Polysomnographic Sleep Is Not Clinically Impaired in Vietnam Combat Veterans with Chronic Posttraumatic Stress Disorder

Thomas D. Hurwitz, Mark W. Mahowald, Michael Kuskowski, and Brian E. Engdahl

Background: *Because sleep is typically disturbed in posttraumatic stress disorder (PTSD), this study was undertaken to evaluate a group of Vietnam combat veterans with the disorder using clinical polysomnographic techniques.*

Methods: *Eighteen Vietnam combat veterans with PTSD and 10 healthy non–combat-exposed Vietnam era veterans participated in 2 nights of polysomnographic study and a multiple sleep latency test.*

Results: *No significant differences between subjects and controls were noted except for greater sleep onset latency to stage 2 ($p < .03$), and lower arousals/hour from stages 3 & 4 ($p < .04$) on night 2, and lower subjectively estimated total sleep time on night 1 ($p < .005$) in the case of PTSD subjects. Otherwise, results from the second night served to replicate those from the first, and no significant differences appeared on 2 successive nights for any polysomnographic variable. No daytime hypersomnolence was detected.*

Conclusions: *Polysomnographically recorded sleep was notably better than expected in the presence of clinically significant PTSD with typical histories of disrupted sleep. In these subjects, there is no clinically significant sleep disorder or typical pattern of sleep disturbance detectable by standard polysomnography.* Biol Psychiatry 1998; 44:1066–1073

Key Words: Posttraumatic stress disorder, sleep, polysomnography, Vietnam combat veterans

Introduction

Sleep disturbance has been labeled as the hallmark of posttraumatic stress disorder (PTSD) (Ross et al 1989), and the prototypic symptom has been the nightmare (van der Kolk et al 1984). Clinicians typically find patients complaining of sleep onset and maintenance difficulties as well as poorly restorative sleep. Nightmares are generally not experienced by patients sleeping in laboratory situations, and this may be attributed to the safe, "guarded," hospital environment (Woodward 1995). Nonetheless, patients typically complain of severe and chronic disturbances of sleep, often associating these symptoms with daytime sequellae such as fatigue, poor concentration, etc. Some reports describe sleep disorders such as obstructive sleep apnea (OSA) (Dagan and Lavie 1991), periodic limb movements of sleep (PLMS) (Brown and Boudewyns 1996), and, less often, REM-sleep behavior disorder (Hefez et al 1987; Inman et al 1990) in patients with PTSD. It would seem reasonable to expect to observe a number of these disorders in a population of men in their fifth decade of life and beyond.

Of the published polysomnographic (PSG) studies of PTSD, 14 (six case reports/series and eight case control studies), reporting on up to 266 subjects, have involved Vietnam combat veterans studied, for the most part, 15–25 years after their combat exposure (Brown and Boudewyns 1996; Dow et al 1996; Fuller et al 1994; Kramer and Kinney 1988; Kramer et al 1982; Mellman et al 1991, 1995a, 1997; Ross et al 1994a, 1994b; van der Kolk et al 1984; Woodward et al 1996a, 1996b, 1996c). In two of the eight studies reporting REM latency (RL), the value was elevated (Kramer et al 1982; Kramer and Kinney 1988), and in one it was decreased at a trend level of significance (Dow et al 1996). One other paper, without formally reporting all quantitative data, describes RL less than 40 min and some sleep-onset REM periods (SOREMPs) in a number of cases (Greenberg et al 1972). In the three studies reporting REM density (RD), it was increased with borderline statistical significance in one (Ross et al 1994a), increased in one (Mellman 1997), and unremarkable in one (Dow et al 1996). Sleep architecture was generally unremarkable other than decreased slow-wave sleep in two reports (Fuller et al 1994; Woodward et al 1996a), decreased REM% in one (Kramer and Kinney

From the Minneapolis Veterans Affairs Medical Center, Minneapolis, Minnesota (TDH, MK, BEE); Hennepin County Medical Center, Minneapolis, Minnesota (MWM); and University of Minnesota Medical School, Minneapolis, Minnesota (TDH, MWM, MK, BEE).

Address reprint requests to Thomas D. Hurwitz, MD, Minneapolis VA Medical Center, 116A, 1 Veterans Drive, Minneapolis, MN 55417.

Received August 26, 1997; revised January 28, 1998; accepted February 25, 1998.

