antibody is associated with a favourable outcome.⁶

ELIZABETH MILLER
Immunisation Division, PHLS Communicable
Disease Surveillance Centre,
61 Colindale Avenue,
London NW9 5EQ

- McIntosh D, Isaacs D. Varicella zoster virus infection in pregnancy. *Arch Dis Child* 1993; **68**: 1-2.
- Miller E, Cradock-Watson JE, Ridehalgh KS. Outcome in newborn babies given anti-varicella-zoster immunoglobulin after perinatal maternal infection with varicella-zoster virus. *Lancet* 1989; **ii**: 371-3.
- Department of Health. *Immunisation against infectious disease*. London: HMSO, 1992.
- Brunnell PA. Varicella in pregnancy, the fetus, and the newborn: problems in management. *J Infect Dis* 1992; **166** (suppl 1): S42-7.
- Rubin L, Leggadro R, Elie MT, Lipsitz P. Disseminated varicella in a neonate: implications for immunoprophylaxis of neonates postnatally exposed to varicella. *Pediatr Infect Dis J* 1986; **5**: 100-2.
- Baba K, Yabuuchi H, Takahashi M, Ogra PL. Immunologic and epidemiologic aspects of varicella infection acquired during infancy and early childhood. *J Pediatr* 1982; **100**: 881-5.

Dr McIntosh and Professor Isaacs comment:

Our table 2 was in fact obtained from an earlier article published by the same authors in the Communicable Disease Report 4th April 1986 (CDR 86/14), which cannot be used as a reference. We had overlooked the increased numbers of children studied in Dr Miller's 1989 *Lancet* article,¹ and regret this. We do not necessarily agree, however, with her subsequent conclusions.

The severity of neonatal chickenpox depends on the dose of virus as well as the presence or absence of maternal antibody. Epidemiologically most of the severe and fatal cases have been babies whose mother developed chickenpox five or fewer days before delivery. In the paper of Miller *et al* 16 of 19 'severe' neonatal chickenpox cases were babies whose mother's rash was 4 days before to 2 days after delivery.¹ The 1991 Red Book has continued to recommend VZIG for maternal chickenpox at -5 to +2 days,² even though the JCVI has been persuaded to change to -7 days on theoretical grounds.

There have been anecdotal reports of fatal postnatally acquired chickenpox, but there are also fatal cases in apparently immune competent children and adults. It is not known whether the incidence of fatal disease is higher in postnatally infected neonates than later in life, and the Red Book does not recommend VZIG for these babies.

- Miller E, Cradock-Watson JE, Ridehalgh KS. Outcome in newborn babies given anti-varicella-zoster immunoglobulin after perinatal maternal infection with varicella-zoster virus. *Lancet* 1989; **ii**: 371-3.
- American Academy of Pediatrics. *Varicella-zoster infections. Report of the Committee on Infectious Diseases*. 22nd Ed. Illinois: AAP, 1991: 517-24.

Proportion of neonates with antibodies to varicella zoster at birth according to onset of maternal rash²

Onset of maternal rash before delivery (days)	Proportion of babies with detectable antibody*
≤2	0/60
3-5	31/56
6	4/9
7	5/6
8-14	38/38

* >1 arbitrary unit detected by enzyme linked immunosorbent assay.

Echocardiographic assessment of cardiac function in shocked very low birthweight infants

EDITOR,—In their excellent article Gill and Weindling state that neonatal serum electrolyte values are not routinely measured immediately after birth because they, 'invariably reflect maternal levels'.¹ While this statement may be true, and is a frequent excuse for not determining newborn serum electrolyte concentrations early on, obstetricians rarely provide appropriate maternal results. Neonatologists suffer from this lack of information and are frequently surprised at serum electrolyte values eight or 12 hours after birth.

Electrolyte concentrations hold considerable interest for caretakers of sick newborns. Either the obstetricians must regularly provide maternal values at parturition or the neonatologist should measure the baby's concentration shortly after birth. To wait eight or 12 hours because early values reflect maternal levels, yet not to know maternal or newborn levels, is a cop out.

For Gill and Weindling not to have presented such information slightly tarnishes an otherwise excellent, informative report.

JOHN W SCANLON
Columbia Hospital for Women Medical Center,
2425 L Street Northwest,
Washington,
DC 20037, USA

- Gill AB, Weindling AM. Echocardiographic assessment of cardiac function in shocked very low birth weight infants. *Arch Dis Child* 1993; **68**: 17-21.

