Chapter 9
Mechanical Support of the Failing Right Heart

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Case

A 66-year-old man with diabetes mellitus, hypertension, and tobacco use presented with 1 week of exertional chest pain, progressive shortness of breath, and orthopnea. In the Emergency Department (ED), blood pressure was 80/60 mmHg, heart rate 40 beats-per-min, respiratory rate of 20 breaths-per-min and oxygen saturation of 88% in ambient air. Initial labs were remarkable for N-terminal pro b-type natriuretic peptide (Nt-pro BNP) of 5000 pg/mL (normal < 400 pg/mL), troponin T of 5 ng/mL (normal < 0.04 ng/mL), and lactic acid of 5.5 mmol/L (normal < 2.2 mmol/L). The bedside electrocardiogram revealed new Q waves in the inferior leads. Physical examination revealed poor mentation, elevated jugular venous pressure (JVP) of >15 cm H2O with

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positive hepatojugular reflux, clear lung fields, and cold and clammy lower extremities.

9.1 What Is the Initial Assessment?

The first step towards managing this patient was recognizing that he was in cardiogenic shock. In the 1970s, Forrester et al. demonstrated four hemodynamic profiles in patients with acute myocardial infarction [1]. These profiles were based on congestion (pulmonary artery capillary pressure: PCWP > 18 mmHg) and perfusion (cardiac index: CI > 2.2 L/min/m^2). Profile IV patients (those with congestion and hypoperfusion), representing those in cardiogenic shock, have an increased risk of mortality [1, 2].

The coldness and clamminess in the lower extremities suggested decreased tissue perfusion. This suspicion corroborated with the narrow pulse pressure (difference between systolic and diastolic blood pressures) of 20 mmHg (normal ~40 mmHg). A pulse pressure less than 25% of systolic blood pressure is indicative of decreased left ventricle (LV) stroke volume [3].

The elevated JVP in this patient served as evidence for elevated filling pressures which was consistent with his symptoms of shortness of breath and orthopnea. In the Forrester classification system he would be classified as "cold and wet", which supports the clinical suspicion of cardiogenic shock in this hypotensive patient. Q waves in the inferior leads (II, III, avF), imply a late presentation of inferior ST elevation myocardial infarction. This patient was stabilized, then referred to the cardiac catheterization laboratory for ongoing chest pain.

Case Continued

In the ED, he was given ASA 325 mg and atorvastatin 80 mg. He was placed on 3 L per min of oxygen delivered through a nasal cannula with oxygen saturation of 94%. Norepinephrine

was started at 0.1 mcg/kg/min to maintain systemic perfusion. Left heart catheterization (LHC) revealed a right-dominant system with 90% stenosis of the right coronary artery (RCA), status post drug eluting stent to the subtotal occlusion of the ostial RCA. Right heart catheterization (RHC) revealed: Right atrium pressure (RAP): 20 mmHg, Pulmonary artery pressures (PAP): 38/19 [25] mmHg, PCWP: 20 mmHg, cardiac output (CO): 3.8 L/min, CI: 1.9 L/min/m^2, blood pressure:75/60 mmHg. Bedside transthoracic echocardiogram (TTE) at the coronary care unit revealed a preserved left ventricle ejection fraction (LVEF) with a severely hypokinetic right ventricle (RV).

9.2 What Should Be the Next Steps in the Management of this Patient?

The first step would be to either increase the norepinephrine (and/or add vasopressin) to achieve a MAP >65 mmHg. The pulse pressure of 15 mmHg (20% systolic blood pressure) and cardiac index of 1.9 (<2.2 L/min/m^2) were concerning for ongoing cardiogenic shock. An inotrope (dobutamine or milrinone) needed to be started to improve myocardial contractility. Despite the LVEF being normal, the severe RV dysfunction on echocardiogram raised the suspicion of RHF in the setting of right ventricle myocardial infarction (RVMI). Chapter 3 extensively reviews the different imaging findings in RHF. On the hemodynamic profile, the pulmonary artery pulsatility index (PAPi) < 1.0 and the right-left heart pressures mismatch evidenced by RAP/ PCWP >0.86 confirmed RHF. The hemodynamic assessment of RHF is reviewed later in this chapter.

