Consensus Document

Executive Summary of the SCAI/HFSA Clinical Expert Consensus Document on the Use of Invasive Hemodynamics for the Diagnosis and Management of Cardiovascular Disease

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Scope of Document

Modern insight into cardiovascular pathophysiology first emerged through detailed invasive hemodynamic studies performed in the cardiac catheterization laboratory. Understanding the evolution of these concepts and their clinical utility remain fundamental to practice. Although noninvasive methods are now routinely used, invasive catheter-based assessments remain the gold standard for hemodynamic evaluation of cardiovascular disease.

On behalf of the Heart Failure Society of America (HFSA) and the Society for Cardiovascular Angiography and Interventions (SCAI), we recently published a consensus document on this topic [1]. We defined clinically relevant hemodynamic concepts and their invasive assessment; described clinical scenarios in which invasive hemodynamics should be strongly considered; and emphasized the need for maintenance of quality and continuous professional development. Below is an executive summary of this document that highlights clinical scenarios and recommendations, and in particular those that involve the management of patients with heart failure.

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Clinical Scenarios and Recommendations

Governing Principles

Following review of clinical and noninvasive evaluations, the decision to pursue hemodynamic evaluation in the cardiac catheterization laboratory should be based on the incremental utility of data that will be derived from the procedure. To understand this utility, it is necessary to consider the details of proper invasive assessment, its limitations, and how these data will either complement, or in some cases, supplant the current clinical assessment based on noninvasive testing (Table I). Operators skilled in safely obtaining and immediately interpreting accurate hemodynamic data should perform invasive hemodynamic evaluations. A summary of the most common indications for invasive hemodynamic assessment separated by clinical indications follows.

Determination of Etiology of Dyspnea

The most commonly assessed index of diastolic function is ventricular filling pressure—left ventricular end-diastolic pressure (LVEDP) or pulmonary capillary wedge pressure (PCWP). Although these indices are critically important in clinical care, they are not direct measures of diastolic function. For example, LVEDP might be elevated because of prolonged relaxation, elevated passive chamber stiffness, or external restraint from pericardial constriction. Nonetheless, an elevation in filling pressures is typically related to diastolic dys-function and should prompt further consideration in terms of etiology. The presence of a markedly elevated LVEDP is generally suggestive of increased LV diastolic stiffness, with an important exception being patients with simultaneous elevation in right heart filling pressure (e.g., with severe tricuspid regurgitation or right heart failure) [2]. In this circumstance, left heart pressures are elevated because of enhanced ventricular interdependence in a manner analogous to pericardial constriction.

A common indication for right heart catheterization is to determine whether exercise intolerance (exertional dyspnea

Table I. Key Clinical Recommendations for Invasive Hemodynamic Evaluations

Determination of etiology of dyspnea
1. In patients presenting with exercise intolerance, in which noninvasive and resting invasive measurements are inconclusive, provocative testing in the cardiac catheterization laboratory should be considered to determine the presence or absence of heart failure as the cause of dyspnea.
2. Cycle ergometry exercise is the most physiologically relevant and sensitive stressor and is preferred over other provocative maneuvers such as saline loading or arm exercise.

Pulmonary hypertension
1. Invasive assessment of pulmonary hemodynamics is required for patients with pulmonary hypertension who are being considered for pulmonary selective vasodilator therapy.
2. Invasive assessment of pulmonary hemodynamics should be considered when there is diagnostic uncertainty regarding pulmonary hypertension based on noninvasive data. This assessment should establish the diagnosis according to WHO classification.
3. Invasive assessment of pulmonary hemodynamics should be performed to monitor and assess the effectiveness of pulmonary hypertension therapies.

Advanced heart failure
1. Invasive assessment of pulmonary hemodynamics with or without vasodilator challenge is required for patients being considered for cardiac transplantation or inotrope therapy.
2. Invasive assessment of pulmonary hemodynamics can be used to assess the risk of right ventricular failure with advanced heart failure therapies.
3. Invasive assessment of pulmonary hemodynamics may be considered to define volume status when unclear based upon noninvasive assessments, particularly in the setting of cardiorenal syndrome.

