Use of a Removable Mandibular Neuroprosthesis for the Reduction of Posttraumatic Stress Disorder (PTSD) and Mild Traumatic Brain Injury/PTSD–Associated Nightmares, Headaches, and Sleep Disturbances

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ABSTRACT

Introduction: Posttraumatic stress disorder (PTSD) has been associated with nighttime headaches (HAs), nightmares (NMs), and difficulty falling or staying asleep (sleep disturbances [SD]). The authors of the current study evaluated the correlative elements of using a removable mandibular neuroprosthesis (RMN) and the reduction of these symptoms in participants diagnosed with PTSD or mild traumatic brain injury (mTBI)/PTSD. The RMN device is a form of specialized dental splint that has a potential to reduce the painful stimuli of bruxing and potential upregulation of threat response systems that may occur during sleep. **Method:** A sample of 32 individuals was selected through random assignment from a volunteer base of 200 volunteers for examination by self-report according to an A-B-A-B design. The sample included 25 men and 7 women between the ages of 21 and 65; 21 had military experience and 11 were civilians. Participants were asked to rate the frequency and intensity of their HAs, NMs, and SD during each phase. Their responses were scored using a custom survey (equivalent forms reliability) that provides ratio-scaled results for symptom frequency and intensity. The original number of participants was 35 with three participants dropping out before the conclusion of the study. **Results:** Survey scores for PTSD-related sleep symptoms were relatively high at baseline ($\bar{x}$ = 0.52) and significantly lower in the first experimental phase ($\bar{x}$ = 0.20). Scores in the second experimental phase were likewise lower ($\bar{x}$ = 0.38). Significant reductions in symptoms were reported across all three dimensions. **Discussion:** All participants reported some improvement in symptoms while using the device. No participants reported worsening of any symptoms as a result of using the RMN. Participants commonly reported that improvements in symptoms were immediate and did not diminish over time. Data indicate that there is a negative correlation between the use of an RMN and the reduction of HAs, NMs, and SD in persons diagnosed with PTSD or mTBI/PTSD.

KEYWORDS: *PTSD, mTBI, nightmares, headaches, sleep disturbances, DIMS, bruxism, bruxing, splint*

Introduction

Approximately 22% of armed services personnel who served in Iraq or Afghanistan between 2002 and 2008 were diagnosed with PTSD.[1] The most commonly reported symptoms of PTSD include reliving an event through NMs and SD.[2] Previous research has indicated that about 61% of persons with PTSD may report experiencing NMs and about 74% may report experiencing insomnia.[3]

SDs are a frequent residual complaint after treatments of PTSD that are termed successful and may constitute a core feature of the disorder.[4] As many as 48% of patients who no longer meet PTSD diagnostic criteria after treatment with cognitive-behavioral therapy may still report insomnia.[5] Implosive therapy, involving systematic exposure to traumatic memories and skills training interventions directed at improving social competence in interpersonal interactions, has been shown to improve some outward symptoms of PTSD, although sleep symptoms may still occur.[6] SDs are associated with an increased risk of suicide ideation and attempt,[7] and improved sleep has been correlated with improvement in the severity of PTSD symptoms.[8]

In addition, mTBI has been associated with depression,[9] multiple neurological memory problems,[10] and HAs independent of PTSD.[11] Recent data indicate that approximately 500 of every 100,000 persons visiting emergency departments in the United States have an mTBI, making it the most common neurological condition in the United States.[12] Treatment of these symptoms is complicated by a common comorbidity with PTSD.[13] Sleep difficulties may mediate the effect of a positive mTBI screening on the later development of mental health disorders and may act as an early indicator for risk of developing PTSD or depression.[14]

Sleep bruxism is an arousal-related phenomenon[15] that has been associated with PTSD.[16] Bruxism leads to masticatory muscle tenderness and temporomandibular joint

(TMJ) pain.[17] Previous research indicates that an intraoral splint may be used to treat HAs in bruxing patients.[18] The neuromodulator was classified by the US Food and Drug Administration in 1998 as a nonsignificant risk.[19] The authors intended in this study to replicate and expand on the previous findings that sleeping with an RMN may alleviate chronic NMs, chronic HAs, and SDs in military and civilian participants with a diagnosis of PTSD.[20]

The current study was constructed in an A-B-A-B format mandating that participants refrained from RMN use after the initial week of use and resumed use after a week of nonuse. The initial case series, a retrospective observational study, recorded general patient impressions of improvement that were perceived and recorded as a percentage improvement in three symptoms: HAs, NMs, and SDs. Discontinuation of the use of the device did not occur in the initial case series. This study used specific pretreatment and posttreatment inventory questions (derived from validated psychological inventories), which were subjected to statistical validation as useful for measuring change in the clinical outcome measures in HA, NM, and sleep continuity. These outcome measures were subjected to statistical validation for internal consistency as well as for effect of the device use. The initial case series did not use statistical methodology for analysis of treatment device effect or internal validation.

