Maternal and uteroplacental haemodynamic state in pre-eclamptic patients during spinal anaesthesia for Caesarean section

J. KARINEN, J. RÄSÄNEN, S. ALAHUHTA, R. JOUPPILA AND P. JOUPPILA

Summary

We have studied the effects of crystalloid (Ringer's acetate 1 litre) preloading and subsequent spinal anaesthesia in 12 pre-eclamptic parturient patients undergoing elective Caesarean section. Maternal placental uterine artery circulation was measured using a pulsed colour Doppler technique with simultaneous measurement of maternal haemodynamic state. Despite preloading, mean maternal systolic arterial pressure (SAP) decreased significantly and marked maternal hypotension (SAP < 80 % of baseline value) was recorded in two patients after induction of spinal anaesthesia. Mean central venous pressure increased significantly after preload, but decreased to baseline shortly after induction of spinal anaesthesia. Mean pulsatility index (PI) in the uterine artery did not change during preload or spinal block. In one patient, uterine artery PI increased significantly when SAP decreased to 71 % of the baseline value, 14 min after induction of spinal anaesthesia. These results suggest that preload with crystalloid solution does not prevent maternal hypotension in pre-eclamptic patients, and that changes in uterine artery velocity waveforms were minor when SAP was 80 % or more of baseline during spinal anaesthesia. These changes did not appear to have any major effect on the clinical condition of the neonate, as assessed by Apgar score and umbilical artery pH values. (Br. J. Anaesth. 1996; 76: 616–620)

Key words


Pre-eclamptic parturient patients present a challenge to the anaesthetist because of the problems that pre-eclampsia poses to the fetus and mother. The well-being of the fetus is jeopardized by poor placental perfusion which may result from immunologically mediated abnormal trophoblastic invasion or microvascular disease [1] and is reflected as high vascular resistance in the uterine arteries [2]. The mother may develop cardiopulmonary and cerebrovascular emergencies in addition to acute renal failure, severe thrombocytopenia and activation of the coagulation cascade [3].

Extradural anaesthesia is widely accepted as the method of choice for Caesarean section for pre-eclamptic parturients. There is evidence to suggest that extradural anaesthesia blunts the haemodynamic and neuroendocrine stress responses caused by Caesarean delivery in women with severe pre-eclampsia [4]. Because uterine blood perfusion is reduced in pre-eclampsia, it is important to prevent maternal hypotension; measures used during regional anaesthesia include left uterine displacement, volume preloading and vasopressor infusion. With these prophylactic measures, it has been shown that plain bupivacaine as an anaesthetic for extradural Caesarean section does not change uteroplacental circulation in hypertensive patients [5].

Severe maternal hypotension is more common during spinal anaesthesia for Caesarean delivery than during extradural anaesthesia. This is one reason why spinal anaesthesia has not been considered widely as an alternative to extradural anaesthesia in pre-eclamptic parturient patients. Preloading and vasopressor infusion did not prevent hypotension during spinal anaesthesia [6, 7]. However, it is crucial to avoid hypotension, especially in pre-eclamptic patients, because of the threat of impaired uterine circulation. We have not found any earlier studies on uteroplacental circulation in pre-eclamptic patients immediately after induction of spinal anaesthesia. Therefore, in this study we examined the effects of crystalloid preloading and subsequent spinal anaesthesia on maternal, and especially uteroplacental, haemodynamic state in pre-eclamptic patients, using a pulsed colour Doppler technique during the first 20 min of spinal anaesthesia.

Patients and methods

The study was approved by Oulu University Ethics Committee and written informed consent was obtained from each patient. We studied 12 pre-eclamptic patients (gestation 31–40 weeks) undergoing elective Caesarean section. Of the 12 patients who volunteered for the study, six had severe pre-eclampsia with an arterial pressure constantly greater than 160/110 mm Hg and proteinuria exceeding 5.0 g 24 h⁻¹. The other six patients had an arterial
pressure of 140/90–160/110 mm Hg and proteinuria of 0.3–5.0 g 24 h⁻¹. Ten patients were receiving an antihypertensive peroral regimen of labetol 300–600 mg day⁻¹ and one patient was also receiving nifedipine 5–10 mg day⁻¹. None of the patients was in labour. Exclusion criteria included eclampsia, chronic hypertension, chronic renal disease, thrombocytopenia (platelet count < 100 000 ml⁻¹) and maternal haemorrhage or signs of fetal distress requiring emergency Caesarean section.

