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 N_2O is used, continuous on-line oxygen analysis is mandatory. The electromechanical fail-safe would force anesthetists to use their analyzers whenever using N_2O.

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Hypotension after Intravenous Cimetidine in Intensive Care Unit Patients

To the Editor:—Iberti *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 ± 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 Iberti *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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