JACC: CASE REPORTS VOL. 21, 2023

CASE REPORT

INTERMEDIATE

EDUCATIONAL CLINICAL CASE SERIES

The Value of Right Heart Catheterization

Case Series Showing Benefits in a Variety of Diagnoses

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ABSTRACT

Although the right heart catheterization (RHC) was first introduced in 1945, its use in the quantitative hemodynamic assessment of patients has remained of questionable benefit. With recent advances in pharmacotherapies and mechanical support devices, RHC has been increasingly used to assess and help tailor the management of more complex patient scenarios. We present a case series in which the use of the RHC was helpful in making complex medical decisions. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2023;21:101959) © 2023 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Right heart catheterization (RHC) is an invasive procedure that allows for the direct measurement of intracardiac pressures and cardiac output in addition to the indirect measurement of pulmonary and systemic vascular resistance. Although the routine use of RHC has declined since its first introduction, it can provide invaluable data for tailoring therapy in a variety of cardiac conditions highlighted in this series of cases where it altered medical decision making.[1,2]

LEARNING OBJECTIVES

- To review the foundations of RHC including components of a normal pressure waveform, major procedural risks, and key indications.
- To summarize the evolving utilization of RHC and highlight its current importance with modern pharmacotherapy and advanced therapies.
- To illustrate three specific situations where information from a RHC directly impacted medical decision making.

PATIENT 1

A 51-year-old female presented with worsening cough and dyspnea and new-onset lower extremity edema for the past 7 days. She first noted dyspnea and a cough 1 year before this admission and was diagnosed with asthma; pulmonary function testing was not performed. Two months before the current admission she had been admitted for shortness of breath and treated for multifocal pneumonia demonstrated on a chest computed tomography scan. Her symptoms did not improve, prompting the present admission. Her review of systems was significant for 2 years of polyarthralgias of the hands, shoulders, knees, and ankles and dysphagia to thin liquids associated with a 10-pound weight loss. On examination, she was febrile to 104.7 °F, heart rate was 118 beats/min, and she was hypoxic to 88% on room air. She had elevated jugular venous pressure, diffuse rhonchi,

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John W. Hirshfeld Jr, MD, served as Guest Editor-in-Chief for this paper.

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 April 11, 2023; accepted July 6, 2023.

