

# Temporomandibular Disorders, Non-Carious Cervical Lesions and Implant Failures: is Occlusion Guilty or Innocent?

<sup>1</sup>Cleide Gisele Ribeiro, <sup>2</sup>Jair Rodriguez Ivich, <sup>3</sup>Antonio Carlos Cardoso, <sup>4</sup>Cimara Fortes Ferreira

<sup>1</sup>Professor, Hospital Maternidade Therezinha de Jesus-HMTJ/JF and Suprema-Faculdade Ciencias Medicas e da Saude, Juiz de Fora, Minas Gerais, Brazil

<sup>2</sup>Private practice, Pegasso 3315 Col. La-Calma, Guadalajara, Lalisco, Mexico

<sup>3</sup>Professor of Prosthodontics of the Department of Stomatology of the University of Santa Catarina, Florianopolis, Santa Catarina, Brazil

<sup>4</sup>Associate Professor, Director of Implant Dentistry, Department of Periodontics, University of Tennessee School of Dentistry, Memphis, TN, USA

Received: September 07, 2020; Accepted: September 14, 2020; Published: September 17, 2020

\*Corresponding author: Cimara Fortes Ferreira, DDS, MSc, PhD, MDS, Associate Professor in Periodontology, Director of Implant Dentistry, University of Tennessee Health Science Center College of Dentistry, Dunn Dental Building – 5th floor S503, 875 Union Ave - Memphis TN 38163, Tel. No: 901/448-4494; Fax. No: 901/448-1294; E-mail: cimarafortes@hotmail.com

## Abstract

We are always looking for the cause of these failures to justify results that aren't always predictable. Alternatively, everyone pivots and blames occlusion as the root cause of the innumerable failures and pathologies such as Temporomandibular Disorders (TMD), Non-Carious Cervical Lesions (NCCLs) and dental implant failures. In this literature review relevant published studies were searched for in PubMed, from 2015 to 2020. It was concluded that TMD and NCCLs are multifactorial diseases, of which occlusion is not a causative factor; in addition to the biological failures of dental implants. However, regarding the mechanical failures of dental implants, occlusion can be considered a causative factor.

**Keywords:** Occlusion, Temporomandibular Disorders, Non-Carious Cervical Lesions, Implant-Supported Prosthesis Failures

## Introduction

In dentistry, there isn't a field so discussed, debated, interpreted, "conceptualized", loved, and hated as occlusion. Occlusion has been linked to adjectives; such as complex, difficult, boring, and confusing. It is also possible that there isn't another field in dentistry with so many philosophies, as occlusion. There were times where it was of much interest, and times of little interest; times where it was much taught, and times where it was less taught. Further, there isn't a field in dentistry that has received more blame for failures or unsuccessful restorative work, from full-arch rehabilitations to single-unit restorations, than occlusion.

This disorientation could have been caused by factors, such as the growing diversity of concepts in occlusion transforming practical procedures into theoretical concepts; changes in the definitions of important occlusal-related terms; insufficient education about occlusal principles; as well as, diversity in concepts used in the same college by different professors. The coexistence of various and conflicting occlusion philosophies and disagreement regarding basic occlusion terminologies have

created confusion around this topic[1]. Another issue is the variability in methods used to teach occlusion in dental schools. Occlusion can be taught within multiple disciplines rather than as a stand-alone subject. The risk with this fragmented approach is that occlusion may be taught from a "biased viewpoint of one discipline or more confusingly, been given dogmatically by multiple departments with conflicting philosophies"[2-4].

The glossary of prosthodontic terms defines occlusion as the "act or process of closure, being closed or shut off, and the static relationship between the incising or masticating surfaces of the maxillary or mandibular teeth or tooth analogs"[5]. Dental occlusion is considered the core of Dentistry[1]. Nevertheless, occlusion is a concept that requires reflective critical thinking and problem-solving skills to understand its concepts, rather than the "psychomotor skills of operative dentistry". This lack of coordination may adversely affect students' perception of occlusion if students are not exposed to the topic in an integrated conceptual manner. The variability in teaching delivery methods may be mitigated if occlusion is taught within a single department that would receive input from other clinical and basic science faculty. This research reaffirms the need for a dedicated key

faculty to oversee the coordinated delivery of this subject[4].

