

Review Article

Fluid Dynamics of Ventricular Filling in Heart Failure: Overlooked Problems of RV/LV Chamber Dilatation

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D iastolic cardiac dysfunction as a component of heart failure is nowadays sufficiently recognized as to be a part of the coding for congestive heart failure in the International Classification of Diseases (ICD-10, codes I50.30-33). This recognition of diastole by the World Health Organization has been accompanied by major strides in the appraisal of diastolic function,¹⁻⁸ made possible by technology that encompasses multi-sensor cardiac catheterization,^{1,2,9-11} and invasive and noninvasive digital imaging modalities.^{1,12-22} It is now widely appreciated that enhancing diastolic filling has clinical merit and that the significance of diastolic dysfunction is far reaching.^{23,24} Nonetheless, the fact that in health and disease diastolic dynamics are dependent on a large number of factors and their interactions^{1-3,22,25} has complicated the evaluation of the multifactorial causes of the observed ventricular filling abnormalities.

As our pathophysiologic understanding of heart failure has progressed over the past 3-4 decades, remodeling with ventricular enlargement has surfaced as a central mechanism, relevant to heart failure progression and to the prediction of outcomes.¹ The present survey presents accumulating evidence that, besides leading to augmented myocardial wall stresses and impaired systolic function,^{26,27} ventricular

chamber enlargement *per se* can encumber ventricular diastolic filling through fluid dynamic mechanisms.

This radical viewpoint embodies findings and concepts developed through investigations of integrative diastolic function in animal models of heart disease and failure^{11,17,28-32} at the Duke Center for Emerging Cardiovascular Technologies. They concern interactions of mechanisms of cardiac fluid dynamics with atrioventricular (A-V) dynamic geometry and inflow patterns, and diastolic function alterations accompanying chamber enlargement, which are simple to understand, but important in their clinical implications. The clinical studies that we will review, which exemplify and confirm the underlying fluid dynamic principles, also demonstrate the practical value of these principles in managing heart failure with myocardial remodeling and right and left ventricular (RV/LV) dilatation.

Myocardial diastolic function

The initial phase of the research, which led to the integrative diastolic function framework depicted in Figure 1, entailed baseline and longitudinal studies on subacute-to-chronic canine surgical models of RV volume overload (VO), pressure overload and myocardial ischemia, utiliz-

ing right-heart multisensor Millar catheters, real-time 3D echocardiography, and specially designed pulse-transit ultrasonic dimension transducers.^{17,33,34} Only chronically instrumented awake dogs were studied, because of the important limitations of acute studies under conditions of anesthesia, recent surgery, and open chest.³³⁻³⁶

Regarding diastolic myocardial function, in contrast to pressure overload and myocardial ischemia, no significant change from control in the RV time constant of relaxation, τ (tau), was found with RV VO.^{33,37} The only significant change in VO was a raised RV diastolic pressure asymptote, reflecting increased diastolic constraint from elevated right heart volumes.^{1,2,33,36,37} These results suggest that the relaxation mechanism is unimpaired in subacute-to-chronic RV volume overload.³³ They are consistent with earlier LV findings by Zile and coworkers on a comparable canine VO model,³⁸ and with subsequent clinical findings in children with diverse conditions producing RV volume overload.³⁹

Filling pressure vs. volume relationships

A sigmoidal model^{1,34} for passive filling pressure versus volume relations and the resultant myocardial compliance formulations showed that the maximum RV myocardial compliance, which is attained during early filling, becomes significantly reduced from control with pressure overload and ischemia but not with VO. In contrast to pressure overload and ischemia, in VO with ventricular enlargement the pressure-volume relationship shifts far to the right and downward over much of the filling process; end-diastolic myocardial compliance actually increases in VO compared to control, while end-diastolic pressure stays unchanged.^{1,3,34} These RV results are again congruous with the earlier LV data obtained by Zile et al in a similar VO canine model.³⁸

Fluid dynamically disadvantaged filling in dilated ventricles

The intriguing feature of the foregoing findings is that VO engenders minimal abnormalities in myocardial properties relative to control. Since the RV chamber size in VO (end-diastolic volume 60 ± 29 mL) increased markedly ($p < 0.05$) from control (45 ± 21 mL), reflecting myocardial creep and remodeling,^{1,3,34} it was hypothesized that it might be responsible for unrecognized dynamic filling changes relative

to control. Therefore, a fuller understanding of integrative diastolic ventricular function in the setting of chamber enlargement can be forthcoming only if the scope of the investigations is expanded beyond myocardial mechanics, which consider merely myocardial relaxation and compliance changes.⁴⁰ A comprehensive examination of ventricular filling was called for, utilizing combined sonomicrometric, digital imaging, and computational fluid dynamics (CFD) methods, to look for fluid dynamic underpinnings of diastolic dysfunction in dilated ventricles.^{17,28,29,32}

