# MACEDONIAN DENTAL REVIEW

ISSN 2545-4757 | 2 43 2020

Macedonian Dental Review is publishing by the Faculty of Dentistry, University „Ss. Cyril and Methodius“, Skopje, Republic of North Macedonia and Macedonian Dental Society

**http://stomfak.ukim.edu.mk/msp/**

**MACEDONIAN DENTAL REVIEW** **Contents**

**2020** ● Year **XXXXIII** ● Nummber 2 ● стр. 49-84



**Macedonian Dental Review** is publishing by the Faculty of Dentistry, University „Ss. Cyril and Methodius“, Skopje, Republic of North Macedonia and Macedonian dental society.

**Editor in chief** - Prof. **Kjiro Ivanovski**, PhD, e-mail: kiroivanovski@stomfak.ukim.edu.mk
**Associate editor -** Prof. **Elizabeta Georgievska** PhD, Department of Pediatric and Preventive Dentisty, e-mail: egjorgievska@stomfak.ukim.edu.mk
**Secretary** - **Vlatko Kokalanski**, MBA, e-mail: vkokolanski@stomfak.ukim.edu.mk

**Adress - Macedonian Dental Review,** str. Majka Tereza br. 43 Skopje, Republic of North Macedonia, **http://stomfak.ukim.edu.mk/msp/**

**2020** ● година **XXXXIII** ● Број 2 ● Страна 49 - 84



**Македонски стоматолошки преглед** го издава Стоматолошкиот факултет при Универзитетот „Св. Кирил и Методиј" Скопје, Република Северна Македонија и Македонското стоматолошко друштво.

Одговорен уредник: Проф. д-р **Ќиро Ивановски**, e-mail: kiroivanovski@stomfak.ukim.edu.mk
Заменик одговорен уредник: Проф. д-р **Елизабета Ѓоргиевска**, e-маил: egjorgievska@stomfak.ukim.edu.mk
Секретар на списанието: м-р **Влатко Коколански**, e-mail: vkokolanski@stomfak.ukim.edu.mk

**Адреса - Македонски стоматолошки преглед**, ул. Мајка Тереза бр. 43 Скопје, Република Северна Македонија
**http://stomfak.ukim.edu.mk/msp/**

UDC: 616,314–77:616.314–022.7

# THE IMPACT OF DIFFERENT SURFACE CHARACTERISTICS OF FIXED-PROSTHODONTICS RESTORATIVE MATERIALS ON THE EXISTENCE OF PATHOGENIC BACTERIA

# ВЛИЈАНИЕ НА РАЗЛИЧНИ КАРАКТЕРИСТИКИ НА МАТЕРИЈАЛИ ЗА ФИКСНО-ПРОТЕТИЧКИ НАДОМЕСТОЦИ ВРЗ ПРИСУСТВОТО НА ПАТОГЕНИ БАКТЕРИИ

**Bislimi F., Bajevska J., Bajevska J.**

Department of Prosthodontics, Faculty of Dentistry, UKIM, Skopje, North Macedonia

## Abstract

Dental plaque on the teeth enamel and surfaces of restorative materials plays an important role in the pathogenesis of oral health. Therefore, there is a great interest in the production of materials that reduce or inhibit dental plaque formation. The purpose of this paper is to present the influence of different surface characteristics of restorative materials on bacterial adhesion through a literature review. Articles published in the electronic bibliographic databases (Medline-Pubmed) have been searched for the following terms: dental plaque or biofilm and restorative materials (composites, porcelain, titanium, Co-Cr alloys, and zirconia), dental plaque or biofilm and surface characteristics, dental plaque or biofilm and surface roughness, dental plaque and surface free energy. Surface characteristics such as surface roughness (SR), surface free energy (SFE), and chemical composition can affect bacterial adhesion and plaque formation. From the literature review it can be concluded that the surface of the materials has a decisive influence on the formation of bacterial plaque and, above all, its roughness. Increasing the coefficient of roughness of the surface and also increasing the surface free energy leads to the formation of biofilm on the surface of the materials. Since papers presented different methodological approaches, the results yielded different and sometimes contradictory outcomes. **Keywords:** dental plaque, biofilm, bacterial cells, restorative materials, surface roughness, surface free energy.

## Апстракт

Формирањето дентален плак во емајлот на забите и површините на реставративните материјали игра важна улога во патогенезата на оралното здравје. Затоа, постои голем интерес за производство на материјали кои го намалуваат или го инхибираат формирањето дентален плак. Целта на овој труд е преку литературен преглед да се објасни влијанието на различни карактеристики на различни реставративни материјали врз бактериската адхезија. Во пребарувањето се користени следните елементи: дентален плак или биофилм и реставративни материјали (композити, порцелан, титаниум, легури на Co-Cr, циркониум диоксид), дентален плак или биофилм и површински карактеристики, дентален плак или биофилм и површинска грубост, дентален плак или биофилм и површинска слободна енергија. Карактеристиките на површината на денталните материјали како површинска грубост (SR), површинска слободна енергија (SFE) и нивниот хемиски состав, може да имаат влијание на бактериската адхезија и формирање на плакот. Од литературниот преглед може да се констатира дека површината на материјалите има одлучувачко влијание врз формирањето на бактерискиот плак, а пред сè, неговата грубост. Зголемувањето на коефициентот на грубост на површината, а исто така и зголемувањето на т.н. слободна површинска енергија води до формирање биофилм на површината на материјалите. Бидејќи во трудовите постои голема хетерогеност и различни методолошки пристапи, добиените резултати дадоа различни вредности, а некогаш и контрадикторни. **Клучни зборови:** биофилм, дентален плак, бактериски клетки, реставративни материјали, површинска грубост, слободна површинска енергија.

## Introduction

Fixed prosthetic restorations can be made of different materials, such as metal (titanium, chromium - cobalt alloys), types of ceramics, composites, and other contemporary materials.

Particular attention should be given to the type of materials used in the manufacture of fixed-prosthetic restorations, because they are in direct contact with periodontal tissues and can easily compromise their health.

Bacterial accumulation in the gingival margin areas of the tooth and restorative materials is a key factor in

encouraging secondary decay, which is one of the main reasons for the replacement of restorations[1,2]. Therefore, there is a growing interest in the production of materials that reduce or inhibit dental plaque formation[3].

Dental plaque, as an oral biofilm, is recognized as a key factor for decay and periodontal inflammation in humans. Bacterial colonization of dental surfaces or dental materials - such as dental filling materials, dental implants or prosthetic restorations, begins immediately after exposure to the oral environment. In the process of plaque formation, early colonizers, including Streptococcus sanguinis, adhere to the salivary layer covering the dental surfaces[4]. This initial adhesion is an important step in the formation of a biofilm that may affect the dental plaque composition.

More than 700 different bacterial species have been found in the oral cavity, of which more than 50% cannot be cultivated. Microflora of the teeth, tongue, buccal epithelium, soft and hard palate, and vestibulum consists 20-30 different dominant species at each site, and the number of dominant species per individual ranges from 30 to 70. The most common species belongs to Gemella, Granulicatella, Streptococcus and Veillonella[5, 6].

A number of factors have been identified that influence the formation of biofilm, such as surface roughness and surface free energy. Microscopic studies of early dental plaque formations have shown adhesion of the initial colonized bacteria along the cracks and pits in the enamel, indicating the influence of surface structure on bacterial adhesion[7]. Mjor et al.[8] have reported that the margins of dental restorations stimulates bacterial recolonization and acid production as metabolic substances of cariogenic bacteria.

In addition, many studies have found that there is a variation in the effect of different types of restorations on the growth of specific bacteria on dental plaque according to its material composition[9, 10, 11].

Various studies have shown that restoration margins have always been suitable sites for plaque accumulation and reproduction of bacteria that results with gingival inflammation and even tooth loss in some cases. Restoration materials and cement can affect periodontal tissues in different ways and result in gingivitis and gingival damage. These developments usually occur in restorations with subgingival margins and may be due to physical or chemical characteristics of the materials. In most cases, these destructive reactions are due to the roughness of the surfaces of dental materials rather than their composition[12]. In such cases the rough surface results in gingivitis and accumulation of more plaque.

The literature emphasizes the fact that besides the biochemical factor, the non-specific physio-chemical factor also plays an important role in the adhesion phenomenon. From the physio-chemical aspect, the phenomenon of bacterial adhesion is explained by two theories: thermodynamic and classical. The thermodynamic theory of bacterial biofilm formation is explained by the action of free surface energy (SFE) between adjacent surfaces and liquids. In contrast, classical DLVO theory (Derjalung, Landau, Verwey, Overbeek) explains the mechanism of biofilm formation as a long-term reaction between a bacterial cell and the tooth surface due to attractive van der Waals forces and repulsive electrostatic forces[13].

## Aim of the paper

The purpose of this paper is to evaluate and compare the influence of surface characteristics of restorative materials such as surface roughness, surface free energy, and surface chemistry on bacterial adhesion of different restorative materials used in making fixed prosthetic restorations.

## Materials and methods

Articles published in the electronic bibliographic databases (Medline-Pubmed) have been searched for the following terms: dental plaque or biofilm and restorative materials (composites, porcelain, titanium, Co-Cr alloys, and zirconia), dental plaque or biofilm and surface characteristics, dental plaque or biofilm and surface roughness, dental plaque and surface free energy. In vitro and in vivo studies are included in this study. The papers contained great heterogeneity, different methodological approaches and outcome changes.

### Surface Roughness

Surface roughness (SR) measurement is an important aspect in determining the surface properties that influence biofilm formation. Different techniques can be used to evaluate this parameter. Research on this topic includes qualitative assessments [atomic force microscope (AFM) and scanning electron microscopy (SEM)] and quantitative methods (2D and 3D surface analysis profile). Quantitative profile analysis can be performed with a contact diamond laser and a non-contact laser[14].

Because surface topography is three-dimensional in nature, 3D surface topography measurement provides a more realistic analysis of the surface and gives a complete description of the surface topography. Laser or white light profilometry enables the three-dimensional study of the specimen's (or material's) surface without any contact[15].

For the purpose of qualitative evaluation, measurement is usually used to observe the scratches and imperfections of material surfaces. However, SEM is a limitation in defining surface topography, as it only allows morphological evaluation of the sample surface[16].

For visual and high resolution qualitative analysis of surface topography, the use of AFM seems more appropriate. In addition to SEM, AFM can offer more detailed topography of the surface, providing three-dimensional surface analysis in nanometric resolution.

## Surface Free Energy

Surface Free Energy (SFE) is described as all solid surface energy equivalent to the liquid surface voltage. It is defined[17] as "the work required to increase the surface area of a substance by 1cm$^2$" and is an important factor in determining surface reactivity. Several different approaches can be used to determine SFE by measuring the angle of contact (θ) formed by different liquids (distilled water, ethylene glycol, and glycerol) that are different in hydrophobicity on a certain surface.

## Chemical Composition

The chemical composition of the dental material will further affect the bacterial adhesion since both proteins and microorganisms can chemically attach or get attracted to components in the material, by van der Waal forces, acid-base reactions or electrostatic interactions[18]. In most patients, there will be several different materials present in the mouth simultaneously which can interfere with the biofilm formation and the microbiota in general. The chemical interaction between material and microorganisms can lead to alterations in the surface properties over time[19].

### *In vivo and in vitro experiments to study the development and adhesion of bacteria*

In-vivo experiments by Glantz[20] in 1969 show that surfaces characterized by high free surface energy (SFE) are more susceptible to bacterial adhesion. Research by Quirynen et al.[21] show a correlation between the value of free surface energy and the amount of plaque. Low-SFE surfaces are characterized by less mature supra - and subgingival plaque. Comparing the interdependence between the free surface energy and the degree of surface roughness, it has been shown that the degree of surface roughness is an important factor for bacterial adhesion.

Imgard Hauser G et al.[4] performed an in vitro study that aimed to compare the adhesion of Streptococcus sanguinis on integrated dental implants and restorative materials versus human tooth enamel, and also to determine the viability of bacteria that initially adhere. In their study, the testing materials were titanium, gold, ceramics, and composites. Rectangular test specimens were used and polished. Surface roughness was measured with a Hommel tester. The viability of the adhered bacteria was estimated using a double fluorescence staining method that allows differentiation of vital and non-vital bacterial cells according to Decker[22]. The surface roughness of the tested materials was Ra = 0.24 μ which corresponds to the average roughness of the enamel surface.

The obtained results showed differences in cell adhesion and vitality of the adhered cells, thereby indicating different characteristics of the substrate material. It has been shown that the physical and chemical properties of the materials - such as surface free energy, hydrophobicity and roughness, as well as the composition (composition) of the material, influence the initial bacterial adhesion[3, 23]. The number of adhered Streptococcus sanguinis cells per mm$^2$ was significantly greater in surfaces of titanium, gold, and ceramics than in enamel, whereas bacterial adhesion in the composites was significantly lower, and their vitality was lower compared to those found on the enamel surface. The percentage of adhered vital Streptococcus sanguinis was higher in enamel (92.5%) whereas it was significantly lower in all four tested restorative materials (41.5-69.1%). These results are in accordance with other studies[24, 25, 13]. However, it was noted that fewer bacteria were retained in the composites, although the hydrophilic properties of the surfaces were similar to the other materials tested. In addition, some dental restorations release metal ions or fluorides in the medium - with a possible impact on the vitality of the adhered bacteria[22, 26]. This may enhance the explanation for the low percentage of vital adherent cells in the restorative materials used in this study.

Bulem Yzugullu et al.[27] in their study investigated the effect of feldspar porcelain surface treatment on the adhesion of Streptococcus mutans. Ninety-six porcelain specimen discs were fabricated and divided into six equal groups according to surface treatment: group 1- fine-grit diamond polishing, group 2 - self-glazing, group 3 - overglazing, group 4 - overglazing followed by a finishing procedure and then overglazing, group 5 - Pearl Surface polishing and group 6 - Diamond Twist SCLTM polishing. Mean Ra values and standard deviation of porcelain samples after different surface treatments showed statistically significant disparities in surface roughness. The contact angle was also influenced by the procedures used to process the surfaces. The high values of the contact angle led to all the specimens hav-

ing hydrophobic surface properties. The group of specimens glazed twice after grinding (group 4), which largely eliminated surface irregularities and reduced surface roughness values (Ra = 0.8 μm), showed the lowest bacterial adhesion, while the highest bacterial adhesion value was present in the group of specimens having the highest surface roughness value (Ra = 1.6 μm) (group 1). Reglazing after grinding may therefore decrease bacterial adhesion beneficially.

Quirynen and Bollen[23] suggest that surface roughness and surface free energy are the major factors affecting bacterial adhesion. They further show that the influence of surface roughness is greater than the surface free energy and surface hydrophobicity. Generally, roughness of surfaces promotes bacterial adhesion while smooth surfaces minimize it.[23, 28] According to Bollen et al.[29] roughness of surfaces less than Ra = 0.2 μm has no quantitative and qualitative effects on bacterial adhesion. In addition, variations around this value have a negligible effect on bacterial adhesion. In this study, the roughness of all samples is about 0.2 μm, hence, it turns out that differences in bacterial adhesion cannot be explained in terms of surface roughness. This would mean that any observation of differences in bacterial adhesion is likely due to other properties of the surface and the composition of the used materials.

The literature emphasizes the fact that on the surface of restorative materials, there is greater accumulation of plaque and retention of bacteria than on the surface of enamel[10, 11]. The amount and composition of bacterial biofilm in prosthetic restorations has been found to vary and depend on the type of restorative materials[30]. Restoration materials such as ceramics, composites, titanium, and Cr-Co-Mo alloys have been tested in chambers with special conditions that imitate the oral cavity (laminar flow chamber). The adhesion of Streptococcus mutans showed significant differences between different materials. The highest values of bacterial adhesion were found in composite samples, while the lowest values of bacterial adhesion were found in chromium-cobalt alloys and titanium restorations. Streptococcus mutans showed moderate adhesion to ceramic specimens which was larger than the alloys and lower than the composites. According to some authors, these result values are due to the bacteriostatic properties of the metals used30. This findings are contrary to the findings of Imgard Hauser et al.[4].

Jalalian et al.[31] evaluated the adhesion of Streptococcus mutans to zirconia, feldspar porcelain, titanium alloys, and indirect composite resin. The study used 10 samples (5mm diameter and 1mm thick) of each material. The enamel was used as a reference value. Bacterial adhesion was determined using a scanning electron microscope. The results showed the highest bacterial adhesion in the composite specimens, while the lowest bacterial adhesion was observed in the zirconia specimens. The effect of surface roughness was also studied, but no correlation was found between surface roughness and bacterial adhesion. Yet, another study conducted by Yu et al.[32] showed that increased surface roughness of zirconia and its hydrophobicity resulted in increased surface forces of adhesion and early attachment of Streptococcus mutans.

The density and morphological aspects of the biofilm adhered to different prosthetic restoration materials were investigated by Julio et al.[33], using 60 cylindrical specimens divided into four groups: porcelain, Co-Cr alloy, titanium, and zirconium. Procedures were then performed in a microbiological laboratory for the cultivation of biofilm in human saliva. The unit of measurement used for counting colonies on surfaces was CFU/cm$^2$, analyzed by spectrophotometry (absorption) and scanning electron microscopy (FEG-SEM). The highest absorption values and number of CFU/cm$^2$ colonies were recorded in biofilms grown in Co-Cr alloys within the first 24 hours and after 48 hours, compared to the other materials used in the study. FEG-SEM images also showed higher biofilm density in Co-Cr alloys.

The results of this study show that ceramic surfaces induce a low density of biofilm associated with a small number of colonies. This may be related to the low level of free energy intensity found on ceramic surfaces. Also, titanium-developed biofilm reveals low density, which can be explained by the presence of passive titanium oxide film (mainly $TiO_2$). Biofilm morphology was also observed by scanning electron microscopy, which showed a lower biofilm growth after 48 hours in porcelain and zirconia compared to titanium. The biofilm that developed on zirconia and titanium also showed a slight increase in density after 48 h growth compared with that of 24 h growth in relation to the same material. However, porcelain biofilm density remained stable for periods of 24 and 48 hours. In fact, the present results indicate a trend towards higher accumulation of oral biofilms of prosthetic structures based on CoCr alloys when compared to those based on titanium or zirconia.

In most studies, human tooth enamel was treated as a place with the lowest adhesion level of dental plaque compared to restorative materials, but in an in vitro study by Jalalian et al.[34] comparing the adhesion level of Streptococcus mutans in polished IPS e. max, feldspar porcelain and enamel met different results. The study was conducted in vitro. Porcelain samples were polished with an ultradent 0.5 μm diamond polish for 60 seconds while the enamel samples were not polished. They were

then exposed to a standard bacterial suspension of Streptococcus mutans in a microbiological laboratory at a concentration of 1x106 mg/ml. The results showed higher adhesion of Streptococcus mutans to enamel samples, while the lowest value of adhesion were encountered in IPS e.max.

## Conclusion

From the literature review it can be concluded that the latest research on bacterial adhesion to materials used for dental reconstruction, the surface of the material has a decisive influence on the formation of bacterial plaque, and above all its roughness. Increasing the surface roughness coefficient above the Ra value of 0.2 μm and increasing the surface free surface energy leads to the formation of biofilm on the surface of materials.