Valvular heart disease
1. An invasive hemodynamic evaluation with or without provocative maneuvers is recommended to resolve discrepancies between clinical findings and noninvasive imaging data in patients with valvular disease when surgical or catheter-based therapy is being considered.
2. Invasive hemodynamic studies of patients with valvular disease should be performed with simultaneous measurement of multiple central cardiac chambers.
3. Invasive hemodynamic evaluations are beneficial for patients with valvular regurgitation in certain scenarios, such as eccentric jets with difficult quantitation, prosthetic valves with possible acoustic shadowing, and acute lesions in which color flow Doppler might be limited.

Pericardial disease
1. An invasive hemodynamic evaluation should be strongly considered for all patients with suspected constrictive pericarditis because of the frequently complex pathophysiology and the need for high diagnostic specificity when considering surgery.
2. Invasive studies for constrictive pericarditis should entail examination of the dynamic respiratory criteria.
3. An invasive hemodynamic study is typically not required for the diagnosis of cardiac tamponade.

Congenital heart disease
1. Cardiac catheterization should be performed for patients with shunts when there is evidence of elevated pressures, chamber enlargement, or symptoms that are out of proportion to the size of the congenital lesion, and prior to closure of shunts.
2. Cardiac catheterization should be performed to assess the hemodynamics of patients with congenital heart disease with known or suspected right ventricular failure, especially in palliated single ventricle physiology.
3. Cardiac catheterization should be performed to determine the severity of obstructions in series.

Cardiogenic shock and mechanical circulatory support
1. Invasive hemodynamic assessment, with measurement of ventricular filling pressures, cardiac output, and systemic vascular resistance, is recommended for the diagnosis of cardiogenic shock.
2. Continuous hemodynamic monitoring with a pulmonary artery catheter is recommended for acute management of patients receiving therapy with mechanical circulatory support.
3. Pulmonary artery catheterization is useful to guide withdrawal of mechanical circulatory and pharmacologic support in patients with myocardial recovery from cardiogenic shock.
4. In patients without recovery of myocardial and end-organ function, hemodynamic monitoring is useful to assess candidacy for and transition to advanced heart failure therapies, including durable mechanical circulatory support and heart transplantation.

and/or fatigue) has a cardiac or noncardiac etiology. Many patients with heart failure (HF) display normal physical findings, normal natriuretic peptide levels, normal echocardiographic indices, and even normal filling pressures at rest, but marked elevation in PCWP during exercise [3,4]. Many of these patients also present impairments in cardiac output (CO) reserve with exercise, despite normal CO at rest [5,6]. Exercise echocardiography may be useful in some patients when high quality images can be obtained, but is often limited or equivocal. Invasive hemodynamic cardiopulmonary exercise testing is the gold standard to identify or exclude HF as the cause of exercise intolerance in these patients. Leg exercise presents much greater hemodynamic stress and is preferable to upper extremity exercise. Saline loading can be considered if exercise cannot be performed, but provides less diagnostic information and is not recommended to identify or exclude heart failure [7]. When available, high-fidelity micromanometers can be used to obtain an accurate examination of pressure contours, which makes interpretation of central pressures during exercise far more accurate [4].

Pulmonary Hypertension

Pulmonary hypertension (PH) is a common risk factor for various cardiovascular outcomes [8,9]. PH can occur passively as a sole consequence of elevated PCWP (i.e., pulmonary venous hypertension or postcapillary PH) or as a manifestation of pulmonary vascular disease. The latter is characterized by the presence of a normal left atrial pressure and markedly elevated pulmonary vascular resistance (PVR), i.e., pulmonary arterial hypertension (PAH) or precapillary PH. "Mixed" PH or PH "out of proportion to left heart disease" are terms used to describe conditions in which both PCWP and PVR are elevated [10]. This frequently occurs as a result of chronic left-sided congestion (e.g., mitral valve disease), which leads to remodeling of the pulmonary arterial circulation. Although noninvasive assessments reliably screen for PH and its consequences (e.g., right ventricular dysfunction), they are limited in specificity for World Health Organization (WHO) classification and guiding therapy. Thus, invasive assessment is key to managing all patients with PH.

