170 mmHg; below this value, the lower the PaO₂, the greater the increase in V̇E. It also has been shown that the depression of afferent chemoreceptor electrical activity follows a time course that is closely related to fall of V̇E. Therefore, the O₂ test of Dejours is used to evaluate the O₂-related chemoreflex contribution to ventilation. In any case, it is widely accepted that, under normoxic conditions, the O₂-related peripheral chemosensitivity influences respiration.

We did not study profound hypoxemia under halothane anesthesia for ethical reasons. We have shown that almitrine can restore the O₂-related contribution to ventilation, since its analeptic effect was abolished in hypoxic conditions. PaCO₂ was not statistically different between air (56.2 ± 1.4 mmHg) and O₂ breathing (57.2 ± 1.9 mmHg) and cannot explain our results. Dr. Ward is wondering if the same phenomenon could be observed in more profound hypoxia. Our purpose was not to study the complex mechanisms that occur during hypoxia but to observe if “almitrine bismesilate, a specific stimulant of the peripheral chemoreceptors, could restore peripheral chemosensitivity.” Our results clearly show that, if not complete, a partial restoration of the “hypoxic” drive to breathing, under normoxic conditions during halothane anesthesia, was obtained with almitrine bismesilate.

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The Environment, Carbon Dioxide, and the Milieu Intérieur

To the Editor:—I read with surprise of the concern of Dr. C. Stanley Jones regarding inspired carbon dioxide. The surprise was due to the editorial acceptance of a letter smacking of scare tactics and homilies (i.e., the green house effect and the milieu intérieur). Some perspective is provided by quantitation and some comfort by physiology, which should dispel the fears of CO₂ and rebreathing.

Firstly, the CO₂ level of the atmosphere has not been constant at 0.033 ± 0.001% (or 330 ± 10 ppm). A much lower level existed 25,000 years ago, approximately 225 parts per million, and a higher level even earlier, 350 parts per million, 130,000 years ago. Fear of the current secular rise in atmospheric CO₂ is based on theoretic concerns of global mean temperature rather than on man’s respiration and acid base balance. Recent reports are, to quote Philip Abelson, “less panicky” than earlier statements. The present estimate of doubling time is 220 years. This would give 700 parts per million CO₂ early in the 23rd century.

Secondly, a 10-fold increase in the inspired CO₂ (i.e., 3,300 parts per million or 0.33% FICO₂) is less than that regularly tolerated for long times in closed environments such as submarines. Given no central responsiveness, it would raise PaCO₂ only 2 mm/Hg or alternately, given infinite responsiveness, an increase in minute volume of less than 250 ml/min would be required to restore CO₂ to normal. Dr. Jones should note that if his patients are breathing spontaneously, their arterial CO₂ probably have increased six to ten times this much because of anesthetic depression of drive. Use of the Lack system or other Mapleson A modification would further lower the inspired CO₂, as the Bain system is inefficient in this mode of ventilation. If their ventilation is assisted or controlled, the extra 250 ml/min needed is truly negligible.

Finally, one might point out that the only major deleterious effect of increasing arterial CO₂ is a slightly greater decrease in arterial oxygen tension. Breathing air at altitude might produce a hypoxic stress, but
breathing 40% oxygen enriched inspirates fully compensates for a 1% rise in $P_{CO_2}$ at the top of Mount Everest. At sea level, 1% oxygen enrichment compensates for a 5 torr increase in $P_{CO_2}$. The minor acidosis is initially only 0.03 pH units with no ventilatory responsiveness. The beneficial effects of $CO_2$ on cerebral and coronary blood flow, on ventilation perfusion matching in the lung, on oxygen unloading in tissue (to cite the most important effects only), should, in light of present knowledge, allay apprehension that the milieu intérieur is compromised by a bit of inspired $CO_2$.

I think our patients would not be safer if we used nonrebreathing techniques. They would be poorer, since in the end they pay for their consumed supplies.

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Can We Do without $O_2$ Analyzers?

To the Editor—Dr. Zorab’s\(^1\) letter suggests that the use of $N_2O$ premixed with $O_2$ would prevent the occurrence of hypoxic accidents during anesthesia without the need for an oxygen analyzer. In another letter, Dr. Feingold\(^2\) raises the question of whether instruments such as the currently available $O_2$ analyzers will produce incremental improvements in anesthesia safety. We would like to present a case that may help define the need for $O_2$ analyzers.

REPORT OF A CASE

Patient 1, a 63-year-old man, was admitted for a superficial temporal to middle cerebral artery bypass. After induction, anesthesia was maintained with 50% oxygen/nitrous oxide and isoflurane delivered by an Ohio isoflurane vaporizer mounted in the fresh gas supply line to the circle. One hour into the procedure, the surgeon noted dark blood. All connections to the patient were confirmed as being secure, good breath sounds were heard, and the patient’s chest was seen to rise with each breath. The flow meter settings were verified at 50% oxygen, and the disconnect alarm had not indicated a pressure loss. The blood remained dark, however, and so the patient was given 100% oxygen and his ventilatory status again checked. At this point, a precipitous drop in blood pressure occurred. The ventilator then was disconnected and manual ventilation attempted. Despite the use of the oxygen flush, the bag would not fill and a disconnect of the machine fresh gas flow then was noted. The problem was corrected, the surgery canceled, and the patient recovered without sequelae.

Many of our anesthesia machines were put into service before the advent of isoflurane and thus now have an “add-on” vaporizer interposed in the machine-to-circle fresh gas line (fig. 1). There is a one-way valve in this vaporizer, which will prevent pressure loss in the advent of a disconnect between machine and vaporizer.\(^3\) If a descending bellows ventilator is utilized (i.e., Drager), the patient will continue to be ventilated indefinitely—albeit with expired gas plus whatever room air is entrained. In this situation, the inhaled mixture becomes hypoxic.\(^4\) Only a monitor of circle gas concentrations reliably will alert one to this type of disconnect before signs of hypoxic changes. This is true even in the presence of disconnect alarms or anesthesia machines “incapable” of delivering hypoxic mixtures.

There have been other reports,\(^3\)-\(^5\) of accidents having these similar features: failure of fresh gas inflow due to a disconnection or leak; maintenance of circle pressure due to a check valve or high outflow resistance; a falling weighted bellows type of ventilator. In these cases,

![Fig. 1. Configuration of equipment. The function of the vaporizer check valve is shown schematically in inhalation and exhalation.](http://anesthesiology.pubs.asahq.org/pdfaccess.ashx?url=/data/journals/jasa/931419/)

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