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EDITOR'S NOTE

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This edition of "THE REVIVAL" discusses the art and science of right heart catheterization in the context of advanced heart failure and cardiac transplant. It sheds light on the prognostic and therapeutic role of right heart catheterization enlisting the normal ranges of various basic hemodynamic measurements as well as advanced calculated indices. Elevated pulmonary vascular resistance is a significant issue which can adversely affect the outcomes of cardiac transplant.

This review also focuses on the various vasoreactvity manoeuvres to assess reversibility of pulmonary vascular resistance thus helping prognosticate patients awaiting cardiac transplant or durable left ventricular assist device implants. Plus, the practical tips on interpreting waveforms in the setting of severe mitral regurgitation and constrictive pericarditis adds value to the review particularly for those in the early training phase of their cardiology careers.

Warm regards,
Dr. Talha Meeran

SUB EDITOR'S NOTE

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Right heart catheterization remains central to decision-making in advanced heart failure and transplant care, where accurate hemodynamic interpretation often determines outcomes. The review article in this issue of the REVIVAL provides a clear and practical framework—moving from waveform fundamentals to advanced indices such as PAPi, RVSWI, and cardiac power output—while highlighting the critical role of pulmonary vascular assessment and vasodilator testing in transplant candidacy.

At a time when non-invasive surrogates are widely used, this review reinforces the irreplaceable value of invasive hemodynamics in complex, high-risk patients. It serves as a concise yet comprehensive guide for clinicians seeking to translate numbers at the bedside into sound, patient-centered clinical decisions.

Warm regards,
Dr. Aditi Singhvi, FACC

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RIGHT HEART CATHETERIZATION IN ADVANCED HEART FAILURE AND TRANSPLANT EVALUATION: BASICS, WAVEFORM INTERPRETATION, AND HEMODYNAMIC DECISION-MAKING

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Abstract

Right heart catheterization (RHC) serves as the gold standard for hemodynamic assessment in pulmonary hypertension and heart failure, providing precise data on pressures and waveforms that non-invasive imaging cannot fully replicate. This comprehensive review details the technical aspects of RHC—from catheter positioning to the interpretation of pulmonary capillary wedge pressure (PCWP)—while emphasizing the importance of advanced indices like PAPi and RVSWI in predicting right ventricular failure. A major clinical focus is placed on vasodilator testing for risk stratification in heart transplant and mechanical circulatory support candidates, alongside the application of Forrester hemodynamic profiling to guide therapeutic interventions. By contrasting invasive measurements with echocardiographic surrogates and identifying specific findings for conditions like constrictive pericarditis, the review establishes a robust framework for managing complex heart failure and transplant patients in modern clinical practice.

Introduction

Right heart catheterization (RHC), often performed via a pulmonary artery catheter, remains the **gold standard** for the comprehensive hemodynamic evaluation of pulmonary hypertension (PH). This invasive procedure is instrumental in distinguishing between pre-capillary and post-capillary PH by providing precise measurements of cardiac output and pulmonary vascular resistance (PVR). Beyond diagnosis, RHC is a critical component of transplant assessment, helping clinicians determine whether a patient requires a heart transplant alone or a combined heart-lung transplant. Additionally, it serves as a Class IIa indication for managing complex heart failure and cardiogenic shock, where real-time hemodynamic data is essential to guide therapeutic interventions and optimize patient outcomes. [1,2]

Methods

The Swan-Ganz catheter (figure 1), or pulmonary artery catheter (PAC), is a sophisticated diagnostic tool characterized by its balloon-tipped, flow-directed design. It features several specialized components that allow for multi-parametric hemodynamic monitoring: the distal port, typically color-coded yellow, is positioned in the pulmonary artery to provide continuous pressure readings, while the blue proximal port sits in the right atrium to measure central venous pressure and facilitate thermodilution bolus injections. A red-coded balloon at the tip is intermittently inflated to allow the catheter to float into a "wedged" position for measuring pulmonary capillary wedge pressure (PCWP). Furthermore, an integrated thermistor at the distal end detects temperature fluctuations, which are essential for calculating cardiac output. [1,2]

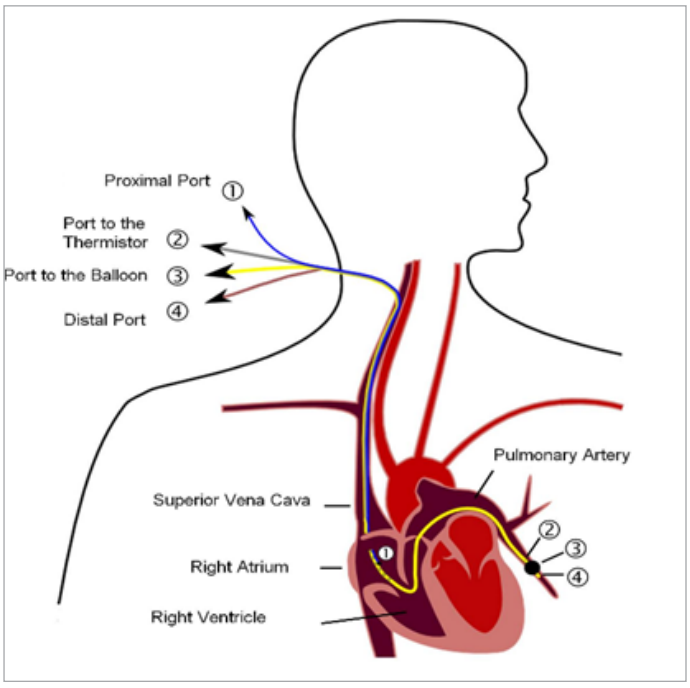


Figure 1: Placement of a multi-lumen pulmonary artery catheter. The distal tip (4) is positioned in the pulmonary artery for pressure monitoring, while the proximal port (1) rests in the right atrium for CVP monitoring and fluid administration. The thermistor (2) and balloon (3) components are essential for thermodilation cardiac output measurements and wedge pressure acquisition, respectively.

