



Perioperative right ventricular function and dysfunction in adult cardiac surgery—focused review (part 2—management of right ventricular failure)

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Abstract

The single most important factor in improving outcomes in right ventricular (RV) failure is anticipating and recognizing it. Once established, a vicious circle of systemic hypotension, and RV ischemia and dilation, occurs, leading to cardiogenic shock, multi-organ failure, and death. RV dysfunction and failure theoretically can occur in three settings—increase in the pre-load; increase in after load; and decrease in contractility. For patients deemed low risk for the development of RV failure, when it occurs, the correction of underlying cause is the most important and effective treatment strategy. Therapy of RV failure must focus on improving the RV coronary perfusion, lowering pulmonary vascular resistance, and optimizing the pre-load. Pre-load and after-load optimization, ventilator adjustments, and improving the contractility of RV by inotropes are the first line of therapy and should be initiated early to prevent multi-organ damage. Mechanical assist device implantation or circulatory support with extracorporeal membrane oxygenation (ECMO) may be needed in refractory cases.

Keywords Right ventricular failure · Right-sided heart failure · Perioperative · Management · Assist devices

When Harvey's discovery Like an earthquake Had broken up Galenism and other outworn sophistries His masterly work stood forth against a more lurid background of folk superstitions-of Vampires, Witch-burning, Magic, Cabbalism, Astrology, Alchemy, Chiromancy And water-casting. Harvey's discovery was the first step to a transfiguration of medicine; and though after Harvey there arose much false physiology and there with again great floods of medical sophistry Yet from his time medicine has had to reckon with physiology The only source of scientific nosology and therapeutics.—Thomas Clifford Allbutt M.A., M.D. Science and Medieval Thought. London C. J. Clay & Sons 1901 P. 111

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Introduction

The single most important factor in improving outcomes in right ventricular (RV) failure is anticipating and recognizing it. Once established, a vicious circle of systemic hypotension, and RV ischemia and dilation, occurs, leading to cardiogenic shock, multi-organ failure, and death [1]. Therapy of RV failure must focus on improving the RV coronary perfusion, lowering pulmonary vascular resistance (PVR), and optimizing the pre-load. Apart from these, specific causes of RV failure should be identified and corrected if possible [1].

RV dysfunction and failure theoretically can occur in three settings: (1) increase in the pre-load; (2) increase in after-load; (3) decrease in contractility.

However, in clinical setting, these three factors are intermingled and the management depends on optimization of each parameter. The management strategies are based on the following principles:

- 1). Optimizing RV pre-load
- 2). Reducing RV after-load
- 3). Maximizing coronary perfusion
- 4). Increasing RV contractility

- 5). Optimizing myocardial oxygen delivery and oxygen consumption
- 6). Correction of arrhythmias and atrioventricular (AV) conduction to maintain adequate stroke volume of the right and left ventricles

Optimizing RV pre-load

RV pre-load augmentation to a filling pressure of 8–12 mm of Hg is a reasonable first step in management of RV dysfunction. This will lead to an improvement in cardiac output in the presence of RV dysfunction, but not in RV failure. Pre-load may be suboptimal after cardiac surgery due to blood loss, positive pressure ventilation, and third space loss. However, fluid challenge might prove deleterious in the presence of high filling pressures due to the shift of interventricular septum (IVS) to the left, further worsening of the cardiac output. Hence, in the intensive care unit setting, intense diuresis and, if not effective, aggressive ultrafiltration by continuous renal replacement therapy (CRRT) may be initiated to keep the central venous pressure (CVP) below 12 mm of Hg [2]. Targeting a negative fluid balance improves the RV function. A large amount of volume may need to be removed to improve the RV function owing to the flatness of RV Frank-Starling curve. Venous congestion can lead to cardiorenal syndrome and acute kidney injury [3]. Hence, CRRT should be initiated early to improve the outcome. Rescue therapy with CRRT in acute kidney injury due to cardiorenal syndrome is associated with high in-hospital mortality [4].

Reducing RV after-load

As mentioned earlier, pulmonary circulation is a low pressure circuit with low vascular resistance. An increase of systemic pressure from 100 to 140 mmHg decreases the left ventricular (LV) stroke volume by 10%. In contrast, increase in mean pulmonary artery pressure (PAP) from 10 to 30 mm Hg decreases the stroke volume by more than 40%. Moreover, such an increase in PAP reduces RV coronary perfusion leading to sub-endocardial ischemia and reduced contractility [2]. Hence, optimization of RV after-load plays an important role in the management of RV failure.

