Invasive Hemodynamic Assessment in Heart Failure

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KEYWORDS

• Hemodynamics • Heart failure • Systole • Diastole
• Ventricular-arterial interaction • Cardiovascular function

The concept of hemodynamics was born in 1628 with Harvey's description of the circulation, but its growth was limited for centuries by the inability to measure pressure and flow accurately. The work of Starling, Wiggers, and other hemodynamic physiologists in the first part of the twentieth century, along with introduction of cardiac catheterization by Cournand and Richards in the 1940s ushered in the golden era of hemodynamics. From the late 1970s to early 1990s, there was an explosion of clinical and basic research as new methods were developed to quantify ventricular systolic and diastolic properties more definitively. Enthusiasm for such characterization subsequently waned, however, as therapies directly targeting hemodynamic derangements, such as inotropes, were found to hasten mortality. This change coincided with a paradigm shift in the way heart failure was conceptualized, from a disease of abnormal hemodynamics to one of neurohormonal derangements, abnormal cell signaling, and maladaptive remodeling. As such, many "gold-standard" methods for characterizing load, contractility, diastole, and, ventricular-arterial interaction were not adopted into clinical practice. A working understanding of each element remains paramount to interpret properly the hemodynamic changes in patients who have acute and chronic heart failure, however.

Routine cardiac catheterization provides data on left heart, right heart, systemic and pulmonary arterial pressures, vascular resistances, cardiac output, and ejection fraction. These data are often then applied as markers of cardiac preload, afterload, and global function, although each of these parameters reflects more complex interactions between the heart and its internal and external loads. This article reviews more specific, gold standard assessments of ventricular and arterial properties and how these relate to the parameters reported and used in practice, and then discusses the re-emerging importance of invasive hemodynamics in the assessment and management of heart failure.

CARDIAC CONTRACTILITY: LOOKING BEYOND THE EJECTION FRACTION

The most universally accepted index of contractility used in practice, the EF, unfortunately is also one of the least specific.[1] As with any parameter measuring the extent of muscle shortening or thickening, it is highly sensitive to afterload and really is an expression of ventricular–arterial coupling rather than of contractility alone. EF also is affected by heart size, because its denominator is end-diastolic volume (EDV), leading many to propose that EF is more a parameter of remodeling than of contractility. EF commonly is used to classify different "forms" of heart failure (low versus preserved EF).[2] This approach is appealing, given its binary nature and ease of application in practice, but the realities of how a patient develops signs and symptoms of heart failure are far more complex,[3] and in this regard EF serves as a somewhat arbitrary marker.

More specific measures of contractility have been developed but because of their complexity

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Heart Failure Clin 5 (2009) 217–228
doi:10.1016/j.hfc.2008.11.008
1551-7136/08/$ – see front matter

remain used principally in research. The maximal rate of pressure rise during isovolumic contraction (dP/dt_{max}) can be assessed using a high-fidelity micromanometer and is used widely as a measure of contractility. dP/dt_{max}, however, is dependent on cardiac filling (ie, is preload dependent) and heart rate, and it may not always reflect contractile function that develops after cardiac ejection is initiated.[4] In patients who have cardiac dyssynchrony, the lack of coordinated contraction in early-systole reduces dP/dt_{max} because the force developed by the early activated wall is dissipated by stretching of the still relaxed opposite wall.[5] dP/dt_{max} is quite sensitive to this phenomenon (**Fig. 1**), but in this case it reflects chamber mechanics rather than intrinsic muscle function.