Occlusion is considered one of the main factors for temporomandibular disorders (TMD), Non-Carious Cervical Lesions (NCCLs), conventional restorative and dental implant failures. However, can occlusion be blamed for these failures if four teeth only occlude momentarily during swallowing and occasionally during mastication? Besides, most of the time our teeth should be separated while in the mandibular rest position[6]. Some occlusal alterations, such as; absence of anterior guidance, absence of canine guidance, vertical trespass, premature contact in Centric Relation (CR), partial edentulism, altered Occlusal Vertical Dimension (OVD), are capable of causing restorative failures or tooth fractures? After all, is occlusion guilty or innocent?

Hence, it is worthwhile to recall Becker and Kaiser's[7] patient-friendly statement that "it is presumptuous to state nature's intent for an ideal occlusion, and therefore it is recommended to avoid occlusal therapy for individuals who appear to be functioning in health, even if their occlusal scheme does not fit a concept of optimum occlusion." The rare occurrence of a so-called "perfect" or "ideal" occlusion in natural dentitions indicates that "nature does not require such perfection"[8]. About 70% of North American youths have some form of malocclusion, mostly crowding of teeth within an Angle class I occlusion[9]. Because of the large prevalence of "mal" occlusions, most of which are not bad at all[10], it has recently been suggested to eliminate the term "malocclusion" from the dental literature.

It is important to note that a particular occlusal scheme is not a predictor of disease and that there is a significant overlap of occlusal features between individuals with and without jaw disorders[11]. Due to great controversies in the theme of occlusion, the purpose of this review was to answer the following clinical question: Is occlusion guilty or innocent of TMD, NCCLs, and failure of conventional and Implant-Supported Prostheses (ISP)?

## Materials & Methods

### Search strategy

Relevant published studies were searched for in PubMed, from 2015 to 2020 using the following keywords: 1) TMD: occlusion is guilty or innocent? "TMD"+"occlusion" resulting in 42 results; 2) Non-carious cervical lesions (NCCLs) and Occlusion: guilty or innocent?; 3) "Non-carious cervical lesions"+"occlusion" = 9 articles; " Non-carious cervical lesions "; 4) "occlusion" resulting in 9 articles; Non-carious cervical lesions and occlusion resulting in 10 articles; and, "Non-carious cervical lesion causes" resulting in 26 articles; 5) Failure in implant dentistry: occlusion is guilty or innocent?; 6) "dental implants "+" occlusion" resulting in 158 articles; "occlusion and dental implants failure" resulting in 53 articles ; "dental prosthesis failures"+" occlusion" resulting in 63 articles.

## Results

Articles published in English in the last 5 years were used in this literature review. Full-text, Randomized Controlled Trials (RCTs), retrospective studies, prospective studies, cross-sectional, case series, and case reports involving human subjects and in vitro studies were searched. Articles included in this review addressed occlusion and malocclusion as possible causative factors for the researched pathologies. Articles that addressed treatment strategies were not included in this literature review.

Regarding occlusion being a causative factor for TMD, 42 articles were found, and after the criteria analysis was conducted, 23 articles remained. Regarding NCCLs and occlusion: guilty or innocent, 26 articles were found, however only 11 analyzed occlusion as a possible causative factor. Regarding failure in implant dentistry as innocent or guilty, there were 158 articles in total, but only 13 were used in this literature review.

### Temporomandibular disorder: is occlusion guilty or innocent?

The relationship between dental occlusion and TMD is still a controversial topic in dentistry. TMD research focused on single-factor explanations of TMD, such as the TMJ, muscles, or dental occlusion; however, its findings showed little supporting evidence[12]. TMD is not caused by a single factor but by several factors, including psychological factors, occlusal factors, traumas, and factors related to joint pathology, deep pain input, and parafunction, can either cause TMD, increase the risk of TMD, impair healing, or enhance the progression of pre-existing TMD[13]. The etiology of TMD has been considered one of the most controversial issues in clinical dentistry.