RV after-load can be reduced either by the adjustment of ventilator parameters or by the use of pulmonary vasodilators. Avoiding hypoxia, hypercarbia, and high positive end expiratory pressure (PEEP), maintaining near normal pH and maintaining systemic saturation more than 95% might reduce the PAP. Since high tidal volume increases the RV after-load, it is important to ventilate with low tidal volume. If the RV failure persists after the optimization of ventilator parameters, pulmonary vasodilators become the next line of therapy.

Pulmonary vasodilators used for this purpose includes inhaled nitric oxide (NO), phosphodiesterase-5 inhibitors (PDE-5), prostacyclin derivatives, endothelin receptor antagonist, and guanylate cyclase stimulator. Inhaled NO is the commonly used pulmonary vasodilators with rapid onset of action and extremely short half-life. Multiple studies have shown improvement of RV function with the use of inhaled NO [5, 6]. Weaning from inhaled NO can cause rebound pulmonary hypertension (PH) and RV failure, which may be reduced with concomitant administration of PDE-5 inhibitors like sildenafil. However, PDE-5 inhibitors can cause systemic vasodilatation with resultant hypotension and its action can extend up to 18 h [2, 7]. Prostacyclin derivatives increase cyclic adenosine monophosphate (cAMP) levels and its inhaled form causes pulmonary vasodilatation and positive inotropic effect without systemic side effects. It is currently the drug of choice to reduce PVR after cardiac surgery [8]. Compared with NO, there is no special equipment required for administration or for toxicity monitoring. Administration of epoprostenol is accomplished via a nebulizer in the ventilator circuit; typical doses in the post-bypass setting are 30–50 ng/kg/min [8]. The other pulmonary vasodilators like endothelin receptor antagonists (ambrisentan, bosentan etc.) and guanylate cyclase stimulator (riociguat) are currently not indicated in acute RV failure setting [9].

Improving RV contractility

Inotropes are used for increasing the RV contractility. Dopamine and epinephrine increase the RV contractility and heart rate, resulting in an increase in oxygen demand. At higher doses, they also cause pulmonary vasoconstriction. Dobutamine acts mainly on the β_1 and β_2 receptors. At lower doses, it causes pulmonary vasodilatation and hence improves the RV contractility. However, it increases the risk of arrhythmias and causes systemic vasodilatation [10]. Milrinone is a phosphodiesterase-3 inhibitor which acts by increasing intracellular cAMP. It improves the RV contractility and causes pulmonary vasodilatation. It has less chronotropic effect than dobutamine. However, bolus doses may cause systemic hypotension [10, 11]. Levosimendan is a calcium-sensitizing agent which improves RV systolic and diastolic functions and causes pulmonary and systemic vasodilatation [12]. Currently, milrinone and levosimendan are the drugs frequently used for the management of RV failure.

Increasing coronary flow

Decrease in systemic vascular resistance and hypotension can lead to decrease in coronary blood flow. Hence, a vasopressor may be required to improve coronary perfusion pressure. Pure α_1 stimulant like phenylephrine should be avoided due to

pulmonary vasoconstriction and reflex bradycardia. At lower doses, epinephrine has a positive inotropic effect. Hence, it is used as the first line of treatment to improve the systemic diastolic pressure and mean pressure. Higher doses might result in pulmonary vasoconstriction [13]. Vasopressin causes vascular smooth muscle contraction by its action on V1 receptor. In doses of 0.1–0.3 μ per min, it also causes pulmonary vasodilatation in addition to systemic vasodilatation. Higher doses of vasopressin may cause splanchnic, digital, and coronary vasospasm. Intra-aortic balloon pump (IABP) is useful in biventricular failure for the improvement of LV performance. Diastolic pressure augmentation by IABP improves the coronary flow and hence the RV perfusion [2, 10].

Improving myocardial oxygen delivery

Myocardial oxygen supply is improved by using optimal ventilator parameters and 100% fraction of alveolar oxygen (FaO_2). Myocardial oxygen consumption is reduced by avoiding tachycardia.

