

# DENTISTRY

Modern Analysis, Finding and Researches

Editor

Ayşe MEŞE



LIVRE DE LYON

2023

Dentistry

# Dentistry

Modern Analysis, Finding and Researches

**Editor**  
**Ayşe MEŞE**



LIVRE DE LYON

Lyon 2023



# Dentistry

Modern Analysis, Finding and Researches

**Editor**

**Ayşe MEŞE**



LIVRE DE LYON

Lyon 2023

## **Dentistry Modern Analysis, Finding and Researches**

**Editor** • Prof. Dr. Ayşe MEŞE • Orcid: 0000-0002-1612-5516

**Cover Design** • Motion Graphics

**Book Layout** • Motion Graphics

**First Published** • October 2023, Lyon

**ISBN:** 978-2-38236-609-7

**copyright © 2023 by Livre de Lyon**

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted in any form or by any means, electronic, mechanical, photocopying, recording, or otherwise, without prior written permission from the Publisher.

**Publisher** • Livre de Lyon

**Address** • 37 rue marietton, 69009, Lyon France

**website** • <http://www.livredelyon.com>

**e-mail** • [livredelyon@gmail.com](mailto:livredelyon@gmail.com)



LIVRE DE LYON

## PREFACE

Dear readers;

Dentistry, which has an important place in the healthcare community, needs to follow technological developments and implement innovations in basic areas. We are proud to present to you, our book, “**Dentistry Modern Analysis Finding and Researches**”, which will serve this purpose, consists of 8 different chapters covering current topics. In each chapter, new developments and research on the subject are included in addition to classical information, and it is aimed to contribute to the literature.

I would like to thank my colleagues who contributed to the preparation of this book, which I am proud to edit, as well as the publishing organization and publishing house employees. I hope this book will be an important resource for all readers.

**Kind regards...**  
**Prof. Dr. Ayşe MEŞE**



# CONTENTS

<b>PREFACE</b>	I
<b>CHAPTER I. HALITOSIS</b>	1
<i>Buse Başak FEYİZOĞLU</i>	
<b>CHAPTER II. GINGIVAL RECESSON: ETIOLOGY AND MANAGEMENT</b>	13
<i>Buse Başak FEYİZOĞLU</i>	
<b>CHAPTER III. PROSTHETIC TECHNIQUES USED TO IMPROVE GINGIVAL AESTHETICS</b>	25
<i>Özlem SARAÇ ATAGÜN</i>	
<b>CHAPTER IV. DIAGNOSIS AND TREATMENT PLANNING IN PERIODONTICS</b>	35
<i>Pınar ŞAYAN</i>	
<b>CHAPTER V. ORAL HEALTH STATUS IN CHILDREN WITH TYPE 1 DIABETES MELLITUS</b>	45
<i>Muhammet Ali ORUÇ &amp; Semih Ercan AKGÜN</i>	
<b>CHAPTER VI. AUTOGENOUS TOOTH TRANSPLANTATION</b>	57
<i>Zeynep GÜMÜŞER</i>	
<b>CHAPTER VII. SURGICAL TREATMENT OPTIONS OF OBSTRUCTIVE SLEEP APNEA SYNDROME</b>	69
<i>Ipek Necla GULDIKEN &amp; Yigit Can GULDIKEN</i>	
<b>CHAPTER VIII. USE OF DWI-MRI IN DENTOMAXILLOFACIAL RADIOLOGY</b>	81
<i>Büşra ERYİĞİT &amp; Taha Emre KÖSE &amp; Muhammed Enes NARALAN</i>	





# CHAPTER I

## HALITOSIS

**Buse Başak FEYİZOĞLU**

(DDS, PhD), E-mail: [busebasakyilmaz@gmail.com](mailto:busebasakyilmaz@gmail.com)

ORCID: 0000-0001-5276-2486

### 1. Introduction

**H**alitosiis, fetor ex ore, fetor oris, oral malodor and bad breath are terms used to refer to unpleasant or offensive odor emanating from the oral cavity when breathing or speaking. The term fetor ex ore has been used to describe malodors that arise from conditions within the mouth and associated sinuses. Halitosis is derived from the Latin *halitus* meaning breathed air and *osis* is condition.(1,2)

Oral malodor has often been overlooked by the dental profession, despite its significant impact on patient visits to the dentist. It may be considered the third most common reason for dental appointments, trailing only behind dental caries and periodontal disease. However, there is a noticeable shift in this trend, as emerging oral malodor clinics are now providing potential remedies by targeting the bacterial populations in the tongue and periodontal tissues.(3)

### 2. Prevalence

Halitosis prevalence has been investigated in various groups of individuals across different regions of the world using convenience samples. These studies present varying assessments and cutoff points, making it difficult to obtain precise estimates of halitosis prevalence. A population-based cross-sectional study from North Italy reported prevalence of halitosis as 53.51%(4), while Hammad et al. indicated that the prevalence of halitosis was 78% in a sample of Jordanian population.(5) Bornstein et al. evaluated the prevalence of halitosis in a young male adult population in Switzerland. According to their study, ~20% of the recruits reported suffering at least occasionally from halitosis.(6) A cross-sectional study from Northwest Ethiopia revealed a

prevalence of halitosis as 44.2% among the study participants.(7) Likewise prevalence of halitosis in Japanese school children was found to be 44.9%.(8) According to a clinical research among Chinese subjects, 65.9% had halitosis. (9) A review by Akaji et al. in 2014 claimed that the overall prevalence ranges from 22% to 50%(10), similar to the systematic review in 2017 which reported the prevalence of halitosis as 31.8%.(11) It is a clear fact that halitosis is a prevalent problem and it has negative impacts on people's social interactions and quality of life.(1)

### **3. Classification**

In general, halitosis can be divided into two subgroups as primary and secondary. Primary halitosis refers to the expiration air from the lungs, while secondary halitosis arises from the upper respiratory tract or the oral cavity.(12)

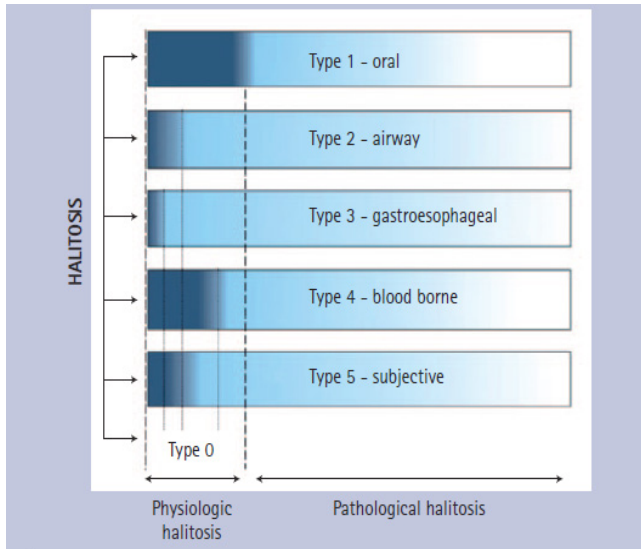
According to Miyazaki et al. halitosis is clinically categorized into three groups: genuine halitosis, pseudo-halitosis and halitophobia. Genuine halitosis is divided into physiological or pathological, and the latter is split into oral and extra-oral.(13) Pseudo-halitosis refers to the situation where the patient claims to experience bad breath without an actual existence. It can be treated simply by counseling and oral hygiene procedures.(14) Halitophobia assessed as psychosomatic disorders related to dentistry affect at least 0.5-1% of the adult population.(15) It can be described as a fear of having halitosis. The halitophobic patients show a disproportionate concern and are obsessed with their breath. It is also verified that pseudo-halitosis patients accept the clinician's diagnosis after receiving explanations, while halitophobics never believe the nonexistence of oral malodor.(16)

In 2010 Tangerman and Winkel suggested intra- and extra-oral halitosis, depending on the place where it originates. Extra-oral halitosis can be subdivided into non-blood-borne halitosis, such as halitosis from the upper respiratory tract including the nose and from the lower respiratory tract, and blood-borne halitosis.(17)

Due to the lack of standardization in these classifications and their inability to clearly define various types of breath odour, a new classification system independent of subjective descriptions was proposed in 2014. According to that system, halitosis has 6 types: type 0 (physiologic), type 1 (oral), type 2 (airway), type 3 (gastroesophageal), type 4 (blood borne), type 5 (subjective). (18) Relationship among these types was shown in Fig. 1.

## 4. Etiology

Most of the patients suffering from halitosis have oral causes. However a small, but important percentage of oral malodor cases have an extra-oral etiology. (19) Main cause of halitosis is the microbial degradation in the oral cavity and volatile sulphur compounds (VSCs) that are formed through this process.(20)



**Fig. 1** New etiologic classification proposed by Aydin et al.(18)

### 4.1. Oral Causes

#### 4.1.1. Tongue Coating

Tongue coating, including bacteria, desquamated cells, and saliva is the most important factor inducing halitosis.(21) As a consequence of its large and papillary surface area, the dorsum of the tongue can retain large amounts of desquamated cells, leucocytes, and micro-organisms.(22) According to the study of Quirynen et al. 76% of the halitosis patients visited their multidisciplinary breath odour clinic have an intra-oral cause with tongue coating being most frequently seen.(23)

Scientific analysis of odors using chemical and organoleptic analysis demonstrated that the major elements in the production of oral malodor are volatile sulfur compounds (VSC), primarily hydrogen sulfide (H<sub>2</sub>S), methylmercaptan (CH<sub>3</sub>SH), and dimethyl sulfide (CH<sub>3</sub>SCH<sub>3</sub>).(2) These gases originate from the degradation of sulphur-containing amino acids such as cysteine, cystine, methionine by various oral bacterial species.(24) Tanaka et al. revealed that the

five periodontal pathogens (*P. gingivalis*, *Tannerella forsythia*, *P. intermedia*, *Prevotella nigrescens* and *Treponema denticola*) in tongue coating samples are major contributors to VSC production.(25)

#### **4.1.2. Periodontal Disease**

VSCs, as mentioned before, have been reported to be major contributors to halitosis. Gram-negative anaerobic microorganisms, such as *Porphyromonas gingivalis*, *Treponema denticola*, and *Tannerella forsythia*, which are also associated with periodontal diseases, are the main cause of increasing VSC levels.(26) Periodontal disease is a chronic inflammatory condition of the tooth-supporting tissues that results in bone destruction if not treated.(27,28) It is highly prevalent in adult-aged populations all over the world and its advanced form is one of the main reasons of tooth loss in adults.(29) Patients with periodontal disease were reported to be at higher risk for halitosis than individuals with healthy periodontal tissues. (30) According to the review of Geest et al. periodontal disease can be an additional, but less important, contributor of halitosis as not all patients with periodontal disease have oral malodor, and periodontally healthy patients can present with malodor.(31)

#### **4.1.3. Xerostomia**

Xerostomia, or commonly known as dry mouth, can also be an important factor inducing halitosis. Decreased saliva flow leads to an increased plaque accumulation on teeth and tongue. Hyposalivation also results in the loss of antimicrobial effectiveness of saliva. Several factors can contribute to xerostomia, including diabetes, medication, Sjögren syndrome, mouth breathing, stress, alcohol abuse and depression. It is also associated with aging.(20)

#### **4.1.4. Other Oral Causes**

Healing wounds, exposed tooth pulps, fixed orthodontic appliances, pericoronal infections, caries and insufficient oral hygiene are other oral contributing factors to halitosis.(1,32)

### **4.2. Non-oral Causes**

#### **4.2.1. Blood-borne Halitosis**

Metabolites with unpleasant odors, absorbed from anywhere in the body can be carried to the lungs through the bloodstream. If the malodorous

substances have sufficient concentrations, they can be expelled through exhaling, imparting an unpleasant odor to the breath, causing blood-borne halitosis. Methylmercaptan (CH<sub>3</sub>SH) is proved to be the main cause inducing blood-borne halitosis, while hydrogen sulfide (H<sub>2</sub>S) and dimethyl sulfide (CH<sub>3</sub>SCH<sub>3</sub>) are linked to intra-oral halitosis.(33) Conditions causing blood-borne halitosis can be listed as following; systemic diseases, metabolic disorders, certain foods and medications.(17)

#### ***4.2.2. Non-blood-borne Halitosis***

Nasal infections such as sinusitis and throat infections such as tonsillitis are one of the main reasons of non-blood-borne halitosis.(34) Other conditions suggested by Tangerman et al. are respiratory system infections, lung diseases and stomach disorders.(17)

### **5. Diagnosis**

The recognition of halitosis typically commences when the individual expresses concern about having bad breath or has been informed of it by someone else. However, it's noteworthy that the patient's self-reported complaint of malodor is the least dependable criterion for documenting oral malodor.(3)

#### ***5.1. Organoleptic Measurements***

Organoleptic measurement is a sensory test assessed by the examiner's perception of a subject's oral malodor. It is considered as the "gold standard" to diagnose halitosis, although the objectivity and reproducibility of organoleptic measurement are limited. That is because of the the capability of the human nose to detect and categorize not only VSCs but also other organic compounds emitted during exhalation, which are perceived as unpleasant. Another advantage is that it doesn't require any special equipment.(35,36)

Organoleptic measurement can be easily conducted by sniffing the patient's breath and assigning a score to the degree of oral malodor. A straw or plastic tube is introduced into the patient's mouth. As the patient exhales slowly, the examiner assesses the odor at the opposite end of the tube or straw. A privacy screen equipped with an aperture for the straw or tube can be employed to create a barrier between the examiner and the patient. The odor of nasal breath can also be evaluated by inserting a tube into one of the nostrils while closing the other nostril with a finger (Fig. 2) This method helps prevent the dilution of the odor with room air.(37) Nevertheless, the reliability and reproducibility of

this method pose challenges, prompting ongoing research projects aimed at enhancing its effectiveness.(38)



**Fig. 2** Organoleptic measurement(37)

### ***5.2. Gas Chromatography***

Gas chromatography is utilized to quantify the concentration of VSCs in samples of saliva, tongue coating, or expired breath.(38) It has the capability to detect odorous molecules even at low concentrations and the results are highly objective, reliable and reproducible.(39) However, it comes with drawbacks such as high cost, bulkiness, and the necessity for a well-trained operator. The process is time-consuming, and the machine cannot be applied in daily practice, thus limiting its usage to research contexts.(40) Combining gas chromatography of with organoleptic scoring is considered the most effective method for detecting oral halitosis.(41)

### ***5.3. Sulphide Monitoring***

Sulfide monitors analyze the total sulfur content in the air exhaled from the subject's mouth. A portable monitor has been created for assessing volatile sulfur compounds. Patients are instructed to abstain from talking for 5 minutes before the measurement. The monitor is calibrated to ambient air, and the measurement is conducted by inserting a disposable tube into the patient's mouth, connecting it to the monitor, while the patient breathes through the nose. Electrochemical reactions with sulfur-containing compounds in the breath produce an electric current directly proportional to the levels of volatile sulfur-containing compounds.(38,40) Sulfide monitoring is a relatively cost-effective and user-friendly method; however, it has the limitation of not detecting certain crucial odors.(38)

#### 5.4. BANA Test

Three species linked to periodontal disease; *Treponema denticola*, *Porphyromonas gingivalis*, and *Tannerella forsythia* generate VSCs as mentioned before.(26,42) Detection of these compounds in plaque and/or tongue samples could offer supplementary insights into factors contributing to halitosis. The presence of these organisms in plaque samples can be identified by the detection of an enzyme or enzymes that break down benzoyl-DL-arginine-anaphthylamide(BANA), resulting in the formation of a colored compound.(3) In patients with periodontal disease, a more robust correlation was observed between BANA test results and measurements from the sulfide monitor.(43)

### 6. Treatment

The first step in the treatment of halitosis is accurate diagnosis. Every patient, regardless of the type of halitosis, should receive a professional oral health care examination. Periodontal and restorative treatment should be performed if needed in order to treat the contributing factors of halitosis.(39)

Mechanical removal of biofilm and reducing microorganisms on the dorsum of the tongue is of paramount importance in managing halitosis. The study of Choi et al. confirm the effectiveness of mechanical tongue cleaning in reducing both bad breath and tongue coating, regardless of the adopted technic.(44) According to the systematic review by Van der Sleen et al. mechanical approaches have the potential to effectively reduce tongue coating and halitosis, however the authors didn't make a firm statement due to lack of evidence.(45) Gonçalves et al. proposed a new tongue hygiene technique, the X technique, and showed that it has significant positive impact on organoleptic scores and the quantity of bacterial colonies found on the dorsum of the tongue.(46)

Considering that malodor is generated by the presence of microorganisms, any treatment approach affecting the oral microbiota holds the potential to reduce halitosis. Mouthrinses containing antibacterial agents such as chlorhexidine and cetylpyridinium chloride are reported to have a significant impact on decreasing the levels of microorganisms responsible for halitosis on the tongue.(47) Combining the two are proved to achieve the best results regarding anti-halitosis effect and anti-microbial activity on salivary bacterial counts.(48) Additionally mouthrinses containing chlorine dioxide and zinc have the potential to effectively neutralize odorous sulfur compounds.(47)

If oral interventions prove ineffective in reducing or eliminating halitosis, it is advisable to refer patients to a physician. In the absence of suspected medical causes, the initial specialist for referral should be an otorhinolaryngologist,



followed by a gastroenterologist. A psychologist or psychiatrist should be included if halitophobia is suspected.(35) A nutritionist assessment can also be beneficial for treatment of halitosis patients.(49)

Masking agents such as mouthrinses, chewing gums, mint tablets and sprays have only short-term effect on reducing halitosis.(39) Nevertheless they can be employed in order to satisfy the patient. Chewing gum usage may reduce halitosis, particularly through increasing saliva secretion.(35)

## 7. Conclusion

Halitosis treatment should be taken seriously, and a multifactorial thorough assessment should be adopted in order to achieve good results. It is largely caused by intra-oral factors but also a limited number of cases result from extraoral or systemic problems. Therefore it is vital for health care professionals, including general physicians, periodontologists, ENT specialists and psychiatrists to understand its etiology and risk factors for accurate diagnosis and appropriate patient treatment. Further scientific investigations are required to effectively address and treat halitosis.

## References

1. Wu J, Cannon RD, Ji P, Farella M, Mei L. Halitosis: prevalence, risk factors, sources, measurement and treatment – a review of the literature. *Aust Dent J.* 2020;65(1):4–11.
2. Messadi D V, Younai FS. Halitosis. *Dermatol Clin.* 2003;21(1):147–55.
3. Loesche WJ. Microbiology and treatment of halitosis. *Curr Infect Dis Rep.* 2003;5(3):220–6.
4. Aimetti M, Perotto S, Castiglione A, Ercoli E, Romano F. Prevalence estimation of halitosis and its association with oral health-related parameters in an adult population of a city in North Italy. *J Clin Periodontol.* 2015;42(12):1105–14.
5. Hammad M, Darwazeh A, Al-Waeli H, Tarakji B, Alhadithy T. Prevalence and awareness of halitosis in a sample of Jordanian population. *J Int Soc Prev Community Dent.* 2014;4(6):S178–86.
6. Bornstein MM, Stocker BL, Seemann R, Bürgin WB, Lussi A. Prevalence of Halitosis in Young Male Adults: A Study in Swiss Army Recruits Comparing Self-Reported and Clinical Data. *J Periodontol.* 2009;80(1):24–31.
7. Teshome A, Derese K, Andualem G. The prevalence and determinant factors of oral halitosis in northwest ethiopia: A cross-sectional study. *Clin Cosmet Investig Dent.* 2021;13:173–9.

8. Ueno M, Ohnuki M, Zaitso T, Takehara S, Furukawa S, Kawaguchi Y. Prevalence and risk factors of halitosis in Japanese school children. *Pediatr Int*. 2018;60(6):588–92.
9. Du M, Li L, Jiang H, Zheng Y, Zhang J. Prevalence and relevant factors of halitosis in Chinese subjects: A clinical research. *BMC Oral Health*. 2019;19(1):1–11.
10. Akaji EA, Folaranmi N, Ashiwaju O. Halitosis: a review of the literature on its prevalence, impact and control. *Oral Health Prev Dent [Internet]*. 2014;12(4):297–304. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/25525639>
11. Silva MF, Leite FRM, Ferreira LB, Pola NM, Scannapieco FA, Demarco FF, et al. Estimated prevalence of halitosis: a systematic review and meta-regression analysis. *Clin Oral Investig*. 2018;22(1):47–55.
12. Özer NE, İlhan B. Halitozis : Güncel Sınıflama , Tanı ve Tedavi Yaklaşımları Halitosis : New Classification , Diagnosis and Management. *EÜ Dişhek Fak Derg*. 2021;42(3):227–37.
13. Miyazaki H, Arao M, Okamura K, Kawaguchi Y, Hoshi K, Yaegaki K. Tentative classification of halitosis and its treatment needs. *Niigata Dent J*. 1999;32:7–11.
14. Madushankari GS, Yamunadevi A, Selvamani M, Mohan Kumar KM, Basandi PS. Halitosis – An overview: Part-I – Classification, etiology, and pathophysiology of halitosis. *J Pharm Bioallied Sci*. 2015;7:339–43.
15. Çoban Z, Sönmez I. Halitosis: a review of current literature. *Meandros Med Dent J*. 2017;18:164–70.
16. Falcão DP, Vieira CN, Batista De Amorim RF. Breaking paradigms: A new definition for halitosis in the context of pseudo-halitosis and halitophobia. *J Breath Res*. 2012;6(1):17–22.
17. Tangerman A, Winkel EG. Extra-oral halitosis: An overview. *J Breath Res*. 2010;4:1–6.
18. Aydin M, Harvey-Woodworth CN. Halitosis: A new definition and classification. *Br Dent J [Internet]*. 2014;217:E1. Available from: <http://dx.doi.org/10.1038/sj.bdj.2014.552>
19. Campisi G, Musciotto A, Fede O Di, Marco V Di, Craxì A. Halitosis: Could it be more than mere bad breath? *Intern Emerg Med*. 2011;6(4):315–9.
20. Bollen CML, Beikler T. Halitosis: the multidisciplinary approach. *Int J Oral Sci*. 2012;4(2):55–63.

