Review Article

Invasive Cardiopulmonary Hemodynamic Assessment in Patients with Advanced Heart Failure: How to Interpret?

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Abstract

Invasive cardiopulmonary hemodynamic assessment by means of a pulmonary artery catheter is an important tool for evaluating patients with advanced heart failure. It makes it possible to definitively diagnose pulmonary hypertension and, when pulmonary hypertension is present, to classify it as isolated post-capillary or combined pre- and post-capillary. Its use is mandatory in evaluation for heart transplantation and mechanical circulatory assist device implantation. Furthermore, it can be very useful in the management of cardiogenic shock.

Introduction

The use of catheters for invasive cardiac assessment has been described since the beginning of the twentieth century.[1] However, it was only starting in the 1970s that pulmonary artery catheters (PAC) began to be used for hemodynamic assessment at the bedside of critical patients. Their use became popular in subsequent years, to the extent that, by the end of the 2000s, approximately 1.5 million catheters were being sold annually in the United States.[2] Studies with negative results for routine use of PAC in critical patients in intensive therapy[3] and in patients with symptomatic heart failure (HF) with signs of severity but without cardiogenic shock[4] led to a reduction in their use. However, more recent data on patients with cardiogenic shock in the contemporary era incorporating the use of mechanical circulatory assist devices in treatment have demonstrated an association of PAC use with greater survival.[5]

Currently, PAC are recognized as useful and recommended in some clinical scenarios, including the following:[6-8]

- assessment of valvular and congenital diseases, especially when there is disagreement between the clinical and echocardiographic findings, as well as in the assessment of pulmonary hypertension (PH) and pulmonary reactivity before correction;
- diagnosis, prognostic assessment, and reactivity test to guide therapy for pulmonary arterial hypertension;
- early diagnosis of HF with preserved ejection fraction in patients with dyspnea;
- assessment and management of patients with advanced HF, both for indication of advanced therapy (transplantation or mechanical circulatory assist devices) and for assistance in management of cardiogenic shock.

Keywords

Heart Failure; Hemodynamic Monitoring; Pulmonary Hypertension

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Definition and classification of pulmonary hypertension

One of the main objectives of using PAC in advanced HF is assessment of PH. The definition of PH has recently been modified, and it is currently diagnosed in the presence of mean pulmonary artery pressure (mPAP) above 20 mmHg at rest, thus reducing the previously used cut-off value of 25 mmHg.[9]

A study with invasive assessment of 1,187 healthy individuals showed that the mean value for mPAP was 14.0 ± 3.3 mmHg, and this value was independent of sex and ethnicity.[10] Considering this normal value, mPAP > 20 mmHg would be 2 standard deviations above. Moreover, observational studies have demonstrated that small elevations in pulmonary pressure (mPAP between 20 and 25 mmHg) have a prognostic impact on symptoms, hospitalization, and mortality.[11] In a meta-analysis of 15 studies, the risk ratio for mortality was 1.52 among patients with mPAP of 19 to 24 mmHg when compared to patients with lower pressures.[12]

PH is currently classified into 5 groups that combine clinical conditions with similar pathophysiological mechanisms, clinical presentation, hemodynamic characteristics, and therapeutic management (Table 1).[9,13]

Pulmonary hypertension in heart failure

The main characteristic of group 2 PH is the presence of elevated pulmonary artery occlusion pressure (PAOP) (> 15 mmHg), which reflects increased left ventricular filling pressure. This group accounts for 65% to 80% of patients with PH.[14]

Group 2 PH results primarily from increased left ventricular filling pressures due to systolic and/or diastolic ventricular dysfunction or to aortic or mitral valve disease. This increased left chamber pressure is transmitted retrogradely to the pulmonary circulation (post-capillary component). Persistent elevation of pressure in this area leads to endothelial dysfunction with increased vasoconstrictor action, decreased available nitric oxide, and desensitization of vasodilation induced by natriuretic

peptides.[15] Subsequently, activation of inflammatory mediators and metabolic factors occurs, which will lead to vessel remodeling, with intimal fibrosis and hypertrophy of the middle layer of pulmonary arterioles, which are histological changes similar to those observed in primary pulmonary arterial hypertension.[16] The prevalence of PH in the population with HF with reduced ejection fraction is estimated at 40% to 75%.[14]

