Physiological–Emotional Reactivity to Nightmare-Related Imagery in Trauma-Exposed Persons With Chronic Nightmares

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Script-driven imagery was used to assess nightmare imagery-evoked physiological–emotional reactivity (heart rate, skin conductance, facial electromyogram, subjective ratings) in trauma-exposed persons suffering from chronic nightmares. Goals were to determine the efficacy of nightmare imagery to evoke physiological–emotional reactivity, correlates (mental health, nightmare characteristics) of reactivity, and consequences (sleep and health problems) of reactivity. Nightmare imagery resulted in significant reactivity relative to control imagery. No mental health variable (posttraumatic stress disorder status, depressive symptoms, dissociation) or nightmare characteristic (months experienced, frequency, similarity to trauma) was associated with reactivity level. However, nightmare imagery-evoked autonomic responses were associated with greater sleep disturbance and reported health symptoms, even when nightmare frequency was controlled. These results suggest nightmare-related autonomic reactions may contribute to sleep and health disturbance.

Chronic nightmares threaten psychological well-being, a fact that may be especially true for trauma-exposed persons. Presence of nightmares following trauma exposure is associated with greater sleep disturbance (Resnick & Newton, 1992),

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posttraumatic stress disorder (PTSD) symptom severity (Erman, 1987; Esposito, Benitez, Barza, & Mellman, 1999), and functional impairment (Davis, Rhudy, Byrd, & Wright, 2007; Schreuder, Kleijn, & Rooijmans, 2000). Nightmares appear to be particularly problematic for trauma-exposed persons who go on to develop PTSD. The presence of nightmares and sleep disturbances in the immediate aftermath of a traumatic event are associated with current and consequent symptom severity (Koren, Arnon, Lavie, & Klein, 2002; Mellman, David, Bustamante, Torres, & Fins, 2001). Specifically, studies find that the presence and severity of nightmares after a trauma are associated with overall levels of reported distress and overall severity of re-experiencing symptoms (Erman, 1987; Esposito et al., 1999; Schreuder et al., 2000). Research indicates that 50% to 88% of individuals with PTSD report chronic nightmares (Forbes, Creamer, & Biddle, 2001; Kilpatrick et al., 1998; Neylan et al., 1998), a prevalence rate that is higher than the prevalence for trauma-exposed persons without PTSD (Neylan et al., 1998). There is also evidence that among persons reporting chronic nightmares, those with PTSD experience greater nightmare-related distress (van der Kolk, Blitz, Burr, Sherry, & Hartmann, 1984). Although chronic nightmares are particularly problematic for those with PTSD, PTSD-negative individuals still experience significant nightmare-related psychological problems and difficulties (Davis et al., 2007; Schreuder et al., 2000). Moreover, successful cognitive-behavioral treatments (CBTs) that specifically target chronic nightmares reduce symptoms of PTSD, depression, and panic; therefore, chronic nightmares could be a significant maintaining factor of psychological distress (Davis & Wright, 2007; Forbes et al., 2003; Germain & Nielsen, 2003; Krakow et al., 2001; Krakow et al., 2000).

Although the negative effects of chronic nightmares likely stem from many factors, physiological–emotional arousal subsequent to nightmare-related thoughts and cues may contribute. For example, thoughts related to nightmares could elicit significant physiological arousal that increases the latency to sleep, thus promoting nightmare-related distress, decreased sleep quality and quantity, and perhaps nightmare maintenance. Moreover, chronic physiological arousal is known to have a detrimental impact on physical and mental health (McEwen, 1998). Thus, repeated exposure to nightmares and nightmare-related stimuli could place patients at increased risk for developing chronic physical problems, such as cardiovascular disease (Blanchard, 1990; Boscarino & Chang, 1999). Although research finds heightened arousal in terms of panic symptoms upon waking from a nightmare (Davis et al., 2007; Davis & Wright, 2007), it is unclear whether heightened arousal may also be experienced with waking exposure to nightmare cues. To date, no research has examined this issue.

By contrast, numerous studies have examined reactivity to trauma cues in persons exposed to trauma. These studies consistently find that PTSD-positive individuals have a heightened physiological response to trauma cues compared
with PTSD-negative individuals (Carson et al., 2000; Lindauer et al., 2006; Orr et al., 1998; Pitman et al., 2001; Pole, 2007; Schmahl et al., 2004; Shin et al., 2004). Research also finds that heightened reactivity is specific to trauma cues and is not evidenced in response to other fear or stress–related stimuli (Orr et al., 1998; Orr, Pitman, Lasko, & Herz, 1993; Pitman et al., 1990; Pitman, Orr, Forgue, de Jong, & Claiborn, 1987; Shalev, Orr, & Pitman, 1993). Greater physiological–emotional reactivity has further been shown to have relevance in predicting chronic PTSD and treatment response, with heightened reactivity generally being associated with better treatment outcome (Orr & Roth, 2000).

Although nightmare content is not always associated with traumatic events in persons exposed to trauma (Halliday, 1987; Hartmann, 1998), nightmare content is likely to evoke fear-like subjective and physiological reactivity. Thus, procedures used to study physiological–emotional reactivity to trauma cues in persons with PTSD (e.g., script-driven imagery) can be adapted to study reactivity to nightmare cues. If nightmare cues are found to elicit significant physiological–emotional arousal, it would be possible to examine whether participant characteristics influence the degree of reactivity (e.g., PTSD status), whether individual differences in the degree of physiological–emotional reactivity correlate with health and sleep problems, or both.

