Archives of Disease in Childhood, 1989, **64**, 270–282

Short reports

Early hyperkalaemia in very low birthweight infants in the absence of oliguria

L P BRION*, G J SCHWARTZ,† D CAMPBELL,* AND A R FLEISCHMAN*

*Department of Pediatrics, Divisions of *Neonatalogy and †Nephrology, Albert Einstein College of Medicine, Bronx, New York, USA*

SUMMARY We reviewed 1552 admissions to a neonatal intensive care programme; seven, all with a birth weight <1500 g, developed early onset, non-oliguric hyperkalaemia (potassium concentration >7·0 mmol/l). Although their perinatal variables were similar to those of a normokalaemic group, hyperkalaemic infants had a higher incidence of intraventricular haemorrhage and developed increased concentrations of plasma creatinine by 7 days of age.

Hyperkalaemia in prematurity has been recognised for many years as a serious problem with important consequences.[1] Although hyperkalaemia is a frequent complication of acute oliguric renal failure, it is much less commonly found in infants with mild renal impairment and normal urine output.[2] This study reviews the incidence of hyperkalaemia in the first 3 days of life in very low birthweight infants in the absence of oliguria and acute renal failure.

Patients and methods

We reviewed the charts of 1552 neonates admitted over a period of 17 months (1 July 1983 to 30 November 1984) to our regional neonatal intensive care programme. Only patients who developed hyperkalaemia (potassium concentration of >7·0 mmol/l on two non-haemolysed samples) during the first 72 hours of life in the presence of a urine output of >1·0 ml/kg/hour were included for study. We then created a retrospective normokalaemic control group by matching each patient with an infant of similar birth weight. All infants had serum electrolyte concentrations measured at least at 24, 48, and 72 hours of age. All patients with a birth weight <1500 g or a gestational age <35 weeks had ultrasonographic examinations of the head performed during the first week of life. Data are presented as mean (SE).

Results

Seven infants developed early non-oliguric hyperkalaemia, an incidence of 3·2% (7/221) and 4·4% (6/135) for <1500 g and <1250 g birthweight infants respectively. None had received potassium before hyperkalaemia was diagnosed at 9–70 hours of age, although five out of seven infants had received a packed cell transfusion with less than seven day old cells. The electrocardiogram was abnormal in five out of seven patients including prolongation of the QRS and PR interval, ST depression or peaked T waves, or both; three patients had ventricular tachycardia. All patients received potassium exchange resin enemas and combinations of glucose, insulin, bicarbonate, and calcium. In two patients the arrhythmia resolved only after administration of lignocaine or bretylium tosylate or both. Hyperkalaemia persisted for a period of one to seven days. Mean potassium concentration in the urine was 34 (12·5) mmol/l (n=4) and mean fractional excretion of potassium was 62 (22)%. Fractional excretion of sodium was 12·4 (6·5)% and the urine sodium: potassium ratio was 9·4 (7·6). Although the initial biochemical values and urine output were similar in hyperkalaemic patients and in controls, four hyperkalaemic infants developed a plasma creatinine concentration >132·6 μmol/l during the first week of life, compared with none in the control group ($p<0·001$). The peak plasma creatinine concentration in the hyperkalaemic patients resulted in a lower estimated glomerular filtration rate[3] than found at comparable postnatal age in the controls (6·9 (0·7) compared with 10·7 (0·9) ml/min/1·73m^2, $p<0·05$, Student's t test) and than in previously reported premature infants with gestational age of 25–32 weeks (13·9 (0·9) ml/min/1·73 m^2, n=26, $p<0·01$).[3]

The weight loss from birth to the time of onset of hyperkalaemia was similar in hyperkalaemic patients and in controls (4·6 (1·8)% compared with 3·8 (2·0)%). Although perinatal variables appeared

Table *Hyperkalaemic neonates compared with matched controls. Results are mean (SE) and range except for Apgar scores which are median (range)*

