ISSN 2689-8268 | Volume 8

American Journal of Surgery and Clinical Case Reports

Case Report Open Access

Temporomandibular Joint Versus Apnea A Cause-and-Effect Relationship! Clinical Case Report

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Received: 16 Dec 2024
Accepted: 31 Dec 2024
Published: 06 Jan 2025
J Short Name: AJSCCR

Citation:

Arnaldo Scarlati. Temporomandibular Joint Versus Apnea A Cause-and-Effect Relationship! Clinical Case Report. Ame J Surg Clin Case Rep. 2025; 8(4): 1-6

1. Abstract

Obstructive sleep apnea and hypopnea syndrome (OSAHS), in addition to the numerous associated comorbidities, greatly influences the quality of sleep in patients. Several episodes of total (apnea) or partial (hypopnea) interruption of airflow for at least 10 seconds make up this multifactorial sleep disorder, being the object of studies by Physicians, Dentists, Physiotherapists and Speech Therapists whose consensus is based on the application of surgical or conservative methods with the purpose of removing a probable obstruction that prevents the passage of air. This article presents a conservative protocol, supported by the physiology of breathing, swallowing, and maintenance of basal muscle tone, with promising results in relation to sleep quality. An intraoral device was used to maintain the spatial position of the TMJs in their respective cavities, so that there was no compression of the joint noble spaces. The protocol, lasting 3 months, proved to be efficient in improving sleep quality and associated symptoms. The 31-year-old female patient reported improvement in all complaints presented in the anamnesis.

2. Introduction

OSAHS is a chronic, progressive, disabling respiratory disorder with severe systemic repercussions characterized by recurrent collapse of the upper airways during sleep, as a consequence of posterior positioning of the tongue, resulting in a substantial reduction (hypopnea) at 30% of the flow along with a 4% desaturation of O2, or a 90% drop (apnea) of the airflow to the lungs for about 10 seconds, persisting or impeding respiratory efforts, according to the American Academy of Sleep Medicine (2020) [1-4]. OSAHS is said to be “central” when breathing ceases for at least 10 seconds, without abdominal thoracic movements, that is, there is no apparent central neurological command for breathing to occur; it is obstructive, with greater prevalence, when there is cessation of flow but with active abdominal thoracic movements, that is, with central respiratory commands with the flow interrupted by a probable obstruction of the upper airways (UA); mixed when central and obstructive occur concomitantly. [5, 6,3]. Another classification implies the number of events (minimum 10 seconds) per hour; MILD: 5-15 events, MODERATE: 16-30 events, SEVERE: more than 30 events/hour [7, 1]. Among the etiological factors, muscle hypotonicity (use of alcohol, relaxing drugs, sedentary lifestyle, aging and mouth breathing) stand out, as well as predisposing factors such as tissue hypertrophy, obesity, craniofacial deformities, etc.[5,8, 9,4]. The main signs and symptoms described include snoring, nocturnal movement, sleepwalking, morning headache, excessive tiredness, feeling of suffocation, irritation, depression, anxiety, choking, non-restorative sleep [2,3,4,5,9].

Polysomnography is considered the gold standard for assessing sleep architecture, in addition to providing the apnea-hypopnea index (AHI) by quantifying events per hour [8,10]. In addition to this, questionnaires such as the Epworth Index or the Pittsburg Sleep Quality Index also help in the qualification of the syndrome. Treatment with a CPAP (positive pressure) device is the technique of choice for severe cases because of its cost-effectiveness. They are effective in the short term, but have low adherence because they are relatively invasive and require a high level of cooperation from patients [5,11]. Intraoral appliances are used to control obstructive conditions, but they do not cure, in addition to presenting possible side effects in the short and long term [12]. These devices, the mandibular advancement devices (MAD) are widely used, with relative acceptance by patients, although they cause

short-term side effects such as hyper salivation, xerostomia, gingival discomfort, dental and temporomandibular joints or long-term discomfort such as occlusal changes, temporomandibular joint or muscular dysfunctions due to the maintenance of the non-physiological mandibular position [10-16].

