

contrast clonidine, a relatively specific alpha-2 agonist that acts indirectly by reducing central nervous system sympathetic outflow, would not be blocked by the actions of ethacrynic acid.⁴ The mechanism for poor absorption in critically ill patients proposed by the authors may account for the ability of rectally administered clonidine to control hypertension in their patient when oral clonidine failed.

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In reply: We thank Dr. Hosking for his comments and concern that the use of ethacrynic acid may have interfered with the vasodilator actions of both nitroglycerin and nitropruside.

We reexamined our cases and noted that the first patient received ethacrynic acid, but as a secondary measure late in the course of managing her hypertension. None of the other five cases received ethacrynic acid. Therefore, we do not feel that ethacrynic acid interfered with the vasodilators in the first case. We will certainly keep this drug interaction in mind for future reference.

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Fail-safe Oxygen Analyzers

To the Editor:—Whenever nitrous oxide is supplied to an anesthesia circuit, there is a real possibility that a hypoxic mixture will be delivered secondary to human error in setting the flow controls. Less commonly, inaccurate or leaky flowmeters in conjunction with N₂O can lead to hypoxia. Use of an oxygen analyzer allows early detection of these errors but is often vitiated by another human error, failure to turn the analyzer on. A modification of

the anesthesia machine could guarantee that nitrous oxide will only be used when the oxygen analyzer is on.

An electromechanical fail-safe valve should be placed in the nitrous oxide intermediate pressure line. This valve would only be opened by a solenoid that is activated when the oxygen analyzer is turned on. Nitrous oxide could not be delivered unless the O₂ analyzer is turned on! If the machine is equipped with air or N₂, these lines should also have electromechanical fail-safes. Of course the low O₂ alarm must have a default preventing settings below 20%.¹ Perhaps the fail-safe analyzer should be further refined to shut the valves automatically whenever reading below 20%, thus allowing only oxygen to be delivered to

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the circuit. Conceivably, this might replace the traditional oxygen pressure fail-safe. However, initially it would be prudent to install the electromechanical fail-safe valve in the intermediate pressure lines in series with the traditional fail-safe rather than as a replacement for it.

Whenever N₂O is used, continuous on-line oxygen analysis is mandatory. The electromechanical fail-safe would force anesthetists to use their analyzers whenever using N₂O.

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Hypotension after Intravenous Cimetidine in Intensive Care Unit Patients

To the Editor:—Iberty *et al.* have recently documented adverse hemodynamic effects in 22 of 24 intensive care unit patients after intravenous administration of cimetidine as a single 2-min bolus injection.¹ Ninety-two per cent of these patients demonstrated a decrease in mean arterial pressure (MAP) greater than 10 mmHg. We feel that several aspects of this observation require additional comment.

The authors did not report renal function of the study patients nor were serum cimetidine concentrations determined. This may be important in light of our observations of markedly elevated cimetidine serum concentrations after an intravenous bolus administration of cimetidine to patients with severely impaired renal function (SIRF) compared with normal volunteers (NV).² Serum cimetidine concentrations 3 min after the end of the 300 mg 2-min iv bolus ranged from 32 to 78 mg/l and from 6 to 17 mg/l in the SIRF and NV groups, respectively. We noted a significant reduction in the volume of the central compartment in the SIRF group compared with the NRF group (0.091 ± 0.119 l/kg *vs.* 0.320 ± 0.160 l/kg, respectively; mean \pm SD).

On the basis of these results and preliminary evidence that cardiovascular toxicity was associated with elevated peak serum cimetidine concentrations,³⁻⁵ we recommended in 1983 that cimetidine be administered by iv infusion over at least 30 min to avoid excessive peak serum cimetidine concentrations and thus reduce the risk of cardiovascular toxicity.

It would be useful to know the renal function as well as other clinical parameters of the study patients of Iberty *et al.*¹ to assess the relationship of these parameters with the decrement in MAP. Furthermore, the measurement of peak serum cimetidine concentrations would have been

useful in order to assess whether there was any correlation between serum drug concentration and decrement in MAP.

We suggest that cimetidine be administered by slow intravenous infusion, rather than by rapid intravenous bolus, in all patients in order to reduce the risk of cardiovascular toxicity due to excessive serum cimetidine concentrations.

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