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Review

Importance of right heart catheterization in advanced heart failure management

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Abstract

Patients with chronic congestive heart failure belong to a population with reduced quality of life, poor functional class, and increased risk of mortality and morbidity. In these patients, assessment of invasive hemodynamics both serves therapeutic purposes and is useful for stratification roles. The right heart catheterization has become a cornerstone diagnostic tool for patients in refractory heart failure or cardiogenic shock, as well as for the assessment of candidacy for heart replacement therapies, and the management of patients following mechanical circulatory assist device implantation and heart transplantation.

Keywords: Pulmonary artery catheter; Swan-Ganz catheter; Right heart catheterization; Invasive hemodynamics; Cardiogenic shock; Heart failure; Acute decompensated heart failure

1. Introduction

The history of right heart catheterization (RHC) began in 1929, when Dr. Werner Forssmann, as a surgical trainee in Germany, advanced a 4 French urinary tract catheter from the antecubital fossa to the right atrium and confirmed the position by fluoroscopy [1]. Decades later, Drs. Cournand and Richards won the Nobel Prize in medicine for describing the concept of invasive hemodynamics using RHC [2]. In 1970, a case series of 100 consecutive pulmonary catheterizations with the flow-directed catheter was published [3], validating modern-day RHC developed by Drs. Ganz and Swan [3] (Fig. 1). Since then, RHC has been utilized in many clinical scenarios and endorsed by many societal guidelines (Tables 1,2). It is the objective of this review to summarize the current understanding and indications of RHC.

2. The role of right heart catheterization and invasive hemodynamic monitoring in patients with acute decompensated heart failure

The utility of invasive hemodynamic evaluation via RHC in patients with acute decompensated heart failure is still under debate. Before the 1970s, an invasive hemodynamic assessment was essential in the diagnosis of structural heart disease [4]. In the late 1970s, however, significant advancements were made in echocardiography. Specifically, Doppler echocardiography permitted the estimation of pressure gradients across stenotic valves and of intracardiac chamber pressures with a good correlation between invasive measurements and Doppler estimations in most circumstances [5,6]. The use of full Doppler echocardiographic examination has been proposed as a complete, viable replacement to RHC in stable patients with heart failure. Temporelli *et al.* [7] found an excellent correlation between Doppler echocardiographic results and direct hemodynamic values obtained from RHC. However, RHC remains a crucial tool in the hemodynamic evaluation due to known limitations of echocardiography. Right heart filling pressures are hampered in patients with mechanical ventilation, indwelling cardiac devices can generate artifacts, and patient habitus or dressings after cardiac surgery may make correct alignment of Doppler waveforms impossible and hemodynamic calculations inaccurate. Complex hemodynamic scenarios with multiple high-velocity jets can also render isolating the correct waveform challenging. Furthermore, continuous echocardiography over hours and days to provide real-time hemodynamic data in response to interventions is not feasible in the same manner that continuous hemodynamic monitoring with RHC is feasible.

Despite the advances in technology, physical examination remains crucial in the management of heart failure patients. Thus, it is important to recognize the strengths and limitations of the clinical examination in these patients. In general, the ability to estimate congestion by physical examination is superior to the recognition of an inadequate perfusion status [8]. The physical examination is accurate in the assessment of elevated cardiac filling pressures in only 60–70% of cases, mostly due to elevation in jugular ve-