However, evaluation of surface chemistry may also be suitable for studying the biological behavior of restorative materials. Restorative materials submitted in different surface treatment protocols may show similar roughness and SFE values, but a different chemical surface composition may affect their biological performance.

Dental ceramics is a material that has the least ability to absorb bacteria on its surface compared to other materials. Comparing different types of ceramics, zirconia is the material with the lowest degree of bacterial adhesion.

Since the cited articles presented different methodological approaches, the results yielded different and sometimes contradictory outcomes.

## Reference


1. Deligeorgi V., Mjor IA., Wilson NH. An overview of reasons for the placement and replacement of restorations. Prim Dent Care. 2001; 8:5-11.
2. Fontana M., Gonzalez-Cabezas C. Secondary caries and restoration replacement: an unresolved problem. Compend Contin Educ Dent. 2000; 21:15-18.
3. Boeckh C., Schumacher E., Podbielski A., Haller B. Antibacterial activity of restorative dental biomaterials in vitro. Caries Res. 2002; 36:101-107.
4. Hauser-Gerspach I, Kulik EM., Weiger R., Decker EM., Von Ohle C., Meyer J. Adhesion of streptococcus sanguinis to dental implant and restorative materials –in vitro. Dent Mater J. 2007; 26(3): 361-6.
5. Mager DL., Ximenez-Fyvie LA., Haffajee AD., Socransky SS. Distribution of selected bacterial species on intraoral surfaces. J Clin Periodontol. 2003; 30:644-654.
6. Aas JA., Paster BJ., Stokes LN., Olsen I., Dewhirst FE. Defining the normal bacterial flora of the oral cavity. J Clin Microbiol 2005; 43:5721–5732.
7. Lee BC, Jung GY, Kim DJ, Han JS. Initial bacterial adhesion on resin, titanium and zirconia in vitro. J Adv Prosthodont. 2011; 3: 81-4.
8. Mjor IA. The frequency of secondary caries at various anatomical locations. Oper Dent. 1985; 10:88-92.
9. Mjor IA, The reasons for replacement and the age of failed restorations in general dental practice. Acta Odontol Scand, 1997; 55:58-63.
10. Beyth N, Domb AJ, Weiss EI. An in vitro quantitative antibacterial analysis of amalgam and composite resins. J Dent. 2007; 35:201-206
11. Satou J, Fukunaga A, Satou N, Shintani H, Okuda K. Streptococcal adherence on various restorative materials. J Dent Res.1988; 67:588-591.
12. Willershausen B, Kottgen C, Ernst CP. The influence of restoration materials on marginal gingival. Eur j Med Res.2001; 29; 6(10): 433-9.
13. Teughles W, Van Assche N, Sliepen I. Quirynen M. Effect of material characteristics and/or surface topography on biofilm development. Clin Oral Imp Res. 2006; 17(supl. 2):68–81.
14. Wassell RW, McCabe JF, Walls AW. Wear characteristics in a two-body wear test. Dent Mater. 1994; 10:269-274.
15. Park JW, Song CW, Jung JH, Ahn SJ, Ferracane JL. The effects of surface roughness of composite resin on biofilm formation of Streptococcus mutans in the presence of saliva. Oper Dent 2012; 37:532-539.
16. Van Meerbeek B, Vargas M, Inoue S, Yoshida Y, Perdigão J, Lambrechts P, Vanherle G. Microscopy investigations. Techniques, results, limitations. Am J Dent 2000; 13:3D-18D.
17. Ahn SJ, Lim BS, Lee SJ. Surface characteristics of orthodontic adhesives and effects on streptococcal adhesion. Am J Orthod Dentofacial Orthop. 2010; 137:489-495.
18. Sakaguchi, R.; Powers, J. Craig's Restorative Dental Materials, 13th ed.; Elsevier: Philadelphia, PA, USA, 2012.
19. Øilo M, Bakken V. Biofilm and Dental Biomaterials, Materials 2015; 8: 2887-2900; doi:10.3390/ma8062887.
20. Glantz. P.O. On wettability and adhesiveness, Odont Revy. 1969; 20 (suppl. 17), 5-124.
21. Quirynen M, Marechal M, Busscher HJ, Weerkamp AH, Darius PL, van Steenberghe D. The influence of surface free energy and surface roughness on early plaque formation. An in vivo study in man. J Clin Periodontol. 1990 Mar; 17(3):138-44.
22. Decker EM. The ability of direct fluorescence- based two colour assays to detect different physiological states of oral streptococci. Lett Appl Microbiol. 2001; 33:188-192.
23. Quirynen M, Bollen CM. The influence of surface roughness and surface-free energy on supra- and subgingival plaque formation in man. A review of the literature. J Clin Periodontol 1995; 22:1-14.
24. Takatsuka T., Konishi N., Nakabo S., Hashimoto T Torii Y., Yoshiyama M. Adhesion in vitro of oral streptococci to porcelain, composite resin, cement and human enamel. Dent Mater J 2000; 19:363-372.
25. Grivet M., Morrier JJ., Benay G., Barsotti O. Effect of hidrophobicity on in vitro streptococcal adhesion to dental alloys. J Materials Sci: Mater Med 2000; 11:637-642.
26. Tanner J., Robinson C., Söderling E., Vallittu P. Early plaque formation on fibre-reinforced composites in vivo. Clin Oral Invest. 2005; 9:154–160. English.
27. B. Yuzugullu, C. Celik, TB Ozcelik, S Erkut, P Yurdakul, Y Ocal, B Sener. The effect of different polishing sequences on the adhesion of streptococcus mutans to feldspatic porcelain. The Journal of Adhesion, 2016; 92(12):939-49.
28. Sardin Morriier j-j, Benay G, Barsoti O. In vitro streptococcal adherence on prosthetic and implant materials. Interactions with psychochemical surface properties. J Oral Rehab. 2004; 31:140-148.
29. Bollen CM, Papaioannou W, Van Eldere J, Schepers E, Quirynen M,Van Steenberghe D. The influence of abutment surface roughness on plaque accumulation and periimplant mucositis. Clin Oral Impl Res. 1996; 7: 201-211.

30. Rosentritt M, Hahnel S, Gröger G, Mühlfriedel B, Bürgers R, Handel G. Adhesion of Streptococcus mutans to various dental materials in a laminar flow chamber system. J Biomed Mater Res B Appl Biomater. 2008 Jul; 86(1):36-44.
31. Jalalian E, Mostofi ShN, Shafiee E, Nourizadeh A, Nargesi RA, Ayremlou S. Adhesion of Streptococcus mutans to Zirconia, Titanium Alloy and some other Restorative Materials: "An in-vitro Study". Advances in Bioscience and Clinical Medicine. 2015; 3(2):13-20.
32. Pei Yu, Chuanyong Wang, Jinglin Zhou, Li Jiang, Jing Xue, and Wei Li. Influence of Surface Properties on Adhesion Forces and Attachment of Streptococcus mutans to Zirconia In Vitro. BioMed Research International. Volume 2016 (2016), Article ID 8901253, 10 pages.
33. Júlio C. M. Souza, Raquel R. C. Mota, Mariane B. Sordi, Bernardo B. Passoni, Cesar A. M. Benfatti, Ricardo S. Magini. Biofilm Formation on Different Materials Used in Oral Rehabilitation. Brazilian Dental Journal (2016) 27(2): 141-147.
34. E. Jalalian, GH. Mofrad, M. Rahbar, A. Mohseni, M. Mohebbi. In Vitro Adhesion of Streptococcus Mutans to Polished IPS e.max and Feldspathic Porcelain. Journal of Islamic Dental Association of IRAN (JIDAI) Autumn 2015; 27, (4).

UDC: 616,311,4–006.326

# LIPOMA ON THE FLOOR OF THE MOUTH – A RARE CASE WITH UNUSUAL LOCALIZATION

# ЛИПОМ НА ПОДОТ НА УСТАТА – РЕДОК СЛУЧАЈ СО НЕОБИЧНА ЛОКАЛИЗАЦИЈА

**Kirkov A.[1]*, Koneski F.[1], Benedetti A.[1], Iliev A.[1], Stamatoski A.[1]**

University Clinic for Maxillofacial Surgery in Skopje, Ss. Cyril and Methodius University in Skopje, Faculty of Dentistry, Department of Maxillofacial Surgery

## Abstract

Oral lipomas as benign tumors are a rare entity in the oral cavity. However, certain locations that contain adipose tissue may be a place of origin for lipomas. This report presents a case of lipoma on the floor of the mouth in a 74-year old male patient, with evident swelling, difficulties in speech, eating, and talking. Clinical examination revealed tender oval yellowish mass located in the right sublingual space. Infection or cyst as a possible differential diagnosis were ruled out, besides the specific localization. Under local anesthesia, the mass with dimensions of 3.3×2.3×1 cm was totally extirpated from the right sublingual space and histopathological evaluation revealed a clear picture of lipoma, consisting of mature adipose cells. The follow up of six months did not reveal any recurrence or other morbidity events. It is of major importance to take into consideration the vital anatomic structures in order to avoid injuries to the salivary duct or lingual nerve during the surgical technique. A thorough differential diagnosis should be made before making the decision for treatment, especially because previously there were presented cases of malignant transformation of a buccal lipoma into liposarcoma. **Keywords:** oral lipoma, floor of the mouth, differential diagnosis. The authors declare that there is no conflict of interest in regard to this study. The study has not received any funding.

## Апстракт

Оралните липоми како бенигни тумори се редок ентитет во оралната празнина. Сепак, тие може да се појават на одредени локализации кои содржат масно ткиво. Во овој приказ се презентира случај на липом на подот на устата кај 74-годишен маж, со евидентен оток, потешкотии во говорот, исхраната и зборувањето. Клиничкиот преглед покажа тврда овална жолтеникава маса локализирана во десниот сублингвален простор. Инфекција и циста како можна диференцијална дијагноза беа исфрлени, и покрај специфичната локализација. Во локална анестезија, масата со димензии 3,3×2,3×1 цм беше целосно екстирпирана од десниот сублингвален простор, а хистопатолошката анализа покажа јасна слика на липом, кој се состоеше од зрели масни клетки. Шест месеци подоцна, кај пациентот не беше забележан рецидив, ниту други знаци на морбидитет. Од големо значење е при хируршката техника да се земат предвид важните анатомски структури со цел да се избегне повреда на изводните канали на плунковните жлезди и на јазичниот нерв. Пред да се донесе одлуката за третман, потребно е детално да се проучи диференцијалната дијагноза, особено поради фактот што претходно се пријавени случаи на малигна трансформација на букален липом во липосарком. **Клучни зборови:** орален липом, под на устата, диференцијална дијагноза

## Introduction

Lipomas are benign tumors with mesenchymal origin, consisting of mature adipocytes and are relatively common finding in the body. The head and neck region is affected by around 15-20% of all lipomas[1], but their occurrence in the oral cavity is rare, with a rate of 1:50,000 patients, which counts for 1-4.4% of all benign tumors in the mouth[2]. Buccal mucosa is the most affected site, because of the present fat tissue. However, lipomas may also develop on the lips, tongue, palate and rarely the floor of the mouth[3, 4]. The etiology of oral lipomas is not elucidated, but several hypotheses exist, including local chronic trauma as most speculated. Most of the studies have shown no gender preferences, although there are reports for slightly higher prevalence in males[5]. Generally, lipomas are growths resulting from developmental defects, but in the oral cavity they are most commonly associated with trauma, and they are more prevalent in adults older than 40. Clinically, lipomas are painless, well defined, slow growing submucosal or superficial lesions, encapsulated with thin fibrous

capsule and covered with intact mucosa[3, 6]. In addition, some reports show intramuscular infiltrative types[7, 8]. Literature reviews show that the mean diameter of lipomas in the oral cavity is 22 mm[9]. Although they do not pose a serious health threat, lipomas may cause aesthetic and functional disturbances, especially if located in a visible area or if they interfere with the function of mastication[10, 11].

However, the clinical diagnosis of lipoma on the floor of the mouth may be challenging, because of the unusual localization, nonspecific appearance, small incidence and other more likely differential diagnosis, such as ranulas.

This paper reports a case of lipoma on the floor of the mouth, preliminary giving a clinical appearance of ranula.

## Case presentation

A 74-year old man was referred to the University Clinic for Maxillofacial Surgery in Skopje, complaining on painless growing mass on the sublingual area on the right side intraorally, which persisted for several weeks. Although it did not cause any pain, the mass was a reason for discomfort and interfered with the function of eating, speech, and swallowing. The anamnesis revealed a history of high blood pressure, controlled with appropriate antihypertensive medication, therefore not contributing to any specific issues.

The clinical intraoral examination revealed a relatively firm mass in the right sublingual area, ovoid in shape, extending from the central incisor on the second molar (Figure 1). The overlying mucosa did not show any signs of inflammation, neither a change in color or consistency. The mass was not fixed to the mucosa and it was not fluctuant. No ulcers or exophytic lesions were noted. The mass dimensions were roughly 4x2 cm, which caused discomfort during tongue movements and hence, it interfered with the function of speech and eating. There was no history of pus discharge, bleeding, pain, or huge variations in the size associated with taking food or drinks. Upon manual extraoral pressure in the submandibular area, the swelling bulged and was more evident in the mouth. However, palpation revealed that the mass was limited to the area above the mylohyoid muscle, indicating that it did not spread from the sublingual space. No significant cervical lymphadenopathy was noted. Blood analysis showed normal range of parameters important for ruling out infection or other disturbances that may be associated with the mass.

Regarding the dental status, the patient was partially edentulous, having fixed metal-composite bridge constructions on the both sites of the jaws, which lasted for more than 10 years. The remaining teeth were characterized with advanced attrition. There were no detected changes on the oral mucosa in terms of ulcers, change in color or other possible growths.

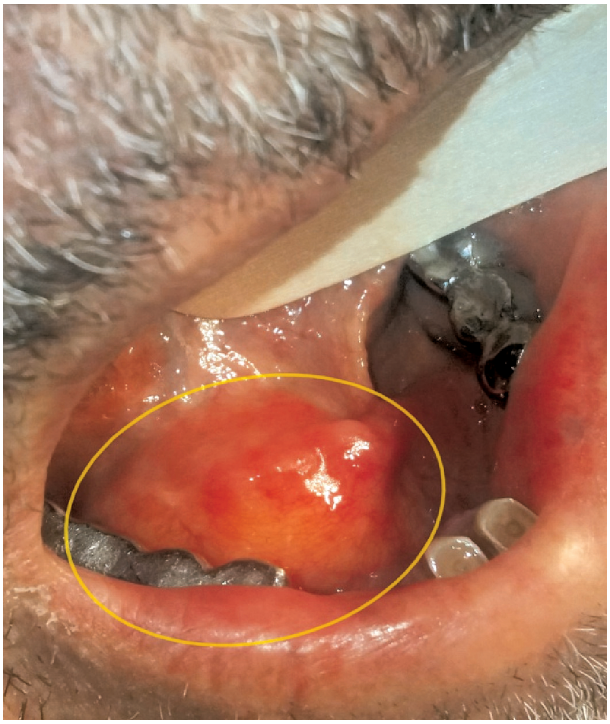

***Figure 1.*** *Intraoral appearance of a mass in the right sublingual space. Note the metal-composite bridge constructions on both sides of the lower jaw.*

Taking into consideration the nature of the sublingual mass, its tendency not to regress, not causing any specific symptoms, except the discomfort and difficulties to speak, eat, and swallow due to the limited movement of the tongue, a decision for total extirpation of the mass was made. The procedure was performed under local anesthesia. 2 ml of solution of 2% Lidocaine hydrochloride (40 mg) and adrenaline (0,025 mg) were injected in the area of innervations of the lingual nerve, in the submucosal zone around 1 cm below the second lower molar. Additionally, a small amount of anesthetic solution was injected around the mass that was subject to extirpation. Incision was made over the highest point of the growth, taking into consideration the anatomic path of the Wharton's duct of submandibular gland and maintaining at least 1 cm distance from it. After the initial incision, a dissection of the remaining overlying mucosa was done laterally, to the extent where the mass was located (Figure 2). Then, a blunt dissection of the adjacent mucosa, connective tissue and in part of the muscle fibers was performed, taking care not to cause any injury

on the lining of the mass. The mass itself was yellowish, with smooth surface, covered with a very thin capsule. Having it carefully clumped on the periphery on its anterior aspect, with further combination of blunt dissection techniques, the mass was completely extirpated (Figures 3 and 4). The remaining surgical site was carefully examined for possible remains of the mass, and satisfactory hemostasis was achieved (Figure 5). The wound was sutured by primary intention, with non-absorbable 3-0 silk sutures. Finally, salivation was checked by manual stimulation of the submandibular gland, to make sure the Wharton's duct was intact. Once the anesthetic period of the injected solution was over, the patient was asked for any changes in sensitivity of the tongue and mucosa in the sublingual area. He confirmed no changes, approving that there was no damage to the lingual nerve.

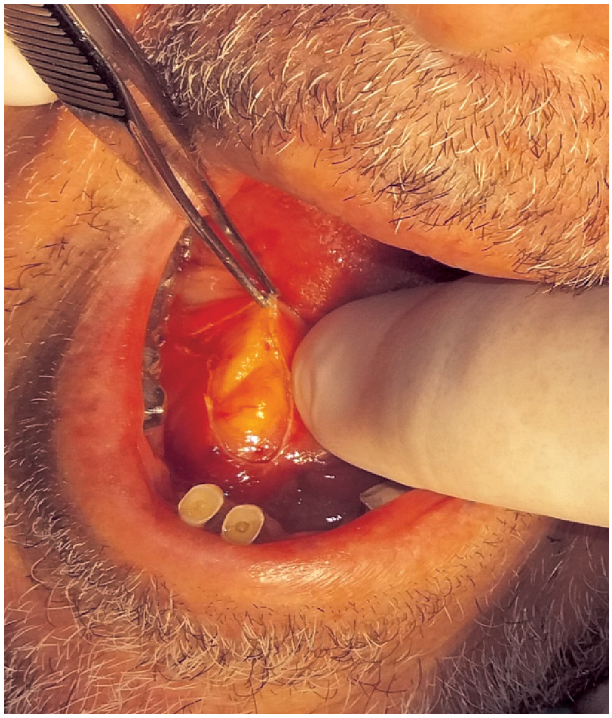

***Figure 2.*** *After the initial incision was made, a yellowish lobulated mass just beneath the mucosa was noted.*

A specimen with dimensions 3.3×2.3×1 cm (Figure 6) was sent for histopathological evaluation and was prepared with Hematoxylin and Eosin staining. Microscopic analysis revealed mature adipose tissue lobules, comprised of benign adipocytes, separated by the connective tissue springs and incorporated with capillaries. On the surface, a thin capsule was noted. Hence, a histopathological diagnosis of lipoma was set.

