Normal saline i.v. fluid load decreases uterine activity in active labour

T. G. Cheek, P. Samuels, F. Miller, M. Tobin and B. B. Gutsche

Summary
This study was designed to observe the effects of i.v. fluid infusion on uterine activity during normal labour in women receiving an extradural block. Thirty-four women in spontaneous labour at term gestation were allocated randomly and prospectively to one of three i.v. pre-extradural fluid load groups: group A, no fluid load; group B, normal saline 500 ml; and group C, normal saline 1000 ml. Continuous internal measurement of uterine activity was observed before, during and after fluid infusion and extradural block. In groups A and B, uterine activity did not change. In group C, uterine activity decreased after infusion of saline (P < 0.01) and returned to baseline over the next 20 min. Extradural block was not associated with a change in uterine activity. Hypotension was not increased in the group that received no fluid preload. (Br. J. Anaesth. 1996; 77: 632–635)

Key words
Fluids, i.v. Anaesthetic techniques, extradural. Uterus, activity.

There is disagreement on the effect of lumbar extradural block on uterine activity and its influence on the progress of labour.1–5 It is presently common obstetric practice in the USA to infuse 500–1000 ml of i.v. normal saline as initial treatment in the tocolytic therapy of preterm labour.6 The purpose of this study originated from the question: if an i.v. fluid load of normal saline 500–1000 ml effectively inhibits uterine activity in more than 50% of subjects in preterm labour, what is the effect of similar fluid load during normal active labour? If uterine activity is inhibited by such a fluid load, could this explain observations of decreased uterine activity after extradural block, a procedure normally preceded by an i.v. fluid load? This study was designed to observe the individual effects of i.v. fluid load and extradural block on uterine activity during normal active labour.

Patients and methods
After obtaining approval from the Institutional Committee for Human Studies and informed consent, we studied 34 healthy, ASA I–II term parturients in spontaneous labour. Patients were allocated randomly and prospectively to one of three pre-extradural i.v. fluid load groups: group A, no fluid load; group B, normal saline 500 ml infused over 10 min; group C, normal saline 1000 ml infused over 10 min. All subjects received maintenance i.v. fluids: 5% glucose 125 ml h⁻¹ in 1/2; normal saline from admission to the labour ward to just before the time of study (mean = 550 ml). None of the subjects received any medication before the study. Both primiparae and multiparae were studied. Extradural analgesia was performed with the subject in the lateral position with a total of 12 ml of 0.25% bupivacaine without adrenaline (a 3-ml test dose followed by 9 ml). After insertion of the extradural, continuous uterine displacement with a right hip wedge was maintained throughout the study. Fetal heart rate and maternal arterial pressure were noted at 5-min intervals and treatment for maternal hypotension was recorded. Maternal arterial pressure was determined at 1-min intervals for 5 min and at 5-min intervals thereafter for the first 30 min after local anaesthetic injection. If maternal arterial pressure decreased to less than 100 mm Hg for more than 1–2 min, i.v. ephedrine was given in 5-mg increments to obtain an arterial pressure of >100 mm Hg.

Continuous internal measurement of uterine activity was divided into 7–10-min intervals: 10 min of baseline uterine activity, 10 min of fluid infusion, 20 min (2 × 10 min) for recovery from possible fluid load effects, and for 30 min after extradural injection. Measurements were continuous after both fluid load and extradural injection, with 10 min allowed between measurements for insertion of the extradural catheter. Intrauterine pressure was measured with catheters positioned by an obstetrician in the uterine cavity. These were placed electively for the purpose of the study and not for obstetric indications. Quantitative measurement of...
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uterine activity units, frequency, duration and intensity of contractions were calculated from the area under the graph curve produced on tracing paper from the fetal heart monitor (Corometrics). Calculation of uterine activity for each interval was made by the same obstetrician (P. S.) who was unaware of the amount of fluid load or the time of extradural injection. One uterine activity unit is equivalent to 50 mm Hg s⁻¹.⁷

Two subjects were excluded in group A because precipitous delivery occurred before extradural block was performed. One subject each from groups B and C received fluid load measurements but rapidly progressed to delivery before extradural block could be accomplished. Constraints during the measurement period included avoidance of change in subject position, voluntary pushing or intraterine pressure system flushing. Measurements were not made if the subject vomited, in the presence of asymmetric tracing or if there was a suspicion of system malfunction. Cervical dilatation at the time of measurements was 4–6 cm.

