

## Last Word on Point:Counterpoint: Left ventricular volume during diastasis is the physiological in vivo equilibrium volume and is related to diastolic suction

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TO THE EDITOR: We thank the commentators for sharing their perspectives and we would like to expand on them as a group—with a focus on causation. Although equilibrium volume definitions may appear semantic, disagreement leads to experimentally differentiable consequences about suction, which as Little and Ohara (see Ref. 5) point out, should not be lost.

Thus we step back and consider the physiology. When the ventricle fills with blood, what is the cause, and what is the effect?

There is no controversy regarding the cause of late (post-diastatic) atrial filling (Doppler A-wave). As described (4), atrial contraction not only causes atrial and ventricular pressure rise, but generates elastic stretch. If the R-wave following atrial relaxation is delayed (as in 1st degree AV block), then late diastolic mitral regurgitation ensues, causing LVP to fall as the LV recoils back toward diastasis. This is significant, because it highlights the role of elastic elements. As ventricular shape or volume changes, elastic elements are either loaded or unloaded, with predictable consequences.

To us the cause of early filling (Doppler E-wave) is unambiguous. While it is possible for excessively high left atrial pressure to contribute to filling, as Remme and Smiseth's (see Ref. 5) mathematical model suggests, physiological observations rule out an atrial push as the sole E-wave generator. First, the observation that  $dP/dV < 0$  at the start of all E-waves demonstrates that the LV expands faster than it can fill. Second, the atrium is a conduit (1), as the LV aspirates blood from the lungs (Doppler pulmonary vein D-wave) while atrial pressure drops. Furthermore, if  $V_0 < ESV$  was the elastic equilibrium, then during the E-wave, elastic elements would be stretched even farther away from  $V_0$ . Because we never observe a ventricle recoiling back from diastasis volume toward  $V_0$ , but do observe ventricles recoiling toward diastasis in first degree AV block, we conclude that defining  $V_0 < ESV$  as equilibrium is incompatible with the physiology.

Our causal considerations are based on statics and dynamics and supported by experiment (1, 3). This lead us [and others (2, 4)] to

conclude that when all ventricles fill in early diastole (always with  $dP/dV < 0$ ), elastic restoring forces, unmasked by the relaxation process are the cause. As the ventricle expands, elastic elements return toward equilibrium and the degree of recoil can be observed ultrasonically or hemodynamically (6).

Importantly, the concept is heart rate independent; at higher heart rates the ventricle may pass through its equilibrium (diastasis) volume without stopping before atrial filling. In the case of dilated cardiomyopathy, or heart failure with elevated LAP, restoring forces may be weak and early filling (suction) may be modest or poor, but diastasis remains the volume at which forces are balanced and equilibrium is achieved. As Little and Ohara note, the impairment of suction as a cause of heart failure is key- and our causal kinematic paradigm provides a clear path to better understanding cardiac (patho)physiology.

Thus the causality of elastic recoil unmasked by relaxation leads us to conclude: LV volume during diastasis *is* the physiological in vivo equilibrium volume and *is* related to diastolic suction.

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