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ORIGINAL ARTICLE

Autonomic responses to tooth clenching and handgrip test

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ABSTRACT

Objective: The aim was to study the differences in autonomic nervous system activation between maximal tooth clenching task and handgrip test during and after the tasks. Also, the possible activation of trigeminocardiac reflex during the clenching task was explored.

Material and methods: We compared autonomic responses to maximal tooth clenching and handgrip in 28 participants. Responses in heart rate variability, heart rate, and blood pressure were evaluated before, during, and after tests. Although all study participants were considered healthy during recruitment, 14 of them showed painful temporomandibular disorders in the clinical examination, which was taken into account in the analyses.

Results: Handgrip and tooth clenching caused similar autonomic responses. However, tooth clenching seemed to activate the trigeminocardiac reflex shown as clenching-related vagal activation. The painful signs of temporomandibular disorders may interfere with the heart rate variability both at the baseline and during both tests causing significant variation in them.

Conclusions: Both handgrip and tooth clenching affect the autonomic nervous system function. Tooth clenching differs from the handgrip due to trigeminocardiac reflex. Painful signs of temporomandibular disorders are interfering with the results of the tests and maybe underestimated in the studies of autonomic responses to both tasks.

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Introduction

The autonomic nervous system (ANS) regulates and integrates internal organs' functioning unconsciously. Analysis of heart rate variability (HRV) is one of the most popular methods to evaluate ANS responses [1]. HRV denotes changes in the interbeat interval (time interval between consecutive heartbeats), which are a consequence of the continuous regulation of the heart rate by the ANS [1,2]. Analysis of HRV allows differentiation of the sympathetic and parasympathetic nervous systems' activity [2].

As it is almost impossible to directly measure the function of the ANS, there is a group of clinical tests to measure the end-organ responses to the perturbations caused by clinical or experimental provocations [3]. One of those is the handgrip test (HGT) which induces an increase in heart rate (HR) and cardiac output, as well as raises systolic (SBP) and diastolic (DBP) blood pressures [4–6] by evoking reflex responses in the autonomic nervous system. Thus, HGT is a useful tool for the studies of autonomic system functions.

During HGT the contraction of the hand's muscles causes the autonomic nerve responses. However, the autonomic responses of contraction of masticatory muscles caused by tooth clenching are not well known. The different locations of the activated muscle groups might cause different responses. One of the reasons is that the masticatory muscles are innervated by the trigeminal nerve and the clenching could activate the so-called trigeminocardiac reflex (TCR) which affects the autonomic nerve function [7]. Zhang et al. have studied the effects of static unilateral clenching tasks and compared its effects on ANS function with those of the HGT. Sympathetic activity during unilateral jaw clenching tasks in healthy participants turned out to be lower than that during HGT [8]. However, the studied clenching task was unilateral, it was not performed with maximal clenching force and post-test changes were not monitored [8]. Chewing has shown similar effects by raising HR and blood pressure (BP) apparently due to modulation of the autonomic nervous activity [9–11]. However, opposite findings have also been made. In Takeuchi et al. [12], prolonged low-level tooth clenching lowered HR. In addition, the mandibular extension has been shown to induce hypotensive and bradycardic effects [13]. Studies of bruxism have shown that tooth clenching is a way to relieve stress during psychic

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overloading [14] likely due to its effects on the activity of the autonomic nervous system and hypothalamic-pituitary-adrenal axis [15]. Altogether, more studies focusing on the effects of maximal tooth clenching (MTC) on the autonomic nervous system's responses would be needed.

Autonomic responses during MTC may be influenced by pain if the participant suffers from temporomandibular disorders (TMD). Indeed, the frequency of painful TMD signs (pTMD) is as high as 38% in the Finnish population. [16]. However, TMD's pathophysiology is probably multifactorial and there are different physiological, psychological, genetical, and behavioural factors, which may expose, onset or maintain TMD [17]. Also, a new concept of TMD, being a stochastic process between multiple risk factors has been proposed [18]. Yet, it is linked to ANS dysfunction [19–23] as patients with TMD have hyperactive sympathetic and reduced parasympathetic nervous system activity even when resting [19]. Since it may affect the results, the pTMD was taken into account in the present study.

