The Use of Cutaneous Oximetry in the Prescription of Long-term Oxygen Therapy*

Brian W Carlin, M.D.;† Jack L. Clausen, M.D.; and Andrew L. Ries, M.D., F.C.C.P.

Using current Medicare guidelines for the prescription of long-term oxygen therapy, we studied the impact on decision-making of substituting cutaneous oxyhemoglobin saturation measurements (SaO₂) for direct arterial oxygen tension measurements (PaO₂). Fifty-five patients with chronic lung disease and resting hypoxemia were studied. More than 80 percent of patients with a resting PaO₂ of 7.33 kPa (55 mm Hg) or less had a cutaneous oximetry SaO₂ greater than 85 percent. These patients would not have met the guidelines for long-term oxygen therapy if the cutaneous oximetry measurements were used instead of direct PaO₂ measurements. Substituting a threshold criterion of 88 percent instead of 85 percent resulted in fewer patients being denied oxygen therapy but also included patients with PaO₂ values greater than 7.33 kPa (55 mm Hg). We conclude that cutaneous oximetry cannot be substituted equivalently for PaO₂ measurements in prescribing long-term oxygen therapy.

The beneficial effects of long-term oxygen therapy (LTOT) have been documented by two landmark studies.1,2 The high cost of this therapy, often more than $300 per month, has prompted third party carriers to establish strict prerequisites for reimbursement. Medicare guidelines for qualifying levels of hypoxemia include either an arterial oxygen tension (PaO₂) less than or equal to 7.33 kPa (55 mm Hg) or an arterial oxygen saturation (SaO₂) less than or equal to 85 percent measured either directly on arterial blood using spectrophotometry or indirectly using cutaneous oximetry.3

Two considerations led us to question the validity of the Medicare guideline for SaO₂ measured by cutaneous oximetry. First, under normal circumstances (pH = 7.40; arterial carbon dioxide tension [PaCO₂] = 40; temperature, 37°C), a PaO₂ of 7.33 kPa (55 mm Hg) should be reflected by an SaO₂ of 88 percent, not 85 percent. Second, although previous studies have demonstrated that the inaccuracy (95 percent confidence limits) of ear oximetry measurements is ±4-5 percent compared to SaO₂ measurements by CO-oximetry,4 the study defining the impact of using cutaneous oximetry as the sole basis for the prescription of LTOT has not been performed. We conducted this study to examine this issue.

*From the Department of Medicine, Division of Pulmonary and Critical Care Medicine, University of California School of Medicine, San Diego.
†Fellow American Lung Association of California.
Research supported in part by NIH SCOR grant No. 510035-23906, NIH Pulmonary Training grant HL-07702, and NIH grant RR00827 from the Division of Research Resources for Clinical Research Centers.
Manuscript received October 21; revision accepted January 20.
Reprint requests: Dr Ries, UCSD Medical Center, 225 Dickinson Street, San Diego 92103

Materials and Methods

Patient Selection

We examined the data obtained from all patients referred to our pulmonary function laboratory from May 1984 to November 1985 for evaluation of arterial blood gas (ABG) changes during exercise. All patients with a resting PaO₂ of 8.65 kPa (65 mm Hg) or less and concomitant measurement of SaO₂ by cutaneous oximetry were included. If a patient had more than one study conducted during this period, only results from the first study were included.

Blood Gas and Ear Oximetry Measurements

In each study ABG samples were drawn (within 5 s) into heparinized 3-ml syringes, immediately placed into ice, and analyzed as soon as possible. Samples were collected with the patient in the upright seated position breathing room air for at least ten minutes. They were analyzed in duplicate for pH, carbon dioxide tension (PaCO₂), and oxygen tension (PaO₂) using IL-813 and IL-513 (Instrumentation Laboratories, Lexington, MA) blood gas analyzers. Routine quality control of these measures includes the use of tonometered blood at least once every eight hours and one-point gas calibration checks before each determination.5 In our laboratory, the routine quality control data from using blood tonometered to a Po₂ of 6.67 kPa (50 mm Hg) indicate that the inaccuracy (observed minus target value) of single measurements of Po₂ is 0.093 ± 0.067 kPa (0.7 ± 0.5 mm Hg) (mean ± 1.0 SD). The SaO₂ carboxyhemoglobin (COHb), and methemoglobin (MetHb) were measured using a four-wavelength spectrophotometer (IL-282 CO-oximeter, Instrumentation Laboratories). The absolute inaccuracy of measurements of SaO₂ by spectrophotometry (IL-282) is difficult to define because of the absence of primary reference standards. Daily quality control checks using commercially prepared bovine hemoglobin solutions (IL-282 CO-oximeter Control, Instrumentation Laboratories) were within published assay values. The average imprecision (observed minus mean) of our repeated SaO₂ measurements using blood tonometered to an SaO₂ of approximately 85 percent is 0.5 percent ± 0.3 percent (mean ± 1.0 SD).

