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Nikolaus Netzer, Arn H. Eliasson, Cordula Netzer and David A. Kristo

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Overnight Pulse Oximetry for Sleep-Disordered Breathing in Adults*

A Review

Nikolaus Netzer, MD; Arn H. Eliasson, MD, FCCP; Cordula Netzer, MD; David A. Kristo, MD, FCCP

Pulse oximetry is a well-established tool routinely used in many settings of modern medicine to determine a patient's arterial oxygen saturation and heart rate. The decreasing size of pulse oximeters over recent years has broadened their spectrum of use. For diagnosis and treatment of sleep-disordered breathing, overnight pulse oximetry helps determine the severity of disease and is used as an economical means to detect sleep apnea. In this article, we outline the clinical utility and economical benefit of overnight pulse oximetry in sleep and breathing disorders in adults and highlight the controversies regarding its limitations as presented in published studies.

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Key words: COPD; desaturation; pulse oximetry; sleep; sleep apnea syndromes; upper airway resistance syndrome

Abbreviations: AHI = apnea-hypopnea index; NPSG = nocturnal polysomnography; ODI = oxygen desaturation index; OSA = obstructive sleep apnea; RDI = respiratory disturbance index; Sa_{O_2} = arterial oxygen saturation

Pulse oximetry is one of the most widely used tools to determine a patient's cardiorespiratory stability. Over the last 40 years, it has often replaced arterial blood gas analysis because the arterial oxygen saturation (Sa_{O_2}) frequently gives a sufficient amount of information about a person's respiratory patterns.[1,2] In the early years of pulmonary medicine, pulse oximetry was the key means to identify patients with pickwickian syndrome or severe sleep apnea syndrome by detecting the saw-tooth pattern on oxygen desaturation waveforms (waveform derived as a plot of Sa_{O_2} vs time).[3] Very few clinics had access to other devices such as pneumotachographs, esophageal catheters, and respiratory effort belts. With the broader use of nocturnal polysomnography (NPSG) in sleep medicine, pulse oximetry has kept its key role in the interpretation of NPSG but has lost its status as the sole objective diagnostic parameter for respiratory disturbance events.[4,5]

In the past 5 years, debate has centered on the effectiveness of overnight pulse oximetry as a screening tool to identify patients with sleep-disordered breathing from the larger group of patients with simple snoring and those with excessive daytime sleepiness from other causes.[6–8] This controversial discussion has arisen from needs to reduce the cost for diagnostic procedures in sleep disorders while technologic advances have made pulse oximeters handier, cheaper, and more reliable.[9,10]

Using keywords, we found 1,558 articles listed in the PubMed database over the last 5 years that are related to pulse oximetry. One individual reviewed these publications by evaluating the abstracts. Screening these publications for relevance revealed that 79 of these articles contained useful information to outline the actual role of overnight pulse oximetry in the diagnosis and treatment of sleep-disordered breathing. We reviewed the full text of these 79 articles. Eleven key articles from previous years were also reviewed for important background information. All articles were studied for strategies to use in the interpretation of data gathered during overnight pulse oximetry.

Interpretation and Technical Aspects of Overnight Pulse Oximetry

Common sense dictates that pulse oximetry can be a useful tool only if the user knows how to interpret the oximetry data. In a survey performed in 1997 with 203 respondents, only 36% of intensive care nurses, 4% of medical technicians, and 50% of

*From the Pulmonary and Critical Care Medicine Service, Department of Medicine, Walter Reed Army Medical Center, Washington DC.