The aim of this study was to determine what kind of differences there are in ANS activation between MTC and HGT during and after the tasks. The possible TCR in connection with MTC was also a subject of interest. Because it turned out that quite a few of the 'healthy' participants were found to have subclinical TMD, found in their clinical TMD examination, we wanted to study, in addition, if there were differences in the autonomic responses between participants with and without pTMD.

Materials and methods

The study plan was approved by the Research Ethics Committee of the Hospital District of Northern Savo, Kuopio, Finland. All participants provided written consent. The study group consisted of 28 healthy participants (17 women and 11 men). The participants were students and staff of the University of Eastern Finland and their relatives or friends. The average age of the participants was 31.8 ± 1.8 years. The average duration of performed MTC until volitional exhaustion was 97.9 ± 8.2 s, 92.1 ± 10.1 s in women and 106.7 ± 14.2 s in men. Healthy adults were included as study subjects but those with any general disease and/or medication were excluded. The participants did not have any additional pain conditions or any neurologic or cardiovascular diseases. None of the participants met the diagnostic criteria for chronic migraine or medication overuse headache which were screened as the study group was the same as the healthy controls used by Zaproudina et al. [24]. Clinical oral examination was performed by a TMD specialist and subclinical findings of pTMD were documented. Of the 28 participants, 14 turned out to have pTMD (13 muscle-related, one joint-related), which, however, did not show subjective symptoms of TMD. Nine of those subjects were women. Self-reported mood (on scale 0–21, where 0 = no depression and 21 = depression) [25] and presence of autonomic disorders such as sweating, rumble, faintness (13-dimensional questionnaire, 0–39, where 0 = no symptoms and 39 = the worst symptoms possible) scores were collected. The autonomic disorder score scale was also used by Zaproudina et al. [24]. However, it has not been validated, and accordingly, an English translation of it has been presented in Supplementary Appendix 1 for the present report.

The protocol of MTC included two 5 s periods of clenching and 5 s breaks followed by maximal clenching until exhaustion or if any pain was felt by the participant [24]. A Portapres device (Finapres Medical Systems, Amsterdam, the Netherlands) was used to measure systolic (SBP) and diastolic (DBP) blood pressures [24]. A modified chest lead V5 with Biopac MP150 system (V5Biopac Systems Inc., Goleta, CA, USA) was used to record the electrocardiogram for HR and HRV analysis [24]. To verify the duration and intensity of teeth clenching the electromyography (EMG) registrations were performed using an ME6000 biosignal monitor (Mega Electronics Ltd, Kuopio, Finland). The electrodes were placed on both masseters, temporalis, and trapezius muscles. The reference electrodes were placed behind the ear. The EMG registrations were done on 21 of the 28 participants due to a lack of resources during the last seven recordings. EMG sample frequency was 1000 Hz. The BP measurements failed in two participants due to technical difficulties. Therefore those two participants were left out from the BP analysis.

For HGT, an adjustable hand dynamometer (Hand Dynamometer, Saehan, Changwon, South Korea) was used. For each participant, the dynamometer was adjusted individually. The forearm was positioned approximately horizontally while the elbow was on the side, not leaning to the flank area. Cues were given to the participants 1 min, 30 s, and 10 s before starting the task. The protocol of HGT included 3 min of gripping the dynamometer using 30% of the participants' maximal gripping force, which was determined individually using the dynamometer before any of the measurements related to the tasks in the actual experiments.

Data analysis

HRV was analysed using Kubios HRV software [26]. In addition to mean HR, the standard deviation of beat intervals (SDNN), root mean square of successive beat interval differences (RMSSD), low frequency (LF: 0.04–0.15 Hz) and high frequency (HF: 0.15–0.4 Hz) powers and LF/HF ratio were computed, as well as the mean values of HR, LF and HF power, LF/HF ratio, RMSSD and SDNN ($n = 28$), and SBP and DBP in participants ($n = 26$) 2 min (mean/SD) before and after MTC and HGT. The on-test value of MTC was the mean value of the parameter during the whole clenching time and in HGT it was the 2-min mean value. On-test changes were computed by subtracting the mean baseline values of the mean on-test values. Post-test changes were computed correspondingly by subtracting the mean baseline values of the mean post-test values. Also, maximal and minimal on-test and post-test values of HR, SBP and DBP were determined. Maximal increases were computed by subtracting the mean baseline values of the mean maximal values. Maximal decreases were absolute values computed by subtracting the mean baseline values of the minimal values. The SPSS Statistics 25.0 (IBM Corp., Armonk, NY, USA) was used for the

