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Effects of epidural analgesia on uterine artery Doppler in labour

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Editor's key points

- Previous studies have shown that in some circumstances, epidural analgesia is also associated with a reduction in uterine blood flow.
- In this study in labouring women, uterine artery pulsatility index increased (suggesting reduced uterine blood flow) during contractions 30 min after epidural ropivacaine 0.1%.
- These changes were more marked than in a group of women in labour without analgesia.
- No significant changes in uterine blood flow were seen during periods of uterine relaxation.

Background. The effects of epidural anaesthesia on maternal uteroplacental blood flow in the presence of uterine contractions remain unclear. The aim of our study was to evaluate the effects of epidural analgesia with bolus doses on uterine artery pulsatility index (UtA-PI) during labour.

Methods. In a prospective case–control study, UtA-PI was measured during uterine contraction and relaxation in nulliparous women in active labour with (epidural group) and without (control group) epidural analgesia. Patients in the two groups were matched for gestational age at delivery, American Society of Anesthesiologists physical status score, and cervical dilatation at the beginning of labour. In the epidural group, an epidural catheter was placed after prehydration with 500 ml i.v. saline, and sufentanil 2 μg ml^{-1} (5 ml) and ropivacaine 1 mg ml^{-1} (20 ml) were administered. UtA-PI was measured before (T0), 30 min (T30), and 90 min (T90) after the first administration of epidural analgesic drugs, during both uterine relaxation and contraction.

Results. Fifty-two patients were included in the study, 33 in the epidural group and 19 in the control. UtA-PI was significantly higher in the epidural compared with the control group, only at T30 and during contraction. There were no differences in the rate of oxytocin augmentation, mode of delivery, birth weight, and umbilical artery pH between the two groups.

Conclusions. Epidural analgesia using ropivacaine 1 mg ml^{-1} (20 ml) significantly reduced placental blood flow only transiently during uterine contraction 30 min after the injection. These changes did not seem to affect neonatal outcomes.

Keywords: anaesthetics local, ropivacaine; analgesia, epidural; analgesia, obstetric; analgesics, opioid, sufentanil; blood flow velocity; Doppler ultrasonography; labour, obstetric

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The effects of epidural anaesthesia on maternal uterine blood flow velocity waveforms have already been reported.[1–4] However, previous studies did not compare Doppler indices during the contraction and relaxation periods, and there were no control parturients in labour without epidural or other analgesia. The aim of our study was to evaluate the effects of analgesia with a top-up technique on uterine artery Doppler during labour, in particular during the contraction and the relaxation phases.

Methods

This was a prospective case–control study performed during a 6 month period in the labour ward of Brescia University Hospital, Italy. Uterine artery Doppler indices were measured during uterine contraction and relaxation in nulliparous women in active labour with (cases: epidural group) and without (control group) epidural analgesia. The study was approved by the local ethical committee, and all women gave their written informed consent. Inclusion criteria were: gestational age $\geq$37 weeks; singleton pregnancy with an engaged vertex presentation; no significant medical or obstetric complications; normal platelet count and coagulation profile; active labour with cervical dilatation $>$3 cm and uterine contractions occurring at least every 5 min; normal cardiotocography (CTG) [baseline fetal heart rate (FHR) between 110 and 160 beats min^{-1}, baseline variability $>$5 beats min^{-1}, presence of accelerations, and absence of decelerations]; no evidence of fetal or maternal compromise before or during labour. Exclusion criteria were: maternal age $<$18 yr, poor parturient compliance, pre-eclampsia, gestational diabetes, and cardiovascular diseases. Patients in the two groups were