The initial case series was single practitioner validated by retrospective chart review. The current study separated the treatment arm from the evaluation arm, using nonclinical evaluators administering validated effect instruments to determine effect.

Materials and Methods

Ethical approval for the current study was obtained from Fox Commercial Institutional Review Board LLC (study #140130-001). The study was conducted independently and apart from any university, firm, or government agency. Full disclosure was provided to each participant and signed informed consent documents were required from each as a condition for inclusion in the study.

Participants

All participants were volunteers who responded to advertisements on six radio stations owned by PMB Broadcasting in Columbus, Georgia; television news segments run by WRBL and WTVM; representatives at the 2014 Thunder in the Valley air show (Columbus); and flyers that were distributed from Dr Moeller's oral surgery practice.

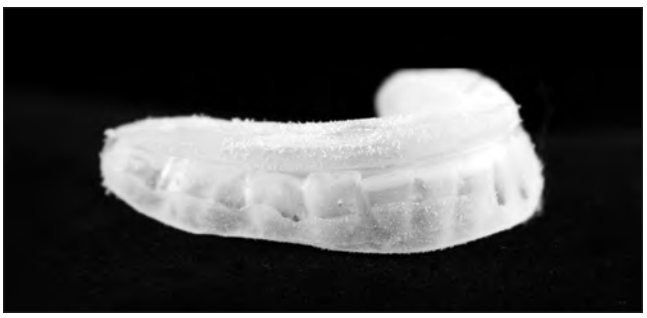
To be included in the study, each participant was required to be between the ages of 21 and 65 at the time of participation; to be in reasonably good physical and mental health as determined by Dr Moeller; to have been diagnosed with PTSD by a licensed professional; to have been symptomatic for at least 3 years before participation; and to express qualifying sensitivity according to masticatory muscle trigger point (MMTP) assessments as determined by Dr Moeller.

The sample included 32 individuals (n = 32), 25 males and 7 females. Of all participants, 21 had military experience and 11 were of civilian symptomatic categorization. In age, 5 were between 21 and 25 years; 7 were between 26 and 30 years; 8 were between 31 and 40 years; 7 were between 41 and 50 years; and 5 were between 51 and 60 years. In terms of race, 11 were white, 10 were African-American, and 11 reported race as "other." Twelve participants were taking psychotropic medications before and during the present study. The original number of participants was 35, with three participants dropping out before conclusion of the study.

Apparatus

The RMN (Figure 1) is constructed by taking an alginate impression of the mandibular teeth and using it to make a model in dental stone. A soft RMN is then fabricated from thermoplastic material on the model and is refined manually at chair-side for maximum interarch dental stability. The device is constructed to separate the maxillary and mandibular arches to a distance that will reduce MMTP sensitivity and will be thick enough to reduce dental pain on maximum clinching.

Figure 1 *The removable mandibular neuromodulating prosthesis.*



Photograph by John Duffey, 2014.

Methods

Participants were asked to complete a self-report survey that described their PTSD-related sleep symptoms (nighttime HAs, NMs, and SDs). The design consisted of the standard four phases: baseline (A1), experimental (B1), control (A2), and second experimental (B2), in that order. Participants were first asked to report their experiences for the previous 7 nights, and these scores were designated as A1. Each was then asked to wear

the RMN while sleeping for 7 consecutive nights. When these 7 nights were completed, participants were asked to report their experiences for the previous 5 nights of sleep, allowing 2 nights to acclimate to the sensations of sleeping with the device in their mouth. These scores were designated as B1.

Participants were then asked to refrain from using the device for the next 7 nights and to report the experiences of all 7. These scores were designated as A2. Each was then asked to resume the use of the device for another 7 nights and, when completed, to report their experiences for the last 5. These scores were designated as B2. When participants were instructed to use the device while sleeping, they were also instructed to use the device any time they napped during the day.

Measures

The present study measured the frequency and intensity of three dimensions of PTSD sleep symptoms (HAs, NMs, and SDs) by self-report, according to two forms of an equivalent forms reliable survey referred to as the PTSD Night Symptoms Index (PNSI). The PNSI-7 (a 7-day version of the survey, included as Appendix A) was administered during the baseline and control phases (A1 and A2). The PNSI-5 (a 5-day version of the survey, included as Appendix B) was administered during the experimental phases (B1 and B2). The PNSI inventories were validated through comparative scoring with the Disturbing Dreams and Nightmares Severity Index (DDNSI) for PNSI items 2a and 2b, the Veterans Administration (VA)/Department of Defense (DoD) Pain Supplemental Questionnaire (VDPSQ) for PNSI items 1a and 1b, and the Iowa Sleep Disturbances Inventory (ISDI) for PNSI items 3a and 3b. PNSI scores tended to reflect similar scores seen in related items of the aforementioned assessment inventories and thus render reliable data.

