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RV instantaneous intraventricular diastolic pressure and velocity distributions in normal and volume overload awake dog disease models

Ares Pasipoularides,^{1,2} Ming Shu,² Ashish Shah,¹ Alessandro Tucconi,² and Donald D. Glower¹

¹Department of Surgery, Division of Cardiac and Thoracic Surgery, and ²Center for Emerging Cardiovascular Technologies, Duke University Medical Center, Durham, North Carolina 27710

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Pasipoularides, Ares, Ming Shu, Ashish Shah, Alessandro Tucconi, and Donald D. Glower. RV instantaneous intraventricular diastolic pressure and velocity distributions in normal and volume overload awake dog disease models. Am J Physiol Heart Circ Physiol 285: H1956-H1965, 2003. First published July 24, 2003; 10.1152/ ajpheart.00372.2003.—Intraventricular diastolic right ventricular (RV) flow field dynamics were studied by functional imaging using three-dimensional (3D) real-time echocardiography with sonomicrometry and computational fluid dynamics in seven awake dogs at control with normal wall motion (NWM) and RV volume overload with diastolic paradoxical septal motion. Burgeoning flow cross section between inflow anulus and chamber walls induces a convective pressure rise, which represents a "convective deceleration load" (CDL). High spatiotemporal resolution dynamic pressure and velocity distributions of the intraventricular RV flow field revealed time-dependent, subtle interactions between intraventricular local acceleration and convective pressure gradients. During the E-wave upstroke, the total pressure gradient along intraventricular flow is the algebraic sum of a pressure decrease contributed by local acceleration and a pressure rise contributed by a convective deceleration that partially counterbalances the local acceleration gradient. This underlies the smallness of early diastolic intraventricular gradients. At peak volumetric inflow, local acceleration vanishes and the total adverse intraventricular gradient is convective. During the E-wave downstroke, the strongly adverse gradient embodies the streamwise pressure augmentations from both local and convective decelerations. It induces flow separation and large-scale vortical motions, stronger in NWM. Their dynamic corollaries on intraventricular pressure and velocity distributions were ascertained. In the NWM pattern, the strong ring-like vortex surrounding the central core encroaches on the area available for flow toward the apex. This results in higher linear velocities later in the downstroke of the E wave than at peak inflow rate. The augmentation of CDL by ventriculoannular disproportion may contribute to E wave and E-to-A ratio depression with chamber dilatation.

intraventricular gradients; functional imaging; echocardiography; heart failure; ventricular function

UNDERSTANDING OF INTRAVENTRICULAR blood flow and pressure distributions during ejection has led to improved insights into systolic loading and ventricular function (15, 16). A similar understanding is currently being developed for left ventricular diastolic flow and transvalvular and intraventricular pressure gradients (23, 25–28). We have recently demonstrated (19) that large-scale vortical motions develop in the RV chamber during the downstroke of the E wave, similar to those occurring during left ventricular filling (1, 2, 23, 25– 28), and that chamber dilatation tends to reduce their intensity, especially when combined with diastolic paradoxic septal motion (PSM), as shown in Fig. 1.

Right-sided instantaneous intraventricular diastolic pressure gradients are smaller than their left-sided counterparts (5) and generally do not render themselves to reliable micromanometric multisensor catheter measurement. In contrast, transvalvular rightsided pressure differences can be measured under experimental hyperdynamic conditions (Fig. 2) and demonstrate dynamic characteristics, including timing relative to inflow velocity, similar to those reported for the left-sided atrioventricular transvalvular pressure drop by Isaaz (11), who applied a simplified linear momentum equation to its noninvasive assessment. In a patient with a nonobstructive mitral valve, in whom mitral flow and cardiac dimensions recorded by Doppler echocardiography were digitized and analyzed, the calculated mitral transvalvular pressure difference had already decreased considerably (11) at the time of peak velocity. The right-sided multisensor catheter measurements displayed in Fig. 2 exhibit analogous behavior. It is noteworthy that, according to Braunwald (3a), the diagnosis of tricuspid stenosis requires an atrioventricular mean gradient (by fluid-filled catheter) of only 2 mmHg and that a mean gradient of 5 mmHg is associated with overt systemic venous congestion. The smallness of the normal early RV diastolic intraventricular pressure gradients not only renders their measurement by catheter unreliable, it also conceals important fluid dynamic mechanisms that underlie them.

