Choice of Oximeter Affects Apnea-Hypopnea Index*

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Study objectives: Current Medicare guidelines include an apnea-hypopnea index (AHI) ≥ 15 events per hour, in which all hypopneas must be associated with 4% desaturation, to qualify for reimbursement for therapy with continuous positive airway pressure (CPAP). The present data demonstrate the effect of pulse oximeter differences on AHI.

Design: Prospective study, blinded analysis.

Setting: Academic sleep disorder center.

Patients: One hundred thirteen consecutive patients (84 men and 29 women) undergoing diagnostic sleep studies and being evaluated for CPAP based on the Medicare indications for reimbursement.

Interventions: Patients had two of four commonly used oximeters with signal averaging times of 4 to 6 s placed on different digits of the same hand during nocturnal polysomnography.

Measurements and results: Apneas and candidate hypopneas (amplitude reduction, > 30%) were scored from the nasal cannula airflow signal without reference to oximetry. Candidate hypopneas then were reclassified as hypopneas by each oximeter if they were associated with a 4% desaturation. Although the use of three oximeters resulted in a similar AHI (bias, < 1 event per hour), the fourth oximeter showed an overall increase in AHI of 3.7 events per hour. This caused 7 of 113 patients to have an AHI of ≥ 15 events per hour (meeting the Medicare criteria for treatment) by one oximeter but not when a different oximeter was used. More importantly, when our analysis was limited to those patients whose number of candidate hypopneas made them susceptible to the threshold value of 15 events per hour, 7 of 35 patients who did not meet the Medicare AHI standard for treatment by one oximeter were reclassified when a different oximeter was used.

Conclusion: In the present study, oximeter choice affected whether the AHI reached the critical cutoff of 15 events per hour, particularly in those with disease severity that was neither very mild nor very severe. As oximetry is not a technique that produces a generic result, there are significant limitations to basing the definition of hypopnea on a fixed percentage of desaturation in determining the eligibility for CPAP therapy.

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Key words: Medicare; obstructive sleep apnea; oximetry; sleep apnea syndromes

Abbreviations: AASM = American Academy of Sleep Medicine; AHI = apnea-hypopnea index; AI = apnea index; CPAP = continuous positive airway pressure; NPSG = nocturnal polysomnogram

The procedure currently recommended for manually scoring hypopneic respiratory events on nocturnal polysomnography depends on oxygen desaturation data obtained by pulse oximetry. One of the justifications advanced for this recommendation is that the inclusion of desaturation in the definition of hypopnea appears to increase the reliability of the respiratory index obtained. Implicit in this use is the assumption that the measurement of oxygen saturation itself is reliable. While this may be true for repeated measures with a single oximeter, there are data to suggest that different oximeters may measure different values during a single event of desaturation. Several authors have demonstrated the dependence of clinical outcomes on the brand of oximeter and its settings. For example, Brouillette et
al showed that the number of nonartifactual desaturations in a pediatric population with sleep-related breathing disorders was significantly affected by oximeter choice and signal averaging time. Recently, Davila et al demonstrated that the number of events associated with a 3% desaturation varies with changes in the set averaging time (ie, 3 to 12 s), even using a single brand of oximeter, and that this variation can affect clinical treatment. Finally, Trang et al showed that significant differences in the numbers of desaturations in a pediatric population occurred when two different oximeters with similar averaging times were used simultaneously.

The relevance of these differences in oximeter behavior has increased since Medicare has revised its definition of hypopnea. While previously the definition of hypopnea required only a reduction in the amplitude of the flow signal, under the new rules hypopnea needs to be associated with oxygen desaturation of at least 4%. By physiologic criteria alone, patients must have an apnea-hypopnea index (AHI) of ≥15 events per hour (with this definition of hypopnea) to be eligible for Medicare reimbursement of therapy with continuous positive airway pressure (CPAP). Although the guidelines allow reimbursement for CPAP therapy at a lower AHI in the presence of documented excessive daytime somnolence or comorbid disease, the major justification for the treatment of episodic obstruction of the airway remains the frequency with which it occurs (ie, the AHI). Under the present Medicare definition, this is critically linked to the desaturation produced by each hypopnea. Thus, oximetry now plays a prominent role in determining the eligibility of patients for therapy of sleep-disordered breathing, especially in patients with borderline numbers of respiratory events. This study was designed to determine whether differences between pulse oximeters in data acquisition and processing might lead to meaningful differences in the classification of patients according to current US Medicare guidelines for the reimbursement for CPAP therapy.