The correction of metabolic derangements such as acidosis, alkalosis or anemia is essential in RHF. It is imperative that appropriate oxygenation is delivered to promote decreased myocardial oxygen demand. Hypoxia and hypercapnia would cause an acute increase in RV afterload which would decrease RV stroke volume [4]. In intubated patients,

elevated positive end-expiratory pressure (PEEP) increases intrathoracic pressure, which can reduce venous return leading to a decrease in preload, thus exacerbating RHF [5].

The failing RV is sensitive to arrhythmias, especially those that cause atrioventricular (AV) dyssynchrony such as atrial fibrillation, supraventricular tachycardia, and ventricular arrhythmias. Anti-arrhythmic agents or direct current cardioversion or defibrillation should be used to ensure sinus rhythm. Our patient was bradycardic, which likely reflected the poor AV nodal conduction due to RVMI. Atrial pacing could be used to increase his heart rate to augment the cardiac output [6] in patients who have epicardial wires or permanent pacemakers in place. Pharmacological strategies include chronotropic agents such as dopamine, isoproterenol, epinephrine, and theophylline.

Although, only 5% of patients in the "Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock" (SHOCK) trial had predominant RV failure, their in-hospital mortality (53.1%) was comparable to patients with LV failure [60.8% p = 0.296] [7]. This observations speaks to the need to be proactive and aggressive in the management of RV failure. However, it needs to be pointed out that "Isolated" RV shock was an exclusion criteria in the SHOCK trial, hence the findings cannot be directly applied to our patient.

9.3 What Is the Pathophysiology of RHF?

The etiology for acute RHF in this patient is RVMI. The pathologic signature of RVMI is necrosis of the LV posterior/inferior wall, septum, and posterior right ventricular free wall. The latter is usually contiguous with the septum and in rare occasions to the anterior right ventricular free wall [8, 9]. The RCA is the most common culprit vessel; however, the involvement of the RV free wall is dependent on the location of the occlusion relative to the RV branches. The occlusion must be proximal to the RV branches to cause RVMI. In a

left dominant system, the left circumflex and the left anterior descending artery could cause RVMI depending on the epicardial vessel giving rise to the RV branches. Cohn et al., were the first to demonstrate the hemodynamic profile of RVMI when they showed RAP >15 mmHg [10]. The distinctive hemodynamic profile is characterized by RHF, low output and clear lungs.

The pathophysiology of RHF is explored in Chap. 1. RHF starts with an initial insult (such as ischemia) to the RV as seen in our patient or as a result of trauma/surgery, air embolus during cardiac surgery, and inflammation (myocarditis). Other potential etiologies include pulmonary arterial hypertension, pulmonary embolism, and acute respiratory distress through the increase of RV afterload. The RV is also preload sensitive, hence the progressive dilatation and worsening tricuspid regurgitation from massive blood transfusion or fluid infusion could cause ventricular interdependence.

The mechanisms for RV failure include: (a) Ventricular interdependence: An increase in RV pressure and volume results in a left shift of the interventricular septum. The consequence of this shift is the reduction of the LV diastolic filling, which contributes to the decline of cardiac output [11]. (b). Pericardial constraint: An acute increase in RV volume can worsen the pericardial constraint, which is transmitted to the septum causing the interventricular septum to shift to the left, thus increasing the LV filling pressure and decreasing the effective cardiac output [12]. (c) Finally, an increase in the RV filling pressures results in the reduction of coronary flow, due to coronary sinus congestion. The reduced coronary flow would contribute to further ischemia [13].

9.4 What Is the Medical Management of RV Cardiogenic Shock?

The medical management of acute RHF should focus on treating the underlying cause. In acute RVMI, coronary artery reperfusion is essential. Management should target

the optimization of preload, contractility, and afterload. In cases with low intravascular volume, cautious fluid infusion is required to increase contractility as per the Frank Starling curve. The central venous pressure (CVP) should be monitored closely during fluid infusion to ensure that it does not exceed 12–15 mmHg in those who are volume depleted [14]. On the other hand, RV volume overload from excess preload can shift the interventricular septum to the left resulting in interventricular interdependence as discussed above.

