# Actigraphy in the Assessment of Insomnia 

Annie Vallières, MPs; Charles M. Morin, PhD<br>Université Laval, Québec, Canada


#### Abstract

Objective: The present study explores the clinical utility and sensitivity of actigraphy as an outcome measure in the treatment of chronic insomnia. Design: Following a screening-adaptation night, polysomnography, actigraphy, and sleep-diary data were collected in the sleep laboratory for 2 baseline nights and 2 posttreatment nights. Setting: A university-affiliated sleep disorders center. Participants: Seventeen participants with chronic primary insomnia. Mean age was 41.6 years. Interventions: Participants took part in a treatment protocol investigating different sequential treatments for insomnia (these results are reported elsewhere). Measurements and Results: Compared to polysomnography, both actigraphy and sleep-diary instruments underestimated total sleep time and


sleep efficiency and overestimated total wake time. Also, actigraphy underestimated sleep-onset latency while the sleep diary overestimated it as compared to polysomnography. Actigraphy data were more accurate than sleep-diary data when compared to polysomnography. Finally, actigraphy was sensitive in detecting the effects of treatment on several sleep parameters.
Conclusions: These results suggest that actigraphy is a useful device for measuring treatment response and that it should be used as a complement to sleep-diary evaluation.
Key Words: Insomnia, actigraphy, accuracy, sleep-wake perception
Citation: Vallières A; Morin CM. Actigraphy in the assessment of insomnia. SLEEP 2003;26(7):902-6.
data convergent with PSG measures and be more accurate than the sleep diary. ${ }^{4}$ Of the studies that have investigated the accuracy of actigraphy, 5 have used, actigraphy and sleep-diary data that were not obtained from the same nights as the PSG recordings. ${ }^{5-9}$ Only 2 studies ${ }^{10,11}$ have reported results on these 3 measures obtained from the same nights. Five studies compared actigraphy to sleep diary alone, ${ }^{7,12-16}$ and 3 studies ${ }^{9,10,13}$ evaluated actigraphy's sensitivity to treatment response.

These studies indicate that, depending on the participants' insomnia diagnosis (eg, psychophysiologic or sleep-state misperception), actigraphy underestimates ${ }^{13}$ or overestimates total sleep time (TST) ${ }^{6,10,17}$ compared to PSG. The agreement coefficients between PSG and actigraphy for detecting TST vary from $.81^{8}$ to $.88^{17}$ and are lower than for good sleepers (above $90 \%{ }^{3}$ ). The magnitude of the discrepancies between those assessment devices varies from 25 minutes ${ }^{5}$ to 49 minutes. ${ }^{6}$ Regardless of the direction of the discrepancies, actigraphy has been found to be sensitive to treatment effect. ${ }^{9,10,13}$ Moreover, data on time in bed (TIB) suggest that actigraphy is a reliable measure of adherence to behavioral treatment procedures. 9,13

Despite technologic improvements and innovations in recent years, ${ }^{11,18,19}$ the accuracy and clinical usefulness of actigraphy in the assessment of insomnia is still controversial. Discrepancies may result from the inclusion of subjects with sleep-state misperception, a factor that can contribute to the underestimation of sleep time, ${ }^{6}$ or the use of heterogeneous samples comprising subjects with other sleep disorders or coexisting psychologic disorders. ${ }^{17}$ The paucity of studies using multiple PSG and parallel nights of actigraphy and sleep-diary recordings of sleep makes it difficult to understand these discrepancies. There is, therefore, a need to compare PSG, actigraphy, and sleep-diary data collected for the same nights of sleep and on more than 1 night using a homogeneous sample of insomnia patients both before and after treatment. The present study further evaluates the accuracy, sensitivity, and clinical utility of actigraphy in documenting treatment response in chronic insomnia. Polysomnography, actigraphy, and a sleep diary are used for a total of 4 nights, including 2 at baseline and 2 after treatment.

