Nature Optimizes the Swirling Flow in the Human Left Ventricle

Gianni Pedrizzetti*
Dipartimento Ingegneria Civile, Università di Trieste, Piazzale Europa 1, 34127 Trieste, Italy
Federico Domenichini†
Dipartimento Ingegneria Civile, Università di Firenze, V. Santa Marta 3, 50139 Firenze, Italy
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The asymmetry of the blood flow in the human left ventricle is commonly assumed to facilitate the following ejection of blood in the primary circulation. The intraventricular flow is here studied by the numerical solution of the governing equations written in a prolate spheroid geometry with moving walls. The physiological parameters are taken from pediatric clinical data; then, the entering jet is artificially displaced to modify the asymmetry of the flow. The analysis of flow patterns confirms that the physiological case looks to best comply with the transition from the filling to the ejection phase. The flow energy dissipation is found to be minimized about the physiological conditions. An unnatural asymmetry, as given by cardiac diseases or valvular replacement, could reduce the efficiency of the heart pump by over 10%, thus augmenting the work required by the cardiac muscle.

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The mechanics of the blood flow inside of the heart chambers represents a central issue of the biological research. Despite the allowance of imaging diagnostic tools that permit, to some extent, intraventricular flow visualization, and of some experimental and numerical studies, several aspects are still not clear in their relation to physiology. The visualization of flow in the heart chambers is, in fact, still of little usage in diagnosis. For this, the challenge is represented by the search for a synthetic description of the available information and its relation with the heart function. The present work is centered on the fluid dynamics of the left ventricle following the path drawn in previous studies, with the goal to show some evidence that the physiological geometric arrangement of the left ventricular chamber looks naturally designed to minimize the energy dissipation of the flow. Analogous issues are found in technological systems where a chamber is inserted in a fluid conduct to enhance the local mixing or heat exchange. Here the asymmetric location of inlet and outlets is often designed to create a symmetry break of the flow pattern that enhances the local diffusion and also increases the dissipative phenomena.

The fluid dynamics in the human left ventricle has been investigated by several physical [1–3] and computational models [4–8]. These and current imaging techniques, like echocardiography [9,10] and magnetic resonance [11,12] that allow nowadays its direct visualization, give a general description that can be synthesized as follows. The flow is primarily characterized by a strong compact jet that enters the chamber through the mitral orifice during the ventricular filling (diastolic) phase. The jet has two distinct pulses: the early-filling wave (E wave), during ventricular relaxation, followed by the A wave produced by the atrial contraction. At the end of the diastole, the ventricle begins to contract (systole), the active phase produced by the action of the myocardium. In systole, pressure rises in the chamber, the mitral valve closes (to prevent backflow) while the aortic valve opens and, through this, blood is ejected in the primary circulation.

Notwithstanding the apparent simplicity of the cardiac cycle, the intraventricular fluid dynamics presents a variety of complex phenomena that have an influence on the heart function. The diastolic entering jet has an almost irrotational core surrounded by a shear layer that rolls up and immediately arranges into a compact ring-shaped vortex structure (the jet head) that enters the cavity. Moreover, as the orifice is displaced with respect to the axis of the cavity, the jet is redirected toward one wall [8,12]. Flow visualizations, made on a vertical plane cutting the ventricle and the inlet orifice, show the development of a persistent recirculating cell [1,2], which gives evidence of the eventual asymmetric blood pattern as sketched in Fig. 1(a). The

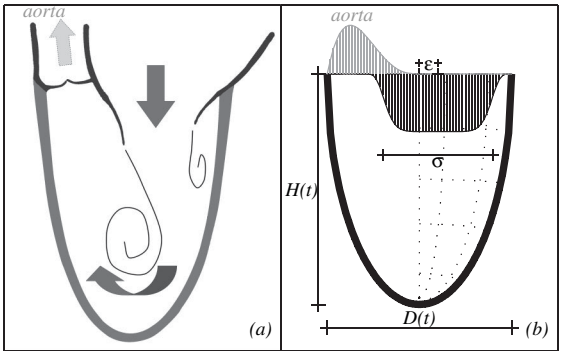

FIG. 1. Sketch of the physical problem on a vertical plane across the ventricle: (a) the flow that enters through the mitral orifice organizes itself in a circulatory pattern that redirects the flow to the aorta; (b) schematic representation of the velocity profiles and model parameters.

