Epidural Anaesthesia as an Anticonvulsant in the Management of Hypertensive and Eclamptic Patients in Labour

D. A. MERRELL, M. A. T. KOCH

SUMMARY

Lumbar epidural anaesthesia was used as an anticonvulsant in a series of 1106 patients with hypertension in labour, including 7 patients with eclampsia. Six of the 1074 patients in whom epidural analgesia was successful subsequently had convulsions. Of these 4 had convulsions after delivery, which took place at least 21 hours after the last dose of local anaesthetic, and the 2 others within 5 minutes of receiving the first dose of bupivacaine. We conclude that before it wears off epidural anaesthesia is an effective way of preventing eclampsia and that other anticonvulsant therapy is not required, although the latter may be necessary before the procedure. The possible mechanism of the anticonvulsant action is discussed.


One of the major problems facing the obstetrician in the management of hypertensive patients in labour is that of identifying those at risk of developing eclampsia and therefore requiring anticonvulsant therapy. The distinction between pre-eclampsia, especially when it is superimposed on pre-existing hypertension, and other causes of hypertension in pregnancy may be difficult (if not impossible) without renal biopsy, particularly if the patient is first seen late in pregnancy. Even when pre-eclampsia is suspected it may prove impossible to predict which patients will have convulsions. The selection of patients for anticonvulsant therapy is therefore based on the clinical diagnosis of pre-eclampsia and the estimation of the likelihood of convulsions occurring, neither of which can always be accurate.

We think that it is safer to assume that all women with hypertension in labour are liable to develop eclampsia and that all should be provided with anticonvulsant cover. There is a reluctance to do this routinely because it will be unnecessary in the majority of cases and the side-effects of the drugs — particularly fetal sedation — aggravate the intra-uterine asphyxia sometimes associated with hypertension in pregnancy and are compounded by the use of narcotic analgesics.

PATIENTS AND METHODS

All patients with a blood pressure of 140/90 mmHg or more were classified as hypertensive. In a 20-month period, 2 399 patients received epidural anaesthesia during labour. Of these 1 106 (46%) were considered hypertensive and are included in this series (Table I). Seven of these patients had one or more fits.

The causes of hypertension in the patients in this series were varied, and although a pre-eclamptic element was thought more likely in some than in others, no attempt has been made to separate them for reasons outlined above. Some had been in the antenatal wards before labour, where hydralazine and methyldopa were given if the blood pressure was persistently above 170/110 mmHg. Anticonvulsants were not routinely used before the epidural anaesthesia except in eclamptic patients and in the 73 (6.6%) whose knee jerks were considered to be brisk. Patients received an intravenous injection of diazepam 10 mg to stop convulsions; the other eclamptics and the hyper-reflexic patients were given intramuscular magnesium sulphate 4 g. After administration of the epidural anaesthetic anticonvulsants were not used again except: (i) in 32 (2.9%) patients in whom epidural anaesthesia failed; (ii) in the event of a subsequent convolution; and (iii) after delivery.

Local anaesthetic agents were injected via an indwelling epidural catheter. The epidural space was entered via the lumbar spine, the lumbar vertebral spaces 2:3 and 3:4.
TABLE I. CLASSIFICATION OF THE PATIENTS

<table>
<thead>
<tr>
<th>Age and parity</th>
<th>Hypertension</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mild (≥140/90 mmHg)</td>
<td>Moderate (≥150/100 mmHg)</td>
</tr>
<tr>
<td>Nullipara &lt;20</td>
<td>144</td>
<td>168</td>
</tr>
<tr>
<td>Nullipara ≥20</td>
<td>138</td>
<td>131</td>
</tr>
<tr>
<td>Multipara any age</td>
<td>106</td>
<td>160</td>
</tr>
<tr>
<td>Total</td>
<td>388 (35%)</td>
<td>459 (41.5%)</td>
</tr>
</tbody>
</table>

TABLE II. CLASSIFICATION OF PATIENTS WITH ECLAMPTIC CONVULSIONS

The convulsion took place:

<table>
<thead>
<tr>
<th>Group</th>
<th>No. (incidence)</th>
<th>Before the epidural</th>
<th>After the epidural</th>
<th>After the epidural had worn off</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nullipara &lt;20</td>
<td>6 (1.5%)</td>
<td>3</td>
<td>-</td>
<td>3*</td>
</tr>
<tr>
<td>Nullipara ≥20</td>
<td>5 (1.5%)</td>
<td>4</td>
<td>-</td>
<td>1†</td>
</tr>
<tr>
<td>Multipara any age</td>
<td>2 (0.5%)</td>
<td>0</td>
<td>2‡</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>13 (1.2%)</td>
<td>7§</td>
<td>2</td>
<td>4</td>
</tr>
</tbody>
</table>

* The convulsion took place after caesarean section under general anaesthesia in 1, 2½ hours after delivery in 1, and 4 hours after top-up and 3 minutes after delivery in 1.
† The convulsion took place 60 hours after delivery.
‡ The convulsion took place 5 minutes after administration of the epidural anaesthetic in 1 and 3 minutes after in the other.
§ Epidural anaesthesia was unsuccessful in 3 of these patients, all of whom subsequently had convulsions.

being preferred. Because of obesity and oedema in this region in some of the patients higher approaches occasionally had to be used, the highest space being that between the 12th thoracic and 1st lumbar vertebral spines. After a test dose of 2 ml 2% xylocaine (Xylocard), an initial dose of 7 ml 0.5% bupivacaine (Marcaine) was injected. If control of the blood pressure or relief of the pain was inadequate, 2 ml increments of 0.5% bupivacaine were injected up to a maximum total volume of 15 ml. Top-up doses of 7 ml bupivacaine 0.5% were given when the hypertension recurred or pain returned, and the volume of bupivacaine was increased before delivery if the previous doses had been found not to anaesthetize the perineum.