matched for gestational age at delivery in weeks, American Society of Anesthesiologists physical status score, and cervical dilatation at the beginning of labour, with a 2:1 case:control ratio. All women labouring without analgesia admitted to the labour ward immediately after each second case were assessed and enrolled in the study as controls when meeting inclusion and matching criteria with one of the previous two cases. Uterine contractions were assessed with an external tocodynamometer during labour and with manual palpation during uterine artery Doppler assessment. Epidural analgesia was at the woman's request and, in the absence of any contraindications, was administered by the anaesthetist present on the labour ward. Each patient received an i.v. infusion of 500 ml of saline solution, which was completed 20 min before the beginning of the study. In the epidural group, an epidural catheter was placed in the L3–4 interspace under sterile conditions, and after a negative aspiration test, a dose (3 ml) of lidocaine 2% was injected. The control group received no analgesics during the study. Maternal arterial pressure and heart rate were monitored at 5 min intervals. Once it was determined that no adverse effects such as maternal hypotension or fetal bradycardia had occurred, sufentanil 2 μg ml^{-1} (5 ml) and ropivacaine 1 mg ml^{-1} (20 ml) were administered to ensure a diminished pin-prick sensation caudal to the T10 dermatome. Efficacy of analgesia was assessed by a midwife using 100 mm visual analogue pain scores (VAS), where 0 represented 'no pain' and 100 was 'worst possible pain' at 5 min intervals for the first 30 min after bolus injection. When VAS was $\geq$40, a second dose of local anaesthetic was required and patients were excluded from the study. Uterine artery pulsatility index (UtA-PI) during uterine relaxation and contraction was measured before (T0), 30 min (T30), and 90 min (T90) after the beginning of analgesia in the epidural group and at the beginning of labour (T0), 30 min (T30), and 90 min (T90) later in Group B. The right and left uterine arteries were identified by colour flow at the apparent crossover with the external iliac arteries, and pulsed-wave Doppler was used to obtain waveforms. When three similar, consecutive waveforms were obtained, the PI was measured and the mean UtA-PI calculated. An Aloka SSD 1700 ultrasound machine with 3.5 MHz convex transabdominal probe was used. Maternal arterial pressure parameters were recorded at T0, T30, and T90. The fetus was monitored by continuous CTG. The details of the FHR record, uterine activity patterns, along with birth weight, Apgar scores at 5 min, and umbilical arterial pH were recorded. For statistical analysis, Student's *t*-test, Mann–Whitney *U*-test, χ^2 test, Fisher's exact probability test, ANOVA, and Pearson's correlation test were used as appropriate. *P*-values $<$0.05 were considered statistically significant.

Results

Seventy-two patients were enrolled in the study: 51 in the epidural group and 25 in the control group. Subsequently, 15 women in the epidural group and nine in the control group were excluded from the study because of incomplete data (n=14), poor compliance (n=4), inadequate analgesia with VAS $>$40, or accidental dural puncture (n=2). Among the 52 remaining patients, 33 received top-up epidural analgesia and 19 did not. Maternal and delivery characteristics in both groups are listed in Table 1. Mean cervical dilatation at T0 was 4 cm in both groups. Mean UtA-PI values during uterine contraction and relaxation are shown in Table 2. As expected, in both groups, mean UtA-PI was significantly higher during contraction than relaxation at T0, T30, and T90 (all $P<0.01$, not shown in the table). During relaxation, the mean UtA-PI was not significantly different between the epidural and control group. However, in the epidural group, 30 min after administration of epidural analgesia (T30), the mean UtA-PI measured during contraction was significantly higher than in the control group. There were no statistically significant differences at T0 and T90. Table 3 shows mean arterial pressure during relaxation and contraction in both groups at T0, T30, and T90. None of the women receiving analgesia required ephedrine for hypotension. Pearson's test showed that the UtA-PI during contraction at T30 in the epidural group did not significantly correlate with maternal mean arterial pressure (r=0.35, P=0.21). FHR pattern was classified as normal in all patients at the time of inclusion in the study (T0); there were no cases of significant fetal tachycardia, bradycardia, or decelerations, at T30 and T90 in either group.

Uterine activity pattern was recorded in both groups at T0, T30, and T90. In the epidural group, the median (range) number of contractions was 4 (2–6) in 10 min at T0, 4 (2–6) in 10 min at T30, and 4 (2–6) in 10 min at T90. In the

Table 1 Maternal characteristic and delivery characteristics in patients with (epidural group) and without (control group) analgesia. Continuous variables are shown as mean (SD) or median (inter-quartile range), as appropriate

	Epidural group (n=33)	Control group (n=19)
Maternal age (yr)	30 (28–33)	28 (26–33)
Gestational age at delivery (weeks)	39^{+4} (39^{+0}–40^{+3})	39^{+4} (39^{+2}–40^{+1})
ASA physical status score	I (I–I)	I (I–I)
Cervical dilatation at the beginning of labour (cm)	4 (3–4)	4 (4–5)
Spontaneous labour (n)	27	17
Induction of labour with prostaglandins (n)	6	2
Oxytocin for augmentation of labour (n)	10	6
Caesarean section (n)	3	2
Operative vaginal delivery (n)	8	6
Birth weight (g)	3630 (452)	3420 (387)
Umbilical artery pH	7.23 (0.06)	7.25 (0.10)
Umbilical artery base excess (mmol litre^{-1})	−3.7 (2.9)	−3.7 (2.9)
Apgar score at 5 min	9 (9–10)	10 (9–10)

Table 2 Mean (SD) UtA-PI during uterine contraction and relaxation in patients with (epidural group) and without (control group) analgesia. *$P<0.01$ between the epidural and control groups. †$P<0.01$ within the group over time

	Relaxation		Contraction	
	Epidural group	Control group	Epidural group	Control group
T0 (baseline)	0.68 (0.10)	0.66 (0.16)	1.03 (0.27)†	1.00 (0.20)
T30 (30 min)	0.78 (0.23)	0.61 (0.22)	1.30 (0.37)†	1.00 (0.28)*
T90 (90 min)	0.74 (0.17)	0.69 (0.17)	1.25 (0.42)†	1.00 (0.30)

