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The effect of nasal continuous positive airway pressure on the symptoms of Gulf War illness

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Abstract

Purpose We performed a pilot study to determine whether nasal continuous positive airway pressure (CPAP) alleviates the symptoms of veterans with Gulf War illness (GWI) and sleep disordered breathing (SDB).

Methods Eighteen male veterans with GWI and SDB recruited by advertisement, participated in a randomized, single-masked, sham-controlled treatment trial. Participants received 3 weeks of treatment during sleep with either therapeutic nasal CPAP or sham nasal CPAP. Using validated questionnaires, pain, fatigue, cognitive function, sleep disturbance, and general health were assessed by self-report before and after treatment. One of the participants assigned to therapeutic CPAP was excluded from the trial before starting treatment, leaving 17 participants.

Results Compared to the nine sham nasal CPAP recipients, the eight participants receiving therapeutic nasal CPAP experienced improvements in pain (34%; $p=0.0008$), fatigue (38%; $p=0.0002$), cognitive function (33%; $p=0.004$), sleep quality (41%; $p=0.0003$), physical health (34%; $p=0.0003$), and mental health (16%; $p=0.03$).

Conclusions Our findings in this pilot study suggest that nasal CPAP may greatly improve symptoms in veterans with GWI and SDB.

Introduction

Up to half the veterans of the first Persian Gulf War experience a group of symptoms including fatigue, insomnia, body pain, mood and cognitive disturbances known as Gulf War illness (GWI) [1–3]. Because the cause of these symptoms is unknown, treatment is directed at symptoms rather than at a specific etiology with limited efficacy [4].

The symptoms of GWI are not unique to veterans of the first Gulf War. Non-veterans with headache syndromes, fibromyalgia, and irritable bowel syndrome, known as the functional somatic syndromes (FSS), experience the same variety of symptoms as veterans with GWI [5, 6]. Because these syndromes have been associated with sleep disordered breathing (SDB) [7–10], in a previous study [11], we compared the inspiratory airflow dynamics during sleep between veterans with GWI and healthy veterans of the first Gulf War finding a much higher prevalence of SDB among veterans with GWI. Thus, veterans with GWI resemble other FSS patients in their symptoms and in having SDB.

Although FSS patients are characterized by SDB, the role of SDB in their symptoms remains uncertain. In a clinical series of female fibromyalgia patients, splinting of the pharyngeal airway during sleep with nasal continuous positive airway pressure (CPAP) reduced the severity of their symptoms of fatigue, pain, gastrointestinal discomfort, and insomnia [10]. The study, however, was not performed

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with a sham nasal CPAP control. To establish a role for SDB in the symptoms of the FSS, a sham-controlled CPAP study is needed.

We hypothesized that SDB contributes to the symptoms of GWI. To investigate this hypothesis, we randomized veterans with GWI and SDB into a sham-controlled pilot study of treatment with nasal CPAP to examine the effect upon their symptoms. As secondary objectives, we investigated the impact of treatment with nasal CPAP on standard parameters of sleep and correlated the post-treatment changes in sleep parameters with changes in symptoms of GWI.

Methods

Study design

In our study of inspiratory airflow dynamics during sleep in GWI, we identified 18 male veterans with GWI and SDB [11] who had been recruited by advertisement. These 18 participants were randomized into a masked, parallel-group comparison of the effects of nasal CPAP vs. sham on the symptoms of GWI. The protocol was approved by the institutional review board of the DVA Medical Center-Northport and was registered as a clinical trial (www. clinicaltrials.gov; identifier: NCT00252629).

Study participants

Our definition of GWI has been fully discussed in our previous study [11]. Briefly, the 18 veterans with GWI and SDB who participated in this treatment trial were deployed to the Persian Gulf between August 1990 and August 1991 and reported onset after August 1990 of fatigue, pain involving at least two body regions and cognitive dysfunction (memory or concentration problems). All three symptoms had lasted for more than 6 months, were present at the time of screening, and were unexplained by any clearly defined organic illness. The participants were recruited between January 2006 and July 2008 and were registered in the Gulf War Veterans Registry. Exclusion criteria included alcohol abuse, active clinical depression, active post-traumatic stress disorder, current use of opiates, and a prior diagnosis of sleep apnea.

