EFFECT OF MATERNAL POSITION ON FETAL HEART RATE DURING EXTRADURAL ANALGESIA

K. HUOVINEN AND K. TERAMO

SUMMARY

Using continuous cardiotocography, the effect of maternal position on fetal heart rate (FHR) was studied during extradural analgesia (EA) with either 50 or 100 mg of etidocaine without adrenaline for labour and vaginal delivery. Of 79 healthy parturients, 39 were supine and 40 were in the lateral position. FHR was normal in all patients during the control period before EA, and remained normal during EA in 53. Transient abnormal patterns occurred in 26 patients, 22 (56%) in the supine group and four (10%) in the lateral group, a significant difference (P<0.001). The decrease in arterial pressure (AP) did not differ between the groups, but the frequency of abnormal FHR associated with a small or moderate decrease in AP (30% or less) was greater in the supine (51%) than in the lateral group (3%).

Extradural analgesia (EA) has been reported to have beneficial effects on mother and fetus and on the newborn infant (Pearson and Davies, 1974; Thalme, Belfrage and Raabe, 1974; Zador and Nilsson, 1974). However, continuous cardiotocographic recordings of the fetal heart rate (FHR) have revealed a varying number of abnormal FHR patterns in almost every series reported for this type of analgesia. There are few studies in which FHR changes have been absent (Thalme, Belfrage and Raabe, 1974). Usually, abnormal FHR patterns occur in less than 15% of fetuses (Printz and McMaster, 1972; Maltau, 1975; Jouppila et al., 1977), but on occasion between 20 and 50% (Schifrin, 1972), increasing to 77% (Zilianti et al., 1970). The main changes in FHR have been late deceleration or prolonged fetal bradycardia (Printz and McMaster, 1972; Schifrin, 1972; Maltau, 1975; Jouppila et al., 1977). There are reports of a loss of beat-to-beat variation (Boehm, Woodruff and Growdon, 1975; Raabe and Belfrage, 1976).

Both maternal hypotension (Brotanek et al., 1973; Maltau, 1975; Jouppila et al., 1977) and infusion of oxytocin during EA (Schifrin, 1972) have been shown to be associated with changes in FHR. The supine position has also been considered harmful to the fetus, especially during extradural block (Crawford, Burton and Davies, 1972). Many studies have described this for Caesarean section under general anaesthesia (Ansari et al., 1970; Crawford, Burton and Davies, 1972; Downing et al., 1974), but there is still little direct evidence of a harmful effect of the maternal supine position on the fetus during extradural block for vaginal delivery.

METHODS

Patients

We studied 79 healthy normotensive parturients with a single fetus in vertex presentation without signs of fetal distress. Sixty-eight mothers were primiparae and 11 multiparae. The maternal age ranged from 17 to 39 yr, the duration of gestation from 260 to 295 days and the cervical dilatation at induction of analgesia from 2 to 8 cm. All mothers were in the active phase of the first stage of labour (table I).

| TABLE I. Clinical data of patients in the supine and lateral position receiving lumbar extradural analgesia (EA) for labour with vaginal delivery |
|-----------------|-----------------|-----------------|-----------------|-----------------|
|                 | Supine (n = 39) | Mean ± SD       | Lateral (n = 40) | Mean ± SD       |
| Age (yr)        | 26 ± 4          | 25 ± 3          |
| Gestation (days)| 282 ± 9         | 280 ± 9         |
| Cervical dilatation (cm) | 4.8 ± 1.1     | 4.8 ± 1.3     |
| Maternal arterial pressure before EA (mm Hg) | | |
| Systolic        | 128 ± 13        | 122 ± 11        |
| Diastolic       | 86 ± 9          | 82 ± 13         |

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Analgesia

Using a Portex catheter inserted in the extradural space at the L1-2 or L2-3 interspace, etidocaine (Duranest) 50 or 100 mg without adrenaline was administered in volumes of 5, 10 or 30 ml. The concentrations of etidocaine solutions ranged from 0.17 to 1%. The maternal position (supine or lateral) for induction of EA was selected at random (table II).