This patient had elevated right and left filling pressures, so his fluid status needed to be optimized with a diuretic. Optimally, intravenous diuretics should be titrated to keep the CVP between 8 and 12 mmHg and PCWP <18–22 mg Hg [14]. In the setting of fluid overload refractory to diuresis, continuous veno-venous hemofiltration (CVVH) or ultrafiltration might be needed to achieve negative fluid balance. Inotropes such as epinephrine, dobutamine or milrinone can be used to keep cardiac index >2.2 L/min/m^2. Inotrope choice can be institution-dependent and stylistic. Dobutamine acts via β1 receptor stimulation, but may also cause vasodilatation due to β2 effects. We opted to not use dobutamine as an initial strategy in this patient because he was hypotensive. Epinephrine was not a first choice either due to the concern for demand ischemia. Inhaled and parenteral epoprostenol and nitric oxide would be the agents of choice in cases with elevated pulmonary artery pressures to decrease the RV afterload [15].

Case Continued

Despite maximal medical therapy (norepinephrine 1 mcg/kg/min, vasopressin 0.04 units/min, milrinone 0.5 mcg/kg/min, inhaled epoprostenol 30 ng/kg/min), the patient continued to have unfavorable RHC hemodynamics: RAP 18 mmHg, PA 30/18 [12] mmHg, PCWP 15 mmHg, CO 4.3 L/min, CI 2.1 L/min/m^2. The Shock Team was consulted for the consideration of RV mechanical support.

9.5 Which Percutaneous Mechanical Support Options Are Available for Acute RHF?

Intra-aortic balloon pump (IABP) is the most commonly used percutaneous mechanical support in LV failure. IABP has no direct effect on RHF; however, indirect support is achieved through the promotion of coronary perfusion during diastole. IABP's utilization in biventricular failure is based on the concept that its LV afterload reduction effect will reduce the RV filling pressures. In the SHOCK Trial, IABP usage was similar between RV and LV cardiogenic shock [7].

The axial flow Impella RP (Abiomed Inc., Danvers, Massachusetts-USA) and extracorporeal centrifugal flow TandemLife Protek Duo (TandemLife, Pittsburgh, PA) bypass the RV by delivering blood from the right atrium to the pulmonary artery. The Impella RP (shown in Fig. 9.1) is placed through the femoral vein into the inferior vena cava (IVC)/right atrial junction (inlet), and then advanced through the tricuspid valve into the main pulmonary artery (outlet). In the RECOVER RIGHT trial, which included 30 patients with cardiogenic shock post left ventricular assist device (LVAD) implantation, cardiotomy or myocardial infarction, Impella RP was shown to improve the hemodynamic profile of patients through the decrease in RAP and increase in CI [16]. The overall survival at 30 days was 73.3% post device explant or hospital discharge.

In the United States, the Federal Food and Drug Administration (FDA)'s mandated post approval study (PAS) showed approximately 29% (12/42) survival. The disparity between the pre-market approval (PMA) study and the PAS mortality was attributed to patient selection. The survival rate was 64.3% in the PAS cohort who strictly met the PMA criteria [17]. Patients with cardiogenic shock >48 h, cardiac arrest, or with pre-implant hypoxic or ischemic neurologic event were not in the PMA; hence, appropriate patient selection is imperative for Impella RP use. Impella RP is European CE marked.