Polysomnography

Participants underwent an initial full-night polysomnogram to characterize sleep architecture and respiration. Sleep, EKG, and oxyhemoglobin saturation were monitored using standard clinical methods [12]. Airflow was monitored with a nasal pressure catheter and thoraco-abdominal movement with piezoelectric belts.

To be consistent with prior studies from this group [9, 10], polysomnograms were staged using 30-s epochs and strict Rechtschaffen and Kales criteria [13] except for the scoring of arousals using the 3 s frequency shift criterion [14] and the consolidation of NREM stages 3 and 4 into stage N3 [15]. For each participant, we determined standard clinical sleep parameters and the total sleep stage shifts (shifts from deeper to lighter sleep with increasing depth of sleep ordered as wake, N1, N2, N3, and REM) and sleep stage shift index (sleep stage shifts/total sleep time).

Sleep disordered breathing events were defined as: apnea, a decrease of inspiratory airflow to below 20% of waking levels lasting at least 10 s and hypopnea, a decrease of inspiratory airflow to below 50% of waking levels associated with an arousal from sleep. Arousals preceded by three or more breaths with an inspiratory airflow plateau that was above 50% of waking airflow were quantified as respiratory event-related arousals (RERAs) [16].

Following the initial polysomnogram, all participants underwent a second polysomnogram to determine the minimum nasal CPAP needed to eliminate inspiratory aiflow limitation (IFL) during supine sleep. Sleep was monitored using the same methods as for the initial polysomnogram. Each participant wore a nasal mask in series with a heated pneumotachograph to measure airflow. Inspiratory effort was measured by supraglottic pressure (Psg) using a catheter inserted trans-nasally (MPC-500, Millar Instruments, Houston, TX). Once the participant was asleep and the presence of IFL was confirmed (a plateau of inspiratory airflow despite the continued decrease of Psg), CPAP was increased in 1 cmH_2O increments until the resolution of IFL (airflow increased and Psg decreased in parallel; therapeutic CPAP; Ptherapeutic). After approximately 3 h on Ptherapeutic (which usually included REM sleep), CPAP was then lowered to the minimum level (4 cmH_2O) and left for the duration of the study to facilitate masking of the participants during the treatment trial.

Pre-treatment evaluation of patient-reported outcomes

The patient-reported outcomes of this study were assessed for 1 week before treatment using the following visual analogue scales (VAS) and questionnaires:

- Pain—increasing levels were rated 0–10 by VAS daily.
- Fatigue—increasing impact was rated 1–7 using the Fatigue Severity Scale [17] (FSS) on days 1 and 7 (averaged).
- Cognition—increasing difficulty with memory, ability to think, and ability to concentrate was rated 0–10 daily by VAS.

- Sleep quality—increasing sleep disturbance was rated 0–21 with the Pittsburgh Sleep Quality Index [18] (PSQI) on days 1 and 7 (averaged).
- General health—(mental and physical components) was assessed with the Short Form 36 [19] (SF-36) on day 1.

Each day, after completing the questionnaires, the participants mailed the evaluation back to the research team with the postmark serving to confirm completion the previous day.

Treatment trial

Participants were randomized to receive nasal CPAP at Ptherapeutic or sham nasal CPAP (pressure below 1 cmH$_2$O) using a previously utilized sham circuit [20]. Participants were asked to sleep with their breathing machines (the terms CPAP or pressure were never used) for at least 5 h per night for 3 weeks. We chose a 3-week trial duration based upon the duration of our nasal CPAP trial in fibromyalgia [10]. Compliance was assessed by software in each generator (the Respironics Aria—LX CPAP System). Contact with participants during the treatment trial occurred only when initiated by the participant.

Post-treatment evaluation of outcomes

During the third week of treatment, subjects repeated the battery of questionnaires completed during the pre-treatment week. On completing the trial, all participants underwent a third polysomnogram using their assigned treatment monitored in the same fashion as the first polysomnogram. Analysis of the third polysomnogram was performed by an investigator, masked to the assigned treatment and to the respiratory channels during sleep staging (the airflow channel would be expected to show IFL among sham participants). Only after completing the sleep staging did the investigator score respiration.