The present study was undertaken to characterize instantaneous dynamic pressure and velocity distribution patterns in the diastolic RV intraventricular blood flow field throughout the E wave. Albeit much smaller in magnitude than their systolic counterparts, diastolic intraventricular pressure gradients are linked to functionally significant, subtle, and more diverse dynamic

Address for reprint requests and other correspondence: D. D. Glower, Dept. of Surgery, PO Box 3851 Med. Ctr., Duke Univ., Durham, NC 27710.

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Fig. 1. Under control conditions of normal wall motion (NWM), filling is accompanied by anterior-directed motion of both the free and septal right ventricular (RV) walls (black arrows, A). In chamber dilatation with paradoxic septal motion (PSM), the septal motion is directed toward the left ventricle (back-pointing black arrow, B). Interposed is the intermediate pattern of dilatation with normal motion (gray arrows). By increasing both intraventricular mass and effective rotation radii, an increased chamber size leads to smaller effective recirculating velocities and vortex strength, as the "whirling dervish" tops suggest: with a wider girth and arms extended, spinning is slower in B than A. Although instantaneous volumetric inflow rates at control were smaller than with volume overload, after vortex development higher linear core velocities were present at control than with chamber dilatation. The more intense vortex ring squeezes the central core area more in A. The width of each arrow in the top panels is proportional to central core area; the length, to linear velocity.

phenomena. In particular, the smallness of normal early RV intraventricular pressure gradients is attributed to the partially counterbalancing actions of convective deceleration and local acceleration during the upstroke of the E wave. New perspectives have been gained on intriguing flow-associated facets of diastolic ventricular physiology and their modification by chamber dilatation.

METHODS

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Instrumentation and data acquisition. Because of the complicated dynamic RV geometry, this investigation required application of a recently developed functional imaging method, which comprises real-time, three-dimensional (3D) cardiac imaging and computational fluid dynamics (CFD). Figure 3 summarizes this method, which has been detailed in a recent publication (20). Geometric fidelity between the actual dynamic geometry of the RV chamber and its computational reconstruction is crucial to obtaining accurate CFD results. The functional imaging method has been experimentally validated (20) under control and volume overload conditions and shown (19) under both conditions to yield RV diastolic flow field velocity patterns that are in complete agreement with available left-sided experimental and clinical findings (1, 2, 23, 25, 28). Chronically instrumented, unrestrained large animals can be used in several serial or parallel studies within a period of a few weeks, following successful surgical sonomicrometric crystal implantation and recovery. Accordingly, real-time combined echocardiographic and sonomicrometric data for the present investigation were obtained in additional experiments on seven 20- to 30-kg dogs in which the method had been previously (20) validated.

The Duke University Institutional Animal Care and Use Committee approved animal care and procedures. With the dogs under anesthesia, sonomicrometric transducers were sewn as usual for dynamic RV chamber volume using the shell subtraction method (6, 7, 18, 20). Each dog recovered for 7–10 days before control combined 3D echocardiographic and sonomicrometric data acquisition in a lightly sedated (morphine 0.7 mg/kg) awake state. Sonomicrometric data were digitized at 400 Hz. Subsequently, volume overload (VO) was induced by tricuspid regurgitation according to a surgical protocol developed in our laboratory (18–20, 22), and combined echocardiographic and sonomicrometric data were acquired 2–3 wk later, similarly to control.

Method of tricuspid regurgitation. After control measurements, the dogs were sedated with intramuscular morphine (0.7 mg/kg). The right jugular vein was exposed, and a 8-Fr 25-cm introducer sheath was introduced into the right ventricle under fluoroscopic guidance. A 6-Fr urologic biopsy forceps (Circon Instruments; Santa Barbara, CA) was then advanced via the sheath, and multiple passes were taken to sever chordae until 3-4+ regurgitation was developed, with elevation of right atrial pressures assessed micromanometrically (Fig. 2*B*,*a*). The vein was ligated and the wound closed.