recirculating cell is considered to act as a sort of reservoir that stores some kinetic energy and facilitates the blood ejection [1,2,8,12,13]. Therefore the flow asymmetry seems to play a role in reducing the energy dissipation and, in turn, in diminishing the energy that the myocardial muscle must produce to eject the blood into the primary circulation. This work is devoted to verify whether this asymmetric intraventricular flow arrangement influences the heart function.

A model of the blood flow inside the left ventricle is employed for the study. It is based on the numerical solution of the three-dimensional Navier-Stokes equation into a half prolate spheroid geometry with moving walls [8]. The chamber geometry is thus defined by the time-varying functions $D(t)$ and $H(t)$ (t is the time) that are the temporal variations of the equatorial diameter and of the major semiaxis. They specify the variation of the ventricular volume $\mathcal{V}(t)$, and represent the forcing of the system. The valves are assumed to be circular and held in a fixed open or closed position depending on the sign of $d\mathcal{V}/dt$. Given the entering flow $d\mathcal{V}/dt > 0$, a blunt velocity profile is assigned at the equatorial plane

$$v(r, \theta) \propto \exp\left\{-\left[\frac{(r\cos\theta - \varepsilon)^2 + (r\sin\theta)^2}{\sigma^2}\right]^4\right\}, \qquad (1)$$

where (r, θ) are polar coordinates [8]. In the profile (1), sketched in Fig. 1(b), the parameter σ is the characteristic dimension of the jet and ε is its displacement with respect to the central axis. The exit (aortic) profile is taken proportional to $r^3(1 - r)\exp\{-(\theta - \pi)^2/\sigma^2\}$, although its shape has little or no influence on the intraventricular flow. The system is thus completely defined by the time-varying geometry $D(t)$, $H(t)$, and by the parameters σ and ε in the profile (1). In dimensionless terms, the flow is characterized by the Stokes and Strouhal numbers, $\beta = D_0^2/(\nu T)$ and $\mathrm{St} = D_0/(U_0 T)$, where $\nu = 3 \times 10^{-6}$ m^2/s is the kinematic viscosity of the blood, assumed to be a Newtonian fluid. T is the heartbeat period [the period of $D(t)$ and $H(t)$], the reference length scale, D_0, is taken as the diameter at the beginning of the filling phase [the minimum value of $D(t)$], and U_0 is the peak inflow velocity $4\max[(\sigma D)^{-2} d\mathcal{V}/dt]/\pi$.

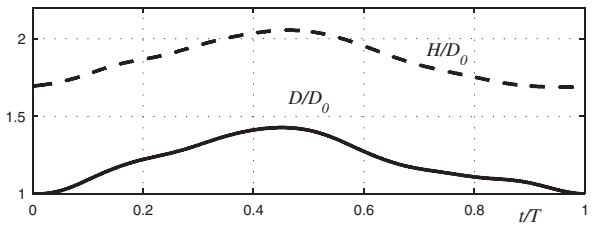

FIG. 2. Time variation of the geometry parameters, $H(t)$ and $D(t)$, as evaluated from an echocardiographic study of a prematurely born child weighing 0.8 kg. The reference length and time scales are $D_0 = 9.2$ mm and $T = 480$ ms, respectively.

