HAEMODYNAMIC EFFECTS OF POSITION CHosen FOR INSERTION OF EPIDURAL CATHETER
A comparison of the lateral and sitting position

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The ideal haemodynamic position for insertion of a lumbar epidural catheter in the obstetric patient is unknown. At present individual preference determines choice of the lateral or sitting position. However cases have occurred of hypotension in patients placed in the lateral position with hips flexed to maximize spinal flexion.

To investigate this further we evaluated the effect of maternal posture on cardiac output. Cardiac output was measured using the principal of thoracic bioimpedance (Bomed NCCOM3 Monitor). This technique has previously been employed in pregnant patients and has been shown to have acceptable correlation with both invasive and non-invasive thermodilution and M-mode echocardiography as a means of determining cardiac output.

We studied 20 healthy patients at term with a singleton pregnancy and 20 female controls. All subjects provided their own baseline values.

Subjects were placed sequentially in four positions: supine; 15° left tilt (wedged position); left lateral with hips and spine flexed; and sitting with spine and neck flexed. Each study period lasted 5 min. Measurements of thoracic impedance, ejection velocity, heart rate and derived variables of cardiac index and stroke volume were recorded every 12 beats. Maternal blood pressure and fetal heart rate were recorded. Statistical analysis was by repeat measures analysis of variance.

The wedged position was taken as baseline. In the supine position there was a reduction in cardiac index (CI) of 8.7%, 4.69–4.28 litres/min⁻¹. The largest changes occurred in the left lateral position in which CI was reduced by 22.7% from 4.69 to 3.63 litres/min⁻¹. In the sitting position CI was reduced only in the order of 6.6% from 4.69 to 4.38 litres/min⁻¹ (Table 1). There was a similar reduction in the control group between the wedged and lateral positions in that the CI dropped by 12.6%. The changes in CI were significant (P<0.01).

In the patient group the stroke index was reduced by 10% in the supine position, 21.9% in the left lateral position and 12.8% in the sitting position. These differences were statistically significant (P<0.01).

Between the wedged and lateral positions there was a significant reduction in systolic blood pressure in the patient group by 9.8% from 118 to 106 mmHg. There were no significant changes in maternal or fetal heart rate.

The study demonstrates that, of the two positions chosen by anaesthetists for the insertion of epidurals, the flexed left lateral position has significantly adverse effects on maternal cardiac output. Therefore in the compromised patient it may be preferable to perform the regional block in the sitting position.

Table 1. Summary data for cardiac index

<table>
<thead>
<tr>
<th></th>
<th>Supine</th>
<th>Wedge</th>
<th>Left lateral</th>
<th>Sitting</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controls</td>
<td>Mean Litres/min</td>
<td>5.34 (4.73,6.04)</td>
<td>5.28 (4.67,5.96)</td>
<td>4.61 (4.09,5.21)</td>
</tr>
<tr>
<td>(95% CI)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patients</td>
<td>Mean Litres/min</td>
<td>4.28 (3.80,4.82)</td>
<td>4.69 (4.17,5.28)</td>
<td>3.63 (3.22,4.09)</td>
</tr>
<tr>
<td>(95% CI)</td>
<td></td>
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</tr>
</tbody>
</table>

Model used — log (cardiac index) — transformation used to achieve normality of the data.
THE GENETIC PREDISPOSITION TO PRE-ECLAMPSIA

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There is increasing evidence that there is a familial tendency to pre-eclampsia. In order to research this possibility, a study was set up with the University of Iceland to investigate the incidence of pre-eclampsia through four generations. Iceland was used as it has a small island population which has been fairly static over this century.

All pregnancies delivered in the National Maternity Hospital in Reykjavik between 1931-1947 were studied. The total number was 7453. The index pregnancies were those with definite pre-eclampsia or eclampsia. 156 index pregnancies were found.

The descendants were followed through three or four generations. It was found that there was an increased incidence of the condition in the daughters and granddaughters of the index pregnancies compared with the daughters-in-law. This finding was consistent with a single gene inheritance with incomplete penetrance of around 48%.

Therefore other factors are involved which may also be genetic. There is evidence of immunological abnormalities related to malplacentation. However, studies have failed to discover linkage to the HLA-DR4 gene. Our preliminary studies show no evidence of linkage to the renin gene.

