Pulse oximetry in severe carbon monoxide poisoning

NB Hampson

Chest 1998;114;1036-1041

Pulse Oximetry in Severe Carbon Monoxide Poisoning*

Neil B. Hampson, MD, FCCP

Study objectives: **To evaluate the accuracy and quantitate the error of pulse oximetry measurements of arterial oxygenation in patients with severe carbon monoxide (CO) poisoning.**
Design: **Retrospective review of patient clinical records.**
Setting: **Regional referral center for hyperbaric oxygen therapy.**
Patients: **Thirty patients referred for treatment of acute severe CO poisoning who demonstrated carboxyhemoglobin (COHb) levels >25%, with simultaneous determinations of arterial hemoglobin oxygen saturation by pulse oximetry (Spo_2) and arterial blood gas (ABG) techniques.**
Measurements and results: **COHb levels and measurements of arterial oxygenation from pulse oximetry, ABG analysis, and laboratory CO oximetry were compared. Spo_2 did not correlate with COHb levels. Spo_2 consistently overestimated the fractional arterial oxygen saturation. The difference between arterial hemoglobin oxygen saturation (Sao_2) calculated from ABG analysis and Spo_2 increased with increasing COHb level.**
Conclusions: **Presently available pulse oximeters overestimate arterial oxygenation in patients with severe CO poisoning. An elevated COHb level falsely elevates the Sao_2 measurements from pulse oximetry, usually by an amount less than the COHb level, confirming a prior observation in an animal model. Accurate assessment of arterial oxygen content in patients with CO poisoning can currently be performed only by analysis of arterial blood with a laboratory CO-oximetry.**
(CHEST 1998; 114:1036–1041)

Key words: carbon monoxide; oxygenation; poisoning; pulse oximetry

Abbreviations: ABG=arterial blood gas; CO=carbon monoxide; COHb=carboxyhemoglobin; MetHb=methemoglobin; O_2Hb=oxyhemoglobin; $O_2Hb\%$=fractional oxygen saturation; pHa=arterial pH; RHb=reduced or deoxyhemoglobin; Sao_2=arterial hemoglobin oxygen saturation; Sao_2(calc)=arterial hemoglobin oxygen saturation calculated from arterial blood gas values; Spo_2=arterial hemoglobin oxygen saturation measured by pulse oximetry

Pulse oximetry is widely utilized in emergency medical settings for the immediate evaluation of a patient's oxygenation status. The technology provides an instantaneous, noninvasive, *in vivo* estimate of arterial hemoglobin saturation with oxygen. Emergency department pulse oximetry screening with detection of unsuspected hypoxemia has been shown to result in significant changes in medical treatment,[1,2] leading some to recommend that routine pulse oximetry be performed when evaluating unselected patients presenting with illness.[2]

Four species of hemoglobin typically circulate in adult blood–oxyhemoglobin (O_2Hb), reduced or deoxyhemoglobin (RHb), methemoglobin (MetHb), and carboxyhemoglobin (COHb). At normobaric ambient pressure, arterial oxygen content is almost completely accounted for by oxygen bound to hemoglobin. Dissolved oxygen contributes little to arterial oxygen content, even during administration of supplemental oxygen. As such, oxygen delivery to tissue is dependent on the concentration of O_2Hb. A variety of terms exist to describe the oxygen saturation of arterial hemoglobin.[3,4] Fractional saturation is measured spectrophotometrically by a CO-oximetry as $O_2Hb/(O_2Hb + RHb + COHb + MetHb) \times 100\%$. Functional saturation has been described as $O_2Hb/(O_2Hb+RHb)\times100\%$. Functional saturation therefore excludes hemoglobin species that cannot carry oxygen and describes the oxygen bound to hemoglobin as a percentage of the oxygen binding capacity.

Pulse oximetry estimates of arterial hemoglobin oxygenation are generally accurate because dyshemoglobins are usually present in only small amounts. In such instances, functional hemoglobin saturation approximates fractional hemoglobin saturation. Among the reported sources of error for the technique, however, is the presence of a significant

*From the Section of Pulmonary and Critical Care Medicine, Virginia Mason Medical Center, Seattle, WA.
Manuscript received October 2, 1997; revision accepted April 6, 1998.
Correspondence to: Neil B. Hampson, MD, Virginia Mason Clinic C7-PUL, 1100 Ninth Ave, Seattle, WA 98111, email: cidnbh@vmmc.org

quantity of MetHb or COHb.[5–8] Significant dyshemoglobinemia results in divergence between functional and fractional hemoglobin saturation.