ISSN 2666-0849 https://doi.org/10.1016/j.jaccas.2023.101959

ABBREVIATIONS AND ACRONYMS

EDP = end-diastolic pressure

LV = left ventricle

PA = pulmonary artery

PCWP = pulmonary capillary wedge pressure

PVR = pulmonary vascular resistance

RA = right atrium

RAP = right atrial pressure

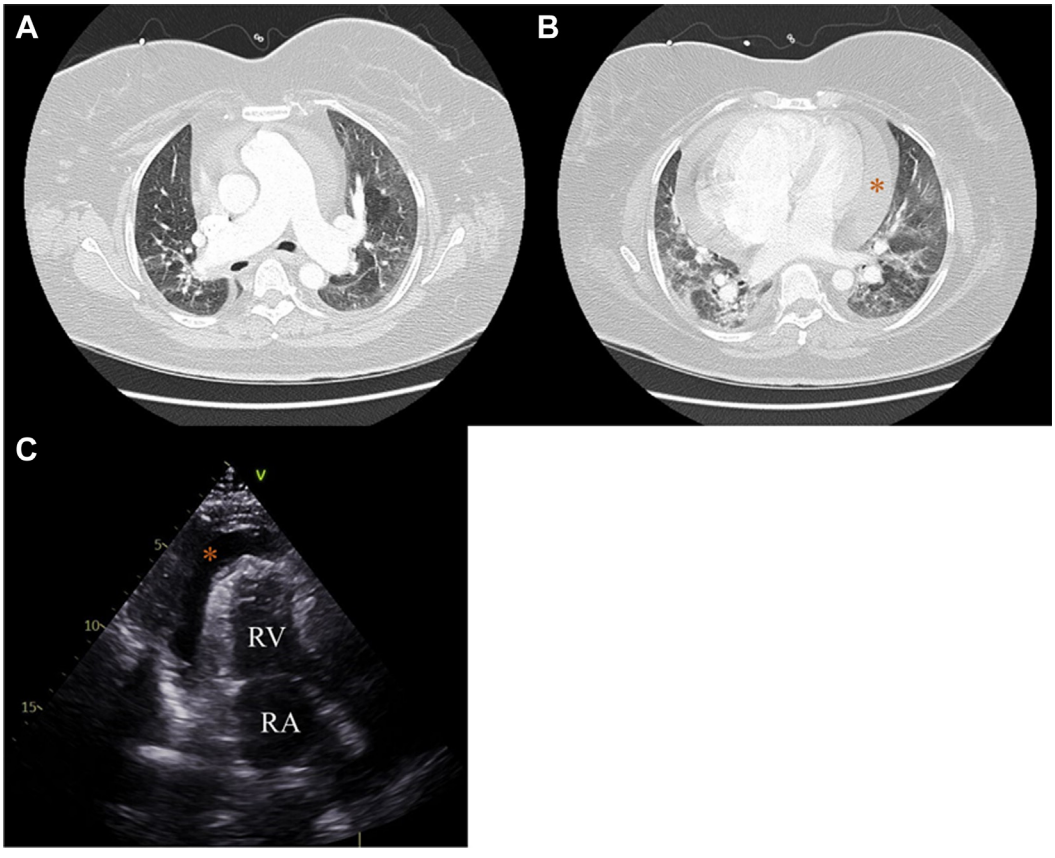
RV = right ventricle

and 2+ lower extremity edema. Laboratory abnormalities included an elevated high-sensitivity troponin level that peaked at 138.0 ng/L (normal: ≤18 ng/L), D-dimer 1.38 μg/mL (normal: <0.49 μg/mL), N-terminal pro-B-type natriuretic peptide (NT-proBNP) 267 pg/mL (normal: ≤125 pg/mL), and C-reactive protein 8.00 mg/dL (normal: <0.8 mg/L). A chest computed tomography scan revealed a moderate-to-large pericardial effusion, bilateral ground glass opacities, and a prominent main pulmonary artery (PA) (Figure 1). In addition to the pericardial effusion, a transthoracic echocardiogram (TTE) showed hyperdynamic left ventricular (LV) systolic function (with an ejection fraction of 75%), dilated right ventricle (RV) with decreased function, severe pulmonary hypertension with an estimated PA systolic pressure of 66 mm Hg, and brief late diastolic collapse of the right atrium (RA). In the setting of tamponade physiology, a pericardiocentesis was performed with placement of an indwelling pericardial catheter.

Further evaluation to identify a unifying diagnosis included negative blood, sputum, and urine cultures. Autoimmune evaluation was significant for positive antibodies to Ro (also known as Sjogren's syndrome-associated antibody) and histidyl tRNA synthetase (anti-Jo-1).

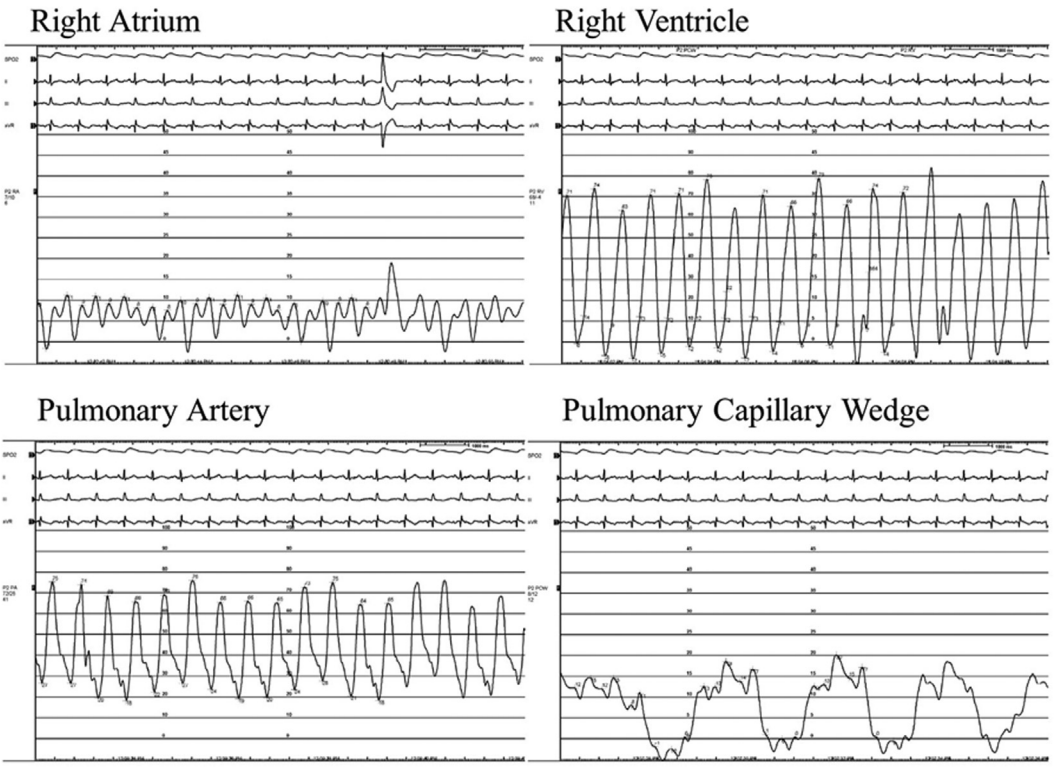
Given her ongoing hypoxia after pericardiocentesis and imaging findings with signs of pulmonary hypertension, a RHC on 4 L nasal canula was performed which revealed a right atrial pressure (RAP) of 6 mm Hg with notable lack of fall in inspiration, consistent with positive Kussmaul's sign, elevated PA pressure of 72/25 mm Hg (mean, 41 mm Hg), pulmonary capillary wedge pressure (PCWP) with significant respiratory variation with 12 mm Hg measured at end

FIGURE 1 Significant Imaging From Patient 1



Patient 1's imaging showing the enlarged pulmonary artery **(A),** pericardial effusion **(B, C; orange asterisk)**, and diffuse ground class opacities **(B).** RA = right atrium; RV = right ventricle.