0006-3223/98/$19.00
PII S0006-3223(98)00089-4

1988), and increased REM% in another (Ross et al 1994a). Sleep efficiency (SE) was significantly reduced from that of control subjects in one paper at 81% (Mellman et al 1995a), and total sleep time (TST) was reduced in one (Dow et al 1996). In the eight papers citing SE and sleep onset latency (SOL), parameters which would describe insomnia, the former averaged 86.8% (range 81.1–93%) and the latter, 18.0 min (range 7–32.3) (Dow et al 1996; Fuller et al 1994; Kramer and Kinney 1988; Mellman et al 1995a, 1997; Ross et al 1994a; Woodward et al 1996a, 1996b). These values are hardly indicative of severe sleep disturbance. Two studies include multiple sleep latency test (MSLT) data to document daytime sequellae of sleep fragmentation. In one report of 2 cases, both had excessive sleepiness and SOREMPs (Mellman et al 1991), and the same research group has more recently described up to 8 of 11 PTSD patients having SOREMPs during daytime naps (Mellman et al 1997; Mellman 1997). Additional literature on PSG-monitored sleep in patients with PTSD other than Vietnam combat veterans has appeared with a similarly inconsistent variability of findings (Astrom et al 1989; Dagan et al 1991; Glaubman et al 1990; Hefez et al 1987; Hudson et al 1991; Kaminer and Lavie 1991; Kinzie et al 1994; Lavie et al 1979; Lavie and Hertz 1979; Mellman et al 1995b; Peters et al 1990; Schlossberg and Benjamin 1978; van Kammen et al 1987). Differences between published findings may relate in part to variable recency of trauma exposure, small sample sizes, and the presence of potentially confounding comorbid psychiatric disorders among the PTSD subjects. The difficulty of drawing specific conclusions about the nature and role of sleep disturbance in PTSD has been acknowledged (Woodward 1995).

The present investigation was undertaken to evaluate the sleep of Vietnam combat veterans with PTSD utilizing conventional PSG to provide a clinically oriented description of sleep associated with this psychiatric illness. It was hypothesized that, in a group of middle-aged men with frequent complaints of sleep disturbance, there would be PSG documentation of sleep disorders that might contribute to symptoms of PTSD.

Subjects

Twenty-one Vietnam combat veterans were recruited from clinical and community sources at the Minneapolis Department of Veterans Affairs Medical Center, the local Vets Center, and from participants in another study of psychophysiological reactivity recruited from the same sources. Two with untreated obstructive sleep apnea who dropped out of the study and 1 with previously undiagnosed narcolepsy (with onset of hypersomnolence well before service in Vietnam) were not included among 18 subjects on whom analyses are based. One man was included with the use of nasal continuous positive airway pressure therapy (CPAP) following 9 months of effective therapy and, thereby, no sleep-disordered breathing. One subject contributed only 1 night of data and was excluded from analyses involving night 2. Subjects averaged 45.4 ± 5.6 (mean ± SD) years of age. All were outpatients; 14 were currently involved in some outpatient group or individual psychotherapy, while 4 were in no current treatment.

Axis I psychiatric diagnoses (American Psychiatric Association 1987) were assessed utilizing the Structured Clinical Interview for DSM-III-R–Nonpatient version (SCID-NP-III-R) (Spitzer et al 1990). This form of the instrument was utilized because the study originally planned recruitment from nonclinical sources, and any psychotic symptoms that it might not detect would have been noted by the screening psychiatrist (TDH). PTSD was assessed with the SCID module for PTSD (Spitzer and Williams 1987). Diagnostic interviews were conducted by a PhD or masters level psychologist trained in SCID administration and experienced in the assessment of combat-related PTSD. Current axis I comorbidity was found in 10 of 18 (56%) subjects, including dysthymia in 2/18 (11%), major depression in 4/18 (22%), panic disorder in 5/18 (28%), obsessive–compulsive disorder in 2/18 (11%), social phobia in 2/18 (11%), alcohol abuse in 1/18 (6%), and bipolar II disorder in 1/18 (6%). The SCID PTSD module can yield a quantitative severity score between 17 (no symptoms present) and 51 (all symptoms present). These scores and those on the Mississippi Scale of Combat-Related PTSD (MISS) (Keane et al 1988), the Keane PTSD subscale (PK) (Keane et al 1984) of the Minnesota Multiphasic Personality Inventory-2 (MMPI-2), and the Impact of Event Scale (IES) (Horowitz et al 1979) document the magnitude of PTSD in the subjects and are listed in Table 1. Scores in excess of 107 on the MISS have been associated with clinically significant PTSD in Vietnam veteran outpatient samples (Keane et al 1988). Moderate levels of anxiety and depressive symptoms were also documented in these individuals on the Hamilton Depression Rating Scale (HDRS) (Hamilton 1960), Beck Depression Inventory (Beck et al 1988a), Beck Anxiety Inventory (BAI) (Beck et al 1988b), Zung Self-Rating Anxiety Scale (SAS) (Zung 1965), and the Symptom Checklist-90 (SCL-90) (Derogatis 1977).