Optimal placement of the pulmonary artery catheter is contingent upon its positioning within the West Zones of the lung (figure 2), specifically targeting **West Zone 3** at the lung base. Physiologically, Zone 3 is characterized by a pressure gradient where arterial and venous pressures exceed alveolar pressure (Pa > Pv > PA), ensuring continuous blood flow. This positioning is vital for clinical accuracy because it maintains an uninterrupted column of blood between the catheter tip and the left atrium, allowing the pulmonary capillary wedge pressure (PCWP) to serve as a reliable surrogate for left atrial pressure. Conversely, placing the catheter in Zones 1 or 2 can lead to measurement errors, as higher alveolar pressures may interrupt flow and cause the PCWP to reflect respiratory artifacts or alveolar pressure rather than true left-sided cardiac filling pressures as described in table 1.[1,2]

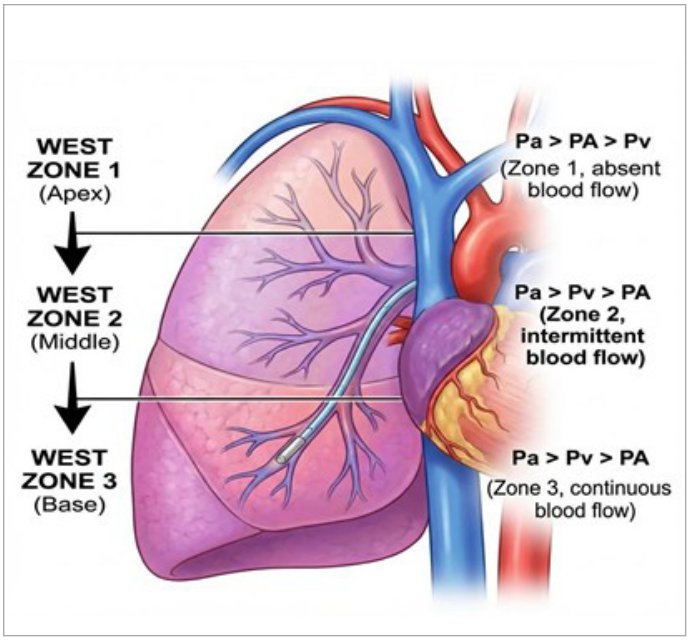


Figure 2. Pulmonary Blood Flow and West Zones.

The lung is divided based on the interaction between alveolar (PA), arterial (Pa), and venous (Pv) pressures:

Zone	Region	Pressure Relationship	Flow Characteristics	Clinical Significance
Zone 1	Apex (Top)	PA > Pa > Pv	Minimal/Absent: Alveolar pressure compresses capillaries, creating "dead space."	Usually only present in disease (shock, hemorrhage) or during positive pressure ventilation.
Zone 2	Middle	Pa > PA > Pv	Intermittent: Flow occurs when arterial pressure rises above alveolar pressure (recruitment).	Flow is determined by the difference between arterial and alveolar pressure ($P_a - P_A$).
Zone 3	Base (Bottom)	Pa > Pv > PA	Continuous: Gravity increases hydrostatic pressure; capillaries stay open.	Gold standard for PA Catheter tip placement to ensure accurate pressure readings.

Table 1: PA = Alveolar pressure; Pa = Arterial pressure; Pv = Venous pressure; (>) indicates pressure dominance.

Procedural Technique and Reference Hemodynamics

The execution of a right heart catheterization typically begins with obtaining vascular access via the right internal jugular or femoral veins, through which the catheter is advanced sequentially from the right atrium (RA) into the right ventricle (RV), the pulmonary artery (PA), and finally to the wedge position (PCWP). Establishing these baseline values is critical for identifying hemodynamic abnormalities in resting supine adults. Normal physiologic ranges include a mean RA pressure of **2–6 mmHg** and a mean PCWP of **6–12 mmHg**. During the transit through the right heart, systolic pressures in both the RV and PA are expected to fall between **15–25 mmHg**, though the PA maintains a higher diastolic floor of **8–15 mmHg** compared to the near-zero diastolic pressure of the RV as shown in table 2. Accurate interpretation of these approximate reference values is essential for the differential diagnosis of various cardiovascular and pulmonary pathologies.[1,2]