The present study was designed to examine physiological–emotional reactivity to nightmare-related imagery relative to standard emotionally evocative imagery in trauma-exposed persons (with and without PTSD) who experience chronic nightmares. Physiological reactivity was assessed via four measures. Activity in the corrugator (frowning) and frontalis (outer eyebrow raise) muscles was assessed by electromyogram (EMG) because activity in these muscles has been shown to positively correlate with the subjective experience of negative affect (e.g., Lang, Greenwald, Bradley, & Hamm, 1993; Orr, McNally, Rosen, & Shalev, 2004). Heart rate (HR) and skin conductance (SC) were measured to assess autonomic reactivity, with SC being specifically tied to sympathetic activation (Dawson, Schell, & Filion, 2000). In addition, subjective emotional reactivity was assessed from ratings of emotional valence (unpleasantness–pleasantness) and arousal (calm–excited; Bradley & Lang, 1994).

We predicted that nightmare-related imagery would lead to significantly greater physiological–emotional reactivity than other emotionally evocative imagery. We also expected that poorer mental health (due to reduced coping resources) and more severe nightmares would correlate with greater reactivity to nightmare imagery. Because evidence suggests dissociation is associated with reduced physiological reactivity to fear cues (Griffin, Resick, & Mechanic, 1997; Lanius et al., 2002; Pole et al., 2005), we predicted that dissociation would be negatively associated with reactivity to nightmare imagery. Finally, it was expected that greater physiological reactivity to nightmare imagery would correlate with greater sleep disturbance and more self-reported health...
problems. If these research hypotheses are supported, this study would represent the first step toward developing methods to more comprehensively assess psychophysiological variables that contribute to chronic nightmares and their maintenance (Lang, 1988; Orr & Roth, 2000).

**METHOD**

**Participants**

Men \((n = 10)\) and women \((n = 25)\) from the community were recruited to participate in an ongoing study evaluating the efficacy of a CBT for chronic nightmares in trauma-exposed persons. Ages ranged from 22 to 63 years \((M = 39, SD = 12.02)\). Most were married \((38\%)\) or divorced \((21\%)\), and \(82\%\) had at least some college education. The most frequent types of trauma reported were car accident \((60\%)\), unwanted sexual contact \((57\%)\), physical assault with a weapon \((51\%)\), and physical assault without a weapon \((49\%)\). Thirty-eight percent met criteria for current PTSD, and \(53\%\) met criteria for lifetime PTSD. Six participants dropped out before physiological assessments could be conducted, and 1 participant was not exposed to their nightmare script due to equipment problems; therefore, the final sample included \(28\) participants. Participants were recruited by fliers, e-mail, and radio ads and were eligible to participate if they were \(\geq 18\) years old, had previous exposure to a traumatic event, and had nightmares \(\geq 1\) times per week for the previous 3 months. Exclusion criteria were psychosis or mental retardation, active suicidality or parasuicidal behaviors, and current drug or alcohol dependence. Participants were asked to refrain from consuming alcohol, nicotine, or caffeine \(24\) hr prior to the physiological assessments. Participants were not excluded for medication use, but preliminary analyses suggested that neither psychotropic medications nor other medications (e.g., beta blockers, analgesics) altered physiological–emotional reactivity in the present study (all \(p \geq .05\)). A \$20\) gift certificate was provided for participation.

**Procedure**

All procedures were approved by the University of Tulsa institutional review board. After an initial phone screening, eligible participants underwent a diagnostic evaluation (baseline psychological assessment) by a trained upper level graduate student after informed consent was obtained. During this evaluation, questionnaires and structured interviews (see later discussion) were administered to assess inclusion or exclusion criteria as well as demographics, PTSD status,
PTSD symptom severity and frequency, depressive symptoms, dissociative experiences, nightmare characteristics, sleep problems, and health problems. Furthermore, the Nightmare Content Interview (NCI) was used to obtain nightmare content that was used later by different experimenters to generate a personalized nightmare script for physiological assessment of script-driven imagery. (The participant was not involved in the script generation.) Following this psychological evaluation, participants were randomly assigned to CBT for nightmares or a waitlist control group and then scheduled for the baseline physiological assessment conducted on a later day. For this study, only data collected during psychological and physiological baseline assessments were used. Between the psychological and physiological assessment, experimenters generated a 30-sec script (approximately 100 words) from the nightmare content. This script and the standard emotional scripts (see later discussion) were recorded onto the computer in the voice of the third author (Amy E. Williams).

During the physiological assessment, participants were seated comfortably in a recliner and instrumented for facial EMG, SC, and electrocardiogram (ECG) following a thorough explanation of the procedures. Once the experimenter left the room, the session started with a 5-min habituation period followed by a 3-min relaxation training period (diaphragmatic breathing) that was presented by computer. During script-driven imagery, five scripts (see later discussion) were presented in random order during which physiological responses were recorded. Physiological reactivity to each script was derived from a 2-min recording epoch (30-sec baseline period, a 30-sec period during which the script was presented over headphones by computer, a 30-sec imagery period in which the participant was asked to engage in the script imagery “as if it were happening to them,” and a 30-sec recovery period in which the participant was asked to clear their mind and relax). After each recovery period, the participant rated their emotional reaction to the script-driven imagery using the Self-Assessment Manikin (SAM; Bradley & Lang, 1994), and then HR was monitored to ensure that it returned to pre-script baseline before the next recording epoch began (generally 1–2 min). Once all scripts were presented, participants were thanked for their time.

