JACC: HEART FAILURE VOL. 12, NO. 7, 2024

PUBLISHED BY ELSEVIER

STATE-OF-THE-ART REVIEW

Practical Guidance for Hemodynamic Assessment by Right Heart Catheterization in Management of Heart Failure

Navin Rajagopalan, MD,[a] Barry A. Borlaug, MD,[b] Alison L. Bailey, MD,[c] Peter M. Eckman, MD,[d] Maya Guglin, MD, PhD,[e] Shelley Hall, MD,[f] Matthew Montgomery, MD,[g] Gautam Ramani, MD,[h] Prateeti Khazanie, MD, MPH[i]

HIGHLIGHTS

- Measurement and interpretation of hemodynamic data are integral aspects of treating patients with heart failure.
- Right heart catheterization should be performed when patients present with cardiogenic shock, with suspected or known heart failure but do not respond to medical management, and when patients are being assessed for advanced surgical options.
- More data are needed to demonstrate benefit of right heart catheterization in heart failure, in both improving survival and preventing rehospitalizations and end-organ damage.

ABSTRACT

Heart failure is a clinical syndrome characterized by the inability of the heart to meet the circulatory demands of the body without requiring an increase in intracardiac pressures at rest or with exertion. Hemodynamic parameters can be measured via right heart catheterization, which has an integral role in the full spectrum of heart failure: from ambulatory patients to those in cardiogenic shock, as well as patients being considered for left ventricular device therapy and heart transplantation. Hemodynamic data are critical for prompt recognition of clinical deterioration, assessment of prognosis, and guidance of treatment decisions. This review is a field guide for hemodynamic assessment, troubleshooting, and interpretation for clinicians treating patients with heart failure. (J Am Coll Cardiol HF 2024;12:1141–1156) © 2024 by the American College of Cardiology Foundation.

From the [a]Division of Cardiology, University of Kentucky, Lexington, Kentucky, USA; [b]Department of Cardiovascular Medicine, Mayo Clinic, Rochester, Minnesota, USA; [c]Centennial Heart at Parkridge, Chattanooga, Tennessee, USA; [d]Alina Health Minneapolis Heart Institute, Minneapolis, Minnesota, USA; [e]Krannert Cardiovascular Research Center, Indiana University, Indianapolis, Indiana, USA; [f]Baylor University Medical Center, Dallas, Texas, USA; [g]Division of Cardiology, Newark Beth Israel Medical Center, Newark, New Jersey, USA; [h]Division of Cardiology, University of Maryland, Baltimore, Maryland, USA; and the [i]Division of Cardiology, University of Colorado-Anschutz Medical Campus, Aurora, Colorado, USA.

The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

Manuscript received December 22, 2023; revised manuscript received March 26, 2024, accepted March 28, 2024.

ISSN 2213-1779/$36.00 https://doi.org/10.1016/j.jchf.2024.03.020

ABBREVIATIONS AND ACRONYMS

CO = cardiac output

CPO = cardiac power output

CS = cardiogenic shock

HF = heart failure

HFpEF = heart failure with preserved ejection fraction

HFrEF = heart failure with reduced ejection fraction

LVAD = left ventricular assist device

PAP = pulmonary artery pressure

PCWP = pulmonary capillary wedge pressure

PH = pulmonary hypertension

PVR = pulmonary vascular resistance

RAP = right atrial pressure

RHC = right heart catheterization

RV = right ventricle

VO_2 = whole body oxygen consumption

Assessment of invasive hemodynamic status via right heart catheterization (RHC) is integral for the treatment of patients with heart failure (HF). Many noninvasive hemodynamic parameters can be measured using echocardiography.[1] However, RHC is critical when patients have refractory symptoms despite medical management, have symptoms that are out of proportion to objective data, or have unexplained dyspnea despite appearing euvolemic.

The majority of patients living with HF in the community are treated by primary care clinicians, hospitalists, advanced practice providers, and general cardiologists. Thus, the purpose of this guide is to review invasive hemodynamic data for clinicians to optimize the care of patients with HF and to aid in earlier recognition of cardiogenic shock and advanced HF. After a description of the basic principles, techniques, and pitfalls of performing RHC, we describe its role in the management of HF (with both reduced and preserved ejection fraction) and in the management of cardiogenic shock. We also review the optimal application of invasive hemodynamics to guide clinical decision making for left ventricular assist device (LVAD) therapy and heart transplantation. The goal is to help clinicians in practice develop a deeper understanding of how HF specialists use hemodynamic data to assess, treat, and risk stratify patients across the HF spectrum.

PATHOPHYSIOLOGY

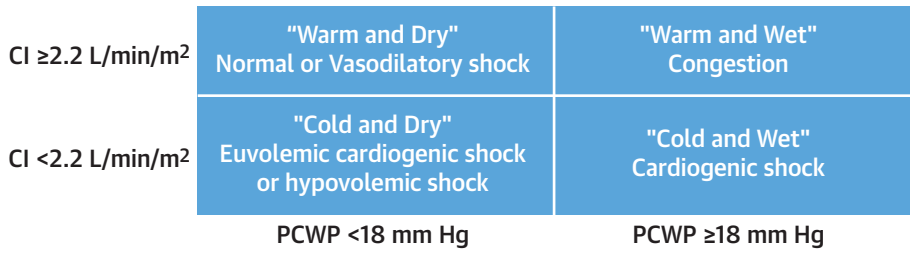
HF is a clinical syndrome that results in an inability of the heart to fulfill the circulatory demands of the body without requiring an increase in intracardiac pressures at rest or with exertion.[2] A reduction in cardiac output (CO) can be present but is not required for the diagnosis. Even when CO is normal at rest, many HF patients do not increase output with activity.[3,4] Elevated systemic venous pressures are also important in HF. Studies have shown that high central venous pressures lead to renal venous congestion and are more strongly associated with worsening renal function as opposed to reduced CO. Moreover, increases in pulmonary capillary wedge pressures are associated with subclinical lung congestion, impaired exercise capacity, and increased risk of HF hospitalization and death.[4-6]

Patients with HF can present with clinical evidence of worsening end-organ function despite maintained CO. Intermittent and sustained elevation in intracardiac pressures in HF lead to end-organ damage, particularly in the lungs and kidneys, as a result of maladaptive neurohormonal and inflammatory pathways.[4] Evidence of volume overload and congestion are known to be adverse prognostic signs in HF,[7] emphasizing the importance of using diuretic agents and guideline-directed medical therapy to normalize intracardiac filling pressures and improve hemodynamic status. Without appropriate decongestion, chronically elevated left-sided filling pressures and pulmonary congestion can lead to changes in the pulmonary vasculature and resultant secondary pulmonary hypertension (PH).[8] The development of PH and subsequent right ventricular dysfunction are adverse prognostic signs in HF, regardless of the left ventricular ejection fraction (LVEF).[9]

Patients can stratified into 4 categories based on volume status (wet or dry) and CO (warm or cold), which can aid in treatment and prognostication (**Figure 1**).[10,11] However, the assignment of patients to 1 of these categories based on physical examination findings has limitations, particularly in patients with chronic and advanced HF.[12] Elevated right-sided pressures as seen with jugular venous distension may not correspond with elevated left-sided filling pressures; conversely, some patients may have high left-sided pressures in the absence of jugular venous distension.[13] In these settings where volume status is uncertain or when patients fail to respond to diuretics, RHC can inform medical decision making. Additionally, low CO can be difficult to appreciate in patients with chronic HF, inasmuch as end-organ damage and elevated lactate may not be present until the final stages of the disease process.[4,14] Unlike patients with acute myocardial infarction, patients with chronic HF can experience low CO insidiously and report symptoms interfering with quality of life, instead of symptoms more commonly associated with cardiogenic shock. Early identification of these patients with invasive hemodynamic measurements is critical for risk stratification and to identify candidates for inotropic support, LVAD therapy, cardiac transplantation, and palliative care.[15]

For patients with normal LVEF and unexplained dyspnea, RHC with exercise stress testing is useful in determining whether patients have heart failure with preserved ejection fraction (HFpEF) or a noncardiac cause of dyspnea.[16] Such testing requires special equipment with a cycle ergometer and is more commonly performed at tertiary medical centers. Approximately one-third of patients with HFpEF have normal pulmonary capillary wedge pressure (PCWP) at rest, with elevations in filling pressures

FIGURE 1 Hemodynamic Classification of Heart Failure Patients



Right heart catheterization to classify patients based on cardiac index and pulmonary capillary wedge pressure (PCWP) can help facilitate optimal medical therapy. CI = cardiac index; PCWP = pulmonary capillary wedge pressure.

exclusively during exercise.[6] These patients frequently have normal or near-normal levels of natriuretic peptides; physical examination results or echocardiographic abnormalities suggestive of congestion may be absent.[17] However, patients with earlier stages of exercise-induced elevation in PCWP still have a 2.4-fold increased risk of HF hospitalization or death and may not receive treatment without invasive exercise evaluation or unless clinical suspicion is high.[6]

ESSENTIALS AND COMMON PITFALLS OF RHC

Vascular access for RHC is typically obtained via the internal jugular, femoral, or brachial veins under ultrasound guidance. The use of ultrasound allows the procedure to be performed safely without the need to stop systemic anticoagulation. The balloon-tipped catheter is then advanced through the right heart to obtain right atrial pressure (RAP), right ventricular (RV) pressure, pulmonary artery pressures (PAPs) (systolic, diastolic, mean), and PCWP (**Figure 2**). Vascular access through the right internal jugular vein can eliminate the need for fluoroscopy because the balloon-tipped catheter is more naturally directed toward the PA.