Multisensor catheter measurements. A 25-cm 8-Fr introducer sheath was passed through the exterior jugular vein to the right ventricle to allow passage of a custom-made rightheart catheter (Millar Instruments; Houston, TX). The multisensor catheter has two micromanometers spaced 5 cm apart; the distal micromanometer is at the tip and a Doppler velocimeter is 3 cm away from the distal micromanometer. The micromanometers and velocimeter were soaked in a saline bath at $36-38^{\circ}$ C for a minimum of 3 h; the micromanometers were simultaneously balanced, and all sensors were calibrated immediately before use.

Once proper functioning of the micromanometers and velocimeter had been verified, the catheter was advanced and guided by fluoroscopy until the velocimeter reached the tricuspid orifice. Proper positioning of the velocimeter ensured that the proximal and distal micromanometers were located inside the right atrium and the right ventricle, respectively. In a steady physiological state, right atrial and ventricular pressures and linear tricuspid inflow velocity were obtained and digitized at 400 Hz (Fig. 2B). As pointed out above and discussed further in Functional imaging compared with multisensor catheterization, reliable multisensor catheter measurements of pressure gradients in the RV diastolic intraventricular flow field are not physically possible. Transvalvular (Fig. 2A) atrioventricular pressure differences such as those presented in Fig. 2B, b are, in fact, indicative of the best that is achievable by direct multisensor ("high-fidelity") catheter measurement.

3D real-time echocardiography and image segmentation. Real-time 3D images were obtained using the Volumetrics (Durham, NC) real-time 3D-ultrasound scanner (RT3D). RV endocardial border detection yielded individual inner chamber surface points, which were converted into 3D Cartesian coordinates and stored in a four-dimensional matrix (Fig. 3, *inset 1*). Each matrix represented the chamber surface throughout one cardiac cycle.

Reconstruction of endocardial border points and mesh generation. The extracted endocardial edges were in layered form and were processed to generate instantaneous inner surface coordinates in 3D. To obtain flow field simulations at a temporal resolution of 400 Hz, it was necessary to approxH1958



imate RV geometry at instants between successive RT3D images. Under steady-state conditions, RV geometry was calculated at these intermediate times as a quadratic weighted average of the geometry in contiguous RT3D frames. Representative RV reconstructions are depicted in Fig. 3, inset 2. Dynamic RV chamber volume, using SSM and sonomicrometric dimensions, and its time derivative or "inflow rate" were also calculated and compared with those obtained from the RT3D data (cf. Fig. 3, inset 3). This allowed adjustment of the instantaneous volume and velocity boundary conditions in the CFD simulations as needed, according to a scheme detailed in a recent paper on the method (20). Mesh generation followed, using a combination of FIDAP (or fluid flow analysis CFD package) and proprietary software, to obtain the instantaneous meshes representing the dynamic 3D domain for the simulation of the RV intraventricular diastolic flow field (Fig. 3, inset 4).

Computer simulations, postprocessing, and plots of flow variables. The instantaneous boundary condition assigned to each external nodal point was the velocity vector describing the direction and speed of instantaneous motion of the point (Fig. 3, inset 4). Simulations were carried out on a CRAY

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Fig. 3. Functional imaging comprises three parts. First, a semiautomated segmentation aided by intraluminal contrast medium locates the RV endocardial surface. Second, a geometric scheme for dynamic RV chamber reconstruction applies a time interpolation procedure to the RT3D data to quantify wall geometry and motion at 400 Hz. Finally, the RV endocardial border motion information is used for mesh generation on a computational fluid dynamics (CFD) solver to simulate development of the early RV diastolic inflow field. Boundary conditions (tessellated endocardial surface nodal velocities) for the solver are directly derived from the endocardial geometry and motion information. Postprocessing visualization provides flow field dynamic plots of pressure and velocity. See text and Refs. 19 and 20 for more details.

T-90, assuming that blood is a Newtonian, incompressible fluid, with kinematic viscosity of 0.04 Stokes, and mass density of 1.05 g/cm³, and that intraventricular flow is governed by the Navier-Stokes equations. The CFD solutions yielded values of the dependent flow field variables (velocity and pressure) at literally thousands of discrete points in space and time within each RV chamber simulated. From these "high-density of information" data sets, FIDAP's postprocessing module extracted high resolution plots of instantaneous intraventricular flow velocity and pressure distributions (Fig. 3, *insets 5a* and 5b, and Figs. 4–7).