Materials and Methods

Subjects and Design

Nocturnal polysomnograms (NPSGs) were obtained in 113 consecutive patients (84 men and 29 women), recorded in two of four rooms at the New York University Sleep Disorders Center between August 2002 and February 2003. These patients were referred to the sleep center for suspected sleep disorders, were evaluated by a sleep physician, and subsequently were scheduled for diagnostic sleep studies. The assignment of the patients to the rooms equipped with two paired oximeters was random. One hundred eight of the 113 patients were being evaluated for obstructive sleep apnea-hypopnea syndrome. The other patients were being evaluated for insomnia or parasomnias. The mean age of the patients was 46 years (range, 10 to 80 years). The mean BMI was 31 (range, 15.0 to 55.5). The protocol was approved by the institutional review board of the New York University School of Medicine.

Techniques

All patients underwent full NPSG. Central and occipital EEGs, bilateral electrooculograms, and submental electromyograms were used to monitor sleep. An anterior tibialis electromyogram was recorded to detect leg movements. A bipolar ECG was simultaneously recorded for cardiac monitoring. Respiratory airflow was monitored using a nasal cannula/pressure transducer system (Protech; Mukilteo, WA) and an oral thermistor. Respiratory effort was monitored using rib and abdominal piezoelectric strain gauges (Sleepmate; Midlothian, VA). Body position was monitored using a sleep position sensor (Protech). All signals were acquired digitally (Sleep-Scan system; Bio-Logic; Mundelein, IL).

For each NPSG, signals from two of four commonly used pulse oximeters were recorded simultaneously (Radical; Masimo; Irvine, CA; models 395 or 295; Nellcor Puritan Bennett; Pleasanton, CA; or Sat-Trak; SensorMedics; Yorba Linda, CA). Sensors were always placed on the same hand and were assigned to random fingers. The Masimo oximeter was set to a signal averaging time of 4 s, the SensorMedics oximeter was set to a time of 6 s, and the two Nellcor oximeters had fixed intervals described as being "between 4 and 6 s." Thus, all devices were set between 4 s and 6 s for the averaging interval, which was the closest they could be matched.

Scoring of Polysomnographic Records

Sleep was scored on 30-s epochs using the standard scoring rules of Rechtschaffen and Kales. Arousal scores were scored using American Sleep Disorders Association criteria. Arousals were scored based on American Sleep Disorders Association criteria. On a separate pass, the nasal cannula airflow signal alone was used to score respiratory events without reference to oximetry. Apneas were defined as an airflow amplitude of < 10% of baseline values on both the nasal cannula airflow signal and the thermistor signal for > 10 s. “Candidate” hypopneas were identified based on a visually defined reduction in airflow of at least 30% compared with the amplitude of the baseline flow as recommended by both the American Academy of Sleep Medicine (AASM) guidelines and Medicare guidelines. Each candidate hypopnea was evaluated repeatedly and separately for the presence of 4% desaturation while using each oximeter. This was done using a computer algorithm that was triggered by the manually defined event and that inspected the saturation tracing automatically. Thus, the same respiratory events were evaluated for each oximeter, and no rater effects were involved in the repetitive calculation of AHI. Desaturation was defined as the difference between the maximum oxygen saturation occurring during the event and the nadir oxygen saturation occurring up to 30 s after the termination of the event. See Figure 1 for examples of the linking of events to desaturation. For each oximeter, candidate hypopneas found to be associated with a desaturation of ≥ 4% were considered to be valid and were tabulated with apneas to produce an AHI for that oximeter.

Subgroup Analysis

In the Medicare definition of eligibility for CPAP therapy reimbursement, oximetry only affects patients in whom the number of hypopneas determines whether the AHI is > 15.
events per hour or < 15 events per hour. Thus, if the apnea index (AI [without hypopneas included]) is > 15 events per hour, there is no effect of classifying hypopneas. Likewise, if the total event count (including apneas and all candidate hypopneas) is < 15 events per hour, no reclassification of these events can cause the AHI to reach the target of 15 events per hour. Thus, we examined separately those patients having AHI values that potentially depended on oximeter performance. This was defined by excluding from analysis all patients with an AI of > 15 events per hour and all patients with an apnea plus candidate hypopnea index of < 15 events per hour.

