MATERNAL HYPOTENSION AND NEONATAL ACIDAEMIA DURING CAESAREAN DELIVERY UNDER SPINAL ANAESTHESIA

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P. R. OLANG', D. C. WAMALWA and OMONDI-OGUTU

ABSTRACT

Objectives: To determine the incidence of neonatal acidaemia following delivery through caesarean section under spinal anaesthesia and determine the prevalence of maternal hypotension during Caesarean section under spinal anaesthesia and its correlation with neonatal acidaemia.

Design: Prospective observational study

Setting: Kenyatta National Hospital (KNH), Nairobi, Kenya.

Subjects: One hundred and seventy-two ASA I and II consecutive term patients undergoing elective Caesarean section under spinal anaesthesia

Results: Forty three babies (27.2%) were born with neonatal acidaemia, defined as umbilical arterial blood pH≤ 7.2. There was, however, no significant difference in the five minute Apgar scores between the acidotic and non-acidotic neonates. Twenty eight patients (17.7%) developed maternal hypotension (systolic Blood Pressure less than 100mmHg). The hypotension was readily corrected within two minutes (mean of 1.43 minutes) of onset using vasopressors and boluses of intravenous fluids.

Conclusions: A short period (<2 minutes) mean of 1.43 minutes of maternal hypotension has no significant effect on the neonate as assessed by five minute Apgar Scores. Similarly, neonatal acidaemia following Caesarean delivery under spinal anaesthesia does not seem to have any short-term effects on neonatal well-being.

INTRODUCTION

American College of Obstetrics and Gynaecology (ACOG) Committee Opinion No. 348 of November, 2006 states that; “umbilical cord blood gas and acid-base assessment are the most objective determinants of the foetal metabolic condition at the moment of birth. Moderate and severe newborn encephalopathy, respiratory complications and composite complication scores increase with an umbilical arterial base deficit of 12-16 mmol/L” (1).

Maternal hypotension is the most common complication of spinal anaesthesia during caesarean section and is associated with reduced utero-placental perfusion. Although this hypotension can be catastrophic and poses a threat to both mother and neonate, it is not always severe and is readily treatable. If short-lived (not more than two minutes), it does not significantly affect neonatal outcome (2).

Whereas several protocols have been formulated to prevent maternal hypotension, none has been shown to totally eliminate this risk (3-5).

Up until ten years ago, all Caesarean sections at Kenyatta National Hospital were performed under general anaesthesia. This trend has now changed and more mothers have Caesarean delivery under spinal anaesthesia. Since general anaesthesia has been associated with a significant maternal and neonatal morbidity attributable mainly to difficult/failed tracheal intubations and aspiration (3), in keeping with current obstetric anaesthesia guidelines, it has been recommended that regional anaesthesia for Caesarean section be the preferred choice in this institution (1, 3).

Kenyatta National Hospital is the premier referral and teaching hospital in Kenya. It runs a very busy obstetric care unit with over 3000 deliveries annually. The Caesarean section delivery rate at 46-52% in the institution is mainly due to many high risk patients referred from other hospitals in the region. The labour ward has two operating rooms, one of which operates
on a 24-hour basis to cover emergencies. The second operating room handles all elective cases.

With the gradual introduction of regional anaesthesia since 1998, currently over 90% of the Caesarean deliveries are done under spinal anaesthesia. This change in trend towards spinal anaesthesia has led to significant lowering of the morbidity and mortality associated with the anaesthesia during Caesarean delivery. Another factor that has encouraged the introduction of spinal anaesthesia for Caesarean delivery in Kenya is its lower cost as compared to general anaesthesia (2). Indeed, in hospitals that are sponsored by non-governmental organisations in Kenya, Caesarean sections are almost exclusively performed under spinal anaesthesia not only because of its lower cost but also because of shortage of equipment for general anaesthesia and highly trained anaesthesia providers in most rural parts of the country. General anaesthesia is, however, still practised widely in most hospitals since it is safer in the hands of anaesthesia providers not skilled in regional techniques (1).