RESULTS

Intraventricular flow field. All seven dogs exhibited normal wall motion (NWM) at control; three had normal and four had diastolic paradoxical septal motion (PSM) in volume overload (Fig. 1). In the following sections, we examine throughout the entire E wave, illustrative RV intraventricular pressure and velocity distributions obtained by the functional imaging method in individual dog hearts exhibiting NWM at control and PSM in volume overload. The differences illustrated in Figs. 4–7 between volume-overloaded and normal-size chambers are only quantitative, i.e., they pertain to "degree or intensity" rather than qualitatively different phenomena. Development of axial velocities and pressures throughout the E wave. Figure 4A shows the peak linear velocity on the RV axis (V_{max}) throughout the E wave. The peak volumetric inflow rate (E_{pk}) ensues 37.5 ms after the onset of RV filling at control and 32.5 ms in VO. As exemplified in this typical dog heart, in volume overload with PSM the instant at which V_{max} reaches its E wave maximum tends to coincide with the instant of E_{pk} (Fig. 4A, arrow). On the other hand, in the control (NWM) pattern, the value that V_{max} reaches at the time of E_{pk} is only a local maximum. It is only in the final period of the E wave, as the velocity rises continuously and far exceeds the level attained at the time of E_{pk} , that the maximum value of V_{max} is attained.

Figure 4B demonstrates the evolution of the corresponding intraventricular axial RV diastolic pressure gradient. Arrows again indicate $E_{\rm pk}$ times. For both the NWM and the PSM patterns, inception of RV diastolic filling is associated with a sharp decline in the value of apical pressure (ΔP) from the pressure at the tricuspid anulus. This corresponds to an accelerating (negative) pressure gradient. Upon reaching an early minimum, ΔP then starts to rise toward zero for both patterns as the instant of $E_{\rm pk}$ is approached. For both patterns, the instant of $E_{\rm pk}$ ensues within 2.5 ms of the instant when ΔP first becomes positive. ΔP remains above zero after



Fig. 4. A: peak linear velocity along the RV inflow axis ($V_{\rm max}$) through the E wave for NWM and volume overload with PSM. Note that with NWM, $V_{\rm max}$ continued to rise late in the E wave although the volumetric inflow rate was decreasing. B: development of the axial RV intraventricular pressure gradient ($\Delta P =$ apical pressure – tricuspid anulus pressure) throughout the E wave. ΔP inverts from negative (favorable) to positive (adverse) around the time of peak volumetric inflow velocity ($E_{\rm pk}$) and remains positive throughout the remainder of the E wave. Note that more prominent fluctuations in the magnitude of the pressure gradient occur after $E_{\rm pk}$ with NWM than under volume overload with PSM. Such fluctuations are indicative of strong large-scale vortical motions. The axial pressure distributions that correspond to *instants* A–J are discussed in detail in the text and in Figs. 5 and 6.

the occurrence of $E_{\rm pk}$, reflecting an adverse (positive) gradient for flow toward the apex. For both patterns, the downstroke of the E wave was associated with low-frequency fluctuations of the pressure gradient. In the last 20–30 ms of the E wave, the fluctuation was much more prominent in the control (NWM) pattern, as shown in Fig. 4B.

Axial pressure distributions around the time of E_{pk} . The time of E_{pk} is a critical point in the development of the axial intraventricular pressure distribution: before it, global inflow is accelerating (E-wave upstroke), whereas following it, global inflow is decelerating (Ewave downstroke). Accordingly, the period between 20 and 42.5 ms from the onset of the E wave, which straddles E_{pk} , was subdivided into 2.5-ms intervals (*instants* A–J in Fig. 4B) and analyzed in detail. Representative pressure distribution "snapshots" are shown in Figs. 5 and 6.

Figure 5 shows the pressure distribution, in reference to tricuspid anulus pressure, along the inflow axis for the control (NWM) condition. The interval illustrated began with *instant* C (i.e., 25 ms from the onset of the E wave) and ended with *instant* J (i.e., 42.5 ms from the onset of the E wave); $E_{\rm pk}$ corresponded to *instant* H (cf. Fig. 4B). *Instants* C-J correspond to "snapshots" of the pressure distribution, which show its gradual transformation in time.