Table 3 Mean (SD) maternal mean arterial pressure (mm Hg) during uterine contraction and relaxation in patients with (epidural group) and without (control group) analgesia

	Relaxation		Contraction	
	Epidural group	Control group	Epidural group	Control group
T0 (baseline)	89 (10)	88 (7)	93 (9)	89 (10)
T30 (30 min)	88 (8)	87 (9)	93 (8)	95 (11)
T90 (90 min)	88 (11)	93 (10)	91 (11)	97 (9)

control group, the median (range) number of contractions was 4 (3–5) in 10 min at T0, 4 (3–5) in 10 min at T30, and 4 (3–5) in 10 min at T90. Differences in uterine activity were not significant between different times nor between the groups. Caesarean section was performed in three of the 33 patients in the epidural group and in two of the 19 patients in the control group because of failure of labour to progress (Table 1).

Discussion

In our study, UtA-PI measured during contraction was significantly increased 30 min after administration of ropivacaine 0.1% in women labouring with epidural analgesia when compared with PI measured in women labouring without analgesia. This increase in uterine arterial impedance, however, was not associated with neonatal acidosis or low Apgar scores at birth. Chen and colleagues[1] suggested that continuous epidural analgesia for painless labour with bupivacaine 0.075% increased uterine blood vessel impedance possibly reducing uterine blood flow during labour. However, in this study, there were no controls labouring without epidural analgesia. Similar results were shown by a study evaluating uterine circulation during analgesia for Caesarean section demonstrating that the UtA-PI increased significantly after ropivacaine 0.5% administration, but only on the non-placental side and not until sensory analgesia had reached the T6–T4 level.[5] In our study, we used ropivacaine at a lower concentration, and sensory analgesia reached the T10 level which according to the ASA guidelines corresponds to a lower level and less profound spinal block.[6] Ropivacaine is a long-acting amide local anaesthetic that has been shown in animal studies to have less dysrhythmogenic and cardiotoxic potential than bupivacaine, and its i.v. administration has not been associated with any detrimental effects on uterine blood flow in pregnant ewes.[7–10]

We demonstrated a significant increase in UtA-PI 30 min after the onset of epidural, but this increase did not persist 90 min after the beginning of analgesia, when the action of ropivacaine would be diminished. During pregnancy, uterine blood flow is proportional to perfusion pressure and inversely related to vascular resistances depending on vascular tone, myometrial tone, and contractions. At the end of the pregnancy, the uterine vascular bed is maximally dilated without chances of further dilation or autoregulation. We hypothesize that increased impedance measured during uterine contraction at T30 might be related to an insufficient increase in preload because of epidural anesthesia-induced sympathetic block.

Our hypothesis is confirmed by a previous study from Manninen and colleagues[11] showing that the PI of maternal femoral artery decreased after onset of epidural analgesia, indicating epidural-induced vasodilation. It has been suggested that maternal hypotension related to epidural analgesia is associated with an increase in the Doppler indices for the uterine arteries.[11–13] In our study, we performed prehydration with 500 ml of saline solution and we did not record any significant case of maternal hypotension. Moreover, we could not demonstrate a relationship between UtA-PI during contraction at T30 in the epidural group and maternal mean arterial pressure. These findings underline the importance of administration of i.v. fluids before epidural analgesia in order to prevent reduction of placental blood flow. Our hypothesis is that at T30, the time of peak action of ropivacaine, myometrial contraction increased vascular resistance at such a point that increased preload was not enough to prevent a transient detrimental effect on placental blood flow. In our study, we did not measure force of uterine contractions, so we cannot define to what extent the effect of epidural analgesia in increasing uterine artery resistance could be related to increased force of uterine contractions.

Modifications of uterine blood flow induced by epidural analgesia were not associated with neonatal acidosis or low Apgar scores at birth. A possible explanation is that during uterine relaxation, mean UtA-PI was not significantly different between the epidural and control group, and UtA-PI measured during contraction was not significantly different in the two groups before epidural (T0) and at ropivacaine's nadir (T90). In conclusion, our results show that, given adequate i.v. saline pre-hydration, epidural analgesia using

ropivacaine 0.1% significantly reduces placental blood only transiently 30 min after the injection, when the action of local anaesthetic peaks, and during uterine contraction only. These changes do not seem to affect FHR pattern, uterine activity, or neonatal outcomes. In this study, we used sufentanil 10 μg in 5 ml of saline plus ropivacaine 0.1% (20 ml). Smaller dose combinations have been suggested to be equally effective for epidural labour analgesia.[14 15] It may be that smaller dose combinations would have led to smaller effects on placental flow. Further studies are needed to evaluate if in patients with increased impedance in uterine artery, effects of epidural analgesia might be clinically significant.

Conflict of interest

None declared.

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