The Incidence and Neonatal Effects of Maternal Hypotension During Epidural Anesthesia for Cesarean Section

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Cesarean sections are frequently performed during epidural anesthesia. A major side effect of epidural anesthesia is maternal hypotension leading to metabolic abnormalities in umbilical cord blood.1,2 Previous studies of the effects of maternal hypotension on neonatal acid-base status have reported data from only small numbers of patients undergoing elective or repeat cesarean section during epidural anesthesia.1-4 The incidence of hypotension reported has varied according to the definition of hypotension, the type of prophylaxis, the extent of epidural block, and the frequency of blood pressure monitoring.

To determine whether a specific prophylactic and therapeutic regimen could reduce the incidence of maternal hypotension, we studied a large population of laboring and nonlaboring women undergoing repeat and primary cesarean sections during epidural anesthesia. We determined the incidence of maternal hypotension and its effects on neonatal clinical and acid-base status.

MATERIALS AND METHODS

Over a 4-yr period, we consecutively studied 583 parturients delivered by cesarean section during epidural anesthesia. All women were in the 38th to 42nd week of pregnancy. Subjects with preeclampsia, Rh-sensitized fetuses, and multiple or growth-retarded fetuses were excluded from the study, which was conducted with the approval of the University of California Committee on Human Research.

Five groups of women were identified: group I — repeat cesarean section, no labor (N = 183); group II — planned primary cesarean section, no labor (N = 39); group III — repeat cesarean section, labor, no fetal distress (N = 88); group IV — primary cesarean section, labor, no fetal distress (N = 229); and group V — primary cesarean section, labor, mild-to-moderate fetal distress (N = 44). Mild-to-moderate fetal distress was defined as evidence of meconium with fetal heart rate abnormalities during the first stage of labor or fetal heart rate abnormalities characterized by persistent tachycardia, late decelerations, or variable decelerations. Breech presentation and cephalopelvic disproportion were considered indicators for primary cesarean section in nonlaboring women, and failure-to-progress, cephalopelvic disproportion, and breech presentation determined most of the cesarean sections in laboring women.

To prevent hypotension before delivery, all patients were given a rapid iv infusion of 1 L of lactated Ringer's solution immediately before administration of epidural anesthesia. In addition, within 30 min of administration of the block, 272 patients (47%) were given ephedrine, 25-50 mg im. This injection was not given in the remaining patients when clinical observation indicated no clear prophylactic benefit. An epidural block was then performed with patients in the lateral decubitus position, following which patients were placed in slight Trendelenburg position with a left lateral uterine tilt of 30°. All patients were administered oxygen by nonbreathing face mask. Choice of local anesthetic was determined by the attending anesthesiologist: 70% of all patients received bupivacaine, 24% chloroprocaine, and 6% lidocaine to obtain anesthesia to, approximately, a T-4 sensory dermatome level.

Blood pressure was monitored at least once per min-
ute up to the time of delivery with a blood pressure cuff and either a Dinamap® vital sign monitor (Critikon 845) or Arteriosonde® automated blood pressure monitor (Roche 1216). Hypotension was defined as a systolic blood pressure lower than 100 mmHg or a greater than 30% decrease from baseline. Baseline maternal blood pressure was recorded while the patient was in the left lateral tilt position. Hypotension was treated by infusing additional lactated Ringer’s solution, increasing the head-down position, and manually displacing the uterus to the left when possible. If blood pressure did not return to greater than 100 mmHg within 30–60 s, incremental iv doses of ephedrine were administered to restore normal blood pressure.

