1916 - 2026

An Acad Bras Cienc (2026) 98(2): e20251179 DOI 10.1590/0001-3765202620251179
Anais da Academia Brasileira de Ciências | *Annals of the Brazilian Academy of Sciences*
Printed ISSN 0001-3765 I Online ISSN 1678-2690
www.scielo.br/aabc | www.fb.com/aabcjournal

HEALTH SCIENCES

Impact of CPAP treatment for obstructive sleep apnea on stomatognathic system function: a study on bite force, thickness, and temperature of the masseter and temporal muscles

GUILHERME G.C. GOMES, MARCELO PALINKAS, EDSON D. VERRI, ANA LÍVIA. L. ROSSI, DANIEL S. DE SOUSA, DANILO H. LATTARO, JÚLIA C. HALLAK, LUCIANO M.A. FERREIRA, SIMONE C.H. REGALO & SELMA SIÉSSERE

Abstract: Obstructive sleep apnea (OSA) affects the body due to repeated obstruction of the upper airways. Continuous positive airway pressure (CPAP) is a treatment that can influence muscle function. This nine-month longitudinal study evaluated the effects of CPAP on molar bite force and on the thickness and surface temperature of the masseter and temporal muscles in adults. The study included 12 adults aged 20–70 with moderate or severe OSA, confirmed by polysomnography, all using CPAP. Molar bite force was measured with dynamometer; muscle thickness was evaluated by ultrasound at rest and during maximum contraction; and temperature was assessed using infrared thermography. Measurements were taken before treatment and at 3, 6, and 9 months. Data were analyzed using repeated-measures ANOVA with Bonferroni correction ($p<0.05$). Although no significant differences in bite force were observed, only a non-significant tendency toward increased maximum molar force on both sides was noted after nine months. Ultrasound showed a significant increase in right temporal muscle thickness at rest and during contraction, and a left-side increase at rest. Nine months of CPAP therapy in adults with moderate to severe obstructive sleep apnea resulted may influence craniofacial muscle morphology without significantly altering stomatognathic function.

Key words: obstructive sleep apnea, CPAP, masticatory muscles, stomatognathic system.

INTRODUCTION

Obstructive Sleep Apnea (OSA) is a disorder characterized by the partial or complete collapse of the upper airways due to reduced muscle tone, leading to episodes of hypopnea or apnea (Da Silva et al. 2023, Maréchal et al. 2023, Ulualp & Kezirian 2024). This condition induces intermittent hypoxia, sleep fragmentation, and inflammatory and oxidative responses, significantly impacting the quality of life of affected subjects (Fontanilles Arbones et al. 2024).

The stability of the upper airways depends on the interaction of various anatomical structures, primarily the pharynx, with emphasis on the oropharynx, the region where most obstructive episodes occur (Bilston & Gandevia 2014, Mayoral & Lagravère 2024). Predisposing factors include increased soft tissue volume, obesity, and craniofacial abnormalities, which can compromise airway patency (Galtieri et al. 2024).

The gold standard treatment for OSA is continuous positive airway pressure (CPAP) therapy, which maintains a constant airflow, preventing the collapse of soft structures (Lo et al. 2025). Adherence to CPAP is influenced by the comfort of the interface used, with nasal masks offering better tolerance and effectiveness in reducing obstructive respiratory events (Bahammam et al. 2017). Prolonged use of CPAP may induce neuromuscular and structural changes, particularly in the stomatognathic system, which is responsible for chewing, phonation, and breathing.

The stomatognathic system consists of masticatory muscles, the temporomandibular joint, and associated structures, playing an essential role in maintaining mandibular posture and the functionality of the upper airways. The electromyographic activity of masticatory muscles, such as the masseter and temporal muscles, is linked to the compensatory response to apnea episodes, contributing to the restoration of airflow (Li et al. 2023, Kaongampanich et al. 2025). However, continuous use of CPAP may impact the function of these muscles, altering bite force, muscle thickness, and even the surface temperature of the involved structures.

The assessment of the impact of CPAP on the stomatognathic system can provide valuable information to optimize the therapeutic approach for OSA. Measures such as maximum molar bite force, the thickness of the masseter and temporal muscles, and infrared thermography have been used to understand possible muscular and functional adaptations over time (Clemente et al. 2020, Gomes et al. 2022).

The aim of this study was to analyze the effects of OSA treatment with CPAP on the stomatognathic system by evaluating bite force, muscle thickness, and surface temperature at different stages of the treatment. The null hypothesis adopted in this study assumes that the use of CPAP does not cause changes in the function of the stomatognathic system over time.

METHODS

Study design and sample selection

This longitudinal study was approved by the ethics committee of the at Ribeirão Preto School of Dentistry, São Paulo, Brazil (process # 45122721.2.0000.5419). All subjects were informed about the objectives and procedures and formally consented to participate by signing the informed consent form. The selection of subjects was carried out through an open invitation to the population. All were subjected to OSA treatment with CPAP and were followed up before the start of therapy and at 3, 6, and 9 months. The diagnosis of OSA was made through polysomnography conducted by a sleep specialist in a specialized clinic during a single night of sleep.

Inclusion criteria were: subjects with natural dentition, the presence of all four first permanent molars, normal occlusion, clinical diagnosis of OSA, use of CPAP, ages between 20 and 70 years, of both sexes. Subjects were excluded if they had ulcerations, open wounds, or skin hypersensitivity, cognitive deficits, other decompensated neurological or systemic pathologies, edentulous subjects or those using full or removable dentures; also, those with periodontal diseases, using muscle relaxants that could affect neuromuscular physiology, or those undergoing orthodontic, speech therapy, or otorhinolaryngological treatments.

A priori sample size calculation was performed based on a primary variable from an international study, which was the tonic electrical activity of the genioglossus muscle during NREM sleep, in subjects with obstructive sleep apnea using CPAP and in a control group (Zhou et al. 2019). The values used for the calculation were 1.23 [0.73%] for the patient group and 2.54 [1.12%] for the control group. A significance level of α = 0.05 was considered. The effect size obtained was 1.38, and the test power was 96%. The minimum sample size was determined to be 12 subjects. The calculation was performed using the software G*Power 3.1 (Franz Faul from the University of Kiel, Germany).