Statistics

This was a pilot study to investigate hypotheses about the nature of the symptoms of GWI and a possible modality of treatment. The sample size of 9 per treatment group was based on our experience with fibromyalgia patients [10] for whom nasal CPAP produced a large treatment effect. All statistical analyses were conducted using SAS Version 8.2 for Windows.

All demographic and baseline symptom and sleep parameters were compared between treatment groups with an unpaired Student's t test. The effects of the treatments on changes in symptom severity and sleep parameters were compared with ANOVA including a treatment effect and baseline covariate. The interactions of treatment effect and baseline covariate were dropped because they were generally not statistically significant. The effect sizes for the symptom severities were computed as the standardized mean difference (SMD) utilizing the least squares mean differences (from the ANOVA) in post-treatment symptom levels divided by the root mean squared error (from the ANOVA). We interpreted the effect sizes using the widely accepted guidelines of Cohen for SMD, defining 0.2 as small, 0.5 as medium, and 0.8 as large [21].

Pearson correlations were computed to clarify the relationships between changes in symptom severities and changes in sleep parameters. Additionally, partial correlations were computed, i.e., the correlation between change in severity of a symptom and change in a sleep parameter after removing from both (via linear regression) the correlation with a second sleep parameter. Partial correlation can be thought of as the extent to which two variables would be correlated in a population where all subjects were at the mean of a third variable, i.e., where the effect of the third variable on each of the two variables had been removed. The entire correlation analysis was conducted without reference to the treatment effect.

Results

Following randomization of our participants but before treatment, one participant assigned to active treatment enrolled in a PTSD treatment program and was excluded (Table 1, #17) leaving eight participants assigned to active and nine assigned to sham treatment. Table 2 summarizes demographic characteristics, baseline symptom levels, and sleep parameters measured in the first polysomnogram. Symptom levels were generally slightly lower in the sham group, and the quality of sleep (assessed by sleep parameters) was generally slightly poorer, but there were no significant differences between the groups. The perception of overall physical health as reflected by the physical component of the SF-36 among participants randomized to active CPAP, however, was significantly lower than that of those randomized to sham (Table 2). Compliance with assigned treatment was comparable between the active and sham groups (265.1±90.2 min/night vs. 266.6±100.8 min/night, respectively; $p=0.98$).

Efficacy is summarized in Table 3 and Fig. 1. Participants were 100% compliant with mailing back their questionnaires. The participants receiving nasal CPAP experienced substantial improvements in their symptoms (reductions of 34% in pain and 38% in fatigue, improvements of 33% in cognitive function, 41% in sleep quality, 34% in physical health, and 16% in mental health).

Table 1 Anthropometric and sleep data of the GWI participants

Participant	Age	BMI	Snoring	AHI	RERA/h	Ptherapeutic	Assignment
1	46	33	+	9	6	7	Sham
2	43	24	−	10	25	7	Sham
3	41	30	−	10	37	11	Sham
4	42	34	+	60	11	10	Sham
5	44	34	+	45	2	8	Sham
6	45	33	−	2	9	7	Sham
7	47	32	+	8	7	8	Sham
8	41	30	−	5	11	9	Sham
9	37	26	−	9	15	9	Sham
10	39	33	+	7	19	11	Active
11	37	34	+	47	27	13	Active
12	50	35	+	91	14	12	Active
13	41	36	+	10	29	10	Active
14	43	29	−	10	13	7	Active
15	43	30	−	5	23	9	Active
16	40	32	+	9	4	8	Active
17	45	27	?	6	8	9	Active
18	38	33	−	3	7	9	Active
Mean+SD	42±4	31±3		19±25	15±10	9±2	

BMI body mass index in kilogram per square meter

Snoring +; loud snoring reported at least once or twice a week (PSQI)

Snoring −; loud snoring reported less than once a week (PSQI)

Snoring ?; the patient withdrew prior to completing the PSQI

Ptherapeutic; the level of nasal CPAP that eliminates inspiratory airflow limitation

Assignment; the treatment group to which the participant was randomized

Participants receiving sham experienced no change or slight worsening of symptoms. Differences between the groups were all statistically significant.

The effect of treatment upon the participants' sleep parameters is demonstrated in Table 4. There were significantly greater decreases in AHI, sleep stage shifts, and sleep stage shift index on nasal CPAP vs. sham, reflecting decreased SDB and increased sleep consolidation (Fig. 2).

