

TRAUMA ASSOCIATED SLEEP DISORDER: A NEW NIGHTMARE OR A WRINKLE ON AN OLD FAVORITE?

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The emergence of a brand-new sleep disorder is a rare and beautiful thing. In many ways, it is like watching the birth of a baby... it is inspiring, labor intensive, full of future possibilities and usually messy as heck. It begins as a germ of an idea in the mind of one or a handful of people, growing slowly, being fed by an increasing number of observers and subsequent observations. Until one day, it is ready to hatch, set forth upon the world, only to then have some detractors argue that it is nothing new, but rather just a twist on an old disorder. Sometimes these exchanges can get quite messy and sometimes it is something so clear and obvious that the idea moves quickly and uncontested through its infancy, until it matures onto the pages of Principles and Practice of Sleep Medicine.

A few days ago, a paper came across my desk (ok, so do we really have papers and desks?... it was actually a link to a website,¹ and it came across my Facebook feed) about some interesting research coming out of Madigan Army Medical Center at Joint Base Lewis, McChord, Washington. Col. Vincent Mysliwicz, MD, a sleep researcher, had noticed some peculiar sleep behaviors from some military veterans of the Iraq and Afghanistan wars. These veterans usually (but not necessarily) suffered from Post-Traumatic Stress Disorder (PTSD) and had the typical nightmares that are associated with it. What made it unusual, was the emergence of dream enactment behaviors in some of these patients. "These behaviors were troubling", Col. Mysliwicz reported. "They'd strike out, scream, yell at their spouse and even run around their bed and at times hurt themselves or others." Then in 2013, Col. Mysliwicz came across one episode in his sleep laboratory. After establishing that this is something new, Col. Mysliwicz and colleagues labeled what they saw as "trauma associated sleep disorder," or TSD. They define TSD as "dream-enactment behaviors relating to trauma-related nightmares." Post-Traumatic Stress Disorder may or may not be present, but a precipitating stressful trauma and nightmares are cardinal features of TSD.²



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So, let's first discuss what this is NOT. It is not somnambulism or sleep walking. Sleep walking occurs during the deeper stages of sleep, not during rapid eye movement (REM) sleep, which is often associated with dreaming. The sleep walker does not recall dreaming and when studied in a sleep laboratory, these episodes arise out of N3 "slow wave" or "deep sleep." In essence, the sleep walker has trouble waking up from deep sleep, and is stuck in a limbo state between sleep and wakefulness.^{3,4} On the other hand, the symptomology of TSD not only requires the presence of dreams, but also those that are highly emotionally charged and associated with the precipitating trauma.

Trauma Associated Sleep Disorder is also not REM Sleep Behavior Disorder (RBD). The proposed etiology is vastly different from that of RBD. Most cases of RBD are proposed to arise from neuro-degenerative causes. Damage to portions of the brainstem can cause abnormalities in certain aspects of REM sleep. Of particular interest are manipulations that affect the regulation of muscle tone in REM sleep. Lesions of several regions in the pons and medulla can cause REM sleep to occur without the normal loss of muscle tone⁵. Simply put, that means it is damage to the brain itself that causes the movements in REM sleep. We know that there are brain mechanisms that "paralyze" the body during REM sleep that prevent us from acting out our dreams. In classic RBD, neuro-degeneration causes these mechanisms to break down, the protective paralysis then fails, and we enact our dreams. The presence of neuro-degenerative disorders has been shown to increase the risk of RBD by as much as 50 percent, including Parkinson's Disease and Multiple System Atrophy.⁶ RBD in people without these disorders could predict an increased risk of developing them in the future, predicting up to 80 percent of as-of-yet undiagnosed Parkinson's and Lewy Body Dementia.⁷

To be sure, not all RBD is due to underlying neurological disorders. Other factors that influence the intensity of REM sleep may also play a role in the genesis of RBD, including alcohol consumption or its withdrawal, strokes, brain tumors, sleep deprivation and medication use. In the case of alcohol or medication use, RBD may only be acute, rather than chronic as it is in most other cases.⁶ Certain medications, particularly some of the selective serotonin reuptake inhibitors (SSRIs) and selective noradrenergic reuptake inhibitors (SNRIs) appear to increase EMG tone during REM sleep, and may or may not precipitate or aggravate RBD.⁸