Scores for each of the three dimensions may be calculated by multiplying the measure of frequency, as the number of nights on which symptoms were experienced (item A of each dimension), by the perceived intensity of the symptoms (item B of each dimension). Each product is then divided by the highest possible score to provide a value on a scale between 0 and 1 for the purpose of comparison. Total scores for each completed survey may be calculated by adding together the products of each dimension and then dividing by the highest possible score for the whole survey.

The frequency measure of each dimension is a value on a ratio scale. The intensity measure of each dimension is a value on an ordinal scale. The products of these two numbers may be sensibly ranked against one another but should not be subjected to further quantitative analysis. Significant differences may be examined according to a nonparametric test, such as the Wilcoxon signed-rank test used here, and central tendencies may be expressed as medians.

Results

Total scores, which incorporate frequency and intensity from all three symptoms, were relatively high at baseline ($\bar{x} = 0.53$). Total scores from the B1 phase were significantly lower ($\bar{x} = 0.21$), $z = -2.53$, $p = .0001$. Those from the B2 phase were likewise lower ($\bar{x} = 0.34$), $z = -2.94$, $p = .0001$. Scores from each survey dimension, which incorporate frequency and intensity for one symptom, were lower during the B1 and B2 phases than during the A1 and A2 phases (Table 1).

All survey dimensions show significant reductions in reports of symptoms. Scores from B1 were significantly lower than baseline scores for HAs ($z = -2.80$, $p = .005$), for NMs ($z = -2.01$, $p = .04$), and for SDs ($z = -2.93$, $p = .003$). Scores from B2 were likewise lower for HAs ($z = -2.13$, $p = .03$), NMs ($z = -2.60$, $p = .001$), and SDs ($z = -2.93$, $p = .003$). The mean scores for both A1 and A2 phases for each category maintained exceptional similarity that indicates no impact on the results due to maturation.

Table 1 *Sleep Symptom Scores for All Participants*

	Phase A1	Phase B1	Phase A2	Phase B2
HAs	0.39	0.19	0.40	0.19
NMs	0.43	0.20	0.37	0.16
SDs	0.78	0.28	0.65	0.26

There were 14 participants who had been diagnosed with an mTBI and 15 participants who were confirmed clinically to not have an mTBI before participation in the present study. Thirteen participants were taking psychotropic medications during the study, while 16 did not take any psychotropic medications during the study. These numbers and conditions remained constant throughout the study.

Overall hypothesis testing (H_0: $\mu = 0.51$; H_1: μ not = 0.51) through one-sample *t*-test shows baseline and return-to-baseline figures ($\bar{x}_1 = 0.51/\bar{x}_2 = 0.51$ with 95% confidence intervals (CIs)$_1$ of 0.45–0.57 and 95% CI$_2$ of 0.45–0.57 ($p_1 = .98$, $p_2 = .94$) are at the parameter mean (μ) (accept H_0). This indicates that the sample means ($\bar{x}$) for A1 and A2 represent the parameter mean (μ) with 95% CI. Overall hypothesis testing (H_0: $\mu = 0.50$; H_1: μ not = 0.50) through one-sample *t*-test shows treatment phases 1 and 2 ($\bar{x}_1 = 0.21/\bar{x}_2 = 0.34$ with 95% CI$_1$ being 0.16–0.26 and 95% CI$_2$ being 0.28–0.42 ($p_1 = .0001$, $p_2 = .0001$) are significantly different from μ (reject H_0) (Table 2).

Table 2 *Sleep Symptom Scores for Participants With mTBI*

	A1	B1	A2	B2
Headaches	0.38	0.15	0.50	0.16
Nightmares	0.68	0.25	0.56	0.25
Sleep disturbances	0.88	0.22	0.53	0.22

Medians of raw values of survey items illustrate a connection between sleep symptoms experienced by participants with and without mTBI. Raw values of symptom frequency from B1 and B2 were multiplied by a coefficient (7/5) to put them on the same scale as values from A1 and A2. Survey instruments from B1 and B2 collected data pertaining to the previous 5 nights, while those from A1 and A2 pertained to the previous 7 nights. Raw values of symptom intensity did not require any adjustments.

The survey items pertaining to HA frequency (Figure 2) and HA intensity (Figure 3) clearly show that reports of HA symptoms were lower during the experimental phases. They also show that HA reports tended to return slightly above baseline when use of the RMN was discontinued for a week. Reports of HAs were lower at the end of the experiment than during baseline and control phases.

Those survey items pertaining to NM frequency (Figure 4) and NM intensity (Figure 5) show that reports of NMs were lower during the experimental phases. Reports of NM frequency returned near baseline when use of the device was discontinued, but reports of NM intensity remained notably lower than baseline during that same phase. Reports of NMs were lower at the end of the experiment than during baseline and control phases.