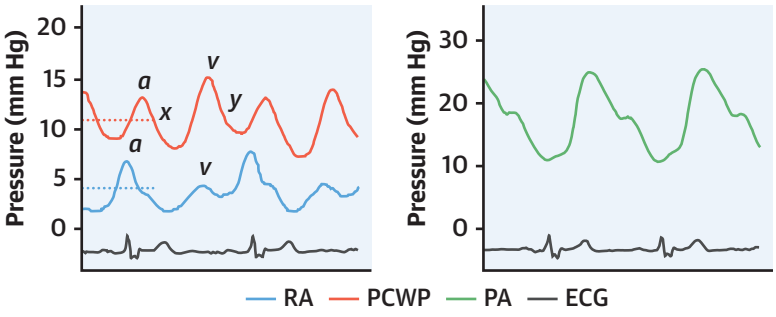
Proper zeroing technique of the pressure transducer is critical for data accuracy (**Table 1**). Before obtaining pressure measurements, the transducer should be zeroed at the level of the right atrium, usually measured at the midthoracic level.[18] If there are hemodynamic data that appear inconsistent with the patient's clinical presentation, the pressure transducer should be re-zeroed, and the catheter and lines should be flushed with saline solution to ensure accurate measurements. Pressures should be recorded at end-expiration during normal respiration when the lungs are at functional residual capacity to avoid the impact of negative pleural pressures.[19] Conscious sedation can alter a patient's breath cycle; oversedation is a common pitfall that can affect data accuracy. In patients with lung disease or morbid obesity, there can be prominent changes in intrathoracic pressures during the respiratory cycle.[18] With significant respiratory variation in RAP and PCWP tracings, both the mean and end-expiratory values should be noted, although the clinical significance of this variation is uncertain.[20]

CO is calculated using the direct Fick method or thermodilution and divided by body surface area to obtain cardiac index. For determination of Fick CO, mixed venous oxygen saturation is collected from the main PA, and arterial saturation is recorded from noninvasive oximetry or arterial blood sampling. Whole body oxygen consumption (VO_2) should be directly measured, because assumed values deviate >25% from true VO_2 in many patients (causing similar deviations in CO).[21] Fick CO is the gold standard, but direct measurement of VO_2 is often not available. The use of assumed VO_2 values has led to the reporting of "indirect" Fick CO, which should be avoided because of the potential for error. Thermodilution is the preferred method when direct VO_2 measurements cannot be obtained. Thermodilution-based CO may be influenced by the presence of tricuspid regurgitation, but studies have shown reasonable accuracy even in these settings.[22] Potential errors of thermodilution method include variations in the volume of saline solution injected, transient arrhythmias, changes in preload caused by prominent respiratory variation, and catheter malpositioning.[23] Thermodilution CO is unreliable when an intracardiac shunt is present.

For patients presenting for their first RHC, blood from the superior vena cava and PA should be sampled to check for differences in oxygen saturation resulting from an occult intracardiac shunt. A shunt

FIGURE 2 Basics of RHC Hemodynamics

Variable	Normal Range (mm Hg)
Right atrium	0-6
Right ventricle	Systolic: 20-34 Diastolic: 0-6
Pulmonary artery	Systolic: 20-34 Diastolic: 8-12 Mean: ≤20
Pulmonary capillary wedge pressure	4-14



Normal values associated with right heart catheterization (RHC) along with representative hemodynamic tracings. For right atrial pressure (RAP) and PCWP, the a-wave represents atrial contraction, and the v-wave represents venous return into the atria. X- and y-descents are also noted. ECG = electrocardiogram; PA = pulmonary artery; RA = right atrium; other abbreviation as in Figure 1.

should also be considered if pulmonary artery saturation is higher than expected for the clinical situation. It is important to record the patient's systemic blood pressure during the procedure. Elevations in blood pressure may be caused by high systemic vascular resistance (SVR), which can have important therapeutic implications in the treatment of patients with HF, such as the need for maximizing afterload reduction. Common calculations to perform during RHC are shown in Table 2.

Several clinical conditions resulting in characteristic hemodynamic tracings may be detected at the time of RHC and are important to recognize. Constrictive pericarditis can be missed when patients have refractory volume overload and present with a presumed diagnosis of HFpEF or restrictive cardiomyopathy.[24] RA pressure is often elevated with accentuated "y" descent (more prominent than the "x descent) resulting from rapid early diastolic filling of the ventricles, with a positive Kussmaul sign (paradoxical increase in RA pressure with inspiration). These findings are not specific to constrictive pericarditis, being commonly observed in patients with advanced HF with a failing, noncompliant RV, indicating poor prognosis.[25] Dynamic respiratory maneuvers during simultaneous left and right heart catheterization provide greater sensitivity and specificity to distinguish constriction from myocardial disease based on the presence or absence of enhanced ventricular interdependence and intracardiac-intrathoracic dissociation (Figure 3A).[24]

Mean PCWP should be reported at mid a-wave (or c-wave for patients in atrial fibrillation) at end-expiration to provide the most accurate surrogate for left ventricular end-diastolic pressure. A common finding is a prominent v-wave in the PCWP tracing and can be indicative of hemodynamically significant mitral regurgitation (Figure 3B). However, a prominent v-wave is most often related to reduction in operant left atrium compliance, resulting from either a stiff left atrium or one that is filled to the steep portion of its pressure-volume relationship. This distinction between significant mitral regurgitation and reduced left atrial compliance is important in the era of catheter-based mitral valve repair for patients with HF. Echocardiography is the main tool for distinguishing between these states, but RHC can play a supplemental role when clinical and echocardiographic data are discordant.[26] The presence of significant v-waves should be noted on the final report. When PCWP is averaged over the entire cardiac cycle in patients with significant v-waves, left ventricular end-diastolic pressure will be overestimated. In this situation, the operator should report the value of the mean PCWP at mid a-wave and the mean PCWP over the entire cardiac cycle. Clinical consideration should be used to estimate how the v-wave amplitude might influence the reported pulmonary vascular resistance

TABLE 1 Common Clinical Occurrences During Right Heart Catheterization

	Mechanism	Troubleshooting
Improper zeroing	• If pressures are consistently higher or lower than expected, the level of the transducer may be incorrect	• Pressure transducer should be zeroed to atmospheric pressure at the left of the right atrium, which is located at midthorax level • Lines should be flushed to remove any air bubbles
Overdamping	• Can result in false reduction of systolic pressures and elevation of diastolic pressures • Clues are waveforms that appear "rounded" and loss of dicrotic notch in PA waveform • Causes include catheter kinking or thrombus, air in the system, or loose connections	• Catheter and the tubing leading to pressure transducer should be inspected and flushed
Catheter whip	• Excessive movement of catheter in vessel can cause erratic waveforms	• Reposition catheter • Partial or complete deflation of balloon • Ensure tubing between catheter and transducer is not being moved or manipulated during waveform acquisition
Under-wedged PCWP tracing	• If balloon catheter is inadequately wedged, the higher PA pressure waveform will interfere with PCWP waveform, leading to false elevation of recorded PCWP • PCWP waveform may look similar to PA waveform	• Measure PCWP saturation in all cases, and saturation should be within 5% of systemic saturation • If unable to wedge, consider direct measurement of LVEDP
Overwedged PCWP tracing	• Characterized by loss of a-waves and v-waves and lack of respiratory variation • Inflating balloon fully in distal PA may result in overwedging and false elevation of PCWP • Can occur when end-hole of catheter is not parallel to the vessel but compressed against side wall • Overwedging can cause pulmonary artery rupture	• Avoid full inflation of balloon in distal PA • Deflate balloon and withdraw catheter to the proximal PA, and inflate balloon and advance
Significant respiratory variation	• Due to large changes in intrathoracic pressures • More commonly observed in patients with obesity or lung disease	• Report should note the presence of significant respiratory variation • Report average pressure over entire respiratory cycle as well as pressures at end-expiration • Avoid sedation
Mitral regurgitation leading to tall v-waves in PCWP	• With large v-waves, mean PCWP may be significantly higher than end-diastolic PCWP, which is typically what is recorded, because it corresponds to LVEDP	• Large v-waves contribute to the increase in mean PAP, and therefore mean PCWP should be used to calculate PVR • Report should note presence of v-waves as well as mean PCWP and end-expiratory PCWP
Inaccurate CO (thermodilution)	• Direct Fick CO is considered gold standard but usually not available because of inability to directly measure oxygen consumption • Changes in preload caused by large changes in intrathoracic pressures (respiratory variation) • Operator error is possible, particularly among new operators/trainees, leading to variation in volume of saline solution injected • Thermistor abutting vessel wall	• Average 3-5 measurements and ensure reproducibility • Injection of saline solution at same point in respiratory cycle • Good PA tracing indicates thermistor is not against a vessel wall • Inaccurate in intracardiac shunts and should not be reported • Preferred method when oxygen consumption cannot be directly measured to calculate Fick CO
Inaccurate CO (Fick)	• Oxygen consumption should be directly measured • Assumed oxygen consumption values available in most laboratories are not reliable in patients with obesity or systemic disease such as HF	• Avoid reporting Fick CO if oxygen consumption is not directly measured

CO = cardiac output; HF = heart failure; LVEDP = left ventricular end-diastolic pressure; PA = pulmonary artery; PAP = pulmonary artery pressure; PCWP = pulmonary capillary wedge pressure; PVR = pulmonary vascular resistance.

(PVR) vs the true resistance to flow across the lungs. Prominent v-wave can also be caused by a ventricular septal defect, which should be considered depending on the clinical context (ie, recent myocardial infarction) and thereby necessitate shunt evaluation via oximetry.

