# Pharmacological Treatment of Nightmares in Post-Traumatic Stress Disorder: Evidence, Limitations, and Clinical Considerations

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## Abstract

Nightmares constitute a major symptom of post-traumatic stress disorder (PTSD) and are linked to distress, impaired sleep quality, psychiatric comorbidity, and increased suicide risk. The neurobiological mechanisms underlying PTSD-related nightmares only partially overlap with those of insomnia, signaling a need for specific therapeutic approaches. This review summarizes the current evidence on pharmacological treatments for nightmares associated with PTSD, focusing on efficacy, tolerability, and quality of evidence. A narrative review of the literature was carried out using PubMed and Google Scholar for publications up to 1 July 2025**.** English-language studies of pharmacological interventions focused on reducing nightmare frequency or severity in adults with PTSD were included. Randomized controlled trials (RCTs), observational studies (including open-label studies and chart reviews), case series, and systematic reviews and meta-analyses were considered. Efficacy, adherence, and adverse effects were evaluated. Alpha-1 adrenergic antagonists, particularly prazosin, are the most extensively studied agents and have demonstrated modest benefit in multiple trials and meta-analyses, although large RCTs have produced mixed results. Other adrenergic agents (such as doxazosin and clonidine), cannabinoid-based treatments (such as nabilone), sedating antidepressants, and second-generation antipsychotics have shown preliminary or adjunctive benefits, primarily in small or methodologically limited studies. Adverse effects, including hypotension, metabolic complications, and daytime sedation, restrict the widespread use of several agents. No pharmacological treatment has demonstrated unequivocal efficacy for PTSD-related nightmares in large, high-quality trials. Current evidence supports cautious, individualized use of selected agents, with careful risk–benefit assessment. Large, well-designed RCTs are needed to establish clear treatment recommendations.

**Keywords:** post-traumatic stress disorder; nightmares; prazosin; drug therapy; veterans; military personnel

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## INTRODUCTION

Post-traumatic stress disorder (PTSD) is a clinical syndrome that may develop following exposure to actual or threatened death, serious injury, or sexual violence. It is characterized by four symptom clusters: intrusive symptoms, avoidance, negative alterations in cognition and mood, and alterations in arousal and reactivity [1]. Nightmares are classified within the intrusion cluster, and their underlying mechanisms remain only partially understood. Clinically, nightmares are among the most distressing symptoms of PTSD and present a significant therapeutic challenge.

Although nightmares occur during sleep and may be accompanied by comorbid insomnia, accumulating evidence indicates that PTSD-related nightmares are neurobiologically distinct, at least in part, from insomnia. Neuroimaging and polysomnographic studies suggest that nightmares are primarily associated with dysregulation of neural circuits involved in fear memory, emotional processing, and autonomic arousal. Central mechanisms include amygdala hyperactivity, impaired inhibitory control by the medial prefrontal cortex (mPFC), and hippocampal dysfunction, resulting in recurrent re-experiencing of traumatic memories during sleep, particularly in rapid eye movement (REM) sleep [2,3]. In contrast, insomnia in PTSD is more closely related to non-REM sleep disruption, reduced frontal delta activity, and generalized hyperarousal. This evidence supports the conceptualization of nightmares and insomnia as partially independent symptoms with distinct biological substrates, suggesting that they may require different treatment strategies [4].

PTSD-related nightmares are strongly associated with psychiatric comorbidity, including anxiety and depressive disorders, increased feelings of hopelessness, and impaired daytime functioning [5]. They are also linked to other sleep disturbances such as sleep avoidance and insomnia [6], and could worsen daytime PTSD symptoms [7]. Nightmares have been identified as an independent risk factor for suicidality, with chronic and persistent nightmares conferring a particularly elevated risk for suicidal ideation, attempts, and non-suicidal self-injury [5,8]. These associations underline the importance of early and effective intervention. However, the role of pharmacotherapy remains unclear, as trials of the most extensively studied agent, prazosin, have produced conflicting results. Given that much of the available evidence is derived from military veterans and service members, this issue is directly relevant to military medicine. This review summarizes the current evidence on pharmacological treatments for PTSD-related nightmares in adults, emphasizing efficacy, tolerability, and quality of evidence.

