



THEORETICAL REVIEW

Posttraumatic stress disorder and sleep-disordered breathing: a review of comorbidity research



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SUMMARY

Posttraumatic stress disorder (PTSD) and sleep-disordered breathing (SDB) are common disorders, but limited data address their co-morbidity. Emerging research indicates PTSD and SDB may co-occur more frequently than expected and may impact clinical outcomes. This review describes historical developments that first raised suspicions for a co-morbid relationship between PTSD and SDB, including barriers to the recognition and diagnosis of this co-morbidity. Objective diagnostic data from polysomnography studies in PTSD patients reveal widely varying prevalence rates for co-morbidity (0–90%). Use of standard, recommended technology (nasal cannula pressure transducer) versus older, less reliable technology (thermistor/thermocouple) appears to have influenced objective data acquisition and therefore SDB rates in sleep studies on PTSD patients. Studies using higher quality respiratory sensors demonstrated the highest prevalence of SDB in PTSD patients. Clinical relevance, theoretical models and research recommendations are discussed. The lack of widely acknowledged, tested, or proven explanatory models and pathophysiological mechanisms to understand the relationship between these two disorders may prove formidable barriers to further investigations on prevalence and clinical relevance, albeit both conditions are associated with waking or sleeping hyperarousal activity, which may inform future studies.

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Introduction

Conceptualizations in the scientific literature about post-traumatic stress disorder (PTSD) and sleep were sparse until the past decade [1–4], at which point a clinical sleep medicine perspective on PTSD emerged [5–16]. Yet, this newer information has been slow to diffuse into the psychiatric or psychological literature where PTSD research commonly omits sleep as a primary variable of interest. While insomnia and nightmares are frequently reported sleep symptoms in PTSD [2,7,8,16,17] rare attention is given to sleep-disordered breathing (SDB) either in terms of prevalence or in developing theories addressing co-morbid sleep disorders and PTSD.

This review article examines the possible co-morbid relationship between SDB and PTSD, first by providing a brief overview of SDB pathophysiology followed by an outline of the historical developments that initially raised suspicions for a potential co-morbid relationship between SDB and PTSD. To shed more light on this historical progression, we describe key barriers that likely inhibited recognition and diagnosis of SDB in PTSD patients. With these backdrops, we describe our literature search and results for the known prevalence of SDB in PTSD patients as well as possible clinical relevance of this alleged co-morbidity. Finally, we delve into theoretical models and research recommendations to facilitate hypothesis development about this co-morbidity.

Assessment of sleep-disordered breathing

SDB is a common sleep disorder, affecting as many as 9–24% of the adult population [18]. Obstructive sleep apnea (OSA) is the most common form of sleep breathing problem, which comprises three

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Abbreviations

AASM	American Academy of Sleep Medicine
AHI	Apnea hypopnea index (total number of apneas and hypopneas divided by total hours of sleep)
FLE	Flow limitation event
HPA	Hypothalamic-pituitary-adrenal
NCPT	Nasal cannula pressure transducer
OSA	Obstructive sleep apnea
PAP-T	Positive airway pressure therapy
PSG	Polysomnography
PTSD	Posttraumatic stress disorder
RDI	Respiratory disturbance index (total number of apneas, hypopneas, and flow limitation events divided by total hours of sleep)
RERA	Respiratory effort-related arousal
SDB	Sleep-disordered breathing
SpO ₂	Blood oxygen saturation
TBI	Traumatic brain injury
UARS	Upper airway resistance syndrome

precisely defined obstructive breathing events known as apneas, hypopneas, and respiratory effort-related arousals (RERA), each of which collapses the airway and reduces airflow for 10 s or longer [19,20]. Apneas reflect near or total cessation of airflow; hypopneas reflect roughly 30%–70% reduction in airflow; and, RERAs, which also are termed flow limitation events (FLEs) or, more generally, upper airway resistance syndrome (UARS), comprise a less than 30% reduction in airflow but which otherwise remains ill-defined [20]. Apneas and hypopneas are often accompanied by oxygen desaturations or fluctuations; whereas RERAs show limited or no changes in oxygenation. All three sleep breathing events trigger cortical arousals or awakenings after which normal breathing resumes, and these episodes may recur throughout the sleep period, which provides a rationale for viewing SDB as a generator of nocturnal hyperarousal activity. One additional breathing event potentially relevant to patients with PTSD is the central apnea: changes in the central nervous system, usually attributed to wide fluctuations in carbon dioxide levels, cause breathing cessation while asleep despite an unobstructed airway [21]. Prior research indicates anxiety patients [22] as well as traumatic brain injury patients [23] show risks for central apneas.

