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# Airway Obstruction in Lungs Obtained from an Asthmatic Donor Complicating Heart–Lung Transplantation

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Severe bronchospasm following transplantation of heart and lungs obtained from a nonasthmatic donor into an asthmatic recipient has previously been reported.[1] There have been no previous reports, however, of respiratory problems encountered during transplantation of lungs obtained from asthmatic donors. We report a case of extreme difficulty in pulmonary ventilation complicating transplantation of heart and lungs obtained from an asthmatic donor into a nonasthmatic recipient.

## Case Report

The donor, a 15-yr-old boy, had sustained a severe head injury 3 days previously. Ventilatory support had been commenced immediately on admission to a neurosurgical intensive care unit, and conservative management aimed at restricting further neurologic damage had been instituted. Brain stem death was confirmed on the third day, and the patient was referred for multiple organ donation.

The only previous medical history was of asthma requiring occasional treatment with a salbutamol inhaler. There was no other history of atopy.

During the period of ventilation in intensive care, regular chest physiotherapy and tracheal suctioning had been performed until confirmation of brain stem death, and there had been no requirement for bronchodilator therapy; peak airway pressures never exceeded 20 $cmH_2O$. Clinical examination on the day of organ donation revealed course crepitations dorsally, and small quantities of viscid secretions were obtained on tracheal suctioning. There was no evidence of infection: the patient was afebrile, with negative microbiologic cultures and a normal chest x-ray. Arterial blood gases before transfer to the operating room for organ harvesting were as follows: *p*H, 7.5; $P_{CO_2}$, 34 mmHg; and $Pa_{O_2}$, 170 mmHg at an $FI_{O_2}$ of 0.4.

The donor operation proceeded uneventfully until just before cannulation of the pulmonary artery when a rise in peak airway pressure from 20–35 $cmH_2O$ was noted. The only drugs administered up to this point were pancuronium, vasopressin, and metaraminol. Intravenous fluids had been limited to colloid in the form of 20% human albumin solution and crystalloid as 4% dextrose/saline. Using the technique of a single cold pulmonary artery flush for lung preservation,[2] a mixture of prostacylin (epoprostenol; Flolan [Burroughs Wellcome]), albumin, mannitol, and autologous blood at 4 °C was flushed through the pulmonary circulation *via* a cannula in the main pulmonary artery. The aorta was cross-clamped, cardioplegia administered through the aortic root, and the heart–lung bloc excised after full inflation of the lungs and application of the tracheal cross-clamp. Following our usual practice, the lungs were deflated to two thirds of their vital capacity before immersion of the organs in ice cold normal saline for transportation.

The recipient, a 43-yr-old man with end-stage pulmonary sarcoidosis, was anesthetized and supported on cardiopulmonary bypass pending arrival of the donor organs. When the donor heart–lung bloc was examined at the transplant center, it was noted that the lungs would not fully deflate. However, as the degree of deflation was sufficient to allow implantation into the recipient's chest and to permit surgical access for anastomosis of donor and recipient circulation and trachea to be performed, the operation proceeded in the normal way.

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Reperfusion of the transplanted organs was achieved after a donor-to-recipient ischemic time of 110 min. Before separation from cardiopulmonary bypass, pulmonary ventilation was commenced, initially by manual compression of the reservoir bag of the anesthetic circuit. At this stage, it was noted that despite inflation pressures in excess of 50 $cmH_2O$, minimal inflation of the lungs was being achieved and, furthermore, that minimal deflation of the lungs was occurring on cessation of application of positive pressure.

No further attempt at ventilation was made, and cardiopulmonary bypass was continued while rigid and flexible bronchoscopy were performed. Drug therapy at this stage consisted of isoproterenol by infusion and methylprednisolone, which was routinely administered during heart–lung transplantation. In addition, an intravenous (iv) infusion of salbutamol was commenced. At bronchoscopy, the narrow, constricted caliber of the airways was noted, and diffusely distributed mucous plugs were found, particularly in the right lung. Bronchial lavage was performed, and the bronchi were cleared of fluid and debris by suctioning. On recommencement of ventilation, inflation/deflation was more easily accomplished, with peak airway pressures now being within acceptable limits (30 $cmH_2O$) and the lungs deflating adequately during expiration.

The recipient was separated from cardiopulmonary bypass without difficulty; his trachea was extubated within 8 h after operation; and he experienced no subsequent respiratory complications. Lung function tests performed 2 weeks posttransplantation were not indicative of an obstructive airways pattern.

## Discussion

In this case, the increase in airway pressure during the donor operation and the incomplete deflation of the lungs before implantation in the recipient suggest that bronchospasm was triggered before removal of the heart–lung bloc from the donor and that airway obstruction persisted during transportation despite denervation. In view of the crepitations and viscid tracheal secretions noted on examination of the donor before surgery, we postulate that accumulation of bronchial secretions, and possibly the release of humoral factors during the donor operation to which the asthmatic lung may be more sensitive, led to the development of bronchospasm with subsequent inspissation of material in the airways contributing to air trapping.

The bronchoscopic findings of narrowed airways with edema fluid and mucous plugs are identical to the postmortem findings in the lungs of asthmatics who die as a result of status asthmaticus.[3]

The main implication of this case is that caution needs to be exercised when considering the presence of even relatively mild asthma in potential lung donors. Since the heart–lung donor pool is already fairly limited,[4] we would hesitate to suggest that such mild asthma should constitute a contraindication to this form of organ donation.

It should be kept in mind, however, that the potential triggers for bronchospasm present themselves in many guises during organ procurement and transplantation. For example, physiotherapy tends to be discontinued after confirmation of brain stem death, allowing the accumulation of secretions in the respiratory tract; intravenous preparations ranging from antibiotics to colloid solutions are administered as routine; cardiopulmonary bypass, used by some organ procurement teams during heart–lung acquisition,[2] is inevitably accompanied by release of anaphylotoxins[5]; and ischemia during the donor-to-recipient transfer results in the intrapulmonary generation of inflammatory mediators.[6]

In conclusion, we presented a case of airway obstruction resulting from bronchospasm and mucous plug formation in lungs obtained from an asthmatic donor that seriously complicated heart–lung transplantation in a nonasthmatic recipient.

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