Cognitive-Behavioral Treatment for Chronic Nightmares in Trauma-Exposed Persons: Assessing Physiological Reactions to Nightmare-Related Fear

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Cognitive-behavioral treatments (CBTs) that target nightmares are efficacious for ameliorating self-reported sleep problems and psychological distress. However, it is important to determine whether these treatments influence objective markers of nightmare-related fear, because fear and concomitant physiological responses could promote nightmare chronicity and sleep disturbance. This randomized, controlled study (N = 40) assessed physiological (skin conductance, heart rate, facial electromyogram) and subjective (displeasure, fear, anger, sadness, arousal) reactions to personally relevant nightmare imagery intended to evoke nightmare-related fear. Physiological assessments were conducted at pretreatment as well as 1-week, 3-months, and 6-months posttreatment. Results of mixed effects analysis of variance models suggested treatment reduced physiological and subjective reactions to nightmare imagery, gains that were generally maintained at the 6-month follow-up. Potential implications are discussed. © 2010 Wiley Periodicals, Inc. J Clin Psychol 66: 1–18, 2010.

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Frequent nightmares are associated with chronic distress (e.g., Kilpatrick et al., 1998; Schreuder, Kleijn, & Rooijmans, 2000) and poor, long-term functioning (e.g., Harvey & Bryant, 1998), but they may also contribute to the initiation and/or maintenance of posttraumatic stress disorder (PTSD) in trauma-exposed individuals (Neylan et al., 1998; Ross, Ball, Sullivan, & Caroff, 1989). Despite the pervasive negative impact of nightmares, they can be particularly resistant to interventions that
broadly target PTSD symptomatology (e.g., Clark et al., 1999; Forbes, Creamer, & Biddle, 2001). However, cognitive-behavioral treatments (CBTs) that specifically target nightmares appear to be more efficacious, showing improvements in subjective sleep quality and reductions in the severity and frequency of idiopathic and trauma-related nightmares (e.g., Davis & Wright, 2007; Krakow et al., 2001). We find it interesting that these interventions also improve symptoms of PTSD and depression, although these are not specifically targeted in treatment (e.g., Davis & Wright; Krakow et al., 2001).

From a theoretical standpoint, it may be important to assess objective indicators of nightmare-related fear and negative affect as an outcome of nightmare treatments, because fear may promote and maintain nightmare chronicity. For example, Levin and Nielsen (2007) have argued that posttraumatic nightmares (and even distressing non-posttraumatic nightmares) reflect a failure of fear extinction processes that would normally occur during dreaming—processes that are mediated by a putative neural circuit involving the amygdala, medial prefrontal cortex, anterior cingulate cortex, and hypothalamus. In Levin and Nielsen’s conceptualization, nightmares are pathological versions of fear memory networks that are highly resistant to extinction (Foà & Kozak, 1986; Lang, 1979) and lead to responses such as avoidance, physiological reactivity, and affective distress. Successful amelioration of nightmares requires the extinction of the fear memory network, breaking linkages among (a) the nightmare content, (b) physiological, verbal, and behavioral responses of fear, and (c) the meaning associated with these responses (Foà & Kozak, 1986; Lang, 1979; Levin & Nielsen, 2007). Following Levin and Nielsen’s line of reasoning, successful nightmare treatments should reduce objective measures of nightmare-related fear.

One experimental paradigm that can be used to assess physiological reactions to nightmare-related fear is script-driven imagery. In most script-driven imagery experiments, participants are exposed to a personally relevant fear script and asked to imagine it as vividly as possible while physiological reactions are assessed (e.g., heart rate [HR], skin conductance [SC], facial electromyogram [EMG]). This method of fear elicitation has been used extensively in individuals with PTSD and other anxiety disorders with results indicating a marked increase in physiological-emotional reactions to the personal fear imagery (e.g., Cuthbert et al., 2003; Lindauer et al., 2006; Orr, 1997). As further validation, imaging research employing the script-driven imagery paradigm have found it can activate neural circuitry involved with fear and negative emotions (e.g., amygdala, anterior cingulated; e.g., Etkin & Wager, 2007; Lanius et al., 2007; Lindauer et al., 2004; Shin et al., 2004), and treatment outcome studies have found that physiological responses to script-driven fear are reduced by successful interventions (e.g., Lindauer et al., 2006; Shalev, Orr, & Pitman, 1992).

If Levin and Nielsen’s (2007) hypothesis is correct, then assessing physiological reactions to nightmare-related imagery should provide a method of assessing the strength of the fear memory network that maintains the nightmares. In a previous study, we have shown that personal nightmare imagery evokes robust physiological and negative emotional reactions in trauma-exposed nightmare sufferers, an effect that is independent of mental health status (PTSD, depressive symptomatology, dissociation; Rhudy, Davis, Williams, McCabe, & Byrd, 2008). Specifically, nightmare imagery led to HR acceleration, increased SC (a measure of sympathetic nervous system activation), and increased tension in facial muscles as assessed from EMG (indicative of facial displays of negative emotion and fear). Rhudy et al. (2008) also noted that autonomic responses (HR, SC) evoked by nightmare imagery were
positively correlated with self-reported sleep problems and health symptoms. Together, these results suggest the script-driven imagery paradigm is an ideal method to assess the effects of CBT for nightmares on physiological reactivity to nightmare-related fear.

