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Masseter Deoxygenation in Adults at Risk for Temporomandibular Disorders

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Abstract

Patients with muscular temporomandibular disorder (TMD) present with abnormal oxygenation of the jaw muscles. Nonetheless, the deoxygenation pattern of jaw muscles of healthy subjects with frequent wake-time tooth-clenching episodes, who are at greater risk for TMD, has never been investigated. This case-control study compared the deoxygenation of the masseter during standardized tasks between TMD-free individuals with frequent self-reports of wake-time clenching and those with infrequent self-reports. University students ($N = 255$) filled out the Oral Behavior Checklist. Fourteen females with high versus low scores—high parafunctional (HP) group ($n = 7$, ≥80th percentile of score distribution) versus low parafunctional (LP) group ($n = 7$, ≤20th percentile)—completed 2 sessions during which they clenched at their maximum voluntary contraction (MVC) for 2 min and at 10% to 20% MVC for 20 min. Tissue oxygen saturation (StO_2) and changes in oxygenated hemoglobin, deoxygenated hemoglobin, and total hemoglobin of the masseter were measured via near-infrared spectroscopy and analyzed with a generalized mixed effect model. A significant interaction effect (task × study group) was found on all outcome measures, indicating that the deoxygenation pattern of the HP group differed from the LP group (all $P < 0.001$). MVC of the masseter induced an almost 5-times-greater reduction of StO_2 in the HP group as compared with the LP group ($P = 0.023$). However, the relative increase in StO_2 at rest after the MVC was similar between groups ($P > 0.05$). At the end of the prolonged MVC task (10% to 20%), the blood flow (change in total hemoglobin) was almost 6 times higher in the LP group as compared with baseline. On the contrary, it increased minimally in the HP group (all $P < 0.001$). Healthy individuals at risk for TMD have abnormalities in masseter deoxygenation. Future prospective studies are needed to test whether this contributes to the onset of muscular TMD.

Keywords: near-infrared spectroscopy, musculoskeletal pain, masseter muscle, mastication, facial pain, bruxism

Introduction

Temporomandibular disorders (TMDs) are the most common cause of orofacial pain, significantly affecting quality of life (Cioffi et al. 2014; Slade et al. 2016). Oral behaviors such as tooth clenching contribute to the onset, progression, and maintenance of TMD (Huang et al. 2002; Michelotti et al. 2010).

Wake-time tooth clenching is highly prevalent among people with TMD (Michelotti et al. 2010). Experimental clenching induces TMD-like symptoms among healthy subjects (Farella et al. 2010; Koutris et al. 2013). The contributing role of oral behaviors to TMD was further supported by a prospective cohort study (Ohrbach et al. 2013) and by the significant pain reduction after habit-reversal treatment (Gramling et al. 1996; Townsen et al. 2001; Durham et al. 2016).

Wake-time clenching and TMD myalgia could be linked to impaired blood perfusion and jaw muscle deoxygenation. In healthy subjects, sustained jaw muscle activity induces intramuscular hypoxia, pain, fatigue, and the onset of TMD-like symptoms (Delcanho et al. 1996; Suzuki et al. 2016). In patients with TMD myalgia, masseter oxygenation during sustained isometric contraction is reduced as compared with that of healthy controls (Delcanho et al. 1996). However, it is unknown whether abnormalities in jaw muscle deoxygenation may be already present among healthy individuals at a greater risk of TMD.

Muscle deoxygenation can be investigated with near-infrared spectroscopy (NIRS) to identify the concentration of oxygen in a specific region, termed tissue oxygen saturation (StO_2). StO_2 is computed as the ratio (%) between oxygenated hemoglobin (O_2Hb) and total hemoglobin (tHb = oxygenated + deoxygenated hemoglobin [HHb]). An increase in StO_2 could indicate greater availability of O_2Hb due to increased blood flow or reduced metabolic activity. However, its decrease could indicate decreased blood flow or increased metabolic activity (Boushel et al, 2001).

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A supplemental appendix to this article is available online.