**Emotional Scripts**

Every script used to generate imagery was approximately 100 words long. The script was recorded digitally in a slow-paced, monotone voice such that the length was approximately 30 sec. Each participant’s personal nightmare script was constructed from subjective and sensory content assessed by the NCI (see later discussion) and written in second person. Non-personal, standard scripts obtained from other researchers using script-driven imagery (Peter Lang, personal communication, April 16, 2004; Scott Orr, personal communication, January 27, 2004) were used as control stimuli and included the following content: pleasant
NIGHTMARE-RELATED IMAGERY

("You are lying on a sandy beach on a warm summer day.... You feel relaxed and content, enjoying the warmth of the sun...."), neutral ("You are sitting in a lawn chair on your porch on a summer afternoon...."), action ("... You are riding your bicycle on a quiet country road. You breathe heavily and sweat runs down your face...."), and fear ("... You have never addressed such a large group before. Your palms have become sweaty, and you tense up....").

Measures

Demographic questionnaire. The demographic questionnaire is a self-report measure designed to obtain standard information about the participants including age, marital status, educational achievement, ethnicity, vocational status, and household income.

Clinician-Administered PTSD Scale (CAPS; Blake et al., 1990). The CAPS is a structured interview that assesses lifetime and current PTSD from 17 questions. The symptom endorsement method 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., 1990; Weathers et al., 1999). A CAPS total score was created by summing frequency and intensity scores for all items.

Beck Depression Inventory–Second Edition (BDI–II; Beck, Steer, & Brown, 1996). The BDI–II is a reliable, 21-item self-report inventory used to assess depression symptoms. Responses are made on a 4-point scale, yielding total scores ranging from 0 to 63.

Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). The PSQI is a reliable and valid self-report instrument that assesses sleep problems and quality for the 1 month prior to the assessment (Buysse et al., 1989). A global sleep problem score ranges from 0 to 21 (higher scores = poorer quality), and is obtained by summing seven component scores for the following: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medication, and daytime dysfunction. Single items were used to assess minutes taken to fall asleep and hours slept per night.

PSQI Addendum (PSQI-A) for PTSD (Germain, Hall, Krakow, Shear, & Buysse, 2005). The PSQI–PTSD Addendum (PSQI-A) is a seven-item scale that assesses PTSD-related sleep and nighttime behaviors. The items assess frequency of sleep problems in the past month ranging from 0 (not during the
past month) to 3 (three or more times per week). Scores range from 0 to 21. The PSQI-A has an internal consistency of .85 and good convergent validity.

**Modified PTSD Symptom Scale–Self-Report (MPSS–SR; Resnick, Best, Kilpatrick, Freedy, & Falsetti, 1993).** This reliable and valid instrument assesses the frequency (0 = not at all, 3 = five or more times per week/very much/almost always) and severity (0 = not at all distressing, 4 = extremely distressing) of 17 PTSD symptoms (Falsetti, Resick, Resnick, & Kilpatrick, 1992, as cited in Falsetti, Resnick, Resick, & Kilpatrick, 1993; Wright, Davis, Inness, & Stem, 2003).

**Trauma-Related Nightmare Survey (Davis, Wright, & Borntrager, 2001).** This scale assesses nightmare symptoms as well as nightmare-related cognitions, emotions, and behaviors. The panic upon waking subscale is a sum of 14 endorsed symptoms (e.g., chest pain or discomfort, numbness or tingling sensations, derealization) assessed from the following prompt: “After waking from the nightmare, do you experience any of the following symptoms?” Internal consistency was α = .83. A single item assessed whether nightmares were “exactly or almost exactly like the trauma,” “similar to the trauma,” or “unrelated to the trauma” (nightmares similar/replicates trauma).

**Dissociative Experiences Scale (DES; Bernstein & Putnam, 1986).** The DES is a reliable, self-report questionnaire that assesses frequency of 28 dissociative experiences on a 0% to 100% scale with 10-point intervals (Carlson & Putnam, 1993; Dubester & Braun, 1995).

**Pennebaker Inventory of Limbic Languidness (PILL; Pennebaker, 1982).** This reliable, 54-item, self-report questionnaire assesses the frequency of common physical symptoms. It is scored using a 5-point Likert scale that ranges from 0 (have never or almost never experienced the symptom) to 4 (experiencing the symptom more than once a week).