<table>
<thead>
<tr>
<th>Material position during EA</th>
<th>Amount of etidocaine without adrenaline (mg)</th>
<th>No. of parturients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Supine</td>
<td>100</td>
<td>12</td>
</tr>
<tr>
<td>Lateral</td>
<td>100</td>
<td>12</td>
</tr>
<tr>
<td>Supine</td>
<td>50</td>
<td>27</td>
</tr>
<tr>
<td>Lateral</td>
<td>50</td>
<td>28</td>
</tr>
</tbody>
</table>

Initially a 2.5-ml test dose was given and the remainder was administered over the succeeding 5-10 min. In the lateral group half the dose was given with the mother in one lateral position and the remainder with her in the contralateral position. These mothers remained in the lateral position during the entire period of study. The other patients remained supine before and during extradural block and for the whole period of EA, provided no maternal or fetal complication occurred (table II). If abnormal FHR changes were observed, the mother was turned immediately to a lateral position.

Fetal and maternal monitoring

FHR was monitored continuously using a scalp electrode, and uterine contractions were monitored with either an internal or an external tocrometer (cardiotocographs: Hewlett-Packard, model 8020 A with transducer adapter 15137 or Corometrics Fetal Monitor, model III). FHR patterns were evaluated visually, decelerations according to Hon and Quilligan (1967), short-term base line variability (beat-to-beat) according to Hammacher (1969). In addition, serial fetal scalp capillary blood samples were obtained for pH and acid-base balance determinations (data published separately). Artefacts caused by fetal blood sampling on FHR were not taken into account.

We considered the FHR to be abnormal when there was late deceleration during two or more successive uterine contractions, bradycardia over two uterine contractions or over 5 min, and a loss of beat-to-beat variability under 5 beat min\(^{-1}\) for 10 min or longer (silent type of FHR base line variability).

Maternal arterial pressure (AP) was recorded by auscultation with an ordinary sphygmomanometer from the arm (upper arm in the lateral parturients) between uterine contractions at 5-10-min intervals.

The study commenced with a 30-60-min control period before the induction of EA. During this time the mother was in the same position as for the extradural injection. Oxytocin infusion was not given from 30 min before to 30 min after the induction. EA was given only if the FHR pattern was normal and maternal AP stable during the control period. Thus each parturient served as her own control before EA. If the mother’s AP during EA decreased by more than 20 mm Hg, saline was infused i.v. at a rapid rate (1000 ml/15 min). No vasopressor was used.

Fisher’s exact test between two frequencies and Student’s unpaired t test between two means were used for statistical analyses.

RESULTS

Fetal heart rate (FHR)

FHR was normal in 53 of the 79 patients during EA (table III). Transient pathological FHR was observed in the remaining 26 patients, 22 of whom were supine and four were in the lateral position (table III). Typical examples are shown in figures 1, 2, 3 and 4.

<table>
<thead>
<tr>
<th>FHR changes</th>
<th>Frequency of FHR changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Supine (n = 39) Lateral (n = 40)</td>
</tr>
<tr>
<td>No changes</td>
<td>15 (38.5%) 34 (85.0%)</td>
</tr>
<tr>
<td>Early decelerations</td>
<td>2 (5.1%) 2 (5.0%)</td>
</tr>
<tr>
<td>Abnormal</td>
<td>21 (53.9%) 4 (10.0%)</td>
</tr>
<tr>
<td>Late decelerations</td>
<td>Silent type of FHR base line variability</td>
</tr>
<tr>
<td>1 (2.6%) 0</td>
<td></td>
</tr>
</tbody>
</table>

These abnormal FHR changes commenced 8–23 min after the onset of EA and usually lasted 10–25 min, but occasionally longer (fig. 4). The frequency in the supine group (56%) was significantly different from that in the lateral group (10%) (P<0.001).

The short-term variability of FHR, as judged by eye, remained unchanged in the 53 fetuses with
Fig. 1. Abnormal fetal heart rate (late deceleration type fetal bradycardia) associated with maternal supine position and unchanged maternal arterial pressure during lumbar extradural block induced by etidocaine 50 mg without adrenaline. (22-yr-old, para 1, 280th day of gestation, cervical dilatation 4 cm.) Bradycardia began 14 min after induction, associated with an increased beat-to-beat variability (saltatory type). It was followed at 25–48 min by a decrease in the short term variability. The fetal scalp capillary pH decreased from 7.41 to 7.32 unit in 25 min.