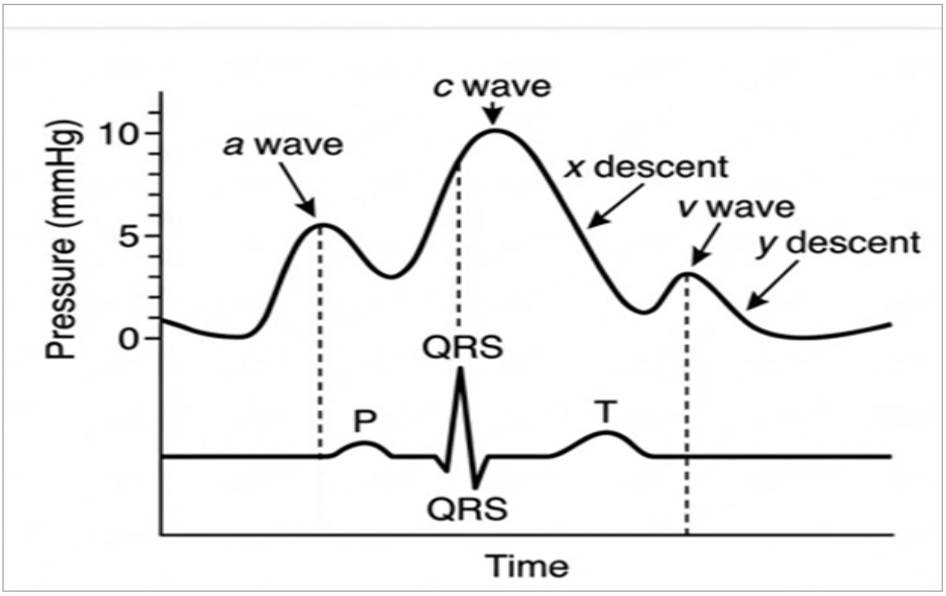


Figure 3. Central Venous Pressure (CVP) Waveform Correlation with ECG.

The graphic depicts the mechanical events of the cardiac cycle relative to electrical activity:

Chamber / Vessel	Systolic (mmHg)	Diastolic (mmHg)	Mean (mmHg)
Right Atrium (RA)	-	-	2 - 6
Right Ventricle (RV)	15 - 25	0 - 8	-
Pulmonary Artery (PA)	15 - 25	8 - 15	10 - 20
PCWP (Wedge)	-	-	6 - 12

Table 2: mmHg = Millimeters of mercury; PCWP = Pulmonary Capillary Wedge Pressure (reflects left atrial pressure).

Right Atrial (RA) Waveform Analysis

Interpretation of the right atrial (RA) pressure trace(figure 3) is essential for diagnosing valvular and structural heart diseases. The normal RA waveform is composed of three distinct positive waves and two descents: the **'a' wave**, representing atrial contraction, follows the **P wave** on an electrocardiogram (ECG) and may be enlarged in cases of tricuspid stenosis or right ventricular hypertrophy. The **'c' wave** corresponds to tricuspid valve closure and its subsequent bulging into the atrium, while the **'v' wave** reflects passive atrial filling against a closed tricuspid valve; a prominent **'v' wave** is a classic sign of tricuspid regurgitation. Complementing these peaks are the **'x' descent**, caused by atrial relaxation and the downward movement of the septum, and the 'y' descent, which represents rapid atrial emptying upon the opening of the tricuspid valve. Notably, a steep **'y' descent** is often indicative of constrictive pericarditis.(1,2) as shown in table 3.

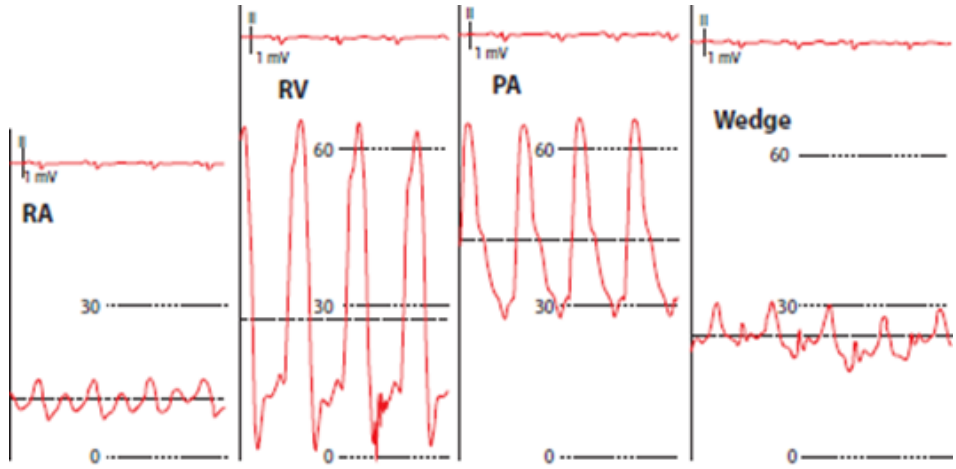
Figure 3. Central Venous Pressure (CVP) Waveform Correlation with ECG. The graphic depicts the mechanical events of the cardiac cycle relative to electrical activity:

Wave/Descent	Cardiac Event	ECG Correlation
a wave	Atrial contraction	End of P wave
c wave	Ventricular systole (valve bulge)	End of QRS
x descent	Atrial relaxation	ST segment
v wave	Venous filling of atrium	End of T wave
y descent	Tricuspid valve opening	Before next P wave

Table 3: Central Venous Pressure (CVP) Waveform Correlation with ECG

During right heart catheterization, distinguishing between right ventricular (RV) and pulmonary artery (PA) waveforms (figure 4) is essential for confirming correct catheter tip progression. While both chambers exhibit similar systolic pressures, typically ranging from **15–25 mmHg**, they are differentiated by their diastolic characteristics. The transition

from the RV to the PA is marked by a sudden and significant rise in diastolic pressure; whereas the RV diastolic pressure drops to near-zero levels **(0–5 mmHg)**, the PA maintains a higher diastolic floor of **8–15 mmHg**. Furthermore, the PA waveform features a distinct **dicrotic notch** on its downstroke, which represents the physical closure of the pulmonic valve—a morphological feature entirely absent in the RV trace. Identifying these physiological landmarks ensures the clinician has successfully crossed the pulmonic valve before proc eeding to obtain wedge pressure measurements.[1,2]



Source: Jesse B. Hall, Gregory A. Schmidt, John P. Kress: *Principles of Critical Care*, 4th Edition: www.accessmedicine.com

Figure 4. Normal Pressure Waveforms During Pulmonary Artery Catheter Insertion. The series displays the characteristic pressure changes (in mmHg) as the catheter tip advances through the right heart.