The main objective of this study was to determine the prevalence of neonatal acidemia and maternal hypotension during spinal anaesthesia for Caesarean section as practised at the Kenyatta National Hospital. The secondary objective was to correlate neonatal acidemia and the duration of maternal hypotension.

MATERIALS AND METHODS

Between November 2009 and May 2010, 172 consecutive patients undergoing elective Caesarean sections under spinal anaesthesia were recruited into the study. The exclusion criteria were as follows:

(a) declined to consent for spinal anaesthesia
(b) declined to be included in the study
(c) had to undergo general anaesthesia for failed spinal anaesthesia or any other reason
(d) went into labour before the scheduled elective surgery
(e) developed non-reassuring foetal status before surgery
(f) patients with a history of bad obstetric history such as stillbirths, neonatal deaths, intrauterine deaths and, therefore, could have had other reasons for neonatal acidemia
(g) previously delivered babies with gross congenital abnormalities

All eligible patients scheduled for elective Caesarean section were counselled on the procedure of spinal anaesthesia. Those who consented gave an informed consent to have spinal anaesthesia, which was to be conducted as per the hospital’s standard guidelines which were prominently displayed in the operating room for ease of reference.

Protocol: Each patient was preloaded with at least 500 ml of crystalloid (Normal Saline-0.9% or Ringers Lactate/Hartmann’s solution), hooked up to the physiological monitor and observations of vital signs were recorded. Under aseptic conditions, 2% lignocaine 2-3 mls was infiltrated into the skin and subcutaneous tissues at the L3-4 or L4-5 interspaces. After three to four minutes, lumbar puncture was performed (observing aseptic techniques) at either of the interspaces with the patient seated or in lateral decubitus position using G25 Quincke point spinal needle and free flow of cerebrospinal fluid (CSF) ensured. One point five millilitres plain or heavy bupivacaine0.5% plus25µg (0.5 ml)fentanyl was then injected into the intra-thecal space. A sterile dressing was placed over the puncture site, and the patient was gently eased to lie in the supine position. Vital signs observations (BP, Pulse, SpO2) were recorded every two and a half minutes. Hypotension (systolic BP < 100 mmHg) was treated immediately with boluses of ephedrine (6 mg) or epinephrine (50 µg) as appropriate and recorded.

At delivery of the baby, the surgeon applied double clamps to the cord proximal to the baby allowing for a five centimetres cord segment with one clamp on the placental side before separation. An assistant took 2 mls of blood samples in two heparinised syringes from the UA (Umbilical Artery) and UV (Umbilical Vein) in the isolated portion of the cord. A prescribed data sheet with the details of the mother accompanied the samples to the ICU (Intensive Care Unit) laboratory within thirty minutes of collection for analysis of blood gases, pH and base excess. A clamped segment of the cord is stable for blood gas and pH assessment for at least sixty minutes. The laboratory used Rapid lab Model 348 for blood gas analysis. Quality control was maintained by means of a special kit used for calibration every morning. Umbilical artery pH ≤ 7.2 was considered as neonatal/foetal acidosis. All the data sheets were collected by the investigators daily from the operating room. The rest of the anaesthetic management of the patient was as per the protocol.

Data analysis: The data were cleaned and coded before input into SPSS version 16.0. The incidence of neonatal acidemia and maternal hypotension was calculated. The analysis was done using Pearson correlation coefficient to determine associations between maternal hypotension and neonatal acidemia and maternal hypotension and volume of preload. The results were considered statistically significant at P<0.05. Multiple regression analysis was applied to determine correlation between the variables.
RESULTS

A total of 172 patients were recruited into the study, but the total number of umbilical artery blood samples analysed was 158. Fourteen samples clotted and were not, therefore, available for analysis. Baseline characteristics of the patients studied are shown in Table 1.

