In conclusion, parents are not present any more during ward rounds. However, we have not formally evaluated this approach. Parents are now formally informed about major issues after the main ward round by the consultants, in the presence of the doctor on duty. On minor issues, doctors and nurses inform the parents while they visit their baby.

H D Dellagrammaticas, N Lacovidou
Department of Paediatrics, University of Athens, Greece

Correspondence to: H D Dellagrammaticas, NICU, 2nd Department of Paediatrics, University of Athens, Aglaia Kyriakou Children's Hospital, 11527 Athens, Greece; hdellagr@ath.forthnet.gr

Competing interests: None declared.

Reference

1 **Bramwell R**, Weindling M. for the FVWR Research Team. Families' views on ward rounds in neonatal units. *Arch Dis Child Fetal Neonatal Ed* 2005;**90**:F429–31.

Prolonged hyperinsulinaemic hypoglycaemia in newborns with intrauterine growth retardation

Intrauterine growth retardation (IUGR) is an important cause of neonatal mortality, morbidity and poor neurological outcome.[1] Hypoglycaemia as a consequence of IUGR is a major risk factor for neurodevelopmental impairment. The factors that predispose these patients to hypoglycaemia include failure of counter-regulation, immaturity of the enzyme systems regulating glycogenolysis, gluconeogenesis, ketogenesis, reduced adipose tissue stores, hyperinsulinism or increased sensitivity to insulin.[2–4] Hypoglycaemia in patients with IUGR is usually thought to be transient, lasting for a few days. However, the precise duration of transient hyperinsulinaemic hypoglycaemia is unclear.

We report on our experience of prolonged hypofattyacidaemic hypoketotic hyperinsulinaemic hypoglycaemia (between 3 and 9 months' duration) in 20 infants with symmetrical IUGR who required treatment with diazoxide and chlorothiazide. A total of 20 consecutive patients referred to the London Centre for Paediatric Endocrinology and Metabolism, Great Ormond Street Children's Hospital NHS Trust, London, UK, with symmetrical IUGR (birth weight <3rd centile) and persistent hypoglycaemia (hypoglycaemia persisting for >10 days at the referring hospital) were recruited into the study. The mean birth weight and mean gestational age of the whole cohort were 2.1 kg and 38 weeks, respectively.

In each patient, the hypoglycaemia was characterised by inappropriate insulin secretion, increased glucose clearance (>10 mg/kg/min), blunting of the serum cortisol and glucagon counter-regulatory hormonal responses, and resistance to growth hormone as shown by low serum levels of insulin-like growth factor 1 and insulin-like growth factor-binding protein 3 and raised serum levels of growth hormone.

All patients required treatment with diazoxide (5–10 mg/kg/day) and chlorothiazide (7–10 mg/kg/day) to correct their hypoglycaemia. The mean age for starting diazoxide was 18 days. Each of these patients was then followed up at 3-monthly intervals to assess their response to diazoxide withdrawal. In 10 patients, administration of diazoxide and chlorothiazide was stopped at age 3 months, in seven patients at 6 months and in the remaining three patients at 9 months.

In summary, some infants with IUGR may continue to have hypofattyacidaemic hypoketotic hyperinsulinaemic hypoglycaemia beyond the first a few weeks of life. As this hypoglycaemia is associated with a lack of alternative substrates for the brain to use (such as ketone bodies), recognition and treatment of this group of patients is important. If unrecognised, this may have important implications for neurodevelopmental outcome of these patients. Further studies are needed to understand the underlying mechanism of these observations.

O Fafoula, H Alkhayyat, K Hussain
London Centre for Paediatric Endocrinology and Metabolism, Great Ormond Street Children's Hospital NHS Trust, London, UK

Correspondence to: K Hussain, Unit of Biochemistry, Endocrinology and Metabolism, Institute of Child Health, University College London, 30 Guilford Street, London WC1N 1EH, UK; K.Hussain@ich.ucl.ac.uk

doi: 10.1136/adc.2006.095919

Funding: Research at the Institute of Child Health and Great Ormond Street Hospital for Children NHS Trust benefits from R&D funding received from the NHS Executive.