statistical analyses. Mann-Whitney *U*-test, *t*-test, and Fisher's Exact test were used to examine differences in characteristics between the study subjects. All of the HRV, HR, and BP parameters were normally distributed except for LF/HF ratio, RMSSD, and Std. RR. Normality was assessed using Shapiro–Wilk test and visual check using histograms with normal curve overlay. Paired samples *t*-test and Wilcoxon signed-rank test were used accordingly to detect if there were any differences in baseline values between the two tests because two different tests were compared in the same participant group. Mann-Whitney *U*-test was used to detect if there were any differences in the measured parameters between participants with or without subclinical pTMD since a nonparametric test of two independent samples was indicated. MTC- and HGT- induced changes in the mean values of HRV parameters and blood pressures were compared between the tests with a Linear mixed model, adjusted for age and sex. A stepwise linear regression analysis was used to identify the predictors of the measured changes. The level $p < .05$ was considered significant.

Results

At the baseline, mean levels of HR and HRV parameters and BP levels did not differ (Paired samples *t*-test) between HGT and MTC (Table 1). With Mixed model analysis, the dynamics of changes in mean values of HRV parameters did not differ between the tests except for that of LF power [Estimate (Est.) 0.1, confidence interval (CI) 0.005; 0.19, $p < .05$] and SDNN (Est. 4.32, CI 0.56; 8.07 $p < .05$), both being lower for HGT compared to MTC. Also, the changes in SBP (Est. −5.54, CI −8.98; −2.09, $p < 0.01$) and DBP (Est. −4.03, CI −5.92; −2.14, $p < .01$) differed between the tests with a greater DBP on-test level increase in HGT than in MTC (test × time interaction, $p < .05$).

As compared to baseline levels, the on-test decrease in SDNN (Est. −5.02, CI −10.0; −0.04, $p < .05$) and increase in SBP (Est. −4.57, CI −9.13; −0.01, $p = .05$) and on-test DBP levels (Est. −4.4, CI −6.91; −1.88, $p < .01$) were lower in MTC than in HGT (Table 1). Also, there was a test x time interaction for DBP (Est. −5.84, CI −10.67; −1.01, $p < .05$).

As compared to baseline levels, the post-test levels of SBP (Est. −4.62, CI −7.75; −1.48, $p < .01$) and DBP (Est. −2.35, CI −3.95; −0.75, $p = .01$) were greater in HGT (Table 1).

The possible determinants of MTC- and HGT-induced autonomic changes (age, sex, height, weight, pTMD, mood and autonomic scores, duration of test, the baseline values of measured parameters) were entered stepwise into the model to study, whether they were independently associated with the measured changes (Table 2). Sex and autonomic scores were found to predict the baseline levels of HRV parameters. On-test levels in both tests were dependent on test duration, baseline levels, autonomic and mood scores but also on the presence of pTMD for autonomic parameters in both MTC and HGT. Post-test levels in both tests were dependent on baseline levels and sex. For the post-test changes, pTMD and mood were predictors of autonomic changes only in HGT. The duration of the test predicted the post-test levels of HF power and DBP in MTC.

Of the 28 participants, 14 had pTMD exposed in the clinical examination although they did not have any subjective symptoms. There were some differences between participants with and those without pTMD. For example, baseline

Table 1. The mean baseline, on-test, post-test, test changes, maximal and minimal values of heart rate (HR), low (LF) and high frequency (HF) power, LF/HF ratio, square root of the mean squared differences of successive RR intervals (RMSSD) and standard deviation of beat intervals (SDNN) ($n = 28$), systolic (SBP) and diastolic (DBP) blood pressures of maximal tooth clenching (MTC) and handgrip test (HGT) in the participants ($n = 26$).

