cular steroid has little influence on its immunosuppressive effect. Dexamethasone, which is one of the most potent corticosteroids, has comparatively little immunosuppressive property.⁶

The results of our study show that the use of dexamethasone in the treatment of bronchopulmonary dysplasia is not associated with an appreciable increase in the incidence of infection, although the possibility of a Type II error prevents us from ruling out a small effect. Steroids should not be withheld for fear of predisposing to infection.

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Neonatal pneumomediastinum with isolated mitral obstruction

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Abstract

While mediastinal air accumulation in the ventilated newborn is usually a benign entity, tension pneumomediastinum can embarrass both respiratory and cardiac function. A case of isolated left ventricular inflow obstruction caused by a pneumomediastinum in a premature infant is described that resolved with high frequency ventilation. The development of pneumomediastinum and haemodynamic compromise in a premature infant warrants echocardiographic examination.

Intrathoracic air accumulation is a common complication of mechanical ventilation in the newborn intensive care unit. The clinical consequences of pneumomediastinum and pulmonary interstitial emphysema in the premature infant have been well described.^{1,2} Pneumomediastinum is a less commonly encountered result of barotrauma, often having minimal pulmonary or cardiac consequences. We here describe a case of pneumomediastinum in a low birthweight premature infant that caused haemodynamic compromise secondary to isolated left ventricular inflow obstruction. High frequency ventilation was successfully used to decompress the mediastinum and restore normal cardiac function.

Case report

A boy, weighing 750 g, was born vaginally at 25 weeks' gestation to a 23 year old woman (gravida 4, para 3) with an uncomplicated prenatal course. Labour was spontaneous and Apgar scores were 4 at 1 minute and 5 at 5 minutes with immediate intubation in the delivery room. During the first 12 hours of life, the patient required increasing ventilator rates and pressures secondary to severe hyaline membrane disease. A small patent arterial duct was diagnosed by echocardiography at 12 hours

of age and indomethacin treatment was instituted.

During the next 12 hours, the ventilatory peak pressures and rates were modestly weaned. The infant's chest radiograph, however, showed signs of worsening disease with the appearance of interstitial emphysema. During the second day of life there were increasing ventilator requirements. Peak ventilatory pressures of 30 cm H₂O (mean airway pressure of 17 cm H₂O) and rates of 70 breaths/minute and 100% oxygen were needed to maintain a pH of 7.24, a carbon dioxide arterial pressure of 7.8 kPa and an oxygen arterial pressure of 7.8 kPa.

On day three of life, the patient continued to require maximal respiratory support. A chest radiograph showed free air in the mediastinum and increasing opacity of the lung fields (fig 1a). Subcutaneous emphysema was also noted. Repeat echocardiography was performed to assess the efficacy of the indomethacin treatment. Pulsed Doppler examination confirmed that the arterial duct had closed. The four chamber view, however, showed a large mass like effect superior and posterior to the left atrium (fig 1b). Pulsed Doppler examination showed evidence of inflow obstruction at the mitral valve with turbulent flow and a peak inflow velocity of 100 cm/second (fig 1c). Simultaneous tricuspid inflow was laminar with a peak inflow velocity of 20 cm/second (fig 1d). Postulating that a tension pneumomediastinum was compromising mitral inflow and possibly contributing to pulmonary oedema, strategies to decompress the mediastinum were considered. In view of the infant's precarious condition, surgical decompression was thought to be too risky. High frequency jet ventilation at a rate of 420 breaths/minute was instituted and a reduction of mean airway pressure from 17 to 8 cm/H₂O was effected.

Over the next 12 hours, the infant's chest radiograph showed resolution of mediastinal air (fig 2a) with concurrent improvement in arterial

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Figure 1a, Infant's chest radiograph showing mediastinal and subcutaneous air accumulation as well as opacified lung fields. **1b**, Echocardiogram (four chamber view) showing large mass effect (m) superior to the left atrium. Asterisk indicates area of mitral valve commissural opening; ra, right atrium; rv, right ventricle; lv, left ventricle; s, superior; and l, left. **1c**, Pulsed Doppler examination of mitral inflow before high frequency jet ventilation treatment. Note turbulent flow as well as abnormal 100 cm/second peak inflow velocity. **1d**, Pulsed Doppler examination of tricuspid inflow before high frequency jet ventilation showing normal laminar flow and peak inflow velocity of 20 cm/second.

Figure 2a, Infant's chest radiograph after high frequency jet ventilation showing resolution of mediastinal air. **2b**, Repeat echocardiogram (four chamber view) after high frequency jet ventilation showing resolution of mass effect on left atrium; ra, right atrium; rv, right ventricle; lv, left ventricle; la, left atrium.

blood gases. Repeat echocardiography showed resolution of the mass effect impinging on the left atrium (fig 2b) and normal mitral inflow.

Discussion

The clinical consequences of intrathoracic air accumulation in the neonate secondary to

mechanical ventilation have been well described.^{1 2} Air trapped in the pleura, lung parenchyma, or mediastinum, especially if under tension, can embarrass respiratory and cardiac function.

A tension pneumomediastinum can interfere with the normal intrathoracic pressure associations, compromising normal venous return

patterns to the right and left sides of the heart.³ This may be reflected in low right and left ventricular inflow velocities on pulsed Doppler examination. Most cases of pneumomediastinum in the neonate result in anterior air accumulation with negligible cardiac or respiratory sequelae. A large isolated posterior mediastinal air accumulation is much less common, but may also lead to respiratory decompensation as has recently been described.⁴

The above case illustrates a previously undescribed cardiac side effect of pneumomediastinum in the neonate. The echocardiogram showed an air filled mass compressing the superior, posterior left atrial wall impinging upon the mitral valve. In contrast with the low velocity signal that would have been obtained had the systemic or pulmonary veins been obstructed, the pulsed Doppler pattern confirmed obstruction at the mitral valve level. Venous return through the tricuspid valve, however, as evidenced by Doppler flow, was normal. This localised cardiac effect is postulated to have resulted from the regional differences of air accumulation in the posterior mediastinum.

The physiological consequences of acute mitral valve obstruction are varied depending on the severity. Both pulmonary oedema and low cardiac output are possible sequelae. In our case, the increased opacity of the lung fields on the chest radiograph paralleled the development

of mitral obstruction. It is not clear, however, whether the change in the chest radiograph reflected pulmonary oedema, worsening lung disease, or both. Similarly, although cardiac output was compromised during this time, the relative contribution of mitral obstruction is difficult to quantify. Clinical improvement rapidly followed resolution of the pneumomediastinum.

Surgical decompression has been described as the treatment of choice for tension pneumomediastinum.^{2,4} High frequency ventilation, however, has been recently utilised in the neonate to treat life threatening pulmonary air leaks⁵; this was effectively employed to decrease the air leakage and decompress the mediastinum in our patient. Normal left ventricular venous inflow was demonstrated after resolution of the mediastinal air.

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