Survey items pertaining to SD frequency (Figure 6) and SD intensity (Figure 7) show that reports of SDs were much lower during the experimental phases. Both SD measures remained lower than baseline when the device was discontinued and were lower at the end of the experiment than during baseline and control phases. Reductions in reports of SDs were the most pronounced of the three symptoms measured.

Figure 2 *Headache frequency scores collected during the study. Solid lines = PTSD; dashed lines = mTBI/PTSD.*

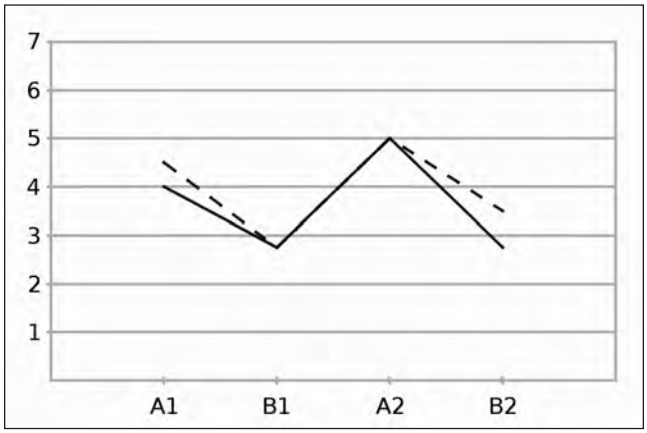


Figure 3 *Nightmare frequency scores collected during the study. Solid lines = PTSD; dashed lines = mTBI/PTSD.*

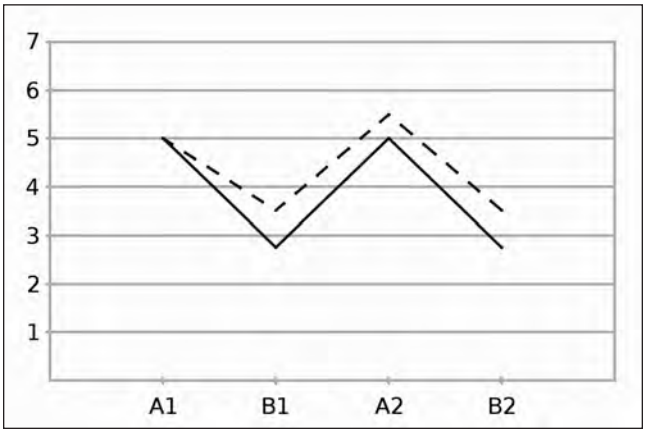


Figure 4 *Nightmare frequency scores collected during the study. Solid lines = PTSD; dashed lines = mTBI/PTSD.*

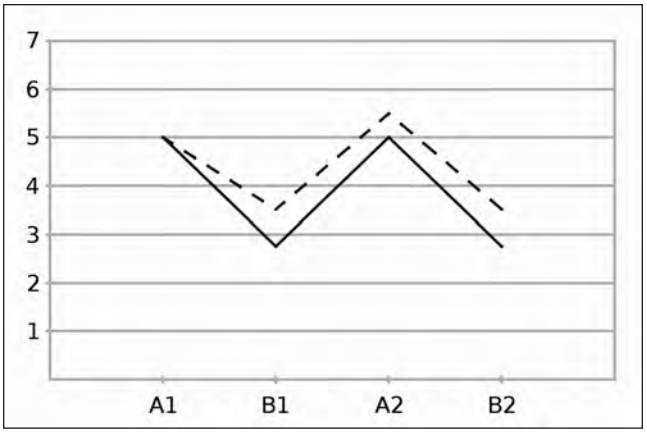


Figure 5 *Nightmare intensity scores collected during the study. Solid lines = PTSD; dashed lines = mTBI/PTSD.*

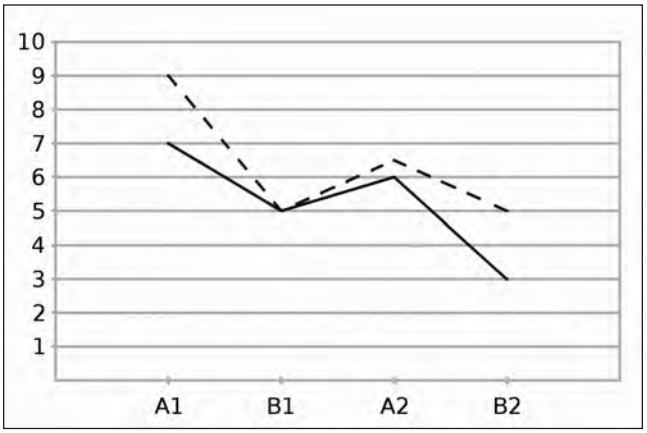


Figure 6 *Sleep disturbance frequency scores collected during the study. Solid lines = PTSD; dashed lines = mTBI/PTSD.*