Competing interests: None declared.

References

1 **Aucott SW**, Donohue PK, Northington FJ. Increased morbidity in severe early intrauterine growth restriction. *J Perinatol* 2004;**24**:435–40.
2 **Hawdon JM**, Weddell A, Aynsley-Green A, *et al*. Hormonal and metabolic response to hypoglycaemia in small for gestational age infants. *Arch Dis Child* 1993;**68**:269–73.
3 **Collins JE**, Leonard JV, Teale D, *et al*. Hyperinsulinaemic hypoglycaemia in small for dates babies. *Arch Dis Child* 1990;**65**:1118–20.
4 **Hussain K**, Aynsley-Green A. The effect of prematurity and intrauterine growth restriction on glucose metabolism in the newborn. *Neoreviews* 2004;**5**:e365–9.

Neonatal pure red cell aplasia due to anti-M

Haemolytic disease of the newborn (HDN) is caused by maternal immunoglobulin (Ig) G acting against antigens expressed on mature fetal red cells. Anti-M antibodies active at 37°C are a rare cause of HDN.[1] We report data, arising from the investigation of a neonate with severe transient pure red-cell aplasia, indicating that anti-M can cause hypoplastic HDN by inhibition of erythroid precursor growth, as frequently occurs with anti-Kell antibodies.[2]

A female neonate presented at 4 weeks of age with severe hypoplastic anaemia (haemoglobin 3.7 g/dl, reticulocytes 11×10^9/l) and mild jaundice (bilirubin 30 μmol/l). Direct anti-globulin test was negative. A bone marrow aspirate showed reduced but morphologically normal erythroid precursors. The patient was transfused twice before spontaneous increases in reticulocyte and haemoglobin values were noted at 74 days of age, the haemoglobin value reaching and remaining within normal limits until discharge from follow-up at 15 months.

The patient's blood group was O M+N+ and that of her mother A M−N+. At presentation, maternal serum contained anti-M with both IgM and IgG components.

In view of this finding, experiments were carried out to determine the role of anti-M in causing anaemia. With local research ethics committee approval and parental consent, marrow mononuclear cells from the patient and group O controls (ie, children with acute lymphoblastic anaemia in remission, where excess sample was available at a scheduled marrow examination) were cultured by standard assay (Methocult System, Stem Cell Technologies, Vancouver, Canada) and in the presence of inert serum (from the child's father) and dilutions of maternal serum. On the basis of 95% ranges derived from repeated analysis of two samples (data not shown), reductions in erythroid colony numbers (burst-forming and colony-forming units–erythroid combined) in the patient and in controls 2 and 3 with 10% and control 3 with 1% maternal serum were considered significant. No inhibition was seen in a patient with the NN phenotype (table 1). In experiments with absorbed maternal serum, the considerable inhibition of erythroid growth was abolished in one of two samples by prior absorption with group OM+N− cells but not by OM−N+ cells (data not shown).

The M antigen is expressed on immature erythroid precursors[3] and it is plausible that precursor cell growth would be inhibited by anti-M. Our patient's clinical picture and our in vitro data provide strong circumstantial evidence that anti-M should join anti-Kell as a cause of reticulocytopenic HDN.

R F Hinchliffe, B Nolan, A J Vora
Department of Paediatric Haematology, Sheffield Children's NHS Trust, Sheffield, UK

R Stamps
National Blood Service, Sheffield

Table 1 Erythroid precursor colony numbers (per 5×10^4 nucleated cells) in patient and control bone marrow samples and the effects of both neutral serum and maternal serum (containing anti-M) at two dilutions

Subject	Type	Standard assay	+10% inert serum	+10% maternal serum	+1% maternal serum
Patient	MN	39	43	10 (−74)	NT
Control 1	MN	46	44	30 (−35)	44
Control 2	MN	49	40	21 (−57)	36 (−27)
Control 3	MM	63	73	32 (−49)	43 (−32)
Control 4	NN	21	20	26 (+24)	31 (+48)

NT, not tested.
Figures in brackets indicate the percentage change from the value obtained with the standard assay, where this was >20%.