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Acute and Chronic Right Heart Failure: A Narrative Review of Mechanical Circulatory Support

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Abstract

Historically, the Right Ventricle (RV) has been an underrepresented component of the growing body of literature regarding the circulatory system. Unfortunately, evidence has shown RV failure is closely associated with hospital admissions and increased short- and long-term mortality. RV failure can be categorized as acute or chronic, but the primary mechanisms underlying the development of its decompensation are diminished contractility, circulatory overload, and pressure overload. While medical management of Right Heart Failure (RHF) has its role in hemodynamic optimization, not all patients respond as expected. The role of mechanical circulatory support MCS continues to grow as more options become available. This review aims to present an overview of the physiology of the RV as it relates to pulmonary circulation, the pathophysiology of right ventricular failure, distinctions between acute and chronic developments, medical management, and options available for mechanical circulatory support.

Abbreviations: RV: Right Ventricle; RHF: Right Heart Failure; MCS: Mechanical Circulatory Support; LV: Left Ventricle; TF: Transcription Factors; PVR: pulmonary vascular resistance; RHC: Right Heart Catheterization; RAP: Right Atrial Pressure; RA: Right Atrium; PA: Pulmonary Artery; PAWP: Pulmonary Artery Wedge Pressure; CO: Cardiac Output; ARHF: Acute right heart failure; PE: pulmonary embolism; RVMI: Right Ventricle Myocardial Infarction; MI: Myocardial Infarction; PAH: Pulmonary Arterial Hypertension; CHD: Congenital Heart Disease; IABP: Intra-Aortic Balloon Pumps; LVAD: Left Ventricular Assistive Device; RVAD: Right Ventricular Assistive Device; MVAD: Miniature Ventricular Assist Device; VA-ECMO: Veno-Arterial Extracorporeal Membrane Oxygenation; SVC: Superior Vena Cava; IVC: Inferior Vena Cava.

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The Right Ventricle and Pulmonary Physiology

The differences between the RV and left ventricle (LV) begin at development. Progenitor cell populations known as heart fields will grow into their respective ventricle based on cellular markers and Transcription Factors (TF) [1-3]. TF Islet-1 is expressed in cells that will develop into the future RV but not be expressed in future LV cells [1]. In utero, the RV has a similar wall thickness as the LV while being the dominant chamber. At birth, a dramatic decrease in Pulmonary Vascular Resistance (PVR) allows for the regression of wall thickness. Generally, the structure of the RV will be thin-walled, crescent-shaped, and have a mass of 1/6 that of the LV, despite supplying the lungs with 100% of our body's cardiac output [1-3].

The fundamental role of the RV is to provide the lungs with deoxygenated blood for gas exchange. With our first breath, the pulmonary circulation instantly becomes a low-pressure circuit at rest and during exercise. This contrasts with systemic circulation, where increased cardiac output during exercise is associated with a significant increase in blood pressure. The difference between the two circulations is based on the concept of recruitment. As right ventricular cardiac output increases, vessel circuits are added in parallel to the existing capillary network, obviating any rise in pulmonary arterial pressure [51]. The ability of the lung to selectively perfusion regions of the lung based on ventilation as well as body position is fundamental to the model proposed by Dr. West in 1964, in which he categorized the lung fields into Zones 1 through 3 based on the degree of blood flow and aeration of alveoli, pulmonary arterioles, and venules [52]. With this model in mind, we find that blood flow through the lungs depends on the relationship between the driving pressure between the pulmonary arterial and venous systems and the trans mural pressure exerted on the pulmonary vessels by alveolar distention. Pulmonary vessels, unlike systemic vessels, have much higher compliance and act as compressible resistors (i.e., Starling resistors). Therefore, inspiration-associated over-distention of alveoli will compress these small vessels and consequently increase RV afterload, thus impeding RV ejection during inspiration [53].

Two determinants of proper RV function are preload and afterload. Traditionally, echocardiography provides a non-invasive and accessible method of measuring RV function. However, a patient's body habitus or unskilled user may diminish the exam quality. Fortunately, Right Heart Catheterization (RHC) is an invasive method for measuring intracardiac pressures. Through this, we can measure Right Atrial Pressure (RAP), Pulmonary Artery Wedge Pressure (PAWP), and cardiac output (CO) [4]. The most straightforward approach to quantify RV dysfunction is measuring the ratio of RA to PAWP [5].

