Sleep Pathophysiology in Posttraumatic Stress Disorder and Idiopathic Nightmare Sufferers

Anne Germain and Tore A. Nielsen

**Background:** Nightmares are common in posttraumatic stress disorder (PTSD), but they also frequently occur in idiopathic form. Findings associated with sleep disturbances in these two groups have been inconsistent, and sparse for idiopathic nightmares. The aim of the present study was to investigate whether sleep anomalies in PTSD sufferers with frequent nightmares (P-NM) differ from those observed in non-PTSD, idiopathic nightmare (I-NM) sufferers and healthy individuals.

**Methods:** Sleep measures were obtained from nine P-NM sufferers, 11 I-NM sufferers, and 13 healthy control subjects. All participants slept in the laboratory for two consecutive nights where electroencephalogram, electro-oculogram, chin and leg electromyogram, electrocardiogram, and respiration were recorded continuously.

**Results:** Posttraumatic nightmare sufferers had significantly more nocturnal awakenings than did I-NM sufferers and control subjects. Elevated indices of periodic leg movements (PLMs) during rapid eye movement (REM) and non-REM sleep characterized both P-NM and I-NM sufferers.

**Conclusions:** Posttraumatic nightmare sufferers exhibit more nocturnal awakenings than do I-NM sufferers and control subjects, which supports the hypothesis of hyper-arousal in sleep in PTSD sufferers; however, elevated PLM indices in both P-NM and I-NM sufferers suggest that PLMs may not be a marker of hyperarousal in sleep of PTSD sufferers. Rather, PLMs may be a correlate of processes contributing to intense negative dreaming.

**Key Words:** Idiopathic and posttraumatic nightmares, sleep disturbances, periodic leg movements in sleep

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Introduction

Rapid eye movement (REM) sleep disturbances and nightmares have been suggested to be the hallmark of posttraumatic stress disorder (PTSD) (Ross et al 1989). The presence of REM sleep disturbances remains equivocal, however, and no profile of sleep disturbances unique to PTSD has yet been established (e.g., see Pillar et al 2000 for review). One study showed that the nature of posttraumatic nightmares may shape sleep profiles in PTSD inpatients (Woodward et al 2000). Nightmares are part of the intrusion symptom cluster of PTSD (American Psychiatric Association 1994) and are reported by as many as 75% of PTSD patients (e.g., Kilpatrick et al 1994). Although spontaneous awakenings following distressing dreams are rarely observed when PTSD patients sleep in a laboratory environment, chronic and frequent distressing dreams (associated or not with a sudden awakening) may nevertheless influence sleep profiles in PTSD patients in a pervasive manner. In other words, we postulate that sleep disturbances in PTSD may be in part due to nightmare pathophysiology, rather than being exclusively attributable to PTSD-specific processes.

Two studies conducted with individuals reporting frequent nightmares but who do not suffer from PTSD observed sleep disturbances comparable to those observed in PTSD patients, including increased or unaltered phasic REM sleep activity, decreased total sleep time, increased number and duration of nocturnal awakenings, and decreased slow-wave sleep (Fisher et al 1970; Newell et al 1992). Another study (van der Kolk et al 1984) compared sleep complaints in nightmare sufferers with PTSD (n = 15) and individuals with idiopathic nightmares (n = 10). Posttraumatic stress disorder–related nightmares tended to occur earlier in the night, were more frequent, and were more often associated with gross body movements than were idiopathic nightmares; however, a lack of objective sleep measures obviates the possibility of determining more specifically whether these two groups had different types of sleep disturbances. Together, these observations suggest that the relationship between chronic frequent nightmares and sleep anomalies may not be exclusive to PTSD. Rather, these findings further support the notion.
that sleep disturbances in PTSD patients may be in part a function of the nightmare psychopathology rather than other, more global PTSD processes.

In fact, no study has investigated whether PTSD patients with frequent nightmares differ from idiopathic nightmare sufferers on laboratory-recorded sleep parameters. Idiopathic nightmare sufferers offer a unique opportunity to determine whether the sleep disturbances observed in PTSD patients are due to intrinsic pathophysiological factors proper to PTSD, or whether some of these anomalies are attributable to nightmare pathophysiology. Thus, the goal of the present study was to investigate whether the sleep attributes of PTSD patients with frequent nightmares differ from those of idiopathic nightmare sufferers and healthy participants matched for age and gender.

