

REVIEW ARTICLE

# Nightmares and their treatment options

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## Abstract

Nightmares are dream experiences accompanied by severe anxiety or fear, they are an issue in both the healthy population and patients with numerous mental disorders. Themes of nightmares may be related to the future, or may be related to past events, with traumatic, unresolved or inadequately processed events being recalled during nightmares. In case of nightmares related to past events, it is generally appropriate to return to these events during therapy or consider behavioral changes. In case of nightmares regarding the future, therapy should focus on improving patients' coping skills. The goal of treatment is not merely to "improve sleep". There are several possible therapeutic approaches to be used in the treatment of nightmares in PTSD. As for psychotropic drugs, prazosin was assessed, with a positive effect on sleep architecture. The most recommended psychotherapeutic approach is imagery rehearsal therapy (IRT), using the principle of creating a new dream scenario that is satisfactory for the patient and its imagery rehearsal.

## INTRODUCTION

Nightmares are dream experiences accompanied by severe anxiety or fear. They belong to parasomnias. A recollection of dream content is vivid and sometimes detailed. The usual topics are threats to safety, life (one's own or someone else's, often a close person's) or self-esteem. These terrifying or scary topics repeatedly occur in nightmares. In a typical episode, vegetative symptoms develop. Verbal behavior or body movements may or may not be present. After waking from a terrifying dream, individuals are rapidly oriented and alert. They are able to fully communicate with those around them and to retell the dream, either immediately on awakening or in the morning. The occurrence

of nightmares may not be associated with feelings of sleep deprivation or fatigue on the following day.

From time immemorial, nightmares have been paid attention to. They were mentioned as early as in the Bible. Frequently, they were perceived as messages to the future and therefore interpreted in various ways. The first medical study on nightmares was published by John Bond in 1753. According to his theory, nightmares are caused by unknown ghosts or spirits who lay upon a person to constrict the chest and complicate breathing, leading to a sense of suffocation, crushing or sexual violence (Stein 2010).

From an evolutionary point of view, nightmares may have a protective warning meaning for an individual. If a person experiences a traumatic event, it is

important for him or her to be careful in the future until he or she is sure that the event will not repeat or that he or she will be able to manage it. Such increased watchfulness may be reflected in the quality of sleep, being not sufficiently deep and frequently interrupted by minimal external stimuli that did not wake the individual before. Thus, dreams may include nightmares reminding the person of the experienced event that are remembered even after awakening. In all cultures, dreams have traditionally been associated with expecting messages to the future. These were either interpreted by someone competent (e.g. the Biblical Joseph) or interpretations were compiled in the form of dream books. In the case of a positive prediction, these interpretations could be reassuring. In the case of a negative prediction, they could lead to preparation for a threat, its acceptance and coping with the expected fate.

#### Diagnosing nightmares

In the DSM-III, nightmares were categorized as mental disorders in the group of sleep disturbances (DSM-III, 1980). They were similarly classified in the ICD-10 (1992). In the latest valid American classification, DSM-IV-TR, the criteria for diagnosing nightmares are as follows (Tasman *et al* 2003):

- Repeated awakenings from the major sleep period or naps with detailed recall of extended and extremely frightening dreams, usually involving threats to survival, security, or self-esteem. The awakenings generally occur during the second half of the sleep period.
- On awakening from the frightening dreams, the person rapidly becomes oriented and alert (in contrast to the confusion and disorientation seen in sleep terror and some forms of epilepsy).
- The dream experience, or the sleep disturbance resulting from the awakening, causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
- Exclusion criterion: The nightmares do not occur exclusively during the course of another mental disorder (e.g. delirium, posttraumatic stress disorder – PTSD) and are not due to the direct physiological effects of a substance (e.g. a drug of abuse, a medication) or a general medical condition.

Nightmares may not be associated with other mental disorders. Nightmares may occur as early as in childhood. However, they are more frequent in children suffering from separation anxiety disorder (van der Kolk *et al* 1991) or following traumatic experience that has not been adequately processed (Pennebaker 1985). Similarly in adulthood, nightmares may occur separately. However, more frequently than in children, they are associated with other mental disorders. They are typical symptoms in PTSD but may be also seen in depression, bipolar disorder, generalized anxiety disorder and personality disorders, especially borderline, schizoid and

schizotypal personality disorders (Avidan & Zee 2006; Sadock *et al* 2009; Yudofsky & Hales 2009; Schredl *et al* 2012).

