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Mandibular Distalisation for Tooth Wear in Patients Requiring Mandibular Protrusion for Sleep Apnoea

Keywords

OSA
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ABSTRACT

Background: Pathological tooth wear is an increasing concern and may require intervention and occlusal rehabilitation. Often the treatment includes distalisaton of the mandible to restore the dentition in centric relation. Obstructive sleep apnoea (OSA) is another condition treated my mandibular repositioning but in this case by an advancement appliance. The authors have a concern that there could be a group of patients with both conditions where distalisation for their tooth wear management would be contrary to their OSA treatment. This paper aims to look at this potential risk. Methods: A literature search was carried out using the following keywords (OSA or sleep apnoea or apnea or snoring or AHI or Epworth score) and for tooth surface loss (TSL or distalisation or centric relation or tooth wear or full mouth rehabilitation). Results: No studies were identified which considered the effect of mandibular distalisation on OSA. Conclusion: There is a theoretical risk that dental treatment involving distalisation may adversely affect patients at risk of OSA or worsening their condition due to the modification of airway patency. Further study is recommended.

INTRODUCTION

A recent epidemiology study was undertaken on 240 patients referred to the restorative department at King's College Hospital for the provision of a mandibular advancement appliance (MAA). The study revealed that twice as many male patients were being referred for treatment of mild to moderate sleep apnoea with a MAA than female patients and the overall mean age for referred patients was 52.[1]

A similar study investigating the epidemiology of tooth surface loss on 671 consecutive referrals to the same restorative department yielded similar results. The mean age for patients referred for assessment and treatment of their tooth surface loss was also 52 years of age. Furthermore the male to female ratio of patients was 1.7:1.[2]

Figure 1 depicts the age distribution of patients being referred to the restorative department at the Dental Hospital for OSA and TSL respectively. The number of patients in each age range is displayed above each bar.

This overlap in demographic indicates that there could be a subgroup of patients being referred to the restorative department at King's College Hospital who are potentially at risk of both tooth surface loss and OSA.

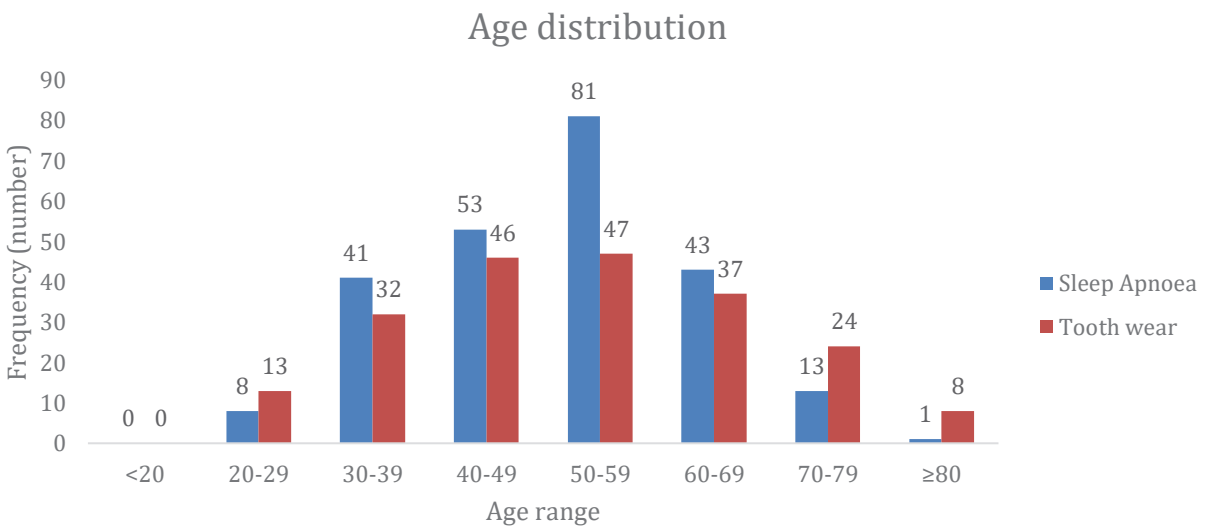

Figure 1: Age distribution of patients being referred for OSA and TSL treatment at Kings College Hospital.

