SHORT COMMUNICATION

Obstructive sleep apnea in combat-related posttraumatic stress disorder: a controlled polysomnography study

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Background: Obstructive sleep apnea (OSA) may be highly prevalent in posttraumatic stress disorder (PTSD) and may exacerbate PTSD complaints.

Objective: Our objective was to determine whether the prevalence of OSA was high in a sample of Dutch veterans with PTSD as compared to age- and trauma-matched controls, and whether OSA was associated with more severe PTSD complaints.

Methods: We determined the apnea hypopnea indices (AHI) with polysomnographic registrations in 20 veterans with PTSD, 24 veterans without PTSD, and 17 healthy controls. PTSD severity and nightmare complaints were assessed with the Clinician-Administered PTSD Scale (CAPS).

Results: The prevalence of an AHI >10 was 29% in PTSD, 21% in trauma controls, and 29% in healthy controls ($\chi^2 = 0.60$, $df = 2$, $p =$ n.s.). The mean CAPS score in patients with OSA ($n = 6$) was significantly higher than in patients without OSA ($p < 0.05$), while nightmare severity was similar in PTSD patients with OSA as compared to PTSD patients without OSA ($p =$ n.s.). Furthermore, there was a significant correlation between AHI and CAPS score in PTSD patients ($r = 0.46$, $p < 0.05$, $df = 14$).

Conclusions: Our results indicate that PTSD is not necessarily associated with a higher prevalence of OSA. However, PTSD severity was related to OSA, which may possibly mean that comorbid OSA leads to an increase in PTSD complaints. However, future research should indicate whether OSA exerts a negative influence on PTSD, and treatment of OSA alleviates PTSD symptoms.

Keywords: *PTSD*; *sleep*; *OSA*; *polysomnography*; *apnea*

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Received: 10 September 2011; Revised: 13 November 2011; Accepted: 21 November 2011; Published: 26 December 2011

Traumatic memories are frequently experienced during nightmares in patients suffering from posttraumatic stress disorder (PTSD). PTSD patients are vigilant at night and complain of nightmares, frequent awakenings, and non-restorative sleep (Neylan et al., 1998). Despite often severe subjective complaints, objective sleep measures, such as total sleep time and the amount of rapid eye movement (REM) sleep, are generally unaffected in PTSD. More subtle changes in polysomnographic (PSG) recordings, such as increased stage 1 sleep, decreased REM density, and a higher number of awakenings, have been reported (Kobayashi, Boarts, & Delahanty, 2007; Van Liempt, Vermetten, Lentjes, Arends, & Westenberg, 2011); however, it is unclear how these alterations relate to insomnia and nightmares. Mechanisms responsible for the nightly complaints remain to be further explored (Spoormaker & Montgomery, 2008).

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Citation: European Journal of Psychotraumatology 2011, **2:** 8451 - DOI: 10.3402/ejpt.v2i0.8451
(page number not for citation purpose)

Earlier research has shown that posttraumatic nightmares and other posttraumatic complaints may be related to obstructive sleep apnea (OSA) (Krakow et al., 2000, 2001a; Yesavage et al., 2010). When OSA is highly prevalent in PTSD and related to PTSD symptoms, reducing respiratory events may be a putative focus for the treatment of posttraumatic complaints (Hurwitz & Khawaja, 2010). Thus far, only uncontrolled studies reported a high prevalence of OSA in PTSD.

Objective

To examine the putative relationship between PTSD and OSA, we determined the apnea hypopnea indices (AHI), body mass index (BMI), PTSD severity, and nightmare complaints in veterans with PTSD, veterans without PTSD, and healthy controls.

Method

Participants

Twenty-three male veterans with PTSD were recruited through the outpatient clinic of the Military Mental Healthcare, Utrecht, the Netherlands. PTSD patients with habitual benzodiazepine usage ($n = 1$) and substance abuse were excluded ($n = 2$). Patients who used benzodiazepines, alcohol, or drugs weekly or monthly were instructed to refrain from these substances on the day of the sleep recordings. Five PTSD patients used a selective serotonin reuptake inhibitor.