The purpose of the present study was to assess the impact of Exposure, Relaxation, & Rescripting Therapy (ERRT) on physiological and subjective measures of fear and negative affect (HR, SC, facial EMG) in response to personal, nightmare-related imagery. ERRT is a brief, three-session treatment that directly targets nightmares in trauma-exposed persons. A case study (Davis, De Arellano, Falsetti, & Resnick, 2003), a case series (Davis & Wright, 2005), and a randomized controlled trial (Davis & Wright, 2007) have previously demonstrated the efficacy of ERRT on other outcome measures. Specifically, immediate improvements (1 week posttreatment) were noted in the reported frequency and severity of nightmares, sleep problems, PTSD symptom frequency and severity, feeling rested upon waking, and depression, with treatment gains persisting to the 6-month follow-up (e.g., only 16% reporting a nightmare in the previous week: Davis & Wright). To date, no study has examined the influence of treatment for nightmares on objective measures of nightmare-related fear. However, given the positive effects ERRT has on other outcome measures, we predicted it would reduce physiological and subjective emotional reactions to nightmare imagery and that treatment gains would persist for at least 6 months.

Methods

Participants

Participants were eligible to participate if they were ≥18 years old, had previous trauma exposure, and nightmares ≥1 time/week for the previous 3 months. Exclusion criteria included psychosis, mental retardation, suicidality/parasuicidal behaviors, or current drug/alcohol dependence. Participants were asked to refrain from consuming alcohol, nicotine, or caffeine 24 hours before physiological assessments. Figure 1 depicts the recruitment flowchart and final participant numbers. Of the 40 participants randomized to the study, most were female (73.2%), married (42.9%), and employed full time (46.3%). Ages ranged from 21 to 63 years (M = 38 yrs, SD = 12). Trauma exposure comprised the following items (participants could list more than one): serious accident (63.4%), natural disaster (34.1%), diagnosed with serious illness (19.5%), sexual assault/rape (53.7%), attacked with weapon (53.7%), attacked without weapon (43.9%), serious injury/physical damage (31.7%), threat of death/serious injury (48.8%), and witnessed others seriously injured or violently killed (41.5%). Written informed consent was obtained from all participants after a complete description of the study was provided.

Cognitive Behavioral Treatment for Nightmares: Exposure, Relaxation, and Rescripting Therapy (ERRT)

For a thorough description of ERRT, see Davis and Wright (2007). ERRT was administered 2 hours a week for three weeks. Session 1 included psychoeducation (about trauma, PTSD, nightmares, and sleep hygiene) and training in progressive muscle relaxation (PMR). Homework included practicing PMR, changing one’s sleep habit, and monitoring nightmares and psychological symptoms. Session 2 included review of homework and instructions for exposure and rescripting.
Exposure included writing out the nightmares, reading it aloud, and identifying relevant themes (McCann, Sakheim, & Abrahamson, 1988; Resick & Schnicke, 1993). Participants were instructed to rescript their nightmare any way they wanted but were encouraged to use the themes (e.g., increase sense of power if nightmare theme was powerlessness) in writing and read aloud the rescripted nightmare. Session 2 ended with diaphragmatic breathing and homework (visualize rescripted dream, PMR, change another sleep habit, practice breathing, monitor symptoms). Session 3 included homework review, discussion of rescripting process, and problem-solving difficulties.

**Questionnaires**

For the present study, several questionnaires were administered to assess demographic information and to determine whether there were group differences (completers vs. noncompleters) on important psychological and sleep-related variables.

A self-report demographic questionnaire was used to obtain information about age, marital status, educational achievement, ethnicity, vocational status, and...
household income. The symptom endorsement method of scoring the Clinician-Administered PTSD scale (CAPS; Blake et al., 1990) was used to determine current and lifetime PTSD diagnoses. This method has adequate sensitivity and specificity rates (Weathers, Ruscio, & Keane, 1999) and good reliability (Blake et al.; Weathers et al.). The Modified PTSD Symptom Scale Self Report (MPSS-R-SR; Resnick, Best, Kilpatrick, Freedy, & Falsetti, 1993) assessed the frequency and severity of 17 PTSD symptoms (Falsetti, Resnick, Resick, & Kilpatrick, 1993; Wright, Davis, Inness, & Stem, 2003). The Beck Depression Inventory-2 (BDI-2; Beck, Steer, & Brown, 1996) was used to assess depressive symptomatology. The Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) was used to assess sleep problems and quality for the 1-month before the assessment (Buysse et al.). A global sleep quality score was obtained by summing seven component scores (subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction). Scores ranged from 0 to 21 (higher scores = poorer sleep quality). Additionally, the Pittsburgh Sleep Quality Index Addendum for PTSD (PSQI-A; Germain, Hall, Krakow, Shear, & Buysse, 2005) was used to assess PTSD-related sleep/nighttime behaviors. Scores can range from 0 to 21 with higher scores reflecting greater sleep problems. A computer version of the Self-Assessment Manikin (SAM; Bradley & Lang, 1994) was used to assess subjective valence/pleasure (unpleasant–pleasant) and arousal (calm–excited) in response to nightmare imagery (Rhudy, Williams, McCabe, Nguyen, & Rambo, 2005). For this study, the valence/pleasure scale was reverse scored to assess displeasure. Scores ranged between 1 and 9 for each dimension (higher scores = greater displeasure or arousal). Additionally, participants were asked to rate their subjective fear, anger, and sadness in response to the imagery using 21-point Likert-type scales (0 = Not at all, 20 = Extremely).