In the case of carboxyhemoglobinemia, a false reading from pulse oximetry may be especially misleading. Carbon monoxide (CO) binds avidly to hemoglobin at sites that normally carry oxygen, reducing arterial oxygen binding capacity. When arterial hemoglobin is partially saturated with CO, pulse oximetry measurements have been shown to overrepresent true arterial fractional hemoglobin saturation with oxygen in both an animal model[9] and limited numbers of humans.[10–13] The present study was performed to further investigate this phenomenon in a large group of patients with severe CO poisoning and to evaluate the degree of pulse oximetry error in the presence of high levels of COHb in humans.

Materials and Methods

Records of all patients treated with hyperbaric oxygen for acute CO poisoning at Virginia Mason Medical Center in Seattle from December 1988 to November 1997 were screened. Those with measured COHb values >25.0% as determined by CO oximetry of arterial or venous blood were reviewed in detail. Charts of patients with documented simultaneous measurement of arterial hemoglobin oxygen saturation by pulse oximetry (Spo_2), arterial blood gas (ABG) values (arterial pH [pHa], $Paco_2$, Pao_2), and COHb were included in the final analysis. For this group with complete simultaneous data, patient demographic information and details of the CO exposure were abstracted in addition to the laboratory data.

Spo_2 measurements were compared with COHb and simultaneous arterial hemoglobin oxygen saturation (Sao_2) values determined from ABG analysis. "Sao_2(calc)" was calculated from ABG values for Pao_2, pHa, and temperature per standard method[14] and is an estimate of functional arterial hemoglobin saturation. In cases in which CO oximetry had been performed on arterial blood, a direct measurement of arterial oxygen saturation, O_2Hb, was available for comparison with Spo_2. The ratio of O_2Hb to total hemoglobin was defined as fractional oxygen saturation ($O_2Hb\%$). Finally, the magnitude of the effect of COHb elevation on Spo_2 was evaluated by comparing the difference between Sao_2(calc) and Spo_2 with the COHb level.

Least squares linear regression was used to compare COHb with Spo_2 and the Sao_2(calc)-Spo_2 difference. The technique of Bland and Altman[15] for assessing agreement between two methods of clinical measurement was used for the comparison of Spo_2 with $O_2Hb\%$ by CO oximetry.

Results

In the decade reviewed, 740 patients were treated for acute CO poisoning with hyperbaric oxygen. Among those, 30 had COHb levels >25.0% in conjunction with documented simultaneous ABG analysis and measurement of Spo_2. As these measurements were performed in the emergency departments of referring hospitals and abstracted from medical records transferred with the patient, information regarding the brand or model of pulse oximeter, ABG analyzer, and CO-oximetry was not available.

A total of 22 male and 8 female patients comprised the study group. They ranged in age from 20 to 91 years, averaging 46±19 years (mean±SD). Sources of the CO exposure included motor vehicles (21), fires (5), gasoline generators (2), propane stove (1), and miscellaneous gasoline engine (1). The reason for the exposure was accidental in 15 instances and intentional in 15 instances. Twenty-four of the patients (80%) lost consciousness with the poisoning episode.

Laboratory data are summarized in Table 1. COHb levels ranged from 25.2 to 54.0% (mean, 36.9%±8.9%). In all 30 patients, Spo_2 was >90%. All patients except three (patients 10, 12, 30) were receiving supplemental oxygen when data were obtained. Figure 1 compares Spo_2 with COHb levels. While the slope of the best fit line was significantly different from zero (slope=−0.13, y intercept =101.3, r=−0.40, p=0.0304), it can be seen from the graph that Spo_2 shows only minor dependence