In the present work, the required parameters and functions were extracted from an echocardiographic study of a healthy early-born child of 0.8 kg. This choice was driven by the computational effort that presently limits the numerical application to small hearts (pediatric studies). The profiles $D(t)$ and $H(t)$ are shown in Fig. 2 and correspond to a Stokes parameter $\beta = 54$. The diameter of the mitral jet σ was estimated to be equal to $0.65D$ during the first pulse (E wave) and $0.45D$ during the second pulse (A wave), and the resulting value of the Strouhal number is $\mathrm{St} = 0.07$. The eccentricity was estimated about $\varepsilon = 0.125D$.

The Navier-Stokes equations [14] are written on a prolate spheroid moving system of coordinates and numerically solved with a spectral representation along the azimuthal coordinate and centered second order finite differences on the meridian plane. Time is advanced using a fractional step method with a third order Runge-Kutta scheme. The system is started from rest and the computations have been carried for several periods to ensure the periodicity of the solution that is already found on the third period. The solutions have been obtained with a 48×96 grid in the meridian plane and 8 harmonics in the azimuthal direction. The details about the mathematical formulation, the numerical method, and the required spatial resolutions are given in [8].

The overall development of the intraventricular dynamics has been described above and reported in detail in [8]. A snapshot of the solution at the end of the filling period, when the inlet flow is approaching zero before the start of contraction, is given in Fig. 3. Figure 3(a) shows the velocity vectors and the distribution of vorticity on the transversal (symmetry) plane. In Fig. 3(b), the three-dimensional flow structure is presented in terms of the distribution of the scalar λ_2, whose negative extremes

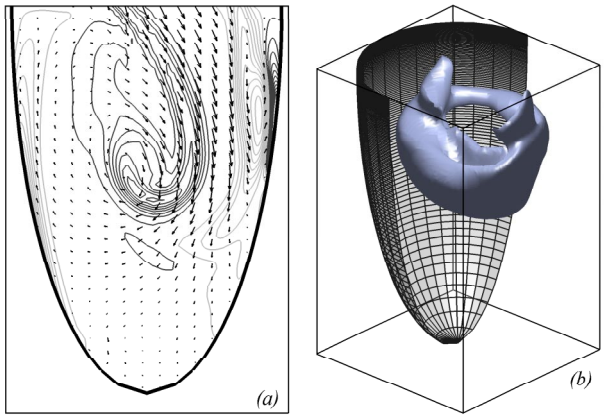

FIG. 3 (color online). Flow field for the natural case $\varepsilon = 0.125D$, at $t = 0.4T$. (a) Velocity vector and distribution of the vorticity on the symmetry plane; vorticity levels from -315 to 315, step 30, negative (clockwise) levels are black, and positive levels are light gray; (b) isosurface of the vortex indicator λ_2 at $\lambda_2 = -1500$.

correspond to the trace of coherent vortices [15]. As suggested in [12], the entering jet is redirected toward the nearby wall [right of Fig. 3(a)]. The jet head, formerly a vortex ring, is partly dissipated on that side, while the opposite vortex portion eventually occupies the center of the cavity. This asymmetric arrangement is seen, on the transversal plane, as that of a central vortex structure that dominates the entire flow field. The circulating flow at the end of diastole appears to be naturally invited toward the opening aortic valve. A careful analysis of the vortex structure further clarifies that the out-of-plane three-dimensional flow converges toward the same region and enhances the two-dimensional picture [13].