## METHOD

## Participants

Participants were recruited through newspaper advertisements or by physician referrals. Inclusion criteria were a) being between 30 and 50

## Disclosure Statement

This research was supported by a studentship from the Fond de la Recherche en Santé du Québec awarded to the first author and by a grant from the National Insitute of Mental Health (H60413) to the second author.

## Submitted for publication November 2002

## Accepted for publication June 2003

Address correspondence to: Charles M. Morin, PhD, École de psychologie, Université Laval, Pavillon FAS, Québec (QC), Canada, G1K 7P4;
Tel: 418-656-3275; Fax: 418-656-5152; E-mail: cmorin@psy.ulaval.ca
years of age; b) reporting insomnia, defined as a sleep-onset latency (SOL), wake after sleep onset (WASO), or early-morning awakening equal to or longer than 60 minutes at least 4 nights a week for the past 6 months; c) reporting significant distress or daytime impairment as evaluated by the Insomnia Severity Index (score of 2 or higher on a 0 [not at all] to 4 [very much] Likert scale); and (d) cessation, at least 1 month prior to treatment, of any sleep or other psychotropic medication that could alter sleep. Exclusion criteria were a) presence of another sleep disorder such as sleep apnea (respiratory disturbance index $>15$ ), periodic limb movements during sleep (periodic limb movement index $>$ 15), or circadian rhythm disorder; b) evidence that insomnia was related to a medical condition; c) presence of major depression, anxiety disorder, alcohol or substance abuse, or any other psychopathology as diag-
nosed with the Structured Clinical Interview for DSM-IV (SCID-IV ${ }^{20}$ ); d) currently in psychotherapy; and (e) regular use of a medication interfering with sleep (eg, antihistaminic, corticosteroid). Although more stringent, these criteria are consistent with those of the DSM-IV ${ }^{21}$ for chronic primary insomnia.

The sample included 17 participants ( 7 men and 10 women) with a mean age of 41.6 years ( $\mathrm{SD}=5.7$; range, $34-50$ ). The average education level was 15.2 years ( $\mathrm{SD}=3.0$; range, $10-19$ years). Fourteen were working, and 3 were unemployed. The average insomnia duration was 11.8 years ( $\mathrm{SD}=6.2$ ), and the mean age of insomnia onset was 29.8 years ( $\mathrm{SD}=7.7$ ). One participant presented with sleep-onset insomnia, 9 with sleep-maintenance insomnia, and 7 with mixed insomnia.

Table 1—Relative and absolute discrepancies between actigraphy and polysomnography and between sleep diary and polysomnography $(\mathrm{N}=17)$.

|  | Relative discrepancies |  |  |  | Absolute discrepancies |  |  |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Night 1 |  | Night 2 |  | Night 1 |  | Night 2 |  |
|  | Mean (SD) | d | Mean (SD) | d | Mean (SD) | d | Mean (SD) | d |
| Total sleep time |  |  |  |  |  |  |  |  |
| ACT-PSG | -8.56 (59.97) | 1.06 | -38.29 (45.29) | 0.19 | 44.57 (39.32) | -0.96 | 45.59 (37.36) | -0.36 |
| SD-PSG | -72.23 (100.30) |  | -46.89 (72.41) |  | 82.40 (91.52) |  | 59.17 (62.02) |  |
| Total wake time |  |  |  |  |  |  |  |  |
| ACT-PSG | 6.66 (60.12) | -0.99 | 35.36 (46.43) | -0.39 | 45.21 (38.37) | -0.90 | 43.31 (38.55) | -0.63 |
| SD-PSG | 66.39 (95.60) |  | 53.49 (75.52) |  | 79.65 (84.06) |  | 67.67 (62.15) |  |
| Sleep efficiency, \% |  |  |  |  |  |  |  |  |
| ACT-PSG | -2.74 (12.46) | 1.08 | -8.23 (9.81) | 0.23 | 9.21 (8.51) | -1.02 | 9.93 (7.93) | -0.41 |
| SD-PSG | -16.23 (22.24) |  | -10.48 (14.79) |  | 17.93 (20.80) |  | 13.16 (12.29) |  |
| Sleep-onset latency |  |  |  |  |  |  |  |  |
| ACT-PSG | -14.16 (21.59) | -1.79 | -7.01 (11.77) | -2.50 | 14.16 (21.59) | -0.60 | 11.27 (7.41) | -1.63 |
| SD-PSG | 24.51 (32.13) |  | 22.39 (20.63) |  | 27.05 (29.86) |  | 23.34 (19.47) |  |
| Time in bed |  |  |  |  |  |  |  |  |
| ACT-PSG | 4.00 (8.13) | 0.48 | 0.39 (7.58) | -0.35 | 6.28 (6.40) | -0.31 | 6.02 (4.33) | -0.39 |
| SD-PSG | 0.07 (11.24) |  | 3.06 (10.97) |  | 8.29 (7.25) |  | 7.71 (8.16) |  |

