Carbon Monoxide and General Anesthesia

To the Editor:

In the report “False-Low Carbon Monoxide Diffusing Capacity Measurement After General Anesthesia” by Gilbert and colleagues (February 1996), the authors attribute the measurement of high-mixed expired carbon monoxide (CO) concentrations in their postoperative patients to nitrous oxide. They claim that it would be “a physiologic impossibility” for expired CO concentrations to be higher than the inspired concentration utilized in diffusing capacity (DL) measurement.

It should be pointed out that a patient who has been intoxicated with CO will clear the gas via exhalation after removal from the source of exposure. When measuring DL, it is assumed that CO back pressure is zero. If the patient’s carboxyhemoglobin (COHb) level is elevated, it is necessary to correct the measured DL to account for a false-low value.

Furthermore, a number of reports have been published that document the development of elevated COHb levels during general anesthesia. The mechanism by which this occurs is thought to be due to generation of CO by a chemical interaction within the breathing circuit between the carbon dioxide absorbent and halogenated volatile anesthetics.

The patients reported by Gilbert et al.1 received isoflurane in addition to nitrous oxide. It is possible that the measured expired CO concentrations were real, as a result of CO exposure during general anesthesia. Based on the kinetics of COHb elimination, it is not inconceivable that they would still be clearing CO when the DL measurements were performed 2 to 4 h later. When patients demonstrate unusually low CO diffusing capacity measurements following general anesthesia, COHb levels should be obtained.

To the Editor:

We would like to respond to the letter of Drs. Hampson and McDonald concerning their Communication to the Editor, “False-Low Carbon Monoxide Diffusing Capacity Measurement After General Anesthesia.”

Drs. Hampson and McDonald point out that severe carboxyhemoglobin toxicity could conceivably produce an expired carbon monoxide concentration larger than the inspired, and therefore produce a negative value for diffusing capacity (DL). This may be true but hardly seems pertinent to the phenomenon that we are describing.

We were not aware of the recent work showing that anesthetic gases could react with carbon dioxide absorbing agents in anesthetic circuits to produce carbon monoxide; this effect is apparently more pronounced when the absorbing agent is dry. The highest value of carboxyhemogoblin that we noted in the medical literature under operating room conditions was 7.6%; this could certainly lower the calculated DL but probably not produce a negative value.

The experimental subject described in our letter3 was simply breathing dry nitrous oxide from a tank via a nasal cannula. The mechanism noted above would obviously not be working under these conditions.

We appreciate the interest of Drs. Hampson and McDonald and believe they have pointed out another mechanism in addition to the one we proposed, which could account for the low values of DL following general anesthesia.

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Reference

Pleural Fluid Cholesterol and Lactate Dehydrogenase for Separating Exudates From Transudates

To the Editor:

The study by Costa and colleagues (November 1995)3 concluded that measurements of pleural fluid cholesterol and lactate dehy-