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Correspondence to: Arn H. Eliasson, MD, FCCP, Pulmonary and Critical Care Medicine Service, Department of Medicine, Walter Reed Army Medical Center, Building 2, Ward 77, 6900 Georgia Ave, Washington, DC 20307; e-mail: aheliasson@aol.com

anesthesia technicians believed that they had received adequate training in interpreting pulse oximetry data. Only 68.5% correctly stated what pulse oximeters actually measure.[11] These survey results were found despite the fact that practice guidelines for pulse oximetry were published in 1991 by the American Association for Respiratory Care.[12]

The interpretation skills of overnight pulse oximetry start with a knowledge of normal oxygen saturation values during sleep. In a key validation study published in 1996 in *CHEST*, the authors noted a normal overnight mean (the so-called Sat 50) Sao_2 of 96.5% (± 1.5%) in 350 healthy subjects.[13] Sao_2 decreased slightly with increasing age, the values ranging from 96.8% in the age group of 1- to 10-year-old patients to 95.1% in the age group > 60 years (Table 1). Ethnicity, gender, and weight did not significantly influence normal values. In a group of 21 asthmatic patients, Sao_2 did not decrease significantly, but significantly lower values were found in a group of 25 patients with obstructive sleep apnea (OSA) where mean "lowest Sao_2" of 65.9% (± 22.6%) was measured vs 90.4% (± 3.1%) in normal subjects and 89.0% (± 5.3%) in asthmatic subjects.

Normal Sao_2 values at night differ with altitude of course. In six healthy subjects, normal mean Sao_2 values of 97.3%, 83.0%, and 71.0% were measured respectively at 500 m, 4,200 m, and 6,400 m (three subjects) of altitude during sleep.[14]

The high-quality, portable pulse oximeters of today deliver accurate values of Sao_2 that differ from arterial blood gas probes by < 0.5% (± 1.8%); there are no significant differences if probes measure at the fingertips or ears.[15] Due to the fact that measurements are taken by performing a "running average" with a moving window that varies from 1 to 15 s in length, the speed of response to onset of oxygen breathing is on average 9 to 10 s with finger and ear probes.[15,16] However, the speed of response is markedly slower with toe probes.[15] The default settings for the averaging time are different for various pulse oximeters, and must be known by the user. For overnight pulse oximetry in sleep medicine, it is important that the oximeter be set to the shortest time interval for measurement.[16] The typical cyclical drop in Sao_2 in patients with OSA lags 45 to 60 s behind a respiratory event and should be accurately detected at this measurement speed.[15] Due to movements during sleep, the artifact rate is higher in overnight pulse oximetry, compared to daytime Sao_2 measurements. With measurement intervals set on high speed, artifacts are recognized by most pulse oximeters due to a missing pulse signal, although this is controversial. In a validation study of three different oximeters, Barker and Shah[17] revealed that one oximeter displayed the Sao_2 value within 7% of control only 76% of the time after patient motion; another oximeter did so 87% of the time; and only one of the three oximeters did so 99% of the time. Another study[18] also showed that pulse oximeters detect only 18% (± 11%) of all artifacts in infants.

There is no universally accepted definition of an oxygen desaturation in sleep-disordered breathing. However, in most publications, an oxygen desaturation is defined as a decrease of ≥ 4% from baseline Sao_2.[6,19–22] Rauscher et al[19] tested the detection of apneas and hypopneas by searching for rapid resaturations of ≥ 3% Sao_2 within 10 s at the end of a respiratory event vs detecting a decrease ≥ 4% Sao_2 in a 40-s interval. They found the resaturation to be a more accurate sign of respiratory events than the actual desaturation.[19] Taha et al[23] defined an oxygen desaturation as a fall in oxyhemoglobin saturation of ≥ 2% if the rate of descent was > 0.1%/s but < 4%/s.

Whereas one definition of an oxygen desaturation is in common use, no such uniform definition exists for a normal or abnormal oxygen desaturation index (ODI; oxygen desaturations per hour of sleep). There are generally three cutoff points for an abnormal ODI that appear to mirror the definition of an

Table 1—*Descriptive Statistics of Normal Oxygen Values in Different Age Groups

Age Group, yr	Patients, No.	Low Sat (SD), %	Sat 10 (SD), %	Sat 50 (SD), %
All ages	350	90.4 (3.1)	94.7 (1.6)	96.5 (1.5)
≤ 1	30	90.7 (2.6)	95.2 (1.0)	96.4 (1.2)
1–10	180	90.1 (3.6)	95.1 (1.5)	96.8 (1.4)
10–20	46	90.4 (2.7)	94.5 (1.8)	96.5 (1.6)
20–30	12	92.0 (3.4)	94.8 (1.1)	96.3 (1.0)
30–40	24	91.5 (2.2)	94.8 (1.3)	96.3 (1.1)
40–50	25	91.1 (2.0)	94.2 (1.7)	96.0 (1.3)
50–60	16	90.4 (1.9)	93.6 (1.6)	95.8 (1.7)
≥ 60	17	89.3 (2.8)	92.8 (2.3)	95.1 (2.0)