The natural asymmetric flow arrangement looks favorable to facilitate the blood ejection. This asymmetry is a consequence of the physiological eccentricity ε of the mitral orifice. Potential modifications of the flow are here analyzed by artificially varying the eccentricity parameter. The solution on the symmetry plane in the nearly symmetric condition, $\varepsilon = 0.02D$, is shown in Fig. 4(a) at the end of the filling phase. The flow field presents an approximately central jet that develops into a vortex pair (a cross section of a vortex ring) that is almost symmetric and induces a weak lateral backflow toward the aortic opening. The case $\varepsilon = 0.25D$, larger than the physiological value, is shown in Fig. 4(b); the jet is strongly redirected toward the lateral wall, and the vortex head on that side closely interacts with the wall boundary layer. This effect leads to secondary vortex structures, and it is expected to increase local energy dissipation. The backflow toward the aorta also appears to be weaker than in the physiological case of Fig. 3. It must be pointed out that the dynamics here discussed is a two-dimensional view of a more complex three-dimensional flow field, where the development of the axial component of the vorticity, Fig. 3(b), facilitates the flow redirection toward the aortic orifice [8]. The 3D view of the vorticity structure for the cases of Fig. 4, here not reported, shows the almost absence of axial vorticity for $\varepsilon = 0.02D$; in the case $\varepsilon = 0.25D$, the vortex structure is highly deformed, irregular, and partially dissipated.

These results qualitatively support the hypothesis that the healthy left ventricle develops a flow apparently favorable to efficiently eject the blood during contraction. This point is here analyzed by quantifying the temporal evolution of the total viscous dissipation

$$\mathcal{P}(t) = \frac{T^3}{\rho D_0^5} \int_{V(t)} \boldsymbol{T}\tilde{\mathrm{a}}\tilde{\mathrm{N}}\boldsymbol{D}\, dV. \qquad (2)$$

$\mathcal{P}(t)$ is the dimensionless power dissipated by the local stresses to deform the fluid elements over the entire cavity, $\boldsymbol{T}$ is the stress tensor, and $\boldsymbol{D}$ is the rate of strain tensor [14], while the prefix in front of the integral makes the result dimensionless and ρ is the fluid density. The time evolution of $\mathcal{P}(t)$ is shown in Fig. 5(a) for six solved cases that differ in the value of ε only. During the initial stage of the diastolic filling, no significant differences have been detected, with the exception of the case $\varepsilon = 0.25D$, where a greater instantaneous dissipation takes place because of the close interaction between the entering eccentric jet and the wall boundary layer. In the interval between the conclusion of the ventricular filling and the beginning of the ejection, $0.35 < t/T < 0.5$, differences are noticeable and directly reflect how the filling flow well couples with the following flow ejection pattern. The cases with $\varepsilon = 0.125D$ and $\varepsilon =$

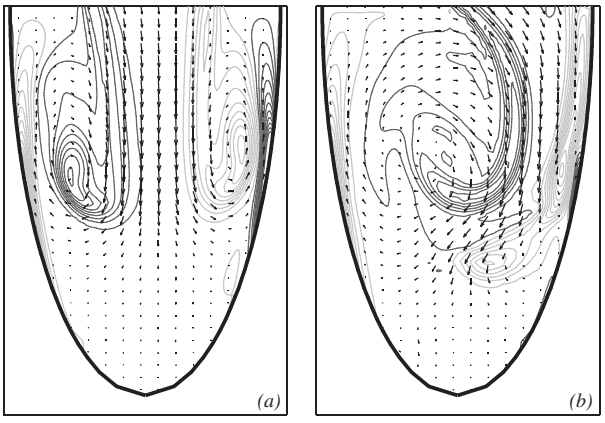

FIG. 4. Velocity vector and distribution of the vorticity on the symmetry plane at $t = 0.4T$; vorticity levels from -315 to 315, step 30, negative (clockwise) levels are black, and positive levels are light gray; (a) $\varepsilon = 0.02D$, (b) $\varepsilon = 0.25D$.

