Right Ventricular Failure After Cardiac Surgery: Management Strategies

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Right ventricular failure after cardiac surgery is a difficult clinical dilemma. We review the physiology of right ventricular failure in addition to current management strategies to address it.

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Right ventricular failure after cardiac surgery is an uncommon but potentially catastrophic complication. Being a system in series, the normal left heart can only provide a stroke volume based on its preload from the right ventricle (RV). Therefore, a failing RV presents a magnificent task to manage in the postoperative cardiac surgical patient. Although much understanding has been achieved in left heart failure, understanding of the right heart remains more illusive with regard to first considering it as a possible diagnosis for a floundering postoperative patient, then proceeding to confirm the diagnosis, and finally managing this clinical problem.

Here, we briefly review the right ventricular anatomy and physiology followed by focused attention on etiologies, diagnosis, and management of RV dysfunction/failure after cardiac surgery.

RIGHT VENTRICULAR ANATOMY AND PHYSIOLOGY

The RV is triangular shaped from a lateral view and crescent shaped in its cross section with coarse trabeculations and an interventricular septum that is convexly curved toward the RV.1 Compared with the left ventricle (LV), the RV is thin walled and highly compliant with a low-pressure pulmonary arterial system. Being a system in series, in the absence of significant shunts or valvular regurgitation, the RV ejects the same amount of stroke volume as the LV.2

Like the LV, the physiology of the RV follows the Frank-starling relationship of the heart and is closely linked to preload, contractility, and afterload. An abnormality in any one of these three parameters may lead to RV dysfunction, and therefore, treatment strategies revolve around optimizing these factors.

The preload of the RV (end-diastolic RV volume (EDRVV)) determines RV stroke volume via two mechanisms: directly by its sheer volume, and indirectly by its effect on RV contractility. In clinical practice, EDRVV is not readily available so end-diastolic RV pressure or right atrial pressure (RAP) is substituted as an indicator of preload. Being highly compliant, the RV distends as preload is increased—unfortunately this can be to the detriment of the chamber. As preload increases, the RV-free wall distends and the interventricular septum changes from being convex to concave bowing into the LV. This phenomenon known as ventricular interdependence eliminates the septum’s role in RV contractility and decreases left ventricular compliance.3,4 The preload is further affected by atrial contraction, which contributes a significant (10%-40%) portion of RV preload depending on RV compliance and function. In situations where RV compliance is diminished (eg, RV infarction), the atrial contraction is vital to maintaining preload. In addition, as heart rate increases, diastolic filling time is diminished and the atrial contraction plays a larger role in preload.3,5

The afterload of the RV is primarily determined by the pulmonary valve, the pulmonary vasculature resistance, and the left heart function. In clinical practice, pulmonary vascular resistance (PVR) is widely used as an afterload indicator and is a function of pulmonary arterial pressure (PAP), left atrial pres-
SURE, and cardiac output (CO). Chronically, slow increases in this afterload allow time for the RV to compensate by correspondingly increasing its mass. However, acute increases in RV afterload are not well tolerated.

Contractility is the ability of the RV myocardium to generate force. The relatively low-resistance pulmonary vasculature requires the stroke work of the RV to be about one-fourth that of the LV.8 Hence, the RV muscle is much smaller in mass compared with the LV.

ETIOLOGIES OF RV FAILURE

A number of preoperative, intraoperative, and postoperative factors can affect the preload, contractility, and afterload of the RV. Capillary leak, third spacing, copious urine output, and bleeding are common after cardiac surgery, leading to intravascular hypovolemia. Conversely, acute excessive fluid or transfusion administration can also impair RV function. Failed tricuspid or pulmonary valve repairs leading to moderate-severe regurgitation may result in excessive preload and subsequent RV failure. Extracardiac factors such as of positive-pressure mechanical ventilation with positive end-expiratory pressure (PEEP) have been shown to decrease preload by increasing intrathoracic pressure and increasing afterload by changing transpulmonary dis-tending pressures.9

RV contractility can be compromised by several intraoperative factors, such as suboptimal myocardial protection, myocardial stunning after long durations on cardiopulmonary bypass, air or thromboembolism to the right coronary artery, and mechanical occlusion or kinking of the right coronary button or bypass graft. RV contractility is also depressed by postoperative complications, such as sepsis, arrhythmias, and volume overload. Postoperative LV dysfunction can lead to hypoperfusion of the RV, whereas preoperative RV dysfunction is obviously exacerbated by these perioperative factors.