Ten healthy non–combat-exposed Vietnam era veterans, averaging 46.5 ± 3.2 (mean ± SD) years old, without current axis I psychiatric illness by SCID (except for 1 with tetrahydrocannabinol abuse abstinent for 2 weeks, and 1 with adjustment disorder but no significant depressive or anxiety symptoms on psychometric instruments) served as control subjects. On the PTSD module they did not meet criterion A (trauma exposure), indicating that the

Table 1. Age and Psychological Data

	PTSD subjects (n = 18)		Non-PTSD controls (n = 10)	
	Means ± SD	Range	Means ± SD	Range
Age (years)	45.4 ± 5.6	38–63	46.5 ± 3.2	41–49
SCID	44.3 ± 4.0	39–51	N/A	
MISS	117.5 ± 16.3	88–146	N/A	
Keane	25.1 ± 11.6	10–43	4.0 ± 3.4	0–10
HDRS	10.3 ± 4.9	2–21	0	
BDI	17.2 ± 11.1	2–43	2.6 ± 3.6	0–10
BAI	15.1 ± 11.3	1–41	2.1 ± 3.1	0–10
SAS	41.3 ± 10.5	30–73	26.1 ± 4.0	21–29
SCL-90 (General)	1.22 ± 0.7	0.3–3.5	0.18 ± 0.16	0.02–0.52
IES (Total)	43.2 ± 16.5	0–70	N/A	
IES (Intrusion)	21.2 ± 8.4	0–35	N/A	
IES (Avoidance)	22.3 ± 10.5	0–40	N/A	

instrument is not applicable to the group. Similarly, the MISS and IES do not apply to non–combat-exposed subjects. Scores on the other psychometric measures shown in Table 1 were not indicative of psychopathology.

All subjects reported abstinence from illicit drugs, alcohol, and any psychotropic medication for at least 2 weeks prior to sleep studies, and all were required to have negative urine toxicology screens and breath alcohol determinations when studied in the laboratory. The study protocol was approved by the committees for the use of human subjects in research at the Hennepin County Medical Center and the Minneapolis VA Medical Center, and all participants provided written informed consent to participate. Each was paid a stipend of $100.

Methods and Materials

Subjects provided extensive sleep histories using a clinical sleep disorders questionnaire as well as a semistructured clinical interview including the HDRS by a psychiatrist experienced in sleep medicine (TDH). Subjects were asked to sleep in the laboratory for two successive nights. During the first evening in the laboratory, all subjects completed the BDI, BAI, SAS, MMPI-2, and SCL-90. Urine toxicology and breathalyzer alcohol screening was done at the beginning of the first night of study. Recordings were obtained on Grass Model 78 polygraphs monitoring electro-oculogram (EOG), electroencephalogram (EEG) using the standard central scoring channel (C3 referenced to the opposite mastoid) and eight additional bipolar leads (F7–T3, T3–T5, T5–O1, F8–T4, T4–T6, T6–O2, F3–P3, and F4–P4), electromyogram (EMG) from submental chin muscles as well as bilateral anterior tibialis and extensor digitora muscles, electrocardiogram (EKG), oral–nasal airflow, and tachographic monitor of cardiac rate. Recordings were made at a paper speed of 10 mm/sec with approximately 5 min recorded hourly at 15 mm/sec to screen for epileptiform activity in the EEG. There was continuous technologist observation as well as audiovisual recording. If clinically significant OSA was suspected during the first night of study, the second night was recorded with full monitoring of airflow, thoracic and abdominal ventilatory movements, and oximetry. One subject had been treated for 9 months for OSA and was subsequently studied using an effective level of nasal continuous positive airway pressure. Multiple sleep latency testing (MSLT) (Carskadon and Dement 1987) was performed following spontaneous awakening after the first night's sleep. Criteria for latencies included the onset of the first three consecutive epochs of stage 1 or a single epoch of any other sleep stage. A morning questionnaire inquiring about subjective perceptions of sleep latency, duration, and quality was completed following awakening each morning. Recordings were scored visually by a single, experienced technologist, blind to diagnosis, using standard criteria for 30-sec epochs (Rechtschaffen and Kales 1968), and all PSGs were reviewed thoroughly by board-certified sleep specialists (TDH, MWM). Brief arousals were defined by 1) longer than 2-sec periods of alpha frequency EEG activity or clear wakefulness with or without augmentation of the submental EMG; 2) the presence of an EEG K-complex or desynchronization of EEG if clearly associated with an extremity movement or apnea; or 3) a sleep stage shift if associated with an extremity movement or apnea. REM latency was designated as the interval between the first epoch of sleep and the first epoch of REM, excluding any intervening wakefulness. REM density was calculated by the method of the Western Psychiatric Institute and Clinic (Taska and Kupfer 1983).

