CASE REPORTS

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Circulatory Failure after Anesthesia Induction in a Patient with Severe Primary Pulmonary Hypertension

Laurent Höhn, M.D., Alexandre Schweizer, M.D.,* Denis R. Morel, M.D.,† Anastase Spiliopoulos, M.D.,‡ Marc Licker, M.D.**

PRIMARY pulmonary hypertension (PPH) is an uncommon, almost uniformly fatal disease that generally affects young adults.[1] The risk of death is best correlated with right ventricular hemodynamic indices and New York Heart Association functional class.[2] Although anticoagulants, calcium-channel blockers and prostacyclin have shown great promise, lung transplantation remains the only viable treatment option in patients who remain symptomatic and deteriorate during treatment.[3]

We describe a patient with severe PPH in whom general anesthesia and mechanical ventilation precipitated the onset of cardiac failure and necessitated urgent cardiopulmonary bypass.

Case Report

A 48-yr-old man with PPH was scheduled to undergo lung transplantation after a 3-yr history of progressive dyspnea with home oxygen therapy. Right-sided heart catheterization showed elevated pulmonary artery pressures (125/59 mmHg) with normal cardiac output (4.5 l/min) and mixed venous oxygen saturation (Smv_{O_2}) of 74%. Pulmonary hypertension was unresponsive to inhaled nitric oxide, and treatment trials with calcium antagonists and prostacyclin failed to improve dyspnea and exercise tolerance. Twelve-lead electrocardiography (ECG) showed sinus tachycardia with right-axis deviation and right atrial and right ventricular hypertrophy. Lung volumes were within normal values, and resting arterial partial pressure of oxygen (Pa_{O_2}) was 57 mmHg while breathing room air.

Before surgery, electrocardiography leads and a pulse oximeter probe were placed, and arterial and pulmonary artery catheters were inserted during local anesthesia for continuous monitoring of mean arterial pressure (MAP), mean pulmonary arterial pressure (MPAP) and central venous pressure. After a 3-min oxygenation period, arterial oxygen saturation (Sp_{O_2}) and Smv_{O_2} increased (Sp_{O_2} from 91 to 95% and Smv_{O_2} from 72 to 78%), with no change in systemic and pulmonary artery pressures (150/100 mmHg and 130/62 mmHg, respectively). General anesthesia was induced with incremental doses of fentanyl (250 μg) and midazolam (3.5 mg). Succinylcholine (75 mg) was given to facilitate airway intubation with a double-lumen tracheobronchial tube, and controlled mechanical ventilation was initiated using low tidal volumes (5–7 ml/kg). Anesthesia was maintained with 0.5–1% isoflurane in oxygen.

As shown in figure 1, anesthesia induction was associated with a marked decrease in MAP (from 125 to 85 mmHg). A large increase in MPAP (from 80 to 115 mmHg) occurred in response to tracheal intubation, with MPAP exceeding the level of MAP. Within 2 min after the start of positive-pressure ventilation, right ventricular failure developed, as indicated by high end-expiratory central venous pressure, a dramatic decrease in cardiac output (from 3.9 to 1.2 l/min) and by low Sp_{O_2} ($< 85\%$), Smv_{O_2} (30%), and expired carbon dioxide values ($< 2\%$). Hemodynamics and gas exchange improved transiently with the administration of epinephrine (two repeated doses of 50 μg followed by an infusion of 0.01 $\mu g \cdot kg^{-1} \cdot min^{-1}$). At that time, transesophageal echocardiography (TEE) showed marked dilatation of the right cardiac chambers, with massive tricuspid dilatation and underfilling of the left cardiac cavities caused by a leftward shift of the interatrial and interventricular septa (fig. 2A). Shortly after thoracotomy (10 min after the start of positive-pressure ventilation), hemodynamics dramatically deteriorated (MAP of 50 mmHg with sinus rhythm at 50 beats/min); therefore, heparin (300 IU/kg) was administered and cardiac arrest ensued; open-chest cardiac massage was initiated while the femoral vessels were cannulated. Immediately after the institution of partial cardiopulmonary bypass (3.5 l/min, pump flow), MAP increased, right-sided cardiac pressures (MPAP and central venous pressure) decreased, and arterial oxygenation recovered to normal values.

