Does Sleep Bruxism Contribute to Headache-Related Disability After Mild Traumatic Brain Injury? A Case-Control Study

Yoshitaka Suzuki, DDS, PhD
Postdoctoral Fellow
Faculty of Dental Medicine
Université de Montréal
Montréal, Québec, Canada

Caroline Arbour, RN, PhD
Assistant Professor
Faculty of Nursing
Université de Montréal
Montréal, Québec, Canada

Samar Khoury, PhD
Postdoctoral Fellow
Alan Edwards Center for Research on Pain
McGill University
Montréal, Québec, Canada

Jean-François Giguère, PhD, MD
Neurosurgeon and Clinical Professor
Division of Trauma Research
Departments of Surgery and Neurological Sciences
Hôpital du Sacré-Coeur de Montréal
Montréal, Québec, Canada

Ronald Denis, MD
Surgeon and Clinical Professor
Division of Trauma Research
Departments of Surgery and Neurological Sciences
Hôpital du Sacré-Coeur de Montréal
Montréal, Québec, Canada

Louis De Beaumont, PhD
Assistant Professor
Center for Advanced Research in Sleep Medicine (CARSM)
Hôpital du Sacré-Coeur de Montréal
Montreal, Québec, Canada

Gilles J. Lavigne, DMD, PhD
Professor
Faculty of Dental Medicine
Université de Montréal
Montréal, Québec, Canada

Correspondence to:
Dr Gilles J. Lavigne
6128 succursale Centre-ville
Montréal (Québec)
Canada H3C3J7
Fax: +1-514-343-2233
Email: gilles.lavigne@umontreal.ca

Aims: To explore whether traumatic brain injury (TBI) patients have a higher prevalence of sleep bruxism (SB) and a higher level of orofacial muscle activity than healthy controls and whether orofacial muscle activity in the context of mild TBI (mTBI) increases the risk for headache disability. **Methods:** Sleep laboratory recordings of 24 mTBI patients (15 males, 9 females; mean age ± standard deviation [SD]: 38 ± 11 years) and 20 healthy controls (8 males, 12 females; 31 ± 9 years) were analyzed. The primary variables included degree of headache disability, rhythmic masticatory muscle activity (RMMA) index (as a biomarker of SB), and masseter and mentalis muscle activity during quiet sleep periods. **Results:** A significantly higher prevalence of moderate to severe headache disability was observed in mTBI patients than in controls (50% vs 5%; $P = .001$). Although 50% and 25% of mTBI patients had a respective RMMA index of ≥ 2 episodes/hour and ≥ 4 episodes/hour, they did not present more evidence of SB than controls. No between-group differences were found in the amplitude of RMMA or muscle tone. Logistic regression analyses suggested that while mTBI is a strong predictor of moderate to severe headache disability, RMMA frequency is a modest but significant mediator of moderate to severe headache disability in both groups (odds ratios = 21 and 2, respectively). **Conclusion:** Clinicians caring for mTBI patients with poorly controlled headaches should screen for SB, as it may contribute to their condition. *J Oral Facial Pain Headache 2017;31:306–312. doi: 10.11607/ofph.1878*

Keywords: *headache, polysomnography, rhythmic masticatory muscle activity, sleep bruxism, traumatic brain injury*

Traumatic brain injury (TBI) results from a sudden impact imparted directly or indirectly to the head by an external mechanical force.[1] According to the Centers for Disease Control and Prevention, approximately 1.7 million civilians a year experience a TBI in the United States.[2] Mild TBI (mTBI) accounts for 85% of all TBI and is defined as a concussion after a force to the head with a transient alteration in mental status or loss of consciousness.[3,4] Even in milder forms, a TBI is often accompanied by a myriad of somatic, neuropsychiatric, and cognitive symptoms; indeed, over 50% of mTBI patients complain of headaches, and 15% complain of widespread pain in the months following injury.[4–7] Sleep-wake disturbances (eg, hypersomnia, insomnia, circadian rhythm misalignment, and sleep apnea) have also been reported after mTBI,[4,8–10] with more than one-third of mTBI patients complaining of sleep disturbances in the weeks following injury.[11]

Sleep bruxism (SB) is a sleep movement disorder characterized by teeth clenching or grinding and/or bracing or thrusting of the mandible.[12] Rhythmic masticatory muscle activity (RMMA) is a research biomarker of SB.[13–15] SB is reported to increase the odds of tension-type headaches (odds ratio [OR] = 3.1 to 3.8) in healthy individuals,[16] and it has also been found that SB subjects are at higher risk for transient morning jaw muscle pain.[17,18] Furthermore, recent sleep studies have observed that temporomandibular disorder (TMD) patients or patients with SB/teeth grinding report transient morning headaches.[17,19–22] Morning headache, jaw pain (in the masseter and temporalis muscles and in the temporomandibular joint [TMJ]), and fatigue are used

