IJODR

CORE Provided by Heidelberger Dokumenten

A cognitive model of recurrent nightmares

Victor I. Spoormaker

Max Planck Institute of Psychiatry, Germany

Summary. Nightmares are a prevalent mental disorder resulting in disturbed sleep, distress, and impairment in daily functioning. Elaborating on previous theoretical models for anxiety disorders, sleep disorders and dreaming, this study introduces a cognitive model of recurrent nightmares, the central element of which concerns representation of the nightmare's repetitive storyline in the memory as a script. It is suggested that activation of this script during REM sleep results in a replay of the nightmare, and that activation occurs through perceived similarity between dream elements and the nightmare script. The model proposes a central role for cognitive processes in the persistence of nightmares over time. The success of cognitive-behavioural treatments is explained and clinical implications are discussed.

Keywords: Nightmares; Cognition; Theory; Script; PTSD

1. Introduction

Recent years have seen a vast increase in studies on efficacy of nightmare treatments and on affective, sleep and personality correlates of nightmares, but attempts to integrate and understand these studies have been rare. One (recent) neuro-cognitive model proposes a function and mechanism of a variety of negative dreaming including nightmares (Levin & Nielsen, 2007), on which the current model elaborates. Aim is to highlight and describe the cognitive processes involved in recurrent nightmares.

2. What is a nightmare?

Nightmare definitions in research and clinical practice vary according to several criteria. For instance, the Diagnostic and Statistical Manual of Mental Disorders (4th ed., text rev. – DSM-IV-TR; (American Psychiatric Association, 2000) defines a nightmare as (1) an extremely frightening dream (2) that leads to awakening. Nightmares can only be diagnosed if (3) they do not occur exclusively during the course of another mental disorder and (4) if they lead to significant distress or functional impairments. The DSM-IV-TR definition is the narrowest definition of nightmares; the International Classification of Sleep Disorders (2nd ed. – ICSD-2; (American Academy of Sleep Medicine, 2005) definition does not limit the range of negative emotions to fear alone as studies have shown that anger, sadness or guilt are also prevalent negative emotions in nightmares (Rose, Perlis, & Kaszniak, 1992; Zadra, Pilon, & Donderi, 2006)

The 'awakening' criterion has also been disputed. The assumption is that nightmares leading to awakening are more intense than nightmares without awakening (usually referred to as bad dreams; (Zadra & Donderi, 2000), but nightmares and bad dreams are both associated with distress (Blagrove, Farmer, & Williams, 2004). The third criterion of the DSM-IV-TR – not occurring exclusively during the course of another disorder – excludes post-traumatic nightmares in Post-traumatic Stress Disorder (PTSD) from a diagnosis, and prevents specific attention and treatment in the clinical practice. Problematic is that few studies have shown that treatment of PTSD leads to a reduction in nightmares (see section 1.3). The fourth criterion on distress is not part of the ICSD-2 definition, although not nightmare frequency but the separate construct of nightmare dist-

Coresponding address: Victor I. Spoormaker, Max Planck Institute of Psychiatry, Kraepelinstr. 2-10, 80804 Munich, Germany. email: spoormaker@mpipsykl.mpg.de

Submitted for publication: January 2008 Accepted for publication: March 2008 ress seems crucial in determining whether nightmares are a problem or not (Belicki, 1992).

Levin & Nielsen (2007) suggested a dimensional scale for dreaming, ranging from dysphoric dreams to post-traumatic nightmares. Negative emotions occur in normal dreams (only 20-25% of explicitly stated emotions are positive, yet this figure becomes around half when self-rated moods are included; (Hall & Van de Castle, 1966; Kramer, Winget, & Whitman, 1971; Schredl & Doll, 1998; Strauch & Meier, 1989) and increasing in emotional intensity are bad dreams (nightmares that do not lead to awakening), nightmares and finally, post-traumatic nightmares, which are assumed to be the most intense. In the research literature, consensus seems to be developing to the following definition: all negative emotions can occur in nightmares, nightmares lead to awakening and bad dreams do not and nightmare distress is an independent but crucial construct. Although there is little evidence in favour of excluding post-traumatic nightmares from a clinical diagnosis (3rd DSM-IV-TR criterion), studies have yet to focus on that topic.

2.1 A cognitive model of recurrent nightmares

In their recent review, Levin & Nielsen proposed a neuro-cognitive mechanism and function for of all types of nightmares and bad dreams. They argue that by its continuous presentation of novel and unexpected elements, dreaming provides an 'ever-changing sequence of contexts to supply the formation of [fear] extinction memories'. Bad dreams are thought to be examples of accomplished fear extinctions, in which a fear memory is neutralised, while nightmares are thought to be examples of failed fear extinctions – i.e. where the process is disrupted by awakening or too intense emotional distress.

The aim of this model is to elaborate on this well-supported model and to describe how nightmares become repetitive and frequent as nightmares associated with clinical distress are typically recurrent, whether as a more or less exact replication of a traumatic event (post-traumatic) or as a broader theme like being chased (idiopathic). The proposed model is a description of the cognitive processes involved in nightmare persistence and associated distress, with specific considerations for current nightmare treatments and predictions about new nightmare treatments.

Besides the work of Levin and Nielsen (2007), the current model draws heavily from, and owes much to other cognitive models about affective disorders (Beck, 1976; Ehlers & Clark, 2000; Ehlers & Steil, 1995; Rachman, 1980), sleep disorders (Harvey, 2002), dream formation (Domhoff, 2001; Hartmann, 1996a; Seligman & Yellen, 1987), in particular regarding the role of expectations (La-Berge & Rheingold, 1990), and scripts (Haberlandt, 1997).



2.2 Nightmares: prevalence and problem

Although most prevalence studies have focused exclusively on college students, studies of the general population have indicated that nightmares are highly prevalent, with up to 70% suffering from occasional nightmares (Hublin, Kaprio, Partinen, & Koskenvuo, 1999) and around 3–5% of the adult population suffering from frequent nightmares (Hublin et al., 1999; Klink & Quan, 1987; Spoormaker & van den Bout, 2005; Stepansky et al., 1998). A similar percentage is estimated to "have a current problem with nightmares" (Nielsen & Zadra, 2000).

