

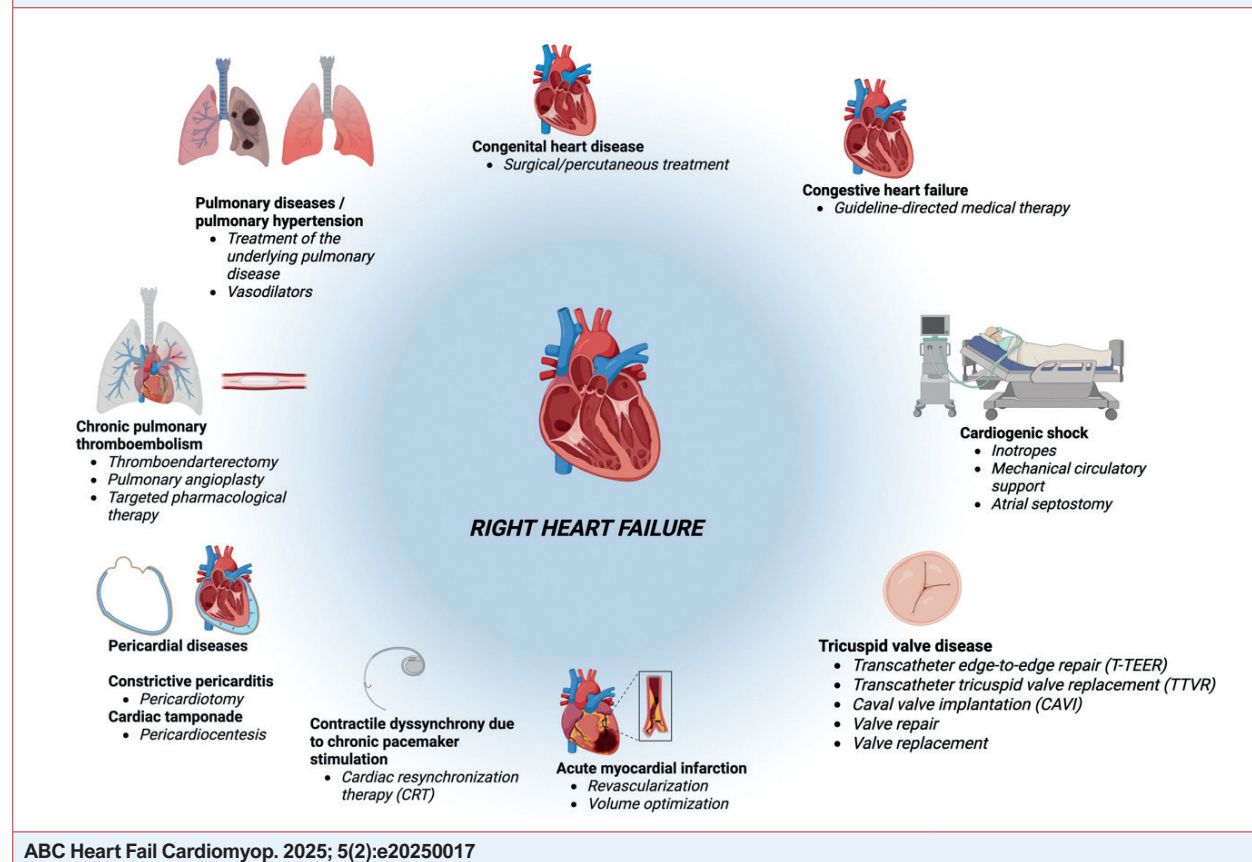
Advanced Management of Right Heart Failure: An Illustrative Case

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Central Illustration: Advanced Management of Right Heart Failure: An Illustrative Case



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Abstract

Right heart failure (RHF) is a serious condition characterized by the inability of the right ventricle (RV) to generate sufficient output for pulmonary circulation,

leading to systemic congestion and hypoperfusion. Proper management of RHF is essential to improve prognosis and quality of life, requiring individualized treatment based on hemodynamic assessment.

Keywords

Heart Failure; Pulmonary Hypertension; Heart Ventricles

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This article reviews pathophysiology, diagnosis, and therapeutic options for RHF, including strategies for preload optimization, afterload reduction, and advanced circulatory support. Additionally, it presents a clinical case of a patient with severe RHF secondary to chronic pulmonary thromboembolism and primary tricuspid regurgitation, treated with a hybrid approach involving balloon pulmonary angioplasty and pulmonary thromboendarterectomy combined with surgical valve repair, resulting in significant functional improvement. This review highlights the importance of personalized strategies and combined therapies in the management of advanced RHF.

Introduction

Right heart failure (RHF) is a clinical syndrome characterized by right ventricular (RV) dysfunction that results in inadequate pulmonary blood flow and/or increased filling pressures, either at rest or during demanding physiological conditions, such as exercise, growth, or pregnancy.¹ This condition may occur in isolation or in association with LHF, and may result from different pathophysiological mechanisms, which will be described in this article.¹

RHF is associated with a poor prognosis, especially when combined with LHF. In addition to patients with left ventricular dysfunction, patients with pulmonary arterial hypertension who present with RHF also have a worse prognosis.²

This article aims to review the pathophysiology, diagnosis, and treatment options for RHF. Furthermore, a clinical case managed by the authors of this chapter at the Hospital de Clínicas de Porto Alegre will be presented.