Methods and Materials

Participants

Self-referred nightmare sufferers were recruited mainly from advertisements in the University of Montreal’s campus newspaper and following a short televised documentary on the study and treatment of nightmares that aired in the evening. To enter the study, participants had to be at least 18 years of age and to report recalling more than one nightmare per week for a minimum of 6 months. They were excluded if 1) they were currently under medications known to influence sleep and dreams; 2) they were currently suffering from a major psychiatric disorder other than PTSD; 3) they reported currently suffering from another sleep problem; 4) they suffered from a neurologic disorder; 5) they reported irregular sleep–wake schedules or had undergone jet lag; 6) they reported using alcohol or drugs on a regular basis; or 7) they were currently engaged in legal proceedings involving events related to their nightmares. Eligibility was ascertained during extensive clinical interview conducted by either one of the two authors. Nine individuals with PTSD and nightmares (P-NM; four men and five women, age [mean ± SD] = 39.0 ± 12.1 years) and 11 individuals with idiopathic nightmares (I-NM; five men and six women; age = 28.2 ± 5.3 years) met these criteria and participated in the study.

For participants who reported that the onset of nightmares occurred after exposure to a traumatic event (i.e., P-NM sufferers), PTSD status was determined using the Clinician’s Assessment of Posttraumatic Stress (Blake et al 1990). Three P-NM sufferers (patients 1, 2, and 9) met DSM-IV criteria for current depressive episodes in the severe range; however, all three sufferers attributed their current depressive symptoms to the PTSD-related events (i.e., financial problems, social isolation, and imminent prison release of the aggressor); they were thus included. One I-NM patient (patient 1) reported a history of substance abuse but had been abstinent for 6 years, whereas none of the PTSD patient did. Seven men and six women (age = 32.6 ± 11.2 years) constituted the control group. These subjects were paired for age and gender to the I-NM and P-NM sufferers. They were recruited from an advertisement in the same university newspaper. Because of limited resources available for the present study, eligibility of control subjects was assessed during a telephone interview and through the completion of self-report measures (see below). All reported being good sleepers, free of sleep and dreaming disturbances, and otherwise met the same inclusion and exclusion criteria.

The Sacré-Cœur Hospital Ethics Committee approved the study. Written and verbal consent was obtained from all participants.

Self-Report Measures

At intake, all participants completed the Beck Depression Inventory (BDI; Beck et al 1961), the Beck Anxiety Inventory (BAI; Beck et al 1988), the Nightmare Distress Questionnaire (NDQ; Belicki 1992), and the Posttraumatic Symptom Scale, self-report version (PSS-SR; Foa et al 1993). The BDI is a 21-item self-report questionnaire that assesses severity of the behavioral, cognitive, emotional, and somatic symptoms associated with depression. The BAI is a similar 21-item self-report checklist that assesses the severity of anxiety-related symptoms. Both the BDI and the BAI are scored by summing responses for each of the 21 items, with each item rated on a 0–3 scale. The NDQ is a 13-item self-report scale that measures the level of waking distress associated with the experience of nightmares. This instrument has been shown to be reliable; high scores are significantly correlated with interests in pursuing therapy for nightmares (Belicki 1992). Finally, the PSS-SR is a measure of PTSD severity according to DSM-III-R criteria (American Psychiatric Association 1987), and it evaluates the severity of intrusion, avoidance, and arousal symptoms in the preceding 2-week period. On all measures, higher scores reflect greater symptom severity.