The time of E_{pk} (Fig. 5, *instant* H) is pivotal for this transformation. Before this time, the pressure declines through most of the inflow axis to levels lower than that at the tricuspid anulus: this yields a favorable pressure gradient for flow toward the apex. The decline is monotonic for Fig. 5, *instant C*. For *instants D–G* in Figure 4B (cf. Fig. 5, *instants F* and *G*), a rise in pressure within the first centimeter beneath the inflow orifice precedes the monotonic decline in the remainder of the inflow axis. At the instant of E_{pk} (Fig. 5, *instant H*), pressure rises in the first centimeter from the orifice, shows a slight depression in the midregion of the axis, and increases again toward the apex. After E_{pk} , the pressure increases monotonically away from the tricuspid anulus toward the apex, and most of the rise occurs within the first centimeter of the inflow axis (Fig. 5, *instants I* and J). This yields an adverse (positive) pressure gradient for flow toward the apex and augurs flow field instability.

Figure 6 presents pressure computed at the successive instants labeled in Fig. 4B and is comparable to Fig. 5. Figure 6 provides representative snapshots of the development of the pressure distribution for the condition of volume overload with PSM. The pressure differences along the inflow axis were higher in volume overload with PSM; however, the development pattern before, at, and after the instant (Fig. 6, *instant F*) of $E_{\rm pk}$ was similar to that of Fig. 5.

Development of intraventricular RV axial velocities throughout the E wave. Figure 7 presents intraventricular velocity distributions along the RV inflow axis from a typical dog heart. Figure 7A pertains to control with NWM, and Fig. 7B pertains to volume overload with PSM. Three "snapshots" are overlaid for each state: the first followed the onset of the E wave by ~ 10 ms; the next corresponds to the peak of the E wave; the last preceded the end of the E wave by ~ 10 ms.

At any given instant of time, and for both operating states under examination, blood velocity decreases away from the inflow orifice toward the apex, reflecting convective deceleration of the intraventricular flow, strongest in the vicinity of the tricuspid anulus. The

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Fig. 5. Normal wall motion. Apportionment of the instantaneous axial pressure gradient along the main inflow axis of the RV chamber around the time of the peak of the E wave. *Instants C-J*: pressures computed at the successive instants of time labeled in Fig. 4B. Note that at the time of peak volumetric inflow rate (*instant H*, $t = t_{E_{pk}}$), the pressure distribution is transformed from a predominantly favorable (accelerating) to a predominantly adverse (decelerating) axial pressure gradient, in reference to apically directed flow. Arrowheads at the *right* margin of each panel indicate the zero pressure level with reference to tricuspid anulus pressure.

magnitudes of the linear velocities are very low beyond the initial 2.0 cm, or so, of the inflow axis. This is a kinematic evidence for the intense convective deceleration in the proximity of the inflow orifice. It is distinct from the flow field visualization-derived evidence, embodied in previously reported (19) instantaneous color flow maps and velocity vector renderings.

The two patterns differ primarily in the magnitudes of their velocity distributions during the downstroke of the E wave. In volume overload with PSM, the magnitudes of the corresponding velocity distributions were roughly proportional to those of the volumetric inflow rates applying at the three instants of time. Thus, at peak inflow rate, the axial linear velocity ensemble was also at its maximum echelon above the abscissa (Fig. 7*B*). In distinct contrast to this, under control (NWM) conditions, late in the E wave the linear velocity magnitudes in the basal and midchamber region of the axis rose to levels substantially higher than their levels at the instant of peak volumetric inflow (Fig. 7A). In fact, these basal region velocity levels late in the E wave were under control conditions comparable to the enhanced velocity levels applying at E_{pk} during volume overload.

DISCUSSION

Left ventricular filling dynamics and transvalvular and intraventricular pressure-flow relationships have been studied experimentally by various groups (1, 2, 4, 5, 11, 13–15, 23, 25, 26, 28), most recently through elegant, meticulous experimental measurements of intraventricular pressure and velocity on anesthetized, thoracotomized dogs (27). Earlier experiments had demonstrated (5) that right-sided patterns of diastolic pressure gradients between inflow anulus and apex are similar to left-sided ones, but significantly smaller in magnitude. The present study describes detailed distributions of intraventricular pressure and velocity and contributes to our understanding of RV diastolic fluid dynamics.