ACT-PSG $=$ discrepancy between actigraphy and polysomnography; SD-PSG $=$ discrepancy between sleep diary and polysomnography. $d=$ Cohen's $d$ effect size (Cohen, 1988).


## Measures

## Initial Screening and Clinical Evaluation

The initial screening included a 20 -minute telephone questionnaire administered to determine participants' eligibility for the study. A subsequent multimeasure pretreatment evaluation was composed of a semistructured sleep-history interview ${ }^{22}$ to diagnose insomnia, the SCID-IV ${ }^{20}$ to evaluate the presence of psychologic disorders, and a physical examination. Participants were enrolled in a 10 -week treatment that comprised medication (zopiclone) and cognitive behavior therapy. Additional information about the treatment protocol and treatment outcome is reported elsewhere. ${ }^{23}$

## Polysomnography

The PSG montage included electroencephalographic, electromyographic, and electrooculographic monitoring. Sleep stages, respiratory disturbance, and limb movements were scored by an experienced clinician according to standard criteria. ${ }^{24}$ Respiration (airflow, tidal volume, and oxygen saturation) and anterior tibialis electromyographic readings were recorded during the first night to detect sleep apnea and periodic limb movements. Variables used for the present study were total wake time (TWT), TST, SOL, sleep efficiency (SE: ratio of TST to TIB), and TIB.

## Actigraphy

When participants slept in the laboratory, they also wore an actigraph from IM Systems (Individual Monitoring Systems, Inc., Baltimore, MD). Data were processed and scored for the following variables: TST, TWT, SOL, SE, and TIB with the IM Systems, Inc., software and algorithm (version 3.15a). The variable WASO was not used in the study because it was not directly computed by IM System's software.

## Sleep Diaries

Following each night spent in the laboratory, participants completed their sleep diaries. From these
diaries, an estimate was computed for a nightly average of TWT, TST, SOL, SE, and TIB.

## Procedures

Participants came to the laboratory for PSG recordings on 3 consecutive nights at baseline and 2 consecutive nights at posttreatment. On those same nights, they wore an actigraph, and the next morning, they completed their sleep diary. Same-night data from diaries, actigraphy, and PSG were compared. Data from the first baseline night were not used to allow for an adaptation to the laboratory. Thus, the 2 nights using the 3 devices (PSG, actigraphy, and sleep diary) before treatment and the 2 nights after treatment were used in the analysis. Pretreatment readings were obtained from 17 participants for a total of 34 nights. At posttreatment, the sample was smaller as 1 participant dropped out of treatment, 5 participants did not return for their laboratory evaluation, 1 came for only 1 night, 1 refused to wear the actigraph because it was uncomfortable to sleep with it during the night, and the data for 1 subject were unavailable due to a broken device. Thus, a total of 16 nights were obtained for 8 participants after treatment. The sleep variables compared across the 3 assessment tools were TST, TWT, SOL, SE, and TIB.