According to the presence or absence of functional or morphological alteration of pulmonary arterioles associated with the post-capillary component, PH in HF can be further classified as isolated post-capillary or combined pre- and post-capillary (Table 2). What indicates the presence of alterations in the pulmonary vasculature is increased pulmonary vascular resistance (PVR), which is calculated by dividing the transpulmonary gradient (TPG) by the cardiac output.[9] In turn, TPG corresponds to the difference between mPAP and PAOP, where > 15 mmHg indicates the presence of an associated pre-capillary component.[17]

Indications for use of pulmonary artery catheter in patients with advanced heart failure

Invasive cardiopulmonary hemodynamic assessment by PAC continues to be an important tool in patients with advanced HF (Figure 1). The main recommendations for the use of PAC in patients with advanced HF are as follows:

- Patients being evaluated for heart transplantation (HT) with the objective of evaluating the presence of PH (class of recommendation: I, level of evidence: B);[8]
- Every 3 to 6 months in patients listed for HT, especially in the presence of previous PH or worsening HF (class of recommendation: I, level of evidence: B);[18]
- Patients who are candidates for implantation of long-term left ventricular assist devices (VAD) with the objective of assessing right ventricular (RV) function and predicting RV failure after VAD implantation (class of recommendation: I, level of evidence: C);[7]
- Patients with refractory symptoms or cardiogenic shock, with the objective of assisting in hemodynamic optimization (class of recommendation: IIa, level of evidence: B).[8]

Practical aspects

Techniques for cardiopulmonary hemodynamic assessment

The standardization of techniques for correct assessment of the hemodynamic parameters obtained with PAC is essential to clinical and hemodynamic diagnosis, as well as to the implementation of appropriate treatment.

Table 3 summarizes the main practical aspects of techniques for assessing cardiopulmonary hemodynamics with right catheterization.[11]

The normal values of intravascular and cavity pressures and saturation are displayed in Table 4.

Table 1 – Classification of pulmonary hypertension

Group 1	Pulmonary arterial hypertension
Group 2	Pulmonary hypertension due to left heart disease
Group 3	Pulmonary hypertension due to lung disease and/or hypoxia
Group 4	Chronic thromboembolic pulmonary hypertension and other pulmonary artery obstructions
Group 5	Pulmonary hypertension with unclear multifactorial mechanisms

Table 2 – Classification of pulmonary hypertension in heart failure

Isolated post-capillary PH	mPAP > 20 mmHg
	PAOP > 15 mmHg
	PVR < 3 Woods
	TPG < 15 mmHg
Combined pre- and post-capillary PH	mPAP > 20 mmHg
	PAOP > 15 mmHg
	PVR ≥ 3 Woods
	TPG ≥ 15 mmHg

mPAP: mean pulmonary artery pressure; PAOP: pulmonary artery occlusion pressure; PH: pulmonary hypertension; PVR: pulmonary vascular resistance; TPG: transpulmonary gradient.

