Disturbed EEG Sleep, Paranoid Cognition, and Somatic Symptoms Identify Veterans with Post-Traumatic Stress Disorder.

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Introduction:

The disabling behavior symptoms of military combatants, who were involved in the Viet Nam war, provided the impetus to characterize those psychological disturbances as post-traumatic stress disorder (PTSD). This psychiatric diagnosis first appeared in the third edition of the APA Diagnostic and Statistical Manual (DSM 3), with its seminal feature being a specific precipitating stressful event. Subsequently, two major revisions of the DSM better characterized PTSD's behavioral diagnostic features. There are 3 clusters of behaviour symptoms in DSM 4 (1) and 4 cluster groups in DSM 5 (2). These 4 clusters groups comprise: re-experiencing, avoidance, negative mood and cognitions, such as depressive, hostile, aggressive, and paranoid thinking, and behavioral arousal including sleep disturbances. In addition to its behavioral features, military medical personnel have described a variety of chronic disabling physical symptoms following exposure to combat (3). These troublesome physical complaints lack any objective physical pathology. Where the focus is on unexplained widespread variable musculoskeletal pain and chronic fatigue, the current preferred diagnosis is fibromyalgia (formerly known as rheumatism or fibrositis). This diagnosis overlaps with such equally medically-unexplained syndromes as Gulf War Syndrome, chronic fatigue syndrome, combat fatigue, (with previous labels including: Da Costa's syndrome, disordered action of the heart, neurasthenia or neuro-circulatory asthenia, and effort syndrome). Patients with fibromyalgia have unrefreshing sleep with disturbances in EEG sleep physiology. Moreover, experimentally induced sleep physiological disturbances in normal subjects and animals result in increased pain sensitivity and pain symptoms. Clinical and epidemiological studies show that chronic sleep disturbances are integral to a vicious self-perpetuating cycle of chronic pain, fatigue, negative

mood and cognition in patients with fibromyalgia. These symptoms may be precipitated by a distressing traumatic industrial or motor vehicle accident (4). Therefore the purpose of this study was to determine whether the disordered physiology of the sleeping/waking brain may provide the link between the behavioral and somatic symptoms of those military personnel suffering from post-combat chronic PTSD. The analyses comprised a 2-part strategy—first to establish differences between treatment-resistant PTSD and non-PTSD military personnel in multiple domains comprising EEG sleep physiology, behavioral, and the medically unexplained pain and fatigue somatic symptoms and second, to generate a decision-tree probability model for PTSD based on these differences. That is, the key behavioral elements that distinguish chronic PTSD from non-PTSD military comprise a specific stressful traumatic event (i.e., combat), altered sleep EEG physiology, persistent unexplained somatic symptoms, and negative cognitive behavior.

We employed a case-controlled study of PTSD in which both case and control groups included veterans with and without experience in combat zones, enabling us to separate PTSD symptoms from those that might be associated with combat experience alone, and to assess combat experience along with other patient measures when building models. We measured behavior symptoms including, negative cognition (depressive, hostile, aggressive, and paranoid thinking), and chronic physical health concerns including, disturbances in EEG sleep physiology, sleep quality, and medically unexplained somatic symptoms (e.g. musculoskeletal pain and fatigue).

Methods:

Subjects:

The subjects comprise consecutive patients referred between 2008 to 2014 from an Operational Stress Injury (OSI) clinic of the Dept of Veteran Affairs of Canada. The OSI clinic specializes in the assessment and treatment of PTSD and other operational stress injuries in the Canadian military and veteran forces. The diagnosis of PTSD was determined by a psychiatrist employing a compehensive psychiatric examination and the Clinician-Administered PTSD Scale for DSM-4 (CAPS) (5) where the CAPS mean rating was 77.38 s.d., 21.36. In order to control for commonly disrupted and insufficent sleep problems in the military, we compared sleep and symptoms of these exposed war-combat CF veterans with persistent symptoms of PTSD to CF personnel without a history of combat exposure who complained of chronic insomnia . This latter group of subjects were referred from Canadian Dept. of National Defence (DND) medical ambulatory clinics for diagnostic assessment of a possible chronic primary sleep disorder such as sleep apnea or restless legs/sleep-related periodic limb movement disorder.

