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TWELVE WEEKS OF OCCLUSAL SPLINT TREATMENT REDUCES BLOOD PRESSURE RESPONSES TO HYPERTENSIVE STIMULI IN THOSE WITH SLEEP BRUXISM

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Sabella M. & Jarvis S. Sleep bruxism (SB) is associated with increased sympathetic tone, a risk for the future development of hypertension. It is unclear whether those with SB would demonstrate exaggerated responses to hypertensive stimuli and whether treatment with an occlusal splint could mitigate these responses. **Objectives**: This study compared hemodynamic responses to the cold pressor test (CPT) and static handgrip (SHG) to fatigue between controls (CON) and those with SB to determine whether those with SB have exaggerated responses. **Methods**: Nineteen subjects (9 CON, 10 SB) had heart rate (HR) and blood pressure (BP) responses measured during a CPT and SHG with post-exercise circulatory arrest (PECA). SB subjects underwent a 12-wk occlusal splint intervention followed by a repeat of the experimental protocol. **Results**: No BP differences existed with pre- (PRE) vs. post-intervention (POST) in the SB subjects. CPT induced larger ΔSBP (10±10 vs. 19±7 mmHg; $p < 0.05$) and ΔDBP (8±8 vs. 17±9 mmHg; $p < 0.05$) in SB subjects compared to CON. POST had attenuated ΔSBP (15±9 vs. 8±5 mmHg; $p < 0.03$) and ΔDBP (18±10 vs. 8±6 mmHg; $p < 0.05$) during CPT. SB subjects experienced greater ΔDBP (21±11 vs. 31±8; $p < 0.05$) during SHG which was not different during POST. **Conclusions**: SB individuals may be at increased risk for the development of hypertension based on exaggerated BP responses to hypertensive stimuli. Twelve weeks of occlusal splint treatment attenuated some of these responses.

Key Words: sympathetic tone, hypertension, cold pressor test, static handgrip to fatigue, sleep bruxism, controls, post-exercise circulatory arrest

INTRODUCTION

Approximately 8% of the general adult population is affected by sleep bruxism (SB) (Lavigne, Kato, Kolta, & Sessle, 2003), a sleeping disorder characterized by nocturnal teeth grinding. SB is associated with enhanced daytime sympathetic tone (Malpas, 2010), as well as surges in sympathetic activity that precede SB episodes in the form of brief awakenings termed microarousals (Nashed et al., 2012). Microarousals can occur spontaneously or be triggered through external stimuli (Satoh & Harada, 1973). Regardless of the origin, there exists a shift in sympathovagal balance towards sympathetic dominance during these episodes (Huynh et al., 2006). Heart rate (HR) and blood pressure (BP) increase, likely due to the pressor response created by the forceful bite (Okada et al., 2009). It has been proposed that the trigeminocardiac reflex is initiated to lower sympathetic cardiovascular responses (Schames, Schames, Schames, & Chagall-Gungur, 2012). For example, manipulation of the trigeminal branches during SB lead to vagal stimulation, thus countering the sympathetic activation prior to SB (Lang, Lanigan, & van der Wal, 1991).

Individuals with elevated sympathetic activity have demonstrated exaggerated responses to tests that expose them to a hypertensive stimulus, such as the cold pressor test (Wood, Sheps, Elveback, & Schirger, 1984). These responses identify individuals that are increased risk for the future development of hypertension and cardiovascular events (Wood et al.,

1984). For example, long-term follow-up studies have shown that individuals with hyper-reactive BP responses during childhood or their younger years were associated with the future development of hypertension (Chaney & Eyman, 1988; Kasagi, Akahoshi, & Shimaoka, 1995; Menkes et al., 1989; Wood et al., 1984).