TOOTH SURFACE LOSS

Tooth wear (TW) is the irreversible surface loss of dental hard tissues from causes other than dental caries, trauma or as a result of developmental disorders.[3] It is a physiological process with the normal vertical loss of enamel estimated to be between 20 and 38 µm per annum.[4]

TW becomes pathological when this process is excessive.[5] The thickness of enamel is reduced, exposing underlying dentine. This causes teeth to change colour from the white of the enamel to a yellow hue of the underlying dentine.[6] This in turn causes hypersensitivity and functional issues as the thin tooth structure starts to chip.

There are three universally accepted aetiologies for TW, namely erosion, attrition and abrasion.[6] Abrasion is a fourth aetiological factor, but its existence and aetiology are questioned.[7]

Erosion is the loss of dental hard tissue by chemical dissolution by nonbacterial acids from dietary or gastric sources.[8] Attrition is the loss of dental hard tissue due to tooth to tooth contact whilst abrasion is the loss of dental hard tissue due to tooth contact with exogenous substances.[9] TW aetiologies usually occur in combination with a dominant primary factor, which can make accurate diagnosis challenging.[10]

The most recent Adult Dental Health Survey reported that the prevalence of TW is increasing for all ages. It revealed that 77% of adults showed some tooth wear in their anterior teeth, 15% showed moderate wear and 2% showed severe.[11] Life expectancy is increasing and patients are maintaining their dentitions for longer, therefore the prevalence of tooth wear in the older population is increasing.[12]

Erosive TSL is also a problem in the younger population. Up to 1:3 young adults[18-35] exhibit signs of enamel wear such as incisal edge translucency and loss of surface anatomy.[13] Research points to the frequent consumption of extrinsic acid sources such as carbonated drinks and fruit juices as the primary etiological factor for this.[14]

The management of tooth wear should aim to protect the remaining tooth structure, control symptoms, stabilise the occlusion and restore aesthetics.[15]

OBSTRUCTIVE SLEEP APNOEA

Obstructive sleep apnoea (OSA) is the most common sleep related breathing disorder, characterised by recurrent episodes of partial or complete airway obstruction during sleep.[16] Airway obstruction leads to hypoxaemia and arousal from sleep.[17] OSA causes daytime sleepiness, neurocognitive dysfunction, impaired quality of life and increases the risk of mortality from a cerebrovascular incident.[18]

It is estimated that 1.5 million adults in the UK suffer from OSA but only 15% of cases are diagnosed.[19] OSA is therefore a common sleep disorder, especially in middle-aged men, with a male to female ratio of 2:1.[1] Obesity is a significant risk factor for OSA and a body mass index of over 25 kg/m² has a 93% sensitivity for OSA.[18]

The condition is diagnosed using overnight pulse oximetry or polysmography which measures a patient's Apnoea Hypopnea index (AHI) score. AHI measures the number of times a patient stops breathing per hour of sleep. Five episodes of apnoea an hour is diagnostic of mild OSA, 10-15 episodes per hour is diagnosed as moderate OSA and more than 15 episodes an hour being classified as severe OSA.[21] Patients can also be given a subjective questionnaire called the Epworth sleepiness scale (ESS) that measures the severity of daytime sleepiness.[22]

Continuous positive airway pressure (CPAP) is considered the gold standard treatment for OSA, however compliance is only around 50% due to side effects such as nose bleeds, sinusitis and problems sleeping due to the bulk of the appliance.[23] MAAs are often used in the management of mild and moderate cases of OSA. MAAs may be recommended in severe OSA where patients are unable to tolerate CPAP.[20]

MAAs function by bringing the mandible into a more anterior position (protrusion) with minimal increase in OVD. Typical advancement with a MAA should be between 50 and 70 percent of the patient's maximum mandibular protrusion depending on the severity of their OSA.[24] This repositioning opens the upper airway by bringing the tongue forwards preventing it from resting on the back of the throat and obstructing the airway.[25]

THE RELATIONSHIP BETWEEN TOOTH SURFACE LOSS AND OBSTRUCTIVE SLEEP APNOEA - SHOULD WE BE CONCERNED?

