Highlanders were also less likely than other respondents to report having consumed alcohol in the previous week.

**Levels of alcohol consumption**—Table IV shows the mean levels of alcohol consumption reported by men and women in the three areas. Significant differences were reported in the mean amounts consumed during the previous week by both sexes in the three areas. Among men, those in Tayside drank the most; whereas among women, those in Kent were the heaviest drinkers. Highlanders drank the least. These differences did not appear to arise from either sampling or response bias (Crawford, and Crawford et al, unpublished observations).

**TABLE IV**—Mean levels of self reported alcohol consumption (units*): among respondents who had consumed alcohol during previous week

<table>
<thead>
<tr>
<th>Respondents</th>
<th>Area</th>
<th>Highland</th>
<th>Tayside</th>
<th>Kent</th>
<th>Region</th>
<th>F = 5.7; df = 2,1407; p &lt; 0.003</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men</td>
<td></td>
<td>15 4</td>
<td>21 1</td>
<td>16 9</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Women</td>
<td></td>
<td>15 5</td>
<td>6 6</td>
<td>7 2</td>
<td></td>
<td>p = 0.001</td>
</tr>
</tbody>
</table>

*Each unit equivalent to half pint (285 ml) ordinary beer, lager, etc, or single glass of wine or spirits. Each unit contains approximately 10 cl/79 g absolute alcohol. 15 (Area) x 2 (respondent’s sex) analysis of variance with scores loged to base 10.

**Conclusions**

These results do not support the view that levels of alcohol consumption in the community mirror officially recorded rates of treated morbidity for alcohol dependence. In this respect this general population survey produced results fully compatible with those of our clinical survey. Together these studies suggest that the widespread belief that alcohol dependence is vastly more commonplace in the north than in the south of Britain is more of a myth than a reality. Regional differences in other indicators of alcohol misuse remain to be explained. Several policy options exist whereby alcohol misuse may be curbed. These range from controlling the price and availability of alcohol to attempting to make public drinking more relaxed and socially integrated. Some of these are reviewed elsewhere.13

This study was funded by the Economic and Social Research Council and the Medical Research Council. Additional support was given by the Scotch Whisky Association and by the Brewers’ Society. Fieldwork was efficiently conducted by Survey Research Associates, London. Some of the data will be included in a Glasgow University PhD thesis.

References


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Reliability of cardiotocography in predicting baby’s condition at birth

P CURZEN, J S BEKIR, D G McLINTOCK, M PATEL

Abstract

A prospective study of 6825 labours was undertaken to determine the relationship between the Apgar scores of the babies at one minute and the cardiotocograph tracing in labour. The sensitivity of an abnormal tracing was 35.2% for babies who needed intermittent positive pressure ventilation and 20.0% for babies who did not but who had Apgar scores of less than 7. The sensitivity of an abnormal tracing for all babies with an Apgar score of less than 7 was 23.2%. The positive predictive value of an abnormal tracing was 8.7% for babies who needed intermittent positive pressure ventilation and 18.7% for babies who did not but who had an Apgar score of less than 7. The positive predictive value of an abnormal tracing was 27.4% for all babies with an Apgar score of less than 7. The specificity of the tracing was 93.4% for babies with an Apgar score of 7 or over. The relatively high incidence of false positive predictions might be explained on the grounds that abnormalities in the cardiotocograph tracing are a more sensitive indicator of hypoxia than the Apgar score. False negative predictions might have been due to adverse factors other than hypoxia—for example, fetal trauma, compression of the head, infection, and analgesia in labour.

These findings suggest that the current overdependence on fetal monitoring by cardiotocography alone should be examined and that other reliable indicators for non-hypoxic fetal distress should be sought.