Emotional Scripts for Script-Driven Imagery

Experimenters constructed a personal nightmare script for each participant from content assessed by the Nightmare Content Interview, a structured interview developed for the present study. The Nightmare Content Interview asked participants to report on their most recurrent, or most recent (if a single recurring nightmare was not available), nightmare. General instructions asked them to “briefly describe your recurrent nightmare,” and then follow-up questions probed for cognitive, somatic, and sensory details of the nightmare. In addition, nonpersonal scripts (fear = public speaking, neutral = sitting on a lawn chair, pleasant = lying on beach, action = riding a bicycle) were obtained from other researchers using script-driven imagery (Peter Lang, personal communication; Scott Orr, personal communication) and used as control stimuli. All personal and nonpersonal scripts were approximately 100 words long, written in second person, and recorded onto the computer by a female experimenter in a slow-paced voice (length = 30-s). Thus, the computer presented all scripts and the order of scripts was randomized both within- and between-subjects.

We have previously demonstrated the validity of script-driven imagery for evoking nightmare-related fear and negative emotions (Rhudy et al., 2008). Specifically, in 28 participants, nightmare imagery was shown to evoke significant displeasure (SAM valence ratings reversed scored) and arousal ($M_s = 8.28$ and 7.61, respectively). Moreover, variability in these reactions was small ($SEM_s = 0.25$ and 0.38, respectively), suggesting that despite the nightmare scripts being different for each
participant, they were consistently distressing. Data from the current study also support the validity of using script-driven imagery to evoke nightmare-related fear (see Table 1).

We have also shown that reactions to nightmare imagery correlate with other treatment outcome variables (Rhudy et al., 2008). For example, HR reactivity was associated with global sleep problems (as assessed by the Pittsburgh Sleep Quality Index; \( r = 0.55 \)), reported hours slept per night (\( r = -0.59 \)), subjective panic upon waking (\( r = 0.58 \)), reported health symptoms (as assessed by the Pennebaker Inventory of Limbic Languidness; \( r = 0.54 \)), and minutes taken to fall asleep (\( r = 0.41 \)). SC reactivity was associated with minutes taken to fall asleep (\( r = 0.50 \)), hours slept per night (\( r = -0.40 \)), and panic upon awakening (\( r = 0.63 \)). Displeasure reactions were associated with PTSD-related sleep behaviors (\( r = -0.46 \)) and corrugator EMG reactions were associated with minutes taken to fall asleep (\( r = 0.41 \)). Together, these data provide external validity for the use of the script-driven imagery to assess treatment outcome.

Prior analyses on physiological-emotional reactions to imagery at baseline suggested only the personally-relevant, nightmare script produced robust physiological responses (Rhudy et al., 2008). Of the nonpersonal scripts, the fear script led to the largest change in SC (0.28 DmS), corrugator EMG (1.22 DmV), and lateralis frontalis EMG (0.25 DmV), and the action script led to the largest change in HR (2.56 Dbpm). When these reactions are compared with the personally relevant, nightmare imagery (see Fig. 2), it is clear that reactions to nonpersonal scripts were much smaller. Therefore, to avoid floor effects (i.e., it would be difficult to observe reliable decreases in such small reactions), only reactions to the nightmare script were used to assess treatment response in the present study. However, preliminary analyses that examined the influence of treatment on physiological reactions to the fear script (the nonpersonal script that evoked the most consistent responses) found that treatment did not alter those reactions (Davis et al., 2008).

<table>
<thead>
<tr>
<th>Rating scale</th>
<th>Baseline</th>
<th>Posttreatment/baseline #2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SEM</td>
</tr>
<tr>
<td>SAM displeasure (1–9)</td>
<td>Treatment</td>
<td>8.33</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>8.43</td>
</tr>
<tr>
<td>Fear (1–20)</td>
<td>Treatment</td>
<td>16.67</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>15.95</td>
</tr>
<tr>
<td>Anger (1–20)</td>
<td>Treatment</td>
<td>9.28</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>10.48</td>
</tr>
<tr>
<td>Sadness (1–20)</td>
<td>Treatment</td>
<td>12.89</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>12.90</td>
</tr>
<tr>
<td>SAM arousal (1–9)</td>
<td>Treatment</td>
<td>8.00</td>
</tr>
<tr>
<td></td>
<td>Control</td>
<td>7.76</td>
</tr>
</tbody>
</table>

