Anesthesiology
59:159, 1983

A Plea for the Routine Use of Oxygen Analyzers

To the Editor:—I would like to respond to opinions stated by Alfred Feingold, M.D., concerning the continuous use of oxygen analyzers.[1] In addition to questioning their usefulness, he suggests that currently available analyzers are unreliable and require frequent maintenance and calibration. This simply is not true. Models that I have used over the past 6 years are very reliable, need little maintenance, and can be calibrated rapidly. Incorporating a check and calibration of the oxygen analyzer during the first daily check of the anesthesia machine requires less than 30 sec. In our hospital, oxygen analyzers are used during all general anesthetics, and they are turned *on*.

Mazze pointed out the need for continuous in-line oxygen monitors some 10 years ago.[2] Currently available oxygen analyzers accurately will analyze and display the inspired oxygen concentration and rapidly will detect and warn of hypoxic mixtures caused by either machine malfunctions or human error. Because hypoxia during anesthesia may be difficult to detect, this information is invaluable.[3] An oxygen analyzer obviously will not detect a lack of ventilation of a patient's lungs, but another simple monitor, the precordial or esophageal stethoscope, will.

Dr. Feingold also suggests that proportional flow devices may be a better way to assure that minimum safe concentrations of oxygen are delivered to patients. Although I cannot understand why any new anesthesia machine sold today should be able to deliver any less than 21% oxygen, mechanical malfunction and delivery of the incorrect gas to anesthesia machines will continue to occur. In these cases, only an oxygen analyzer monitoring the inspired gases or the patient would detect hypoxia quickly.

In this state over the past 10 years, more than one anesthetic death resulting from the accidental administration of hypoxic gases has occurred when oxygen analyzers were not being used. In June 1982, the South Carolina Department of Health and Environmental Control added the following standard: "Anesthesia apparatus shall be equipped with a device to measure the oxygen component of the gas being inhaled by the patient. The device shall emit an audible and/or visual alarm should the proportion of oxygen fall below a safe level."[4]

In short, oxygen analyzers are relatively inexpensive, are reliable, and are easy to maintain and use. They will detect low concentrations of oxygen in the inspired gases early and will save lives. Let's use them until a better monitor is available.

WILLIAM E. MARKS, JR. M.D.
Director of Anesthesiology
Easley Baptist Hospital
Easley, South Carolina 29640
Clinical Associate Professor
Department of Anesthesiology
Medical University of South Carolina
Charleston, South Carolina

REFERENCES

1. Feingold A: Oxygen Analyzer as Viewed by the public, attorney, and an anesthesiologist. ANESTHESIOLOGY 57:553–554, 1982
2. Mazze RI: Therapeutic misadventures with oxygen delivery systems: The need for continuous in-line oxygen monitors. *Anesth Analg (Cleve)* 51:787–792, 1972
3. Knill RL, Gelb AW: Peripheral chemoreceptors during anesthesia: Are the watchdogs sleeping? ANESTHESIOLOGY 57:151–152, 1982
4. South Carolina Department of Health and Environmental Control Regulation No. 61-16: Minimum Standards for Licensing of Hospitals and Institutional General Infirmaries in South Carolina. Section 611.3, 1982. 2600 Bull Street, Columbia, South Carolina

(Accepted for publication January 17, 1983.)

Anesthesiology
59:159–160, 1983

Antagonism of Nitroprusside Effects by Cyanide May Not Be Clinically Relevant

To the Editor:— Recent papers[1–3] have attempted to demonstrate that plasma accumulation of cyanide may be responsible for resistance in patients who require large doses of nitroprusside (SNP) to achieve hypotension, by directly antagonizing the vasodilator effects of the drug. This has been discussed most recently by Kruszyna *et al.*,[3] who showed that this action of SNP on isolated rabbit aortic strips, contracted by various concentrations of norepinephrine *in vitro*, were antagonized partially by cyanide. These results are interesting, but one must consider them in the clinical context. Concentrations of cyanide used during these experiments were of the order of 40–100 μmol/l. Analysis of blood samples from many hundreds of patients receiving SNP in the operating theater and intensive therapy unit (ITU) has convinced us that plasma levels during clinical