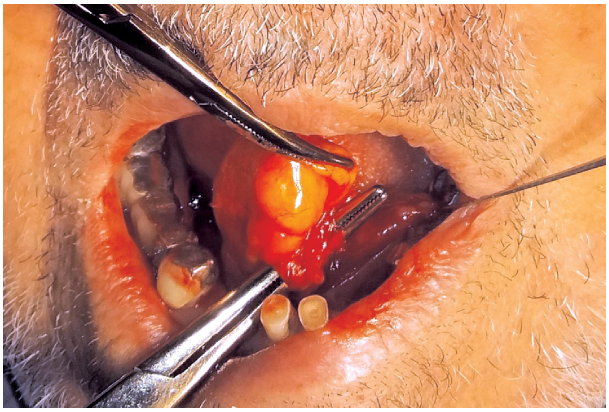

***Figure 3.*** *Blunt dissection was performed to detach the tumor from the surrounding mucosa, connective tissue and in part the muscle fibers, in order to avoid damage of the Wharton's duct and the lingual nerve.*

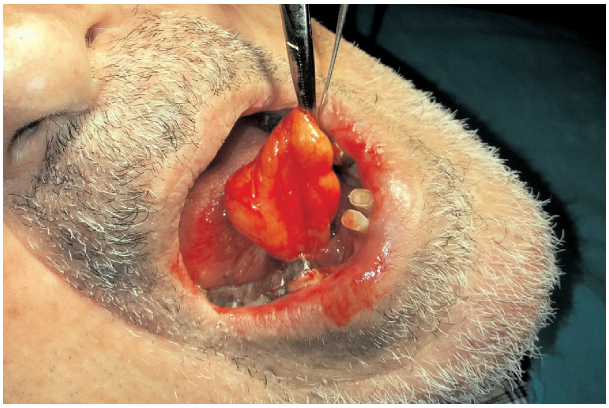

***Figure 4.*** *Final step of tumor extirpation.*

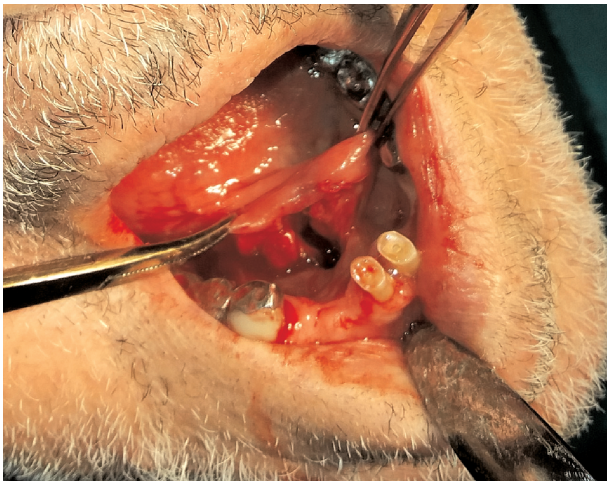

***Figure 5.*** *Surgical site after extirpation of the tumor and after achieving satisfactory hemostasis.*

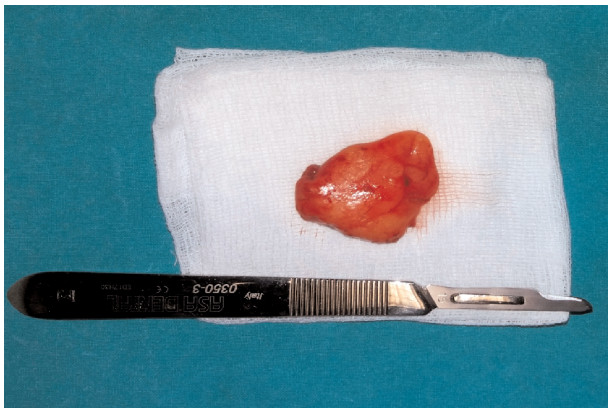

***Figure 6.*** *The mass sent for histopathological evaluation had the following dimensions: 3.3×2.3×1 cm (tumor placed next to a surgical instrument for comparison).*

Sutures were removed one week after the surgery. The follow-up of six months did not reveal any signs of morbidity associated with the surgical procedure, nor any signs of recurrence.

## Discussion and conclusion

Although oral lipomas are a rare entity, they may occur at any site where adipose tissue is present. The most common oral location is the inner side of the cheek. However, this report presents a case of a lipoma on the floor of the mouth, extending through the right sublingual space. They may occur in the lips, tongue, gingival, and palate. Lipomas are benign tumors which may remain undiagnosed until they reach certain dimensions, like in our case. Because the sublingual space may be more commonly associated with a number of pathological changes, some of which are very similar to each other, a thorough history and detailed checkup should be performed before deciding on the treatment. In terms of differential diagnosis, when such a mass is present in this area, the general dentist or surgeon should think of several possible conditions. Retention or extravasation cyst, known as ranula or mucocele, which in this zone presents as a swelling limited to the above-mylohyoid muscle space, derived from an obstruction in the ducts of the sublingual gland or the gland itself, should be ruled out. Other types of cysts may also be taken into consideration, especially developmental dermoid or epidermoid cysts. This diagnosis was ruled out because the mass in our case was not connected with the overlying mucosa, but was slightly mobile. Infection spread in the sublingual space, including abscesses may also be a possibility, but in our case, there were no signs of infection – there was no redness, pain, increased temperature, nor pus discharge or signs of damage of the mucosa. Finally, a possible tumor lesions, including benign or malignant ones, shall be taken into consideration.

A previous review shows that the mean age of patients with oral lipomas is 50.2 years[5], which is consistent with our case where the patient was over the age of 70. However, there are several reports when younger patients were diagnosed with this kind of benign tumors[5, 12]. Literature also shows that there are no predilections regarding the gender, despite the fact that lipomas are slightly more common in men. The exact reason and mechanism of occurrence of intraoral lipomas are not fully elucidated, but several hypotheses include chronic irritation or trauma, poor dental and oral status, hereditary and developmental disturbances[7].

Clinical characteristics may play an important role in deciding whether the mass will be removed or monitored. Because there were no signs of regression, the mass in our case was completely removed. The final diagnosis was set by the histopathological evaluation, which usually reveals adipocytes larger than normal, organized in lobules and encapsulated, with low vascularization. As in the case in this report, atypical changes are not seen by rule. However, a case of transformation of a lipoma on the buccal mucosa which was initially diagnosed with biopsy, into liposarcoma[13]. This possibility underlines the necessity of careful examination or deciding to remove any lesion that does not show signs of regression. In exceptional cases, imaging may be used for proper evaluation of the nature and extent of the mass. MRI may play a particularly important role since lipomas are well detected due to the high signal intensity. Sialolipomas appear on both T1- and T2-weighted images on MRI, hence being another indication for this imaging tool[14, 15]. However, different diagnostic imaging modalities such as MRI, CT, or ultrasound may not be completely accurate in distinguishing lipomas from well-differentiated liposarcomas[16]. Hence, the microscopic analysis is the gold standard for setting a final diagnosis. Histopathologically, lipomas are differentiated from liposarcoma if there are no signs of lipoblastic proliferation, adipocytes with various sizes, increased nucleous to cytoplasm ratio, signs of atypia or hyperchromatia, or bizarre stromal cells in fibrous septa, between adipocytes or in vessel walls[17, 18].

Interestingly, lipomas differ metabolically from normal fat cells even though they are histologically similar. It has been shown that the fat of lipoma is not used for energy production during starvation periods, as it happens with the normal adipose tissue[19].

Not only preliminary diagnosis may be challenging, but also certain considerations and precautions should be

taken into account when planning and performing the surgical procedure on the floor of the mouth. Important anatomical structures should be kept in mind and careful techniques should be implemented. These include the duct of the submandibular and sublingual gland and the lingual nerve, which may pass this area very superficially, posing a risk of damage. Therefore, the incision should be planned at least 1 cm away from the projected path of the duct, while sharp dissection should be avoided in order to prevent nerve damage, especially in cases where possible muscle extension of the mass is present.

## Reference


1. Trandafir D, Gogalniceanu D, Trandafir V, Caruntu ID. Lipomas of the oral cavity – a retrospective study. Rev Med Chir Soc Med Nat Iasi. 2007; 111:754–758.
2. De Visscher JG. Lipomas and fibrolipomas of the oral cavity. J Oral Maxillofac Surg 1982; 10(3):177–181.
3. Fregnani E.R., Pires F.R., Falzoni R., Lopes M.A., Vargas P.A. Lipomas of the oral cavity: clinical findings, histological classification and proliferative activity of 46 cases. Int J Oral and Maxillofac Surg, 2003; 32(1): 49–53.
4. Furlong M.A., Fanburg-Smith J.C., Childers E.L.B. Lipoma of the oral and maxillofacial region: site and subclassification of 125 cases. Oral Surg, Oral Med, Oral Path, Oral Rad Endod, 2004; 98(4): 441–450.
5. Bandéca MC, de Pádua JM, Nadalin MR, Ozório JE, Silva-Sousa YT, da Cruz Perez DE. Oral soft tissue lipomas: a case series. J Can Dent Assoc. 2007; 73(5):431-434.
6. Weiss SW, Goldblum JR, editors. Benign lipomatous tumors. In: Enzinger and Weiss's soft tissue tumors. 4th ed. St. Louis: Mosby; 2001. pp. 571–639
7. Tan MS, Singh B. Difficulties in diagnosing lesions in the floor of the mouth – report of two rare cases. Ann Acad Med Singapore. 2004; 33 suppl:72-76.
8. Garavaglia J., Gnepp D.R. Intramuscular (infiltrating) lipoma of the tongue. Oral Surg Oral Med Oral Path, 1987; 63(3):348-350.
9. Manor E., Sion-Vardy N., Joshua B.Z., Bodner L. Oral lipoma: analysis of 58 new cases and review of the literature. Ann Diagn Pathol, 2011; 15(4):257–261.
10. Annibali S, Cristalli MP, Monaca GL, Giannone N, Testa NF, Russo LL, et al. Lipoma in the Soft Tissues of the Floor of the Mouth: A Case Report. The Open Otorhinolaryng J, 2009; 3:11-13.
11. Hosein AT, Razavi SM, Khabazian A. Lipoma in Oral Mucosa: Two Case Reports. Dent Res J. 2010; 7:41-3.
12. Punjabi VH, Patel S, Pathak J, Swain N. Fibrolipoma of Lip in a Young Individual: A Rare Presentation. J Cont Dent, 2017; 7(3):181-184.
13. Yamada K., Dohara Y., Nagata M., Kawashima K., Yamashita S. A case of liposarcoma of the cheek. Jpn J Clin Onc, 1979; 9(1):123–129
14. Bancroft L.W., Kransdorf M.J., Peterson J.J. O'Connor M.I. Benign fatty tumors: classification, clinical course, imaging appearance, and treatment. Skeletal Radiol, 2006; 35(10):719–733.
15. Taira Y., Yasukawa K., Yamamori I., Iino M. Oral lipoma extending superiorly from mandibular gingivobuccal fold to gingiva: a case report and analysis of 207 patients with oral lipoma in Japan. Odontol, 2012; 100(1):104–108.
16. Chikui T., Yonetsu K., Yoshiura K., Miwa K., Kanda S., Ozeki S., Shinohara M. Imaging findings of lipomas in the orofacial region with CT, US, and MRI. Oral Surg Oral Med Oral Path Oral Radiol Endodon, 1997; 84(1): 88–95.
17. Orita Y., Nishizaki K., Ogawara T., Yamadori I., Yorizane S., Akagi H, Masuda Y. Liposarcoma of the tongue: case report and literature update. Ann Oto Rhyno Laryn, 2000; 109(7):683-686.
18. Nascimento A.F., McMenamin M.E., Fletcher C.D. Liposarcomas/atypical lipomatous tumors of the oral cavity: a clinicopathologic study of 23 cases. Ann Diagn Pathol, 2002; 6(2):83-93.
19. Kumaraswamy SV, Madan N, Keerthi R, Singh Shakti. Lipomas of oral cavity: case reports with review of literature. J Maxillofac Oral Surg, 2008; 4:394–397.

UDC: 616.833.15–009.7

# THERAPEUTICAL PROCEDURES IN PATIENTS WITH TRIGEMINAL NEURALGIA - LITERATURE REVIEW

# ТЕРАПЕВТСКИ ПРОЦЕДУРИ КАЈ ПАЦИЕНТИ СО ТРИГЕМИНАЛНА НЕУРАЛГИЈА - РЕВИЈАЛЕН ТРУД

**Shushak Z.[1], Popovski V.[2], Mitikj J.[3], Veljanovski D.[4]**

[1]PhD Candidate, DDM, DDS - Private health institution ,,Dentoria-1 Specialist practice for oral surgery and implantology" Ohrid, Republic of North Macedonia, [2]PhD, MSD, MFS, DDM, MD - Clinic for maxillofacial surgery, University „Ss. Cyril and Methodius" Skopje, Republic of North Macedonia, [3]PhD Candidate, DDM, MSD, trainee in periodontology - Private health institution „Molar Dent" Skopje, Republic of North Macedonia, [4]PhD Candidate, DDM, DDS, - Private health institution „Optimum dental practice", Skopje, Republic of North Macedonia

## Abstract

Trigeminal neuralgia (TN) is the most common pathological condition of the cranial nerves and the most common cause of pain in the orofacial region. More and more literary data indicate that a in significant number of patients, TN is caused by compression on the root of the trigeminal nerve, close to its entry into the pons in the aberrant arterial or venous loop leading to pathohistological changes in the nerve while in most of the same, the etiological factor for the pathology of TN remains a mystery. Numerous medical and surgical procedures are available, usually without a number of randomized clinical trials and studies of placebo-controlled groups. As a result, most patients experience refractory pain, which has a significant effect on reducing their quality of life. It is still unclear how available treatment methods can be best used. Medical treatment consists of anticonvulsant drugs, muscle relaxants and neuroleptic agents, alternative treatments with botulinum toxin. In patients resistant to drug therapy, promising i.e. recommended surgical procedures include microvascular decompression, gamma-knife surgery, and percutaneous techniques of the Gasser ganglion. In this paper we will present a systematic review of the literature and investigate the outcome of different types of therapeutic procedures in patients with trigeminal neuralgia. **Keywords:** Trigeminal neuralgia, anticonvulsant drugs, pain.

## Апстракт

Тригеминалната неуралгија (ТН) е најчестата патолошка состојба на кранијалните нерви и најчест причинител на болка во орофацијалната регија. Се повеќе литературни податоци укажуваат дека кај значителен број на пациенти, ТН е предизвикана од компресија врз коренот на тригеминалниот нерв, близу до неговиот влез во понсот во аберантната артериска или венска јамка која доведува до патохистолошки промени во нервот додека кај поголемиот број од истите, етиолошкиот фактор за патологијата на ТН останува мистерија. Достапни се бројни медикаментозни и хируршки процедури, обично без поголем број рандомизирани клинички испитувања и студии контролирани со плацебо групи. Како резултат на тоа, повеќето пациенти доживуваат рефракторна болка, која има значително влијание врз намалување на квалитетот на живот. Сè уште останува неизвесно како е најдобро да се употребат достапните методи на терапија. Медикаментозниот третман се состои од антиконвулзивни лекови, мускулни релаксанти и невролептични агенси, алтернативни третмани со ботулински токсин. Кај пациентите отпорни на медикаментозна терапија, перспективни т.е. препорачливи се хируршки процедури, микроваскуларна декомпресија, гама-нож хирургија и перкутани техники на гасеровиот ганглион. Во овој труд ќе направиме систематски преглед на литературата и ќе го истражиме исходот од различните видови терапевтски процедури кај пациенти со тригеминална неуралгија. **Клучни зборови:** Тригеминална неуралгија, антиконвулзивни лекови, болка.

## Introduction

Trigeminal neuralgia (TH) is defined by the International Classification of Headache Disorders as „*Disorder characterized by recurrent, unilateral short electrical pain in the form of shock, with sudden onset and termination, limited to the distribution of one or more trigeminal nerve divisions, and caused by insignificant stimuli*."[1] The new classification sets 3 etiological categories of: idiopathic TN (without neurovascular contact or neurovascular contact without morphological changes of the trigeminal root), classic TN (due to neurovascular compression with morphological changes of the trigeminal root) and secondary TN (due to a neurological disease such as tumor of the cerebellopontine angle or multiple sclerosis). Also, 2 phenotypes are classified: clear paroxysmal TN (only with paroxysmal pain) and TN with continuous constant pain[2]. In early descriptions, the disorder was called tic douloureux[3]. The incidence is estimated at 4 to 13 people per 100,000 per year.

The pain is limited to an area innervated by one or more branches of the trigeminal nerve. In 60% of cases it is only one branch, maxillary (V2) or mandibular (V3), while in 35% of cases both branches are involved. On the other hand, the ophthalmic branch (V1) is rarely included (in less than 4% of patients)[4].

TN symptomatology is very characteristic, patients report intense stabbing, severe pain localized on the face, nose, teeth, or jaws caused by provocation at the trigger points or by spontaneous (sudden) onset. Depending on whether it is primary or secondary, TN may vary based on the characteristics of the disease. In patients with neurological deficits, extratrigeminal symptoms, bilateral occurrence, and appearance in young individuals are very characteristic[5].

Accurate diagnosis of the orofacial pain is the first step in successful disease management. This is primarily based on the patient's history, because there are still no definite (specific) paraclinical-diagnostic or therapeutic-clinical tests. In 1936, Riley's classic pain analysis highlighted 11 essential issues, which should be included in the history of pain. These aspects apply today, too[6]. As part of diagnostic procedures, it is recommended to use magnetic resonance imaging (MR), because a lack of clinical features may preclude secondary TN. If MR is unavailable or contraindicated, trigeminal reflexes shall be used to distinguish between primary and secondary TN[7]. Differential diagnostics should be distinguished: maxillary sinusitis, temporomandibular disorders, postherpetic neuralgia, posttraumatic trigeminal neuralgia, glossopyrosis, glosopharyngeal neuralgia, idiopathic facial pain, SUNA and SUNCT[8]. There is also a serious lack of evidence for diagnostic criteria[9].

The purpose of this review paper is to systematize the evidence obtained from previously published articles on the outcome of various therapeutic procedures in patients diagnosed with trigeminal neuralgia.

There are a number of conservative and surgical therapeutic procedures for TN. The general recommendation is to start drug therapy and consider surgical treatment in patients who are refractory to it[10]. There is still a lack of sufficient number of studies that directly compare medical and surgical treatment. Medical therapy begins as monotherapy, followed by a combination therapy with various drugs that can be used when the effectiveness of monotherapy is low[11,12]. Antiepileptic drugs **Carbazepine** and **Oxcarbazepine** are the first line of treatment in TN[13]. **Lamotrigine** and **Baclofen** are considered a second line of treatment, while **Topiramate, Levetiracetam, Gabapentin, Pregabalin** and **botulinum toxin type A** are alternative treatments[14]. There are several surgical treatment options available to **TN** patients and it is important to choose the most appropriate surgery for the patient[15]. **Microvascular decompression** is a non-destructive procedure and is considered the golden standard of surgical therapy[16]. The other widely used technique is the **gamma knife**[17]. Of the destructive techniques, the most widely used are **the percutaneous techniques of the Gasser ganglion**, which have the advantage of selectively targeting the affected trigeminal divisions[18].