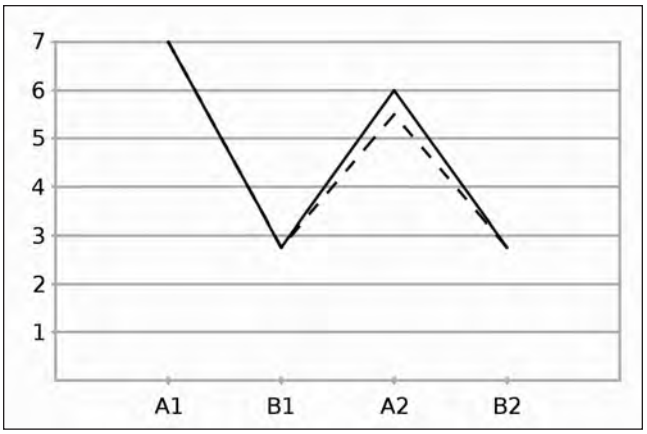
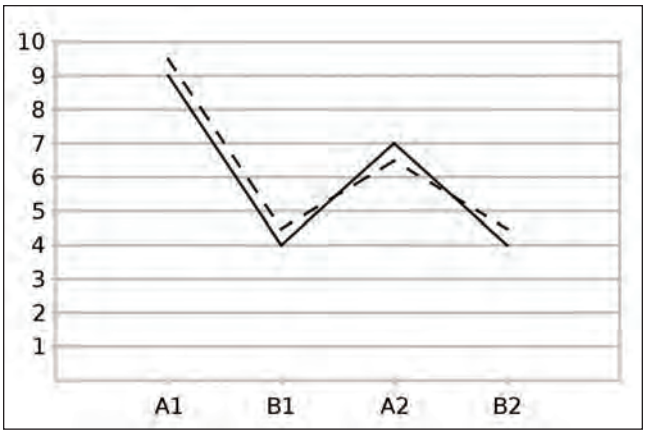


Figure 7 *Sleep disturbance intensity scores collected during the study. Solid lines = PTSD; dashed lines = mTBI/PTSD.*



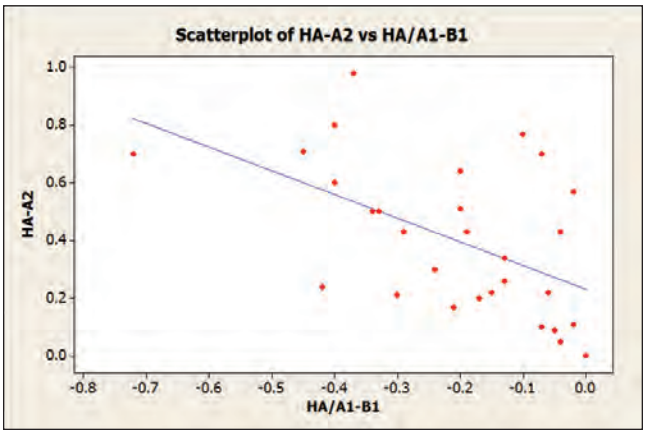
Discussion

Although this is a correlational study where causation cannot be determined, there is room to suggest and support further experimental investigation into a possible cause-and-effect relationship. A reduction of movement during bruxing may lead to decreased activity across the trigeminal nerve (CN-V), which may also contribute to the observed reduction in symptoms. The device may reduce bruxing frequency through behavioral response blocking, abolishing the association between the bruxing behavior and any reinforcing stimuli. It is also possible that the effect on CN-V may precipitate a neuromodulatory effect in the brain.

It is known that not all bite guards will provide for the same correlation as the RMN. The most striking difference between common bite guards and the RMN is thickness. A thin bite guard may inadequately reduce pressure on the TMJ and inadequately reduce stimulation of CN-V. If a portion of the effect depends on behavioral response blocking, then it may also be true that an ideal interarch distance must be achieved to eliminate reinforcing stimuli.

It was noted during the study that participants who were under the influence of pain relievers or muscle relaxers tended to score lower on MMTP examination, but these artificially lowered scores were not associated with lesser symptoms. No participants reported worsening of any symptoms as a result of using the RMN at any point. One participant noted increased salivation, but no other side effects or adverse reactions were reported. No medication changes occurred in any subject during the course of this study. The distribution histogram shows a normal bell curve indicating an equal distribution of mTbi, non-mTbi, pharmacotherapy, and nonpharmacotherapy subjects (Figure 8). Thus, confounding factors such as brain injury and presence of psychotropics are balanced with factors of nonpsychotropic and absence of brain injury in PTSD subjects. (Variables of medication, injury, and the absence of either/both are kept constant during experimentation and do not compete/interfere with the independent variable's effect on the dependent variable.)