FIGURE 2 **Hemodynamic Pressure Waveforms From Patient 1**



Hemodynamic pressure waveforms from the right heart catheterization of patient 1 showing right atrial pressure (RAP) of 6 mm Hg, but with notable lack of fall in inspiration, consistent with positive Kussmaul's sign, and elevated pulmonary artery pressure of 72/25 mm Hg (mean, 41 mm Hg). Pulmonary capillary wedge pressure showed significant respiratory variation with 12 mm Hg measured at end expiration.

expiration, cardiac index 3.32 L/min/m^2 by estimated Fick method with venous oxygen saturation (SvO_2) 72.7%, pulmonary vascular resistance (PVR) 4.8 WU (normal: ≤1.5 WU), and systemic vascular resistance of 2,091 dyn/s/cm^{-5} (reference range, 800 to 1,200 dyn/s/cm^{-5}) (Figure 2, Table 1). Based on these findings, and with her positive Sjogren's syndrome-associated antibody and anti-Jo-1, her hypoxia was thought to be from WHO group I pulmonary hypertension due to antisynthetase syndrome and possible Sjogren's syndrome. In consultation with the department of rheumatology, she was started on a solumedrol taper, rituximab, and hydroxychloroquine.

RHC is essential in the diagnostic workup of pulmonary hypertension and to rule out WHO group II disease due to left-sided heart disease. Furthermore, RHC is important for accurate risk stratification of patients with pulmonary arterial hypertension. Several features on invasive hemodynamics are associated with an increased 1-year mortality, including RAP >14 mm Hg, PVR >5 WU, SvO_2 <60%, and cardiac index <2 L/min/m^2, none of which were present in this patient.[3]

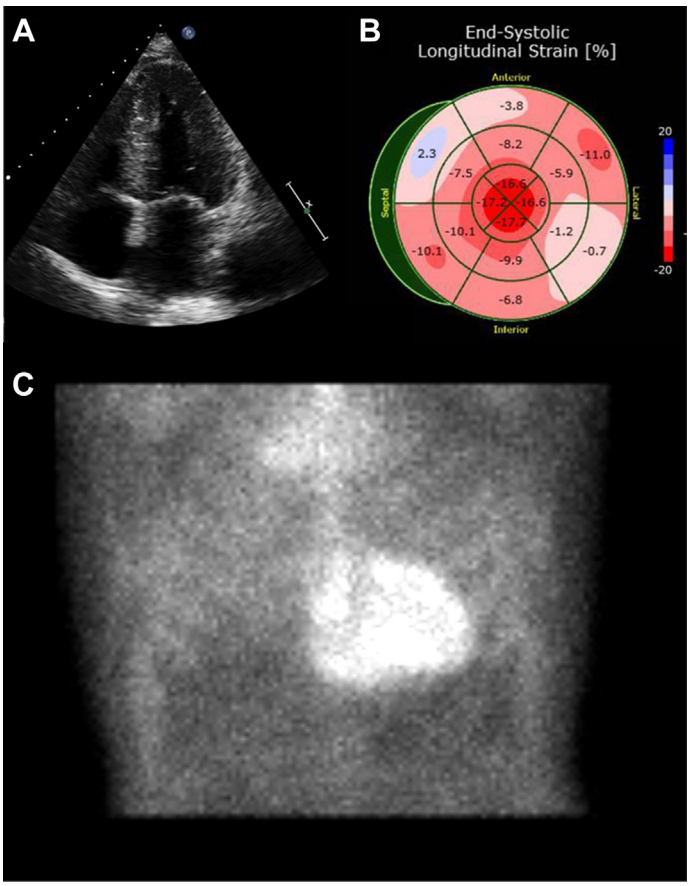
TABLE 1 **RHC Hemodynamics**

	Patient 1	Patient 2	Patient 3[a]	Patient 3[b]
RAP, mm Hg	6	12	23	13
RVP, mm Hg	68/11	60/16	50/25	37/13
PAP, mm Hg	72/25 (41)	60/27 (39)	40/33 (35)	37/15 (23)
PCWP, mm Hg	12	27	25	8
Cardiac index (Fick), L/min/m^2	3.32	1.29	2.01	2.15
PVR, WU	4.8	5.2	2.0	3.2
PAPI	7.8	2.75	0.3	1.7
Systemic vascular resistance, dyn/s/cm^{-5}	2,091	3,278	875	1,010

[a]On milrinone 0.25 μg/kg/min. [b]Left ventricular assist device speed 5,500 RPM.

PAPI = pulmonary artery pulsatility index; PCWP = pulmonary capillary wedge pressure; PVR = pulmonary vascular resistance; RAP = right atrial pressure; RHC = right heart catheterization; RVP = right venous pressure.

FIGURE 3 Significant Imaging for Patient 2



Transthoracic echocardiogram in the apical 4-chamber view with increased wall thickness and nondilated left ventricle **(A)** and strain pattern **(B)** with preserved apical segments. Nuclear scan **(C)** with diffuse, 3+ uptake of technetium-99m hydroxymethylene diphosphonate.

In this case, RHC confirmed the diagnosis of pulmonary hypertension, ruled out left-sided heart disease as the underlying etiology, and provided accurate risk stratification of pulmonary arterial hypertension. With this information, treatment was targeted to her antisynthetase syndrome with significant improvement in clinical status and her oxygen saturation to 98% on room air.