## Materials and methods

In the paper, we did research and meta-analysis of all available literary data using a wide range of data up to date (from 1966 to 2020) for all studies related to **TN** treatment. Research was conducted electronically using studies published in English. The databases used in the selection of studies are: **PubMed/MEDLINE, Embase and Cohrane Library.**

## Results and discussion

This review paper provides an overview of the results, measures, and prognosis of the end result by using the TN medication and surgical treatments until today. Treatments are performed by experienced therapists from different countries around the world and highlight the variability of the choice of measures used to achieve a positive result.

### *Conservative procedures*

Regardless of the classification system, TN treatment always begins with drug therapy. Of the drugs currently used for treatment, all were originally developed for other indications (antiepileptics, myorelaxants, opioids, etc.). In addition, only a small proportion have been examined in large controlled studies, and many of the studies so far have methodological inconsistencies[19].

### *Anticonvulsant drugs*

**Carbamazepine** acts inhibitory on sodium channels and reduces the excitability of nerve membranes. It also highlights gamma aminobuteric acid gamma receptors GABA composed of alpha 1, beta 2, and gamma 2 under units relevant to its role in the reduced transmission of neuropathic pain. **Oxcarbazepine** is a keto analogue of carbamazepine. With long-term treatment, carbamazepine (200–1200 mg/daily) or oxcarbazepine (300–1800mg/daily) remain the most effective drugs in the early stages of TN[20]. Literature also contains described situations that require even higher doses. If

these medications become ineffective or result in poor tolerance, then other medications should be considered. From systematic examinations[21] and randomized controlled trials[22-24], carbamazepine proves more effective than placebo groups, but more patients withdraw from use due to side effects. The most common side effects include somnolence, dizziness, and orthostatic hypotension. Oxcarbazepine has comparable efficiency in regard to cocarbazepine, but greater tolerance[25], except for the risk of hyponatraemia and low drug interaction potential[26, 27]. In the study of Zakrzewska, there is evidence that **Lamotrigine was** used as additional effective therapy[28], while there is little evidence that other anticonvulsant drugs like **clonazepam, gabapentin, pregabalin and valproate** have a beneficial effect[29]. Several review papers have investigated the comparative efficacy and safety of anticonvulsant drugs[30-32]. A paper based on 16 randomized controlled trials compared the effects of carbamazepine and gabapentin. Gabapentin is associated with fewer side effects than carbamazepine and oxcarbazepine[33]. We still have difficulty determining the best treatment for pain relief with minimal side effects. The failure of treatment is usually not due to the ineffectiveness of the medication, but rather to the side effects that cause discontinuation of treatment or reduction of the dose to an ineffective level. Combined treatment (carbamazepine or oxcarbazepine with lamotrigine, baclofen, pregabalin or gabapentin) should be taken into account when carbamazepine or oxcarbazepine may not reach the required dose due to side effects. Each of the drugs has clinical application as monotherapy, although the available evidence are weak[34].

### *Muscle relaxants*

**Baclofen** (60-80 mg/daily), skeletal muscle relaxant is a GABA analogue that activates GABAb receptors and reduces excitatory neurotransmission. Clinical trials have shown that baclofen is effective as monotherapy or in combination with carbamazepine in the treatment of TN[35]. According to the research of Hassan and associates[36], after carbamazepine, baclofen shows the greatest effectiveness in the treatment of TN. Typical side effects include drowsiness, dizziness, fatigue, hypotension.

### *Opioids*

Opioids are drugs used to treat acute and chronic pain. According to numerous studies, it is not recommended as the first line of therapy due to the danger of side effects that would occur during abuse and overdose[38, 39]. In recent years, Fentanyl has been used in the treatment of many chronic types of pain40. Coven, in his research, uses fentanyl in the treatment of TN type 2 as a blockage in the sphenopalatinal ganglion and gets successful results[41]. However, according to Cochrane, in the review papers there is insufficient evidence for the use of opioids in the treatment of neuropathic pain[42].

### *Botulinum toxin A*

**Botulinum toxin** is a neurotoxin produced by the bacterium Clostridium botulinum. It blocks the release of acetylcholine on the neuromuscular synapse. It binds to C-fibers, has an analgesic effect and reduces muscle spasms[43]. In 2002, Micheli and associates[44] published a successful treatment of a patient with hemifacial spasm associated with TN with onabotulinumtoxin, which opens up new possibilities for its use. In the Bohluli study[45], 47% of patients did not require further treatment, nonsteroidal anti-inflammatory drugs were sufficient to relieve pain in 33% of patients, while 20% of patients again required anticonvulsant therapy after toxin administration. Recently, three examinations demonstrated that botulinum toxin could provide a clinically significant benefit in the treatment of TN[46-48]. A recent review by Jiangshan suggests that botulinum toxin is effective and safe in the treatment of TN and peripheral neuropathic pain[49]. The duration of the therapeutic effect and the doses that would be applied for the given pathology are issues that shall be researched in the future in well-thought-out examinations.

## *Surgical procedures*

Patients who have persistent pain or cannot tolerate drug therapy due to side effects are referred for surgical treatment. These procedures have different success rates and risk profiles. Three descriptive studies have been identified that address the issue when TN patients should be offered surgical treatment[20,50,51]. Studies have shown that patients who are refractory to drug therapy may prefer early surgical treatment. A prospective study reported that 65% of patients referred to specialist centers could be managed with drug therapy for 2 years after referral with satisfactory results. 35% of them underwent surgery[52].

### *Microvascular decompression*

**Microvascular decompression** (MVD) is a type of neurosurgery used in the treatment of TN caused by vertebrobasilar compression. Surgery is not without risk, it can cause recurrent facial pain and other side effects (insult, paralysis, paresis, lethal outcome). It was first

introduced by Jannetta[53] and after many years of improvement, MVD has become the most effective of TN surgical treatments .MVD in TN is a surgical treatment that has the least chance of failure, according to a study of 195 patients by Hitchon[54]. However, there are still patients who cannot achieve long-term outcomes. For patients who are not suitable for pure MVD, MVD combined with partial sensory rhizotomy can be considered as an effective alternative[55]. Leakage of vascular structure results in incomplete relief of symptoms after intervention[56]. Endoscopically assisted microsurgery helps to optimize surgical procedures, especially in identifying the overall course of the cranial nerve and avoiding the leakage of vascular structures[57]. The initial success rate of the therapy is usually high, but in 5% of the patients there is little or no pain relief after MVD. 10-30% of patients have recurrent neuralgia on follow-up, with an annual risk of recurrence of 1 to 4%[58,59]. In a recent review and meta-analysis[60], it has been concluded that about three-quarters of patients with TN resistant to drug therapy have been relieved of pain after MVD. Shorter duration of pain, arterial compression, and type 1 Burchiel classification predict a more favorable outcome.

## *Gamma knife*

**Gamma knife radiosurgery** is an increasingly used, minimally invasive treatment option for TN patients who are refractory to drug treatment[61]. The use of radiosurgery in the treatment of TN dates back to Leksell (Sweden, 1950) who performed radio ganglionotomies on the gasserian ganglion[62]. Later, he began using the Gamma knife used in the form of multiple focal rays from cobalt-60 sources. Several retrospective and several prospective studies have reported good short-term and medium-term safety and effectiveness of the gamma knife[63,64,65]. The long-term outcome has not yet been well documented, as evidenced by a historical cohort study[66]. According to one review paper, the gamma knife is an effective single and multiple treatment option. Their cumulative research suggests that patients treated once show the same control of facial pain compared to patients who have been treated multiple times. The second group of patients is more likely to experience numbness and other sensory changes in the face compared to the first group[67].

## *Percutaneous techniques of the gasserian ganglion*

Professional procedures performed at the level of the ganglion, trigeminal radiofrequency thermocoagulation (RFT), percutaneous balloon compression (PBC), percutaneous glycerol rhizolysis of the gastric ganglion (PRGR) are more effective than peripheral procedures, but no approach can guarantee long-term pain relief. The first two procedures are non-destructive and can cause sensory loss and distension[68].

RFT of the Gasser ganglion is a widely accepted, minimally invasive technique for the treatment of TN. Efforts are still being made to improve the details of each procedure, reduce perioperative pain, reduce surgical complications, and enhance analgesic efficacy[69]. Understanding and mastering surgical details varies between different hospitals[70]. Two RFT approaches are most commonly used: conventional radio frequency CRF and pulsed radio frequency PRF. According to previous examinations, CRF is the preferred treatment option (higher pain reduction rate, less frequent recurrent pain)[71]. New technologies, such as 3D printed lead board that allow for more precise interventions, have been proven in recent studies[72].

In 1983, Mullan and Lichtor introduced PBC by modifying the technique of nerve compression while performing a craniotomy. Initial studies have shown that PBC is successful, safe, with a small number of relapses, making it particularly popular with elderly patients[73,74]. In one review[75], the authors presented data from the largest cohort of patients with the longest follow-up for this procedure. The PBC procedure has the advantage of being a quick and simple procedure that can be performed in a short period of general anesthesia without discomfort to the patient. This makes it an attractive choice in TN treatment. In their study, Bergenheim and associates[76] presented the complications of the procedure (cardiovascular stress, local haemorrhage, postoperative sensory disturbance, masseur muscle weakness, affections, and transient diplopia). Measures to minimize side effects have been proposed.

In 1981, Hakanson introduced the PRGR technique in which he uses anhydrated glycerol and has since found use as a routine procedure[77]. Several studies have shown that PRGR is a simple, safe, relatively inexpensive method of treating TN[78,79]. The number of side effects and complications during the intervention is relatively small. A second application may be effective in recurrent pain and in patients who do not respond to the first application[80]. Predictive success factors include patients without persistent facial pain, patients with nomadic facial pain during glycerol injection, and patients with new trigeminal defects after PRGR[81]. One of the alternatives to the new PRGR technique is neuronavigation to place the needle in the oval opening and inject glycerol under sedation. This technique is the simplest and safest if the surgeon has previous experience in it[82].

## Conclusion

Conservative (drugs) therapy with antiepileptic drugs, an individualized dose of CARBAMAZEPIN is still the gold standard in the treatment of TN worldwide. A combined therapy, guided by the literature base, did not make a significant difference in the fight against pain compared to monotherapy, and the use of alternative therapies in the form of botulinum toxin treatment requires further studies to determine the exact dose and the time period of drug action. Surgical procedures and studies that have been developed are a good basis for progress in their development using new 3D technology and the introduction of new recommendations and work protocols that would drastically reduce the percentage of risks and complications and make them more accessible to patients.

## Reference


1. Headache Classification Committee of the International Headache Society. The International Classification of Headache Disorders, 3rd edition (beta version). Cephalalgia, 33 (2013), pp. 629-808.
2. Cruccu G, Finnerup NB, Jensen TS, et al. Trigeminal neuralgia: new classification and diagnostic grading for practice and research. Neurology 2016; 87: 220–228.
3. Cole CD, Liu JK and Apfelbaum RI. Historical perspectives on the diagnosis and treatment of trigeminal neuralgia. Neurosurg Focus 2005; 18: 1–15.
4. Eder G, Juliana G. C, and Gerald W. Z. Trigeminal neuralgia: An overview from pathophysiology to pharmacological treatments. Molecular Pain 2020; 16: 1–18.
5. Stine M, Giulia D S, Lars B, Giorgio C. Trigeminal neuralgia – diagnosis and treatment. Cephalalgia 2017, Vol. 37(7) 648–657.
6. Anne M Hegarty, Joanna M Zakrzewska. Differential Diagnosis for Orofacial Pain, Including Sinusitis, TMD, Trigeminal Neuralgia. Dent Update 2011; 38: 396–408
7. Bendtsen, L., Zakrzewska, J.M., Abbott, J., Braschinsky, M., Di Stefano, G., Donnet, A., Eide, P.K., Leal, P.R.L., Maarbjerg, S., May, A., et al. (2019). European Academy of Neurology guideline on trigeminal neuralgia. European Journal of Neurology 26, 831–849.
8. Zakrzewska, J.M., 2013. Differential diagnosis of facial pain and guidelines for management. British Journal of Anaesthesia 111, 95–104. doi:10.1093/bja/aet125.
9. Zebenholzer, K., Wober, C., Vigl, M., Wessely, P. and Wober-Bingol, C. (2006) Facial pain and the second edition of the International Classification of Headache Disorders. Headache 46: 259263.
10. Obermann, M., 2010. Treatment options in trigeminal neuralgia. Therapeutic Advances in Neurological Disorders. doi:10.1177/1756285609359317
11. Cheshire WP. Trigeminal neuralgia: for one nerve a multitude of treatments. Expert Rev Neurother 2007; 7:1565–1579.
12. Maarbjerg S, Di Stefano G, Bendtsen L, Cruccu G. Trigeminal neuralgia – diagnosis and treatment. Cephalalgia 2017; 37: 648–657.
13. Gambeta, E., Chichorro, J.G., W. Zamponi, G., 2020. Trigeminal neuralgia: An overview from pathophysiology to pharmacological treatments. Molecular Pain. doi: 10.1177/1744806920901890.
14. Al-Quliti, K.W. (2015). Update on neuropathic pain treatment for trigeminal neuralgia: The pharmacological and surgical options. Neurosciences 20, 107–114.
15. Parmar, M., Sharma, N., Modgill, V., and Naidu, P. (2013). Comparative Evaluation of Surgical Procedures for Trigeminal Neuralgia. Journal of Maxillofacial and Oral Surgery 12, 400–409.
16. Sivakanthan, S., Van Gompel, J.J., Alikhani, P., Van Loveren, H., Chen, R., and Agazzi, S. (2014). Surgical management of trigeminal neuralgia: Use and cost-effectiveness from an analysis of the medicare claims database. Neurosurgery 75, 220–226.
17. Mihajev I., Stevanov Z., Radovanov M., Velickovic P.: Neuralgijatrigeminalnognerva-prikazslucaja, Aktuelnostiizneurologije, psihijatrijeigranicnihpodrucja, God XI, Br.3, 2003
18. Bick, S. K. B., &Eskandar, E. N. (2017, July 1). Surgical Treatment of Trigeminal Neuralgia. Neurosurgery Clinics of North America. W.B. Saunders. https://doi.org/10.1016/j.nec.2017.02.009.
19. Zakrzewska, JM, McMillan R. Trigeminal neuralgia: the diagnosis and management of this excruciating and poorly understood facial pain. Postgrad Med J 2011; 87:410e416.
20. Zakrzewska, JM, Wu, J, Mon-Williams, M, Phillips, N, Pavitt, SH. Evaluating the impact of trigeminal neuralgia. Pain 2017; 158: 1166– 1174.
21. Wiffen PJ, Derry S, Moore RA, Kalso EA. Carbamazepine for chronic neuropathic pain and fibromyalgia in adults. Cochrane Database Syst Rev 2014; 4:CD005451.
22. Campbell FG, Graham JG, Zilkha KJ. Clinical trial of carbazepine (tegretol) in trigeminal neuralgia. J NeurolNeurosurg Psychiatry 1966; 29: 265–267.
23. Nicol CF. A four year double-blind study of tegretol in facial pain. Headache 1969; 9: 54–57.
24. Rasmussen P, Riishede J. Facial pain treated with carbamazepin (Tegretol). Acta NeurolScand 1970; 46:385–408.
25. Di Stefano G, La Cesa S, Truini A, Cruccu G (2014) Natural history and outcome of 200 outpatients with classical trigeminal neuralgia treated with carbamazepine or oxcarbazepine in a tertiary Centre for neuropathic pain. J Headache Pain 9(15):34
26. Liebel JT, Menger N, Langohr H (2001) Oxcarbazepine in der Behandlung der Trigeminusneuralgie. Nervenheilkunde 20:461–465.
27. Beydoun A (2000) Safety and efficacy of oxcarbazepine: results of randomized, double-blind trials. Pharmacotherapy 20:152S–158S.
28. Zakrzewska JM, Chaudhry Z, Nurmikko TJ, et al. Lamotrigine (lamictal) in refractory trigeminal neuralgia: results from a double blind placebo controlled crossover trial. Pain 1997; 73:223–30.
29. Qin, Z., Xie, S., Mao, Z., Liu, Y., Wu, J., Furukawa, T.A., Kwong, J.S.W., Tian, J., and Liu, Z. (2018).Comparative efficacy and acceptability of antiepileptic drugs for classical trigeminal neuralgia: A Bayesian network meta-analysis protocol. BMJ Open 8.
30. Yuan M, Zhou HY, Xiao ZL, et al. Efficacy and safety of gabapentin vs. carbamazepine in the treatment of trigeminal neuralgia: a meta-analysis. Pain Pract2016; 16:1083–91.
31. Wiffen PJ, Derry S, Moore RA, et al. Carbamazepine for acute and chronic pain in adults. Cochrane Database Syst Rev 2011; 1:CD005451.
32. Moore RA, Straube S, Wiffen PJ, et al. Pregabalin for acute and chronic pain in adults. Cochrane Database Syst Rev 2009; 3:CD007076.
33. Yuan M, Zhou HY, Xiao ZL, et al. Efficacy and safety of gabapentin vs. carbamazepine in the treatment of trigeminal neuralgia: a meta-analysis. Pain Pract 2016; 16: 1083–1091.
34. Cruccu G, Gronseth G, Alksne J, et al. AAN-EFNSguidelines on trigeminal neuralgia management. Eur JNeurol 2008; 15: 1013–1028.
35. Puri N, Rathore A, Dharmdeep G, Vairagare S, Prasad BR, Priyadarshini R, Singh HP. A clinical study on comparative