#### Nightmares in PTSD

More than 70% of war veterans and civilians with PTSD present with persistent and severe posttraumatic nightmares and sleep disturbances (Harvey *et al* 2003). PTSD nightmares remind patients of traumatic events in the past. While falling asleep, people may experience flashbacks of traumatic events, inducing severe anxiety and preventing them from falling asleep. These flashbacks may even take a form of pseudohallucinations. Also with respect to this fact, it is surprising that these issues have been dealt with by only few researchers. Ross *et al.* (1989) claim that sleep disturbances, namely the presence of nightmares, are the key symptoms of PTSD. Nightmares affect not only the sleep itself but also sleep-related behavior. Individuals having severe PTSD nightmares often develop fear from more sleep or falling asleep (Zayfert & DeViva 2004). Biological findings in PTSD reveal increased activity and reactivity of the autonomic nervous system, apparent from rapid heartbeat and blood pressure fluctuations but also abnormal sleep architecture (e.g. sleep fragmentation and prolonged sleep latency, generally reduced sleep time). When comparing nightmares in persons suffering from PTSD and in those without PTSD, nightmares are found to be more frequent in PTSD, occurring earlier in the sleep cycle, sticking to the topic more frequently and repeating a real event (Inman *et al* 1990). Nightmares may be associated with other types of problematic behavior (e.g. fearing going to bed, safety behavior before falling asleep, late-night binge-eating, sleeping in an armchair) (Spoormaker & Montgomery 2008). Some studies consider PTSD nightmares as the key feature predicting chronicity of sleep problems (Kobayashi *et al* 2008).

Relationship between PTSD severity and levels of sleep disturbances was studied by Germain *et al.* (2004). The authors studied 367 persons with PTSD lasting for 6 months to more than 30 years. The severity of PTSD was most significantly correlated with sleep disturbances. Nightmares mean not only the presence of terrifying dream experiences during sleep but there are other parameters of impaired sleep. Sleep deprivation gradually accumulates, with subsequent development of chronic stress with increased levels of inflammatory cytokines and sympathetic activation (McEwen 2006). Sleep disturbances subside after successful PTSD therapy (Spoormaker & Montgomery 2008; Germain *et al* 2008). However, the time relation between PTSD development, sleep disturbances and nightmare occurrence is unknown. Whether a traumatic experiences is followed by nightmares first and then PTSD develops, or there is a different time sequence (Koren *et al* 2002). Some studies showed that predictors of PTSD development may be polysomnographically confirmed sleep disturbances

and presence of nightmares (Mellman *et al* 2002; 2004). This would mean that sleep disturbances develop earlier and are not secondary manifestations of PTSD.

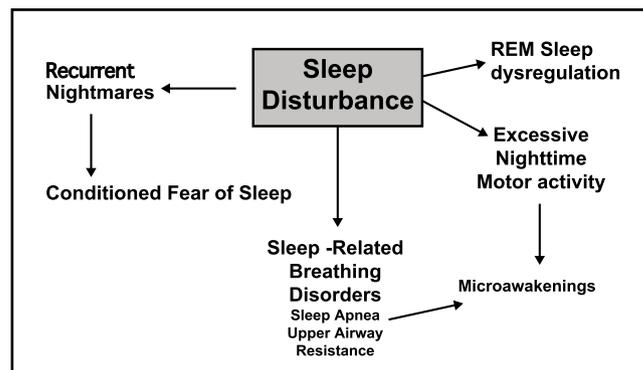
#### Neurobiological findings in PTSD sleep disturbance