Pulmonary Capillary Wedge Pressure (PCWP) Dynamics

The pulmonary capillary wedge pressure (PCWP)(figure 5) serves as a vital indirect measure of left atrial (LA) pressure and left ventricular end-diastolic pressure (LVEDP), provided mitral valve disease is absent. Morphologically, the PCWP waveform resembles the right atrial trace, containing 'a' and 'v' waves, though it appears damped and delayed because the pressure pulse must travel through the pulmonary capillary bed before reaching the catheter tip. Clinical interpretation of these waveforms is diagnostic; for instance, a pathologically large 'v' wave suggests mitral regurgitation due to systolic overfilling of the left atrium. Furthermore, a PCWP exceeding **15 mmHg** is a hallmark indicator of post-capillary pulmonary hypertension, signifying that the primary pathology resides within the left heart. (1,2) normal hemodynamic pressure values for the right heart and pulmonary vasculature, which are essential for assessing cardiac output and lung circulation health is shown in table 4.

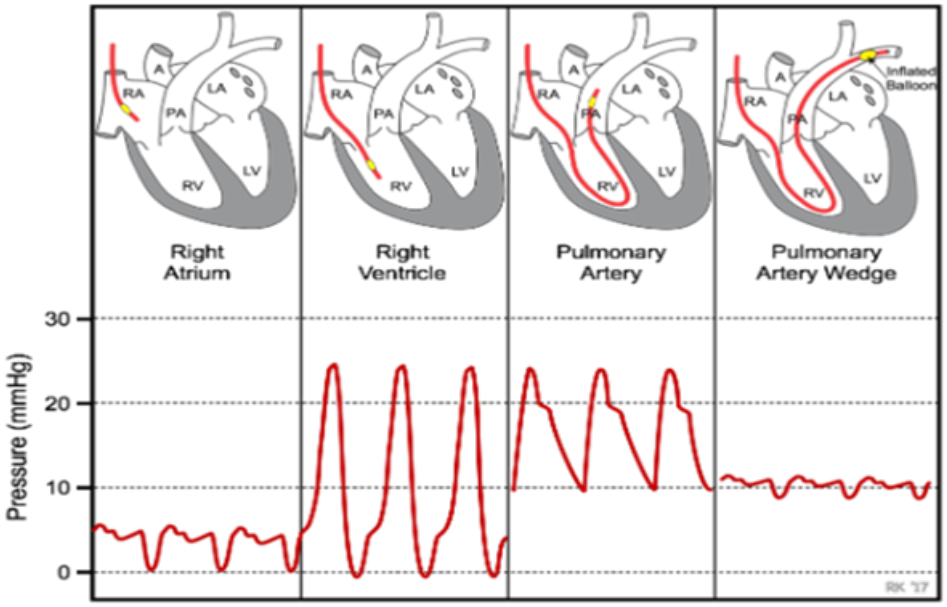


Figure 5. Anatomical Progression and Pressure Waveforms During Pulmonary Artery Catheterization. The schematic illustrates the catheter's path through the heart chambers alongside the characteristic pressure tracings (in mmHg) encountered during insertion.

Location	Typical Systolic (mmHg)	Typical Diastolic (mmHg)
Right Atrium	2–8 (Mean)	—
Right Ventricle	15–25	0–8
Pulmonary Artery	15–25	8–15
Wedge (PCWP)	6–12 (Mean)	—

Table 4: normal hemodynamic pressure values for the right heart and pulmonary vasculature, which are essential for assessing cardiac output and lung circulation health.

Interpretation of the Thermodilution Curve

Thermodilution is a clinical standard for assessing cardiac output (CO)(figure 6) during right heart catheterization, where the morphology of the resulting temperature-time curve provides immediate diagnostic insight. A normal or high cardiac output is characterized by a curve with a rapid upstroke and quick decay, resulting in a small area under the curve because the cold bolus is washed out of the heart rapidly. Conversely, in patients with low cardiac output or heart failure, the curve exhibits a slow upstroke and a prolonged, "broad" decay, leading to a larger area under the curve as the indicator lingers in the chambers. It is essential to monitor for artifacts such as irregular bumps, which may be caused by patient movement, respiration, or incomplete curves due to catheter contact with the vessel wall, as these can compromise the accuracy of the CO calculation.[2,3]

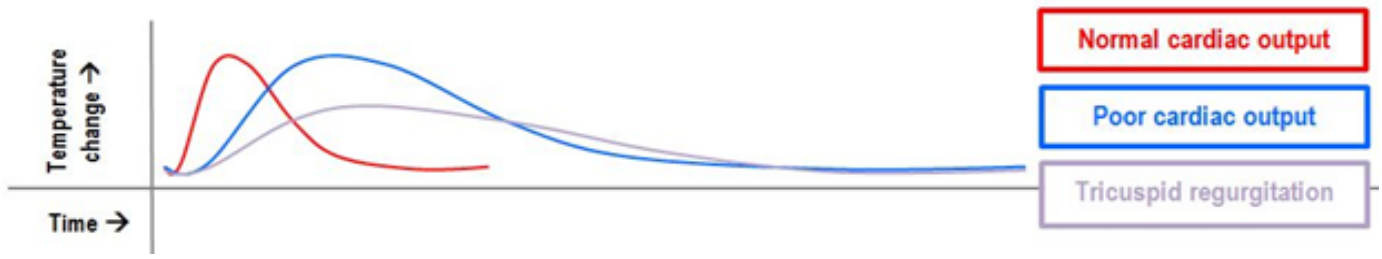


Figure 6. The thermodilution cardiac output curve illustrates how blood temperature changes over time following the injection of a cold saline bolus, with the area under the curve being inversely proportional to cardiac output. A normal curve features a rapid peak and a swift return to baseline, signifying efficient flow, whereas a poor cardiac output curve is characterized by a slower rise and a prolonged "tail," reflecting a reduced flow rate. In cases of tricuspid regurgitation, the curve appears flattened and irregular because the backflow of blood disrupts the consistent transit of the cold indicator, often leading to inaccurate or underestimated measurements of the heart's actual output.