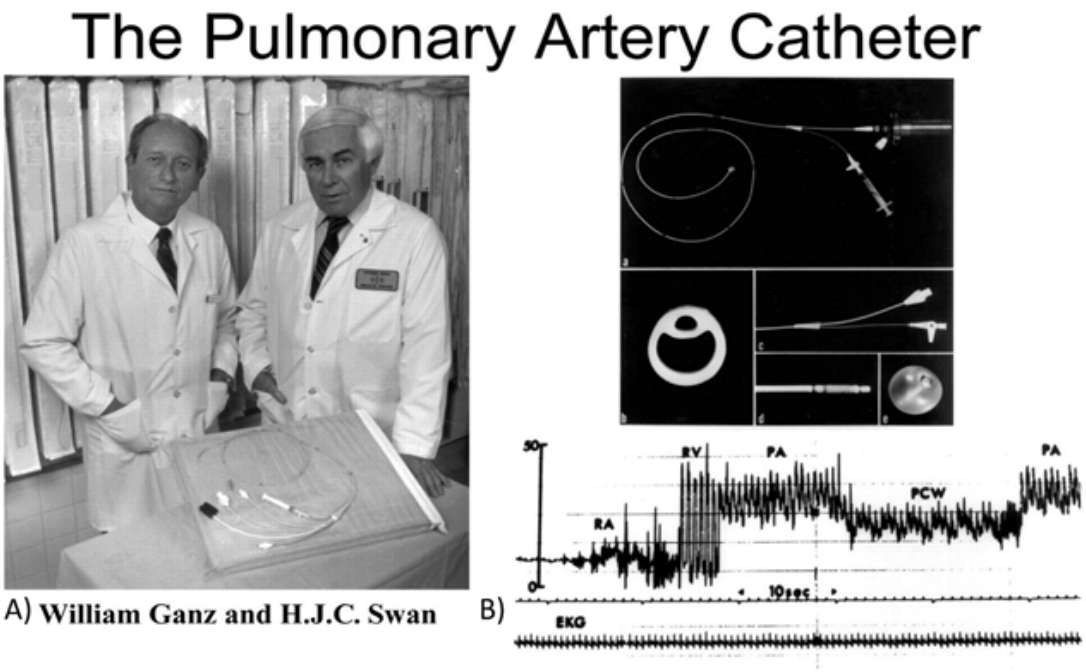

Fig. 1. The development of the pulmonary artery catheter. (A) Drs. Swan and Ganz. (B) Swan-Ganz catheter and waveforms generated after floated from the right atrium to the pulmonary arteries. (Figure courtesy of Dr. Peter Ganz, San Francisco General Hospital, University of California, San Francisco).

nous pressure [9]. Drazner *et al*. [10] demonstrated a concordant relationship between elevated right atrial pressure and pulmonary capillary wedge pressure in approximately 80% of patients with decompensated heart failure. Thus, the role of RHC in the clinical scenarios of a patient with elevated right-side pressures that are not correlated with elevated left-sided filling pressures (e.g., in patients with advanced lung disease and pulmonary hypertension), remains invaluable.

The widespread use of RHC has been called into question by some clinicians, primarily because of the Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness (ESCAPE) trial [11]. That randomized controlled study included nearly 400 patients with heart failure comparing RHC to clinical assessment alone. The study excluded patients for whom RHC may have been warranted by significant renal dysfunction (serum creatinine >3.5 mg/dL), prior use of moderate dose inotropes, or pulmonary hypertension. In addition, the ESCAPE trial did not study patients with cardiogenic shock in whom inotropic and vasopressor therapy is frequently used. The study concluded that the use of RHC did not demonstrate a mortality benefit or shorter hospitalization in stable, hospitalized patients. Therefore, the use of RHC should be restricted to complex and severe cases.

The American College of Cardiology Foundation (ACCF)/American Heart Association (AHA) [12] and European Society of Cardiology (ESC) [13] guidelines for the management of heart failure outline the clinical conditions in which the use of RHC should be considered (Table 1). The guidelines emphasize that RHC is not routinely recommended for all patients with decompensated heart failure. Rather, invasive hemodynamic monitoring should be considered in patients with decompensated heart failure refractory to the initial therapy, for patients with unclear volume status and ongoing hypoperfusion, or patients being considered for advanced therapies.

3. The role for invasive hemodynamic monitoring in patients with cardiogenic shock

Cardiogenic shock (CS) is the most severe form of heart failure with significant mortality and morbidity [14]. CS is a consequence of myocardial muscle dysfunction either due to ischemic or non-ischemic culprit event, manifested by detrimental organ perfusion and could progress into multiorgan failure.

CS, defined by clinical presentation and invasive hemodynamic criteria, is characterized by a state of tissue hypoperfusion secondary to inadequate cardiac output (CO) in the absence of hypovolemia [15] (Table 3). Within this context, the utility of RHC in the diagnosis, staging, phenotyping, and monitoring of patients in CS is invaluable, especially in the presence of complex hemodynamic interplays such as valvular disease and/or pulmonary hypertension. In 2019, the Society for Cardiovascular Angiography and Interventions released a consensus statement on the classification of CS, which stratifies patients in five stages (A through E in ascending levels of severity) (Table 4, Ref. [15]). While physical exam findings and labo-

Table 1. Right heart catheterization recommendations. 2013 ACCF/AHA and 2021 ESC HF management guidelines.