Sleep is regulated by those areas of brain where PTSD-related changes were found as well. This suggests that stress response, arousal of the organism, fear conditioning and extinction, emotional memory and sleep may be significantly biologically interconnected. The most frequently reported finding is increased amygdala activity together with decreased activation of the medial prefrontal cortex (mPFC) and hippocampal dysfunction (Bremner *et al* 2005). These changes seem to be associated with fear conditioning (Etkin & Wager 2007; Bremner *et al* 2008). Imaging studies have shown that exposure to stimuli associated with trauma leads to excessive activation of the amygdala and decreased activation of the mPFC/ anterior cingulate cortex and hippocampus (Bremner *et al* 2008). The extent of these alterations correlates with clinical severity of PTSD symptoms (Moreya *et al* 2008) and seems to be increased in repeated exposure (Gilboa *et al* 2004). Persistent increased level of noradrenaline (NA), a monoamine playing a crucial role in fear conditioning and partly inhibiting hippocampal function, may support maintenance of PTSD symptoms including nightmares (Bremner *et al* 2005; Orr *et al* 2000). Excessive noradrenergic activity is assumed to mediate some of re-experiencing symptoms, heightened arousal and alertness and sleep disturbances in PTSD (Debiec & LeDoux 2006; Mellman *et al* 1995; Gottesmann 2008; Strawn & Geraciotti Jr 2008). Apart from the noradrenergic system, both the dopaminergic and serotonergic systems contribute to the development of nightmares (Pagel & Helfter 2003).

#### Polysomnography in PTSD

Changes in the brain's electrical activity occurring during sleep may be monitored with electroencephalography (EEG) recording. EEG supplemented with electrocardiography, electrooculography (measuring eye movements), electromyography of facial and lower extremity muscles, and monitoring of respiration rate, respiratory movements and oxygen saturation is referred to as polysomnography. Today, alterations in both electrical activity and metabolism may be monitored. Most frequently, changes in regional cerebral blood flow (rCBF) are monitored by single-photon emission computed tomography.

Unlike wakefulness, the REM stage of sleep is characterized by relatively increased rCBF in the amygdala, paralimbic and arousal controlling areas (the medial pontine tegmentum and thalamus), and reduced rCBF in the lateral prefrontal, parietal and sensory cortex. During the non-REM stage of sleep, rCBF is decreased in the pons, reticular formation, thalamus and association cortex, and increased in the sleep-promoting areas



**Fig. 1.** Sleep disturbances in PTSD (adapted from Lydiard & Hamner 2009)

such as the dorsal pontine tegmentum and basal forebrain (Nofzinger 2002). With the knowledge of activation and deactivation of various parts of the brain, non-REM sleep is considered a low activity status with a restorative function whereas REM sleep seems to be associated with learning, memory and emotional processing (Germain *et al* 2008).

Increasing knowledge, however, suggests that REM sleep disturbances play an important role in the development of PTSD (Mellman *et al* 2002; Gilboa *et al* 2004). When comparing PTSD patients with healthy controls, they were found to have more frequent transitions from REM to stage 1 sleep, awakenings during the REM stage of sleep, changes in REM sleep density and shorter REM episodes (Mellman *et al* 2002; Breslau *et al* 2004). This fragmentation of the REM stage may be due to excessive noradrenergic activity (Mellman *et al* 2004), premature termination of REM sleep or disturbances in the normal rhythm of cholinergic REM-on and aminergic REM-off signals (Germain *et al* 2008; Gottesmann 2008; Gilboa *et al* 2004). REM fragmentation may mimic REM deprivation with all behavioral and metabolic consequences (McEwen 2006).

Dreams occur in both the REM and non-REM stages of sleep. Those in REM sleep appear to be more emotionally charged than those in non-REM sleep. The sleep stage does not seem to determine the degree of emotional distress experienced during the nightmare, but it probably does account for the amount of visual imagery present (van der Kolk 1987).

Limb movements in sleep were monitored in a study by Ross *et al.* (1994). In a group of war veterans, lower limb motor activity was studied using electromyography of the anterior tibialis muscle. During REM sleep, a higher percentage of prolonged twitches was observed in PTSD subjects as compared with healthy controls. Patients with PTSD were more likely to have period limb movements during non-REM sleep (Mellman *et al* 1995).

