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EDITORIAL COMMENT

The Dynamic Vortex of a Beating Heart

Wring Out the Old and Ring in the New!*

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"I find the great thing in this world is not so much where we stand as in what direction we are moving."
—Johann Wolfgang von Goethe (1)

In nature, fluid motion often chooses to spiral rather than stream linearly. A beating heart, with its helical myofiber architecture and twisting-untwisting motion, also reveals spiraling streams of blood flow. This mechanism is highly conserved in vertebrates (2). Why does nature choose to move blood in a helical manner, and how does that make normal cardiac function efficient? These are important but complex questions that are just being answered.

The left ventricle (LV), both at rest and during exercise, normally needs only a small amount of left atrial (LA) pressure to fill adequately, suggesting that LV filling is more than just a passive process. Both systolic and diastolic dysfunction have impaired filling in common and need elevated LA pressures to fill the LV, especially during exercise. Multiple mechanisms may be involved, but loss of the normal helical filling—vortex—may be important.

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In this issue of the *Journal*, Martínez-Legazpi et al. (3) used currently-available 2-dimensional (2D) color flow Doppler technology to find a rather easy way to deconstruct ventricular filling into rotational (vortex) and nonrotational components. They show that the vortex contributes more to LV filling in dilated cardiomyopathy (DCM), especially with spherical hearts, whereas the smaller vortex component in hypertrophic cardiomyopathy (HCM) might contribute to diastolic dysfunction. Because vortices fill the ventricle with minimal cost in terms of energy and pressures, they may help LV filling in disease states without a proportionate need for high filling pressures. These data add to the growing body of evidence that link specific forms of LV remodeling with characteristic flow patterns.

WHAT ARE VORTICES AND WHY DO THEY FORM?

The cardiovascular system's dynamic contours create time-varying and spatially-complex patterns of blood flow. Flow coming in from different directions is melded into a compact, nonturbulent mass of fluid and channeled towards preferential flow lines, often in the form of vortices, ring-shaped regions of rotating blood flow (2,4). Mitral leaflets and the trabeculated endocardium further modulate the flow, facilitating continuous asymmetric redirection of blood flow to the outflow tract.

Vortices have different formation time, size, shape, strength, depth, and direction depending on the time of the cardiac cycle, as well as valve and chamber geometry (**Figure 1**). Which characteristic of the vortex is most important is not clear, but a tightly compact, persistent ring seems to provide the best flow propagation (4,5). Interestingly, vortex properties depend on chamber function, but vortices also modulate diastolic LV wall lengthening and recoil (5). Vortex characteristics may thus be a signature of myocardial health and disease.

WHAT ARE THE ADVANTAGES OF VORTEX-MEDIATED FILLING?

Vortices, whether in tubes, aquatic motion, or nature, seem to transport fluid more efficiently than

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in a straight jet by providing a compact hydrodynamic channel (6). Vortices help multidirectional streams of blood merge without collision and energy loss. Their ability to add volume to the LV without a significant increase in pressure benefits chamber compliance, a mechanism that may be important in stiff hearts (2). This is especially important when gradient-dependent filling slows down in early to mid-diastole. Vortical flows continue in late diastole and preferentially move blood into the LV outflow; the resulting volume and stretch appear to "prime the pump" and help maintain momentum into the isovolumetric contraction phase (2). Finally, vortices help controlled coaptation of valve leaflets during closure.

The most intriguing advantages of vortex formation may be to couple flow, stretch, and cellular response, whether it is via mechano-induction of gene programs, stretch-dependent cellular signaling, or maybe even shear-dependent modulator release, which can all affect chamber remodeling and contractility (7). Fluid forces and vortices are epigenetic modulators in the development of cardiac chambers and valve geometry (8), and vortices may help distribute flow-related stress loads, which in turn might be able to modulate myocyte and matrix geometry (7,9).

VORTEX RING AND DIASTOLIC FUNCTION. The general trend in vortex biology seems to consider inefficient vortex formation as pathology that directly contributes to systolic and diastolic dysfunction. Indeed, preliminary observations have suggested that vortex formation is impaired in heart failure in proportion to some elements of prognosis (10). However, very few studies have modulated vortex formation in vivo in a controlled manner to see if deficient vortex formation impairs cardiac function, and none have shown that reversing it improves function (11). This study was unique in quantifying the vortex contribution to ventricular filling and thus extending it to diastolic dysfunction: normal and DCM ventricles ported 15% to 20% of LV filling via vortex, but only 5% of LV filling in HCM patients was mediated by vortex.