Time from uterine incision-to-delivery of the infant was recorded by the anesthesiologist. Apgar scores at 1 and 5 min and time-to-sustained respiration (TSR) were determined by a pediatrician unaware of either the choice of anesthetic or the presence or absence of hypotension. At delivery, umbilical venous (UV) and arterial (UA) blood-gas samples were obtained from a doubly clamped segment of umbilical cord and analyzed immediately. Base excess (BE) was calculated using a Severinghaus slide ruler.5

The incidence of hypotension for all patients was distributed into categories defined by the presence or absence of labor and the use of im ephedrine prophylaxis. These data were analyzed using the Chi-square test and Fisher’s exact test. Within each of the five subject groups, infants of normotensive mothers were compared with infants of hypotensive mothers for Apgar score, TSR, and umbilical blood acid-base status. Infants of normotensive mothers in all groups were compared for umbilical blood acid-base status. Comparisons of Apgar scores and TSR were analyzed using the Chi-square test and Fisher’s exact test. Blood acid-base data were compared using analysis of variance and Duncan’s multiple range test; only paired UA and UV acid-base values were used for statistical comparison. UV and UA blood acid-base data and uterine incision-to-delivery times are presented as mean values and standard errors of the mean. A P value of less than 0.05 was considered significant.

**RESULTS**

**Maternal Hypotension.** The overall incidence of maternal hypotension was 29%; 92% of whom were given iv ephedrine to normalize blood pressure. Nonlaboring mothers (groups I, II) had a significantly higher incidence of hypotension than laboring mothers (groups III, IV, V), 36% versus 24% (P < 0.01, fig. 1). However, the presence of transient maternal hypotension did not affect the clinical condition of neonates. There were no significant differences in the Apgar scores or TSR of infants of normotensive and hypotensive mothers within any of the five study groups.

The comparison of UV and UA blood-gas values for infants of normotensive and hypotensive mothers within each study group indicated no significant differences in UV and UA Po2 and Pco2 values (tables 1, 2). Statistically significant differences appeared in the values for UV pH (group I), UA pH (groups I, II, and IV), and BE (group II). However, the mean difference in acid-base status for these infants was never greater than 0.04 pH units or 2 mEq base excess. Umbilical blood acid-base values for neonates who experienced mild-to-moderate distress during labor (group V) did not differ significantly in the presence versus absence of maternal hypotension.

When the mean umbilical blood acid-base values of infants of all normotensive mothers were compared, infants of mothers in group V had significantly lower values for UA base excess and UV pH, Po2, and base excess than infants of mothers in the other four groups. Infants from groups III and V had significantly lower UA, pH and significantly higher UV Pco2 values, compared with groups I, II, and IV (tables 1, 2).

**Ephedrine Prophylaxis.** Hypotension occurred in 26% of the mothers treated prophylactically with ephedrine and in 32% of those not treated (P = NS). Although the incidence of hypotension in certain groups tended to decrease with the prophylactic use of ephedrine (groups I, II, and IV), the differences within groups were not statistically significant (table 3). Laboring mothers given prophylactic ephedrine had a 20% incidence of hypotension and nonlaboring mothers, a 33% incidence (P
Table 1. The Effect of Maternal Hypotension on Umbilical Venous Acid-base State

<table>
<thead>
<tr>
<th>Group</th>
<th>$\text{pH}$</th>
<th>$\text{PaCO}_2$</th>
<th>$\text{PCO}_2$</th>
<th>BE</th>
</tr>
</thead>
<tbody>
<tr>
<td>I (N = 145) Normal BP Hypotension</td>
<td>7.33 ± 0.01</td>
<td>31.6 ± 0.6</td>
<td>41.5 ± 0.5</td>
<td>−3.7 ± 0.3</td>
</tr>
<tr>
<td>II (N = 25) Normal BP Hypotension</td>
<td>7.31 ± 0.01*</td>
<td>32.4 ± 0.8</td>
<td>43.1 ± 0.7</td>
<td>−4.3 ± 0.5</td>
</tr>
<tr>
<td>III (N = 69) Normal BP Hypotension</td>
<td>7.33 ± 0.01</td>
<td>31.0 ± 1.0</td>
<td>41.8 ± 0.9</td>
<td>−5.5 ± 0.5</td>
</tr>
<tr>
<td>IV (N = 174) Normal BP Hypotension</td>
<td>7.32 ± 0.01</td>
<td>30.9 ± 0.7</td>
<td>42.6 ± 0.7†</td>
<td>−4.2 ± 0.4</td>
</tr>
<tr>
<td>V (N = 35) Normal BP Hypotension</td>
<td>7.32 ± 0.01</td>
<td>29.0 ± 0.6</td>
<td>41.0 ± 0.4</td>
<td>−4.4 ± 0.2</td>
</tr>
</tbody>
</table>