Fig. 2. Abnormal fetal heart rate (late decelerations) associated with supine position and maternal hypotension during lumbar extradural block induced by etidocaine 100 mg without adrenaline. (26-yr-old, para 1, 285th day of gestation, cervical dilatation 6 cm.) The late decelerations at 15 and 26 min after the block coincided with smallest maternal systolic pressures. The fetal scalp capillary blood pH decreased from 7.29 before the block to 7.20 unit 43 min after the induction.
FIG. 3. Abnormal fetal heart rate (late deceleration type of bradycardia) associated with lateral recumbent position and unchanged arterial pressure during lumbar extradural block induced by etidocaine 50 mg without adrenaline. (19-yr-old para 1, 271st day of gestation, cervical dilatation 5 cm.) Bradycardia occurred 14 min after the induction, associated with an increased beat-to-beat variability. There are three consecutive late deceleration waves between 26 and 34 min. The fetal scalp capillary pH remained unchanged.

FIG. 4. Abnormal fetal heart rate (late decelerations) associated with maternal lateral recumbent position and maternal hypotension during lumbar extradural block induced by etidocaine 50 mg without adrenaline. (21-yr-old para 1, 266th day of gestation, cervical dilatation 4 cm.) Late decelerations beginning 16 min after the induction are visible over a period of 48 min. At 26–31 min they are associated with an increase and later at 32–48 min with a decrease in beat-to-beat variability.
normal FHR during EA. Eight of 26 fetuses with an abnormal heart rate during EA had a normal beat-to-beat variation, one had a silent pattern and the remaining 17 exhibited a saltatory pattern transiently (beat-to-beat variation more than 25 beat min\(^{-1}\)) alternating with normal short-term variability (figs 1, 3 and 4).

**Maternal arterial pressure (AP)**

The mean systolic AP before EA was similar in both groups (table II). The mean maximal decrease (± SD) in maternal AP during EA was 17±17 mm Hg in the supine and 17±16 mm Hg in the lateral group. In mothers with a normal FHR during EA the mean decrease in systolic AP was 18±15 mm Hg in the supine group and 16±17 mm Hg in the lateral. In mothers with an abnormal FHR during EA, the mean maximal decrease in systolic AP was not significantly different between the groups (18±20 mm Hg supine, 28±20 mm Hg lateral).

A decrease in systolic AP of more than 30% was observed in nine of the 79 patients (11.4%), five being lateral and four supine. The association of the decrease in maternal AP with the frequency of abnormal FHR changes is shown in table IV. When the systolic AP decreased more than 30% there were three fetuses of five with an abnormal heart rate in the lateral group in comparison with four of four in the supine group. The frequency of abnormal FHR during EA with etidocaine 50 mg was 12 of 27 (44.4%) compared with nine of 12 (75%) with etidocaine 100 mg in the group of supine mothers. In the lateral group the frequency of abnormal FHR was two of 28 fetuses (7.1%) with etidocaine 50 mg and two of 12 (16.7%) with 100 mg.

The maximal decrease in systolic AP during EA with etidocaine 50 mg (10±12 mm Hg) differed significantly from the corresponding decrease with 100 mg (21±17 mm Hg) (P<0.005).

**DISCUSSION**

Some authors describe EA as a safe technique for maternal analgesia (Zador and Nilsson, 1974; Raabe and Belfrage, 1976; Jouppila et al., 1977), although they noted the same types of abnormal FHR change during the block as occurred in the present study. These FHR changes probably indicated fetal asphyxia (Beard et al., 1971), but they were transient. Although the long-term effects of these FHR changes are unknown, it is obviously desirable that the frequency of pathological FHR changes should be as small as possible after EA.

In the present study the unfavourable fetal effects of the maternal supine position during obstetric EA were clearly demonstrated. Pathological FHR patterns were much more common in the supine position (56%) compared with the lateral recumbent position (10%). In three of the four fetuses developing a pathological FHR with the mother lateral, there was a marked decrease in maternal AP (table IV). A reduction in uterine placental blood flow (UPBF) as a result of hypotension was the probable cause of these FHR changes. Although the frequency of pathological FHR patterns during EA with etidocaine 100 mg was greater in both the supine and lateral group than with etidocaine 50 mg, it is likely that the FHR changes resulted from hypotension and not from a direct effect of the local anaesthetic on the fetus as hypertension was greater with 100 mg than with 50 mg of etidocaine. Infusion of a plasma expander immediately before the induction of EA has been shown to prevent the subsequent development of both maternal hypotension and a decrease in UPBF (Huovinen et al., 1977).

Vasoactive drugs should be used with great caution as they may diminish UPBF (Ralston, Shnider and de Lorimier, 1974), and this could harm the fetus further.