Table 1
Demographic Data

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean (SD)</th>
<th>Median</th>
<th>Interquartile Range</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (Yrs)</td>
<td>29 ± 4.6</td>
<td>29</td>
<td>7.0</td>
<td>28.4 - 29.8</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>158 ± 8.7</td>
<td>159</td>
<td>9.3</td>
<td>157.1 - 159.8</td>
</tr>
<tr>
<td>Weight (Kg)</td>
<td>78.2 ± 10.9</td>
<td>76.3</td>
<td>11.8</td>
<td>76.4 - 79.9</td>
</tr>
</tbody>
</table>

The demographic data shown in Table 1 indicate that there were no extremes of age, height or weight in the study population. In fact, none of the patients in this study had a height ≤155cm which has been associated with a higher risk of maternal hypotension when "usual" doses of local anesthetics are used for spinal anaesthesia. Results of neonatal umbilical arterial and venous blood gas analyses are shown in Tables 2(a) and 2(b).

Table 2 (a)
Umbilical Arterial Blood Gas Analysis

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean ± (SD)</th>
<th>Median</th>
<th>Interquartile Range</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH (Units)</td>
<td>7.247 ± 0.1</td>
<td>7.273</td>
<td>0.12</td>
<td>7.231 - 7.262</td>
</tr>
<tr>
<td>PCO₂ (Kpa)</td>
<td>6.3 ± 1.5</td>
<td>6.1</td>
<td>1.85</td>
<td>6.04 - 6.49</td>
</tr>
<tr>
<td>PO₂ (Kpa)</td>
<td>2.0 ± 1.0</td>
<td>1.9</td>
<td>1.17</td>
<td>1.89 - 2.18</td>
</tr>
<tr>
<td>HCO₃ (mmol/l)</td>
<td>19.8 ± 3.6</td>
<td>20.1</td>
<td>4.3</td>
<td>19.3 - 20.4</td>
</tr>
<tr>
<td>B/E (mmol/l)</td>
<td>-7.5 ± 4.5</td>
<td>-6.8</td>
<td>5.1</td>
<td>-8.1 - -6.8</td>
</tr>
<tr>
<td>O₂ Sat (%)</td>
<td>18.0 ± 15.1</td>
<td>13.3</td>
<td>17.8</td>
<td>15.7 - 20.3</td>
</tr>
</tbody>
</table>

Table 2 (b)
Umbilical Venous Blood Gas Analysis

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Mean ± (SD)</th>
<th>Median</th>
<th>Interquartile Range</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH (Units)</td>
<td>7.284 ± 0.1</td>
<td>7.314</td>
<td>0.13</td>
<td>7.27 - 7.30</td>
</tr>
<tr>
<td>PCO₂ (Kpa)</td>
<td>5.7 ± 1.5</td>
<td>5.5</td>
<td>1.78</td>
<td>5.51 - 5.95</td>
</tr>
<tr>
<td>PO₂ (Kpa)</td>
<td>3 ± 1.2</td>
<td>2.8</td>
<td>1.64</td>
<td>2.79 - 3.14</td>
</tr>
<tr>
<td>HCO₃ (mmol/l)</td>
<td>19.7 ± 3.3</td>
<td>19.9</td>
<td>4.0</td>
<td>19.2 - 20.2</td>
</tr>
<tr>
<td>B/E (mmol/l)</td>
<td>-6.7 ± 4.2</td>
<td>-6.1</td>
<td>4.4</td>
<td>-7.4 - -6.1</td>
</tr>
<tr>
<td>O₂ Sat (%)</td>
<td>33.4 ± 20.0</td>
<td>33.4</td>
<td>32.4</td>
<td>30.8 - 32.4</td>
</tr>
</tbody>
</table>

Forty three babies (27.2%) were born with neonatal acidaemia defined as umbilical arterial blood pH≤7.2. The mean umbilical arterial and venous pH was just over 7.2 with a very narrow range of 0.12 and 0.13 respectively. There was a tendency towards neonatal acidaemia as exemplified by the narrow ranges in both umbilical arterial and venous pH. Umbilical arterial and venous base deficits were less than 10mmol/L which is far from the range of 12-16 mmol/L associated with significant neonatal complications (1).
Table 3
Early Neonatal Outcome and Maternal Blood Pressure