Polysomnography

All participants slept in the laboratory for two consecutive nights. Sleep recordings were performed with a 32-channel montage that measured electroencephalogram (EEG) with the international 10–20 electrode placement system (FP1, FP2, F3, F4, F7, F8, C3, C4, T3, T4, T5, T6, P3, P4, O1, O2, Fz, Cz, Pz), eye movements (LOC-A2, ROC-A1), EMG (submental and right tibialis), oral-nasal airflow, and electrocardiogram. A referential montage (with linked-ears reference) was used to record the 19 EEG channels. Recordings were performed using RHYTHM version 10.0 (Stellate Systems, Montréal, Québec, Canada) and were scored manually, according to Rechtschaffen and Kales (1968) criteria using HARMONY version 4.1 (Stellate Systems), by an experienced polysomnographic technician who had not conducted the sleep recordings and who was blind to the purpose of the study. Sleep onset latency (SOL) was computed as the interval between lights out and the first episode of any sleep stage. Periodic leg movements (PLMs) were scored according to Coleman’s criteria (Coleman 1982), and the PLM indices were computed as the number of PLM × 60/number of minutes of sleep. Rapid eye movement density was computed as the absolute number of REMs (Tashihana et al 1994) during the last
5 minutes of each REM sleep episode and then averaged over all REM sleep episodes. Micro-arousals were identified as abrupt changes in EEG frequency with a minimal duration of 3 sec and a maximal duration of 10 sec and could include alpha or theta frequencies but not spindles. A minimal interval of continuous sleep of 10 sec was necessary to score a second micro-arousal (American Sleep Disorders Association 1992). The first night was considered an adaptation night, and only results collected from the second night are reported. Bedtime was between 10:00 PM and midnight, depending on each participant’s usual bedtime. The morning awakening was conducted between 6:00 and 8:00 AM, again depending on each participant’s typical schedule. In the morning, electrodes were removed and participants were free to go for the day. Before leaving and after the first recording night, they were reminded to avoid caffeine consumption and naps during that day. All participants received a monetary compensation of $20 per night slept in the laboratory.

Statistical Analyses

Statistica 5.1 software (StatsSoft, Tulsa, OK) was used. Square root transformations were applied to SOL, wake time after sleep onset, number of awakenings, REM latency, REM density, and all variables related to PLM. Logarithmic transformations were applied data related to sleep and REM sleep latencies, sleep stages, sleep efficiency, and REM sleep efficiency. One-way analyses of variance (ANOVARs) were then computed and Newman-Keuls post hoc comparisons performed. One-way ANOVAs and Newman-Keuls post hoc comparisons were also conducted to assess group differences on the self-report measures of psychological distress. The significance level was set at .05.

Results

Sample Characteristics and Self-Report Measures

Table 1 presents information on the age, gender, nightmare chronicity, type of trauma, and PTSD severity (when applicable) of all nightmare sufferers. Four of the I-NM sufferers also reported past traumatic events, but the onset of nightmares preceded the trauma in all cases. None of these met the criteria for past or current PTSD. Age tended to differ across the three groups \(F(2,32) = 3.11, p = .06\); P-NM sufferers were relatively older than I-NM sufferers \(p = .05\). (Because the present study does not have sufficient statistical power to further discriminate regarding the contribution of age from that of nightmare etiology, age was not entered as a covariate in subsequent analyses.) Nightmare chronicity did not differ across the three groups \(F(1,19) = .09, \text{ ns}\). Control subjects did not report significant levels of psychological distress. One control subject reported a traumatic event (motor vehicle accident) 2 years before

Table 1. Descriptive Information on P-NM and I-NM Patients

<table>
<thead>
<tr>
<th></th>
<th>Gender</th>
<th>Age</th>
<th>NM Chronicity</th>
<th>Trauma</th>
<th>PTSD Severity</th>
<th>PTSD Chronicity</th>
</tr>
</thead>
<tbody>
<tr>
<td>P-NM</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>M</td>
<td>28</td>
<td>3</td>
<td>Car crash</td>
<td>Severe</td>
<td>3</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>46</td>
<td>4</td>
<td>Rape</td>
<td>Severe</td>
<td>4</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>58</td>
<td>53</td>
<td>Sexual abuse(^{b})</td>
<td>Severe</td>
<td>14</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>35</td>
<td>25</td>
<td>Physical abuse(^{b})</td>
<td>Moderate</td>
<td>25</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>40</td>
<td>10</td>
<td>Physical and sexual abuse(^{c})</td>
<td>Moderate</td>
<td>10</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>40</td>
<td>3</td>
<td>Rape</td>
<td>Moderate</td>
<td>3</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>46</td>
<td>20</td>
<td>Parachute accident</td>
<td>Moderate</td>
<td>20</td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>22</td>
<td>5</td>
<td>25-foot fall</td>
<td>Moderate</td>
<td>5</td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>36</td>
<td>2.5</td>
<td>Physical assault</td>
<td>Severe</td>
<td>2</td>
</tr>
<tr>
<td>I-NM</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>F</td>
<td>36</td>
<td>30</td>
<td>Kidnapped(^{b})</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>M</td>
<td>19</td>
<td>13</td>
<td></td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>30</td>
<td>10</td>
<td></td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>F</td>
<td>27</td>
<td>9</td>
<td>Father was shot</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>36</td>
<td>25</td>
<td></td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>M</td>
<td>24</td>
<td>19</td>
<td>Witnessed armed robbery</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>25</td>
<td>13</td>
<td></td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>30</td>
<td>25</td>
<td>Intruder in the house</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>M</td>
<td>29</td>
<td>24</td>
<td></td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>F</td>
<td>31</td>
<td>2</td>
<td></td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>F</td>
<td>23</td>
<td>2.5</td>
<td></td>
<td>None</td>
<td></td>
</tr>
</tbody>
</table>