Introduction

Continuous monitoring of the fetal heart rate in labour is now widespread, but the fetal benefits of this have been questioned.1 Earlier papers reporting the results of randomised controlled trials showed that continuous monitoring was associated with a
significant increase in the incidence of operative deliveries but there was no definite fetal advantage. 2-5 We report a prospective study of 6825 consecutive labours in which we compared the clinical condition of babies who were delivered operatively because of fetal distress diagnosed by cardiotocography and babies whose cardiotocograph tracing had been normal.

Patients and methods

We analysed the computerised results of 6825 consecutive labours. Some mothers with uncomplicated obstetric histories and pregnancies were monitored only by intermittent auscultation of the fetal heart at intervals of 15 minutes and were not considered further. All the rest were monitored continuously by cardiotocography, the recordings being interpreted at the time by the obstetric registrar on duty. Operative delivery for fetal distress was performed if the cardiotocograph tracing showed late decelerations accompanied by baseline tachycardia or loss of baseline variation, or both.6 The clinical condition of the newborn babies was assessed by the Apgar score at one minute and by the need for intermittent positive pressure ventilation. The data were analysed by the department of computer medicine at Westminster Hospital.

Results

From 1 January 1978 to 31 December 1982 there were 6825 deliveries. Of these, 683 were monitored only by intermittent auscultation of the fetal heart and were not studied further. Continuous monitoring of the fetal heart rate by cardiotocography was used in 5962 labours (87%). Table I shows the relation between the cardiotocograph tracing in labour and the condition of the baby at birth. In 492 labours (8.3%) operative delivery was undertaken because of an abnormal tracing.

<table>
<thead>
<tr>
<th>Cardiotocograph tracing</th>
<th>Mode of delivery</th>
<th>&lt;7, IPPV not needed</th>
<th>&lt;7, IPPV needed</th>
<th>7 or over</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Forceps</td>
<td>10 (3%)</td>
<td>36 (12%)</td>
<td>251 (85%)</td>
<td>297</td>
</tr>
<tr>
<td>Normal</td>
<td>Ventouse</td>
<td>1 (4%)</td>
<td>4 (8%)</td>
<td>8 (13)</td>
<td>13</td>
</tr>
<tr>
<td>Normal</td>
<td>Caesarean</td>
<td>32 (18%)</td>
<td>52 (29%)</td>
<td>98 (53%)</td>
<td>182</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>43</td>
<td>92</td>
<td>357</td>
<td>492</td>
</tr>
</tbody>
</table>

IPPV = Intermittent positive pressure ventilation.

Sensitivity of an abnormal cardiotocograph tracing—Forty three babies (35.2%) who needed intermittent positive pressure ventilation and 92 (20.0%) of those who did not but who had Apgar scores of less than 7 were delivered operatively because of an abnormal tracing. The sensitivity of cardiotocography for all babies with Apgar scores of less than 7 was 23.2%.

Positive predictive value of an abnormal cardiotocograph tracing—Of the babies delivered operatively because of an abnormal tracing, 43 (8.7%) needed intermittent positive pressure ventilation and 92 (18.7%) did not but had Apgar scores of less than 7. The positive predictive value of cardiotocography for all babies with Apgar scores of less than 7 was 27.4%.

Specificity of cardiotocography—Of the 5379 labours in which the babies had an Apgar score of 7 or over, 5022 (93.4%) had normal cardiotocograph tracings.

Table II shows the relation, in cases with an abnormal cardiotocograph tracing, between the mode of delivery and the condition of the baby at birth. The positive predictive value of an abnormal tracing for babies who needed intermittent positive pressure ventilation was 3.2%, when delivery was by forceps and 18.0% when delivery was by caesarean section (SE = 3.0; 0.001 < p < 0.001). The positive predictive value of an abnormal tracing for babies who did not need ventilation but had Apgar scores of less than 7 was 12.9%, when delivery was by forceps and 29.0%, when delivery was by caesarean section (SE = 3.9; 0.01 < p < 0.05). The positive predictive value of an abnormal tracing for all babies with an Apgar score of less than 7 was 15.1%, when delivery was by forceps and 47.2% when delivery was by caesarean section (SE = 4.2; p < 0.001).