Factors affecting the PVR present the primary determinants of RV afterload. Increases in RV afterload are caused by pulmonary vasoconstriction, pulmonary vascular bed compression or reduction, and mechanical obstruction. Pulmonary vasoconstriction is induced by hypoxia, hypercarbia, metabolic acidosis, protease, and cytokine release often associated with large transfusions and cardiopulmonary bypass. Afterload may also be increased if the pulmonary vascular bed is compressed or reduced by mechanical ventilation/PEEP, acute respiratory distress syndrome, pulmonary embolism, and pneumothorax. A failing left heart postoperatively results in rising left atrial pressures, leading to increased PAP’s. Preexisting elevated RV afterload because of left-sided valvular disease or intrinsic lung disease can be exacerbated after cardiac surgery. Etiologies of RV dysfunction based on Frank-starling relationship of the heart are summarized in Figure 1.

In recent years, an increase incidence of RV failure has occurred secondary to the successful implantation of left ventricular assist devices (LVADs). The incidence has been reported to be 13% in some reports.8 A number of mechanisms have been suggested, including a leftward shift in the ventricular septum, leading to increased RV preload and a subsequent increase in wall tension beyond its optimal range, leading to increased wall stress and diminished contractility.8 Other theories revolve around the increased systemic blood flow from the LVAD, which increases RV preload further leading to RV dilatation, greater tricuspid regurgitation (TR), and in return a vicious cycle which ensues.8 Finally, the RV function may have been poor to begin with and unveils itself once the preload increases.10

Preoperative TR, either primary or secondary to left side lesion, is intimately related to RV function. Severe TR is frequently associated with RV dysfunction.11 Postoperative residual TR makes the management of RV dysfunction even more challenging; therefore, a more liberal application of tricuspid anuloplasty should be considered in patients with a dilated tricuspid annulus and/or moderate TR.12

DIAGNOSING RV FAILURE IN THE CARDIAC SURGICAL INTENSIVE CARE UNIT

RV function is a reflection of preload, afterload, and contractility. Because of its complex geometry and high susceptibility to loading conditions, accurate assessments of RV function are challenging and require an integrative approach. Although newer modalities such as 3-dimensional echocardiography are promising, pulmonary artery catheters and 2-dimensional echocardiography are still the gold standard tools used for assessment of RV function in clinical practice. Magnetic resonance imaging is an excellent tool for assessing RV function, but its role is limited in the intensive care patient because of the inability of these ill patients to undergo the lengthy investigation. Pulmonary artery catheters provide important hemodynamic parameters, such as RAP, PAP, CO, mixed venous oxygen saturation, PVR, and RV stroke work index (RVSWI). The RVSWI incorporates the mean PAP, RAP, heart rate, and cardiac index into its formulation, providing an assessment of RV function. Intrinsic to the formula is the differ-
ence between the mean PAP and RAP. If this difference is small, the RVSWI will be small, suggesting that the RV is providing very little contractility to blood entering its chamber.

The American Society of Echocardiography strongly recommends routine use of at least one of the following parameters of RV systolic function. These include tricuspid annular plane systolic excursion, RV fractional area change, tissue imaging of tricuspid annular velocity, and RV index of myocardial performance (Tei index). RV size and wall thickness can be readily available by echocardiography and should also be taken into account in the assessment of RV function as a part of integrative approach. RV size measurements (basal, midcavity, and base-to-apex) are best performed in apical or midesophageal 4 chamber views. Although ejection fraction can be routinely calculated for the RV, its complex geometry, lack of standard measurement techniques, and variability based on preload and afterload results in the RV ejection fraction being precise but not always accurate. Therefore, despite these methods, assessment of the RV in clinical practice often remains mostly qualitative. Practical parameters used to assess the RV are listed in Table 1.

