonary sites of infection [39].

Most commonly, bacterial pneumonia results from aspiration of oropharyngeal flora into the lower respiratory tract, failure of host defense mechanisms to eliminate them, multiplication of the microorganisms, and subsequent tissue destruction [40]. It is likely that most pathogens first colonize the surfaces of the oral cavity or pharyngeal mucosa before aspiration [40]. These pathogens can colonize from an exogenous source or emerge following overgrowth of the normal oral flora after antibiotic treatment. Common potential respiratory pathogens (PRPs) such as Streptococcus pneumoniae, Mycoplasma pneumoniae, and Haemophilus influenzae can colonize the oropharynx and be aspirated into the lower airways. Other species thought to comprise the normal oral flora, including A. actinomycetemcomitans and anaerobes such as P. gingivalis and Fusobacterium species, can also be aspirated into the lower airways and cause pneumonia [36].

Generally accepted risk factors that predispose an individual to nosocomial pneumonia include the presence of underlying diseases such as chronic lung disease, congestive heart failure, or diabetes mellitus; age >70 years; mechanical ventilation or intubation; a history of smoking; previous antibiotic treatment; immunosuppression; a long preoperative stay; and prolonged surgical procedures [41,38].

Pneumonia can result from infection by anaerobic bacteria. Dental plaque would seem to be a logical source of these bacteria, especially in patients with periodontal disease. Such patients harbor a large number of subgingival bacteria, particularly anaerobic species. Among the oral bacterial species implicated in pneumonia are A. actinomycetemcomitans [42], Actinomyces israelii [43], Capnocytophaga species [44], Eikenella corrodens [45], Prevotella intermedia, and Streptococcus constellatus [46].

There are several proposed mechanisms to explain the propensity for PRPs to colonize the oropharynx of susceptible patients. First, compromised individuals such as diabetics and alcoholics may be prone to oropharyngeal colonization by PRPs [36]. These individuals are thought to be more likely to aspirate and are also known to be at greater risk of periodontal disease [47]. Thus, the extensive dental plaque of these subjects may provide surfaces to which PRPs might adhere to provide a reservoir for infection to distal portions of the respiratory tract [48].

Second, the oral surface of subjects at high risk for pneumonia, such as hospitalized patients, may somehow become modified to provide receptors for the adhesion of PRPs [36]. Poor oral hygiene increases the plaque load and therefore the level of hydrolytic enzymes in saliva. The source of these enzymes has been attributed to plaque bacteria [49] or polymorphonuclear leukocytes, which enter the saliva through the inflamed gingival sulcus [50]. These proteolytic enzymes may alter the characteristics of the mucosal surfaces, resulting in increased colonization by pathogenic bacteria [51].

Limeback [52] noted a relationship between poor oral hygiene and aspiration pneumonia among elderly residents of chronic care facilities. He subsequently found that the nursing homes with the least number of dental visits had the most deaths due to pneumonia.

A study by Scannapieco et al. [53] has shown that individuals with respiratory disease (n = 41) have significantly higher oral hygiene index scores than subjects without respiratory disease (n = 193; P = 0.044). Logistic regression analysis of data from these subjects, which considers age, race, gender, smoking status, and simplified oral hygiene index (OHI), suggests that subjects having the median OHI value are 1.3 times more likely to have a respiratory disease than those with an OHI of 0. (OHI is a composite index which scores debris and calculus deposition on tooth surfaces.) Similarly, subjects with the maximum OHI value are 4.5 times more likely to have a chronic respiratory disease than those with an OHI of 0.

Loesche and Lopatin [21] have studied oral and dental conditions in over 350 elderly individuals that may predispose individuals for aspiration pneumonia. They used the periodontal disease score as the outcome and compared the upper tertile of the periodontal disease score with the lower tertiles. The individuals with "definite" aspiration pneumonia were 3.3 times more likely to have a higher periodontal disease score (95% CI = 1.06 to 10.3; P = 0.05) than the individuals without pneumonia. The odds ratio pattern and wide CIs suggest that an important association exists between poor periodontal status and aspiration pneumonia.

6) Low Birth Weight:

Pregnancy can influence gingival health. Changes in hormone levels during pregnancy promote an inflammation termed pregnancy gingivitis [54]. This type of gingivitis may occur without changes in plaque levels [55]. Oral contraceptives may also produce changes in gingival health. Some birth control pill users have a high gingival inflammation level but a low plaque level. Birth control pills may cause changes such as alteration of the microvasculature, gingival permeability, and increased synthesis of estrogen PGs [56].

Oral infections also seem to increase the risk for or contribute to low birth weight in newborns. Low birth weight, defined as a birth weight of <2,500 g, is a major public health problem in both developed and developing countries. The incidence of preterm delivery and low birth weight has not decreased significantly over the last decade and remains at about 10% of all live births in the United States [57].

Low birth weight in preterm infants remains a significant cause of perinatal morbidity and mortality. Compared to normal-birth-weight infants, low-birth-weight infants are more likely to die during the neonatal period [58], and low-birth-weight survivors face neurodevelopment disturbances [59], respiratory problems [60], and congenital anomalies [61]. They also demonstrate more behavioral abnormalities as preschoolers [62] and may have attention deficit hyperactivity disorder [63]. For low birth weight, all these factors need further elucidation.

Risk factors for preterm low-birth-weight infants include older (>34 years) and younger (<17 years) maternal age, African-American ancestry, low socioeconomic status, inadequate prenatal care, drug, alcohol, and/or tobacco abuse, hypertension, genitourinary tract infection, diabetes, and multiple pregnancies. Although increasing efforts have been made to diminish the effects of these risk factors through preventive interventions during prenatal care, they have not reduced the frequency of preterm low-birth-weight infants [64].

Evidence of increased rates of amniotic fluid infection, chorioamnion infection, and chorioamnionitis supports an association between preterm birth or low birth weight and infection during pregnancy [57]. Histologically, the chorioamnion is often inflamed, even in the absence of any bacterial infection in the vagina (vaginosis) or cervical area. This suggests that distant sites of infection or sepsis may be targeting the placental membranes. Vaginosis, caused by gram-negative, anaerobic bacteria, is a significant risk factor for prematurity and is usually associated with the smallest, most premature neonatal deliveries [65]. The biological mechanisms involve bacterially induced activation of cell-mediated immunity leading to cytokine production and the ensuing synthesis and release of prostaglandins (PG), which appears to trigger preterm labor [65]. Elevated levels of cytokines (IL-1, IL-6, and TNF-) have been found in the amniotic fluid of patients in preterm labor with amniotic fluid infection [66]. These cytokines are all potent inducers of both PG synthesis and labor. Intra-amnionic levels of PGE2 and TNF- rise steadily throughout pregnancy until a critical threshold is reached to induce labor, cervical dilation, and delivery [64].

As a remote gram-negative infection, periodontal disease may have the potential to affect pregnancy outcome. During pregnancy, the ratio of anaerobic gram-negative bacterial species to aerobic species increases in dental plaque in the second trimester [55]. The gram-negative bacteria associated with progressive disease can produce a variety of bioactive molecules that can directly affect the host. One microbial component, LPS, can activate macrophages and other cells to synthesize and secrete a wide array of molecules, including the cytokines IL-1 , TNF- , IL-6, and PGE2 and matrix metalloproteinases [57]. If they escape into the general circulation and cross the placental barrier, they could augment the physiologic levels of PGE2 and TNF- in the amniotic fluid and induce premature labor.

Human case-control studies have demonstrated that women who have low-birth-weight infants as a consequence of either preterm labor or premature rupture of membranes tend to have more severe periodontal disease than mothers with normal-birth-weight infants [57].

A case-control study of 124 pregnant or postpartum mothers was performed, using mothers with normal-birth-weight babies as controls [64]. Assessments included a broad range of known obstetric risk factors, such as tobacco and drug use, alcohol consumption, level of prenatal care, parity, genitourinary infections, and nutrition. Each subject received a periodontal examination to determine the clinical attachment level. Mothers of preterm low-birth-weight infants and primiparous mothers of preterm low-birth-weight infants (n = 93) had significantly worse periodontal disease than the respective mothers of normal-birth-weight infants (controls). Multivariate logistic regression models, controlling for other risk factors and covariates, demonstrated that periodontal disease is a statistically significant risk factor for preterm low birth weight, with adjusted odds ratios of 7.9

and 7.5 for all preterm low-birth-weight cases and primiparous preterm low-birth-weight cases, respectively. These data indicate that periodontal disease represents a previously unrecognized and clinically significant risk factor for preterm low birth weight as a consequence of either preterm labor or premature rupture of membranes.