Figure 8 *Scatter plot with regression line for the effect of the RNM on headaches.*



Data suggest that use of an RMN may be considered a useful adjunct to psychological and pharmaceutical therapy, which should be explored further under experimental designs. Cognitive-behavioral and exposure therapies have been shown to be comparable to each other in reducing the experience of general PTSD symptoms.[21] Prazosin, a selective α_1 blocker and selectivemelatonin receptor (MT_3) antagonist, has been shown to reduce the experience of trauma NMs in PTSD patients.[22] Because some participants were on antidepressant and pain relief agents while displaying the same correlative response as those who were not, it is possible that these three therapies may be administered simultaneously, and it is therefore recommended that future studies should explore the effects of a combination treatment of sleeping with an RMN, psychotherapy, and prazosin on persons experiencing PTSD and mTBI symptoms.

Scatter plot analysis with regression lines in HAs, NMs, and SD indicates a strong negative correlation between

baseline and treatment phases (see Figures 9 through 11). The SD scores, rendered in a boxplot graph (Figure 12), further support the strong negative correlation between treatment and baseline phases seen in the previous line graphs.

Figure 9 *Scatter plot of nightmare scores and effect of RNM in phases A1 and B1 with regression line.*

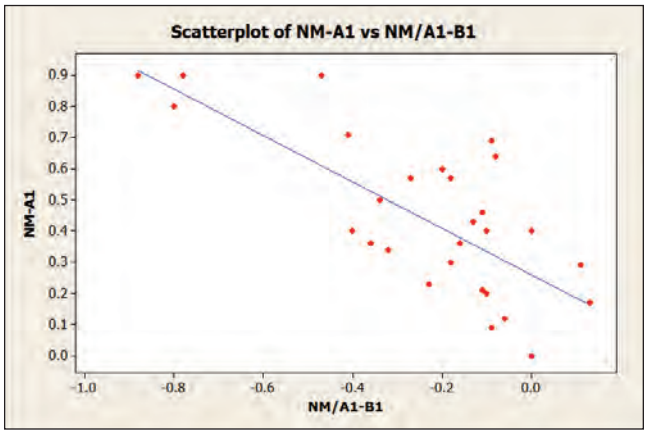


Figure 10 *Scatter plot of disrupted sleep and effect of RNM in phases A2 and B2 with regression line.*

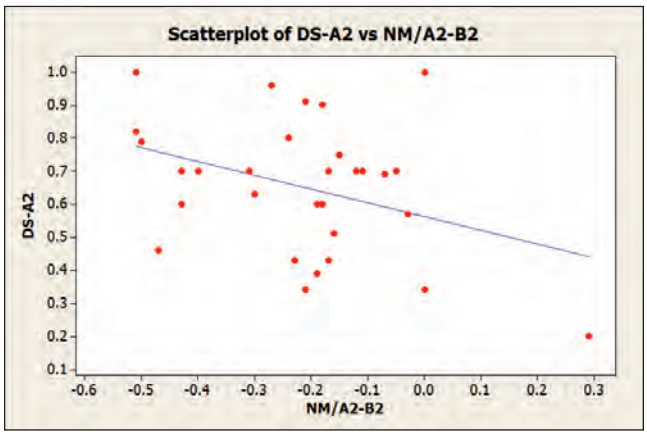


Figure 11 *Box plot graph of disrupted sleep levels across all phases of the study.*

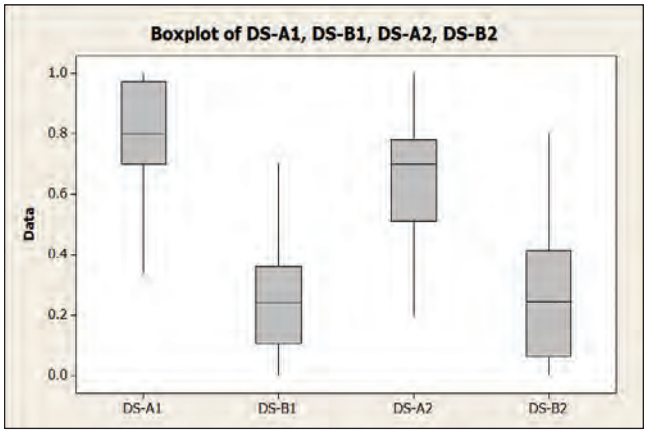
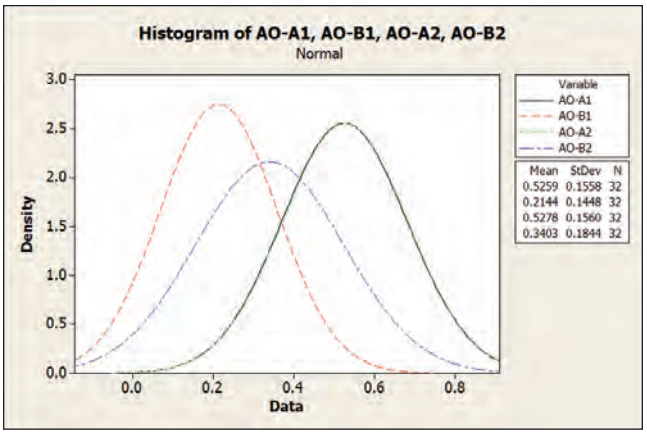