Table III shows the relation, in cases with a normal cardiotocograph tracing, between the mode of delivery and the condition of the baby at birth. The proportion of babies who needed intermittent positive pressure ventilation was 0.6% of those delivered spontaneously, 1.8% of those delivered by forceps (SE = 0.49; 0.01 < p < 0.05), and 11.7%, of those delivered by caesarean section (SE = 2.12; p < 0.001). The proportion of babies whose Apgar score was less than 7 was 5.8%, of those delivered spontaneously, 9.9% of those delivered by forceps (SE = 1.12; p < 0.001), and 38.5% of those delivered by caesarean section (SE = 3.22; p < 0.001).

Discussion

The purpose of this study was to assess the value of cardiotocography on an in service basis, with tracings being analysed during routine management in the labour ward. The criteria for recognising an abnormal tracing were easy to apply in practice, and blind retrospective checking of tracings was not done.

We used the Apgar score at one minute as one of our criteria for assessing the value of cardiotocography despite criticisms that have been made of this criterion. Other workers have shown that the Apgar score does not usually reflect the degree of acidosis at delivery; 73% of babies with severe acidosis at birth were reported to have an Apgar score of 7 or over at one minute.7 Such a gross discrepancy, however, between severe acidosis and babies that were clinically well must leave open to question whether the biochemical or the clinical criterion is more accurate. It has also been argued that an abnormal cardiotocograph tracing during labour may well be due to fetal acidosis at that time, which then improves before the baby is delivered due to interim measures—for example, encouraging the mother to adopt a lateral tilt posture.8 These explanations have been offered to account for the false positive predictions of cardiotocography. The correlation between the tracing and fetal acidosis is well established.9 False negative tracings have been attributed to non-hypoxic factors in labour—for example, metabolic, infective, or traumatic factors—which would not produce an abnormal tracing because they are not necessarily associated with fetal acidosis.9 Our study produced some evidence in support of this. In those cases in which the cardiotocograph tracing was normal throughout labour there was an appreciably larger proportion of babies with Apgar scores of less than 7 or requiring intermittent positive pressure ventilation.
among those delivered operatively than among those delivered normally. The operative deliveries were undertaken for failure to advance in labour due to uterine, pelvic, or fetal factors, the fetal factors being either malpresentation or malposition.

We applied the Apgar score in our study because it remains widely used as a simple clinical index of the condition and behaviour of newborn babies. So far, nothing better has been found to replace it.

This study showed that both the sensitivity and the predictive value of an abnormal cardiotocograph tracing in labour for babies born with an Apgar score of less than 7 were poor, whether or not the babies required intermittent positive pressure ventilation. A normal tracing was obtained in 77% of all babies with an Apgar score of less than 7 and 65% of those who needed intermittent positive pressure ventilation. Cardiotocography gave a false positive prediction in 73% and a false negative prediction in 8% of all babies.

The positive predictive value of cardiotocography was significantly better in those babies delivered by emergency caesarean section compared with those delivered by forceps. Most caesarean sections were performed in the first stage of labour, and all forceps deliveries were performed in the second stage. We do not think that the assessment of the cardiotocographic tracings was different for the two groups, which suggests that cardiotocography is more reliable in the first stage than in the second stage of labour.

References

(Accepted 29 August 1984)

SHORT REPORTS

Generalised epileptic fits in renal transplant recipients given cyclosporin A

Cyclosporin A is widely used to induce immunosuppression after organ transplantation. Reported neurological side effects include tremor, ataxia, confusion, paraparesis, and quadriparesis.1 Epileptic fits have not been reported in patients given renal transplants but may occur after bone marrow transplantation.2 In our unit cyclosporin A (5-10 mg/kg/day) is administered 12 hourly to achieve predose whole blood concentrations of 200-400 μg/l, as measured by radioimmunoassay.

