Intraventricular vortex flow changes in the infarcted left ventricle: numerical results in an idealised 3D shape

Federico Domenichini* and Gianni Pedrizzettib

aDipartimento di Ingegneria Civile e Ambientale, Università di Firenze, V. S. Marta 3, Firenze, Italy;
bDipartimento di Ingegneria Civile e Ambientale, Università di Trieste, P.le Europa 1, Trieste, Italy

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The cardiac diagnostic process is primarily based on the evaluation of myocardial mechanics whereas little is known about blood dynamics that is rarely considered to this purpose. The intraventricular blood flow is analysed here for akinetic and dyskinetic myocardial motion corresponding to the presence of an ischaemic pathology. This study is performed through a 3D numerical model of the left ventricular flow. Results show that the presence of an anterior–inferior wall infarction leads to the shortening and weakening of the diastolic mitral jet. A region of stagnating flow is found near the apex and close to the ischaemic wall. These results are in agreement with previous clinical findings based on echographic imaging. The described phenomena are also noticed for moderate degrees of the ischaemic pathology and suggest a potential value of the study of the intraventricular flow to develop early diagnostic indicators.

**Keywords:** left ventricle; infarction; computational fluid dynamics

1. **Introduction**

The left ventricle (LV) is the most energetic element of the human heart, a deficiency in its contractility reflects immediately in the allowance of oxygenated blood in the whole body and possibly gives rise to symptomatic pathology up to cardiac arrest. A systolic dysfunction, however, is often preceded by a weaker, possibly asymptomatic, lack of efficiency that is more difficult to detect, and whose identification represents a challenge to physicians (Wilson et al. 1998). Diagnosis at the early stage of a pathology plays a central role in cardiology. An early detected heart malfunction can be controlled by a light pharmacological therapy. Pathologies found at an advanced stage may produce irreversible modifications or could require invasive approaches. It is therefore customary to pursue a deeper understanding of LV mechanics in pathophysiological conditions. And to develop the ability of describing changes that can be observable and eventually employed in a physically grounded development of diagnostic techniques.

This study is focused on the regional LV ischaemia and its relation with intraventricular fluid dynamics. Ischaemia is a primary systolic dysfunction caused by the partial or total occlusion of a coronary vessel, leading to the decrease in blood perfusion in the myocardial muscular territory. In terms of mechanical function, an ischaemic segment presents a reduced contractile activity and a progressive akinetic behaviour, when its motion is only due to the passive transport of nearby segments. The result of an important or long-lasting ischaemic pathology is dyskinesia. The pathologic segment moves in the direction opposite to the nearby tissue, because the muscle has locally lost its functionality and the segment expands during contraction because of pressure increase in the chamber. It is worth to remark that, given the periodicity of the heart cycle, an abnormal motion during systole is likewise reflected during the diastole. A dyskinetic segment that moves outward during the contractile phase necessarily displaces inward in the filling one. Therefore, a systolic dysfunction has an indirect influence on the diastolic phase (Sengupta et al. 2006).

The diagnosis of cardiac contractile function is largely based on the evaluation of the tissue mechanics by means of medical imaging techniques such as magnetic resonance imaging (MRI) and echocardiography. Global indexes (like Ejection Fraction, EF), which are routinely estimated, give measures of the pumping efficiency of the LV. The segmental LV contractile strength has been initially evaluated by measuring thickening and inward motion (M-mode echocardiography) and, more recently, by measuring the myocardial strain by tissue Doppler echocardiography, tagged-MRI or, lately, 2D-strain techniques. These techniques allow, within certain limitations, to quantify the regional contractility with good accuracy and reproducibility. On the other side their applicability in the clinical practice is still debated.