Clinical Case

A 48-year-old woman with a history of pulmonary hypertension due to chronic thromboembolism (CTEPH) and severe tricuspid regurgitation was transferred to the Hospital de Clínicas de Porto Alegre with severe RV dysfunction and cardiogenic shock. She presented with INTERMACS classification 3 (progressive deterioration) and required inotropic (milrinone 0.4 µg/kg/min) and vasopressor (norepinephrine 0.1 µg/kg/min) support to maintain systemic perfusion. Echocardiography revealed RV hypertrophy and dilation, significant free wall hypokinesis, increased pulmonary artery systolic pressure (PASP = 75 mmHg), and

severe tricuspid regurgitation due to a posterior leaflet flail (vena contracta = 9 mm), suggesting pulmonary hypertension secondary to chronic pulmonary thromboembolism (PTE) associated with tricuspid valve incompetence. Angiotomography (AngioCT) of the pulmonary arteries demonstrated amputation due to chronic thrombosis of the left lower lobe branch, as well as areas of stenosis and dilation in the pulmonary arterial system, in addition to significant dilation of the right heart chambers with inversion of the interventricular septum.

Right heart catheterization was performed, revealing precapillary pulmonary hypertension with elevated pulmonary vascular resistance and hemodynamic signs (invasive and noninvasive) of poor prognosis (Table 1). Furthermore, pulmonary arteriography demonstrated bilateral filling defects consistent with chronic pulmonary embolism and the absence of perfusion of the left lower lobe.

Given the severity of the condition and the initial contraindication for pulmonary endarterectomy due to clinical instability, percutaneous balloon pulmonary angioplasty (BPA) was chosen as rescue therapy. Supported by high-flow nasal cannula oxygen therapy, the patient underwent two BPA sessions targeting segmental and subsegmental branches of the right pulmonary artery in the following locations: A3 (anterior segment of the upper lobe), A4 (lateral segment of the middle lobe), A6 (superior segment of the lower lobe), A7 (basal medial segment of the lower lobe), and A8 (basal anterior segment of the lower lobe). The procedure was successful, with significant global hemodynamic improvement (Table 1), allowing progressive reduction of inotropes and complete

Table 1 – Hemodynamic data of the BPA strategy as a bridge therapy for definitive surgical treatment

Exam	Parameters evaluated	Pre-Balloon Pulmonary Angioplasty	After Balloon Pulmonary Angioplasty	After Definitive surgical treatment
Right heart catheterization	Right atrium	20 mmHg	10 mmHg	3 mmHg
	Right ventricle	90 mmHg	58 mmHg	
	Pulmonary artery	90/35/53 mmHg	58/27/37 mmHg	47/22/30 mmHg
	Pulmonary artery occlusion pressure	12 mmHg	12 mmHg	*
	Cardiac index	2.5 L/min/m ²	3.2 L/min/m ²	2.7 L/min/m ²
	Pulmonary vascular resistance	8.5 Wood	4.5 Wood	*
	Total pulmonary resistance	7,5 Wood	6.6 Wood	6.3 Wood
	Pulmonary vascular compliance	1.1 ml/mmHg	1.67 ml/mmHg	1.7 ml/mmHg
Echocardiogram	FAC	20%	23%	24%
	RV Dimensions	52 mm	53 mm	45 mm
	Left ventricular ejection fraction	75%	76%	53%

**Hemodynamic measurements obtained immediately before removal of the pulmonary artery catheter in the postoperative period of thromboendarterectomy. Under these circumstances, pulmonary artery occlusion pressure (PAOP) is not measured due to the risk of rupture of the areas manipulated during the procedure. Source: authors.*

withdrawal of norepinephrine after the second session. Endovascular therapy allowed the discontinuation of both vasoactive drugs and oxygen therapy, resulting in discharge from the intensive care unit. The six-minute walk test after two BPA sessions covered a distance of 347 m without significant desaturation. Serial echocardiograms showed partial recovery of RV function with reduced ventricular dilation.