As patients wear away tooth tissue they often reduce and even remove their anterior guidance resulting in mandibular protrusion as they move towards an edge-to-edge position. This position is not considered ideal for restoring the dentition at an increased vertical dimension. Therefore centric relation is used to help determine a reproducible mandibular position to rehabilitate the patient given the lack of suitable ICP. The process of distalisation may well move the mandible posteriorly by several mm. Distalisation (moving the mandible into Centric Relation) can be achieved with a removable appliance or jig, indirect or direct restorative materials.[15]

The distilisation of the mandible will reduce the volume and patency of the upper airway, making airway obstruction by the soft palate more likely. The tongue's muscle attachments to the mandible mean it mirrors the mandibles movements. The posterior movement of the tongue increases its tendency to fall backwards, touching the back of the throat and obstructing the airway.[25]

We know that TW and OSA share a similar demographic of patients.[1,2] Theoretically some patients will be in both demographics and may receive mandibular distilisation into centric relation, which could adversely affect their OSA or place them at risk of developing OSA. Reducing airway patency could risk increasing a patient's AHI score, making them more prone to developing OSA or worsening existing OSA.

The aim of this paper is to investigate this potential risk and determine what recommendations there are in the literature regarding this anomaly in mandibular repositioning.

METHODS

A literature review was conducted on PubMed using the PubMed Advanced Search Builder. The following key terms were used for sleep apnoea (OSA or sleep apnoea or apnea or snoring or AHI or Epworth score) and for tooth surface loss (TSL or distalisation or centric relation or tooth wear or full mouth rehabilitation). Reference lists of the included articles were then hand searched to avoid missing relevant literature. All articles in the initial literature search were assessed using the following inclusion and exclusion criteria.

Table 1. Inclusion and exclusion criteria used for initial literature review paper selection.

Inclusion criteria	Exclusion criteria
Published in the last 20 years	Studies investigating Tooth wear or Sleep apnoea independently and not their relationship to each other
Published in English	Non-english language studies
Consideration of how mandible position could affect airway patency	Publication prior to 2002
Consideration of how mandible position could affect or change OSA score	Studies which did not consider airway patency or AHI score
Studies which considered the relationship between OSA and Tooth surface loss	

The literature review was conducted systematically, using the method depicted in Figure 2.

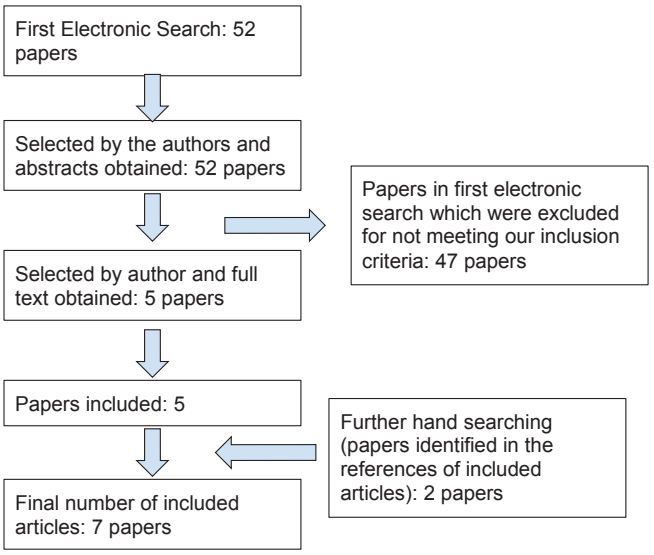

Figure 2: Search strategy used to perform the Pubmed literature review.

RESULTS

Studies meeting our inclusion criteria are summarised in Table 2. Overall seven papers were included in our study. Two papers were identified by the hand searching of the reference lists of papers selected from the literature search. The potential association between tooth wear and sleep disorders is reported in the literature, however we were not able to identify any publications that considered the impact of mandibular distalisation on AHI score. No papers considered the potential adverse effect of managing tooth wear with regard to OSA.

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Table 2. Included studies from the literature search.