**Statistical Analysis**

Data on the number of respiratory events were analyzed both for the entire number of events and for the number of events when incorporated into an AHI that had been calculated for each subject by each oximeter. For each comparison between oximeters, mean biases and variability in the AHIs were calculated and further evaluated using a Bland-Altman plot. Analyses were performed on both the entire data set (113 patients) and the subgroup of patients whose data made them susceptible to reclassification (35 patients).
RESULTS

Figure 2 shows the data for the oximeter pairs used in the first 28 subjects. All pairs shown by open symbols exclude oximeter A and fall close to the line of identity. Thus, oximeters B, C, and D produced similar AHIs (bias ± 2 SDs = 0.3 events per hour ± 1.7) but appeared to be consistently different from AHIs determined using oximeter A (bias ± 2 SDs = 7.1 events per h ± 9.6). Because the purpose of this study was to demonstrate the potential for a disagreement between oximeters, we chose to pursue our comparisons using only pairs of oximeters that included oximeter A. Additional data were thus collected always pairing oximeter A with either oximeter B (53 patients) or oximeter C (48 patients). In all studies, only sections of the NPSG in which both oximeter signals were valid were used for analysis, and in no case was this < 95% of the study.

Figure 3, top, shows the difference in AHI between oximeter A and oximeters B or C in the complete data set. A systematic bias of 3.7 more events per hour was seen with oximeter A when compared with oximeters B or C. The Bland-Altman plot (Fig 3, bottom) reveals a consistent bias over the full range of AHI values, with oximeter A producing higher values than oximeters B or C. The systematic nature of this bias implies that any effect that this bias has on the classification of patients (i.e., over or under a cutoff value) will occur at any cutoff value chosen and therefore the analysis was only performed at a cutoff value of 15 events per hour.

Of the initial 113 subjects in this study, 101 patients were studied using oximeter A vs oximeter B or C. Of these 101 patients, 75 had a candidate AHI of ≥ 15 (i.e., before the hypopneas were evaluated for desaturation). Of these 75 patients, 40 had an AI of ≥ 15 events per hour, so that the count of hypopneas did not influence the AHI qualifications for Medicare reimbursement of CPAP. This left 35 subjects in whom the results of oximetry affected the AHI and could influence CPAP eligibility. Twenty of these subjects experienced such mild desaturation by all oximeters that the AHI fell below the cutoff value of 15 events per hour independently of the oximeter used. Finally, 15 subjects had an AHI that remained above the cutoff value of 15 events per hour determined using at least one oximeter.

Table 1 provides a breakdown of the number of apneas and hypopneas affected by the choice of oximeter in both the entire data set and in the subgroup of 35 patients whose diagnosis could be influenced by the results of oximetry. In the entire group, the percentage of apneas with 4% desaturation differed by oximeter (oximeter A, 81% of all apneas had 4% desaturation; oximeter B or C, 65% of all apneas had 4% desaturation). For candidate hypopneas, the percentage of events associated with 4% desaturation was lower but was affected by choice of oximeter (oximeter A, 41% of all candidate hypopneas had 4% desaturation; oximeter B or C,
25% of all candidate hypopneas had 4% desaturation). Oximeter A consistently detected a greater number of respiratory events with 4% desaturations than did oximeters B or C. Figure 4 shows the data for the individual subjects for apneas and hypopneas. Overall, the total percentage of respiratory events with 4% desaturation was determined to be 58% by oximeter A and 43% by oximeters B or C. Similar results occurred in the subgroup. Of the 10,604 candidate hypopneas in the entire group, 1,693 hypopneas (16%) were classified differently by oximeter A vs those classified by oximeter B or C, and of 5,104 candidate hypopneas in the subgroup, 778 hypopneas (15%) were classified differently by oximeter A. In the potentially oximeter-dependent subgroup, the use of oximeter B or C reduced the AHI by 40% compared to the AHI obtained when using oximeter A (from 14.5 to 10.5 events per hour). Figure 5 shows that 7 of 35 patients (20%) were eligible by AHI for Medicare reimbursement of CPAP therapy only if one used the measurements obtained with oximeter A. Eligibility for reimbursement was not affected for the remaining 28 of 35 subjects despite the change in their AHI. There were no cases in which oximeter B or C produced a higher AHI than did oximeter A.