Ear oximetry readings from either a Hewlett-Packard HP 47201A, a Biox IIA (pulse-type), or both used concomitantly, were recorded by a separate observer simultaneously with the withdrawal of each
ABG sample. Both instruments were used following the manufacturer's recommendations. Prior to attaching the Hewlett-Packard ear probe, the ear pinna was massaged with an alcohol pad for 30 to 60 s. Ear perfusion was then checked with a Hewlett-Packard perfusion indicator to ensure adequate perfusion (indicator ≤ 2 percent error). The ear probe was then attached and secured with the cover provided and by wrapping the head with a gauze bandage. A similar procedure was used to attach the Biox ear oximeter probe. The probe was attached to the pinna with either the manufacturer's retainer or by tape and a headband. The instrument was used in the "fast" response mode, and the calibration was checked before each study using the "test" switch. Measurements made when the oximeter indicated "low perfusion" were excluded.

Results

Fifty-five patients with a PaO₂ of 65 mm Hg or less qualified for inclusion in this study. There were 30 males and 25 females with a mean age of 60.6 (range, 17-82) years. They included 47 caucasian, four black, and four hispanic subjects. Pulmonary function tests (lung volumes, maximal expiratory flows) revealed patterns of obstruction in 36, restriction in nine, combined disease in three, and were within normal limits in seven. Forty-four patients had measurements made with the HP 47201A, 50 patients with the Biox IIA, and 39 patients with both.

Figure 1 presents the relationship between resting PaO₂ and cutaneous oximetry SaO₂ measurements. These results demonstrate that greater than 80 percent of patients with a resting PaO₂ of 7.33 kPa (55 mm Hg) or less had a cutaneous oximetry SaO₂ level of more than 85 percent (14 of 17 with the HP 47201A [Fig 1, top], 18 of 21 with the Biox IIA [Fig 1, bottom]). Based on current Medicare guidelines, these patients would not qualify for oxygen therapy if the cutaneous oximetry SaO₂ measurements were used in place of PaO₂ measurements.

Using direct CO-oximetry SaO₂ measurements from arterial blood and the 85 percent criterion, 17 of 22 patients with a resting PaO₂ measurement less than 7.33 kPa (55 mm Hg) would be denied therapy. The correlation coefficients (r) between the cutaneous SaO₂ measurements and CO-oximetry SaO₂ measurements of available hemoglobin were 0.604 (standard error of the estimate [SEE] of SaO₂ = 1.82 percent) with the HP 47201A and 0.595 (SEE of SaO₂ = 2.17 percent) with the Biox IIA. No patient had a measured SaO₂, by either cutaneous or direct method, less than 85 percent when the PaO₂ was greater than 7.33 kPa (55 mm Hg). Therefore, no patient would have received oxygen therapy inappropriately based on the SaO₂ measurements made by cutaneous oximetry.

Because under normal circumstances a PaO₂ of 55 mm Hg corresponds more closely to an SaO₂ of 88 percent rather than 85 percent, we also analyzed the data using 88 percent saturation as a threshold criterion for initiation of oxygen therapy. Using this criterion, the number of patients with a PaO₂ of 7.33 kPa (55 mm Hg) or less who would be denied therapy based on cutaneous oximetry measurements would be reduced to four of 17 (24 percent) with the HP 47201A, 12 of 21 (57 percent) with the Biox IIA, and 13 of 22 (59 percent) with the CO-oximeter. However, some patients with a PaO₂ greater than 7.33 kPa, 55 mm Hg (range, 7.60 to 8.66 kPa [57 to 65 mm Hg]) had a cutaneous oximetry SaO₂ level less than 85 percent (two of 27 [seven percent] with the HP 47201A and six of 29 [21 percent] with the Bixo IIA).

