Antenatally diagnosed renal pelvis dilatation

EDITOR,—We were interested in the findings of Jaswon et al regarding the outcome of babies with antenatally detected renal pelvis dilatation (ARPD).1 Since October 1997 it has been the practice in our unit to perform micturating cysto-urethrogramography (MCUG) on all babies with ARPD, defined as a renal anteroposterior renal pelvis diameter of greater than 5 mm, even in the presence of a normal six week postnatal ultrasound scan of the renal tract.2

Over the 16 months since this practice has been established we identified 29 fetuses with ARPD from 3397 total deliveries (0.9%). Twenty four of 29 (83%) had ARPD of 5–10 mm, and five of 29 had ARPD of greater than 10 mm. Fifty five per cent were boys, 34% girls, and the sex was not identified in three fetuses who were lost to follow up. Postnatal follow up data were available for 18 of 29 babies. Postnatal abnormalities were detected in 13 of 18 (72%) of the babies. Six of 13 (46%) had vesico-uretrectal reflux (VUR). Other diagnoses were mild unilateral pelvi-ureteric junction obstruction (n=2), posterior urethral valves (n=1), duplex kidney with obstructed upper moiety requiring heminephrectomy (n=1), bladder diverticulum without ureteric obstruction (n=1), and idiopathic hydronephrosis (n=2).

Of the six babies with VUR, four had ARPD of 5–10 mm, and two had ARPD greater than 10 mm. One baby had normal postnatal ultrasound scans (performed at 3 days, and at 6 weeks of age) with bilateral grade II VUR on MCUG. One baby with reflux had focal uptake defects on dimercaptosuccinic acid (DMSA) scans but no high uptake defects on DMSA, which we and others2 have reported higher levels of malondialdehyde (MDA) in cord blood at birth: the e


How phototherapy affects the relation between serum bilirubin and plasma malondialdehyde in neonates

EDITOR,—The study by Yigit et al showed that serum malondialdehyde (MDA) concentrations were higher in infants with hyperbilirubinaemia than in controls, but there was no significant correlation between serum MDA and bilirubin concentrations in jaundiced neonates with hyperbilirubinaemia. The results of our study confirmed those of Yigit et al, but we also investigated whether treating neonates with phototherapy increases the risk of oxygen free radical injury. We evaluated plasma MDA which is an index of free radical induced lipid peroxidation, and serum bilirubin concentrations, in blood samples taken from healthy, term neonates with non-haemolytic hyperbilirubinaemia (n=19). Total bilirubin > 15.0 mg/dl before and after phototherapy. These were compared with those taken from healthy neonates without hyperbilirubinaemia (n=22), total bilirubin < 1.0 mg/dl at birthweight, gestational age, and postnatal age did not differ between the study and control groups (p > 0.05). All babies were fed their mothers’ milk. MDA concentrations were determined using the modified method of Stocks and Dornmand3 with the thiobarbituric acid (TBA) test.

Plasma MDA concentrations in neonates with jaundice (0.99 (SEM 0.15) nmol/ml) were significantly higher than those of the healthy infants (0.89 (0.16) nmol/ml) (p=0.038). There was a significant difference between the MDA concentrations of the study group before and after phototherapy (0.99 (0.15) vs 0.85 (0.20) nmol/ml; p=0.0016). However, no significant correlation was found between plasma MDA and serum bilirubin concentrations before and after phototherapy (r=0.16, p > 0.05; r=0.09, p > 0.05).

Bilirubin is an important free radical scavenger in early neonatal life in response to oxidative stress. Phototherapy reduces the concentrations of circulating bilirubin, but there are conflicting data on red cell membrane lipid peroxidation secondary to phototherapy.1 4 Our data suggest that phototherapy does not induce lipid peroxidation in healthy term infants with non-haemolytic hyperbilirubinaemia.

REFERENCES
hours, beyond that period there was no significant effect of delivery route on MDA levels. In our study published in this journal we again found no significant difference in MDA levels according to mode of delivery. We are not sure what Dr Manzar means by “the normal range”. Our control group gives the norm for this population under this condition. Only a large scale study would determine more precisely the normal range of MDA in infants up to 10 days old. His final query reflects the fact that Dr Manzar has missed a key point about MDA and bilirubin. All human beings, not just neonates, have a detectable level of MDA. Our study is the first to show that MDA levels are higher in babies with hyperbilirubinaemia than in normal infants.

Furthermore, we investigated the difference between babies with and without haemolytic jaundice. Only those with haemolytic jaundice showed a positive correlation between MDA and bilirubin levels. This difference is clearly illustrated in the figures in our paper, as well as explained in the third paragraph of the results section.


Pulmonary hypoplasia: alternative pathogenesis and antenatal therapy in diaphragmatic hernia

Editor,—We wish to comment on some aspects of the important problem raised by Porter et al relating to congenital diaphragmatic hernia (CDH).

Pulmonary hypoplasia in CDH may not be attributable to the diaphragmatic defect and visceral herniation alone. There is evidence from CDH models to indicate that, before the established hernia, lung development is abnormal from the outset in the embryonic period. This has substantial implications for the type and timing of potential treatments.

Intrauterine tracheal occlusion for CDH is the subject of a US National Institutes of Health clinical trial (Harrison MR, personal communication). Numerous studies in experimental CDH have shown that although antenatal glucocorticoid treatment does not correct overall lung size, it improves structural, biochemical, molecular and functional abnormalities in the hypoplastic lung. As a potential pharmacological fetal treatment, antenatal steroids and bilirubin levels. This di...
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