evaluation of the effectiveness of carbamazepine and combination of carbamazepine with baclofen or capsaicin in the management of trigeminal neuralgia. Niger J Surg 2018; 24: 95–99.
36. Hassan S, Khan NI, Sherwani OA, Bhatt W, Asif S. Trigeminal neuralgia: an overview of literature with emphasis on medical management. Int Research J Pharmcol. 2013; 3:235–238.
37. Khalid W, Quliti A. Update on neuropathic pain treatment for trigeminal neuralgia Neurosciences 2015 Apr; 20(2): 107–114.
38. Colloca L, Ludman T, Bouhassira D et al. Neuropathic pain. Nat Rev Dis Primers 2017; (3):17002.
39. Attal N & Bouhassira D. Pharmacotherapy of neuropathic pain: which drugs, which treatment algorithms? Pain 2015; 156 Suppl1:S104–114.
40. Sivakumar W, Karsy M, Brock A, Schmidt RH: Postoperativepain control with the fentanyl patch and continuous paravertebral anesthetic infusion after posterior occipitocervical junction surgery. Cureus17:e645, 2016.
41. Coven I, Dayisoylu EH: Evaluation of sphenopalatine ganglion-blockade via intra oral route for the management of atypical trigeminal neuralgia. Springerplus 5: 906, 2016
42. Clinical Pharmacist, December 2017, Vol 9, No. 12, online | DOI: 10.1211/CP.2017.20203641.
43. Himshweta Das., et al. "Therapeutic Effect of Botulinum Toxin in Trigeminal Neuralgia: Case Report". EC Dental Science 18.5 (2019): 992-996.
44. Micheli F, Scorticati MC, Raina G (2002). Beneficial effects of botulinum toxin type for patients with painful tic convulsif. Clin Neuropharmacol 25:260–262.
45. Bohluli B, Motamedi MH, Bagheri SC et al (2011) Use of botulinum toxin A for drug-refractory trigeminal neuralgia: preliminary report. Oral Surg Oral Med Oral Pathol Oral RadiolEndod 111:47–50.
46. Morra, M.E., Elgebaly, A., Elmaraezy, A., Khalil, M., Altibi, A. M., Vu, T.L., Hirayama, K. (2016).Therapeutic efficacy and safety of botulinumtoxin. A Therapy in trigeminalneuralgia: Asystematic review and meta-analysis of randomized controlled trials.The Journal of Headache and Pain,17(1), 63.
47. Shackleton, T., Ram, S., Black, M., Ryder, J., Clark, G.T.,& Enciso, R. (2016).The efficacy of botulinum toxin for the treatment of trigeminal and postherpetic neuralgia: Asystematic review with meta-analyses. Oral Surgery, Oral Medicine, Oral Pathology and Oral Radiology,122(1),61–71.
48. Lakhan, S.E., Velasco, D.N., & Tepper, D. (2015). Botulinum toxin-A for painful diabetic neuropathy: A meta-analysis. Pain Medicine, 16(9), 1773–1780.
49. Wei J, Zhu X, Yang G, et al. Theefficacy andsafety of botulinum toxin type A in treatment of trigeminal neuralgia and peripheral neuropathic pain: A meta-analysis of randomized controlled trials. BrainBehav. 2019; 9:e01409.
50. Zakrzewska, JM, Patsalos, PN. Long-term cohort study comparing medical (oxcarbazepine) and surgical management of intractable trigeminal neuralgia. Pain 2002; 95: 259– 266.
51. Zakrzewska, JM, Lopez, BC, Kim, SE, Coakham, HB. Patient reports of satisfaction after microvascular decompression and partial sensory rhizotomy for trigeminal neuralgia. Neurosurgery 2005; 56: 1304– 1311.
52. Heinskou, T, Maarbjerg, S, Rochat, P, Wolfram, F, Jensen, RH, Bendtsen, L. Trigeminal neuralgia – a coherent cross-specialty management program. J Headache Pain 2015; 16: 66.
53. Jannetta PJ. Arterial compression of the trigeminal nerve at the pons in patients with trigeminal neuralgia. J Neurosurg. 1967; 26(1 Suppl):159–162. doi:10.3171/jns.1967.26.1part2.0159
54. Hitchon P, Holland M, Noeller J, Smith M, Moritani T, Jerath N, He W. Options in treating trigeminal neuralgia: Experience with 195 patients. Clin Neurol Neurosurg. 2016 Oct; 149:166-70.
55. Liu, R., Deng, Z., Zhang, L., Liu, Y., Wang, Z., and Yu, Y. (2020). The long-term outcomes and predictors of microvascular decompression with or without partial sensory rhizotomy for trigeminal neuralgia. Journal of Pain Research 13, 301–312.
56. Jarrahy R, Eby JB, Cha ST, Shahinian HK. Fully endoscopic vascular decompression of the trigeminal nerve. Minim Invasive Neurosurg. 2002; 45:32–5.
57. Lovely TJ, Jannetta PJ. Microvascular decompression for trigeminal neuralgia: surgical technique and long-term results. Neurosurg Clin N Am. 1997; 8:11–29.
58. Sanchez-Mejia RO, Limbo M, Cheng JS, et al. Recurrent or refractory trigeminal neuralgia after microvascular decompression, radiofrequency ablation, or radiosurgery. Neurosurg Focus 2005; 18:1–6.
59. Theodosopoulos PV, Marco E, Applebury C, et al. Predictive model for pain recurrence after posterior fossa surgery for trigeminal neuralgia. Arch Neurol2002; 59:1297–302.
60. Katherine Holste, MD, Alvin Y Chan, MD, John D Rolston, MD, PhD, Dario J Englot, MD, PhD, Pain Outcomes Following Microvascular Decompression for Drug-Resistant Trigeminal NeuraIgia: A Systematic Review and Meta-Analysis, Neurosurgery, Volume 86, Issue 2, February 2020, Pages 182–190.
61. Longhi, M., Rizzo, P., Nicolato, A., Foroni, R., Reggio, M., and Gerosa, M. (2007). Gamma knife radiosurgery for trigeminal neuralgia: Results and potentially predictive parameters - Part I: Idiopathic trigeminal neuralgia. Neurosurgery 61, 1254–1260.
62. L. Leksell, "Sterotaxic radiosurgery in trigeminal neuralgia," Acta Chirurgica Scandinavica, vol. 137, no. 4, pp. 311–314, 1971.
63. Dhople AA, Adams JR, Maggio WW, Naqvi SA, Regine WF, Kwok Y: Long-term outcomes of Gamma Knife radiosurgery for classic trigeminal neuralgia: implications of treatmentand critical review of the literature. Clinical article. J Neurosurg 111:351–358, 2009.
64. Kondziolka D, Zorro O, Lobato-Polo J, Kano H, Flannery TJ, Flickinger JC, et al: Gamma Knife stereotactic radiosurgery for idiopathic trigeminal neuralgia. J Neurosurg112:758–765, 2010.
65. Régis J, Metellus P, Hayashi M, Roussel P, Donnet A, BilleTurc F: Prospective controlled trial of gamma knife surgery for essential trigeminal neuralgia. J Neurosurg 104:913–924, 2006.
66. L. Leksell, "Sterotaxic radiosurgery in trigeminal neuralgia," Acta Chirurgica Scandinavica, vol. 137, no. 4, pp. 311–314, 1971.
67. Elaimy, A.L., Hanson, P.W., Lamoreaux, W.T., Mackay, A.R., Demakas, J.J., Fairbanks, R.K., Cooke, B.S., Thumma, S.R., and Lee, C.M. (2012). Clinical Outcomes of Gamma Knife Radiosurgery in the Treatment of Patients with Trigeminal Neuralgia. International Journal of Otolaryngology 2012, 1–13.
68. Peters, G., and Nurmikko, T.J. (2002). Peripheral and gasserian ganglion-level procedures for the treatment of trigeminal neuralgia. Clinical Journal of Pain 18, 28–34.
69. Nie, F., Su, D., Shi, Y., Chen, J., Wang, H., Chen, Y., Qin, W., and Wang, S. (2014). A prospective study of x-ray imaging combined with skin stimulation potential-guided percutaneous radiofrequency thermocoagulation of the gasserian ganglion for treatment of trigeminal neuralgia. Pain Medicine (United States) 15, 1464–1469.
70. Bendersky M, Hem S, Landriel F, et al. Identifyingthe trigeminal nerve branches for transovale radiofrequency thermolesion: "No pain, no stress". Neurosurgery 2012; 70(2):259–63.
71. Guo, J., Dong, X., and Zhao, X. (2016). Treatment of trigeminal neuralgia by radiofrequency of the Gasserian ganglion. Reviews in the Neurosciences 27, 739–743.
72. Deng, M., Cai, H., Fang, W., and Long, X. (2018). Three-dimensionally printed personalized guide plate for percutaneous radiofrequency thermal coagulation in idiopathic trigeminal neuralgia. International Journal of Oral and Maxillofacial Surgery 47, 392–394.

73. Mullan S, Lichtor T. Percutaneous microcompression of the trigeminal ganglion for trigeminal neuralgia. J Neurosurg1983; 59:1007–12.
74. Ying, X., Wang, H., Deng, S., Chen, Y., Zhang, J., and Yu, W. (2017). Long-term outcome of percutaneous balloon compression for trigeminal neuralgia patients elder than 80 years: A STROBE-compliant article. Medicine (United States) 96.
75. Skirving, D. J., & Dan, N. G. (2001). A 20-year review of percutaneous balloon compression of the trigeminal ganglion. Journal of Neurosurgery, 94(6), 913–917.
76. Bergenheim, A.T., Asplund, P., and Linderoth, B. (2013). Percutaneous retrogasserian balloon compression for trigeminal neuralgia: Review of critical technical details and outcomes. World Neurosurgery 79, 359–368.
77. Asplund, P., Blomstedt, P., and Tommy Bergenheim, A. (2016). Percutaneous Balloon Compression vs Percutaneous Retrogasserian Glycerol Rhizotomy for the Primary Treatment of Trigeminal Neuralgia. Neurosurgery 78, 421–428.
78. Kodeeswaran M, Ramesh V G, Saravanan N, Udesh R. Percutaneous retrogasserian glycerol rhizotomy for trigeminal neuralgia: A simple, safe, cost-effective procedure. Neurol India 2015;63:889-94.
79. Kondziolka, D., & Lunsford, L. D. (2005). Percutaneous retrogasserian glycerol rhizotomy for trigeminal neuralgia: technique and expectations. Neurosurgical Focus, 18(5).
80. Xu, L., and Xu, M. (2010). Analysis of factors influencing the efficacy of percutaneous retrogasserian glycerol rhizotomy in patients with idiopathic trigeminal neuralgia. Scientific Research and Essays 5, 2446–2450.
81. Pollock, B.E. (2005). Percutaneous retrogasserian glycerol rhizotomy for patients with idiopathic trigeminal neuralgia: A prospective analysis of factors related to pain relief. Journal of Neurosurgery 102, 223–228.
82. Paranathala, M.P., Ferguson, L., Bowers, R., and Mukerji, N. (2018). Percutaneous retrogasserian glycerol rhizotomy for trigeminal neuralgia: an alternative technique. British Journal of Neurosurgery 32, 657–660.

UDC: 616.314–77:612.24–072.7

# SPIROMETRIC EVALUATION OF RESPIRATORY CAPACITY AT USERS OF TOTAL DENTURES (REVIEW)

# СПИРОМЕТРИСКА ПРОЦЕНА НА РЕСПИРАТОРНИОТ КАПАЦИТЕТ КАЈ КОРИСНИЦИТЕ НА ТОТАЛНИ ПРОТЕЗИ

**Tanturovski F.[1], Bajevska J.[2] Bajevska J.[3]**

[1]Dental Clinic Dentin – Skopje, North Macedonia, [2]Deparment of Prosthetics, Faculty of Dental Medicine, 'Ss. Cyril and Methodius" University, Skopje, North Macedonia, [3]PZU "Bajevski" Skopje

## Abstract

Dental prosthetic appliances will function properly only if aligned with all the other components that comprise the masticatory organ. For a dental prosthesis to be therapeutic, it is expected to restore the effects of lost teeth and establish the function of: mastication, swallowing, phonation, aesthetics, and normal breathing, thus performing prophylaxis of living tissues. The purpose of this literature review is to determine the impact of complete removable dentures on the respiratory capacity of their carriers through spirometry test evaluation. Several authors have performed examinations to see if complete removable dentures have an impact on spirometry test results. **Key words:** Spirometry, edentulism – total tooth loss, removable total dentures.

## Апстракт

Протетичкиот додаток правилно ќе да ја врши функцијата само ако е вклопен со сите останати компоненти кои го сочинуваат џвакалниот орган. Се очекува тој да биде терапевтско средство со кое ќе се лечат последиците од изгубените заби и ќе се воспостават функциите на: џвакање, голтање, фонација, естетика и нормално дишење, така што при тоа ќе врши профилакса на живите ткива. Целта на овој литературен преглед е да се одреди влијанието на тоталните протези врз респираторниот капацитет кај нивните носители, преку спирометриска процена. Повеќе автори правеле испитувања за да воочат дали тоталните протези имаат влијание врз спирометарските проценi (тестови). **Клучни зборови:** клучни зборови: спирометрија, беззабност, тотални протези.

## Introduction

Over the last few decades, teeth of civilized nations have collapsed rapidly and massively. This problem is generalized around the world. It is present in all nations, regardless of gender and age. On the other hand, people from developing countries are affected proportionally to the extent of their economic development. This shows that there is a growing need for all kinds of dental prosthetic appliances, and dental prosthetics is being given more and more significance.

Dental prosthetics has long been important in terms of oral and general human health, so it was economically necessary to analyze the principles and adopt protocols and procedures for making removable complete dentures. It's all based on science and blends with the contemporary technological capabilities.

In 1903, the famous dental historian Geist - Jakobi claimed that dental prosthetics was a skill and had little to do with medicine. Until World War I, only the extracted tooth was studied. Those understandings changed radically when he began to study the tooth as part of a sophisticated and complex gnatological (masticatory) system.

Further advances in dentistry were related to the knowledge of the temporomandibular joint, its mechanics and kinetics. It was found that there was some relationship between the shape of the teeth and the movement of the joint. It is understood that the teeth, the upper and lower jaw, the skeletal muscles and the temporomandibular joint were an integral part of the single gnatological system and that they all together constituted the functional unit - apparatus mandibulo-maxillaris, known as the stomatogenic system in the new Anglo-

American literature. The understanding of the functional unity of the teeth, the connective tissue connecting them to the jawbone, all soft tissues and glands, the cheek and tongue muscles, and the temporomandibular joint (TMJ), which have become dominant in the science of dental prosthetics, places dental prosthetics equal with medicine.

The gnatological system is a very complicated integral part of the human body, made up of many components. Dysfunction or lack of function of any of its components changes the function of the entire system. With the loss of teeth, only one component of that system is missing, and the prosthetic appliance has to take over all the functions of that component. Prosthetic therapy must take into account all of these relationships. Only this condition enables proper treatment of any defects in the gnatological system, i.e. each treatment should begin with a view of achieving a functional unit, that is to say, known in the literature as oral rehabilitation.

Dental prosthetic appliance can only perform its function properly if it is aligned with all the other components that make up the gnatological system. The modern prosthetist is aware that a dental prosthesis can fit successfully into that system only if the damaged functions of the gnatological system are well known. These data have, over the last decades, led to a significant development of the science of dental prosthetics and conviction that it is not just a mechanical-craft problem, but that every singular prosthetic appliance is a biological and medical challenge. Today, the prerequisite for understanding and solving prosthetic problems requires knowledge from several sciences, such as chemistry, physics, biology, histology, physiology, pathology, bacteriology, hygiene, surgery, and medical psychology. Without the knowledge of all these disciplines, today's modern dental prosthetics cannot be imagined. And so, by applying general medicine and studying how living tissue responds to a foreign body, the underlying biological science of dental prosthetics is gradually evolving.

It was ascertained that removable prosthesis is a therapeutic appliance that restores the effects of lost teeth and that its task was not only to establish masticatory, aesthetic, and phonetic rehabilitation, but also prophylactic to the harmful effects that the removable prosthetic denture can cause on living tissue and mucosa. If we keep in mind the prophylactic properties of the removable prosthetic appliance, the modern prosthetist should also have in mind the basic setting of general medicine, which also applies to dentistry, which is primum non nocere, i.e. with the prosthetic appliance, primarily the patient must not be harmed.

The removable prosthesis is a foreign body, which necessarily causes some tissue damage. The prophylactic task is to minimize the harmful impacts, although today's remedies cannot yet be prevented[1].

In other words, if the dental prosthesis is poorly prophylactic, hygienic or laboratory-made, it will have a harmful effect on the surrounding tissues.

To ensure proper retention and stability of the complete removable dentures, they must be extended to the soft palate in the maxilla and to the retro-mandibular space in the mandible[2,3,4,5]. This means that the volume of the oral cavity is reduced and some crucial functions, such as speech and chewing efficiency, become limited[6,7,8]. Breathing is one of the most important vital functions and can be described as the exchange of gases between the living organism and the atmosphere, which is necessary to carry out the metabolic processes in the body (the living organism) [9]. In the process of breathing through the mouth, oral tissues and existing removable complete prosthesis in the mouth are the first structures to come into contact with the air that passes through the upper airways. It has been found that edentulism causes changes in the pharyngeal muscle[10, 11].

Lung functional tests are appropriate tests for the physiological evaluation of the respiratory system, for the diagnosis of pathological processes and for appropriate clinical management[12]. Spirometry is just one of the diagnostic methods for measuring lung function. It measures static and dynamic pulmonary volumes and flow capacities (vital capacity - VC, forced vital capacity - FVC, 1 second forced expiratory volume – FEV1, Tiffany's index - FEV1 / FVC, top expiratory flow for 25% of FVC - FEF75, forced expiratory flow to 50% of FVC - FEF50, forced expiratory flow to 75% of FVC - FEF25)[13]. The spirometry test has an appropriate quality control protocol and appropriate standards, which is the latest of the current standard for performing spirometry evaluations. These measurements are performed under the same conditions using the same spirometer. Spirometer calibration is daily required due to environmental factors (room temperature, air pressure, relative humidity), which adjusts the BTPS standard[14]. Prior to testing, it is necessary to obtain information on the patient's sex, age, body height, and weight, which are then compared to individual standards (expected values of the respondent). That standard, today, is embedded in the spirometer and does not need to be calculated because the device automatically calculates it. The European Coal and Steel Community Standard (CECA II)[15] is the most commonly used today.

After entering the appropriate patient data into the spirometer, the patient should stand upright and comfortably. The laboratory assistant explains the need and the way to perform the test by demonstrating the correct technique for performing the test. Patients undergo sev-

eral rehearsals until they have mastered the proper technique needed for proper spirometry. The patient should be encouraged to complete the exhalation process. It is important in this procedure to limit the number of trials (rehearsals and actual measurements) to 8 or less, to avoid patient exhaustion and inadequate results.

The spirometry test is performed by deep exhalation, deep inhalation and deep exhalation again through the spirometer pipe. This is repeated three times. The air must not pass through the nose, so a nose clip is used. During each measurement of the spirometry test, the technique of performing should be evaluated for each patient to avoid the appearance of an artefact in the final results. Possible artefacts may include the following: insufficient or incomplete inhalation, lack of labor on inhalation, additional inhalation, insufficient mouth closure, slow onset of exhalation, temporary exhaustion, partial nasal exhalation, coughing during the first second of exhalation etc. Technically, satisfactory spirometry evaluation should be done three times. These three measurements should be consistent (reproducible). Of these, two should not differ more than 100 ml (for FVC and FEV1), i.e. 150 ml from each other. When 3 satisfactory measurements are achieved, those with the highest FVC and FEV1 are selected and used for interpretation (compared to the individual standard), to calculate the percentages of expected values for that person/patient (measured value)/default (expected value x 100%). Thus, the calculated percentage serves for clinical evaluation of spirometry test results[14,16].

Patients with complete tooth loss often face problems while performing spirometry during their regular control of certain diseases, as their vertical dimension is lost. At the same time, rotation and displacement of the lower total denture, which is more mobile than the upper, reduces the posterior pharyngeal space. This is more symptomatic in patients suspected of having extrathoracic airway obstruction, as well as obstructive sleep apnea, parathyroid lymphadenopathy, etc.

Edentulism, also causes increased pharyngeal obstruction with worsening cardio-respiratory symptoms. Some authors recommend that the prosthesis should be in the mouth when performing spirometry, while others recommend removal of the prosthesis during the spirometry procedure[17,18,19].