Parameter	Baseline (mean/SD)	During test (mean/SD)	Post-test (mean/SD)	On-test changes (mean/SD)	Post-test changes (mean/SD)
MTC (Duration at mean 97.9 s (8.2))					
HR	64.34 (7.13)	73.42 (9.06)	62.23 (8.70)	9.08 (7.96)	−2.11 (3.41)
LF power	3.07 (0.36)	2.93 (0.37)*	3.04 (0.42)	−0.13 (0.43)	−0.03 (0.41)
HF power	2.97 (0.38)	2.76 (0.54)	3.00 (0.34)	−0.21 (0.56)	0.03 (0.25)
LF/HF	1.73 (1.52)	2.49 (2.83)	1.57 (1.39)	0.77 (3.13)	−0.15 (1.93)
RMSSD	0.06 (0.02)	0.05 (0.03)	0.06 (0.03)	−0.01 (0.03)	0.01 (0.01)
Std. RR	0.05 (0.02)	0.05 (0.02)*	0.06 (0.02)	−0.01 (0.02)*	0.00 (0.01)
SBP	148.32 (18.04)	167.05 (24.19)*	146.16 (18.18)*	18.72 (15.26)*	−2.16 (6.38)*
DBP	77.75 (18.51)	87.85 (18.49)*	76.85 (17.09)*	10.10 (7.76)*	−0.90 (2.94)
Maximal values		Max.	Min.	Max. increase	Max. decrease
HR		81.07 (8.75)	59.21 (9.64)	16.17 (6.97)*	6.03 (3.58)
SBP		177.43 (26.63)*	140.44 (20.49)	29.34 (19.11)*	8.22 (6.92)
DBP		97.01 (21.89)*	72.42 (19.46)	19.29 (10.06)*	5.41 (3.71)
HGT (Duration at mean 175.9 s (2.9))					
HR	64.14 (8.21)	73.84 (9.89)	62.57 (9.13)	9.70 (6.74)	−1.56 (4.65)
LF power	2.98 (0.44)	2.84 (0.36)*	2.93 (0.40)	−0.14 (0.42)	−0.06 (0.38)
HF power	2.96 (0.37)	2.66 (0.44)	3.01 (0.34)	−0.30 (0.41)	0.05 (0.29)
LF/HF	1.65 (18.2)	2.37 (2.38)	1.35 (1.36)	0.73 (2.45)	−0.30 (1.19)
RMSSD	0.06 (0.03)	0.04 (0.02)*	0.06 (0.03)	−0.02 (0.03)	0.00 (0.02)
Std. RR	0.05 (0.02)	0.04 (0.02)*	0.05 (0.02)	−0.01 (0.02)*	0.00 (0.01)
SBP	148.60 (18.45)	175.20 (21.23)*	153.81 (18.86)*	31.79 (35.88)*	10.40 (33.50)*
DBP	78.73 (17.72)	94.89 (17.72)*	79.56 (17.74)*	18.62 (15.25)*	3.28 (15.88)
Maximal values:		Max.	Min.	Max. increase	Max. decrease
HR		82.65 (10.58)	60.76 (10.04)	17.60 (8.37)*	4.86 (4.92)
SBP		189.95 (22.82)*	146.06 (19.62)	40.31 (19.20)*	6.06 (9.39)
DBP		105.32 (17.61)*	75.09 (18.01)	25.89 (11.63)*	4.85 (5.16)

*$p < .05$ between the MTC and HGT tests; Paired samples *t*-test, Wilcoxon signed-rank test, Mixed model analysis.

Table 2. Predictors of changes of baseline, on-test and post-test values of heart rate (HR), low (LF) and high frequency (HF) power, LF/HF ratio, square root of the mean squared differences of successive RR intervals (RMSSD) and standard deviation of beat intervals (SDNN) ($n = 28$), and systolic (SBP) and diastolic (DBP) blood pressures in the participants ($n = 26$) in maximal tooth clenching (MTC) and handgrip test (HGT).