	Recommendations	COR	LOE
2013 ACCF/AHA HF Management Guideline	Invasive hemodynamic monitoring is recommended in patients with respiratory distress or impaired systemic perfusion when clinical assessment is inadequate	I	C
	Invasive hemodynamic should be considered for carefully selected patients with acute HF with persistent symptoms and/or when hemodynamics are uncertain	IIa	C
	Routine use of invasive hemodynamic monitoring is not recommended in normotensive patients with acute HF	III	B
2021 ESC HF Management Guideline	Right heart catheterization is recommended in patients with severe HF being evaluated for heart transplantation or MCS	I	C
	Right heart catheterization should be considered in patients where HF is thought to be due to constrictive pericarditis, restrictive cardiomyopathy, congenital heart disease, and high output states.	IIa	C
	Right heart catheterization should be considered in patients with probable pulmonary hypertension, assessed by echo in order to confirm the diagnosis and assess its reversibility before the correction of valve/structural heart disease.	IIa	C
	Right heart catheterization may be considered in selected patients with HFpEF to confirm the diagnosis.	IIb	C

COR, Class of Recommendation; LOE, level of Evidence; HF, Heart Failure; MCS, Mechanical Circulatory Support; HFpEF, Heart Failure with Preserved Ejection Fraction; ACC, American College of Cardiology; AHA, American Heart Association; ESC, European Society of Cardiology.

Table 2. Overview of applications of right heart catheterization in advanced heart failure patients.

Clinical scenario	Important applications	Key/Unique measurements and calculations
Acute decompensated heart failure	Evaluation of volume status and output state, especially when clinical exam/echo is equivocal	CVP, PCWP, CO, CI
Cardiogenic shock	Diagnosis, risk stratification, real-time titration of pharmacological vasoactive medications, decision support for implantation/titration/weaning of MCS	CVP, PCWP, CO, CI
Evaluation for OHT/VAD	INTERMACS profiling, standard hemodynamic measurements, and titrations as above	CVP, PCWP, CO, CI
Evaluation after OHT/VAD	Titration of vasoactive medications, necessary for biopsy, evaluate for RH failure, predict mortality	Endomyocardial biopsy (post-transplantation)
Evaluation of concomitant pulmonary hypertension	The gold standard for PH diagnosis, important to assess response to therapy, delineate into pre-/post-capillary (or mixed) profile	mPA, PVR, PCWP, response to vasodilator challenge
Evaluation of right heart failure	Post-surgery (including OHT or VAD), in the setting of pulmonary hypertension or significant valvular disease, identifying isolated RHF	PAPI, CVP, PVR, RVSWI

CI, cardiac index; CO, cardiac output; CVP, central venous pressure; OHT, orthotopic heart transplantation; mPA, mean pulmonary pressure; PAPI, pulmonary artery pulsatility index; PCWP, pulmonary capillary blood pressure; PH, pulmonary hypertension; PVR, pulmonary vascular resistance; RHF, right heart failure; RVSWI, right ventricular stroke work index; VAD, ventricular assist device.

ratory biomarkers figure into the schematic, invasive hemodynamic parameters from RHC become a crucial addition to the assessment, especially at the more advanced (C–E) stages of CS (Table 5).

The classical CS invasive hemodynamic profiling is "cold and wet" when low CO is associated with and with elevated systemic vascular resistance (SVR), however, there are several other CS profiling we recognized only with the use of invasive hemodynamic assessment (Table 6, Ref. [14]). Other less frequent subtypes of CS are normotensive and right ventricular (RV) CS. Normotensive CS patients have falsely 'normal' systemic blood pressure due to an elevated SVR but in the presence of low CO. In a post-MI CS registry of over 1000 patients, 5% of those had evidence of hypoperfusion with a systolic blood pressure of more than 90 mmHg [16]. RV shock is present in approximately 5% of patients with post-myocardial infarction CS. Several invasive hemodynamic calculations can be crucial in the determination of right ventricular (RV) versus left ventricular (LV) CS: right atrial pressure (RAP) more than 15 mmHg (mostly validated in post LV assist device (LVAD) patients), the ratio of the RAP and PCWP more than 0.8, pulmonary artery pulsatility index (PAPI) lower than 1.0, and RV stroke work index (RVSWI) <600 mmHg $\times$ mL/m^2 are present in RV failure [17,18].