Methodologies for Cardiac Output Calculation

In the clinical setting of right heart catheterization, cardiac output (CO) is primarily determined using two validated techniques: the Fick principle and the thermodilution method. The **Fick principle** is considered the gold standard for evaluating patients in low-output states or those with severe tricuspid regurgitation. It relies on the relationship between oxygen consumption and the arteriovenous oxygen difference, requiring simultaneous sampling of arterial blood and mixed venous blood from the pulmonary artery. Alternatively, **thermodilution**—utilizing the Stewart-Hamilton equation—is the standard clinical method due to its ease of use. This technique involves injecting a cold saline bolus into the right atrium; a distal thermistor then measures the temperature "washout" in the pulmonary artery. Because the CO is inversely proportional to the area under the resulting temperature-time curve, a larger area indicates a lower output state. To ensure physiological relevance across different body sizes, these values are often indexed to body surface area (BSA) to calculate the **Cardiac Index (CI)**, with a normal range typically falling between **2.5 and 4.0 L/min/m^2**.[2,3]

Hemodynamic Parameters for Transplant Evaluation

In the specialized context of heart transplant evaluation, derived hemodynamic parameters are essential for assessing the reversibility of pulmonary vascular changes. **Pulmonary Vascular Resistance (PVR)**, calculated by dividing the pressure gradient across the lungs (mPAP - PCWP) by the cardiac output (CO), is a critical metric; a fixed PVR exceeding **3–4 Wood Units** often serves as a contraindication for isolated heart transplantation because of the high risk of right ventricular failure in the donor organ. Additionally, clinicians utilize the **Transpulmonary Gradient (TPG)**, which represents the overall pulmonary vascular tone and should normally remain below **12 mmHg.** The **Diastolic Pulmonary Gradient (DPG)**—the difference between the pulmonary artery diastolic pressure and the wedge pressure—is also calculated as a specific marker for vascular remodeling, with a normal value typically being less than **7 mmHg**. Together, these metrics allow for a precise distinction between passive pulmonary congestion and intrinsic pulmonary vascular disease.[2,3]

Hemodynamic Classification of Pulmonary Hypertension

The hemodynamic classification of pulmonary hypertension (PH) is essential for identifying the underlying primary driver of the disease and tailoring appropriate treatment strategies. PH is defined by a mean pulmonary artery pressure (mPAP) exceeding **20 mmHg,** and is further categorized based on the pulmonary artery wedge pressure (PAWP) and pulmonary vascular resistance (PVR). **Pre-capillary PH**, often associated with pulmonary arterial hypertension (PAH) or lung disease, is characterized by a PAWP of less than **15 mmHg** and a PVR of > **2 Wood Units** (WU), indicating the pathology resides within the pulmonary vasculature. In contrast, **isolated post-capillary** PH is driven by passive congestion from left heart disease, typically presenting with a PAWP > **15 mmHg** but a normal PVR of less than **2 WU**. A third category, **combined pre- and post-capillary PH**, represents a mixed pathology where both a high wedge pressure **(> 15 mmHg)** and elevated vascular resistance **(> 2 WU)** are present, suggesting that chronic left-sided heart failure has led to secondary remodeling of the pulmonary vessels.[2,4]

Advanced Metrics for Right Ventricular Performance

Evaluation of right ventricular (RV) function is paramount in determining surgical candidacy, particularly for Left Ventricular Assist Device (LVAD) implantation. Two advanced hemodynamic indices—the Pulmonary Artery Pulsatility Index (PAPi) and the Right Ventricular Stroke Work Index (RVSWI)—serve as critical surrogates for RV performance. PAPi is calculated as the ratio of pulmonary artery pulse pressure to right atrial pressure (PAPi = {PASP - PADP}/{RAP}), where a value below **1.8–2.0** indicates a high risk for post-operative RV failure, and a value less than **1.0** signifies severe, fixed dysfunction. Complementing this, RVSWI measures the stroke work generated by the RV per heartbeat, adjusted for body size (RVSWI = (mPAP - RAP) *SI *0.0136). A low RVSWI (normal range: **7–12 g-m/m^2**) coupled with a high RAP is a profound clinical indicator of RV failure. Together, these metrics allow for a more nuanced assessment of the right heart's ability to handle hemodynamic shifts than standard pressure readings alone.[2]

Cardiac Power Output (CPO) as a Prognostic Metric

Cardiac Power Output (CPO) has emerged as the most potent hemodynamic predictor of mortality for patients presenting with cardiogenic shock. Calculated as the product of cardiac output (CO) and mean arterial pressure (MAP) divided by a constant of 451 (CPO = {CO * MAP}/{451}), this value represents the total pumping energy of the heart measured in Watts. A CPO threshold of less than **0.6 W** is highly clinically significant, as it indicates a failure of cardiac reserve and correlates with a high risk of mortality. Identifying a low CPO is a critical step in clinical decision-making, often serving as a definitive implication for the immediate need for Mechanical Circulatory Support (MCS) to stabilize the patient[5]