The subjects consented to be assessed in the Centre for Sleep and Chronobiology in Toronto for specialized comprehensive sleep/wake clinical and laboratory investigations of their chronic treatment-resistant symptoms. The research was reviewed and approved by the DND, Institute of Military and Veterans Research Institute in association with Defense Research and Development Canada.

See table 1 for the description of the subjects: Gender, Mean age, Mean BMI, and the independent variables: Combat experience (Combat 1=Yes, 0= No or Unknown) and presence of treatment-resistant PTSD (PTSD 1= Yes, 0= No) were independent variables. Note that the sample of subjects from the OSI clinic also included those with combat exposure and no PTSD, as well as those with PTSD and no combat exposure. In order to control for commonly disrupted

and insufficient sleep problems in the military, we compared sleep and symptoms of CF veterans with persistent symptoms of PTSD to CF personnel without PTSD who complained of chronic sleep difficulties. There were no significant differences in the two groups' ages and BMI values. There were no differences comparing noncombat females (n=9) to noncombat males (n=50) with MANOVA statistic, so that both genders were included in the study. Combat experience included those patients who had served in combat zones (e.g. Gulf-war, Afghanistan), and those who developed treatment-resistant PTSD symptoms following deployment to so-called "U.N. peace-keeping missions" in dangerous combative environments i.e., the Balkans (Bosnia and Croatia), Africa/Middle East (Suez Canal zone, Rwanda, Somalia, and Cyprus). Six cases of PTSD occurred in sleep-disturbed, non-combat CF patients. Distressing noncombat traumatic events were quite varied. They included: sexual assault, witness to a major airplane crash, engagement in dangerous service with the military police, witness of a suicide of a soldier, and participation in a military training exercise.

All treatment-resistant PTSD subjects were functionally impaired despite psychological and lengthy pharmacological treatments [median 32.5 mon. (range 6 to 249 mons.)].
All PTSD patients had received cognitive behavioural treatments which included trauma-focussed behavior therapy such as prolonged exposure and various types of psychological support e.g., individual, marital, family counselling and group therapies.
At the time of the sleep study, they were taking at least 2 drug class combinations of antipsychotics (e.g.risperidone, quetiapine, aripiprazole), and various antidepressants (e.g., mirtazepine, bupropion, venlafaxine, citalopram).13 received an anticonvulsant mood modulator (sodium valproate or topiramate). 9 received pain medication (opiates:3; NSAIDs:6). Only 2

patients had found that their alpha1- adrenergic receptor blocker (prazosin) satisfactorily reduced recurrent nightmares. Seven received benzodiazepine sedative hypnotics;16 received medication for cardiovacular problems, and 3 were treated for type 2 diabetes.

In comparison to the PTSD patients, the 59 with unremitting sleep problems and no PTSD used fewer antidepressants (n=9), anticonvulsants (n=2) antipsychotics (n=3) and sedative hypnotics (n=1), (all Chi square $p<.005$). They were prescribed similar cardiovacular drugs (n=13),antidiabetic drugs (n=4), but more NSAIDs (n=11) and no opiates. The records that were made available did not specify why they were given the psychotropic drugs.
The psychotropic treatments were not withdrawn in order to avoid potential adverse effects and permit full co-operation of the patients to participate in the various physiological and behavioral proceedings.

Those subjects who had been previously identified by the military physicians as having problems with alcohol and/or substance abuse/dependency had been treated for their condition(s) before the time of referralfor their sleep study. For this study, all subjects completed a self-rated standardized question on the types, amount, and frequency of use of alcohol and recreational drugs over the previous year and during the two weeks prior to their sleep study. During the two weeks prior to the sleep study neither group abused alcohol and there were no differences in their alcohol habits. During the previous year 8 combat-related PTSD subjects used cannabis vs. none of the sleep disturbed, non-PTSD subjects (Chi Square $p<.01$).Within the two weeks prior to the sleep study only 2 combat-related PTSD subjects had occasionally taken cannabis drugs vs. none were used in the noncombat, sleep-disordered group (Chi Square p= n.s.).