*Low Sat = lowest oxygen saturation during the night; Sat 10 = saturation below which the patient spent 10% of the time; Sat 50 = median saturation during the night; data from Gries and Brooks.[13]

abnormal apnea-hypopnea index (AHI; apneas and hypopneas per hour of sleep) for that study. The threshold for an abnormal ODI is either ≥ 5 desaturations per hour,[6,20,21,24,25] ≥ 10 desaturations per hour,[7,8,22] or ≥ 15 desaturations per hour.[26–30] There is little evidence of one definition having greater validity than the others.

To properly interpret overnight oximetry data, an understanding of the Sa_{O_2} vs time waveform morphologies is essential.[31,32] The waveforms can help discriminate between obstructive apneas and hypopneas, as well as between obstructive and central apneas, and can give evidence of Cheyne-Stokes respiration.[33] While obstructive apneas show the typical saw-tooth waveform with a rapid increase in Sa_{O_2} during or after the arousal, the "teeth" are not as sharp in hypopneas and are sometimes completely missing in central apneas (Fig 1, 2). Central apneas can act as the great masquerader of oximetry waveforms. Especially when part of Cheyne-Stokes respiration, they show a more regular symmetrical wave due to the more regular breathing pattern, compared to those of obstructive apneas. However, single central apneas not in conjunction with Cheyne-Stokes respiration can also have a saw-tooth configuration in the oximetry waveform.

The length of the desaturation waveform can also help to distinguish desaturations due to COPD from desaturations caused by obstructive apneas or hypopneas. The desaturations secondary to COPD tend to last much longer and have a much lesser degree of slope in the waveform.[34,35] This is also important for the diagnosis of OSA in the presence of COPD, the so-called overlap syndrome.

The automatic interpretation of the Sa_{O_2} waveform is often a part of modern NPSG and portable oximetry software. However, the programs are not yet able to replace interpretation by hand. The same may be true for the interpretation of heart rate variability, but here the experience with automatic analysis is much greater because of the long experience with automatic analysis in ECG-Holter systems. The heart rate slows during and at the end of an upper-airway obstruction (apnea or hypopnea) due to a reflex bradycardia with high negative intrathoracic pressure (involuntary Mueller maneuver). There is a rapid increase in the pulse with rebreathing during the arousal. This strategy does not apply to the interpretation of central apneas, because there is no negative intrathoracic pressure during a central apnea. Adult criteria for the interpretation of overnight pulse oximetry may not be valid for the evaluation of sleep-disordered breathing in children and adolescents due to different patterns of normal respiration and gas exchange.[36,37]

Sensitivity and Specificity of Overnight Pulse Oximetry in Screening for Sleep-Disordered Breathing

Over the last decade, a debate in the literature has questioned whether or not pulse oximetry could effectively screen patients for sleep-disordered