When there is uncertainty whether the hemodynamic tracing represents an accurate PCWP, blood should be aspirated to measure oxygen saturation. This value represents the oxygenation of the postcapillary pulmonary circulation, which should be within 5% of the systemic oxygen saturation (>90%-95%) in the absence of hypoxia. If this is not the case, the catheter should be repositioned to obtain an accurate estimate of left atrial pressure.[27]

CLINICAL SCENARIOS WHERE RHC PLAYS A KEY ROLE

CARDIOGENIC SHOCK. Cardiogenic shock (CS) is defined as severe myocardial dysfunction with resultant decreased CO and systemic hypoperfusion, complicated by tissue ischemia and/or hemometabolic derangement.[28] To help identify and risk stratify patients with CS, the SCAI (Society for Cardiovascular Angiography and Interventions) in 2019 published an

TABLE 2 Calculations to Obtain During RHC

	Formula	Normal Range	Significance
Transpulmonary gradient, mm Hg	Mean PAP − PCWP	<12	• Chronic elevations in left-sided filling pressures in HF can lead to reactive pulmonary vascular remodeling and elevation in TPG
Pulmonary vascular resistance, WUs	TPG/CO	<2	• PVR can be elevated with chronic HFrEF and HFpEF and is associated with increased CV mortality • Elevated PVR >3 WUs is a risk factor for RV failure after heart transplantation, and acute pulmonary vasodilator testing is recommended to assess for reversibility
Systemic vascular resistance, dyne·s/cm^5	$\frac{MAP - RAP}{CO} \times 80$	900-1,200	• Often elevated in CS or decompensated HF • Low SVR in CS may indicate coexisting inflammatory response or active infection
Pulmonary artery pulsatility index	$\frac{PA\ systolic - PA\ diastolic}{RAP}$	>2.0	• Estimates RV pulsatile load and contractile strength and by using RAP, also incorporates a measure of RV congestion [68] • PAPI <1.85 is a risk factor for RV failure after LVAD [92]
Right ventricular stroke work index, g × m/m^2/beat	(Mean PAP-RAP) × (CI/HR × 1,000) × 0.0136	>8	• Estimates RV workload and contractility through measurement of RV pressure (mean PAP) and flow (cardiac index) • Low RVSWI is associated with RV failure after LVAD
Cardiac power output, Watts	$\frac{(MAP - RAP) \times CO}{451}$	>1.0	• Incorporates measures of both pressure and flow to estimate cardiac pumping capability • Used in cardiogenic shock as prognostic factor and in decision to escalate therapy

CS = cardiogenic shock; HR = heart rate; LVAD = left ventricular assist device; MAP = mean arterial pressure; PAPI = pulmonary artery pulsatility index; RA = right atrial; RV = right ventricle; RSWI = right ventricular stroke work index; SVR = systemic vascular resistance; TPG = transpulmonary gradient; other abbreviations as in Table 1.

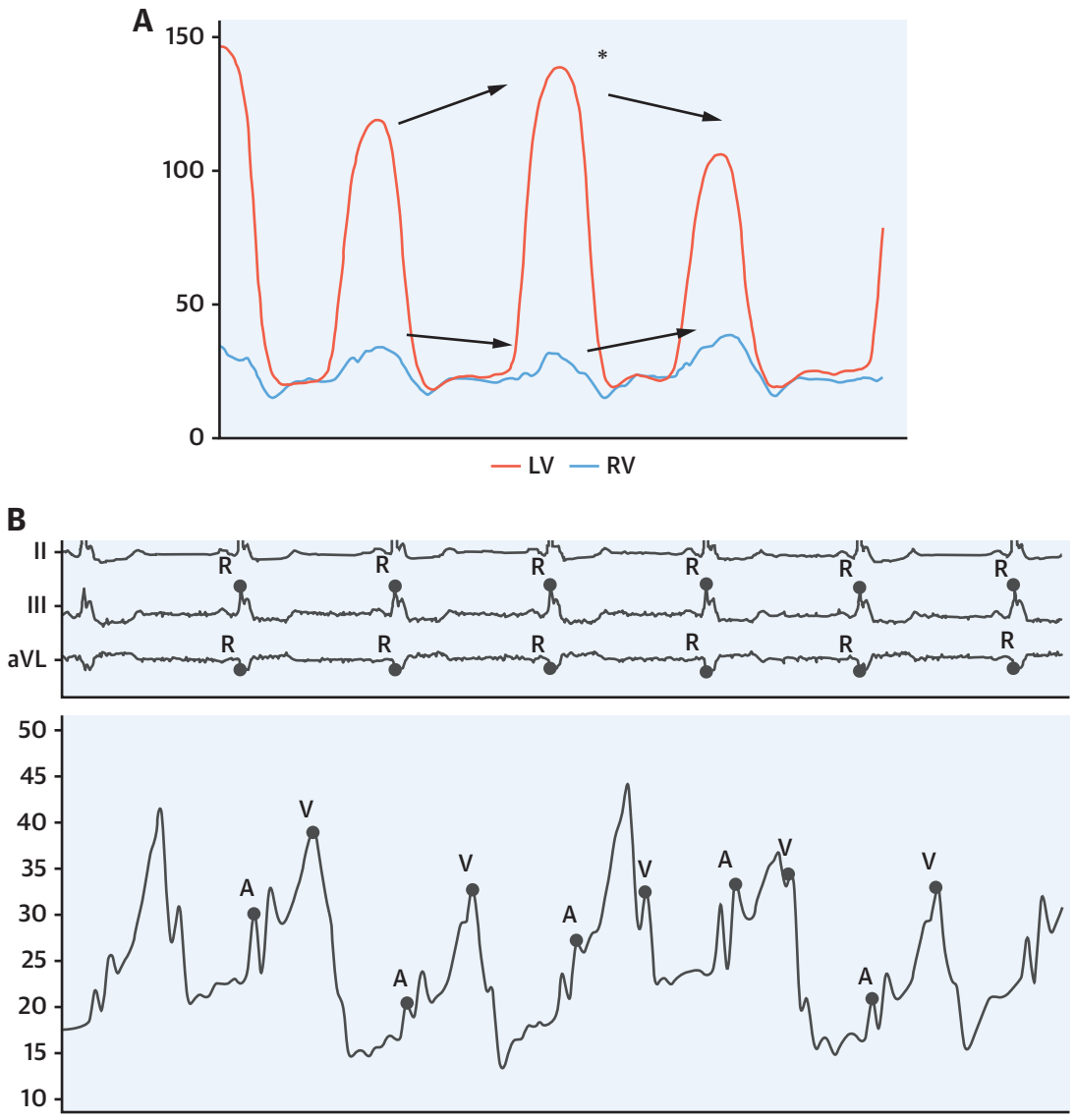
updated consensus statement in which CS was categorized into 5 stages (Figure 4).[29] Hemodynamic data via RHC and biochemical markers have a prominent role in this classification algorithm. Hypoperfusion characterized by elevated lactate and leading to worsening renal and hepatic function is more ominous than hypotension, which is often normal in the early onset of CS.[30,31] Although there are no prospective clinical trials, observational studies suggest mortality benefit with the use of RHC in CS.[32,33] Continuous monitoring enables treatment to hemodynamic goals, such as RAP 8-12 mm Hg, PCWP ≤15 mm Hg, and cardiac index ≥2.2 L/min/m^2. Monitoring enables more rapid recognition of clinical deterioration if these targets are not achieved, leading to earlier escalation to inotropic support, temporary mechanical circulatory support, or more durable advanced HF therapies.[34] Finally, RHC can distinguish the different hemodynamic profiles of shock in hypotensive patients, including cardiogenic and vasodilatory shock. Mixed shock is an important consideration when patients have elements of CS along with low SVR usually seen with vasodilatory shock.

CS as a result of progressive HF (HF-CS) has a different clinical presentation compared with CS as a result of acute myocardial infarction (AMI-CS).[34] Abrupt myocardial dysfunction in AMI-CS leads to rapid clinical deterioration, whereas the insidious onset of low CO seen in HF-CS allows for chronic adaptations that may conceal low CO until the body is ultimately no longer able to compensate. Patients with HF-CS are more likely to have severe hemodynamic derangements but preservation of end-organ function and normal serum lactate.[34,35] The complexity of CS, driven by overlapping clinical and physical findings, varying disease phenotypes, and rapid progression can make prompt diagnosis challenging.

CO is used to measure overall cardiovascular function in patients with CS. Further calculation of cardiac power output (CPO) combining both flow (CO) and pressure (MAP) measurements, can provide more prognostic information (Table 2).[36] In clinical practice, RAP is often omitted from CPO calculations; however, given that it is frequently elevated in CS, this can lead to error. Although an analysis by the Cardiogenic Shock Working Group failed to demonstrate that CPO was predictive of mortality, a smaller study found an association between CPO and in-hospital mortality (using RAP in the formula).[37,38] The clinical value of CPO lies in its ability to identify patients with CS who are at risk for further deterioration. In patients with low CPO (<0.6 W) in the setting of systemic hypotension despite increasing vasopressor and/or inotropic support, escalation to temporary support devices should be considered.[39,40] Even among patients who are not in shock, the inability to enhance CPO with exertion is prognostic, further supporting its clinical utility.[41]

RHC can also determine whether CS is resulting from left ventricular, right ventricular, or