The purpose of the LV is to efficiently convey propulsion to the blood. Therefore, the LV fluid dynamics is directly affected by the LV function, and its analysis...
may give additional information to the diagnostic process. The intraventricular flow has a peculiar arrangement associated with vortices (Kilner et al. 2000; Cooke et al. 2004) that are structures sensible to the dynamics of the surrounding boundary (endocardium). Normal flow develops with an asymmetric vortex structure that permits an efficient filling (Cheng et al. 2004; Domenichini et al. 2005) and a natural redirection towards the outflow track (Bolzon et al. 2003). Numerical studies have shown, in ideal or approximate models, the potential development of changes in the presence of specific pathologies (Lemmon and Yoganathan 2000; Baccani et al. 2002a), and the relevance of the asymmetry of the mitral orifice for the efficiency of the pumping work (Pedrizzetti and Domenichini 2005). These studies, and other experimental and numerical studies cited therein, have clarified several aspects of the cardiac fluid dynamics. Nevertheless, the understanding of the modifications of intraventricular flow due to pathologies is still at an early stage and further developments are needed before these findings can be translated into the clinical phase.

Recent imaging processing techniques are starting to develop diagnostic tools that permit, to some extent, the visualisation of intraventricular vortex flow (Hong et al. 2002b; Domenichini et al. 2005). The blood is assumed to be a Newtonian incompressible fluid, governed by the Navier-Stokes and continuity equations

\[
\frac{\partial \mathbf{v}}{\partial t} + \mathbf{v} \cdot \nabla \mathbf{v} = -\frac{1}{\rho} \nabla p + \nu \nabla^2 \mathbf{v}, \quad \nabla \cdot \mathbf{v} = 0, \quad (1)
\]

where \( v \) and \( p \) are the velocity and the pressure, respectively, \( \rho = 1060 \text{ kg/m}^3 \) and \( \nu = 3.3 \times 10^{-6} \text{ m}^2/\text{s} \) are the density and the kinematic viscosity respectively. The system (1) is completed with the boundary conditions. At the cavity wall, the no-slip condition is enforced. At the equatorial plane, \( z = 0 \), which corresponds to the region below the mitral and aortic orifices, the entering/exiting jets are reproduced imposing their velocity profiles (Pedrizzetti and Domenichini 2005). The mitral entry-jet is assumed here with an elliptic blunt shape

\[
v_c(x,y,z=0) = V_0 \exp\left[-\frac{(x-x_0)^2}{\sigma_x^2} - \frac{y^2}{\sigma_y^2}\right], \quad (2)
\]

where \( x_0 > 0 \) is the centre of the mitral jet, \( \sigma_x \) and \( \sigma_y \) are a measure of its dimensions. The reference velocity \( V_0 \) is evaluated to allow the exact quantity of fluid required by the LV volume expansion to flow into the cavity. From (2), it follows that \( y = 0 \) is the plane passing through the mitral and the aortic orifices, the posterior LV wall and the anterior intra-ventricular septum (IVS); it corresponds approximately to the apical 3 chamber (A3C) projection in echocardiographic imaging, see Figure 1. The reference healthy dynamics of the LV walls has been taken from the analysis of echocardiographic images of a young subject, with \( EF = 55\% \), whose temporal variation of the cavity volume is reported in Figure 2.

A myocardial infarction is artificially reproduced modifying the wall dynamics to mimic different degrees of occlusion of the left anterior descending coronary (LAD). This is achieved starting from the healthy case and reducing the longitudinal and circumferential strain of the anterior and inferior IVS at median-apical level. This region, see Figure 1, corresponds to a circumferential sector in the range \( \pi/2 < \theta < 7\pi/6 \) in the lower half of the LV wall, \( \theta \) is the polar azimuthal coordinate. At each instant of time, the healthy strains at every point of the wall are multiplied by a reduction coefficient

\[
c(\eta, \theta) = 1 - A \exp\left[-\left(\frac{\theta - \theta_c}{2\theta_c}\right)^4 - \left(\frac{\eta - \eta_c}{2\eta_c}\right)^4\right], \quad (3)
\]
AV = Equation 3, are also plotted. The darker area marks the ischaemic volume, \( V \) influenced by the infarction is defined by (3) with centre at when strain is assumed zero as reference. The region is evaluated starting from the tele-diastole (ECG R-wave) once the strain values are known, the pathologic geometry of the cavity \( V \), \( \text{cm}^3 \), Figure 2. Volume of the cavity \( V(t) \) for the healthy ventricle (EF = 55%), continuous line, and for an ischaemic ventricle with regional akinesia (EF = 40%), dashed line.

