

Use of a Noninvasive Pulse CO-Oximeter to Measure Blood Carboxyhemoglobin Levels in Bingo Players

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BACKGROUND: Though smokers are known to have elevated blood carboxyhemoglobin (COHb), due to inhalation of carbon monoxide (CO) in cigarette smoke, limited data exist regarding COHb levels in nonsmokers exposed to secondhand smoke. **METHODS:** COHb was measured using a new noninvasive pulse CO-oximeter (Rad-57, Masimo, Irvine, California) in 38 subjects entering a bingo hall where smoking was allowed, then again as they exited 3 hours later. **RESULTS:** The mean \pm SD baseline COHb for the entire group was $3.3 \pm 1.8\%$, for the 23 nonsmokers it was $2.2 \pm 0.7\%$, and for the 15 smokers it was $4.9 \pm 1.9\%$. The nonsmokers' mean COHb was unchanged, at $2.2 \pm 0.8\%$, whereas the smokers' mean COHb fell to $3.2 \pm 1.9\%$. **CONCLUSIONS:** The nonsmokers were not significantly exposed to CO from secondhand smoke in the setting we tested. The smokers probably consumed fewer cigarettes while playing bingo than they did prior to arrival. The Rad-57 pulse CO-oximeter is easy to use in the ambulatory setting. *Key words:* CO-oximetry; carboxyhemoglobin; carbon monoxide; smoking; pulse oximetry; monitoring, noninvasive. [Respir Care 2006;51(7):758–760. © 2006 Daedalus Enterprises]

Introduction

Carbon monoxide (CO) is an odorless, colorless, toxic gas produced as a by-product of burning carbon fuel. Almost all burning produces some CO; the amount depends on the material burned and the efficiency of the combustion. When inhaled, CO binds to hemoglobin in red blood cells passing through the lungs, forming carboxyhemoglobin (COHb). Because CO binds to hemoglobin much more tightly than does oxygen, and CO occupies the sites normally used to bind and carry oxygen from the lungs to the tissues, one mechanism of CO toxicity is that it decreases the oxygen content of arterial blood, which reduces peripheral oxygen delivery.¹

Since CO binds so avidly to hemoglobin, COHb remains in the circulation for hours and is thus a marker of

recent exposure to CO. Average COHb levels are different in smokers and nonsmokers, because smokers regularly inhale CO with cigarette smoke. The average COHb level in nonsmokers is $< 1\%$, whereas the average level in smokers is about 4% (Table 1).² There is obviously a range of values among individuals in each category; some have higher levels and some lower than the average. To look at it a different way, 90% of nonsmokers have a COHb of $< 1.33\%$ and 90% of smokers have a COHb of $< 7.56\%$ (see Table 1). Of the 2% of smokers whose COHb exceeds 10%, COHb has been reported as high as 15–20% immediately after smoking.^{3–5}

While it is well known that cigarette smokers have elevated COHb, there are only limited data on whether nonsmokers exposed to secondhand cigarette smoke experience increases in COHb.⁶ The likely reason for such sparse information is that measuring COHb has, until recently, required obtaining a blood sample and using a laboratory CO-oximeter. The development of a new pulse CO-oximeter (Rad-57, Masimo, Irvine, California) has made measuring COHb rapid, noninvasive, and readily available at any location. The Rad-57 measures blood COHb in addition to conventional pulse oximetry variables.⁷ To examine the CO effect of secondhand smoke on nonsmokers, we used the Rad-57 to measure COHb in individuals exposed while playing bingo in a smoky environment.

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Table 1. Carboxyhemoglobin Levels in Persons 3–74 Years of Age in the United States

Smoking Status	Percent COHb (mean \pm SD)	Percent COHb (75th Percentile)	Percent COHb (90th Percentile)
Never smokers	0.83 \pm 0.67	0.97	1.33
Current smokers	4.30 \pm 2.55	5.89	7.56
All smoking statuses combined	1.94 \pm 2.24	2.38	5.49

COHb = carboxyhemoglobin
(Data from Reference 2.)