Cardiac power output (CPO) calculated as mean arterial pressure multiplied by CO and divided by 451 is a strong parameter to assess LV systolic function, especially for patients with chronic systolic heart failure with acute decompensation and a strong predictor of morbidity and mortality [19].

Table 3. 2015 SCAI/ACC/HFSA/STS definition of cardiogenic shock.

Blood pressure criteria	SBP <90 mmHg for >30 min, or the need for supportive measures to maintain an SBP > or =90 mmHg
	Decrease in MAP >30 mmHg below baseline
Hemodynamic criteria	CI < or =1.8 L/min/m^2 without support or <2.2 L/min/m^2 with support
	PCWP > or =15 mmHg

SBP, systolic blood pressure; MAP, mean arterial pressure; CI, Cardiac index; PCMP, Pulmonary capillary wedge pressure; SCAI, Society for cardiovascular angiography and interventions; ACC, American college of cardiology; HFSA, Heart failure society of america; STS, Society of thoracic surgeons.

Table 4. The SCAI stages classification of cardiogenic shock (adapted from Baran *et al.* [15]).

Stage/Name	Description
A "At risk"	The patient is not currently experiencing signs or symptoms of CS but is at risk (i.e., MI, CHF)
B "Beginning CS"	The patient has clinical evidence of relative hypotension without hypoperfusion
C "Classic CS"	The patient manifests with hypoperfusion that requires intervention (inotrope/pressor or MCS) beyond volume resuscitation to restore perfusion
D "Deteriorating/Doom"	The patient is like category C but is getting worse and failing to respond to initial interventions
E "Extremis"	The patient has circulatory collapse, frequent refractory cardiac arrest with ongoing CPR, or is being supported by multiple simultaneous acute interventions including ECPR

Modifier A: cardiac arrest—the presence of cardiac arrest, at any stage, would be considered a subtype (i.e., B_A, C_A, D_A, E_A). CS, cardiogenic shock; MI, myocardial infarction; CHF, congestive heart failure; MCS, mechanical circulatory support; CPR, cardiopulmonary resuscitation; ECPR, extracorporeal membrane oxygenation-facilitated CPR; SCAI, Society for Cardiovascular Angiography and Interventions.

Table 5. Hemodynamic criteria associated with each stage of cardiogenic shock.

SCAI shock stage	Hemodynamic criteria
A	Normotensive and if hemodynamics done: CI >2.5, CVP <10, PA Sat >54%
B	SBP <90 mmHg, or MAP <60 or >30 mmHg drop in blood pressure. If hemodynamics done: CI >2.2, PA Sat >65%
C	Drugs/devices used to maintain blood pressure above stage B values. Hemodynamic Criteria: CI <2.2, PCWP >15, RAP/PCWP >0.8, PAPI <1.85, CPO <0.6
D	Any of stage C parameters requiring multiple pressors or addition of mechanical circulatory support
E	Persistent hypotension and worsening hemodynamics despite maximal support

CI, cardiac index; CPO, cardiac power output; MAP, mean arterial pressure; PAPI, pulmonary artery pulsatility index; PA Sat, pulmonary artery saturation; PCWP, pulmonary capillary blood pressure; RAP, right atrial pressure; SBP, systolic blood pressure.

Table 6. Cardiogenic shock clinical phenotypes (adapted from van Diepen *et al.* [14]).

		Volume status	
		Wet	Dry
Peripheral Perfusion	Cold	Classic CS ("Cold & Wet")	Euvolemic CS ("Cold & Dry")
		High SVR, low CI, high PCWP	High SVR, low CI, low/normal PCPW
	Warm	Mixed CS ("Warm & Wet")	Vasodilatory shock (no CS)
Status		Normal SVR, low CI, high PCWP	Low SVR, high CI, low PCWP

CS, cardiogenic shock; CI, cardiac index; PCWP, pulmonary capillary wedge pressure; SVR, systemic vascular resistance.