The functional imaging (FI) method for the study of intracardiac flow

The ensuing fluid dynamic studies^{17,28,29} revealed important but previously unrecognized mechanisms responsible for the filling impairment in the volume-overloaded, dilated ventricles. These underlying mechanisms were revealed and investigated by the FI method.^{1,17} The method comprises real-time, 3D echocardiographic (RT3D) and sonomicrometric measurements combined with CFD simulations of the intracardiac flow field;^{1,11,22,31,32} it was applied to ascertain diastolic filling dynamics in individual dogs, studied at control and in RV dilatation induced by subacute-to-chronic VO.^{17,28,29} Although real time 3D echocardiographic imaging^{1,17} was used in our studies, these techniques are applicable generally to modern digital imaging methods.^{1,13,41-46}

The FI method yields values of velocity and pressure at literally thousands of discrete points in space and time within each filling chamber simulated under control and chamber dilatation conditions. The vast amount of diagnostically invaluable qualitative and quantitative spatiotemporal information can be envisaged only when transformed into graphic representations. High-resolution plots of instantaneous intraventricular velocity and pressure distributions can be extracted from the FI simulation datasets, using interactive graphics modules.^{1,17,28-32} They reveal time-dependent, subtle interactions (detailed below) between the local acceleration and convective acceleration components of the total pressure gradient.^{1,11,22,29} The smallness of this total early diastolic intraventricular pressure gradient renders most directly gauged measurements of it—even by solid-state multisensor catheter—unreliable. Regrettably, this smallness also conceals the underlying dynamic mechanisms, which it is clinically advantageous to know, especially in managing heart failure with ventricular dilatation.^{11,22}

Local and convective acceleration components of the total RV/LV pressure gradient

In the intraventricular diastolic flow field, the velocity is a function of both time and space. Generally, there can be acceleration of fluid passing through a point in a flow even when the velocity at the given point is constant, i.e. even if the local (or temporal) acceleration, defined as the rate of change of velocity with respect to time, is zero.^{1,9,11,22,47} Streamlines connect velocity vectors in a flow field at a given instant. When streamlines are plotted so that the distances between them correspond to equal volumetric flux, the resulting plot gives information about regions of high and low velocities.^{1,9,48,49} Closely spaced streamlines indicate relatively high linear velocities, and vice-versa. Accordingly, converging streamlines in a flow region imply convective (i.e. flow-associated) acceleration; diverging streamlines imply convective deceleration along the stream (Figures 1, right and 2, top).^{1,9,28,48,49}

Convective deceleration load (CDL) and diastolic ventriculoannular disproportion (DVAD) in chamber dilatation¹

Tricuspid transvalvular phasic pressure differences measured in dogs by multisensor right-heart catheters^{1,29} under experimental hyperdynamic conditions, which accentuate them, exhibit dynamic characteristics, including their timing relative to the inflow velocity, similar to those shown for the mitral transvalvular pressure drop using clinical human data.^{1,2,11,50} Instantaneous RV intraventricular diastolic pressure gradients are smaller than their LV counterparts^{1,11,22,29} and are not generally amenable to reliable direct measurement, even by “high-fidelity” micromanometric catheter, as noted above.

The FI method has shown^{1,22,28-32} that up to the E-wave peak, instantaneous inflow streamlines extend from the tricuspid orifice to the RV endocardial surface in an expanding fan-like pattern (Figures 1, right and 2, top). This pattern agrees with the clinical measurements of Rodevand et al⁵¹ in the left ventricle: using Doppler echocardiography techniques, they showed that, during early transmitral flow acceleration, all intraventricular velocities are directed toward the chamber walls; however, after the peak of the E-wave, retrograde velocities ensue around the central inflow, connoting vortical flow.¹ Comparable streamline patterns were also obtained in humans using three-directional velocity-encoded MRI.^{52,53}