The subjects with and without PTSD completed psychological and physical symptom self-reports prior to a single over-night EEG sleep study. Because sleep disturbance has the potential to promote negative cognition such as irritability, aggressive behaviour, paranoid and depressive thinking that may occur with PTSD (6,7,8) we employed the Symptom Checklist-90 [SCL-90] subscales for hostility and paranoid thinking (9,8) and the Beck Depression Inventory [BDI] (10). Physical symptoms were self-rated from 1 to 5 with the Wahler Physical Symptom Inventory[WPSI] (11,12).The number of symptoms self-rated as 4 or 5 on the WPSI scale (corresponding to "about twice a week" or "nearly every day") were tallied in order to determine the prevalence of troublesome symptoms. Frequency of regional pain symptoms (13), and severity of fatigue (range 1 "full of energy" to 7 "totally physically exhausted") (14), and Stanford Sleepiness Scale (15, 16)) were self-rated before and after the overnight sleep.

The frequency of sleep -related behavioural disturbances during the previous 2 months were rated on the Sleep-Wake questionnaire with a graded scale of 0-3 (0- never, 1- sometimes, 2- often, and 3- always). The behavioural disturbances during sleep included: frequency of awakening during the night due to nightmares or unpleasant dreams; told that you had screamed or shouted; disturbing (bad) dreams during sleep. Abnormal cognition included self-ratings of frequency of harmful behavioural to self and others noted in BDI Q9 (10): thoughts of killing self-rated on a scale of 0-3. Abnormal temper outbursts which could not be controlled included: urges to beat, injure, or harm someone; urges to break or smash things; shouting or throwing things (questions from the SCL 90 which were rated on a scale of 0-4: (0-Not at all, 1-A little bit, 2-Moderately, 3-Quite a bit, 4-Extremely)(11).

Physiological assessment of sleep comprised standardized EEG polysomnography (PSG). The PSG included electroencephalogram (EEG C3, C4) electro-oculogram, submental and bilateral anterior tibialis electromyogram, single anterior lead electrocardiogram, measures of respiration comprising measures of airflow with oral-nasal thermistors, respiratory impedance plethysomography, and pulse oximetry. An experienced registered PSG technologist completed blind ratings of sleep physiological indices [17, 18]. The sleep EEG stages were scored according to the original Rechtschaffen and Kales criteria (17), which was required for a computerized physiological sleep measures of EEG sleep stability known as the Cyclical Alternating Pattern (CAP) (19-21). Sleep EEG CAP is characterized by sequences of transient EEG changes in nonREM sleep that occur distinctively from the background EEG activities. They comprise 3 subtypes: CAP $_{A1}$ is related to favorable sleep stability. CAP_{A2} and $CAP_{A3,}$ are progressive indices of poor sleep quality or sleep instability (19). These abnormal physiological features have been observed in patients who suffer from chronic widespread musculoskeletal pain and fatigue (22, 23), as well as sleep-related breathing disorders (24). Analysis of nocturnal sleep EEG CAP was determined by an Embla computerized automatic detection system (21, 22).

Data analyses

SAS Software Multivariate Analysis of Variances models (MANOVAs) were fit into four groups of dependent variables. These included: PSG and sleep EEG CAP indices; self-ratings of behavior including negative cognition, pre- and post PSG sleepiness and fatigue; physical symptom severity. Combat experience (Combat 1=Yes, 0= No or Unknown) were the independent variables that were compared to whether or not they had treatment-resistant PTSD

