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

MYRON J. LEVIN, M.D.
Clinical Professor, Anesthesia
University of Health Sciences
The Chicago Medical School
Chicago, Illinois 60637

KANTEERAVA BALASARASWATHI, M.D.
Clinical Instructor, Anesthesia
Loyola University Medical Center
Chicago, Illinois 60626

(Accepted for publication September 19, 1977.)

* Foregger Company—Model 710.

Anesthesiology
48:153, 1978

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] "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.

BARRY L. ZIMMERMAN, M.D.
Major, Medical Corps
Anesthesiology/Operative Service
Brooke Army Medical Center
Fort Sam Houston, Texas 78234

REFERENCES

1. Birch AA, Boyce WH: Effects of droperidol–dopamine interaction on renal blood flow in man. ANESTHESIOLOGY 47:70–71, 1977
2. Goldberg LI: Cardiovascular and renal actions of dopamine: Potential clinical applications. Pharmacol Rev 24:1–29, 1972
3. Robie NW, Goldberg LI: Comparative systemic and regional hemodynamic effects of dopamine and dobutamine. Am Heart J 90:340–345, 1975

(Accepted for publication September 21, 1977.)

Anesthesiology
48:153–154, 1978

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 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