PATIENT 2

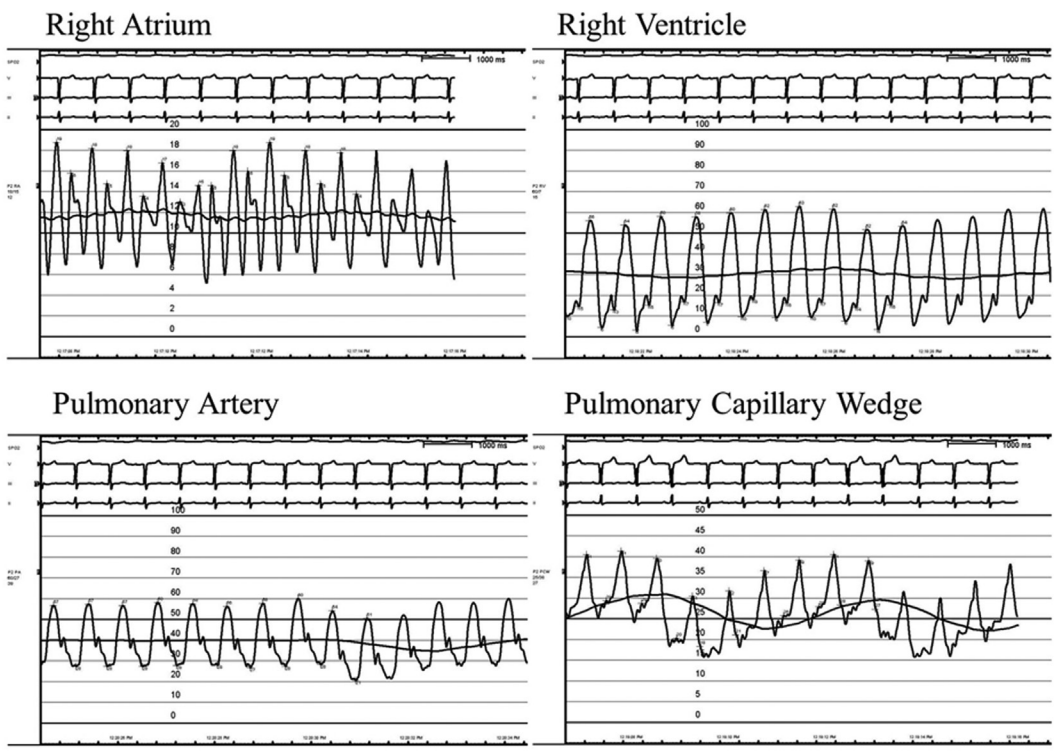
A 65-year-old male with a past medical history of well-controlled asthma and bilateral carpal tunnel syndrome was referred for evaluation of a recently diagnosed cardiomyopathy.

Approximately 10 months before presentation he was noted to have profound fatigue and dyspnea with routine exercise. His vitals were within normal limits. On physical examination, he had no murmur, rub, or gallop, significant jugular venous distention, or lower extremity edema. TTE revealed global hypokinesis with reduced systolic function (left ventricular ejection fraction [LVEF] of 31%), severely increased LV wall thickness (intraventricular septum of 1.8 cm, normal: 0.6-1.0 cm; posterior wall thickness 1.8 cm, normal: 0.6-1.0 cm), and moderate left atrial dilation (Figure 3A). Global longitudinal strain was abnormal and regional longitudinal strain was reduced in the basal and mid-LV segments but preserved in the apical segments (Figure 3B). Coronary angiography revealed 3-vessel coronary artery disease. He was referred to cardiothoracic surgery as part of the heart team evaluation. RHC revealed RAP of 12 mm Hg with v waves to 19 mm Hg with steep y decent, a notable lack of fall of RAP with inspiration consistent with positive Kussmauls's sign, PCWP of 27 mm Hg with large v waves to 38 mm Hg, PA pressure 60/27/39 mm Hg, and severely reduced cardiac index of 1.29 L/min/m^2 (Figure 4, Table 1). This pattern of RA waveform with large v waves and steep y decent, positive Kussmaul's sign, and large v waves on PCWP tracing in the absence of significant mitral regurgitation was concerning for severe restrictive cardiomyopathy or constrictive pericarditis.

Although he had severe multivessel coronary artery disease on his cardiac catheterization, his history of bilateral carpal tunnel syndrome, the degree of ventricular hypertrophy along with the strain pattern on TTE, the lack of LV dilation, and restrictive pattern on RHC raised suspicion of a concurrent infiltrative cardiomyopathy. A subsequent nuclear scan revealed diffuse 3+ uptake of technetium-99m hydroxymethylene diphosphonate suggestive of transthyretin (TTR) cardiac amyloidosis (Figure 3C). Serum free kappa, free lambda, and kappa/lambda ratio were within normal limits ruling out light chain amyloidosis. Genetic testing found a substitution of valine to isoleucine at position 142 on the TTR gene confirming the diagnosis of hereditary TTR amyloidosis for which he was started on tafamadis. Based on the data obtained from this RHC, a decision was made to pursue heart transplantation for which he is currently listed.