Table 1—*Laboratory and Pulse Oximetry Data*

Patient	COHb	pHa/$Paco_2$/Pao_2	Sao_2(calc)	Spo_2	$O_2Hb\%$
1	44.3	7.38/34/306	100	91	
2	45.5	7.35/24/>250	100	100	
3	40.9	7.26/36/398	100	100	
4	30.7	7.45/30/169	99	98	68.0
5	47.7	7.41/28/132	99	91	
6	50.2	7.50/27/314	100	95	
7	25.2	7.36/38/237	100	100	
8	28.2	7.42/36/67	93	94	69.1
9	29.4	7.38/32/262	100	97	70.2
10	42.4	7.35/34/78	95	94	56.3
11	25.7	7.39/44/115	98	97	71.1
12	25.7	7.44/37/77	95	96	72.0
13	54.0	7.43/31/390	100	96	
14	34.9	6.99/40/532	100	98	64.7
15	43.0	7.43/34/305	100	95	
16	28.7	7.14/37/391	100	94	70.0
17	29.0	7.36/29/554	100	99	69.0
18	33.5	7.34/38/198	99	98	65.2
19	46.1	7.39/37/396	100	99	
20	27.0	7.47/36/516	100	99	
21	40.7	7.24/27/277	100	92	
22	41.4	7.12/22/457	100	97	55.6
23	26.6	7.40/44/430	100	100	71.6
24	43.3	7.32/37/382	100	99	55.6
25	46.3	7.46/36/250	100	92	52.7
26	44.0	7.36/29/245	100	98	
27	25.5	7.37/45/192	99	98	
28	44.8	7.36/44/515	100	91	54.6
29	29.1	7.40/34/103	97	100	
30	32.2	7.47/34/114	98	96	

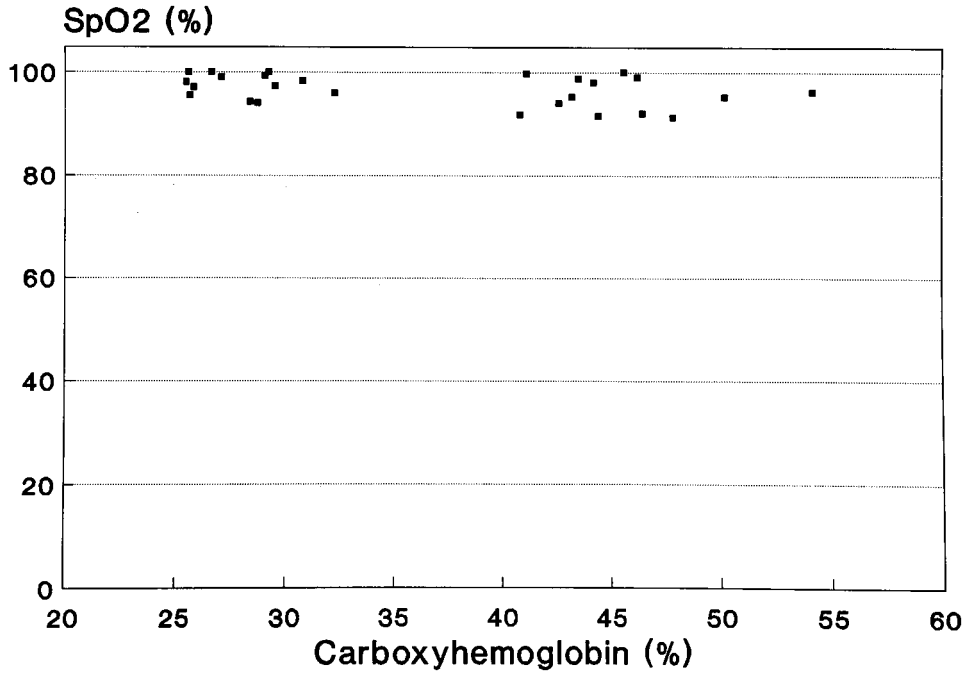

FIGURE 1. Comparison of Spo_2 with simultaneous blood COHb measurements.

on COHb, requiring an 8% increase in COHb to observe a 1% decrease in Spo_2.