The diverging intraventricular streamlines during the E-wave upstroke imply that the RV/LV inflow is undergoing a convective deceleration (see preceding Section). Consequently, the total instantaneous intraventricular pressure gradient along the inflowing blood stream is the algebraic sum of a streamwise (viz. in the flow direction) pressure decrease contributed by the local acceleration, and a Bernoulli pressure augmentation contributed by the convective deceleration. The Bernoulli pressure augmentation partially counterbalances the streamwise pressure fall engendered by the simultaneously applied local acceleration of the inflow.^{1,11,22,29}

This mutually offsetting action underlies the astounding smallness of the total early diastolic intraventricular pressure gradients in animals and humans.^{1,2,11,22,29,54} In ejection, on the other hand, throughout the upstroke of the ejection waveform the intraventricular convective and local acceleration effects act jointly in the same sense,^{1,9,11,27,47,48} actually reinforcing each other. These contrasting dynamic interactions of its components underlie the fact that the total intraventricular pressure gradient (measured by multimicromanometric catheter) is much less prominent during the upstroke of the diastolic E-wave than during the upstroke of the ejection waveform; this is especially true under hyperdynamic conditions, such as during exercise, when both convective and local acceleration components are augmented.

At peak volumetric inflow the local acceleration vanishes, so that the total intraventricular gradient is represented by the convective component, which is adverse (i.e. the flow encounters higher pressure as it moves downstream), and flow persists under its previously built-up momentum, just as forward car motion persists transiently after the driver shifts from accelerator to brake. These considerations led me to formulate a new mechanism, the “convective deceleration load,” or CDL,^{1,11,17,22,28-32} as an important determinant of diastolic inflow and filling dynamics in health and especially in heart failure with ventricular dilatation.

Ventricular dilatation in RV/LV failure depresses the E-wave, furthering atrial overload

The magnitude of CDL strongly affects the attainable peak E-wave velocities. The larger the ventricle, the larger the CDL (Figures 1, right and 2, bottom). A ventricle enlarged in end-systole/early diastole (e.g. dilated cardiomyopathies and dilatation in heart fail-

ure) provokes a disproportionate increase in CDL and more difficult RV/LV inflow.^{1,11,22,28,29,31,32} Moreover, the pressure-rise representing CDL is proportional to the square of the velocities involved; accordingly, matters may be exacerbated in sympathoadrenally mediated tachycardia, which accompanies heart failure and causes diastolic filling time to wane.

The augmentation of CDL with acute or chronic chamber dilatation may contribute to depressed E-wave amplitudes and E/A ratio abnormalities, and to elevated atrial pressures and upstream congestion. Ensuing atrial overload (active boosting of difficult ventricular inflow by an atrial A-wave “kick”) may account for the association of atrial fibrillation with clinical ventricular enlargement in the absence of coronary disease.²⁹ The greater the discrepancy between the sizes of the RV/LV endocardial surface and the A-V valve annulus, the larger the CDL becomes (Figures 1, right and 2). These considerations underlie my formulation of the clinically important concept of a fluid dynamic “diastolic ventriculoannular (inflow valve) disproportion”^{1,22,28,29,32} (see DVAD in Figure 1, right), which is the counterpart, or diastolic analog, of the “systolic ventriculoannular (outflow valve) disproportion”, which I had formulated in a systolic clinical fluid dynamics survey in JACC.⁹

Diastolic large-scale intraventricular vortical motions

Early on during the downstroke of the E-wave, the intraventricular total pressure gradient becomes strongly adverse: it now embodies pressure augmentations in the downstream flow direction accruing from both local and convective decelerations.^{1,11,22,28,29} This induces flow instability and large-scale vortical motions, which are considerably more intense in the normal-sized than in the dilated RV chamber (Figures 1, right and 2, bottom);^{1,28,29,32} parallel results for the normal-sized and the dilated LV chamber have been found in clinical and animal studies and in computational simulations.⁵⁵⁻⁵⁹ These findings are in harmony with the groundbreaking discoveries of Taylor and Wade, who studied vortex formation in both the right and left ventricles during diastolic filling.⁶⁰ Vortex formation time has in recent years been proposed as an echocardiographic index of diastolic function based on the formation of intraventricular filling vortices, but no evidence is available that it has clinical or physiological applicability or value.⁶¹

