

mechanical ventilators or those undergoing other forms of intensive care—and with little handling or disturbance. The value of this technique lies in its ability to measure non-invasively kidney size and to detect structural abnormalities. Further studies will establish the frequency of such abnormalities and allow study of the natural history of such conditions.

We thank Dr C Metreweli for helpful advice.

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## Neonatal pericardial effusion associated with central eventration of the diaphragm

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**SUMMARY** A normal infant born at term developed tachypnoea. A massive pericardial effusion associated with absent central tendon of the diaphragm and eventration into the pericardium was found. Surgical correction was performed and the baby is now well and developing normally.

#### Case report

A 3.8-kg boy, the second son of healthy unrelated white parents, was born by caesarean section after induction of labour failed at 41 weeks' gestation. He required no resuscitation and general examination was normal. His 29-year-old mother had clinical signs of polyhydramnios from 32 weeks' gestation, which was confirmed by ultrasound scan but no fetal abnormality was seen. After birth the infant was apparently well until 21 hours of age when tachypnoea at rest was noticed. The respiratory rate was 80–90 per minute with subcostal recession and nasal flaring; crepitations were heard at the bases of both lungs, but air entry and chest expansion were normal. Peripheral perfusion was good, and all the pulses were present and of normal volume. Systolic blood pressure was 60 mmHg in both the arms and legs. The heart sounds were soft and there were no murmurs or pericardial rub. The liver was palpable 3 cm below the costal margin but there was no peripheral oedema.

Radiographs of the chest showed a greatly enlarged cardiac shadow (Fig. 1). The electrocardiogram was of normal voltage with an axis of  $+120^\circ$  and without evidence of ventricular enlargement. The  $\text{Pao}_2$  rose to 210 mmHg in 50% inspired

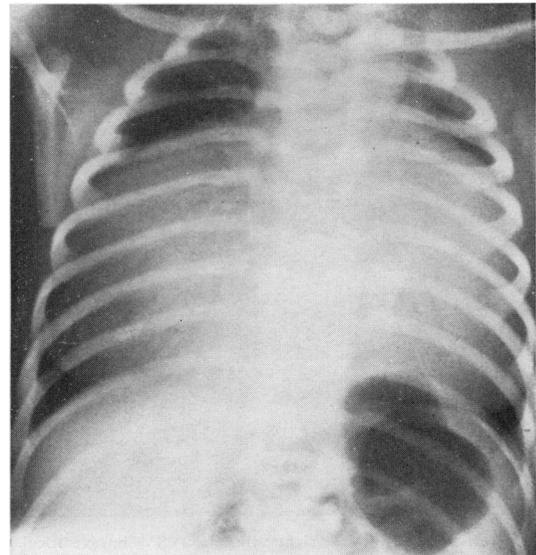


Fig. 1 Chest radiograph showing large cardiac shadow and evidence of pulmonary venous congestion.

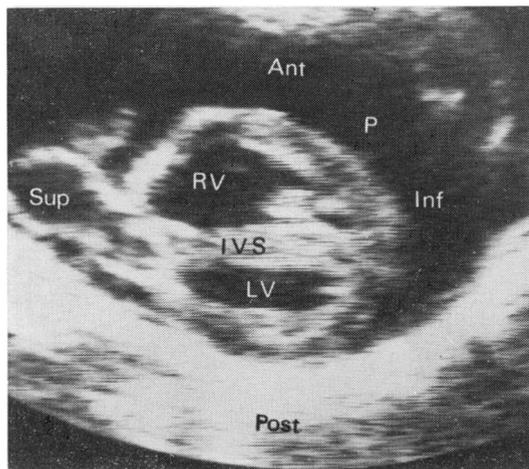
oxygen concentration, excluding congenital heart disease with a right-to-left shunt. Radial arterial pressure (by intra-arterial catheter) was 80/60 mmHg with no pulsus paradoxus.

Real time echocardiography (ATL 860C ultrasound imager) showed a heart of normal size with normally related chambers, valves, and great vessels but there was a large pericardial effusion (Fig. 2).

Plasma electrolytes and albumin concentration were normal as were haematological studies. No bacterial pathogens were isolated from cultures of superficial swabs, urine, blood, or cerebrospinal fluid. Antinuclear factor was not detected either in the infant's blood or in that of his mother. Nor were antibodies present to Q fever, psittacosis, *Mycoplasma pneumoniae*, cytomegalovirus (IgM), herpes simplex, or coxsackie viruses. Maternal blood did not contain antibodies to adenovirus or toxoplasma (IgM) but rubella HA1 antibodies were detected at a titre of 1 in 20.

At 70 hours of age pericardiocentesis was performed and a No 4 Gensini catheter was left in the pericardial sac and 70 ml of straw-coloured fluid drained over 48 hours. Within 4 hours the respiratory rate decreased to 50 per minute, without any change in the blood pressure.

The protein concentration of the pericardial fluid was 45 g/l and that of the plasma 61 g/l, suggesting a transudate. Cultures of the fluid were sterile, microscopical examination showed some epithelial cells and the predominant cell types were bone



LV = left ventricle, RV = right ventricle, IVS = intraventricular septum.