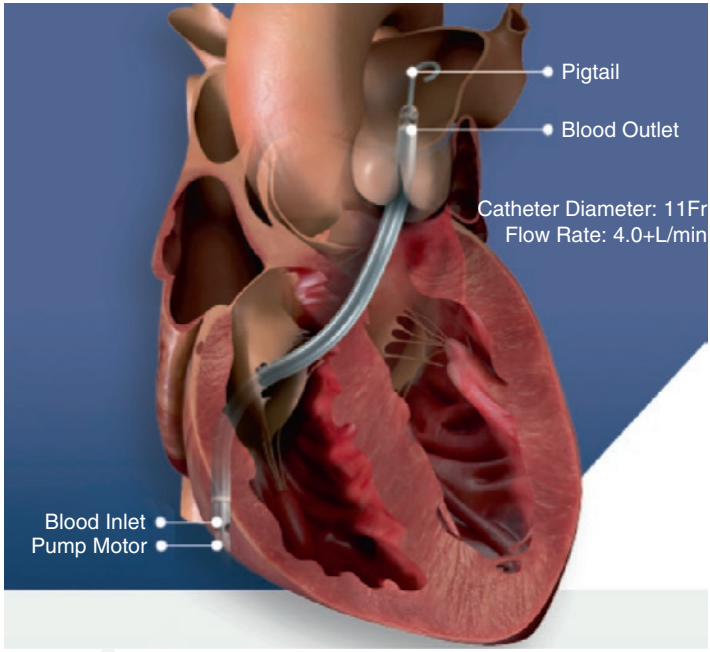

FIGURE 9.1 Schematized Impella RP. Blood is drawn from the inlet (sits in the inferior vena cava) and then delivered at the outlet (in the pulmonary artery). This figure is courtesy of Abiomed Inc., Danvers, Massachusetts-USA

The TandemLife Protek Duo (TandemLife, Pittsburgh, USA), shown in Fig. 9.2 has a proximal lumen in the right atrium while the distal lumen is in the pulmonary artery. The dual lumen cannula is inserted through the right internal jugular vein and the TandemLife Protek Duo can provide up to 4.5 L/min of flow. Blood is drained from the right atrium into an extracorporeal centrifugal pump and then delivered into the pulmonary artery. It provides the advantage of groin-free insertion, allowing patients to be mobilized and rehabilitated while in the hospital. In cases of lung failure, an oxygenator can be added to the TandemLife Protek Duo [18].

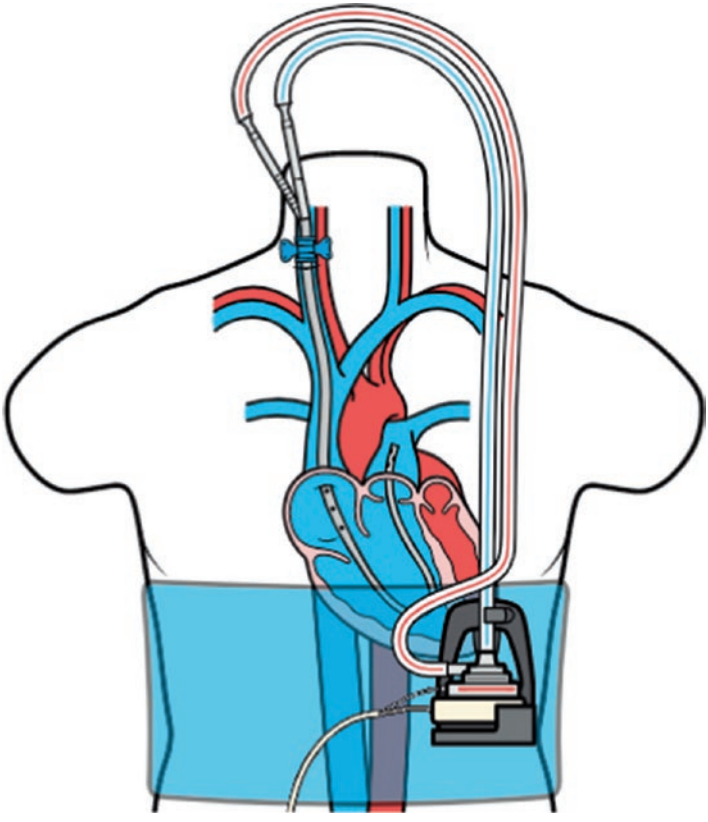

FIGURE 9.2 Schematization of blood circulation through the TandemLife Protek Duo. Venous blood (blue color) is drawn from the right atrium into an extracorporeal centrifugal pump (shown on the right). Oxygenated blood (red color) returns through the dual lumen cannula into the pulmonary artery. This Figure is courtesy of TandemLife, Pittsburgh, USA

Recently, a two-center, retrospective review of 17 patients showed successful wean of TandemLife Protek Duo in 23% (n = 4) patients. Although the device served as a bridge to right ventricular assist device (RVAD) in 35% (n = 6) of the cohort, the mortality rate was still high (n = 7) [19]. In the trial

above, the indications for TandemLife included elevated RAP despite aggressive medical therapy; inability to wean inotrope or vasopressor support while on continuous flow LVAD; and clinical signs of RV failure. Most of the patients in this series had RHF after LVAD implantation.