In addition to abnormal lower limb movements, PTSD patients are known to have breathing problems. Most attention has been paid to sleep apnea syndrome

**Tab. 1.** REM fragmentation sleep disturbances in PTSD (adapted from Lydiard & Hamner 2009)

1. Increased REM density
2. More frequent, shortened REM episodes
3. More awakenings from REM sleep
4. More REM to stage 1 transitions
5. Shorter latency to move from REM to stage 1
6. More REM-preceded awakenings with panic, startle and physiological arousal

(Krakow *et al* 2004; Ocasio-Tascon *et al* 2006; Kobayashi *et al* 2007; Lamarche & De Koninck 2007). However, it is difficult to determine whether breathing problems during sleep result in or from PTSD, or whether they are generally related to sleep disturbances.

#### Sleep and attachment

Children having nightmares are most frequently comforted by their mothers. If children feel secure, nightmares are rare and often related to terrifying events. They may be more frequent after return from a hospital or summer camp. They are probably best solved by the mother's safe embrace and general calming of the child. Research has shown that individuals with anxious attachment in childhood had a higher frequency of REM dreams with aggression and self-denigrating themes than those with secure attachment (McNamara *et al* 2011). Although the relationship between nightmares and attachment in childhood has not yet been studied, it may be hypothesized that anxious attachment in childhood may predispose to nightmares. Similarly, individuals with anxious attachment in childhood are more vulnerable to PTSD if they experience traumatic events. According to van der Kolk *et al.* (1991), the probability that PTSD symptoms including nightmares resolve is not dependent on the history of trauma but rather on the ability to be provided security and calming from another person. The character of an individual's bonding with a close person reflects strategies learned to cope with negative influences. A more secure bond with a partner creates a safe haven that helps the victim regulate anxiety, sadness, anger and fear in a manner strengthening positive self-perception and the relationship. The safe haven with the partner help the victim solve, in a constructive way, emotionally charged re-experiencing symptoms such as nightmares, pervasive thoughts and memories (Kobak & Cole 1991).

#### Drug-induced nightmares

Nightmares may be induced by numerous psychopharmaceuticals as well as other drugs. Clinical studies or case reports have demonstrated the ability to induce nightmares in cholinergic agonists (donepezil, rivastigmine), beta blockers (atenolol, bisoprolol, propa-

nolol), noradrenergic agents (tramadol, methyl dopa), selective serotonin reuptake inhibitors (SSRIs; sertraline, fluoxetine, paroxetine), drugs possessing both antiserotonergic and antiadrenergic properties (risperidone, venlafaxine), dopaminergic agents (bupropion, levodopa, selegiline), amphetamine-like drugs (bethanidine, fenfluramine) and GABAergic agents (gabapentin, nitrazepam, zopiclone) (Pagel & Helfter 2003). Also abrupt discontinuation of drugs suppressing REM sleep (a dream-related stage) may lead to increased dreaming and nightmares.

#### **TREATMENT OF NIGHTMARES**

If we accept the hypothesis of an adaptive function of nightmares in an individual, it is reasonable to consider their significance for the patient's life before therapy is initiated. Nightmares related to the past should be eliminated by focusing either directly on nightmares or on their causes, i.e. traumatic events producing nightmares. In nightmares related to the future, it may be important to think about their significance for the patient and behavioral changes aimed at coping with feared situations in the future should be preferred to attempted elimination of nightmares. In particular, such changes should involve an increase in the ability to cope with life's problems in general.

For PTSD-related nightmares, the drugs of first choice are SSRIs (Stein *et al* 2006). This is true despite the fact that in studies, these drugs were shown to have only a minimal effect on sleep disturbances and no direct effect on nightmares (Maher *et al* 2006; Lydiard & Hamner 2009). However, if the other PTSD symptoms resolve during the treatment, nightmares are likely to be reduced or disappear. Unfortunately, this effect does not occur in all patients and nightmares may remain as one of residual symptoms that have to be dealt with by psychotherapy.

When considering PTSD therapy without the use of psychopharmaceuticals, the therapy of first choice should be cognitive behavioral therapy (CBT) with exposure therapy, shown to be effective in a few studies assessing the effect on sleep as well (Foa *et al* 1999; Foa 2006). In a study of 48 civilians with PTSD treated with CBT, a total of 24% of the subjects no longer met the DSM-IV-TR criteria and continued to have clinically significant sleep disturbances (primary insomnia) (Zayfert & DeViva 2004). Some of the patients benefited from additional insomnia-targeted CBT. One open-label study (Krakow *et al* 2001a) and one controlled study (Krakow *et al* 2001b) of subjects with PTSD-related nightmares and insomnia assessed the effectiveness of imagery rehearsal therapy (IRT). Although they showed significant improvement of sleep and PTSD outcome measures, some of the subjects continued to report insomnia, occasional nightmares and other PTSD residual symptoms even after completion of the studies.