Table 7. Pulmonary hypertension hemodynamic definitions from 6th World Symposium on Pulmonary Hypertension.

	Isolated pre-capillary PH (WHO groups 1, 3, 4, 5)	Combined PH (WHO group 2)	Isolated post-capillary PH (WHO group 2)
PCWP (mmHg)	<15	>15	>15
mPAP (mmHg)	>20	>20	>20
PVR (WU)	≥3	≥3	<3

Given the high mortality rate of CS, several institutions have begun deploying 'shock teams' to streamline the expedient diagnosis and management of CS patients. Ideally formed by an advanced heart failure, an interventional

Table 8. Hemodynamic criteria for diagnosis of primary graft dysfunction.

Classification of PGD	Severity of PGD	Hemodynamic criteria
Left ventricular PGD	Mild: one of the following criteria must be met	RAP >15 mmHg, PCWP >20 mmHg, CI <2.0 L/min/m^2 lasting more than one hour requiring low dose inotropes
	Moderate PGD: must meet one criterion from A and another from B	A. RAP >15 mmHg, PCWP >20 mmHg, CI <2.0 L/min/m^2, hypotension with MAP <70 mmHg (lasting more than one hour)
		B. High dose inotropes with an inotrope score >10 or newly placed IABP
	Severe PGD	Dependence on left or biventricular mechanical support including
Right ventricular PGD	Diagnosis requires either both A and B or C alone	A. Hemodynamics with RAP >15 mmHg, PCWP <15 mmHg, CI <2.0 L/min/m^2
		B. TPG <15 mmHg and/or pulmonary artery systolic pressure <50 mmHg
		C. Need for RVAD

BiVAD, biventricular assist device; CI, cardiac index; ECMO, extracorporeal membrane oxygenation; IABP, intra-aortic balloon pump; LVAD, left ventricular assist device; PCWP, pulmonary capillary wedge pressure; RAP, right atrial pressure; RVAD, right ventricular assist device; TPG, transpulmonary pressure gradient.

Table 9. Guidelines for right heart hemodynamic assessment following heart transplantation.

Scenario		Class recommendation	Evidence level
1.	Suggestion of worsening allograft function or concerns for rejection	I	C
2.	Consideration of heart re-transplantation	I	C
3.	Pulmonary hypertension management in heart transplant recipients	IIa	B
4.	At the time of surveillance cardiac biopsy in an asymptomatic patient ≤6 months vs. >6 months	IIa (≤6 months) IIb (>6 months)	C
5.	Suspected tamponade related to endomyocardial biopsy or immunosuppression	IIb	C
6.	Persistent concerns for congestion or renal dysfunction to assess hemodynamics and filling pressures	IIb	C
7.	Concerns of allograft vasculopathy or restrictive physiology	IIb	C

Table 10. Important pulmonary hemodynamics to assess for heart re-transplant candidacy.

Hemodynamic parameter	Abnormal value
Pulmonary vascular resistance	>5 wood units
Indexed pulmonary Vascular resistance	>6
Transpulmonary gradient	>16 mmHg
Pulmonary artery Systolic pressure	>60 mmHg with any one of the above

cardiologist, intensivist, and cardiovascular surgeon, a cardiogenic shock would take time-sensitive decisions, execute a treatment and choose the most appropriate intervention depending on the patient's status in a multidisciplinary approach, from the escalation of vasoactive drugs to temporary mechanical circulatory support (TMCS) devices in the acute shock phase. Decided which patients might need escalation on therapy for potential recovery or advanced heart failure candidacy, like heart transplantation, needs to be taken into account. Tehrani *et al.* [20] published a framework for the design and implementation of shock teams and protocols for both post-acute myocardial infarction CS (AMI-CS) and acute or chronic non-ischemic CS (non-AMI-CS). RHC after suspected CS helps to stratify the need for escalation of shock therapy and earlier utilization of TMCS. Survival of CS at their institution increased from 47% to 77% after implementation of risk scores based on invasive hemodynamic parameters and other clinical markers, with a 39% absolute increase in survival. The authors also developed a multifaceted risk score for CS that included the CPO and PAPI for the 30-day risk of mortality [20]. Similarly, the National Cardiogenic Shock Initiative implemented an algorithm designed to address AMI-CS. This algorithm uses invasive hemodynamic assessment and has changed the paradigm from solely "door-to-balloon" to "door-to-support" with the placement of TMCS before coronary revascularization if the cardiac index (CI) is <2.2 L/min/m^2 and LV end-diastolic pressure is >15 mmHg [21].