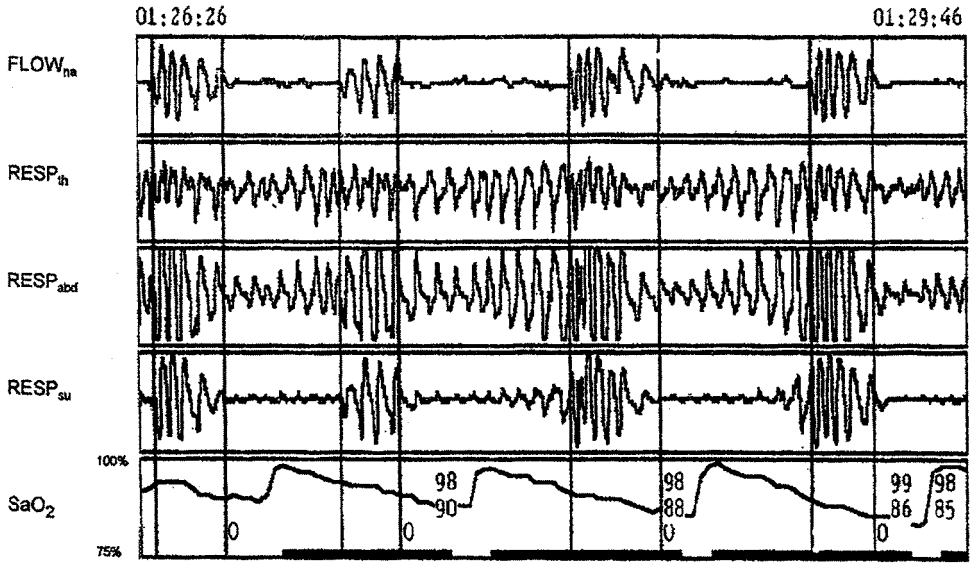

Figure 1. Respiratory patterns during a 3-min time period for a patient with OSA syndrome showing obstructive apneas with typical saw-tooth morphology of the pulse oximetry curve. $Flow_{na}$ = nasal and oral airflow; $Resp_{th}$ = thoracic respiratory effort; $Resp_{abd}$ = abdominal respiratory effort; $Resp_{sum}$ = sum signal of thoracic and abdominal respiratory effort; O = obstructive apnea.

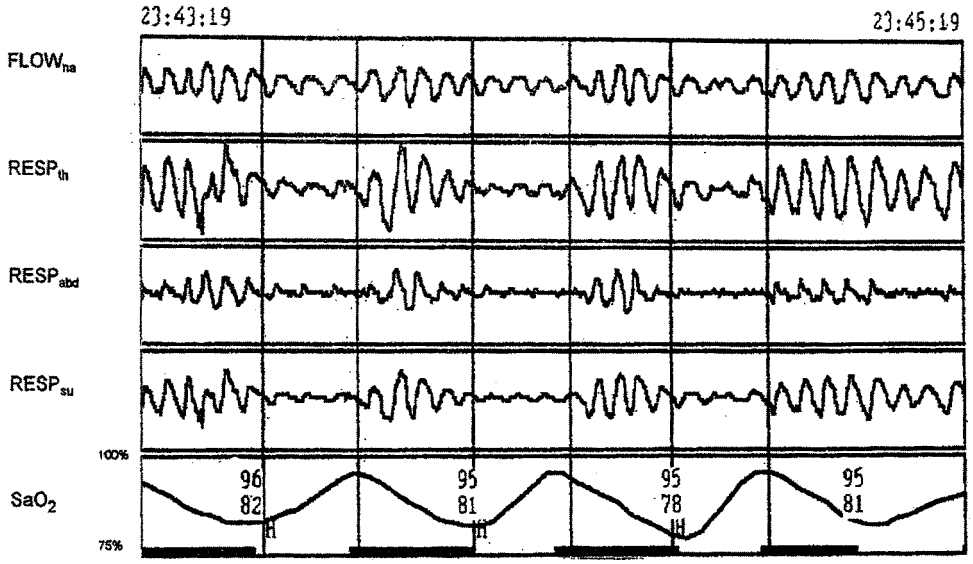

FIGURE 2. Respiratory patterns during a 3-min time period for a patient with OSA syndrome showing obstructive hypopneas with a more regular up-and-down waveform of the pulse oximetry curve. H = hypopnea; see Figure 1 legend for definition of abbreviations.

breathing and possibly replace NPSG in many patients. Deegan and McNicholas[28] reported 250 consecutive Irish patients who underwent NPSG. In one third of these patients, patient history and pulse oximetry data would have been sufficient to make a diagnosis. In the other two thirds, a final diagnosis could be established only by NPSG.[28]