Nightmares disturb the sleep (Kales et al., 1980; Krakow, Tandberg, & Scriggins, 1995), produce daily distress (Berquier & Ashton, 1992; Zadra & Donderi, 2000), and may induce physical complaints (Köthe & Pietrowsky, 2001). Chronic insomnia and sleep-disordered breathing (sleep apnoea) are also related to nightmares (Klink & Quan, 1987; Krakow, Melendrez et al., 2001). Nightmares are one of the main symptoms of PTSD (American Psychiatric Association, 2000; Kilpatrick et al., 1998; Schreuder, van Egmond, Kleijn, & Visser, 1998; van der Kolk, Blitz, Burr, Sherry, & Hartmann, 1984) and strongly related to general psychopathology and neuroticism (e.g., (Blagrove et al., 2004; Zadra & Donderi, 2000). The question is whether nightmares are part of an underlying disorder or a separate disorder warranting specific attention.

2.3 Anxiety symptom or separate disorder?

Krakow, Hollifield and Schrader (2000) noted that it is still a prevailing view among health professionals that nightmares are secondary to some other mental disorder (e.g. PTSD or another affective disorder). For instance, according to the DSM-IV-TR nightmares can only be diagnosed when they "do not occur exclusively during the course of another mental disorder" (American Psychiatric Association, 2000). In this view, nightmares are a symptom of a larger syndrome: a nightly symptom of anxiety that does not require separate attention. After all, when the accompanying disorder has been treated, nightmares are thought to disappear (Kaplan & Sadock, 1998). However, recent publications have shown otherwise.

For instance, idiopathic nightmares are not part of an underlying anxiety or other mental disorder, as relationships with affective complaints have typically been weak or absent (these nightmares are more related to general psychopathology level or the personality factor neuroticism, for a review see (Spoormaker, Schredl, & van den Bout, 2006).

Even when nightmares occur during the course of for instance PTSD, they are one of the most frequent symptoms (60-80%) in PTSD patients (Kilpatrick et al., 1998; Neylan et al., 1998; Spoormaker & Montgomery, 2008). Several lines of evidence are incompatible with the view that nightmares are a secondary symptom. First, evidence suggests that nightmares or related REM disruptions (fragmentations) function as a risk-factor for the development of PTSD (Harvey & Bryant, 1998; Mellman, Bustamante, Fins, Pigeon, & Nolan, 2002; Mellman, David, Bustamante, Torres, & Fins, 2001; Mellman, David, Kulick-Bell, Hebding, & Nolan, 1995). Second, treatment of PTSD does not necessarily lead to a reduction in nightmare frequency, e.g. in 38 randomised controlled trials on PTSD included in a meta-analysis (Bisson et al., 2007) just one showed a reduction in nightmares, while five others found no or minimal changes on 'sleep disturbances' and 32 did not bother to measure or report this cardinal feature (Spoormaker & Montgomery, 2008). Third, treatment specifically focused on post-traumatic nightmares, whether cognitive-behavioural or pharmacological, leads to impressive reductions in general PTSD-symptom-severity (Krakow, Hollifield et al., 2001; Raskind et al., 2007). So there is strong evidence to suggest that even post-traumatic nightmares are more than a secondary symptom of PTSD; they are either a core feature of the disorder or a disruption that may develop into a separate disorder during the course of PTSD. All the more reason to treat nightmares as a separate complaint warranting specific treatment - after all, untreated nightmares can persist over decades (Schreuder, Kleijn, & Rooijmans, 2000).

2.4 Idiopathic and post-traumatic nightmares

Although idiopathic and post-traumatic nightmares are usually not separated in research studies, it is a common notion that these are different types of nightmares, with post-traumatic nightmares as the most emotionally intense. Two differences between both are obvious: 1) post-traumatic nightmares are often exact replications of a the traumatic event (Schreuder et al., 1998) and idiopathic nightmares are, if repetitive, more related to a broader theme; 2) idiopathic nightmares usually occur during REM sleep in the third part of the night and post-traumatic nightmares occur earlier in the night or during REM or light non-REM sleep (Hartmann, 1996b). A recent review (Wittmann, Schredl, & Kramer, 2007) showed that, in a re-analysis of Woodward et al.'s polysomnographic data on posttraumatic nightmares (Woodward, Arsenault, Murray, & Bliwise, 2000) around 40% of post-traumatic nightmares occurred outside REM sleep.

However, differences between post-traumatic and idiopathic nightmares should not be overemphasised, as there seem to be more similarities than differences. Both consist of visual (dream) hallucinations, both have a repetitive storyline, comparable accompanving emotions and sleep correlates (Germain & Nielsen, 2003), and both show the same response to cognitive-behavioural treatment (Krakow, Kellner, Pathak, & Lambert, 1995; Krakow, Melendrez et al., 2001). Moreover, it is worth noting that although post-traumatic nightmares can be replications of (parts of) the original traumatic event, post-traumatic nightmares are often not exact replications. In one study, 79% of post-traumatic nightmares of Vietnam veterans had distortions (Esposito, Benitez, Barza, & Mellman, 1999). Moreover, three studies found that up to half of the nightmares in PTSD-patients are idiopathic (Esposito et al., 1999; Mellman et al., 2001; Schreuder et al., 2000). Most relevant for the current model is that both types of nightmares are recurrent nightmares (whether exact replications or more skeleton-like structures) that persist over vears.

3. An overview of the model

The repetitive storyline of recurrent nightmares is based upon a real-life traumatic event (post-traumatic nightmares) or upon a broader theme such as being chased or losing a relative (idiopathic nightmares). The current model proposes that information about the storyline of a recurrent nightmare is represented in the memory as a fixed expectation pattern: a script.

This script describes a typical sequence of events and allows for variability. For instance, a person with recurrent nightmares in which he/she is chased may be chased by different attackers in different settings. As most dream memories, the script is poorly integrated into the autobiographical memory. Dream memories do not have a context in time, place, subsequent, and other previous information necessary for integration into the autobiographical memory. The nightmare script is not only an isolated but also a highly distressing memory that, unlike other dream memories, is clearly recalled after its occurrence and during the day.

If neutral or ambiguous dream elements are interpreted as threatening and similar to elements of a recurrent nightmare script, the nightmare script is activated and due to the highly visual mode of the brain in REM sleep (Hobson, Stickgold, & Pace-Schot, 1998), the nightmare is replayed (see Fig. 1). This replay strengthens the nightmare script, a process mediated by the emotional intensity of the nightmare. Perceived similarity between dream elements and the nightmare script is mediated by the interpretive bias and accessibility of the script. Normalisation and integration of the script is prevented through several cognitive and behavioural processes, in particular cognitive avoidance.