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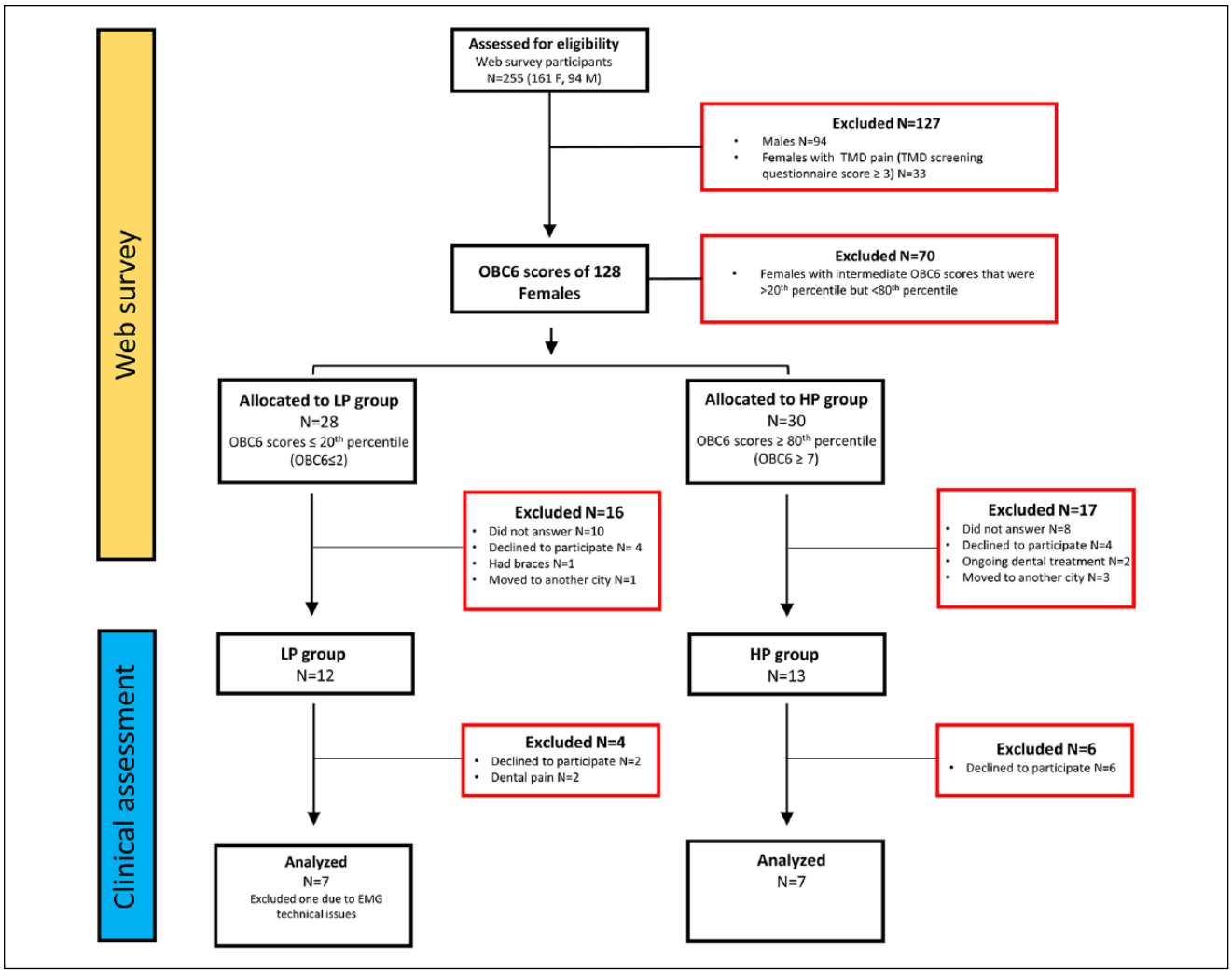

Figure 1. Recruitment of research participants and allocation to study groups. F, female; HP, high parafunctional; LP, low parafunctional; M, male; OBC6, 6 items from the Oral Behavior Checklist; TMD, temporomandibular disorder.

This study aimed to compare the deoxygenation of the masseter during standardized tasks between TMD-free participants with frequent self-reports of wake-time clenching (at a greater risk for TMD; Ohrbach et al. 2013) and those with infrequent self-reports (at a lower risk). It was hypothesized that the pattern of deoxygenation of the masseter would differ between these groups.

Materials and Methods

Participants

This case-control study was conducted at the University of Toronto between May and August 2017. University students ($N = 255$) completed the Oral Behavior Checklist (Markiewicz et al. 2006) through a web survey (Chow and Cioffi 2018; Fig. 1). Scores from 6 behaviors from the checklist (OBC6)—characterized by pressing attitudes against soft tissues, objects, or teeth—were summed and tabulated (Cioffi et al. 2018): 1) grinding teeth together during waking hours; 2) clenching teeth together during waking hours; 3) pressing, touching, or holding teeth together during times other than eating; 4) biting, chewing, or playing with tongue, cheeks, or lips; 5) holding bite objects between the teeth, such as hair, pipe, pencils, pens, and fingers; and 6) chewing gum. Men were excluded, as were women if they had a TMD pain screening questionnaire score ≥3 (Gonzalez et al. 2011). Two groups were selected: high parafunctional (HP) from the ≥80th percentile of the frequency distribution and low parafunctional (LP) from the ≤20th percentile (Fig. 1).