Statistical analysis included paired Student’s t test for patient variables. Analysis of variance over time within and between groups was determined for uterine activity with Tukey’s test because of the number of subjects involved.

Results

There were no differences between groups in maternal age, height, weight, gestational age or cervical dilatation at the start of the study (table 1).

In group A (maintenance fluid only), uterine activity did not change during any period. In group B (fluid infusion of 500 ml i.v.), there was a decrease in uterine activity after the infusion, but this was not significant. In group C (infusion of 1000 ml i.v.), uterine activity decreased after the infusion (P < 0.01) but returned to baseline over the next 20 min. Mean uterine activity did not decrease in any group after induction of extradural block. Uterine activity in the three groups in response to fluid load and extradural block is summarized in figure 1. There were no differences between groups in changes in maternal arterial pressure and there were no abnormal changes in fetal heart rate during the study. Duration of labour was not different between groups after initiation of the study.

There were no differences in incidence and severity of hypotension between the groups.

Discussion

Several factors associated with extradural block are reported to decrease uterine activity, including inadvertent aorto-caval compression, maternal hypotension, β agonist activity from low-dose adrenaline in the local anaesthetic solution, and possible direct local anaesthetic effects on the myometrium. After controlling for these variables, our data indicated that an i.v. fluid load before regional block caused a transient decrease in uterine activity.

Rout and co-workers have reported attenuated protection against hypotension with pre-spinal i.v.

![Figure 1](image_url) Uterine activity (mean, SD) after no fluid preload (0 ml) and after i.v. infusion of 500 and 1000 ml. B = Baseline, I = infusion, Extra. = extradural injection.

**Table 1**  Patient characteristics (mean (sd or range)) or number

<table>
<thead>
<tr>
<th></th>
<th>Group A (no preload)</th>
<th>Group B (500 ml)</th>
<th>Group C (1000 ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>11</td>
<td>8</td>
<td>11</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>20.7 (16–31)</td>
<td>22.7 (18–33)</td>
<td>21.1 (16–29)</td>
</tr>
<tr>
<td>Gestational age (weeks)</td>
<td>39.5 (38–40)</td>
<td>39 (37–40)</td>
<td>39 (38–40)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>76.4 (6)</td>
<td>79.7 (6.5)</td>
<td>75.5 (9.5)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>64.7 (0.7)</td>
<td>65.3 (0.5)</td>
<td>62.8 (1.8)</td>
</tr>
<tr>
<td>Nullipara</td>
<td>8</td>
<td>5</td>
<td>7</td>
</tr>
<tr>
<td>Multipara</td>
<td>3</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Cervical dilatation at study (cm)</td>
<td>5 (0.3)</td>
<td>4 (0.3)</td>
<td>4 (0.4)</td>
</tr>
<tr>
<td>I.v. fluids before study (ml h⁻¹)</td>
<td>125</td>
<td>125</td>
<td>125</td>
</tr>
<tr>
<td>Forceps</td>
<td>3/11</td>
<td>2/8</td>
<td>3/11</td>
</tr>
<tr>
<td>Vaginal delivery</td>
<td>9/11</td>
<td>7/8</td>
<td>9/11</td>
</tr>
<tr>
<td>Caesarean delivery</td>
<td>2/11</td>
<td>1/8</td>
<td>2/1</td>
</tr>
</tbody>
</table>
fluid loading during Caesarean section. On a smaller scale we found that subjects who received no fluid load had no greater incidence or severity of hypotension after 0.25% bupivacaine 12 ml than subjects who received 1000 ml i.v. fluid preload. It may be that as all subjects received maintenance i.v. fluids, intravascular volume was adequate to compensate for the increased vascular capacitance associated with a T9 (mean) sympathectomy. Our data suggest that a fluid load of 1 litre i.v. did not decrease the incidence of maternal hypotension in labouring patients.

The incidence of slowing or arrest of labour, as defined by failure of cervical dilatation in the absence of extradural block, was reported by Bottoms, Sokol and Rosen as approximately 20%, with 5.5% defined as secondary arrest requiring obstetric intervention. Arrest or slowing of labour was associated most commonly with decreased uterine activity. This indicates that under normal conditions one of five parturients experiences decreased uterine activity during labour, independent of external medication.