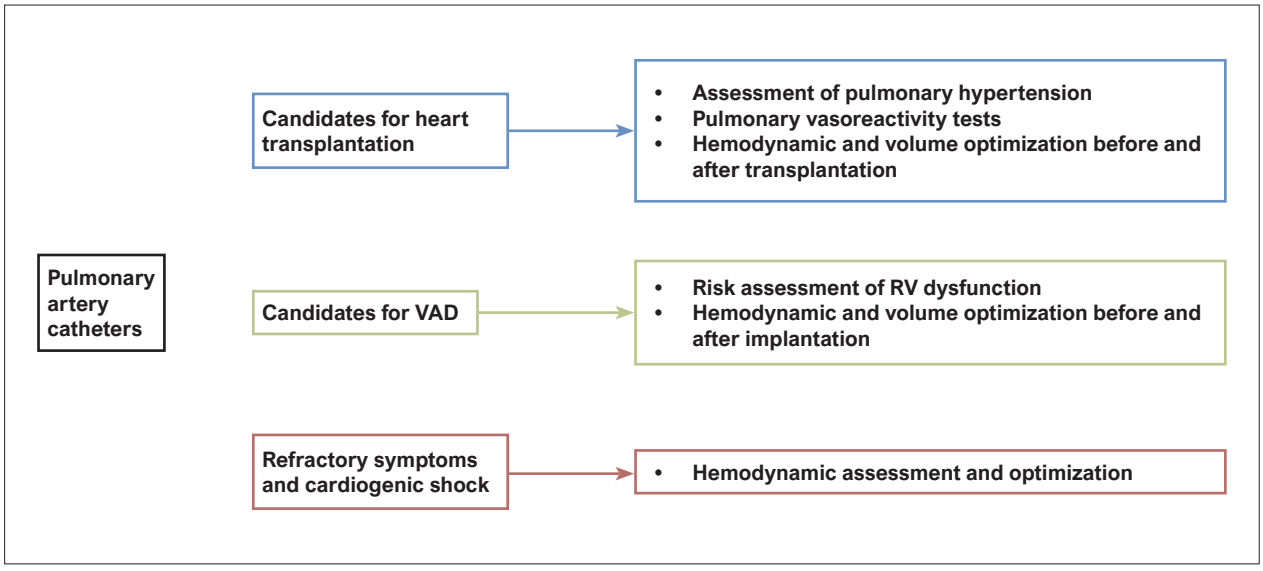

Figure 1 – *Indications for invasive cardiopulmonary hemodynamic assessment in patients with advanced heart failure. RV: right ventricle; VAD: ventricular assist device.*

Table 3 – Techniques for cardiopulmonary hemodynamic assessment

Preparation and positioning	Prior confirmation of catheter positioning (radiography or radioscopy)
	Full view of the hemodynamic monitor
	Patient in a supine position, with legs extended; avoid taking measurements while the patient is talking, coughing, or moving.
	Leveling of pressure transducers (RAP, PAP), which should be zeroed to atmospheric pressure at the level of the LA (mean distance between the patient's anterior sternum and back).
Quality assessment of tracings	Identify the presence of adequate pressure curves, without interference or artifacts.
	Aspiration of air bubbles from catheters and subsequent lavage can minimize artifacts.
Determination of right cardiac and pulmonary pressures	Pressure measurements should be performed during spontaneous breathing without performing the Valsalva maneuver.
	Measurements at the end of expiration are preferable.
	Measure right atrium, right ventricle and pulmonary artery pressures.
Measurement of pulmonary capillary pressure or PAOP	Measurement should be performed with an expiratory pause, without performing a Valsalva maneuver.
	If PAOP is very high and is questioned, correct confirmation of pulmonary artery occlusion can be achieved by measuring oxygen saturation > 90%.
	Mean PAOP pressure is generally correlated with LA pressure (and LVEDP).
	Presence of important MR, giant V wave, and AF may overestimate PAOP and should be reported.
Cardiac output measurements	Thermodilution measurements are preferable over indirect Fick calculation.

LA: left atrium; LVEDP: left ventricular end-diastolic pressure; MR: mitral regurgitation; PAOP: pulmonary artery occlusion pressure; PAP: pulmonary artery pressure; RAP: right atrial pressure.

Assessment of candidates for heart transplantation

The presence of PH with elevated PVR is classically associated with increased mortality after HT due to RV graft dysfunction, especially in individuals with PH with a pre-capillary component who do not show vasoreactivity in tests with pulmonary vasodilators.[18,19] The *Brazilian Guidelines for Cardiac Transplantation* consider this non-reactive (fixed) pre-capillary PH as a contraindication to HT.[20]

Thus, invasive cardiopulmonary hemodynamic assessment with a PAC is indicated for all patients who are candidates for HT.[20] In addition to identifying the presence of PH, it makes it possible to determine the hemodynamic factors that are possibly responsible and, in patients with PH with a pre-capillary component, to assess the reduction of pulmonary pressures with vasoreactivity tests. Invasive cardiopulmonary hemodynamic assessment

Table 4 – Normal values of intravascular and cavity pressures and saturation

	Systolic/diastolic pressure (mean) (mmHg)	Saturation (%)
Right atrium	(5-8)	70
Right ventricle	26/2	70
Pulmonary artery	26/8 (14)	70
Pulmonary occlusion	(8)	100
Left atrium	(8)	98
Left ventricle	120/8	98

also assists in hemodynamic optimization by adjusting blood volume, cardiac output, and pulmonary pressure before HT (Chart 1).