## Data Analysis Plan

Data analysis included 3 steps. The first step involved computing relative and absolute discrepancies between actigraphy and PSG, and between sleep diary and PSG. Relative discrepancies were computed by retaining negative or positive signs of the differences between actigraphy and PSG. Absolute discrepancies were computed based only on absolute values of actigraphy minus PSG (ie, $\mid$ actigraphy - PSG $\mid$ ). For example, if actigraphy recorded 350 minutes of sleep time and PSG 375 minutes, then the relative discrepancy was equal to -25 and the absolute discrepancy was equal to 25 . Means of relative and absolute differences were then computed. A repeated-measure analysis of variance (ANOVA) with 2 within-subject effects ( 2 nights and 2 differences) was used to evaluate the magnitude of the discrepancies (actigraphy - PSG and sleep diary - PSG). In addition, Cohen's $d$ effect sizes were computed for each

Table 2-Median, minimum, and maximum actigraphic and sleep diary objective estimates for each night and sleep variable


For nights 1 and $2, \mathrm{~N}=17$ and for nights 3 and $4, \mathrm{~N}=8$. Actigraphy/PSG $=$ ratio of actigraphy data to polysomnography data X 100 ; Sleep diary/PSG = ratio of sleep diary data to polysomnography data X 100. Both ratios were adapted from Edinger and Fins (1995) objective sleep estimate (OSE) formula.
dependant variable. This statistic expresses the difference between 2 means in standard deviation units. ${ }^{25}$ Thus, Cohen's $d$ provides a standardized magnitude of difference between means. The second step was to assess the accuracy of measurement of each device with the objective sleep time estimate (OSE) formula proposed by Edinger and Fins²: OSE $=($ MSE $/$ MSA $) \times 100$. In this formula, MSE represents minutes of sleep estimated by the sleep diary or actigraphy and MSA represents the minutes of actual sleep time obtained by PSG. Therefore, an OSE value of $100 \%$ indicates a perfect concordance between the actigraphy or sleep diary and the PSG assessment. This formula was adapted to compute an objective estimate of each sleep variable for actigraphy and the sleep diary. Thus, MSE becomes the estimated value and MSA the actual value obtained by PSG. Descriptive statistics of the sample distribution were computed for the OSE for each variable. The next step to assess the accuracy involved computing a Spearman correlation coefficient between each device at the 2 baseline nights. The Spearman correlation coefficient was used because of the small sample size. The fourth and final step was to compute descriptive statistics for all dependent variables at baseline and after treatment in order to examine the sensitivity of the 3 devices to treatment effect. A repeated-measure ANOVA with 3 within-subject effects ( 2 times, 2 nights, and 3 devices) was used to control for the internight variability of each subject (intrasubject effect). In order to decrease type I error, the $\alpha$ was adjusted with Bonferroni to .01 .

## RESULTS

## Relative and Absolute Discrepancies Between Devices

Means and SD of relative and absolute discrepancies for all sleep variables between actigraphy and PSG, as well as between sleep diary and PSG, are presented in Table 1. Repeated-measure ANOVAs with 2 with-in-subject effects ( 2 nights and 2 discrepancies) indicate significant differences for relative discrepancies for SOL, $F(1,14)=26.21, p<.0001$. Therefore, relative discrepancies between actigraphy and PSG were significantly smaller than relative discrepancies between sleep diary and PSG at both nights. There was no other significant difference for absolute or relative discrepancies for any of the remaining variables. There was a large effect size on night $1(d=1.06)$ for relative discrepancies and a moderate effect size on nights 1 and $2(d s=-0.96$ and -0.36 , respectively) for absolute discrepancies. Magnitudes of the discrepancies between sleep diary - PSG and actigraphy - PSG for TIB were much smaller and nonsignificant.

## Objective Estimates

The distributions of objective estimates of TST (OSE; sleep diary/PSG and actigraphy/PSG) for each night are illustrated in Figure 1. Visual inspection of the data revealed that the majority of the participants had an OSE slightly lower than or close to 100 , indicating that sleep diary and actigraphy estimates of TST were slightly lower than the PSG measures. In addition, the range of actigraph OSE scores was narrower than the range of sleep-diary OSE scores. Objective estimates of TST from actigraphy were generally closer to PSG measures than were diary estimates. Descriptive statistics (medians, minimum, and maximum) of actigraphy and sleep-diary OSE for all sleep variables are presented in Table 2. Again, the data revealed that for all sleep variables, the range of OSE scores was smaller for actigraphy than for sleep diary. Medians for TST and SE were near 100 and were similar for both devices. However, the range of OSE scores for each sleep variable was more restricted for actigraphy than for sleep diary. The TWT medians indicated that both devices overestimated TWT relative to PSG. For SOL, objective estimates revealed that actigraphy underestimated SOL and sleep diary overestimated it.