The change in sleep stage shifts among all participants from pre-treatment to post-treatment polysomnogram was highly and significantly correlated with the change in most measures of symptom levels (Table 3). In fact, after adjusting for differences between participants in change in sleep stage shifts, only one partial correlation remained statistically significant, change in stage 2 as a percent of total sleep time vs. change in fatigue ($p=0.001$). In contrast, after adjusting for differences between participants in changes of any other sleep parameter, the partial correlation between change in sleep stage shifts and changes in most symptoms remained statistically significant.

Discussion

We conducted this pilot study to test our hypothesis that SDB plays a role in the symptoms of GWI. In a 3-week, sham-controlled trial of nasal CPAP, veterans with GWI receiving nasal CPAP experienced a marked symptomatic improvement while those receiving sham did not improve.

Among all participants, the change in symptoms correlated with the change in sleep stage shifts from the initial polysomnogram to the polysomnogram after treatment. This pilot study provides preliminary evidence that SDB plays a role in the symptoms of GWI.

While SDB may play a role in the symptoms of GWI, how can one know that the combination of SDB, fatigue, and cognitive dysfunction in this middle-aged, overweight, veteran population is not simply OSA/H? Our participants, many of them patients at the DVA Medical Center-Northport, have been diagnosed with GWI since their service in the Persian Gulf. At the time their symptoms began, they were approximately 15 years younger and closer to their active duty weight (presently, 14 of the 18 GWI participants have values of BMI≥30 kg/m^2 which is uncharacteristic of active duty personnel). Furthermore, 8 of 17 participants who completed the PSQI did not report snoring (Table 1), a finding that is not typical of OSA/H patients. Finally, while fatigue and cognitive dysfunction are symptoms of OSA/H [22] and may be expected to improve with nasal CPAP, pain is not a recognized symptom of OSA/H, and the improvement of pain by nasal CPAP is a characteristic of fibromyalgia [10] and not OSA/H. While the characteristics of our participants are not typical of OSA/H patients, how pharyngeal collapse during sleep relates to the symptoms of GWI remains a question.

Compared to the treatments available for FSS, how significant was the therapeutic effect of nasal CPAP in this study? In the one multicenter treatment trial comparing

Table 2 Demography and baseline

	Therapeutic CPAP (N=8) mean±SD	Sham CPAP (N=9) mean±SD	p value
Demography:			
Age	41.4±4.1	42.9±3.1	0.40
BMI	32.8±2.4	30.7±3.5	0.19
Baseline symptoms:			
Pain VAS	5.9±2.4	4.4±3.1	0.28
FSS	5.3±1.5	5.1±1.1	0.74
Cognitive VAS	6.1±2.3	5.2±3.1	0.50
PSQI	13.8±5.0	10.2±3.5	0.11
SF-36 physical	32.7±6.9	45.5±12.0	0.02
SF-36 mental	32.4±10.6	39.4±9.1	0.16
Baseline sleep parameters:			
TST (min)	374.8±57.2	308.0±117.1	0.16
Sleep efficiency (%)	85.6±9.5	69.4±23.9	0.09
Sleep latency (min)	8.7±11.6	18.3±23.3	0.31
REM latency (min)	101.3±39.5	110.7±84.6	0.78
WASO (min)	53.9±44.9	112.0±103.7	0.16
N1 (% TST)	36.1±24.6	31.7±11.6	0.64
N2 (% TST)	44.9±20.2	46.2±13.5	0.88
N3 (% TST)	7.5±5.6	9.9±10.4	0.57
Stage REM (% TST)	11.6±3.7	12.0±7.4	0.88
AHI (events/h)	22.7±31.0	17.5±20.4	0.68
RERA index (events/h)	17.0±9.1	13.7±10.9	0.51
Sleep stage shifts (events)	43.9±15.8	36.0±7.0	0.20
Sleep stage shift index (events/h)	6.6±2.6	8.2±4.3	0.36