Vortices represent rotatory motions of a mul-

titude of fluid particles around a common center and rotating flows have fascinated people for centuries. Leonardo da Vinci described vortices within the sinuses of Valsalva in his *Quaderni d'Anatomia* in 1513.^{1,9,62} Large-scale vortices (of a size comparable to that of the RV or LV chamber) dissipate little flow energy through viscous (frictional) effects. However, interactions of these vortices generate intermediate- and small-scale eddies, which are strongly dissipative. Thus, there is a cascade of energy from large, through intermediate, to small eddies, where rotational kinetic energy is dissipated as heat.^{1,25,28,62}

Diastolic LV vortices are well visualized using cardiac MRI and color Doppler-based echocardiographic vector flow mapping.^{1,41-45,52} They were previously investigated by Bellhouse in mechanical analogs of the left heart;⁶³ he observed that the mitral valve leaflets opened wide early in diastole, then moved increasingly toward closure, and were almost fully closed by end-diastole, whether atrial systole was present or not. Atrial systole caused partial A-V valve reopening before resumption of closure. Bellhouse compared A-V valve closure rates for two different end-systolic volumes, while maintaining all other parameters identical. When the operating end-systolic LV volume was small, a strong “ring-vortex” (akin to a confined smoke-ring) formed within the chamber. This vortex initiated mid-diastolic leaflet movements toward closure, and the valve was nearly closed by end-diastole. In contrast, when the end-systolic volume was abnormally large, as in heart failure, no vigorous ring-vortex formed, and the valve moved sluggishly toward closure, so that it was only 25% closed at end-diastole, and reversed flow was required to seal it.⁶³

Bellhouse concluded that the vortex plays an important role in the normal mechanism of closure of the mitral valve, ensuring minimal regurgitation by pushing the mitral anteromedial and posterolateral leaflets toward each other prior to LV myocardial contraction.⁶³

The facilitatory role of the filling vortex in diastolic filling

However, no other plausible useful function had been proposed for the intraventricular filling vortex until the comprehensive diastolic function studies at the Duke Center for Emerging Cardiovascular Technologies.^{1,3,11,22,28-34} Out of these pioneering studies, which integrated intracardiac flow phenomena with myo-

cardial relaxation and compliance dynamics, the hypothesis was advanced of an assisting role for the intraventricular diastolic vortex in diastolic filling. The key to this useful physiological role²⁸ of the RV/LV diastolic vortices lies in their impounding of a certain amount of flow energy, and this becomes manifest as a decrease in the pressure energy of the inflowing blood. By pre-empting an inflow-impeding Bernoulli pressure rise in the fanning streamline flow between A-V valve annulus and the expanding endocardial surface of the chamber (see Figures 1, right and 2), the diastolic vortex facilitates greater diastolic filling and stroke volume maintenance. It does so by shunting the inflow kinetic energy, which would otherwise contribute to the adverse convective Bernoulli pressure rise, into the kinetic energy of its gyratory motion. This rotational energy is ultimately dissipated as heat.^{1,22,25,28}

Heart failure with reduced vortex strength induces cardiac epigenetic changes

In recent publications,^{1,22,64,65} I have developed the hypothesis that changing “environmental” forces associated with diastolic RV and LV gyratory flows during filling exert important, albeit still unappreciated, epigenetic actions that influence functional and morphological cardiac adaptations. Mechanisms analogous to Murray’s law of hydrodynamic shear-induced endothelial cell modulation of vascular geometry are likely to connect RV/LV adaptations^{1,22,64,65} to the variable laminar vortical shear and centrifugal “squeeze” forces, which are exerted by the poloidally spinning diastolic vortex ring (cf. inset c, Figure 2) on the endocardium and myocardial walls. By Newton’s Third Law, the centrifugal force is directed away from the center of vortical rotation and is exerted by the revolving blood on the ventricular walls that provide its centripetal acceleration.

Clinical validation of the flow-encompassing diastolic filling framework

In view of its facilitatory role for ventricular inflow, as discussed above, the diastolic vortex assists filling by eliminating CDL to a variable degree that depends on vortex intensity.^{1,22,28,29,32} In the normal-sized chamber, the strong expanding ring vortex surrounding the central inflow core encroaches on the area available for flow toward the apex. This forces blood fluid elements in the central core (inflowing jet) to augment

their velocities as they pass into and through the region that is occupied by the expanding toroidal vortex surrounding the core.