3. Justification

In view of the high prevalence of unwanted side effects with the use of MAD, whose criterion for mandible advancement depends on a positive result in the biological response of the disorder to titration on the polysomnograph, we used a non-invasive, reversible alternative that seeks to act on basal muscle tone, and prevent interference in the control of breathing and swallowing [17]. Because it is a device with a reduced size, whose objective is to change the spatial relationship of the mandible heads within their respective cavities without necessarily a mandibular advancement, better adherence to treatment is expected with a great prospect of cure [18]. The AAS1 is an intraoral functional orthopedic appliance whose main function is to keep the mandibular heads in physiological spatial position, determine a balanced mandibular bio-mechanics in order to promote sensorimotor coherence at the cortical level. There are analog versions, printed in 3D resin or acetate. In this clinical case, the most cost-effective version (AAS1-ACT) was chosen.

4. Methodology

The 31-year-old female Caucasian patient presented several complaints in the anamnesis, such as:

- Non-restorative sleep, a quality attested by the Pittsburg questionnaire with a score of 16.

- TMD of the severe type according to the Fonseca index.

- Bruxism of the “clenching” type day and night.

- Persistent chronic headache along 4 years.

- Tingling and pain in the right arm.

- Restriction of cervical movements in flexion, hyperextension, and lateral rotation.

- No isometric activity of the mandible elevator muscles.

- Laterality movement of the mandible with restriction and non-reciprocal bilateral “clicking” occurring at the beginning of the opening.

A total tomography of the face was requested, in dental occlusion, as well as the preparation of 100% virtual gnatostatic models according to the Zampieri-Scarlati protocol, with reference to the horizontal plane of Camper. We found a spatial position of the mandible heads suggesting compression of the joint spaces [18]:

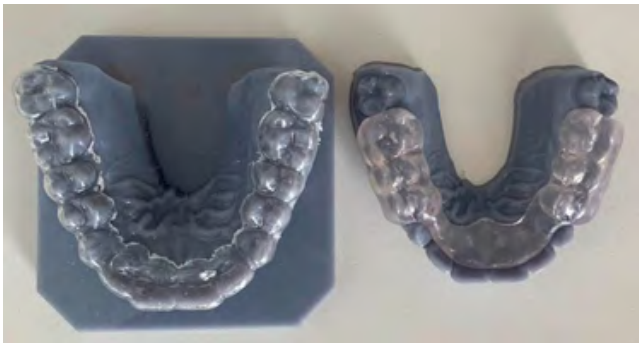
Right upper joint space = 3mm

Right posterior joint space = 3mm

Left upper joint space = 3mm

Left posterior joint space = 3mm

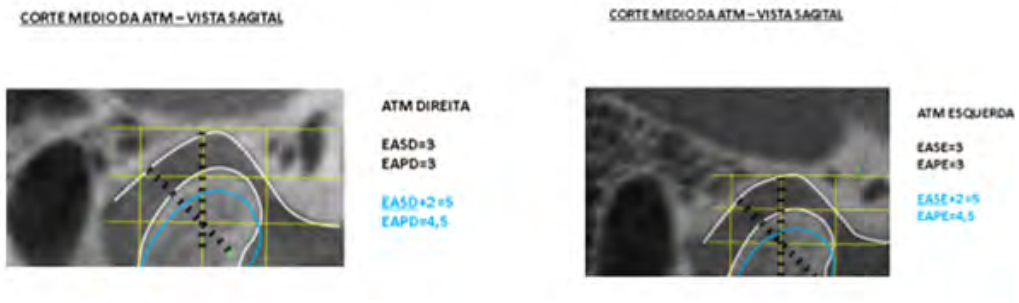
The new spatial ratio of the mandibular heads was calculated and the reverse therapeutic posture change (RTPC) maintained by the AAS1 device [18]. After assembling the models in the Gnatophore, the accessory of vertical adjustment of +2 mm and adjustment of the incisal pin at +1 mm (without the need for sagittal advancement) were used. The entire process, including the design for the production of the 3D printed model to obtain the AAS1-ACT (AAS1 version in thermo-processed acetate) was executed in the digital flow where the Meshmixer and Exocad software were used. The splint obtained was finished on a Gnatophore (joint device) that uses gnatostatic models and respects the laws of development of Planas, which shows positive results in relation to sleep quality [19]. The progressive use of the AAS1 for 3 months followed the physiotherapy protocol applied together with the device, which does not require any type of occlusal adjustment, except for those necessary for adaptation when appropriate [18]. It is worth noting that only the use of splint without applied physical therapy provides little effect on muscle response [20].