Although occlusion is often considered one of the causative factors of TMD, the relationship between TMD and malocclusion is an extremely critical issue in Dentistry. Which types of malocclusion could be related to TMD? A study suggested that Angle's class II and class III malocclusion are associated with TMD[14]. Nevertheless, a study showed that patients that needed to undergo orthodontics and orthognathic surgery to correct their malocclusion had a significant incidence of TMDs when compared to a control population. However, after treatment, the incidence of TMDs did not differ from that of a control population. The reasons for these findings are not clear[15].

Which are the occlusal alterations already considered causative factors for TMD? Occlusal anomalies such as anterior open bite, unilateral posterior crossbite, horizontal trespass, absence of 5 or more posterior teeth, and distance from Centric Relation (CR) to maximum intercuspation (MI) discrepancy being greater than 2 mm[16-19]. Another study found significantly high occlusion time, right lateral disclusion time, left lateral disclusion time, and protrusion disclusion time in participants with pain-related TMD, with headaches, and in those presenting intra-articular joint disorders as compared to the control group[20]. To reduce the

incidence of deflective occlusal contacts, reflex contraction of masticatory muscles can occur, leading to altered mandibular position and to the prolongation of occlusion time[21]. The reason disclusion time in various excursive movements increases in participants with TMD remains unknown.

Next, does the increase in the OVD lead to TMD? Does decreasing the OVD lead to TMD? Can the stomatognathic system adapt to changes in OVD? In conclusion, much of the concepts regarding OVD and TMD are unfounded by scientific evidence. Traditional beliefs have been based on case reports and anecdotal opinions rather than on well-controlled clinical trials. The available evidence is weak and seems to indicate that the stomatognathic system can adapt rapidly to moderate changes in OVD. Nevertheless, it should be taken into consideration that in some patients it may show mild transient symptoms, but these are most often self-limiting and without major consequences. In conclusion, there is no indication that permanent alteration of the OVD will produce long-lasting TMD symptoms[22]. Another study evaluated the correlation between a reduced dental arch and TMD. They concluded that the lack of posterior tooth support does not cause the progression of TMD[23].

An alleged causal relationship between “malocclusion” and TMDs has been proposed. Therefore, diseased individuals should have a higher frequency of the purported causal factor than its absence as being the basic pre-requisite to broaden the depth of an assessment of causal hypothesis[24]. Studies support the absence of consistent, clinically relevant associations between TMD and the various features of dental occlusion[12, 23, 25-33]. Based on these results, irreversible occlusal changes of prosthodontic or orthodontic rehabilitation cannot be recommended for the management or even the prevention of such TMD.

This literature review showed that occlusion is innocent as a possible causative factor of TMD. Such observation should ideally lead to an end of the so-called “gnathological era”[24] of aetiological thinking in the TMD field, in which normal variability in the interindividual features of dental occlusion has been considered a pathological sign. Dental clinicians are thus encouraged to move forward and abandon the old-fashioned gnathological paradigm.

#### **Non-carious cervical lesions (NCCLs): isocclusion guilty or innocent?**

NCCLs are the result of a pathological condition characterized by the loss of tooth structure at the Cementoenamel Junction (CEJ) that is unrelated to dental caries. The prevalence of NCCLs increases with age, most certainly because older people are exposed to the etiological factors for longer periods when compared to the younger ones[34,35]. The worldwide prevalence of NCCLs among adults is 46.7%[35].

Of all possible etiological factors for NCCLs, occlusal stress forces have received maximum attention over the years.

Nevertheless, the correlation between heavy occlusal forces and the progression of NCCLs is controversial, in that some studies state that stress concentration in the cervical areas of the tooth contributes to their occurrence[36-38], however, it is never a single factor responsible for the occurrence and progression of NCCLs[34,39]. Another study that evaluated only the progression of the NCCLs, concluded that perhaps factors other than occlusal forces are more important in initiating NCCLs; nevertheless, occlusal forces are responsible for their progression[40]. The present conclusion concurs with studies that show that occlusal wear, or wear facets without NCCLs, did not indicate a direct association with occlusal discrepancies[40,41].

