Right Ventricular Failure Post CPB

31st Annual Meeting & Workshops
Society of Cardiovascular Anesthesiologists
San Antonio, Texas
April 18-22, 2009

Alina M. Grigore, M.D.
Albert C. Perrino, Jr., M.D.
Jonathan B. Mark, M.D.

Learning Objectives

Participants will be able to
a. Describe preoperative and intraoperative risk factors for perioperative RV failure
b. Discuss the perioperative diagnosis of RV failure based upon hemodynamic and echocardiographic monitoring
c. Describe a physiologic approach to the primary medical management of patients with acute RV failure
d. Discuss the types of advanced medical and surgical therapies available for refractory RV failure

Case Presentation (Part 1)

A 63-year-old, 75 kg, 5’ 5” female is scheduled for coronary artery bypass surgery because of increasing symptoms of chest pain and dyspnea with minimal exertion. Preoperative cardiac catheterization showed 95% proximal stenosis of the RCA, 90% stenosis of the mid LCX, 90% proximal stenosis of the LAD, and right dominance. A preoperative transthoracic echocardiogram showed LVEF 45%, with inferior severe hypokinesia, RV hypokinesia, and mild mitral and tricuspid regurgitation. She has a past medical history of myocardial infarction (2004) treated with a stent to the RCA. Other medical problems include a history of syncope, treated with a DDD pacemaker (2005), type 2 diabetes, hypertension, chronic obstructive pulmonary disease, and obstructive sleep apnea requiring CPAP. Her preoperative medications include simvastatin, metoprolol, lisinopril, insulin, aspirin, and albuterol and atrovent inhalers. Preoperative lab results show: Hb 12 g/dl, platelets 225K, PT INR 1.18, creatinine 1.2 mg/dl. ECG shows heart rate 68, normal sinus rhythm, Q waves in leads 2, 3, and aVF. Chest x-ray shows mild cardiomegaly and flattening of the diaphragms.

Questions (Part 1)

1. What preoperative risk factors does this patient have for post CPB RV dysfunction? Can any of these be modified prior to surgery?
2. What is the significance of coronary dominance? Does this have any influence on your perioperative management?
3. What perioperative monitoring would you use and why?
4. How would you induce and maintain anesthesia in this patient?
Case Presentation (Part 2)

The patient is monitored with a 5-lead ECG, left radial arterial catheter, right internal jugular pulmonary artery catheter, and TEE. Anesthetic induction is uncomplicated and the patient undergoes a three-vessel CABG operation without difficulty. The patient remains hemodynamically stable before and during bypass. Of note, bronchospasm following tracheal intubation requires treatment with albuterol. The patient is now ready to be weaned from cardiopulmonary bypass.

Questions (Part 2)

1. Which inotropic agents would be indicated, if any, and why?
2. Are any other therapies indicated prior to weaning from bypass?

Case Presentation (Part 3)

The patient is weaned from bypass with milrinone 0.375 mcg/kg/min and epinephrine 0.05 mcg/kg/min. Initial hemodynamic variables show HR 90 with AV-sequential pacing, BP 90/45 mmHg, PAP 55/20 mmHg, CVP 15 mmHg, and CO 3.8 L/min. Initial TEE examination shows moderate LV dysfunction, inferior akinesia, RV hypokinesia, mild MR, and moderate TR. Mechanical ventilation parameters appear to be similar to those seen prior to bypass (tidal volume 600 ml, respiratory rate 12, FiO2 100%, SpO2 97%, peak airway pressure 34 cm H2O).

Questions (Part 3)

1. Given these hemodynamic variables, can you estimate the systemic and pulmonary vascular resistances?
2. Are these hemodynamic variables acceptable at this time?
3. Should her medical management be changed now, and if so, why?
4. Are the TEE findings of concern? What other TEE images and measurements would be useful at this time?

Case Presentation (Part 4)

Protamine 250 mg IV is administered. Approximately 5 minutes later, the peak airway pressure increases to 45 cm H2O, SpO2 82%. The patient becomes hypotensive, despite continued AV sequential pacing at 90, BP 70/40 mmHg, PAP 55/25 mmHg, CVP 22 mmHg, and CO 2.2 L/min. Also of note, the ECG shows persistent ST-segment elevation in lead 2 and V5, varying between 1-3 mm since the end of bypass.

Questions (Part 4)

1. What is the differential diagnosis of the cardiopulmonary deterioration at this time?
2. What role might the diabetes play in these events?
3. Is this a protamine reaction? Are there specific diagnostic tests that would help make this diagnosis?
4. How might the TEE examination help guide treatment at this time?
5. What are the advantages and disadvantages of returning to cardiopulmonary bypass at this time?

**Case Report (Part 5)**

Heparin is given, and cardiopulmonary bypass is reinstituted. After resting the heart for 20 minutes, two additional attempts to wean from bypass are unsuccessful. Intraoperative TEE reveals severely depressed RV function, akinetic RV diaphragmatic and free walls.

**Questions (Part 5)**

1. What medical treatment options are available at this point?
2. Would an intraaortic balloon pump be helpful in this situation?
3. Are additional inhaled agents indicated? If so, are there advantages to using inhaled nitric oxide vs. prostaglandin I2 vs. milrinone?
4. What alternative surgical treatments could be considered?
5. What are the indications/contraindications for RV mechanical support? (RVAD versus ECMO)
6. What VADs are available on the market for RV support?
7. If an RVAD is placed, how do hemodynamic and TEE monitoring help identify problems with the device?