Bilateral sequential lung transplantation was uneventful and adequate gas exchange was easily achieved. Temporary inotropic support was necessary during weaning from cardiopulmonary bypass. At the end of surgery, transesophageal echocardiography showed marked improvement of cardiac function, with normalization of right ventricular size and interventricular septum position (fig. 2B).

Postoperatively, the patient slowly emerged from anesthesia. Pressure support ventilation was necessary for 10 days, and the patient was discharged, fully ambulant and self-caring, from the hospital 70 days after undergoing transplantation. At the 2-yr follow-up, the patient's lung function volumes and arterial gas exchange are within normal values (forced vital capacity of 85%, and forced expiratory volume in 1 s of 75% of predicted values and arterial P_{O_2} of 95 mmHg breathing room air).

* Staff Anesthesiologist.

† Associate Professor, Division of Anesthesiological Investigations.

‡ Chief-Surgeon of the Thoracic Surgical Unit.

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Address reprint requests to Dr. Licker: Division d'Anesthésiologie, Hôpital Universitaire de Genève, CH-1211 Geneve 14, Switzerland. Address electronic mail to: marc-joseph.licker@hcuge.ch

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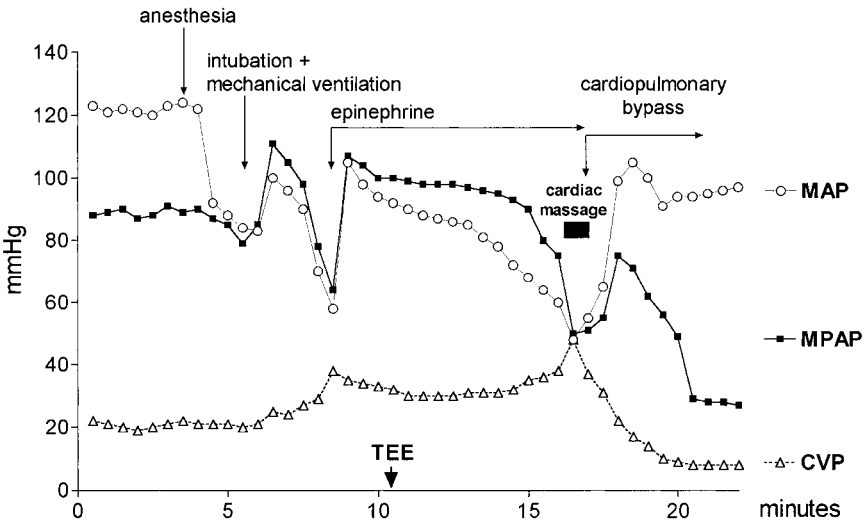

Fig. 1. Hemodynamic time course during anesthesia induction, tracheal intubation, and mechanical ventilation in a patient with severe pulmonary hypertension. CVP = central venous pressure; MAP = mean arterial pressure; MPAP = mean pulmonary arterial pressure; TEE = transesophageal echocardiography.

Discussion

Despite several reports concerning the effects of epidural anesthesia in patients with severe PPH,[4–6] there is a paucity of literature regarding the management of general anesthesia and lung ventilation. Myles *et al.*[7] described a single case of near-fatal anesthesia induction for heart–lung transplantation that also necessitated direct cardiac compression and urgent cardiopulmonary bypass.

In the current case, anesthesia induction and positive-pressure ventilation precipitated the onset of circulatory failure. First, the anesthetic-induced suppression of sympathetic tone was associated with decreased cardiac output and a 35% decrease in MAP. Although catecholamines administered exogenously or released in response to tracheal intubation transiently restored MAP, there was a further increase in MPAP. Second, the institution of positive-pressure ventilation impeded sys-