Why, despite many efforts to demonstrate that occlusal forces are the main cause of NCCLs, its etiology remains poorly understood or controversial? Because NCCLs may have several etiological factors[36,39,42,43]. Some causative agents are associated with NCCLs, such as; the use of a hard toothbrush bristles, the horizontal brushing technique[41,44]; dynamic occlusal parameters, left lateral disocclusion time, right lateral disocclusion time,[44] in addition to a thinner buccal plate and excessive lateral load during mandibular excursive movements, favoring the pre-molars to be the teeth with higher prevalence to NCCLs. These may lead to higher flexion of the tooth to the buccal direction, amplifying deformations in the cervical region[34]. However, the few clinical studies available were not able to confirm a positive association between occlusal loading and NCCLs[34,41].

It can be concluded that NCCL can be considered a disease, a lesion, or simply dental wear without specific etiology, or with multiple etiologies. It is almost impossible to blame occlusion as the etiological factor or even as an adjuvant in the development of this lesion. We all apply forces, may they be function or parafunction, the problem is labeling the disease with a specific etiology based on computerized studies, known worldwide as abfraction[45]. It is understood that occlusion is innocent for those reasons.

#### **Failures in implant dentistry: is occlusion guilty or innocent?**

Dental implants show a high success rate[46]. However, it is known that the conventional or dental implant restorative work fails in the short-, medium- and long-term. We need to understand why this happens and determine the etiology. We, clinicians, are not prepared for failures.

In the case of dental implants, failures can be divided into early and late[47]. The early failures occur before the implant is submitted to loading, and the late failures occur after the prosthesis is delivered. The late failure is frequently associated with traumatic occlusion[48]. Historically this condition is identified as “occlusal overloading”, to indicate that these forces surpass the adaptive capacity of the oral tissues. This definition

was recently reviewed as a “traumatic occlusal force” by the consensus report on the classification of periodontal and peri-implant diseases and conditions[49]. Can “traumatic occlusal forces” lead to peri-implantitis or peri-implant bone loss? Can “traumatic occlusal forces” lead to mechanical failures? How do we standardize the forces and define what is considered “traumatic occlusal forces” and how do we evaluate those in the clinical setting, since the opportunity to test such an intentional and standardized “traumatic occlusal force” in humans remains inappropriate and unethical?

It seems that the evidence of overloading dental implants leading to hard and/or soft tissue defects is very scarce[50,51]. There is evidence from observational studies that patients exhibiting poor plaque control and not attending regular periodontal maintenance visits are at higher risk of developing peri-implantitis, but overload alone, as the etiology of bone loss in humans, remains unclear[52]. Nevertheless, another study suggested that even when implant-prosthetic characteristics play a protective role; e.g. the use of screw-retained instead of a cement-retained prosthesis, there is a higher marginal bone loss observed under inappropriate occlusal patterns, such as inadequate anterior and canine guidances or inadequate occlusal contacts[53]. These findings are in agreement with previous clinical study[54], which reported significant differences in the marginal bone loss around implants of individuals with inadequate anterior guidance, non-working side contacts, and lateral group function involving teeth and implants. It is important to note that due to the lack of periodontal mechanoreceptor feedback, the control of the muscles of mastication is reduced and does not decrease the load applied to implant-supported restorations, resulting in greater stress on peri-implant crestal bone[53]. Other types of occlusal trauma, such as large cantilevers, parafunctional habits/bruxism, steep cusp inclines, poor distribution of force; e.g., limited contacts, interferences, may cause complications that range from biomechanical failures (screw loosening or screw fracture, porcelain chipping, cemented prosthesis debonding[55-57], to marginal bone loss or complete loss of the osseointegration of the implant[58]. A complete vertical trespass, a significant horizontal trespass or retroclination, increases the occlusal complexity, leading to the potential for adverse horizontal forces on abutment components. Thus, the clinician should keep implant occlusion in mind when placing or restoring an implant to protect the implant and surrounding peri-implant bone[58].