Case reports

Case 1—Thirty days after cadaveric renal transplantation this patient developed right sided headache and blurring of vision and then had three generalised epileptic fits. On recovery neurological examination showed nothing abnormal. He was metabolically stable (plasma concentration of sodium 138 mmol/l, potassium 4.8 mmol/l, calcium 2.4 mmol/l, 9-0 mg/100 ml, bicarbonate 18 mmol/l, urea 33 mmol/l (199 mg/100 ml), creatinine 345 μmol/l (3.9 mg/100 ml)). No acute change in renal function was noted. A computed tomogram (CT) of the brain was normal. Cyclosporin A concentration was 584 μg/l.

Case 2—Four days after cadaveric renal transplantation this patient reported visual flashes and then had four generalised epileptic fits. On recovery neurological findings were normal. His metabolic state was stable (plasma concentration of sodium 139 mmol/l, potassium 4.9 mmol/l, bicarbonate 22 mmol/l, urea 36 mmol/l (217 mg/100 ml), creatinine 892 μmol/l (10-1 mg/100 ml), calcium 2-3 mmol/l (9-2 mg/100 ml)). Electroencephalography showed a normal rhythm. Cyclosporin A concentration was 1075 μg/l.

Case 3—Two months after a second transplant operation this patient felt unwell and saw rainbow bands; she then began jerking her head to the left and had five generalised epileptic fits. On recovery neurological examination elicited weakness of the left side of her mouth and exaggerated reflexes on the right side with clonus of the knee. These signs cleared over the next two days. She was metabolically stable (plasma concentration of sodium 135 mmol/l, potassium 4-2 mmol/l, urea 56 mmol/l (337 mg/100 ml), creatinine 467 μmol/l (10-5 mg/100 ml), calcium 2-55 mmol/l (10-2 mg/100 ml)).

CT showed a low density area in the white matter of the right parietal lobe, which had disappeared at the next examination one week later. Cyclosporin A concentration was 533 μg/l.

Common clinical features

Each of these patients had generalised epileptic fits for the first time in their lives while receiving cyclosporin A after renal transplantation. Phenytoin was prescribed as an anticonvulsant measure and the cyclosporin A reduced to achieve blood concentrations of 200-400 μg/l (recommended range). The patients had no further fits. None had an acute rejection episode, although the third patient, whose renal transplant function was poor initially, required chronic ambulatory peritoneal dialysis for two months after the episode. One month later her transplanted kidney was functioning well. These patients were receiving no other drugs which might have been responsible for the fits. Their blood pressures were stable and there was no evidence of hypertensive encephalopathy. The patients were followed up for four, 18, and three months, respectively; there was no recurrence of fits and all three transplants were functioning well (serum creatinine concentration < 300 μmol/l (< 3-4 mg/100 ml)).

When the epileptic fits occurred in cases 1 and 2 an initial diagnosis of rejection was made, but this was not proved.

Comment

Epileptic fits in patients with transplants may be caused by infection, rejection, or metabolic upset. So far as we could tell these were ruled out in our patients: all had normal cerebrospinal fluid findings, the serum viral titres did not rise, and their weight and metabolic states were stable. Space occupying lesions and other organic lesions of the central nervous system were also unlikely, as CT scans and electroencephalograms showed no persistent abnormality. Epileptic fits after renal transplantation may occur due to encephalopathy associated with an acute rejection episode,4 but none of our patients had acute rejection at the time.

Epileptic fits in the absence of a conventional cause in three of our patients over 18 months led us to suspect that cyclosporin A might be responsible or at least a predisposing factor. Although epileptic fits caused by cyclosporin A have not been reported in renal transplant recipients, epileptic fits have been reported in association with cyclosporin A given after bone marrow transplantation.5

1. }
2. }
3. }
4. }
5. }