Obstructive sleep apnea (OSA) is a prevalent and chronic disorder characterized by recurrent collapse of the upper airway during sleep producing frequent apnea and hypopnea events. Eleven percent of women and 25% of men aged 40 years or older have at least 15 apneas plus hypopneas per hour of sleep (apnea-hypopnea index, AHI).1 OSA is associated with multiple adverse outcomes, including pathological daytime sleepiness,7 a ~2-fold increase in motor vehicle accidents,8 depression,4 hypertension,9 and all-cause mortality.6 The main treatments include continuous positive airway pressure (CPAP), mandibular advancement splint devices, weight loss, and upper airway or bariatric surgery. Upper airway function is typically more compromised in the supine posture, such that therapies designed to discourage supine sleep have a significant potential treatment role, as an adjunct or primary treatment in appropriately selected patients.

In OSA patients, unfavorable gravitational effects in supine sleep lead to an increased propensity for and frequency of upper airway collapse,7,8 higher airway opening pressures,9 and more prolonged and severe respiratory events with greater oxygen desaturation.10 Positional OSA, traditionally defined as supine AHI ≥ twice that of non-supine postures, has a prevalence of 50% to 60%.7,11,12 According to a more recent and stringent definition requiring a supine AHI ≥ twice that of non-supine AHI and normalization of AHI in non-supine postures,13 positional OSA patients who meet these criteria should remain within normal limits by using effective positional therapy alone to simply avoid supine sleep. Approxi-
mately 30% of clinic-referred OSA patients exhibit positional OSA on this basis.\textsuperscript{13,14}

Snoring is a cardinal feature of OSA and results from vibrations of a partially obstructed upper airway during sleep.\textsuperscript{15} Snoring may have deleterious health consequences in its own right, including vibration damage-induced carotid atherosclerosis\textsuperscript{16,17} and potentially carotid plaque rupture and stroke. Snoring very frequently disrupts the sleep of others, particularly affecting bed partner sleep and quality of life.\textsuperscript{18} While there are some anecdotal reports of marked reductions in snoring with supine sleep avoidance, only one study appears to have examined the impact of positional therapy on snoring in positional OSA patients via objective snoring measurements.\textsuperscript{19} This used indirect flow measurements without sound recordings, and found that snoring time remained unchanged with a severely restrictive positioning apparatus that was not tolerated by 22% (5/23) of patients studied.\textsuperscript{19}

Several positional therapies have been devised, including the classic “tennis ball technique,” in which a tennis ball strapped to the back is used to discourage supine sleep. Although this technique is effective,\textsuperscript{20} recent data from our laboratory strongly suggest that patients rarely comply with this treatment longer term.\textsuperscript{21} After an average follow-up time of ~30 months, only ~6% of patients reported continuing use, the main reason for discontinuation being excessive discomfort associated with the ball-on-back design.\textsuperscript{21} While a minority of patients report having learned to avoid supine sleep without continued treatment,\textsuperscript{21,22} objective data to support the long-term success of supine avoidance and treatment adherence are currently lacking for supine-avoidance therapies in general.\textsuperscript{23}

The aims of this study were to evaluate the position recording accuracy and acute treatment effectiveness of a novel body position orienting device. The device monitors sleep position and activates a vibration alarm on the sternum, designed to facilitate effective vibration transmission with minimal disturbance to others, to alert and discourage the patient from sleeping supine. An abstract of the preliminary results has previously been published.\textsuperscript{24}

### METHODS

This project was approved by the Repatriation General Hospital and University of Adelaide Human Research and Ethics Committees. All participants gave written informed consent.

**Body Position Orienting Device**

The position monitoring and supine alarm device (Buzz-POD, Gorman ProMed Pty Ltd, Victoria, Australia) is a small, light-weight, battery-powered device (80 × 40 × 20 mm, 50 g, 2 × AAA batteries) that is fastened to the chest using a Velcro strap (**Figure 1**). The device logs body posture (left, right, prone, supine) at 1 Hz, using internal position-sensitive tilt switches. A vibrating component can be programmed to remain inactive or to activate after the detection of 5 consecutive sec of supine sleep (to facilitate movement/turning without unnecessary vibrations). A patient event-mark button allows for recording of bed and rise times to identify the sleep period and facilitate monitoring of treatment adherence. The device can store 3 weeks of continuous position, alarm, and event data and is configured and data uploaded through custom software via universal serial bus (USB) connection and personal computer.