Occlusal splints, commonly used to treat SB, are known to decrease electromyographic (EMG) activity in the jaw muscles that are associated with SB episodes (Amorim, Vasconcelos Paes, de Faria Junior, de Oliveira, & Politti, 2012). Occlusal splint treatment has been reported to show improvement in aspects of sleep structure, such as increased slow wave activity sleep, which is inversely related to hypertension (Fung et al., 2011; Sjoholm, Kauko, Kemppainen, & Rauhala, 2014). However, it is also unclear whether wearing an occlusal splint may inhibit the beneficial parasympathetic influence of the trigeminocardiac reflex by preventing manipulation of the mandibular branch. Use of this splint has been shown to lower maximal bite force contraction and EMG to the masseter (Amorim et al., 2012). Blood pressure rises during sustained contractions of the masseter muscle. By inhibiting the muscle mechanoreceptor response created by the masseter, the increase in blood pressure would not occur.

The relationship between SB and cardiovascular risk factors is not clearly understood. Therefore, the objective of this study was to determine the relationship between SB and BP responses to hypertensive stimuli. It was hypothesized that individuals with SB would have an exaggerated BP response to the compared to the control subjects. it was also hypothesized that SB subjects would demonstrate improvements in BP responses after 12-wks of treatment with an occlusal splint.

METHODS

Subjects

Nineteen subjects (6 men, 13 women) gave written informed consent to participate in this study which was approved by the Institutional Review Board at Northern Arizona University. The study followed the guidelines set forth in the *Declaration of Helsinki*. Subjects were divided in the SB group (n = 10) and the control (CON) group (n=9). SB subjects were initially recruited from the university campus and the nearby community using flyers and mass emails. During the medical screening the participants filled out a questionnaire assessing their daily symptoms of sleep bruxism. Symptoms included headaches upon waking, pain in the location of the temporomandibular joint upon waking, tooth sensitivity, and if they were using substances, medically or recreationally, that could have been a factor in their sleep bruxism. Decisive inclusion in the study was determined by the subject's diagnosis by a dentist and whether or not the subject needed treatment, failing to do so was exclusionary. SB subjects could also already have an occlusal splint but been inconsistent with treatment (<2 nights per week). Inclusionary criteria were: ≥18 yrs, body mass index (BMI) ≤35 kg/m^2, no evidence of depression or neurological disorders, and no smoking or living with a smoker. Also, failing to be diagnosed by a dentist or use treatment was exclusionary. During screening, a detailed medical history was obtained, and a supine 12-lead resting electrocardiogram and BP measurement was performed.

Experimental Design

24-Hr Ambulatory Blood Pressure

Subjects with SB wore a 24-hr ambulatory BP monitoring device to confirm normotensive status for the study. BP via brachial artery was taken every 30 minutes during waking hours and every hour during sleep (Oscar 2, Morrisville, NC). The 24-hr ambulatory BP monitoring was repeated after the 12-wk occlusal splint intervention.

Study visits. Subjects arrived at the lab having fasted for ≥4 hrs. Subjects also avoided strenuous physical activity for ≥12 hrs and caffeine and alcohol for ≥48 hrs prior to the visit. The study visits included a cold pressor test (CPT) and static handgrip to fatigue (SHG) with post-exercise circulatory arrest (PECA). PECA was performed by occluding blood flow to the arm by inflating a blood pressure cuff to 250 mmHg. Prior to instrumentation the subject performed three maximal voluntary contractions with his/her dominant hand. The protocol was performed in the same order and under the same lab conditions during the second study visit.

Cold Pressor Test

Subjects laid in the supine position while beat-by-beat BP (Finometer MIDI, ADInstruments, Colorado Springs, CO) and heart rate (ECG module, ADInstruments, Colorado Springs, CO) were continuously measured. Following a 1 min baseline (BL), the cold pressor test was performed by submerging the hand in a 50:50 mixture of water and ice (2-4 C°) for 2 min, followed by a 3 min recovery (REC) period. BP via electrosphygmomanometry (SunTech Tango+, Morrisville, NC) was obtained during min 1 and 2 of cold pressor test and min 1 and 3 of REC.

Static Handgrip to Fatigue with Post Exercise Circulatory Arrest

The subject was required to maintain 30% of his/her maximal voluntary contraction until fatigue. Fatigue was defined as inability to maintain 80% of the desired force. A 2-min PECA began immediately following failure by inflating a BP cuff to 250 mmHg around the upper arm.