Parameter	Baseline Predictor, β; Sign.	On-test Predictor, β; Sign.	Post-test Predictor, β; Sign.
MTC			
HR	Autonomic scores 0.46*	Baseline, 0.49** Duration, −0.40*	Baseline, 0.93**
LF power	ns	Duration, 0.56**	Baseline, 0.48*
HF power	Sex, −0.41*	Duration, 0.47** Baseline, 0.42* pTMD, 0.36*	Baseline, 0.77** Duration, 0.30*
LF/HF	Sex, 0.52**	Age, −0.44*	Baseline, 0.69**
RMSSD	ns	Baseline, 0.48** pTMD, 0.44** Duration, 0.35*	Baseline, 0.91 **
SDNN	ns	Baseline, 0.59** pTMD, 0.61** Duration, 0.38** Autonomic scores, −0.34**	Baseline, 0.82** Sex, −0.24*
SBP	ns	Baseline, 0.86** Mood scores, 0.31*	Baseline, 0.93**
DBP	ns	Baseline, 0.93** Mood scores, 0.27** Autonomic scores, −0.16*	Baseline, 0.98** Duration, 0.07* Age, 0.06*
HGT			
HR	Autonomic scores 0.50*	Baseline, 0.78** Mood scores, 0.51** Autonomic scores, −0.32*	Baseline, 0.86**
LF power	ns	Baseline, 0.53** Autonomic scores, −0.39*	Baseline, 0.51** Mood scores, 0.42*
HF power	Autonomic scores, −0.45*	Baseline, 0.62** Mood scores, −0.50** pTMD, 0.42**	Baseline, 0.80** pTMD, 0.36** Sex, −0.25*
LF/HF	ns	Mood scores, 0.50*	Baseline, 0.71**
RMSSD	Autonomic scores −0.41*	Baseline, 0.58** Mood scores, −0.49** pTMD, 0.34*	Baseline, 0.92** pTMD, 0.23*
SDNN	ns	Baseline, 0.61** Mood scores, −0.48** pTMD, 0.34*	Baseline, 0.89** pTMD, 0.25* Sex, −0.24*
SBP	ns	Baseline, 0.69** Autonomic scores, −0.35*	Baseline, 0.81**
DBP	ns	Baseline, 0.88** Autonomic scores, −0.25*	Baseline, 0.93**

Data were analyzed by logistic linear multivariate stepwise regression model. LF: low frequency and HF: high frequency power; MTC: maximal tooth clenching; SBP: systolic blood pressure; DBP: diastolic blood pressure; pTMD: painful signs of temporomandibular disorders.
*$p < .05$, **$p < .01$.

levels of SDNN were lower in participants with pTMD ($p < .05$, Mann–Whitney *U*-test). Also, the baseline level of LF power tended to be lower in those participants. As compared to baseline levels, there was a lower on-test decrease in MTC in participants with pTMD in HF power ($p < .05$), RMSSD ($p < .05$) and SDNN ($p < .01$, test × time × pTMD interactions, Mixed model analysis) than in those without pTMD. There were no significant differences in post-test-changes compared to baseline levels between participants with or without pTMD. Also, pTMD was associated with higher autonomic (Mann–Whitney *U*-test, $p < .05$) but not mood scores.

Any corrections for multiple analyses were not performed since all the statistical analyses compared not more than two groups. Also, the statistically tested parameters are positively related to each other. Using a correction for multiple analyses would in this case unnecessarily increase the risk of false-negative results.

The EMG of the masticatory muscles was registered in 21 of the 28 participants. Of the 7 participants of which EMG data was not recorded, 3 had pTMD. Graphs were made of the EMG data and those graphs were analyzed visually. Some of the participants ($n = 3/21$) clearly clenched their teeth during HGT but most of them ($n = 14/21$) did not. The results were unclear in some of the cases ($n = 4/21$). Figure 1 shows an example of a participant with pTMD clenching his teeth during HGT.