FIGURE 3 Examples of Hemodynamic Tracings

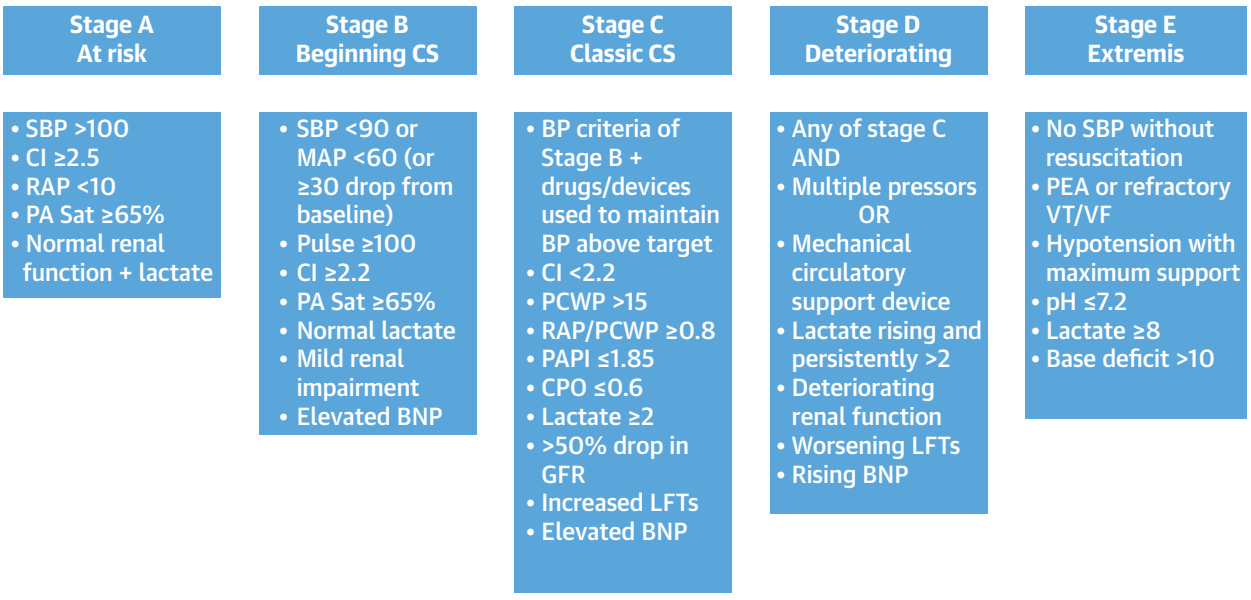


(A) Ventricular interdependence in constrictive pericarditis. With inspiration (asterisk), left ventricular (LV) systolic pressure (red) falls as the pressure gradient from the pulmonary veins to the left heart decreases, leading to acute LV underfilling and fall in stroke volume. At the same time, right ventricular (RV) systolic pressure (blue) rises as venous return to the right heart is amplified, increasing RV stroke volume and RV pressure in ensuing beat. The opposite occurs during expiration. (B) PCWP tracing in a patient with severe mitral regurgitation. Tall v-waves >30 mm Hg are observed. Abbreviation as in **Figure 1**.

biventricular failure (**Central Illustration**).[42] Right HF is a driver of poor outcomes in CS, with several hemodynamic variables associated with increasing mortality: RAP >15 mm Hg, RAP/PCWP ratio >0.6, pulmonary artery pulsatility index (PAPI) <1.5, and RV CPO <0.3 W(where mean PAP replaces MAP in the CPO equation).[38,43] Although isolated RV shock is rare, biventricular CS is increasingly recognized. The RV is particularly sensitive to volume and pressure overload, highlighting the importance of diuretic agents to lower RAP and PCWP and improve RV mechanics.[44] It remains to be seen if improved detection of RV failure by RHC will lead to improved outcomes in patients with CS.

Patients in CS are often initially stabilized with vasopressors and inotropic support. Serial

FIGURE 4 Cardiogenic Shock Classification

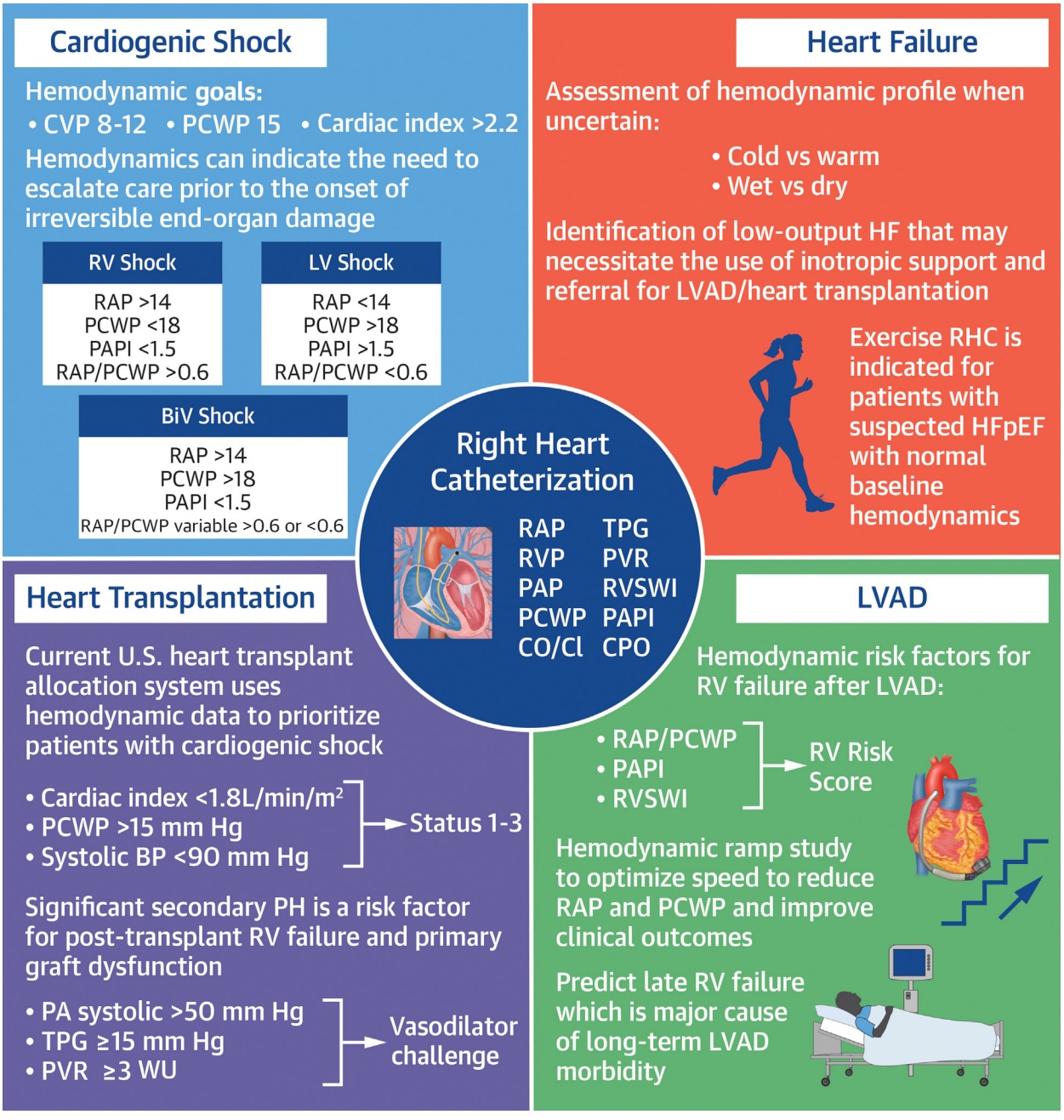


Hemodynamic features associated with the 5 Society for Cardiovascular Angiography and Interventions stages of cardiogenic shock. BNP = B-type natriuretic peptide; BP = blood pressure; CPO = cardiac power output; CS = cardiogenic shock; GFR = glomerular filtration rate; LFT = liver function test; MAP = mean arterial pressure; PA = pulmonary artery; PAPI = pulmonary artery pulsatility index; PEA = pulseless electrical activity; RAP = right atrial pressure; Sat = saturation; SBP = systolic blood pressure; VF = ventricular fibrillation; VT = ventricular tachycardia; other abbreviations as in Figure 1.

assessment of invasive hemodynamics is essential to ascertain the adequacy of initial stabilization efforts. Patients who do not require vasopressors may respond to vasodilator therapy, allowing the weaning of inotropic support. Careful assessment of hemodynamic status while medical therapy is up-titrated can allow for stabilization without the need for escalation. However, patients who do not respond or whose condition deteriorates may require escalation, including temporary mechanical support, to prevent end-organ damage. After the implantation of temporary mechanical circulatory support in the left ventricle, RV, or both ventricles, hemodynamic monitoring should be used to assess for clinical stability/deterioration and for potential weaning and device removal.[45] If temporary support fails, patients may need evaluation for advanced HF therapies, such as LVAD or heart transplantation to prolong survival.[15] Such evaluation involves medical, psychosocial, and financial considerations, and these therapies are not long-term options for all patients. Durable RV or biventricular support devices are clinically challenging because these patients are often unable to be discharged. Thus, for patients with severe refractory RV or biventricular failure, transplantation is often the only feasible long-term therapy if they qualify. Complex decision making for patients with CS necessitates that HF and surgical specialists are integrated into CS teams to determine the optimal treatment options that are consistent with patients' goals and values.[46]

HFrEF. Volume overload is prevalent among patients hospitalized with acute decompensated heart failure with reduced ejection fraction (HFrEF). Although RHC is not appropriate for routine use, it can be helpful when volume status is uncertain or when patients do not respond to diuretics. Unfortunately, clinicians in practice may hold diuretic agents inappropriately because of concerns for worsening renal function and cardiorenal syndrome.[47] In these settings, RHC can be useful to demonstrate elevated filling pressures, indicating that renal venous congestion is contributing to renal dysfunction and emphasizing the need to administer diuretic agents and vasodilators. An important caveat in using RHC pressures to evaluate volume status is that correlation between pressure and volume may be modest. How to incorporate measures of volume status with

CENTRAL ILLUSTRATION Use of Right Heart Catheterization in Heart Failure



Rajagopalan N, et al. J Am Coll Cardiol HF. 2024;12(7):1141–1156.