Therefore, 12 adult subjects with moderate or severe OSA using CPAP (fixed or automatic) participated in the study. The analysis of the stomatognathic system included the measurement of maximum molar bite force, as well as the evaluation of the thickness and surface temperature of the masseter and temporal muscles

Molar bite force analysis

Data regarding maximum molar bite force were collected using a digital dynamometer, model IDDK (Kratos - Equipamentos Industriais Ltda., Cotia, São Paulo, Brazil), with a capacity of up to 980,7 N. The device featured two rods equipped with teflon discs at their ends, on which the bite force was applied. The bite force was recorded with the subject seated, with their hands resting on their thighs. The dynamometer was disinfected with alcohol and protected by disposable latex finger cots, which were placed on the bite rods as a biosafety measure. Prior to beginning data collection, tests were conducted with the dynamometer to ensure the reliability of the procedure. Measurements were taken in the region of the first permanent molars, both on the right and left sides. The bite force was recorded three times, with two-minute intervals between recordings, alternating between the right and left sides. For the study, the maximum molar force was considered (Palinkas et al. 2010, Esposto et al. 2024).

Masseter and temporal muscles thickness analysis

To measure of the masseter and temporal muscles thickness, a portable ultrasound device (Titan; SonoSite Inc, Bothell, WA, USA) with a 13 MHz linear transducer was used. During the examination, the subjects remained seated in a comfortable chair, with an upright posture, feet flat on the floor, palms resting on their thighs, and the body slightly reclined, without head fixation. The environment was kept calm and with reduced lighting. Instructions and explanations were provided to the participant, requesting that they remain relaxed during the procedure (Palinkas et al. 2010, Donizetti Verri et al. 2019).

The location of the masseter and temporal muscles, on both sides, was determined by palpation and the movement of the linear transducer. The transducer was covered with water-based electroconductive gel to prevent the presence of air and ensure the quality of the images. The transducer was then positioned perpendicular to the muscle fibers, with the masseter muscle belly located approximately 2.75 cm above the mandibular angle, towards the upper eyelid. The anterior portion of the temporal muscle was located about 1.25 cm posterior and superior to the outer corner of the eye (Mendes da Silva 2017). Three ultrasound images of the masseter and temporalis muscles on both sides were captured at rest and during maximum voluntary contraction (MVC), with a two-minute interval separating each image.

Skin temperature in the region of the masseter and temporal muscles analysis

A thermographic camera (FLIR Systems Inc., model FLIR C2 - autofocus) was used, along with a computer for data analysis using the FLIR Ignite software. The camera settings followed specific parameters: emissivity adjusted to 0.98, with the camera positioned at a distance of 0.90 meters from the evaluated area (right and left lateral faces), mounted on a tripod and perpendicular to the floor. The environment was maintained under controlled conditions, with the ambient temperature during data collection ranging between 22 and 23°C, relative humidity of the air equal to or less than 60%, and no computers or electronic devices nearby (Moreira et al. 2017).

Before the data collection began, the subjects were instructed, when necessary, to tie their hair back to expose the areas of the ears and the masseter and temporal muscles bilaterally. The skin of the face was cleaned with a tissue dampened with alcohol, and foam markers were placed around the masseter muscles. The subjects remained at rest for 20 minutes to allow facial thermoregulation (Hegedűs 2018).

The thermographic evaluation was performed using the software with the polyarea tool. Initially, the jaw area was marked with the tool, followed by the selection of the masseter muscle area, starting from the temporal process of the zygomatic bone and the body of the mandible. To mark the temporal muscle area, the polyarea tool was used again to delineate the anterior portion of the temporal muscle in the temporal fossa, the hairline, the temporal process of the zygomatic bone, and the zygomatic process of the frontal bone. The software was configured with an emissivity of 0.98, a distance of 0.90 meters, and the Rainbow HC color palette.

Statistical analysis

After obtaining the data for the analyzed variables, the Shapiro-Wilk normality test was applied, which confirmed a normal distribution. The statistical analysis of the data was performed using SPSS software, version 20.0 (SPSS Inc., Chicago, IL, USA). A repeated-measures (ANOVA) was applied, followed by Bonferroni post-hoc correction, with a significance level set at $p < 0.05$.

RESULTS

Table I presents the data obtained before CPAP treatment and after 3, 6, and 9 months of therapy. No statistically significant differences were detected in bite force; however, a progressive increase in maximum molar force on both sides was noted after nine months. Similarly, no significant changes were found in the thickness of the masseter and temporal muscles, either at rest or during maximum voluntary contraction. Despite this, a gradual increase in masticatory muscle thickness was observed after nine months of CPAP use. Thermographic analysis also revealed no significant differences, though a transient rise in temperature was recorded at the third month, followed by a gradual decline thereafter.

Table II compares the measurements taken before treatment and after nine months of CPAP use. While bite force did not differ significantly, an overall increase in maximum molar force was evident at the end of the follow-up period. Ultrasound analysis demonstrated a significant increase in the right temporal muscle thickness at rest ($p = 0.01$) and maximum voluntary contraction ($p = 0.04$), as well as in the left temporal muscle at rest ($p = 0.04$).

A visual inspection of individual trajectories revealed progressive changes in several participants, with ariability in muscle thickness, bite force, and thermographic patterns across the follow-up periods, complementing the statistical findings (Figures 1–4).

