CHANGES IN FETAL $\text{tcPO}_2$ VALUES OCCURRING DURING LABOUR IN ASSOCIATION WITH LUMBAR EXTRADURAL ANALGESIA

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Summary

Forty-six of 64 high risk labours were managed with continuous lumbar extradural analgesia. Fetal heart rate (FHR) and continuous transcutaneous $P_O_2$ ($\text{tcPO}_2$) measurements were made in the 64 patients. Abnormal fetal heart rate patterns and low $\text{tcPO}_2$ values associated with the onset of the extradural block were noted in 9% of these cases. A decrease in maternal arterial pressure and uterine hypertonus appeared to be responsible, singly or in combination, for the changes. These effects and the changes in FHR were not seen in the 18 mothers not receiving extradural analgesia. The supine position was associated with slightly smaller fetal $\text{tcPO}_2$ values than the preferred lateral positions, with a significant worsening of the fetal $\text{tcPO}_2$ values after induction of the extradural block although, overall, extradural analgesia neither improved nor impaired the fetal $\text{tcPO}_2$.

Continuous lumbar extradural analgesia has been reported to improve (Pearson and Davies, 1974) and to impair (Schifrin, 1972) fetal acid-base status in the first stage of labour when compared with infants of mothers not receiving extradural blockade. The circumstances which influence the fetus are changes in uteroplacental perfusion (Schifrin, 1972; McDonald, Bjorkman and Reed, 1974; Pearson and Davies, 1974; Jouppila et al., 1977) and the degree to which the acid-base state of the mother is altered. Changes in fetal heart rate (FHR) have occurred with the use of local analgesic agents and it was our intention to determine whether these changes were associated with alterations in the degree of fetal oxygenation.

Patients and methods

Sixty-four patients in spontaneous labour were studied, 46 of whom received continuous lumbar extradural analgesia. All mothers and their fetuses were monitored using the transcutaneous $P_O_2$ technique. Twelve patients did not receive extradural analgesia because they were assessed initially as having FHR patterns suggestive of moderate fetal hypoxaemia. These patients were observed carefully and were found, on subsequent reassessment, to be comparable to the group initially selected for extradural analgesia. These patients were used as controls in addition to a further six patients randomly allocated to the non-extradural group since their initial assessment did not contraindicate the use of this form of analgesia. Thus 18 patients, comparable clinically to the 46 treated patients, were used for comparison. In addition, comparisons were made within the extradural group, before and after the administration of the analgesic agent.

The mothers had been classified as "high-risk" because of evidence of pre-eclampsia, hypertension or diabetes, with some fetuses having substantial intrauterine growth retardation, abnormal antepartum fetal heart rate (FHR) tests, or being pre- or post-term. Twenty-eight fetuses of the 46 mothers selected for extradural analgesia had FHR abnormalities. Eighteen fetuses had mild to moderate variable decelerations, 10 had loss of beat-to-beat variability alone or in combination with occasional decelerations. All fetuses were in the cephalic presentation. The gestational ages as derived from the last menstrual period or by serial measurements of biparietal diameter were 36–42 weeks. Continuous lumbar extradural analgesia was commenced in the active phase of labour, with cervical dilatation of between 4 and 8 cm. Maternal arterial pressure (AP), intrauterine pressure (IUP), FHR and maternal and fetal $\text{tcPO}_2$ were measured.

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The patients were preloaded with lactated Ringer's solution 500-1000 ml and placed in the left lateral position. Using a mid-line approach and loss of resistance with a 10-ml syringe containing air to identify the extradural space, a test dose of 1.5% lignocaine 1-2 ml was given. Bupivacaine 0.5% without adrenaline was then injected to the extradural space, the definitive dose depending on the weight, height and age of the patient, satisfactory pain relief being the end-point. The patients were then placed supine for 8-10 min, after which they were transferred to the left lateral position.

After injection of the analgesic agent, measurements of systolic and diastolic arterial pressures were obtained using a mercury sphygmomanometer every 2 min for 20 min and then every 5 min for 10 min. The sensory level affected by the extradural block was noted.

The transcutaneous PO$_2$ (tcPO$_2$) electrode was applied to the fetal head as described by Huch and colleagues (1977). The tcPO$_2$ of the mother and fetus were recorded on a Litton Oxymonitor (Litton Medical Electronics, Elk Grove, Illinois). The FHR and uterine contractions (UC) were recorded on either a Corometrics 101B (Corometrics, Wallingford, Connecticut) or a Sonicaid FM3R (Sonicaid, Frederickburg, Virginia) fetal monitor. All data were transferred to a Hewlett-Packard multichannel recorder HP7758A (Hewlett-Packard, Waltham, Massachusetts). Blood-gas tensions from umbilical cord blood were measured on a Corning 165 analyser (Corning, Medfield, Massachusetts).