Invasive hemodynamic assessment remains essential in patients who are in CS when the etiology is unknown or not well clarified. Moreover, continuous hemodynamic monitoring of the response to therapeutic regimens may help guide medical therapy. Several observational studies have shown that "tuning up" the hemodynamics in circulatory shock patients (i.e., target a PCWP less or equal than 15 mmHg and a RAP less or equal than 8 mmHg while avoiding systemic hypotension and progressive renal insufficiency) has led to a significant change in the critical care management and is associated with improved outcomes [22].

Table 11. New definition of right ventricular failure following LVAD implantation.

Type of right heart failure	Definition
Early acute RHF	RVAD implanted at time of LVAD operation
Early post-implant RHF	i. RVAD implanted within thirty days after LVAD implantation
	OR
	ii. Failure of wean from inotropic or vasopressor medications or inhaled nitric oxide within fourteen days after LVAD implantation
	OR
	iii. Initiating the above support within thirty days of implant for a minimum of fourteen days
Late RHF	i. Implantation of an RVAD after thirty days of LVAD implantation
	OR
	ii. Any hospitalization greater than thirty days post LVAD implantation which requires intravenous diuretics or inotropic support for at least seventy two hours

Table 12. Signs of right heart failure following left ventricular assist device implantation.

Criteria	Signs
Hemodynamic	i. Elevated central venous pressure of at least >16 mmHg
	ii. Mixed venous saturation <50%
	iii. Cardiac index <2.2 L/min/m^2
	iv. Absence of cardiac tamponade and reduction in pump flow of >30% from the previous baseline
Physical exam	i. Ascites
	ii. Limiting edema
	iii. Increased central venous pressure at least halfway up the neck in an upright patient
Signs of end organ dysfunction	i. Renal failure with serum creatinine >2× baseline value
	ii. Liver injury with an elevation of at least 2× upper limit of normal in AST/ALT or total bilirubin >2.0
	iii. Lactate >3.0 mmol/L

4. The role for right heart catheterization in advanced heart failure therapy (stage D) and heart replacement therapy candidacy

Invasive hemodynamic assessment is recommended by both American and European heart failure guidelines for the management of heart failure patients being considered for durable LV assist device (LVAD) or cardiac transplantation. In addition, to evaluate the reversibility of pulmonary hypertension, the use of RHC may have important prognostic implications and may help clinicians in their decision to implant an LVAD or to transplant [23]. For example, a study of 657 consecutive heart transplant recipients in Italy from 2000–2018 found that right heart failure (RHF) defined by low PAPI (<1.68) was associated with the need for renal replacement therapy and primary graft dysfunction [24]. For both LVAD and transplant patients, in an analysis of the previously described ESCAPE trial, the authors found that an increased RAP/PCWP ratio was associated with adverse clinical outcomes [25].

Pulmonary hypertension occurs frequently in patients with heart failure and has important therapeutic and prognostic implications in both inpatient and outpatient management [26]. The gold standard for diagnosis and monitoring of pulmonary hypertension is right heart catheterization. With the publication of proceedings from the 2018 6th World Symposium on Pulmonary Hypertension, updated definitions and subtypes of pulmonary hypertension were elaborated. Most conspicuously, the threshold for mean pulmonary artery pressure was established at 20 mmHg from the prior cutoff of 25 mmHg [27]. The traditional classification into WHO groupings 1–5 remained unchanged, with WHO group 2 defined as PH due to left heart disease. In addition to etiological groupings, the proceedings defined 3 different hemodynamic profiles: isolated pre-capillary pulmonary hypertension, combined pre-/post-capillary hypertension, and isolated post-capillary pulmonary hypertension [27] (Table 7). Of these profiles, combined pre-/post-capillary PH and isolated post-capillary PH are found in patients with left heart disease. Typically, a transpulmonary gradient (mPA-PCWP) greater than 12 mmHg with a diastolic pulmonary gradient (diastolic PA pressure–PCWP) below 3 mmHg is seen in these patients.