**Nightmare Content Interview (NCI).** This structured interview was used to gather content from the participant’s most recurrent nightmare, or most recent nightmare if a single recurring nightmare was not available, and create personal nightmare scripts. Participants were asked, “First, I would like you to briefly describe your recurrent nightmare,” and then follow-up questions probed for additional cognitive (“Tell me what was going through your head…”), somatic (“What is your heart doing, are you sweating?…”), and sensory information (“What did you hear, see, smell, taste?”).
**Self-Assessment Mankin (SAM) (Bradley & Lang, 1994).** A computerized version of the SAM (Rhudy, Williams, McCabe, Nguyen, & Rambo, 2005) assessed affective valence (unpleasant–pleasant) and arousal (calm–excited) reactions to script-driven imagery. The SAM is a two-item questionnaire that consists of two sets of five pictographs depicting affective valence/pleasure and arousal. To respond, an indicator was placed on or between any of the five pictographs, and ratings ranged between 1 and 9 for each dimension (higher scores = greater pleasure–valence or arousal).

**Apparatus and Physiological Outcomes**

Sound-attenuating headphones and a video camera allowed the experimenter to communicate with and monitor the participant from an adjacent room. A computer with dual 17-in. flat-panel monitors and A/D board (National Instruments, PCI–6036E) presented all script stimuli and SAM questionnaires, as well as acquired and stored physiological data. One monitor was used by the experimenter to assess physiological signals and experimental timing, whereas the other monitor was used by the participant to complete the SAM questionnaire. All physiological signals were sampled at 250 Hz and collected and filtered using a Grass Instruments Model 15LT Bipolar Amplifier with Quad AC (15A54) and Dual DC (15A12) modules. SC was measured using an adaptor (Grass, Model SCA1) for the 15A12 amplifier, and electrodes filled with isotonic paste (EC33, Grass Instruments) were placed on the distal volar surface of the nondominant index and middle fingers. Miniature electrodes to measure corrugator 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 ECG were placed on the forearms. Facial EMG and ECG electrodes were applied by degreasing the skin, followed by slight skin abrasion using NuPrep gel to reduce impedances below 10 KΩ, and then EC60 gel (Grass Instruments) was placed in the electrodes before application. Facial EMG activity was used as a physiologic measure of negative affect (Orr et al., 1998), whereas ECG and SC were used to assess autonomic reactivity (Dawson et al., 2000).

**Physiological Data Reduction and Analyses**

ECG was converted offline to HR in beats per minute. The raw signals for HR, SC, and facial EMG signals were visually inspected for artifacts in 5-sec bins. Bins that did not contain artifacts were then averaged into 30-sec phases (baseline, script, imagery, recovery). Physiological reactivity was defined as the change in the raw physiological signals during imagery relative to baseline; therefore, the averaged raw signal (for HR, SC, and facial EMG) during the
baseline phase was subtracted from the averaged signal during the imagery phase. These change scores were then subjected to statistical analysis.

Chi-square analyses (for nominal variables) and independent samples t tests (for interval or ratio scale variables) were used to compare participants included in the analysis to those not included on study variables, and to compare participants with and without a current diagnosis of PTSD. To assess whether physiological–emotional reactivity was greater during personal nightmare imagery in persons with and without current PTSD, a 5 (Imagery Type: pleasant, neutral, action, fear, nightmare) × 2 (PTSD Status) doubly multivariate repeated measures analysis of variance was used to analyze all physiological and subjective reactions simultaneously (to control Type-I error rate). (It is noteworthy that results from this analysis were the same when lifetime PTSD was used instead of current PTSD.) However, to interpret significant effects, this analysis was followed up with individual analyses of variance (ANOVA)s on each reaction. When necessary, Greenhouse–Geisser adjustments were used to correct for violations of sphericity in ANOVA models. To control for Type-I error rate, significant main effects of imagery type were followed up by a priori contrasts that only compared nightmare imagery to standard imagery (i.e., comparisons among standard imagery were not conducted). All other significant effects were followed up with Bonferroni adjusted comparisons. Partial eta squared ($\eta^2$) is reported for the effect size of F tests. To provide a measure of effect size for the degree of reactivity, Cohen’s $d$ is reported for the comparison between reactions to nightmare imagery relative to standard fear script imagery.

To determine whether poorer mental health, less dissociative tendencies, and more severe nightmare characteristics correlated with greater physiological–emotional reactivity to nightmare imagery, zero-order correlations were calculated between physiological–emotional reactivity outcomes and current or lifetime PTSD status, PTSD severity, PTSD frequency, depressive symptoms, dissociative experiences, months since nightmares started, nightmare frequency, and similarity of nightmare to trauma. To determine whether physiological reactivity to nightmare imagery correlated with greater sleep disturbance and more reported health problems, zero-order correlations were also calculated between physiological and subjective reactions to nightmare imagery and the global sleep quality index, PTSD-related sleep behaviors, minutes taken to fall asleep, hours slept per night, panic upon awakening, and health symptoms. For comparison, correlations between physiological–emotional reactivity to fear imagery and consequences of nightmares were also conducted. To interpret correlation analyses, a more stringent alpha level ($p \leq .01$) was chosen that reduced family-wise Type-I error, but also took into consideration the problem of augmenting a Type-II error rate.
RESULTS

Characteristics of Participants Included in the Study (Table 1)