Intervention

After the PRE measurements were obtained, the SB subjects wore a nighttime occlusal splint prescribed by a dentist for 12-wks. Occlusal splints were made of hard acrylic resin and covered the entire arch of the teeth. Soft splints and nociceptive trigeminal inhibiting splints were not permissible. Subjects were queried at weeks 4 and 8 of the intervention to assess treatment compliance and if they were experiencing difficulties using their occlusal splints. Prior to the experimental protocol, the subjects were informed of the possible beneficial uses of using an occlusal splint, such as protection from chipping or fracturing teeth and possibly relief of tension type headaches. Information regarding effects on the cardiovascular system was limited to possible overall health references and quality of life increases due to decreases in pain.

Statistical analysis

Beat-by-beat BP and HR for BL were condensed into 1 min averages. Cold pressor test and REC were condensed into 30 sec averages. Static handgrip was divided into 5 even stages [20, 40, 60, 80, and 100% (fatigue)]. PECA was reduced to 1 min averages and REC for static handgrip was reported as 1 min averages.

All values are expressed as means ± SD. A repeated measures ANOVA [group (CON, SB) × stage] was used to compare BP and HR responses. For the cold pressor test, comparisons between BL, CPT1, CPT2, and REC were made. For the static handgrip to fatigue, comparisons between BL, fatigue (100%), PECA1, PECA2, and REC were made. T-tests were used to compare BP and HR changes (Δ) from BL between CON and SB groups during CPT1, CPT2, fatigue, PECA1, and PECA2. Paired t-tests were used to compare these differences PRE vs POST in the SB subjects. Significance was defined as $p < 0.05$.

RESULTS

Subject characteristics

Eight of the initial 10 SB subjects completed both PRE and POST visits. These subjects reported occlusal splint compliance of $\geq$5 nights per week throughout the intervention. None of these subjects reported discomfort related to the treatment or a need for refinement from any cause, such as soft tissue interference, during the check-up or on the day of the final experimental protocol, though one subject who did not complete the follow-up protocol reported removing the mouth guard during the night due to its excessive bulk.

There was no difference in age (CON: 19±1 vs SB: 22±5 yrs.), height (172±9 vs 168±10 cm), weight (66.7±10.7 vs 69.0±13.8 kg), or body mass index (22.8±2.5 vs 24.2±3.7 kg/m^2) between the two groups of subjects.

24-Hr Ambulatory Blood Pressure Monitoring for Sleep Bruxism Subjects

SBP (120±8 vs 122±8 mmHg, *P*=0.03, for PRE vs POST, respectively) and DBP (70±7 vs 71±6 mmHg, $p = 0.04$) were both higher during the overall phase during POST testing. This was likely due to higher SBP (124±9 vs 128±10 mmHg, $p = 0.02$) and DBP (73±7 vs 76±7 mmHg, $p = 0.04$) during the awake phase after the intervention. No differences in SBP (107±9 vs 107±8 mmHg, p = NS) and DBP (59±7 vs 58±8 mmHg, p = NS) were observed between PRE vs POST during the sleep phase.

Cold Pressor Test

Heart rate. There was a trend toward a main effect of lower HR among SB subjects compared to CON (p = 0.07), but overall was not significant. The cold pressor test elicited the same absolute HR in the CON and SB groups. There were also no differences in HR between PRE vs POST. HR changes from BL (ΔHR) did not differ at CPT1 or CPT2 between CON and SB subject or between PRE and POST.

Blood pressure. As expected, the cold pressor test induced increases in SBP and DBP during CPT in CON and SB groups (all p < 0.01). SB subjects demonstrated lower SBP during the BL for cold pressor test and REC (both p < 0.05). However, no significant differences were seen in absolute SBP and DBP between the groups during the cold pressor test. SBP and DBP were similar between PRE and POST in SB subjects. However, ΔSBP and ΔDBP (changes from BL, Table 3) during CPT1 were larger in SB subjects than in CON (both p < 0.05). ΔSBP during CPT2 was also larger in SB subjects (p < 0.05). ΔSBP and ΔDBP during POST were attenuated compared to PRE testing during CPT1 (both p < 0.05) but ΔSBP was not significant during CPT2 (PRE vs POST, p = 0.07).