In addition to the standard collection of hemodynamic data, vasoreactivity testing and exercise testing are sometimes important in the evaluation of this patient population. For example, vasoreactivity testing (preferably with inhaled

nitric oxide at 10–20 ppm) is performed in patients with PAH (WHO group 1 PH) wherein an absolute drop in mPA by 10 mmHg to an absolute value of less than 40 mmHg while maintaining cardiac output. In advanced HF patients with WHO group 2 PH being considered for transplantation, vasoreactivity testing may be used to assess for fixed PH and prognosticate before transplantation [42]. Exercise testing or fluid challenge during RHC can also be used to unmask pulmonary hypertension in patients with borderline hemodynamic profiles [28].

Finally, precise diagnosis with meticulous attention to detail is crucial when evaluating the heart failure patient with PH for several reasons. Administration of therapies directed towards PAH (WHO group 1 PH) has met with mostly negative results including worsening of HF and increased mortality across a range of PAH therapies [26]. Furthermore, the degree, presence of, and vasoreactivity of PH in patients with advanced HF can have important prognostic consequences when considering the placement and adjustment of advanced therapies including temporary and durable mechanical circulatory support as well as transplantation.

5. The role of invasive hemodynamic monitoring in post-heart replacement therapy management

5.1 The role of invasive hemodynamic monitoring in the management of primary graft dysfunction

The leading cause of death among patients receiving heart transplantation is primary graft dysfunction (PGD) [29]. PGD is defined as LV and/or RV graft failure occurring in the immediate post-transplant period in the absence of an anatomic or immunologic etiology. Early and accurate diagnosis is therefore critical for optimizing outcomes [30]. All patients receiving heart transplantation leave the operating room to assess for PGD and to optimize hemodynamics. The severity of PGD is based on RHC criteria [31] (Table 8).

5.2 The role of invasive hemodynamic monitoring in the long-term management of heart transplant recipients

RHC plays an important prognostic role after cardiac transplantation. In the long-term management of heart transplant recipients, there are only two Class 1 recommendations that require RHC, namely in the assessment of patients that may require heart re-transplantation and in patients who present with signs suggestive of allograft dysfunction (Table 9). The International Society for Heart and Lung Transplantation (ISHLT) position statement also includes Class 2 recommendations for RHC in the management of heart transplant recipients [32].

RHC, in combination with an endomyocardial biopsy, can potentially differentiate patients who are hemodynamically stable from those patients with substantial allograft dysfunction and abnormal hemodynamics to further assist in therapeutic intervention. Patients without hemodynamic compromise in the setting of rejection can be treated with anti-rejection treatment. Those with compromised hemodynamics may require diuretics, pressors, and inotropic assistance (Fig. 2). Therefore, the need for an RHC in these scenarios is critical for prognostication, diagnosis, and management [32,33]. Additionally, RHC has a Class 1 recommendation for patients under consideration for heart re-transplantation [34]. For these patients, RHC is needed to assess for pulmonary vascular resistance, pulmonary artery pressures, and transpulmonary gradient [35] (Table 10). A patient who remains refractory to medical therapy regarding this specific hemodynamics may not be eligible for heart re-transplantation [35,36]. More importantly, patients with elevated pulmonary pressures are at a high risk of morbidity and mortality [37,38].

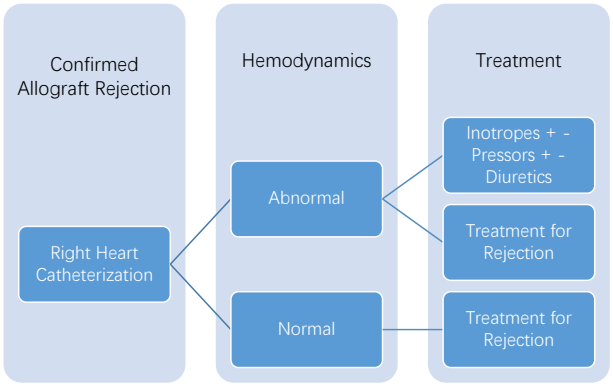

Fig. 2. Right heart catheterization guided treatment mechanism in the setting of rejection.