(PTSD 1= Yes, 0= No).See Table 1. Because there were only 6 patients with PTSD =1 and Combat = 0, we did not test for PTSD x Combat experience interactions. EEG sleep measures included sleep architecture and computerized measure of EEG sleep stability, i.e., sleep EEG CAP rate. MANOVA permitted the testing the significance of suites of measures, while accounting for relationships among the dependent variables as well as the relationships between independent and dependent variables. Initial data exploration showed skewed distributions, and outliers, particularly for the sleep physiology measures. Normality assumptions were likely violated. For significance testing, MANOVA and ANOVA models were fit to ranked measures; tests on ranked data provided nonparametric tests (25-28). All inferences were based on these ranked nonparametric tests. Because multiple testing inflates Type 1 error, or the probability of rejecting the null-hypothesis when true, P-values from ANOVAs were adjusted for multiple testing within each of the four independent variable groups using the adaptive step-down Holm method (29). This method controlled for the error rate within a family of tests (family-wise error). The test does not assume independence between the various dependent variables in these ANOVAs.

We used three methods to identify distinguishing characteristics of PTSD and non-PTSD patients: Canonical Discriminant Analysis (CDA), Logistic Regression, and Decision Trees. PTSD was the dependent variable. Patient measures and combat experience were the independent variables.

We fit a Chi-square Automatic Interaction Detection (CHAID)-like decision tree algorithm using SAS Enterprise Miner (28). The CHAID approach finds splitting rules that create pure segments of data using a Chi-Square test of association with a significance cut-off (alpha of 0.05) where all

p-values are adjusted for multiple testing using a Bonferroni method. Missing values were addressed through multiple imputation using the predictive mean matching method (30,31), which is more appropriate for non-normal measures (32). BDI was excluded from decision tree and regression modeling because this self-rated test includes both somatic symptoms such as sleep, pain, fatigue and abnormal cognitive behavioural data.

Results

PTSD differed from non-PTSD subjects in prevalence of suites of physical and behavioural health problems, while controlling for the effects of combat exposure. MANOVA tests showed significant differences between PTSD and non-PTSD subjects on sleep measures ($P < 0.01$), psychological ($P < 0.0001$), and physical symptoms self-reports ($P < 0.01$). The MANOVA test showed no significant difference between PTSD and non-PTSD on subtypes of sleep EEG CAP measures ($P > 0.05$).

While controlling for effects of combat exposure, individual ANOVAs showed differences between PTSD and non-PTSD groups in their distributions of onset to EEG sleep, sleep efficiency, EEG stage 4 %, REM sleep onset latency, REM % and EEG CAP indices measures including CAP frequency, CAP A2, CAP A3 / CAP A1+A2+A3(22). See Table 2. Those with PTSD took longer to fall asleep, had less efficient sleep (i.e., less time asleep relative to time in bed), less deep (stage 4) sleep, lower percent of time in REM sleep, a prolonged delay to onset of REM sleep. The higher CAP frequency index in the nonPTSD group is consistent with their complaint of chronic poor quality of sleep in the absence of a specific combat-related traumatic

experience. These sleep EEG differences may be influenced by more frequent use by the PTSD group of psychotropic drugs.

There were no group differences in prevalence of such primary sleep disorders as obstructive sleep apnea or sleep-related periodic leg movements. The prevalence of PTSD subjects with an index of obstructive sleep apneas/hypopneas per hour of sleep>5 per hour (n=35) did not differ from NonPTSD subjects (n=25, Chi square=n.s.). Furthermore, there were no statistical differences in their frequencies of sleep EEG arousals from sleep apnea/hypopneas or with sleep-related periodic leg movements (PLM) (see Table 2).

Behavioural disturbances during sleep were more common among the PTSD veterans. Of the 39 PTSD subjects, 32 were more likely to be awakened by nightmares vs.11 of 59 non-PTSD subjects (chi square test, $p<0.05$). 21 PTSD subjects vs. 6 non-PTSD reported screaming or shouting during sleep (Chi square $p <0.05$). Seven of those in the combat-related PTSD group also reported inadvertently striking, but not seriously injuring, their partner during sleep whereas none of the non-PTSD group described any abnormal sleep-related behavior.

PTSD patients had greater mean ratings on the WPSI with more frequent physical problems (commonly musculoskeletal pain) and post sleep fatigue (Table 3).