Note: SEM = standard error of the mean; SAM = Self-Assessment Manikin. *Indicates the Bonferroni adjusted mean comparison between baseline and posttreatment was significant at \( p < 0.05 \).
Participants sat alone in a sound attenuated chamber while being monitored from an adjoining room by video camera. A PC with dual 17'' flat panel monitors and A/D board (National Instruments, PCI-6036E) presented scripts and SAM questionnaires, as well as acquired and stored physiological data. One monitor was used by the experimenter to track physiological signals, whereas the participant used the other monitor to complete the SAM. Signals were sampled at 250 Hz and collected/filtered using a Model 15LT Bipolar Amplifier (Grass Technologies, West Warwick, RI) with Quad AC (15A54) and Dual DC (15A12) modules. SC was measured using an adaptor (Grass, Model SCA1) for the 15A12, and electrodes filled with isotonic paste (EC33, Grass) were placed on the distal volar surface of the nondominant index and middle fingers. Miniature electrodes to measure corrugator electromyogram (EMG) and lateralis frontalis EMG were placed according to the recommendations of Fridlund and Cacioppo (1986) on the left-side of the face. Electrodes for electrocardiogram (ECG) were placed on the forearms to assess heart rate (HR). Facial EMG and ECG electrodes were applied by degreasing the skin, slightly abrading the skin using NuPrep gel (≤10 KΩ), and filling the electrodes with EC60 gel (Grass). Facial EMG activity was used as a physiologic measure of negative affect (e.g., Orr et al., 1998), because the corrugator pulls the eyebrow down into a frown and the lateralis frontalis raises the outer eyebrow as in displays of fear. HR and SC were used to assess autonomic reactivity, with SC being primarily driven by the sympathetic nervous system (Dawson, Schell, & Filion, 2000).

Procedure

All procedures were approved by the University of Tulsa ethics review board. At an initial laboratory visit, eligible participants were assessed on psychological variables.
(demographics, PTSD symptom severity, depressive symptoms, nightmare characteristics, and sleep problems), and a structured interview (Nightmare Content Interview) assessed details of their nightmares to generate a personal nightmare script. Then, participants were randomly assigned to treatment or a waitlist control group. In a subsequent laboratory visit (referred to as “baseline assessment” from here forward), participants were seated in a recliner, instrumented for facial EMG, SC, and ECG, and then sat quietly during a 5-minute habituation period after the experimenter left the room. Computer-presented instructions guided the participant through a 3-minute diaphragmatic breathing exercise. During script-driven imagery, emotionally charged scripts were presented in random order (randomized within-subjects and between-subjects) and participants were instructed to “imagine it as if it were happening to you.” Physiological reactivity to imagery was derived from 2-minute epochs (30-s baseline, 30-s computer-presented script, 30-s imagery, 30-s recovery). After each 2-minute epoch, the participants rated their subjective reactions to the imagery using the Self-Assessment Manikin and emotion descriptors (i.e., fear, anger, sadness), and then the heart rate was monitored until it returned to prescript levels (generally 1–2 min). Although several scripts were presented to evoke emotional imagery, this study focused only on physiological reactions to personal nightmare scripts. Reactivity to script-driven imagery was assessed again 1 week after Exposure, Relaxation, and Rescripting Therapy or an equivalent waiting period for the control group. At that time, control participants were offered treatment. Reactivity to script-driven imagery was assessed again in all participants at 3-months and 6-months posttreatment. To minimize experimenter bias, experimenters that conducted physiological assessments were blinded to participants’ group assignments. Participants received a $20 gift card for each completed assessment period.

**Physiological Data Reduction**

ECG was converted offline to heart rate (HR) in beats per minute (BPM). The raw signals for HR, SC, and facial EMG were visually inspected for artifacts in five s bins. Bins that did not contain artifacts were then averaged into 30-s phases (baseline, script, imagery, recovery). Physiological reactivity due to script-driven imagery was defined as the average change in the raw physiological signals during the 30-s imagery phase minus the average activity during the 30-s baseline phase.

**Data Analysis**

**Preliminary analyses.** Preliminary analyses were conducted to determine whether interpretations of treatment outcomes for the controlled portion of this study (i.e., baseline assessment and posttreatment/baseline 2; see Fig. 1) may have been confounded by attrition. To do so, chi-square analyses (for nominal variables) and independent samples t-tests (for interval or ratio scale variables) were used to compare those who completed assessments 1 and 2 with those who did not on relevant study variables assessed at baseline (demographics, sleep quality, psychological symptoms, physiological-emotional reactivity).

**Analysis of treatment response.** First, to assess treatment response relative to the control group, a 2 (Time: baseline assessment vs. posttreatment/baseline #2) × 2 (Group: treatment vs. waitlist) mixed effects ANOVA was conducted separately for each physiological (corrugator EMG, lateralis frontalis EMG, HR, SCR) or
subjective (SAM displeasure, SAM arousal, fear, anger, sadness) outcome using the SPSS 14.02 MIXED procedure. Participant ID number was used as the grouping variable, and the error structure of repeated measures was modeled as AR1. A major advantage of the MIXED procedure is that cases with incomplete or missing data are not dropped from the analysis. Thus, participants who dropped out are still included in the analyses. However, for comparison, a second set of analyses were conducted that carried the last value forward to replace missing data to see whether this altered the conclusions.

To determine the magnitude of the treatment effects for the comparison to waitlist controls, first the change over time for the treatment group was defined as follows: Cohen’s $d_{\text{treatment}} = (\text{posttreatment mean} - \text{baseline mean}) / \text{pooled standard deviation}$. Then, change over time for the control group was defined as follows: Cohen’s $d_{\text{control}} = (\text{baseline assessment 2 mean} - \text{baseline assessment 1 mean}) / \text{pooled standard deviation}$. The treatment effect size was defined as Cohen’s $d_{\text{treatment}} - d_{\text{control}}$. Cohen (1988) provides guidelines for interpreting $d$ (small = 0.20, medium = 0.50, large = 0.80).