## Correlations Between Devices at Baseline

Spearman correlation analyses revealed significant positive correlations between PSG and actigraphy only at the second baseline night on

TWT, TST, and SE ( $r \mathrm{~s}=.52, .71$, and .57 , respectively, $p \mathrm{~s}=.05, .003$, .03). For TIB, there were significant positive correlations between devices at each of the 2 nights (for night $1: r \mathrm{~s}=.91, .90$, and $.88, p \mathrm{~s}=$ .0001 . For night $2: r s=.97, .94$, and $.97, p s=.0001$ ). There was no other significant correlation between devices.

## Sensitivity of the 3 Devices to Treatment Effect

Table 3 presents means and SD for 5 dependent sleep variables (TST, TWT, SE, SOL, and TIB) as measured with the 3 devices (PSG, actigraphy, and sleep diary) for 2 nights at baseline and 2 nights after treatment. Repeated-measure ANOVAs with 3 within-subject effects ( 2 times, 2 nights, and 3 devices) revealed significant decreases for TWT and TIB and a significant increase for SE from baseline to posttreatment, $F \mathrm{~s}(1,6)$ $=45.83,31.80$, and 33.41, respectively, $p \mathrm{~s}<.001$. Repeated-measure ANOVAs also revealed a significant decrease in TST and SOL from baseline to posttreatment, $F \mathrm{~s}(1,6)=5.71$ and 6.30 , respectively, $p \mathrm{~s}<.05$, which would have been significant at $p=.01$ with a sample size of 19 . These time effects from baseline to posttreatment indicated that changes were detected over time with all these assessment devices.

Significant device effects were obtained for SOL, $F(2,12)=8.16, p=$ .006 , as well as for TWT, SE, and TST, $F \mathrm{~s}(2,12)=4.39,4.57$, and 3.68 , respectively, $p s=.04$. These later effects would have been significant at $p=.01$ with a sample size of 22 . These effects indicated that data collected with the 3 devices differed for these sleep variables. Pairwise comparisons revealed that only sleep-diary data differed significantly from PSG data $(p s=.03)$ for these 3 sleep variables. No night effect or interaction such as night by device or device by time effects was significant.

## CONCLUSION

The present findings provide evidence supporting the sensitivity and clinical utility of actigraphy in objectively documenting treatment response in chronic insomnia. Indeed, actigraphy detected changes on all

Table 3-Polysomnography, actigraphy, and sleep-diary data for each night and each sleep variable ( $\mathrm{N}=8$ ).

