Model and influence of mitral valve opening during the left ventricular filling

Bernardo Baccani\textsuperscript{a}, Federico Domenichini\textsuperscript{a,}\textsuperscript{*}, Gianni Pedrizzetti\textsuperscript{b}

\textsuperscript{a}Dipartimento di Ingegneria Civile, Universit\textsuperscript{a} di Firenze, Via S. Marta, 3, Firenze 50139, Italy
\textsuperscript{b}Dipartimento di Ingegneria Civile, Universit\textsuperscript{a} di Trieste, Italy

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Abstract

The flow inside a model left ventricle during filling (diastole) is simulated by the numerical solution of the equations of motion under the axisymmetric approximation. The left ventricle is taken with a truncated ellipsoid geometry, and a simple conceptual model is introduced to simulate the presence of the moving mitral valve. A relevant role during the left ventricle diastolic flow, as already discussed by other authors, is played by the travelling vortex wake that is formed from the transmitral jet during the early filling acceleration phase. The presence of a moving valve is found to produce a non-simultaneous spatial development of the entering bulk flow and a slightly more complex vortex wake structure; the results are discussed in comparison with fixed valve ones. They are analysed also in terms of M-mode representation suggesting a physical interpretation of the pattern detected in the clinical measurements that extends the one given previously on the basis of fixed valve models.

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1. Introduction

The flow inside of the left ventricle represents one of the most interesting problem in biological fluid dynamics, depending on its relevance on the global functionality of the heart pump. Nowadays it is known that the diastolic phase has a relevant role during the complete heartbeat period, and it is widely accepted that minor dysfunctions during the filling phase are often early indicators of heart failure (Mandinov et al., 2000). Therefore, an increasing effort has been put on the analysis of the left ventricle diastolic flow, in order to understand the major phenomena involved and to develop physically based schemes for the interpretation of clinical echographic measurements.

The principal fluid phenomena involved in the left ventricular diastolic flow are related to the presence of vortex structures that develop with the strong jet that enters through the mitral valve. Such phenomena have been initially recognised by visualisation of the ventricular flow (Bellhouse, 1972; Reul et al., 1981; Wieting and Stripling, 1984; van Dijk, 1984), and afterwards confirmed by analyses based on colour Doppler mapping and Magnetic Resonance Imaging (Kim et al., 1994, 1995; Firstenberg et al., 2000; Kilner et al., 2000; Tonti et al., 2001, to cite few of them). Results that have been further confirmed by in vitro experiments (Steen and Steen, 1994). A further step in the understanding of the flow features has been obtained by the numerical analysis of the problem; different numerical techniques have been used to study the details of the ventricular flow (Vierendeels et al., 2000; Lemmon and Yogathan, 2000; Baccani et al., 2002a).

On the basis of the recent literature results, the most relevant features of the intraventricular flow can be synthesised as the formation of vortex sheets attached to the valve during the strong accelerating stage of the E-wave, the detachment of the fully formed vorticity wake at the deceleration of the entering flow, and the translation of the vortex structure toward the ventricle apex accompanied by the interaction with the induced boundary layer at the ventricle walls.
This complex dynamics has been described in detail under the assumption of axial symmetry of the flow and a fixed valvular orifice (Vierendeels et al., 2000; Baccani et al., 2002b). Within the limits of the idealised modelling, the numerical results give a physically based description of the ventricular flow. The comparison between numerical results and measured data has shown the capability of the mathematical modelling to clarify several phenomena observed during the clinical practice. In particular, the representation of the results in terms of space–time maps of the axial velocity, computed on the ventricle axis, has clarified the physical meaning of the well known M-mode velocity pattern detected by the cardiologists (Garcia et al., 1998), where a major feature is strictly correlated with the vortex structures evolution.

The most remarkable difference between clinical data and mathematical modelling, also present in the experimental results (Steen and Steen, 1994) made with rigid valvular orifice, is observable during the initial stage of the diastole when the mitral valve motion is expected to play an important role in the birth and development of the vortex sheets.