Invasive hemodynamic assessment is an essential part of the evaluation for patients with advanced heart failure, especially when heart transplantation or left ventricular assist device (LVAD) are being considered. Specifically in patients with cardiac amyloidosis, hemodynamic profiling is very informative for risk stratification in addition to established clinical scores. Reduced cardiac index occurs in approximately 55% of patients with cardiac

FIGURE 4 **Hemodynamic Pressure Waveforms of Patient 2**



Hemodynamic pressure waveforms from the right heart catheterization of patient 2 showing RAP of 12 mm Hg and v waves of 19 mm Hg with steep y decent, and notable lack of fall of RAP with inspiration, consistent with positive Kussmaul's sign, pulmonary artery wedge pressure of 27 mm Hg with large v waves to 38 mm Hg, and pulmonary artery pressure 60/27/39 mm Hg. Abbreviation as in **Figure 2**.

amyloidosis and is the strongest hemodynamic predictor associated with the need for cardiac transplantation/LVAD, and a predictor of increased risk of death, heart failure admissions, and reduced functional capacity in 1 study.[4]

In this case of newly diagnosed coronary artery disease and cardiac amyloidosis, RHC allowed objective quantification of his risk with severely reduced cardiac index, prompting an expedited heart transplantation evaluation.

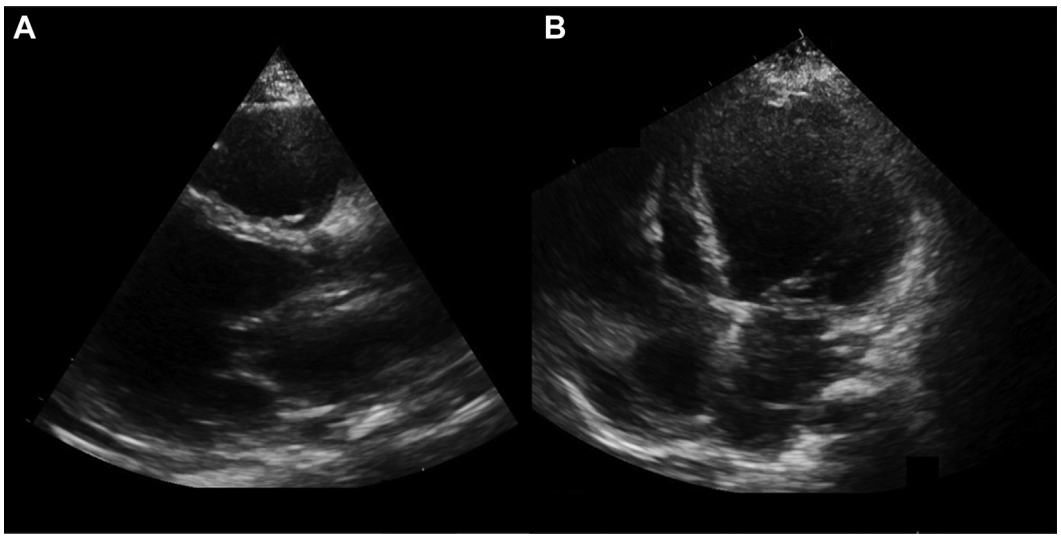
PATIENT 3

A 28-year-old woman with a history of obesity (body mass index 51 kg/m^2), peptic ulcer disease, and obstructive sleep apnea was transferred to our inpatient heart failure unit with concerns for cardiogenic shock. One year before this presentation, she was diagnosed with heart failure with reduced ejection fraction (HFrEF) (LVEF 15%-20%) due to viral myocarditis. Titration of guideline-directed medical therapy was hampered due to gastrointestinal side effects, and she had since been admitted multiple times for acute decompensated HFrEF.

One week before transfer, she presented to an outside hospital for acute decompensated heart failure with cardiogenic shock. Initial diuretic trials were unsuccessful. The initiation of milrinone was complicated by hypotension requiring discontinuation. She was transferred to our institution for further evaluation and treatment of her cardiogenic shock.

Vital signs revealed a heart rate 117 beats/min, blood pressure 107/65 mm Hg, respiratory rate 20 breaths/min with an oxygen saturation of 99% on 2 L nasal cannula. She had prominent jugular venous distention, abdominal distension, and 3+ lower extremity edema. Her labs on admission were significant for an elevated creatinine 1.44 mg/dL (reference range: 0.70-1.40 mg/dL) and NT-pro-BNP 3,130 pg/mL (normal: ≤125 pg/mL). Complete blood cell count, liver function tests, and whole blood lactate were within normal limits. TTE revealed global