Static Handgrip to Fatigue with Post Exercise Circulatory Arrest

No differences in maximal contraction force were observed CON vs. SB (38.1±13.9 vs. 41.9±15.9 kg, p = 0.59). Time to fatigue was also similar between groups (167.9±17.0 vs. 201.8±42.6 sec, p = 0.23). Maximal force contraction did not differ PRE vs POST in SB subjects (42.2±15.0 vs. 40.3±16.5 kg, p = 0.16) nor did time to fatigue (199.1±46.8 vs. 208.0±56.7 sec, p = 0.61)

Heart Rate. No group (CON vs SB) or intervention (PRE vs POST) differences in HR existed during static handgrip to fatigue and PECA. As expected, HR increased from BL in all groups during 100% SHG (all P<0.001). No ΔHR differences between CON and SB or PRE vs POST were observed during static handgrip or PECA.

Blood pressure. There was no difference in SBP during static handgrip or PECA between CON vs SB or PRE vs POST. Conversely, DBP was higher in SB subjects during static handgrip and PECA (all p 0.05). All subjects demonstrated significantly higher SBP and DBP during static handgrip and PECA (CON vs SB and PRE vs POST, all p < 0.001). ΔSBP was similar among CON vs SB during static handgrip and PECA; however, ΔDBP was larger during static handgrip in SB subjects (p < 0.05) and bordered significance during PECA (p = 0.06). No difference was seen in ΔDBP response to static handgrip between PRE and POST SB subjects. Table 3 outlines ΔSBP did not change and ΔDBP remained elevated in SB subjects after occlusal splint intervention during static handgrip and PECA.

Table 1

Blood pressure and heart rate during the cold pressor test.

Variable	Cold pressor test			
	BL	**CPT1**	**CPT2**	**REC**
HR, beats/min				
Control	75 ± 15	83 ± 20	79 ± 20	67 ± 8
Sleep Bruxism	63 ± 11	69 ± 12	67 ± 11	61 ± 8
Pre-Int	62 ± 12	66 ± 7	64 ± 8	61 ± 9
Post-Int	63 ± 9	66 ± 11	63 ± 9	63 ± 7
SBP, mmHg				
Control	129 ± 12**	132 ± 16	139 ± 13*	127 ± 15**
Sleep Bruxism	116 ± 9	136 ± 10*	136 ± 10*	115 ± 7
Pre-Int	115 ± 10	134 + 10	135 ± 10	114 ± 7
Post-Int	114 ± 10	121 ± 10	129 ± 10	115 ± 8
DBP, mmHg				
Control	68 ± 5	76 ± 9	79 ± 7*	68 ± 9
Sleep Bruxism	64 ± 8	81 ± 14*	81 ± 16*	64 ± 11
Pre-Int	64 ± 8	82 ± 16	82 ± 18	64 ± 12
Post-Int	64 ± 7	71 ± 5	80 ± 9	69 ± 10

Note. Values are mean differences from baseline ± SD. HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure. Pre-Int, pre-intervention; Post-Int, post-intervention. *Difference from BL, $p < 0.05$. **Difference within stage between Control and SB, $p < 0.05$.

Table 2

Blood pressure and heart rate during static handgrip to fatigue with PECA.