What role do deficient vortices (as in HCM) play in mediating diastolic dysfunction? Although the authors conclude that unfavorable vortex morphology was important, the study does not clarify its unique role in HCM. Despite a small vortex, E/e′ (a rough measure of LA pressure) was not much different than in DCM, implying that vortex loss did not require proportionately higher LA pressures to fill the LV and maintain stroke volume in HCM patients. Because chamber sphericity was a major determinant of vortex contribution to filling, type of remodeling might be a more important factor for LV operating characteristics than the vortex by itself.

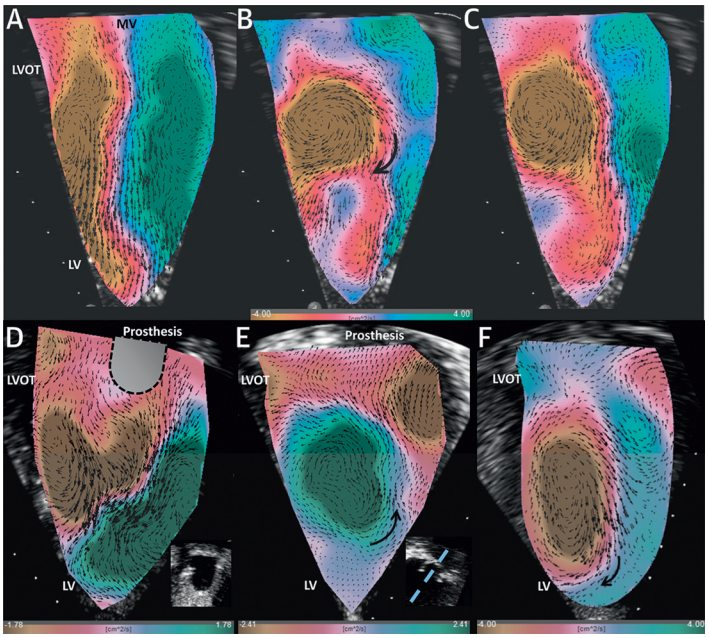

FIGURE 1 LV Vortex During Diastolic Phases Normally and in Surgically-Intervened Mitral Valves

Development of larger clockwise blood flow rotation **(arrow)** is delineated in a normal heart during early diastole **(A)**, diastasis **(B)**, and late diastole **(C)** using high-resolution echocardiographic contrast particle imaging velocimetry. Flow redirection is markedly disorganized for a ball-in-cage prosthesis **(D)**, reversed posteriorly **(arrow)** for a tilted bioprosthetic valve **(E)**, and better preserved with mitral valve repair **(F)**. LV = left ventricle; LVOT = left ventricular outflow tract.

As with many studies in this field, the cause and effect is suggestive but not fully clear: are vortex size and flow calculations simply the consequence of a larger chamber size in DCM and a smaller chamber in HCM? Importantly, HCM volumes in this group may not represent the usual HCM population, and many DCM subjects had significant mitral regurgitation; this could affect vortex characteristics. Vortex biology, although very attractive, remains hypothetical; distorted vortex formation may be a well-correlated marker of cardiac structure, remodeling, and function, and other mechanisms might compensate for their absence.

IMPLICATIONS AND LIMITATIONS. Using 2D color flow Doppler, Martínez-Legazpi et al. (3) provide a convenient method for studying vortices. However, many physical assumptions related to the boundary conditions and the study's complex mathematical modeling will require further validation. Interestingly, DCM patients had worse diastolic dysfunction

and deformation parameters, but their vortex-mediated filling was "better" than in HCM. The reason for this paradox is not clear. Although the present investigation suggests that vortices carried more fluid in DCM, others have shown that vortex size and strength are reduced in patients with heart failure with reduced ejection fraction (10). The parameters (vorticity, energy, dissipation) that would best correlate with a clinical endpoint were not investigated and should be addressed in future studies.

CONCLUSIONS. Martínez-Legazpi et al. (3) help to discern the role of diastolic filling vortex as a key physiological contributor of LV filling. On a wider scale, the study symbolizes a shift in our thinking that the heart is just a simple pressure generating mechanism. While the systolic "wring" helps eject the "old" volume, the diastolic vortex "ring" helps conserve the "new" momentum of arriving blood. This implies a slingshot-like mechanism for efficient handoff of blood from diastole to systole. It is also time to use more sophisticated measures of diastolic function than those we use today. Advances in visualization techniques and ensuing research will help us close knowledge gaps. Noninvasive assessment of LV fluid mechanics may assist in designing optimal surgical strategies, valve scaffolds, and therapeutic interventions in the near future.

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