FIGURE 5 Transthoracic Echocardiogram of Patient 3



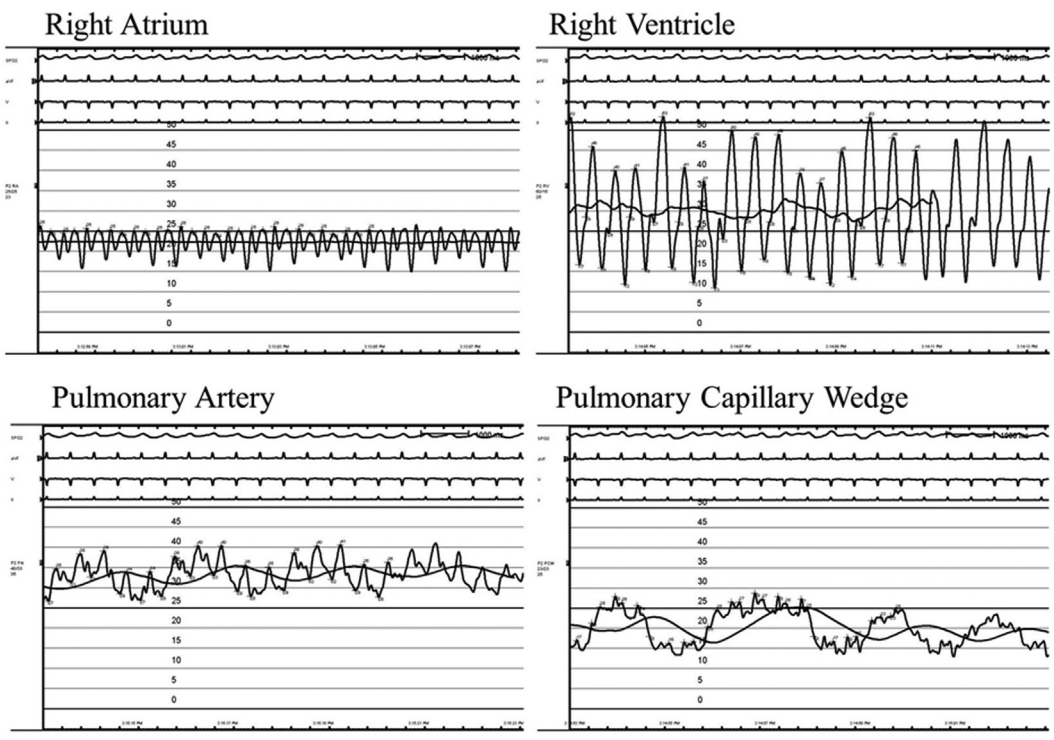
Transthoracic echocardiogram in the **(A)** parasternal long axis and **(B)** apical 4-chamber view showing severe biventricular dilation and dysfunction.

hypokinesis with an LVEF of 15%, severely dilated RA, moderately dilated RV, and moderate tricuspid regurgitation (Figure 5). LV end-diastolic diameter was 7.8 cm (normal: 4.2-5.8 cm). Over the next 2 days, continuous infusions of intravenous furosemide (40 mg/h) and milrinone (0.25 μg/kg/min) were initiated without significant diuresis. RHC revealed RAP of 23 with elevated v waves to 28 mm Hg, intermittent steep y descents, and notable lack of fall of RAP with inspiration consistent with positive Kussmaul's sign, square root pattern of RV waveform, reduced PA pulse pressure, elevated PCWP of 25 mm Hg measured at end expiration, and decreased cardiac index despite milrinone infusion (Figure 6, Table 1). This pattern on RHC waveforms with positive Kussmaul's sign, equalization of RAP and PCWP, and reduced of PA pulsatility index (PA systolic-PA diastolic/RA), raised suspicion for restrictive cardiomyopathy, constrictive pericarditis, or severe biventricular failure. Her milrinone dose was increased and dobutamine was added to help facilitate decongestion. Despite inotropic support, she had progressive lactic acidosis and acute kidney injury.

In the setting of severe obesity, she was deemed not to be a candidate for heart transplantation. Despite dual inotropic support, she required escalation to mechanical circulatory support. Given the severe RV dysfunction on TTE and RHC with severely reduced PA pulsatility index of 0.3 (normal: >2), there was a concern that she would need upfront biventricular support. Because of the increased procedural risk for upfront biventricular support, initial decision was to undergo surgically implanted isolated LV support with a microaxial temporary LVAD with 5.5 L/min of flow and reassess its impact on RV parameters with continuous monitoring with RHC. Likely because of low PVR, isolated LV support led to improvement in RAP (23 to 12 mm Hg) and PCWP (25 to 18 mm Hg) with resolution of her acute kidney injury and lactic acidosis.

On day 25, the patient was taken for implantation of durable LVAD with HeartMate III (Abbott). Although current risk models have only modest power to predict post-LVAD RV failure, severely reduced PA pulsatility index on RHC is associated with significant risk of post-LVAD RV failure.[5] Upfront right ventricular assist device (RVAD) is preferred over delayed RV support to mitigate end organ dysfunction.[6,7] Furthermore, intraoperative insults to RV may unmask severe RV failure and lead to irreversible end-organ damage and vasoplegia. Therefore, our patient underwent upfront extracorporeal RVAD via the right internal jugular vein. Her postoperative course was complicated by bleeding requiring mediastinal exploration and reintubation eventually requiring tracheostomy. She required prolonged RV support, but her RVAD was able to be weaned and ultimately removed on hospital day 50.

FIGURE 6 **Hemodynamic Pressure Waveforms of Patient 3**



Hemodynamic pressure waveforms from the right heart catheterization of patient 3 showing RAP of 23 mm Hg with elevated v waves to 28 mm Hg, intermittent steep y descents, and notable lack of fall of RAP with inspiration consistent with positive Kussmaul's sign, square root pattern of RV waveform, reduced pulmonary artery (PA) pulsatility index (PA systolic-PA diastolic/RA), elevated pulmonary capillary wedge pressure of 25 mm Hg measured at end expiration. Abbreviations as in Figures 1 and 2.