Right Heart Failure

Right Heart Failure (RHF) has had multiple proposed definitions over the years, but none have gained formal acceptance. In 2008, the European Society of Cardiology defined RHF as "a common clinical manifestation of heart failure characterized by breathlessness, fatigue, evidence of RV dysfunction, raised jugular venous pressure, peripheral edema, hepatomegaly, and gut congestion."7 By 2018, the American Heart Association proposed RHF to be defined as "a clinical syndrome with signs and symptoms of heart failure resulting from abnormal RV structure or function, caused by the inability of the RV to support optimal circulation…"7 Despite the varying verbiage the core concept is that the RV is unable to fill or reject blood. Several factors contribute to low cardiac output (CO) in the setting of RV failure, such as RV systolic dysfunction, tricuspid regurgitation, tachyarrhythmias, or poor preload [1-3,6,7].

The progression of RHF is dependent on the RV's ability to adapt to injury or stress. The patient presentation can range from asymptomatic to refractory RHF [5,6]. Key factors such as the type/severity of the injury, chronicity, and age at the time of onset will determine RV adaptation [6-8]. Significant consideration must also be paid to neurohormonal activation, gene expression, and pattern of ventricular remodeling for their roles in the progression of RHF. Excessive sympathetic adrenergic stimulation adversely affects ventricular remodelling [7,8]. Elevated catecholamine levels associated with pulmonary artery hypertension increase pulmonary vascular resistance, thus lowering cardiac index [6-8].

LV systolic function directly correlates with the prevalence and severity of right ventricular disease (RVD) [7]. One study comparing 100 heart failure patients with both reduced and preserved ejection fraction, found a prevalence of 50% of RVD in preserved ejection, but 73% in reduced. Furthermore, RVD complicating heart failure with reduced ejection fracture is associated with a 2.4 increase in the risk of mortality compared to no RVD [7]. Ventricular interdependence plays a vital role in RHF, especially in the acute setting, as dilatation can cause a leftward shift of the interventricular septum and compromise the LV [1,3,7,8].

Acute Right Heart Failure

Acute Right Heart Failure (ARHF) results from impaired forward flow secondary to impaired function of the ventricle, valves, or conduits. Developing ARHF is associated with a mortality ranging from 6% to 14%.1 The RV is thin-walled because the highly compliant, low-resistance pulmonary circuit allows forward flow with low peak systolic pressure. This has the added advantage of avoiding the need for isovolumic contraction and relaxation phases, as seen with the LV. Thus, the RV adapts better to volume overload than pressure overload or contractile dysfunction [1,7,8].

The most common cause of acute RV pressure overload is a pulmonary embolism (PE) [8]. The anatomic location as well as clot burden are closely related to the degree of RV failure and hemodynamic instability.1,8 The initial obstruction leads to an immediate increase in PVR but is exacerbated by hypoxic pulmonary vasoconstriction, secondary to a ventilation-perfusion mismatch [1]. The RV can momentarily adapt to the pressure overload before RV dilatation causes a leftward septal shift and compromises LV filling. Echocardiographic evidence of RV dysfunction is present in 25% to 60% of patients with a PE [7,9]. Such findings are associated with poorer prognosis and mortality ranging from 2.4 to 3.5 times greater than those without RV dysfunction [9]. Patients who are hemodynamically stable on presentation have an expected mortality of 4%, while patients in cardiogenic shock have an expected mortality between 20% and 50% [8].

An acute Right Ventricle Myocardial Infarction (RVMI) is often the cause of ARHF in the setting of inferior Myocardial Infarction (MI) [1,3-4,7,8]. Of all inferior acute ST-elevation myocardial infarction, RV involvement occurs in 30% to 50% of cases [7,8]. The disruption of blood flow via the right coronary artery, results in ischemic damage to the myocardium of the RV-free wall and interventricular septum [1-5]. Impaired contractility

reduces the RV systolic function leading to poor RV stroke volume.1,8 Ultimately, the left heart can only pump what the right heart can deliver, thus hypotension is the most commonly seen consequence of RVMI [10].

Chronic Heart Failure

Chronic heart failure is often a consequence of progressive increases in RV afterload, as seen in Pulmonary Arterial Hypertension (PAH) or mitral valve disease [1,2,6,8,11]. A hallmark adaptive change to the elevated filling pressure is myocyte hypertrophy. As the hypertrophy continues, the RV begins to undergo isovolumic phases of contraction and higher end-diastolic volume [1,11]. Eventually, RV adaptation will no longer overcome the increasing afterload leading to oxidative stress, inflammation, and impaired angiogenesis to the myocardium.1 Ultimately, myocytes are replaced by fibrotic tissue further compromising the function of the RV [1,2,11].