RHC provides important diagnostic and prognostic information for the full spectrum of heart failure, including cardiogenic shock, prioritization of candidates for heart transplantation, and treatment of LVAD patients. BiV = biventricular; CO = cardiac output; CVP = central venous pressure; HFpEF = heart failure with preserved ejection fraction; LV = left ventricle; LVAD = left ventricular assist device; PH = pulmonary hypertension; PVR = pulmonary vascular resistance; RHC = right heart catheterization; RV = right ventricle; RVSWI = right ventricular stroke work index; TPG = transpulmonary gradient.

hemodynamic measurements is an area of active investigation.[48]

When a patient has low CO and elevated SVR with maintained perfusion, initiation and careful titration of vasodilatory therapy can improve hemodynamic status. This can include angiotensin receptor-neprilysin inhibitors and other oral guideline-directed medical therapy.[49,50] If decompensation is particularly severe, nitroprusside can be considered. This strategy can allow for continued up-titration of neurohormonal therapies and clinical stabilization without the urgent need for advanced HF therapies. For patients with chronic HF on guideline-directed medical therapy, low-output HF may represent disease progression requiring inotropic support, particularly if there is evidence of hypoperfusion (**Table 3**).[2,51] In the United States, the only approved inotropic medications are both parenteral: dobutamine and milrinone. The risks of extended inotropic support include hypotension, arrhythmias, and risks of infection and venous thrombosis from long-term intravenous access.[52] Given these risks and that early clinical studies demonstrated increased mortality with inotrope use instead of prolonged survival, inotropic support should be considered as a bridge to clinical improvement, bridge to advanced HF therapies such as LVAD and heart transplantation, or palliation.[53] Decisions regarding candidacy for LVAD and heart transplantation are complex, and inotropic support can stabilize a patient's condition while a comprehensive evaluation is ongoing.

HFpEF. The incidence and prevalence of HFpEF have grown by epidemic proportions, overtaking HFrEF as the dominant cause of HF.[16,54] The criteria for diagnosis of HFpEF have evolved over the years. Although abnormalities on echocardiogram such as left ventricular hypertrophy may be present, in about one-third of patients with HFpEF, left ventricular filling pressures are normal at rest and only increase during exercise, meaning that physical examination findings of hypervolemia at rest are absent. Natriuretic peptides are normal in approximately one-third of individuals with HFpEF, particularly in those with obesity, a common comorbidity in HFpEF.[55] Clinicians must have a low threshold for considering HFpEF as the diagnosis in patients with unexplained dyspnea or exercise intolerance despite euvolemia on examination and normal natriuretic peptides.

The gold standard diagnostic test for HFpEF is an invasive hemodynamic exercise stress test.[16] This involves measurement of cardiac hemodynamic status via RHC at rest and with exercise. The normal response to exercise is a modest increase in both mean PA pressure (<3.0 mm Hg/L/min increase in CO) and PCWP (<25 mm Hg) with a PVR that decreases from rest because of vascular distension, recruitment, and flow-mediated vasodilation.[16,56] Exercise echocardiography has been proposed as an alternative over invasive hemodynamic exercise, but inadequate sensitivity has been a limitation in studies to date.[16]

TABLE 3 Clinical Criteria to Suggest the Need for Right Heart Catheterization in Patients With Chronic HFrEF

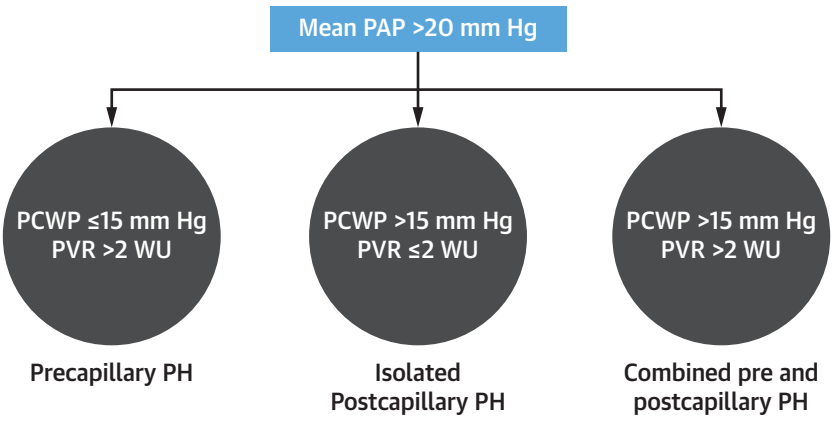
Worsening fatigue
Hypotension with narrow pulse pressure
Cool extremities and S3 gallop
Down-titration of guideline-directed medical therapy
Recurrent HF hospitalizations with no other precipitating factors identified
Worsening renal function
Worsening liver function test results (increasing bilirubin, transaminases)

HFrEF = heart failure with reduced ejection fraction; other abbreviation as in **Table 1**.

Exercise testing is usually performed at institutions with access to supine or upright cycle ergometers. Exercise protocols vary across centers but include escalating exercise workload to patient fatigue; simultaneous measurements of RAP, PAP, and PCWP; CO measurement using either direct Fick or thermodilution; measurement of gas exchange using a metabolic cart, and assessment of arterial and venous oximetry.[19] Detailed discussion regarding exercise hemodynamics is beyond the scope of this paper, but some points are worth mentioning. Upright exercise is more typical of daily life but requires specialized equipment, which may not be readily available at all centers. Intracardiac pressures are lower when the individual is in the upright position than in a supine position, both at rest and during exercise. Small studies have suggested that changes with exercise are similar in both positions.[57] Elevation in left heart filling pressures during exercise is prognostic, irrespective of body position.[5,6] Hemodynamic data measured during end-expiration are higher than average values, demonstrating that respiratory-averaged and end-expiratory measurements cannot be used interchangeably.[58] One study demonstrated that end-expiratory PCWP more strongly correlated with lung congestion as compared with PCWP averaged over the entire respiratory cycle.[59]

PULMONARY HYPERTENSION CAUSED BY HF. PH is common in both HFrEF and HFpEF and is often suggested by echocardiography. Numerous studies have shown that elevated PA pressures are an adverse prognostic factor in HF.[60,61] Although an echocardiogram can suggest PH, RHC is required to confirm

FIGURE 5 Diagnosis of Pulmonary Hypertension



Hemodynamic definitions for precapillary and postcapillary pulmonary hypertension. PAP = pulmonary artery pressure; PH = pulmonary hypertension; PVR = pulmonary vascular resistance; other abbreviation as in Figure 1.

the diagnosis. The World Symposium on PH revised the hemodynamic definition of PH to mean PAP >20 mm Hg (from a previous definition of >25 mm Hg). PH phenotypes are determined by the PCWP, CO, and PVR (Figure 5).[62]

RHC is not always necessary when PH is suspected by echocardiogram. In many situations, patients with HF have elevated PA pressures caused by an increase in left-sided filling pressures (ie, isolated postcapillary PH). There may be a subset of patients with HF who experience PH that is "out of proportion" to left heart disease; these patients have both an elevated PCWP and elevated PVR, or combined pre- and post-capillary PH. These patients display more adverse hemodynamic data, greater RV dysfunction, and more severe impairments in pulmonary gas exchange, and they are at high risk for adverse clinical events, but treatment with pulmonary arterial vasodilators has not shown benefit.[63,64] For patients with normal LVEF, RHC can identify patients with precapillary PH in the absence of HF, on the basis of elevated mean PAP and normal PCWP. Distinguishing these patients from patients with HFpEF is important because beneficial treatment options exist for those with pulmonary arterial hypertension. Another form of PH that has gained recent attention is exercise-induced PH, in which an exaggerated increase in mean PAP is observed during exercise RHC, although detailed discussion is beyond the scope of this review.[65]

LVADs. Guidelines recommend RHC within 1 to 2 weeks of elective LVAD implantation and immediately before surgery for critically ill patients to assess for the presence and severity of RV dysfunction.[66] RV failure remains a leading cause of morbidity and mortality after durable LVAD implantation. Echocardiography provides information regarding RV size and function and severity of tricuspid regurgitation, but invasive hemodynamic assessment is paramount in the risk assessment for RV failure. Indices that suggest higher incidence of RV failure after LVAD implantation include RAP >15 mm Hg, RAP/PCWP ratio >0.63, and pulmonary artery pulsatility index <1.85.[67,68] No single parameter has demonstrated clear superiority, so the incorporation of hemodynamic data with clinical and echocardiographic data may offer optimal risk assessment.[69]

Hemodynamic optimization before LVAD implantation can be helpful to relieve congestion, minimize RV overload, and maintain end-organ perfusion.[66] With continuous hemodynamic monitoring, diuretic agents to achieve RAP <15 mm Hg and inotropic support to optimize RV function are recommended.[70,71] In patients with severe RV dysfunction, right-sided ventricular assist device support may be planned at the time of LVAD surgery to better support the circulation and improve outcomes, although prospective data are lacking.[72] Continuous hemodynamic monitoring is frequently used during the immediate postoperative period in the intensive care unit to guide inotropic support, diuretics, and pulmonary vasodilators such as nitric oxide, to optimize right-sided hemodynamic status, and to monitor LVAD flow. The onset of hypotension may be caused by hypovolemia, RV failure, or inadequate LVAD support, and knowledge of hemodynamic status is

TABLE 4 Use of RHC in Patients With Durable LVAD

	Rationale	Recommendation Level[a]
Recurrent HF symptoms	• RV failure • Device malfunction • Inadequate LV unloading	Class I
Heart transplantation evaluation	• Assess TPG and PVR • Reversibility testing if needed (PVR >3 WUs) • Serial testing if extended wait-time	Class I
Ramp study	• Optimize LVAD speed to lower RAP and PCWP • Improve functional status and possibly prevent LVAD complications	Class IIa
Myocardial recovery	• Assess hemodynamic status with serial lowering of pump speed • Confirm normal hemodynamic status before LVAD explantation	Class IIa

[a]Based on the 2023 International Society for Heart and Lung Transplantation Guidelines for Mechanical Circulatory Support: A 10-Year Update (Saeed et al[66]).