Veno-arterial extracorporeal membrane oxygenation (VA-ECMO) takes blood from the right atrium (via the femoral vein), passes through the oxygenator and then delivers oxygenated blood to the femoral artery (percutaneous) or the aorta (surgical). Sometimes, a distal perfuser is placed in the superficial femoral artery or the posterior tibial artery to overcome lower extremity ischemia from the large-bore cannulas. Although VA-ECMO reduces preload to both ventricles, it causes elevation of LV afterload.

The Impella RP and TandemHeart Protek Duo tend to decrease the RV preload but increase the left ventricle preload since the outlet or distal lumen is located in the pulmonary artery. In biventricular failure, increasing the LV preload (Impella RP and TandemHeart) or LV afterload (VA-ECMO) could worsen pulmonary edema.

Case Continued

Based on the patient's tenuous hemodynamic values, an Impella RP was implanted through the right femoral vein. Over the next week, vasopressors as well as inhaled epoprostenol were weaned off. Despite this, he remained on a high level of support with RAP 16 mmHg, PA 34/18 [23] mmHg, PCWP 18 mmHg, CO 4.2 L/min, CI 2.1 L/min/m^2, MAP 70 mmHg on milrinone 0.5 mcg/kg/min and the Impella RP. Due to the duration of support required by the patient (the Impella RP is FDA approved for up to 14 days of support), the Advanced Heart Failure Team was consulted for the placement of an RVAD.

9.6 Which Hemodynamic Parameters Are Useful for the Assessment of RV Function?

Invasive hemodynamic monitoring with a pulmonary artery catheter is critical to understanding RV pathology. Cohn et al. identified RAP>15 as a marker of hemodynamically significant RVMI [10]. Presence of RAP >15 mmHg is a risk factor in for RHF in continuous flow-left ventricular assist devices (CF-LVAD) [20]. A right–left heart pressure mismatch is another clue to RHF. The normal RAP is ~5 and PCWP ~10, therefore a normal RAP/PCWP is ~0.5. Lopez-Sendon et al. demonstrated that an RAP/PCWP >0.86 correlated with pathologic evidence of RVMI [21]. Subsequently, RAP/ PCWP have been shown to be associated with increased mortality or hospitalization in patients with advanced heart failure [22]. In a study involving contemporary LVAD, a CVP > 15 and an RAP/PCWP > 0.63 accurately predicted RHF [23].

Recently, pulmonary artery pressure index (PAPi) has been proposed as a marker of RHF and is defined as the pulmonary artery systolic pressure minus the pulmonary artery diastolic pressure divided by the RAP (PASys-PADia)/RAP. In a retrospective study of an inferior wall myocardial infarction cohort, hemodynamically derived RAP/PCWP, PAPi and the right ventricular stroke work (RVSW) were compared to qualitative echocardiographic grading of RV systolic function. RVSW was calculated as Stroke Volume/[mean PA pressure − PCWP] × 0.0136. PAPi appeared to have the strongest association with the echocardiographic estimates of RV systolic function ($r = -0.731$, $p < 0.001$) [24]. In the aforementioned study, PAPi showed a high sensitivity (88.9%), specificity (98.3%) and accuracy (97.1%) in predicting the need for a percutaneous RV support device.

Similarly, PAPi < 1.85 was shown to have the highest predictive value in predicting RHF (94% sensitivity and 81%

Table 9.1 Hemodynamic parameters used to evaluate right ventricular failure

Hemodynamic parameter	RAP (mmHg)	RAP/PCWP	PAPi	RVSWI
Without LVAD	>15(10)	>0.86(21)	<1.0(24)	
With LVAD	>15(20)	>0.63(23)	<1.85(25)	<0.3–0.6 (25)

LVAD left ventricle assist device, *RAP* right atrial pressure, *PCWP* PA capillary wedge pressure, *PAPi* pulmonary artery pulsatility index = (PA systolic pressure – PA diastolic pressure)/RAP, *RVSWI* right ventricle stroke work index = [mean PA pressure – mean RA] × stroke volume index
Stroke volume index = cardiac index/heart rate

specificity) in a cohort of patients with RHF after CF-LVAD implantation [25]. Other hemodynamically derived parameters evaluated in this study included: pulmonary artery elastance (PAE): [PA systolic pressure/stroke volume]; pulmonary artery compliance (PAC): stroke volume/(PA systolic pressure—PA diastolic pressure); and RV stroke volume index (RVSWI): [mean PA pressure—mean RA] x stroke volume index. The stroke volume index is calculated from cardiac index/ heart rate. These other hemodynamic parameters were not as sensitive as PAPi. Table 9.1 reflects the hemodynamic parameters used in the clinical setting to define RHF.