**Validation of Position Recording Accuracy**

Seventeen patients undergoing routine diagnostic sleep studies at the Adelaide Institute for Sleep Health were recruited and wore the position monitoring device in addition to standard polysomnographic measures, with the device programmed to record sleep position without vibration. For comparison, an infrared video camera (ICX-IR480/25M, Finest Security System Co. Ltd, Songshan District, Taipei, Taiwan) inside the study bedroom and in-laboratory position sensor (Compumedics P/N 7000-0057-00 sampled at 1 Hz) was used to record sleep position throughout the night.

**Data and Statistical Analysis**

Device and in-lab posture data recorded at 1 Hz were classified as left, right, supine, or prone in each 30-sec epoch according to the predominant posture registered in each epoch. The same posture classifications were applied to video recordings on an equivalent 30-sec epoch basis using playback at 8 times scored normal speed by an investigator (JB) blinded to position monitoring device, and using in-lab position sensor data.

Agreement was evaluated using the group-averaged κ statistic\textsuperscript{25} (i.e., the chance-corrected proportion of agreement) determined from each epoch throughout the night within each patient. Kappa values can range from −1 (complete disagreement) to +1 (perfect agreement). Agreement was also assessed via Bland-Altman analysis\textsuperscript{26} of supine time according to the 2 measurement methods.

**Efficacy of Supine-Avoidance Treatment in Positional OSA Patients**

**Subjects**

Positional OSA patients (overall AHI ≥ 15/h; supine AHI ≥ twice the non-supine AHI; ≥ 20 min of sleep in supine and non-

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**Figure 1**—Subject wearing the position monitoring and supine alarm device

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supine postures; and non-supine AHI < 15/h) identified from diagnostic sleep studies conducted at the Adelaide Institute for Sleep Health between January and July 2009 were invited to participate. Apneas and hypopneas were scored from polysonography records using the Chicago criteria. A cutoff of 15/h using these criteria corresponds to approximately 5/h using more recent AASM “recommended” criteria. Exclusion criteria were existing treatment(s) for OSA; mobility-limiting problems inhibiting lateral sleep; and a cardiac pacemaker (given unknown potential for device interference). Where applicable, patients’ bed partners were also invited to participate, via informed consent, to complete questionnaires regarding patient snoring and potential sleep disturbance from the device.

Protocol

Patients were instructed to wear the position monitoring device each night in a 3-week in-home randomized controlled crossover trial. The trial comprised 1 week of active vibration and 1 week of inactive vibration in a random order, separated by an intervening washout week without vibration to control for potential carryover or training effects, with a previous study by an intervening washout week without vibration to control for and 1 week of inactive vibration in a random order, separated crossover trial. The trial comprised 1 week of active vibration device each night in a 3-week in-home randomized controlled protocol via informed consent, to complete questionnaires regarding patient snoring and potential sleep disturbance from the device.

Data Analysis

Nightly supine time as a percentage of the sleep period was determined based on the interval between successive event markings in conjunction with sleep diary-recorded bed and rise times. Patient noncompliance was assessed and deemed present if only one posture registered for an entire nightly period, strongly suggesting failure to wear the position monitoring device.

To investigate the temporal characteristics of supine periods during inactive and active treatment, the frequencies of transitions to the supine posture lasting ≥ 5 sec were averaged across nights within each condition. For inactive treatment, the average duration of these periods was calculated, while for active treatment, average alarm time was determined for alarm events of any length.

Type III monitor recordings were analyzed using the accompanying software (Download 3 v1.2, Stowood Scientific Instruments Ltd) following randomization of all fully de-identified records by another investigator to ensure analysis blinding. Raw signals were manually reviewed before analysis and periods in which the cannulae or finger sensor were detached from the patient or in which the patient was clearly not asleep (e.g., talking with partner in sound recording) were excluded. In the remaining periods, Download 3 automatically calculated the apnea index, hypopnea index, and AHI as the number of apneas, hypopneas, and apneas plus hypopneas, respectively, per hour of recording. Apnea and hypopnea were defined as a reduction in airflow amplitude of > 90% and of 50% to 90%, respectively, lasting 10-100 sec. Minimum overnight oxygen saturation and 4% oxygen desaturation index were also calculated. AHI values from this type III monitor have previously been validated against laboratory polysomnography-determined AHI, with an area under the receiver operator curve for AHI ≥ 10/h of 0.96.