PTSD patients described significantly more BDI depressive symptoms. See Table 4. Further, seven PTSD subjects reported suicidal intentions vs. none of the non-PTSD subjects.

More PTSD subjects reported hostility and paranoid notions on the SCL-90. (Table 4).Nineteen subjects reported domestic and/or violent social behavior with 3 being charged and arrested for assaultive behaviour. In contrast, none of the non-combat non-PTSD subjects reported any form of social misbehavior (Chi square, $p<.001$).

Canonical Discriminant Analysis (CDA), and in particular Logistic Regression and Decision Trees identified the most salient combinations of measures that characterize PTSD. All five runs of CDA, which used multiply imputed datasets, showed significant discriminant functions ($P< 0.001$). Excluding combat exposure, the top six variables that are most correlated ($|r| > 0.4$) with these functions and were the most discriminating characteristics of PTSD, comprise the following features: BDI total score, paranoid score, REM onset latency, SCL-90 hostility scale score, WPSI symptom score and reduced REM% sleep. Regression models that minimize Schwartz Criteria reveal that PTSD is described by REM onset latency, paranoid score and combat exposure. Subjects with combat experience had 4.7 times the odds of having PTSD compared to those without combat exposure. Because SSRI and SNRI anti-depressants may cause a delay in REM onset latency (33) and both antipsychotic as well as anticonvulsant neurotropic drugs may influence EEG sleep physiology we also re-ran the selection routine while forcing these drugs as variables into the model. The selection routine chose the same variables. PTSD in these subjects is still described by REM onset latency, paranoid score and combat exposure, even while controlling for the use of antidepressant, antipsychotic and anticonvulsants.

The CHAID–like decision tree analysis reveals the key traits that distinguish PTSD from non-PTSD and their relative importance. Paranoid score was the most important variable in the tree

model, followed by REM onset latency, exposure to combat and WPSI severity score based on each measure's contribution to impurity reduction across the tree (relative importance: 1, 0.79, 0.77, and 0.49 respectively). For subjects who were exposed to combat, longer delays to onset of REM sleep (REM onset latency $\geq$ 175.5 min.) lead to higher propensity of exhibiting PTSD (0.79 or 79%). Lower REM onset latency and high paranoid scores ($\geq$ 7.5) lead to high PTSD propensity (0.857 or 85.7%), whereas low paranoid scores (<2.5) lead to low PTSD propensity. With no combat experience, PTSD is still highly probable (0.83 or 83%) with higher paranoid scores ($\geq$ 2.5) and higher somatic symptom ratings (WPSI $\geq$ 11). On the other hand, those non-combat subjects with lower paranoid and somatic symptom ratings were likely to have a low propensity for PTSD (See Fig.1).

Discussion:

This study provides provisional evidence for the key clinical and physiological sleep features that characterize veterans who remain functionally disabled with PTSD despite various drug and behavioral treatments. The results are consistent with DSM 5 behavioural symptomology. The association of malfunctions of the sleeping/waking brain and medically unexplained physical symptoms with DSM 4 behavioral symptoms, identified in the MANOVA and CDA analyses, broaden our understanding of chronic PTSD. These psychological and somatic symptoms are consistent with our hypothesis that EEG sleep disturbances may provide the link between negative cognition and mood, and somatic symptoms. While ANOVA and MANOVA statistics show these symptoms to be independent of combat stressor effects, the war-zone experience itself is a key characteristic of PTSD amongst military personnel, as shown by both the logistic

regression and decision tree models (see Fig 1). Our decision tree model in Figure 1 shows that combat military service, disturbed REM sleep, paranoid thinking, and medically unexplainable somatic symptoms (commonly musculoskeletal pain) characterize CF veterans with chronic PTSD. However, the regression model, while identifying combat and altered EEG sleep, did not include somatic symptoms.