	Hyperkalaemic neonates (n=7)		*Control group (n=7)*	
Gestational age (weeks)	27·4 (0·9)	25–32	27·7 (0·5)	26–29
Birth weight (g)	936 (109)	620–1450	935 (84)	710–1360
Apgar score at 1 minute	5	1–8	3	1–7
Apgar score at 5 minutes	6	2–10	6	1–9
Plasma concentrations of:				
Sodium (mmol/l)	144 (3)	131–158	142 (2)	132–146
Potassium (mmol/l)	8·8 (0·5)†	7·2–10·4	5·2 (0·3)	4·3–6·3
Bicarbonate (mmol/l)	19 (1)	16–22	20 (2)	17–23
Creatinine (μmol/l)	132·6 (17·7)	97·2–229·8	106·1 (4·7)	79·6–123·8
Blood urea nitrogen (mmol/l)	8·9 (1·8)	2·5–15·7	7·1 (0·7)	3·2–9·3
Urine output (ml/kg/hour)‡	2·7 (0·7)	1·2–4·8	2·2 (0·5)	1·2–4·7
Estimated glomerular filtration rate (ml/min/1·73 m^2)§	9·0 (1·1)	4·7–14·0	11·2 (0·7)	9·4–14·3
Maximum plasma concentrations of:				
Potassium (mmol/l)	9·4 (0·6)†	8·0–11·2	5·9 (0·3)	4·4–6·9
Creatinine (μmol/l)	168·0 (26·5)	106·1–327·1	107·6 (8·4)*	79·6–132·6
Estimated glomerular filtration rate (at maximum creatinine concentration) (ml/min/1·73 m^2)	6·9 (0·7)**	3·3–8·6	10·7 (0·9)	8·1–14·7
Increase in plasma creatinine concentration during first week of life (μmol/l)	44·2 (17·7)	+8·8 to +97·2	4·4 (9·4)*	−26·5 to +44·2

Laboratory values were obtained for each pair of infants at the same postnatal age—that is at the time of diagnosis of the hyperkalaemic patients for the initial values and at the peak value during the first week of life for the maximum plasma creatinine and potassium concentrations.
Results different from controls by unpaired *t* test:†$p<0·05$ and **$p<0·01$.
‡Urine output at time of entry to study; *n=6.
§Estimated glomerular filtration rate (GFR) is calculated from the formula: 0·29×length (cm) ÷ plasma creatinine concentration (μmol/l).

to be similar in the hyperkalaemic and in control patients, the incidence of intraventricular haemorrhage was six out of seven in the hyperkalaemic group, as compared with one out of seven in the control group ($p<0·05$, Fisher's exact test). Four patients in the hyperkalaemic group died subsequently after becoming normokalaemic, two from respiratory failure and two from necrotising enterocolitis, whereas no patients died in the control group.

Details of both groups of patients are shown in the table.

Discussion

Hyperkalaemia may result from increased potassium load or decreased renal potassium excretion, or both. Factors in low birthweight infants that may contribute to increased load include endogenous potassium from perinatal asphyxia, tissue necrosis, extravasated blood (including intraventricular haemorrhage), acidosis and starvation, and increased exogenous load from blood transfusion. Decreased renal potassium excretion may result from a combination of immaturity of tubular function as well as renal damage and poor response to aldosterone.[4–6]

The incidence of early hyperkalaemia in low birthweight infants in our series was similar to that reported in the literature.[5] Whether intraventricular haemorrhage or periventricular leucomalacia, or both, is due to hypotension and cerebral vascular spasm resulting from hyperkalaemia,[5 6] or hyperkalaemia is caused by intraventricular haemorrhage cannot be concluded from this study. Some patients in our study lacked any obvious predisposing factors at the time of diagnosis.

There appear to be no clear clinical indicators to predict which low birthweight infants will develop hyperkalaemia. Therefore we recommend that serum potassium concentrations be checked every six to eight hours in the very low birthweight infant during the first days of life.

References

[1] Usher R. Respiratory distress syndrome in prematurity. I.