	BL	20%	40%	60%	80%	100%	PECA1	PECA2	REC
Variable									
HR, Beats/min									
Control	66 ± 8	77 ± 14	83 ± 10	87 ± 9	91 ± 12	92 ± 16	71 ± 16	67 ± 10	65 ± 9
Sleep Bruxism	63 ± 7	73 ± 10	79 ± 6	83 ± 7	87 ± 9	95 ± 11*	68 ± 12	66 ± 13	61 ± 7
Pre-Int	62 ± 7	71 ± 9	78 ± 6	83 ± 7	87 ± 10	94 ± 12*	67 ± 13	65 ± 15	60 ± 8
Post-Int	63 ± 8	72 ± 10	76 ± 11	77 ± 10	82 ± 11	87 ± 14*	65 ± 7	63 ± 12	61 ± 7
SBP, mmHg									
Control	117 ± 14	134 ± 12	140 ± 16	145 ± 21	150 ± 25	155 ± 23*	135 ± 16*	140 ± 17*	126 ± 12
Sleep Bruxism	126 ± 21	135 ± 25	145 ± 25	154 ± 25	160 ± 23	167 ± 23*	152 ± 26*	156 ± 24*	121 ± 29
Pre-Int	130 ± 16	136 ± 21	148 ± 22	157 ± 22	163 ± 20	169 ± 20*	157 ± 24*	162 ± 21*	125 ± 26
Post-Int	131 ± 12	138 ± 14	146 ± 17	154 ± 19	158 ± 20	163 ± 21*	156 ± 19*	162 ± 19*	138 ± 16
DBP, mmHg									
Control	52 ± 8	58 ± 9	64 ± 9	67 ± 11	70 ± 11	73 ± 12* **	60 ± 7 * **	65 ± 9* **	52 ± 8
Sleep Bruxism	61 ± 16	67 ± 19	76 ± 19	82 ± 19	85 ± 21	92 ± 22*	75± 19*	81 ± 21*	59 ± 19
Pre-Int	63 ± 13	69 ± 15	78 ± 14	85 ± 15	89 ± 16	96 ± 18*	79 ± 16*	85 ± 18*	62 ± 14
Post-Int	61 ± 13	67 ± 10	76 ± 14	82 ± 15	86 ± 15	89 ± 16*	77 ± 14*	84 ± 17*	64 ± 10

Note. Values are mean differences from baseline ± SD. PECA, post-exercise circulatory arrest; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure. Pre-Int, pre-intervention; Post-Int, post-intervention. *Difference from BL, $p < 0.05$. **Difference within stage between Control and SB, $p < 0.05$.

Table 3

Blood pressure and heart rate changes during cold pressor test and static handgrip to fatigue with PECA.

Variable	Cold pressor test		Static handgrip to fatigue	
	ΔCPT1	**ΔCPT2**	**100%, at fatigue**	**PECA2**
ΔHR, Beats/min				
Control	6 ± 11	4 ± 12	25 ± 14	1 ± 7
Sleep Bruxism	6 ± 10	4 ± 10	32 ± 10	3 ± 10
Pre-Int	4 ± 9	2 ± 10	32 ± 10	3 ± 12
Post-Int	3 ± 6	0 ± 7	24 ± 17	0 ± 8
ΔSBP, mmHg				
Control	3 ± 6	10 ± 10	38 ± 28	22 ± 16
Sleep Bruxism	16 ± 18*	19 ± 7*	39 ± 8	30 ± 11
Pre-Int	15 ± 9	20 ± 8	39 ± 9	32 ± 11
Post-Int	8 ± 5†	15 ± 8	32 ± 16	25 ± 11
ΔDBP, mmHg				
Control	8 ± 8	12 ± 5	21 ± 11	12 ± 8
Sleep Bruxism	17 ± 9*	17 ± 12	31 ± 8*	20 ± 8
Pre-Int	18 ± 10	18 ± 13	32 ± 8	22 ± 7

Post-Int	8 ± 6†	16 ± 6	28 ± 11	23 ± 16

Note. values are mean differences from baseline ± SD. PECA, post exercise circulatory arrest; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure. *Difference in change from baseline compared to CON, $p < 0.05$. †Difference in change from baseline compared to pre-intervention, $p < 0.05$.

DISCUSSION

The major findings of this study are SB subjects demonstrated: 1) exaggerated BP reactivity to both the cold pressor test and static handgrip compared to the CON group and 2) lowered BP responses to cold pressor test after 12-wks of occlusal splint intervention. The findings of the present study support the hypothesis that people with SB might be at increased risk for the future development of hypertension. Correspondingly, the results indicate that use of an occlusal splint may be beneficial in lowering ΔSBP responses to the cold pressor test after a 12-wk intervention. This is the first to examine a predictive measurement of BP responses to cold pressor and static handgrip before and after 12-wks of occlusal splint intervention.

