

## CASE REPORTS

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## Deliberate Hypoventilation in a Patient with Air Trapping during Lung Transplantation

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TRANSPLANTATION of one or both lungs is an evolving surgical treatment for pulmonary insufficiency caused by a variety of lung diseases. One critical decision during lung transplantation is whether to use cardiopulmonary bypass (CPB). Although circulatory support may be useful, especially during pneumonectomy, CPB during lung transplantation often is associated with both early graft dysfunction and coagulopathy. In the absence of severe pulmonary hypertension, unilateral lung transplantation often can be carried out without CPB, as can bilateral lung replacement using the "bilateral sequential" lung transplant technique.<sup>1</sup> Still, some patients undergoing single or double lung transplantation will develop cardiac or respiratory instability during the procedure that is serious enough to require CPB. Such instability may be caused by acute right ventricular failure when the pulmonary artery (PA) is clamped or by inadequate ventilation or oxygenation, especially during pneumonectomy. An additional reason for cardiorespiratory instability during this pro-

cedure is hyperinflation of the lungs (air trapping), which leads to decreased venous return, decreased cardiac output, and systemic hypotension.<sup>2</sup> We report a case in which a patient undergoing bilateral sequential lung transplantation developed hemodynamic instability as a result of air trapping. We used deliberate hypoventilation to increase venous return. Although this deliberate hypoventilation was accompanied by severe respiratory acidemia, the patient's oxygenation remained adequate. Deliberate hypoventilation was well tolerated and allowed the transplant to proceed without CPB.

### Case Report

The patient was a 28-year-old, 54-kg woman with end-stage lung disease secondary to cystic fibrosis and repeated lung infections. Preoperative medications included prednisone, warfarin, famotidine, and albuterol *via* an inhaler. Pulmonary function tests indicated the presence of severe obstructive lung disease: forced vital capacity (FVC) 1.51 L, forced vital capacity in 1 s (FEV<sub>1</sub>) 0.79 L (26% of predicted), FEV<sub>1</sub>/FVC 52%, diffusing capacity for carbon monoxide 15.20 (77% of predicted). Sequential bilateral lung transplantation *via* a "clamshell" thoracosternotomy was planned.

Immediately before the procedure, the patient was dyspneic and receiving oxygen while sitting upright. Frequent paroxysms of coughing produced copious, tenacious brown sputum. Auscultation of the chest disclosed distant breath sounds and rhonchi. Laboratory examination was notable for a serum bicarbonate level of 42 mEq/L, a hemoglobin level of 10.8 g/dl, a prothrombin time of 14 s, and a normal electrocardiogram.

Two 16-G intravenous catheters and a 20-G femoral arterial catheter were inserted before the induction of anesthesia. The blood pressure was 140/70 mmHg, and the electrocardiogram showed sinus tachycardia at a rate of 120 bpm. Blood gas analysis during spontaneous ventilation with supplemental oxygen at 6 L/min by nasal cannulae

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revealed a fully compensated severe respiratory acidosis and severe hypoxemia (table 1). Anesthesia was induced rapidly with 500  $\mu\text{g}$  fentanyl, 18 mg etomidate, and 80 mg succinylcholine. A 37-Fr left endobronchial tube was placed. The systolic blood pressure decreased to 85–90 mmHg after induction of anesthesia and initiation of positive-pressure ventilation. We inserted an oximetric PA catheter after induction because we believed that the patient would not tolerate the supine position for placement before induction. The PA pressure was 35/22 mmHg, the CVP was 13 mmHg, the cardiac output was 8.8 L/min, and the mixed venous oxyhemoglobin saturation was 80%. Transesophageal echocardiographic examination revealed mild left and right ventricular hypertrophy with preserved ventricular function and a dilated main PA. Analysis of blood gases during mechanical ventilation with a Siemens 900C ventilator (Solna, Sweden) revealed compensated respiratory acidosis and a marked increase in oxygen tension compared with the preinduction value. Anesthesia was maintained with additional fentanyl, midazolam, and vecuronium.