Twenty-four trauma controls (TCs; veterans without PTSD) and 17 healthy controls (HCs; civilians or service members who were naïve for deployment) were recruited through advertisements in veteran-related magazines and newspapers. TCs were matched for age, year, and region of deployment with the PTSD group and were excluded when they had a Clinician-Administered PTSD Scale (CAPS) score of 18 or higher. HCs were matched for age with the PTSD group and were excluded when reporting significant psychotrauma in the past according to the CAPS.

All control subjects were medically healthy males without a history of psychiatric disorders and without sleep complaints. Written consent was obtained from all participants, after a complete written and verbal description of the study. The study was approved by the Institutional Review Board of the University Medical Centre of Utrecht, the Netherlands.

Procedures

All veterans were screened for psychiatric illness using the structured clinical interview for DSM IV axis I disorders [severe combined immunodeficiency (SCID)] (Spitzer, Williams, Gibbon, & First, 1992). The diagnosis of PTSD (assessed with the SCID) was confirmed by the CAPS (Blake et al., 1995) and after consensus by two clinicians (SvL., E.V.). Only patients with a CAPS score above 50 were included. Trauma controls were included if they met the A1 criterion for PTSD (the person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to physical integrity of self or others) but had a CAPS score below 18 and did not meet DSM IV criteria for PTSD or any other life time axis I disorder. Subjects were screened for medical conditions by history taking and physical examination. As a high BMI is associated with AHI, weight and length were determined.

PSG recordings were obtained during one night at Kempenhaeghe, the Netherlands, with Brainlab Real Time©, including electrooculography for vertical and horizontal eye movements, electromyography (chin, left and right m. tibialis anterior), electrocardiography, and electroencephalography (F0-C0, F3-C3, P3-O1, C4-A1, O2-A2), airflow at the nose (nasal pressure), movement of the thorax and abdomen by inductance plethysmography, and arterial oxygen saturation at the index finger.

Data analyses

Sleep data were analyzed according to criteria of Rechtschaffen and Kales (1968) by an experienced sleep technician who was blinded to group identity. Apneas and hypopneas were scored when an airflow reduction of 50%–90% (hypopnea) or >90% (apnea) was detected for at least 10 seconds, except when such airflow reductions occurred after arousals or during wake.

An AHI >15 per hour is the cutoff for obstructive sleep apnea syndrome (Epstein et al., 2009). An AHI >10 is also considered clinically relevant and used as an exclusion criterion in sleep studies (Engdahl, Eberly, Hurwitz, Mahowald, & Blake, 2000; Mellman, Nolan, Hebding, Kulick-Bell, & Dominguez, 1997). We compared both indices between groups. In comparisons of mean CAPS scores between PTSD patients with and without OSA, we used the cutoff of AHI >10.

Statistical analyses

Differences between the groups were tested in SPSS 17.0. An ANOVA was used to test group differences when variables were normally distributed. In other cases, a nonparametric Mann–Whitney U test was used. With a χ^2 test, we examined whether OSA was more prevalent in PTSD compared with controls. Correlations were analyzed with a Pearson's test and were controlled for age and BMI. Differences were considered significant when p-values were smaller than 0.05.

Citation: European Journal of Psychotraumatology 2011, 2: 8451 - DOI: 10.3402/ejpt.v2i0.8451

Table 1. Demographic characteristics

	PTSD ($n=20$)	TC ($n=24$)	HC ($n=17$)	Comparison
Age (*M*, SD)	40.75 (8.45)	37.71 (6.91)	35.06 (8.26)	$df=60$, $F=2.5$, $p=0.095$
Alcohol intake (*M*, SD)	7.50 (8.00)	5.33 (7.07)	7.29 (5.28)	$df=60$, $F=0.96$, $p=0.391$
BMI (*M*, SD)	27.86 (4.86)	24.64 (2.90)	23.65 (2.42)	$df=53$, $F=6.7$, $p=0.003$*
CAPS (*M*, SD)	67.50 (11.02)	4.00 (5.11)	0 (0)	$df=60$, $F=571.0$, $p<0.001$
Number of A1 trauma (*M*, SD)	8.92 (2.18)	7.24 (2.91)	1.93 (1.49)	$df=60$, $F=36.3$, $p<0.001$**

Notes: BMI, body mass index; HC, healthy controls; *M*, mean; PTSD, posttraumatic stress disorder; SD, standard deviation; TC, trauma controls.
*PTSD >TC, ($p=0.036$); PTSD >HC, ($p=0.006$); TC =HC, ($p=1.0$).
**PTSD =TC, ($p=0.16$); HC <PTSD, ($p<0.001$); HC <TC, ($p<0.001$).