Table 1 Case Reports of Sleep Bruxism Following Brain Injury[23–27]

Author	Situation	Orofacial activity
Ivanhoe et al[23]	Male, 28 y Sports accident Glasgow Coma Scale: 3; attenuated artifact of the right middle fossa (CT)	Onset: 2 mo No history of bruxism Severe and continuous tooth grinding
Kesikburun et al[24]	Male, 21 y Car accident Coma for 2 weeks, superior thalamic hemorrhage (CT)	Onset: 2 mo Severe tooth grinding
Millwood and Fiske[25]	7 males, 21–58 y 3 females, 43–63 y Road traffic accident (4), industrial accident (1), fall (1), vascular injury (2), anoxic injury (2) Persistent vegetative or minimally conscious state	Lip injury by recurrent biting (all), bruxism (3), noise created by bruxism (2), facial hypersensitivity (eg, biting the lip when the face was touched; 9)
Pidcock et al[26]	Male, 7 y Acute increased intracranial pressure secondary to malfunction of intraventricular shunt Old left thalamic injury consistent with in utero damage and new occipital and brainstem lesions consistent with herniation (MRI)	Onset: 2 mo No history of bruxism Increased muscle tone from 19 days postinjury Severe bruxism
Van Zandijacke and Marchau[27]	Female, 32 y Car accident Inhomogenous hyperdensity in the left parietotemporal area (CT)	Onset: 4 mo Jaw clenching Chewing movement Very loud and continuous tooth grinding

CT = computed tomography; MRI = magnetic resonance imaging.

as clinical indicators of SB.[13,19,21] Five case reports have documented secondary SB in severe TBI patients (Table 1),[23–27] and mTBI may also lead to SB. However, the lack of quantitative SB muscle activity data (eg, electromyographic [EMG] activity) and sufficiently powered study designs in past studies cast doubt on the association between TBI and SB. It has also been reported that higher background masseter muscle tone during quiet sleep time may contribute to myofascial TMD, which has masticatory muscle pain.[20] Again, there is little evidence to support any association between TBI and these orofacial muscle activities.

Just as in individuals with no brain injuries, it could be hypothesized that orofacial muscle activity during sleep could be present in some mTBI patients and may contribute to exacerbation of headache. Therefore, the aims of this study were to explore whether mTBI patients have a higher prevalence of SB/RMMA and a higher level of orofacial muscle activity than healthy controls and whether orofacial muscle activity in the context of mTBI increases the risk for headache disability.

Materials and Methods

Study Design, Sample, and Ethics

A case-control study design was used. Data from a convenience sample of 24 mTBI patients (15 males, 9 females; age [mean ± standard deviation (SD)] 38 ± 11 years; 2.4 ± 1.1 months postinjury) and 20 healthy controls (8 males, 12 females; 31 ± 9 years) were extracted from a larger-scale study addressing the co-occurrence of pain and sleep disorders after mTBI.[28] The smaller number of participants in the present study was due to age and gender matching (original sample = 102 mTBI patients and 22 controls undergoing genetic analyses).

Patients seeking care for first-time mTBI at the emergency department of a tertiary trauma hospital were invited by a research nurse to participate in a 2-night laboratory polysomnographic (PSG) assessment. A trauma neurosurgeon (J.F.G.) confirmed mTBI by basing the diagnosis on the World Health Organization Task Force criteria[29]; ie, a score of 13 to 15 on the Glasgow Coma Scale upon presentation for health care and a period of loss of consciousness, mental confusion, and/or posttraumatic amnesia not exceeding 30 minutes postimpact. Patients with a history of psychiatric or neurologic conditions and/or acute/chronic pain prior to the injury, nightshift workers, patients over 65 years old, and patients using medication or any substance known to influence sleep were excluded.

A control group of healthy individuals was recruited by referrals and local newspaper advertisements. Individuals with a previous history of brain injury or orofacial pain disorders or with a confirmed diagnosis of a sleep disorder (eg, insomnia, periodic limb movement, or rapid eye movement [REM] disorder behavior) were excluded.

The study was approved by the ethics board of the Hôpital du Sacré-Coeur de Montréal (CIUSSS du Nord-de-l'Île-de-Montréal), Quebec, Canada. All participants provided written informed consent prior to participating.

Questionnaires

All participants completed the Migraine Disability Assessment Scale (MIDAS),[30] which contains five questions referring to the number of days that headache interfered with general life activities (such as work or household work) in the last 3 months. Total score ranged from 0 to 21+, and the degree of headache disability was rated as: Grade I = minimal or infrequent disability (score 0–5); Grade II = mild or infrequent disability (score 6–10); Grade III = moderate disability (score 11–20); and Grade IV = severe disability (score 21+). Participants scoring Grade III or higher on the MIDAS were considered liable to have moderate to severe headache disability.