With hemodynamic stabilization and functional improvement, the patient was reevaluated for definitive treatment. Bilateral pulmonary thromboendarterectomy was indicated and performed, combined with tricuspid valve repair at the same surgical time, without intraoperative complications. Under total circulatory arrest with deep hypothermia, thrombi were removed from lobar, segmental, and subsegmental arteries bilaterally. Even vessels previously treated with BPA were treated. During surgery, a rupture of part of the chordae tendineae of the posterior leaflet was confirmed, causing significant tricuspid valve dysfunction. The valve repair consisted of fusion of the posteroanterior commissure, resulting in a competent bicuspid valve. The patient's postoperative course was favorable. Anticoagulation was initiated approximately 6 hours after surgery. She required low-dose vasopressors for 48 hours. It was decided to restart riociguat on the third postoperative day due to the mean pulmonary artery pressure (maintained between 28 and 33 mmHg) to facilitate management of residual tricuspid regurgitation (due to annular dilation related to the large right atrial enlargement). The patient was discharged on the 13th postoperative day in New York Heart Association (NYHA) functional class I. The combination of BPA and pulmonary thromboendarterectomy, combined with tricuspid valve repair, proved to be an effective strategy for significant recovery of RV function and clinical improvement in a complex case of RHF associated with chronic PTE refractory to pharmacological management.

Etiology and pathophysiology of right heart failure

RHF results from conditions that compromise RV function through different pathophysiological mechanisms that will be described in this topic (Figure 1 - Central illustration).

Pressure overload

Pulmonary arterial hypertension (PAH) – Group 1

Pulmonary vascular disorder characterized by progressive arteriolar remodeling, with chronic elevation of pulmonary vascular resistance, in the absence of secondary causes.

Pulmonary hypertension associated with left heart disease – Group 2

It results from a chronic increase in pulmonary venous pressure, secondary to left ventricular dysfunction (with reduced or preserved ejection fraction), valve dysfunction (mitral or aortic), and other conditions related to left heart disease that lead to venocapillary hypertension.

Pulmonary hypertension associated with lung disease and/or hypoxia – Group 3

Includes chronic obstructive pulmonary disease (COPD), pulmonary fibrosis, and combined lung disease, in which chronic hypoxic vasoconstriction, parenchymal destruction, and remodeling of the pulmonary vascular bed cause increased resistance to blood flow. Hypoxemia without primary lung disease, such as hypoventilation syndrome, high-altitude PH, and developmental lung diseases, is also included in this group.

Pulmonary hypertension associated with pulmonary artery obstructions – Group 4

Pulmonary hypertension due to chronic PTE is characterized by persistent obstruction of the pulmonary arteries by organized thrombi, with secondary remodeling of the remaining vascular bed. Sarcomas and vasculitis are rarer causes.

Pulmonary hypertension of uncertain and/or multifactorial mechanism – Group 5

It involves systemic diseases, hematologic and metabolic disorders, where multiple factors – including chronic inflammation and endothelial dysfunction – contribute to elevated pulmonary artery pressure.

Congenital obstructive heart disease of the RV outflow tract

Such as pulmonary stenosis, tetralogy of Fallot, and pulmonary atresia.

Conditions that raise intrathoracic pressure

Mechanical ventilation with excessive positive pressure, tension pneumothorax, and massive pleural effusions.

Systemic acidosis

The presence of acidosis from any cause induces pulmonary arterial vasoconstriction, increasing pulmonary vascular resistance and RV afterload.³

Volume overload

Congenital heart disease with left-to-right shunt

Patent ductus arteriosus (PDA), atrial septal defect (ASD), and ventricular septal defect (VSD) promote chronic pulmonary hyperflow, causing RV dilation and, eventually, secondary pulmonary hypertension (e.g., Eisenmenger syndrome).

Right valve regurgitation

Primary or secondary tricuspid regurgitation, as well as pulmonary regurgitation after surgical repairs, increase RV end-diastolic volume, increasing preload and contributing, in the long term, to ventricular dilation.

Chronic atrial fibrillation

Chronic atrial fibrillation can lead to dilation of the right atrium and tricuspid annulus, promoting functional tricuspid insufficiency and, consequently, RV volumetric overload.

Systemic hypervolemia

Hypervolemia from extracardiac causes (polytransfusion, excessive use of serum therapy, dialysis-dependent renal failure, and cirrhosis) can also lead to RV volume overload.⁴

Contractile dysfunction

Coronary artery disease

Acute occlusion of the proximal right coronary artery can result in acute RV systolic dysfunction, leading to hemodynamic instability. Patients with chronic RV ischemic disease may also develop RHF.⁵

Myocarditis and infiltrative diseases

Inflammatory processes, such as viral myocarditis, as well as diseases such as sarcoidosis and amyloidosis, promote fibrosis and myocardial infiltration, affecting RV systolic and diastolic function.⁶

Arrhythmogenic right ventricular dysplasia (ARVD)

Genetic disease characterized by fibroadipose replacement of the RV myocardium, resulting in progressive dysfunction, ventricular arrhythmias, and a high risk of sudden death.

Use of left ventricular assist device (LVAD)

LVAD implantation may exacerbate RV dysfunction by increasing venous return and redistributing preload, imposing an additional challenge on an already compromised RV.

Post-heart transplant

RV dysfunction in the post-transplant period may occur due to suboptimal myocardial preservation, preexisting pulmonary hypertension, or graft-recipient incompatibility.

Ischemic or mechanical injury after cardiectomy

During cardiac surgeries, the RV may suffer direct injury or perioperative ischemia, especially in the context of prolonged extracorporeal circulation, resulting in transient or persistent contractile impairment.