Patients with post-capillary PH generally do not need to undergo a pulmonary vasoreactivity test, given that the main hemodynamic components are hypervolemia and increased left ventricular filling pressures (increased systemic vascular resistance). Thus, diuretics and systemic vasodilators are the basis for volume and hemodynamic optimization and consequent reduction in pulmonary pressure (Chart 1).

Patients with PH with combined pre- and post-capillary components should undergo a pulmonary vasoreactivity test with the objective of identifying a component that is reactive to vasodilators (pulmonary vascular vasoconstriction). In these cases, the drop in pulmonary pressure and normalization of TPG and PVR with the pulmonary vasoreactivity test allow candidacy for HT (Chart 1).

In patients whose pulmonary pressure does not reduce or whose PVR does not normalize, treatment for hemodynamic optimization guided by invasive monitoring should be maintained for at least 24 to 48 hours, considering the use of diuretics (or even ultrafiltration), vasodilators, and inotropes. Left ventricular decompression strategies such as intra-aortic balloon can be considered, with the objective of reducing pulmonary pressures.[18]

The persistence of significant PH with high TPG and PVR, even after these strategies, is considered a contraindication for HT. In this scenario, VAD implantation as a bridge to later candidacy is a supportive option in selected patients. Left ventricular decompression obtained with the use of a VAD can lead to reduced pulmonary artery pressure and PVR in the medium term, making the patient a candidate for HT.[21-23] In patients with advanced HF and significant PH with a persistent pre-capillary component, heterotopic HT or combined heart-lung transplantation (in qualified centers), VAD implantation as a target therapy, and palliative care are options for treatment and support (Figure 2).

Some measurements during cardiopulmonary hemodynamic assessment may eventually be in disagreement with the patient's actual hemodynamics and lead to errors in interpretation. Table 5 describes some common errors and problems that occur during invasive cardiopulmonary assessment.

Chart 1 – Cardiopulmonary hemodynamic assessment in candidates for heart transplantation with pulmonary hypertension

	Hemodynamic assessment	PH classification	Suggested approach	New condition	Diagnosis	HT
PASP ≥ 50 mmHg	PAOP < 15 mmHg TPG ≥ 15 PVR ≥ 3 Woods	Pre-capillary PH	Pulmonary vasoreactivity Nitric oxide	PASP < 50 mmHg TPG < 15 PVR < 3 Woods	Reactive PH	✓
				PASP ≥ 50 mmHg TPG ≥ 15 PVR ≥ 3 Woods	Fixed PH	✘
	PAOP ≥ 15 mmHg TPG < 15 PVR < 3 Woods	Post-capillary PH	RAP > 12 mmHg: Diuretics SVR > 1200 $_{dynas/s/cm^5}$:SNP	PASP < 50 mmHg TPG < 15 PVR < 3 Woods		✓
	PAOP ≥ 15 mmHg TPG ≥ 15 PVR ≥ 3 Woods	Combined pre- and post-capillary PH	Pulmonary vasoreactivity SNP	PASP < 50 mmHg TPG < 15 PVR < 3 Woods	Reactive PH	✓
				PASP ≥ 50 mmHg TPG ≥ 15 PVR ≥ 3 Woods	Fixed PH	✘

HT: heart transplantation; PAOP: pulmonary artery occlusion pressure; PASP: pulmonary artery systolic pressure; PH: pulmonary hypertension; PVR: pulmonary vascular resistance; RAP: right atrial pressure; SNP: sodium nitroprusside; SVR: systemic vascular resistance; TPG: transpulmonary gradient.

Assessment of candidates for ventricular assist device implantation

RV dysfunction is one of the main causes of death and early morbidity after VAD implantation.[24,25] Accurate assessment of the risk of RV dysfunction during the early postoperative period after VAD implantation is important for planning eventual temporary circulatory support for the RV, which may attenuate the risk of postoperative mortality.[26-28]

Clinical, laboratory, echocardiographic, and hemodynamic assessment are part of the majority of scores that predict RV dysfunction after VAD implantation; however, these tools are still not totally reliable in correctly predicting RV dysfunction in this scenario,[29,30] making this assessment challenging.