In another 1:1 matched case-control study (55 pairs), the hypothesis that poor oral health of the pregnant woman is a risk factor for low birth weight was evaluated [67]. The effect of the periodontal and dental caries status of the woman on the birth weight of the infant was evaluated at the time of delivery by conditional logistic regression analysis, while controlling for known risk factors for low birth weight. Mothers of low-birth-weight infants are shorter, less educated, and married to men of lower occupational class, have fewer areas of healthy gingiva and more areas with bleeding and calculus, and gain less weight during the pregnancy. Conditional logistic regression analyses indicate that mothers with more healthy areas of gingiva (odds ratio [OR] = 0.3, 95% CI = 0.12 to 0.72) and those who are taller (OR = 0.86, 95% CI = 0.75 to 0.98)

have a lower risk of giving birth to a low-birth-weight infant. The authors conclude that poor periodontal health of the mother is a potential independent risk factor for low birth weight.

In a recent case-control study, 48 case-control subjects had their gingival crevicular fluid (GCF) levels of PGE2 and IL-1 measured to determine whether mediator levels are related to current pregnancy outcome [68]. In addition, the levels of four periodontal pathogens were measured by using microbe-specific DNA probes. The results indicate that GCF PGE2 levels are significantly higher in mothers of preterm low-birth-weight infants than in mothers of normal-birth-weight infants (controls) (131.4 ± 21.8 versus 62.6 ± 10.3 ng/ml [mean ± standard error], respectively, at P = 0.02). Furthermore, among the primiparous mothers of preterm low-birth weight infants, there is a significant inverse association between birth weight (as well as gestational age) and GCF PGE2 levels at P = 0.023. These data suggest a dose-response relationship for increased GCF PGE2 as a marker of current periodontal disease activity and decreasing birth weight. Four organisms associated with mature plaque and progressing periodontitis, Bacteroides forsythus, P. gingivalis, A. actinomycetemcomitans, and Treponema denticola, are detected at higher levels in mothers of preterm low-birth-weight infants than in controls. These data suggest that biochemical measures of maternal periodontal status and oral microbial burden are associated with preterm birth and low birth weight.

Offenbacher et al. [64] concluded that 18.2% of preterm low-birth-weight babies may result from periodontal disease a previously unrecognized and clinically important risk factor for preterm birth and low birth weight.

However, it should be noted that periodontal disease pathogens are necessary but not sufficient for periodontal disease expression. The role of the host's inflammatory response appears to be the critical determinant of susceptibility and severity [69]. The association between periodontal disease and low birth weight may reflect the patient's altered immune-inflammatory trait that places the patient at risk for both conditions. Thus, periodontitis may be a marker for preterm delivery susceptibility as well as a potential risk factor. Indeed, the data from animal models suggest that even if periodontal disease is not the primary cause of prematurity, in a subset of patients it may serve as a contributor to the morbidity of the condition.

7) Diabetes Mellitus:

Diabetes mellitus is a clinical syndrome characterized by hyperglycemia due to an absolute or relative deficiency of insulin. It affects more than 12 million people in the United States. Diabetes mellitus is characterized by metabolic abnormalities and long-term complications involving the eyes, kidneys, nervous system, vasculature, and periodontium [70]. Diabetes is commonly categorized as type 1, or insulin dependent, and type 2, non-insulin dependent. The fundamental derangement in insulin-dependent diabetes is the hypoproduction of insulin due to destruction of the beta cells of the pancreas. In non-insulin-dependent diabetes, the derangement involves resistance of target tissue to insulin action [1].

Although the precise etiology is still uncertain in both main types of primary diabetes, environmental factors interact with a genetic susceptibility to determine which of those with the genetic predisposition actually develop the clinical syndrome and the timing of its onset. Environmental factors in insulin-dependent diabetes include virus, diet, immunological factors, and pancreas disease. In non-insulin-dependent diabetes, environmental factors such as lifestyle, age, pregnancy, pancreas pathology, and insulin secretion and resistance are included [71].

Severe periodontal disease often coexists with severe diabetes mellitus. Diabetes is a risk factor for severe periodontal disease. The converse possibility that periodontal disease either predisposes or exacerbates the diabetic condition has received more and more attention. Recently, a new model was presented by Grossi and Genco [72], in which severe periodontal disease increases the severity of diabetes mellitus and complicates metabolic control. They propose that an infection-mediated upregulation cycle of cytokine synthesis and secretion by chronic stimulus from LPS and products of periodontopathic organisms may amplify the magnitude of the advanced glycation end product (AGE)-mediated cytokine response that is operative in diabetes mellitus. The combination of these two pathways, infection and AGE-mediated cytokine upregulation, helps explain the increase in tissue destruction seen in diabetic periodontitis and how periodontal infection may complicate the severity of diabetes and the degree of metabolic control, resulting in a two-way relationship between diabetes mellitus and periodontal disease or infection.

It is well established that diabetics are more likely to develop periodontal disease than nondiabetics [73] and that the disease severity is related to the duration of diabetes [74]. One plausible biologic mechanism for why diabetics have more severe periodontal disease is that glucose-mediated AGE accumulation affects the migration and phagocytic activity of mononuclear and polymorphonuclear phagocytic cells, resulting in the establishment of a more pathogenic sub gingival flora. The maturation and gradual transformation of the sub gingival micro flora into an essentially gram-negative flora will in turn constitute, via the ulcerated pocket epithelium, a chronic source of systemic challenge. This in turn triggers both an "infection-mediated" pathway of cytokine upregulation, especially with secretion of TNF- and IL-1, and a state of insulin resistance, affecting glucose-utilizing pathways. The interaction of mononuclear phagocytes with AGE-modified proteins induces upregulation of cytokine expression and induction of oxidative stress. Thus, monocytes in diabetic individuals may be "primed" by AGE-protein binding. Periodontal infection challenge to these primed phagocytic cells may, in turn, amplify the magnitude of the macrophage response to AGE-protein, enhancing cytokine production and oxidative stress. Simultaneously, periodontal infection may induce a chronic state of insulin resistance, contributing to the cycle of hyperglycemia, nonenzymatic irreversible glycation, and AGE-protein binding and accumulation, amplifying the classical pathway of diabetic connective tissue degradation, destruction, and proliferation. Hence, the relationship between diabetes mellitus and periodontal disease or infection becomes two way. A self-feeding two-way system of catabolic response and tissue destruction ensues, resulting in more severe periodontal disease and increased difficulty in controlling blood sugar [72].

Certain metabolic end products such as glycated hemoglobin are thought to contribute to the degenerative retinal and arterial changes commonly found in diabetic subjects. The concentration of glycated hemoglobin in serum is a direct function of the time that hemoglobin is exposed to elevated glucose levels [1]. A longitudinal study of diabetes and periodontal disease has been carried out in the Pima tribe, an Indian population in the United States having a prevalence of non-insulin-dependent diabetes of about 50%. This is the highest reported prevalence of non-insulin-dependent diabetes in the world [75]. Poor glycemia control was defined as the occurrence of glycated hemoglobin of 9% or more at follow-up. The results indicated that severe periodontitis at baseline is associated with increased risk of poor glycemic control at follow-up two or more years later. These findings suggest that severe periodontitis may be an important risk factor in the progression of diabetes, and control of periodontal infection is essential to achieve long-term control of diabetes mellitus. Grossi and Genco [72] reexamined the studies that addressed the effect of periodontal treatment on metabolic control of diabetes mellitus [76,77]. Six of these studies included type 1 patients, and two studies [76,77] included type 2 patients. Periodontal treatment was divided into two groups, mechanical treatment only and with systemic antibiotics as an adjunct to mechanical treatment. The results show that the effect of periodontal treatment on diabetic metabolic control is dependent on the mode of therapy. When mechanical periodontal treatment alone is provided, regardless of the severity of periodontal disease or degree of diabetes control, the treatment outcome is strict improvement in periodontal status or a local effect. On the contrary, when systemic antibiotics are included with mechanical therapy, an improvement in diabetes control, measured as a reduction in glycated hemoglobin or reduction in insulin requirements, is achieved. Therefore, one may propose that control of the chronic gram-negative periodontal infection should be part of the standard treatment of the diabetic patient.

