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Effects of Maternal Position on Epidural Anesthesia for Cesarean Section, Acid–Base Status, and Bupivacaine Concentrations at Delivery

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In 25 patients excellent clinical anesthesia for elective cesarean section was obtained with lumbar epidural block using an average dose of bupivacaine of 130 mg (18 ml of 0.75 per cent solution). Supplemental drugs were not needed. All infants had normal Apgar scores at delivery. Ten patients were kept in a 35–40-degree semi-sitting supine position during induction, while 15 patients were similarly semi-sitting but turned into the left lateral position. Maternal position did not affect the adequacy of the anesthesia or the clinical condition of the infants, but did alter acid–base state and bupivacaine concentrations in the infants. At delivery, the infants whose mothers had been supine had significantly lower *p*H values in umbilical cord blood than those whose mothers had been in the lateral position. Also, higher concentrations of bupivacaine were found in the umbilical vein blood of infants whose mothers were supine. (Key words: Anesthesia: obstetric. Anesthetic techniques, regional: lumbar epidural. Anesthetics, local: bupivacaine. Acid–base equilibrium: fetal.)

THE USE of lumbar epidural anesthesia for elective cesarean section is increasing, yet there continues to be uncertainty about the details of technique, drug dose, and outcome. This paper presents and assesses an anesthetic technique using bupivacaine, 0.75 per cent. We present the effects of altering maternal position during induction of anesthesia on the adequacy of anesthesia, fetal outcome, maternal and fetal acid–base status, and blood levels of bupivacaine at delivery.

Methods

Twenty-five healthy women, without maternal or fetal complications, undergoing elective primary or repeat cesarean section at term, were selected at random. Informed consent was obtained in accordance with the protocol approved by the Research Advisory Committee of the Boston Hospital for Women. With an intravenous infusion of dextrose, 5 per cent, in lactated Ringer's solution established, the patients were placed in the left lateral decubitus position. An epidural catheter was inserted at the L2–3 interspace and directed 1–2 cm caudad. A test dose of bupivacaine, 2 ml of 0.75 per cent (15 mg), without epinephrine, was injected. All patients were then placed in the supine semi-sitting position, with the head up at a 35–40-degree angle. After 3–5 min had elapsed without evidence of subarachnoid or intravascular injection, the remainder of the bupivacaine was administered to a total dose, which averaged 130 mg (18 ml of 0.75 per cent solution).

All patients were kept supine and semi-sitting for about 10 min until the level of hypesthesia to pin prick had reached T10. In the ten patients in Group A, the uterus was not displaced during this time; in the 15 patients in Group B, the uterus was manually displaced to the left. For the next 30 min, during the development of full surgical anesthesia, the patients in Group A remained supine and semi-sitting, while those in Group B were kept in the left lateral decubitus position with the head up at the same angle.

The patients were then taken to the operating room, where approximately 20 min elapsed during preparation for operation and during delivery of the infant. During this period, all patients in both groups were supine and tilted to the left with a blanket roll under the right hip and torso. In the operating room, all patients received oxygen through a plastic face mask at a flow rate of 6 l/min.

In all patients, 1 liter of intravenous fluid was infused within 15 min following institution of epidural anesthesia and a second liter of fluid was infused slowly thereafter. Maternal blood pressure and heart rate were measured every minute for the first 15 min and every 3 min thereafter. Fetal heart rate was measured by auscultation at 5-min intervals. Maternal hypotension was considered to be present when systolic blood pressure decreased more than 30 torr or to below 100 torr. Hypotension was immediately corrected by left uterine displacement and increasing the rate of intravenous fluid infusion. In no instance did hypotension last longer than 2 min. Vasopressor drugs were not given.