Methods

The subjects were recruited for study participation as they entered a community center on a Saturday night to play bingo. Cigarette smoking was allowed within the building. Smokers sat on one side of the room and nonsmokers on the other. Windows and doors were left ajar to provide ventilation.

Blood COHb levels were measured, with the Rad-57, at the time of entry and again upon exit, following the 3-hour bingo session. The smoking status of each individual was initially determined by participant self-report and was subject to reporting inaccuracy.

Data were first analyzed for the group as a whole, and then in subgroups, according to reported or suspected smoking status. Since the normal nonsmoker mean COHb level plus 2 standard deviations is 2.17% (see Table 1) and the Rad-57 displays COHb measurements in whole percentages, those with COHb $>$ 3% were classified as probable smokers. Therefore, one subgroup comprised those individuals who reported being current smokers plus those who reported being nonsmokers but who presented with elevated COHb ($>$ 3%). The other subgroup included those who reported nonsmoker status and whose baseline COHb was $<$ 3%. Statistical analysis was performed using a 1-tailed or 2-tailed paired *t* test and the Wilcoxon signed rank test.

The institutional review board of Virginia Mason Medical Center approved the study, as did the board of directors of the fraternal organization that operates the bingo hall.

Results

Thirty-eight subjects were recruited from the approximately 40 individuals present in the bingo hall that evening. Of these, 7 were self-reported smokers with baseline COHb of 2–9% and mean \pm SD COHb of 5.4 \pm 2.6%. Of the 31 self-reported nonsmokers, 8 had elevated baseline COHb: range 4–6%, mean \pm SD 4.4 \pm 0.7%, which is not statistically different than the self-reported smokers ($p = 0.382$). These 15 individuals were combined to form the subgroup of (probable) “smokers.” The remaining 23 subjects composed the subgroup of (probable) “nonsmokers.”

Summary results for the whole population and the 2 subgroups are shown in Table 2. The average baseline COHb for the entire group was 3.3 \pm 1.8%. Smokers had significantly

Table 2. Carboxyhemoglobin Levels in Recruited Bingo Players

	Carboxyhemoglobin (%)		
	Entire Group (<i>n</i> = 38)	Nonsmokers (<i>n</i> = 23)	Smokers (<i>n</i> = 15)*
Before Bingo	3.3 \pm 1.8	2.2 \pm 0.7	4.9 \pm 1.9
After Bingo	2.6 \pm 1.4	2.2 \pm 0.8	3.2 \pm 1.9

*The smokers group includes 7 self-reported smokers and 8 self-reported nonsmokers who had elevated baseline carboxyhemoglobin.

higher baseline COHb than did nonsmokers ($p < 0.0001$). Secondhand smoke was clearly visible in the room by the end of the evening, and numerous individuals complained of eye irritation from it. Despite this, COHb levels remained stable among the nonsmokers and decreased among the smokers over the course of the evening (see Table 2). Because the average COHb changes for the groups were less than the precision of the measurement device (3%), it was not possible to assess the statistical significance of the quantitative change. Therefore, the Wilcoxon signed-rank test was used to assess qualitative changes from baseline. The trend among smokers was significant, as 13 of 15 had lower COHb after bingo ($p = 0.006$) (Fig. 1). No significant pattern of change was seen among the nonsmokers ($p = 0.965$).