4. The nightmare script

Frequent nightmares typically have a repetitive storyline. In a recent study of 188 college students with nightmares we found that, of all nightmares, including non-frequent, occasional nightmares, about 60% have a repetitive storyline (Lancee & Spoormaker, unpublished



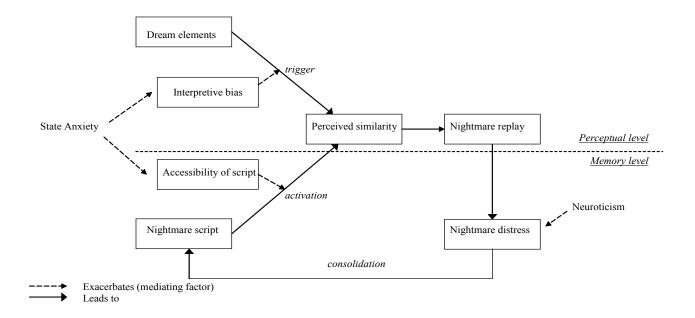


Figure 1. A cognitive model of recurrent nightmares. If there is perceived similarity between elements of a normal dream and the nightmare script, the nightmare script is activated and the nightmare replayed. This strengthens the nightmare script if nightmare distress is high (mediated by the personality factor neuroticism) through cognitive and behavioural processes. Both accessibility of the script and interpretive bias for ambiguous dream elements are mediated by state anxiety.

data) and 91% of the participants with more than one nightmare a week reported that their nightmares had a repetitive storyline. In a sample of schoolchildren, 44% of most recent nightmares were reported to be repetitive (Schredl & Pallmer, 1998).

The current model suggests that the repetitive storyline of nightmares is represented in the memory as a script, similar to a mental groove for verbal behaviour or a schema for procedural knowledge. A nightmare script is a typical sequence of dreamed events or images, where one image is automatically followed by the next (for an example, see Table 1). The nightmare script is strongly connected with and evokes highly adverse emotions when activated, most commonly intense fear.

4.1 Mediating variable: nightmare distress

The distress caused by nightmares determines whether nightmares become recurrent (and problematic). The same dream about being chased can lead to feelings of excitement in some persons (e.g., thrill-seekers) while it can be frightening for other persons and horrifying for anxious persons. This distress is proposed to be exacerbated by the personality factor neuroticism - the tendency to experience negative emotions - and related constructs like trait anxiety / thrill seeking. After all, neuroticism and/or general psychopathology is strongly related to nightmares distress, i.e. distress caused by nightmares, which seems more a personality trait than a characteristic of the nightmare.

The tendency to experience negative emotions affects the intensity and emotional distress of the nightmare. An anxious person is likely to get aroused by a chase, leading to intense negative emotions and awakenings, while a thrill-seeker may not get aroused to the same extent and may just dream on. Awakening from a nightmare prevents normalisation (or fear extinction – Levin & Nielsen, 2007) and instead, strengthens connections between intense emotions and expectations about the storyline, creating the (highly emotional) nightmare script.

Moreover, the tendency to experience negative emotions will also lead to negative appraisals of the nightmare event itself, exacerbating nightmare distress. Nightmare distress will be higher when appraisals of the nightmare are negative:

- "Something is seriously wrong with me";
- "I must be going crazy";
- "Something bad is going to happen";
- "I am still having the same problems as in the past"; or
- "Will I never grow out of it!?"

In short, the current model suggests that neuroticism affects both the emotional intensity of the nightmare and its evaluation afterwards. This is not to be confused with the hypothesis (Belicki, 1992) that persons with high neuroticism overestimate nightmare intensity and distress in retrospect; one study showed that the retrospective intensity of one self-selected nightmare was unrelated to neuroticism scores (Schredl, Landgraf, & Zeiler, 2003). Instead, this model

Table 1. Example of a typical sequence of events in a common recurrent nightmare.

Title nightmare script: BEING CHASED

Scene 1: meeting the attacker

Walking in the dark

Seeing an unfamiliar person

Scene 2: chase starts

Unfamiliar person starts moving towards dreamer Dreamer starts walking and eventually runs away The unfamiliar person follows

Scene 3: climax

The attacker gets nearer

Dreamer unable to run fast; slows down

The attacker catches up

--> Intense fear; awakening

proposes that the reaction to threatening elements in the nightmare is more emotionally intense in persons with high neuroticism; just as they respond more emotionally intense to daily events than for instance thrill-seekers. Additionally, negative appraisals about the nightmare are more likely to occur in persons with high neuroticism. In turn, nightmare intensity and negative evaluations - which can both be categorised under the concept of nightmare distress - determine whether nightmares become recurrent and problematic.

4.2 Cognitive avoidance

Cognitive avoidance is proposed to be the mechanism by which an occasional nightmare with high nightmare distress could become recurrent. When a nightmare has a highly negative impact and is appraised in a negative manner, cognitive avoidance starts directly after awakening. The person tries not to think about the nightmare because it evokes too much fear and/or because he/she may believe that thinking about the nightmare will increase the chance of experiencing it again.

Cognitive avoidance persists throughout the day (e.g. "It was only a dream" – (Schredl & Pallmer, 1998) and has several consequences. First, it makes memory integration of the nightmare script into the (autobiographical) memory less probable. Due to cognitive avoidance, the script is more likely to become an isolated and independent memory.

Second, it prevents emotional normalisation of the script (e.g., fear extinction) – both the type of emotion and its intensity will remain unaltered. Every time the nightmare occurs (see 3.3) the images are accompanied by the same negative and intense emotions. In this way, cognitive avoidance maintains the strong association between the script and its accompanying emotions.

Third, cognitive avoidance also prevents brainstorming about alternate responses to the threatening situation in the nightmare. This reduces the chance that the script will alter spontaneously. Each time the nightmare occurs, associations between the dream images and events are strengthened – and the typical sequence gets fixed. Both emotions and dream images/event become robust to change (strong fear memory).