A “snore” was automatically scored when there was a spike in sound intensity ≥ 50 decibels (dBA), whether during inspiration or expiration. This threshold was chosen because it has been used previously to identify snoring, and was above background noise (mean ± SD, 31.4 ± 1.8 dB) and normal breathing sounds (which registered up to 45 dB). Following automated analysis, sound recordings were manually reviewed, and identifiable artifacts such as teeth grinding were removed. Snoring was subsequently quantified by frequency (noises/h) and by mean duration (in milliseconds). While the analysis software did not allow snoring intensity to be examined directly, snoring frequency was calculated for “soft” (≥ 50, < 60 dB), “moderate” (≥ 60, < 70 dB) and “loud” (≥ 70 dB) snores as well as overall (≥ 50 dB). The 50, 60, and 70 dB cutoffs correspond approximately to the loudness of a quiet conversation, a ringing telephone, and a vacuum cleaner, respectively.

Statistical Analysis

The primary endpoint was AHI, and all analyses were conducted on an intention-to-treat basis. The sample size chosen was based on the results of 2 previous crossover trials of positional therapy. Using AHI as the primary endpoint, we estimated that a within-subject difference of 9/h between active and inactive treatment (SD 11/h) could be detected with a sample size of 15, 2-tailed significance level of 5%, and power of 80%.
To test for potential learning and carryover effects on supine time among patients randomized to active treatment first, intervening supine time data were analyzed in isolation for treatment order and order by night effects using linear mixed model analysis with an autoregressive covariance structure (SPSS v17.0, SPSS Inc., Chicago, IL, USA). Mixed-model analysis was also used to compare AHI and other endpoints between conditions (inactive vs active) and, where applicable, across nights (1-7). Treatment order and AHI recording night (6 vs 7) were initially included as factors in relevant models to assess for order and night effects, but were subsequently removed when nonsignificant. Where applicable, Bonferroni-adjusted custom post hoc contrasts were used to further examine significant interaction and/or main effects. Unless otherwise indicated, data are expressed as mean ± SEM or, if non-normally distributed, as median (interquartile range, IQR). p < 0.05 was considered statistically significant.

RESULTS

Validation of Position Recording Accuracy
Patients were predominantly male (11/17 patients) with a mean ± SD age and body mass index of 49.7 ± 15.7 years and 34.6 ± 8.4 kg/m², respectively. Based on a median of 830 epochs per patient (IQR: 715-905), the median κ statistic of posture recording agreement between the position monitoring device and video recordings was 0.95 (IQR 0.88-1.00), indicating close agreement. A Bland-Altman plot of supine time agreement is shown in Figure 2. The mean difference (bias) in supine time between the position monitoring device and video recordings was 0.3%, with 95% limits of agreement of −4.0% to 4.6%. Agreement between video and in-laboratory position sensor recordings were similar but appeared to be more variable (median κ 0.96, IQR 0.47-0.99, supine time bias 1.1%, 95% limits of agreement −13.6% to 15.5%).

Efficacy of Supine Avoidance in Positional OSA Patients
Thirty-five positional OSA patients were approached. Ten declined (3 reported they rarely slept supine, 2 had upcoming travel commitments, and 5 gave no reason), and 9 were already on OSA treatment. Thus, 16 patients with supine-predominant OSA consented and were randomized into the trial, with all but one completing the study protocol. This patient withdrew on the first night due to intolerance of the position monitoring device chest strap and vibration alarm, leaving no data available for analysis. All 15 patients who completed the study contributed AHI and snoring data, but supine time data were not available for one patient because of a position monitoring device upload failure. Seven patient bed partners completed the questionnaires.