LV = left ventricle; RAP = right atrial pressure; RHC = right heart catheterization; other abbreviations as in Tables 1 and 2.

crucial for correct diagnosis. Distortion of RV anatomy after LVAD implantation can unmask underlying RV dysfunction even if it was not apparent preoperatively.[73]

Even after LVAD support, many patients continue to demonstrate abnormal hemodynamic profiles, including elevated RAP and PCWP, and abnormal functional capacity.[74,75] LVAD speed changes (ie, ramp optimization) during RHC and echocardiography can improve hemodynamic variables in such patients.[76] Hemodynamic goals to attain include RAP <12 mm Hg and PCWP <18 mm Hg, along with intermittent opening of the aortic valve, and minimal mitral regurgitation.[66] Observational studies suggest that hemodynamic ramp optimization can improve functional capacity, reduce readmissions, and lower LVAD complications.[77,78] The 2023 ISHLT (International Society for Heart and Lung Transplantation) Guidelines for Mechanical Circulatory Support recommend RHC to select an appropriate LVAD speed, improve hemodynamic status, and optimize RV function (Class IIa recommendation).[66] Thus, invasive ramp tests may become the standard of care for the treatment of patients with LVADs, although further study is necessary.

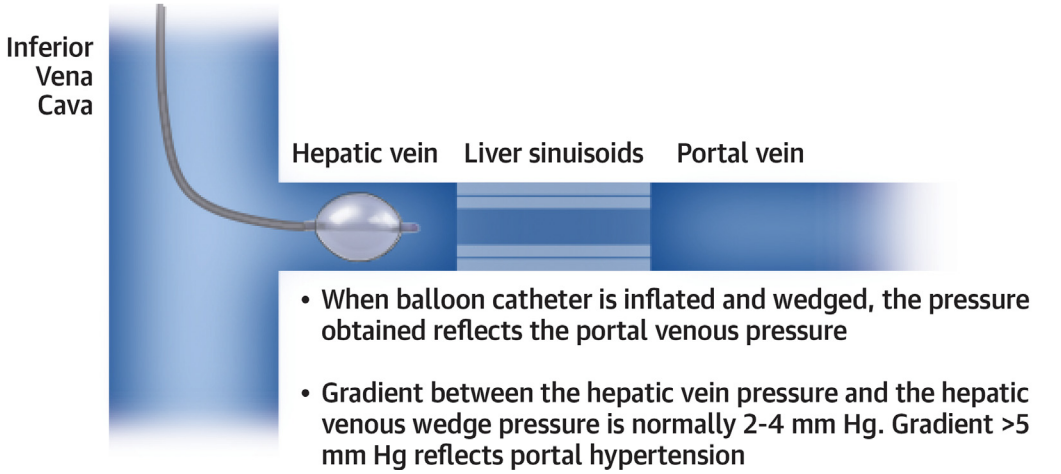
Late RV failure is increasingly recognized as a cause of morbidity and mortality in patients with LVADs.[79] It is unclear whether the cause is suboptimal LV unloading and subsequent PH, or long-term progression of the myopathic process. Early signs and symptoms of RV failure include fatigue, early satiety, lower extremity edema, arrhythmias, and hepatic and renal dysfunction. These findings may be difficult to identify early, particularly in elderly patients with comorbidities. A high index of suspicion is required, and RHC can lead to earlier recognition. RHC is also indicated when patients are being assessed for myocardial recovery (Table 4).

HEART TRANSPLANTATION. The United States heart transplantation allocation policy from 2018 requires hemodynamic data for risk stratification and prioritization on the waitlist. Inotropic support is indicated for patients with PCWP >15 mm Hg and cardiac index <2.2 L/min/m^2. Escalation to mechanical circulatory support is indicated when cardiac index is low (<1.8 L/min/m^2 if no inotropic support, <2.0 L/min/m^2 with inotropic support) along with SBP <90 mm Hg.[80] The intent of incorporating hemodynamic data for prioritization was to use objective criteria demonstrating cardiogenic shock to ensure that donor hearts were prioritized to the "sickest" patients. Unfortunately, given the shortage of organs, the use of exception requests and other clinical practices have led to patients obtaining higher prioritization on the waitlist independently of hemodynamic criteria.[81] Evaluation of the allocation system is under way to realign its goals more closely with clinical practice.

RHC is necessary before heart transplantation to evaluate for severe pulmonary vascular disease.[82] Owing to the risk of donor RV failure after transplantation, elevated PVR >5 WUs or transpulmonary gradient (TPG) >16 mm Hg is considered at least a relative contraindication to transplantation. Vasodilator challenge is recommended for patients with PA systolic pressure >50 mm Hg and either a TPG ≥15 mm Hg or a PVR ≥3 WUs.[83] Common vasodilators used include inhaled nitric oxide, prostacyclin, nitroprusside, milrinone, and nitroglycerin. The selection of a specific agent may depend on institutional preference and the specific hemodynamic profiles of the patient. For example, nitroprusside is ideal if a patient has a high SVR but may cause symptomatic hypotension if baseline MAP is borderline. A decrease in the PVR to <3 WUs with SBP >85 mm Hg constitutes a favorable response associated with low risk for RV failure postoperatively. When an acute vasodilator challenge is unsuccessful, hospitalization with continuous monitoring of hemodynamic status is recommended. A reduction in PVR can often be documented in 24 to 48 hours after treatment with inotropic agents, diuretics, and vasoactive agents.[83] Temporary mechanical circulatory support can also be considered. However, if PH remains severe and is irreversible, options such as

FIGURE 6 Assessment of Portal Hypertension

Hepatic vein pressure (mm Hg)	Hepatic vein wedge pressure (mm Hg)	Hepatic venous pressure gradient (mm Hg)	Clinical condition
6	8	2	Normal
6	18	12	Portal hypertension, no venous congestion
18	22	4	Venous congestion, no portal hypertension
18	28	10	Portal hypertension and venous congestion



Schematic depiction showing measurement of hepatic vein wedge pressure, which provides an estimate of portal venous pressure, and clinical correlation of obtained measurements.

durable LVAD or combined heart/lung transplantation may be considered. Long-term LVAD support may reduce PVR to allow for subsequent heart transplantation.[84]

PORTAL HYPERTENSION. Patients with HF may present with abnormal liver function tests and abnormal hepatic radiographic findings, especially those with severe systemic venous congestion. It is critical to determine whether liver abnormalities are related to intrinsic portal hypertension or to systemic venous congestion, because portal hypertension from liver disease could disqualify patients from LVAD or heart transplantation.[85]

The most common approach to assessing portal vein pressure requires measurement of hepatic vein wedge pressure.[86] A normal pressure gradient between the hepatic vein and the hepatic vein wedge (ie, portal pressure) is defined as 1 to 5 mm Hg; portal hypertension is suspected when the gradient is >5 mm Hg (Figure 6). In patients with elevated hepatic vein pressure with a normal pressure gradient, liver abnormalities are likely related to HF.[87]

HIGH-OUTPUT HF. High-output HF is an uncommon clinical syndrome and is defined as signs and symptoms of HF with cardiac index >4.0 L/min/m^2.[88] Leading causes include obesity, liver disease, and arteriovenous fistulas. The creation of a fistula increases venous return and preload, reduces SVR, and increases CO, which over time may lead to high-output HF, RV failure, and PH.[89] One approach to fistula evaluation involves measurement of baseline hemodynamic status, and then repeated measurement of hemodynamic status after 1 minute of fistula compression, which has been shown to be safe.[90] Whether this test is predictive of long-term improvement in hemodynamic status if the arteriovenous fistula is modified is unknown. Takedown or

surgical modification in some patients can lead to cardiac reverse remodeling or improvement in HF severity.[91]

CONCLUSIONS

Despite improvement in medical therapy, HF patients remain at risk for decompensation and hospitalization. Clinicians must have a detailed understanding of hemodynamics to treat patients with HF successfully, avoid end-organ damage, and potentially improve cardiovascular outcomes. Emerging data support the value of hemodynamic assessment in the full spectrum of HF, ranging from cardiogenic shock to exercise testing in patients with suspected HFpEF. Understanding the role of invasive hemodynamics is critical for clinicians to optimize the treatment of patients with HF.

FUNDING SUPPORT AND AUTHOR DISCLOSURES

Dr Rajagopalan has served as a consultant for Abbott Laboratories. Dr Borlaug is supported by R01 HL128526, R01 HL162828, and U01 HL160226 from the National Heart, Lung, and Blood Institute and W81XWH2210245 from the United States Department of Defense, as well as research grant funding from AstraZeneca, Axon, Medtronic, Novo Nordisk, and Tenax Therapeutics, and has served as a consultant for Amgen, Aria, BD, Boehringer Ingelheim, Cytokinetics, Edwards Lifesciences, Eli Lilly, Merck, Novo Nordisk, NGM, NXT, and VADovations, and is named inventor (US Patent number 10,307,179) for the tools and approach for a minimally invasive pericardial modification procedure to treat heart failure. Dr Bailey has served as a consultant for OptumRx and Nordisk. Dr Eckman has served as a consultant for Daxor. Dr Hall has served as a consultant for Abbott Laboratories, Abiomed, Evaheart, CareDx, and Natera. Dr Ramani has served as a consultant for Merck and has received research grant funding from United Therapeutics and Janssen. Dr Khazanie has research grant support from the National Institutes of Health (K23 HL145122) and the University of Colorado's Ludeman Family Center for Women's Health Research. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

ADDRESS FOR CORRESPONDENCE: Dr Navin Rajagopalan, Division of Cardiology, University of Kentucky, 900 South Limestone Street, Lexington, Kentucky 40536, USA. E-mail: nra224@uky.edu. X handle: @nrajag.

REFERENCES

1. Beigel R, Cercek B, Siegel RJ, Hamilton MA. Echo-Doppler hemodynamics: an important management tool for today's heart failure care. *Circulation*. 2015;131:1031–1034.