Other studies[38,39] are more encouraging about the use of overnight oximetry as a less expensive substitute for NPSG. In 1991, Cooper et al[25] studied a group of 41 patients with suspected sleep apnea and found that the sensitivity and specificity of pulse oximetry for identifying OSA was dependent on the AHI. For patients with an AHI $\geq$ 25 events per hour, the sensitivity was 100% and the specificity 95%. For patients with AHI $\geq$ 15 events per hour, these values decreased to 75% and 86%; for patients with AHI $\geq$ 5 events per hour, to 60% and 80%, respectively. The authors concluded that pulse oximetry is an effective tool for screening patients with moderate-to-severe sleep apnea. In the same year, Williams et al[7] reported a sensitivity of 78% and specificity of 100% when screening patients with an AHI $\geq$ 10 events per hour. In a study of 116 subjects, Rauscher et al[8] reported a sensitivity of 94% and a specificity of 45% for detecting OSA with an AHI $\geq$ 10 events per hour and 95% and 45% with an AHI $\geq$ 20 events per hour, respectively. Within the past 5 years, 11 articles on this topic were published, revealing a broad range of sensitivity and specificity values for pulse oximetry as a screening tool for sleep-disordered breathing.[26,27,29,30,40–46] The values for sensitivity range from 31 to 98% and for specificity from 41 to 100% (Table 2). These validation studies deserve critical comment. Some authors used methods of pulse oximetry that are not yet available to the general public. The utility of these new technologies may not be borne out with further investigation.[30] Other authors looked only at a limited patient group in the spectrum of severity of OSA. Findings from these studies may not be applicable to OSA patients with different levels of severity from those studied.

Overnight Pulse Oximetry in Combination With Other Parameters

Pulse oximetry is the most important parameter for identifying sleep-disordered breathing in many portable multichannel sleep apnea screening devices. The next most commonly measured parameters are snoring sound via microphone,[47,48] oronasal airflow measured via thermistor or nasal pressure cannula,[49–51] and ECG recording.[52] One author[52] argues that the full ECG provides information about the comorbidity of cardiovascular disease in sleep apnea better than pulse oximetry alone. In 1998, Lojander et al[53] described pulse oximetry in combination with a bed sensitive to static charge in order to measure body movements. However, if compared to the sensitivity and specificity values of pulse oximetry alone as a screening tool, the combination of other parameters with pulse oximetry does not offer much improvement.[49,51]

Another interesting strategy may be the combina-

Table 2—*Sensitivity and Specificity of Pulse Oximetry When Used To Screen for OSA Compared to NPSG: Results From 11 Published Studies

Author/Year	Study Population, No.	AHI/ODI Cutoff Point	Screening Specificity, %	Screening Sensitivity, %
Ryan et al[27]/1995	69	≥ 15	100	31
Levy et al[29]/1996	301	≥ 15	94	77
Rodriguez Gonzalez-Moro et al[40]/1996	96	NA	69	91
Schafer et al[41]/1997	114	NA	41 (92†)	94
Lacassagne et al[44]/1997	329	≥ 15	57.8	89
Sano et al[26]/1998	40	≥ 15	83.3	73.5
Olson et al[45]/1999	113	≥ 15	70	88
Golpe et al[43]/1999	116	≥ 10	97	84
Brouillete et al[46]/2000	349	NA	96	58
Nuber et al[42]/2000	70	NA	77.8	85.2–91.8‡
Vazquez et al[30]/2000	246	≥ 15	88	98

*NA = not available.
†Combined with questionnaire.
‡Higher sensitivity after rereading unclear desaturations.

tion of a validated questionnaire with overnight pulse oximetry. Chervin and Aldrich[54] state that the addition of the Epworth Sleepiness Scale alone does not appear to be helpful for the diagnosis of sleep-disordered breathing compared to NPSG and oximetry. However, there is a report[42] that the combination of a questionnaire and pulse oximetry doubles the specificity of oximetry as a screening tool for sleep apnea. This approach invites further validation.