|  | Polysomnography |  | Actigraphy |  | Sleep Diary |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | Mean | (SD) | Mean | (SD) | Mean | (SD) |
| Total sleep time (minutes) |  |  |  |  |  |  |
| Night 1 | 391.63a | (52.64) | $362.57^{\text {a }}$ | (49.95) | $327.14^{\text {a }}$ | (94.11) |
| Night 2 | $388.03^{\text {a }}$ | (56.07) | $349.00^{\text {a }}$ | (50.13) | $309.43^{\text {a }}$ | (53.41) |
| Night 3 | $349.00^{\text {b }}$ | (52.85) | $316.29{ }^{\text {b }}$ | (30.74) | $307.14{ }^{\text {b }}$ | (67.69) |
| Night 4 | $362.76{ }^{\text {b }}$ | (49.53) | $333.43{ }^{\text {b }}$ | (34.65) | $322.86^{\text {b }}$ | (81.64) |
| Total | 372.85 | (17.78) | 340.32 | (10.16) | 316.64 | (24.21) |
| Total wake time (minutes) |  |  |  |  |  |  |
| Night 1 | $75.56^{\text {a }}$ | (51.57) | 104.29a | (50.37) | $136.43{ }^{\text {a }}$ | (92.63) |
| Night 2 | $71.20^{\text {a }}$ | (30.49) | $109.29^{\text {a }}$ | (55.42) | $165.71^{\text {a }}$ | (78.82) |
| Night 3 | $36.29^{\text {b }}$ | (24.40) | $72.00^{\text {b }}$ | (19.33) | $80.00^{\text {b }}$ | (49.24) |
| Night 4 | $24.51^{\text {b }}$ | (12.23) | $50.29{ }^{\text {b }}$ | (15.48) | $61.43{ }^{\text {b }}$ | (45.53) |
| Total | 51.89 y | (8.58) | 83.96 yz | (12.49) | 110.89 z | (17.07) |
| Sleep efficiency (\%) |  |  |  |  |  |  |
| Night 1 | $84.49^{\text {a }}$ | (10.99) | $77.86^{\text {a }}$ | (10.43) | $70.60^{\text {a }}$ | (19.74) |
| Night 2 | $85.00^{\text {a }}$ | (7.65) | $76.43{ }^{\text {a }}$ | (10.66) | $67.53{ }^{\text {a }}$ | (12.17) |
| Night 3 | $91.06^{\text {b }}$ | (7.64) | $81.43{ }^{\text {b }}$ | (4.16) | $78.96{ }^{\text {b }}$ | (13.20) |
| Night 4 | $94.09^{\text {b }}$ | (4.17) | $85.86{ }^{\text {b }}$ | (3.98) | $83.16^{\text {b }}$ | (13.67) |
| Total | 88.66 y | (2.35) | 80.39 yz | (2.41) | $75.06{ }^{\text {z }}$ | (4.09) |
| Sleep-onset Latency (minutes) |  |  |  |  |  |  |
| Night 1 | $16.40^{\text {a }}$ | (8.87) | $6.57{ }^{\text {a }}$ | (2.88) | $30.00^{\text {a }}$ | (20.41) |
| Night 2 | $17.70^{\text {a }}$ | (7.83) | $4.43{ }^{\text {a }}$ | (5.00) | $40.00^{\text {a }}$ | (27.84) |
| Night 3 | $9.73{ }^{\text {b }}$ | (7.12) | $4.00{ }^{\text {b }}$ | (3.27) | $30.71^{\text {b }}$ | (40.04) |
| Night 4 | $6.86{ }^{\text {b }}$ | (1.82) | $3.29{ }^{\text {b }}$ | (3.45) | $17.86{ }^{\text {b }}$ | (13.18) |
| Total | 12.67 y | (2.21) | 4.57 yz | (0.73) | $29.64{ }^{\text {z }}$ | (8.15) |
| Time in bed (minutes) |  |  |  |  |  |  |
| Night 1 | $463.87^{\text {a }}$ | (22.54) | $466.86{ }^{\text {a }}$ | (22.77) | $463.57^{\text {a }}$ | (24.28) |
| Night 2 | 455.23a | (34.89) | 458.29a | (40.07) | $460.86^{\text {a }}$ | (38.99) |
| Night 3 | $381.73{ }^{\text {b }}$ | (33.66) | $388.29{ }^{\text {b }}$ | (35.41) | 387.14b | (34.14) |
| Night 4 | $384.31^{\text {b }}$ | (39.91) | $383.86{ }^{\text {b }}$ | (41.01) | $385.71{ }^{\text {b }}$ | (41.07) |
| Total | 421.29 y | (10.46) | 424.32 ${ }^{\text {z }}$ | (10.03) | 424.32 y | (10.59) |

For each variable, means in the same column and in the same row that do not share the same subscripts ( ${ }^{\mathrm{a}}$ and $^{\mathrm{b}}{ }^{\mathrm{b}}$ or ${ }^{\mathrm{y}}$ and ${ }^{\mathrm{z}}$ ) differ at $p<.05$.
sleep variables after treatment. Furthermore, discrepancies between actigraphy and PSG were smaller than those obtained between sleep diary and PSG, suggesting that actigraphy was more accurate than the sleep diary. Actigaphic data correlated positively with most of the PSG data. Correlations were higher for TST and TIB than for SOL or TWT, suggesting that actigraphy is more accurate for global than for more discrete sleep variables. Objective estimates of TST and SE were generally slightly lower than or close to $100 \%$ for actigraphy. Furthermore, the range of scores of OSE for actigraphy was smaller than those for the sleep diary.

