

# Carbon Monoxide Poisoning and SpO<sub>2</sub>: A Case Report

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## Abstract

A six year old patient undergoing surgery as the first case on a Monday morning had an unexplained fall in SpO<sub>2</sub>. The cause was found to be carbon monoxide toxicity due to the degradation of the volatile anesthetic by desiccated carbon dioxide absorbent. The diagnosis was confirmed by co-oximetry analysis of an arterial blood sample.

## INTRODUCTION

The potential for carbon monoxide (CO) toxicity due to the degradation of volatile anesthetics by desiccated carbon dioxide absorbents is well known. Diagnosis of that entity is difficult unless it is suspected based on knowledge of the potential problem. We present a case of severe intraoperative CO poisoning where the diagnosis was suggested only by a moderate decrease in SpO<sub>2</sub>.

## CASE REPORT

A 2yr old girl, weighing 16 kilograms; ASA physical status I, was anesthetized for an oral surgery procedure as the first case on a monday morning. Following mask induction with sevoflurane/nitrous/oxygen, a peripheral IV was placed. Neuromuscular blockade was established and the patient's trachea easily intubated. Maintenance of anesthesia was started with desflurane 8% and oxygen. Ten minutes after intubation, the patient's blood pressure decreased from a baseline of 90/35 to 65/15 mm/hg. Concurrently the SpO<sub>2</sub> decreased from 100% to 98%. The blood pressure responded immediately to 10mcgs of phenylephrine IV and the desflurane level was reduced, however the SpO<sub>2</sub> continued to fall to 89%. An immediate exploration for the cause failed to identify an obvious etiology. An arterial blood gas was drawn; results were a pH 7.43, PaCO<sub>2</sub> 31 and PaO<sub>2</sub> 498. Being aware of the potential for carbon monoxide production, a blood sample for co-oximetry evaluation had also been sent to the lab. The carboxyhemoglobin was 63%. The inhalational anesthetic was immediately changed to sevoflurane and CO<sub>2</sub> absorbent replaced with fresh Baralyme. The patient's SpO<sub>2</sub> gradually increased to 100%. Forty-five minutes after replacement of the Baralyme, an arterial sample was drawn for co-oximetry evaluation. This

had a carboxyhemoglobin of 42%. After another forty-five minutes, the carboxyhemoglobin was 23%. Forty-five minutes later the co-oximetry value for carboxyhemoglobin (COHb) was 6%.

The duration of anesthesia was 180 minutes. The neuromuscular blockade was reversed and the patient emerged from anesthesia with no apparent sequelae. The patient remained in the Post Anesthesia Care Unit with routine monitoring for more than 2 hours. While further co-oximetry samples were not taken, the patient was able to maintain a SpO<sub>2</sub> of 100% on room air for more than an hour after discontinuing supplemental oxygen. She was discharged home after her mother was informed and had received instructions regarding the carbon monoxide exposure during anesthesia. The mother was called the day after surgery and again four weeks later. She reported no abnormalities in the child's behavior or activity. Motor skills and her behavior were reported unchanged from prior to surgery.

## DISCUSSION

Carbon monoxide (CO) production by the degradation of volatile anesthetics was initially reported by Fang and Eger in 1994 in the Anesthesia Patient Safety Foundation newsletter.<sup>(1)</sup> Case reports followed.<sup>(2)</sup> The awareness of this potential problem has reduced the incidence or at least the reported incidence of similar cases. However, knowledge of a potential problem and the ability to diagnose the problem may not be analogous. During our department's morbidity and mortality conference, the audience, was almost unanimous in thinking that CO was not the cause. The majority felt that CO does not have a significant effect on the pulse oximetry reading. <sup>(3,4)</sup> One of the most severe case

of CO poisoning previously reported (COHb 36%) was diagnosed mainly due to an erroneous gas analyzer readings.<sup>(2)</sup> In our case, the diagnosis of CO poisoning was facilitated by a clinically significant decrease in SpO<sub>2</sub>. This is consistent with animal studies done by Barker and Tremper. Their work showed that SpO<sub>2</sub> decreased with high COHb concentrations. A COHb concentration of 70% produced a SpO<sub>2</sub> of 90%, ( $\text{SpO}_2 = 99.3 - (\text{COHb} \times 0.1)$ ).<sup>(3)</sup> This has also been noted by others.<sup>(4,6,7,8)</sup>

The reason for this can be explained by the difference between pulse oximetry and co-oximetry. Both function based on the principle of differential light absorbance. Pulse oximetry employs two wavelengths of light, 940 nm and 660 nm. Oxyhemoglobin predominantly absorbs light at 940 nm and reduced hemoglobin predominantly absorbs light at 660 nm. Using the relation of those differences, an algorithm estimates the oxygen saturation. The presence of carboxyhemoglobin can interfere with that analysis. Carboxyhemoglobin (COHb) also absorbs light at 660 nm but does not absorb at 940 nm. Meanwhile a co-oximeter measures light absorbance at six or more discrete wavelengths. Using these data and the known absorbance spectra of the various hemoglobin species, the co-oximeter calculates the concentration of each species and reports the percentages of reduced, oxygenated, carboxyhemoglobin and methemoglobin.

While knowledge has certainly minimized intraoperative carbon monoxide poisoning, it is still a potential problem. The pulse oximetry literature contains the information necessary to deduce the diagnosis from the clinical signs and situation, leading to obtaining appropriate blood analysis for confirmation. However, most of the literature on the effect of carboxyhemoglobin was published before the case reports of the "first-case Monday-morning syndrome". The pulse oximeter's accuracy is certainly affected by the presence of

carbon monoxide. This effect is only minimal until the carboxyhemoglobin concentration is well above a toxic level.

### CONCLUSION

In conclusion, even though the pulse oximeter overestimates arterial Hemoglobin saturation, and hence is not a reliable monitor to assess oxygenation of patients with CO poisoning, one should definitely include CO poisoning in the differential diagnosis of decreasing intraoperative SpO<sub>2</sub>.

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