**Discussion**

This article demonstrates that the choice of oximeter can have an important effect on the calculation of AHI when desaturation is incorporated into the definition of hypopnea. As AHI is used in the Medicare definition of eligibility for CPAP therapy reimbursement, some patients consistently had an AHI of ≥15 events per hour when evaluated using data from one commonly used oximeter, but not when using data from other oximeters. This difference occurred even when all adjustable oximeter parameters (such as averaging interval) were made as

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**Table 1—Events With 4% Desaturation by Oximeter A vs B, C, and D***

<table>
<thead>
<tr>
<th>Variables</th>
<th>All</th>
<th>Subgroup†</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Apnea</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total apneas</td>
<td>8,283</td>
<td>905</td>
</tr>
<tr>
<td>Apneas with 4% desaturation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>by oximeter A</td>
<td>6,668 (81)</td>
<td>713 (72)</td>
</tr>
<tr>
<td>Apneas with 4% desaturation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>by oximeter B/C</td>
<td>5,411 (65)</td>
<td>520 (52)</td>
</tr>
<tr>
<td><strong>Hypopnea</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total candidate hypopneas</td>
<td>10,604</td>
<td>5,104</td>
</tr>
<tr>
<td>Hypopneas with 4% desaturation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>by oximeter A</td>
<td>4,327 (41)</td>
<td>1,584 (31)</td>
</tr>
<tr>
<td>Hypopneas with 4% desaturation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>by oximeter B/C</td>
<td>2,634 (25)</td>
<td>806 (16)</td>
</tr>
<tr>
<td><strong>All events</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total events</td>
<td>18,887</td>
<td>6,099</td>
</tr>
<tr>
<td>Events with 4% desaturation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>by oximeter A</td>
<td>10,995 (58)</td>
<td>2,297 (38)</td>
</tr>
<tr>
<td>Events with 4% desaturation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>by oximeter B/C</td>
<td>8,045 (43)</td>
<td>1,326 (22)</td>
</tr>
</tbody>
</table>

*Values given as No. (%).
†Thirty-five patients whose AHIs potentially depended on oximeter performance. These patients were identified by excluding from analysis all patients with AIs of >15 events per hour and all patients with AI plus candidate hypopnea index of <15 events per hour.
nearly identical as possible. Using one oximeter resulted in an increase in the number of events (ie, apneas and hypopneas) with 4% desaturation across the full spectrum of disease severity. The greatest potential for clinical impact is in those patients with mild-moderate numbers of respiratory events.

Although we found that the magnitude of the change in AHI associated with the choice of oximeter was small (mean for the entire group of patients, 3.7 events per hour), it was consistent in direction, with one oximeter resulting in a higher value than the others. Because of this, the choice of oximeter will affect calculations that depend on desaturation in a manner that is different from the biological variability or imprecision in measurement known to affect the AHI for other reasons (eg, night-to-night variability), as it represents a systematic bias rather than a random error. To place the magnitude of this systematic effect in perspective, one can examine the effect of changing the cutoff values of the AHI used to classify subjects for oximeters B and C. For our group of 35 subjects, lowering the AHI cutoff value to 8 events per hour for oximeters B and C would result in a nearly identical classification to that of using 15 events per hour for data from oximeter A. Furthermore, the significance of a systematic change of 3 events per hour (as occurs with choosing oximeter A vs oximeter B or C) in the AHI is shown by comparing this to the increase in AHI (2.6 to 5 events per hour over the follow-up period of 5 to 8

Figure 4. Oximeter A detected more apneas and hypopneas with 4% desaturation than oximeters B and C for all patients. Events with 4% desaturation are shown on the x-axis for oximeters B and C and on the y-axis for oximeter A.

Figure 5. A comparison of the AHIs determined using oximeter B and C with that using oximeter A in the subgroup of patients with borderline respiratory events. The dashed lines indicate the Medicare cutoff value of 15 events per hour. A total of 7 of 35 patients (upper left quadrant) were classified as having an AHI of >15 events per hour by oximeter A only. Note that 20 of 35 subjects did not meet the Medicare cutoff of 15 events per hour when AHI was defined using desaturation from any oximeter.
years) that was reported in two large epidemiologic studies. Had either of these studies been performed with a change of oximeter (or algorithm) over the period of follow-up (which does not appear to have happened as far as we can establish), the entire statistically significant epidemiologic finding could have been introduced due to a change in the oximeter that was used for the monitoring.