In 15 patients, COHb determination was performed by CO oximetry analysis of arterial (rather than venous) blood, providing measured values for O_2Hb, reduced or deoxyhemoglobin, COHb, and MetHb, in addition to O_2Hb%. In those patients, O_2Hb% ranged from 52.7 to 72.0% (mean, 64.4%±7.2%). Spo_2 is compared with O_2Hb% in Figure 2. By the comparison method of Bland and Altman,[15] there is significant discrepancy between the two measurements (bias 31.9; 95% confidence interval, 28.3, 35.5). The difference between Spo_2 and fractional Sao_2 ranged from 24.0 to 43.4%. COHb was greater than the Spo_2-fractional Sao_2 difference in 10 patients, equal in 1 patient, and less in 4 patients.

As was the case with Spo_2, Sao_2(calc) from ABG analysis was >90% in all patients. As can be seen in Table 1, Sao_2(calc) was less than Spo_2 in 3 patients, equal in 4 patients, and greater than Spo_2 in 23 patients. The difference between them is graphed in Figure 3 as a function of COHb. Linear regression analysis demonstrates a weak but statistically significant correlation between increasing COHb and the increasing difference between Sao_2(calc) and Spo_2 ($r=0.55$, $p=0.0018$).

DISCUSSION

Pulse oximetry is a noninvasive method for measurement of arterial oxygenation. Several excellent reviews describing the method have been published.[3,8,16] In brief, the technique transmits two wavelengths of light through tissue (typically 660 and 940 nm), measuring changes in absorbance at each wavelength over time. Light absorption by tissue is cyclic due to cardiac cycling and the resultant pulsation of arterial blood into the tissue bed. The pulse oximeter measures the change in light transmitted during diastole (background) from that during arterial pulsation and attributes the difference to absorbance by arterial blood entering the monitored field. Each form of hemoglobin has a characteristic light absorption pattern (extinction curve) (Fig 4). O_2Hb and RHb are the two forms of hemoglobin present in arterial blood in greatest amounts under normal conditions. Because they have different extinction curves, it is possible to estimate the relative amounts of each present, utilizing knowledge of their light absorption characteristics at each of the two wavelengths.

To measure Spo_2, pulse oximetry creates ratios of the intensity of light transmitted at each of the two wavelengths during both the constant and fluctuating (pulsatile) phases. The technique then combines those ratios to produce a reading from a "look-up" table of saturation values based on experimental data from healthy adult volunteers and programmed into the device.[3,16] The degree to which Spo_2 approximates either fractional or functional saturation depends on the degree to which hemoglobin species other than O_2Hb and RHb are present in the

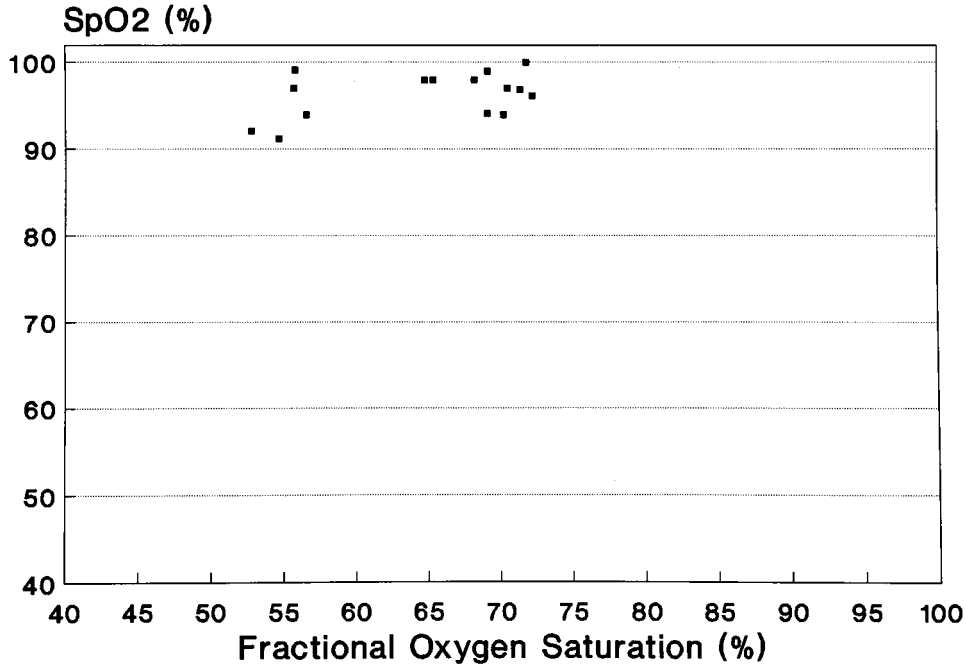

FIGURE 2. Comparison of Spo_2 with simultaneous $O_2Hb\%$ measured by CO oximetry.

monitored tissue, as well as to the average levels accounted for in the "look-up" table by the device manufacturer.

