Last Word on Viewpoint: Is left ventricular volume during diastasis the real equilibrium volume, and what is its relationship to diastolic suction?

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TO THE EDITOR: We appreciate the thoughtful commentaries (1, 5, 8), to which we respond thematically. We cite additional references (1a, 2–4, 6, 7) and the prior Viewpoint references.

TRANSMURAL VS. TRANSMITRAL PRESSURE GRADIENTS

Existence of diastolic transmural pressure gradients ($\Delta P_{transmural}$) is not questioned; however, $\Delta P_{transmural}$ are the results, not the causes, of suction. Even short-axis ventricular slices with zero $\Delta P_{transmural}$ spring apart when cut (see Viewpoint, Ref. 22). Furthermore, in contrast to open-chest settings, $\Delta P_{transmural}$ and transmitral gradients are not interchangeable in closed-chest settings.

If $dP_{LV}/dV_{LV}<0$ and the atrium is intact, the recoil overcomes residual unrelaxed elements and the endocardium recoils faster than the aspirated blood can enter. Suction is the resultant sum of tissue-motion generated forces on the blood, and therefore includes any effects that $\Delta P_{transmural}$ may have on the tissue.

The duration and magnitude of $dP_{LV}/dV_{LV}<0$ determines the extent of suction. Thus suction exists with LV dilatation, but the duration of suction and the associated aspirated volume is diminished relative to healthy hearts because ESV is barely below $V_{diastasis} = V_{equilibrium}$.

When atrial pressure rises such that $P_{LA}>P_{peri}$, the atrium exerts outward force on the pericardium, and although this may increase the atrioventricular pressure gradient it does not cause suction. Indeed, ventricles can fill without atria (see Viewpoint, Ref. 28). Instead, suction is powered by elastic energy stored by titin (Viewpoint, Ref. 11) and other sources (3, 6) and involves recoiling tissue-generated P_{LA}-P_{LV} being converted to motion of blood.

MECHANISMS OF SUCTION

Our Viewpoint unifies two definitions of suction: one invoking $dP_{LV}/dV_{LV}<0$ and the other relying on $ESV<V_{Eq}$. We show that the laws of statics and dynamics, independent of specific ventricular recoil mechanisms, establish that suction-initiated filling brings the ventricle to mechanical equilibrium at $V_{diastasis}$.

RELIABILITY OF MEASUREMENTS

Functional imaging elegantly elucidates details inaccessible to catheters, but these details are consequences of ventricular suction. Demonstration of ventricular suction only requires that $dP_{LV}/dV_{LV}<0$. Indeed Pasipoularides (5), in agreement with others, observes RV pressures decreasing as RV filling proceeds.

IS DIASTASIS HEMODYNAMIC STASIS?

Conclusions from Carlsson et al. (1a) regarding diastasis must consider heart rate (HR), because diastasis is lost when HR > 80 (2). Indeed, in contrast to the two lower HR subjects from Carlsson et al., the remaining six subjects had HR ≥ 80 and lack diastasis.

Intracavity flow exists during early diastole (3), although by diastasis there is no net volume change and intracavity swirling diminishes to minimal levels (Fig. 2*E*; Ref. 7).

When L-waves are present, or in other cases where LV volume continuously changes, $V_{diastasis}$ is not achieved. However, these cases are exceptions to the rule, and most ventricles possess diastasis (see online supplemental video).

Load variation impacts transmitral gradients, transmitral flow, and $V_{diastasis}$. We stress that while $V_{diastasis}$ is a static equilibrium state for a particular beat, specific $V_{diastasis}$ values are *expected* to vary with load. Thus filling pressure variation may not directly correlate with changes in suction.

CONCLUSION

The physiological mechanisms governing diastole remain an active field of research. Future and current work regarding diastolic function must abide by the constraint that all hearts are mechanical suction pumps ($dP_{LV}/dV_{LV}<0$) at mitral valve opening and initiate filling at a volume below their equilibrium volume ($ESV<V_{equilib}=V_{diastasis}$).

REFERENCES

1. **Ade CJ, Wong BJ.** Commentary on Viewpoint: Is left ventricular volume during diastasis the real equilibrium volume, and what is the relationship to diastolic suction. *J Appl Physiol*; doi:10.1152/japplphysiol.90325.2008.
1a. **Carlsson M, Cain P, Holmqvist C, Stahlberg F, Lundback S, Arheden H.** Total heart volume variation throughout the cardiac cycle in human. *Am J Physiol* 287: H243–H250, 2004.
2. **Chung CS, Karamanoglu M, Kovács SJ.** Duration of diastole and its phases as a function of heart rate during supine bicycle exercise. *Am J Physiol Heart Circ Physiol* 287: H2003–H2008, 2004.
3. **Jöbsis PD, Ashikaga H, Wen H, Rothstein EC, Horvath KA, McVeigh ER, Balaban RS.** Visceral pericardium: macromolecular structure and contribution to passive mechanical properties of the left ventricle. *Am J Physiol Heart Circ Physiol* 62: 3379–3387, 2007.
4. **Peskin CS, McQueen DM.** A three dimensional computational method for blood flow in the heart: immersed elastic fibers in a viscous incompressible fluid. *J Comput Phys* 81: 372–405, 1989.
5. **Ritman EL, Pasipoularides A, Arts T, Delhaas T, Sengupta PP, Khandheria BK, Tajik AJ, Boussuges A, Regnard J.** Commentaries on Viewpoint: Is left ventricular volume during diastasis the real equilibrium volume, and what is the relationship to diastolic suction. *J Appl Physiol*; doi:10.1152/japplphysiol.zdg-8134.vpcomm.2008.
6. **Robinson TF, Factor SM, Sonnenblick EH.** The heart as a suction pump. *Sci Am* 254: 84–91, 1986.
7. **Sengupta PP, Khandheria BK, Korinek J, Jahangir A, Yoshifuku S, Milosevic I, Belohlavek M.** Left ventricular isovolumic flow sequence during sinus and paced rhythms: new insights from use of high-resolution Doppler and ultrasonic digital particle imaging velocimetry. *J Am Coll Cardiol* 49: 899–908, 2007.
8. **Yildiz M.** Commentary on Viewpoint: Is left ventricular volume during diastasis the real equilibrium volume, and what is the relationship to diastolic suction. *J Appl Physiol*; doi:10.1152/japplphysiol.90338.2008.

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