Maintaining sinus rhythm

Maintaining sinus rhythm is important to optimize the stroke volume. Atrial pacing is important in this regard. In patients having conduction block after cardiac surgery, AV sequential or atrio-biventricular pacing is employed. Isolated RV pacing is hazardous as it worsens biventricular function due to ventricular dyssynchrony [14]. Bradycardia should be avoided in the presence of severe tricuspid valve regurgitation (TR). Severe RV dysfunction and failure can cause tachy- and bradyarrhythmias. Tachyarrhythmias can be supraventricular or ventricular and can lead to rapid hemodynamic deterioration. Common tachyarrhythmias are sinus tachycardia, atrial fibrillation, and atrial flutter. Rate control with beta blockers or calcium channel blockers will precipitate cardiovascular collapse; hence, they should be avoided [14]. Rate or rhythm control with amiodarone or ibutilide or electrical cardioversion can be attempted along with the correction of the underlying cause. Bradyarrhythmias in RV failure generally indicate a pre-terminal stage in RV failure or of severe damage to the conduction system due to an RV infarct [14]. Epinephrine boluses and temporary pacing through epicardial wires may be tried to support the heart rate and systemic blood pressure.

RV dysfunction and failure-specific situations and management

Intra-operative RV failure

Intra-operative RV failure is the main reason for difficulty in weaning from cardiopulmonary bypass (CPB) or failure

Table 1 Stratification of patients based on risk for development of RV failure

Low risk	CABG, AVR, MV repair
High risk	PAH, long CPB/ACC time, TV repair
Very high risk	Pre-operative RV dysfunction/RV infarct Heart transplant LVAD Pulmonary thromboendarterectomy

RV right ventricle, CABG coronary artery bypass grafting, MV mitral valve, PAH pulmonary arterial hypertension, CPB cardiopulmonary bypass, ACC aortic cross-clamp, LVAD left ventricle assist device

Table 2 Mechanism of RV failure

Associated with left heart failure
Increased PVR
RV ischemia or infarct
RV after-load mismatch

RV right ventricle, PVR pulmonary vascular resistance

to sustain the hemodynamics after coming off CPB. Intra-operative echocardiography assessment is vital for diagnosing and finding the cause (refer to part I of this article). Eye balling the function of RV is a very useful indicator. Even though some amount of RV dysfunction occurs after cardiac surgery, bulging of the free wall of RV increased CVP and narrow arterial tracing suggesting low cardiac output should alert the surgeon about the possibility of severe RV dysfunction. Patients for cardiac surgery can be stratified based on their risk for the development of RV failure (Table 1). When patients develop severe RV dysfunction or failure intra-operatively, correction of the underlying cause is of paramount importance. RV should be decompressed by going back on CPB, and this will limit further ischemic damage.

RV failure can occur due to a variety of reasons (Table 2). RV after-load mismatch occurs when tricuspid valve repair is attempted in functional TR with RV dysfunction and severe pulmonary arterial hypertension (PAH). Intracoronary or graft air embolism usually causes only a transient RV dysfunction which is often resolved by resting the heart on CPB and elevating the mean arterial pressure. Myocardial protection requires frequent cardioplegia delivery in adequate doses supplemented with topical and systemic cooling. Relying only on retrograde cardioplegia for myocardial protection can lead to severe RV dysfunction [15]. Excessive blood and blood product usage increase the chance of RV dysfunction and failure. Apart from pre-operative correction of anaemia, intra-operative strategies to minimize blood loss should be considered [16]. This includes paying meticulous attention to haemostasis and maintenance of normothermia during CPB to reduce coagulopathy, using blood-conserving

strategies such as avoidance of excessive haemodilution, use of closed extracorporeal circuit, and autologous priming of the CPB circuit. Specific causes RV failure associated with commonly performed cardiac surgical procedures are enumerated in Table 3.