\(^{a}\)Nightmare chronicity provided in years, and did not differ between groups.  
\(^{b}\)Trauma occurred in childhood.  
\(^{c}\)Trauma occurred during adolescence.

P-NM, posttraumatic stress (PTSD) nightmare; I-NM, idiopathic nightmare; M, male; F, female
participating in the study but did not meet present past or current PTSD symptoms. Table 2 presents mean scores on the self-report measures, including prospective nightmare frequency for the three study groups. Significant group differences were observed on all measures of psychological distress. The P-NM group endorsed significantly higher scores on the BDI, BAI, NDQ, and PSS-SR than did the control group (all \( p < .05 \)). The P-NM group also endorsed higher scores on the BDI, PSS-SR, and NDQ than the I-NM group (all \( p < .05 \)). The I-NM group endorsed higher scores than did control group on the NDQ (\( p < .001 \)), whereas the two groups were comparable on the BDI, BAI, and PSS-SR.

### Polysomnography

Mean polysomnography scores for the three groups are presented in Table 3. Sleep efficiency significantly differed across the three study groups [\( F(2,29) = 3.50, p = .04 \)]. This was attributable to differences in the total number of nocturnal awakenings [\( F(2,29) = 5.61, p = .002 \)], and total wake time after sleep onset [\( F(2,29) = 4.13, p = .03 \)] across the three groups. The P-NM group exhibited more nocturnal awakenings than both I-NM and control groups (\( p = .007 \) and \( p = .01 \), respectively). The latter two groups did not differ on these three measures. The groups did not differ on any of the REM sleep parameters.

Mean percent sleep stage scores are presented in Table 4. None of these sleep stage variables, including REM sleep percent, differentiated the groups.

### Periodic Leg Movements during Sleep

Table 5 presents results for PLMs in REM and non-REM sleep. Differences across the three groups were found for all PLM indices, with and without associated micro arousals, in both REM and non-REM sleep. In all cases, P-NM and I-NM sufferers exhibited elevated PLM indices compared with control subjects, but did not differ from one another.

### Discussion

Consistent with prior studies (Engdahl et al. 2000; Kramer and Kinney 1988; Lavie et al. 1979; Mellman et al. 1995;

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### Table 2. Scores on Measures of Anxiety, Depression, Nightmare Distress, and Posttraumatic Symptom Severity (when applicable), for P-NM, I-NM, and Control Subjects

<table>
<thead>
<tr>
<th></th>
<th>P-NM (n = 9)</th>
<th>I-NM (n = 11)</th>
<th>CTL (n = 13)</th>
<th>( F ) (df)</th>
<th>( p )</th>
<th>Post-hoc Comparisons</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anxiety</td>
<td>18.22 (9.58)</td>
<td>9.27 (10.55)</td>
<td>6.58 (5.07)</td>
<td>5.07 (2.31)</td>
<td>.01</td>
<td>P-NM &gt; CTL, ( p &gt; .01 )</td>
</tr>
<tr>
<td>Depression</td>
<td>21.56 (11.10)</td>
<td>7.36 (6.18)</td>
<td>3.33 (3.47)</td>
<td>17.49 (2.31)</td>
<td>&lt; .001</td>
<td>P-NM &gt; I-NM, ( p &lt; .001 )</td>
</tr>
<tr>
<td>Nightmare Distress</td>
<td>39.89 (7.37)</td>
<td>34.81 (6.75)</td>
<td>13.25 (11.44)</td>
<td>24.18 (2.31)</td>
<td>&lt; .001</td>
<td>P-NM &gt; CTL, ( p = .001 )</td>
</tr>
<tr>
<td>Posttraumatic Symptom</td>
<td>34.31 (6.93)</td>
<td>5.40 (4.06)</td>
<td>2.50 (2.84)</td>
<td>129.42 (2.29)</td>
<td>&lt; .001</td>
<td>P-NM &gt; I-NM, ( p &lt; .001 )</td>
</tr>
</tbody>
</table>

