plained by hepatic storage (uptake) rather than by metabolism of halothane. They apparently failed to understand that: "Equilibrium between blood and liver was approached from high to low concentrations to eliminate the effect of uptake" (ANESTHESIOLOGY 34: 233). We agree that at the higher concentrations there may have been uptake into the liver, which would give an overestimate of metabolism. Thus, if uptake (or storage) were to manifest itself as a concentration difference across the liver, it should have done so at the higher concentrations. As we reported, the fractional extraction of halothane was lowest at these concentrations. Moreover, as we decreased the partial pressure of halothane in the inflowing hepatic blood, we should expect to see a reversal of the halothane partial pressure difference between arterial blood and liver. Halothane should then move from liver to blood, thus causing the venous concentration of halothane to exceed that in arterial blood. Since we found the opposite, we conclude that "storage" cannot explain our data and, if anything, would only strengthen the conclusion we reached: the fractional metabolism of halothane increases as the partial pressure of halothane in the liver decreases. Our only error may be that the fractional differences we measured across the liver underestimate rather than overestimate the total halothane metabolism.

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Formulation of *d*-Tubocurarine

To the Editor:—Dowdy and her colleagues [1] have shown clearly that the antibacterial preservatives in the commercial preparations of *d*-tubocurarine have a marked negative inotropic action, and that *d*-tubocurarine by itself has a slight positive inotropic action. Because of the confusion that exists in the naming of solutions of *d*-tubocurarine in different countries, we should like to point out that the solution "Tubarine (miscible)," which is widely used in Great Britain, contains no antibacterial or antioxidant preservatives. In our experience, administration of *d*-tubocurarine, 30 mg, has been followed by highly significant decreases in blood pressure in both healthy and poor-risk adults.[2, 3]

Dowdy *et al.* do not say that an action on the heart of *d*-tubocurarine or preservatives is responsible for the clinical hypotension, but we think it worthwhile to point out that the marked hypotension which does occur with the dosage used in Great Britain must be due to histamine liberation, or, more probably, to ganglionic blockade, and has no connection with preservatives. This hypotension has not been observed in comparable studies with pancuronium.[2, 3]

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REFERENCES

1. Dowdy EG, Holland WC, Yamanaka I, *et al.*: Cardioactive properties of *d*-tubocurarine with and without preservatives. ANESTHESIOLOGY 34:256, 1971
2. McDowell SA, Clarke RSJ: A clinical comparison of pancuronium with *d*-tubocurarine. Anesthesia 24:581, 1969
3. Lyons SM, Clarke RSJ: Unpublished observations