Taken together, these results suggest that actigraphy is a reliable method for assessing sleep-wake patterns and for monitoring treatment response among insomnia patients. First, the results show that actigraphy is as sensitive to changes in sleep parameters as PSG and sleep diary. Second, the most important difficulty with actigraphy appears to be in estimating SOL, which is underestimated by actigraphy and overestimated by the sleep diary. Therefore, using actigraphy with patients with primarily sleep-onset difficulties may lead to an underestimation of insomnia severity. Such limitations need to be taken into account when interpreting SOL data without PSG. Third, actigraphy provides a reliable method for assessing TIB, an important treatment target when using sleep-restriction procedures. This result concurs with those of previous studies that have used actigraphy as an outcome measure ${ }^{9,10,13}$ and provides additional evidence supporting the use of actigraphy as a reliable measure of compliance with behavioral treatment for insomnia. Actigraphy may actually promote treatment compliance. Indeed, because patients know that the actigraph monitors movement, they may be more inclined to adhere to prescribed behavioral recommendations. Therefore, actigraphy not only is an assessment tool to monitor outcome, but can also promote treatment compliance at home.

The findings regarding discrepancies show that using absolute discrepancies alone may mask important differences between PSG and actigraphy. Indeed, our results on absolute discrepancies concur with those of previous studies, ${ }^{6,10,17}$ where it was shown that actigraphy relative to PSG inflated sleep time. On the other hand, examinations of the relative discrepancies ( -8.56 and -38.29 minutes) and of the OSE suggest that actigraphy compared to PSG underestimates TST, which is also consistent with at least 1 other study. ${ }^{13}$ Therefore, there is a need to develop other methods to analyze data collected from different assessment devices and methods that will lead to more convergent findings about the underestimation or overestimation of sleep-wake parameters with actigraphy. Additional investigations are needed on this issue.

The present results should be interpreted cautiously given some methodologic limitations, including the small sample size and missing data due to technical problems. In addition, the results may not generalize to samples of patients with insomnia secondary to medical ${ }^{16}$ or psychiatric disorders. The fact that the actigraph was damaged during treatment underscores the importance of using more than 1 assessment device to measure sleep and wakefulness. Furthermore, since insomnia is a complex syndrome including physiologic and psychologic components, ${ }^{1}$ it is necessary to use multiple measures to capture all of its different components. ${ }^{26}$

In conclusion, this study provides additional evidence supporting the clinical utility of actigraphy for assessing sleep among insomnia patients. Our findings are consistent with the conclusions of a recent update of practice parameters on the role of actigraphy in the study of sleep. ${ }^{27}$ The potential impact of actigraphy on promoting adherence to behavioral treatment should also be investigated in additional studies. Finally, these preliminary findings should be replicated with larger samples, and, until then, actigraphy should be used only as an adjunct to PSG or the sleep diary.