Single lung ventilation during surgical dissection was associated with mild hypoxemia (oxygen saturation by pulse oximeter decreasing to 90%) and marked hypotension (systolic blood pressure of 60–70 mmHg), which was treated with infusion of crystalloid and hetastarch, dobutamine at 10  $\mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ , and bolus doses of epinephrine (10–30  $\mu\text{g}$ ). The PA systolic pressure increased to 46 mmHg without a change in the PA diastolic pressure, and the CVP increased to 17 mmHg. The mixed venous oxyhemoglobin saturation decreased to 58%. These values reverted to baseline when both lungs were ventilated. Severe hemodynamic instability returned during single-lung ventilation for the right pneumonectomy. At this point, we stopped ventilation to evaluate whether the instability resulted from air trapping. Blood pressure increased to baseline values (systolic blood pressure 90–110 mmHg) approximately 30 s after we stopped positive-pressure ventilation. We discovered that hemodynamic stability and adequate oxygenation could be maintained by deliberate hypoventilation (exhaled minute volume of 5 L/min). In consultation with the surgeons, we decided to proceed with the pneumonectomy

without CPB. The systolic PA pressure increased to 70–80 mmHg when the right PA was clamped, but right ventricular function as assessed by transesophageal echocardiographic examination did not deteriorate, and the cardiac output remained 4.6–7.7 L/min. Blood gas analysis during deliberate hypoventilation showed severe acute respiratory acidosis superimposed on the chronic respiratory acidosis and chronic metabolic alkalosis, with adequate oxygenation (table 1). Despite the low pH, the patient remained normotensive and without arrhythmias. When the transplanted right lung was reperfused, she developed hypotension as a result of vasodilation, which was treated with a norepinephrine infusion. Ventilation of the implanted lung with normal tidal volumes and an increased respiratory rate was well tolerated and resulted in a progressive return of the blood gas values to reference values. The recipient's left lung was removed and the donated left lung implanted without further incident. At the conclusion of the procedure, the patient was taken to the intensive care unit. She awoke 4 h after arrival in the intensive care unit, and her endotracheal tube was removed 27 h after the conclusion of the transplantation procedure. She exhibited no neurologic deficits 2 weeks after the procedure.

## Discussion

Conacher,<sup>3</sup> in a recent review of anesthetic techniques for lung transplantation, stated that "the procedure is a pneumonectomy in a patient who, under normal circumstances, would be adjudged as unfit for such an operation." Lung transplant recipients often have more serious derangements of cardiopulmonary physiology than those encountered in other patients, and many lung transplant recipients require CPB during pneumonectomy. The major disadvantage of CPB is that anticoagulation frequently is associated with severe hemorrhage. In a retrospective review of 37 consecu-

**Table 1. Ventilatory Variables and Arterial Blood Gas Values**

Event	Time* (min)	FiO <sub>2</sub>	V <sub>T</sub> (ml)	Rate (min)	V <sub>E</sub> (L/min)	PIP (cmH <sub>2</sub> O)	pH <sub>a</sub>	Pa <sub>o</sub> <sub>2</sub> (mmHg)	Pa <sub>co</sub> <sub>2</sub> (mmHg)	HCO <sub>3</sub> (mEq/L)	BE
Preinduction	10	NC	—	—	—	—	7.39	49	72	44	+16
Postinduction	52	1.0	500	20	10	45	7.34	304	75	40	+13
Both lungs											
Hypoventilation	180	1.0	—	—	5	25	6.99	106	162	40	+5
Left lung only											
Hypoventilation	250	1.0	—	—	5	25	6.94	301	153	33	–1
Left lung only											
Transplanted	320	1.0	500	15	7.5	30	7.24	426	71	31	+3
Right lung only											
Transplanted	350	0.8	500	20	10	42	7.37	437	46	27	+2
Right lung only											
Both transplanted lungs	625	0.6	520	22	11.4	38	7.44	308	37	25	+2