5.3 The role of invasive hemodynamic monitoring in post-left ventricular assist device management

In the past decade, the definition of RHF has varied across studies. As a result, estimates of RHF after LVAD implantation have varied from 3–40% [39,40]. Additionally, the prior definition of RHF endorsed by the Society of Thoracic Surgeons Interagency Registry for Mechanically Assisted Circulatory Support (STS-INTERMACS) was complex and therefore challenging to apply [40]. As a result, outcomes for RHF following LVAD placement were not presented in the most recent 2020 STS-INTERMACS annual report [36,41]. The academic research consortium has since then attempted to simplify the definition while adding the timing from LVAD implantation and the need for chemical or mechanical RV support [39] (Table 11). In addition to the criteria provided in Table 11 for accurate diagnosis, the patient suspected of RHF must have at least two clinical signs suggestive of RHF (Table 12).

Hemodynamic criteria predictive of the development of RHF following LVAD placement are well studied. However, the studies varied by trial design and used differ-

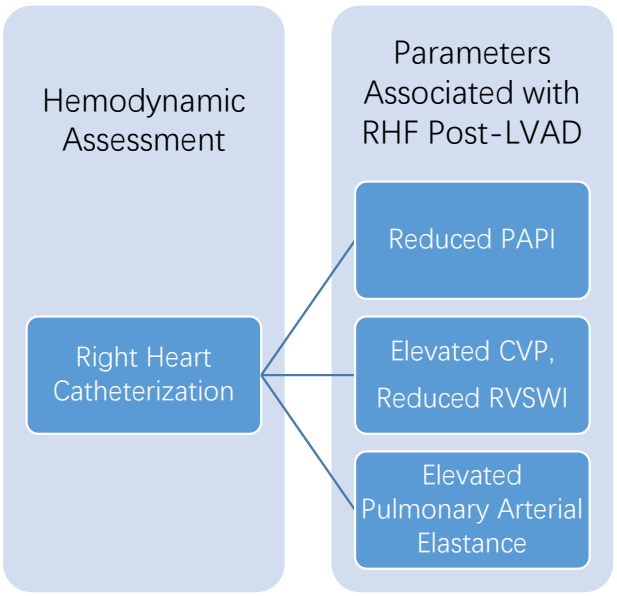

Fig. 3. Hemodynamic parameters associated with right heart failure post left ventricular assist device implantation. CVP, central venous pressure; PAPI, pulmonary artery pulsatility index; RVSWI, right ventricular stroke work index.

ent definitions. Kang *et al.* [42] reported that the PAPI score was an independent predictor of RHF and the need for RVAD support after LVAD implantation. In a 2017 systematic review and meta-analysis of observational studies, Bellavia *et al.* [44] found that elevated CVP and reduced RV stroke work index were the hemodynamic parameters most strongly associated with the development of RHF post LVAD implantation. A 2019 multicenter study of 375 patients by Muslem *et al.* [45] evaluated the relationship between RHC hemodynamic data within the preceding 30 days of LVAD implantation and investigated the relationship to RHF using the INTERMACS definition. The authors found that higher pulmonary arterial elastance (the ratio of systolic pulmonary artery pressure to stroke volume) was a significant predictor of severe RHF following LVAD implantation (Fig. 3) [45].

More importantly, RHF after LVAD implantation requiring RVAD support reduces overall survival to 50% at 1 year and increases morbidity. The current, simplified and more standardized definition for RHF post-LVAD, may potentially allow a more accurate assessment of the hemodynamic variables that need optimization to prevent post-LVAD RHF.

6. Conclusions

For patients with advanced heart failure, RHC will remain an important standard of care for diagnostic and prognostic purposes as well as for long-term management.

Author contributions

Authors AA and JMC contributed equally. CYGM contributed substantially. Each author was involved in every step of the writing process.

Ethics approval and consent to participate

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Conflict of interest

The authors declare no conflict of interest.

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