Vasoplegia

As with other vistas in the treatment of impaired hemodynamics, the role of the RV in potentiating multi-organ dysfunction is relatively unexplored. A seminal paper studying the impact of the RV in promoting vasoplegia showed that compared to patients undergoing left-sided surgery, adult patients with right-sided congenital heart disease had double the incidence of vasoplegia after cardiac surgery [17]. The study hypothesizes that this could be due to the congestive side effects of right-sided dysfunction on downstream organs, such as the liver. This is backed by experimental evidence from several studies that show venous congestion triggers the release of inflammatory mediators, such as tumour necrosis factor- α and cytokines, such as IL-6, which link right-sided dysfunction to vascular stress and a vasodilatory state in patients with right-sided heart failure [18, 19]. In addition, functional liver impairments are strongly correlated to pressure in the inferior vena cava which is in turn linked to right atrial (RA) pressure [20]. The liver is a prime source of vasodilatory cytokines [21]. As a direct extension of this consequence, in clinical studies of left ventricular assist device (LVAD) implantation, the strongest correlate of a high vasoactive inotropic score proximate to surgery is significant RV impairment [22]. It therefore seems safe to assume that one of the key steps to prevent vasoplegia

after cardiac surgery is to focus on protecting the RV from functional impairment to the extent possible, and, when functional impairment occurs, to limit RV volume overload. Finally, in situations such as when there is a pre-operative history of PAH, coronary perfusion abnormalities in the RV can also ensue analogous to the left coronary bed.

Protamine reaction

Protamine is a highly cationic protein developed to neutralize the anticoagulant effect of heparin. Protamine is notorious for the acute severe reactions, when used to neutralize the effect of heparin after cardiac surgery. The protamine reactions are classified into three—type I associated with significant hypotension owing to histamine release, type II linked with anaphylactic and anaphylactoid reactions, and type III related to acute pulmonary hypertension (APH). APH increases RV after-load leading to acute RV failure and subsequently low cardiac output [23].

APH is mediated through various immunologic and non-immunologic mechanisms. Literature suggests that the increase in thromboxane-B₂ (TxB₂) plasma levels after protamine administration is the perpetrator of APH. Heparin-protamine complexes activate complement pathway resulting in elevated levels of C5a anaphylatoxins and thromboxanes in susceptible patients [24].

Management of APH may be challenging. Prostacyclin (PGI₂) is a prostaglandin, which stimulates the release of NO from the endothelial cells, resulting in vasodilatation along with platelet anti-aggregatory effect. Inhaled and intravenous (IV) formulations have been tried in APH [25, 26].

Table 3 Causes of RV failure

General causes	
1. Air	
2. Myocardial protection	
3. Increased PVR	Protamine reaction Bronchospasm Tension Pneumothorax/ Lung collapse
Specific causes	
1. CABG	Right graft kink, thrombosis
2. AVR	Right coronary obstruction
3. MVR	Severe paravalvular leak Stuck valve PAH/pre-operative RV dysfunction
4. TV repair	After load mismatch
5. Pulmonary thromboendarterectomy	Residual obstruction Reperfusion injuries
6. Aortic root replacement	RCA button kink, occlusion, or dissection

RV right ventricle, PVR pulmonary vascular resistance, CABG coronary artery bypass grafting, AVR aortic valve replacement, MVR mitral valve replacement, PAH pulmonary arterial hypertension, TV tricuspid valve, RCA right coronary artery

The side effects include systemic hypotension, increased bleeding tendencies, nausea, and vomiting.

Inhaled NO at starting doses of 40 ppm has been tried successfully in the treatment of APH. NO stimulates membrane guanylate cyclase, increasing the production of cyclic guanylate monophosphate, resulting in pulmonary vasodilatation. The side effects associated with NO use include methemoglobinemia, accumulation of free oxygen radicals, and nitrogen dioxide. Abrupt discontinuation of PGI₂ and NO can cause rebound PH [27].

RV failure after heart transplant

Recently, RV failure after heart transplant has become a well-recognized entity. A normal RV exposed to the increased PAP might suffer acute failure, resulting in circulatory collapse. Despite advances in perioperative management in the current era, RV dysfunction accounts for 50% of all cardiac complications and 19% of all early deaths in patients after heart transplantation [28]. The assessment of PH plays a pivotal role during the evaluation of suitable heart transplant recipient. Various revisions in the recipient inclusion criteria have been made over the years, with clear understanding that the RV is not able to bear with a sharp increase in its after-load. This basically led to excluding all the patients that had severe PH [29]. Any situation with a raised LV end-diastolic pressure, as in heart failure, leads to a “reactive” pulmonary vasoconstriction which in turn leads to an irreversible increase in PVR. PH and increased PVR are not only associated with post-transplant morbidity from acute RV failure and perioperative mortality, but also with post-transplant infections and arrhythmias [30, 31]. Though the terms PH and increased PVR have been used interchangeably, it is important to understand that increased PH can occur without an increase in PVR. Understanding this plays a pivotal role in the success or failure of a transplanted heart.