Table I. **Mean values ± standard error of molar bite force (N), muscle thickness (cm), and surface temperature (°F) of the masseter and temporal muscles across periods: before CPAP treatment and after 3, 6, and 9 months. Statistical analysis was performed using repeated-measures ANOVA with Bonferroni post-hoc correction ($p < 0.05$) *. Different superscript letters within the same row indicate significant differences between periods according to the post-hoc test.**

Variables	Periods				p-value	Cohen's d
	Before	3 months	6 months	9 months		
Bite Force						
Right molar	391.8±72.0	402.0±74.0	366.8±63.8	401.1±64.7	0.82	0.14
Left molar	366.9±69.3	378.3±64.5	435.6±70.3	402.1±70.4	0.07	0.50
Muscle thickness Rest						
Right masseter	1.37±0.07	1.37±0.07	1.37±0.09	1.37±0.07	0.99	0.00
Left masseter	1.38±0.07	1.36±0.07	1.37±0.08	1.39±0.07	0.97	0.14
Right temporal	0.61±0.05[a]	0.68±0.03[b]	0.67±0.04[c]	0.69±0.03[d]	0.04*	1.86
Left temporal	0.53±0.04	0.58±0.02	0.58±0.02	0.61±0.03	0.09	2.18
Muscle thickness Maximum voluntary contraction						
Right masseter	1.52±0.08	1.55±0.07	1.53±0.08	1.57±0.09	0.88	0.59
Left masseter	1.56±0.08	1.59±0.08	1.60±0.08	1.60±0.07	0.90	0.52
Right temporal	0.74±0.05	0.78±0.03	0.78±0.04	0.82±0.03	0.10	1.82
Left temporal	0.68±0.04	0.73±0.02	0.73±0.02	0.73±0.03	0.21	1.40
Surface temperature						
Right masseter	89.6±32.6	89.6±32.7	88.3±32.5	87.9±33.2	0.32	0.05
Left masseter	87.2±32.6	88.7±32.8	86.7±32.6	86.0±33.0	0.16	0.04
Right temporal	90.7±32.5	91.8±32.5	89.5±32.7	88.6±33.4	0.06	0.07
Left temporal	88.3±32.5	90.6±32.7	88.3±32.6	87.2±33.2	0.06	0.03

DISCUSSION

The results showed that, over the nine-month follow-up, no significant differences were found for most of the analyzed variables, leading to acceptance of the null hypothesis. The only exception was the right temporalis muscle thickness at rest, which demonstrated a significant difference across time, indicating that this parameter alone did not confirm the null hypothesis.

International research indicates that subjects diagnosed with obstructive sleep apnea experience recurrent episodes of upper airway obstruction, resulting from the repetitive collapse of these dynamic structures (Panou et al. 2025). This obstruction can lead to the development of a compensatory breathing pattern, often affecting muscle function (Wilcox et al. 1990).

Table II. **Mean values ± standard error of molar bite force (N), muscle thickness (cm), and surface temperature (°F) of the masseter and temporal muscles across periods: before CPAP treatment and after 9 months of use. Statistical analysis included the p-value from the Bonferroni post-hoc correction (p < 0.05) *.**

Variables	Periods		p-value	
	Before	9 months		Cohen's d
Bite Force				
Right molar	391.8±72.0	401.1±64.7	0.84	0.13
Left molar	366.9±69.3	402.1±70.4	0.38	0.50
Muscle thickness Rest				
Right masseter	1.37±0.07	1.37±0.07	0.95	0.00
Left masseter	1.38±0.07	1.39±0.07	0.72	0.14
Right temporal	0.61±0.05	0.69±0.03	0.01*	1.86
Left temporal	0.53±0.04	0.61±0.03	0.04*	2.18
Muscle thickness Maximum voluntary contraction				
Right masseter	1.52±0.08	1.57±0.09	0.59	0.59
Left masseter	1.56±0.08	1.60±0.07	0.46	0.52
Right temporal	0.74±0.05	0.82±0.03	0.04*	1.86
Left temporal	0.68±0.04	0.73±0.03	0.15	1.41
Surface temperature				
Right masseter	89.6±32.6	87.9±33.2	0.25	0.05
Left masseter	87.2±32.6	86.0±33.0	0.35	0.04
Right temporal	90.7±32.5	88.6±33.4	0.20	0.06
Left temporal	88.3±32.5	87.2±33.2	0.45	0.03

The results of this study showed that although the ultrasound images of the masseter and temporal muscles did not reveal significant differences over the nine months of CPAP treatment, except for the right temporalis muscle at rest, the analysis of the time factor alone (before and after nine months of treatment) indicated significant changes in three muscles. Additionally, clinical observations suggested changes in muscle morphology over time. Muscle thickness, as an indicator of functional adaptation, is directly influenced by the mechanical load applied to the muscles (Palinkas et al. 2010). Although many comparisons did not reach statistical significance, the Cohen's d values help contextualize the magnitude of the observed changes and indicate that some muscle adaptations may have been physiologically relevant despite limited statistical power.

Evidence suggests that the increase in temporalis muscle thickness observed after nine months of the treatment proposed in this study