Our EEG sleep results complement previous studies that show the importance of disturbances in REM and nonREM sleep. Those with chronic PTSD take longer to fall asleep, have reduced sleep efficiency, reduced stage 4 (deep) sleep, reduced and delayed onset to REM sleep. The higher EEG CAP frequency in the nonPTSD subjects, a sensitive marker for sleep instability (38), may be related to their primary complaint of disturbed and unrefreshing sleep. A meta-analysis of previous polysomnographic studies of military and civilian PTSD patients vs. those without PTSD showed similar findings with more stage 1 (light) sleep, less slow-wave (deep) sleep and alterations in REM (34). Furthermore, our findings are consistent with previous PTSD sleep studies which show prolongation in the onset to REM sleep (35, 36). Unlike previous studies that associate sleep-related breathing disorders to sleep EEG CAP (37) and to PTSD in the military (38), this study found no statistical differences in apnea/hypopnea index, sleep-related respiratory arousals, or frequency of periodic limb movement arousals between those with combat-related PTSD and those sleep disturbed nonPTSD military subjects.

In this study of CF veterans with chronic post-combat PTSD, a prolonged delay in the onset to REM sleep is accompanied by negative mood and negative cognitive symptoms. Whereas most tricyclic, SSRI, and SNRI antidepressant drugs may reduce and delay the onset to REM sleep

(33), the delay in onset to REM sleep persists when use of antidepressant, as well as anticonvulsant and psychotropic drugs are statistically-controlled. A previous sleep EEG study, however, that compared the sleep of drug-free combat-related PTSD patients with major depression patients and normal subjects showed that the PTSD group had similar reduction in REM sleep and no delay in onset to REM sleep (38). Overall, our results are supportive of Germain's hypothesis and previous EEG sleep research that disturbed REM and non-REM sleep contribute to maladaptive stress and trauma responses in PTSD (39).

Ideally, further studies of sleep physiology should involve comparisons between a group of non-sleep-altering medicated sleep-disturbed subjects with PTSD and age-matched normal healthy sleepers who are acclimatized to the novelty of the lab environment. In order to affirm the importance of a disorganization of the REM/Non REM sleep as a possible key factor to the emergence of PTSD, it would be preferable to examine the EEG sleep before any drug administration, which may influence sleep physiology. The design of the research did not permit a comparative study with those who had benefitted from specific pharmacological and/or behavioral treatment. A future study employing similar physiological and behavioral methodology should comprise a large group of randomized, early-identified combat-exposed vs. noncombat-exposed subjects who are free of potentially sleep EEG confounding psychotropic drugs. Such research may pave the way to determine what pharmacological and behavioural methods would be useful in those early-detected post-combat military with PTSD.

Our decision to include measures of negative cognition and mood in PTSD patients, diagnosed using DSM-4 criteria, highlights their importance in the current DSM 5 criteria. This finding

highlights the importance of paranoid and hostile behaviour in those with PTSD following a major traumatic experience (6). Their presence in these veterans with chronic PTSD, despite their various drug and behavioral treatments, may stem from an inability in civilian life to overcome their combat-ready vigilant mindset, which becomes ingrained in military training. Such cognitive and behavioral abnormalities may contribute to the increased prevalence of dysfunctional domestic and social behaviour in U.S. veterans with PTSD (40), and in post-combat British veterans (41).

In conclusion, the results of this study and previous clinical research support the notion that chronic disturbed sleep physiology, unexplained musculoskeletal pain and fatigue symptoms should be considered as integral to irremediable post combat PTSD. Moreover the presence of hostile and paranoid ideation may be a cautionary signal to the potential for antisocial behaviour. Further studies are required to determine whether civilians with PTSD who are victims of psychologically stressful events, (e.g. sexual assault, non-physically injurious MVA, industrial and environmental disasters) may demonstrate similar distinguishing features as noted in Figure 1.

Tables and Figures

Table 1. Sample sizes and subject descriptions.

	PTSD-YES Combat -Yes	PTSD-YES Combat-No	PTSD-NO Combat-Yes	PTSD-NO Combat-No
Total Number of Subjects	**33**	**6**	**28**	**31**
Number of Females	**0**	**0**	**2**	7
Number of Males	**33**	**6**	**26**	**24**
Mean Age	**44.4**	**46.8**	**40.5**	**41.6**
Mean BMI	**30.1**	**32.5**	**30.1**	**32.5**

Table 2

Results of ANOVAs Sleep Physiology measures. All P-values from Ranked (non-parametric) tests are adjusted using the Adaptive Holm method to control family wise error rate. Least squares means (LSMean for PTSD NO (0) and YES (1) are adjusted to remove combat effects.

