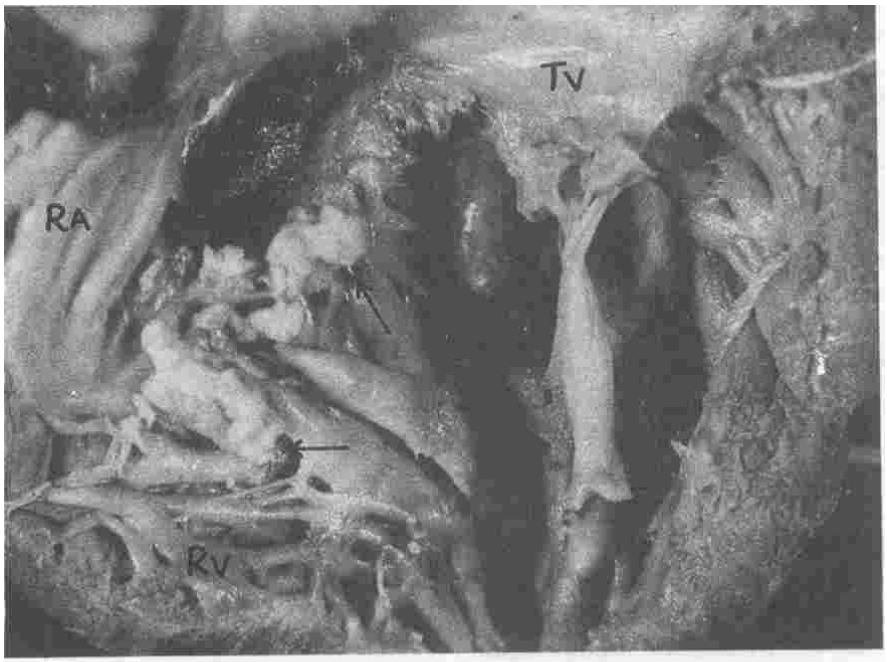

Fig. 1. Tricuspid valve (TV) with nodular white vegetations (arrows) over atrial aspect of the leaflets (RA-Right atrium, RV-Right ventricle).

gestive of persistent fetal circulation clinically or histopathologically

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Renal Infarction Due to Umbilical Artery Catheters

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Umbilical artery catheters allow reliable blood sampling and blood pressure moni-

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toring with minimal handling in critically ill infants, but carry a moderate risk of infarction of the distal extremities and necrotising enterocolitis from thromboembolism(1,2). There is evidence that almost all umbilical artery catheters develop thrombi, but only a minority cause clinical problems(3). Many neonatologists feel their advantages outweigh their risks, and favor the tip being placed in the 'high' thoracic (T_{4-12}) rather than 'low' lumbar (L_{3-5}) position, because there is a lower rate of tip thrombi affecting the legs, though the risk of necrotising enterocolitis is unaltered(l,2).

Clearly, a thrombus dislodged into the renal arteries could cause renal ischemia or infarction, whereas this could not happen with a 'low' catheter unless there was gross retrograde flow during a rapid fluid infusion. Thus, authors considering hypertension secondary to renal artery thromboembolism argue in favor of low placed umbilical artery catheters because most reported cases have occurred with thoracic catheters(4,5). This advice was formulated twenty years ago when antihypertensive drugs were limited and the majority of babies with neonatal hypertension died (4). Considering that hypertension occurs in less than 2% of cases, whereas lower limb compliations are common, and considering medical management of neonatal blood pressure has improved greatly in the last two decades, this may no longer be the best advice.

We describe two infants observed on routine examination to develop hypertension during the second week of life due to renal infarction, both of whom had umbilical artery catheters placed in the high position.

Case Reports

Case 1: An 880 g boy was born by caesarean secrion because his mother developed fulminating pre-eclampsia at 26 weeks gestation. Despite surfactant therapy he required ventilation from birth for eight days because of hyaline membrane disease, and a umbilical artery catheter was placed with its tip at T_8 on day one. His plasma sodium concentration and daily blood pressure measurements were normal until day eight. Thereafter, the plasma sodium fell, and the systolic blood pressure rose; by day twelve they had reached 122 mmol/1, and 108 mm Hg. The hyponatremia was secondary to a high renal salt loss (urinary sodium, 182 mmol/1), necessitating a daily sodium supplement of 18 mmol/kg until the hypertension was controlled. Plasma creatinine concentration, urine microscopy and stick testing for blood, glucose and protein, and urinary vanillyl mandelic acid excretion were all normal, but the plasma renin activity and aldosterone concentration were both elevated at 100 pmol/ml/h, and 4,000 pmol/1. Renal tract ultrasonography was normal, but dimercaptosuccinic acid scanning showed an irregular right kidney that contributed only 43% of the overall function. He still requires regular propranolol at 3.5 years to maintain normal blood pressure.

Case 2: A 3425 g boy was born vaginally at 34 weeks gestation to a 25 year old primigravida with insulin-dependent gestational diabetes, 36 hours after she had a laparotomy for abdominal pain which revealed a fibroid. Despite surfactant therapy he required ventilation from birth for eight days because of hyaline membrane disease, and an umbilical artery catheter was placed with its tip at T_9 on day one. Daily blood pressure monitoring was normal until day ten when it rose to 104 mm Hg systolic, and thereafter he required treatment for hypertension. Plasma creatinine and sodium concentrations were normal, as were

urine microscopy and stick testing for blood, glucose and protein, and urinary vanillyl mandelic acid excretion. Subsequent renal imaging by ultrasonography, dimercapto-succinic acid scan and aortogram revealed a small, irregular, poorly functioning and minimally perfused left kidney. The hypertension remained difficult to control, and subsequent scans demonstrated relative decline in left kidney function; he has been normotensive since a left nephrectomy at fourteen months. Histology showed patches of normal kidney scattered among large areas of infarction.

Discussion

These two babies had a similar clinical course; normal blood pressure was observed throughout the first week, and hypertension detected in the second week, when they had come off ventilatory support. Both had thoracic umbilical artery catheters, and imaging evidence of unilateral kidney damage. We did not perform immediate aortograms, but its seems almost certain that they each had a thrombus that embolised from the catheter tip into one renal artery; spontaneous renal artery emboli are excessively rare, presumably traversing the arterial duct. Also, one case had evidence of renin-driven hypertension, and secondary hyperaldosteronism causing an uncontrolled natriuresis and hyponatremia(6), and the other had reduced renal perfusion and patchy ischemia on histology.

Early control of blood pressure was achieved without difficulty using antihypertensive drugs, many of which were not available twenty years ago. Both had persistent hypertension, treated in one case by propranolol for 3.5 years so far. In the other, drug side effects could not be justified in a child whose kidney had only minimal residual function, and he underwent nephrectomy at fourteen months. The high placement of umbilical artery catheters carries a risk of renal artery embolism, but a reduced risk of harming the legs compared to low placed catheters(l,2). Because the management of neonatal hypertension has become easier, and because leg ischemia is a more frequent complication, catheters should be placed routinely in the high position.

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