Replicability of Psychometric Differences Between Obstructive Sleep Apnea, Primary Snoring, And Periodic Limb Movement Disorder

James E. Aikens, Ph.D., Wallace B. Mendelson, M.D., and Erin K. Baehr, M.A.

We previously documented that periodic limb movement disorder (PLMD) patients report more psychopathological symptoms on the MMPI than obstructive sleep apnea (OSA) patients, who in turn report more symptoms than primary snorers (PS). The purpose of this study was to replicate and extend these findings on an independent patient sample using an alternative measure, the Symptom Checklist 90 - Revised (SCL-90-R). Of 109 consecutive diagnostic polysomnography (PSG) referrals to a Sleep Disorders Clinic, we studied every patient with PLMD (n=11, 6 males and 5 females; mean age = 55 years). Two comparison groups were developed by randomly selecting one OSA and one PS patient from those matching PLMD subjects on gender and age. Subjects completed the SCL-90-R before undergoing sleep interview, physical examination, 12-channel diagnostic PSG, and daytime MSLT. Analysis of covariance indicated that, even after controlling for MSLT latency, the SCL-90-R General Severity Index was significantly higher (and more likely to be elevated) in the PLMD subjects than in OSA or PS subjects. PLMDs also scored significantly higher than both of the other groups on the Obsessive-Compulsive, Interpersonal Sensitivity, and Depression scales. Corresponding differences emerged in the prevalence of elevated SCL-90-R scores; elevation was 1.7 to 3.6 times more likely in PLMD than in OSA and PS. We conclude that irrespective of daytime sleepiness, patients with PLMD are more likely than those with PS or OSA to experience obsessionality, compulsivity, concentration difficulties, dysphoria, fatigue, low motivation, and interpersonal oversensitivity. Thus, the relative ranking of psychopathology symptoms previously observed in patients with PLMD, OSA, and primary snoring was replicated across different samples and measures. (Sleep and Hypnosis 1999;4:212-216)

Key words: sleep apnea, snoring, periodic leg movement disorder, psychological distress.

INTRODUCTION

Although several studies document psychological disturbance in various sleep disorders, most existing work concerns insomnia, probably because psychological deviations are presumed to have no particular etiologic role in disorders such as periodic limb movement disorder.

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(PLMD) or obstructive sleep apnea (OSA) syndrome. For example, although psychopathology has been widely documented in OSA (1-9), it is correlated with poor nocturnal oxygen saturation (1,2,5), appears to improve following OSA treatment (10-12), and at least in the case of major depressive disorder, tends not to manifest itself until after OSA onset (13). Even though probably attributable to OSA, such problems may nonetheless be clinically significant (1,2,4,5,9) and thus become an ancillary target of treatment. Finally, we have recently noted that psychometric responses of patients with primary snoring (but not OSA) fall midway between those of OSA patients and normals (4) and are also of a magnitude suggesting clinical significance. Considerably less is known about the type of psychopathology accompanying PLMD, a nocturnal movement disorder which occurs at the rate of 8.6 per 100,000 (14) and is most common among the older adults.
Mendelson (16) noted that 30% of PLMD patients report a history of depression treatment, and Mosko and colleagues (17) documented that PLMD patients report more depressive symptoms than patients with OSA do. A few studies directly compare PLMD to other sleep-disordered groups or healthy normals using the Minnesota Multiphasic Personality Inventory (MMPI; 18), which assesses a variety of psychiatric symptoms and personality traits. Zorick and colleagues (19) found that PLMD patients (including those with restless legs syndrome; RLS) were older and had the fewest MMPI elevations of all sleep disordered groups studied (mean 1.0 + s.e.m. = .36), which included insomnia associated with psychiatric disorder, psychophysiological insomnia, and sleep state misperception (i.e., those patients with insomnia complaint but no objective findings on PSG). In contrast, we have found that patients with PLMD (excluding all those with RLS) average 3.2 MMPI elevations (+SD = 2.6, s.e.m. = .51), a fairly high rate (3). In fact, the high rate of MMPI elevations (88%) found for PLMD was indistinguishable from that recorded in combined insomnia and psychiatric disorder, and significantly greater than rates seen in either OSA or psychophysiological insomnia. PLMD patients specifically showed greater elevation on scales 2 (Depression), 7 (Psychasthenia), and 8 (Schizophrenia), suggesting that they may be more likely to experience a wide range of depressive symptoms accompanied by anxious guilt, tension, and worry, as well as possibly social alienation and diminished mental concentration. The puzzling discrepancy with Zorick et al. (19) could possibly be due to differences in sampling and data analysis, but clearly suggests a need for further research.