Discussion

Until recently, measuring an individual’s COHb level required drawing a blood sample and measuring it in a laboratory with a CO-oximeter or estimating it from measured CO in exhaled breath.⁸ Laboratory CO-oximeters use multiple wavelengths to distinguish the various forms of hemoglobin (oxyhemoglobin, deoxyhemoglobin, carboxyhemoglobin, and methemoglobin). Conventional 2-wavelength pulse oximeters are incapable of measuring COHb.⁹ The Rad-57, which has been approved by the Food and Drug Administration, uses 8 wavelengths of light to noninvasively and rapidly (within seconds) measure COHb, in addition to the regular pulse-oximetry-measured oxygen saturation (S_{pO_2}) and heart rate. Pulse-CO-oximetry-measured COHb has been termed “ S_{pCO} .” The Rad-57 is accurate with S_{pCO} values up

PULSE CO-OXIMETER TO MEASURE BLOOD CARBOXYHEMOGLOBIN

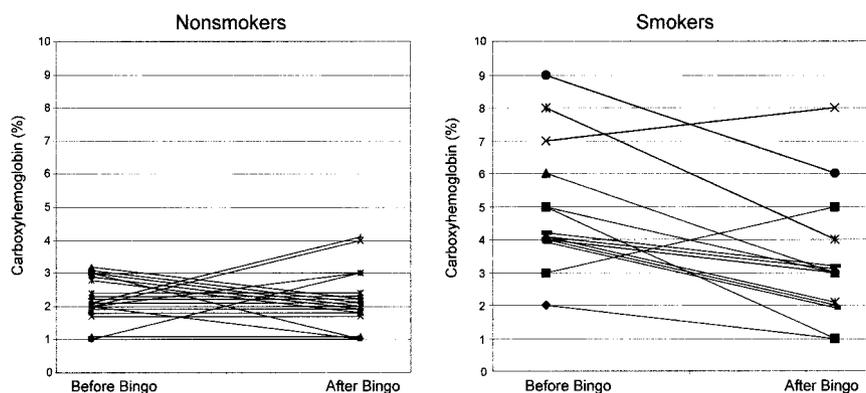


Fig. 1. Carboxyhemoglobin levels in 23 nonsmokers (left) and 15 smokers (right) before and after exposure to secondhand smoke at a bingo game.

to 40%, with an accuracy range of $\pm 3\%$ around the measurement.⁷ The device is the same size as a conventional handheld pulse oximeter, is battery-powered, and uses a fingertip probe for measurement.

In the present study, it does not appear that secondhand cigarette smoke in the setting tested posed a significant risk for CO exposure, despite the fact that several nonsmokers complained about smoke exposure when leaving the bingo hall at the end of the evening. Surprisingly, COHb levels declined in the subgroup of smokers over the course of the evening, despite the fact that they were allowed to smoke without scrutiny. This may have occurred because they were occupied by the game and consumed fewer cigarettes during the 3-hour period than they would have otherwise.

It should be noted that our group of “probable” smokers included 7 self-reported smokers and 8 self-reported nonsmokers, who had similar COHb elevations. Though they were suspected to be nonreporting smokers, it is also possible that they were exposed to another source of CO before arriving, such as a malfunctioning home furnace, faulty automobile exhaust system, or a high level of secondhand smoke from a spouse. In that case, their COHb levels may have declined in the bingo hall because of lower ambient CO exposure. All subjects were provided their measured COHb levels in writing and advised to seek further evaluation if their COHb was unexpectedly elevated.

Biomarkers such as expired CO or urinary total cotinine have been suggested by others as more meaningful markers of cigarette consumption than patient recall and report.¹⁰ However, the utility of such markers has been thought to be limited by the requirement to obtain blood or urine specimens, the expense, and/or the delay in result availability. The Rad-57 pulse CO-oximeter has potential clinical application for this purpose, especially if future studies show that S_{pCO} measurements correlate well with documented cigarette consumption.

To our knowledge, this is the first study that has measured the effect of secondhand cigarette smoke on COHb

levels in nonsmokers in a real-life situation. The only other study we are aware of exposed experimental subjects to secondhand smoke in a small confined room as part of a coronary-blood-flow study.⁶ Passive smoking caused a small increase in average COHb (mean difference 1.3%).

Conclusion

Despite the fact that COHb levels in the nonsmokers did not change in our study, secondhand smoke exposure should still be avoided because of other health risks that it carries.¹¹

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