21. Calil C, Liberato FL, Pereira AC, de Castro Meneghim M, Goodson JM, Groppo FC. The relationship between volatile sulphur compounds, tongue coating and periodontal disease. *Int J Dent Hyg.* 2009;7(4):251–5.

22. Porter R, Scully C. Oral malodour (halitosis). *BMJ.* 2006;333:632–5.

23. Quirynen M, Dadamio J, S VDV, M DS, Dekeyser C, M VT. Characteristics of 2000 patients who visited a halitosis clinic. *J Clin Periodontol.* 2009;36:970–5.

24. Lee C-H, Kho H-S, Chung S-C, Lee S-W, Kim Y-K. The Relationship Between Volatile Sulfur Compounds and Major Halitosis-Inducing Factors. *J Peridontology.* 2003;74(1):32–7.

25. Tanaka M, Yamamoto Y, Kuboniwa M, Nonaka A, Nishida N, Maeda K, et al. Contribution of periodontal pathogens on tongue dorsa analyzed with real-time PCR to oral malodor. *Microbes Infect.* 2004;6(12):1078–83.

26. Lee YH, Shin S II, Hong JY. Investigation of volatile sulfur compound level and halitosis in patients with gingivitis and periodontitis. *Sci Rep [Internet].* 2023;13(1):1–11. Available from: <https://doi.org/10.1038/s41598-023-40391-3>

27. Han J, Menicanin D, Gronthos S, Bartold PM. Stem cells, tissue engineering and periodontal regeneration. *Aust Dent J.* 2014;59(SUPPL. 1):117–30.

28. Highfield J. Diagnosis and classification of periodontal disease. *Aust Dent J.* 2009;54:S11–26.

29. Könönen E, Gursoy M, Gursoy UK. Periodontitis: A multifaceted disease of tooth-supporting tissues. *J Clin Med.* 2019;8(8):1135.

30. Apatzidou AD, Bakirtzoglou E, Vouros I, Karagiannis V, Papa A, Konstantinidis A. Association between oral malodour and periodontal disease-related parameters in the general population. *Acta Odontol Scand.* 2013;71(1):189–95.

31. De Geest S, Laleman I, Teughels W, Dekeyser C, Quirynen M. Periodontal diseases as a source of halitosis: A review of the evidence and treatment approaches for dentists and dental hygienists. *Periodontol* 2000. 2016;71(1):213–27.

32. Zalewska A, Zatoński M, Jablonka-Strom A, Paradowska A, Kawala B, Litwin A. Halitosis - a common medical and social problem. A review on pathology, diagnosis and treatment. *Acta Gastroenterol Belg.* 2012;75(3):300–9.

33. Tangerman A. Halitosis in medicine: A review. *Int Dent J.* 2002;52(3 SUPPL.):201–6.

34. Ferguson M, Aydin M, Mickel J. Halitosis and the tonsils: A review of management. *Otolaryngol - Head Neck Surg.* 2014;151(4):567–74.

35. Rösing CK, Loesche W. Halitosis: An overview of epidemiology, etiology and clinical management. *Braz Oral Res.* 2011;25(5):466–71.
36. Nakhleh MK, Quatredeniens M, Haick H. Detection of Halitosis in Breath: Between the Past, Present and Future. *Oral Dis.* 2018;24(5):685–95.
37. Murata T, Yamaga T, Iida T, Miyazaki H, Yaegaki K. Classification and examination of halitosis. *Int Dent J.* 2002;52:181–6.
38. van den Broek AMWT, Feenstra L, de Baat C. A review of the current literature on aetiology and measurement methods of halitosis. *J Dent.* 2007;35(8):627–35.
39. Kapoor U, Sharma G, Juneja M, Nagpal A. Halitosis: Current concepts on etiology, diagnosis and management. *Eur J Dent.* 2016;10(2):292–300.
40. Kini VV, Pereira R, Padhye A, Kanagotagi S, Pathak T, Gupta H. Diagnosis and Treatment of Halitosis: An Overview. *J Contemp Dent.* 2012;2(3):89–95.
41. Tangerman A, Winkel EG. Intra- and extra-oral halitosis: Finding of a new form of extra-oral blood-borne halitosis caused by dimethyl sulphide. *J Clin Periodontol.* 2007;34(9):748–55.
42. Amou T, Hinode D, Yoshioka M, Grenier D. Relationship between halitosis and periodontal disease - associated oral bacteria in tongue coatings. *Int J Dent Hyg.* 2014;12(2):145–51.
43. Figueiredo LC, Rosetti EP, Marcantonio E, Marcantonio RAC, Salvador SL. The Relationship of Oral Malodor in Patients With or Without Periodontal Disease. *J Periodontol.* 2002;73(11):1338–42.
44. Choi HN, Cho YS, Koo JW. The effect of mechanical tongue cleaning on oral malodor and tongue coating. *Int J Environ Res Public Health.* 2022;19(1).
45. Van der Sleen MI, Slot DE, Van Trijffel E, Winkel EG, Van der Weijden GA. Effectiveness of mechanical tongue cleaning on breath odour and tongue coating: a systematic review. *Int J Dent Hyg.* 2010;8(4):258–68.
46. Gonçalves AC de S, Martins MCN, Paula BL de, Weckwerth PH, Franzolin S de OB, Silveira EMV. A new technique for tongue brushing and halitosis reduction: The X technique. *J Appl Oral Sci.* 2019;27(14):1–8.
47. Fedorowicz Z, Aljufairi H, Nasser M, Outhouse TL, Pedrazzi V. Mouthrinses for the treatment of halitosis. *Cochrane Database Syst Rev.* 2016;2016(5).
48. Roldán S, Herrera D, Santa-Cruz I, O'Connor A, González I, Sanz M. Comparative effects of different chlorhexidine mouth-rinse formulations on volatile sulphur compounds and salivary bacterial counts. *J Clin Periodontol.* 2004;31(12):1128–34.

49. Dal Rio ACC, Nicola EMD, Teixeira ARF. Halitosis - An assessment protocol proposal. *Braz J Otorhinolaryngol* [Internet]. 2007;73(6):835–42. Available from: [http://dx.doi.org/10.1016/S1808-8694\(15\)31180-0](http://dx.doi.org/10.1016/S1808-8694(15)31180-0)

## CHAPTER II

# GINGIVAL RECESSION: ETIOLOGY AND MANAGEMENT

**Buse Başak FEYİZOĞLU**

*(DDS, PhD), E-mail: busebasakyilmaz@gmail.com*

*ORCID: 0000-0001-5276-2486*

### 1. Introduction

**G**ingival recession can be described as the apical migration of the gingival margin and thereby exposure of the root surface to the oral environment. (1) It may occur in a specific area or affect the entire gingiva, and could be associated with one or multiple surfaces. Epidemiological studies establish that gingival recession has a high prevalence rate, approximately half of the population exhibit one or more surfaces with gingival recession regardless of the oral hygiene.(2,3) There is also supporting evidence that gingival recession becomes more prevalent with age although it is not a direct consequence of ageing.(4–6)

Aesthetic appearance can be affected by gingival recession, which is a common concern among patients. Especially those with pronounced gingival display often refer to dental clinics seeking treatment. However their attention rarely extends beyond the facial aspect of the front teeth.(7) In addition to aesthetic worries, another undesirable outcome of gingival recession is the exposure of root surfaces to the oral cavity. It is frequently associated with dentine hypersensitivity, non-carious cervical lesions, root caries and compromised plaque control.(8) A non-carious cervical lesion (NCCL) is defined as the erosion of tooth at the gingival one-third level, attributed to factors other than dental caries. Its etiology is accepted to be multifactorial such as abrasion due to mechanical forces, corrosion and abfraction (Fig. 1).(9)



**Fig. 1.** Canine with gingival recession and a deep NCCL defect.(9)

Bearing in mind these various considerations, the dental professional needs to take into account both aesthetic and dental health considerations related to gingival recession, along with individual patient factors, while formulating a treatment strategy.

## **2. Etiology**

### **2.1. Anatomical Factors**

The definition “periodontal phenotype” is used for describing the combination of bone morphotype (buccal bone plate thickness) and gingival phenotype (gingival volume, gingival thickness). Jepsen et al. suggested using the term “phenotype” rather than “biotype”, postulating that its expression includes the latter.(1) Several studies report that patients with thin phenotypes tend to develop more gingival recession lesions.(1,10,11)

Another anatomical factor related to gingival recession is dehiscence of the alveolar bone (Fig. 2). Depending on the buccal bone plate thickness, vertical bone loss leads to localized resorption specifically at the buccal bone level. This results in bone dehiscence on the affected root, forming a V-shaped cavity in the bone contour, causing a local decrease in the gingival bone support. Although for a specific period, it might be covered with periosteum, as the gingival soft tissues tend to keep up with cervical bone level, gingival recession will be most likely established in that area. Likewise when there is a bone window on the buccal bone plate, so called fenestration, the tooth become more susceptible to gingival recession formation (Fig. 3).(2,12)



**Fig. 2.** Bone dehiscence associated with gingival recession.(13)



**Fig. 3.** Severe canine dehiscence and premolar fenestration.(12)

Among potential etiological and predisposing factors, the quantity of keratinized gingiva has been proposed. Nevertheless, studies have not shown a clear causative relationship in this regard. Evidence indicates that gingival recession is not necessarily a natural consequence even in the absence of keratinized gingiva.(14)

Other anatomical factors associated with gingival recession are abnormal tooth position in the arch, the shape of the tooth and aberrant path of eruption of the tooth. These factors are connected and could lead to a thinner-than-usual alveolar osseous plate, making it more prone to resorption.(2,13)



## ***2.2. Physiological Factors***

Orthodontic tooth movement does not necessarily result in damage to gingival tissues. However, clinical studies have indicated that, during orthodontic treatment the movement of incisors beyond the osseous envelope of the alveolar process are more likely to develop gingival recessions. In such cases, prior to gingival retraction, orthodontic movement has caused dehiscence at the bone crest due to shifting a tooth towards an area with exceptionally thin bone (Fig. 4).(15,16) Considering the buccal periodontal structure as a predisposing factor for gingival recession, it can be asserted that proper orthodontic planning, customized to specific regions with thin buccal bone plates, can prevent gingival recession from occurring.(12)



**Fig. 4.** Alveolar dehiscences present over the root surfaces.(17)

## ***2.3. Pathological Factors***

### ***2.3.1. Trauma***

Various forms of trauma, such as traumatic toothbrushing, improper flossing, perioral/intraoral piercing, aberrant frenal attachment, occlusal injury and tobacco chewing, are reported to be an etiological factor of recession.(2,13)

Flossing trauma lesions frequently manifest in highly motivated patients who didn't receive proper instruction about flossing techniques. Confirming the

diagnosis of these injuries can often be achieved by having patients demonstrate their oral hygiene procedures. The lesions can be reversible or irreversible depending on the connective tissue involvement. Red clefts are reversible where the lesions are confined within the connective tissue, while white clefts are irreversible due to involvement of the whole connective tissue thickness (Fig. 5).(13)



**Fig. 5.** Red clefts (upper row) and white clefts (lower row)(13)

The impact of toothbrushing has been investigated by numerous researchers, and there is a general consensus that vigorous or improper use of the toothbrush can lead to recession.(2) According to Heasman et al. the main toothbrushing factors linked to the initiation and advancement of gingival recession are the frequency and method of brushing. Secondary factors encompass the frequency of changing toothbrushes and the hardness of bristles.(18)

### ***2.3.2. Periodontal Disease***

Periodontal disease, such as plaque-induced gingivitis and periodontitis, can be regarded as a significant factor linked to gingival recession, particularly for teeth with thin gingival tissues and/or mucogingival conditions. Inadequate cleaning can result in localized inflammation that the delicate tissue cannot endure, potentially leading to gingival recession in patients with thin phenotype (Fig. 6).(7,17)



**Fig. 6.** Recession as a result of inflammation.(17)

Periodontal disease management is frequently associated with the occurrence of gingival recession. The reduction in probing depth observed after successful nonsurgical treatment of periodontal disease is a combination of both gingival recession and gain in attachment level. Anterior teeth, thin and edematous tissues are likely to exhibit more gingival recession in response to periodontal treatment.(17)

### **3. Management**

Various root coverage techniques have been experimented in order to shift the position of the gingival margin coronally. Improving esthetics, alleviating dental hypersensitivity, and preventing caries and NCCLs are commonly cited as the primary indications of root covering procedures.(19) Regardless of the surgical approach, the ultimate objective of a root coverage procedure is achieving complete coverage of the recession defect and the optimal integration of the covering tissue with the adjacent soft tissue.(20)

The decision-making process for selecting a surgical technique to cover a root recession should involve a thorough assessment of the local anatomical characteristics and the patient's preferences. Local characteristics need to be considered can be cited as: the dimensions of root exposure, the number of recession defects, the height and width of the inter-dental soft tissue, the qualities of the keratinized tissues around the root exposure, the presence of root caries or cervical abrasions, the vestibulum depth and muscle insertions.(21)

#### **3.1. The Free Gingival Graft**

The free gingival graft (FGG) is defined as a soft tissue graft taken from the palate, including the overlying epithelium. It was introduced to increase the keratinized tissue. Many studies investigated the healing process and risk factors affecting the outcomes of the procedure.(22)

There has been two major limitations reported regarding FGGs. The first one is that the graft undergoes significant shrinkage (around 30%) during the healing process, resulting in the need for harvesting a wider graft than the recipient site. This makes postoperative discomfort and complications at the donor site inevitable. The other limitation is the poor esthetic outcome of the FGG in terms of color, surface and scarring. However in the presence of mucogingival defects, FGG is still indicated in order to re-establish keratinized tissue and gingival thickness.(22,23)

Agudio et al. compared the long-term efficacy of a FGG with contralateral untreated sites and reported stabilisation of the gingival margin and prevention of further recessions after FGG, while untreated sites exhibiting increased recession depth or development of recessions.(24)

### ***3.2. Coronally Advanced Flap and Its Combinations***

Coronally advanced flap(CAF) procedures are based on the coronal shift of the soft tissues located apically to the recession on the exposed root surface to cover the exposed root.(25) It can be applied for the treatment of both single or multiple recession defects (Fig. 7).(19) Cairo et al. confirmed its safety and reliability as a periodontal plastic surgery and its association with consistent recession reduction and frequently with complete root coverage(CRC).(20)

De Sanctis & Zucchelli recently proposed a modified coronally advanced flap technique for single recession sites and tested the technique on isolated recession-type defects in the upper jaw, in a case-series study, reporting a 97% degree of root coverage and 85% complete root coverage.(21)



**Fig. 7.** Coronally advanced flap for multiple recessions.

Several techniques and graft substitutes have been proposed in combination with CAF in order to achieve best results in terms of esthetics and function. Among them, connective tissue graft(CTF) + CAF demonstrate the strongest potential of achieving complete root coverage, together with better esthetic results(Fig. 8).(20,22) It is postulated that CTG functions as a biological filler, enhancing the adaptation and stability of the flap to the root during the early stages of wound repair. Consequently, the gingival phenotype becomes thicker, increasing the potential of achieving complete root coverage.(26) A 5-year long-term clinical study concluded that CAF+CTG achieved better outcomes than CAF alone in terms of CRC.(27) A randomized controlled clinical trial by Cairo et al. established similar effectiveness of both CAF+CTG and CAF alone regarding CRC after 12 months. They proposed CTG addition under CAF in cases where a thin phenotype is present.(28)



**Fig. 8.** Coronally advanced flap and connective tissue graft.(25)

Various connective tissue substitutes have been suggested to overcome patient morbidity and requirement for a second surgical site.(29) Collagen matrices in that matter are claimed to have advantages such as not requiring human donor, thereby having no risk of disease transmission. Another positive factor about collagen matrix is that there is no size limitations. Tonetti et al. concluded that there are significantly more patient benefits deriving from the avoidance of autologous soft tissue grafting by replacing them with collagen matrix-based devices in multiple adjacent recessions.(30) Mathias-Santamaria et al. evaluated the use of collagen matrix in combination with CAF to treat gingival recession with NCCL. Comparison of the two approaches, as in CAF alone and CAF+collagen matrix(CM), resulted in no significant deference

between the two approaches in terms of coverage parameters. Nevertheless they reported a significantly higher increase in keratinized tissue width and keratinized tissue thickness for the CAF+CM group.(31)

#### 4. Conclusion

Gingival recession is highly prevalent, posing an elevated risk for root caries and potential disruptions to patient comfort, function, and esthetics. While addressing the causes of gingival recession can reduce its incidence and severity, effective management and prevention can still be challenging. Further development and improvement of clinical trials with a primary focus on patient outcomes, especially in terms of esthetics and morbidity are required in order to advance in the treatment of gingival recessions.

#### References

1. Jepsen S, Caton JG, Albandar JM, Bissada NF, Bouchard P, Cortellini P, et al. Periodontal manifestations of systemic diseases and developmental and acquired conditions: Consensus report of workgroup 3 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *J Clin Periodontol*. 2018;45(December 2017):S219–29.
2. Kassab MM, Cohen RE. The etiology and prevalence of gingival recession. *Jada*. 2003;134(2):220–5.
3. Seong J, Bartlett D, Newcombe RG, Claydon NCA, Hellin N, West NX. Prevalence of gingival recession and study of associated related factors in young UK adults. *J Dent*. 2018;76(March):58–67.
4. Heasman PA, Ritchie M, Asuni A, Gavillet E, Simonsen JL, Nyvad B. Gingival recession and root caries in the ageing population: a critical evaluation of treatments. *J Clin Periodontol*. 2017;44:S178–93.
5. Vignoletti F, Di Martino M, Clementini M, Di Domenico GL, de Sanctis M. Prevalence and risk indicators of gingival recessions in an Italian school of dentistry and dental hygiene: a cross-sectional study. *Clin Oral Investig*. 2020;24(2):991–1000.
6. Teixeira DNR, Zeola LF, Machado AC, Gomes RR, Souza PG, Mendes DC, et al. Relationship between noncarious cervical lesions, cervical dentin hypersensitivity, gingival recession, and associated risk factors: A cross-sectional study. *J Dent [Internet]*. 2018;76(June):93–7. Available from: <https://doi.org/10.1016/j.jdent.2018.06.017>



7. Merijohn GK. Management and prevention of gingival recession. *Periodontol 2000*. 2016;71(1):228–42.

8. Imber JC, Kasaj A. Treatment of Gingival Recession: When and How? *Int Dent J [Internet]*. 2021;71(3):178–87. Available from: <https://doi.org/10.1111/idj.12617>

9. Zucchelli G, Gori G, Mele M, Stefanini M, Mazzotti C, Marzadori M, et al. Non-Carious Cervical Lesions Associated With Gingival Recessions: A Decision-Making Process. *J Periodontol*. 2011;82(12):1713–24.

10. Kim DM, Bassir SH, Nguyen TT. Effect of gingival phenotype on the maintenance of periodontal health : An American Academy of Periodontology best evidence review. *J Periodontology*. 2020;91:311–38.

11. Maroso FB, Gaio EJ, Rösing CK, Fernandes MI. Correlation between gingival thickness and gingival recession in humans. *acta Odontol Latinoam*. 2015;28(2):162–6.

12. Jati AS, Furquim LZ, Consolaro A. Gingival recession : its causes and types , and the importance of orthodontic treatment. *Dental Press J Orthod*. 2016;21(3):18–29.

13. Zucchelli G, Mounssif I. Periodontal plastic surgery. *Periodontol 2000*. 2015;68(205):333–68.

14. Closs QL, Branco P, Rizzato SD, Raveli DB, Rösing CK. Gingival margin alterations and the pre-orthodontic treatment amount of keratinized gingiva. *Braz Oral Res*. 2007;21(1):58–63.

15. Liu Y, Li CX, Nie J, Mi CB, Li YM. Interactions between Orthodontic Treatment and Gingival Tissue. *Chinese J Dent Res*. 2023;26(1):11–8.

16. Joss-Vassalli I, Grebenstein C, Topouzelis N, Sculean A, Katsaros C. Orthodontic therapy and gingival recession : a systematic review. *Orthod Craniofacial Res*. 2010;13:127–41.

17. Baker P. Gingival Recession – causes and management. *Prim Dent J*. 2019;8(8):40–7.

18. Heasman PA, Holliday R, Bryant A, Preshaw PM. Evidence for the occurrence of gingival recession and non- carious cervical lesions as a consequence of traumatic toothbrushing. *J Clin Periodontol*. 2015;42(16):237–55.