MANAGEMENT OF RV FAILURE

The goal of treatment in RV failure is to optimize preload, reduce RV afterload, and increase RV contractility. With this in mind, we review current treatment options in RV failure.

INTRAVENTRICULAR INOTROPES

Dobutamine

Dobutamine is primarily a beta agonist with minimal alpha receptor agonist activity. It works through $\beta_1$ receptor-mediated increases in myocar-
Dial contractility and $\beta_2$ stimulation, which induces vasodilation and decreases afterload. Dobutamine functions by increasing cyclic adenosine monophosphate (cAMP) levels, resulting in inotropic stimulation. At doses up to 5 $\mu$g/kg/min, dobutamine increases myocardial contractility and reduces PVR and systemic vascular resistance (SVR). At doses exceeding 10 $\mu$g/kg/min, dobutamine leads to tachycardia and increased oxygen consumption without providing any additional improvement in PVR.\textsuperscript{14}

**Isoproterenol**
Isoproterenol is a nonselective beta agonist. It has both positive inotropic and chronotropic properties, thereby increasing CO. It also produces pulmonary and peripheral vasodilation.\textsuperscript{15}

**Milrinone**
Milrinone is a selective phosphodiesterase III inhibitor. It exerts a positive inotropic action as well as a vascular smooth muscle relaxing effect, resulting in decreasing PVR, SVR, and increasing RV contractility.\textsuperscript{16} Because systemic vasodilation occurs, there is often the need to coadminister pressors. Milrinone functions by increasing cAMP levels.\textsuperscript{3} Milrinone and dobutamine can work synergistically because both drugs increase cAMP levels via 2 separate mechanisms.

**Levosimendan**
Levosimendan sensitizes troponin C to intracellular calcium, resulting in increased contractility without increased oxygen consumption.\textsuperscript{17,18} It also has a vasodilatory effect by opening adenosine triphosphate-sensitive potassium channels in smooth muscle to cause smooth muscle relaxation.\textsuperscript{19} This results in improved diastolic function, decrease in PVR, and improved myocardial contractility without increasing oxygen consumption.

**PULMONARY VASODILATORS**
Pulmonary vasodilators should be used in conjunction with drugs used to increase RV contractility. Systemic administration of pulmonary vasodilators may also reduce systemic blood pressure. Hypotension can exacerbate RV ischemia and drop RV contractility. Intravenous pulmonary vasodilators run the risk of worsening ventilation-perfusion matching.\textsuperscript{20}

**Inhaled Nitric Oxide (iNO)**
Nitric oxide is a potent, rapidly acting, and selective pulmonary vasodilator. It decreases PVR by stimulating cyclic guanosine monophosphate release in smooth muscle cells. Rapid inactivation by hemoglobin in the pulmonary capillaries prevents systemic vasodilation.\textsuperscript{21} Nitric oxide has a short half-life, so it needs to be continuously delivered into the

<table>
<thead>
<tr>
<th>Parameters Used in Assessing Right Ventricular Function</th>
<th>Reference Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary artery catheter</td>
<td></td>
</tr>
<tr>
<td>Right atrial pressure</td>
<td>1-6 mm Hg</td>
</tr>
<tr>
<td>Pulmonary systolic artery pressure</td>
<td>15-30 mm Hg</td>
</tr>
<tr>
<td>Pulmonary diastolic artery pressure</td>
<td>6-12 mm Hg</td>
</tr>
<tr>
<td>Pulmonary capillary wedge pressure</td>
<td>6-12 mm Hg</td>
</tr>
<tr>
<td>Cardiac output</td>
<td>2.4-4 L/min/m²</td>
</tr>
<tr>
<td>Pulmonary artery resistance index</td>
<td>200-400 dyne·sec(^{-1})·cm(^{-2})/m²</td>
</tr>
<tr>
<td>RV stroke work index</td>
<td>4-8 g m/m²</td>
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<tr>
<td>RVEF</td>
<td>46%-50%</td>
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</tbody>
</table>