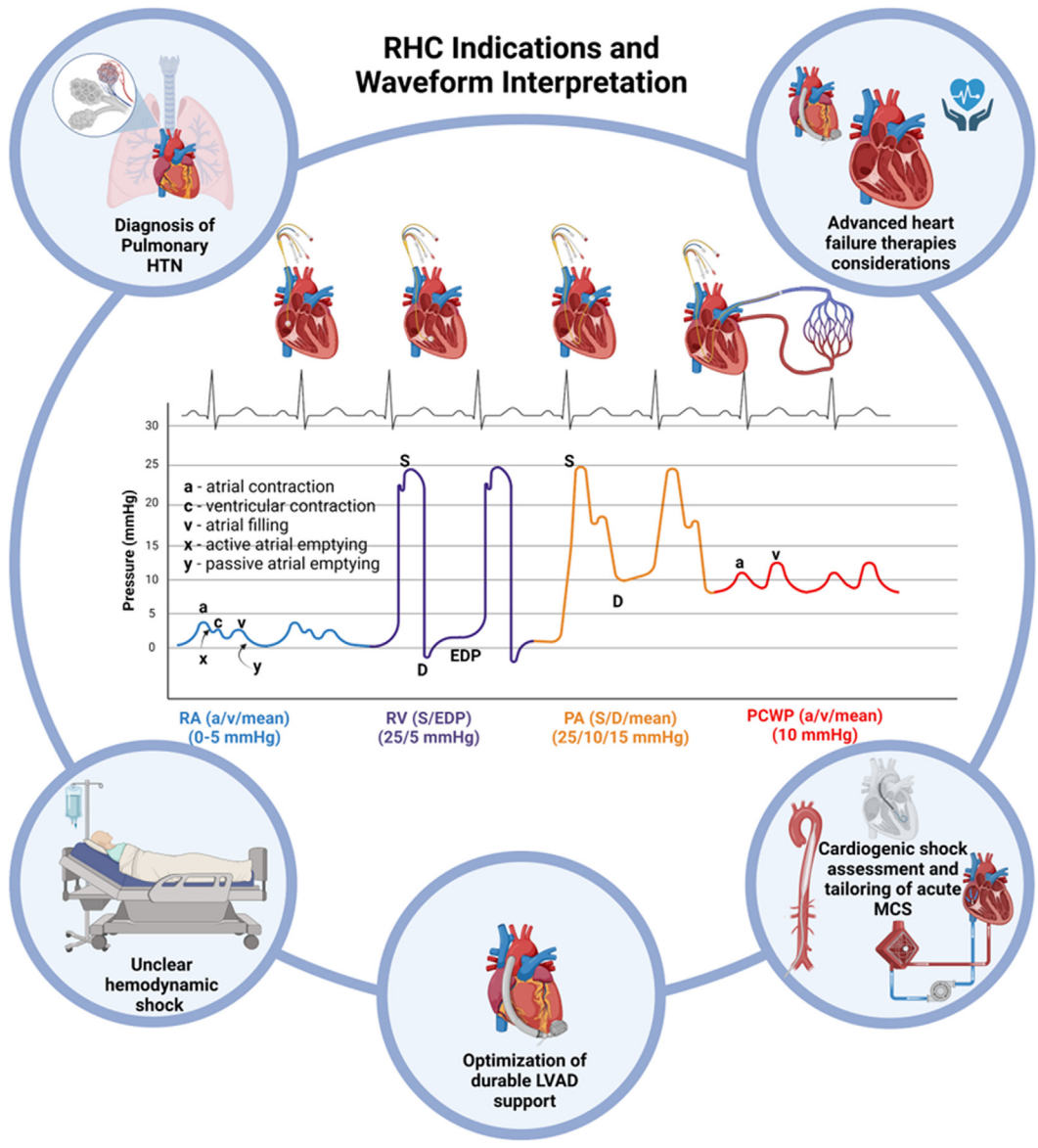
On hospital day 69, her LVAD speed was optimized by testing various settings under continuous TTE and RHC hemodynamic evaluation (Table 1). She was discharged on hospital day 83 after optimization of her functional status. In this case, the RHC was essential in guiding proper selection of initial temporary mechanical circulatory support (MCS), monitoring response to therapy, and choice for more durable MCS. After recovery from surgery, a RHC with LVAD ramp study was also instrumental in optimization of LVAD support and need for further inotropic support for RV.

DISCUSSION

RHC provides a wealth of information about a patient's hemodynamics that can be used to make clinical decisions, as presented in this case series. In addition to the quantitative assessment of the RHC, qualitative measures provide crucial insights into underlying pathophysiology as shown in our cases. As the catheter is advanced through the right-sided cardiac chambers, distinct waveforms are seen that can both identify the location of the catheter tip and provide additional information based on the waveform itself that can be used in clinical decision making (Figure 7). Normal RA waveform consists of 3 distinct positive inflections (a, c, v) and 2 negative deflections (x, y). RV waveforms have characteristic appearance with systolic peak, early diastolic decline, and end-diastolic plateau. PA pressure has systolic peak and diastolic plateau, separated by dicrotic notch reflecting closure of the pulmonic valve. PCWP usually consists of 2 distinct positive inflections (a, v) and 2 negative deflections (x, y). PCWP usually has v wave predominance, as compared to RA waveform, which usually has a wave predominance, consistent with RA coupling to low pressure, high compliance pulmonary circuit, and LA coupling to low compliance, high pressure systemic circuit (Figure 7).