In the second set of analyses, the participants in the waitlist control group who completed the treatment were combined with the immediate treatment group to determine the long-term benefits of treatment (i.e., treatment response at 3-month and 6-month follow-up). These analyses were conducted with mixed effects analysis of variances (ANOVAs) that included only the effect of time as a nominal variable with four levels (pretreatment, posttreatment, 3-month, and 6-month). Again, a second set of comparison analyses were conducted after carrying the last value forward to replace missing data.

For all MIXED ANOVAs, significant effects were followed up using Bonferroni corrected mean comparisons to control for family-wise error rate (reported $p$ values are the probability of Type I error after Bonferroni corrections were made). In the SPSS MIXED procedure, degrees of freedom for the denominator of $F$ tests are estimated using Satterthwaite approximation and are, therefore, reported with decimals.

Hypotheses

For the analyses that compared active treatment to the waitlist control, it was predicted that ERRT would decrease physiological and subjective reactivity to nightmare imagery. Therefore, significant Group $\times$ Time interactions were expected, such that the simple effect of time should be significant for participants randomized to treatment but not the control group. For analyses of the long-term treatment response in all participants (including control participants after being provided treatment), it was predicted that posttreatment gains would be maintained at 3-month and 6-month follow-up periods.

Results

Preliminary Analyses

Data from 1 of the 40 participants was lost due to equipment problems. Participants who completed baseline assessment and posttreatment/baseline 2 (completers, $n = 30$) did not differ on any relevant variables from those who did not complete both assessments (noncompleters, $n = 9$), except noncompleters reacted with less subjective arousal to the nightmare imagery (Table 2). Thus, it does not appear that
attrition confounded analyses that compared the treatment group with waitlist controls.

Analysis of Treatment Response: Comparisons to the Waitlist Control Group

Physiological outcomes. Figure 2 illustrates these data. The Group × Time interaction was significant for the following: corrugator EMG, $F(1, 35.34) = 4.93, p = 0.03$; lateralis frontalis EMG, $F(1, 31.46) = 5.17, p = 0.03$; HR, $F(1, 34.32) = 5.51, p = 0.03$; and SC, $F(1, 36.20) = 6.16, p = 0.02$. Bonferroni comparisons that decomposed the interactions indicated that all physiological reactions to nightmare imagery were lower at posttreatment than baseline for the treatment group (all $p$s < 0.01), but physiological reactions of the control group did not change over time (all $p$s > 0.40). Cohen’s $d$ treatment effect sizes were large: corrugator EMG = 1.13; lateralis frontalis EMG = 0.89; HR = 0.93; and SC = 0.99. Together, these results suggest physiological reactivity to nightmare imagery was significantly reduced by CBT.

Analyses of physiological outcomes that used the last value carried forward method to handle missing data resulted in the exact same conclusions. The Group × Time interaction was significant for all physiological measures (all $p$s ≤ 0.046), and the baseline versus posttreatment comparison was significant for the treatment group (all $p$s ≤ 0.01), but no change over time was noted in the control group (all $p$s > 0.40).

Subjective outcomes. Table 1 presents means and standard errors for subjective outcomes. The Group × Time interaction was significant for the following: SAM

Table 2
Comparison of Completers and Noncompleters on Psychological, Sleep, and Baseline Physiological-Emotional Reactivity Variables