Statistical analysis was performed using SPSS version 6.1. Multivariate 2 × 2 repeated measures analyses of variance (RM-MANOVAs) with subject group (PTSD, control) as a between-subjects factor and observation night (night 1, night 2) as the within-subject factor, were used to analyze sets of PSG outcome variables as defined in Table 2, representing sleep onset, continuity, architecture, and REM items. Prior to the multivariate analyses, angular transformation was applied to percentage variables to normalize their distributions (Zar 1984). In view of the possibilities of type II error associated with low statistical power and of isolated significant differences between groups, comparisons on a single variable basis using matched and independent group t tests were also performed to increase statistical power at the risk of increased type I error. Relationships between PSG variables and measures of PTSD severity

Table 2. Groups of Polysomnographic Parameters for Repeated Measures MANOVA

Sleep onset	Sleep continuity, 1	Sleep continuity, 2	Sleep architecture	REM measures
Latency to stage 1	% stage W	Total sleep time	% stage 1	% stage REM
Latency to stage 2	% stage 1	Sleep efficiency	% stage 2	REM latency
Latency to 5 min of sustained sleep	Sleep efficiency	Arousals/hour of sleep	% stages 3 & 4	REM density
	Arousals/hour of sleep		% stage REM	

were evaluated with the use of Pearson product–moment correlations.

Results

Sleep Disorders Questionnaire

In response to a sleep disorders questionnaire, the 18 PTSD subjects reported many symptoms of disturbed sleep and daytime sleepiness as tabulated in Table 3. In spite of these symptoms, only 6 subjects endorsed the symptom of "insomnia," suggesting that more of their concerns were about sleep disruption rather than difficulty falling asleep. PTSD subjects estimated their usual sleep duration to be shorter and SOL longer than did controls. In a clinical interview, 15 of the 18 reported a history of nightmares, bad dreams, and/or panicky arousals; 1/18 restless legs, 7/18 snoring, and 1/18 choking spells. Only 3 control subjects gave a history of any of these symptoms.

Comparisons of Nights 1 and 2

Repeated measures MANOVA for both groups revealed only one significant main effect of observation night, for the sleep continuity-2 set of variables [$F(3,23) = 4.28$, $p = .015$]. This was not true of the similar sleep continuity-1 set and therefore relates to the surprisingly shorter total sleep time on the second night [372 ± 78 (mean ± SD) as opposed to 421 ± 78 min on night 1] for PTSD subjects. Matched *t* tests demonstrated no statistically significant differences between nights for any PSG variables in the PTSD group. For control subjects, the only differences to reach statistical significance on *t* tests were higher sleep efficiency (86.2% ± 8.4 as opposed to 81.8% ± 7.5, $p < .03$) and lower % wake (13.8% ± 8.4 as opposed to 18.2% ± 7.5, $p < .04$) on the second night. These are of modest clinical significance, and there are otherwise no significant indications of a first night effect influencing these data. With this large number of comparisons, the number of significant results is not greater than what might be expected to result from chance variation. PSG data expressed as means from the 2 nights appear in Table 4.