Values are mean (SD). P-NM, posttraumatic stress (PTSD) nightmare; I-NM, idiopathic nightmare; CTL, control subjects.
van der Kolk et al (1984), P-NM sufferers exhibited lower sleep efficiency than did I-NM sufferers and healthy control participants. Poorer sleep efficiency in P-NM sufferers was due to increases in the number and duration of nocturnal awakenings in P-NM sufferers compared with I-NM and control subjects. Posttraumatic nightmare sufferers did not differ from I-NM sufferers or control subjects on any of the REM sleep and sleep stage measures. Both P-NM and I-NM sufferers exhibited elevated PLM indices compared with control subjects. Despite the relatively small sample sizes attributable to stringent selection criteria, and the consequent lack of statistical power to control for the potential confound of age on these sleep parameters, the present study nevertheless provides preliminary support for the hypothesis that sleep anomalies in PTSD may in part be a function of PTSD-specific processes, and partially attributable to pervasive effects of chronic frequent nightmares.

The findings that the P-NM group exhibited more nocturnal awakenings than the other two groups further support the hypothesis that a lowered arousal threshold characterizes sleep in PTSD (Brown and Boudewyns 1996; Mellman et al 1995; Ross et al 1989). The present results may not contradict findings from three previous studies that have shown that PTSD patients exhibit higher awakening thresholds during sleep than do control subjects (Dagan et al 1991; Lavie et al 1998; Schoen et al 1984). It remains possible that emotional–attentional processes are shifted inward toward intensified negative sleep mentation in PTSD sufferers, rather than outward toward external stimuli. Intensified oneiric processes may thus be related to an abnormal emotional–attentional shift during sleep, increased number of nocturnal awakenings, and an increase in body movements during sleep.

The finding that all PLM indices were elevated in both groups of nightmare sufferers relative to control subjects does not support the hypothesis that PLMs are a specific correlate of hyperarousal in P-NM patients (Brown and Boudewyns 1996; Ross et al 1989, 1994). Examination of the PLM inter-movement intervals indicated that gross body movements, as well as PLMs, may be more frequent in both groups of nightmare sufferers. Despite the low PLM indices and the questionable clinical significance of PLMs (e.g., Mahowald 2001; Monplaisir et al 2000), the present observations nevertheless bear empirical significance in indicating that PLMs/gross body movement may be closely related to nightmare pathophysiology, or more globally, to abnormal oneiric processes. The direction of this relationship, however, remains unclear. Abnormal central motor activation patterns may be translated at higher cortical levels into vivid terrifying dream imagery (Ross et al 1994) in both P-NM and I-NM sufferers. Alternatively, intense negative dreaming may facilitate the release of motor inhibition during all sleep stages. This is not to say that leg or body movements are necessarily

### Table 4. Average Percent Sleep Stages for the Three Study Groups: P-NM Sufferers, I-NM Sufferers, and Control Participants

<table>
<thead>
<tr>
<th>Group</th>
<th>P-NM (n = 9)</th>
<th>I-NM (n = 11)</th>
<th>CTL (n = 13)</th>
<th>F</th>
</tr>
</thead>
<tbody>
<tr>
<td>% S1</td>
<td>12.2 (5.6)</td>
<td>8.2 (3.1)</td>
<td>8.4 (4.2)</td>
<td>2.36b</td>
</tr>
<tr>
<td>% S2</td>
<td>64.5 (7.3)</td>
<td>60.6 (8.6)</td>
<td>64.2 (4.4)</td>
<td>1.23b</td>
</tr>
<tr>
<td>% S3</td>
<td>3.6 (3.7)</td>
<td>8.4 (4.9)</td>
<td>5.1 (3.9)</td>
<td>1.78c</td>
</tr>
<tr>
<td>% S4</td>
<td>.2 (.4)</td>
<td>1.7 (2.7)</td>
<td>.8 (1.5)</td>
<td>.03d</td>
</tr>
<tr>
<td>% REM</td>
<td>19.5 (5.1)</td>
<td>21.2 (4.4)</td>
<td>21.5 (3.6)</td>
<td>.81</td>
</tr>
</tbody>
</table>

Values are mean (SD). P-NM, posttraumatic stress (PTSD) nightmare; I-NM, idiopathic nightmare; CTL, control subjects; PLMs, periodic leg movements; MA, micro-arousals.