Patients were given 30 ml of sodium citrate 0.3 mol litre⁻¹ orally, 60 min before entering the operating theatre, where they were placed in the supine position with a left lateral tilt. Monitoring comprised a three-lead ECG, pulse oxymetry and automated non-invasive arterial pressure (Cardiocap, Datex, Instrumentarium Group, Finland). Oxygen 3 litre min⁻¹ was administered via a nasal cannula. A peripheral vein was cannulated for administration of i.v. fluids, and a vein in the right antecubital fossa was cannulated during local anaesthesia with a 16-gauge central venous catheter for continuous monitoring of central venous pressure (CVP) [8]. Correct location of the tip of the CVP catheter was confirmed by recording the CVP waveform. Baseline maternal heart rate (HR), non-invasive systolic (SAP) and diastolic (DAP) arterial pressures and CVP were recorded, and the first Doppler ultrasound examination was performed before i.v. preloading. In all patients the bladder was catheterized to measure urine output during Caesarean section.

The maternal uterine artery (main branch on the placental side of the uterus) was identified by colour Doppler near the cervix before its divisions and the blood velocity waveform recorded using the pulsed Doppler method (with a 3.75-MHz sector probe, 120-Hz high-pass filter). Pulsatility index (PI = (systolic peak velocity–end-diastolic velocity)/mean velocity during cardiac cycle) was measured from the blood velocity waveform profile. Three consecutive correctly imaged blood velocity waveforms were analysed and the mean value was used in further analysis. All blood flow velocity waveform recordings were obtained during periods of fetal rest without breathing movements. Maternal HR, SAP, DAP and CVP readings were recorded every 2 min during the study.

After the first Doppler measurement, patients received a preload of crystalloid (Ringer’s acetate) solution 1 litre over a 15–20-min period. Immediately after preloading, the second Doppler measurement was obtained. Patients were then placed in a right lateral position, an extradural catheter was inserted 3 cm cephalad through the L1–2 interspace, and an atraumatic catheter was inserted into the subarachnoid space at the L3–4 interspace and 0.5 % hyperbaric bupivacaine 2.5 ml (range 2.4–2.6 ml) was injected intrathecally. Patients were then returned to the supine position with a left lateral tilt. Doppler measurements from the same uterine artery were obtained at 2-min intervals during the first 20 min after injection of bupivacaine. Each Doppler measurement required an average of 1 min and was performed by the same obstetrician (J.R.) in every case. During these measurements sensory block was assessed by loss of cold sensitivity and extended to T4 in most patients. After completion of the ultrasound measurements, patients were prepared for surgery. Plain 0.5 % bupivacaine was given later via the extradural catheter if the spinal block began to wear off.

After induction of spinal anaesthesia, crystalloid solution was infused at approximately 1000 ml per 60 min until delivery. The infusion rate of the crystalloid solution was adjusted carefully according to CVP in all subjects to avoid overhydration. The time from induction of spinal anaesthesia to delivery was recorded. Maternal hypotension was defined as a decrease in SAP to 80 % of baseline and was treated by increasing the rate of crystalloid infusion. The minimum percentage value of SAP was recorded. If hypotension was not corrected within 1–2 min, ephedrine 5 mg was injected i.v. repeatedly until SAP was at least 80 % of baseline. A paediatrician assessed the condition of the neonates at delivery using Apgar scores at 1 and 5 min, and blood samples from double-clamped umbilical arteries were obtained immediately after delivery for determination of acid–base status.