19. Stefanini M, Marzadori M, Aroca S, Felice P, Sangiorgi M, Zucchelli G. Decision making in root- coverage procedures for the esthetic outcome. *Periodontol 2000*. 2018;0(14):1–11.

20. Cairo F, Pagliaro U, Nieri M. Treatment of gingival recession with coronally advanced flap procedures : a systematic review. *J Clin Periodontol*. 2008;35(8):136–62.

21. de Santics M, Zucchelli G. Coronally advanced flap : a modified surgical approach for isolated recession-type defects. *J Clin Periodontol.* 2007;34:262–8.
22. Zucchelli G, Tavelli L, Mcguire MK, Rasperini G, Feinberg SE, Wang H-L, et al. Autogenous soft tissue grafting for periodontal and peri-implant plastic surgical reconstruction. *J Periodontology.* 2019;00:1–8.
23. Zuhr O, Baumer D, Hürzeler M. The addition of soft tissue replacement grafts in plastic periodontal and implant surgery : critical elements in design and execution. *J Clin Periodontol.* 2014;41(15):123–42.
24. Agudio G, Cortellini P, Buti J, Pini-Prato G. Periodontal Conditions of Sites Treated With Gingival-Augmentation Surgery Compared to Untreated Contralateral Homologous Sites : A 18- to 35-Year Long-Term Study. *J Periodontology.* 2016;87(12):1371–8.
25. Cairo F. Periodontal plastic surgery of gingival recessions at single and multiple teeth. *Periodontol 2000.* 2017;75(40):296–316.
26. Cairo F, Cortellini P, Pilloni A, Nieri M, Cincinelli S, Amunni F, et al. Clinical Efficacy of Coronally Advanced Flap with or without Connective Tissue Graft for the treatment of Multiple Adjacent Gingival Recessions in the Aesthetic Area. A Randomized Controlled Clinical Trial. *J Clin Periodontol.* 2016;43(10):849–56.
27. Gp P, Cairo F, Nieri M, Franceschi D, Rotundo R, Coronally CP, et al. Coronally advanced flap versus connective tissue graft in the treatment of multiple gingival recessions : a split-mouth study with a 5-year follow-up. 2010;644–50.
28. Cairo F, Nieri M, Tonetti M. Coronally Advanced Flap and Composite Restoration of the Enamel with or without Connective Tissue Graft for the treatment of single maxillary gingival recession with non-carious cervical lesion. A randomized controlled clinical trial. *J Clin Med.* 2020;47(3):362–71.
29. Tavelli L, Ravidà A, Amo FS, Tattan M, Wang H. Comparison between Subepithelial Connective Tissue Graft and De-epithelialized Gingival Graft : A systematic review and a meta-analysis. *J Int Acad Periodontol.* 2019;21(2):82–96.
30. Tonetti MS, Cortellini P, Pellegrini G, Nieri M, Bonaccini D, Allegri M, et al. Xenogenic collagen matrix or autologous connective tissue graft as adjunct to coronally advanced flaps for coverage of multiple adjacent gingival recession : Randomized trial assessing non- inferiority in root coverage and superiority in oral health- . *J Clin Periodontol.* 2018;45:78–88.
31. Mathias-Santamaria IF, Silveira CA, Rossato A, de Melo MAS, Bresciani E, Santamaria MP. Single gingival recession associated with non-carious cervical lesion treated by partial restoration and coronally advanced



flap with or without xenogenous collagen matrix. A randomized clinical trial evaluating the coverage procedures and restorative pro. J Periodontol. 2022;93(4):504–14.

## CHAPTER III

# PROSTHETIC TECHNIQUES USED TO IMPROVE GINGIVAL AESTHETICS

**Özlem SARAÇ ATAGÜN**

*(Asst. Prof.), Department of Periodontology, Gülhane Faculty of Dentistry,  
University of Health Sciences, Ankara, Turkey,  
E-mail: ozlemsarac2806@hotmail.com,  
ORCID: 0000-0002-2964-8244*

### 1. Introduction

**T**he development of both surgical and prosthetic treatments targeted at enhancing or maintaining aesthetic traits is a result of growing patient and physician awareness of the significance of gingival and smile aesthetics. (1) Buccal cervical cavities that are concurrent with unsightly recession defects may need to be restored to prevent future loss of tooth surface, lower plaque retention, or lessen dentinal sensitivity. The loss of papillae may also be unattractive when recession is more widespread, particularly in individuals who have received periodontal therapy. The term “black triangle syndrome” is used for these situations.(2) It is challenging to reconstruct this missing interdental papilla surgically. According to Tarnow et al., the interdental papilla should form when there is a minimum of 5 mm between the contact site and alveolar crest. (3) Technique-sensitive, recommended surgical procedures for reconstructing gingival architecture around recession or alveolar abnormalities may call for a transplant from a different surgical site, which would increase morbidity.(4) Bone grafting may be necessary to promote implant recovery in edentulous regions that have obvious vertical and horizontal abnormalities, especially in the aesthetic zone.(5) It should be remembered that surgical treatments are the first choice when the patient accepts the possible complications and treatment challenges and is systemically fit.

A treatment option is to match gum and tooth proportions with fixed or removable methods to provide an aesthetic harmony to the smile. However, the presence of teeth of different lengths in the aesthetic zone, manifested by the 'high smile line', is a factor that makes prosthetic rehabilitation very difficult. A variety of surgical procedures have been recommended to enhance soft tissue volume if there is vertical soft tissue loss in edentulous gaps before the placement of a permanent bridge.(6) Although the primary goal of these approaches is to enhance the pontic's emerging profile, it should be highlighted that the morbidity of a potential second surgical site is a crucial factor. Even after surgery, the outcomes might occasionally be unexpected and inadequate in terms of appearance and functionality.(7)

This chapter will describe both conventional and modern methods used in the prosthetic management of soft tissue aesthetics.

## **2. Restorations With a Gingival Color on Natural Teeth**

In cases where gingival recession is localized, the etiology of the recession must first be determined and addressed before any restoration is performed.(8) As the effects of gingivitis can be challenging to control following restoration, it is crucial to establish and maintain gingival health. Non-carious cervical lesions that are left untreated could cause hypersensitivity by exposing the root dentine. Cervical lesions with caries may progress and cause pulp exposure.(9)

The first description of the use of gingivally colored composites in the treatment of a cervical deformity was made by Zalkind and Hochman.(10) In recent years, gingivally-colored resins, composites and ceramics have been frequently used to repair gum recessions.(11) To avoid plaque retention, it's critical to pay close attention to the cervical shape of the restoration. This can be done by employing methods for applying the composite with the right tools, effective moisture management, and curing.(12) A pseudo gingival sulcus can be produced over the restoration.

When close to the gingival sulcus, moisture control might make composite restorations difficult. On the other hand, the clinician controls the form, color and characterization of the restoration directly.(13) In cases where the enamel is still sound, the bond can be predicted. Staining colours that can be utilized for characterization are included in composite kit packages. Composites also have the benefit of being easily replaced and removed with little harm to the underlying tooth tissue. In order to enhance cleanability and characterization, the composite can also be easily adjusted after curing.(1, 13) This technique's

drawback is that it can eventually necessitate a new restoration owing to color instability.

Another technique for enhancing the proportions of individual teeth is the use of gingival porcelain in conjunction with conventional tooth-colored porcelain.(14, 15) The formation of a margin on the root dentine and the removal of sound tooth tissue may be necessary for these restorations. At the same time, the marginal interface of the restoration must be carefully evaluated to avoid further recession. The doctor cannot assess the final restoration's and shade's cleanability because they have no direct control over the restoration's form. Furthermore it could be challenging to communicate color and character to the technician. It is challenging to refurbish, and replacement work may require extensive removal of healthy tooth tissue, endangering the periodontium. The cement lute may intrude into the cervical gingival margin, resulting in trauma and subsequent plaque retention.(7, 14, 15)

### **3. Implant Restorations with Gingival Coloring**

The maxillary esthetic zone presents various difficulties for implant rehabilitation. Using dental porcelain that is gingiva-colored allows for anterior implant restorations to provide aesthetically pleasing results that are clinically acceptable.(16) Applying pink gingiva-colored porcelain to the cervical region of an implant-fixed dental prosthesis can help restore interproximal papillae, pleasing mucogingival curves, and natural crown ratios while obviating the need for difficult surgical procedures. The smooth, homogeneous interface between the pink porcelain gingiva and the residual tissues may boost patient comfort, simplifying and shortening the cost and length of treatment.(17)

Due to the requirement to preserve a crown insertion path in relation to neighboring teeth, the degree of application of gingivally colored porcelain in respect to gingival embrasures may be constrained. In contrast, the use of customized abutments with gingival porcelain gives more latitude for filling gingival embrasure spaces where interproximal papillae are absent since there are fewer restrictions on the abutment's insertion path than there are for a crown. (18)

Utilizing gingiva-colored pink porcelain to replace hard and soft tissues has the potential to produce an aesthetic result and boost patient satisfaction. The smile line, the extent of the prosthetic rehabilitation, the anatomical area, the vertical and horizontal transition between the prosthesis and the natural gingiva, the contact surface, the framework material, and the color all have an impact

on the achievement of satisfactory esthetic results when using gingiva-colored pink porcelain.(19) However, there isn't much variety in the shade guides now available for pink dental ceramic materials, and the pink tones are frequently oversaturated.(18)

The magnitude of the smile line may limit the usage of pink porcelain, and the procedure is highly recommended for low to moderate smile lines. It is challenging to conceal the transition when the patient has a prominent smile line. When creating a cervical prosthesis out of gum-colored pink porcelain, careful preparation should be made throughout diagnostic planning, laboratory design, and clinical processes. Prosthetic maintenance and cleaning might also be a problem. Convex cervical outlet profiles and clearly specified design guidelines should be used to provide long-term cleanability.(18, 19)

#### **4. Soft Tissue Aesthetics for Multiple Teeth and Edentulous Spans**

##### ***4.1. Gingival Prosthesis***

The use of a “gingival prosthesis” can significantly enhance aesthetics when there is generalized tissue loss due to periodontal disease or when there is a substantial variance in gingival border heights, especially in individuals with a high smile line.(20) Resin, silicone, or copolyamide can all be used to create gingival prosthesis, and it has been demonstrated that all three are satisfactory in terms of color stability.(21) Gingival prosthesis have been demonstrated to support the lip and prevent food trapping. Additionally could be successful for resolving phonetic issues.(22) With a gingival prosthesis, air leakage through interdental voids that affect phonetics can be avoided in cases of marked interdental papillae loss.(1)

Before beginning any serious treatment, it is feasible to show the patient a waxed-up result or even place a try-in prosthesis directly in the mouth for evaluation. Its use may be restricted to specific clinical scenarios when oral hygiene can be maintained, the intended aesthetic outcome is feasible or esthetics are not a priority, and a fixed prosthesis is already being planned for the region. With a removable prosthesis, more tissue can be restored while still being able to be cleaned properly. With detachable prosthodontic materials, it is simpler to shape an optimal contour, and lost tissue can be restored without interfering with the other dental parts.(20) When replacing fixed teeth, a mixed strategy that combines both permanent and detachable components may be used, including the use of dental attachments to improve support and retention.(23)

They might be built immediately at the chair side or indirectly. The indirect method calls for an impression that, ideally, uses a specialized tray, catches the interproximal spaces and the whole depth of the sulcus for the span of the proposed prosthesis.(20) Before providing, a frenectomy can be considered for patients who have a low labial frenum; otherwise, it may be more likely that the thin acrylic in this location will shatter midline.(1) A gingival prosthesis can be used to administer and sustain contact with desensitizing chemicals against root dentine to assist alleviate symptoms of dentine hypersensitivity, a recognized side effect of periodontal therapy.(24) A gingival prosthesis could distribute fluoride formulations and help remineralize early carious lesions in individuals prone to root caries, such as those with head and neck cancer and who received radiation treatment.(25)

#### ***4.2. Gingival Porcelain on Fixed Bridges***

Gingivally colored materials can be used to create permanent bridgework to correct obvious vertical flaws in edentulous spaces and improve vertical and horizontal pontic proportions. In fact, to maximize the aesthetics of the final restoration, patients who have undergone surgical procedures to improve soft tissue topography might still need to employ gingivally colored porcelain.(26)

The integration of gingival porcelain in the final restoration should be considered at the planning stage while keeping in mind the constraints of adjunctive surgical procedures. This preparation may include a diagnostic wax-up or a CT scan for potential gingival and dental restorations.(27)

The use of gingivally colored porcelain can provide the impression of interdental papilla where many implants have been positioned as optimally as possible. Where there are implant pier abutments or the implants are placed near together, it might be challenging to maintain.(1)

The intersection of the gingivally colored porcelain with the natural gingival tissue may be apparent in individuals with a high smile line, possibly not acceptable for the aesthetically conscious patient. Furthermore, the majority of gingivally colored porcelain may produce a large ridge lap if the implant position is relatively palatal in the upper anterior region, which can be challenging to clean. By developing a laboratory temporary with gingival material incorporated for examination by the patient, dentist, and technical staff before definitive restoration, these issues can be found and resolved.(1)

### ***4.3. Soft Tissue Management in Partial Denture Patients***

Soft tissue appearance in patients with partial or total removable prostheses is an important issue with certain principles. The characterization of the denture base, the use of proper root contouring, and stippling have been identified as features in the development of a natural-looking complete denture when considering aesthetics in complete denture construction.(28) The inclusion of staining, in addition to pseudo recession and gingival inflammation, into the denture teeth has also been recorded for patients who want a prosthesis that is identical to their prior aged dentition. Pre-extraction information, photographs, and study models can be used to recreate tooth placements, gingival contours, and measurements in immediate replacement dentures and the final prosthesis when the prior natural dentition has recently been extracted.(29)

### ***4.4. Fixed and Removable Implant Prosthesis***

Implant-supported overdentures and implant-supported fixed complete dentures are both successful treatment modalities.(30) There are a few important considerations when making these prostheses to maintain gingival aesthetics.

Instead of tooth-colored composite resin, gingivally colored composite can be used to hide abutment screw holes that are placed lingual to prosthetic teeth. Patients who have a fixed implant prosthesis may experience the same issues with phonetics as those patients who have long-term periodontal disease. Using detachable, directly or indirectly manufactured gingival prostheses can mask multi-unit abutments and reduce unintentional air loss during speech. These restorations go through a similar clinical and analytical process as gingival prosthesis.(31)

## **5. Conclusions**

Proper prosthetic component selection is crucial for aesthetics. Utilizing pink-colored composites is an innovative technique to treat papillary deficit and recession in people without surgery in order to meet their cosmetic goals and preserve oral cleanliness. Using gingival ceramic and ceramic abutments are some prosthetic adjustments to optimize esthetic in implant prosthesis.

## **References**

1. Alani A, Maglad A, Nohl F. The prosthetic management of gingival aesthetics. *British dental journal*. 2011;210(2):63-9.

2. Clark D. "Restoratively driven papilla regeneration: correcting the dreaded 'black triangle'". *Texas dental journal*. 2008;125(11):1112-5.
3. Tarnow DP, Magner AW, Fletcher P. The effect of the distance from the contact point to the crest of bone on the presence or absence of the interproximal dental papilla. *Journal of periodontology*. 1992;63(12):995-6.
4. Imber JC, Kasaj A. Treatment of Gingival Recession: When and How? *International dental journal*. 2021;71(3):178-87.
5. Donkiewicz P, Benz K, Kloss-Brandstätter A, Jackowski J. Survival Rates of Dental Implants in Autogenous and Allogeneic Bone Blocks: A Systematic Review. *Medicina (Kaunas, Lithuania)*. 2021;57(12).
6. Mishra N, Singh BP, Rao J, Rastogi P. Improving prosthetic prognosis by connective tissue ridge augmentation of alveolar ridge. *Indian journal of dental research : official publication of Indian Society for Dental Research*. 2010;21(1):129-31.
7. Sonune SJ, Kumar S, Jadhav MS, Martande S. Gingival-colored Porcelain: A Clinical Report of an Esthetic-prosthetic Paradigm. *International journal of applied & basic medical research*. 2017;7(4):275-7.
8. Zucchelli G, Mounssif I. Periodontal plastic surgery. *Periodontology* 2000. 2015;68(1):333-68.
9. Donovan TE. Clinical management of root caries. *Journal (Indiana Dental Association)*. 2009;88(1):23-4.
10. Zalkind M, Hochman N. Alternative method of conservative esthetic treatment for gingival recession. *The Journal of prosthetic dentistry*. 1997;77(6):561-3.
11. Miletic V, Trifković B, Stamenković D, Tango RN, Paravina RD. Effects of staining and artificial aging on optical properties of gingiva-colored resin-based restorative materials. *Clinical oral investigations*. 2022;26(11):6817-27.
12. Aydın N, Topçu FT, Karaoğlanoğlu S, Oktay EA, Erdemir U. Effect of finishing and polishing systems on the surface roughness and color change of composite resins. *Journal of clinical and experimental dentistry*. 2021;13(5):e446-e54.
13. Lucena C, Benavides-Reyes C, Ruiz-López J, Tejada-Casado M, Pulgar R, Pérez MM. Relevant optical properties for gingiva-colored resin-based composites. *Journal of dentistry*. 2022;126:104316.
14. Hannon SM, Colvin CJ, Zurek DJ. Selective use of gingival-toned ceramics: case reports. *Quintessence international (Berlin, Germany : 1985)*. 1994;25(4):233-8.



15. Capa N. An alternative treatment approach to gingival recession: gingiva-colored partial porcelain veneers: a clinical report. *The Journal of prosthetic dentistry*. 2007;98(2):82-4.
16. Kamalakidis S, Paniz G, Kang KH, Hirayama H. Nonsurgical management of soft tissue deficiencies for anterior single implant-supported restorations: a clinical report. *The Journal of prosthetic dentistry*. 2007;97(1):1-5.
17. Priest GF, Lindke L. Gingival-colored porcelain for implant-supported prostheses in the aesthetic zone. *Practical periodontics and aesthetic dentistry : PPAD*. 1998;10(9):1231-40; quiz 42.
18. Paspaspyridakos P, Amin S, El-Rafie K, Weber HP. Technique to Match Gingival Shade when Using Pink Ceramics for Anterior Fixed Implant Prostheses. *Journal of prosthodontics : official journal of the American College of Prosthodontists*. 2018;27(3):311-3.
19. Viana PC, Kovacs Z, Correia A. Purpose of esthetic risk assessment in prosthetic rehabilitations with gingiva-shade ceramics. *The international journal of esthetic dentistry*. 2014;9(4):480-9.
20. Barzilay I, Irene T. Gingival prostheses--a review. *Journal (Canadian Dental Association)*. 2003;69(2):74-8.
21. Lai YL, Lui HF, Lee SY. In vitro color stability, stain resistance, and water sorption of four removable gingival flange materials. *The Journal of prosthetic dentistry*. 2003;90(3):293-300.
22. Carvalho W, Barboza EP, Gouvea CV. The use of porcelain laminate veneers and a removable gingival prosthesis for a periodontally compromised patient: a clinical report. *The Journal of prosthetic dentistry*. 2005;93(4):315-7.
23. Brygider RM. Precision attachment-retained gingival veneers for fixed implant prostheses. *The Journal of prosthetic dentistry*. 1991;65(1):118-22.
24. Greene PR. The flexible gingival mask: an aesthetic solution in periodontal practice. *British dental journal*. 1998;184(11):536-40.
25. Aguiar GP, Jham BC, Magalhães CS, Sensi LG, Freire AR. A review of the biological and clinical aspects of radiation caries. *The journal of contemporary dental practice*. 2009;10(4):83-9.
26. Palmer RM, Palmer PJ, Newton JT. Dealing with esthetic demands in the anterior maxilla. *Periodontology 2000*. 2003;33:105-18.
27. Salama M, Coachman C, Garber D, Calamita M, Salama H, Cabral G. Prosthetic gingival reconstruction in the fixed partial restoration. Part 2: diagnosis and treatment planning. *The International journal of periodontics & restorative dentistry*. 2009;29(6):573-81.

28. Waliszewski M, Shor A, Brudvik J, Raigrodski AJ. A survey of edentulous patient preference among different denture esthetic concepts. *Journal of esthetic and restorative dentistry : official publication of the American Academy of Esthetic Dentistry [et al]*. 2006;18(6):352-68; discussion 69.

29. Ali A, Hollisey-McLean D. Improving aesthetics in patients with complete dentures. *Dental update*. 1999;26(5):198-202.

30. Tsigarida A, Chochlidakis K. A Comparison Between Fixed and Removable Mandibular Implant-Supported Full-Arch Prostheses: An Overview of Systematic Reviews. *The International journal of prosthodontics*. 2021;34:s85-s92.

31. Délben JA, Goiato MC, Gennari-Filho H, Gonçalves Assunção W, Dos Santos DM. Esthetics in implant-supported prostheses: a literature review. *The Journal of oral implantology*. 2012;38(6):718-22.