Figure 12 *Frequency distributions for all phases of the study illustrating a normal representative sample for each.*



Speculated Mechanisms for Action of the RNM

PTSD and mTBI/PTSD are associated with increased sympathetic activity. The sympathetic nervous system is activated under the conditions of physical, psychological, and psychosocial stress. This activation may also affect motor function by modulating afferent activity from muscle spindles that are highly concentrated in jaw-closing muscles.[24,27] Emotionally stressful states measured by urinary catecholamines may affect the development of bruxism.[26] It is known that rhythmic masticatory muscle episodes, seen in electromyographic studies of sleep bruxism, are preceded by physiological activation of the central nervous and sympathetic cardiac systems.[29] Transcranial magnetic stimulation of sleep bruxing patients suggested that an abnormal excitability of the central jaw motor pathways may be present in sleep bruxing patients and that this increased excitability could derive from an impaired modulation of brainstem inhibitory circuits and not from altered cortical mechanisms. The authors indicated that this supported the contention that bruxism is mainly centrally mediated and that it involves subcortical structures.[28] In a nonhuman primate study, it was suggested that the onset of rhythmic masticatory muscle activity and sleep bruxing episodes during sleep are under the influences of brief transient activity of the brainstem arousal–reticular ascending system contributing to the increase in activity in autonomic–cardiac and motor modulatory networks.[29] One such network is the central pattern generator in the trigeminal nucleus, which produces masticatory movements during mastication. The output of these neurons is modified by inputs that may descend from higher centers in the brain and by feedback from sensory receptors.[25]

Mechanoreceptors in the muscles of mastication, and the periodontal ligaments around the roots of the teeth have particularly powerful effects on movement parameters.[34,35] It is possible that periodontal mechanoreceptors

and muscle spindles of jaw closing muscles facilitate adrenal nerve activity.[23]

In addition, it has been concluded that gradual increase of intraoral vertical dimension of occlusion may result in decreased masseter muscle sensitivity. This suggests that peripheral sensory plasticity may occur after changes in occlusal vertical dimension.[37] The effects of varying the vertical thickness of an occlusal splint on nocturnal electromyographic activities of the masseter and temporalis muscles has been measured.[33] It is possible that the neurons of the trigeminal mesencephalic nucleus play an important role in the regulation of occlusal vertical dimension (distance between the jaws) because this nucleus receives projection from the jaw-closing muscle spindles and periodontal mechanoreceptors.[31]

It is also suggested that changes in sympathetic outflow to muscle spindles can change rhythmic movement patterns in the masticatory systems of rabbits. This research has important implications for the control of motor functions in states of high sympathetic activity.[30]

Masseter length has also been found to determine muscle spindle reflex excitability during jaw closing movements. In a study using lateral cephalometric radiographs to establish facial heights in patients, it was found that shorter facial heights were associated with stronger spindle reflexes.[36]

The RMN increases the vertical dimension of occlusion and may change the jaw muscle spindle discharge characteristics. It also may decrease the periodontal mechanoreceptor feedback to the trigeminal nucleus The trigeminocervical nucleus is the largest nucleus in the brain, and its tracts radiate widely to include the thalamus and the dorsal prefrontal cortex. Increased output from the trigeminocervical nucleus may radiate to the prefrontal cortex, where dreaming occurs. Clinically, the patients report that their NMs are transformed to nonthreatening military-themed dreams.

There may also exist a positive feedback loop between the bruxism causing pain, which may in turn cause an increase in output from sympathetic upregulation, which in turn increases the sensitivity of the jaw muscle spindles, which then causes the bruxism to increase. The increased upregulation of the trigimenal nucleus from jaw muscle spindle and periodontal output tracts may cause increase in output to the reticular activating system. This may account for the high number of patients falling asleep immediately after RNM placement. The shorter facial height patients, many of whom have deep bites (upper incisors well overlapping the mandibular incisors), seem to have some of the most dramatic results from use of this device.

The wear that occurs on the occlusal surface of the device from bruxing will cause the symptoms (NMs, SDs, etc.) to recur after varying periods of time. Restoration of the original vertical dimension of occlusion (original RNM distance) brings the same clinical result as the original device. This may support the idea that the jaw muscle spindles play the most important role in the physiological action of this device.

Sample size, although small, represents a group of PTSD/mTBI subjects with significant manifestation of comorbid disorder processes (i.e., HAs, NMs, and SDs), which represents a legitimate statistical representation to measure response to this therapy as a pilot study for further research. Certainly, future research, including true experiment designs, should be conducted to define a cause-and-effect relationship between the RMN and the reduction in sleep symptomology in PTSD subjects. Additional use of this device as an adjunct to cognitive-behavioral therapy and acute resolution therapy should be explored.