### Table 5. Scores for Periodic Leg Movements in REM and non-REM Sleep,* with and without Micro-arousals for P-NM Sufferers, I-NM Sufferers, and Control Participants

<table>
<thead>
<tr>
<th>Group</th>
<th>P-NM (n = 9)</th>
<th>I-NM (n = 11)</th>
<th>CTL (n = 13)</th>
<th>Post-hoc Comparisons</th>
</tr>
</thead>
<tbody>
<tr>
<td>PLMs</td>
<td>7.0 (5.6)</td>
<td>8.8 (8.9)</td>
<td>1.9 (2.7)</td>
<td>8.93 (p &lt; .001)</td>
</tr>
<tr>
<td>PLMs + MA</td>
<td>2.1 (1.7)</td>
<td>1.9 (1.2)</td>
<td>.6 (.9)</td>
<td>7.70 (p = .001)</td>
</tr>
<tr>
<td>REM PLMs</td>
<td>14.2 (8.6)</td>
<td>18.61 (17.9)</td>
<td>5.2 (9.1)</td>
<td>7.96 (p = .002)</td>
</tr>
<tr>
<td>REM PLMs + MA</td>
<td>2.7 (2.7)</td>
<td>1.8 (1.8)</td>
<td>.5 (1.0)</td>
<td>1.77 (ns)</td>
</tr>
<tr>
<td>Non-REM PLMs</td>
<td>5.2 (4.9)</td>
<td>5.7 (7.9)</td>
<td>1.0 (1.4)</td>
<td>5.94 (p = .007)</td>
</tr>
<tr>
<td>Non-REM PLMs + MA</td>
<td>1.9 (1.5)</td>
<td>1.7 (1.4)</td>
<td>.6 (1.0)</td>
<td>6.03 (p = .006)</td>
</tr>
</tbody>
</table>

Values are mean (SD). REM, rapid eye movement; P-NM, posttraumatic stress (PTSD) nightmare; I-NM, idiopathic nightmare; CTL, control subjects; PLMs, periodic leg movements; MA, micro-arousals.

*Square root transformation performed before analyses. Mean values are presented in original units.

All df = 2,30
correlates of nightmare content, in a manner analogous to the motor correlates of REM-sleep behavior disorder (Mahowald and Schenck 2000), but rather to claim that body movements may reflect an underlying disposition of system dysfunction that produces intense negative dream-
ing both in and out of PTSD. Neuroimaging developments in the study of sleep (e.g., Nofzinger et al 1998) may clarify the neuroanatomic substrates underlying the relationships between waking anxiety, disturbing dreams, and increased motor activity during sleep in P-NM and I-NM sufferers.

The main limitations of the present study are inherent to the sample sizes in both groups of nightmare sufferers due to the use of stringent selection criteria. Replications with larger samples are required to further characterize the specific contribution of nightmare etiology in shaping sleep disturbances both in and out of PTSD. Larger samples will provide the statistical power necessary to control for the potential confounding effects of age on sleep, as well as to investigate which qualities of nightmares (e.g., frequency vs. distress) more directly influence sleep parameters in PTSD and non-PTSD patients. Another limitation concerns the absence of a group of PTSD subjects without frequent nightmares; however, because as many as 75% of PTSD patients endorse frequent nightmares (e.g., Kilpatrick et al 1994), the feasibility of recruiting nonmedicated PTSD patients without nightmares remains uncertain. Finally, eligibility of control participants did not include the use of structured diagnostic interview. Although the levels of psychopathology endorsed by the control participants were below clinically significant thresholds, the possibility that subsyndromal psychopathologies may have been present cannot not be ruled out.

Despite these limitations, this is the first study to investigate the pathophysiology of sleep in nonveteran P-NM sufferers in comparison with I-NM sufferers and healthy individuals. The results support and refine the sleep-related hyperarousal hypothesis during sleep in PTSD but indicate that increased motor activity during sleep is a correlate of chronic frequent nightmares both inside and outside of PTSD. In addition, the inclusion of I-NM sufferers allowed us to provide new data regarding the neurophysiologic correlates of abnormal dreaming processes during sleep in non-PTSD nightmare sufferers.

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