FiO<sub>2</sub> = fraction inspired oxygen; V<sub>T</sub> = tidal volume; V<sub>E</sub> = expired minute volume; PIP = peak inspiratory pressure; pH<sub>a</sub> = arterial pH; Pa<sub>o</sub><sub>2</sub> = partial pressure of oxygen in arterial blood; Pa<sub>co</sub><sub>2</sub> = partial pressure of carbon dioxide in arterial blood; HCO<sub>3</sub> = bicarbonate; BE = base excess; NC = nasal cannulae.

\* Time is measured in minutes since the start of anesthetic management.

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tive patients undergoing double lung transplantation at our institution from January 1991 through June 1992, support with CPB was associated with significantly increased transfusion of packed erythrocytes, platelets, and cryoprecipitate (table 2).

The decision to use CPB during lung transplantation usually is based on the development of hemodynamic instability or severe gas-exchange abnormalities. Our patient's hemodynamic instability was the result of air trapping and improved during deliberate hypoventilation. Obstruction of expiratory airflow prevents complete exhalation of the delivered tidal volume, such that intrapleural pressure remains positive throughout the respiratory cycle (auto positive end-expiratory pressure (PEEP)).<sup>2</sup> Air trapping of a degree severe enough to result in auto- or intrinsic PEEP can be encountered even in the absence of lung disease if the frequency of mechanical ventilation is sufficiently rapid,<sup>2,4</sup> but it is found most commonly in patients with severe expiratory airflow obstruction. The presence of auto-PEEP is highly negatively correlated with FEV<sub>1</sub> (percent predicted) and highly positively correlated with pulmonary flow resistance and resting hypercarbia.<sup>5</sup>

In a previous case report of the anesthetic management of a patient undergoing bilateral sequential lung transplantation, only mild air trapping was encountered during single lung ventilation, with an increase in partial pressure of carbon dioxide (P<sub>CO<sub>2</sub></sub>) from 54 to 62 mmHg.<sup>6</sup> Curiously, a recent report of 10 patients undergoing bilateral sequential lung transplantation did not identify air trapping as a significant problem during single lung ventilation.<sup>7</sup> Although moderate hypercarbia was encountered during single lung ventilation in

a series of 11 patients with pulmonary fibrosis undergoing single lung transplantation, air trapping was not detected.<sup>8</sup> On the other hand, air trapping has been identified as a frequent complication of single lung ventilation during single lung transplantation in patients with obstructive lung disease.<sup>9-11</sup> However, the degree of ensuing hypercarbia reported was mild (P<sub>CO<sub>2</sub></sub> 69 mmHg).<sup>9</sup> Deliberate hypoventilation in our patient provoked severe hypercarbia and acidemia, which were partially offset by a preexisting metabolic alkalosis. Many clinicians would be uncomfortable tolerating the degree of acidemia present in our patient. We accepted the severe hypercarbia because our patient's hemodynamics were stable and her oxygenation was acceptable.

In addition to deliberate hypoventilation, PEEP has been used as a means to ameliorate air trapping. Low levels of PEEP (up to 10 cm H<sub>2</sub>O) decreased expiratory resistance during controlled mechanical ventilation and decreased the work of breathing during assisted ventilation in patients with severe airflow obstruction and auto-PEEP.<sup>12,13</sup> However, the application of PEEP requires close monitoring, because if the level of PEEP exceeds the level of auto-PEEP, further air trapping may result.<sup>13</sup> Although we did not apply PEEP to our patient, its cautious application may be justified in similar circumstances.

