

CARBON MONOXIDE POISONING

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33 Noninvasive Measurement of Blood Carboxyhemoglobin with Pulse CO-Oximetry

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CONTENTS

33.1 Introduction	739
33.2 New Pulse CO-Oximetry Equipment	740
33.3 Conclusion	743
References	743

33.1 INTRODUCTION

Carbon monoxide (CO) poisoning causes symptoms that range from headache, nausea, vomiting, and dizziness to loss of consciousness, pulmonary and cardiac failure, and even death. Since the milder symptoms of CO poisoning are nonspecific, patients may be misdiagnosed with conditions such as viral illness, food poisoning, or motion sickness, depending upon the circumstances of the exposure. Diagnosis of CO poisoning requires both clinical awareness and biological confirmation of exposure.

Among the many mechanisms of toxicity of CO,¹⁻⁵ its effect on hemoglobin has been known for over a century. When inhaled, CO binds to hemoglobin in red blood cells transiting the pulmonary capillaries, forming carboxyhemoglobin (COHb). Because CO binds to hemoglobin in sites normally used to transport oxygen, the result is a decrease in the oxygen content of arterial blood and an associated reduction in peripheral oxygen delivery.

Since CO binds to hemoglobin much more avidly than oxygen, COHb remains in the circulation for hours and is a biomarker that can be measured to document recent exposure to CO. Until recently, determination of an individual's COHb level required drawing a blood sample and measuring it in a laboratory with a benchtop CO-oximeter or estimating it by measuring exhaled CO.⁶ Laboratory CO-oximeters use multiple wavelengths to spectrophotometrically distinguish and quantify the various hemoglobin species present (oxy-, deoxy-, carboxy- and methemoglobin).

33.2 NEW PULSE CO-OXIMETRY EQUIPMENT

Pulse oximetry is a technique widely utilized 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. Conventional pulse oximeters transmit two wavelengths of light through tissue (typically 660 and 940 nm), measuring changes in absorbance at each wavelength over time and calculating the functional saturation of hemoglobin with oxygen (SpO_2).

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 animals⁷ and humans.⁸ COHb and O_2Hb have similar absorption characteristics at 660 nm, but not at 940 nm (Figure 33.1). As a result, pulse oximeters measure COHb similarly to O_2Hb . Although some have suggested that the two species are measured identically, this is not true and the difference becomes apparent at high COHb levels.⁸

A new pulse CO-oximeter, developed by Masimo Corporation⁹ (see Addendum), utilizes eight wavelengths of light and is able for the first time to provide a noninvasive measurement of COHb ("SpCO") in seconds, in addition to conventional oximeter variables SpO_2 and pulse rate. The accuracy of the device has been demonstrated by the manufacturers up to 40% SpCO, with a range of $\pm 3\%$ around the measurement (Figure 33.2).⁹

Because the instrument is relatively new, only a few independent studies of it are available. In a series of 31 patients reporting to a pulmonary function laboratory for testing, arterial blood analysis by laboratory CO-oximetry confirmed the stated