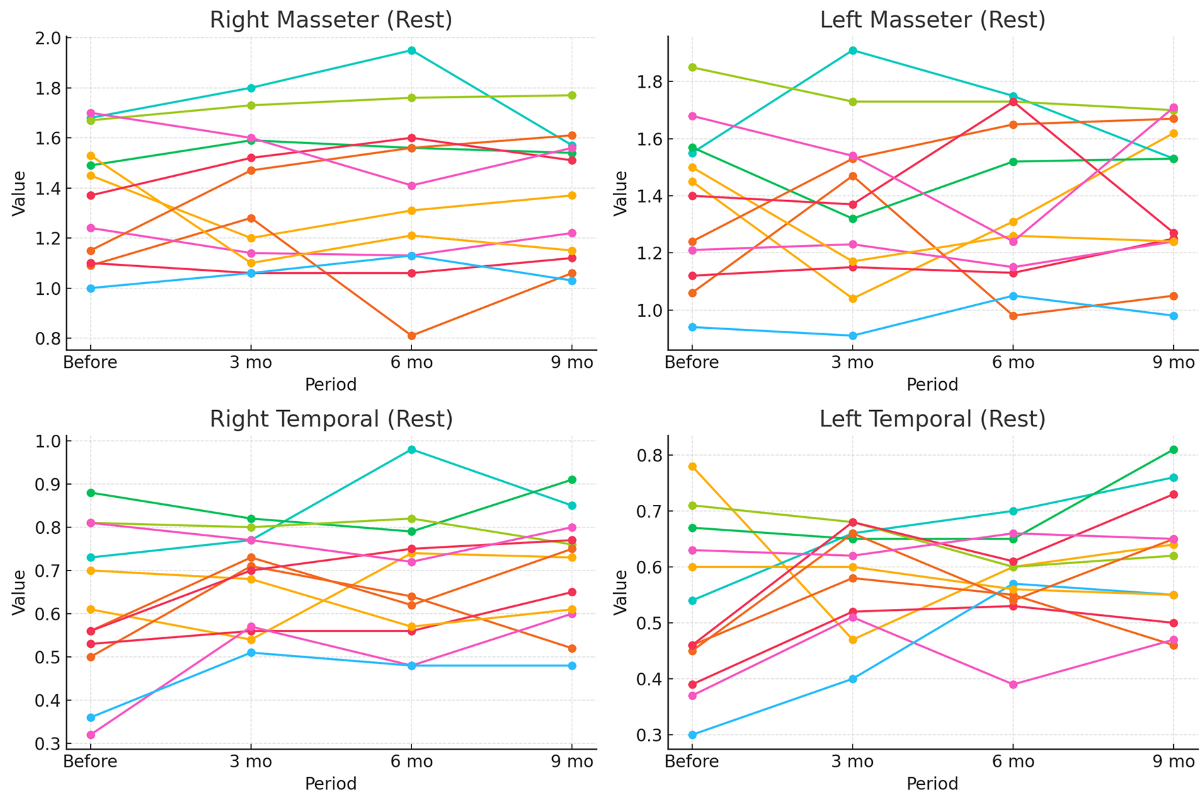

Figure 1. Individual trajectories of muscle thickness at rest (right/left masseter; right/left temporalis) across before, 3, 6, and 9 months (mo). Repeated-measures ANOVA showed significant increases for the right temporalis (p = 0.01) and left temporal (p = 0.05), with no significant changes in masseter muscles. Plots illustrate intra-individual variability and overall temporal muscle adaptation.

may be related to physiological and functional factors. Treatment of obstructive sleep apnea with CPAP improves blood oxygenation (Yang et al. 2024), reduces episodes of hypoxia (Pun et al. 2021), and supports muscle recovery and strengthening (Granton et al. 1996). In addition, many patients with OSA may exhibit bruxism (Massahud et al. 2022). However, the correlation between these two conditions is not yet well established, and further studies are needed to clarify this relationship (Błaszczyk et al. 2024) CPAP use may also alter muscle activation patterns, potentially leading to compensatory hypertrophy; however, sleep bruxism was not evaluated in this study sample.

Another important factor is the reorganization of neuromuscular function, as OSA can lead to dysfunction in orofacial muscle activity, and its correction may result in changes in muscle tone (Sambale et al. 2024). Additionally, CPAP exerts continuous positive pressure, stimulating the muscles responsible for stabilizing the airways (Galtieri et al. 2024). Theoretically, this may promote adaptations such as hypertrophy of the masticatory muscles, which are key dynamic components of the stomatognathic system and have a direct functional relationship with the craniomandibular region.

Bite force is an important methodological biomechanical parameter for analyzing the efficiency of the masticatory system (Palinkas et al. 2010, Bueno et al. 2025). Factors such as muscle tone, neuromotor adaptation, and the

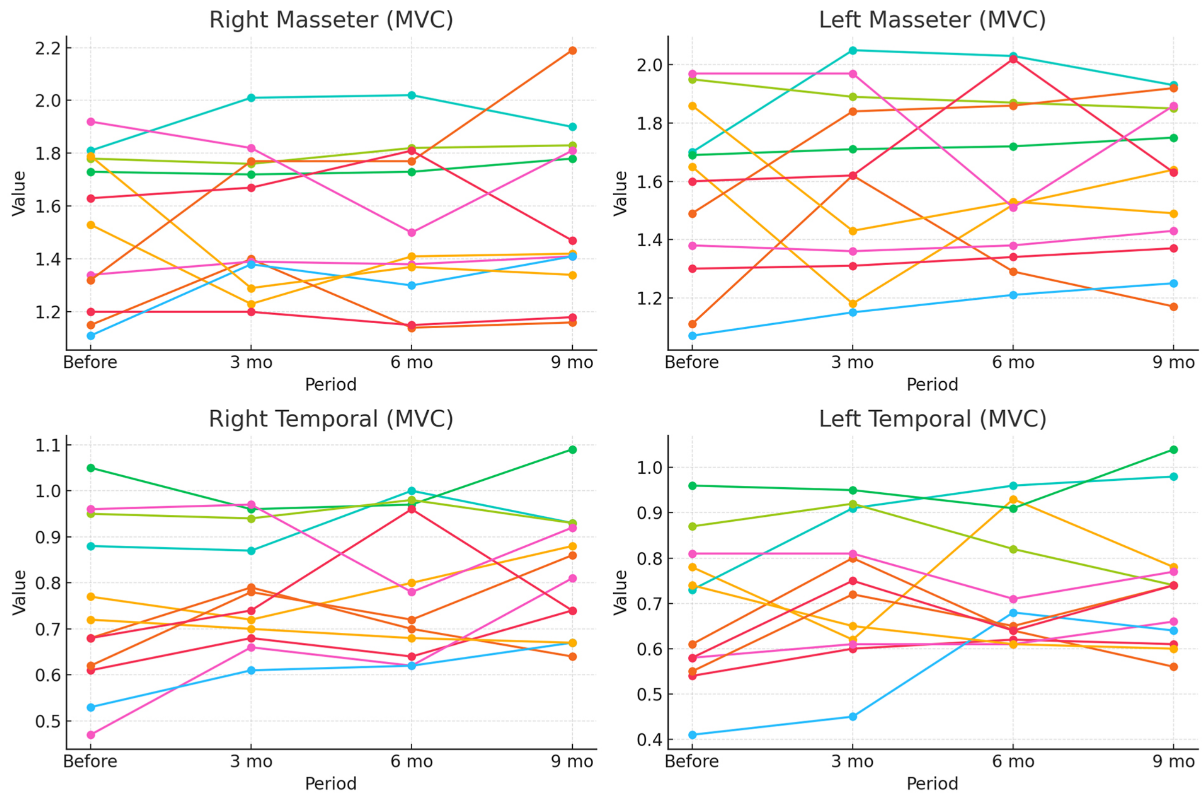

Figure 2. Individual trajectories of muscle thickness during MVC for the right/left masseter and temporalis muscles across before, 3, 6, and 9 months (mo). Repeated-measures ANOVA revealed a significant effect of period only for the right temporalis muscle (p = 0.04). No significant changes were observed in the other muscles. Lines show variability in contractile adaptation over time.

integrity of the temporomandibular joints influence the ability to generate force (Moritani 1993). The literature indicates that subjects with obstructive sleep apnea may experience a reduction in force due to chronic muscle fatigue resulting from nocturnal respiratory effort (Zhang et al. 2022).