A simple attempt to analyse the influence of the mitral valve is reported in the present work. Following the assumptions reported in (Baccani et al., 2002b) the diastolic flow is analysed in an idealised left ventricle. The mathematical model and the numerical scheme do not allow the explicit introduction of moving valvular leaflets; thus, the presence of the mitral valve is simulated imposing different fluid dynamical boundary conditions at the mitral plane. The effect of the valvular motion is therefore replaced by the introduction into the ventricular flow of a vorticity distribution, which simulates the boundary layer developing at the valvular leaflets. Notwithstanding the simplicity of the model, the results show that the properties of the early entering flow are modified significantly by such inlet condition, which leads to an initial pattern closer to clinical ones. This is evidenced by the M-mode representation of the axial velocity.

2. Methods

The mathematical formulation of the problem follows closely that reported in (Baccani et al., 2002b), and it is here only briefly recalled. The motion of a Newtonian fluid inside a model left ventricle is analysed in the axisymmetric approximation. The ventricle is assumed to be half of a prolate spheroid. The fluid dynamics is forced by the imposed motion of the ventricle wall specified by the time laws of the major semiaxis and principal diameter, \( H(t) \) and \( D(t) \); these are derived by available clinical measurements of a healthy subject, a young athlete. From such data the entering discharge \( Q(t) \) is obtained, Fig. 1. Thorough the article we refer to dimensionless quantities, unless otherwise stated, in the terms described here. The dimensionless Navier–Stokes equations are characterised by the group \( \text{Re}_T = U^2 T/\nu \), where \( \nu \) is the kinematic viscosity of the fluid, the time scale \( T \) is the heartbeat period, the velocity scale \( U \) is the peak inlet velocity during the systolic phase. The equations of motion are written in the moving, boundary-fitted prolate spheroid system of coordinates (Baccani et al., 2002a). The no-slip condition is imposed at the ventricle walls, and symmetry conditions are given on the axis, \( r = 0 \), of the ventricle. Particular attention is posed to the definition of the inlet flow conditions at the mitral plane.

The mitral aperture is modelled as a thin orifice with diameter \( D_v(t) \), with the ratio \( D_v(t)/D(t) \) kept constant in time, and different inlet conditions have been used. The fixed-open orifice case is analysed imposing an irrotational entering profile (Baccani et al., 2002a, b). In order to reproduce the presence of the opening valve,
which in the present model cannot be explicitly simulated, different inlet conditions are adopted as follows. An additional parameter $b(t)$ is introduced, it represents the angle between a virtual rigid opening valve and the mitral plane. The value $b = 0$ represents a completely closed valve, i.e. no transmitral flow $Q = 0$; we assume that a complete opening, with a value $b = \pi/2$, corresponds to a value $Q = Q_{\text{lim}}$. During the intermediate instants $b(t)$ is linearly related to $Q(t)$, as shown in Fig. 1. The initial period of the diastole necessary to the complete valve opening has been derived from the clinical data, and a corresponding value $Q_{\text{lim}}$ of the entering discharge has been estimated. Once the valvular opening $b(t)$ is instantaneously known, the projection on the mitral plane of the valve radius, of size $D_v \cos b/2$, gives the portion of the mitral orifice which is partially closed; on this region the profile of the entering velocity is assumed to be linear, simulating the adherence of the fluid to a rigid valve rotating with angular velocity $d\beta/dt$. An irrotational profile is imposed on the remaining open part of the mitral orifice. The same approach allows to simulate the presence of a curvature on the leaflets. For this the previous model has been slightly modified adding a perturbing parabolic contribution to the linear profile such that the velocity is smaller in the central portion and higher at the edge of the leaflet with respect to the linear profile. The parabolic perturbation is characterised by its maximum value $\epsilon$, taken as a percentage of the maximum one of the linear profile. In all cases, the entire continuous inflow profile, in correspondence of the instantaneous $Q(t)$ and $b(t)$, is computed at once as a solution of a linear system. Details on the numerics and the related technical points can be found in Baccani et al. (2002b).

3. Results

The problem has been analysed assuming the forcing discharge in Fig. 1 and varying the opening period of the virtual valve in a narrow range around 0.025, that has been estimated from the available data as a mean significant value; as a consequence, the value $Q_{\text{lim}}$ results in the range $2.3-2.7 \times 10^{-4}$. In what follows the results obtained with an instantaneous opening, $Q_{\text{lim}} = 0$, are compared with those derived with the value $Q_{\text{lim}} = 2.5 \times 10^{-4}$; all the other analysed cases show a very similar behaviour, and they are not reported for shortness.

