Method of Ephedrine Administration and Nausea and Hypotension during Spinal Anesthesia for Cesarean Section

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Nausea alone or with vomiting during spinal anesthesia for cesarean section is a common occurrence. These symptoms have been attributed to hypotension.1,2 While hypotension may be harmful to the newborns,3,4 nausea and/or vomiting may disturb both patients and surgeons.

Prophylactic measures such as acute expansion of the intravascular volume and left uterine displacement attenuate the incidence of hypotension.5–6 Ephedrine, the most widely used vasopressor in obstetrics, is commonly used either as a prophylactic intramuscular injection7 or by intravenous administration after the development of frank hypotension. Neither of these approaches is completely satisfactory. Our idea was to administer ephedrine intravenously as soon as there was any drop of maternal blood pressure from the baseline level. Purpose of this project was to assess the effectiveness of this regimen in the prevention of hypotension, nausea alone, or with vomiting. We also proposed to examine maternal and neonatal acid-base status following this technique.

MATERIALS AND METHODS

Sixty healthy parturients, scheduled for elective primary or repeat cesarean section at term, were studied. The anesthetic technique was the same in all cases. No premedication was given. Maternal blood pressure was first measured after placing the patient in a left lateral position with a 15° tilt by means of a blanket roll under the right side and hip. This value was noted as the baseline blood pressure. The parturients received 1500 ml 5 per cent dextrose in lactated Ringer’s solution intravenously within 15–20 min before induction of anesthesia. Tetracaine, 8–9 mg (0.8–0.9 ml 10 per cent dextrose) in a 0.5 per cent solution, was injected through a 26-gauge spinal needle usually inserted at L2–L3 interspace. All lumbar punctures were performed with the patient in the right lateral position.

After injection of the anesthetic solution, the patient was replaced in the left lateral position with a 15° tilt. Oxygen at 6 l/min was administered via a plastic disposable face mask from the time of induction of spinal anesthesia until delivery of the baby. Continuous electrocardiographic monitoring was used routinely. Blood pressure was measured at 30-s intervals for the first 15 min and every 3 min thereafter. Maternal hypotension was noted to be present when systolic blood pressure fell more than 30 mmHg or below 100 mmHg.

Patients were assigned to one of three groups according to the response of the blood pressure. In Group A (n = 22), there was no change from the baseline blood pressure and none received ephedrine. In Group B (n = 18), because of hypotension (as defined above) ephedrine was given intravenously in repeated doses of 10 mg each until the blood pressure rose to at least 100 mmHg. In Group C (n = 20), intravenous ephedrine (10–30 mg) was administered as soon as any fall from baseline pressure was detected. The two treatment groups (Group B and Group C) were done in sequence.

The incidence of nausea alone or with vomiting was noted by the anesthesiologists by observing and questioning all patients. The time from the injection of the local anesthetic to the delivery of the infant was recorded as the induction-delivery (I-D) interval and the time from incision of the uterus to the delivery of the infant was noted as the uterine incision-delivery (U-D) interval.

At delivery, samples of maternal radial artery blood as well as samples of umbilical artery and vein blood were collected from a doubly clamped segment of umbilical cord. Blood gases and pH were immediately determined on each sample in duplicate with a Radiometer microelectrode system. Base deficit was calculated using the Siggard-Anderson Nomogram.8 Apgar scores were determined at 1 and 5 min of age by a pediatrician who was not aware to which study group the patient belonged.

All data were analyzed for statistical significance by computation of multiple correlation coefficients, t tests, or chi-square method where appropriate. A P value of < 0.05 was significant.

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RESULTS

There were no differences in gestational age, maternal age, height, weight, or infant birth weight among the groups. In no patient did baseline systolic blood pressure exceed 140 mmHg. Sensory block was adequate and extended to T4 for most patients using pin prick for evaluation. There were no differences in the induction-delivery intervals or the uterine incision-delivery intervals among the groups (table 1).

The incidence of nausea alone or with vomiting was clearly correlated with development of maternal hypotension (table 1). None of the parturients in Group A complained of nausea alone or with vomiting, two mothers (10 per cent) had these symptoms in Group C, whereas two-thirds of all patients (66 per cent) in Group B developed nausea or both nausea and vomiting ($P < 0.01$).

Acid-base data (table 2) showed normal maternal values with no differences among the groups.

The infants of mothers who developed hypotension (Group B) had significantly lower average $pH$ values in umbilical artery and umbilical vein blood at birth ($P < 0.001$) compared with babies of mothers who did not develop hypotension (Groups A and C). Umbilical artery base-deficit and $\Delta$ base deficit also were higher in Group B ($P < 0.001$).

The percentage of newborns with low Apgar scores (<7) was higher in Group B (80 per cent) at 1 min; however there were no differences in Apgar scores at 5 min among the groups (table 1). No patient in any group developed postpartum hypertension.

DISCUSSION

Maternal hypotension during spinal anesthesia for cesarean delivery is a persistent problem in approximately 80 per cent of cases without proper prophylaxis. Prehydration and left uterine displacement reduce the incidence to 50 to 60 per cent. Use of prophylactic intramuscular ephedrine as suggested by Gutsche, further lowers the incidence to 24 per cent. This use of ephedrine may result in postpartum hypertension because of synergism with oxytocic drugs. The advantage of our approach is that ephedrine is used only when necessary and virtually eliminates hypotension without any demonstrable side effects.