Table 1—Baseline demographic and clinical characteristics of patients completing the efficacy trial (n = 15)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Patient Characteristics</strong></td>
<td></td>
</tr>
<tr>
<td>Age (y)</td>
<td>58.2 ± 13.9</td>
</tr>
<tr>
<td>Sex (M/F)</td>
<td>13/2</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>83.5 ± 13.1</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>169.8 ± 10.5</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>28.8 ± 2.5</td>
</tr>
<tr>
<td><strong>Laboratory sleep study</strong></td>
<td></td>
</tr>
<tr>
<td>Total sleep time (h)</td>
<td>6.7 ± 1.2</td>
</tr>
<tr>
<td>Sleep efficiency (%)</td>
<td>81.4 ± 9.2</td>
</tr>
<tr>
<td>Supine sleep time (h)</td>
<td>2.3 ± 1.3</td>
</tr>
<tr>
<td>Supine sleep (%)</td>
<td>36.4 ± 20.6</td>
</tr>
<tr>
<td><strong>Arousal Index (events/h)</strong></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>17.3 ± 6.9</td>
</tr>
<tr>
<td>Respiratory</td>
<td>5.3 ± 3.3</td>
</tr>
<tr>
<td>Spontaneous</td>
<td>6.0 ± 2.4</td>
</tr>
<tr>
<td><strong>Apnea-hypopnea Index (events/h)</strong></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>24.1 ± 10.5</td>
</tr>
<tr>
<td>REM</td>
<td>30.5 ± 18.4</td>
</tr>
<tr>
<td>NREM</td>
<td>22.8 ± 10.8</td>
</tr>
<tr>
<td>Supine</td>
<td>51.3 ± 23.3</td>
</tr>
<tr>
<td>Non-supine</td>
<td>9.7 ± 3.9*</td>
</tr>
<tr>
<td>REM: NREM ratio</td>
<td>1.5 (IQR 0.8 - 2.2)</td>
</tr>
<tr>
<td>Supine: non-supine ratio</td>
<td>5.3 (IQR 3.3 - 9.2)</td>
</tr>
</tbody>
</table>

Values are mean ± SD or median (IQR). M, male; F, female; BMI, body mass index; AHI, apnea-hypopnea index. *p < 0.001 vs supine.
in-home supine time with inactive treatment (36.6% ± 5.7% vs 19.3% ± 4.3%, p = 0.002).

The frequency of transitions to the supine position was not different between inactive and active treatment (1.7 ± 0.5 vs 1.3 ± 0.3 transitions/h). During inactive treatment supine periods lasted 8.2 ± 1.9 min. During active treatment, average alarm and supine time (excluding programmed 5-sec delay) was 9.2 ± 4.1 sec before transition to another posture.

Total AHI was reduced in the order of 45% with active treatment (p = 0.03), via reduced apnea (p = 0.02) and hypopnea indices (p = 0.04, Figure 3). There was a significant corresponding decrease in 4% oxygen desaturation index (5.5 ± 0.3 to 3.4 ± 0.2 dips/h, p = 0.014) and increase in minimum overnight oxygen saturation (84.3% ± 1.3% to 88.3% ± 0.9%, p = 0.02). Although the group mean AHI was substantially reduced with active treatment, the number of patients achieving an AHI < 15/h was not different between active (10/15) and inactive treatment (9/15).

There was a trend for an overall reduction in snoring frequency in the order of 20% with active treatment (p = 0.084), but this was not statistically significant (p = 0.084), and there was no treatment by snoring intensity interaction effect. There were also no differences in mean snore duration (inactive 1126 ± 169 vs active 1082 ± 96 msec), or bed partner snoring scale scores (3.0 ± 0.9 vs 3.0 ± 0.9, n = 7) to support reduced snoring with supine-avoidance. Bed partners’ sleep disturbance from the supine vibration alarm increased slightly but significantly from 0.7 ± 0.6 to 2.9 ± 0.8 (p = 0.01), corresponding to “a little” disturbance.

**DISCUSSION**

This study demonstrates the clinical utility of this position monitoring and supine alarm device as an accurate monitor of overnight posture and a highly effective supine-avoidance therapy, almost completely abolishing the supine posture and significantly reducing overnight AHI, albeit with persistent snoring in positional OSA patients.
The position monitoring device showed high posture classification agreement (average $\kappa = 0.95$) and supine time measurements with negligible systematic bias, with limits of agreement within 5% compared to simultaneous in-laboratory video recordings. Accurate position monitoring is an important consideration with this form of supine-avoidance treatment, given that false omissions would allow patients to sleep untreated when supine, and false detections would unnecessarily disturb patient and (potentially) bed partner sleep.