The abnormality could be related to the cardiovascular system. A retrospective study of the incidence of a family history of cardiovascular disease was set up in Glasgow. Fifty four patients with severe pre-eclampsia were compared with 100 normotensive controls. A significantly higher incidence of cardiovascular disease was found in the families of the pre-eclamptic patients. Could this be a sign of a general vascular abnormality? It would appear to support the use of low dose aspirin as early therapy of the condition.

Can the knowledge of the family history be used as a predictor of higher risk of pre-eclampsia? An investigation is still under way in Glasgow to answer this. We have screened 1000 primigravida with a simple questionnaire. Initial results show that 35% would appear to be at high risk and 35% at low risk. If these findings are confirmed, intervention studies directed at those at high risk will be possible.

MAGNESIUM SULPHATE IN THE MANAGEMENT OF ECLAMPSIA AND SEVERE PRE-ECLAMPSIA

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Eclampsia is but one manifestation, albeit a dramatic and serious one, of severe multiorgan disease. Magnesium sulphate is not an accepted anticonvulsant but its efficacy in eclampsia is assumed from wide usage and large published series. Magnesium does not cross the blood-brain barrier in significant amounts and therefore its anticonvulsant efficacy must be in actions such as selective vasodilation, calcium channel blocking and inhibition of catecholamine release.

In Cape Town we arrest convulsions with clonazepam 1 mg i.v. followed by magnesium sulphate 4 g i.v. in a slow loading dose and then 1–2 g i.v. hourly by continuous infusion. This regime produces safe, effective but often unpredictable serum levels as these are rapidly influenced by urinary output. This regime was found to be more effective and safer than phenytoin or diazepam in two recently published series.1,2

The need for anticonvulsant therapy, and the possible role of magnesium sulphate, in the management of severe pre-eclampsia is less certain. It is important first to understand the underlying disease process and particularly the haemodynamic changes that are involved.

Finally, magnesium, in a dose of 3 g, can lessen the hypertensive surge during intubation,3 and can potentiate the action of non-depolarizing muscle relaxants, but probably not that of depolarizing agents such as suxamethonium.
ASPIRIN AND ITS RELEVANCE TO PRE-ECLAMPSIA

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Low dose aspirin is already widely used in pregnancy for the prophylaxis of pre-eclampsia, intrauterine growth retardation and to improve fetal outcome in lupus. Meta-analysis of the 10 small trials that have already been performed suggests that aspirin reduces the risk of these complications from 15% in a high risk placebo treated group to 38%. The proposed mechanism of low dose aspirin is irreversible inhibition of platelet cyclooxygenase preventing ADP stimulated platelet aggregation and thromboxane formation. But in contrast to high dose aspirin, low dose aspirin does not prevent the synthesis of prostacyclin from vascular endothelium.

Such an inhibition of platelet aggregation also has the potential for increasing the bleeding risk and indeed the bleeding time has been increased in some but not all series of patients exposed to low dose aspirin. However it is not clear that the effects of aspirin that have been demonstrated in vitro and the prolongation of bleeding time represent a true increased risk of maternal bleeding and particularly of epidual haematoama. For example, platelets can still adhere to the endothelium even if not stimulated by ADP. Reanalysis of the data from the Collaborative Perinatal Project shows that there was slightly less bleeding in 2269 mothers exposed to aspirin in the last 10 days of pregnancy compared to 7606 unexposed. There has been no excess bleeding risk demonstrated in the 10 studies that have already been reported concerning the use of low dose aspirin in pregnancy. CLASP is a large multicentre placebo controlled trial of 60 mg of aspirin per day in which 10 000 patients will eventually be randomized. In January 1991 the data monitoring committee examined the data of the 3400 women who had already been delivered. 1069 women were known to have had epidual blocks and haemorrhage was reported on three occasions, in all instances limited to blood-stained fluid in the cannula during treatment. One was in a woman on aspirin and two in women on placebo. There is therefore no evidence at present of any increased risk of bleeding in association with the use of low dose aspirin in pregnancy. Further data will be available when the outcomes of CLASP and other large trials are reported.