where \( \eta \) is the spheroidal coordinate along the wall, ranging from \( \eta = 0 \) at the apex to \( \eta = \pi/2 \) at the base (Baccani et al. 2002b); the relation between \( \eta \) and the vertical coordinate \( z \) is

\[
z = H \sqrt{\frac{4H^2 + D^2}{4H^2 - D^2}} \cos(\eta).
\]

Once the strain values are known, the pathologic geometry is evaluated starting from the tele-diastole (ECG R-wave) when strain is assumed zero as reference. The region influenced by the infarction is defined by (3) with centre at \( \theta_c = 5\pi/6 \) and \( \eta_c = \pi/8 \), and extension \( \theta_e = \pi/3 \) and \( \eta_e = \pi/10 \); an annular extension has been assumed for the longitudinal strain only to avoid LV tilting. The factor \( A \) represents the entity of the infarction: small values, say \( A < 1 \), simulate a reduced wall motion. Values little larger than 1 correspond to a regional akinesia, higher ones to dyskinetic motion. Variations of \( A \) in the range 1–2 lead to values of the EF from 45 to 32%.

The system (1) is solved in a computational box, with the LV boundaries immersed therein and managed through a version of the immersed boundary (IB) method (Mittal and Iaccarino 2005). Periodicity is assumed along \( x \) and \( y \) axes to allow a spectral representation of the variables and fast solution methods of the Poisson problem for the mass conservation. Centred second-order finite differences on a 3D staggered grid are employed for the temporal advancement of the Navier–Stokes equation, performed with a third-order Runge–Kutta scheme. The velocity components are then transformed in their Fourier’s representation, to compute the divergence and solve Poisson’s problem for the instantaneous irrotational correction of the pressure field (Verzicco and Orlandi 1996). With this method, the \( N_x \times N_y \times N_z \) Poisson’s system is reduced to the solution of \( N_x \times N_y/2 \) tridiagonal linear systems of dimension \( N_z \) that can be achieved with a fast direct method. The corrections to the velocity and pressure fields are computed in Fourier’s space and transformed again in their physical counterparts.

The LV wall is represented by a 2D mesh, in the \( \eta-\theta \) space, whose both position and velocity are known at each time step. Any interpolation is used to impose the ‘inner’ boundary condition at the wall, but the wall velocities are imposed to the closest point of the computational 3D grid during the advancement of the Navier–Stokes Equation (Domenichini 2008). The accuracy of the IB method and the choice of some numerical parameters have been verified by comparing the results with those obtained with a numerical code written in the body-fitted system of coordinates (Domenichini et al. 2005), showing almost undistinguishable solutions. The results here reported have been obtained with a computational grid \( N_x = N_y = N_z = 128 \). Several preliminary runs for the healthy case have been performed doubling the computational grid in all the directions to guarantee the convergence of the solution. The chosen grid has proven to be a good compromise between the convergence of the solution and the computational effort. The dimensions of the numerical box have been eventually set to \( L_x = L_y = 6.6 \text{ cm} \), \( L_z = 7 \text{ cm} \), the independence of the solution on the transversal dimensions has been verified doubling \( L_x \) and \( L_y \), showing a negligible influence of the assumption of periodicity. The time step of integration has been fixed to \( \Delta t = T/1024 \), \( T \) being the heartbeat period, to satisfy the stability conditions and accuracy of the unsteady IB approach (Domenichini 2008).
3. Results

The flow is analysed in terms of vorticity and velocity on the transversal plane, y = 0, that crosses the mitral and outflow tracks, and in terms of the 3D vortex structure identified by the so-called $\lambda_2$-method (Jeong and Hussain 1995). The healthy case (EF = 55%) is reported in Figure 3. Hereinafter, the velocity vectors are sampled from the finer computational grid to enhance the readability. During the early filling phase, the entering jet develops a compact ring-shaped vorticity structure, whose 2D cross section is represented by two counter-rotating vortices. The vortex on the anterior LV side (left in Figure 3(a) and (c)) occupies the central part of the cavity, whereas the other (right) one creeps along the posterior wall and interacts with the viscous boundary layer. During the following evolution, the primary vortex structure deeply penetrates the ventricle, whereas a second one associated to the A-wave phase develops below the mitral plane. The deformation of the primary structure is noticeable, due to its self-induced dynamics, the left vortex occupies the large part of the transversal plane, pushing the right one on the wall, where it is partly dissipated within the boundary layer (Domenichini et al. 2005). During systole, the interaction between these structures gives the complex 3D field shown in Figure 3(d), which dissipates and is finally ejected through the aortic orifice.