2. Heidenreich PA, Bozkurt B, Aguilar D, et al. 2022 AHA/ACC/HFSA guideline for the management of heart failure: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. *J Am Coll Cardiol*. 2022;79:e263–e421.

3. Abudiab MM, Redfield MM, Melenovsky V, et al. Cardiac output response to exercise in relation to metabolic demand in heart failure with preserved ejection fraction. *Eur J Heart Fail*. 2013;15:776–785.

4. Verbrugge FH, Guazzi M, Testani JM, Borlaug BA. Altered hemodynamics and end-organ damage in heart failure: impact on the lung and kidney. *Circulation*. 2020;142:998–1012.

5. Eisman AS, Shah RV, Dhakal BP, et al. Pulmonary capillary wedge pressure patterns during exercise predict exercise capacity and incident heart failure. *Circ Heart Fail*. 2018;11:e004750.

6. Omote K, Verbrugge FH, Sorimachi H, et al. Central haemodynamic abnormalities and outcome in patients with unexplained dyspnoea. *Eur J Heart Fail*. 2023;25:185–196.

7. Drazner MH, Stevenson LW. Relief and prevention of congestion in heart failure enhance quality and length of life. *Circulation*. 2019;140:1380–1382.

8. Berthelot E, Bauer F, Eicher JC, et al. Pulmonary hypertension in chronic heart failure: definitions, advances, and unanswered issues. *ESC Heart Fail*. 2018;5:755–763.

9. Guazzi M, Borlaug BA. Pulmonary hypertension due to left heart disease. *Circulation*. 2012;126:975–990.

10. Baudry G, Coutance G, Dorent R, et al. Prognosis value of Forrester's classification in advanced heart failure patients awaiting heart transplantation. *ESC Heart Fail*. 2022;9:3287–3297.

11. Nohria A, Tsang SW, Fang JC, et al. Clinical assessment identifies hemodynamic profiles that predict outcomes in patients admitted with heart failure. *J Am Coll Cardiol*. 2003;41:1797–1804.

12. Thibodeau JT, Drazner MH. The role of the clinical examination in patients with heart failure. *J Am Coll Cardiol HF*. 2018;6:543–551.

13. Pham DD, Drazner MH, Ayers CR, et al. Identifying discordance of right- and left-ventricular filling pressures in patients with heart failure by the clinical examination. *Circ Heart Fail*. 2021;14:e008779.

14. Adamo L, Nassif ME, Novak E, LaRue SJ, Mann DL. Prevalence of lactic acidaemia in patients with advanced heart failure and depressed cardiac output. *Eur J Heart Fail*. 2017;19:1027–1033.

15. Guglin M, Zucker MJ, Borlaug BA, et al. Evaluation for heart transplantation and LVAD implantation: JACC council perspectives. *J Am Coll Cardiol*. 2020;75:1471–1487.

16. Borlaug BA, Sharma K, Shah SJ, Ho JE. Heart failure with preserved ejection fraction: JACC scientific statement. *J Am Coll Cardiol*. 2023;81:1810–1834.

17. Obokata M, Reddy YNV, Melenovsky V, Sorimachi H, Jarolim P, Borlaug BA. Uncoupling between intravascular and distending pressures leads to underestimation of circulatory congestion in obesity. *Eur J Heart Fail*. 2022;24:353–361.

18. D'Alto M, Dimopoulos K, Coghlan JG, Kovacs G, Rosenkranz S, Naeije R. Right heart catheterization for the diagnosis of pulmonary hypertension: controversies and practical issues. *Heart Fail Clin*. 2018;14:467–477.

19. Hsu S, Fang JC, Borlaug BA. Hemodynamics for the heart failure clinician: a state-of-the-art review. *J Card Fail*. 2022;28:133–148.

20. Grinstein J, Houston BA, Nguyen AB, et al. Standardization of the right heart catheterization and the emerging role of advanced hemodynamics in heart failure. *J Card Fail*. 2023;29:1543–1555.

21. Grafton G, Cascino TM, Perry D, Ashur C, Koelling TM. Resting oxygen consumption and heart failure: importance of measurement for determination of cardiac output with the use of the Fick principle. *J Card Fail*. 2020;26:664–672.

22. Sternberg ME, Nicolazzi J, Patel MR, et al. Thermodilution assessment of cardiac index in patients with tricuspid regurgitation. *J Invasive Cardiol*. 2023;35:E122–E125.

23. Jansen JR. The thermodilution method for the clinical assessment of cardiac output. *Intensive Care Med*. 1995;21:691–697.

24. Geske JB, Anavekar NS, Nishimura RA, Oh JK, Gersh BJ. Differentiation of constriction and restriction: complex cardiovascular hemodynamics. *J Am Coll Cardiol*. 2016;68:2329–2347.

25. Nadir AM, Beadle R, Lim HS. Kussmaul physiology in patients with heart failure. *Circ Heart Fail*. 2014;7:440–447.

26. Stone GW, Lindenfeld J, Abraham WT, et al. Transcatheter mitral-valve repair in patients with heart failure. *N Engl J Med*. 2018;379:2307–2318.

27. Viray MC, Bonno EL, Gabrielle ND, et al. Role of pulmonary artery wedge pressure saturation during right heart catheterization: a prospective study. *Circ Heart Fail*. 2020;13:e007981.

28. Ginsberg F, Parrillo JE. Cardiogenic shock: a historical perspective. *Crit Care Clin*. 2009;25:103–114, viii.

29. Baran DA, Grines CL, Bailey S, et al. SCAI clinical expert consensus statement on the classification of cardiogenic shock: this document was endorsed by the American College of Cardiology (ACC), the American Heart Association (AHA), the Society of Critical Care Medicine (SCCM), and the Society of Thoracic Surgeons (STS) in April 2019. *Catheter Cardiovasc Interv*. 2019;94:29–37.

30. Jentzer JC, Burstein B, Van Diepen S, et al. Defining shock and preshock for mortality risk stratification in cardiac intensive care unit patients. *Circ Heart Fail*. 2021;14:e007678.

31. Naidu SS, Baran DA, Jentzer JC, et al. SCAI SHOCK stage classification expert consensus update: a review and incorporation of validation studies. *J Am Coll Cardiol*. 2022;79:933–946.

32. Garan AR, Kanwar M, Thayer KL, et al. Complete hemodynamic profiling with pulmonary artery catheters in cardiogenic shock is associated with lower in-hospital mortality. *J Am Coll Cardiol HF*. 2020;8:903–913.

33. Kadosh BS, Berg DD, Bohula EA, et al. Pulmonary artery catheter use and mortality in the cardiac intensive care unit. *J Am Coll Cardiol HF*. 2023;11:903–914.

34. Narang N, Blumer V, Jumean MF, et al. Management of heart failure-related cardiogenic shock: practical guidance for clinicians. *J Am Coll Cardiol HF*. 2023;11:845–851.

35. Narang N, Dela Cruz M, Imamura T, et al. Discordance between lactic acidemia and hemodynamics in patients with advanced heart failure. *Clin Cardiol*. 2021;44:636–645.

36. Lim HS. Cardiac power output revisited. *Circ Heart Fail*. 2020;13:e007393.

37. Baldetti L, Pagnesi M, Gallone G, et al. Prognostic value of right atrial pressure-corrected cardiac power index in cardiogenic shock. *ESC Heart Fail*. 2022;9:3920–3930.

38. Thayer KL, Zweck E, Ayouty M, et al. Invasive hemodynamic assessment and classification of in-hospital mortality risk among patients with cardiogenic shock. *Circ Heart Fail*. 2020;13:e007099.

39. Fincke R, Hochman JS, Lowe AM, et al. Cardiac power is the strongest hemodynamic correlate of mortality in cardiogenic shock: a report from the SHOCK trial registry. *J Am Coll Cardiol*. 2004;44:340–348.

40. Saxena A, Garan AR, Kapur NK, et al. Value of hemodynamic monitoring in patients with cardiogenic shock undergoing mechanical circulatory support. *Circulation*. 2020;141:1184–1197.

41. Alogna A, Omar M, Popovic D, et al. Biventricular cardiac power reserve in heart failure with preserved ejection fraction. *Eur J Heart Fail*. 2023;25:956–966.

42. Abraham J, Blumer V, Burkhoff D, et al. Heart failure-related cardiogenic shock: pathophysiology, evaluation and management considerations: review of heart failure-related cardiogenic shock. *J Card Fail*. 2021;27:1126–1140.

43. Jain P, Thayer KL, Abraham J, et al. Right ventricular dysfunction is common and identifies patients at risk of dying in cardiogenic shock. *J Card Fail*. 2021;27:1061–1072.

44. Kanwar MK, Everett KD, Gulati G, Brener MI, Kapur NK. Epidemiology and management of right ventricular-predominant heart failure and shock in the cardiac intensive care unit. *Eur Heart J Acute Cardiovasc Care*. 2022;11:584–594.

45. Randhawa VK, Al-Fares A, Tong MZY, et al. A pragmatic approach to weaning temporary mechanical circulatory support: a state-of-the-art review. *J Am Coll Cardiol HF*. 2021;9:664–673.

46. Papolos AI, Kenigsberg BB, Berg DD, et al. Management and outcomes of cardiogenic shock in cardiac ICUs with versus without shock teams. *J Am Coll Cardiol*. 2021;78:1309–1317.

47. Rangaswami J, Bhalla V, Blair JEA, et al. Cardiorenal syndrome: classification, pathophysiology, diagnosis, and treatment strategies: a scientific statement from the American Heart Association. *Circulation*. 2019;139:e840–e878.

48. Biegus J, Borlaug BA, Testani JM. Congestion and decongestion assessment in heart failure: pressure, volume, or both? *J Am Coll Cardiol HF*. 2023;11:1152–1156.