The analysis of maximum right and left molar bite force in this study showed that, although no significant differences were found across the evaluated periods, there was a trend toward higher maximum molar bite force after nine months of CPAP treatment. Improved oxygenation and sleep quality resulting from CPAP use may reduce fatigue and enhance muscle strength (Leelasittikul et al. 2024).

The increase in bite force observed after nine months of CPAP use, even in the absence of a statistically significant difference, may be explained by a more favorable balance between functional demand and muscle capacity. Improved oxygenation during sleep reduces the effects of oxidative stress caused by intermittent hypoxia, an essential mechanism for supporting the recovery of contractile strength (Zhang et al. 2024). Furthermore, stabilizing the breathing pattern may decrease compensatory recruitment of accessory muscles, thereby promoting more efficient function of the masseter and temporalis muscles by preserving the integrity of the associated physiological systems.

Surface skin temperature reflects tissue metabolism and local blood flow, serving as a

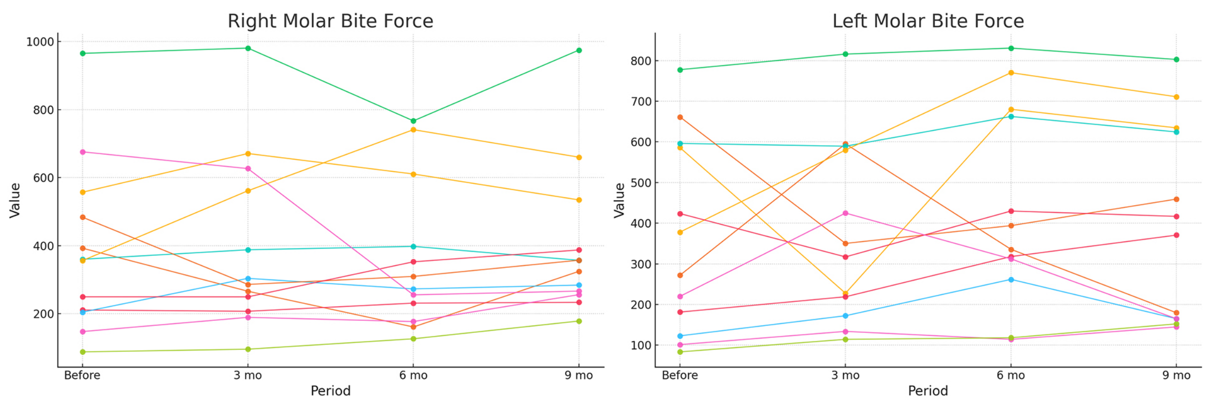

Figure 3. Individual trajectories of right and left molar bite force across before, 3, 6, and 9 months (mo). Repeated-measures ANOVA revealed no significant period effects; however, heterogeneous individual responses are evident, with some participants showing progressive increases in bite force.

relevant indicator of muscle activity and blood perfusion (Korman et al. 2024). Thermal changes may signal variations in these processes, especially in response to new stimuli or physiological adaptations (Chung et al. 2024). In the context of using CPAP as treatment for OSA in this study, the thermal increase observed in the third month may be associated with an initial rise in muscle activity related to blood perfusion, This occurs because CPAP directly influences the breathing pattern and the postural stability of the jaw, requiring neuromuscular adjustments (Masumi et al. 1996).

With prolonged use of the device, the progressive reduction in temperature until the ninth month could indicate the stabilization of muscle metabolism, as decreases in muscle temperature are consistent with a transition to more efficient metabolic functioning during repeated or sustained activity, leading to lower heat production and a more balanced perfusion–metabolism response (Morris et al. 2005). This pattern suggests that after the initial adjustment phase, the metabolic demand becomes more balanced, resulting in a lower need for increased blood perfusion. In this context, infrared thermography has proven to be a promising tool for detecting muscular thermal variations (Clemente et al. 2020, Cecilio et al. 2025). Its application enables the detection of subtle changes in neuromuscular function over time, offering valuable insights into the adaptive responses to interventions affecting muscle activity.

Limitations

This study has some limitations that should be considered. The small sample size, although justified by a power calculation, limits the generalizability of the findings; furthermore, the effect size used for this calculation was based on genioglossus EMG data rather than the primary outcomes of this study. The absence of a control group without OSA or without CPAP limits causal inference, as temporal effects cannot be separated from treatment effects. Changes in OSA-related outcomes, including the Apnea–Hypopnea Index and downstream markers, may reflect not only CPAP therapy but also the natural progression of the disease over months to years, even without treatment. Therefore, the observed effects cannot be attributed exclusively to CPAP and should be interpreted with caution (Sahlman et al. 2007). The nine-month follow-up period may also have been insufficient to detect broader structural muscle adaptations.