## Objectives

The aim of this literature review was to determine the impact of complete dentures on the respiratory capacity of their carriers through performing a spirometry test.

## Materials and methods

The material consists of reviewed articles that examine spirometry test results in patients with complete tooth loss restored with complete removable dentures. The articles were acquired by research in international journals, as well as PubMed and NCBI database. Research was done with reviewed articles.

## Discussion

Previous studies have shown that there is a strict relationship between orofacial conditions and the upper respiratory tract[20,21,22,23,24,25]. However, until the end of the 20$^{th}$ century, clinical findings were not used to assess the respiratory functions of various dental conditions, such as partial or total tooth loss. The most significant clinical records of the relationship between the oral condition and the respiratory function appeared only in the late nineties of the twentieth century.

Bucca et al[26] reported that the Apnea-Hypopnea Index (AHI) showed nearly double values during sleep in patients with total tooth loss who did not wear dentures. Patients were 44 years of age and had obstructive sleep apnea (OSA) and chronic obstructive pulmonary disease (COPD), who wore total dentures because of loss and extraction of teeth. Cephalometric analyzes of patients revealed a significant narrowing of the anteroposterior oropharyngeal space from 1.5 to 0.6 cm. Following these striking findings, they expanded their studies to six male toothless patients with obstructive sleep apnea (OSA). The authors observed that removal of total dentures significantly reduced the retropharyngeal space and that sleeping with removed dentures was associated with a decrease in mean and low arterial blood saturation, while increasing the Apnea-Hypopnea index[26]. The authors concluded that removing the complete removable dentures significantly reduces the retropharyngeal distance in patients with obstructive sleep apnea (OSA) during sleep. They advised patients not to remove complete removable dentures while sleeping to avoid the risk of developing obstruction of the respiratory tract[26,27].

In another study for evaluating the impact of complete removable dentures on the Apnea-Hypopnea Index (AHI), 34 patients with obstructive sleep apnea (OSA) were examined. Ariska et al.[28] found that wearing complete removable dentures reduced the Apnea-Hypopnea Index (AHI) in 19 patients, whereas it increased the index in 8 patients during sleep. Interestingly, the improvement of the Apnea-Hypopnea Index (AHI) is not associated with a decrease in apnea score but with a

decrease in hypopnea score. In addition, there was no significant difference between the different prosthetic situations (with and without prosthesis in the mouth) with correlation to the mean and low oxygen saturation index in percentages ($SpO_2$) and desaturation index.

In another study, Bucca et al.10 performed spirometry studies on 76 toothless patients, of whom 36 were asymptomatic, 22 had a chronic obstructive pulmonary disease (COPD) and 18 had a interstitial pulmonary disease (ILD). These studies were performed to determine the effect of total dentures on the respiratory function. In addition, they reported that in asymptomatic patients and ILD patients, lung performance was slightly improved when complete removable dentures were in the mouths of patients. The authors have so far found no significant differences in COPD patients if they wore or didn't wear their dentures. According to Bucca et al.[26], values of maximal expiratory flow rate (MEF), forced expiratory flow rate to 50% (FIF50), forced inspiratory flow rate to 50% (FEF50) were increased in asymptomatic patients, while peak expiratory values flow (PEF) and forced expiratory flow (FEF50) were increased in ILD patients. There was no significant difference in the studies on forced vital capacity (FVC) and excretory volume at 1% (FEV1) in any patient group.

Contrary to previous studies, Almeida et al.[29] performed a polysomnographic evaluation of 23 non-OSA patients. They observed that wearing a complete removable denture during sleep increased the AHI index in mild cases. The results of this study were consistent with those of Almeida et al. However, when compared with a spirometry test, subjects should be considered to have different sleeping positions during polysomnography, as opposed to sitting upright in standard spirometry.

Bucca et al.[30] compared results of PSG (polysomnography) in toothless individuals while sleeping with and without dentures in the mouth, and the results showed that the AHI index and the main oxygen saturation were significantly worse in the nights of toothless, denture-free patients than in patients that wore dentures in the mouth at night.

Bucca et al.[31] with the help of spirometry, reported that toothless patients with prosthesis had improvement in some parameters when performing the spirometry. The quality of sleep parameters, such as: sleep efficiency, sleep 1, sleep 2, sleep 3, REM sleep percentages, AHI index, oxygen saturation and excitement index, may all avoid the Obstructive Sleep Apnea Syndrome (OSAS) when using the prostheses.

According to Bucca et al.[30] attention should be paid to individual toothless patients who sleep with their dentures in order to prevent OSAS problems. Erivigni et al.[32] performed cephalometric analyzes in patients with total dentures and their studies found that wearing dentures encouraged changes in the position of the jaw and the tongue, as well as in the pharyngeal space, which could be used as a useful tip to reduce apnea in total prosthesis carriers.

Pellegrino R, Viegi G, Brusasco V et al.[33] have also found that maximal inspiratory flow (PIF) is significantly reduced during extratoracal air obstruction, since airway pressure (which is approximately equal to atmospheric pressure) cannot resist the negative intraluminal pressure produced by the inspiratory. As a comparison, this has little effect on intratracheal air obstruction.

Toothless patients with obstructive sleep apnea may or may not have dentures in their mouth during spirometry tests during lung function tests to assess intratracheal airway (such as distinguishing obstructive from restrictive lung diseases), but they always have to use prostheses for extrathoracic airway compression (as in cases of obstructive sleep apnea, paratracheal tumors, paratraheal lymphadenopathy and laryngeal inflammation).

Piskin B et al.[34] observed the impact of total dentures on spirometry parameters in toothless patients. A total of 46 carriers of total dentures were included in the study. Respiratory functions were examined in four different oral conditions: no prosthesis, only upper, only lower, and both oral prostheses. Spirometric analyzes used forced vital capacity (FVC), peak expiratory flow (PEF), forced expiratory volume in 1 second (FEV1) and forced expiratory flow between 25% and 75% (FEF 25-75).

Data were analyzed with Friedman, Wilcoxon and T-test. ($p = 0.05$). Results showed that there was a significant difference between the parameters with presence or absence of oral dentures ($p < 0.05$).

In all spirometry parameters, the most significant difference was found between the forced vital capacity in the absence of dentures in the oral cavity, the forced vital capacity in the presence of the lower denture as well as in the forced expiratory volume in 1 second without dentures in the mouth, forced expiratory volume in 1 second with the presence of the upper total denture only.

It has been reported that complete removable dentures can adversely affect spirometry values in toothless patients.

Bucca CB et al.[35] compared the values of FVC, FEV1, PEFR, FEF50%, FIV1 and FIF50% recorded with and without prosthesis in three groups of toothless patients: 36 asymptomatic patients with normal spirometry, 22 patients with chronic obstructive pulmonary disease (COP) and 18 with interstitial pulmonary disease (ILD). In 14 subjects, the retropharyngeal space was examined by cephalometry.

Respondents with normal spirometry and interstitial pulmonary disease showed significantly low prosthesis-

free air flow, while subjects with chronic obstructive pulmonary disease showed no significant difference in spirometry values with or without prosthesis.

The retropharyngeal space was significantly increased by removing the dentures (from 1.52 +/- 0.07 to 1.16 +/- 0.09 cm, SEM, p <0.0001).

These findings indicate that in toothless patients with normal or restrictive pulmonary function, airflow values, with or without prosthesis, have small but significant differences. Also, such differences do not play a role in the results of spirometry analyzes, so toothless patients are recommended to wear the dentures during spirometry analyzes.

Carossa S. et al.[36] have investigated whether toothless patients with an increased pharyngeal muscle will have an impact on spirometry analysis. Spirometry tests were conducted in 58 toothless patients, with and without dental prosthesis, 36 of whom were asymptomatic, with normal pulmonary function, and 22 had chronic obstructive pulmonary disease (COPD).

In 10 patients, the retropharyngeal space with and without prosthesis was measured by cephalometry. In the group of patients with normal pulmonary function, removal of the prosthesis caused a significant reduction in lung volume and air flow values, while in patients with obstructive pulmonary disease, it didn't have any impact.

In both groups, the retropharyngeal space was significantly reduced by removing the dentures.

Loss of teeth, by reducing extratracheal airway calibration, significantly affects spirometric measurements in patients with normal lung function but not in patients with chronic lung disease.

Majumdar S. et al.[37] examined spirometry functions. Patients and controls (n = 10) for each of the groups of males and females were examined by spirometry using a computerized electronic spirometer with tolerance assessed by a modified Harvard step test.

Patients with type 2 diabetes mellitus (n = 4), post-tuberculosis (n = 7), hypothyroid (n = 6), collagen vascular disease group (n = 6) showed restrictive spirometry results, and IHD (ischemic heart disease) patients showed significant limitations in the tolerance values examined.

Many studies suggest that loss of teeth causes decrease in the size and tonus of pharyngeal muscles, which can cause apnea in these individuals[38].

The morphological changes caused by tooth loss consist of a reduced vertical dimension of occlusion, a decrease in the lower third of the face, and anterior rotation of the mandible[39].

Also, loss of teeth causes an unnatural position of the thongue[40].

All of these disorders cause occlusive disorders and airway obstruction[41].

Usually, the purpose of oral appliances (dental prosthesis) is to increase the airflow (to restore the space as much as possible to a more natural position in the presence of teeth) during sleep by repositioning the mandible forward and downward[42].

Bucca et al.[41] compared the results of polysomnography in toothless patients with and without dentures, and showed that the AHI index and the main oxygen saturation were significantly worse at night in patients that slept without dentures than in patients who slept with the dentures.

Bucca et al.[38] also investigated patients with obstructive sleep apnea syndrome (OSAS) using spirometry tests. From the spirometry test results they found that the values improved when patients had prostheses in their mouths.

The quality of sleep parameters, such as sleep efficiency, first phase of sleep, second phase of sleep, third phase of sleep, percentage of REM sleep, AHI index, oxygen saturation and arousal index, could be improved in the OSAS patient group by the use of complete dentures.

Traditionally, dentists advise their patients to sleep without total dentures at night to prevent the risk of overnight oral irritation[43].

According to Bucca et al.[41] special consideration should be given to patients who sleep with total dentures in order to reduce OSAS problems.

Erovigni et al.[44] performed cephalometric analyzes in patients with total dentures. They found that wearing dentures stimulated changes in the TMJ and in the tongue position, as well as in the pharyngeal airflow space, which in turn reduced apnea in patients.

Bucca et al.[45] and Gupta et al.[46] confirmed that removal of dentures significantly reduces the retropharyngeal space, and thus denture-free sleep is associated with a significant decrease in the apnea hypopnea index (AHI) and a decrease in hemoglobin blood saturation. Authors have highlighted that wearing dentures overnight will reduce the effects of OSA.

Carossa et al.[47] described the effect of complete removable dentures and their impact on the reduced pharyngeal space and showed a significant reduction in PAS (posterior airway space). They concluded that wearing dentures induces modifications in the position of the tongue, the TMJ, and the pharyngeal space.

Gupta et al.[48] examined patients with total tooth loss using cephalometry with increasing vertical jaw alignment. They found a significant correlation between PAS and the retropharyngeal space in patients with no teeth in the mouth and in patients with complete dentures. They

concluded that increasing the vertical dimension of the occlusion within the permissible range was advantageous for patients with OSA.

Ariska et al.[49] and Bucca et al.[50] investigated the risk of OSA in total tooth loss with and without total dentures. They concluded that the absence of dentures reduces oxygen saturation and reduces the retropharyngeal space, which in turn results a worsened situation of OSA patients.

In a study by Tsuda et al.[51], it was demonstrated that toothless favors air obstruction during sleep. After all, both the AHI and the main $SaO_2$ index were significantly worse in patients who slept without dentures than in patients who slept with dentures.

## Conclusion

This paper provides research from authors who have worked on the issue of respiratory function in patients wearing complete removable dentures. Their research was done to determine if complete removable dentures have an effect on airflow in the airways.

Patients with obstructive pulmonary disease are advised not to remove their complete removable dentures while sleeping to avoid the risk of upper airway collapse (worsening of results). This data confirms the findings that complete removable dentures in the oral cavity tend to restore the condition in the oral cavity to as normal as possible (vertical dimension and position of surrounding tissues).

On the other hand, in patients who did not use to have respiratory problems, a difference was observed in the results of spirometry depending on the individual.

Spirometry test results in cases who wore the complete removable dentures had an impact in some individuals, while that wasn't the outcome with other examined subjects.

This data cannot confirm with certainty whether patients should remove prostheses during examinations and overnight sleep, as some tests have shown no changes, while other have shown improvement in performing spirometry with oral prostheses.

Some individuals who have difficulties getting used to new conditions, do not fully accept the prostheses and this interferes while performing spirometry tests and wearing them at night while sleeping.

As mentioned earlier, for a complete removable denture to be appropriately accepted by the patient (its user), it must be maximally extended, thereby reducing the volume of the oral cavity and impeding the oral function. Therefore, it is traditionally accepted by dentists to advise their patients to sleep without the complete removable dentures in the mouth at night in order to prevent the risk of oral irritation.

From dental point of view, as a rule for the use of complete removable dentures, it should be noted that prosthesis should be removed from the oral cavity at a certain time of the day to avoid the risk of oral irritation. This period is usually overnight, because the person is inactive, with all the functions reduced to a minimum and can be easily applicable. For people active during the day, removing dentures from the oral cavity for tissue rest would be an additional obligation that can be overlooked by other daily tasks.

In patients with a clinical history of airway obstruction, it should be noted that for better results, dentures may be worn at night if it's not in another way problematic for them, but note that the break from wearing the prosthesis intended for rest of the oral tissues should not be neglected and should be practiced at other parts of the day.

## Reference


1. M. Suvin. Bioloski temelji protetike – totalna proteza 1988; VII dopunjeno izdanje: 1 – 4.
2. Roessler DM. Complete denture success for patients and dentist. Int Dent J 2003; 53: 340 – 345.
3. Koike T, Ishizaki K, Ogami K, et al. Influence of anterior palatal coverage on perception and retention in complete dentures. J Prosthet Dent 2011; 105: 272 – 279.
4. Zarb GA, Bolender CL, Eckert SE, et al. Proshodontic treatment of edentulous patients. Complete Dentures and Implant-Supported Proshteses. St. Louis: Mosby, 2004.
5. Mc Guire L, Millar K, Lindsay S. A treatment trial of an information package to help patients accept new dentures. Behav Res Ther 2007; 45: 1941 – 1948.
6. Stelzle F, Ugrinovic B, Knipfer C, et al. Automatic, computer-based speech assessment on edentulous patients with and without complete dentures – preliminary results. J Oral Rehabil 2010; 37: 209 – 216.
7. Goiato MC, Ribeiro PDP, Garcia AR, et al. Complete denture masticatory efficiency: a Literature review. J Calif Dent Assoc 2008; 36: 683 – 686.
8. Gokce HS, Gokce S, Akin E, et al. Effects of complete denture wearing on the head posture and posterior airway space. J Dental Sci 2011; 6: 6 – 13.
9. Pierce R. Spirometry: An essential clinical measurement. Aust Fam Phusician 2005; 34: 535 – 539.
10. Bucca C, Carossa S, Colagrande, et al. Effect of edentulism on spirometric test. Am j Respir Critr Care Med 2001; 162: 1018 – 1020.
11. Gupta P, Thombare R, Singhal S, Pakhan AJ. Obstructive sleep apnea and edentulism role of complete dentures/oral appliance from prosthodontics respective: a review. Indian J Sleep Med 2010; 5: 116 – 119.
12. American Thoracic Society. Medical section of the American Lung Association. Am Rev Respir Dis 1991; 144: 1202 – 1218.
13. Chronic Obstructive Pulmonary Disease: National clinical guideline for management of chronic obstructive pulmonary disease in adults in primary and secondary care. Thorax 2004; 59 (Suppl 1):1-232.

