Another perspective on ACEP policy on critical issues in carbon monoxide poisoning: *Invited commentary*

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The American College of Emergency Physicians (ACEP) recently published its official policy on the evaluation and management of patients with acute carbon monoxide (CO) poisoning [1], *an update of the policy previously published in 2008.*

Three questions regarding CO poisoning were posed to a 30-person ACEP subcommittee, which employed a comprehensive literature review to attempt to draw conclusions.

While this is a major step forward, we feel that their conclusions warrant comment.

ACEP Question 1

Can pulse CO-oximetry be used in the emergency department (ED) to accurately diagnose [carbon monoxide] CO toxicity?

ACEP Final Recommendation

"Do not use pulse CO-oximetry to diagnose CO toxicity in patients with suspected acute CO poisoning."

Our Comment

The ACEP committee based this recommendation on detailed analysis of five clinical studies of diverse size, methods and results. Its opinion appears related to the variability that has been demonstrated between simultaneous pulse and laboratory CO-oximetry carboxyhemoglobin measurements (SpCO and COHb, respectively).

However, it was not possible for the committee to actually answer the question posed. A pulse CO-oximeter is not designed to measure CO toxicity. Toxicity represents the adverse effects of a poison. In CO poisoning, it is assessed many ways, including level of consciousness, hemodynamic parameters, neurological status, or by measuring blood correlates of tissue toxicity such as lactate or arterial pH. COHb is a marker of CO exposure, not toxicity. A soaking wet person may show evidence of having been in water, but that finding does not diagnose drowning.

The pulse CO-oximeter also cannot diagnose CO poisoning. The diagnosis of CO poisoning is a clinical one, based upon a confirmed exposure to CO, an elevated COHb level if measured shortly after exposure, and a consistent clinical presentation.

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A pulse CO-oximeter does have the potential to diagnose recent CO exposure. After CO inhalation, the COHb level rises, then slowly decreases, returning to normal by 24 hours or less. This provides the opportunity to identify those who have been exposed.

To answer the question of whether pulse CO-oximeters can accurately diagnose CO exposure in ED patients with suspected CO poisoning, one would need to identify such a group

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and determine how often their SpCO level was elevated above normal when laboratory COHb was simultaneously elevated.

In one small study where the data are expressed in that fashion [2], ten (10) patients with suspected CO poisoning demonstrated laboratory COHb levels above 10%, nine of whom had SpCO levels over 10%. While this suggests a possible application, the manufacturer's website cautions:
"SpCO with Masimo devices is not intended to replace laboratory blood testing. Blood samples should be analyzed by laboratory instruments prior to clinical decision making." Indeed, case reports and series have revealed that when compared with blood COHb measurement, pulse CO-oximetry readings (SpCO) can exhibit both false negatives [3] and false positives [4,5]. A prospective study of 1,363 emergency patients found a wide range of SpCO values compared with COHb levels [6].

The pulse CO-oximeter cannot diagnose CO poisoning.

Our Recommendation

When the SpCO is elevated, venous COHb should be measured for confirmation. A low SpCO value should not exclude possible CO poisoning if clinical suspicion exists.

ACEP Question 2

Does hyperbaric oxygen therapy, as compared to normobaric oxygen therapy, improve long-term neurocognitive outcomes?

ACEP Recommendation

"Emergency physicians should use HBO_2 therapy or high-flow normobaric therapy for acute CO-poisoned patients. It remains unclear whether HBO_2 therapy is superior to normobaric oxygen (NBO_2) therapy for improving long-term neurocognitive outcomes."

Our Comment

The ACEP committee based its recommendation on the re-review of several randomized trials that have been the subject of two Cochrane reviews and discussed at length in the literature over the past two decades. A few observations are warranted.

First, hyperbaric vs. normobaric oxygen trials in CO poisoning have used widely divergent protocols. None of those which utilized a maximum treatment pressure of 2.0 ATA have demonstrated a positive benefit of HBO_2. However, some protocols performed at 2.5-3.0 ATA have been positive. It would appear that 2.0 ATA may not provide an adequate dose of HBO_2 for CO poisoning, and these studies should be considered for exclusion from future meta-analysis.

Second, the committee again failed to answer the question posed. All randomized studies except one have used only short-term outcomes (two to six weeks) as endpoints, and some have had low follow-up rates [7]. It has been shown that neurological dysfunction from CO poisoning can continue to improve for three to 12 months [8], so long-term outcomes are very appropriate to assess. Only the Weaver trial measured long-term outcomes

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(one year) [9]. As such, the report by Weaver, et al. should be the sole basis for the answer to this question. In it, neurocognitive outcomes were significantly better in the hyperbaric group at 12 months.

Finally, the ACEP policy in this area is noncommittal, recommending only that practitioners use HBO_2 or NBO_2 oxygen for CO poisoning. This is not much guidance for the physician on the front line at 3:00 in the morning. Perhaps the committee's unwillingness to make a stronger statement relates to lack of experience in the field. A Pub Med search reveals only four publications on CO poisoning among the 30 members, besides the clinical policy itself.

In 2012, a consensus expert opinion paper on the clinical aspects of CO poisoning reviewed the same literature as the ACEP [10]. Written by four CO experts with over 150 combined publications on CO poisoning, the authors recommended consideration of HBO_2 in all cases of acute, symptomatic CO poisoning. The same year, the Chinese Medical Association published a national guideline that also analyzed all randomized trials in the area, recommending consideration of HBO_2 in all cases of acute CO poisoning as soon as possible [11].

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Our recommendation

Until better information is available to guide patient selection, clinical management should be guided by the best information available. The Weaver study is most similar to clinical practice and provides evidence that HBO_2 reduces long-term neurocognitive sequelae. HBO_2 should at least be considered for all patients with acute, symptomatic CO poisoning.

ACEP Question 3

Can cardiac testing in the ED predict morbidity and mortality?

ACEP Recommendation

"In ED patients with moderate to severe CO poisoning, obtain an ECG and cardiac biomarker levels to identify acute myocardial injury, which can predict poor outcome."

Our Comment

The ACEP guideline reviews studies demonstrating poorer short- and long-term outcomes when a CO-poisoned patient suffers cardiac injury with poisoning, as compared to one who does not. It should come as no surprise that having heart disease worsens one's prognosis and longevity. It has long been recommended that evaluation for myocardial injury be performed in the case of significant CO poisoning. What would really help is a study that shows how to use that information to reduce chances of an undesirable outcome.

Our Recommendation

Continue to evaluate for myocardial injury in patients with significant acute CO poisoning. Indicators of myocardial injury should prompt cardiac evaluation, as CO poisoning may unmask occult coronary artery disease.

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