## METHODS

This narrative literature review used PubMed and Google Scholar to identify studies published up to 1 July 2025. The search included combinations of the following keywords: post-traumatic stress disorder, PTSD, acute stress disorder, ASD, nightmares, pharmacological treatment, drug therapy, prazosin, doxazosin, clonidine, terazosin, cyproheptadine, and nightmare treatment.

Studies were eligible for inclusion if published in English, involving adults (≥18 years) diagnosed with PTSD, and evaluating the efficacy of pharmacological interventions for trauma-related nightmares. Randomized controlled trials (RCTs), prospective and retrospective observational studies (including open-label studies and chart reviews), case series, and systematic reviews and meta-analyses were considered. Studies focusing exclusively on pediatric populations, non-pharmacological interventions, conference abstracts, or articles without available full text were excluded. The review draws on 24 primary clinical studies (10 RCTs and 14 open-label, observational, case-series, or case-report studies), which are summarized in the Results and Table 1; systematic reviews, meta-analyses, clinical practice guidelines, and background sources were used for context.

Titles and abstracts were screened for relevance, followed by full-text assessment of potentially eligible articles. Studies were selected based on their relevance to the efficacy, tolerability, and adherence associated with pharmacological treatments for PTSD-related nightmares. Reference lists of relevant articles were also reviewed to identify additional eligible studies. The level of evidence for each agent was graded according to the Oxford Center for Evidence-Based Medicine (CEBM) 2009 levels of evidence (Table 1).

## RESULTS

Table 1 summarizes the pharmacological agents most frequently reported in the literature, their proposed mechanisms of action, major adverse effects, study designs, and principal findings. The following sections discuss these agents in more detail, organized by pharmacological group.

| Medication | Mechanism of action | Main adverse effects | Study design and population | Main findings | Ref. | Level* |
|---|---|---|---|---|---|---|
| **Adrenergic agents** | | | | | | |
| Prazosin | α1-adrenergic antagonist | Hypotension, dizziness, fatigue, syncope | Multiple RCTs and meta-analyses; veterans and civilians | Reduction in nightmare frequency and intensity; modest efficacy; mixed results in large trials | 9–18, 21, 36 | 1a |
| Doxazosin | α1-adrenergic antagonist | Hypotension, dizziness, fatigue | Case reports, chart review, small crossover RCT; PTSD | Mixed findings; no significant benefit in RCTs (2024 NMA); less extensively studied than prazosin | 18, 21–24 | 2b |
| Clonidine | Central α2-adrenergic agonist | Hypotension, dry mouth, fatigue | Retrospective chart review; veterans with PTSD | Preliminary positive findings; additional controlled studies needed | 25 | 4 |
| **Cannabinoids** | | | | | | |
| Nabilone | Synthetic cannabinoid (CB1 agonist) | Dizziness, dry mouth, sedation, mood changes | Single RCT; PTSD | Preliminary positive findings in small studies; evidence limited to one RCT; not approved for PTSD | 26, 27 | 2b |
| **Histamine receptor antagonists** | | | | | | |
| Hydroxyzine | H1 antihistamine | Sedation, dry mouth, confusion, headache | Single RCT; PTSD | Positive effect on nightmares; further studies required | 36 | 1b |
| Cyproheptadine | Serotonin antagonist and antihistamine | Sedation, weight gain, dry mouth | Case series, open-label trial, one RCT; PTSD | No benefit in open-label or placebo-controlled trials; benefit limited to case reports | 19, 21, 34, 35 | 2b |
| **Second-generation antipsychotics** | | | | | | |
| Risperidone | Second-generation antipsychotic | Weight gain, sedation, metabolic syndrome, extrapyramidal symptoms | One adjunctive RCT (n = 267); case series; veterans with PTSD | Small improvement in sleep quality and nightmares in one RCT; no clear benefit in 2024 NMA; not first-line | 18, 19, 28, 29 | 1b |
| Olanzapine | Second-generation antipsychotic | Weight gain, sedation, metabolic syndrome | Case series; two small RCTs; PTSD | Mixed results in two small RCTs; not recommended as first-line treatment | 19, 20, 25, 30–32 | 2b |
| Quetiapine | Second-generation antipsychotic | Sedation, weight gain, metabolic syndrome | Historical cohort (chart review); veterans with PTSD | Similar to prazosin in one cohort but more discontinuations; no significant benefit in 2024 NMA | 18, 20, 33 | 2b |
| **Sedating antidepressants** | | | | | | |
| Mirtazapine | Serotonin and noradrenergic antagonist (NaSSA) | Sedation, weight gain, dry mouth | Case series; PTSD | Benefit reported in case series; no significant benefit in RCTs (2024 NMA) | 18, 37 | 4 |
| Trazodone | Serotonin antagonist and α1-blocker (SARI) | Sedation, dry mouth, dizziness | Retrospective survey; veterans with PTSD | Preliminary positive findings; not approved for PTSD | 19, 39 | 4 |