Exclusion criteria included dental fixed prostheses (≥3 teeth), ongoing orthodontic or dental treatment, neurologic disorders, intake of drugs affecting the central nervous system or masticatory muscles, orofacial pain, severe malocclusion/craniofacial deformities, and/or asymmetries. Exclusion criteria were evaluated with the survey and a clinical examination (Schiffman et al. 2014).

NIRS and Ultrasonography

A wireless NIRS device with standard nominal 760- and 850-nm wavelengths (PortaLite mini; Artinis Medical Systems BV) was used to measure StO_2, O_2Hb, HHb, and tHb (Boushel et al. 2001) of the right masseter. The device has 1 receiver and 3 transmitter optodes, with interoptode distances of 16, 21, and 26 mm. The skin was cleaned with alcohol and the optode placed over the belly of the masseter, as detected with manual palpation. Hypoallergenic tape secured the NIRS optode in position and limited extraneous light. Time-synchronized changes in chromophore levels of O_2Hb, HHb, tHb, and StO_2 were monitored in real time, sampled at 10 Hz, filtered (moving Gaussian), and stored for analysis with Oxysoft software (Artinis Medical Systems).

Ultrasonography (Vivid i Diagnostic Ultrasound System; GE Healthcare) connected to a linear array transducer (13 MHz) was used to determine the thickness of the subcutaneous tissue and the masseter (Serra et al. 2008) during rest and maximum voluntary contraction (MVC). The measurements were repeated twice to the nearest 0.1 mm by a single operator (N.S.). The average of the measurements (rest and MVC) was computed to determine which interoptode distance to use for data analysis (i.e., the one reaching the center of the muscle belly).

Surface Electromyography

After the cheek was cleaned with a gel (Nuprep; Weaver and Company), surface electromyography (sEMG) electrodes (Duotrode, 12.5-mm diameter; Myotronics) were mounted in pairs (interelectrode distance of 19 mm) on the left masseter (Castroflorio et al. 2005). The electromyographic (EMG) signals were amplified, band pass filtered between 20 and 400 Hz (PowerLab 8/35; ADInstruments) with analog to digital sampling at 10 kHz, and stored for data analysis (LabChart 8.1; ADInstruments).

Pressure Pain Thresholds

Pressure pain thresholds (PPTs; Ohrbach and Gale 1989) were measured with a digital algometer (Wagner Force One, FDIX 25; Wagner Instruments) with a 1-cm^2 rubber tip to evaluate jaw muscle tenderness (Cioffi et al. 2017; Cioffi et al. 2018). The tester (N.S.) was blinded to the allocation of subjects to groups. Pressure was increased at approximately 20 kPa/s. When pain was perceived, subjects rang a bell, which halted the pressure stimulus, and the PPT was read from the display. In random order, measurements were collected 3 times at 1-min intervals from the superficial masseter and anterior temporalis muscles (both sides) and thenar eminence in the right hand.

Experimental Protocol

Height, weight, and blood pressure were measured. All testing was performed in the sitting position. PPTs were evaluated at baseline and at the end of the protocol. Next sEMG electrodes and NIRS optodes were positioned. Participants closed their eyes, breathed slowly, relaxed their jaw muscles, and kept their teeth apart slightly for 30 s. Thereafter, they were asked to clench their teeth as hard as possible for 3 s, 3 times, separated by 5-s intervals. The average EMG amplitude recorded during 30-s rest and 3-s MVCs were set as 0% and 100% MVC, respectively.

Participants underwent 2 experimental sessions. In session A, participants clenched at MVC for 2 min. A 2-min rest was provided before and after the MVC (rest A and B). In session B, they performed two 10-min clenching tasks (MVC10A and MVC10B) by targeting 10% to 20% sEMG on a computer monitor, shown as a green band on the EMG output by LabChart. Three rests (rest A, 2 min; rest B, 1 min; rest C, 2 min) were interspersed before, in between, and after MVC10A and MVC10B.

At the end of session A and every 2 min during MVC10A and MVC10B (session B), participants indicated if they had tooth pain (yes/no) and rated pain and fatigue of the right/left masseter and temporalis with a visual analog scale (VAS; 0 to 100 mm; left endpoint, *no pain/fatigue*; right endpoint, *the worst pain/fatigue I can imagine*). Thereafter, the right masseter was scanned with ultrasound. The experimenter (N.S.) was blinded to group allocation.

This study conformed with STROBE guidelines for observational studies and was approved by the local Research Ethics Board (No. 34585). All participants provided informed consent.