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normotensive (N=125) Mean ± SD</th>
<th>Hypotensive (N=31) Mean ± SD</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ApH</td>
<td>7.247 ± 0.11</td>
<td>7.245 ± 0.1</td>
<td>0.926</td>
</tr>
<tr>
<td>VpH</td>
<td>7.285 ± 0.11</td>
<td>7.276 ± 0.11</td>
<td>0.684</td>
</tr>
<tr>
<td>Apgar Score (1 Min)</td>
<td>8.8 ± 0.85</td>
<td>8.8 ± 0.6</td>
<td>1.000</td>
</tr>
<tr>
<td>Apgar Score (5 Min)</td>
<td>9.8 ± 0.58</td>
<td>9.8 ± 0.45</td>
<td>1.000</td>
</tr>
</tbody>
</table>

Key:
ApH = Umbilical Arterial Blood pH
VpH = Umbilical Venous Blood pH.

Twenty eight patients (17.7%) developed maternal hypotension (systolic Blood Pressure less than 100mmHg) but there was no statistically significant difference in the prevalence of neonatal acidaemia and low Apgar scores among babies born to normotensive and hypotensive mothers (Table 3). We had expected to find a positive correlation between maternal hypotension and neonatal acidaemia and low Apgar scores.

**DISCUSSION**

This study showed that maternal hypotension occurring for short periods of time before delivery had no significant effect on neonatal outcome. The findings are similar to those of Karinen et al who observed a higher incidence of maternal hypotension in the crystalloid preload group (62%) than in the colloid group (38%). They concluded that these changes seemed not to have had any major effect on the clinical condition of the newborn, as assessed by Apgar scores and umbilical artery pH values. It is important to note that all the hypotensive episodes in this study were corrected within two minutes of detection and hence the observed good general neonatal outcome seen (1, 2). Maternal hypotension was noted to have been poorly correlated with umbilical venous blood pH. This result is at variance with studies done by Ueyama et al (7) who concluded that maternal hypotension had a strong correlation with neonatal acidaemia. A study by Socol et al also showed that all mothers with hypotension delivered babies with umbilical venous blood pH 7.2 and associated raised base excess. They, however, noted that the neonatal Apgar scores at ten minutes were normal and so were the neonatal neuro-behaviour scores within the first 24 hours of follow up (8). In this study there was no effect on the babies noted at birth. Although we did not make any specific follow up on the babies during the first 24 hours, there were no reports of any adverse effects on the babies during the post-operative reviews on the day after surgery.

A study by Corke et al concluded that babies born to mothers with hypotension were significantly more acidotic than controls although acid-base levels were still within normal limits. Neonatal neuro-behavioural studies were, however, found to have been normal in both groups at four and 24 hours. They, like us, also concluded that a short period (<2 minutes) of hypotension was not harmful to the neonate (8). Using random-effect meta-analysis, twenty-seven studies reporting on neonatal acid-base data with different types of anaesthesia were used by Reynolds et al to compare umbilical artery pH and base deficit. They found that cord pH was significantly lower with spinal than with both epidural and general anaesthesia and the base deficit was also significantly higher for spinal anaesthesia (9).

Ramin et al noted that the use of ephedrine, an alpha-agonist, during regional anaesthesia was associated with elevated plasma Arginine Vasopressin (AVP) and Adrenocorticotropic Hormone (ACTH) concentrations compared to those in women receiving general anaesthesia. At the time of delivery, 12 out of 91 infants (13.1%) had acidaemia (11). Since ephedrine is commonly used for the treatment of maternal hypotension, it can be inferred that the resulting neonatal acidaemia was either due to maternal hypotension or the ephedrine used to treat the hypotension.

In conclusion, maternal hypotension occurring during spinal anaesthesia for Caesarean section is known to affect the early neonatal acid base balance if left untreated for periods exceeding two minutes. This study has not shown any significant relationship between neonatal acidaemia and maternal hypotension. This, we believe, is because the anaesthetists meticulously followed the protocol provided and did not allow prolonged maternal hypotension to occur. The use of Vasopressors, speed of injection of local anaesthetic and the measures taken to prevent supine hypotension syndrome may have had a confounding effect in this study (12).
REFERENCES


12. Anderson L., Walker J., Brydon C.,Serpell M. G.: Rate of injection through Whitacre needles affects distribution of spinal anaesthesia