

Invasive Hemodynamics of Myocardial Disease

Systolic and Diastolic Dysfunction (and Hypertrophic Obstructive Cardiomyopathy)

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KEYWORDS

- Hemodynamics • Congestive heart failure • Hypertrophic cardiomyopathy • Ischemia
- Infarction

KEY POINTS

- Heart failure is a clinical diagnosis that is supported by various laboratory, imaging, and invasive hemodynamic measures. There is no single diagnostic test.
- A variety of structural and/or functional myocardial abnormalities can lead to the inability of the heart to fill or eject blood.
- Invasive hemodynamic assessment remains of great importance in the evaluation of patients with myocardial disease or hypertrophic cardiomyopathy.
- Several different hemodynamic measures are available to aid in diagnosing systolic and diastolic dysfunction.
- A deep understanding of pressure-volume loops and hemodynamic tracings is necessary in order to appreciate variation in pathology of myocardial disease.

INTRODUCTION

Invasive assessment of cardiovascular function provides greater insight into the mechanisms of disease not only in classic heart failure due to dilated cardiomyopathy and cardiogenic shock but also in disorders, such as heart failure with preserved ejection fraction (HFpEF), and may explain how patients who have different forms of heart failure respond to various therapies. From the cardiac catheterization laboratory data, hemodynamic pressure waveforms, in addition to echocardiographic information,

provide data on left and right heart function, systemic and pulmonary arterial resistances, and cardiac-vascular coupling relationships. These data form therapeutic parameters, such as cardiac preload, afterload, and global left ventricular (LV) function to guide and gauge the efficacy of both medical and mechanical interventions.

ASSESSMENT OF CARDIAC CONTRACTILITY

The major manifestation of the diseased myocardium is the failure to produce normal contraction

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and in some cases normal relaxation. Systolic contractile effort can be characterized by both global and regional myocardial function as measured by the extent of muscle shortening or thickening producing ejection of blood.^{1,2} The ejection of blood from the ventricle can be quantitated by global ejection fraction (EF), segmental LV wall motion, or cardiac output or cardiac work. Contractile ejection is highly sensitive to afterload and really is an expression of ventricular ejection into the arterial resistance, a phenomenon known as vasculo-ventricular coupling. EF is also affected by heart size. Because the denominator of LVEF is end-diastolic volume (EDV), the EF value is more a parameter of remodeling than of contractility. Unfortunately, for simplicity, EF is used as a more binary labeling of myocardial diseases into those with normal versus reduced EF. In reality, the causes and presentation of patients with heart failure is much more complex.

THE PRESSURE-VOLUME LOOP AND LEFT VENTRICULAR FUNCTION

Parameters of cardiac function can be derived from relations between cardiac pressure and volume. Pressure-volume (PV) loops can be obtained invasively to assess peak systolic and end-diastolic pressures, stroke work, maximal rate of pressure increase during isovolumetric contraction (dP/dt_{max}), maximal ventricular power (PWR_{max}), elastance, efficiency, and other unique parameters (Fig. 1). By examining the PV loop, myocardial function can be viewed over a range of preload volumes to determine load-independent, cardiac-specific measures.³

LV hemodynamics can be represented by a PV loop, which plots the changes of these variables over a cardiac cycle.⁴⁻⁶ Each PV loop represents one cardiac cycle (see Fig. 1). Beginning at end-diastole (point a), LV volume has received the atrial contribution and is maximal. Isovolumetric contraction (a to b) increases LV pressure with no change in volume. At the end of isovolumetric contraction, LV pressure exceeds aortic pressure, the aortic valve opens, and blood is ejected from the LV into the aorta (point b). Over the systolic ejection phase, LV volume decreases; as ventricular repolarization occurs, LV ejection ceases and relaxation begins. When LV pressure decreases to less than the aortic pressure, the aortic valve closes, a point also known as the end-systolic PV point (ESPV) (point c). Isovolumetric relaxation occurs until LV pressure decreases to less than the atrial pressure, opening the mitral valve (point d).

The stroke volume (SV) is represented by the width of the PV loop, the difference between end-systolic volume (ESV) and EDV. The area within the loop represents stroke work. Load-independent LV contractility, also known as E_{max} , is defined as the maximal slope of the ESPV points under various loading conditions, the line of these points is the ESPV relationship (ESPVR). Effective arterial elastance (E_a), a measure of LV afterload, is defined as the ratio of end-systolic pressure to SV. Under steady-state conditions, optimal LV contractile efficiency occurs when the ratio of $E_a:E_{max}$ approaches 1.

CONTRACTILITY, MYOCARDIAL WORK, AND CARDIAC POWER OUTPUT

Contractility is an intrinsic property of cardiac tissue that determines strength of contraction in response to ventricular load and reflects the level of activation, formation, and cycling of actin and myosin cross-bridges. At a constant preload and afterload, increased contractility results in increased extent and velocity of shortening (Fig. 2A). Contractility is affected by the adrenergic state, drugs, and myocyte injury or loss. Although an increase in heart rate leads to an increase in contractility in normal myocardium (positive force-frequency relationship), this relationship is blunted or even negative in failing myocardium. The most commonly used surrogate variables of contractility, such as EF and peak rate of increase of ventricular pressure ($+dP/dt$), are load dependent. The use of the end-systolic PV relationship to determine end-systolic elastance (E_{es}) has been suggested as a load-independent method of assessing myocardial contractility (Fig. 2B–D).

dP/dt_{max} can be assessed using a high-fidelity micromanometer and is used widely as a measure of contractility. dP/dt_{max} , however, depends on cardiac filling (ie, is preload dependent) and heart rate; it may not always reflect contractile function that develops after cardiac ejection is initiated.² The relationship between end-systolic pressure and volume from a variety of variably loaded cardiac contractions yields the ESPVR.⁷ This slope provides the E_{es} . The E_{es} may then provide further information regarding contractile function and chamber stiffness.

The relationship between stroke work and preload, measured from multiple beats under different loading conditions, generates a Sarnoff curve; its slope, often termed *preload recruitable stroke work*, provides a contractile index

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