Data Analysis

Primary outcome measures were StO_2 and changes (Δ) in O_2Hb, HHb, and tHb. Data normality was tested with the Kolmogorov-Smirnov test. Between-group differences in body mass index (BMI) and blood pressure (during rest and MVC) were tested with independent sample *t* tests. Analysis of covariance was used to test differences in masseter thickness after controlling for BMI. Multivariate analysis of variance was used to test the effect of the experimental session on PPTs in both groups with the mean of the 2 trials obtained at each location, after discarding the first measurement.

A generalized mixed effect model was used to test between- and within-group differences in primary outcome measures. Each subject was included in the model as a random factor, while task and study group (HP vs. LP) were included as fixed factors. The Bonferroni method was used to adjust for multiple comparisons ($\alpha = 0.05$).

Differences in the frequency of tooth pain in both groups were assessed with chi-square tests. VAS scores for pain/fatigue, as compared between right and left sides and between muscles, were not different (all $P > 0.05$) and were averaged for overall scores. The Mann-Whitney *U* test was used to test between-group differences in VAS scores for pain/fatigue. Correlations between pain/fatigue VAS scores and time were tested by computing Spearman's correlation coefficients.

The statistical significance was set at $P < 0.05$. SPSS 24 (IBM Corp.) was used. The allocation of subjects to groups was masked in the final data set, blinding the operator (I.C.).

Results

A flow diagram depicting participant recruitment and information about exclusion is reported in Figure 1. The OBC6 scores of 128 eligible women out of 255 students were examined (≤20th and ≥80th percentiles of OBC6 score distribution). Initially, 70 women were excluded because of intermediate scores. The final sample included 7 females (mean ± SD age, 23.7 ± 5.5 y) with OBC6 scores ≥80th percentile (OBC6 ≥8; HP) and 7 females (27.6 ± 5.8 y) with scores ≤20th percentile (OBC6 ≤3; LP; Fig. 1). This sample was sufficient to achieve a power of 80% for assessing between-group differences in relative changes of StO_2 during MVC ($d = 1.4$, $\alpha = 0.05$).

The study groups had similar BMI (HP, 23.5 ± 3.2; LP, 20.8 ± 3.17 g/m^2) and blood pressure (all $P > 0.05$). The mean masseter thickness during rest (HP, 11.1 ± 4.2 mm; LP, 12.8 ± 4.5 mm; $P = 0.485$) and MVC (HP, 13.8 ± 4.2 mm; LP, 14.1 ± 4.9 mm; $P = 0.554$) were similar between groups. PPTs at all locations were not different between groups at baseline and did not change after the experiment, $F(3, 21) = 1.745$, $P = 0.189$, Wilk's $\Lambda = 0.800$, partial $\eta^2 = 0.200$.