Thus, there are conflicting reports about the degree of psychological disturbance experienced by patients with PLMD. We sought to clarify this issue by repeating the study with an alternative assessment instrument (Symptom Checklist 90 - Revised, or SCL-90-R; 20) and an independent patient sample. Although the MMPI (and its recent revision, the MMPI-2) is multidimensional and well-established, its disadvantages include its length, item overlap between scales, and emphasis on traits rather than discrete behaviors.

METHODS

Subjects

The 33 subjects were drawn from a pool of 109 consecutive clinical patients who underwent overnight diagnostic polysomnography (PSG) after being referred for diagnostic evaluation to a university sleep disorders clinic. As would be expected for a clinical series, the presenting complaints and reasons for referral were quite diverse, so for the purposes of this descriptive study we classified subjects on the basis of sleep disorder diagnosis. We selected every patient diagnosed with PLMD (n = 11; 6 males and 5 females, none of whom had RLS or major psychiatric disorder). These patients averaged 55.4 years of age (+11.1), and had a mean periodic leg movement index of 14.9 (+19.9) and mean sleep efficiency of 70.9 (+15.4). All subjects with primary psychiatric disorder accounting for their sleep disturbance were excluded. Two comparison groups were developed by randomly selecting one OSA and one PS patient from those matching PLMD subjects on age (+/-2 years) and gender. The demographic and polysomnographic characteristics of all three groups are summarized in Table 1.

Table 1. Means and standard deviations (-) for demographic and polysomnographic variables.

<table>
<thead>
<tr>
<th></th>
<th>PLMD (n=11)</th>
<th>OSA (n=11)</th>
<th>PS (n=11)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>55.4 – 11.1</td>
<td>55.0 – 12.1</td>
<td>53.6 – 12.2</td>
</tr>
<tr>
<td>Periodic leg movement index (PLMI)</td>
<td>14.9 – 19.9</td>
<td>3.1 – 7.5</td>
<td>1.0 – 1.6</td>
</tr>
<tr>
<td>Apnea-hypopnea index (AHI)</td>
<td>1.8 – 1.8</td>
<td>19.9 – 14.6</td>
<td>1.8 – 1.6</td>
</tr>
<tr>
<td>Sleep latency (min)</td>
<td>34.7 – 31.4</td>
<td>28.8 – 21.2</td>
<td>37.5 – 39.9</td>
</tr>
<tr>
<td>Total sleep time (min)</td>
<td>266.2 – 69.8</td>
<td>300.8 – 68.0</td>
<td>356.6 – 51.0</td>
</tr>
<tr>
<td>Sleep efficiency</td>
<td>70.9 – 15.4</td>
<td>74.4 – 15.9</td>
<td>83.4 – 11.9</td>
</tr>
<tr>
<td>MSLT mean latency (min)</td>
<td>7.4 – 6.6</td>
<td>7.5 – 7.6</td>
<td>8.2 – 5.0</td>
</tr>
</tbody>
</table>

a. PLMD mean > OSA and PS means (ANOVA; p < .05).
b. OSA mean > PLMD and PS means (ANOVA; p < .05).
c. PS mean > PLMD and OSA means (ANOVA; p < .05).

RESULTS

For descriptive data on the major study variables (except SCL-90-R data), see Table 1. As could be expected on the basis of sleep diagnosis, preliminary analysis of variance comparisons revealed that the PLMD group had the highest PLM index (PLM1; F(2,30) = 4.03, p (2-tailed) < .05), the OSA group had the highest apnea hypopnea...
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of clinical significance for individual group members, we no alteration of the results.

daytime sleepiness. Thus, we repeated all ANOVAs with effects may be influenced by the psychological impact of depressive disorder (24), we sought to rule out that the SCL-90-R mean scores by group. Overall effects emerged for more than 55% of subjects and a mean elevation rate followed up with pairwise comparisons, which indicated (F(2,30)= 7.99, p<.05), and in no case did the OSA group differ from the PS group scored higher than the OSA and PS groups (all ps <.05), and in no case did the OSA group differ from the PS group scored higher than the OSA and PS groups (all ps <.05).

group. Because several researchers have commented on the association between excessive daytime somnolence and depressive disorder (24), we sought to rule out that the effects may be influenced by the psychological impact of daytime sleepiness. Thus, we repeated all ANOVAs with MSLT latency data entered first as a covariate, but observed no alteration of the results.