CONCLUSION:

A large number of studies have suggested that oral infection, especially periodontitis, are a potential contributing factor to a variety of clinically important systemic diseases. Endocarditis has been studied most extensively. It appears that dental procedures and oral infection meet currently accepted epidemiological criteria for causation of endocarditis. However, there is still not sufficient evidence to claim a causal association between oral infection and other systemic diseases.

Epidemiological research (cross-sectional and longitudinal studies) can identify relationships but not causation. If some types of periodontal disease merely constitute an oral component of a systemic disorder or have etiological features in common with systemic diseases, periodontal and systemic diseases might frequently occur together without having a cause-effect relationship.

Therefore, further research must be done before the potential for oral infections to cause damage in other sites of the body can definitely be established.

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Review Article

DYNESTHETIC INTERPRETATION OF ESTHETICS IN COMPLETE DENTURE

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Abstract

Function & esthetics are inseperable & interdependent factors of prosthodontic success.The development & use of adequate function is the first step followed by final refinement of prosthodontic service i.e. the dentogenic phase. The dynesthetic and dentogenic concept, when applied, provides a more natural, harmonious prosthesis, which not only is desired by patients, but also is a quality of care they deserve. Outstanding esthetics can be achieved by simple guidelines, using tooth molds specifically sculpted for males and females, arranging prosthetic teeth to correspond with personality and age and sculpting the matrix (visible denture base) with more natural contours. There is no reason for edentulous individuals to be provided with care of any less quality than that available with other procedures, such as crowns, bridges, veneers, or implant restorations.

Key words
Esthetics, Dentogenic Concept, Dynesthetic Concept

INTRODUCTION

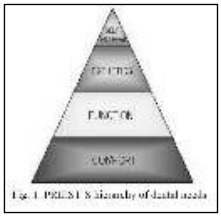
Webster's third new international dictionary defines "esthetic" as appreciative of,responsive to or zealous about the beautiful; having a sense Of beauty or fine culture".It is truly said that "beauty lies in the eyes of the beholder".

But to look at the patient & interpret him in the denture & make him look beautiful to other people is the real challenge in front of the dentist. The branch of dentistry dealing with the ways of achieving life-like results is esthetic dentistry.

Esthetic dentistry can be defined "as the art and science of dentistry applied to create or enhance beauty of an individual within functional and physiological limits." It is the art of dentistry in its purest form. The purpose is to use function as the foundation of esthetics

Cosmetic dentistry is application of the principles of esthetics and certain illusionary principles, performed to signify or enhance beauty of an individual to suit the role he has to play in his day-to-day life or otherwise

Dr. PRIEST gave a hierarchy of dental needs (fig. 1).



According to him, there are basically four levels of patients needs - first comfort, then function followed by esthetics & lastly self esteem. He says that a person cannot rise to the upper level until the lower level is achieved i.e. if the patient is not comfortable with his prosthesis, he cannot be satisfied with its function. Once satisfaction with comfort & function is achieved, the patient becomes conscious about esthetics & once all three are acceptable to the patient; the prosthesis definitely helps in increasing his self esteem.

PERSONALITY IN TREATMENT PLANNING

BAKER & SMITH in 1939 classified personalities into:

Group I: are well adjusted individuals. These patients are successful in their life & career & want treatment for esthetic reasons. Ideal patients.

Group II: are people with unassertive or inadequate personalities who use their disfigurement as a shield & unconscious defense. A subset consists of passive people who are grateful for any assistance or aid given.

Group III: are prepsychotic or psychotic people for whom the facial abnormality was focal point of deviant personality. Any esthetic correction serves only to disrupt rationalization process

Acc. to LEVIN (1988)

Driven: focuses on results, time conservation oriented.

Expressive: wants to feel good, highly emotional

Amiable: reacts poorly to pressure, emotional, fears consequences

Analytical: requires endless details, highly exacting

ESTHETICS IN DENTURES

"DENTURE ESTHETICS" is defined as the

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cosmetic effect produced by a dental prosthesis which affects the desirable beauty, attractiveness, character and dignity of the individual.

FRUSH & FISHER in 1956 gave dentogenic concept & its dynesthetic interpretation to give a more natural & individual appearance to the dentures of a patient.
FISHER states that "Utilize the approach of an artist while analyzing the patient first as to sex, i.e. Male or female, then as to personality i.e. Vigorous or delicate, & then as to age, i.e. young, middle aged or old."

The original concepts of these important considerations belong to WILHEHN ZECH, a sculptor in Switzerland. He observed that "it was possible for a sculptor to express vigor & delicacy in his arts of work. So, it should be possible for the dentist also to do so in his artificial denture." This was an integral part of thinking that led to the whole concept of "DENTOGENIC RESTORATIONS".

The Dentogenic theory of esthetics is a basic esthetic concept for all phases of dentistry where appearance is a factor. It is explained as the prosthodontic appearance interpretation of three vital factors which every patient possesses: sex, personality & age, Hence, also known as SPA concept.

The word "dynesthetic" uses the word "dynamics" as applied to the fine art of producing 'life-effect' in a denture. Dynesthetics are the secondary factors of dentogenic restoration. The dynesthetic and dentogenic concept, when applied, provides a more natural, harmonious prosthesis, which not only is desired by patients, but also is a quality of care they deserve. Outstanding esthetics can be achieved by using these simple guidelines.

DYNESTHETIC INTERPRETATION OF DENTOGENIC CONCEPT:

To apply the dynesthetic techniques in prosthodontics requires knowledge of dentogenic concept.The selection of artificial teeth, their subsequent sculpturing, the individual & detailed positions of these teeth, & the color & contours of the denture base are all parts of the dynesthetic concept & can be done in a comprehensive manner with reference to patient's age, sex & personality as primary factors.

In other words, the dentogenic concept is basically how we perceive a patient's appearance & the procedures carried out to make that appearance a reality is dynesthetic concept.

The dynesthetic techniques are rules which concern three important divisions of denture fabrication: The tooth, its position & its matrix (visible denture base). The dynesthetic techniques that can be accomplished by the technician in the laboratory as supplementary help to dentist:

Physiologic tooth selection:

Our purpose is to create an illusion of natural dentition. Hence, artificial tooth shades should be selected according to physiologic color changes seen in progressively aging, undiseased natural teeth.

For e.g.: the Young have a solid & opaque color with little or no color texture. Therefore, select teeth with blue incisal edge & a yellowish unmarred body. On the other hand, older people due to wearing of mamelons & exposure of dentino-enamel junction exhibit more of grey tinge instead of blue. So, Slightly discolored teeth or teeth with changed color texture can be used. But that does not necessarily mean darker teeth. The old can have lighter shaded teeth than the young. It basically is influenced by the habits & personal grooming of the patient.

Special considerations can be given to smokers. For such cases canary yellow, pikgray, gray-brown etc., shades of teeth are available. Tobacco stains can also be incorporated in patient having a long standing tobacco chewing habit. Special shades can be used to depict for fixed, removable partial dentures, restorations etc. to give a more natural appearance to the denture.

Mold selection:

The selection of an acceptable personality mold involves its subsequent treatment for abrasion, erosion, depth grinding, shaping & polishing. (fig. 2)

The progressive abrasion of the teeth occurs with age of the person. In artificial teeth it can be depicted by making an oblique cut at incisal edge.
Erosion also increases with age & can be duplicated by grinding & polishing the artificial tooth. It should be achieved to a lesser degree in anterior teeth than posteriors.

Depth grinding gives the feeling of depth to a tooth, the third dimension, for realism. It gives the feel of sex & personality to the denture. It is done on the mesial surface of the central incisor. The mesio-labial line angle of the central incisor is ground in a flat cut, following the same curve as the mesial contour of the tooth on order to move the deepest visible point of the tooth further lingually. This is followed by careful rounding, smoothening & polishing. A flat, thin, narrow tooth is delicate looking while a thick, heavily carved tooth (severe depth grinding) is symbolic of maleness.

Midline:

The facial midline serves to evaluate the location and axis of the dental midline and the medio-lateral discrepancies in tooth position. The dental midline, if perpendicular to the inter-pupillary line and coinciding with the bridge of the nose and the philtrum, produces an attractive orientation of the smile.

