

informed us that to his knowledge only one type of gas machine* from a single manufacturer employs a truly fail-safe device, whereby closing the flowmeter valve on the oxygen line will automatically shut off all other gas flow. It becomes obvious that if nitrous oxide or other gases continue to be delivered in the absence of oxygen, as is readily possible with the usual apparatus, "fail-safe" is a misnomer. We contend that no-one administering anesthesia should ever rely on this type of equipment, and accordingly it becomes an expensive and dangerous addition to the machine. The one brand of equipment containing an actual fail-safe

* Foregger Company—Model 710.

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Cardiovascular vs. Renal Effects of Dopamine

To the Editor:—The communication by Birch and Boyce purports to demonstrate that the butyrophenone drug droperidol does not prevent "dopamine-induced renal vasodilatation" when given in a dose of 0.1 mg/kg.¹ Their evidence for this is a measured increase in renal blood flow when dopamine was infused into patients that was not significantly attenuated by droperidol administration. However, the dosage of dopamine used (20 µg/kg/min) would not be expected selectively to stimulate only the vascular dopaminergic receptors that would be blocked by droperidol.² This selective stimulation typically occurs only at very low doses of dopamine where cardiac output and systemic blood pressure are unaffected. Robie and Goldberg found that at a dose of dopamine of 1.25 µg/kg/min in dogs, cardiac output, total peripheral resistance, and systemic blood pressure did not change significantly, but renal vascular resistance decreased and renal blood flow increased significantly.³ At the high dose ranges used by Birch and Boyce, cardiac output should be markedly increased by beta-receptor stimulation.^{2,3} Cardiac output *per se* was not measured, but the authors did state that [dopamine]

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In reply:—It was not the purpose of our study to show whether droperidol prevented dopamine-induced renal vasodilation. This was done by Yeh and others with a similar drug, haloperidol.¹ Ours was a study of droperidol-dopamine interaction. We agree that the increased blood flow following dopamine in patients pretreated with droperidol could be entirely on the basis of increased cardiac output or some combination of increased cardiac output and increased renal blood flow. The important point is that the renal

mechanism could well be emulated by other manufacturers and by this manufacturer in its other models.

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"increased systolic blood pressure, diastolic blood pressure, and renal blood flow." These changes would be the expected results of a large increase in cardiac output. Drs. Birch and Boyce have demonstrated that if you increase cardiac output with a potent inotropic agent, renal blood flow will increase. This is hardly surprising. The study should be repeated using more appropriate doses of dopamine.

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blood flow increased following the usual clinical doses of dopamine and droperidol, 0.1 mg/kg.

To determine how much of the effect resulted from increased cardiac output and how much from direct vasodilation would require more sensitive equipment than we presently have available. For example, using low-dose dopamine, 1.25 µg/kg/min, the renal blood flow changes in Robie's dogs were only about 50 ml/min.² This is within the error of our flow probes used in man. Another problem we had with our patients

was that during the operation the patients were in the severely flexed lateral position, which may make cardiac output determinations invalid. We chose a dose of dopamine of 20 $\mu\text{g}/\text{kg}/\text{min}$ because that is the dose most commonly used following cardiopulmonary bypass, and this is where the question first arose. To elucidate the exact mechanism will probably require animal studies where cardiac output and renal blood flow can be monitored with more ease and accuracy.

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Intracardiac Catheters Unnecessary in Neurosurgical Anesthesia

To the Editor:—The placement of a central venous catheter in patients undergoing neurosurgical procedures in the sitting position has become an accepted technique. Dr. Munson and colleagues have advocated the placement of a pulmonary-artery catheter to further facilitate the withdrawal of entrained venous air.¹ After a brief trial of right atrial catheterization, we abandoned placement of any type of intracardiac catheter for three reasons. First, there are serious risks associated with both right atrial²⁻⁵ and pulmonary arterial catheterization.⁶⁻⁹ Second, attaining proper catheter placement may be time-consuming and troublesome. Third, our experience in a large institution with an active neurosurgical service has led us to believe that with proper management, venous air embolism is not the great hazard some believe it to be.

To verify our clinical impression, all neurosurgical procedures done with the patients in the sitting position during the last five years were reviewed. Anesthesia technique included placement of an esophageal stethoscope, intravenous infusion of large volumes of lactated Ringer's solution, and the use of continuous positive airway pressure. The cases of 461 patients, including 87 undergoing posterior-fossa craniotomy and 374 undergoing cervical laminectomy, were reviewed. All patients had inhaled nitrous oxide, at flow-meter concentrations ranging from 50 to 66 per cent.

Three patients (0.7 per cent) experienced air embolism sufficient to produce a murmur audible with an esophageal stethoscope and changes in vital signs. In all three tachycardia to 120–150 beats/min developed, and two showed decreases in systolic blood pressure of 20–30 torr. Packing the wound and discontinuing nitrous oxide effected prompt recovery in all three cases.

We are now using a Doppler monitor for detecting air embolism, but were not during the period re-

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viewed. This probably accounts for our low incidence compared with that reported by others.¹⁰ This failure of detection, however, in no way detracts from the fact that with proper management, the incidence of clinically important air embolism is very low; hence our conclusion that routine use of central venous or pulmonary-artery catheters is not necessary.

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