Hemodynamic Compromise Associated with Air Trapping Following Coronary Artery Bypass Surgery*

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Cardiovascular collapse due to pulmonary hyperinflation was noted in a patient with chronic obstructive pulmonary disease following median sternotomy for cardiac surgery. Treatment included bronchodilator therapy to reduce airway obstruction, limitation of minute ventilation, and increasing time available for exhalation. High inspiratory flow rates and expiratory retard may be beneficial.

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PAP = \text{pulmonary artery pressure; PCWP = pulmonary capillary wedge pressure}\]

Cardiac tamponade causes hypotension by extracardiac mechanical limitation of ventricular filling, decreased venous return, and subsequent reduction in cardiac output. A less recognized cause of hypotension in patients undergoing median sternotomy and cardiac operations is mechanical limitation of ventricular filling due to high intrathoracic pressure transmitted by hyperinflated lungs which compromise cardiac function. This phenomenon, descriptively termed, “air trapping,” is seen in patients with pulmonary diseases including loss of elastic recoil or increased airway resistance. When supported with mechanical ventilation and standard inspiratory/expiratory time (I:E) ratios, patients with airway disease fail to empty peripheral air space units during exhalation creating progressive air trapping and hyperinflation.

Hemodynamic and gas exchange embarrassment are potential consequences of air trapping. Patients undergoing cardiac surgery are at risk for dynamic hyperinflation because of the loss of restriction by chest wall and parietal pleura on lung expansion during operation. Reapproximation of the sternum with resumed pleural space restriction may magnify the hemodynamic compromise if this process is not recognized and corrected.

**CASE REPORT**

A 65-year-old woman with chronic bronchitis was admitted with triple vessel disease for coronary artery bypass surgery. Preoperative pulmonary function tests revealed FEV₁ of 30 percent and FVC of 55 percent of predicted. Chest x-ray examination demonstrated bullous lung disease. Prior to instituting cardiopulmonary bypass, the arterial blood gas revealed: pH 7.32; partial pressure of carbon dioxide (PaCO₂), 48 mm Hg; partial pressure of oxygen (PaO₂), 318 mm Hg; fraction of inspired oxygen (FIO₂), 100 percent. After induction of anesthesia using nitrous oxide/narcotic technique, mechanical ventilation was instituted using a ventilator (Siemens 900C, Sweden) with a tidal volume of 12 ml/kg and a rate of ten breaths per minute. The inspiratory flow rate was 90 L/min. Arterial pressure was 110/80 mm Hg at this time with pulmonary artery pressure of 20/10 mm Hg and pulmonary capillary wedge pressure (PCWP) of 8 mm Hg. The patient was weaned from cardiopulmonary bypass with low dose dobutamine (<5 μg/kg/min). When attempts were made to close the chest 15 minutes after bypass, precipitous hypotension and bradycardia occurred and peak airway pressure increased to 100 cm H₂O. A norepinephrine infusion was titrated to maintain the systolic pressure greater than 100 mm Hg. Hemodynamic measurements revealed PAP of 32/20 mm Hg and PCWP of 22 mm Hg. The chest was immediately reopened. Upon opening the chest, the blood pressure returned to initial levels after bypass. The lungs were hyperinflated and herniating through the incision. No ischemic changes were noted on ECG monitors and coronary artery grafts were intact. Secretion output was minimal and wheezing was not appreciated.

The following measures were taken: tidal volume and respiratory rate were reduced and I:E ratio was changed to prolong the exhalation phase. The plane of anesthesia was unchanged. Twenty-five minutes following these measures, the chest was closed without difficulty. Peak airway pressure decreased to 30 cm H₂O. The patient remained hemodynamically stable and was extubated the next day.

**DISCUSSION**

Acute hypotension on sternal reapproximation following cardiopulmonary bypass can originate in heart-lung interaction in addition to purely vascular compromise. Kinking of coronary artery bypass grafts, valvular malfunction, tamponade secondary to persistent bleeding and compromise of the great veins due to adjacent clot with inadequate returning blood volume are prominent considerations. Median sternotomy with opening of the pleural space provides a greater degree of pulmonary hyperinflation than other thoracic incisions. The consequences of hyperinflation will thus be most pronounced in this group of patients.

Bergman demonstrated air trapping and occurrence of intrinsic positive end-expiratory pressure in a group of anesthetized, paralyzed patients with normal lungs when the respiratory frequency was increased from 10 to 22 breaths per minute. The magnitude of hyperinflation is dependent on several factors. These include tidal volume, mechanical time constant of the respiratory system (time constant = compliance × resistance), and time available for expiration. Air trapping and hyperinflation are more likely to occur in patients with COPD who require high minute ventilation for adequate alveolar ventilation. COPD produces greater time constants for complete passive exhalation.