The influence of a moderate reduction in the wall mobility ($A = 1$ in (3)), with a diminution of the EF from 55 to 45%, is depicted in Figure 4. The entering jet penetrates the ventricle less deeply and with lower levels of vorticity. As a consequence, the self-induced dynamics and dissipation are weaker resulting in a more regular vortex structure at the beginning of the systole, Figure 4(c) and (d). A more severe level of infarction, with a dyskinetic behaviour ($A = 2$ in (3)), is shown in Figure 5, with an increased shortening of the mitral jet and lower levels of vorticity. The large part of the ventricle is not influenced by the vortex structures and by flow circulation. During systole, the flow redirected to the aortic outflow comes from the central and upper parts of the ventricle. Velocity is always almost null close to the apex, where the flow is nearly stagnating during the entire heart cycle. The steady-streaming flow, that is the flow averaged over the heartbeat period, is a synthetic description of the dominant circulation persisting during the cycle. It is shown from the healthy case, EF = 55% (Figure 6(a)), to EF = 45, 40, 32% ($A = 1, 1.4, 2$ in Equation (3)) in Figure 6(b)–(d). The circulatory pattern, that characterises the redirection of blood from the inlet to the outflow track, becomes progressively shorter and weaker, the stagnating area at the apex becomes more extended. The reduction in the jet length is related to the diminution of the EF as given by the simple calculation $L_{jet} = \int_{\text{dia}} Q dt/A_{MV} = (V_{ed} - V_{es})/A_{MV} = EFV_{ed}/A_{MV}$ from which it follows that the jet length, in first approximation, is proportional to the EF and

$$\frac{L_{jet}}{H_{ed}} = \frac{V_{ed}}{H_{ed}A_{MV}} = \frac{\pi D_{ed}^2}{6A_{MV}}. \quad (4)$$

This rough estimate assumes a 1D unsteady jet that maintains a constant direction during the filling. In the present case, the jet also expands transversally, its shortening is further enhanced by the lateral motion at the base, because the small mobility at the apex reduces the capability of accommodating fluid therein.

4. Discussion

This work focused on describing how the reduced mobility of the myocardium, as caused by a LAD coronary stenosis, is reflected in the left ventricular fluid dynamics. To this aim, the contraction of a healthy prolate spheroid cavity has been progressively reduced to reproduce different degrees of ischaemia and infarction. The problem has been analysed via numerical solution of the fluid dynamics equations.

The work takes the simplifying assumptions of a smooth endocardial wall, and papillary muscles are not included. These would add disturbances to the wake structure, enhancing its dissipation, but are not expected to modify the global phenomenological outcome. Similarly, the healthy ventricular shape has been taken from an ideal geometry, and the pathologic motion of the ischaemic myocardium has been defined artificially rather than on the basis of clinical recordings. Therefore, the results are aimed to evidence changes in the fluid dynamics pattern, as would be expected in specific pathological conditions, although the effective correspondence in clinical practice is suggested but not demonstrated here.

Moreover, the held-open mitral valve used here does not account for the leaflets dynamics, which can indeed modify the entry jet development. A previously tested inflow profile has been employed; such a design has shown to reproduce the natural pattern of the intraventricular flow (Domenichini et al. 2005; Pedrizzetti and Domenichini 2005), in agreement with the ones observed in clinical studies (Beppu et al. 1988; Hong et al. 2007; Sengupta et al. 2007; Hong et al. 2008). In addition, a recent study on leaflet opening (Pedrizzetti and Domenichini 2007) partially supports this approximation showing that the vortex from the longer leaflet (here the anterior) does not develop significantly until the leaflet reaches its final open position. In general, particular care must be used in modelling the transmitral flow for which limited information are normally available. At the present state of knowledge, it is not immediate to ensure a realistic modelling of the transmitral flow or, in alternative, of the leaflets geometry and their material properties. In this
study, the simple entry flow model has been designed to give a realistic intraventricular flow and to reduce the impact of this issue. At the same time the possible modifications of the valvular dynamics due to the presence of a pathology are not addressed in this study that is focused on the effect of the wall dyskinesia only.