References

2. Brent R L. Personal communication.

BIRTH ASPHYXIA AND CEREBRAL PALSY

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The traditional belief that birth asphyxia is a common cause of brain damage is being challenged. Renewed interest was prompted by the rising trend in litigation following birth asphyxia. There is no satisfactory clinical definition of 'birth asphyxia'. The so-called markers of fetal asphyxia including cardiotocography, fetal scalp pH, meconium staining of the liquor, and the Agpar score do not tell us the extent to which the brain of the fetus suffered an hypoxic-ischaemic insult. Probably the most reliable clinical indicator of such an insult during labour is the occurrence, within 24 h of birth, of neonatal encephalopathy comprising, in its fully developed form, seizures, abnormality of muscle tone, and loss or severe reduction in level of consciousness.

About 2 per 1000 children have cerebral palsy, defined as a non-progressive disorder of movement caused by a brain abnormality which has its origins before birth or in early infancy. Current evidence suggests that, when the brain is damaged by intrauterine asphyxia resulting in impairment or handicap, then the dominant abnormality is cerebral palsy. Mental retardation alone is not caused by fetal asphyxia. Similarly, epilepsy or visual or hearing impairment in the absence of cerebral palsy is not caused by fetal asphyxia. If conventional markers of birth asphyxia are present yet there is no encephalopathy [or only mild encephalopathy], then a normal outcome can be anticipated. Conversely, probably 80–90% of babies
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who survive a severe hypoxic-ischaemic encephalopathy go on to develop cerebral palsy.

Only 10% of cases of cerebral palsy are caused by fetal asphyxia, and in these cases the type of cerebral palsy is almost always spastic quadriplegia. There remains the possibility that in some of these children there was a predetermined brain abnormality operating before the onset of labour making the fetus vulnerable to asphyxia during labour. The following must apply before it can be said that a child's impairment is caused by 'birth asphyxia' (adapted from American Academy of Pediatrics):

1. Evidence of severe asphyxia during labour (e.g. CTG, fetal scalp pH).
2. The baby must be in a poor condition at birth and require resuscitation.
3. Occurrence of severe neonatal encephalopathy.
4. Dominant impairment must be cerebral palsy.
5. Investigations point to no other cause.

Paediatricians may unwittingly make life difficult in court for their obstetric colleagues by jumping to erroneous conclusions when faced with a baby who fails to breathe promptly at birth.

THE ANAESTHETIC CONTRIBUTION TO MATERNAL MORTALITY IN NEW ZEALAND 1969–1986

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In New Zealand the Maternal Mortality Research Act came into force in 1969. Since then, and until 1986, the Maternal Death Assessment Committee has reviewed all reported deaths 'occurring to a woman during pregnancy or within a period of 3 months after pregnancy'. This review of all the data includes an assessment by the author of the notes of all anaesthesia-related deaths.

There were 393 reported maternal deaths and 24 (6.1%) were anaesthesia-related. A comparison with other reports from around the world is presented in Table 1. It must be remembered that differing classifications of maternal death will bias these figures.

The classification of several of the New Zealand anaesthesia-related deaths was difficult because of the inadequacy of the patient notes and detail available to the committee, whose opinions were retrospective. Aspiration was demonstrated in 8 patients; failed intubation in 3 patients; severe bronchospasm occurred in 4 patients and other airway or ventilation problems in a further 4 patients. Cardiac arrest from other causes occurred in 4 patients and there was 1 proven anaphylaxis. Mask anaesthesia and obesity were among the risk factors. No patients died from complications of regional anaesthesia.

Whilst the denominator, that is the number of epidurals given or even the number of general anaesthetics given in New Zealand for obstetrics is unknown, figures for the National Womens Hospital, Auckland may be a guideline. In 1990 there were 7831 births, 1359 by caesarean section. A total of 2602 epidurals were administered: 1032 for caesarean section, 731 where the outcome was assisted vaginal delivery and 839 where the outcome was normal vaginal delivery. It is interesting to compare the National Womens Hospital practice in 1970, 1980 and 1990 (Table 2).

We hope that the trend to epidural anaesthesia will further lessen the risk of harm to the mothers of New Zealand.