Echocardiography

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Reference Range</th>
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<tbody>
<tr>
<td>Visual estimation</td>
<td></td>
</tr>
<tr>
<td>Fractional area change</td>
<td>&gt;35%</td>
</tr>
<tr>
<td>Tricuspid annular plane systolic excursion</td>
<td>&gt;1.6 cm</td>
</tr>
<tr>
<td>Pulsed Doppler peak velocity at the annulus</td>
<td>&lt;10 cm/s</td>
</tr>
<tr>
<td>Pulsed Doppler myocardial performance index</td>
<td>&gt;0.40</td>
</tr>
<tr>
<td>Tissue Doppler myocardial performance index</td>
<td>&gt;0.55</td>
</tr>
<tr>
<td>RV diameter (basal) at end diastole</td>
<td>2.4-4.2 cm</td>
</tr>
<tr>
<td>RV diameter (midcavity)</td>
<td>2.0-3.5 cm</td>
</tr>
<tr>
<td>RV diameter (base-to-apex)</td>
<td>5.6-8.6 cm</td>
</tr>
<tr>
<td>Wall thickness</td>
<td>&lt;0.5 cm</td>
</tr>
</tbody>
</table>

RV, right ventricle.
ventilator circuit. Because nitric oxide is delivered as an inhalational agent, it only reaches ventilated regions of the lung, dilating those capillaries, resulting in better ventilation-perfusion matching and oxygenation. However, intravenous pulmonary vasodilators dilate all the lung capillaries. This nonselectivity can lead to increasing pulmonary blood flow to regions of poorly ventilated blood, causing ventilation-perfusion mismatching. iNO provides synergistic pulmonary vasodilation with intravenous prostacyclin, inhaled iloprost, and oral sildenafil. The use of iNO is limited by potential methemoglobinemia and rebound pulmonary hypertension after rapid discontinuation.22

Prostaglandin E1 and Prostacyclin

Prostaglandin E1 and prostacyclin are both potent pulmonary vasodilators. As a result, they reduce PVR, improve RV performance, and increase CO. Epoprostenol is an intravenous prostacyclin with a short half-life and is often used in the intensive care. It decreases PAP and PVR and increases CO; however, its use is limited by the systemic hypotension that ensues.23 Inhaled iloprost is a potent pulmonary vasodilator with antiplatelet and antiproliferative effects. As it is not an intravenous infusion there is less resulting hypotension.

Phosphodiesterase 5 (PDE5) Inhibitors

PDE5 inhibitors (eg, sildenafil) increase downstream cyclic guanosine monophosphate signaling by blocking its degradation. PDE5 inhibitors reduce PVR but also reduce SVR. They also can exert milrinone-like effects through phosphodiesterase III inhibition, improving RV function.24 Sildenafil has been shown to act synergistically with iNO and to decrease rebound pulmonary hypertension after iNO withdrawal.25

Endothelin Receptor Antagonists

Endothelin receptor antagonists block endothelin A and B receptors in vascular smooth muscle, resulting in decreased PAP. Its use is limited by relatively long half-lives and potential hepatotoxicity.26

VENTILATORY MANAGEMENT

Positive-pressure inspiration increases intrathoracic pressure and RAP, which result in a decrease in RV filling and RV stroke volume. During spontaneous inspiration, the opposite happens. There is a decrease in intrathoracic pressure and RAP, increasing RV filling and RV stroke volume. These changes in EDRVV will eventually have an effect on LV diastolic compliance through ventricular interdependence. Continued dilation of the RV contributes to worsening RV failure, and as the ventricular septum shifts toward the LV, continued dilation of the RV causes a decrease in LV diastolic compliance and decreased LV end-diastolic volume and CO.