## CHAPTER IV

# DIAGNOSIS AND TREATMENT PLANNING IN PERIODONTICS

**Pınar ŞAYAN**

*(Dental Specialist), Department of Periodontology, Adana, Turkey*

*E-mail: pnrsayan44@gmail.com*

*ORCID: 0000-0001-7620-1181*

### **1. Introduction**

**G**ingiva, periodontal ligament, root cementum, and alveolar bone proper are the tissues that compose the periodontium. Periodontitis is an inflammation based chronic disease affecting periodontium early seen in gingiva and if continues noticed with destruction of the connective tissue attachment, characterized slowly progressing and painless. At first prevention, then treatment of periodontal diseases are based on accurate diagnosis, elimination of causative factors, risk management and compensation of the detrimental effects of disease. Accurate periodontal diagnosis is essential for sensible treatment planning. It is necessary to understand the needs and expectations of the patient for care. Optimal clinical results can only be obtained if the requirements of the patient can be matched with the realistic assessment of the condition and the expected outcomes of the treatment. Hence the aspirations of the patient must be taken seriously and acknowledged in the assessment in order to ensure consistency with the clinical situation. Accurate diagnosis can be made through certain anamnesis consist of patients verbal history, systemic status, clinical and radiographic examination and laboratory tests as needed. The data obtained as a result of the anamnesis taken from the patient should be documented. (1,2)

Bleeding, mobility of teeth, calculus (Fig.1), diastemas according to pathologic migration, breath malodor, masticatory dysfunctions, aesthetic problems, bad mouth taste, etc. are complaints of patients who have periodontal diseases. (1,3)



(Fig.1)(Periodontal disease due to intense calculus and plaque accumulation.)

## 2. Examination

### 2.1. Dental History

In the dental history bleeding of the gums, tooth mobility, itching of the gums, pain (necrotizing periodontal diseases, etc.)(Fig.2), burning of the gums (desquamative gingivitis, etc.), sensitivity during chewing, cold and hot sensitivity, reasons to go to a dentist before, history of orthodontic treatment, cause of bleeding, clenching and grinding, etc. should be questioned. (1-3)



(Fig.2)(Necrotizing periodontal diseases are very painful, in comparison to the painless concept of periodontal diseases. The ulcers are coated with a yellowish-white or grayish slough called a pseudomembrane.)

## 2.2. Medical History

Systemic status should be asked to the patient during medical history.

- Heart diseases,
- Diabetes mellitus,
- hypertension (Fig.3),
- chemotherapy,
- immunosuppressive therapy,
- drugs used (bisphosphonates, phenytoin (Fig.3), calcium channel blockers (Fig.4), etc.),
- allergy,
- bleeding disorders,
- hormonal changes,
- pregnancy,
- nutritional deficiencies (Iron, folic acid, Vitamin C, D, Calcium, etc.)
- stress and obesity,
- host response,
- family story,
- previous operations, etc. (1,2,4)

The possibility of underlying systemic factors should be considered when unusual gingival or periodontal problems are encountered that cannot be explained locally. Oral findings of systemic disease should be analyzed and, if necessary, the patients should be consulted with their physicians. Medical laboratory tests are also helpful in the diagnosis of systemic diseases. (1,4)



(Fig.3)(Generalized papillary overgrowth in a patient using a calcium channel blocker for hypertension disease.)



(Fig.4)(Drug-induced inflammatory gingival overgrowth in a patient using phenytoin medication and inadequate oral hygiene)

### ***2.3. Local Factors***

Local factors should be investigated in periodontal diseases.

- Bacterial plaque is the main cause of the gingival inflammation.
- Infection,
- poor oral hygiene,
- radiation therapy,
- impacted third molars,
- oral jewelry,
- toothbrush trauma,
- chemical irritation,
- orthodontic therapy,
- piercing,
- aberrant frenal attachments,
- dry mouth,
- food impaction,
- poor restorations and prosthesis,



-habits (mouth breathing, smoking, smokeless tobacco, bruxism, clenching and grinding, wedging toothpicks between teeth, application of fingernail pressure against gingiva, occupational habits, and dietary habits, etc. are some of them.) etc. are predisposing factors for periodontal diseases. (1,2,5,6)

#### ***2.4. Radiographic Examination***

Radiographs play a complementary role in the history and clinical examination findings. With radiographs; the bone level, pattern of bone destruction and periodontal ligament space width, as well as radiodensity, trabecular pattern, and marginal contour of the interdental bone furcation involvement, periodontal abscess and dental implants can be evaluated. Orthopantomographs provide general information about dental arch, developmental anomalies, pathologic lesions of surrounding structures but are not suitable for certain diagnosis of periodontal diseases. The most used technique in terms of diagnosis and follow-up is the periapical films using parallel technique. Cone-Beam Computed Tomography screening is a valuable three-dimensional imaging technique for the diagnosis of intrabony defects, fenestrations and dehiscences, furcation involvement and buccal / lingual bone destruction, and implant site imaging. (1,2)

The diagnostic models and intraoral photographs obtained from the patient are also to be used in the first evaluation. (1,2)



(Fig.5)(Diabetes Mellitus with deep periodontal pockets, gingival growth, papilloma and plasma cell gingivitis.)





(Fig.6)(Aggressive periodontitis is characterized by alveolar bone loss, which progresses very rapidly despite the absence of plaque and calculus, in patients at a young age, without underlying systemic disease.)

### ***2.5. Clinical Examination***

- Extraoral examination (Temporomandibular junction, etc.)
- Oral cavity (Lips, floor of mouth, tongue, soft and hard palate, oropharynx, etc.)
- Oral hygiene (Plaque, calculus, teeth stains, materia alba, etc.)
- Breath malodor (May be caused by intraoral and extraoral areas.)

- Teeth (Tooth mobility, pathologic migration of the teeth, wasting disease of the teeth, sensitivity to percussion, proximal contact relations, etc.)
- Implants
- Periodontium (1,2,6-8)

### ***2.6. Periodontal Examination***

Probing should be performed with a scaled, blunt periodontal probe. To detect the deepest penetration areas around each surface of the tooth/root, the probe should be applied with a circumferential ‘walked’ motion. A force of the weight of the probe, approximately 0.75 N, has been found to be correct. The probing angle should be positioned as parallel to the tooth surface as possible.

Probing periimplant tissues should be performed with lighter forces than normal dental probing to avoid damaging these tissues, a force of 0.25 N is recommended. Curved periodontal probes of 3 mm (Nabers furcation probe) are useful for clinical detection of bone and attachment loss in furcation involvement.

Slight degree of mobility due to periodontal ligament is seen in healthy teeth which is known as physiologic tooth mobility. Mobility beyond the physiological limit is considered as an indicator of periodontal disease severity and also helps to determine the prognosis of the disease. Tooth mobility is evaluated by a metal instrument and one finger. Mobility is scored according to the Miller Index as follows;

Mobility 1: Slightly more than normal

Mobility 2: Moderately more than normal

Mobility 3: Severe mobility and vertical mobility in the facio-lingual and mesio-distal directions. (1,2,7,8)

Periodontal charting records the patient’s periodontal condition and helps for correct diagnosis. Also the patient’s charts are guide for dentists on recall visits and the response of the periodontal areas to treatment is evaluated according to the comparison of the charts.

Periodontal examination should be systematic, should not begin immediately with insertion the periodontal probe into the gingival crevice and it should begin with visual assessment of biofilm and calculus accumulation, changes in inflammation of the soft tissue, and assessment of marginal bleeding. (1,2,7,9,10)

After the visual assessment of periodontal status, the gingival crevice should be carefully probed to understand the subgingival area, the response of the gingival tissue to probing is observed in terms of resistance to probe penetration, depth of probe penetration, bleeding on probing, and pain on probing. Attachment loss, attachment level, attached gingiva, periodontal pockets, furcation involvement, periodontal abscess should be evaluated and recorded to the periodontal chart.

Attachment level is defined as the location where the dentogingival junction begins coronally on a tooth. Clinical evaluation of the attachment level has become routine in the evaluation of clinical response in periodontal treatment. (1-3,10-12)

### 3. Treatment Planning

Treatments should be considered as immediate, intermediate, and long-term goals.

The immediate goals are to eliminate infectious problems, which may hinder the general health of the patient, and to restore the oral cavity to a healthy state. Patient education about periodontal procedures, endodontics, caries control, oral surgery and treatment of oral mucosal membrane pathologies may be required, if necessary, referring to other dental and medical specialties. Intermediate goals are the reconstruction of a healthy dentition that fulfills all functional and aesthetic requirements, take into consideration the age, health and desires of the patient. After active and infectious diseases are eliminated and necessary treatments are made, the long-term goals are to prevent this and maintain health through, patient instruction on oral hygiene and loyalty to come recall visits and dentists professional support therapy. (1-3)



(Fig.7) (Temporary splints)

Treatment planning protocols start with emergency treatment. Extraction of hopeless teeth, temporary splints (Fig.7), etc. One week after the first visit, phase 1 (non-surgical phase) begins. This step includes plaque control and oral hygiene instruction of the patient. Phase 2 treatment includes surgical operations, implant placement. Phase 3 treatment (restorative phase) includes periodontal examination, restorative and / or prosthetic treatment. Phase 4 is the supportive periodontal therapy in other words the maintenance phase. This treatment involves assessment of the deepened sites with bleeding on probing, instrumentation of such sites, fluoride application to prevent dental caries, tooth mobility, occlusion control, etc. The aim of this treatment is to prevent reinfection and disease recurrence. (1-3)

#### **4. Conclusion**

Reliable diagnosis of periodontal disease is necessary for sensible treatment planning. Knowing the patient's necessities and standards is essential to providing appropriate care. Only when the patient's needs align with a realistic assessment of their condition and the expected outcomes of their therapy can optimal clinical outcomes be achieved. Therefore, in order to maintain consistency with the therapeutic condition, the patient's desires must be taken seriously and acknowledged in the evaluation. A patient's verbal history, systemic status, clinical and radiographic examination, and any necessary laboratory testing can all be used to make an accurate diagnosis. Documentation of the information acquired from the patient's anamnesis is necessary.

#### **References**

1. Carranza FA, Newman MG. Clinical Periodontology. Philadelphia: W.B. Saunders; 2019.
2. Lindhe J. Clinical Periodontology and Implant Dentistry. John Wiley & Sons, Ltd; 2015.
3. Armitage GC. Periodontal Diseases: Diagnosis. Annals of Periodontology. 1996;1(1):37-215.
4. Kinane DF, Peterson M, Stathopoulou PG. Environmental and other modifying factors of the periodontal diseases. Periodontology 2000. 2006;40(1):107-119.
5. XHONGA FA. Bruxism and its effect on the teeth. Journal of Oral Rehabilitation. 1977;4(1):65-76.

6. Ratcliff PA, Johnson PW. The Relationship Between Oral Malodor, Gingivitis, and Periodontitis. A Review. *Journal of Periodontology*. 1999;70(5):485-489.

7. Kaldahl WB, Kalkwarf KL, Patil KD, Molvar MP. Relationship of Gingival Bleeding, Gingival Suppuration, and Supragingival Plaque to Attachment Loss. *Journal of Periodontology*. 1990;61(6):347-351.

8. Wu CP, Tu YK, Lu SL, Chang JH, Lu HK. Quantitative analysis of Miller mobility index for the diagnosis of moderate to severe periodontitis - A cross-sectional study. *Journal of Dental Sciences*. 2018;13(1):43-47.

9. Armitage GC. Periodontal diagnoses and classification of periodontal diseases. *Periodontology 2000*. 2004;34(1):9-21.

10. Armitage GC, Svanberg GK, Loe H. Microscopic evaluation of clinical measurements of connective tissue attachment levels. *Journal of Clinical Periodontology*. 1977;4(3):173-190.

11. Badersten A, Nilveus R, Egelberg J. Effect of nonsurgical periodontal therapy. VII. Bleeding, suppuration and probing depth in sites with probing attachment loss. *Journal of Clinical Periodontology*. 1985;12(6):432-440.

12. Badersten A, Nilveus R, Egelberg J. Scores of plaque, bleeding, suppuration and probing depth to predict probing attachment loss 5 years of observation following nonsurgical periodontal therapy. *Journal of Clinical Periodontology*. 1990;17(2):102-107.

## CHAPTER V

# ORAL HEALTH STATUS IN CHILDREN WITH TYPE 1 DIABETES MELLITUS

**Muhammet Ali ORUÇ<sup>1</sup> & Semih Ercan AKGÜN<sup>2</sup>**

*<sup>1</sup>(Asst. Prof. Dr; M.D.) Samsun University, Faculty of Medicine,  
Department of Family Medicine, E-mail: muhammetalioruc@gmail.com  
ORCID: 0000-0002-4320-8579*

*<sup>2</sup> (DDS, Pediatric Dentistry Specialist) Ministry of Health,  
Bafra Oral and Dental Health Hospital,  
E-mail: semihercanakgun@gmail.com  
ORCID: 0000-0001-7266-8593*

### 1. Introduction

**T**ype 1 Diabetes Mellitus (T1DM) is a chronic disease characterized by insulin deficiency and the destruction of pancreatic  $\beta$ -cells, affecting more than half a million children worldwide. (1)

Many epidemiological studies have reported that T1DM increases the risk of cardiovascular diseases, kidney diseases, and cognitive regression in children and adolescents. Many systemic complications, such as micro- and macroangiopathies, retinopathy, neuropathy, and nephropathy, have been recognized as common complications of diabetes. (2-5)

Diabetes can also be seen with some syndromic conditions. It may also occur as a component of diseases such as Friedreich ataxia, Prader-Willi syndrome, Bardet-Biedl syndrome, Klinefelter syndrome, Turner syndrome, and Down syndrome. (6)

Among the warning symptoms of T1DM, polydipsia, polyuria, polyphagia, decreased level of consciousness, weight loss, difficulty in concentrating, dehydration, blurred vision, abdominal pain, weight loss, hypotension, hyperventilation, as laboratory findings glycosuria, ketonuria, and hyperglycemia are frequently observed. (7, 8)

Studies have shown that T1DM plays a significant role in the development of oral diseases, such as periodontitis and dental caries, and changes in the oral microflora. (9, 10)

Individuals with diabetes show oral complications more frequently than healthy individuals. Periodontal disease is the one of the most common complication of diabetes supports these data. (11, 12)

In children with T1DM, although dietary sucrose consumption is limited, ignorance of oral care habits can lead to oral health-related morbidities and poor glyceemic control. (8)

T1DM has a significant effect on oral health, intraoral treatment, and outcomes. Therefore, pediatric oral health professionals and medical doctors should be aware of the oral effects of T1DM. (8, 13, 14)

## **2. Intraoral Manifestations of Type 1 Diabetes**

### **2.1. Caries Risk**

Dental caries are the most common disease, especially in children, and have become an international health problem. (15) It has become an important problem that can affect the quality of life of the child and his family, since it can start early periods of life, progress rapidly in children at high risk, and often delay treatment. (16) However, the precise incidence of dental caries in T1DM patients, particularly in those with poor metabolic control, remains a matter of debate. (17-21) Additionally, diabetes causes changes in saliva content and flow rate, which may affect oral microflora. (22-26)

The number of Lactobacilli and *Streptococcus mutans* (*S. mutans*) is higher in patients with active caries, regardless of diabetes status. Cariogenic bacteria, especially *S. mutans* and *Lactobacillus casei* (*L.casei*), were observed in higher numbers in those with poor diabetes control than in healthy children. (11, 27)

Recent studies have reported no difference in caries incidence between children with controlled T1DM and their healthy peers. (28-30) The reason for this is the restriction of carbohydrate intake in patients with diabetes. Advances in insulin treatment regimens have now led to disease control, thus allowing children with T1DM to have a diet similar to that of healthy children. (31)

The multifactorial origin of dental caries makes it difficult to specify the precise factors responsible for the relationship between T1DM and dental caries. Altered salivary content and amount in T1DM patients causes slow oral clearance of glucose in the mouth, resulting in reductions in the pH level of dental plaque, thus increasing the risk of caries. (32)



Children with T1DM are at a higher risk of dental caries than healthy children, mainly due to the hypofunction of the salivary gland, high glucose concentrations in their saliva, and delayed metabolic control. (33) In a study comparing diabetic and non-diabetic children, a positive correlation was reported between caries and salivary glucose levels. (34) It has also been emphasized that the incidence of caries is higher in the permanent teeth of children whose diabetes is not well-controlled. (35, 36)

## ***2.2. Periodontal Disease Risk***

The immune response of diabetes patients to pathogenic microorganisms responsible for periodontal disease is exacerbated. Furthermore, during periodontal infection, pro-inflammatory cytokines may enter the bloodstream, leading to increased insulin resistance and ultimately inadequate glycemic control. (37, 38)

There is a double-sided relationship between diabetes and periodontal disease, and periodontitis causes difficulties in glycemic control in patients with T1DM. (39) Many studies have reported that periodontal therapy has a favorable effect on the metabolic control of diabetes. On the other hand, high glucose content in oral fluids of patients with T1DM contributes to bacterial growth, increases dental plaque formation, and leads to periodontal disease. (11, 12, 40, 41)

There is a relationship between diabetes mellitus (DM) and periodontal disease, especially in uncontrolled or hyperglycemic patients. Metabolic disorders in periodontal tissues can reduce the resistance of diabetics to infections, thus affecting the development of inflammatory periodontal disease. (42, 43)

Diabetes increases periodontal tissue destruction risk from the age of six, depending on the period of the disease. In a study including T1DM and non-diabetic control groups (6-18 years), it was shown that diabetic children had gingivitis, periodontal tissue destruction, and more dental plaque accumulation than the controls. Children had a 2.8 times higher risk of periodontal disease than the control group. (44) In another study, it was observed that diabetic children had more gingivitis, earlier dental eruption, and a higher prevalence of gingival bleeding in primary/permanent dentition than their non-diabetic counterparts. (45)

The risk of periodontitis is approximately three times higher in diabetic people, and periodontitis is known as the '6th chronic complication of diabetes.



Therefore, diabetes is considered a predisposing factor for periodontitis, as the severity of periodontal destruction correlates with glycemic level, as measured by HbA1c. (46)

Hyperglycemia increases glucose levels in saliva and gingival sulcus fluid, and glucose in the oral cavity increases oral inflammation by increasing the proliferation of periodontopathogenic and cariogenic bacteria. Studies have reported that compared with healthy or well-controlled diabetics, uncontrolled diabetics have higher proinflammatory mediator levels in the gingival sulcus fluid, resulting in significant periodontal destruction. (47)

Microangiopathy can be observed in hyperglycemic patients. Glucose is used by endothelial cells to a greater extent than normal and creates more glycoproteins. The basement membrane thickens and weakens, so that blood vessels leak proteins and bleed easily. These vascular changes reduce the elimination of chemotaxis, adhesion, phagocytosis, and migration of antigens by polymorphonuclear cells, leading to the progression of periodontitis. (48)

Hyperglycemia also increases glycation end products. These glycosylated products form complex molecules. It can reduce the solubility of collagen and increase the levels of proinflammatory cytokines responsible for the breakdown of bone and connective tissue. (48, 49)

Periodontal destruction in children with diabetes can begin very early in life and become more evident as children progress towards adolescence. Therefore, the early signs of periodontal disease should be considered. (30) In studies conducted, an improvement was observed in variables such as glycemic status, blood pressure, weight, and body mass index in patients with diabetes mellitus who regularly participated in interdisciplinary health support programs. (50, 51)

### ***2.3. Salivary Alterations***

Saliva plays an important role in maintaining dental and oral mucosal health, and changes in its volume and quality can alter oral health status. T1DM is known to cause microvascular diseases that can affect salivation. (52)

Decreased salivary flow has been reported in T1DM patients. Poor glycemic control may cause hyposalivation in children with diabetes. (53) This reduction in salivary volume causes a decrease in salivary clearance, buffering ability, and antimicrobial activity. This may lead to unfavorable effects on the healing of other oral infections. (54) Additionally, an increase in salivary glucose concentration may also affect the development of dental caries. (24)

Hyperglycemia increases glucose levels in saliva and gingival sulcus fluid, and glucose in the oral cavity increases oral inflammation by accelerating the proliferation of periodontopathogenic and cariogenic bacteria. (47)

#### ***2.4. Effect on Tissue Healing***

Delayed bone healing and poor soft tissue regeneration in patients with diabetes are complications that can be observed after oral surgery. This may be attributed to delayed vascularization and decreased blood flow, immunity, and growth factor production in patients with T1DM. (55)

#### ***2.5. Taste Disorder***

Taste changes are also associated with diabetes. (56) Children with T1DM have a lower salivary flow rate, pH, and buffering capacity than healthy children but have a higher glucose content. (21, 23, 57) Significantly higher glucose and urea levels in saliva were observed in 80% of patients with diabetes mellitus and 10% of healthy subjects. (58)

These findings suggest that DM is associated with xerostomia. There is a relationship between the degree of dry mouth and glucose levels in saliva.