The ability to reliably and accurately record position in the home setting is a major advantage of this device over previous supine-avoidance approaches, since the ongoing evaluation of supine avoidance effectiveness and treatment adherence otherwise remain inevitably speculative. While longer-term treatment adherence and effectiveness of this and other supine-avoidance treatments remain largely unknown, the ability to continuously log overnight body position should allow for objective measures of long-term outcomes in patients with supine-predominant OSA and/or snoring, an area of sleep medicine for which data are currently lacking.

In this study, supine time during inactive treatment was in the order of 20%, consistent with 10% to 34% reported in the few previous in-home studies, and presumably reflective of typical overnight supine time in the home setting. In contrast, supine sleep time in diagnostic in-laboratory sleep studies in the same patients was significantly higher (~35%), consistent with previous reports of high in-laboratory supine sleep times (50-60%). This likely indicates a combination of verbal encouragement to sleep supine in the clinical laboratory setting, designed to examine worst-case respiration in sleep, plus restricted movement imposed by leads attached to patients. Such in-laboratory bias towards supine sleep may overestimate the therapeutic potential of supine-sleep avoidance in the more typical home environment. Consistent with previous studies of supine sleep avoidance therapies, AHI was reduced in the order of 50%, despite only 19% supine time during inactive treatment, underscoring the supine predominance of AHI in positional OSA patients. On the basis of in-laboratory recordings and by experimental design, all patients in this study were expected to achieve an AHI < 15 events/h with abolition of ~35% supine time. Although group data suggest normalization of AHI in most patients, AHI responses were highly variable, and the number of patients achieving AHI < 15/h with active treatment (10/15) was not different from that with inactive treatment (9/15), indicating substantial AHI variability, and potential confounding of patient selection by elevated supine time in the laboratory setting. Evaluation of more typical supine time in the home versus in-laboratory setting may be needed to more appropriately target supine-avoidance treatment to those patients most likely to benefit.

Snoring

Overall snoring frequency appeared to be reduced in the order of 20% or ~40 snores/h, but this and all other indices of snoring improvement, including bed partners’ satisfaction regarding snoring, showed no improvement with supine-avoidance treatment. This finding is consistent with another study that examined the impact of positional therapy on objective measurements of snoring in positional OSA patients, which also found no significant change with treatment. In a less direct evaluation, Nakano and coworkers also found no significant difference in snoring intensity or snoring time between the supine and lateral sleep postures in 51 OSA patients, although no distinction was made between patients with and without positional OSA.

Snoring was reported to be relatively mild in terms of bed partner complaints. Nevertheless, persistent snoring with supine-avoidance therapy may limit the utility of this approach in patients with supine-predominant OSA. Snoring is one of the primary complaints in the clinical presentation of OSA that frequently disrupts bed partner sleep, affecting their own quality of life and that of the snorer. Snoring may also contribute to cardiovascular risk. Effective positional therapy to reduce AHI but with only a small reduction in snoring may be of greatest benefit to patients who regularly sleep alone (5/15 patients in this study and 17% to 25% in others). However, for the majority of positional OSA patients, adjunct or alternative therapies are likely necessary to relieve snoring if snoring is a primary complaint or significant clinical concern.

The reasons for persistent snoring in positional OSA patients despite highly effective supine-avoidance and significantly reduced AHI are not entirely clear. While not statistically significant, and potentially a type II error, a 20% reduction in snoring frequency and/or small downward shift in the frequency of louder snores might be all that should be expected from a ~20% reduction in supine time in positional OSA patients selected on the basis of the supine predominance of AHI and not snoring. Unlike apneas, which arise from complete obstruction of the upper airway and relative silence until apnea resolution, hypopneas, which dominate AHI, are associated with partial airway occlusion more relevant to snoring. Although hypopneas were significantly reduced and might be expected to improve snoring indices, partial upper airway collapse and flow limitation are likely prevalent during periods of relative breathing stability between conventionally scored respiratory events. At least in this positional OSA patient group, partial upper airway collapse, flow limitation, and snoring may therefore persist via conversion of apneas and more severe hypopneas into snoring events, continued snoring between respiratory events, and potentially reduced arousals and non-flow limited breaths during transient wakefulness. This does not discount that snoring may nevertheless be substantially resolved in patients with milder upper airway dysfunction and supine-predominant snoring. Further studies are needed to separately evaluate the supine-predominance of snoring and AHI to most appropriately target supine-avoidance treatment.