**Discussion**

**Hemodynamic identification of RV dysfunction**

Although the use of pulmonary artery catheters (PACs) remains controversial, and large clinical trials in critically ill medical and surgical patients have failed to show survival benefits for patients monitored with these catheters, no large scale trial has been performed in patients like the one in this case, who is undergoing heart surgery and is at risk for right ventricular dysfunction. Many cardiac surgical centers continue to use PACs for all heart surgery, and others use these catheters selectively. The hemodynamic signature of acute RV failure is readily identified with PAC monitoring and consists of a low cardiac output associated with a high CVP that is disproportionately increased compared to the left heart filling pressure. Volume administration often results in further increases in CVP without a concomitant increase in stroke volume, cardiac output, or blood pressure. The left-sided filling pressure may be overestimated by the PA diastolic pressure owing to an increased pulmonary vascular resistance. In this setting, the PAWP may be much lower than the PADP. With severe RV dysfunction (particularly when the pericardium is intact), the dilated RV displaces the ventricular septum leftward, reducing LV compliance, and increasing LV filling pressure, despite a reduced LV volume or preload. In addition, PAP may not be elevated, simply because the failing RV cannot generate sufficient systolic pressure. In these confusing clinical circumstances, TEE is particularly useful for diagnosis.
Echocardiographic findings in patients with RV dysfunction

Transesophageal echocardiography can provide several quantitative and qualitative measurements of RV structure and systolic function. Wall thickness greater than 5 mm is abnormal and suggests chronic pressure overload. Quantitative assessment of RV function with in the midesophageal four-chamber view has been used to calculate RV fractional area change (RVFAC = [RV end-diastolic area – RV end-systolic area]/RV end-diastolic area × 100%). Normally the RV end-diastolic cross-sectional area is less than 60% of the LV end-diastolic cross-sectional area. With dilatation, however, the RV changes its shape from triangular to round, with concomitant enlargement of the right ventricular outflow tract (RVOT) and flattening of the ventricular septum from right to left. With RV pressure overload, a maximal leftward septal shift is noted at end-systole, whereas RV volume overload is associated with a maximum reversed septal curvature in mid-diastole. As RV dilation becomes moderate or severe, the RV replaces the LV in forming the cardiac apex in the ME 4-chamber view, and the end-diastolic cross-sectional area of the RV may equal or exceed that of the LV. Tricuspid annular plane systolic excursion (TAPSE) is another simple measure, and values of TAPSE less than 20 mm suggests RV systolic dysfunction. Pulsed wave Doppler evaluation of blood flow in the hepatic vein may reveal attenuation of the systolic inflow wave, suggesting elevated central venous pressure or tricuspid regurgitation. Using continuous wave Doppler to measure the velocity of the tricuspid regurgitant jet allows calculation of the peak RV systolic pressure. Finally, tricuspid regurgitation is often encountered in conjunction with RV dysfunction, owing to tricuspid annular dilatation and increased RV afterload.
Use and Echocardiographic Assessment of Ventricular Assist Devices in RV Failure

Ventricular assist devices (VADs) are connected to the heart or placed within the heart to assume some of the workload and allow the ventricles to rest, undergo reverse remodeling, and recover contractile function. Factors to be considered during device selection include the expected duration of support, type of support needed (right, left or biventricular assist), overall cost, device-related mobility, and FDA approval status. The latest indications for mechanical assistance include reversible ventricular dysfunction occurring after cardiac surgery, bridge to heart transplantation, and destination therapy for non-transplant candidates. Mechanical assistance has become an important tool in the surgical management of patients with failing hearts. Currently there are various cardiac assist devices available for both short- and long-term support of both ventricles. Based on the device related blood flow characteristics, VADs are classified as nonpulsatile or pulsatile, and depending on the site of implantation, they are categorized as extracorporeal or intracorporeal. Most of the extracorporeal devices, nonpulsatile and pulsatile, are now used for short to medium-term support. Nonpulsatile devices have either centrifugal or axial flow patterns. The most commonly used devices for RV support (Thoratec and Lexitronix Centrimag) have an inflow cannula placed inside of the RA and an outflow cannula placed either in the RV or PA. In the presence of RV failure with increased pulmonary vascular resistance, the use of an RVAD could potentially lead to lung damage and is not recommended. Extracorporeal membrane oxygenation (ECMO) would be the technique of choice in this particular clinical setting.

Intraoperative TEE examination should begin by assessing the adequacy of ventricular decompression. The supported RV should be relatively empty, with the ventricular septum slightly deviated toward the decompressed right chamber. Tricuspid valve and RVAD inlet flow patterns can be used to adjust the pump flow rates and achieve desired loading conditions. Ruling out the presence of PFO during the post-bypass period is also important, because increased right-sided pressure may cause right to left shunting and hypoxemia. RVAD inlet and outlet cannulae must be assessed for their position and patency. Color flow and spectral Doppler are used to assess flow through the RVAD and measure the inflow gradient. If the inlet is partially obstructed, high-velocity aliased flow at the cannula orifice will be noted in association with ventricular distension and an elevated inflow gradient. Doppler techniques can also be used to assess VAD output. Different flow patterns are seen with pulsatile and continuous flow devices. With chest closure, VAD inlet and outlet obstruction could occur and reassessment of the flow patterns is advisable.

References