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**Brief Report** 

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# Noninvasive pulse CO-oximetry expedites evaluation and management of patients with carbon monoxide poisoning Neil B. Hampson MD\*

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#### Abstract

**Purposes:** Pulse CO-oximetry (Rad-57; Masimo Corp, Irvine, CA) has been available since 2005. To date, all published clinical studies have focused on clinical reliability and whether the device enhances case finding through screening of various populations. This study examines whether use of pulse CO-oximetry shortens the time to diagnosis and treatment of patients with carbon monoxide (CO) poisoning. **Basic Procedures:** Data from the joint Undersea and Hyperbaric Medical Society/Centers for Disease Control and Prevention CO poisoning surveillance system from August 2008 to July 2011 were analyzed. Of 1711 cases of CO poisoning treated with hyperbaric oxygen in the United States and reported through the system, 1606 had their initial carboxyhemoglobin (COHb) level measured by laboratory CO-oximetry and 105 by pulse CO-oximetry. Patients were selected from the laboratory CO-oximetry group to match each of the 105 patients evaluated by pulse CO-oximetry in 5 characteristics— age, sex, race/ethnicity, intent of poisoning, and occurrence of loss of consciousness. Measures of timeliness in measurement and management were compared between the 2 groups.

**Main Findings:** Patients with initial COHb measurement by pulse CO-oximetry had significantly shorter time to measurement of COHb, higher average levels of COHb, and shorter time from the end of CO exposure to the initiation of hyperbaric oxygen treatment. On average, patients evaluated by pulse CO-oximetry reached the hyperbaric chamber 1 hour faster than did patients evaluated by laboratory CO-oximetry (P < .01).

**Principle Conclusions:** Pulse CO-oximetry is associated with more rapid diagnosis and initiation of hyperbaric oxygen therapy in CO-poisoned patients compared with laboratory CO-oximetry. The impact on clinical outcome remains to be determined.

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#### 1. Introduction

Basic principles of toxicology include identification and quantification of the poison involved, clearance of the toxin from the body, and administration of antidotes, all as rapidly as possible. In the case of carbon monoxide (CO) poisoning, the toxin is CO, clearance is accelerated by the administration of oxygen, and a subset of the most severely poisoned patients are treated with hyperbaric oxygen [1].

Diagnosis of CO poisoning typically includes presentation with a history compatible with exposure, symptoms typical of the syndrome, and demonstration of an elevated blood carboxyhemoglobin (COHb) level [1]. Historically, COHb levels have been measured on arterial or venous blood samples using a laboratory CO-oximeter. This has required that the patient or his/her blood sample be transported to a

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hospital with laboratory CO-oximetry capability. In one region of the country, it was demonstrated that even within the past decade, only one-half of acute care hospitals had laboratory CO-oximetry capability [2], likely due to the expense of the instrumentation. Additional delay arises when it is determined that hyperbaric oxygen therapy is appropriate for the patient, and transfer to yet another medical center with hyperbaric capability must be coordinated and accomplished.

The RAD-57 (Masimo Corp) is a handheld, noninvasive pulse CO-oximeter, capable of measuring COHb at the scene of discovery by transilluminating the fingertip with multiple wavelengths of near-infrared light [3]. Since it was introduced to the market in 2005, it has been adopted and used by many first-responder units such as paramedics and firefighters. No one, however, has demonstrated that the use of the device has reduced the time to measurement of a COHb level or the time to hyperbaric oxygen treatment of CO poisoning. This study examines those variables in a large population of patients referred for hyperbaric oxygen treatment of CO poisoning across the United States, comparing the timing of diagnosis and treatment in those with initial COHb measurement by hospital laboratory CO-oximetry vs those initially evaluated with pulse CO-oximetry.

#### 2. Materials and methods

From August 2008 to October 2011, the Undersea and Hyperbaric Medical Society maintained an online system for surveillance of cases of CO poisoning treated with hyperbaric oxygen. When a patient was treated in a facility enrolled in this voluntary program, facility staff logged on to a secure Web site and entered nonidentifiable demographic and epidemiologic data about the exposure. The system was been financially supported by and operated in conjunction with the Centers for Disease Control and Prevention, in an effort to expand surveillance of CO poisoning and also to test the hypothesis that teaming with a medical specialty society in this fashion is an effective and efficient way to perform disease surveillance. Complete details of the development and operation of the program are available in other publications [4,5].