VAS visual analogue scale, *FSS* fatigue severity scale, *PSQI* Pittsburgh sleep quality index; *SF-36* Short Form-36 Health Survey with physical and mental components, *TST* total sleep time, *WASO* wake after sleep onset (total wake time from sleep onset to final awakening of the night), *AHI* apnea hypopnea index, *RERA* respiratory event-related arousal (an arousal associated with inspiratory flows above the threshold for hypopnea), *Sleep stage shift* a shift from a deeper to a lighter sleep stage with increasing depth of sleep ordered: wake, N1, N2, N3, and REM

Table 3 Post-treatment symptoms

Questionnaire	Therapeutic CPAP (N=8) mean±SD	Sham CPAP (N=9) mean±SD	Effect size	p value	Correlation with SSS (p value)
Pain VAS post	3.9±2.3	4.8±3.3	2.14	0.0008	0.51 (0.037)
Change from baseline	−2.0±0.9	0.4±1.2			
FSS post	3.3±1.3	5.3±1.1	2.55	0.0002	0.71 (0.002)
Change from baseline	−2.1±0.9	0.2±1.0			
Cognitive VAS post	4.1±2.6	5.6±3.4	1.67	0.004	0.64 (0.006)
Change from baseline	−2.0±1.7	0.4±1.0			
PSQI post	8.2±1.8	10.8±3.2	2.67	0.0003	0.59 (0.016)
Change from baseline	−5.7±3.5	0.6±1.7			
SF-36 physical post	43.8±4.0	41.0±10.0	2.79	0.0003	−0.41 (0.104)
Change from baseline	11.1±3.9	−4.5±5.9			
SF-36 mental post	37.6±7.8	33.9±9.9	1.29	0.03	−0.58 (0.015)
Change from baseline	5.3±7.3	−5.5±7.0			

CPAP continuous positive airway pressure, *VAS* visual analogue scale, FSS fatigue severity scale; *PSQI* Pittsburgh sleep quality index, *SF-36* the Short Form-36 Health Survey with physical and mental components, *SSS* sleep stage shifts
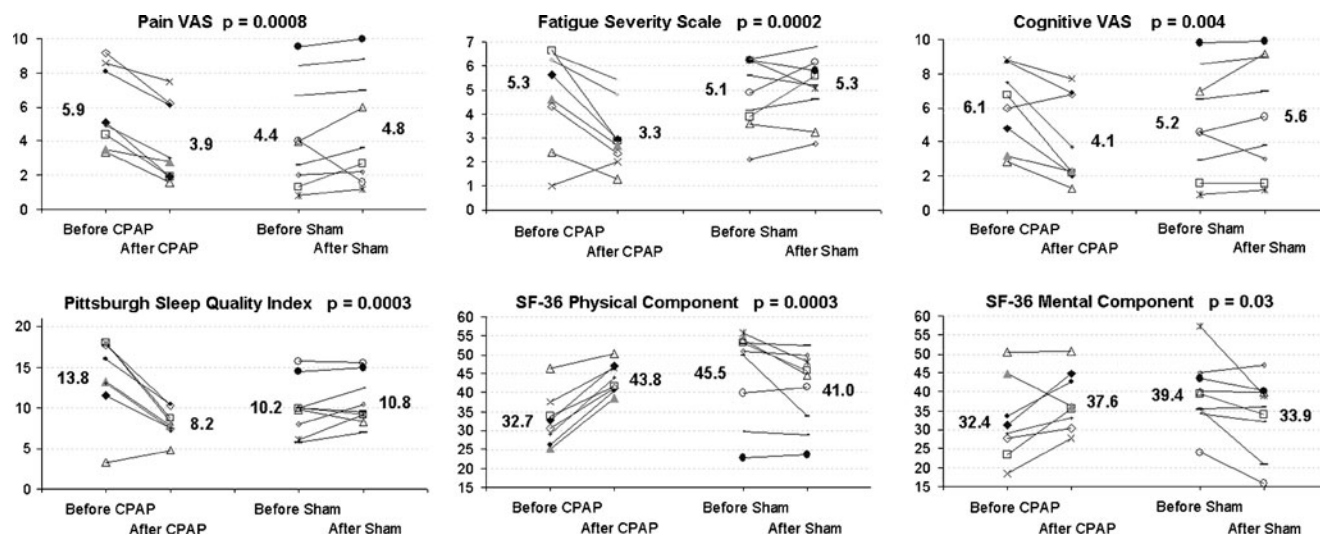
Fig. 1 This figure demonstrates the changes in patient-reported outcomes following 3 weeks of treatment with either nasal CPAP (eight participants) or sham nasal CPAP (nine participants). The scales/questionnaires used to assess the outcomes are described in the "Methods" section

