useful agent in the management of other children with severe hypertension who are nonresponsive to the usual medical management.

Two important changes in the renin-angiotensin system were observed after treatment with captopril. Captopril lowered plasma aldosterone by blocking the production of angiotensin II, a known stimulator of adrenal aldosterone production. Captopril also increased plasma renin levels, presumably by interrupting the negative feedback loop which controls renin secretion.

Improved creatinine clearance after captopril, which was seen in our patient, has been reported in adults.⁵ The cause of this change is unknown, but renal vasodilatation, decrease in nephrosclerosis, and resolution of the haemolytic syndrome may each have played a role in the patient reported here.

Captopril has several apparent advantages over minoxidil, another agent useful in severe hypertension.⁷ Minoxidil causes pronounced sodium retention which must be aggressively managed with dietary sodium restriction and diuretics. While receiving captopril and frusemide our patient was able to tolerate a high salt diet for treatment of immobilisation hypercalcaemia without adverse effects on blood pressure. The ability to tolerate salt results, in part, from lowered aldosterone levels. The hirsutism caused by minoxidil has limited its usefulness in children.

This patient has had hepatitis throughout the illness. The aetiology of the hepatitis is unknown. The possibility that our patient had an exacerbation of hepatitis due to captopril cannot be completely excluded. However, according to the Squibb monitor,

hepatotoxicity has not been observed in other patients receiving captopril.

Captopril was supplied by Squibb under a humanitarian protocol.

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Oxygen embolus during mechanical ventilation with disappearance of signs after death

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SUMMARY Oxygen embolus is thought to be a rare complication of mechanical ventilation in preterm infants. In the patient described there was clinical and radiological evidence of embolisation within the heart and great vessels, but at necropsy gas was seen only in the cerebral arteries.

Case report

This boy (birthweight 900 g) was the second of

undiagnosed twins delivered by lower segment caesarean section from a woman with severe pre-eclampsia at 29 weeks' gestation. He was transferred to this hospital at 4 hours, receiving artificial ventilation because of idiopathic respiratory distress syndrome (LS ratio 1.5: 1). Despite ventilation at low peak pressure (14/2 cm of water) he developed pulmonary interstitial emphysema followed by a pneumothorax at 26 hours. A chest drain was inserted but he became acidotic and hypoxic, responding to high rates of ventilation as well as to buffer and

tolazoline. When 40 hours old, while ventilated in 100% O₂ at a pressure of 26/2 cm of water, he became bradycardic and hypoxic. Transillumination of the chest failed to show a pneumothorax and there was no improvement in his condition after reintubation. During resuscitation blood was withdrawn from the umbilical artery catheter and this was found to be mixed with gas. Just before death crepitus was detected in the neck, and the skin over the blood vessels of the face and neck became white. An x-ray film at this time (Fig. 1) showed gas in the portal circulation, in the chambers of the heart, and the vessels of the neck. A chest x-ray film taken at the start of the necropsy examination 24 hours later (Fig. 2) showed no evidence of intravascular gas, but examination of the infant showed features of hyaline membrane disease and pulmonary interstitial emphysema, with gas bubbles present only in the anterior cerebral arteries.

Fig. 1 Chest x-ray film at the time of death showing gas outlining the cardiac chambers, with gas in the hepatic arteries as well as in major vessels of the head and neck. Oxygen in the carotid artery is shown by arrow.

Fig. 2 Chest x-ray film taken 24 hours after death.

Discussion

Gas embolism has been described in infants receiving mechanical ventilation.¹⁻⁶ In previous reports pulmonary interstitial emphysema,^{1 2 4 5} or pneumothorax^{1 4} was evident before embolisation occurred. Although many of the infants reported were ventilated at high peak inspiratory pressures, in others the pressure was less than 30 cm of water,^{1 5} as in our case.

It is suggested that gas enters the pulmonary venous system and is sucked into the systemic circulation when alveoli burst during mechanical ventilation and when the intra-alveolar pressure exceeds the pulmonary venous pressure.^{2 6} In experiments on cat lungs, Grosfeld *et al.*⁷ showed that at ventilatory pressures of 50 cm of water pneumothorax and pneumomediastinum occurred, while at 70 cm of water there was intracardiac gas embolisation.

We suspect that oxygen embolisation occurs more frequently than has been reported, as infants may collapse and die too quickly for a chest x-ray film to be performed in life. Radiology at necropsy has been recommended as an aid to detection of air embolism,⁸ but this may be misleading in cases of oxygen embolism as this gas will diffuse out of the circulation after death.

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