Hemodynamic Profiling and Tailored Therapy

The Forrester Classification(figure 7) provides a framework for hemodynamic profiling in heart failure, allowing clinicians to categorize patients into four distinct phenotypes based on their perfusion and congestion status. Patients

identified as "Wet" or congested exhibit a pulmonary capillary wedge pressure (PCWP) greater than **18 mmHg** and generally require interventions such as diuretics or vasodilators to reduce fluid overload. Conversely, those categorized as "Cold" present with a low perfusion state, defined by a cardiac index (CI) of less than **2.2 L/min/m²**, often necessitating the use of inotropes or mechanical circulatory support. The clinical objective of this tailored therapy is to transition the patient from high-risk profiles—such as "Cold and Wet"—back to the stable "Warm and Dry" (Profile A) state before considering hospital discharge or advanced options like transplantation.[6] as shown in figure table 5.

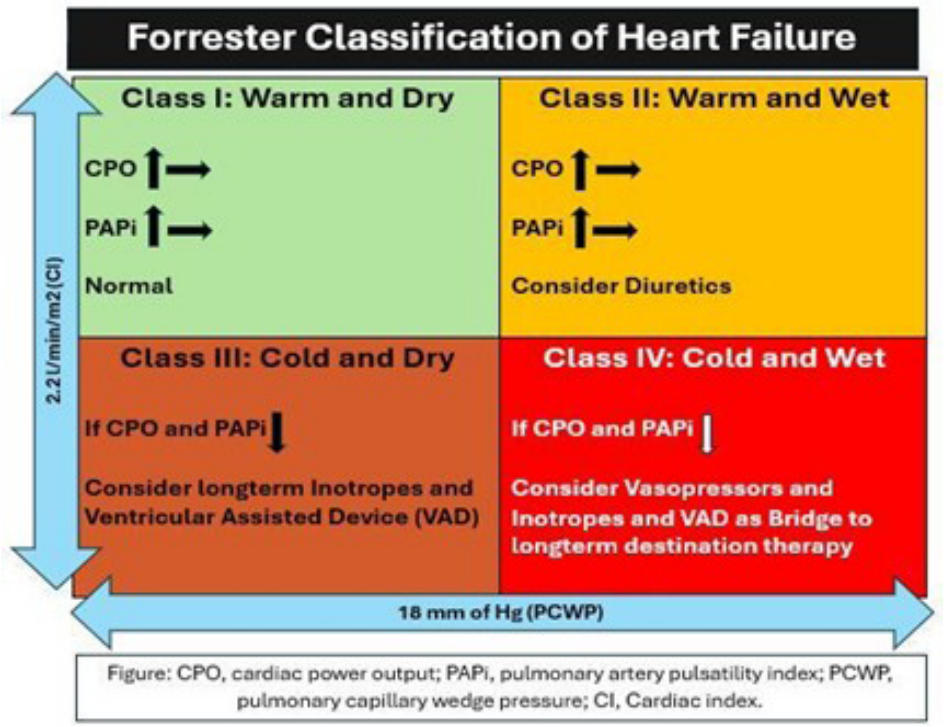


Figure 7. **Forrester Classification of Heart Failure.** This 2x2 matrix categorizes hemodynamic subsets based on Cardiac Index (CI) and Pulmonary Capillary Wedge Pressure (PCWP)

Class	Description	Hemodynamic Profile	Clinical Manifestation	Typical Treatment
I	Warm & Dry	CI > 2.2; PCWP < 18	Normal: Adequate perfusion and no congestion.	Observation or optimization of oral meds.
II	Warm & Wet	CI > 2.2; PCWP > 18	Congestion: Fluid overload in the lungs.	Diuretics and/or vasodilators.
III	Cold & Dry	CI < 2.2; PCWP < 18	Hypoperfusion: Low flow but no fluid in lungs.	Careful volume expansion (fluids).
IV	Cold & Wet	CI < 2.2; PCWP > 18	Cardiogenic Shock: Poor perfusion and congestion.	Inotropes, vasopressors, or mechanical support.

Table 5: Hemodynamic Indices,CI (Cardiac Index): Cardiac output adjusted for body surface area (L/min/m2); indicates perfusion.PCWP (Pulmonary Capillary Wedge Pressure): Indicates "wetness" or left-sided filling pressures (mmHg).

In the final stages of heart transplant candidacy evaluation, specific hemodynamic thresholds are utilized to mitigate the risk of acute right ventricular (RV) failure in the donor heart post-transplantation. High-risk profiles or contraindications include a fixed Pulmonary Vascular Resistance (PVR) greater than 5 Wood Units (WU) despite optimal medical therapy, a Transpulmonary Gradient (TPG) exceeding 15 mmHg, or a Diastolic Pulmonary Gradient (DPG) greater than 7 mmHg. Furthermore, a mean pulmonary artery pressure (mPAP) in the range of 35–40 mmHg, particularly when associated with a normal or high cardiac output, signals a significant risk for post-transplant complications. Conversely, favorable clinical outcomes are associated with a PVR of less than 3 Wood Units or hemodynamic values that demonstrate significant improvement during vasodilator testing. The primary goal of identifying these parameters is to ensure that the recipient's pulmonary vasculature can accommodate the output of the new donor heart without inducing right-sided heart failure.[2]