#### ***2.6. Oral Candidiasis and Bacterial Infections***

Fungal infections are more common in patients with DM than in healthy individuals because of associated immunodeficiency. (59, 60) An increased amount of *Candida albicans* has been detected in patients with T1DM compared to healthy subjects. (59-61)

Decreased metabolic control, increased blood and saliva glucose concentration, low salivary secretion, prolonged disease duration, impaired chemotaxis and phagocytosis defects due to polymorphonuclear leukocyte deficiency, and decreased tissue resistance to infection are associated with increased *Candida* carriage and clinical manifestations of candidiasis. (60, 62)

Diabetic patients have impaired defense mechanisms; therefore, they are considered immunocompromised, and recurrent bacterial infections are seen more frequently in individuals with poor metabolic control. Therefore, patients with DM are more prone to deep neck infections of bacterial origin than patients without diabetes. (63)

In a study comparing the severity of maxillofacial infections of odontogenic origin, the susceptibility of microorganisms to antibiotics, the type of microorganisms, and the duration of hospitalization in diabetic patients with

healthy individuals, it has been determined that DM patients stay longer in the hospital owing to severe infection and need more time to control their blood sugar. (64)

### 3. Conclusion

In order to prevent dental caries/periodontal diseases in children with T1DM, it is vital to provide metabolic control of diabetes, perform regular intraoral examinations, and apply the necessary treatments promptly. It is important that children with T1DM are followed up by a multidisciplinary team, including dentists, in coordination with medical doctors. It is recommended that educational oral health programs be implemented that target patients and family members.

### References

1. Novotna M, Podzimek S, Broukal Z, et al. Periodontal Diseases and Dental Caries in Children with Type 1 Diabetes Mellitus. *Mediators Inflamm.* 2015;2015,379626.
2. Firatli E, Yilmaz O, Onan U. The relationship between clinical attachment loss and the duration of insulin-dependent diabetes mellitus (IDDM) in children and adolescents. *J Clin Periodontol.* 1996;23(4):362-366.
3. Ahmadizar F, Fazeli Farsani S, Souverein PC, et al. Cardiovascular medication use and cardiovascular disease in children and adolescents with type 1 diabetes: a population-based cohort study. *Pediatr Diabetes.* 2016;17(6):433-440.
4. Helve J, Sund R, Arffman M, et al. Incidence of End-Stage Renal Disease in Patients With Type 1 Diabetes. *Diabetes Care.* 2018;41(3):434-439.
5. Tonoli C, Heyman E, Roelands B, et al. Type 1 diabetes-associated cognitive decline: a meta-analysis and update of the current literature. *J Diabetes.* 2014;6(6):499-513.
6. Schmidt F, Kapellen TM, Wiegand S, et al. Diabetes mellitus in children and adolescents with genetic syndromes. *Exp Clin Endocrinol Diabetes.* 2012;120(10):579-585.
7. Hong Y, Hassan N, Cheah YK, et al. Management of T1DM in children and adolescents in primary care. *Malays Fam Physician.* 2017;12(2):18-22.
8. Nirmala S, Saikrishna D. Dental care and treatment of children with diabetes mellitus-an overview. *J Pediatr Neonatal Care.* 2016;4(2),00134.

9. Karjalainen KM, Knuutila ML. The onset of diabetes and poor metabolic control increases gingival bleeding in children and adolescents with insulin-dependent diabetes mellitus. *J Clin Periodontol*. 1996;23(12):1060-1067.
10. Arheiam A, Omar S. Dental caries experience and periodontal treatment needs of 10- to 15-year old children with type 1 diabetes mellitus. *Int Dent J*. 2014;64(3):150-154.
11. Siudikiene J, Machiulskiene V, Nyvad B, et al. Dental caries and salivary status in children with type 1 diabetes mellitus, related to the metabolic control of the disease. *Eur J Oral Sci*. 2006;114(1):8-14.
12. Loe H. Periodontal disease: The sixth complication of diabetes mellitus. *Diabetes care*. 1993;16(1):329-334.
13. Babu SR, Eisenbarth GS. Juvenile diabetes. *Indian J Med Res*. 2012;136(2):179-181.
14. Alberti KG, Zimmet PZ. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. *Diabet Med*. 1998;15(7):539-553.
15. Colak H, Dülgergil CT, Dalli M, et al. Early childhood caries update: A review of causes, diagnoses, and treatments. *J Nat Sci Biol Med*. 2013;4(1):29-38.
16. Grindefjord M, Dahllöf G, Modéer T. Caries development in children from 2.5 to 3.5 years of age: a longitudinal study. *Caries Res*. 1995;29(6):449-454.
17. Coelho A, Paula A, Mota M, et al. Dental caries and bacterial load in saliva and dental biofilm of type 1 diabetics on continuous subcutaneous insulin infusion. *J Appl Oral Sci*. 2018;26,e20170500.
18. Rafatjou R, Razavi Z, Tayebi S, et al. Dental Health Status and Hygiene in Children and Adolescents with Type 1 Diabetes Mellitus. *J Res Health Sci*. 2016;16(3):122-126.
19. Akpata ES, Alomari Q, Mojiminiyi OA, et al. Caries experience among children with type 1 diabetes in Kuwait. *Pediatr Dent*. 2012;34(7):468-472.
20. Miralles L, Silvestre FJ, Hernández-Mijares A, et al. Dental caries in type 1 diabetics: influence of systemic factors of the disease upon the development of dental caries. *Med Oral Patol Oral Cir Bucal*. 2006;11(3):256-260.
21. Carneiro V, Fraiz F, Ferreira FM, et al. The influence of glycemic control on the oral health of children and adolescents with diabetes mellitus type 1. *Arch Endocrinol Metab*. 2015;59(6):535-540.

22. Ben-Aryeh H, Cohen M, Kanter Y, et al. Salivary composition in diabetic patients. *J Diabet Complications*. 1988;2(2):96-99.

23. Moore P, Guggenheimer J, Etzel K, et al. Type 1 diabetes mellitus, xerostomia, and salivary flow rates. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2001;92(3):281-291.

24. López ME, Colloca ME, Páez RG, et al. Salivary characteristics of diabetic children. *Braz Dent J*. 2003;14(1):26-31.

25. Mata AD, Marques D, Rocha S, et al. Effects of diabetes mellitus on salivary secretion and its composition in the human. *Mol Cell Biochem*. 2004;261(1-2):137-142.

26. Busato IM, Ignácio SA, Brancher JA, et al. Impact of clinical status and salivary conditions on xerostomia and oral health-related quality of life of adolescents with type 1 diabetes mellitus. *Community Dent Oral Epidemiol*. 2012;40(1):62-69.

27. Bolgöl BS, Celenk S, Ayna BE, et al. Evaluation of caries risk factors and effects of a fluoride-releasing adhesive material in children with insulin-dependent diabetes mellitus (IDDM): initial first-year results. *Acta Odontol Scand*. 2004;62(5):289-292.

28. Edblad E, Lundin SA, Sjödin B, et al. Caries and salivary status in young adults with type 1 diabetes. *Swed Dent J*. 2001;25(2):53-60.

29. Moore PA, Weyant RJ, Etzel KR, et al. Type 1 diabetes mellitus and oral health: assessment of coronal and root caries. *Comm Dent Oral Epidemiol*. 2001;29(3):183-194.

30. Lalla E, Cheng B, Lal S, et al. Periodontal changes in children and adolescents with diabetes: a case-control study. *Diabetes Care*. 2006;29(2):295-299.

31. Ciglar L, Skaljic G, Sutalo J, et al. Influence of diet on dental caries in diabetics. *Coll Antropol*. 2002;26(1):311-317.

32. Hase JC, Birkhed D. Salivary glucose clearance, dry mouth and pH changes in dental plaque in man. *Arch Oral Biol*. 1988;33(12):875-880.

33. Sharma M, Tiwari S, Singh K, et al. Occurrence of bacterial flora in oral infections of diabetic and non-diabetic patients. *J Life Sci Med Res*. 2011;32:1-6.

34. Siudikiene J, Machiulskiene V, Nyvad B, et al. Dental caries increments and related factors in children with type 1 diabetes mellitus. *J Caries Res*. 2008;42(5):354-362.

35. Lal S, Cheng B, Kaplan S, et al. Gingival bleeding in 6- to 13-year-old children with diabetes mellitus. *Pediatr Dent*. 2007;29(5):426-430.

36. Orbak R, Simsek S, Orbak Z, et al. The influence of type-1 diabetes mellitus on dentition and oral health in children and adolescents. *Yonsei Med J*. 2008;49(3):357-365.
37. Orlando VA, Johnson LR, Wilson AR, et al. Oral Health Knowledge and Behaviors among Adolescents with Type 1 Diabetes. *Int J Dent*. 2010;2010,942124.
38. Khader YS, Dauod AS, El-Qaderi SS, et al. Periodontal status of diabetics compared with nondiabetics: a meta-analysis. *J Diabetes Complications*. 2006;20(1):59-68.
39. Mirza B, Syed A, Izhar F, et al. Bidirectional relationship between diabetes and periodontal disease: review of evidence. *J Pak Med Assoc*. 2010;60(9):766-768.
40. Moore PA, Orchard T, Guggenheimer J, et al. Diabetes and oral health promotion: a survey of disease prevention behaviors. *J Am Dent Assoc*. 2000;131(9):1333-1341.
41. Kinane DF, Peterson M, Stathopoulou PG. Environmental and other modifying factors of the periodontal diseases. *Periodontol 2000*. 2006;40:107-119.
42. Ainamo J, Lahtinen A, Uitto VJ. Rapid periodontal destruction in adult humans with poorly controlled diabetes. A report of 2 cases. *J Clin Periodontol*. 1990;17(1):22-28.
43. Chittenden SJ, Shami SK. Microangiopathy in diabetes mellitus: I. Causes, prevention and treatment. *Diabetes Res*. 1991;17(3):105-114.
44. Dakovic D, Pavlovic MD. Periodontal disease in children and adolescents with type 1 diabetes in Serbia. *J Periodontol*. 2008;79(6):987-992.
45. Lalla E, Cheng B, Lal S, Kaplan S, et al. Diabetes mellitus promotes periodontal destruction in children. *J Clin Periodontol*. 2007;34(4):294-298.
46. Preshaw PM, Alba AL, Herrera D, et al. Periodontitis and diabetes: a two-way relationship. *Diabetologia*. 2012;55(1):21-31.
47. Ryan ME, Usman A, Ramamurthy NS, et al. Excessive matrix metalloproteinase activity in diabetes: inhibition by tetracycline analogues with zinc reactivity. *Curr Med Chem*. 2001;8(3):305-316.
48. Grover HS, Luthra S. Molecular mechanisms involved in the bidirectional relationship between diabetes mellitus and periodontal disease. *J Indian Soc Periodontol*. 2013;17(3):292-301.
49. Ryan ME, Ramamurthy NS, Sorsa T, et al. MMP-mediated events in diabetes. *Ann N Y Acad Sci*. 1999;878:311-34.

50. Franz MJ, Bantle JP, Beebe CA, et al. Nutrition principles and recommendations in diabetes. *Diabetes Care*. 2004;27 Suppl 1:36-46.

51. Koerber A, Peters KE, Kaste LM, et al. The views of dentists, nurses and nutritionists on the association between diabetes and periodontal disease: a qualitative study in a Latino community. *J Public Health Dent*. 2006;66(3):212-215.

52. Meurman JH, Collin HL, Niskanen L, et al. Saliva in non-insulin-dependent diabetic patients and control subjects: The role of the autonomic nervous system. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 1998;86(1):69-76.

53. Harrison R, Bowen WH. Flow rate and organic constituents of whole saliva in insulin-dependent diabetic children and adolescents. *Pediatric Dent*. 1987;9(4):287-291.

54. Pruitt KM. The salivary peroxidase system: thermodynamic, kinetic and antibacterial properties. *J Oral Pathol*. 1987;16(8):417-420.

55. Abiko Y, Selimovic D. The mechanism of protracted wound healing on oral mucosa in diabetes. Review. *Bosn J Basic Med Sci*. 2010;10(3):186-191.

56. Leite RS, Marlow NM, Fernandes JK, et al. Oral health and type 2 diabetes. *Am J Med Sci*. 2013;345(4):271-273.

57. Kao CH, Tsai SC, Sun SS. Scintigraphic evidence of poor salivary function in type 2 diabetes. *Diabetes Care*. 2001;24(5):952-953.

58. Chávez EM, Borrell LN, Taylor GW, et al. A longitudinal analysis of salivary flow in control subjects and older adults with type 2 diabetes. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2001;91(2):166-173.

59. Vazquez JA, Sobel JD. Fungal infections in diabetes. *Infect Dis Clin North Am*. 1995;9(1):97-116.

60. Guggenheimer J, Moore PA, Rossie K, et al. Insulin-dependent diabetes mellitus and oral soft tissue pathologies: II. Prevalence and characteristics of *Candida* and *Candidal* lesions. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2000;89(5):570-576.

61. Aly FZ, Blackwell CC, Mackenzie DA, et al. Factors influencing oral carriage of yeasts among individuals with diabetes mellitus. *Epidemiol Infect*. 1992;109(3):507-518.

62. Shenoy MP, Puranik RS, Vanaki SS, et al. A comparative study of oral candidal species carriage in patients with type1 and type2 diabetes mellitus. *J Oral Maxillofac Pathol*. 2014;18(Suppl 1):60-65.

63. Huang TT, Tseng FY, Liu TC, et al. Deep neck infection in diabetic patients: comparison of clinical picture and outcomes with nondiabetic patients. *Otolaryngol Head Neck Surg.* 2005;132(6):943-947.

64. Rao DD, Desai A, Kulkarni RD, et als. Comparison of maxillofacial space infection in diabetic and nondiabetic patients. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2010;110(4):7-12.





## CHAPTER VI

# AUTOGENOUS TOOTH TRANSPLANTATION

**Zeynep GÜMÜŞER**

*E-mail: gumuser.zeynep@gmail.com*

*ORCID: 0000-0002-7834-4343*

### 1. Introduction

A fixed dental restoration to compensate for losing a missing molar tooth in adolescents is not indicated because bone growth is ongoing. Still, it is necessary to replace missing teeth to prevent resorption of the alveolar bone and migration/elongation of adjacent teeth. (1) Although dental implant treatment is considered the best option to compensate for missing tooth/teeth, it may not apply to every patient. It is expensive, time-consuming, and contraindicated for patients with continuous bone growth. (2-4)

Autogenous tooth transplantation (ATT) is the preferred biological replacement for posterior missing teeth in adolescents when an immature donor tooth is available. (1, 5-8) The essence of ATT is transferring a tooth from one socket to another within the same person. (9)

Autogenous tooth transplantation offers several advantages over dental fixtures and prostheses, as it is the only biological option. The procedure is reliable and predictable, especially for adolescents. After undergoing ATT, regeneration of bone and periodontal tissues is expected; both result in significant function and aesthetics. (10) The donor tooth's periodontal ligament prevents bone resorption at the alveolar crest, distributes the continuation of growth and provides harmony with teeth in the opposing arch. Orthodontics can also be applied after ATT. (11) In addition, donor tooth's periodontal ligament and bone contain proprioceptive receptors, which are expected to maintain functionality, while this prevents disproportionate forces that may occur during biting and chewing. Periodontal tissue fibres suspend forces between bone and root and

ensure homogeneous distribution. This may be another significant difference between a biological tooth replacement and other options, such as dental fixtures and prostheses.

## **2. Indications and Contraindications**

If a suitable donor tooth is available, it can be used in certain situations:

- Congenital missing teeth, impacted teeth, or ectopic eruption of teeth,
- In cases where teeth have deep caries and excessively damaged crowns,
- Persistent chronic periapical periodontitis or lesions,
- Missing teeth due to iatrogenic causes. (4, 12-16)

For a successful tooth transplantation, the donor tooth should have a simple shape to extract without any damage to periodontal tissues, and the recipient site should be wide enough to embed the donor tooth. For immature tooth roots, the optimal Morrees' stage is 4-5. Although stage 6 can also be suitable, it's important to ensure that the apical foramen is open. If the apical foramen/foramina of the donor tooth is/are less than 1mm wide, the patient should be under 30 for a better chance of success. (16)

Before performing the ATT procedure, it is essential to consider the patient's systemic condition and any obstacles to regenerative healing. Additionally, the recipient site and interocclusal space should be evaluated, and the process should be practical and achievable. If there is insufficient interocclusal space or buccolingual or mesiodistal narrowness, ATT is contraindicated.

## **3. Prognostic Factors**

Prognostic factors refer to all the factors that can influence the outcome of a treatment. (12, 17) According to the literature review by Almpani et al. (12), the success of tooth transplantation is affected by factors such as patient age and gender, root development stage of the donor tooth, the sufficiency of bone volume in the recipient area, root anatomy, preservation during the exposure time, fixation of the donor tooth to the recipient site, and the surgical technique used. Also, operation time and postoperative care affect the prognosis. (12) In addition, factors such as the surgeon's experience, patient's oral hygiene level, presence of acute or chronic infection, presence of occlusal contacts during the healing period, timing, and quality of endodontic treatment of the donor tooth were reported as prognostic factors.

A retrospective study conducted by Aoyama et al. (18) revealed that the success of ATT is significantly affected by the subject's age (over 40), the depth of probing (over 4mm), and the presence of caries, restorations, or root canal treatment. Other factors such as multiple or complex/divergent roots, the use of a maxillary tooth as the donor tooth, the donor tooth preferred from the opposing arch, and the absence of teeth in the recipient area for 2.5 months or longer also impact success rates.

#### **4. Preoperative Planning**

In an autotransplantation procedure without access to 3D imaging, the donor tooth extraction should be prioritized to prepare the recipient socket. This is because the proper size of the recipient socket can only be determined after examining the root morphology of the donor tooth. (4) In this case, the success of the ATT procedure depends entirely on the surgeon's experience. The exposure time of the donor tooth after extraction and the fitting attempts in the recipient socket directly affect the result. (9)

Thanks to 3D imaging and Computer-Aided Rapid Prototyping (CARP), personalized templates can be created as a copy of the donor tooth. This allows the recipient site to be prepared without using the donor tooth during fitting attempts and to reduce the exposure time. (9, 19-21)

#### **5. Procedure**

In most cases of ATT, the donor tooth is placed into the empty recipient socket immediately after extraction. However, this order may change depending on current conditions to provide advantages in the treatment. ATT can be single-phased and immediate, as well as single-phased early, single-phased delayed, or two-phased. (22)

In conventional ATT, the extracted tooth is used as a template to create the neo-alveolus, the newly performed recipient site alveolus, for a perfect fit. In the conventional method, the socket is either prepared based on radiographic images, and the donor tooth is extracted after this preparation, or the socket will be prepared after the donor tooth is extracted and the root morphology is examined. If the extracted tooth from the recipient site is multi-rooted, the interradicular septum is removed. In the presence of a lesion due to chronic inflammation, the apical part of the extraction socket should be curetted until it is ensured that the lesion is completely eradicated. Meanwhile, the integrity of the facial and lingual bone lamellae must be preserved. It is recommended

to leave the periodontal tissues in the recipient socket walls untouched due to their potential of progenitor cells. (16) Removing a minimum amount of tissue is important, making the procedure minimally invasive. Flap design should be planned accordingly. Unless necessary for facial bone protection, avoid creating a full-thickness flap in the recipient area. (19) If the flap is not elevated, even if a fracture occurs in the facial bone during extraction, the broken piece will be able to be fed as it remains attached to the periosteum. If the recipient site is adequately prepared, the donor tooth can be extracted atraumatically. While the donor tooth is luxated with the elevator, care should be taken not to damage the cementum, periodontal ligament, and most importantly, the Hertwig's Epithelial Root Sheath (HERS), which is the most critical factor for root development and revascularization. (2) After extraction, fitness is checked by manipulating the donor tooth into the recipient socket without touching the root surface. The donor tooth must be embedded in an infraocclusal position in the recipient area. Therefore, precise positioning may be possible after several fitting attempts. If this is the case, the donor tooth should not be moved to the extraoral area, if possible, between fitting attempts. The donor tooth can be placed in its socket in the room without pressure and kept there. (4) When a donor tooth cannot be saved in its original alveolus, storing it in gauze soaked with 0.9% saline solution, physiological saline solution, or fetal calf serum between the fitting attempts is preferable.

As per Tsukiboshi's research (4, 16), the typical process in ATT involves the following steps:

1. Antibiotics are started preoperatively (a few hours before the operation) on the patient.

2. Disinfection of the surgical area is provided; local anaesthesia is applied to the donor and recipient areas.

3. The tooth on the receiving side is extracted.

4. The donor tooth is extracted. Before the socket is prepared, the donor tooth's anatomical root shape and periodontal status should be evaluated. Care must be taken not to damage the periodontal tissues. To separate as much PDL as possible with the root, an intrasulcular incision should be made before starting the luxation, luxation should be made slowly and carefully, and the tooth should be atraumatically removed.

5. The mesiodistal width of the crown and root(s) of the donor tooth and the lengths of the roots are measured. After the donor tooth is examined, it will be

placed back in its original socket. If it must be kept extraoral, isotonic solutions will be appropriate. Pure, sterile water is not recommended as it is hypotonic and may damage periodontal cells.

6. Preparation of the recipient area is performed. The socket is prepared slightly wider than the root of the donor tooth. A rotary instrument with a system cooled with saline solution at low speed is used during the preparation.

7. Harmonization of the recipient socket and donor tooth root is performed. The donor tooth is placed in the recipient socket, avoiding pressure, and fitness is checked. At this time, too much infraocclusal alignment of the donor tooth should be avoided.