The only significant difference found between persons included versus excluded was that those included in the study reported sleeping longer (more hours) per night ($M = 4.57$ vs. $5.96$; $t = 1.99$, $p = .05$). Participants without and with current PTSD were similar on most variables ($ps > .05$) except global sleep problems ($M = 11.00$ vs. $16.00$; $t = 3.27$, $p = .003$), CAPS total score ($M = 44.05$ vs. $82.14$; $t = 7.13$, $p < .001$), PTSD severity ($M = 19.33$ vs. $42.50$; $t = 4.07$, $p < .001$), PTSD frequency ($M = 17.47$ vs. $34.50$; $t = 3.86$, $p = .001$), depressive symptoms ($M = 18.21$ vs. $28.56$; $t = 2.12$, $p = .04$), and

<table>
<thead>
<tr>
<th>Variable</th>
<th>mean or %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean</td>
<td>39.64 (12.06)</td>
</tr>
<tr>
<td>Sex (female)</td>
<td>68%</td>
</tr>
<tr>
<td>Race (White)</td>
<td>75%</td>
</tr>
<tr>
<td>Relationship status (living with other or married)</td>
<td>46%</td>
</tr>
<tr>
<td>Months since nightmare started (TRNS), mean</td>
<td>206.81 (140.87)</td>
</tr>
<tr>
<td>Nightmares per week (TRNS), mean</td>
<td>3.46 (1.82)</td>
</tr>
<tr>
<td>Nightmare similar to or replicates trauma (TRNS)</td>
<td>56%</td>
</tr>
<tr>
<td>Panic upon waking (TRNS), mean</td>
<td>5.44 (3.72)</td>
</tr>
<tr>
<td>Global sleep problems (PSQI), mean</td>
<td>12.80 (4.35)</td>
</tr>
<tr>
<td>PTSD-related sleep behaviors (PSQI-A), mean</td>
<td>9.70 (9.0)</td>
</tr>
<tr>
<td>Minutes taken to fall asleep (PSQI), mean</td>
<td>39.64 (28.67)</td>
</tr>
<tr>
<td>Hours slept per night (PSQI), mean</td>
<td>5.96 (1.54)</td>
</tr>
<tr>
<td>CAPS total score, sum</td>
<td>59.29 (24.32)</td>
</tr>
<tr>
<td>PTSD current (CAPS)</td>
<td>32%</td>
</tr>
<tr>
<td>PTSD lifetime (CAPS)</td>
<td>46%</td>
</tr>
<tr>
<td>PTSD severity (MPSS–SR), mean</td>
<td>26.22 (17.04)</td>
</tr>
<tr>
<td>PTSD frequency (MPSS–SR), mean</td>
<td>22.52 (12.96)</td>
</tr>
<tr>
<td>Depressive symptoms (BDI–II), mean</td>
<td>21.54 (12.80)</td>
</tr>
<tr>
<td>Dissociative experiences (DES), mean</td>
<td>17.00 (15.60)</td>
</tr>
<tr>
<td>Health symptoms (PILL), mean</td>
<td>78.64 (31.61)</td>
</tr>
</tbody>
</table>

Note. N = 28. Standard deviations are shown in parentheses. TRNS = Trauma-Related Nightmare Survey; PSQI = Pittsburgh Sleep Quality Index; PSQI-A = PSQI-Addendum; CAPS = Clinician-Administered PTSD Scale; PTSD = posttraumatic stress disorder; MPSS–SR = Modified PTSD Symptom Scale–Self-Report; BDI–II = Beck Depression Inventory–Second Edition; DES = Dissociative Experiences Scale; PILL = Pennebaker Inventory of Limbic Languidness.
dissociative symptoms ($M = 12.31$ vs. $26.90; t = 2.53, p = .02$). Participants with current PTSD had worse symptomatology.

Physiological–Emotional Reactivity to Nightmare Imagery (Table 2)

The doubly multivariate analysis found a significant main effect of imagery type, $F(24, 305) = 9.38, p < .001, \eta^2 = .38$. No other effects were significant ($ps > .17$). To interpret this main effect, ANOVAs were conducted on each physiological–emotional measure. A significant main effect of imagery type was found for valence (pleasure) ratings, arousal ratings, HR, SC, and corrugator EMG. Lateralis frontalis EMG was not significantly affected by imagery. Compared to all other standard imagery types, nightmare imagery resulted in lower valence (greater displeasure) ratings ($ps < .001$), higher arousal ratings ($ps \leq .01$), larger HR responses ($ps < .01$), and larger SC ($ps < .004$). Corrugator reactivity was greater during nightmare imagery than pleasant and neutral ($ps < .005$), but the comparisons of nightmare imagery to action ($p = .07$) and fear ($p = .08$) imagery did not reach significance. Cohen’s $d$ for the nightmare versus fear imagery comparisons were as follows: valence $= 1.37$, arousal $= 0.79$, HR $= 0.66$, SC $= 0.79$, corrugator EMG $= 0.62$, and lateralis frontalis EMG $= 0.21$. The PTSD status main effect was nonsignificant for all analyses ($Fs < 1.78, ps > .18$). The Imagery Type $\times$ PTSD Status interaction was only significant for HR, and suggested HR responses were lower for persons with PTSD during pleasant and neutral imagery ($ps < .03$); but PTSD versus non-PTSD comparisons for action ($p = .07$), fear ($p = .59$), and nightmare imagery ($p = .15$) were nonsignificant. Together, these data suggest that nightmare imagery resulted in significant physiological–emotional reactivity relative to standard imagery, with medium to large effect sizes for most comparisons between nightmare and fear imagery. It is interesting to note that PTSD status did not have a significant effect on reactivity to nightmare imagery.