Values are presented as mean (SD, range or 95 % confidence intervals (CI)), where appropriate. Values for maternal SAP were also computed as percentage changes from baseline values for later analysis. Paired Student’s t test was used for evaluation of maximum haemodynamic changes from baseline. Analysis of variance for repeated measures was used to assess the statistical significance of the haemodynamic changes during spinal anaesthesia. The association between the changes in maternal haemodynamic variables and uterine artery PI was assessed by calculating Pearson’s correlation coefficient. Differences were considered statistically significant if P < 0.05. All statistical tests were performed on a standard PC using the program SPSS for MS Windows release 6.1 (SPSS Inc., USA).

Results

Baseline maternal and newborn data are shown in tables 1 and 2. Mean maternal HR did not change significantly during preloading and spinal anaes-

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Maternal characteristics (mean (SD or range) or number). Maternal systolic (SAP), mean (MAP) and diastolic (DAP) arterial pressures are mean values 1 day before Caesarean section</th>
</tr>
</thead>
<tbody>
<tr>
<td>n = 12</td>
<td></td>
</tr>
<tr>
<td>Maternal</td>
<td></td>
</tr>
<tr>
<td>Age (yr)</td>
<td>27.3 (21–37)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>73.9 (9.3)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>160.2 (4.9)</td>
</tr>
<tr>
<td>SAP (mm Hg)</td>
<td>170.5 (149–194)</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>127.2 (112–141)</td>
</tr>
<tr>
<td>DAP (mm Hg)</td>
<td>105.5 (88–125)</td>
</tr>
<tr>
<td>Proteinuria (g 24 h⁻¹)</td>
<td>6.8 (1–14)</td>
</tr>
<tr>
<td>Weeks of pregnancy</td>
<td>34.9 (31–40)</td>
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<tr>
<td>Parity (n)</td>
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<tr>
<td>Primiparae</td>
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<td>Multiparae</td>
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Atropine was not required. Mean maternal SAP and DAP decreased significantly after induction of spinal anaesthesia (\(P < 0.05\)) (fig. 2). Mean SAP decreased from 169 (95% CI 157–180) mm Hg to a minimum of 137 (120–154) mm Hg (\(P < 0.001\)) and mean DAP from 112 (105–118) mm Hg to 90 (79–101) mm Hg (\(P < 0.001\)) during spinal anaesthesia. The mean maximum percentage decrease in SAP from baseline was 19 (12–26)%. The incidence of maternal hypotension was 17% (two of 12 patients). Of these two patients, one had marked hypotension and her SAP decreased to 51% (80 mm Hg) of baseline, 6 min after induction of spinal anaesthesia. In this patient, sensory block extended to C5 and the patient needed assisted ventilation of the lungs with 100% oxygen via a face mask and a bag for 10–15 min. Oxygen saturation remained at 98–99%, however, and hypotension responded to the increased crystalloid infusion and ephedrine 10 mg i.v. The patient remained conscious and cooperative and had no convulsions and, therefore, general anaesthesia was not considered. This patient delivered a 1925-g male infant with Apgar scores of 9 and 8 at 1 and 5 min, respectively. Unfortunately, determination of umbilical artery pH was not managed satisfactorily. In the other hypotensive patient, SAP decreased to 71% (124 mm Hg) of the baseline value and was also corrected rapidly with ephedrine 10 mg.

Mean CVP increased significantly after preloading from 2.3 (95% CI 1.0–3.7) mm Hg to 6.8 (4.8–8.9) mm Hg (\(P < 0.001\)) and returned towards baseline values shortly after induction of spinal anaesthesia, after which CVP tended to increase until delivery (fig. 3). In two patients, CVP increased to 10 and 13 mm Hg after preload, but decreased immediately after induction of spinal anaesthesia to 5 and 6 mm Hg, respectively. The total volume of crystalloid infused before delivery was approximately 2108 (range 1500–2400) ml. No patient had any signs of pulmonary oedema during the study.