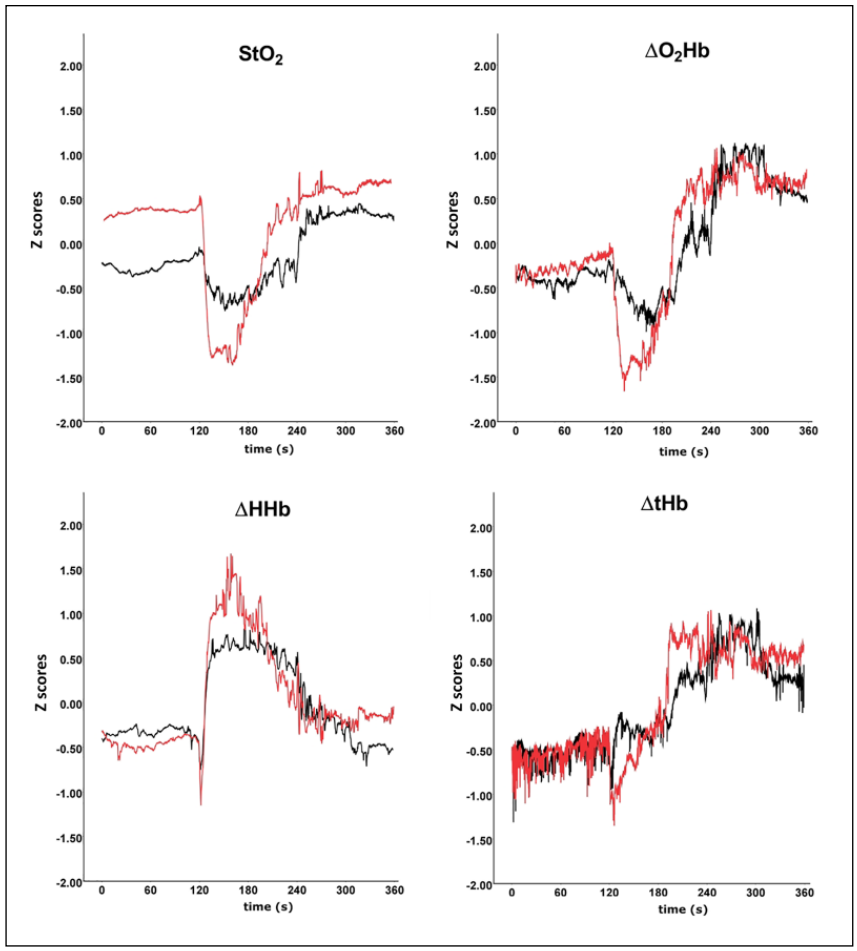

Figure 2. Slopes (mean *Z* scores) for StO_2 and changes in chromophores (ΔO_2Hb, ΔHHb, and ΔtHb) measured during session A (rest A, 2 min; maximum voluntary contraction, 2 min; rest B, 2 min) in the high (HP, red) and low (LP, black) parafunctional groups. HHb, deoxygenated hemoglobin; O_2Hb, oxygenated hemoglobin; StO_2, oxygen tissue saturation; tHb, total hemoglobin. This figure is available in color online.

Hemodynamic Parameters during Session A

Significant interactions task × group ($P < 0.001$) were found for StO_2, ΔO_2Hb, ΔHHb, and ΔtHb (Figs. 2, 3; Appendix Table 1). As compared with rest A, StO_2 decreased during MVC in LP and HP (all $P < 0.001$) and was the greatest during rest B (all $P < 0.001$). StO_2 did not differ between groups ($P > 0.05$) during the tasks. However, the relative decrease in StO_2 during MVC was greater in the HP group (−10% ± 8.0%; LP, −2.2% ± 3.6%; $P = 0.023$; Fig. 4).

ΔO_2Hb was greater during rest B as compared with baseline rest A and MVC in the HP and LP groups (all $P < 0.001$). However, in the LP group, no differences were found between rest A and MVC ($P = 0.074$; Fig. 3).

ΔHHb was the greatest in both groups during MVC (all $P < 0.001$) but was greater in rest B than rest A in only the HP group ($P < 0.001$; LP, $P = 0.065$). ΔtHb increased significantly from baseline (rest A) to rest B in both groups (all $P < 0.001$). There were no between-group significant differences for ΔO_2Hb, ΔHHb, and ΔtHb ($P > 0.05$; Fig. 3).

At the end of session A, VAS scores for muscle pain (LP, 34.3 ± 32.8 mm; HP, 34.6 ± 31.4 mm; $P = 0.967$) and fatigue (LP, 33.9 ± 29.1 mm; HP, 34.4 ± 34.6 mm; $P = 0.954$) and frequency of tooth pain ($\chi^2 = 0$, $df = 1$, $P > 0.99$) were similar between groups.

Hemodynamic Parameters during Session B

A significant task × group interaction ($P < 0.001$) was found for StO_2. In the LP group, StO_2 increased from rest A to rest C (all $P < 0.001$; Fig. 5, Appendix Table 2). No differences were found between MVC10A and MVC10B ($P = 0.935$). In HP, StO_2 decreased significantly during MVC10A and MVC10B as compared with rest A (all $P < 0.001$), and it increased during the following resting sessions (all $P < 0.001$). During rest A, StO_2 was significantly greater in the HP group than the LP group ($P = 0.044$).

A significant task × group interaction (all $P < 0.001$) was found for ΔO_2Hb, ΔHHb, and ΔtHb. ΔO_2Hb increased significantly throughout the session (all $P < 0.001$) in the LP group. In HP, ΔO_2Hb was the greatest during rest B and lowest during MVC10A (all $P < 0.001$). In both groups, there were no differences between rest C and MVC10B (all $P > 0.05$). ΔHHb was