Pericardial diseases

Constrictive pericarditis

Pericardial thickening and calcification (the most common causes of which are previous viral pericarditis, radiotherapy, cardiac surgery, tuberculosis, and collagen diseases) restrict RV expansion, resulting in early ventricular filling and diastolic collapse, with elevated right atrial pressure.

Cardiac tamponade

Acute accumulation of pericardial fluid, under high pressure, leads to diastolic collapse of the right chambers, impairing venous return and causing obstructive shock. The most common etiologies are trauma, iatrogenic complications, malignant neoplasms, infections, uremia, infarction, and collagen diseases.⁷

Other mechanisms

Chronic pacemaker stimulation

Chronic RV pacing, especially through apical pacemaker devices, can induce non-physiological electrical activation patterns, contributing to contractile dyssynchrony and adverse RV remodeling. In addition to the deleterious effects of chronic RV pacing, the presence of pacemaker device leads can mechanically interfere with tricuspid valve function, resulting in its incompetence.⁸

Ventricular interdependence

Due to shared anatomy and a common pericardium, changes in volume or pressure in one ventricle directly affect the other. In RHF, RV dilation can shift the interventricular septum to the left, compromising LV diastolic filling and systolic function, exacerbating global heart failure.

Imbalance between oxygen supply and demand

RV hypertrophy increases the metabolic demand of the myocardium. Simultaneously, elevated end-diastolic pressures can compromise RV coronary perfusion, especially during episodes of hypotension or hypoxemia, leading to myocardial ischemia and contractile dysfunction.⁷

Obesity and insulin resistance

Obesity and insulin resistance are potential modifiable risk factors with a prognostic impact on RHF that deserve to be considered. In patients with dilated cardiomyopathy or pulmonary hypertension, diabetes is associated with worsening RV systolic and diastolic function. The mechanisms involved include fibrosis, inflammation, microvascular ischemia, and myocardial lipid accumulation.^{9,10}

Obesity negatively impacts RV function by increasing preload and afterload, and compromising contractility. Obese individuals with heart failure and preserved ejection fraction have greater RV dilation, elevated pulmonary pressures, and poorer sarcomere performance, as well as greater ventricular interdependence and pericardial restriction, compared to non-obese individuals. Furthermore, sleep-disordered breathing, common in obesity, also contributes to RV dysfunction by increasing pulmonary and systemic pressures.¹¹⁻¹³

Clinical manifestations

Symptoms

The main symptoms of RHF reflect systemic venous congestion, reduced cardiac output, and tissue hypoperfusion:

- Dyspnea and exercise intolerance.
- Orthopnea and paroxysmal nocturnal dyspnea (less frequent).
- Peripheral edema.
- Ascites and abdominal distension.
- Pain or discomfort in the right upper quadrant.
- Nausea and vomiting (congestion of the gastrointestinal tract and edema of the intestinal wall).
- Weight loss and cardiac cachexia (advanced stages).

Physical examination

Signs of Systemic Venous Congestion

- Pathological jugular distension.
- Hepatojugular reflux.
- Kussmaul's sign (absence of physiological decrease or, in some cases, paradoxical increase in jugular venous pressure during inspiration).
- Hepatomegaly and pain on liver palpation.
- Ascites.
- Peripheral edema.

Cardiopulmonary Findings

- B1 normal or hypophonic; B2 with hyperphonic.
- Systolic or holosystolic murmur at the right lower sternal border (functional tricuspid regurgitation). This murmur intensifies during deep inspiration, a finding known as the Rivero-Carvalho maneuver.
- Fixed splitting of B2: if associated atrial septal defect.
- Pulsus paradoxus: cardiac tamponade and, occasionally, in advanced RHF.
- Jugular venous pulse with abnormal waves:
 - ◇ Giant A wave: Suggests RV diastolic dysfunction (e.g., tricuspid stenosis or significant hypertrophy).
 - ◇ Prominent V wave: Indicative of significant tricuspid regurgitation.
- Diastolic murmur of pulmonary regurgitation: early diastolic murmur, usually decrescendo, indicative of severe pulmonary hypertension.
- Third RV sound: rapid ventricular filling - RV systolic dysfunction.
- Fourth RV sound: forced atrial filling of a non-compliant right ventricle.
- Left parasternal impulse: Sign of hypertrophy or dilation of the right ventricle.
- Pleural effusion.

Advanced Systemic Manifestations

- Arterial hypotension.
- Reduced peripheral perfusion.
- Central or peripheral cyanosis.^{7,14,15}

Complementary exams

Several complementary tests can be used in RHF; the main ones are summarized in Table 2.