Study	Year of Publication	Study design	Purpose of paper as stated in the abstract
We H, T T, M K. (27)	2022	Case report	How TMD and airway dimension may be associated
Hang W, Gelb M (30)	2016	Opinion article	Considering airway patency in TMD and orthodontic treatment
Durán-Cantolla J, Alkhraisat M, Martínez-Null C, Aguirre J, Guinea E, Anitua E (31)	2015	Prospective cohort study	Frequency of Obstructive Sleep Apnea in Tooth Wear patients
Allred R, Shaha D, Stanford (32)	2021	Blinded Observational study	Tooth wear in patients undergoing sleep studies
Wetselaar P, Manfredini D, Ahlberg J, Johansson A, Aarab G, Papagianni C *et al* (33)	2019	Narrative overview	The association between Tooth wear and Sleep disorders
Nikolopoulou M, Naeije M, Aarab G, Hamburger H, Visscher C, Lobbezoo F (28)	2011	Cohort study	The effect of raising the bite without protrusion on obstructive sleep apnoea
Gagnon Y, Mayer P, Morisson F (29)	2004	Pilot study	How occlusal splints may worsen an individual's OSA (measured by changes in AHI score)

DISCUSSION

Several papers of interest were identified during our literature search. One case report considered how increasing the vertical dimension with an occlusal splint when treating TMD could reduce airway volume and impact on the patient's risk of developing OSA. This was a case report of a single individual being treated for TMD and not TSL. The patient studied was also a 17 year old girl and did not fit the demographic of individuals at high risk of TSL and OSA who are more typically male aged 50 years of age.[1] Interestingly, the clinician opted against making this patient a splint in centric relation and instead opted for centric occlusion as this was a more anterior position which would have less impact on airway patency.[26]

One publication explored the possible indirect association between tooth surface loss and obstructive sleep apnoea syndrome.[30] A statistically significant positive correlation was found between AHI and tooth wear severity within their sample (as AHI severity increases so does the severity of tooth wear). These authors concluded that patients with TW had a high frequency of OSA and that tooth wear assessment could be a tool to identify patients at risk of OSA (30).[30] A positive correlation between the severity of OSA and sleep bruxism has been identified in the literature[31] where the authors believe patients with more severe OSA could have higher levels of night time parafunction which would accelerate their TW.

Two publications identified outside of the literature search from the reference lists of included papers investigated how temporarily increasing the vertical occlusal height (raising the bite) without mandibular protrusion when providing an occlusal splint for TMD could affect OSA. Although AHI scores were adversely affected in approximately half of cases, there was no statistically significant difference in AHI in either study.[27,28] In order to validify the accuracy of the findings of these papers, further randomised controlled trials need to be conducted.

Raising the bite using a removable splint is similar to increasing the occlusal vertical dimension in full mouth rehabilitation for the treatment of tooth surface loss as it involves the addition of material to teeth to replace tooth tissue that has been lost. We know from MAA studies that increasing the OVD without protrusion can increase AHI scores although not by a statistically significant level.[34] So increasing OVD in TW cases may also increase AHI scores. This aspect of TW treatment was not considered in this paper but may be a significant factor. In the authors' opinion the distalisation process is likely to have a greater adverse effect on OSA than the increase in OVD.

Furthermore, the results from the included studies were obtained with removable mandibular appliances and not fixed restorations which would be less bulky and may affect the patient's airway in a different way to a splint.

No studies have investigated the potential impact of distalising the mandible on AHI or OSA. The recognised treatment for mild and moderate cases of OSA is mandibular protrusion which is the opposite to the often-used distalisation of the mandible in prosthodontics prior to full mouth rehabilitation in the management of tooth wear. The potential risk remains unknown and requires further investigation.

After identifying an overlap in demographic for OSA and TW patients the authors suggest that it may be beneficial to screen patients at high risk of OSA prior to mandibular distilisation.

CONCLUSION

Clinicians need to consider the possible impact of restoring patients in centric relation on airway patency and OSA risk. TW rehabilitation in centric relation may need to be avoided in patients at risk of OSA as the potential morbidity associated with increased AHI scores may take precedence over restorative treatment.

Further research is required to investigate this potential association and to allow more objective conclusions and clinical relevance to be provided.

The authors declare no conflicts of interest.

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