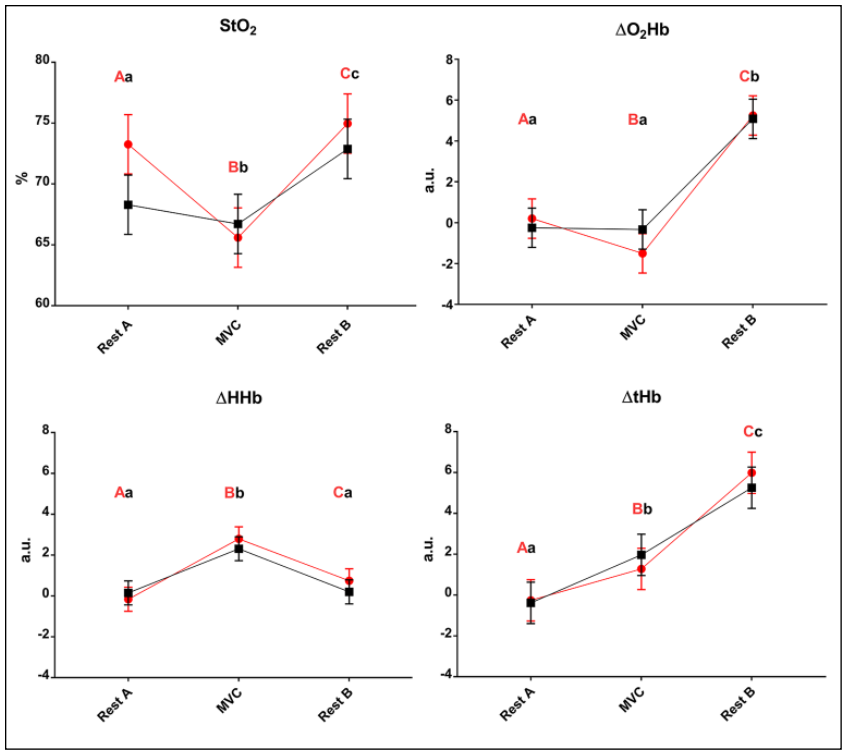

Figure 3. Mean values for StO_2 and changes in chromophores (ΔO_2Hb, ΔHHb, and ΔtHb) measured during session A (rest A, 2 min; MVC, 2 min; rest B, 2 min) in the high (HP, red circle) and low (LP, black squares) parafunctional groups. Error bars indicate SEM. Significant differences ($P < 0.05$) between tasks within the same group are indicated by different capital letters (HP/red group) or lowercase letters (LP/black group). Same letters indicate no significant differences between tasks. During session A, no between-group significant differences were found. au, arbitrary units; HHb, deoxygenated hemoglobin; MVC, maximum voluntary contraction; O_2Hb, oxygenated hemoglobin; StO_2, oxygen tissue saturation; tHb, total hemoglobin. This figure is available in color online.

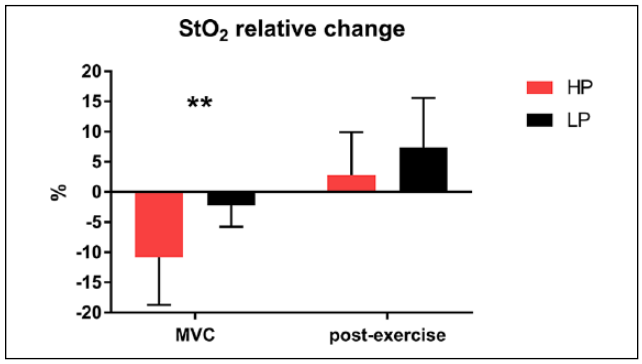

Figure 4. Mean value of StO_2 relative changes during MVC and at postexercise in the HP (red) and LP (black) groups. Error bars indicate SD. **Between-groups difference at $P < 0.05$. HP, high parafunctional; LP, low parafunctional; MVC, maximum voluntary contraction; StO_2, oxygen tissue saturation. This figure is available in color online.

the highest during MVC10B and the lowest during rest A (all $P < 0.001$) the LP group. In the HP group, it was the highest during MVC10A and MVC10B (but with no differences between these 2 time points, $P = 0.919$) and lowest in rest C (all $P < 0.001$). ΔtHb increased progressively, reaching the highest value during MVC10B and then decreased during rest C in both groups (all $P < 0.001$). There were no differences between rest B and MVC10B ($P = 0.967$) in the HP group and between rest B and rest C in the LP group ($P = 0.183$). No between-group differences in ΔO_2Hb, ΔHHb, and ΔtHb during the tasks were found (all $P > 0.05$).

There were no significant between-group differences in VAS pain and fatigue scores (median [IQR] pain: HP, 22 [51] mm; LP, 27 [43] mm; $P = 0.147$; fatigue: HP, 37 [46] mm; LP, 33 [59] mm; $P = 0.856$). Overall pain and fatigue VAS scores increased significantly with time in the HP and LP groups (pain: HP, $r = 0.188$, $P = 0.001$; LP, $r = 0.167$, $P = 0.005$; fatigue: HP, $r = 0.266$, $P < 0.001$; LP, $r = 0.274$, $P < 0.001$; Appendix Fig. 1).

Tooth pain was more frequently reported in the HP group ($\chi^2 = 29.1$, $df = 1$, $P < 0.001$) than the LP group.