Coupling of the right ventricle with the pulmonary artery

Coupling between RV contractility and pulmonary circulation-imposed afterload (RV-PA coupling) is a key hemodynamic concept for assessing RV adaptation to pulmonary hypertension. This coupling is represented by the ratio of RV systolic elastance (Ees), a load-independent contractility parameter, to effective arterial elastance (Ea), which reflects afterload. Under normal conditions, this ratio (Ees: Ea) ranges from 1.5 to 2.0, indicating efficient coupling, in which RV-generated energy is optimally transferred to the pulmonary circulation.²⁸

In pulmonary hypertension, a compensatory increase in Ees initially occurs to counteract the increased afterload (Ea) and maintain coupling. However, as RV contractile capacity is exhausted, Ea continues to increase disproportionately to Ees, leading to progressive RV decoupling and hemodynamic compromise. Ees: Ea values below 0.6–0.8 indicate severe decoupling and are associated with a worse prognosis.²⁹

Direct measurement of RV-PA coupling requires right-sided catheterization with pressure-volume curve analysis, a complex method that is difficult to apply in daily clinical practice. Alternatively, there are echocardiographic parameters that estimate this value less accurately.^{14,29}

Treatment

The management of RHF should be based on an individualized approach, taking into account the blood volume status, underlying etiology, and chronicity of the condition. General measures are recommended, including nutritional support, physical activity, including cardiopulmonary rehabilitation, immunizations, contraception, travel and procedural guidance, and psychological counseling. Therefore, a multidisciplinary approach is essential for successful care.²⁵

The treatment of RHF varies depending on the scenario involved (e.g., whether it is acute, chronic, or chronic-exacerbated). Therefore, it is important to establish therapeutic goals to guide necessary monitoring and management in clinical practice.^{30,31}

Preload optimization

Blood volume adjustment

- In patients with chronic RHF and significant fluid retention, the use of loop diuretics is indicated. Thiazide diuretics may be associated in refractory cases.
- In cases of refractory congestion, ultrafiltration may be considered to remove excess volume while avoiding side effects related to high doses of diuretics.³² It is worth remembering that pharmacological treatment does not just include fluid withdrawal to improve RV performance. In acute situations (such as acute myocardial infarction with RV involvement), it may be

Table 2 – Complementary exams used in ICD

Exam	Subitem	Main Considerations
Laboratory	BNP/NT-proBNP	Assess myocardial stress and prognosis. Correlates with RHF severity.
	Liver function	Assess chronic liver congestion. Elevated transaminases, GGT, and AF.
	Kidney function	Monitor renal perfusion. Elevated creatinine and urea nitrogen; ↓ GFR.
	Blood count	Assess hematologic changes.
Electrocardiogram	Electrocardiogram	Right axis deviation, right atrial dilation, RV hypertrophy (e.g., R waves in V1), right bundle branch block, atrial arrhythmias, S1Q3T3 pattern suggesting PTE.
Echocardiogram	TAPSE	<17 mm indicates RV dysfunction.
	S' wave (tissue Doppler)	≤9,5 cm/s indicates RV dysfunction.
	FAC	<35% indicate RV dysfunction.
	Strain RV	Early changes in contractility (especially in the free wall).
	Morphology and dimensions	Allows the identification of remodeling, such as dilation or hypertrophy (chronic overload).
	Valvular heart disease	Includes analysis of the tricuspid and pulmonary.
	Ventricular interdependence	Displacement of the interventricular septum to the left.
Transesophageal Echocardiogram	Transesophageal Echocardiogram	It may be indicated to evaluate shunts, valvular anomalies, and posterior structures.
Bedside Ultrasound	VExUS	Assessment of systemic congestion. Includes hepatic, portal, and renal veins.
	VCI	Indirect estimation of CVP.
	McConnell's Sign	Sign suggestive of acute PTE.
	RV/LV ratio, thrombi, and cardiac tamponade	Important in immediate management.
	Vascular ultrasound	Venous thrombosis suggests a thromboembolic etiology for RHF.
	Pleuropulmonary ultrasound	Signs of congestion (presence of B lines, pleural effusion); Pneumothorax; Pulmonary infarction.
Magnetic Resonance Imaging	RV volumes and ejection fraction, and shunts	Gold standard for volumes and RVEF. Stroke and end-systolic volume index. Identification of intracardiac shunts.
	Tissue characterization	Late gadolinium enhancement.
	Strain and pericardium assessment	Useful in ARVC, pericarditis, and myocarditis.

Review Article

Right heart catheterization	PH Stratification	Identification of pre- and post-capillary components.
	Advanced parameters	– Pulmonary vascular compliance – Pulmonary artery pulsatility index (PAPI) – RV functional reserve; – RV work index; – Relationship between right atrial pressure and PAOP; – Cardiac power (CPO)
	Therapeutic planning and mechanical support	Essential for support escalation.
	Complementary steps, such as provocative testing (water and/or exercise) and vasoreactivity.	They can guide therapeutic strategy (pharmacological treatment or decision about heart transplant).
	Pulmonary arteriography	Carried out with the Right heart catheterization for BPA planning.
Chest CT Angiography	Chest CT Angiography	Visualizes thrombi, airways, and structural changes. Helps in the surgical or percutaneous planning of CTEPH.
Ventilation-Perfusion Scintigraphy	Ventilation-Perfusion Scintigraphy	Fundamental in CTEPH. Allows differentiation between vascular and parenchymal changes.
Pulmonary function tests	Spirometry Lung volumes Pulmonary diffusion Submaximal and maximal exercise testing	Functional assessment in patients with PH (as a cause or consequence of PH). Evaluation of baseline and follow-up exercise capacity.
PET CT	PET CT	Helps identify cardiac sarcoidosis, neoplasms, and vasculitis.
Abdominal ultrasound	Doppler examination	Evaluation of chronic liver disease and schistosomiasis.
Polysomnography	Polysomnography	Investigate sleep-disordered breathing.