Table 4 Post-treatment sleep parameters

Sleep parameter	Therapeutic CPAP (N=8) mean±SD	Sham CPAP (N=9) mean±SD	p value
TST (min)	358.1±39.7	329.7±71.5	0.97
Change from baseline	−16.7±56.4	21.7±81.0	
Sleep efficiency (%)	88.0±5.9	76.4±15.5	0.36
Change from baseline	2.5±11.7	7.0±15.9	
Sleep latency (min)	11.4±9.3	25.3±18.1	0.12
Change from baseline	2.7±18.1	7.0±24.6	
REM latency (min)	126.5±91.7	199.4±88.8	0.11
Change from baseline	25.2±92.4	88.8±144.3	
WASO (min)	43.0±27.8	75.5±57.5	0.57
Change from baseline	−10.9±55.7	−36.6±66.9	
Stage 1 (% TST)	19.3±10.1	26.8±12.4	0.16
Change from baseline	−16.8±23.4	−4.9±13.9	
Stage 2 (% TST)	54.7±7.9	56.6±12.4	0.74
Change from baseline	9.7±16.8	10.4±19.9	
Slow wave sleep (% TST)	9.2±8.6	5.4±6.9	0.27
Change from baseline	1.8±8.9	−4.4±10.5	
Stage REM (%TST)	16.8±7.4	11.1±6.1	0.11
Change from baseline	5.3±9.7	−0.9±8.4	
AHI (events/h)	0.5±0.7	11.1±7.0	0.0009
Change from baseline	−22.2±30.9	−6.4±20.5	
RERA index (events/h)	4.5±2.5	16.9±14.3	0.055
Change from baseline	−11.9±8.8	3.2±20.8	
Sleep stage shifts (events)	26.3±5.7	39.1±9.4	0.002
Change from baseline	−17.6±12.9	3.1±9.7	
Sleep stg shift idx (events/h)	4.4±0.8	7.5±3.0	0.026
Change from baseline	−2.2±2.4	−0.7±4.4	

CPAP continuous positive airway pressure, *WASO* wake after sleep onset (wake time between sleep onset and the final awakening of the night), *TST* total sleep time, *AHI* apnea hypopnea index, *RERA* respiratory event-related arousal (an arousal associated with inspiratory flows above the threshold for hypopnea), *Sleep stage shift* a shift from a deeper to a lighter sleep stage with increasing depth of sleep ordered: wake, stage 1, stage 2, slow wave sleep, and REM, *Sleep Stg Shift Idx* sleep stage shift index, *HR* heart rate, *bpm* beats per minute
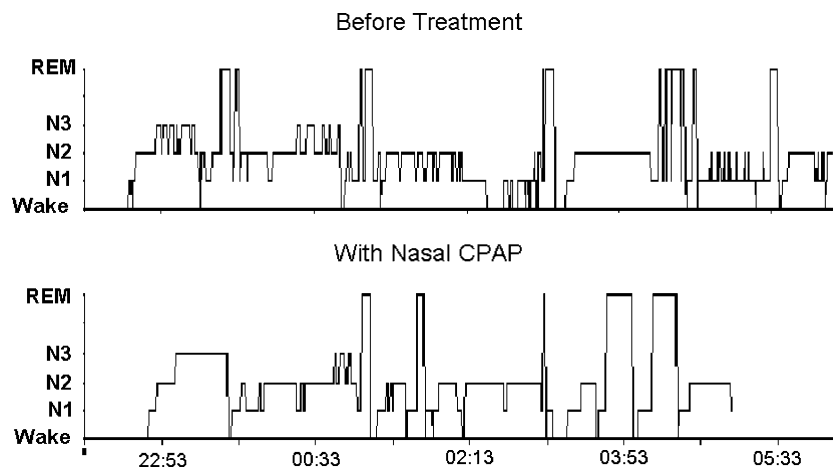
Fig. 2 This figure contains two hypnograms (plots of sleep stages against time) that demonstrate increasing sleep consolidation (decreasing sleep stage shifts) for a participant receiving therapeutic nasal CPAP (age 43 years; BMI 30.0 kg/m^2; baseline AHI 5/h; baseline RERA index 23/h).