Table 1. Anaesthesia for caesarean sections — National Womens Hospital

<table>
<thead>
<tr>
<th>Year</th>
<th>Caesarean sections</th>
<th>General anaesthesia</th>
<th>GA + epidural</th>
<th>Epidural</th>
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<tr>
<td>1970</td>
<td>357</td>
<td>320</td>
<td>9</td>
<td>28</td>
</tr>
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<td>1980</td>
<td>749</td>
<td>476</td>
<td>31</td>
<td>242</td>
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<tr>
<td>1990</td>
<td>1359</td>
<td>303</td>
<td>24</td>
<td>1032</td>
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Table 1.

<table>
<thead>
<tr>
<th>Country</th>
<th>Period</th>
<th>Maternal deaths</th>
<th>Anaesthesia related deaths</th>
<th>%</th>
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<tr>
<td>USA</td>
<td>1974–1986</td>
<td>5119</td>
<td>184</td>
<td>3.6</td>
</tr>
<tr>
<td>Canada</td>
<td>1970–1980</td>
<td>100</td>
<td>15</td>
<td>15</td>
</tr>
<tr>
<td>Australia</td>
<td>1964–1981</td>
<td>715</td>
<td>64</td>
<td>8.95</td>
</tr>
<tr>
<td>South Africa</td>
<td>1980–1982</td>
<td>737</td>
<td>35</td>
<td>4.75</td>
</tr>
<tr>
<td>Germany (38%)</td>
<td>1971–1980</td>
<td>328</td>
<td>21</td>
<td>6.4</td>
</tr>
<tr>
<td>New Zealand</td>
<td>1969–1986</td>
<td>393</td>
<td>24</td>
<td>6.1</td>
</tr>
</tbody>
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FURTHER INVESTIGATIONS OF OMEPRAZOLE IN OBSTETRIC PATIENTS

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The routine use of antacid therapy during labour is controversial but most anaesthetists would agree that patients requiring regional or general anaesthesia should be treated. A recent survey\(^1\) has shown that suppression of acid secretion by H\(_2\) receptor blockade, supplemented by a saline antacid if operative obstetrics becomes necessary, is being widely used. Nevertheless H\(_2\) receptor blockers by inhibiting liver enzymes and reducing hepatic blood flow could influence the pharmacokinetic pattern of other drugs. Omeprazole inhibits the proton pump of the parietal cell by way of a metabolite which is produced only in the highly acid milieu existing there.\(^2\) The parent drug in clinical doses has been shown to have no discernable effects elsewhere and the active metabolite has a very short half-life when absorbed. Non-obstetric studies indicated that a single dose provided a marked reduction in acid output for periods up to 24 h.\(^3\) Investigation of its effects as pre-anaesthetic antacid therapy was indicated and two investigations of its efficacy, side-effects and placental transfer in patients undergoing elective caesarean section were carried out.

Results of study 1\(^3\)

Twenty women were given 80 mg omeprazole as enteric coated capsules at 20:00 on the evening prior to surgery. Eighty five percent of patients anaesthetized some 12–16 h later had gastric aspirates of pH > 2.5 and volume < 25 ml. Placental transfer was limited, 16 and 13 of the umbilical vein and artery samples respectively having levels below the measurement limit. No omeprazole-related maternal of fetal side-effects were observed.

Results of study 2

This investigation was randomized and double-blind in a similar patient population to study 1. There were two main study groups. Group 1 were given 40 mg omeprazole at 20:00 on the evening before surgery with a similar dose at 06:00 on the morning of operation. Group 2 received 80 mg dose at 06:00 on the morning of operation. The two other series, groups 3 and 4 received corresponding omeprazole therapy to 1 and 2 but in addition were given 10 mg metoclopramide i.m. 20 min before induction of anaesthesia. Aspiration of gastric contents immediately post-induction showed that 87, 73, 100 and 81% of patients in groups 1, 2, 3 and 4 respectively had aspirates of pH > 2.5 and volume < 25 ml. No significant side-effects attributable to the treatments were recorded.

Omeprazole, because of its unique action on the acid output of the parietal cell, and limited placental transfer may provide an appropriate antacid treatment for the parturient. However, its evaluation in the emergency situation is required and its use would still pose the problem of existing stomach acid together with a delayed onset time. It does give hope of a single antacid therapy for labours up to 12 h duration.

References