8. The flap is adapted and sutured. The most critical surgical procedure is the tight fixation of the gingival flap around the donor tooth. This prevents bacterial invasion, stabilizes the clot between the tooth and bone wall, and promotes optimal attachment formation. The flap must be adapted and sutured before the donor tooth is placed to ensure a tighter and more compliant closure. This technique is especially important for the second molar region.

9. After suturing the flap, the socket mouth is desired to be slightly narrower than the diameter of the donor tooth. The donor tooth is pushed through this opening slowly, without disproportionate pressure, and inserted into the socket. The placed donor tooth is first splinted with an occlusal suture. If the tooth is unstable despite the suture and must be aligned occlusally, splinting is performed with wire and composite. If the tooth is not fixed, but the occlusal adjustment is not required, splinting can be delayed for 2-3 days because it is difficult to fix the wire with composite despite postoperative bleeding since the area cannot be kept dry.

10. It is necessary to make sure that the tooth is not in occlusion. If sutures are to be used for stabilization, ideally, the occlusal contacts should be mopped extraorally before the tooth is positioned in the socket, avoiding damage to the PDL. If the donor is to be splinted with braces, it is possible to adapt in the mouth after the splint is placed. It should be as conservative as possible, and surfaces should not be mopped so much that they must be restored to provide post-healing function.

11. Postoperative radiographs should be taken. The position of the donor tooth in the recipient socket should be evaluated before and after splinting. The preservation of viable cells on the cementum surface and good adaptation of tissues are the most critical aspects for the success of dental autotransplantation.

(4, 9)

Therefore, in the conventional method, the number of attempts to control the compatibility of the donor tooth, the distance between the bone walls and the root of the donor tooth, the extra-alveolar time, the experience of the surgeon, and the level of trauma during the extraction of the donor tooth affect the prognosis of the autotransplant. (23, 24) Apart from the mechanical injury that may occur on the root surface and HERS during the manipulation of the donor tooth into the socket for optimum compatibility, the nutrition of the cells in the periodontal tissues will be disrupted during the time outside the socket; their biochemistry will be adversely affected, which will result in the damage and death of living cells. (4, 9) The greater the number of manipulations of the donor tooth into the socket and the longer the extra-alveolar time, the greater the risk of bacterial contamination. (23, 25) Unlike the technique in surgery performed by using the replica produced with CARP as a template, all fitting trials are performed with a donor tooth replica. If the position of the donor tooth and the socket-donor compatibility are assured, the extraction of the donor tooth is performed as the last step, and the donor tooth is placed in the recipient socket within seconds. This ensures that the above-mentioned negativities are eliminated or minimized. In late/non-socket cases where the recipient area has been edentulous for more than 2.5 months, preparing the artificial socket is difficult, which negatively impacts success. The reason for this is the extra time lost due to the difficulty in preparing the socket and the mechanical damage during the fitness checks. (18) Especially in such cases, a replica of the donor tooth produced by CARP is required for a predictable result. (9, 21)

### ***5.1. Splinting and Postoperative Process***

In the early 2-week postoperative stage, the transplant is stabilized at the infraocclusal level, protecting it from excessive forces and allowing a slight mobility. The goal is to allow the formation of a functional periodontal space. (14, 26) In the study of Bauss et al. in 2002 (27), it was mentioned that occlusal suture fixation is advantageous over a semi-rigid wire fixation. It was found that cases fixed with splint had a significantly higher incidence of pulp necrosis or ankylosis. If the donor tooth is stable enough at the infraocclusal level and the interocclusal distance is enough, a tight 8 suture can be seated between the cusps and will be sufficient to prevent loss of donor tooth. Instead of suturing the occlusal suture as a single suture, it is also possible to suture as two separate sutures intersecting occlusally, and it has been mentioned in the literature that this is more advantageous. (4, 15, 16) If the donor tooth is expected to move

due to tongue movements or chewing forces, a passive semi-rigid fixation is recommended. To avoid any kind of forces on the transplant, it's better to include only one adjacent tooth when using a stainless-steel wire for fixation. (16) In cases where splinting cannot be performed due to postoperative bleeding, it is possible to perform the semi-rigid splinting 2-3 days postoperative. (4) If there is no vertical mobility in the tooth, splinting should be limited to 7-10 days. In cases where vertical mobility is observed, the splint should be removed after the vertical mobility disappears. (28, 29)

Although there are conflicts in the literature on the use of antibiotics after ATT, in general, the use of postoperative antibiotics is for preventive purposes and the patient does not leave the clinic without prescribing antibiotics. Chung et al. (30) recommend prescribing systemic antibiotics to prevent complications and improve success rates of ATT. Again, after ATT, anti-inflammatory non-opioid analgesics will also be prescribed. Apart from this, postoperative oral hygiene and diet directives given to the patient are important. Until the healing of the periodontal ligament is completed, the patient, who will avoid solid foods for 2-8 weeks and will be directed to relatively soft foods, will inevitably consume carbohydrate-rich and fibre-free foods, which will lead to more plaque accumulation. The patient should be warned about this and encouraged to drink water and brush after each food intake. According to the American Society of Endodontists, if the apex of the donor tooth is closed, root canal treatment should start 7 to 14 days after ATT. If the donor tooth is splinted, the splint must be removed after the first session. Endodontic treatment should be completed by the 8th-22nd week after surgery. (4, 11, 31, 32)

## **6. Follow-up**

After the first week following ATT, the sutures will be removed. If there is no serious mobility that threatens healing, there is no harm to removing the occlusal sutures even if the tooth is not splinted. (15) If the tooth is splinted and there is no vertical mobility, it is preferred to remove the splint at the end of the second week. Follow-ups are ideally performed at the end of each month in the first 3 months, and every 6 months from the 6th postoperative month. (15) Monthly clinical and radiological controls in the first three months are very important for the early detection of inflammatory root resorption. Early detection of inflammatory resorption can be effectively controlled by applying root canal treatment to the donor tooth. In clinical evaluations, mobility, sensitivity in percussion and palpation, formation of papillae, and healing are evaluated



according to the period. In addition to these, the gingival and periodontological condition of the tooth is evaluated in clinical controls. Sensitivity is checked as an indirect sign of vitality. CO<sub>2</sub>, Electric Pulp Test (EPT), and Laser Doppler Flowmetry (LDF) are reliable and accurate tests. Although CO<sub>2</sub> and EPT are less reproducible, they are less time-consuming than LDF. (33) Subjective tests can be applicable at the end of the 3rd month at the earliest. (34) It is possible to get a positive response from the sensitivity test from the revitalized donor tooth, usually around the 6th month postoperatively. A clinical sign of success is the eruption of the transplanted tooth. (15) Orthodontic extrusion may be considered when the tooth does not erupt despite a healthy periodontal ligament. In radiological evaluations, bone healing, continuity of periodontal ligament and lamina dura, and obliteration of the pulp and root canals, which can be observed around the 6th month, are checked. Obliteration is the radiological sign of revascularization. Whether the roots continue to develop or not can be observed radiologically at the 6th month controls. (2) Complications due to iatrogenic injury, necrosis, and contamination in pulp, periodontal tissues, and HERS are seen in the early and late follow-up stages. Infection and healing disorders are the first complications that may be seen in the early period, which are not specific to ATT. The most crucial complication due to pulp/periodontal ligament injury/contamination is inflammatory root resorption. If inflammatory resorption can be diagnosed at an early stage, long-term survival of the donor tooth can be achieved with disinfection of the root canal system. The radiological signs of resorption can be observed at the end of the 8<sup>th</sup> week. (15)

Ankylotic healing occurs in cases where the root surface is injured and repair with new cementum is not possible. Ankylotic healing is a form of non-physiological healing in which the root is resorbed, and the apposition of the bone is seen instead of the root tissue. Ankylosis can be radiologically detected from the fourth week, but it may also manifest up to a year after healing. (14, 15) Ankylosis has no known cure. Ankylotic resorption was observed to progress more rapidly in children and young adults than in adult patients. (4, 35) It is crucial to create a personalized plan for each patient considering that age has an impact on the prognosis.

## **7. Success and Survival**

Survival is the functional existence of the transplanted tooth in the recipient area without showing any symptoms. (36) Success is the recovery and function of the donor tooth without any pathology and the need for additional intervention.

(9) Success rates range between 79-100% and 57-100% in cases performed with conventional methods. (4, 37) Regularly evaluating the transplants in postoperative period is crucial for their short- and long-term success. The first signs of success in the early postoperative period are uneventful healing, absence of infection, and decreased mobility of transplant. Infection and occlusal trauma can cause early transplant loss by hindering healing of periodontal tissues and pulp. Considering the possibility of inflammatory resorption in the first three months of the transplant, monthly radiological follow-ups should be performed. (15) It is known that the cause of donor tooth loss in the first year is usually due to inflammatory resorption. (38) In the long term, donor tooth loss is often caused by ankylosis. (15) Currently, there is no agreement on the specific criteria that define success. However, most researchers define success as the donor tooth being vital and functional without any pathology after ATT. (39)

## 8. Conclusion

Today, autotransplantation is the only viable option for physiological tooth replacement. In the presence of a suitable donor tooth, dental autotransplantation can be the gold standard for the early treatment of single tooth deficiency in adolescents. Dentists should be conscious and informative, and the patient's tooth should always be prioritized over any prosthesis.

## References

1. Clark HB, Jr., Tam JC, Mitchell DF. Transplantation of developing teeth. *J Dent Res.* 1955;34(3):322-8.
2. Paulsen HU, Andreasen JO, Schwartz O. Pulp and periodontal healing, root development, and root resorption subsequent to transplantation and orthodontic rotation: a long-term study of autotransplanted premolars. *Am J Orthod Dentofacial Orthop.* 1995;108(6):630-40.
3. Mendes RA, Rocha G. Mandibular third molar autotransplantation--literature review with clinical cases. *J Can Dent Assoc.* 2004;70(11):761-6.
4. Tsukiboshi M. Autotransplantation of teeth: requirements for predictable success. *Dent Traumatol.* 2002;18(4):157-80.
5. Apfel H. Transplantation of the unerupted third molar tooth. *Oral Surg Oral Med Oral Pathol.* 1956;9(1):96-8.
6. Hale ML. Autogenous transplants. *J Am Dent Assoc.* 1954;49(2):193-8.
7. Miller HM. Transplantation and reimplantation of teeth. *Oral Surg Oral Med Oral Pathol.* 1956;9(1):84-95.

8. Slagsvold O, Bjercke B. Autotransplantation of premolars with partly formed roots. A radiographic study of root growth. *Am J Orthod.* 1974;66(4):355-66.

9. Verweij JP, Jongkees FA, Anssari Moin D, Wismeijer D, van Merkesteyn JPR. Autotransplantation of teeth using computer-aided rapid prototyping of a three-dimensional replica of the donor tooth: a systematic literature review. *Int J Oral Maxillofac Surg.* 2017;46(11):1466-74.

10. Reich PP. Autogenous transplantation of maxillary and mandibular molars. *J Oral Maxillofac Surg.* 2008;66(11):2314-7.

11. Kim S, Lee SJ, Shin Y, Kim E. Vertical Bone Growth after Autotransplantation of Mature Third Molars: 2 Case Reports with Long-term Follow-up. *J Endod.* 2015;41(8):1371-4.

12. Almpiani K, Papageorgiou SN, Papadopoulos MA. Autotransplantation of teeth in humans: a systematic review and meta-analysis. *Clin Oral Investig.* 2015;19(6):1157-79.

13. Andreasen JO. Periodontal healing after replantation and autotransplantation of incisors in monkeys. *Int J Oral Surg.* 1981;10(1):54-61.

14. Andreasen JO, Paulsen HU, Yu Z, Ahlquist R, Bayer T, Schwartz O. A long-term study of 370 autotransplanted premolars. Part I. Surgical procedures and standardized techniques for monitoring healing. *Eur J Orthod.* 1990;12(1):3-13.

15. Tsukiboshi M, Yamauchi N, Tsukiboshi Y. Long-term Outcomes of Autotransplantation of Teeth: A Case Series. *J Endod.* 2019;45(12S):S72-S83.

16. Tsukiboshi M, Tsukiboshi C, Levin L. A step-by step guide for autotransplantation of teeth. *Dent Traumatol.* 2023;39 Suppl 1:70-80.

17. Sugai T, Yoshizawa M, Kobayashi T, Ono K, Takagi R, Kitamura N, et al. Clinical study on prognostic factors for autotransplantation of teeth with complete root formation. *Int J Oral Maxillofac Surg.* 2010;39(12):1193-203.

18. Aoyama S, Yoshizawa M, Niimi K, Sugai T, Kitamura N, Saito C. Prognostic factors for autotransplantation of teeth with complete root formation. *Oral Surg Oral Med Oral Pathol Oral Radiol.* 2012;114(5 Suppl):S216-28.

19. Lee SJ, Jung IY, Lee CY, Choi SY, Kum KY. Clinical application of computer-aided rapid prototyping for tooth transplantation. *Dent Traumatol.* 2001;17(3):114-9.

20. Verweij JP, Anssari Moin D, Wismeijer D, van Merkesteyn JPR. Replacing Heavily Damaged Teeth by Third Molar Autotransplantation With the Use of Cone-Beam Computed Tomography and Rapid Prototyping. *J Oral Maxillofac Surg.* 2017;75(9):1809-16.

21. Verweij JP, van Westerveld KJH, Anssari Moin D, Mensink G, van Merkesteyn JPR. Autotransplantation With a 3-Dimensionally Printed Replica of the Donor Tooth Minimizes Extra-Alveolar Time and Intraoperative Fitting Attempts: A Multicenter Prospective Study of 100 Transplanted Teeth. *J Oral Maxillofac Surg.* 2020;78(1):35-43.
22. Tschammler C, Angermair J, Heiligensetzer M, Linsenmann R, Huth KC, Nolte D. Primary canine auto-transplantation: a new surgical technique. *Oral Surg Oral Med Oral Pathol Oral Radiol.* 2015;119(2):158-69.
23. Anssari Moin D, Derksen W, Verweij JP, van Merkesteyn R, Wismeijer D. A Novel Approach for Computer-Assisted Template-Guided Autotransplantation of Teeth With Custom 3D Designed/Printed Surgical Tooling. An Ex Vivo Proof of Concept. *J Oral Maxillofac Surg.* 2016;74(5):895-902.
24. Day PF, Lewis BR, Spencer RJ, Barber SK, Duggal M. The design and development of surgical templates for premolar transplants in adolescents. *Int Endod J.* 2012;45(11):1042-52.
25. Lee SJ, Kim E. Minimizing the extra-oral time in autogeneous tooth transplantation: use of computer-aided rapid prototyping (CARP) as a duplicate model tooth. *Restor Dent Endod.* 2012;37(3):136-41.
26. Mejare B, Wannfors K, Jansson L. A prospective study on transplantation of third molars with complete root formation. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2004;97(2):231-8.
27. Bauss O, Schilke R, Fenske C, Engelke W, Kiliaridis S. Autotransplantation of immature third molars: influence of different splinting methods and fixation periods. *Dent Traumatol.* 2002;18(6):322-8.
28. Pogrel MA. Evaluation of over 400 autogenous tooth transplants. *J Oral Maxillofac Surg.* 1987;45(3):205-11.
29. Sange S, Thilander B. Transalveolar transplantation of maxillary canines. A follow-up study. *Eur J Orthod.* 1990;12(2):140-7.
30. Chung WC, Tu YK, Lin YH, Lu HK. Outcomes of autotransplanted teeth with complete root formation: a systematic review and meta-analysis. *J Clin Periodontol.* 2014;41(4):412-23.
31. Akiyama Y, Fukuda H, Hashimoto K. A clinical and radiographic study of 25 autotransplanted third molars. *J Oral Rehabil.* 1998;25(8):640-4.
32. Salinas TJ, Eckert SE. In patients requiring single-tooth replacement, what are the outcomes of implant- as compared to tooth-supported restorations? *Int J Oral Maxillofac Implants.* 2007;22 Suppl:71-95.
33. Chen E, Abbott PV. Evaluation of accuracy, reliability, and repeatability of five dental pulp tests. *J Endod.* 2011;37(12):1619-23.

34. Andreasen JO. Challenges in clinical dental traumatology. *Endod Dent Traumatol.* 1985;1(2):45-55.

35. Czochrowska EM, Stenvik A, Bjercke B, Zachrisson BU. Outcome of tooth transplantation: survival and success rates 17-41 years posttreatment. *Am J Orthod Dentofacial Orthop.* 2002;121(2):110-9; quiz 93.

36. Aslan BI, Ucuncu N, Dogan A. Long-term follow-up of a patient with multiple congenitally missing teeth treated with autotransplantation and orthodontics. *Angle Orthod.* 2010;80(2):396-404.

37. Cross D, El-Angbawi A, McLaughlin P, Keightley A, Brocklebank L, Whitters J, et al. Developments in autotransplantation of teeth. *Surgeon.* 2013;11(1):49-55.

38. Abela S, Murtadha L, Bister D, Andiappan M, Kwok J. Survival probability of dental autotransplantation of 366 teeth over 34 years within a hospital setting in the United Kingdom. *Eur J Orthod.* 2019;41(5):551-6.

39. Lucas-Taule E, Bofarull-Ballus A, Llaquet M, Mercade M, Hernandez-Alfaro F, Gargallo-Albiol J. Does Root Development Status Affect the Outcome of Tooth Autotransplantation? A Systematic Review and Meta-Analysis. *Materials (Basel).* 2022;15(9).

## CHAPTER VII

# SURGICAL TREATMENT OPTIONS OF OBSTRUCTIVE SLEEP APNEA SYNDROME

**Ipek Necla GULDIKEN<sup>1</sup> & Yigit Can GULDIKEN<sup>2</sup>**

<sup>1</sup>(Assistant Professor), *Istinye University School of Dentistry, Istanbul, Turkey,*  
*Department of Oral and Maxillofacial Surgery*  
ORCID: 0000-0003-1266-7913

<sup>2</sup>(Specialist MD), *Bahcesehir University, Goztepe Medical Park Hospital,*  
*Department of Neurology, Istanbul, Türkiye.*  
ORCID: 0000-0002-6954-5215

### 1. Introduction

**S**urgical interventions are crucial in addressing obstructive sleep apnea (OSA) in different cases. Typically, surgery is employed when anatomical stenosis is the primary root of the problem. Conservative approaches may not be well-received or inadequate, hence surgical intervention is needed. The surgical approach varies depending on the location of the airway obstruction. Surgical procedures are typically performed in the nasopharynx, oropharynx (retrolingual), and hypopharynx (retropalatal). They may be combined to meet the patient's specific needs. Nevertheless, procedures aimed at reducing upper airway collapse rarely achieve complete relief of OSAS symptoms.

While such interventions are generally effective, they do have limitations in achieving complete eradication of all symptoms. Often, patients require additional postoperative treatment and follow-up. Surgical procedures are typically performed in conjunction with other treatment modalities as part of a comprehensive treatment plan. The goal of surgical interventions is to increase overall quality of life and improve sleep quality by reducing airway obstruction and widening the airway. This approach can decrease the risk of cardiovascular and metabolic complications associated with OSAS by enhancing sleep duration and oxygenation.

In addition, a multifaceted approach is critical in the planning of surgical treatment. Physicians must develop a tailored treatment plan that considers the patient's symptoms, anatomical characteristics, and comorbidities. This ensures favorable outcomes that account for the distinctive necessities and circumstances of each patient (1).

## **2. Surgical Treatments for OSAS**

### **Upper Respiratory Tract Surgery**

These methods aim to address the upper respiratory tract and are not a standalone cure for OSAS. Yet, in combination with other treatments, they can augment the efficacy of treatment for OSAS. The following procedures are frequently performed in patients with nasal passage stenosis (2):

- Septoplasty
- Endoscopic sinus surgery
- Inferior turbinectomy
- Rhinoplasty
- Nasal valve surgery

While nasal surgery alone does not consistently impact AHI in OSAS patients, evidence suggests it can enhance snoring, subjective sleep quality, daytime sleepiness, sleep-related quality of life measures, and other significant OSAS outcomes (2,3).

### **Uvulopalatopharyngoplasts (UPPP)**

It was initially employed by Ikematsu in 1952 to address snoring and is also referred to as 3-P. The tissues eliminated through this technique comprise the following:

- Palatine tonsils (adenoids/nasal passages)
- Posterior soft palate
- Uvula

Thus, during sleep, diminished muscle tone causes the soft tissues that vibrate during breathing to elongate, widening the airway passage. The most commonly utilized surgical procedure for treating OSAS is UPPP, which boasts the highest success rate after maxillo-mandibular advancement (MMA).

Despite UPPP's substantial success in reducing snoring alone, its efficacy in treating apnea drops to approximately 50%. The likelihood of success is higher when UPPP is combined with tongue root surgery or MMA. The primary complication of UPPP is velopharyngeal insufficiency caused by excessive resection. Including the musculus levator veli palatine in the resection elevates the risk of this complication. The sensation of a foreign object lodged in the throat is another potential complication. Furthermore, the high concentration of ceruminous glands in the uvula results in moisture within the oropharynx. This factor may cause speech and swallowing difficulties post-UPPP (3,4).