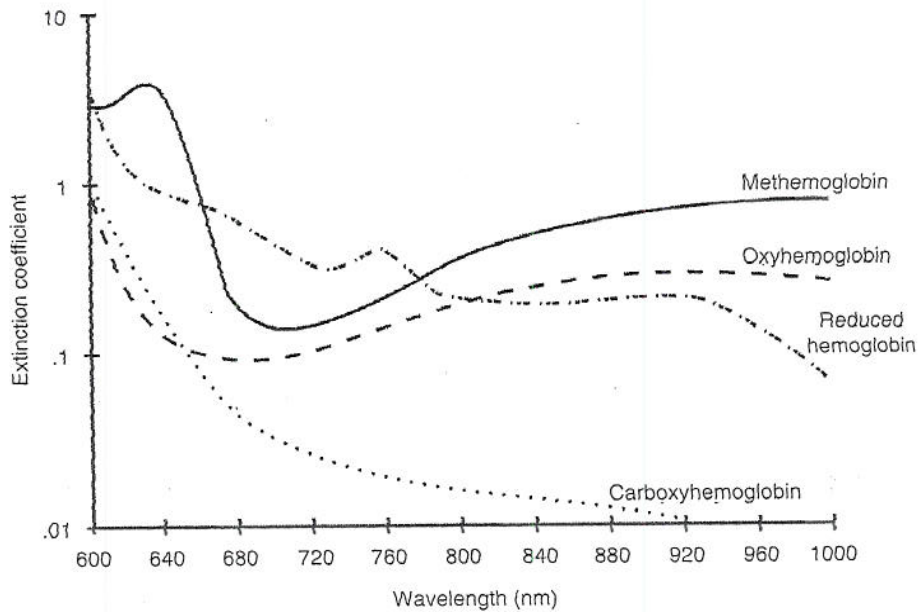


FIGURE 33.1 Absorption spectra of four hemoglobin species.

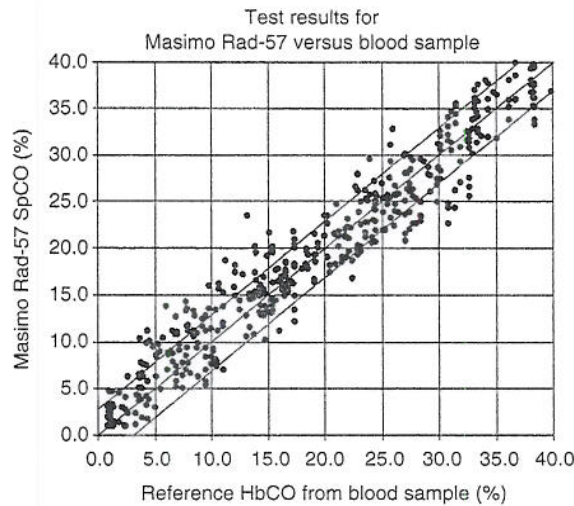


FIGURE 33.2 Pulse CO-oximetry SpCO measurements versus simultaneous laboratory CO-oximetry COHb levels in normal volunteers (From Masimo Corporation website. Rad-57 Pulse CO-oximeter. Available at: <http://www.masimo.com/rad-57/index.htm>. Accessed: September 26, 2005.)

accuracy of the device.¹⁰ However, the range for COHb in that population was only 0.8–9.3%. In a clinical laboratory study, ten volunteers breathed 500 ppm CO until COHb was raised to 15%.¹¹ SpCO correlated with CO-oximeter COHb with a precision of 2.19%.

The pulse CO-oximeter has also been used to measure baseline COHb levels in patients presenting to another pulmonary function laboratory for assessment of pulmonary diffusing capacity (DLCO).¹² The SpCO value obtained was then utilized to “correct” the measured DLCO when severity of impairment was graded by the interpreting physician. In another study, the pulse CO-oximeter was utilized in an ambulatory research setting to measure the blood COHb levels of smokers and nonsmokers exposed to second-hand cigarette smoke.¹³ In a case report, the device was used to continuously monitor COHb during treatment of a victim of CO poisoning resulting from smoke inhalation.¹⁴ At an actual initial COHb of 35%, the SpCO was 39%. After 150 min of normobaric 100% oxygen, the COHb was 5% with an SpCO of 6%. This same group has since reported on using the device to screen patients presenting to their emergency department (ED).¹⁵ Over 1700 patients had SpCO measured at triage. Not surprisingly, they found that self-reported smokers exhibited higher SpCO readings than nonsmokers ($5.3\% \pm 3.8\%$ versus $2.9\% \pm 2.7$; $p < .00001$). More importantly, they identified three cases of unsuspected CO poisoning that were confirmed through laboratory analysis. In all clinical studies to date, the device has been found to be convenient and easy to use.