Other Applications

Overnight pulse oximetry is frequently being used to assess the response to the surgical interventions for OSA as well as the effectiveness of therapy with continuous positive airway pressure. However, this clinical practice is not established in the literature, and validation of its use for this indication is lacking. Continuous pulse oximetry is also in frequent use in a variety of other settings, including preoperative evaluations, the operating room, postanesthesia recovery suites, ICUs, and stroke units.[55–61] This has led to an increasing awareness of sleep-disordered breathing as a comorbidity in patients being treated for other diagnoses or as a symptom of other diseases, such as stroke,[60,61] neuromuscular diseases,[62,63] and cardiovascular diseases.[64–66] As continuous pulse oximetry has become more accessible and more widely employed, physicians in specialties other than sleep medicine have become accustomed to recognizing oximetry waveforms suggestive of sleep apneas. These coincidental observations are frequently leading to patient referrals for definitive diagnosis and treatment of OSA.

Attempts have been made to capitalize on the continuous measurement of heart rate provided by pulse oximetry. Computerized analysis of the heart rate variability makes it possible to detect sleep apnea syndrome via the pulse signal.[67,68] Using this method, Keyl et al[68] report a sensitivity of 90% and a specificity of 77% for the detection of OSA in patients with daytime sleepiness. Some authors[69,70] believe that the interpretation of heart rate changes delivers a better pulse oximetry indicator for OSA than interpretation of the Sa_{O_2} signal, especially if it is done using automation. Another aspect used by some investigators is the waveform generated by the displacement of capillary walls by the intermittent pulse signal or so-called "plethysmographic" pulse. Shamir et al[71] and Schnall et al[72] describe that apneas lead to transient peripheral vasoconstriction. Schnall et al[72] conclude in their publication that pulsatile finger blood flow patterns can be clearly diagnostic of OSA and other conditions of sleep-disordered breathing.

Future developments with pulse oximetry will undoubtedly show marked improvements in artifact detection. Signal delivery will become more reliable and less vulnerable to interruptions by movement using the same technique employed in portable compact disk players to memorize signals (using new paradigms for oximeter signal processing).[17,73] The spectral analysis of oximetry data facilitates precise analysis with a reported sensitivity of 94% and specificity of 65% for OSA.[74] Photon density wave differentials and noninvasive optical oximetry with a living tissue oximeter may allow monitoring of regional tissue oxygenation in the heart or brain in conjunction with sleep apnea.[75,76] Another promising innovation is the improvement of adhesive probes that would allow for pulse oximetry in sites other than digits and ears.[77]

Limitations

While pulse oximetry is a useful clinical tool in sleep medicine, it suffers from major limitations due to the nature of the parameters that are monitored.[78–80] Limitations result from problems with blood flow, hemoglobin, or a lack of change in oxygen saturation.

Pulse oximetry relies on pulsatile blood flow for its measurements and is vulnerable to the effects of poor peripheral arterial blood flow. Therefore, body movements, vasoconstriction, and hypotension can cause artifacts through an interruption of the pulse signal. In sleep medicine, movement artifacts are common since patients often have fragmented sleep with a lot of body movements. Oximeters do not always detect movement artifacts, and this would tend to overestimate desaturations.[17]

Changes in the hemoglobin structure and quantity will also cause artificially high (in cases of methemoglobinemia and carboxyhemoglobinemia) or low readings (anemia) that are not due to respiratory disturbances.[78] Anemia would also tend to be misread by overestimating respiratory-caused desaturations. Tissue optics in very obese patients can cause the same effect.[81] Herer et al[82] found that oximetric data do not reliably predict OSA in obese patients. Mower et al[83] studied SaO_2 data from 12,096 patients at the UCLA Emergency Medicine Center. They believed that no conclusions could be drawn from the data due to high variations in respiratory rates among the patients and the artifacts that this caused.[83]

Another type of limitation of pulse oximetry is due to the inability of technology to detect other forms of sleep-disordered breathing where oxygen desaturation does not occur. These disorders include upper airway resistance syndrome or pure central sleep apnea in diseases like Ondine's curse. A normal minute ventilation in upper airway resistance syndrome maintains normal oxygen levels, but high respiratory workload causes arousals and daytime sleepiness. Understandably, pulse oximetry would appear normal in this setting.[24,84]

The limitations of pulse oximetry might not have much impact in the sleep laboratory, where several other parameters are monitored to aid in the interpretation of the study. However, these limitations become of major importance in the application of pulse oximetry alone as a screening tool for breathing-disordered sleep.