The flow evolution in the case $Q_{\text{lim}} = 0$ is shown in Fig. 2, where the instantaneous velocity and vorticity fields are plotted, and is here briefly summarised. During the initial stage of the E-wave, the flow presents the almost instantaneous birth of a wake vortex attached to the mitral edge. Such a wake grows in size and interacts with the boundary layer at the ventricle wall. The primary wake immediately rolls-up into a well-defined vortex ring that tends to lift the opposite-sign vorticity from the wall boundary layer, Fig. 2a at $t = 10/256$. Afterward the boundary layer separates from the wall, as shown in Fig. 2b at $t = 20/256$, and the vortex wake detaches from the mitral edge to eventually form a free

![Image of a figure showing instantaneous velocity and vorticity fields](image_url)

Fig. 2. Instantaneous velocity (left) and vorticity (right) fields, fixed-open valve: (a) $t = 10/256$, (b) $t = 20/256$, (c) $t = 50/256$. Vorticity levels: from 10 to 80, step 10 (black) and symmetric negative values (grey).
vortex structure at the end of the accelerating phase, \( t \sim 0.1 \). The vortex ring translates for self-induced motion toward the ventricle apex dragging the vorticity at the wall. During the deceleration of the E-wave this phenomenology repeats in a weaker form, Fig. 2c at \( t = 50/256 \). The even weaker vortex wake that forms during the A-wave has no time to develop and detach from the mitral edge. The details of these phenomena may vary, depending of the specific ventricle geometry and on the tissue kinematics (or inlet flow profile). Phenomena modification associated to different conditions can be found in Vierendeels et al. (2000) and Baccani et al. (2002a, b).

A synthetic, although partial, description of the flow dynamics is given by the space–time evolution of the velocity along the axis of symmetry. This representation, Fig. 3, is analogous to the standard Doppler M-mode performed during clinical routine (Garcia et al., 1998). An important feature is noticed during the initial dynamics \((t < 0.1)\); it presents an essentially vertical structure, that is the maxima of the velocity at different depths along the axis are contemporaneous, as a consequence of incompressibility. The following phase \((t > 0.15)\) shows the trace of the primary vortex motion which, detached from the mitral plane, travels inside of the ventricle in an otherwise irrotational field (Steen and Steen, 1994; Vierendeels et al., 2000; Baccani et al., 2002a, b), with a propagation velocity (the slope of the dashed line in Fig. 3) estimated as \( V_p \approx 0.18 \).

The flow dynamics is modified when \( Q_{\text{lim}} \) is greater than zero, the velocity and vorticity fields are shown in Fig. 4 in correspondence of a linear inlet profile (straight leaflets). The initial dynamics is characterised by the presence of a vortex sheet that develops at the virtual valve location and reproduces, in the present model, the boundary layer developing over the moving leaflets. During the opening period, the sheet rotates following the assumed leaflet motion, up to the final, approximately vertical, shape corresponding to a valve completely open. At that time, the inflow becomes irrotational, with the exception of the thin vortex wake developing at the edge on the mitral plane, Fig. 4a at \( t = 10/256 \). The valvular vortex sheet stretches inside of
the ventricular cavity, and a vortex wake develops at the entering section. During the following period, the vortex sheet rolls up into a free vortex and the attached vortex wake develops in a manner that is analogous to the previous case, compare Fig. 4b, at $t = 20/256$, with Fig. 2b. Eventually a more complex vorticity field is found because of the combined inflow phenomena that produce a double vortex wake. It is shown in Fig. 4c where also the inflow influence of the partly closed valve during diastasis (see Fig. 1b) can be observed.

The space–time map of the axis velocity is reported in Fig. 5 where some remarkable differences from that of Fig. 3 are found. In particular, the M-mode pattern presents two main traces with a finite slope. The first trace ($t < 0.15$) is essentially absent in Fig. 3; it represents the propagation of the local velocity maxima from the mitral plane toward the apex. This evolution is intimately related to the increase of the effective orifice area due to the valvular opening whose dynamics is reproduced in an approximate way with the present model. The second trace ($t > 0.15$) corresponds to the motion of the primary vortex detached from the mitral plane, and it is only marginally influenced by the valve dynamics. A linear approximation of such traces is plotted in Fig. 5, in order to estimate the propagation velocities, found to be approximately 0.55 and 0.2, respectively. These values can be assumed as a direct measure of the propagation velocity expressed in m/s, given the choice of the scaling quantities.