The use of oximetry as a confirmatory signal for respiratory events has been accorded variable importance in the past 20 years. Opinion on how to define hypopnea varies, and only limited data on outcomes exist to justify any particular choice. The AASM task force published an article in 1999 on the definition of the obstructive sleep apnea-hypopnea syndrome and the measurement techniques used for respiratory events. They did not require desaturation in the definition of hypopnea when flow was markedly reduced, but supported using desaturation in the definition of hypopnea when the events were subtle. The AASM issued a position paper in 2001 that argued that only events with 4% desaturation should be counted as hypopnea, referencing an increased reproducibility and interscorer reliability. However, Whitney et al also showed that interscorer reliability is still reasonable when reducing the desaturation requirement to 2% (93% vs 99% agreement). Furthermore, Oeverland et al found that while all patients with events that had been counted using the requirement of 4% desaturations for respiratory events had significant disease, 36% of those with detectable and probably significant respiratory events (ie, a 50% reduction in flow or arousal) were missed by this criterion. Thus, there remains a considerable debate over what level of desaturation should define a “significant” respiratory event and whether oximetry alone should define such an event.

Despite this controversy, Medicare has chosen to incorporate 4% desaturation in its definition of hypopnea, making the measurement of oximetry a crucial component of defining the AHI. This requires that differences between oximeters be considered. The measurement of desaturation after a respiratory event needs to be consistent across devices to allow any “critical value” of desaturation to be used, as proposed by the Medicare guidelines. The present study shows that this is not currently the case.

Beyond the clinical relevance of the eligibility for CPAP therapy if defined by AHI alone, variability in the oximeter measurement of desaturation after a respiratory event and its use as part of the definition of the AHI may have an impact on research protocols where multicenter enrollment is required (eg, the eligibility of a subject for recruitment may be influenced by the choice of oximeter).

Previous investigators have shown that the nadir of a saturation decline is affected by the averaging time. The difference in the detection of saturation decline is greatest between signal averaging times of 3 and 12 s. In our study, there was no detectable difference between oximeter B or C and oximeter D, all of which used signal averaging times of 4 to 6 s. Thus, it is difficult to attribute the entire effect that we observed to differences in the signal averaging times of the oximeters we tested.

The method of measuring desaturation requires both the specification of an amount and a time during which desaturation will be evaluated. We chose to define the nadir of the desaturation related to a respiratory event as the lowest saturation occurring within 30 s of the end of the event. While it is possible that circulatory delays, like those seen in patients with congestive heart failure, may prolong the time to the nadir of desaturation, a review of our data showed this did not occur. In our experience, searching for a nadir in a period > 30 s after an event introduces a possible artifact as it risks capturing the desaturation resulting from a subsequent event. Although other algorithms could have been chosen to define the nadir associated with an event, we do not think that the specific algorithm we used contributed to our result. As shown in Figure 1 and consistently in our data, there was little difference in the time to nadir between oximeters, only in the value achieved. In addition to defining the choice of a nadir, the desaturation resulting from an event depends on the chosen starting value; we chose to make this the highest saturation recorded during the respiratory event defined by the airflow signal. This peak value may be higher than a true baseline saturation, due to the “overshoot” following a previous event. We have found little discussion of the issue of what the “baseline” needs to be to calculate the desaturation produced by an event and no guidance as to how to define this in the absence of a period of stable breathing close to the time of an event. However, we think that in our data the choice of baseline saturation that we used (maximum value within the duration of the event) is unlikely to have been the sole cause of the differences we observed between oximeters, although it may have magnified the effect.

Other than user parameters (such as the averaging interval), there are several possible explanations for differences between the effect of different oximeters on the AHI. These include patient factors, proprietary hardware issues, differences in signal-processing algorithms, and interactions be-
tween these factors. The type of probe (eg, finger, ear, and toe) also may have an effect\textsuperscript{4,17} but did not affect our data as all oximeters were used with a finger probe.

Patient-related factors that might have artifici-
tually created our results include a difference in
blood flow to the two fingers used by the different
oximeters or a difference in light transmission
through fingers (eg, from skin thickness or anat-
omy). To address these issues, we did a substudy
using two identical oximeters on different fingers
in 10 patients and could demonstrate no differ-
ences in either the actual superimposed oximetry
signals or the resultant AHI\textsuperscript{18} calculated (data not
shown). Furthermore, there is a study in abstract
form that did not find any effect on oximetry of
changing the hand on which an oximeter probe
was placed.\textsuperscript{18}

Instrumentation and signal-processing algorithm
issues clearly vary between devices. As far as we can
determine from the available documentation, all
devices use two wavelengths of light and calculate a
similar ratio of their transmission intensities to derive
oxygen saturation. However, techniques for noise
cancellation, filtering, and pulse-to-pulse value com-
binations differ in proprietary ways that may underlie
the apparent different sensitivities that these devices
have to changes in saturation, even if the steady-state
saturations that are measured agree. The problem is
not just one of calibration itself but may lie in the
response characteristics of an oximeter when given a
changing saturation signal.

