

the authors that the methemoglobinemia may have contributed to the respiratory distress. Alternatively, methemoglobinemia and respiratory failure both may have been due to the same factors. In effect, the oxidation of hemoglobin into methemoglobin is believed to be due to oxygen-derived toxic radicals (hydrogen peroxide, superoxide anion, singlet oxygen and hydroxyl ion).⁵⁻⁸ The same activated species of oxygen are also thought to play a key role in the pathogenesis of adult respiratory distress syndrome (ARDS).⁹⁻¹¹ The simultaneous occurrence of ARDS and methemoglobinemia, to our knowledge, has not been reported. Certain conditions, however, such as paraquat intoxication and oxygen toxicity that are typically associated with ARDS recently have been linked to methemoglobinemia.^{12,13} Furthermore, we recently had a patient with ARDS who, incidentally, was found to have significant methemoglobinemia and evidence of severe tissue hypoxia.

Thus, in addition to impairing oxygen-carrying capacity of the blood, methemoglobinemia either may predispose a patient to acute respiratory failure, as suggested by Zurick *et al.*, or share with it the same pathophysiology, namely the toxic action of the oxygen-derived radicals.

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Another Example of Hypoxic Gas Mixture Delivery

To the Editor:—Delivery of a hypoxic gas mixture due to mechanical failure of the oxygen supply system to an anesthesia machine is a serious problem. Many causes of this type of mishap have been reported.¹⁻⁵ I would like to bring to the attention of practicing anesthesiologists another variation causing this potential disaster.

An anesthesiologist, working in an operating room suite equipped with a central supply of oxygen and a central supply of nitrous oxide commenced administering general anesthesia for an intraabdominal procedure. After an intravenous induction, the intention was to maintain the patient on a 50:50 nitrous oxide:oxygen gas mixture at a total flow rate of 4 l/min. Upon opening the needle valve on the nitrous oxide flow meter, it appeared that a much greater than usual number of turns had to be used to reach a nitrous oxide flow of 2 l/min. The bobbin in the flow meter of

nitrous oxide was rotating well, with no suggestion of it sticking in the column. After several minutes, it was noted that the bobbin of the nitrous oxide flow meter suddenly was rising in the column without further adjustment to the valve, and within a few seconds, it indicated a nitrous oxide flow rate of 8 l/min. The oxygen flow meter was still at 2 l/min. The nitrous oxide flow immediately was turned back to 2 l/min and kept under constant observation for the rest of the procedure. A later check with the hospital engineer showed that, by an unfortunate set of circumstances, the central supply of nitrous oxide was nearly exhausted right at the moment that the gas mixture was started. During the procedure, the central supply switched from main to standby operation, and the line pressure shot up accordingly.

In order to prevent this problem from recurring in

the future, it was emphasized that the time of switching from a main to an auxiliary supply should occur before line pressure drops due to near exhaustion of the main storage bank.

Even though new models of anesthesia machines and central gas supplies are becoming more widely distributed with evermore sophisticated safeguards, the vigilance of the individual anesthesiologist cannot be overemphasized.

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Concerning the Actions and Efficacy of Different Antacids

To the Editor:—We read with interest the article, "Effectiveness of Bicitra® as a Preoperative Antacid," by Gibbs *et al.*¹ In the introduction, the authors stated that "Although Bicitra® contains approximately the same amount of sodium citrate as does 0.3 M sodium citrate, the pH of Bicitra® is 4.8. Therefore, Bicitra's® buffering or neutralizing capacity might be less than sodium citrate." Also they stated in the discussion that "the lower mean pH with Bicitra® may be explained in part by the lower pH of the Bicitra® solution (4.8 vs. 8.5 for 0.3 M sodium citrate). The diluent effect of the higher pH solution may contribute to the greater efficacy of sodium citrate." These statements imply that the lower pH of Bicitra® is the main reason why it is less effective than sodium citrate.

Chemically, Bicitra® is a "buffering" agent (it contains a weak acid, citric acid, and its corresponding salt, sodium citrate); it is not an antacid. Theoretically, Bicitra® should be less effective in neutralizing acid in solutions than 0.3 M sodium citrate solution. This is not due to its lower pH (hence lower in diluent effect) but rather to its nature of being a "buffer" solution.

We also have been interested in a commercially available clear-liquid antacid. We have evaluated several antacids, including Bicitra®, in an animal lung model.

From our data the antacid that is comparable with or even better than 0.3 M sodium citrate solution is Alka-Seltzer® Effervescent Antacid.²

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