

weight, was safe and effective in controlling the emergence-related hypertension in all patients. Both systolic and diastolic hypertension were controlled with minimal changes in heart rate, even at a total dose of 2.5 mg/kg. None of the patients experienced any untoward side effects, such as hypotension, significant bradycardia, bronchospasm, or electrocardiographic changes that may occur following beta- or alpha-adrenergic blocking drugs.

REFERENCES

1. Prys-Roberts C, Greene LT, Meloche R, Foëx P: Studies of anesthesia in relation to hypertension. II: Haemodynamic consequences of induction and endotracheal intubation. *Br J Anaesth* 43:531-541, 1971
2. Wallach R, Karp RB, Reves JG, Oparil S, Smoth LR, James TN: Pathogenesis of paroxysmal hypertension developing during and after coronary bypass surgery: A study of hemodynamic and humoral factors. *Am J Cardiol* 46:559-565, 1980
3. Cumming AMM, Brown JJ, Lever AF, Robertson JIS: Intravenous labetalol in the treatment of severe hypertension. *Br J Clin Pharmacol* 13 (Suppl):93s-96s, 1982
4. Ronne-Rasmussen JO, Anderson GS, Bowal-Jensen N, Andersson E: Acute effect of intravenous labetalol in the treatment of systemic arterial hypertension. *Br J Clin Pharmacol (Suppl)*:805s-808s, 1976
5. Bhatia SK, Frohlich ED: Hemodynamic comparison of agents useful in hypertensive emergencies. *Am Heart J* 85:367-373, 1973
6. Cressman MD, Vidt DG, Gifford RW, Moore WS, Wilson DJ: Intravenous labetalol in the management of severe hypertension and hypertensive emergencies. *Am Heart J* 107:980-985, 1984
7. MacCarthy EP, Bloomfield SS: Labetalol: A review of its pharmacology, pharmacokinetics, clinical uses and adverse effects. *Pharmacotherapy* 3:193-219, 1983
8. Sybertz EJ, Sabin CS, Pula KK, Vander Vliet G, Glennon J, Gold EH, Baum T: Alpha and beta-adrenoceptor blocking properties of labetalol and its R,R-isomer, SCH 19927. *J Pharmacol Exp Ther* 218:435-443, 1981
9. Richards DA, Tuckman J, Prichard BNC: Assessment of alpha- and beta-adrenoceptor blocking actions of labetalol. *Br J Pharmacol* 3:849-855, 1976
10. Richards DA, Prichard BNC, Boakes AJ, Tuckman I, Knight EJ: Pharmacological basis for antihypertensive effects of intravenous labetalol. *Br Heart J* 39:99-106, 1977
11. Richards DA, Prichard BNC, Dobbs RJ: Adrenoceptor blockade of the circulatory response to intravenous isoproterenol. *Clin Pharmacol Ther* 24:264-273, 1978

Anesthesiology
67:416-418, 1987

Epinephrine Prolongs Lidocaine Spinal: Pain in the Operative Site the Most Accurate Method of Determining Local Anesthetic Duration

D. C. MOORE, M.D.,* H. S. CHADWICK, M.D.,† L. B. READY, M.D.‡

Previous investigations report conflicting information with regard to the effect of adding epinephrine to solutions of hyperbaric lidocaine for spinal anesthesia. Chambers *et al.* concluded that anesthesia was not significantly prolonged by the addition of epinephrine to lidocaine for spinal anesthesia in patients undergoing transurethral prostatectomy.¹ Spivey arrived at the same conclusion after studying women undergoing vaginal deliveries.² Both studies involved the use of thoracic dermatome regression of sensory level to define duration of the block. Leicht and Carlson, however, also using such regression, found that the addition of epinephrine did prolong the analgesia of lidocaine spinal blockade for lower abdominal surgery.³

We question whether thoracic dermatome regression accurately reflects the duration of spinal blockade for surgical procedures involving the perineum and lower extremities which are innervated by the lumbosacral nerves. To clarify this issue, a blinded study was undertaken using hyperbaric lidocaine spinal anesthesia with or without the addition of epinephrine in day-care patients undergoing arthroscopic knee procedures. The definitive endpoint of subjective discomfort was used to determine the duration of anesthetic action.