As I detail elsewhere – see Chap. 14 in *Heart’s Vortex*¹ – a comparison of the shapes of the aliasing area in color M-mode Doppler (CMD) echocardiograms of the left ventricle and the spatiotemporal pattern of intraventricular flow, revealed by e.g. direct LV MRI velocimetry, points to the elongation of the aliasing area in a CMD echocardiogram as the manifestation of the expansion of the toroidal vortex and its migration toward the ventricular apex throughout the downstroke of the E-wave. Thus, the clinical evaluation of ventricular diastolic function by CMD echocardiography may, in my view,¹ reveal mainly the evolution of the size and shape and only secondarily the shifting localization of the intraventricular diastolic filling vortex with progressive ventricular expansion. In the CMD map, the recirculating vortical flow can at times be recognized as an E-wave pattern of forked distinct secondary “flames”.

The vortical encroachment normally causes higher linear velocities (cm/s) to actually occur later – during the downstroke of the E-wave – than the peak volumetric (mL/s) inflow rate.^{1,22,29} In dilated ventricles this effect is blunted, so that linear and volumetric peak velocities tend to coincide.^{1,22,29} These findings are consistent with meticulous clinical measurements published by Yamamoto et al,⁶⁶ who compared normal intraventricular velocity patterns to those found with LV enlargement, in dilated cardiomyopathy and in hypertensive heart disease. The regional diastolic velocity patterns at 1, 2, and 3 cm from the mitral tip toward the apex were recorded simultaneously with the mitral inflow velocity pattern, using multigated pulsed-Doppler. In the dilated left ventricles, linear velocities decreased strikingly from the mitral tip toward the apex.⁶⁶ In the new flow-encompassing framework for diastolic filling,^{1,22,28,29,32,64,65} this abnormal velocity distribution accrues from the convective deceleration of the inflowing blood (see foregoing discussion of CDL and DVAD), which in the dilated chambers persists unabated by a strong encroaching toroidal vortex (see Figures 1, right and 2).

In view of the integrative diastolic function framework of Figure 1, the detailed clinical findings of Yamamoto et al⁶⁶ regarding chamber dilatation are now clearly perceived as a manifestation of the strong convective deceleration occurring as the in-