Correlates of Physiological–Emotional Reactivity to Nightmare Imagery

No mental health or nightmare variable (PTSD status or symptoms, depressive symptoms, dissociative experiences, or nightmare characteristics) was significantly related to physiological or subjective reactivity to nightmare imagery at the $p \leq .01$ level. The average correlations with outcome variables were generally small (average $rs$: valence $=.12$, arousal $=.16$, HR $=.27$, SC $=.18$, corrugator EMG $=.09$, lateralis frontalis EMG $=.13$). The only correlation that approached significance was the relation between MPSS–SR PTSD symptom frequency and HR ($r = .42, p < .05$).
### TABLE 2
Descriptive and Inferential Statistics (From Univariate ANOVAs) for Physiological and Subjective Reactivity to Imagery Grouped by Imagery Type and PTSD Status

<table>
<thead>
<tr>
<th>Variable</th>
<th>No Current PTSD</th>
<th>Current PTSD</th>
<th>ANOVA Results</th>
<th>Imagery Type</th>
<th>Imagery × PTSD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subjective reactions</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Valence ratings</td>
<td>M 7.83 7.94 5.61 3.61 1.72</td>
<td>8.00 7.78 6.78 4.00 1.56</td>
<td>77.53***</td>
<td>.76</td>
<td>0.79</td>
</tr>
<tr>
<td>SEM 0.34 0.37 0.45 0.46 0.25</td>
<td>0.48 0.53 0.63 0.65 0.35</td>
<td>42.02***</td>
<td>.63</td>
<td>1.37</td>
<td>.05</td>
</tr>
<tr>
<td>Arousal ratings</td>
<td>M 1.89 1.67 5.06 6.00 7.61</td>
<td>2.56 3.33 5.22 6.11 7.11</td>
<td>13.02***</td>
<td>.35</td>
<td>3.91*</td>
</tr>
<tr>
<td>SEM 0.44 0.44 0.47 0.51 0.38</td>
<td>0.62 0.62 0.67 0.72 0.54</td>
<td>15.84***</td>
<td>.39</td>
<td>0.85</td>
<td>.03</td>
</tr>
<tr>
<td>Autonomic reactions</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (Δbpm)</td>
<td>M 0.70 0.86 2.56 2.20 4.09</td>
<td>−2.34 −2.43 −0.35 1.48 8.19</td>
<td>8.64**</td>
<td>.27</td>
<td>0.95</td>
</tr>
<tr>
<td>SEM 0.71 0.45 0.85 0.72 1.52</td>
<td>1.07 0.68 1.27 1.08 2.28</td>
<td>10.02**</td>
<td>.21</td>
<td>1.00</td>
<td>.03</td>
</tr>
<tr>
<td>SC (ΔμS)</td>
<td>M −0.17 −0.14 0.02 0.28 0.96</td>
<td>−0.24 −0.21 −0.22 0.23 1.44</td>
<td>13.02***</td>
<td>.35</td>
<td>3.91*</td>
</tr>
<tr>
<td>SEM 0.06 0.05 0.09 0.20 0.28</td>
<td>0.09 0.07 0.13 0.30 0.42</td>
<td>15.84***</td>
<td>.39</td>
<td>0.85</td>
<td>.03</td>
</tr>
<tr>
<td>Facial EMG reactions</td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Corr. EMG (ΔμV)</td>
<td>M −0.43 −0.63 1.19 1.22 5.71</td>
<td>−2.03 −0.74 2.64 3.04 4.07</td>
<td>8.64**</td>
<td>.27</td>
<td>0.95</td>
</tr>
<tr>
<td>SEM 0.44 0.31 0.98 0.61 1.85</td>
<td>0.65 0.45 1.43 0.88 2.70</td>
<td>10.02**</td>
<td>.21</td>
<td>1.00</td>
<td>.03</td>
</tr>
<tr>
<td>Lat. Fr. EMG (ΔμV)</td>
<td>M −0.15 −0.04 0.14 0.25 0.99</td>
<td>−0.36 0.00 0.16 0.53 0.27</td>
<td>1.02 .04</td>
<td>0.32</td>
<td>.01</td>
</tr>
<tr>
<td>SEM 0.13 0.08 0.09 0.14 0.81</td>
<td>0.20 0.12 0.13 0.20 1.21</td>
<td>1.02 .04</td>
<td>0.32</td>
<td>.01</td>
<td></td>
</tr>
</tbody>
</table>

**Note.** ANOVA = analysis of variance; PTSD = posttraumatic stress disorder; Pleas. = pleasant imagery; Neu. = neutral imagery; Act. = action imagery; NM = personal nightmare imagery; HR = heart rate; Δbpm = change in beats per minute; SC = skin conductance; ΔμS = change in microSiemens; EMG = electromyogram; Corr. = corrugator muscle; ΔμV = change in microvolts; Lat. Fr. = lateralis frontalis muscle.