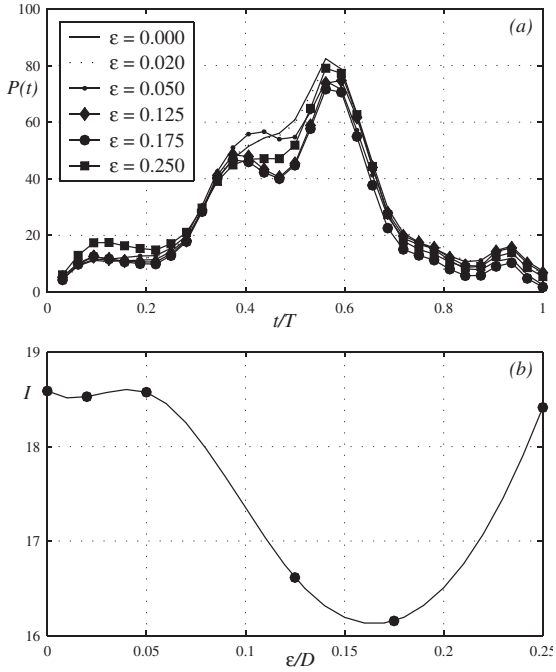

FIG. 5. Time variation of the instantaneous viscous dissipation, $P(t)$, for different values of the displacement ε of the mitral orifice (a), and (b) total viscous dissipation I as a function of ε, (●) computed results, (−) interpolating curve.

$0.175D$ show low values of energy dissipation in this period.

The total energy dissipation during filling until the peak of ejection (at time $t_{sys} = 0.57T$),

$$\mathcal{I} = \int_0^{t_{sys}} \mathcal{P}(t)dt, \quad (3)$$

is shown in Fig. 5(b) as a function of ε. This curve has a minimum for ε almost corresponding to physiological values. When the mitral orifice is more centered, dissipation increases (of a percentage between 10% and 15%) because the flow pattern at the end of filling is not favorable to the ejection, and fluid elements are subjected to a more significant strain to redirect the flow toward the exit. On the opposite, when the mitral orifice is highly eccentric the entering flow collides with the ventricular wall, enhancing the dissipative interaction with the boundary layer. The numerical results confirm the general hypothesis, emphasized by the argument and the nice visualizations given by [12], that the natural swirling flow found inside healthy ventricles is an efficient physiological adaptation to facilitate the ejection of blood. The increase of mechanical work reported here, which corresponds to a loss of efficiency of the heart pump, is not negligible with respect to the about 50% increase of pressure losses found with aging (from 18–30 to 60–70 yr old) or about 40% reduction during exercise (average values, e.g., [16]).

The results presented here show that the intraventricular flow pattern in healthy subjects has a natural structure that is optimal in terms of minimization of the energy dissipation. When this natural arrangement is broken, the increased dissipation has a potential consequence in increasing the work required by the cardiac muscle to eject in the circulation an equally energetic flow, thus augmenting the consumption of the oxygen to activate the additional chemical work. Alternately, the same muscular stress made on a chamber with a suboptimal asymmetry ejects a less energetic blood (lower pressure) in the aorta. This could activate physiological control mechanisms (possibly pressure rise, hypertension) to allow a sufficient perfusion of the primary circulation network.

A reduction of asymmetry is a typical consequence of several cardiac diseases, such as the increase of blood pressure (hypertension) that leads to ventricular dilatation (dilated cardiomyopathy), one of the most common diseases with aging. Flow symmetrization is commonly observed in these cases, although no clinical trial about this effect has been made, and it is not yet used for diagnostic purposes. However, any physiological issue goes beyond the scope of this work: the potential increase of mechanical work represents one, possibly marginal or central, among the many physiological phenomena concurring to the heart function (e.g., the primary requirement is the maintenance of a sufficient amount of blood, i.e., oxygen, ejected from the ventricle to the circulation). This work is aimed to show evidence of a natural symmetry break in the normal heart whose quantification may depend on the individual geometry and its time profile.

The geometry of the mitral valve (here assumed as circular and fixed open), in particular, the asymmetry of its leaflets, is another important aspect that influences flow pattern. The results suggest that mitral valve diagnosis and repair should consider the impact of the therapy on flow symmetry. The design of mechanical prosthesis, or the selection of biological ones, will influence the asymmetry of the flow and therefore the function of the heart.

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*Electronic address: giannip@dic.univ.trieste.it
†Electronic address: federico@ingfi1.ing.unifi.it

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