An ideal parameter of contractility would assess inotropic state independently of preload, afterload, heart rate, and remodeling.[1] This assessment still remains somewhat elusive, but parameters derived from relations between cardiac pressure and volume have come the closest to achieving it. As shown in **Fig. 1**B, a series of variably loaded pressure–volume (PV) loops can be obtained to assess systolic, diastolic, coupling, and energetic properties. Stroke work, dP/dt_{max}, maximal ventricular power, elastance, efficiency, and other parameters are assessed, and by examining these variables over a range of preload volumes, one can derive more load-independent, cardiac-specific measures.[6]

The relationship between end-systolic pressure and volume from a variety of variably loaded cardiac contractions yields the end-systolic pressure–volume relationship (ESPVR),[7] its slope being the end-systolic elastance (Ees) (see **Fig. 1**B). The Ees conveys information about both contractile function and myocardial

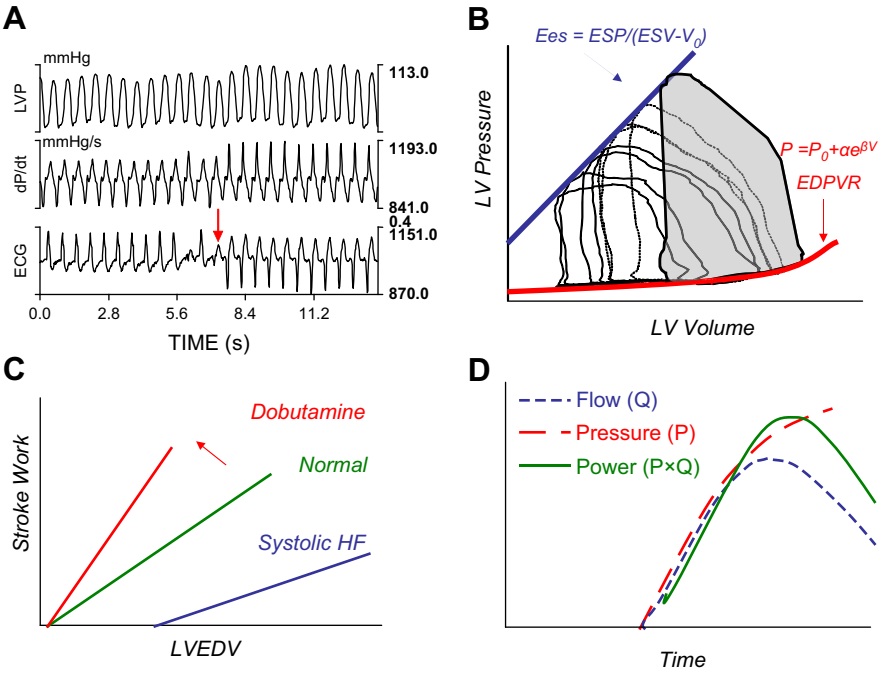

Fig. 1. (*A*) Time plot of left ventricular pressure (LVP), first derivative of pressure (dP/dt), and EKG in a patient who has heart failure with left bundle-type conduction delay. At the arrow, the patient received bi-ventricular stimulation resulting in an abrupt rise in dP/dt_{max}. (*B*) PV loops obtained at baseline and during transient caval occlusion (decreasing LV volumes—loops moving right to left). The slope of the EDSPV derived from multibeat analysis defines ventricular Ees, a load-independent measure of contractility. By measuring diastolic pressure and volume during diastasis at variably loaded beats, the end-diastolic PV relationship (EDPVR) is obtained. The shaded area subtended by the baseline loop represents the stroke work performed by the ventricle. ESP, end-systolic pressure; ESV, end-systolic volume; V_0, volume axis intercept of ESPVR. (*C*) The slope of the relation between systolic chamber performance (stroke work) and preload (left ventricle end-diastolic volume, LVEDV) determines the preload recruitable stroke work. This relationship shifts up and to the left, as indicated by the arrow, with an increase in contractility, as with dobutamine, or down and to the right with systolic heart failure (HF). (*D*) LV power ($P \times Q$, *solid line*) is determined by the product of simultaneously measured pressure (P, *dashed line*) and flow (Q, *dotted line*). When indexed to preload, this calculation produces another load-independent measure of LV chamber contractility.