Mean PI of the uterine artery did not change significantly (\(P = 0.28\)) during the study (fig. 4) and there was no significant correlation with changes in SAP. In one patient, there was a marked increase in uterine artery PI from 0.73 to 4.22 during a period of

Table 2 Newborn characteristics (mean (range) or number).
<table>
<thead>
<tr>
<th>I–D time = induction of anaesthesia to delivery time. (*n = 7)</th>
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<tbody>
<tr>
<td>Newborn weight (g)</td>
</tr>
<tr>
<td>Umbilical artery pH*</td>
</tr>
<tr>
<td>Apgar scores (n)</td>
</tr>
<tr>
<td>1 min (&lt; 7)</td>
</tr>
<tr>
<td>5 min (&lt; 7)</td>
</tr>
<tr>
<td>I–D time (min)</td>
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</tbody>
</table>

Figure 1 Mean (95% CI) changes in maternal heart rate (HR) during the study. There were no significant changes. B = Before preload, A = after preload, S = spinal anaesthesia. Time = minutes after injection of intrathecal bupivacaine.

Figure 2 Mean (95% CI) changes in maternal systolic (SAP) and diastolic (DAP) arterial pressures during the study. SAP and DAP decreased significantly after induction of spinal anaesthesia (\(*P < 0.05\)). B = Before preload, A = after preload, S = spinal anaesthesia. Time = minutes after injection of intrathecal bupivacaine.

Figure 3 Mean (95% CI) changes in central venous pressure (CVP) during the study. CVP increased significantly after preload (**\(P = 0.001\)) and returned towards baseline immediately after induction of spinal anaesthesia. B = Before preload, A = after preload, S = spinal anaesthesia. Time = minutes after injection of intrathecal bupivacaine.

Figure 4 Mean (95% CI) changes in pulsatility index (PI) of the uterine artery during the study. There were no significant changes. B = Before preload, A = after preload, S = spinal anaesthesia. Time = minutes after injection of intrathecal bupivacaine.
maternal hypotension, when SAP decreased to 71 % (124 mm Hg) of baseline, 12 min after induction of spinal anaesthesia. In the case of the most marked decrease in SAP to 51 % of the baseline value, PI of the uterine artery increased from 0.73 to 2.17. We also divided the group into two subgroups of mild and severe pre-eclampsia and found no significant difference in changes in maternal haemodynamic variables (HR, SAP, DAP and CVP) or utero-placental circulation (uterine artery PI) between these subgroups.

Eight of the 12 newborns were small for gestational age (<2.5th percentile). Umbilical artery pH was obtained in seven patients and varied from 7.14 to 7.42. All neonates had Apgar scores of 7 or more at 5 min. After operation, all mothers were observed intensively for the first 24 h, and recovery was uncomplicated in every case.

Discussion

We found reasonably stable maternal and utero-placental haemodynamic values in pre-eclamptic parturient patients undergoing Caesarean section during spinal anaesthesia after prophylactic pre-loading with 1 litre of crystalloid solution. Severe hypotension was seen in only two patients, and this was corrected rapidly by increasing the crystalloid infusion and giving small doses of ephedrine 5–10 mg i.v. However, the absolute mean decrease in SAP in our study was 19 %, which is similar to the decrease of 23 % reported in an earlier study [9]. That retrospective study failed to reveal any significant difference between extradural and spinal anaesthesia for Caesarean section in severely pre-eclamptic parturients with regard to changes in maternal haemodynamics or total intraoperative ephedrine doses used.

The cardiovascular state of severely pre-eclamptic patients has been studied intensively and hyperdynamic function of the left ventricle has been demonstrated in 73–80 % of patients [10, 11]. In these two studies, there was a normal-to-high cardiac output with inappropriately high peripheral resistance. Cotton and colleagues [11] hypothesized that the increased left ventricular afterload contributes to an increase in pulmonary capillary wedge pressure despite the hypovolaemic state associated with pre-eclampsia and that central venous pressure may therefore reflect the intravascular volume status more accurately than pulmonary capillary wedge pressure. Because none of our patients had pulmonary oedema or persistent oliguria, we used CVP monitoring instead of pulmonary capillary wedge pressure to assess central blood volume during preloading and spinal anaesthesia.