Discussion

One of the aims of the present study was to compare autonomic responses to MTC and HGT by measuring HR, HRV and BP changes in healthy participants. It was found that MTC- and HGT-induced autonomic responses resemble each other as shown in the HR and HRV parameters. In both tests, HF power and RMSSD decreased on-test and increased post-test reaching approximately the baseline level. This indicates a decrease in parasympathetic activity during the tests followed by a post-test increase. In both MTC and HGT LF

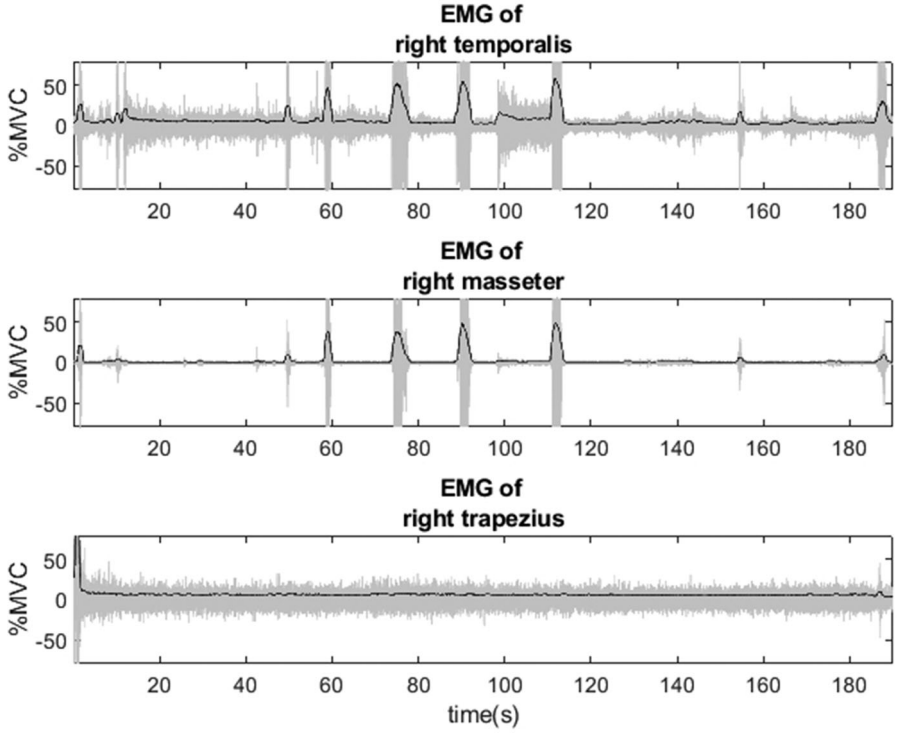

Figure 1. The electromyography (EMG) registration of one participant's right-side m. temporalis, m. masseter and m. trapezius showing significant activation of the masticatory muscles during HGT. The grey colour indicates the raw EMG signal and the black line is root mean square (RMS) amplitude proportion to the maximal contraction of the muscles.

power also decreased on-test and increased post-test but stayed below the baseline level. However, LF/HF ratio increased on-test and decreased strongly post-test reaching a value lower than baseline. These findings indicate that sympathovagal balance shifts towards sympathetic dominance during the tests, whereas parasympathetic dominance takes place after the tests. It should be noted that in both tests the LF/HF ratio decreased below the baseline levels indicating that parasympathetic activity is more dominant after both tests compared to baseline.

We also examined, if TCR would be activated during MTC. TCR can be triggered by stimulating structures innervated by the trigeminal nerve [7]. For example during MTC, a muscle group innervated by the trigeminal nerve, namely masticatory muscles are activated. Accordingly, TCR may have an impact on the autonomic responses to MTC [7]. TCR increases the activity of the parasympathetic nervous system and either increases or decreases sympathetic activity or that may stay unaltered [7]. The increased activity of the parasympathetic nervous system decreases the heart rate and lowers blood pressure [27]. Mixed model analysis showed that LF power, SDNN, DBP and SBP were significantly different between the two tests. Yet, the SDNN is dependent on the length of the recording [2] and the different durations of the tests may make the on-test value of this parameter unreliable to some extent. TCR activation during and after MTC could be one of the factors, that may have caused some of the differences between the two tests in the other parameters. Also, there was a statistically significant difference in on-test and post-test SBP and DBP values between the tests. Both of their on-test and post-test values were higher in HGT and their on-test maximal values and increases were greater in HGT. Interestingly, the post-test SBPs were lower than at the baseline after MTC but higher after HGT. This could be partly a result of TCR, lowering the BP on-test and post-test in MTC.