### **Radiofrequency**

Radiofrequency treatment of the soft palate aims to create a voluntary fibrosis of the soft palate, making it more tense and firm, and thus more resistant to gravity in the sleeping position. However, this method alone is only relatively effective for snoring and less effective for apnea.

### **Tonsillectomy**

Another surgical method is isolated tonsillectomy and tonsillotomy in patients with large tonsils. It is generally preferred in pediatric OSAS. The extent to which tonsil hypertrophy contributes to OSAS in adults remains unclear. Tonsillectomy combined with adenectomy is accepted as first-line treatment in pediatric patients with severe OSAS and adenotonsillar hypertrophy. It has been reported that this type of surgery shows significant improvement in AHI severity, oxyhemoglobin saturation and sleep quality in obese patients with OSAS (3,5).

### **Palate Implants**

This procedure, also known as the Pillar procedure, aims to reduce snoring and mild to moderate AHI by creating fibrosis in the surrounding soft tissues with a polyester implant placed in the soft palate. The resulting fibrosis hardens the soft palate and increases the distance between the palate and the posterior wall of the pharynx during sleep. This procedure is a relatively minimally invasive method that can be performed under local anesthesia (2,4).

### **Resective and Positional Tongue Surgery**

When the primary underlying cause of OSAS is the posterior positioning of the tongue which leads to retrolingual obstruction and narrowing of the oropharynx, surgical intervention is considered. Thus, one can opt for surgical



techniques that bring the posteriorly placed tongue root forward or techniques that reduce hypertrophy of the tongue. The objective of tongue reduction procedures is to enhance the lower pharyngeal airway by decreasing the volume of tongue tissues. Tongue tissue advancement surgeries aim to enlarge the same area utilizing an alternate mechanism (1-3).

Several surgical procedures can be used to reduce tongue volume, including midline glossectomy, hyoid suspension, genioglossal advancement, tongue root stabilization, partial glossectomy, and radiofrequency application to the tongue root. Some of these are described as follows (6):

- **Lingual Tonsillectomy:** Improves the respiratory tract by removing the blocked lingual tonsil tissue.
- **Mid-Line Glossectomy:** Resection of the midline tongue base tissue.
- **Submucosal Lingualplasty:** Resection of the submucosal lingual tissue of the tongue root.
- **Radiofrequency Tissue Ablation:** Radiofrequency application to the tongue root is another simple surgical approach based on the principle of opening the posterior airway by reducing the tongue root volume. The procedure is performed in several sessions around the circumvallate papillae at intervals of several weeks.

Tongue tissue advancement procedures aim to advance the base of the tongue and pharyngeal muscles and thus widen the lower pharyngeal airway (2,6):

- **Base of Tongue Suspension:** A common technique for tongue root surgery is the 'Sleep In' approach: with a non-resistant suture passed through the tongue root, the tongue is pulled forward and connected to the lingual part of the lower jaw. The aim is to create a base of tongue sling fixed to the mandible and to prevent retrolingual collapse.
- **Genioglossal Advancement:** The genial tubercle of the anterior mandible is advanced forward by creating an osteotomy line. In this technique, a small window is opened at the attachment point of the genioglossus to the mandible and this bony lamella is rotated 90 degrees and reattached to the mandible with the help of a mini-plate.
- **Hyoid Semic suspension:** The hyoid bone is suspended to the thyroid cartilage or mandible using permanent sutures. The aim is to stabilize the base of the tongue and hypopharynx.

Genioglossal advancement is a procedure that involves hyoid bone suspension, also referred to as hyoid sling, or can be performed in isolation. This procedure can expand the posterior airway by approximately 1-1.5 mm. Despite these benefits, there are some limitations to this method. Firstly, the area that can be gained is restricted. Secondly, the movement alone is not enough to eliminate apnea. Due to the mandible not being pulled forward, there is no additional space for the tongue (1,6).

Multiple studies have shown that lower pharyngeal and laryngeal procedures can improve respiratory physiology and quality of life during sleep. However, reductions in AHI of up to 50% have only been reported in mild cases where the problem is isolated to the base of the tongue. It is important to explain technical term abbreviations when first used to ensure clarity and comprehension.

The reported side effects vary depending on the surgical techniques employed. Pain, bleeding, tongue infection, airway complications, changes in taste, and dysphagia are commonly observed as complications of partial glossectomy, lingualplasty, and lingual tonsillectomy. These relatively simple surgical procedures do not result in significant restrictions on tongue movement; however, when performed in isolation, they are insufficient in the treatment of OSAS (1,7,8).

### **Sliding Genioplasty**

This genioplasty technique is an advancement genioplasty that is occasionally recommended for patients with micrognathia due to retrognathic mandible. Since there is a risk of loss of tooth vitality, it may be considered in patients with significant retrognathia, that is, patients positioned >2 cm behind the subnasal vertical line. This procedure rarely involves the entire genial tubercle, so it does not affect the extraction of the genioglossal muscle. It is likely to pull the geniohyoid muscle, which may lead to an unfavorable vector at the root of the tongue. Sliding genioplasty is mainly suitable for patients with obstruction in the area behind the epiglottis. It is performed through subperiosteal dissection with exposure of the lower border of the anterior mandible. The osteotomy is performed by incising both lateral margins of the parasymphysis along its inferior border and then extending this incision horizontally under the mental neurovascular bundles. It should be performed in patients with both functional and aesthetic indications. When the anterior osteotomy is combined with the geniotubercle through adjacent vertical window osteotomies, the operation is defined as Mortised Genioplasty (9).

## **Multistage Surgical Approach and Maxillo-Mandibular Advancement (MMA)**

Since moderate to severe OSAS is often characterized by stenosis in multiple parts of the airway passage, surgical interventions targeted at a single site are insufficient for effective treatment. In 1986, Riley et al. were the first to recognize the importance of opening multiple obstructions with multilevel surgical interventions in OSAS patients. Today, multilevel surgery is widely accepted as a treatment option for OSAS patients with obstructions in multiple areas. However, as this alternative is not feasible for all patients, Maxillo-Mandibular Advancement (MMA), a method that enables airway expansion at multiple levels, has gained popularity and widespread acceptance over time. This treatment, which previously entailed a significant morbidity rate and a lengthy recuperation period, now necessitates a shorter and simpler recovery period owing to enhancements in orthodontic and surgical fixation methods. Due to its numerous advantages and high success rate in treating OSAS, dental occlusion has become a more popular method of treatment compared to the past (1,10,11).

In cases with mild to moderate maxillary retrognathia, where the incisor-molar positions are in close proximity, minimal mandibular advancement can be achieved through the use of dental occlusion. In this scenario, advancing the mandible alone does not achieve enough airway dilatation necessary to treat OSAS. Bilateral orthognathic surgery (or MMA) is one of the leading surgical treatment options for OSAS, with a success rate of approximately 85%, which can vary inversely with the severity of OSAS (11-13).

In the MMA procedure, the maxilla and mandible are advanced simultaneously to enlarge the upper airway. To accomplish this goal, Le Fort I osteotomy is typically performed on the upper jaw, and bilateral sagittal split ramus osteotomy (BSSRO) is generally preferred for the lower jaw. Although patients typically undergo postoperative orthodontic treatment for dental closure before orthognathic surgery, in the context of maxillomandibular advancement surgery for obstructive sleep apnea syndrome (OSAS), preserving the existing occlusion by bringing the upper and lower jaw segments forward without preoperative orthodontic treatment may be possible in most cases, providing a time-saving benefit (8,11).

The oropharynx, hypopharynx, and nasopharynx, to a lesser extent, are the anatomical regions aimed at expanding through maxillo-mandibular advancement, resulting in multilevel expansion.

The advantageous positive airway alteration produced by this procedure is effective during the early stages following MCI. After these surgeries, patients experience a more favorable improvement than that seen with UPFP. Additionally, significant relief is observed in the early postoperative period when compared to other isolated surgeries (1, 8, 13, 14).

According to Riley et al., the successful surgical protocol is as follows:

Phase 1: UPPP and/or Genioglossal Advancement and Hyoid Bone Suspension (Success rate is reported to be 75% on average in moderate OSAS patients, while the level of symptom relief and improvement in severe OSAS cases has been found to be below 50%.

Phase 2: Maxillo-Mandibular Advancement (MMA) (Success rate hovers around 95% even in severe cases) (15).

In general, surgical techniques used in severe OSAS cases are categorized as follows (14):

- Single-segment Le Fort I Osteotomy described by Bell
- Bilateral Sagittal Split Ramus Osteotomy (BSSRO) as modified by Epker
- Genioglossus and Hyoid Bone Suspension applied according to the Riley and Powell Method

The observed superiority of MMA over other surgical options and even the multilevel surgical method in the treatment of OSAS is that it can provide expansion of the entire retropalatal and retrolingual airway by expanding the skeletal frame. With MMA, it has been proven that the pharyngeal airway volume can be expanded up to 60% (1,3,12).

However, in some cases, the airway stenosis may be specifically maxillary when viewed from the sagittal plane, or it may be at the mandibular level, but at a lower level. If the condition causing this condition is a severe maxillary or mandibular retardation, the AHI scores of these patients can be significantly reduced with isolated maxillary or mandibular advancement (16).

The long-term success of orthognathic surgery is usually evaluated based on the relapse rate. Research in this area shows that MMA, especially with 'overcorrection' as much as aesthetic parameters allow, maintains clinical success, with approximately 90% of patients continuing to experience therapeutic benefit for more than 4 years. Studies on patient-oriented outcomes emphasize that more than 90% of patients have improved quality of life with gains in productivity, social outcomes, and physical activity level (25,30,33).

### 3. Current Approaches

Interestingly, palatal resection techniques such as UPPP are now considered obsolete in OSAS surgery and modern reconstructive techniques such as expansion sphincter pharyngoplasty are replacing conventional surgical approaches due to better clinical outcome and fewer side effects. In addition, upper airway stimulation and hypoglossal nerve (12th cranial nerve) stimulation, an emerging treatment option for moderate to severe OSAS, has been reported to have a success potential of up to 75% in patients with OSAS. Hypoglossal nerve stimulation is also a relatively new and popular technique. In this method, a stimulation electrode is surgically implanted in the branches of the nerve and a sensing electrode is surgically implanted in the chest wall. Both are then connected to a neurostimulator, which is placed in a surgically created pocket just below the collarbone. The sensing electrode determines when breathing starts and sends a signal to the neurostimulator. The stimulation electrode activates the hypoglossal nerve, which controls the movement of the tongue. The activated motor nerve causes the tongue to move forward. This method aims to prevent upper airway obstruction by preventing the tongue from collapsing backwards during apnea (1,8,12,13,15).

Examples of relatively less invasive procedures that can be considered current for OSAS include reducing the size of the tongue with radiofrequency, hardening the soft palate with stents, advancing only the anterior part of the lower jaw and displacing the hyoid bone. None of these procedures alone has been shown to achieve a high success rate in the treatment of OSAS. Isolated applications may be useful only in selected cases (7,15,17).

### 4. Conclusion

Obstructive Sleep Apnea Syndrome (OSAS) involves recurrent episodes of apnea and hypopnea caused by upper airway obstruction, typically due to anatomical abnormalities. Surgical treatments are vital in managing OSAS, particularly when conservative treatments prove ineffective or intolerable. Nonetheless, the effectiveness of surgical interventions in fully eliminating OSAS symptoms is often restricted. Anatomic stenosis and obstruction may occur in the nasopharynx, oropharynx, and hypopharynx. For each region, different surgical procedures can be implemented (1).

The choice of surgical treatment strategy depends on the location and severity of the anatomical obstruction. Examples of common surgical approaches are uvulopalatopharyngoplasty (UPPP), maxillomandibular advancement

(MMA), genioglossus advancement (GA), and hypoglossal nerve stimulation. Proper evaluation of the patient and selection of the appropriate surgical method are essential for a successful surgical intervention (7).

According to studies, UPPP can alleviate the symptoms of OSAS, but it usually fails to achieve a significant reduction in the apnea-hypopnea index (AHI) (4). On the other hand, MMA and GA are generally more successful, but these procedures are more invasive and carry significant morbidity risks (1). Hypoglossal nerve stimulation can significantly reduce AHI, but it is only appropriate for specific patients (15).

Surgical methods for OSAS are not definitive and have pros and cons. When planning surgical treatment, it is necessary to consider the severity of the patient's symptoms, anatomical characteristics, comorbidities, and treatment preferences (19). Long-term follow-up and evaluation of patients after surgical intervention is necessary to assess treatment efficacy and implement additional strategies if needed (20).

In conclusion, surgical treatment of OSAS can benefit specific patients, but careful evaluation and selection must precede implementation. Patients and healthcare professionals should comprehend the confines and plausible hazards linked with the offered surgical alternatives and suitably regulate their anticipations for every therapy method.

## References

1. Barrera JE. Skeletal Surgery for Obstructive Sleep Apnea. Vol. 13, Otolaryngologic Clinics of North America. W.B. Saunders; 2016. p. 1433–47.
2. Hanna J, Izzo A. Surgical Treatment Options for Obstructive Sleep Apnea. In: Updates in Sleep Neurology and Obstructive Sleep Apnea. IntechOpen; 2021.
3. Hendler BH, Costello BJ, Silverstein K, Yen D, Goldberg A. A protocol for uvulopalatopharyngoplasty, mortised genioplasty, and maxillomandibular advancement in patients with obstructive sleep apnea: An analysis of 40 cases. *Journal of Oral and Maxillofacial Surgery*. 2001;59(8):892–7.
4. Kent D, Stanley J, Aurora RN, Levine C, Gottlieb DJ, Spann MD, et al. Referral of adults with obstructive sleep apnea for surgical consultation: an American Academy of Sleep Medicine clinical practice guideline. Vol. 17, *Journal of Clinical Sleep Medicine*. American Academy of Sleep Medicine; 2021. p. 2499–505.

5. Salzano G, Maglitto F, Bisogno A, Vaira LA, De Riu G, Cavaliere M, et al. Obstructive sleep apnoea/hypopnoea syndrome: Relationship with obesity and management in obese patients. Vol. 41, *Acta Otorhinolaryngologica Italica*. Pacini Editore S.p.A./AU-CNS; 2021. p. 120–30.

6. Emanuelli E, O'Connor MK, Garg RK. Genioglossus Advancement: Technique Modification for Improved Chin Contour. *Plast Reconstr Surg Glob Open*. 2023 Mar 8;11(3):E4846.

7. Patel SR. Obstructive sleep apnea. *Ann Intern Med*. 2019 Dec 3;171(11):ITC81–96.

8. Lye KW, Waite PD, Meara D, Wang D. Quality of Life Evaluation of Maxillomandibular Advancement Surgery for Treatment of Obstructive Sleep Apnea. *Journal of Oral and Maxillofacial Surgery*. 2008 May;66(5):968–72.

9. Dos Santos JF, Abrahão M, Gregório LC, Zonato AI, Gumieiro EH. Genioplasty for genioglossus muscle advancement in patients with obstructive sleep apnea-hypopnea syndrome and mandibular retrognathia. *Braz J Otorhinolaryngol*. 2007;73(4):480–6.

10. Barrera JE. Skeletal Surgery for Obstructive Sleep Apnea. Vol. 49, *Otolaryngologic Clinics of North America*. W.B. Saunders; 2016. p. 1433–47.

11. Coleta KED, Wolford LM, Gonçalves JR, dos Santos Pinto A, Cassano DS, Gonçalves DAG. Maxillo-mandibular counter-clockwise rotation and mandibular advancement with TMJ Concepts® total joint prostheses. Part IV - Soft tissue response. *Int J Oral Maxillofac Surg*. 2009 Jun;38(6):637–46.

12. Coleta KED, Wolford LM, Gonçalves JR, dos Santos Pinto A, Cassano DS, Gonçalves DAG. Maxillo-mandibular counter-clockwise rotation and mandibular advancement with TMJ Concepts® total joint prostheses. Part II - Airway changes and stability. *Int J Oral Maxillofac Surg*. 2009 Mar;38(3):228–35.

13. Goncalves JR, Buschang PH, Goncalves DG, Wolford LM. Postsurgical Stability of Oropharyngeal Airway Changes Following Counter-Clockwise Maxillo-Mandibular Advancement Surgery. *Journal of Oral and Maxillofacial Surgery*. 2006 May;64(5):755–62.

14. Riley RW, Powell NB, Guilleminault CP, Alto S. Inferior sagittal osteotomy of the mandible with hyoid myotomy-suspension: A new procedure for obstructive sleep apnea.

15. Riley RW, Powell NB, Li KK, Troell RJ, Guilleminault C. Surgery and obstructive sleep apnea: Long-term clinical outcomes. *Otolaryngology-Head and Neck Surgery*. 2000;122(3):415–21.

16. Chan ASL, Sutherland K, Schwab RJ, Zeng B, Petocz P, Lee RWW, et al. The effect of mandibular advancement on upper airway structure in obstructive sleep apnoea. *Thorax*. 2010;65(8):726–32.
17. Elez F. Obstructive sleep apnea syndrome. *Turkiye Aile Hekimligi Dergisi* [Internet]. 2008;12(2):65–9. Available from: <https://www.turkailehekderg.org/jvi.aspx?un=TAHD-77785&volume=12&issue=2>
18. Sankri-Tarbichi AG. Obstructive sleep apnea-hypopnea syndrome: Etiology and diagnosis. *Avicenna J Med*. 2012 Jan;02(01):3–8.
19. Ng A, Gotsopoulos H, Darendeliler AM, Cistulli PA. Oral Appliance Therapy for Obstructive Sleep Apnea. Vol. 4, *Respir Med*. 2005.
20. Curado TF, Berger S, Polotsky VY. Pharmacotherapy of obstructive sleep apnea: Is salvation just around a corner? Vol. 199, *American Journal of Respiratory and Critical Care Medicine*. American Thoracic Society; 2019. p. 1186–7.





## CHAPTER VIII

# USE OF DWI-MRI IN DENTOMAXILLOFACIAL RADIOLOGY

**Büşra ERYİĞİT<sup>1</sup> & Taha Emre KÖSE<sup>2</sup> &  
Muhammed Enes NARALAN<sup>3</sup>**

*<sup>1</sup>(Research Assistant). Recep Tayyip Erdoğan University,  
Faculty Of Dentistry, Oral and Maxillofacial Radiology  
Department. busra.eryigit@erdogan.edu.tr  
ORCID :0009-0005-0938-8579*

*<sup>2</sup>(Assoc. Prof., PhD), Recep Tayyip Erdoğan University,  
Faculty Of Dentistry, Oral and Maxillofacial Radiology Department.  
tahaemre.kose@erdogan.edu.tr  
ORCID:0000-0003-3601-0393*

*<sup>3</sup>(Asst. Prof.), Recep Tayyip Erdoğan University, Faculty of Dentistry,  
Oral and Maxillofacial Radiology Department.  
muhammedenes.naralan@erdogan.edu.tr  
ORCID: 0000-0002-2444-4322*

### 1. Introduction

**M**agnetic resonance imaging (MRI), a non-invasive imaging method, produces anatomically accurate three-dimensional pictures. Applications for it include monitoring treatment progress and diagnosing diseases. Protons in water, which are prevalent in living tissues, are used by the technology to detect changes in the rotational axis of protons.(1) MRI is a vital tool for oncological assessment because it offers superb soft tissue contrast. To identify or classify lesions, T1 and T2 relaxation alterations might not be enough. To deal with such circumstances, diffusion-weighted imaging

(DWI) was created. To provide contrast in images, apparent diffusivity—the displacement of tissue water brought on by random, thermal motion—is employed.

## 2. General Information

Anatomically accurate three-dimensional images are created using a non-invasive imaging technique known as magnetic resonance imaging (MRI). Its applications include disease detection, diagnosis, and therapy monitoring. The technology relies on detecting changes in the axis of rotation of protons in water, which are abundant in living tissues.

MRI offers a wide range of examinations and applications, including:

- Imaging of the head area: Brain, eyes, inner ear, ear structures, pituitary gland, temporomandibular joint (TMJ), cerebral arteries, and vein systems.
- Neck imaging: Larynx, pharynx, salivary glands, tongue, and surrounding structures.
- Cardiovascular imaging: Lungs, heart, and large vessels associated with the heart.
- Abdominal imaging: Intra-abdominal organs and lower abdomen.
- Spinal pathologies: Neck, back, and lumbar region.
- Extremity and joint imaging: Shoulders, arms, elbows, wrists, hands, hips, thighs, knees, legs, ankles, and feet.
- Whole-body angiography: Detailed imaging of blood vessels throughout the body.
- Specialized techniques: MR spectroscopy, cranial and abdominal diffusion imaging, perfusion MRI, MRCP (Magnetic Resonance Cholangiopancreatography), MR pyelography, and MR myelography.
- CSF (Cerebrospinal Fluid) flow studies.
- Kinematic investigations: Assessing joint movements.
- Whole-body metastasis screening: Detecting cancer spread throughout the body.
- Dynamic tissue imaging: Assessing liver, breast, and tumour tissues.
- Regional MR angiographic examinations: Detailed evaluation of blood vessels in specific areas.