The definitions of the FHR patterns follow the criteria of Hon and Quilligan (1967). The normal range for the fetal tcPO$_2$ is yet to be established, but published data (Huch et al., 1977; Willcourt et al., 1981) indicate that a range of 2–3.3 kPa is associated with fetal well being.

**RESULTS**

One hundred and sixty-six hours of fetal tcPO$_2$ measurements were obtained from the 64 labours; 53 of these from labours in which extradural analgesia was not used. Non-recurring late decelerations with exaggerated FHR variability occurred five times in four fetuses during the 113 h of extradural analgesia, but were not seen in the 53 h without the extradural blockade. Two of the four fetuses were in the group that did not demonstrate any FHR abnormalities before blockade, one fetus had diminished FHR variability and the fourth fetus had demonstrated occasional mild variable decelerations before induction of the extradural block. Following the sudden severe decelerations that were temporally related to the onset of the extradural block, each fetus demonstrated a return to the previous FHR pattern.

Although the supine position generally decreased fetal tcPO$_2$ by 0.27 kPa, the decrease was as great as 0.8 kPa in three fetuses, but excluding the four instances reported above, these changes were not associated with fetal distress. All other fetal tcPO$_2$ values were essentially unaltered by extradural analgesia. The two lateral positions were compared before and after the onset of the block and were found to give comparable fetal tcPO$_2$ values. In 18 of the 46 mothers, the right lateral position gave greater tcPO$_2$ values than the left before the block, with changes of 0.27–0.4 kPa after blockade. The left lateral position was associated with greater fetal tcPO$_2$ values than the right in 28 patients before and after the extradural block (table I). The better of two lateral positions is referred to as the "preferred" lateral position.

In the first patient with abnormal changes, a decrease in the fetal tcPO$_2$ from 2.13 to 1.46 kPa occurred after the mother had been placed in the supine position (fig. 1). A further decrease occurred 8 min after the administration of the extradural analgesic. The uterine contractions as measured by an internal pressure catheter became more frequent and, although less intense than before the blockade, caused the uterine pressure to remain between 2.7 and 4 kPa for 5 min (normal 0.67–2.7 kPa). The FHR deceleration was accompanied by a decrease in tcPO$_2$ which began to recover after the uterine contractions had ceased. However, a sustained pattern of contraction recurred, and decreases in FHR and tcPO$_2$ were noted. The maternal arterial pressure (AP) decreased from 122/80 mm Hg to

| Table I Effects of position and continuous lumbar extradural analgesia on fetal tcPO$_2$ (kPa). The lateral position in this table refers to that side which gave the greater fetal tcPO$_2$ values. n = number of observations made in each position after allowing 10 min for stabilization. Statistical analysis was performed using a two-tailed t-test: Supine v. lateral = P < 0.1 (n.s.); lateral v. lateral extradural = P < 0.01 (n.s.); supine extradural v. lateral extradural = P < 0.001 |
|---------------------|-----------------|------------------|-----------------|------------------|
|                     | Supine v. lateral | Supine extradural | Lateral extradural |
| Range               | 1.87–2.38 (n = 19) | 1.87–3.73 (n = 19) | 1.06–2.4 (n = 20) | 2–4.0 (n = 20) |
| Mean ± SD           | 2.4 ± 0.2       | 2.5 ± 0.4        | 1.6 ± 0.28      | 2.67 ± 0.45     |
Fig 1  First patient. Records of maternal transcutaneous $P_{O_2}$ (Mat. tcPO$_2$), fetal transcutaneous $P_{O_2}$ (Fet. tcPO$_2$), fetal heart rate (FHR) and uterine contractions (UC). Injection of analgesic 8 min before onset of fetal bradycardia.

$90/55$ mm Hg during the first deceleration. Oxygen was administered to the mother, she was placed in the right lateral position and the rate of fluid infusion increased, whereupon improvement was seen in FHR and tcPO$_2$. The fetus was delivered 1 h later with an umbilical artery (UA) pH of 7.24 unit and Apgar scores of 8 at 1 min and 9 at 5 min. Gestational age was assessed as being 39 weeks.