Aside from headache, participants were asked to report the presence (yes or no) of other clinical indicators of SB, such as poor sleep quality, awareness of tooth grinding and/or jaw clenching during sleep (ie, as a proxy for sleep bruxism), and pain in various body sites (frontal and occipital head, neck, face, shoulder, elbow, hand, sternum, pelvic, upper and lower back, knee, and foot). The morning following PSG recording, participants were also asked to report the presence (yes or no) and intensity of global body pain and fatigue on a 0–100 mm visual analog scale (0 = complete absence of symptoms, 100 = the worst possible level imaginable). The Beck Anxiety Inventory (BAI)[31] and the Beck Depression Inventory (BDI-II) were used to assess mood.[32]

PSG Recording

All participants slept for 2 consecutive nights in a light- and sound-attenuated room at the hospital sleep laboratory.[28] The first night was intended for habituation and the second night for data analysis. PSG recordings were performed by using a 32-channel Grass PSG recorder with 0.1- to 100-Hz filter bandpass. The montage comprised 11 leads for electroencephalographic (EEG) derivations, placed according to the standard system (eg, C3-A2, O2-A1): bilateral electrooculogram (EOG) recordings; mentalis, right masseter, and right tibialis (anterior leg) EMG recordings; and 3 electrocardiogram (ECG) derivations.[28] Thoracic and abdominal straps, a nasal airflow cannula, and a pulse oximeter were used to monitor respiration. Signals were digitized at 256-Hz and 512-Hz sampling rates for EEG and ECG recordings, respectively. Infrared video recordings were performed to detect abnormal movements and behaviors during sleep.

PSG Data Analysis

Sleep stages were scored by using 20-second epochs based on EEG, EOG, and mentalis EMG activity according to modified Rechtschaffen and Kales criteria.[33] Total sleep time (time from sleep onset to offset, excluding any awake time), sleep efficiency (percentage of total sleep time of the time spent in bed), sleep latency (time from lights off to sleep onset), and rapid eye movement (REM) latency (time from lights off to REM sleep onset) were computed. The percentage of time spent in each sleep stage (ie, stages N1, N2, N3, and REM) and the micro-arousal index (MAI; frequency of micro-arousal events), apnea-hypopnea index (AHI; frequency of apnea-hypopnea events), and periodic limb movement index (PLMI; frequency of periodic limb movement events) were also computed.

RMMA frequency was scored from EMG traces for the right masseter based on a 20% maximum voluntary clenching (MVC) threshold and video recording data. RMMA was further categorized as a phasic RMMA episode if it included ≥ 3 repetitive bursts over 2 seconds (burst duration: 0.25 to 2.00 seconds; burst interval: < 2 seconds) observed in the masseter EMG recording.[13,34] A burst episode sustained over 2 seconds was considered a tonic RMMA episode.[13,34] A combination of phasic and tonic activity was scored as a mixed RMMA episode.[13,34] RMMA frequency as the number of episodes divided by total sleep time (TST) and prevalence of RMMA episodes ≥ 2/hour and ≥ 4/hour were estimated.[13,18,34]The mean peak amplitude and root mean square (RMS) amplitude for RMMA episodes were computed.[35,36]

For sleep background EMG activity of the mentalis and right masseter muscles over each sleep stage, 20 resting epochs free of any artifacts or motor or respiratory events were randomly selected. Resting epochs during wakefulness were selected from within the period from lights off to sleep onset. For each resting epoch, RMS amplitudes were computed as an estimate of the sleep background EMG activity for the mentalis and right masseter.[20,37]

Statistical Analyses

Student *t* tests (for continuous variables) and chi-square tests (for nominal or categorical variables) with Bonferroni correction were used to compare PSG sleep data, anxiety, depression, and self-reported headache disability, sleep quality, morning body pain, and fatigue in mTBI patients and control participants. Logistic regression was performed to investigate whether mTBI and RMMA frequency could predict moderate to severe levels of headache disability (MIDAS grade ≥ III). Analyses were performed using SPSS Statistics for Windows, version 22.0 (IBM), and significance was set at $P < .05$.

Results

Demographic and Clinical Characteristics

Overall, gender and body mass index were not different in mTBI and control participants, but mTBI patients were on average 7 years older than controls. The most frequent cause of mTBI was a vehicle accident (Table 2).