Cost-effectiveness

Bennet and Kinnear[10] call pulse oximetry "sleep on the cheap" in their 1999 editorial because it generates a lot of data at a very low cost. Perhaps the only competitor for cost-effectiveness is a structured and validated questionnaire. In other fields of medicine, the cost-effectiveness of pulse oximetry is more or less accepted.[85] In sleep medicine, the clinical value of overnight pulse oximetry alone for the diagnosis of sleep apnea syndrome has become controversial since NPSG has been widely available. However, the recent advent of managed care and pressures for cost reduction have stimulated a variety of investigations[22,86–89] that substantiate the economies of overnight pulse oximetry at home as a screening test for sleep-disordered breathing. Epstein and Dorlac[22] state that initial diagnosis with home-based overnight pulse oximetry would save $4,290 per 100 patients vs diagnostic NPSG or split-night studies. However, they showed that oximetry is not very sensitive for patients with mild sleep apnea.[22] Chiner et al[89] subsequently analyzed how many NPSGs could be saved by overnight pulse oximetry in the initial diagnosis for patients with differing severity of OSA. They concluded that in 275 suspected cases, of which 216 patients were confirmed to have OSA, pulse oximetry could have saved 140 polysomnographic studies in the group with a respiratory disturbance index (RDI) ≥ 5, 119 in the group with an RDI ≥ 10, and 10 in the group with an RDI ≥ 15.[89] Because of its low cost, there is almost no alternative to overnight pulse oximetry as a sole diagnostic tool, except for patient history and questionnaires.[90] If the pressure for cost reduction

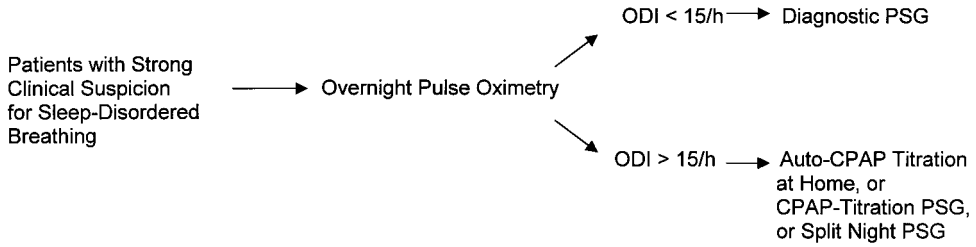

Figure 3. Flow diagram for the use of overnight pulse oximetry to screen for sleep-disordered breathing with a descending progressive therapeutic approach for patients with an ODI > 15 desaturations per hour. PSG = polysomnography; CPAP = continuous positive airway pressure.

continues, proposals may arise to perform pulse oximetry with reusable finger and ear probes, or validated questionnaires may become the sole "procedure" of first choice in the diagnostic evaluation of sleep disorders.[91]

Conclusion

Overnight pulse oximetry is a very useful tool for the diagnosis of sleep-disordered breathing. Authoritatively establishing a final diagnosis is very difficult without oximetry data. As a screening tool for the diagnosis of OSA, pulse oximetry is cost-effective and shows substantial accuracy. Sensitivity and specificity remain controversial, however, and deserve further clarification through controlled studies. Technical limitations, limited user knowledge, and the lack of consensus on interpretation of data all play a role in diminishing the value of pulse oximetry as a diagnostic tool. The authors suggest a flow diagram to delineate the clinical use of overnight pulse oximetry as a screening tool for sleep-disordered breathing (Fig 3). The establishment of clinical practice guidelines that outline technical requirements and strategies for interpretation, along with improved automated analysis, may improve the clinical utility of pulse oximetry in the future.

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Nikolaus Netzer, Arn H. Eliasson, Cordula Netzer and David A. Kristo

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