Pre-eclamptic parturients have been shown to suffer from relative hypovolaemia throughout their pregnancy compared with normal parturients [12], and rapid volume expansion with crystalloid [13, 14] or colloid solution [15, 16] has therefore resulted in beneficial effects in pre-eclamptic patients. Infusion of crystalloid solution 15 ml kg⁻¹ led to a decrease in systemic vascular resistance [13, 14] and an increase in cardiac output [14] in pre-eclamptic mothers. Infusion of 5 % albumin 500–1000 ml [15] or 3.5 % colloid plasma substitute 200–600 ml [16] also resulted in an increase in cardiac index and a decrease in systemic vascular resistance. After rapid volume expansion, mean SAP was unchanged [16] or slightly reduced [15]. Grunewald and colleagues [13] demonstrated no change in uterine artery PI after rapid volume expansion. Hence, volume expansion in pre-eclamptic patients with either crystalloid or colloid solution appears to be a reasonable first measure to restore central blood volume and to stabilize maternal haemodynamic state, especially before induction of central neural block. In our study, mean initial CVP was 2.3 mm Hg, it increased markedly to 6.8 mm Hg after volume expansion and remained slightly less than 6 mm Hg after induction of spinal anaesthesia. These stable values during spinal anaesthesia, which were at physiological normal levels, imply that spinal block did not significantly affect maternal central blood volume in pre-eclamptic patients, as there were no large alterations in CVP.

Mean maternal HR did not change significantly during the study despite the significant decrease in SAP. There is at least one potential explanation for this stability. Of the 12 patients in our study, 10 were using labetalol. This drug may have prevented a compensatory increase in maternal HR. Labetalol, on the other hand, does not appear to affect uteroplacental blood flow in pre-eclampsia [17, 18].

Robson and colleagues [19] have reported a decrease in uteroplacental blood flow secondary to reduced maternal cardiac output and stroke volume during spinal anaesthesia in healthy parturients. In this study, uterine artery PI values did not change significantly during preload or spinal anaesthesia. This is in contrast with our recent findings [20], which demonstrated occasionally high but transient increases in uterine artery PI in some healthy parturients during spinal anaesthesia. These increases, which reflected increased vascular resistance, did not, however, relate to any obvious changes in individual maternal SAP, HR, or CVP. This difference in the changes in uterine artery resistance (PI) between normal and pre-eclamptic parturients was surprising, and we can only speculate on the underlying reasons. Although we did not have a control group in this study, we are able to compare our results with those of an earlier study by our team using extradural anaesthesia [5]. In that study, Alahuhta and colleagues demonstrated no change in uterine artery PI in hypertensive mothers with chronic fetal asphyxia after extradural anaesthesia with plain bupivacaine and no patient had profound hypotension or required a vasopressor. Eneroth-Grimfors and colleagues [21] observed decreased vagal control of the heart in pre-eclampsia. These changes in the cardiovascular state of pre-eclampsia together with the α and β blocking agent, labetalol, used by the majority of our patients may account for the different results obtained in healthy and pre-eclamptic parturients during spinal anaesthesia.

Our finding, that uteroplacental blood flow did not change markedly during spinal anaesthesia, is in accordance with our earlier data [22]. In that study, placental blood flow, recorded with an isotope...
technique, did not change in seven healthy parturients and even increased in one patient with pre-eclampsia during spinal anaesthesia. In the 12 pre-eclamptic patients in this study, a marked increase in uterine artery PI as a sign of increased vascular resistance was seen in only one patient simultaneously with a period of severe hypotension lasting 4–6 min after induction of spinal anaesthesia. This was, however, corrected promptly with adequate measures. In the other patient, with even more marked hypotension, uterine artery PI increased only slightly and normalized rapidly. It seems therefore that PI levels in uterine arteries are stable and not sensitive to a moderate decrease in maternal arterial pressure. The good condition of the neonates was a further indication of undisturbed uterine haemodynamic state during the study. The small number of pre-eclamptic subjects examined here may, however, have limited the possibility of observing significant changes in uterine artery PI values after marked maternal hypotension.

**Acknowledgement**

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**References**