In addition, cognitive avoidance leads to several behavioural responses that disrupt sleep. Examples are: getting out of bed, making something to eat or drink, delaying bedtimes, sleeping with lights on and several other safety behaviours practised to cope with the fear of going to sleep that is common in nightmare sufferers (Haynes & Mooney, 1975; Krakow, Hollifield, & Schrader, 2000; Spoormaker, Schredl et al., 2006), which is why nightmares may have an etiological role in the development of insomnia, especially in PTSD patients.

4.3 Activation of the script

Activation of the nightmare script results in a replay of the nightmare. During REM sleep, the visual system of the brain is highly active: it continuously produces hallucinations by projecting memories and novel elements before the eyes. Our brain can be viewed as a hyperactive visual 'dream machine' during REM sleep (Hobson, 1999). As a result, activation of the script during REM sleep will lead to a sequence of visual hallucinations. As a script is a sequence of strongly interconnected images, the first image of the nightmare will result in the next (and so on), and the nightmare elaborates according to the typical sequence of nightmare events.

The nightmare script is thought to become activated when elements in a neutral dream resemble elements of the script. When a person has, for instance, nightmares about being chased by an attacker in the dark, a running person in a dream can be interpreted as a 'threatening' element, in this case: a possible attacker. The running person in the dream is intrinsically neutral, but if there is perceived similarity to the script, this image activates the script and the nightmare starts. Dark alleys or unfamiliar or running persons are other intrinsically neutral but 'hazardous' dream stimuli that may activate the 'being chased' nightmare script (see Table 2).

Vague physical similarity would be sufficient in perceiving dream elements as similar to those of the script (comparable to poor stimulus discrimination in PTSD – Ehlers & Clark, 2000). The nightmare can therefore be activated even if the dream elements are rather different from the original nightmare. This is why recurrent idiopathic nightmares employ varying persons and context – these depend on the neutral dream elements that preceded and activated the script. In a similar vein, the common distortions in post-traumatic nightmares can be explained.

The model proposes that two cognitive processes mediate perceived similarity between dream elements and script: 1) the interpretive bias and 2) the accessibility of the script. These two processes are both affected by current levels of anxiety.

4.3.1 Interpretive bias

The interpretive bias denotes the evaluation of a particular stimulus: is it threatening or not? It has repeatedly been found that anxious individuals are more likely than non-anxious individuals to interpret ambiguous stimuli as threatening. An experimental lexical decision task with homographs (words with both threat-related and neutral meanings, e.g. 'batter') showed that high trait-anxious persons were more prone to interpret these words as threatening (Richards & French, 1992). Similar results were found in a lexical decision task with ambiguous sentences in persons with test anxiety (Calvo, Eysenck, & Estevez, 1994) and with sentence pairs (with the first one being ambiguous) in high and low trait-anxious persons (MacLeod & Cohen. 1993). This interpretive bias is thought to reflect an anxious mood state (Eysenck, Mogg, May, Richards, & Mathews, 1991) and has been reported in generalised anxiety disorder (Hazlett-Stevens & Borkovec, 2004) and social anxiety (Constans, Penn, & Hope, 1999).

According to the current model, this interpretive bias is also present in during REM sleep. Hazardous dream elements are similar to homographs in lexical decision tasks in the sense that they are ambiguous: they can be interpreted as threatening or as neutral. This interpretation of an ambiguous dream element determines the further course of the dream – whether it will activate a nightmare (interpretation: threatening) or continue as a normal dream (interpretation: neutral). It is predicted that persons with recurrent nightmares have a negative interpretive bias for hazardous dream elements: hazardous dream elements should lead to shorter reaction times for the subsequent recognition of threatening words than neutral dream elements.

Table 2. Examples of dream elements that may start a particvular script.

Script	Dream elements
Being chased	Running persons, strangers, dark surroundings.
Losing a relative	Being with relative, unfamiliar surroundings.
Drowning	Swimming, water: rivers, lakes, or seas.
Falling	High grounds: cliffs, mountains, or skyscrapers.
Re-experiencing a car crash	Driving a car, highway, traffic.
Re-experiencing a war trauma	Colleagues, noises, surroundings resembling war-territory (e.g., jungle, desert).



Another prediction is that the interpretive bias in nightmares reflects an anxious mood state. With high levels of state anxiety, hazardous dream elements are more easily interpreted as threatening. A high state anxiety alters the interpretive bias in such a way that a larger number of hazardous dream elements can be interpreted as threatening; the pool of hazardous dream elements increases. For instance, with a nightmare script about being chased, instead of running dream personages, all moving dream objects may be interpreted as threats when state anxiety is high. In contrast, when state anxiety is low(er), the pool of hazardous dream elements decreases and instead of all running dream-personages, only male dreampersonages running towards the dreamer may be interpreted as threats and activate the script. In accordance with this prediction is the repeatedly found result that state anxiety is strongly related to nightmare frequency (De Koninck & Brunette, 1991; Köthe & Pietrowsky, 2001; Schredl, 2003).

4.3.2 Accessibility of the script

The accessibility of the script concerns the ease with which the script is activated: the threshold for activation. With a high accessibility (and low threshold) the script is easily triggered, and with a low accessibility (and high threshold) the script is more difficult to activate. Accessibility mediates the likelihood that a certain dream element will activate the script. For instance, if the activation of the nightmare script is compared to the firing of a neuron, then the interpretive bias relates to how many other neurons would send their spike, and accessibility to the polarisation of the neuron itself, i.e. how easily it reaches the threshold and fires.

The accessibility of the script is a function of (poor) memory integration and disrupted fear extinction. Post-traumatic nightmares are examples of strong fear memories in which there is little place for novel elements and fear extinction. Strong fear memories are highly accessible and in its most extreme case, the script is so highly accessible that it becomes activated by any dream element preceding it – a phenomenon that has been reported in PTSD-patients, for whom post-traumatic nightmares could as start as early as the first REM episode (Hartmann, 1996b).

4.4 Cognitive-behavioural treatment of nightmares

Several cognitive-behavioural techniques are effective in reducing nightmare frequency (for a review see Spoormaker, Schredl et al., 2006) but theoretical developments integrating these findings have been lacking. The current model explains the efficacy of these treatments with different mechanisms. In addition, it predicts a new cognitive-behavioural treatment of nightmares.