Drs Gill and Weindling comment:

Unfortunately Dr Scanlon has failed to quote exactly as written in the paper regarding serum electrolyte levels in the first few hours after birth. In our discussion we stated 'Serum calcium and potassium concentrations were not routinely measured in the first few hours after birth as they invariably reflect maternal levels. Subsequent measurements of calcium and potassium after 12 hours of age did not show any difference between the groups.' As the infants did not receive additional calcium and potassium during the first 24 hours we cannot accept that the serum electrolytes were likely to be abnormal at the time of the echocardiogram. To our knowledge, we have not seen any research evidence to suggest that electrolytes should be measured routinely in the first few hours after birth in all preterm infants.

Changes in cerebral artery blood flow velocity after intermittent cerebrospinal fluid drainage

EDITOR,—We agree with the findings of Kempley and Gamsu¹ but dispute their suggestion that these may be used to time cerebrospinal fluid (CSF) drainage in posthaemorrhagic hydrocephalus (PHH).

In a similar study of PHH and ventricular dilatation, we found the same reduction in pulsatility index and increase in mean flow velocity after CSF taps, but because of the very wide range of normal values, were not able to construct an 'action line' to indicate the need for intervention.² In addition, a number of factors unrelated to intracerebral haemodynamics (such as hypercapnia or the presence of a patent ductus arteriosus) may influence the pulsatility index and cerebral blood flow velocity in the ill newborn with

PHH. This is not the case in older children with hydrocephalus in which a clear clinical role for Doppler ultrasound exists.³

We believe that the changes observed by Kempley and Gamsu are acute changes and are of interest in so far as they may reflect the process of autoregulation but that they are unlikely to guide the timing of intervention. Furthermore, it is possible, in the absence of accurate measurements of the angle of insonation, that the flow velocity changes merely reflect a change in vessel distortion with CSF taps rather than a true change in flow velocity.

Intervention should continue to be guided by the rate of head growth and symptoms of raised intracranial pressure⁴ rather than changes in Doppler parameters.

MICHAEL W QUINN
Department of Child Health,
University of Exeter,
Postgraduate Medical School,
Church Lane,
Heavitree,
Exeter EX2 5SQ

MALCOLM I LEVENE
Department of Child Health,
University of Leeds,
Leeds General Infirmary,
Leeds LS2 9NS

- Kempley ST, Gamsu HR. Changes in cerebral artery blood flow velocity after intermittent cerebrospinal fluid drainage. *Arch Dis Child* 1993; **69**: 74-6.
- Quinn MW, Ando Y, Levene MI. Cerebral arterial and venous flow velocity measurements in post-haemorrhagic ventricular dilatation and hydrocephalus. *Dev Med Child Neurol* 1992; **34**: 863-9.
- Quinn MW, Pople I. Middle cerebral artery pulsatility with blocked cerebrospinal fluid shunts. *J Neurol Neurosurg Psychiatry* 1991; **55**: 325-7.
- Ventriculomegaly Trial Group. Randomised trial of early tapping in neonatal posthaemorrhagic ventricular dilatation. *Arch Dis Child* 1990; **65**: 3-10.

Dr Kempley and Dr Gamsu comment:

Dr Quinn and Professor Levene suggest that the changes in blood flow velocity (BFV) we observed after intermittent CSF drainage may be due to alterations in the angle of insonation from changes in vessel distortion. This is not the case, as in all measurements we visualised the artery and corrected for the angle of insonation.

We agree that at present, intervention in posthaemorrhagic hydrocephalus should be guided by the rate of head growth and by symptoms and signs of raised intracranial pressure. However, both of our studies demonstrated considerable variations in cerebral artery BFV with this form of management.^{1 2} Compromise of the cerebral circulation by raised intracranial pressure, in combination with other circulatory factors, could cause further cerebral damage in some infants.

The lack of a consistent reduction in cerebral artery BFV in their study² after the onset of ventricular dilatation (ventricular index >2 SD from the mean) may have been because intracranial pressure was not increased at this early stage of ventricular dilatation.

Neither study found a significant correlation between CSF pressure and BFV, perhaps because an infant's ability to maintain cerebral blood flow in the face of raised intracranial pressure depends on a number of factors, such as arterial blood pressure or the presence of a patent ductus arteriosus.

Before dismissing Doppler ultrasound as a tool which could aid decision making in

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 changes in individual infants may be a more fruitful approach which requires further investigation.

- 1 Kempley ST, Gamsu HR. Changes in cerebral artery blood flow velocity after intermittent cerebrospinal fluid drainage. *Arch Dis Child* 1993; **69**: 74-6.
- 2 Quinn MW, Ando Y, Levene MI. Cerebral arterial and venous flow velocity measurements in post-haemorrhagic ventricular dilatation and hydrocephalus. *Dev Med Child Neurol* 1992; **34**: 863-9.

Maternal carboxyhaemoglobinaemia

EDITOR.—We wish to report a case of maternal carboxyhaemoglobinaemia which resulted in evidence of fetal compromise and delivery by caesarean section. We believe 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 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 carboxyhaemoglobin 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 at equilibrium 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 carboxyhaemoglobinaemia 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, if preterm, have 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 carboxyhaemoglobinaemia, particularly in the preterm setting, needs clarification.