	PTSD						
	NO			YES			
Dependent	**LSMean**	**SE**	**n**	**LSMean**	**SE**	**n**	**P-Value**
Sleep Onset Latency (min.)	11.9	1.81	58	17.38	2.47	38	**<0.05***
Sleep Efficiency %	85.5	2.05	58	81.19	2.81	38	**<0.05***
Stage 1 %	12.36	1.7	58	14.56	2.32	38	NS
Stage 2 %	61.76	1.36	57	66.31	1.84	38	NS
Stage 3 %	6.33	0.78	57	5.74	1.06	38	NS
Stage 4 %	2.01	0.44	56	0.91	0.61	36	**<0.05***
REM %	19.14	0.93	57	11.66	1.26	38	**<0.0001***
REM Onset Latency (min.)	124.91	10.61	56	205.9	14.69	35	**<0.001***
Apnea-hypopnea Arousals	74.73	15.92	57	109.6	21.79	37	NS
PLM arousals	17.57	5.16	57	32.98	7.27	36	NS
Spontaneous Arousals	51.48	4.62	58	45.25	6.38	37	NS
CAP A2 Index Cycle	7.07	0.74	56	5.49	1.01	37	NS
CAP A3 Index Cycle	9.16	0.99	56	6.01	1.34	37	NS
CAP A1/CAP A1+A2+A3	0.25	0.02	56	0.24	0.03	37	NS
Total CAP Index Cycle	74.77	4.23	56	57.83	5.75	37	**<0.05***

Table 3.

Physical self-reports. All P-values from Ranked (non-parametric) tests are adjusted using the Adaptive Holm method to control family wise error rate. Least squares means (LSMean) for PTSD NO (0) and YES (1) are adjusted to remove combat effect.

	PTSI						
	NO			YES			
Dependent	**LSMean**	**SE**	**n**	**LSMean**	**SE**	**n**	**P-Value**
Presleep Sleepiness	2.94	0.14	53	2.99	0.20	33	NS
Post sleep Sleepiness	3.03	0.14	51	3.27	0.19	34	NS
Presleep Fatigue	3.67	0.20	53	4.38	0.28	33	NS
Post sleep Fatigue	2.93	0.18	51	3.88	0.24	34	**<0.01***
Presleep Pain	3.02	0.51	52	5.25	0.71	34	NS
Post sleep Pain	3.19	0.57	51	5.16	0.78	34	NS
Somatic Symptoms (WPSI)	5.44	0.63	57	10.15	0.88	37	**<0.001***

Table 4.

Results of ANOVAs on raw and ranked data for SCL-90 subscales paranoid and hostility and BDI psychological self-reports. All P-values from Ranked (non-parametric) tests are adjusted using the Adaptive Holm method to control family-wise error rate. Least squares means (LSMean) for PTSD NO (0) and YES (1) are adjusted to remove combat effects.

	PTSD						
	NO			YES			
Dependent	**LSMean**	**SE**	**n**	**LSMean**	**SE**	**n**	**P-Value**
Hostility Score	3.02	0.63	53	6.56	0.84	36	**<0.001***
Paranoid Score	2.05	0.48	56	5.68	0.66	35	**<0.0001***
BDI Score	9.88	1.05	56	22.74	1.47	36	**<0.0001***

Figure 1.

CHAID like decision tree with BDI removed from analysis. The model shows the percentage (probability x 100) of PTSD given patient combat experience, REM Latency, and Paranoid and WPSI scores. Red segments show groups of patients with higher probabilities of PTSD than the overall average, while green segments show groups of patients with lower probabilities.
N.B. Because both PTSD and non PTSD subjects are shown, the figure uses the DSM-4 PTSD term to describe each of the segments.