CASE REPORTS

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Severe Carbon Monoxide Poisoning during Desflurane Anesthesia

Peter D. Berry, M.B., Ch.B., Daniel I. Sessler, M.D.,† Merlin D. Larson, M.D.‡*

AFTER reports of carbon monoxide production within anesthetic breathing circuits,§[1–3] Fang *et al.*[4] have identified degradation of volatile anesthetics by desiccated carbon dioxide absorbents as the source of carbon monoxide. The possibility of severe intraoperative carbon monoxide toxicity was highlighted by Frink *et al.*[5] who demonstrated extremely high blood carboxyhemoglobin (COHb) concentrations in an animal model using dry Baralyme with desflurane. The danger of intraoperative carbon monoxide exposure is increased by its indistinct clinical features in the anesthetized patient. We present the most severe case of intraoperative carbon monoxide exposure yet reported, in which the diagnosis was suggested by a combination of moderately decreased oxygen saturation by pulse oximetry (Sp_{O_2}) and an erroneous gas analyzer reading.

This case report is accompanied by an Editorial View. Please see: Woehlck HJ: Severe intraoperative CO poisoning: Should apathy prevail? ANESTHESIOLOGY 1999; 90:353–4.

* Visiting Assistant Professor, Department of Anesthesia and Perioperative Care, University of California, San Francisco.

† Professor, Department of Anesthesia and Perioperative Care, University of California, San Francisco; Professor, Ludwig Boltzmann Institute for Clinical Anesthesia and Intensive Care; Director Outcomes Research™ and Vice-Chair, Department of Anesthesia and General Intensive Care, University of Vienna.

‡ Professor, Department of Anesthesia and Perioperative Care, University of California, San Francisco.

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Address correspondence to Dr. Sessler: Department of Anesthesia and Perioperative Care, University of California, San Francisco, 374 Parnassus Avenue, 3rd Floor, San Francisco, California 94143-0648. Address electronic mail to: sessler@vaxine.ucsf.edu. On the World Wide Web: www.outcomes.ucsf.edu

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§ Lentz RE: CO poisoning during anesthesia poses puzzles. APSF Newsletter 1994; 9:13–24

Case Report

A 24-yr-old woman, ASA physical status 1, was anesthetized for a clinical research study that involved combined epidural and general anesthesia. The subject's weight was 62 kg; height was 1.66 m; hematocrit level was not measured. She had undergone an identical general anesthetic 2 weeks previously as part of the same study, with no alteration of Sp_{O_2} or other complications. After insertion of an intravenous cannula, 3 ml lidocaine, 1.5%, was infiltrated into the T10–T11 interspace, and an epidural catheter was positioned uneventfully using an 18-gauge Tuohy needle. Preoperative Sp_{O_2} was 99% (Nellcor Puritan Bennet, Pleasanton, CA).

The subject was preoxygenated with a fresh gas flow of 8 l/min oxygen *via* the anesthetic machine circle breathing system (Ohmeda 8000 Anesthesia System; Datex-Ohmeda, Helsinki, Finland). General anesthesia was induced with 180 mg intravenous propofol, followed by 10 mg intravenous vecuronium. She was then mask ventilated with desflurane in oxygen at a tidal volume near 15 ml/kg at approximately 12 breaths/min for approximately 3 min. The Ohmeda Tec 6 desflurane vaporizer (Datex-Ohmeda) was set between 6 and 10% to rapidly achieve a target end-tidal desflurane concentration of 5%. The trachea was then intubated without difficulty. The lungs were mechanically ventilated with a minute volume of 7 l/min, a fresh gas flow of 2 l/min with 100% oxygen, and a desflurane vaporizer setting of 6%. Five minutes after induction of anesthesia, Sp_{O_2} decreased to 93%. Bilateral auscultation of the lungs was normal, endotracheal suction returned no secretions; end-tidal pressure of carbon dioxide (P_{CO_2}) was 34–35 mmHg. Heart rate was 96 beats/min, and blood pressure was 110/60 mmHg, both similar to preinduction levels. The three-lead electrocardiogram tracing showed normal rhythm and QRS- and ST-segment appearance. Automated ST-segment analysis was not available on the monitor used. Clinical appearance of the subject was entirely normal, with no cyanosis and no "cherry red" appearance. Ten minutes after induction of anesthesia, the Datex Capnomac Ultima end-tidal gas analyzer (Datex-Ohmeda)—set in "automatic" mode—indicated the presence of enflurane followed after a few minutes by "mixed agent." Until this point, it indicated desflurane. No enflurane vaporizer was attached to the anesthetic machine. At this point, carbon monoxide toxicity because of desiccation of the carbon dioxide absorbent was suspected, and the Baralyme (Allied Healthcare Products, St Louis, MO) was therefore immediately replaced with fresh Baralyme. The time interval from induction of anesthesia to replacement of the Baralyme was approximately 15 min. After an additional 15 min, an arterial blood sample was obtained for cooximeter analysis with the following results: oxygenated hemoglobin (HbO_2): 63%; COHb: 36%; and methemoglobin (MetHb): 1%. The study