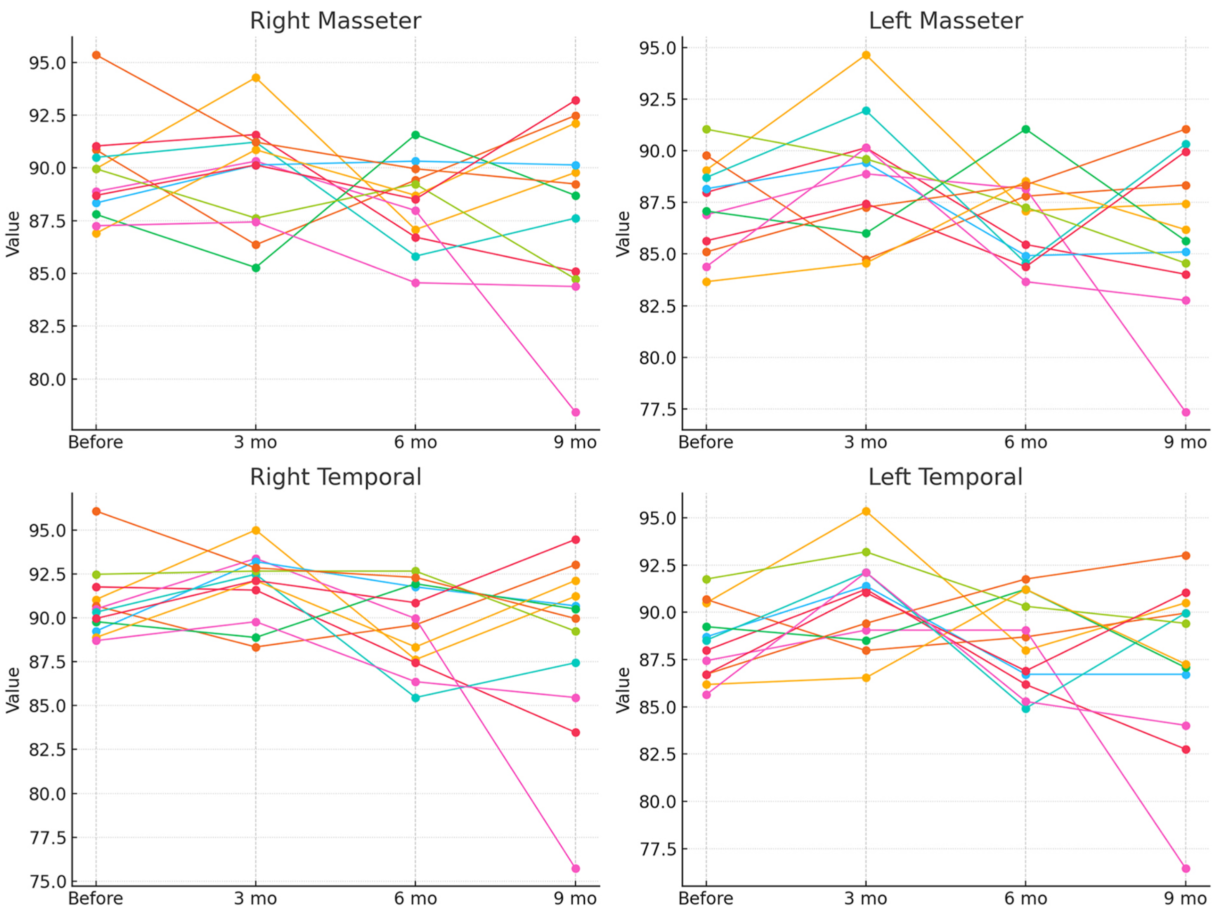

Figure 4. Individual thermographic trajectories for right/left masseter and temporalis muscles across before, 3, 6, and 9 months (mo). No significant temperature changes were detected in repeated-measures ANOVA, although individual variability is observable across periods.

Factors such as sleep bruxism, which may affect the function and thickness of the masticatory muscles, were not assessed. Therefore, caution is warranted when interpreting these results, and future studies should include larger samples, appropriate control groups, and longer follow-up periods.

CONCLUSIONS

The authors suggest that 9 months of CPAP therapy in adults with moderate to severe obstructive sleep apnea results may influence craniofacial muscle morphology without significantly altering stomatognathic function.

ABBREVIATIONS

OSA, obstructive sleep apnea,
CPAP, continuous positive airway pressure,
NREM, non-rapid eye movement.

Acknowledgments

This was not an industry supported study. This research was supported by Fundação de Amparo à Pesquisa do Estado de São Paulo (FAPESP/SP/Brazil) and Conselho Nacional de Desenvolvimento Científico e Tecnológico (CNPq) process number: 312586/2021-3. The authors have indicated no financial conflicts of interest. This work was conducted at the Ribeirão Preto School of Dentistry, University of São Paulo, São Paulo, Brazil.

REFERENCES

BAHAMMAM AS, SINGH T, GEORGE S, ACOSTA KL, BARATAMAN K & GACUAN DE. 2017. Choosing the right interface for positive airway pressure therapy in patients with obstructive sleep apnea. Sleep Breath 21: 569-575.

BILSTON LE & GANDEVIA SC. 2014. Biomechanical properties of the human upper airway and their effect on its behavior during breathing and in obstructive sleep apnea. J Appl Physiol(1985)116: 314-324.

BŁASZCZYK B, WALISZEWSKA-PROSÓŁ M, WIĘCKIEWICZ M, PORĘBA R, NIEMIEC P, PRZEGRAŁEK J & MARTYNOWICZ H. 2024. Sleep bruxism (SB) may be not associated with obstructive sleep apnea (OSA): A comprehensive assessment employing a systematic review and meta-analysis. Sleep Med Ver 78: 101994.

BUENO PM, KIEMLE TRINDADE PA, MEDEIROS LH, FIDÉLIS DA SILVA LV & TRINDADE-SUEDAM IK. 2025. Long-term effects of orthognathic surgery on masticatory function in individuals with cleft lip and palate: A prospective study. J Oral Biol Craniofac Res 15: 33-40.

CECILIO FA, SIÉSSERE S, BETTIOL NB, GAUCH CG, DE VASCONCELOS PB, GONÇALVES LMN, ANDRADE LM, REGALO IH, REGALO SCH & PALINKAS M. 2025. Effect of intervertebral disc degeneration on the stomatognathic system function in adults. Cranio 43: 401-409.

CHUNG YT, YEH CY, CHEN CC, LAI CH, LIN YH, LIN CY, SHU YC & KO NY. 2024. Early changes in skin surface temperature predict body temperature increases in patients with fever: A pilot study. Intensive Crit Care Nurs 83: 103652.

