TO THE EDITOR: The Viewpoint by W. Zhang et al. (3) advances our understanding of the age-old question about the meaning of end diastole of the left ventricle as well as its corollary, diastolic suction. The discussion omits several additional mechanisms that may also be contributing to left ventricular (LV) filling. One plausible explanation of the recoil generated by the myocardial muscle is the erectile effect of the intramyocardial microcirculation being distended as blood enters it again after the systolic “blanching” ceases (2). Another plausible mechanism is that the recoiling LV myocardium merely advances over the stationary blood in the ventricle and atrium—much like a sock pulled (or in this case pushed) over a foot.

Given these multiple plausible mechanisms, the question arises as to how much of the ventricular filling is due to suction. Suction generated by the ventricle should be reflected by negative transmural pressure during early diastolic relaxation. That this is true only in the late systolic phase was suggested by Kenner and Wood (1) using simultaneous percutaneous measurement of pericardial and intracardiac pressures. Their data show that although pericardial pressure is transiently more negative during systole, that pressure is essentially constant at pleural pressure after the aortic valve closes, suggesting return to a positive transmural pressure during diastole.

In summary, while I do not disagree with the observations and conclusions drawn from them as presented in this Viewpoint, I believe that these other mechanisms should also be considered as possible contributors to this elusive issue.

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DOES LEFT VENTRICULAR SUCTION EXIST?
TO THE EDITOR: A vacuum chamber does not attract matter, but matter is pushed out of regions with high pressure (Wikipedia, item “suction”).

Mitrail flow is energized by the summed action of left atrium (LA) and left ventricle (LV). At initiation of mitral flow, the LA contributes energy by push (pLA-pPERI)·dVLV. Following the ideas of Nikolic, the LV sucks when during filling the cavity pressure (pLV) is below intrapericardial pressure (pPERI), which was atmospheric in his open chest preparations. The LV contributes energy by (pPERI-pLV)·dVLV, which value is practically always negative, i.e., the LV does not suck, but is weakly pushing.

Why talking about suction? LV energy is the sum of elastic recoil by the passive matrix and active contraction. At initiation of mitral flow, passive recoil delivers energy to filling. The still partly activated myocardium however generates stress while being stretched, implying consumption of mechanical energy.
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parently, the weak passive suction component is overruled by the effect of stretching of still partly activated myocardium.

A consistent definition of suction is supply of mechanical energy by the LV wall during filling, implying negative transmural pressure. If we would accept the proposed definition of suction by dpLV/dVLV < 0, with increasing LA pressure together with LV dilatation, suction would stay high, whereas a dilated LV is accepted to have less or no suction at all.

In summary, the left ventricle can suck, but it never does.

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IS DIASTASIS REALLY A PHASE OF HEMODYNAMIC STASIS?

TO THE EDITOR: The Viewpoint by Zhang et al. (5) reiterates diastasis as a period of zero-motion (static) condition over a finite time interval where left ventricular (LV) wall mechanics and transmitial flow are both absent. The following observations, however, would suggest that diastasis may be more complex than just a period of equilibrium and stasis.

1) First, changes in LV volume and deformation during diastasis do not reveal a halted phase of mechanical relaxation, rather, LV continues relaxing and lengthening, attaining progressively higher volumes (1).

2) Volume change in diastasis result from large-scale intracavitary vortical motions that develop during the down stroke of E-wave (3). Large vortices never unwind smoothly. Rather, they break up into smaller eddies, dissipating and maintaining a steady outward force on the LV endocardial surface. This facilitates continued filling and an increase in LV volume.

3) Indeed filling mechanisms in diastasis are heightened in some failing hearts. Flow in diastasis may be augmented, leading to genesis of mid-diastolic filling wave, also referred to as “L-wave” (2).

There are practical limitations, therefore, in separating early diastolic suction from diastasis because LV volume changes during both phases occur on a continuum. Recent studies have redefined suction as an active state wherein postystolic regional shortening (beyond aortic valve closure) produces dynamic shortening-relaxation gradients within LV wall that hasten the process of diastolic restoration (4). The cross-over point of postystolic shortening into relaxation may therefore better define the period in diastole when active LV suction ceases to operate.

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2. Wang J, Khoury DS, Thohan V, Torre-Amone G, Nague SF. Global diastolic strain rate during the isovolumetric relaxation period, is well related to hemodynamic indices of LV relaxation (2). Furthermore, Notomi et al. (1) highlighted that ventricular untwisting provided a temporal link between relaxation and diastolic suction. Consequently, and as underlined by Zhang et al. (3), the definition of the diastolic suction should not be limited to changes in pressure and volume, but should also integrate the heart deformations leading to the restoration of a nonstressed LV shape (equilibrium volume).

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