Although less common, chronic volume overload, as seen in tricuspid regurgitation, pulmonic stenosis/regurgitation, or septal defects, will also lead to RV failure [1,2,7,8]. Generally, these patients develop chronic RHF as a sequela from Congenital Heart Disease (CHD). The more common associations are tetralogy of Fallot, pulmonary atresia, hypoplastic left heart syndrome, and transposition of the great arteries. Interestingly, with advances in pediatric surgical technique and medical care, CHD patients are living longer into adulthood predisposing them to the progression of RHF[1].

Medical Management

Medical management of RHF begins with identifying and treating any reversible causes. While management is tailored to its specific cause, the focus is the optimization of preload, afterload, and contractility [5,8,11]. Preload should be managed with volume expansion or diuretic therapy with an emphasis on avoiding venous congestion [5,11]. Because PAH significantly increases afterload, providing locally acting or systemic pulmonary vasodilators will reduce pulmonary artery pressures and thus increase forward flow [4,5]. The significant hemodynamic changes associated with RHF can be improved with vasopressors or inotropic agents. Unfortunately, some RHF is refractory to medical management, necessitating mechanical support.

Mechanical Circulatory Support

Although medical treatment can significantly improve a patient's hemodynamics and symptomatology, treatment failure is always possible. Patients' refractory to chemical agents may benefit from Mechanical Circulatory Support (MCS). MCS has multiple indications and functions, including but not limited to bridge to transplantation or recovery. Timing of implantation is critical as outcomes are unlikely to improve after the onset of multiorgan failure.5 When selecting appropriate candidates, several factors include age, comorbidities, the potential for recovery, and advanced heart failure therapies (i.e., heart transplant) [12]. The primary role of RV support devices is to deliver blood from the Right Atrium (RA) to the Pulmonary Artery (PA) via cannulation in a continuous flow manner. This is achieved by a device's Rotations Per Minute (RPM) and the RA-PA pressure gradient [5,13]. Because the pressure gradient is relatively low compared to the LV, the set RPM is the primary determinant of flow [5,13]. However, in patients with severe PAH, the pressure gradient between the RA and PA is significantly elevated, which may hinder flow requiring higher RPMs [13].

Balloon counter-pulsation pumps

Intra-Aortic Balloon Pumps (IABPs) are among the most used circulatory support device due to their established safety and efficacy [14]. It is designed to increase myocardial oxygen supply while decreasing myocardial oxygen demand [15]. However, IABPs are designed primarily as assistive devices for the LV – inserted through the femoral artery and into the thoracic aorta. IABPs inflate during diastole to increase diastolic aortic pressure, thereby increasing coronary perfusion pressure, and maximizing oxygen supply [14]. During systole, IABPs rapidly deflate creating a negative pressure which functions to offload the struggling LV and decrease myocardial oxygen demand [14]. Generally speaking, IABPs are not ideal solutions for a failing RV – they have only indirect effects on the right ventricle by reducing LV filling pressure and, in turn, reducing RV afterload. Clinical data on the efficacy of IABPs in RV failure conflict. Whereas Krishnamoorthy et al. found that IABPs provided inadequate support for patients with biventricular failure, Boeken et al. and Arafa et al. showed that hemodynamics did indeed improve with IABP implantation in patients with primarily RV failure [16-18].

Liakopoulos et al. also found that IABPs alone did not restore RV function [19]. However, by combing the mechanical support treatment with a phenylephrine infusion investigators were able to increase both CI and SvO2 levels and restored cardiac function [19]. Recent studies have shown that IABPs improve hemodynamics in patients with out-of-proportion RV dysfunction [20]. Additionally, prolonged IABP support may optimize RV function in patients with biventricular end-stage heart failure preparing for LVAD placement-such optimization is essential for reducing the risk of RVF following LVAD implantation [21]. Given conflicting data, further study is necessary to understand the possibility of IABPs as RVADs, however, its well-established use gives it the potential to be implemented widely.

Rotary Flow RVAD

Rotary flow RVADs were first developed in the early 1990s, where they were initially deemed more effective in treating RV failure than balloon counter-pulsation pumps. Modern RVADs function through rotary pumps that transfer the rotational kinetic energy into circulation.5 Rotary blood pumps have become increasingly popular as efficiency and durability increased over the years. In addition to reducing or even eliminating ventricular work, they effectively increase and provide systemic perfusion [22].

