but collectively they are significant and hence appropriate investigations should be considered. It is particularly important to consider IMD where more than one case of hydrops has occurred in the same family or a history of spontaneous abortion or unexplained deaths in close family members. We would like to describe our protocol for the investigation of fetal and neonatal hydrops for IMD. Investigations are considered when the more common causes of hydrops have been excluded.

From the dead fetus a skin biopsy can be taken, and this should be taken as soon as possible. The cultured cells can be assayed for β-galactosidase (GM1 gangliosidosis, Morquio’s disease type B, galactosialidosis), β-glucuronidase (Sty’s disease), β-glucosidase (Gaucher’s disease type A), α-n-acetylglucosaminidase-6-sulphate sulphatase (Morquio’s disease type A), α-sialic acid (asialic aciduria) and cholesteral esterification (Niemann-Pick C). DNA from the cultured cells can be investigated for expanded trinucleotide repeat sequence found in myotonic dystrophy.

In cases where hydrops has been diagnosed antenatally amniotic fluid can be tested for β-thalassemia, and this should be taken as soon as possible. The cultured cells can be assayed for α-thalassemia disorders. Other assays can be undertaken on cultured amniotic fluid cells or chorionic villus tissue (direct or cultured). In the live neonate investigations which can be performed on blood include α-thalassemia, β-thalassemia, α-oligosaccharide and α-fetoprotein (neonatal enzyme deficiency). Cultured skin fibroblasts can be investigated for the presence of mycosaceous enzymes, α-mannosidase, α-glucosidase, α-fucosidase, α-iduronidase, α-neuraminidase and α-arylsulphatase activities.

Do maternal β-sympathomimetics influence the development of retinopathy in the premature infant?

EDITOR,—Recent studies suggest that a number of factors other than the injudicious delivery of oxygen contribute to the development of retinopathy in the premature infant.1,2 One hundred and fifty infants on our neonatal unit who required oxygen treatment for more than 60 days were reviewed retrospectively in order to determine the range of factors contributing to this pathologic.

All infants requiring oxygen for more than two weeks were examined at two weekly intervals by an ophthalmic surgeon (ES). Fourteen cases of retinopathy greater than stage 3 were identified. These patients were matched for gestation (±1 week), birth weight (±50 g), sex, and ethnic group with infants who had oxygen requirements for more than 60 days, but did not have retinopathy. The two sets of cases showed no significant differences in their Apgar scores, requirement for exchange transfusions, or the incidence of clinically significant patent ductus arteriosus.

Mothers of eight of the retinopathy group had received infusions of β-sympathomimetics (seven ritodrine, one salbutamol) to arrest premature labour: only one of the non-retinopathy group had received ritodrine (p=0.008). This observation may be of physiological importance, as animal experiments have suggested that the retinal circulation is strongly influenced by these pharmacological agents acting jointly through the sympathetic nervous system and endothelial factors.3 β-Sympathomimetics are known to induce transient ischaemic changes in the myocardium of the newborn if given over long periods by intravenous infusion.4 Maternal ritodrine has been associated with reduced blood pressure in the newborn.5,6 In the premature infant in whom antenatal retinal perfusion is influenced by β-sympathomimetics, the retinal circulation may therefore be a predisposition to damage by other factors, including inappropriate oxygen tensions. This combination of insults to retinal perfusion could lead to the development of ischaemia, and predispose to retinopathy of prematurity. This observation merits review in a larger series of infants. Further, it casts doubt as to the safety of β-sympathomimetics which are widely used in the therapy of premature labour with little evidence to support their efficacy.

CA MICHIE S BRAITHWAITE E SCHULENBERG D HARVEY
Department of Paediatrics and Neonatal Medicine, Royal Postgraduate Medical School, Hammersmith Hospital, London W12 0NN


placental factors and other causes of adverse perinatal outcome (see table). Apgar scores are a convenient and easy method of assessing the health of the newborn, but there is evidence that they are not a good indicator of longer-term outcome.7,8

Despite a period of three months (November 1993 to January 1994) 12 babies had C reactive protein estimations before and after immunisation. The median value preimmunisation was 4 mg/l (range 0.7–8.2 mg/l), and 48 hours post-immunisation 3 mg/l (range 1.5–15 mg/l). The median duration of increased C reactive protein was two days. During the initial period of the study one of the babies had a full infection screen based on the raised C reactive protein and clinical findings (systemically unwell, postimmunisation).

Plasma C reactive protein is known to increase after immunisation with diphtheria, pertussis, and tetanus vaccines in malnourished neonates.8,9 To the best of our knowledge it has not been reported in premature infants who have been immunised. It is important to realise that immunisation leads to an increase in the levels of C reactive protein concentrations so that unnecessary investigations may be avoided.


Diagnosis and management of non-immune hydrops in the newborn

EDITOR,—The article by Stephenson and colleagues concerning the diagnosis and management of non-immune fetal hydrops was both informative and comprehensive. The number of reported associations was particularly thorough.1 I would like to make a minor point regarding the inclusion of asplenia syndrome among the cardiovascular associations.

Asplenia syndrome (right atrial isomerism) is characterised by complex cardiac defects including primitive atrioventricular septal defects and pulmonary stenosis, of which are listed in Stephenson’s table.1 Nevertheless, it is far more common to see polysplenia syndrome (left atrial isomerism) in association with fetal hydrops.2,3 Left atrial isomerism is well associated with the common atrioventricular block, as well as other structural cardiac lesions.4 Complete heart block can occur, rarely, in right atrial isomerism, as in one case in Schmidt’s study.
(v 17 cases with left atrial isomerism). It is thought that the fetal bradycardia and/or structural cardiac lesions contribute to low cardiac output and consequent hydrops, with a dismal prognosis.5–9

After browsing the literature, I remain perplexed that right atrial isomerism is detected so infrequently in utero, given the typical severity of its lesions. However, the current data from fetal echocardiography centres indicate that left atrial isomerism accounts for many more cases of fetal hydrops than right atrial isomerism. Apparently, it is the combination of heart block with structural cardiac defects which puts the fetus at high risk for hydrops, as the prognosis is much better for heart block without structural abnormalities.3,4

COLIN K PHOON
Division of Pediatric Cardiology, University of California Medical Center, San Francisco, CA 94143-0544, USA


Cerebral autoregulation of preterm neonates – a non-linear control system?

EDITOR.—We read with interest the detailed paper of Zernikow et al on cerebral auto-regulation of preterm neonates.1 However we would caution against the use of the term autoregulation. This term implies maintaining a constant cerebral perfusion in the face of a changing cerebral perfusion pressure.2 The slow wave cycles we, and others, have described in the cerebral blood flow velocity values occur independently of blood pressure and are indices of velocity and not flow.3 They undoubtedly reflect some underlying physiological control but it would be incorrect to describe this as autoregulation.

M Y ANTHONY
John Radcliffe Hospital, Oxford OX3 9DU


Professor Jorch and coauthors comment:

We use the term ‘autoregulation’ in a more general meaning to describe the autonomous regulation of cerebral haemodynamics. It addresses the complexity of regulation, is not limited to the mean arterial blood pressure–cerebral blood flow autoregulation relationship, and the regulatory result is not necessarily the constancy of cerebral blood flow.1 If this was not clear from our paper, we thank Dr Anthony and Professor Levene for their comment.

Diagnosis and management of non-immune hydrops in the newborn

Colin K Phoon

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