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hours. On no occasion was there more than a temporary improvement in PO_2 , while the disturbance to the infant during the manipulation required to place the endotracheal tube was considerable. In particular, passage of the endotracheal tube into the right main bronchus without occlusion of the right upper lobe was extremely difficult.

One of the infants in whom selective intubation was carried out subsequently underwent a thoracotomy, where multiple pleurotomies were performed. Again, improvement was modest and short lived.

In four of the five infants, including the infant in whom surgery was performed, spontaneous resolution and recovery occurred subsequently. The fifth infant died of cor pulmonale complicating bronchopulmonary dysplasia after a period of time at home.

We feel that selective bronchial intubation has a place in the management of pulmonary interstitial emphysema but that any improvement may be limited. In particular, improvement may fail to occur when pulmonary interstitial emphysema is associated with bronchopulmonary dysplasia, rather than as a complication of the idiopathic respiratory distress syndrome. The radiological distinction between these two is not always possible.

Reference

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Non-immunologic hydrops fetalis

Sir,

We read with interest the paper by Iliff *et al*¹ and would like to add a further cause of non-immunologic hydrops fetalis.

At routine ultrasound examination a 32 year old primigravida who was 23 weeks pregnant was found to have a grossly hydropic infant, with ascites and displacement of the fetal heart to the left. No irregular blood group antibodies were found and viral serology was negative. A detailed examination of the fetal heart using echocardiography showed normal cardiac anatomy, although the right atrium was rather dilated.

The presence of gross hydrops was confirmed after delivery at 27 weeks' gestation and despite attempts at resuscitation, the infant died 30 minutes after birth. Subsequent investigations failed to show any evidence of haemolysis, blood group incompatibility, congenital infection, or chromosome abnormality.

A full necropsy showed that there was atresia of the right main bronchus associated with noticeable enlargement of the right lung and a shift of the heart and mediastinum to the left. The left lung was compressed and severely hypoplastic. There was also moderately severe tracheo-bronchomalacia with partial deficiency of the tracheal cartilage and collapse of the posterior membrane.

Histology of the right lung showed distension with

an accelerated maturation and numerous cup shaped alveoli. The left lung showed failure of expansion but the maturity was consistent with the gestational age; hyaline membranes were noted together with occasional inhalation of squames. Bronchial cartilage was present in both lungs. This cause of non-immunologic hydrops fetalis has not previously been recorded, although cases of similar nature, for example adenomatoid malformation of the lung and intrathoracic tumours causing mediastinal displacement, have been the subject of previous reports.²

References

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Transient hyperphosphatasemia

Sir,

We were interested to read Arthur *et al*'s short report on the benign nature of transient hyperphosphatasemia.¹ Although this is the first account in the English published reports, the syndrome has recently been reviewed by Nathan.² He described six cases in 1980 and reviewed the 28 patients previously reported: four more cases were added by Rosalki and Foo.³ Only one of these patients was found to have a malignancy.

At this hospital, an 18 month old infant presenting with Letterer-Siwe disease was found to have an alkaline phosphatase value of 4153 IU/l (normal adult range 90 to 330 IU/l). This increased to 12 978 IU/l over the next week, while other biochemical tests of liver function remained normal. An ultrasound and computed tomogram of the liver, and a bone scan showed no abnormalities. Isoenzyme analysis was typical of that found in transient hyperphosphatasemia and the alkaline phosphatase activity returned to normal over the following month. In another patient, a 2 year old with leukaemia on UKALL VIII maintenance treatment, alkaline phosphatase increased to 2050 IU/l after a period of normal liver function tests. As the child's other parameters of liver function were all normal, further investigations were deferred and within a month the alkaline phosphatase had reverted to its previous value. Failure to appreciate the possibility of transient hyperphosphatasemia could have led to unnecessary further investigation in the first patient and an inappropriate reduction in 6-mercaptopurine dosage in the second. Thus it should be noted that hyperphosphatasemia may also be a benign finding in children with an already established malignancy and may not be indicative of any deterioration in their condition.

References

- ¹ Arthur LJH, Hill PG, Attenburrow AA. Transient hyperphosphatasemia. *Arch Dis Child* 1984;59:996-7.