The RHC is an invasive procedure with risks that must be balanced against the potential benefits. The

FIGURE 7 **Right Heart Catheterization Indications and Waveform Interpretation**



Various indications for right heart catheterization (RHC) across a spectrum of cardiac diseases including diagnosis of pulmonary hypertension, identification of the etiology of shock, choosing optimal mechanical circulatory support (MCS) in those with cardiogenic shock, and optimizing advanced therapies such as left ventricular assist device. The center of the figure compares normal pressure waveforms obtained in the RA, RV, PA, and pulmonary capillary wedge pressure (PCWP) position with labeling of the typical tracings for each. D = diastole; EDP = end diastolic pressure; HTN = hypertension; LVAD = left ventricular assist device; S = systole; other abbreviations as in Figures 1 and 6.

risk of mechanical complications, most commonly arterial puncture and hematoma, are more common during femoral (12.8%-19.4%) than internal jugular (6.3%-11.8%) access.[8] Subsequent advancement of the catheter to the PA can cause lethal arrhythmias including sustained ventricular tachycardia (3%) or ventricular fibrillation (1.3%).[9] Right bundle branch block, although typically transient, can occur in approximately 5% of patients; therefore, care must be taken in those with pre-existing left bundle branch block to avoid progression to complete heart block. Other less frequent complications of catheter advancement include PA rupture, which carries a high risk of death, and catheter knotting. Lastly,

catheter-associated infections, endocarditis, and venous thromboembolism can occur, although risk of each of these is higher when the catheter is left in place for continued monitoring.

Although prior studies have found no benefit to routine use of RHC, it has been shown to be useful in certain situations where the benefits have outweighed the potential risks.[10,11] ESC/ERS guidelines from 2022 for pulmonary hypertension recommend comprehensive hemodynamic assessment with RHC for diagnosis and subclassification, which ultimately drives therapy.[12] Some retrospective studies of patients with cardiogenic shock have also found improved mortality in those with complete hemodynamic profiling with RHC, which might be attributable to timely selection of optimal mechanical circulatory support based for each patient's hemodynamic profile.[13,14] Other areas that have not yet been rigorously tested but might benefit from RHC include diagnosis and treatment of undifferentiated shock, mixed cardiogenic and septic shock, moderate and severe valvular heart disease, cardiac tamponade, intracardiac shunting, or the management of specific conditions such as acute renal failure, decompensated cirrhosis, or severe preeclampsia and eclampsia. The overall trend in RHC use represents a shift from the indiscriminate use in the critically ill patients to a more judicious use in clinically appropriate situations such as has been demonstrated with this series.

The cases in this series highlight examples of 3 clinical scenarios in which RHC helped define the diagnosis, risk stratify, and ultimately design treatment plan for each of the patients; specifically, subclassifying pulmonary hypertension for appropriate treatment, defining the severity of cardiomyopathy, and choosing the optimal MCS for a patient in cardiogenic shock along with optimization of LVAD parameters (**Figure 7**).

FUNDING SOURCE AND AUTHOR DISCLOSURES

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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REFERENCES

1. Wiener RS, Welch HG. Trends in the use of the pulmonary artery catheter in the United States, 1993-2004. *JAMA*. 2007;298(4):423-429.

2. Ikuta K, Wang Y, Robinson A, Ahmad T, Krumholz HM, Desai NR. National trends in use and outcomes of pulmonary artery catheters among Medicare beneficiaries, 1999-2013. *JAMA Cardiol*. 2017;2(8):908-913.

3. Galiè N, Channick RN, Frantz RP, et al. Risk stratification and medical therapy of pulmonary arterial hypertension. *Eur Respir J*. 2019;53(1):1801889.

4. Martens P, Bhattacharya S, Longinow J, et al. Hemodynamic profiling and prognosis in cardiac amyloidosis. *Circ Heart Fail*. 2023;16(3):e010078.

5. 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(2):110-116.

6. Bhama JK, Bansal U, Winger DG, et al. Clinical experience with temporary right ventricular mechanical circulatory support. *J Thorac Cardiovasc Surg*. 2018;156(5):1885-1891.

7. Kumar S, Derbala MH, Nguyen DT, et al. A multi-institutional retrospective analysis on impact of RV acute mechanical support timing after LVAD implantation on 1-year mortality and predictors of RV acute mechanical support weaning. *J Heart Lung Transplant*. 2022;41(2):244-254.

8. McGee DC, Gould MK. Preventing complications of central venous catheterization. *N Engl J Med*. 2003;348(12):1123-1133.

9. Sprung CL, Pozen RG, Rozanski JJ, Pinero JR, Eisler BR, Castellanos A. Advanced ventricular arrhythmias during bedside pulmonary artery catheterization. *Am J Med*. 1982;72(2):203-208.

10. Harvey S, Harrison DA, Singer M, et al. Assessment of the clinical effectiveness of pulmonary artery catheters in management of patients in intensive care (PAC-Man): a randomised controlled trial. *Lancet*. 2005;366(9484):472-477.

11. Connors AF Jr, Speroff T, Dawson NV, et al. The effectiveness of right heart catheterization in the initial care of critically ill patients. SUPPORT Investigators. *JAMA*. 1996;276(11):889-897.

12. Humbert M, Kovacs G, Hoeper MM, et al. 2022 ESC/ERS guidelines for the diagnosis and treatment of pulmonary hypertension. *Eur Heart J*. 2022;43(38):3618-3731.

13. 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(11):903-913.

14. Hernandez GA, Lemor A, Blumer V, et al. Trends in utilization and outcomes of pulmonary artery catheterization in heart failure with and without cardiogenic shock. *J Card Fail*. 2019;25(5):364-371.

KEY WORDS acute heart failure, chronic heart failure hemodynamics, right-sided catheterization