AAS1-ACT (Acetate) AAS1-RSN (3D Printed)

Figure 1: Inter-occlusal splint AAS1.

Figure 2: (a) Gnatostatic model showing the spatial position of the dental arches in maximum occlusion, sagittal views, related to Camper's horizontal plane.



(b) mid-sagittal TMJ slices with the positions of the mandible heads in maximum occlusion (white) and in the RTPC (blue).

Figure 3: Gnathostatic models with RTPC.

5. Results

In the fourth month of use, the new evaluation showed:

1 - Improved sleep quality with a reduction in the PSQI score from 16 to 1.

2 - TMD from SEVERE at the beginning of treatment to MILD according to the Fonseca index.

3 - Symptoms were controlled with elimination of headache, tingling and pain in the arm, in addition to bruxism.

4 – The mandibular movement was released and there was no clicking sound at the mouth opening.

5 - The patient reports restful sleep after the protocol.

6. Discussion

This case report opens new questions in the management of OSAS always associated with much controversy.

To what extent would a collapse in the soft tissues obstruct the passage of air from reaching the pulmonary alveoli? Paradoxically, would this collapse be resistant to the point of overcoming the force of the negative pressure generated by the contraction of the diaphragm, causing the pressure of 1 atmosphere of the external environment to balance as it diffuses in the environment of lower pressure?.

Would CPAP supposedly produce a positive pressure preventing the collapse in which the pressure of the environment that is hundreds of times greater has not been able to?.

A quick anatomical review of the pharyngeal region and a thorough analysis of the physiology of breathing, swallowing and maintenance of muscle tone, opens new perspectives for understanding the pathophysiology of the syndrome.

7. Anatomy

According to the orientation of the Sterling model (Figure 4.a), the passage of air through the pharynx runs through a "rigid" tube corresponding to the nasopharynx. Inside the box there is a flexible tube corresponding to the oropharynx and finally it passes through another "rigid" tube, the hypopharynx, before entering the trachea. According to the model, the probable site of obstructive collapse would occur in the oropharynx when exhaled air at high speed is subjected to the Venturi effect (negative pressure) that would be responsible for the collapse of tissues. Thanks to this phenomenon, it is possible to drain and clean all the paranasal sinuses. This effect is fleeting and occurs in lung expirations where rapidly, under normal conditions, the negative pressure is equalized with the external pressure. When we observe the layers of tissue that make up the tube corresponding to the pharynx, we have anatomically in its posterior and lateral aspect (Figure 4b):

1 - Laminate Ciliary Mucous Tissue Lining

2 - Pharyngobasilar fascia. It is formed of connective tissue, thick in the connection with the base of the skull that tapers to the hypopharyngeal region. In the central part of the structure, from the cranial base to the hypopharyngeal region, we have the pharyngeal raphe, which serves as the insertion of the pharyngeal constrictor muscles. This entire structure is also called the "framework" of the pharynx, suggesting a resistant structure.