Tris-hydroxymethyl-aminomethane can ameliorate the acidemia associated with hypercarbia, because it can buffer carbonic acid and reduce P<sub>CO<sub>2</sub></sub> by a nonpulmonary route. However, buffering a significant amount of carbon dioxide with tris-hydroxymethyl-aminomethane requires the administration of a large solute load. Attempting to buffer 25% of the carbon dioxide load for 2 h requires 500 mOsm of tris-hydroxymethyl-aminomethane, resulting in a total solute load of 1,000 mOsm.<sup>14</sup> Because we generally seek to minimize fluid administration intraoperatively to avoid pulmonary edema in the transplanted lung in the postoperative period, we do not see a prominent role for tris-hydroxymethyl-aminomethane in this setting.

Hypercarbia affects cardiac function. Hypercapnic acidosis of a degree similar to that encountered in our patient (pH 6.9) decreased developed tension of isolated strips of ventricular muscle to 30% of the control value.<sup>15</sup> Moderately severe hypercarbia (pH 7.09, P<sub>CO<sub>2</sub></sub> 92 mmHg) decreased the slope of the left ventricular end-systolic pressure-volume relation in dogs by approximately 20%, indicating a substantial decrease

**Table 2. Blood Component Transfusion during the Immediate 72-h Perioperative Period in Patients Undergoing Double Lung Transplantation with or without CPB**

Component	With CPB (n = 20)	Without CPB (n = 17)	P
Packed erythrocytes	5.9 ± 5.4	2.5 ± 2.5	0.02
Platelets	19.7 ± 12.9	0.5 ± 1.9	<0.0001
Cryoprecipitate	7.0 ± 7.5	0.3 ± 1.5	0.0006

CPB = cardiopulmonary bypass.

Data are from 37 consecutive patients undergoing double lung transplantation at the University of Pittsburgh from January 1991 through June 1992. Values are expressed in units (mean ± SD). Groups with and without CPB were compared using the Kruskal-Wallis rank-sum test.

in contractility.<sup>16</sup> Myocardial depression induced by hypercapnia is reversible with  $\beta$ -adrenergic agonists, and it has been hypothesized that this myocardial depression may be due to antagonism of calcium ions by hydrogen ions.<sup>17</sup> However, hypercapnia actually *increased* cardiac output by a combination of a decrease in afterload and an increase in venous return (probably due to catecholamine release).<sup>16</sup> Patients with right ventricular dysfunction may be at special risk, since hypercapnia could diminish the ability of the right ventricle to compensate for an increase in afterload such as that occurring with clamping of the PA during pneumonectomy. Indeed, mild hypercapnic acidosis (pH 7.27,  $P_{CO_2}$  49 mmHg) was associated with a four-fold greater increase in right ventricular end-diastolic pressure during an increase in right ventricular afterload in healthy dogs.<sup>18</sup> Hypercapnia also can cause serious cardiac dysrhythmias in the presence of halothane.<sup>19</sup>

Hypercapnia also exhibits potent cerebral effects. Severe, prolonged respiratory acidosis induced by ventilating rats with 50%  $CO_2$  resulted in a 0.6-unit decrease in intracellular brain pH, *i.e.*, a decrease normally associated with metabolic failure.<sup>20</sup> However, all of the animals subsequently awoke, behaved normally, and exhibited brain histology that was no different from that of control animals.<sup>20</sup>

Severe hypercapnia also is well tolerated in humans. The accidental administration during anesthesia of an estimated fractional inspired carbon dioxide of 35% for approximately 30 min was associated with a pH of 6.86 and a  $P_{CO_2}$  of 248 mmHg but no obvious sequelae.<sup>21</sup> Indeed, a degree of hypercapnia (pH 6.72–6.97,  $P_{CO_2}$  130–250 mmHg) comparable to that in the present report was well tolerated by healthy volunteers in the pioneering work describing the physiology associated with apneic oxygenation.<sup>22</sup> The authors of that classic study induced apnea for as long as 55 min without sequelae, provided oxygenation was maintained. Thus, in a sense, our management represents a modification of an anesthetic technique used for pulmonary procedures in the past.