protocol was aborted and the subject was ventilated with 100% oxygen using a fresh gas flow of 8 l/min. Anesthesia was maintained with a desflurane vaporizer setting of 6%. Twenty minutes after replacement of the Baralyme, the anesthetic agent analyzer no longer indicated mixed agent, and instead indicated an end-tidal desflurane concentration of 5–5.5%, which was consistent with the vaporizer setting. The Sp_{O_2} returned to 99%. Cooximetry was repeated 45 min after the original sample, and COHb concentration was 21%. After an additional hour, COHb concentration decreased to 12%. Neuromuscular blockade was then antagonized, the anesthetic was discontinued, and the subject emerged from anesthesia with no apparent abnormal sequelae. The total duration of anesthesia was approximately 140 min. The subject remained in the postanesthesia care unit with routine monitoring for 2 h and was then discharged to home. Before discharge, she was informed about her carbon monoxide exposure.

She was telephoned 2 weeks later and interviewed in person 6 months later. She reported no adverse sequelae of the event. Specifically, there were no subjective alterations in her emotional state or motor control. Her occupation required typing expertise, and this was reportedly unchanged. She thought that her recovery had been no different after carbon monoxide exposure than after her similar uncomplicated anesthetic 2 weeks previously. Formal neuropsychologic testing was not performed.

Inquiry at the time of the anesthetic and subsequently revealed that (despite the appearance of recent use) the anesthetic machine had not been used for several days and had probably been left switched on and connected to the oxygen pipeline for this entire period. It was not possible to establish the fresh gas flow during this period of disuse, nor the exact configuration of the circuit. The room used for the study was located within the operating room suite, and therefore not in a "remote location"; it was, however, not used for surgical cases and was used only infrequently for other anesthetic purposes.

Discussion

In vitro studies have shown that carbon monoxide is produced when desiccated carbon dioxide absorbent reacts with volatile anesthetic agents.[4,6] Animal studies have shown that this reaction can occur within anesthetic breathing circuits[5] and can cause extremely high blood COHb concentrations. Reports of elevated COHb concentrations detected intraoperatively in humans imply that significant carbon monoxide toxicity might occur in patients *via* this mechanism.§[1–3] The COHb concentrations in these reports range from 7 to 32%. The COHb concentration of 36% that we report in this case is therefore the most severe yet reported. The precise mechanism by which carbon monoxide causes tissue injury is not known. However, the best-understood and most-popular explanations are (1) occupation of oxygen binding sites on hemoglobin and leftward shift of the oxygen–hemoglobin dissociation curve, with consequent reduction in tissue oxygen delivery, and (2) inhibition of cytochrome oxidase by carbon monoxide, leading to impaired energy metabolism and free-radical production.[7]