Discussion

The results of this study demonstrate that greater than 80 percent of patients with chronic lung disease who would qualify for ambulatory oxygen supplementation under the current Medicare criteria for resting PaO₂ (7.33 kPa [55 mm Hg] or less) would not qualify for such therapy on the basis of a simultaneous SaO₂ measured by cutaneous oximetry (85 percent or less). Changing the current Medicare criteria to include a more appropriate threshold criterion of 88 percent SaO₂, instead of 85 percent, would reduce the number.
of patients denied this therapy using cutaneous oximetry measurements. However, a small number of patients who would not qualify based on a PaO₂ measurement would then qualify based upon a cutaneous SaO₂ measurement less than 88 percent.

Cutaneous oximetry offers a rapid, noninvasive, and portable means for the estimation of arterial oxyhemoglobin saturation. The HP 47201A (no longer in production) uses an eight-wavelength method in this determination, while newer oximeters, such as the Biox IIA, use a two-wavelength method associated with arterial pulsation. By comparing the absorbance of transmitted light before and during arterial pulsation, absorbance due to arterial oxyhemoglobin can be separated from static background absorbance attributable to skin pigmentation, soft tissue components, and venous blood.

Recent studies, however, have demonstrated some limitations of cutaneous oximeters. For SaO₂ values greater than 70 percent, the absolute inaccuracy (95 percent confidence limits) of the cutaneous measurement of SaO₂ when compared with direct measurement of SaO₂ is ±4.0 to 5.0 percent; for SaO₂ values less than 70 percent, the error may be appreciably larger. For monitoring changes in SaO₂, the inaccuracy is ±3.0 percent.

Invalid measurements can also occur in conditions which result in decreased cutaneous blood flow, or elevated bilirubin, MetHb, or COHb levels. Decreased perfusion may be sensed by the oximeter thus triggering a warning light. We did not include measurements made when this light was on. In this study, no patient had either clinical evidence of jaundice or laboratory evidence of an elevated bilirubin. Two patients had a MetHb level greater than 1 percent while five had a COHb level greater than 4 percent. Exclusion of data from these patients did not appreciably improve the relationship between the cutaneous oximetry and direct PaO₂ measurements. The factors responsible for the discrepancies we observed included the shape of the oxyhemoglobin dissociation curve for PaO₂ values near 60 mm Hg, the inherent inaccuracy and imprecision of cutaneous oximetry measurements, and an occasional aberrant cutaneous oximetry measurement. For example, one patient in this study who, in the absence of recognized technical problems, had a cutaneous oximetry SaO₂ of 95 percent with a directly measured SaO₂ of 91 percent, COHb of 2.1 percent, and PaO₂ of 54 mm Hg.

Our study does not permit conclusions about whether direct measurements of PaO₂ are better than cutaneous oximetry SaO₂ measurements for predicting efficacy of long-term oxygen therapy. Single measurements of either may not be the optimal way to predict who will respond to such therapy. Continuous measurements of SaO₂ during normal activities (including sleep) with portable cutaneous oximeters may provide parameters (eg, mean SaO₂, percentage time with SaO₂ less than 90 percent, etc) which better identify those patients who might benefit most from long-term oxygen therapy.

In summary, based on the results of this study, we conclude that cutaneous oximetry measurements currently cannot be substituted equivalently for measurements of PaO₂ for decisions regarding initiation of long-term oxygen therapy. This conclusion is in accord with the recommendations of a recent conference on home oxygen therapy. Cutaneous oximetry may be useful for screening; if the SaO₂ is less than 85 percent with the patient at rest, then the need for direct measurement of PaO₂ is obviated. Additional studies are needed to explore the other potential roles for cutaneous oximetry in identifying patients likely to benefit from ambulatory oxygen therapy.

ACKNOWLEDGEMENTS: We wish to thank Jeffrey Johnson and Catherine Fonzi for technical contributions, Lela Prewitt for data processing and statistical assistance, and Kenneth M. Moser, M.D., for review of the manuscript.

REFERENCES

3 Criteria for Medicare coverage of oxygen services in the home. Federal Register April 5, 1985, 50.