The effect of extradural block on labour and uterine activity has been debated almost from the beginning of its use in obstetrics. Unfortunately, the clinical evidence offered in these arguments is largely anecdotal, uncontrolled, non-randomized and usually retrospective. Friedman and Sachtleben, in 1959, reported that caudal block to T10 did not alter uterine activity but that higher levels of block were associated with longer labours. This report was not based on a prospective, randomized series and maternal position or arterial pressure after caudal block was not reported. Lowensohn and colleagues prospectively studied the effect of extradural lignocaine and propitocaine (a drug no longer in use) and reported a decrease in uterine activity after each injection of lignocaine that lasted approximately 20 min.

This study was flawed by the addition of adrenaline to the local anaesthetic injectate, and neither the amount of preblock fluid loading nor the assurance of uterine displacement were reported. Quantitation of uterine activity during labour cannot be done with external tocodynamometry. Accurate measurement requires intra-cavity placement of either a transducer or a fluid-filled catheter with a pressure transducer connected to a fetal monitor where the signal is expressed as a continuous tracing calibrated in mm Hg. Earlier investigators estimated uterine activity in Montevideo units where peak height of the contraction was multiplied by the number of contractions. Hon and Paul described a more precise method for quantitation of uterine activity measurement in which the integral under the uterine contraction curve was calculated and expressed in “torr seconds”. In this study, uterine activity was estimated by directly measuring the area under the uterine contraction curve and above baseline tone.

Despite strict adherence to uterine displacement, avoidance of maternal hypotension and pre-block i.v. fluid load, transient decreases in uterine activity are still observed occasionally after extradural block for analgesia in labour. Normal uterine activity requires a complex interplay between ongoing uterine smooth muscle biochemical changes and a set of external maternal and fetal signals. These external signals may be neural, or hormonal; the uterine smooth muscle and vascular system are innervated by sympathetic and peptidergic fibres and their transmitter and receptor content changes during pregnancy. Our data suggest that prehydration before extradural block attenuated uterine activity. There are several possible explanations for decreased uterine activity observed after a fluid load. Valenzuela, Cline and Hayashi speculated that oxytocin and vasopressin secretion are inhibited directly at the neurohypophysis. Transient dilution of plasma oxytocin concentrations by a fluid load may temporarily attenuate uterine activity. Both of these suggestions are in dispute because the effect of oxytocin on uterine activity is no longer thought to be dependent on circulating oxytocin concentrations but related rather to the number of myometrial oxytocin receptors and their degree of activity.

Another suggestion is that the temperature (~22°C) of the infused i.v. solution may affect uterine activity. This is unlikely because cooling of the blood by 1 litre of room temperature saline would be both small and rapidly dissipated.

Rapid volume expansion increases transmural atrial distending pressure. This stimulates vagal neural afferents which may decrease posterior pituitary hormone secretion and sympathetic (particularly renal) nerve activity, and perhaps efferent peptidergic nerve activity. We speculate that the mechanism may lie either in a sudden humoral release, such as atrial natriuretic peptide (ANP) after rapid atrial distension, or a direct mechanical effect on the uterine vasculature producing local release of endothelial vasoactive peptides. Bek, Ottesen and Fahrenkrug have shown ANP to be a potent inhibitor of rat uterine smooth muscle contraction.

Recently Eledjam and co-workers reported a 172% increase in ANP plasma concentrations of labouring women 15 min after i.v. fluid challenge with dextran 40 250 ml. ANP is a 28 amino acid peptide which is secreted directly by the atrium into the coronary veins, and causes vasodilatation, natriuresis and diuresis, and inhibition of secretion or end-organ effects of several vasoactive hormones, including angiotensin II and noradrenaline. Concentrations of ANP increase slightly during pregnancy, and increase further with volume loading. In contrast, Grunewald and colleagues infused 0.9% Ringer’s lactate 15 ml kg⁻¹ in normal parturients over 30 min. This caused a decrease in ANP from 6.5 to 4.6 pmol litre⁻¹. This may indicate that different i.v. infusates have different effects on ANP concentrations. ANP receptors are present in the non-pregnant rat uterus and inhibit uterine contraction and blood flow. The time course of ANP release and action is consistent with the role of ANP in decreasing uterine activity during volume loading. However, the overall role of ANP in parturition is unknown.

References
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