RESULTS

Eclampsia (Table II)

There were 13 eclamptics in this series, 7 of whom had had convulsions before administration of the epidural anaesthetic. In 3 of these 7, attempts to place the epidural catheter were unsuccessful, and in all 3 convulsions recurred later in labour. The 4 who were successfully anaesthetized did not have any further fits. Two of the multiparous patients had fits, one 3 and the other 5 minutes after the first injection of bupivacaine. All the other patients who had convulsions did so after delivery and at least 2½ hours after their last top-up dose of anaesthetic.

Perinatal Mortality

The 1 074 patients gave birth to 1 108 infants, of which 49 died in the perinatal period — a perinatal mortality rate of 44.2 per 1 000 total births. Of the 49 infants 23 (46.9%) were already dead at the time of the epidural, 16 (32.7%) died because of obstetric or paediatric problems, and a set of twins died in utero following the spinal anaesthetic mentioned below. This left 8 perinatal deaths for which no obvious cause could be found. Two perinatal deaths were definitely and 8 others possibly related to the epidural anaesthesia, a procedure-related perinatal mortality rate of 9 per 1 000 total births.

Major Maternal Problems

Rupture of the uterus under epidural anaesthesia occurred in 3 patients; 1 of these required a second laparotomy on the 2nd postoperative day for a broad-ligament haematoma, during which she died. One patient had a respiratory arrest following an inadvertent spinal anaesthetic and recovered fully after three-quarters of an hour of assisted ventilation.

DISCUSSION

We interpret our results as showing that anticonvulsant cover is provided while the epidural anaesthetic is effective, and the procedure continues to be used routinely as the treatment of choice in our department. Epidural
anaesthesia offers a simple and safe means of providing the hypertensive patient potentially at risk with both anticonvulant cover (without the risk of fetal sedation) and adequate analgesia for the labour and for elective assisted delivery.

Four patients had convulsions after the epidural anaesthesia had worn off, one of them within minutes of removal of the endotracheal tube after general anaesthesia for a caesarean section. Another had convulsions 60 hours after delivery; although this is outside the accepted time limit of puerperal eclampsia, the case has been included as such in this series. The remaining 2 patients both had convulsions after delivery, 1 after 2½ and the other after 4 hours. No patient in whom epidural anaesthesia was successful had convulsions while the blockade lasted. This is similar to the findings of Willocks and Moir, although they recommend in a later paper that chlorpromazine should be used in conjunction with the epidural anaesthesia.

The possible mechanism of the anticonvulsant action of epidural anaesthesia is obscure, but may involve one or more of the following: (i) reduction of the blood pressure, although this has not previously been considered adequate; (ii) reduction of the sensory input to the brain from the blocked area and the decrease of fear and anxiety produced by the pain of labour, vaginal examinations and manipulations, etc.; (iii) local anaesthetic depression of the central nervous system, although this may be preceded by an excitatory phase which might stimulate a convulsion; and (iv) an increase in the splanchnic blood flow. If vasospasm is a feature of pre-eclamptic toxemia, under-perfusion of some organs would be expected; it is possible that in its under-perfused state one of these organs might produce the 'toxin' that perpetuates the pre-eclamptic disease process. This might be the uterus, as suggested by Page, but might equally well be the liver, the gut or the kidney. The vasospasm might be responsive to the vasodilatation that follows the sympathetic blockade of epidural anaesthesia, perhaps enhancing perfusion of that organ and reducing the release of the 'toxin'.

It is known that denervation of the kidney increases the excretion of sodium and water and that epidural anaesthesia prevents the retention of sodium occurring after upper abdominal surgery. Increased sodium excretion might be advantageous in pre-eclampsia, where the ion is retained in excess. Evidence for such an action in pre-eclampsia comes from reports of increased urine output and diminution of oedema following epidural anaesthesia, although this might well have been due to the associated bed rest. In their study of 20 patients with gestational hypertension James and Davies were unable to demonstrate any change in the glomerular filtration rate.

The problems experienced in our series were: (i) the hypertension sometimes recurred before the return of pain, and blood pressure should be monitored as a guide for repeat doses of local anaesthetic; (ii) there was a high incidence of occipitotransverse and posterior positions late in the second stage of labour, although the anaesthesia facilitated rotation of the fetus with Kjelland's forceps; (iii) owing to the restlessness of some of the eclamptic patients successful placement of the epidural catheter was not always possible; (iv) 2 patients had convulsions almost immediately after the injection of bupivacaine — these were indistinguishable from the toxic convulsions seen in some normotensive patients, but have been included here as eclampsia. This possibly indicates that in some patients fits may be stimulated by the injection of fluids into the epidural space, or that sufficient local anaesthetic may be absorbed and produce central nervous system stimulation.

We consider the procedure-related perinatal mortality rate of 9 per 1 000 total births relatively low for such a high-risk group; continuous fetal monitoring by cardiotocography might have reduced it still further. A more detailed analysis of the perinatal mortality and neonatal problems is to be reported separately.

**CONCLUSION**

We are able to confirm that in the management of patients with pre-eclampsia and eclampsia anticonvulsant therapy is not required after the establishment of functional lumbar epidural anaesthesia, but that it may be necessary both before the procedure in certain patients and after delivery in all. The regimen appears to be associated with acceptably low procedure-related perinatal mortality.

**REFERENCES**