As expected, a significantly higher number of mTBI patients than controls reported a moderate to severe level of headache disability (50% vs 5%) and poor sleep quality (54% vs 0%) (Table 2). In contrast, no significant between-group difference was found in participant awareness of tooth grinding and/or jaw clenching during sleep. However, the morning following PSG recording, mTBI patients reported a higher prevalence than controls of overall morning fatigue (88% vs 30%, respectively), as well as higher morning body pain intensity (median VAS scores of 22 vs 0) and morning fatigue intensity (median VAS score of 28 vs 9). Nearly half of the mTBI patients reported neck pain (52.2%) and lower back pain (47.8%) (Fig 1). For headache pain, the prevalences of frontal and occipital headache in mTBI patients were 39% and 13%, respectively. Lastly, mTBI patients had higher levels of anxiety and depression than controls, as revealed by higher mean BDI-II scores (12 vs 2, respectively) (Table 2).

Table 2 Effects of Sociodemographic Variables, Self-reported Sleep Macro-Architecture, and RMMA Index on PSG Data for mTBI Patients and Controls

	mTBI (n = 24)	Controls (n = 20)	*t* test or χ^2 *P* value
Sociodemographic			
Gender (male/female)	15/9	8/12	NS
Age (y)	38 ± 11	31 ± 9	.019
Body mass index (kg/m^2)	25 ± 5	23 ± 4	NS
Time elapsed since TBI (d)	72 ± 33		
Cause of TBI (%, self-reported):			
Motor vehicle accident	62		
Fall	21		
Sports-related accident	17		
Sleep bruxer (RMMA index ≥ 2/h)	12 (50)	10 (50)	NS
Sleep bruxer (RMMA index ≥ 4/h)	6 (25)	4 (20)	NS
Self-reports			
Moderate to severe headache disability (MIDAS grade ≥ III)	12 (50)	1 (5)	.001
Poor sleep quality	13 (54)	0 (0)	.000
Aware of sleep bruxism	8 (33)	5 (25)	NS
Morning body pain	15 (63)	5 (25)	.013[a]
Body pain intensity, mean score (0–100 VAS)	22 (0–90)	0 (0–51)	.002
Morning fatigue	21 (88)	6 (30)	.000
Fatigue intensity, mean score (0–100 VAS)	28 (0–80)	9 (0–48)	.000
BAI total anxiety score	7 ± 9	2 ± 5	.016[a]
BDI-II total depression score	12 ± 10	2 ± 4	.000
Sleep macro-architecture[b]			
Total sleep time (min)	409.2 ± 54.6	431.8 ± 60.7	NS
Sleep efficiency (%)	90.6 ± 6.8	93.2 ± 8.0	NS
Sleep latency (min)	20.4 ± 16.2	14.5 ± 23.1	NS
REM latency (min)	79.6 ± 25.9	76.5 ± 20.0	NS
% Stage N1	5.9 ± 3.0	4.7 ± 1.5	NS
% Stage N2	52.0 ± 10.5	52.2 ± 10.3	NS
% Stage N3	18.1 ± 9.5	21.3 ± 6.7	NS
% Stage REM	18.9 ± 5.0	22.0 ± 6.7	NS
AHI (/hr)	2.8 ± 3.0	2.0 ± 1.6	NS
MAI (/hr)	14.5 ± 5.7	14.7 ± 9.4	NS
PLMI (/hr)	6.9 ± 9.9	4.8 ± 6.9	NS
RMMA index for PSG data[b]			
Overall (ep/h)	2.5 ± 2.1	2.4 ± 1.2	NS
Phasic (ep/h)	1.0 ± 1.1	0.9 ± 1.0	NS
Tonic (ep/h)	1.3 ± 1.0	1.3 ± 0.9	NS
Mixed (ep/h)	0.2 ± 0.3	0.3 ± 0.2	NS
Peak amplitude (μV)	148.1 ± 42.8	167.9 ± 39.2	NS
RMS amplitude (μV)	15.5 ± 4.3	15.7 ± 2.5	NS

Values represent mean ± SD or number (frequency). Morning body pain and fatigue intensity show median (min–max). [a]Nonsignificant after Bonferroni correction ($P > .006$). [b]Five mTBI patients and two controls were excluded from RMMA analysis due to an AHI > 10. RMMA = rhythmic masticatory muscle activity; PSG = polysomnographic; mTBI = mild traumatic brain injury; VAS = visual analog scale; BAI = Beck Anxiety Inventory; BDI-II = Beck Depression Inventory-II; AHI = apnea-hypopnea index; MAI = microarousal index; PLMI = periodic limb movement index; ep = episodes; RMS = root mean square.