We formulated the hypothesis (19) that throughout the upstroke of the E wave there should be a convective deceleration and a rise in pressure in the forward flow direction between inflow orifice and chamber walls, and this was corroborated by the present findings (Figs. 4–7). This convective rise in pressure is proportional to the square of applying intraventricular velocities and is higher under daily activities and with increased venous return than in the lightly sedated experimental state.

Convective effects should strongly impact velocity and pressure. Combined with the accentuation of the adverse pressure gradient by the local deceleration of flow during the downstroke of the E wave, this leads to flow field instability, flow separation, and large-scale H1962







vortical motions (19). The present high spatiotemporal resolution pressure and velocity distribution findings complement visualization maps of the intraventricular velocity field (19, 20).

Intraventricular pressure field and its gradient. The spatial distribution of the time-varying intraventricular pressure values obtained by the CFD simulations during the filling process, determines the instantaneous force acting on flowing blood particles within the filling chamber. The assemblage of the unsteady pressure values on all of the intraventricular flow field points is the instantaneous pressure field.

A major advantage of the functional imaging method is that much more extensive information can be extracted compared with experimental measurements; it yields values of the field variables at literally thousands of discrete points in space and time. From this high-density information, we can extract informative snapshots of instantaneous velocity and pressure distributions, as is demonstrated in Figs. 4–7. Catheterization measurements, on the other hand, traditionally (1, 2, 4, 5, 13-15, 23-26, 28) have been limited to global quantities (chamber "pressure" values in Fig. 2A) or to values at a small number of points in space and time. It is impossible to obtain through multisensor (micromanometric/velocimetric) catheters high-density spatiotemporal diastolic pressure and velocity distributions, such as those in Figs. 4-7. Thus functional imaging reveals information on important dynamic flow behavior that was previously inaccessible. It is instructive to analyze the instantaneous distribution of pressure along the line of the chamber flow axis, as we have done in our studies of the fluid dynamics of ejection (3, 8-10, 12, 16, 17, 21). Discrete points along the axis exhibit different patterns of pressure values at any given time during filling.

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Local and convective components of the diastolic pressure gradient. Convective effects significantly impact diastolic intraventricular velocity distributions and pressure gradients. With the exception of the instant of peak volumetric inflow (dQ/dt = 0), both local and convective accelerations make their individual contributions to the total pressure gradient in the course of the E wave. At any given instant, the velocity of blood flow tends to decrease away from the inflow orifice toward the apex (Fig. 7).

Because acceleration is associated with a drop in pressure in the flow direction, the local acceleration $(\partial v/\partial t)$ component causes a pressure decrease from the orifice to the apex during the upstroke of the E wave. The total intraventricular pressure gradient is then the algebraic sum of the pressure decrease contributed by the local acceleration and the pressure rise contributed by the convective deceleration. This convective





Fig. 7. Velocity distributions along the main inflow axis of the RV chamber. A: results from functional imaging simulations under control conditions (NWM). B: results from simulations under volume overload with PSM. Note that under control conditions, the velocities along the axis in the basal region of the RV chamber are substantially higher late in the E wave than their levels at $E_{\rm pk}$, although the volumetric inflow rate is then much lower. It is a corollary of conservation of mass or "continuity of flow" and is graphically explained by the *insets* accompanying the velocity distribution plots. The length of the arrows is proportional to linear velocity; their width, to volumetric inflow rate.

intraventricular pressure rise (Figs. 5 and 6) during the upstroke of the E wave had not been previously demonstrated.

At $E_{\rm pk}$ the local acceleration component vanishes, and the total pressure gradient is convective. In the E-wave downstroke, the strongly adverse pressure gradient is the sum of pressure augmentations along the flow path from both local and convective decelerations. Soon after the onset of the downstroke of the E wave, the overall adverse pressure gradient causes flow separation and inception of recirculation with a vortical motion surrounding the central inflowing stream (19). Subsequent interaction of the central stream with the expanding vortex, which encroaches on it, is responsible for the continuous rise of the peak RV axis velocity throughout the downstroke of the E wave that is typical under normal conditions (NWM) and is demonstrated for the first time, to our knowledge, in Fig. 4.