Results

Demographic data

In Table 1, demographic variables are shown. PTSD patients ($n=20$), TCs ($n=24$), and HCs ($n=17$) did not differ in age or alcohol intake per week. The mean CAPS score was 67.50 (SD =11.01) in PTSD patients and 4.00 (SD =5.12) in TCs. Of two PTSD patients, four TCs and one HCs BMI data were missing. PTSD patients ($n=18$) had a higher BMI than both TCs ($n=20$) and HCs ($n=16$) (respectively, 27.86 (SD =4.86); 24.64 (SD =2.90) and 23.65 (SD =2.42), (ANOVA $F(2,51)=6.66$, $p<0.01$).

Prevalence of OSA

The AHI was higher than 15 in 10% of the PTSD patients, 13% of the TCs, and 12% of the HCs ($\chi^2=0.07$, $df=2$, $p=$ n.s.). Twenty-nine percent of the PTSD patients, 21% of TCs, and 29% of HCs had an AHI >10 per hour ($\chi^2=0.60$, $df=2$, $p=$n.s.). The mean AHI was 6.79 (SD =5.58) in PTSD patients, 7.18 (SD =6.50) in TCs, and 5.68 (5.91) in HCs (ANOVA, $F(2,58)=0.32$, $p=$n.s.).

OSA and PTSD severity

The patients with an AHI >10 ($n=6$) exhibited significantly higher CAPS scores ($M=77$, SD =9) than patients without OSA ($M=63$, SD =9) ($n=14$) (Mann–Whitney *U*test: $z=-2.36$, $p<0.05$).

With a partial correlation, controlling for age and BMI, a significant relation between the CAPS and AHI was found in PTSD patients (Pearson, $r=0.46$, $p<0.05$, $df=14$). (see Fig. 1). The three subscales (B, C, D) of the CAPS were not significantly related to the AHI in PTSD. However, the correlation between the D-subscale (hyperarousal) and AHI was at trend-level significance in PTSD patients (Pearson, $r=0.36$, $p=0.08$, $df=14$).

OSA and nightmares

The mean nightmare score was 3.33 (SD =2.56) in patients with OSA (AHI >10, $n=6$) and 3.21 (SD =2.29) in patients without OSA ($n=14$) (ANOVA, $F(1, 19)=0.1$, $p=$n.s.). Similarly, there was no relationship between B2 score of the CAPS and AHI, when corrected for BMI and age (Pearson's test $r=-0.076$, $p=$n.s, $df=14$).

Discussion

Our study shows that the occurrence of OSA in PTSD patients was 10% and was not increased compared with TCs and HCs, despite a higher BMI of PTSD patients. Furthermore, we found a relationship between PTSD severity and the AHI, whereas nightmare frequency was not related to the occurrence of apneas. The observations from our study contrast with previous reports. Several studies reported high indices of AHI in 69%–91% of the PTSD patients (Krakow et al., 2001b, 2002, 2004; Yesavage et al., 2010). In these studies, screening instruments for detecting OSA may have been more sensitive than in our study, especially because some studies defined a cutoff of five events per hour. As none of the previous studies included a control group, it cannot be concluded that the incidence of OSA is elevated in PTSD. Another explanation for the high incidence of OSA in some