14. Miller MR, Hankinson J, Brusasco V, Burgos F, Casaburi R, Coates A, Crapo R, Enright P, van der Grinten CP, Gustafsson P, Jensen R, Johnson DC, MacIntyre N, McKay R, Navajas D, Pedersen OF, Pellegrino R, Viegi G, Wanger J. ATS/ERS Task Force. Standardisation of spirometry. Eur Respir J 2005; 26(2):319-38.
15. Quanjer PH, Tammeling GJ, Cotes JE, Pedersen OF, Peslin R, Yernault JC. Lung volumes and forced ventilatory flows. Report Working Party Standardization of Lung Function Tests, European Community for Steel and Coal. Official Statement of the European Respiratory Society. Eur Respir J 1993; 6 (Suppl 16):5-40.
16. Hyatt RE, Scanlon PD, Nakamura M. Interpretation of Pulmonary Function Tests. A Practical Guide. Second Edition. Lippincot Philadelphia: Williams and Wilkins, 2003.
17. Morris AH. Clinical pulmonary function testing: a manual of uniform laboratory procedures, 2nd ed. Salt Lake City, UT. Intermountain Thoracic Society; 1984.
18. Waner J. Pulmonary function testing: a practical approach, 1st ed. Baltimore: Williams & Wilkins; 1992.
19. Cotes JE. Assessment of bellows and mechanical attributes of the lung. In: Cotes JE, editor Lung function, 5th ed. Oxford: Blackwell Scientific Publications; 1993.
20. Solow B, Skov S, Ovesev J, et al. Airway dimensions and head posture in obstructive sleep apnea. Eur J Orthod 1996; 18: 571 – 579.
21. Choi JK, Hur YK, Lee JM, et al. Effects of mandibular advancement on upper airway dimension and collapsibility in patients with obstructive sleep apnea using dynamic upper airway imaging during sleep. Oral Surg Oral Med Oral Pathol Oral Radiol Ednod 2010; 109: 712 – 719.
22. Lowe AA, Gionhaku N, Takeuchi K, et al. Three dimensional CT reconstructions of tongue and airway in adult subjects with obstructive sleep apnea. Am j Orthod Dentofacial Orthop 1986; 90: 364 – 374.
23. Clark GT, Arand D, Chung E, et al. Effect of anterior mandibular positioning on obstructive sleep apnea. Am Rev Respir Dis 1003; 147: 624 – 629.
24. Muto T, Yamazaki A, Takeda S. A cephalometric evaluation of the pharyngeal airway space in patients with mandibular retrognathia and prognathia, and normal subjects. Int J Oral Maxillofac Surg 2008; 37: 228 – 231.
25. Martin O, Muelas L, Vinasa MJ. Nasopharyngeal cephalometric study of ideal occlusions. Am J Orhod Dentofacial Orghop 2006; 130: 436. El-436. e9.
26. Bucca C, Carossa S, Pivetti S, et al. Edentulism and worsening of obstructive sleep apnea. Lancet 1999; 353: 121 – 122.
27. Bucca C, Cicolin A, Brussino L, et al. Tooth loss and obstructive sleep apnea. Respir Res 2006; 17: 7 – 8.
28. Arisaka H, Sakuraba S. Tamaki K, et al. Effects of wearing complete dentures during sleep on the Apnea – hypopnea index. Int J Prosthodont 2009; 22: 173 – 177.
29. Almeida FR, Furuayama RJ, Chaccur DC, et al. Complete denture wear during sleep in elderly sleep apnea patients-a preliminary study. Sleep Breath 2011; DOI: 10.1007/s11325-011-0587-9.
30. Bucca C, Cicolin A, Brussino L, Arienti A, Graziano A, Erovigni F, et al. Tooth loss and obstructive sleep apnoea. Respir res 2006; 7:8
31. Bucca CB, Carossa S, Colagrande P, Brussino L, Chiavassa G, Pera P, et al. Effect of edentulism on spirometric tests. Am J Respir Crit Care Med 2001; 163:1018-20
32. Erovigni F, Graziano A, Ceruti P, Gassino G, De Lillo A, Carossa S, Cephalometric evaluation of the upper airway in patients with complete dentures. Minerva Stomatol 2005; 54: 293-301.
33. Pellegrino R, Viegi G, Brusasco V, Crapo RO, Burgos F, Casaburi R et al. Interpretative strategies for lung function test. EurRespir J. 2005; 26: 948-68.
34. Piskin B1, Sipahi C, Karakoc O, Atay A, Ciftci F, Tasci C, Akin H, Arisan V, Sevketbeyoglu H, Turker T. Effects of complete dentures on respiratory performance: spirometric evaluation. Gerodontology. 2014 Mar; 31(1): 19-24.
35. Bucca CB1, Carossa S, Colagrande P, Brussino L, Chiavassa G, Pera Pm Rolla G, Preti G. Effect of edentulism on spirometric tests. Am J Respir Crit Care Med. 2001 Mar; 163(4): 1018-20.
36. Carossa S1, Pera P, De Lillo A, Corsalini M, Lombardo S, Buvva C. The influence of edentulism on spirometric values. Minerva Stomatol. 2000 Sep; 49(9): 405-8.
37. Majmudar S1, Sen S. Mandal SK. A hospital-based study on pulmonary function tests and exercise tolerance in patients of chronic obstructive pulmonary disease and other diseases. J Indian Med Assoc. 2007 Oct; 105(10): 565-6, 568, 570 passim.
38. Bucca CB, Carossa S, Colagrande Pm Brussino L, Chiavassa G, Pera P et al. Effect of edentulism on spirometric tests. Am J Respir Crit Care Med 2001: 163: 1018-20.
39. Ivanhoe JR, Cibirka RM, Lefabvre CA, Parr GR. Dental considerations in upper airway sleep disorders: A review of the literature. J Prosthet Dent 1999: 82:685-98.
40. Kotsiomiti E, Kapari D. Resting tongue position and its relation to the state of the dentition: A pilot study. J Oral Rehabil 2000:27: 349-54.
41. Bucca C, Cicolin A, Brussino L, Arienti A, Graziano A, Erovigni F et al. Tooth loss and obstructive sleep apnea. Respir Res 2006:7:8.
42. Marklund M. Predictors of long-term orthodontic side effects from mandibular advancement devices in patients with snoring and obstructive sleep apnea. Am J Orthod Dentofacial Orthop 2006:129:214-21.
43. Budtz-Jörgensen E. Prognosis of overdenture abutments in the aged: Effect of denture wearing habits. Community Dent Oral epidemiol 1992:20:302-6.
44. Erovigni F, Graziano A, Ceruti P, Gassino G, De Lillo A, Carossa S, Cephalometric evaluation of the upper airway in patient with complete dentures. Minerva Stomatol 2005:54:293-301.
45. Bucca C, Carossa S, Pivetti S, Gai V, Rolla G, et al. (1999) Edentulism and worsening of obstructive sleep apnoea. Lancet 353: 121-122.
46. Gupta P, Thombare R, Singhal S, Pakhan AJ (2010) Obstructive sleep apnea and edentulism-role of complete dentures/oral appliance from prosthodontics respective: a review Indian J Sleep Med 5: 116-119. Bucca C, Cicolin A, Brussino L, Aroenti A, Graziano A, et al. (2006) Tooth loss and obstructive sleep apnea, Respir Res 7:8.
47. Carossa S, Bucca C, de Lillo A, Corsalini M, Rizzatti A, et al. (2000) Correlation between edentulism, sleep disorders and arterial hypertension. Preliminary research. Minerva Stomatol 49: 399-404.
48. Gupta P, Thombare R, Pahlan AJ, Singhal S (2011) Cephalometric evaluation of the effect of complete dentures on retropharyngeal space and its effect on spirometric values in altered vertical dimension. ISRN Dent 51:69.
49. Arisaka H, Sakuraba S, Tamaki K, Watanabe T, Takeda J, et al. (2009) Effects of wearing complete dentures during sleep on the apnea-hypopnea index. Int J Prosthodont 22: 173-177.
50. Buvva C, Cicolin A, Brussino L, Arienti A, Graziano A, et al. (2006) Tooth loss and obstructive sleep apnea. Respir Res 7:8
51. Tsuda H, Almeida FR, Walton JN, Lowe AA (2010) Questionnaire-based study on sleep-disordered breathing among edentulous subjects in a university oral health center. Int J Prosthodont 23: 503-506.

UDC: 616.314–77:612.24–072.7

# IMPACT OF COMPLETE REMOVABLE DENTURES ON THE RESPIRATORY TRACT AIRFLOW

# ВЛИЈАНИЕ НА ТОТАЛНИТЕ ПРОТЕЗИ ВРЗ ПРОТОКОТ НА ВОЗДУХ ВО РЕСПИРАТОРНИОТ ТРАКТ

**Tanturovski F.[1], Bajevska J.[2]**

[1]Dental Clinic Dentin – Skopje, North Macedonia, [2]Deparment of Prosthetics, Faculty of Dental Medicine, 'Ss. Cyril and Methodius" University, Skopje, North Macedonia

## Abstract

Prosthetic restoration performs its function properly only if it fits with all other components that contain the masticatory organ. **The aim of** this paper is to determine the effect of complete removable dentures on the respiratory tract airflow through the process of spirometry. In our study, we used spirometry (measurement of respiration), which is the most common test for lung function, especially the amount (volume) and/or speed (flow) of air that can be inhaled and exhaled. The tests were performed on 8 patients with complete removable dentures who were healthy individuals. Spirometry was used as a method of work at the Institute of Medical Physiology and Anthropology at the Medical Faculty, "Ss. Cyril and Methodius" University in Skopje. Examinations were performed in different oral conditions: when both prostheses were present, when only the upper prosthesis was present, when only the lower prosthesis was present, and when both prostheses were not present in the oral cavity. The research was done to see if total dentures have an effect on airflow in the airway, or if their presence has an effect on the normal functioning of the respiratory tract. **Keywords:** complete removable dentures, spirometry, respiratory tract.

## Апстракт

Протетската реставрација правилно ја врши функцијата само ако е вклопена со сите останати компоненти кои го сочинуваат џвакалниот орган. **Целта** на овој труд е да се одреди влијанието на тоталните протези врз протокот на воздух во респираторниот тракт преку процесот на спирометрија. Во нашава студија користиме спирометрија (мерење на дишењето), која претставува најчест тест за функцијата на белите дробови, посебно на количината (волуменот) и/или брзината (протокот) на воздух кој може да се вдишува и издишува. Испитувањата се изведени кај 8 пациенти кои носат тотални протези и се здрави индивидуи. Како метод за работа се користеше спирометрија при Институтот за медицинска физиологија и антропологија при Медицинскиот факултет, Универзитет „Св. Кирил и Методиј" Скопје. Испитувањата се изведуваа во различни орални состојби: кога се присутни двете протези, кога е присутна само горната протеза, кога е присутна само долната протеза, и кога не се присутни протезите во усната празнина. Истражувањето беше направено со цел да се воочи дали тоталните протези имаат влијание врз протокот на воздух во дишните патишта, односно дали нивното носење има влијание врз нормалното функционирање на респираторниот тракт. **Клучни зборови:** тотални протези, спирометрија, респираторен тракт.

## Introduction

A dental prosthesis is a therapeutic tool that treats the consequences of lost teeth, and its task is not only to establish chewing, aesthetic, and phonetic rehabilitation but also to prevent harmful effects caused by the prosthesis of living tissue and mucous membranes. If we bear in mind the prophylactic property of the prosthesis, the modern prosthetist should also take care of the basic set of general medicine, which also applies to dentistry, and that is "primum non nocere", i.e. with the prosthetic treatment, the patient must not be harmed in the first place.

The prosthesis is a foreign body that necessarily causes some tissue damage. The prophylactic task is to minimize the harmful effects, although with today's knowledge it is still not possible to prevent it[1].

Today, the dental prosthesis is expected to be a therapeutic tool that will treat the effects of lost teeth and will establish the function of chewing, swallowing, phonation, aesthetics, and normal breathing, thus preventing damage on living tissues. In other words, if the prosthesis is poorly made prophylactically, hygienically, or with lack of laboratory skills, it will adversely affect the surrounding tissues.

Spirometry is just one of the diagnostic methods for measuring lung function (respiratory tract). It measures static and dynamic lung volume and flow capacities (vital capacity - VC, forced vital capacity - FVC, forced expiratory volume in 1 second - FEV1, Tiffany's index –

FEV1/FVC, peak expiratory flow PEF, forced expiratory flow on 25% of FVC - FEF75, forced expiratory flow on 50% of FVC – FEF50, forced expiratory flow on 75% of FVC - FEF25[2]. Spirometry measurement has an appropriate quality control protocol and proper standards, the last of which is published and is the current standard for performing spirometry measurements. These measurements are performed under the same conditions, using the same spirometer. Spirometer calibration is required daily due to surrounding factors (room temperature, air pressure, relative humidity), which would correct the measured volume in standard conditions (BTPS standard)[3]. Before testing, it is necessary to obtain information about the patient's gender, age, body height, and weight, which are then compared to individual standards (the respondent's expected values). That standard is embedded into the spirometer and does not need to be calculated, because the device automatically calculates it. Today, the European Coal and Steel Community (CECA II) standard is the most commonly used[4].

After entering the appropriate patient data in the spirometer (gender, age, weight, and height), the patient should stand upright and comfortably. The laboratory technician explains the need and the method of performing the test by demonstrating the correct technique for performing the test. Several tests (drills) are performed with the patient until he/she masters the correct technique, required for a properly performed spirometry. The patient should be encouraged to complete the exhalation process. In this procedure, it is important to limit the number of trials (drills and actual measurements) to 8 or less, to avoid exhaustion of the patient and improperly obtained results.

Spirometry is measured by deep exhalation, deep inhalation, and deep exhalation again through the pipe of the spirometer. This is repeated 3 times. The air must not escape through the nose, so patients are pinched on the nose with a nose clip.

During each measurement, the technique for each patient should be evaluated to avoid the appearance of an artifact in the final results. Possible artifacts include: insufficient or incomplete inhalation, shortness of breath during exhalation, additional inhalation during exhalation, unsatisfactory closing of the mouth, slow onset of exhalation, temporary cessation of exhalation, part of nasal exhalation, coughing during the first second of exhalation, etc. Technically, satisfactory spirometry measurement should be done three times. Those three measurements should be consistent (reproductive). Two of those should not differ more than 100 mL (for FVC and FEV1), i.e. 150 mL from each other. When 3 satisfactory measurements are made, the one with the highest number of FVC and FEV1 is selected, and it is later used for interpretation (compared to the individual standard) to calculate the percentages of the expected values for that person (measured value/standard (expected value) x 100%). The calculated percentage, thus serves as clinical estimate of spirometry measurements[3, 5].

## Purpose of the paper

The aim of this paper is to determine the effect of complete removable dentures on the respiratory tract airflow of their carriers through spirometry evaluation.

## Materials and methods

Tests were performed on 8 patients with complete removable dentures. Patients were selected at their regular check-up at the Department of Fixed and Removable Dental Prosthodontics at the PHO Dental Clinical Center "St. Pantelejmon", and we were guided by certain criteria, which were:

- Patients with complete tooth loss;
- Patients with complete removable dentures;
- Patients wearing the complete removable dentures for less than 5 years;
- Patients did not complain about existing dentures (wear them daily);
- There were no errors in the production of existing prostheses;
- Patients do not smoke;
- Patients do not to suffer from any respiratory diseases, such as asthma or chronic obstructive pulmonary disease;
- Patients do not have any cardiovascular or other systemic diseases.

Spirometry was used as a working method for this paper at the Institute of Medical Physiology and Anthropology at the Medical Faculty, University "St. Cyril and Methodius" Skopje. The required parameters for our study were: forced vital capacity - FVC, peak expiratory flow - PEF, forced expiratory volume in 1 second - FEV1, forced expiratory flow 25-75% - FEF25-75. Patients were sent to the Institute of Medical Physiology, where they rested for 5 minutes and were placed in a chair next to the spirometer. The laboratory technician gave them instructions on how to perform the test properly. The patient data (ordinal number, name, surname, date of birth, height, weight) were registered. Next was the placement of a nose clip on the nose. After

3 seconds the spirometer was set. The pipe of the spirometer was then placed in the mouth and the patient breathed normally several times, then he/she exhaled maximally, inhaled maximally, and exhaled rapidly and vigorously. This procedure was repeated three times in the same order, and each patient stood in an upright position. Results were printed on paper suitable for the spirometer.

These measurements were repeated in four oral conditions, with both total prostheses in the mouth, only the upper complete denture, only the lower complete denture, and without dentures in the mouth (as a control group).

## Results

The results obtained from the research set as aims for the realization of our paper, are shown in the following tables and graphs. In this study, we used the correlation coefficient and the Chi square test of independence with significance level p=0.05. The null hypotheses of this study states that complete removable dentures do not affect the respiratory capacity in their carriers.

The control group comprises patients without complete removable dentures.

This table shows the number of respondents and their average age.

**Table 1.** Average values of the respondents classified by gender

| GENDER | NUMBER OF PATIENTS | AGE |
|---|---|---|
| MALE | 4 | 73.25 |
| FEMALE | 4 | 74.30 |
| TOTAL/AVERAGE | 8 | 73.76 |

The following tables show the values obtained during the spirometry analysis in female patients

**Table 2.** Values of each parameter from the spirometry analysis at different examined oral conditions for the **first female patient**

| Spirometry (Spirometer parameter) | Condition in the oral cavity | | | |
|---|---|---|---|---|
| | With both prostheses | Without both prostheses | Without upper denture | Without lower denture |
| FVC | 3.04 | 3.40 | 3.28 | 3.32 |
| FEV1 | 2.96 | 3.12 | 3.00 | 3.08 |
| PEF | 4.10 | 3.63 | 3.45 | 3.86 |
| MMEF 25/75 | 3.27 | 3.33 | 2.78 | 3.31 |

**Table 3.** Values of each parameter from the spirometry analysis at different examined oral conditions in the **second female patient**

| Spirometry (Spirometer parameter) | Condition in the oral cavity | | | |
|---|---|---|---|---|
| | With both prostheses | Without both prostheses | Without upper denture | Without lower denture |
| FVC | 3.60 | 1.40 | 1.92 | 4.60 |
| FEV1 | 1.72 | 1.20 | 1.76 | 1.92 |
| PEF | 1.77 | 1.48 | 1.82 | 1.96 |
| MMEF 25/75 | 1.35 | 3.51 | 1.68 | 1.44 |

**Table 4.** Values of each parameter of the spirometry analysis at different examined oral conditions in the **third female patient**

| Spirometry (Spirometer parameter) | Condition in the oral cavity | | | |
|---|---|---|---|---|
| | With both prostheses | Without both prostheses | Without upper denture | Without lower denture |
| FVC | 1.84 | 1.96 | 1.72 | 1.88 |
| FEV1 | 1.60 | 1.76 | 1.52 | 1.64 |
| PEF | 1.76 | 2.01 | 1.84 | 1.85 |
| MMEF 25/75 | 1.39 | 1.55 | 1.31 | 1.38 |

**Table 5.** Values of each parameter from the spirometry analysis at different examined oral conditions for the **fourth female patient**

| Spirometry (Spirometer parameter) | Condition in the oral cavity | | | |
|---|---|---|---|---|
| | With both prostheses | Without both prostheses | Without upper denture | Without lower denture |
| FVC | 4.88 | 1.44 | 2.12 | 2.56 |
| FEV1 | 2.60 | 1.22 | 1.79 | 2.48 |
| PEF | 3.60 | 2.39 | 3.08 | 3.01 |
| MMEF 25/75 | 0.73 | 3.76 | 2.68 | 2.70 |

The following tables show the values obtained during spirometry analysis in male patients.

**Table 6.** Values of each parameter from the spirometry analysis at different examined oral conditions for the **first male patient**

| Spirometry (Spirometer parameter) | Condition in the oral cavity | | | |
|---|---|---|---|---|
| | With both prostheses | Without both prostheses | Without upper denture | Without lower denture |
| FVC | 3.52 | 4.36 | 3.80 | 3.32 |
| FEV1 | 3.32 | 4.48 | 3.60 | 3.28 |
| PEF | 3.85 | 7.29 | 5.59 | 5.98 |
| MMEF 25/75 | 2.90 | 6.83 | 4.68 | 3.74 |

**Table 7.** Values of each parameter from the spirometry analysis at different examined oral conditions for the **second male patient**

| Spirometry (Spirometer parameter) | Condition in the oral cavity | | | |
|---|---|---|---|---|
| | With both prostheses | Without both prostheses | Without upper denture | Without lower denture |
| FVC | 4.00 | 3.04 | 3.96 | 4.04 |
| FEV1 | 3.44 | 2.16 | 2.88 | 2.72 |
| PEF | 3.67 | 2.32 | 3.09 | 3.08 |
| MMEF 25/75 | 3.18 | 2.72 | 2.46 | 2.72 |

**Table 8.** Values of each parameter from the spirometry analysis at different examined oral conditions for the **third male patient.**

| Spirometry (Spirometer parameter) | Condition in the oral cavity | | | |
|---|---|---|---|---|
| | With both prostheses | Without both prostheses | Without upper denture | Without lower denture |
| FVC | 2.40 | 2.16 | 2.48 | 2.32 |
| FEV1 | 2.32 | 2.12 | 2.44 | 2.28 |
| PEF | 3.26 | 3.81 | 3.57 | 3.22 |
| MMEF 25/75 | 1.87 | 1.81 | 2.40 | 1.56 |

**Table 9.** Values of each parameter from the spirometry analysis at various examined oral conditions for the **fourth male patient**

| Spirometry (Spirometer parameter) | Condition in the oral cavity | | | |
|---|---|---|---|---|
| | With both prostheses | Without both prostheses | Without upper denture | Without lower denture |
| FVC | 2.32 | 1.32 | 2.08 | 2.68 |
| FEV1 | 2.08 | 1.98 | 2.04 | 2.52 |
| PEF | 2.20 | 2.25 | 2.48 | 3.67 |
| MMEF 25/75 | 1.82 | 8.32 | 2.24 | 3.15 |

**Table 10.** Average values of spirometry parameters for the male respondents

| Spirometry (Spirometer parameter) | Average spirometric analysis in the appropriate oral cavity | | | |
|---|---|---|---|---|
| | With both prostheses | Without both prostheses | Without upper denture | Without lower denture |
| FVC | 3.06 | 2.72 | 3.08 | 3.09 |
| FEV1 | 2.79 | 2.69 | 2.74 | 2.70 |
| PEF | 3.25 | 3.92 | 3.68 | 3.99 |
| MMEF 25/75 | 2.44 | 4.92 | 2.95 | 2.79 |

**Table 11.** Average values of spirometry parameters for the female respondents

| Spirometry (Spirometer parameter) | Average spirometric analysis in the appropriate oral cavity | | | |
|---|---|---|---|---|
| | With both prostheses | Without both prostheses | Without upper denture | Without lower denture |
| FVC | 3.34 | 2.05 | 2.26 | 3.09 |
| FEV1 | 2.22 | 1.83 | 2.02 | 2.28 |
| PEF | 2.81 | 2.38 | 2.55 | 2.67 |
| MMEF 25/75 | 1.69 | 3.04 | 2.11 | 2.21 |

The following tables show the results obtained from our analysis.