A 1987 laboratory study by Barker and Tremper[9] examined the effect of an elevation in COHb on pulse oximetry measurements in dogs. They measured the degree to which a pulse oximeter overestimated arterial hemoglobin oxygen saturation in the presence of carboxyhemoglobinemia, demonstrating that Spo_2 declines to a small degree as COHb

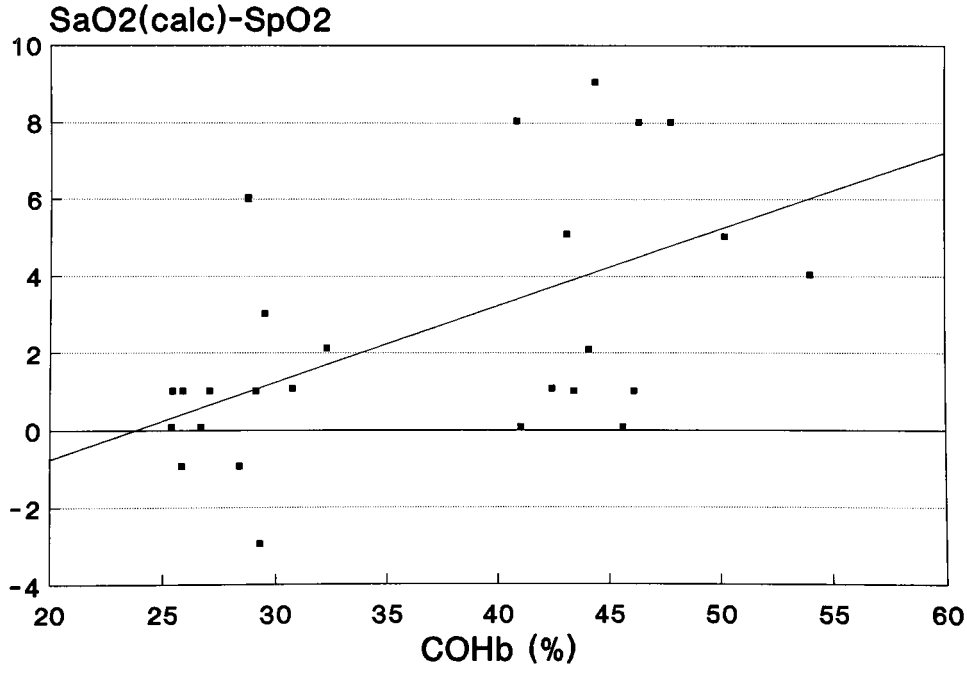

FIGURE 3. Comparison of COHb levels with the difference between Sao_2(calc) and simultaneous pulse oximetry measurement (Spo_2) (slope of line=0.199, y intercept=−4.75, SD of residuals from the line=2.75).

increases, at constant inspired oxygen concentration. In an animal breathing 100% oxygen (predicted Sao_2 100%), Spo_2 was found to measure approximately 94% when COHb was raised to 50%. A number of subsequent publications, however, reported that pulse oximeters interpret COHb almost exactly as O_2Hb and that Spo_2 represents the sum of O_2Hb and COHb.[8,10–13,17] It has been observed that pulse oximeters could only interpret COHb as O_2Hb if the two species had identical absorption coefficients at both 660 nm and 940 nm, which they do not.[4] COHb and O_2Hb have similar absorption characteristics at 660 nm, but not at 940 nm (Fig 4). The effect of COHb on Spo_2 must therefore be something other than exact measurement as O_2Hb, a concept suggested by the early work of Barker and Tremper.[9]

In the present study, Spo_2 correlated poorly with extremely elevated COHb levels and grossly overestimated O_2Hb% in a group of patients with severe impairment in oxygen binding capacity due to COHb formation (Figs 1, 2). While other investigators have described similar findings, data from only a small number of patients with CO poisoning of this severity have been published. An extensive English-language literature search yielded comparisons of pulse oximetry readings with other measures of arterial oxygenation in only seven patients with COHb elevation to the degree reported herein (COHb>25.0%).[10–12] These limited clinical data have made it difficult to accurately describe the degree of effect of extreme elevation of COHb on Spo_2.