Because group means do not directly address the issue of clinical significance for individual group members, we also analyzed the prevalence of elevated SCL-90-R scores. Chi-squared frequency analysis was used to analyze covariance between group membership and elevation. The results were consistent with the ANOVA results. Compared to the OSA or PS groups, elevation in the PLMD group was 3.6 times more likely on GSI (95% c.i.: 1.6 - 8.2; X2(1) = 10.5, p<001), 1.7 times more likely on Obsessive-compulsive (95% c.i. 1.1 - 2.5; X2(1) = 4.4, p<05), 3.5 times more likely on Interpersonal sensitivity (95% c.i. 1.3 - 9.4; X2(1) = 6.8, p<005), and 2.2 times more likely on Obsessive-compulsive (95% c.i. 1.3 - 3.8; X2(1) = 7.5, p<005). Finally, subjects with PLMD were almost twice as likely to meet caseness criteria (OR = 1.7; 95% c.i. 1.1 - 2.5; X2(1) = 4.4, p<05).

**DISCUSSION**

We found evidence of several significant differences by sleep disorder in behavioral and psychiatric complaints. Patients with PLMD showed higher mean scores (and rates of clinical elevation) on the GSI, the most reliable and valid SCL-90-R indicator of general psychiatric distress. This was attributable to differences on the Obsessive-compulsive, Interpersonal sensitivity, and Depression subscales. This generally concurs with our prior MMPI-based findings, in that PLMD patients manifested relatively high levels of obsessional thinking and compulsive behaviors, concentration difficulties, marked feelings of personal inadequacy and discomfort during social interactions, as well as multiple depressive symptoms (e.g., dysphoric mood, low energy, uncontrollable worry, reduced general motivation, low self esteem, hopelessness, suicidal ideation, etc.). Finally, none of these differences were readily attributable to daytime sleepiness, insofar as there were no associations between psychometric scores and MSLT latency, and the statistical analysis outcome was unaffected by the removal of MSLT-related covariance.

The prevalence rates listed in Table 2 can be appreciated by noting that the norm-referenced T score cutoff of 63 approximately corresponds to the 90th percentile relative of the SCL-90-R community normative sample. Thus, only 10% of "nondistressed" individuals would be expected to score above the cutoff T of 63. Our prior study indicated that 88% of PLMD patients had at

**Table 2. Means, standard deviations (-), and percentage of subjects with an elevated score (T score > 63) for SCL-90-R variables.**

<table>
<thead>
<tr>
<th>SCL-90-R subscale</th>
<th>PLMD Mean – SD</th>
<th>PLMD % Ss elev.</th>
<th>OSA Mean – SD</th>
<th>OSA % Ss elev</th>
<th>PS Mean – SD</th>
<th>PS % Ss elev</th>
</tr>
</thead>
<tbody>
<tr>
<td>General Severity Index</td>
<td>65.0 – 7.3a</td>
<td>82%b</td>
<td>49.8 – 15.2</td>
<td>27%</td>
<td>53.0 – 12.1</td>
<td>18%</td>
</tr>
<tr>
<td>Somatization</td>
<td>66.9 – 10.9</td>
<td>73%</td>
<td>62.9 – 11.7</td>
<td>46%</td>
<td>61.6 – 10.8</td>
<td>46%</td>
</tr>
<tr>
<td>Obsessive-compulsive</td>
<td>71.1 – 10.7a</td>
<td>91%b</td>
<td>65.7 – 9.9</td>
<td>55%</td>
<td>65.6 – 7.9</td>
<td>55%</td>
</tr>
<tr>
<td>Interpersonal sensitivity</td>
<td>66.1 – 5.1a</td>
<td>64%</td>
<td>59.9 – 5.2</td>
<td>18%</td>
<td>59.5 – 4.2</td>
<td>18%</td>
</tr>
<tr>
<td>Depression</td>
<td>71.3 – 6.5a</td>
<td>91%b</td>
<td>61.6 – 6.2</td>
<td>36%</td>
<td>62.4 – 6.2</td>
<td>46%</td>
</tr>
<tr>
<td>Anxiety</td>
<td>70.3 – 8.8</td>
<td>64%</td>
<td>63.3 – 6.5</td>
<td>27%</td>
<td>66.8 – 7.0</td>
<td>55%</td>
</tr>
<tr>
<td>Hostility</td>
<td>65.9 – 7.6</td>
<td>55%</td>
<td>63.1 – 5.6</td>
<td>36%</td>
<td>62.8 – 5.8</td>
<td>36%</td>
</tr>
<tr>
<td>Phobic anxiety</td>
<td>68.7 – 6.3</td>
<td>73%</td>
<td>65.3 – 3.1</td>
<td>55%</td>
<td>66.5 – 5.7</td>
<td>55%</td>
</tr>
<tr>
<td>Paranoid ideation</td>
<td>64.9 – 7.8</td>
<td>36%</td>
<td>61.6 – 6.7</td>
<td>27%</td>
<td>61.6 – 8.4</td>
<td>9%</td>
</tr>
<tr>
<td>Psychoticism</td>
<td>64.3 – 11.9</td>
<td>54%</td>
<td>61.2 – 11.6</td>
<td>36%</td>
<td>55.1 – 8.0</td>
<td>27%</td>
</tr>
<tr>
<td>&quot;Caseness&quot;a,b,c</td>
<td>64%</td>
<td>91%</td>
<td>61%</td>
<td>62%</td>
<td>55%</td>
<td>64%</td>
</tr>
</tbody>
</table>