CLEMENTE MP, MENDES J, VARDASCA R, MOREIRA A, BRANCO CA, FERREIRA AP & AMARANTE JM. 2020. Infrared thermography of the crânio-cervico-mandibular complex in wind and string instrumentalists. Int Arch Occup Environ Health 93: 645-658.

DA SILVA NC, DA SILVA GPJT, ONOFRI SMM & PINATO L. 2023. Obstructive sleep apnea and orofacial myofunctional aspects in obesity. Sleep Breath 27: 1351-1358.

DONIZETTI VERRI E ET AL. 2019. Effects of Parkinson's disease on molar bite force, electromyographic activity and muscle thickness of the masseter, temporal and sternocleidomastoid muscles: A case-control study. J Oral Rehabil 46: 912-919.

ESPOSTO DS, PALINKAS M, RIGHETTI MA, TAUBE OLS, FERNANDES ALA, CHIARATTO J, RICARDO V, RUFATO FCTF, REGALO SCH & SIÉSSERE S. 2024. Rheumatoid arthritis and osteoarthritis in adult women: a functional approach to the stomatognathic system. Prague Med Rep 125: 220-231.

FONTANILLES ARBONES E, SALORD OLEO N, GASA GALMES M, PÉREZ RAMOS S, PRADO GALA E, CALVO SÁNCHEZ M, PALLARÈS FONTANET N, SANTOS PÉREZ S & MONASTERIO PONSA C. 2024. Phenotypes of obstructive sleep apnea in women: A real-life cohort study. Sleep Med 121: 295-302.

GALTIERI R, SALLES C, KUSHIDA CA, MEIRA E CRUZ M & SOUZA-MACHADO A. 2024. Morphometric measures and desaturations: Proposal for an index with improved accuracy for obstructive sleep apnea screening. Sleep Med 122: 258-265.

GOMES GGC, PALINKAS M, DA SILVA GP, GONÇALVES CR, LOPES RFT, VERRI ED, FABRIN SCV, FIOCO EM, SIÉSSERE S & REGALO SCH. 2022. Bite Force, thickness, and thermographic patterns of masticatory muscles post-hemorrhagic stroke. J Stroke Cerebrovasc Dis 31: 106173.

GRANTON JT, NAUGHTON MT, BENARD DC, LIU PP, GOLDSTEIN RS & BRADLEY TD. 1996. CPAP improves inspiratory muscle strength in patients with heart failure and central sleep apnea. Am J Respir Crit Care Med 153: 277-282.

HEGEDŰS B. 2018. The potential role of thermography in determining the efficacy of stroke rehabilitation. J Stroke Cerebrovasc Dis 27: 309-314.

KAONGAMPANICH N, HOSIRILUCK N, TRIPRATEEPSILP N, PHOLSIRIPATHOM S & CHATCHAIYAN N. 2025. Sleep Bruxism in Thai obstructive sleep apnea patients. Int Dent J 75: 777-783.

KORMAN P, KUSY K, STRABURZYŃSKA-LUPA A, SILLERO-QUINTANA M, ZARĘBSKA E & ZIELIŃSKI J. 2024. Exploring the correlation of skin temperature and body composition in athletes undergoing exhaustive physical exercise. J Therm Biol 123: 103918.

LEELASITTIKUL K, PUGONGCHAI A, TEPWIMONPETKUN C & SAIPHOKLANG N. 2024. Quality of life, mental health, and CPAP compliance in thai patients with obstructive sleep apnea during COVID-19 pandemic. Sleep Disord 2024: 1373299

LI D, KUANG B, LOBBEZOO F, DE VRIES N, HILGEVOORD A & AARAB G.2023. Sleep bruxism is highly prevalent in adults with obstructive sleep apnea: a large-scale polysomnographic study. J Clin Sleep Med 19: 443-451.

LO JE, SCHMICKL CN, VAIDA F, NEMATI S, SINGH K, SANDS SA, OWENS RL, MALHOTRA A & ORR JE. 2025. The combination of physiology and machine learning for prediction of CPAP pressure and residual AHI in OSA. J Clin Sleep Med 21: 775-782.

MARÉCHAL L, DUMONCEL J, SANTOS F, ASTUDILLO ENCINA W, EVTEEV A, PREVOST A, TORO-IBACACHE V, VENTER RG & HEUZÉ

Y. 2023. New insights into the variability of upper airway morphology in modern humans. J Anat 242: 781-795.

MASSAHUD MLB, BRUZINGA FFB, DINIZ SAM, SERAIDARIAN KKA, LOPES RM, BARROS VM & SERAIDARIAN PI. 2022. Association between sleep bruxism, use of antidepressants, and obstructive sleep apnea syndrome: A cross-sectional study. J Oral Rehabil 49: 505-513.

MASUMI S, NISHIGAWA K, WILLIAMS AJ, YAN-GO FL & CLARK GT. 1996. Effect of jaw position and posture on forced inspiratory airflow in normal subjects and patients with obstructive sleep apnea. Chest 109: 1484-1489.

MAYORAL SANZ P & LAGRAVÈRE VICH M. 2024. Oral appliances for obstructive sleep apnea. Dent Clin North Am 68: 495-515.

MENDES DA SILVA J ET AL. 2017. Influence of mandibular tori on stomatognathic system function. Cranio 35: 30-37.

MOREIRA DG ET AL. 2017. Thermographic imaging in sports and exercise medicine: A Delphi study and consensus statement on the measurement of human skin temperature. J Therm Biol 69: 155-162.

MORITANI T. 1993. Neuromuscular adaptations during the acquisition of muscle strength, power and motor tasks. J Biomech 26: 95-107.

MORRIS JG, NEVILL ME, BOOBIS LH, MACDONALD IA & WILLIAMS C. 2005. Muscle metabolism, temperature, and function during prolonged, intermittent, high-intensity running in air temperatures of 33 degrees and 17 degrees C. Int J Sports Med 26: 805-814.