Table 3. Sleep Disorders Questionnaire

Questionnaire items	PTSD subjects ($n = 18$)	Non-PTSD controls ($n = 10$)
Insufficient sleep	16	2
Waking during the night	18	2
Sleep-related movement	12	1
Sleep-related injury	11	0
"Insomnia"	6	0
Difficulty falling asleep	13	0
Unrefreshing sleep	16	1
Oneiric behavior	12	0 (1 answered "?")
Action-packed dreams	17	2
Daytime sleepiness	11	2
Mean usual sleep duration ± SD	4.9 hour ± 1.5	6.8 hour ± 0.7
Mean usual sleep onset latency ± SD	42.5 min ± 37.1	13.7 min ± 8.0

Comparisons of PTSD and Control Subjects

Repeated measures MANOVA with subject group as a between-subjects factor utilizing PSG data from each night revealed no main effects of group membership on analysis of the variable sets described in Table 2. When PSG data from each night were compared separately by independent *t* tests, no significant differences between PTSD subjects and controls were noted for any sleep variable except for less subjectively estimated total sleep

Table 4. Mean PSG Data ± SD for 2 Combined Nights

	PTSD	Control
TST	396.8 ± 53.5	403.4 ± 28.0
Estimated TST	339.1 ± 85.0	411 ± 85.0
SE	82.0 ± 8.0	85.0 ± 5.3
SOL, to stage 1	12.9 ± 7.8	9.7 ± 4.4
SOL, to stage 2	19.4 ± 10.3	12.5 ± 5.2
SOL, to 5 min sleep	20.1 ± 10.8	16.8 ± 13.2
Estimated SOL	32.7 ± 20.0	23.0 ± 7.6
% wake	18.0 ± 8.0	15.0 ± 5.3
% stage 1	12.8 ± 5.2	8.5 ± 3.0
% stage 2	51.2 ± 10.7	55.0 ± 10.1
% stages 3 & 4	13.3 ± 5.9	12.5 ± 7.0
% REM	22.6 ± 6.6	24.1 ± 3.6
REM latency	75.6 ± 17.2	65.2 ± 20.6
REM density	2.2 ± 0.7	1.9 ± 0.6
Arousals/hour	14.0 ± 5.4	15.0 ± 4.3
Arousals from stages 1 & 2/hour	0.3 ± 0.1	0.3 ± 0.1
Arousals from stages 3 & 4/hour	0.1 ± 0.04	0.2 ± 0.1
Arousals from REM/hour	0.3 ± 0.0	0.4 ± 0.1
MSLT	13.5 ± 4.1	12.7 ± 4.4

time for night 1 (329.3 ± 107.8 vs. 442.5 ± 55.3 min, $p <$ .005), longer sleep onset latency to stage 2 on night 2 (21.0 ± 11.8 vs. 11.5 ± 5.5 min, $p <$ .03), and lower arousals/hour from stages 3/4 on night 2 (0.07 ± 0.04 vs. 0.15 ± 0.14, $p <$ .04) for PTSD subjects. With these exceptions, the absence of significant differences for night 1 was essentially replicated by data for night 2, and there were no significant differences to appear on 2 successive nights for any PSG variable. There were no significant differences between PTSD and control subjects noted for SOL1, latency to 5 min of sustained sleep (SOL5), TST, SE, or percentages of sleep stages 1, 2, 3/4, and REM, during the sleep period or % wake during the total recording period. Neither were there any differences for derived values such as RL, RD, or arousals per hour overall or from stages 1 and 2, or REM.

On a morning questionnaire following a night of recording, PTSD subjects reported subjective estimations of TST to be significantly less (329.3 ± 107.8 vs. 442.5 ± 55.3 min, $p <$ .005), and depth of sleep greater than usual ($p <$ .04) after the first night than did control subjects. After the second night, the only significant differences were that subjective estimations of sleep latency were thought to be longer (30.0 ± 15.9 vs. 18.4 ± 6.6 min, $p <$ .04) and to be greater than usual ($p <$ .04) as compared to reports from control subjects.