The differentiation of pulmonary venous hypertension and PAH is critically important because of their distinctive prognoses and therapeutic strategies, and relies upon accurate measurement of the PCWP, which is normal in PAH and elevated in pulmonary venous hypertension. Most current guidelines define a normal PCWP as ≤ 15 mm Hg, though others have advocated for a somewhat higher partition value of <18 mm Hg. All PCWP measurements should be made at end-expiration, mid-A wave, and confirmed with oximetry, fluoroscopy, and typical waveforms. When equivocal, direct left atrial pressure can be considered. If there is suspicion of left heart disease in the setting of a normal PCWP, direct measurement of LVEDP can help clarify the underlying problem.

Assessments in Advanced Heart Failure

For patients with HF and elevated PVR, particularly those being considered for surgical therapy or heart transplantation, reversibility should be assessed in the catheterization laboratory. While multiple agents can be used for this purpose, caution should be exercised with selective vasodilators, especially with inhaled agents (e.g., nitric oxide or epoprostenol), because of the small risk of pulmonary edema when increases in pulmonary flow overcome impaired left-sided compliance. In general, pulmonary-specific vasodilators should be avoided in patients with PCWP >25 mm Hg; in these cases an agent that unloads both the pulmonary vasculature and left heart, such as nitroprusside or milrinone, is preferred [9].

Invasive measurement also allows the calculation of novel hemodynamic parameters that can be used to predict RV failure with advanced therapies such as heart transplantation and left ventricular assist device implantation, including RV diastolic and systolic reserve, RV stroke work index, and pulmonary artery pulsatility index [11]. Another scenario that arises is the patient with unclear volume status and worsening renal function, where direct assessment of left and right heart filling pressures provides direction in the aggressiveness of further volume reducing therapies, either pharmacologic or using renal replacement therapy. Finally, measures of CO and systemic vascular resistance may be used to direct choice of inotropic and/or vasodilator therapy to optimize hemodynamics. As per HFSA guidelines, routine use of a pulmonary artery catheter to guide management of acute heart failure is not indicated [12,13].

Valvular Heart Disease

For patients with valvular heart disease, an invasive hemodynamic evaluation is recommended to resolve discrepancies between clinical findings and noninvasive imaging data when surgical or catheter-based therapy is being considered. In this setting, simultaneous hemodynamic study of multiple *central* cardiac chambers should be performed. Peripheral pressures should not be used because of problems related to phase delay, damping, and pressure amplification. Dynamic study, with either pharmacologic or exercise provocation, should be considered in low flow states and when resting hemodynamics are not diagnostic of exertional symptoms.

For most patients with valvular stenosis, a Doppler echocardiogram provides the necessary data for clinical decision-making; however, there are limitations to echocardiography. These limitations primarily arise from the angle-dependent nature of Doppler echocardiographic techniques, as well as geometrical assumptions in regard to flow assessment. Diagnostic quality echocardiographic windows cannot be obtained in all patients. When low-flow, low-gradient aortic stenosis is present with hypertension, a hemodynamic study with lowering of systemic blood pressure should be considered, since high aortic impedance can mask the presence of a significant gradient [14].

For patients with valvular regurgitation, invasive hemodynamic assessment can be advantageous in several clinical scenarios, including the presence of eccentric jets (which can be difficult to quantitate), acute regurgitant lesions (in which rapid pressure equalization can limit color flow Doppler assessment), and prosthetic valves (in which acoustic shadowing can mask the regurgitant jet). Studies in the cardiac catheterization laboratory overcome spatial limitations, such that absolute elevations in pressure accurately reflect the pathophysiology and overall hemodynamic burden of these lesions. Exercise hemodynamics may be diagnostic of severe ischemic mitral regurgitation in patients with normal PCWP at rest but marked elevation in PCWP with V waves during exercise [15].

Pericardial Disease

Pericardial disease can be challenging to diagnose because of nonspecific symptoms and signs as well as the frequent presence of coexistent lesions (e.g. valvular, myocardial, and pericardial lesions in radiation-induced heart disease). For patients with possible constrictive pericarditis, a high specificity in the diagnosis is required, given that pericardiectomy, though often life-saving, is a complex procedure that carries risk. Thus, invasive hemodynamic studies should be strongly considered in the assessment of all patients suspected of having constrictive pericarditis.