By studying only full-term pregnant women undergoing scheduled cesarean section without premedication and a uniform spinal anesthesia technique, we minimized factors that might contribute to maternal nausea alone or with vomiting. Even though the observer was aware of the patients' treatment, the diagnosis of nausea alone or with vomiting was probably objective because of being based on spontaneous complaints or obvious retching by the patient. In this study, nausea alone or with vomiting occurred in those patients who developed hypotension. If frank hypotension was prevented, nausea alone or with vomiting was not observed. This supports the suggestion that the mechanism of the nausea alone or with vomiting may be maternal hypotension and hypoxemia in the vomiting center.

Fetal acid-base status also was significantly better in Groups A and C where systolic blood pressure remained above 100 mmHg compared with Group B patients who developed frank hypotension. Hypotension reduces uterine perfusion and is associated with neonatal acidosis, even when it is promptly treated with intravenous ephedrine. We observed a similar result in the babies of mothers of Group B. On the other hand, maintenance of blood pressure close to baseline level with or without ephedrine kept the umbilical artery $pH$ high in both Group A and Group C. Finally, the percentage of low Apgar scores at 1 min was significantly higher in newborns whose mothers developed frank hypotension.

In conclusion, following induction of spinal anesthesia for cesarean section, the prompt intravenous adminis-

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### Table 1. Patient Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Group A (n = 22)</th>
<th>Group B (n = 18)</th>
<th>Group C (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline blood pressure (mmHg)</td>
<td>125 ± 4*</td>
<td>120 ± 3</td>
<td>123 ± 4</td>
</tr>
<tr>
<td>L-D interval (min)</td>
<td>15 ± 2</td>
<td>13 ± 2</td>
<td>15 ± 2</td>
</tr>
<tr>
<td>U-D interval (s)</td>
<td>90 ± 10</td>
<td>110 ± 12</td>
<td>100 ± 10</td>
</tr>
<tr>
<td>Incidence of nausea and vomiting (per cent)</td>
<td>0</td>
<td>66*</td>
<td>10</td>
</tr>
<tr>
<td>Apgar score (&lt;7) (per cent)</td>
<td>1 min</td>
<td>0</td>
<td>83*</td>
</tr>
<tr>
<td></td>
<td>5 min</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

* Mean ± SE.
† $P < 0.05$.

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### Table 2. Acid-Base and Blood Gas Data

<table>
<thead>
<tr>
<th></th>
<th>Group A</th>
<th>Group B</th>
<th>Group C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal artery $pH$</td>
<td>7.44 ± 0.01*</td>
<td>7.40 ± 0.01</td>
<td>7.43 ± 0.01</td>
</tr>
<tr>
<td>BE $\pm$ mEq/l</td>
<td>1.3 ± 0.6</td>
<td>2.8 ± 0.6</td>
<td>0.8 ± 0.8</td>
</tr>
<tr>
<td>Umbilical ven $pH$</td>
<td>7.36 ± 0.01</td>
<td>7.31 ± 0.01†</td>
<td>7.38 ± 0.01†</td>
</tr>
<tr>
<td>Umbilical artery $pH$</td>
<td>7.30 ± 0.01†</td>
<td>7.23 ± 0.01†</td>
<td>7.30 ± 0.01†</td>
</tr>
<tr>
<td>BE</td>
<td>−3.00 ± 0.5</td>
<td>−7.3 ± 0.6†</td>
<td>−2.7 ± 0.7†</td>
</tr>
<tr>
<td>$\Delta$ BE (umbilical artery BE − maternal artery BE)</td>
<td>−1.7 ± 0.3</td>
<td>−4.5 ± 0.8†</td>
<td>−1.9 ± 0.5</td>
</tr>
</tbody>
</table>

* Mean ± SE.
† $P < 0.05$.
‡ BE = base excess.
tration of ephedrine as soon as any fall in maternal blood pressure is detected prevents further fall in blood pressure and markedly reduces the incidence of nausea alone or with vomiting. In addition, acid-base status in the infants of parturients so treated is normal and identical to that in the patients who did not experience a fall in blood pressure and hence did not receive ephedrine. This simple alteration in clinical practice seems to provide significant improvement in clinical care of mothers and babies.

REFERENCES


Assessment of Risk Factors Related to the Acid Aspiration Syndrome—Gastric pH and Residual Volume

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Stress and emotional upset apparently increase gastric acid production.1-2 Since the presurgical period is stressful for many children, we examined risk factors for the acid aspiration syndrome in a prospective randomized study. Factors investigated included anxiety, gastric volume and pH, inpatients vs. outpatients, and the number of previous anesthetic experiences. Our intent was to define a subgroup of patients at increased risk for acid aspiration.

MATERIALS AND METHODS

This protocol was approved by the Subcommittee on Human Studies of our institution. Fifty-one ASA physical status I children ages 3-17 years were randomly chosen for this study. Group I (n = 16) consisted of children anesthetized for the first time; all were inpatients. Group II (n = 16) consisted of pediatric outpatients anesthetized for the first time. Group III (n = 19) consisted of children who had been hospitalized and anesthetized on multiple occasions; all of this group were plastic surgical patients at least one year post-acute burn injury. No patient had a known history of ulcer disease or was taking a medication that would interfere with gastric emptying.

The level of anxiety prior to induction was assessed by a physician not involved in the study. Each patient was scored as either anxious or comfortable. The term anxious meant that the child was obviously nervous and uncomfortable with the events about him. Comfortable meant that there were no overt signs of anxiety such as crying, agitation, clinging to the parent, or refusing to talk.

Children were not premedicated. Induction of anesthesia in children under age 7 consisted of 20 to 30 mg/kg methohexital (100 mg/ml) administered rectally by a physician in the presence of a parent. Older children were induced with intravenous thiopental; all patients were maintained with halothane, nitrous oxide, and oxygen.

After induction of general anesthesia gastric samples were obtained through a Salem Sump® catheter (Argyle)