In the second patient (fig. 2), following administration of the definitive dose of analgesic, exaggerated FHR variability was observed and was followed by a late deceleration occurring in association with a decrease in fetal tcPO$_2$. The intrauterine pressure tracing showed a “coupling” pattern with a low resting pressure. Oxygen was administered to the mother before the deceleration in fetal heart rate. There was no significant change in the maternal arterial pressure at any time during the FHR deceleration (AP 110/76–115/75 mm Hg). Umbilical artery pH was 7.26 unit and Apgar scores were 7 at 1 min and 9 at 5 min. The fetus was assessed as being 41 weeks.

In the example shown in figure 3, exaggerated FHR variability occurred with a decrease in the fetal tcPO$_2$ 13 min after the regional block. The intrauterine pressure increased and remained increased for 8 min during which further changes in FHR and fetal tcPO$_2$ were seen. The maternal AP changed slightly from 130/84 to 120/78 mm Hg.

The position of the mother was changed from the right to the left lateral position and the rate of infusion increased. This mother was already breathing oxygen, but because of the poor application of the mask the maternal tcPO$_2$ values were not as high as expected. Following a further injection of $0.5\%$ bupivacaine into the extradural space, the same sequence of events occurred. Thirty minutes later the baby was delivered. The UA pH was 7.16 unit and the Apgar scores were 6 at 1 min and 8 at 5 min. Compression of the fetal tcPO$_2$ electrode resulted in erroneously low tcPO$_2$ values at delivery. Gestational age was 41 weeks by dates.

The changes seen in the fourth patient (fig. 4) are similar to those already described, but the fetal tcPO$_2$ changes were not as marked. The uterine contractions were, however, recorded by an external tocodynamometer so that changes in intrauterine pressure could not be appreciated or quantitated.

Exaggerated FHR variability was seen with a decrease in fetal tcPO$_2$. The maternal arterial pressure decreased from $114/72$ to $98/58$ mm Hg during the period of abnormal fetal recording. This 39-
FIG. 2. Second patient: Legends as in figure 1. Analgesic administered 10 min before changes in fetal heart rate.

FIG. 3. Third patient: Legends as in figure 1.
week fetus had a UA pH of 7.21 unit and the Apgar scores were 8 at 1 min and 8 at 5 min.

In all instances the mothers were in the supine position when the FHR abnormalities occurred.

DISCUSSION
Continuous lumbar extradural analgesia is a preferred method of pain relief in many obstetric centres. The hazards of the technique for the fetus are well recognized and relate primarily to changes in the utero-placental circulation, to toxic effects of the drugs on the fetus (Schifrin, 1972; Pearson and Davies, 1974; McDonald, Bjorkman and Reed, 1974) and, secondarily, to malpresentations of the fetus (Hoult, MacLennan and Carrie, 1977).

A decrease in blood flow in the utero-placental circulation occurs by redistribution of the maternal intravascular compartment with decreases in venous return and vasoconstriction above the block, or by an increase in intrauterine tone, or both (McDonald, Bjorkman and Reed, 1974; Malthau, 1975). It has been postulated that when the block is below T10, this effect is decreased (Abouleish, 1976). Maintenance of a supine position further aggravates the already compromised uteroplacental circulation, increasing the chances of fetal distress.

These observations of 46 labours managed under extradural blockade show five instances in four fetuses of fetal bradycardia with changes in fetal tcPO₂ and in the pattern of uterine contraction, temporally related to the administration of extradural analgesia. While 28 of the 46 patients who received extradural analgesia had fetuses with abnormal FHR patterns before blockade, this particular type of deceleration was seen only in close relationship to the administration of the extradural analgesia. A decrease in the fetal tcPO₂ accompanied the changes in FHR with a return to the previous tcPO₂ values once the episode had passed. Clearly, avoidance of the supine position is essential if the frequency of fetal hypoxaemia is to be decreased. Alternately placing the mother in the right or left lateral position while waiting for the block to become established would seem preferable to keeping the mother supine. Otherwise, the effects of the extradural analgesia were neither to improve nor impair the fetal tcPO₂ values provided the mothers were in the preferred lateral position. The supine position was always associated with a lower value than the lateral positions with the effect more pronounced after institution of the block.

The frequency of fetal bradycardia and lower fetal tcPO₂ values in our study was 9% (four out of 46). Malthau (1975) in reviewing other studies cited a frequency of fetal distress of between 10 and 77%, while he had a 6% frequency in his own study.