Conclusions

The use of an RMN is positively correlated with a reduction in the experience of PTSD-related sleep symptoms independent of psychological and pharmaceutical therapies. Scores for nighttime HAs, NMs, and difficulties falling and staying asleep significantly lessened while the device was in use. Reductions were seen in both frequency and intensity. Participants commonly reported that improvements in symptoms were immediate and did not diminish over time.

Disclosures

The authors have nothing to disclose.

Funding and Affiliation

This original research was not affiliated with any university or other institution. Primary financial support was provided by Dr Donald Moeller and secondary support by research staff. All research materials were generated and constructed by Dr Moeller and staff. No third-party manufacturers were involved in this study. The authors have no financial relationships relevant to this article to disclose.

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LTC (Ret) Moeller, DC graduated from dental school at the University of California with his DDS, medical school at the University of Alabama with his MD, and graduate school at Hampton University with an MA in biological sciences. He did his residency in oral and maxillofacial surgery at Walter Reed Army Medical Center and his transitional medical residency at Columbus Regional Medical Center in Columbus, Georgia. He is retired from the US Army Dental Corps. While in the Army, he was deployed as a medical corpsman to Vietnam and as an oral and maxillofacial surgeon during Desert Storm. He is board certified in oral and maxillofacial surgery and has a private practice in Columbus, Georgia. He occasionally teaches health science courses at Columbus Technical College in Columbus, Georgia.

Mr Duffey is an ordained minister and former military law enforcement officer (USA-MP) specializing in crimes against humanity, children, and domestic violence. He has an interest in the study and treatment of posttraumatic stress disorder in military, law enforcement, and abuse/trauma victims. He is currently studying psychology at Auburn University with a goal of achieving a clinical PhD.

Ms Goolsby is a student at Columbus State University, expecting a bachelors of science in psychology in May 2015. She aspires to attain a PhD in a field related to clinical research in psychology.

Mr Gallimore is a student of psychology at Columbus State University, expecting a bachelors of science in December 2014. He has 15 years of experience in a number of computer-related, technical fields. He intends to acquire a PhD in cognitive and behavioral psychology in order to work in professional research settings.

APPENDIX A

PTSD NIGHT SYMPTOMS INDEX (PNSI-7)

PARTICIPANT NUMBER: _____ FORM SERIES: _____

Please take your time and answer the following six questions as accurately as you can. Do not provide your name or any other identifiable information on this page. All information recorded here will be stored anonymously. Thank you for your participation.

No.	ITEM	1	2	3	4	5	6	7	8	9	10
1a	On how many nights over the last 7 nights have you experienced headaches?								/ / /	/ / /	/ / /
1b	On a scale of 1 to 10, with 10 being the most severe, what was the intensity of these headaches?										
2a	On how many nights over the past 7 nights have you experienced nightmares?								/ / /	/ / /	/ / /
2b	When you experienced these nightmares what was their average intensity on a scale of 1 to 10 (10 being the most severe)										
3a	On how many nights over the past 7 nights did you have difficulty falling asleep or staying asleep?								/ / /	/ / /	/ / /
3b	On those sleepless nights, how difficult was it to get useful rest? (scale of 1 to 10, with 10 being impossible)										

Please allow staff to complete this form below this line. Thank you.

AREA	A	B	Product	n/70 Score
1				/70 =
2				/70 =
3				/70 =

Note: Area 1 = HEADACHE, Area 2 = NIGHTMARE, Area 3 = SLEEP DISTURBANCE

OVERALL

1	2	3	SUM	n/210	%
				/210	

APPENDIX B

PTSD NIGHT SYMPTOMS INDEX (PNSI-5)

PARTICIPANT NUMBER: _____ FORM SERIES: _____

Please take your time and answer the following six questions as accurately as you can. Do not provide your name or any other identifiable information on this page. All information recorded here will be stored anonymously. Thank you for your participation.

No.	ITEM	1	2	3	4	5	6	7	8	9	10
1a	On how many nights over the last 5 nights have you experienced headaches?						/ / /	/ / /	/ / /	/ / /	/ / /
1b	On a scale of 1 to 10, with 10 being the most severe, what was the intensity of these headaches?										
2a	On how many nights over the past 5 nights have you experienced nightmares?						/ / /	/ / /	/ / /	/ / /	/ / /
2b	When you experienced these nightmares what was their average intensity on a scale of 1 to 10 (10 being the most severe)										
3a	On how many nights over the past 5 nights did you have difficulty falling asleep or staying asleep?						/ / /	/ / /	/ / /	/ / /	/ / /
3b	On those sleepless nights, how difficult was it to get useful rest? (scale of 1 to 10, with 10 being impossible)										

Please allow staff to complete this form below this line. Thank you.

AREA	A	B	Product	n/50 Score
1				/50 =
2				/50 =
3				/50 =

Note: Area 1 = HEADACHE, Area 2 = NIGHTMARE, Area 3 = SLEEP DISTURBANCE

OVERALL

1	2	3	SUM	n/150	%
				/150	