Embryology of Bronchial Atresia

To the Editor:

We were interested by the report by Williams and Schuster on the association of bronchial atresia and bronchogenic cyst (Chest 1985; 87:396-98). We are aware of two similar cases in previous publications. Case 2 of Tsuji et al showed bronchial mucocele and hyperinflation of the right upper lobe, together with a mediastinal bronchogenic cyst. Case 2 of Brocard and Gallouedec had bronchial atresia and mucocele of the posterior segmental bronchus of the left lower lobe and a bronchogenic cyst in paraspinal position, as was confirmed pathologically.

Other congenital anomalies have been associated with bronchial atresia: pericardial defect, strium septum defect and left-sided inferior vena cava, congenital cystic adenomatoid malformation (CCAM) Stocker type III, unilateral agenesis of the kidney, anomalous venous drainage of the left upper lobe, pulmonary sequestration, and fusion of apophyseal joints C2-C3.

These associated defects occur early in embryologic development; eg, the pericardium is normally formed in the fifth week and the interstitial septum is normally complete in the eighth week. CCAM Stocker type III is believed to result from an injury at the time of early budding of the lung, and renal agenesis occurs prior to 31 days. These associations indeed point towards the early phases of budding of the lung (fourth to sixth week) as the origin of bronchial atresia, at least for a number of cases.

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Hypokalemia from Usual Salbutamol Dosage

To the Editor:

It is well known that both salbutamol and theophylline poisoning produce hypokalemia and hyperglycemia, and that in both cases these changes have been reversed by administration of propranolol.1,2

Hypokalemia induced by salbutamol has been described only during overdosage.3,4 However it might be possible, we think, that therapeutic concentrations of salbutamol also produce hypokalemia. We describe a case of a 34-year-old male nurse and football player who took 6 mg (0.08 mg/kg) salbutamol half an hour before a 30 min, 100 W exercise test, which was performed as described by Tarssanen et al, in order to study serum potassium changes during exercise with medication.

As we know, potassium concentration normally rises during exercise.3,4 However, after taking salbutamol, our patient's serum potassium concentration rised remarkably less than during the exercise test without medication. Serum potassium concentration was 0.5 mmol/l lower than before the exercise and the patient became hypokalemic (3.4 mmol/l) 30 min after the exercise (Fig 1). The patient felt palpitation and he received 40 mg propranolol. About two hours later, he went to play football. Thereafter, he felt unusual palpitation and tiredness that, we suppose, might be due to profound hypokalemia.

Due to our exercise test results, we suggest that hypokalemia may be noticeable also during ordinary salbutamol treatment. Rapid potassium changes, especially hypokalemia, during exercise may achieve dangerous rhythm disturbances.

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FIGURE 1. Serum potassium levels during exercise with salbutamol 6 mg (— — — — —) and without medication (———).