3 - Next, we have the buccopharyngeal fascia in close contact with the cervical fascia. This connection is not rigid to allow movement of the pharyngeal tube, but the adhesion maintained by the negative pressure between the fascia prevents it from coming forward, since it is connected to the cervical spine. Every description casts doubt on the possibility of tissue collapse, at least of the lateral and posterior part of the oropharynx. The responsibility for the mechanical collapse, supposedly caused by the negative pressure after expiration, would have to be attributed to the anterior sector corresponding to the dorsum of the tongue and the soft palate. The analysis of this of the anterior sector of the pharynx has two readings: the first with the patient with the closed lip and the second with the mouth half-open with open lips. With the lips closed, after swallowing, an environment of sub-atmospheric pressure is created inside the mouth. The tongue is passively placed on the roof of the mouth and the soft palate, including the uvula, remains "glued" to the dorsum of the tongue until the next swallowing occurs, and so on. During this condition, the possibility of a pharyngeal collapse attributed to the anterior sector is practically nil. With the lip open, the pressure of the environment causes the tongue to remain theoretically free and the possibility of collapse would even be admissible, remembering that it would have to act against 16 muscles of the tongue plus the positive pressure of 1 atmosphere.

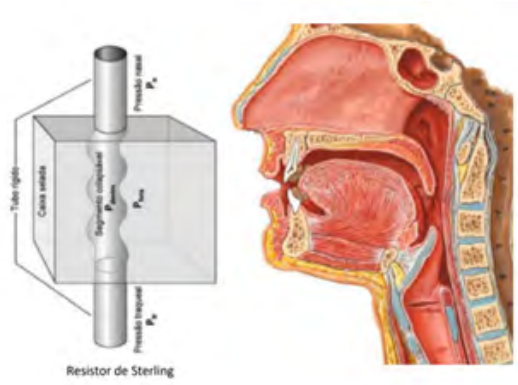


Figure 4: Sterling box and anatomy of the face.

8. Respiration

A respiratory event (apnea or hypopnea) will always occur after an exhalation, i.e., with an empty lung, when the current volume will theoretically have been completely expelled from the lungs. The Venturi effect, the result of the exhaled air that caused the "negative pressure" causing the collapse according to Colin Sulivan, inventor of CPAP, no longer exists, since the gaseous mixture of the ambient air, of higher pressure (1 atm) invades the space of the lower pressure and equalizes the space of the entire pharyngeal region in respect to the general law of gases. If a pressure of 8 to 15cm H2O (CPAP) is able to maintain positive pressure preventing a collapse, why wouldn't an atmospheric pressure of 1,033 cm H2O (1 ATM) do it?

Another aspect to be considered is the neural control in charge of the respiratory centers in the brainstem. The perfect synchrony of the neuromuscular system maintains the motor command over the diaphragm by the phrenic nerve; on the neck, intercostal, and abdominal muscles in charge of their respective nerves; and the trapezius and ECM muscles by the XI encephalic or accessory pair independently but simultaneously and synchronously, depending on the respiratory category to which the patient belongs [21].

- Category 1 - lying down, sleeping, with all atonic skeletal muscles except for the phrenic nerve and some ocular and mandibular muscles.
- Category 2 - sitting position, where the diaphragm muscle and all the muscles of the neck enter into synchronous activity, in addition to the intercostal and abdominal muscles commanded by the respective nerves.
- Category 3 - standing, or in physical activity, all muscles of category 2 and including the upper trapezius muscle and the ECM.

In addition, it is worth mentioning that inspiration activates the anterior muscles of the neck, promoting a rectinization of the cervical lordosis, which would facilitate the opening of the pharynx.

The careful analysis of the anatomical conditions of the upper airways, the neuromuscular control of breathing plus the physical behavioral events of the gases in respect to the general laws that govern them, lead at least to a question about the structural obstructive character preventing or hindering the airflow.