A new finding was that some of the participants also clenched their teeth during HGT. This may have altered the results as the autonomic responses during HGT may not have been purely due to gripping the hand but there may have been some clenching effect involved, too. Since the number of such participants was too low, we could not statistically address the possible mixing effects of clenching or if the clenching during HGT was associated with pTMD.

We also examined if there were differences in the autonomic responses between participants with and without pTMD. The subclinical TMD indicated by pTMD assessed in the clinical examinations may have affected the autonomic responses. Interestingly, pTMD was a predictor of autonomic responses in both MTC and HGT at on-test but post-test only in HGT. Also, pTMD affected several HRV parameters in the baseline as well as on-test in MTC and both on-test and post-test in HGT. In addition, the mixed model analysis showed that there were test $\times$ time $\times$ pTMD interactions in some of the HRV parameters and differences in on-test changes compared to baseline levels in those same HRV parameters between participants with or without pTMD. One of the mechanisms why pTMD may affect the autonomic responses could be that clenching would cause pain in participants with pTMD (even in those with a subclinical condition) and pain-induced increase in sympathetic activity [28]. This may happen not only in response to MTC but also to

HGT in the participants who clenched their teeth during HGT. Thus, there may be some unwanted masticatory muscle activation during HGT, which might have altered the autonomic responses. As mentioned above, the number of participants clenching during HGT was small. Accordingly, clenching, may not have affected the results of HGT significantly. Also, it is possible that some of the subjects clenched their fists or contracted some other muscles during MTC. This was not measured and if it occurs, it may also affect the results. This should be measured in future studies. Except for TMD, several other chronic pain conditions, such as fibromyalgia [29,30] and chronic neck pain [31–33] are linked to abnormal ANS function. In all these chronic pain conditions, the time domain parameters of HRV are lowered [23] which is a sign of diminished ANS function. The pTMD seems to affect the autonomic responses even in HGT. Thus, methodologically, participants with pTMD should be excluded when studying the responses of the ANS to MTC but possibly also to HGT. In addition, the mood and autonomic scores were predictors of some of the parameters. Higher autonomic scores were associated with pTMD. Thus, autonomic scores indicated abnormal ANS function as could be expected. Mood [34] and depression [35] are also associated with differences in ANS function.

As told before, Zhang et al. [8] compared the pre-test and on-test periods of HGT and unilateral, not maximal force, clenching task indicating that sympathetic activity during the clenching task turned out to be lower than during HGT. In the present study, MTC seemed to cause similar autonomic responses as HGT. Bilateral, maximal clenching in the present study may explain the differences in the results compared to those of Zhang et al. [8]. However, TCR may cause some of the differences between the two tests. In addition, we also examined the post-test effects on the activity of ANS as previously only the pre- and on-task periods have been studied. Also, the pTMD was taken into account.

One deficiency in this study is the short and heterogeneous clenching time which may make the on-test HRV parameters less reliable. This might be the reason why the TCR was only shown in the HR and BP parameters after the MTC task. Also, the participants were informed to clench with their maximal clenching force until volitional exhaustion, which, in fact, was the reason for the variable clenching times. For HGT we used the dynamometer at 30% of the participants' maximal gripping force for a given standard time. Such standardization would be helpful for MTC tasks in future studies to make the two tasks better comparable and results more reliable. Also, we did not control the participants' breathing and some of them may have held their breaths during MTC and HGT, which also causes autonomic responses. Breath-holding, alias Valsalva manoeuvre, can alter all HRV [36], BP and HR [37] parameters. Also, the pain caused by the clenching in participants with pTMD may have affected the responses of the autonomic nervous system. Finally, the pre-test stress may have influenced the autonomic responses at baseline.

Conclusions

The handgrip test and maximal tooth clenching seem to cause similar autonomic responses. However, MTC seems to activate the trigeminocardiac reflex, which causes on-test and post-clenching vagal activation as seen in the BP parameters. Furthermore, the presence of painful signs in relation to TMD did cause significant differences in or may influence the HRV parameters in the baseline levels and on-test responses. This may be underestimated in the studies of autonomic responses to MTC and HGT. In conclusion, there is a need for further studies and investigations with a higher number of participants, such without pTMD, to clarify the autonomic responses caused purely by maximal tooth clenching.

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