BNP: B-type natriuretic peptide; NT-proBNP: N-terminal pro-B-type natriuretic peptide; RHF: right heart failure; GGT: Gamma-glutamyl transferase; AF: Alkaline phosphatase; GFR: Glomerular Filtration Rate; RV: Right Ventricle; PTE: Pulmonary Thromboembolism; TAPSE: Tricuspid Annular Plane Systolic Excursion; FAC: Fractional Area Change; VExUS: Venous Excess Ultrasound Score; CVP: Central Venous Pressure; IVC: Inferior Vena Cava; ARVC: Arrhythmogenic Right Ventricular Cardiomyopathy; PH: Pulmonary Hypertension; BPA: balloon pulmonary angioplasty; CTEPH: Chronic Thromboembolic Pulmonary Hypertension; PET-CT: Positron Emission Tomography and Computed Tomography. Sources:¹⁶⁻²⁷

necessary to administer fluids cautiously to increase preload, correct hypotension, and preserve systemic perfusion. On the other hand, in acute PTE, volumes greater than 500 ml tend to worsen instability.⁵

Atriotomy with Septostomy

Atrioseptoplasty, creating a controlled interatrial shunt, may be a therapeutic option in advanced cases of RHF with pulmonary hypertension. This intervention aims to reduce right

atrial pressure, improve systemic cardiac output, and alleviate symptoms of congestion, acting as a bridge or palliative therapy in selected patients.³³ However, this therapy should not be performed in acute situations and/or in patients with very high right atrial pressure (RAP) (> 20 mmHg) as cardiac output is partly dependent on filling volume (preload), as well as in already hypoxemic patients (systemic saturation < 90%) as the procedure can induce hypoxemia due to the right-to-left shunt.

Tricuspid regurgitation treatment

Tricuspid regurgitation, often associated with RHF, can have different approaches:

- **Conventional Surgery:** In cases of significant tricuspid regurgitation associated with annular dilation or organic lesions, valve repair or valve replacement can restore valve competence and improve RV function.
- **Contemporary Treatments:**
 - ◇ **Triclip:** A percutaneous device that uses a clipping technique to approximate the tricuspid valve leaflets. The Triluminate study demonstrated that transcatheter tricuspid valve repair is safe and effective, resulting in a significant reduction in tricuspid regurgitation and improved patient quality of life.³⁴
 - ◇ **Tricvalve:** A bicaval percutaneous valve system developed to treat severe tricuspid regurgitation. Its implantation aims to correct valve incompetence and reduce RV volume overload. The single-arm, prospective, non-randomized TRICUS and TRICUS Euro studies demonstrated potential improvements in quality of life and device safety.³⁵ It is worth noting that randomized studies are needed to confirm these hypotheses.
 - ◇ **Percutaneous valve implantation:** The largest clinical trial of valve implantation in the tricuspid position is the TRISCEND study, which evaluated the EVOQUE transfemoral valve replacement system. This randomized, controlled, multicenter clinical trial demonstrated that transcatheter implantation of the EVOQUE valve is safe and effective for treating severe tricuspid regurgitation, potentially providing an improvement in patients' quality of life (hierarchical composite outcome assessed).³⁶

Afterload reduction

Percutaneous and surgical treatment of chronic PTE

In patients with chronic PTE, pulmonary thromboendarterectomy is the gold standard, promoting pulmonary artery unblocking and immediate reduction of RV afterload. Surgical treatment can reduce mortality, normalize hemodynamics, and improve functional capacity.³⁷ When thromboendarterectomy is not feasible due to high surgical risk or anatomical compromise of segmental or subsegmental branches alone, BPA emerges as an effective percutaneous alternative to improve pulmonary hemodynamics and functional capacity.^{38,39}

Pulmonary vasodilators

The use of specific vasodilators, such as prostanoids, phosphodiesterase-5 inhibitors (e.g., sildenafil), endothelin receptor antagonists, and guanylate cyclase stimulators, aims to reduce pulmonary vascular resistance, thereby decreasing right ventricular afterload and improving cardiac

output. Sotatercept, an activin receptor ligand trap, has a different mechanism of action from other vasodilators, as it acts by reversing vascular remodeling. The choice of agent should be based on the underlying etiology and individual response, and is often indicated in cases of pulmonary arterial hypertension (group 1).^{3,25}

In the context of chronic PTE, particularly in patients not eligible for pulmonary endarterectomy or with persistent hypertension after surgery, riociguat is currently the only drug approved with a specific indication. This drug promotes vasodilation and reverse remodeling of the pulmonary vasculature. Robust evidence of efficacy was demonstrated in the study CHEST-1,⁴⁰ and more recently reinforced in the RACE trial,⁴¹ which compared riociguat to BPA in patients with inoperable chronic PTE. The study showed significant improvement in pulmonary hemodynamics and functional capacity in the riociguat-treated group. Furthermore, one of the main findings of the RACE study was a significant reduction in periprocedural complications in patients treated with riociguat before BPA.

