Guggiari *et al.*[9] in a series of head-injury patients receiving nimodipine, should be of value by reducing transmural pressure within the aneurysm. However, nimodipine could compromise cerebral perfusion by reducing arterial blood pressure if vasospasm were present or by increasing intracranial pressure if it were already high. Since patients with subarachnoid hemorrhage may have intracranial hypertension because of mass effect, cerebral infarction, and edema or hydrocephalus, the potential additive effects on cerebral blood volume and intracranial pressure of nimodipine and vasodilator anesthetics cause concern and suggest the need for further prospective studies on the effects of nimodipine on cerebral blood flow and on the influence of nimodipine on the response of patients with subarachnoid hemorrhage to anesthetic drugs.

In conclusion, we reviewed our experience with the anesthetic management of patients who received nimodipine or placebo in a double-blind study on the effects of nimodipine prophylaxis on cerebral vasospasm. Nimodipine produced small, apparently desirable alterations in perioperative hemodynamic variables and did not otherwise appear to affect anesthetic management.

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Epidural Blockade for Cesarean Section Comparing Lidocaine Hydrocarbonate and Lidocaine Hydrochloride

COLM P. COLE, M.D.,* GRAHAM H. MCMORLAND, M.B., F.R.C.P.(C),† JAMES E. AXELSON, PH.D.,‡ LEONARD C. JENKINS, M.D., F.R.C.P.(C)§

Lidocaine hydrocarbonate (Xylocaine CO_2®), when compared with lidocaine hydrochloride for regional analgesia, including labor and delivery, has been reported to have a faster onset and spread, while producing a more intense motor block.[1] The purpose of this study was to compare in a randomized, prospective, double-blind fashion, lidocaine–HCl and lidocaine–CO_2 in terms

* Resident in Anesthesiology.

† Clinical Associate Professor of Anesthesiology and Associate in Obstetrics and Gynecology; Head, Division of Obstetric Anesthesia.

‡ Professor of Biopharmaceutics and Pharmacokinetics.

§ Professor and Head, Department of Anesthesiology.

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Address reprint requests to Dr. Cole: Department of Anesthesiology, Grace Hospital, 4490 Oak Street, Vancouver, B. C., Canada, V6H 3V5.

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of onset, duration, and serum concentrations reached during epidural blockade for cesarean section.

Materials and Methods

Twenty patients, all ASA I and II, undergoing elective cesarean sections under epidural blockade, randomly were assigned to a lidocaine–HCl (2%) or to a carbonated lidocaine (1.73%) group after informed consent and institutional medical research committee approval was obtained. (Both lidocaine solutions contained 17.3 mg of lidocaine base per milliliter).

All patients received the same anesthetic technique. This consisted of sodium citrate premedication (30 ml 0.3 M po 2 h preoperatively), intravenous preloading (Ringer's lactate 2,000 ml), and insertion of the epidural catheter at the L2–3 interspace, with the patient in the left-lateral position. A 17-gauge Tuohy needle was used, employing 1 ml 0.25% bupivacaine for skin anesthesia and a loss of resistance technique using normal saline to identify the epidural space. The patient then was placed in the supine position with 15 degrees of left-lateral tilt and supplemental oxygen applied (6 l/min flow via face mask). Both groups were similar in terms of maternal age, weight, and parity. The only difference between the two groups was the agent used, lidocaine–CO_2, or lidocaine–HCl, both without epinephrine.

The anesthetic agent was administered via the epidural catheter as follows: a test dose of 2 ml was administered at time zero. Three minutes later, a dose of 6 ml was administered that was repeated every 3 min until a total dose of 20 ml had been given (9 min after time zero) or the sensory blockade had reached an adequate level. If further anesthesia was required, additional agent was given in 5-ml increments every 5 min until the patient was deemed sufficiently anesthetized for surgery to commence. After this time, no further agent was given until the duration of the anesthetic blockade had been determined.

The carbonated lidocaine slowly was drawn up, using an 18-gauge needle and a 20-ml syringe immediately prior to its administration. The syringe remained capped between administrations of the agent.

The anesthesiologist administering the agent and evaluating its effect was not aware of the identity of the agent being used. The onset of sensory blockade was evaluated by the patient's subjective impression of onset and by analgesia to pin prink and ice. Spread of the block was evaluated every 2 min using ice with the end points of bilateral anesthesia to S2, 3, 4, and T4 and of the time of furthest dermatome spread or complete spread being recorded. The duration was recorded as that time from time zero (administration of the test dose) to the time the block had regressed two segments from the complete spread level. Motor block was assessed using the Bromage[1] scale from 0 to 4, with a 4 being assigned to that patient unable to move her feet when complete spread was reached and a score of 0 assigned to that patient able to move both hips and knees.