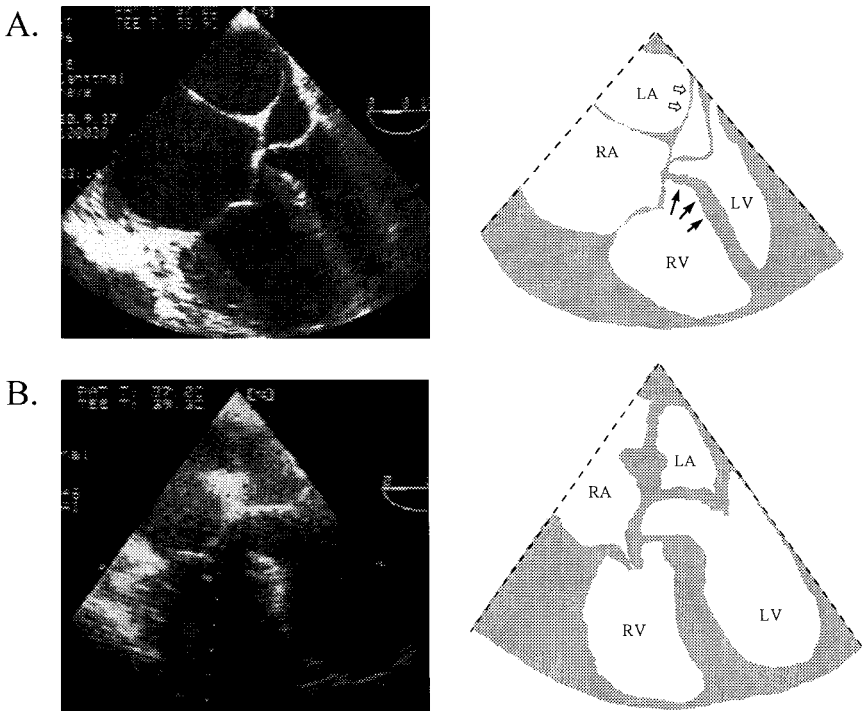

Fig. 2. Echocardiographic four-chamber view of the heart at end-systole. After anesthesia induction and during mechanical ventilation, the enlarged right atrium and ventricule (RA and RV) cause a leftward shift of the interatrial and interventricular septa (arrows) and collapse of the left atria (LA) and left ventricule (LV). After successful bilateral lung transplantation and weaning from cardiopulmonary bypass, transesophageal echocardiography shows normalization of the dimensions of the cardiac cavities.

temic venous return and could increase right ventricular afterload by closing of small pulmonary arteries.[8] Third, right ventricular dilatation with tricuspid regurgitation and leftward septal shift resulted from right-sided pressure overload. Subsequently, incomplete filling of the left ventricle with decreased diastolic compliance further impaired left ventricular function.[9] A positive feedback mechanism involving interventricular coupling could be triggered, whereby the decreased left ventricular function (*via* septal motion) tends to reduce right ventricular systolic function and *vice versa.*[10] Taken together, ventricular interdependence–positive feedback mechanisms and the series connections between the pulmonary and systemic circulation are implicated in the development of circulatory failure during anesthesia induction and positive-pressure mechanical ventilation.

In many patients with long-lasting PPH, pulmonary vasodilators are ineffective because of irreversible hypertrophic and fibrotic changes within the pulmonary arteries.[11] In the current case, preoperative tests involving inhaled nitric oxide, prostaglandins, calcium-channel blockers, or nitroglycerin failed to show beneficial effects; therefore, we did not consider the administration of these agents. Isoflurane was chosen in this patient because it was shown to lower pulmonary vascular resistance.[12] Any factor that worsens pulmonary hypertension (hypoxia, acidosis, light anesthesia, nitrous oxide) or that may decrease ventricular function (hyperinflation, depressant anesthetics, hypothermia) should be avoided. A pulmonary artery catheter (with mixed venous oximetry) and transesophageal echocardiography are the best ways to track cardiac function and fluid management during lung transplantation.

A cautious approach consists of cannulation of the femoral vessels, either immediately after induction or when the patient is still breathing spontaneously, during local anesthesia and light sedation. Because cardiopulmonary bypass is always indicated in lung transplantation for PPH,[13] and given the risk of circulatory collapse after anesthesia induction and mechanical ventilation, such a protocol has been adopted in our institution for all patients with PPH undergoing lung transplantation.

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