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stages of the infusion of the drug or may develop gradually in the later stages, but never after the drug has been withdrawn!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.

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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.

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Use of Oxygen Analyzers Should Be Mandatory

To the Editor:—Dr. Feingold[1] is correct that advice from a sensational television show and fear of looking bad in court are not good reasons for using oxygen analyzers. But there are other, compelling reasons to do so.

Incorrect settings of gas flows are not the only cause of hypoxia. Other causes include inaccurate or leaking flowmeters,[2,3] rebreathing secondary to low fresh gas flows,[4] failure of the oxygen source on a machine not equipped with an oxygen failure safety valve, delivery of a gas other than pure oxygen through the oxygen flowmeter,[5–11] and failure of systems designed to ensure a safe oxygen–nitrous oxide mixture.[12] Observation of the color of the blood or skin are unreliable methods of detecting hypoxia, as are monitoring of heart rate and blood pressure.[13]

In addition to detecting hypoxia, oxygen analyzers can warn of inadvertent increases in oxygen that lead to patient awareness or damage to the lungs or eyes.[14–16] When used with closed system techniques, they provide much information about the patient's uptake of oxygen and other gases.[17]

An oxygen analyzer can achieve its purposes only if it is used. This condition can be met if anesthesia personnel appreciate its role in enhancing patient safety. Our experiences over the past 5 years are that it is possible to keep these devices functioning properly without undue effort. We have found that having specific places on the anesthesia record for noting oxygen percentages and alarm settings helps to remind people to use them.

It is true that the analyzers presently available could stand improvement. We hope that the manufacturers of analyzers and anesthesia machines take note of Dr. Feingold's excellent suggestions. In the meantime, how many tragic cases of hypoxic death or brain damage can we tolerate while waiting for the perfect device?

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