4.4.1 Increasing the threshold for activation: Relaxation

Relaxation reduces nightmare frequency (Burgess, Gill, & Marks, 1998; Miller & DiPilato, 1983) by reducing current levels of (state) anxiety, which are positively related to nightmare frequency. The present cognitive model suggests that through relaxation and the reduction of state anxiety both the interpretive bias and the accessibility of the script will alter – the pool of 'hazardous' dream elements activating the nightmare script diminishes, whereas the threshold for activation of the script becomes higher. As a result, dream elements will less frequently be perceived as similar to the nightmare script, and activation is therefore less likely.

4.4.2 Overcoming cognitive avoidance: Exposure

Exposure (systematic desensitisation) is an effective treatment for nightmares (Burgess et al., 1998; Burgess, Marks, & Gill, 1994; Celluci & Lawrence, 1979) that is usually accompanied by relaxation, but has shown better outcomes in nightmare reduction than relaxation alone (Burgess et al., 1998). One study found that the effects of exposure were maintained over four years (Grandi, Fabbri, Panattoni, Gonnella, & Marks, 2006). With exposure, participants are instructed to write down a nightmare and to re-experience it using their imagination. Exposure breaks through cognitive avoidance, which is, according to the proposed model, a main mechanism responsible for the persistence of a script. By breaking through cognitive avoidance, exposure makes integration and consolidation of the script into the (autobiographical) memory more likely, and emotional normalisation of the script is more likely to occur.

4.4.3 Changing the script: Cognitive-restructuring

Cognitive-restructuring techniques restructure the script. The introduction of novel, nightmare-incompatible elements into the nightmare may serve the proposed function of fear extinction (Levin & Nielsen, 2007) but certainly breaks down the expectation pattern of the script. Two cognitive-restructuring techniques have been evaluated in randomised controlled trials: imagery rehearsal therapy (IRT) and lucid dreaming treatment (LDT).

IRT (Marks, 1978) has developed into a technique in which participants are instructed to change a recurrent nightmare any way they wish, which they then have to rehearse in imagination. IRT has shown positive long-term results in randomised trials, with large effect sizes (> 1) for nightmare frequency reduction after one to three group sessions (Krakow, Hollifield et al., 2001; Krakow et al., 2000; Krakow, Kellner et al., 1995). In addition, IRT improved the (subjective) sleep quality and decreased PTSD symptom severity in sexual assault survivors with PTSD (Krakow, Hollifield et al., 2001). Two studies with longer follow-ups (18 and 30 months) showed that the effects of IRT are maintained over a longer period (Krakow, Kellner, Neidhardt, Pathak, & Lambert, 1993; Krakow, Kellner, & Pathak, 1994) and IRT is currently the treatment-of-choice for nightmares (Spoormaker, Schredl et al., 2006).

LDT is an alternative cognitive-restructuring technique. Lucid dreaming refers to a person realising that he/she is dreaming in the dream itself. Lucid dreaming has been verified by volitional eyemovements during REM sleep (LaBerge, Nagel, Demen, & Zarcone, 1981) and is a learned cognitive skill (Purcell, Mullington, Moffit, Hoffman, & Pigeau, 1986; Zadra, Donderi, & Pihl, 1992). Particularly because nightmares can spontaneously 'trigger' lucidity in nightmares (Schredl & Erlacher, 2004), lucid dreaming may be an appropriate technique for treating nightmares. Only two series of cases (Spoormaker, van den Bout, & Meijer, 2003; Zadra & Pihl, 1997) and one randomised trial (Spoormaker & van den Bout, 2006) have been conducted to evaluate the effects of LDT and these indicate that, in one to three sessions, LDT reduces nightmare frequency (without affecting anxiety levels or sleep quality as IRT).

The current model predicts that in a randomised comparison with adequate power, cognitive-restructuring techniques show better outcomes in nightmare frequency reduction than exposure.

4.4.4 Restructuring negative appraisals: Paradoxical intention

Another hypothesis regarding cognitive-behavioural treatment is that since negative appraisals / nightmares distress are proposed to be crucial in the creation and persistence of nightmare scripts, alleviating negative appraisals would lead to a reduction in nightmares. This means that paradoxical intention, similar as employed in insomnia (Morin et al., 1999), would show positive treatment outcomes. In other words, encouraging nightmare sufferers to 'have nightmares' would reduce the chances that they actually have one. When negative appraisals are replaced by more positive appraisals (i.e., it is good or functional to have nightmares), nightmare distress will be lowered and script formation halted. In line with this novel technique derived from the model is the finding that people have significantly fewer nightmares in the sleep laboratory or even when measured with polysomnography (Spoormaker, Schreuder, de Weerd, Kleijn, & Kamphuisen, 2006). The costly procedure and efforts aimed at measuring nightmares, could give people the idea that they have to perform, i.e. have a nightmare.

Paradoxical intention could credibly be introduced as part of a cognitive-restructuring technique. The aim of IRT or LDT is to change the nightmare, and in order to change it, one should have a nightmare or rather: as many as possible. Persons could be encouraged in more or less subtle ways and a comparative study of IRT with and without paradoxical intention should show better outcomes for the former condition.

5. Clinical implications

As mentioned before, it is still a common view among health professionals that nightmares are secondary to some other mental disorder and do not warrant specific treatment. This is problematic because idiopathic nightmares have weak relationships with specific anxiety and depressive complaints and because even when nightmares occur during the course of another mental disorder such as PTSD, there is little evidence that shows that they recede once PTSD has been treated (Spoormaker & Montgomery, 2008). However, specific treatment of post-traumatic nightmares does lead to both nightmare reduction and PTSD-symptom-severity (Krakow, Hollifield et al., 2001). For instance, a re-analysis of 38 RCTs on PTSD treatment showed that 12-14 sessions of cognitivebehavioural trauma-focused treatment resulted in minimal changes on sleep disturbances while a three-session cognitive-behavioural nightmare-focused treatment did reduce both nightmare frequency and PTSD-symptom-severity (Spoormaker & Montgomery, 2008).

It seems therefore more fruitful for mental health professionals to adopt a new view on nightmares. This new view should stress that no matter how they are induced, nightmares may develop into an independent and persistent disorder warranting specific attention. The DSM-IV-TR has a separate diagnosis for idiopathic nightmares; we suggest professionals use it more often. And although posttraumatic nightmares cannot receive a specific diagnosis according to the DSM-IV-TR, we suggest that professionals more closely monitor the effects of PTSD treatment on post-traumatic nightmares. Cognitive-behavioural treatment that focuses on memory integration and emotional normalising of the traumatic event (i.e., nightmare script) is likely to reduce nightmare frequency, but when there is no effect, specific nightmare treatment should be considered.