Among the 40 data fields of information collected about each case were questions about the initial COHb level, including the time from the end of CO exposure to measurement, as well as the method used. Also included was a question about the time from the end of CO exposure to the initiation of hyperbaric oxygen treatment. In the first 3 years of operation (August 1, 2008–July 31, 2011), a total of 1912 patients treated with hyperbaric oxygen for CO poisoning at 63 facilities in 42 US states were reported. It has been estimated that the system captured approximately one-half of patients receiving hyperbaric oxygen therapy for CO poisoning, based on historical data [6]. Of the 1921 patients reported, 118 were excluded from this analysis because their poisoning was treated longer than 24 hours after CO exposure. Because many hyperbaric facilities in the United States limit therapy to those referred within 12 or 24 hours of exposure [7], that limit was applied in the present analysis to avoid bias introduced by patients reported from only a subset of facilities.

An additional 92 patients were excluded from analysis because details of their COHb level or the method used to measure it were unknown or not reported. The remaining 1711 patients formed the study population. Among these, 1606 (94%) had their initial COHb level measured by laboratory CO-oximetry and 105 (6%) by pulse oximetry. It is presumed that most measurements by pulse CO-oximetry were performed at the scene by first responders, but some may have been performed in emergency department triage settings. That information was not collected by the surveillance system.

To compare the much smaller pulse CO-oximetry group with a group of similar size and baseline characteristics, the database was queried in an attempt to identify a cohort of laboratory CO-oximetry patients who matched each of the 105 pulse CO-oximetry patients in 5 characteristics—sex, age ( $\pm 1$  year), race/ethnicity, intent of poisoning (accidental vs intentional), and occurrence of loss of consciousness. When more than 1 patient from the laboratory CO-oximetry group matched all 5 characteristics of a patient evaluated by pulse CO-oximetry, the patient evaluated by laboratory COoximetry treated first was selected.

Methods of data analysis included descriptive statistics, 2-tailed Fisher exact test, and t test.

#### 3. Results

For the 105 patients evaluated by pulse CO-oximetry, it was possible to obtain a match for 5 of 5 characteristics from the group of patients evaluated by laboratory CO-oximetry in 95 instances. The remaining 10 patients matched with a laboratory CO-oximetry patient in 4 of 5 characteristics. In 8 cases, race/ethnicity was the mismatch. In 2 cases, age was the mismatch because it was outside the range of  $\pm 1$  year. Data for baseline characteristics for the 2 groups are shown in Table 1. Patients evaluated by laboratory CO-oximetry used for analysis were treated at 37 different hyperbaric facilities. The 105 patients evaluated by pulse CO-oximetry were treated at 18 different facilities, 15 of which also treated patients evaluated by laboratory CO-oximetry. Only 4 o 105 patients in the pulse CO-oximetry group were treated at 3 facilities that did not also treat patients in the laboratory COoximetry group included in the analysis.

The reported time from the end of CO exposure to COHb measurement for laboratory CO-oximetry cases was  $1.7 \pm 1.2$  hours (mean  $\pm$  SD; range, 0.15-6.25 hours) and  $1.1 \pm 1.3$  hours for pulse CO-oximetry cases (range, 0.0-7.0 hours) (P < .01)

#### Pulse CO-oximetry expedites management of CO poisoning

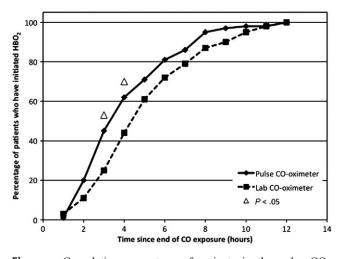
Table	Baseline characteristics and measures of time from the
end of	CO exposure to COHb measurement and initiation of
hyperba	aric oxygen therapy

	Laboratory CO-oximeter, n = 105	Pulse CO-oximetry, n = 105	
Sex	62% male	62% male	P = NS
Age (y)	$34 \pm 21$	$34 \pm 22$	P = NS
Race/Ethnicity			
Non-Hispanic white	45%	40%	P = NS
Black	29%	28%	P = NS
Hispanic white	24%	26%	P = NS
Other	3%	5%	P = NS
Intent (% accidental)	91%	91%	P = NS
Loss of consciousness	31%	31%	P = NS
Time to COHb	$1.7 \pm 1.2$ hours	$1.1 \pm 1.3$ hours	<i>P</i> < .01
COHb	$21.9\pm1.2\%$	$25.9\pm8.9\%$	P < .01
Time to HBO <sub>2</sub>	$5.3\pm2.6\ hours$	$4.4\pm2.3\ hours$	<i>P</i> < .01
~			

Continuous data are expressed as mean + SD. Abbreviation: NS, not significant.