Another failure that can be caused by occlusion is implant fracture, which is rare[59]. To reduce the rate of implant failure, cantilever structures should be avoided, and large diameter implants should be used in the molar and premolar sites when possible. It is necessary to decrease the prosthetic buccolingual width of the crown, lower the inclination of the cusps, and place occlusal contact points close to the long axis of the implant fixture. Finally, passive adjustment of the prosthesis through the proper

path of insertion should be made[56]. In the present investigation, these data were recorded but not further reviewed. Rather, the effect of bruxism, the absence of a nightguard, and porcelain veneering material was analyzed and eventually associated with increased risk for chipping of the prosthetic material of the implant-supported fixed complete denture protheses[55].

It is important to understand that occlusion is a dynamic process. Even if we conduct an adequate occlusal adjustment, it may not be maintainable indefinitely. Even in adulthood, the 3-dimensional positioning of natural teeth in the dental arches is constantly changing as a consequence of continued slow tooth eruption and mesialization, which ranges from approximately 0.1 to 0.2 mm annually[60]. Dental implants cannot mimic the positional changes of the natural teeth because of the absence of the periodontal ligament. Due to the need for a light occlusion on dental implants, to compensate for the lack of periodontal ligament, the continuous eruption of the opposing teeth may play an important role in the subsequent changes in occlusal force distribution and occlusal trauma on the ISP. A study showed that the occlusion of ISP changed over the 3-year follow-up period, which was mainly reflected in the increasing occlusal force and occlusal contact time of the ISPs. The occlusion of ISPs should be carefully monitored during follow-up examinations, and occlusal adjustment should be considered when necessary[57].

It can be concluded from the studies evaluated in this section, that there isn't evidence that occlusion is guilty of the peri-implant hard and/or soft tissue defects, being, therefore, innocent. There is, however, evidence that traumatic occlusion can lead to mechanical failures, such as; prosthetic screw loosening, prosthetic screw fractures, porcelain chipping, crown debonding, and even implant fractures, being occlusion in these cases considered guilty.

## Conclusion

It is known that restorative work, from single-unit restorations to full arch reconstructions, may fail in the short-, medium- or long-term. Therefore, we are always looking for the cause of these failures to justify results that aren't always predictable. Dentists are not prepared for failures and always assume the cause of any problem must have been the result of their negligence, which the patient reinforces. Alternatively, everyone pivots and blames occlusion as the root cause of the innumerable failures or pathologies, such as TMD, NCCLs, and dental implants. It needs to be understood that DTM and NCCLs are multifactorial diseases, and that occlusion as a causative factor is considered innocent of these accusations. Regarding the biological failures of dental implants, occlusion is considered innocent. However, regarding the mechanical failures, occlusion can be considered guilty.