*p < .05, **p < .01, ***p < .001.
TABLE 3
Zero-Order Correlations Between Physiological–Emotional Reactions to Nightmare Imagery and Problems With Sleep and Health

<table>
<thead>
<tr>
<th>Reactions to Nightmare Imagery</th>
<th>Global Sleep Problems (PSQI)</th>
<th>PTSD Sleep Behaviors (PSQI-A)</th>
<th>Minutes Taken to Fall Asleep (PSQI)</th>
<th>Hours Slept Per Night (PSQI)</th>
<th>Panic Upon Waking (TRNS)</th>
<th>Health Symptoms (PILL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valence rating</td>
<td>−0.09</td>
<td>0.46***</td>
<td>−0.25</td>
<td>−0.02</td>
<td>0.04</td>
<td>0.09</td>
</tr>
<tr>
<td>Arousal rating</td>
<td>0.01</td>
<td>−0.17</td>
<td>0.31</td>
<td>0.04</td>
<td>0.04</td>
<td>−0.06</td>
</tr>
<tr>
<td>HR (Δbpm)</td>
<td>0.55*</td>
<td>0.20</td>
<td>0.41***</td>
<td>−0.59*</td>
<td>0.58*</td>
<td>0.54*</td>
</tr>
<tr>
<td>SC (ΔµS)</td>
<td>0.33</td>
<td>0.19</td>
<td>0.59*</td>
<td>−0.40***</td>
<td>0.63**</td>
<td>0.16</td>
</tr>
<tr>
<td>Corr. EMG (ΔµV)</td>
<td>0.18</td>
<td>−0.05</td>
<td>0.41***</td>
<td>−0.13</td>
<td>−0.01</td>
<td>−0.11</td>
</tr>
<tr>
<td>Lat. Fr. EMG (ΔµV)</td>
<td>−0.01</td>
<td>−0.18</td>
<td>−0.24</td>
<td>0.26</td>
<td>0.22</td>
<td>0.03</td>
</tr>
</tbody>
</table>

Note. Significant correlations are in bold type. PSQI = Pittsburgh Sleep Quality Index; PSQI-A = PSQI-Addendum; TRNS = Trauma-Related Nightmare Survey; PILL = Pennebaker Inventory of Limbic Languidness; HR = heart rate; Δbpm = change in beats per minute; SC = skin conductance; ΔµS = change in microSiemens; Corr. = corrugator muscle; EMG = electromyogram; Lat. Fr. = lateralis frontalis; ΔµV = change in microvolts.

Correlates of Negative Consequences of Chronic Nightmares From Physiological and Subjective Reactivity to Nightmare Imagery (Table 3)

Autonomic reactions were the most reliable correlates of poor sleep and health, demonstrating large effect sizes. HR was associated with increased global sleep problems, fewer hours slept per night, panic upon waking, and health symptoms. SC was associated with time to fall asleep and panic upon awakening. It is interesting to note that subjective reactions were unrelated to these variables. For exploratory purposes, partial correlations were conducted that controlled for number of nightmares per week (nightmare chronicity). All relationships remained significant. More important, physiological–emotional reactions to fear imagery were unrelated to any of these outcomes, with the strongest correlation being between valence ratings and PTSD-related sleep behaviors ($r = -.28, p > .05$). Moreover, Fisher’s $r$-$to$-$z$ tests for non-independent correlations indicated nightmare and fear imagery correlations were different at $p \leq .01$, except between SC and minutes taken to fall asleep ($p = .13$). Therefore, autonomic reactions to nightmare imagery appear related to sleep disturbance and health symptomatology.

DISCUSSION

The present study examined physiological–emotional reactions to nightmare imagery in trauma-exposed persons suffering from chronic nightmares. Results
suggested nightmare imagery increased reports of displeasure (lower valence) and arousal, as well as increased HR, SC, and corrugator (frowning) muscle activity. Lateralis frontalis muscle activity was not affected. Of particular interest is the fact that PTSD status did not moderate reactivity to nightmare imagery. This suggests nightmares may have a significant physiological–emotional impact on all sufferers, regardless of comorbid psychopathology. Although PTSD status did interact with imagery type for HR, this stemmed from the fact that HR decelerated in persons with PTSD during pleasant and neutral imagery compared to persons without PTSD. Results from the ANOVA suggested HR reactivity to nightmare imagery did not differ by PTSD status. This analysis was supported by correlational analyses showing that PTSD symptomatology (current–lifetime PTSD diagnosis, symptom severity, symptom frequency) was not associated with nightmare imagery-evoked HR. One possible explanation for this lack of moderation by PTSD could have been that participants with PTSD had greater anxious apprehension about being exposed to their nightmare. This apprehension could have elevated resting HR, making it difficult to evoke greater reactivity above baseline (i.e., a ceiling effect). This could also explain why HR decelerated during relaxing and pleasing imagery (neutral, pleasant). To minimize anticipation and emotional carry-over effects associated with our within-subjects design, script order was randomized between individuals. However, this tactic may not have eliminated all anticipatory and carry-over effects in the PTSD-positive individuals.

Our prediction that mental health would influence the level of nightmare imagery-evoked physiological–emotional reactivity was not supported. Neither PTSD nor depressive symptoms were significant correlates. This is surprising given that mental health problems in trauma-exposed persons are typically associated with impaired coping (e.g., Stallard & Smith, 2007) and heightened physiological–emotional reactivity to stressful events (Keane et al., 1998; Orr et al., 2004; Shalev, Orr, & Pitman, 1992). Analyses also suggested that dissociative tendencies were not associated with blunted physiological–emotional responses (Griffin et al., 1997). However, this may have resulted from the explicit instructions to engage in imagery rather than avoid it. Future research could address this issue by varying the instruction set.