The 2022 ESC European Guideline on Pulmonary Hypertension strongly recommends the use of riociguat as first-line therapy in patients with chronic PTE who are not candidates for surgery or who have residual hypertension after endarterectomy. Riociguat use should be monitored by a specialized team, with regular functional and hemodynamic monitoring.³

Treatment of underlying lung disease

Group 3 pulmonary hypertension is commonly associated with three conditions: COPD/emphysema, pulmonary fibrosis, and hypoventilation syndromes. Therapy should be targeted to each of these underlying lung diseases. It is worth remembering that in cases of hypoxemia, oxygen support is indicated, as is ventilatory support (for conditions related to hypoventilation).³

Treatment of left heart failure

RHF secondary to left heart disease should be treated according to the underlying disease. LHF (with reduced or preserved EF) and valvular heart disease (aortic stenosis/insufficiency and mitral stenosis/insufficiency) should be treated according to current guidelines to alleviate RV afterload. Right-sided catheterization is indicated to understand RV function better and to define the pre- and post-capillary components of PH.³

Specific treatments

Surgical or percutaneous repair of congenital heart diseases

Treatment of RHF related to congenital heart disease often requires interventions to correct structural alterations that impact RV preload and afterload. Correction of these abnormalities can be performed through percutaneous or surgical approaches, depending on the anatomy and degree of hemodynamic impact.

However, it is crucial to recognize that patients with severe pulmonary hypertension associated with intracardiac shunts are at high risk of complications, especially Eisenmenger syndrome. In these cases, defect correction can precipitate acute RV failure and hemodynamic collapse. According to the European guideline for Congenital Heart Disease in adults, pulmonary vascular resistance ≥ 5 Wood's units with optimized clinical treatment or physiology compatible with Eisenmenger syndrome (with desaturation during exercise) contraindicate defect correction. In cases with borderline values, therapeutic decisions should always consider the individual risk-benefit analysis and the possibility of prior treatment with pulmonary vasodilators before definitive intervention.^{42,43}

Pericardial diseases

Cardiac tamponade is a medical emergency that requires immediate intervention to relieve cardiac compression caused by fluid accumulation in the pericardial space. The treatment of choice is pericardiocentesis. Constrictive pericarditis, on the other hand, is a chronic condition characterized by thickening and stiffness of the pericardium. The definitive treatment is pericardiectomy.⁷

Acute myocardial infarction

RV infarction with acute RHF requires a specific therapeutic approach: early revascularization of the culprit coronary artery, preferably by primary angioplasty, is essential to limit the extent of the infarction and preserve RV function.

Volume replacement should be carefully adjusted to maintain adequate preload and optimize cardiac output, avoiding both hypovolemia and systemic congestion. The use of nitrates and other vasodilators should be avoided, as they can reduce preload and worsen hypotension. In patients with right-sided cardiogenic shock, vasopressor support may be necessary to maintain systemic perfusion, while inotropic agents can be used to improve RV contractility.

Conduction disturbances are common in RV infarction, especially atrioventricular blocks and sinoatrial node dysfunction, and may justify the need for a temporary pacemaker in cases of symptomatic bradyarrhythmias or significant hemodynamic impairment.⁵

Treatment of pacemaker-related right heart failure

Treatment of RHF related to chronic RV pacing by a pacemaker involves strategies to mitigate the adverse effects of this therapy. Biventricular pacing (Bi-V) may be considered as a treatment for this condition.⁴⁴

Other alternatives are the search for more physiological stimulation in the His bundle or in the left branch area, which has shown superior results to isolated RV stimulation.⁸

The presence of transvalvular device leads can compromise tricuspid valve leaflet coaptation, leading to significant tricuspid regurgitation. Specific interventions to treat lead-induced tricuspid regurgitation, such as lead replacement or

percutaneous procedures like the Triclip, may be considered to correct valve incompetence.⁴⁵

Cardiogenic shock: Use of inotropes

Cardiogenic shock secondary to RHF is characterized by the right ventricle's inability to generate sufficient output for the pulmonary circulation, resulting in severe systemic hypoperfusion. Immediate management aims to restore ventricular contractility and improve pulmonary blood flow through the use of inotropic agents. Among the most commonly used drugs, dobutamine is frequently indicated for its selective positive inotropic effect, increasing RV contractility without causing significant vasoconstriction. In cases of associated hypotension, norepinephrine may be added to maintain systemic blood pressure (essential for maintaining coronary perfusion) without compromising cardiac output. Vasopressin may also be added.