6. Limitations

The present model has a few limitations. First, it focuses on a specific type of nightmares: recurrent nightmares. Other types of negative dreams, including bad dreams and dysphoric dreaming, fall outside the scope of this model.

Furthermore, it is unsure whether the proposed model is supported by brain mechanisms during REM sleep. For example, an interpretive bias and the activation of a script concern active and complex cognitive processes that may well require aminergic activity. However, neuropharmacological evidence shows that aminergic activity is diminished during REM sleep (Hobson et al., 1998). Nightmares may differ from normal REM sleep in this regard, as serotonergic and dopaminergic agents have been found most likely to induce nightmares (Pagel & Helfter, 2003; Thompson & Pierce, 1999), whereas a noradrenergic antagonist (Prazosin) has an ameliorating effect on nightmare frequency (Raskind et al., 2003; Raskind et al., 2007). But to date, the necessary presence of neuropharmacological mechanisms or the necessary brain activation for such complex cognitive processes during REM sleep have not yet been sufficiently proven.

7. Conclusions

A conceptual model for the persistence of recurrent nightmares has been introduced that addresses the role of cognitive processes in recurrent nightmares. The central element of this model concerns the representation of the nightmare in a script, a consequence of cognitive avoidance due to negative appraisals and nightmare distress. Activation of this script results in a replay of the nightmare through perceived similarity between dream elements and elements of the script. This mechanism is suggested to depend on the accessibility of the script and the interpretive bias, which are in turn both affected by current levels of anxiety. The script, based upon a real-life traumatic event or a broader theme, is unlikely to be altered, integrated, or normalised without specific cognitive-behavioural treatment. Although the present model has its limitations, its testable predictions and treatment implications should be able to aid and guide future studies as well as mental health professionals.

Acknowledgements

The author would like to thank Michael Schredl, Jaap Lancee, Jan van den Bout and Karolijne van der Houwen for their valuable comments. This study was supported by the Netherlands Organisation for Scientific Research.

References

- American Academy of Sleep Medicine. (2005). International classification of sleep disorders: Diagnostic and coding manual (2 ed.). Westchester, IL: Author.
- American Psychiatric Association. (2000). Diagnostic and Statistic Manual of Mental Disorders (4th ed.). Washington DC: American Psychiatric Press.
- Beck, A. T. (1976). Cognitive therapy and the emotional disorders. New York: Penguin.
- Belicki, K. (1992). Nightmare frequency versus nightmare distress: relations to psychopathology and cognitive style. Journal of Abnormal Psychology, 101, 592-597.
- Berquier, A., & Ashton, R. (1992). Characteristic of the frequent nightmare sufferer. Journal of Abnormal Psychology, 101, 246-250.
- Bisson, J. I., Ehlers, A., Matthews, R., Pilling, S., Richards, D., & Turner, S. (2007). Psychological treatments for chronic post-traumatic stress disorder. British Journal of Psychiatry, 190, 97-104.
- Blagrove, M., Farmer, L., & Williams, E. (2004). The relationship of nightmare frequency and nightmare distress to well-being. Journal of Sleep Research, 13, 129-136.
- Burgess, M., Gill, & Marks, I. M. (1998). Postal self exposure treatment of recurrent nightmares: a randomised controlled trial. British Journal of Psychiatry, 172, 257-262.
- Burgess, M., Marks, I. M., & Gill, M. (1994). Postal self-exposure treatment of recurrent nightmares. British Journal of Psychiatry, 165, 388-391.
- Calvo, M. G., Eysenck, M. W., & Estevez, A. (1994). Ego-threat interpretive bias in test anxiety. Cognition and Emotion, 8, 127-146.
- Celluci, A. J., & Lawrence, P. S. (1979). The efficacy of systematic desentisation in reducing nightmares. Journal of Behavioural Therapy and Experimental Psychiatry, 9, 109-114.
- Constans, J., Penn, D., & Hope, G. I. D. (1999). Interpretive biases for ambiguous stimuli in social anxiety. Behaviour Research and Therapy, 37, 643-651.
- De Koninck, J., & Brunette, R. (1991). Presleep suggestion related to a phobic object: successful manipulation of reported dream affect. Journal of General Psychology, 118, 185-200.
- Domhoff, G. W. (2001). A new neurocognitive theory of dreams. Dreaming, 11, 13-33.
- Ehlers, A., & Clark, D. M. (2000). A cognitive model of post-traumatic stress disorder. Behaviour Research and Therapy, 38, 319-345.
- Ehlers, A., & Steil, R. (1995). Maintenance of intrusive memories in posttraumatic stress disorder: A cognitive approach. Behavioural and Cognitive Psychotherapy, 23, 217-249.
- Esposito, K., Benitez, A., Barza, L., & Mellman, T. A. (1999). Evaluation of dream content in combat-related PTSD. Journal of Traumatic Stress, 12, 681-687.
- Eysenck, M. W., Mogg, K., May, J., Richards, A., & Mathews, A. (1991). Bias in interpretation of ambiguous sentences related to threat in anxiety. Journal of Abnormal Psychology, 100, 144-150.
- Germain, A., & Nielsen, T. A. (2003). Sleep pathophysiology in posttraumatic stress disorder and idiopathic nightmare sufferers. Biological Psychiatry, 54, 1092-1098.
- Grandi, S., Fabbri, S., Panattoni, N., Gonnella, E., & Marks, I. M. (2006). Selfexposure treatment of recurrent nightmares: waiting-list-controlled trial and 4-year follow-up. Psychotherapy and Psychosomatics, 75, 384-388.
- Haberlandt, K. (1997). Cognitive Psychology (2 ed.). Boston: Allyn and Bacon.
- Hall, C. S., & Van de Castle, R. (1966). The content analysis of dreams. New York: Appleton-Century-Crofts.
- Hartmann, E. (1996a). Outline for a theory on the nature and functions of dreaming. Dreaming, 6, 147-160.
- Hartmann, E. (1996b). Who develops PTSD nightmares and who doesn't. Cambridge, MA: Harvard University Press.
- Harvey, A. G. (2002). A cognitive model of insomnia. Behaviour Research and Therapy, 40, 869-893.
- Harvey, A. G., & Bryant, R. A. (1998). The relationship between acute stress disorder and posttraumatic stress disorder: a prospective evaluation of motor vehicle accident survivors. Journal of Consulting and