Results also did not support our prediction that greater nightmare chronicity (months since nightmares started, number of nightmares per week) and similarity of the nightmare to a previously experienced trauma would be associated with greater reactivity. However, these null results need to be interpreted with caution because our small sample size may have reduced our power to detect significant relations (e.g., some correlations between HR reactivity and mental health had medium effect sizes). Power analyses, however, suggested that a large sample size (between 250 and 2,800) would have been needed to detect most of the correlational effects determined to be nonsignificant in the current study.
Even the correlations with HR would have required over 100 participants. In addition, our one-time assessment of physiological reactivity to scripts during this baseline assessment may have led to low reliability and stability of our dependent variables. Inconsistent with this interpretation however, we did find that individual differences in physiological reactivity to nightmare imagery was associated with sleep and self-reported health problems. Consistently, greater HR reactivity was associated with poor sleep and health outcomes. Also, greater SC was associated with longer time taken to fall asleep and greater panic upon waking. It is interesting to note that participants’ subjective or facial emotional reactions were unrelated to sleep and health problems. Therefore, autonomic arousal resulting from nightmare-related thoughts and imagery may play an important role in sleep disturbance. For example, increased autonomic arousal could interfere with getting to sleep, returning to sleep upon awakening from a nightmare, and amount of restful sleep (sleep quality). Indeed, sleep latency has been found to positively correlate with contemporaneous HR (Bonnet & Arand, 2005). In addition, frequent and exaggerated autonomic arousal may increase allostatic load, thus resulting in poorer health (McEwen, 1998). In particular, the finding of increased HR reactivity may place persons suffering from chronic nightmares at increased risk of developing cardiovascular disease (Blanchard, 1990; Boscarino & Chang, 1999). Given the potential importance of autonomic reactivity in nightmare-related problems, this suggests future studies should consider other, more dynamic, measures of autonomic reactivity, such as HR variability.

Together, these results suggest that reducing nightmare-related autonomic arousal could improve sleep and health. However, longitudinal studies are needed to determine the direction of the relations noted in these cross-sectional analyses (perhaps greater sleep disturbance and health problems result in greater nightmare-related autonomic reactivity). Our laboratory is currently following these participants after they undergo CBT for nightmares to determine whether physiological–emotional arousal is impacted by psychological treatment. If so, this would add to the extant literature suggesting psychological (e.g., Davis & Wright, 2007; Forbes, Phelps, & McHugh, 2001; Germain & Nielsen, 2003) and pharmacological (e.g., Raskind et al., 2003) interventions are efficacious for reducing nightmare-related problems.

Study Limitations

In addition to the limitations noted earlier, there are a few other issues worthy of mention. The present study used personal nightmare content to evoke nightmare imagery. In future studies, another personally relevant script unrelated to the nightmare should be used to control for physiological–emotional reactivity due to personal relevance. Although the present design did not allow us to con-
trol for personal relevance, reactivity to the nightmare imagery was large and significantly different from all other imagery types.

In the majority of studies, script-driven imagery is used to examine physiological–emotional reactivity to fear cues, including trauma-reminders. For the present study, we assume that nightmare content, regardless of its relation to an experienced trauma, would serve as a fear cue to elicit physiological–emotional reactivity. Our results are consistent with this hypothesis and suggest that nightmare-related thoughts could promote avoidance of sleep (during which exposure to nightmare content is imminent). Nonetheless, it is important to note that nightmare imagery is different from most other fear cues in that it does not represent a specific environmental or sensory event.

For 56% of participants, the nightmare imagery was trauma related (the nightmare was similar to, or replicated, the participant’s trauma experience). Although analyses suggested the trauma similarity of the nightmare was not associated with the degree of physiological–emotional reactivity to nightmare imagery, our sample size was not large enough to determine whether PTSD status interacted with trauma similarity of the nightmare. For example, it is possible that persons who are PTSD-positive and whose nightmares are related to their trauma have greater physiological–emotional reactivity to nightmare imagery than persons who are PTSD-negative.

Finally, to assess subjective reactions to imagery, the SAM was used. Although the SAM is well-validated and commonly used in studies of subjective reactivity (e.g., Bradley, Codispoti, Cuthbert, & Lang, 2001; Bradley & Lang, 1994), single items are used to measure valence and arousal. Moreover, participants generally rated their nightmare imagery as highly unpleasant (low valence) and arousing, thus limiting response variability. These issues could have impacted our ability to detect significant relations between subjective emotional reactivity and other study variables.

Despite these limitations, this study contributes to our understanding of chronic nightmares and their impact in trauma-exposed persons. It is the first study, to our knowledge, to use script-driven imagery to examine physiological–emotional reactivity to nightmare imagery in persons suffering from chronic nightmares. Although preliminary, these data suggest physiological reactivity may be an important variable to consider when assessing and treating chronic nightmares. In particular, nightmare cue-evoked autonomic reactivity may be an important therapeutic target.

REFERENCES