9. Swallowing

In view of the above, how would the pathophysiology of this syndrome be explained? One of the paths could be in the swallowing process. I am not going to talk about the whole physiology of swallowing, which is well described in the treatises on medical physiology and neuroscience. Special attention should be given to the passage from the oral phase to the pharyngeal phase, which is involuntary. At this exact moment, where the undulatory movement of the tongue exerts mechanical pressure on the receptors existing in the pillars of the fauces and in the pre-epiglottic folds, the transition from the oral phase of swallowing to the pharyngeal phase is observed. The afferent by the glossopharyngeal nerve (in the pre-epiglottic fold) and the vagus nerve (in the pillar of the fauces) follow to the nucleus of the solitary tract. The information triggers a cascade of highly complex responses to the swallowing nucleus, with special attention to the branch that from there reaches the respiratory nucleus. This afferent towards the respiratory nucleus aims to INHIBIT the phrenic nerve during swallowing, which stops activating the diaphragm muscle, which is responsible for the highest percentage of lung volume increase during swallowing. This physiological process prevents anything that transits through the oropharynx from being sucked into the lungs at that time [17]. It is worth mentioning that only the phrenic nerve is inhibited, while the others responsible for activating the neck, intercostal, abdominal and accessory muscles continue to be active depending on the patient's respiratory category.

10. Baseline Postural Tone

Among the various factors that contribute to the onset of OSAHS and snoring, basal muscle tone is perhaps the most important of them. Adequate muscle tone and competent lip sealing are decisive so that oral tissues do not stimulate the sensors of the pharyngeal phase of swallowing during sleep. Basal, or supra segmental tone is regulated by the reticular formation (RF) in the brainstem. This control implies facilitating or inhibiting the gamma system, which is directly responsible for maintaining supra segmental tone, due to the positive or negative influence of the cognitive cortex. The RF receives these positive or negative influences by the excitation of various cortical systems. For example, the stimulus in the visual or auditory cortex has a positive effect on the RF facilitating area that benefits the gamma system, making all skeletal muscles go into tone. On the other hand, harmful stimuli in the somesthetic system (temperature, touch, acute and chronic pain, spatial position of the joints and their dynamics) negatively influence the area that facilitates RF and positively influence the area that inhibits RF. The end result is poor tone due to the inhibition of the gamma system [17]. It is known that the temporomandibular joint has an important representation in the sensory and motor cortex. Any inappropriate position or masticatory dysfunction of the mandibular heads has a significant influence on the somesthetic system with repercussions on baseline postural tone [17].

Everything leads us to believe that the impaired tone favors the installation of SAHOS. MADs end up positively influencing disease control, improving tone, preventing false stimulation of the pharyngeal phase of swallowing, reducing the AHI to bearable levels, controlling the syndrome and probably snoring. However, the criteria used for mandibular advancement are questionable, even those that use the Gauge instrument, in an attempt to eliminate a supposed "mechanical obstruction" without taking into account the short and long-term side effects caused.

Let's assume that a patient, regardless of the AHI presented, belongs to respiratory category 2 or 3 at the time of sleep, that is, there was an inversion of respiratory categories. Most of these patients do not have physiological breathing (exclusively through the nose), inefficient pulmonary breathing, or even more serious processes such as emphysemas, etc. If this patient has a compression of the bilaminar zone (retro discal) with invasion of the mandible head below the 3 mm necessary for the functional spaces of the TMJs, most likely his basal muscle tone will be impaired. When submitted to a polysomnography exam, whenever there is a false swallowing caused by the mechanical stimulation of the dorsum of the tongue on the receptors, the phrenic nerve will be inhibited, where 1 respiratory event will be counted if this inhibition lasts 10 seconds or more, which will be categorized as "central" type in category 1 individuals, "obstructive" type in the other categories without airflow record; of the Hypopnea type when there is a 30% reduction in the flow associated with O2 desaturation for any category. The patient will remain in "apnea" until true swallowing occurs or until the level of CO2 in the bloodstream is perceived (e.g., sinuses and carotid bodies) and the respiratory nucleus is stimulated by the adrenaline rush [17].

The results of the case presented show that the change in reverse therapeutic posture (from the spatial position of the mandible heads - RTPC) calculated by the algorithm is sufficient to control OSAHS without exaggerated advances. In this clinical case, only one vertical adjustment and one adjustment in the lower facial height were necessary, without the need for mandibular advancement.

9. Conclusion

A detailed study of the upper airways involving anatomy, respiratory physiology, swallowing physiology, control of supra segmental postural tone and the respective neurological interaction is necessary for a better understanding of OSAHS. MADs should not exaggerate their titrations, thus reducing their side effects, sometimes irreversible.