use seldom exceed 1 μmol/l, and most are considerably below this. Thus, the cyanide levels quoted by these authors are far in excess of those usually encountered in clinical practice. Even our most resistant patients rarely have exceeded a plasma cyanide of 3 μmol/l, and cyanide levels of 40–100 μmol/l must be seen to be quite unrelated to the clinical situation. Kruszyna *et al.* conceded this point in their article, but argued further that the ratio of CN^- to SNP concentration is as important as the absolute CN^- concentration; in fact, a level of cyanide greater than that of SNP may be necessary for resistance to develop. This situation might occur, for example, after cessation of SNP infusion, because, in the baboon, plasma CN^- concentration fell more slowly than that of SNP. On the contrary, resistance to the hypotensive effects of SNP usually is seen in the early stages of the infusion of the drug or may develop gradually in the later stages, but never after the drug has been withdrawn!

An increase in cardiac output and, therefore, resistance to hypotensive action during administration of SNP, may be achieved, however, by cyanide itself.[4] A rise of 20–25% above initial levels was seen after infusion of SNP into dogs for 45 min and coincided with the maximum plasma cyanide levels.[5] This effect of cyanide would provide an alternative explanation for the reduction of tachyphylaxis produced by thiosulfate in children infused with SNP.[6]

Furthermore, a survey of the available literature shows that most patients reported to exhibit resistance to SNP were relatively young, and it is particularly common in children. Resistance to hypotensive drugs of all kinds is well described in young patients and usually is associated with the development of tachycardia, probably mediated by the baroreceptor response, which is active in this group. Since beta-adrenoreceptor antagonist drugs have been used more widely as an adjuvant to induced hypotension, both with SNP and other agents, reports of resistance have become uncommon and most clinicians appear to have become much less concerned with the problem.

DR. P. V. COLE, M.B., CH.B., F.F.A.R.C.S.
Consultant Anaesthetist

C. J. VESEY, M.SC.
Senior Biochemist

Anaesthetic Laboratory
St. Bartholomew's Hospital
West Smithfield,
London, EC1A 7BE, England

REFERENCES

1. Tremblay NAG, Davies DW, Volgyesi G, Kadar D, Steward DJ: Sodium nitroprusside: Factors which attenuate its action. Studies with isolated gracilis muscle of the dog. Can Anaesth Soc J 24:641–650, 1977
2. Grayling GW, Miller ED, Peach MJ: Sodium cyanide antagonism of the vasodilator action of sodium nitroprusside in the isolated rabbit aortic strip. ANESTHESIOLOGY 49:21–25, 1978
3. Kruszyna H, Kruszyna R, Smith RP: Nitroprusside increases cyclic guanylate monophosphate concentrations during relaxation of rabbit aortic strips and both effects are antagonised by cyanide. ANESTHESIOLOGY 57:303–308, 1982
4. Krasney JA: Cardiovascular responses to cyanide in awake sinoaortic denervated dogs. Am J Physiol 220:1361–1366
5. Simpson PJ, Adams L, Vesey CJ, Cole P: Some physiological and metabolic effects of sodium nitroprusside and cyanide in the dog. Br J Anaesth 51:81–87, 1979
6. Davies DW, Greiss L, Kadar D, Steward DJ: Sodium nitroprusside in children: Observations on metabolism during normal and abnormal responses. Can Anaesth Soc J 22:553–560, 1975

(Accepted for publication January 17, 1983.)

Anesthesiology
59:160–161, 1983

In reply:— The contention of Cole and Vesey that the reversal of nitroprusside-induced relaxation of isolated rabbit aortic strips may not be relevant to the rare patient who exhibits nitroprusside resistance is acceptable to us. We went to some lengths to point out how remote that possibility might be and to encourage the pursuit of other explanations. Certainly, we never implied that antagonism could occur after the drug has been withdrawn, which is a contradiction in terms. It also was a weakness in one study,[1] which proported to show that cyanide failed to antagonize the effects of nitroprusside in dogs, because it was not clear that the two agents ever were infused contemporaneously. What is of interest to us is that the observation may be trying to tell us something of fundamental importance about the mechanism of action of an interesting drug. This is particularly true, because we have shown recently[2] that cyanide also reverses the inhibition of human platelet aggregation by nitroprusside. Thus, the drug appears to act on vascular smooth muscle and on platelets by a common mechanism.

ROGER P. SMITH, PH.D.
Professor and Chairman