The two types of continuous-flow rotary pumps – axial and centrifugal – differ in how they facilitate blood flow. Axial pumps are analogous to an Archimedes screw. In this system, the propeller is housed within a chamber. As the propeller turns it physically draws blood into the inlet and pushes it through the outlet to overcome resistance and pressure; the direction of blood flow is parallel to the impeller's central axis. Examples of axial pumps discussed in this section are the Impella RP and the Heart Ware MVAD. On the other hand, centrifugal pumps function through a bladed, spinning disk that captures fluid and throws it tangent to the blade tips; the direction of blood flow is perpendicular to the impeller's central axis [22,23]. An example of a centrifugal pump includes the CentriMag. Compared to axial pumps, centrifugal pumps have larger diameters, lower pump speeds, and higher hydraulic efficiency because they need not overcome suction resistance [22,23]. Due to these differences, different rotary flow pumps should be used for different patient

indications and may result in varying outcomes, even if hemodynamic performance is similar [22].

Centrimag

The Levtronix Centrimag is a pump designed for temporary uni- or biventricular support after acute cardiogenic shock. With a maximum flow rate of 10 L/min, it can effectively support patients for up to 30 days [24]. The Centrimag is unique in that its motor has no bearings – it runs off a magnetically levitated spinner, which eliminates the need for bearings, shafts, and seals on the pump. In fact, there are no moving parts except the levitating rotor. Due to minimal friction and heat generation affecting blood flow, hemolysis and thrombosis rates are significantly reduced following implantation [24]. The streamlined design allows easy use in centers not equipped with transplant programs or ventricular assist devices. Other benefits of the Centrimag include reliable device function and low rates of device-related complications [25].

As an RVAD, the Centrimag is positioned in the right atrial appendage, allowing outflow into the pulmonary artery. It provides complete right ventricular unloading and circulatory support after cardiogenic shock [25]. Though the Centrimag is an effective temporary device for RHF, overall outcomes based on survival and weaning rates have remained static over time [26]. This stagnancy is observed despite advances in care management and technology, thereby revealing that further research into RHF management is necessary [26]. Of note and for an unknown reason, female patients seem at higher risk of Centrimag weaning failure than male patients. Further study into hemodynamic differences between genders is necessary to explain why [26].

VA-ECMO

Veno-Arterial Extracorporeal Membrane Oxygenation (VA-ECMO) devices are percutaneously delivered to patients with RV failure. They contain a centrifugal pump that can pass blood at a rate of 8 L/min to decompress RV and provide complete hemodynamic support indirectly. VA-ECMO is unique because it has the ability to oxygenate blood and remove carbon dioxide through sweep gas flow [27,28]. VA-ECMO placement entails femoral artery and vein cannulation, after which a cannula is positioned either in the RA or SVC-RA junction to drain deoxygenated blood. The blood is passed through the device, oxygenated, and returned to hemodynamics by reducing RV preload, decreasing trans-pulmonary flow, reducing LV end-diastolic volume and pressure, and improving systemic perfusion; allowing for hemodynamic stabilization [27]. These devices provide complete, albeit temporary, support for patients with biventricular HF or cardiac arrest. They may be safely and quickly positioned in patients at bedside, making them reliable RVADs for emergencies.

VA-ECMO have been shown to be more effective than continuous-flow external VADs in patients with severe primary graft dysfunction in heart transplants. Those treated with VA-ECMO required less support time and experienced lower rates of renal failure and mortality [29]. However, a recent, large national cohort study of 940 LVAD patients requiring MCS for RVF found that patients treated with VA-ECMO had higher adjusted and unadjusted mortality, reoperation, and significant complication risks than those treated with traditional RVADs.30 Additionally, VA-ECMO have not achieved positive outcomes in isolated RV recovery; they should not be the primary choice for RV MCS.28 These caveats encourage further critical evaluation of VA-ECMO efficacy.

While VA-ECMOs is effective, further study is necessary to determine how HF subtypes – such as dilated LV cavities or depressed baseline LV ejections – affect outcomes [27]. To date, these variables as they relate to different levels of support have been studied computationally or in animal models; thus, more clinical data are needed to characterize these interdependent relationships accurately.

Impella RP

Similar to the TandemHeart, the Impella RP is also a minimally invasive, percutaneous device that enables direct RV bypass [31]. Inserted through the femoral vein with fluoroscopic guidance, the pump is laid across the tricuspid and pulmonary valves. Recently, a new method was used to implant the Impella RP without fluoroscopic guidance successfully; the device may be inserted through the femoral vein under transesophageal echocardiography guidance, thereby eliminating the extra step of fluoroscopy [32]. After implantation, the Impella RP unloads the right ventricle by pumping blood from the IVC directly to the outflow of the pulmonary artery; its maximum is 4.0 L/min at 33,000 rpm [31]. Thus, it allows a prompt and significant reduction of RV preload and direct augmentation of pulmonary artery flow.

