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COMMENTARY

The diagnostic challenge of dream-enactment behaviors

Commentary on Baltzan M, Yao C, Rizzo D, Postuma R. Dream enactment behavior: review for the clinician. *J Clin Sleep Med*. 2020; 16(11):1949–1969. doi:10.5664/jcsm.8734; and Barone DA. Dream enactment behavior—a real nightmare: a review of post-traumatic stress disorder, REM sleep behavior disorder, and trauma-associated sleep disorder. *J Clin Sleep Med*. 2020;16(11): 1943–1948. doi:10.5664/jcsm.8758

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Dream-enactment behaviors (DEBs) are purposeful-appearing movements and/or vocalizations that are presumed to arise from rapid eye movement (REM) sleep given their deliberate character. DEB, the prototypical clinical manifestation of REM sleep behavior disorder (RBD), is distinctly relevant because RBD is a robust biomarker for subsequent development of an α synucleinopathy. Although RBD is frequently the primary diagnostic consideration, there are other etiologies as well as mimickers of DEB, which routine polysomnography may not identify. Given the nature and complexity of DEB with the patient's lack of awareness during the clinical events, a thorough history and examination are paramount to establish context. Careful questioning of the bed partner is an essential step in identifying the probable diagnosis and developing an appropriately tailored workup. In this issue of the *Journal of Clinical Sleep Medicine*, Baltzan et al[1] provide a meticulous examination of the causes, associations, and proposed pathophysiologic mechanisms of DEB, which serves as an excellent reference.

The authors deliver a brilliant review emphasizing the proposed mechanisms at play, which provides a more intimate understanding of the fundamental processes driving DEB. Importantly, they shed light on situations when DEB may be considered normal, which is rarely discussed elsewhere. For example, brief and episodic dream-related motor activity is commonly reported in otherwise normal postpartum women. The proposed pathophysiologic mechanism is that the highly intense dream content momentarily overrides REM sleep inhibition of the motor neuron. This mechanism is further potentiated by sleep disruption, which is a common occurrence during the postpartum period. Similarly, posttraumatic stress disorder (PTSD) with DEB is thought to be mediated by heightened dream content, overwhelming motor neuron inhibition. Baltzan et al report on an interesting hypothesis of genetic predisposition, as proposed by Cartwright.[2] The threshold at which dream intensity overwhelms the atonia neural circuitry is perhaps genetically determined, which could explain why not all patients with PTSD and traumatic experiences exhibit DEB.

There were some notable gaps in this review. When managing patients with RBD, tolerance issues and suboptimal responses with clonazepam and melatonin prompt clinicians to consider alternative medications. Pharmacological agents such as pramipexole, rotigotine, acetylcholine esterase inhibitors such as donepezil, and rivastigmine, to name a few, can be tried in selective cases depending on the clinical context.[3–5] When examining behaviors that can be confused with dream enactment, the authors do not discuss malingering and expanded as periodic limb movements in sleep.[6,7] We applaud the exhaustive examination and synthesis required to produce this outstanding review of DEB for the clinician.

Also found in this issue of the *Journal of Clinical Sleep Medicine* is Dr Barone's focused review on DEB in PTSD, RBD, and the proposed parasomnia, trauma-associated sleep disorder (TASD).[8] The important question raised in this review is whether TASD is a subtype of RBD or a distinct sleep disorder. This article provides an examination of 3 disorders exhibiting overlapping features. Increased electromyographic activity during REM sleep has been reported in patients with PTSD.[9] Furthermore, RBD and TASD both demonstrate similar REM sleep without atonia,[10] and PTSD has been linked to RBD.[11] An association between PTSD and RBD was illustrated in a large prospective study of 394 veterans in the absence of antidepressant usage.[12] In addition, there is literature linking the pathology observed with PTSD of locus ceruleus and peri-locus ceruleus, which is similar to the pathophysiologic process observed in RBD.[13] Hyperactive locus ceruleus and peri-locus ceruleus regions due to trauma are thought to be responsible for REM sleep without atonia in TASD. The overdrive phenomenon is the etiology proposed for DEB in TASD.[8]

Several features distinguish TASD from RBD, as reported in this review. In TASD, there is an inciting traumatic experience that becomes incorporated into the DEB. This is somewhat analogous to a flashback experienced in PTSD but is

characterized by motor behavior/vocalization rather than dream imagery.[14] Polysomnographic demonstration of sympathetic activation during REM sleep is a unique feature of TASD.[14] Hence, adrenergic receptor blockade with prazosin is effective in TASD as opposed to RBD.

Barone appropriately acknowledged the need for longitudinal studies in patients with TASD and RBD related to selective serotonin reuptake inhibitors to determine their risk of synucleinopathy phenoconversion. These prospective studies can provide clinical guidance for patient management and offer insight into the pathophysiologic mechanisms. Evidence-based treatments are needed for symptomatic management of these parasomnias and identifying a neuroprotective agent for patients destined to develop a synucleinopathy. Perhaps a different marker of increased sympathetic activity during REM sleep can be identified to distinguish patients with TASD from other causes of DEB. Refinement and stratification of the "inciting traumatic experience" observed in TASD into those associated with psychological trauma with or without traumatic brain injury may help advance understanding of the pathophysiology of TASD and its possible association with RBD.

CITATION

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DISCLOSURE STATEMENT

The authors report no conflicts of interest.