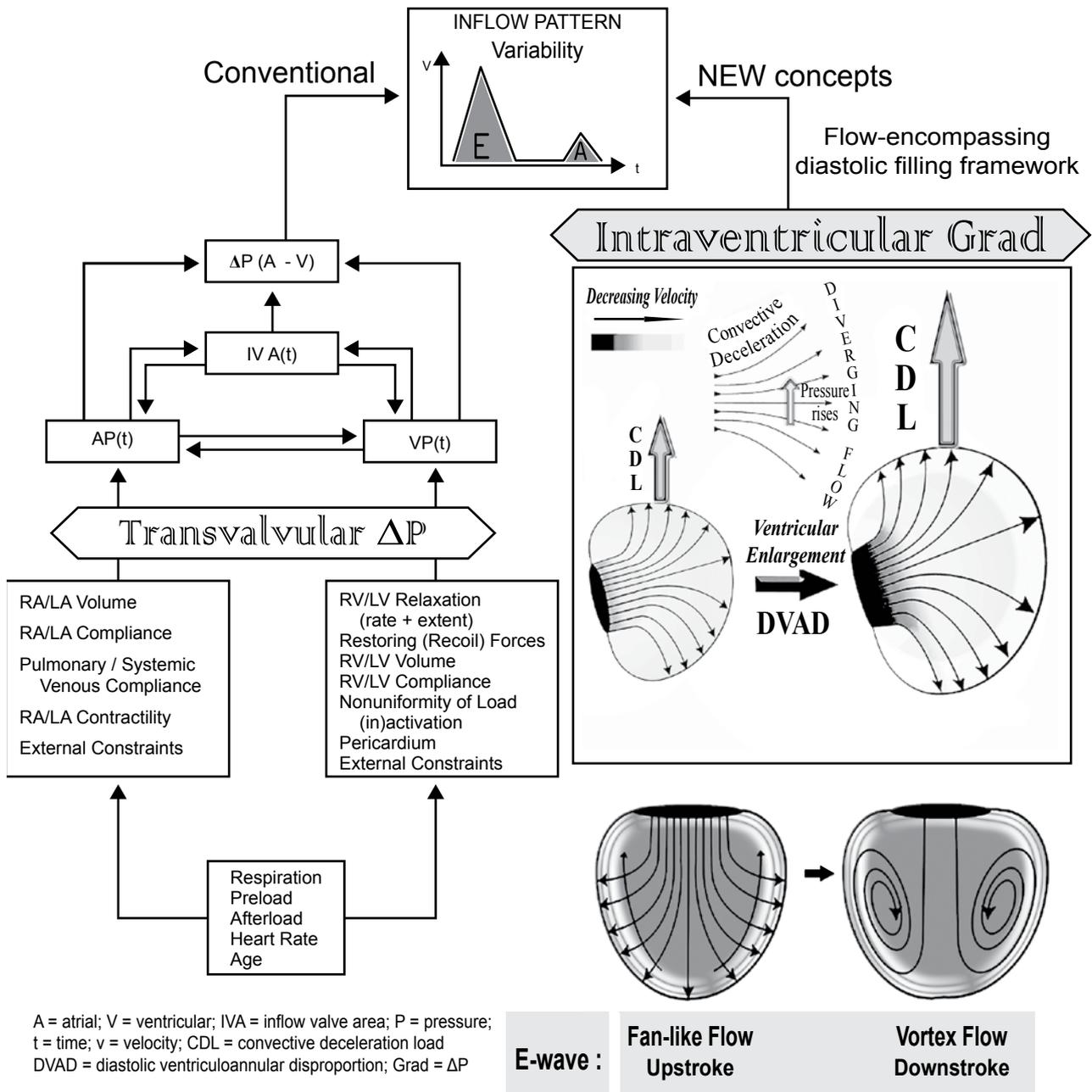


Figure 1. The new intraventricular flow-encompassing diastolic filling framework. The determinants of variable ventricular inflow patterns and diastolic filling in health and disease include factors intrinsic and extrinsic to the right and left ventricles. The new intraventricular flow-encompassing diastolic filling framework (right) complements and acts in parallel with the traditional diastolic function paradigm (left). Fundamental fluid dynamic mechanisms pertaining to CDL and DVAD, which are reviewed in the text, can impair filling regardless of coexisting conventionally recognized abnormalities. These mechanisms are operational particularly in the setting of compensated or decompensated heart failure with chronic or acute RV/LV chamber enlargement or dilatation. [Adapted, modified, from Pasipoularides A. Heart's Vortex: Intracardiac Blood Flow Phenomena. Shelton, CT: PMPH-USA, 2010. 960 p., with permission of the publisher.]

flow fans out in the absence of a strong, encroaching diastolic vortex (see Figures 1, right and 2). In contrast, in subjects with normal LV function and size, the simultaneously recorded velocities at these sites

were remarkably uniform.⁶⁶ This reflects the presence of the ring-vortex surrounding the central incoming stream and preventing fanning out of the streamlines with ensuing strong convective deceleration.

