Published in final edited form as:

Resuscitation. 2009 September ; 80(9): 1082–1083. doi:10.1016/j.resuscitation.2009.05.030.

Reply letter to:

Survival Following Cardiac Arrest Associated with Carbon Monoxide Poisoning

Bryn E. Mumma, David Shellenbarger, Clifton W. Callaway, Kenneth D. Katz, Francis X. Guyette, and Jon C. Rittenberger*

To the Editor:

In his letter, Dr Hampson raises worthwhile questions regarding the time course and cause of cardiac arrest in our 93-year-old patient who had a cardiac arrest following carbon monoxide (CO) poisoning, received therapeutic hypothermia (TH), and achieved good neurological outcome. We provide further data to address Dr. Hampson's concerns and to allow our audience to draw their own conclusions.

Our patient presented to an outside emergency department (ED) with a carboxyhemoglobin level of 35%. Treatment with 100% oxygen was initiated, and upon arrival at our facility approximately five hours later, his Glasgow Coma Score (GCS) was six (Eyes - 1; Verbal - 1; Motor - 4). His carboxyhemoglobin level on arrival at our facility had decreased to 11.7%. In preparation for possible hyperbaric oxygen therapy, bilateral myringotomies were performed with fentanyl 100 mcg given for pain control. Although he could be oxygenated and ventilated without difficulty, his size 6.0 tracheal tube was changed to a larger 8.0 tube. Etomidate 30 mg and succinylcholine 200 mg were injected for the rapid sequence induction. Oxygen saturation by pulse oximetry remained at 97% or higher, and chest radiography confirmed correct placement of the tracheal tube. Approximately 30 minutes after this procedure, the patient suffered a witnessed pulseless electrical activity (PEA) arrest. Following return of spontaneous circulation, his GCS was three. His electrocardiogram (ECG) showed atrial fibrillation with rapid ventricular response at a rate of 135 beats per minute. His troponin on arrival was 0.29 ng mL⁻¹, and it rose to 0.92 ng mL⁻¹ eight hours later; subsequent measurements at eight-hour intervals were 0.84, 0.65 and 0.96 ng mL⁻¹.

His initial GCS of six suggests that he experienced cerebral hypoxia related to his CO exposure, and the decline in GCS to three following cardiac arrest suggests that the injury was exacerbated by cerebral anoxia during his cardiac arrest. Given his insult, protocolised post-cardiac arrest care including TH was initiated.¹ An echocardiogram on hospital day three showed a nondilated left ventricle with normal segmental motion and preserved systolic function. Increased left ventricular wall thickness, interpreted as being consistent with left ventricular hypertrophy, was noted. As described previously, the patient showed excellent neurological recovery by hospital day six. Following a complicated hospital course, he was discharged to hospice care

© 2009 Elsevier Ireland Ltd. All rights reserved.

*Corresponding author: University of Pittsburgh, Department of Emergency Medicine, Iroquois Building, Suite 400A, 3600 Forbes Avenue, Pittsburgh, PA 15261, United States

Conflict of interest statement

No conflicts of interest to disclose.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

on hospital 19. He died one day later from complications of a bowel obstruction. His documented medical problems before presentation included hypertension, mild congestive heart failure, and renal insufficiency. His creatinine was 2.8 mg dL⁻¹ and potassium was 3.9 mmol L⁻¹ on presentation. He had no known pulmonary disease.

We believe that this man's CO poisoning was a significant factor in his PEA cardiac arrest. A close temporal association exists between the two events, and neither his ECG nor his echocardiogram show signs of focal ischemia to suggest underlying coronary artery disease as the cause. Furthermore, we believe that his neurological improvement following TH suggests that TH may benefit certain patients who arrest in the setting of CO poisoning.

References

1. Rittenberger JC, Guyette FX, Tisherman SA, DeVita MA, Alvarez RJ, Callaway CW. Outcomes of a hospital-wide plan to improve care of comatose survivors of cardiac arrest. *Resuscitation* 2008;79:198–204. [PubMed: 18951113]