Vasodilator Testing in Heart Transplant Candidacy

Vasodilator testing is a mandatory component of the hemodynamic evaluation for patients being considered for orthotopic heart transplantation (OHT). The primary objective of this assessment is to establish a baseline pulmonary vascular resistance (PVR) and determine if an elevated PVR is reactive (reversible) or fixed (irreversible). Identifying a reactive PVR is clinically favorable, whereas fixed pulmonary hypertension often serves as a contraindication for transplantation due to the high risk of acute donor right heart failure. Various pharmacological agents are utilized during the procedure, with nitroprusside being the most commonly preferred systemic vasodilator. Other options include nitroglycerin, adenosine, inhaled nitric oxide at concentrations of 20–40 ppm, and intravenous milrinone, which provides both inotropic and vasodilatory effects. Additionally, 100% oxygen may be administered specifically to patients whose pulmonary hypertension is induced by hypoxia.[1,2]

To ensure clinical precision, the **protocol for vasodilator testing** during right heart catheterization follows a structured, sequential approach aimed at evaluating pulmonary vascular reactivity. The process begins with recording a complete **baseline hemodynamic set**, including right atrial (RA) pressure, pulmonary artery (PA) pressure, pulmonary capillary wedge pressure (PCWP), cardiac output (CO), and mean arterial pressure (MAP). A vasodilator—most commonly **nitroprusside** at 0.3–0.5 mcg/kg/min or **inhaled nitric oxide (iNO)** at 20 ppm—is then initiated at a low dose and carefully titrated every 5–10 minutes. During this phase, continuous monitoring of mPAP, PCWP, and CO is essential to evaluate efficacy, while MAP is tracked as a safety measure to prevent systemic hypotension. Titration is concluded once specific PVR criteria are achieved, if hypotension occurs, or if no further hemodynamic improvement is observed. The protocol culminates in a final assessment, repeating the full hemodynamic set at the maximum tolerated dose to calculate the final PVR and determine transplant eligibility.[2]

To confirm successful pulmonary vascular resistance (PVR) reversibility during vasodilator testing, specific hemodynamic criteria must be met to classify a patient as having reactive pulmonary hypertension. The primary goal is to observe a significant reduction in PVR, defined as a final value of less than 3 Wood units or a relative decrease of more than 20–30% from the patient's recorded baseline. Throughout this titration process, systemic safety must be prioritized by maintaining a systolic blood pressure above 85–90 mmHg. **A favorable response** profile is characterized by a simultaneous decline in mean pulmonary artery pressure (PAP), transpulmonary gradient (TPG), and PVR, all while preserving or improving the cardiac output or cardiac index. Achieving these targets indicates that the pulmonary hypertension is predominantly reactive rather than fixed, which is a critical factor in determining suitability for advanced therapies like heart transplantation.

Clinical decision-making regarding pulmonary hypertension (PH) reversibility is a primary determinant of surgical candidacy for advanced heart failure therapies. Patients exhibiting a **favorable response**, characterized by a pulmonary vascular resistance (PVR) falling below 3 Wood Units (WU) or showing significant reduction, are cleared to proceed with orthotopic heart transplantation (OHT) alongside medical optimization. In **borderline cases**, where PVR remains between 3–5 WU but demonstrates a 20–30% decrease, a Left Ventricular Assist Device (LVAD) may be utilized as a "bridge to transplant" to allow for secondary remodeling of the pulmonary vasculature, with a planned re-evaluation via right heart catheterization after 3–6 months. Conversely, **irreversible or fixed PH**, defined by a PVR exceeding 5 WU despite maximal vasodilator challenge, serves as a contraindication for isolated OHT, necessitating alternatives such as heart-lung transplantation or a transition to palliative care.[2]

While right heart catheterization remains the invasive gold standard, non-invasive echocardiography provides a critical screening tool for estimating hemodynamic parameters using validated formulas. Left atrial pressure, or pulmonary capillary wedge pressure (PCWP), can be estimated via Tissue Doppler Imaging (TDI) using the Nagueh formula: PCWP = 1.24 * (E/e') + 1.9, where an E/e' ratio greater than 15 suggests elevated filling pressures. Similarly, mean pulmonary artery pressure (mPAP) is derived by first calculating systolic PAP (4v2 + RAP) and then applying the Chemla equation: mPAP = 0.61 * SPAP + 2. Advanced metrics like pulmonary vascular resistance (PVR) can be approximated using the Abbas formula, which relates the maximum tricuspid regurgitation velocity (TRV{max}) to the velocity-time integral of the right ventricular outflow tract (VTI_{RVOT}). These non-invasive assessments, including the calculation of the transpulmonary gradient (TPG = mPAP - PCWP), allow for preliminary hemodynamic profiling and the identification of patients who require further invasive evaluation[2,7,8,9,10]

To ensure the accuracy of a right heart catheterization (RHC), clinicians must be vigilant against common interpretation pitfalls that can skew hemodynamic data. One significant technical error is over-wedging, which occurs when the balloon is inflated too far into the pulmonary capillary, potentially leading to a falsely elevated pulmonary capillary

wedge pressure (PCWP) and an artificially low pulmonary vascular resistance (PVR). Furthermore, the patient's volume status must be optimized to "dry weight" prior to the procedure to establish reliable baseline pressures. In low cardiac output states, high PVR values may actually underestimate the true severity of pulmonary hypertension, necessitating a repeat RHC after the initiation of inotropic support. Identifying high-risk indicators is equally crucial; advanced right ventricular (RV) failure—marked by a cardiac index below 2.0 and sharply rising right atrial pressures—suggests poor tolerance for major interventions. Ultimately, chronic and severe fixed pulmonary hypertension carries a near-fatal risk of acute RV failure in a donor heart, as the new organ may be unable to tolerate the sudden afterload demands.[1]