Interplay of convective with local acceleration effects underlies nonuniformity of the pressure gradient along the chamber axis. The monotonic decrease in pressure along the inflow axis epitomized in Fig. 5, *instant C*, reveals that the local acceleration effect completely dominates the convective during the first 25 ms of the upstroke of the E wave. During the subsequent interval up to $E_{\rm pk}$, the impact of an intense convective deceleration becomes prominent in the segment of the axis immediately beneath the tricuspid orifice. During this interval, although the volumetric inflow rate continues to accelerate toward its peak, the acceleration magnitude has begun to diminish. Hence, the favorable (negative) pressure gradient contributed by the local acceleration is reducing in magnitude. On the other hand, the magnitude of the inflow velocity near the tricuspid orifice has risen to higher levels. Consequently, the adverse pressure gradient contributed by the convective deceleration now overpowers in the first 1–2 cm of the inflow axis the favorable pressure gradient contributed by the local acceleration. This explains why pressure is rising in the first 1-1.5 cm of the axis and decreasing in the remaining 6.5-7 cm (Fig. 5, instants F and G). The reduction in velocity. because of the intense convective deceleration in the immediate vicinity of the tricuspid orifice (cf. Fig. 7), allows the local acceleration to regain its dominance, generating a pressure drop in the remaining segment of the inflow axis, after the initial 1-2 cm.

Because at the instant of the peak volumetric inflow the local acceleration vanishes, the convective deceleration contributes exclusively to the total pressure gradient, resulting in a predominantly monotonic pressure rise along the inflow axis (Fig. 5, *instant H*). The sharp rise in the magnitude of the adverse pressure gradient near the tricuspid inflow orifice is notable.

During the downstroke of the E wave, the local acceleration turns into local deceleration. The local and the convective components now both add to the total pressure gradient. As the rate of deceleration increases, so does the magnitude of the pressure rise, as seen in Fig. 5, *instants I* and *J*, and this leads to subsequent flow separation and vortex formation (cf. color maps in Ref. 19).

The rise in the linear velocity within the basal region of the chamber during the downstroke of the E wave under control conditions is an up to now unrecognized phenomenon. It is a corollary of the large-scale vortical motion, which develops within the chamber and is intense at control but diminishes in strength and extent with chamber dilatation. The greater filling vortex strength at control manifests itself dynamically in the more prominent pressure fluctuations late in the E wave in Fig. 4.

Figure 6 shows a qualitatively similar spatiotemporal development pattern of the pressure gradient along the inflow axis in volume overload with PSM. For instance, the pressure distribution at the time of $E_{\rm pk}$ (Fig. 6, *instant F*) has the same characteristics as that of Fig. 5, *instant H* ($E_{\rm pk}$ panel). In the dilated chamber there is, however, a much sharper accentuation of the pressure gradient near the inflow orifice, a result of diastolic ventriculoannular disproportion.

Analogous distributions demonstrating that the overwhelming proportion of the intraventricular convective acceleration occurs in the immediate vicinity of the outflow orifice were found in studies of ejection (8-10, 12, 16, 17, 21).

Clinical implications: convective deceleration load and ventriculoannular disproportion. The dynamic pressure and velocity distributions in Figs. 4-7 corroborate flow visualization findings (19, 20) that led us to formulate the "convective deceleration load" (CDL) mechanism. The greater the discrepancy, or "diastolic ventriculoannular disproportion," between the sizes of the endocardial surface and the inflow orifice, the larger the CDL (Fig. 8). The rise in pressure representing CDL is proportional to the square of applying velocities. From this, a larger ventricle in early diastole engenders a disproportionate increase of CDL and a much more difficult inflow. Matters should be worsened with tachycardia, which accompanies heart failure. Such augmentation of CDL may contribute to depressed E-wave amplitude and E-to-A ratio abnormalities in acute or chronic chamber dilatation. Ensuing atrial overload (active support of filling) may account for the association (R. Paterson, personal communications) of atrial fibrillation with clinical ventricular enlargement in absence of coronary disease.

Doppler echocardiographic implications. Previous papers, discussed by Isaaz (11), had demonstrated that Doppler echocardiography using the simplified Bernoulli equation is not accurate for calculating the transvalvular pressure drop across a nonobstructive mitral valve and had emphasized the contribution by local acceleration to diastolic transvalvular pressure differences across a nonobstructive mitral valve.