## References

1. Turp JC, Greene CS, Strub JR. Dental occlusion: a critical reflection on past, present and future concepts. *Journal of oral rehabilitation*. 2008;35(6):446-453.
2. Esposito CJ, Farman AG, Veal SJ. Occlusion in the dental curriculum: 1980-1981. *J Prosthet Dent*. 1983;49(3):419-426.
3. Lee DJ, Wiens JP, Ference J, Donatelli D, RM Smith, Dye BD, et al. Assessment of occlusion curriculum in predoctoral dental education: report from ACP Task Force on Occlusion Education. *J Prosthodont*. 2012;21(7):578-587.
4. O'Carroll E, Leung A, Fine PD, Boniface D, Louca C. The teaching of occlusion in undergraduate dental schools in the UK and Ireland. *Br Dent J*. 2019;227:512-517.
5. The glossary of prosthodontic terms. *J Prosthet Dent*. 2005;94:10-92.
6. CM. *Science And Practice Of Occlusion*: Quintessence Publishing Co., Inc. 1997.
7. Becker CM, Kaiser DA. Evolution of occlusion and occlusal instruments. *J Prosthodont*. 1993;2(1):33-43.
8. Ross B. Satisfactory occlusal relations for the individual with a craniofacial anomaly. *Int J Prosthodont*. 2003;18(4):284-287.
9. Bryant SR. The rationale for management of morphologic variations and nonphysiologic occlusion in the young dentition. *Int J Prosthodont*. 2003;16 Suppl:74-75:89-90.
10. Palla S. The interface of occlusion as a reflection of conflicts within prosthodontics. *Int J Prosthodont*. 2005;18(4):304-306.
11. Pullinger A. Establishing better biological models to understand occlusion. I: TM joint anatomic relationships. *Journal of oral rehabilitation*. 2013;40(4):296-318.
12. Ohrbach R, Dworkin SF. The Evolution of TMD Diagnosis: Past, Present, Future. *J Dent Res*. 2016;95(10):1093-1101.
13. List T, Jensen RH. Temporomandibular disorders: Old ideas and new concepts. *Cephalalgia*. 2017;37(7):692-704.
14. Bertoli FM, Bruzamolín CD, Kranz A, Losso EM, Brancher JA, Souza JF. Anxiety and malocclusion are associated with temporomandibular disorders in adolescents diagnosed by RDC/TMD. A cross-sectional study. *Journal of oral rehabilitation*. 2018;45(10):747-755.
15. Moraissi EA, Perez D, Ellis E 3rd. Do patients with malocclusion have a higher prevalence of temporomandibular disorders than controls both before and after orthognathic surgery? A systematic review and meta-analysis. *Journal of cranio-maxillo-facial surgery : official publication of the European Association for Cranio-Maxillo-Facial Surgery*. 2017;45(10):1716-1723.
16. Caldas W, Conti AC, Janson G, Conti PC. Occlusal changes secondary to temporomandibular joint conditions: a critical review and implications for clinical practice. *Journal of applied oral science*. 2016;24(4):411-419.
17. Liu X, Zhou KX, Yin NN, Zhang CK, Shi MH, Zhang HY, et al. Malocclusion Generates Anxiety-Like Behavior Through a Putative Lateral Habenula-Mesencephalic Trigeminal Nucleus Pathway. *Front Mol Neurosci*. 2019;12:174.
18. Nokar S, Sadighpour L, Shirzad H, Shahrokhi Rad A, Keshvad A. Evaluation of signs, symptoms, and occlusal factors among patients with temporomandibular disorders according to Helkimo index. *Cranio*. 2019;37(6):383-388.
19. Jussila P, Krooks L, Napankangas R, Pakkila J, Lähdesmäki R, Pertti P, et al. The role of occlusion in temporomandibular disorders (TMD) in the Northern Finland Birth Cohort (NFBC) 1966. *Cranio*. 2019;37(4):231-237.
20. Jivnani HM, Tripathi S, Shanker R, Singh BP, Agrawal KK, Singhal R. A Study to Determine the Prevalence of Temporomandibular Disorders in a Young Adult Population and its Association with Psychological and Functional Occlusal Parameters. *J Prosthodont*. 2019;28(1):e445-e449.
21. Baldini A, Nota A, Cozza P. The association between Occlusion Time and Temporomandibular Disorders. *J Electromyogr Kinesiol* 2015;25(1):151-154.
22. MorenoHay I, Okeson JP. Does altering the occlusal vertical dimension produce temporomandibular disorders? A literature review. *Journal of oral rehabilitation*. 2015;42(11):875-882.
23. Reissmann DR, Anderson GC, Heydecke G, Schiffman EL. Effect of Shortened Dental Arch on Temporomandibular Joint Intra-articular Disorders. *J Oral Facial Pain Headache*. 2018;32(3):329-337.
24. Manfredini D, Lombardo L, Siciliani G. Temporomandibular disorders and dental occlusion. A systematic review of association studies: end of an era? *Journal of oral rehabilitation*. 2017;44(11):908-923.
25. Manfredini D, Poggio CE. Prosthodontic planning in patients with temporomandibular disorders and/or bruxism: A systematic review. *J Prosthet Dent*. 2017;117(5):606-613.
26. Manfredini D, Perinetti G, Guarda-Nardini L. Dental malocclusion is not related to temporomandibular joint clicking: a

