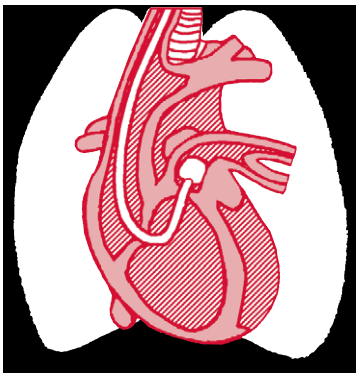

CASE CONFERENCE

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Difficult Weaning From Cardiopulmonary Bypass in the Lateral Position Caused by Lung Collapse

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ATRIAL SEPTAL DEFECTS (ASDs) are usually repaired through a median sternotomy under general anesthesia and cardiopulmonary bypass (CPB). However, a right anterolateral thoracotomy[1] or right-limited posterior thoracotomy[2] approach can be used for cosmetic reasons for closure of an ASD. The anesthetic management is similar to any other cardiac surgical procedure; however, tracheal intubation with a left-sided double-lumen endobronchial tube (DLT) is preferred (if feasible) to permit collapse of the right lung and to facilitate surgical exposure.[1] In the lateral thoracotomy position, in a paralyzed patient during two-lung ventilation, the dependent lung receives more perfusion and the nondependent lung receives more ventilation, whereas during one-lung ventilation the dependent lung is well ventilated and well perfused.[3] Airway collapse in the dependent lung during two-lung or one-lung ventilation would be expected to result in severe ventilation-perfusion mismatch (desaturation) and increased afterload to the right ventricle. A repeated failure of weaning from CPB after ASD closure is described in a child operated through a right limited posterior thoracotomy who developed collapse of the dependent lung because of complete obstruction of the left bronchus with very thick secretions. The left lung could not be expanded despite repeated tracheal suctioning and washing under fiberoptic bronchoscopy. The patient was finally weaned from CPB after reducing tidal volume to one third of the required tidal volume. The pathophysiology of the intraoperative complications, management, and implications of these complications for cardiac surgical procedures approached through a right thoracotomy are discussed.

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CASE REPORT*

A 5-year-old female weighing 12.5 kg presented for an ASD closure. The patient had a history of recurrent respiratory infections. General examination revealed her heart rate to be 100 beats/min, blood pressure to be 110/74 mmHg, and respiratory rate (RR) to be 16 breaths/min. Precordial auscultation revealed a systolic ejection murmur in the left second intercostal space and a mid-diastolic murmur in the tricuspid area. On chest auscultation, the breath sounds were vesicular, and no added sounds were heard. A preoperative chest radiograph revealed plethoric lung fields. Laboratory investigations were within normal limits. The electrocardiogram revealed normal sinus rhythm. Transthoracic echocardiography revealed a 1.5-cm ostium secundum ASD with a large left-to-right shunt.

The child was premedicated with intramuscular morphine (1.5 mg) and glycopyrrolate (0.1 mg). In the operating room, monitoring started with an electrocardiogram, pulse oximetry, and noninvasive blood pressure. Anesthesia was initiated with intravenous thiopental (50 mg), fentanyl (50 μg), and pancuronium (2.0 mg). The trachea was intubated with a 5.0-mm internal diameter cuffed endotracheal tube (ETT). The ETT was taped at the 13-cm mark at the lips; its position was confirmed by auscultation and advancing it to the mainstem bronchus and backing out slowly. Mechanical ventilation was initiated with a tidal volume (Vt) of 120 mL and a RR of 20 breaths/min; the peak inspiratory pressure (PIP) was 18 cmH_2O, and the end-tidal carbon dioxide was 28 to 30 mmHg. Anesthesia was maintained with inhaled isoflurane (0%-2%) in a mixture of oxygen and nitrous oxide (50:50), intravenous fentanyl (50 μg), midazolam (2.0 mg), pancuronium (1.0 mg), and a morphine infusion of 40 μg/kg/h. The femoral artery and right internal jugular vein were then cannulated for arterial blood pressure and central venous pressure monitoring.

The patient was then placed in the left lateral decubitus position for a limited right posterior thoracotomy incision, and the heart was approached through the fourth intercostal space.

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Arterial blood gas (ABG) analysis during the prebypass period revealed pO_2 and pCO_2 of 192 mmHg and 38 mmHg, respectively. After heparin administration, 300 IU/kg, and achieving an activated coagulation time greater than 400 seconds, aortobicaval cannulation was performed and CPB was then initiated. After attaining full flow on CPB, mechanical ventilation was discontinued. Under mild hypothermic CPB (32°C) and cold blood cardioplegia, the ASD was closed with a pericardial patch. After deairing of the heart, mechanical ventilation was initiated with 100% F_IO_2 using a Vt of 120 mL and a RR of 20 breaths/min. The PIP was 24 cmH_2O, and the right lung was well expanded. After rewarming to 35°C, the patient was gradually weaned from CPB. Within a few minutes, the arterial oxygen saturation (SpO_2) decreased to less than 80% and the right ventricle became distended. CPB was reinstituted; the trachea was suctioned; and, after a short period of rest on CPB, the patient was weaned again from CPB. However, like the previous attempt, within moments, the patient experienced desaturation, and the right ventricle became distended. CPB was once again reinstituted.

