Our data confirm the findings of Stevenson et al.: iCT concentrations are raised throughout pregnancy. In at-risk pregnancies iCT levels were not altered, although a statistically non-significant tendency toward higher levels in EPH-gestosis was observed. The striking increase in iCT concentrations in cord blood (50–300 pg/ml) over the corresponding levels in the mother before delivery (undetectable to 170 pg/ml) indicates an important physiological function for calcitonin in the newborn. This seems to be remarkable in view of the altered vitamin-D metabolism in vitro of the fetoplacental unit. 1,2,125-DHCC values are raised in pregnant women but low in fetal tissue, suggesting that in the newborn the physiological role of calcitonin is greater than that of protection against the bone-resorbing action of 1,25-DHCC.

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RENAL MEDULLARY HÆMMORRHAGE AND ECHOVIRUS INFECTION

SIR,—If renal medullary haemorrhage is seen at post mortem in a neonate, echovirus infection should be considered. Nagy et al. 1 described three fatal cases of echovirus 11 infection in which renal medullary haemorrhage was seen, and we have now seen two cases, one definite and one speculative.

The first patient was a full-term male infant (birthweight 3060 g) who within 60 h of a rapid uncontrolled delivery had meningitis and features of disseminated intravascular coagulation. He became jaundiced with a bilirubin of 169 mmol/l (normal 3.5–20), alkaline phosphatase 118 IU/l (normal 30–90), and aspartate transaminase 460 IU/l (normal 10–40). He died on the 6th day. Echovirus 11 was cultured from cerebrospinal fluid and from throat and rectal swabs taken before death. The mother had been febrile (38°C) on the morning of delivery and both the father and a 2-year-old sibling had had a “flu-like” illness 2 days previously. Neutralising antibody titre to echovirus 11 in paired sera from the father indicated a recent infection (neutralising titre 4 rising to 128).

Necropsy showed slightly swollen kidneys together weighing 31 g (normal 25 ± SD 5).1 Slicing showed cortical pallor and severe medullary haemorrhage (figs. 1 and 2) and there were occasional petechiae in the pelvicalyceal system. There was no renal vein thrombosis. Other positive findings included thymic petechiae, bilateral adrenal medullary haemorrhage, and a right subdural haemorrhage with bilateral tentorial tears. The liver weighed 107 g (normal 13±3) and histologically showed massive haemorrhagic necrosis. Echovirus 11 was cultured from liver, lung, and heart. Kidney tissue was not cultured.

The clinical course in the second full-term neonate was similar. A septicemia-like illness developed on the fifth day followed by jaundice and bleeding due to disseminated intravascular coagulation (DIC) with death on the seventh day. No pathogenic bacteria were cultured from blood, throat, rectum, or cerebrospinal fluid. No virological cultures were done. Necropsy findings were similar to those in the first case, as were the histological appearances in the kidneys and liver. Electronmicroscopy and immunofluorescence failed to reveal the presence of virus in either case.

Case 1: renal medullary haemorrhage.

Upper: magnification about × 14.
Lower: magnification × 5.

Fatal echovirus 11 infections have been described in neonates,3 and the Cambridge workers plan to publish a larger series. Similar renal changes have been described in the neonatal kidneys of fatal echovirus 64 and echovirus 19 infections. Thomas3 described perinatal haemorrhagic necrosis of renal pyramids in six neonates showing identical macroscopic and microscopic features, and occurring after prolonged labour.

There are five possible methods by which renal medullary haemorrhage could have occurred: outflow obstruction (e.g., renal vein thrombosis), bleeding diathesis (e.g., DIC), effects due to echovirus 11 infection in the kidneys (direct or indirect), hypoxia, and intrarenal shunting of blood. In both these patients there is evidence of DIC and in the first patient disseminated echovirus 11 infection. We suggest that the pathogenesis of the renal medullary haemorrhage to be multifactorial, probably due to a combination of DIC, intrarenal shunting, and the effects of echovirus infection. We suggest that culture for echovirus should be done if these appearances are seen at necropsy.

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