The features of the face usually slant one way or another. Hence, eccentric midline if not too exaggerated is acceptable & may lead to illusion of natural dentition. But it should always be vertical or with slight labial inclination to incisal & occlusal plane.

Speaking line:

It is the incisal length or the vertical composition of the anterior teeth. The final evaluation of incisal length is made when the patient is speaking. While speaking seriously, the tip of lateral incisors should be seen.
As for central incisors, with lips at rest – Young woman = 3 mm, Young man = 2mm,
Middle age = 1.5mm, Old age = 0 – 2mm should be seen.

Smiling line:

Smile line is a curve whose path follows the incisal edges of central incisors up & back to the incisal edges of lateral incisors & thence to the tips of the cuspids.
It is very important as is the primary factor of esthetics. In females it

follows the curve of lower lip while in males the lateral incisors are above the centrals & the cuspids are arranged at the level of central incisors. Smile line flattens with age resulting in a straight incisal plane which is said to have a "gull wing appearance". (fig. 3)

CENTRAL INCISORS POSITION

Dominance is an important physical attribute of dento-facial composition. It exists when a strong centralized structure is surrounded by well demarcated, characterized structures. This role is played by the central incisors as they are the first teeth to be seen. Their placement controls – Midline, Speaking line & Smiling line composition, Lip support & Labioversion.

They are the basis of personality mold selection. Their shape depicts the personality & position determines the strength & action of the dentogenic composition.

They can be placed in three different ways to give a vigorous personality to the denture: by placing the cervical end of one incisor out, by placing one central incisor bodily ahead of the other or by combined rotation of two central incisors with the distal surface forward with one incisor depressed at cervical end & the other depressed incisally.

They must contrast sharply in size with lateral incisors. This can be achieved by either selecting a larger sized central incisors & canines or by taking the lateral incisors from the smaller sized mold.

They must be depth ground for the feeling of depth & hence the life likes appearance of the dentures.

LATERAL INCISOR POSITION:

The position is subordinated in importance to that of central incisors. In dentogenics, it is basically the sex determining tooth.

Its rotation will harden (masculine) or soften (feminine) the composition. The lateral incisors rotated to show its mesial surface, whether overlapping centrals or not, gives softness or youthful coquettishness to the smile.

This effect can also be achieved by rounding or squaring the edges. Squaring of incisal edges leads to a masculine appearance.

They should have asymmetric long axis & the tips should be visible when speaking seriously.

Cuspid position:

The position of the cuspid is important as it supports the anterior arch form at its widest part & also controls the size of buccal corridor.

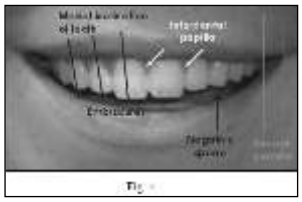
They should be carefully positioned so as to dominate the lateral incisors & complete the upward curve of smile line. The three basic requirements for their placement are that they should be rotated to show mesial surface with their cervical end out & the long axis should be vertical when observed from the side.

They represent the age factor of dentogenic restorations. The cuspid tip should be artistically ground (in an curvature) so as to imply abrasion against opposing teeth.Hence, amount of grinding done is dependent on the age of the patient but it should always follow a curve.

Their arrangement is also helpful in depiction of sex of the patient. If they are positioned such that the smile line follows the curve of lower lip, then the composition appears feminine. Also, prominent cuspids that are rotated to show about 2/3rd of the labial surface are symbolic of male characters while in females only mesial 1/3rd is visible.

Long axis of teeth:

Each combination of tooth inclinations in a smile is unique. The long axis of the natural teeth varies, although in very minute degrees. The direction of the anterior teeth in an esthetic smile shows the medial tipping of the axial inclinations which increases as one moves further from the midline.(Fig. 4)



This progression, therefore, should be exaggerated in the dentogenic restoration as an artistic device.

EMBRASURES

The pattern of silhouetting created by the edges and separations between the maxillary anterior teeth against the darker background of the mouth helps define a good-looking smile. It represents a divergence of the proximal surfaces of the anterior teeth from the contact point. These spaces between the edges of the teeth known as embrasure spaces follow a pattern that develops between the central incisors and then progress laterally. The size and volume of the incisal embrasures between teeth increase as the dentition progresses away from the midline. In other words, the incisal embrasure space between the lateral incisor and the central incisor should be larger than the incisal embrasure between the central incisors & so on. (Fig. 4)

INTERDENTAL PAPILLA

It forms the main part of tooth matrix. This is the part of the denture base that is visible when the patient speaks or laughs.

If correctly formed, they accomplish four purposes i.e. they create a hygienic interdental area, they determine the outline form of the tooth, they act as a complimentary factor in age interpretation, & they also bring a degree of color reflection to the interdental area which creates the illusion of natural dental composition.

The papilla must extend to the point of tooth contact & must be convex in all directions to make them self cleansing & hygienic. Also they must be of various lengths to give a more natural appearance to the prosthesis.(Fig. 4)

The tip of the papilla at its lowest point must terminate at the juncture of the labial face & lingual face of the tooth. It should never slope inward to terminate toward the lingual portion of the proximal surface rather turns upwards & back to form the bottom of the groove which is known as lingual cutaway.

The papilla must be shaped according to the age of the patient. In young people have papilla that are at the contact point, finely stippled, pointed & tight against the tooth while in the old age the interdental papilla are convex, rounded & shortened due to recession. The middle aged lies somewhere in between.

LINGUAL CUTAWAY

It is a groove in interdental area which begins at contact point if the teeth are together or at the tip of the interdental papilla if a diastema is present. It widens & deepens according to natural divergence of lingual proximal tooth surfaces. It fades away into palatal surface of the denture.

While incising, this polished channel helps in sweeping the food & keeping the area clean.

BUCCAL CORRIDOR

It is the space created between the buccal surface of the posterior teeth & the corner of the lip when the patient smiles. It brings about a harmonious cohesion between the various elements of the smile. (Fig. 4). Its size & shape are controlled by the position & slant of the cuspid. It helps by preventing the "sixty tooth smile" or "molar to molar smile" which is often characteristic of a denture.

Obliteration of these essential spaces by dental elements like bulky canines, wide arches or over-contoured restorations can lead to an unattractive smile. Excessive buccal corridor seen in cases of missing premolars or palatally placed posteriors and a constricted arch also appear unaesthetic.

SPACES

Spaces between teeth are highly effective but their size & position must be artistically & hygienically formed.

Diastemas should always be placed asymmetrically on either side of the dental arch & a midline diastema is to be avoided as it appears unesthetic. They should be Vshaped to shed food. When placed between posterior teeth allows for additional spillways for food & creates additional cutting edges from the marginal ridges.

The width of the diastema should be such that it is inconspicuous in a denture.Spaces which are too wide appear as black holes. Conversely, too narrow spaces are not hygienic.

GUM LINE

Esthetic conditions related to gingival health and appearance are an essential component of effective smile design. Inflamed, uneven gingival lines detract from a pleasing smile. Blunted papilla and asymmetric gingival crests become part of the overall esthetic picture. (fig. 5)

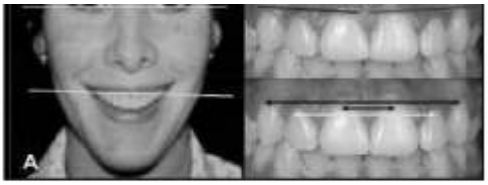


Fig. 5: a) slight canting of gum line
b) gingival zenith
c) gum line symmetric on both sides
d) lateral incisor line is lower than central & canine

The gum line at the cervical ends of the teeth should be parallel to intepupillary line for most pleasing appearance, although slight canting in maxillary teeth is allowed for more natural appearance.

The gum line should be symmetrical on both sides of the midline with the gum line over the lateral incisor below a straight line drawn from central to canine gum lines

The gum line should follow the concept of GINGIVAL ZENITH: i.e. The height of contour of gingival margin is slightly distal to long axis in maxillary central incisor, on the axis in lateral & slightly distal in case of canines.

