posthaemorrhagic hydrocephalus, we should ask whether the aim of treatment is to maintain CSF pressure within the normal range, regardless of other circulatory factors, or whether the maintenance of cerebral perfusion is more important.

The wide normal range of cerebral artery BFV does mean that a single 'action line' is not appropriate for all infants. Study of longitudinal change in individual infants may be a more fruitful approach which requires further investigation.


Maternal carboxyhaemoglobinemia

EDITOR,—We wish to report a case of maternal carboxyhaemoglobinemia which resulted in evidence of fetal compromise and delivery at a cesarean section. We believe that the case highlights both a lack of management guidelines and adequate facilities for treating this serious condition in pregnancy.

Case report

A 21 year old, non-smoking primigravida at 39 weeks' gestation accidentally inhaled carbon monoxide from a faulty gas heater for approximately two hours. On arrival at accident and emergency, three hours later, she had a headache, felt drowsy, and had reduced fetal movements (blood pressure 155/75, pulse 100/min). She had been transferred in oxygen and had normal arterial blood gases (pH 7.43, carbon dioxide tension 4.27 kPa, oxygen tension 14.7 kPa, base excess −2.2 mmol/l) but the carboxyhaemoglobin percentage was raised at 13%. Fetal heart monitoring revealed fetal tachycardia, diminished variability and decreased late decelerations. Emergency caesarean section was performed and 10 minutes after delivery a maternal carboxyhaemoglobin estimation was 3%.

The girl, with a birth weight of 3590 g, had normal Apgar scores but the carbon monoxide level in cord blood was 16.2%. After delivery no neurological compromise was noted and blood gases remained satisfactory in air. Mother and child were subsequently discharged at 5 days and no problems were noted at follow up.

Faulty gas heaters remain a major reason for 1000 deaths annually from carbon monoxide poisoning.1 Stillbirths have been recorded at relatively low maternal levels of carboxyhaemoglobin because fetal carbon monoxide concentrations can exceed maternal concentrations and also there is an additional shift of the fetal haemoglobin dissociation curve to the left.2 In this case we encountered a pregnant patient with mild to moderate carboxyhaemoglobinemia who was symptomatic with evidence of fetal distress. Some authorities, particularly in the US, would advocate hyperbaric oxygen (HBO) in this situation,3 which might avoid the high anaesthetic risk in caesarean section. It would also reduce the considerable risk of delivering a neonate requiring intensive monitoring which might affect, if preterm, in addition respiratory distress syndrome.

Our inquiries indicate that just 10 main

land UK hyperbaric units accepted civilian carbon monoxide poisoning cases in 1988–90 and only one was actually equipped with operating facilities. Therefore HBO is a management option that few paediatric/obstetric teams can currently contemplate, though it has considerable potential clinical advantages.

The management of fetal distress in mild to moderate maternal carboxyhaemoglobinemia, particularly in the preterm setting, needs clarification.


Ureaplasma and mycoplasma central nervous system infections in neonates

EDITOR,—Waite et al describe two studies in Birmingham, Alabama in which Mycoplasma hominis and Ureaplasma urealyticum were isolated from the cerebrospinal fluid (CSF) of neonates.1 2 In the first study of 100 predominantly preterm babies, U urealyticum and M hominis were isolated from the CSF of eight and five of the neonates respectively.1 The mothers were generally of low socioeconomic status and few had received antenatal care. In their second study, of 318 predominantly full term neonates, the mothers of whom had received private obstetric care, U urealyticum and M hominis were isolated from nine infants.2 They suggested that culture for these organisms should be attempted on all CSF specimens from neonates with progressive hydrocephalus, CSF pleocytosis, or evidence of congenital infection.2

We prospectively studied the babies admitted to our neonatal intensive care unit over a 15 month period. We cultured 42 CSF specimens from 35 neonates for M hominis and U urealyticum, as part of the microbiological investigation of suspected sepsis or therapeutic tap for hydrocephalus. The mean gestational age was 32 weeks (range 24–41 weeks), mean birth weight 2140 g (range 700–4400 g), and mean age at first sampling was 10 days (range 8–35 days). The mothers were from varied socioeconomic backgrounds and all had received antenatal care.

No CSF specimen yielded a growth of M hominis or U urealyticum. Although our study is small, our findings are more in keeping with Fries et al5 and M hominis prevalence rates reported by Shaw et al6 in Liverpool than those quoted by Waite et al. As UK rates of maternal colonisation with these organisms are similar to elsewhere it seems that other factors must be in play producing differences in neonatal central nervous system infection rates. Further studies are probably justified. However, like Shaw et al we do not feel that CSF culture for these organisms is indicated in our population.

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