**Table 1. Pharmacologic agents evaluated for the treatment of PTSD-related nightmares.** Agents are grouped by drug class. For each agent, the main mechanism, adverse effects, evidence base, key findings, supporting references, and level of evidence are shown. PTSD, post-traumatic stress disorder; RCT, randomized controlled trial; CB1, cannabinoid receptor type 1; NaSSA, noradrenergic and specific serotonergic antidepressant; SARI, serotonin antagonist and reuptake inhibitor; CEBM, Center for Evidence-Based Medicine. *Oxford Center for Evidence-Based Medicine (CEBM) 2009 levels of evidence. NMA, network meta-analysis. Reference numbers refer to the reference list.

**Adrenergic agents**. α1-Adrenergic antagonists, particularly prazosin, have been the most widely used pharmacological options for the treatment of PTSD-related nightmares [9]. The most extensively studied agent in this category is prazosin, an α1-adrenergic receptor antagonist originally approved for hypertension and benign prostatic hyperplasia. An early open-label report of four Vietnam War veterans described a marked reduction in the frequency and severity of nightmares with prazosin [10]. Subsequent small-scale studies in both military and civilian populations indicated possible efficacy [11,12]. A larger placebo-controlled parallel-group trial later supported its clinical use [13], and guidelines then included prazosin [14]. However, a large multicenter, double-blind RCT in 304 veterans with chronic PTSD found no significant benefit of prazosin compared with placebo with respect to global PTSD symptoms, sleep quality, or nightmare severity over 26 weeks [15]. Two subsequent meta-analyses including 429 and 575 patients, respectively, nevertheless reported a statistically significant reduction in nightmare-related symptoms associated with prazosin [16,17]. The most comprehensive synthesis to date, a 2024 network meta-analysis of 99 RCTs (10,481 participants), found prazosin to be the only agent significantly more effective than placebo for nightmares (20 RCTs, 1,398 participants; standardized mean difference −0.44, 95% CI −0.84 to −0.04), with very low to moderate confidence in the estimates [18]. In 2018, the American Academy of Sleep Medicine downgraded its recommendation for prazosin [19]. The current US Department of Veterans Affairs and Department of Defense (VA/DoD) guideline for the management of PTSD makes a weak recommendation for prazosin for the treatment of PTSD-related nightmares [20].

Doxazosin, another α1-adrenergic antagonist with a longer half-life than prazosin, has been studied less extensively. Reviews and case reports describe early clinical experience [21,22]. A retrospective chart review of 51 patients suggested potential benefit, although the lack of a control group and sample heterogeneity limit interpretability [23]. A small double-blind, placebo-controlled crossover pilot trial in veterans (n = 8) found a significant reduction in self-rated PTSD symptoms but no significant change in the total Clinician-Administered PTSD Scale (CAPS) score; nightmares were not reported as a separate outcome [24]. In the 2024 network meta-analysis, the two available RCTs of doxazosin showed no significant benefit over placebo [18]. Researchers have also investigated clonidine, a centrally acting α2-adrenergic agonist. A retrospective chart review in veterans (n = 79) reported partial improvement in 63–72% of patients, with clinically significant improvement observed in approximately half [25].