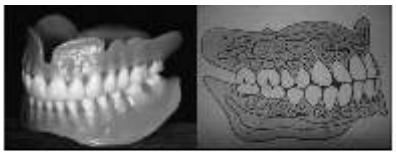
DENTURE BASE CONTOURS

The denture base contour is convex, vertically from the denture base border to the tip of interdental papilla in the anterior region. Denture characterization is defined as "the modification of the form & color of denture base & the teeth to produce a more life-like appearance."

Denture base can be divided into two portions: matrix (visible portion of the base) & the nonvisible part. The nonvisible part should provide a smooth & self-cleaning surface. The matrix on the other hand requires special attention & should have over accentuated characterization to give a more natural appearance to the denture. Some interpretation of age, sex & personality should be made in gum matrix. Denture base characterization can be achieved by:

Stippling: (fig.6) In natural dentition, the papillae and marginal gingiva are smooth, but the band of attached gingiva in between has a stippled or orange-peel appearance. It is less prominent in old age. This creation of minute pore-like depressions on the attached gingiva portion of denture base to create this orange peel appearance is known as stippling.
Stippling of the areas representing the attached gingiva may be accomplished in a variety of ways: positive stippling & negative stippling. Positive stippling is achieved by blow-wax technique while negative stippling is done by making pores in the wax pattern using a tooth brush or Robinson bristle brush. The result of positive stippling is more natural looking which seems to collect less debris and calculus, and is easier to clean than the indentations made by negative stippling techniques.

Festooning: (fig.6) The contour of the gingiva presents a festooned appearance with intermittent elongated prominences corresponding



to the root contours. It is recommended that casts from patients with natural teeth be used as guides for gingival waxing and festooning. Without festooning and stippling, light is not randomly reflected and the denture becomes a smooth, pink mirror appearing artificial & lifeless.
Tinting & staining of base: The soft tissue shade guide is used to select a denture base material.The same shade guide can also be used to select other tissue colors and unusual characteristics, i.e., blotches of melanin, heamangiomas etc. Characterization of artificial teeth with stained incisors, cracks or simulated restorations may also be

recorded. This is done at the appointment when the artificial teeth are selected. A simple method of charting is needed to record these observations. The denture tinting chart may be reproduced and used to provide instructions for the dental laboratory. Denture staining can be done before or after the curing of the dentures.

CONCLUSION:

Denture esthetics has been discussed in terms of the anatomic-physiologic and artistic phases. A dentist must learn to see and try to understand the laws of physics, physiology, and psychology governing the perception of natural teeth & must apply the same principles to reproduce life-like teeth substitutes. On the other hand, the technician should carefully follow all the laid down guidelines & help the dentist in achieving the primary objective of patient satisfaction.

The changing trend in field of esthetic dentistry will reorient the art element involved in esthetic dental creations but its established scientific guidelines will always provide the sound basis for this change. By using these principles, the right smile can be created, one that reflects personality and character of the patient with all the benefits to health, spirit and mind that comes from confidence.

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Review Article

OBESITY AND PERIODONTAL DISEASE

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Abstract

Obesity is the most common nutritional disorder and is a significant risk factor for numerous adult diseases, and may be a factor in the incidence of periodontitis. Obesity has a significant association with periodontitis in terms of BMI, waist-to-hip circumference ratio, body fat, and maximum oxygen consumption. Chronic Inflammation has multidirectional link with periodontal disease, obesity and other chronic conditions.

Key words
Obesity, BMI, Periodontitis

INTRODUCTION

Periodontal disease is no longer identified as only an oral health problem but also a public health issue as it is associated with systemic health. Many mediators have been recognized for this relationship like chronic inflammation, infection and genetic predisposition.[1] Apart from these mediators, nutrition has been postulated as an alternative mediator.[2] Obesity, the most common nutritional disorder in America[3](Kopelman 2000) is a significant risk factor for numerous adult diseases, and may be a factor in the incidence of periodontitis. Obesity has a significant association with periodontitis in terms of BMI, waist-to-hip circumference ratio, body fat, and maximum oxygen consumption. The body mass index has always been considered a simple method for analysis of the nutritional status. These findings suggest that periodontitis may be aggravated by certain conditions associated with obesity for example, "the metabolic syndrome", a clustering of dyslipidemia and insulin resistance[5]

Obesity is well-known to be a significant risk factor for various adult diseases, such as type 2 diabetes, hyperlipidemia, hypertension, cholelithiasis, arteriosclerosis, and cardiovascular and cerebrovascular disease (Kopelman et al 2000). Obesity is also known to increase mortality from these and other health disorders. Among these obesity-related diseases, the risk of cardiovascular disease has been shown to be increased by periodontitis in some epidemiological studies.[6]

CLASSIFICATION

Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have an adverse effect on health leading to reduced life expectancy and health related problem.7It is defined by body mass index (BMI) and further evaluated in terms of fat distribution via the waist-hip ratio and total cardiovascular risk factor. Obesity in children and adolescents is defined not as an absolute number, but in relation to a historical normal group, such that obesity is a BMI greater than the 95th percentile.[8]

BMI	Classification
< 18.5	Underweight
18.5–24.9	Normal weight
25.0–29.9	Overweight
30.0–34.9	Class I obesity
35.0–39.9	Class II obesity
>40.0	Class III obesity

Metric: BMI = kilograms / meters2

As Asian populations develop negative health consequences at a lower BMI than Caucasians, some nations have redefined obesity; the Japanese have defined obesity as any BMI greater than 25 while China uses a BMI of greater than 28.[9]

WHR (waist to hip ratio) is used as a measurement of obesity, which in turn is a possible indicator of other more serious health conditions. A WHR of 0.7 for women and 0.9 for men have been shown to correlate strongly with general health.

Elevated waist circumference Men ‡ 40 inches (102 cm) Women ‡ 35 inches (88 cm)

PREVALENCE

The prevalence of periodontal disease is 76% higher among young obese (body mass index ‡ 30 kg/m2) individuals aged 18–34 years than in normal-weight individuals[10] and that weight is associated with increased risk of periodontitis among those aged 17–21 years [11].

ADIPOSE TISSUE

Adipose tissue is loose connective tissue composed of adipocytes. It is not only a passive triglyceride

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reservoir, but also produces high levels of cytokines and hormones, collectively called adipokines or adipocytokines [12], which may in turn affect the periodontal tissues. Some act locally and others via systemic circulation affect various body parts. Several studies have demonstrated a close involvement of adipokines (such as leptin, adiponectin and resistin) in inflammatory processes [13],[1]

Role of adipokines in inflammation

1.Hormone like proteins: Leptin and adiponectin

Leptin that plays a key role in regulating energy intake and energy expenditure, including appetite and metabolism. It is similar in some action with insulin. Most of patients suffering from obesity have leptin resistance. Leptin can elevate blood pressure and contribute to atherosclerosis and CVD[15] But leptin in relation to periodontitis related with obesity still need to be examined. Adiponectin is a protein hormone that modulates a number of metabolic processes, including glucose regulation and fatty acid catabolism.[16] there is inverse association between adiponectin and markers of inflammation. Low levels of adiponectin are associated with an increased risk of coronary artery disease and some other features related to metabolic syndrome.

2. Cytokines (TNF and IL-6)

Pro-inflammatory cytokines, such as tumor necrosis factor-a and interleukin- 6, may form a multidirectional link among periodontitis, obesity and other chronic diseases [17].It is mainly secreted from macrophages accumulated in adipose tissue. It is a member of a group of cytokines that stimulate the acute phase reaction. It has regulatory role in periodontal disease by stimulating Bone resorption, Collagen degradation, Activation of endothelial cell (ICAM/VCAM), Enhanced IL 8 production, Expression of MCP-1. It contributes to poor health by increasing insulin resistance and inducing C reactive protein and systemic inflammation.IL 6 is produced in greater quantity by deep abdominal fat. It is associated with Up regulation of acute phase protein (CRP/serum amyloid),Increase procoagulant activity of monocytes,Stimulation of LDL receptor gene in hepatocytes. Elevated levels are associated with increased risk of cardiovascular events.