The patient diagnosed with PH need to be classified into “fixed” or “reactive” PH. When baseline hemodynamics are reversed after the administration of pulmonary vasodilators, such as NO, 100% oxygen, sodium nitroprusside, or adenosine, it is called reactive PH which clearly has a better prognosis [32, 33]. The other parameters that are used for risk stratification are systolic PAP (SPAP), transpulmonary gradient (TPG), and PVR index (PVRI). Fixed PH is defined as having a PVR ≥ 4 Wood units (WU), a PVRI ≥ 6 WU/m² (particularly useful in the paediatric and small-size patient population), a SPAP ≥ 60 mm Hg, or a TPG ≥ 15 mm Hg [31, 34]. Each of these parameters plays an independent role, and coming to a clinical conclusion requires a good deal of experience and understanding of the interplay of hemodynamics. The concept of “vasodilator conditioning” has evolved in patients in whom previously irreversible

PH becomes reactive, secondary to inotropic support over a prolonged period [31]. Thus, a patient who was earlier thought to be unsuitable for transplant can sometimes turn into a favourable candidate. Unfortunately, it is also seen that patients having normal PVR pre-operatively do not mean they do not have the potential of having a rise in PVR and RV failure post-transplantation. Myocardial protection and deleterious effects of CPB have a role to play in such cases.

Though the cause may be multi-factorial, RV failure occurs due the donor heart not being able to adjust to a high pulmonary after-load, as that of the recipient. Inadequate myocardial protection and reperfusion injury may add to the insult. RV failure results in ventricular dilatation, ischemia, and poor contractility which results in IVS deviation that in turn compromises the LV output. The important clinical strategies that help in optimizing the RV hemodynamics are maximizing coronary perfusion through maintenance of aortic pressure, reducing pre-load to a distended and ischemic RV, decreasing RV after-load by reducing PVR, optimizing myocardial oxygen delivery, and limiting ventricular oxygen consumption [2]. Arrhythmias and conduction disturbances should be treated accordingly, to maintain a good cardiac output. In summary, the basic tenets of treatment of RV failure are judicious administration of intravenous (IV) fluids, high inhaled fraction of inspired oxygen (FiO₂) to facilitate pulmonary vasodilatation, and inotropic support. Milrinone and levosimendan form the mainstay of initial therapy. Inhaled NO is instituted before leaving the operating room, where the initial therapy is not very effective. “Conditioning” of the vascular bed is tried with inhaled NO, in patients with known PH, by initiating NO therapy even before the initiation of CPB [31]. IABP may help to tide over RV dysfunction resulting from ischemia or reperfusion injury. Finally, a crucial decision regarding the need for right ventricular assist device (RVAD) implantation has to be made in the operating room, with the decision based on multiple parameters such as overall hemodynamics, size, and function of the ventricles, as estimated by the transesophageal echocardiography (TEE), oxygenation, and renal function. Effective management of the RV in the post-operative period requires a through planning and evaluation of the RV, prior to the operation [31].

RV failure after LVAD implantation

The use of LVADs has increased over the last 2 decades serving mostly as a bridge to heart transplantation, bridge to recovery, or as a destination therapy [35, 36]. It has proven to have increased the quality of life in those with end-stage heart failure by improving end-organ function and improving functional capacity. A broad spectrum of mild RV dysfunction to a fulminant RV failure can happen in patients after LVAD.