SB and Responses to the Cold Pressor Test

SB subjects in this study sample had a prominently augmented mean ΔSBP increase, approximately double than that of CON subjects during cold pressor test. One advantage of cold pressor testis it is a highly reproducible test (Zhao et al., 2012). Exaggerated BP responses, such as those observed during long term studies of 20-, 28-, and 36-years, have demonstrated that these responses are strong evidence for risk of the future development of hypertension (Kasagi et al., 1995; Menkes et al., 1989; Wood et al., 1984). Consistent with Menkes et al. (1989), when the maximal SBP response to cold pressor test is augmented (≥20 mmHg), subjects are more likely to develop hypertension before the age of 45 yrs. Taken together, these studies and this one indicate that SB subjects may be at risk for future development of hypertension. It is of interest to note that the subjects in the current study were young and normotensive yet exhibited cold pressor test responses comparable to hypertensive individuals.

ΔSBP and ΔDBP during cold pressor test were reduced in POST SB subjects. These results suggest that the use of an occlusal splint was beneficial in reducing the reactivity to the cold pressor test. A possible mechanism for this reduction could be due to a decrease in the number SB episodes with occlusal splint treatment. For example, a short term (two-night study) with an occlusal splint intervention showed a 41% decrease in the number of SB episodes per hour (Dube et al., 2004). Subjects also demonstrated significantly lower bite contraction force after occlusal splint treatment (Amorim et al., 2012). Thus, wearing an occlusal splint may attenuate the BP responses that normally occur during SB episodes. The potential decrease in the number of SB episodes could have led to diminished sympathetic dominance and less exaggerated responses to the CPT. Also, the subjects did not report lifestyle changes, such as diet or exercise, or health changes that would account for the results when questioned during the follow-up visit, but BP can be altered by numerous mental and physiological conditions, such as dealing with family issues or traumatic events, which should be kept into consideration.

SB Responses to Static Handgrip to Fatigue and Post Exercise Circulatory Arrest

In our study, SB subjects had higher DBP during static handgrip and PECA, as well as a larger ΔDBP during static handgrip. DBP responses to static handgrip exercise have been previously used to predict hypertension in a 14-yr follow-up trial (Chaney & Eyman, 1988). The initial responses in our SB sample could be due to sensitized mechano- or chemo-receptors, a common trait found in hypertensive individuals (Victor, Rotto, Pryor, & Kaufman, 1989). Mechano- and chemo-receptors present in skeletal tissue are responsible for the cardiovascular pressor responses during static exercise (Victor et al., 1989). In addition to rises in HR and BP during the microarousals that precede SB episodes (Huynh et al., 2006; Nashed et al., 2012), there are further escalations in BP when the rhythmic jaw contractions occur in SB subjects (Nashed et al., 2012). For example, Okada et al. (2009) observed significant increases in BP during isometric biting at

50% of maximal bite force. Therefore, increases in BP observed in SB subjects may be the result of masseter and temporalis (muscles of mastication) contractions similar to those observed in static handgrip using the forearm muscles. The exaggerated response could also be due to the activation of chemoreceptors in the periodontal tissue (Okada et al., 2009), or a combination of both. Notably, studies in patients with heart failure and hypertensive individuals have attributed abnormally high BP responses to static handgrip to sensitized mechanoreceptors in the working skeletal muscles that lead to larger sympathetic activation than those observed in normotensive subjects (Hoel, Lorentsen, & Lund-Larsen, 1970; Middlekauff & Chiu, 2004). Thus, it is possible that SB subjects may exhibit similar characteristics as repeated rhythmic jaw contractions may also have a sensitization effect (Middlekauff & Chiu, 2004). Interestingly, Okada et al. (2009) showed attenuated BP responses during the second trial of isometric biting after administration of a nerve block to the periodontal tissue. This further supports that increases in cardiovascular variables are attributed to the pressor response (Okada et al., 2009).