3. Acute phase protein C reactive peptide

Elevated levels are associated with obesity and cardiovascular disease. It also predicts the risk of progression to type 2 diabetes mellitus. [18] CRP levels are associated with periodontal disease and levels respond to periodontal therapy

4. Proteins associated with vascular hemostasis: plasminogen activator inhibitor 1

It is regulatory protein of coagulation cascade. It acts by inhibiting fibrinolysis and extracellular matrix degradation. It contributes to obesity related complications like diabetes and coronary thrombi

5. Others

Increased levels of angiotensinogen which is secreted from adipose tissue are seen in obesity. It has vasoconstrictive effect and contributes to hypertension. Obesity is also associated with increased levels vascular endothelial growth factor which play role in hypertension and atherogenesis.

Obesity and periodontitis

A variety of potential mechanisms could explain an association between obesity and periodontitis.

1.Overweight young subjects may have unhealthy dietary patterns with insufficient micronutrients and excess sugar and fat content, and such dietary patterns may increase the risk for periodontal disease.[10]

2 Changes in host immunity and/or increased stress levels, which are often associated with gain of excess fat early in life, may also play a role. [11]

3.The underlying biological mechanisms for the association of obesity with periodontitis are not well established. However, adipose-tissue derived cytokines and hormones may play a role. [13]

4. Obesity may also influence periodontal disease status by increasing lipid and glucose blood levels , which may in turn have deleterious consequences for the host response by altering T cells and monocyte/macrophage/ macrophage function, as well as increasing cytokine production.[1]

Several recent studies have suggested a relationship between periodontal disease and obesity. In Saito's study of Japanese adults, increasing body mass index and waist: hip ratio was associated with increasing risk of periodontitis. Haffajee AD et al (2009)[20] concluded that an overgrowth of T. forsythia occurs in the subgingival biofilms of periodontally healthy, overweight and obese individuals that might put them at risk for initiation and progression of periodontitis and risk was significantly higher in obese periodontally healthy/gingivitis individuals. Kongstad J et al (2009)[21] reported that BMI may be inversely associated with clinical AL but positively related to BOP. Lundin et al. recently noted a correlation between tumor necrosis factor-a in the gingival crevice fluid and body mass index .[22] Mohammad Taghi Chitsazi et al demonstrated correlations between obesity, waist circumference, elevated CRP levels and severity of periodontitis.[23] Wood N et al (2003) found the significant correlations between body composition and periodontal disease (with WHR being the most significant, followed by BMI.[2]

Longitudinal studies with more precise measures of adiposity will provide better insights into the relationship between periodontal disease and obesity

Treatment

Weight loss therapy is recommended for patients with a body mass index of ‡30 and for patients with a body mass index of 25–29.9, or a high-risk waist circumference, and two or more risk factors.. Faster rates of weight loss are no more effective over the long term.

Behavioural therapy, serving as a useful adjunct to dietary therapy, includes self-monitoring stress management; problem-solving, contingency management, and social support. If lifestyle changes do not lead to weight loss in 6 months, pharmacotherapy should be considered. The two medications currently available for the treatment of obesity are sibutramine, and orlistat. A new drug on the horizon is rimonabant.

Weight loss surgery is recommended for well informed and motivated patients who have clinically severe obesity (body mass index 40) or a body mass index of 35 and serious comorbid conditions. Two types of operations are routinely performed: those that restrict gastric volume (banded gastroplasty) and those that, in addition to limiting food intake, also alter digestion (Roux-en-Y gastric bypass).

CONCLUSION

Obesity is a complex and multifactorial disease. Its relationship with periodontal disease and other chronic disease is well documented but underlying mechanism is under investigation. Chronic Inflammation has multidirectional link with periodontal disease, obesity and other chronic conditions. A periodontist can educate his patients about related information and can help in improving oral and overall health of patient.

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Review Article

EFFECT OF SEX HORMONES ON THE PERIODONTIUM

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Abstract

Sex hormones have long been considered to play an influential role on periodontal tissues, bone turnover rate, wound healing and periodontal disease progression. This review focuses on the effects of sex hormones on the periodontium. This review article will analyze how these hormones influence the periodontium at different life stages such as puberty, menstruation, pregnancy, menopause and post-menopause. Moreover, the effects of contraceptives and hormone replacement therapies on the periodontium will be discussed. It is clear that sex steroid hormones play significant roles in modulating the periodontal tissue responses. The influence of sex hormones can be minimized with good plaque control and with hormone replacement. Despite profound research linking periodontal condition with sex hormones kinetics, more deflnitive molecular mechanisms and therapy still remain to be determined.

Key words
Sex Hormones, Periodontal Disease, Pregnancy, Menopause

Introduction

Hormones are specific regulatory molecules that have potent effects on the major determinants of the development and the integrity of the skeleton and oral cavity including periodontal tissues. It is clear that periodontal manifestations occur when an imbalance of these steroid hormones take place. The Bacterial plaque has been established as the primary etiologic factor for the initiation of periodontal disease.1 However, it has also been shown that without a susceptible host the periodontal pathogens are necessary but not sufficient for disease to occur. Sexual hormones have been suggested as important modifying factors that may influence the pathogenesis of periodontal diseases. 2, 3

Steroid Sex Hormones

Steroid sex hormones are derived from cholesterol and as a common structure they have three rings of six carbon atoms. They are believed to play an important role in the maintenance of the skeletal integrity, including the alveolar bone. The steroid sex hormones such as estrogen and estradiol have been known for their effect on bone mineral metabolism. Other bone turnover-related hormones include progesterone, testosterone and dihydrotestosterone, androstenedione, dihydroepiandrostenedione, and sex hormone-binding globulin. Among these, estrogens, progesterone, and testosterone have been most linked with periodontal pathogenesis. 3

Effects of androgens on the periodontal tissues 4

• Inhibit prostaglandin secretion
• Enhance osteoblast proliferation and differentiation
• Reduce IL-6 production during inflammation
• Enhance matrix synthesis by periodontal ligament fibroblasts and osteoblasts 4

Effects of estrogen on the periodontal tissues4

• Decreases keratinization while increasing epithelial glycogen that results in the diminution in the effectiveness of the epithelial barrier
• Increases cellular proliferation in blood vessels
• Stimulates PMNL phagocytosis
• Inhibits PMNL chemotaxis
•Suppress leukocyte production from the bone marrow
•Inhibits proinflammatory cytokines released by human marrow cells
• Reduces T-cell mediated inflammation
•Stimulates the proliferation of the gingival fibroblasts
•Stimulates the synthesis and maturation of gingival connective tissues
•Increases the amount of gingival inflammation with no increase of plaque. 4

Effects of progesterone on the periodontal tissues 4

•Increases vascular dilatation, thus increases permeability
•Increases the production of prostaglandins
•Increases PMNL and prostaglandin E2 in the gingival crevicular fluid (GCF)
•Reduces glucocorticoid anti-inflammatory effect
•Inhibits collagen and noncollagen synthesis in periodontal ligament fibroblast
•Inhibits proliferation of human gingival fibroblast proliferation
•Alters rate and pattern of collagen production in

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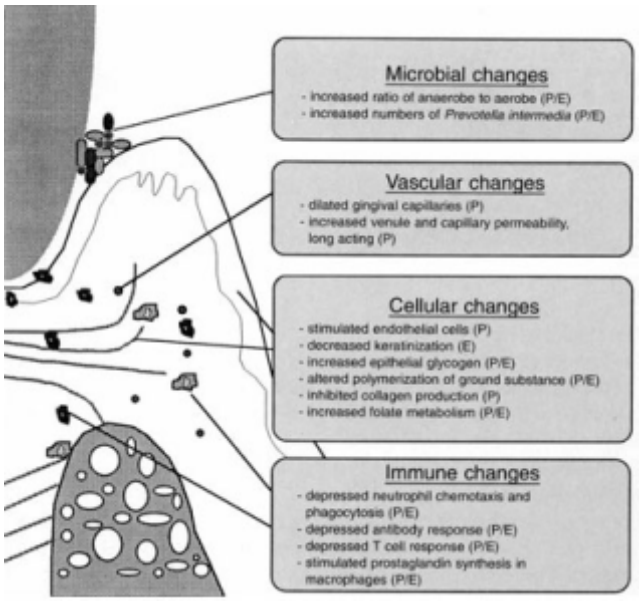
gingiva resulting in reduced repair and maintenance potential
•Increases the metabolic breakdown of folate which is necessary for tissue maintenance and repair. 4



Influence of estrogen and progesterone on the periodontal environment. P: effect of progesterone; E: effect of estrogen. 5
Courtesy: (Amar, Chung 1994) 5

Factors Infuencing Sex Hormone Effects on the Periodontium
1)Gender
2)Age
3)Hormone supplements

1) Gender
Gender plays an important role in changes of the bone density throughout the entire skeleton. It is also known that women are much more affected than men (e.g. osteoporosis). Lau et al. (2001) reported that 80% of the osteoporotic patients are female, correlating with the higher frequency of hip fractures in females, who are also more likely to experience hormonal imbalance throughout their lives than males. 6 In addition, when the influence of gender on periodontal disease was studied, females were considered for several years to be more affected than males, although contradicting data have been reported. This disparity seems to be simply correlated with the fact that females are more likely to seek dental care than males. 3

2) Age
The biological changes on the periodontal tissues during different time points such as puberty, the menstrual cycle, pregnancy, menopause, and oral contraceptive use have heightened interest in the relationship between steroid sex hormones and the health of the periodontium. Females seem to be more prone to hormone imbalance than males.