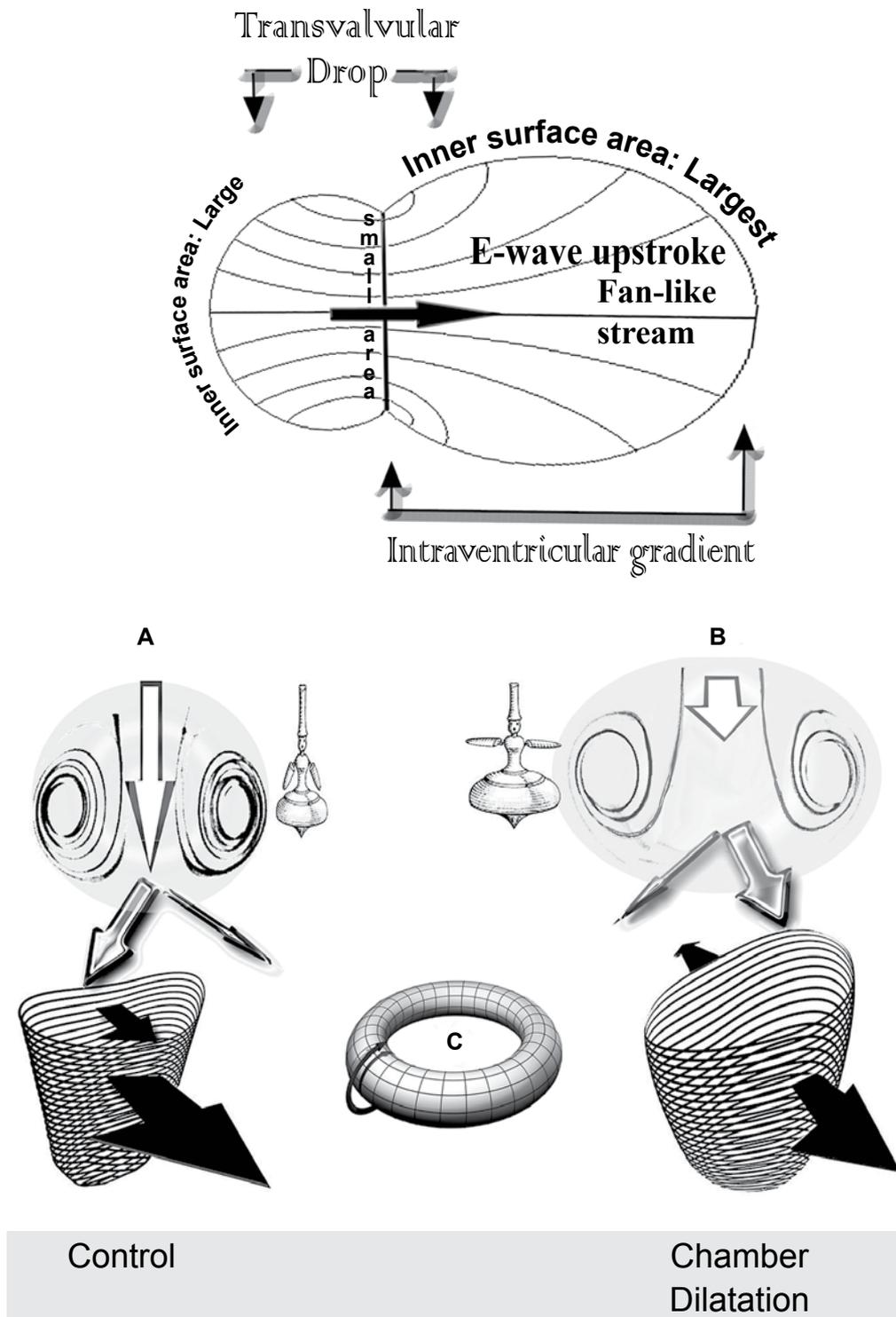


Figure 2. Fundamental fluid dynamic mechanisms pertaining to intracardiac diastolic flow. Top: During the E-wave upstroke, flow is confluent between the atrial endocardium and the atrioventricular valve orifice and diffluent between the latter and the ventricular chamber walls. There is convective acceleration up to the orifice and convective deceleration beyond it. Transvalvular pressure drops embody convective acceleration, whereas intraventricular gradients involve convective deceleration that counterbalances the simultaneous local acceleration gradient during the E-wave upstroke. Bottom: By increasing both intraventricular rotating blood mass and effective rotation radii, increased chamber size yields smaller recirculating velocities and vortex strength, as the “whirling dervish” tops suggest: with arms extended and wider girth, spinning is slower in B than in A. The stronger vortex ring in the normal-sized chamber (A) encroaches more powerfully on the central core than the weaker vortex in the enlarged chamber (B). Inset C gives a schematic representation of the toroidal vortex surrounding the inflowing core stream and rotating poloidally, i.e., in the sense of the curved arrow.

Using sophisticated digital processing of color Doppler M-mode recordings, Yotti et al⁵⁵ obtained spatiotemporal distribution measurements of LV diastolic intraventricular pressure gradients. Their findings in dilated LV cardiomyopathy and their conclusions are in complete agreement¹¹ with our previously published^{17,28,29} fluid dynamic analyses and propositions regarding the convective deceleration load, the diastolic ventriculoannular disproportion, and the impairment of E-wave peak velocities and E/A wave ratios in dilated ventricles. The Editorial accompanying the elegant study by Yotti et al in *Circulation* underscored this agreement.⁶⁷

Fluid dynamic concepts useful in managing RV/LV dilatation in heart failure

Conditions leading to eccentric hypertrophy and remodeling with chamber enlargement, as seen e.g. in congestive heart failure or after myocardial infarction, should increase DVAD and the Bernoulli-induced diastolic CDL while concurrently reducing the intensity of the filling vortex. These conditions have been shown to participate in the filling impairment linked to chamber enlargement.^{1,11,17,22,28-32,64} The converse applies for medical treatments and surgical interventions, such as the Batista procedure (partial left ventriculectomy), that reduce the size of dilated ventricles or attenuate/reverse ventricular remodeling and chamber enlargement.^{1,22,26} Medical interventions include cardiac resynchronization therapy to reverse the mechanical remodeling seen in congestive heart failure, and the administration of cell therapy or angiotensin-converting enzyme (ACE) inhibitors to attenuate ventricular remodeling and operating volume elevations after myocardial infarction. Such interventions should act, in part, by decreasing the CDL and by diminishing the DVAD,^{29,32} as well as by promoting stronger diastolic intraventricular vortical motions within the less-dilated chamber.²⁸