The difference between pulse oximetry Spo_2 and O_2Hb% has been described as the "pulse oximetry gap."[10] COHb has been reported to approximate the pulse oximetry gap in previous studies involving limited numbers of patients.[10–12] In the present study, the pulse oximetry gap was less than COHb in 19 patients (73%). This suggests that while the presence of COHb falsely elevates the Spo_2 readings made by presently available pulse oximeters, the amount of that elevation is generally less than the COHb percentage. The similar extinction coefficients for COHb and O_2Hb at 660 nm contribute to this phenomenon, but the differing extinctions at 990 nm result in a contribution to the pulse oximeter reading that is usually less than the amount of COHb present. In other words, COHb is seen similarly, but not entirely, as O_2Hb by current generation pulse oximeters. Barker and Tremper[9] first noted this in their canine study.

The degree to which Spo_2 is less than Sao_2(calc) is dependent on the amount of COHb present (Fig 3). As COHb increases to extreme levels, the fact that it is not interpreted exactly as O_2Hb becomes increasingly apparent, especially when COHb is >40%. Clinical reports on this topic have included only three patients with COHb levels >40%,[10,12] likely contributing to difficulty in recognizing the phenomenon. When published data from those patients are reexamined closely, however, the pulse oximetry gap (measured or calculated) is indeed seen to be less than the COHb percentage in two of the three. It is

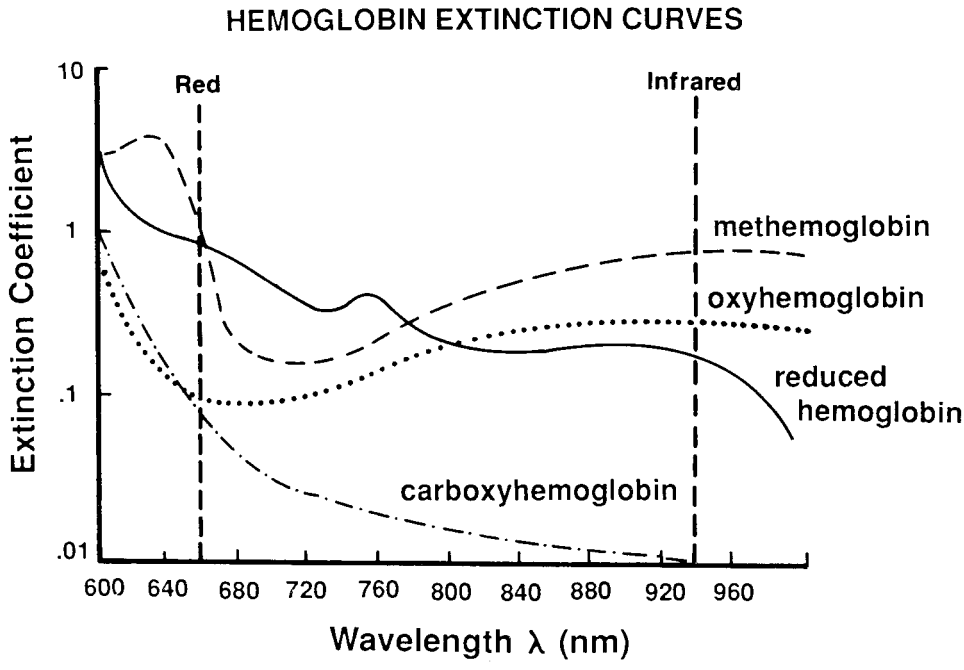

FIGURE 4. Absorption spectra of four hemoglobin species (reproduced with permission from Tremper and Barker[9]).

interesting to compare the degree to which Spo_2 is less than the predicted Sao_2 in the current group of patients with that observed in dogs by Barker and Tremper.[9] For a patient with COHb 50%, the Sao_2 predicted from ABG analysis is approximately 5% greater than the observed Spo_2 (Fig 3), almost identical to the value seen in their animal model.