In summary, deliberate hypoventilation ameliorates the hemodynamic instability induced by air trapping and may obviate the need for CPB during lung transplantation in patients with severe obstruction to expiratory flow. Although marked acidemia may result, previous studies in both animals and humans indicate that it is well tolerated.

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## Prolonged Neuromuscular Block Following Mivacurium

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MIVACURIUM chloride is a new short-acting nondepolarizing neuromuscular blocking agent that undergoes hydrolysis by plasma cholinesterase.<sup>1,2</sup> The rate of hydrolysis of mivacurium *in vitro*, as catalyzed by purified human plasma cholinesterase, is between 70% and 88% of the rate of succinylcholine. We report a case of prolonged neuromuscular block in a young, healthy patient who presented for elective surgery.

### Case Report

A 22-year-old woman was admitted for extraction of impacted wisdom teeth. Medical history was not significant except for polycystic ovarian disease. The patient was taking oral contraception containing 150  $\mu\text{g}$  desogestrel and 30  $\mu\text{g}$  ethinyl estradiol (Marvelon, Organon, Cambridge, UK) for the previous 12 months. She had no known allergies. There was no known history of significant illness in the family, and neither the patient nor anyone in her immediate family had any experience of anesthesia. The patient was 146 cm tall and weighed 54 kg, and physical examination revealed no abnormal physical findings. Results of preoperative routine biochemistry and hematology investigations were normal.

The patient was premedicated with 20 mg temazepam and was anesthetized with 250 mg thiopental, 0.1 mg fentanyl, and 66% ni-

trous oxide in oxygen until intubating conditions were assessed, after which 0.5–0.75% halothane was added to the inspired gas mixture. Monitoring consisted of electrocardiogram, noninvasive blood pressure, oxygen saturation, end-tidal carbon dioxide concentration, and temperature. Monitoring of the neuromuscular block was carried out by transcutaneous stimulation of ulnar nerve at the wrist with supramaximal stimuli in a train-of-four (TOF) mode at 2 Hz every 12 s. The resultant force of contraction of adductor pollicis muscle was measured and recorded using a force transducer and a neuromuscular function analyzer (Myograph 2000, BioMeter, Denmark). The detailed neuromuscular monitoring was carried out because the patient was in a study assessing the clinical effects of mivacurium, which at this stage is not licensed for general use in the United Kingdom.

After stabilization of control responses over 5 min, 11 mg (0.2 mg/kg) mivacurium was administered over 10 s. The first measurable effect (lag time) and complete neuromuscular block (onset time) developed in 34 and 90 s, respectively (fig. 1). Tracheal intubation was carried out 2.5 min after mivacurium administration, and the conditions were excellent. No signs of recovery were observed in the neuromuscular function at the usual expected time of about 20–25 min. The surgery lasted 70 min, but there was still no measurable degree of recovery. The patient was kept anesthetized, and monitoring of the neuromuscular block continued.

The first signs of recovery from neuromuscular block were observed 235 min after the initial administration of mivacurium. The rate of recovery was very slow, taking 7 min for the first response in TOF (T1) to recover from 5–6% of control. It was decided that neostigmine be administered to hasten the recovery. Accordingly, 2.5 mg neostigmine with 0.5 mg glycopyrrolate was administered 242 min after the first administration of mivacurium, when the T1 had attained a height of 6%. The T1 increased to 25% and 50% in 1.7 and 4.3 min, respectively, and to a maximum height of 68% in 8.0 min, after which no further recovery was observed. The maximum TOF ratio attained was 0.34 in 11 min. The patient started to breathe spontaneously at this stage.

Fifteen minutes after the initial administration of neostigmine, another dose of 1.25 mg neostigmine with 0.25 mg glycopyrrolate was administered. The T1 recovered to 80% of its control height 9 min after the second dose of neostigmine. The TOF ratio reached a maximum of 0.45 another 3 min later, when no further recovery was

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