### Pharmacotherapy of nightmares

For the treatment of nightmares, numerous psychopharmaceuticals with various mechanisms of effect have been tested. So far, no drugs have been found to ensure sleep without nightmares following their administration. At present, there is evidence about the effectiveness of prazosin in reducing PTSD-associated nightmares in both veterans (Raskind *et al* 2003, 2007) and civilians (Taylor *et al* 2008) in placebo-controlled studies. Prazosin is a centrally and peripherally acting alpha-1 adrenergic receptor antagonist. Originally, it was labeled to treat hypertension (in doses of up to 20 mg daily). It is also used off-label for benign prostatic hyperplasia (2 mg daily) and Raynaud's phenomenon (0.5–3 mg daily). In veterans with PTSD, doses of 10–15 mg daily were well tolerated (only some patients dropped out of the study due to orthostatic hypotension) (Raskind *et al* 2003, 2007). In a placebo-controlled study of 13 civilians, sleep criteria were also assessed. After a 1-week washout period, the subjects were divided into two arms (prazosin 3 mg at bedtime vs. placebo). When compared with placebo, prazosin therapy was more effective in improving sleep disturbances and reducing nightmares and was also associated with prolonged overall sleep time. Moreover, the overall REM sleep time as well as individual REM episodes were found to be longer (Taylor *et al* 2008). Clinical experiences have shown that to suppress symptoms of nightmares, 1 mg daily doses of prazosin administered for 1–2 weeks are sufficient. If ineffective, prazosin doses should be increased to 15–20 mg daily over several weeks and at the same time, hypotension should be monitored. The most frequent adverse effects observed in approximately 10% of cases are postural vertigo and sedation. In a small study, PTSD patients in whom nightmares significantly improved also decreased their response to traumatic stimuli during the day (Taylor *et al* 2006).

Apart from prazosin, other adrenergic drugs have been tested. Clonidine and guanfacine, alpha-2 adrenergic receptor agonists, were shown to be effective against PTSD symptoms in both children and adults (Boehnlein & Kinzie 2007; Strawn & Geraciotti Jr 2008). Also administration of propranolol, a beta adrenergic receptor antagonist, resulted in a mild decrease in the number of persons who had developed PTSD following traumatic events; however, its effect on sleep has not been sufficiently described (Pitman *et al* 2002).

Another tested drug is gabapentin, an anticonvulsant with anxiolytic, antinociceptive and sleep-inducing effects. It acts upon the noradrenergic, serotonergic and GABAergic systems and substance P (Taylor *et al* 1998). In their study, Hamner *et al.* (2001) reported an off-label effect of gabapentin (300–2100 mg daily) as an adjunct to existing pharmacotherapy in 30 veterans with PTSD. Its administration led to a decrease in the number of nightmares and sleep disturbances. Out of 30 veterans, 24 (77%) showed moderate or greater improvement in disturbed sleep and overall PTSD severity after adjunctive

therapy with gabapentin. Both gabapentin and pregabalin, another antiepileptic, affect reuptake and release of gamma-aminobutyric acid (GABA) (Lydiard 2003). Preliminary preclinical findings suggest that gabapentin, and probably also pregabalin, may be promising as an alternative or adjunctive drug to SSRIs in PTSD patients with persistent sleep disturbances. Additionally, other agents have been investigated, of which nefazodone, trazodone, mirtazapine, levomepromazine and quetiapine may be effective in the treatment of PTSD-related sleep disturbances (van Liempt *et al* 2006); however, their effect on nightmares has not been reported. Trazodone was tested in 4 patients with advanced cancer experiencing nightmares. In a half of them, nightmares subsided (Tanimukai *et al* 2013).