Milrinone, a phosphodiesterase III inhibitor, is also considered in cases of associated pulmonary hypertension due to its selective inotropic and vasodilatory properties for the pulmonary circulation. However, its use requires caution in patients with significant hypotension (due to its effect on systemic vascular resistance). Another inotropic agent to consider is levosimendan, a calcium sensitizer and potassium channel facilitator that has shown hemodynamic improvement in RHF. On the other hand, digitalis has no role as an inotropic agent in RHF and may even be harmful. The choice of inotropic agent should be based on a precise hemodynamic assessment, preferably guided by parameters obtained by right-sided catheterization, aiming to balance improvement in ventricular contractility with maintenance of systemic perfusion.⁴⁶⁻⁴⁹

Mechanical circulatory support

In cases of cardiogenic shock refractory to pharmacological treatment, mechanical circulatory support (Table 3) emerges as a crucial strategy for hemodynamic stabilization and as a bridge to recovery, heart transplantation, or definitive therapeutic decision.

Bilateral heart, cardiopulmonary, and lung transplantation

Transplantation represents the definitive therapy for patients with advanced RHF refractory to other strategies. The choice of transplant type depends fundamentally on the underlying etiology: patients with severe PAH without significant left ventricular impairment are preferred candidates for bilateral lung transplantation; individuals with complex congenital heart disease, particularly those with complex intracardiac anatomy or univentricular circulation, may require heart-lung transplantation. In cases of refractory biventricular failure, isolated heart transplantation may be considered. Careful selection of candidates, including assessment of comorbidities, functional status, surgical risk, and absolute or relative contraindications, should be performed by a multidisciplinary team specialized in heart failure and transplantation, in accordance with updated international guidelines.⁵¹

Table 3 – Mechanical circulatory support devices

Device	Indication/Use	Observations
Veno-Arterial ECMO (VA-ECMO)	Biventricular support; bridge to decision in critically ill patients.	Cannulas can be inserted into the RA and PA for immediate relief of afterload.
RV Assist Devices (Tandem-RVAD, Protek Duo)	Specific RV support; bridge to transplant or recovery.	Implantable or percutaneous; do not directly affect the LV.
Intra-Aortic Balloon Pump (IABP)	Selected cases; reduces LV afterload and improves RV-LV coupling.	Controversial for RV; may be useful after ECMO for afterload relief.
Percutaneous device (Impella RP)	Refractory shock; continuous flow to the PA and relief of RV overload.	Percutaneous insertion; temporary use.

RA: right atrium; PA: pulmonary artery; ECMO: extracorporeal membrane oxygenation; RV: right ventricle; LV: left ventricle. Source:^{50,51}

Palliative care

Palliative care represents a multidisciplinary approach aimed at alleviating suffering in patients with chronic diseases such as RHF. It should be initiated early and maintained throughout the disease's progression. It involves symptomatic control, communication about prognosis, and care planning, including treatment limitations. Although all professionals should be involved in this management, specialized teams contribute to better clinical decisions and quality of life, and current guidelines recommend their structured integration.^{52,53}

Conclusion

Right heart failure is a complex condition with a significant impact on patient prognosis, especially when associated with pulmonary hypertension and structural RV disease. Effective management requires an individualized approach, based on a detailed hemodynamic assessment and identification of the underlying etiology. Strategies such as preload optimization, afterload reduction, and advanced circulatory support are essential to stabilize patients and improve RV function. It is important to recognize each condition, allowing for targeted interventions that can bring significant benefits to patients.

The clinical case presented reinforces the importance of a multidisciplinary approach and escalated treatment in patients with severe RHF. It is worth noting that there are few reports of rescue BPA for the treatment of cardiogenic shock, and this is the first case in the literature in which percutaneous treatment was used as a bridge to surgical treatment.⁵⁴ Furthermore, the percutaneous procedure performed in the case cited in this article had some quite notable characteristics. In addition to periprocedural hemodynamic and clinical instability, the decision was made to perform dilations in more proximal vessels than usually recommended. This approach was part of a shared judgment decision by the multidisciplinary team,

which was aware of the associated risks, but it culminated in significant clinical and hemodynamic improvement, allowing surgical treatment. The combination of percutaneous and surgical therapies enabled the recovery of right ventricular function and the patient's clinical rehabilitation, highlighting the need for personalized strategies for each hemodynamic profile. With advances in diagnostic and therapeutic techniques, continued improvement in the management of RHF is expected, reducing morbidity and improving clinical outcomes for these patients.

Author Contributions

Conception and design of the research and Critical revision of the manuscript for content: Gazzana MB, Wainstein RV; Writing of the manuscript: Crivelaro PCF, Machado GP, Lorenzi W, Benedetto IG, Valle FH.

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This study is not associated with any thesis or dissertation work.

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