Tolerability of the Position Monitor and Supine Alarm

This is the first trial to evaluate patient compliance with positional therapy objectively, rather than by patient self-report, which may be unreliable. Although one patient withdrew on the first night of active treatment, overall treatment compliance was high and was not different between active and inactive treatment, indicating that most patients tolerate active therapy in the short-term. Compliance with traditional tennis ball based supine-avoidance appears to be very poor, primarily due to its inherent discomfort. Being fastened to the chest rather than the back, this new position monitoring device may be more comfortable, and the vibratory alarm less intrusive to the bed partner poten-
tially facilitating improved longer-term compliance. A vibratory stimulus may also be more effective than an auditory alarm, given the presence of competing loud snoring and the potential for comorbid hearing loss. Although bed partners reported higher sleep disturbance during active treatment, this corresponded to only a “little” disturbance, and alarms were relatively infrequent. Nevertheless, further trials are clearly needed to investigate longer-term treatment compliance and acceptability.

Methodological Considerations

The main weakness of this study was the use of a type III device to estimate AHI using the total sleep period including any wake time as the denominator. We specifically chose the home setting most relevant to OSA and snoring and elected to estimate AHI with a simplified device that included objective snoring measurements, but without EEG, EOG, and EMG leads associated with full polysomnography, as these further restrict body movements and likely confound the study of posture and intervention effects of primary interest in this study. Polysomnography leads likely contributed to bias towards increased supine time in the laboratory setting in this and in other studies, and may overestimate the potential efficacy of positional therapies in the home environment. However, in the absence of sleep measurements it is possible that disrupted sleep with more wake time could have artifactually lowered AHI with active versus inactive treatment. Several observations strongly argue against this and the main findings are consistent with similar studies. The frequency of posture transitions was similar, and patient-reported sleep quality was not different between inactive and active treatment. In addition, alarms were infrequent and of short duration during active treatment. Of particular note is that snoring frequency did not change with active treatment, a finding consistent with another report, and difficult to reconcile on the basis of increased wake time. Finally, if AHI on active treatment exclusively represents non-supine events without changes in wake time, the addition of 19% supine time (ST) to a non-supine AHI (AHI_{NS}) of ~14 events/h with a 5.3-fold higher supine versus non-supine AHI ratio (AHI_{NS}/AHI_NS) would be expected to increase total AHI to ~25 events/h (Expected AHI_{Total} = AHI_{NS} × AHI_{Ratio} × ST + AHI_{NS} × (1-ST) = 14 × 5.3 × 0.19 + 14 × 0.81 = 25.4). This is entirely consistent with supine sleep avoidance with minimal additional wake time with active treatment. Any increase in wake time should have further reduced AHI calculated using the total sleep period as the denominator.

In summary, these data indicate this new position monitoring and supine alarm device accurately monitors sleep position and is an effective therapy for reducing AHI, but without relieving snoring, at least in patients with supine-predominant OSA. Effects on sleep quality and longer-term outcomes of effectiveness, adherence, and bed partner tolerance compared to alternative therapies remain to be established for positional OSA patients and for positional snorers without OSA.

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Positional Therapy in Supine-Predominant OSA


ACKNOWLEDGMENTS

This project was supported by a Flinders Medical Centre Foundation Grant. The authors are very grateful to Michael Gorman for providing the position monitoring devices and technical support, and to Dr. Lyn Davies for advice on snoring analysis. This work was performed at Adelaide Institute for Sleep Health, Repatriation General Hospital.

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Submitted for publication December, 2010
Submitted in final revised form January, 2011
Accepted for publication January, 2011
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DISCLOSURE STATEMENT

This was not an industry supported study. The authors have indicated no financial conflicts of interest.