Abbreviations: *N1* non-rapid eye movement stage 1 sleep; *N2* non-rapid eye movement stage 2 sleep; *N3* slow wave sleep; *REM* rapid eye movement sleep

CBT to exercise for the symptoms of GWI, the improvements in fatigue, pain, and cognitive function at 3 months for participants receiving the combined treatments did not exceed 5–10% [23]. Further, in a recent large review of pharmacologic treatment trials among fibromyalgia patients, the effect size of treatment upon a variety of symptom outcomes (including pain, fatigue, and sleep) rarely exceeded 0.9 (a large effect size) [24]. In contrast to the CBT/exercise treatment trial [23], the improvement in fatigue, pain, cognitive dysfunction, and sleep disturbance among our eight veterans receiving Ptherapeutic ranged from 33% to 41%, and the smallest effect size we observed for any outcome was 1.29 (Table 3; to our knowledge, this is the first study to measure the effect size of nasal CPAP as treatment for a functional somatic syndrome). Therefore, relative to results of previous studies of CBT, exercise, and pharmacologic treatment of the FSS, the clinical effect of nasal CPAP upon the symptoms of GWI in this 3-week pilot study was profound.

The proportionate change that we observed in the total of sleep stage shifts and the severity of GWI symptoms with treatment may provide a clue concerning how nasal CPAP affects the symptoms of GWI. Although previous investigators have demonstrated increased sleep stage shifts [25–28] in the polysomnograms of patients with fibromyalgia, irritable bowel syndrome, restless legs syndrome, and primary insomnia compared to those of healthy controls, the significance of this finding is uncertain. Voss [29] has proposed that increased sleep stage shifts may be a mechanism to adapt the individual's sleep to the stress of a hostile environment. By continually shifting from deeper sleep stages characterized by decreased arousability to lighter sleep stages, the individual is protected from environmental threats (intruders) or internal threats (pharyngeal collapse) [29]. Building upon Voss's hypothesis, we propose that pharyngeal collapse during sleep may function as a stressor increasing sleep stage shifts in veterans with GWI. Nasal CPAP, by splinting the pharyngeal airway during sleep, removes this stressor and decreases the number of sleep stage shifts (Fig. 2). By extension, the correlation of the change in sleep stage shifts with the change in symptoms of GWI suggests that the change in symptoms also results from the change of the individual's response to the stress of pharyngeal collapse during sleep. Thus, our findings lead us to the hypothesis that nasal CPAP relieves the symptoms of GWI by overcoming an important stressor among veterans with GWI, pharyngeal collapse during sleep.

From the previous discussion, it appears that the symptoms of GWI are related to an increased number of sleep stage shifts. However, in our comparison of inspiratory airflow dynamics during sleep between veterans with GWI and healthy veterans of the first Gulf War, participants with symptoms of GWI did not have an increased total of sleep stage shifts relative to controls with no symptoms [11]. This finding may have been related to increased sleep stage shifts among our controls spending their first night in a sleep laboratory (first night effect) [30]. When we developed the protocol for our study of inspiratory airflow dynamics during sleep [11], we utilized a single night of polysomnography because comparing sleep parameters between our GWI participants and healthy controls was not a primary outcome. Studies that have demonstrated increased sleep stage shifts among patients with functional somatic syndromes have monitored sleep for 2–3 nights [25–28], and such a design may have demonstrated increased sleep stage shifts among our veterans with GWI.

One feature of our study design must be noted. Treatment assignment was single-masked. Although we did not utilize a double-masked design, all primary outcomes were participant-reported, and contact between investigators and participants during treatment was minimal. Therefore, we do not believe that our design introduced a significant investigative bias.

Our study makes an important contribution to the literature on GWI. Given our findings and the absence of effective treatment for veterans with GWI [4], physicians managing these patients might choose to refer them for polysomnography and to treat even mild pharyngeal collapse during sleep. Further, our findings in this initial effort suggest that the relationship between SDB and the symptoms of GWI warrants further study.

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