Antipsychotic drugs were also used for the treatment of nightmares, one of them being risperidone. In 10 acutely burned hospitalized patients, low doses of risperidone (0.5–2 mg at bedtime; average dose 1 mg) diminished nightmares as early as after 1 or 2 days of administration (Stanovic *et al* 2001). Olanzapine was tested as an adjunct to existing therapy in 5 patients with intractable nightmares. In all patients, nightmares improved rapidly (Jakovljevic *et al* 2003). Furthermore, aripiprazole was found to be effective in the treatment of nightmares in 5 patients with combat-related PTSD (Lambert 2006).

### Psychotherapy

At present, the most tested psychotherapeutic approach to the treatment of nightmares is CBT. Generally, CBT for sleep disorders is aimed at thorough education, working with dysfunctional thoughts and emotions, and the use of sleep diaries or logs. There are numerous approaches to the treatment of nightmares. Generally, they may be divided into direct and indirect. The first possible indirect approach is relaxation training.

#### Progressive deep muscle relaxation

One study was carried out on the effect of *progressive deep muscle relaxation* (PDMR) which is widely used in the treatment of other disorders as well. Its principle is induction of tension and subsequent muscle relaxation. Patients are asked to repeatedly tense and relax various muscle groups. In their study, Miller and DiPilato (1983) divided 32 women with nightmares into three groups. One was treated with PDMR, the second with systematic desensitization and subjects in the third group received no therapy. In 21 patients from the two groups receiving treatment, the frequency of nightmares decreased by 80%; in 12 of them, nightmares disappeared completely.

#### Recording nightmares

Another possible indirect approach is simple recording of nightmares. With this technique, patients just write down their nightmares without altering them or reading them repeatedly.

#### Exposure therapy

Another alternative is the use of direct approaches, one of which is *exposure therapy*. This technique utilizes the principle of gradually increasing exposure. Patients are asked to write down their own hierarchy of anxiety-causing dreams or situations. The usual way is to put situations on a scale from one to ten (but the number of steps may be higher). Then the patient himself or herself selects the step to start with (one of the lowest steps is usually recommended). He or she is exposed to the selected situation or dream and instructed to remain in that situation or recall the dream until anxiety attenuates. The patient is exposed to the same dream or situation repeatedly until the experienced anxiety is acceptable for him or her. Then another issue is dealt with in the same manner. When using this technique, it is important to compile the initial hierarchy adequately. The difficulty steps between individual tasks should not be great and should be evenly distributed. As in the case of other techniques, patient motivation is important in exposure therapy. The effectiveness of exposure therapy in the treatment of nightmares was confirmed in a randomized controlled study of 170 adults with nightmares carried out by Burges et al. (1998). The effect was studied at the end of 4-week therapy and 6 months later. The subjects were distributed into three groups, one undergoing exposure therapy, another using relaxation (both in the home setting, according to a manual) and the remaining one was on a waiting list. The best improvement but also the highest dropout rate were seen in the exposure therapy group.

#### Systematic desensitization

Another option is the use of *systematic desensitization*. Unlike exposure therapy, this technique is supplemented with education on anxiety and working with it. The effectiveness of systematic desensitization was investigated in the aforementioned study by Miller and DiPilato (1983). As already mentioned, patients in both treated groups (PDMR and systematic desensitization) decreased their frequency of nightmares. After a 15-week follow-up, there was no difference between the two groups. In the 25th week, however, there was a statistically significant difference in the decrease of nightmare intensity in favor of systematic desensitization. No recent study has been published on this issue. A 1992 study by Kellner et al. (1992) investigated the effectiveness of systematic desensitization as compared with rehearsal instructions. The first group was treated with one session of desensitization and instructed on how to practice it. The other group was explained how to change the nightmare and rehearse the new version. In both groups, there was a significant reduction in the frequency of nightmares at 7-months follow-up. There were no significant differences between the effects of the two techniques.