PALINKAS M, NASSAR MS, CECÍLIO FA, SIÉSSERE S, SEMPRINI M, MACHADO-DE-SOUSA JP, HALLAK JE & REGALO SC. 2010. Age and gender influence on maximal bite force and masticatory muscles thickness. Arch Oral Biol 55: 797-802.

PANOU T, ROUKAS K, CHADIA K, NENA E, GOUVERI E, PAPANAS N & STEIROPOULOS P. 2025. Obstructive sleep apnoea and type 1 diabetes mellitus: a neglected relationship? Exp Clin Endocrinol Diabetes 133: 40-50.

PUN M, BEAUDIN AE, RANERI JK, ANDERSON TJ, HANLY PJ & POULIN MJ. 2021. Impact of nocturnal oxygen and CPAP on the ventilatory response to hypoxia in OSA patients free of overt cardiovascular disease. Exp Neurol 346: 113852.

SAHLMAN J, PUKKILA M, SEPPÄ J & TUOMILEHTO H. 2007. Evolution of mild obstructive sleep apnea after different treatments. Laryngoscope 117: 1107-1111.

SAMBALE J, KOEHLER U, CONRADT R, KESPER K, CASSEL W, DEGERLI M, VINIOL C & KORBMACHER-STEINER HM. 2024. Is sleep bruxism in obstructive sleep apnea only an oral health related problem? BMC Oral Health 24: 565.

ULUALP SO & KEZIRIAN EJ. 2024. Advanced diagnostic techniques in obstructive sleep apnea. Otolaryngol Clin North Am 57: 371-83.

WILCOX PG, PARÉ PD, ROAD JD & FLEETHAM JA. 1990. Respiratory muscle function during obstructive sleep apnea. Am Rev Respir Dis 142: 533-539.

YANG Y, JIA D, CHENG L, JIA K & WANG J. 2024. Continuous positive airway pressure combined with small-tidal-volume ventilation on arterial oxygenation and pulmonary shunt during one-lung ventilation in patients undergoing video-assisted thoracoscopic lobectomy: A randomized, controlled study. Ann Thorac Med 19: 155-164.

ZHANG M, LU Y, SHENG L, HAN X, YU L, ZHANG W, LIU S & LIU Y. 2022. Advances in molecular pathology of obstructive sleep apnea. Molecules 27: 8422.

ZHANG X, ZHOU H, LIU H & XU P. 2024. Role of oxidative stress in the occurrence and development of cognitive dysfunction in patients with obstructive sleep apnea syndrome. Mol Neurobiol 61: 5083-5101.

ZHOU YQ, ZHAO D, YIN GP, LI JJ, CAO X, ZHANG YH & YE JY. 2019. Effectiveness of continuous positive airway pressure ventilation in improving genioglossus electromyography in patients with moderate to severe obstructive sleep apnea. Zhonghua Er Bi Yan Hou Tou Jing Wai Ke Za Zhi 54: 584-590.

GUILHERME G.C. GOMES[1]
https://orcid.org/0000-0002-7970-0717

MARCELO PALINKAS[1,2]
https://orcid.org/0000-0002-3445-8154

EDSON D. VERRI[1]
https://orcid.org/0000-0002-2403-3953

ANA LÍVIA L. ROSSI[1]
https://orcid.org/0009-0005-8988-589X

DANIEL S. DE SOUSA[3]
https://orcid.org/0009-0002-1658-8251

DANILO H. LATTARO[1]
https://orcid.org/0009-0002-2030-4274

JÚLIA C. HALLAK[1]
https://orcid.org/0000-0002-8210-9098

LUCIANO M.A. FERREIRA[1,4]
https://orcid.org/0000-0001-7536-9193

SIMONE C.H. REGALO[1,2]
https://orcid.org/0000-0003-4110-8299

SELMA SIÉSSERE[1,2]
https://orcid.org/0000-0001-9756-3771

[1]Universidade de São Paulo, Faculdade de Odontologia de Ribeirão Preto, Departamento de Biologia Básica e Oral, Avenida do Café, s/n, Monte Alegre, 14040-904 Ribeirão Preto, SP, Brazil

[2]Instituto Nacional de Ciência e Tecnologia Translacional em Medicina (INCT.TM), Hospital das Clínicas da Faculdade de Medicina de Ribeirão Preto (FMRP-USP), Avenida dos Bandeirantes, 3900, Campus Universitário, 14049-900 Ribeirão Preto, SP, Brazil

[3]Universidade de São Paulo, Faculdade de Filosofia, Ciências e Letras, Avenida Bandeirantes, 3900, Vila Monte Alegre, 14040-901 Ribeirão Preto, SP, Brazil

[4]Escola Superior de Saúde Egas Moniz (ESSEM), Campus Universitário Egas Moniz, Quinta da Granja, Travessa da Granja, 2829, 511, Monte de Caparica, Almada, Portugal

Correspondence to: **Marcelo Palinkas**
E-mail: palinkas@usp.br

Author contributions

G Gomes, E Verri, S Regalo and S Siéssere contributed to the design of the study. G Gomes, M Palinkas, AL Rossi, D Sousa, D Lattaro, J Hallak and L Ferreira made substantial contributions to data collection and analysis. G Gome, M Palinkas, S Regalo and S Siéssere edited the manuscript. All authors participated in drafting the manuscript, and author S Siéssere critically revised it. All authors read and approved the final version of the manuscript and agreed with the authors' order of presentation.

How to cite

GOMES GGC, PALINKAS M, VERRI ED, ROSSI ALL, DE SOUSA DS, LATTARO DH, HALLAK JC, FERREIRA LMA, REGALO SCH & SIÉSSERE S. 2026. Impact of CPAP treatment for obstructive sleep apnea on stomatognathic system function: a study on bite force, thickness, and temperature of the masseter and temporal muscles. An Acad Bras Cienc 98: e20251179. DOI 10.1590/0001-3765202620251179.

Manuscript received on October 30, 2025;
accepted for publication on January 3, 2026

Handling editor

João Duarte

Data availability

The data sets used and/or analyzed during the current study are available from the corresponding author on reasonable request.