**Cannabinoid-based agents**. Another proposed mechanism involves modulating the endocannabinoid system. Nabilone, a synthetic cannabinoid and CB1 receptor agonist, has shown preliminary efficacy. A small randomized, double-blind, placebo-controlled crossover trial in Canadian military personnel with treatment-resistant PTSD-related nightmares (n = 10) showed significant improvement [26]. A retrospective chart review of 47 patients reported improvement in nightmare symptoms in approximately 72% of cases [27]. However, the evidence base remains limited, and the VA/DoD guideline recommends against the use of cannabinoids for PTSD [20].

**Second-generation antipsychotics**. Evidence regarding the use of second-generation antipsychotics for PTSD-related nightmares remains limited [18]. These agents are not recommended as first-line treatments and are generally considered adjunctive options, particularly for patients with comorbid psychotic symptoms, severe flashbacks, or bipolar disorder [20]. Risperidone, which acts as an antagonist at serotonin, dopamine, and α-adrenergic receptors, has been studied mainly as adjunctive therapy. In a 24-week multicenter RCT in 267 veterans with antidepressant-resistant, military-related PTSD, adjunctive risperidone produced small but statistically significant improvements in sleep quality (Pittsburgh Sleep Quality Index) and nightmare severity (CAPS) compared with placebo [28]. A case series in veterans also reported benefit [29]. Olanzapine, a dopamine D2 and serotonin 5-HT2C antagonist, can increase slow-wave sleep and suppress REM sleep [19]. Small case series and retrospective reviews have reported rapid improvement or remission of nightmares when olanzapine was combined with selective serotonin reuptake inhibitors (SSRIs) [25,30]. Two small randomized trials produced mixed results: one demonstrated advantage over placebo as adjunctive

therapy [31], while the other found no benefit as monotherapy [32]. Most of these studies have methodological limitations and small sample sizes. Quetiapine is used for its sedative properties in PTSD. A historical cohort study using retrospective chart review compared quetiapine and prazosin in 237 veterans and found similar reductions in nocturnal symptoms; however, quetiapine discontinuation due to adverse effects was more frequent (35% vs. 18%) [33]. In the 2024 network meta-analysis, risperidone and quetiapine were associated with a high risk of somnolence without significant therapeutic benefit [18].

**Histamine receptor antagonists**. Cyproheptadine, an antihistamine with antiserotonergic properties that suppresses REM sleep, has yielded inconsistent results [21]. Although a review has reported benefit [21], an open-label trial in veterans (n = 36; efficacy analysis in n = 16) found no significant improvement in nightmare-related outcomes at doses of 4–8 mg/day [34]. A double-blind, placebo-controlled RCT in combat veterans (n = 69) likewise found no benefit: nightmare severity and sleep quality were nonsignificantly worse with cyproheptadine than with placebo [35]. Despite the limited evidence, the American Academy of Sleep Medicine still lists cyproheptadine as a possible adjunctive treatment [19]. A randomized placebo-controlled trial comparing prazosin and hydroxyzine (maximum dose 100 mg) over 8 weeks in 100 patients with PTSD found that both agents improved nightmares relative to placebo, with prazosin demonstrating greater efficacy [36].