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TABLE 1. Patient Characteristics

	Group A Supine (n = 10)	Group B Lateral (n = 15)
Maternal age (years)	30 ± 1.7*	29 ± 1.6
Maternal height (in)	64 ± 1	63 ± 1
Maternal weight (lb)	159 ± 6	152 ± 6
Gestational age (weeks)	40 ± 0.2	40 ± 0.2
Total dose of bupivacaine (mg)	130 ± 8	129 ± 4
Induction–delivery interval (min)	58 ± 2	59 ± 2
Infant birth weight (g)	3556 ± 120	3660 ± 80
Apgar score		
1 min	8 (7–9)†	9 (7–9)
5 min	9 (8–10)	9 (9–10)

* Mean ± SE.
† Median (range).

The time from the injection of the final dose of local anesthetic to the delivery of the infant was recorded as the induction–delivery interval. At delivery, samples of blood from a maternal radial artery and an antecubital vein were collected, as well as samples of umbilical artery and vein blood from a doubly clamped segment of umbilical cord. Blood-gas and *p*H values were immediately determined for all samples in duplicate with a Radiometer microelectrode system. Base deficit was calculated using the Siggaard-Andersen nomogram.[1] The concentrations of bupivacaine in the blood samples were determined by a previously described gas chromatographic technique sensitive to a concentration of 0.02 μg/ml of bupivacaine with a reproducibility of ±10 per cent.[2] The data were analyzed statistically using t tests for unpaired data and the chi-square method.

Results

A comparison of the two groups of patients (table 1) revealed no significant difference in maternal ages, heights and weights, or gestational ages and weights of the infants. In both groups, excellent surgical anesthesia was obtained with a sensory level to pin prick of at least T6; supplementation was not necessary either before or after delivery of the infant. The average total doses of bupivacaine were identical in the two groups, as were the induction–delivery intervals. Maternal hypotension was seen in three of ten parturients in Group A (supine) and in two of 15 in Group B (lateral), not a statistically significant difference. Since acid–base and bupivacaine values in the hypotensive patients did not differ significantly from those in patients who remained normotensive, the data were combined for analysis.

No abnormality of fetal heart rate was detected during anesthesia, confirming the findings of others who have used bupivacaine to provide epidural anesthesia for cesarean delivery.[3,4] All infants had Apgar scores of 7 or more at both 1 and 5 min of age, had uncomplicated hospital stays, and were discharged with their mothers. The average *p*H of maternal arterial blood (MA) was significantly lower in the parturients of Group A than in those of Group B, but there was no difference in base deficits (table 2). Examination of umbilical vein (UV) blood revealed significantly lower *p*H and higher P_{CO_2} values for the patients who had been in the supine position (Group A). Similarly, in umbilical artery (UA) blood, the average *p*H was significantly lower for the supine group. The calculated base deficit was significantly greater in both umbilical vein and umbilical artery blood of Group A. The fetal–maternal base deficit difference (Δ base deficit), calculated by subtracting maternal artery blood base deficit from umbilical artery blood base deficit (UA − MA), averaged 1.3 mEq/l in the lateral Group B, compared with 6.4 mEq/l in the supine Group A, a highly significant difference.

The average concentration of bupivacaine in maternal venous blood in the supine Group A (0.64 μg/ml) was not significantly higher than that in the lateral Group B (0.54 μg/ml) (table 3). There was a statistically significantly higher concentration of bupivacaine in the umbilical vein blood of the supine group but not in the umbilical artery blood. Sub-

TABLE 2. Acid–Base and Blood-gas Data

	Group A Supine (n = 10)	Group B Lateral (n = 15)
Maternal artery (MA)		
*p*H	7.39 ± 0.007*	7.42 ± 0.008†
P_{O_2} (torr)	204 ± 6	203 ± 8
P_{CO_2} (torr)	34 ± 1.6	31 ± 1.3
Base deficit (mEq/l)	2.8 ± 0.6	2.1 ± 0.6
Umbilical vein (UV)		
*p*H	7.28 ± 0.006	7.34 ± 0.01†
P_{O_2} (torr)	31 ± 1.4	33 ± 1.2
P_{CO_2} (torr)	47 ± 0.7	39 ± 1.4†
Base deficit (mEq/l)	6.3 ± 0.5	4.3 ± 0.7†
Umbilical artery (UA)		
*p*H	7.20 ± 0.01	7.30 ± 0.007†
P_{O_2} (torr)	16 ± 1.5	21 ± 1.7
P_{CO_2} (torr)	58 ± 2.8	52 ± 2.1
Base deficit (mEq/l)	9.2 ± 0.9	3.4 ± 0.7†
Δ Base deficit: UA base deficit − MA base deficit (mEq/l)	6.4 ± 1.2	1.3 ± 0.5†