Changes in potassium in the serum and the electrocardiogram and the effect of therapy. *Pediatrics* 1959;**25**:562–76.
[2] Grylack L, Medani C, Hultzen C, *et al.* Non-oliguric renal failure in the newborn. A prospective evaluation of diagnostic indices. *Am J Dis Child* 1982;**136**:518–20.
[3] Brion LP, Fleischman AR, McCarton C, Schwartz GJ. A simple estimate of glomerular filtration rate in low birth weight infants during the first year of life: non-invasive assessment of body composition and growth. *J Pediatr* 1986;**109**:698–707.
[4] Gruskay J, Costarino AT, Polin RA, Baumgart S. Non-oliguric hyperkalemia in the premature infant weighing less than 1000 grams. *J Pediatr* 1988;**113**:381–6.
[5] Shortland D, Trounce JQ, Levene MI. Hyperkalaemia, cardiac arrhythmias, and cerebral lesions in high risk neonates. *Arch Dis Child* 1987;**62**:1139–43.
[6] Edvinsson L, Lou HC, Tucde K. On the pathogenesis of regional cerebral ischemia in intracranial hemorrhage: a causal influence of potassium? *Pediatr Res* 1986;**20**:478–80.

Correspondence to Dr A R Fleischman, Albert Einstein College of Medicine, Jacobi Hospital, Room 8S15, Pelham Parkway South, Bronx, New York 10461, USA.

Accepted 26 September 1988

Twinning rates and social class in Great Britain

M MURPHY AND B BOTTING*

*Department of Community Medicine and General Practice, Radcliffe Infirmary, Oxford and *Medical Statistics Division, Office of Population Censuses and Surveys, London*

SUMMARY We examined like and unlike sex twinning rates in Great Britain by social class over the period 1974–85. Although twinning rates are believed to have changed over that period, we found no evidence of differential change by social class, suggesting that any factors affecting twinning are widespread in the population.

A secular decline in twinning rates from the early 1950s until the 1970s has been observed almost worldwide, largely confined to dizygotic twinning. In Great Britain, as elsewhere, the dizygotic twinning rate may now have plateaued, with monozygotic twinning stable throughout or latterly increasing. While demographic change may account for part of these trends, the phenomenon is poorly understood. A number of environmental factors have been proposed, including changing patterns of use of ovulatory stimulants and the contraceptive pill and dietary exposures to pesticides or stilboestrol.[1 2] As exposure to these agents may well have varied in different socioeconomic groups, we examined twinning rates in Great Britain, 1974–85, for any evidence of differential changes by social class.

Methods and results

We obtained numbers of like sexed and unlike sexed live and stillborn twins and total confinements 1974–85 for England and Wales from the Office of Population Censuses and Surveys (OPCS) and for Scotland from the General Register Office (GRO(S)). The data were available by father's social class for legitimate births only in Scotland, but it was possible to include jointly registered illegitimate births as well for England and Wales. This allows a larger number of births to be analysed by social class in a consistent way over a period of time when the rising number of illegitimate births raises important questions of selection bias if they are excluded from social class analysis.[3]

The figure shows 1974–85 trends in like sexed, unlike sexed, and total twinning rates for England and Wales by aggregated social class, indirectly standardised for maternal age in each case using the twin rates for 1974–85 as standard. Data for 1981 for England and Wales are not available. There is little evidence of a difference in the trends in twinning rates between the non-manual and manual social classes, though twinning rates are higher in the manual group. Confidence intervals around the regression coefficients for each of the three pairs of time trends confirm this (table). Although not shown here, analysis by less aggregated social class groups (I and II, III, IV and V) showed similar patterns in both England and Wales and Scotland, though with a possibly more pronounced increase in like sexed and total twinning rates in the 1980s in social class III and the combined social classes IV and V particularly in Scotland.

Discussion

Discontinuities in the social class assignment of births because of change in the Registrar General's classification in 1979 are small,[4] and statistical fluctuations due to small numbers of events will also have been minimised by restricting the analysis to