(Table 1). Carboxyhemoglobin levels for the 2 groups were  $21.9\% \pm 10.2\%$  and  $25.9\% \pm 8.9\%$ , respectively (P < .01) (Table 1). When COHb levels from the total pool of patients evaluated by laboratory CO-oximetry ( $23.4\% \pm 10.4\%$ ; n = 1606) was compared with those of the pulse CO-oximetry group, the difference was also statistically significant (P < .02).

With regard to the time from the end of CO exposure to the initiation of hyperbaric oxygen treatment, the total population of 210 analyzed averaged  $4.9 \pm 2.5$  hours (range, 1.0-24.0 hours; median, 5.0 hours) (Table 1). For those with initial COHb measurement by laboratory CO-oximetry, the elapsed time was  $5.3 \pm 2.6$  hours (range, 1.0-12.0 hours) vs  $4.4 \pm 2.3$  hours for pulse CO-oximetry (range, 1.0-12.0 hours) (P < .01).



**Figure.** Cumulative percentage of patients in the pulse COoximetry group and the laboratory CO-oximetry group who have initiated hyperbaric oxygen treatment by hour for the first 12 hours after the end of CO exposure.

#### 4. Discussion

When the RAD-57 pulse CO-oximeter was introduced in 2005, it appeared to offer 2 potential opportunities [8]. First, it could be used as a screening tool to detect unsuspected cases of significant CO poisoning [9]. Mass screening at sites of potential CO exposure, screening of workers at increased risk for occupational CO exposure (eg firefighters), and screening of individuals presenting with nonspecific symptoms of illness were all seen as potential applications that might yield results in the form of discovering persons with otherwise unsuspected significant CO exposure.

The instrument has been shown to be useful for rapid mass screening [10,11]. Studies screening either emergency medical service patients or emergency department patients for occult CO poisoning have each identified some cases, but the yield has been generally low [12-14]. The accuracy and reliability of the instrument in the clinical setting have been questioned and also supported [15-17].

The second potential opportunity, that of accelerating diagnosis and treatment, has not yet been reported. Our results are consistent with pulse CO-oximetry decreasing both of these important time delays. Patients evaluated with pulse CO-oximetry had significantly shorter times from CO exposure to COHb determination and hyperbaric oxygen treatment.

The reduction in time to diagnosis accounted for approximately one-half of the reduction in time to hyperbaric treatment. This suggests that pulse CO-oximetry is expediting management both by reducing the time spent on diagnostic testing, as well as subsequent triage. As would be predicted, initial COHb levels were higher in the pulse CO-oximetry group ( $25.9\% \pm 8.9\%$  vs  $21.9\% \pm 10.2\%$ ) because they were measured an average of 0.6 hours, or 36 minutes, faster after the end of CO exposure than the laboratory CO-oximetry group.

One can only speculate whether shortening the delay to hyperbaric treatment by this amount of time improves clinical outcomes. Although hyperbaric oxygen has been demonstrated in one study to reduce the incidence of subsequent cognitive sequelae by approximately one-half as compared with normobaric oxygen therapy, the exact influence of specific periods of delay has yet to be determined [18,19]. In the absence of such information, most clinicians place an outside limit on the delay within which they will treat CO poisoning with hyperbaric oxygen and then attempt to administer therapy as rapidly as reasonable within that window. Because the demonstrated mechanisms of hyperbaric oxygen's effect in CO poisoning include correction of tissue hypoxia, restoration of cellular energy metabolism, limitation of brain lipid peroxidation, prevention of neuronal apoptosis, and attenuation of inflammation, it seems logical from a toxicologic standpoint that interrupting the toxin's (CO) effects earlier rather than later is an appropriate goal [1].

The major limitation to this analysis is that the end of CO exposure is frequently difficult to determine, and estimates must be often made when calculating the time to measurement of COHb and the time to the initiation of hyperbaric oxygen treatment. Any error in this regard would presumably be negated by similar errors across both study groups, but the potential for bias does exist for this variable.

#### 5. Conclusions

In summary, this is the first study that suggests that early application of pulse CO-oximetry technology in the COpoisoned patient may result in earlier diagnosis and more rapid initiation of definitive therapy. The significance in clinical outcome from the amount of time saved is unknown. Nonetheless, it seems reasonable to consider further study of this simple and inexpensive technology for its potential benefit and to hope that even more time delay to treatment can be saved through increased use and adoption of standardized patient management algorithms.

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