In the context of severe mitral regurgitation (MR) (figure 8), right heart catheterization reveals a pathognomonic finding known as the giant 'v' wave on the pulmonary capillary wedge pressure (PCWP) tracing. This occurs during ventricular systole when the incompetent mitral valve allows high-pressure blood from the left ventricle to eject backward into the left atrium, causing a massive pressure spike. A critical diagnostic challenge is distinguishing this giant 'v' wave from a pulmonary artery (PA) systolic wave, as both can appear morphologically similar. Precise identification is achieved by correlating the pressure trace with an electrocardiogram (ECG); the pathological 'v' wave typically occurs in late systole, appearing after the T wave, whereas a standard PA systolic wave generally aligns with the T wave.[12]

Right heart catheterization is a definitive diagnostic tool for identifying constrictive pericarditis(figure 9), typically characterized by the "square root" sign (also known as the dip-and-plateau sign). This phenomenon occurs during diastole when an initial rapid drop in pressure is followed by an abrupt rise and plateau because the rigid pericardial shell prematurely limits ventricular filling. A hallmark of this condition is diastolic equalization, where the restricted filling causes the mean right atrial (RA), right ventricular (RV) diastolic, pulmonary artery (PA) diastolic, and pulmonary capillary wedge pressure (PCWP) to converge at a similar level. Furthermore, enhanced respiratory variation—where the pressures in the left and right ventricles move in opposite directions during the respiratory cycle (ventricular discordance)—serves as a key physiological feature to distinguish constriction from restrictive cardiomyopathy.[12]

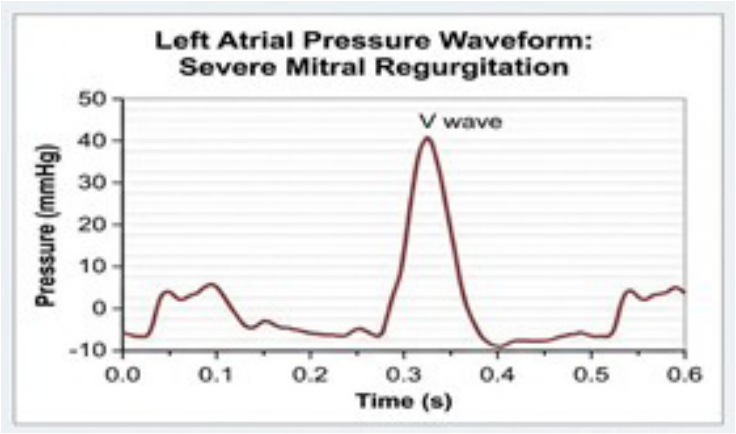


Figure 8. Left Atrial Pressure Waveform in Severe Mitral Regurgitation. The tracing demonstrates the hemodynamic impact of valvular incompetence on atrial pressures.

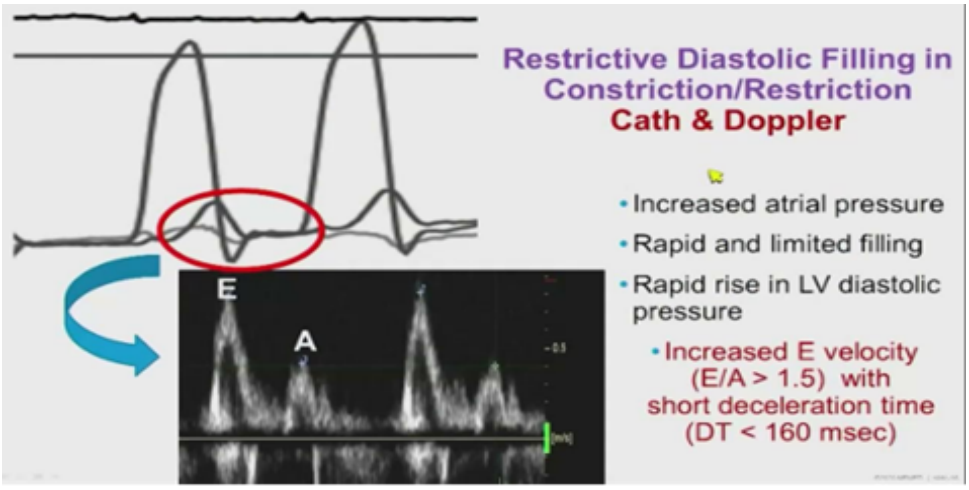


Figure 9. Hemodynamic and Doppler Characteristics of Restrictive Diastolic Filling. The composite image illustrates the physiological hallmarks of restrictive or constrictive cardiomyopathy.Doppler Echocardiography (Bottom): Shows a restrictive filling pattern with an increased E velocity and a significantly reduced A velocity (E/A ratio > 1.5).Clinical Indicators: Note the short deceleration time (DT < 160 msec), indicating an abrupt cessation of flow as the stiff ventricle reaches its elastic limit early in diastole.

Conclusion

RHC remains the definitive modality for hemodynamic assessment in advanced heart failure, pulmonary hypertension, and heart transplant evaluation, providing information that cannot be replicated by non-invasive imaging alone. A structured approach to waveform interpretation, calculation of PVR and gradients, integration of advanced RV indices, and standardized vasodilator testing is essential to minimize transplant risk and optimize use of mechanical support. Echo-based formulas offer useful screening surrogates but should complement rather than replace invasive measurements when irreversible PH and post-transplant RV failure are major concerns.

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