* $P < 0.01$, normal blood pressure vs. hypotension (within group).
† $P < 0.05$, compared with normotensive values in groups I, II, and IV.
‡ $P < 0.05$, compared with normotensive values in groups I, II, III, and IV.

< 0.05). Among those not given ephedrine, laboring mothers had a 27% incidence of hypotension and non-laboring mothers, a 41% incidence ($P < 0.05$). Infant outcome, measured by Apgar score, TPSR, and umbilical blood-gas values, did not differ among hypotensive mothers, regardless of prophylactic maternal treatment with im ephedrine.

Uterine Incision-to-delivery Time. The mean uterine incision-to-delivery times did not differ significantly among the five groups. They were 127 ± 5 s for group I, 158 ± 11 s for group II, 119 ± 6 s for group III, 120 ± 4 s for group IV, and 109 ± 8 s for group V.

Discussion

Lumbar epidural anesthesia, a technique commonly used for cesarean section, may result in maternal hypotension with decreased uterine arterial blood flow and interstitial perfusion. Abnormal fetal heart rate patterns and significant fetal acidosis can result.2,6–8,12 Although the incidence of hypotension reported for this type of anesthesia varies (5–80%),1,4,13–15 our study of 583 parturients indicated an overall incidence of 29%. Non-laboring mothers had a significantly higher incidence of hypotension than laboring mothers.

Certain techniques can minimize the maternal hemodynamic and fetal physiologic changes accompanying lumbar epidural anesthesia during cesarean section, including rapid iv infusion of crystalloid,13,15–17 left uterine displacement after induction of epidural anesthesia,18 prompt correction of maternal hypotension, and uterine incision-to-delivery time of less than 3 min.19

An additional technique, the prophylactic administration of im ephedrine, reportedly prevents maternal hypotension during spinal anesthesia for cesarean section.20 Although some researchers have recommended the prophylactic use of im ephedrine during epidural anesthesia,14 Rolbin et al. found no significant decrease in the incidence of hypotension during epidural anesthesia when 25 patients given 25 mg of prophylactic im

Table 2. The Effect of Maternal Hypotension on Umbilical Arterial Acid-base State

<table>
<thead>
<tr>
<th>Group</th>
<th>$\text{pH}$</th>
<th>$\text{PaCO}_2$</th>
<th>$\text{PCO}_2$</th>
<th>BE</th>
</tr>
</thead>
<tbody>
<tr>
<td>I (N = 145) Normal BP Hypotension</td>
<td>7.24 ± 0.01</td>
<td>16.2 ± 0.5</td>
<td>52.9 ± 0.9</td>
<td>−4.1 ± 0.3</td>
</tr>
<tr>
<td>II (N = 25) Normal BP Hypotension</td>
<td>7.21 ± 0.01*</td>
<td>16.5 ± 0.6</td>
<td>56.7 ± 1.2</td>
<td>−5.1 ± 0.5</td>
</tr>
<tr>
<td>III (N = 69) Normal BP Hypotension</td>
<td>7.24 ± 0.01</td>
<td>16.4 ± 1.0</td>
<td>51.6 ± 1.7</td>
<td>−4.5 ± 0.6</td>
</tr>
<tr>
<td>IV (N = 174) Normal BP Hypotension</td>
<td>7.20 ± 0.02†</td>
<td>18.4 ± 3.8</td>
<td>50.3 ± 5.0</td>
<td>−6.4 ± 0.8†</td>
</tr>
<tr>
<td>V (N = 35) Normal BP Hypotension</td>
<td>7.22 ± 0.01‡</td>
<td>15.6 ± 0.7</td>
<td>54.7 ± 1.2</td>
<td>−5.2 ± 0.5</td>
</tr>
<tr>
<td></td>
<td>7.23 ± 0.01</td>
<td>15.2 ± 1.0</td>
<td>54.3 ± 1.5</td>
<td>−4.8 ± 0.6</td>
</tr>
<tr>
<td></td>
<td>7.24 ± 0.01</td>
<td>15.8 ± 0.7</td>
<td>51.0 ± 0.7</td>
<td>−5.1 ± 0.3</td>
</tr>
<tr>
<td></td>
<td>7.20 ± 0.01†</td>
<td>14.1 ± 0.6</td>
<td>53.6 ± 1.1</td>
<td>−6.5 ± 0.6</td>
</tr>
<tr>
<td></td>
<td>7.20 ± 0.01‡</td>
<td>14.1 ± 1.1</td>
<td>55.3 ± 1.5</td>
<td>−6.6 ± 0.7§</td>
</tr>
<tr>
<td></td>
<td>7.19 ± 0.02</td>
<td>13.6 ± 2.0</td>
<td>58.7 ± 3.9</td>
<td>−6.3 ± 0.9</td>
</tr>
</tbody>
</table>