The AAS1-ACT inter occlusal splint used in this case proved to be efficient in relation to the complaints presented, promoting only the decompression of the noble tissues of the TMJs, regulating the mandibular dynamics and regulating the functions of breathing and swallowing. This case report opens new horizons for further studies to be implemented to better understand the pathophysiology of OSAS.

References

1. Cavalcanti AL, Souza LS. Therapy of Obstructive Sleep Apnea Syndrome: Literature review. Odontol Clin Cientif. 2006; 5(3): 189-193.
2. Nabarro PAD, Hofling RTB. Effectiveness of the Balters Bionator apparatus in the treatment of snoring and sleep apnea. Rev Dental Press Orthod and Ortoped Facial. 2008; 13(4): 36-44.
3. Poluha RL. Dentistry in obstructive sleep apnea syndrome: diagnosis and treatment. RevBras Odontol. 2015; 72: 87-90.
4. Dekon SFC. Role of the dental surgeon in the treatment of primary snoring and obstructive sleep apnea. Rev. Odontol Araçatuba. 2012; 32: 70-74.
5. Ramos LVT, Furquin LZ. Device for obstructive sleep apnea. Rev Clin Ortod Dental Press. 2004; 3(2): 21-26.
6. Vinha PP. Snoring and sleep apnea: presentation of a new intraoral device and treatment protocol. RGO. 2010; 58(4): 515-520.
7. Hanzel DA. Response of obstrutive sleep apnea do fluoxetine and protriptylinbe. Chest. 1991; 100(2): 416-421.
8. Dal-Fabbro C. Clinical and polysomnographic evaluation of the BRD device in the treatment of Obstructive Sleep Apnea Syndrome. Dental Press J Orthod. 2010; 15(1): 107-117.
9. Bittencourt LRA. General approach to patients with obstructive sleep apnea syndrome. Rev Bras Hypertension. 2009; 16(3): 158-163.
10. Duran-Cantolla J. Efficacy of mandibular advanced device in the treatment of obstructive sleep apnea syndrome: a randomized controlled crossover clinical trial. Med Oral Patol Oral Cir Bucal. 2015; 20: 605-615.
11. Anitua E. Minimizing the mandibular advancement in an oral appliance for the treatment of obstructive sleeo apnea. Sleep Med,jan,pp. 2017; 699-703.
12. Almeida MAO. Treatment of obstructive sleep apnea and hypopnea syndrome with intraoral appliances. Rev Bras Otorrin. 2006; 72(5): 699-703.
13. Cistulli PA. Treatment of snoring and obstructiva sleep apnea with mandibular repositioning appliances. Sleep Med Rev. 2004; (6, dec): 443-457.
14. Hoffstein V. Review of oral appliances for treatment of sleep-disordered breathing. Sleep Breath. 2007; 11: 1-22.
15. Aarab G. Effects of an oral appliance with different protrusion positions at a vertical dimensio on obstructive sleep apnea. Clin Oral Invest. 2010; 339-345.
16. Ishiyama H. Effect of jaw-opening exercice on prevention of TMJ disorders pain associated with oral appliance therapy in obstructive sleep apnea patients. A randomized, double-blind, placebo controlled trial. J Prosthod Res. 2016; 376: 27-37.
17. Douglas, C.R. Treatise on Medical Physiology applied to the health sciences. Hobe Editora, SP. 1988.
18. Scarlati A. Spatial evaluation of the head of the mandible in patients with TMD by means of computed tomography. 2014.
19. Barbosa DF. Combined Therapy for Associated Orofacial Disorders – a Challenging Case Report. Eur J Dent. 2022; 16(4): 947-949.
20. Bana LF. Evaluation of ASA1 in electromyographic activity of the temporalis and masseter muscles. Dissertation presented to the FOP of the State University of Campinas SP – master's defense. 2024.
21. Campignion P. Respirations. Breathing for a healthy life. Summus Editorial, 2ed – SP. 1998.