Changing of lung volumes alters PVR. Hyperinflation leads to marked increases in PVR by increasing transpulmonary pressure. Increases in transmural pulmonary artery pressure increases RV afterload, which impedes RV ejection, thereby decreasing RV stroke volume and ultimately resulting in RV dilation. If RV dilation and RV pressure overload persists, ischemia of the RV may ensue with potential progressive failure. PVR also increases at small lung volumes owing to the combined effect of hypoxic pulmonary vasoconstriction and extra-alveolar vessel collapse. PVR is minimal at functional residual capacity.

High levels of PEEP can narrow the capillaries in the well-ventilated lung areas, diverting blood flow to those areas, increasing ventilation-perfusion mismatch, and leading to worsening hypoxia.27 Studies conducted on pigs demonstrated a significant decrease in inferior cava flow and RV CO even for low PEEP levels of 5 and 10 cm H2O. The most pronounced impairment of both took place in the inspiration phase.28 This finding is further exacerbated in hypovolemic patients.

Hypoxic pulmonary vasoconstriction is a phenomenon exclusive to the pulmonary vasculature. Pulmonary vessels constrict with hypoxia and relax in the presence of hyperoxia. The same increase in tone occurs during acidemia. This reaction is mediated by a decrease in nitric oxide synthesis and release as well as changes in intracellular calcium fluxes in pulmonary vascular smooth muscle. Nitric oxide synthesis is inhibited by both hypoxia and acidosis. Optimal ventilator management consists of the use of low tidal volumes and low PEEP with strict avoidance of hypercapnia and acidosis.7

VOLUME MANAGEMENT

In the setting of RV dysfunction, both hypovolemia and hypervolemia will reduce CO. If preload is too low, RV CO will not be adequate. When the RV is excessively distended, wall tension increases according to the Frank-Starling mechanism and muscle fiber length is increased, beyond a certain point at which ventricular function will fail. RV volume overload is very important to recognize and treat promptly. Not only does a dilated RV lead to RV failure, but as the ventricular septum shifts toward the LV, there is a decrease in LV diastolic compliance and LV end-diastolic volume resulting in decreased
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CO. Management involves fluid removal by using diuretics or hemofiltration. When administering intravenous fluids, an increase in RAP without a concomitant increase in CO would indicate that no further volume replacement is necessary.

MECHANICAL SUPPORT AND SURGICAL TREATMENT STRATEGIES

For intractable RV failure that fails to respond to aggressive medical therapy, one must consider the use of temporary right ventricular mechanical support. Counterpulsation with an intra-aortic balloon pump is an option to consider in patients who need slightly more support than medical therapy can provide. Although the intra-aortic balloon pump does not decrease RV afterload, it does improve coronary perfusion and, therefore, may provide the additional support required to see the patient through the perioperative period. Temporary right ventricular support has been used frequently for RV failure after LVAD implantation. As mentioned previously, severe TR can worsen RV failure, and therefore, some have advocated a liberal approach to tricuspid valve annuloplasty at the time of LVAD implantation to avoid this complication. Mechanical support with continuous flow pump support can be considered using central or peripheral cannulation techniques. Cannulation strategies can be venoarterial (extracorporeal membrane oxygenation) with an oxygenator, or right atrial to pulmonary artery with/without an oxygenator, depending on the presence of lung injury. Most often right ventricular assist device (RVAD) support is provided with extracorporeal devices. There have been some case reports of using implantable continuous axial flow pumps to support both the LV and RV, but widespread use of this technique has not disseminated because of persistent complications with pulmonary overcirculation. The most common set-up for mechanical right ventricular support is using a continuous flow extracorporeal pump with right atrial and pulmonary artery cannulation. Using this configuration, the chest can be closed because the cannulae can be tunneled through the skin. RVAD support can be slowly weaned, and using serial echocardiography, a decision can be made regarding explantation.

CONCLUSIONS

Right ventricular failure is indeed a great management challenge in the postoperative cardiac surgical patient. Keys to successful treatment are identification of patients at greatest risk, an early suspicion for dysfunction peripheratively, and finally a broad management approach with experienced critical care support. Using these strategies, successful outcomes can be achieved.