In patients without significant levels of COHb or MetHb, functional oxygen saturation approximates fractional saturation. In such instances, pulse oximetry should be expected to reliably estimate both, assuming that another of the known causes for device error does not interfere. Among patients with markedly elevated COHb levels, however, Spo_2 grossly overestimates $O_2Hb\%$ potentially providing a false sense of security with regard to arterial oxygenation. In the present study, two thirds of the patients demonstrated Spo_2 values at or above the normal range ($97\pm1\%$), despite significant reduction in arterial oxygen content due to carboxyhemoglobinemia.

While screening of emergency department patients with pulse oximetry may identify patients with previously unsuspected hypoxemia, the potential for false-negative results in patients with dyshemoglobinemia must be recognized. This is an issue of significant magnitude, as >40,000 patients with CO poisoning are estimated to present to emergency departments in the United States each year.[18] Until pulse oximeters are developed that accurately account for elevations in COHb and MetHb, physicians must maintain high vigilance for CO poisoning and remember to assess both COHb levels and arterial oxygen content with CO oximetry in appropriate clinical situations.

References

1 Lambert MA, Crinnion J. The role of pulse oximetry in the accident and emergency department. Arch Emerg Med 1989; 6:211–215

2 Mower WR, Sachs C, Nicklin EL, et al. Effect of routine emergency department triage pulse oximetry screening on medical management. Chest 1995; 108:1297–1302

3 Tremper KK, Barker SJ. Pulse oximetry. Anesthesiology 1989; 70:98–108

4 Ralston AC, Webb RK, Runciman WB. Potential errors in pulse oximetry: I. Pulse oximeter evaluation. Anaesthesia 1991; 46:202–206

5 Lindberg LG, Lennmarken C, Vegfors M. Pulse oximetry—clinical implications and recent technical developments. Acta Anaesthesiol Scand 1995; 39:279–287

6 Mardirossian G, Schneider RE. Limitations of pulse oximetry. Anesth Prog 1992; 39:194–196

7 Ralston AC, Webb RK, Runciman WB. Potential errors in pulse oximetry: III. Effects of interference, dyes, dyshaemoglobins and other pigments. Anaesthesia 1991; 46:291–295

8 Severinghaus JW, Kelleher JF. Recent developments in pulse oximetry. Anesthesiology 1992; 76:1018–1038

9 Barker SJ, Tremper KK. The effect of carbon monoxide inhalation on pulse oximetry and transcutaneous Po_2. Anesthesiology 1987; 66:677–679

10 Buckley RG, Aks SE, Eshom JL, et al. The pulse oximetry gap in carbon monoxide intoxication. Ann Emerg Med 1994; 24:252–255

11 Gonzalez A, Gomez-Arnau J, Pensado A. Carboxyhemoglobin and pulse oximetry. Anesthesiology 1990; 73:573

12 Graybeal JM, Russell GB, Basile J. Oxygenation of the carbon monoxide poisoned patient. Respir Care 1991; 36:57–62

13 Vegfors M, Lennmarken C. Carboxyhaemoglobinaemia and pulse oximetry. Br J Anaesth 1991; 66:625–626

14 Severinghaus JW. Oxyhemoglobin dissociation curve correction for temperature and pH variation in human blood. J Appl Physiol 1958; 12:485–486

15 Bland JM, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. Lancet 1986; 1:307–310

16 Wukitsch MW, Petterson MT, Tobler DR, et al. Pulse oximetry: analysis of theory, technology, and practice. J Clin Monit 1988; 4:290–301

17 Alexander CM, Teller LE, Gross JB. Principles of pulse oximetry: theoretical and practical considerations. Anesth Analg 1989; 68:368–376

18 Hampson NB. Emergency department visits for carbon monoxide poisoning in the Pacific Northwest. J Emerg Med 1998; 16:695–698