* $P < 0.01$, normal blood pressure vs. hypotension (within group).
† $P < 0.05$, normal blood pressure vs. hypotension (within group).
‡ $P < 0.05$, compared with normotensive values in groups I, II, and IV.
§ $P < 0.05$, compared with normotensive values in groups I, II, III, and IV.
TABLE 3. The Effects of IM Ephedrine Prophylaxis on the Incidence of Hypotension*

<table>
<thead>
<tr>
<th></th>
<th>Group I (n = 183)</th>
<th>Group II (n = 39)</th>
<th>Group III (n = 88)</th>
<th>Group IV (n = 229)</th>
<th>Group V (n = 44)</th>
</tr>
</thead>
<tbody>
<tr>
<td>No ephedrine prophylaxis</td>
<td>34 (43%)</td>
<td>7 (35%)</td>
<td>11 (26%)</td>
<td>38 (27%)</td>
<td>8 (28%)</td>
</tr>
<tr>
<td>Ephedrine prophylaxis</td>
<td>36 (35%)</td>
<td>4 (21%)</td>
<td>12 (26%)</td>
<td>10 (12%)</td>
<td>8 (50%)</td>
</tr>
</tbody>
</table>

Values represent numbers of hypotensive patients, expressed as percentages in parentheses.

* No significant differences within groups.

Ephedrine (12%) were compared with 25 controls (12%). Our data tend to support these findings. Hypotension occurred in 26% of our patients treated with prophylactic ephedrine versus 32% of those not treated; this difference was not significant.

The regimen we used to prevent hypotension was more effective in reducing maternal hypotension in laboring mothers than in nonlaboring mothers. Antoine and Young report a similar observation: following a regimen which included hydration with 0.5–1.0 l of a balanced salt solution, left uterine displacement, and oxygen administration, 62% of nonlaboring mothers and 35% of laboring mothers developed a greater than 20% decrease in systolic blood pressure following administration of epidural anesthesia for cesarean section. An explanation for the greater incidence of hypotension among nonlaboring mothers may be that most are fasted for at least 8 h and are not hydrated intravenously until just prior to epidural block. In contrast, laboring mothers undergoing nonelective cesarean section often undergo continuous intravenous hydration during labor and may, therefore, be less hypovolemic and less prone to hypotension.