* Mean ± SE.
† $P < 0.05$.

traction of the mean umbilical artery value from that for the umbilical vein yielded average bupivacaine concentration differences of 0.07 μg/ml in the supine group and 0.02 μg/ml in the lateral group.

Calculation of the fetal/maternal concentration ratios (UV/MV) gave values of 0.42 in Group A and 0.31 in Group B.

Discussion

Left uterine displacement in pregnant patients at term undergoing anesthesia for either vaginal or cesarean delivery has been widely accepted. Nevertheless, in a recent survey of obstetric anesthesia services,§ 43 per cent of hospitals reported omission of this practice when epidural anesthesia was used for cesarean section. It had also not been our practice at the Boston Hospital for Women because of the impression that tilting resulted in a higher incidence of inadequate anesthesia. This notion was supported by two recent reports[5,6] where patients were kept in the lateral position throughout induction of epidural anesthesia for cesarean section. In the first,[5] seven of 13 patients reported inadequate anesthesia; in the second,[6] only seven of 15 anesthesias were rated as excellent by the anesthesiologist.

In designing the protocol, the semi-sitting position during induction was selected for all patients in an attempt to ensure adequate block of the sacral nerves so as to obviate maternal pain during delivery of the presenting part from the pelvis and traction on the vagina. In addition, as Ueland *et al.* have shown,[7] cardiac output at term is higher in the sitting position than in the supine position. Finally, since posture affects spread of local anesthetic in the epidural space,[8] the dose of bupivacaine we selected was 25 per cent higher than that in the two reported series,[5,6] in an attempt to obtain adequate anesthesia, both cephalad and caudad.

Clinically, excellent anesthesia was achieved in both groups of patients, with an upper sensory level to pin prick of at least T6 and excellent sacral anesthesia. No patient needed supplemental drugs either before or after delivery of the infant. The incidence of maternal hypotension was 30 per cent in the supine group, compared with 14 per cent in the lateral group, not a statistically significant difference in this small group of patients. Hypotension in all cases was transient and easily and rapidly corrected without the use of vasopressor drugs. At delivery,

§ Clark RB, Ostheimer GW: S.O.A.P. survey: Cesarean section with left uterine displacement. Anesthesiology Review 3:21–22, 1976.

TABLE 3. Bupivacaine Concentrations (μg/ml)

	Group A Supine (n = 10)	Group B Lateral (n = 15)
Maternal vein (MV)	0.64 ± 0.06*	0.54 ± 0.03
Umbilical vein (UV)	0.26 ± 0.04	0.16 ± 0.02†
Umbilical artery (UA)	0.19 ± 0.04	0.14 ± 0.02
Concentration ratio: UV/MV	0.42 ± 0.05	0.31 ± 0.02†
Concentration difference: UV − UA	0.07 ± 0.01	0.02 ± 0.01†

* Mean ± SE.
† $P < 0.05$.

Apgar scores, acid–base status, and bupivacaine levels were not different in those patients who had been hypotensive compared with those who had remained normotensive.

No change in fetal heart rate was detected in either group of patients, and all infants were delivered in excellent clinical condition with normal Apgar scores. Thus, both maternal and fetal outcomes were excellent using the usual clinical criteria.

However, there were significant differences between the two groups in acid–base values. While maternal arterial blood *p*H was normal in either group, the average value in the supine group (7.39) was lower than that in the lateral group (7.42). While statistically significant, this difference is probably not of biologic importance, nor is its cause apparent. Blood-gas values and base deficits did not differ between the two groups. In four reports of epidural anesthesia for cesarean section,[5,6,9,10] maternal arterial blood *p*H values at delivery ranged from a low of 7.37[5] to a high of 7.44[6]; in both the latter series, the patients had been tilted throughout. In patients who had remained supine, maternal arterial blood *p*H values averaged 7.38[9] and 7.43.[10] It appears that maternal position during epidural anesthesia neither substantially nor consistently affects maternal acid–base status.