PAUL BUSS
R SUBAK-SHARPE*
M MCCABE†
Departments of Paediatrics,
Obstetrics*, and
Accident and Emergency†,
Royal Gwent Hospital,
Cardiff Road,
Newport,
Gwent NP9 2UB

- 1 Meredith T, Vale A. Carbon monoxide poisoning [Editorial]. *BMJ* 1988; **296**: 77-9.
- 2 Longo LD. The biological effects of carbon monoxide on the pregnant woman, fetus and newborn infant. *Am J Obstet Gynecol* 1977; **129**: 69-103.
- 3 Van Roeson KB, Campoersi EM, Moon RE, et al. Should HBO be used to treat the pregnant patient for acute carbon monoxide poisoning? *JAMA* 1989; **261**: 1039-43.

Ureaplasma and mycoplasma central nervous system infections in neonates

EDITOR.—Waites *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* was isolated from five infants and *M. hominis* 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.³

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 hours-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 the 0.7% *U. urealyticum* and 0% *M. hominis* prevalence rates reported by Shaw *et al*⁴ in Liverpool than those quoted by Waites *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.

This study was supported by a grant from the Welsh Scheme for the Development of Health and Social Research.

J L HOVENDEN
M C ATKINS
M DRAYTON*
Departments of Microbiology and Paediatrics*,
University Hospital Wales,
Heath Park,
Cardiff CF4 4XN

- 1 Waites KB, Rudd PT, Crouse DT, et al. Chronic Ureaplasma urealyticum and Mycoplasma hominis infections of central nervous system in preterm infants. *Lancet* 1988; **i**: 17-21.
- 2 Waites KB, Duffy LB, Crouse DT, et al. Mycoplasma infections of cerebrospinal fluid in newborn infants from a community hospital population. *Pediatr Infect Dis J* 1990; **9**: 241-5.
- 3 Waites KB, Crouse DT, Cassell GH. Ureaplasma and mycoplasma CNS infection in newborn babies. *Lancet* 1990; **i**: 658-9.
- 4 Shaw NJ, Pratt BC, Weindling AM. Ureaplasma and mycoplasma infections of the central nervous system in preterm infants. *Lancet* 1989; **ii**: 1530.

Congenital diaphragmatic hernia: influence of associated malformations on survival

EDITOR.—In the interesting article by Sweed and Puri they made no mention of an important autosomal recessive condition that includes congenital diaphragmatic hernia.¹ Fryns' syndrome describes infants who have severe diaphragmatic hernia associated with skeletal, palatal, and renal abnormalities.²⁻⁵

The outcome for infants with Fryns' syndrome is universally poor. Rapid identification of this syndrome in a baby born in our unit with severe diaphragmatic hernia enabled us to save the child and family the trauma of an emergency transfer to a surgical unit, so that the baby could die with peace and dignity with both parents in attendance. Urgent diagnosis was achieved through immediate access to regional colleagues in neonatology and clinical genetics. In this case, in addition to a diaphragmatic hernia, there was cleft palate, palpable polycystic right kidney, facial dysmorphic features of mid-face hypoplasia, and a small left pinna and hypoplastic nails.

This recessive inherited syndrome is not uncommon. Of 121 cases of congenital diaphragmatic hernia reported in the Northern region between 1985 and 1992, five (4%) had Fryns' syndrome (Edmund Hey, personal communication). Knowledge of this syndrome, therefore, with its implications for immediate care and the risks in future pregnancies is important for colleagues working with newborn babies.

P M JONES
Children's Services,
Health Centre,
Escomb Road,
Bishop Auckland,
Co Durham DL14 6AB

- 1 Sweed Y, Puri P. Congenital diaphragmatic hernia: influence of associated malformations on survival. *Arch Dis Child* 1993; **69**: 68-70.
- 2 Fryns JP, Moerman F, Goddeeris P, Bossuyt C, Van den Berghe H. A new lethal syndrome with cloudy cornea, diaphragmatic defects and distal limb deformities. *Hum Genet* 1979; **50**: 65-70.
- 3 Lubinsky M, Severn C, Rapoport JM. Fryns syndrome: a new variable multiple congenital anomaly (MCA) syndrome. *Am J Med Genet* 1983; **14**: 461-6.
- 4 Young ID, Simpson K, Winter RM. A case of Fryns syndrome. *J Med Genet* 1986; **23**: 82-8.
- 5 Meinecke P, Fryns JP. The Fryns syndrome: diaphragmatic defects, craniofacial dysmorphism, and distal digital hypoplasia. *Clin Genet* 1985; **28**: 516-20.