Within the limitations of the present model, it has been shown that the anterior–inferior wall infarction is expected to have a relevant impact on the flow dynamics. This is represented by a reduction in the mitral jet strength during the diastole, visualised by its shortening and by the lower intensity of the vorticity structure, which characterises the ventricular flow (Kilner et al. 2000; Domenichini et al. 2005). These effects are connected to the diminution of the ejection fraction, which is closely related to the dimensionless length of the entering jet (4). These results give a physically based background to the clinical observations discussed in (Beppu et al. 1988). In addition, they show that the fluid adjacent to the tissue with low mobility presents a nearly stagnating region, so that the vortex tends to keep away from the ischaemic area.

It is eventually remarkable that the phenomena described above are already noticeable for moderately low level of disease. This may suggest a potential value of intraventricular flow for the development of early diagnostic indicators, either in normal and stress tests. A value that should, however, be carefully verified through a dedicated clinical experimentation.

A remark must be added about a further limitation of the model. The tissue dynamics, even in the healthy case,
has been assumed as essentially composed of radial and longitudinal motions neglecting the ventricular torsion. From the fluid perspective, the torsion is seen as a differential rotation along the ventricle wall. This affects the flow for viscous adherence, giving a wall boundary layer whose thickness is proportional to \( \sqrt{\nu T} \); it takes values of few millimetres and is not expected to influence the bulk flow. An additional simulation has been performed in the healthy condition adding a differential rotation along the LV wall. With reference to clinical data, a rotation ranging from \(-15^\circ\) clockwise (if seen from the equatorial plane) at the apex to \(+10^\circ\) counter-clockwise at the base has been imposed during systole, releasing it during diastole. The rotation profile is distributed along the ventricular length to be zero at approximately half of the ventricle height, being the instantaneous velocity of rotation proportional to the radial one. Results are shown in Figure 7; as was expected, they are about indistinguishable from those in the absence of torsion (Figure 3(a) and (c)). Therefore, we can conclude with a good degree of confidence that the LV torsion, which plays an important role in the function of the myocardial tissue, has a negligible influence on the intraventricular flow.

5. Conclusions

The results presented were reproduced in an idealised LV under carefully controlled although simplified conditions. They show that in the presence of apical akinesia (Figure 4), and even more for dyskinesia (Figure 5), the flow enters less deeply inside the cavity. A stagnating region develops at the apex and close to the wall segment with reduced mobility, where the fluid is not cleared out by the vortex structure created by the mitral inflow. The little influence

Figure 6. Steady streaming flow on the transversal plane across mitral and aortic orifices of the LV in (a) healthy conditions (EF = 55%), in (b) condition of moderate regional akinesia (EF = 45%), (c) akinesia (EF = 40%) and (d) relevant dyskinesia (EF = 32%). Distribution of the normal component of the vorticity field and on-plane velocity vectors. Vorticity values, \( s^{-1} \), are shown in the colour bar. Unit vector corresponds to 10 cm/s.

Figure 5. Flow in the LV in regional dyskinetic condition, EF = 32% at peak diastole (a) and (b) \((t/T = 9/32)\), and during systole (c) and (d) \((t/T = 18/32)\). Panels (a) and (c): Flow on the transversal plane across mitral and aortic orifices, distribution of the normal component of the vorticity field and on-plane velocity vectors. Vorticity values, \( s^{-1} \), are shown in colour bars. Unit vector corresponds to 50 cm/s. Panels (b) and (d): 3D vortex structure visualised by isosurface of the \( \lambda_2 \) field, value \( \lambda_2 = -450 \text{ s}^{-2} \) and \( \lambda_2 = -200 \text{ s}^{-2} \), respectively.

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of twist and torsion on the intraventricular flow has been demonstrated. These information supply a physical back-
ground for phenomena that could realise in realistic 
physiological conditions, and encourage a clinical verifica-
tion to progress in diagnostic methods based on the study of 
the flow dynamics.

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