Sleep Variables

Five mTBI and two control participants were removed from the sleep architecture analysis due to high AHI (> 10, suggesting sleep apnea), leaving 19 mTBI participants and 18 controls for the final PSG data analysis. No significant between-group differences were observed in sleep variables, and all PSG values were within normal clinical ranges (Table 2).[13]

Orofacial Muscle Activity During Sleep

No significant difference was found between groups in the number of subjects presenting an RMMA index of ≥ 2/hour or ≥ 4/hour. Nevertheless, 33% of mTBI participants were aware of SB (ie, self-reported jaw clenching and/or tooth grinding). On the other hand, peak and RMS amplitudes of RMMA in mTBI patients were not significantly different from controls (Table 2).

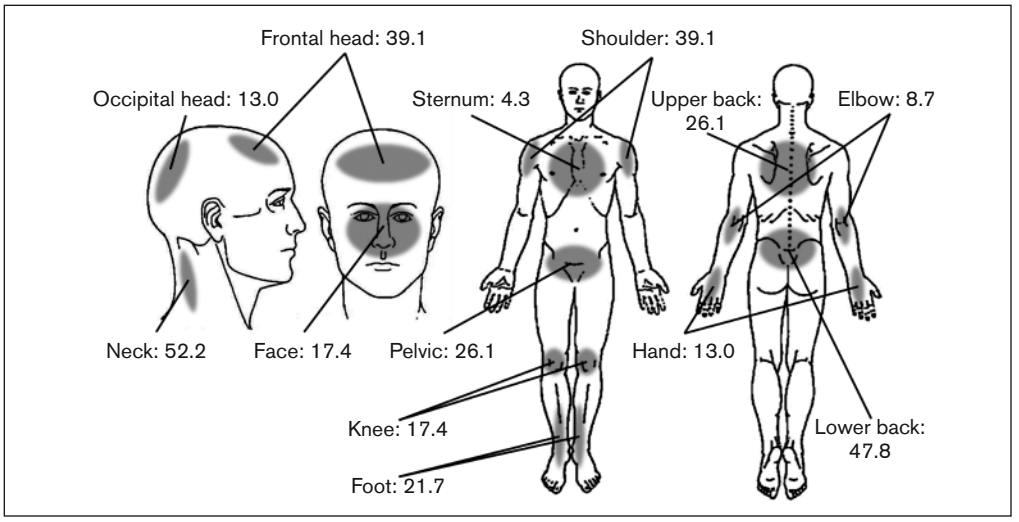

Fig 1 Pain sites in mTBI patients (n = 23; 1 patient missing) and percentage of patients reporting pain for each site.

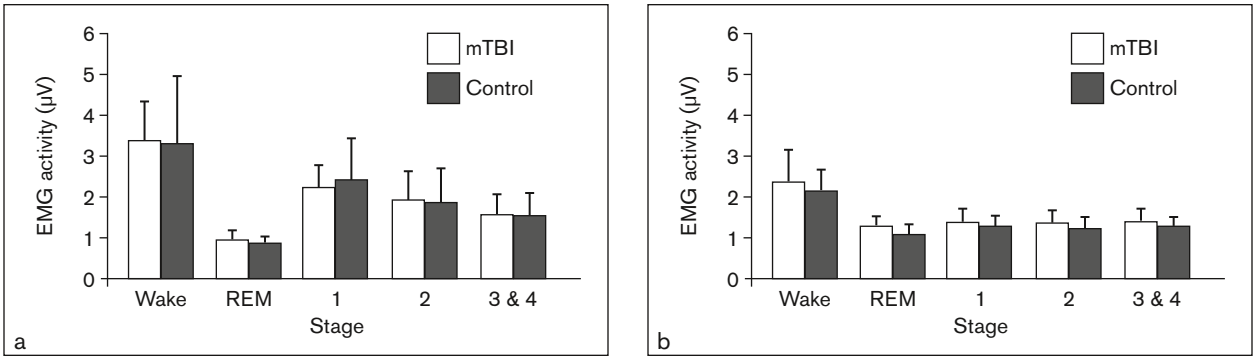

Fig 2 **(a)** Mentalis and **(b)** masseter electromyographic (EMG) activity according to sleep stages in mTBI patients and controls. Although EMG activity dropped during sleep, no significant between-group differences were observed. The columns and error bars show mean and standard deviation (SD). Due to contamination of heartbeat on PSG data, the final sample size of background sleep muscle tone analysis was 12 mTBI patients and 16 controls for the mentalis muscle and 16 mTBI patients and 16 controls for the masseter muscle.

Due to contamination of heartbeat on PSG data, the final sample size for the analysis of background muscle activity during sleep included 12 mTBI patients and 16 controls for the mentalis muscle and 16 mTBI patients and 16 controls for the masseter muscle. Background muscle activity during sleep (excluding RMMA periods) for the mentalis and masseter muscles did not significantly differ between groups (Fig 2).