## REFERENCES

1. Sateia MJ, Doghramji K, Hauri PJ, Morin CM. Evaluation of chronic insomnia. An American Academy of Sleep Medicine review. Sleep 2002;23:243-308.
2. Edinger JD, Fins AI. The distribution and clinical significance of sleep time mispercep-
tions among insomniacs. Sleep 1995;18:232-9.
3. Sadeh A, Hauri PJ, Kripke DF, Lavie P. The role of actigraphy in the evaluation of sleep disorders. Sleep 1995;18:288-302.
4. Verbeek I, Klip EC, Declerck AC. The use of actigraphy revised: the value for clinical practice in insomnia. Percept Mot Skills 2001;92:852-6.
5. Jean-Louis G, von Gizycki H, Zizi F, Spielman A, Hauri PJ, Taub H. The actigraph data analysis software: I. A novel approach to scoring and interpreting sleep-wake activity. Percept Mot Skills 1997;85:207-16.
6. Hauri PJ, Wisbey J. Wrist actigraphy in insomnia. Sleep 1992;15:293-301.
7. Monk TH, Reynolds CF III, Kupfer DJ, et al. The Pittsburgh sleep diary. J Sleep Res 1994;3:111-20.
8. Mullaney DJ, Kripke DF, Messin S. Wrist-actigraphic estimation of sleep time. Sleep 1980;13:83-92.
9. Schmidt-Nowara WW, Beck AA, Jessop CA. Actigraphic assessment of a treatment trial of sleep restriction in chronic insomnia. Sleep Res 1992;21:259.
10. Friedman L, Benson K, Noda A, et al. An actigraphic comparison of sleep restriction and sleep hygiene treatments for insomnia in older adults. J Geriatr Psychiatry Neurol 2000;13:17-27.
11. Verbeek I, Arends J, Declerck G, Beecher L. Wrist actigraphy in comparison with polysomnography and subjective evaluation in insomnia. Sleep-Wake Research in the Netherlands. 1994;5:163-9.
12. Beck AA, Schmidt-Nowara WW, Jessop CA. Relation of actigraphy to sleep logs in insomniacs. Sleep Res 1992;21:171.
13. Brooks JO, Friedman L, Bliwise DL, Yesavage JA. Use of the wrist actigraph to study insomnia in older adults. Sleep 1993;16:151-5.
14. Krahn LE, Lin SC, Wisbey J, Rummans TA, O'Connor MK. Assessing sleep in psychiatric inpatients: nurse and patient reports versus wrist actigraphy. Ann Clin Psychiatry 1997;9:203-10.
15. Wicklow A, Espie CA. Intrusive thoughts and their relationship to actigraphic measurement of sleep: towards a cognitive model of insomnia. Behav Res Ther 2000;38:679-83.
16. Wilson KG, Watson ST, Currie SR. Daily diary and ambulatory activity monitoring of sleep in patients with insomnia associated with chronic musculoskeletal pain. Pain 1998;75:75-84.
17. Cole RJ, Kripke DF, Gruen W, Mullaney DJ, Gillin JC. Automatic sleep/wake identification from wrist activity. Sleep 1992;15:461-9.
18. Jean-Louis G, von Gizycki H, Zizi F, et al. Determination of sleep and wakefulness with the actigraph data analysis software (ADAS). Sleep 1996;19:739-43.
19. Sadeh A, Sharkey KM, Carskadon MA. Activity-based sleep-wake identification: an empirical test of methodological issues. Sleep 1994;17:201-7.
20. First MB, Spitzer RL, Gibbon M, Williams JBW. Structured Clinical Interview for DSM-IV Axis I Disorders, Clinical Version (SCID-IV). Washington, DC: American Psychiatric Association; 1997.
21. Diagnostic and Statistical Manual of Mental Disorders. 4th ed. Washington, DC: American Psychiatric Association; 1994.
22. Morin CM. Insomnia: Psychological Assessment and Management. New York: The Guilford Press; 1993.
23. Vallières A, LeBlanc M, Guay B, Morin CM. Sequential use of medication and behavioral therapies for chronic insomnia. Sleep 2002;25:A69
24. Rechtschaffen A, Kales A. A manual of standardized terminology, techniques, and scoring system for sleep stages of human subjects. Los Angeles: Brain Information Service/Brain Research Institute, UCLA; 1968.
25. Cohen J. Statistical Power Analysis for the Behavioral Sciences. 2nd ed. Mahwah, NJ: Lawrence Erlbaum Associates, Inc; 1988.
26. Kazdin AE. Research Design in Clinical Psychology. 3rd ed. Boston: Allyn \& Bacon; 1998.
27. Ancoli-Israel S, Cole R, Alessi C, Chambers M, Moorcroft W, Pollack CP. The role of actigraphy in the study of sleep and circadian rhythms. Sleep 2003;26:342-92.