Comparisons of Subjective Perceptions and Objective Measurements

When subjective perceptions were compared with objective PSG values, PTSD subjects underestimated TST by 82 min ($p <$ .02) after night 1 and likewise, by 26 min ($p <$ .02) after night 2. Their estimated SOLs for night 1 (36.8 ± 33.0 min) were significantly greater than objective SOL1 (13.7 ± 17.0 min, $p <$ .02) but not significantly so for SOL2 (20.4 ± 20.5 min) or SOL5 (22.9 ± 21.9 min). For night 2, their estimated SOL (30.0 ± 15.9 min) was greater than SOL1 (15.3 ± 10.5 min, $p <$ .001), SOL2 (21.0 ± 11.8 min, $p <$ .04), and SOL5 (20.4 ± 13.4 min, $p <$ .04).

Control subjects overestimated TST by 27 min after night 1 and underestimated it by 11 min after night 2, both statistically nonsignificant. SOLs estimated by control subjects (27.5 ± 13.8 min) were also significantly greater than SOL1 (10.0 ± 7.8 min, $p <$ .0001), SOL2 (13.6 ± 9.7 min, $p <$ .0001), and SOL5 (19.2 ± 20.3 min, $p <$ .04) after night 1. After night 2, estimated SOL (18.4 ± 6.6 min) significantly exceeded SOL1 (9.4 ± 6.1 min, $p <$ .005); and SOL2 (11.5 ± 5.5 min, $p <$ .02), but was nonsignificantly greater than SOL5 (14.5 ± 8.8 min).

Measurement of Daytime Sleep Propensity

MSLT revealed normal mean sleep latencies of 13.5 ± 4.1 min with no REM sleep in the PTSD group and 12.6 ± 4.4 min also with no REM in the control group. Neither of these would indicate any significant excessive daytime sleepiness. REM sleep is normally absent from the brief daytime naps monitored during this test.

Other PSG Observations

Polysomnographically recorded sleep was notably better than expected in subjects with clinically significant PTSD and typical histories of disordered sleep. Consistent with the frequently reported absence of frank nightmares during sleep in the laboratory setting, only 5 PTSD subjects recalled any dreams with violent imagery after night 1, and 3 reported them following night 2. Subjects generally experienced satisfying sleep with clinically unremarkable PSGs. Overall arousal indices from nights 1, 2, and combined nights are well below what would be clinically significant. This is supported by the MSLT, where mean sleep latency for five nap opportunities was normal. The absence of REM sleep during daytime naps is consistent with unremarkable timing and physiological manifestations of nocturnal REM sleep. Page by page visual inspection of our PSGs by sleep specialists very familiar with the REM-sleep behavior disorder (TDH, MWM) revealed no abnormality of electromyographic atonia in submental, extensor digitora, or anterior tibialis muscles during REM sleep. There is, therefore, no evidence in this group of subjects to suggest any clinically important abnormality of REM tonic or phasic motor manifestations inherent in the sleep of patients with PTSD.

There was no observational evidence of clinical seizures nor any epileptiform activity in the EEG. Though PTSD patients often describe very long sleep latencies and even a tendency toward delayed sleep phase, there was no indication of any circadian rhythm sleep disorder nor any serious insomnia in this group. Of the latencies to stages 1 and 2, and 5 min of sustained sleep, the only significant difference between subjects and controls was that for stage 2 on night 2, but this is still quite benign at a mean ± SD of 21.0 ± 11.8 min. Four PTSD subjects demonstrated PLMS, with indices of 16–96 per hour; however, arousal indices below 20/hour, MSLT mean sleep latencies greater than 10 min, and no symptoms of restless legs syndrome all suggest that the PLMS are of doubtful clinical significance. This is consistent with a recent report indicating the unclear clinical correlations of PSG-recorded PLMS (Mendelson 1996).

Relationship between Sleep and PTSD Severity

Possible relationships between PSG variables and measures of PTSD severity as measured by the SCID score, MISS, PK, and IES were explored using Pearson product–moment correlations. In view of the absence of significant PSG differences between nights, these comparisons were made with mean data from the 2 nights. The intrusion subscale of the IOE was found to correlate with % stages 3 & 4 ($r = .48$, $p < .05$). There were otherwise no significant correlations between these measures of PTSD severity and any PSG variable.

Psychiatric Comorbidity

Because of the possibility that PSG findings in PTSD subjects could be influenced by comorbid mood disorder, data were compared between 6 subjects with and 11 without current major depression or dysthymia. Current mood disorder subjects had mean ± SD scores on the SCL-90 depression scale, HDRS, and BDI of 2.1 ± 1.2, 12.0 ± 5.9, and 22.2 ± 10.9, respectively. Scores of those without were 1.2 ± .74, 9.5 ± 4.5, and 15.8 ± 10.4. There were no significant differences between these groups for any PSG parameter.