I) Puberty
Puberty is a complex process of sexual maturation resulting in an individual capable of reproduction. 3, 7 It is also responsible for changes in physical appearance and behavior,3,8,9,10 that are related with increased levels of the steroid sex hormones, testosterone in males and estradiol in females During puberty, the production of sex hormones increases to a level that remains constant for the entire normal reproductive period. Changes in hormone levels have been related with an increased prevalence of gingivitis followed by remission 3, a situation that is not necessarily associated with an increase in the amount of dental plaque. 3,11 The sub gingival microflora is also altered during this period since the bacterial counts increase in number, and there is a prevalence of certain bacterial species such as Prevotella intermedia (Pi) and Capnocytophaga species.3,12,13 Pi has been shown to possess the ability to substitute estrogen and progesterone for menadione (vitamin K) as an essential growth factor.14 Capnocytophaga species, which often increase during puberty, have been associated with the increased bleeding tendency observed during this period of time. 3, 13

Clinical and microbial changes in the periodontal tissues during puberty
•Increased gingival inflammation without accompanying an increase in plaque levels.
•Increased prevalence of certain bacterial species such as P.intermedia and Capnocytophaga species. 4

ii) Menstrual cycle
The menstrual cycle is controlled by the secretion of sex hormones over a 25-30-day period and is responsible for continued ovulation until menopause. 3, 15 In humans, the menstrual cycle can be divided into two phases: a follicular or proliferative phase, and a luteal or secretory phase. During the first phase, there is an increase in estrogen levels. At the same time, the luteinizing hormone stimulates progesterone secretion and ovulation. After ovulation, the luteal phase is characterized by an increase in progesterone and estrogen secretion. At the end of this phase, and if fertilization has not occurred, the plasma levels of progesterone and estradiol decline because of the demise of the corpus luteum. Generally, the periodontium does not exhibit evident changes during the menstrual cycle. Nonetheless, two different clinical findings have been observed in the oral cavity: gingival bleeding and increased production of gingival exudate. 3, 16

Clinical changes in the periodontal tissues during menstruation
•Bleeding and swollen gingiva
•An increase in gingival exudate
• A minor increase in tooth mobility 4

iii) Pregnancy
Some of the most remarkable endocrine alterations accompany pregnancy. During this period, both progesterone and estrogen are elevated due to continuous production of these hormones by the corpus luteum. By the end of the third trimester, progesterone and estrogen reach peak plasma levels of 100 and 6 ng/ml, respectively, which represent 10 and 30 times the levels observed during the menstrual cycle. Susceptibility to infections (e.g. periodontal infection) increases during early gestation due to alterations in the immune system 3, 17 and can be explained by the hormonal changes observed during pregnancy 18, suppression on T-cell activity, decreased neutrophil chemotaxis and phagocytosis, altered lymphocyte response and depressed antibody production 19, chronic maternal stress, and even nutritional deficiency associated with increased nutritional demand by both the mother and the fetus. These immunologic changes might also be responsible for periodontal pathologic conditions observed during pregnancy such as pregnancy gingivitis 20, 21, pregnancy granuloma, periodontitis, and dental caries. The increased synthesis of PGE2 observed when estradiol and progesterone are present in higher concentrations, such as occurs during pregnancy, may also contribute to these pathologic changes. 3 On the other hand, periodontal pathogens such as Pi and Porphyromonas gingivalis (Pg) can also use female sex hormones such as progesterone or estradiol as a source of nutrients. These

bacteria are generally increased in the gingival crevicular fluid of pregnant women, a situation that is positively correlated with the severity of pregnancy gingivitis. 3

Clinical and microbial changes in the periodontal tissues during pregnancy
• Increased gingival probing depths
• Increased gingival inflammation
• Increased gingival crevicular fluid flow
• Increased bleeding upon probing
• Increased tooth mobility
• Increased incidences of pyogenic granulomas
• Increased numbers of periodontopathogens especially P. gingivalis & P.intermedia 4

iv) Menopause and postmenopause
In the premenopausal women, the principal circulating estrogen is 17b-estradiol. As women approach menopause, the levels of estrogen begin to drop mainly during the late follicular and luteal phase of the menstrual cycle. 22 As a result of this physiologic situation, irregular cycles start to occur. Frequently, the time frame between regular cycles and the cessation of menstrual periods, called perimenopausal transition, is 2-7 years. During this period, the concentration of circulating estrogen decreases while follicle-stimulating hormone (FSH) and luteinizing hormone (LH) concentrations increase. Consequently, the effects of estrogen are reduced, therefore compromising the anti-inflammatory effect of this hormone on the periodontium. 3

Progesterone is another sex hormone that may play an important role in bone metabolism during pre- and post menopause. 23 It is believed that ovarian deficiency and associated alterations, but not aging, are the predominant causes of bone loss during the first two decades after menopause. Researches have shown that progesterone may compete with glucocorticoids for an osteoblast receptor and inhibit the glucocorticoid-induced osteoporosis. Therefore, postmenopausal bone density reduction may be the result of a combination of the inhibition of osteoclast downregulation by reduced estrogen and the increased cortisol inhibition of osteoblasts via the reduction of competition with progesterone. 3

Clinical changes in the periodontal tissues during menopause and postmenopause
•Reduction in epithelial keratinization
•A reduction in salivary gland flow
•Drying of the oral tissues
•Redness and abnormal paleness of the gingival tissues
•Bleeding on probing and brushing 4

3) Hormone replacement
As addressed above, females experience hormonal changes under both physiological (e.g. menstrual cycle, pregnancy) and nonphysiological conditions (e.g. hormone therapy, use of oral contraceptives.

I) Contraceptives
The influence of contraceptives on the periodontium is increases in inflammation and in the amount of gingival exudates, increase in the prevalence of dry socket after dental extraction, and accelerated progression of periodontal disease (higher gingival index scores and more loss of attachment). 3

Impact of contraceptives on clinical and microbial features of periodontal tissues
•Inflammation ranges from mild edema and erythema to severe inflammation with hemorrhagic or hyperplastic gingival tissues
•A 50 per cent increase in gingival fluid volume
•A 16-fold-increase in Bacteroides species 4

ii) Hormone replacement therapy in postmenopausal women
Estrogen deficiency is the dominant pathogenic factor for osteoporosis in women.24 Although hormonal replacement in an adequate dosage can slow or prevent bone loss 25, only a small percentage of postmenopausal women receive such therapy, and many who do fail to comply with the prescribed regimen because of the fear of cancer, irregular bleeding, and other minor side effects. Progesterone alone is not effective in preventing postmenopausal bone and tooth loss 26, but when combined with estrogen it is believed to uncouple formation and resorption to diminish bone resorption induced by estrogen. 3

Clinical changes in the periodontal tissues during menopause and postmenopause
•Reduction in epithelial keratinization
•A reduction in salivary gland flow
•Drying of the oral tissues
•Redness and abnormal paleness of the gingival tissues
•Bleeding on probing and brushing 4

Effects of HRT on the periodontal tissues
•A protection takes place against tooth loss
•Reduction in gingival bleeding
•Reduction in the risk of edentulousim 4