- a) Pathophysiology: After a successful LVAD implantation, the output of the RV has to increase to match the LVAD work. With the successful working of LVAD, the pre-load on the RV increases and, therefore, RV after-load has to be decreased in order to improve compliance. This in turn causes the pulmonary capillary wedge pressure (PCWP), PAP, and RV systolic pressure (RVSP) to reduce. However, leftward IVS shift and change in contractility pattern after LVAD implantation (especially in LV unloading) may impair RV contractility. In theory, the reduction in the left atrial (LA) pressure is favourable for the RV, but in reality, the leftward IVS more prominently contributes to RV dysfunction. Any pre-existing TR can worsen after the LVAD implantation, secondary to abnormal position of the IVS and high LVAD flows, which distort the tricuspid annulus. Other causes leading to RV failure are post-CPB inflammatory response, increased PVR following the pump run, increased pre-load, and blood product transfusion [37].
- b) Pre-operative risk factors for post-operative right heart failure are as follows [38]:
- 1) Indication of LVAD destination therapy has a higher risk than bridge to recovery or transplantation [39]
 - 2) Female sex is at higher risk compared to male [38]
 - 3) Pre-operative circulatory failure (need for inotropes/mechanical support/IABP) [40]
 - 4) Pre-operative end-organ dysfunction—patients on ventilator, liver dysfunction, renal dysfunction, coagulation abnormalities, presence of sepsis, and low platelet count [40]
 - 5) Pre-operative severe RV dysfunction [41]
 - 6) Presence of pulmonary vascular disease [38]
 - 7) Others include non-ischemic cardiomyopathy, redo surgery, presence of pre-operative TR [37, 38]

Conversely, the following haemodynamic parameters indicate a less likelihood of patient developing a RV failure after LVAD implantation [38]:

- CVP \leq 8 mmHg; • PCWP \leq 18 mmHg; • CVP/PCWP \leq 0.66; • PVR \leq 2 WU; • Right ventricular stroke work index (RVSWI) \geq 400 mmHg mL/m².

Risk scoring systems have been proposed by Matthews JC, Fitzpatrick JR 3rd, and Drakos SG [40, 42, 43] and but none have been validated on prospective studies [44].

iii) Diagnosis and classification of right heart failure after LVAD

Right heart failure is diagnosed by the following criteria [45]:

A) Documentation of elevated CVP by:

1. Direct measurement (e.g. right heart catheterization) with evidence of a CVP or RA pressure (RAP) $>$ 16 mmHg.
2. Findings of significantly dilated inferior vena cava with absence of inspiratory variation by echocardiography.
3. Clinical findings of elevated jugular venous distension at least halfway up the neck in an upright patient.

B) Manifestations of elevated central venous pressure characterized by:

1. Clinical findings of peripheral edema (\geq 2 + either new or unresolved),
2. Presence of ascites or palpable hepatomegaly on physical examination (unmistakable abdominal contour) or by diagnostic imaging,
3. Laboratory evidence of worsening hepatic (total bilirubin $>$ 2.0 mg/dL) or renal dysfunction (creatinine $>$ 2.0 mg/dL).

If the patient meets the definition for right heart failure, the severity of the right heart failure should be graded as mild, moderate, or severe [45] (Table 4).

iv) Pulmonary artery pulsatility index (PAPi)

The PAPi is the ratio of PA pulse pressure divided by RAP. PA pulse pressure provides an estimate of RV pulsatile load and contractile strength. By normalizing PA pulse pressure to RAP, the PAPi incorporates RV congestion as another indicator of RV failure. PAPi $<$ 1.0 was a highly sensitive indicator of RV failure in the setting of an acute myocardial infarction [46] and PAPi $<$ 1.85 was a sensitive predictor of RV failure after LVAD implantation [47].

v) Prevention of right heart failure

The main stay of prevention of RV failure lies in optimizing the pre-load, contractility, and after-load during the perioperative period. Adequate ventilation, maintaining acid–base balance, sinus rhythm, and temperature play crucial roles [41]. Pre-load is optimized by aggressive diuresis or continuous veno-venous haemodialysis to maintain CVP below 15 cm of water. Pre-operative use of pulmonary vasodilators, when PAP is high, and the use of IABP to improve contractility are other means adopted to protect the RV [41].

vi) Management of RV failure after LVAD:

The inotropes of choice in the management of RV failure are milrinone, levosimendan, and dobutamine. They improve contractility and cause pulmonary vasodilation. Epinephrine

Table 4 Right heart failure after LVAD-severity grade [40]**Mild right heart failure**

Post-implant inotropes, inhaled nitric oxide, or intravenous vasodilators not continued beyond post-op day 7 following VAD implant