Discussion

This study represents the first to examine the hemodynamic parameters of the masseter among healthy participants with higher versus lower risk of TMD. MVC of the masseter induced a 5-times-greater StO_2 reduction among subjects with a higher frequency of oral behaviors (HP) as compared with a lower frequency (LP). Nonetheless, the relative increase in StO_2 at the end of the session was similar between groups (Fig. 4). Moreover, during a prolonged submaximal contraction (10% to 20% MVC), StO_2 steadily increased among LP individuals. Conversely, it dropped significantly among HP ones, although they had greater StO_2 at baseline (Fig. 5). The blood flow through the muscle (tHb) was almost 6 times higher at the end of the submaximal exercise in the LP group as compared with baseline (Fig. 5). On the contrary, it increased minimally in the HP group. Of interest, the PPTs (Michelotti et al. 2012; Al-Harthy et al. 2016; Cioffi et al. 2018) and thickness of the masseter did not differ between the groups and fell within normal ranges (Reis Durao et al. 2017). These results suggest that the HP group had less oxygen than the LP group during the same clenching task, that postexercise hyperemia may not allow a quick recovery for HP subjects, and that mechanisms different from vascular compression may be responsible for limited oxygen delivery in the HP group. It was previously reported that those with myogenous TMD (Delcanho et al. 1996) had reduced oxygenation of the masseter as compared with healthy subjects. Our report revealed that such abnormality is already present among healthy individuals at risk of TMD.

For this study, we decided to exclude those with orofacial pain to eliminate a confounding variable, pain, which could have affected the measures and data analysis (Shimada et al. 2013). Also, we use a threshold of 10% MVC because a contraction of about 5% MVC is sufficient to bring the teeth into contact (Roark et al. 2003) and clenching episodes >10% MVC

are highly frequent among those with myogenous TMD (Cioffi et al. 2017).

In agreement with previous studies (Delcanho et al. 1996; Suzuki et al. 2016), both exercises significantly influenced the hemodynamic parameters. During MVC, the greater relative decrease in StO_2 of the HP group versus the LP group (Fig. 4), with the similar increase in tHb (Fig. 3), could suggest a greater mismatch between oxygen delivery and oxygen consumption in the HP group. A vigorous isometric contraction (MVC) has 2 major effects on the oxygenation of the muscle fibers (Delcanho et al. 1996). First, the shortening and subsequent lateral expansion of muscle fibers induce a mechanical compression of blood vessels (which contrasts vasodilation) and contributes to local ischemia (drop of StO_2). After a few seconds, the local blood perfusion can increase through vasodilation, and more oxygen is delivered to the muscle fibers (increase of StO_2), favoring oxidative metabolism (exercise hyperemia). However, at ≥70% MVC or >30% MVC in other muscles (Kahn et al. 1998; van Beekvelt et al. 2003), the blood flow to the muscle is severely impaired, and the extravascular mechanical compression overcomes the vasodilation response (Delcanho et al. 1996). After the exercise is completed, postexercise hyperemia occurs, with StO_2 becoming greater than preexercise. The changes that we found in StO_2, O_2Hb, HHb, and tHb at the end of session in both groups are consistent with a postexercise hyperemia (Fig. 3).