49. Martyn T, Faulkenberg KD, Albert CL, et al. Acute hemodynamic effects of sacubitril-valsartan in heart failure patients receiving intravenous vasodilator and inotropic therapy. *J Card Fail*. 2021;27:368–372.

50. Stevenson LW, Dracup KA, Tillisch JH. Efficacy of medical therapy tailored for severe congestive heart failure in patients transferred for urgent cardiac transplantation. *Am J Cardiol*. 1989;63:461–464.

51. Morris AA, Khazanie P, Drazner MH, et al. Guidance for timely and appropriate referral of patients with advanced heart failure: a scientific statement from the American Heart Association. *Circulation*. 2021;144:e238–e250.

52. Gustafsson F, Damman K, Nalbantgil S, et al. Inotropic therapy in patients with advanced heart failure: a clinical consensus statement from the Heart Failure Association of the European Society of Cardiology. *Eur J Heart Fail*. 2023;25:457–468.

53. Nizamic T, Murad MH, Allen LA, et al. Ambulatory inotrope infusions in advanced heart failure: a systematic review and meta-analysis. *J Am Coll Cardiol HF*. 2018;6:757–767.

54. Bozkurt B, Ahmad T, Alexander KM, et al. Heart failure epidemiology and outcomes statistics: a report of the Heart Failure Society of America. *J Card Fail*. 2023;29:1412–1451.

55. Verbrugge FH, Omote K, Reddy YNV, Sorimachi H, Obokata M, Borlaug BA. Heart failure with preserved ejection fraction in patients with normal natriuretic peptide levels is associated with increased morbidity and mortality. *Eur Heart J*. 2022;43:1941–1951.

56. Pieske B, Tschope C, de Boer RA, et al. How to diagnose heart failure with preserved ejection fraction: the HFA-PEFF diagnostic algorithm: a consensus recommendation from the Heart Failure Association (HFA) of the European Society of Cardiology (ESC). *Eur Heart J*. 2019;40:3297–3317.

57. Mizumi S, Goda A, Takeuchi K, et al. Effects of body position during cardiopulmonary exercise testing with right heart catheterization. *Physiol Rep*. 2018;6:e13945.

58. Baratto C, Caravita S, Soranna D, et al. Current limitations of invasive exercise hemodynamics for the diagnosis of heart failure with preserved ejection fraction. *Circ Heart Fail*. 2021;14:e007555.

59. Reddy YNV, Obokata M, Wiley B, et al. The haemodynamic basis of lung congestion during exercise in heart failure with preserved ejection fraction. *Eur Heart J*. 2019;40:3721–3730.

60. Maron BA, Kovacs G, Vaidya A, et al. Cardiopulmonary hemodynamics in pulmonary hypertension and heart failure: JACC review topic of the week. *J Am Coll Cardiol*. 2020;76:2671–2681.

61. Vanderpool RR, Saul M, Nouraie M, Gladwin MT, Simon MA. Association between hemodynamic markers of pulmonary hypertension and outcomes in heart failure with preserved ejection fraction. *JAMA Cardiol*. 2018;3:298–306.

62. Maron BA. Revised definition of pulmonary hypertension and approach to management: a clinical primer. *J Am Heart Assoc*. 2023;12:e029024.

63. Omote K, Sorimachi H, Obokata M, et al. Pulmonary vascular disease in pulmonary hypertension due to left heart disease: pathophysiologic implications. *Eur Heart J*. 2022;43:3417–3431.

64. Tampakakis E, Leary PJ, Selby VN, et al. The diastolic pulmonary gradient does not predict survival in patients with pulmonary hypertension due to left heart disease. *J Am Coll Cardiol HF*. 2015;3:9–16.

65. Forbes LM, Bull TM, Lahm T, Make BJ, Cornwell WK 3rd. Exercise testing in the risk assessment of pulmonary hypertension. *Chest*. 2023;164:736–746.

66. Saeed D, Feldman D, Banayosy AE, et al. The 2023 International Society for Heart and Lung Transplantation guidelines for mechanical circulatory support: a 10- year update. *J Heart Lung Transplant*. 2023;42:e1–e222.

67. Bellavia D, Iacovoni A, Scardulla C, et al. Prediction of right ventricular failure after ventricular assist device implant: systematic review and meta-analysis of observational studies. *Eur J Heart Fail*. 2017;19:926–946.

68. Kapur NK, Esposito ML, Bader Y, et al. Mechanical circulatory support devices for acute right ventricular failure. *Circulation*. 2017;136:314–326.

69. Soliman OII, Akin S, Muslem R, et al. Derivation and validation of a novel right-sided heart failure model after implantation of continuous flow left ventricular assist devices: the EUROMACS (European registry for patients with mechanical circulatory support) right-sided heart failure risk score. *Circulation*. 2018;137:891–906.

70. Ali HR, Kiernan MS, Choudhary G, et al. Right ventricular failure post-implantation of left ventricular assist device: prevalence, pathophysiology, and predictors. *ASAIO J*. 2020;66:610–619.

71. Gulati G, Grandin EW, DeNofrio D, Upshaw JN, Vest AR, Kiernan MS. Association between postoperative hemodynamic metrics of pulmonary hypertension and right ventricular dysfunction and clinical outcomes after left ventricular assist device implantation. *J Heart Lung Transplant*. 2022;41:1459–1469.

72. Takeda K, Naka Y, Yang JA, et al. Outcome of unplanned right ventricular assist device support for severe right heart failure after implantable left ventricular assist device insertion. *J Heart Lung Transplant*. 2014;33:141–148.

73. Han JJ, Acker MA, Atluri P. Left ventricular assist devices. *Circulation*. 2018;138:2841–2851.

74. Sailer C, Edelmann H, Buchanan C, et al. Impairments in blood pressure regulation and cardiac baroreceptor sensitivity among patients with heart failure supported with continuous-flow left ventricular assist devices. *Circ Heart Fail*. 2021;14:e007448.

75. Tran T, Muralidhar A, Hunter K, et al. Right ventricular function and cardiopulmonary performance among patients with heart failure supported by durable mechanical circulatory support devices. *J Heart Lung Transplant*. 2021;40:128–137.

76. Uriel N, Sayer G, Addetia K, et al. Hemodynamic ramp tests in patients with left ventricular assist devices. *J Am Coll Cardiol HF*. 2016;4:208–217.

77. Imamura T, Jeevanandam V, Kim G, et al. Optimal hemodynamics during left ventricular assist device support are associated with reduced readmission rates. *Circ Heart Fail*. 2019;12:e005094.

78. Uriel N, Burkhoff D, Rich JD, et al. Impact of hemodynamic ramp test-guided HVAD speed and medication adjustments on clinical outcomes. *Circ Heart Fail*. 2019;12:e006067.

79. Rajapreyar I, Soliman O, Brailovksy Y, et al. Late right heart failure after left ventricular assist device implantation: contemporary insights and future perspectives. *J Am Coll Cardiol HF*. 2023;11:865–878.

80. Shore S, Golbus JR, Aaronson KD, Nallamothu BK. Changes in the United States adult heart allocation policy: challenges and opportunities. *Circ Cardiovasc Qual Outcomes*. 2020;13:e005795.

81. Topkara VK, Clerkin KJ, Fried JA, et al. Exception status listing in the new adult heart allocation system: a new solution to an old problem? *Circ Heart Fail*. 2021;14:e007916.

82. Rivinius R, Helmschrott M, Ruhparwar A, et al. Elevated pre-transplant pulmonary vascular resistance is associated with early post-transplant atrial fibrillation and mortality. *ESC Heart Fail*. 2020;7:176–187.

83. Velleca A, Shullo MA, Dhital K, et al. The International Society for Heart and Lung Transplantation (ISHLT) guidelines for the care of heart transplant recipients. *J Heart Lung Transplant*. 2023;42:e1–e141.

84. Gulati G, Ruthazer R, Denofrio D, Vest AR, Kent D, Kiernan MS. Understanding longitudinal changes in pulmonary vascular resistance after left ventricular assist device implantation. *J Card Fail*. 2021;27:552–559.

85. Goyal A, Dalia T, Ranka S, et al. Impact of biopsy proven liver fibrosis on patients undergoing evaluation and treatment for advanced heart failure surgical therapies. *Am J Cardiol*. 2023;194:46–55.

86. Lu Q, Leong S, Lee KA, et al. Hepatic venous-portal gradient (HVPG) measurement: pearls and pitfalls. *Br J Radiol*. 2021;94:20210061.

87. Suk KT, Kim DJ. Staging of liver fibrosis or cirrhosis: the role of hepatic venous pressure gradient measurement. *World J Hepatol*. 2015;7:607–615.

88. Reddy YNV, Melenovsky V, Redfield MM, Nishimura RA, Borlaug BA. High-output heart failure: a 15-year experience. *J Am Coll Cardiol*. 2016;68:473–482.

89. Reddy YNV, Obokata M, Dean PG, Melenovsky V, Nath KA, Borlaug BA. Long-term cardiovascular changes following creation of arteriovenous fistula in patients with end stage renal disease. *Eur Heart J*. 2017;38:1913–1923.

90. Aitken E, Kerr D, Geddes C, Berry C, Kingsmore D. Cardiovascular changes occurring with occlusion of a mature arteriovenous fistula. *J Vasc Access*. 2015;16:459–466.

91. Rao NN, Stokes MB, Rajwani A, et al. Effects of arteriovenous fistula ligation on cardiac structure and function in kidney transplant recipients. *Circulation*. 2019;139:2809–2818.

92. Morine KJ, Kiernan MS, Pham DT, Paruchuri V, Denofrio D, Kapur NK. Pulmonary artery pulsatility index is associated with right ventricular failure after left ventricular assist device surgery. *J Card Fail*. 2016;22:110–116.

KEY WORDS heart failure, hemodynamics, right heart catheterization