- logistic regression analysis in a patient population. *Angle Orthod.* 2014;84(5):310-315.
27. Shroff B. Malocclusion as a Cause for Temporomandibular Disorders and Orthodontics as a Treatment. *Oral Maxillofac Surg Clin North Am.* 2018;30(3):299-302.
28. Su N, Liu Y, Yang X, Shen J, Wang H. Association of malocclusion, self-reported bruxism and chewing-side preference with oral health-related quality of life in patients with temporomandibular joint osteoarthritis. *Int Dent J.* 2018;68(2):97-104.
29. Sousa ST, Mello VV, Magalhaes BG, Mariana PL, Vasconcelos, Farias Gomeset, al. The role of occlusal factors on the occurrence of temporomandibular disorders. *Cranio.* 2015;33(3):211-216.
30. Slade GD, Ohrbach R, Greenspan JD, RB Fillingim, E Bair, AE Sanders, et al. Painful Temporomandibular Disorder: Decade of Discovery from OPPERA Studies. *J Dent Res.* 2016;95(10):1084-1092.
31. Okeson JP. Evolution of occlusion and temporomandibular disorder in orthodontics: Past, present, and future. *American Journal of orthodontics and dentofacial orthopedics.* 2015;147(5 Suppl):S216-223.
32. Ferreira MC, Toledo PI, Dutra KL, FM Stefani, AL Porporatti, CF Mir, et al. Association between chewing dysfunctions and temporomandibular disorders: A systematic review. *Journal of oral rehabilitation.* 2018;45(10):819-835.
33. Banafa A, Suominen AL, Sipila K. Factors associated with signs of temporomandibular pain: an 11-year-follow-up study on Finnish adults. *Acta odontologica Scandinavica.* 2020;78(1):57-63.
34. Teixeira DNR, Zeola LF, Machado AC, Rafaella RG, Souza PG, Mendes DC, et al. Relationship between noncarious cervical lesions, cervical dentin hypersensitivity, gingival recession, and associated risk factors: A cross-sectional study. *J Dent.* 2018;76:93-97.
35. Teixeira DNR, Thomas RZ, Soares PV, Cune MS, Gresnigt MM, Slot DE. Prevalence of noncarious cervical lesions among adults: A systematic review. *J Dent.* 2020;95:103285.
36. Peumans M, Politano G, Van Meerbeek B. Treatment of noncarious cervical lesions: when, why, and how. *Int J Esthet Dent.* 2020;15:16-42.
37. Leal NMS, Silva JL, Benigno MIM, Bemerguy EA, Meira JBC, Ballester RY. How mechanical stresses modulate enamel demineralization in non-carious cervical lesions? *J Mech Behav Biomed Mater.* 2017;66:50-57.
38. Igarashi Y, Yoshida S, Kanazawa E. The prevalence and morphological types of non-carious cervical lesions (NCCL) in a contemporary sample of people. *Odontology.* 2017;105(4):443-452.
39. Arenal A, Menendez L, Gonzalez I, Riesgo JA, Velasco A, Lanchares H. Non-carious cervical lesions and risk factors: A case-control study. *Journal of oral rehabilitation.* 2019;46(1):65-75.
40. Sawlani K, Lawson NC, Burgess JO, Jack EL, Keith KE, Daniel AG, et al. Factors influencing the progression of noncarious cervical lesions: A 5-year prospective clinical evaluation. *J Prosthet Dent.* 2016;115(5):571-577.
41. Yang J, Cai D, Wang F, D He, L Ma, Y Jin, et al. Non-carious cervical lesions (NCCLs) in a random sampling community population and the association of NCCLs with occlusive wear. *Journal of oral rehabilitation.* 2016;43(12):960-966.
42. Nascimento MM, Dilbone DA, Pereira PN, Duarte WR, Geraldeli S, Delgado AJ. Abfraction lesions: etiology, diagnosis, and treatment options. *Clinical, cosmetic and investigational dentistry.* 2016;8:79-87.
43. Yoshizaki KT, Rios LF, Sobral MA, Aranha AC, Mendes FM, Scaramucci T. Clinical features and factors associated with non-carious cervical lesions and dentin hypersensitivity. *Journal of oral rehabilitation.* 2017;44(2):112-118.
44. Haralur SB, Alqahtani AS, AlMazni MS, Alqahtani MK. Association of Non-Carious Cervical Lesions with Oral Hygiene Habits and Dynamic Occlusal Parameters. *Diagnostics (Basel).* 2019;9(2):43.
45. Rees JS, Hammadeh M, Jagger DC. Abfraction lesion formation in maxillary incisors, canines and premolars: a finite element study. *European journal of oral sciences.* 2003;111(2):149-154.
46. Adler L, Buhlin K, Jansson L. Survival and complications: A 9- to 15-year retrospective follow-up of dental implant therapy. *Journal of oral rehabilitation.* 2020;47:67-77.
47. Esposito M, Hirsch JM, Lekholm U, Thomsen P. Biological factors contributing to failures of osseointegrated oral implants. (I). Success criteria and epidemiology. *European journal of oral sciences* 1998;106(1):527-551.
48. Quirynen M, Naert I, Steenberghe D. Fixture design and overload influence marginal bone loss and fixture success in the Branemark system. *Clin Oral Implants Res.* 1992;3(3):104-111.
49. Jepsen S, Caton JG, Albandar JM, Bissada F, Bouchard P, Cortellini P, et al. Periodontal manifestations of systemic diseases and developmental and acquired conditions: Consensus