It is felt that the 40,000 cases of CO poisoning diagnosed each year in US EDs underestimate the actual incidence and that many more cases are either not seen in an ED or are not diagnosed when seen.¹⁶ Because clinicians have traditionally only

ordered blood measurement of COHb when the condition was suspected, it is likely that there has been a tendency to measure COHb in the more symptomatic patient or in those whose exposure history was known. EDs, emergency medical support (EMS) providers and other first-responders will begin using the new pulse CO-oximeter soon. Since EMS providers and paramedics commonly use a pulse-oximeter to measure SpO₂ at the scene, one can predict that many instances of elevated SpCO will be discovered among patients without a classic history or recognized exposure to CO. A suggested triage and management plan for patients with elevated SpCO levels has recently been published to address this issue (Figure 33.3).¹⁷

Furthermore, many hospitals have not had the ability to measure COHb until now. One recent study of a four-state region found that less than one-half of the acute care hospitals had laboratory CO-oximetry available.¹⁸ This is due to the expense of the instrument, as suggested by the fact that hospitals without CO-oximeters tend to be located in smaller communities. Since pulse CO-oximeters are significantly less expensive, their availability will undoubtedly contribute to increased diagnosis of CO poisoning. Even though most hospitals without CO-oximetry report that they currently send blood samples to other laboratories for COHb measurement, the attendant delay appears to affect timeliness of diagnosis and management. In the same study, over 90% of CO-poisoned patients referred to a regional hyperbaric oxygen treatment facility came from hospitals able to measure COHb. Since hyperbaric treatment is

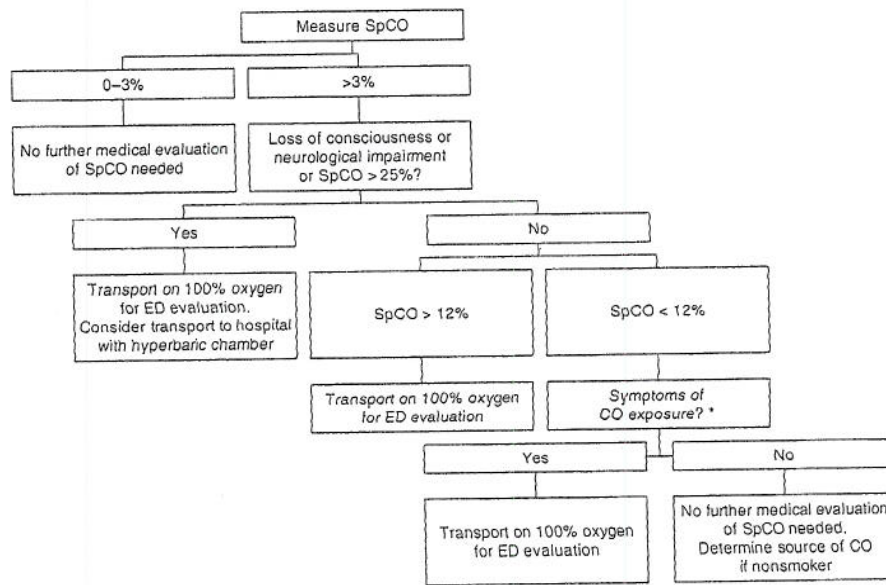


FIGURE 33.3 Algorithm for individuals possibly exposed to carbon monoxide, on the basis of pulse CO-oximetry SpCO measurement (From: Hampson, N.B., Weaver, L.K. Noninvasive CO measurement by first responders: A suggested management algorithm. *J. Emerg. Med. Serv.* 2006, 24(suppl.): 10–12.

* Common symptoms of CO exposure include nausea, vomiting, headache, dizziness, weakness, and loss of consciousness.

felt to be more effective when administered early,¹⁹ rapid identification of poisoned individuals is of great importance.

33.3 CONCLUSION

The new pulse CO-oximeter represents a major advance in field and ED screening of individuals for CO exposure and poisoning. Because many of these will initially be discovered to have an elevated SpCO level by first-responders, it is important that triage and management protocols be available. As use of the device increases in all venues, the number of individuals diagnosed with CO poisoning each year is likely to increase dramatically.

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