Our data also suggest to us that there may be an
interaction among several of the above factors,
specifically patient physiology and oximeter algo-
rithm. We suspect that this interaction resulted in
greater differences between the two oximeter
readings in specific subjects. Three of the seven
patients who were eligible for Medicare-reim-
bursed CPAP therapy determined by oximeter A
alone had marked sinus arrhythmia. West et al\textsuperscript{19}
have shown that a reduced heart rate leads to
delays in detecting changes in oxygen saturation.
Since pulse oximetry requires data that are linked
to heart rate, pulse variability might have contrib-
uted to differences in oximeter measurement in a
dynamic setting, either through modifying the
value of saturation assigned to a single beat, or
because of internal averaging or noise suppression
algorithms. Our available data did not allow this
issue to be fully addressed, and further evaluation
of this finding is needed.

In summary, our data show that in a group of
patients with sleep-disordered breathing the use of
different oximeters results in the calculation of dif-
f erent AHI and this may affect the patients’ eligibil-
ity for CPAP therapy under current Medicare rules.
However, our data do not allow one to choose the
AHI determined using one oximeter as being more
correct than that determined using the other oxime-
ters, as there is no reference standard by which to
judge the desaturations seen using the different
instruments. We particularly caution against the
interpretation that one oximeter is necessarily the
best at detecting the physiologic changes. Our data
merely suggest that current oximeters differ enough
that the 4% desaturation criterion currently required
by Medicare may not be an effective operational
definition of the conceptual criterion it is intended to
represent.

\section*{References}
\begin{thebibliography}{18}
\bibitem{1} Meoli AL, Casey KB, Clark RW, et al. Hypopnea in sleep-
disordered breathing in adults. Sleep 2001; 24:469–470
\bibitem{2} Department of Health and Human Services Centers for
Medicare and Medicaid Services. Medicare coverage issues
manual 12–26-2001; transmittal 150, section 60–17. Wash-
ington, DC: Department of Health and Human Services
\bibitem{3} Tsai WH, Flemons WW, Whitelaw WA, et al. A compari-
sion of apnea-hypopnea indices derived from different
definitions of hypopnea. Am J Respir Crit Care Med 1999;
159:43–48
\bibitem{4} Young D, Jewkes C, Spittal M, et al. Response time of pulse
oximeters assessed using acute decompression. Anesth Analg
\bibitem{5} van Oostrom JH, Melker RJ. Comparative testing of pulse
\bibitem{6} Brouillette RT, Lavergne J, Leimanis A, et al. Differences in
pulse oximetry technology can affect detection of sleep-disor-
\bibitem{7} Davila DG, Richards KC, Marshall BL, et al. Oximeter’s
acquisition parameter influences the profile of respiratory
disturbances. Sleep 2003; 26:91–95
\bibitem{8} Trang H, Bourreghda S, Leske V. Sleep desaturation: compar-
\bibitem{9} Rechtschaffen A, Kales A. A manual of standardized terminol-
ogy, techniques, and scoring system for sleep states of
Office, 1968; 204
\bibitem{10} Sleep Disorders Atlas Task Force. EEG arousals: scoring
\bibitem{11} Tishler PV, Larkin EK, Schluchter MD, et al. Incidence of
sleep-disordered breathing in an urban adult population: the
relative importance of risk factors in the development of
sleep-disordered breathing. JAMA 2003; 289:2320–2327
\bibitem{12} Young T, Palta M, Dempsey J, et al. The occurrence of
328:1230–1235
\bibitem{13} American Academy of Sleep Medicine. Sleep-related breath-
ing disorders in adults: recommendations for syndrome def-
inition and measurement techniques in clinical research.
Sleep 1999; 22:667–689
\bibitem{14} Whitney CW, Gottlieb DJ, Redline S, et al. Reliability of
scoring respiratory disturbance indices and sleep staging.
Sleep 1998; 21:749–757
\bibitem{15} Oeverland B, Skatvedt O, Kvaerner KJ, et al. Pulse oximetry:
sufficient to diagnose severe sleep apnea. Sleep Med 2002;
3:133–138
\end{thebibliography}