The clinical features of carbon monoxide toxicity after smoke inhalation correlate poorly with COHb levels; nevertheless, a COHb level of 36% would generally be associated with severe headache, nausea, vomiting, and syncope; levels greater than 50% are usually associated with coma and convulsions.[8] In patients with coronary artery disease, however, COHb levels as low as 2.9–4.5% can exacerbate myocardial ischemia.[9,10] Similarly, smoke inhalation with relatively mild carbon monoxide exposure (COHb levels <30%) may produce various neuropsychologic abnormalities 3–21 days after exposure.[11] Complex batteries of tests have been used to detect abnormalities after smoke inhalation. However, no comparable data exist for patients exposed to carbon monoxide during anesthesia. In common with previously reported lesser exposures, our subject had no immediate overt adverse consequences. She also denied having abnormalities at 2 weeks and 6 months afterward. Formal neuropsychologic testing, however, would have had greater power to detect subtle abnormalities.

Production of carbon monoxide within breathing circuits occurs when desiccated carbon dioxide absorbent comes into contact with and degrades volatile anesthetics. Production is greatest with desflurane, isoflurane, and enflurane; the most probable source of carbon monoxide is the $-CHF_2$ moiety, which is missing on halothane and sevoflurane. The amount of carbon monoxide produced within a breathing circuit also depends on other factors: the type of absorbent and the degree of desiccation, the temperature, the concentration of volatile agent, and the fresh gas flow used. For instance, at a given water content, Baralyme produces more carbon monoxide than does soda lime. The concentration of carbon monoxide within the circuit also varies with time, tending to peak in the first 30 min.[4,5,12] Furthermore, blood COHb concentrations will be affected by fractional inspired oxygen tension (Fi_{O_2}) because of competitive binding of oxygen to hemoglobin.[13] Thus, production of carbon monoxide and carbon monoxide toxicity is to some degree predictable, although many factors are involved.

Diagnosis of carbon monoxide intoxication during anesthesia is difficult because the main clinical features of toxicity are masked by anesthesia. Furthermore, there is no routinely available means to reliably identify the presence of carbon monoxide within the breathing circuit, nor to detect when carbon dioxide absorbent has been desiccated. In our case, early diagnosis of carbon monoxide production was facilitated by the presence of a clearly erroneous gas analyzer reading (it is not possible to accidentally fill a Tec 6 desflurane vaporizer with enflurane). This false reading probably resulted from trifluoromethane,

which is produced along with carbon monoxide by volatile-agent degradation. Because it has an infrared absorption profile similar to enflurane, it is identified as such by some gas analyzers, including the Datex Capnomac Ultima.[6] Our case, therefore, confirms the potential role of unexpected gas analyzer readings in the clinical detection of carbon monoxide production. Many gas analyzers, however, do not show clearly erroneous readings,[6] which suggests that the routine incorporation of a carbon monoxide detection module into gas analyzers may be justified.

A second important diagnostic feature of our case was the observed moderate decrease in Sp_{O_2} without other identifiable causes. It is widely believed that Sp_{O_2} remains unchanged during carbon monoxide toxicity, with COHb being detected as HbO_2 by most pulse oximeters.[14,15] Our case, however, suggests that significant COHb concentrations may moderately decrease Sp_{O_2}. This is consistent with animal studies in which Sp_{O_2} decreased with high COHb concentrations,[16] with a COHb concentration of 70% producing a Sp_{O_2} of 90%.

Interestingly, in a recent case report,[1] an unexpectedly rapid change of soda lime color to blue after induction of anesthesia was associated with desiccation and carbon monoxide production. This may imply that desiccated absorbent has less capacity to absorb carbon dioxide and, therefore, may reveal its presence by becoming exhausted and changing color more rapidly than expected.