When obstruction worsens or ventilation increases (increased metabolism or dead space, mechanical hyperventilation) functional residual capacity increases. Dynamic hyperinflation may follow this increase. During passive mechanical ventilation, in patients with COPD, conventional ventilator settings including fixed I:E ratios and flow rates may shorten the available exhalation time leading to air trapping and hyperinflation of the lungs.

Wallis and associates have demonstrated direct mechanical compression of the heart secondary to lung hyperinflation in an open chest animal preparation. Hemodynamic consequences of dynamic hyperinflation include reduction in cardiac output.
in transmural pressure and venous return to the heart. The increase in intrathoracic pressure associated with PEEP may spuriously elevate filling pressures measured with a pulmonary artery catheter (as in our patient). This may falsely assure adequate preload and misguide hemodynamic management in COPD patients. Our patient demonstrated hypotension and elevation of filling pressures when an attempt was made to close the sternum. On opening the chest, blood pressure and filling pressures returned to normal.

Once air trapping is recognized, therapeutic measures should be aimed at controlling factors contributing to dynamic hyperinflation. These include bronchodilator therapy to relieve airway obstruction, reduction in minute ventilation, and maximizing time available for exhalation. Minute ventilation should be reduced to the minimum that will maintain adequate pH. Other measures to reduce air trapping and improve gas exchange include use of high flow rates and expiratory flow retard. These techniques are thought to improve ventilation/perfusion relationships in the face of alveolar inhomogeneity.

In conclusion, pulmonary hyperinflation and consequent PEEP should be considered in the differential diagnosis of hypotension occurring following chest closure in patients undergoing coronary artery bypass surgery. COPD patients have a greater propensity for developing air trapping. Failure to recognize air trapping as a cause for hemodynamic embarrassment may lead to hemodynamic mismanagement.

REFERENCES


A Case of Delayed Postoperative Cardiac Tamponade with Unusual Echocardiographic Findings*

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Postoperative cardiac tamponade due to localized compression of the atria occurred in a 64-year-old man after aortic valve replacement and repair of an ascending aortic dissection. The clinical findings were subtle and the echocardiographic findings were unusual. Color Doppler flow imaging assisted in making the diagnosis of localized atrial compression.

Delayed cardiac tamponade is an uncommon complication following cardiac surgery. Early recognition and management are essential for satisfactory outcome. Echocardiography and right heart catheterization usually provide a definitive diagnosis. A case of late postoperative cardiac tamponade with unusual echocardiographic findings follows.

CASE REPORT

A 64-year-old man presented with an acute ascending aortic dissection requiring emergency surgical repair consisting of aortic valve replacement (25 mm Duromedics) and insertion of a woven Dacron aortic graft. Postoperatively, he was anticoagulated with warfarin sodium with a prothrombin time of 22 s. He was discharged from the hospital on the 11th postoperative day, but four days later he was readmitted with recurrent atrial fibrillation and complaints of orthopnea, dyspnea, anorexia, and difficulty swallowing. The prothrombin time was 29.8 s (2.37 times control). There was a large left pleural effusion and cardiac tamponade was suspected. No significant anterior pericardial fluid accumulation was seen on the echocardiogram, but parasternal views demonstrated a 4-cm posterior loculated pericardial effusion causing compression of both right and left atria. The left atrium appeared as a small slit-like orifice. This finding was confirmed on apical views (Fig 1). Increased peak early transmitral Doppler flow velocities (1.4 m/s) were consistent with a decrease in the functional left ventricular inflow orifice secondary to atrial compression. In addition, a loculated effusion was present around the right atrium (best seen on apical and subcostal views, Fig 1 and 2), with severe right atrial compression and impairment of right ventricular diastolic filling (right ventricular early inflow velocity, 1.8 m/s). Color Doppler flow imaging was helpful in distinguishing the compressed atrium from the adjacent pericardial effusion by demonstrating flow from the inferior vena cava through the compressed right atrium into the right ventricle. There was no apparent respiratory variation in right and left ventricular inflow velocity curves. Left ventricular size and systolic function were normal. The prostatic aortic valve and graft appeared normal, with no Doppler evidence for prostatic valve dysfunction.

On right heart catheterization, there was a trend toward equalization of diastolic intracardiac pressures: central venous pressure, 19 mm Hg; pulmonary artery diastolic pressure, 21 mm Hg; and pulmonary capillary wedge pressure, 20 mm Hg. The

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