Fig. 2 Cross sectional echocardiogram showing the ventricular mass surrounded by pericardial fluid (P).

marrow elements. There was no excess of inflammatory cells nor were malignant cells seen.

Serial chest radiographs showed an unusually high gas shadow in relation to the left hemidiaphragm. Fluoroscopy of the diaphragm showed that a part of the body of the stomach apparently herniated through an anterior midline defect into the enlarged pericardial sac. The baby was referred for surgical repair of the diaphragmatic defect.

At thoracotomy the findings were extremely unusual. The central tendon of the diaphragm and pericardial floor were missing. Attenuated diaphragmatic muscle separated the eventrated liver, stomach, and bowel from the heart which was displaced posteriorly and compressed against the vertebral column. The abdominal viscera were reduced into the abdominal cavity and the defect repaired by antero-posterior mattress plication sutures through the muscle and covered by redundant pericardium to create a new floor to the pericardial sac.

The child made an uneventful recovery and the postoperative chest radiograph was normal. Barium enema ruled out malrotation of the gut or other associated gastrointestinal abnormalities.

## Discussion

Pericardial effusion in the newborn is rare. The most common cause in a non-hydropic baby is an intrapericardial teratoma although this has always been associated with acute cardiorespiratory distress and tamponade.<sup>1,2</sup> Peritoneo-pericardial diaphragmatic herniations are even more rare; only seven cases have been reported in children under age 3 years.<sup>3-6</sup> In only one<sup>6</sup> was a pericardial effusion also present, ventilatory support was required from birth and several other congenital abnormalities were present. Our case is unique because the baby was otherwise normal; his only symptom was moderate tachypnoea, and the diaphragmatic defect was that of eventration.

Neonatal pericardial effusion should be considered in the presence of an unexpectedly large cardiac shadow on chest radiograph. Ideally pericardiocentesis could be followed by instillation of CO<sub>2</sub> into the pericardial sac to allow identification of an intrapericardial mass. Teratomas and other cardiac tumours do not move with respiration unlike herniated peritoneal contents.

The mechanism of accumulation of the pericardial fluid is unknown. It may be due to mechanical irritation of the pericardium or to the effect of repeated cycles of negative pressure drawing fluid across the thin eventrated diaphragm and underlying peritoneum.

Surgical correction was by a plication which greatly reduced the area of attenuated muscle and

eliminated paradoxical movement. Despite the gross backward displacement of the heart the child did not suffer the haemodynamic consequences of cardiac tamponade, probably because this developmental defect was associated with considerable distension of the pericardial sac.

We thank Dr Pickering for permission to report this case, also Dr J Van Jones, and Dr E W Fletcher.

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## Serosal miliary Crohn's disease in association with probable coeliac disease

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**SUMMARY** A 2-year-old boy with peritoneal miliary Crohn's disease is described. In addition to vague gastrointestinal symptoms, ascites was a prominent feature. The jejunal mucosa was markedly atrophic, compatible with a diagnosis of coexistent coeliac disease.

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The incidence of Crohn's disease seems to be increasing. It occurs infrequently below school age and is rare before 2 years of age. Although the disease may affect large areas of bowel, in those with atypical manifestations the diagnosis may be reached with difficulty. Recently, a young child with persistent vague gastrointestinal symptoms but obvious ascites was found at laparotomy to have serosal miliary Crohn's disease. Previous cases with this presentation have been aged between 13 and 53 years.<sup>1–4</sup>

#### Case report

A 27-month-old boy presented with a 2-week history of loose, watery stools, episodic vomiting, and striking abdominal distension. He was adequately nourished and of average height. A plain radiograph of the abdomen showed colonic dilatation and some air-fluid levels. He was managed conservatively and within 48 hours the symptoms

had resolved spontaneously. One week later he developed general malaise, anorexia, constipation, and loss of weight. Abdominal distension was again prominent, but no localised abdominal lesion was palpable. Laboratory studies were unhelpful and he continued to be unwell.

Exploratory laparotomy showed no abnormalities; the appendix was histologically normal, as were some adjacent lymph nodes which contained reactive hyperplasia.

The child remained unwell, was anorexic, and had a persistently distended abdomen. During the next 3 weeks body weight declined from 12.9 to 11.7 kg and total serum protein from 56 to 46 g/l. Serum albumin was 27 g/l, alkaline phosphatase (ALP) 111 U/l, cholesterol 2.6 mmol/l (100.39 mg/100 ml), and iron 2.4 µmol/l (13.4 µg/100 ml); prothrombin time and blood count were each within normal limits. Peroral jejunal biopsy showed profound villus atrophy, with pronounced crypt hyperplasia, increased intraepithelial lymphocytes, and a heavy mixed inflammatory cell infiltrate, findings compatible with active coeliac disease. He was started on a gluten-free diet which produced an immediate improvement in appetite and weight. The serum proteins increased to 73 g/l.

His condition soon deteriorated however, and within 6 weeks he was found to have pronounced abdominal distension now accompanied by ascites,