MRI has been widely used in dentistry, especially in TMJ evaluations, imaging of salivary glands, lymph nodes, masses in the head and neck, thyroid

and parathyroid glands, nasopharynx, tongue, and muscle examinations. It can also be used in caries detection and three-dimensional examination of the pulp. (1)

### **2.1. MRI**

The main type of nucleon sensitive to MRI, and the most prevalent atom in the body, is water (H<sub>2</sub>O). Thus, hydrogen nucleons are crucial for MRI. In isolation, water functions as a magnetic dipole for the unpaired proton-based hydrogen nucleons. In the absence of an external effect, these magnetic dipoles are randomly oriented and possess no net magnetic field (2).

When an external magnetic field is applied to the sample, net magnetization occurs, which can be in one of two ways: Parallel to the external magnetic field (spin up) or anti-parallel (opposite) to the magnetic field (spin down). These orientations represent the dipole's low- and high-energy states, respectively. Nuclei can transition from one energy state to another by absorbing or releasing a specific amount of energy. This transition is known as resonance, and it can involve electromagnetic energy in the radiofrequency pulse (RF) region of the electromagnetic spectrum (3).

When an external magnetic field is applied, the North and South poles of the nucleons do not perfectly align with the direction of the magnetic field. Instead, a slight tilt from a point where the axes of spinning protons were parallel to the flux of an external magnet causes them to oscillate or wobble, known as precession. The rate or frequency of precession is called the Resonant or Larmor frequency, and it varies with the intensity of the applied magnetic field. Magnetic fields with intensities ranging from 0.1 to 4.0T are employed for MR imaging (3).

When nuclei are exposed to an external magnetic field, they produce two energy states: Spin-up in the direction of the field and spin-down in the opposite direction. The interaction of these two energy levels results in a weak net magnetic moment, or magnetization vector, aligned parallel to the applied magnetic field. When an electromagnetic wave with a Larmor frequency matching that of the tissue's protons is directed towards tissue with protons aligned in the Z axis by the external magnetic field (imaging magnet), the protons absorb energy and rotate away from the direction induced by the imaging magnet.

The angle of rotation increases with the duration of the radiofrequency (RF) pulse. If the RF pulse is intense enough and rotates the net tissue magnetization vector into a transverse plane (XY plane) perpendicular to the longitudinal

alignment (Z-Axis), all protons will process in phase. This is known as a 90° RF pulse or a flip angle of 90°. At this moment, a receiver coil induces the strongest RF signal, which depends on the presence and bonding strength of hydrogen in the molecules.

The presence of loosely bonded or mobile hydrogen atoms in soft tissues and liquids results in a visible signal. The “proton density” or “spin density” of the tissue refers to the measurement of the concentration of loosely bound hydrogen nuclei available to generate the signal (3).

When the RF pulse is cut off, two processes occur simultaneously:

1. The nuclei release energy and return to their initial state of spin. This process is called relaxation, and the energy loss is measured as a signal known as free induction decay.

2. The transversely aligned nuclei start to realign to their original longitudinal orientation with the main magnetic field and net magnetization regions. This realignment is made possible by transferring energy from individual hydrogen nuclei (spin) to the surrounding molecules (Lattice).

## ***2.2. T1 And T2 Weighted Images***

A quick signal recovery time combined with a quick RF pulse repetition time (TR) results in T1-weighted images. A tissue with a short T1 produces an intense MR signal and appears bright white in a T1-weighted image because T1 is an all-exponential growth time constant. In an MR image, a tissue with a long T1 produces a signal of low intensity and looks dark. Second, transverse magnetization is lost as a result of the diphas caused by the magnetic moments of nearby hydrogen nuclei starting to interfere with one another. (4) T2 relaxation time / transverse (Spin) relaxation time is the unit of time used to express the rate of loss of transverse magnetization. The amplitude and duration of the detected radio signal, as well as the transverse magnetization, all rapidly decay (exponentially) to zero. Long signal recovery times and long RF pulse repetition times are used to acquire a T2-weighted image. Long T2 tissues generate a strong signal that is bright in the image. One with a short T2 is dark in the image and produces a signal of low intensity. (5)

Fat has the shortest T1 relaxation time and the lightest signal compared to other tissues and therefore appears bright in the image; T1-weighted images are also referred to as “fat images.” This kind of image’s strong image contrast makes it possible to see high anatomical detail. Thus, T1-weighted

images can be used to show small anatomical regions that call for high spatial resolution. (5)

Water has the longest T2 relaxation time of any substance, making it appear bright in T2 Weighted images. Normal tissues typically have a shorter T2 time than abnormal tissues. T2 weighted images are most frequently used when a doctor is searching for tumours and inflammatory changes. Anatomy illustrations are more frequently made using T1-weighted images. In order to separate the different tissues by contrast resolution in practice, images must frequently be acquired with both T1 and T2 weighting.

### ***2.3. Diffusion-Weighted Magnetic Resonance Imaging (DWI-MRI) And Apparent Diffusion Coefficient (ADC)***

MRI is a crucial tool in oncological evaluation, providing excellent soft tissue contrast. However, changes in T1 and T2 relaxation may not be sufficient to detect or characterize lesions. Diffusion-weighted imaging (DWI) is developed to address such situations.

Apparent diffusivity, which refers to the displacement of tissue water due to random, thermally driven motion, is used to create image contrast. DWI is a versatile tool for characterizing tissue structure and identifying disease processes. The amount of motion in DW-MRI is related to the mean path length that protons take due to thermally driven, random motion during a specific observation time period.

DWI-MRI uses two additional magnetic field gradients to make the MR signal motion-dependent. Signal loss occurs when water molecules move between the application of these gradients, depending on the mean diffusional path length. The apparent diffusion coefficient (ADC) map is utilized in DWI-MRI, providing information about diffusion and perfusion components.

High diffusion areas appear as bright regions with a high ADC value in the ADC map. Preferential diffusion directions can be analysed using diffusion tensor imaging (DTI), which is valuable for assessing tissue architecture like fiber tracts in the brain.

DWI-MRI is widely used in whole-body malignancy screening and assessing treatment response. It is employed in breast cancer diagnosis and detection of cancer extension. ADC values can help distinguish benign and malignant liver lesions and may predict treatment response in hepatic metastases.

Additionally, DWI-MRI, particularly with diffusion tensor imaging, aids in defining tumour margins and their relationship with fiber tracts, contributing to lesion characterization in the head and neck.

Overall, DWI-MRI is a valuable tool for investigating tissue microstructure, providing crucial information for oncological evaluation and treatment response assessment.

#### ***2.4. Dentomaxillofacial Radiology Usage***

Diffusion weighted imaging has been studied many lesions located in the maxillofacial region from malignancies to bone lesions.

Wang et al. conducted a study on 81 lesions in the head and neck area, including non-Hodgkin lymphoma, carcinoma, benign solid masses, and benign cystic lesions without odontogenic origin. The study found statistically significant differences in the mean ADC values among these groups. The order of ADC values from higher to lower was as follows: Benign cystic lesions, benign solid tumours, carcinomas, and lymphomas. Additionally, poorly differentiated carcinomas and lymphomas showed similar ADC values, indicating shared histologic and cytologic features. (6)

Yuan et al. investigated 42 palatal lesions, including 31 patients with malignant lesions (such as squamous cell carcinoma, adenoid cystic carcinoma, mucoepidermoid carcinoma, MALT lymphoma, malignant pleomorphic adenoma, malignant melanoma, and lymphoepithelial carcinoma) and 11 patients with benign lesions (pleomorphic adenoma, benign lymphoepithelial lesion, and inflammation). The study found that malignant lesions had lower ADC values compared to benign palatal lesions. (7)

In the literature, it has been shown that DWI-MRI detects lymph node metastasis with 98% sensitivity and 88% specificity. DWI-MRI is reported to be superior to conventional MRI in assessing lymph nodes smaller than 1 cm, but there is no significant difference in its ability to detect lymph nodes larger than 1 cm. DWI can help detect cancer-involved structures and differentiate between metastatic and reactive lymph nodes. (8)

Muraoka et al. reported the CT and MRI results of 5 patients with central vascular anomalies. They found a wide range of ADC values and variable signal intensities for these lesions. The researchers concluded that the presence of arteriovenous and venous architecture may explain these findings. (9)

Oda et al. conducted a study investigating the DWI of various maxillofacial lesions, including squamous cell carcinoma, medication-related osteonecrosis, odontogenic abscess, ranula, osteoradionecrosis, haemangioma, pleomorphic adenoma, odontogenic keratocysts, nasopalatine duct cyst, malignant melanoma, and basal cell carcinoma. They reported

that ranula and nasopalatine duct cyst had mean ADC values of  $2.69 \pm 0.59$  and  $2.34 \pm 0.12$ , respectively, which were significantly higher than all other lesions in the study. Odontogenic abscess had the lowest mean ADC value of  $0.67 \pm 0.36$ . Among the malignant lesions, squamous cell carcinoma had the highest ADC values, while malignant melanoma had the lowest ADC values in the study. (10)

In one study, Muraoka et al. (11) investigated patients with different types of osteomyelitis: 6 with chronic diffuse sclerosing osteomyelitis (CDSO), 21 with acute suppurative osteomyelitis (ASO), and 13 with chronic suppurative osteomyelitis (CSO). They found statistically significant differences in the mean ADC values between ASO and CSO, as well as between CSO and CDSO. However, there was no significant difference in the mean ADC values between ASO and CDSO. The mean ADC values were reported in the order of  $ASO > CDSO > CSO$ .

In another study on osteomyelitis, Muraoka et al. investigated the efficiency of diffusion-weighted imaging (DWI) in patients with osteomyelitis. They divided the patients into three groups: Non-osteomyelitis ( $n=22$ ), acute osteomyelitis ( $n=27$ ), and chronic osteomyelitis ( $n=21$ ). The mean ADC values for the three groups were  $0.87 \pm 0.15 \times 10^{-3}$ ,  $1.24 \pm 0.11 \times 10^{-3}$ , and  $1.07 \pm 0.13 \times 10^{-3}$ , respectively. There were statistically significant differences in ADC values between all three groups. (4)

In the study by Ogura et al. (12), they investigated patients with osteoradionecrosis and medication-related osteonecrosis of the jaw using CT, MRI, and scintigraphy. They included 13 patients with medication-related osteonecrosis of the jaw (MRONJ) and 7 patients with osteoradionecrosis (ORN). They observed high signal intensity on Short Tau Inversion Recovery (STIR) and DWI, and low signal intensity on ADC mapping in all patients with MRONJ and ORN.

DWI imaging is also utilized in the evaluation of the TMJ. Muraoka et al. (13) conducted a study to investigate the ADC values of inflammatory connective tissue around the mandibular condyle in healthy individuals and patients with rheumatoid arthritis (RA). The study found that the mean ADC values were  $1.26 \pm 0.11 \times 10^{-3}$  for the control group (healthy individuals) and  $1.60 \pm 0.19 \times 10^{-3}$  for the RA group. The ADC values of the RA group were significantly higher than those of the control group, suggesting differences in tissue characteristics between the two groups, potentially related to inflammation in the TMJ.



Based on traditional imaging modalities (panoramic radiography, computed tomography, traditional MR imaging) alone, some types of cystic lesions of the mandible are difficult to differentiate from one another (14). However, DWI imaging may help to differentiate the lesions, and the literature shows some promise.

Eida et al. (14) conducted a study investigating ADC-based differentiation of cystic lesions of the mandible. They evaluated 27 cystic lesions, including ameloblastomas, simple bone cysts, dentigerous cysts, radicular cysts, and keratocystic odontogenic tumours, located in the mandible. The study results showed that the overall ADC values of ameloblastoma and simple bone cysts were larger than those of dentigerous cysts, radicular cysts, and keratocystic odontogenic tumours. However, there was no difference in ADC values between ameloblastoma and simple bone cysts themselves, nor between radicular cysts, keratocystic odontogenic tumours, and dentigerous cysts.

Han et al. (15) conducted a similar study in collaboration with Eida et al. to investigate DWI images of unicystic ameloblastomas, calcifying cystic odontogenic tumors (CCOT), multicystic ameloblastoma, and dentigerous cysts. They reported that unicystic ameloblastomas had much higher ADC values compared to CCOT and dentigerous cysts. However, the mean ADC values of CCOT and dentigerous cysts were not significantly different from each other.

In Wamasing et al.'s study (16), a total of 127 cystic lesions, including 80 dentigerous cysts, 39 odontogenic keratocysts, and 8 unicystic ameloblastomas, were investigated using MRI. The ADC values were able to discriminate between the lesions of dentigerous cysts and unicystic ameloblastomas, as well as odontogenic keratocysts and unicystic ameloblastomas. However, there was no significant difference observed in the ADC values of dentigerous cysts and odontogenic keratocysts. Srinivasan et al. (17) also reported statistically significant different ADC values between odontogenic keratocysts and unicystic ameloblastomas.

Vanagundi et al. (18) investigated the ADC values of odontogenic keratocysts (n=17), unicystic ameloblastomas (n=5), and dentigerous cysts (n=5). They reported that in 14 patients with odontogenic keratocysts, there was restricted diffusion, while the remaining 3 patients showed facilitated diffusion values. On the other hand, all unicystic ameloblastomas and dentigerous cysts showed facilitated diffusion. Unlike Han and colleagues' study, Vanagundi and colleagues suggested that the difference in diffusion values in dentigerous

cysts could be due to the lower concentration of glycosaminoglycans within the included dentigerous cysts in their study.

Ogura et al. (19) assessed different cystic lesions and normal anatomical structures' ADC values in their study. They included 16 patients with various cystic lesions, which consisted of odontogenic keratocysts (n=5), simple bone cyst (n=1), nasopalatine duct cyst (n=3), radicular cyst (n=3), and dentigerous cyst (n=4). The mean ADC values for normal anatomical structures were as follows: spinal cord ( $0.71 \pm 0.20 \times 10^{-3}$ ), Waldeyer's ring ( $0.75 \pm 0.11 \times 10^{-3}$ ), nasal mucosa ( $1.80 \pm 0.19 \times 10^{-3}$ ), and cerebrospinal fluid ( $3.66 \pm 0.47 \times 10^{-3}$ ). Regarding the cystic lesions, the mean ADC values were odontogenic keratocysts ( $1.03 \pm 0.31 \times 10^{-3}$ ), simple bone cyst ( $2.79 \times 10^{-3}$ ), nasopalatine duct cyst ( $2.28 \pm 0.12 \times 10^{-3}$ ), radicular cyst ( $1.82 \pm 0.71 \times 10^{-3}$ ), and dentigerous cyst ( $1.67 \pm 1.06 \times 10^{-3}$ ). The study found that the lowest ADC values in the cyst group were observed in odontogenic keratocysts.

Kojima et al. (20) reported a case of florid cemento-osseous dysplasia associated with a simple bone cyst. They found no hyperintense signal on diffusion-weighted MRI and observed high ADC values in the cystic areas of the mandible.

Vidmar et al. (21) investigated the potential of DWI imaging in endodontic diagnosis. They studied 26 human teeth with or without caries lesions using high-resolution MRI at 2.35 T. The teeth were assessed according to the International Caries Detection and Assessment System (ICDAS) severity score by two independent observers. They reported a negative correlation between the average ADC values of dental pulp and the depth of demineralization.

### 3. Conclusion

DWI MR shows promise in dentistry for diagnosis and treatment determination. However, further studies are necessary to validate and confirm these findings.

### References

1. Eşer G , Duman ŞB , Başaran M , Aşantoğrol F. Manyetik Rezonans Görüntüleme ve Diş Hekimliği. *BSJ Health Sci.* 2022; 5(1): 130-137.
2. Pekar JJ. A brief introduction to functional MRI. *IEEE Eng Med Biol Mag.* 2006;25(2):24-26. doi:10.1109/memb.2006.1607665
3. White SC, Pharoah MJ. *Oral radiology: principles and interpretation.* Mosby, St Louis 2000: 205-6.

4. Muraoka H, Hirahara N, Ito K, Okada S, Kondo T, Kaneda T. Efficacy of diffusion-weighted magnetic resonance imaging in the diagnosis of osteomyelitis of the mandible. *Oral Surg Oral Med Oral Pathol Oral Radiol.* 2022;133(1):80-87. doi:10.1016/j.oooo.2021.06.007.

5. Katti G, Ara SA, Shireen A. Magnetic Resonance Imaging (MRI) – A Review. *International Journal of Dental Clinics.* March 2011; 3(1):65-70.

6. Wang J, Takashima S, Takayama F, et al. Head and neck lesions: characterization with diffusion-weighted echo-planar MR imaging. *Radiology.* 2001;220(3):621-630. doi:10.1148/radiol.2202010063.

7. Yuan Y, Tang W, Jiang M, Tao X. Palatal lesions: discriminative value of conventional MRI and diffusion weighted imaging. *Br J Radiol.* 2016;89(1059):20150911. doi:10.1259/bjr.20150911.

8. Pałasz P, Adamski Ł, Górska-Chrzastek M, Starzyńska A, Studniarek M. Contemporary Diagnostic Imaging of Oral Squamous Cell Carcinoma - A Review of Literature. *Pol J Radiol.* 2017;82:193-202. Published 2017 Apr 7. doi:10.12659/PJR.900892.

9. Muraoka H, Kaneda T, Kondo T, Tokunaga S. Central vascular malformations of the mandible: characteristic findings in computed tomography and magnetic resonance imaging. *Oral Surg Oral Med Oral Pathol Oral Radiol.* 2023;135(6):e123-e130. doi:10.1016/j.oooo.2023.04.006.

10. Oda T, Sue M, Sasaki Y, Ogura I. Diffusion-weighted magnetic resonance imaging in oral and maxillofacial lesions: preliminary study on diagnostic ability of apparent diffusion coefficient maps. *Oral Radiol.* 2018;34(3):224-228. doi:10.1007/s11282-017-0303-y.

11. Muraoka H, Kaneda T, Hirahara N, Ito K, Okada S, Kondo T. Diagnostic Efficacy of Diffusion-weighted Imaging in Distinguishing Chronic Diffuse Sclerosing Osteomyelitis from Suppurative Osteomyelitis of the Mandible. *Magn Reson Med Sci.* 2023;22(3):283-288. doi:10.2463/mrms.mp.2021-0153.

12. Ogura I, Sasaki Y, Sue M, Oda T, Kameta A, Hayama K. Tc-99m hydroxymethylene diphosphonate scintigraphy, computed tomography, and magnetic resonance imaging of osteonecrosis in the mandible: Osteoradionecrosis versus medication-related osteonecrosis of the jaw. *Imaging Sci Dent.* 2019;49(1):53-58. doi:10.5624/isd.2019.49.1.53.

13. Muraoka H, Ito K, Hirahara N, Okada S, Kondo T, Kaneda T. Quantitative Assessment of the Apparent Diffusion Coefficient Values of the Inflammatory Connective Tissue Around the Mandibular Condyle in Rheumatoid Arthritis. *J Oral Maxillofac Surg.* 2021;79(6):1230-1235. doi:10.1016/j.joms.2021.01.014.

14. Eida S, Hotokezaka Y, Katayama I et al. Apparent diffusion coefficient-based differentiation of cystic lesions of the mandible. *Oral Radiol* ;2012;28:109-114.
15. Han Y, Fan X, Su L, Wang Z. Diffusion-Weighted MR Imaging of Unicystic Odontogenic Tumors for Differentiation of Unicystic Ameloblastomas from Keratocystic Odontogenic Tumors. *Korean J Radiol*. 2018;19(1):79-84. doi:10.3348/kjr.2018.19.1.79.
16. Wamasing N, Watanabe H, Sakamoto J, Tomisato H, Kurabayashi T. Differentiation of cystic lesions in the jaw by conventional magnetic resonance imaging and diffusion-weighted imaging. *Dentomaxillofac Radiol*. 2022;51(1):20210212. doi:10.1259/dmfr.20210212.
17. Srinivasan K, Seith Bhalla A, Sharma R, Kumar A, Roychoudhury A, Bhutia O. Diffusion-weighted imaging in the evaluation of odontogenic cysts and tumours. *Br J Radiol*. 2012;85(1018):e864-e870. doi:10.1259/bjr/54433314.
18. Vanagundi R, Kumar J, Manchanda A, Mohanty S, Meher R. Diffusion-weighted magnetic resonance imaging in the characterization of odontogenic cysts and tumors. *Oral Surg Oral Med Oral Pathol Oral Radiol*. 2020;130(4):447-454. doi:10.1016/j.oooo.2020.04.010.
19. Ogura I, Nakahara K, Sasaki Y, Sue M, Oda T. Diffusion-weighted Magnetic Resonance Imaging in Odontogenic Keratocysts: Preliminary Study on Usefulness of Apparent Diffusion Coefficient Maps for Characterization of Normal Structures and Lesions. *Chin J Dent Res*. 2019;22(1):51-56. doi:10.3290/j.cjdr.a41775.
20. Kojima I, Nishioka T, Sakamoto M, et al. Florid Cemento-Osseous Dysplasia-Associated Simple Bone Cyst Showing Marked Irregular Border and High Apparent Diffusion Coefficient Value. *Case Rep Dent*. 2020;2020:8854428. Published 2020 Sep 21. doi:10.1155/2020/8854428.
21. Vidmar J, Cankar K, Nemeth L, Serša I. Assessment of the dentin-pulp complex response to caries by ADC mapping. *NMR Biomed*. 2012;25(9):1056-1062. doi:10.1002/nbm.2770.