a. PLMD mean > OSA and PS means (both ANOVA p <.05, and ANCOVA controlling for MSLT latency p <.05).
b. PLMD proportion > OSA and PS proportions (X2; p<.05).
c. "Caseness" index, operationally defined as elevated General Severity Index (T > 63) or 2 clinical scales elevated (T > 63).
least one MMPI elevation, which is 1.3 to 1.5 times more common than we have recorded in patients with OSA (1-3,5), and 1.3 times more common than in patients with PS (4). This replication across assessment instruments suggests that PLMD - OSA psychometric differences are robust, potentially generalizing across measures as well as samples.

Moving beyond the issue of quantitative consensus with prior data, the particular pattern of SCL-90-R differences suggests qualitative interpretations that also agree with our prior MMPI-based conclusions. This is particularly true with respect to depressive symptoms (as reflected on MMPI scale 2, or Depression) and anxiety symptoms (MMPI scale 7, or Psychasthenia). The lack of differences on the SCL-90-R Anxiety and Phobic Anxiety subscales suggests that the previously observed effect on MMPI scale 7 could reflect obsessive cognition and compulsive behavior rather than phobic or generalized anxiety. However, the SCL-90-R data offered a less convincing confirmation for our MMPI-based observation that clinically significant distress is twice as prevalent in patients with OSA than among those with PS (4). That is, the corresponding effect did not reach statistical significance in the current study, perhaps because the present sample was only about 25% as large as the MMPI study sample.

One possible limitation is the small sample size, although here it should be noted that power analysis indicates that our sample actually provides at least 70% power to detect group differences. Furthermore, we previously obtained the identical result (3) using a different psychological measure (the MMPI) applied to a larger independent patient sample. Other limitations are the cross-sectional design, which cannot permit us to pinpoint the development of psychiatric complaints along the timeline of the sleep disturbance. Reliance upon self report for assessing psychopathology is another limitation. Future work ought to incorporate multiple methods such as spousal report, structured observation, and standardized interview. Finally, psychiatric screening instruments such as the SCL-90-R can overestimate psychopathology in medical populations because of overlap between distress indicators and illness effects (25). However, such confounding would presumably be most likely to inflate Somatization scores, which neither varied by group nor exceeded other subscales. Regarding patient sampling, it has been suggested that persons presenting for sleep evaluation differ systematically from those with insomnia who do not seek services (24). Moreover, many poor sleepers do not complain of sleep disorder (26), and cognitive-affective estimations of ones own sleep, fatigue, and distress level may mediate the distinction between poor sleep as a phenomenon versus a complaint. Although this presents intriguing questions for future research on treatment-seeking, we cannot readily generalize our findings to all individuals with objectively-defined PLMD, OSA, and PS.

Probably the most informative future studies will evaluate the reversibility of psychological complaints with sleep disorder treatment. It will also be extremely interesting to examine the prognostic value of psychometric variables, which are reliable and inexpensive, in terms of predicting response to both medical and behavioral intervention.

REFERENCES