No inotropes continued beyond post-op day 7 following VAD implant

Moderate right heart failure

Post-implant inotropes, inhaled nitric oxide, or intravenous vasodilators continued beyond post-op day 7 and up to post-op day 14 following VAD implant

Severe right heart failure

CVP or right atrial pressure greater than 16 mm Hg

Prolonged post-implant inotropes, inhaled nitric oxide, or intravenous vasodilators continued beyond post-op day 14 following VAD implant

Severe-acute right heart failure

CVP or right atrial pressure greater than 16 mmHg

Need for right ventricular assist device at any time following VAD implant

Death during the VAD implants hospitalization with right heart failure as the primary cause

CVP, central venous pressure; VAD, ventricular assist device; LVAD, left ventricular assist device; *Post-op*, post-operative

can be used to maintain an adequate systemic perfusion, to maintain coronary perfusion. Duration of inotropes has a strong correlation with mortality; hence, early weaning of inotropes is highly recommended, if feasible. Heart rate must be optimal between 80 and 100 beats per min. The drugs that are commonly used in achieving this target are magnesium sulphate (MgSO₄), digoxin, ivabradine if the heart rate is high and dual chamber (DDD) pacing or isoproterenol if the heart rate is low. Amiodarone and lidocaine are used in restoring sinus rhythm [37, 41].

There are fewer roles for surgical management of RV failure except in the following situations. A worsening TR, due to the shift of IVS with a dilated tricuspid annulus, may need correction, as this will help in maintaining venous flow and renal perfusion and significantly reduces post-operative morbidity. There are sporadic reports indicating that creation of a PA to LA shunt helps in reducing the RV after-load and improves function [48, 49]. Intra-operatively, RV failure is recognized when the cardiac index (CI) remains less than 2.0 L/min/m² and CVP is greater than 20 mmHg. Weaning from CPB is not feasible in such situations and a temporary RVAD may need to be considered [50, 51]. During the ventricular assist device (VAD) implantation, meticulous surgical technique, reduced CPB time, maintaining a low PEEP during ventilation, adequate de-airing, and adjustment of LVAD flows under TEE guidance are all factors that play an important role in maintaining the proper configuration of the IVS [37]. Mechanical means to reduce RV after-load have been tried with devices such as PA balloon pumps with little success. Extracorporeal membrane oxygenation (ECMO) circuit is another option; however, ECMO

circuit does not decompress the ventricles as effectively as VADs [52].

Role of mechanical support devices in RV failure

RV failure, not responding to routine management, requires temporary RV support to prevent multi-organ dysfunction. IABP is commonly used to support RV failure. IABP reduces LV after-load, which in turn reduces RV after-load, and increases the coronary perfusion by diastolic pressure augmentation. The role of IABP in acute RV failure is controversial with several recent studies reporting its limited efficacy [53, 54]. Hence, RV is adequately supported only by RVAD or use of ECMO. RVAD implantation can be done percutaneously or surgically. Percutaneously implanted RVAD include axial pump (Impella®, Abiomed, USA) and centrifugal pump (Tandem Heart®, Cardiac Assist, Inc., Pittsburgh, USA; TandemLife Protek Duo® (TPD; TandemLife, USA)). Surgically implanted RVAD uses a centrifugal pump to connect RA to PA either through median sternotomy or through thoracotomy [55] using cannulas, either directly placed or through grafts sewed to RA and PA. It is an attractive option in patients with RV failure, occurring due to RV ischemia or infarct, in which RVAD can be used as a bridge to recovery [56]. However, in patients who develop RV failure due to high PVR, the use of RVAD gives suboptimal results. This is because of low flow attained due to high PVR and increased chance for lung bleeding [56]. Hence, in these conditions, venoarterial (VA) ECMO may be the optimal strategy. VA ECMO will decompress the RV, decrease the PA pressure, and maintain the cardiac output with preservation and recovery of end-organ function [57]. VA ECMO however can decrease the