Hormonal influences on the microbiota
The effects of sex steroid hormones on the subgingival microbiota during pregnancy have been well documented. Kornman & Loesche 27 reported that during the second trimester, plaque levels remained constant, yet gingivitis and gingival bleeding were shown to increase in severity. 28 At the same time, the ratio of subgingival bacterial anaerobes-to-aerobes increased, as well as proportions of Bacteroides melaninogenicus and P. intermedia (2.2-10.1%). Subgingival plaque samples from these patients during the second trimester demonstrated a significantly higher accumulation of estradiol and progesterone than plaque samples at other time periods. Subsequently, both estradiol and progesterone were shown to be selectively accumulated by P. intermedia as a substitute for vitamin K, and thus postulated to be acting as a growth factor for this microorganism. Not all studies have corroborated these findings, and Jonsson et al. 29 found no difference in levels of P. intermedia at any time during pregnancy or between pregnant and nonpregnant controls in a cross-sectional assessment. This has led to speculation that the increase in P. intermedia seen during the second trimester of pregnancy may actually be independent of estrogens or progesterone and may occur for other reasons. Mariotti 2 has made observations in this regard. First, P. intermedia is seen to increase during the second trimester of pregnancy followed by a decline to postpartum values during the third trimester, despite highly elevated hormone levels still present during the third trimester. Additionally, there was no analysis of competitive inhibition with other steroid-like molecules performed in the heretofore cited studies; therefore, it is open to question whether the accumulation of estradiol or progesterone in second trimester plaque samples or pure cultures of P. intermedia was sex steroid hormone specific or merely dependent on the lipophilic nature of the plaque sample. 28

Hormonal influences on the gingival vasculature
The effects of estrogens and progestins on the gingival vasculature could potentially explain the increased edema, erythema, gingival crevicular exudate, and hemorrhagic gingival tissues noted during

pregnancy as well as other stages of the reproductive cycle. An increase in gingival crevicular fluid flow has been correlated to elevated sex steroid levels, which indicates that these hormones may affect vascular permeability in the gingival sulcus. 28

Hormonal influences on cells of the periodontium

The effects of sex steroid hormones on individual cells of the periodontium may also play a significant role in the exaggerated gingival responses seen during the female reproductive cycle and pregnancy. Sex steroid hormones have been shown to directly and indirectly exert influence on cellular proliferation, differentiation, and growth in target tissues, including keratinocytes and fibroblasts in the gingiva.2 Two theories for the actions of the hormones on these cells involve the role hormones may play in altering the effectiveness of the epithelial barrier to bacterial insult, and in affecting collagen maintenance and repair. Estrogens stimulate epithelial proliferation and increase keratinization of the vaginal mucosa. 5 Some evidence also exists that sex hormones may have a similar effect on the oral mucosal and gingival epithelia, and a reduction in the keratinization of gingival epithelium of postmenopausal women has been shown to accompany declining plasma estrogen levels. Fibroblast proliferation and collagen maturation in gingival connective tissues may be affected by both estrogen and progesterone. By altering collagen turnover, estrogens may stimulate the proliferation of gingival fibroblasts, and the synthesis and maturation of gingival connective tissues. Sex steroid hormones have also been shown to increase the rate of folate metabolism in oral mucosa. 30 Since folate is required for tissue maintenance, increased metabolism could deplete folate stores and inhibit tissue repair. Additionally, progesterone in concentrations corresponding to the third trimester of pregnancy has been shown to lower the synthesis of glycosaminoglycans, a major constituent of the connective tissue matrix of gingiva. 28

Influence of Sex Hormones on Periodontal/Implant Wound Healing

At a molecular level, research has also shown that sex hormones have a regulatory effect on growth factors involved in the wound healing such as the keratinocyte growth factor 31, which has been known to have wound healing regulatory effect including stimulation of proliferation, migration, and morphogenesis of pluripotential cells. However, the influence of sex hormones on periodontal wound healing is still largely unknown.3

Conclusion

Sexual hormones play an important role in influencing periodontal disease progression and wound healing. These effects are different depending on the gender as well as the lifetime period analyzed. It is also clear that not all patients and their periodontium respond in the same way to similar amounts of circulating sexual hormones. In addition, the influence of sex hormones can be minimized with good plaque control as well as with hormone replacement therapies; however, the true mechanism of how these interactions actually occur remains to be determined. 3

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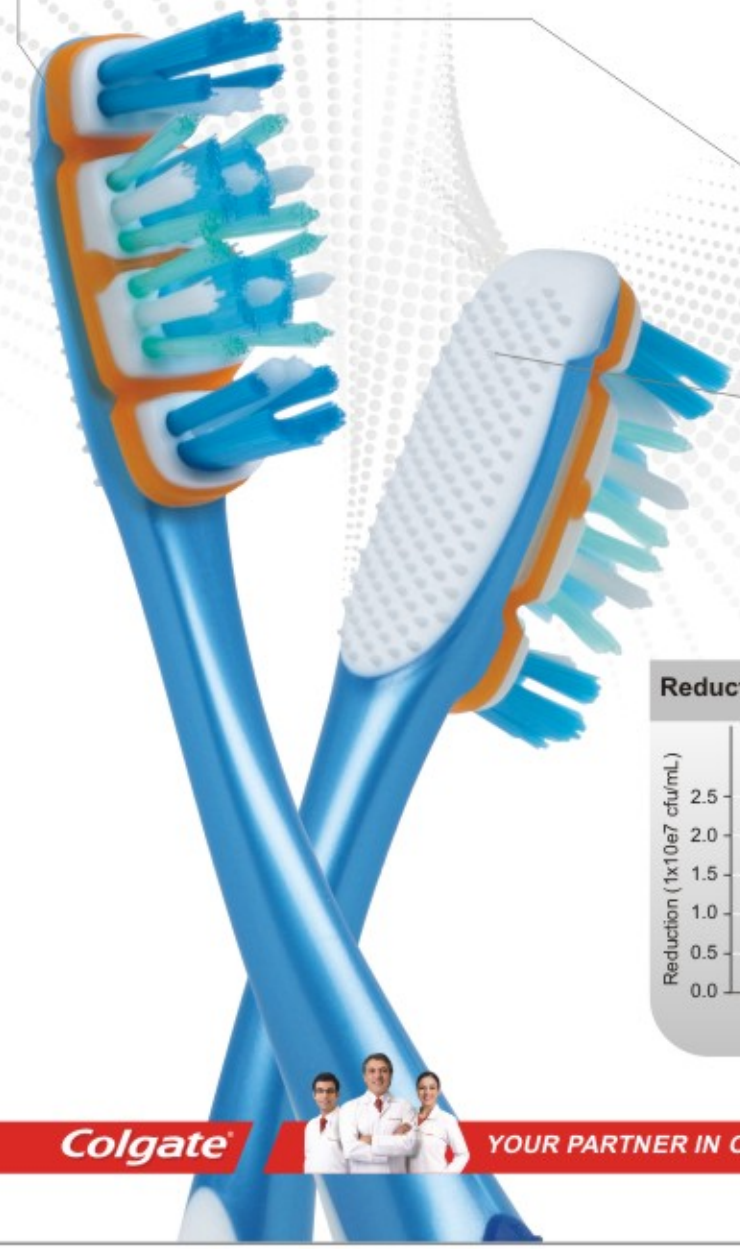

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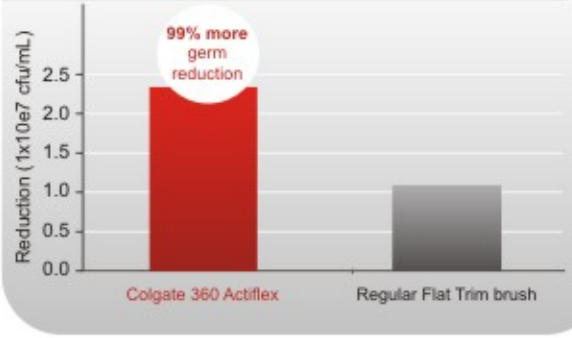

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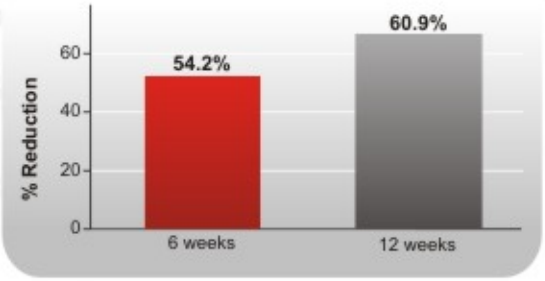

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