Pulmonary hemodynamic assessment assists in prediction of RV dysfunction after VAD implantation.[25] The main hemodynamic parameters and their references for predicting RV dysfunction are described Table 6.

In addition to its role in pre-implantation assessment for VAD, cardiopulmonary hemodynamic assessment can assist in the management of some situations during the postoperative period after implantation, as follows:

- During the early postoperative period:[35]
  - Management of pulmonary hypertension and RV preload;
  - Left ventricular decompression (assisting in the decision to increase or decrease VAD rotations).
- Long-term follow-up:
  - Refractory patients with symptoms of HF: assessment of left ventricular decompression, RV function, and aortic regurgitation;[36]
  - Assessment of optimal VAD rotations (ramp test);[37]
  - Hemodynamic optimization with decoupling between diastolic pulmonary pressure and PAOP.[38]

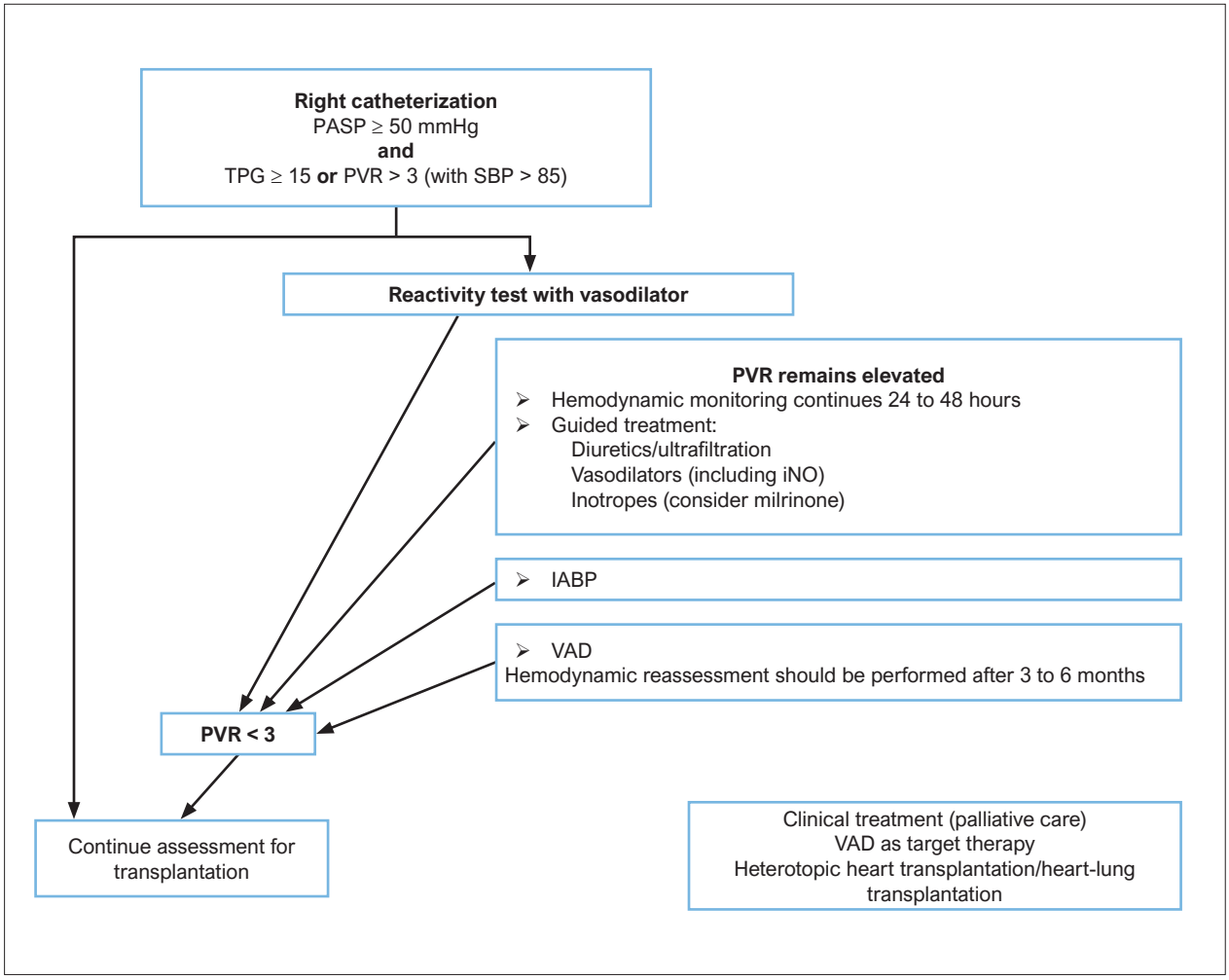

Figure 2 – *Assessment for heart transplantation in patients with advanced heart failure and pulmonary hypertension. IABP: intra-aortic balloon pump; iNO: inhaled nitric oxide; PADP: pulmonary artery diastolic pressure; PASP: pulmonary artery systolic pressure; PVR: pulmonary vascular resistance; SBP: (systemic) systolic blood pressure; TPG: transpulmonary gradient; VAD: ventricular assist device.*

Conclusion

Invasive cardiopulmonary hemodynamic assessment continues to be an important tool for assessing patients with advanced HF, especially in candidates for HT and VAD, as well as in the management of complex patients with unclear hemodynamics and cardiogenic shock.

Appropriate techniques for invasive hemodynamic assessment and correct interpretation of curves and pressures, in a systematic manner, are fundamental to understanding the mechanisms that involve cardiopulmonary hemodynamic changes. They allow guided hemodynamic optimization, thus promoting better clinical outcomes.

Author Contributions

Conception and design of the research; Acquisition of data; Analysis and interpretation of the data; Writing of the manuscript; Critical revision of the manuscript for intellectual content: Biselli B e Seguro LFBC.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Ethics approval and consent to participate