**Table 12.** - for FVC (measurement of lung size (in liters) which is the air volume in the lungs that can be exhaled after previous deep inhalation).

| | Number of patients | Min | Max | Arithmetic mean | Median | Variance | Standard Deviation | | Correlation Coefficient |
|---|---|---|---|---|---|---|---|---|---|
| Without dentures (control group) | 8 | 1.32 | 4.36 | 2.46 | 2.46 | 0.75 | 0.87 | | |
| With dentures | 8 | 1.84 | 4.88 | 3.16 | 3.48 | 0.72 | 0.85 | 0.15 | 0.21 |
| With lower denture | 8 | 1.72 | 3.96 | 2.63 | 2.56 | 0.53 | 0.73 | 0.61 | 0.97 |
| With upper denture | 8 | 1.88 | 4.60 | 3.25 | 3.43 | 0.66 | 0.81 | 0.28 | 0.39 |

When the correlation coefficient is less than ("<") 0.5, it is interpreted that the data measured for the control group compared to the measured data for each of the other three cases (with prostheses, lower prosthesis, upper prosthesis) are poorly correlated.

If the correlation coefficient is greater than or equal to ("≥") at 0.5, the data from the control group compared to the data from each of the three cases separately (with prostheses, lower prosthesis, upper prosthesis) are statistically significantly correlated.

If the correlation coefficient is equal to ("=") to zero ("0"), the measured data from the control group are not in correlation with the measured data for each of the three cases in the oral cavity separately.

Table 12 shows that the correlation coefficient is low in the presence of the two prostheses in the oral cavity and the presence of the upper total prosthesis only, which indicates that their connection is very weak, almost insignificant and has no effect on respiratory capacity when the parameter FVC is considered. In the cases when only the lower prosthesis is present, the correlation coefficient indicates a statistically significant connection, which can be interpreted that the presence of the lower prosthesis in the oral cavity when performing spirometry analysis for the FVC parameter is associated with the respiratory capacity but does not necessarily mean that it has a direct impact on respiratory capacity.

The results of the arithmetic mean and the median, table 12, and the proximity of their values, indicate symmetry of the distribution of measured data for each state for the FVC parameter. The data measured for FVC are close to symmetrical distribution given the fairly close values of median and arithmetic mean and the data are symmetrically distributed in the absence of the complete removable dentures from the oral cavity.

The values of the arithmetic mean and the median, table 13, with the closeness of their values indicate symmetry of data distribution, which allows for them to be easily processed and facilitates the connection analysis. Data measured for the FEV1 parameter are precisely symmetrically distributed with removed dentures from the oral cavity and with only lower dentures in the respondents. For the other two oral conditions, data is quite close to their symmetry.

For the FEV1 parameter (table 13), the correlation coefficient demonstrates that there is a statistically significant relationship between the condition with complete removable dentures in the oral cavity and the condition without dentures in the oral cavity. It can be noted that each condition in the oral cavity (with dentures, only the upper or only the lower removable denture) is related to the respiratory capacity, but does not necessarily mean that they directly affect respiration regarding the FEV1 parameter.

The correlation coefficient (table 14) points out that there is a statistically significant connection between the examined data regarding the PEF parameter and each of the conditions in the oral cavity (with prostheses, only with upper or only with lower denture) is significant when associated with the respiratory capacity, but we cannot conclude that it has a direct impact on it.

Regarding the values of the arithmetic mean and the median from the table, the closeness of their values indicates symmetry of data distribution, i.e. data measured for the PEF parameter is precisely symmetrically distributed when there is only the upper and only the upper prosthesis in the oral cavity. For the other two oral conditions data is quite close to their symmetry.

Table 15 shows that the correlation coefficient is low, which leads to the conclusion that data are poorly relat-

**Table 13.** - FEV1 parameter (a measure of how much air can be exhaled in one second after previous deep inhalation).

| | Number of patients | Min | Max | Arithmetic mean | Median | Variance | Standard Deviation | | Correlation Coefficient |
|---|---|---|---|---|---|---|---|---|---|
| **Without dentures (control group)** | 8 | 1.20 | 4.48 | 2.37 | 2.37 | 0.72 | 0.85 | | |
| **With dentures** | 8 | 1.60 | 3.44 | 2.50 | 2.62 | 0.31 | 0.56 | 0.32 | 0.67 |
| **With lower denture** | 8 | 1.52 | 3.60 | 2.32 | 2.32 | 0.38 | 0.61 | 0.47 | 0.89 |
| **With upper denture** | 8 | 1.64 | 3.28 | 2.50 | 2.66 | 0.22 | 0.47 | 0.32 | 0.79 |

**Table 14.** - PEF parameter (individual maximum air exhalation speed. Typically this is measured in liters per minute (L/min).

| | Number of patients | Min | Max | Arithmetic mean | Median | Variance | Standard Deviation | | Correlation Coefficient |
|---|---|---|---|---|---|---|---|---|---|
| **Without dentures (control group)** | 8 | 1.48 | 7.29 | 3.06 | 2.73 | 2.14 | 1.46 | | |
| **With dentures** | 8 | 1.76 | 4.10 | 3.10 | 3.45 | 0.73 | 0.86 | 0.84 | 0.67 |
| **With lower denture** | 8 | 1.82 | 5.59 | 3.11 | 3.11 | 0.77 | 0.88 | 1.16 | 0.90 |
| **With upper denture** | 8 | 1.85 | 5.98 | 3.37 | 3.37 | 0.83 | 0.91 | 1.16 | 0.87 |

**Table 15.** - MMEF 25/75 parameter (medium forced expiratory flow between 25% and 75% of FVC, which in turn is interpreted as a quantitative measure of small obstructions (<2 mm) in the airways. It can be given in discrete periods, generally defined with how much of the FVC is exhaled)

| | Number of patients | Min | Max | Arithmetic mean | Median | Variance | Standard Deviation | | Correlation Coefficient |
|---|---|---|---|---|---|---|---|---|---|
| **Without dentures (control group)** | 8 | 1.55 | 8.32 | 5.84 | 4.09 | 7.30 | 2.70 | | |
| **With dentures** | 8 | 0.73 | 3.27 | 2.05 | 2.06 | 1.37 | 1.17 | 1.46 | 0.46 |
| **With lower denture** | 8 | 1.31 | 4.68 | 2.57 | 2.57 | 0.54 | 0.74 | 0.48 | 0.24 |
| **With upper denture** | 8 | 1.38 | 3.74 | 2.55 | 2.85 | 0.54 | 0.73 | 0.99 | 0.50 |

ed to each other. Concerning the MMEF 25/75 parameter, none of the different conditions in the oral cavity (with prostheses, only the upper or only the lower denture) has an effect on respiratory capacity.

Regarding the values of the arithmetic mean and the median, (table 15), the closeness of their values indicates the symmetry of data distribution, i.e. data measured for the MMEF 25/75 parameter is precisely symmetrically distributed when there is only the lower prosthesis and when the dentures are present in the oral cavity. For the other two oral conditions, data is quite close to their symmetry.

To show whether oral conditions have an impact or not on respiratory capacity, a zero hypothesis was formulated and tested using the Chi square test of independence. The value d is the result obtained for the x2 test when comparing the control group with each of the three conditions in the oral cavity for each of the four parameters. It is then compared to the value of the Chi square test (24.996). In all cases the value $d \leq 24.996$ from which we draw the conclusion that the tested null hypothesis is accepted, i.e. oral conditions do not affect the respiratory capacity of their carriers for each of the four parameters.

We selected the level of significance ourselves and it can receive a value in the range of 0-100%. We selected our level of significance to be 0.05. In practice, this means that there is a 5% probability that a connection

**Table 16.** - Zero hypothesis tested with X2 independence test

| | FVC | $FEV_1$ | PEF | MMEF 25/75 | Degree of freedom | Significance level α= | The critical value of the test |
|---|---|---|---|---|---|---|---|
| | Result of the statistical test d | | | | | | |
| **With both dentures** | 8.00 | 13.33 | 6.27 | 3.73 | 15 | 0.05 | ≤ 24.996 |
| **Only with lower denture** | 18 | 14.67 | 11.20 | 4.18 | 15 | 0.05 | |
| **Only with upper denture** | 10 | 12.44 | 10.08 | 2.89 | 15 | 0.05 | |

(impact) has been observed between the compared data, i.e. in our case there is a 5% probability that the effect of complete removable dentures on respiratory capacity will be observed. The critical value of the Chi quadratic test is likely to result from a statistical test if the null hypothesis is correct. In our case, the critical value is 24.996. If it is greater than the results obtained for the statistical test d ($d \leq 24.996$), then the null hypothesis is accepted. This means that the probability of the occurrence of such a result, d, for the statistical test is higher than the selected level of significance, i.e. $p > 0.05$.

Since all d values obtained for the statistical test are less than 24.996, we conclude that the hypothesis is accepted, i.e. oral conditions do not affect the respiratory capacity of their carriers for each of the four parameters.

## Discussion

Previous studies have shown that there is a strict relationship between the oropharyngeal condition and the upper respiratory tract[6-11]. However, until the end of the twentieth century, there were no clinical findings to assess respiratory function in a variety of dental conditions, such as for example partial or total tooth loss (edentulism). The most significant clinical records for the relationship between the oral condition and the respiratory function appeared only in the late 1990s.

Bucca et al.[12] reported that the Apnea-Hypopnea Index (AHI) presented almost doubled results during sleep in patients with complete tooth loss in contrast with those who did not wear dentures. Patients were 44 years old and they had obstructive sleep apnea (OSA) and chronic obstructive pulmonary disease (COPD). They wore complete removable dentures due to tooth loss and extraction. Cephalometric analysis of the patients revealed a significant narrowing of the antero-posterior oropharyngeal distance from 1.5 to 0.6 cm. After these significant findings, they expanded their studies to six male patients with OSA, and the authors noted that removing the complete prostheses significantly reduced the retropharyngeal space and that sleeping with prostheses removed from the mouth was associated with lowering the middle and lower arterial blood saturation, as well as an increase in the AHI index[12]. The authors concluded that removing the complete prostheses significantly reduced the retropharyngeal distance in OSA patients during sleep, and they advised patients not to remove their total prostheses while sleeping to avoid the risk of upper airway collapse[12, 13].

Also, in one study aimed to assess the impact of complete removable prostheses on the AHI index in 34 OSA patients, Ariska et al.[14] found that wearing the complete removable dentures reduced the AHI index in 19 patients while increased the index in 8 patients during sleep. Interestingly, the improvement in the AHI index is not related to a reduction in sleep apnea results, but to a reduction in hypopnea results. In addition, there was no significant difference between different prosthetic situations (with prostheses and without prostheses in the mouth) regarding the mean and low $SpO_2$ index and the desaturation index.

The results of this study are consistent with our results showing no effect on reducing the airflow in patients with various prosthetic situations (with dentures and without dentures in the mouth).

In another study, Bucca et al.[15] performed spirometry tests on 76 edentulous patients, of whom 36 were asymptomatic, 22 had chronic obstructive pulmonary disease (COPD), and 18 had interstitial lung disease

(ILD). These tests were performed to determine the effect of total prostheses on respiratory function. In addition, they reported that in asymptomatic patients and ILD patients, lung performance improved slightly when total dentures were in the patients' mouths. The authors added that, so far, no significant differences have been found in COPD patients in terms of whether they wore dentures or not in their mouth. According to Bucca et al.[12], the values of maximum expiratory flow (MEF), forced inspiratory flow at 50% (FIF50), and forced expiratory flow at 50% (FEF50) increased in asymptomatic patients, while peak eccentricity flow (PEF) and forced expiratory flow (FEF50) increased in ILD patients. There was no significant difference in the forced vital capacity (FVC) and the forced expiratory volume of 1% (FEV1) in any group of patients.

Compared to this study, asymptomatic patients, such as those in our study, were found to have better results with the presence of dentures in the oral cavity, which did not correspond to our results where we found that dentures had no effect on respiratory capacity. In contrast to previous studies. Almeida et al.[16] performed a polysomnographic evaluation of 23 edentulous patients with OSA. They found that wearing complete removable dentures during sleep increased the AHI index in mild cases. The results of this study were consistent with those of Almeida et al. However, comparing the two tests, subjects should be considered to have different sleeping positions while performing polysomnography, as opposed to sitting upright in a standard spirometry test.

Based on previously considered studies, the highest average value was found in patients who performed measurements without prostheses in the oral cavity for each spirometry parameter. In addition, the lowest mean value was observed in patients who performed measurements with only the lower total prosthesis present in the oral cavity for FVC and FEV1. The lowest mean values for PEF were obtained in patients wearing only the lower complete removable prosthesis, while in patients with both prostheses in situ, the lowest mean values were obtained for FEF25-75. FEV1 and FVC values in patients who did not have the prostheses in their mouth, were significantly higher than those who wore both prostheses, and only the upper or only the lower prosthesis ($p < 0.05$).

The PEF value showed an insignificant difference between patients who did not wear dentures and patients who wore both dentures. However, values in patients who did not have prostheses in their mouths were significantly higher than those who wore only the upper prosthesis or only the lower prosthesis ($p < 0.05$).

In addition, values in patients who didn't wear total prostheses were significantly higher than in patients with only the upper prosthesis or only the lower prosthesis for the FEF 25-75 value ($p < 0.005$). There was no significant difference in patients who didn't wear any prostheses in the mouth and who wore only the lower total prosthesis ($p > 0.05$).

Comparison of spirometry values between no prostheses in the mouth and the other three different oral conditions with the presence of prostheses in the oral cavity showed that the greatest correlation was found between FVC values where there were no prostheses in the mouth and conditions where there was only a lower total prosthesis ($p < 0.001$), and between the values of FEV1 in conditions of absent total prostheses from the mouth and a condition where only the upper total prosthesis was present ($p < 0.001$). In all spirometry parameters, a high correlation was observed between patients without prostheses in the mouth and where both prostheses were present in the mouth, and also between patients with only the upper prosthesis present and the condition where only the lower prosthesis was in place. ($p < 0.001$, $r > 0.8$). All p values are lower than 0.001.

## Conclusion

In this paper, we examined the problem of respiratory function in patients - carriers of total prostheses. The research was done to see if complete removable dentures have an effect on airflow in the airways, i.e. if their use has an effect on the normal functioning of the respiratory tract.

Our results, as well as the research conducted by other authors, show that in some individuals there was an effect of the complete removable prosthesis on the results of spirometry analyzes, whereas in others there was none.

With this data, it cannot be confirmed with certainty whether patients should remove the prostheses during examinations and at night, because the tests did not show any changes, and in some tests it showed improvement in performing the spirometry with prostheses in the mouth. As a result of the limitations of this study, further examinations are needed with a representative sample of responders and extended examinations to validate current findings.

## Reference


1. M. Suvin. Bioloski temelji protetike – totalna proteza 1988; VII dopunjeno izdanje: 1 – 4
2. Chronic Obstructive Pulmonary Disease: National clinical guideline for management of chronic obstructive pulmonary disease in adults in primary and secondary care. Thorax 2004; 59 (Suppl. 1):1-232.

3. Miller MR, Hankinson J, Brusasco V, Burgos F, Casaburi R, Coates A, Crapo R, Enright P, van der Grinten CP, Gustafsson P, Jensen R, Johnson DC, MacIntyre N, McKay R, Navajas D, Pedersen OF, Pellegrino R, Viegi G, Wanger J. ATS/ERS Task Force. Standardisation of spirometry. Eur Respir J 2005; 26(2):319-38.
4. Quanjer PH, Tammeling GJ, Cotes JE, Pedersen OF, Peslin R, Yernault JC. Lung volumes and forced ventilatory flows. Report Working Party Standardization of Lung Function Tests, European Community for Steel and Coal. Official Statement of the European Respiratory Society. Eur Respir J 1993; 6 (Suppl. 16):5-40.
5. Hyatt RE, Scanlon PD, Nakamura M. Interpretation of Pulmonary Function Tests. A Practical Guide. Second Edition. Lippincot Philadelphia: Williams and Wilkins, 2003.
6. Solow B, Skov S, Ovesev J, et al. Airway dimensions and head posture in obstructive sleep apnea. Eur J Orthod 1996; 18:571 – 579.
7. Choi JK, Hur YK, Lee JM, et al. Effects of mandibular advancement on upper airway dimension and collapsibility in patients with obstructive sleep apnea using dynamic upper airway imaging during sleep. Oral Surg Oral Med Oral Pathol Oral Radiol Ednod 2010; 109:712 – 719.
8. Lowe AA, Gionhaku N, Takeuchi K, et al. Three dimensional CT reconstructions of tongue and airway in adult subjects with obstructive sleep apnea. Am j Orthod Dentofacial Orthop 1986; 90:364 – 374.
9. Clark GT, Arand D, Chung E, et al. Effect of anterior mandibular positioning on obstructive sleep apnea. Am Rev Respir Dis 1003; 147:624 – 629.
10. Muto T, Yamazaki A, Takeda S. A cephalometric evaluation of the pharyngeal airway space in patients with mandibular retrognathia and prognathia, and normal subjects. Int J Oral Maxillofac Surg 2008; 37:228 – 231.
11. Martin O, Muelas L, Vinasa MJ. Nasopharyngeal cephalometric study of ideal occlusions. Am J Orhod Dentofacial Orghop 2006; 130:436. El-436. e9.
12. Bucca C, Carossa S, Pivetti S, et al. Edentulism and worsening of obstructive sleep apnea. Lancet 1999; 353:121 – 122.
13. Bucca C, Cicolin A, Brussino L, et al. Tooth loss and obstructive sleep apnea. Respir Res 2006; 17:7 – 8.
14. Arisaka H, Sakuraba S. Tamaki K, et al. Effects of wearing complete dentures during sleep on the Apnea – hypopnea index. Int J Prosthodont 2009; 22:173 – 177.
15. Bucca C, Carossa S, Colagrande, et al. Effect of edentulism on spirometric tests. Am J respire Critr Care Med 2001; 162:1018 - 1020
16. Almeida FR, Furuayama RJ, Chaccur DC, et al. Complete denture wear during sleep in elderly sleep apnea patients-a preliminary study. Sleep Breath 2011; DOI: 10.1007/s11325-011-0587-9.