Fig. 8. Graphic illustration of the concepts of convective deceleration load (CDL) and diastolic ventriculoannular disproportion (VADP). CDL accrues from the strong deceleration that the inflowing blood experiences during the E-wave upstroke, when "fan-like" flow prevails as a result of the great increase in flow cross-sectional area from tricuspid (mitral) anulus to expanding chamber walls. The greater the inflow rate (Q^2) and the larger the VADP, the greater is the magnitude of CDL.

However, the present work is the first to distinguish the mutually opposed effects of the local acceleration and convective deceleration during the upstroke of the E wave on the intraventricular flow field dynamics. Our findings demonstrate that application of the simplified Bernoulli equation even at the time of the peak of the E wave, when local acceleration vanishes, to calculate an intraventricular pressure decrease makes no sense. Rather than a convective decrease, at the E-wave peak there is a convective increase in the intraventricular pressure away from the tricuspid (mitral) anulus, and flow persists under its previously built-up momentum.

The physiological role of the ventricular vortex in promoting diastolic filling. The pressure and velocity data presented in Figs. 4–7, especially the pressure plots in multipanel Figs. 5 and 6 and their interpretation, corroborate the hypothesis (19) that the dissipative vortical structures facilitate filling and attainment of higher volumes (larger endocardial surface areas) by robbing kinetic energy that would otherwise contribute to the pressure rise representing CDL (Fig. 8).

Functional imaging compared with multisensor catheterization. Multisensor catheters can derive pressure and velocity satisfactorily in many applications, especially involving the stable, "irrotational" (16) intraventricular field of ejection (3, 15-17, 21, 24). However, catheters inside the flow field can directly disturb it. In the case of the RV inflow field, catheter measurements are not practicable. It is impossible to obtain intraventricular pressure and velocity distributions, as in Figs. 4-7, through multisensor catheters. Unavoidable motions of any catheter buffeted by onrushing and whirling flow, global motions of the heart, and, in the case of pressure, attendant hydrostatic effects on the micromanometric sensors vitiate any attempt to measure along the inflow axis instantaneous high axial resolution distributions of pressure and velocity. It is likewise impossible to obtain by catheter high-information-density data series in successive "snapshots," as in Figs. 5-6. Transvalvular atrioventricular pressure differences such as those presented in Fig. 2*B*,*b* exemplify what is at best achievable by direct multisensor catheter measurement; the sensors on the catheter are not really fixed along the inflow axis through the measurement process.

Functional imaging results are in harmony with fundamental fluid dynamics theory and with the experimental in vitro findings on mechanical left heart models by Bellhouse (1, 2) affirming that dilatation decreases vortex strength in the mechanical left ventricular chamber. Moreover, FIDAP results (9, 10) agreed closely (16) with another CFD code developed in our laboratory (8) and with analytic (12, 21), semiempirical correlation (17), and similarity (16) solutions in our investigations of the intraventricular left ventricular ejection flow field, under diverse clinical conditions.

Previously inaccessible subtleties (14, 15) in the dynamics of the RV diastolic flow field can now be investigated by functional imaging, and important but formerly unrecognized aspects of pressure and flow dy-

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namics during ventricular filling can be examined. As shown in Figs. 4-7, inspection of the time-dependent distributions of flow field variables provides a detailed insight into filling dynamics, akin to an ideal high-resolution catheterization experiment.

In conclusion, this is the first work to properly recognize, characterize, and distinguish the complex contributions by local and convective acceleration gradients to diastolic instantaneous intraventricular pressure and velocity distributions in normal and dilated diseased ventricles. It has shown that the smallness of normal (nonobstructive) early RV diastolic intraventricular pressure gradients for inflow is referable to the partially counterbalancing actions of convective deceleration and local acceleration components working concurrently during the upstroke of the E wave. The same mechanism should account for the small left ventricular intraventricular gradients. Detailed dynamic intraventricular pressure and axial velocity distribution patterns have corroborated the concepts (19) of a convective deceleration load and the useful role of the diastolic ventricular vortex in promoting filling. Acquisition and interpretations of Doppler and catheterization measurements should consider such effects more closely than in the past.

The utility and applicability of functional imaging will surely increase rapidly in the near future, with forthcoming advances in digital imaging modalities, computing, and scientific visualization.

DISCLOSURES

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