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Diagnosis of non-immune hydrops in the newborn

EDITOR,—Stephenson *et al* provide a helpful 'personal practice' article in relation to non-immune hydrops of the newborn and a near comprehensive list of reported associations.¹ With such heterogeneity in the causes and associations of non-immune hydrops it is perhaps inevitable that the list is not absolutely complete and I write to highlight one potentially important omission, namely congenital myotonic dystrophy (CMD). Stratton and Patterson recently confirmed this diagnosis by DNA mutation analysis in a case of non-immune hydrops and provide a good literature review of hydropic infants born to mothers with myotonic dystrophy, a total of 16 cases including their own.² Such numbers, although only a minority of those with CMD, suggest a clear cause and effect relationship rather than a chance association. The precise pathophysiology is undetermined but myotonic dystrophy is a multisystem disorder with cardiac muscle and conduction pathways significantly affected in a proportion of patients. There is good evidence that earlier onset, more severe forms of myotonic dystrophy will fair worse in this respect. The

youngest case personally known to me who required a pacemaker did so at 16 years of age. It is reasonable to postulate that non-immune hydrops in CMD may result from unusually severe cardiac muscle involvement with intrauterine heart failure – but this is speculation at present. As Stratton and Patterson point out,² in unexplained non-immune hydrops an examination of the mother and a detailed family history may provide the most important clues. The inheritance of myotonic dystrophy is autosomal dominant and the phenomenon of 'anticipation' is observed, that is, the age of onset of symptoms is earlier, and the severity of most features increased, with succeeding generations. It is virtually always the case that the affected parent of a baby with CMD is the mother and she can be expected to show at least some clinical signs of the disorder. However, the diagnosis must be considered first. Once CMD has occurred in one pregnancy it is highly likely that the next fetus to inherit the myotonic dystrophy gene will also manifest the congenital form. Of course, once a diagnosis of CMD/myotonic dystrophy has been made then genetic counselling and testing can be offered to other family members at risk.

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Patent ductus arteriosus in the newborn

EDITOR,—In situations and places where injectable indomethacin is unavailable, oral indomethacin has been used for closure of the patent ductus arteriosus (PDA). It is, however, extremely difficult to fractionate accurately 25 mg of the capsule (powder) into 0.2 mg sachets or doses, especially when sensitive weighing scales may also not be available. We noted that oral indomethacin powder does not disperse easily in water or other liquid vehicles. We have hence used

mefenamic acid, another non-steroidal anti-inflammatory drug, as an alternative for treatment of symptomatic PDA.^{1,2} Mefenamic acid (Ponstan, Parke-Davis 50 mg/5 ml) was administered orally in three doses of 2 mg/kg/dose at 12 hourly intervals in 25 neonates with symptomatic PDA.³ The mean gestation age of these neonates was 30 weeks, mean birth weight 1320 g, and mean age of administration of mefenamic acid was 16 days. Twenty neonates had pretherapeutic two dimensional echocardiography and Doppler flow studies performed to confirm the diagnosis of PDA and to rule out a ductus dependent state. Twenty two patients (88%) clinically responded to this therapeutic regimen within 48 hours of administration of the last dose of mefenamic acid. Of three non-responders one was subsequently diagnosed to have an endocardial cushion defect and the other two were then administered three doses oral indomethacin (0.25 mg/kg dose) with no therapeutic response.

Our earlier experience with the use of oral indomethacin in 32 preterms of mean gestational age 31 weeks indicated a PDA closure rate clinically of 75% in those treated with indomethacin.

Our initial experience with mefenamic acid treatment, although restricted to 25 cases, suggests that it is effective, safe, well tolerated, and certainly easier to administer than oral indomethacin. In situations where injectable indomethacin is not available, we suggest that mefenamic acid be used given its efficacy and ease of administration of accurate dosage. Further trials need to be conducted in larger number of cases to confirm our observations.

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