On the other hand, we did observe a difference between the two groups of patients in the acid–base values of umbilical cord blood. In umbilical vein blood of the fetuses in Group A, whose mothers had been supine, *p*H values averaged 7.28; P_{CO_2}s, 47 torr; base deficits, 6.3 mEq/l. All three of these values differed significantly from those of fetuses in Group B, whose mothers had been in the lateral position (table 2). In umbilical artery blood, *p*H was significantly higher and base deficit, significantly lower, in the fetuses of Group B. The umbilical vein and umbilical artery blood P_{O_2} values did not differ between the two groups.

The most striking finding was the almost threefold higher base deficit in umbilical artery blood in the supine group compared with the lateral group (9.2 *vs.* 3.4 mEq/l). The calculated fetal–maternal base deficit difference (UA − MA) was five times greater for the fetuses of Group A compared with Group B.

These data suggest a clear advantage of the lateral position with respect to fetal acid–base status. It should be pointed out, however, that all of the values for umbilical cord blood in both groups of patients fell in the ranges accepted as normal for fetal scalp blood in labor[11] and umbilical cord blood at delivery.[12] In addition, none of the infants was clinically depressed, as evidenced by normal Apgar scores following delivery.

The blood-gas and acid–base findings in umbilical cord blood of fetuses in Group A suggest that the acidosis was largely metabolic and of fetal origin, despite the normal values of P_{O_2}. Since the only important difference in the management of these two groups of patients was maternal position, one may speculate that the fetal acidosis was the result of maternal supine position during induction of anesthesia with aortocaval compression, interference with placental blood flow and gas exchange, fetal or placental anaerobic metabolism and acidosis. The biochemical determinants and biologic significance of the acidosis remain to be elucidated.

Our data also suggest that the supine position and the associated fetal acidosis affect the blood levels of bupivacaine in fetal blood at delivery (table 3). Despite identical maternal doses of local anesthetic and induction–delivery intervals, the average concentration of bupivacaine at delivery was significantly higher in the umbilical vein blood for the fetuses of Group A, as was the fetal/maternal (UV/MV) concentration ratio.

Magno *et al.*[4] reported a fetal/maternal concentration ratio of bupivacaine of 0.41 in their series of epidural anesthesias for cesarean section where the maternal arterial blood *p*H values averaged 7.37 and umbilical vein blood *p*Hs, 7.29, values very close to those we observed in Group A. By contrast, in our study of bupivacaine levels following epidural block for vaginal delivery,[13] the fetal/maternal concentration ratios averaged 0.27 and umbilical vein blood *p*Hs, 7.37, values virtually identical to those found in the patients of Group B in the present series.

One hypothesis that may explain these differences invokes the phenomenon of "ion trapping": increased concentration of a weak base, the local anesthetic, on the more acid side of a diffusion barrier, the placenta. We have previously shown with lumbar epidural anesthesia for vaginal delivery[14] an association between fetal acidosis and unusually high fetal/maternal concentration ratios of lidocaine and mepivacaine. Experimentally, this association has also been demonstrated with lidocaine in pregnant sheep.[¶,15] Other mechanisms may be involved, such as alterations in fetal regional and umbilical blood flows, decreased fetal clearance of local anesthetic, altered volumes of distribution, or changes in binding of local anesthetic in blood and tissues.

The final interesting finding was the higher UV − UA concentration difference of bupivacaine that we observed in the more acidotic fetuses of Group A. Sjostrand and Widman[16] compared the tissue distributions of labelled bupivacaine in acidotic and non-acidotic rabbits. They found higher concentrations of bupivacaine in the lungs, livers, and kidneys of the acidotic animals. They suggested that the more acidotic tissues had a higher affinity for bupivacaine. In the same way, more bupivacaine may have been taken up by the tissues of the more acidotic fetuses in Group A, thus causing the higher UV − UA concentration difference. One may then speculate further that fetal acidosis may result not only in higher umbilical vein blood levels of local anesthetic but also in higher levels in tissues, and perhaps greater net transfer of drug from mother to fetus over time.