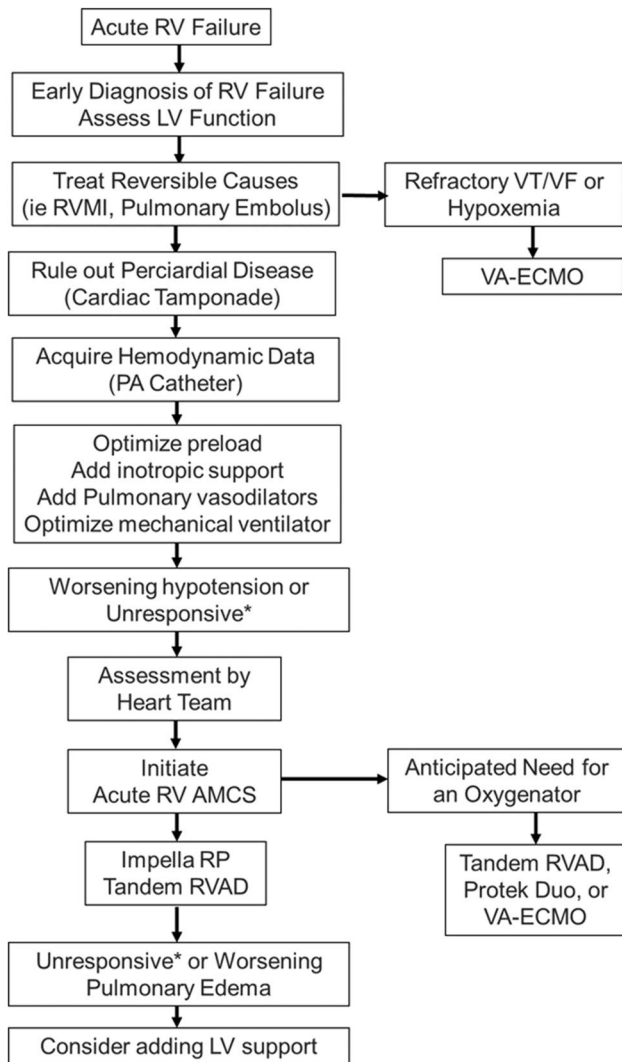


Fig. 1 Proposed algorithm for right ventricular (RV) acute mechanical circulatory support (AMCS) device use in RV failure. LV, left ventricular; PA, pulmonary artery; RVAD, right ventricular assist device; RVMI, right ventricular myocardial infarction; VA ECMO, venoarterial extracorporeal membrane oxygenation; and VT/VF, ventricular tachycardia/ventricular fibrillation. *Unresponsive defined by new or persistent systolic blood pressure <90 mmHg, or cardiac index <2.2 L/min/m², requiring ≥1 inotrope or vasopressor, worsening end-organ perfusion (reproduced with permission from ref [55])

native LV output. The high pressure from the arterial blood delivered retrograde to descending aorta can increase the LV after-load, which can lead to LV failure and increase in the PVR. In these situations, decreasing the ECMO flow or using LV venting or by placing an Impella® device to offload the LV should be considered [58]. However, in cases where the increased PVR is irreversible, weaning from VA ECMO is problematic and it may be considered as a bridge to heart lung transplant [59]. The proposed algorithm for RV mechanical support is depicted in Fig. 1 (reproduced with permission [55]).

RV failure in congenital cardiac surgery

The most extreme cases of right heart failure can occur in patients with congenital heart disease, including those with grown up congenital heart disease. Ebstein anomaly is a classic example; post-operative patients being followed up with pulmonary regurgitation after repair of Tetralogy of Fallot in childhood might be the other common example. Given the diverse anatomic substrate, the variegated presentation, and other factors, this is outside the scope of this review. An open sternum is sometimes a relatively easy to implement treatment modality in the perioperative period for patients whose right-sided chamber function deteriorates during chest closure; this receives affirmation from clinical practice guidelines on the subject [60]. The mechanisms of relief have been described in the first part of this article.

Conclusion

Severe RV dysfunction or failure requires proactive management strategy. For patients deemed low risk for development of RV failure, when it occurs, the correction of underlying cause is the most important and effective treatment strategy. Protamine reaction, even though unpredictable, is very rare. Pre-load and after-load optimization, ventilator adjustments, and improving the contractility of RV by inotropes are the first line of therapy and should be initiated early to prevent multi-organ damage. Mechanical assist device implantation or circulatory support with ECMO may be needed in refractory cases.

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Informed consent Not required.

Animal and human rights Not required being that this is a review article.

Conflict of interest The authors declare no competing interests.

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