TABLE 1. Summary of Recorded Variables*

	Lidocaine–CO_2 (N = 10)	Lidocaine–HCl (N = 10)
Dose		
mg	404 ± 11	388 ± 4
ml	20.2 ± 0.5	19.4 ± 0.2
Onset (min)		
Subjective	5.5 ± 0.90	6.5 ± .86
Objective	7.1 ± 1.4	7.9 ± 1.3
Spread (min)		
S2, 3, 4	13.2 ± 3.7	12.9 ± 1.9
T4	17.6 ± 6.3	16.8 ± 3.9
Comp.	21 ± 5.9	20 ± 6.5
Duration (min)	61 ± 8.9	66 ± 11
Serum concentration (μg/ml)	2.3 ± 0.59	2.4 ± 0.41

* Mean ± SD.

At the time of delivery, a maternal venous blood sample was taken to measure serum lidocaine levels. This sample was centrifuged, the serum frozen, and the Enzyme Multiple Inhibition Technique[2] used to assay serum concentrations, which was done in a blind fashion. The day-to-day precision of the assay was better than 3.1% at a drug concentration of 3.2 mg/l and better than 5.6% at a drug concentration of 8.0 mg/l. The assay was performed by the Emit® technology, with a system CP5000 and an auto-carousel.¶

Basic routine maternal and fetal cardiovascular monitoring continued throughout the precedure, with any unusual events recorded and treated accordingly.

The recorded variables in the two groups (except for motor block) were compared using a Student's t test with a P value of less than 0.05 considered significant.

Results

There were 10 patients in each group. The results, which are expressed as means ± SD, are shown in table 1. There was no statistically significant difference between the two groups in any of the recorded variables. The total doses required to attain satisfactory anaesthetic levels were similar (lidocaine CO_2 404 ± 11 mg, lidocaine–HCl 388 ± 4 mg). The lidocaine–HCl group had less variability, with nine patients receiving 20 ml of

¶ Syva, Palo Alto, California 94304.

anaesthetic solution and one patient 14 ml; while in the lidocaine–CO_2 group, five patients received 20 ml, three required 14 ml, and two required 30 ml.

The onset of the block was about 1 min faster in the lidocaine CO_2 group, which is not statistically significant. Both agents were similar in onset time (approximately 6 min); the total time taken to spread to the S2, 3, 4 dermatomes (approximately 13 min) and to the T4 dermatome (approximately 17 min).

Duration of the block was also similar (approximately 60 min) from the initial administration of the agent. It should be noted that some patients required parenteral fentanyl for visceral pain sensation before the block had regressed the required two segments, to fulfill the criteria of duration in this study. Narcotic supplementation was similar in the two groups with an average of 80 μg of fentanyl in the CO_2 group and 65 μg of fentanyl in the hydrochloride group.

Motor block was not significantly different in the two groups.

There were no observed side effects in terms of sensorium changes, blood pressure, or pulse changes, other than eight of the 20 patients who developed moderate hypotension, which responded rapidly to a small dose (5 mg) of intravenous ephedrine (five in the lidocaine–HCl group, three in the lidocaine–CO_2 group). Moderate hypotension was defined as a decrease in systolic blood pressure of *less* than 30% and to an absolute value of greater than 90 mmHg.

Discussion

Bromage[1] found that the quality of epidural blockade was improved when lidocaine base was dissolved as salts of carbonic acid at P_{CO_2} of 1 atmosphere. He stated that the "carbonated lidocaine" was noted to have a 20–30% shorter latency period, prior to onset of the block, than lidocaine hydrochloride and achieved complete blockade 33% faster. The present study, assessing the two forms of the drug in a prospective, randomized, double-blind manner, did not repeat those findings.

We were unable to confirm Bromage's statement that "analgesia resulting from the CO_2–base solutions is superior in every respect to the blockade produced by equivalent concentration of the hydrochloride salts." While our previous clinical impression agreed with Bromage's statement, this was not confirmed when it was evaluated in a controlled, double-blind manner, in epidural anesthesia for cesarean section.

Other double-blind studies,[3,4] in contrast to our study, have found an increased efficacy of carbonated lidocaine in anesthetizing the lumbosacral nerve roots. This difference may be partly explained by their use of a more upright position and addition of epinephrine to the solutions.

Side effects of the drugs and technique, which we encountered, were minor. The technique achieved patient comfort with minimal need for narcotic supplementation, following the birth of the infant. Despite left lateral tilt of the patient and intravenous preloading with Ringer's lactate solution, there was an incidence (eight of 20 patients) of moderate hypotension (less than a 30% drop in systolic pressure). This was not clinically troublesome and responded very readily to small doses of ephedrine.

In summary, it can be stated that lidocaine hydrocarbonate and lidocaine hydrochloride, used in our study to produce epidural anesthesia for elective cesarean section, are similar and equally effective in terms of onset, spread, and duration of blockade, when used as the sole agents and without addition of epinephrine.

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