<table>
<thead>
<tr>
<th></th>
<th>Completers (n = 30)</th>
<th>Noncompleters (n = 9)</th>
<th>t or χ²</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Psychological symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression symptoms (BDI-2)</td>
<td>19.57 ± 10.89</td>
<td>22.89 ± 14.36</td>
<td>-7.5</td>
<td>.46</td>
</tr>
<tr>
<td>PTSD symptom frequency (MPSS-R)</td>
<td>20.14 ± 10.19</td>
<td>22.75 ± 16.26</td>
<td>-4.3</td>
<td>.68</td>
</tr>
<tr>
<td>PTSD symptom severity (MPSS-R)</td>
<td>24.43 ± 14.36</td>
<td>27.00 ± 18.47</td>
<td>-4.2</td>
<td>.66</td>
</tr>
<tr>
<td>% Current PTSD diagnosis (CAPS)</td>
<td>27% ± 33%</td>
<td>33% ± 33%</td>
<td>.15</td>
<td>.70</td>
</tr>
<tr>
<td>% Lifetime PTSD diagnosis (CAPS)</td>
<td>57% ± 33%</td>
<td>33% ± 33%</td>
<td>1.51</td>
<td>.22</td>
</tr>
<tr>
<td>Sleep problems</td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Global sleep quality (PSQI)</td>
<td>12.86 ± 4.70</td>
<td>12.43 ± 5.19</td>
<td>2.2</td>
<td>.83</td>
</tr>
<tr>
<td>PTSD-related sleep problems (PSQI-A)</td>
<td>9.23 ± 4.50</td>
<td>10.67 ± 5.79</td>
<td>-2.7</td>
<td>.07</td>
</tr>
<tr>
<td>Baseline physiological-emotional reactions</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Corrugator EMG (ΔµV)</td>
<td>8.41 ± 9.55</td>
<td>2.69 ± 4.79</td>
<td>1.63</td>
<td>.11</td>
</tr>
<tr>
<td>Lateralis frontalis EMG (ΔµV)</td>
<td>1.24 ± 3.27</td>
<td>1.28 ± 3.63</td>
<td>-0.3</td>
<td>.98</td>
</tr>
<tr>
<td>Heart rate (Δbpm)</td>
<td>6.36 ± 7.05</td>
<td>2.76 ± 5.69</td>
<td>1.33</td>
<td>.19</td>
</tr>
<tr>
<td>Skin conductance level (ΔµS)</td>
<td>0.99 ± 1.09</td>
<td>0.74 ± 0.82</td>
<td>0.60</td>
<td>.55</td>
</tr>
<tr>
<td>SAM displeasure rating (1–9)</td>
<td>8.53 ± 0.82</td>
<td>7.75 ± 1.75</td>
<td>1.22</td>
<td>.26</td>
</tr>
<tr>
<td>SAM arousal rating (1–9)</td>
<td>8.10 ± 1.27</td>
<td>6.88 ± 2.03</td>
<td>2.13</td>
<td>.04</td>
</tr>
<tr>
<td>Fear rating (0–20)</td>
<td>15.93 ± 4.92</td>
<td>17.13 ± 3.64</td>
<td>-0.64</td>
<td>.52</td>
</tr>
<tr>
<td>Anger rating (0–20)</td>
<td>9.70 ± 7.06</td>
<td>9.50 ± 8.11</td>
<td>.07</td>
<td>.95</td>
</tr>
<tr>
<td>Sadness rating (0–20)</td>
<td>12.43 ± 6.64</td>
<td>13.75 ± 7.72</td>
<td>-0.48</td>
<td>.63</td>
</tr>
</tbody>
</table>

Note: BDI-2 = Beck Depression Inventory, 2nd edition; PTSD = posttraumatic stress disorder; MPSS-R = Modified PTSD Symptom Scale Self Report; CAPS = Clinician-Administered PTSD Scale; PSQI = Pittsburgh Sleep Quality Index; EMG = electromyogram; SAM = Self-Assessment Manikin.
displeasure ratings, $F(1, 30.68) = 13.36$, $p = 0.001$; fear ratings, $F(1, 36.49) = 16.22$, $p < 0.001$; sadness ratings, $F(1, 31.85) = 8.34$, $p = 0.007$; and SAM arousal ratings, $F(1, 31.77) = 4.84$, $p = 0.04$. Bonferroni comparisons that decomposed the interactions indicated that ratings of displeasure, fear, sadness, and arousal were lower at posttreatment compared to baseline for the treatment group (all $ps \leq 0.001$), but subjective reactions of the control group did not change over time (all $ps > 0.31$). Although mean levels of anger were lower at posttreatment than baseline in the treatment group (all $ps < 0.001$), but subjective reactions of the control group did not change over time (all $p > 0.31$). Cohen’s $d$ treatment effect sizes were small to large: SAM displeasure $d = 1.24$; fear $d = 1.20$; anger $d = 0.46$; sadness $d = 0.90$; SAM arousal $d = 0.68$. Together, these analyses suggest that, except for anger, negative emotional reactions to nightmare imagery were reduced but only in the treatment group.

Analyses of subjective outcomes that used the last value carried forward method to handle missing data resulted in the same conclusions, except that the Group $\times$ Time interaction for arousal ratings was not significant ($p = 0.06$). However, for displeasure, fear, and sadness ratings, the Group $\times$ Time interaction was significant (all $ps \leq 0.012$) and the baseline versus posttreatment comparison was significant for the treatment group (all $ps \leq 0.001$), but there was no change over time in the control group (all $ps > 0.37$). Like the analysis with missing data, anger ratings were not affected by treatment (Group $\times$ Time interaction: $p = 0.30$).

Analysis of Treatment Response: Follow-up Analyses

For follow-up analyses, the assessment period most proximal to treatment delivery was used as time 1 (baseline assessment for the treatment group, baseline assessment 2 for the control group; see Fig. 1). Four participants in the control group dropped out before baseline assessment 2, which resulted in available data from 35 participants for these analyses.

Physiological outcomes. Figure 3 presents these data. The main effect of time was significant for the following: corrugator EMG, $F(3, 66.67) = 5.35$, $p = 0.002$; lateralis frontalis EMG, $F(3, 45.31) = 3.88$, $p = 0.02$; HR, $F(3, 65.83) = 6.07$, $p = 0.001$; and SC, $F(3, 50.36) = 10.71$, $p < 0.001$. Bonferroni tests for corrugator EMG and SC reactivity suggested mean comparisons were significant for baseline versus posttreatment ($p \leq 0.006$), baseline versus 3-month ($p \leq 0.02$) and baseline versus 6-month follow-up ($p \leq 0.03$). But, tests that compared means at posttreatment, 3-month, and 6-month assessments were all nonsignificant (all $p > 0.99$) for corrugator EMG and SC reactivity. Lateralis frontalis reactivity was lower during posttreatment than baseline ($p = 0.006$), but all other Bonferroni comparisons were nonsignificant (all $ps > 0.12$). HR reactivity decreased from baseline to posttreatment ($p = 0.002$) and 6-month follow-up ($p = 0.01$), but all other comparisons were nonsignificant (all $ps > 0.18$). Together, these analyses suggested corrugator EMG and SC showed consistent long-term maintenance, whereas reductions in HR reactivity were maintained only at 6-month follow-up, and reductions in lateralis frontalis EMG were not maintained at 3-month or 6-month follow-up. Analyses of physiological outcomes that used the last value carried forward method to handle missing data resulted in the exact same conclusions.

