Comments on Point:Counterpoint: Left ventricular volume during diastasis is/is not the physiological in vivo equilibrium volume and is/is not related to diastolic suction

IN FAVOR OF KATZ’ DEFINITION

TO THE EDITOR: We despair of achieving absolute clarity or agreement in this discussion (4, 6) but we applaud the statements affirming Katz’ criterion (3)—with which we agree.

Brecher, ourselves, and Nikolic have defined an equilibrium volume, Vo, of the passive ventricle but the relevance of this equilibrium volume to the relaxing ventricle is limited in that the recoil energy of the LV can still fill itself, even at volumes greater than Vo.

Kovacs and his colleagues (4) have every right to define the volume at diastasis as an equilibrium volume but, with increasing heart rate, there is no diastasis. Does that mean there is no equilibrium volume? Diastasis would seem to be a luxury of a slow heart rate so it would seem that the relevance of the diastatic volume is limited to an important degree. We question whether the diastatic volume has anything fundamental to do with diastolic suction, beyond the fact that diastasis, if present, occurs after the processes described as diastolic suction have been completed.

Wave intensity analysis defines diastolic suction consistent with Katz’ definition (3), and the magnitude of the energy of diastolic suction is greater after accounting for the passive compliance of the LV (2). Recently Cheng-Baron et al. (1) have described the structural concomitants of ventricular relaxation and recoil.

Katz’ definition (3) remains the gold-standard conceptual foundation of diastolic suction.

REFERENCES


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TO THE EDITOR: We read with interest the Point: Counterpoint articles by Shmuylovich et al. (3) and Yellin and Nikolic (5). Left ventricle myocardial relaxation includes the end of myocardial shortening, the isovolumic relaxation time, and the rapid filling phase. The time rate of relaxation is an important determinant of diastolic function and is regulated by myocyte inactivation, loading conditions and suction of the myocardium. The diastolic suction is associated with intraventricular pressure and volume changes. In clinical transthoracic echocardiography, Doppler indexes were used to study diastolic function. Mitral valve inflow assessed by pulsed wave Doppler remains the cornerstone of diastolic assessment. Several parameters such as early diastolic transmural velocity (E), diastasis (during this period of ventricular filling, the pressures of the left atrium and ventricle are nearly equal), late diastolic transmural velocity (A), early filling deceleration time can be derived from mitral inflow velocities, which are useful for diastolic function. Since transmural E wave is generated by the pressure gradient between left atrium and left ventricle, E-
wave velocity may be influenced by the pressures and compliances of this chambers. Some studies showed that the intraventricular pressure gradient between the apex and base of the left ventricle during early diastolic filling were related to elastic recoil, flow propagation velocity, peak early transmural flow and stroke volume (1, 2, 4). Consequently, in our clinical perspective, left ventricular volume during diastasis may not be limited with diastolic elastic recoil (suction).

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TO THE EDITOR: In this article series, the term “diastolic suction” is used to designate three non-exclusive phenomena, each one identified in a different experimental framework: 1) the subatmospheric pressure generated during early diastole (non-filling experiments; Ref. 4), 2) the negative dP/dV filling phase (PV relationship; Ref. 3), and 3) the positive LA-LV base-apex pressure gradient (multisite pressure mapping). We believe that a consensus definition of diastolic suction should be reached before its physiological determinants can be unambiguously discussed.

The two first definitions were established using a lumped cardiovascular model. However, lumped models fall short to explain the complex interdependent early diastolic phenomena described recently. Myocardial torsion stores energy, which, on release, creates the relaxation expansion-wave and the pressure gradient toward the apex (2). Translating cell and matrix recoil to organ scale requires considering not only three-dimensional chamber geometry, but also intraventricular flow dynamics (5). For a given end-systolic volume, different amounts of energy can be released, depending on the architectural arrangement of fibers. The efficiency of released energy is, in turn, condition by the phenomena ongoing on LV outflow (Ref. 5; decelerating ejection flow) and inflow (generating vorticity and pressure gradients (5) among others).

Using cardiovascular imaging techniques, a number of these properties can be now measured directly in patients (2, 5, 6). Beyond volumetric changes, additional factors related to diastolic suction are becoming clarified, even in the relatively unexplored right ventricle (1). To integrate such a new insight, cardiovascular models need to be improved. However, noninvasive indexes of (or related to) diastolic suction have been well validated; clinical trials assessing their value for improving patient care are therefore warranted.

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TO THE EDITOR: In fact, the focus of this debate (6, 7) is not beyond the old question of “vis a front,” which means force from in front to drive blood flow. Kovacs’ group proposed the ventricular equilibrium volume to explain the suction of heart pump. The reason of fail to be accepted as a new concept is that there is almost no break point for a new framework. There is no doubt for the suction property of ventricles, but it is too weak to emphasize its role as a suction pump (4, 5).

The heart pump is constructed of the four chambers contained in a closed chamber that is the pericardial cavity. Although there is great change of the volume of each chamber, pericardium keeps the heart in a constant total volume during cardiac cycles (1, 3). This result strongly supports the presence of ventricle-atrial interaction, and the atrial may pull (not like Kovacs’ “push”) the ventricles to expand. The force of atrial pulling should include the potential energy in the atrial that exerted by ventricle contraction and atrial active contraction. From this viewpoint, the systolic suction that defined as stretch the atrium and auricle by ventricle contraction is more powerful to explain heart as a suction pump than diastolic suction.

The open chest model destructed the intact heart pump structure, eliminates the ventricle-atrial coupling mechanism, generally applicable to the failing heart. In contrast to the failing heart, normal heart decrease left ventricle end-diastolic pressure during exercising in conscious dogs (2). Thus the author suggests that an intact heart pump drives the blood flow vis a front while vis a torgo.
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EQUILIBRIUM VOLUME AND SUCTION

TO THE EDITOR: Normally, the left ventricle (LV) functions as a suction pump (1). This protects against pulmonary congestion and allows the stroke volume to double during exercise with only a minimal increase in left atrial pressure. LV suction is most clearly demonstrated by the observation that the LV will fill from a zero source pressure. Thus Yellin and Nikolic (3) maintain that ejection to less than the Vo is necessary for suction. However, it is not necessary for the LV to be able to fill from a zero source pressure. It is only necessary that it is able to fill from a normal left atrial pressure.

Shmuylovich et al. (2) propose that the LV volume at diastasis be considered as the equilibrium volume of the ventricle. However, the left ventricular diastatic volume is substantially influenced by the pulmonary venous pressure. Thus, in a normal situation, diastasis occurs at a left atrial pressure of < 10 mmHg; while in a patient with severe heart failure, the diastatic volume will occur at a left atrial pressure of >30.

It is important that semantic arguments over the definition of equilibrium volume or suction do not obscure the very important pathophysiological fact of the performance of the left ventricle as a suction pump. The ability of the LV to increase filling without requiring abnormal elevations of left atrial pressure is essential for the cardiovascular system to meet the body’s needs during stress and the loss of suction is an important cause of heart failure.

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