A high-quality study entails simultaneous assessment of right- and left-sided chambers for examination of ventricular interdependence and dissociation of intracavitary and intrathoracic pressures. These evaluations must be performed with elevated filling pressures and during dynamic respiration. Although traditional hemo-dynamic criteria have been used (e.g., LVEDP-RVEDP ≤ 5 mm Hg), they lack sensitivity and specificity [16]. Invasive examination of reciprocal changes in LV and RV systolic pressure during respiration, which is reflective of enhanced ventricular interdependence, is the most effective diagnostic test when evaluating constriction. Using simultaneous PCWP and LV pressure, the presence of intracavitary–intrathoracic pressure dissociation should also be studied. As in exercise studies, high-fidelity micromanometers (if available) are preferred to enable accurate examination of pressure contours when evaluating for pericardial constriction [16,17].

Congenital Heart Disease

For most patients with shunt lesions, echocardiography with careful examination of the gradients and chamber morphology is sufficient to guide clinical decision-making [18]. Cardiac catheterization is indicated when the gradients suggest elevated pressures, when chamber enlargement and symptoms are out of proportion to the size of the congenital lesion, when new symptoms or findings are not explained by noninvasive testing, and in patients prior to transcatheter or surgical closure of shunts. The goals of these evaluations are to quantify the degree of shunting, to determine the presence of unrecognized lesions, and to evaluate the severity of PH. The finding of an elevated PVR warrants further evaluation with vasoreactivity testing since a calculation of resistance is critical for clinical decision-making. Of note, atrial level shunting is affected by ventricular compliance and the potential presence of atrioventricular valve stenosis. During cardiac catheterization, care should be taken to minimize contamination during oxygen sampling proximal and distal to the lesion.

Adults with congenital heart disease who are most susceptible to heart failure and most likely to benefit from invasive assessment are those with systemic right ventricles and Fontan patients with single ventricle physiology. For example, patients with Fontan baffle obstruction will not generate a significant pressure gradient because of the compliance of the systemic veins and pulmonary vasculature. In addition, many Fontan patients have impaired ventricular function. The invasive assessment of systolic and diastolic dysfunction and their potential etiologies (e.g., chronic volume or pressure overload, prior surgery, cyanosis) remains essential to providing appropriate therapy [19,20] Similarly, assessing the hemodynamic significance of obstructions in series is best performed using invasive techniques.

Cardiogenic Shock and Mechanical Circulatory Support

In the setting of cardiogenic shock, LV contractility and stroke volume are severely reduced, whereas LV enddiastolic volume and arterial afterload are increased to compensate for poor output [21,22]. The net result of increased LV volumes is an increase in overall pressure–volume area (PVA), which correlates directly with increased myocardial oxygen demand [23] This may cause ischemia, trigger arrhythmias, and worsen myocardial damage. For all patients with suspected cardiogenic shock, an invasive hemodynamic assessment is recommended for diagnosis and to expedite therapy [21,24]. Pharmacologic therapy with positive inotropes and vasopressors increases cardiac output and systemic arterial pressure to support end-organ perfusion, but may increase PVA and thus myocardial oxygen demand. Conversely, mechanical circulatory support devices replace native cardiac output, reduce ventricular volume, and increase systemic arterial pressure, and in turn reduce myocardial oxygen demand.

Mechanical circulatory support devices have varying effects on ventricular load, wall stress, and ventricular function [21,25]. Intra-aortic balloon pumps (IABP) augment pulsatile blood flow by inflating during diastole to partially augment coronary perfusion [26]. During systole, IABP deflation can reduce LV wall stress by 20–25%. Roto-dynamic pumps, such as the Impella (Abiomed) and TandemHeart (CardiacAssist), generate continuous, minimally pulsatile blood flow with the net effects of reducing LV volume and pressure while increasing mean arterial pressure without greatly influencing ventricular afterload [21,27]. Other centrifugal pumps, such as the CentriMag (Thoratec), ROTAFLOW (Maquet), and Biomedicus (Medtronic), are more commonly implanted surgically or used for veno-arterial extracorporeal membrane oxygenation (VA-ECMO) [28].

Summary

As healthcare continues to transition from volume to value-based care, it is imperative that professional societies articulate practice standards and expectations for providers to maintain competency and deliver the highest quality care. We believe this executive summary addresses these issues as it pertains to the performance of invasive hemodynamics in patients with heart disease with an emphasis on heart failure. By having such standards, cardiovascular providers, payers, and patients will all benefit by reducing the variability in the use of invasive hemodynamics in cardiovascular disease.

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