Subjective outcomes. Table 3 presents these data. The main effect of time was significant for the following: SAM displeasure ratings, $F(3, 55.81) = 5.35$, $p < 0.001$; fear ratings, $F(3, 50.40) = 18.04$, $p < 0.001$; anger ratings, $F(3, 46.63) = 5.76$,
Long-term effects of cognitive-behavioral treatment for nightmares on physiological reactions to nightmare imagery. Data depicted are from all participants, including those randomized to delayed treatment (waitlist control group). Graphs on the left are facial electromyographic (EMG) reactions (corrugator = left eyebrow muscle, lateralis frontalis = left forehead muscle), whereas graphs on the right are autonomic responses (heart rate [HR]; skin conductance, SC). Error bars are standard error of the mean (SEM). *Indicates significant Bonferroni adjusted mean comparison to baseline (p < 0.05).

Table 3
Subjective Reactions to Nightmare Imagery for Follow-Up Analyses (N = 35)

<table>
<thead>
<tr>
<th>Outcome variable</th>
<th>Baseline</th>
<th>Posttreatment</th>
<th>3-month follow-up</th>
<th>6-month follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SEM</td>
<td>Mean</td>
<td>SEM</td>
</tr>
<tr>
<td>SAM displeasure (1–9)</td>
<td>8.49</td>
<td>0.31</td>
<td>6.69*</td>
<td>0.35</td>
</tr>
<tr>
<td>Fear (1–20)</td>
<td>15.63</td>
<td>0.98</td>
<td>8.48*</td>
<td>1.12</td>
</tr>
<tr>
<td>Anger (1–20)</td>
<td>10.09</td>
<td>1.21</td>
<td>7.90</td>
<td>1.29</td>
</tr>
<tr>
<td>Sadness (1–20)</td>
<td>12.49</td>
<td>1.12</td>
<td>6.97*</td>
<td>1.23</td>
</tr>
<tr>
<td>SAM arousal (1–9)</td>
<td>7.66</td>
<td>0.32</td>
<td>6.02*</td>
<td>0.37</td>
</tr>
</tbody>
</table>

Note: SEM = Standard error of the mean; SAM = Self-Assessment Manikin.
*Indicates statistically significant comparison to baseline based on Bonferroni adjusted tests (p < 0.05).
assessments. However, reductions in anger were not noted at posttreatment, only at 3-month and 6-month follow-ups. Analyses of subjective outcomes that used the last value carried forward method to handle missing data resulted in the exact same results.

Discussion

This study assessed the influence of a CBT (ERRT) for chronic nightmares on physiological and subjective responses to nightmare-related imagery. Physiological reactions to nightmare imagery (corrugator EMG, lateralis frontalis EMG, HR, SC) were reduced following treatment, relative to pretreatment baseline. This effect was not noted after an equivalent wait period in persons randomized to the control group, which suggests reductions in physiological reactivity noted in the treatment group are not due to habituation or exposure to the nightmare script. Important, treatment effect sizes for reductions in physiological reactivity were large (Cohen’s $d = 0.89$ [lateralis frontalis] to 1.13 [corrugator]), even after removing any effect due to changes over time in the control group. Moreover, follow-up analyses that included all participants after being treated indicated reductions in corrugator and SC were maintained 3-months and 6-months posttreatment. We found it interesting that HR reactivity to nightmare imagery, while reduced at posttreatment, was not maintained at the 3-month assessment. However, a significant reduction in HR was noted at the 6-month follow-up relative to pretreatment. Although it is unclear at this time why HR gains may be more variable, it does underscore the importance of measuring follow-up beyond 3-months posttreatment. Treatment gains in lateralis frontalis EMG were not maintained at the 3-month or 6-month assessments. Although more data are needed to explain this observation, it may stem from a floor effect that made significant reductions in lateralis frontalis hard to detect, because lateralis frontalis reactivity at baseline was relatively small (e.g., < 2 DmV compared with > 8 DmV in corrugator EMG at baseline; Fig. 2).

Treatment-related changes in subjective reactions were also noted. Displeasure, fear, sadness, and arousal ratings of nightmare imagery were decreased following treatment, an effect that was not observed in the control group after an equivalent wait period. However, anger was not significantly decreased at the 1-week posttreatment assessment. Effect sizes for treatment-related changes in subjective reactions were generally large (Cohen’s $d = 0.90$ [sadness] to 1.24 [displeasure]), although effect sizes for arousal ($d = .68$) and anger ($d = .46$) were smaller. Follow-up analyses on all participants suggested treatment gains in subjective reactions were maintained at 3-month and 6-month assessments, with anger ratings only showing a significant reduction at the 3-month and 6-month assessments but not 1-week posttreatment. This suggests nightmare-related anger may persist for some time after treatment is over, and it may reflect a consequence of nightmare alleviation (rather than a precursor), given that the number of nightmares experienced per week is an outcome that also showed a delayed response to treatment in the current study (Davis et al., 2009).