The flow dynamics with the perturbed inlet velocity profile (curved leaflets) has been analysed varying the parameter $\varepsilon$ in the range 0–0.15. The space–time map of the axis velocity obtained with $\varepsilon = 0.15$ is reported in Fig. 6. Increasing $\varepsilon$ leads to the growth in intensity of the initial vortex sheet, which therefore shows a prolonged motion toward the ventricle apex, before being captured by the first detached free vortex. Such a difference does not seem to affect significantly the flow evolution as a whole, remaining substantially unchanged the values of $V_p$.

4. Discussion

The diastolic fluid dynamics of a model left ventricle has been analysed numerically solving the equations of motion in the axisymmetric approximation. The ventricle is modelled as half of a prolate spheroid, the mitral valve is assumed to open from an orifice of infinitesimal thickness. The system is forced by the wall motion, whose temporal evolution is derived from clinical data. Particular attention has been posed to the simulation of the valvular opening dynamics by introducing different inlet boundary conditions at the mitral plane. The results obtained with a fixed-open orifice are compared with those of a moving valve.

The flow evolution in the case of fixed valve presents a simultaneous peak flow along the entire ventricle because of incompressibility, and the development of a strong vortex wake from the mitral edge that eventually translates toward the apex and interacts with the ventricular wall, in agreement with previous findings (Steen and Steen, 1994; Vierendeels et al., 2000; Baccani et al., 2002a, b) obtained with a fixed-open valvular orifice.

The modified inlet condition has been constructed, on the basis of clinical observations, as a simple model to reproduce some major effects given by the presence of an orifice with opening leaflets. In fact, the valve takes a finite interval of time to open completely, and during this period the entering flow develops a vorticity layer on the valvular leaflets. The proposed model automatically reproduces the change in time of the effective
valvular area, and the results show some of the main fluid dynamics features expected here. The model allows to simulate the influence of the mitral valve dynamics without solving the complex details of the transmitial motion. The analyses have shown that the ventricular flow is influenced by the valvular motion, but the details of the valve dynamics do not appear to have a fundamental relevance in the global field, depending on the extreme rapidity of the valvular opening.

The M-mode representation of the velocity presents two main propagating traces related to two different physical phenomena, respectively. The former, during flow acceleration, represents the valvular dynamics: it presents a steeper slope that becomes a vertical trace (instantaneous propagation), if observable, in results obtained with fixed valve models (Steen and Steen, 1994; Vierendeels et al., 2000; Baccani et al., 2002a, b). The latter is the propagation of the vortex wake that has been previously observed by several authors. The first trace develops during the initial filling in the region close to the mitral valve, thus it is directly influenced by the properties of the transmitial flow, in particular it is influenced by the evolution of the effective valvular area, here reproduced by modifying the inlet boundary condition. It is less affected by the almost irrotational flow which develops in the large part of the ventricle, where the influence of the wall characteristics is appreciable in the thin viscous boundary-layer only.

The propagation velocity $V_p$, estimated from Color-Doppler M-mode, is a recent clinical indicator of the cardiac function, however its quantification method has always been controversial and a relevant variation of $V_p$ is found in practice (Garcia et al., 1999, and references therein). The present results suggest that the dispersion of the $V_p$ measures could be also imputable to the concurrence of two different physical phenomena and therefore to the mixing of values taken in correspondence of the two of them, giving an interpretative scheme for clinically observed measurements. A confirmation of them may suggest the development of appropriate methods or protocol for the physically based evaluation of $V_p$.

The axisymmetric assumption represents probably the most relevant simplification adopted in this work, it does not allow to describe the three-dimensional effects of the actual dynamics; among these, the geometry of the mitral valve that is far from being axisymmetric and the final stage of the diastolic dynamics, when instability effects take place and the vortex wake is more rapidly dissipated. In view of the axisymmetric results, the flow field is expected to be influenced by the valvular geometry and motion, depending on the significant difference between the anterior and posterior leaflets, which represent the source of the vorticity entering into the ventricle; the real ventricle geometry and wall properties seem to have a minor relevance on the ventricular flow during a large part of the diastolic phase, which appears dominated by the non-linear dynamics of the travelling vortices. Work is currently in progress to extend the modelling to three-dimensional flows.

References