This article does not contain any studies with human participants or animals performed by any of the authors.

Table 5 – Errors and problems in cardiopulmonary hemodynamic assessment

Situation	Comments
Contraindication of HT with a single isolated hemodynamic assessment	Identify hemodynamic component of PH and repeat new measurements after hemodynamic optimization and vasoreactivity tests.
Contraindication of HT with only 1 hemodynamic criterion altered	Always assess all cardiopulmonary hemodynamic variables together (pulmonary pressure, gradients, and PVR)
Disagreement between pulmonary pressure, PVR, and pulmonary gradients	
• Low PVR and elevated gradients	Possibly overestimated cardiac output (e.g., cardiac shunt, measurement error, obesity)
• Elevated PVR and low gradients	Possibly underestimated cardiac output (e.g., important RV dysfunction, severe TR)
• Elevated PVR and gradients, with low pulmonary pressures	Overestimated PAOP (e.g., severe MR) Check patient positioning, leveling, and correct execution of pressure.
Vasoreactivity test with iNO in patients with hypovolemia or very high PAOP	Risk of acute pulmonary edema

HT: heart transplantation; iNO: inhaled nitric oxide; MR: mitral regurgitation; PAOP: pulmonary artery occlusion pressure; PH: pulmonary hypertension; PVR: pulmonary vascular resistance; RV: right ventricle; TR: tricuspid regurgitation.

Table 6 – Cardiopulmonary hemodynamic assessment in candidates for long-term left ventricular assist devices

	Formula	Predictor of RV dysfunction
Pulmonary artery pulsatility index	PASP – PADP / RAP	< 2.0 [31]
CVP / PCP		> 0.63 [32]
RVSWI	[(CI / HR) × (MAP – PCP)] × 0.0136	≤ 5.0 g/m/m^2 [33]
RVSWI + PVR		RVSWI ≤ 5.0 g/m/m^2 PVR > 3.7 Woods [34]
Diastolic pulmonary gradient	PDAP – PCP	≥ 7 [34]

CI: cardiac index; CVP: central venous pressure; HR: heart rate; MAP: mean arterial pressure; PADP: pulmonary artery diastolic pressure; PASP: pulmonary artery systolic pressure; PCP: pulmonary capillary pressure; PVR: pulmonary vascular resistance; RAP: right atrial pressure; RV: right ventricle; RVSWI: right ventricular stroke work index.

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