Together, these results suggest CBT of nightmares results in significant reductions of physiological and subjective reactions to nightmare-related imagery. To our knowledge, this is the first study to demonstrate beneficial effects on objective measures of fear. These findings add to the growing body of literature, suggesting psychological treatments for chronic nightmares can be effective. For example, uncontrolled (Forbes, Phelps, & McHugh, 2001; Forbes et al., 2003) and controlled
(Davis & Wright, 2007; Krakow et al., 2001, 2000; Krakow, Kellner, Pathak, & Lambert, 1995) studies have found that brief CBT is effective in reducing not only subjective reports of nightmare frequency and severity but also symptoms of psychological distress not directly targeted (e.g., posttraumatic stress symptoms, depression). These conclusions were further supported by analyses from the current randomized, controlled trial (reported elsewhere) that indicated ERRT led to improvements on several variables related to sleep and psychological functioning (e.g., nightmare frequency, nightmare severity, hours slept, sleep quality, depression symptoms, panic symptoms upon waking, PTSD symptom frequency), improvements that were maintained at follow-up (Davis et al., in preparation; Davis et al., 2008).

This study demonstrated that thinking about nightmare-related content elicited significant facial muscle tension, HR acceleration, and sympathetic nervous system activation (i.e., skin conductance) prior to treatment. Such responses are likely to reflect the activation of a pathological fear network that could promote nightmare maintenance and sleep disturbance (Foa & Kozak, 1986; Lang, 1979; Levin & Nielsen, 2007). Although not addressed in the current study, it is tempting to hypothesize that heightened physiological reactivity elicited by nightmare-related thoughts at bedtime would interfere with getting to sleep, sleep quality, and returning to sleep after awakening. Consistent with this, research from another laboratory suggests increased HR leads to longer sleep onset latency (Bonnet & Arand, 2005), and our research group has shown that HR and SC evoked by nightmare imagery are associated with self-reported global sleep problems, time taken to fall asleep, hours slept per night (negative correlation), and the degree of panic upon awakening from a nightmare (Rhudy et al., 2008). To address this issue further, Davis and colleagues (in preparation) analyzed data from the current trial to determine whether physiological reactivity to nightmare imagery was associated with treatment-related changes in sleep disturbance and psychological functioning. They found that persons with higher HR reactivity to nightmare imagery at pretreatment baseline demonstrated greater treatment gains in sleep quality, hours slept per night, and depressive symptoms, and that treatment-related decreases in HR reactivity were associated with improvements in sleep quality and depressive symptoms. Thus, there appears to be some overlap between improvements in physiological reactions and traditional outcome measures.

But, physiological reactions may also provide unique and important information not assessed by other variables. For example, frequent or chronic activation of the fear network by nightmares and nightmare-related thoughts could increase stress and allostatic load, the long-term consequences of which could put nightmare sufferers at risk for cardiovascular or other metabolic diseases (Blanchard, 1990; Boscarino & Chang, 1999; McEwen, 1998). Thus, to the degree that physiological reactivity to nightmare imagery assesses activation of the fear network, physiological reactivity may index risk for future health problems. In line with this notion, preliminary data from our laboratory found that HR acceleration evoked by nightmare imagery is positively correlated with self-reported physical symptoms (Rhudy et al., 2008). Although additional longitudinal studies assessing objective health-related outcomes are needed to draw firm conclusions regarding the direction of these effects, the results from the present study suggest that psychological interventions could improve physical health, sleep, and psychological distress by reducing physiological reactivity associated with nightmare-related cognitions. These results also underscore the importance of evaluating both subjective and physiological outcomes in clinical trials of nightmare treatments.
The present study had limitations that should be noted. First, the relatively small sample size and the fact that participants were treatment seeking may limit the generalizability of the results. Although it is important to replicate these findings in a different, more diverse sample, the small sample does not appear to pose a problem for Type II error, because treatment-related effect sizes were generally large. Thus, the sample size provided adequate statistical power. Second, although the dropout rate of 21% (from baseline to 1-week posttreatment) is not unusual for psychological treatments, it may suggest these participants may have been more symptomatic. Nevertheless, analyses indicated that noncompleters did not differ from those that completed the study, with the exception that noncompleters reported less subjective arousal to the nightmare imagery. Although this may suggest they were functioning somewhat better than completers, it may also indicate greater psychological avoidance. And finally, our procedures did not have a way to control for physiological-emotional reactivity due to the personal relevance of the nightmare script. However, physiological responses to the nightmare imagery did not change over time for the waitlist control group, but they did change for the treatment group. This pattern of results would not be expected if the physiological reactivity were due solely to personal relevance. Nonetheless, to overcome this last problem, we recommend that future studies include a control script that is personally relevant but nightmare-irrelevant.

Despite these limitations, the current study suggests CBT (ERRYT) may reduce physiological-emotional arousal to nightmare imagery, an effect that could have long-term positive implications for physical and psychological health. Future research is needed to determine the relationship between reductions in physiological-emotional reactivity and other objective (e.g., polysomnogram, medical utilization) and subjective (e.g., sleep diaries) indicators of treatment outcome.

References