#### Imagery rehearsal therapy

At present, the most recommended approach is *imagery rehearsal therapy* (IRT) (Krakow et al 2001b). The

principle of the technique is to induce a nightmare in imagination after waking up. The patient should try to recall the dream with as many details as possible, including the emotional component. Subsequently, the patient should record the dream, preferably in written. The next task is to change the story. The topic, storyline, end or any other part may be changed so that the story sounds positive to the patient. Ideally, the story should be adapted to fit the patient's own ideas. The therapist should be a guide rather than the new story's author. It is not sufficient to experience the change only once. It is important for the patient to rehearse the new story. He or she may make another change in the story. The positive course of the dream is important, though. This is referred to as a cognitive shift. Krakow et al. (2001b) described the approach as follows: It is a group therapy in three sessions. In the first session, participants are explained two possible views of nightmares, either as a function only of traumatic exposure or as a function of both trauma and learned behaviors. Already at the end of the first session, participants practice pleasant imagery and learn cognitive-behavioral techniques for dealing with negative images. In the second session, the learned and individually practiced skills are used on a single, self-selected nightmare. To process the dream, they use a model by Neidhardt et al. (1992) to write down both the original nightmare and the ideal new scenario. Once they manage this step, participants are encouraged to establish the entire process mentally, without writing down the dream. They are instructed to rehearse this new dream for 5 to 20 minutes daily and not to work on more than two new dreams a week. Importantly, exposure to traumatic events should be gradual and attention should be first paid to less traumatic dreams. During the last session, patients discuss their experiences and progress, and questions may be asked.

According to Marks (1978), imagery is successful because it involves exposure, abreaction and mastery. On the other hand, Bishay (1985) claims that exposure and abreaction are only secondary to mastery. In his observation, storyline alterations are more effective than simple rehearsal of the dream.

The effectiveness of IRT was assessed in several studies such as that of 58 subjects with chronic nightmares by Krakow et al. (1995). Whereas 39 sufferers were treated with IRT, the remaining 19 were controls (on a waiting list). The patients were assessed pre-treatment and at 3-month follow-up. When compared with controls, the treatment group showed a statistically significant decrease in nightmares. Another study by Krakow et al. (2001b), investigated nightmares in 168 sexual assault survivors with PTSD. One group received IRT in three sessions while the controls remained on a control list. After follow-up of 3 and 6 months, treatment reduced the frequency of nightmares as compared with the baseline. There were only nonsignificant improvements in controls. In the treated group, the effect of therapy

remained after both 3 and 6 months, as compared with controls. Thünker and Pietrowsky (2012) studied the effectiveness of IRT in 69 subjects of whom 22 were primarily nightmare sufferers, 21 had major depression and nightmares and 26 had PTSD and nightmares. In the former two groups, all patients were treated. Subjects with PTSD and nightmares were subdivided into those receiving therapy (n=14) and controls (n=12). The therapy comprised eight 50-minute sessions, with both nightmare frequency and the anxiety they caused being assessed. Following therapy, the frequency of nightmares decreased in all treated groups. So did anxiety in all three groups, with the smallest reduction in PTSD subjects. Several other studies are currently under way (Nappi *et al* 2012).

#### Lucid dreaming therapy

There is an IRT variant called lucid dreaming therapy (LDT). During lucid dreaming, patients are made to realize that what they experience is not real but only a dream and to make changes to the dream according to their wishes. The effectiveness of LDT was assessed in a pilot study by Spormaker and van den Bout (2005). Patients were divided into 3 groups. One group received one 2-hour individual LDT session, another one received one 2-hour group LDT session and the remaining participants were placed on the waiting list. Twelve weeks after the intervention, the nightmare frequency following both forms of therapy, group and individual, decreased when compared with controls.

## CONCLUSION

Nightmares are an issue in both the healthy population and patients with numerous mental disorders. Most studies have been concerned with the relationship between nightmares and PTSD. They are also frequently present in patients with borderline personality disorder. Unlike an earlier view that anxiety results from an experienced nightmare, the current view is that anxiety results in the occurrence of nightmares. According to our hypothesis, nightmares play a role in the search for an adaptive solution for incompletely or inadequately solved situation in the past. If psychotherapy or social network support are successful in finding an adaptive solution, nightmares cease to occur. The most recommended psychopharmaceutical to be used in PTSD-related nightmares is prazosin acting upon the autonomic nervous system. The most recommended psychotherapeutic approach is imagery rehearsal therapy, using the principle of creating a new dream scenario that is satisfactory for the patient and its imagery rehearsal.

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