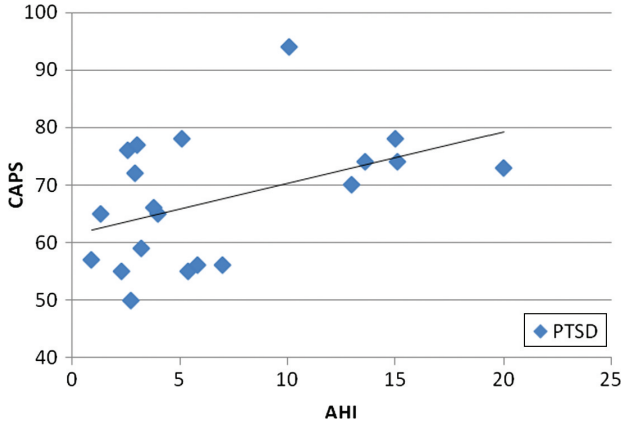

Fig. 1. Relationship between apnea hypopnea index and PTSD severity. Notes: AHI, apnea hypopnea index; CAPS, clinician-administered PTSD scale; PTSD, posttraumatic stress disorder.

previous studies is that the usage of benzodiazepines was not discontinued before sleep recordings, which increases the occurrence of OSA (Dolly & Block, 1982). In our study, participants with regular benzodiazepine usage were excluded, and participants with habitual benzodiazepine usage refrained from sleep medication in the sleep laboratory. Lastly, our study group consisted of middle-aged veterans, whereas other studies included predominantly female PTSD patients. The incidence of OSA may be different in other populations with PTSD. Our study underlines the importance of controlled studies to determine whether OSA is more prevalent in PTSD than in matched controls.

Although controlled studies have not been published on this subject before, our results are in concordance with two sleep studies in PTSD patients, which excluded an equal amount of patients and controls due to OSA (Breslau et al., 2004; Mellman et al., 1997). In contrast, Engdahl et al. (2000) reported that in a sample of elderly war veterans, more PTSD patients were excluded due to OSA than controls.

In our study, PTSD severity was related to OSA. Furthermore, PTSD patients with an AHI >10 exhibited significantly higher CAPS scores compared with PTSD patients without OSA. This may be due to decreased concentration, depression, and irritability that are common complaints in OSA (Saunamäki & Jehkonen, 2007). Also, previous studies have suggested a relationship between PTSD complaints and the occurrence of OSA. In the study of Engdahl et al. (2000), four PTSD patients with OSA improved on overall well-being after treatment with continuous positive airway pressure (CPAP). The relationship between remitted OSA and improvement of PTSD severity was also found in an uncontrolled retrospective study on CPAP treatment in PTSD patients (Krakow et al., 2000).

Our results did not find a relationship between nightmare frequency and OSA. This contrasts with a study in which breathing interruptions, as witnessed by bed partners, were related to nightmare frequency in PTSD patients (De Groen et al., 1993). Reports from the bed partner are less reliable for detecting OSA than polysomnography that was used in the current study. Possibly, bed partners of patients with nightmares are more alert during the night and therefore better aware of breathing interruptions. Therefore, breathing interruptions may be more frequently reported in patients with nightmares in the study of De Groen et al. (1993), in the absence of a relationship between OSA and nightmares.

A limitation of our study is the small sample size. The incidence of OSA in PTSD patients was 10% in our study; however, this cannot be generalized. Furthermore, the power for detecting correlations between OSA and symptom clusters of PTSD may have been too low. Our study consisted of a homogeneous group of middle-aged male veterans. The advantage was that the control groups were well matched. However, the results may not be extrapolated to other PTSD populations.

Conclusions

In summary, our results indicate that PTSD is not necessarily associated with the occurrence of OSA, as some uncontrolled studies suggested. Furthermore, OSA was not related to sleep complaints, while patients with comorbid OSA exhibited higher CAPS scores. Larger controlled studies in different populations need to be performed to provide more reliable estimations of the incidence of OSA in PTSD. As comorbid OSA in PTSD patients may possibly lead to an increase in PTSD symptoms, the influence of OSA, and treatment of OSA, should be further explored, especially in those with refractory complaints to conventional therapeutic strategies.

Acknowledgements

This work was supported by the Dutch Ministry of Defense. We would like to thank Jos van Gorp for the analyses of the sleep recordings.

Conflict of interest and funding

There is no conflict of interest in the present study for any of the authors.

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