Gender- and Age-Matched Comparisons

Additional comparison analyses were made by matching gender (8 males and 9 females) and age. Prevalence of moderate to severe headache disability and SB/RMMA and frequency of RMMA episodes were similar to those observed in the unmatched comparison analysis and led to similar results; ie, prevalence of moderate to severe headache disability was significantly higher in mTBI patients ($P < .005$), and no significant differences between groups were found in the prevalence of SB/RMMA or in RMMA index.

Co-occurrence of mTBI and RMMA as Headache Predictor

From the results of the logistic regression in 37 participants (19 mTBI and 18 controls) without sleep apnea, it appears that the presence of mTBI is not a risk factor for SB/RMMA (Fig 3). On the other hand, while mTBI was a strong predictor of moderate to severe headache disability, RMMA frequency was a modest but significant mediator of moderate to severe headache disability in both groups (ORs = 21 and 2, respectively) (Table 3).

Discussion

The present results have shown that although SB/RMMA may be present in some mTBI patients, the number of subjects presenting a high RMMA prevalence was not significantly different between mTBI patients and controls. Still, SB was found to significantly exacerbate headache complaints in both

healthy and mTBI participants. Thus, even if SB may not be more prevalent after mTBI, it remains a potential variable for clinicians to consider if headache management in mTBI patients is not optimal.

A previous study in the same population found that poor sleep in mTBI patients may be partly due to the presence of pain and a predominance of fast brain activity during sleep, as well as due to other factors, such as mood alteration.[28] Furthermore, it was found that sleep complaints in early-phase mTBI patients constitute a potential predictor of headache (OR = 2.9).[11] From the overall analysis of the MIDAS disability index in the present study, it was observed that 50% of mTBI patients complained of moderate to severe headache vs 5% of controls. The logistic regression results, with an OR approaching 21, support previous findings that mTBI is a strong risk factor for headache disability.[4,5]

The exacerbation of headache by SB/RMMA in both the mTBI patients and healthy controls may be coincidental or due to other unknown factors. A higher prevalence of poor sleep quality, more morning pain and fatigue, and more anxiety- and depression-related symptoms were reported by mTBI patients than by controls. Many interrelated conditions may explain the concomitance between SB and headache in mTBI. Since SB may become a moderate mediator of headache disability in both healthy controls and mTBI patients, clinicians may have to take it in consideration when headache management in mTBI patients is not optimal.

Based on the present data, it could not be confirmed that higher jaw muscle activity during sleep explains the exacerbation of headache. This hypothesis is based on a previous study in which higher masseter background muscle activity during sleep was correlated with morning jaw muscle pain in female patients with chronic myofascial TMD.[20] Another possible explanation would be the presence of more subtle breathing conditions, such as respiratory effort related arousal (RERA), as found in the same chronic TMD/orofacial pain population.[38] Again, due to the small sample size, it could not be concluded that anxiety and/or depression were also influential variables on the findings in SB/headache patients.[39]

This study had several limitations. First, the mTBI patients were recruited from a tertiary trauma emergency department, where they requested medical help for mTBI. Therefore, it is possible that mTBI patients who did not request help would have showed different outcomes. However, it is anticipated that this may have had minimal impact on the present results, as it is estimated that only 20% of mTBI patients do not seek medical help at first-line specialized centers.[1] Second, as a means to reduce the influence of confounding factors in data analysis, subjects with sleep apnea and other sleep movement disorders (eg, periodic limb movement or REM behavior disorder) were excluded from the study. Thus, the role of sleep comorbidities in the relationship between headaches and SB/RMMA remains to be assessed. Last, the sample size of the present study was small. One explanation of such a small sample size for PSG recording (only 23% of mTBI patients included in the authors' previous genetic study[28] agreed to participate in the full sleep study) may be due to the fact that these otherwise healthy young subjects were full-time workers, refraining them from spending 2 nights in the sleep laboratory. It is also known that, in mTBI studies, stringent inclusion and exclusion criteria may contribute to low recruitment rates.[40,41] Furthermore, it is possible that mTBI patients are more hesitant to participate in prospective studies, since most of them expect their condition to rapidly return to normal.

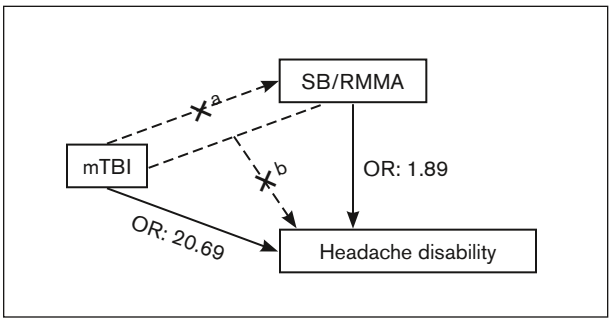

Fig 3 Headache disability predictors by logistic regression. The figure indicates that the presence of mTBI is a risk factor for headache disability but does not mediate SB/RMMA onset **(a, b)**. Secondary higher RMMA frequency is a modest but significant mediator of headache disability independently of mTBI. Hosmer-Lemeshow test results for this model were: χ^2 = 5.10, P = .65. OR = odds ratio.