METHODS

Institutional Human Subjects Review Committee approval was obtained for a study involving 40 patients. Patients were assigned to one of two groups. Group 1 patients received spinal anesthesia with lidocaine not containing epinephrine. Group 2 patients received spinal anesthesia with lidocaine containing epinephrine. Informed consent was obtained, and the possibility of a headache or backache, and the use of a blood patch to treat headache, were explained. Patients were told that

* Clinical Professor.

† Assistant Professor.

‡ Associate Professor.

Received from the Department of Anesthesiology RN-10, University of Washington School of Medicine, Seattle, Washington 98195. Accepted for publication March 30, 1987.

Address reprint requests to Dr. Moore.

Key words: Anesthesia: outpatient. Anesthetic technique: spinal.

a short- rather than a long-acting local anesthetic would be used to avoid a prolonged postoperative stay, and that, if the anesthesia dissipated prior to the completion of surgery, immediate supplementary anesthesia would be administered.

Diazepam 10 mg and fentanyl 0.1 mg were administered iv for sedation. The patients were placed in the lateral decubitus position with the operative side in the dependent position. Lumbar puncture was performed at the L2-3 interspace employing a 26-gauge single-use spinal needle placed through a 20-gauge single-use introducer. Free flow of cerebrospinal fluid was obtained prior to and after injection of the local anesthetic solution. The person administering the spinal block, a resident physician or nurse anesthetist (CRNA), had no knowledge of whether the local anesthetic contained epinephrine.

The choice of which local anesthetic solution would be used was made by the staff anesthesiologist, based on the scheduled duration of the surgical procedure. Lidocaine was chosen for surgery scheduled to last 1.5 h or less, and lidocaine with epinephrine if the procedure was scheduled to last longer. The staff anesthesiologist prepared the local anesthetic solution. The lidocaine dosage was 60 mg (1.2 ml of 5% lidocaine in 7.5% dextrose), to which either 0.2 mg epinephrine (0.2 ml of a 1:1000 concentration) or 0.2 ml sterile water was added, so that the volume in all syringes was always identical (1.4 ml).

The resident or CRNA involved in the cases recorded the following variables: 1) the time of injection of the local anesthetic into the subarachnoid space; 2) the thoracic dermatome level of anesthesia to pin prick achieved at 20 min; 3) the time of onset of pain from either the tourniquet or surgical manipulation; 4) the time of tourniquet deflation; 5) the time surgery ended; and 6) the length of stay in the recovery room. The resident or CRNA administered supplemental iv or general anesthesia, as necessary, when pain occurred. Pain was taken as the endpoint defining anesthetic duration.

Fisher's exact test was used to compare patient's sex, ASA physical status scores, and number of thoracic dermatome levels blocked. Unpaired Student's *t* test was employed to analyze the remaining data. A *P* ≤ 0.05 was considered to be statistically significant.

RESULTS

Twelve residents and three CRNAs participated in the investigation. Twenty patients were studied in each group. None of the patients requested or, because of restlessness, required additional sedation prior to reporting pain.

TABLE 1. Variables with No Significant Differences

	Lidocaine	Lidocaine with Epinephrine
Sex		
Males	18	18
Females	2	2
Physical Status		
Age (yr)*	33 ± 11	33 ± 13
Height (cm)*	174 ± 10	174 ± 12
Weight (kg)*	82 ± 10	86 ± 28
ASA Score		
1	18	16
2	2	4
Upper range of sensory anesthesia	T ₉ -T ₁	T ₈ -C ₅ †
Duration of tourniquet inflation (min)*	70 ± 15	80 ± 19

* Mean ± SD.

† In one patient, the level reached C5; in the others, the maximum dermatome level was T₁.

Mean age, height, weight, ASA physical status scores, sex distribution, and number of dermatomes blocked were similar for each group (table 1). In group 1, ten meniscectomies were performed, compared to 11 in group 2. The duration of surgery was significantly longer (*P* < 0.05) in group 2 (131 ± 42 min) compared to group 1 (109 ± 22 min) (mean ± SD). Similarly, the tourniquet inflation time tended to be longer in group 2, although this did not reach statistical significance (table 1). The duration of anesthesia prior to the occurrence of pain was 87 ± 16 min in group 1, compared to 128 ± 23 min in group 2, representing a highly significant difference (*P* < 0.0005) (fig. 1).