Inadvertent migration of the ETT to the right main bronchus, left pneumothorax, and/or left-lung collapse were suspected. Chest auscultation revealed reduced air entry on the left side. While arranging for a fiberoptic bronchoscope, the left lobe of the thymus was surgically dissected, and, during manual ventilation, the movement of the left pleura was checked. The pleura was not moving; therefore, the left pleura was opened. The left lung was completely collapsed. Under fiberoptic bronchoscopy, thick inspissated secretions were removed from the left main bronchus. However, repeated airway suctioning failed to expand the collapsed lung. Inotropic support (intravenous epinephrine, 0.1 μg/kg/min) was initiated, and 2 more attempts were made to wean from CPB, the first one with normal ventilation and the second one with a larger tidal volume (Vt of 150 mL). Before weaning from CPB, surgical retraction of the right lung was discontinued. During both weaning attempts, the patient could not be separated from CPB. The typical clinical features upon separation were desaturation, hypotension, bradycardia, and hyperinflation of the right lung. Furthermore, the heart was hidden under the hyperinflated right lung.

After remaining on CPB for approximately 15 more minutes, an attempt was again made to wean the patient from CPB. At this time, a Vt of 40 mL was used with a respiratory rate of 25 per minute. The PIP at this time was 12 cmH_2O. This time, after separation from CPB, the systolic arterial pressure and the SpO_2 remained at 70 to 80 mmHg and 80% to 88%, respectively. The right heart did not distend, but the heart rate and $ETCO_2$ were 160 beats/min and 55 mmHg, respectively, yet showed an improving trend. The CVP ranged between 2 and 4 mmHg. Over the following 20 minutes, the Vt was increased to 80 mL, and 50 mL of blood were transfused. The transfusion of blood was based on visual assessment of right ventricular filling and ongoing blood loss. While increasing the Vt, the inflation of the right lung and the right ventricular filling were constantly visually assessed for potential hyperinflation and distention, respectively. The systolic blood pressure remained at greater than 75 mmHg. ABG analysis showed a pO_2 and pCO_2 of 55 mmHg and 65 mmHg, respectively. After preparing for additional blood transfusion (if required), the venous cannulae were removed, and all the cardiotomy sites were checked for surgical hemostasis. Thereafter, the aortic cannula was removed, surgical hemostasis was ensured, and a chest drain was inserted. Anticoagulation was not reversed. Within 15 minutes of decannulation, the ribs and the skin over the thoracotomy wound were suture approximated and the patient turned supine. Within minutes, the SpO_2 spontaneously increased to greater than 95%. The total duration of aortic cross-clamp and CPB times was 46 minutes and 228 minutes, respectively.

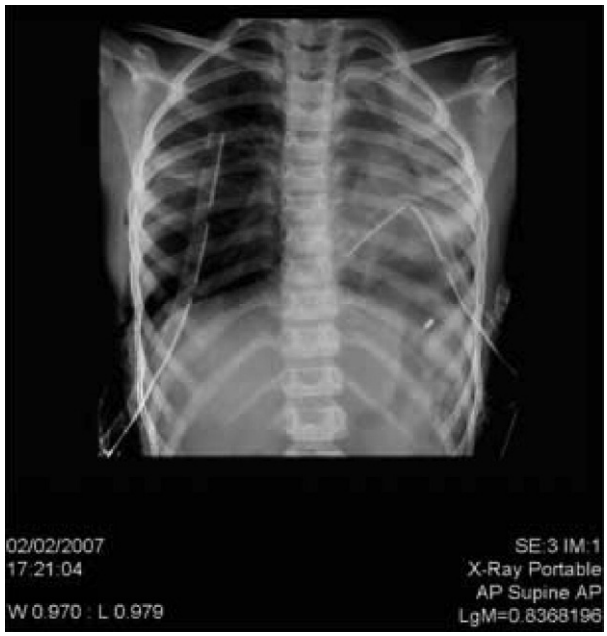

Fig 1. A chest radiograph revealing a collapsed left lung and a hyperinflated right lung.

In the intensive care unit, the patient was ventilated with the same ventilatory settings in the semilateral position (with the left side up). A chest radiograph immediately on arrival to the intensive care unit showed a collapsed left lung and hyperinflated right lung (Fig 1). The patient was hemodynamically stable at this time. Chest physiotherapy and suctioning were done frequently, and mucolite (ambroxol hydrochloride; American Remedies Ltd, Chennai, India) and acetylcysteine granules were administered through a Ryles tube. ABG analysis after half an hour revealed pO_2 (at F_IO_2 of 1.0) and pCO_2 of 405 mmHg and 60 mmHg, respectively. Chest auscultation on the left side revealed air entry with scattered adventitious sounds. Vt was initially increased to 100 mL and then to 120 mL. Anticoagulation was slowly reversed with 50 mg of intravenous protamine. Over the next 5 hours, air entry into the left chest became almost normal. A chest radiograph at this time revealed full expansion of the left lung and marginal hyperinflation of the right lung (Fig 2). Moreover, ABG improved to a pO_2 (at F_IO_2 of 0.6) and pCO_2 of 221 mmHg and 47 mmHg, respectively. On the first postoperative day, the thoracotomy wound was closed in layers. Total chest drain output was 200 mL. The remaining hospital course of the patient was uneventful, and she was discharged on the 7th postoperative day. A chest radiograph before discharge showed no abnormality.