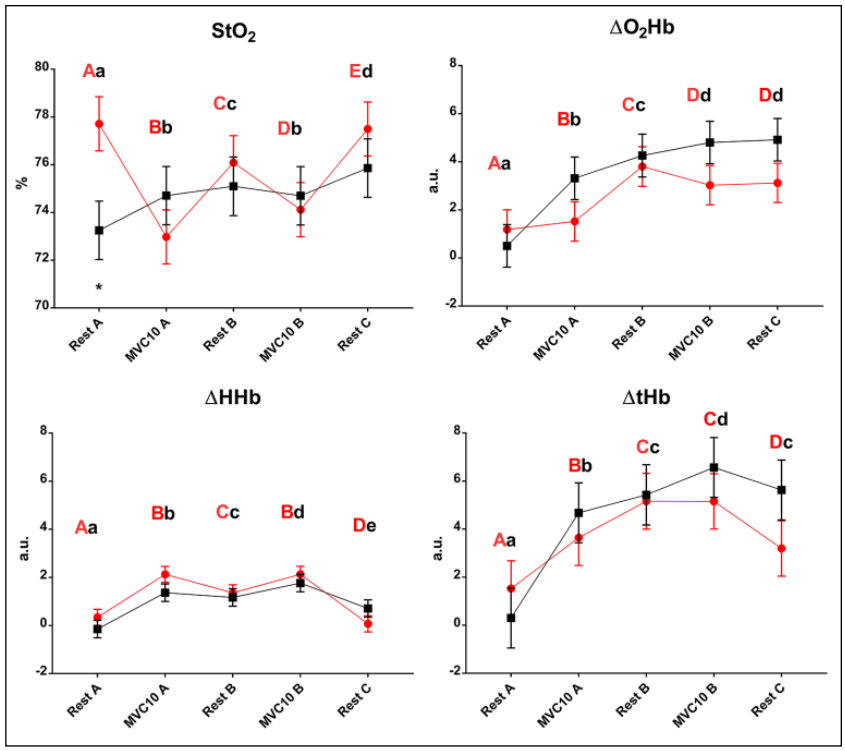

Figure 5. Mean values for StO_2 and changes in chromophores (ΔO_2Hb, ΔHHb, and ΔtHb) measured during session B (rest A, 5 min; MVC10A, 10 min at 10% to 20%; rest B, 2 min; MVC10B, 10 min at 10% to 20%; rest C, 5 min) in the high (HP, red circles) and low (LP, black squares) parafunctional groups. Error bars indicate SEM. Significant differences ($P < 0.05$) between tasks within the same group are indicated by different capital letters (HP/red group) or lowercase letters (LP/black group). Same letters indicate no significant differences between tasks. *Between-group difference at $P < 0.05$. au, arbitrary units; HHb, deoxygenated hemoglobin; MVC, maximum voluntary contraction; O_2Hb, oxygenated hemoglobin; StO_2, oxygen tissue saturation; tHb, total hemoglobin. This figure is available in color online.

The postexercise hyperemia is a critical factor to investigate whether blood flow is sufficient to meet the metabolic demand (Kahn et al. 1998). Insufficient oxygen delivery may be responsible for an early onset of anaerobic metabolism, with release of proalgesic mediators (Delcanho et al. 1996). This predisposes to a greater risk of tissue damage and muscle injuries. Depending on the antioxidant capacity of muscle, reperfusion injury could also occur (Walker 1991). Interestingly, during MVC, the concentration of tHb did not differ between the HP and LP groups (Fig. 3). This leads to a hypothesis that the differences in StO_2 relative changes during MVC are not related to a reduced blood flow during MVC but probably to abnormalities in oxygen extraction/delivery at a local level (Willingham and McCully 2017).

A prolonged submaximal contraction induced decreases of StO_2 in the HP group but not for the LP group. Below 20% of MVC, vasodilating response overcomes the extravascular mechanical compression of the vessels (Delcanho et al. 1996), and the blood flow to the muscle is minimally impaired. It appears that the muscle blood flow provided a better match to oxygen demands during the submaximal contraction among LP individuals than HP.

The possible sequelae of this abnormal pattern of deoxygenation need to be further investigated. It may be responsible for the early onset of anaerobic metabolism during exercise (van Beekvelt et al. 2003) and the increased production of oxygen radicals with poor endothelial function and limited perfusion; it may also cause structural abnormalities within the muscle (Close et al. 2005; Pereira et al. 2015). These significant changes could be responsible for TMD myalgia onset. Also, since patients with TMD spend most of their time keeping their teeth in contact (Cioffi et al. 2017), abnormalities in deoxygenation may contribute to the maintenance and progression of the disease.

This study has some limitations. First, we included only females to eliminate sex-related confounders (Torisu et al. 2006; Slade et al. 2016); therefore, we cannot draw conclusions on men. Second, the EMG visual feedback was constructed with data from the contralateral muscle because it was not possible to fit both the sEMG electrodes and the NIRS probe on the same muscle. However, all participants had Angle class I and no facial asymmetries. As such, it is likely that this issue did not significantly affect measurements. Third, we used EMG feedback instead of bite force. The use of an occlusal transducer for the measurement of bite force could have affected muscle contraction patterns (Augusti et al. 2015).

In conclusion, this study revealed that healthy individuals with frequent wake-time clenching have abnormal masseter deoxygenation. Future prospective studies are needed to test whether this contributes to the onset of muscular TMD. Our study opens new strategies with NIRS for the early screening of patients at risk for TMD myalgia.

Author Contributions

N. Shah, contributed to design, data acquisition, and interpretation, drafted and critically revised the manuscript; L. Melo, contributed to design, data acquisition, and interpretation, critically revised the manuscript; W.D. Reid, contributed to conception, design, data analysis, and interpretation, drafted and critically revised the manuscript; I. Cioffi, contributed to conception, design, data acquisition, analysis, and interpretation, drafted and critically revised the manuscript. All authors gave final approval and agree to be accountable for all aspects of the work.

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