Trauma Associated Sleep Disorder is similar to RBD in its physical expression only, but the etiology may be completely

different. The theorized mechanism of action is psychogenic, rather than somatic; it is not necessarily related to the typical neurodegeneration seen in RBD, rather it may be due to the adrenaline dump from the very intense nightmares these TSD sufferers endure. It is proposed that trauma-related nightmares result in an intense “dump” of adrenaline, which in turn, may overwhelm the body's natural “paralyzing” mechanisms during REM sleep, resulting in behaviors emerging from the dream state. It isn't so much an organic break-down in the paralysis mechanisms themselves, as it is just an overwhelming tsunami of chemicals (likely the hormone adrenaline, but perhaps some involvement of the neurotransmitter noradrenaline as well), causing movement in REM sleep.² So, whereas TSD may mimic RBD in outward appearance, the underlying causes make it something new and exciting.

In a previous paper, first published in 2014, Col. Vincent Mysliwiec et al. reported on four case studies of young male military personnel suffering from this syndrome. The researchers noted their relevant medical history and ran sleep studies on them. Researchers found that these behaviors ranged from vocalizations and somnambulism to combative behaviors that even injured bed partners. Nightmares were replays of the patient's traumatic experiences. All patients had REM without atonia during polysomnography; one of the four patients had an episode of these behaviors and a nightmare, captured during a REM period on the PSG². Subsequent treatment included the use of the antiadrenergic medication Prazosin. Although Prazosin is typically used to treat high blood pressure or benign prostatic hyperplasia⁹ it was chosen as therapy because it also blocks some of the effects of adrenaline.¹⁰ RBD has been previously associated with organic loss of noradrenergic neurons of the locus ceruleus and/or perilocus ceruleus, whereas PTSD appears to be characterized by hyper-adrenergic function of these areas; the overproduction of adrenaline or the “adrenaline dump” discussed earlier. Blockade of the hyper-adrenergic function is thought to be the primary mechanism of action of Prazosin. The researchers predicted that if the proposed “adrenaline dump” model is correct, then this class of medication would reduce the amount of adrenaline released, immediately improving the REM associated movements in TSD. Indeed, researchers showed improved REM related behaviors and nightmares in all patients. Furthermore, patients 1 and 4 reported a return of REM behaviors and nightmares upon discontinuation of Prazosin use.²

All this is new and exciting, but it is sure to have some outright detractors as well, or at least some healthy skeptics and doubters.

As we have already seen, RBD is a disorder that is usually associated with organic, neurodegenerative causes, mostly affecting certain areas of the brain that regulate muscle tone (or lack thereof) in REM sleep. But there are other, less common causes, not related to neurological disorders. Most people readily agree that causes of RBD are already multifactorial in nature, there is no homogeneous etiology. Geneses range from neurodegenerative processes to alcoholism, drug effects, brain trauma or even simple sleep deprivation. Could a sudden and overwhelming release of adrenaline during REM sleep just be another factor to be added to this list? Do we really need to add a whole new sleep disorder, or will tweaking the existing list of known RBD causes suffice? Sometimes the KISS policy (Keep It Short and Simple) makes for the best policy.

This theory is ripe for new and fresh research. Questions going forward that must be answered include whether TSD predicts neuro-degenerative disorders, as RBD does? Does long-term unresolved TSD result in permanent damage to the affected brain areas? Do REM behaviors resolve after long-term remission of PTSD / nightmares? Is psychological counseling and treatment as effective as Prazosin in decreasing or eliminating REM enactment behaviors and associated nightmares? Is TSD present in other traumatic but non-combat related life experiences? Is TSD a seizure in sleep or a short-lived fugue state? Although seizures are known to mimic RBD-like activity,^{11,12} the medical history of TSD patients suggest they are not involved. However, a simple EEG test should suffice to eliminate this possibility.

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