9.7 What Are the Surgical Options for RV Failure?

Our patient was in persistent cardiogenic shock (cardiac index of 2.1 L/min/m^2) despite being supported on an Impella RP. In the RECOVER-RIGHT Trial, which led to FDA approval of the Impella RP, the average time of support was 3.0 ± 1.5 days [16]. FDA approval of Impella RP is up to 14 days. Since our patient had been on Impella RP support for 14 days, it was appropriate to consider alternative therapies. Although there are clear indications for LVAD implantation [26], there are no

guidelines for RVAD implantation. The indications for isolated RVAD implantation in the European Registry for Patients with Mechanical Circulatory Support (EUROMACS) include acute myocardial infarction, failure to wean from cardiopulmonary bypass and post-cardiotomy RV failure [27].

9.7.1 Right Ventricular Assist Device

The CentriMag Right Ventricular Assist device (RVAS) [St. Jude, Minneapolis, MN, USA) has an inflow cannula in the right atrial appendage while the outflow cannula is in the pulmonary artery. The RVAS is an investigational device approved for humanitarian use in the United States for acute RV failure up to 30 days. This device can be used temporarily for isolated RV support, biventricular support, or for RHF after LVAD implantation.

The reported prevalence of RHF after LVAD implantation is approximately 20–30%. The 30-day survival of patients receiving CentriMag RVAS in an LVAD cohort (n = 12) is 50%, while on support for an average of 14 days [28]. CentriMag biventricular support in 12 patients (for a range of 4–22 days) confirmed its utility as a bridge to LVAD (n = 8), bridge to recovery or explant (n = 2), with survival of 83% at 7 and 14 days after implantation [29]. The utility of Centrimag for RV support in post cardiotomy cardiogenic shock, orthotopic heart transplantation (OHT), or LVAD placement has also been shown [17].

There is no approved ambulatory durable RVAD at this time. Contemporary LVADs such as HeartMate III (HM3) [Abbott, North Chicago, IL, USA] and HVAD (Medtronic, MN, USA) have been used in an RVAD configuration. The RV is unloaded by placing the inflow cannula in the right atrium or RV, while the blood is pumped through the outflow graft into the pulmonary artery. The utility of HM3 as an isolated RVAD [speed of 5000 rpm, flow rate of approximately 4.2 L/min], has been shown in a 70-year old male with end stage RHF [30]. To decrease the intraluminal length of the

RVAD inflow cannula, several layers of felt spacers are sutured onto the sewing ring. A query of the EUROMACS registry revealed that a total of eight patients were implanted with the HVAD in the RVAD position for isolated RV failure. 30-day survival was 50%; two patients underwent OHT and the RVAD was explanted in one patient due to RV recovery [27].

Two HM3s were implanted in a biventricular configuration in 14 patients with biventricular failure. Initial RVAD flows were 2.4–5.4 (mean 4.0) L/min at speeds of 4400–6700 (mean 5200) rpm. In this study, eight out of 14 patients continued on BiVAD support for 95–636 (mean 266) days including seven discharges to home [31]. Figure 9.3 shows the chest X-ray of a patient with a biventricular HVAD. The total artificial heart (Syncardia Systems, LLC, Tucson, AZ), a pulsatile device, is another option for biventricular support [32].