Because of the difficulty of detecting of carbon monoxide production and toxicity, prevention is especially important. Various guidelines have been published for prevention of carbon monoxide production; most recently these have concentrated on preventing the use of desiccated carbon dioxide absorbent.‖[2] Baralyme and soda lime both are supplied wet, that is, they contain approximately 13–15% water by weight. The percentage of water that would prevent carbon monoxide production for all anesthetics is probably near 4.8% for soda lime and 9.7% for Baralyme.[4] A fresh gas flow of 5 l/min or more passed through absorbent for 24 h (without a patient) is sufficient to cause critical drying of the absorbent if the reservoir bag is left off the breathing circuit (thus facilitating retrograde movement of gas through the absorber). With the bag in place, drying still occurs, but to a lesser extent. These findings are consistent with the observation in several reports,[1–3] including ours, that carbon monoxide production occurred when anesthetic machines had been unused for 48 h or more. In contrast, it is unlikely that either high- or low-flow anesthesia itself can cause desiccation and carbon monoxide exposure,[17] because water is released as carbon dioxide is absorbed. The Food and Drug Administration has recommended that, where desiccation is suspected, on the basis of a high fresh gas flow in an unused machine over a prolonged period, the carbon dioxide absorbent should be changed.‖ Woehlck *et al.*[2] confirm the success of simple measures, *i.e.*, instructions to technicians and other staff aimed at preventing the use of desiccated absorbent, which were able to reduce (although not completely prevent) intraoperative carbon monoxide exposure in patients. At our institution, this case led to a procedure change, whereby the carbon dioxide absorbent is changed if the anesthetic machine is found with the fresh gas flow on at the beginning of a day. In addition, soda lime has been substituted for Baralyme because it is less likely to produce significant levels of carbon monoxide.#[4]

Our case shows several points regarding the management of intraoperative carbon monoxide exposure. The carbon dioxide absorbent was changed immediately to prevent further carbon monoxide production, and a relatively high fresh gas flow was used subsequently to promote washout of carbon monoxide from the breathing circuit. Furthermore, the subject was ventilated with 100% oxygen to reduce the half-life of COHb. Anesthesia and mechanical ventilation was maintained until the COHb level had decreased to a level not normally associated with overt toxicity in healthy individuals: this was done to facilitate delivery of a high Fi_{O_2}, and to avoid the cardiovascular stimulation of emergence and tracheal extubation occurring against the background of a high COHb level.

In conclusion, we report a case of severe intraoperative carbon monoxide exposure. This case illustrates the possibility of desiccated carbon dioxide absorbent reacting with desflurane to cause significant carbon monoxide exposure in the clinical setting. The presence of an unexpectedly low Sp_{O_2} and an erroneous gas analyzer reading led to the diagnosis in this case. Nevertheless, the diagnosis of intraoperative carbon monoxide exposure is difficult because specific monitoring for carbon monoxide is not routinely available and the clinical features are vague. It is therefore possible that a significant number of undiagnosed carbon monoxide exposures occur. A high index of suspicion, awareness of possible diagnostic feature, and institution of measures to prevent carbon dioxide absorbent desiccation may help to prevent future exposures.

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Intraoperative Burns Secondary to Warmed IV Bags: A Warning

Lorne K. Rosenfield, M.D., Paul J. Pitlyk, M.D.†*

IN positioning for cervical spinal surgery, it is common to place a bolstering device to help support and position the operative site, specifically the anterior aspect of the neck. Ordinarily a 10-lb leather-bound sandbag or a liter bag of intravenous fluid is used for this purpose. During a lengthy surgery, warmed intravenous fluid bags also have been used to treat hypothermia. The purpose of the following two case reports is to promulgate a strong awareness of a hazard in using warmed intravenous fluid bags in such circumstances.

* Chief, Department of Plastic Surgery.

† Assistant Clinical Professor of Neurosurgery, University of California, San Francisco.

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Address reprint requests to Dr. Rosenfield: Department of Plastic Surgery, Mills-Peninsula Hospital, Burlingame, California, 1750 El Camino Real, Suite 405, Burlingame, California 94010.

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Case Reports

Case 1

A 53-yr-old man was taken to surgery on January 6, 1996 for resection of cervical osteophytes *via* the anterior approach. The patient was placed in the supine position, and a liter bag of intravenous fluid was used to position his head in an extended posture. The intravenous fluid bag was obtained from the blanket warmer (set at 114°F) for patient comfort. It was wrapped in a towel and placed between the scapulae