Occlusal Splint

Based on the previous findings of reduced electromyographic activity and bite force, it was hypothesized that 12-wks of occlusal splint use would lead to lower BP responses to the cold pressor test and static handgrip to fatigue. There were improvements in the ΔSBP response to the cold pressor test, but not in the ΔDBP response to static handgrip. Additionally, SBP and DBP were slightly higher during 24-hr BP monitoring after the intervention. There are two possible explanations for this finding. One, it could be due to longer durations of cardiovascular responses during microarousal that would transpire in the event occlusal splints inhibit of the trigeminocardiac reflex and, consequently, its beneficial parasympathetic effects. For example, Takahashi et al. (2013) reported no reduction in microarousal occurrence, even when there were decreases in electromyography activity and the number of SB episodes (Takahashi et al., 2013). This could present a possible reason for the conflicting findings in the results of this study and increased BP after during 24-hr BP monitoring. Two, we cannot also exclude the possibility that the timing of the POST visit played a role. Most of the subjects in this study were college students and some completed their POST testing during their finals week, which may have confounded the results. However, due to the improvements seen in the cold pressor test BP reactivity, the occlusal splint showed that it may be helpful. Previous findings on the efficacy of occlusal splints have also reported conflicting results regarding its use and ability to diminish electromyographic activity (Abekura et al., 2011). The results are consistent with these previous findings of ambiguity regarding occlusal splints. Hence, the continuation of research involving occlusal splint use as treatment for SB is necessary to determine its efficacy regarding SB episodes and systemic effects.

Limitations

Identifying the specific cause of SB was a limitation in this study. The causes of SB are psychosocial factors and dopaminergic disorders malocclusion has been theorized to be a cause of for many years, but there is no valid evidence to support this claim (Manfredini, Visscher, Guarda-Nardini, & Lobbezoo, 2012). Disorder in the dopaminergic system is one of the probable causes of SB (Lobbezoo, Lavigne, Tanguay, & Montplaisir, 1997) because dopamine has motor control functions (Graybiel, Aosaki, Flaherty, & Kimura, 1994). Reductions in dopamine can cause loss of fine motor movement and possible tremors (Graybiel et al., 1994). Though all of the SB subjects were pre-screened for use of neurological drugs and medications affecting the dopaminergic system, including cigarette smoking and recreational drug use, subjects were not explicitly tested for any conditions involving the dopaminergic system. Another reason, not previously mentioned, that RMMA/SB occurs is due to gastroesophageal reflux disease (GERD) (Miyawaki et al., 2003). Chemoreceptors in the esophagus lead to micro-arousal, which then leads to RMMA and swallowing, as well as episodes of bruxing. This mechanism was not tested or quantified in this trial, but could have influenced the results by continuing microarousals regardless of treatment use (Miyawaki et al., 2003). Also, if GERD is the primary source of their SB the beneficial effects from wearing a nighttime mouth guard would likely be minimal. Therefore, the

underlying cause for SB could have been due to stress related factors, disorder in the dopaminergic system, or GERD; malocclusion was ruled out as a cause due to the little evidence supporting this as a SB risk in healthy individuals, gold star diagnoses using electromyography and test to eliminate individuals with GERD or dopaminergic disorders should be done. This study compared sleep bruxers and non-sleep bruxers, as well as non-sleep bruxers before and after using an occlusal splint, however, in future studies a group of bruxers without treatment should also be compared to bruxers undergoing occlusal splint treatment.

CONCLUSION

In conclusion, this study examined BP responses to cold pressor test and static handgrip to fatigue before and after 12-wks treatment with an occlusal splint. Even young, normotensive SB subjects showed risk for the development of future hypertension based on exaggerated responses to cold pressor and static handgrip to fatigue. The risk for hypertension is likely developed from enhanced sympathetic activity. Occlusal splint use showed to be beneficial over a 12-wk duration in lowering the exaggerated SBP response to cold pressor test, likely due to decreased sympathetic tone.

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