A cognitive model of recurrent nightmares



- Clinical Psychology, 66, 507-512. Haynes, S., & Mooney, D. K. (1975). Nightmares: etiological, theoretical and behavioral treatment considerations. Psychological Record, 25, 225-236.
- Hazlett-Stevens, H., & Borkovec, T. (2004). Interpretive cues and ambiguity in generalized anxiety disorder. Behaviour Research and Therapy, 42, 881
- Hobson, J. A. (1999). Dreaming as delirium. Boston: MIT Press.
- Hobson, J. A., Stickgold, R., & Pace-Schot, E. (1998). The neuropsychology of REM sleep dreaming. Neuroreport, 9, 1-14.
- Hublin, C., Kaprio, J., Partinen, M., & Koskenvuo, M. (1999). Nightmares: familial aggregation and association with psychiatric disorders in nationwide twin cohort. American Journal of Medical Genetics, 88, 329-336.
- Kales, A., Soldatos, C., Caldwell, A. B., Charney, D. S., Kales, J. D., Markel, D., et al. (1980). Nightmares: clinical characteristics and personality patterns. American Journal of Psychiatry, 137, 1197-1201.
- Kaplan, H. I., & Sadock, B. J. (1998). Synopsis of psychiatry: Behavioral sciences / clinical psychiatry (8 ed.). Philadelphia: Lippincott.
- Kilpatrick, D. G., Resnick, H. S., Freedy, D., Pelcovitz, P., Resick, S., Roth, S., et al. (1998). Posttraumatic stress disorder field trial: evaluation of PTSD construct criteria A through E. In T. A. Widiger, A. J. Frances, H. A. Pincus, R. Ross, M. B. First, W. Davis & M. Kline (Eds.), DSM-IV Sourcebook. Washington: American Psychiatric Press.
- Klink, M., & Quan, S. (1987). Prevalence of reported sleep disturbances in a general population and their relation to obstructive airway disease. Chest, 91, 540-546.
- Köthe, M., & Pietrowsky, R. (2001). Behavioral effects of nightmares and their correlations to personality patterns. Dreaming, 11, 43-52.
- Krakow, B., Hollifield, M., Johnston, L., Koss, M., Schrader, R., Warner, T. D., et al. (2001). Imagery rehearsal therapy for chronic nightmares in sexual assault survivors with posttraumatic stress disorder: a randomized controlled trial. Journal of the American Medical Association, 286, 537-545.
- Krakow, B., Hollifield, M., & Schrader, R. (2000). A controlled study of imagery rehearsal for chronic nightmares in sexual assault survivors with PTSD; a preliminary report. Journal of Traumatic Stress, 13.
- Krakow, B., Kellner, R., Neidhardt, E. J., Pathak, D., & Lambert, L. (1993). Imagery rehearsal treatment of chronic nightmares: with a thirty month follow-up. Journal of Behaviour Therapy and Experimental Psychiatry, 24, 325-330.
- Krakow, B., Kellner, R., & Pathak, D. (1994). Imagery rehearsal treatment of chronic nightmares: an eighteen month follow-up. Sleep Research, 23. 169.
- Krakow, B., Kellner, R., Pathak, D., & Lambert, L. (1995). Imagery rehearsal treatment for chronic nightmares. Behaviour Research and Therapy, 33(837-843).
- Krakow, B., Melendrez, D. C., Pedersen, B., Johnston, L., Hollifield, M., Germain, A., et al. (2001). Complex insomnia: insomnia and sleepdisordered breathing in a consecutive series of crime victims with nightmares and PTSD. Biological Psychiatry, 49, 948-953.
- Krakow, B., Tandberg, D., & Scriggins, L. (1995). A controlled comparison of self-rated sleep complaints in acute and chronic nightmares sufferers. Journal of Nervous and Mental Disease, 183, 623-627.
- Kramer, M., Winget, C., & Whitman, R. M. (1971). A city dream: A survey approach to normative dream content. American Journal of Psychiatry, 127, 1350-1356.
- LaBerge, S., Nagel, L. E., Demen, W. C., & Zarcone, V. P. (1981). Lucid dreaming verified by volitional communication during REM sleep. Perceptual and Motor Skills, 52, 727-732.
- LaBerge, S., & Rheingold, H. (1990). Exploring the world of lucid dreaming. New York: Ballantine.
- Levin, R., & Nielsen, T. A. (2007). Disturbed dreaming, posttraumatic stress disorder, and affect distress: a review and neurocognitive model. Psychological Bulletin, 133, 482-528.
- MacLeod, C., & Cohen, I. (1993). Anxiety and the interpretation of ambiguity: A text comprehension study. Journal of Abnormal Psychology, 102, 238-247
- Marks, I. M. (1978). Rehearsal relief of a nightmare. British Journal of Psychiatry, 135, 461-465.
- Mellman, T. A., Bustamante, V., Fins, A. I., Pigeon, W. R., & Nolan, B. (2002). REM sleep and the early development of posttraumatic stress disorder. American Journal of Psychiatry, 159, 1696-1701.
- Mellman, T. A., David, D., Bustamante, V., Torres, J., & Fins, A. (2001). Dreams in the acute aftermath of trauma and their relationship to PTSD. Journal of Traumatic Stress, 14, 241-247.
- Mellman, T. A., David, D., Kulick-Bell, R., Hebding, J., & Nolan, B. (1995). Sleep disturbance and its relationship to psychiatric morbidity after Hurricane Andrew. American Journal of Psychiatry, 152, 1659-1663
- Miller, W. R., & DiPilato, M. (1983). Treatment of nightmares via relaxation and desentisation: a controlled evaluation. Journal of Consulting Clinical Psychology, 51, 870-877.