Historical developments

Several papers on the potential role of SDB and posttraumatic stress symptoms or related topics appeared as early as the 1980s; Guilleminault noted a relationship between SDB and anxiety dreams [24]. In 1991, a small study of 24 war veterans demonstrated a 54% rate of SDB in combat related PTSD [25]. Later, de Groen found an association between snoring and anxiety-related dreams [26]. In 1995, another study showed nearly half of combat veterans with PTSD compared with 13% of a non-clinical sample demonstrated apneas and hypopneas, although the number of breathing events per hour was low [27].

In 1998, a study reported on 156 female sexual assault survivors with nightmares and PTSD who manifested symptoms indicating a high potential for sleep apnea [28]. The main finding showed their Pittsburgh sleep quality index [29] global scores exceeded values for patients with depression, insomnia or hypersomnia alone. These data suggested a greater degree of sleep disturbance complexity not fully explained by PTSD.

We speculated this complexity was due to intrinsic physiological sleep disorders disguised as posttraumatic insomnia [30]. For example, 52% of sexual assault survivors suffering from PTSD reported the combination of snoring and daytime sleepiness, which met screening criteria to test for sleep apnea [31]. There was also a potential for either or both sleep breathing and sleep movement disorders among these sexual assault survivors who presented with nightmares, insomnia and posttraumatic stress symptoms. When women with potential physical sleep disorders were compared to those without such symptoms, the presumptive sleep disorders' group correlated significantly with worse PTSD symptom severity [32]. This raised the question of whether or not treatment of physiological sleep disorders might decrease PTSD severity [32].

Also in 1998, Lavie and colleagues reported a controlled study on awakening thresholds in PTSD patients. Among the 12 war veterans in the PTSD group, five exhibited objective evidence for moderate sleep apnea [33], while four of 12 controls also had OSA though of lesser intensity. Lavie and colleagues speculated on whether changes in sleep depth in PTSD patients might increase susceptibility to sleep breathing events, and then expanded this theory by providing a precise pathway wherein anxiety patients might hyperventilate during sleep to produce hypocapnia—a proven risk for triggering apneas [5].

Finally in 1998, a single case report described sleep apnea in a PTSD patient [34], who eliminated PTSD symptoms when treated with positive airway pressure therapy (PAP-T), the gold standard SDB treatment [35]. Two years later, Engdahl reported improvements in psychiatric distress with regular use of PAP-T among four veterans with comorbid PTSD and SDB [36].

From 2000 to 2002, six articles were published on the specific topic of SDB and PTSD, developing hypotheses as well as presenting key objective data. Among the samples of female sexual assault survivors with nightmares and posttraumatic stress symptoms or disorders, the main findings included:

- sleep breathing symptoms were surprisingly common, often present in greater than 50% of a sample [32,37–39];
- presumptively diagnosed sleep breathing disorders presented more like psychiatric-related insomnia rather than classic sleep apnea [32,37,38];
- objectively diagnosed SDB or self-reported SDB symptoms were associated with worse psychiatric distress [37–39];
- and, treatment of sleep breathing disorders in PTSD patients was associated with decreases in nightmares, insomnia and posttraumatic stress [40].

Among this set of papers, the initial objective work used an advance respiratory sensor technology to test 44 consecutive crime victims with posttraumatic stress who sought treatment for nightmares and insomnia; 40 of 44 patients were diagnosed by polysomnography (PSG) with OSA or UARS [41]. From these results, we coined the term “complex insomnia” to describe patients with a primary complaint of insomnia who also suffer co-morbid and usually covert (to the patient and physician or therapist) SDB [41].

Barriers to diagnosis and recognition

While these earlier works were thought-provoking, three prevailing paradigms tended to dilute research on relationships between sleep breathing disorders and PTSD, and each appears to reflect a lack of recognition or application of new perspectives or technology to assess trauma survivors with sleep complaints [6]. Foremost among these barriers is the prevailing view that emphasizes the psychological or psychiatric aspects of PTSD [42–44]. Although there is considerable research on biological aspects of

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