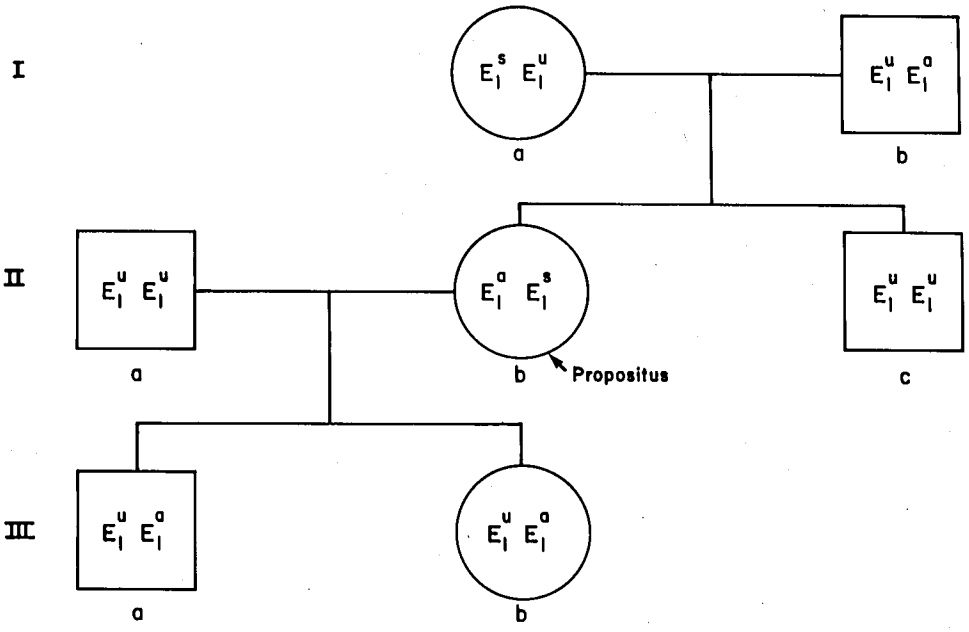

FIG. 1. Apparent genotypes of Case 1 family.

bilical cord blood represents an easy source for analysis. Regional analgesia using amide-type drugs in these parturients was found safe and is our method of choice.

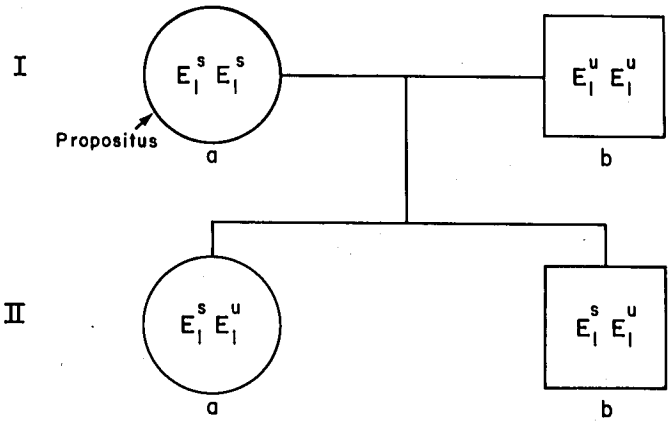

FIG. 2. Apparent genotypes of Case 2 family.

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Beneficial Effects of Halothane on Myocardial Ischemia

To the Editor:—In their recent article, Dr. Verrier and his colleagues[1] were kind enough to cite our earlier studies[2] which demonstrated that halothane produced an increase in coronary vascular resistance. However, their own elegant studies have demonstrated that by using waterfall pressure rather than left ventricular end-diastolic pressure in the calculation of vascular resistance, halothane produces no change. Their studies may therefore reconcile conflicting data in previous investigations on this subject.[2-5]

From our earlier study, we concluded that halothane jeopardized oxygen supply/demand relationships in the myocardium. Because this conclusion was at variance with our own clinical experience using halothane, we subsequently undertook an investigation which was published too recently to be cited in Dr. Verrier's manuscript. In a canine preparation with a one-stage ligation of the anterior descending artery, it was found[6] that halothane 1 per cent inspired significantly improved supply/demand relationships in the ischemic in comparison with normal myocardium, despite producing a 42 per cent reduction in mean arterial pressure. Although the mechanism of this effect was not open to investigation, suspicion rested heavily upon the 15 per cent reduction in heart rate which occurred.

Thus, there appears to be common agreement that with a variety of different animal experimental models of myocardial ischemia,[1,6,7] halothane has a beneficial effect, probably secondary to changes in systemic and myocardial hemodynamics and not by any specific mechanism. These data confirm our teaching that the clinical management of patients is based on maintaining oxygen supply to the myocardium while suppressing those factors which lead to an increase in oxygen demand. However, one would agree with the sentiments expressed by Dr. Merin[8] that unfortunately these studies do not provide additional clinical guidance in managing patients with ischemic heart disease on the tolerance limits of change in cardiac filling pressures, systemic arterial pressure, heart rate, or any other hemodynamic factor.

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Dextran Is Not a Potent Local Anesthetic Adjuvant

To the Editor:—I read with interest the letter by R. E. Loder[1] on the use of dextran as a local anesthetic adjuvant. The scientific literature does not support his contentions, however.

Based upon research conducted at the University of Washington, the Mason Clinic, and my own laboratory at the University of California, Davis, the following statements regarding dextran and regional anesthesia are more realistic:

1) Dextran does not form a macromolecular complex with local anesthetics.[2-3]

2) Dextran's mechanism of action as a local anesthetic adjuvant appears to be a function of its *p*H. The more alkaline the dextran, the greater the prolongation of block.[2]

3) Pharmaceutical dextrans can vary markedly in *p*H. For example, Rheomacrodex® (Pharmacia), the most widely used dextran in the United States, has a *p*H of 4.5 to 5.5 in contrast to Dextraven® (Fisons) which has a neutral *p*H. This difference in *p*H explains some of the contradictory dextran studies that have appeared in the anesthesia literature.

4) The higher molecular weight dextrans are not more potent local anesthetic adjuvants. In a recent study, dextran 40 proved superior to dextran 75, 110, and 150 at prolonging bupivacaine's duration of block in an experimental model. The result we attributed to the *p*H of the dextrans used in the study and not to their respective molecular weights.[4]

5) Finally, dextran is a rather mediocre and impractical local anesthetic adjuvant for clinical use. The maximum prolongation of block that we have been able to elicit experimentally is approximately 50 per cent using a dextran selected specifically on the basis of its alkalinity.

It is evident that the need for dextran as a local anesthesia adjuvant has diminshed since Loder first advocated its use in 1960.[5] The duration of block produced by the