In summary, we have described a technique of lumbar epidural block with bupivacaine for cesarean section where excellent anesthesia was achieved in two groups of patients. In one group, the patients remained supine and semi-sitting throughout induction; in the other, the patients were in the left lateral decubitus, semi-sitting position. Positioning the mother on her side affected neither the adequacy of the block nor the clinical condition of the neonate, but did result in a higher *p*H and lower base deficit values in umbilical cord blood and a lower concentration of bupivacaine in umbilical vein blood at delivery.

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References

1. Siggaard-Andersen O: Blood acid–base alignment nomogram. Scand J Clin Lab Invest 15:211–217, 1963
2. Lurie AO, Weiss JB: Blood concentration of mepivacaine

¶ Radosevich FA, Kennedy RL, Turner T, et al: The effect of maternal and fetal *p*H changes on placental transfer of lidocaine. Abstracts of Scientific Papers, Annual Meeting of the American Society of Anesthesiologists, 1976, pp 533–534.

and lidocaine in mother and baby after epidural anesthesia. Am J Obstet Gynecol 106:850–856, 1970

3. Belfrage P, Irestedt L, Raabe N, et al: General anaesthesia or lumbar epidural block for caesarean section? Effects on the foetal heart rate. Acta Anaesthesiol Scand 21:67–70, 1977
4. Magno R, Berlin A, Karlsson K, et al: Anesthesia for cesarean section. IV: Placental transfer and neonatal elimination of bupivacaine following epidural analgesia for elective cesarean section. Acta Anaesthesiol Scand 20:141–146, 1976
5. Magno R, Kjellmer I, Karlsson K: Anesthesia for cesarean section. III: Effects of epidural analgesia on the respiratory adaptation of the newborn in elective cesarean section. Acta Anaesthesiol Scand 20:73–82, 1976
6. James FM, Crawford JS, Hopkinson R, et al: A comparison of general anesthesia and lumbar epidural analgesia for elective cesarean section. Anesth Analg (Cleve) 56: 228–235, 1977
7. Ueland K, Novy MJ, Peterson EN, et al: Maternal cardiovascular dynamics: IV. The influence of gestational age on the maternal cardiovascular response to posture and exercise. Am J Obstet Gynecol 104:856–864, 1969
8. Bromage PR: Physiology and pharmacology of epidural anesthesia. ANESTHESIOLOGY 28:592–622, 1967
9. Hollmen A, Jagerhorn M: The effects of epidural anaesthesia and caesarean section on foetal and maternal acid–base balance at birth. Acta Anaesthesiol Scand 12:115–123, 1968
10. Fox GS, Houle GL: Acid–base studies in elective caesarean sections during epidural and general anaesthesia. Can Anaesth Soc J 18:60–71, 1971
11. Lumley J, McKinnon L, Wood C: Lack of agreement on normal values for fetal scalp blood. J Obstet Gynaecol Br Commonw 78:13–21, 1971
12. Beard RW: The detection of fetal asphyxia in labor. Pediatrics 53:157–169, 1974
13. Scanlon JW, Ostheimer GW, Lurie AO, et al: Neurobehavioral responses and drug concentrations in newborns after maternal epidural anesthesia with bupivacaine. ANESTHESIOLOGY 45:400–405, 1976
14. Brown WU, Bell GC, Alper MH: Acidosis, local anesthetics, and the newborn. Obstet Gynecol 48:27–30, 1976
15. Biehl D, Shnider SM, Levinson G, et al: Placental transfer of lidocaine: Effects of fetal acidosis. ANESTHESIOLOGY 48: 409–412, 1978
16. Sjostrand U, Widman B: Distribution of bupivacaine in the rabbit under normal and acidotic conditions. Acta Anaesthesiol Scand [Suppl] 50:5–24, 1973