The mean recovery room stay for patients in group 1 was 133 ± 44 min, compared to 149 ± 41 min for those

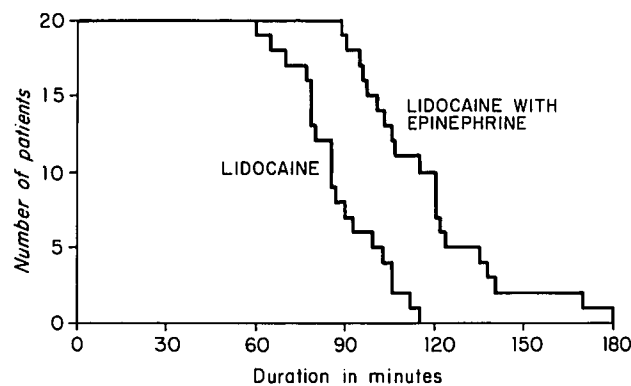


FIG. 1. The graph indicates the number of patients having at least a given duration of spinal anesthesia. In constructing the graph, when surgery ended prior to any report of pain, the surgery end-time was used in determining anesthetic duration.

in group 2 (not significant). No post-anesthetic complications, such as headache or backache, were reported.

DISCUSSION

The classical means of determining the duration of a local anesthetic injected into the subarachnoid space has been to document thoracic dermatome anesthetic regression as measured with pin-prick testing.⁴ To avoid the inaccuracies which might be caused by such an indirect measure, we chose the definitive endpoint of surgical pain to determine duration of action.

Kosody *et al.* showed that dogs given 0.2 mg epinephrine into the subarachnoid space developed dural vasoconstriction primarily in the lumbosacral area.^{5,6} These findings indicate that epinephrine may have a differential vasoconstrictive effect at different segmental levels. If these findings can be extrapolated to local anesthetics containing epinephrine in humans, it would explain why thoracic dermatome regression may not be an accurate method for determining effective local anesthetic duration in the lumbosacral area.

The length of stay in the recovery room did not differ significantly between groups, and was judged to be adequately short for day-care patients. Because of this and the fact that none of our patients reported complications, we believe that spinal anesthesia is a satisfactory option for day-care patients.

Although patients were not assigned to their respective groups in a random fashion, neither the patients nor the resident or CRNA observers were aware of the selection protocol or of which drug solutions were being used. Since actual surgery time often differed consider-

ably from scheduled surgical time, and because of the similar normative data between groups, we believe that the groups were well matched (table 1).

In conclusion, we found that epinephrine significantly prolongs the duration of hyperbaric lidocaine spinal anesthesia for arthroscopic knee procedures. While thoracic dermatome regression of anesthesia may be an appropriate method of studying the duration of action of spinal anesthetics containing epinephrine for upper abdominal surgery, we believe it is unreliable for procedures involving areas innervated by lumbosacral nerves.

The authors wish to acknowledge the following anesthesia residents who participated in this study: S. M. Audenaert, R. L. Claypool, A. G. Gruber, E. D. Kharasch, J. J. Mulroy, C. A. Padavich, D. M. Polaner, A. A. Schultz, S. L. Stoops, D. M. Stout, G. A. Van Norman, K. T. Y. Wong, as well as CRNAs T. V. Cicero, D. L. Milholland, and B. T. Walker.

REFERENCES

1. Chambers WA, Littlewood DG, Logan MR, Scott DB: Effect of added epinephrine on spinal anesthesia with lidocaine. *Anesth Analg* 60:417-420, 1981
2. Spivey DL: Epinephrine does not prolong lidocaine spinal anesthesia in term parturients. *Anesth Analg* 64:468-470, 1985
3. Leicht CH, Carlson SA: Prolongation of lidocaine spinal anesthesia with epinephrine and phenylephrine. *Anesth Analg* 65:365-369, 1986
4. Greene NM: Uptake and elimination of local anesthetics during spinal anesthesia. *Anesth Analg* 62:1013-1024, 1983
5. Kosody R, Palahniuk RJ, Wade JG, Cumming MD: The effect of subarachnoid epinephrine and phenylephrine on spinal cord blood supply. *Can Anaesth Soc J* 31:503-508, 1984
6. Kosody R, Swartz J, Palahniuk RJ, Biehl DR, Wade JG: Spinal cord blood flow following subarachnoid lidocaine. *Can Anaesth Soc J* 32:472-478, 1985