**Sedating antidepressants**. Evidence for noradrenergic and specific serotonergic antidepressants (NaSSAs), such as mirtazapine, remains limited. Observational evidence suggests that combination therapy with SSRIs may reduce nightmare frequency and intensity, with improvement rates of up to 75% reported in a large case series [37]. However, randomized evidence is scarce, and the 2024 network meta-analysis found no significant benefit of mirtazapine over placebo [18]. Mianserin, a tetracyclic antidepressant with a similar receptor profile, was added at 10–30 mg for sleep difficulties to sertraline and trauma-focused cognitive behavioral therapy in an uncontrolled follow-up study of refugees (n = 85). Sleep improved markedly, but nightmares were not assessed separately, and the improvement was not associated with mianserin use [38].

Serotonin antagonist and reuptake inhibitors (SARIs), particularly trazodone, are frequently prescribed for insomnia and nightmares due to their sedative properties [19]. A survey of 74 veterans reported reductions in nightmare frequency and improved sleep quality in 72% of patients receiving trazodone at doses of 50–200 mg nightly [39]. Adverse effects were common, occurring in approximately 60% of patients, and included daytime sedation, dizziness, headache, and, in rare cases, priapism or worsening nightmares.

Nefazodone has shown modest benefit in several open-label trials, with reported reductions of approximately 30% in nightmare frequency; however, concerns regarding rare but severe hepatotoxicity limit its clinical use [40].

## DISCUSSION

The management of sleep disturbances in PTSD presents a significant clinical challenge, with nightmares representing some of the most prominent and difficult-to-treat symptoms. These disturbances can substantially impair quality of life and adversely affect broader aspects of physical and mental health.

Overall, despite a substantial number of published studies, no pharmacological agent has demonstrated definitive efficacy supported by large-scale, high-quality randomized trials. Small sample sizes and methodological weaknesses limit most studies. Consistent with these limitations, the 2024 network meta-analysis found that most individual comparisons were underpowered and that confidence in the effect estimates was very low to moderate [18]. Even prazosin, the most extensively studied medication in this area, has produced inconsistent results in a large trial [15]. Cannabinoid-based agents such as nabilone have shown positive preliminary findings, but small study populations limit the evidence. Second-generation antipsychotics and sedating antidepressants have shown inconsistent benefit, mainly in small or uncontrolled studies, with no significant benefit for mirtazapine, risperidone, or quetiapine in the 2024 network meta-analysis [18], and considerable adverse effects, including metabolic complications and daytime sedation, constrain their use.

Despite the limited empirical foundation, several of these medications appear in clinical guidelines and are supported by meta-analytic evidence. Recent clinical practice guidelines, including the 2023 VA/DoD Clinical Practice Guideline [20], distinguish between pharmacological treatment of broader PTSD symptoms and pharmacological treatment of PTSD-related nightmares. Specifically, these guidelines recommend prazosin as a treatment option for trauma-related nightmares in selected patients, but not for the treatment of overall PTSD symptoms. This distinction reflects the mixed evidence regarding prazosin's effects on global PTSD outcomes while considering its possible benefit for nightmare reduction in certain individuals. Consequently, prazosin should be considered a reasonable option for selected patients with distressing PTSD-related nightmares, rather than a universally recommended first-line pharmacological treatment.

A cautious clinical approach to treatment is warranted. Sedating agents may be considered for sleep disturbances, with the potential secondary benefit of reducing nightmares; however, clinicians should thoroughly assess metabolic and other adverse risks.

**Limitations.** This review has several limitations. It was a narrative review rather than a systematic review; the search was limited to PubMed and Google Scholar and to English-language publications, and study selection was not conducted in duplicate or independently, so selection bias cannot be excluded. No formal risk-of-bias assessment or quantitative synthesis was performed beyond grading levels of evidence, and the included studies varied widely in design, population, outcome measures, and duration, which limits direct comparison between agents.

In conclusion, conducting clinical trials in PTSD populations presents innate challenges, including symptom fluctuation, natural symptom attenuation over time, and difficulties in recruiting patients during acute phases. These problems are notably evident in cases of combat-related PTSD. This review highlights the strong need for large, methodologically robust research programs to evaluate pharmacological treatments for PTSD in general and nightmares specifically. The need for such research is additionally emphasized by the substantial side-effect burden associated with many commonly used medications in this population.

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