Conversely, DVAD and CDL should be augmented initially after surgical interventions (tricuspid or mitral ring annuloplasty), which decrease the effective tricuspid or mitral orifice area relative to the RV or LV chamber volume and inner surface area.⁶⁸ However, the ensuing regression of eccentric hypertrophy and chamber-size normalization remedy this transitory situation. Such fluid dynamic considerations point toward interesting directions for future clinical research, complementing conventional stud-

ies of RV/LV mechanics^{2-6,10,22,23,33-35,37,38,40,67,69,70} and aiming at better management of heart failure.

Conclusions

This article has summarized seminal concepts regarding diastolic function in ventricular dilatation, as accompanies heart failure from multiple etiologies, with primary emphasis on developing the useful framework of Figure 1 for managing ventricular diastolic filling problems. Fundamental fluid dynamic mechanisms can impair filling even in the absence of inflow valvular stenosis, or of abnormalities of myocardial relaxation and recoil and of effective ventricular diastolic compliance, especially in the setting of compensated or decompensated heart failure with chronic or acute RV/LV chamber enlargement/dilatation.

The filling vortex facilitates filling. The CDL is an important determinant of RV or LV inflow during the E-wave (A-wave) upstroke. The larger the chamber, the larger the CDL; this, in turn, strongly affects peak E- and A-wave velocities and underlies the clinically vital concept of DVAD. Vortical motions, ensuing after the E-wave peak, impound inflow kinetic energy, preventing an inflow-impeding Bernoulli pressure rise between inflow orifice and the expanding endocardial surface. This reduces the CDL by a variable amount, depending on vortical intensity, which decreases with chamber dilatation.

In conclusion, the preceding simple ventricular fluid dynamics perspective, with the accompanying flow-encompassing diastolic filling framework, complements conventional assessments of diastolic function in failure/remodeling by encompassing heretofore overlooked but crucial fluid dynamic aspects. Irrespective of any other possible coexisting myocardial and cardiac abnormalities, chamber enlargement in and by itself impairs diastolic filling by enhancing the convective deceleration load and by diminishing the strength of the beneficial, inflow-facilitating, diastolic intraventricular vortex.

Clinical perspective

Cardiac failure and ventricular dilatation are connected with increased morbidity and mortality. A comprehensive understanding of the mechanisms underlying diastolic dysfunction with dilatation is crucial for developing new management strategies and improving prognosis. Chamber enlargement causes generally unappreciated alterations in diastolic cardiac

flow dynamics, with important clinical implications. Convective deceleration is the deceleration (for mass conservation) of flowing blood due to convection (movement) through progressively larger-area cross-sections, from the inflow annulus toward the chamber walls during filling; it induces a convective (Bernoulli) pressure rise. The resultant convective deceleration load (CDL) is an important determinant of RV and LV inflow during the E-wave (A-wave) upstroke. The larger the ventricular chamber, the larger the endocardial surface and CDL. CDL magnitude strongly affects the attainable peak E-wave velocities measured by invasive and noninvasive imaging modalities in the presence of ventricular enlargement, regardless of co-existing myocardial dysfunction/abnormalities. This underlies the concept of diastolic ventriculoannular disproportion (DVAD). Vortical motions, ensuing after the E-wave peak, impound inflow kinetic energy, averting a convective pressure rise between the inflow orifice and the expanding endocardial surface. This reduces the CDL by a variable extent depending on diastolic RV/LV filling vortex intensity, which actually decreases with chamber dilatation. Accordingly, the diastolic vortex facilitates filling to varying degrees, depending on the ventricular chamber volume. These simple fluid dynamics considerations complement conventional assessments of diastolic function in failure and remodeling, contribute to our understanding of the mechanism of action of established (e.g. partial ventriculectomy, cardiac resynchronization, cell therapy, or ACE inhibitors) and new, yet to be developed, therapeutic interventions. They should not be overlooked in the clinical setting.

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