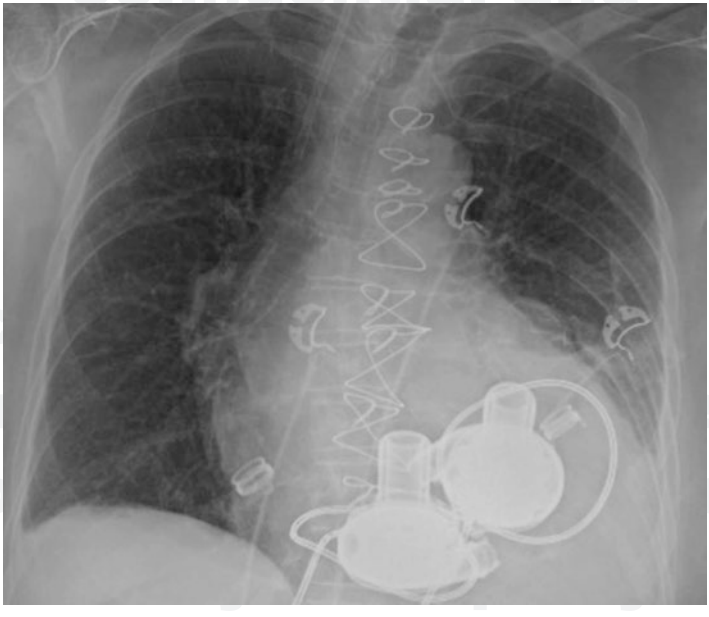

Figure 9.3 Chest radiograph showing a patient with a two HVADs used in a biventricular configuration

9.7.2 *What Are the Indications for Heart Transplant Listing?*

For patients with intractable RHF, heart transplant is the most durable method to correct circulatory dysfunction. Table 9.2 outlines the adapted 2016 International Society for Heart Lung Transplantation (ISHLT) listing criteria for heart transplantation [33]. Advanced age (>70 years), body mass index >35, reversibility of pulmonary hypertension (PVR > 5 wood units), active malignancy, hemoglobin A1C > 7.5, frailty, substance abuse, or red flags on psychosocial assessment could preclude listing for OHT at some heart transplant centers.

A review of the ISHLT registry reveals that 3.1% of patients transplanted were on RVAD prior to transplantation [34]. Limited data exists on the outcomes for OHT in isolated RHF patients. In a series of 12 patients, a mortality rate of

Table 9.2 General indications for heart transplantation

Indications for heart transplant
Refractory arrhythmia
End stage congenital heart disease
Refractory ACC AHA stage D, NYHA class III-IV
Refractory cardiogenic shock requiring continuous inotrope
Refractory angina despite maximal anti anginals or revascularization
Re-transplantation for severe coronary allograft vasculopathy
Estimated Seattle heart failure model 1-year survival of 80% or a heart failure survival score in the high/medium risk
CPET peak VO2 of <14 mL/kg/min without beta blocker, <12 mL/kg/min with beta blockers, young patients <50y (peak VO2 < 50%), BMI > lean body mass–adjusted peak VO2 of <19 mL/kg/min, when RER <1.05, consider VE/VCo2 > 35

NYHA New York Heart Association, *CPET* cardiopulmonary exercise testing, *RER* respiratory exchange ratio, *Vo2* maximum rate of oxygen consumption

50% was reported after OHT, but the majority of these patients were complex congenital patients who are not representative of our patient [35].

Case Conclusion

A Centrimag RVAD was implanted to support the right ventricle. After 5 days, the patient was weaned off all inotropes. A hemodynamic and echocardiographic ramp study revealed a recovered right ventricle. The Centrimag was subsequently decannulated uneventfully, and he was successfully discharged home.

Clinical Pearls

- Initial management of acute RHF should focus on treating the etiology of RHF. Patients need to be stabilized by correcting metabolic derangements, ensuring AV synchrony, appropriate systemic perfusion (MAP > 65 mmHg) and appropriate ventilation.
- Medical management should focus on optimizing the right ventricular preload, afterload and contractility.
- The hemodynamic parameters suggestive of RHF are RAP>15, RAP/ CWP > 0.86 or >0.63 (with LVAD), PAPi <1.0 or <1.85 (with LVAD) and RVSWI <0.3–0.6 (with LVAD).
- Percutaneous mechanical support for acute RHF includes Impella RP, TandemHeart Protek Duo, and ECMO.
- Surgical Options are (1) contemporary durable LVADs implanted as ambulatory RVAD (2) total artificial heart for biventricular support (3) Centrimag RVAD used for temporary support while the RV recovers and (4) heart transplantation for intractable RVF.

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