The Impella RP is effective in patients with RVF of different etiologies – those who developed RVF after LVAD implantation and after cardiac surgery have benefitted from the device [33]. Implantation results in immediate, consistent, and positive hemodynamic effects while it significantly increases CI while decreasing CVP [34]. Hemodynamics remain stable even after explantation, where functional recovery of RV was seen in 78% of patients [34]. Finally, the adverse effects of the Impella RP are minimal. While some patients treated with Impella RP experienced hemolysis and excessive bleeding, these issues had no direct association with the device itself. These patients had already undergone surgical procedures or LVAD implantations, which are established causes of hemolysis [33].

Percutaneous RVAD (Protek Duo)

The Protek Duo percutaneous RVAD is a dual-lumen cannula usually inserted through the right internal jugular or subclavian veins[36]. With the proximal lumen positioned at the RA and the distal lumen to the PA, direct RV decompression and bypass may be obtained [37]. The Protek Duo has been shown safe and effective in patients with different indications for RVAD support -post-cardiotomy, primary respiratory failure, and cardiogenic shock [37]. Data also suggest that the device may have better weaning and mortality profiles than do other surgically placed RVADs.

The Protek Duo can support patients with acute RVF and those with biventricular failure when working in conjunction with a percutaneous LVAD such as Tandem Heart [37]. In addition to restoring RV function in LVAD patients, it may be used in patients with RV primary graft dysfunction following a heart transplant [39,40]. One significant advantage of the Protek Duo is that it is inserted through the upper venous system, thereby allowing patient ambulation and full mobility not possible with devices requiring femoral access. Given the fully percutaneous access site, the Protek Duo may have the potential to significantly decrease rates of bleeding, thromboembolism, and infection rates seen in other VADs [38].

Tandem Heart

The Tandem Heart is a percutaneous centrifugal pump that may support patients in cardiogenic shock or during high-risk coronary interventions. Designed initially as a left atrial-femoral artery bypass system, it may be reconfigured to act as an RVAD.42 TandemHeart utilizes a direct RV bypass system, often through bilateral femoral cannulation, or femoral and internal jugular vein canualtion. The inflow cannula may be positioned in the RA via the left femoral vein and the outflow cannula in the PA through the right femoral vein [43]. An alternative way to position the TandemHeart is through the right jugular vein; this approach has been used alongside the Protek Duo cannula and similarly this fully percutaneous, groin-free strategy is less invasive and allows patient ambulation.

In patients with cardiogenic shock, TandemHeart has been found to raise cardiac index by 20% and reduce Pulmonary Capillary Wedge Pressure (PCWP) by 18.5%; it improves hemodynamic parameters even in patients with inadequate support from an IABP.44 In addition, TandemHeart has been effectively implemented in at least one patient with severe mitral regurgitation for hemodynamic stabilization prior to mitral valve replacement [45].

HeartWare MVAD

The Heartware Miniature Ventricular Assist Device (MVAD) is a continuous axial flow pump that is the smallest of its generation. It allows a maximum flow rate of 10 L/min and operates with a wide-blade rotor design that minimizes cellular trauma [46]. Designed to require minimal surgical access; it does not require outflow graft anastomosis during implantation [47].

Human clinical data on the MVAD are minimal. The device was implanted as an LVAD in the first patient in 2015, but that trial was suspended because of the MVAD's high risk of pump thrombosis. Although, that patient, five years later, was found to have a stable clinical course with no technical malfunctions of the device.48 The MVAD is not yet commercially available today due to several challenges, primarily its size: the small size of the motor results in significant shear stress and rotor heat production during high RPM [48].

While human data are lacking, several studies have shown promising results in animal models. In porcine biventricular assist models, the MVAD successfully replaced cardiopulmonary bypass and fully supported the right ventricle[47]. As an RVAD, the MVAD may be implanted into the right ventricle, and the outflow cannula directed into the PA. While the MVAD demonstrates potential, further study is necessary to confirm its safety in humans. It will be at least a few years before it can be used clinically, especially in children [50].

Discussion

The correlation between RV failure and poor prognosis has become evident over recent years. While medical management is a reliable treatment option, percutaneous MCS devices are proving their reliability in stabilizing patients in cardiogenic shock. These devices serve as a bridging therapy to recovery or transplantation. As these devices become more commonplace, their role in acute and chronic heart failure management will expand. Technological advancements will also allow for a broader patient selection process. There is still much more work to be done before we can know the limits of these devices.

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