

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<sup>6</sup> 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,<sup>1,6,7</sup> 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<sup>8</sup> 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.

G. SMITH, M.D.  
Professor of Anaesthesia  
University of Leicester  
Leicester, England

#### REFERENCES

1. Verrier ED, Edelist G, Consigny PM, et al: Greater coronary vascular reserve in dogs anesthetized with halothane. *ANESTHESIOLOGY* 53:445-459, 1980
2. Smith G, Vance JP, Brown DM, et al: Changes in canine myocardial blood flow and oxygen consumption in response to halothane. *Br J Anaesth* 46:821-826, 1974
3. Vance JP, Brown DM, Smith G, et al: Canine coronary blood flow responses to hypoxaemia: the influence of halothane. *Br J Anaesth* 51:193-197, 1979
4. Vatner SF, Smith NT: Effects of halothane on left ventricular function and distribution of regional blood flow in dogs and primates. *Circ Res* 34:155-167, 1974
5. Domenech RJ, Macho P, Valdes J, et al: Coronary vascular resistance during halothane anesthesia. *ANESTHESIOLOGY* 46:236-240, 1977
6. Smith G, Rogers K, Thorburn J: Halothane improves the balance of oxygen supply to demand in acute experimental myocardial ischemia. *Br J Anaesth* 52:577-583, 1980
7. Bland GHL, Lowenstein E: Halothane-induced decrease in experimental myocardial ischemia in the non-failing canine heart. *ANESTHESIOLOGY* 45:287-293, 1976
8. Merin RG: Is anesthesia beneficial for the ischemic heart? *ANESTHESIOLOGY* 53:439-440, 1980

(Accepted for publication March 17, 1981.)

Anesthesiology  
55:480-481, 1981

### Dextran Is Not a Potent Local Anesthetic Adjuvant

*To the Editor:*—I read with interest the letter by R. E. Loder<sup>1</sup> 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.<sup>2-3</sup>

2) Dextran's mechanism of action as a local anesthetic adjuvant appears to be a function of its *pH*. The more alkaline the dextran, the greater the prolongation of block.<sup>2</sup>

3) Pharmaceutical dextrans can vary markedly in *pH*. For example, Rheomacrodex<sup>®</sup> (Pharmacia), the most widely used dextran in the United States, has a *pH* of 4.5 to 5.5 in contrast to Dextraven<sup>®</sup> (Fisons) which has

a neutral *pH*. This difference in *pH* 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 *pH* of the dextrans used in the study and not to their respective molecular weights.<sup>4</sup>

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 diminished since Loder first advocated its use in 1960.<sup>5</sup> The duration of block produced by the

newer long-acting local anesthetics, bupivacaine or etidocaine, is sufficiently prolonged to meet most clinical requirements. Furthermore, the treatment of postoperative thoraco-abdominal pain by intercostal nerve blocks, the primary use for a dextran-local anesthetic mixture, is declining and maybe supplanted altogether by epidural or intrathecal narcotics.

RICHARD M. ROSENBLATT, M.D.  
*Associate Professor*  
*Department of Anesthesiology*  
*The Ohio State University*  
*Columbus, Ohio*

Anesthesiology  
55:481, 1981

### More Experience with Intrathecal Morphine for Obstetric Analgesia

*To the Editor:*—We read with great interest the recent report by Baraka *et al.* in ANESTHESIOLOGY.<sup>1</sup>

Last year we undertook a similar clinical investigation on 13 healthy parturients with term pregnancies who received intrathecal morphine for the relief of their labor pain. The first group of six patients received intrathecal injection of 2 mg morphine sulfate in 1 ml cerebrospinal fluid and only one patient reported a 60 per cent reduction of her labor pain as evaluated by verbal interview as well as visual analogue score in the subsequent 60 min observation.

The second group of seven patients received intrathecal injections of 2 mg morphine sulfate in 4 ml 0.9 per cent saline (isobaric solution with specific gravity of 1.007), and to our great surprise all except one patient in this group obtained 75–90 per cent pain relief lasting 20–36 hours. Our experience with this second group of patients is similar to that reported by Dr. Baraka and his associates.

We unfortunately were unable to pursue our study further because we too observed a disturbingly high incidence of pruritis (73 per cent), vomiting (66 per cent), somnolence (60 per cent), and urinary retention (40 per cent) which were deemed unacceptable to our patients as well as to our obstetric colleague. Furthermore, in another unrelated postoperative pain study we encountered two alarming incidents where a 26-year-old female patient with postoperative pain developed bradypnea and cyanosis 8.5 hours after intrathecal administration of 2 mg morphine sulfate, and a 20-year-old male patient with a hip fracture developed severe respiratory depression 11.5 hours after intrathecal injection of 1 mg morphine sulfate. Our observation coincided with those reported in the literature.<sup>2–6</sup>

### REFERENCES

1. Loder RE: Lidocaine-dextran solutions. ANESTHESIOLOGY 53:522, 1980
2. Rosenblatt RM, Fung DL: Mechanism of action for dextran prolonging regional anesthesia. Regional Anesth 5:3–5, 1980
3. Scurlock JE, Curtis BM: Dextran-local anesthetic interactions. Anesth Analg (Cleve) 59:335–340, 1980
4. Rosenblatt RM, Lui P, Capener C: Dextran as a local anesthetic adjuvant. ANESTHESIOLOGY 3:S220, 1980
5. Loder RE: A local-anesthetic solution with longer action. Lancet 2:346–347, 1960

(Accepted for publication March 17, 1981.)

Theoretically, the prolonged analgesia provided by intrathecal morphine would be ideal for labor pain since it does not cause any motor, sensory, or sympathetic blockade. However, one must bear in mind the questionable safety of this technique, especially in regard to the unpredictable yet potentially lethal complication of delayed respiratory depression which is beginning to appear in the literature worldwide as experience with intrathecal morphine administration expands with increasing number of patients.

MARTIN S. MOK, M.D.  
*Clinical Associate Professor*  
*Department of Anesthesiology*  
*Harbor-UCLA Medical Center*  
*Torrance, California 90509*

S. K. TSAI, M.D.  
*Department of Anesthesia*  
*Veteran General Hospital*  
*Taipei, Taiwan, R.O.C.*

### REFERENCES

1. Baraka A, Noueihid R, Hajj S: Intrathecal injection of morphine for obstetric analgesia. ANESTHESIOLOGY 54:136–140, 1981
2. Glynn CJ, Mather LE, Cousins MJ, et al: Spinal narcotics and respiratory depression. Lancet 2:356–357, 1979
3. Liolios A, Andersen FH: Selective spinal analgesia. Lancet 2:357, 1979
4. Davies GK, Tolhurst-Cleaver CL, James TL: CNS depression from intrathecal morphine. ANESTHESIOLOGY 52:280, 1980
5. Baskoff JD, Watson RL, Muldoon SM: Respiratory arrest after intrathecal morphine. Anesth Review 7:12–15, 1980
6. McDonald AM: Complication of epidural morphine. Anaesth Intensive Care 8:490–491, 1980

(Accepted for publication March 17, 1981.)