Table 3 Headache Disability Predictors by Logistic Regression

Predictor	OR	95% CI for OR	*P* value
mTBI	20.69	1.90–224.77	.013
RMMA frequency	1.89	1.06–3.40	.032

CI = confidence interval; OR = odds ratio.

Conclusions

The present results suggest that the occurrence of mTBI is not associated with an increased risk of SB. However, clinicians caring for mTBI patients with poorly controlled headaches should still screen for SB, as it may contribute to their condition.

Acknowledgments

The authors would like to thank Jean-François Giguère for confirmation of mTBI diagnoses and Pierre H. Rompré and Christiane Manzini for their assistance. This study was funded by the Québec Pain Research Network of the Fonds de Recherche du Québec – Santé (FRQ-S), the Ronald Denis Foundation, and the Sacré Coeur Research Center. Gilles J. Lavigne holds a Canada Research Chair in Pain, Sleep, and Traumatic Injuries. The authors report no conflicts of interest.

References

1. Haydel M. Management of mild traumatic brain injury in the emergency department. Emerg Med Pract 2012;14:1–24.
2. Coronado VG, Xu L, Basavaraju SV, et al. Surveillance for traumatic brain injury-related deaths—United States, 1997-2007. MMWR Surveill Summ 2011;60:1–32.
3. Cassidy JD, Carroll LJ, Peloso PM, et al. Incidence, risk factors and prevention of mild traumatic brain injury: Results of the WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. J Rehabil Med 2004;43(suppl):s28–s60.
4. Lavigne G, Khoury S, Chauny JM, Desautels A. Pain and sleep in post-concussion/mild traumatic brain injury. Pain 2015; 156(suppl):s75–s85.
5. Huang TY, Ma HP, Tsai SH, Chiang YH, Hu CJ, Ou J. Sleep duration and sleep quality following acute mild traumatic brain injury: A propensity score analysis. Behav Neurol 2015;2015:378726.
6. Ofek H, Defrin R. The characteristics of chronic central pain after traumatic brain injury. Pain 2007;131:330–340.
7. Sommerauer M, Valko PO, Werth E, Baumann CR. Excessive sleep need following traumatic brain injury: A case-control study of 36 patients. J Sleep Res 2013;22:634–639.
8. Ayalon L, Borodkin K, Dishon L, Kanety H, Dagan Y. Circadian rhythm sleep disorders following mild traumatic brain injury. Neurology 2007;68:1136–1140.
9. Duclos C, Dumont M, Wiseman-Hakes C, et al. Sleep and wake disturbances following traumatic brain injury. Pathol Biol (Paris) 2014;62:252–261.
10. Imbach LL, Valko PO, Li T, et al. Increased sleep need and daytime sleepiness 6 months after traumatic brain injury: A prospective controlled clinical trial. Brain 2015;138:726–735.
11. Chaput G, Giguère JF, Chauny JM, Denis R, Lavigne G. Relationship among subjective sleep complaints, headaches, and mood alterations following a mild traumatic brain injury. Sleep Med 2009;10:713–716.
12. Lobbezoo F, Ahlberg J, Glaros AG, et al. Bruxism defined and graded: An international consensus. J Oral Rehabil 2013;40:2–4.
13. American Academy of Sleep Medicine. International Classification of Sleep Disorders: Diagnostic and Coding Manual, ed 3. Westchester, IL: American Academy of Sleep Medicine, 2014.
14. Lavigne GJ, Huynh N, Kato T, et al. Genesis of sleep bruxism: Motor and autonomic-cardiac interactions. Arch Oral Biol 2007; 52:381–384.
15. Mayer P, Heinzer R, Lavigne G. Sleep bruxism in respiratory medicine practice. Chest 2016;149:262–271.
16. De Luca Canto G, Singh V, Bigal ME, Major PW, Flores-Mir C. Association between tension-type headache and migraine with sleep bruxism: A systematic review. Headache 2014;54: 1460–1469.
17. Abe S, Carra MC, Huynh NT, Rompré PH, Lavigne GJ. Females with sleep bruxism show lower theta and alpha electroencephalographic activity irrespective of transient morning masticatory muscle pain. J Orofac Pain 2013;27:123–134.
18. Rompré PH, Daigle-Landry D, Guitard F, Montplaisir JY, Lavigne GJ. Identification of a sleep bruxism subgroup with a higher risk of pain. J Dent Res 2007;86:837–842.