Discussion

These PSG data do not indicate clinically significant gross PSG abnormalities of non-REM or REM sleep in this group of Vietnam combat veterans with chronic PTSD. There are no increases in phasic rapid eye movement density in REM sleep, nor any signs of increased propensity for occurrence of REM sleep during nocturnal recording or daytime MSLTs. Unlike most other studies of Vietnam combat veterans, our sample includes no inpatients and may represent a less acutely disturbed group in spite of their clinical and psychometric manifestations of PTSD. It is not clear how they might otherwise differ. With the exception of the nondiminished nocturnal excretion of 3-methoxy-4-hydroxyphenylglycol reported by Mellman et al (1995a), no studies of PSG in Vietnam combat veterans include any other neuroendocrine markers such as diminished dexamethasone suppression of cortisol (Yehuda et al 1993) or pharmacologic probes such as yohimbine induction of PTSD symptoms (Southwick et al 1993), which might further distinguish study subjects.

The tendencies for these subjects to underestimate TST and overestimate SOL do suggest that their perceptions of sleep disturbance may outweigh objective findings, as in another report of Vietnam combat veterans with PTSD (Woodward et al 1996a) and typical of the subjective insomnia complaint known as sleep state misperception (American Sleep Disorders Association 1997). Though quantitative differences between subjects and controls on these two parameters were not strong, subjects did endorse many more symptoms of sleep disturbance, showing much more contrast between history and benign PSG data.

It may be that the more clinically significant aspect of sleep in chronic PTSD is related to adaptation than hyperarousal. Our subjects were noted to sleep more soundly than expected. They also had fewer arousals/hour from stages 3/4 than did controls on night 2. IES intrusion scale was positively correlated with mean % stages 3/4 for the 2 nights. Visually scored sleep stages, SE, TST, and arousal indices indicate rather continuous sleep but do not address the level of vigilance during sleep, which has been cited as a very important finding, namely the elevation of auditory arousal thresholds in affected subjects during non-REM as well as REM sleep (Dagan et al 1991; Schoen et al 1984; Lavie P, personal communication). Kramer (1993) interprets these findings as evidence that persons suffering chronic PTSD may have a heightened responsiveness to internal events while being less arousable by external stimuli. Depth of sleep may represent a chronic adaptation to trauma. Kaminer and Lavie describe the diminished dream recall of their more well-adjusted Holocaust survivors compared with those less well adjusted (Kaminer and Lavie 1991). The common subjective sleep complaints of chronic PTSD subjects may reflect a breakdown of this adaptation but with enough resilience to permit intact sleep in a safe, neutral environment such as the sleep laboratory.

In conclusion, there is no clinically significant sleep disorder or typical pattern of sleep disturbance detectable by standard PSG to be found in this group of Vietnam combat veterans, which would explain or potentiate their symptoms of well-documented chronic PTSD. It has been suggested that these veterans may sleep more soundly in an environment perceived as safe (Woodward 1995), and some of these men did confirm that impression after their participation. There are, however, no data beyond subjective reports to document the perceived contrast between reported history and laboratory sleep in these subjects. A pattern of unremarkably intact sleep architecture, mild decrease in sleep efficiency, relatively unremarkable sleep onset latencies, normal circadian sleep distribution (no daytime sleepiness nor occurrence of REM sleep), absence of parasomnias and seizures, a tendency toward fewer interruptions of slow-wave sleep, and a trend toward subjective overestimation of difficulty falling asleep and underestimation of total TST emerges in this study population. The most compelling observation in this study is the intact capacity for sleep in these individuals with histories of serious and persistent sleep complaints but who sleep well in a novel situation. This important finding suggests that behavioral therapy techniques such as are

used in cases of psychophysiological insomnia should be investigated for the treatment of sleep complaints of PTSD.

This work was supported in part by grants to Dr. Hurwitz from Hennepin Faculty Associates and to Dr. Engdahl from the United States Department of Veterans Affairs.

The authors wish to thank Ms. Andrea Patterson, Ms. Ruth Elling, and the technical staff of the Minnesota Regional Sleep Disorders Center for their excellent performance of these polysomnographic studies.

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