Our results suggest that the administration of 1 l of crystalloid solution prior to epidural block for cesarean section may provide adequate hydration, particularly for the nonlaboring mother. Several studies indicate that an increase in the volume of hydration provided before epidural anesthesia for cesarean section may substantially decrease the incidence of maternal hypotension. In one study, only 6.7% of patients given epidural anesthesia for elective cesarean section developed hypotension after hydration with 2 l of lactated Ringer's solution. In another study, the incidence of hypotension in women fasted before epidural anesthesia for cesarean section decreased from 45% to 5% following an infusion including 0.5 l each of colloid solution and lactated Ringer's solution versus 1.0 l of lactated Ringer's solution alone. Infusion of the combined mixture may replenish the intravenous volume to a greater degree. Colloid solutions increase oncotic pressure and albumin concentration while crystalloid administration decreases these values. An increase in plasma oncotic pressure promotes expansion of the intravascular fluid volume.

The adverse fetal effects of maternal hypotension are apparent in UV and UA acid-base values. The magnitude of the occasional difference in mean umbilical pH observed in this study (0.02–0.04) is small, and similar to that reported by others. However, greater changes in mean umbilical pH have been associated with maternal hypotension during spinal anesthesia, following less rigid prophylactic regimens than ours. One study of the effect of different hydration regimens preceding spinal anesthesia for cesarean section reported that a hydration volume of less than 1 l was associated with significantly more fetal acidemia than a higher hydration volume. Prompt treatment with ephedrine after a 10-mmHg decrease from baseline systolic blood pressure during spinal anesthesia will reportedly maintain neonatal acid-base status at control level. This suggests that the early administration of iv ephedrine in response to a decrease in maternal blood pressure may help to maintain normal blood pressure and have no adverse effect on the fetus. Had this method of ephedrine administration been used in our study, the incidence of maternal hypotension might have decreased and fetal intragroup metabolic differences might have been insignificant.

Among group V mothers, mild-to-moderate fetal distress was considered an indication for cesarean section. However, these distressed infants demonstrated no deterioration in clinical condition or adverse change in mean umbilical blood-gas values as a result of maternal hypotension. In a series of 126 anesthetics for emergency cesarean section performed for fetal distress, Marx et al. obtained similar UV and UA blood-gas data during general and regional anesthetics. Thus, mild-to-moderate fetal distress alone should not contraindicate regional anesthesia for cesarean section.

Our definition of maternal hypotension was standard, a systolic blood pressure lower than 100 mmHg or 30% lower than the preblock systolic pressure. This is derived from studies by Hon et al. and Zilanti, who
reported that a maternal systolic blood pressure lower than 100 mmHg for approximately 5 min produced abnormal fetal heart rate patterns, and that maternal systolic blood pressures lower than 100 mmHg were associated with fetal acidosis and bradycardia. Although the standard, this definition is limited and, possibly, inadequate, because it cannot differentiate the severity of hypotension among patients or account for patients whose normal blood pressure is low. In addition, baseline blood pressures must be more precisely defined. For example, immediate preblock pressures may be elevated due to patient stress and pain, while periods of sleep may be accompanied by systolic blood pressures lower than 100 mmHg with no associated fetal heart rate abnormalities. A definition of maternal hypotension based on the percent change in systolic or mean blood pressure from some yet undefined average "baseline" pressure may be more useful. It would help prevent overtreatment and undertreatment of patients whose normal blood pressure values reflect the low or high end of the normal blood pressure range.

Epidural anesthesia for cesarean section is a safe technique, and maternal hypotension need not adversely affect the fetus. The overall clinical condition of our neonates according to Apgar scores and TSR was unaffected by maternal hypotension in all patient groups. The use of specific prophylactic measures combined with rapid correction of maternal hypotension attenuated the detrimental effects of maternal hypotension on fetal acid-base status. The higher incidence of maternal hypotension among nlaboring mothers indicates that they are potentially at greater risk for complications with epidural anesthesia. Larger hydration volumes of crystalloid than those used in this study, and more prompt administration of iv ephedrine, may enhance the safety of epidural anesthesia for cesarean section.

REFERENCES


1 James CF, Gibbs CP, Gravenstein JS, van der Aa J: Maternal hypotension: Are current criteria valid (abstract)? ANESTHESIOLOGY 57:A393, 1982