19. Palinkas M, De Luca Canto G, Rodrigues LA, et al. Comparative capabilities of clinical assessment, diagnostic criteria, and polysomnography in detecting sleep bruxism. J Clin Sleep Med 2015;11:1319–1325.
20. Raphael KG, Janal MN, Sirois DA, et al. Masticatory muscle sleep background electromyographic activity is elevated in myofascial temporomandibular disorder patients. J Oral Rehabil 2013;40:883–891.
21. Schmitter M, Kares-Vrincianu A, Kares H, Bermejo JL, Schindler HJ. Sleep-associated aspects of myofascial pain in the orofacial area among temporomandibular disorder patients and controls. Sleep Med 2015;16:1056–1061.
22. Yachida W, Castrillon EE, Baad-Hansen L, et al. Craniofacial pain and jaw-muscle activity during sleep. J Dent Res 2012;91: 562–567.
23. Ivanhoe CB, Lai JM, Francisco GE. Bruxism after brain injury: Successful treatment with botulinum toxin-A. Arch Phys Med Rehabil 1997;78:1272–1273.
24. Kesikburun S, Alaca R, Aras B, Tuğcu I, Tan AK. Botulinum toxin injection for bruxism associated with brain injury: Case report. J Rehabil Res Dev 2014;51:661–664.
25. Millwood J, Fiske J. Lip-biting in patients with profound neuro-disability. Dent Update 2001;28:105–108.
26. Pidcock FS, Wise JM, Christensen JR. Treatment of severe post-traumatic bruxism with botulinum toxin-A: Case report. J Oral Maxillofac Surg 2002;60:115–117.
27. Van Zandijcke M, Marchau MM. Treatment of bruxism with botulinum toxin injections. J Neurol Neurosurg Psychiatry 1990;53: 530.
28. Khoury S, Chouchou F, Amzica F, et al. Rapid EEG activity during sleep dominates in mild traumatic brain injury patients with acute pain. J Neurotrauma 2013;30:633–641.
29. Carroll LJ, Cassidy JD, Holm L, Kraus J, Coronado VG. The WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. Methodological issues and research recommendations for mild traumatic brain injury: The WHO Collaborating Centre Task Force on Mild Traumatic Brain Injury. J Rehabil Med 2004;36(suppl):s113–s125.
30. Stewart WF, Lipton RB, Dowson AJ, Sawyer J. Development and testing of the Migraine Disability Assessment (MIDAS) Questionnaire to assess headache-related disability. Neurology 2001;56(suppl):s20–s28.
31. Beck AT, Epstein N, Brown G, Steer RA. An inventory for measuring clinical anxiety: Psychometric properties. J Consult Clin Psychol 1988;56:893–897.
32. Beck AT, Steer RA, Carbin MG. Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. Clin Psychol Rev 1988;8:77–100.
33. Rechtschaffen A, Kales A. A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Subjects. Los Angeles: UCLA Brain Information Service, 1973.
34. Lavigne GJ, Guitard F, Rompré PH, Montplaisir JY. Variability in sleep bruxism activity over time. J Sleep Res 2001;10:237–244.
35. Lobbezoo F, Lavigne GJ, Tanguay R, Montplaisir JY. The effect of catecholamine precursor L-dopa on sleep bruxism: A controlled clinical trial. Mov Disord 1997;12:73–78.
36. Raphael KG, Sirois DA, Janal MN, et al. Sleep bruxism and myofascial temporomandibular disorders: A laboratory-based polysomnographic investigation. J Am Dent Assoc 2012;143: 1223–1231.
37. Okura K, Kato T, Montplaisir JY, Sessle BJ, Lavigne GJ. Quantitative analysis of surface EMG activity of cranial and leg muscles across sleep stages in human. Clin Neurophysiol 2006;117:269–278.
38. Dubrovsky B, Raphael KG, Lavigne GJ, et al. Polysomnographic investigation of sleep and respiratory parameters in women with temporomandibular pain disorders. J Clin Sleep Med 2014;10: 195–201.
39. Manfredini D, Lobbezoo F. Role of psychosocial factors in the etiology of bruxism. J Orofac Pain 2009;23:153–166.
40. Isokuortti H, Iverson GL, Kataja A, Brander A, Öhman J, Luoto TM. Who gets head trauma or recruited in mild traumatic brain injury research? J Neurotrauma 2016;33:232–241.
41. Luoto TM, Tenovuo O, Kataja A, Brander A, Öhman J, Iverson GL. Who gets recruited in mild traumatic brain injury research? J Neurotrauma 2013;30:11–16.