- report of workgroup 3 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. *J Periodontol* 2018;89 Suppl 1:S237-S248.
50. Hammerle CHF, Tarnow D. The etiology of hard- and soft-tissue deficiencies at dental implants: A narrative review. *J Periodontol*. 2018;89 Suppl 1:S291-S303.
51. Wright SP, Hayden J, Lynd JA, J Willett, C Ucer, Speechley SD, et al. Factors affecting the complexity of dental implant restoration - what is the current evidence and guidance? *Br Dent J*. 2016;221(10):615-622.
52. Bertolini MM, Cury AA, Pizzoloto L, Acapa IRH, Shibli JA, Bordin D. Does traumatic occlusal forces lead to peri-implant bone loss? A systematic review. *Braz Oral Res*. 2019;33(suppl 1):e069.
53. Radaelli MTB, Federizzi L, Nascimento GG, Leite FRM, Boscato N. Early-predictors of marginal bone loss around morse taper connection implants loaded with single crowns: A prospective longitudinal study. *J Periodontal Res*. 2020;55(2):174-181.
54. Koller CD, Pereira-Cenci T, Boscato N. Parameters Associated with Marginal Bone Loss around Implant after Prosthetic Loading. *Braz Dent J*. 2016;27(3):292-297.
55. Papaspyridakos P, Bordin TB, Kim YJ, KE Rafie, Sarah E, Zuhair SN, et al. Technical Complications and Prosthesis Survival Rates with Implant-Supported Fixed Complete Dental Prostheses: A Retrospective Study with 1- to 12-Year Follow-Up. *J Prosthodont*. 2020;29(1):3-11.
56. Jacobsson M, Albrektsson T, Tjellstrom A. Tissue-integrated implants in children. *International journal of pediatric otorhinolaryngology*. 1992;24(3):235-243.
57. Luo Q, Ding Q, Zhang L, Xie Q. Analyzing the occlusion variation of single posterior implant-supported fixed prostheses by using the T-scan system: A prospective 3-year follow-up study. *J Prosthet Dent*. 2020;123(1):79-84.
58. Sheridan RA, Decker AM, Plonka AB, Wang HL. The Role of Occlusion in Implant Therapy: A Comprehensive Updated Review. *Implant Dent*. 2016;25(6):829-838.
59. Carneiro A, Dietrich L, Prudente MS, Silva JP, Jesus CP, Amaral CA, et al. Fracture Resistance of Internal Conical and External Hexagon: Regular and Narrow Implant-Abutment Assemblies. *Implant Dent*. 2016;25:510-514.
60. Heij DG, Opdebeeck H, Steenberghe D, Kokich VG, Belser U, Quirynen M. Facial development, continuous tooth eruption, and mesial drift as compromising factors for implant placement. *Int J Oral Maxillofac Implants*. 2006;21(6):867-878.