Human UPBF decreases synchronously with uterine contractions during EA (Brotanek et al., 1973). This may explain the observation that FHR changes during EA in the present study were mainly late decelerations (table IV) (figs 1, 2 and 4). Uterine hypertonus resulting from EA may also have been responsible for these pathological FHR changes, although hypertonus was not obvious.

Maternal hypotension during obstetric EA has been shown to increase the frequency of pathological FHR changes (Schifrin, 1972; Wingate et al., 1974). This was found in the present study (table IV), although the number of mothers with marked hypotension was small. The fetuses of supine mothers
undergoing Caesarean section under general anaesthesia were considered at risk, even in those with minimal changes in maternal AP (Crawford, Burton and Davies, 1972). We confirmed this observation. Thus, when the decrease in AP was small (30% or less) abnormal FHR patterns occurred in 51% of the supine group while in the lateral group there was only one instance (fig. 3) (table IV). This emphasizes the fact that normal AP does not guarantee normal UPBF.

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REFERENCES


EFFET DE LA POSITION MATERNELLE SUR LA FREQUENCE CARDIAQUE DU FOETUS PENDANT UNE ANALGESIE EXTRADURALE

RESUME

On a étudié à l'aide d'une cardiotocographie en continu l'effet de la position maternelle sur la fréquence cardiaque du foetus (FHR) pendant une analgésie extradurale (EA) administrée à l'aide de soit 50 soit 100 mg d'étidocaine sans adrénaline, pour le travail et l'accouchement par voie vaginale de 79 parturientes dont 39 étaient couchées et 40 dans la position latérale. La FHR était normale chez toutes les patientes dans les deux positions. L'ETE a précédé l'EAD dans 73% des cas. On a constaté sur 26 patientes des signes anormaux transitoires, 22 d'entre elles (85%) ayant une HT comme facteur de risque. La FHR était normale pendant les épisodes d'HT. La perfusion utérine a été mesurée par l'anaesthesie extradurale et l'oxytocine dans 119 patientes pendant la période de travail. On a observé une diminution de la perfusion utérine significative (P < 0,001) chez les patientes couchées, mais aucune différence significative entre les deux groupes. La fréquence des FHR anormales associées à une diminution, modérée ou modérée, de l'AP (30% ou plus) a été plus importante dans la position couchée (31%) que dans la position latérale (3%).
ZUSAMMENFASSUNG

Mittels kontinuierlicher Herz-Tokographie wurde die Wirkung der mütterlichen Position auf die fötale Herztätigkeit (FHR) während extraduraler Analgesie (EA) mit entweder 50 oder 100 mg Etidocain ohne Adrenalin für Wehen und vaginale Geburt studiert. Von 79 gesunden Gebärenden waren 39 in Rückenlage und 40 in lateraler Stellung. Bei allen Patientinnen war FHR während der Kontrollperiode vor EA normal, und blieb normal während EA bei 53 Patientinnen. Völlig normale Erscheinungen ergaben sich bei 26 Patientinnen—22 davon (56%) in Rückenlage und 4 (10%) in lateraler Stellung, also eine wesentliche Differenz ($P < 0.001$). Das Absinken des arteriellen Druckes (AP) unterschied sich in den einzelnen Gruppen nicht, doch war die Häufigkeit von abnormem FHR in Verbindung mit einem kleinen oder mässigen Anstieg von AP (30% oder weniger) höher in der Rückenlage (51%) als in der lateralen Gruppe (3%).

SUMARIO

Se estudió el efecto de la posición maternal en el ritmo cardíaco del feto (RCF), mediante cardiotocografía continua, durante analgesia extradural (AE), con 50 ó 100 mg de etidocaina sin adrenalina, para parto y puerperio vaginal. De las 79 parturientes en buena salud, 39 estaban en supino y 40 en posición lateral. Durante el periodo de control antes de la AE, todas las pacientes tenían un RCF normal, el que se mantuvo normal durante la AE en 53 pacientes. Ocurrieron comportamientos anormales transitorios en 26 pacientes, 22 (56%) del grupo en supino y 4 (10%) en el grupo lateral, lo que constituye una diferencia significativa ($P < 0.001$). La disminución de la presión arterial (PA) no difirió entre los grupos, pero la frecuencia del RCF anormal asociado con una reducción moderada o baja de la PA (30% o menos) era mayor en el grupo en supino (51%) que en el grupo lateral (3%).

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