Acute Myocardial Infarction
Then and Now

To the Editor:

I found the article by Drs. Simmons and colleagues (CHEST 1995; 107:1732-43) very enjoyable. I was relieved at how the medical approach to acute myocardial infarction has changed over the years as a result of the knowledge gained from trials such as the Gruppo Italiano per lo Studio della Streptochinasi nell’ Infarto Miocardio-1 (GISSI-1) and the Second International Study of Infarct Survival (ISIS-2).

I, however, have found one error that I believe needs addressing. In referring to ISIS-2 in their answer to question 1, the authors state: “Nevertheless, in ISIS-2, patients receiving rt-PA within 12 h of symptom onset had a significant reduction in mortality, and those receiving rt-PA between 12 and 24 h enjoyed a reduction in mortality that approached statistical significance.”

In ISIS-2, all patients received streptokinase, so the statement should read: “Nevertheless, in ISIS-2, patients receiving streptokinase within 12 h of symptom onset had a significant reduction in mortality, and those receiving streptokinase between 12 and 24 h enjoyed a reduction in mortality that approached statistical significance.” This statement starts on page 1734 and ends at the top of page 1735.

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To the Editor:

We would like to thank Mr. Brown for his interest in our article. We appreciate his pointing out the above oversight.

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Treatment of Carbon Monoxide Poisoning

To the Editor:

Carbon monoxide poisoning is a significant and frequently preventable injury in the United States. Dr. Shumate’s comments (CHEST 1995; 107:1474) address only one aspect of the injury and its treatment. While treatment of the carboxyhemoglobin level with an exchange transfusion could conceivably lower the blood level more rapidly if hyperbaric oxygen were not readily available, the very important reperfusion syndrome would not be addressed. Simply treating the carboxyhemoglobin levels is not sufficient therapy with our present day understanding of the process. Of equal or perhaps even more importance than the pure hypoxic insult is the intravascular sludging and vasoconstriction caused by leukocyte adherence and the release of cytotoxic substances. It is well demonstrated that hyperbaric oxygen can abort or ameliorate the progressive chain reaction that the hypoxic insult starts. The modern literature documenting the binding of CO to the cytochrome C system shows that the very high gradients of oxygen obtainable in the hyperbaric chamber are needed for reversal of the harmful intracellular effects of CO as well.

Treatment of both the smoke inhalation and the cyanide and other toxic products produced in fire-related CO poisonings are an additional benefit to hyperbaric oxygen.

With good alternative therapy available, the idea of exchange transfusion that both depletes the blood pool and exposes the patient to the risks of hepatitis and AIDS seems inappropriate. One hundred percent oxygen treatment at the scene and thereafter is still indicated, and the current excellent medical transport systems allow rapid transfer to chamber sites of even the most critical patients. All patients with neurologic and cardiac CO effects, carboxyhemoglobin in excess of 25, and pregnancy should be treated with hyperbaric oxygen. Let’s leave bloodletting to history.

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REFERENCES


To the Editor:

In regard to the points that Dr. Hurst makes in his letter, I respond with the following points.

1. While it may be shown that hyperbaric oxygen can ameliorate the progressive chain reaction that the poisoning causes, it is not clear that this effect is independent of the fact that hyperbaric oxygen is the most rapid method of CO removal. One of the articles (Toxicol Appl Pharmacol 1990; 105:340-44) that he cites asserts that rat brain lipid peroxidation products found in CO poisoned rats are a marker for neural damage. This article also shows a decrease in these products among rats treated with hyperbaric oxygen. This article, however, fails to show that this decrease is a unique benefit of hyperbaric oxygen treatment and not simply a function of the rapidity of removal of CO from the poisoned rats.

2. Evidence suggests that severe intracellular hypoxia is necessary before significant CO-cytochrome C binding can take place. Treat the hypoxia and then the CO poisoning of the cytochrome C system should rapidly resolve. Again there is nothing to suggest that hyperbaric oxygen confers a unique treatment advantage other than its rapidity of CO removal.

3. Treatment of smoke inhalation may be a situation where hyperbaric oxygen is especially efficacious; however, cyanide and CO poisonings are synergistic. The most rapid treatment of one poisoning would be expected to lessen the toxicity of the other.

4. As with any treatment, the risks and benefits of therapy must be considered, and administering blood products is not without risks.

I do not disagree that hyperbaric oxygen is the most rapid treatment of CO poisoning when it is readily available. Unfortunately, it is not always readily available and it is in these situations that exchange transfusion could supplement oxygen therapy while the physician is arranging for hyperbaric therapy. The two therapies are