- Morin, C. M., Hauri, P. J., Espie, C. A., Spielman, A. J., Buysse, D. J., & Bootzin, R. R. (1999). Nonpharmacologic treatment of chronic insomnia. An American Academy of Sleep Medicine Review. Sleep, 22, 1-23.
- Neylan, T. C., Mannar, C. R., Metzler, T. J., Weiss, D. S., Zatzick, D. F., & Delucchi, K. L., ETAL. (1998). Sleep disturbances in the Vietnam generation: Findings from a nationally representative sample of male Vietnam veterans. American Journal of Psychiatry, 155, 929-933.
- Nielsen, T. A., & Zadra, A. L. (2000). Dreaming disorders in principles and practices in sleep medicine. In M. H. Kryger, T. Roth & W. C. Dement (Eds.), Principle and Practices of Sleep medicine (3th ed.). Philadelphia: WB Saunders.
- Pagel, J. F., & Helfter, P. (2003). Drug induced nightmares an etiology based review. Human Psychopharmacology, 18, 59-67.
- Purcell, S., Mullington, J., Moffit, A., Hoffman, R., & Pigeau, R. (1986). Dream self-reflectiveness as a learned cognitive skill. Sleep, 9, 423-437.
- Rachman, S. (1980). Emotional processing. Behaviour Research and Therapy, 18, 51-60.
- Raskind, M. A., Peskind, E., Kanter, E., Petrie, E., Radant, A., Dobie, C. T. D., et al. (2003). Reduction of nightmares and other PTSD symptoms in combat veterans by prazosin: a placebo-controlled study. American Journal of Psychiatry, 160, 371-373.
- Raskind, M. A., Peskind, E. R., Hoff, D. J., Hart, K. L., Holmes, H. A., Warren, D., et al. (2007). A parallel group placebo controlled study of prazosin for trauma nightmares and sleep disturbance in combat veterans with posttraumatic stress disorder. Biological Psychiatry, 928-934.
- Richards, A., & French, C. (1992). An anxiety-related bias in semantic activation when processing threat/neutral homographs. The Quarterly Journal of Experimental Psychology, 45A, 503-525.
- Rose, M. W., Perlis, M. L., & Kaszniak, A. W. (1992). Self-reported dream emotion: nightmares and vivid dreams. Sleep Research, 21, 132.
- Schredl, M. (2003). Effects of state and trait factors on nightmare frequency. European Archives of Psychiatry and Clinical Neuroscience, 253, 241-247.
- Schredl, M., & Doll, E. (1998). Emotions in diary dreams. Consciousness and Cognition, 7, 634-646.
- Schredl, M., & Erlacher, D. (2004). Lucid dreaming frequency and personality. Personality and Individual Differences, 37, 1463-1473.
- Schredl, M., Landgraf, C., & Zeiler, O. (2003). Nightmare frequency, nightmare distress and neuroticism. North American Journal of Psychology, 5, 345-350.
- Schredl, M., & Pallmer, R. (1998). Geschlechtsunterschiede in Angstträumen von Schülerinnen. Praxis der Kinderpsychologie und Kinderpsychiatrie, 47, 463-476.
- Schreuder, J., Kleijn, W., & Rooijmans, H. (2000). Nocturnal re-experiencing more than forty years after war trauma. Journal of Traumatic Stress, 13, 453-463.
- Schreuder, J., van Egmond, M., Kleijn, W., & Visser, A. (1998). Daily reports of posttraumatic nightmares and anxiety dreams in Dutch war victims. Journal of Anxiety Disorders, 12, 511-524.
- Seligman, M., & Yellen, A. (1987). What is a dream? Behaviour Research and Therapy, 25, 1-24.
- Spoormaker, V. I., & Montgomery, P. (2008). Disturbed sleep in post-traumatic stress disorder: secondary symptom or core feature? Sleep Medicine Reviews, 12, in press.
- Spoormaker, V. I., Schredl, M., & van den Bout, J. (2006). Nightmares: from anxiety symptom to sleep disorder. Sleep Medicine Reviews, 10(1), 53-59
- Spoormaker, V. I., Schreuder, J. N., de Weerd, A. W., Kleijn, W. C., & Kamphuisen, H. A. C. (2006). Polysomnography reduces nightmare frequency. Sleep-Wake Research in the Netherlands, 17, 113-116.
- Spoormaker, V. I., & van den Bout, J. (2005). The prevalence of sleep disorders in the Netherlands. Sleep-Wake Research in the Netherlands, 16, 155-158.
- Spoormaker, V. I., & van den Bout, J. (2006). Lucid dreaming treatment for nightmares: a pilot-study. Psychotherapy and Psychosomatics, 75, 389-394.
- Spoormaker, V. I., van den Bout, J., & Meijer, E. J. G. (2003). Lucid dreaming treatment for nightmares: a series of cases. Dreaming, 13, 181-186.
- Stepansky, R., Holzinger, B., Schmeiser-Rieder, A., Saletu, B., Kunze, M., & Zeitlhofer, J. (1998). Austrian dream behavior: results of a representative population survey. Dreaming, 8, 23-31.
- Strauch, I., & Meier, B. (1989). Das emotionale Erleben im REM-Traum. Schweizerische Zeitschrift fur Psychologie und Ihre Anwendungen, 48, 233-240.
- Thompson, D. F., & Pierce, D. R. (1999). Drug-induced nightmares. Annals of Pharmacotherapy, 33, 93-98.
- van der Kolk, B., Blitz, R., Burr, W., Sherry, S., & Hartmann, E. (1984). Nightmares and trauma: A comparison of nightmares after combat with lifelong nightmares in veterans. American Journal of Psychiatry, 141, 187-190.
- Wittmann, L., Schredl, M., & Kramer, M. (2007). Dreaming in posttraumatic stress disorder: A critical review of phenomenology, psychophy-



siology and treatment. Psychotherapy and Psychosomatics, 76, 25-39.

- Woodward, S. H., Arsenault, N. J., Murray, C., & Bliwise, D. L. (2000). Laboratory sleep correlates of nightmare complaint in PTSD inpatients. Biological Psychiatry, 48, 1081-1087.
- Zadra, A. L., Donderi, D. C., & Pihl, R. O. (1992). Efficacy of lucid dream in-duction for lucid and non-lucid dreamers. Dreaming, 2, 85-97.
- Zadra, A. L., & Donderi, D. O. (2000). Nightmares and bad dreams: their prevalence and relationship to well-being. Journal of Abnormal Psychology, 109, 273-281.
- Zadra, A. L., & Pihl, R. O. (1997). Lucid dreaming as a treatment for recurrent nightmares. Psychotherapy and Psychosomatics, 66, 50-55.
 Zadra, A. L., Pilon, M., & Donderi, D. C. (2006). Variety and intensity of emo-tions in nightmares and bad dreams. Journal of Nervous and Mental Disease, 194, 249-254.