

Letter To The Editor



Survival in Carbon Monoxide-Associated Cardiac Arrest

To the Editor,

I read with great interest the paper by Bronshteyn and colleagues describing the survival of a patient found in a housefire treated with hyperbaric oxygen (HBO₂) therapy and a Cyanokit for carbon monoxide (CO) poisoning complicated by cyanide poisoning following resuscitation from cardiac arrest (1). Their patient sustained permanent brain injury but was reportedly able to live independently at one year.

In their report, they cite my 2001 publication reporting a lack of survival among 18 consecutive patients treated at our facility in Seattle with HBO₂ after resuscitation from CO-associated cardiac arrest (2). The authors quote me as saying such therapy is “futile” and imply that I was wrong in making that conclusion considering their patient’s course.

In that 2001 paper, the word “futile” appears only once. “The study is also limited by its small size (18 patients). If the next two patients with the syndrome were to survive, the survival rate would be 10%, not 0%. A larger study is needed to conclude that HBO₂ therapy for such patients is futile.”

I would suggest that Dr. Bronshteyn and his co-workers take care not to misquote the literature when they write a paper. All co-authors are responsible for ensuring that prior publications are properly quoted.

Over the two-plus decades since our paper was published, I have maintained that such patients should not be transferred significant distances for hyperbaric treatment because the additional delay will likely only add to their already poor predicted survival rate. In addition, transferring patients with a poor prognosis long distance often precludes their family from being present at their end of life. However, prompt HBO₂ treatment could still be considered if the patient was being evaluated in the ED of a hospital with a capable hyperbaric facility.

As an aside, I have been asked to review numerous case reports describing a patient who survived out-of-hospital cardiac arrest due to CO poisoning. In most of these cases, cardiac arrest has been poorly documented or described. In this case, the patient was said to be pulseless but only required a relatively brief seven minutes of cardiopulmonary resuscitation to restore his circulation. Upon arrival in the emergency department, he was not tachycardic and did not apparently require vasopressors. His venous pH was only 7.22, not suggestive of someone who has been in cardiac arrest. We are not provided his arterial pH.

In rare instances, some individuals appear to have survived this overwhelming insult, but always with evidence of residual brain injury. In 23 years, no one has published a series to contradict my impression that patients experiencing cardiac arrest in conjunction with CO poisoning do not benefit significantly from emergent HBO₂ therapy.

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